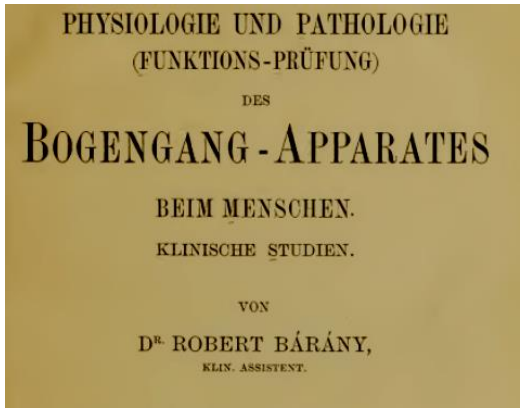
Examinng the dizzy patient: NEW TECHNOLOGY is helpful but a BEDSIDE EXAM based on ANATOMY and PHYSIOLOGY is still the best way to the CORRECT DIAGNOSIS

1965

1992

OVERALL PLAN FOR MY TALKS

- **THIS IS A SPECIALIST'S COURSE: Expect to use your brains and be challenged.**
- **EMPHASIZE FUNDAMENTAL PHYSIOLOGY AND ANATOMY, with applications to the symptoms and signs found in dizzy patients.**
- **The vestibular system is complicated. KEY CONCEPTS will be repeated several times in my talks to aid understanding and memory.**
- **MY ROADMAP will take us through**
  - **What can new technology reliably tell us in the context of the bedside exam.**
  - **Nystagmus and other oscillations.**
  - **How to use eye movements to arrive to the correct diagnosis.**


PHYSIOLOGIE UND PATHOLOGIE
(FUNKTIONS-PRÜFUNG)
DES
BOGENGANG-APPARATES
BEIM MENSCHEN.
KLINISCHE STUDIEN.
VON
DR. ROBERT BÁRÁNY,
KLIN. ASSISTENT.

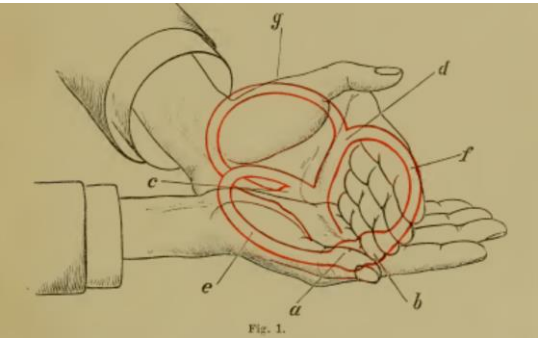


Fig. 1.

TAKE AWAY MESSAGE for the vestibular clinician

- **In the past 25 years there have been sophisticated, quantitative and helpful technological advances in testing the dizzy patient**

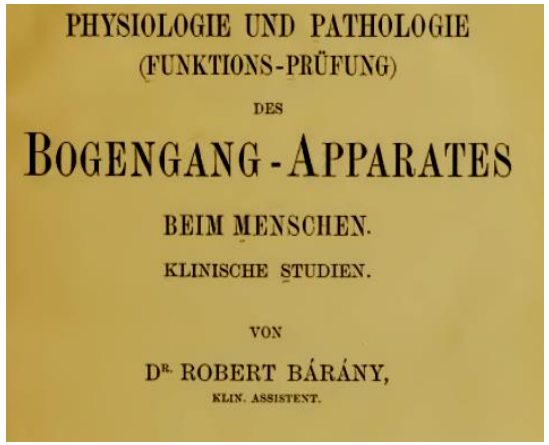
  Video Head Impulse Test (VHIT)

  Quantitative Dynamic Visual Acuity (DVA) testing

  Vestibular-Evoked Myogenic Potentials (cervical and ocular VEMPs)

  High-resolution MRI and CT

  Novel rotational testing paradigms (e.g., off-vertical-axis rotation (OVAR), eccentric rotation, tilt suppression)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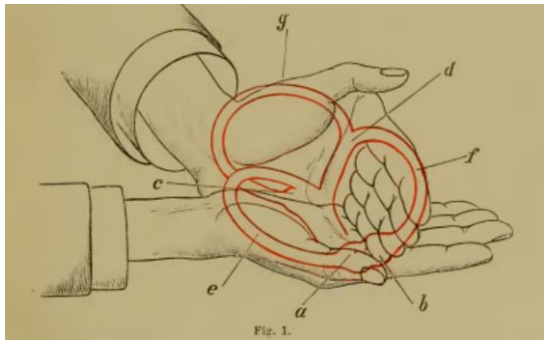


Fig. 1.

TAKE AWAY MESSAGE for the vestibular clinician

But the KEY to successful diagnosis STILL requires

Focused *HISTORY* taking

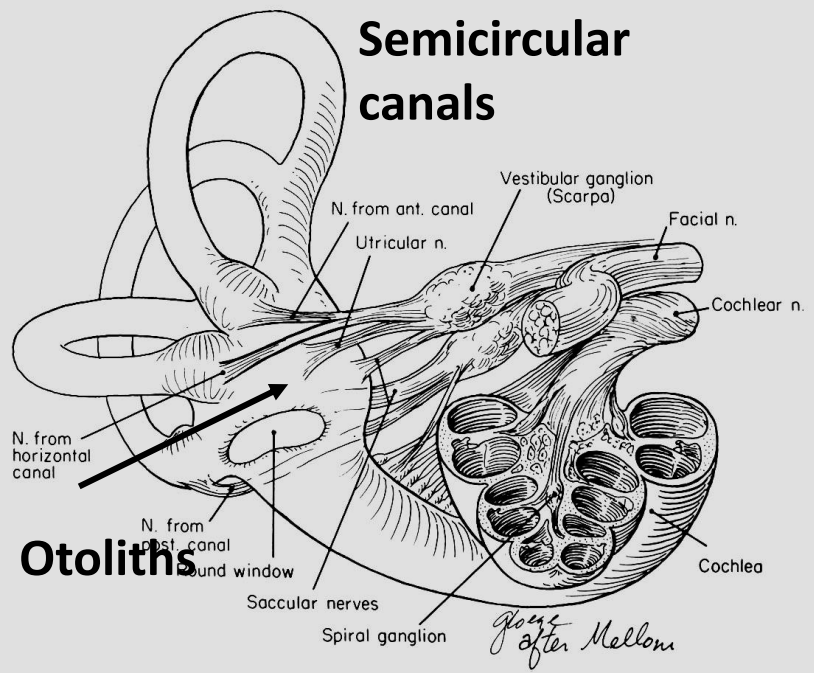
Careful *BEDSIDE CLINICAL EXAMINATION*

Use of *ANATOMICAL AND PHYSIOLOGICAL PRINCIPLES* developed by the European masters of the 19th century

Alexander	Breuer	Hőgyes
Ewald	Bárány	Purkyně
Flourens	Bechterev	Mach

STILL IMPORTANT:

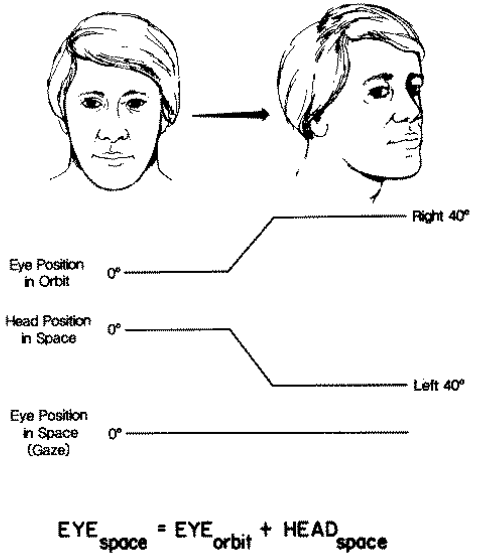
Basic Principles of Vestibular Physiology

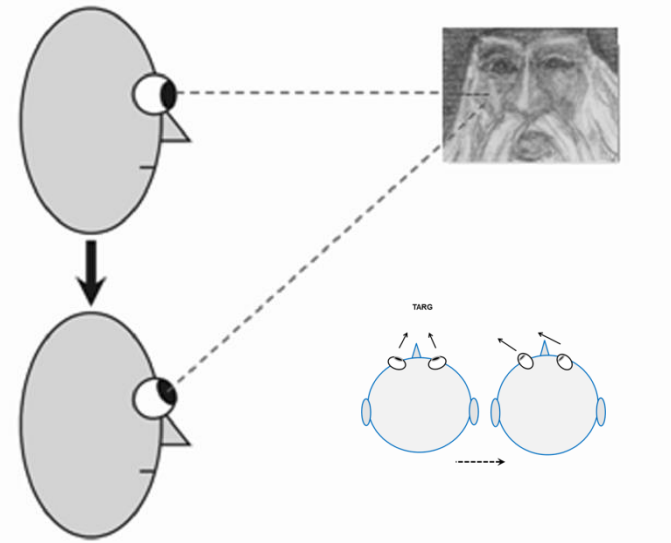


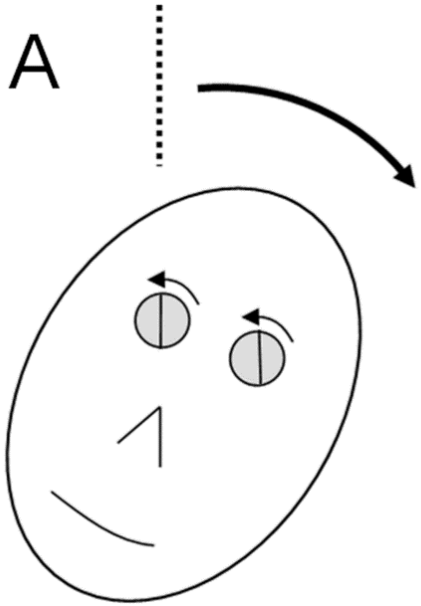
- Two types of acceleration sensors
  - Angular (semicircular canals)
  - Linear (otoliths)
- Two reflexes: vestibulo-ocular reflexes (VOR) and vestibulo-spinal reflexes (VSR)
  - Canal-mediated (*rotational* VOR)
  - Otolith-mediated (*translational* VOR, ocular *counterroll*)
- Functions:
  - Assure *clear vision* during head rotation and translation.
  - Maintain *upright posture* during standing and walking.
  - Help the brain create a conscious *perception* of the position and movement of the body in the environment.

Three Vestibulo-Ocular Reflexes

- **Angular acceleration (canal-mediated, inertial forces)**
  - **(rotational) R-VOR**
- **Linear acceleration (otolith-mediated, inertial or gravitational forces)**
  - **(translational) T-VOR**
  - **(static head tilt) ocular counterroll (OCR)**



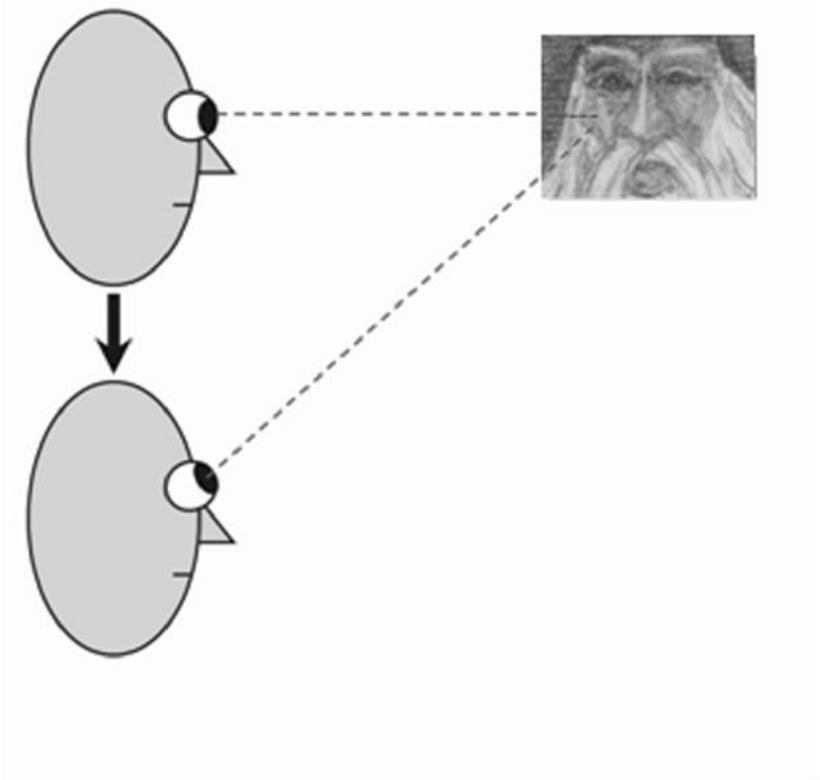


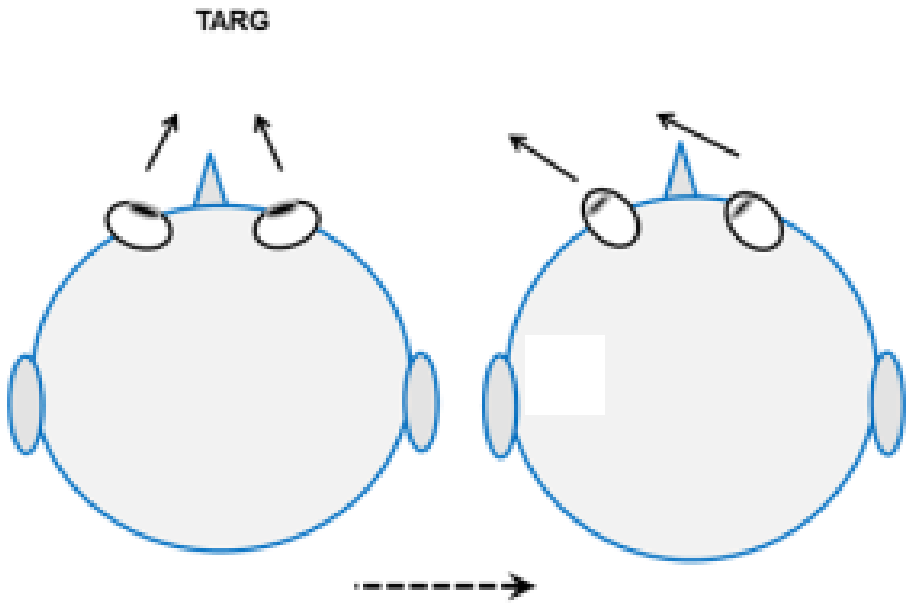


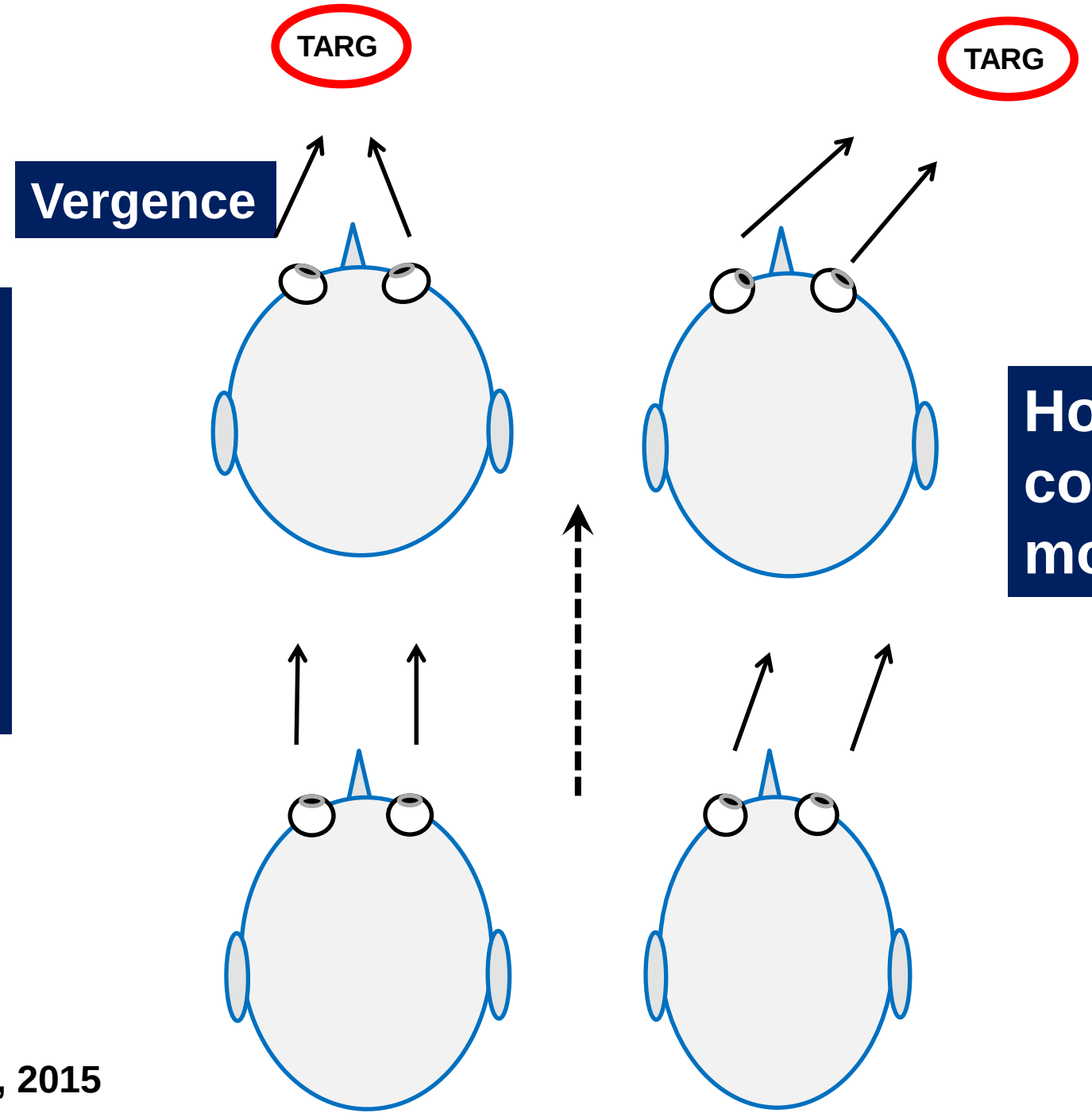
Sometime forgotten: the ROTATIONAL VOR and the TRANSLATIONAL VOR subserve different visual requirements

The pattern of response by the translational VOR is driven by the need for stabilization of images on the FOVEA of both eyes.

Where you are looking and how close the target is determine the pattern of motion of both eyes.







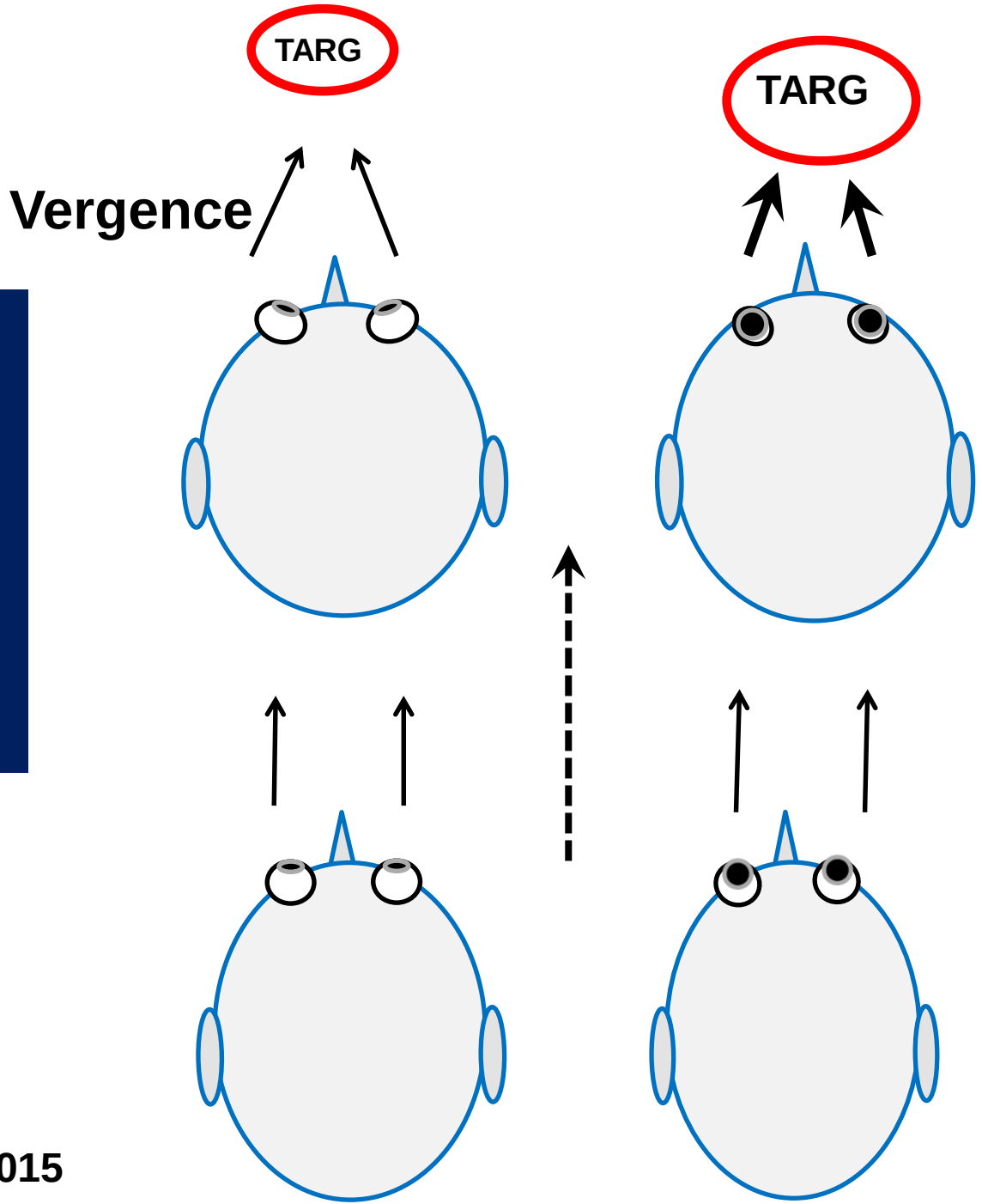
LOOKING AT A NEAR <u>STRAIGHT AHEAD</u> TARGET while translating forward

LOOKING *<u>TO THE SIDE</u>* AT A NEAR TARGET while translating forward

Horizontal compensatory eye movements

Patel, 2015

LOOKING AT A NEAR STRAIGHT AHEAD TARGET while translating forward



LOOKING *UP* AT A NEAR TARGET while translating forward

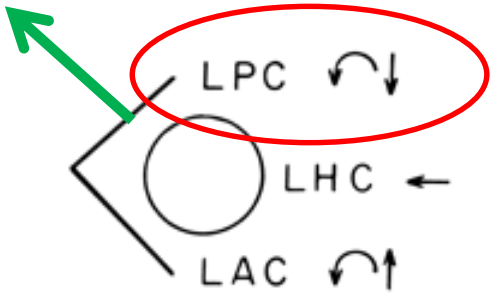
WHY IMPORTANT? In pathology, when a spontaneous nystagmus shows MARKED changes with convergence or eccentric gaze we must think CENTRAL (brainstem-cerebellum) and probably otolith-ocular reflex disorder

Patel, 2015

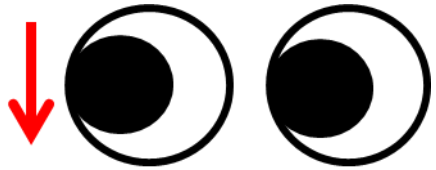
The pattern of response by the rotational VOR is driven by the need for stabilization of images on the entire retina.

The eyes must rotate in a plane parallel to the semicircular canal(s) being stimulated (Ewald's 1st Law), e.g., *BPPV*

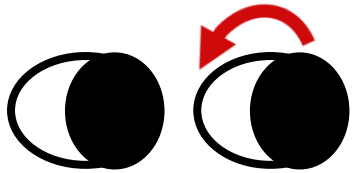
Note how the eye rotates around an axis orthogonal to the plane of the canal being stimulated



Arrows indicate directon of slow phase with stimulation



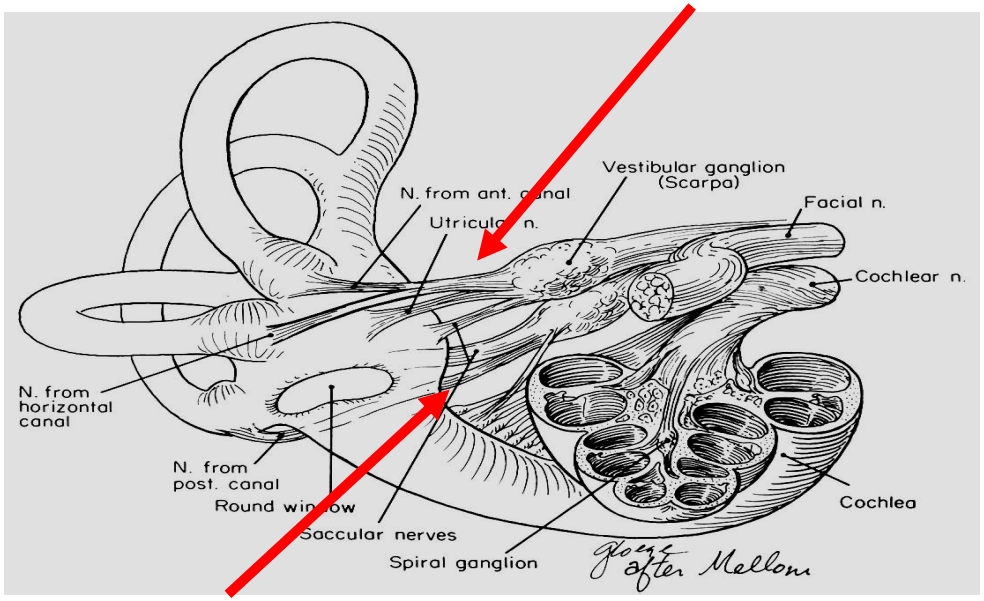
GAZE right



GAZE left

Nerve Supply of the labyrinth

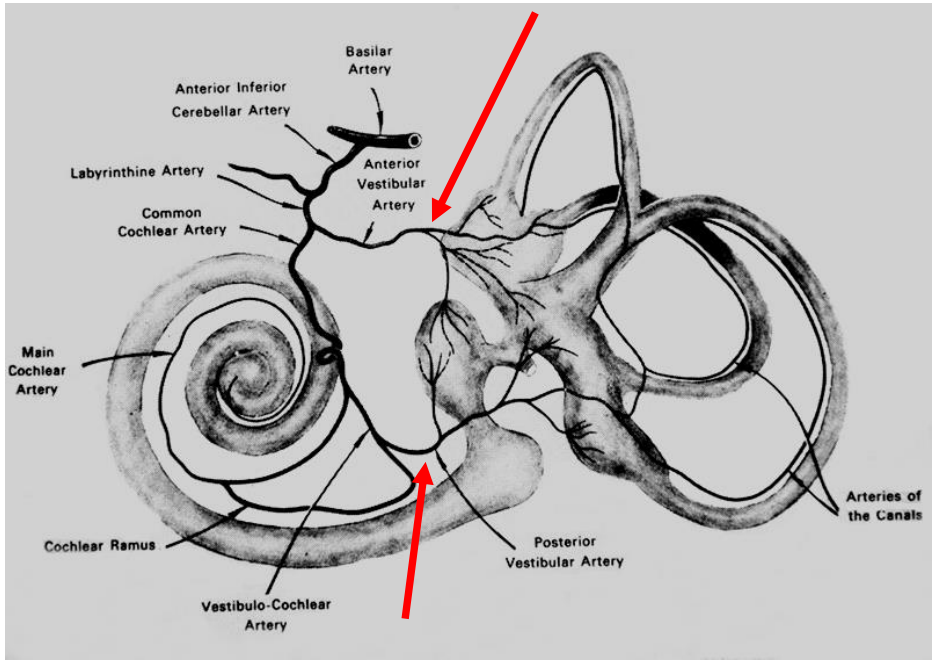
Superior Division: A-SCC, L-SCC, Utricle



Inferior Division: P-SCC, Saccule

Vascular Supply of the labyrinth

Anterior vestibular a. A-SCC, L-SCC, Utricle



Posterior vestibular a. P-SCC, Saccule

Anatomical Principles: Innervation and Blood Supply of the Labyrinth: *Clinical points*

- ***Superior division of the vestibular nerve is most susceptible to damage*** **(long course, focal constriction).**
- **Utricle (and anterior and lateral SCC) likely to be affected and posterior SCC (and saccule) likely to be spared**
- **Making BPPV a not uncommon sequela of vestibular neuritis.**

Anatomical Principles: Innervation and Blood Supply of the Labyrinth: *Clinical points*

- **Anterior vestibular artery is an end artery (hence most susceptible to ischemia).**
- **Thus, *acute isolated vertigo* from posterior circulation hypoperfusion is not uncommon, albeit unusual to recur *over the long term* without other signs or symptoms.**

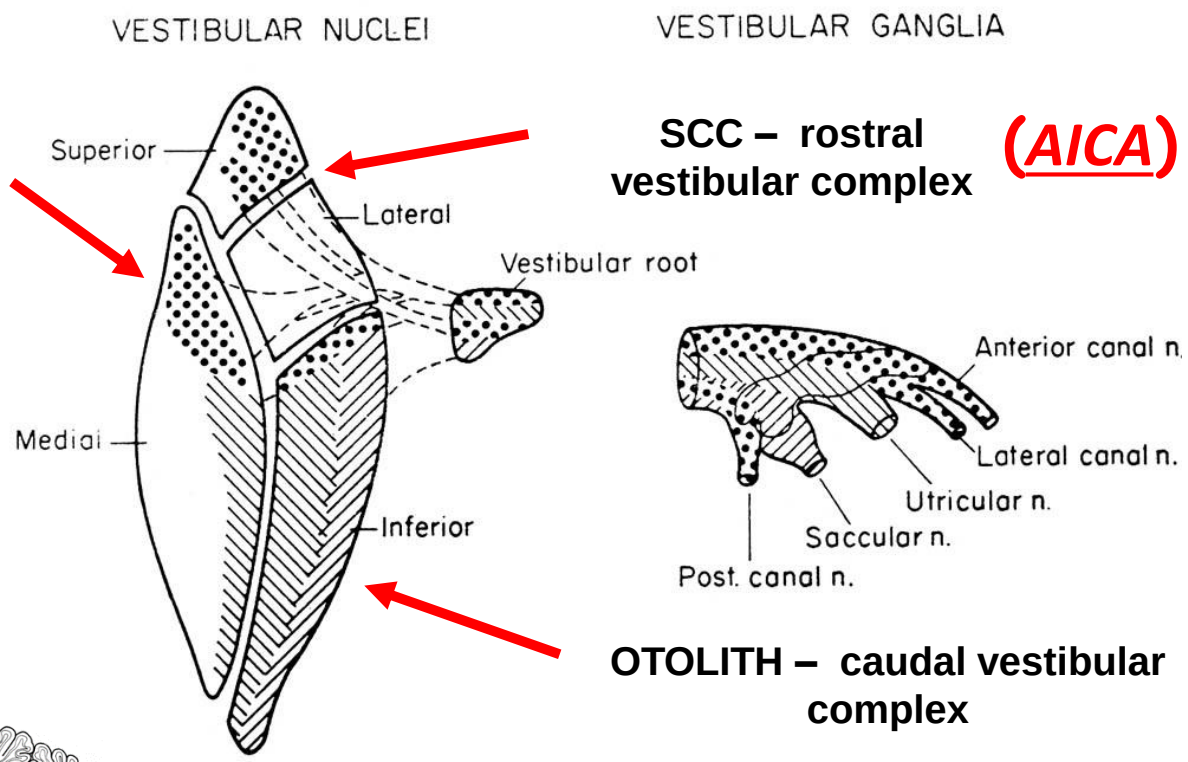
Anatomical Principles: Innervation and Blood Supply of the Labyrinth: *Clinical points*

- **Cochlear branch of the common cochlear -- posterior vestibular artery supplies the *basal turn* of the cochlea (high frequencies).**
- **Hence partial hearing loss from ischemic (and inflammatory) processes on the vestibular nerve is usually *high-frequency***
- **With Ménière's disease (endolymphatic hydrops and its mimics) the hearing loss is usually *low-frequency* (apex of the cochlea).**

MORE KEY ANATOMY:

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

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



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**

Oscillopsia with bilateral vestibular loss

418 THE NEW ENGLAND JOURNAL

MEDICAL INTELLIGENCE

LIVING WITHOUT A BALANCING MECHANISM*

J. C.

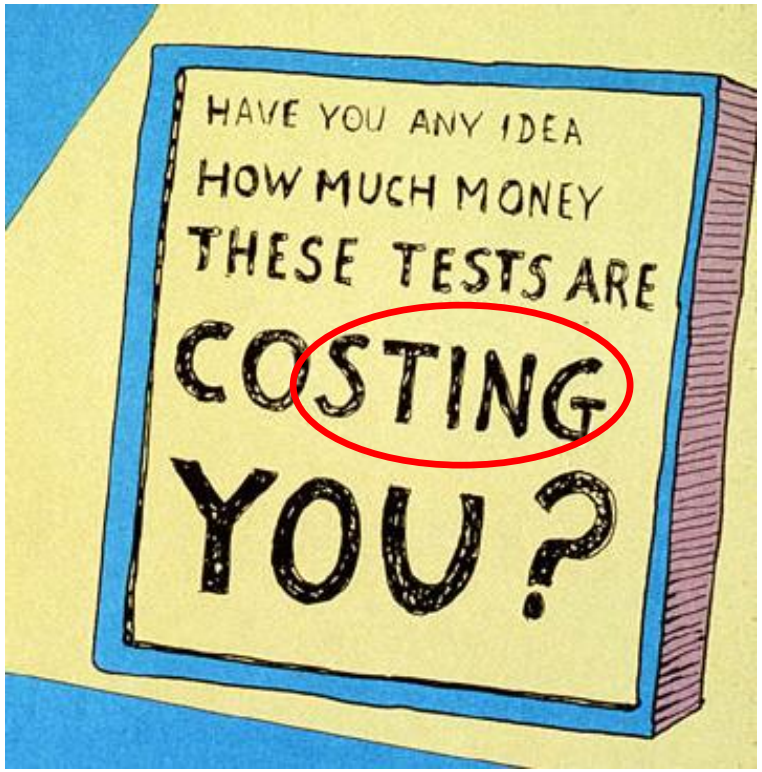
BOSTON

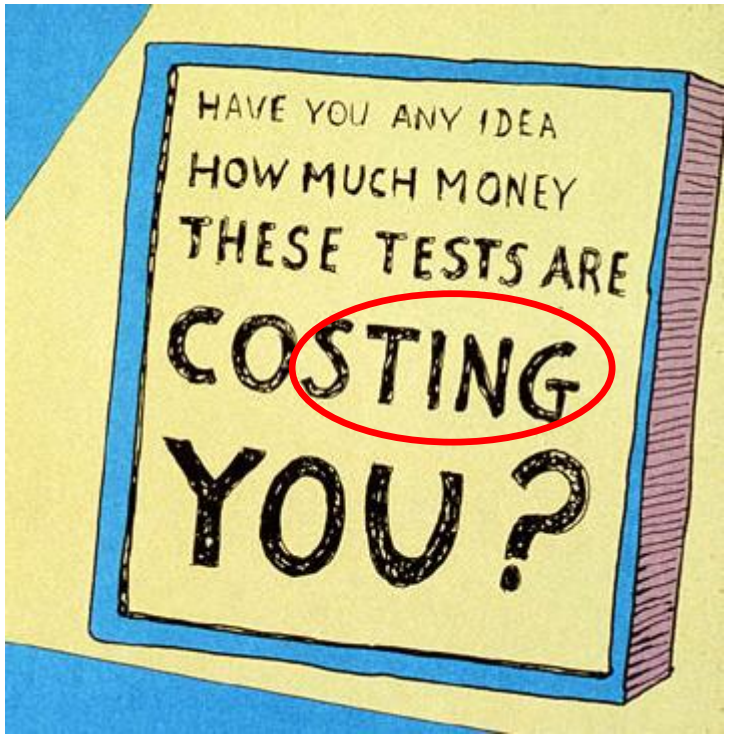
ONE morning, now about four years ago, I arranged in my pajama pocket toothpaste, toothbrush, razor and shaving soap, mounted my

ASK about oscillopsia during head movement

MEASURE Dynamic Visual Acuity (DVA)

Dynamic Visual Acuity (DVA) during head shaking (normal subjects lose one or no lines)


HAVE YOU ANY IDEA
HOW MUCH MONEY
THESE TESTS ARE
COSTING
YOU?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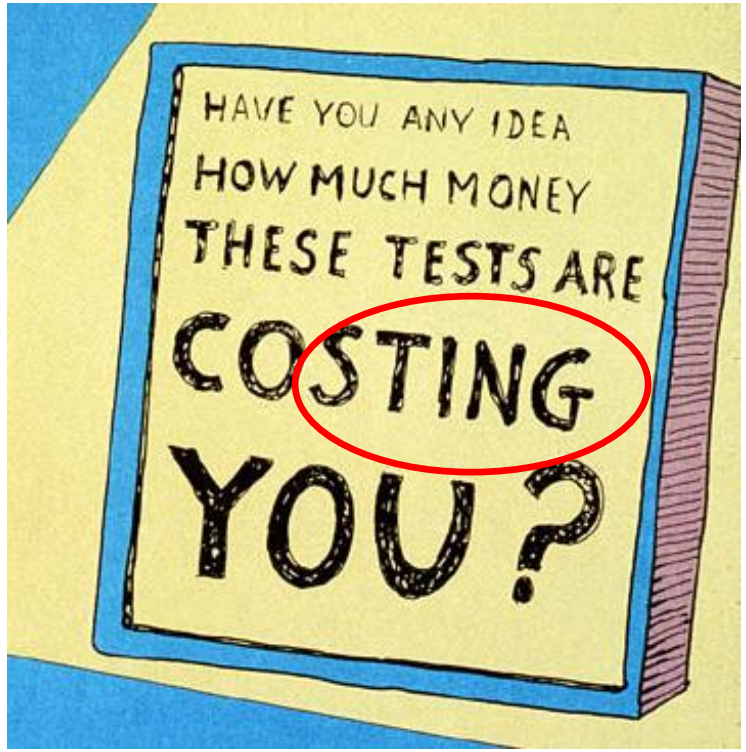
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Dynamic Visual Acuity (DVA) during head shaking

Normal subjects lose one or no lines

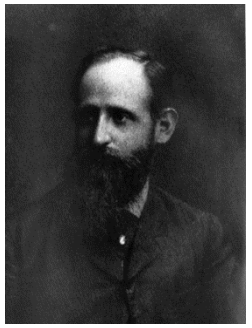
Patients with bilateral loss lose up to 5 lines horizontally and vertically, but less so with roll (torsion)

CLINICAL POINTS

- **Patients who lose same amount of DVA in 'roll' as in horizontal or vertical may be malingering!**
- **At the bedside, there may be a marked loss of DVA when the bedside head impulse test appears NORMAL (e.g., chronic, bilateral vestibular loss in which corrective saccades are hard to see (covert (embedded during head movement, predictive)).**
- **With unilateral loss, DVA will be most affected horizontally and asymmetrically, more so when rotating toward the affected side.**

Ewald

Flourens

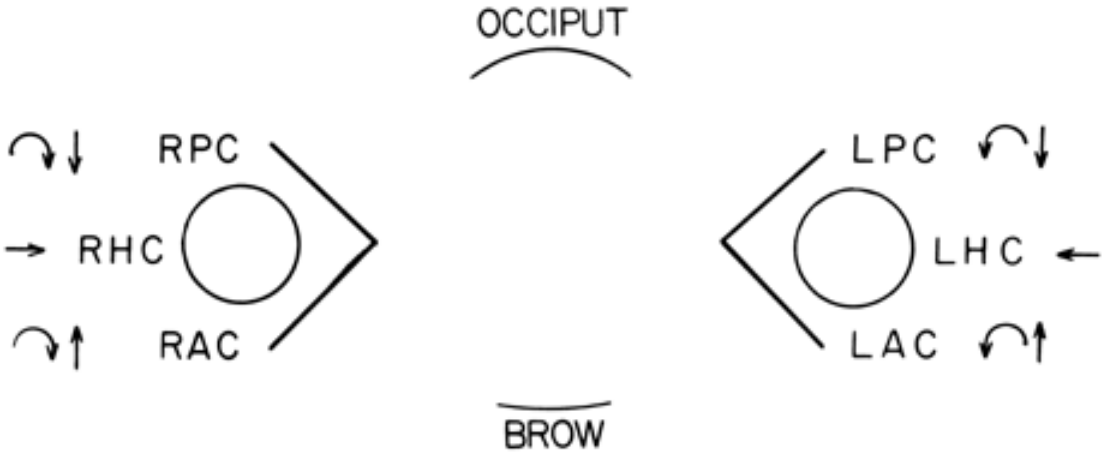


Breuer

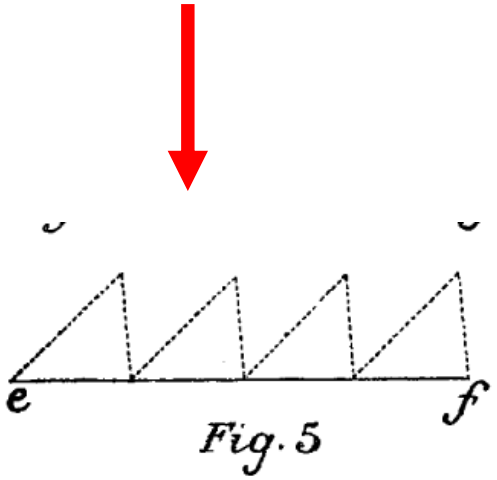
KEY PHYSIOLOGY from three 19th Century Giants

What happens when you stimulate one semicircular canal?

YOU GET NYSTAGMUS: A slow phase, which is the compensatory response to labyrinthine stimulation, and a quick phase, which is the resetting movement.



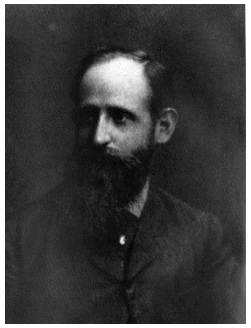
Arrows indicate direction of <u>slow phase</u> with <u>stimulation</u>



Crum Brown 1878

Ewald

Flourens



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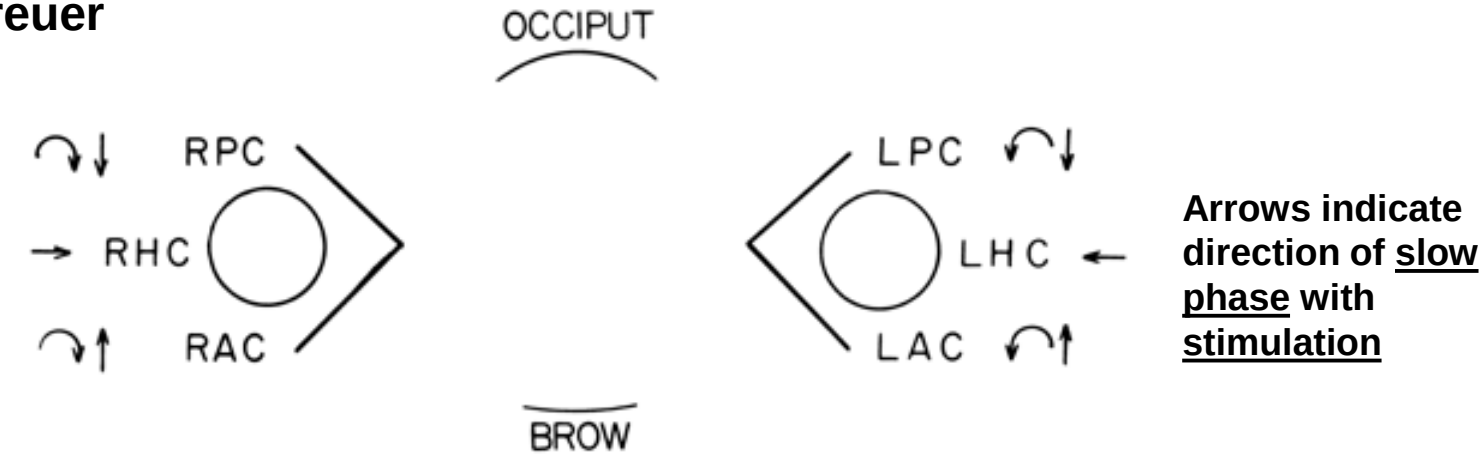


Arrows indicate direction of slow phase with stimulation

Ewald's First Law: **Eyes (head) rotate in a plane parallel to that of head (detected by the SCC in that plane) and so stabilizes gaze (eye-in-space) around all three axes of head rotation.**

Ewald's Second Law: ***The lateral SCC*** **are stimulated better by** ***ampullopetal (excitation) than by*** **ampullofugal (inhibition) fluid flow.**

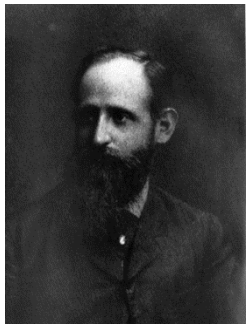
Ewald's Third Law: **The** ***vertical SCC*** **are stimulated better by** ***ampullofugal*** **(*excitation*) than by ampullopetal (inhibition) fluid flow.**

****The excitatory direction of a canal is always when you turn or tilt your head toward that side.**

Vestibular reflexes must stabilize the eyes (and the head) in the environment despite motion of the body so we can SEE when moving

Ewald

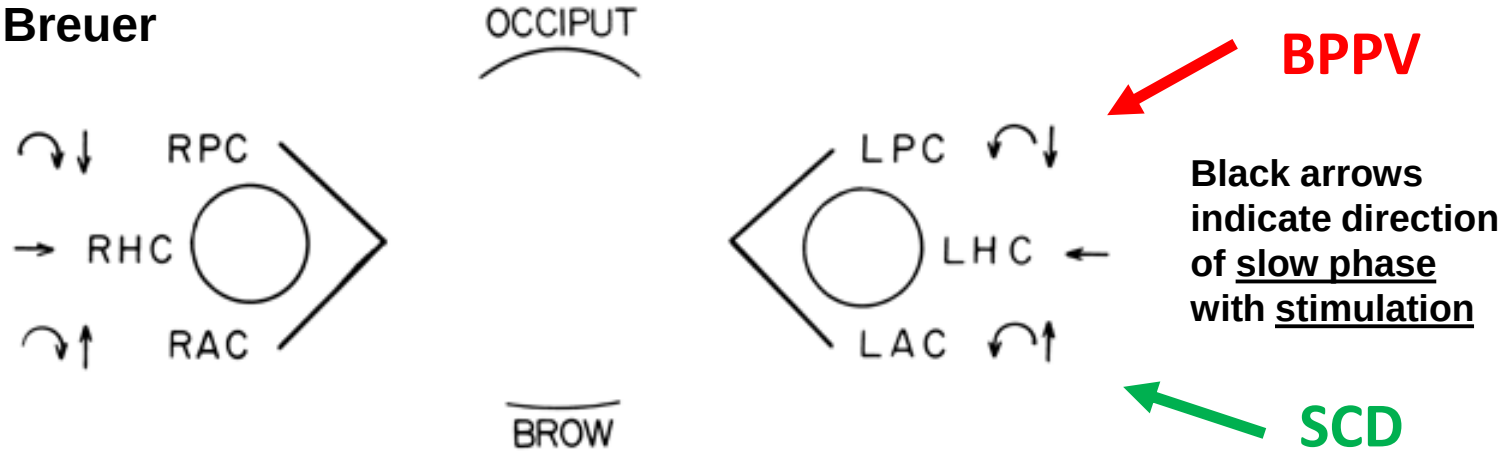
Flourens



Breuer

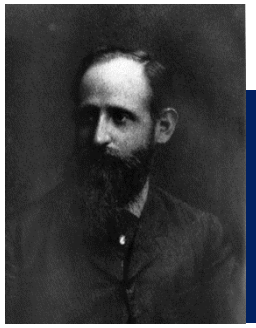
KEY PHYSIOLOGY from three 19th Century Giants

What does this scheme tell the clinician?



- Mixed *vertical-torsional* nystagmus usually is peripheral as it can come from stimulation of a single vertical SCC: BPPV (benign paroxysmal positional vertigo, Upbeat) and SCD (superior canal dehiscence, Downbeat).

The nystagmus typically varies with horizontal eye position from being more torsional to being more vertical, so the globe continues to rotate in the plane of the specific vertical canal. (Ewald's 1st)



Ewald

Flourens

Breuer

KEY PHYSIOLOGY from three 19th Century Giants

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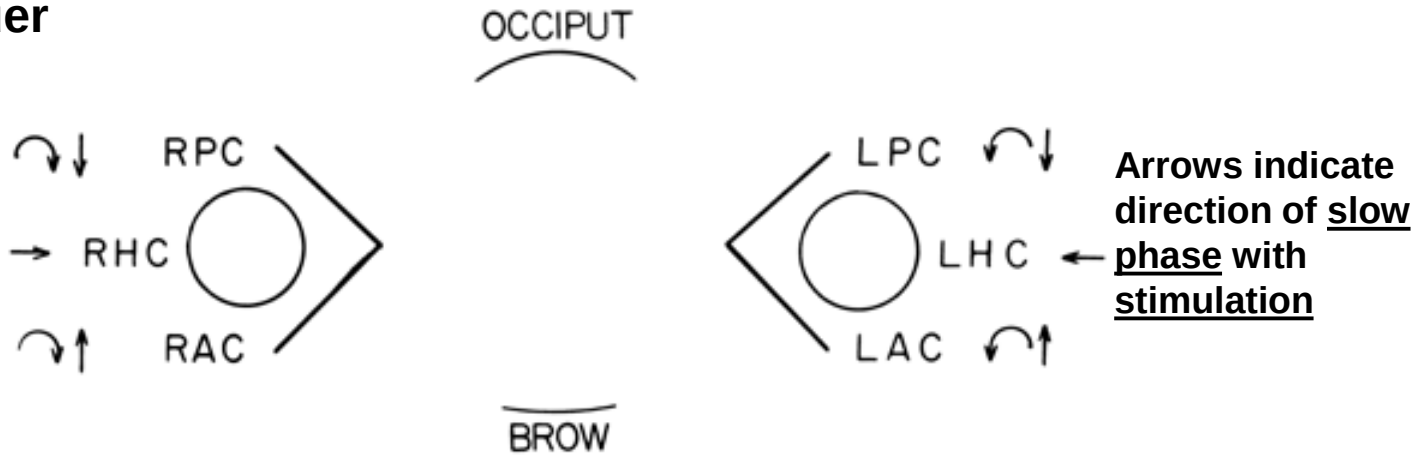
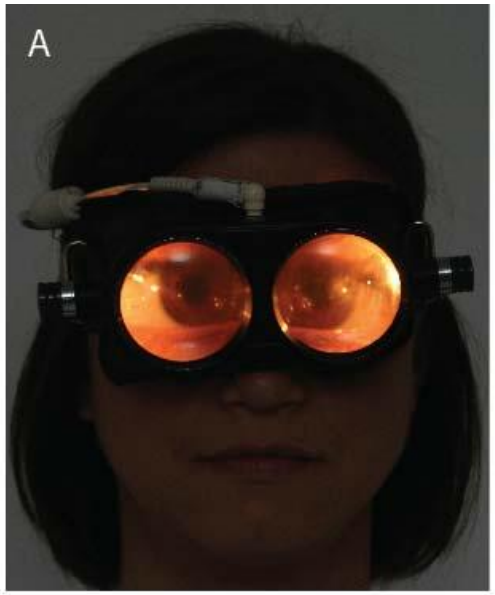
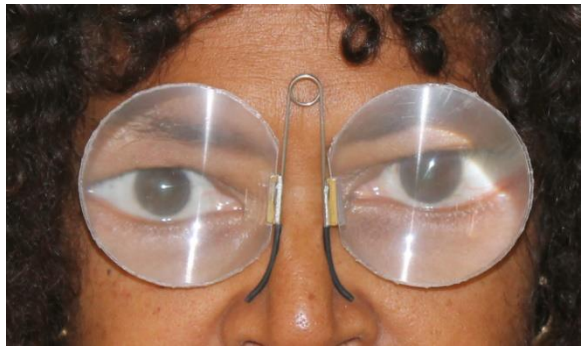


Arrows indicate direction of slow phase with stimulation

- Pure *vertical* and pure *torsional* nystagmus are *central* signs
- Mixed *horizontal-torsional* nystagmus is a *peripheral* sign
- Mixed *vertical-torsional* nystagmus comes from stimulation of a single vertical SCC and is usually a *peripheral* sign

Look for nystagmus: with and without fixation (clinical dictum: fixation suppression of vestibular nystagmus is impaired in central lesions)

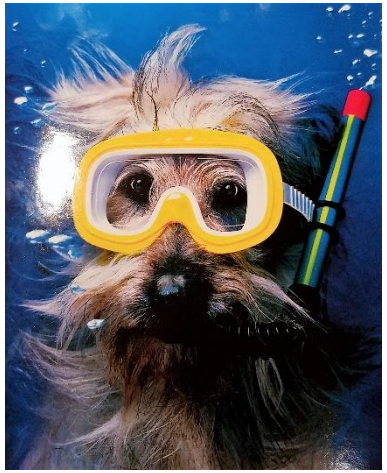




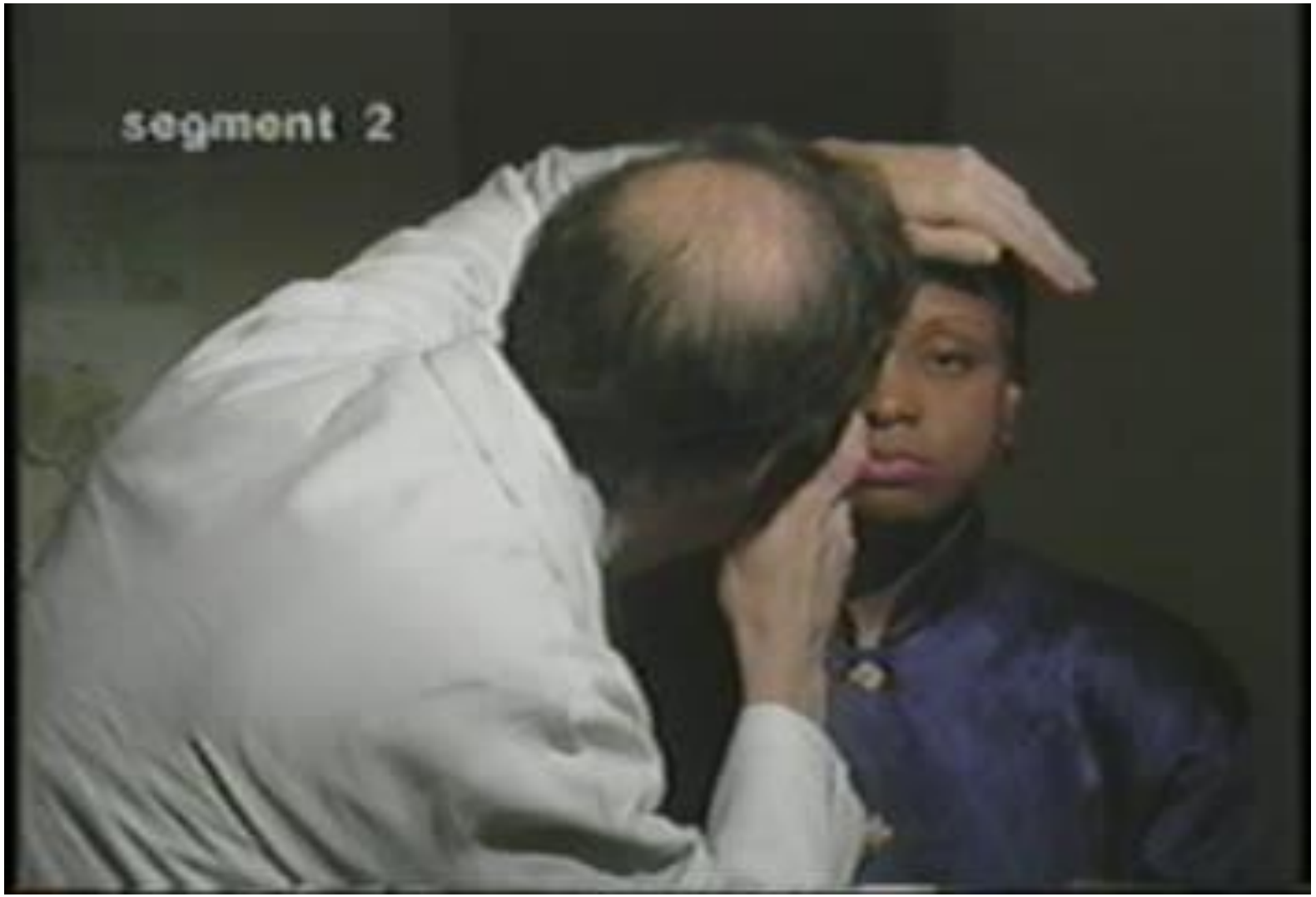
Strupp, Neurology, 2014

Frenzel goggles, ebay

Frenzel Lenses (*best used* with room lights off) to remove fixation

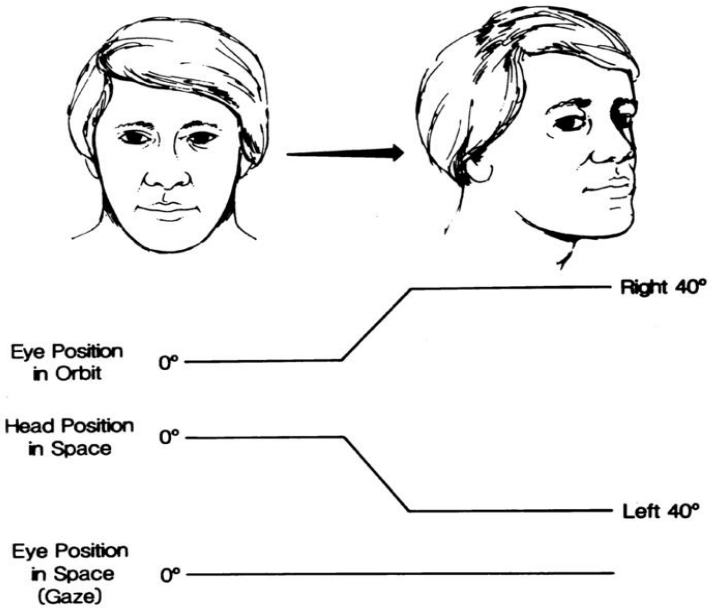


Ophthalmoscope and spontaneous nystagmus



Bedside testing

(rotational) R-VOR



The Head Impulse Test (HIT)

Head impulse testing (HIT) of the vestibulo-ocular reflex (VOR)
LOOK FOR THE CORRECTIVE SACCADE

TECHNICAL POINTS

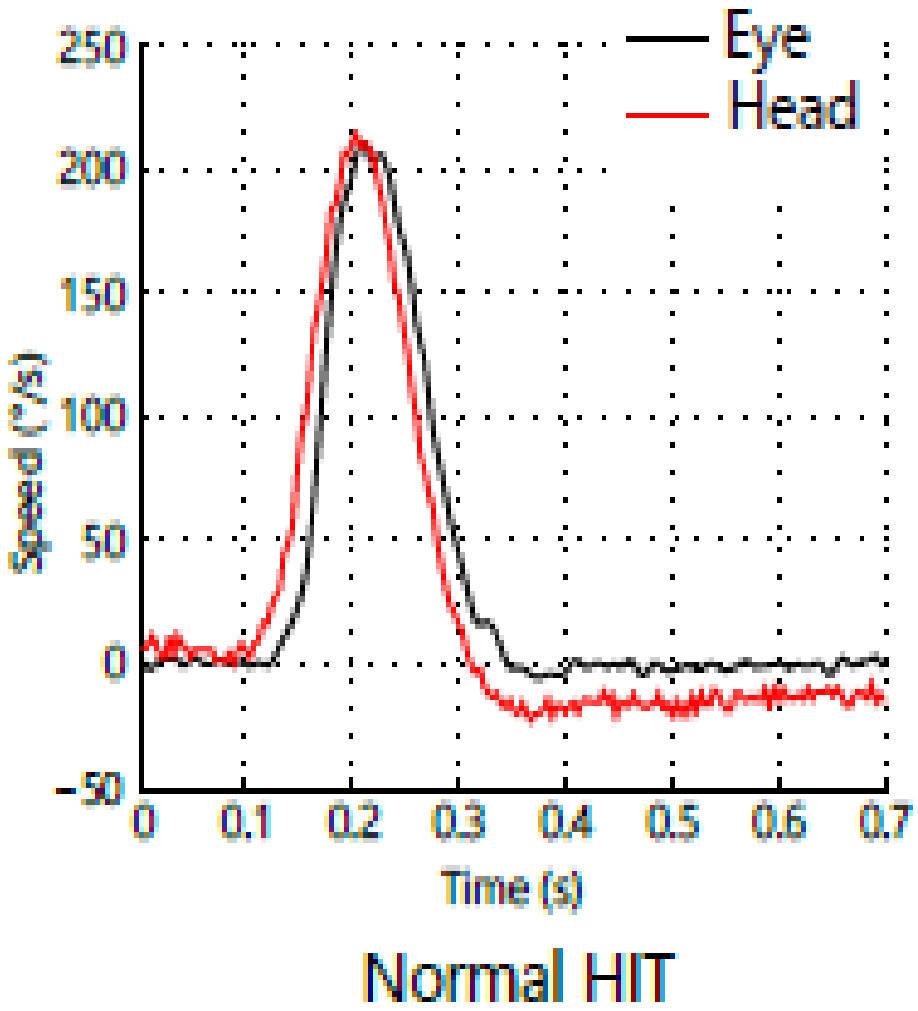
- Abrupt, brief duration, high acceleration
- Small excursion
- BEGIN from center and stop at eccentric to prevent anticipation from obscuring the deficit
- If gaze-evoked nystagmus BEGIN from eccentric and stop at center (to prevent nystagmus from obscuring the deficit)

Testing of the VOR: Head Impulse Test (HIT) in a unilateral peripheral labyrinthine lesion

Catch-up saccade during brief, high-acceleration, head rotation (left-sided loss)

Head-impulse sign in unilateral labyrinthine loss (rotating toward the side of the lesion)

HEAD IMPULSE TEST (HIT)



Normal HIT

Measure HEAD VELOCITY And EYE VELOCITY

Note: EYE speed is inverted so it can be compared with the speed of the HEAD.

The Ratio of Eye to Head (GAIN) should be ONE

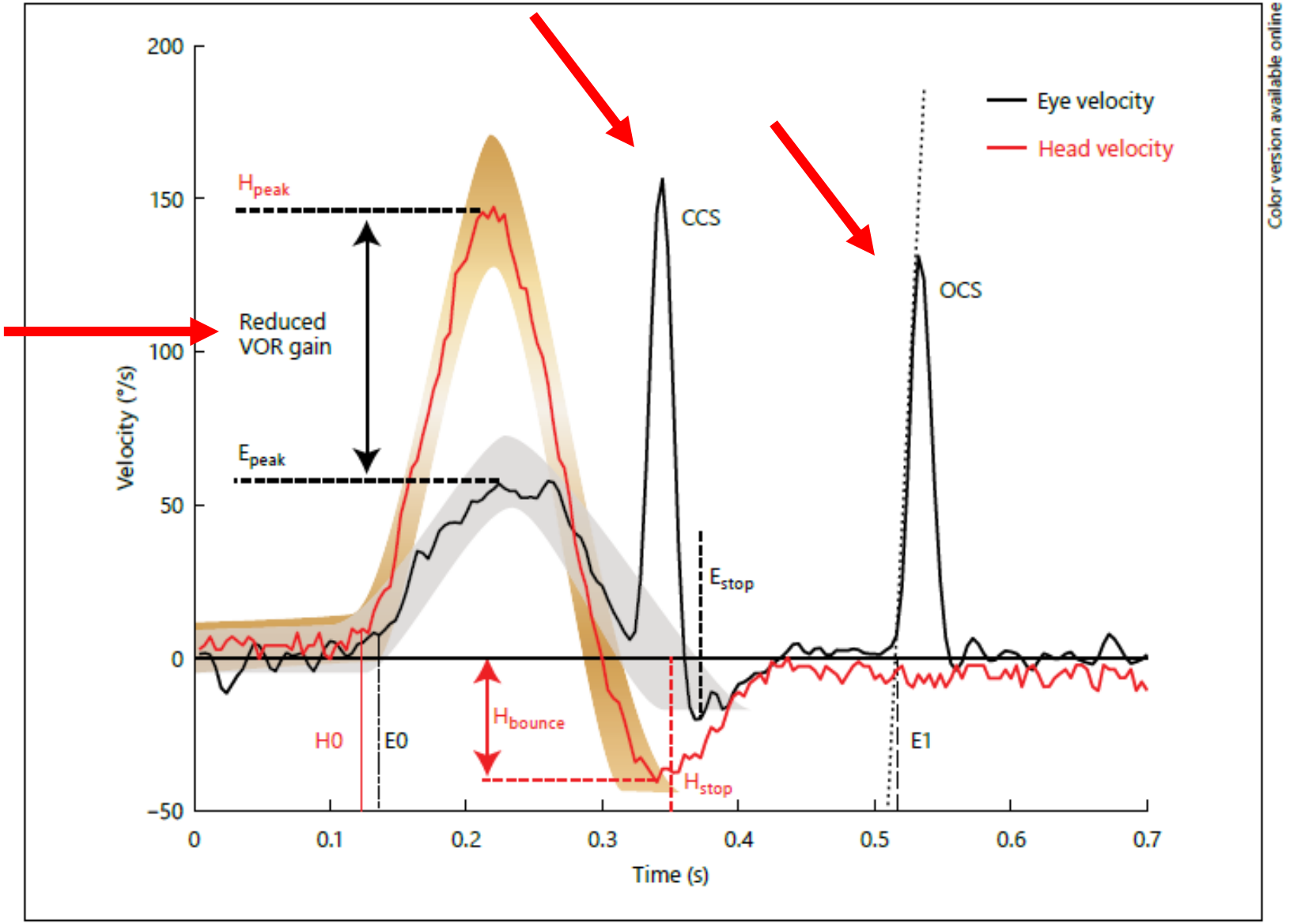
Mantokoudis, 2016

The ideal recorded response when one labyrinth is not working

REDUCED slow-phase VOR gain (EYE/HEAD)

CCS, COVERT corrective saccade

OCS, OVERT corrective saccade



Mantokoudis, 2016

Bilateral head-impulse sign in bilateral labyrinthine loss

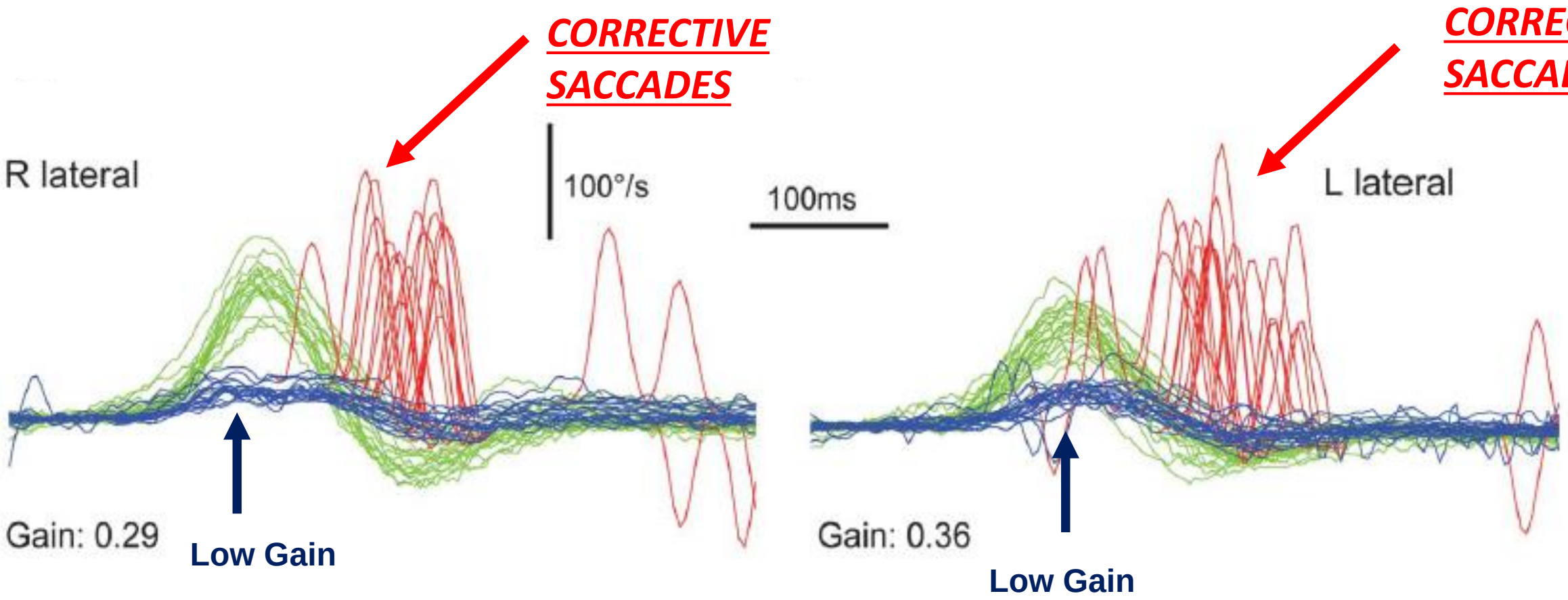
NEVER SWIM AT NIGHT!

We have a powerful dorsal light reflex

The brain always thinks that the sun is above us

Remember: Patients who lose vestibular function bilaterally do NOT have vertigo since there is no tone imbalance or nystagmus. E.g., ototoxicity of antibiotics, autoimmune disorders, syphilis, sarcoidosis, etc

BILATERAL LOSS OF PERIPHERAL LABYRINTHINE FUNCTION



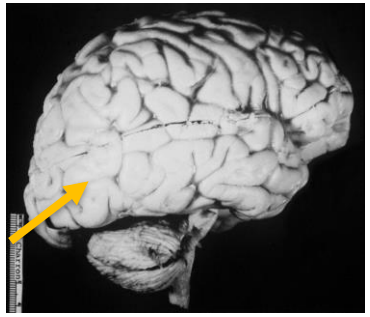
MacDougall, 2016

Abnormal VOR in cerebellar disease: <u>Abnormal direction</u>

What is wrong with this VOR response?

What is wrong with this VOR response?

(Note: Patient is fixating straight ahead)



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

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

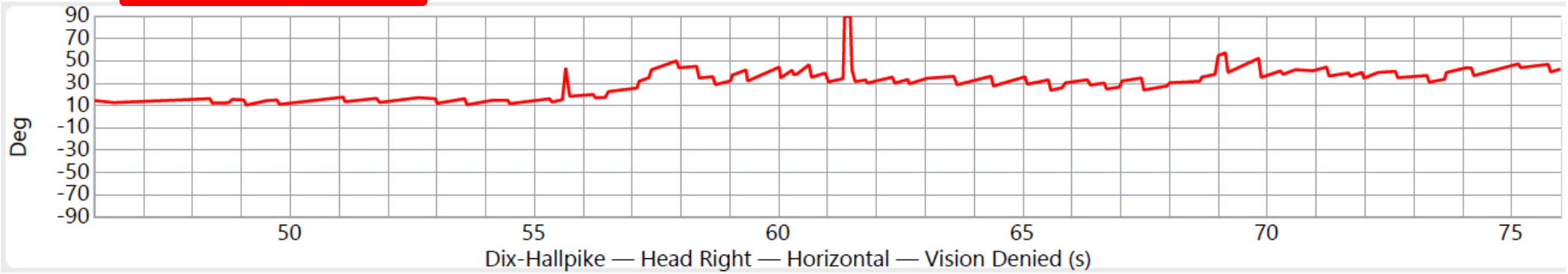
WHAT ABOUT AUTOMATED TECHNOLOGY: VIDEO oculography (VOG)

VIDEO GOGGLES are easy to use and comfortable and give you an immediate, automatically-calculated measures of spontaneous nystagmus and VOR gain (eye movement/head movement), without operator intervention

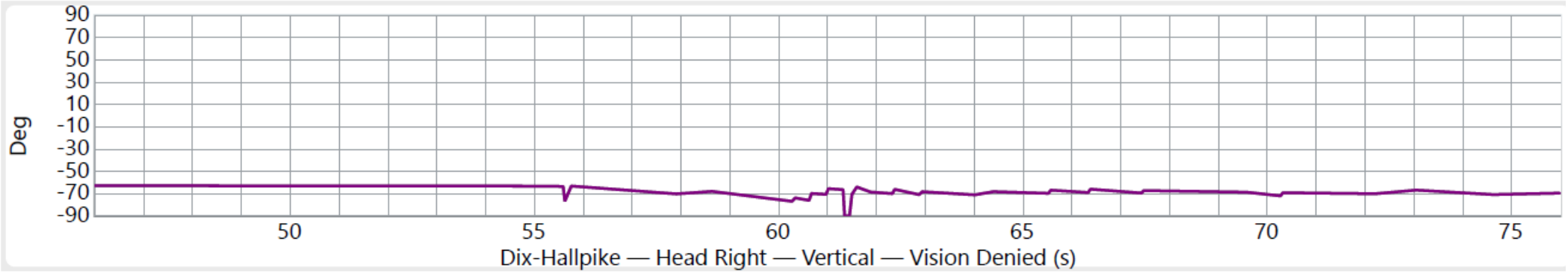
BUT CAN I BELIEVE THE RESULTS??? TWO CASES OF BPPV: ONE FALSE NEGATIVE AND THE OTHER FALSE POSITIVE

An elderly women comes to the Emergency Department with dizziness and imbalance when standing up for a day's duration. BPPV??

Dix-Hallpike — Head Right Test: 9/5/2018 2:57:31 PM
Test Operator: Default Administrator
SPV Peak Horizontal: 9 °/s at 61.5 s

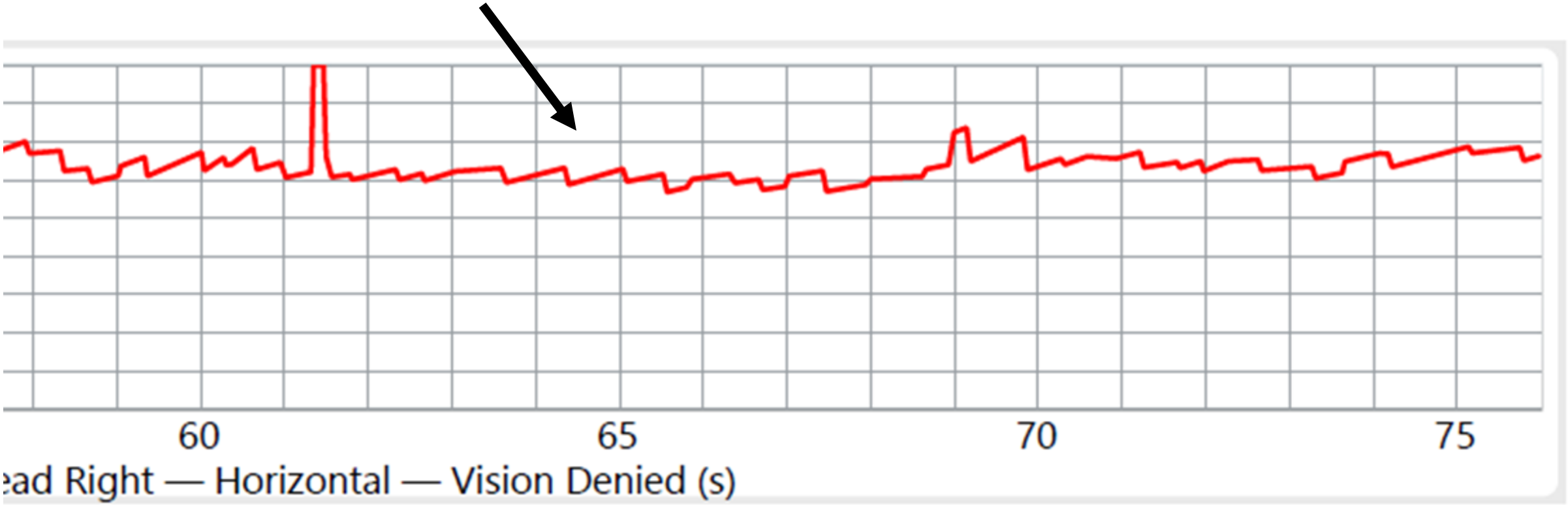


Dix-Hallpike — Head Right Test: 9/5/2018 2:57:31 PM
Test Operator: Default Administrator
SPV Peak Vertical: 0 °/s at 0.0 s

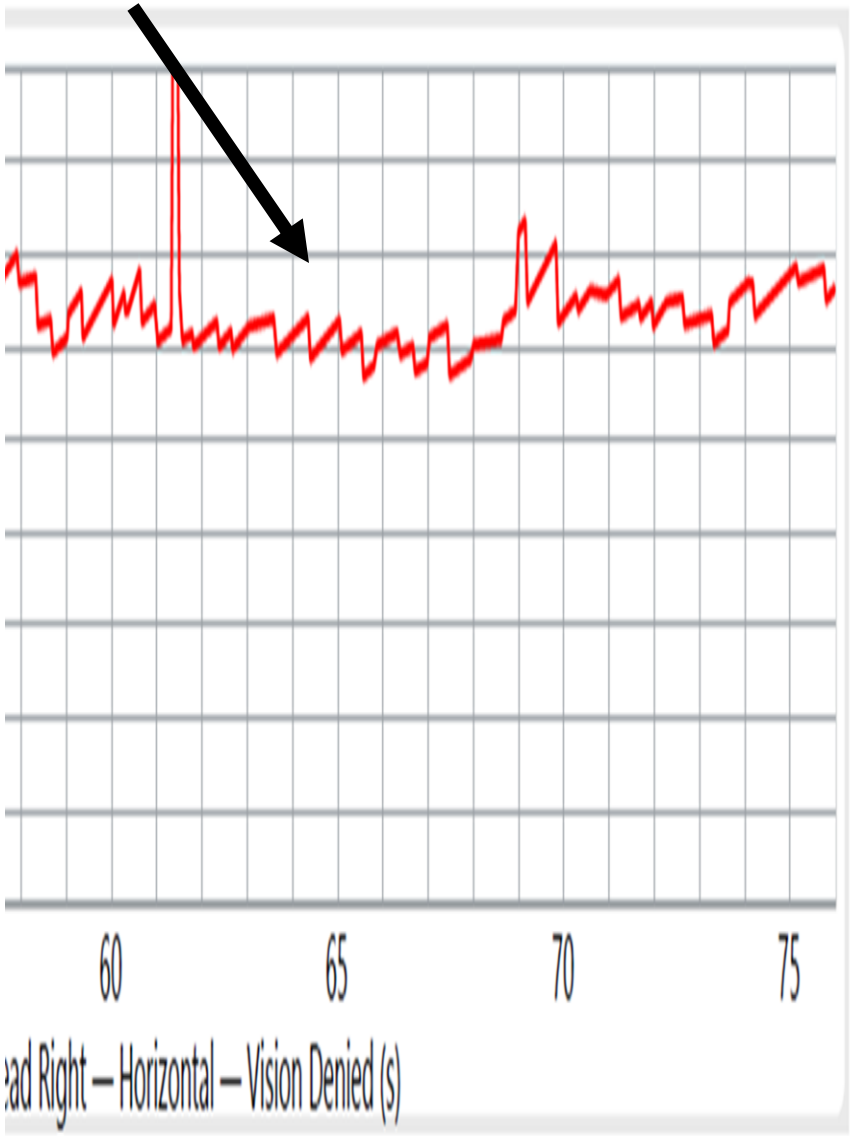


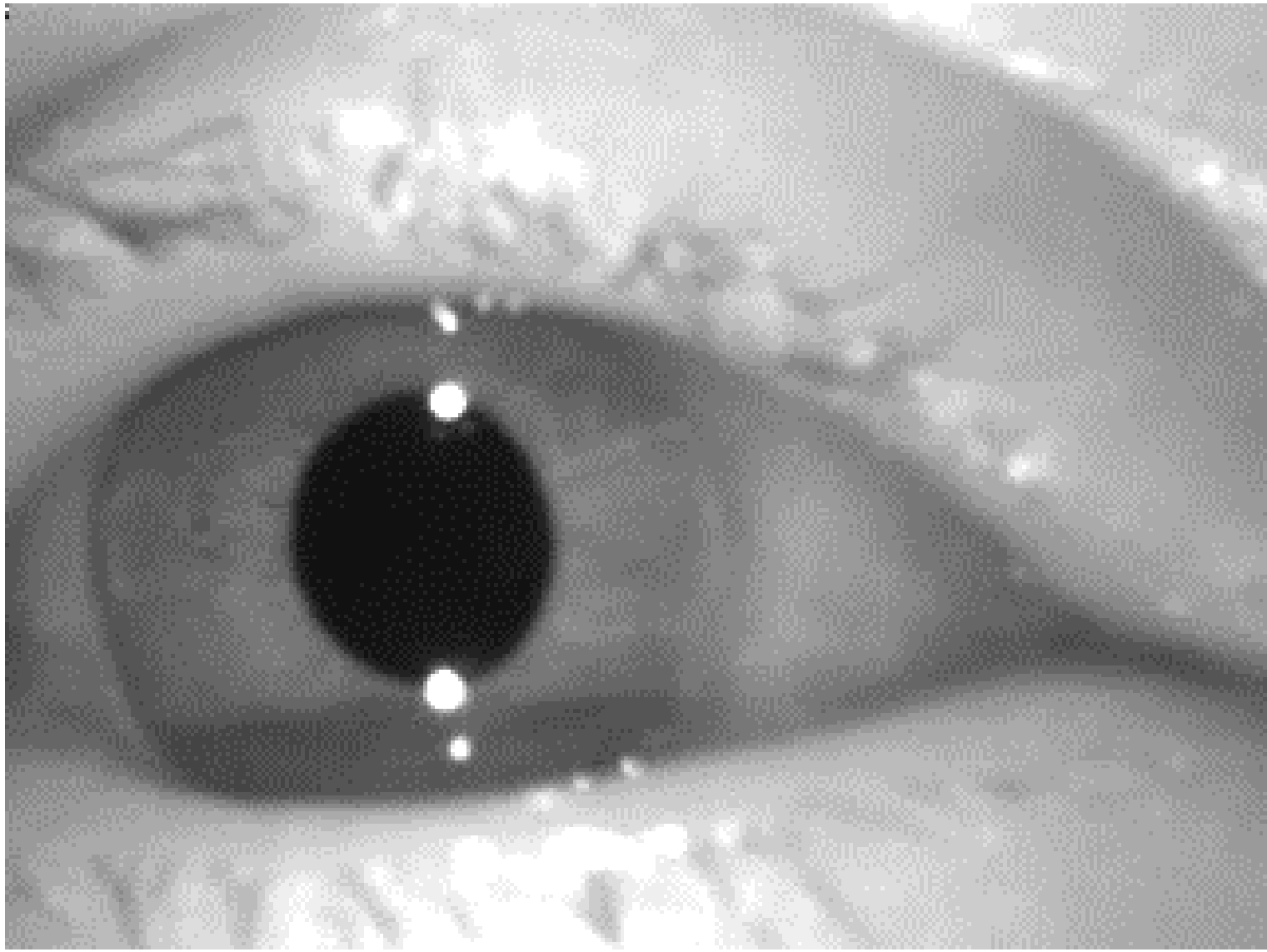
Dix-Hallpike — Head Right Test: 9/5/2018 2:57:31 PM
Test Operator: Default Administrator

HORIZONTAL NYSTAGMUS (9 deg/sec) WITH Dix-Hallpike MANEUVER

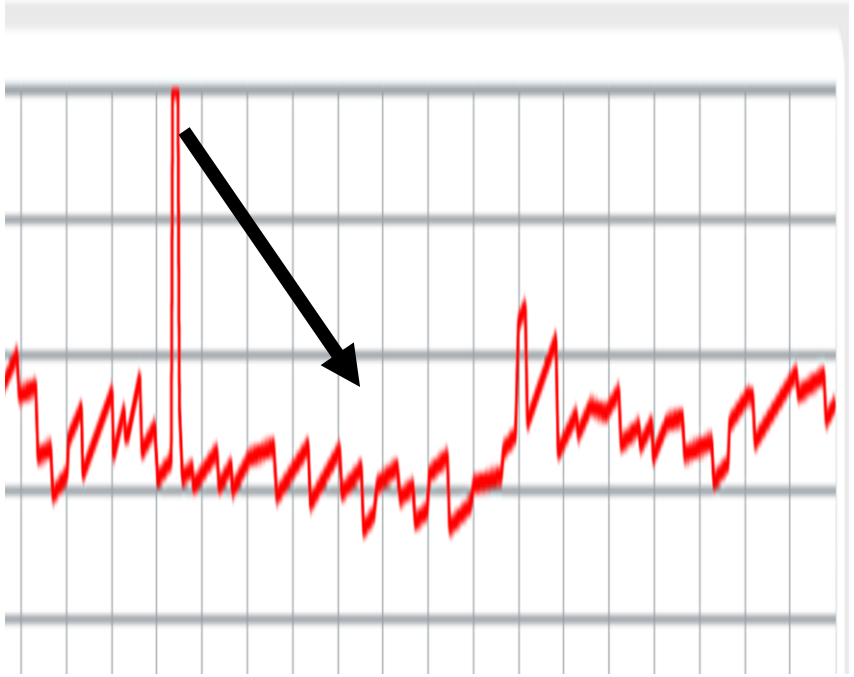


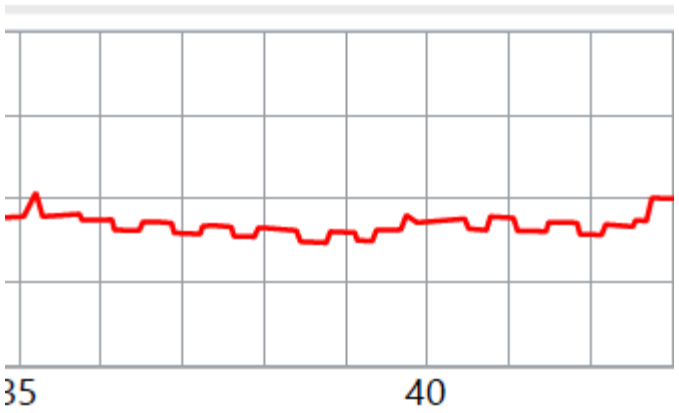
WHERE IS THE NYSTAGMUS?



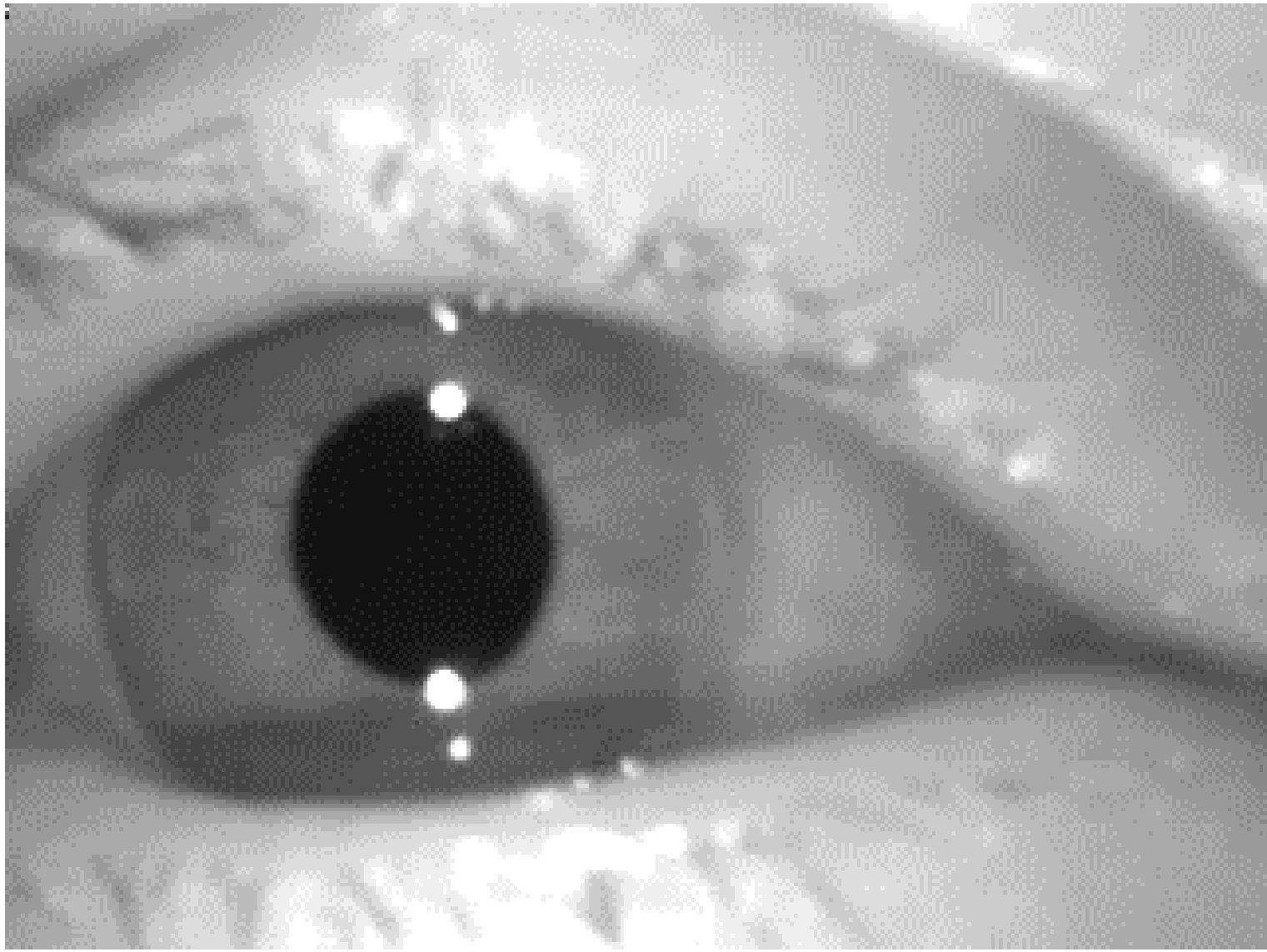


WHERE IS THE NYSTAGMUS?

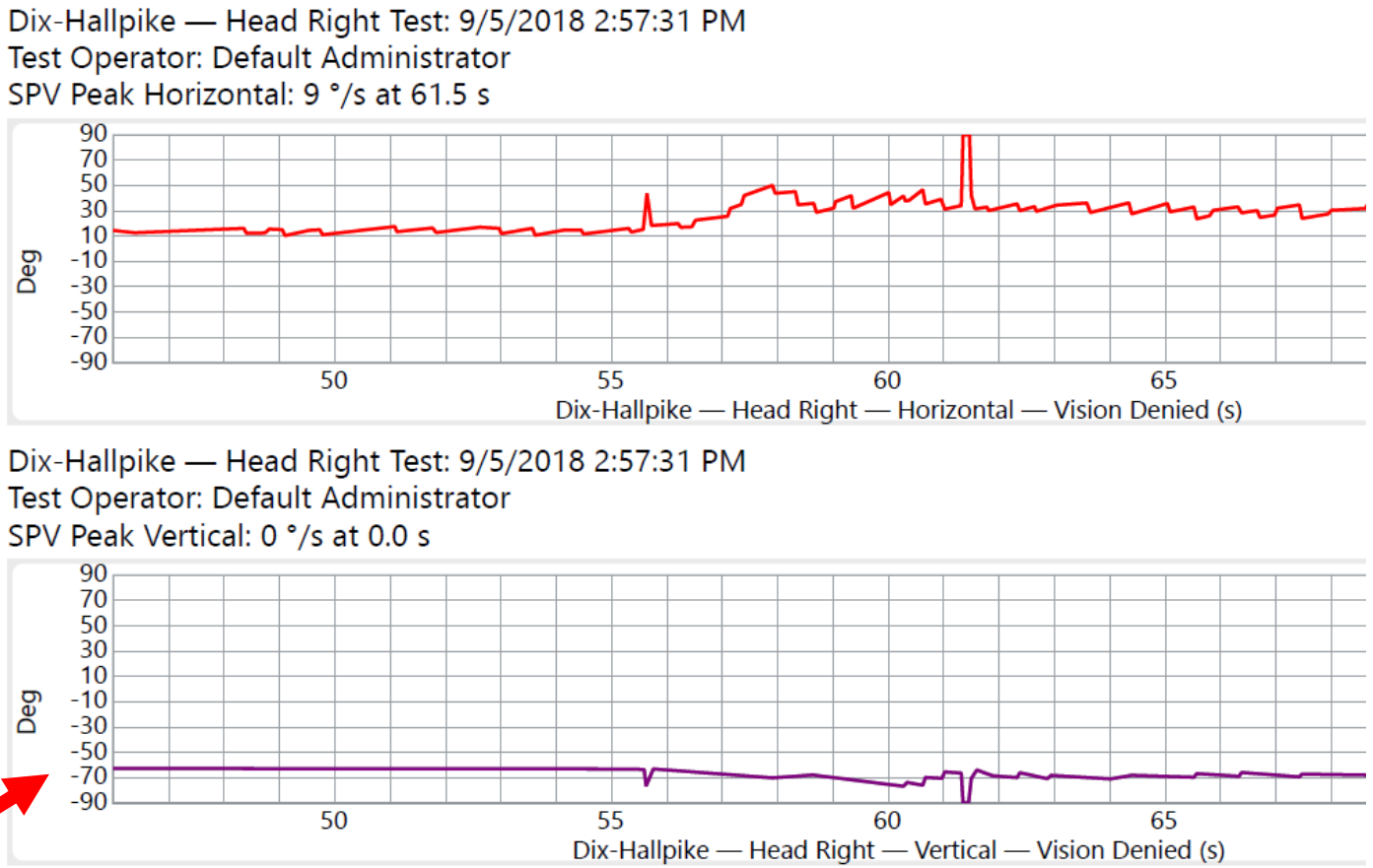




Square Wave Jerks may confuse the nystagmus algorithm??



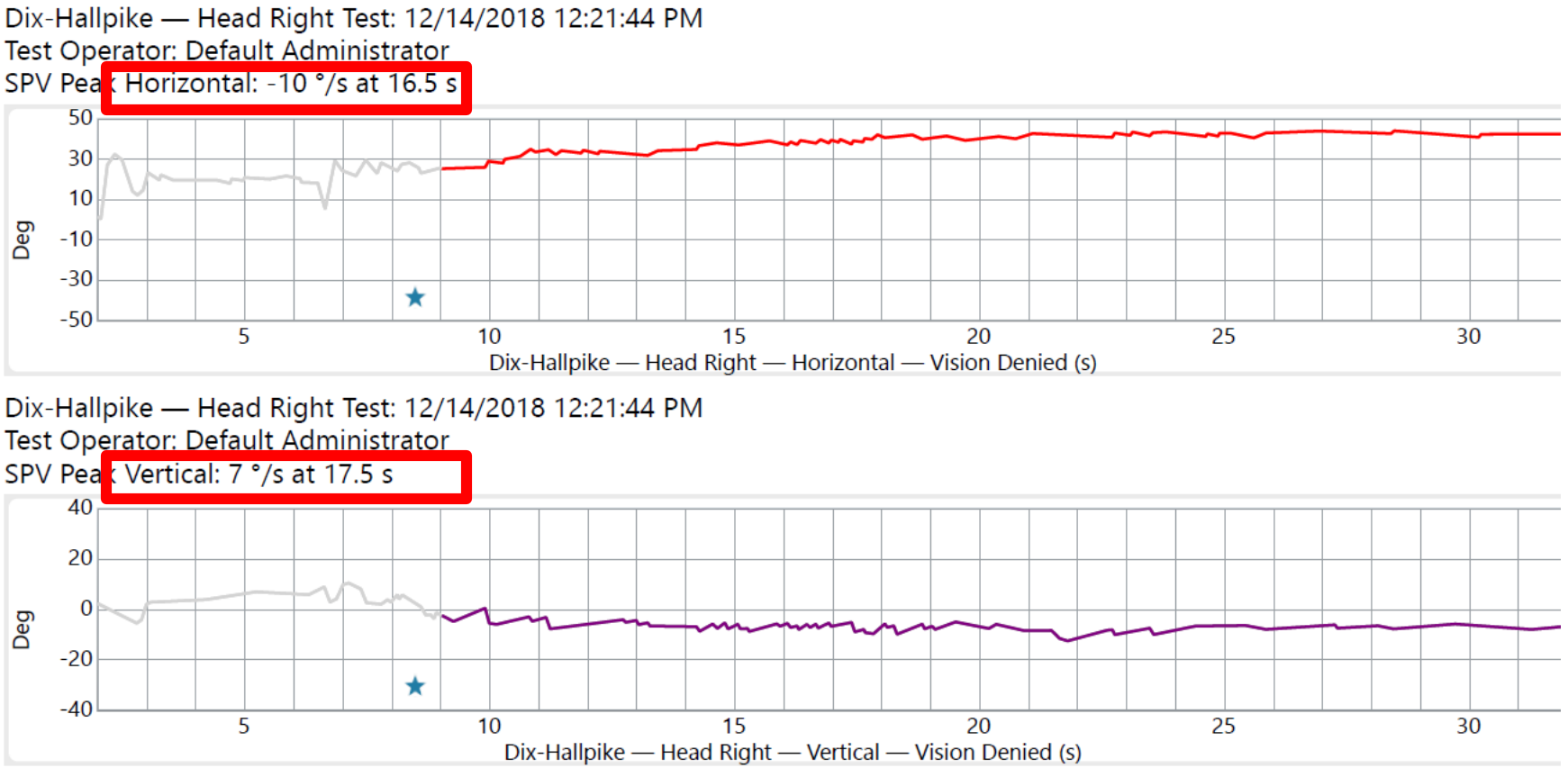
Which is right? The VOG record or the Video

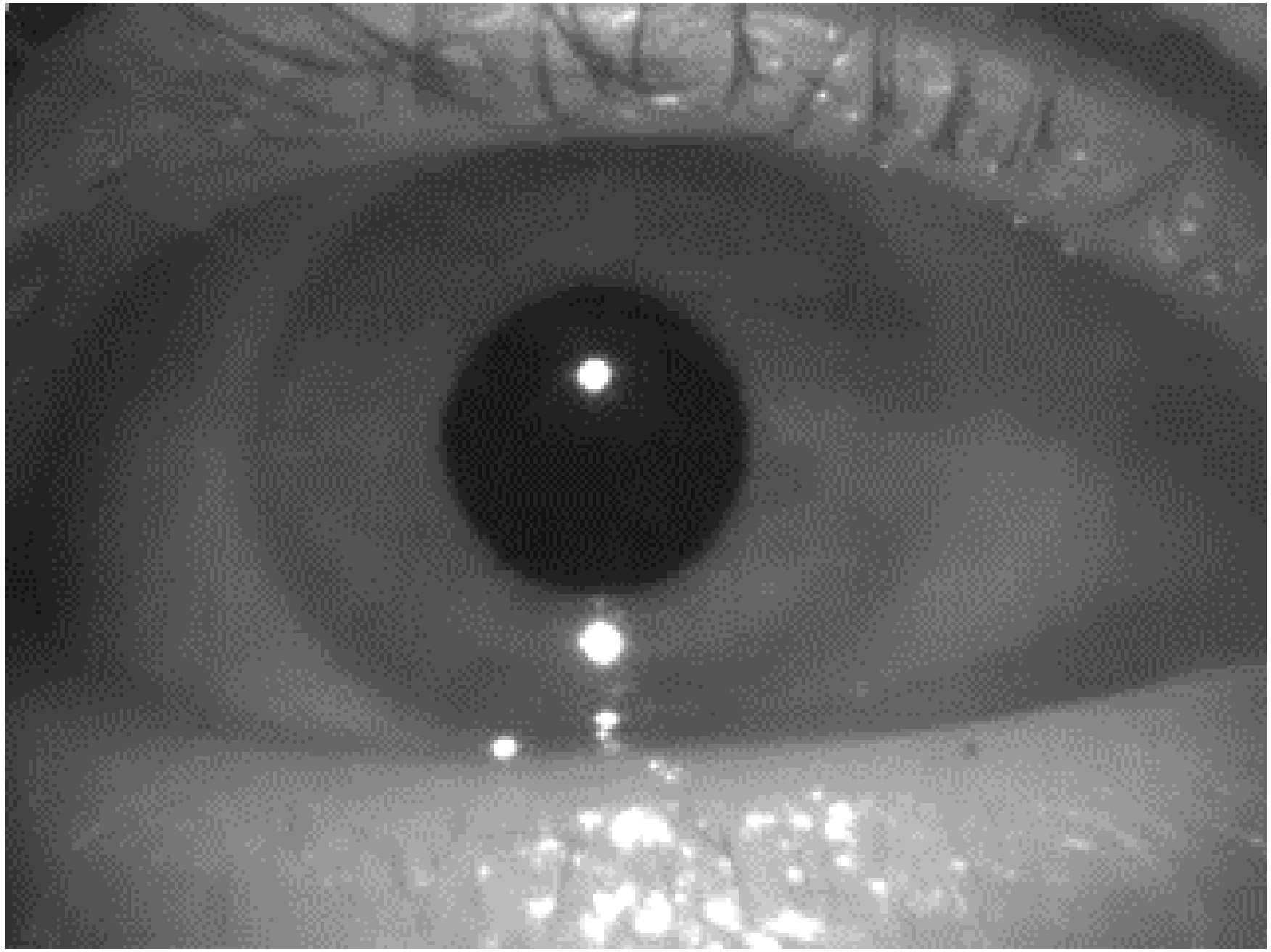


- **62 y.o. male patient who presents to the emergency department today with vertigo.**

  Patient states that he woke up from sleep this morning, and felt like the room was spinning around him. Patient states that this was exacerbated when he tried to stand up, he felt like his balance was off, and like he was going to fall over. He states that that was more like a sharp headache.

VOG Record of Dix-Hallpike maneuver states RB nystagmus > DB nystagmus, with maximum about the same time

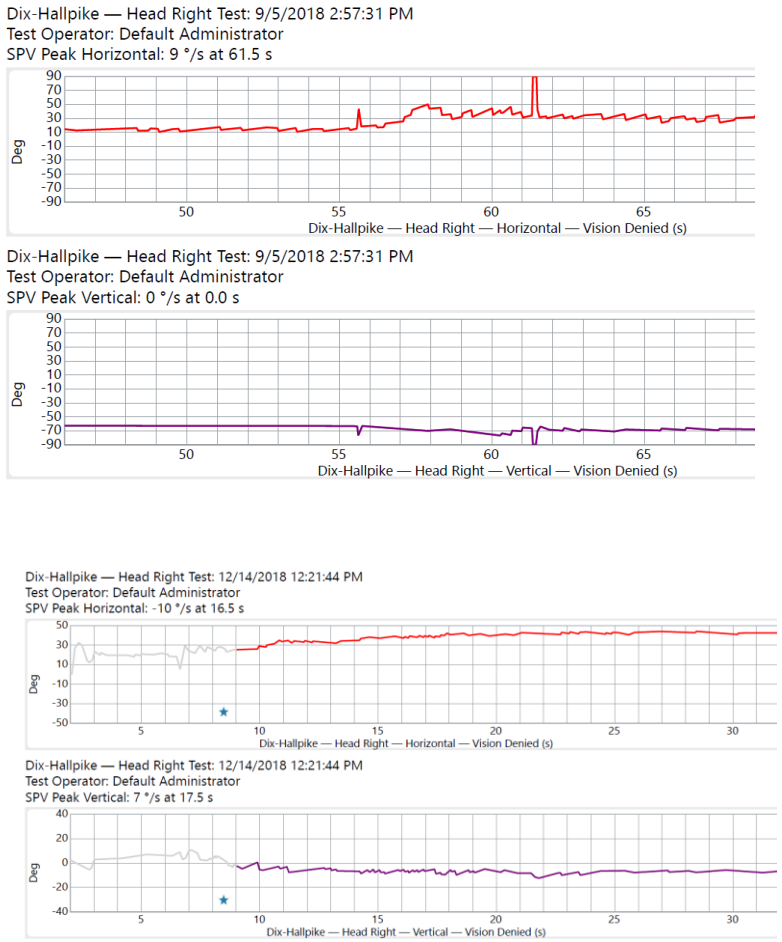




What is the takeaway message?

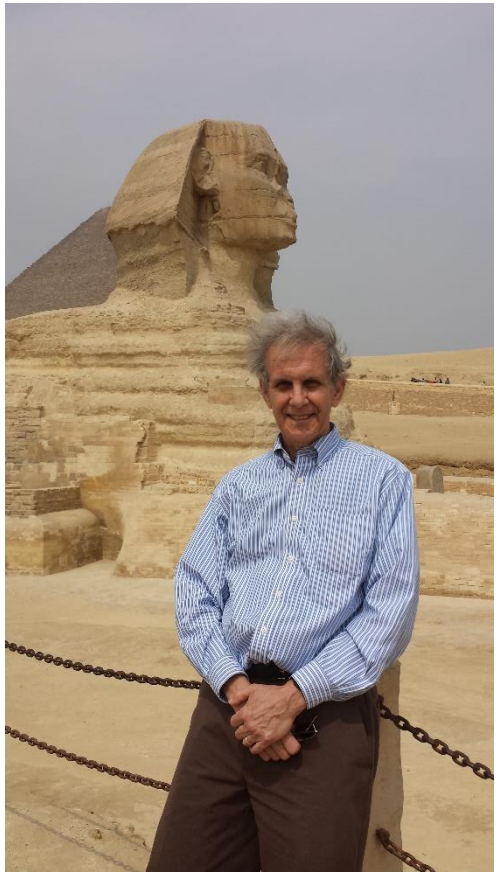
- **Trust your eyes**
- **Trust your brain**
- **Computers and algorithms are subject to human error**

MISLEADING NYSTAGMUS REPORTS



THE CORE ISSUES

AGAIN: Do I believe my own eyes and the interpretation of my brain, or do I believe the eyes and brains and numbers of the VOG machine?



The answer to this riddle is both and neither, and sometimes the truth can only be revealed using BOTH

NOTHING WRONG WITH MAKING A MEASUREMENT!

Quantitative measures **of VOR performance require a** ***quantifying technique.***

VOG goggles to measure VOR slow-phase gain.

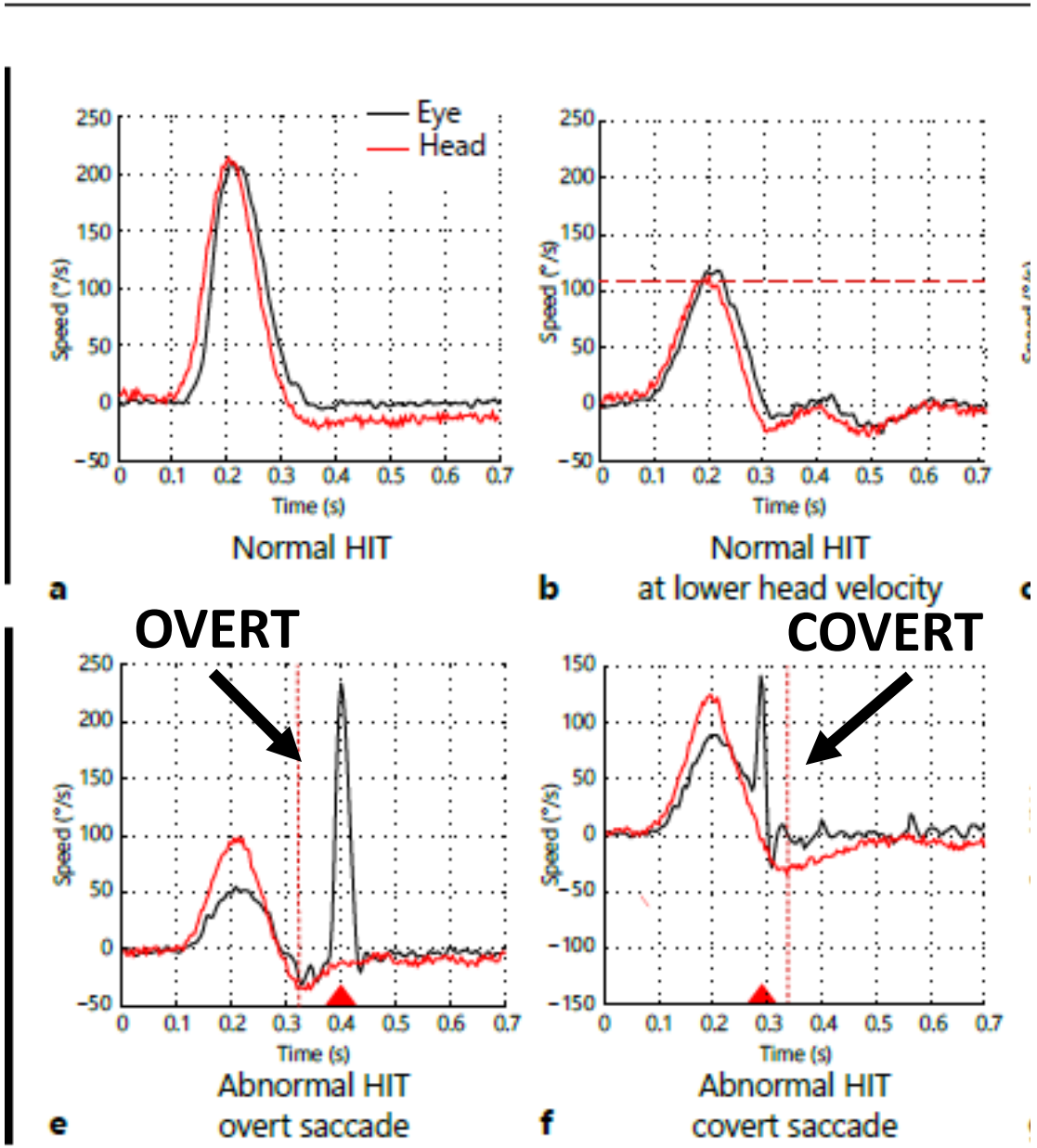
Quantitative DVA (dynamic visual acuity) to measure overall VOR function.

Corrective saccades, indicating an abnormal VOR, can often be seen clinically

IF *OVERT* (after the head movement), they are usually seen and are the signature of an abnormal VOR

IF *COVERT* (during the head movement), they may be only be revealed by VOG, especially in a well-compensated patient .

RELIABLE CORRECTIVE SACCADE RESPONSES

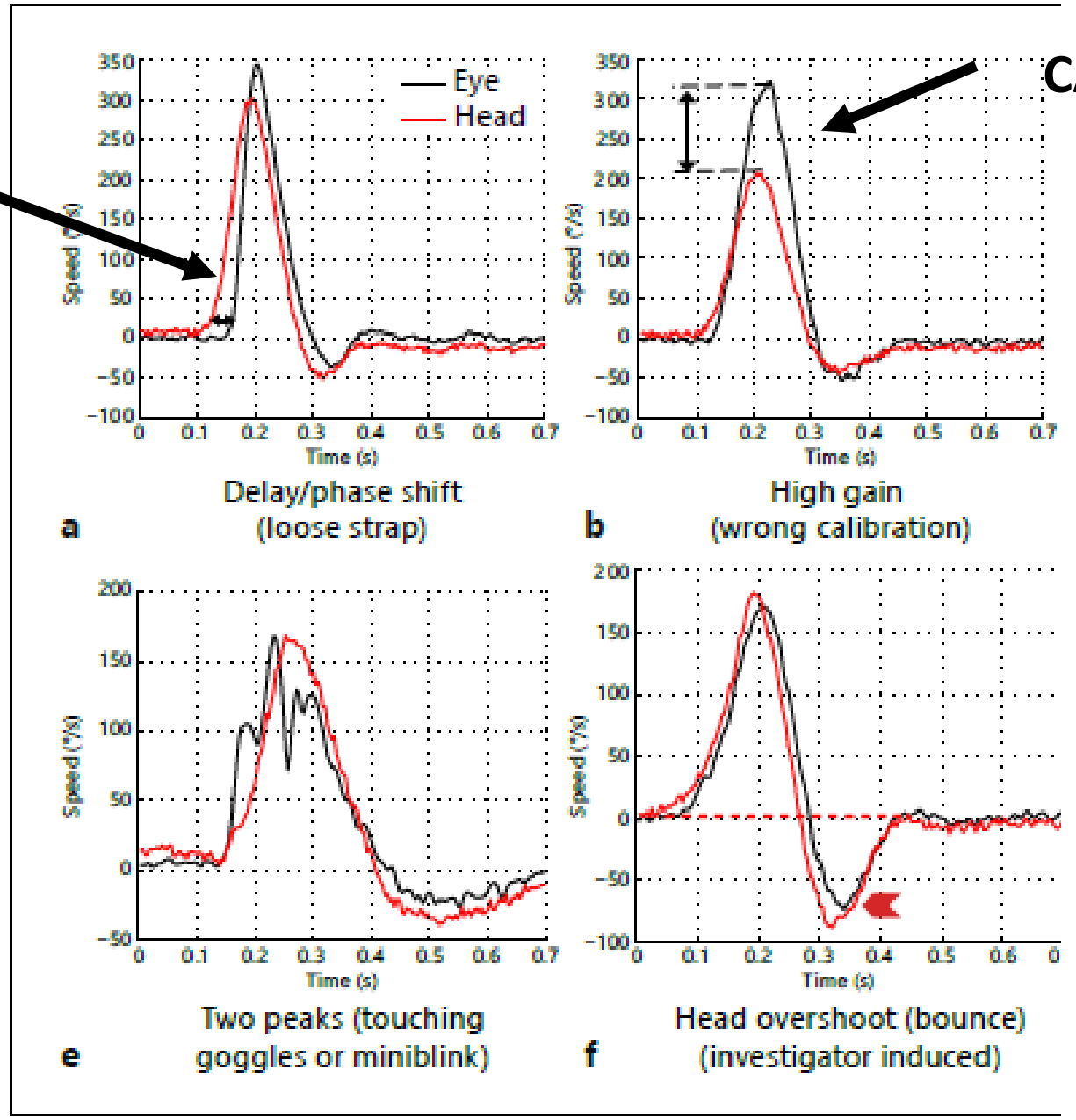


Mantokoudis, 2016

DELAY
GOGGLE SLIP

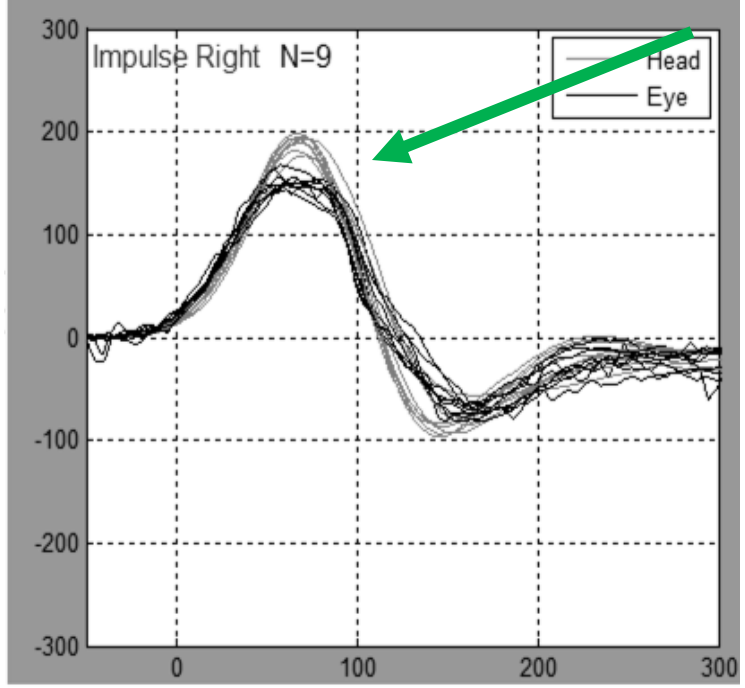
CALIBRATION ERROR

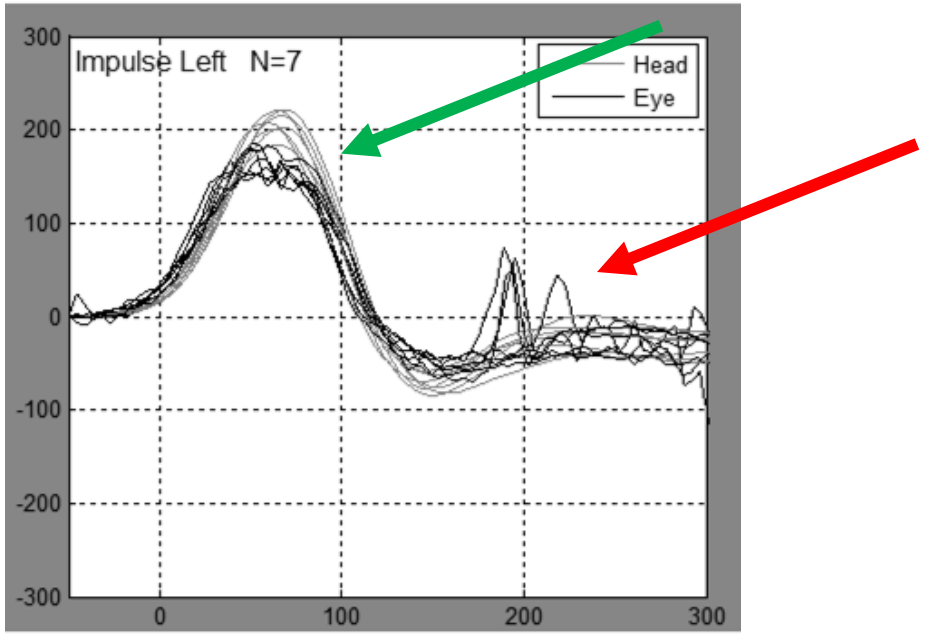
ARTIFACTS



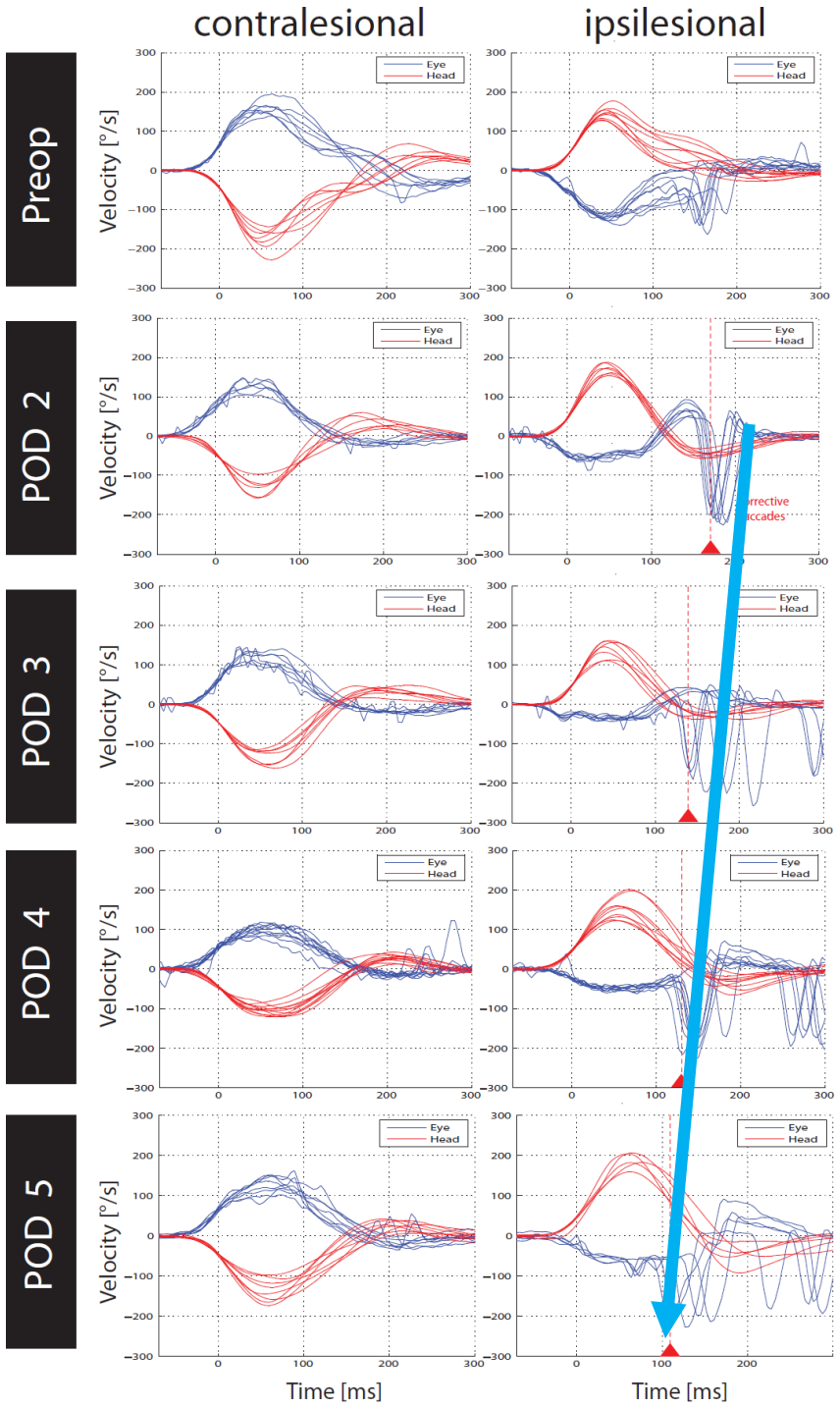
Mantokoudis, 2016

GAIN MAY LOOK THE SAME BUT **SMALL CORRECTIVE SACCADES** ARE THE CLUE TO HYPOFUNCTION





Yacovino, 2017



THINGS ONLY THE MACHINE CAN TELL US

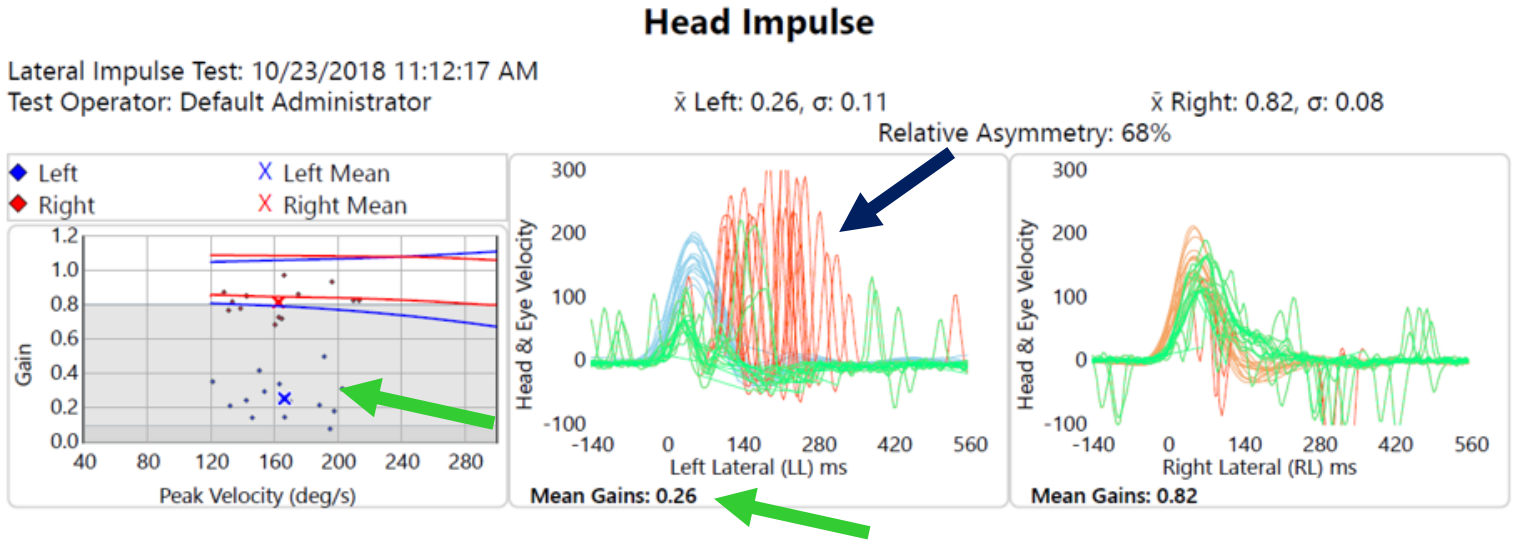
Quantification of changes in corrective saccade performance as a measure of adaptation

Corrective saccades occur earlier and earlier and become covert over the first five post-op days

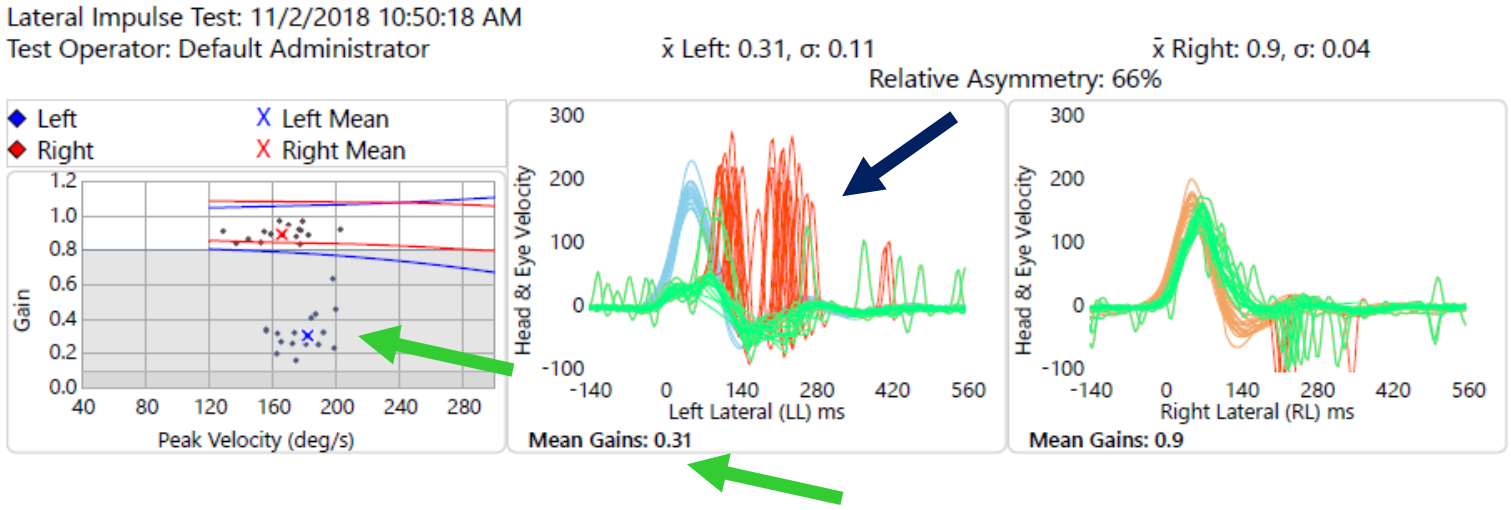
Mantokoudis, 2016

ADAPTATION TO A UNILATERAL VOR DEFICIT: Early and late VHIT with vestibular neuritis

48 hours



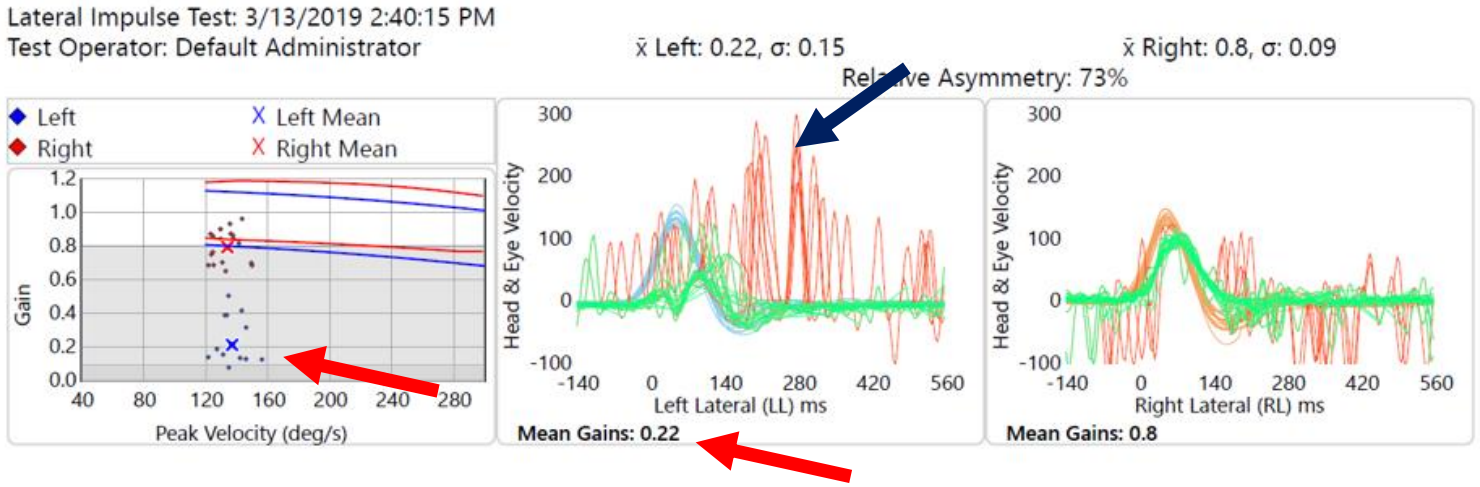
10 days



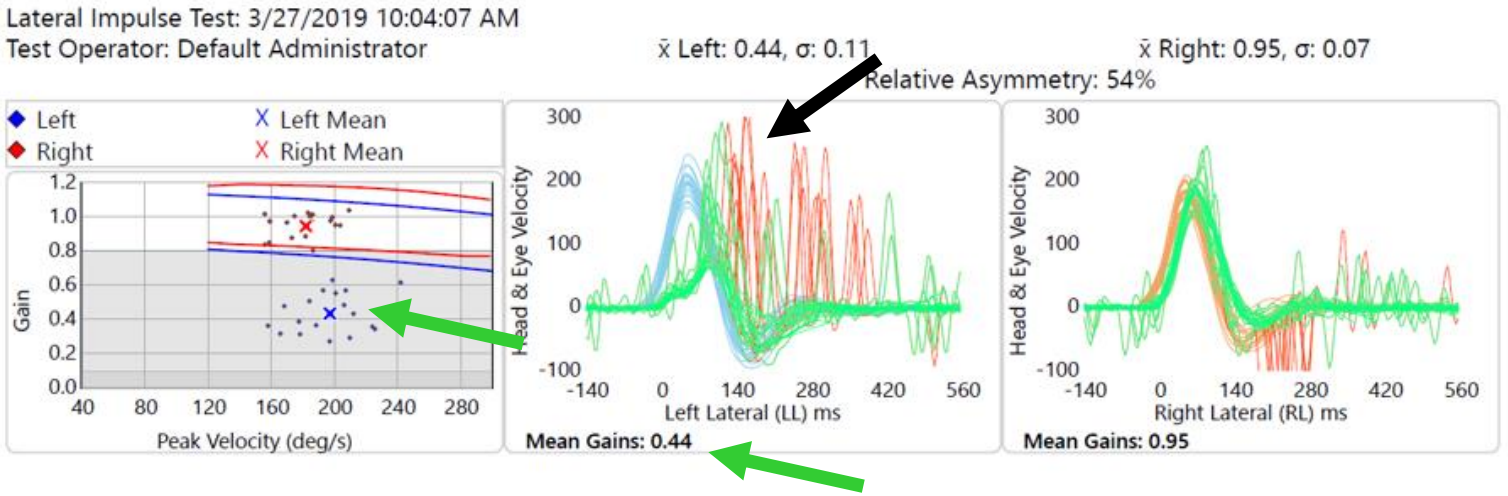
Clustering and tightening of the distributions of covert and of overt saccades with only small change in the slow-phase VOR gain

ADAPTATION TO A UNILATERAL VOR DEFICIT: Early and late VHIT with vestibular neuritis

24 hours



15 DAYS



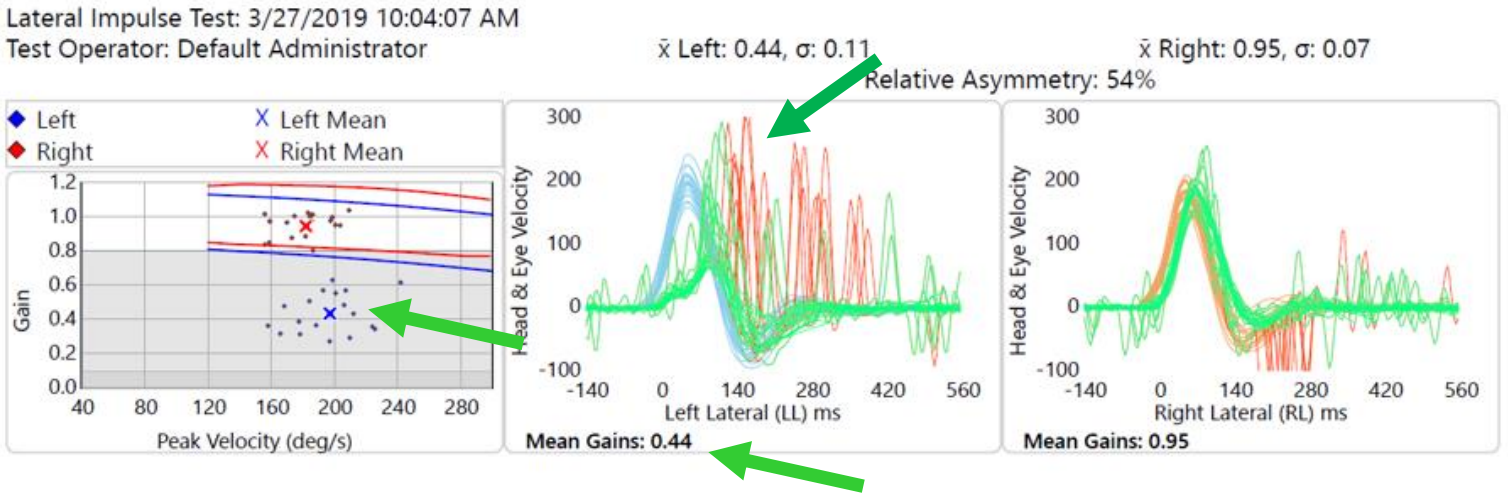
Corrective saccades occurring earlier (covert) with doubling of the slow-phase VOR gain

ADAPTATION TO A UNILATERAL VOR DEFICIT: Early and late VHIT with vestibular neuritis

24 h

CORRECTIVE SACCADES ARE USED TO ADAPT TO A VOR DEFICIT, ESPECIALLY FOR HIGH-SPEED, HIGH ACCELERATION ROTATIONS.

15 DAYS



Corrective saccades occurring earlier (covert) with doubling of the slow-phase VOR gain

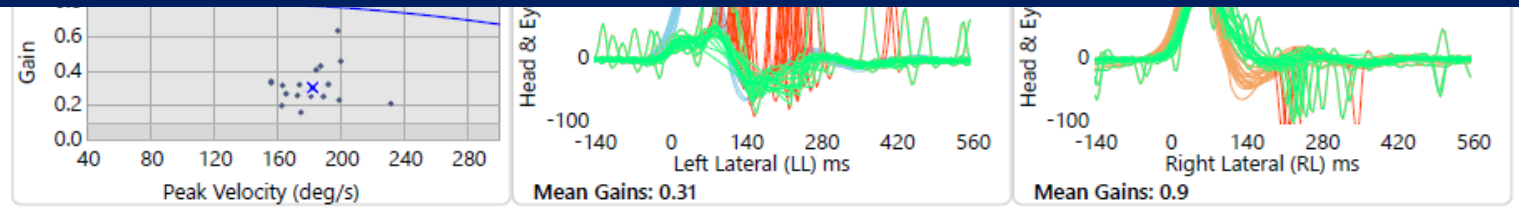
DOES THE VHIT GIVE US THE ANSWER ABOUT COMPENSATION?

Knowing WHEN a saccade occurs is NOT ENOUGH.

To WHERE the saccade takes the fovea is the vital question.

Is the position of the image of interest on the fovea?

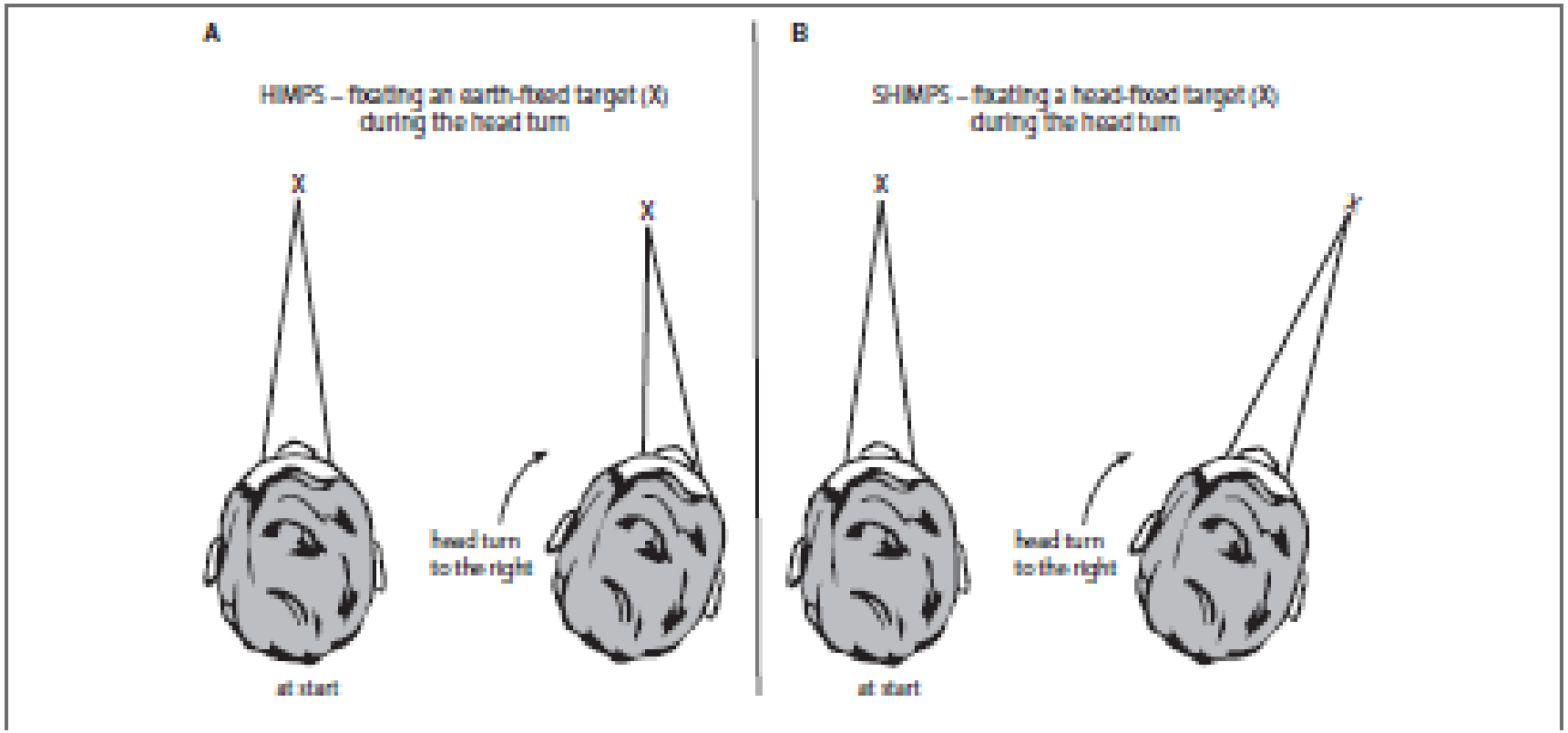
This is not revealed by current VOG recording algorithms.



THE LATEST: HIMP (head impulse) and *SHMP* (suppression head impulse)

HIMP uses a *wall-FIXED* target *stationary* in front of the patient to induce a *normal* VOR response

***SHMP* uses a *head-FIXED* laser target that *moves* with the head to induce a *suppression* VOR response**



MacDougall, 2016

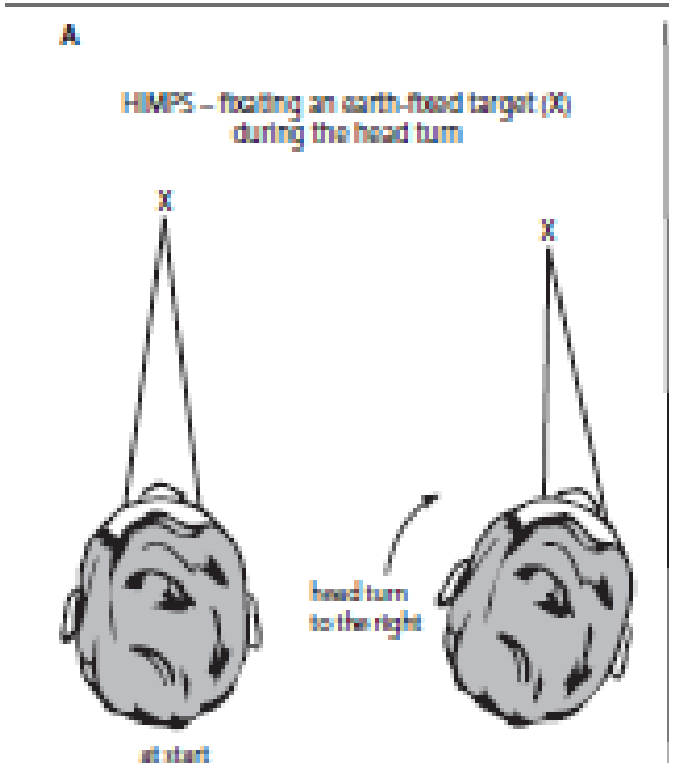
Normal Participant

HIMP

Conventional Head Impulse Paradigm

Earth-Fixed Target

Neurology®



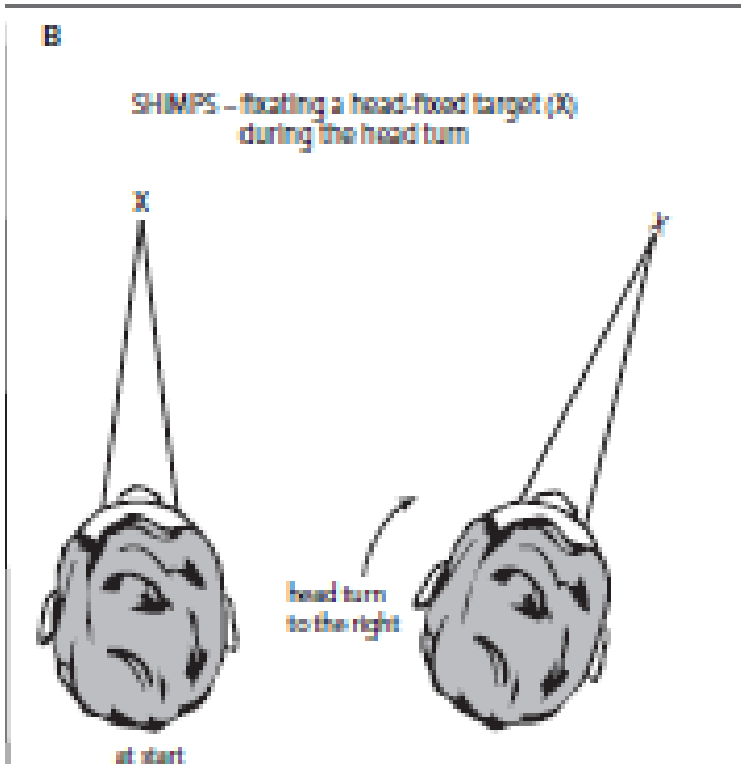
Normal Participant

SHIMP

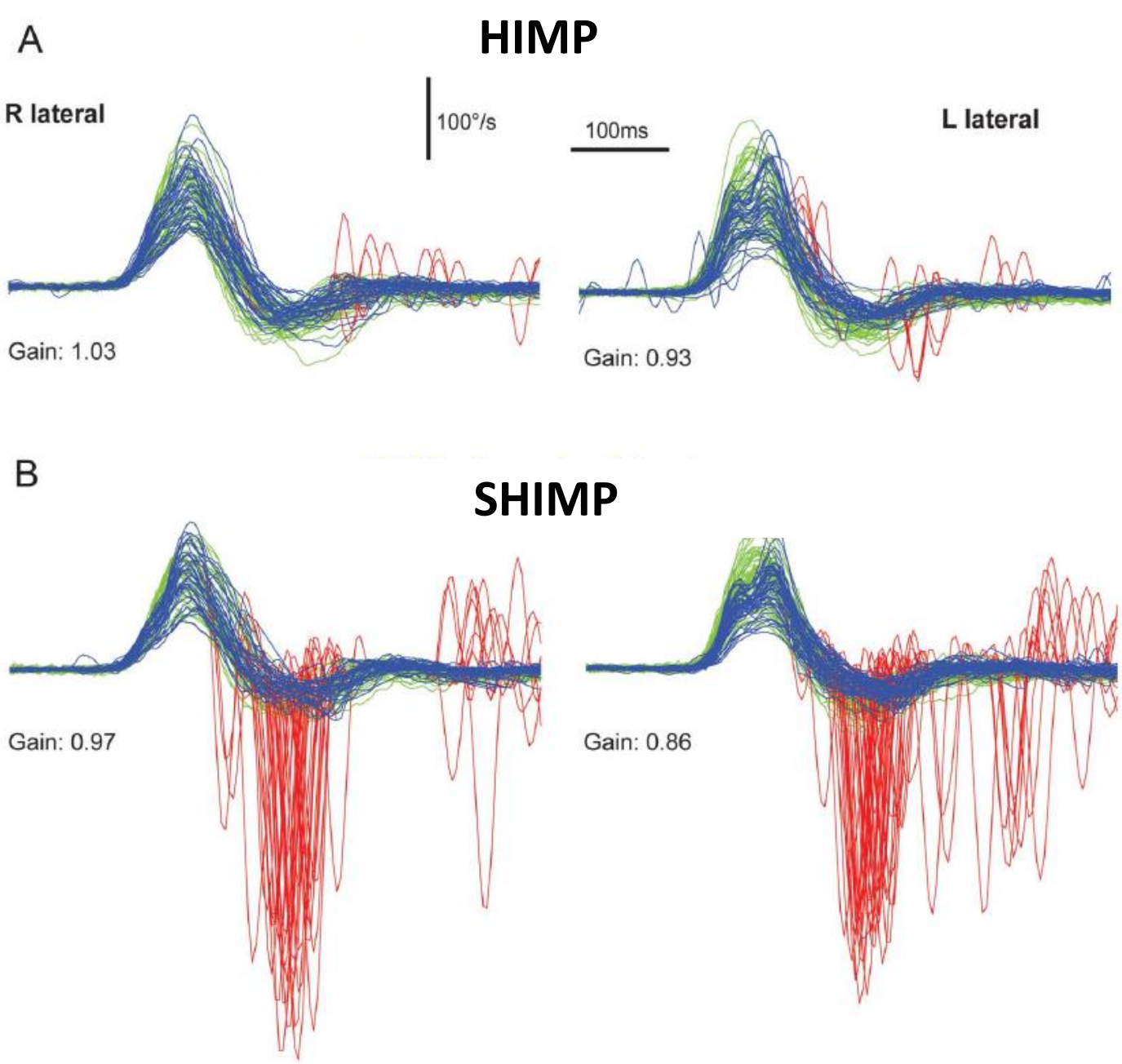
Suppression Head Impulse Paradigm

Head-Fixed Laser Target

Neurology®



NORMAL SUBJECT



NO CORRECTIVE SACCADES in a normal subject

CORRECTIVE SACCADES in a normal subject

MacDougall, 2016

UNILATERAL LOSS PATIENT

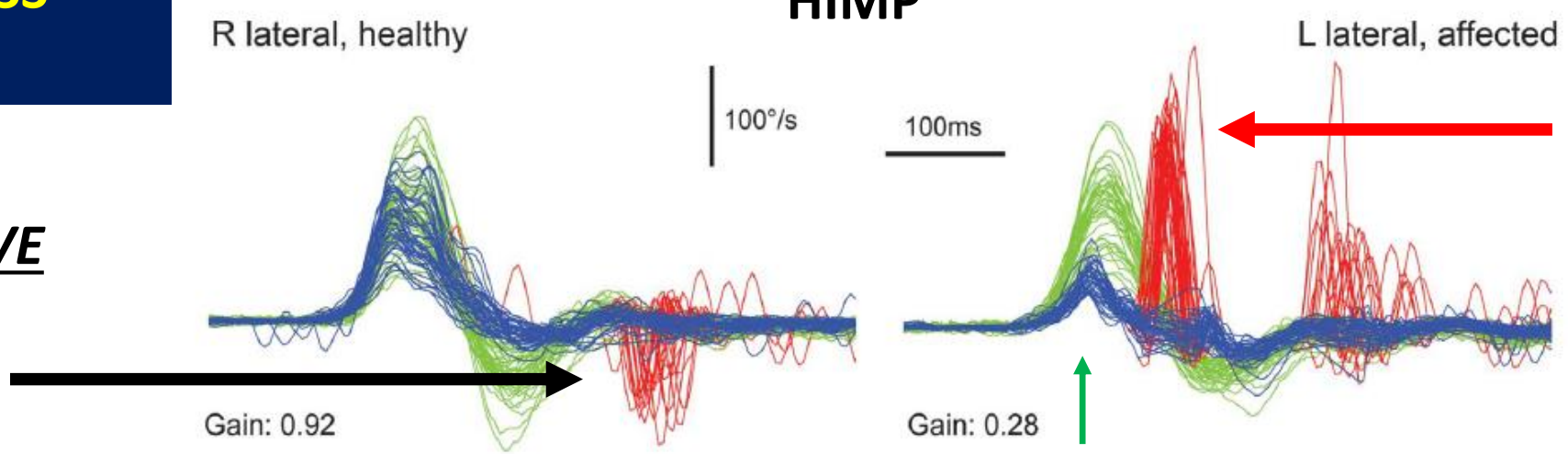
NORMAL SIDE

AFFECTED SIDE

HIMP

NO CORRECTIVE SACCADES

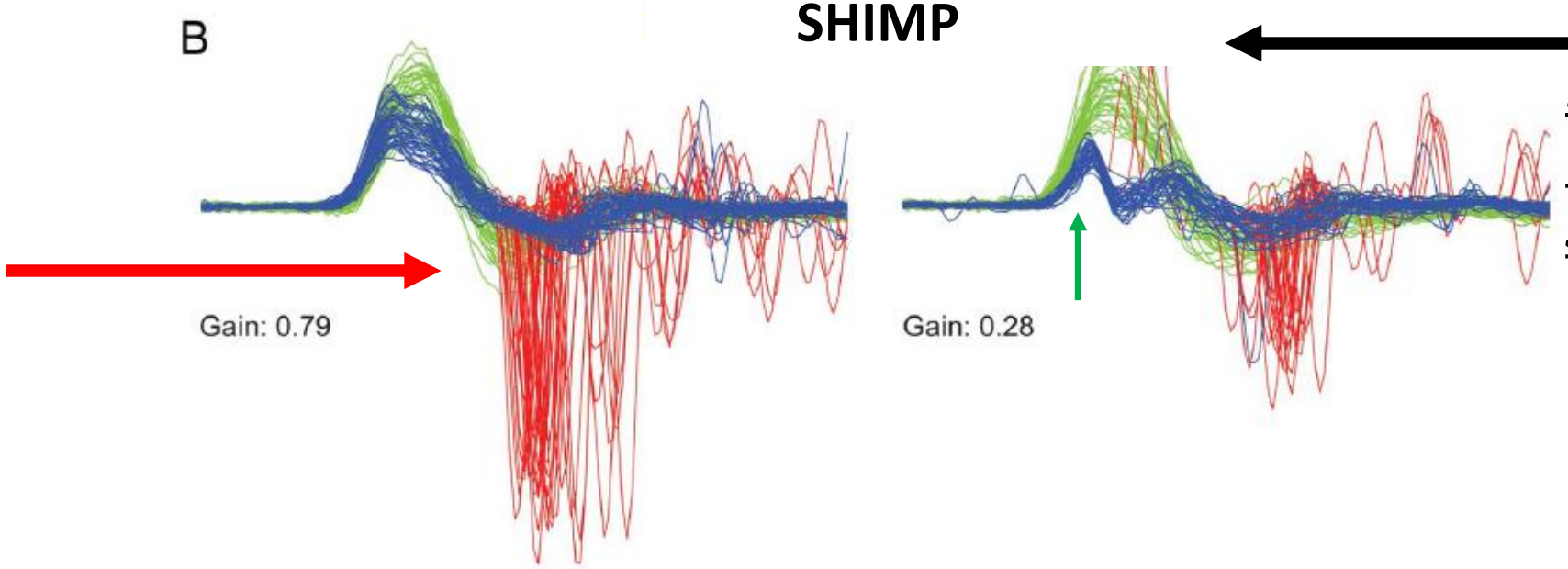
CORRECTIVE SACCADES



SHIMP

CORRECTIVE SACCADES

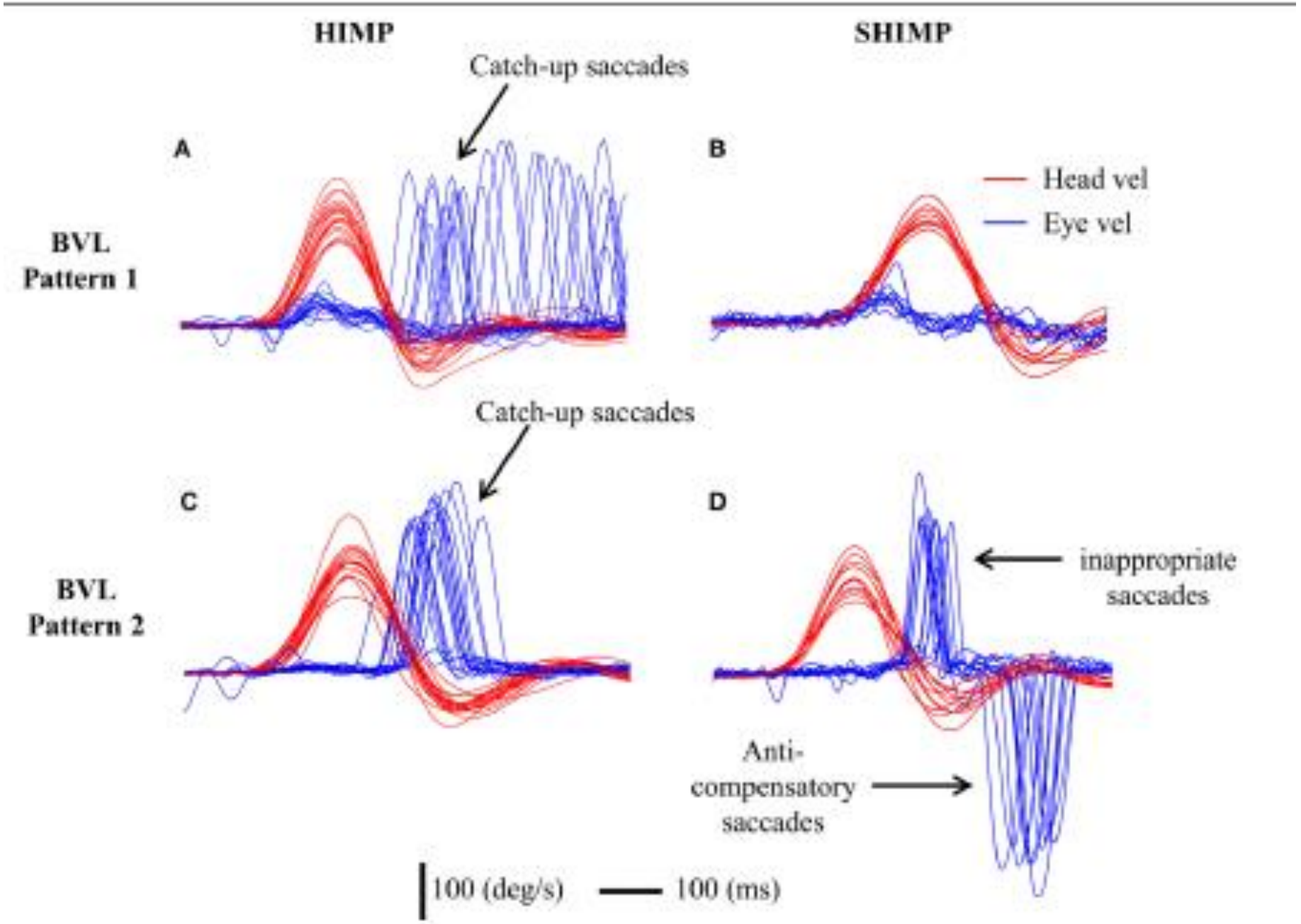
NO CORRECTIVE SACCADES



MacDougall, 2016

BILATERAL LOSS PATIENTS:

PREPROGRAMMED CORRECTIVE SACCADES



Waele, 2017

WHY DO A SHIMP?

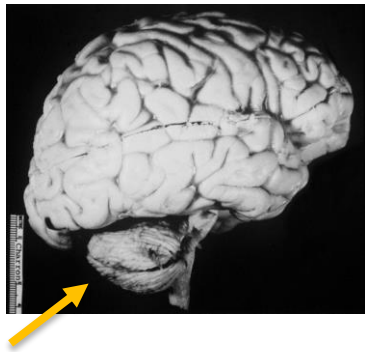
- **IF there is a slow-phase deficit, it may be easier to calculate the slow-phase VOR gain with SHIMP because covert corrective saccades, seen in the HIMP, are no longer needed or present, and so do not confound the measures of the slow-phase response.**

- **SHIMP allows you to study predictive factors on slow-phase gain (the subject knows the target is head fixed and will need to suppress the VOR slow phase.) as well as on saccade generation**

- **SHIMP allows you to study corrective saccade mechanisms (and adaptation) by comparing with corrective saccades in HIMP.**

MORE BEDSIDE THINKING!

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

Patient with cerebellar disease

Impaired **pursuit**

Intact VOR **suppression**

=

VOR

BILATERAL LOSS

Recall VOR suppression is a pursuit function

CEREBELLAR DYSFUNCTION PLUS VOR LOSS

Impaired **pursuit**

Intact VOR **suppression**

=

VOR

BILATERAL LOSS

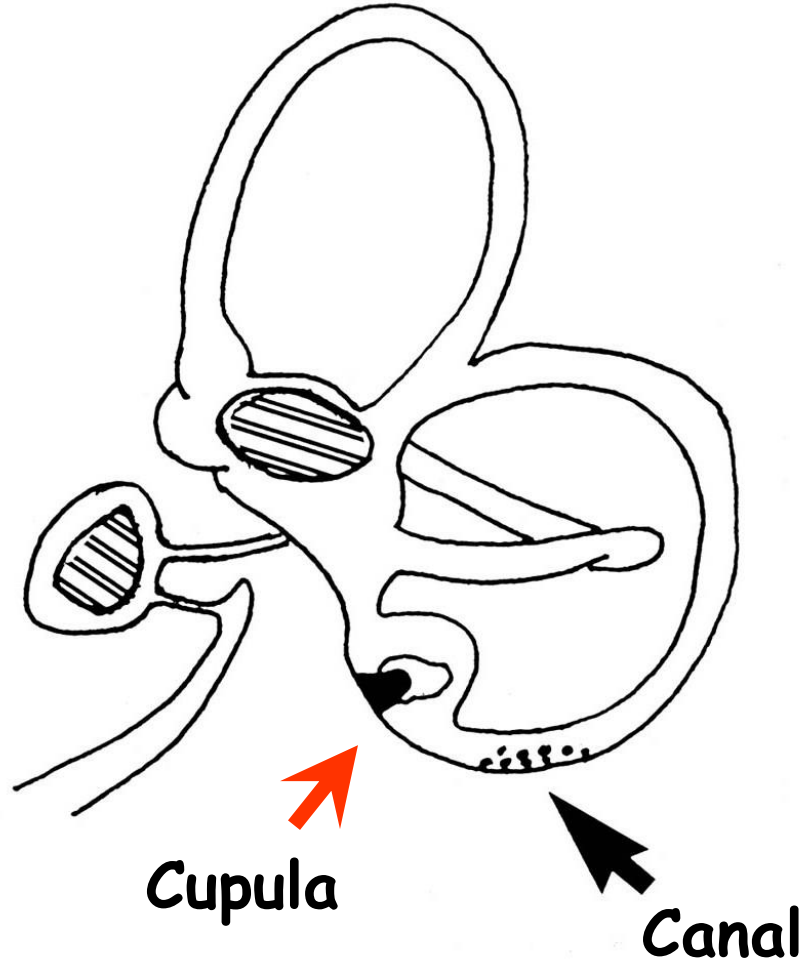
CANVAS syndrome (Cerebellar ataxia, neuropathy, vestibular areflexia syndrome), autosomal recessive

- Recessive disorder ((chromosome 4, RFC1, DNA repair mechanism (replication factor D subunit 1)
- Progressive ataxia
- As many as 25% of LOCA (late onset cerebellar ataxia without a FH may carry this genetic defect)
- Profound sensory neuropathy including on face (ganglioneuropathy)
- Chronic cough
- Loss of sweating and other autonomic features
- Loss of bilateral vestibular function
  - Differential diagnosis of ataxia plus vestibular areflexia
    - Friederich's ataxia
    - SCA3
    - MSA
    - Wernicke's disease
    - CJD
    - Superficial Siderosis
    - CANVAS

One more example: the clinical exam *"trumps"* imaging

- The HINTS bedside algorithm that warns for a stroke in the patient with an acute vestibular syndrome (AVS)
  - Head impulse test NEGATIVE (counterintuitive)
  - Gaze-evoked nystagmus, changing direction on right vs left gaze
  - Skew deviation (vertical misalignment)
  - (HINTS PLUS: unilateral hearing symptoms, inability to walk)
- HINTS is BETTER than an MRI at detecting a possible stroke in the first 48 hrs. Early negative MRI does not EXCLUDE a stroke in patients with positive HINTs
- 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

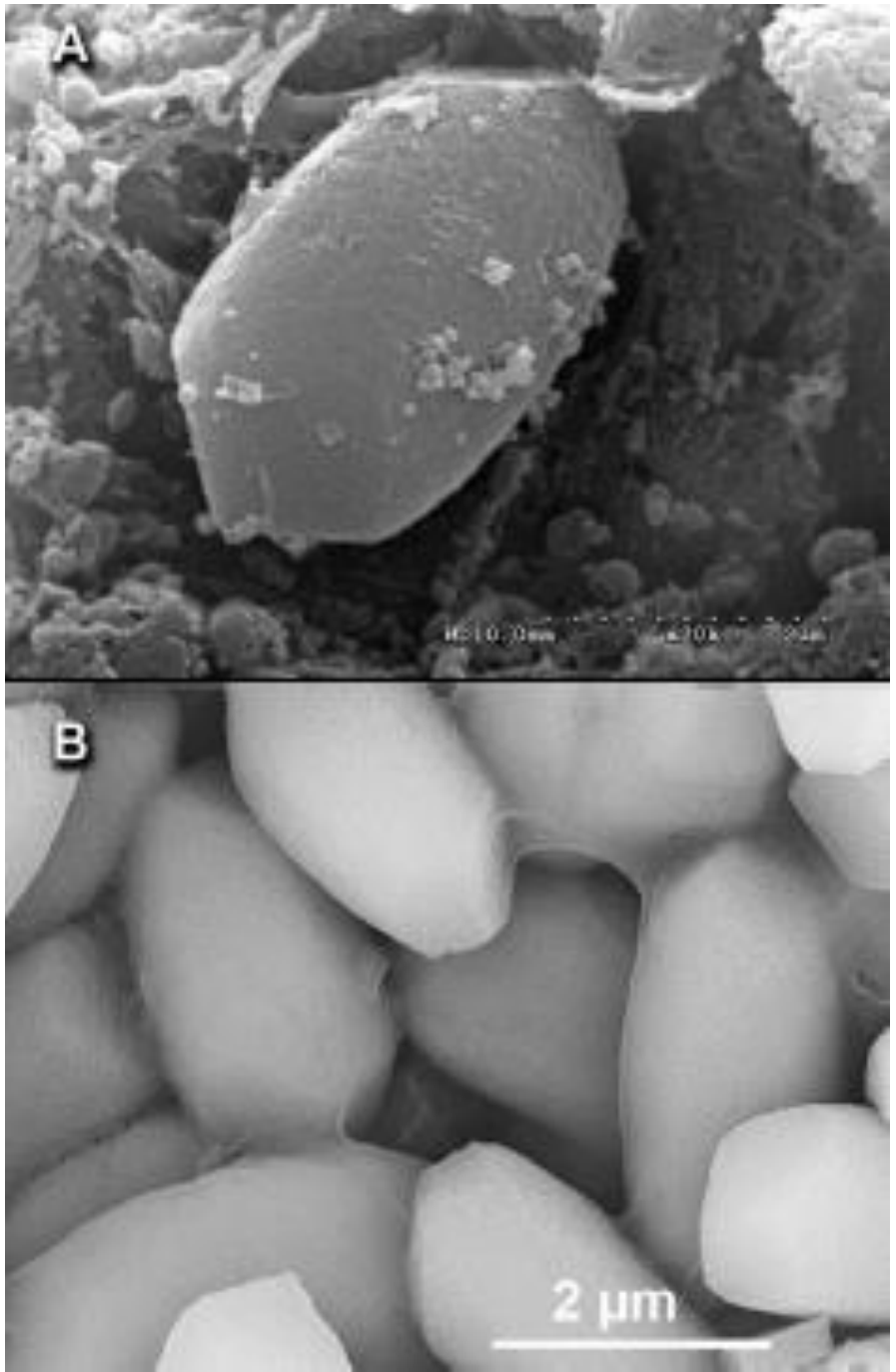
Benign Paroxysmal Positional Vertigo (BPPV)



OTOCONIA

Pathological

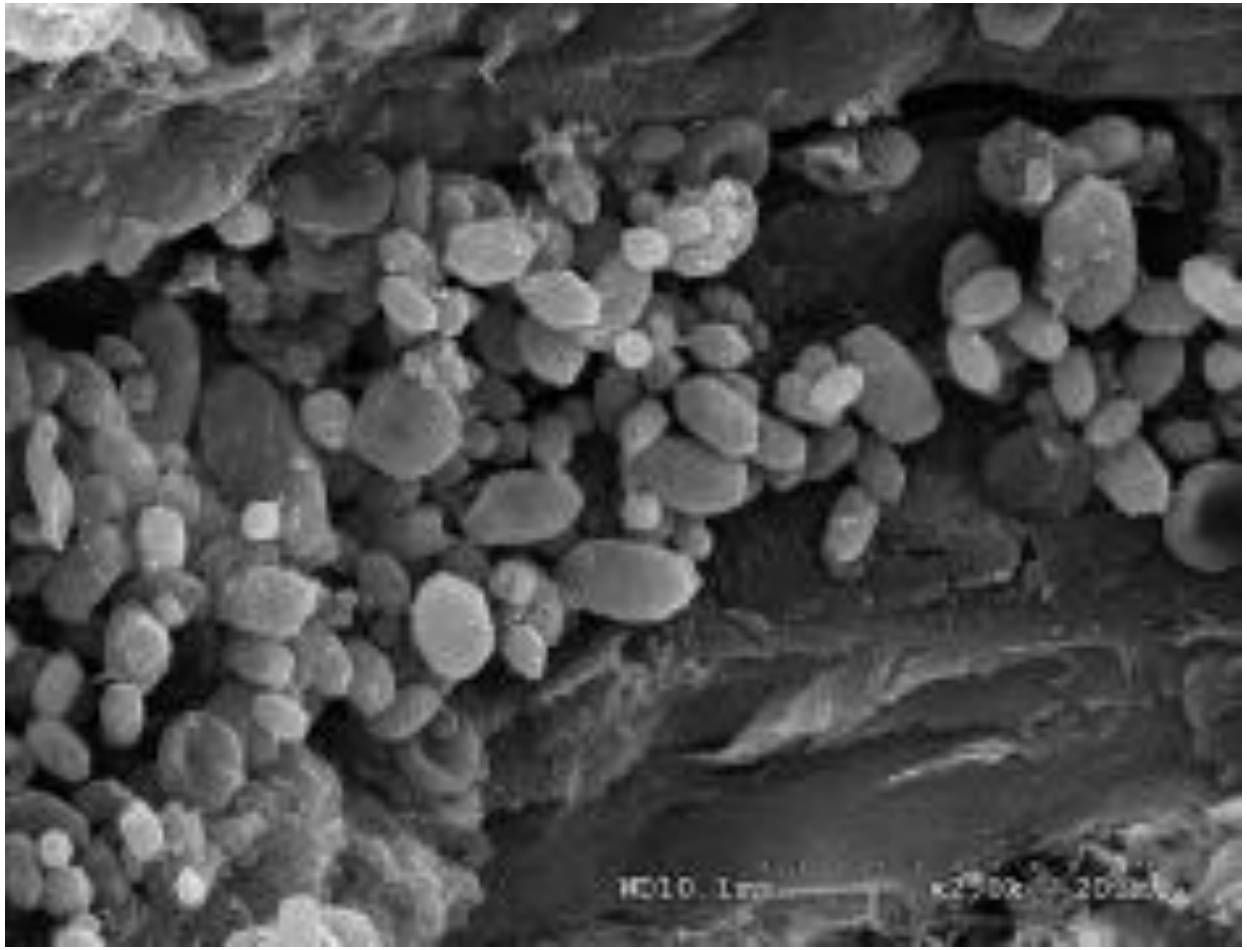
Normal



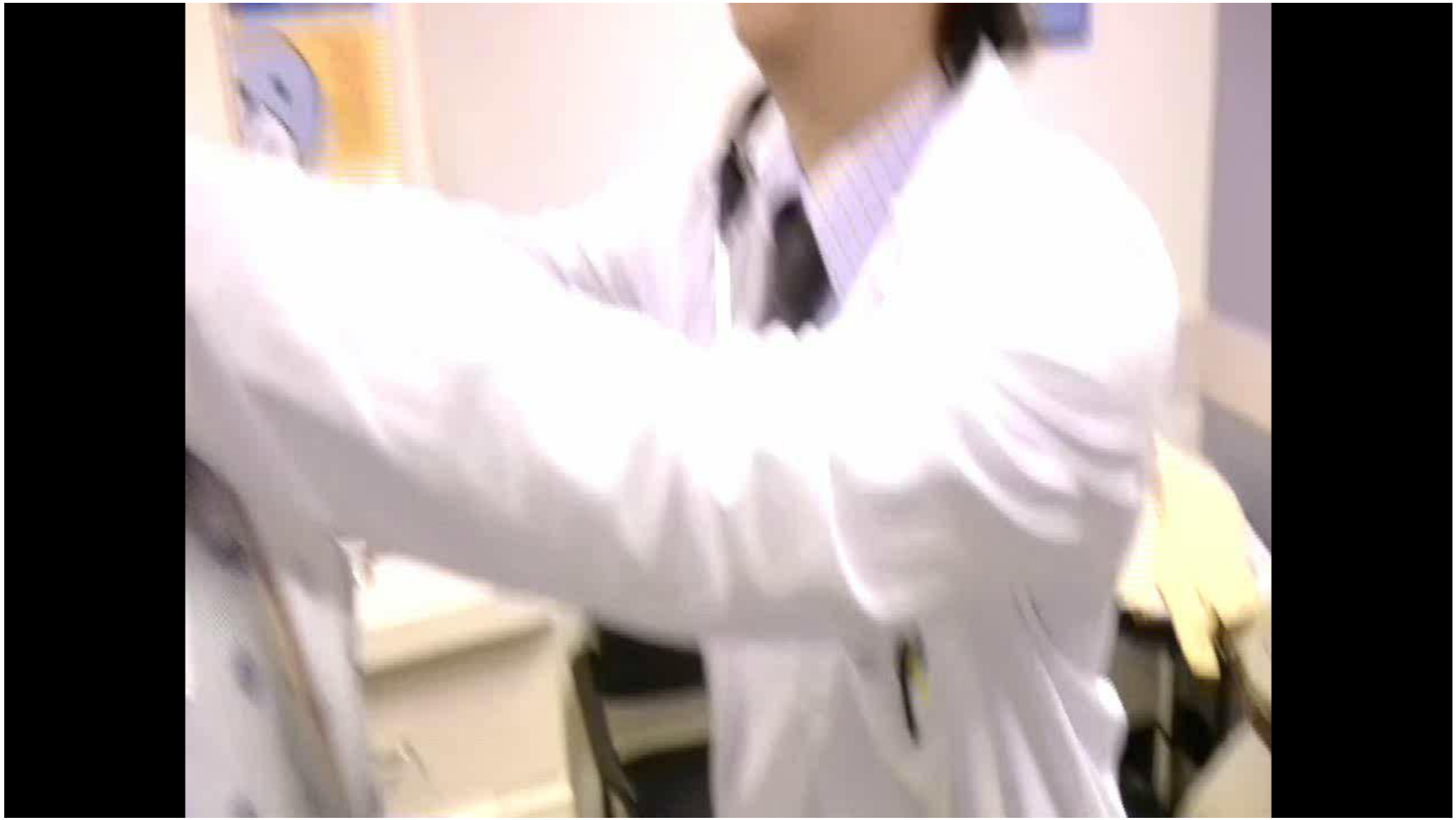
Kao, Parnes, 2017

Pathological

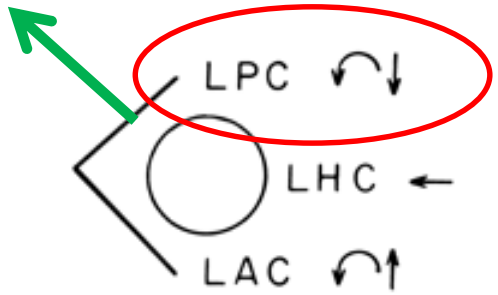
Kao, Parnes, 2017



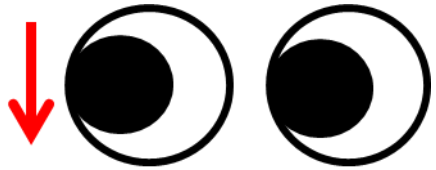
Rolling stones (otoconia) are not always free-floating but also embedded in floating otolithic membranes creating possibilities for peculiar patterns of benign positional nystagmus and “canalolithic jam”



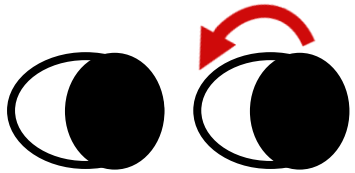
- **Ewald's First Law: Eyes (head) rotates in a plane parallel to that of head rotation as detected by the SCC in that plane. So the VOR stabilizes gaze (eye in space) around all three axes of head rotation NO MATTER WHAT THE DIRECTION OF GAZE, i.e., HEAD-FIXED**



Arrows indicate directon of slow phase with stimulation



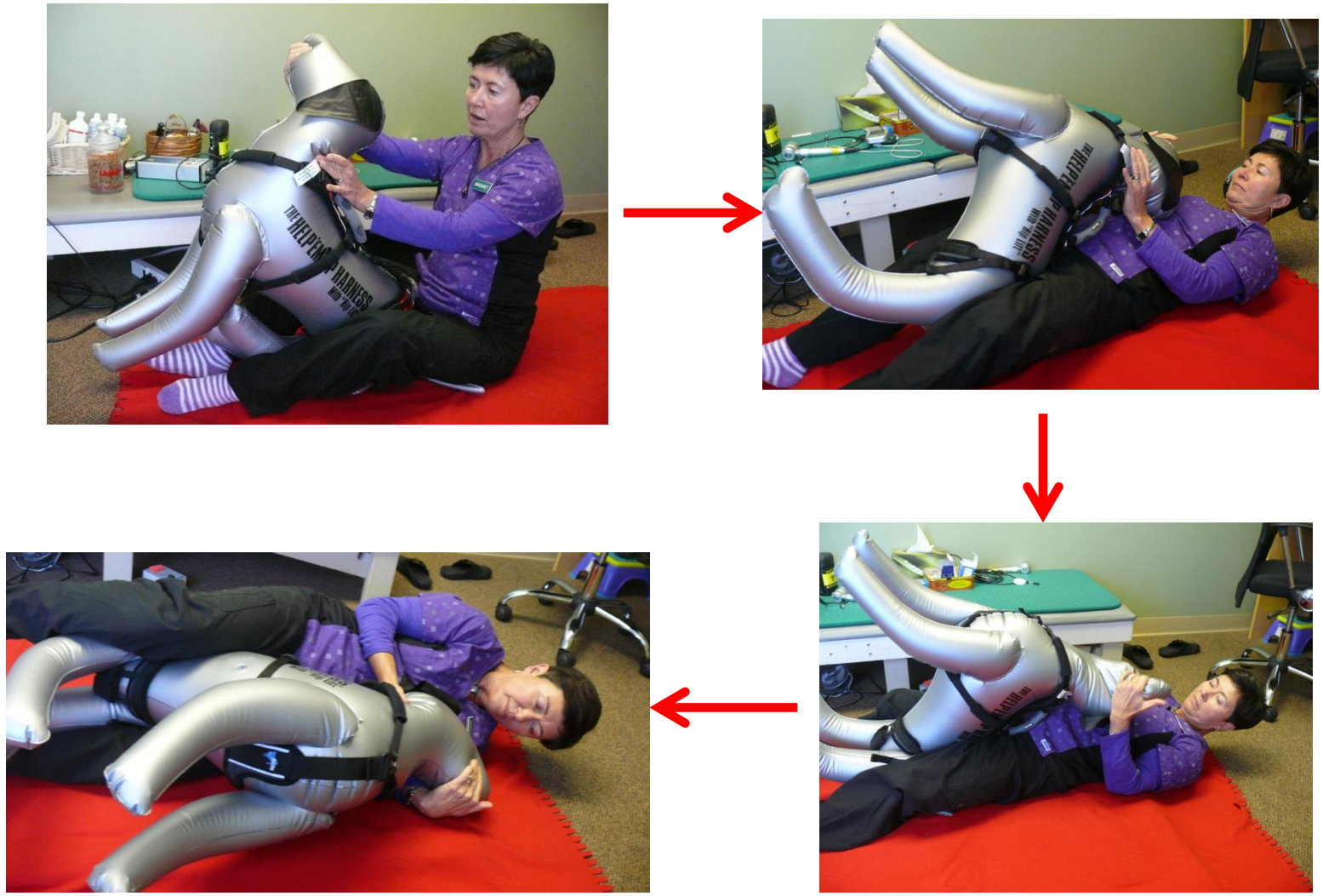
GAZE right



GAZE left

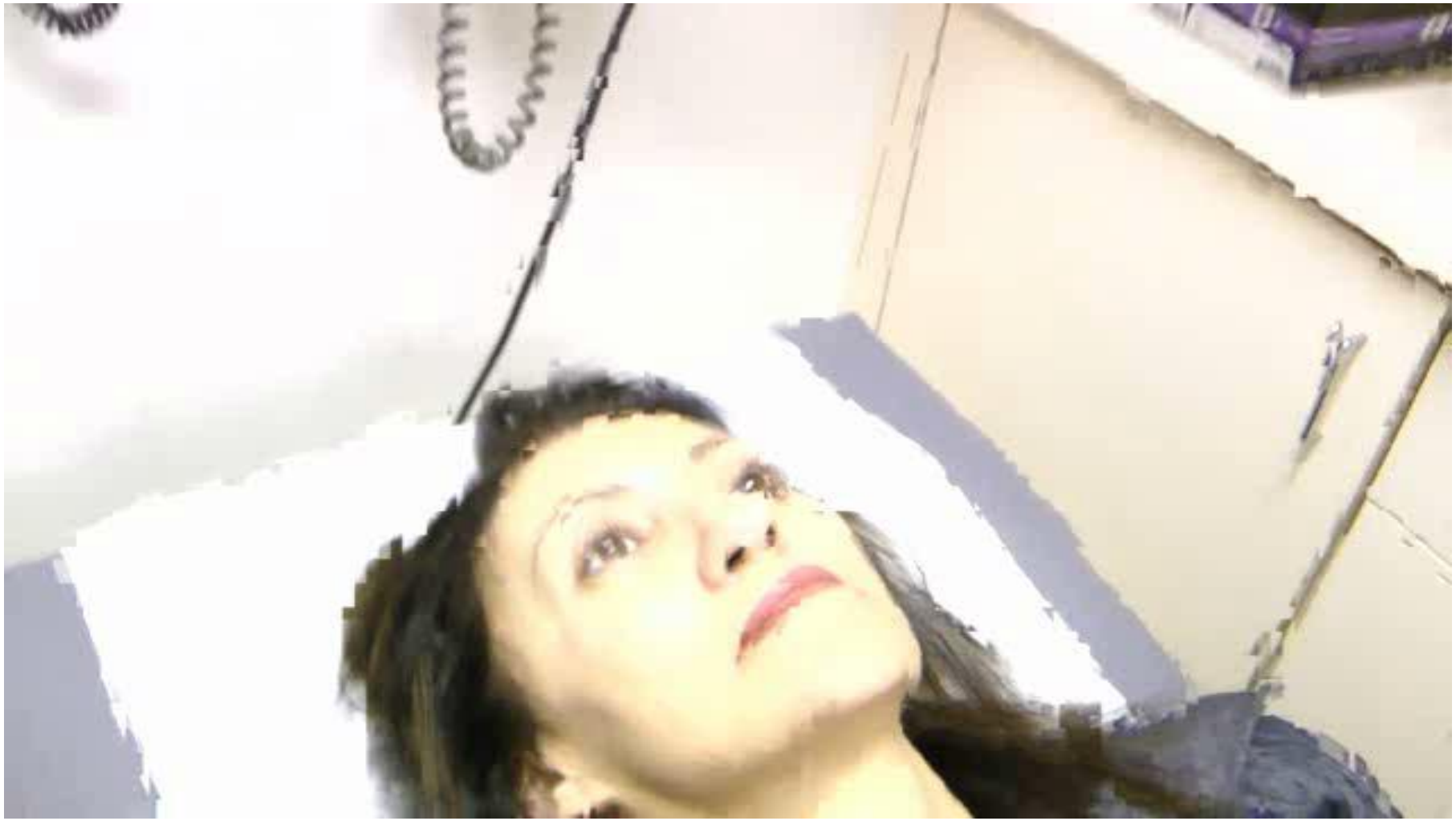
- **CLINICAL IMPLICATIONS: With LEFT BPPV, on gaze right the eyes move vertically and on gaze left torsionally. i.e., the globe rotates around a fixed axis orthogonal to the canal plane, i.e., in a plane parallel to the canal. Same principle applies to superior canal dehiscence(SCD)**

EPLEY RX right BPPV



Kraeling, Topics in Companion Animal Medicine, 2014

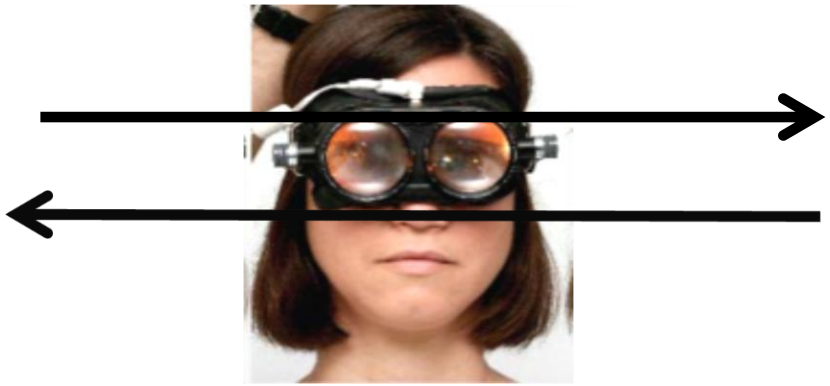
Lateral canal bppv: apogeotropic right ear affected
NOTE the effect of CONVERGENCE



Lateral canal bppv: apogeotropic right ear affected

NOTE: In lateral canal BPPV, when the patient is lying on the side in which the nystagmus is *most intense*, the nystagmus will be *beating toward the affected ear*, i.e., geotropic, with affected ear down and apogeotropic, with affected ear up

PATTERNS OF HEAD-SHAKING INDUCED NYSTAGMUS



Head-shaking induced nystagmus (HSN) in peripheral labyrinthine disease

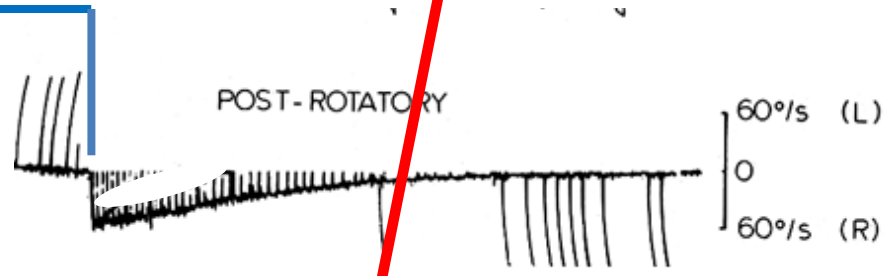
SOMETHING ‘NEW’ FOR THE ACUTE VERTIGO PATIENT

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

- **Normal with peripheral lesions**
- **Impaired <u>with central (nodulus) lesions</u>**

CHAIR

UPRIGHT



TILT

Normal Tilt supp

<u>NO Tilt supp</u>

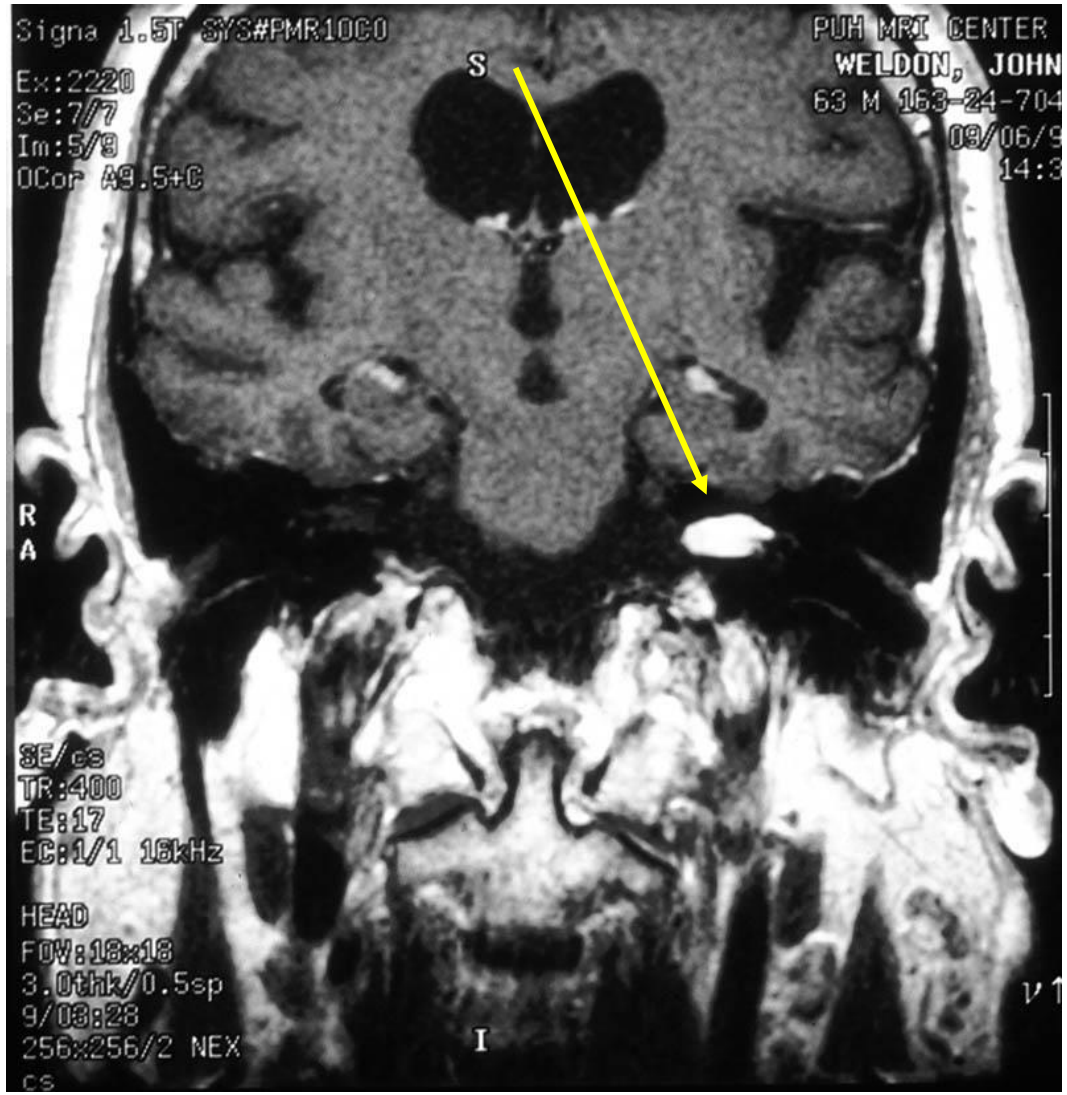
Zuma et al.

Hyperventilation

Patient with slightly asymmetrical hearing loss and mild imbalance

Hyperventilation

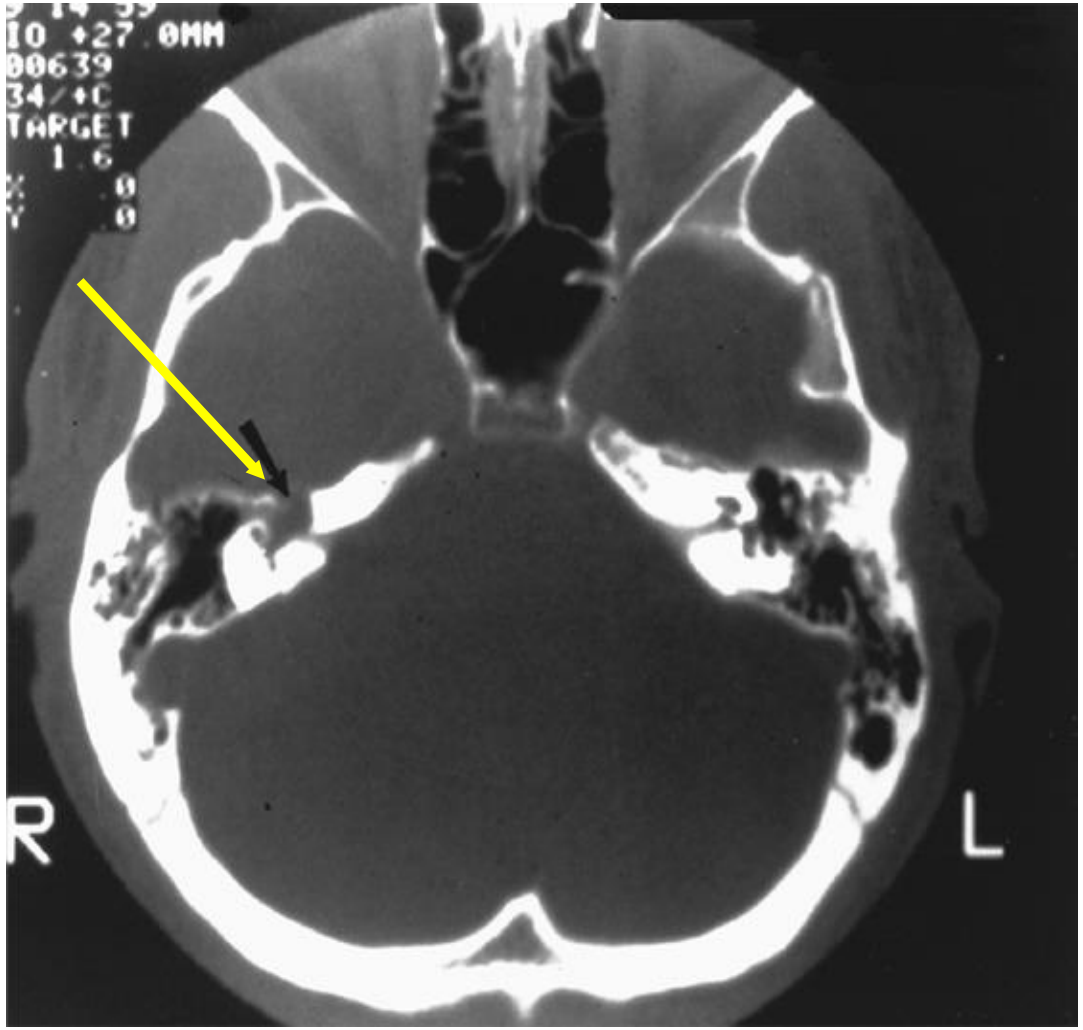
Hyperventilation-induced nystagmus



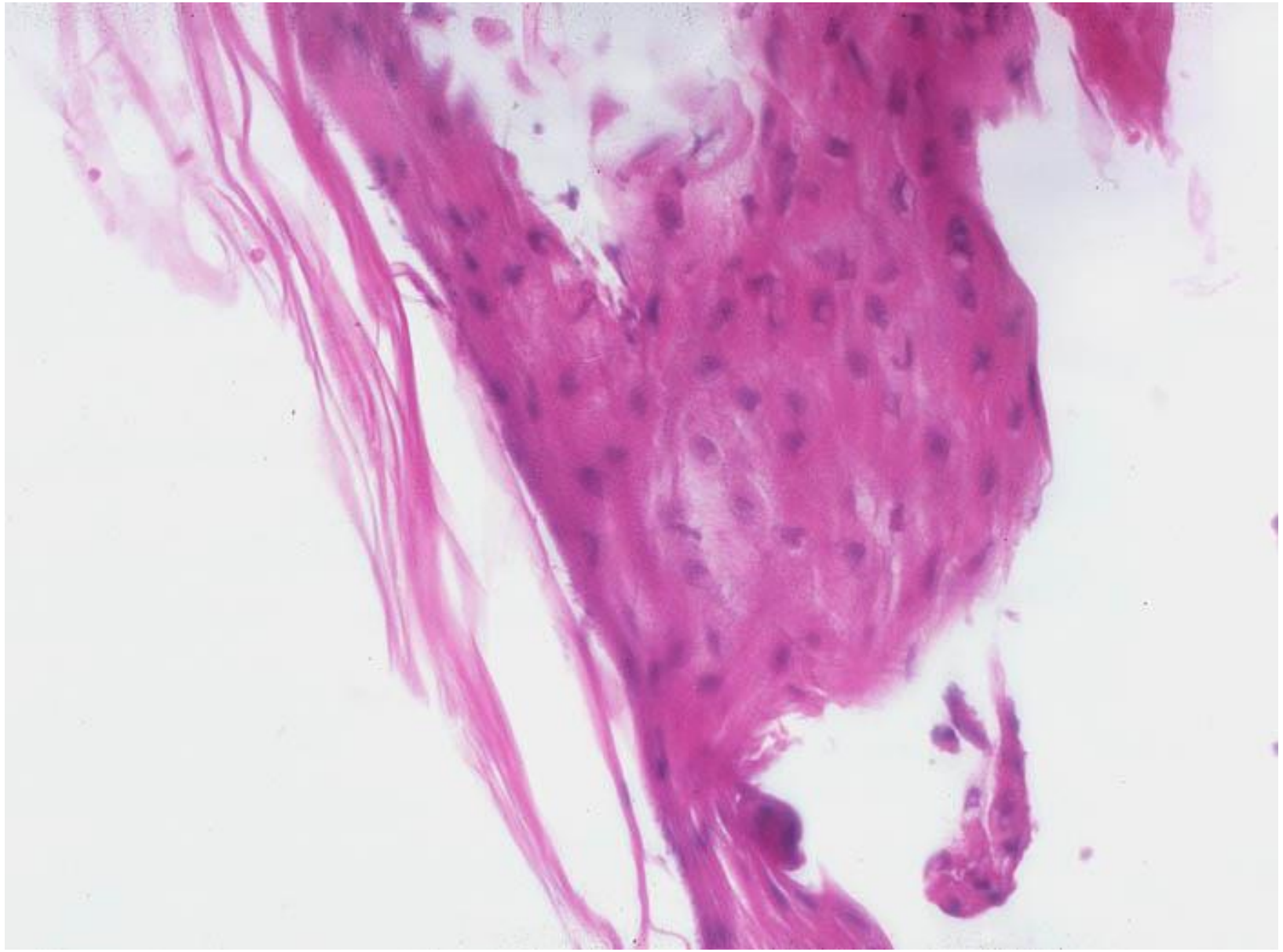
Vestibular Schwanoma

Patient with dizziness when playing lacrosse

Hyperventilation-induced nystagmus



Congenital Epidermoid

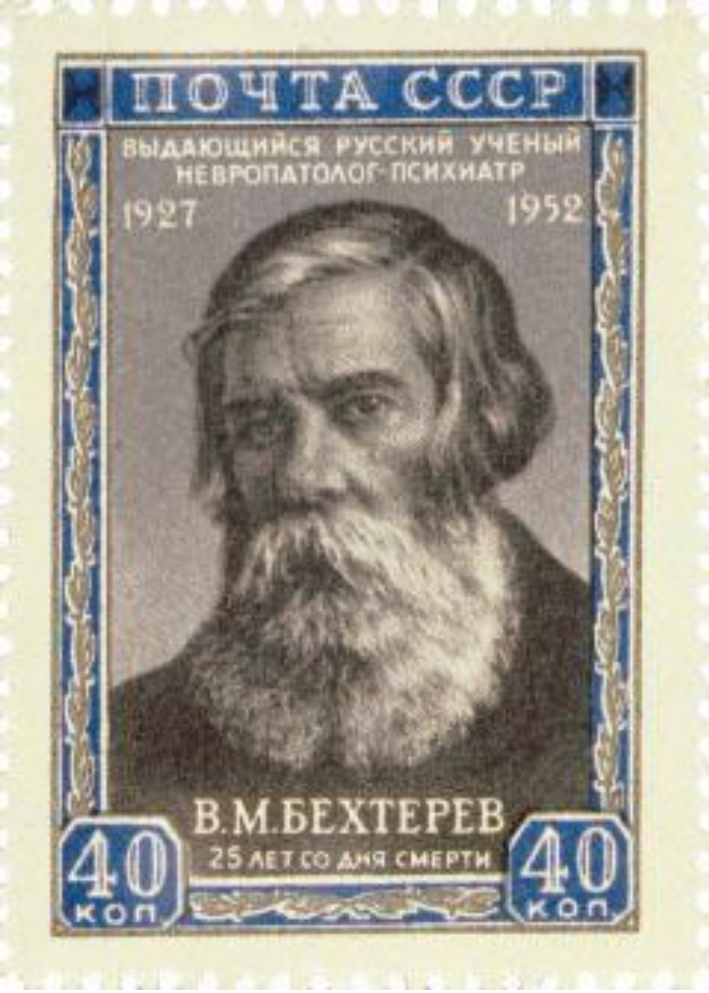


RECOVERY NYSTAGMUS

- **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**

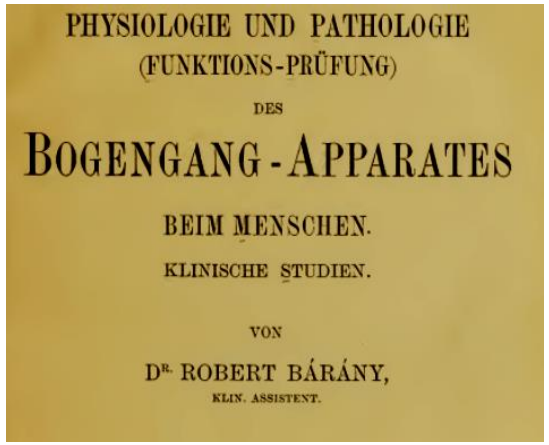
THINK ADAPTATION AT THE BEDSIDE!

Bedside vestibular testing

Bedside vestibular testing

Bedside vestibular testing

Kesselring


PHYSIOLOGIE UND PATHOLOGIE
(FUNKTIONS-PRÜFUNG)
DES
BOGENGANG-APPARATES
BEIM MENSCHEN.
KLINISCHE STUDIEN.
VON
DR. ROBERT BÁRÁNY,
KLIN. ASSISTENT.

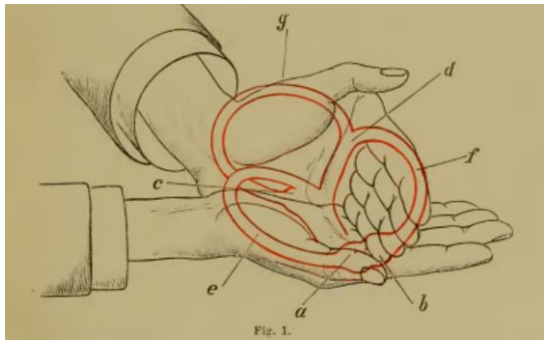


Fig. 1.

TAKE AWAY MESSAGE for the vestibular clinician

The KEY to successful diagnosis STILL requires

Focused *HISTORY* taking

A careful *BEDSIDE CLINICAL EXAMINATION*

Use of *ANATOMICAL AND PHYSIOLOGICAL PRINCIPLES* developed by the European masters of the 19th century

Alexander	Breuer	Hőgyes
Ewald	Bárány	Purkyně
Flourens	Bechterev	Mach

TAKE AWAY MESSAGE for the vestibular clinician

- **MUCH CAN BE LEARNED ABOUT HOW THE BRAIN WORKS**
- **AND HOW TO DIAGNOSE AND TREAT OUR PATIENTS**
- **AT THE BEDSIDE USING YOUR OWN EYES AND EARS!**