1. Context

2. Definitions

3. Classification of Dizziness/Vertigo

4. Related Anatomy

5. Differential Diagnosis of Dizziness/Vertigo

6. Causes of Hearing Disturbances

7. History of Dizzy Patient

8. Physical Examination of the Dizzy Patient

9. Common Causes of Vertigo:
   - Symptoms, Assessment, Management

10. Appendix

Meniere’s disease is overdiagnosed.

Classic triad: Vertigo-Tinnitus-Deafness (sensorineural)

VBI is also overdiagnosed as a cause of vertigo.

It often causes dizziness and sometimes vertigo but rarely in isolation.

½ of the population will have suffered from significant dizziness by age 65 and about a ½ by age 80.

Commonest causes in the general practice:
   - Postural hypotension
   - Hyperventilation

Drug history is very important, including prescribed drugs as well as recreational and illicit drugs.

Vertigo:

‘Vertigo’ comes from the Latin word for turning.

Modern medical definition: ‘A sudden sense of movement’

It should describe a hallucination of rotation of self or the surroundings in a horizontal or vertical direction

Dizziness:

‘Dizzy’ comes from an old English word, dysig, meaning foolish or stupid

It means:
   - Unsteadiness or light headedness –
     - without movement or motion or spatial disorientation

Disequilibrium:

Unsteadiness, imbalance, or loss of equilibrium -

Often accompanied by spatial disorientation.

Dizziness: Vertigo: Disequilibrium: Spatial Disorientation:

> Dizziness
> Vertigo
> Disequilibrium
> Spatial disorientation

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Commonest causes in the general practice:
   - Postural hypotension
   - Hyperventilation

Drug history is very important, including prescribed drugs as well as recreational and illicit drugs.
Hallucination of rotation of self or the surroundings in a horizontal or vertical direction, usually described as spinning, which suggests vestibular dysfunction.

Presentation:
> Usually episodic
> with an abrupt onset
> often associated with nausea or vomiting.

Dysfunction can be located in the PNS or CNS.

Prevalence:
> Peripheral vestibulopathies: 35-55% of dizziness cases
> Central vestibular disorders: ~5% of dizziness cases

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> Central vestibular disorders: ~5% of dizziness cases

Impending faint or loss of consciousness, not associated with an illusion of movement.
> May begin with diminished vision or a roaring sensation in the ears
> This subtype of dizziness results from conditions that compromise the brain’s supply of -
> blood
> oxygen
> or glucose
> May be accompanied by transient neurological signs:
> Dysarthria,
> Visual disturbances
> Extremity weakness
> Prevalence of dizziness cases:
> Clinic: 2%
> ED: 16%

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> Clinic: 2%
> ED: 16%

A sense of imbalance without vertigo that is generally attributed to neuromuscular problems.

Unsteadiness leading to increased risk of fall.
> Unsteadiness or imbalance occurs only -
> when erect and
> disappears when lying down or sitting.

This subtype of dizziness may result from:
> visual impairment
> peripheral neuropathy
> musculoskeletal disturbances
> may include ataxia

Prevalence of dizziness cases: 1-15%

Dizziness described as a vague or floating sensation with the patient having difficulty relating the specific feeling to the clinician.

It includes descriptions of vague lightheadedness, heavy-headedness, or wooziness and cannot be classified as any of the 3 previous subtypes.

Psychiatric disorders are the main cause for this subtype:
> Anxiety
> Depression
> Hyperventilation

Prevalence of dizziness cases: 10 - 25%

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> ED: 16%

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Prevalence of dizziness cases: 10 - 25%
The vestibular apparatus consists of:
- Utricle
- Saccule
- 3 Semicircular canals

The sensory transducers: Hair cells are located in the
- Maculae in the utricle and saccule
- Cristae in the semicircular canals

Utricle & Saccule: Expansions in the membranous labyrinth
- Contain: Macula, a patch of hair cells overlain by the otoconial membrane.
- Otolithic membrane: a gelatinous sheet with calcium carbonate particles, the oticones, lying on its surface or embedded in its top layer.
- Functions: Detect the position of the head and the movements of the head relative to gravity.
- As the head moves, the pull of gravity on the otolithic membrane causes it to lag behind.
- Otolithic membrane shifts with respect to the underlying hair cells, the stereocilia of the hair cells are deflected.
- Depending on the direction of the deflection the cells will be either depolarized or hyperpolarized, resulting in an increase or decrease in the generation of action potentials.
- Macula of the utricle: oriented in the horizontal plane
- Macula of the saccule: oriented in the vertical plane
- 3 semicircular canals:
  - Tubes of membranous labyrinth extending from the utricle
  - Anterior/Posterior/Horizontal: Oriented at right angles to each other
- Ampulla:
  - Expanded end at each canal containing
  - Crista ampullaris a cone-shaped structure, covered in "hair cells"
- Cupula: gel-like structure covering the hair cells
  - Because the gel of the cupula does not contain otocinia, it does not respond to gravity.
  - As the head moves, inertia causes the endolymph within the canals to lag behind and push on the cupula.
  - As a result, the stereocilia of the ampullary hair cells bend, and the electrical properties of the hair cells change
  - Working together, the hair cells send signals to the brain encoding head movement in all 3 planes.

Neurotransmitter released by the hair cells in the maculae and ampullae affects the peripheral processes of the primary sensory neurons whose cell bodies form the vestibular ganglion.

The central processes of the primary vestibular neurons form the vestibular component of CN VIII

They travel with the cochlear afferents through the internal acoustic meatus to the vestibular nuclei at the junction of the pons and the medulla.

Vestibular nuclei integrate signals from the vestibular apparatus with input from the spinal cord, cerebellum, visual system & coordinate motor activities involved in eye & skeletal movements.

Vestibular Nuclear Complex: composed of 4 subnuclei sitting at the pontomedullary junction

- They are named the superior, medial, lateral & inferior nuclei.
- Input predominantly from the semicircular canals but also from the otolith organs projects to the superior and medial nuclei, which send signals in the contralateral ascending MLF to coordinate head and eye movements via cranial nerve nuclei III, IV and VI.
- The medial nucleus also forms a substantial bilateral projection caudally to the cervical spinal cord via the descending MLF to coordinate postural head and neck movements.
- Input predominantly from the otolith organs but also from the semicircular canals projects to the lateral nucleus, which projects ipsilaterally to the spinal cord mostly in the lateral vestibulospinal tract to coordinate postural responses to gravity.
- Inferior nucleus which projects bilaterally to the cervical spinal cord by the descending MLF and to the vestibular parts of the cerebellum and the other vestibular nuclei.
- All vestibular nuclei send a small number of axons via the thalamus to the somatosensory cortex for conscious appreciation of balance and head position.
1. Causes of Dizziness
2. Symptoms Accompanying Peripheral Disease
3. Symptoms Accompanying CNS Disease
4. Vertigo
5. Presyncope
6. Disequilibrium
7. Other dizziness

Peripheral vertigo has distinctive features of onset, duration, and accompanying symptoms
Peripheral vertigo comes in spells and usually lasts -
> seconds (e.g. BPPV)
> minutes (e.g. Ménière’s disease)
> hours (e.g. Vestibular neuritis)
Frequent associated symptoms of peripheral disease -
> Hearing loss
> Tinnitus
> Aural fullness
Position changes exacerbate the dizziness
Lying still lessens the symptoms
In most attacks, the onset is sudden
For the most part, patients feel fine between spells

Central causes of dizziness produce a more variable picture
The sensation may be described in a variety of ways:
> tilting, pushed to one side, lightheadedness, clumsiness, black out
If documented loss of consciousness is present –
> a peripheral aetiology of the dizziness is rarely if ever at fault.
Helpful for localization is the presence of accompanying signs of neural dysfunction:
> dysarthria, dysphagia, diplopia, hemiparesis, severe localized cephalgia, seizures, and memory loss.
Time course of symptoms: More variable from minutes to hours
Effect of movement or position change: Less predictable
These symptoms lead the clinician to suspect brain stem or cortical rather than labyrinthine sources.

Peripheral Disorders
- Labyrinth
  - Labyrinthitis viral/bacterial
  - Vestibulitis
  - Menière’s syndrome
  - BPPV
  - Drugs
  - Trauma
  - Otitis media
- VIII Cranial Nerve
  - Vestibular Neuritis
  - Acoustic neuroma
  - Drugs
- Cervicogenic vertigo

Central Disorders
- Brain stem
  - VBI
- Infarction
- Cerebellum
  - Degeneration
  - Tumours
- Migraine
- MS

Peripheral Disorders
Central Disorders
Central Lesion | Peripheral Lesion
---|---
Vertigo | > Often constant | > Often intermittent
   | > Mild to Moderate | > Severe
Nystagmus | > Sometimes absent | > Always present
   | > Multiple directions | > Unidirectional
   | > Possibly vertical direction (upbeat or downbeat) | > Never Vertical
Hearing loss | > Rarely present | > Often Present
Tinnitus | > Rarely present | > Often Present
Brain stem signs | > Typically present | > Never present

### 5.3. DDx characteristics of central vs peripheral vertigo

- **Central Vertigo**
  - Often constant
  - Mild to moderate
- **Peripheral Vertigo**
  - Often intermittent
  - Severe

### 5.4. Presyncope

- **Sensation** of impending faint or loss of consciousness that is not associated with an illusion of movement.
- Results from conditions that compromise the brain's supply of blood, oxygen, glucose; which affects the function of the cerebral hemispheres or brainstem.
- Symptoms typically arise from vascular, autonomic or cardiac causes.
- **Cardiac disease:** Arrhythmia, outflow obstruction or low cardiac output states
- **Autonomic failure** typically results in postural or orthostatic hypotension.
  > This may occur as part of neurologic conditions such as polyneuropathy (e.g. diabetic neuropathy) or certain disorders of the CNS (Parkinson disease).
  > Many medications can also exacerbate or bring on postural hypotension.
- Vaso-vagal episodes are common and may show inappropriately slow heart rates at the time of the attacks, along with hypotension.
- Anxiety states with hyperventilation can cause presyncope.
  > Hyperventilation results in cerebral vasoconstriction due to the loss of CO2 and the attendant change in blood pH.
- Migraine can also produce presyncope (cerebrovascular instability?)

### 5.5. DDx: Disequilibrium

- **Feeling** of being unsteady on one's feet.
- **Improves** when there are other sensory cues (such as the ability to touch things)
- **Worse**, when the patient's vision is blocked, walking on uneven surface.
- **Useful questions**:
  > "Does it only happen when you're on your feet?"
  > "Does it get much better if you touch things?"
  > "Is the sensation worse in the dark?"
Due to disturbance of sensory or motor control systems that are necessary to maintain the upright posture.

The most common cause, by far, is multisensory deficit.

Gradual decrease in sensory acuity in several systems:
- Diminished sensitivity to joint position (proprioception) in the feet
- Decreased sensitivity of the vestibular system
- Decreasing visual acuity

Many of these patients have polymyopathy that is damaging peripheral nerves

Typically, these patients improve with a cane or other gait assistive device.

Patients with Parkinson's or cerebellar disease often have disequilibrium due to motor difficulties.

Slowed responses in Parkinson's disease or incoordination and cerebellar disease may make the patient entirely unable to walk safely.

The patient perceives this as disequilibrium.

Therefore, the evaluation of the patient with disequilibrium requires both testing of sensation and testing of motor function, including strength, tone and coordination.

Described as a vague or floating sensation

Patient having difficulty describing the sensations.

May be associated with anxiety and depression.

Psychiatric disorders are considered the primary cause of this subtype of dizziness accounting for ~10 - 25% of all dizziness cases.

In older adults, anxiety, depression, and adjustment reactions were factors contributing to dizziness.

Rare form of dizziness attributed to otolith dysfunction

Function of the utricle & saccule:
- Provide information on linear motion & acceleration in the horizontal & vertical directions, respectively.

Function on static head tilt due to the presence of otoconia.

Probably caused:
- by an imbalance of otolith signals due to unilateral vestibular loss

This asymmetry in otolith input to the vestibular nuclei causes the individual to sense a tilt of the environment to the side of the involved inner ear.

Not classified under vertigo:
- As the symptom is tilting of the environment and not spinning

Tilting of the environment tends to challenge the diagnostic classification system.

Ischaemia or infarction in the vertebrobasilar system and its branches unilaterally affecting the vestibular nuclei, the MLF, other nuclei involved in the vestibular mechanism, or the thalamus can also result in a patient reporting a subjective tilt of the visual vertical axis in a frontal plane.

1. Overview
2. Conductive type
3. Sensorineural type (hair cells or nerve)
4. Central type (brain)
5. Psychological (malingering)
6. Mechanism usually unclear
The majority of hearing disturbances are sensorineural, either associated with aging or noise exposure.

Conductive hearing losses can generally be fixed by surgery.

Central hearing losses are very rare.

Psychological hearing losses are also very uncommon.

Cerumen
Perforation of tympanic membrane
Otitis media or fluid
Otosclerosis

Presbyacusis (age related hearing loss)
Noise trauma (industrial)
Acoustic neuroma (rare)
Radiation (rare)
Congenital (rare)
Infection due to syphilis (rare)

Brainstem
Auditory cortex

Malingering

Sudden hearing loss
Key History Questions
1. Description of the Spell
2. Timing
3. Behaviour
4. Triggers
5. Associated Symptoms
6. Otolologic history
7. Social history
8. Review of systems
9. Medication history
10. Family history
11. Previous studies

7. History of Dizzy Patient

» Ask patient to describe his or her sensation without using the word “dizziness.”
» Substitute words:
  > Loss of balance
  > Lightheadedness
  > Tilting
  > Whirling
  > Spinning
  > Blurry Vision
  > Feeling of weakness in the legs
  > Giddiness
  > Others

7.1. Description of the Spell

» If your dizziness comes in spells, how long do the spells usually last?
  > Seconds (Quick Spins)
  > Less than a Minute
  > Minutes to Hours
  > Hours to Days
  > Days to Weeks
  > Months to Years (chronic)

7.2. Timing - Duration

» Constant, intermittent?
» Mild, moderate, severe?
» Other neurologic signs symptoms?

7.3. Behaviour of attacks

BPPV
» Vestibular paroxysmia
  > Nerve compression by blood vessels
  > Nerve damage from vestibular neuritis
  > Nerve damage from a tumour - such as an acoustic neuroma
  > Nerve damage from radiation
  > Nerve damage from surgery on the VIII cranial nerve.

Quick Spins 1-5 Seconds
» BPPV
» Cardiac or cardiovascular
» Panic disorder

» Ménière's
» Migraine
» Stroke and other structural CNS lesions
» Vestibular neuritis/labyrinthitis

» Migraine
» Vestibular neuritis/ Labyrinthitis
» Stroke and other structural CNS lesions
» Drug reactions

» Stroke and other structural CNS lesions
» Fluctuating vestibular disorder (e.g. SCD or fistula)
» Multisensory disequilibrium
» Ototoxicity
» Psychiatric

» Position of head? (usually BPPV)
» Head movement in general?
» Visual stimuli (Visual dependence)
» Pressure changes (Fistula?)
» Eating (Migraine or Menieres)
» Seasons (allergy, migraine)
» Menses (migraine)

» Hearing loss
» Tinnitus
» Vomiting
» Nausea
» Migraines
» Headaches
» Tinnitus
» Hyperacusis/Phonophobia (Migraine)
» Fullness (monaural or binaural)
» Hearing loss (timing, fluctuation, monaural/binaural)
» Ear pain (Migraine or TMJ)

7.6. Otologic History

» Drinking/Smoking/Recreational Drugs
» Married with children?
» Frequent flyer?
» Active or sedentary?
» Working?
» Driving?
» Litigation?
» Menopausal?

7.6. Social History

» Put dizziness into context of whole person
  » Cardiac problems?
  » Vascular risk factors?
  » Endocrine (thyroid/DM) problems?
  » Nervous System
  » Autoimmune?
  » Psychiatric?
  » Surgery?

7.7. Review of Systems

» Centrally acting drugs?
» New drugs
» Blood pressure medications (e.g. Hytrin)
» Diabetic?
» Thyroid?
» Exposure to toxins? (Otoxins, Chemo, Amiodarone)

7.8. Medications

» Similar disorder?
» Migraine?

7.9. Family History

» Ear testing (ENG, Audiogram)
» Brain testing (MRI, MRA, CT, EEG)
» Blood testing (CBC, Chem profile)
» Neck (if relevant)

7.10. Previous Studies
1. Spontaneous Nystagmus
2. Gaze-Evoked Nystagmus
3. Smooth Pursuit
4. Dix-Hallpike Maneuver
5. Limb Coordination Tests
6. Gait Observation
7. Romberg Test

8. Vestibular Examination

8.1. Spontaneous Nystagmus

Action: Ask P to fixate on a stationary target in neutral gaze position with corrected vision (glasses/contact lenses in place).
Observe: for nystagmus or rhythmic refixation eye movements.
Interpretation: If nystagmus is observed, particular attention is paid to the amplitude, direction, and effect of target fixation.
Lesions of the labyrinth and CN VIII produce intense, direction-fixed horizontal-rotary nystagmus.
Nystagmus intensifies when gazing in the direction fast phase.
Lesions of the brain stem, cerebellum, and cerebrum cause less intense, direction-changing horizontal, vertical, torsional, or pendular nystagmus.

8.2. Gaze-Evoked Nystagmus

Action: Ask P to gaze at a target 20-30º to the left & right of center for 20 sec.
Observe: for gaze-evoked nystagmus or change in direction, form, or intensity in spontaneous nystagmus.
Interpretation: The ability to maintain eccentric gaze is under control of the brain stem and midline cerebellum, particularly the vestibulocerebellum.
When these mechanisms fail to hold the eye in the eccentric position, the eye drifts toward the midline, followed by refixation saccades toward the target.
Such gaze-evoked nystagmus is central in origin and always beats in the direction of intended gaze.
In contrast, enhancement of peripheral spontaneous nystagmus occurs without direction change when gazing in the direction of the fast phase.
Causes of gaze-evoked nystagmus include: drug effect (sedatives, antiepileptics), alcohol, CNS tumors, and cerebellar degenerative syndromes.
Rebound Nystagmus
Timothy C. Hain, M.D.
Dario Yacovino, M.D.

Cerebellar Degeneration
Dr Rudi Gerhardt

Missteps
Extrapyramidal disease:
P stands
Arm swing
Interpretation of
Acute unilateral
Observe eyes for nystagmus,
Characteristics
Stride length
Relative
Rapid
R
Interpretation:
Finger
P
Duration:
There
Direction
Action:
Fatigue:
Observe for:
Decline with repeated positioning
2 ways to
Interpretation:
Initiation
Interpretation:
Reversal
Latency:
Action:
Heel
P
Cerebellar
Ask P to walk,
Primarily
Functional
Signs
Fine
Brain
Skeletal
Interpretation:
Cerebellar dysfunction:
Normal
Patients
Fatigue
Observe for:
Make sure:
Latency:
Action:
Step
P
Normal
Romberg’s Test:
>P stands with feet close together with eyes open and then eyes closed.
>Observe for:
>Relative amount of sway with vision present vs absent.
>Interpretation of Romberg’s Test:
>Primarily a test of somatosensory and proprioception and not of vestibular input.
>Patients with compensated bilateral vestibular loss stand normally in both eyes-open and eyes-closed Romberg position because of adequate proprioception from the stable support surface.
>2 ways to make this test more sensitive to vestibular deficits:
1. Tandem stance: The support surface cues are sufficiently altered that vestibular cues play a greater role in maintaining upright posture.
2. P stands on a compliant support surface such as 3-inch foam, somatosensory cues are muted and vestibular cues become more important.
1. Benign Paroxysmal Positional Vertigo
2. Meniere’s Disease
3. Vestibular Neuritis and Labyrinthitis
4. Perilymphatic Fistula
5. Cervicogenic Vertigo
6. Acoustic Neuroma

**Nystagmus Direction**

<table>
<thead>
<tr>
<th>Vertical</th>
<th>Torsion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right</td>
<td>Left</td>
</tr>
<tr>
<td>Up</td>
<td>R PSC</td>
</tr>
<tr>
<td>Down</td>
<td>R ASC</td>
</tr>
</tbody>
</table>

**9. Common Causes of Vertigo**

1. Epidemiology
2. Etiology
3. Symptoms
4. Diagnosis & Evaluation
5. Management
6. Outcome
7. Atypical BPPV

**9.1. BPPV**

» BPPV is a common cause of dizziness
» ~ 20% of all dizziness is due to BPPV
» BPPV can occur in children (Uneri & Turkdogan, 2003)
» The older you are, the more likely it is that your dizziness is due to BPPV
» ~ 50% of all dizziness in older people is due to BPPV

Caused by migration of otoconial debris from the utricle, into the SCC:

- Posterior SCC BPPV: Majority of cases
- Lateral SCC BPPV: 1.3-12% (Korres et al, 2002; Hornbrook 2004)
- Anterior SCC BPPV: 2% (Korres et al, 2002)
- Canalolithiasis: Debris floats in the long arm of the canal, inappropriately causing endolymph to move the cupula
- Cupulolithiasis: Debris sticks to the cupula, making it heavier and more responsive

- The utricle may have been damaged by
  - head injury, infection, or other disorder of the inner ear
  - or may have degenerated because of advanced age
- Otoconia turnover:
  - is slow
  - Otoconia probably dissolve naturally
  - As well as actively reabsorbed by the “dark cells” of the labyrinth
- Most common cause:
  - ~50: head injury
  - Older people: degeneration of the vestibular system
  - There is also a strong association with migraine (Ishiyama et al, 2000)

**9.1.2. Etiology**

- Otoconia turnover:
  - is slow
  - Otoconia probably dissolve naturally
  - As well as actively reabsorbed by the “dark cells” of the labyrinth
- Most common cause:
  - ~50 head injury
  - Older people: degeneration of the vestibular system
  - There is also a strong association with migraine (Ishiyama et al, 2000)
Definition: vertigo
- Timing: seconds – max. 2 minutes
- Behaviour of attacks: Episodic, intermittent pattern
- Triggers: certain head positions
- Associated symptoms: Nausea, vomiting
- Intensity of symptoms: Severe
- Symptoms are almost always precipitated by a change of position of the head with respect to gravity:
  - Getting out of bed or rolling over in bed
  - Use of shampoo bowls at hairdressers
  - Certain Yoga positions
  - "Top shelf vertigo": Head extension & rotation

Diagnosis is based:
- Case history
- Physical examination findings
- Vestibular and auditory test results
- Often: Diagnosis can be made with history and physical examination alone
- Dix-Hallpike test consists of:
  - Observation of a burst of nystagmus
  - Patients sensation of vertigo
- Key observation:
  - vertigo is triggered by lying down, or on rolling over in bed.
- Other conditions that have positional dizziness:
  - get worse on standing rather than lying down (orthostatic hypotension)
  - Patients with central vertigo such as spinocerebellar ataxia
  - may have "bed spins" and prefer to sleep propped up in bed
- These conditions can generally be detected
  - on a careful neurological examination
  - by a family history of other persons with similar symptoms

Upbeating and torsionally with the upper pole of both eyes beating toward the undermost ear (geotropic).
- This latter component becomes more pronounced when the patient looks in the direction of the uppermost ear.

Nystagmus: Posterior SCC
- Dix-Hallpike: Right posterior semicircular canal
- BPPV: posterior SCC upbeating, right torsional nystagmus
Epley Maneuver (right ear)

P is seated on a table, before moving into position B, turn head 45° to side being treated.
P in Dix–Hallpike head hanging position. Particles gravitate in ampullofugal direction and induce subsequent counterclockwise rotatory nystagmus. Position is maintained for 1–2 minutes.
P’s head is then rotated toward the opposite side with the neck in extension through position C and into position D in a steady motion by rolling the P onto the opposite lateral side.
The change from position B to D should take no longer than 3–5 seconds.
Particles continue gravitating in an ampullofugal direction through the common crus into the utricle.
P’s eyes are immediately observed for nystagmus.
Position D is maintained for 1–2 minutes, and then the patient sits back up to position A.

1. Start at side of Dix–Hallpike Test
2. P is held in the right head hanging position for 20–30 sec. or until nystagmus exhausts.
3. Head is turned 90° toward the unaffected side, and is held for 20–30 sec. or until nystagmus exhausts.
4. P is rolled onto unaffected side, turned another 90° so the head is nearly in the face-down position. Position is held for 20–30 sec. or until nystagmus exhausts.
5. P is brought to the sitting position.

(Fife TD et al, 2008)

By 1 week: 94% of patients who underwent the Epley Maneuver were asymptomatic.
At 4 weeks: 85% of patients were asymptomatic.
Are other maneuvers as successful?
Semont maneuver is possibly more effective than no treatment, or sham treatment, or Brandt–Daroff exercises as treatment for posterior canal BPPV.
Are postmaneuver activity restrictions necessary after canalth repositioning procedure?
There seems to be little difference in the rate of treatment success whether or not restrictions were included.
What is the efficacy of Brandt–Daroff exercises, habituation exercises, or patient selfadministered treatments for BPPV?
Patients treated with Brandt–Daroff exercises, “habitation exercises,” did no better than those treated with a sham procedure.

(Fife TD et al, 2008)

9.1.5. Outcome

9.1.6. Atypical BPPV
Prevalence: accounts for 10% - 17%

Direction changing paroxysmal positional nystagmus:
- Horizontal, changes direction when the head is turned ⃝ & ⌒ while supine,
- May be either geotropic or apogeotropic,
- Geotropic form: resulting from free-moving otocional debris in the long arm of the SCC, is generally more responsive to treatment,
- Apogeotropic form: due to otocional material in the short arm of the SCC or attached to the cupula (cupulolithiasis),
- Hence, one seeks to convert the more treatment-resistant apogeotropic to the more treatment-responsive geotropic nystagmus form of horizontal canal BPPV.

Provocation Maneuver:
- Nystagmus & vertigo may be provoked by Dix–Hallpike Maneuver
- More reliably induced by: Supine roll test or Pagnini–McClure maneuver

Cause: Many cases are seen as a consequence of an Epley maneuver.

HC-BPPV differs from PC-BPPV mainly in that:
- Vertigo is more intense
- Triggered by rotary movements of the head or body in supine position
- Nystagmus is horizontal instead of vertical-torsional

Geotropic Variant:
- Most cases
- Rotation to the pathological side from supine position causes a very intense horizontal nystagmus beating towards the undermost ear (geotropic);
- When the patient is rolled to the other, healthy side, there is a less intense horizontal nystagmus again beating towards the undermost ear.

Ageotropic Variant:
- Less frequent
- Presents with horizontal nystagmus beating towards the uppermost ear (ageotropic)
- The side with the less prominent nystagmus is taken to be the affected horizontal SCC.

Lateral canal BPPV side of origin and mechanism are based upon the direction and intensity of nystagmus in the 2 lateral head positions.

1. Very short latency (a few seconds)
2. Paroxysmal character
3. Duration: <1 minute
4. Purely horizontal (geotropic or apogeotropic) position and greater intensity on one side
5. Not fatigable with repeated positioning

Note: Direction of nystagmus in each position determines whether the horizontal canal BPPV is of the geotropic or ageotropic type.
Position 1: P is seated on the examination couch with both the legs hanging out from the same side, arms held close to the body and hands resting on the knees.

Position 2: P is then made to lie down on the uninvolved lateral side with a quick lateral movement and maintained in this position for 2 minutes until the end of evoked geotropic nystagmus.

Position 3: quick 45° rotation of the head towards the floor, position being maintained for 2 minutes.

Position 4: slow return back to the starting position.

Geotropic Variant
- In most cases, rotation to the pathological side from supine position causes a very intense horizontal nystagmus beating towards the undermost ear (geotropic)
- When the patient is rolled to the other, healthy side, there is a less intense horizontal nystagmus again beating towards the undermost ear.
- Start Gufoni's on uninvolved side!

Ageotropic Variant
- Less frequently, the syndrome presents with horizontal nystagmus beating towards the uppermost ear and is usually more intense when the affected ear is uppermost.
- Start Gufoni's on involved side!

79% of the patients treated with the Gufoni's maneuver had complete resolution of symptoms.
6.9% did not show any improvement in their symptoms.
The remaining 13.8% had a conversion into posterior semicircular canal BPPV during treatment and were successfully treated with Epley's maneuver.

Is also rare, and a recent study suggested that it accounts for about 2% of cases of BPPV (Korres et al 2002).
It is diagnosed by a positional nystagmus with components of downbeating and [sometimes] torsional movement on taking up the Dix-Hallpike position (Hain TC 2013).
Anterior canal BPPV is usually transitory and most often is the result of “canal switch” that occurs in the course of treating other more common forms of BPPV.
We identified only two studies specifically addressing the treatment of anterior canal BPPV; both were Class IV studies.
Success rates were between 92% and 97%, though there were no controls to determine whether this represents an improvement over the natural history of this frequently self-resolving form of BPPV (Five et al 2008).

Is a condition in which debris is stuck to the cupula of a semicircular canal, rather than being loose within the canal.
Cupulolithiasis should result in a constant nystagmus.
This pattern is sometimes seen (limouza et al. 1995).
Cupulolithiasis might theoretically occur in any canal - horizontal, anterior or vertical, each of which might have its own pattern of positional nystagmus.
If cupulolithiasis of the posterior canal is suspected, it seems logical to treat with the Epley maneuver.
Other maneuvers have been proposed for lateral canal cupulolithiasis.
There are no controlled studies of cupulolithiasis to indicate which strategy is the most effective.
» Is a hypothetical condition in which debris is present on the vestibule-side of the cupula, rather than being on the canal side.
» For this theory, there is loose debris, close to but unattached to the cupula of the posterior canal, possibly in the vestibule or short arm of the semicircular canal.
» This mechanism would be expected to resemble cupulolithiasis, having a persistent nystagmus, but with intermittency because the debris is movable.
» Very little data is available as to the frequency of this pattern, and no data is available regarding treatment.

*9.16.d. Vestibulolithiasis*

» If debris can get into one canal, why shouldn’t it be able to get into more than one?
» It is common to find small amounts of horizontal nystagmus or contralateral downbeating nystagmus in a person with classic posterior canal BPPV.
» While other explanations are possible, the most likely one is that there is debris in multiple canals.
» Gradually a literature is developing about these situations (Bertholon et al, 2005).

*9.16.e. Multicanal BPPV*

1. Epidemiology
2. Classic Presentation
3. Causation
4. Evaluation
5. Management

*9.2. Meniere’s Disease*

» Prevalence: about 0.2 % of the population has Meniere’s disease.
» The prevalence increases with age, rather linearly, up to the age of 60.
» 1.5:1 ♀/♂ Ratio

*9.2.1. Epidemiology*

» A typical attack of Meniere’s disease is preceded by
  » Aural fullness
  » Hearing fluctuation or changes in tinnitus
  » A Meniere’s episode generally involves
  » Severe vertigo, imbalance,
  » Nausea and vomiting
  » Episodes last for several hours to a day
  » Vertigo-free periods lasting for weeks or months.
  » Following a severe attack, most people find that they are exhausted and must sleep for several hours.
  » There is a large amount of variability in the duration of symptoms.
  » Hearing loss is progressive.
  » Vertigo attacks appear to “burn out” over time.
  » Some patients have sudden “drop” attacks without loss of consciousness.

*9.2.2. Classic Presentation*
Idiopathic endolymphatic hydrops: Distention from either overproduction or retention of endolymph appears to be the cause in most cases. The area of the ear affected is the entire labyrinth, which includes both the semicircular canals and the cochlea. Specifically, there appears to be a problem with the endolymphatic sac's (immune processing area) and/or duct's filtration and excretion function with a possible autoimmune etiology. An association between high levels of ADH and stress has been found in Meniere's patients. Head trauma or previous infection may be factors. Pregnant females may be more prone. Meniere's disease is the 4th most common cause of vertigo.

9.2.3. Causation

The primary conservative approach is based on the theory that increased fluid causes distention and symptoms. Therefore, diuretic therapy (herbal or prescribed) in combination with a salt-restriction diet appears to be effective in managing the vertiginous component of Meniere's disease in ~5% of patients. For a few patients, surgical intervention using decompression of the endolymphatic sac appears effective. Other approaches include transtympanic gentamicin and, for intractable cases, vestibular nerve section. Although, in one study, half of patients had residual subjective complaints, 85% were satisfied with their decision. The possibility of an overlap between Meniere's disease and cervicogenic vertigo warrants a treatment trial of cervical manipulation in patients with Meniere's disease.

9.2.4. Evaluation

Recurrent, sudden onset of vertigo with associated hearing loss or tinnitus is fairly diagnostic of Meniere's disease. Both ears are affected in 30% - 50% of patients over time. Meniere's disease often breaks the general rules of ear disease: Hearing loss in Meniere's usually starts out with a low-frequency sensorineural frequencies. Almost all other ear diseases begin with damage to the highest frequencies first. Patients who fit the vertigo pattern but do not have auditory dysfunction may have recurrent vestibulopathy.

Management

1. Epidemiology
2. Classic Presentation
3. Causation
4. Evaluation
5. Management

During an acute attack, lay down on a firm surface. Stay as motionless as possible, with your eyes open and fixed on a stationary object. Do not try to drink or sip water immediately, as you'd be very likely to vomit. Stay like this until the severe vertigo (spinning) passes, then get up SLOWLY. After the attack subsides, you’ll probably feel very tired and need to sleep for several hours. If vomiting persists and you are unable to take fluids for longer than 24 hours (12 hours for children), contact your doctor.
5% of all dizziness (perhaps 15% of vertigo) is due to Vestibular Neuritis or Labyrinthitis. Occurs in all age groups, but rare in children.

Symptoms of vestibular neuritis & Labyrinthitis typically include:
- Dizziness or Vertigo
- Disequilibrium or Imbalance
- Nausea
- Acutely:
  - the dizziness is constant
  - After a few days:
    - Symptoms are often only precipitated by sudden movements.
  - A sudden turn of the head is the most common 'problem' motion.
  - While patients with these disorders can be sensitive to head position, it is generally not related to the side of the head which is down, but rather just whether the patient is lying down or sitting up.
  - If there are hearing symptoms with dizziness, then labyrinthitis would be the first consideration.

Vestibular neuritis:
- Attributed to a viral infection of the vestibular nerve
- When 1 of the 2 vestibular nerves is infected, there is an imbalance between the 2 sides, and vertigo appears.
- Vestibular neuronitis: another term used for the same clinical syndrome.
- "Neuritis" implies damage to the nerve
- "Neuronitis" implies damage to the sensory neurons of the vestibular ganglion.
- There is also some evidence for viral damage to the brainstem vestibular nucleus (Arbusow et al, 2000), a second potential "neuronitis".
- Labyrinthitis:
  - a combination of the symptoms of vestibular neuritis, with the addition of hearing symptoms.
  - It may be due to a process that affects the inner ear as a whole, or due to a process that affects the 8th nerve as a whole.
  - Labyrinthitis is also always attributed to an infection.
- Vestibular neuritis and labyrinthitis are rarely painful
  - when there is pain it is particularly important to get treatment rapidly as there may be a treatable bacterial infection or herpes infection.

It is important to differentiate vestibular neuritis (peripheral) from cerebellar stroke (central).
- Nystagmus is made worse by looking in the direction of the unaffected side (Alexander’s law) and is reduced by visual fixation with vestibular neuritis.
- Central nystagmus is not reduced by visual fixation and may change direction.
- There are often other neurologic signs and symptoms with cerebellar involvement such as dysarthria, ataxia, and difficulty with repeated supination/pronation or finger-to-nose testing.
Central compensation occurs, and the condition resolves over time.
» Even when nystagmus is present, it is important to begin vestibular training with having the patient focus on a target with head movement in all directions, and eye-head coordination exercises.
» Helpful exercises are to have the patient focus on a target while moving the head up and down and side to side.
» Balance exercises are incorporated as soon as possible.
» Medication may be needed during the acute phase.

9.3.4. Management

- Tai Chi
- Balance
- Cawthorne-Cooksey Exercises

Gaze Stabilization Exercises

Figure 2A: Look straight ahead
Figure 2B: Turn your head 40 degrees towards the right
Figure 2C: Turn your head 40 degrees towards the left

Vestibular Rehabilitation

Tai Chi

Balance

(Hain T, 2003)
Diligence and perseverance are required.

The earlier and more regularly the exercise regimen is carried out, the faster and more complete will be the return to normal activity.

Ideally these activities should be done with a supervised group.

Individual patients should be accompanied by a friend or relative who also learns the exercises.

Cawthorne-Cooksey Exercises

Dr Rudi Gerhardt

1. Epidemiology
2. Classic Presentation
3. Causation
4. Evaluation
5. Management

9.5. Perilymphatic Fistula

Dr Rudi Gerhardt

- Changes in air pressure in the middle ear normally don’t affect the inner ear.
- When a fistula is present, changes in middle ear pressure will directly affect the inner ear, stimulating the balance and/or hearing structures within and causing symptoms.
- There is no classic presentation because onset, intensity, and frequency vary.
- Many patients have a history of barometric pressure changes, as with diving or air flight, or internal pressure development through intense weight lifting.
- Symptoms may include dizziness, vertigo, imbalance, nausea, and vomiting.
- Usually however, patients report an unsteadiness which increases with activity and which is relieved by rest.
- Some people experience tinnitus or aural fullness, many notice a hearing loss.
- Symptoms may get worse with coughing, sneezing, or blowing their noses, as well as with exertion and activity.
- "Valsalva induced dizziness" can also be associated with other medical conditions in for example, the Chiari malformation.

9.5.1. Classic Presentation

Dr Rudi Gerhardt

1. In bed or sitting
   1. Eye movements -- at first slow, then quick
   2. up and down
   3. from side to side
   4. focusing on finger moving from 3 feet to 1 foot away from face
   2. Head movements at first slow, then quick, later with eyes closed
   1. bending forward and backward
   2. turning from side to side
2. Sitting
   1. Eye movements and head movements as above
   2. Shoulder shrugging and circling
   3. Bending forward and picking up objects from the ground
3. Standing
   1. Eye, head and shoulder movements as before
   2. Changing form sitting to standing position with eyes open and shut
   3. Throwing a small ball from hand to hand (above eye level)
   4. Throwing a ball from hand to hand under knee
   5. Changing from sitting to standing and turning around in between
4. Moving about (in class)
   1. Circle around center person who will throw a large ball and to whom it will be returned
   2. Walk across room with eyes open and then closed
   3. Walk up and down slope with eyes open and then closed
   4. Walk up and down steps with eyes open and then closed
   5. Any game involving stooping and stretching and aiming such as bowling and basketball

Head trauma is the most common cause (eg direct blow to the ear).

Fistulas may also develop following rapid or profound changes in intracranial or atmospheric pressure, such as may occur with SCUBA diving, or even just dives into a swimming pool (Klingmann et al, 2007; Rozsasi et al, 2003).

In pregnancy, collagen changes throughout the body, and fistulae may arise spontaneously or in association with delivery.

Children are likely more prone to develop fistulae because of more widely open pathways between the inner ear and the spinal fluid.

Ear surgery, eg for otosclerosis, often creates a fistula.

Some patients develop a fistula, following airplane descent.

Fistulas may be present from birth (usually in association with deafness) or may result from chronic ear infections.

9.5.1. Epidemiology

Dr Rudi Gerhardt

- Opening develops between the middle and inner ear (oval or round window rupture), allowing leakage of perilymph.
- Perilymphatic fistulae are a rare cause of vertigo.
- ‘Dehiscence’ is similar to a fistula, but not as severe.
- Bone is missing, over one of the semicircular canals, uncovering a membrane.
- This dehiscence makes the ear more sensitive to pressure and noise.

9.5.2. Causation

Dr Rudi Gerhardt

- Patients have a history of barometric pressure changes, as with diving or air flight, or internal pressure development through intense weight lifting.
- Symptoms may include dizziness, vertigo, imbalance, nausea, and vomiting.
- Usually however, patients report an unsteadiness which increases with activity and which is relieved by rest.
- Some people experience tinnitus or aural fullness, many notice a hearing loss.
- Symptoms may get worse with coughing, sneezing, or blowing their noses, as well as with exertion and activity.
- "Valsalva induced dizziness" can also be associated with other medical conditions in for example, the Chiari malformation.
Meniere’s disease, which is much more common than fistula, can have identical symptoms, including pressure sensitivity.

- For this reason, fistula diagnoses made in patients without barotrauma are easily questioned.
- Oval window fistulae are often accompanied by hyperacusis and tinnitus.
- Oval window fistulae are largely caused by very loud noises.
- Round window fistulae are mainly characterized by exercise induced dizziness.
- These types of fistulae are mainly induced by barotrauma, such as airplane flights or SCUBA diving.
- There are small amounts of horizontal nystagmus in many persons with dizziness, lacking a diagnosis.

In ~90%, a window fistula fistula will heal itself if activity is restricted.

- Avoidance of activities where there is a possibility of creating high pressure (e.g. airplane travel, Scuba diving, power lifting, horn playing, etc) is usually advised.
- It is usual to wait 6 months before embarking on surgical repair, given that hearing function is reasonable and is stable or improving.

1. Classic Presentation
2. Causation
3. Evaluation
4. Management

It is believed that either overstimulation of upper cervical proprioceptors or degeneration of these proprioceptors or their pathways may cause an imbalance of information leading to a perception of vertigo or disequilibrium.

Findings of upper cervical soft tissue involvement and restricted movements are possible.
- The Fitz-Ritson rotation test may help differentiate.
- The examiner stabilizes the patient’s head while the patient rotates his or her body in a chair.
- If the patient becomes dizzy, a vertebrogenic source is suggested because it is believed that vestibular stimulation is eliminated with this maneuver.
Cervical manipulation may be beneficial and should be applied as a treatment trial. It is important to consider that because of the proprioceptive input of the upper cervical area, cervical manipulation may serve to benefit other causes of vertigo or that there may be an overlap between cervicogenic vertigo and other types.

Patient presents with a complaint of mild but constant hearing loss and dizziness sometimes with associated tinnitus. The onset is gradual and may be ignored initially. There are rarely acute attacks.

Acoustic Neuroma: Benign Schwannoma of CN VIII
Acoustic neuroma occurs in 2 forms:
1. A sporadic form
2. A form associated with Neurofibromatosis
About 95% of all cases are sporadic.
It is located in the cerebellopontine angle, where other cranial nerves are susceptible to compression.
As the tumor grows it may cause brain stem compression.

1. Epidemiology
2. Classic Presentation
3. Cause
4. Evaluation
5. Management

10 acoustic neuromas are newly diagnosed each year per million persons (Evans et al, 2005)
They comprise about:
> 6% of all intracranial tumors
> 30% of brainstem tumors
> 85% of tumors in the region of the cerebellopontine angle
(Anderson et al, 2000)
Acoustic neuromas occur largely in adults -- they are very uncommon in children.
Surgical excision is necessary.