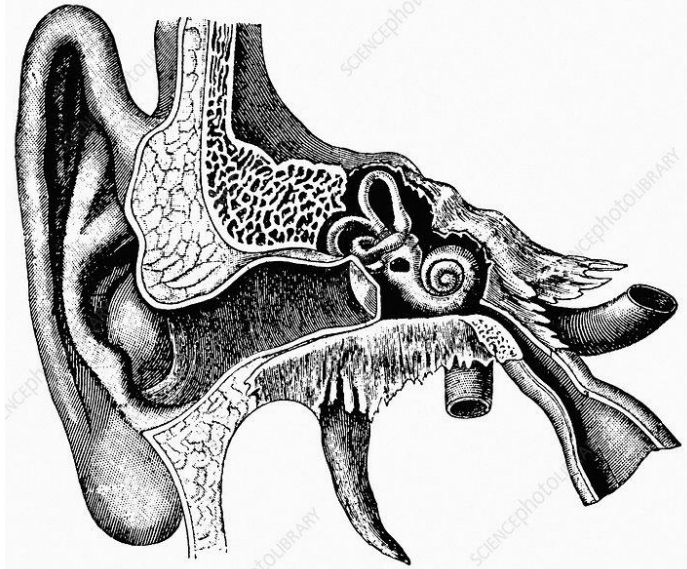


[Video link](#)

[Editing file](#)



REVIEWED BY
ABDULAZIZ ALSAIF



Deafness & Vertigo

Presented by Dr. Abdulaziz Alballa

★ Lecture Objectives:

- Conductive hearing loss (causes and management) (otosclerosis in brief)
- Sensorineural hearing loss (congenital and acquired), presbycusis etc
- Management of SHNL (in brief) {hearing aids and cochlear implant in brief}
- Causes of vertigo (acute and chronic labyrinthitis, Meniere's disease, vestibular neuritis, positional vertigo, etc)
- Investigation of a dizzy patient (in short)

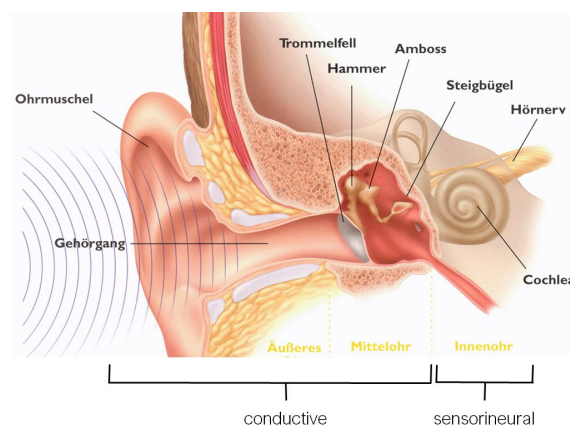
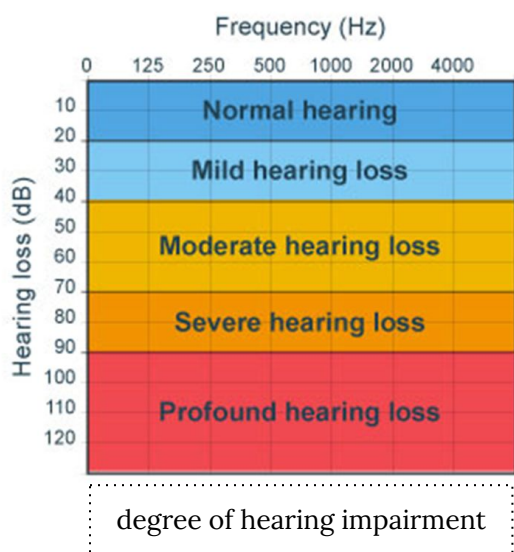
Color Index:

● Introduction

- Hearing loss : is define by the World Health Organization (WHO) as a hearing loss with thresholds ≥ 25 dB on one or both ear.
- Hearing impairment is partial or complete inability to hear from one side or both sides of the ear.

● Types of hearing loss

- Conductive
- Sensorineural



Conductive Hearing loss

| Outer Ear | Middle Ear |
|-----------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------|
| <ul style="list-style-type: none"> ● Inflammation ● Foreign body ● Trauma ● Malformation ● Tumor | <ul style="list-style-type: none"> ● Inflammation ● Trauma ● Malformation ● Tumor |

No conductive hearing loss in inner ear

Conductive Hearing loss

441 slides

- Conduction of sound to the cochlea is impaired.
- Can be caused by external and middle ear disease (auricle, Concha ,Ear canal , Drum , ossicular chain, Eustachian tube).

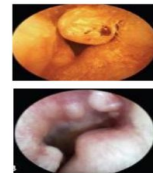
External canal pathology (Outer ear)

● Inflammatory:

- Herpes zoster optics
- Acute otitis externa: It's a common condition involving inflammation of the ear canal. The acute form is caused primarily by bacterial infection, with *Pseudomonas aeruginosa* and *Staphylococcus aureus* the most common pathogens, very painful. As a result of the inflammation, there will be bulging and obstruction
- Auricular perichondritis
- Otitis media: Acute suppurative (ASOM) – Otitis media with effusion (OME) - Chronic otitis media (CSOM).

● Obstruction: Treatment? Removal of the obstruction cause

- Wax: the commonest cause of conductive hearing loss (CHL) (439)
- Foreign body: like insect or Q-tips.
- Tumors: Benign or malignant (osteosarcoma, SCC, BCC).
 - Osteoma: Benign bone tumor, **single, unilateral, pedunculated** broad-base.
 - Exostosis: Benign bone overgrowth, **multiple, bilateral, broad-base**, in cold regions “swimming” (Austria and Scandinavia).
 - Squamous cell carcinoma and basal cell carcinoma



osteoma

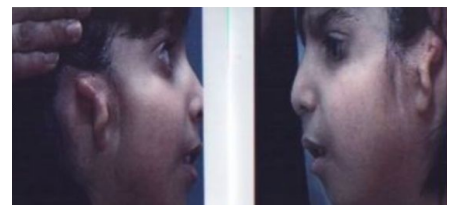
exostosis

● Trauma:

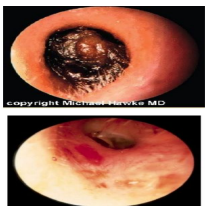
Ear Drum scarring; perforation Skull base fracture blood goes to the external auditory canal > tympanic membrane perforation > blood in the middle ear gives Raccoon eyes sign “periorbital ecchymosis” and battle’s sign. Ear drum Scarring.

● Congenital malformation:

- Atresia = رتق (no ear canal)
- Microtia = صمعا Deformity of the ear auricle
- Stenosis



This patient has abnormal auricle and has No ear canal (microtia) so the sound will not go through to stimulate the middle ear ossicles and tympanic member



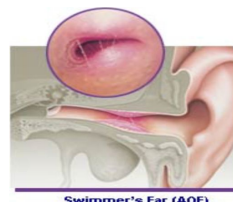
wax



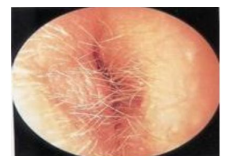
Battle's sign



Raccoon eyes sign



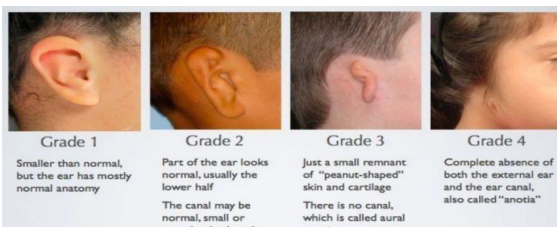
Swimmer's Ear (AOE)



Acute otitis externa



Foreign body



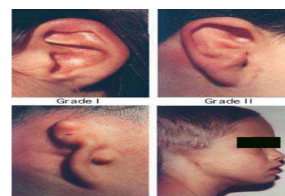
Grade 1
Smaller than normal, but the ear has mostly normal anatomy

Grade 2
Part of the ear looks normal, usually the lower half
The canal may be normal, small or completely closed

Grade 3
Just a small remnant of "peanut-shaped" skin and cartilage
There is no canal, which is called aural atresia

Grade 4
Complete absence of both the external ear and the ear canal, also called "anotia"

Microtia



Grade I

Grade II

Grade III

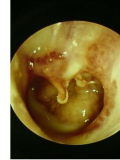
Anotia

Middle ear pathology

- **Inflammatory:**

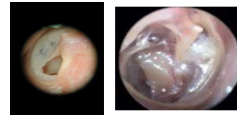
- Chronic otitis media (CSOM).

Chronic Suppurative otitis media



- **Trauma:**

- Barotrauma
- Penetrating injuries of the TM
- Disrupted trauma to the ossicles



Adhesive otitis media



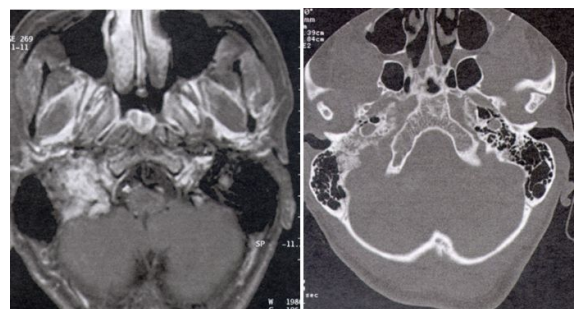
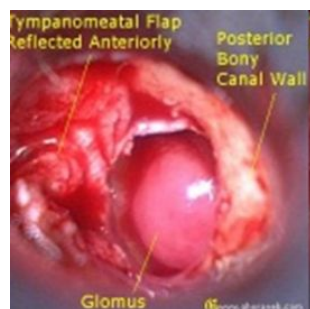
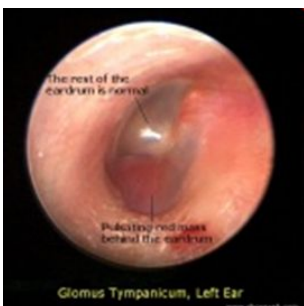
- **Congenital malformation:** As we know there are three ossicles in the middle ear (malleus, incus and stapes) any one affected it will end up with CHL

- Absent & erosion like in chronic ear inflammation or trauma
- **Fixation:** congenital or acquired: (**Otosclerosis** Fixation): the following information is from 439
 - Congenital inherited autosomal recessive disease causes fixation of the footplate (stapes) by new bone formation.**
 - A disease of the bony otic capsule characterized by abnormal replacement of mature bone of the otic capsule by woven bone of greater thickness which makes it harder for the stapes to move and transmit the sound.**
 - 10% otosclerotic lesions (10% symptomatic). (438)
 - Middle-age.**
 - Females: Male, 2: 1.
 - Occur in Caucasian and Europeans mostly.
 - Progressive bilateral hearing loss**
 - One feature that it Worsen during **pregnancy** and improve after delivery (due to hormonal changes).
 - Treatment:
 - Stapedectomy is a surgical procedure (if hearing aid did not work) in which the innermost bone (stapes) of the middle ear is replaced with a small plastic tube of stainless-steel wire “prosthesis” to improve the movement of sound to the inner ear.
 - Ossiculoplasty

- **Tumors:**

- Paraganglioma
- Squamous cell carcinoma
- Schwannoma
- Glomus tumor
Could be originated from carotid or jugular vein

Glomus tympanicum tumors are the **most common vascular tumors of the middle ear**. Glomus tumors may also arise in deep neck (parapharyngeal) space along the course of the vagus nerve

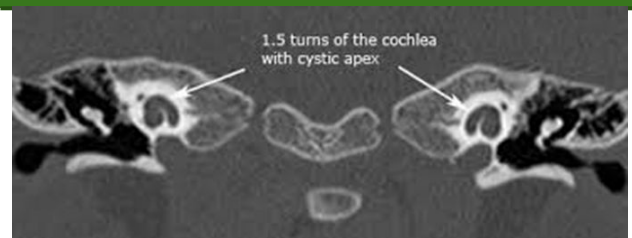


- SNHL is a defect in the conversion of sound into neural signals or in the transmission of those signals to the cortex.
- It can be caused by disease of the inner ear (cochlea), acoustic nerve (CNVIII), brainstem, or cortex.
- It has Two types:
 - **Sensory** (the pathology is within hair cells in cochlea).
 - **Neural** (the pathology is within the auditory nerve and its connection).
- **Etiologies:**
 - Congenital:** Malformation
 - Acquired :** Tumor - Inflammation - Trauma

External canal pathology (Outer ear)

- **Congenital malformation:**

- Mondini malformation
(Most common malformation of cochlea)

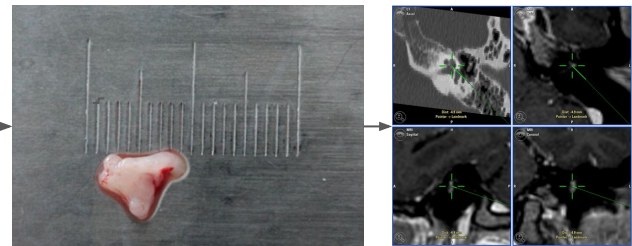


- **Tumors:**

- Vestibular schwannoma
Tumor removed
Stapes head

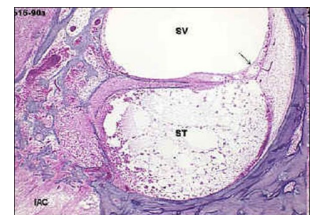


Intra-cochlear schwannoma



- **Inflammatory:**

- labyrinthitis ,meningitis
- Autoimmune (**Cogan syndrome**)

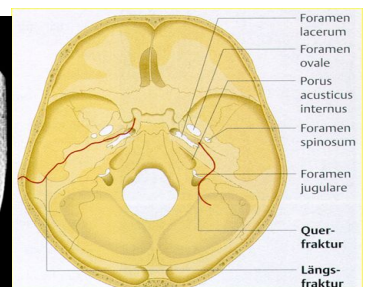


- **Trauma:**

- Temporal bone fracture (common in RTA)
Longitudinal fracture (hearing is preserved) or Transverse fracture (ear will be dead)
The cochlear will be affected when the temporal fracture is transverse
this is due to the anatomical position of it
- Noise exposure (acute or chronic)

- **Ototoxic drugs:**

- Aminoglycosides groups
- Loop diuretics
- Cisplatin



Audiogram:

- Pure tone audiogram: Is true testing of hearing sensation. “You should be a master at this by now:)”
 - Pure tone audiometry provides a measurement of hearing levels by AC and BC and depends on the co-operation of the patient, more than 20 decibel is hear loss
 - The test should be carried out in a soundproofed room. The signal is presented to the patient through earphones (for AC) or a small vibrator applied to the mastoid process (for BC). Signals of increasing intensity at each frequency are presented to the patient, who indicates when the test tone can be heard.
 - The threshold of hearing at each frequency is charted in the form of an audiogram (Figs 3.6–3.8), with hearing loss expressed in decibels (dB).
 - When testing hearing by BC, it is essential to mask the opposite ear with narrow-band noise to avoid cross-transmission of the signal to that ear.
 - Tests the intensity of sound and its frequency (test end at 8000 Hz), the patient decides the threshold by saying if he can or can't hear it.
 - Bone conduction always better in audiogram.

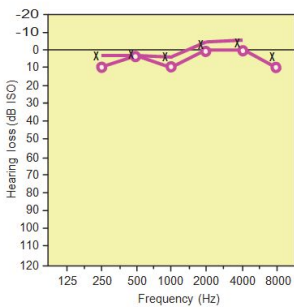


Figure 3.6 A normal pure tone audiogram. o-o-o, right ear; x-x-x, left ear.

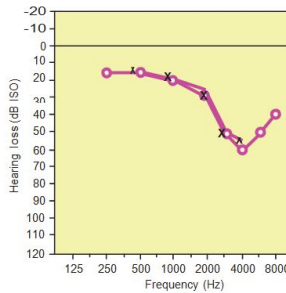


Figure 3.7 A pure tone audiogram showing sensorineural deafness maximal at 4 kHz typical of noise-induced deafness.

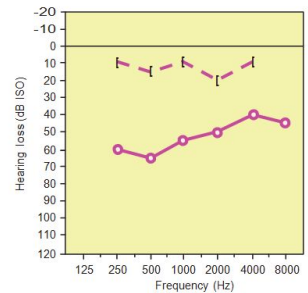
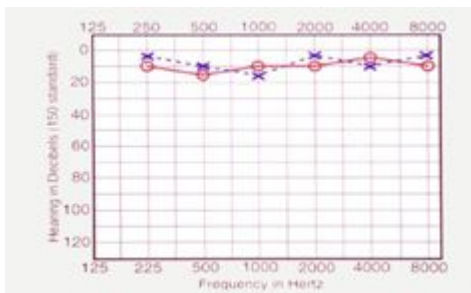


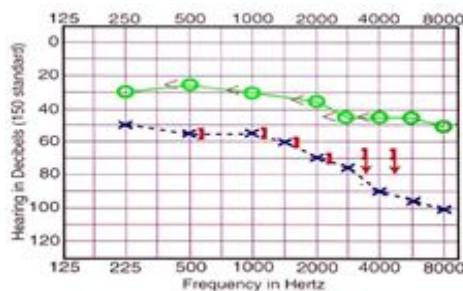
Figure 3.8 A pure tone audiogram showing conductive deafness. The BC (dashed line) is normal but the AC (solid line) is impaired. A case of otosclerosis.

Normal hearing



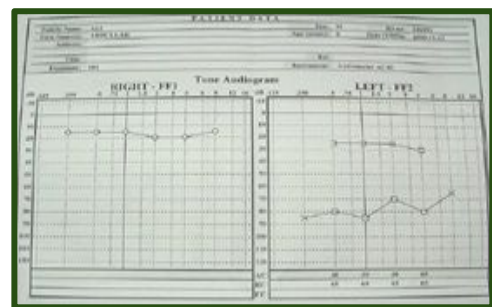
Normally the bone and air overlap each other

Mixed hearing loss



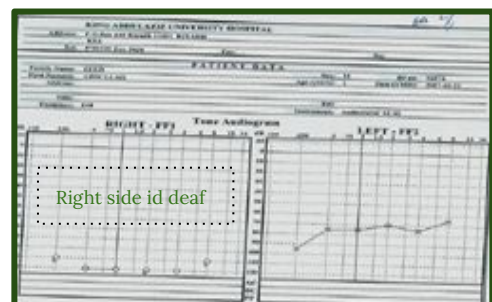
Both air and bone will be down but will NOT be overlapping (with a gap)

Severe Conductive hearing loss



once the bone is normal and air is down more the 10dB then we call it CHL

Severe Sensorineural hearing loss



Both air and bone will be overlapping and they will be down (without a gap)

● Signs of Hearing loss

01

Talking louder than necessary. تلاحظها في كبار السن

02

Turning up volume on the TV or radio.

03

Complaints that other people "mumble".

07

Lip reading:

- Watching a speaker's face intently
- Difficulty "hearing" someone behind
- Having difficulty speaking on the telephone

04

Confusion of similar sounding words نخلة/نحلة

05

Inappropriate responses in conversation.

06

Ringing or buzzing in the ears.

● The impact of hearing impairment:

- Affects Speech if the input is flawed then the output will be flawed too/ if you listen to something in a wrong way then you'll repeat it wrong too, and affecting severity depends on deafness severity.
- Language
- Education
- Social They'll feel left out/isolated and depressed. They'll have thoughts like "what if others are talking about me"
- New studies that show hearing loss is associated with dementia, and the incidences decreased if hearing aids have been used.
- Limit activities, Isolation, Depression, Anxiety, Insecurity, strain relationships, Increase psychosocial difficulties

● Effects of Hearing loss

- Don't enjoy conversations- too much work
- People think you are an idiot
- Scared to try new contacts
- Scared to take new jobs
- Limits your world
- Increases psychosocial difficulties
- Limit activities
- Isolation
- Depression
- Anxiety
- Insecurity
- Strain relationships

● High Risk Criteria For Hearing Loss in Infants:

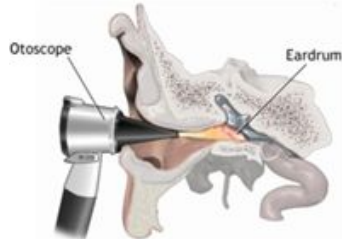
- Family history of hereditary childhood sensorineural hearing loss. First family relatives.
- Hyperbilirubinemia post-delivery, there are two types mild and severe (need transfusions, more risk 'worse')
- Ototoxic medications. Such as Aminoglycosides or chemotherapy like Methotrexate
- Bacterial meningitis, transfer to cochlea
- Birth weight less than 1500 grams, lower body weight = higher chance of having deafness
- In utero infections (toxoplasmosis, syphilis, rubella, cytomegalovirus and herpes), Imp to ask in history
- Craniofacial anomalies (including pinna and ear canal).
- Birth asphyxia.
- Mechanical ventilation lasting 5 days or longer.
- Stigmata or other findings associated with a syndrome known to include a sensorineural and/or conductive hearing loss. Risk factors for adults such as trauma or noise exposure in airplanes (not wearing ear plugs)

After proper Hx keeping in mind all the possibilities regarding the causes and etiology of CHL and SNHL . THEN do full examinations

- General look (syndromic features)
- Complete head and neck exam

Otoscopic / microscopic ear exam for both ears:

the microscope is better because we can see 1- tympanic membrane 2- external auditory canal 3- middle ear



Tuning forks:



- There are 2 tests:

1. **Weber test:** is a quick screening test for hearing. You need to know how to interpret the result.

- It can detect:
 - Unilateral conductive hearing loss (middle ear hearing loss), **deviated to affected ear.**
 - Unilateral sensorineural hearing loss (inner ear hearing loss), **deviated to better ear.**
- The test is useful in determining the type of deafness and in deciding which ear has the better-functioning cochlea. The base of a vibrating tuning fork is held on the middle of the skull and the patient is asked whether the sound is heard centrally or is referred to one or another ear.
- **Interpretation:**
 - In conductive deafness the sound is heard in the deaffer ear. Eliminated outside noise
 - In sensorineural deafness the sound is heard in the better-hearing ear.

2. **Rinne test:**

- Evaluates hearing loss in one ear, it compares perception of sounds transmitted by air conduction to those transmitted by bone conduction through mastoid. **normally air conduction is better.**
- **This test compares hearing in one ear by air conduction (AC), and bone conduction (BC). It is usually performed as follows:**
 - a tuning fork of 512Hz (cycles per second) is struck and held close to the patient's ear (AC); the base is then placed firmly on the mastoid process behind the ear (BC) and the patient is asked to state whether it is heard better by BC or AC (fig. 3.3-check it)
- **Interpretation of Rinne's test:**
 - * If **AC > BC (called Rinne positive)** the middle and outer ears are functioning normally.
 - * If **BC > AC (called Rinne negative)** there is defective function of the outer or middle ear (conductive deafness).
- Try this on yourself. Then gently occlude your outer ear by pressing the tragus, giving yourself a mild temporary conductive deafness. Now repeat the test and you should find that Rinne becomes negative, demonstrating the conductive loss.
- Rinne's test tells you little or nothing about the cochlea. It is a test of middle-ear function.

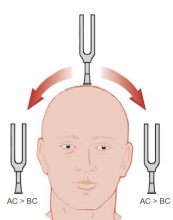


Figure 3.3 Tuning fork tests showing a positive Rinne in each ear and the Weber test referred equally to each ear, indicating symmetrical hearing in both ears with normal middle-ear function.

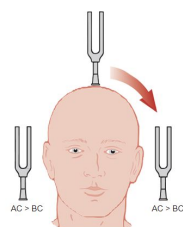


Figure 3.4 Sensorineural deafness in the right ear. The Rinne test is positive on both sides and the Weber test is referred to the left ear.

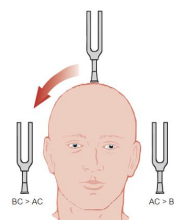
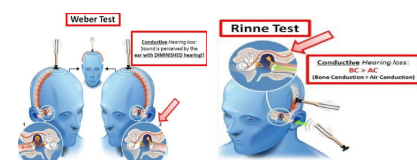


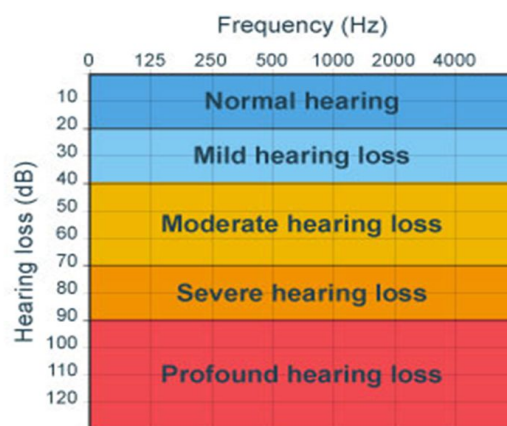
Figure 3.5 Conductive deafness in the right ear. The Rinne test is negative on the right, positive on the left, and the Weber test is referred to the right ear.



- **Speech audiogram:**

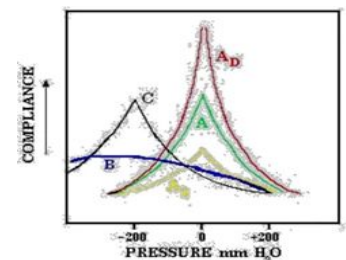
- Speech audiometry measures the ability of each ear to discriminate the spoken word at different intensities.
- A recorded word list is supplied to the patient through the audiometer at increasing loudness levels, and the score is plotted on a graph.
- In some disorders, the intelligibility of speech may fail above a certain intensity level.
- Above a critical threshold, sounds are suddenly perceived as having become excessively loud – loudness recruitment. This suggests a cochlear disorder and is common in elderly patients with presbycusis.

Degree Of Hearing Impairment



- **Tympanogram:**

- It tests
 - Volume
 - Acoustic reflex decay
- It's not a test of hearing, it is a test of **mobility of tympanic membrane** and it will tell us about the status of the tympanic membrane and middle ear status.
- It's a graphic representation of the relationship between the air pressure in the ear canal and the movement of the tympanic membrane.
 - Type A: normal
 - * Type Ad: ossicular disruption with normal TM
 - * Type As: ossicular fixation
 - Type B: fluid (effusion) or perforated tympanic membrane. How to distinguish? By ear canal volume, if high= perforation, if low= effusion. we can distinguish between them by Ear canal volume, if it's high= perforated membrane with or without effusion, if it is normal= OM with effusion, if it's low= Wax/foreign body
 - Type C: peak at the negative side (normally at 0 mm H₂O) = negative pressure in the middle ear (dysfunction).



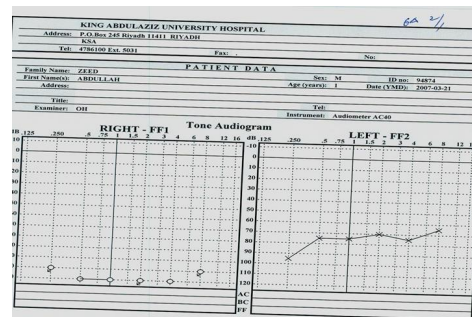
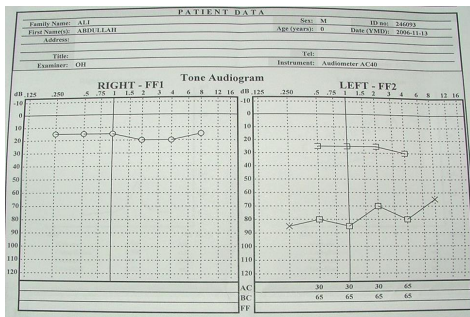
- **Impedance: Acoustic reflex**

- **ABR (Auditory brainstem response)**

- **Emission**

Hearing loss (438 slide)

| Conductive Hearing Loss | Sensorineural Hearing Loss |
|------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------|
| Negative Rinne test (BC > AC) | Positive Rinne test (AC > BC) |
| Weber lateralized to the poorer ear | Weber lateralized to better ear |
| Normal absolute bone conduction | Bone conduction reduced |
| Low frequencies affected more | More often involving high frequencies |
| Audiometry shows bone conduction better than air conduction, with air bone gap. Greater the air bone gap, more is the conductive loss | No gap between air & bone conduction curve on audiometry |
| Loss is not more than 60 dB | Loss may exceed 60 dB |
| Speech discrimination is good | Speech discrimination is poor |
| | There is difficulty in hearing in the presence of noise |



- Infection (chronic otitis media) and trauma might cause mixed HL.

- Conduction of sound to the cochlea is impaired.
- Can be caused by external and middle ear disease (auricle, Concha ,Ear canal , Drum , ossicular chain, Eustachian tube)
- The lesion may lie in the external ear and tympanic membrane, middle ear or ossicles up to stapedio vestibular joint. (433)

1. External canal pathology

- **Congenital:**
 - Atresia = رتق (no ear canal)
 - **Microtia** = صمعاى Deformity of the ear auricle
 - **Anotia** = no auricle at all
- **Inflammatory:**
 - Otitis media: Acute suppurative (ASOM) – Otitis media with effusion (OME) - Chronic otitis media (CSOM).
 - Acute otitis externa: It's a common condition involving inflammation of the ear canal. The acute form is caused primarily by bacterial infection, with Pseudomonas aeruginosa and Staphylococcus aureus the most common pathogens, very painful.
- **Obstruction:**
 - **Wax: the commonest cause of conductive hearing loss (CHL)**
 - Foreign body (438): any form of obstruction can cause CHL like insect or Q-tips
 - Tumors(438): **Benign or malignant (osteosarcoma, SCC, BCC).**
 - **Osteoma: Benign bone tumor, single, unilateral, pedunculated** broad-base.
 - **Exostosis: Benign bone overgrowth, multiple, bilateral, broad-base, in cold regions** "swimming" (Austria and Scandinavia).
 - **Squamous cell carcinoma and basal cell carcinoma**
 - Ear Drum scarring; perforation Trauma: Skull base fracture blood goes to the external auditory canal > tympanic membrane perforation > blood in the middle ear gives Raccoon eyes sign "periorbital ecchymosis" and battle's sign. Ear drum Scarring.



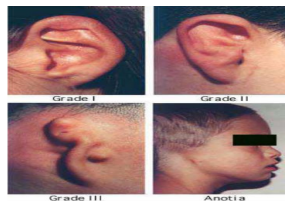
osteoma



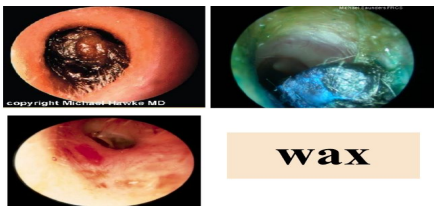
exostosis



Microtia



This patient has abnormal auricle and has No ear canal (microtia) so the sound will not go through to stimulate the middle ear ossicles and tympanic member



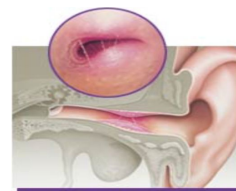
wax



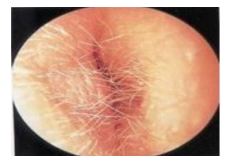
Battle's sign



Raccoon eyes sign



Swimmer's Ear (AOE)



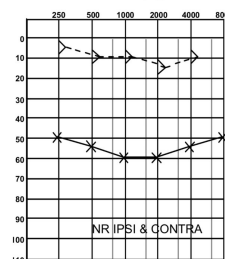
Acute otitis externa



Foreign body

Case(438)

(Conductive hearing loss)



Left ear

2. Tympanic membrane pathology

- Tympanic membrane amplify sound*17 times , It should be tense.
- Tympanic membrane pathology
 - a. **Absent TM caused by perforation:** Fresh blood indicates a recent injury (acute injury).
 - b. Too thick TM caused by **tympansclerosis**. which can Impair the sound transmission to the tympanic membrane
 - i. Tympanosclerosis: Calcification of an old inflamed tissue, **whitish, sclerotic plaques**. (usually it's asymptomatic -most of the time-, but when it's symptomatic it causes CHL) (ask about previous infection in the ear while taking the history because it comes from recurrent infections), It's a condition characterized by the presence of masses of hard, dense connective tissue around the auditory ossicles in the middle ear, also known as myringosclerosis.
 - ii. **Multiple surgeries or infections (myringitis) causes TM scarring and thickening. This could fix the drum and ossicles causing conductive hearing loss.** .
 - c. Too thin TM caused by Secretory otitis media (SOM), Retraction

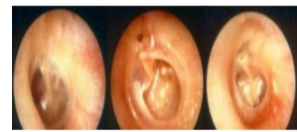


Fixed TM , Fixed ossicles white patches; old infections (Tympanosclerosis)

Middle ear effusion

3. Drum Retraction(438) (Adhesive OM):

- It's also called Atelectasis ear.
- The tympanic membrane gets sucked in because of eustachian tube dysfunction and negative pressure, which will suck the ear-drum inside. We treat it by ventilation tube, which prevents the ear from getting sucked inside by preventing the negative pressure. So perforation and retraction both of them are causes of conductive hearing loss.
- Treatment of adhesive OM is attachment of tube.



Drum Retraction (Adhesive OM)

4. Ossicular chains:

- As we know there are three ossicles in the middle ear (malleus, incus and stapes) any one affected it will end up with CHL
- 1. Absent & erosion like in chronic ear inflammation or trauma
- 2. **Fixation:** congenital or acquired:
 - a. **Otosclerosis:**
 - i. **Congenital inherited autosomal recessive disease causes fixation of the footplate (stapes) by new bone formation.**
 - ii. **A disease of the bony otic capsule characterized by abnormal replacement of mature bone of the otic capsule by woven bone of greater thickness which makes it harder for the stapes to move and transmit the sound.**
 - iii. 10% otosclerotic lesions (10% symptomatic). (438)
 - iv. **Middle-age.**



4. Ossicular chains: cont..

a. **Otosclerosis:** cont..

v. Females: Male, 2: 1.

vi. Occur in Caucasian and Europeans mostly.

vii. **Progressive bilateral hearing loss**

viii. One feature that it Worsen during **pregnancy** and improve after delivery (due to hormonal changes).

ix. Treatment:

- Stapedectomy is a surgical procedure (if hearing aid did not work) in which the innermost bone (stapes) of the middle ear is replaced with a small plastic tube of stainless-steel wire “prosthesis” to improve the movement of sound to the inner ear.

- Ossiculoplasty

x. It has nothing to do with infection.

3. Disrupted trauma or **dislocation**.



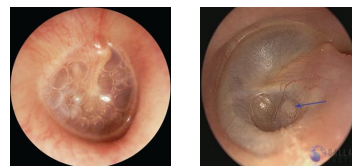
Ossiculoplasty



stapedectomy

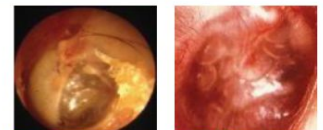
5. Eustachian Tube dysfunction:

- As we know the tube is a part of the middle ear with the Nasopharynx and it's main job is to ventilate the middle ear and equalize the pressure so any defect will end up with OM
- Retraction, Blocking of the eustachian tube lead to negative pressure and retraction.
- **Effusion**, Accumulation of fluids behind tympanic membrane, normally there's Air in middle ear.
- Otitis media: acute suppurative (ASOM), otitis media with effusion (OME), chronic otitis media (CSOM).
- Congenital cholesteatoma
- Squamous cell carcinoma
- Paraganglioma
- Schwannoma



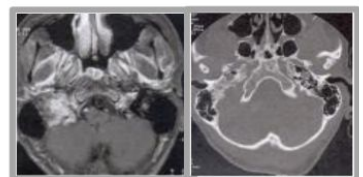
Air-bubbles

Extra



Retraction

Effusion



Sensorineural Hearing loss

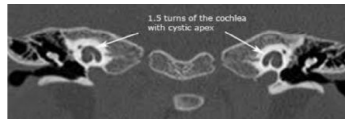
- SNHL is a defect in the conversion of sound into neural signals or in the transmission of those signals to the cortex
- It can be caused by disease of the inner ear (cochlea), acoustic nerve (CNVIII), brainstem, or cortex.
- It has Two types:
 - **Sensory** (the pathology is within hair cells in cochlea).
 - **Neural** (the pathology is within the auditory nerve and its connection) -
- Etiologies(438):
 - Congenital
 - Infection
 - Ototoxic
 - Presbycusis
 - Age
 - Trauma
 - Noise
 - Acoustic neuroma it may be congenital or acquired

A. Congenital (438):

A. Congenital:

1. Inherited
2. Syndromic (less common)
3. Non-syndromic (more common).
4. Congenital infection (TORCH): "Toxoplasmosis, Others (syphilis, varicella-zoster, parvovirus B19), Rubella, Cytomegalovirus, Herpes simplex" Can lead to delay speech and language development.
 - Deafness affects 0.2%
 - SNHL attributed to:
 - 50% genetic factors
 - 20-25% environmental
 - 25-30% sporadic
 - Over 400 syndromes

- Genetic:
 - 75% AR
 - 20% to AD
 - 5% X-linked



Mondini malformation



Waardenburg syndrome (white forelock)

Treacher Collins syndrome (may have conductive HL).

B. Acquired :

1. Trauma (438)

- Temporal bone fracture: affect hearing, balance and facial nerve.
 - Longitudinal fracture:
 - Bleeding from ear
 - **Conductive hearing loss**
 - Uncommon facial nerve paralysis
 - CSF
 - Transverse fracture:
 - **SNHL**
 - Facial nerve paralysis common.
 - CSF
 - Mixed



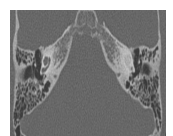
The directions of fracture from lateral to medial



The line going from posterior to anterior

2. Inflammatory

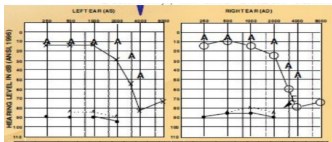
- **Meningitis: 30% of children with meningitis will have cochlear ossification**, in meningitis it goes to cochlear through natural connection of the brain and ear causing ossification, and the new bone being made causes death of outer hair cells, some say the connection is through a blood supply, also it may affect Wernicke's and the temporal lobe causing neural hearing loss
- Labyrinthitis (438)



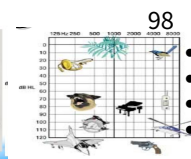
B. Acquired :

3. Noise Exposure Induced SNHL

- Boilermaker's deafness.
- One of the most common occupationally induced disabilities.
- **Those who are working on very noise environment like an airport, factories without protecting their ears they have a special type of hearing loss which is noise induced hearing loss . Follows chronic exposure to less intense sounds than seen in acoustic trauma and is mainly a hazard of noisy occupations. Here its cumulative like blow drying it is NOT REVERSIBLE** (That's why they have devices that measure the noise in factories and airports, loudspeakers in weddings have the same effects)
- Patients will present with Tinnitus only sign, earliest sign:
 - commonly accompanied Noise induced SNHL
 - warning sign
- **Warning sign:** (one gunshot could cause SNHL, and in KSA fireworks), hunting or explosions are one time highly intense shots causing hearing loss straight away, causes damage to hair cells, starting in the basal turn of cochlea. Outer hair cells are affected before the inner hair cells



We can see a normal hearing then a sudden drop at 4000 hz , so any Sensorineural drop of hearing at 4000 we called it noise induced hearing loss



- 90 db for 8 hours
- 95 db for 4 hours
- 100 db for 2 hours
- 105 db for 1 hours

4. Acoustic neuroma (Vestibular Schwannoma):

- Vestibular Schwannoma is a benign nerve tumor in the internal auditory meatus or cerebello-pontine (CP) angle at the base of the skull. It is usually unilateral, except in the very rare familial neurofibromatosis type 2 (NF2), when it may be bilateral. In its early stages, it causes progressive hearing loss and imbalance. As it enlarges, it may encroach on the trigeminal nerve in the CP angle, causing loss of corneal sensation. In its advanced stage, there is raised intracranial pressure and brain stem displacement. Early diagnosis reduces the morbidity and mortality. Unilateral sensorineural deafness should always be investigated to exclude a neuroma. Audiometry will confirm the hearing loss. MRI scanning will identify even small tumors.



5. Autoimmune (438)

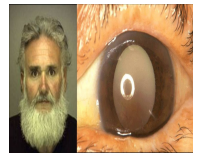
- (Cogan syndrome) with SLE or PRH
- Behcet's syndrome
- RA

B. Acquired :

6. Presbycusis(438)

- **Aging process of human beings, it's associated with grey hair, cataract and SNHL, most common type.** (age related hearing loss, at age 40 we expect patients to have cataract and hearing loss)
- Presbycusis = Deafness + Tinnitus “buzzing” + Recruitment “out of proportion of loudness” (recruitment meaning the patient can't hear, but when he hear, he hear everything louder than it's normal range. Patient will say ‘ why are u screaming?’ The cochlea normally acts as a filter; it decreases loud voices and amplifies the low sounds, here the cochlea is not functioning well)
- Overview of Hearing Loss:
 - #1 handicapping disorder
 - 60% of Americans > 65 HL
 - 90% of > 75 Y have HL
 - HL + degenerative processes of aging
 - Half vestibular symptoms
- Problems with diagnosis includes: shame or embarrassment, HA social stigma, embarrassment prevents 15 million elderly people from getting help.

Inner ear, lenses and hair all derived from ectoderm and all involved in the aging process
Inner ear → presbycusis
Lens → cataract
Hair → white “grey”



7. Ototoxic(438)

- **Antibiotics** “aminoglycosides”, like **Gentamicin**.
- Diuretics, Furosemide they are known to cause oedema and cystic changes in the stria vascularis of the cochlear duct
- Antineoplastics
- Anti Inflammatories
- Antimalarial agents (chloroquine, quinine)
- Ototoxic agents
- Others
- Patients particularly at risk are those: Concomitantly receiving other ototoxic drugs, who have already received aminoglycoside antibiotics, who are receiving high doses of ototoxic drugs with high serum level of drug, who have genetic susceptibility to aminoglycosides (433 team).
- **Higher risk patients:**
 - Renal failure (Elevated peak and trough levels)
 - Liver failure
 - Immunocompromised
 - Collagen-vascular disorders
 - Advanced age (> 65 years)
 - Prior ototoxicity
 - Concurrent use of known ototoxic agents
 - Bacteremia (fever)
 - Treatment course longer than 14 days
 - + ve FHx of AG ototoxicity
 - Preexisting HL or Vestibular

Treat the underlying cause

01

Medical:

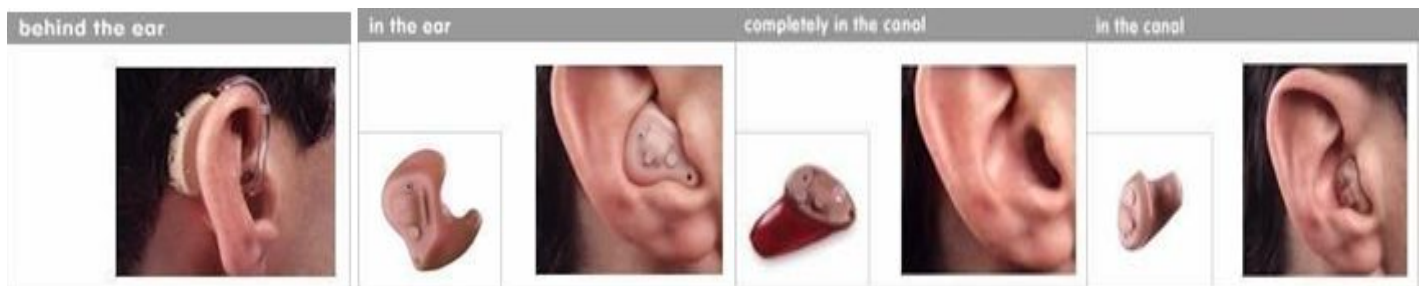
Which pathology can manage with medical ? OM with effusion, otitis externa, some time labyrinthitis, noise related, meningitis and autoimmune disease can be managed with steroids

02

Hearing Aid:

They magnify the sound so it reaches the cochlear amplified.

For any HL type (for conductive, Sensorineural and Mixed HL) “perforation, nerve disturbance” Hearing aids are better in sensory rather than neural loss.



● Surgery:

1

Myringoplasty & ventilation tube:

In case of otitis media with effusion most common procedure performed (done in an eustachian tube infection) **ventilation tube indication:** 1-Persistent effusion 2-Eustachian tube dysfunction 3-Recurrent acute otitis media

2

Myringoplasty & tympanoplasty:

- **Tympanoplasty** (patching as well as reconstruction on the ossicles) **in case of CSOM for 2 indications**
 - To improve hearing
 - To avoid infections

One of the above is enough reason to do tympanoplasty, if the surgery won't affect them then it's discussed with the patient, if decided not to do it then the patient should not swim and be careful with showers so no water can enter the ear

- Myringoplasty > we do only patching of the tympanic membrane

3

- Ossiculoplasty:

- In case of ossicular discontinuity. Replace any one of the ossicles with artificial ossicles
- Either partial or complete (in case all 3 ossicles are involved) ossiculoplasty.

- Stapedectomy

4

Bone Anchored Hearing Aids (B.A.H.A):

- Titanium implants, used in **CHL** (they use titanium because it doesn't react with the body)
 - **BAHA stimulates the cochlea by transmitting the sound waves through the bones in our skull, or bone conduction, thereby bypassing the outer and the middle ear.**
 - Atresia of external ear canal, and microtia
 - Chronic drainage ear not responding to surgery
 - Done for all types of HL (**mainly conductive**) it skips the external and middle ear and goes straight to the cochlear and stimulates the bone directly.
 - Someone with bilateral broken cochlear. (Blind and deaf)
 - We can also reach the brainstem and due to the presence of the cardiac center you can stimulate it and the patient may die but they still want the procedure to be done so it says a lot about the importance of hearing.
-

5

Auditory brainstem implant (A.B.I) (438):

- Implant in the brains
 - When there's no cochlea or nerve we bypass them directly to brainstem. After the procedure is done there won't be a noise protective mechanism so we send the patients to the ICU to monitor them from any cardiorespiratory complication (because cardiac and respiratory centers in brainstem).
-

6

Cochlear implant:

- Audiogram shows **bilateral profound sensorineural hearing loss can be an indication.**
- Patient with SNHL can be managed with hearing aid 🧠 but some time the hearing loss is beyond the capability of the hearing aid and these type of patient we do cochlear implant.
- Putting tiny electrode in the cochlea.
- Prelingual children and postlingual adult (ex; due to trauma)
- It bypass the external, middle and inner ear to stimulate the auditory nerve directly.
- In congenital HL the cochlear implant is ineffective after 5 years (The child will be prelingual and the child would've already learned sign language so it will be difficult to adapt), due to the disappearance of auditory segment from the brain (it gets used up by other centers of other senses that's why their other senses become better like vision for example). But in people who used to hear and then lost their hearing there is no time limit for the usage of cochlear implant, but we prefer to implant within 10 years.
- It's a device consisting of a microphone, signal processor, external transmitter, and implanted receiver; **the receiver is surgically implanted under the skin near the mastoid process above and behind the ear, it stimulate the auditory nerve directly skipping external, middle and inner ear.**
- **The surgery has a risk of hitting the facial nerve**
- So, in the exam if they gave you a cochlear implant picture you should know it, it looks like a regular hearing aid but with a magnet from outside.

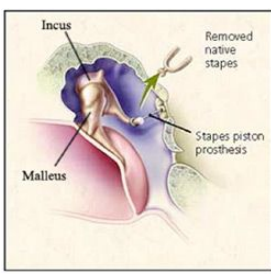
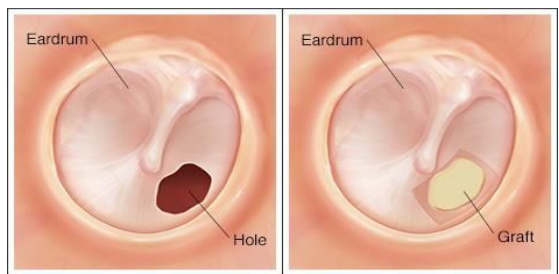
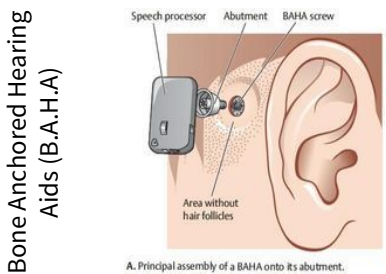
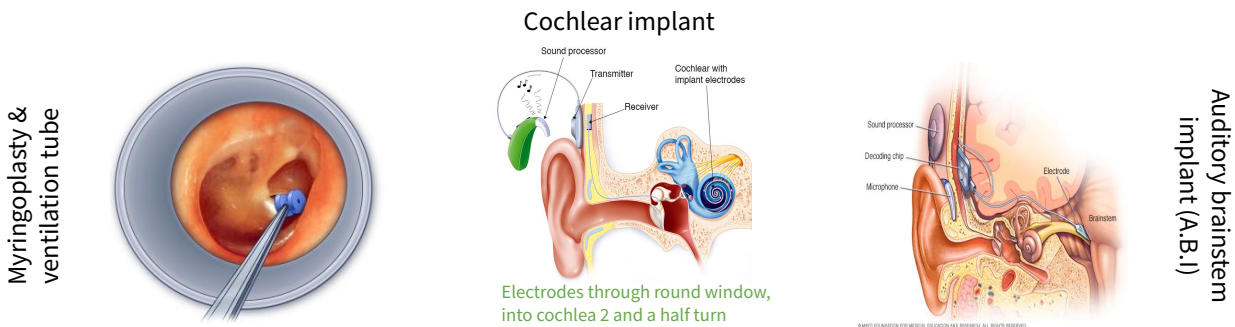
Management of hearing impairment:

441 Slides

- Classical indication of cochlear implant (Candidate):
 - Bilateral sensorineural hearing loss not benefiting from hearing aids, and less than 5 years of age if congenital hearing loss or an adult who is verbal .
 - It is standard practice everywhere to implant for children under 5 years old because of the improvement in the quality of life.
 - We have to make sure that the patient has a cochlea and a nerve prior to implant.

Factors improve/indicate better outcome after cochlear implants:

- Pt used to hear and knows how to speak (postlingual adult).
- Pt who used hearing aid
- Implantation for both ears
- For prelingual child, the earlier the better
- Cooperative environment (parents)



For conductive hearing loss

Myringoplasty & tympanoplasty

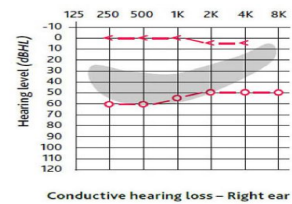
Others management:

- Lip Reading
- Signs Language



Case 1

- History:
 - 12 years old child present with unilateral hearing loss since birth
 - No otorrhea
 - No vertigo
 - No tinnitus
 - No history of trauma
 - No medical problems
- Examination: absence of the right EAC (Rt microtia) and aural atresia
- Pure Tone Audiogram: Right conductive hearing loss
- **Tuning fork:**
 - **Weber: Deviated to the right ear**
 - **Rinne: negative (AC<BC)**
- **Maximum conductive element : 60 dB**
- Management: BAHA (if there is no external auditory canal we can't do surgery because it will reclose itself)

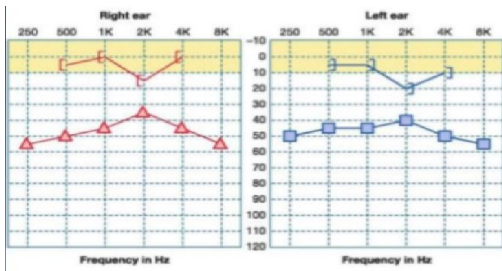
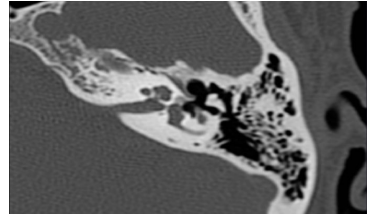


Case 2

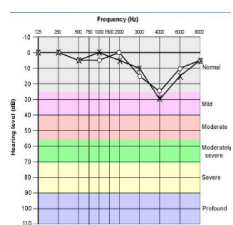
- History:
 - 27 year old woman presented with history of right hearing loss since 3 years
 - Recurrent otorrhea related to water
 - No vertigo
 - No tinnitus
- Examination: grade IV TM perforation, absent TM and ossicles are visible
- Pure Tone Audiogram: Right conductive hearing loss
- **Tuning fork:**
 - **Weber: Deviated to the right ear**
 - **Rinne: negative (AC<BC)**
- **Maximum conductive element : 30-40 dB dB**
- Management: Tympanoplasty



Case 3

- History:
 - 35 year old woman presented with progressive bilateral hearing loss since 4 years
 - No otorrhea
 - No vertigo
 - No tinnitus
 - No Recurrent ear infection
 - No Hx of medications, no medical problems
- Examination: Normal tympanic membrane under microscope
- Pure Tone Audiogram:
 - Tuning fork:
 - **Weber: To the affected ear**
 - **Rinne: Negative (AC<BC)**
- CT scan temporal bone: ossicular fixation (otosclerosis)
- Management: Ossiculoplasty or Stapedectomy

Case 4

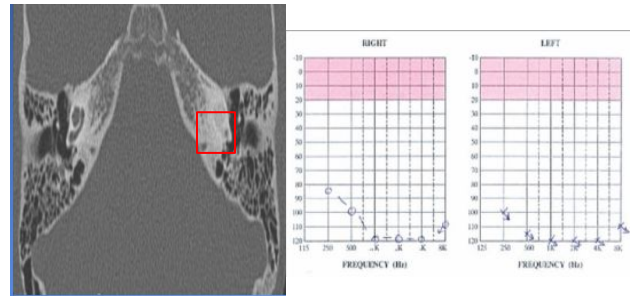
- History:
 - 47 year old man present with history of bilateral ear tinnitus since 8 years
 - Continuous with changing intensity
 - High frequency type
 - No vertigo
 - No otorrhea
 - No ear surgery
 - No medical problems
 - What is missing in the History? **Occupation**
- Pure Tone Audiogram: Bilateral sensorineural hearing loss over 4000 Hz
- Management: Hearing aids and medical management.

Case 5

- History:
 - 6 year old child presents with bilateral profound SNHL post meningitis at age of 3 years.
 - Progressive hearing loss
 - No problem during pregnancy
 - No ICU admission
 - No Recurrent otitis media
 - No localization of sound

Risk factors for hearing loss in children

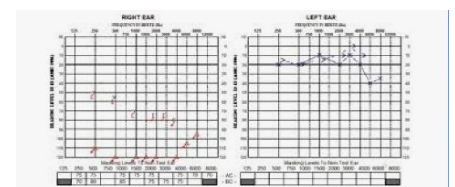
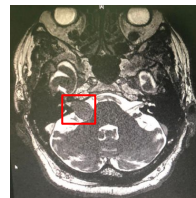
- Examination: Normal tympanic membrane bilateral
- Pure Tone Audiogram (it's not easy to do audiogram for children so sometimes we depend on ABR):
- ABR: absent bilateral
- Speech evaluation: good language
- MRI IAC / CT IAC: Left cochlear ossification



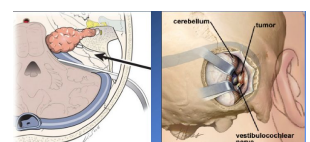
- Management: Cochlear Implant in the right ear **we can't do it in the left side.** Could we use a trial of hearing aid for 6 months? **No because if one side is ossified the other side will be ossified so it's an emergency and we do the implant as soon as we can**

Case 6

- History:
 - 56 year old woman presents with unilateral hearing loss since 8 years
 - Mild instability
 - Right ear tinnitus
 - No true vertigo
 - No recurrent ear infections
 - No medical problems
 - No medications



- Examination: Normal TM bilateral
- Pure Tone Audiogram: Bilateral sensorineural hearing loss over 4000 Hz
 - SDS (speech discrimination score) **the patient is asked to repeat 10 words :**
 - Left ear 100%
 - Rt ear 10% **this means he repeated only 1 word out of 10**
- MRI IAC: RT VS right CP angle which makes it most likely schwannoma
- Management: Surgery through the ear or behind the ear



Secretory Otitis Media (Glue Ear)

- 3 Y
- Recurrent OM
- Hearing Loss



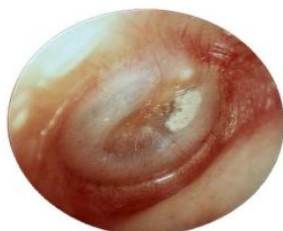
Fracture Base of Skull

- MVA
- Left earache
- Hearing loss



Otosclerosis vs Tympanosclerosis

- 33 y
- No hearing loss
- Ear exam →
Tympanosclerosis



Cochlear implant

- What is this?
Cochlear implant
- Which type of hearing loss?
Indication: bilateral SNHL

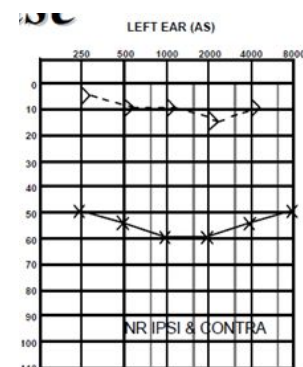


15 years old girl

- What is this?
BAHA
- Which type of hearing loss?
Conductive hearing loss



Conductive hearing loss



Vertigo

Introduction

What are the balance organs? more than just vestibule

1 Inner ear (3 semicircular canals and otolith organ):

If you have severe symptoms of dizziness this is most probably a problem with the inner ear or cerebellum. vision and proprioception problems rarely cause severe symptoms.

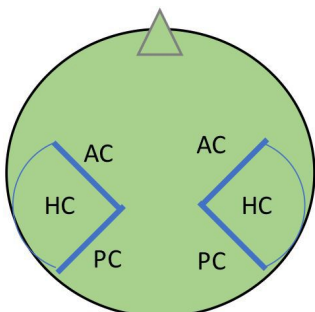
2 Cerebellum.

3 Vision (VOR-Vestibulo Ocular reflex): *The strongest reflex in our body*

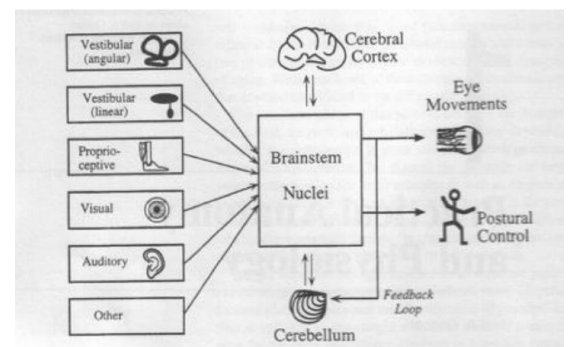
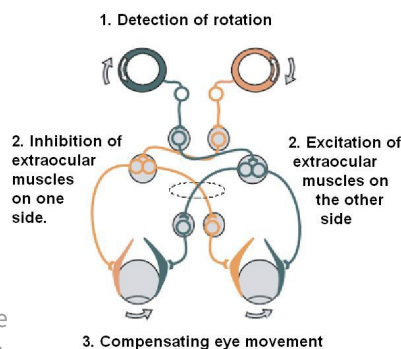
- To stabilize images on the retina during head movement, by moving the eye in direction opposite to the direction of the head, thus keeping image on the center of the visual field. You can test this by performing the finger test: if you move your finger fast and try to follow it with your eyes, the image will not be clear. But if you stabilize your finger and move your head side to side while looking at the finger the finger will be very clear. Why is that? in the first scenario we depend on the eyes to stabilize the finger's image on the retina, but it failed. In the second scenario we are depending on vestibular organ in the inner ear to control the eye movement while head shaking. This is why people with vestibular problems easily get dizzy because there's mismatching between the ear and what they see.
- In High velocities and in the dark our inner ear is what control our eyes via this reflex.**
- Posture control.
- The anatomical components of VOR are:
 - Semicircular canals.
 - Vestibular and oculomotor nuclei in the brainstem.
 - Extra-ocular muscles.

4 Proprioception (Muscles Tone & Joints) To know the position of your joints.

5 Cerebral cortex.



The semicircular canals are paired. We have Horizontal, Anterior, and Posterior (the left and right sides are parallel to each other). They act as sensors to detect any angular movement of the head.



All these inputs (stimulus of an organ) integrate and go to the brain in a systematic way that allows the brain to generate a reflex.

Vertigo

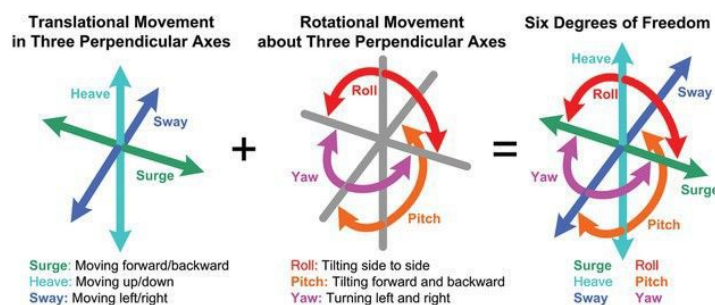
● Physiology of balance and function of vestibular system:

- The body's sense of equilibrium is maintained by input from a number of sources. These include the (eyes, proprioceptive organs especially in the muscles and joints of the neck, peripheral nerves, the labyrinth or 'balance organ' in the inner ear which includes the vestibule and semicircular canals and the cerebral cortex and cerebellum).
- Input from all these sources converge in the brain stem; dysfunction of any of these systems may lead to imbalance, a feeling of unsteadiness, 'vertigo' – a sensation of movement – and a tendency to fall.
- Vertigo may be accompanied by 'nystagmus' – a rapid beating of the eyes to one side – as impulses from the brainstem to the ocular muscles attempt to correct the patient's balance.

Vestibular System:

- Vestibular system is the system which control our posture, control our vision by fix objects on retina.
- Head acceleration and gravity (stimulus) → converting into biological signals (from the inner ear to the brain) → brain develops subjective awareness of head position (orientation) → produce motor reflexes that will maintain both posture (contracts and relax certain muscles) and ocular stability (e.g; If someone asks you to concentrate on something and shakes your head, you will keep your eyes focused on the object of interest despite the shaking). We depends on the ear (vestibular system) to fix object on the retina.
- Is the apparatus of the inner ear that provides stable vision during head movements.
- **Semicircular canals are for Angular Acceleration.** Control posture, balance.
- **Utricle & Saccule:**
 - Macule of the utricle: plan **horizontal**.
 - Macule of the saccule: plan **vertical**.
 - Linear acceleration **horizontal & Vertical** (gravity).

Types of Spatial Movement: 438



- Basic mechanism of detection of rotation:
 - INERTIA.
 - Detects head acceleration, but encodes head velocity (i.e. integrator).

Vertigo

What is Dizziness?

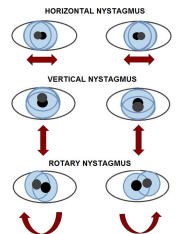
- An illusion of movement of self or environment.
- Exact description is important, just dizziness is too vague:
 - True spinning?
 - Lightheadedness?
 - Unsteadiness?
 - Fainting, passing out?

Most ear problems cause vertigo (true spinning), while LOC is not an ENT problem, refer those patients to neurology.

Dizziness is a broad term, every patient with vertigo has dizziness, you can have dizziness without vertigo. So, **you need to know what do they mean by “dizziness”, is it true spinning (vertigo) or not.**

Definition:

- **Vertigo:** It is an illusion of rotary moving. **The room is moving.**
- **Instability:** Impossibility to maintain one’s body in desired position. could be caused by low BP and low blood sugar. **‘I need to touch the wall’.**
- **Nystagmus:** Is an involuntary conjugated rapid repetitive eye movement:
 - Side to side (horizontal).
 - Up and down (vertical).
 - In a circle (torsional).



How to approach a patient with vertigo ? 438

Management in 5 minutes:

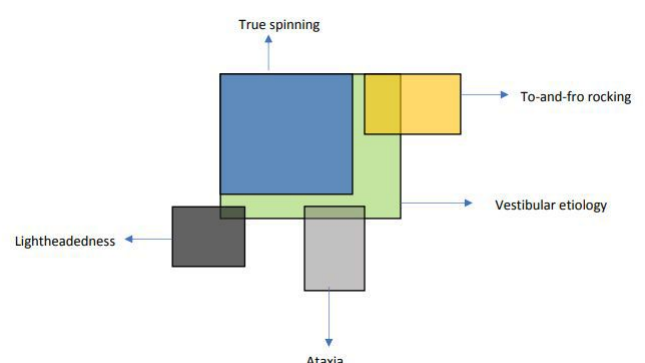
- Vestibular or Non vestibular?
- Central or Peripheral? (Stroke or Otitis media? Your approach and Rx would be different).
- Duration of vertigo and auditory system hearing loss (Was it for sec, min, or days? Was it hearing loss or tinnitus or any other symptom?)
- Proper History (90%) and Physical exam (10%).
- Treatment.

Clue #1: Significance of true spinning:

- **Almost all true spinning is vestibular.**
- All vestibular is not true spinning. (vestibular involvement causes vertigo mainly, but it can cause dizziness rarely).

Vestibular vertigo features:

- Spinning sensation.
- Nausea and vomiting.
- Worse with head movement.
- Ataxia.
- Nystagmus.



Vertigo

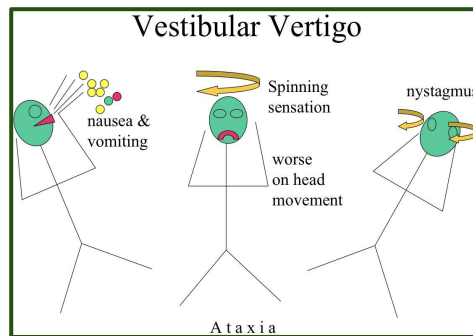
● How to approach a patient with vertigo? ⁴³⁸

Clue #2: Central vs Peripheral causes:

Not in our slides but was mentioned by 439F2 dr, he said it is **important**.

Central

- First think about the life threatening yet treatable causes like CVA before thinking about ENT causes.
- **Neurologic symptoms:**
 - New severe headache.
 - LOC (loss of consciousness).
 - Numbness, weakness.
- **Type of nystagmus**, nystagmus of central causes are different from peripheral causes.
- **Risk factors** (HTN, DM, if they're on Anticoagulants like warfarin).
- No improvement within 48 hours → increases the risk of central causes like ischemia or stroke of the brain.



Peripheral

- **Examples:**
 - Meniere's
 - BPPV (benign paroxysmal positional vertigo).
 - Vestibular neuritis.
- **Ear symptoms.**

Clue #3: Duration of dizziness:

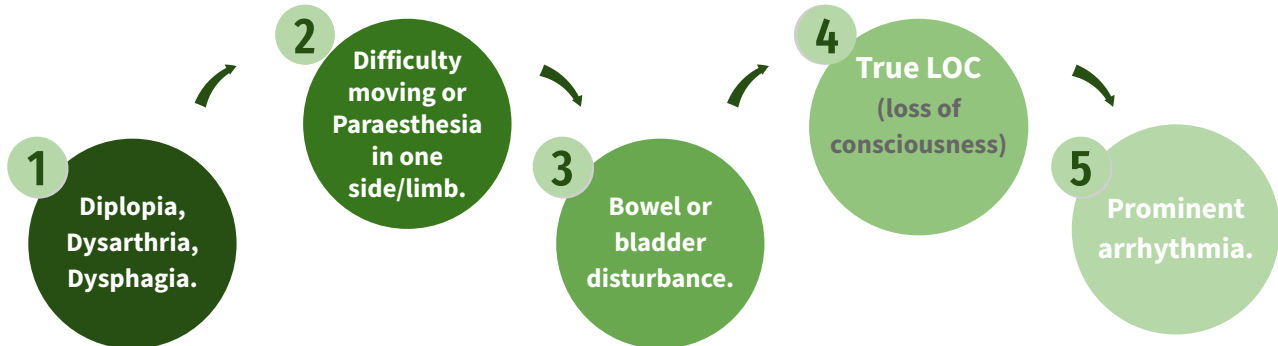
- Otologic: Prime causes.
- **Seconds to minutes:** BPPV (position related, repeated episodes, without hearing loss).
- **Minutes to hours:** Meniere's, Recurrent vestibulopathy, Migraine associated vertigo.
- **Days:** Vestibular neuritis, sudden sensorineural hearing loss with vertigo (labyrinthitis).
- **Constant, no improvement:** never vestibular.

| Vertigo duration | With Hearing Loss | Without Hearing Loss |
|------------------|------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------|
| Seconds-Minutes | ----- | Benign Paroxysmal Positional Vertigo (BPPV) usually less than a minute. |
| Minutes-Hours | Meniere's Disease. | RV (recurrent vestibulopathy), MAV (migraine associated vertigo) also called vestibular migraine (associated with motion sickness). |
| Hours-Days | Labyrinthitis (SSHL "sudden sensorineural hearing loss" with vertigo). | Vestibular Neuritis Lasts for days. |

Vertigo

● How to approach a patient with vertigo ? ⁴³⁸

Worrisome Features (non vestibular): these features could indicate central causes.



Common peripheral clinical diseases (vestibular apparatus + VIII): Know the first 3.

- **Vestibular neuritis**, Inflammation of vestibular nerve.
- **BPPV** 'Benign Paroxysmal Positional Vertigo'.
- **Meniere's disease**.
- Labyrinthine fistula. Vertigo induced by valsalva.
- Superior semicircular canal dehiscence.
- Autoimmune inner ear disease.
- Vestibulopathy.
- Vestibular nerve tumor (vestibular schwannoma).

Can it be more than one type?

- Example ; Vestibular Neuritis followed by BPPV.
- 1st episode vs most recent episode.
- How often, how long , how changing.



History:

- **History is the most important key to diagnosis for a patient with dizziness.**
- The diagnosis of the cause of vertigo or imbalance depends mostly on history, much on examination, and little on investigation.
- Patients will use various terms to describe their imbalance including 'dizziness', 'vertigo', 'funny turns' and 'giddiness'.
- Pay particular attention to timing, i.e. are the symptoms:
 - Constant or episodic.
 - Short lived as in the few minutes of dizziness associated with benign positional vertigo
 - Last for a few hours as in Menière's disease.
 - Are there associated **ear symptoms**:
 - Deafness, tinnitus, earache or discharge, and are there neurological features (loss of consciousness, weakness, numbness, dysarthria and diplopia, or seizures).
 - Note the past medical history and make a record of the patient's medications (ototoxic drug intake: gentamicin and other aminoglycoside antibiotics).

Vertigo



History (cont.):

- What are the questions to ask in history?
 - Frequency: Recurrent, Non -Recurrent.
 - Duration: Seconds, Minutes, Hours to days.
 - Associated auditory symptoms: Tinnitus, Deafness, Fullness.
 - Aggravating and relieving factors: Rolling over in bed, getting up from bed, looking up, Consume salty food.
 - Ear disease or ear surgery.
 - Trauma.
 - Migraine.

extra

| |
|-----------------------------------------------------|
| Episodic with ear symptoms |
| • Migraine |
| • Meniere's disease |
| Episodic without ear symptoms |
| • Migraine |
| • Benign paroxysmal positional vertigo |
| • Transient ischaemic attacks |
| • Epilepsy |
| • Cardiac dysrhythmia |
| • Postural hypotension |
| • Cervical spondylosis |
| Constant with ear symptoms |
| • Chronic otitis media with labyrinthine fistula |
| • Ototoxicity |
| • Acoustic neuroma |
| Constant without aural symptoms |
| • Multiple sclerosis |
| • Intracranial tumour |
| • Cardiovascular disease |
| • Degenerative disorder of the vestibular labyrinth |
| • Hyperventilation |
| • Alcoholism |
| Solitary acute attack with ear symptoms |
| • Viral infection, e.g. mumps, herpes zoster |
| • Vascular occlusion |
| • Labyrinthine fistula |
| • Round-window membrane rupture/head injury |
| Solitary acute attack without aural symptoms |
| • Acute labyrinthitis |
| • Vasovagal faint |
| • Vestibular neuronitis |
| • Trauma |



Examination:

- Complete ENT examination.
- General condition of patient.
- Vital signs, Eye movement (nystagmus).
- Ear examination and Neurological examination including all cranial nerves.
- Hearing test: Tone Audiogram, Speech Audiogram, examining the CN 8.
- Balance test: Romberg, finger to nose test, unterberger test.
- Vestibular examination: Caloric (ENG), swivel chair.



Investigation:

- CT: Skull Fracture, tumor?
- **MRI: Of brain, Tumor?**
- Duplex sonography cervicals.
- VNG.
- Audiogram.
- Head impulse test.
- v-HIT.

extra

| Symptoms | Peripheral | Central |
|---------------------|-----------------|---------------|
| Imbalance | Moderate-severe | Mild-moderate |
| Nausea and vomiting | Severe | Variable |
| Auditory symptoms | Common | Rare |
| Neurologic symptoms | Rare | Common |
| Compensation | Rapid | Slow |



AMBOSS

Differential diagnosis of vertigo

Peripheral Vestibular loss:

Inner ear disease

Examples (will be discussed in details)

- 1-Vestibular neuritis
- 2-BPPV (benign paroxysmal positional vertigo)
- 3-Meniere's disease
Involve vestibular end organs and their 1st order neurons (i.e. the vestibular nerve). The cause lies in the internal ear or the VIIIth nerve. They are responsible for 85% of all cases of vertigo.
- 4- Labyrinthitis

Central vertigo:

Central cause (supranuclear) Above the nucleus that's why they have normal VOR

- 1-Stroke
- 2-Posterior fossa tumor: Vestibular Schwannoma
- 3-migraine
- 4-Multiple sclerosis

Clinical features:

| | Peripheral | Central |
|-----------------------------------------------|------------------|---------------------------------------------------------------------------------------------------------|
| Cranial nerve features Cerebellar features | Typically Absent | Marked Presence of dysmetria, dysphagia, dysarthria , or diplopia Severe ataxia |
| Hearing loss and/or tinnitus | Common | Rare |
| Focal Neurological Deficit | Absent | Present |
| Nausea/Vomiting | Frequent | varies |

Any of the **Dangerous D's** (Dysphagia, Dysarthria, Diplopia, Dysmetria) strongly suggest a central cause of vertigo.

Examination: Head impulse, nystagmus, test of skew (HINTS)

An examination to screen for a **central** cause in acute vestibular syndrome **without** an identifiable trigger (eg: stroke)

| | Peripheral | Central |
|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Head impulse test Evaluates the vestibuloocular reflex (VOR) Ask the patient to fixate on a stationary object in front of them. Rapidly rotate the patient's head 10 degrees from center and assess their ability to maintain a central gaze. | Abnormal head impulse test (impaired VOR) | Normal head impulse test (Normal VOR) → Supranuclear |
| Nystagmus Watch for spontaneous nystagmus at rest and Watch for gaze-evoked nystagmus while examining the extraocular muscles. | <ul style="list-style-type: none"> • Spontaneous horizontal nystagmus (typical) (BPPV has vertical) • The direction of nystagmus does not change with gaze change (<u>unidirectional nystagmus</u>). • The fast phase beats away from the side of the lesion. • Gaze fixation suppresses nystagmus. | <ul style="list-style-type: none"> • May be torsional, horizontal, or vertical • The direction of nystagmus changes with gaze change (<u>gaze-evoked nystagmus</u>). • Gaze fixation does not reduce nystagmus. |
| Test of skew Ask the patient to maintain a fixed central gaze and to keep both eyes open during the examination. Repeatedly cover and uncover alternating eyes, while watching for vertical deviation from the central gaze upon uncovering the eye. | Skew deviation is absent The eye remains in a fixed central gaze when uncovered. | Skew deviation is present A refixation saccade occurs upon uncovering the eye |
| <ul style="list-style-type: none"> • Presence of <u>ANY of the following</u> strongly suggests central vertigo: normal head impulse test, any central-type nystagmus (e.g., direction-changing, vertical, or torsional nystagmus), AND/OR <u>skew deviation</u> • Presence of <u>ALL of the following</u> suggests peripheral vertigo: abnormal head impulse test, only peripheral-type nystagmus (e.g., spontaneous unidirectional horizontal nystagmus), AND no <u>skew deviation</u> | | |

During HINTS testing, think **INFARCT** to identify central causes (e.g., stroke): Impulse **Normal**, Fast-phase **Alternating**, Refixation on Cover Test

Peripheral vestibular loss

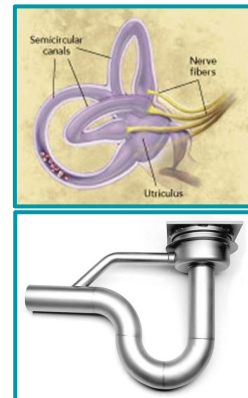


1 BPPV (benign paroxysmal positional vertigo):

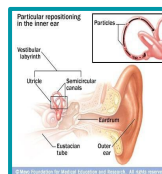
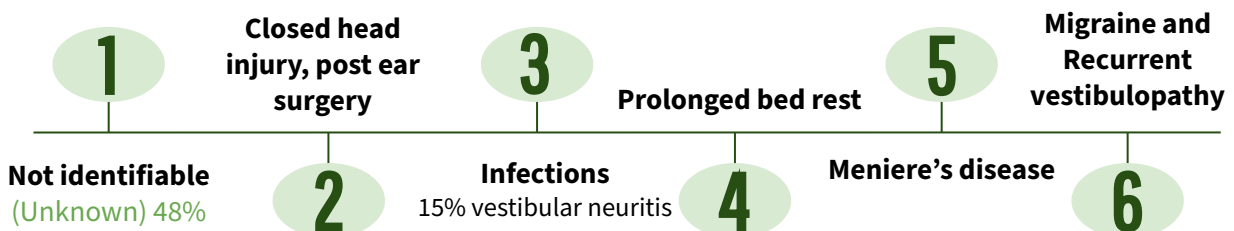
- Most common cause of peripheral vertigo in patients over 40.
- BPPV is the most common cause of vertigo seen by otolaryngologists.
- Represent 20% to 40% of patients with peripheral vestibular disease.

Pathophysiology:

- Canalithiasis theory: degenerative debris from utricle (**otoconia**) → floating freely in the endolymph. migration of free floating otoliths within the endolymph of the semicircular canal.
- Cupulolithiasis theory: Debris adhering to the cupula. Otolith attached to the cupula of the semicircular canal, can affect each of the 3 semicircular canals, **although the posterior canal is affected in >90%**.
- Ear stones “otoliths”, **posterior semicircular canal is the most common canal affected (post SCC). In the examination we test the canal by moving the head down and see if there’s nystagmus.**
- Posterior canal: hangs down like the water trap in drain pipe, allowing the crystals to settle in the bottom of the canal.



Etiology:



Approach:



History (virtually pathognomonic):

Almost pathognomonic once you hear this classical history there is no other differential.

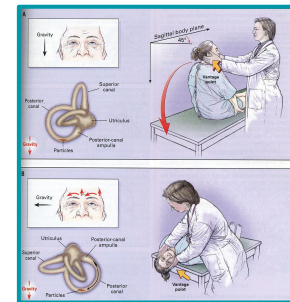
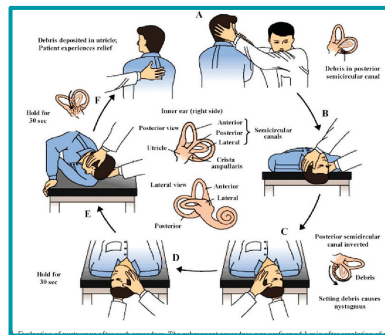
- Severe vertigo.
- **Associated with change in head position. When I lay down I get dizzy when I stand up it goes away**
 - Rolling over or getting into bed.
 - Assuming a supine position.
 - Arising from a bending position.
 - Looking up to take an object off a shelf.
 - Tilting the head back to shave.
- Chronic balance problems. **Not associated with any auditory or neurological symptoms**
 - **Provoked by certain positions (rolling in bed, looking up for shaving and head rotation).**
 - **Suddenly and last in the order of seconds.**
- Bouts of vertigo → remissions.
- Worse on awakening in the morning.
- Only type of vertigo:
 - **Multiple times a day repeated attacks episodic.**
 - **Brief episodes (seconds) should count them, less than 1 minute.**
 - **Unaccompanied by auditory complaints, no tinnitus, hearing loss, discharge, or earache and no neurological symptoms.**

Peripheral vestibular loss



Examination (to confirm the diagnosis):

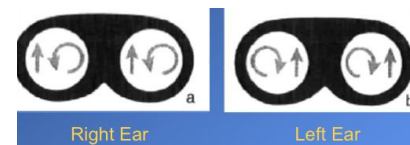
- **Dix hallpike maneuver.**
- Dix hallpike maneuver (Hager 6Ds):
 - 1) Delay (seconds latency).
 - 2) Downward (geotropic).
 - 3) Duration (less than 1 min).
 - 4) Directional changes.
 - 5) Dizziness (Subjective).
 - 6) Disappear (fatigable).



We think it could be due to otolith (oto → ear, lith → stone). Some precipitations in inner ear move from its place and disrupt fluid inside inner ear.

- Five Signs of BPPV Seen with Dix Hallpike Maneuver: “You’re removing the particles back to where they came from”.
 - Geotropic rotatory nystagmus (nystagmus MUST be present for a positive test).
 - Fatigues with repeated maneuver and fixation.
 - Reversal of nystagmus upon sitting up.
 - Latency of ~20 s.
 - Crescendo/decrescendo vertigo lasting 20 s.
- We hold patient head and pull it down then nystagmus will be seen but repeated testing results in abolition (canceling) of the vertigo.
- Nystagmus in BPPV:
 - The nystagmus is a combined vertical upbeating and rotary (torsional) component beating toward the downward eye (affected side).
 - There is often a latency of onset of nystagmus (seconds).
 - Duration of nystagmus is short (<1 minute).
 - The nystagmus disappears with repeated testing (**fatigable**)

Decrease everytime we repeat the test.



Differential diagnosis:

- Postural hypotension (anti-hypertensive drugs / CV problems).
- Fistula.

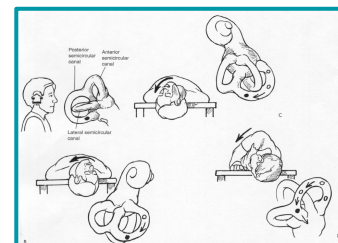


Treatment:

- Aim of the treatment is to move the debris (otoconia) out of the affected semicircular canal back into the vestibule.
- Anti-emetics for nausea/vomiting.
- Particle repositioning maneuvers: **Epley maneuver.**

Epley maneuver:

Placement of the head into the Dix-Hallpike position, there is a 180-degree roll of the head to the position in which the offending ear is up, patient is then brought to the sitting upright position.



Basically, pt will be in laying down position with head rotated to the affected side, he/she will feel vertigo and the physician will be able to notice nystagmus which is the typical diagnostic feature (**Dix hallpike maneuver**).

After vertigo and nystagmus subsided, turn the head to the other side 90° and wait for 30 sec then rotate the whole body 90° for 30 sec and the particle will move out when the pt set up (Epley maneuver).

Peripheral vestibular loss



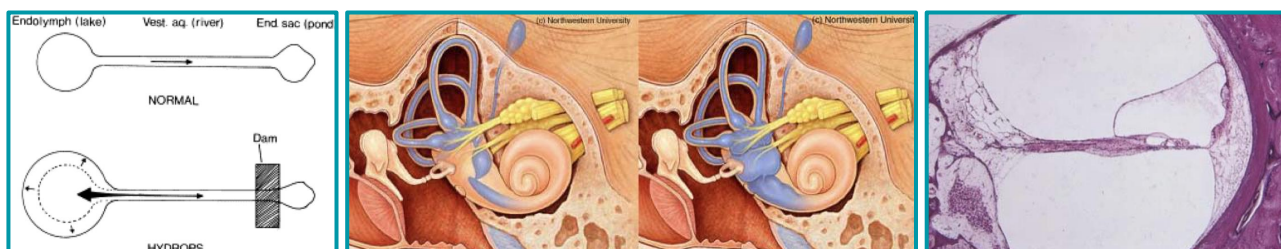
2 • **Meniere's disease:** 20 min to 4 h of vertigo associated with **Tinnitus, Ear(Aural) fullness, Hearing loss.**

Definition

- **Idiopathic endolymphatic hydrops.**
- Recurrent attacks of vertigo **lasting hours. Associated tinnitus, hearing loss, pressure.**
- Can occur at any age but its onset is common between 40-60 years. مرض المشاهير

Pathology:

- Normally, endolymph is secreted by stria vascularis, fills the membranous labyrinth and is absorbed through the endolymphatic sac.
- Decrease endolymphatic reabsorption or **increase production of fluid within inner ear or decrease absorption.**
- Progressive hydrops.
- Membranous ruptures.
- Spillage of large amount of neurotoxic endolymph into the perilymphatic compartment.
- Healing of the membranes.
- Distortion and atrophy of sensory and neural structures.
- **Autopsies revealed scala media is hyperinflated > ruptures.**
- In 10-20% of cases the disease later involves the opposite ear.



Causes:

- It's due to a disease of the inner ear that causes an increase in pressure in the inner ear canal. Overproduction or retention of endolymph (Unknown, autoimmune, ischemia, mumps, syphilis, hypothyroidism, head trauma, previous infection, hormonal (pregnant females are more prone).

Triggers:

- High salt intake, caffeine, stress, nicotine and alcohol. Males affected more than females.

Diseases course:

- Early: predominant vertigo, deafness (partial), normal hearing between (the attacks).
- Later: hearing loss stops fluctuating, progressively worse (50db). **Inner ear lost function of hearing and balance.**

Peripheral vestibular loss



2

Meniere's disease (Cont):



Symptoms:

1. **Vertigo:** usually spinning sensation lasts 20 minutes - 5 hours accompanied by nausea and vomiting with ataxia and nystagmus towards the unaffected ear. **Some people might cry, laugh, experience fear during the attack.**
2. **Fluctuating SNHL 'deafness':** improves after the attack. **Low frequency** (raising Audiogram) fluctuating SNHL - Although deafness is fluctuant repeated attacks can cause significant sensorineural hearing loss.
3. **Tinnitus:** usually low-tone roaring.
4. **Aural fullness:** it also happens before the onset of the attack. Due to increased hydraulic pressure within the inner ear endolymphatic system.



Diagnosis:

1. **History:** *pathognomonic symptoms*
 - **Recurrent attack of vertigo for minutes to hours.**
 - **Associated with tinnitus, fluctuating hearing loss.** (vs BPPV)
 - **Aural (ear) fullness** (unlike vestibular neuritis).
 - Sudden severe attacks may be accompanied by other symptoms of vagal disturbances such as abdominal cramps, diarrhea, cold sweats, pallor and bradycardia.
2. **Physical exam:**
 - **Will not really help you:** unilateral hearing weakness.
3. **PTA (pure tone audiometry):**
 - **LF-SNHL (low frequency sensorineural hearing loss).**
4. You must rule out other possible diagnosis **by CT or MRI to rule out acoustic neuroma for example.**



Management:

- **Education:** **We treat it like hypertension.**
 - Decrease intake of **CATS** (**C**hocolate, **A**lcohol, **I**ea, **S**alt), cessation of smoking, avoid stress.
- Treat the acute attacks (stay away from dangerous places, prevent falls).
- Prevent further attacks.
- Improve hearing.
- Vestibular rehabilitation.
- Follow up bilateral Meniere's disease.
- **Medical** (diuretics, trans-tympanic injection, anticholinergic, antihistamine, phenothiazine, benzodiazepines).
- **We may put ototoxic drugs like gentamicin to destroy and poison the damaged ear.** (First line)
- **Surgical** (we destroy the inner ear ; drill cochlea and nerve). (second line)

Acute attack: prevent falls, head should be restricted, anticholinergics, antihistamines, phenothiazine, benzodiazepines.

Peripheral vestibular loss



3

Vestibular neuritis:

(**labyrinthitis**: a similar syndrome, but with hearing symptoms)
12h to 24h of vertigo, vomiting.

- Disorder that affects the vestibulocochlear nerve of the inner ear.
- 50% may have upper respiratory tract infections.
- 50% infectious illness precede VN (viral infection of vestibular organ) URTI.
- Affect all ages but rare in children.
- Abrupt onset.
- Single, severe, prolonged, spontaneous vertigo, nausea, vomiting and Nystagmus.
- No hearing loss, or severe vertigo: imbalance without hearing loss that resolves over days leaving the residual imbalance that last days to weeks. **Patient is fully awake.** Stroke patients will not be fully awake they are very ill.
- No neurological signs/symptoms.
- Treatment: symptomatic (3 weeks to recover), spontaneous recovery occurs over weeks to months (depend on the age of the patient).
- Neurological origin (stroke), should be eliminated.
- No neurological or auditory symptoms.

Clinical Features:

- Acute phase: severe vertigo (vertigo could turn into instability) with nausea, vomiting, and imbalance lasting 1 to 5 d, Irritative nystagmus (fast phase towards the offending ear) patient tends to veer towards affected side.
- Convalescent phase: imbalance and motion sickness lasting days to weeks, Spontaneous nystagmus away from affected side, gradual vestibular adaptation requires weeks to months.

Investigation



- Audiogram
- Videonystagmography
- v-HIT
- CT Scan
- MRI

Treatment wait it out



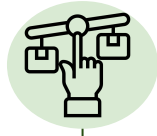
- Usually self-limiting.
- The patient requires only symptomatic treatment.
- Acute phase: bed rest, vestibular sedatives (Gravol®), and diazepam.
- Convalescent phase: progressive ambulation especially in the elderly, vestibular exercise: (involve eye and head movements, sitting, standing, and walking).

Vertigo

● Vertigo consequences:



Possibility of deafness (M. Menière)



Drive prohibition (safety issue - occupations requiring driver's license)



Increased danger of falling (fractures, especially older patients)

Mental stress (possibly psychotherapy)



Impairment of the quality of life



Loss of occupations



Ototoxicity:

- Usually aminoglycosides or chemotherapy **both ears; bilaterally.**
- Complain of oscillopsia.



Investigation:

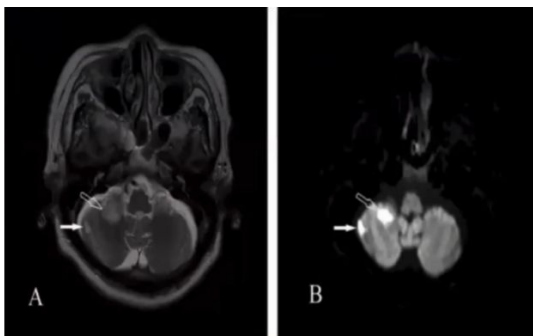
For any patient with dizziness. **99% of the diagnosis depends on the history and examination.** But we do investigations to have a baseline.

- Audiology tests **are routinely done for any dizzy patients, and will be discussed in audiology.**
 - PTA (pure tone audiometry).
 - ENG (Electronystagmography).
 - Posturography.
 - Rotation chair.
- Radiology
 - **CT/MRI** (if we suspect a brain tumor).
- Blood tests (for other diseases)
 - CBC, thyroid, FT-ABS.

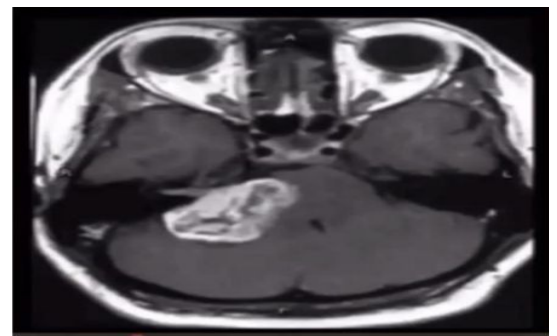
An area for your notes

Part of obj

- Vascular insufficiency e.g; stroke.
- Mass lesion e.g; CPA tumor, acoustic neuroma.
- Multiple sclerosis.
- Epilepsy.
- Arnold-chiari malformation.
- Migraine induced vertigo:
 - Very common in female.
 - Commonly occur below age of 30.
 - Hx of true attacks of migraine with/without vertigo and vice versa or pt present with vertigo only with previous Hx migraine.
 - Diagnosis based on Hx.



Both pictures of MRI indicate stroke.
A: without contrast.
B: with contrast.



- Acoustic neuroma at the right side.
- **Acoustic neuroma:**
 - Pt present similarly like Ménière's disease.
 - Clinical presentation of acoustic neuroma :
 - Tinnitus.
 - Continuous and progressive hearing loss unlike Ménière's disease which is **fluctuating**.
 - Persistent vertigo common on daily bases, while Ménière's disease comes and goes.



Part of obj

1- Acoustic neuroma:

- Benign tumor, arise from vestibular division of VIII.
- Pathogenesis:
 - Starts in the internal auditory canal and expands into cerebellopontine angle (CPA), compressing cerebellum and brainstem
 - When associated with type 2 neurofibromatosis (NF2): bilateral acoustic neuromas, café-au-lait skin lesions, and multiple intracranial lesions
- Clinical presentation:
 - Unilateral tinnitus
 - Hearing loss
 - Dizziness But true vertigo is rare as tumor growth is slow thus compensation occurs.
 - Facial nerve palsy and trigeminal (V1) sensory deficit (corneal reflex) are late complication.
- DDX:
 - Acoustic neuroma mimics Meniere's disease in presentation and **imaging** is the only way to differentiate between them.
- Diagnosis:
 - History
 - PTA (Unilateral SNHL)
 - Radiology (CT, MRI)
 - MRI with Gadolinium contrast is the gold standard.
- Treatment:
 - Expectant management if tumor is very small, or in elderly. Definitive management is surgical excision.

2- CVA:

Elderly patient with chronic disease like (DM, HTN) with sudden attack of vertigo + neurological symptoms. Vertigo is abrupt in onset, lasts several minutes and is associated with nausea and vomiting. Other neurological symptoms like visual disturbances, drop attacks, diplopia, hemianopia, dysphagia and hemiparesis resulting from ischaemia to other areas of brain may also accompany vertigo.

An area for your notes

● A dizzy patient may fit into one of the following scenario:

1- The patient who is having a first ever attack of acute spontaneous vertigo.

- DDX:
 - Acute vestibular neuritis.
 - Cerebellar infarction (stroke).
- How to differentiate?
 - Clinically **if still not sure go for imaging.**
 - Radiology.

2- The patient who has repeated attacks of vertigo, but is seen meanwhile well (**means between attacks he is fine**).

- DDX:
 - Benign paroxysmal positional vertigo *
 - Ménière's disease *
 - *To differentiate between these two above, look for 3 things: (1) vertigo duration, (2) positionally related or not (3) hearing loss.
 - Migraine induced vertigo **common attack that comes and goes and pt will tell you a specific details about his/her migraine attacks.**
 - perilymph fistula **is a communication between middle ear and inner ear by fistula induced surgically or pathologically by a disease, those patients have vertigo attacks that comes and goes which are mostly provoked by straining and lifting heavy objects.**

3- The patient who is off balance **day and night I'm feeling imbalanced, so it's not recurrent and it's not one attack.**

- DDX:
 - Bilateral vestibulopathy **due to autoimmune diseases or ototoxic medications.**
 - Normal pressure hydrocephalus.
 - Posterior fossa tumor.

● Conclusion:

- Proper history is the most important key for diagnosis of a dizzy patient.
- If you can't reach a diagnosis from the first visit don't feel disappointed, try to bring patient back for another assessment.
- A multi specialty (Cardiac, Ophtha, Psych) approach is sometimes appropriate for some complicated cases.
- Investigations should be tailored to the most likely diagnosis. Don't do unnecessary tests when they're not needed.

● Case Scenarios 436:

Case 1

- 50-year-old patient, medically free.
- Sudden dizziness with head movement 3 days ago.
- Horizontal Nystagmus to the left.
- No problems without Head movement.
- Accompanying symptoms (nausea, vomitus).
- No hearing problems, no tinnitus.
- **Answer: benign paroxysmal positional vertigo.**

● Case Scenarios 436:

Case 2

- 32-year-old female patient with recurrent episodes of vertigo.
- Relapsing accompanying hearing loss, tinnitus.
- Frequently nausea / vomiting.
- Fall inclination both sides.
- Persistent hearing loss in the interval (weeks).
- Recurrent inflammation of both eyes.
- Answer: **cogan syndrome**.

Questions 436:

1. Young female complaining of acute persistent vertical vertigo no hearing loss no tinnitus no fullness, but she reported a history of respiratory infection last week. what is the diagnosis?
 - A. BPPV
 - B. Vestibular neuritis
 - C. Meniere's disease
2. A 60-year-old man, complaining of severe tinnitus, episode of vertigo, and hearing loss in his right ear. PTA showed SNHL in the right ear, while the left was normal. What are the suspected finding in tuning fork test in this patient?
 - A. Weber test is lateralized to the right, Rinne test is negative
 - B. Weber test is lateralized to the left, Rinne test is positive.
 - C. Weber test is central, Rinne test is negative.
 - D. Weber test is central, Rinne test is positive.
3. A patient presented with history of hearing loss and vertigo. Pure tone audiometry showed SN hearing loss. ABR(auditory brainstem response) showed abnormal waves. What is the diagnosis?
 - A. Vestibulitis
 - B. Acoustic neuroma
 - C. Meniere disease
 - D. BPPV (Benign Paroxysmal Positional Vertigo)
4. 28-year-old female presented with vertigo which last for minutes with hearing loss and tinnitus. What is most likely the diagnosis?
 - A. Benign paroxysmal positional vertigo
 - B. Vestibular neuritis
 - C. Meniere's disease
 - D. Acoustic neuroma
5. 26 years old female comes with 6 days history of severe vertigo associated with right sided hearing loss. She had a history of chronic suppurative otitis media for many years. On examination there is marginal moist perforation on the right ear drum. There is horizontal nystagmus. What is the most likely cause of vertigo?
 - A. Acute labyrinthitis
 - B. Benign paroxysmal positional vertigo.
 - C. Meniere's disease
 - D. Vestibular neuritis

THANK YOU!

This work was done by:

Sultan Alosaimi

Hamad Alkhenizan

Abdullah AlGhamdi

Reviewed by

Naif Alfahed - Abdullah AlQarni

Team Leaders:

Naif AlFahed

Nawaf AlGhamdi

Abdullah AlQarni

Special Thanks to

439 Team

