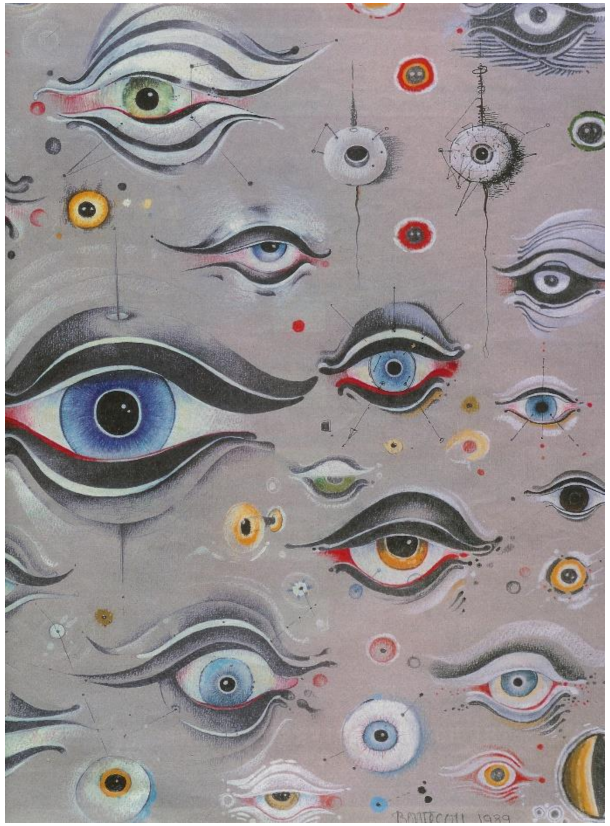
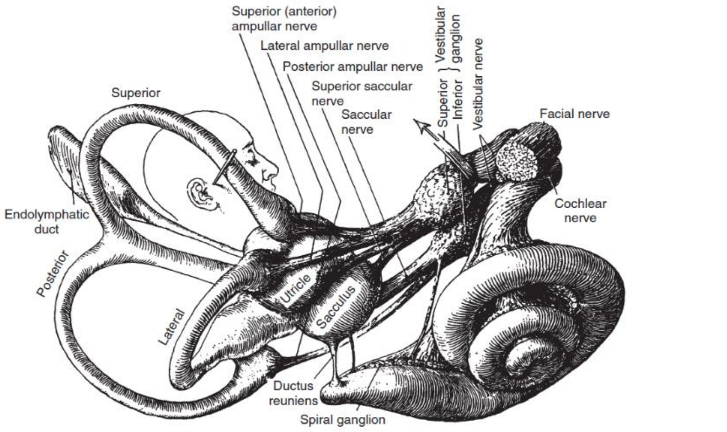
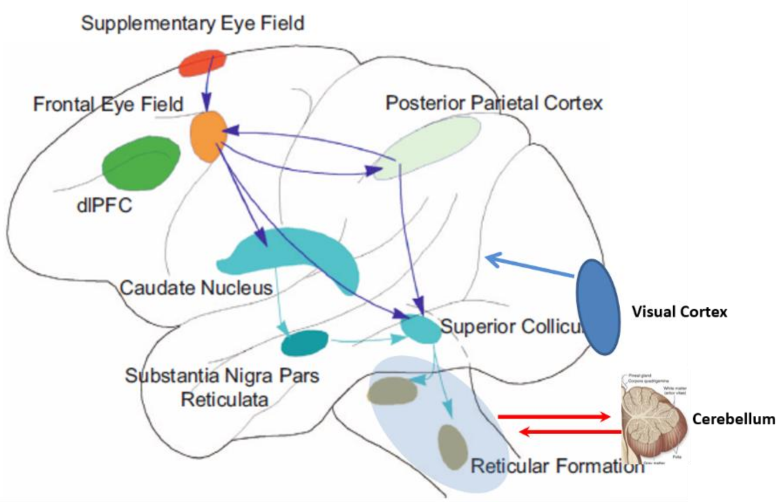
CENTRAL VERUS PERIPHERAL VESTIBULAR DISORDERS: USING EYE MOVEMENTS TO MAKE THE DIAGNOSIS





The vestibular end organs. (From Brodel M. *Three unpublished drawings of the anatomy of the human ear.* Philadelphia: WB Saunders; 1946.)



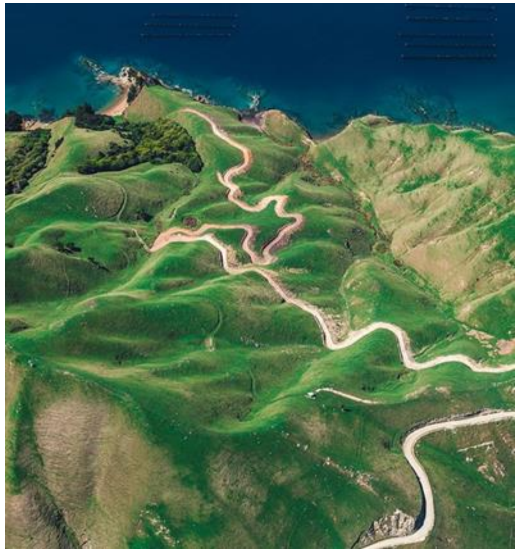
Modified from Pouget 2014

TWO REMINDERS

To understand eye movement disorders one must tackle the phylogenetically-old, primitive, *anläge* (foundation or scaffolding) for all types of eye movements -- The SLOW and QUICK phases of the vestibulo-ocular reflex (VOR)

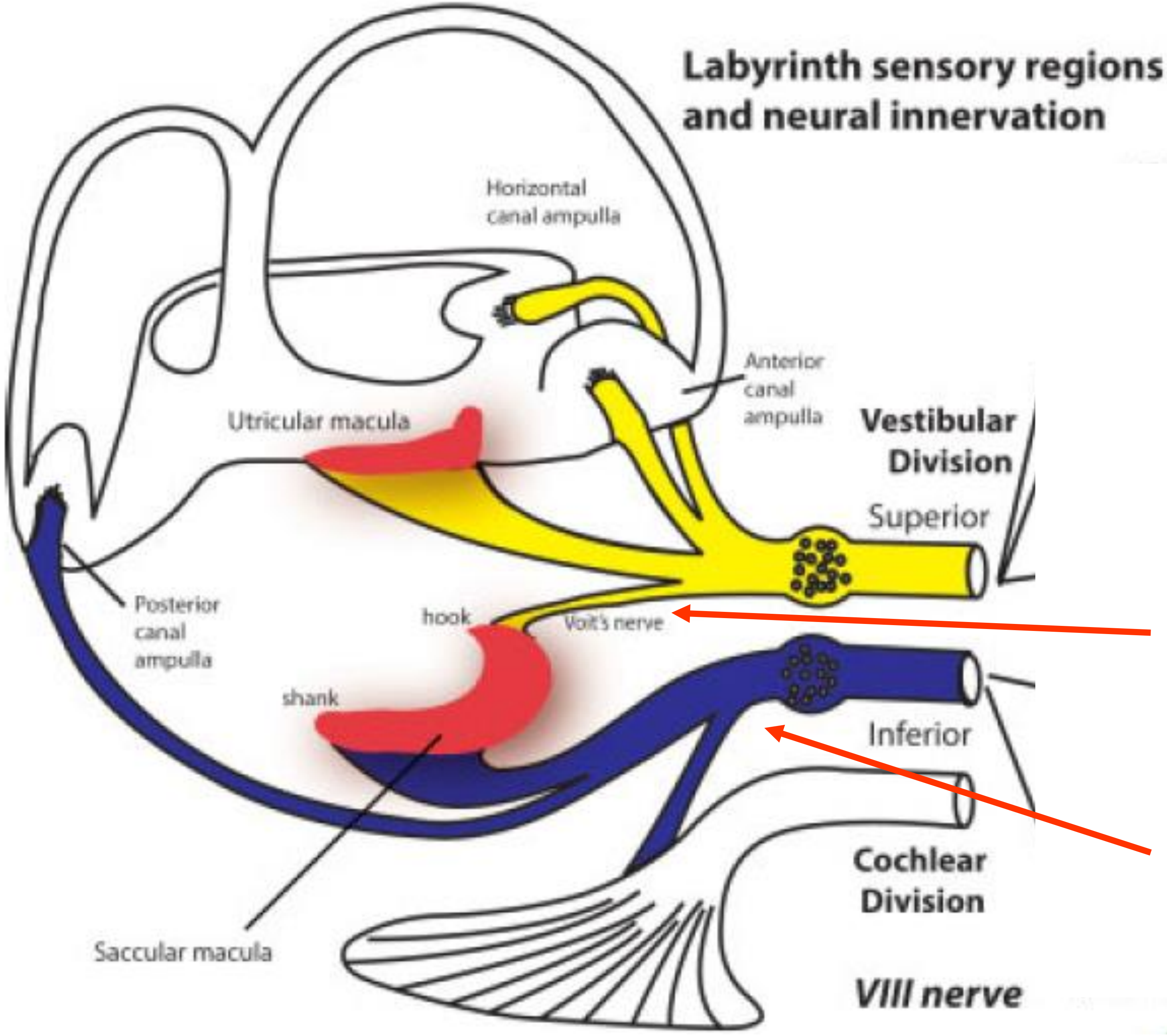
Conversely, the examination of eye movements including alignment, saccades and gaze-holding often offers the best clues to the diagnosis of a patient with a vestibular disorder.

The strategy to distinguish central from peripheral vestibular disorders using eye movements



- Based on the relevant physiology and anatomy

- Know the patterns of
  - Spontaneous nystagmus
    - Effect of fixation
    - Effect of changing eye position
  - Head impulse responses
  - Head-shaking induced nystagmus
  - Vibration-induced nystagmus
  - Hyperventilation-induced nystagmus
  - Valsalva induced nystagmus
  - Positional nystagmus
  - Saccades
    - speed
    - accuracy
    - oscillations



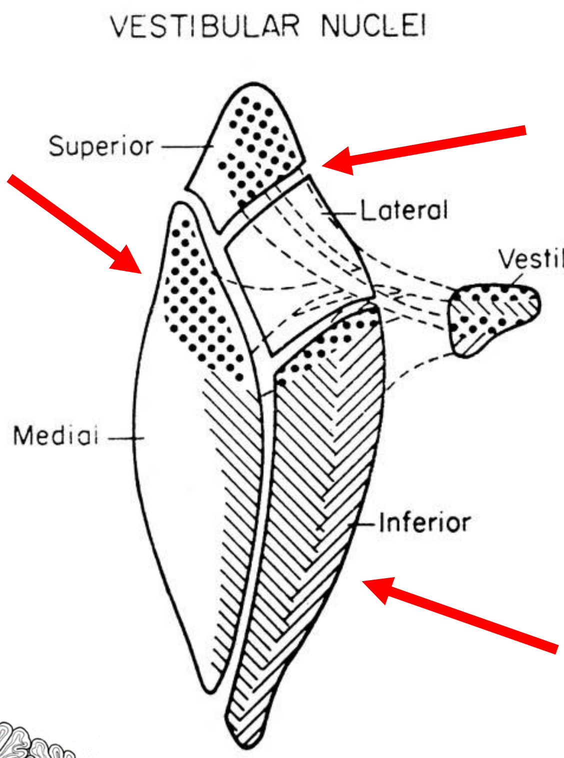
Dual supply of saccule

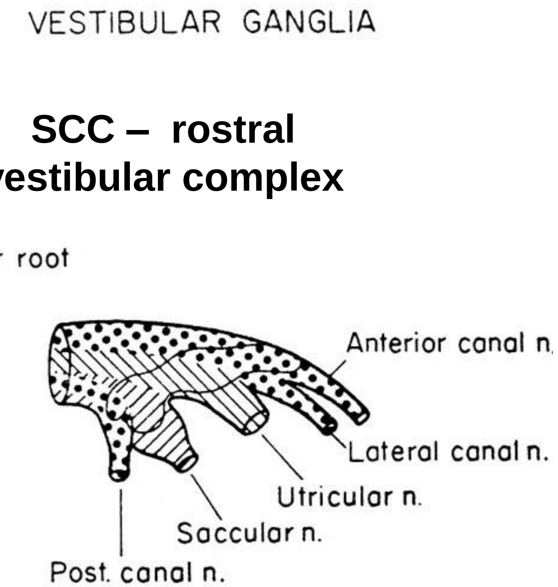
Dual supply of cochlea

Curthoys Manzarri

MORE KEY ANATOMY:

SCC and otolith projections are <u>segregated</u> in the lower brainstem





SCC – rostral vestibular complex

OTOLITH – caudal vestibular complex

VESTIBULOCEREBELLUM

SCC / Otolith – FLOCCULUS: TONSIL, NODULUS:

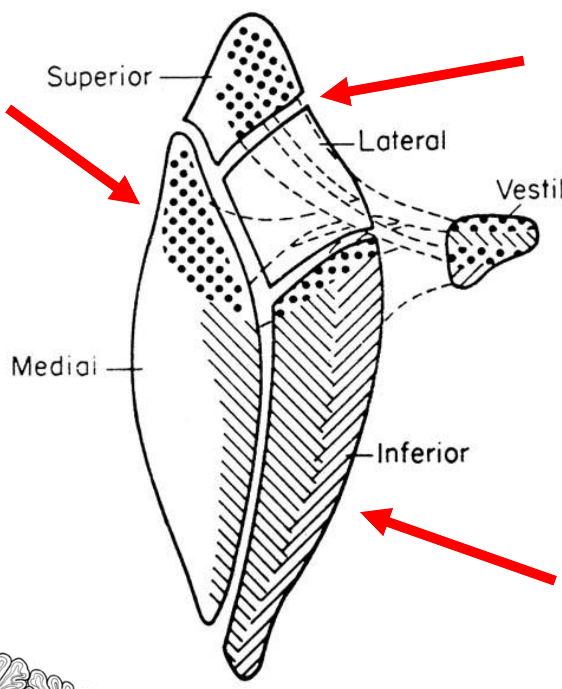
MORE KEY ANATOMY: 

1) Canal and Otolith projections are segregated in the lower brainstem

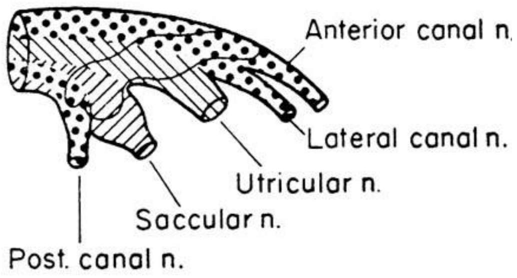
2) Patterns of blood supply to the *central* labyrinthine projections

VESTIBULAR NUCLEI

VESTIBULAR GANGLIA



SCC – rostral vestibular complex ***(AICA)***



OTOLITH – caudal vestibular complex ***(PICA)***

VESTIBULOCEREBELLUM

SCC / Otolith – FLOCCULUS: ***(AICA)***
TONSIL, NODULUS: ***(PICA)***

***AICA*, anterior inferior cerebellar artery**
***PICA*, posterior inferior cerebellar artery**

Central projections from the labyrinth

- SCC projections are primarily to the rostral portions of the vestibular complex (AICA)

- Otolith projections are primarily to the caudal portions of the vestibular complex (PICA)

- **Beware infarct in lateral medulla (Wallenberg's syndrome)** when symptoms and signs are isolated otolith (tilt, vertical diplopia due to skew (vertical misalignment of eyes)).

- SCC and otoliths also project directly to the cerebellum. **Beware of cerebellar infarct and hemorrhage in elderly presenting as 'vestibular neuritis',** especially if they have **HEADACHE** or **CANNOT WALK**, even with assistance. Be especially aware if they have spontaneous nystagmus WITHOUT an abnormal VOR (head impulse sign).

Localization of vestibular nystagmus: KEY POINTS

Pointing to a ***PERIPHERAL*** lesion

- **Nystagmus is increased or brought out by <u>removal</u> of fixation (Romberg sign of VOR)**

- **Mixed horizontal-torsional pattern is characteristic for complete unilateral loss of function**

- **Nystagmus intensifies when looking in the quick-phase direction (Alexander's Law)**

- **Nystagmus obeys Ewald's 1st Law: Eye rotates in a plane <u>parallel</u> to the stimulated canal no matter what the position of the eye in the orbit**

  (E.g., in Benign Paroxysmal Positional Vertigo (BPPV) of the posterior canal (due to detached otoconia in the canal) the nystagmus is more *<u>vertical</u>* when looking *<u>away</u>* from side of the affected labyrinth and more *<u>torsional</u>* when looking *<u>toward</u>* the side of the affected labyrinth)

Localization of vestibular nystagmus: KEY POINTS

Pointing to a *CENTRAL* lesion

- **Fixation suppression of nystagmus usually impaired**
  (Caveat: May not be impaired with unilateral brainstem or cerebellar lesions)

- **Pure vertical or pure torsional nystagmus, and which remains so on eccentric gaze**

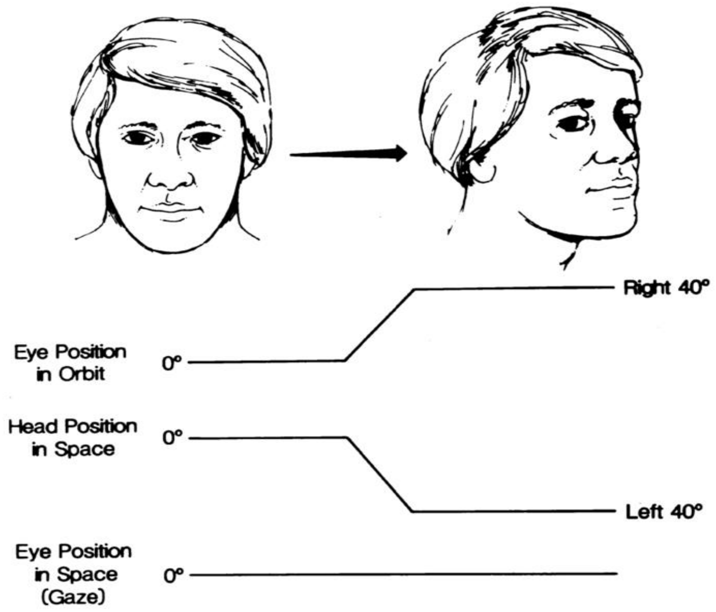
- **Nystagmus considerably modulated with vergence**

- **Nystagmus that diminishes when looking in the direction of the quick phase (anti-Alexander's law)**

(Caveat: Nystagmus that obeys Alexander's law or Ewald's 1st Law can be CENTRAL if it involves the central projections of the semicircular canals)

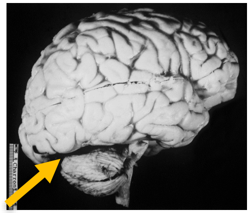
High-Frequency brief VOR

(rotational) R-VOR



The Head Impulse Test

What is wrong with this VOR response?

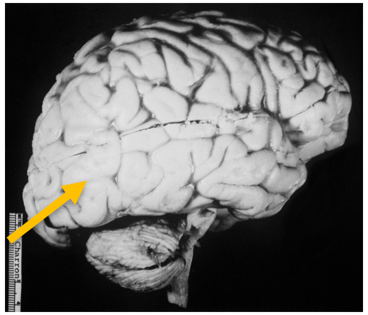


Abnormal VOR in cerebellar disease: Abnormal direction (X-coupling)

Abnormal VOR in cerebellar disease: <u>Increased gain</u>

What is wrong with this VOR response?

(Note: Patient is fixating straight ahead)



Corrective saccades IN THE DIRECTION of head rotation (opposite the slow phase) during fixation of a stationary target indicate a HYPERACTIVE VOR

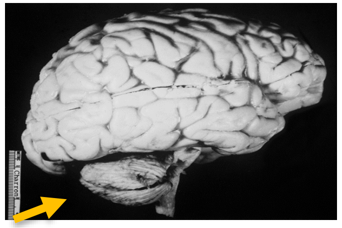
Corrective saccades OPPOSITE THE DIRECTION of head rotation (same as slow phase) during attempted fixation of a target indicate a HYPOACTIVE VOR

Vestibuloocular abnormalities in diffuse cerebellar atrophy

DISSOCIATION of effects of cerebellar lesions on the rotational VOR and on the translational VOR

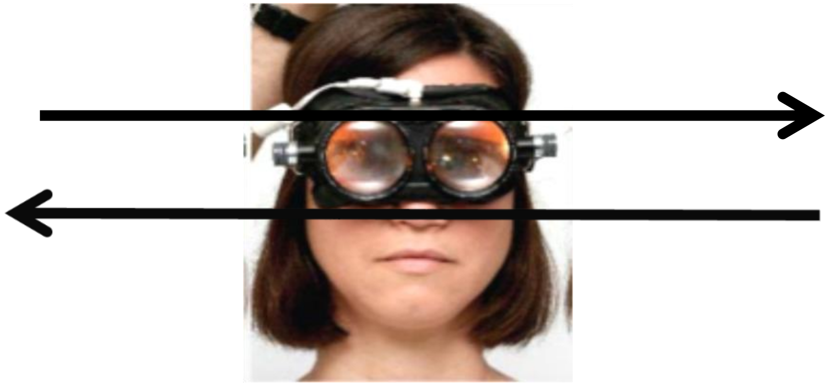
SPARING of *rotational* VOR and markedly IMPAIRED *translational* VOR

DOWNBEAT NYSTAGMUS increased by lateral gaze in SPINOCEREBELLAR ATAXIA TYPE 6



Abnormal processing of information for otolith adjustments to translational VOR which depend on the line of sight

PATTERNS OF HEAD-SHAKING INDUCED NYSTAGMUS



Head-shaking induced nystagmus (HSN) in peripheral labyrinthine disease (Frenzel lenses to eliminate fixation)

The slow phase of HSN is directed toward the paretic labyrinth

Head-shaking nystagmus (HSN) in cerebellar disease

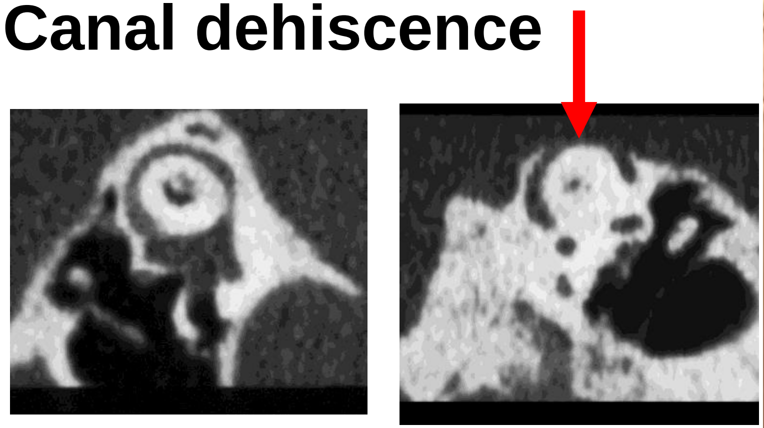
Head-shaking nystagmus (HSN) in cerebellar disease

PEARL: Think central if HSN is

- **Directed DIFFERENTLY than head motion (cross-coupled), e.g, vertical nystagmus with horizontal head-shaking.**
- **Directed <u>OPPOSITE</u> to spontaneous nystagmus**
- **If there is a reversal of the direction of HSN that is <u>EARLY AND STRONG</u> (patients with a peripheral lesion may have a smaller and later reversal of HSN)**

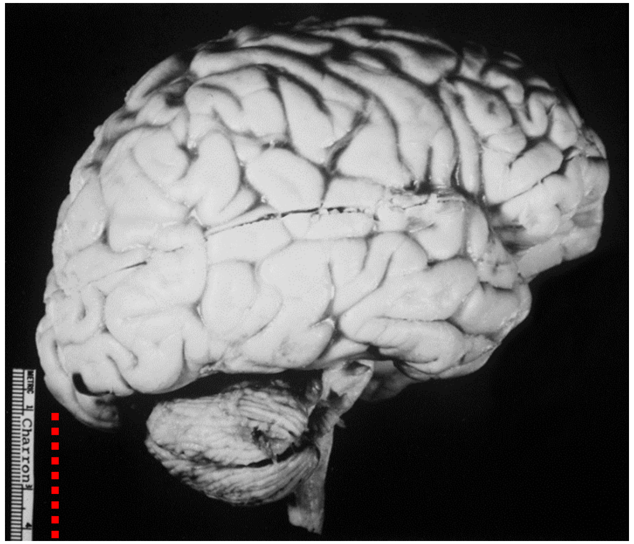
Valsalva-induced vertigo

Superior Semicircular Canal dehiscence



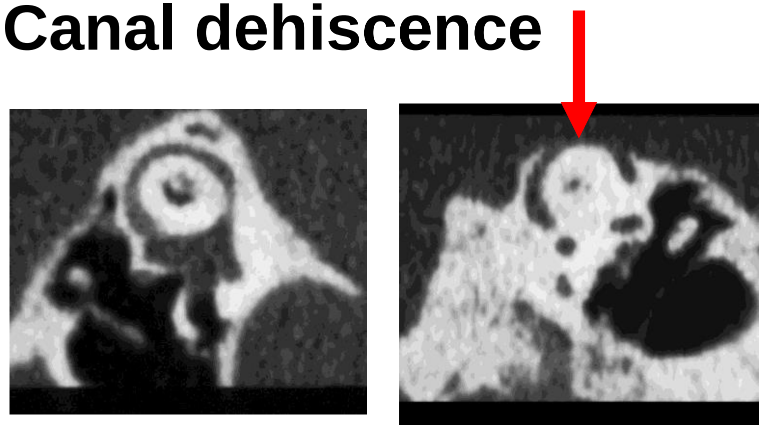
Valsalva-induced nystagmus (can be central or peripheral)

Cranial-cervical junction: Chiari



Cerebellar atrophy: SCA6

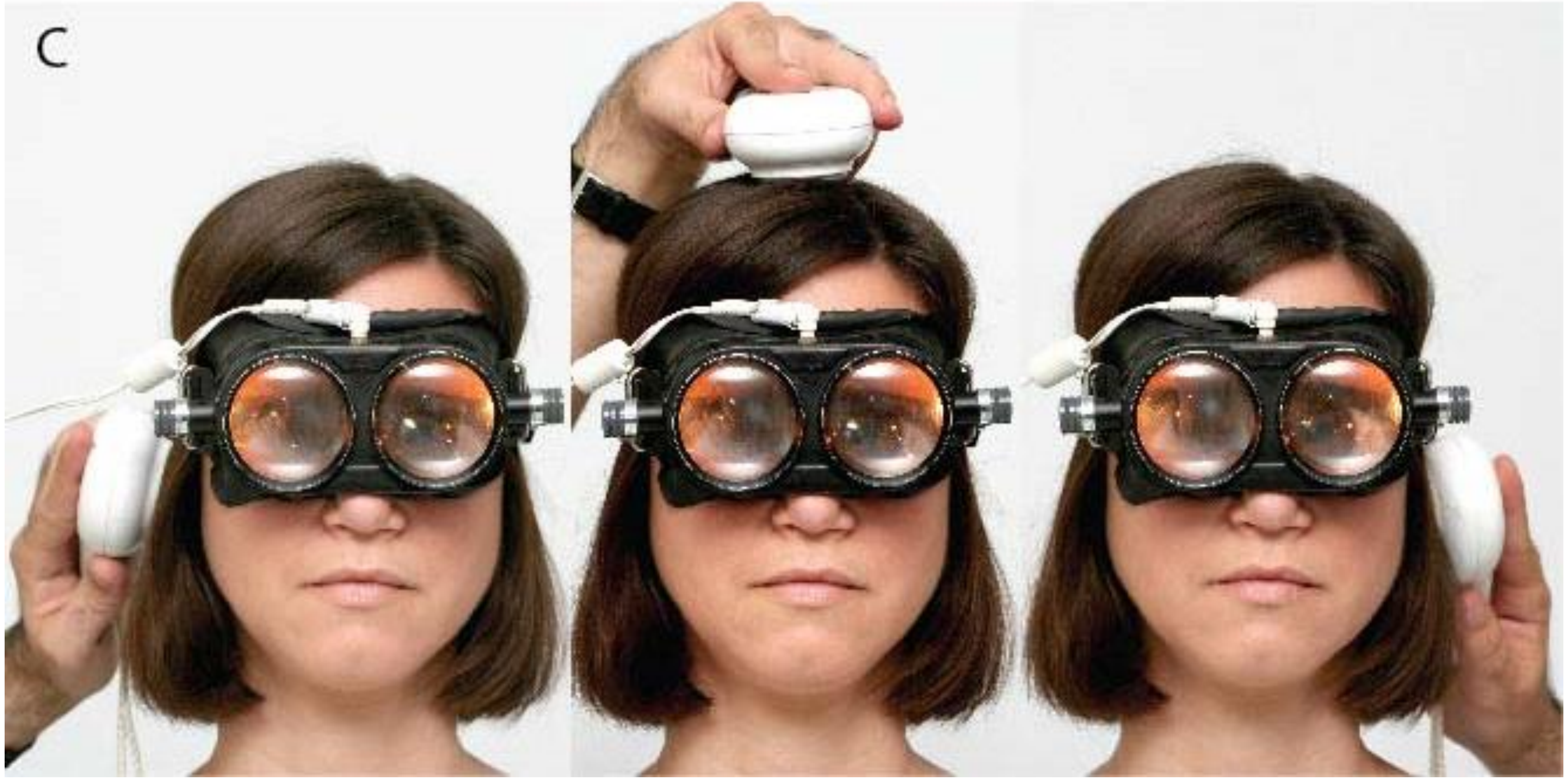
Superior Semicircular Canal dehiscence



PEARL

Remember Valsalva-induced vertigo with cranial-cervical junction anomalies and with labyrinthine fistula such as SCC dehiscence

With a <u>fistula</u> there is often associated NOISE induced vertigo (Tullio phenomenon)

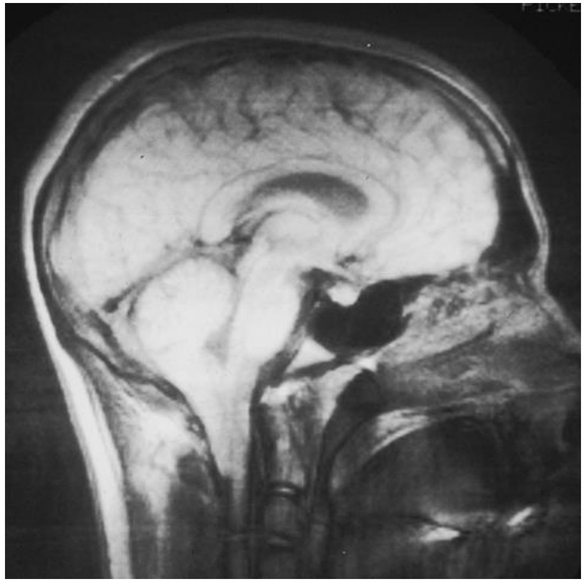


Vibration-induced nystagmus

‘Hot water caloric’ with unilateral (right) loss of labyrinthine function

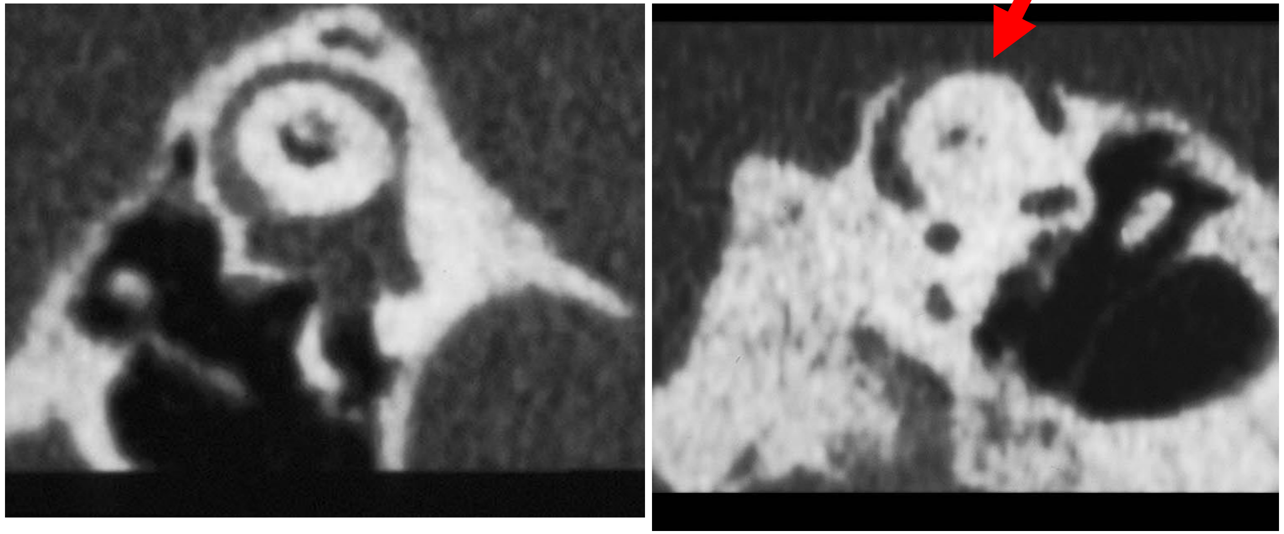
Vibration-induced nystagmus (central lesion, Wernicke's Encephalopathy)

Valsalva-induced nystagmus may be central or peripheral



Cranial-cervical junction anomalies (Chiari)

Fistula (superior canal dehiscence)



AWAYS ask about Valsalva-induced symptoms in patients with headache or with vestibular complaints

Hyperventilation

Hyperventilation-induced (HVN) downbeat nystagmus

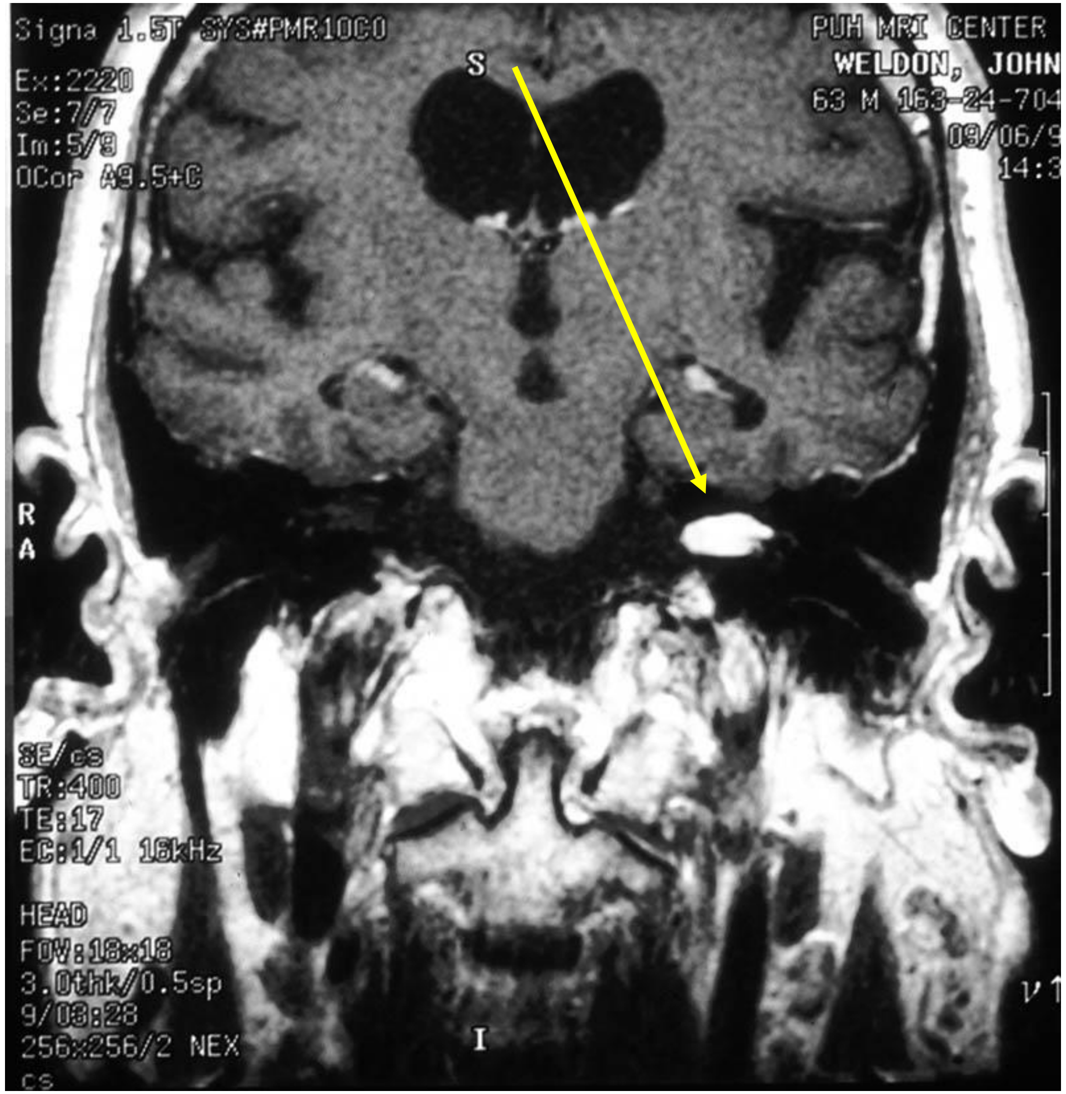
PEARL: HVN

- **Cranial-cervical junction anomalies**
- **Cerebellar degenerations**
- **Compressive lesions on VIII CN (microvascular compression, tumors)**
- **Demyelinating diseases (e.g., MS)**
- **Labyrinthine fistula and SCC dehiscence**

Patient with slightly asymmetrical hearing loss and mild imbalance

Hyperventilation

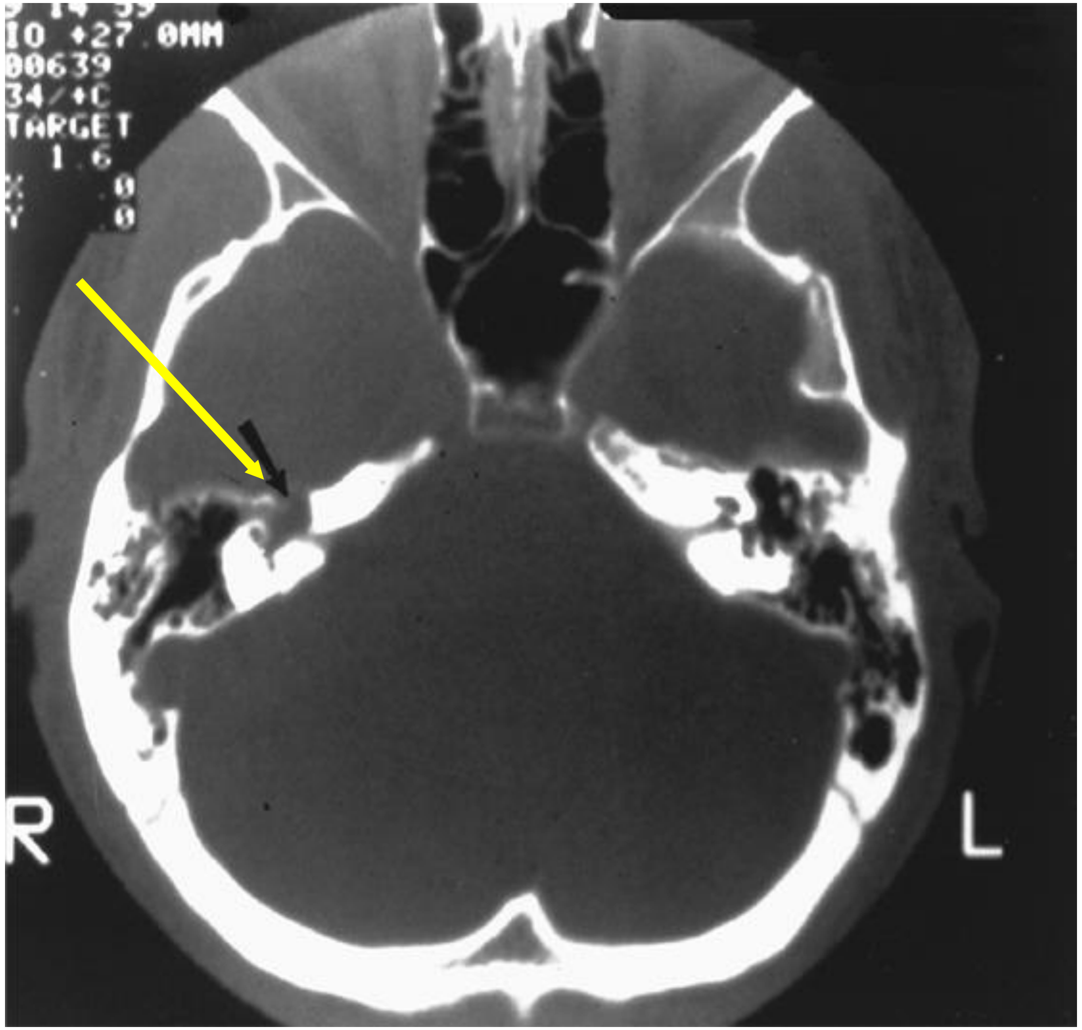
Hyperventilation-induced nystagmus



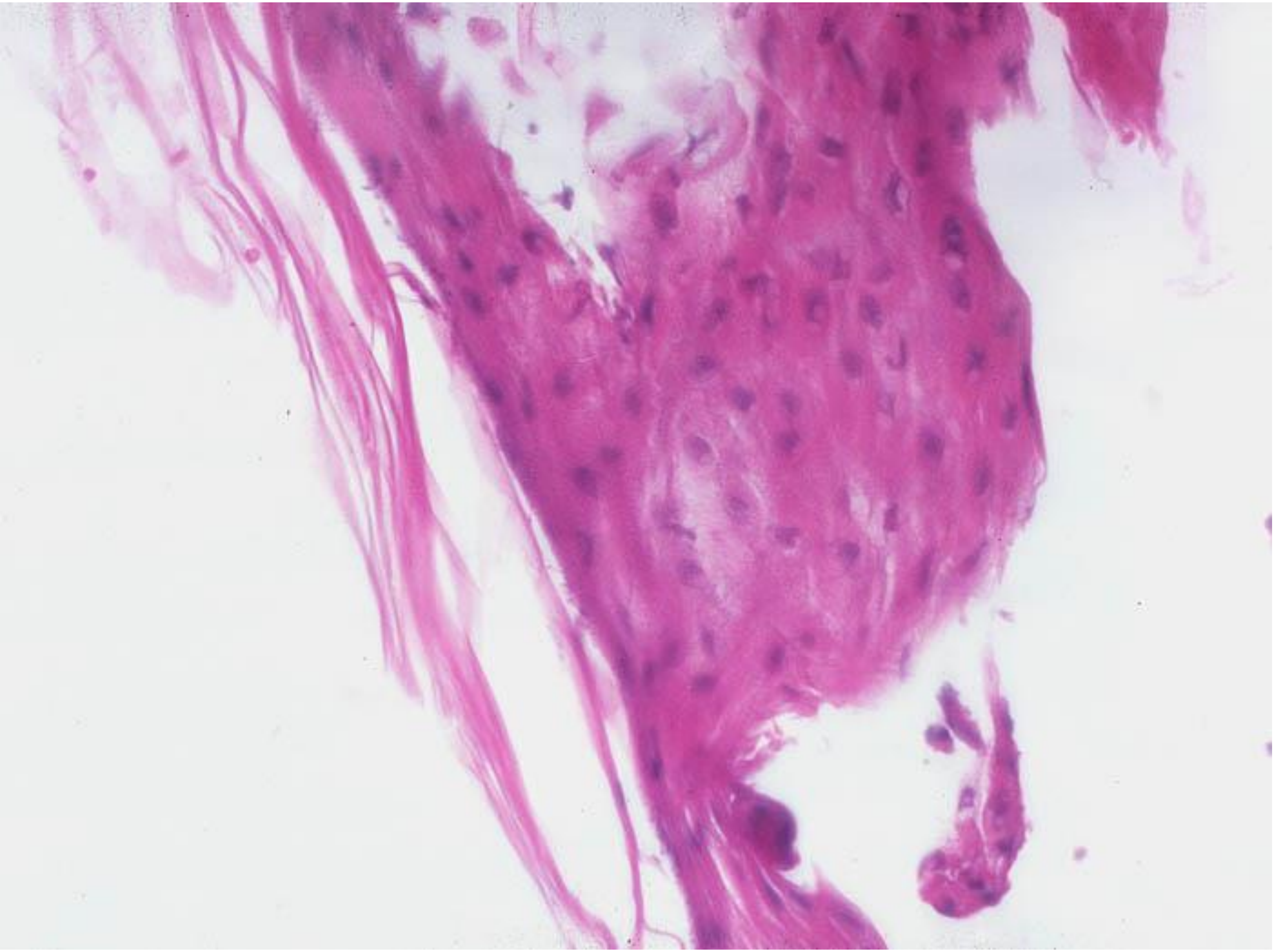
Vestibular Schwanoma

Patient with dizziness when playing lacrosse

Hyperventilation-induced nystagmus



Congenital Epidermoid

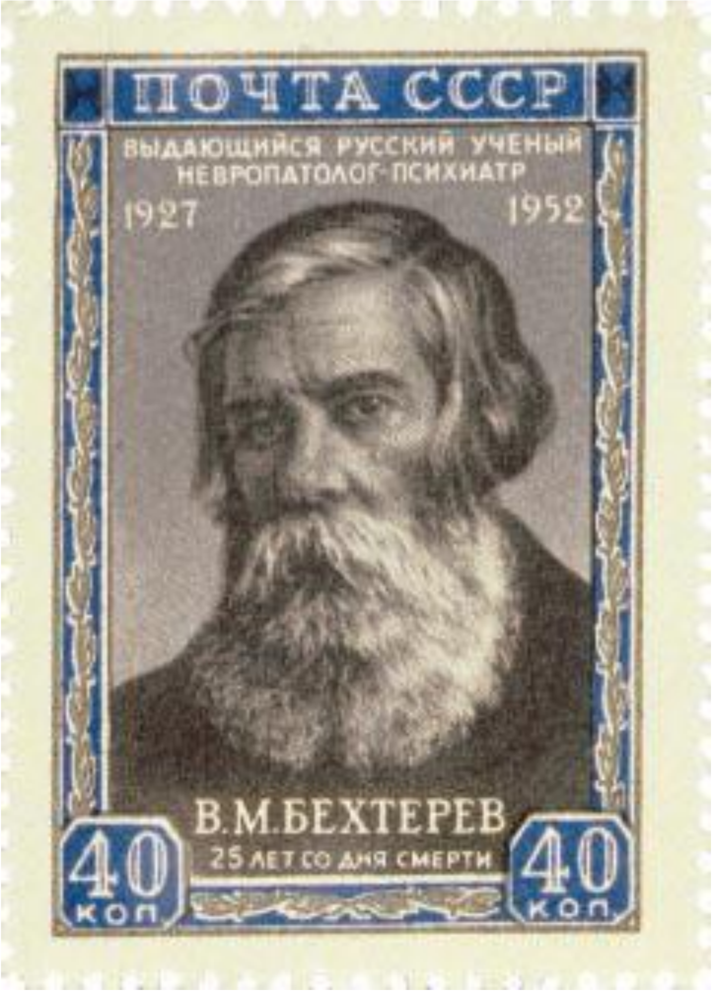


RECOVERY NYSTAGMUS

- **After a unilateral peripheral loss of vestibular function there is a central rebalancing to eliminate the spontaneous nystagmus.**

- **If peripheral function suddenly "recovers" (e.g., by improved conduction on demyelinated nerves because of pH changes due to hyperventilation), a new imbalance is created causing a recovery nystagmus with slow phases directed away from the affected ear.**

Recovery nystagmus and Bechterev's phenomenon



- **Loss of function in one labyrinth produces spontaneous nystagmus that slowly recovers over day**
- **If then, there is loss of function in the other labyrinth, again there is a transient (days) spontaneous nystagmus (slow phase toward the more recently affected labyrinth) even though caloric testing shows NO response in either ear.**

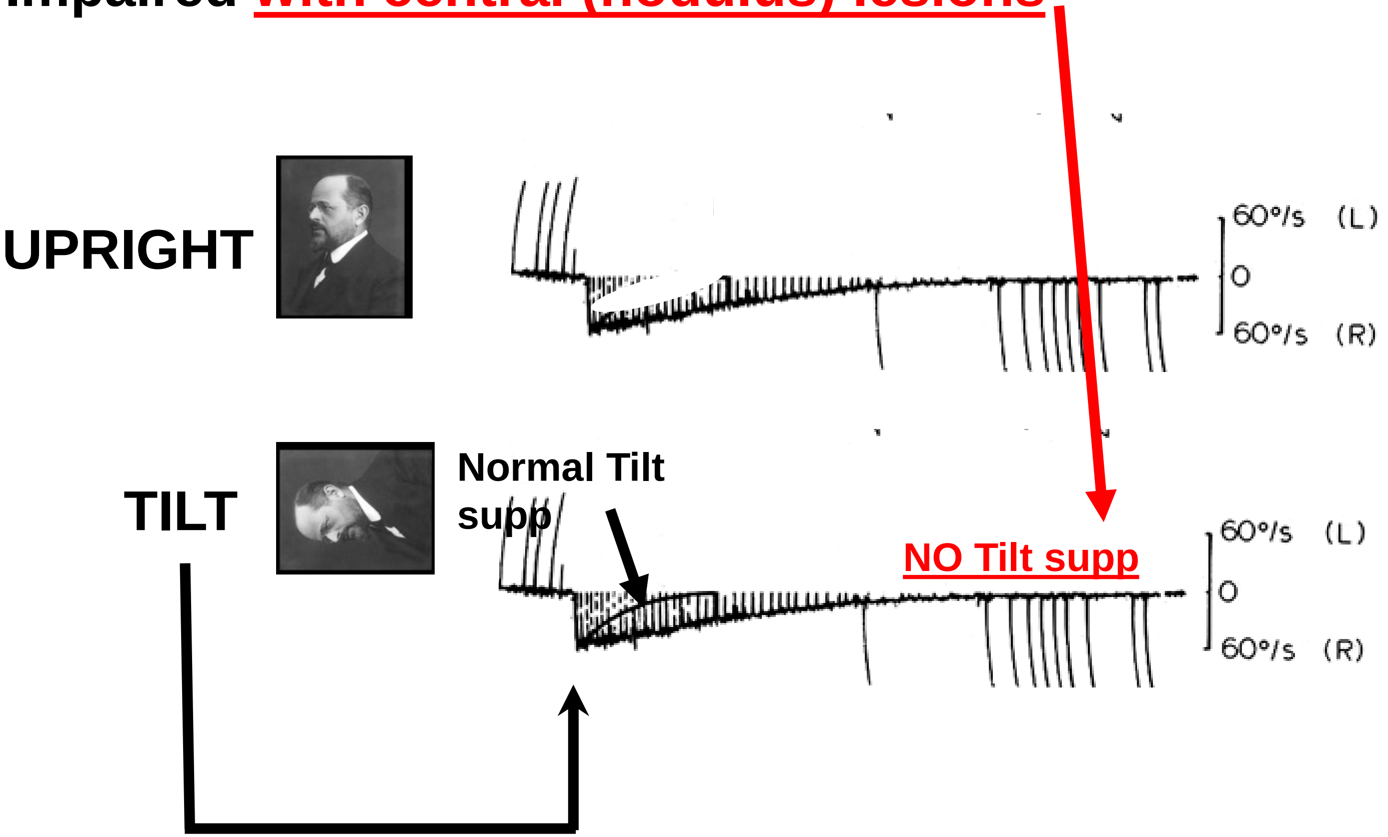
EXAMPLES

- **Hyperventilation induced nystagmus with a demyelinating lesion on VIII nerve**
- **Meniere's syndrome**
- **Post Labyrinthitis**

ANOTHER NODULUS TEST

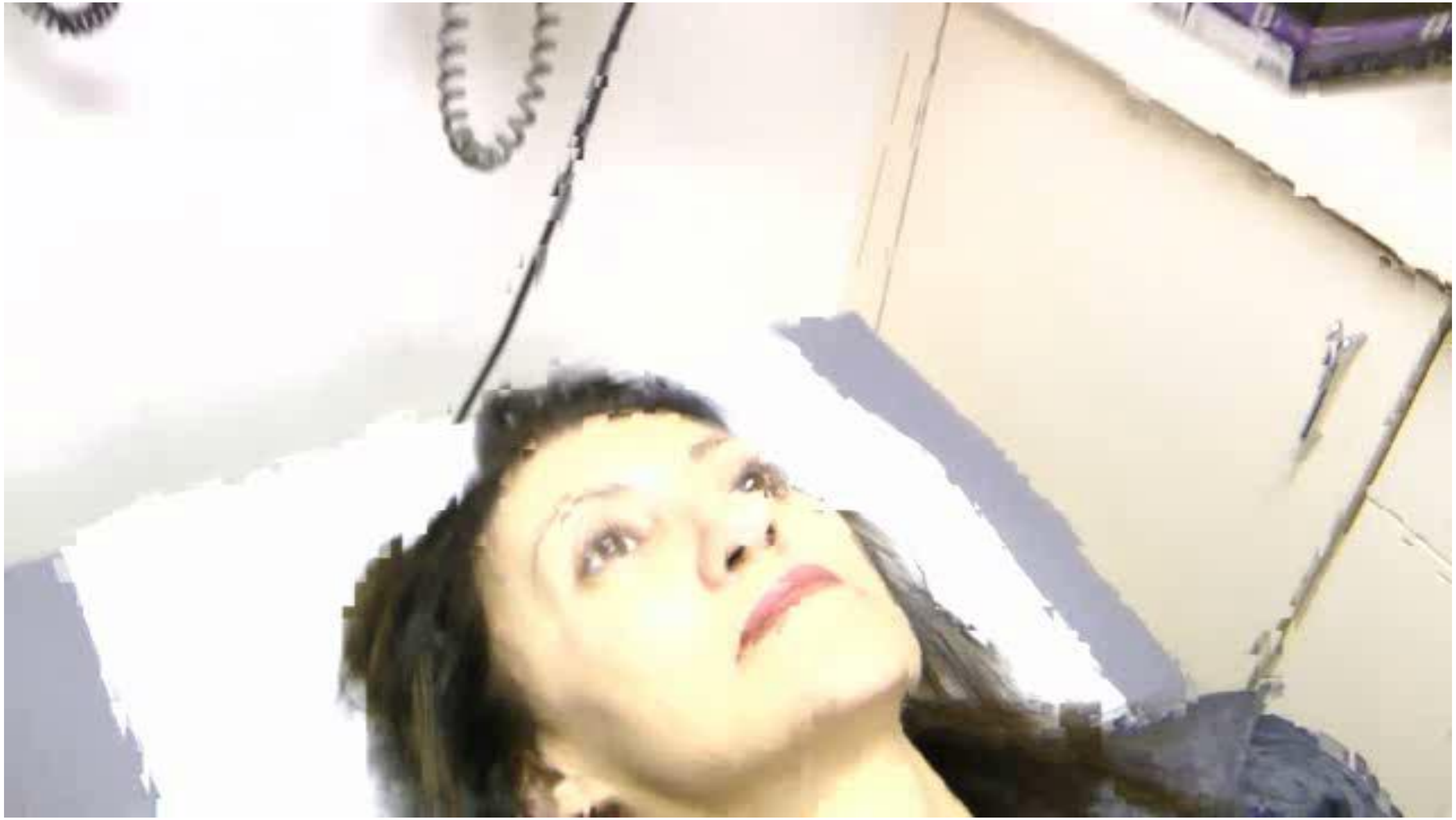
Tilt suppression (Tilt supp) of *head-shaking induced nystagmus.*
(Note the head is tilted just *after the head shaking stops*)

- **Normal with peripheral lesions**
- **Impaired with central (nodulus) lesions**



Zuma et al.

Lateral canal bppv: apogeotropic right ear affected
NOTE the effect of CONVERGENCE



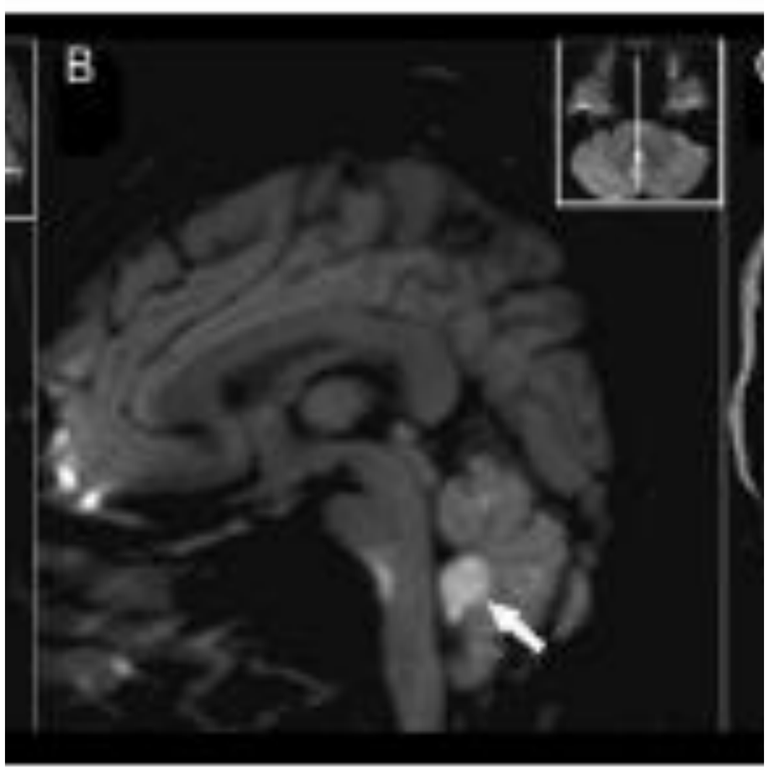
Lateral canal bppv: apogeotropic right ear affected

PEARL: Think central, if horizontal positional nystagmus is associated with a nystagmus in the upright position that has a vertical or torsional component.

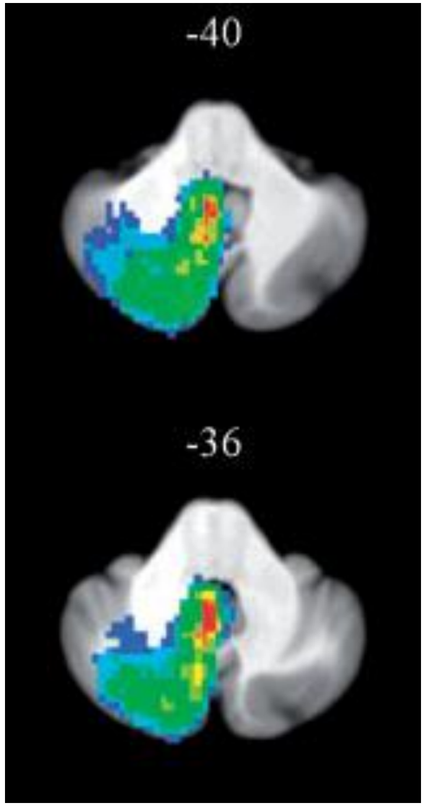
Apogeotropic (beating toward the sky) direction-changing positional nystagmus

Nam et al. 2009

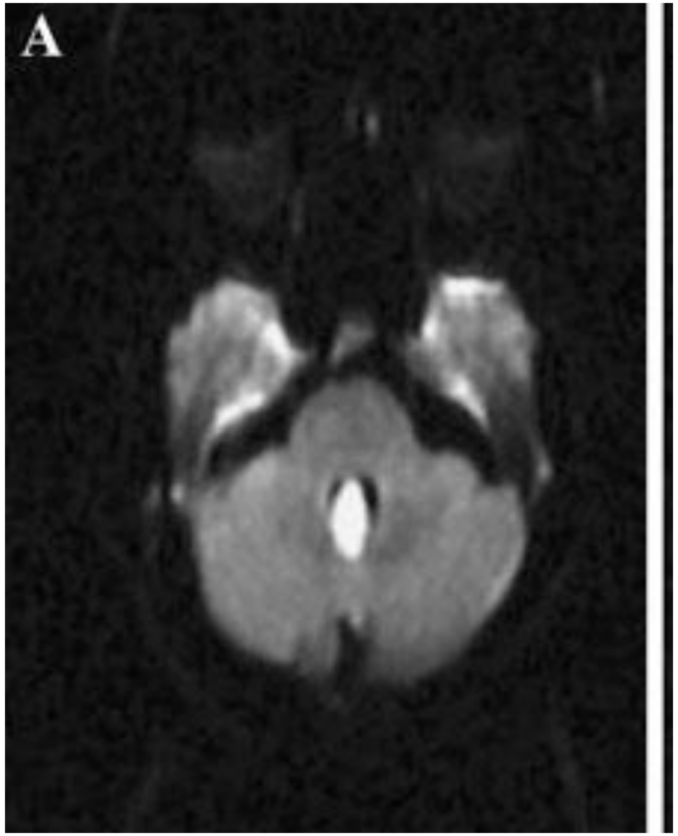
CENTRAL: Apogeotropic (beating to the sky), direction-changing, HORIZONTAL positional nystagmus



Nam et al. 2009



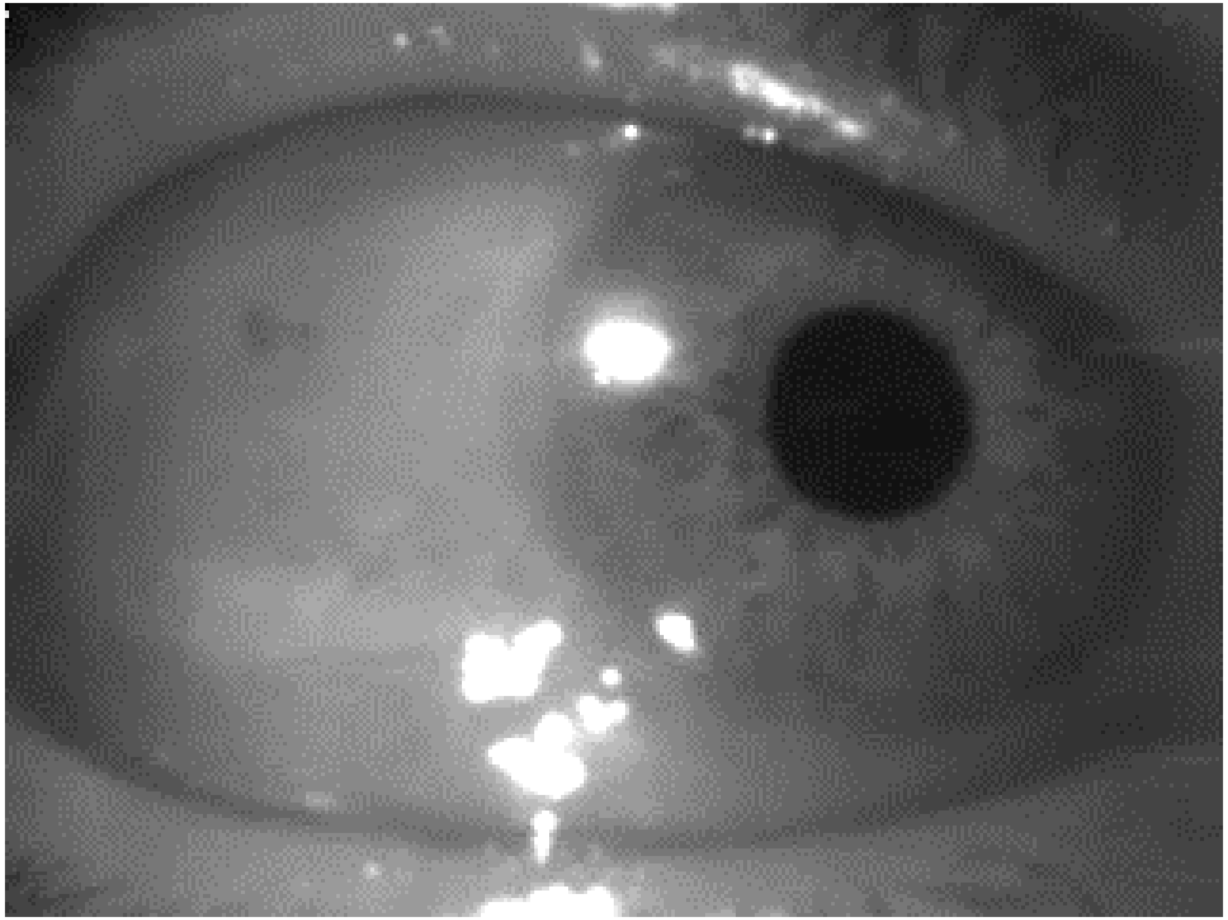
Choi et al. 2018

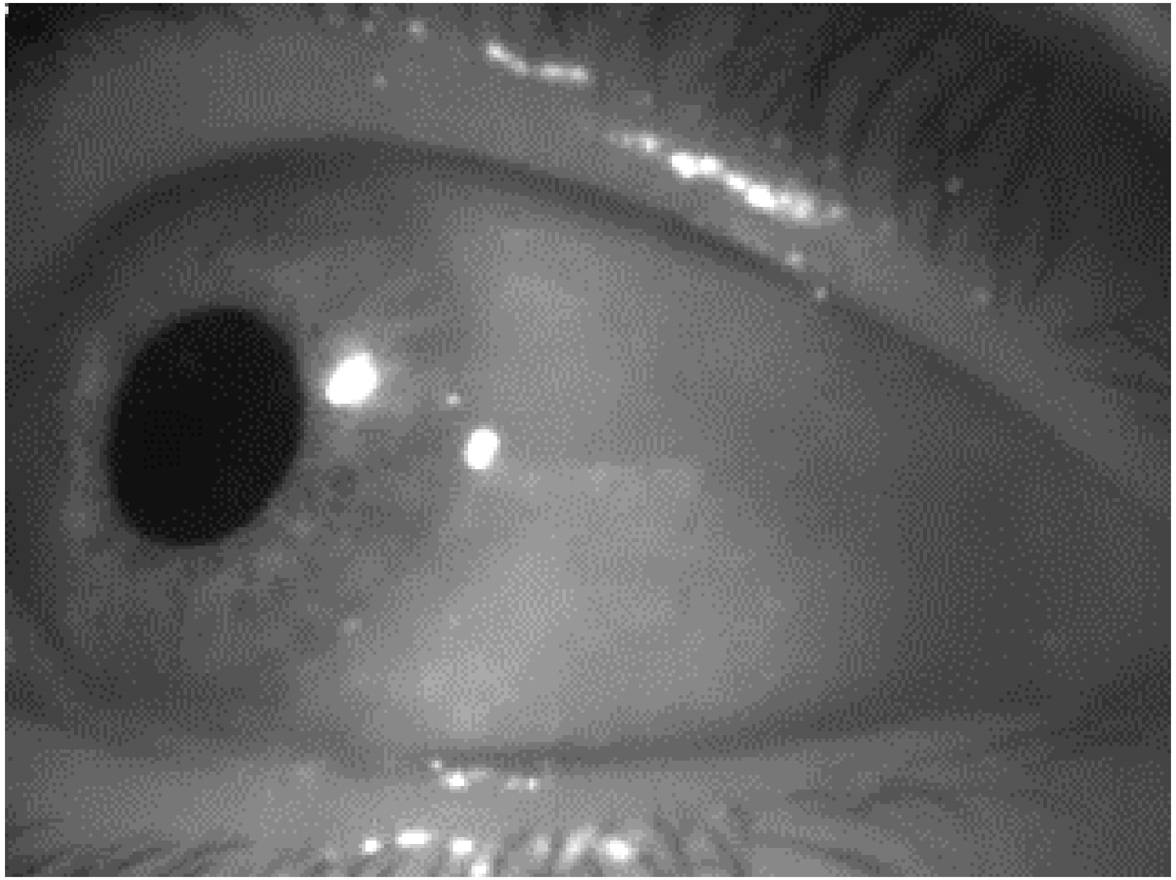


Kim et al. 2011

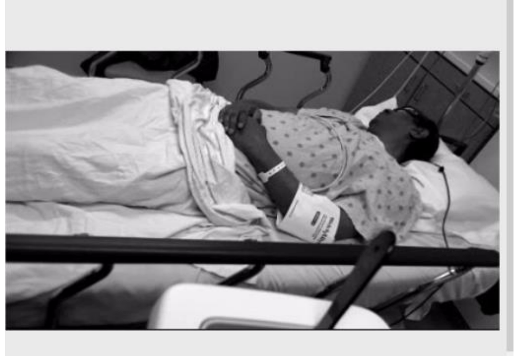
CEREBELLAR NODULUS (RED)

Lateral Canal BPPV (Left, geotropic)

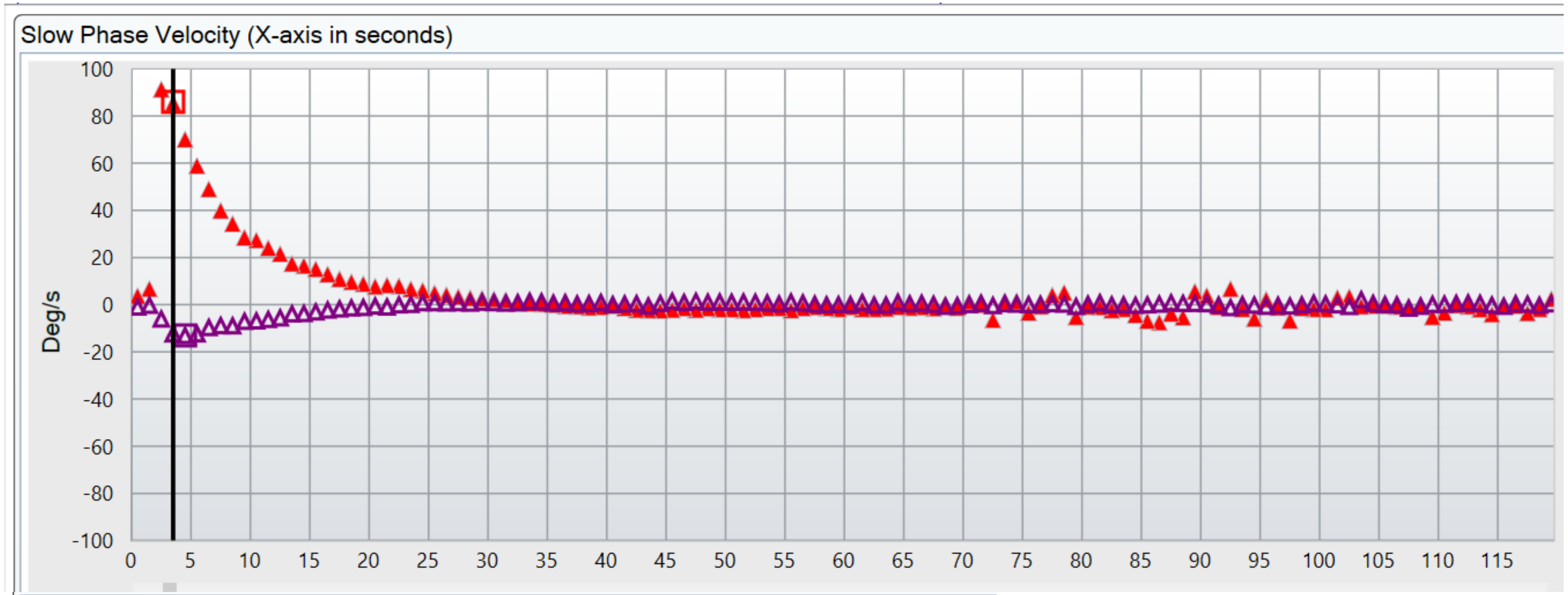




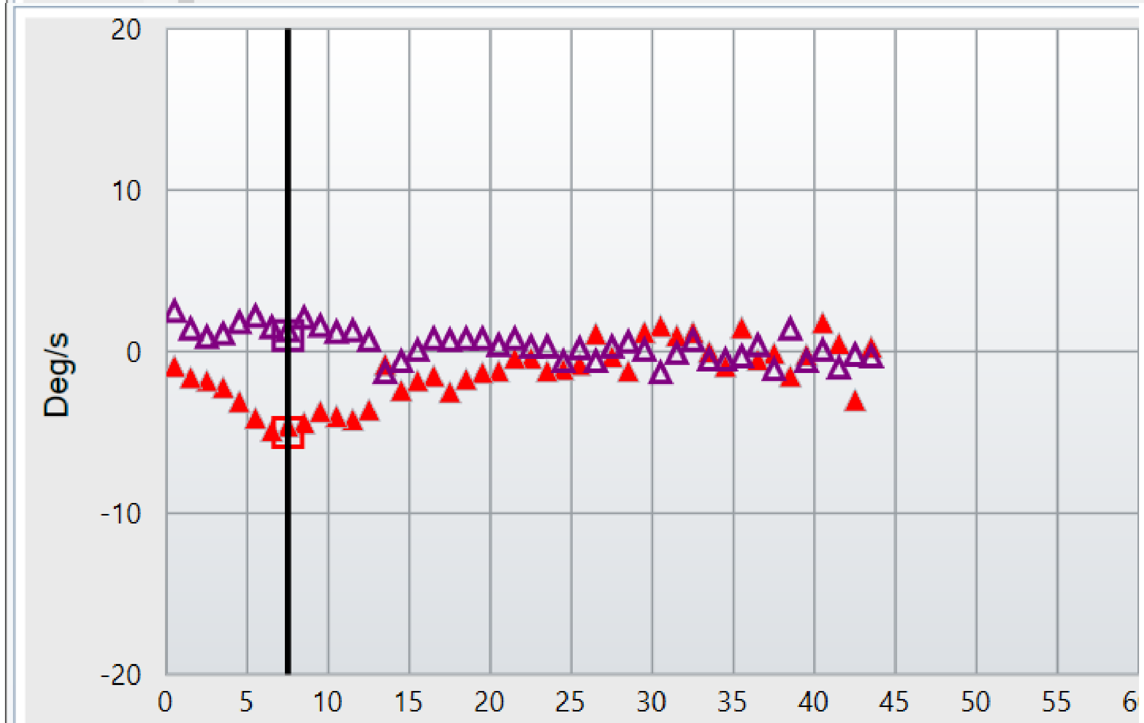
Head left



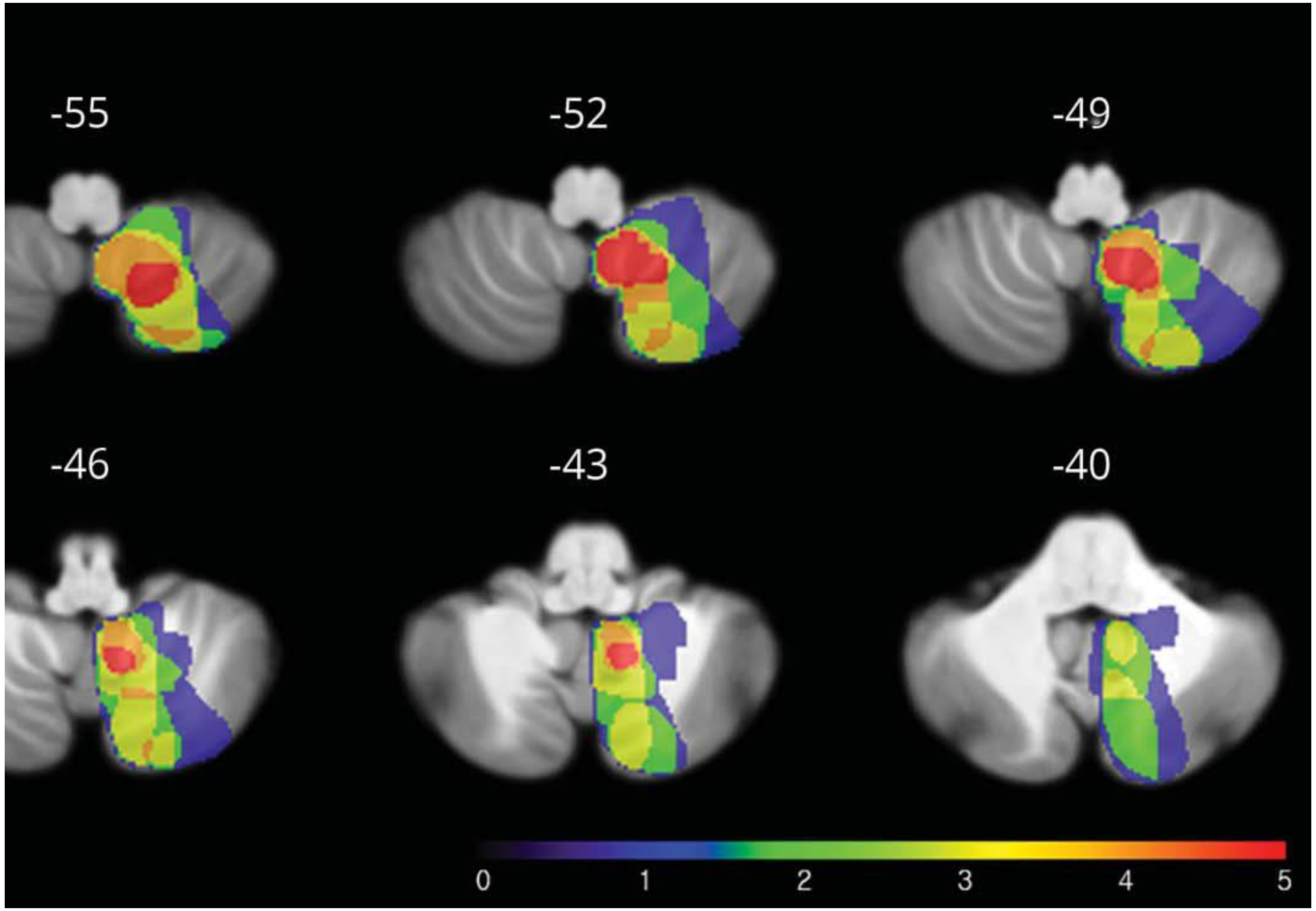
Head right



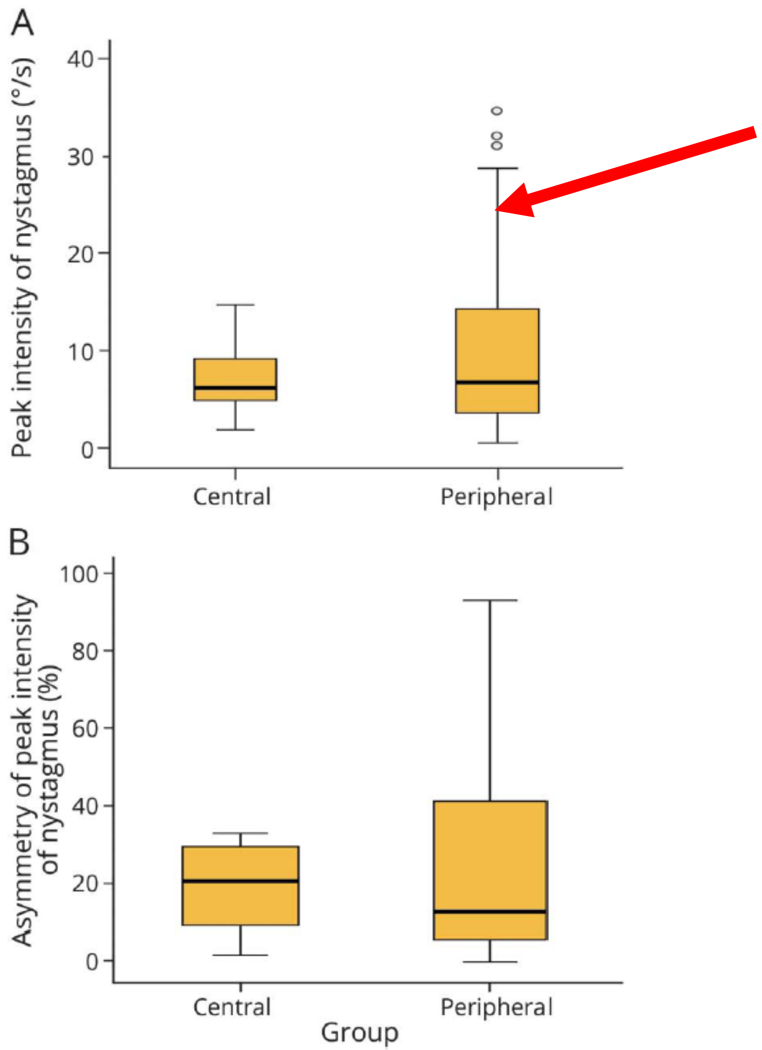
Where MOST intense will be beating toward the affected ear



CENTRAL Horizontal Positional Nystagmus: Geotropic (beat to the ground), direction-changing



CEREBELLAR TONSIL (RED)



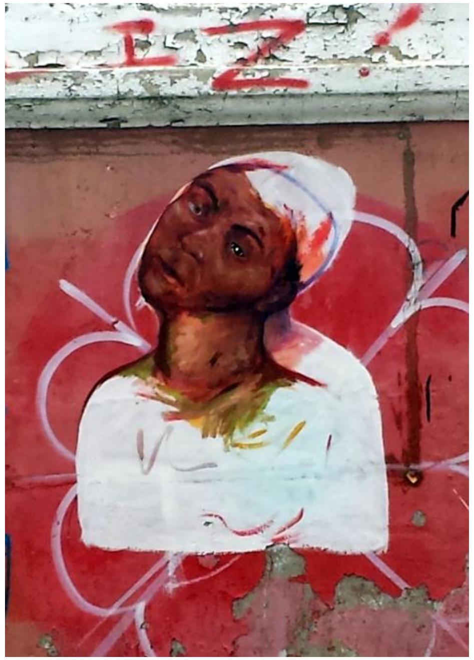
When central BILATERAL impaired pursuit

IF *VERY* high Intensity probably peripheral

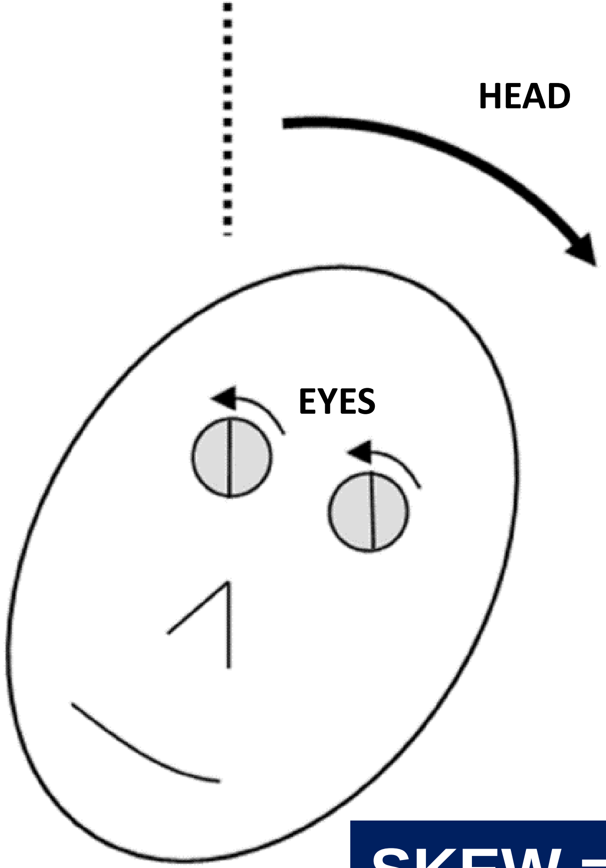
Choi, et al Neurology, 2018

Ocular counterroll (OCR), torsion)
in response to static head tilt: Otolith-ocular reflex

Valparaiso Chile



Tessa the golden retriever, Munich



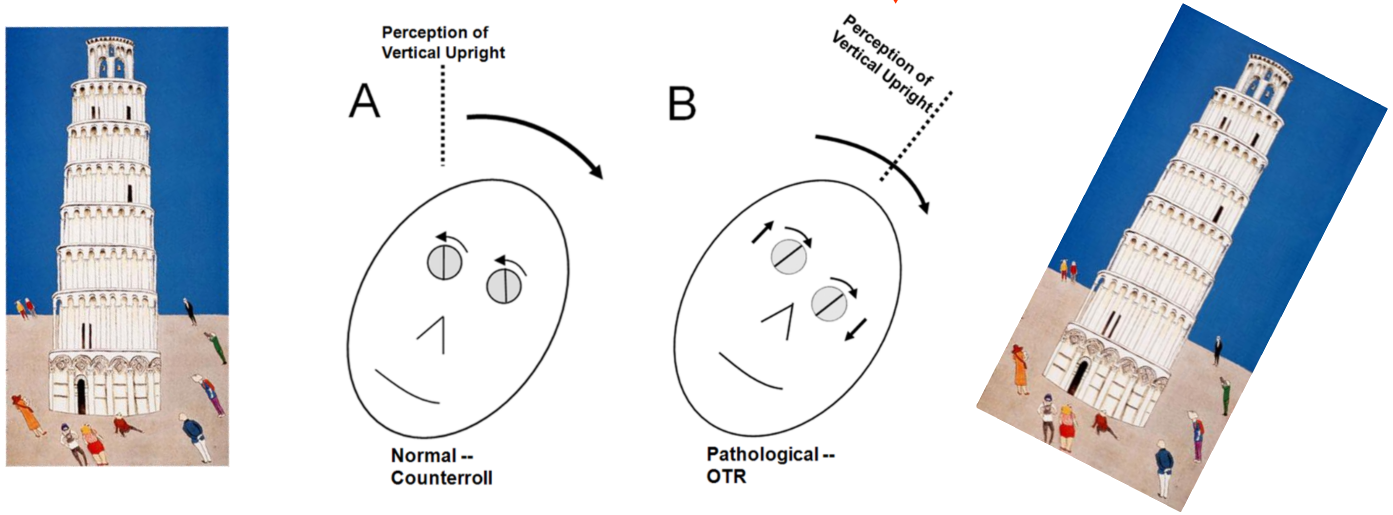
A vestigial reflex, amplitude of counterroll is only about 10% of head tilt but plays a MAJOR ROLE in pathology (*OTR*, Ocular Tilt Reaction with *SKEW DEVIATION*)

SKEW = VERTICAL OCULAR MISALIGNMENT NOT DUE TO AN OCULAR MUSCLE WEAKNESS

Mechanism of abnormal perception of upright in OTR

In normal, frontal-eyed, foveate animals the normal response to a lateral head tilt is pure ocular counterroll or torsion without a skew

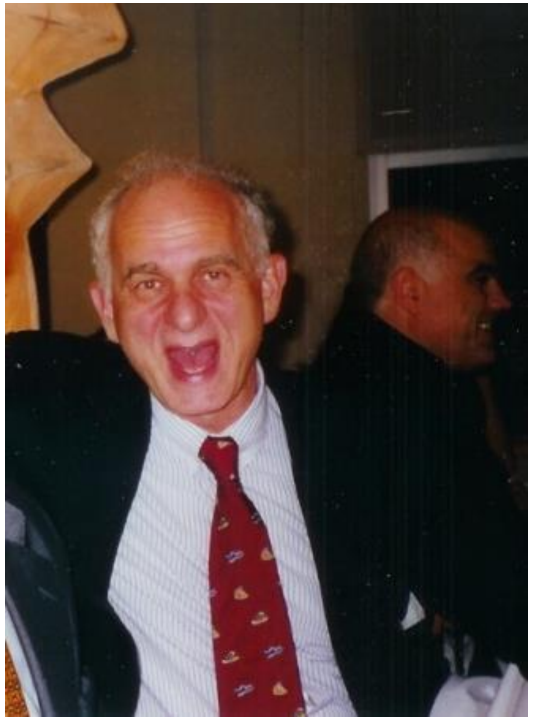
In pathology, in frontal-eyed, foveate animals the abnormal response to a perceived shift of the sense of vertical becomes a "compensatory" head tilt, counterroll and a skew deviation of the eyes that produces <u>vertical diplopia</u>



Ocular Tilt Reaction (OTR)

acute tone imbalance of static utricular righting reflexes (analogous to spontaneous nystagmus from a semicircular canal imbalance)

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

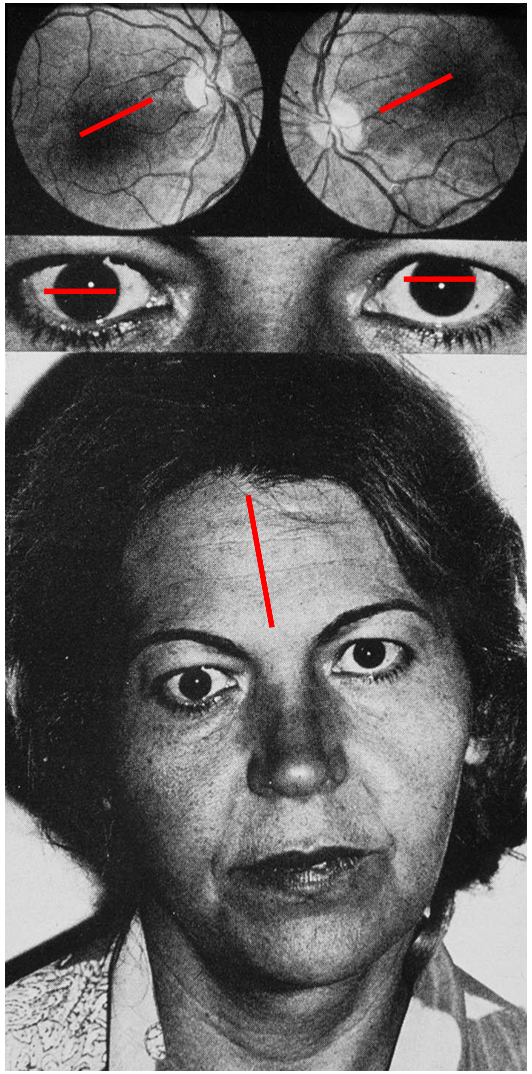


Michael Halmagyi

Counterroll

Skew

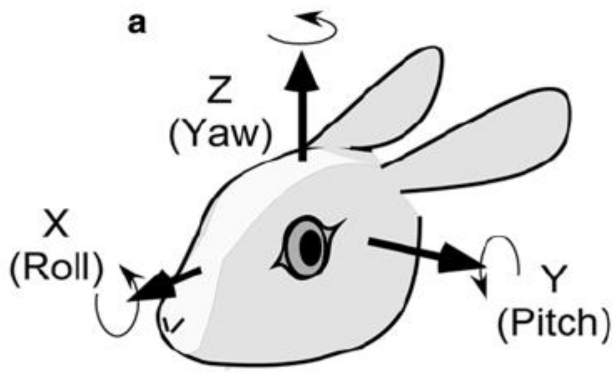
Head Tilt

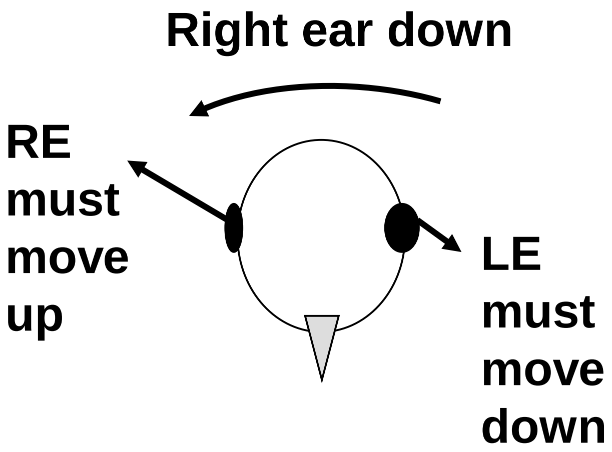


Ann Neuro 1979

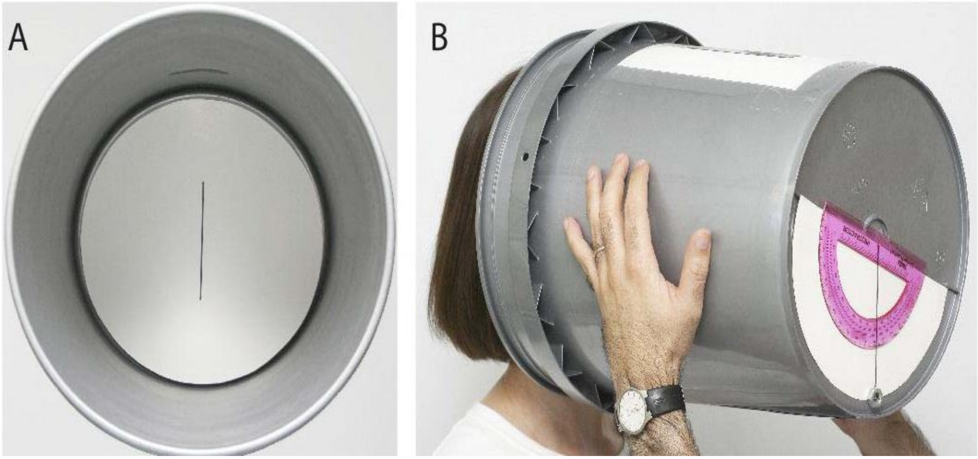
Why a vertical misalignment (skew) in the OTR?

- Emergence of a phylogenetically-old, ocular righting response to lateral tilt.
- In the lateral-eyed rabbit, a lateral tilt (one ear up and the other down) leads to the eyes rotating around the *roll axis* with one eye rotating down and the other up (a physiological skew as part of a normal OTR)
- When there is an imbalance in otolith-ocular pathways in humans, the physiological skew of the rabbit emerges as the pathological OTR



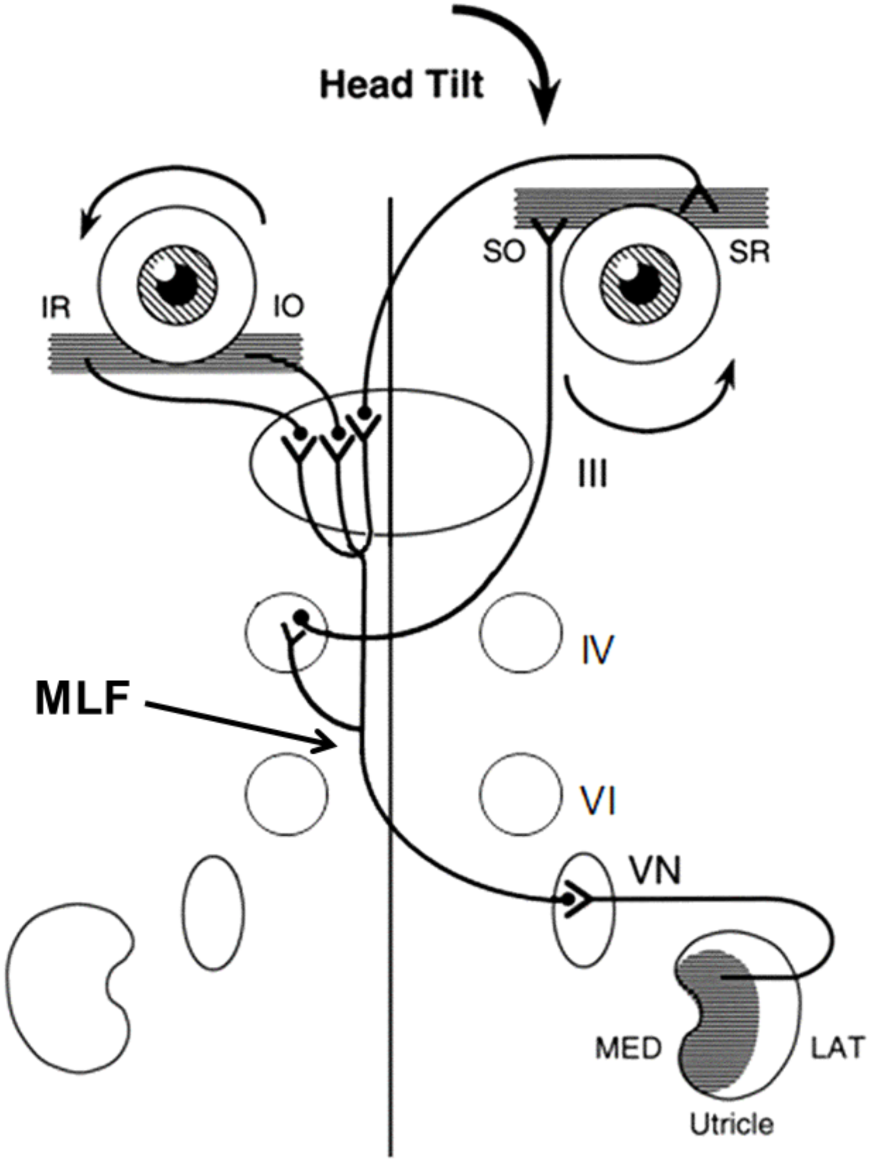


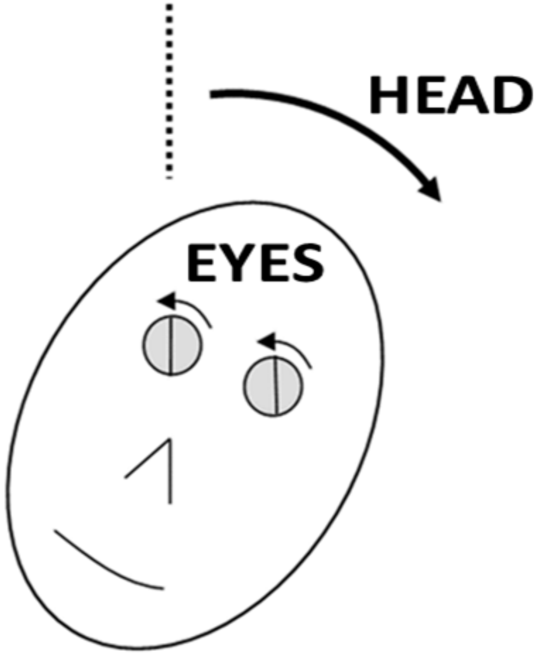
Bucket technique for Subjective Visual Vertical (SVV)
(as a measure of torsion (ocular counterroll))



After Zwergal, Neurology, 09, and Frisen, Neuro-ophthalmology, 2000

Utricular-ocular pathways <u>subserving head tilt</u>

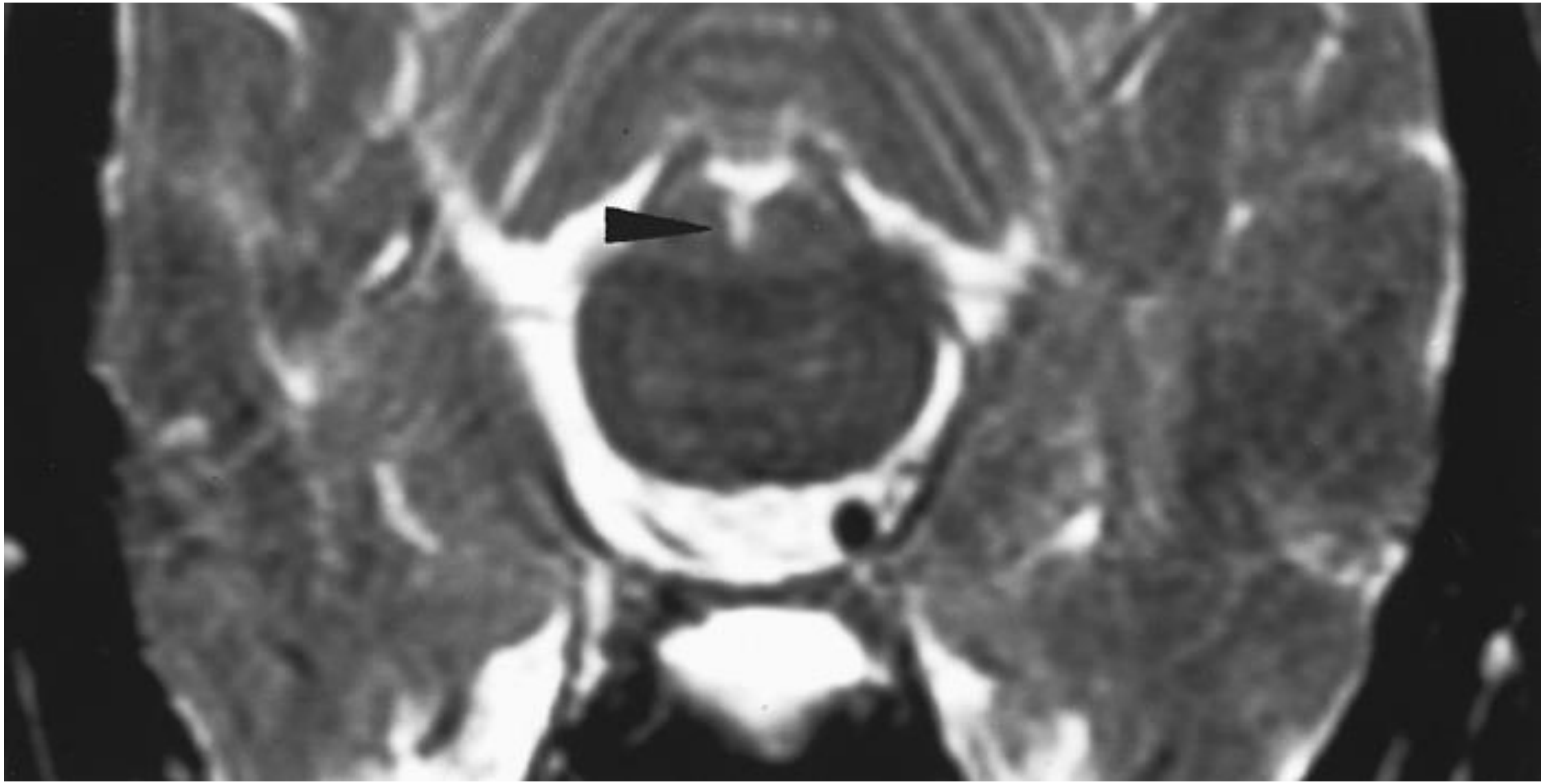




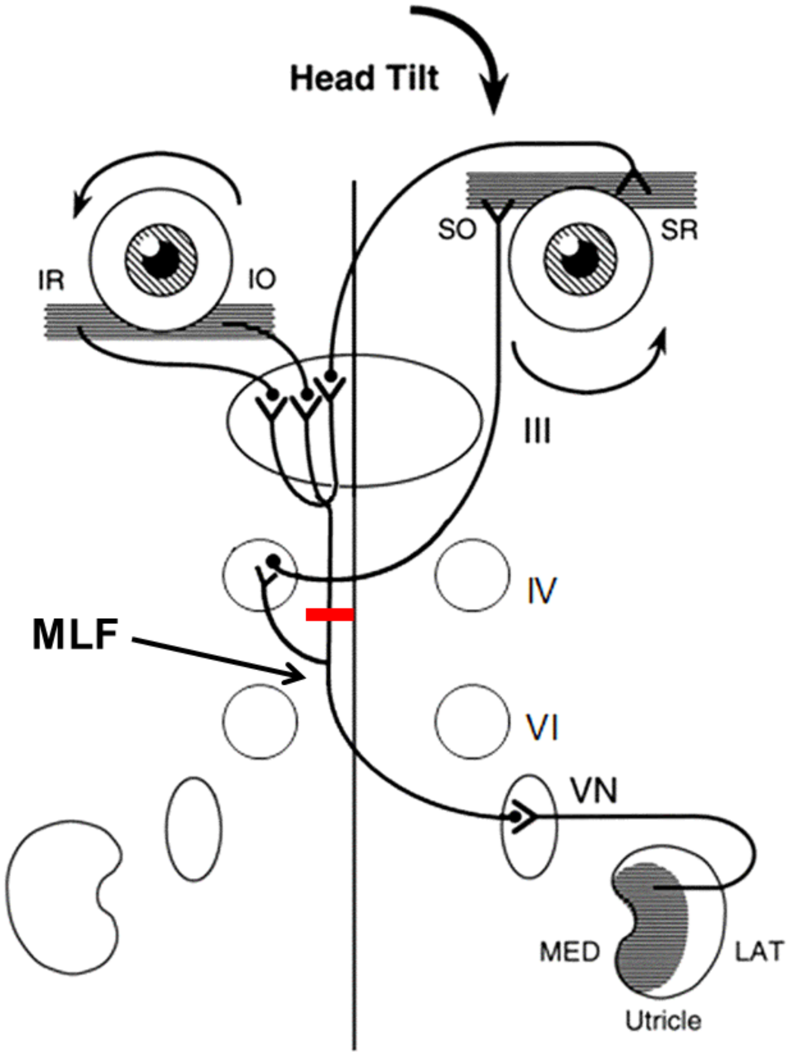
Left hyperdeviation and right head tilt

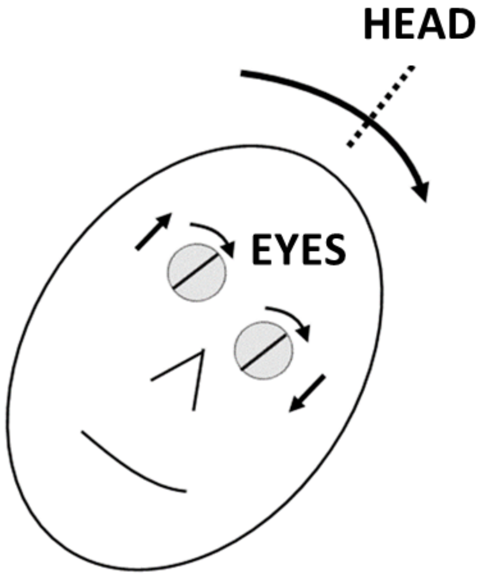
Medial longitudinal fasciculus (MLF) lesion

Internuclear ophthalmoplegia (INO)

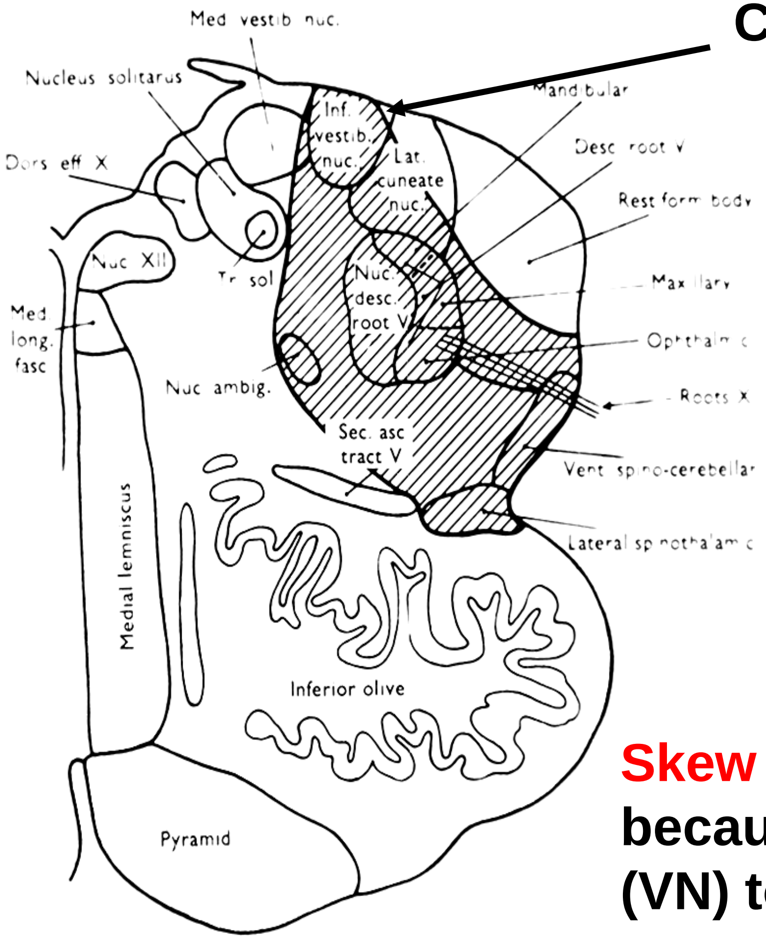


Ocular Tilt Reaction (OTR) in MLF lesions (INO) (skew with eye higher on side of the lesion) due to involvement of utricular-ocular pathways AFTER they cross the midline and transverse the MLF

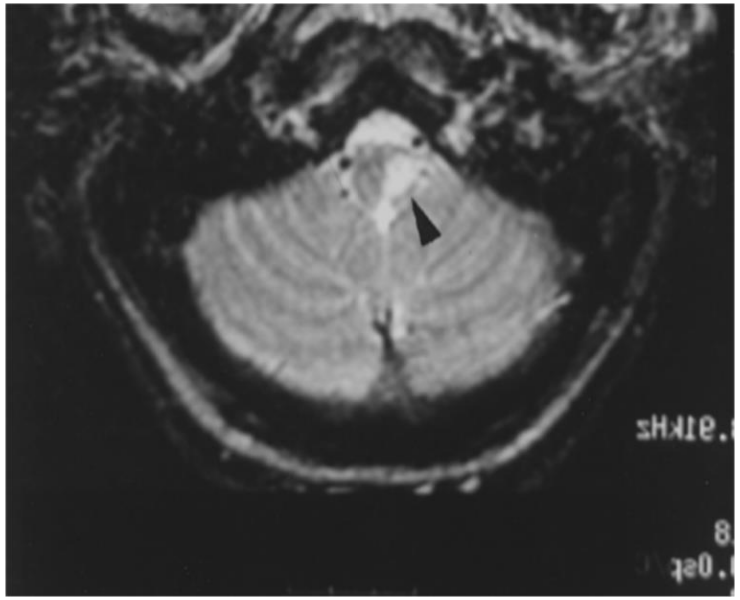




Wallenberg's Syndrome – Posterior Inferior Cerebellar Artery (PICA) distribution infarct involving the dorsolateral medulla



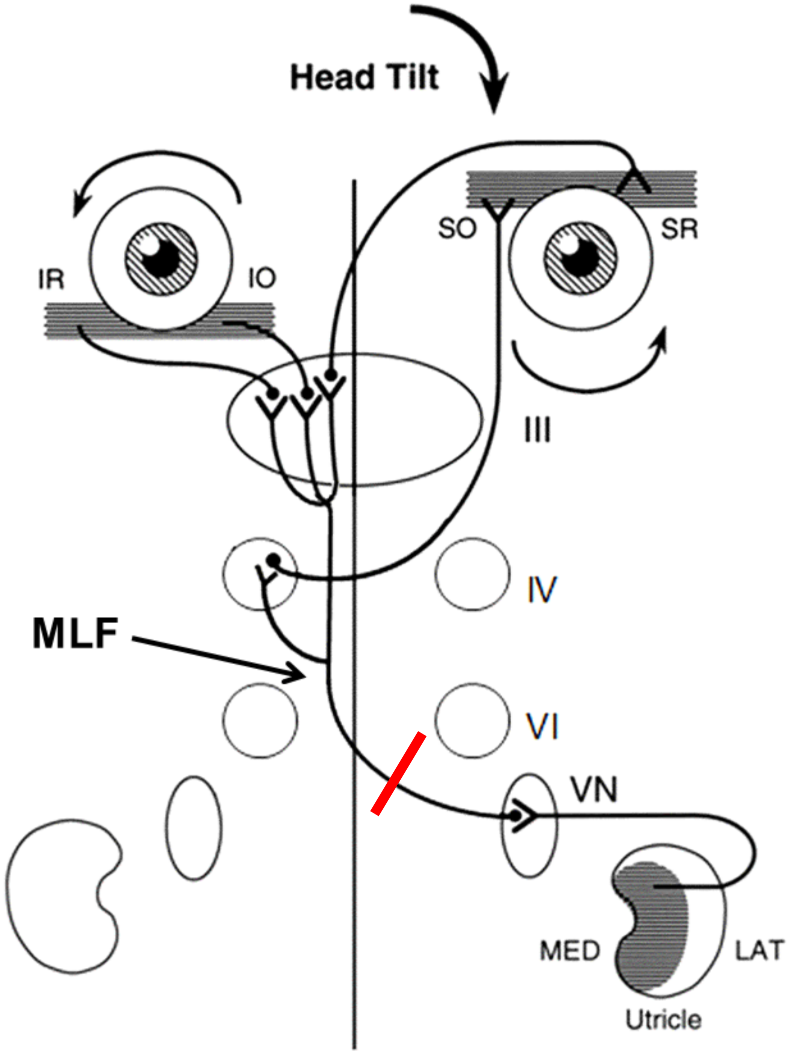
Caudal vestibular nuclei

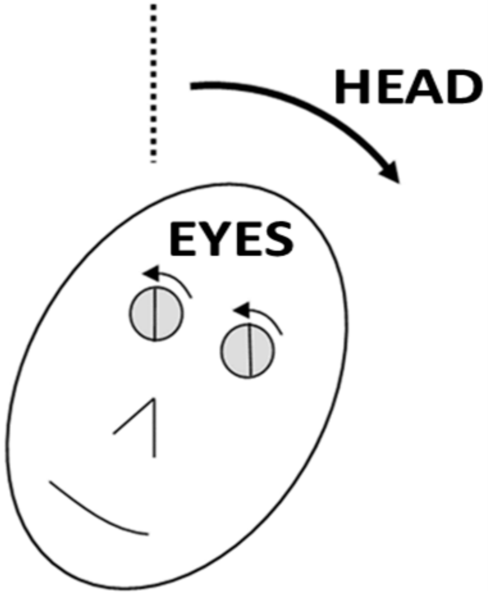


Skew with lower eye on the side of the lesion occurs because of involvement of the caudal vestibular nuclei (VN) to which project mostly otolith inputs.

Skew is part of the OTR (ocular tilt reaction) with a skew, head tilt and ocular counterroll.

Ocular Tilt Reaction (OTR) in lesions in the vestibular nuclei (VN, e.g. Wallenberg syndrome) (skew with eye lower on side of the lesion) due to involvement of utricular-ocular pathways BEFORE they cross the midline and transverse the MLF



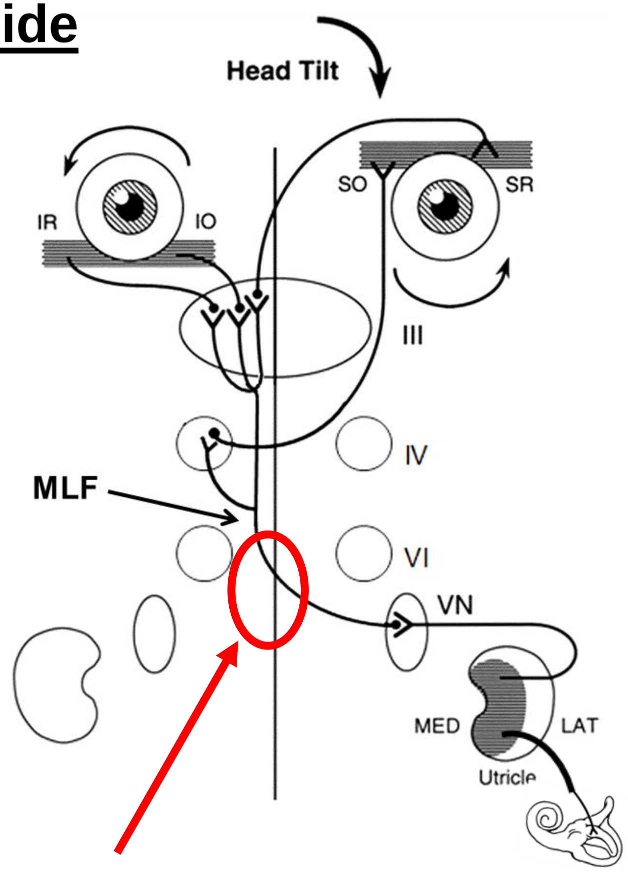


Localization of Skew in Ocular Tilt Reaction (OTR)

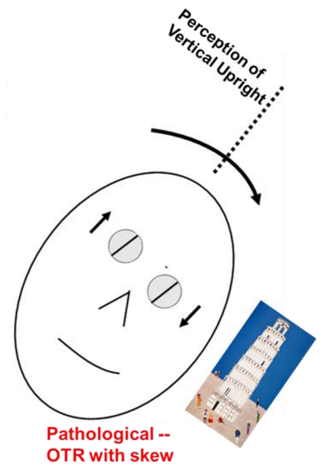
EYE HIGHER on lesion side

Interstitial Nucleus of Cajal (Midbrain)

Medial Longitudinal Fasciculus (INO)



CROSSING of otolith-ocular pathway

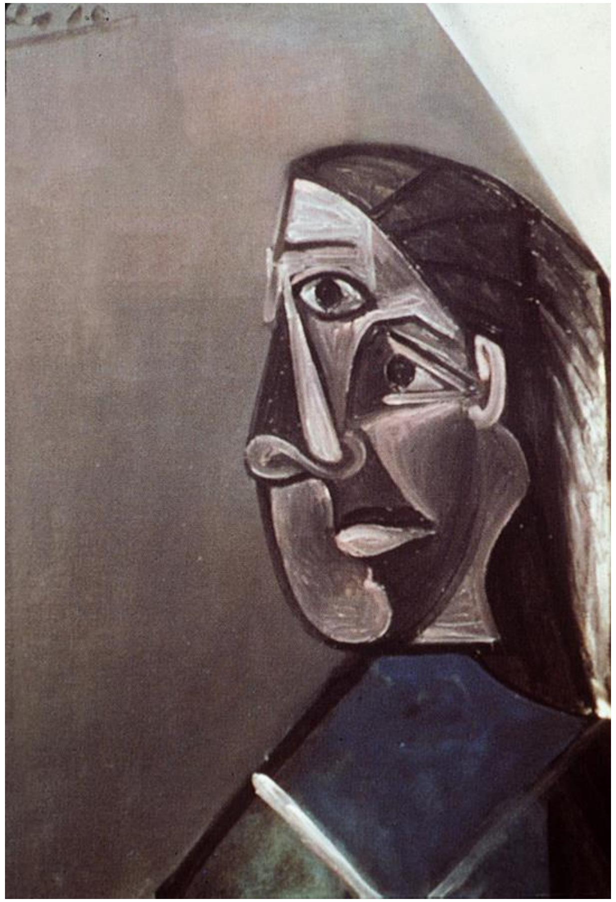


Vestibular nucleus (VN) lesion (e.g., Wallenberg's, PICA)

Vestibular (VIII) nerve lesion (BUT small and transient)

EYE LOWER on lesion side

The alignment changes in patients with cerebellar disease



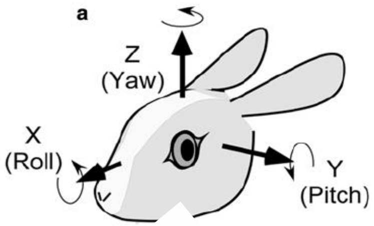
- Esodeviation (eyes turn in with distance viewing, mimics a divergence paralysis): Horizontal Diplopia
- ‘Skew’ (vertical misalignment (alternating hyperdeviation, usually abducting eye is higher)): Vertical Diplopia

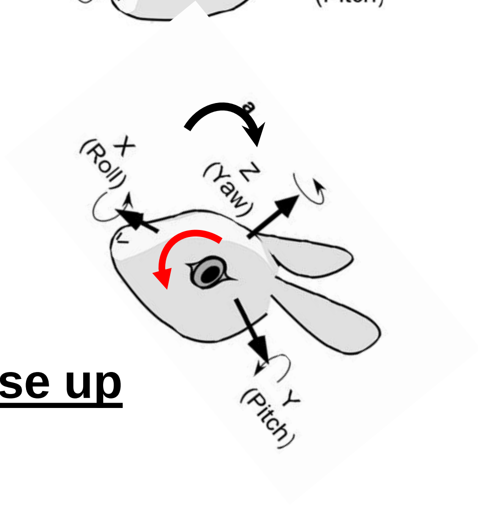
Alignment changes in cerebellar disease

Alignment changes in cerebellar disease

Alignment changes in cerebellar disease

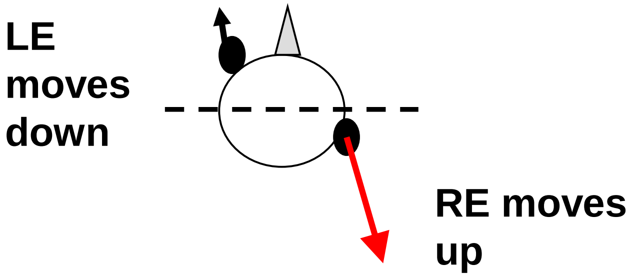
Alternating Skew in Cerebellar Patients: A misinterpretation of head pitch in a "lateral-eyed" animal?

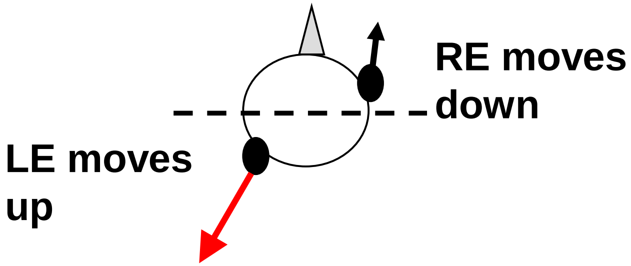




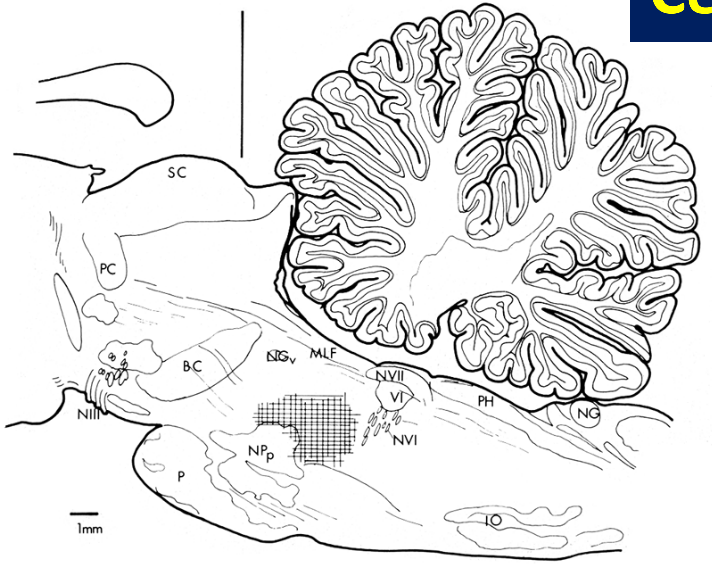
Nose up

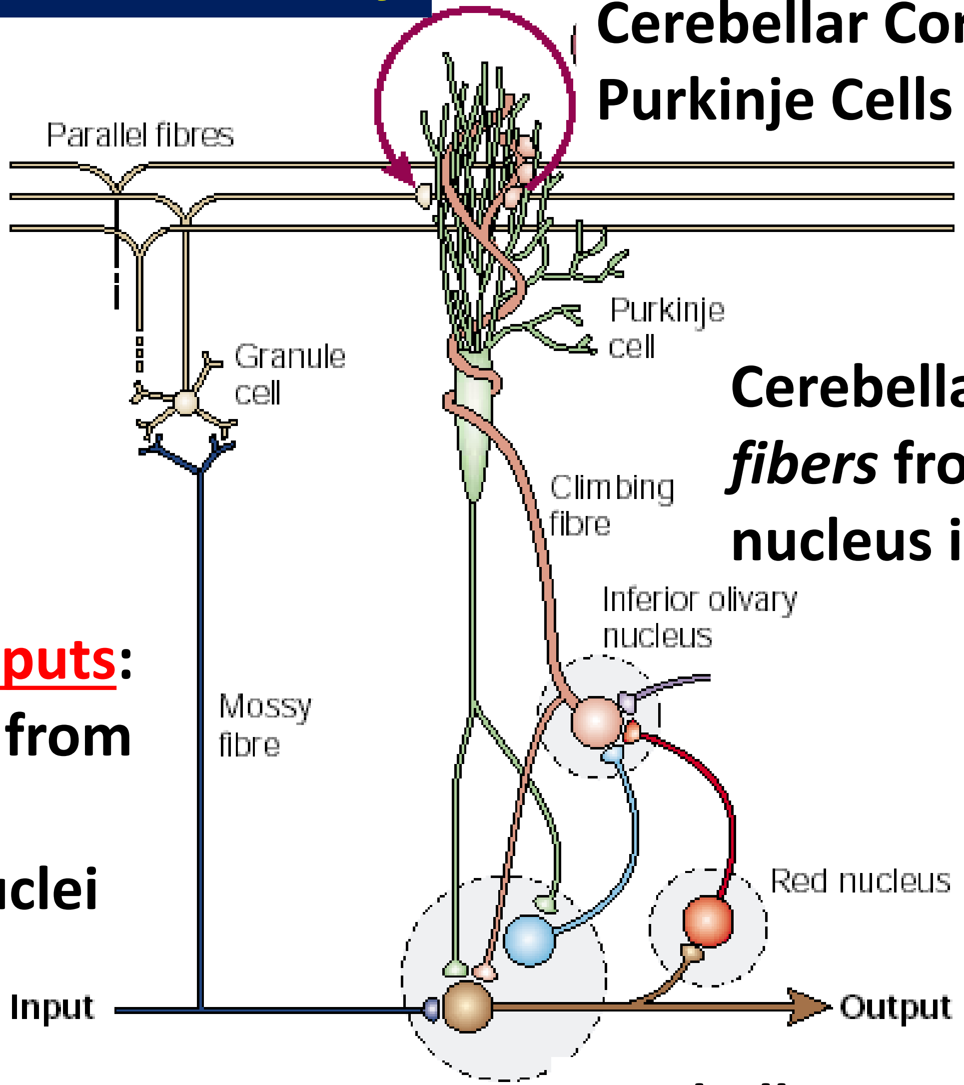
Top View





Cerebellar Circuitry





Cerebellar Cortex, Purkinje Cells

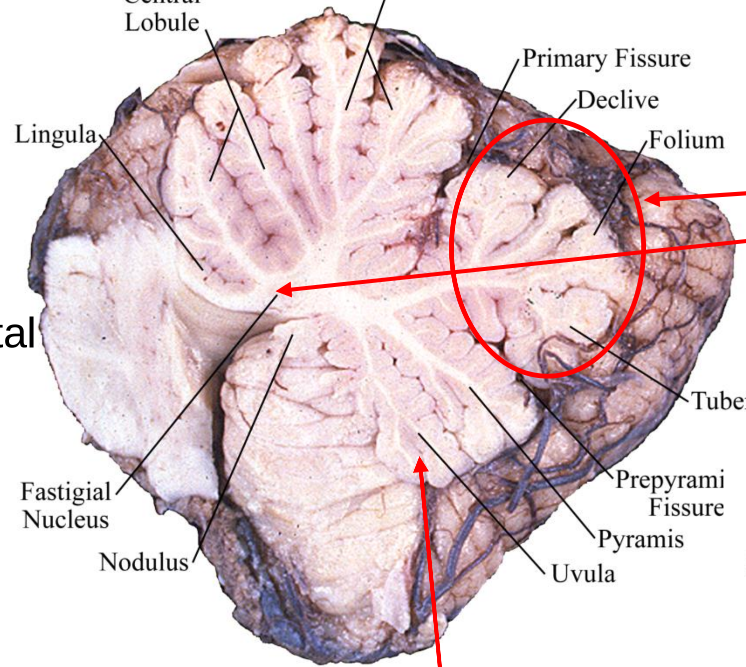
Cerebellar Inputs: *climbing fibers* from inferior olivary nucleus in medulla

Cerebellar Inputs: *mossy fibers* from pontine and vestibular nuclei

Cerebellar Cortex Outputs: deep cerebellar (and vestibular) nuclei

KEY ANATOMY: Three basic functional-anatomical cerebellar syndromes

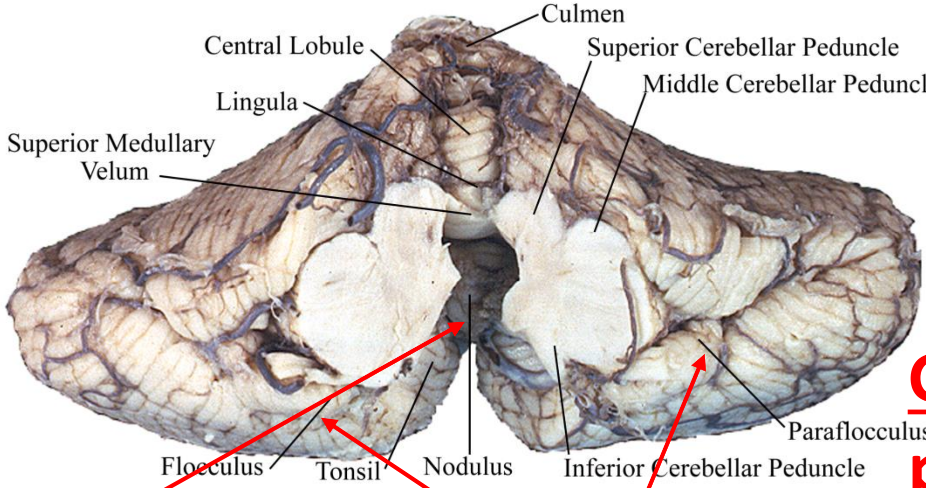
Sagittal View



Syndrome of the dorsal vermis (OMV) & posterior fastigial nucleus (FOR)

Saccade accuracy

Inferior View



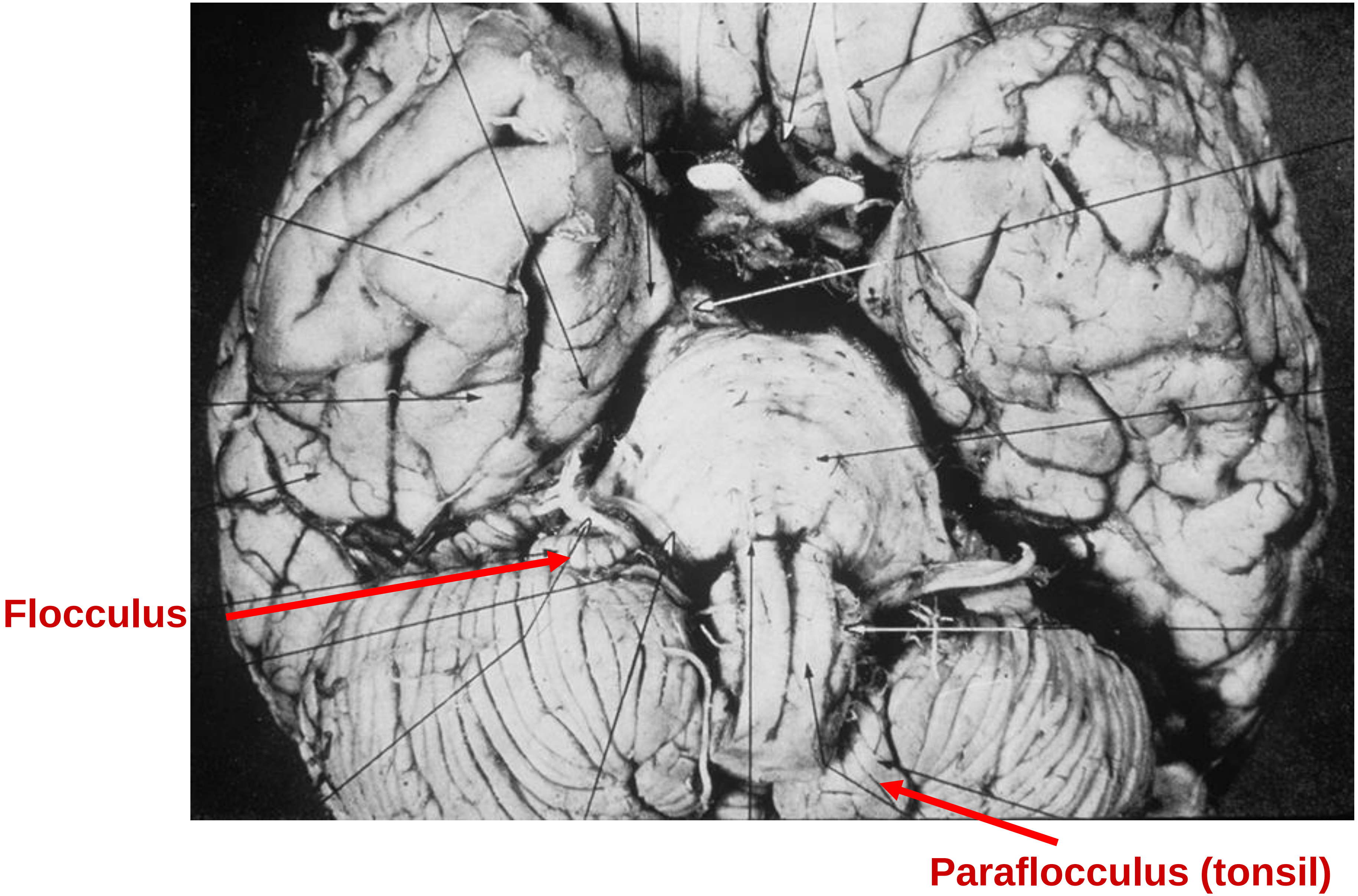
Vestibular (low-freq, slowly changing)

Syndrome of the nodulus & ventral uvula

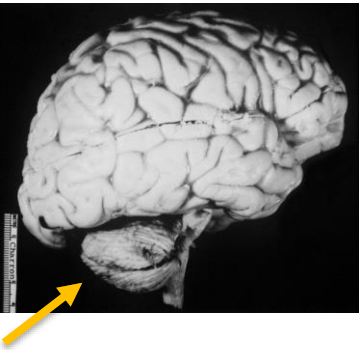
Syndrome of the flocculus and paraflocculus (tonsil)

Gaze-holding, pursuit, vestibular (high-freq, rapidly changing)

Cerebellar flocculus and paraflocculus (tonsils)



Flocculus/Paraflocculus syndrome: Downbeat, gaze-evoked and rebound nystagmus in cerebellar atrophy

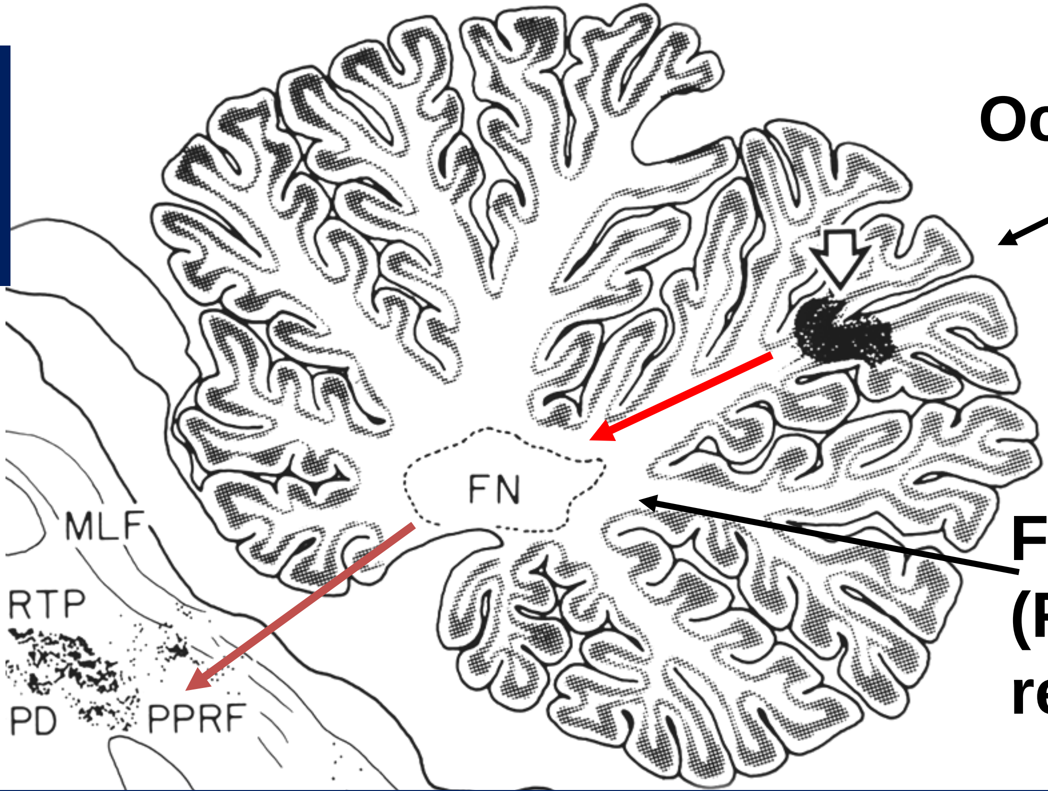


Cerebellar atrophy: SCA6

Flocculus/Paraflocculus syndrome
Impaired pursuit and vestibuloocular reflex (VOR) cancellation (fixation suppression)

Pursuit and VOR cancellation

Cerebellum and saccades



Ocular Motor Vermis

Fastigial Nucleus (Fastigial oculomotor region, called the FOR)

REMEMBER

1) The dorsal vermis contains Purkinje cells and they INHIBIT their target neurons in the posterior fastigial nucleus (FOR)
2) Each FOR projection crosses immediately and then runs astride the contralateral superior cerebellar peduncle
3) Each FOR normally acts to stop ipsilateral saccades by stimulating inhibitory burst neurons (IBN)

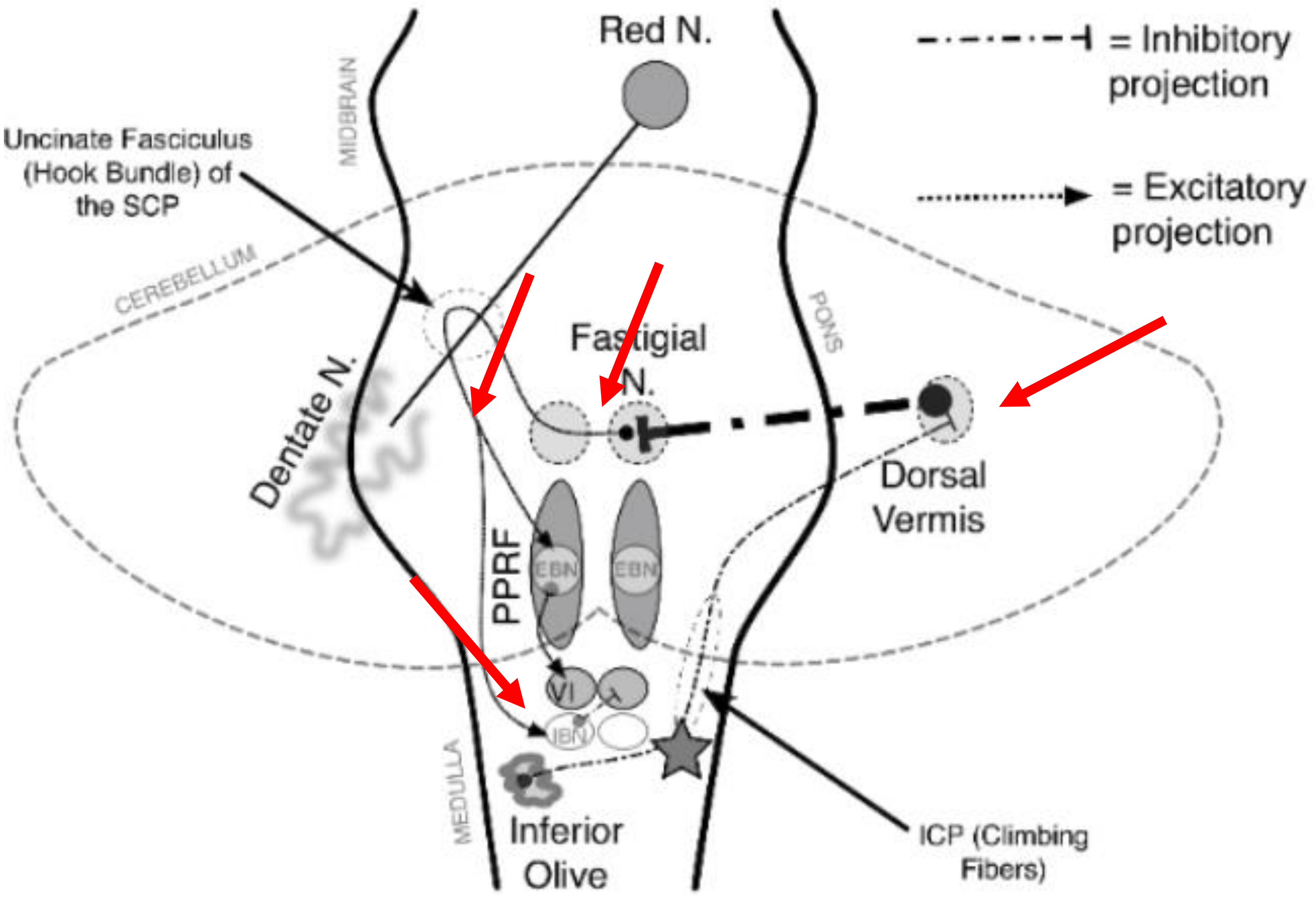
DO NOT PANIC WHEN YOU SEE THE NEXT SLIDES.

There is (clinical) LIGHT at the end of the (anatomical) TUNNEL

Purkinje cells inhibit deep nuclei

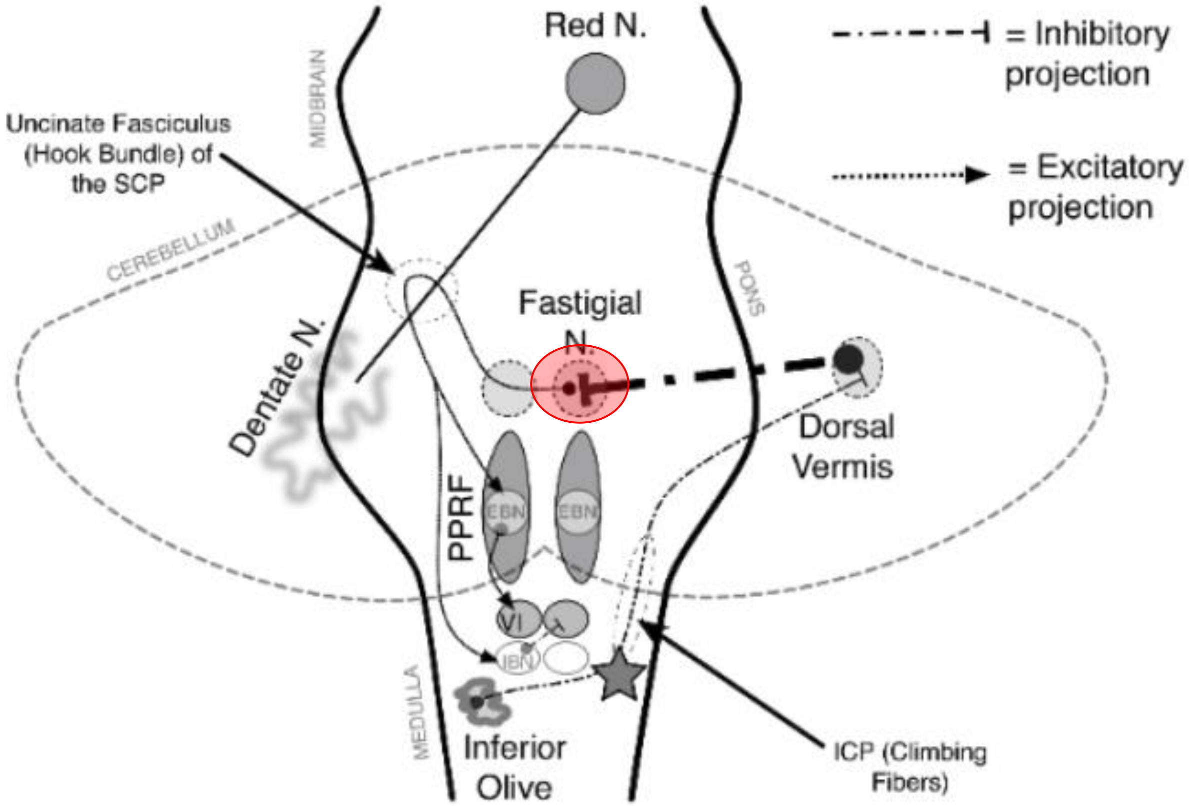
FOR axons immediately cross and project to contralateral brainstem where they normally drive ***inhibitory and excitatory saccade burst neurons***

Activity in ***inhibitory saccade*** burst neurons (the axons of which cross to the abducens nucleus ipsilateral to the FOR) normally work to facilitate contralateral saccades and inhibit ipsilateral saccades



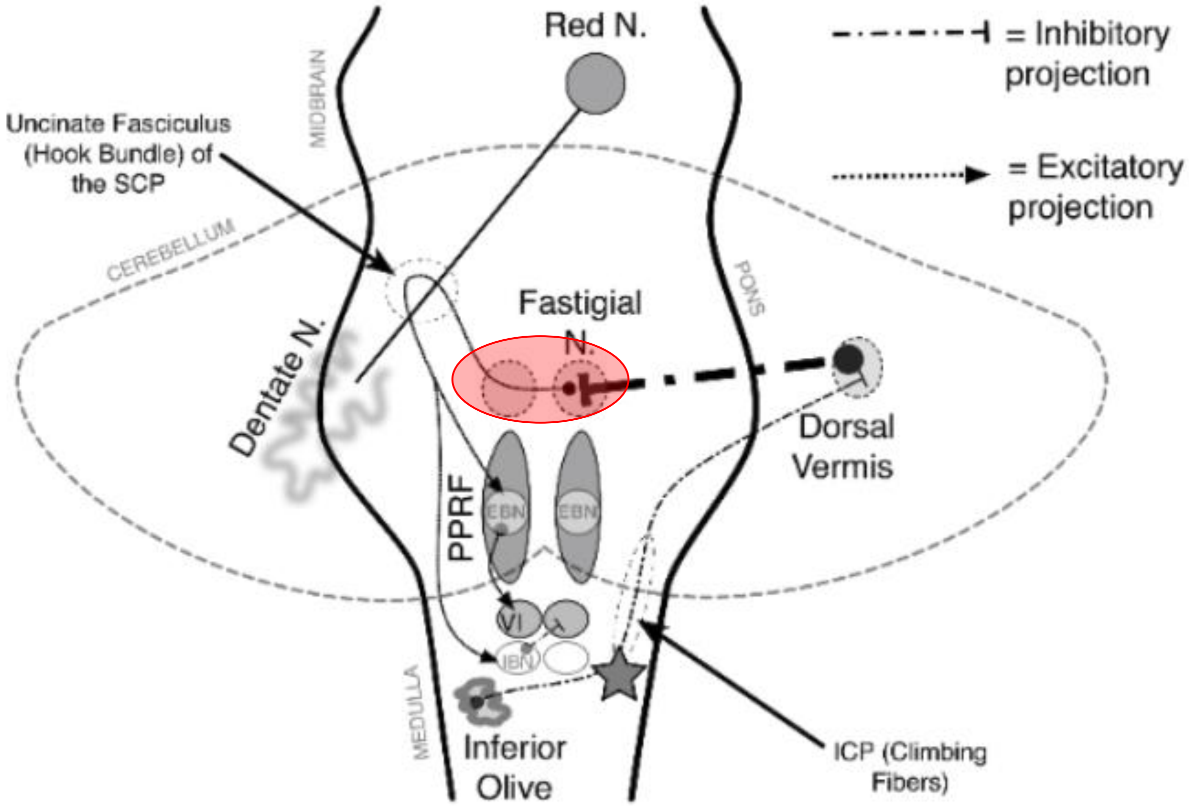
Cerebellum and saccades

- **Fastigial nucleus (FOR)**
  - **Unilateral lesions cause *<u>overshooting ipsiversive</u>* and *<u>undershooting contraversive</u>* saccades primarily because inhibitory burst neurons no longer inhibit ipsiversive saccades**
    - **(NOTE a *<u>unilateral</u>* *<u>structural</u>* lesion is not possible because of immediate crossing of FOR axons. Selective neurotoxins can produce unilateral lesions experimentally)**



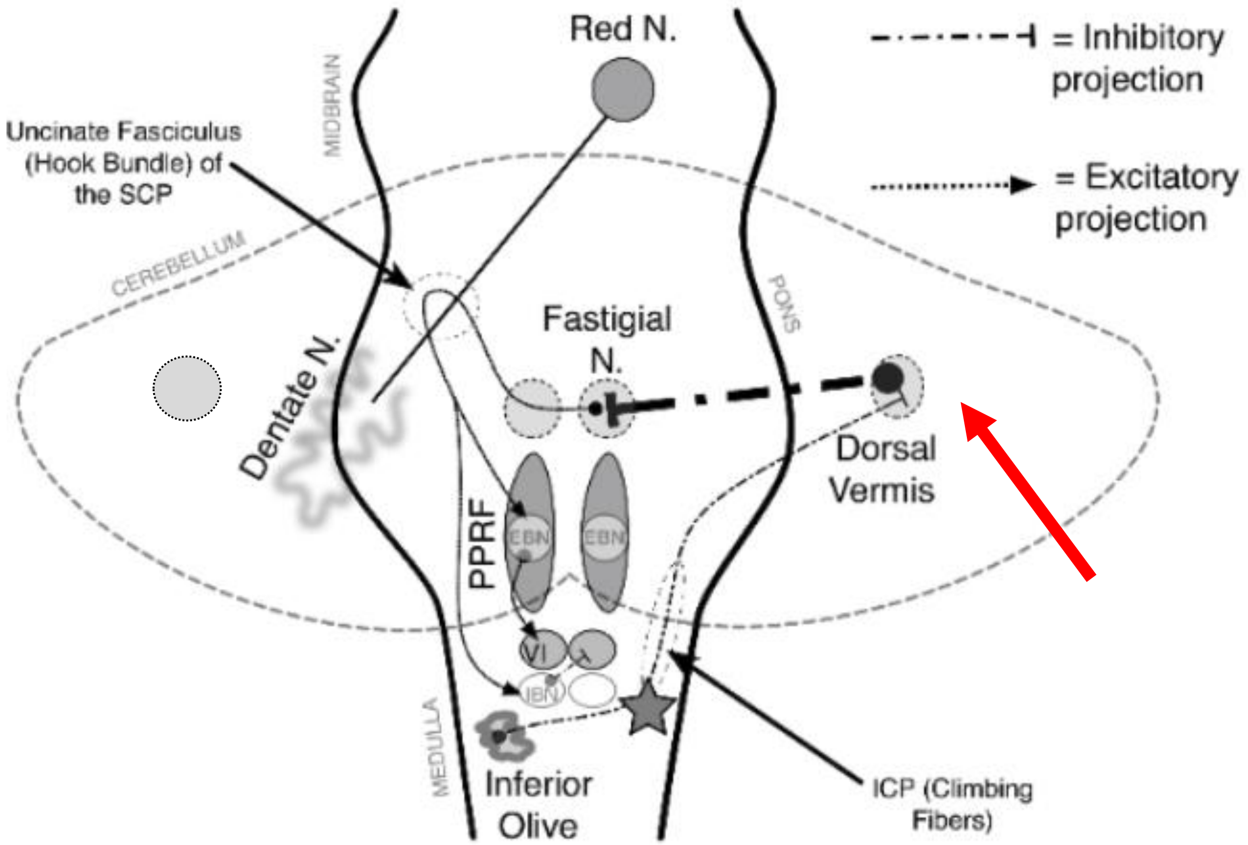
Cerebellum and saccades

- Fastigial nucleus (FOR)
  - Unilateral lesions cause ***overshooting ipsiversive*** and ***undershooting contraversive*** saccades primarily because inhibitory burst neurons no longer inhibit ipsiversive saccades
    - (NOTE a unilateral ***structural*** lesion is not possible because of immediate crossing of FOR axons. Selective neurotoxins can produce unilateral lesions experimentally)
  - Bilateral lesions cause ***HYPERMETRIC*** saccades



Cerebellum and saccades

- Dorsal cerebellar vermis
  - Inhibits fastigial nucleus on the same side (Purkinje cells are inhibitory)
  - Lesions of the vermis have the opposite effect as fastigial nucleus lesions



Cerebellum and saccades

- Dorsal cerebellar vermis
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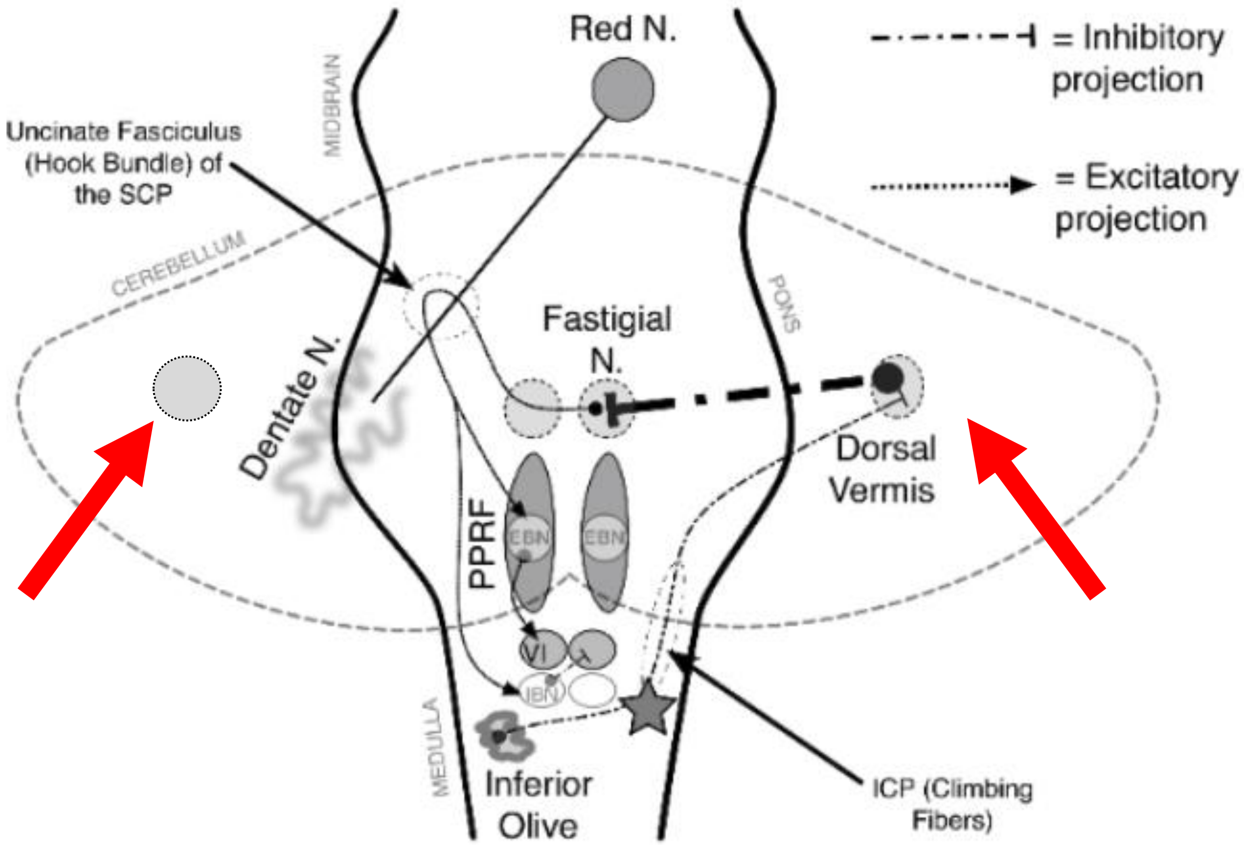
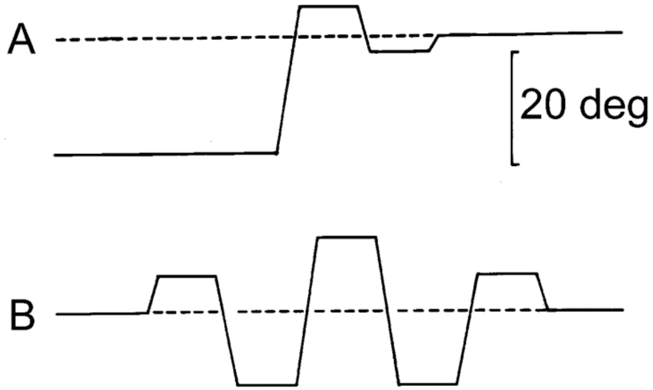
  - Bilateral lesions cause ***HYPOMETRIC*** saccades

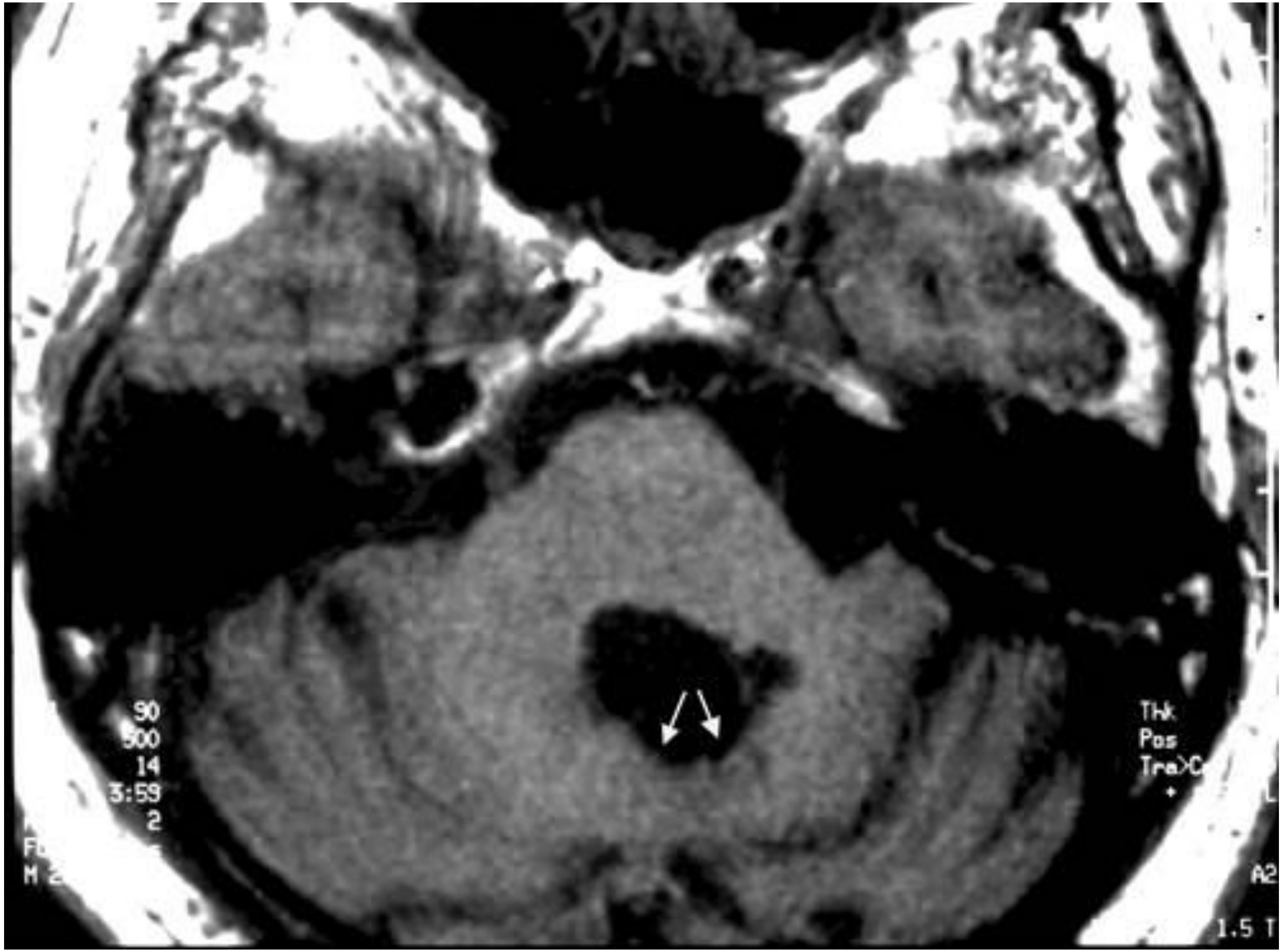


Saccadic overshoot dysmetria with macrosaccadic oscillations



Macrosaccadic Oscillations

Cerebellar fastigial nucleus lesions produce saccade *hypermetria*

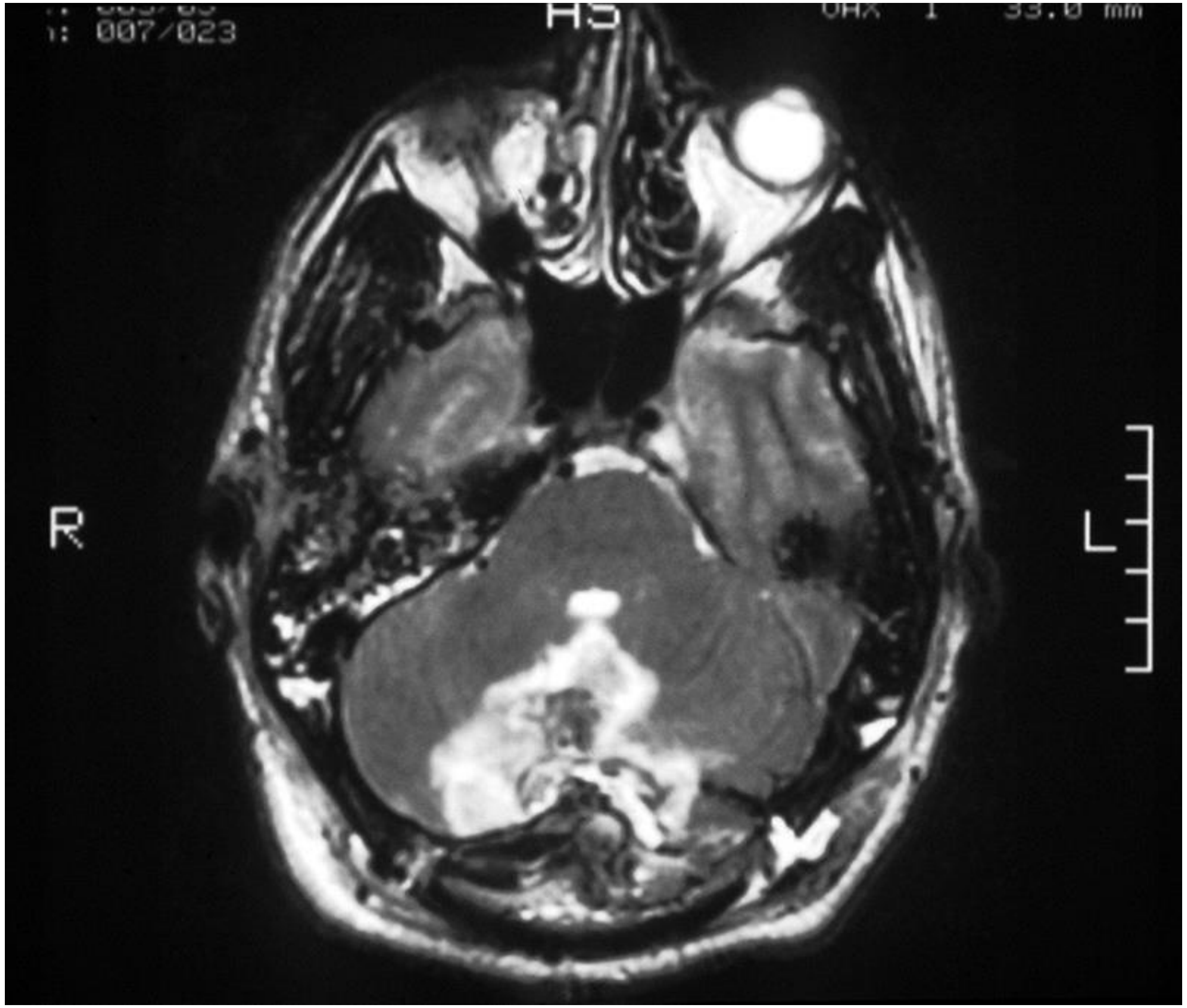


Saccadic undershoot dysmetria

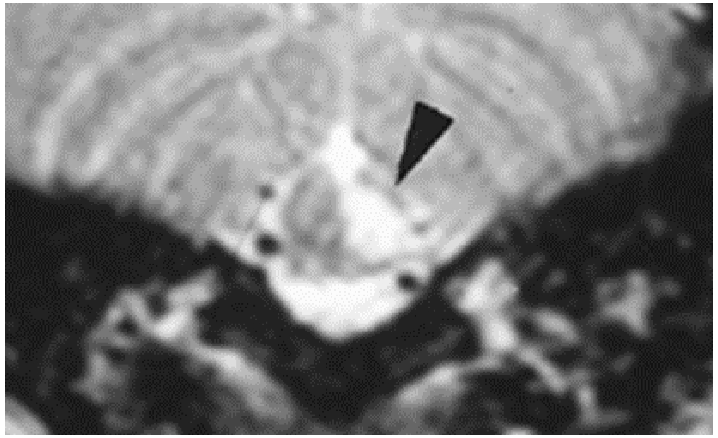
Cerebellar dorsal vermis lesions produce saccade *hypometria*

Hemangiopericytoma

Involving dorsal vermis



Dysmetria of saccades: *Overshoot* to one side, *undershoot* toward the other, called *lateropulsion* (ipsipulsion) of saccades



Wallenberg's Syndrome – Posterior Inferior Cerebellar Artery distribution infarct involving the dorsolateral medulla

Causes a FUNCTIONAL lesion of the cerebellum because of interruption of afferent inputs from the brainstem to the cerebellum

Lateropulsion of saccades: A FUNCTIONAL lesion of the fastigial nucleus (FOR) in Wallenberg's syndrome

Fastigial nucleus (FOR) facilitates contralateral saccades (via contralateral EBN and IBN)

Purkinje cells inhibit deep nuclei

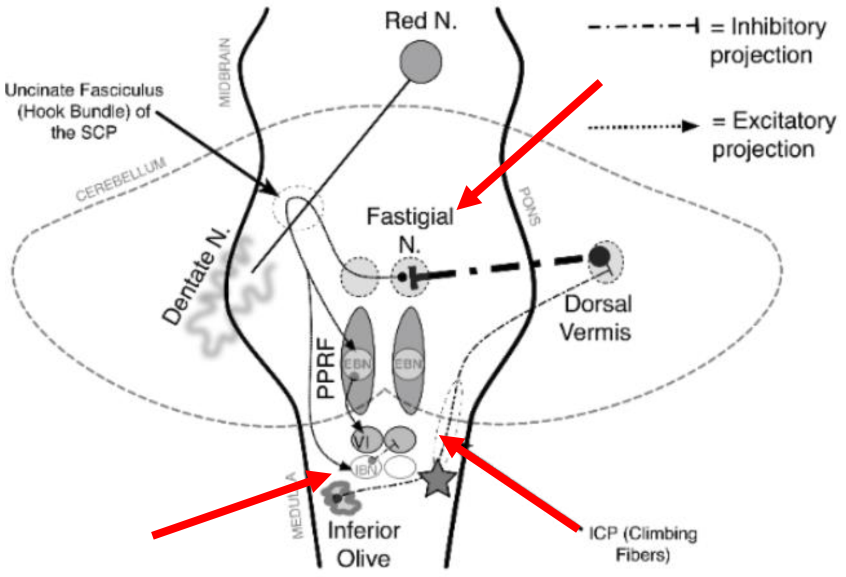
Complex and simple-spike discharge of P-cells are *inversely* related: $R_{ss} \sim 1/R_{cs}$

Lesion interrupts climbing fiber input (complex spikes decrease) to dorsal vermis

Simple-spike (inhibitory) discharge of P-cells increases

FOR activity decreases – *FUNCTIONAL lesion*

FOR axons immediately cross and project to contralateral brainstem where they normally drive *inhibitory saccade burst neurons*



Activity in inhibitory saccade burst neurons (the axons of which cross to the abducens nucleus ipsilateral to the FOR and normally work to facilitate contralateral saccades and inhibit ipsilateral saccades) is decreased leading to

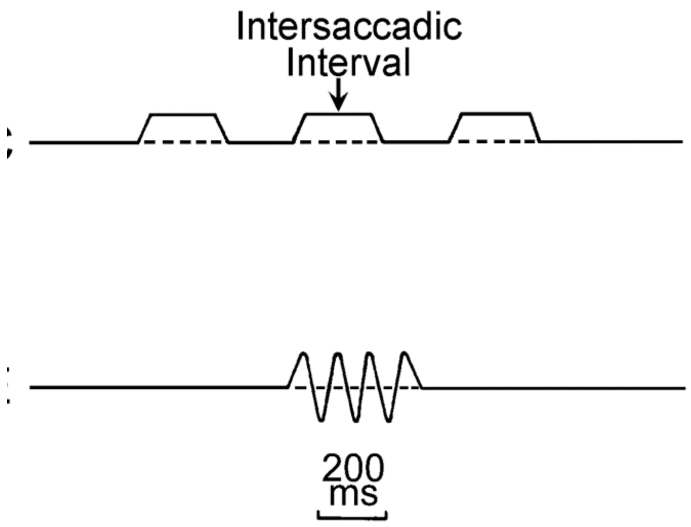
Saccades overshoot toward, undershoot away, from the lesion

Why are saccades hypermetric with a fastigial nucleus lesion and hypometric with a dorsal vermis lesion?

- **The fastigial nucleus normally acts to STOP saccades (via projections to contralateral inhibitory burst neurons (IBN) which inhibit ipsilateral abducens nucleus)**
  - **With a fastigial nucleus lesion saccades will overshoot, producing *hypermetria***
  - **With a dorsal vermis lesion saccades undershoot, producing *hypometria*, since Purkinje Cells (PC), which project to the fastigial nuclei, are inhibitory so the effects of a lesion in the vermis are the opposite of those in the deep nuclei.**

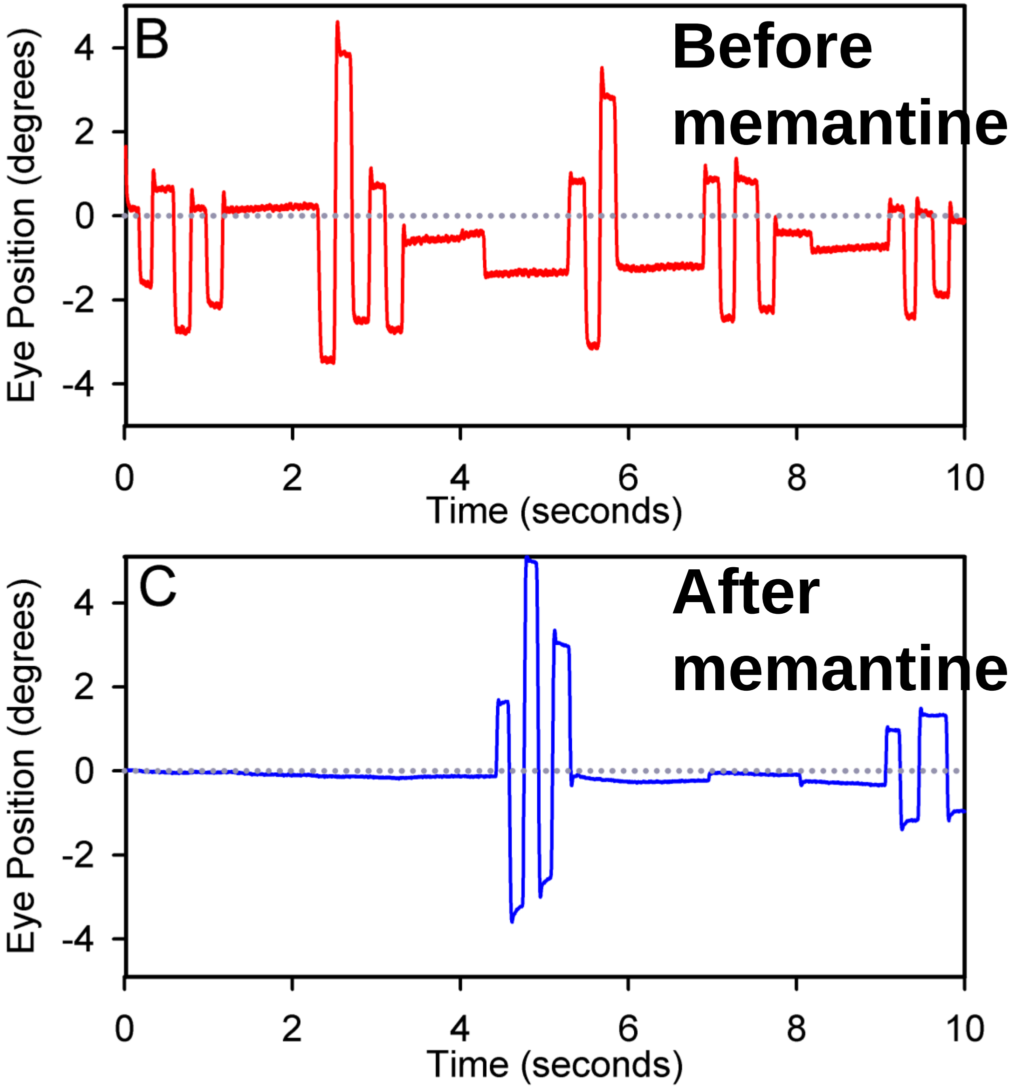
Friederich's Ataxia: Saccadic intrusions (Square wave jerks)

SWJ with a brief intersaccadic interval



Note: Flutter usually refers to <u>horizontal</u> saccade oscillations WITHOUT an intersaccadic interval (i.e., horizontal form of opsoclonus)

Saccadic Intrusions in SCASI: Treatment with memantine



Serra, A. et al. Neurology 2008;70:810-812

Square-wave jerks

- Cerebellar disease
- Elderly
- Progressive Supranuclear Palsy
- Schizophrenia
- Dementing illnesses

Slow vertical saccades

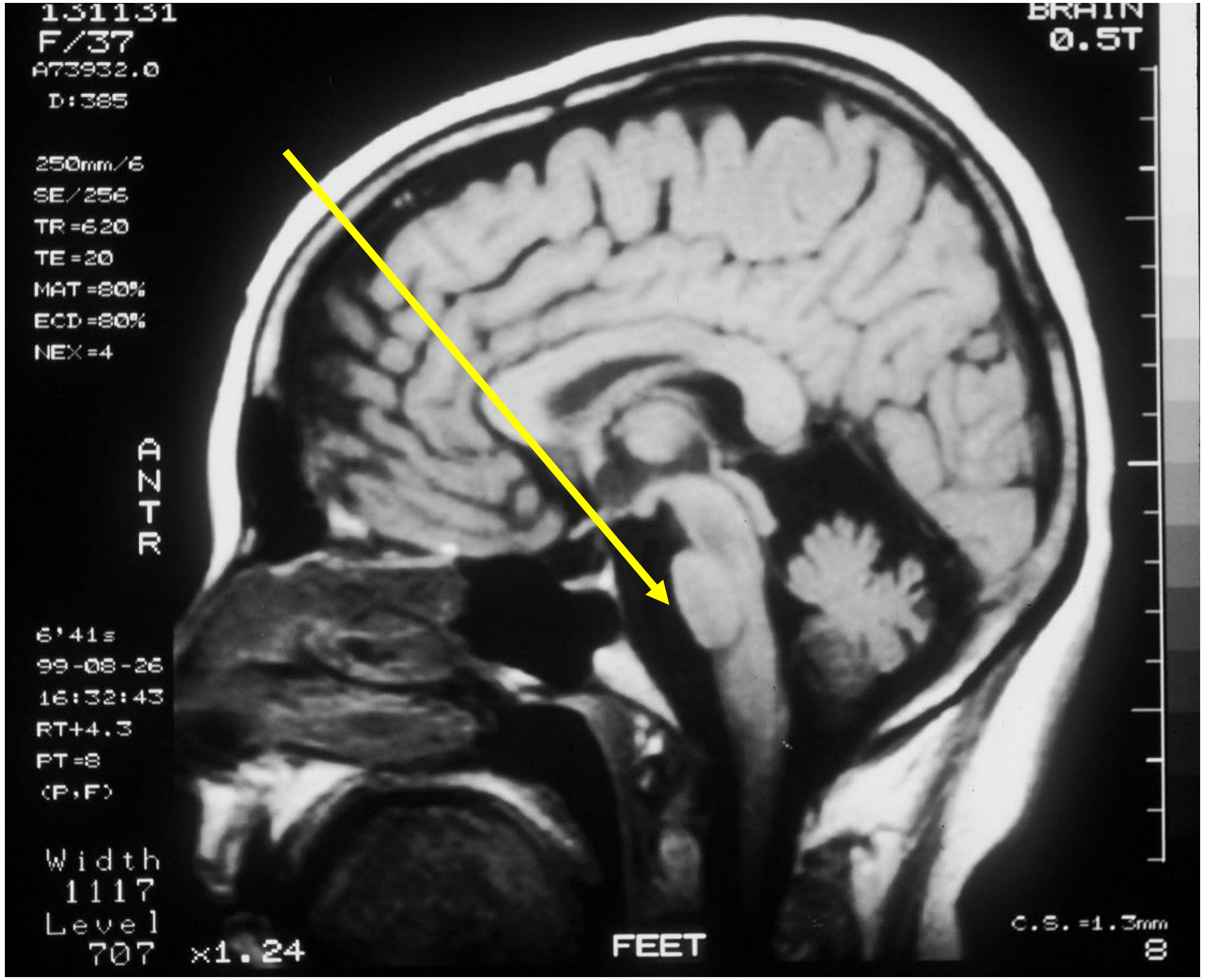
What do YOU see?

MRI in Progressive Supranuclear Palsy (PSP)
Steele-Richardson-Olszewski syndrome

PSP small midbrain, ‘humming bird’ or ‘emperor penguin’ sign

Slow saccades: impaired *pontine* paramedian reticular formation (PPRF) (spinocerebellar ataxia type 2 (SCA2))

MRI in Spinocerebellar ataxia type 2 (SCA2)



SCA2 small pons

Saccadic oscillations: Opsoclonus

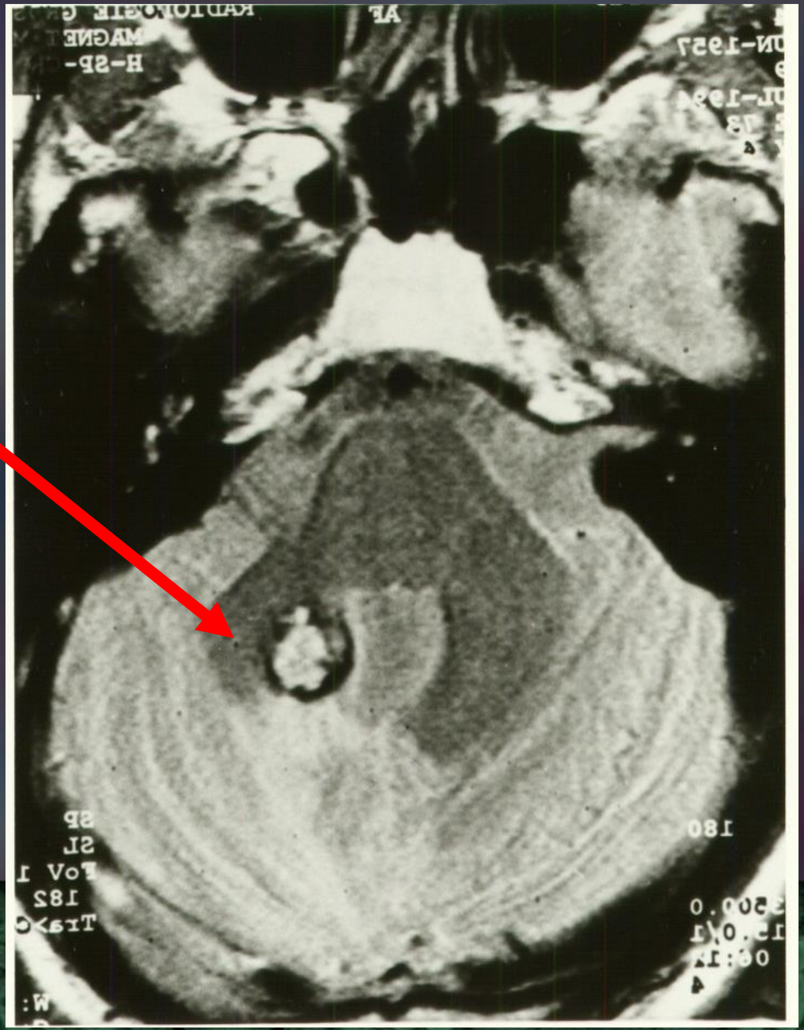
CLINICAL STORY # 2

- Woman in her late thirties with history of ‘migraine’ headaches who suddenly developed a severe headache and imbalance.
- Examination shows a curious torsional nystagmus during vertical smooth pursuit tracking.

TORSION DURING VERTICAL SMOOTH PURSUIT

MRI FINDINGS

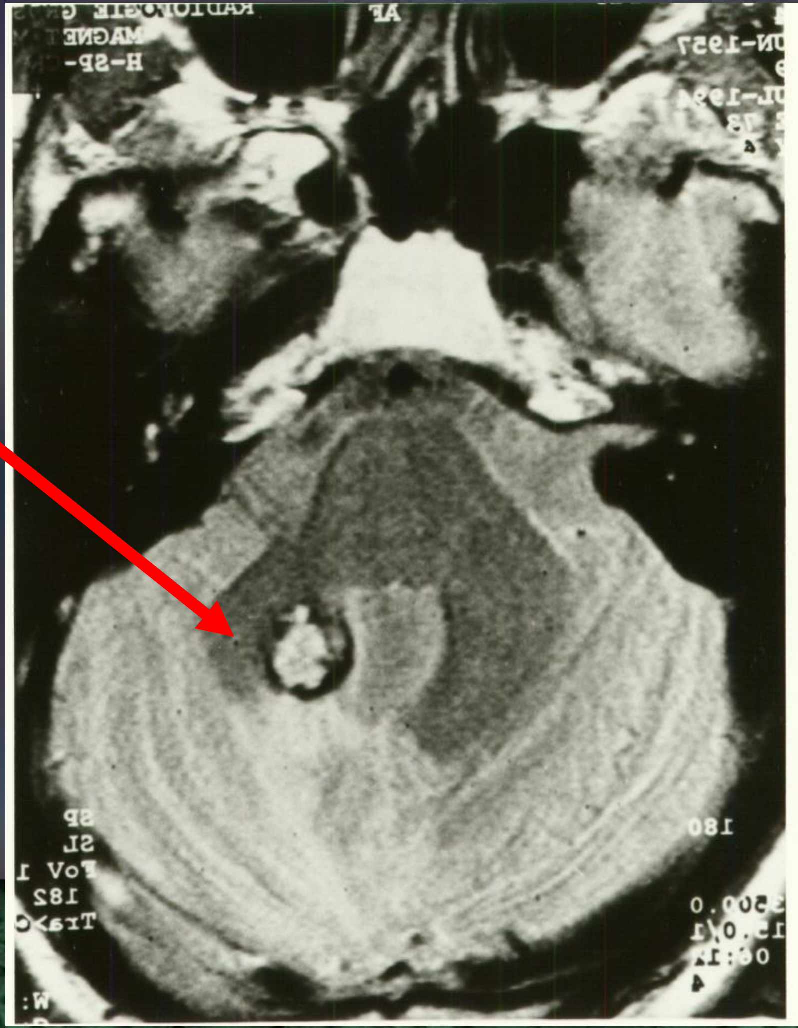
Cavernous angioma in region of right middle cerebellar peduncle



MRI FINDINGS

Cavernous angioma in region of right middle cerebellar peduncle

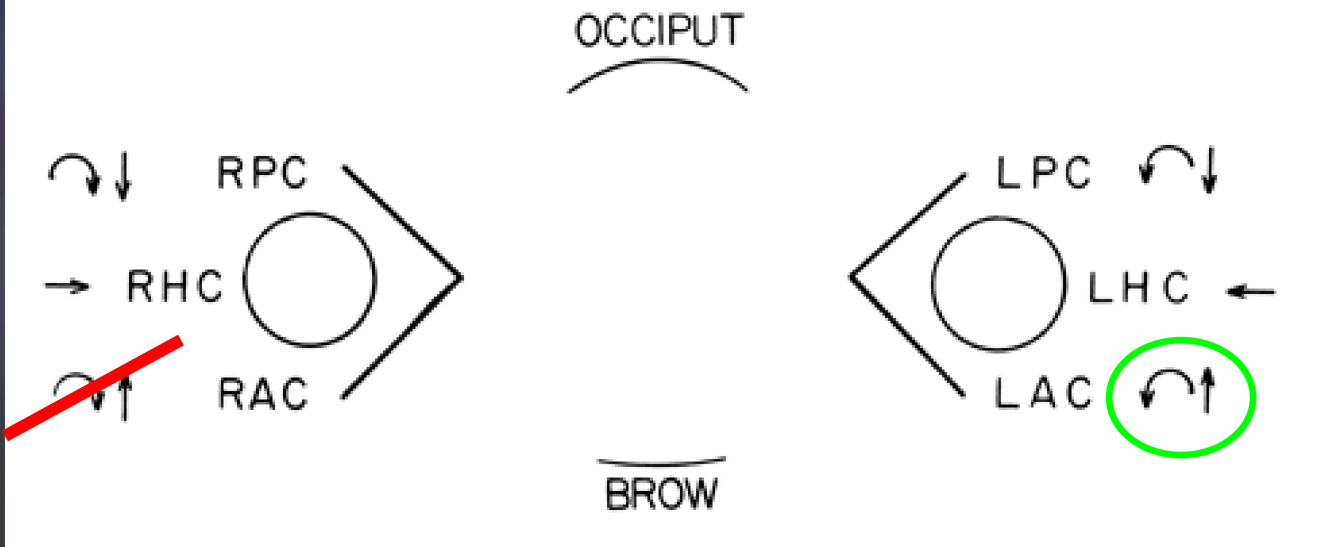
Pattern of abnormal pursuit is virtually pathognomonic for lesions in middle cerebellar peduncle and almost always a cavernous angioma



WHY PURSUIT-INDUCED TORSIONAL NYSTAGMUS?

- **IF vertical pursuit is encoded in a similar way to labyrinthine information, then**
  - **Loss of pursuit information carried in anterior SCC coordinates on the lesion side could lead to the pattern of torsion seen during vertical pursuit in our patients.**

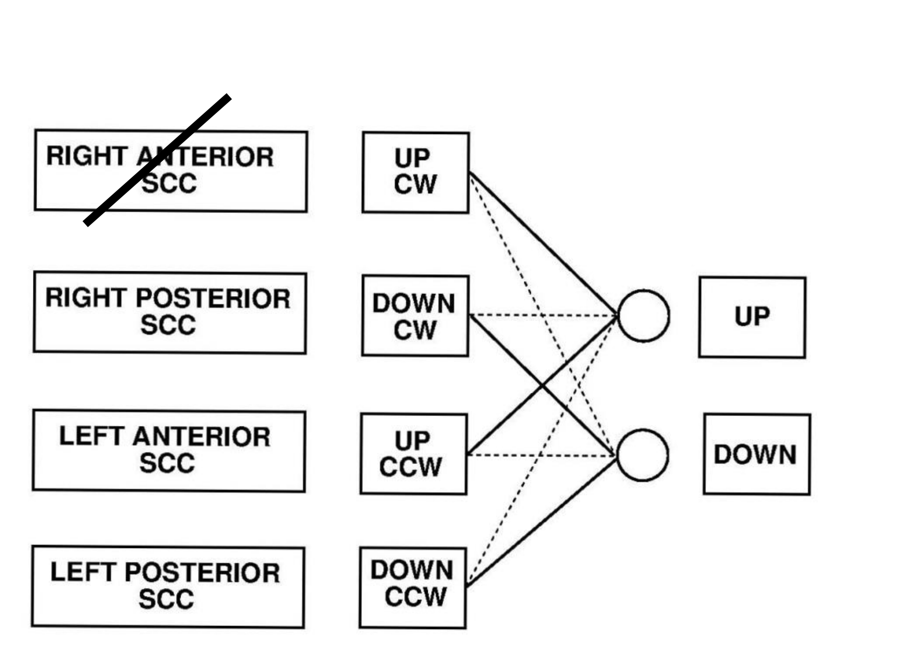
MODEL FOR TORSIONAL NYSTAGMUS DURING VERTICAL PURSUIT



In our patients loss of modulation (excitation and inhibition) of pursuit signals carried in anterior SCC coordinates on the lesion side leads to oppositely directed torsion during up and down vertical pursuit.

During tracking *upwards* the torsion is such that the top poles of the eyes BEAT *away* from the lesion side and the slow phase is *toward* the lesion side.

MODEL FOR TORSIONAL NYSTAGMUS DURING VERTICAL PURSUIT: LOSS OF MODULATION IN ANTERIOR SEMICIRCULAR CANAL PATHWAYS



Significance of pursuit-induced torsional nystagmus?

- **Middle cerebellar peduncle may carry visual information from cerebral-pontine-cerebellar pathways that is encoded in (anterior) SCC coordinates, i.e., *the pursuit system is organized in a labyrinthine coordinate system*.**

- **This emphasizes the importance of the vestibular Anlage for all conjugate eye movements, both slow (vestibular, pursuit, OKN) and rapid (quick phases of nystagmus and saccades). Same principles apply for generation of pure vertical saccades. Unilateral lesions in the midbrain lead to vertical saccades with inappropriate torsion and the direction of torsion tells you the side of the lesion.**

- **Torsion during vertical pursuit may be a useful neuroophthalmological sign of middle cerebellar peduncle lesions, especially cavernous angiomas. Quick phases (top pole) beat away from the side of the lesion during upward pursuit.**

- **After our paper on these patients, neurophysiologists found cells in vestibular nuclei of monkey which supported our hypothesis.**

http://www.gamarjobat.com **Michael R. MacAskill, Kazuo Koga and Tim J. Anderson: Japanese street performer mimes violation of Hering's Law, Neurology 2011;76;1186**
Can Unusual Convergent Gaze in "Kabuki Play" Generalize the Norm of Evaluation System for the Beauty Beyond the Individual Art?-Symmetrical or Asymmetrical- KOGA KAZUO (Nagoya Univ., Res. Inst. of Environ. Med.) IEIC Technical Report (Institute of Electronics, Information and Communication Engineers)VOL.105;NO.479(HIP2005 79-106);PAGE.47-50(2005)