Vestibular Bedside Examination

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Bedside exam of the vestibular system

- Acute vertigo or dizziness is often a challenge in medical practice
- Careful clinical examination (and patient's history), often allows us to get the diagnosis and to formulate the treatment.
- Bedside examination is based on solid anatomical and physiological principles
- Common causes of acute vertigo are well known

Clinical vestibular examination

- Provides the assessment of static and dynamic vestibular function
- ocular motor examination
- provocative maneuvers
- vestibulo-spinal testing
- Depending on the patient's symptoms: sometimes necessary a general physical and neurological examination with assessment of the remaining cranial nerves

Bedside

- Purpose of this course:

- try to specify the clinical meaning and the diagnostic value of the bedside examination with the only help of Frenzel glasses and few “pocket tools”
- to suggest when further testing are necessary (beside the clinical examination)

Bedside examination

- **Static evaluation**
- Spontaneous nystagmus
- OTR
- Saccades
- Pursuit
- **Dynamic evaluation**
- HIT
- HST
- **Miscellanea**
- Vibration
- Hyperventilation
- Valsalva
- Tullio
- **Posture**

Spontaneous Nystagmus

- Most important clinical finding in a patient with acute vertigo
- Hallmark of a static imbalance of tonic activity at the level of the vestibulo-ocular reflexes
- Peripheral SN
- Central SN

Peripheral spontaneous nystagmus

- Unidirectional
- Horizontal (minor torsional component)
- Follows Alexander law: increases in intensity when looking in the direction of quick phase
- Typically inhibited by visual fixation

Frenzel glasses

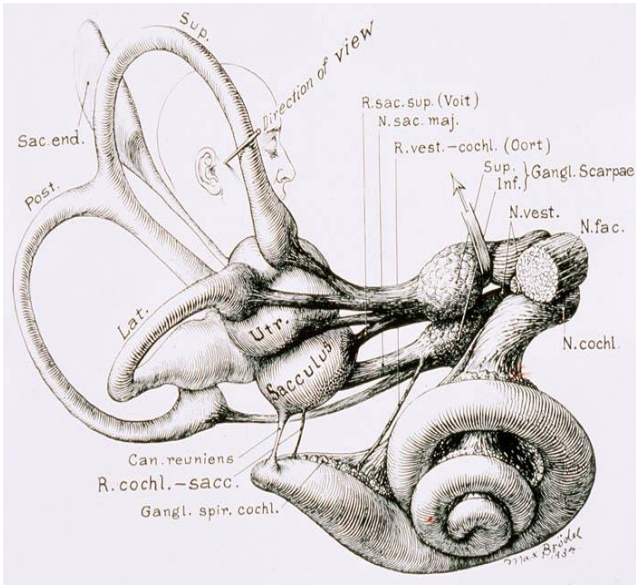
- efficient tool to assess the influence of visual fixation on nystagmus and eye movements

Frenzel goggles, ebay

Typical peripheral nystagmus

- Hallmark of an acute unilateral peripheral disorder
- Vestibular neuritis
- Menière's disease in acute stage
- Lateral canal BPPV (pseudospontaneous)

- Can also be seen in central vertigo
- cerebellar infarction
- vestibular migraine
- Peripheral origin needs to be confirmed
- Head Impulse test (HIT)
- and/or caloric test



Central spontaneous nystagmus

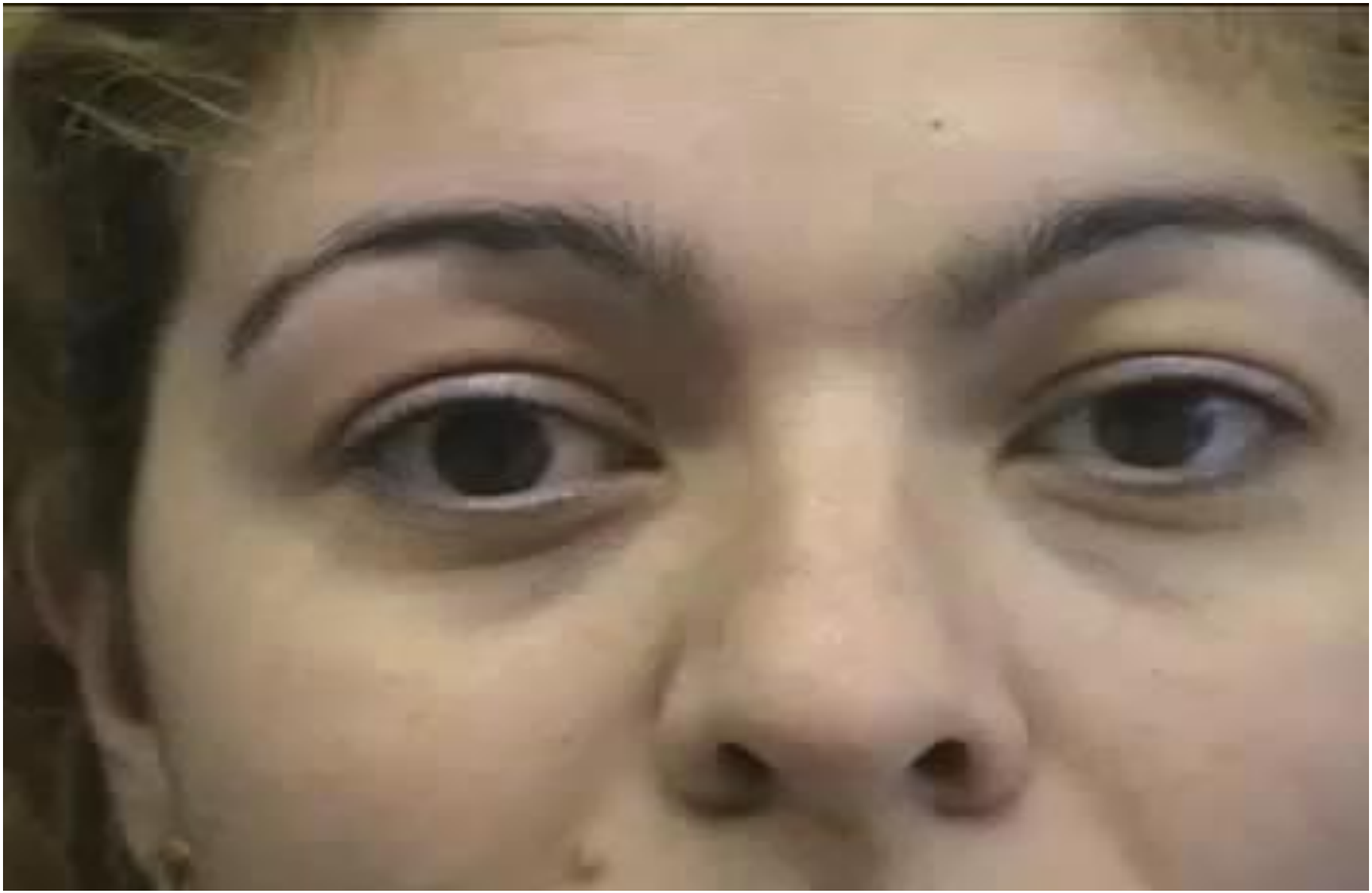
- Quite often easily recognizable
- Unequivocal for the diagnosis of central lesion
- Well detectable with direct inspection of the eyes (poorly affected by fixation)

- pure torsional
- vertical (downbeat/upbeat)
- direction changing nystagmus (gaze evoked, rebound, periodic alternating, dissociated, centripetal)

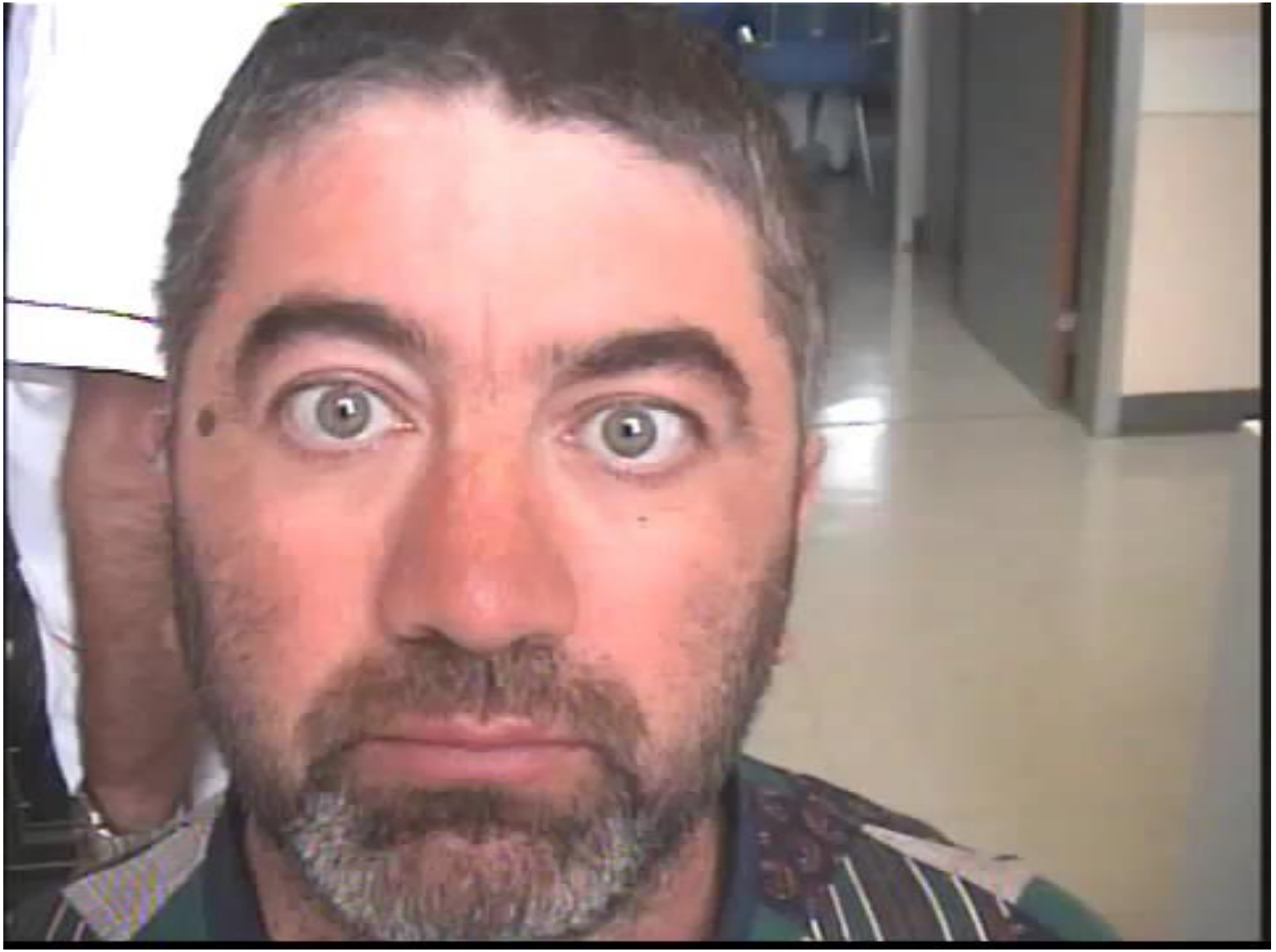
Central spontaneous nystagmus

- Certain kinds of central spontaneous nystagmus: diagnostic value to determine the site of the lesion
- Gaze & rebound: cerebellum (flocculus/paraflocculus)
- Downbeat: cerebellum (nodulus/uvula; tonsil)
- Upbeat nystagmus: midline brainstem lesions
- PAN: nodulus
- Brun's nystagmus: cerebellopontine angle tumors: (low frequency/ large amplitude nystagmus on looking towards the lesion and high frequency/small amplitude looking away from the lesion).

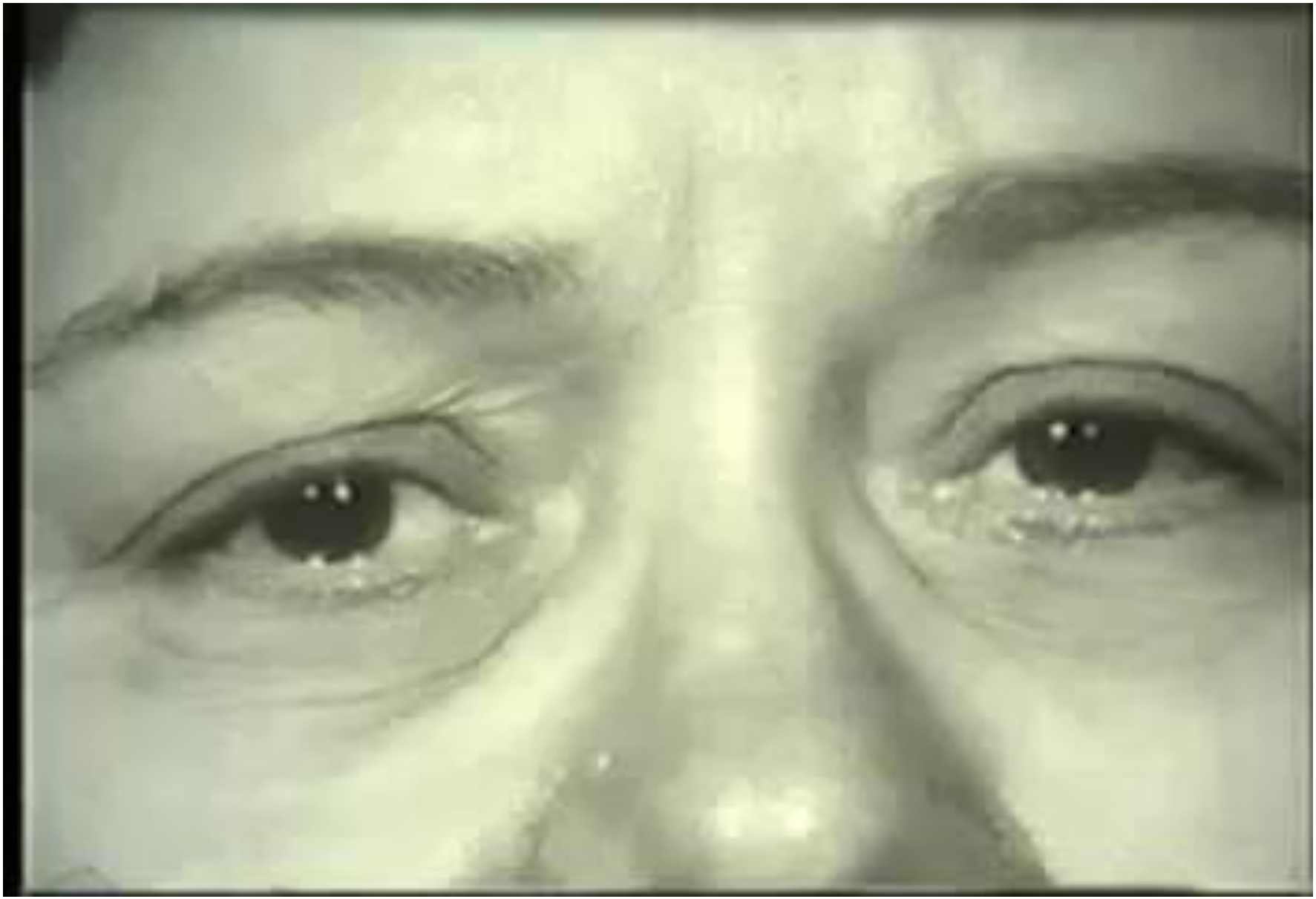
Gaze evoked nystagmus, direction changing, with rebound: abnormal function of the cerebellar flocculus or paraflocculus



Upbeat nystagmus reflects midline brainstem lesions

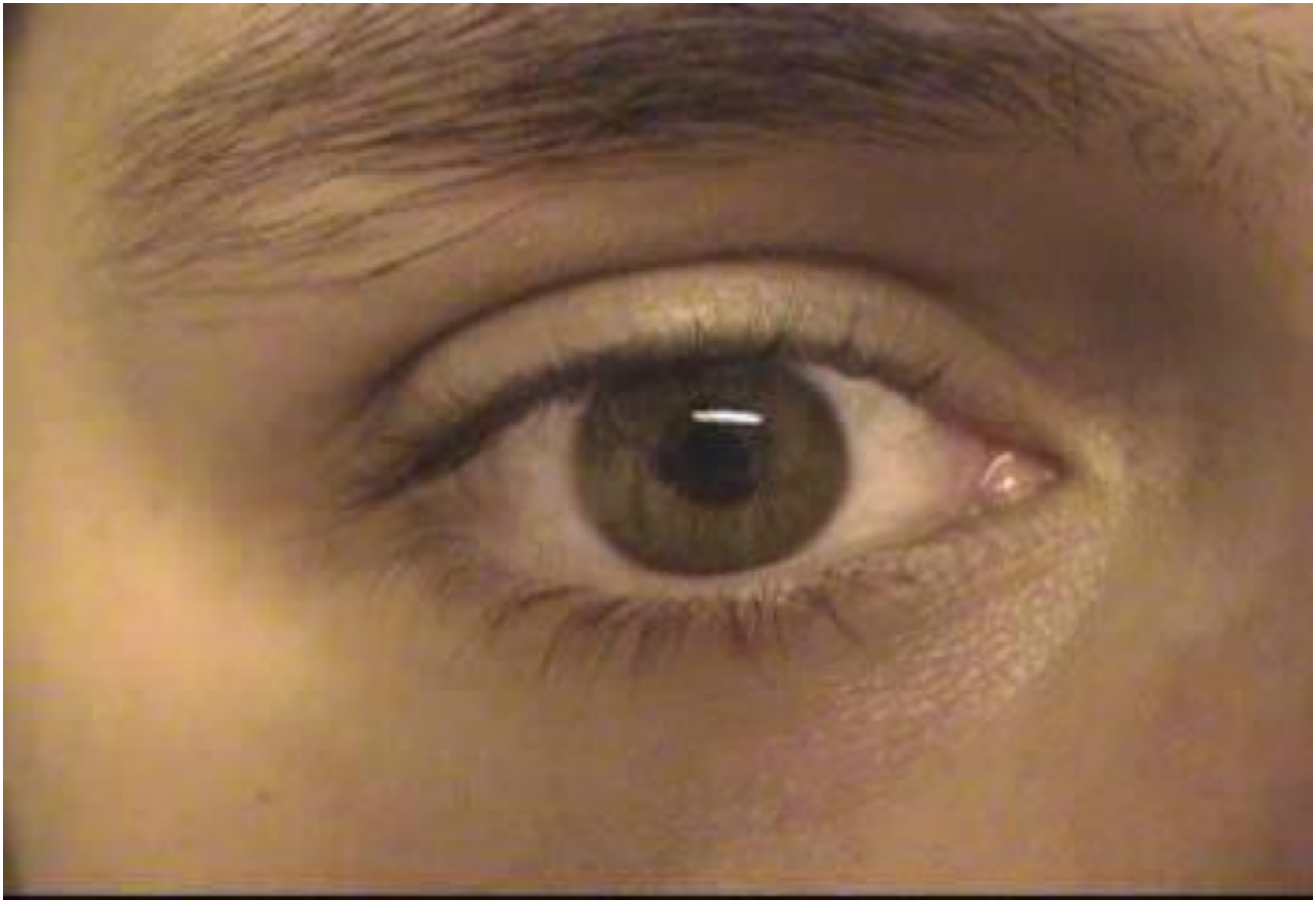


PAN: nodulus



Unilateral INO: FLM

Brun's nystagmus: (low frequency/ large amplitude nystagmus on looking towards the lesion and high frequency/small amplitude looking away from the lesion).



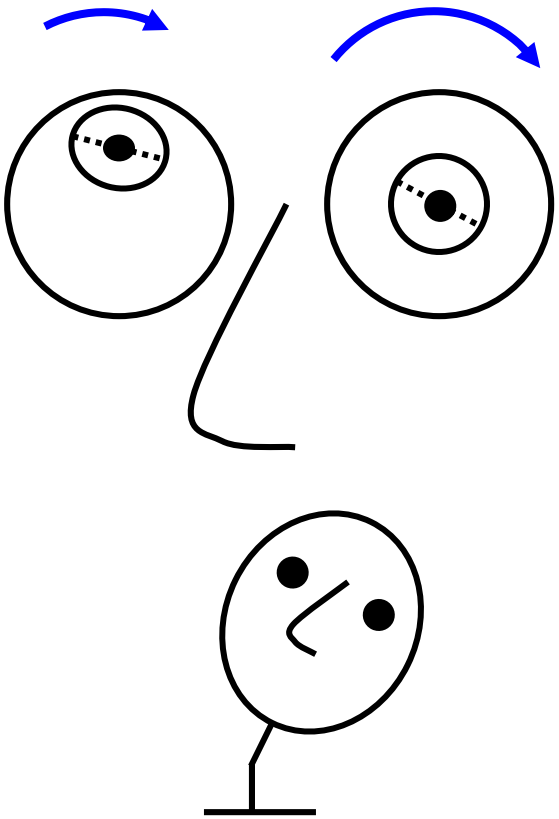
Central nystagmus

- Detection of these nystagmus forms: further laboratory vestibular examinations are unnecessary
- Imaging procedures are needed to identify the lesional site
- Gaze evoked direction changing nystagmus and dizziness: sometimes necessary to investigate cerebellar toxicity (alcohol anticonvulsivant, lithium)

Carbamazepine intoxication

Ocular tilt reaction

Skew Deviation
(vertical eye misalignment)

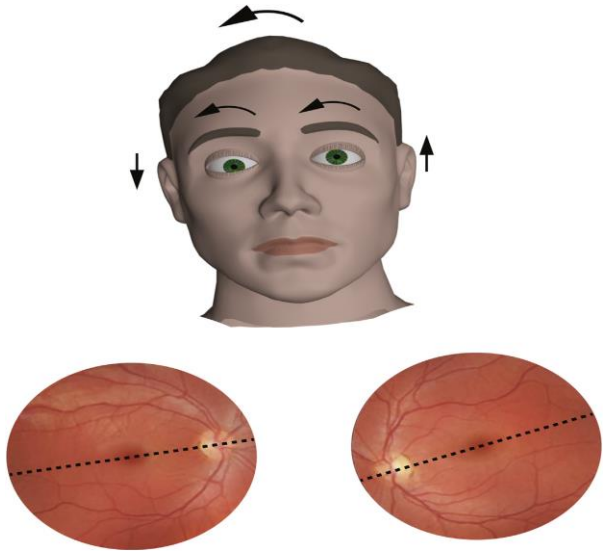


Counterrolling (torsional) rotation of the eyes (dissociated)

head roll tilt

OTR

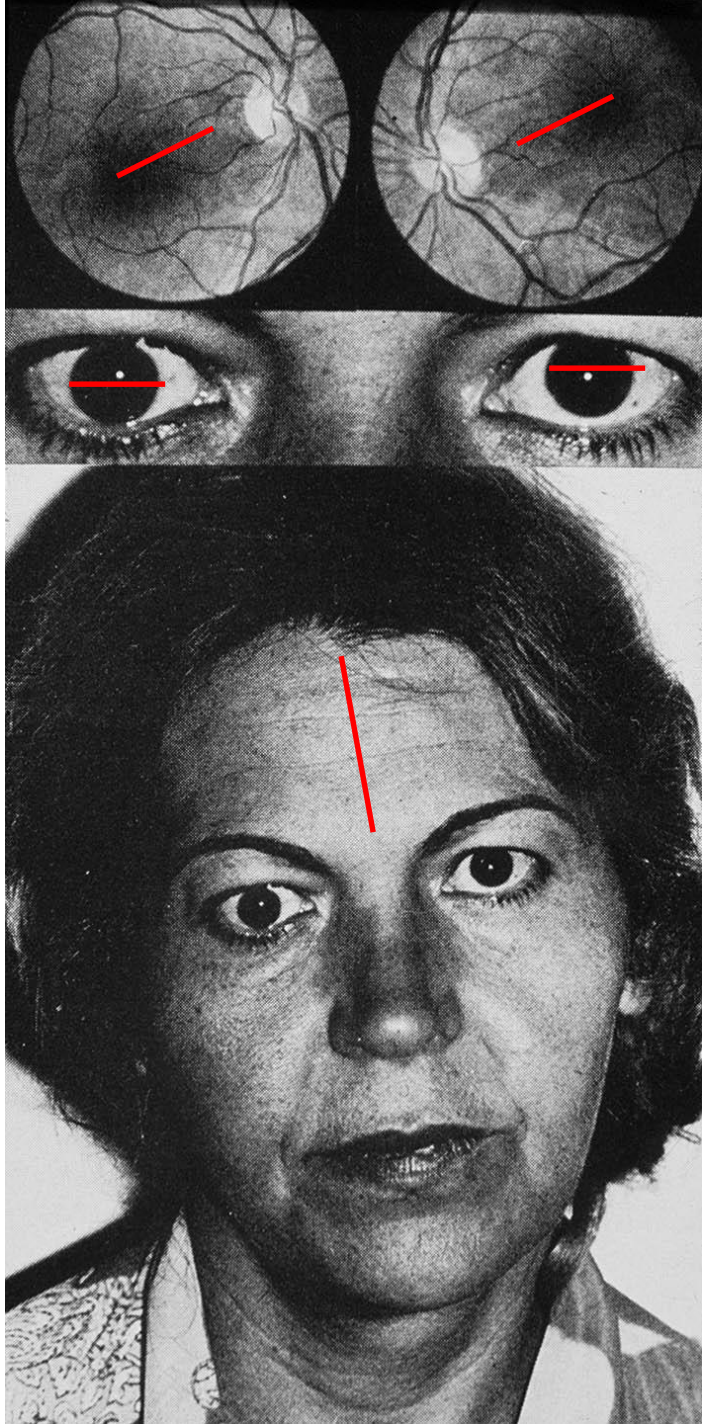
- **Acute tone imbalance of static utricular righting reflexes: analogous to spontaneous nystagmus from a semicircular canal tone imbalance (Michael Halmagyi, Ann Neurol, 1979)**
- **Oculocephalic response mainly due to a the tonic otolith-ocular reflex imbalance (Thomas Brandt, 1994)**



Ocular Counterroll: not detectable at bedside

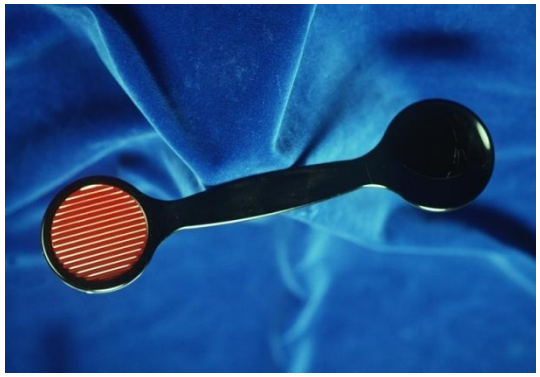
Skew

Head Tilt



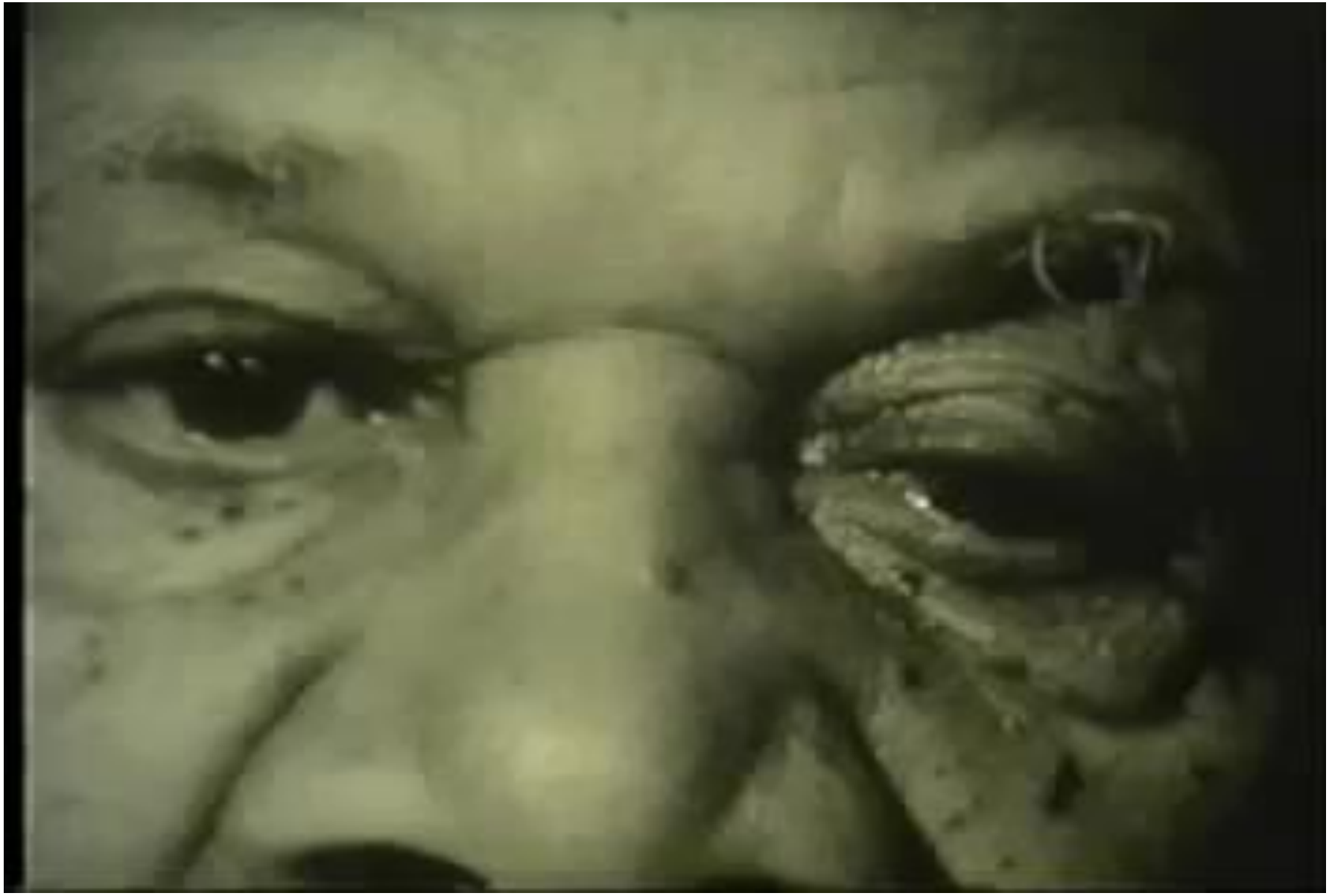
Skew deviation and Ocular Tilt Reaction (OTR)

- Skew Deviation can be manifest or revealed by the alternate cover testing at the bedside
- **Look for a refixation movement when the cover is removed to determine whether the eye was higher or lower.**



Maddox Rod

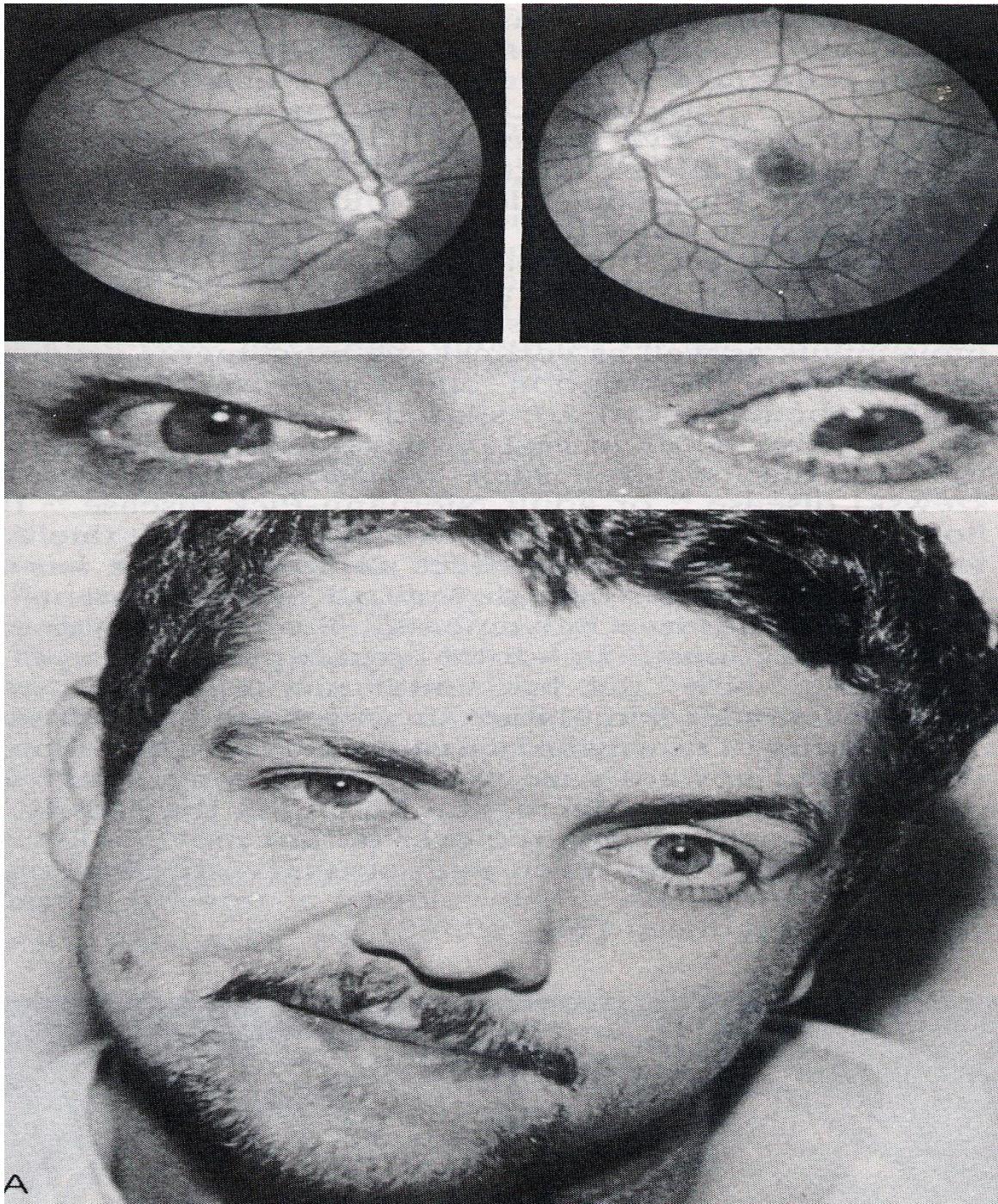
alternate cover testing



SKEW: left eye down

HEAD TILT to the left

Right midbrain lesions
OR
diseases from labyrinth to vestibular nuclei on the left side



Skew deviation and Ocular Tilt Reaction (OTR)

- Head tilt to the affected side and SD (ipsilateral hypotropia): possible in any peripheral vestibular lesion (vestibular neuritis)
- These signs are sometimes skipped because attention is placed on spontaneous nystagmus.
- Head tilt and manifest SD disappear in a few days in peripheral patients
- Large and persistent SD, often associated with diplopia, point to a disorder of the central vestibular system
- Differential diagnosis: superior oblique palsy (the vertical misalignment of the eyes is non-comitant, i.e. it changes in different directions of gaze)

Skew Deviation

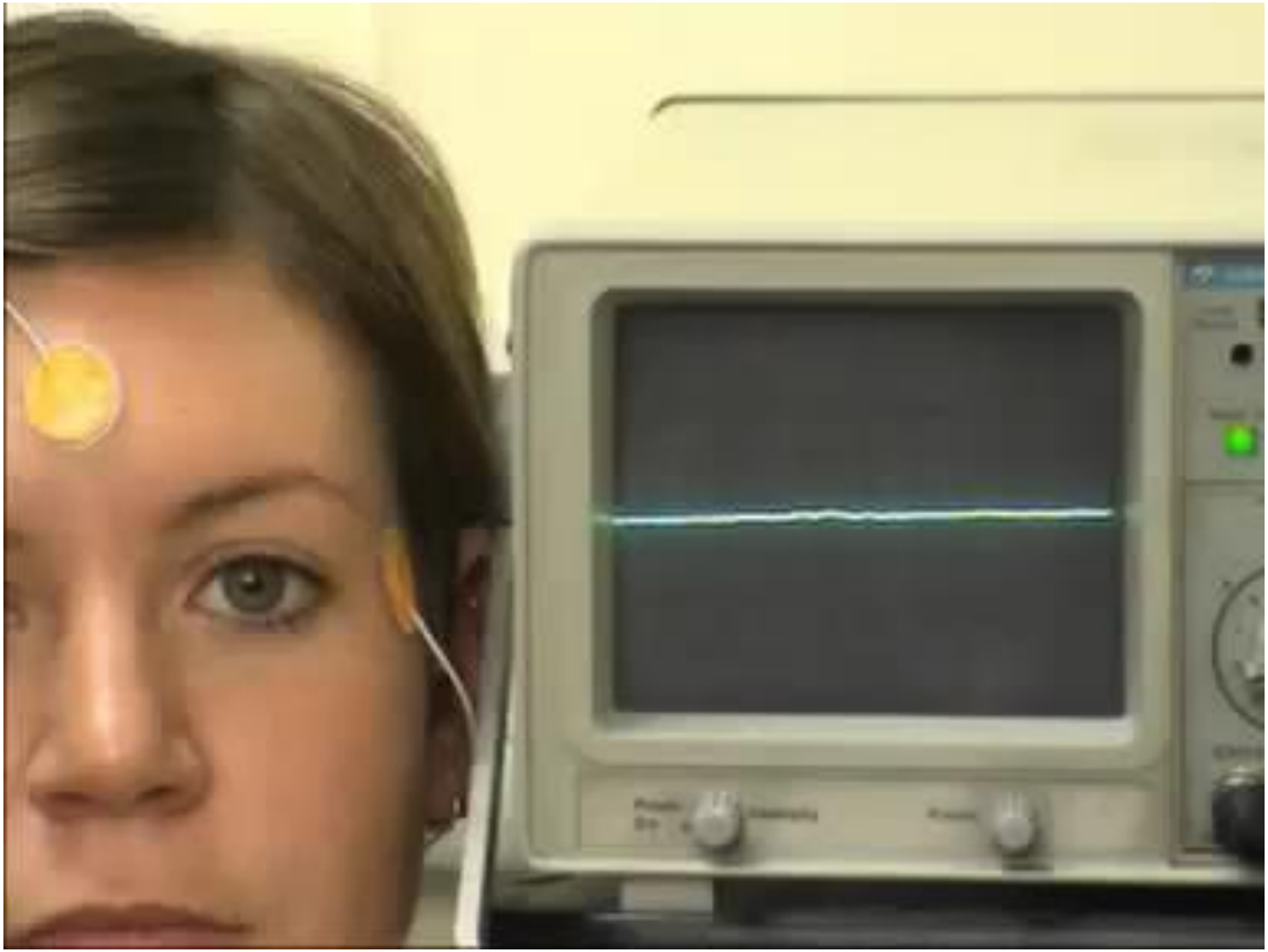
- In summary, the finding of SD in patients with acute vertigo is indicative of an imbalance of the otolithic pathways. In the algorithm “HINTS” it is considered one of the signs that suggests a central vestibular lesion
- This topic deserves some criticism since SD and head tilt are sometimes observed in the first days of vestibular neuritis

Figure 1: The golden retriever, Tessa, with acute left labyrinthine failure

Saccades at the bedside

- Examination of visually induced saccades can provide important topographical information.
- Instruct the patient to fix alternately upon two targets (tip of a pencil-nose of the examiner) located at 50 cm away from the patient
- Examine in each direction in the horizontal and vertical planes

Visually induced saccades



Saccades at the bedside

- Two main abnormalities to be searched, both indicative of central disorders:

- Are they accurate? (wrong size)
- Their velocity is normal? Or too slow?

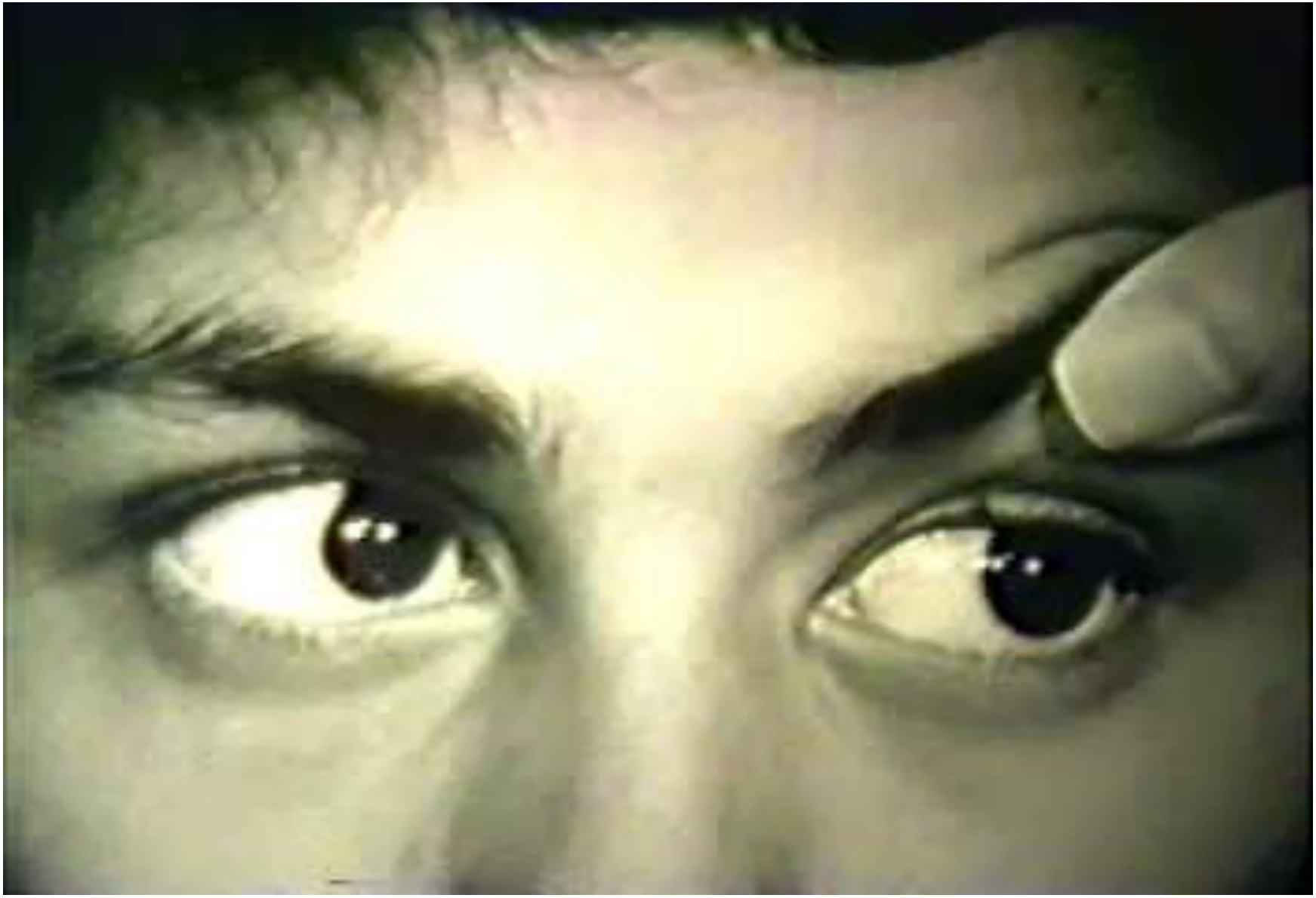
Vermis hypometria (multiple little saccades to reach the target)

Saccadic hypermetria (the eye overcomes and then returns to the target)

Saccadic Dysmetria

- Hypometry : various brainstem and cerebellar diseases
- Hypermetria: cerebellar diseases

Slow saccades



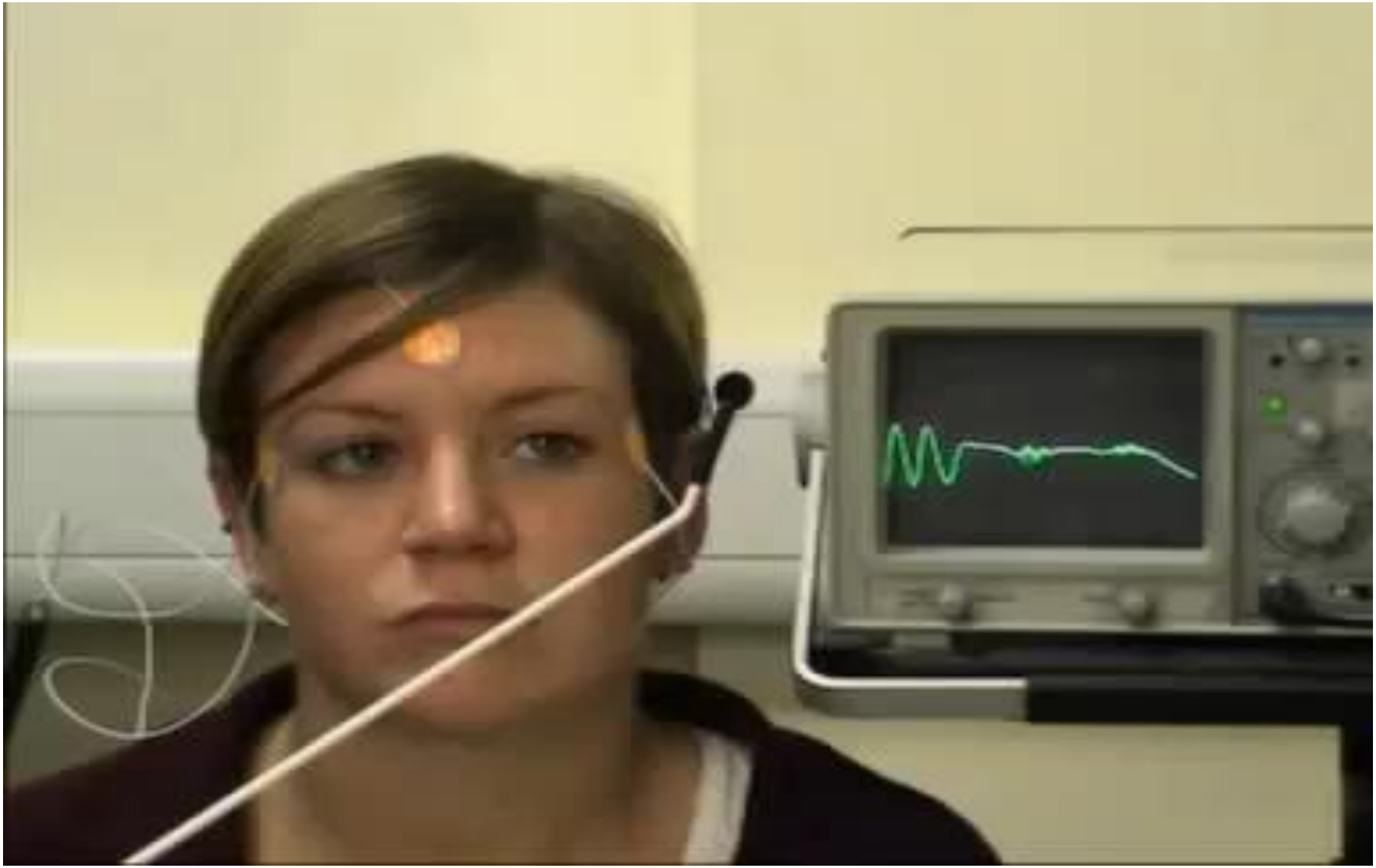
Slow saccades

- Lesions in the brainstem
- horizontally: pons (parapontine reticular formation-PPRF)
- Vertically: midbrain (rostral interstitial nucleus of medial longitudinal fasciculus-RIMLF)

Smooth pursuit

- Slow tracking movements that maintain images of small moving targets on the fovea
- Ask to follow the slow movement of a pen
- Around 50 cm away from the patient
- Slow movement on the horizontal and vertical planes
- Velocity of movement: no more than 30°/sec;

Smooth pursuit

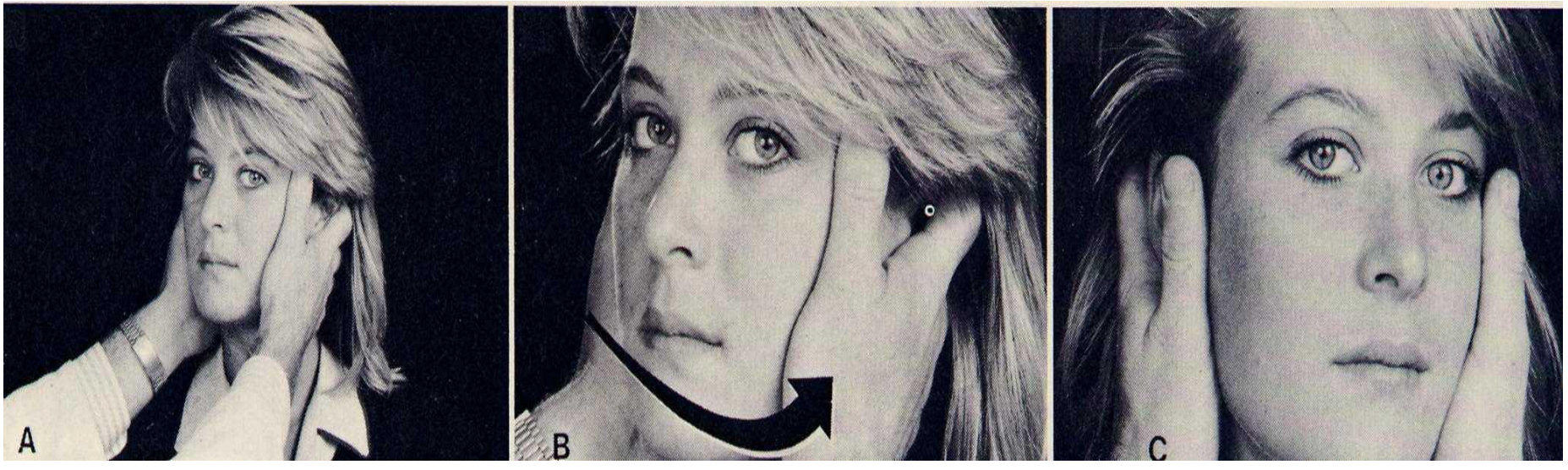


Pursuit

- Inspection of slow eye movements plays a secondary role in the evaluation of a patient with acute vertigo or dizziness
- Pursuit abnormalities are not site-or disease-specific
- If abnormal: quite always accompanied by other ocular-motor alterations
- Peripheral diseases have, as a rule, normal pursuit
- Mild impairment can be due to inattention or medications
- Pursuit after the age of 65-70 years is quite always deteriorated, especially in the vertical plane.

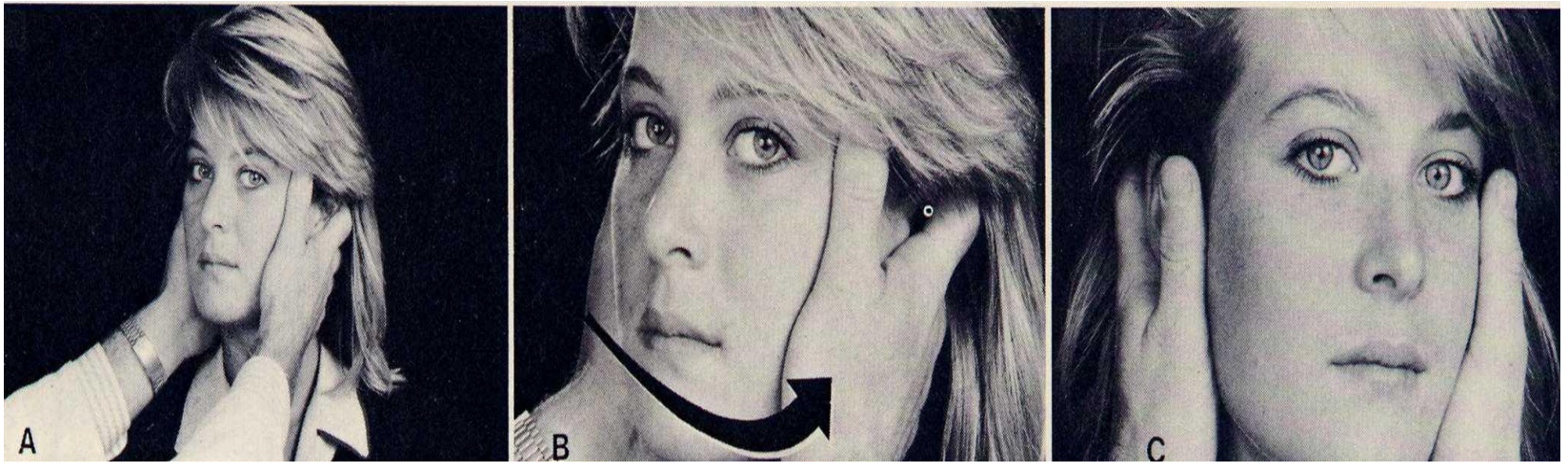
Head Impulse Test†

- Conceived by Halmagy and Curthoys in 1988
- Real revolution in the field of clinical vestibular examination
- Most important method to assess the high-frequency VOR at the bedside



HIT

- Head impulses must be applied in an unpredictable way for time and directions, brief duration, high acceleration, small excursion
- the patient must fixate the examiner's nose while his head is quickly rotated
- Look for catch-up saccades in opposite direction to that of head movement (refixation saccades)
- The corrective saccade is “the bedside signature of a hypofunctioning labyrinth” (Leigh and Zee, 2015)



Left vestibular neuritis

HIT

- HIT towards the affected side: catch-up saccade to the opposite side at the end of the head movement
- Due to the lacking VOR slow phase of the affected canal
- Catch-up saccade (s) generally easy to be detected at the bedside: overt saccades
- Even better in patients with horizontal spontaneous nystagmus.
- Patients with vestibular neuritis observed in the very early stage of the disease: head impulse sign well detectable in about 90% of patients if the vestibular loss is marked (Nuti et al. 2005)

HIT

- The diagnostic value of a positive HIT is very high in patients with spontaneous horizontal nystagmus: allows the diagnosis of peripheral vestibular hypofunction
- Further investigations can be delayed or even omitted
- We can prescribe drugs and physiotherapy, following the patient to verify the expected progressive improvement of signs and symptoms.

HIT

- Over time the adaptation mechanisms make the corrective saccades occur earlier or during the head rotation so that they become harder or impossible to be seen at the bedside *(covert saccades)*
- HIT may be negative or doubtful if
- the vestibular loss is mild
- the saccade is covert or too early
- the lesion is in the central vestibular system.
- Further investigations are needed: caloric test, video head-impulse testing, and eventual MRI
- HIT can also be applied to the vertical canals: the ocular response is often difficult to see at the bedside (the response from the vertical canals is better evaluated by video head-impulse testing)

HINTS

- Patients with “Acute vestibular Syndrome “: important not to miss the diagnosis of stroke in cerebellum or brainstem.
- It has been suggested that a careful bedside vestibular examination is more sensible than MRI in early detection of stroke
- Algorithm **“HINTS”** has been conceived for this purpose:

- Negative **Head Impulse (HI)** sign,
- *or* direction changing gaze evoked **Nystagmus (N)**
- *or* positive **Test** for **Skew** deviation **(TS)**

HINTS

- The presence of any of these signs is considered as a pointer for a possible lesion within brainstem and cerebellar areas receiving projections from the labyrinth (Leigh & Zee, 2015)
- These patients must carefully evaluated and monitored even with early normal MRI (Kattah, 2009)
- Caveat 1: in the first days of vestibular neuritis is quite often detectable a mild skew deviation, which soon disappears
- Caveat 2: Remember an abnormal HIT does not exclude a central (usually AICA) stroke

WHAT ABOUT IMAGING

- HINTS is BETTER than an MRI at detecting a possible stroke in the posterior fossa in the first 48 hrs
- Early negative MRI does not EXCLUDE a stroke in patients with positive HINTs. Such patients have a stroke until proven otherwise. It's necessary to repeat MRI
- **This is even more true for a CT scan. Unless there is headache or a propensity for bleeding, CT scan is USELESS in the patient with AVS.**

DIAGNOSIS OF POSTERIOR FOSSA STROKE IN ACUTE VESTIBULR SYNDROME:

HINTS plus

- Head impulse test NEGATIVE
- Direction-changing, gaze-evoked nystagmus
- Skew deviation

- PLUS

- Unilateral hearing symptoms (another warning of danger for stroke in the territory of anterior inferior cerebellar artery, not only for peripheral labyrinthine disease)
- Inability to walk or to stand unaided

Markers of Central AVS

- Further ‘red flags’ of central vestibular involvement:

- coexistence of throbbing occipital headache
- abnormal pattern of Head Shaking Nystagmus

Head Shaking Test (HST)

- Series of head oscillations for about 10 seconds
- Around 2 cycles per second

- Assess the dynamic balance of the vestibular system and the integrity of **the velocity storage mechanism**.
- Unilateral vestibular hypofunction: asymmetry of vestibular inputs during head rotations
- Asymmetrical accumulation of activity within VSM
- Post HSN reflects discharge of that activity

HST

- Particularly effective to bring out a nystagmus in patients with compensated unilateral vestibular loss
- Valuable information also in patients with acute vertigo or unsteadiness: evident increase in intensity of the spontaneous nystagmus due to a peripheral vestibular loss (reinforces the suspected diagnosis)


segment 12

HST

- Great diagnostic value when it brings out an atypical response
- A cerebellar lesion is probable when:
- HST in the horizontal plane results in a vertical (perverted) nystagmus (Huh 2011, Kim 2005)
- when there is a prompt early reversal of head shaking induced nystagmus
- when it causes the reverse of direction of a pre-existent spontaneous nystagmus (Choi, 2007)
- Central nystagmus patterns are often easily detected without Frenzel glasses

Mastoid Vibration

- Simple, low-price tools, devices
- Mastoid vibration (100 HZ)
- Stimulus to both labyrints, similar to simultaneous caloric test (warm water)

Vibration induced nystagmus

- Unilateral vestibular deficit: only the healthy side is stimulated
- Nystagmus with slow phase toward the affected side (beats toward healthy side)

Compensated right vestibular neuritis

Vibration Test

- Mastoid Vibration: effective method to evoke nystagmus in compensated unilateral vestibular loss.
- Much less important is its usefulness in patients with acute vertigo, both peripheral and central.
- Vibration test increases the intensity of spontaneous nystagmus in vestibular neuritis (not decisive for diagnosis)
- Menière's disease: vibration test can provoke nystagmus towards the affected side

- A labyrinthine fistula must be suspected with strong vibration induced nystagmus

Labyrinthine fistula

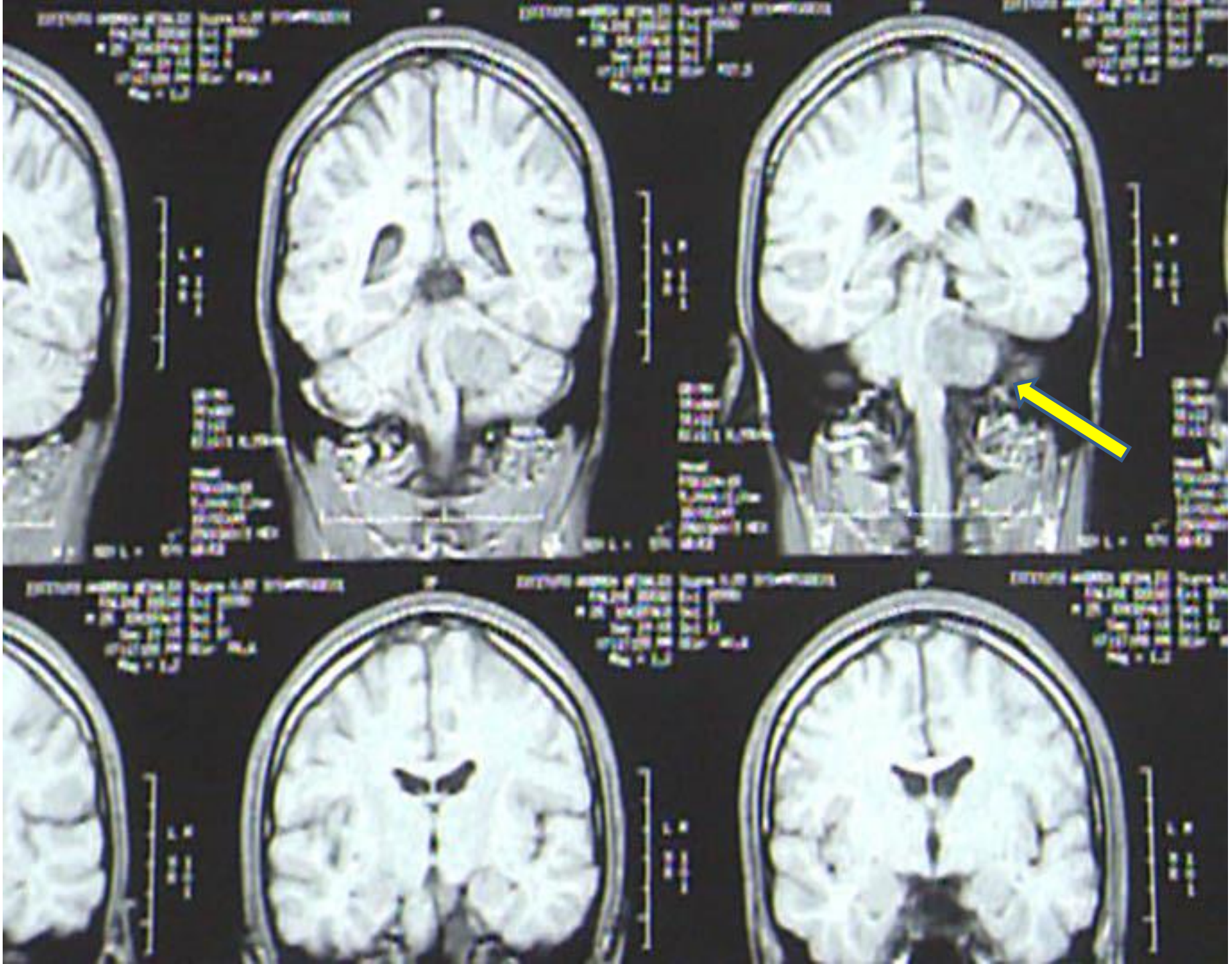
Hyperventilation

- Method: deep hyperventilation for 40 seconds (30 times), taking around one breath per second
- Not necessary in acute vestibular syndrome
- Hyperventilation induced nystagmus: not a marker of a specific disease

Hyperventilation

- Can be useful for diagnosis of:
- VIII° nerve compression (vestibular schwannoma, vascular)
- inflammation of the VIII° nerve
- demyelinating diseases
- perilymphatic fistula
- disorders of the cranio-cervical junction
- cerebellar degenerations

Left Vestibular Shwannoma



Hyperventilation induced nystagmus

- Transitory improvement of nerve conduction in marginally functional, demyelinated, fibers by virtue of its effects upon serum pH and free calcium concentration
- Recovery nystagmus beating toward the affected side

Hyperventilation and anxiety

- May be useful to induce dizziness in patients with anxiety and phobic disorders, without producing nystagmus:

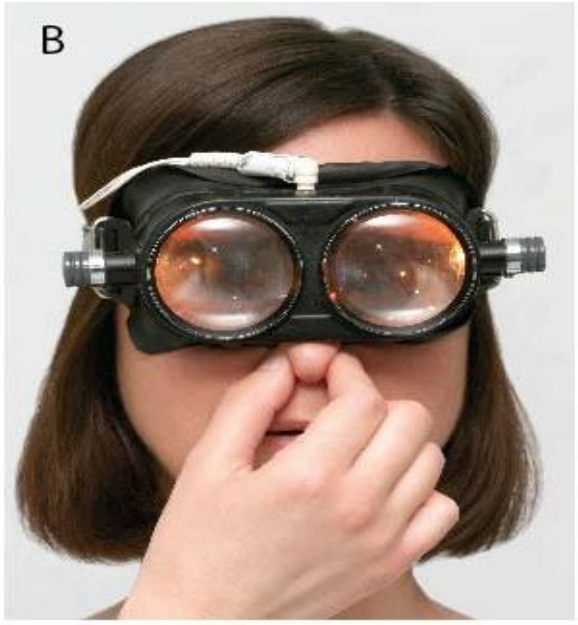
- No typical vertigo
- Dizziness, paresthesias, numbness, lightness

- Caveat: patients with epileptic seizures

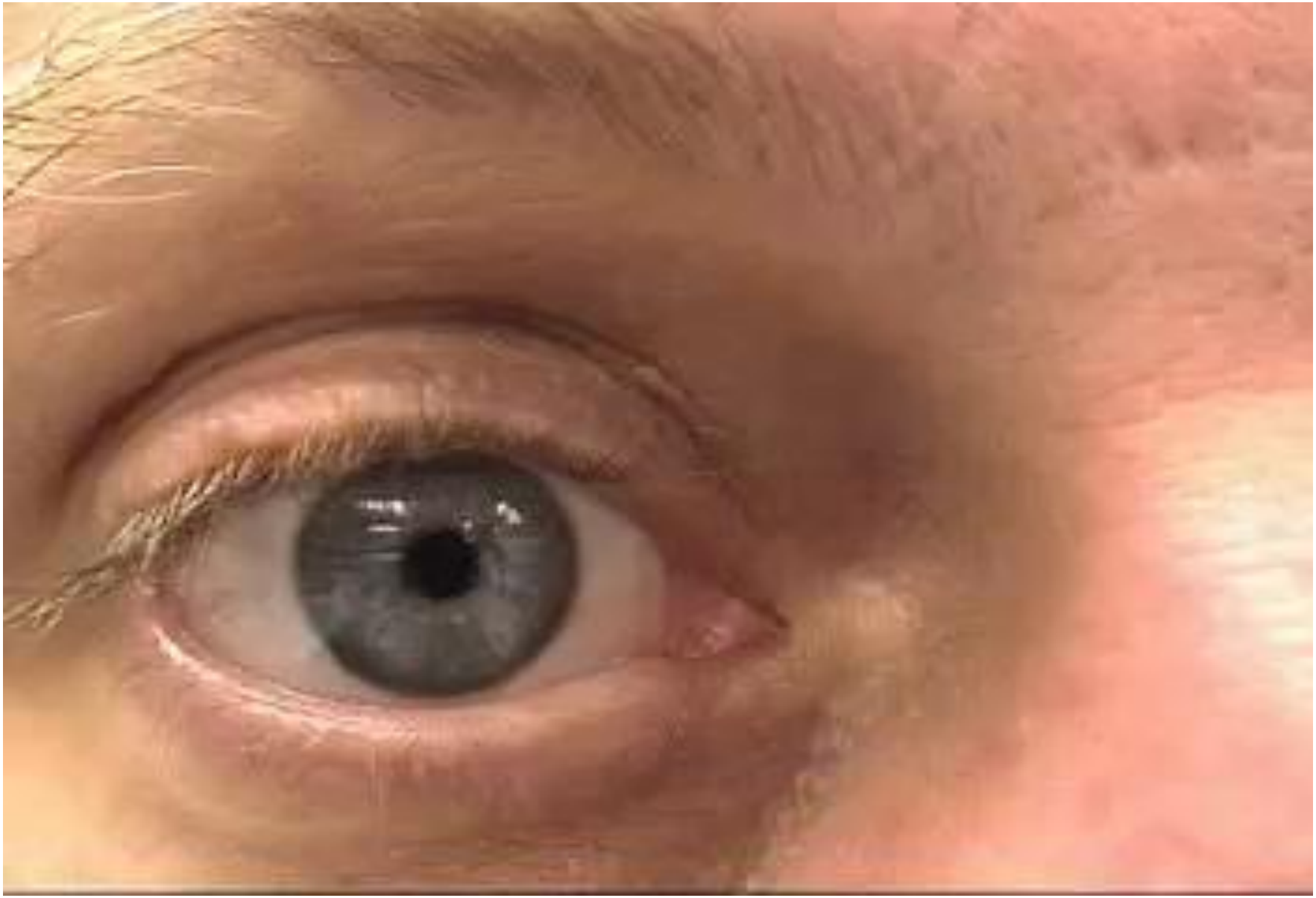
Pressure testing

- Nystagmus or drift of the eyes should also be assessed after positive and negative pressure (Valsalva, tragus compression: Hennebert sign)
- Positive response:

- perilymphatic fistula, superior canal dehiscence
- hypermobile stapes
- occasionally in Menière's disease
- Chiari Malformation

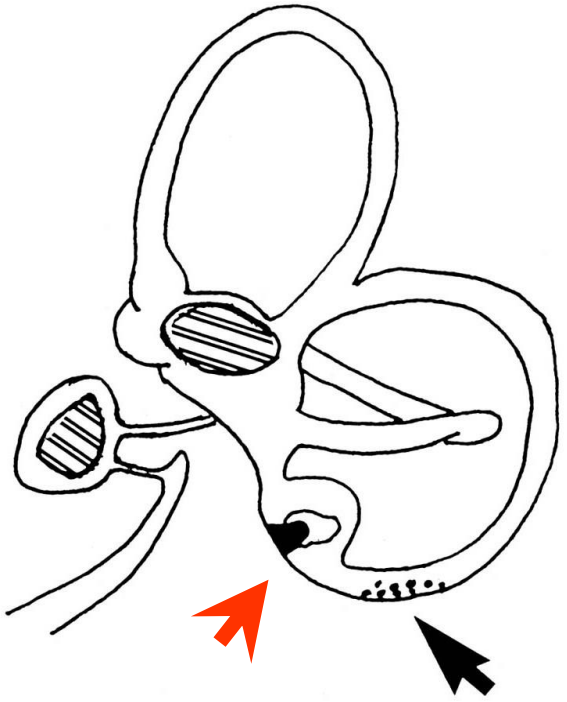


Valsalva’s maneuver in Superior Canal Dehiscence



Positional testing

- Essential part of bedside examination
- Diagnosis of BPPV
- Most common vestibular disease



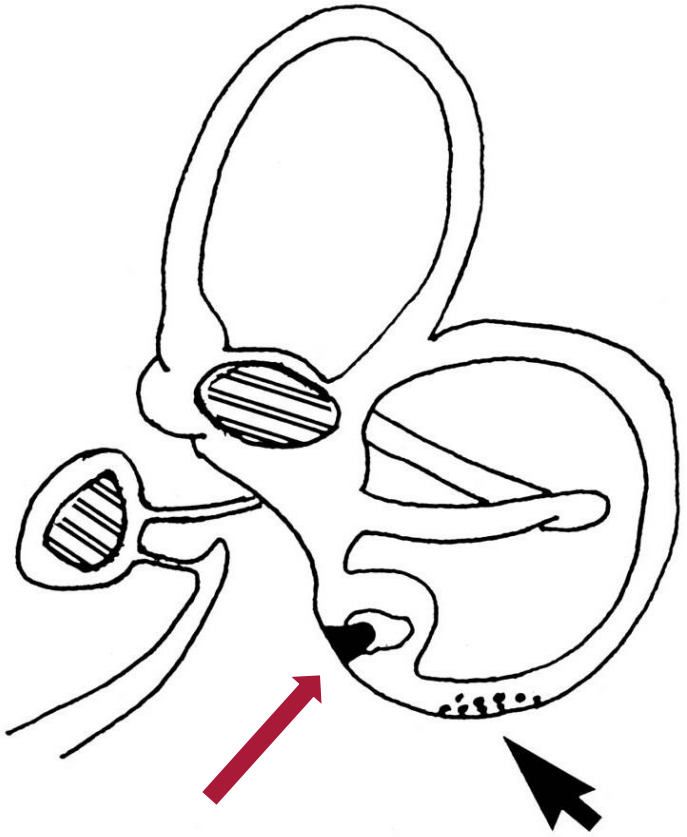
BPPV

- Easily diagnosed by history and clinical exam (even without Frenzel glasses)
- Laboratory testing usually unnecessary
- Pathophysiology reasonably understood
- Usually easily treated with physical therapy, with no medications
- Patients, health care professionals and insurance companies gratified

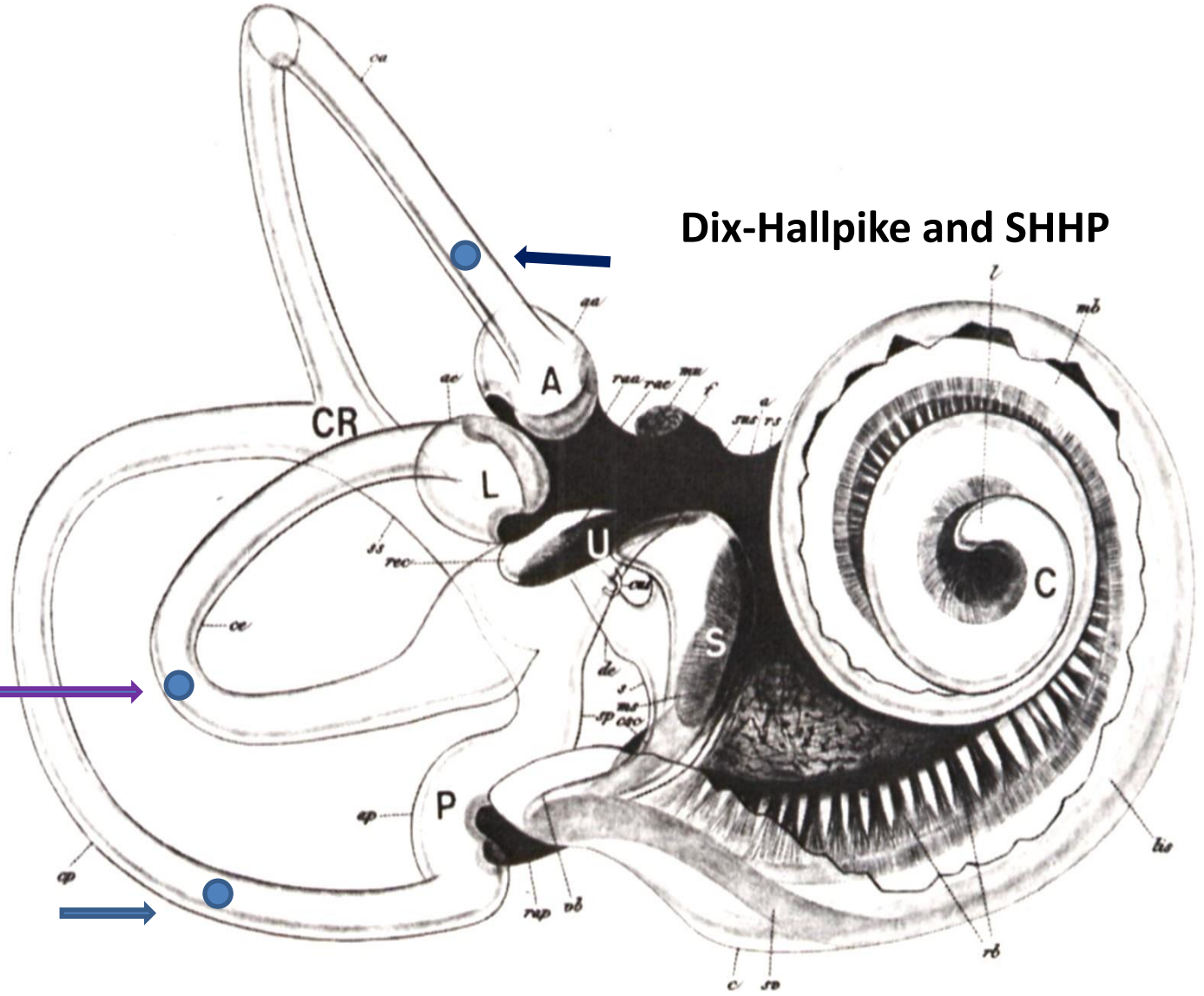
BPPV

Semicircular Canal (usually posterior) becomes gravity sensitive due to floating debris (otoconia dislodged from the macula of the utricle)

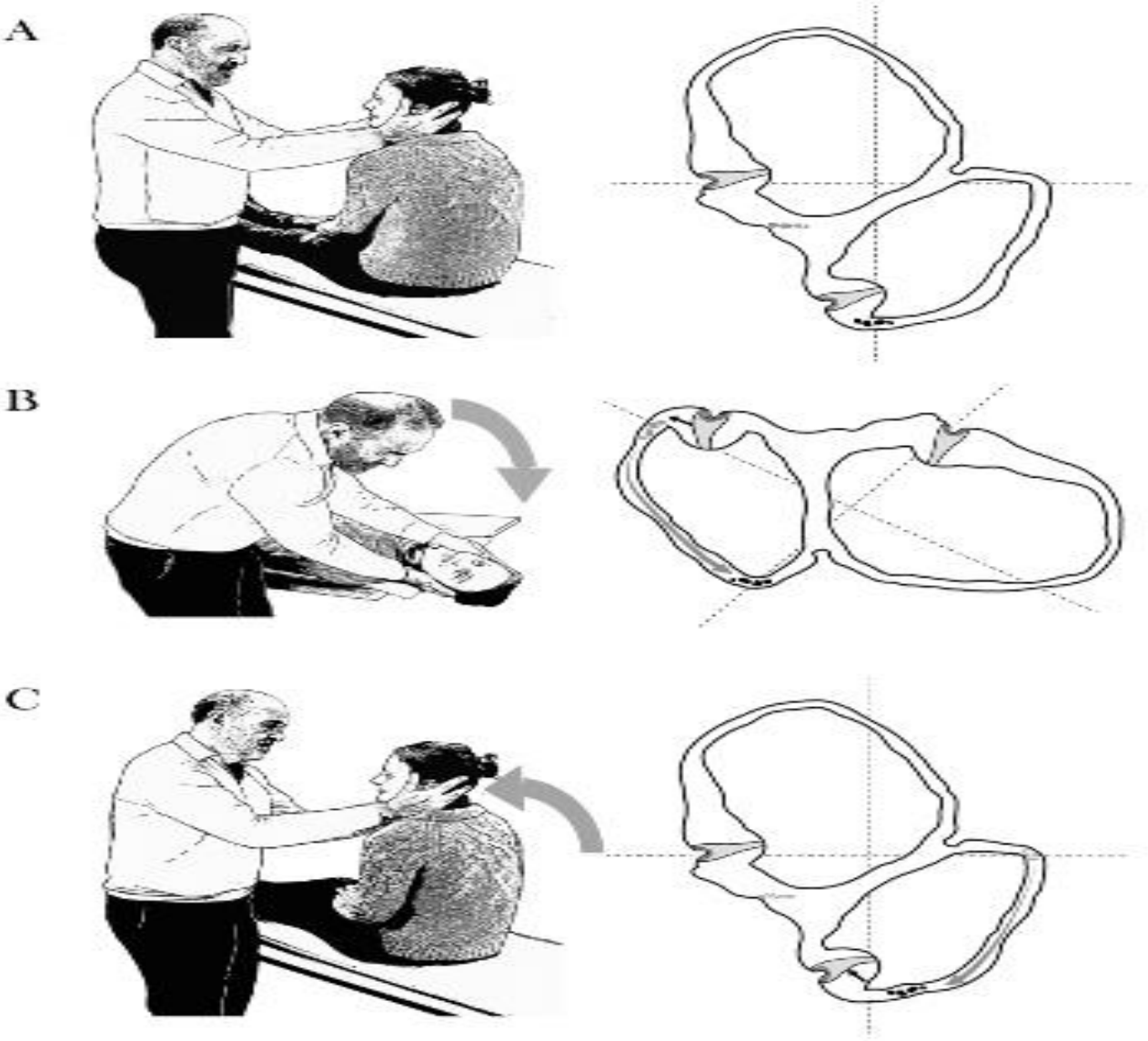
Free-floating in the long arm of the canal (CANALOLITHIASIS)
or
Otoconia get trapped in the posterior SCC on the cupula (CUPULOLITHIASIS)



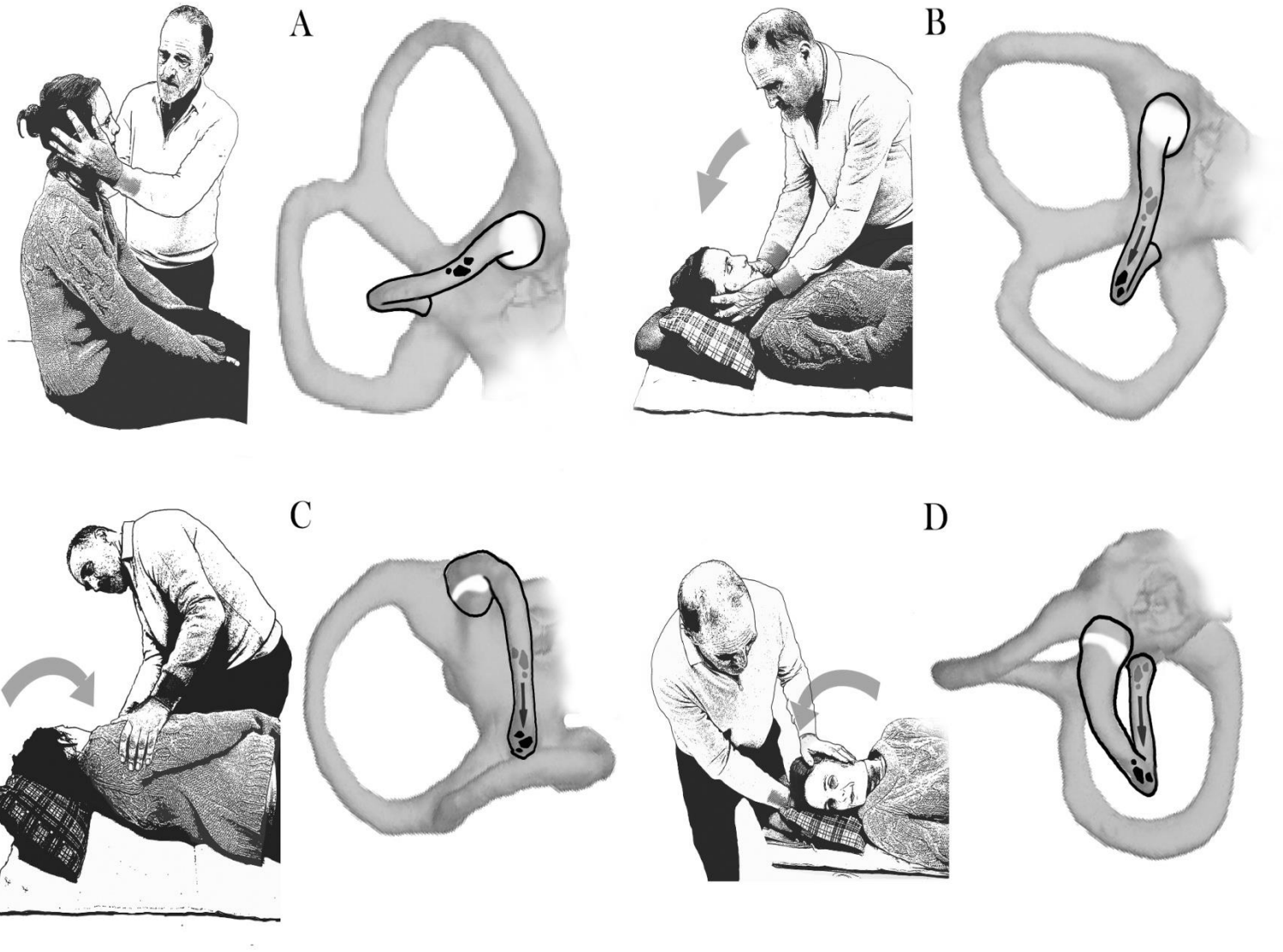
Diagnosis of BPPV



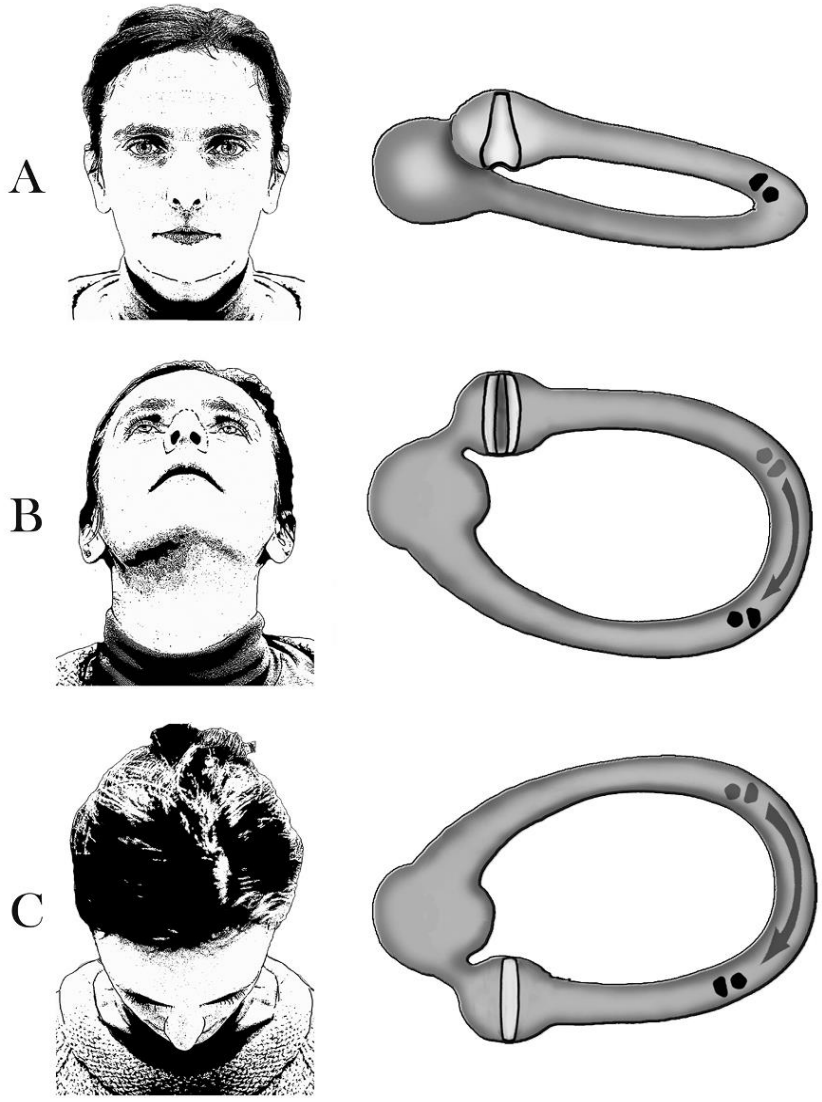
Dix-Hallpike



Supine head-roll test



Bow and Lean (left LC canalolithiasis)



Horizontal PN
away from the affected side
(to the right ear)

Horizontal PN
towards the affected side
(to the left ear)