Vertigo, tinnitus

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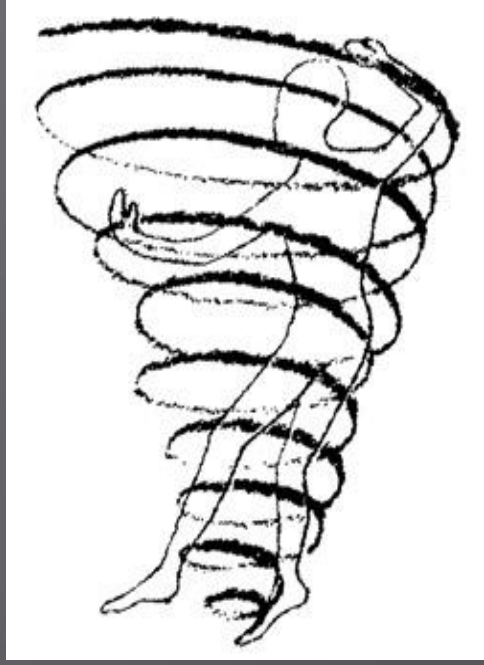
Vertigo



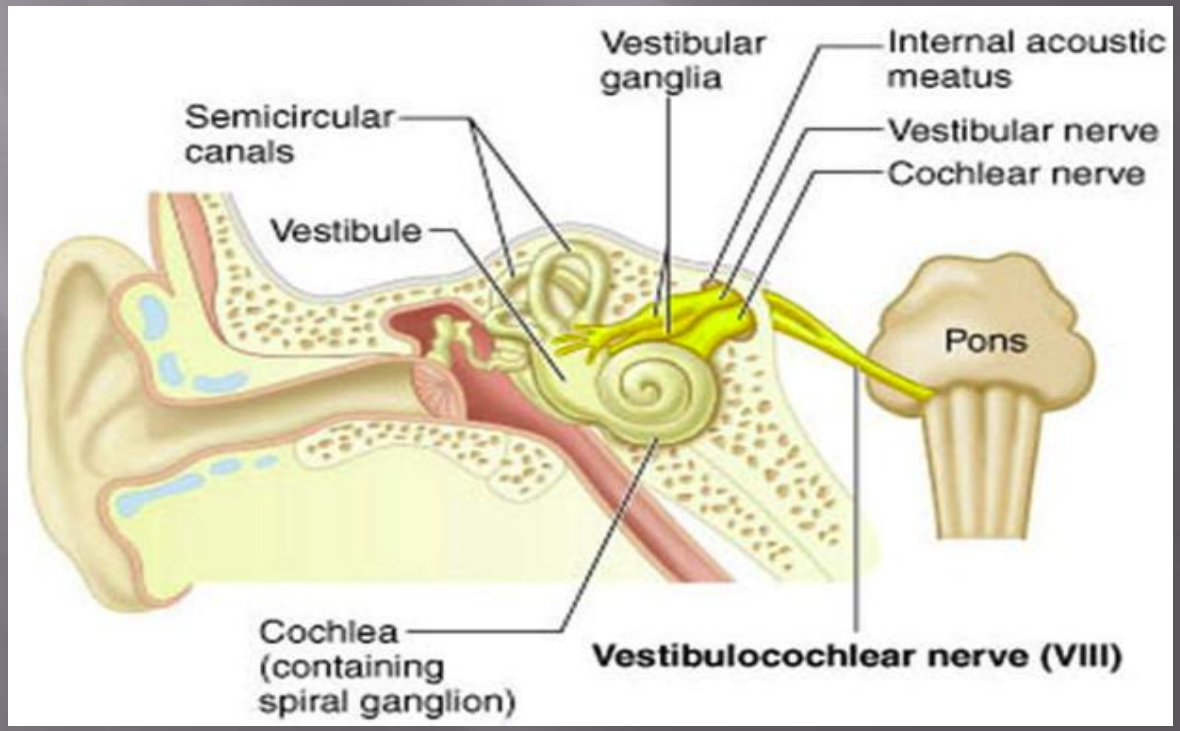
The sensation that you are moving, or everything moves around you.

Causes of vertigo and dizziness

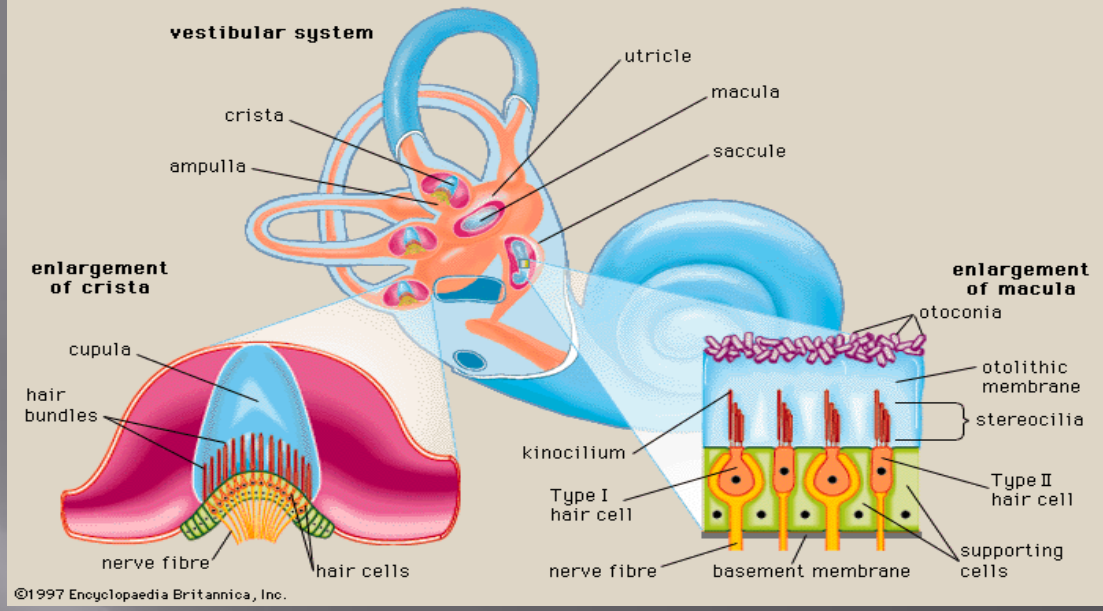
- **ENT disorders**
  - BPPV
  - Vestibular neuritis
  - Ménière-disease (M.D.)
  - Bilateral vestibulopathy
  - Labyrinthitis
  - Fracture of the temporal bone
  - Vestibular schwannoma
  - Superior semicircular canal dehiscence (SSCD)
  - Vestibular migrain
  - Vestibular paroxysmia
- **Neurological diseases**
  - Ischaemia/haemorrhage
  - TIA (tranzient ischaemic attacks)
  - Cerebellar tumors
  - Virus infections
  - Multiplex sclerosis
  - Antiepileptic, anxiolytic drugs
- **Internal diseases**
  - Orthostatic hypotension
  - Hypertension, antihypertensive drugs
  - Metabolic disorders– pl.diabetes mellitus, thyroid
  - Arrhytmia cordis
  - **Heart diseases: 63% has dizziness, 37%: the only symptom!!!**
  - Atherosclerosis
  - Anaemia
  - Toxins, kidney and liver diseases
- **Psychogenic** (panic, phobia)
- **Ophtalmic diseases**

Balance maintenance

- **VISUAL system**
- **PROPRIOCEPTIVE system** (our body's ability to sense where we are in relationship to our surroundings) = kinaesthetic information from the receptors in the skin, muscles, tendon, and joints
- **VESTIBULAR system** (**PERIPHERAL** and **CENTRAL**)



Semicircular canals



Our vestibular system contains three semicircular canals:

- the *horizontal* (or *lateral*),
- the *anterior* (or *superior*)
- the *posterior* (or *inferior*) semicircular canal.

They respond to rotations in their plane.
Anterior and posterior canals may be collectively called *vertical canals*.

Otolith Organs (utricule, saccule)

- While the semicircular canals respond to rotations, the otolithic organs sense linear accelerations.
- The **otoconia crystals** rest on a viscous gel layer, and are heavier than their surroundings. Therefore they **get displaced during linear acceleration**, - produces a sensory signal.

Peripheral disorders

Trauma:

- Fracture
- Perilymph-fistula

Infection:

- Vestibular neuritis
- Herpes zoster oticus
- Labyrinthitis

Vascular:

- Neurovascular compression (VIII. cranial nerve compression by vascular loop)

Tumors:

- Vestibular schwannoma

Others:

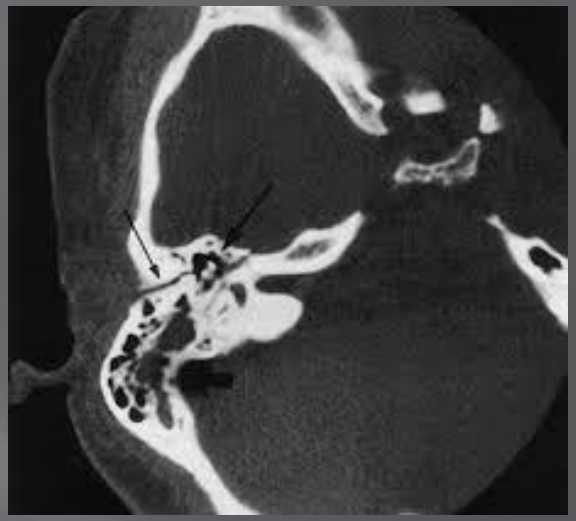
- Ménière's disease
- BPPV (benign paroxysmal positional vertigo)
- Superior semicircular canal dehiscence (SSCD)
- Bilateral vestibulopathy

Trauma

- **Fracture of the temporal (pyramid) bone**

  Cause of vertigo:

  - labyrinth commotion
  - labyrinth injury
  - vestibular nerve injury

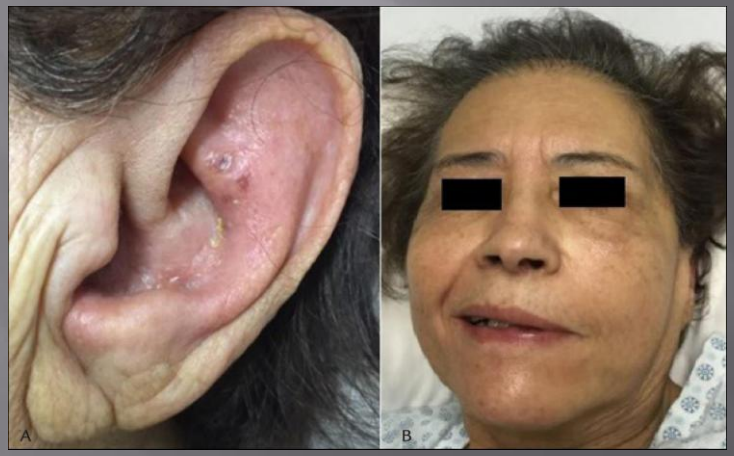


- **Perilymph fistula**
  - Round window / Oval window rupture – due to increased pressure
  - Symptoms: vertigo, tinnitus, progressive hearing loss
  - Therapy: surgery

Infections

Herpes

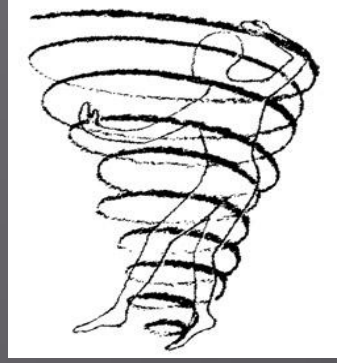
- herpes zoster oticus
- Ramsay-Hunt syndrome: facial nerve (N.VII), cochlear nerve, vestibular nerve (n.VIII) involvement



Labyrinthitis

- Purulent –loss of periferal vestibular function, deafness
- Serous – arousal symptoms, functions may remain

Vestibular neuritis / neuronitis



= Loss of peripheral vestibular function – on one side

Frequent

Cause: Viral infection (HSV) of the vestibular nerve (the branch of the 8th cranial nerve) is believed to be the most common cause / Acute localized ischemia also may be a cause.

Patient history:

- **Sudden onset** - begins in minutes or in a few hours, (viral infection can be before)
- Severe attack of **vertigo** with **nausea and vomiting**
- **Lasts more than 24 hours** (2-3 days), slow improvement, imbalance can remain for months.
- Vertigo even without movement!!! - But motion increases their complaints

Status:

- **Harmonic periferal syndrome**
  - HR (horisonto-rotatory) nystagmus toward the healthy side
  - Patient tends to fall toward the affected side during Romberg tests

  HIT (head impulse test / Halmágyi's test) positive on the affected side!!!= saccadic eye movement turning to the affected side
- videoHIT, Caloric test, VEMP

Vestibular neuronitis

Caloric test: Affected side has impaired or no response to caloric (30°, 44°) stimulation.

Head impulse test (HIT):

Ask the patient to maintain fixation on a fix target, move her head passively, rapidly and randomly to the left and right.

Normal (negative): gaze stays on the fixated target = intact periferal function

Abnormal (positive): the eyes move with the head and a corrective saccad appears after head movement.

Therapy:

Hydration if vomiting persists, antiemetic drugs

Early mobilization!!!– helps the central compensation (eliminates the symptoms).

Corticosteroids, betahistin, vitamin B, antiviral drugs

Ménière's disease (M.D.)
(*not a syndrome!*)

Michael Finney: Vertigo

Affects hearing and balance!
Background: Endolymph hydrops

- ATTACKS (20 minutes-12 hours):
  - **- Vertigo** with nystagmus
  - - Nausea and vomiting
  - **- Hearing loss** on the affected side (sensorineural, fluctuating) – first the low frequencies, than progressive
  - **- Tinnitus** on the affected side (low tone) or **ear fullness**
  - - No neurological signs (like double vision, headache…)

  At least 2 attacks and a documented hearing loss for the dg!!!

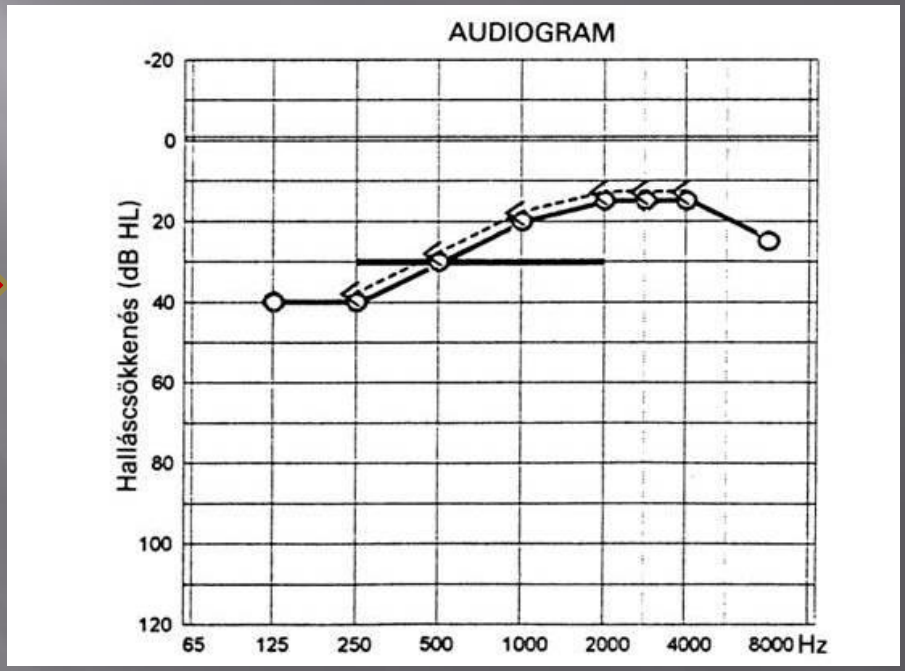
  Dg: **tipical attacks, tipical audiogram, definitive test: ECoG =electocochleography** (shows endolymph hydrops)

- Therapy:
  - Attacks: antiemetic drugs, hydration
  - Betahistine (to prevent attacks)
  - Intratympanal gentamicin = chemical labyrinthectomy
  - Surgery ? (saccotomy, labyrinthectomy, neurectomy – vestibular nerve)
  - **+ rehabilitation (hearing – cochlear implantation, tinnitus, vestibular training)**

Pathophysiology

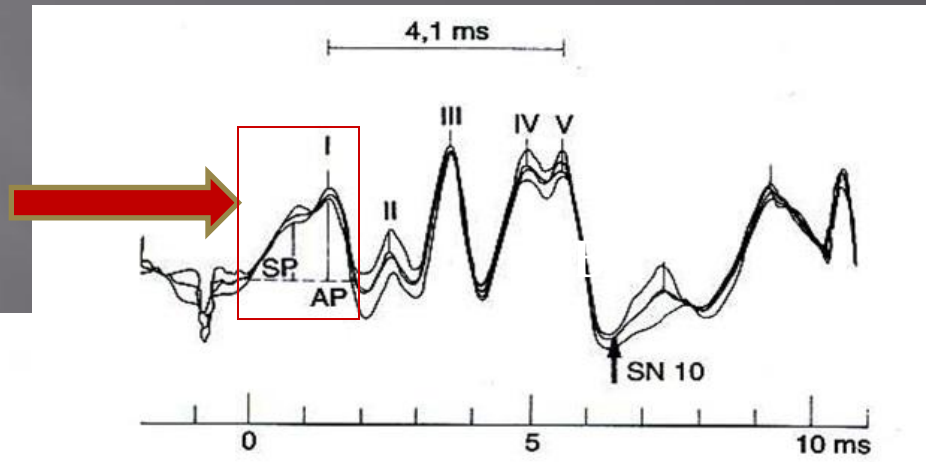
- - Genetic factors
- - Extrinsic factors (trauma, otosclerosis, chronic suppurative otitis)
- - ADH (vasopressin)
- - Allergy
- - Viral infections (CMV)
- - Autoimmune reaction
- - Excytotoxicity, apoptosis

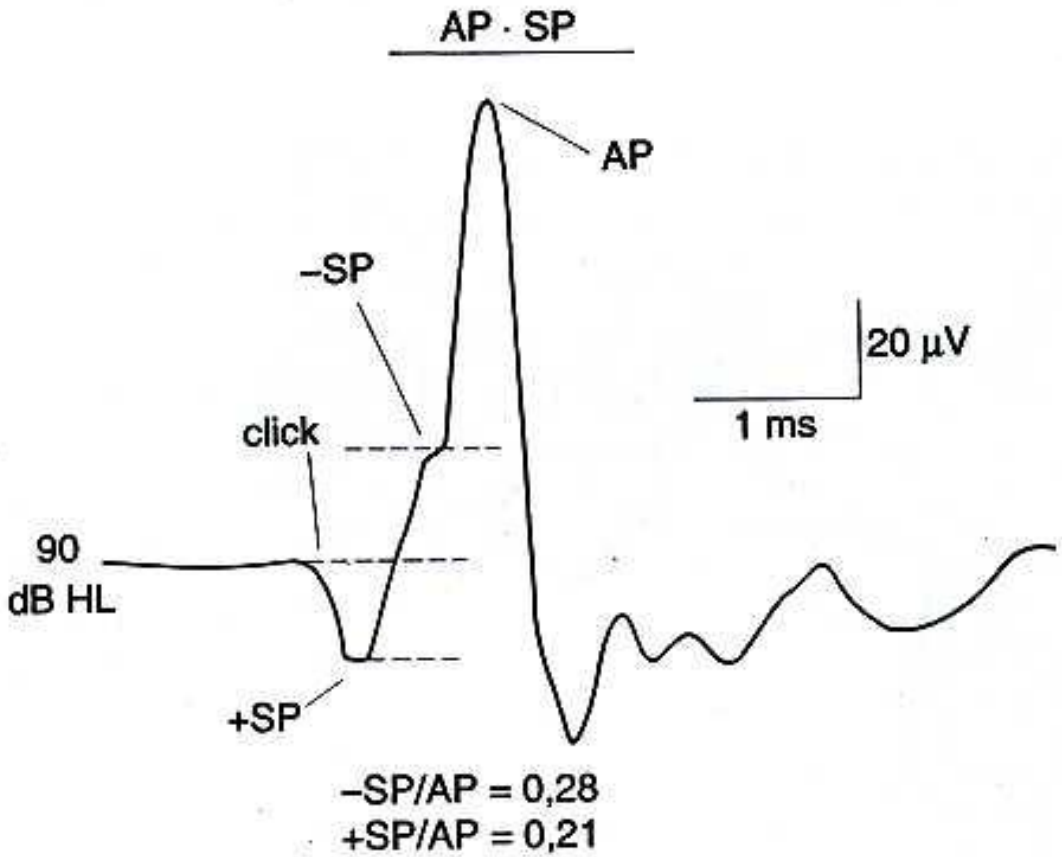
Pure tone audiometry



Objective Audiology

Normal EcoG





BERA / ABR – ECoG

Endolympha hydrops

Diagnostic criteria

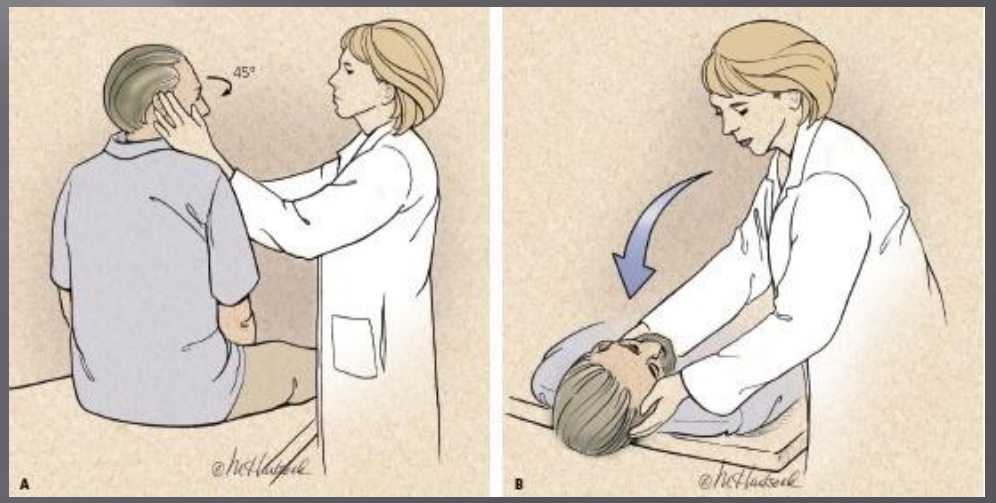
- „**Certain**” : histopathology signs of EL hydrops,
- „**Definite**”: 2 or more tipical vertigo attaks, SN HL measured by audiometry, tinnitus, fullness in the affected ear
- „**Probable**” : at least 1 attack, SN HL, tinnitus, fullness in the affected ear
- „**Possible**” : 1 tipical vertigo attack, no audiometry

BPPV = Benign paroxysmal positional vertigo

Most frequent (older age, trauma, osteoporosis, neuronitis, Ménière, migrain)

Cause: **Canalolithiasis (cupulolithiasis - rare)**

Vertigo **attacks last for max. 1 minute, provoked by a specified head movement**, usually with nystagmus.



- **Posterior canal BPPV**
  - Dg: **Dix-Hallpike maneuver**
  - Therapy: **Epley-maneuver**
- **Horizontal canal BPPV**
  - Dg: **Supine roll test**
  - Therapy: **BBQ maneuver**
- **BPPV type 2.**
  - Dg: Dix-Hallpike maneuver
  - Therapy: sit up from Dix-Hallpike position

Posterior
Semi-Circular
Canal

Dark Cells

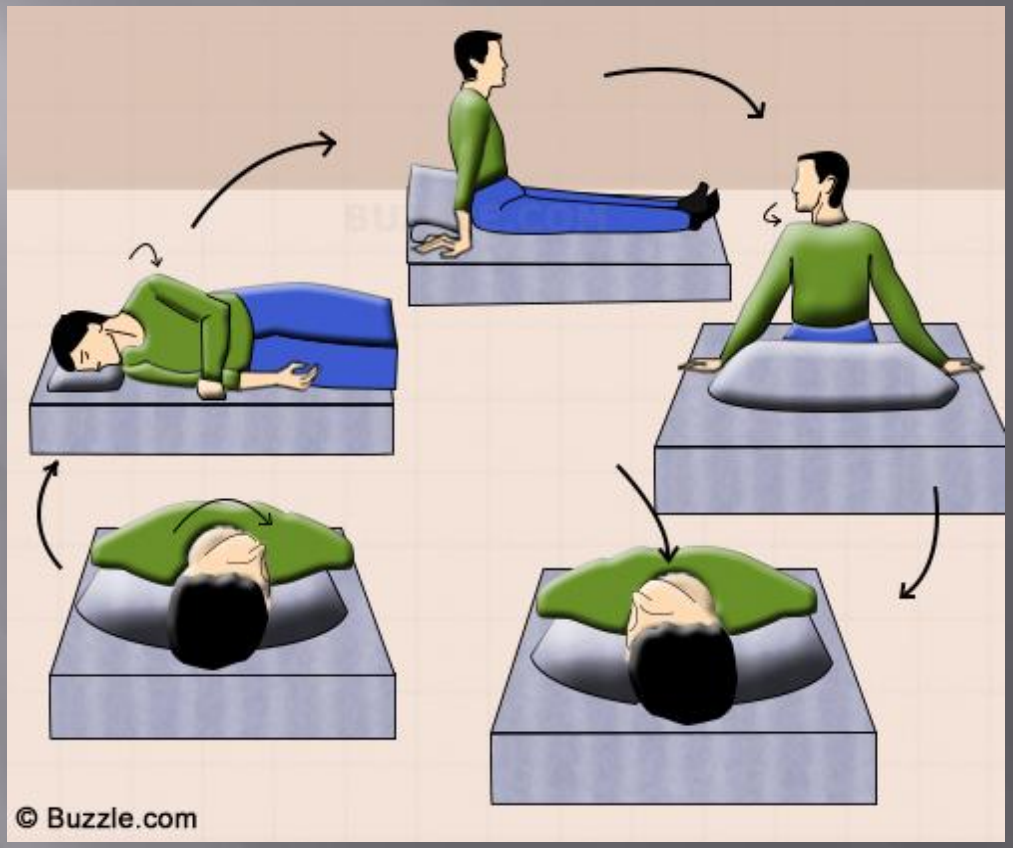
Utricle

Saccule

Otoconia

Displaced
Otoconia

Epley maneuver



The maneuver moves the particles (otoconia) from areas in the semicirculat canal, which cause symptoms (such as vertigo), and repositions them into areas where they do not cause these problems.

Bilateral vestibulopathy

Bilateral loss of peripheral vestibular function **= poor quality of life!**
(fortunately not too frequent)

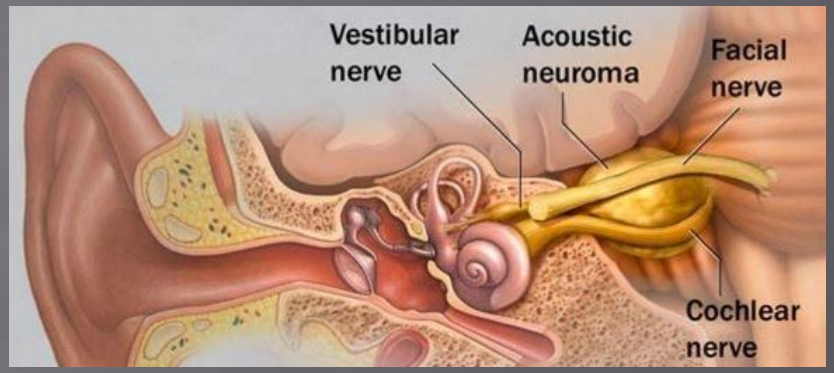
- **Symptoms:**
  - Without movement – no symptom!!! Dizziness while walking!
  - Oscillopsy (can't read and recognize people during walking)
  - Soft ground and darkness makes it worse
  - Hystory: vestibulotoxic drugs, chemotherapy, meningitis, encephalitis, 2 sided Méniére's-disease…

- **Diagnosis:**
  - No nystagmus
  - **Head Impulse Test (HIT) is positive bilaterally** !!!
  - No caloric response

- **Therapy:**
  - Vestibular training, walking sticks
  - Difficult to cure, management (vestibular impantation?)

Prevention!!!

Vestibular schwannoma



Bening tumor, slow growing

Symptoms:

- One sided tinnitus (usually the first symptom)
- Hearing loss on the same side
- Dizziness, dysequilibriometry (rare – due to central compensation)
- Facial nerve involvement (late symptom)

Diagnosis:

- BERA (brainstem evoked response audiometry)– retrocochlear laesion

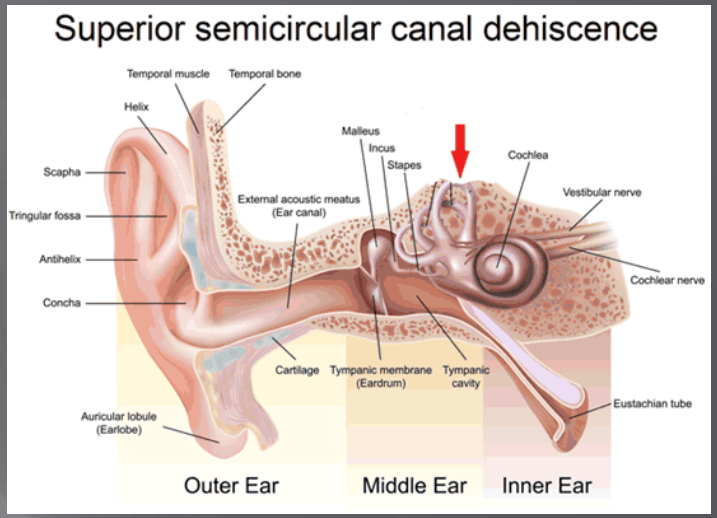
- MRI

Therapy:

- Surgery
- Gamma knife (stereotaxic irradiation)
- Wait and see (MRI, 6 months)

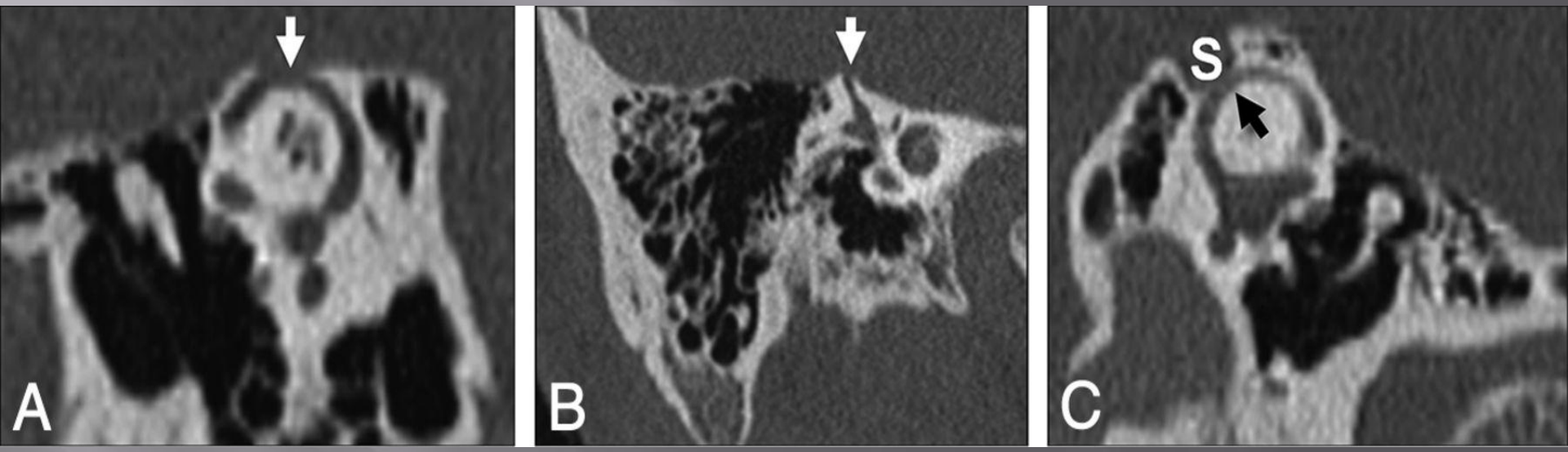
SSCD (superior semicircular canal dehiscence, 3.window syndrome)

Dehiscence of the bony canal
(= third window on the labyrinth)
(Rare)

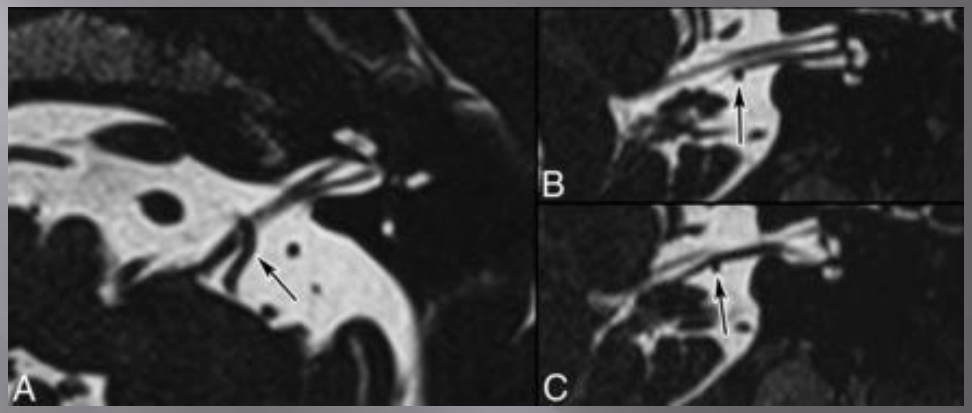


- Symptoms:
  - - **Conductive hearing loss** (air-bone gap)
  - - Vertigo attacks **provoked by pressure / loud noise**, lasting for few minutes (caughing, sneezing, Valsalva)
  - **Positional vertigo**
  - **Autophony** (eg.: hear the moving of their eyes)
- Diagnosis:
  - **Audiogram (air-bone gap) + VEMP (vestibular evoked myogenic potencials)**, HR CT, Hennebert sign = positive fistule test
- Therapy:
  - surgery (?)

SSCD (superior semicircular canal dehiscence, 3.window syndrome)



Neurovascular compression (vestibular paroxysmia)



Causes: AICA - artery compresses the VIII. nerve, demyelinisation

Dg: MRI + patient history

Symptoms:

Attacks of

- - vertigo for seconds or minutes, provoked by head movement
- - hearing loss
- - tinnitus

Therapy:

- Carbamazepin (Tegretol) – antiepileptic drug
- surgery

Vestibular migrain

- ~ CHAMELEON

- HIT can be positive
- Spontaneus nystagmus can be presented
- Central nystagmus can be
- Hearing loss can be
- 20%: endolymphatic hydrops (combined with Ménière-disease)

Therapy = migrain therapy (prophylactic, painkiller)

Criteria:

- - At least 5 vertigo attack, **5 min.- 72 hours**.
- **- Migrain in the patient's history** (with or without aura)
- **- 1 or 2 migrain feature** at more than 50% of vertigo attacks
  - - Headache with at least 2 feature:
    - One sided, pulsatile, severe, physical activity makes it worse
  - - Photophoby/phonophoby
  - - Visual aura

Functional vertigo

History:

- No complaints on the morning
- Physical activity makes it better
- Fear of supermarkets and crowd, agoraphoby

Physical examination:

- No positive findings (MRI neg. - therapeutic)

Phsychotherapy

What makes them feel dizzy???

Patient history!

Most important questions:

- **attacks** or not?
- **between the attacks** do they have complaints?
- **duration** of the attacks
- **lasts more than 24 hours = acut vestibular syndroma!!!**
- **accompanying** symptoms
- **provoking** factors
- **medial history: infection, head or neck injury, drug intake, meningitis / encephalitis** (ototoxic drugs, e.g. :antibiotics, chemotherapy – *bilateral vestibulopathy*)
- **former neuronitis /M. Ménière/migrain** – *BPPV more often occurs*
- **accompanying diseases**

Duration of vertigo

Time	Peripheral	Central
Seconds	BPPV	VB-TIA, aura of epilepsy
Minutes	perilymph fistula	VB-TIA, aura of migraine
(Half) hours	Meniére disease	basilar migraine
Days	vestibular neuronitis labyrinthitis	VB stroke
Weeks, Month	acustic neurinoma, drug toxicity	multiple sclerosis cerebellar degenerations

AVS (acut vestibular syndrome)

Vertigo, >24 hours + nausea/vomiting + intolerance to head movements + NYSTAGMUS (central? periferal?)

- **Vestibular neuronitis (labyrinthitis)**
- **Multiple sclerosis!**
- **Stroke / haemorrhagia in the cerebellum / brainstem!!!**

25% central origin

50% of them have no focal neurological signs !!!!

Deadly D's:

- dysphonia
- dysphagia
- dysarthria
- diplopia
- dysaesthesia
- dysmetria

AVS (acut vestibular syndrome)

CT scan – **X**
MRI – 1.day - **X**

H.I.N.T.S. plus protocol – first 1-2. day: higher sensitivity for central disorders, than MRI.

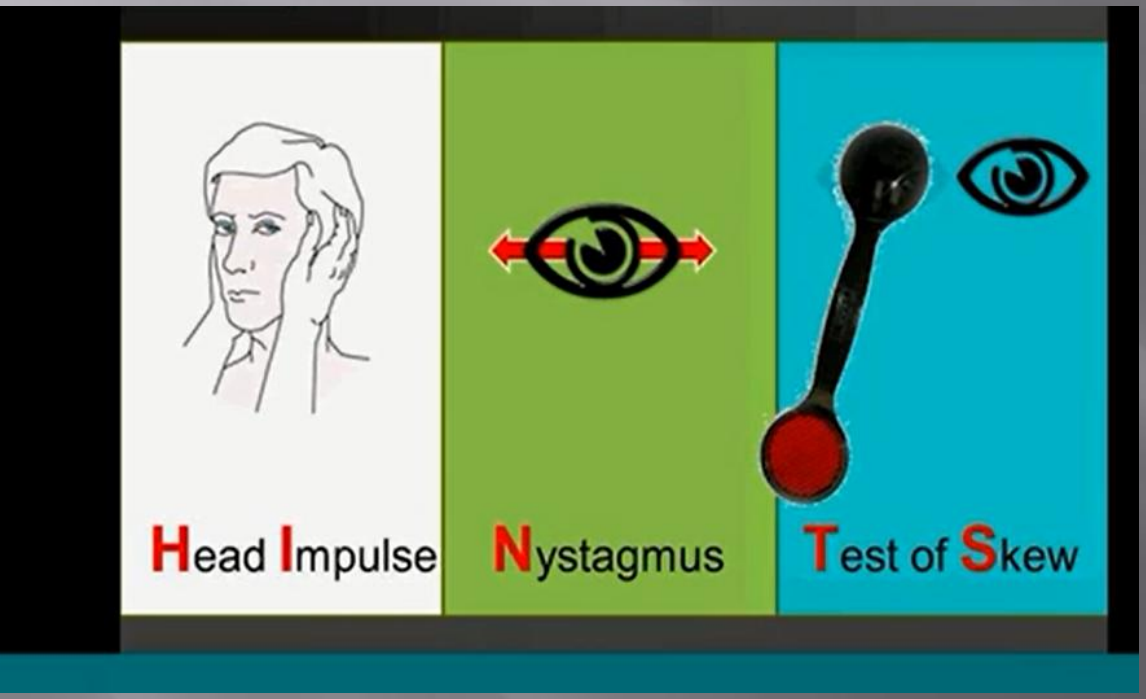
H.I.N.T.S.: Head **I**mpulse test, direction changing **N**ystagmus, **T**est of **S**kew deviation.
plus: sudden hearing loss - *AICA-infarct* (anterior inferior cerebellar artery) /

So if the dizzy patient has:

- negative Head Impulse Test on both side, and/or
- direction changing nystagmus (central nystagmus), and/or
- vertical skew-deviation, and/or
- sudden hearing loss,

probably has central disorder!!!

H.I.N.T.S. +



Sudden hearing loss

Physical examination
(if it's possible, during attack)

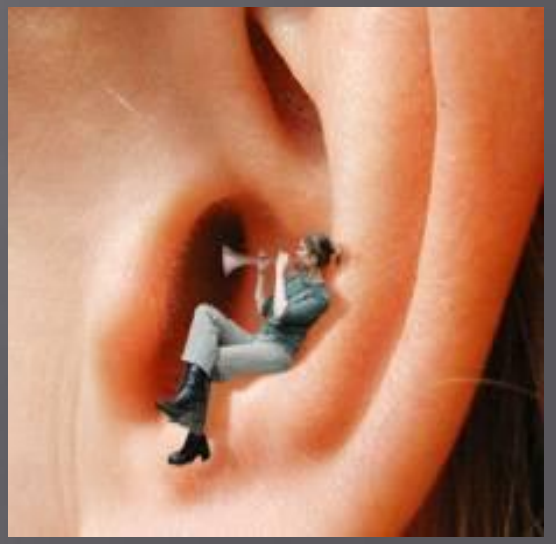
1. **Eardrum** (usually negative!!!)
2. **Spontanous nystagmus - periferal nystagmus**
3. **Head shaking** test
4. **Head Impulse Test** (**HIT**, Halmagyi's test) usually positive in periferal lesions
5. **Skew-deviation** – no vertical skew-deviation
6. **Vestibulo-spinal reflexes** (*Romberg-test, walking blind, Unterberger test, Bárány-test*) toward the affected side
7. **Cranial nerves**
8. **Positional examinations (BPPV / central positional nystagmus)**

9. **Hearing test - with whisper** (if they complain hearing loss)
10. **Tuning fork** (Weber, Rinné) – conductive or sensorineural hearing loss?

Instrumental examinations

- **VNG (Videonystagmography)** – spontaneous nystagmus, caloric examination, positional nystagmus…
- **ENG (Electronystagmography)** – spontaneous nystagmus, caloric examination
- **vHIT (video head impulse test) googles - 6 semicircular canals individually!!!**
- **VEMP (Vestibular evoked myogen potencial) – utriculus, sacculus!!!**
- **ECoG (Electrocochleography)** (Ménière's disease– endolymhatic hydrops)
- **Subjective audiometry**
- **Objective audiometry - BERA**

Tinnitus



Definition

- Tinnitus - a non-auditory, internal sound (ringing, buzzing, hissing, chirping, whistling, or other sounds). - Perception of sound within the human ear in the absence of corresponding sound.

The noise is usually subjective - only the patient can hear it.

- As tinnitus is subjective, it is impossible to measure it using objective tests, only by comparison with noise of known frequency and intensity (=tinnitometry).

- Very common in adults, at least 15 % have or had in their lifetime.
- Most people with chronic tinnitus adjust to the ringing over time, but 1 in 5 will find it disturbing or debilitating.
- 1% very severe – lead to suicide
- Can be uni- or bilateral, fluctuating or permanent, subjective or objective.

Subjective tinnitus

Causes:

- Sudden deafness
- Ménière's disease
- Acute or chronic noise trauma, or middle/inner ear infections
- Presbyacusis
- Hereditary inner ear disease
- Vestibular schwannoma (acustic neurinoma)
- Head and neck injury
- Ototoxic drugs
- Otosclerosis
- TMJ (temporomandibular joint) diseases
- Metabolic diseases: diabetes mellitus, thyreoid disorders…
- Cardiovascular diseases: hypo- or hypertension
- Anaemia,….
- Unknown

Objective tinnitus:

Tinnitus that sounds like a heartbeat may be more serious.

Causes of objective tinnitus:

- Stenosis of the arteries on the neck
- Vascular tumors (haemangioma, glomus tumor)
- Malformations of the heart, artificial valves
- Myoclonus of the middle ear's or soft palate’s muscles
- Open Eustachian-tube
- Spontaneous otoacustic emission (outer hair cell product)

Etiology of the subjective tinnitus

THEORIES:

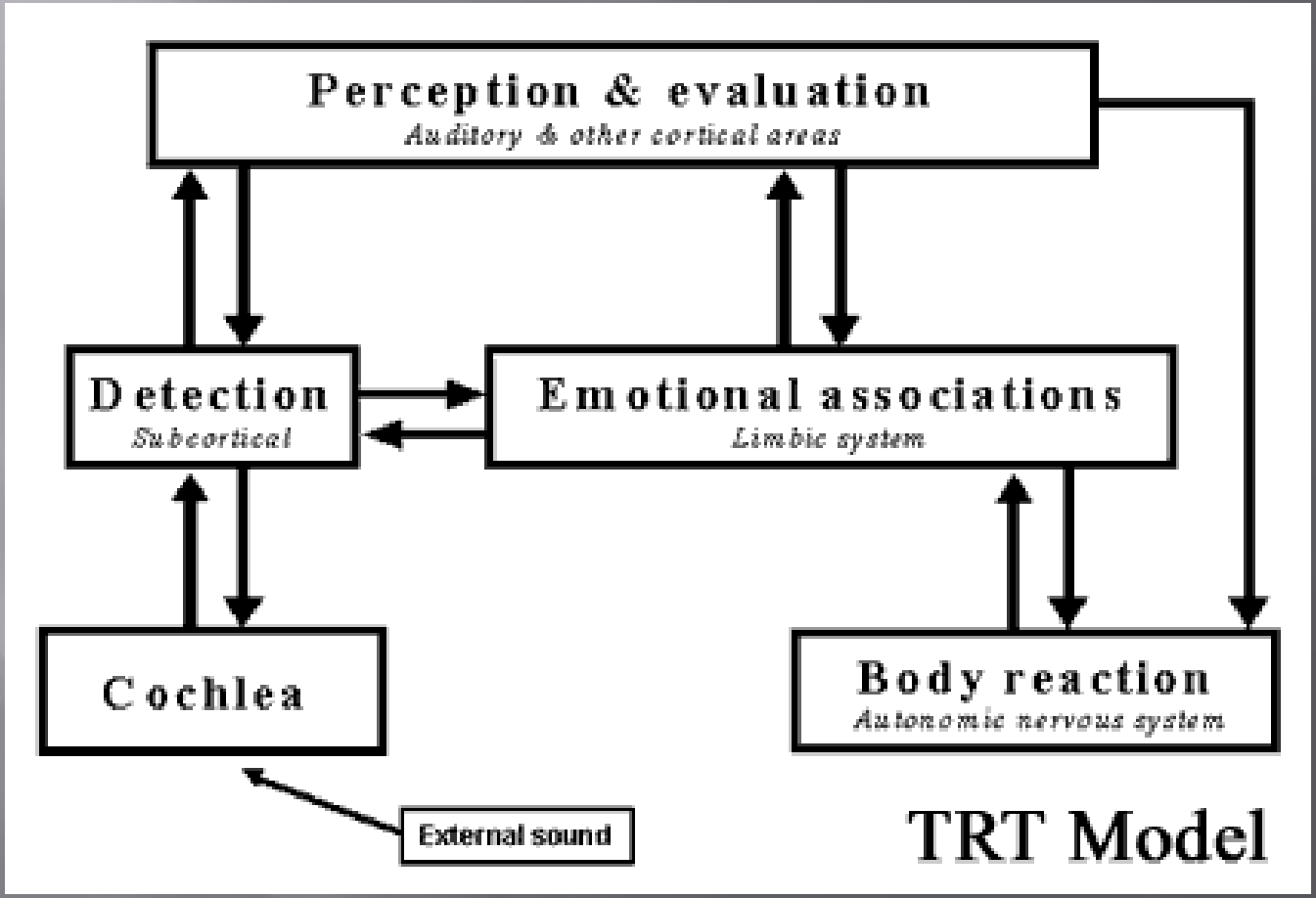
- -spontaneous activity of the cochlear nerve changes because of an inner ear damage (Prolonged exposure to loud sounds, single exposure to a sudden extremely loud noise)
- -endolymph or/and perilymph changes

- **Jastreboff – neurophysiologic model:**

  -Subcortex filtrates many incoming information-protecting software: congenital, learned.

  -Mostly the cause of the tinnitus – inner ear damage – software damage

  -After some time, (months, half year) it „goes" into the brain, and „stuck"!


Perception & evaluation
Auditory & other cortical areas
Detection
Subcortical
Emotional associations
Limbic system
Cochlea
Body reaction
Autonomic nervous system
External sound
TRT Model

Factors –makes tinnitus worse

- caffeine
- alcohol
- nicotine
- sodium
- stress
- noise injury/pollution
- sleeping problems

Tinnitus can cause:

- sleeping problems
- hearing loss
- hyperacusis: decreased sound tolerance
- impaired speech understanding
- frustration, stress, depression
- lack of concentration
- dependence on medicine
- pain/headache

Tyler and Baker, 1983

Examinations

- Examination of the ears with microscope
- Subjective audiometry (pure tone audiometry, speech audometry, tinnitometry)
- Objective tests: tympanometry, stapedial reflex, TOAE (Transient OtoAcustic Emission), DPOAE (Distorsion Product OtoAcustic Emission), ABR (Auditory Brainstem Response)
- MRI (mainly in single sided cases, to rule out vestibular schwannoma)

Therapy

Still we do not have a mechanism-based method to provide a cure for tinnitus!!!

Most people learn to live with tinnitus, but help is available for those who find this difficult.

Prevention!!! - avoid : loud noise, medications (200 drugs) , ototoxic drugs, etc.

Tinnitus re-training therapy:

Aimed primarily at habituating tinnitus-evoked reactions of the brain and body, and secondarily, at habituation of tinnitus perception.

The key is to break the link between tinnitus perception and the negative or fearful emotions associated with it.

Habituation occurs when the patient considers the tinnitus to be an irrelevant stimulus, just like many other environmental sounds (low level traffic noise,...)

So anything that can make the tinnitus sound "irrelevant" will be helpful.

Ignoring it rather than focusing on it can provide relief.

Tinnitus Retraining Therapy

Two key components:

Counseling

So patients will be able to control the negative reactions through education (i.e., the more you know, the more you control)

Sound therapy

Changes the tinnitus-to-background ratio (but not nature of tinnitus) to facilitate habituation:

- **tinnitus masker / noiser** (Uses external noise to mask the perception of tinnitus. Low-level music, white noise, or specialized ear maskers can help.)
- **hearing aid** (they amplify environmental sounds and redirect attention to those noises instead of the tinnitus)

+ *Psychotherapy!!!* *(relaxing techniques)*

Listening to loud music on personal devices, can trigger tinnitus!!!


THANK
YOU
FOR
YOUR
ATTENTION…