Cours d'été de la SSORL
Sommerschule SGORL

Bettlach, 27 août 2016

The Vestibular System: from the periphery to the cortex

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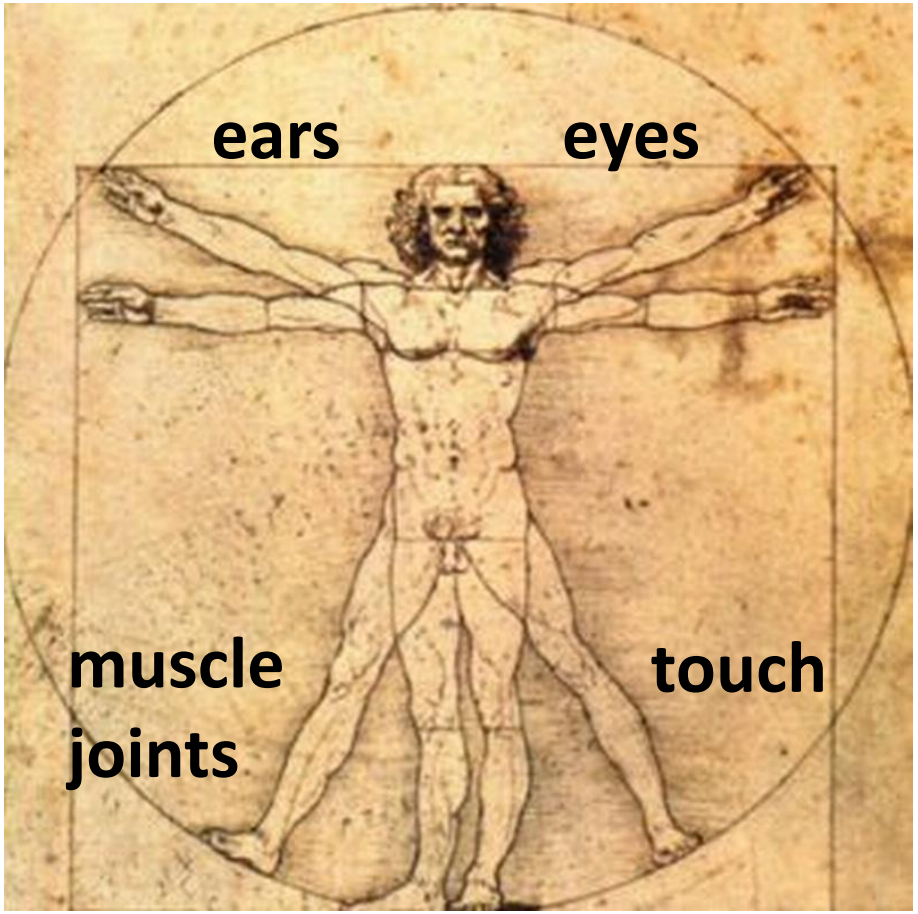
Equilibrium : complex function



<u>Sensory inputs</u>

vestibular

visual

proprioceptive

Equilibrium : vestibular system

multimodal system (6th sense)

Immediate CNS integration of bilateral multisensory informations

- at the subcortical level
  - vestibular nuclei
  - cerebellum: uvula, nodulus, fastigial nuclueus
  - thalamus
- at the cortical level
  - multiple vestibular areas

Vestibular system : functions

- **Stabilization motor reflexes**

  - eye : maintenance of gaze stability during head / visual scene movements

  - body : maintenance of posture

- **Spatial orientation:**

  perception of verticality
  perception of self-motion

Vestibular system

convergent and multimodal system

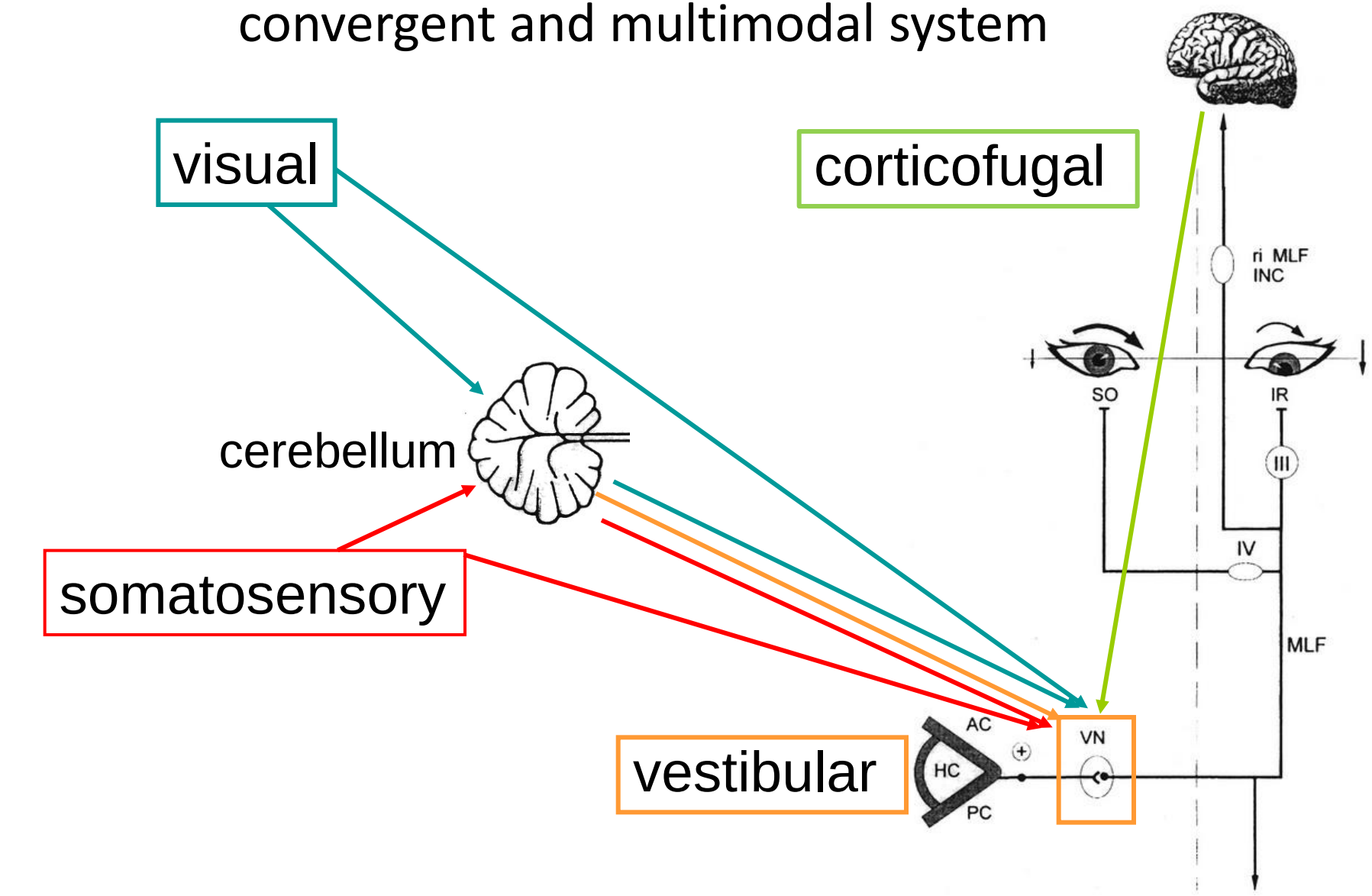
visual

corticofugal

cerebellum

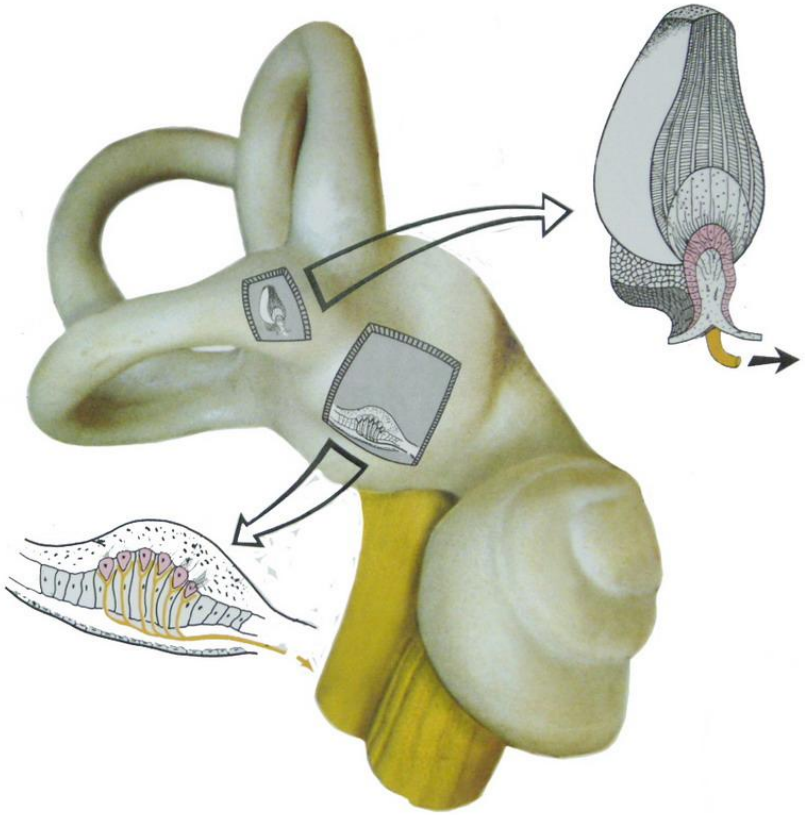
somatosensory

vestibular



Vestibular end organs

5 motion sensors



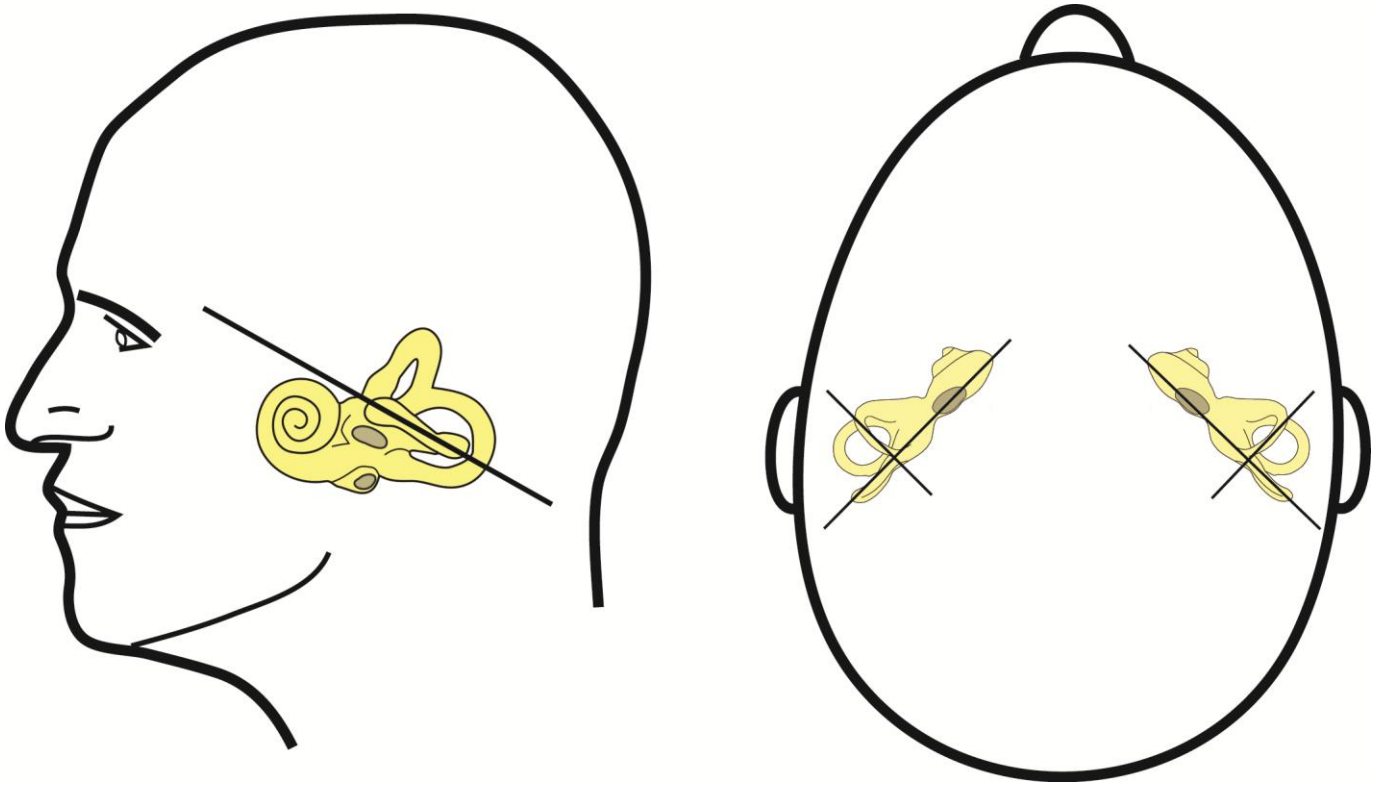
3 semi-circular canals

respond to angular acceleration: rotation

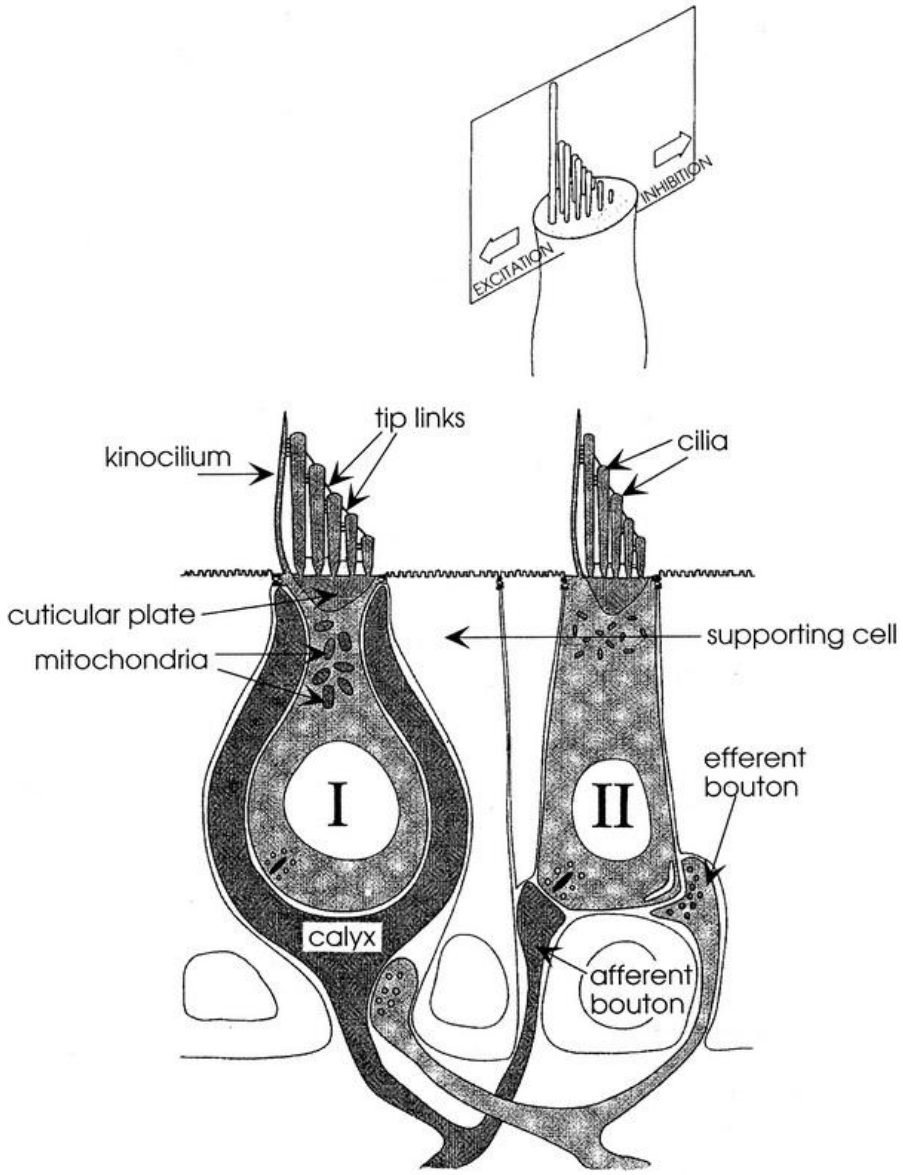
2 otoliths (utricle, saccule)

respond to linear acceleration: gravity, translation

Labyrinth : anatomical orientation



Sensory vestibular cells



Cristae: 23’000 cells

Macula: 52’000 cells

Cupular deflection towards kinocil

⟶ depolarisation (excitation)

Cupular deflection away from kinocil

⟶ hyperpolarisation (inhibition)

Asymmetry of the response:

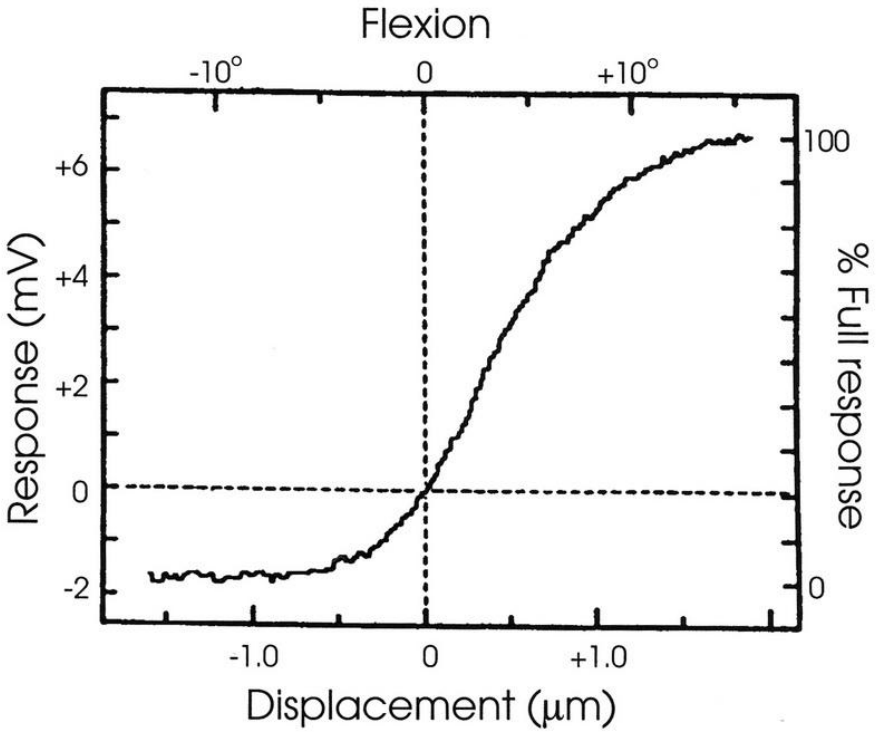
gain excitation > inhibition

Primary vestibular neurons

About 15’000 fibers

Large diameter fibers: connect cell type I

Narrow diameter fibers: connect cell type II



Resting potential: 70-90 spikes/sec

Excitation: ↑ 350 spikes/sec

Inhibition: ↓ 0 spike/sec

Asymmetry of response (Ewald’s law)

Afferent vestibular system

Type I cells and large diameter neurons

low sensitivity to acceleration, phasic response

no high intensity saturation

sensitive to high frequency, high gain

kinetic role

Type II cells and narrow diameter neurons

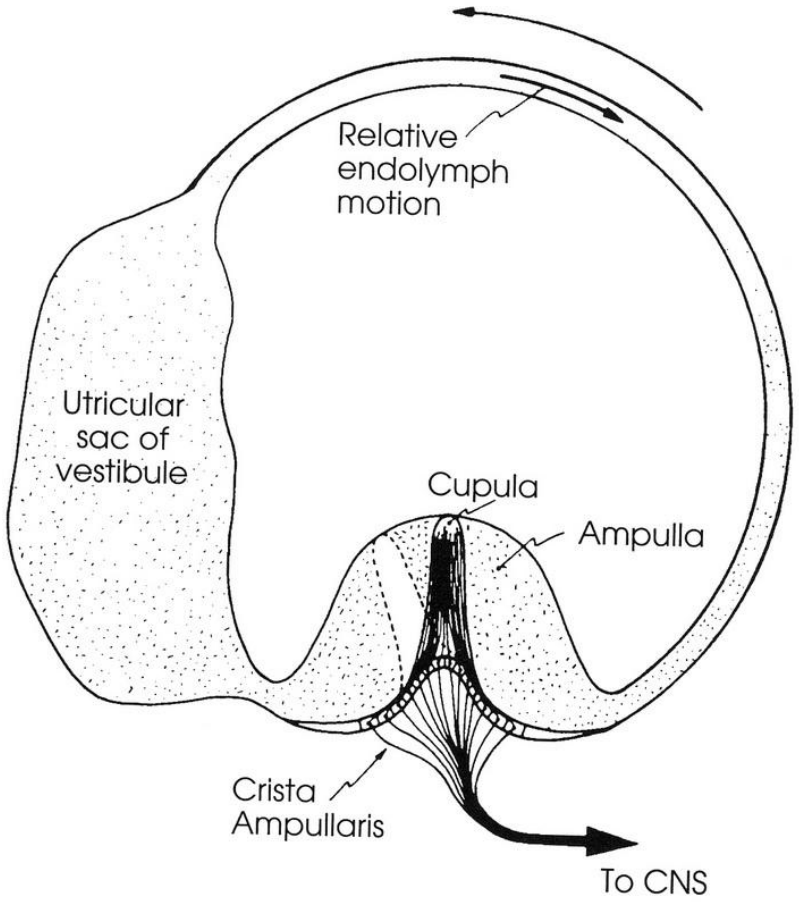
high sensitivity to acceleration, tonic response

high spontaneuous activity, early saturation

sensitive to low frequency, low gain

static role

Semi-circular canals: cristae



Internal diameter: 0.4 mm

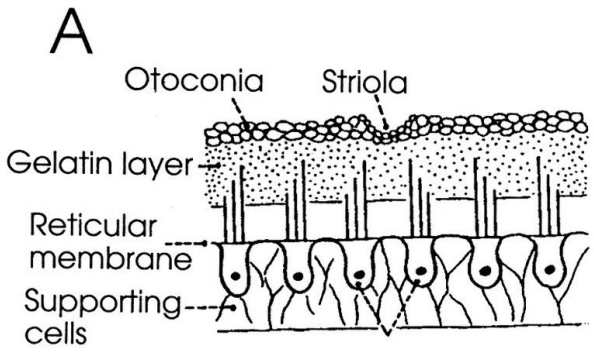
kinocil stimulation in canal axis
no cupula mass effect
⟶ insensitive to gravity

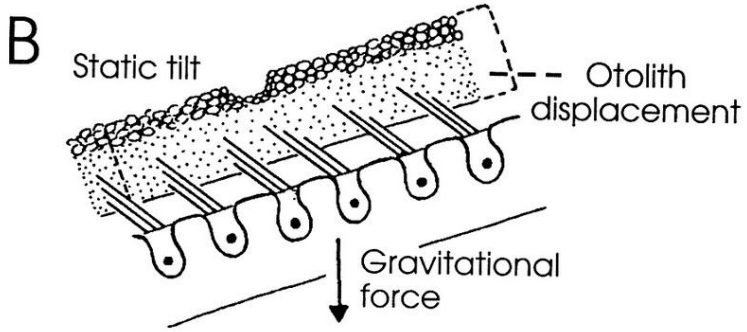
horizontal SCC
kinocil towards utricle
⟶ excitation by ampullopetal (= utriculopetal) endolymph flow

Vertical SCC
kinocil towards canal
⟶ excitation by ampullofugal endolymph flow

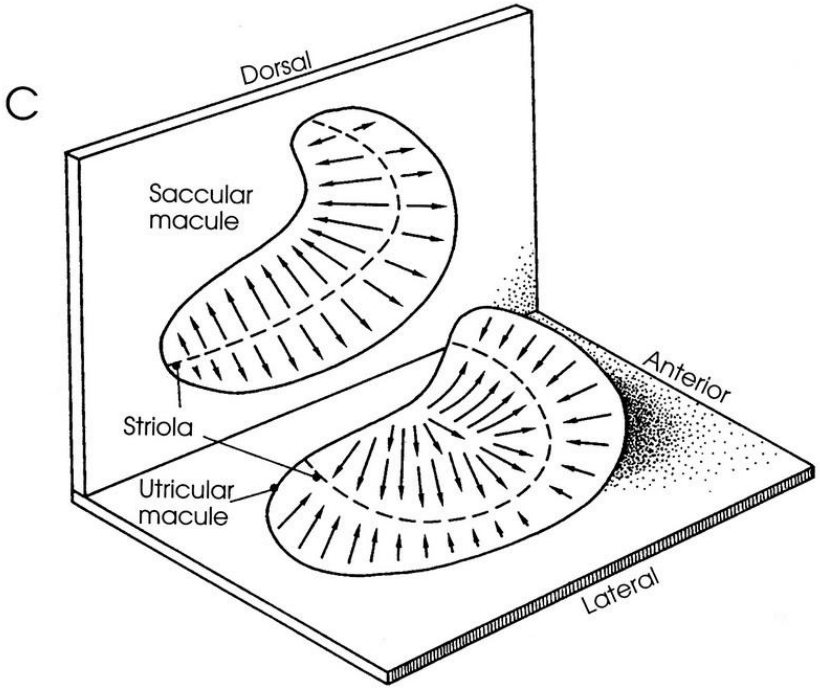
imperfect orthogonal alignment
angular motions stimulate at least 2 or 3 SCCs

Otoliths: macula





Tilt : gain=0.1



Macula: density 2x H_2O ⟶ mass effect
surface < $1mm^2$
thickness: 50µm

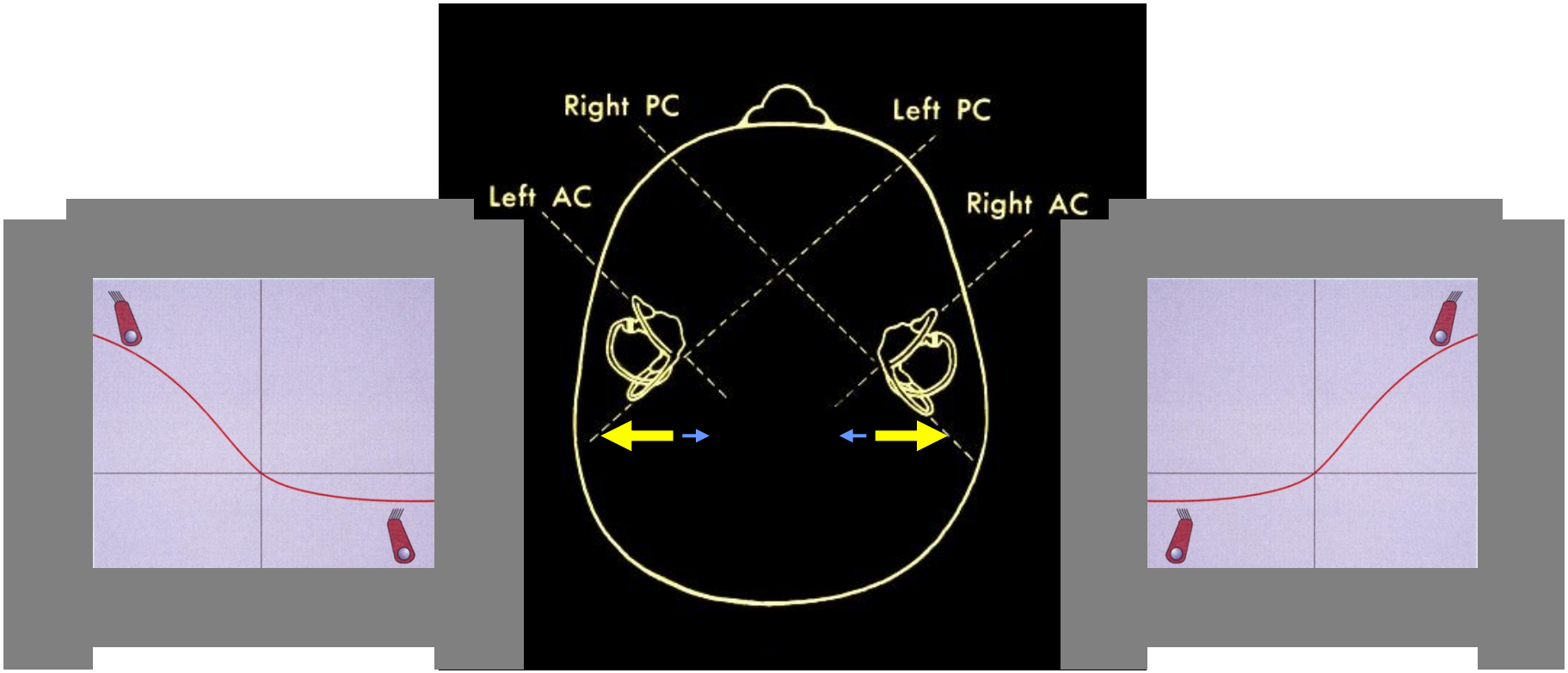
Striola: center

Saccule: vertical, kinocils back to back

Utricle: horizontal, kinocils face to face

Semi-circular canals: Ewald's 2nd law

Asymmetry of response

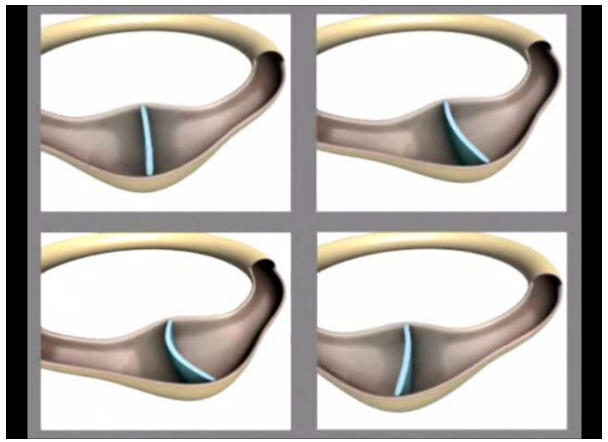


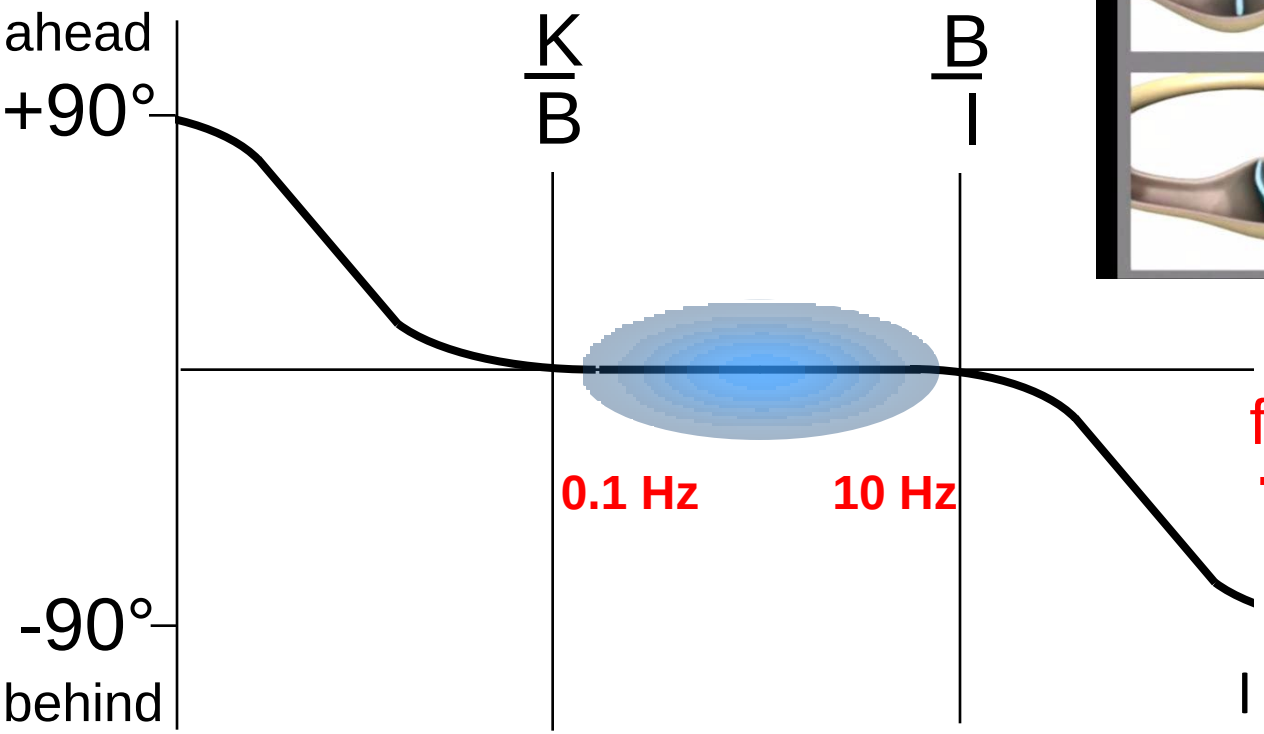
loss of gaze stabilisation (towards bad-side)
especially for fast head movements

Courtesy of H. Kingma

Semi-circular canals: frequency dependence

Phase (timing)

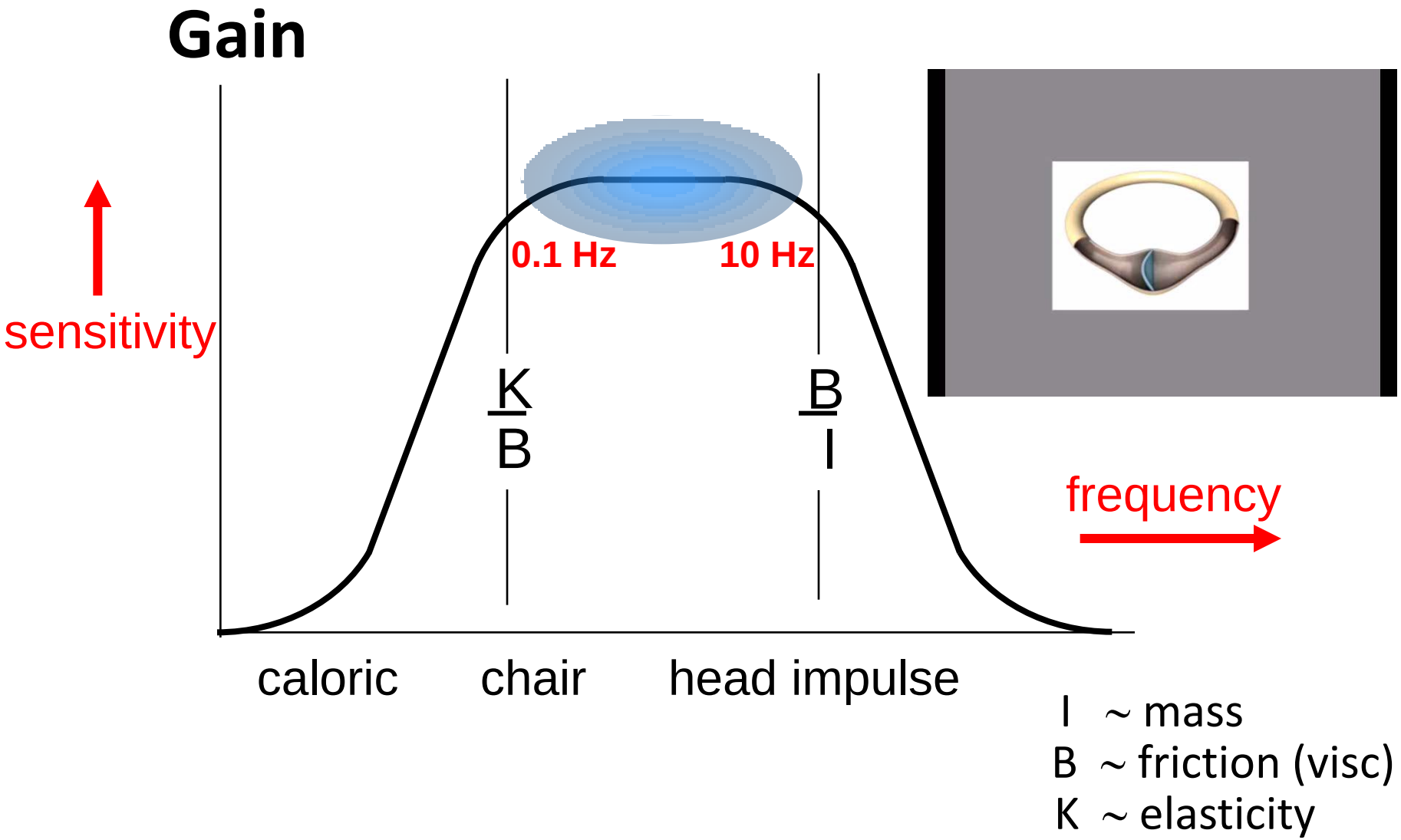




I ~ mass
B ~ friction (visc)
K ~ elasticity

Courtesy of H. Kingma

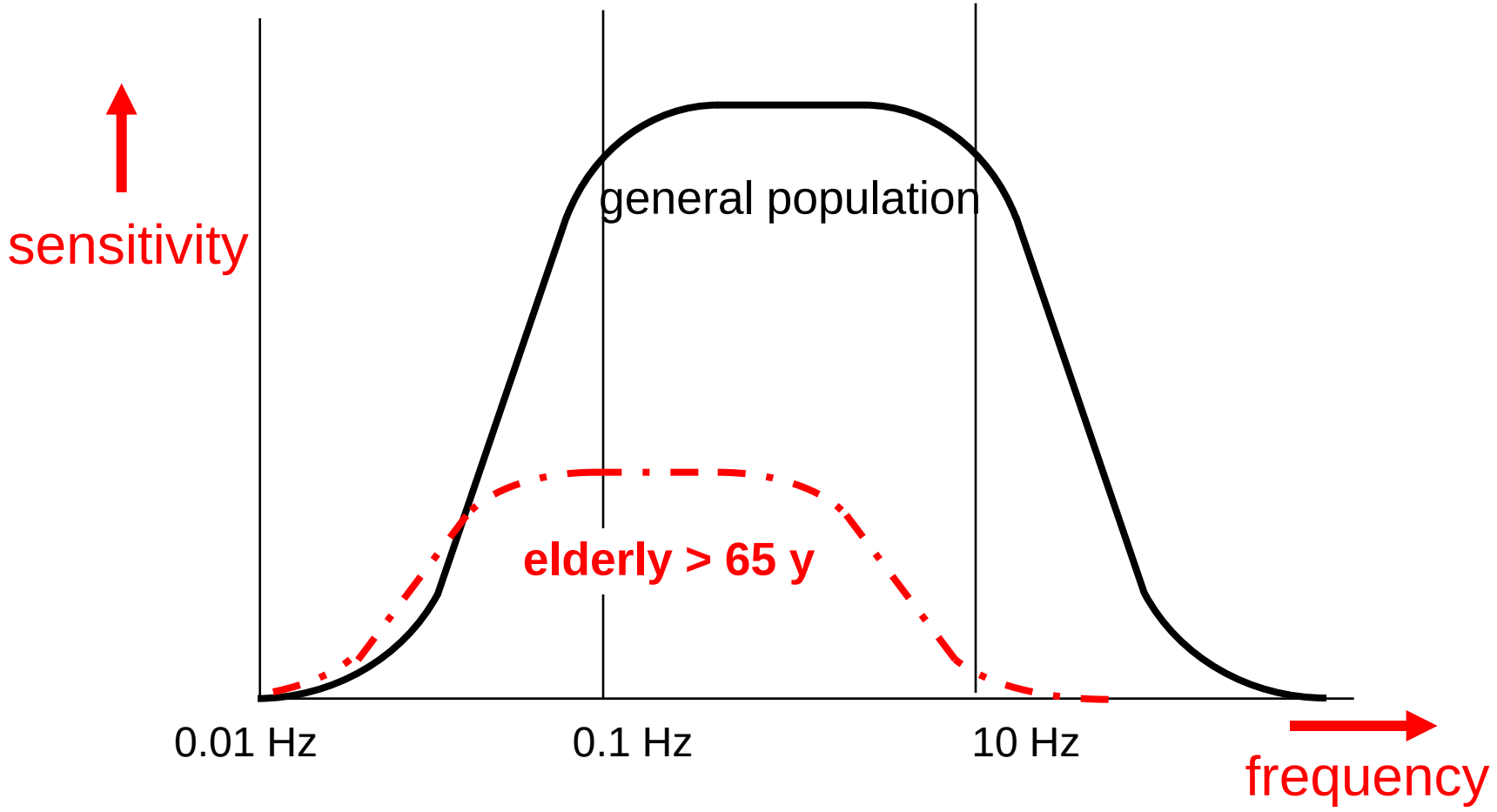
Semi-circular canals: frequency dependence



Courtesy of H. Kingma

Semi-circular canals: frequency dependence

ageing (> 60 y) decrease in sensitivity and gain

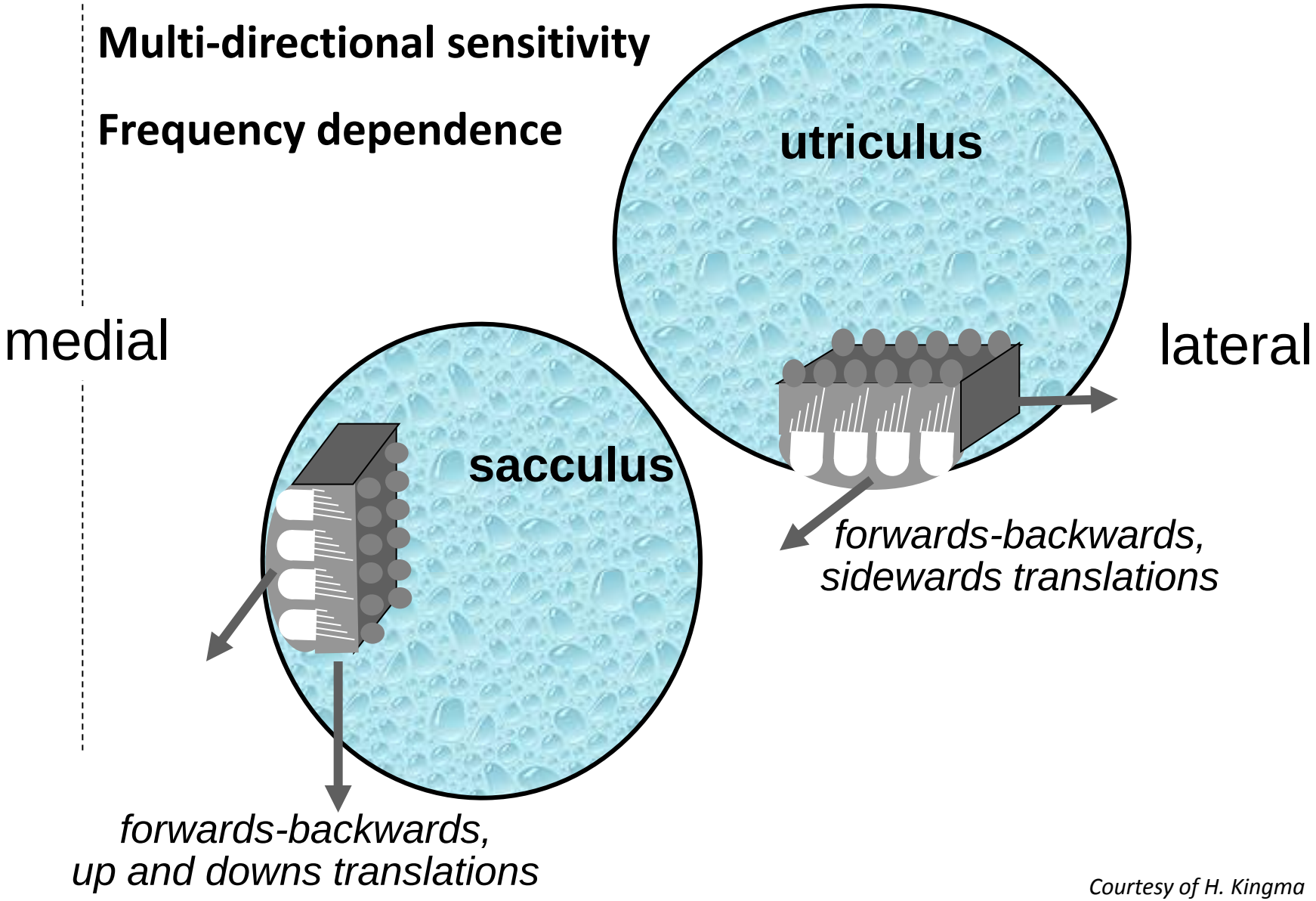


Courtesy of H. Kingma

Otolith organs

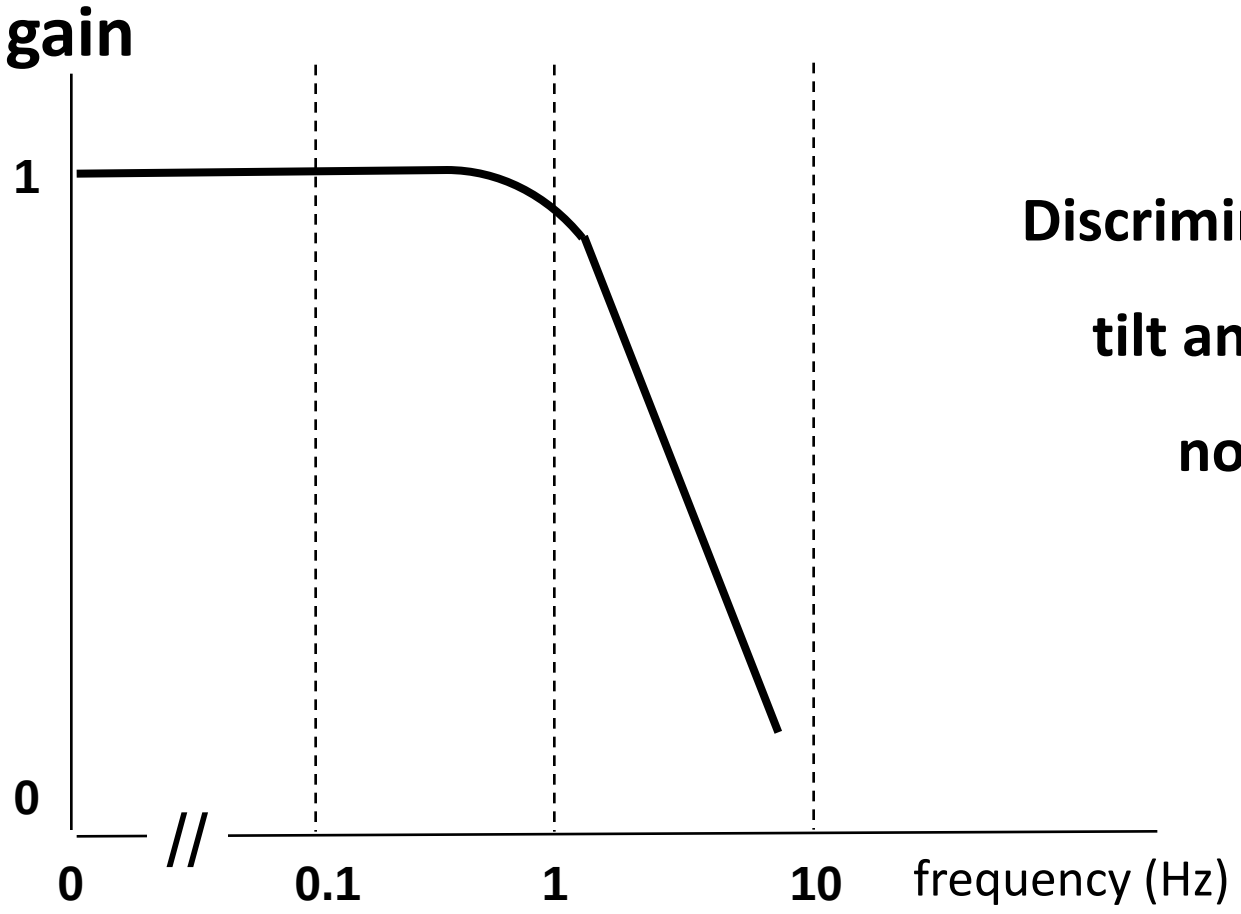
Multi-directional sensitivity

Frequency dependence



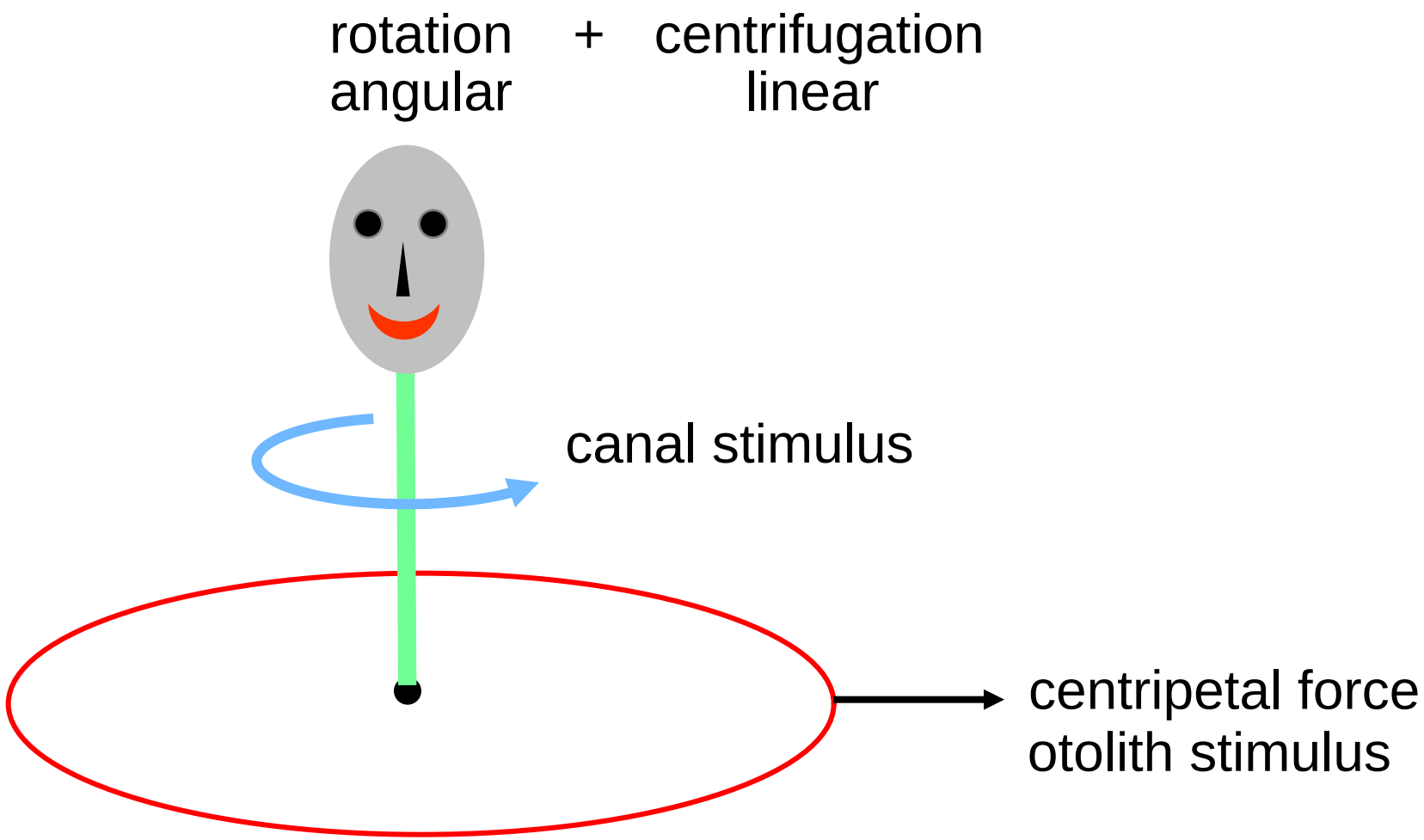
Courtesy of H. Kingma

Otolith organs



Discrimination between tilt and translation not possible

Otolith organs: also stimulated by rotations



Problem of vestibular receptors

Anatomically fixed in the head
→ head-centered reference frame

- rotation problem

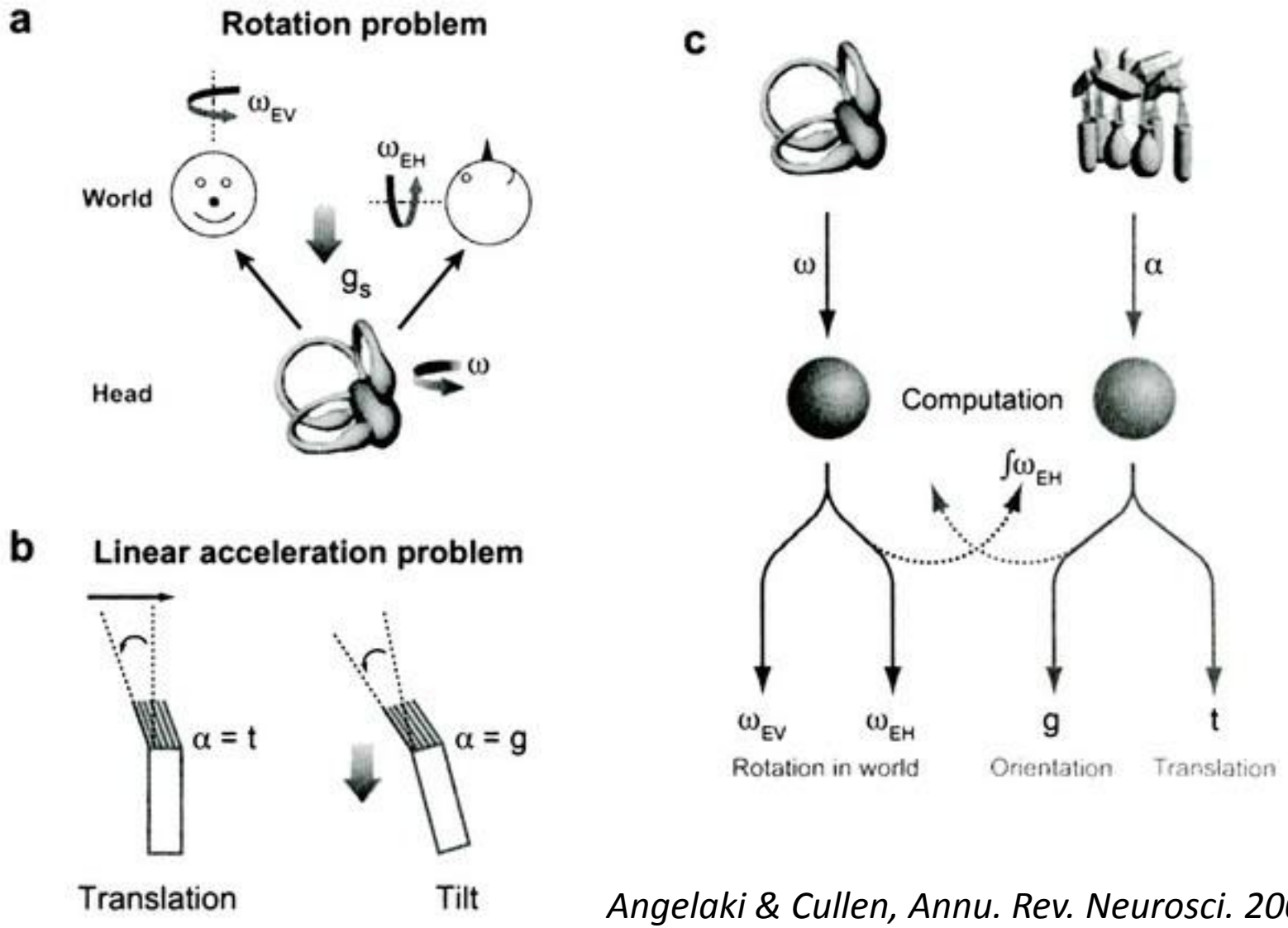
  axis of motion relative to the world

- linear acceleration problem

  distinction between tilt and translation

→ **otolith-canal interaction**

Otolith-canal interaction



Angelaki & Cullen, Annu. Rev. Neurosci. 2008

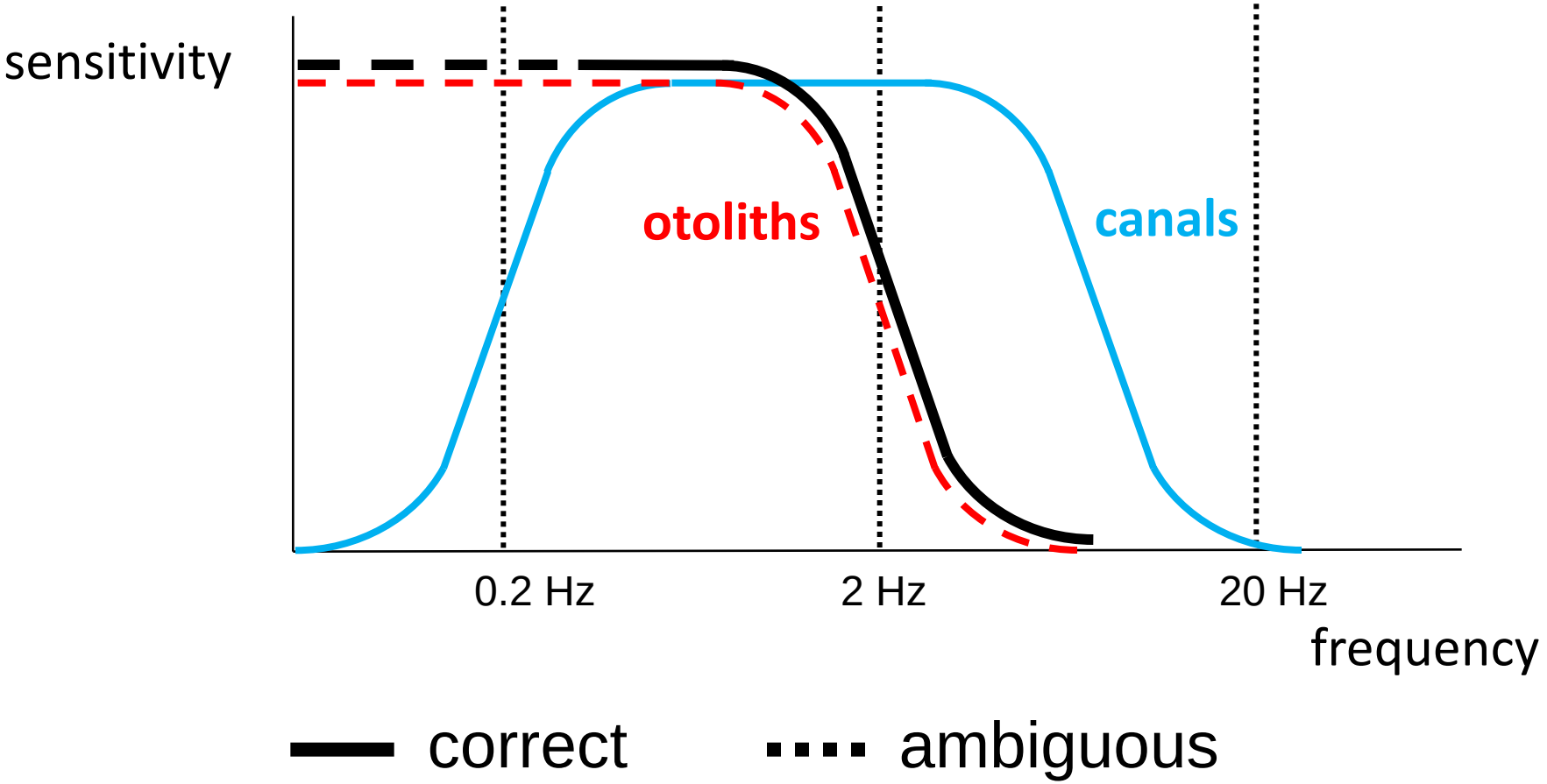
Otolith-canal interaction

- allows adequate coding of movement relative to the world (world-centered coding)

- allows the vestibular system to function as an inertial sensor for navigation and spatial orientation

Otolith-canal interaction

Discrimination between tilt and translation



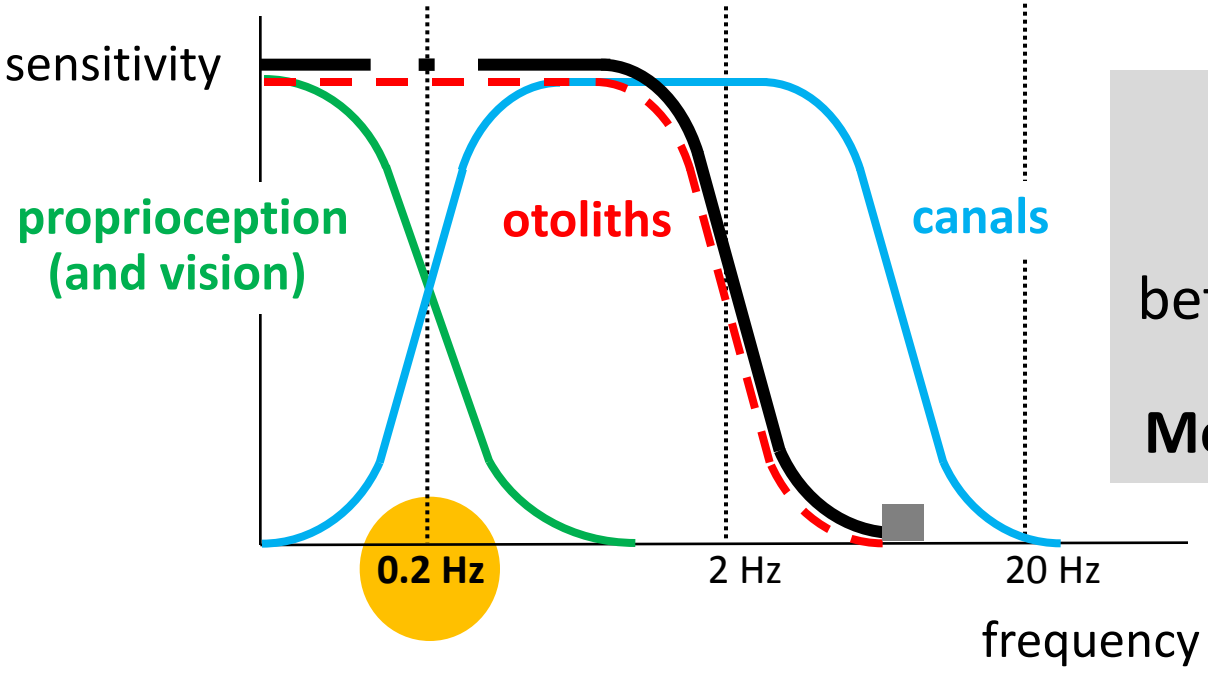
Proprioceptive-vestibular interaction

- allows body-referenced perception of motion
  (body-centered coding)

- distinction between active and passive movement

Proprioceptive-vestibular interaction

Discrimination between tilt and translation



0.2Hz

Persisting ambiguity between tilt and translation

Motion sickness frequency

Visual-vestibular interaction

- maintenance of gaze stability during head / visual scene movements
- maintenance of spatial perception during motion at constant velocity
- do not allow the brain to differenciate vestibular (labyrinthine) from optokinetic (visual) inputs

Gaze stability during head / visual scene movements

visual-vestibular-oculomotor system
(or vestibular-optokinetic system)

- vestibulo-ocular reflex
- optokinetic reflex

} **nystagmus**

- smooth pursuit (target tracking)

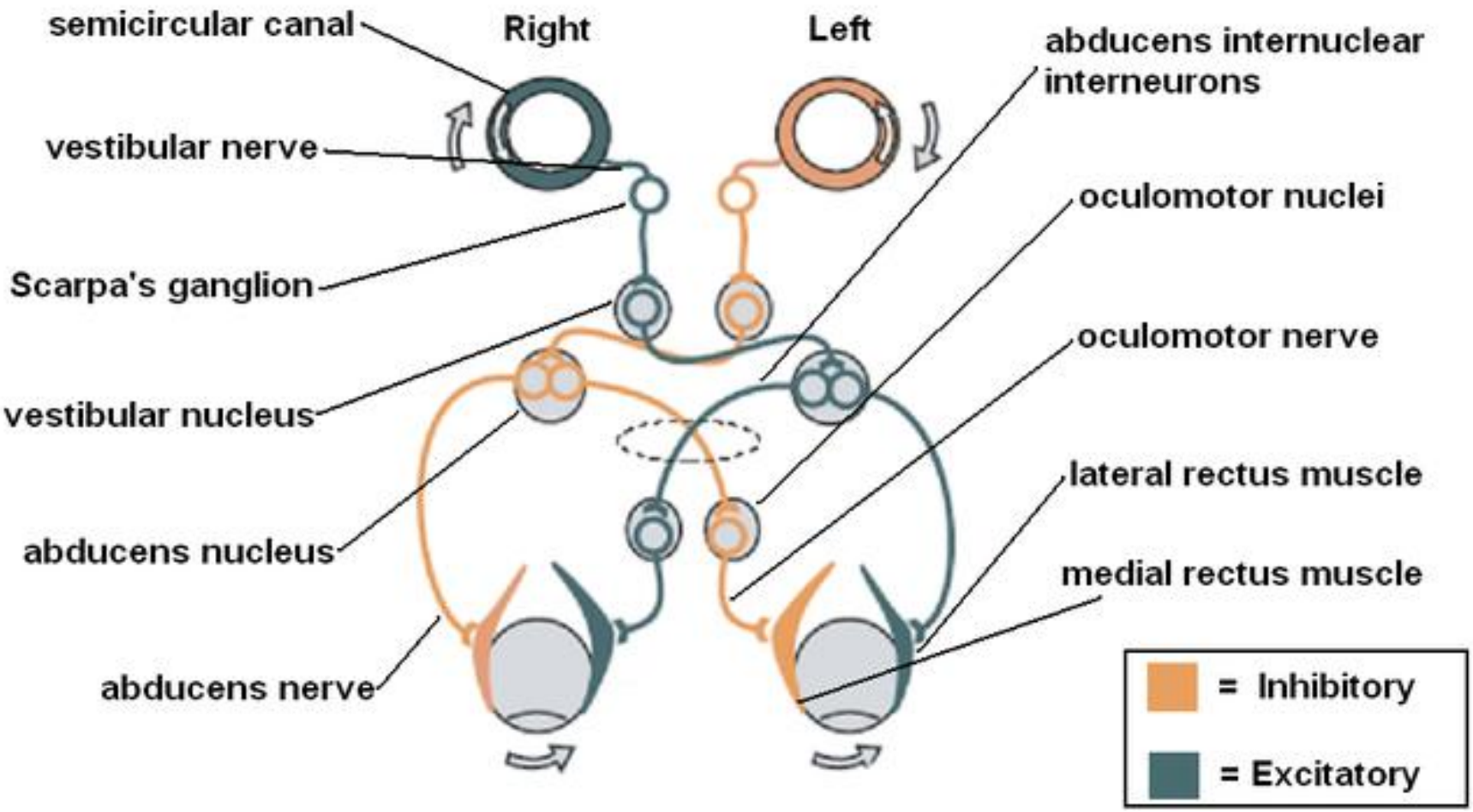
Vestibulo-ocular reflex (VOR)

Effective : natural head movement (0.1 – 5 Hz)

- **direct pathway (rapid)**
  short latency < 8 msec
  brief, fast head movement
  small amplitude
  → **compensatory eye displacement**
  1 – 5Hz and velocity > 100°/sec : gain ≈ 1

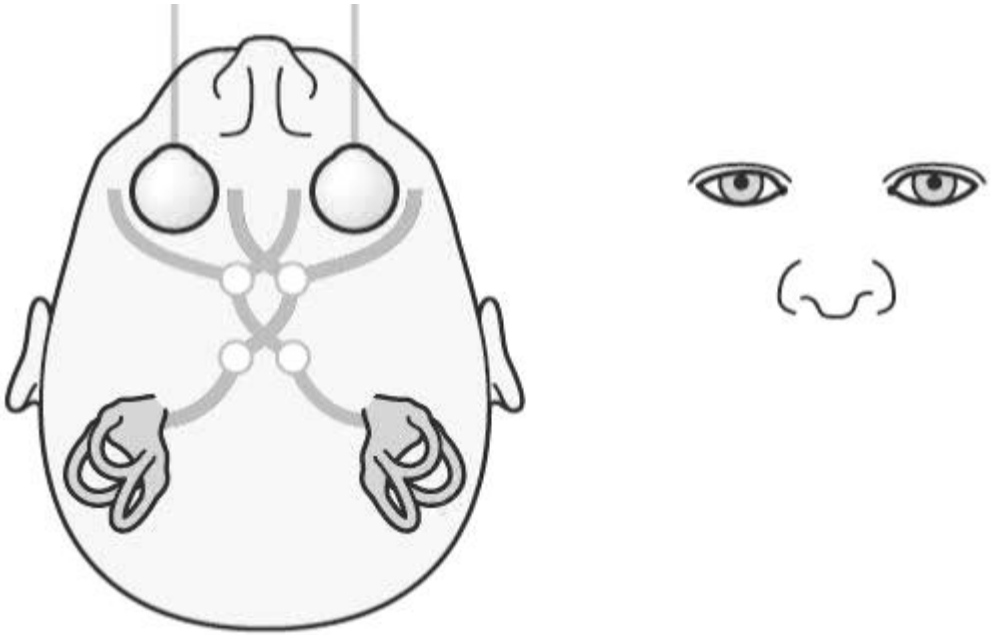
- **indirect pathway (slow)**
  via velocity storage
  slow, continuous head movement
  large amplitude
  → **vestibular nystagmus**

VOR: three neurons reflex arc



VOR : direct pathway

compensatory eye movement



three-neuron chain reflex

VOR : direct pathway

Fast head movement: 0,5 – 5 Hz ; > 100°/sec

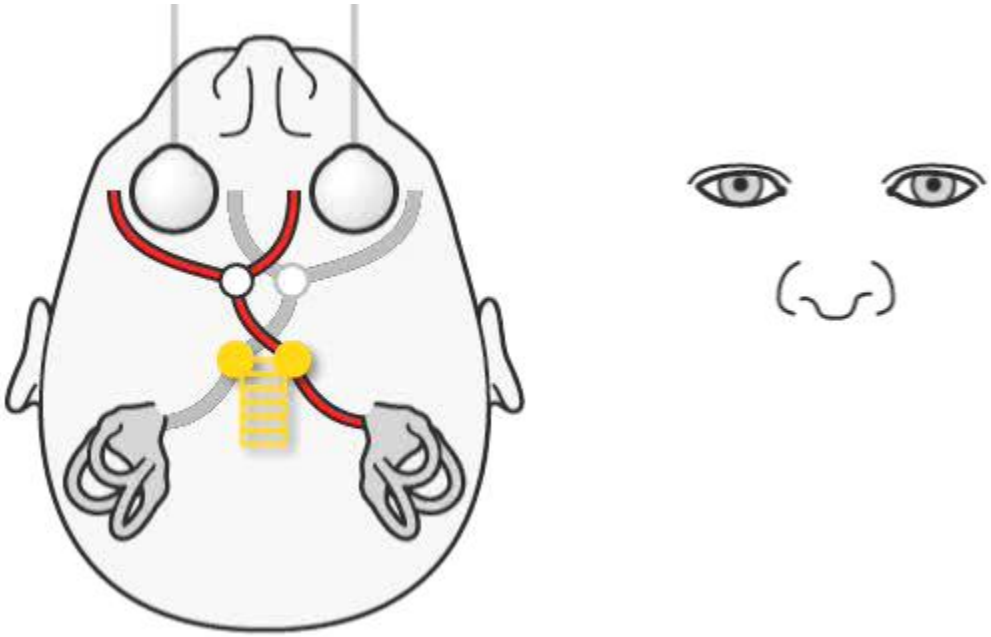
gain ≈ 1

Head thrust

Halmagyi Test (Head Impulse Test)

VOR : indirect pathway

Slow and continuous head movement



three-neuron chain + velocity storage

Vestibular nystagmus

Velocity storage: overview

Definition:

- central integrator mechanism used for producing vestibular nystagmus, OKN and OKAN
- activated by both visual and vestibular rotation cues
- component of the indirect, slow, « low frequency » VOR/OKN pathways that generate nystagmus
- prolonges the time course of VOR response

Velocity storage: overview

Properties / functions

- integration and storage of slow phase eye velocity
  → increase of VOR sensitivity
  → balance /equilibration of VOR during rotation

- mainly involved in *hor*VOR, poor contribution to *vert*VOR

- spatial orientation of nystagmus according to gravito-inertial forces → eye velocity alignment towards gravity

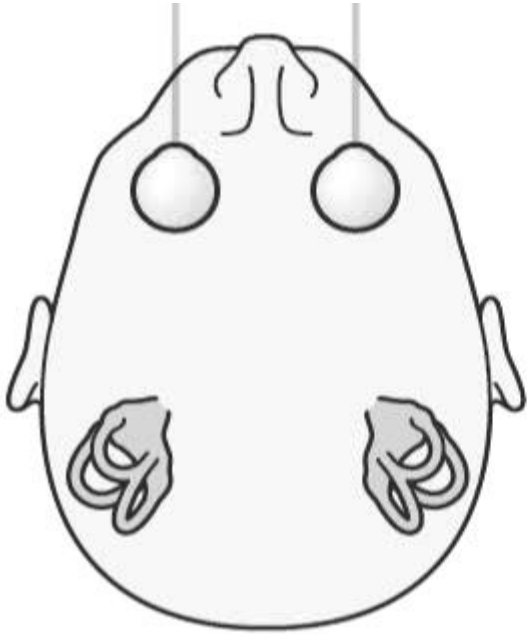
- role in motion sickness susceptibility

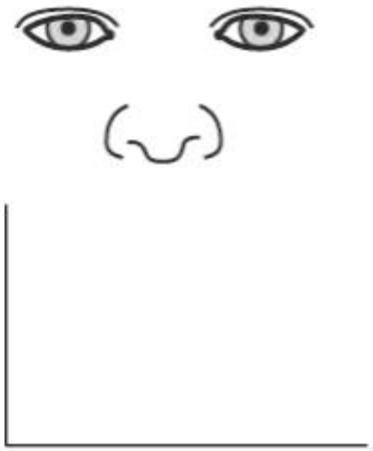
Velocity storage: overview

Neuronal network

- three-neurons canal-ocular arc reflex
- lateral commissural inhibitory fibers connecting
  - medial Vestibular Nuclei
  - descending Vestibular Nuclei
- Nodulus and Uvula
- Nucleus of the Optic Tract, nucleus prepositus hypoglossi

VOR : velocity storage

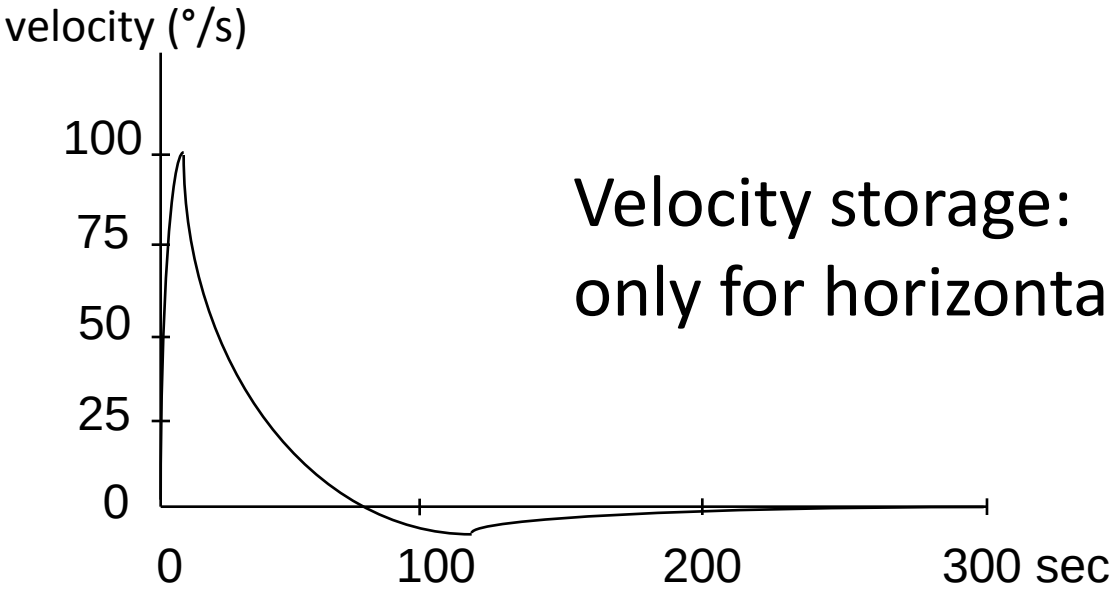




Impulse
(velocity step)

prolonged time discharge
with time constant

VOR : velocity storage



Velocity storage:
only for horizontal canals

Duration (≈ 3x time constant)

$duration_{deflection\ cupula}$	= 6 ms
$duration_{cupula\ back}$	= 20 s
$duration_{velocity\ storage}$	= 60 s
$duration_{central\ adaptation}$	> 300 s

Time constant (τ)

τ_{cupula}	≈ 2 ms
τ_{canal}	≈ 6 s
$\tau_{velocity\ storage}$	≈ 20 s
$\tau_{central\ adaptation}$	>100 s

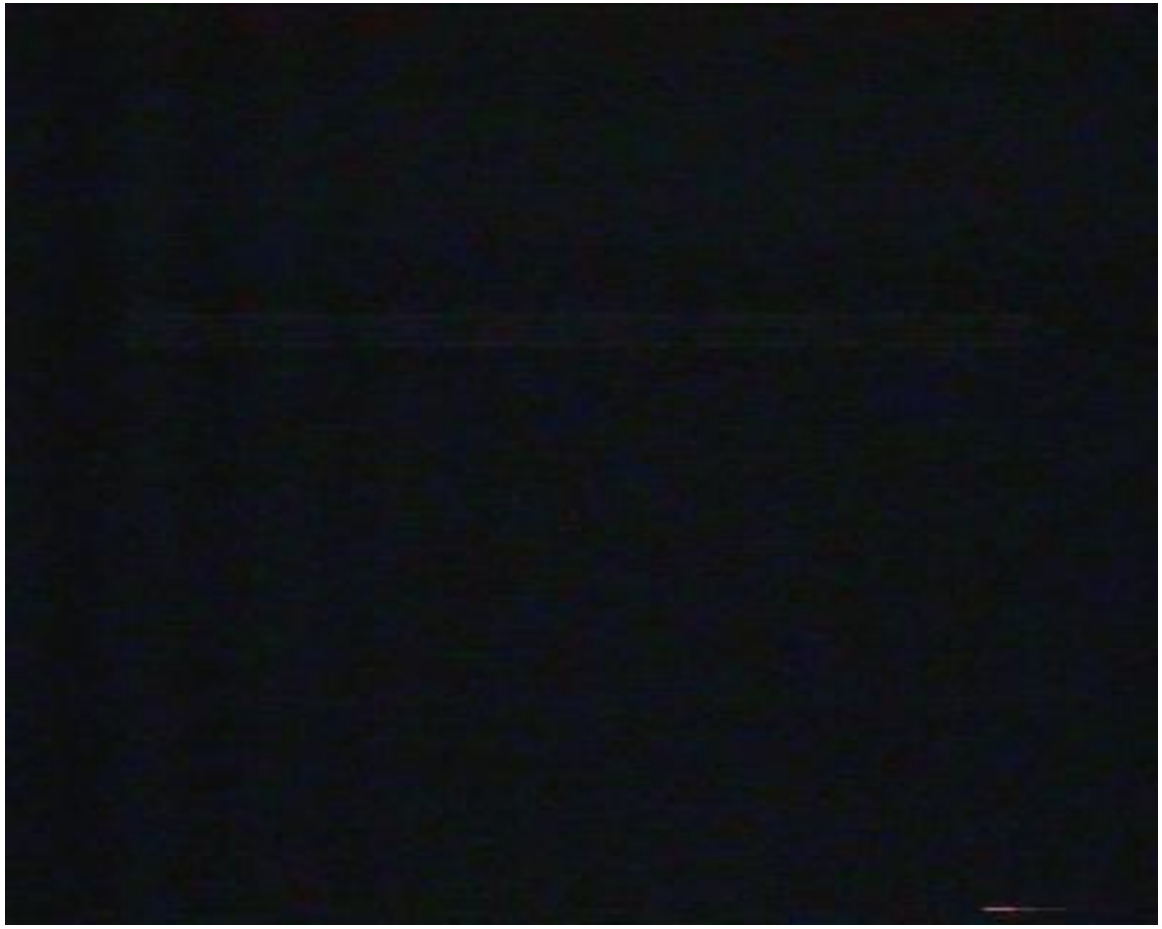
Optokinetic system

Effective : slow image slip on retina
< 1Hz ; velocity < 100°/sec

- **direct pathway = smooth pursuit**
  fast, foveal, volontary (cortical)
  → target tracking
  → visual suppression of vestibular nystagmus

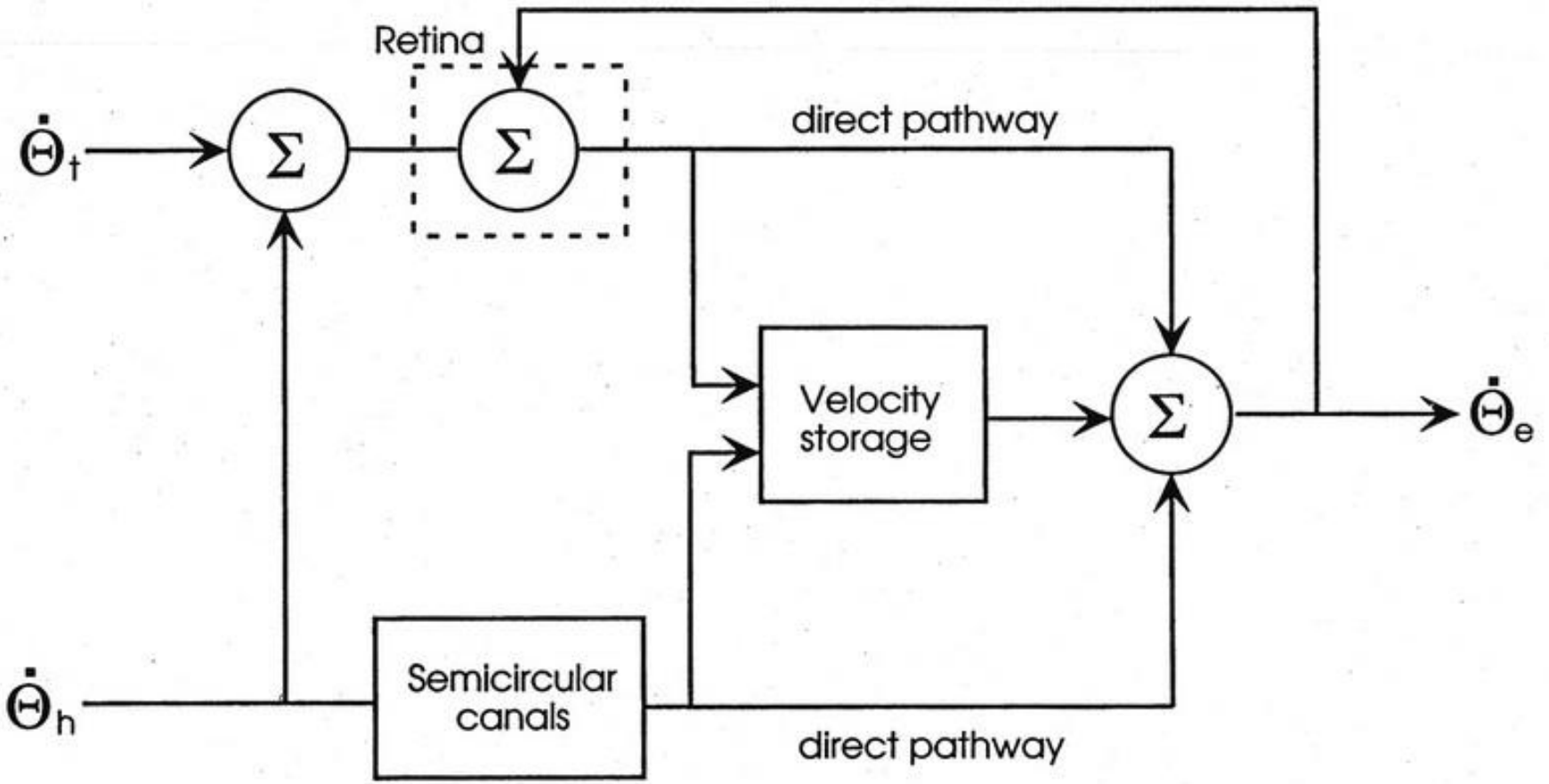
- **indirect pathway**
  slow, extrafoveal, unvolontary (subcortical)
  via velocity storage
  → optokinetic nystagmus

Low frequency < 1Hz



Visual suppression of vestibular nystagmus

Visual-vestibular interaction



In : *Clinical neurophysiology of the vestibular system*
Baloh & Honrubia, 3e ed, CNS, 2001

Vestibular system

Multimodal integration at the cortical level

- multisensory vestibular cortex

  → fusion of the different sensory inputs (multisensory coding)

  → internal representation of space in unique 3-D map

Multisensory vestibular cortex

- no primary vestibular cortex

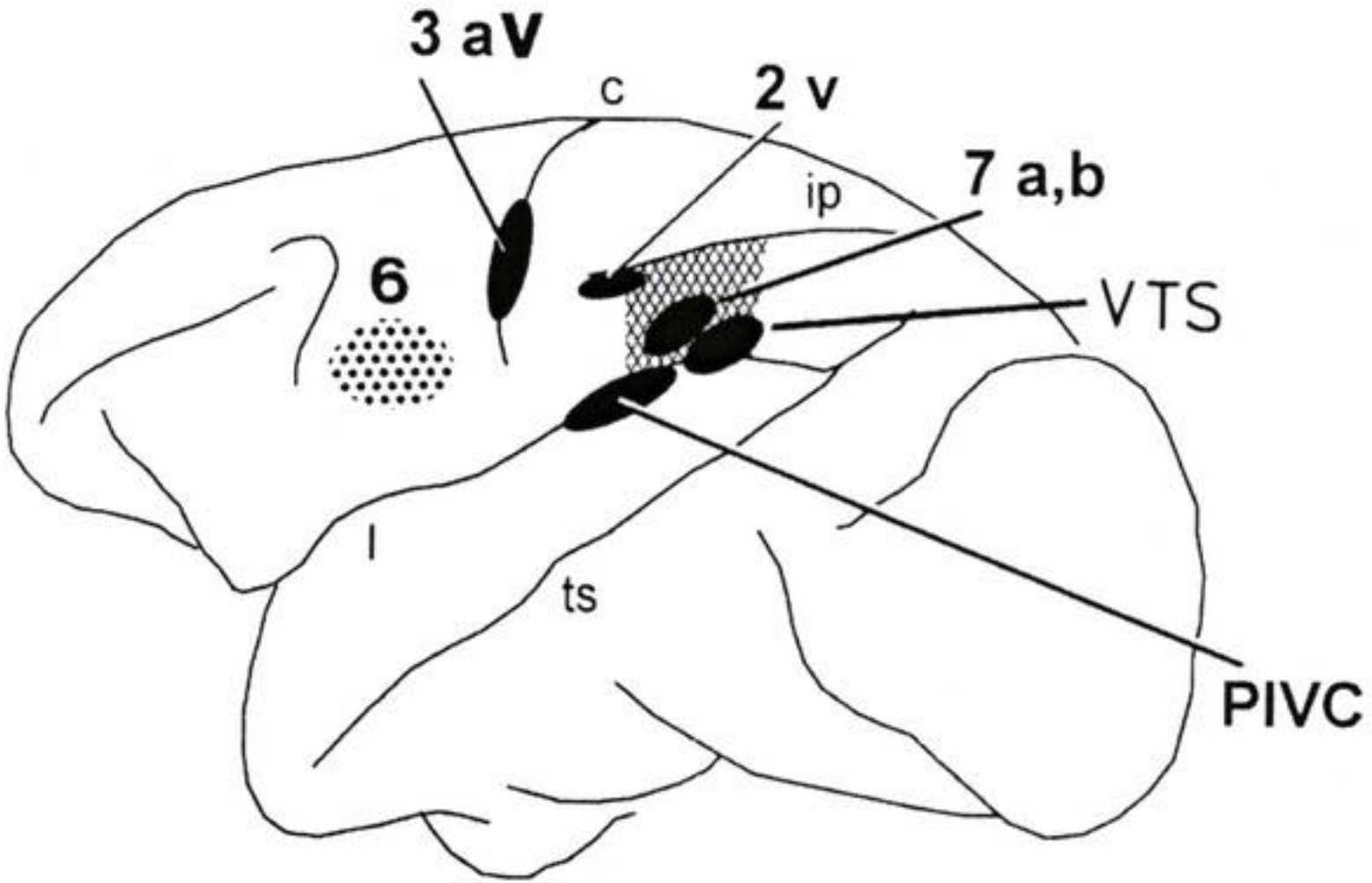
- multiple vestibular cortex areas

  ↳ parieto-insular cortex

  ↳ multisensory neurons

  respond to vestibular, optokinetic and somatosensory stimuli

Vestibular cortex (monkey)



Brandt & Dieterich, Ann. NY Acad. Sci. 1999

Parieto-insular vestibular cortex (PIVC)

- **Afferents** (via vestibular nuclei and thalamus)

  semi-circular canals and otoliths

  optokinetic

- **Efferents** (corticofugal feed-back)

  direct projection to vestibular nuclei

PIVC : functions

- perception of verticality
- perception of self-motion
- internal representation of space and body orientation in unique 3-D coordinates

  unified visual-vestibular-somatosensory map

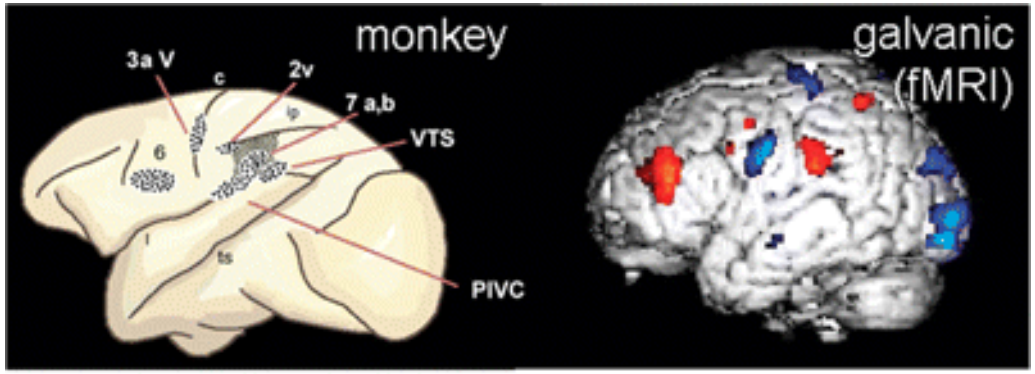
  ↳ egocentric (body-centered)

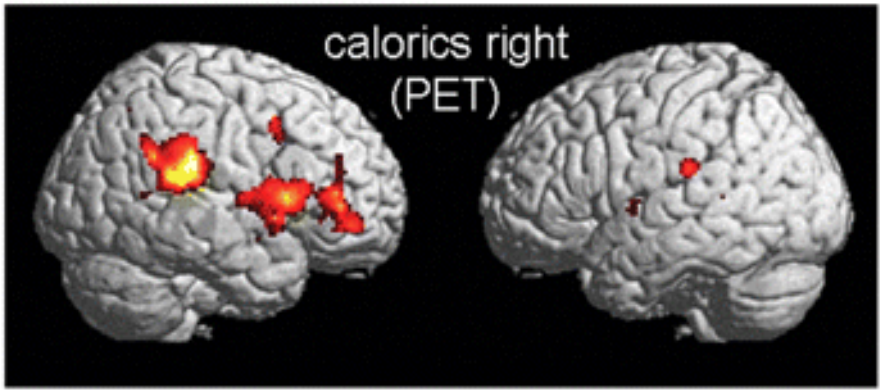
  ↳ exocentric (world-centered)

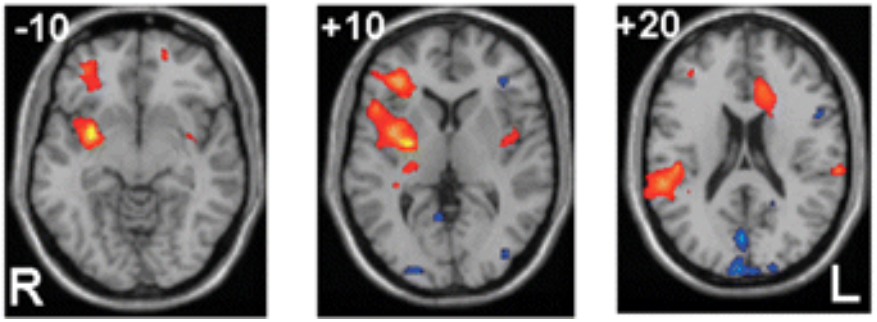
PIVC

- bilateral activation
- right hemispheric dominance (non-dominant hemisphere)
- interaction with primary visual cortex

Illustration of the normal activation-deactivation pattern during unilateral vestibular stimulation in healthy volunteers (activations in yellow-red, deactivations in blue)







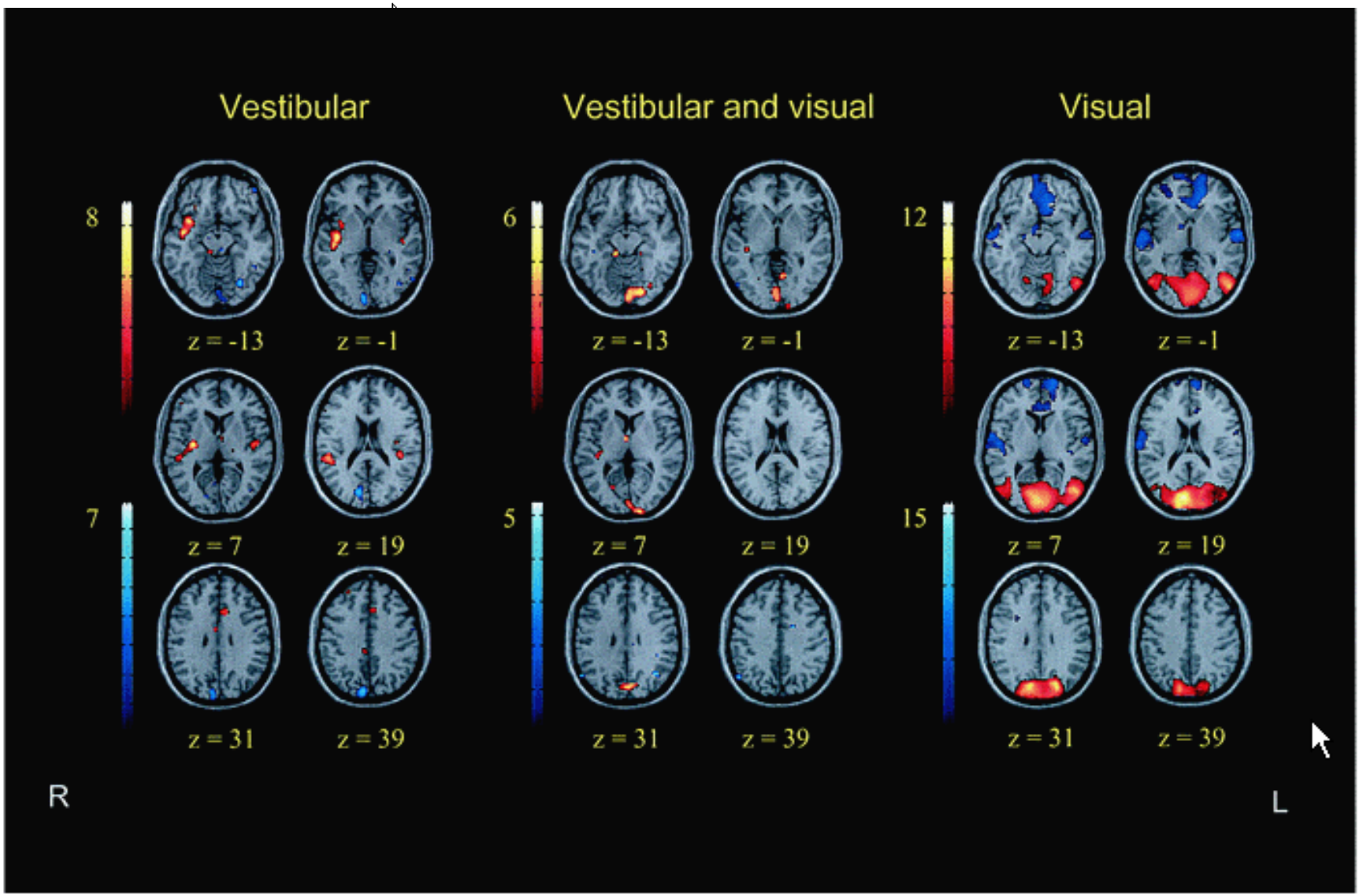
Dieterich, M. et al. Brain 2008 131:2538-2552

Self-motion perception

Mechanism of reciprocal inhibitory visual-vestibular interaction

- optokinetic deactivates the vestibular cortex
- vestibular stimulation deactivates the visual cortex

FIGURE 1. Activation maps (rCBF increases and decreases in PET) showing activations (*red*) and deactivations (*blue*) during unimodal vestibular (**A**), simultaneous (**B**), and unimodal visual (**C**) stimulation versus rest condition (**D**) (group analysis: $n = 13$; $p < 0.001$), superimposed onto the MNI standard brain template. Caloric vestibular stimulation activates the parietoinsular vestibular cortex and deactivates the visual cortex. Visual motion stimulation activates the visual cortex and deactivates the vestibular cortex. During simultaneous visual-vestibular stimulation, activations and deactivations are smaller.



Brandt et al. Ann N.Y. Acad Sci 956: 230-241 (2002)

Reciprocal inhibitory visual-vestibular interaction

- prevents sensory mismatch and perceptual ambiguity

- allows the brain to link the perception of self-motion to the dominant input:

  vestibular (head acceleration)
  visual (constant velocity)
  both

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Bettlach, 27 août 2016

Management of vertigo

PD Dr Raphaël Maire
Unité d'Otoneurologie et Audiologie
Service d'ORL, CHUV, Lausanne

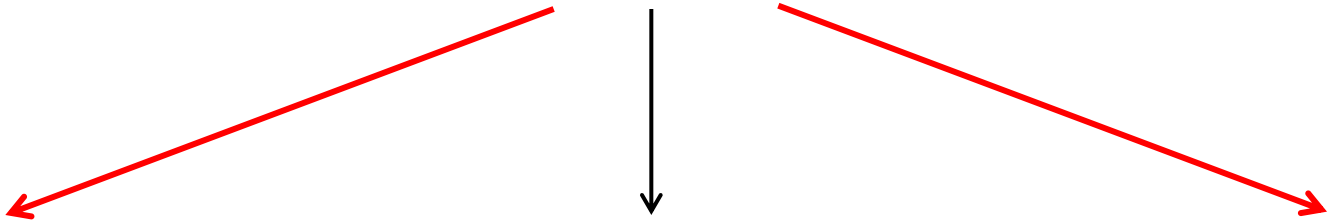
Acute vertigo : main diagnosis

- Benign paroxysmal positional vertigo
- Acute unilateral peripheral vestibular loss
- Menière's attack
- Orthostatic hypotension
- Transient ischemic attacks, Stroke
  - brainstem, cerebellum
- Head trauma
  - temporal bone fracture, perilymph fistula
- Intoxication (drugs, alcohol)

Chronic (*or recurrent)* vertigo : main diagnosis

- uncompensated unilateral peripheral vestibular loss
- Menière's disease
- recurrent vestibulopathy (vestibular Menière?)
- vestibular migraine
- fistula / superior canal dehiscence syndrome
- tumor (vestibular schwannoma, meningioma, glioma)
- vestibular paroxysmia (neuro-vascular compression)
- bilateral vestibulopathy
- post traumatic syndrome
- central vestibular (neurological), drugs side effects
- immune-mediated inner ear diseases, metabolic disorders
- psychophysiologic (sensory ambiguity perception)
  - space and motion discomfort, chronic subjective dizziness, visual vertigo
  - anxiety, phobic disorder

Origin of vertigo



Peripheral	*other*	**central**
Labyrinth and/or VII nerve	*motor* *hypotension* *psychogenic*	cerebellum and/or brainstem

Evaluation of the dizzy patient

History

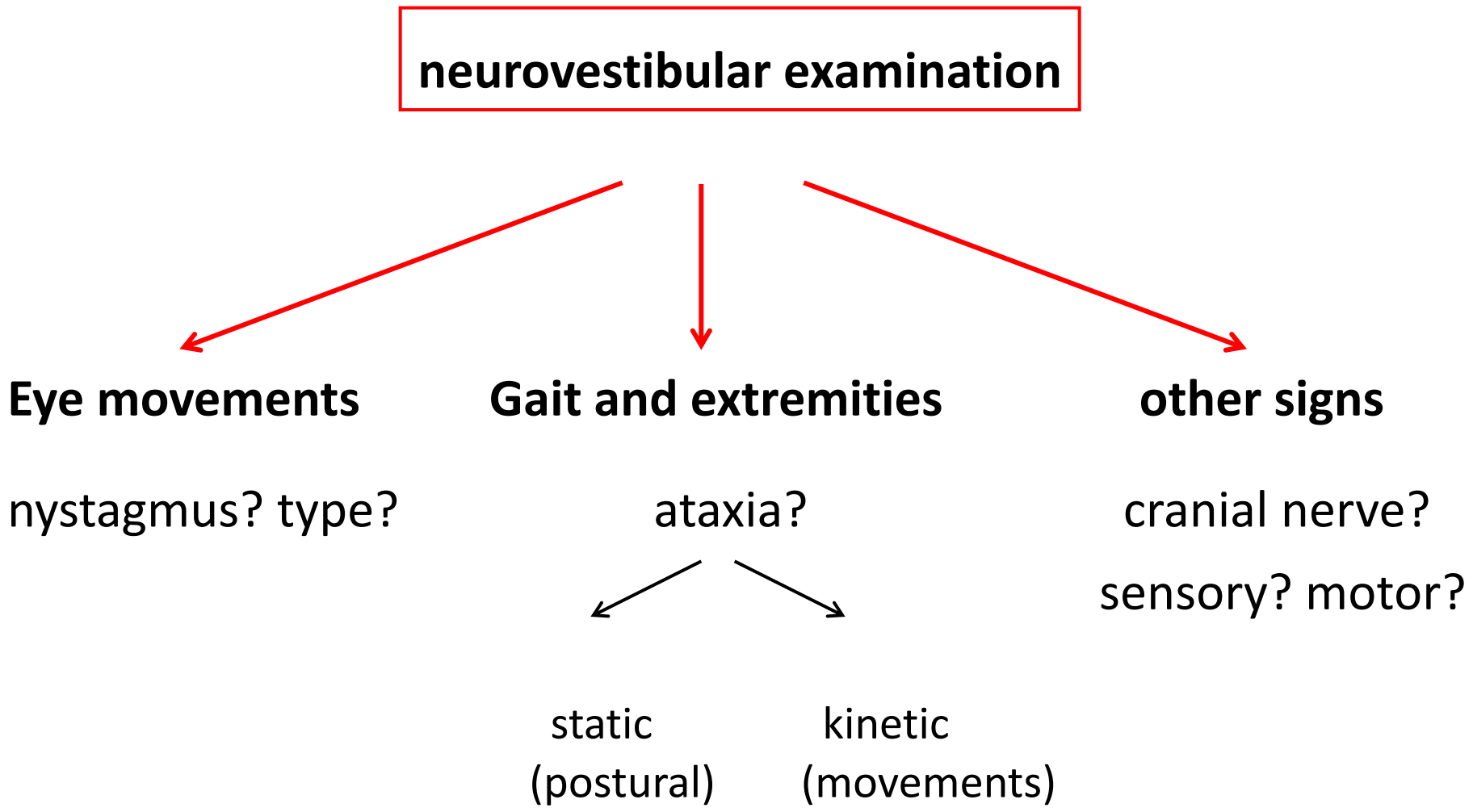
Examination of the vestibular system

- ear examination, including hearing test
- static posture, walking tests
- neurological evaluation
- labyrinthine evaluation

} neurovestibular examination

- ± cardiovascular examination

Evaluation of the dizzy patient



Neurovestibular examination

1) Neurological evaluation

- cranial nerves
- cerebellar tests
- sensory /motor deficit
- state of consciousness

Neurovestibular examination

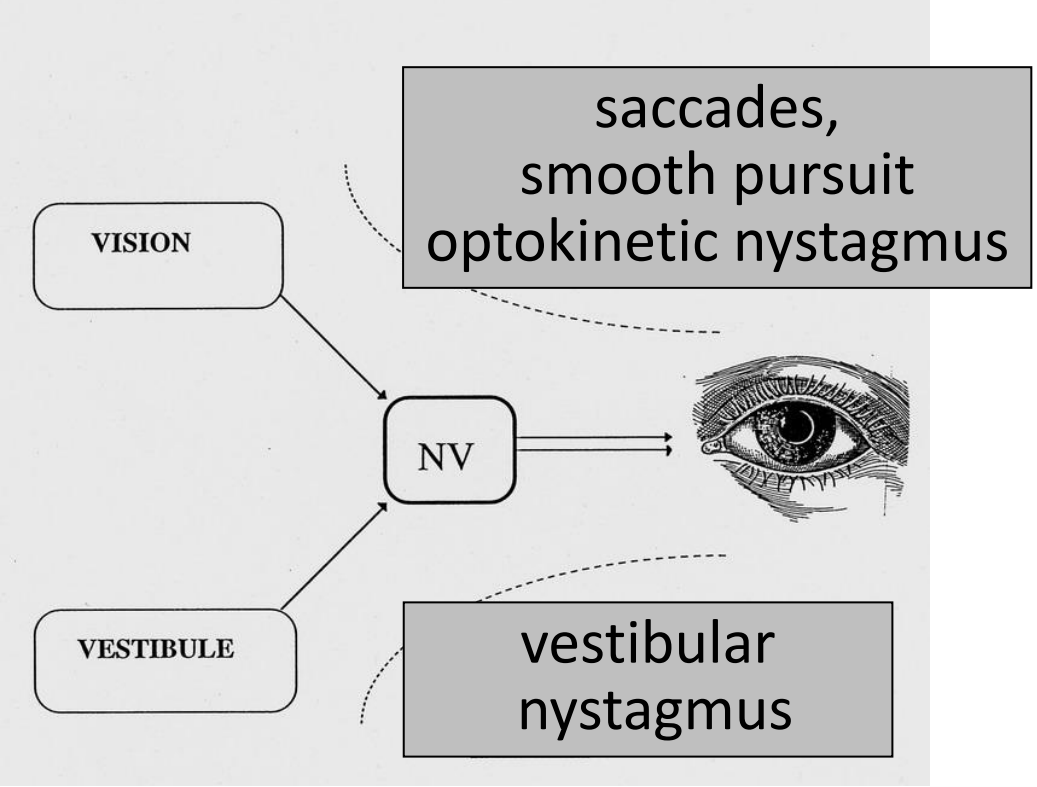
2) Balance (vestibulospinal reflexes)

- Romberg
- Unterberger (Fukuda)
- walking tests

Neurovestibular examination

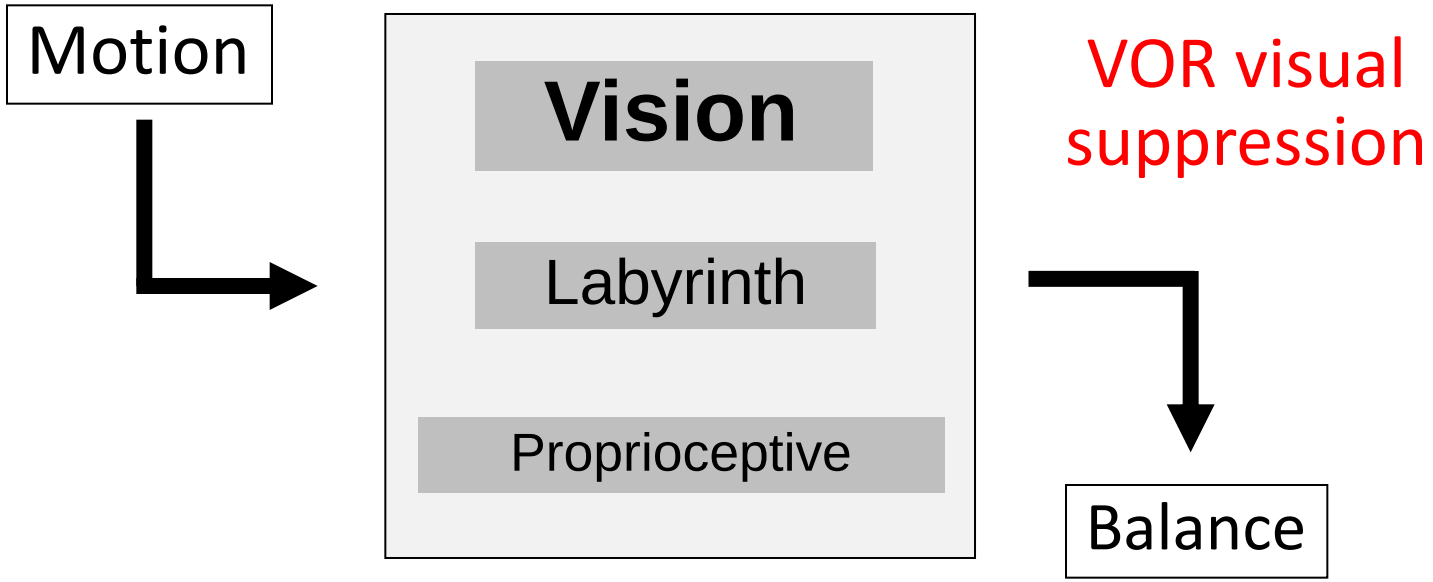
3) Visual-vestibular pathways

Vestibulo-ocular reflex
Optokinetic reflex



Visual-vestibular interaction: hierarchy

- low frequency (<1Hz) : vision > labyrinth



- high frequency (1–4Hz) : labyrinth > vision

Visual-vestibular pathways examination

- at light
- in condition preventing fixation
  - darkness
  - Frenzel glasses
  - infrared videoscopy

Consider visual suppression of vestibular nystagmus

Neurovestibular examination

4) Labyrinthine evaluation

- bithermal binaural caloric testing
  ⟶ low frequency VOR evaluation (<0.1Hz)
- Head-thrust test (Halmagyi)
  ⟶ high frequency VOR evaluation (>1Hz)
- fistula/dehiscence test (Politzer, Valsalva, Tullio)
- rotational testing

Types of pathologic nystagmus

- spontaneous (eyes in primary position)
- gaze-evoked
- head-shaking
- positional / positioning
    - direction fixed, direction changing
- congenital

Pathologic nystagmus

origin	characteristics
peripheral	conjugate (binocular) ↓ by fixation
central	conjugate / disconjugate not ↓ or ↑ by fixation

Spontaneous nystagmus (primary gaze)

- peripheral
  - combined horizontal-torsional, unidirectional
  - asymmetric loss of peripheral vestibular tone (labyrinth or vestibular nerve)

- central
  - often pure horizontal, vertical or torsional
  - may change direction
  - imbalance in central vestibular tone (brainstem or cerebellum)

Acute peripheral vestibular loss

- Vestibular neuritis (idiopathic)
- Labyrinthitis or neurolabyrinthitis
  - herpes zoster oticus
  - complication of otitis media
- Labyrinthine concussion
  - head trauma, temporal bone fracture
- Labyrinthine infarction

Acute peripheral vestibular loss

Effect on the vestibular system

- acute imbalance in central vestibular tone (vestibular nuclei)
- oculomotor, postural and perceptual deficits

Acute peripheral vestibular loss

Symptoms :

- prolonged rotatory vertigo (days)
- perceptual tilt towards affected side
- postural deviation towards affected side
- nausea ± vomiting

Acute peripheral vestibular loss

Clinical signs :

- spontaneous nystagmus towards healthy ear, ↓ by fixation
- head tilt towards affected ear
- tonic eye deviation towards affected ear
- postural deviation towards affected ear
- partial/total caloric weakness of affected ear
- pathologic head thrust towards affected ear (Halmagyi sign)
- no central neurological signs

Acute peripheral vestibular loss

spontaneous

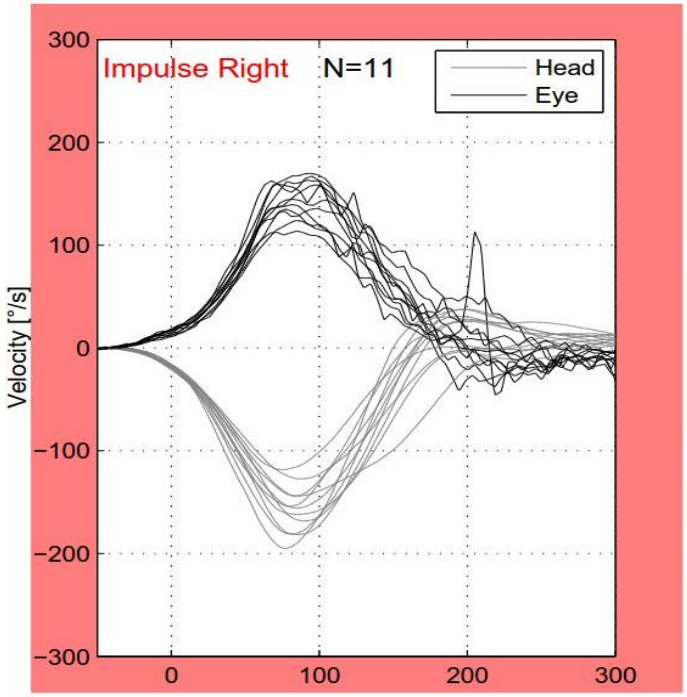
nystagmus

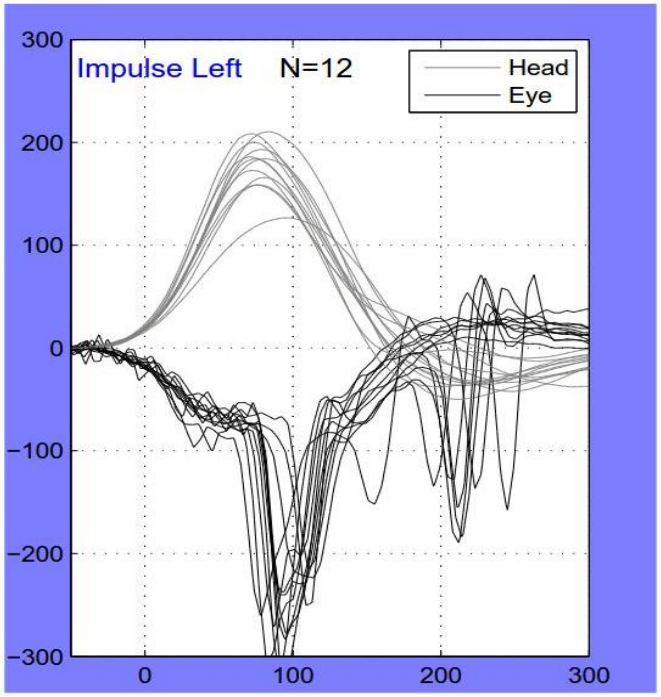
beating

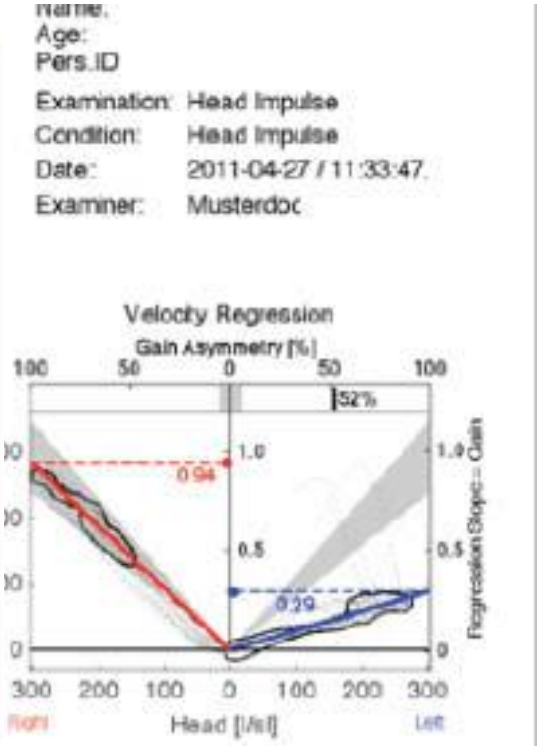
towards healthy side

Pathologic Head Impulse Test (Halmagyi sign)

Video Head Impulse Test







Acute peripheral vestibular loss

Evolution :

- decrease of signs and symptoms with central compensation (3 to 6 weeks)

→ recovery of central vestibular tone (vestibular nuclei)

→ re-equilibration of low frequency VOR (velocity storage)

Central vestibular compensation

Lacour M 1992 – 2010

maximum compensation is reached within 3-12 months

- stimulated by movement and fastened by betahistine
- reduced by immobility and vestibular sedation

STATIC components of compensation are good (at rest)

- powerful neuroplasticity in VN

DYNAMIC components of compensation are poor (during movement)

- impaired automatization of gait and balance
- impaired image stabilization
- hypersensitivity to optokinetic stimuli
- impaired spatial orientation

Effect of betahistine on central compensation after vestibular neurectomy

Cats *Tighilet B et al (jan. 1995) J Vestibular Research*

placebo: recovery in 6 weeks*

betahistine (50mg/kg/day): recovery in 3 weeks*

*posture and locomotor balance

Meniere patients *Redon C et al (apr. 2011) J Clin Pharmacology*

placebo: recovery in ≥ 3 months

betahistine (2x24mg/day): recovery in ≈ 1 month*

* postural stability, subjective visual vertical, head orientation

Acute peripheral vestibular loss

General treatment recommendations

- mobilization and rehabilitation as soon as possible
- avoid immobility and vestibular sedation
- betahistine 48-72mg/day 4-12 weeks
- ± prednisone 1mg/kg/day 7-10 days (according to authors)

Acute peripheral vestibular loss

Evolution :

- if no recovery of canal loss :

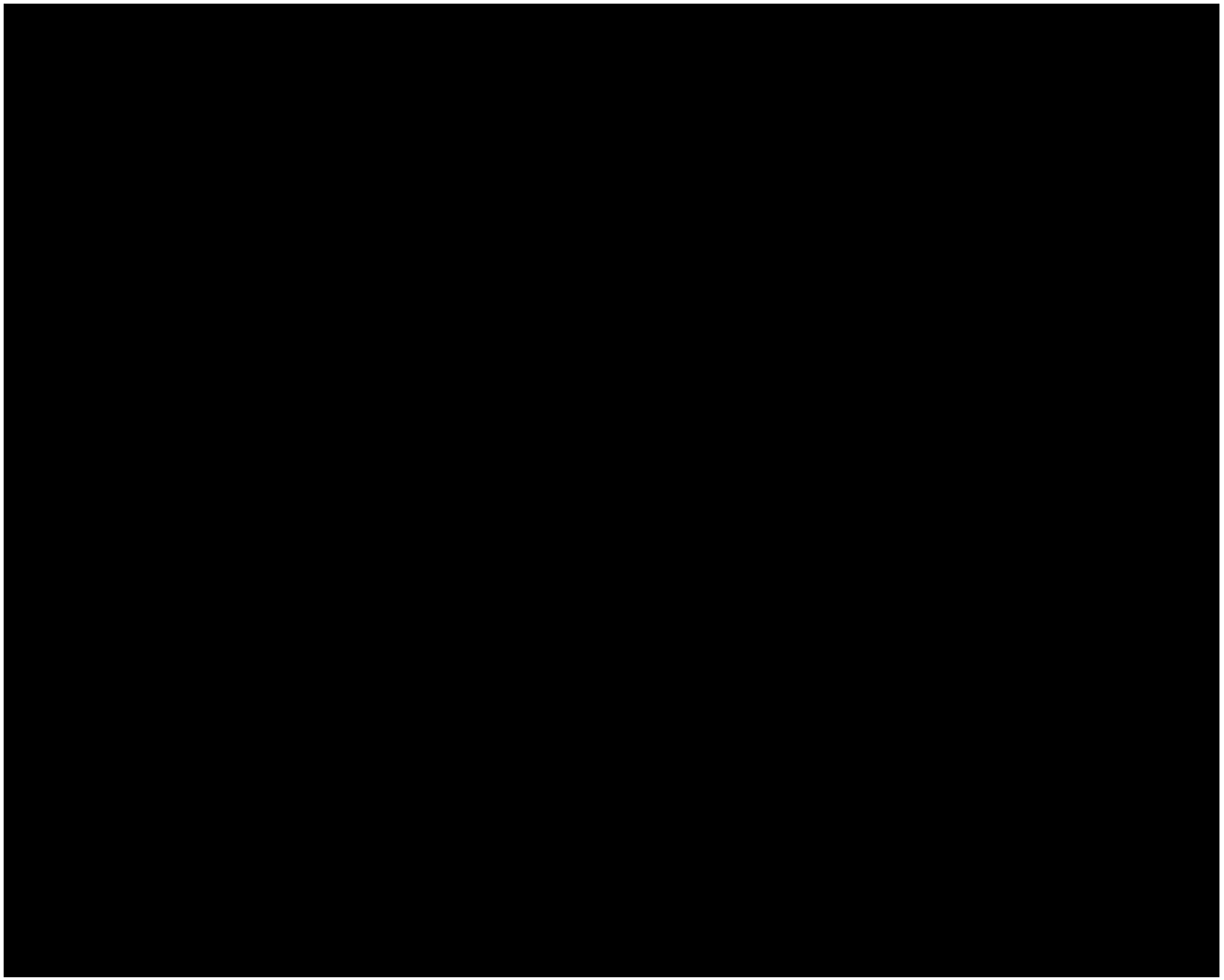
  persisting high frequency VOR deficit towards affected side (Halmagyi sign)

→ oscillopsia, reduced dynamic visual acuity

mechanism : retinal slip due to VOR loss

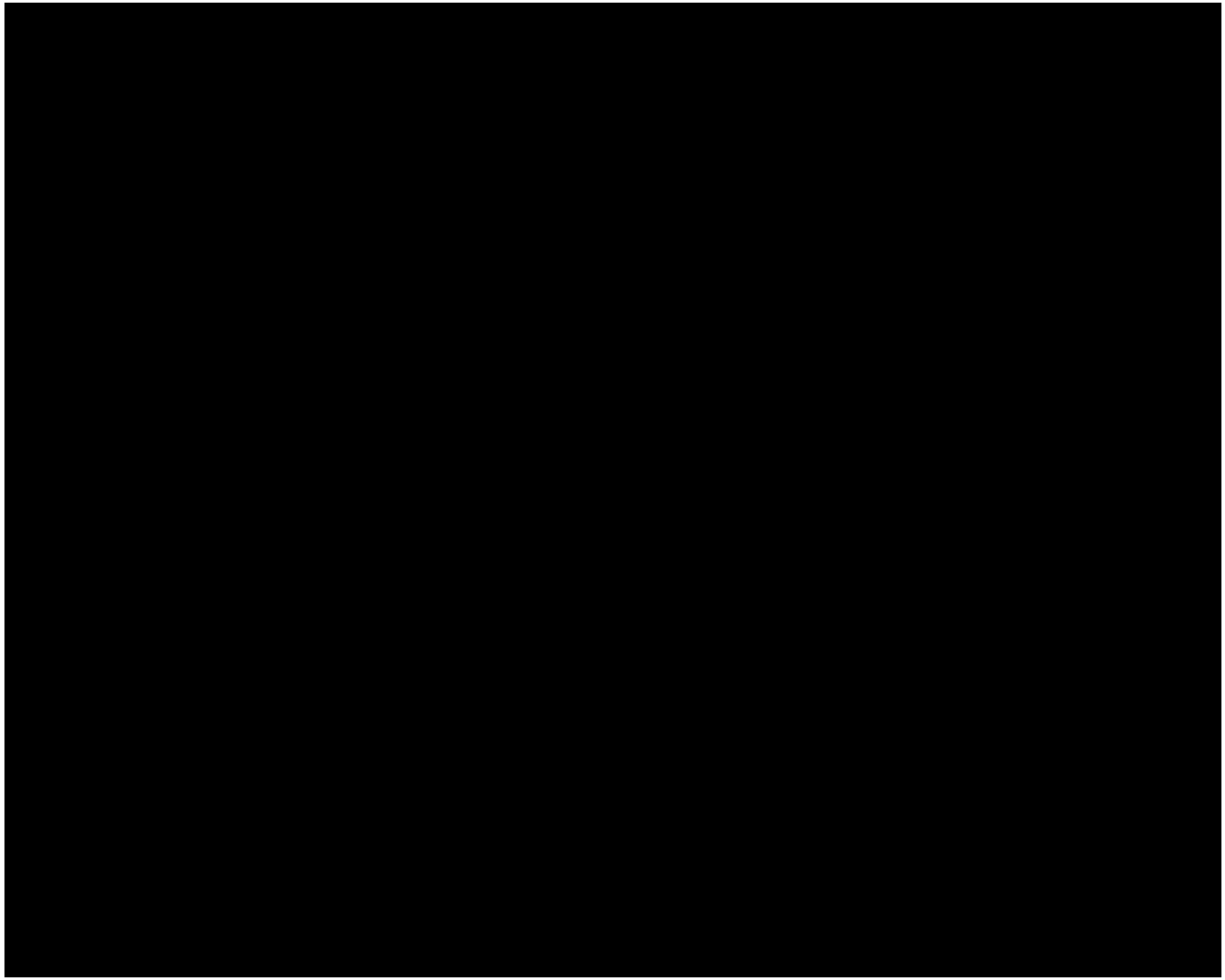
Central (visuo)vestibular deficit

Lack of visual suppression of nystagmus



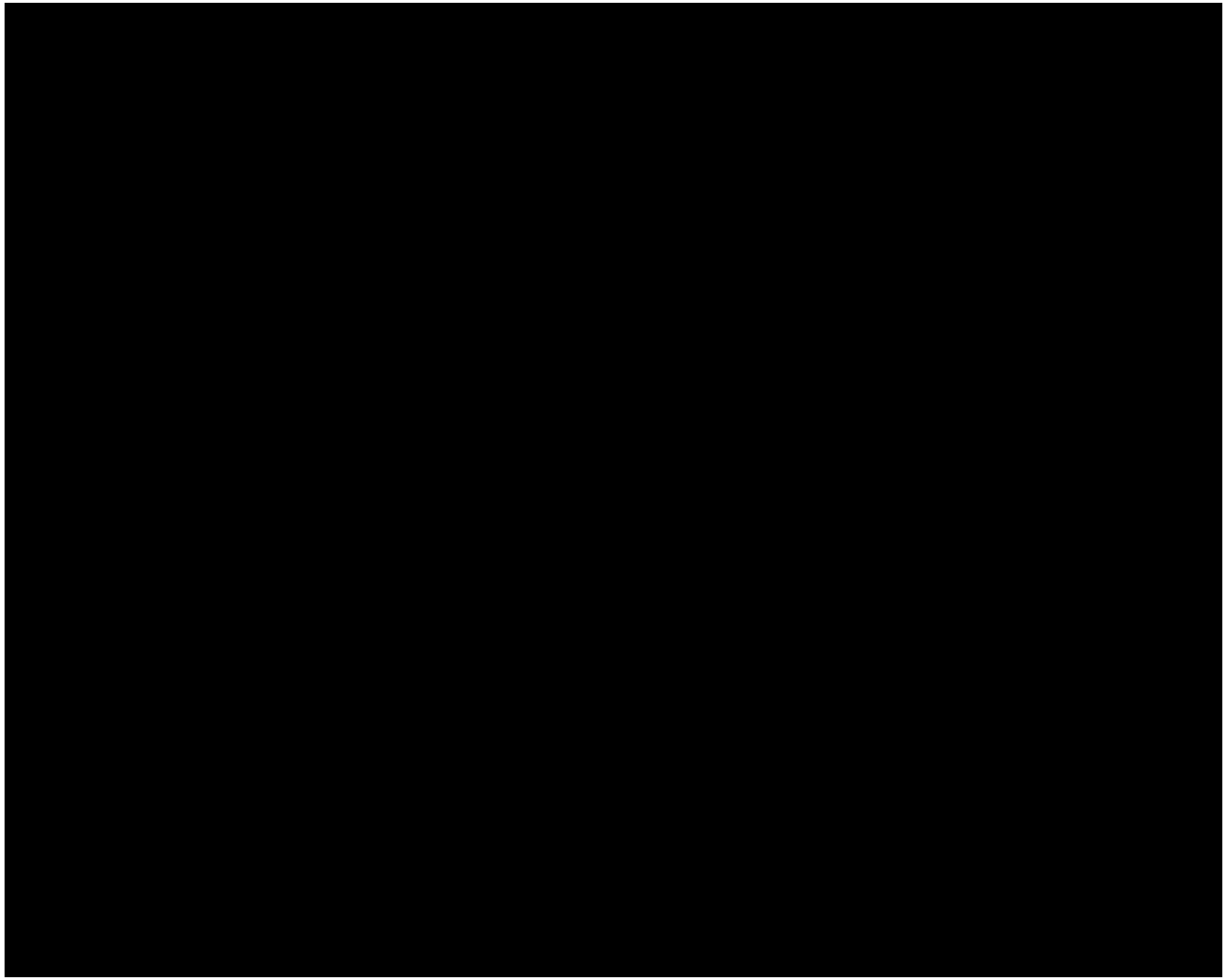
Central (visuo)vestibular deficit

Upbeat nystagmus



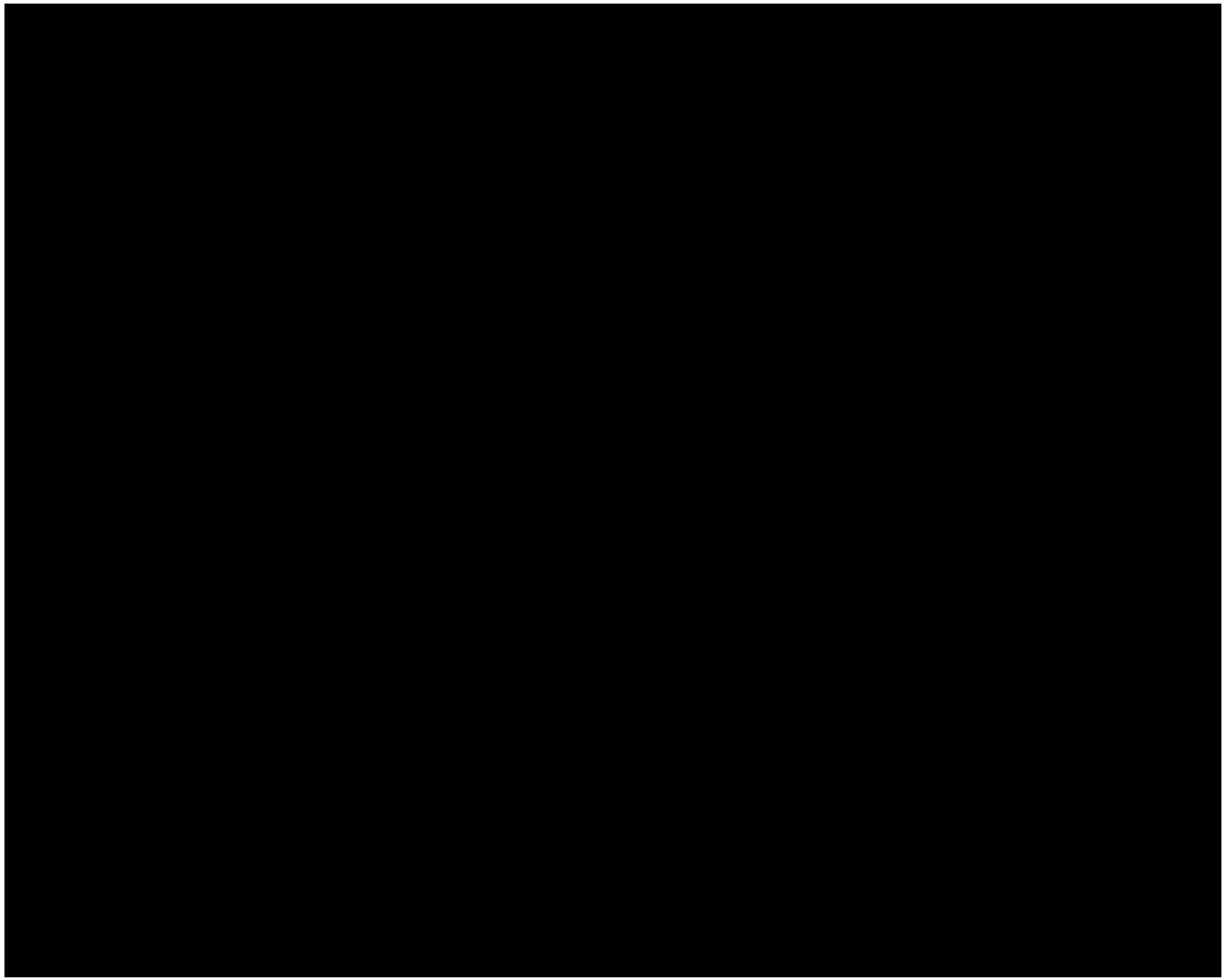
Central (visuo)vestibular deficit

downbeat nystagmus



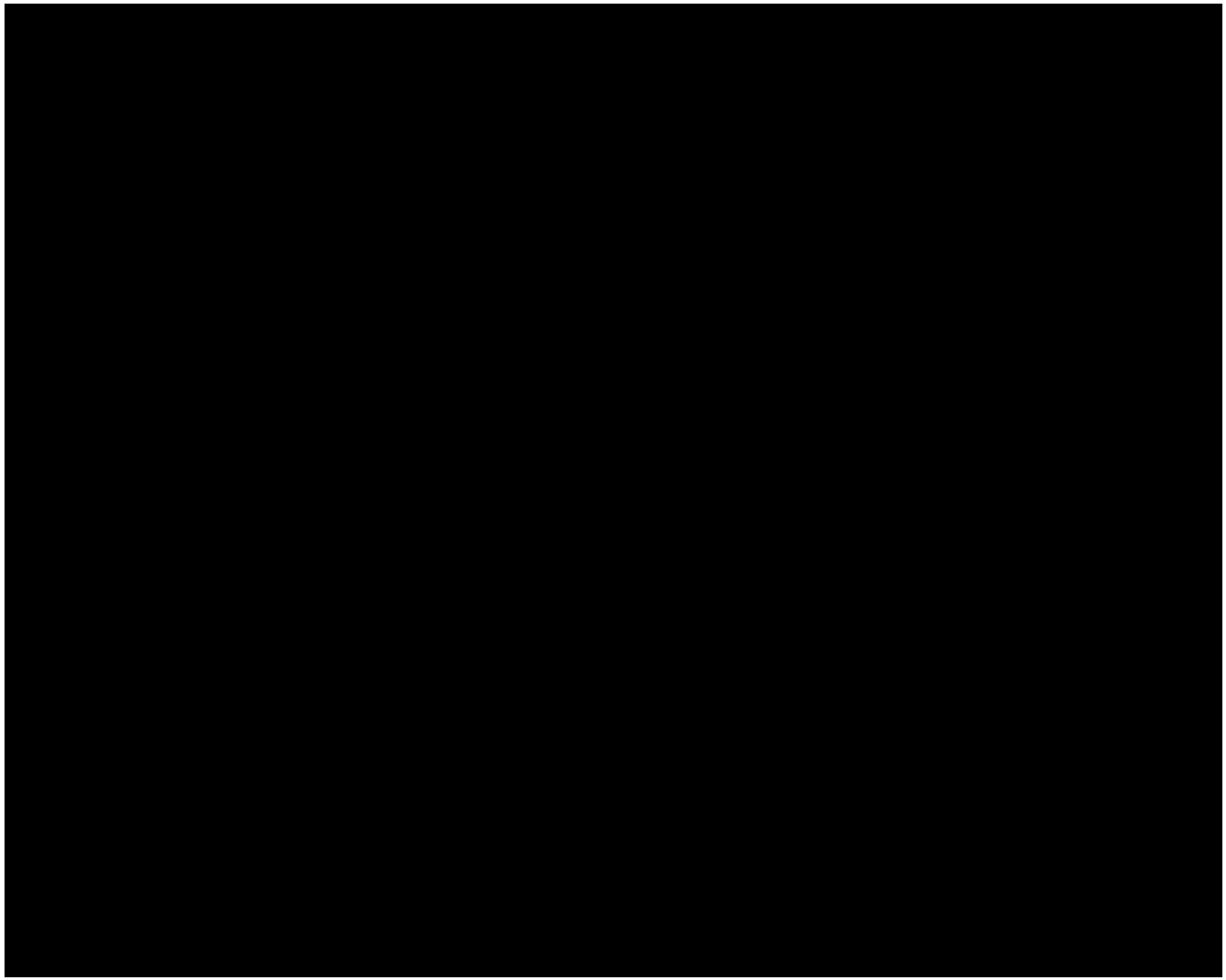
Central (visuo)vestibular deficit

Saccadic hypermetria



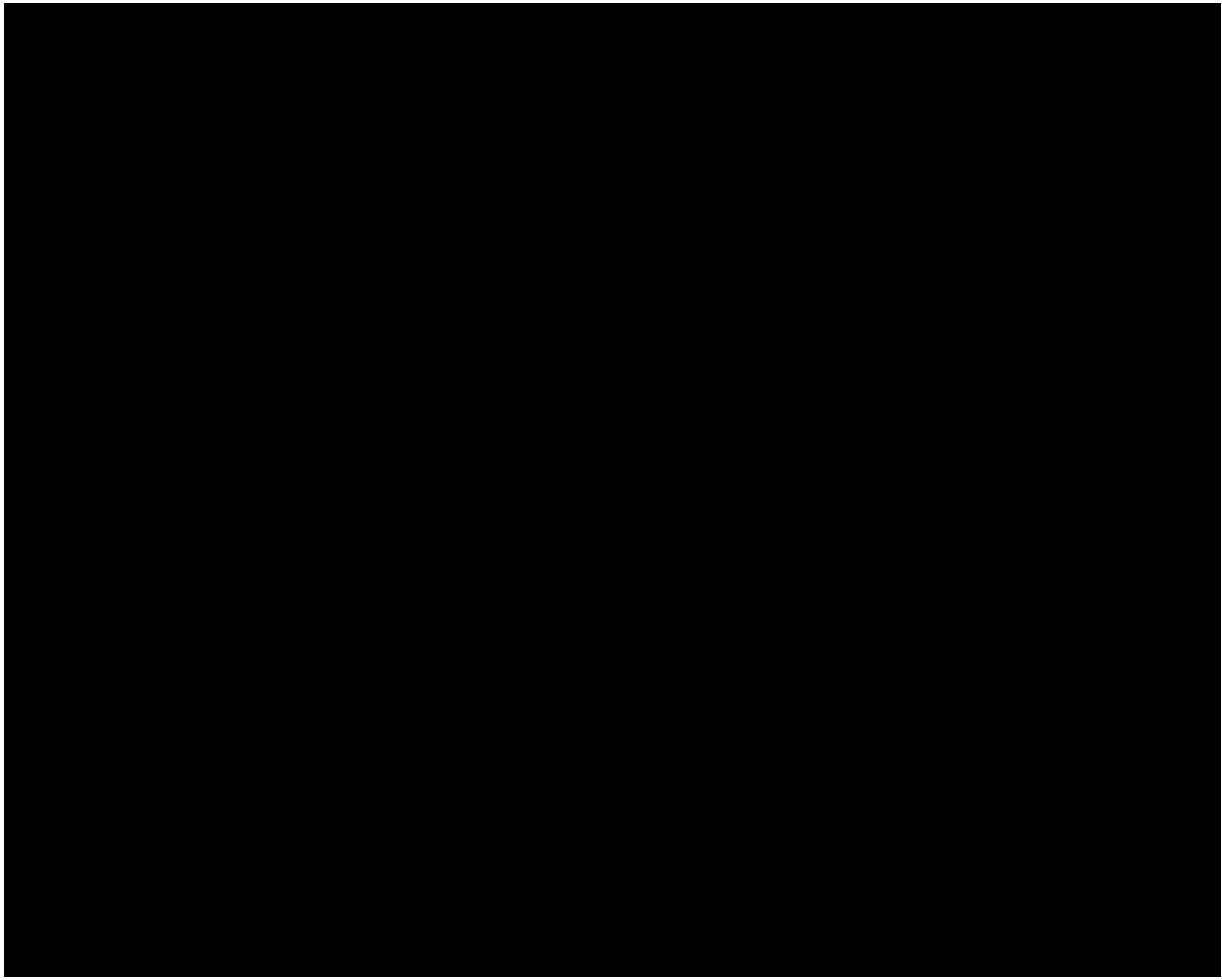
Central (visuo)vestibular deficit

Ocular flutter / opsoclonus



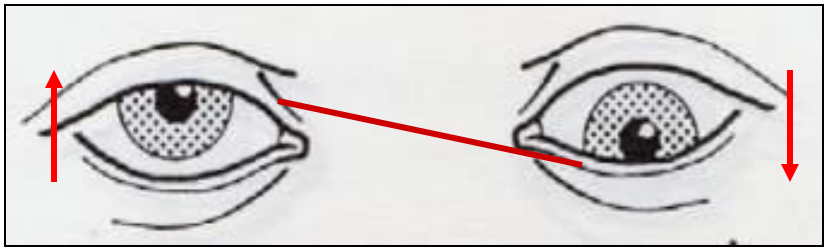
Central (visuo)vestibular deficit

Skew deviation detected by alternate cover test

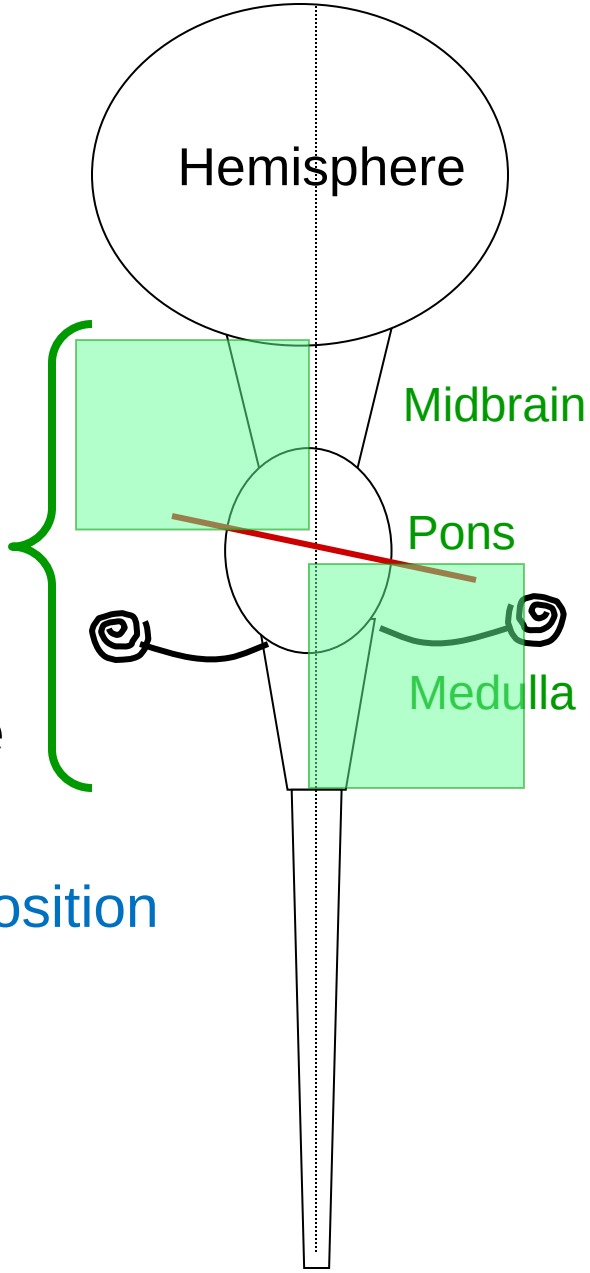


Skew deviation:

How to localize the lesion



Elevation one eye Depression other eye

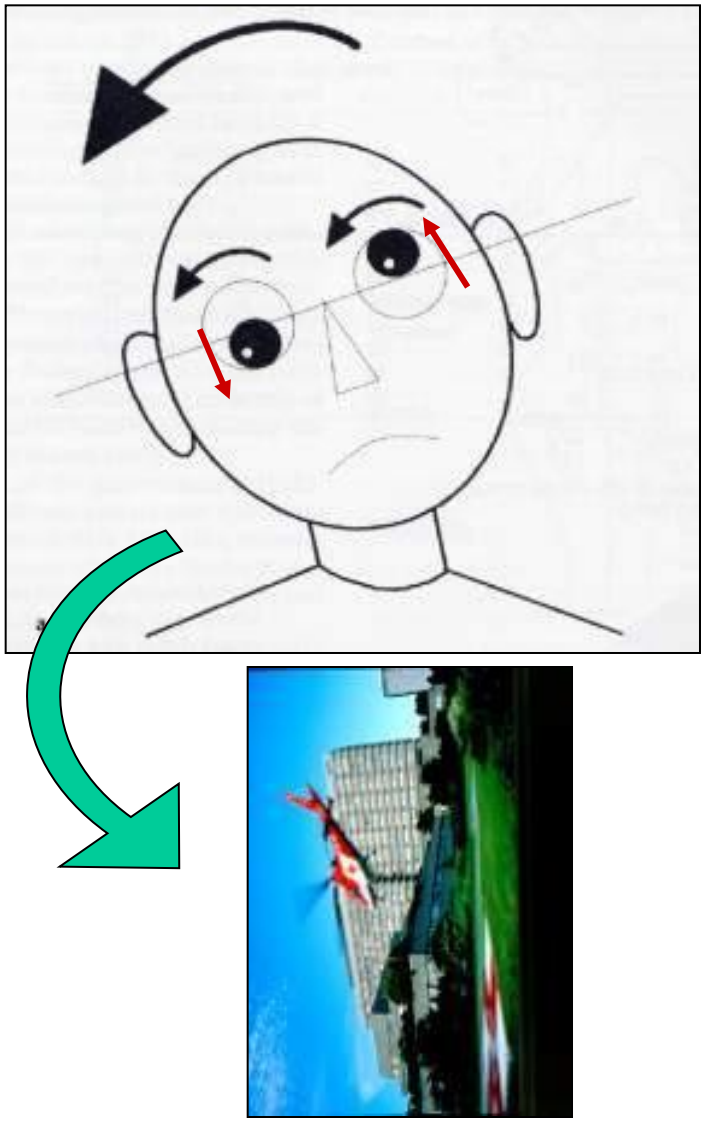


1. Draw an oblique line according to eye position
2. Put this line into the mid-pons
3. Lesions are above the higher part
   or below the lower part of the line

Ocular tilt reaction (OTR)

- Consists of
  1. skew deviation
  2. head lateroflexion
  3. ocular torsion

  (all to the same side)
- May be associated with visual tilt (of the image)



Acute spontaneous vertigo: peripheral or central

Keys for differential diagnosis

Stroke in brainstem and / or inferior cerebellum

HINTS to Diagnose Stroke in the Acute Vestibular Syndrome: Three-Step Bedside Oculomotor Examination More Sensitive Than Early MRI Diffusion-Weighted Imaging . Jorge C. Kattah et al. *Stroke 2009;40;3504-3510*

HINTS : Head impulse – **N**ystagmus – **T**est of **S**kew

normal Head impulse Test both sides

+ direction-changing spontaneous nystagmus or gaze evoked nystagmus

+ skew deviation

→ 100% sensitivity and 96% spécificity for a stroke of the posterior fossa

Inferior vestibular neuritis

J Neurol (2012) 259:1553–1560 Kim JS. Inferior vestibular neuritis.

Table 2 Diagnostic criteria of isolated inferior vestibular neuritis

1	Acute vertigo with nausea/vomiting and imbalance
2	Spontaneous torsional downbeat nystagmus
3	Abnormal head impulse test for the posterior semicircular canal
4	Abnormal cervical vestibular-evoked myogenic potential
5	Normal head impulse tests for the anterior and horizontal semicircular canals
6	Normal calorics
7	Exclusion of central pathologies using neurological examination and brain MRI

Diagnostic criteria of Menière's disease

Definite MD

A. Two ore more spontaneous episodes of vertigo each lasting 20 minutes to 12 hours
B. Audiometrically documented low-to medium frequency sensorineural hearing loss in one ear, defining the affected ear on at least one occasion
C. fluctuating aural symptoms (hearing, tinnitus or fullness) in the affected ear
D. Not better accounted for by another vestibular diagnosis

Lopez-Escamez et al., J vestib Res 25 (2015) 1-7

Diagnostic criteria of Menière's disease

Probable MD

A. Two ore more episodes of vertigo or dizziness, each lasting 20 minutes to 24 hours

B. Fluctuating aural symptoms (hearing, tinnitus or fullness) in the affected ear

C. Not better accounted for by another vestibular diagnosis

Lopez-Escamez et al., J vestib Res 25 (2015) 1-7

Diagnostic criteria of Vestibular Migraine

Vestibular Migraine

A. At least 5 episodes with vestibular symptoms lasting 5 min to 72 hours
B. Current or previous history of migraine with or without aura according to the International Classification or Headache disorders (ICHD)
C. One ore more migraine features (one sided location, pulsating qualitiy, photo-phonophobia, visual aura) with at least 50% of vestibular episodes
D. Not better accounted for by another vestibular or ICHD diagnosis

Lempert et al., J vestib Res 22 (2012) 167-172

Diagnostic criteria of Vestibular Migraine

Probable Vestibular Migraine

A. At least 5 episodes with vestibular symptoms lasting 5 min to 72 hours

B. Only one of the criteria B and C for vestibular migraine is fulfilled (migraine history *or* migraine features during the episode)

C. Not better accounted for by another vestibular or ICHD diagnosis

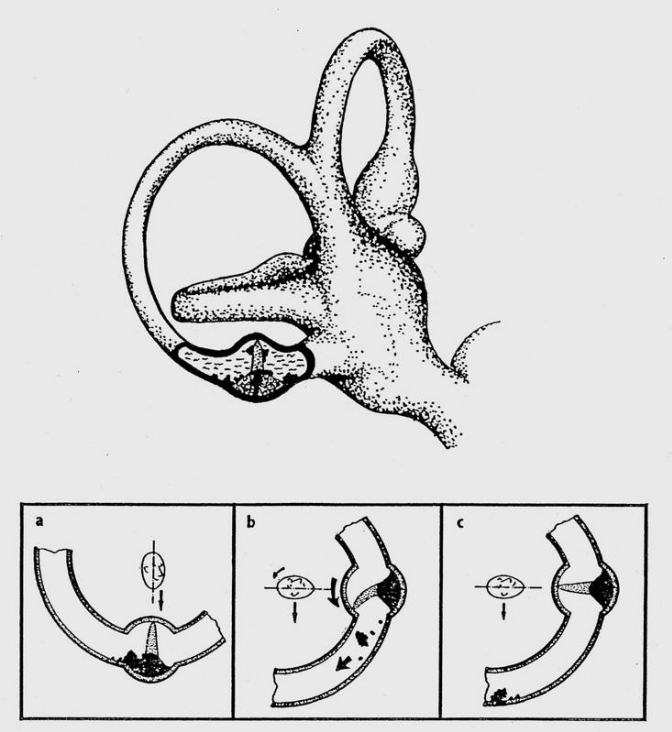
Lempert et al., J vestib Res 22 (2012) 167-172

Benign paroxysmal positional vertigo (BPPV)

Mechanism:
Displaced otoconia into semi-circular canal from otolith organs (utricle or saccule)

canalolithiasis:
Free floating debris in the canal

cupulolithiasis:
Debris attached to or very close to the cupula



BPPV

Most frequent cause of vertigo

Localization:	posterior canal :	80-90%
	horizontal canal:	10-20%
	anterior canal:	rare

Symptoms

brief episodes of vertigo (< 1min) with position change:

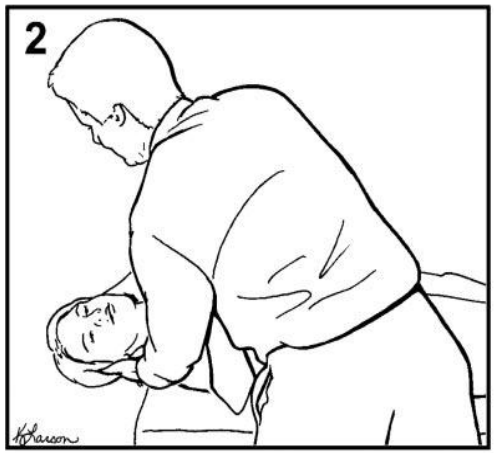
- getting in and out of bed
- turning over in bed
- straightening up
- extending the neck to look up

BPPV: etiology of canalo/cupulolithiasis

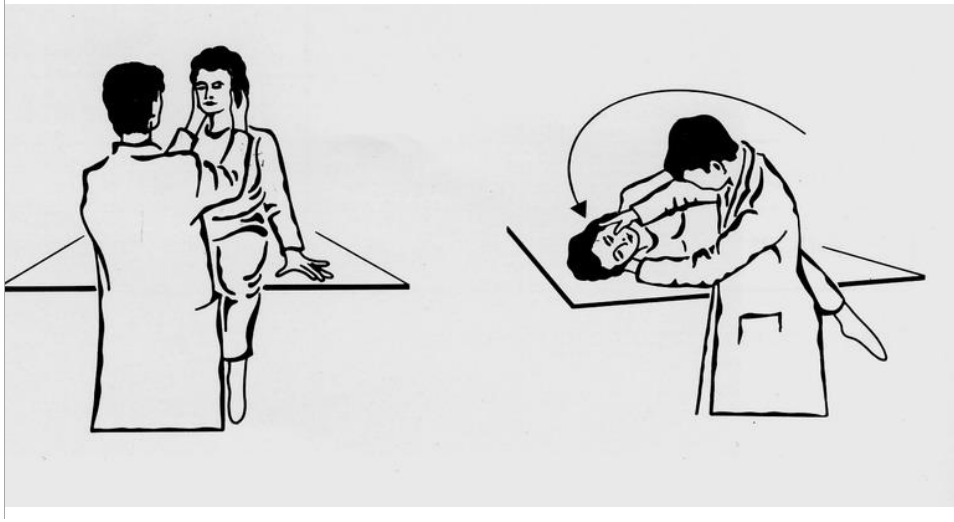
- disturbance in otolith metabolism
  (vascular, ageing, Ca^{2+} metabolism/osteoporosis)
- head trauma (otolith detachment)
- bed rest (clot formation in canals)
- vestibular neuritis, labyrinthitis
- ear surgery
- idiopathic (most frequent)

Posterior BPPV: diagnostic positioning manoeuvre

classical Dix-Hallpike

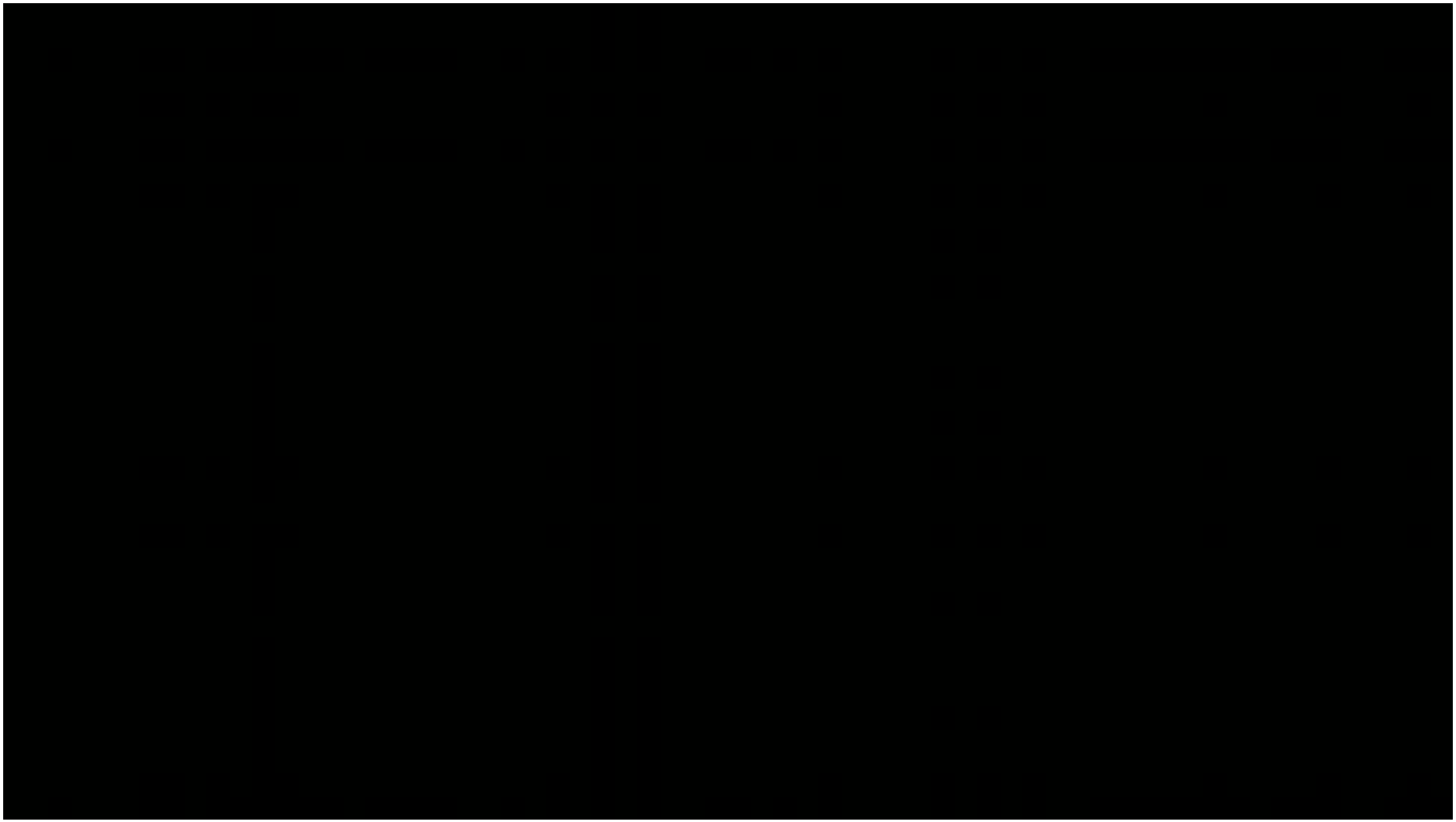


modified Dix-Hallpike

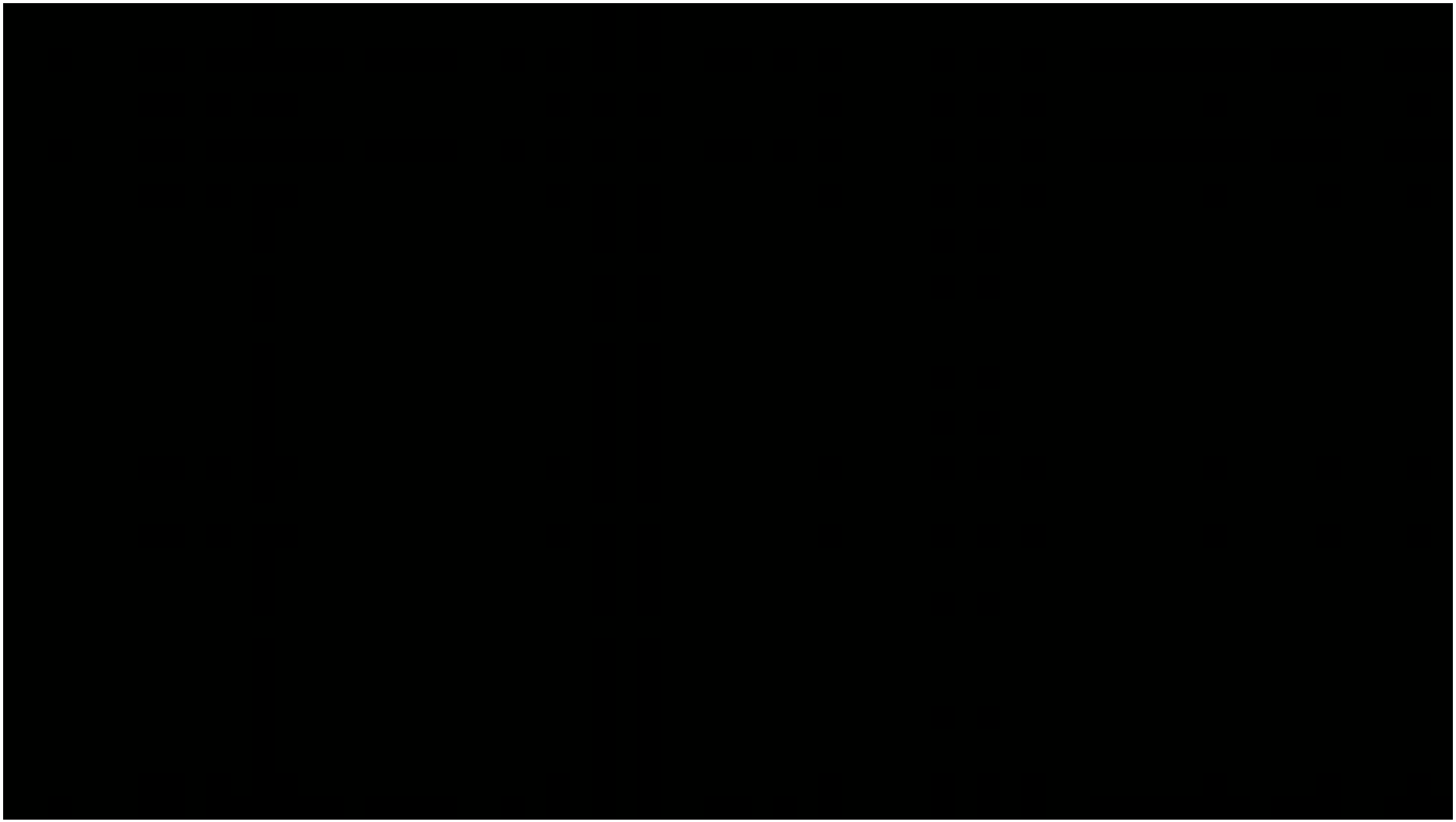


Also diagnostic manœuvre for contralateral *anterior*BPPV

PosteriorBPPV
classical Dix-Hallpike manoeuvre



PosteriorBPPV
modified Dix-Hallpike manoeuvre

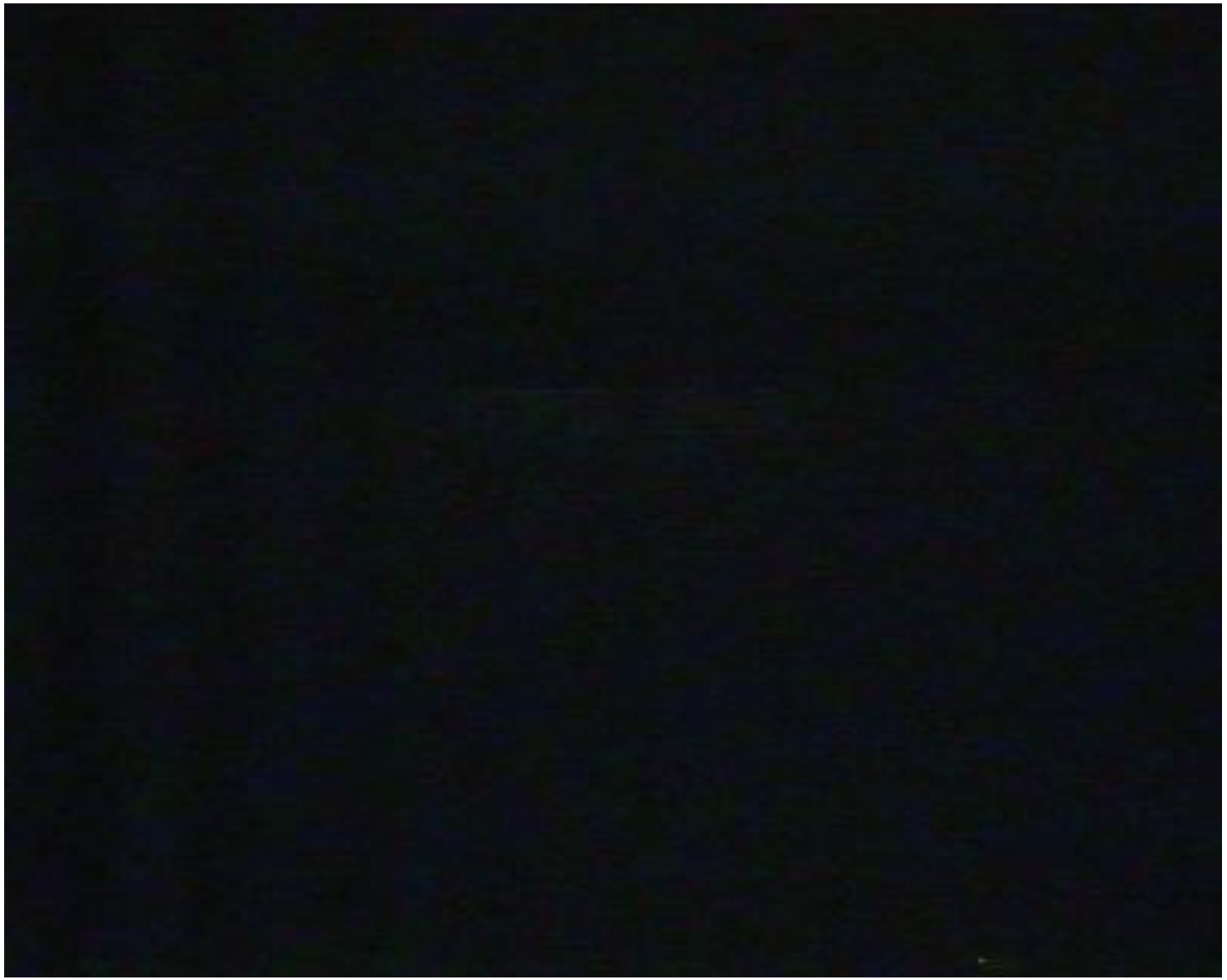


Posterior BPPV

Typical features:

- latency : 1-30 sec
- duration: < 1 minute (paroxysmal)
- characteristic positioning nystagmus: upward vertical and torsional
- fatigability: nystagmus decreases with repeated positioning

Right posterior BPPV



Posterior BPPV

Treatment

- Liberatory manoeuvre (Epley, Semont)

  move the patient around the plane of posterior canal

- Surgery
  - singular neurectomy (Gacek)
  - posterior canal occlusion (Parnes)

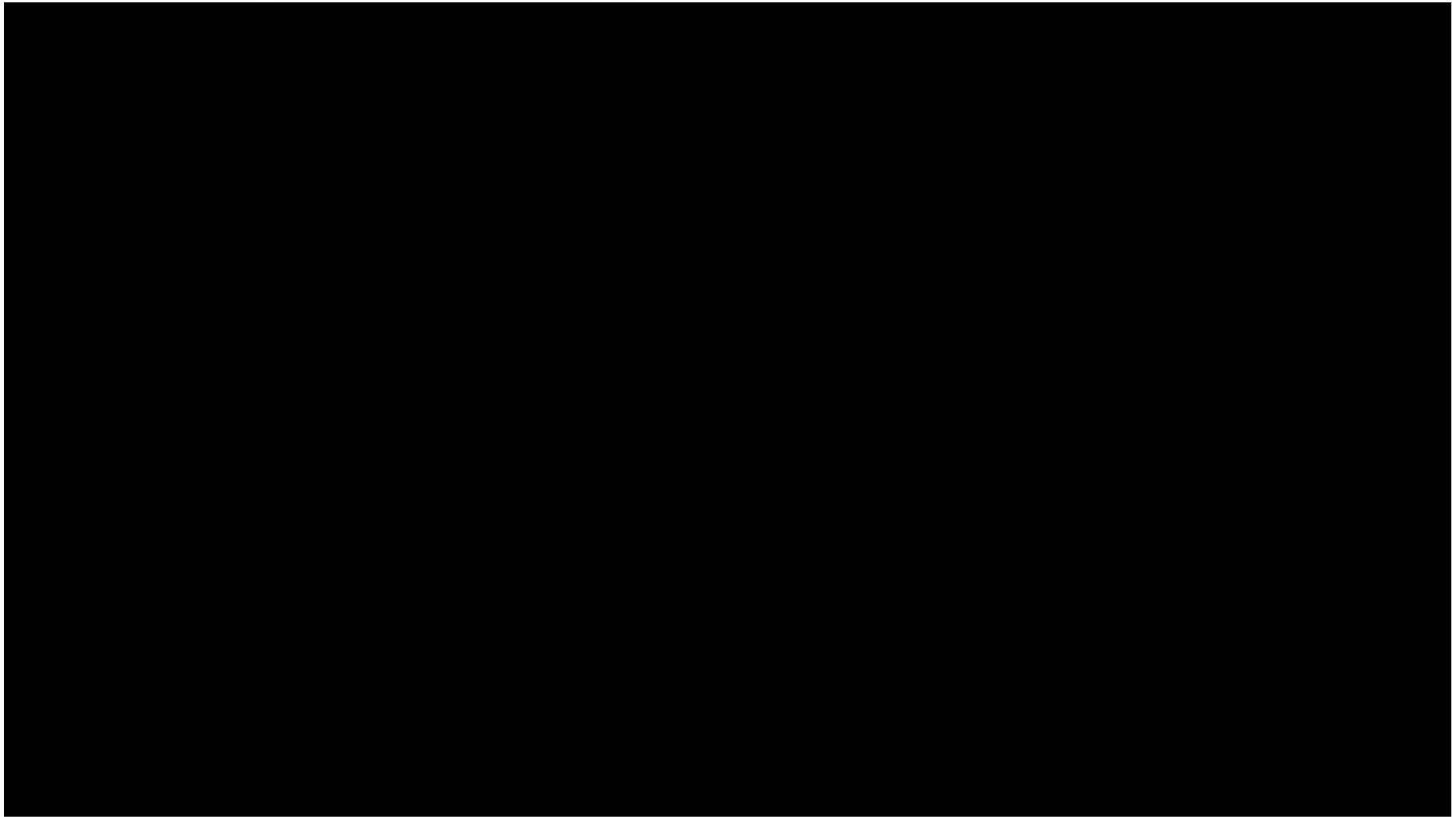
Evolution

- good prognosis

  most BPPV spontaneously disappear within 2 to 10 weeks

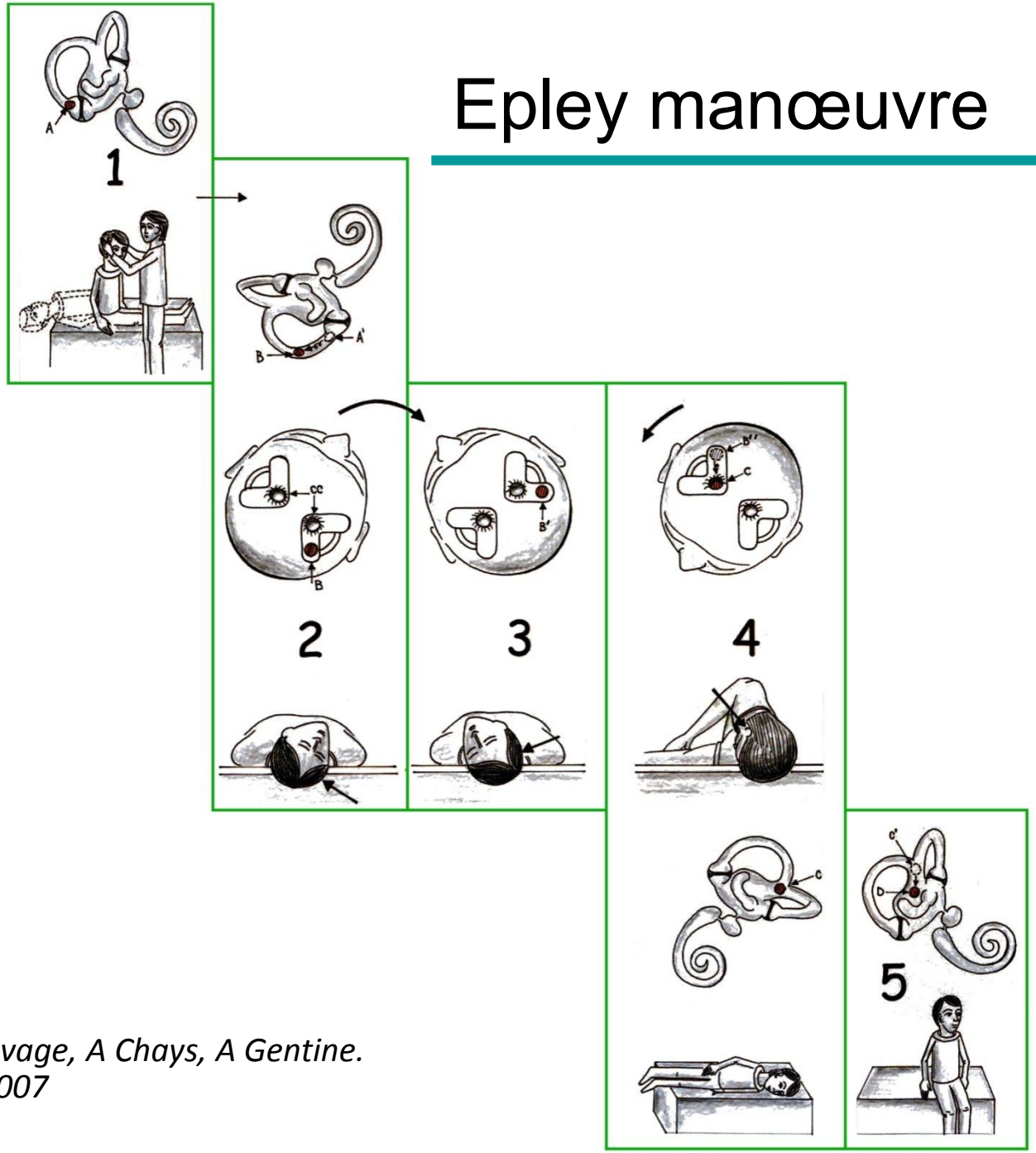
- common recurrences

*posterior*BPPV：Epley liberatory manoeuvre



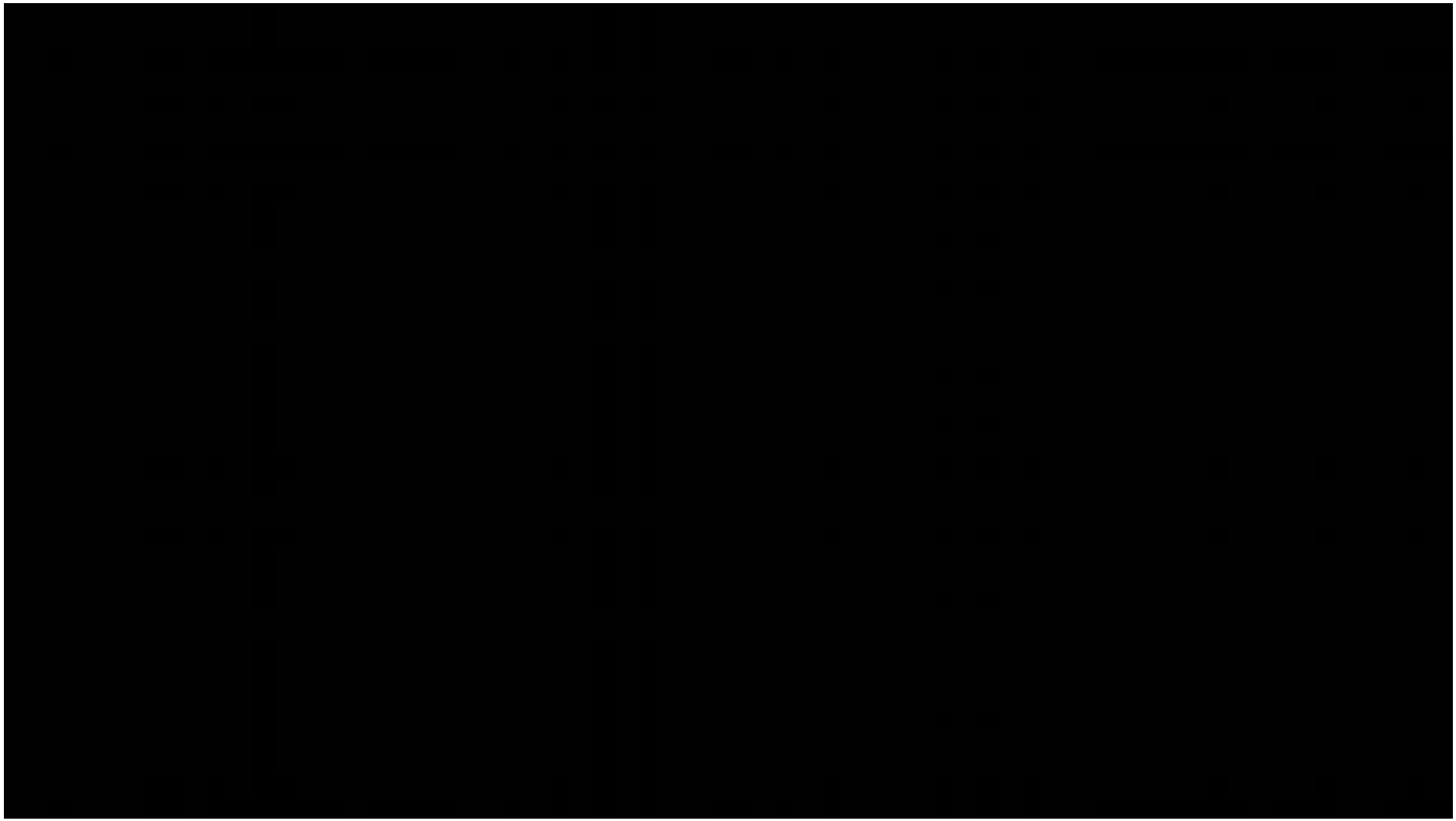
Posterior BPPV

Epley manœuvre

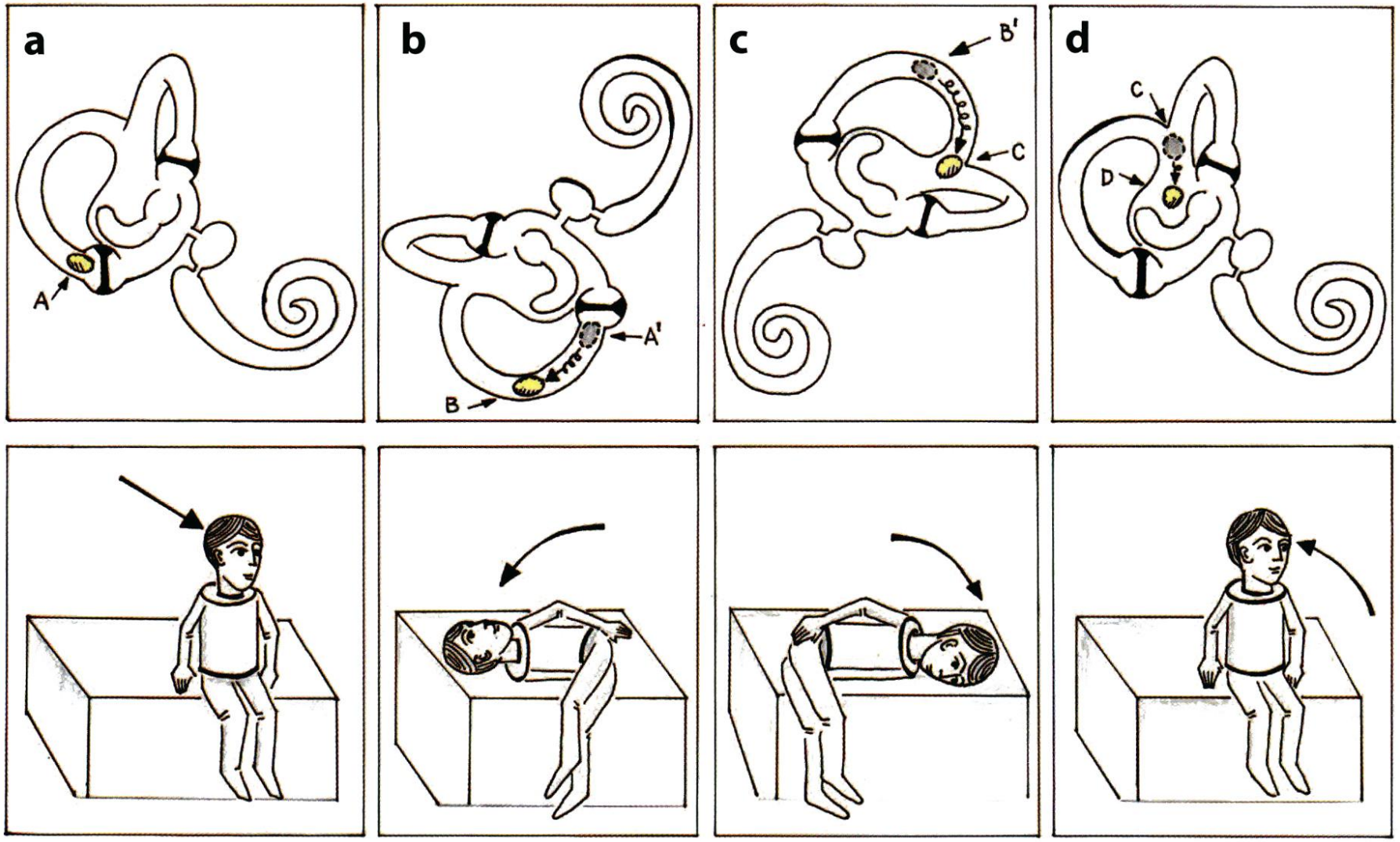


In: « vertiges positionnels » JP Sauvage, A Chays, A Gentine. Société française d'ORL, éditeur, 2007

*posterior*BPPV：Semont liberatory manoeuvre



Posterior BPPV: Semont manoeuvre

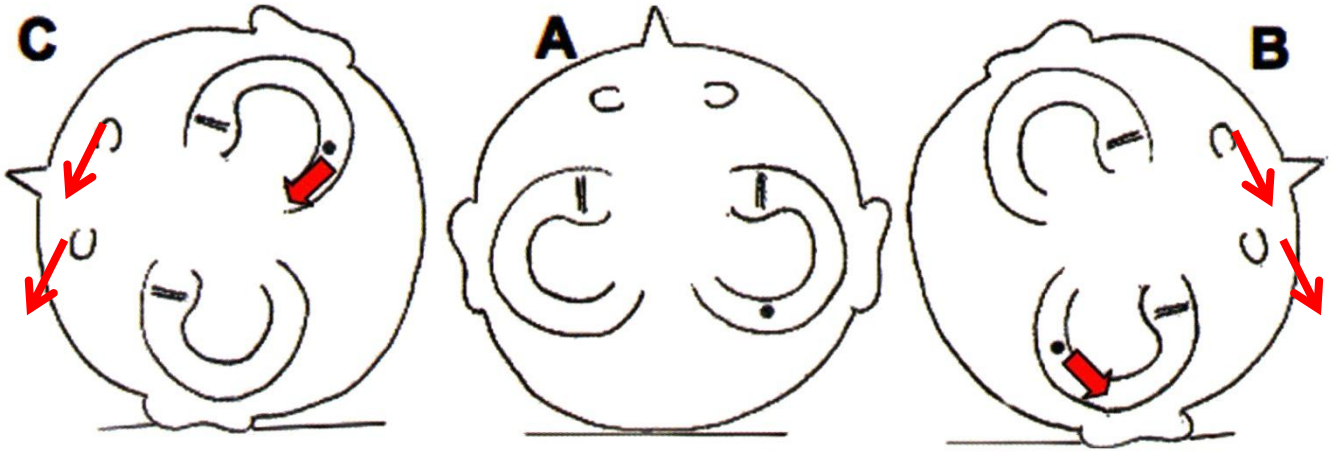


In: « vertiges positionnels » JP Sauvage, A Chays, A Gentine. Société française d'ORL, éditeur, 2007

Horizontal BPPV: positioning nystagmus

Canalolithiasis

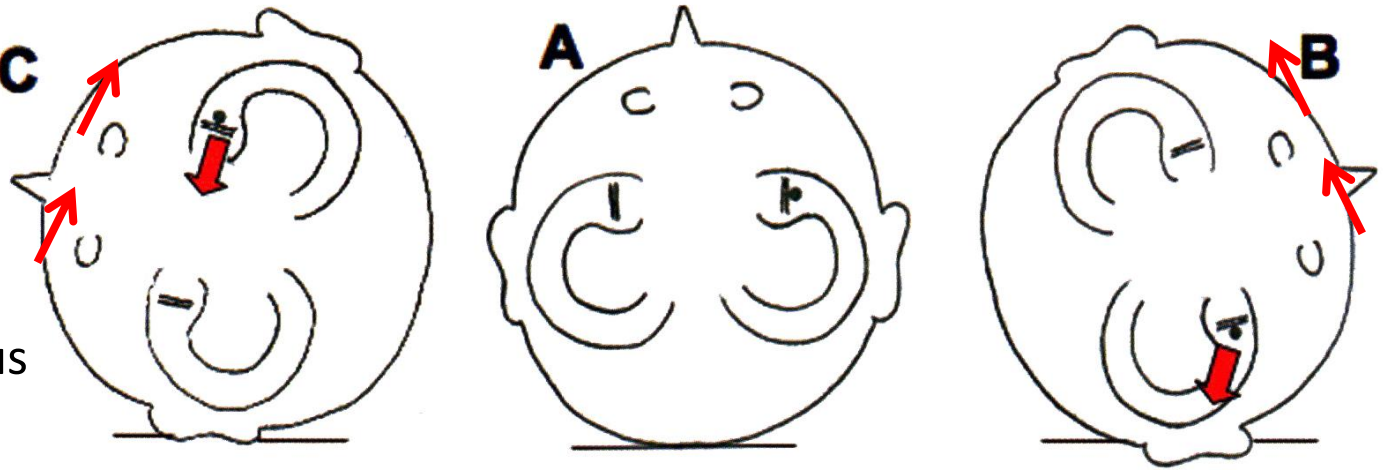
Direction-changing **geotropic** horizontal nystagmus



Supine position

Cupulolithiasis

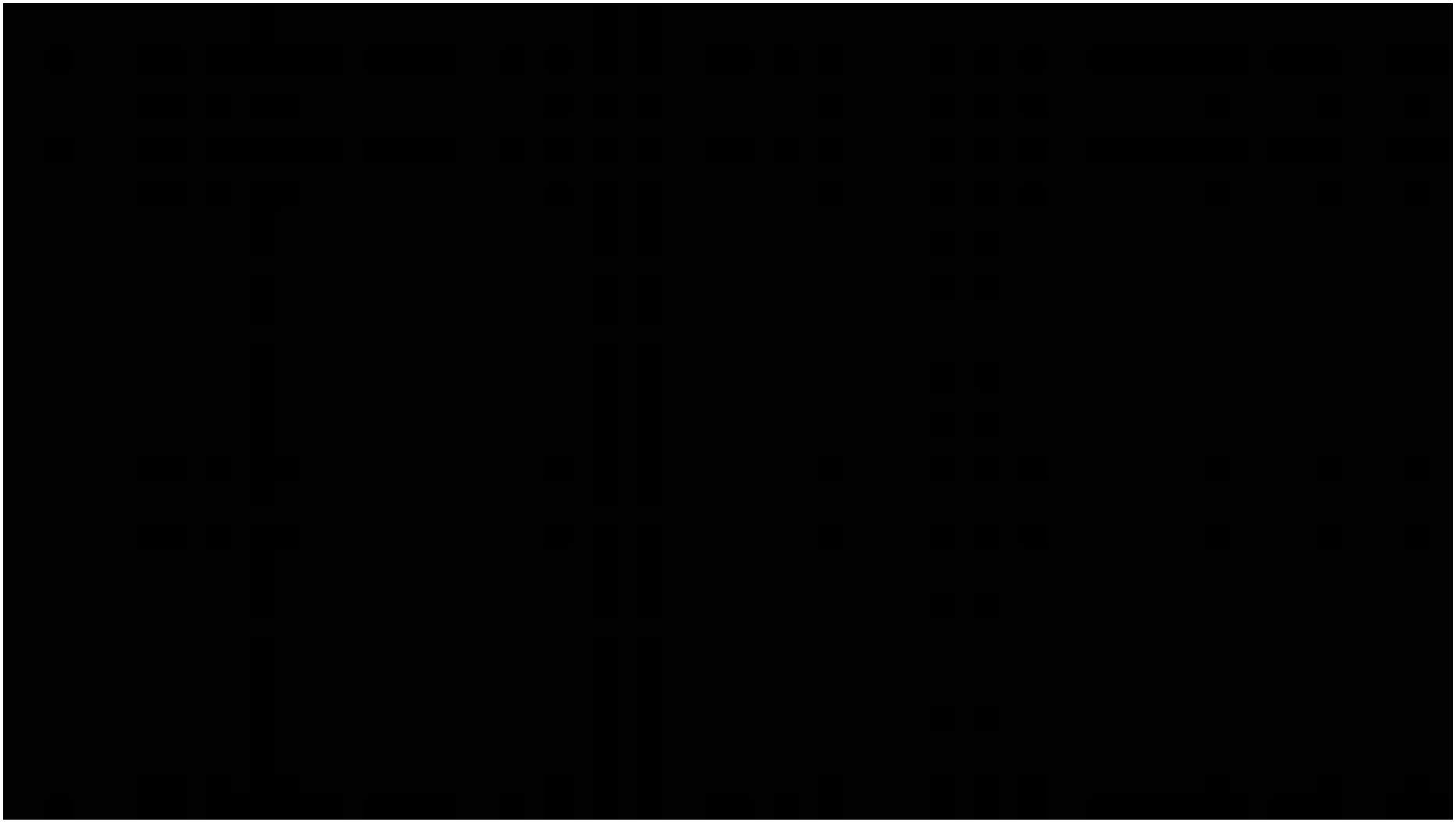
Direction-changing **apogeotropic** horizontal nystagmus



In: « vertiges positionnels » JP Sauvage, A Chays, A Gentine. Société française d'ORL, éditeur, 2007

horBPPV : diagnostic positioning manœuvre

Pagnini-McClure manœuvre, supine roll Test



Horizontal BPPV

- **canalolithiasis** — geotropic, transient nystagmus; greater intensity towards affected side
- **cupulolithiasis** — apogeotropic, persistent nystagmus; greater intensity towards healthy side
- nystagmus not fatigable with repeated positioning
- frequency: canalolithiasis > cupulolithiasis

Evolution

good prognosis
canalolithiasis quickly disappears < 2 weeks
common recurrences

Treatment

liberatory manoeuvre
Lempert (barbecue rotation), Gufoni

sustained position ≈ 12 hours on healthy side (Vannucchi)

Horizontal BPPV

Treatment liberatory manoeuvre

Lempert (barbecue rotation), Gufoni

sustained position ≈ 12 hours on healthy side (Vannucchi)

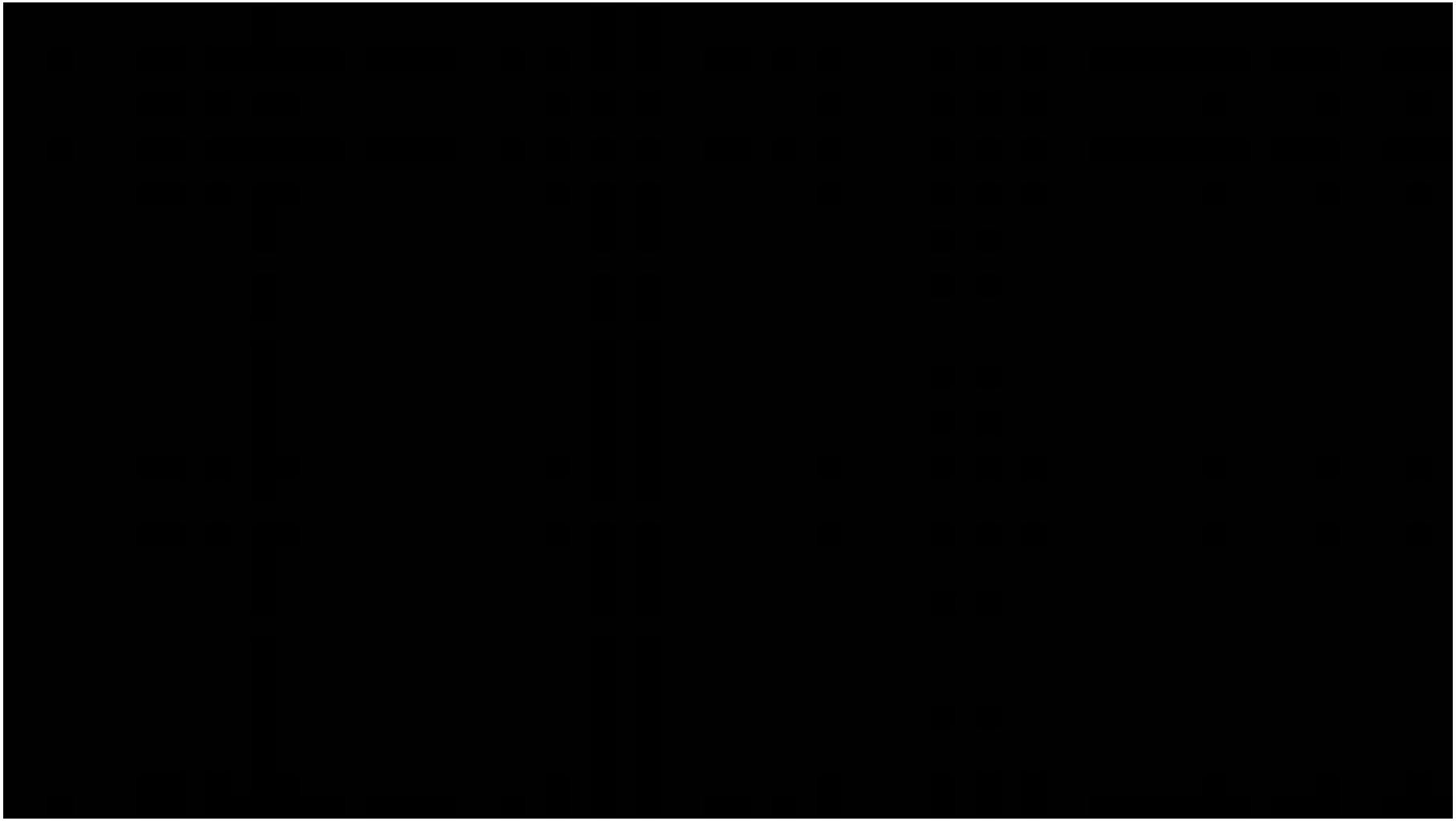
Evolution good prognosis

canalolithiasis quickly disappears < 2 weeks

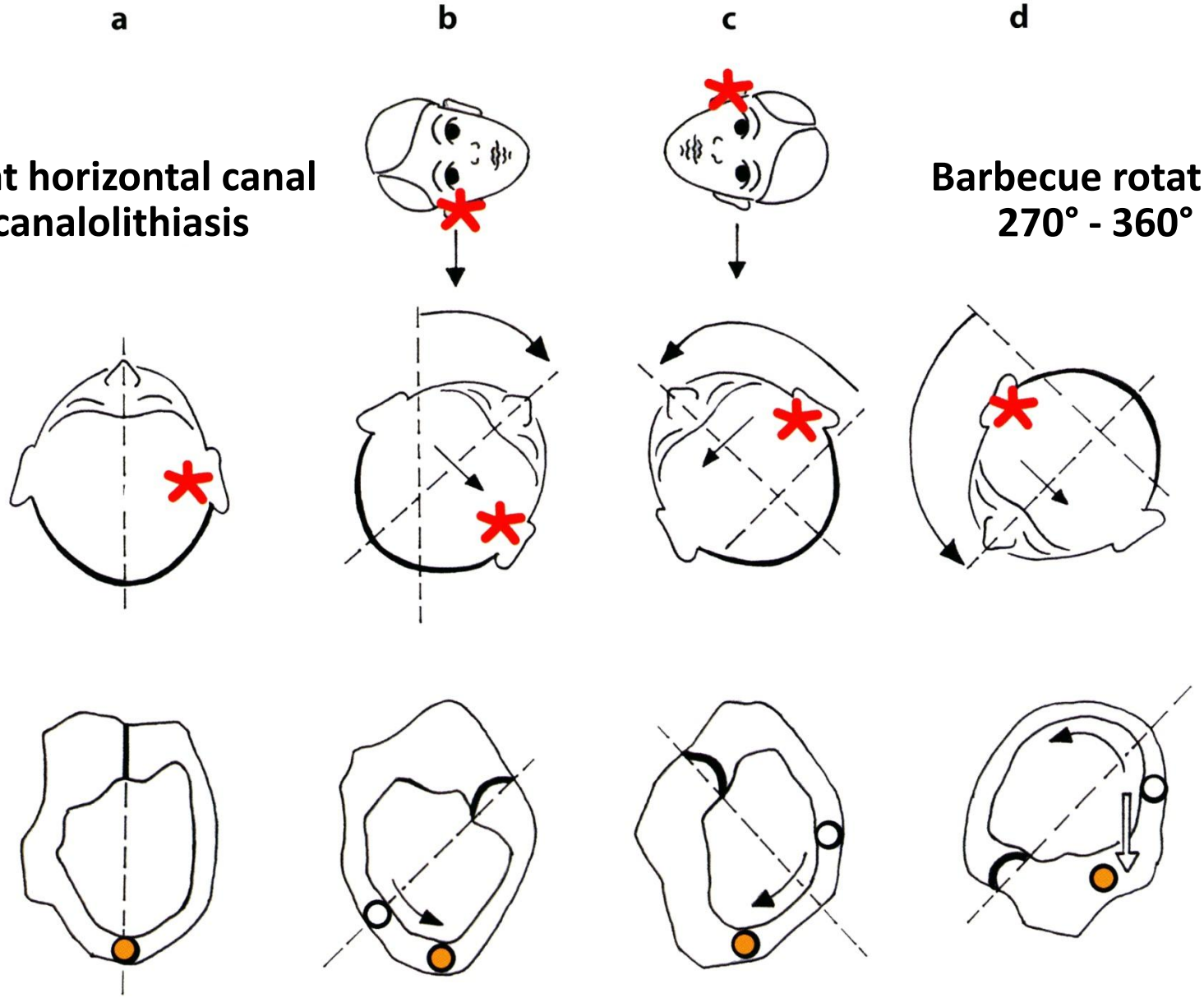
common recurrences

*horizontal*BPPV : Lempert manœuvre

Geotropic variant : towards healthy side (1 turn)
Apogeotropic variant: towards healthy side (1½ turn)



Horizontal BPPV: Lempert manoeuvre



In: « vertiges positionnels » JP Sauvage, A Chays, A Gentine. Société française d'ORL, éditeur, 2007

Anterior BPPV

- same general features than other canals BPPV

  latency, duration < 1 minute

- characteristic positioning nystagmus after Dix-Hallpike manœuvre with strong head hanging position

  downward vertical ± torsional

- rare: spatial orientation of anterior canal allows spontaneous evacuation of clot into the utricle just by sitting up

TABLE 1. *Criteria for determining canal involvement*

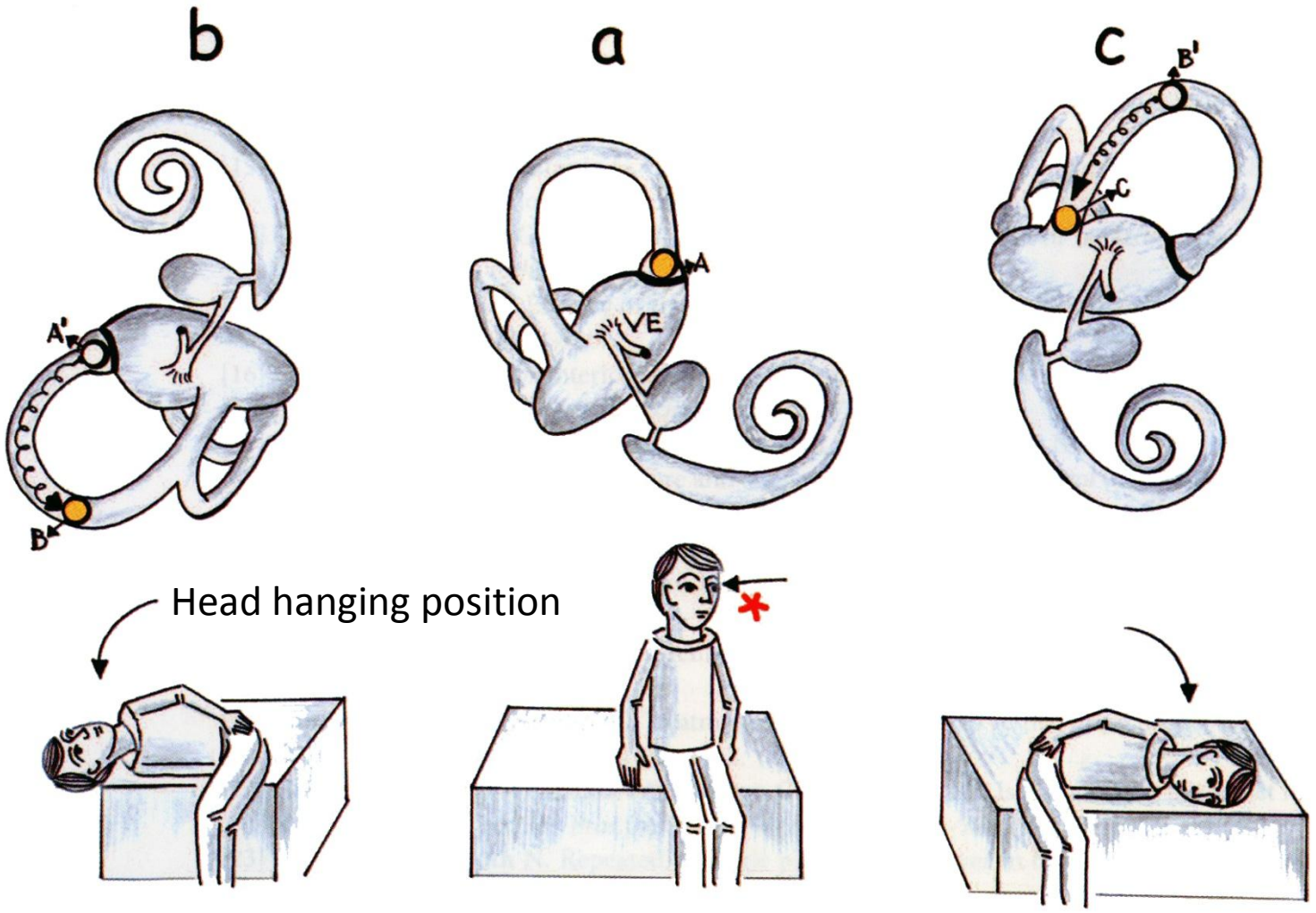
Canal	Positioning test	Induced nystagmus
Posterior semicircular canal	DH with the affected side down	Upbeating nystagmus with torsional component toward the affected side
Anterior semicircular canal	DH with the unaffected side down	Downbeating nystagmus with torsional component toward the affected side
	DH with the affected side down	No nystagmus or downbeating nystagmus with torsional component toward the affected side
	Straight head-hanging test	Downbeating nystagmus with torsional component toward the affected side

DH indicates Dix-Hallpike test.

Park et al., Canal Conversion Between Anterior and Posterior Semicircular in Benign Paroxysmal Positional Vertigo. Otol Neurotol 2013 ; 34 :1725-1728,

Anterior BPPV: liberatory manoeuvre

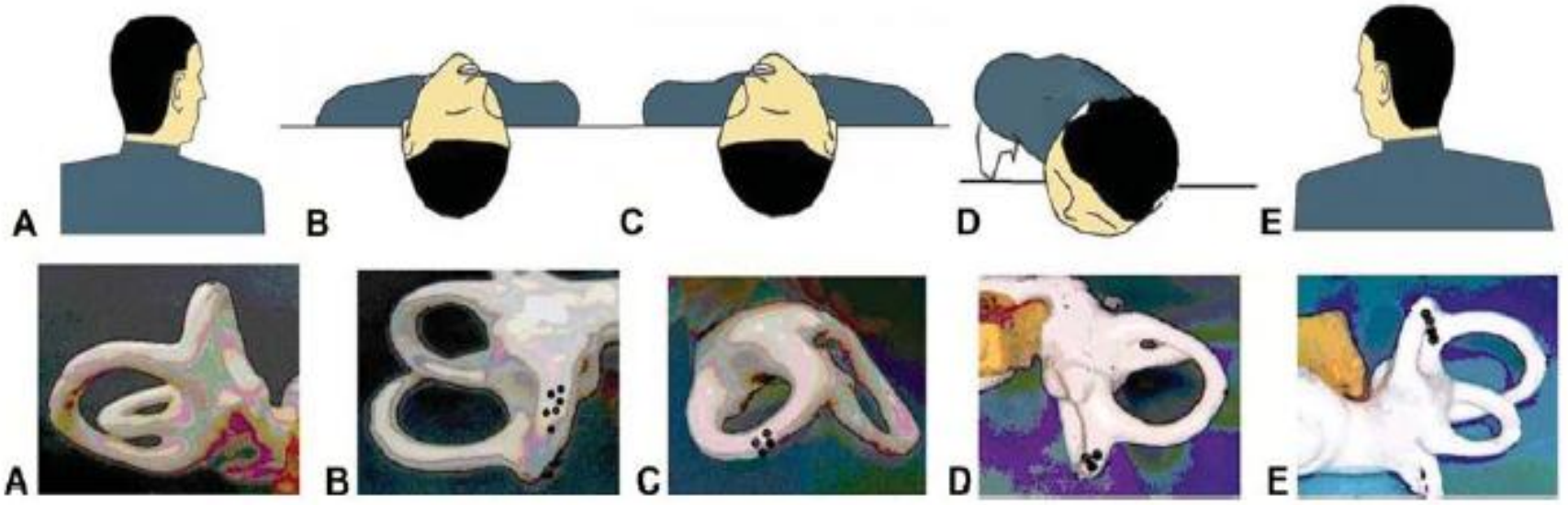
Semont manoeuver Left *ant*BPPV



In: « vertiges positionnels » JP Sauvage, A Chays, A Gentine. Société française d'ORL, éditeur, 2007

Anterior BPPV: liberatory manoeuvre

Epley reverse manoeuver (A) head turned 45° towards healthy side

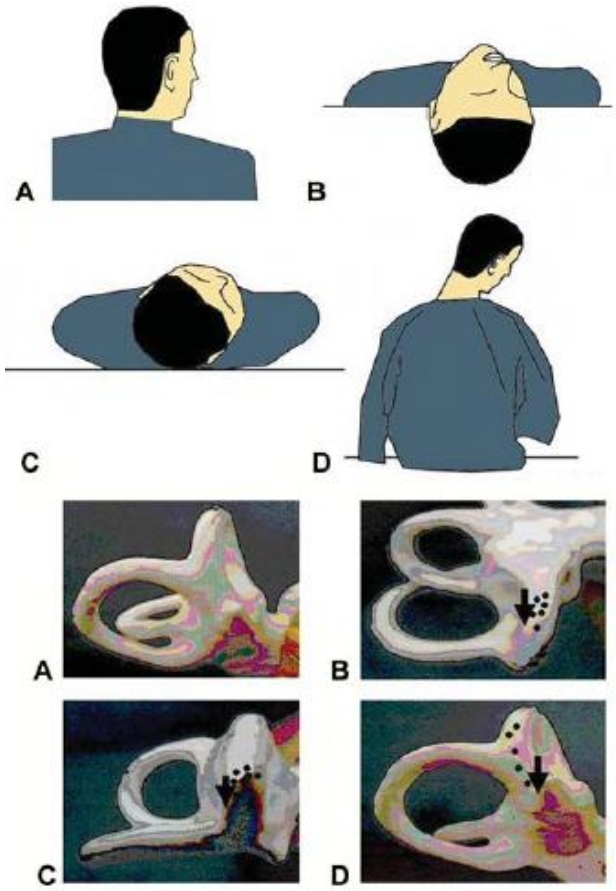


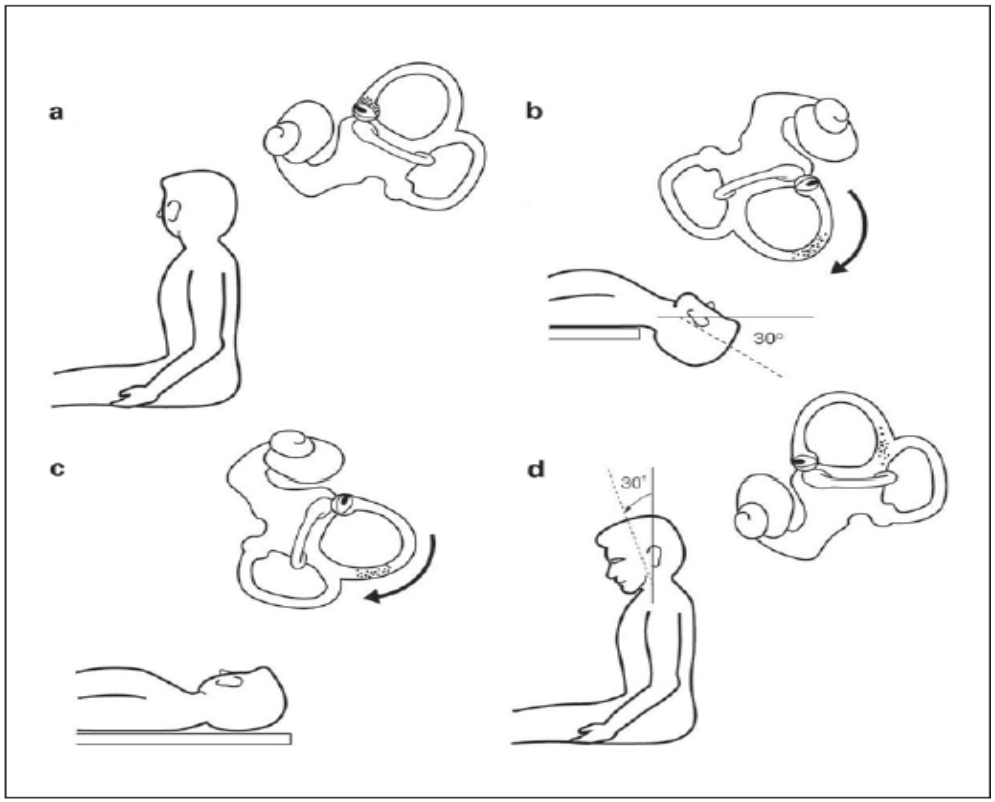
Korres S et al., Canalithiasis of the anterior semicircular canal (ASC) : Treatment options based on the possible underlying pathogenetic mechanisms, International Journal of Audiology 2010; 49 : 606-612

Anterior BPPV: liberatory manoeuvre

Kim manoeuver

(a) head turned 45° towards healthy side
(b) supine position 30° head hanging during 2 minutes.
(c) head elevated in horizontal position during1 minute.
(d) return to sitting position, chin tilted 30° down.



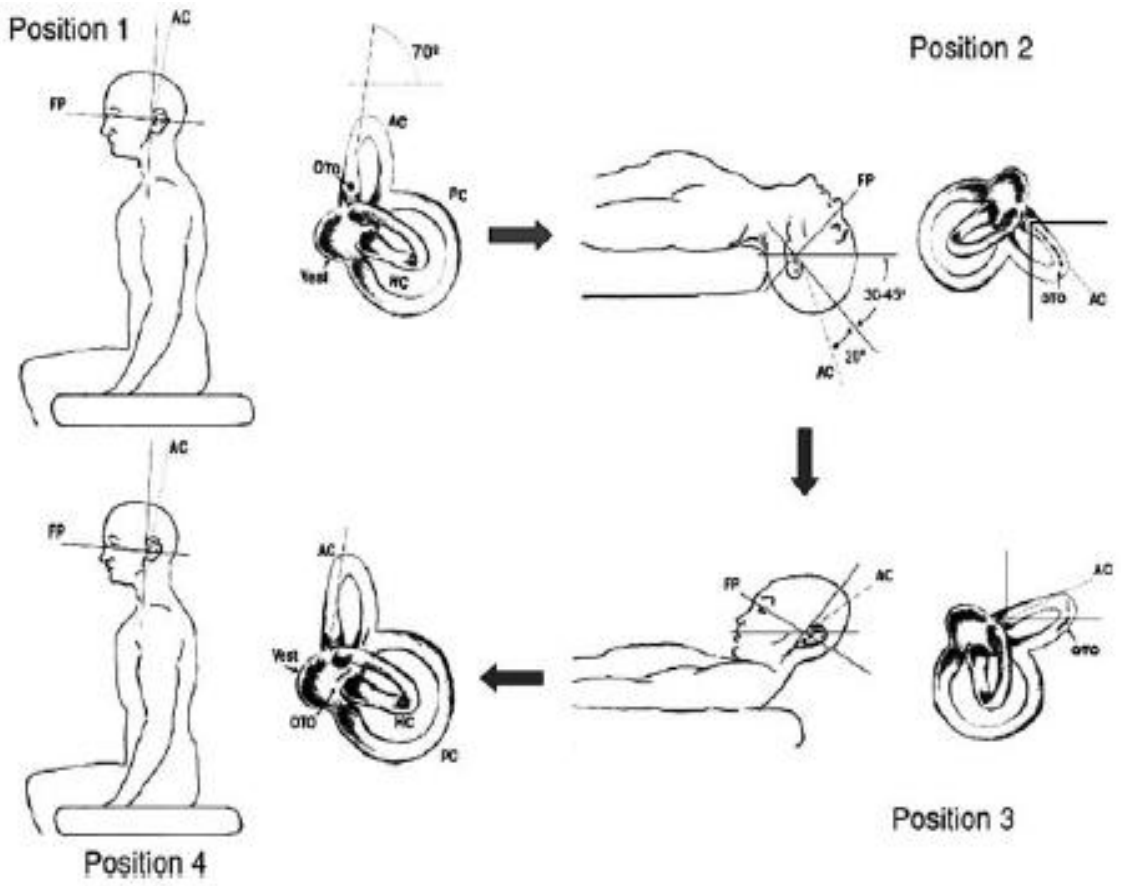


Kim YK et al., The effect of canalith repositioning for anterior semicircular canal canalithiasis. ORL J Otorhinolaryngol Relat Spec 2005 ;67 :56-60.

Anterior BPPV: liberatory manoeuvre

Yacovino manoeuver

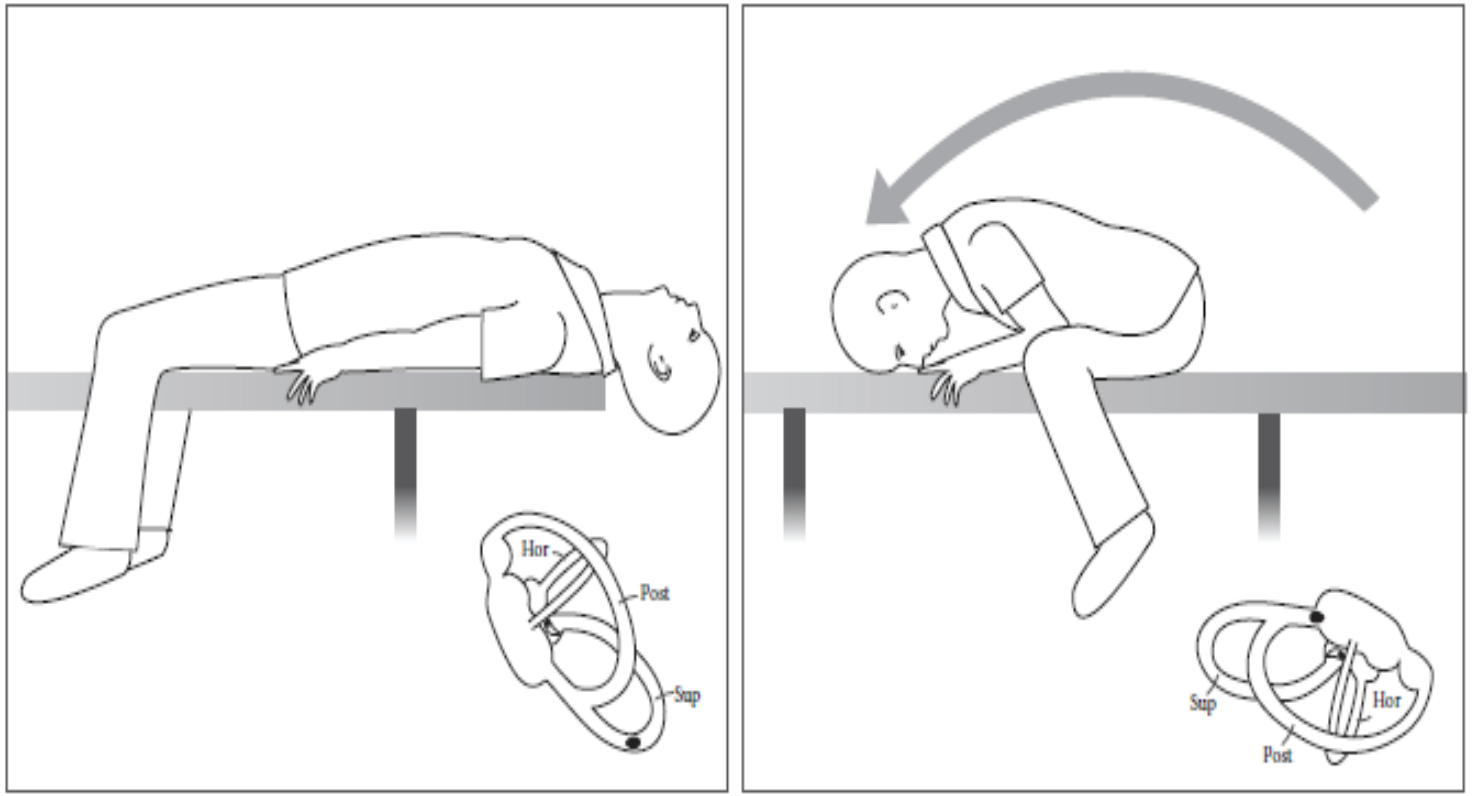
(position 2) head-hanging 30°
(position 3) head elevated chin to chest



Yacovino DA et al., New therapeutic maneuver for anterior canal benign paroxysmal positional vertigo
J Neurol 2009; 256 : 1851-1855

Anterior BPPV: liberatory manoeuvre

Li manoeuver rapid change from head-hanging to face down position



Hornibrook J, Benign Paroxysmal Positional Vertigo (BPPV) : History, Pathophysiology, Office Treatment and Future Directions International Journal of Otolaryngology 2011; Article ID 835671, 1-13