The Inner Ear
Inner ear anatomy
Anatomy

• Inner ear consists of:
  • Bony Labyrinth: filled with a fluid called **perilymph**, consists of 3 parts:
    1. The Vestibule
    2. The Semicircular Canals: 3 in number (Lateral, Posterior, Superior)
    3. The Cochlea: The bony cochlea is a coiled tube making 2.5 to 2.75 turns round a central pyramid of bone called modiolus.

• Membranous Labyrinth: filled with fluid called **endolymph**, consists of:
  1. The Cochlear Duct: contains the organ of Corti.
  2. The Utricle and the Saccule: the sensory epithelium is called the macula. Concerned with Linear Acceleration and deceleration.
  3. The Semi Circular Ducts: 3 in number, correspond to the bony semi circular canal, the sensory epithelium is called the crista ampularis. Concerned with angular acceleration and deceleration.
INNER EAR
- BONY & MEMBRANOUS LABYRINTHS

- Cochlear duct (hearing -2 3/4 turns)
- Oval window
- Round window
- Utricle & Saccule (both for static balance)
- Full size at birth
- In petrous temporal bone
- One continuous cavity
- For hearing & balance
- Vestibulocochlear nerve
- Blood: labyrinthine artery

Membranous labyrinth lies within osseous labyrinth

Semicircular canals (kinetic balance)

Endolymphatic sac & duct

Perilymphatic duct

External meatus

Auditory tube

Round window

Cochlear duct (hearing -2 3/4 turns)

Blood supply via labyrinthine artery

Endolymph

Perilymph

U = Utricle
S = Saccule (both for static balance)
Cochlea

The **cochlea is coiled around** the modiolus. The Modiolus: **central** axis of the cochlea. Contains:
- **cochlear nerve**
- Blood vessels

The cochlear coil extends “up” from its base to (2 3/4 X)

**High** frequency at the **base**, **low** frequency at the **tip**. The cochlea is divided by **Reissner’s membrane** and the **basilar membrane** into 3 scalae(chambers):
- Scala vestibule
- Scala media (cochlear duct)
- Scala tympani
INNER EAR - COCHLEA STRAIGHTENED OUT TO AID UNDERSTANDING

- Stapes in oval window
- Vestibular membrane
- Apex of cochlea
- Scala vestibuli
- Perilymph
- Cochlear duct
- Endolymph
- Scala tympani
- Hair cells lying on basilar membrane
- Tectorial membrane lying on cilia of hair cells
- Ossicles amplify vibrations 15-20 times
- Round window closed with thin membrane

HEARING MECHANISM

Sound waves → Pinna → External meatus →
Tympanic membrane → Ossicles → Stapes →
Vibrations in perilymph → Basilar membrane →
Hair cells (convert acoustic energy to action potentials) →
Tectorial membrane →
Cochlear part of vestibulocochlear nerve (VIII) → Auditory cortex
Sensory Neural Hearing Loss (SNHL) and tinnitus
Sensory Neural Hearing Loss (SNHL)

• Results from lesions of the cochlea, VIIIth nerve or central auditory pathways. It may be present at birth (congenital) or start later in life (acquired).

• The characteristics of sensorineural hearing loss are:
  1. A positive Rinne test, i.e. AC > BC.
  2. Weber lateralized to better ear.
  3. Bone conduction is reduced.
  4. More often involving high frequencies.
  5. No gap between air and bone conduction curve on audiometry
  6. Loss may exceed 60 dB.
  7. Speech discrimination is poor.
  8. There is difficulty in hearing in the presence of noise.
Hearing Loss Symptoms

Hearing loss analysis by history:

1. **Onset:** sudden/gradual
2. **Severity:** impairing daily activity/mild/moderate/severe
3. **Course:** Rapidly progressive/non progressive
4. **associated problems,** such as tinnitus or vertigo
5. **unilateral or bilateral.**
6. **Predisposing factors:** e.g. noise exposure
7. **Family history**
8. **Drug History**
9. **Prenatal, postnatal history (in children)**
Clinical examination

The examination may include the following:

1. Examination of the ear
2. Clinical assessment of the degree of deafness: *Whispered speech test*.
3. Tuning fork tests: Weber’s, Rinne’s
4. Pure Tone Audiometry
5. Otoacoustic emissions: assesses the cochlear function by recording signals produced by the hair cells
6. Auditory brainstem response: assesses the integrity of the cochlea, auditory nerve and brain
SNHL

• Aetiology:
  • Congenital
  • Acquired:
    1. Infections of labyrinth—viral, bacterial or spirochaetal
    2. Trauma to labyrinth or VIIIth nerve, e.g. fractures of temporal bone or concussion of the labyrinth or the ear surgery
    3. Noise-induced hearing loss
    4. Ototoxic drugs
    5. Presbycusis
    6. Meniere’s disease
    7. Acoustic neuroma
    8. Sudden hearing loss
    9. Familial progressive SNHL
   10. Systemic disorders, e.g. diabetes, hypothyroidism, kidney disease, autoimmune disorders, multiple sclerosis, blood dyscrasias.
Presbyacusis

• progressive, bilaterally symmetrical sensorineural hearing loss occurring with age usually.
• The age of onset is variable, but typically some hearing difficulty may be experienced from the mid 60s.
• Genetic factors can cause a tendency to a more advanced and earlier onset of presbyacusis.
• The most common pattern of hearing loss is a symmetrical one in which high frequencies are lost.
• The diagnosis is usually confirmed from the history, examination and a pure tone audiogram.
• Management comprises offering hearing aids bilaterally.
Idiopathic Sudden sensorineural Hearing loss

- Definition: $\geq 30$ dB SNHL in at least 3 adjacent frequencies that occurs over $\leq 3$ days
- Usually unilateral
- Should be evaluated within 72 hours to 1 week of onset maximally (the sooner the better)
Idiopathic Sudden sensorineural Hearing loss (ISSNHL)

• Aetiology:
  • Most often the cause of sudden deafness remains obscure, in which case it is called the idiopathic variety. In such cases, three aetiological factors are considered—viral, vascular or the rupture of cochlear membranes.

• Other aetiological factors which cause sudden deafness and must be excluded are listed below.
  1. Infections. Mumps, herpes zoster, meningitis, encephalitis, syphilis, otitis media.
  2. Trauma. Head injury, ear operations, noise trauma, barotrauma, spontaneous rupture of cochlear membranes.
  3. Vascular. Haemorrhage (leukaemia), embolism or thrombosis of labyrinthine or cochlear artery or their vasospasm. They may be associated with diabetes, hypertension, polycythaemia, macroglobinaemia or sickle cell trait.
  4. Ear (otologic). Meniere’s disease, Cogan’s syndrome, large vestibular aqueduct.
  5. Toxic. Ototoxic drugs, insecticides.
  7. Miscellaneous. Multiple sclerosis, hypothyroidism, sarcoidosis.
  8. Psychogenic.
Idiopathic Sudden sensorineural Hearing loss (ISSNHL)

• Incidence: 8/100,000

• Investigations:
  • CBC and ESR; urea and electrolytes;
  • lipid profile; glucose;
  • thyroid function;
  • syphilitic serology;
  • auto-antibodies;
  • ± MRI (depending on availability).
  • Investigation of choice in children is CT with contrast, 2\textsuperscript{nd} choice is MRI
ISSNHL Management

• 50% will have complete spontaneous recovery.
• high-dose oral corticosteroids: 1 mg/kg for 1 week then do PTA if there is improvement tapper the steroid if no improvements give steroid for another week then tapper
Drug induced SNHL

Temporary vs permanent hearing loss:

**permanent** (hearing loss/vestibular dysfunction):
- Anti-neoplastic: cisplatine/carboplatine
- Aminogylcoside (neomycin, streptomycin, kanamycin, Gentamycin, arbekacin)
- vancomycin
- solvents: Toluene, benzene

**temporary** (hearing loss):
- Aspirin/ NSAIDs (cause more tinnitus than HL)
- Macrolide: erythromycin, clarithromycin, azithromycin
- Quinine

**temporary/permanent** (hearing loss):
- Diuretics: Ethacrynic acid, furosemide
Noise-induced deafness

- Exposure to noise levels louder than 85–90 dB through prolonged employment or recreation in a noisy environment, will lead to death of cochlear hair cells and usually bilateral high-tone sensorineural deafness, initially maximal at 4000 Hz.

- The presentation is often with tinnitus and bilateral hearing loss being noted in middle age, even though the original noise damage occurred in the early adult years.
Hearing loss in children

Risk factors for hearing loss in children:
1. Family history of hearing loss.
2. Prenatal infections (TORCHES).
3. Craniofacial anomalies including those of pinna and ear canal.
4. Birth weight less than 1500 g (3.3 lbs).
5. Hyperbilirubinaemia requiring exchange transfusion.
6. Ototoxic medications included but not limited to aminoglycosides used in multiple courses or in combination with loop diuretics.
7. Bacterial meningitis.
8. Apgar score of 0–4 at 1 min or 0–6 at 5 min.
9. Mechanical ventilation for 5 days or longer.
10. Stigmata or other findings associated with a syndrome known to include sensorineural and/or conductive hearing loss.
Management of SNHL

• Hearing aids
• Bone anchored hearing aids
• Cochlear implant
• Lip reading
• Sign language
• Electronic aids for the deaf
Wearable Hearing aid

Used for CH or SNHL up to profound Hearing loss.

Components:
• Microphone: converts sound to electricity.
• Amplifier
• Receivers
Bone Anchord Hearing Aid (BAHA)

- Used for Bilateral CHL
- Soft BAHA (Head wearable band)
- BAHA (osteointegrated +/- external device)
Cochlear implant

Selection criteria

Children 12-24 months of age
- bilateral profound SNHL hearing loss (> 90 dB)
- minimal hearing aid benefit
- No speech development

Adults
- bilateral severe-to-profound hearing loss (> 70 dB)
- at least 1-3 months pre-operative hearing aids trial
- Not receiving adequate benefit of Hearing aid:
- no evidence of central auditory lesion

Age limit: 12 months
Prevention

• Noise-induced hearing loss is usually permanent and progresses with each exposure. Use proper ear protection when working around loud noises.
• Never put foreign objects in the ear.
  • Do not use cotton swabs to probe or clean the ear canals.
  • Do not put cotton balls or liquids into the ear unless prescribed by a doctor.
• Treat middle ear infections as soon as possible.
• If you are taking medications that can cause hearing loss, hearing should be monitored
Tinnitus
Tinnitus

- Tinnitus is the perception of sound without an external source for more than five minutes at a time, in the absence of any external acoustical or electrical stimulation of the ear and not occurring immediately after exposure to loud noise.

- Disturbing tinnitus occurs in 3% to 5% of individuals with tinnitus.

- Two types of tinnitus are described:
  1. Subjective, which can only be heard by the patient.
  2. Objective, which can even be heard by the examiner with the use of a stethoscope.

- Subjective > objective tinnitus.
<table>
<thead>
<tr>
<th>Subjective tinnitus</th>
<th>Objective tinnitus</th>
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<tbody>
<tr>
<td><em>Otologic</em></td>
<td><em>Vascular</em></td>
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<tr>
<td>Impacted wax</td>
<td>AV shunts</td>
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<tr>
<td>Fluid in middle ear</td>
<td>Congenital AV</td>
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<tr>
<td>Acute otitis media</td>
<td>malformations</td>
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<tr>
<td>Chronic otitis media</td>
<td>Glomus tumour of middle ear</td>
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<td>Ménière’s disease</td>
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<tr>
<td>Presbycusis</td>
<td>Arterial bruit</td>
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<tr>
<td>Noise-induced hearing loss</td>
<td>Carotid aneurysm</td>
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<tr>
<td>Idiopathic sudden SNHL</td>
<td>Carotid stenosis</td>
</tr>
<tr>
<td>Acoustic neuroma</td>
<td>Vascular loop pressing on VIIIth nerve</td>
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<td></td>
<td>in internal auditory canal</td>
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<tr>
<td>Metabolic</td>
<td>High-riding carotid artery</td>
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<td>Hypothyroidism</td>
<td>Persistent stapel artery</td>
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<td>Hyperthyroidism</td>
<td>Venous hum</td>
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<tr>
<td>Obesity</td>
<td>Dehiscent jugular bulb</td>
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<tr>
<td>Hyperlipidaemia</td>
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<tr>
<td>Vitamin deficiency (e.g. B12)</td>
<td>Patulous eustachian tube</td>
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<tr>
<td>Neurologic</td>
<td>Palatal myoclonus</td>
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<tr>
<td>Head injury (labyrinthine concussion)</td>
<td>Idiopathic stapedal or tensor tympani</td>
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<tr>
<td>Temporal bone fractures</td>
<td>myoclonus</td>
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<tr>
<td>Whiplash injury</td>
<td>Dental</td>
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<tr>
<td>Multiple sclerosis</td>
<td>Clicking of TM joint</td>
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<td>Postmeningitic</td>
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<tr>
<td>Brain haemorrhage</td>
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<td>Brain infarct</td>
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<td>Cardiovascular</td>
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<td>Hypertension</td>
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<td>Hypotension</td>
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<td>Anaemia</td>
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<tr>
<td>Cardiac arrhythmias</td>
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<td>Arteriosclerosis</td>
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<tr>
<td>Pharmacologic</td>
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<td>Certain drugs used by the patient</td>
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<td>All ototoxic drugs</td>
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<td>Psychogenic</td>
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<tr>
<td>Anxiety</td>
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<td>Depression</td>
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History

• a general medical evaluation, Assessment of its features. Important features include tonality; noise aspects such as hissing, buzzing, humming, sizzling, and roaring; unilateral-bilateral localization in the head; temporal features (e.g., pulsatile vs. steady); and interaction with external sounds, including masking, inhibition, and exacerbation.

• Basic information about the auditory characteristics of tinnitus includes localization (left, right, in the head, outside of the head), constancy (episodic, fluctuating, constant, pulsatile), pitch, loudness, and sound quality (tonal, hissing, buzzing, clicking, ringing).
Physical examination

- palpation and light compression of the jugular vein may diminish tinnitus of venous origin (a similar effect can be achieved by the Valsalva manoeuvre, during which an increased intrathoracic pressure and decreased venous return, may also reduce tinnitus);
- auscultation of the neck and cranium for the presence of carotid bruit or blood turbulence due to arteriovenous malformation;
- *otoscopy/otomicroscopy* may reveal glomus tumours or tympanal haemangioma;
- oropharyngeal examination could reveal contraction of the soft palate in palatal myoclonus.
Investigations

• Tympanometry may demonstrate myoclonic activity and patulous Eustachian tube.

• Pure tone audiometry may indicate conductive hearing loss secondary to vascular lesions affecting the middle ear.

• Imaging with gadolinium-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) is necessary in most cases.
Management of Tinnitus

• Tinnitus is a symptom not a disease, so the cause of the tinnitus should be sought and treated.

• When no cause could be found, the management would be:
  1. Reassurance and psychotherapy. Many times the patient has to learn to live with tinnitus.
  2. Techniques of relaxation and biofeedback.
  3. Sedation and tranquillizers. They may be needed in initial stages till patient has adjusted to the symptom.
  4. Masking of tinnitus.
Tinnitus Treatment Strategies

• Using Sound to Decrease Loudness and Annoyance of Tinnitus
• Hearing aids
• Cognitive-Behavioral Therapy for Tinnitus
• Tinnitus retraining therapy
• Cochlear Implants
Vertigo is defined as the illusion of movement of the patient or the surroundings.

- **chief complaint** of patients with injury to the vestibular system is usually dizziness not vertigo.
- Never permanent, continuous symptom. Even when the vestibular lesion is permanent, the central nervous system adapts to the defect so that vertigo subsides over days or weeks.
- Constant dizziness lasting months is not vestibular.
- some patients describe it as **constant due to frequent episodic dizziness**.
The diagnosis depends **mostly on history, much on examination and little on investigation.**

Neurological Sx: loss of consciousness; weakness; numbness; dysarthria; diplopia; fitting.

- **Episodic with aural symptoms**
  - Menière’s disease (HOURS)
  - Migraine (MINUTES)

- **Episodic without aural symptoms**
  - Benign paroxysmal positional vertigo (SECONDS)
  - Migraine (MINUTES)
  - Transient ischaemic attacks (MINUTES)
  - Epilepsy
  - Cardiac arrhythmia
  - Postural hypotension
  - Cervical spondylosis

- **Constant with aural symptoms**
  - Chronic otitis media with labyrinthine fistula (SECONDS)
  - Ototoxicity
  - Acoustic neuroma

- **Constant without aural symptoms**
  - Multiple sclerosis
  - Posterior fossa tumour
  - Cardiovascular disease
  - Degenerative disorder of the vestibular labyrinth
  - Hyperventilation
  - Alcoholism

- **Solitary acute attack with aural symptoms**
  - Head injury
  - Labyrinthine fistula (SECONDS)
  - Viral infection, e.g. mumps, herpes zoster
  - Vascular occlusion
  - Round-window membrane rupture

- **Solitary acute attack without aural symptoms**
  - Vasovagal faint
  - Vestibular neuronitis (DAYS)
  - Trauma

**Onset of vertigo**

- Character of vertigo, real vertigo, or dizziness
- Duration
- Relationship to the movements of the head
- Other associated symptoms, tinnitus, hearing loss etc.
Duration of vertigo. (orange color associated with hearing loss)

1. Seconds
   A. benign paroxysmal positional vertigo

2. Minutes
   A. Migraine-associated vertigo
   B. Vertibrobasilar insufficiency

3. Hours
   A. Meniere's disease (endolymphatic hydrops)
   B. Otic syphilis

4. Days
   A. Vestibular neuritis
   B. Labyrinthitis

5. Weeks
   A. CNS disorder
   B. Lyme disease
   C. Multiple sclerosis
   D. Acoustic neuroma
   E. Autoimmune

6. Variable duration
   a. Inner ear fistula
   b. Labyrinthine concussion
   c. Blast trauma or Barotrauma
   e. Superior semicircular canal dehiscence syndrome
### Central vs. Peripheral Vertigo

<table>
<thead>
<tr>
<th></th>
<th>Central Vertigo</th>
<th>Peripheral Vertigo</th>
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</thead>
<tbody>
<tr>
<td><strong>Onset</strong></td>
<td>Gradual</td>
<td>Usually Sudden</td>
</tr>
<tr>
<td><strong>Tinnitus, hearing loss</strong></td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td><strong>Neighbourhood signs (Diplopia, cortical blindness, dysarthria,...)</strong></td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Nystagmus</strong></td>
<td>Pure, vertical, suppress with fixation, &amp; multidirectional</td>
<td>Mixed, horizontal, suppress with fixation, &amp; unidirectional</td>
</tr>
</tbody>
</table>
Diagnosis

History:

- **True vertigo**, any sensation of motion
- Any nausea, vomiting, sweating, and abnormal eye movements.
- Occur when moving or changing positions
- **Duration**
- Constant or come and go
- Any new medications
- Recent head trauma
- Other **hearing symptoms** (ringing or hearing loss).
- Other **neurological symptoms** such as weakness, visual disturbances, altered level of consciousness, difficulty walking, abnormal eye movements, or difficulty speaking
Examination

Joint position sense (proprioception), carried in the dorsal columns of the spinal cord; Vision; Vestibular apparatus

- **Romberg's test** is not a test of cerebellar function, it is a test of the proprioception receptors and pathways function.
  
  A positive Romberg's test has been shown to be 90% sensitive for lumbar spinal stenosis.

- **Unterberger test** used to help assess whether a patient has a vestibular pathology.
  
  It is **not useful for detecting central** disorders of balance.
  
  If the patient rotates to one side they may have a labyrinthine lesion on that side (not enough alone)

- **Walking with eyes closed:** repeat three times, if vestibular deficit is present pt gait is deviated or unsteady towards the same side

- **Turning test:** close eyes, walk straight and turn quickly 180 stop at point pt tend to fall toward the side of vestibular weakness (perilymph fistula)
Examination

• The head thrust test: an examination for chronic peripheral vestibular loss, to diagnose a chronic as well as to identify the side of the hypofunctioning labyrinth. Based on the doll’s eye phenomenon

Fistula test: done when perilymph fistula is suspected by pressing on tragus and checking for nystagmus and symptoms
Examination

• **Nystagmus:** **Definition:** Involuntary, rhythmical oscillation of the eyes away from the direction of gaze, followed by a return of the eyes to their original position
  • named after the fast component of the nystagmus caloric test: (37 +/-7)
• **COWS:**
  • Cold: toward the opposite ear (makes the labyrinthine hypoactive)
  • warm: toward the same ear
  • used to validate a diagnosis of asymmetric function in the peripheral vestibular system.
• one of several tests which can be used to test for brain stem death.
Investigation:
- **CT scan** if a brain injury is suspected
- **Blood tests** to check blood sugar levels.
- **ECG** to look at heart rhythm may also be helpful.
BENIGN PAROXYSMAL POSITIONAL VERTIGO

ESSENTIALS OF DIAGNOSIS

1. Sudden vertigo lasting seconds with certain head positions.
2. No associated hearing loss.
3. Characteristic nystagmus (latent, geotropic, fatigable) with Dix-Hallpike test.

Statistics:

- The posterior semicircular canal (PSC) was affected in the majority of cases of BPPV (93% of cases), with 85% being unilateral, and 8% Bilateral.
- The average age of presentation is in the 5th decade
- no gender bias.
- The incidence may range from 10-100/100,000 per year.
- Nearly 20% of patients seen at vertigo clinics are given the diagnosis of BPPV.
- The rate of recurrence may be 10–15% per year.
BPPV

• Arising as a result of mostly due to Canalithiasis

• Predisposing factors of BPPV:
  • Circumstances in which the head is placed or maintained in an inverted orientation (e.g., dental procedures, visits to the hairdresser).
  • Age, Inactivity, Family history
  • Trauma and vestibular neuritis.
  • Other ear disease; Meniere’s syndrome.

• The triggering positions:
  • rolling over in bed into a lateral position, getting out of bed, looking up and back, and bending over.
Dix Hallpike test in diagnosing BPPV

MANEUVER INTERPRETATION

• A positive test is indicated by a latent period of 1-5 seconds during which the patient is minimally symptomatic.

• followed by the acute onset of vertigo and rotatory nystagmus with a rapid component toward the affected side. A typical duration of symptoms and visible nystagmus is 10-40 seconds.

• repeated to the same side; with each repetition, the intensity and duration of nystagmus will diminish.
Treatment of PSC BPPV

Treatment with Repositioning: First-line therapy for BPPV, use gravity to move canalith debris out of the affected semicircular canal and into the vestibule.

• Epley maneuver, gravity is the stimulus that moves the particles within the canal.

• The maneuver is repeated until no nystagmus is elicited. In this way, the Epley maneuver is effective in more than 90% of cases in eliminating BPPV.
Assistive devices

• *Rotator devices*

No author suggests that such a device is required for treatment, however, they may be useful in patients who are difficult to maneuver due to mobility problems in the cervical spine, and simultaneous treatment of bilateral PSC BPPV may be accomplished with a 360° heels over head rotation.
endolymphatic hydrops

• *Primary idiopathic endolymphatic hydrops* (known as Ménière’s disease) is of unknown etiology

• *Secondary endolymphatic hydrops*: head trauma or ear surgery, and it can occur with other inner ear disorders, allergies, or systemic disorders (such as diabetes or autoimmune disorders).
MENIERE DISEASE

• ESSENTIALS OF DIAGNOSIS
  • Episodic vertigo lasting hours.
  • Fluctuating hearing loss.
  • Tinnitus.
  • Aural pressure. (fullness)

Usually starts Unilateral, but in 25% Bilateral
Stabilizing the body’s fluid and electrolyte levels
• A hydrops diet regimen (HDR): minimizing the use of solutes (salts and sugars); Adequate fluid intake 6-8 glasses/day; Caffeine and alcohol restriction.
• Physicians may prescribe diuretics as part of treatment

IDENTIFYING AND TREATING THE UNDERLYING CONDITION

Creating a safe physical environment
Managing persistent symptoms and changes
• Aminoglycoside therapy: intratympanic gentamicin therapy. Generally 10% risk of worsening the hearing loss
• SURGICAL MEASURES Endolymphatic sac decompression; Vestibular neurectomy; Labyrinthectomy
VESTIBULAR NEURITIS

• ESSENTIALS OF DIAGNOSIS
  • Vertigo lasting days after an upper respiratory infection.
  • No hearing loss.
  • No other neurologic signs or symptoms.

• The proposed etiologies for vestibular neuritis include viral infection, vascular occlusion, and immunologic mechanisms

• The patient may have postural instability toward the injured ear but is still able to walk without falling.
Labyrinthitis

• characteristically is viral-induced endolabyrinthitis and is not potentially fatal.

• However, labyrinthitis secondary to middle ear infection can be fatal if suppurative labyrinthitis and, subsequently, meningitis occur.

• Suppurative labyrinthitis= Vertigo + SNHL permanent.

• Therefore, each call from the emergency department to see a patient in whom severe vertigo and hearing loss occur simultaneously requires the clinician to determine whether the middle ear is normal.
Labyrinthitis

• Route of spread into the labyrinth:

In AOM:
  • Weakened oval window membrane: post stapes surgery
  • Dehiscent oval window membrane: as occurs in congenital labyrinthine deformities

COM:
  • Direct bacterial invasion of the labyrinth through a cholesteatomatous Lateral SCC fistula

• Diffuse Suppurative Labyrinthitis:
  • Cause: suppurative otitis media
  • Pathogens: *S. pneumoniae* (most common), *H. influenzae*, and *Neisseria meningitides*
  • Management: admission, IV antibiotic (*to prevent further bacterial invasion intracranially*, *not to reverse SNHL or vestibular damage*)
Herpes zoster oticus — Ramsay Hunt syndrome, this syndrome is believed to represent activation of latent herpes zoster infection of the geniculate ganglion.

In addition to acute vertigo and/or hearing loss, ipsilateral facial paralysis, ear pain, and vesicles in the auditory canal and auricle are typical features.

Acoustic neuroma
- A type of tumor of the nerve tissue.
- Symptoms include:
  a. Vertigo.
  b. One-sided ringing.
  c. Hearing loss.