Complete and incomplete canalith jam atypical variants of BPPV and the role of the vHIT in the diagnosis

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AVISA
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VIS
Vestibology · Italian Society
Società Italiana di Vestibologia

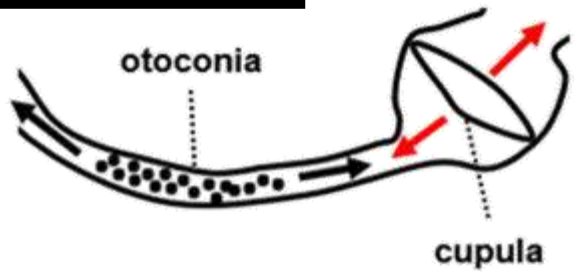
Acknowledgments

Pasquale Malara

Salvatore Martellucci

BPPV: possible scenarios

- **Canalolithiasis:** otoconia are free to float back and forth along the canal
  → **paroxysmal direction-changing nystagmus**

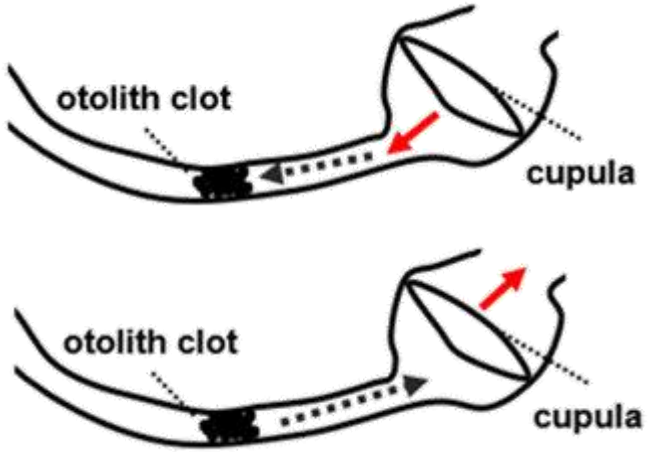


BPPV: possible scenarios

- **Cupulolithiasis:** otoconia adhere to the cupula overloading the cupula itself → **persistent direction-changing nystagmus with a neutral point**

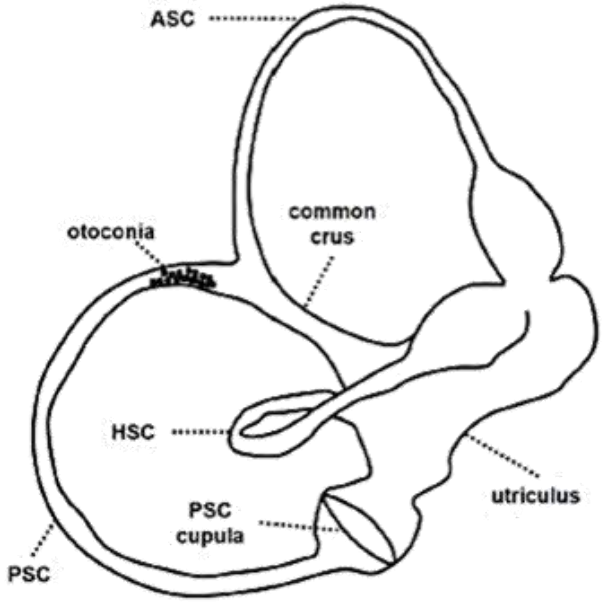
BPPV: possible scenarios

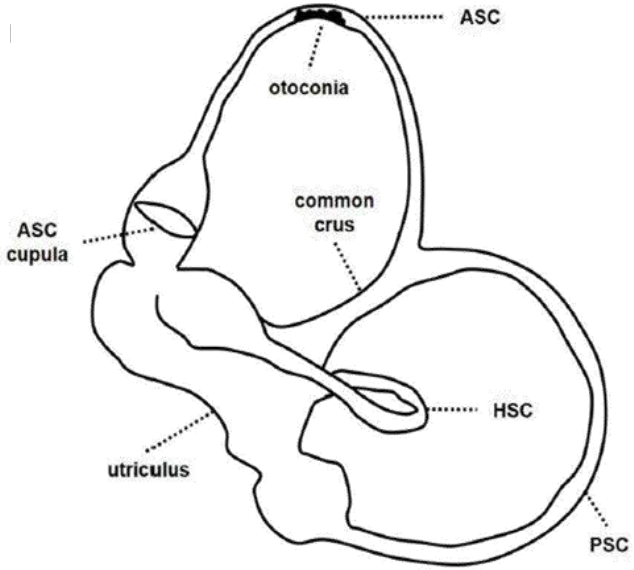
- **Canalith jam:**
  - **Complete:** an otolith clot entirely plugs the canal lumen → **spontaneous nystagmus that can be modulated by gravity**



BPPV: possible scenarios

- **Canalith jam:**
  - **Incomplete:** otoliths are partially entrapped in the canal lumen generating a positional jam → **persistent direction-fixed positional nystagmus**





BPPV: canalith jam

CAVEATS IN PARTICLE REPOSITIONING FOR TREATMENT OF CANALITHIASIS (BPPV)

JOHN M. EPLEY, MD

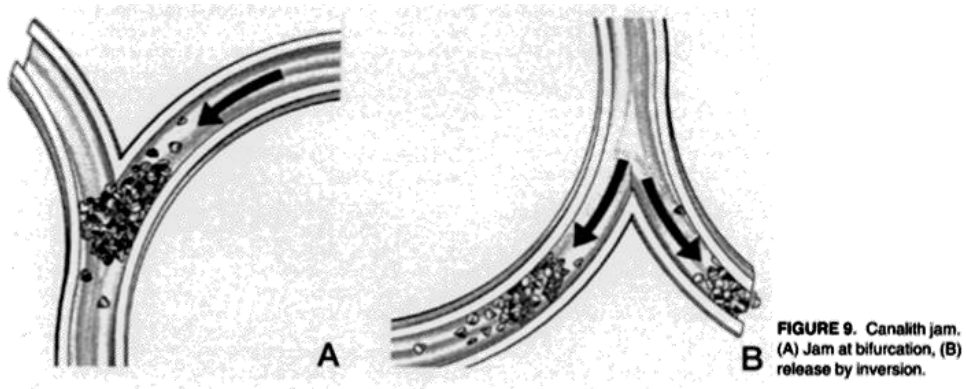


FIGURE 9. Canalith jam. (A) Jam at bifurcation, (B) release by inversion.

- **Pathomechanism:** otoliths migration **from a wider to a narrower segment of the canal**
  - at the outlet of the ampulla
  - at the **bifurcation of the common crus**
  - between the ampulla wall and a deflected cupula not allowing to return to its neutral position

- **It should be suspected** when **sudden conversion from a transient positional nystagmus to a persistent form** which is thereafter **unaffected by change of head position**

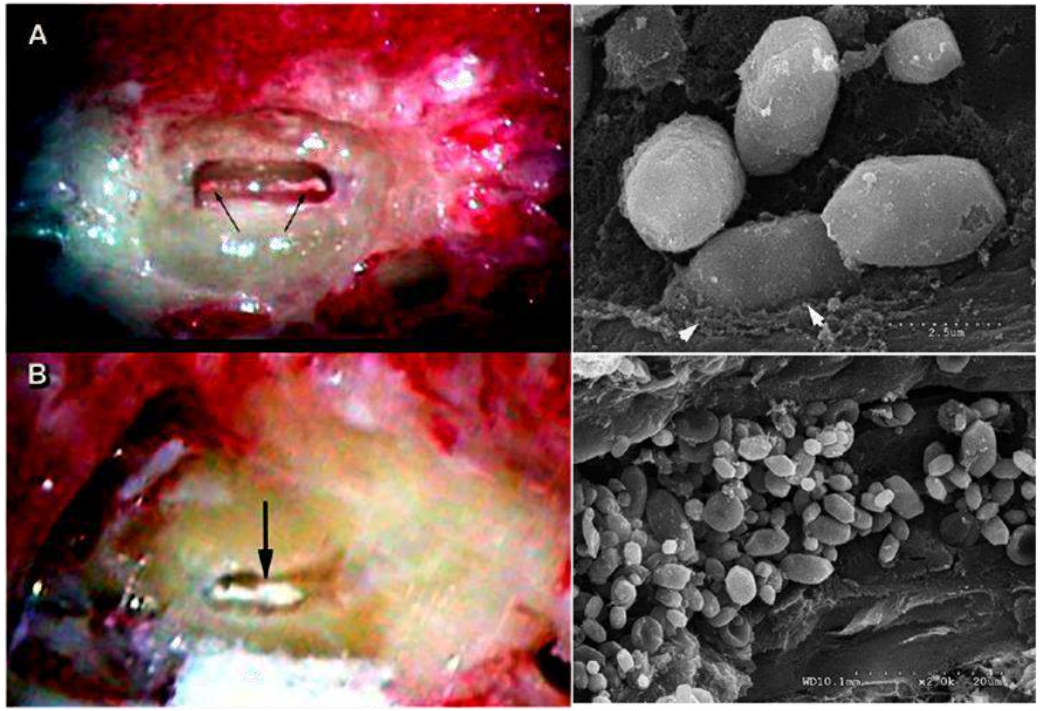
- It usually occurs as a **complication of CRM**, in the presence of 3 signs which suggest **many free particles**:
  - provocation of vertigo by **minimal change of position**
  - **severe positional nystagmus** of long duration (over 12 seconds)
  - involvement of **more than one canal**

BPPV: canalith jam

The Laryngoscope

Otoconia and Otolithic Membrane Fragments Within the Posterior Semicircular Canal in Benign Paroxysmal Positional Vertigo

Wee Tin K. Kao, MD; Lorne S. Parnes, MD; Richard A. Chole, MD, PhD



- Patients with **refractory BPPV** submitted to **surgical plug of the PSC**

o Scanning electron microscope**: otoconia and otolithic membrane fragments partially embedded in a gel matrix** rather than free-floating otoconia

o **Complete** or **incomplete occlusion** of the canal due to otoconial matter **does not seem to be so unlikely**

BPPV: canalith jam

ORL

Original Paper

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High-Resolution Three-Dimensional Magnetic Resonance Imaging of the Vestibular Labyrinth in Patients with Atypical and Intractable Benign Positional Vertigo

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Wolfgang Arnold[a]

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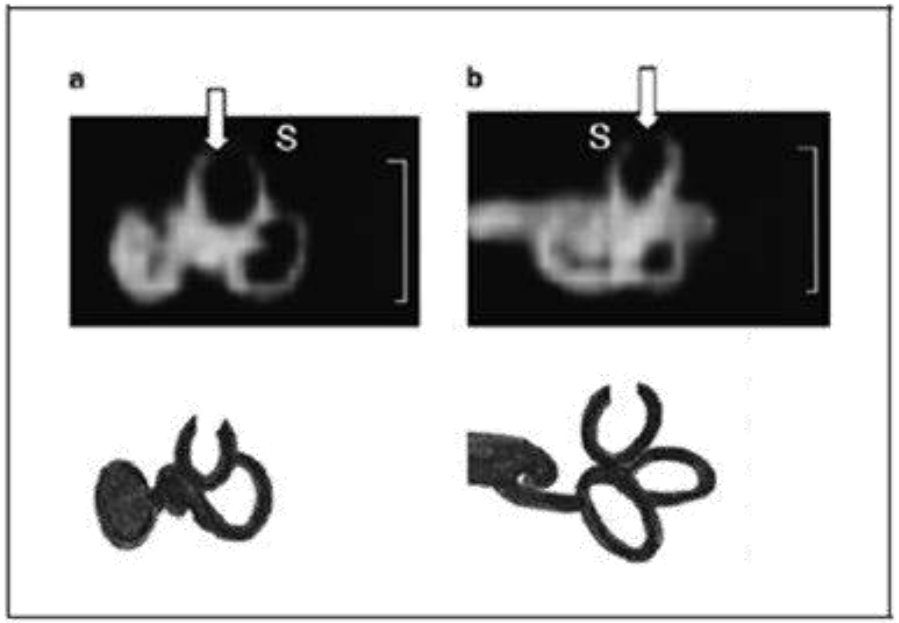
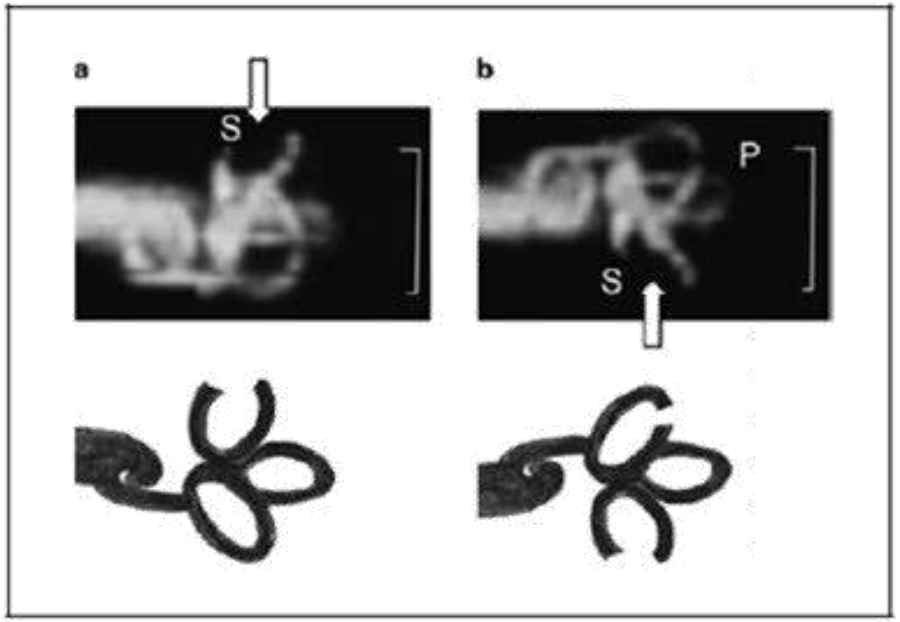
- **«Filling defects»** within semicircular canals on 3D HR MRI in patients with **atypical or refractory BPPV**





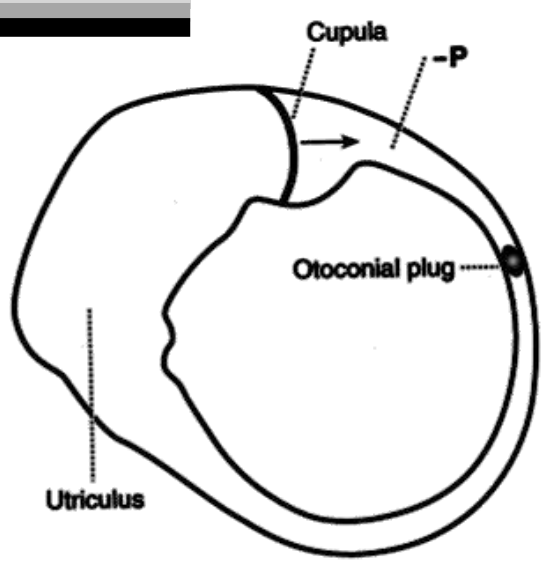
BPPV: canalith jam

Continuous vertigo and spontaneous nystagmus due to canalolithiasis of the horizontal canal

Article abstract—The authors present a patient with benign paroxysmal positional vertigo of the right horizontal semicircular canal who developed persistent vertigo with spontaneous horizontal nystagmus to the left and caloric hypoexcitability on the right after a head shaking maneuver. Both spontaneous nystagmus and canal paresis resolved after repeated shaking of the head. The most probable mechanism of this type of vertigo is plugging of the horizontal canal by otoconial particles with a negative endolymph pressure between plug and cupula.

NEUROLOGY 2001;56:684–686

Michael von Brevern, MD; Andrew H. Clarke, PhD; and Thomas Lempert, MD

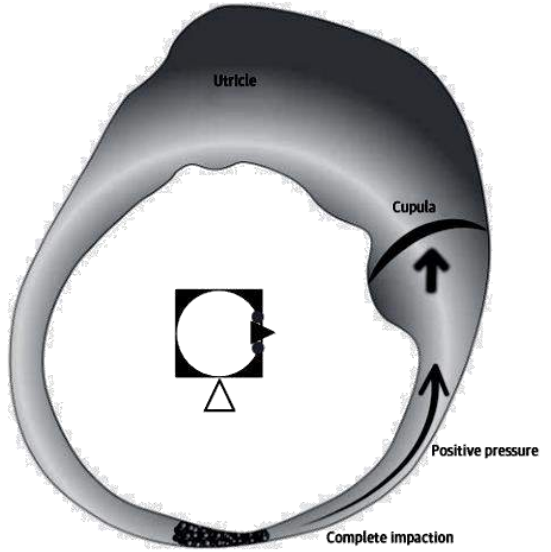


- 1° case of **HSC jam complicating CRM** for HSC-BPPV
- **"Paretic" spontaneous nystagmus** due to a **continuous negative ampullofugal pressure** exerted by the otoliths clot
- **positive bedside horizontal HIT** and **reversible after CRM**

Case Report/Case Series

Persistent Spontaneous Nystagmus Following a Canalith Repositioning Procedure in Horizontal Semicircular Canal Benign Paroxysmal Positional Vertigo

Kyung Min Ko, MD; Mee Hyun Song, MD, PhD; Ji Hong Kim, MD; Dae Bo Shim, MD



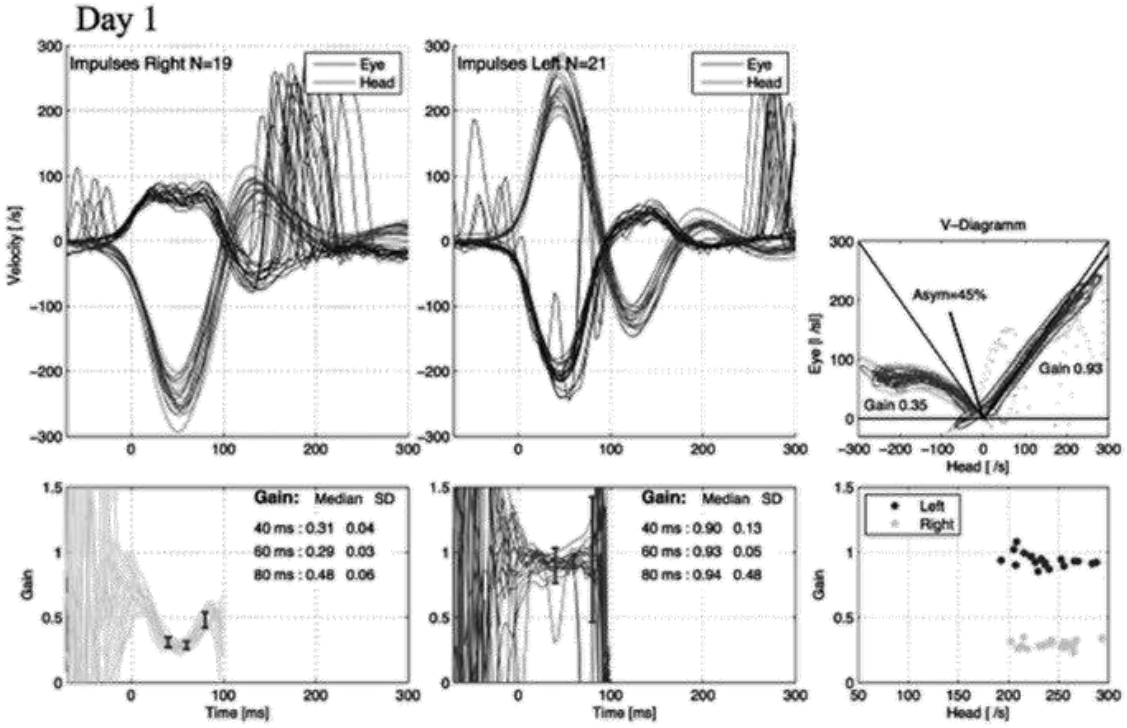
- Another case of **HSC jam complicating wrong CRM** for geotropic HSC-BPPV
- **«Irritative» spontaneous nystagmus** due to **continuous positive ampullopetal pressure** exerted by the otolith clot
- **Normal HIT**
- **Reversible CP after CRM**

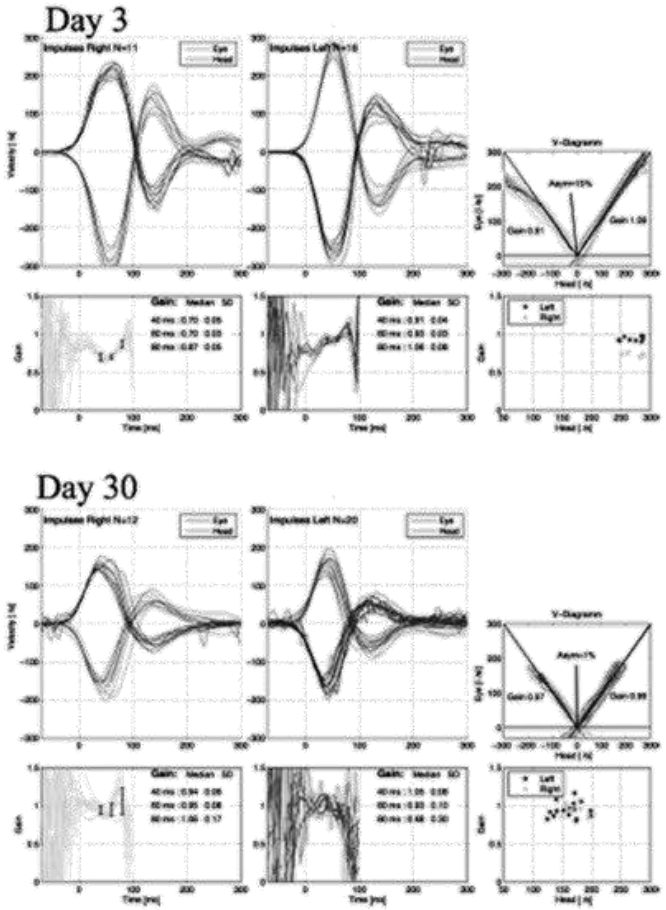
BPPV: canalith jam

Spontaneous Plugging of the Horizontal Semicircular Canal With Reversible Canal Dysfunction and Recovery of Vestibular Evoked Myogenic Potentials

*†‡Leonel Luis, ‡João Costa, §Fernando Vaz Garcia, ||Josep Valls-Solé, ¶Thomas Brandt, and ¶#Erich Schneider

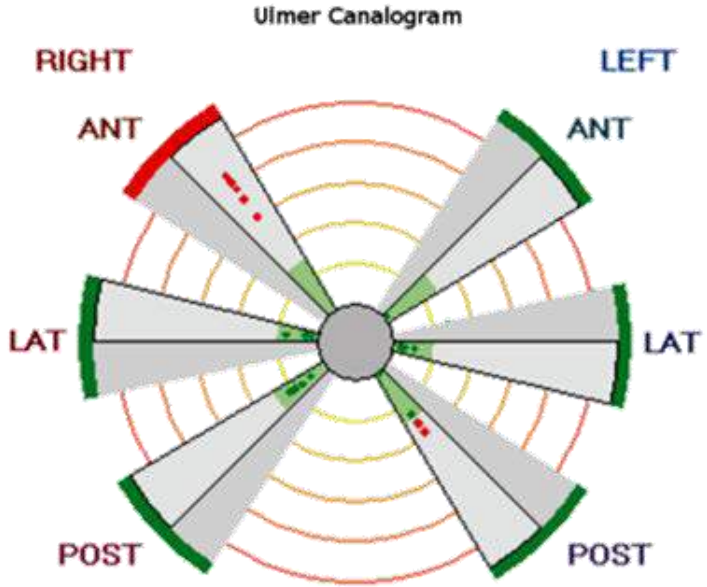
- 1° case of **HSC jam** were the **diagnosis was based on the vHIT**
- vHIT exhibited a **selective high-frequency VOR impairment for the affected canal reversible after CRM**

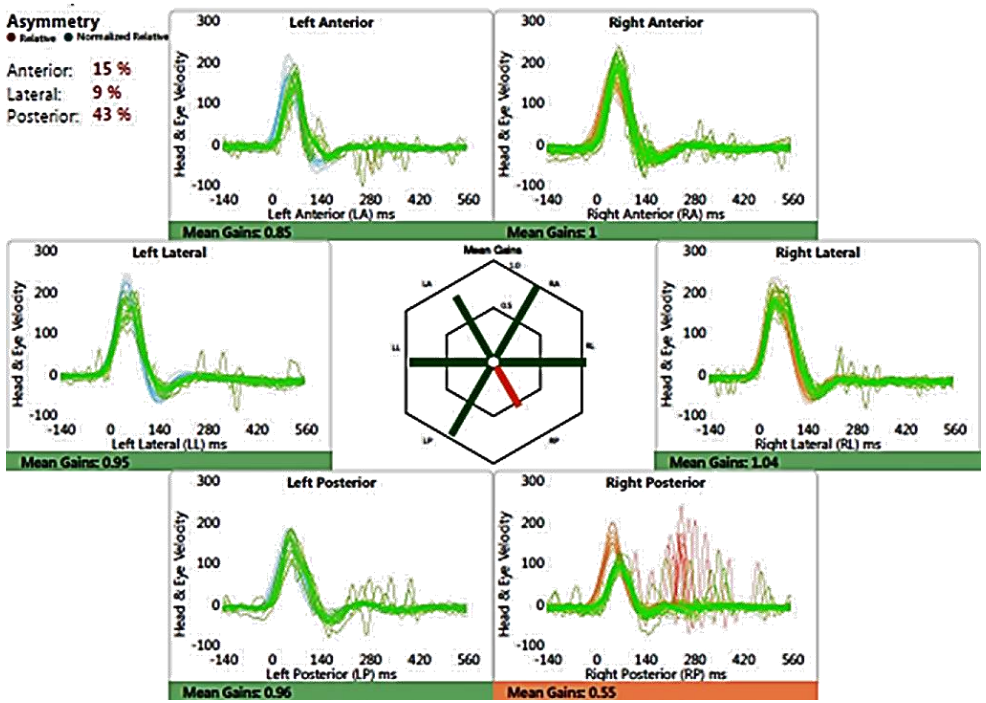


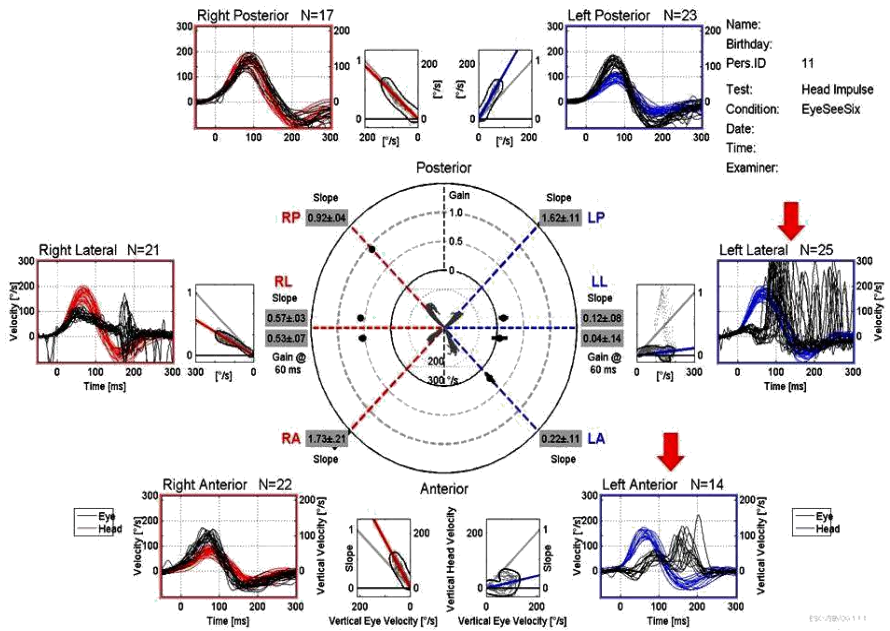


video HIT

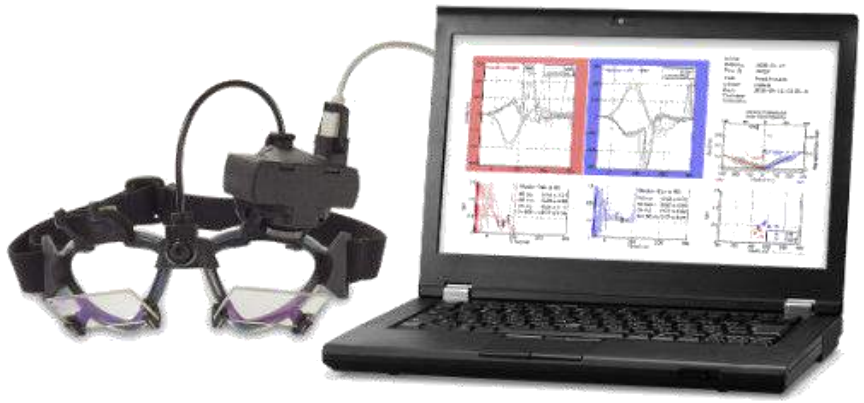
- **High frequency sampling camera** (**100 - 250 frames/sec**) **with sensitivity overlapping Scleral Search Coil** (gold standard)
- Precise measure of the **VOR-gain** (eye/head velocity ratio) **of each of the 6 canals**
- **Target:** (**Type I hair cells**, **phasic irregular afferents**)







video HIT



- If the **“canalith jam”** is **assumed to alter endolymphatic flows leading to persistent deflections of the cupula of the affected canal**
- If **vHIT** can precisely measure **high-frequency responses of ampullary afferents for each single canal**

↓

- It could **potentially provide useful clues to detect the involved canal in case of canalith jam**
- **In case of atypical spontaneous/positional nystagmus,** it could probably contribute in the **precise distinction among peripheral and central disorders**

BPPV: canalith jam

Spontaneous Canalith Jam and Apogeotropic Horizontal Canal Benign Paroxysmal Positional Vertigo: Considerations on a Particular Case Mimicking an Acute Vestibular Deficit

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Spontaneous Plugging of the Horizontal Semicircular Canal With Reversible Canal Dysfunction and Recovery of Vestibular Evoked Myogenic Potentials

*†‡Leonel Luis, ‡João Costa, §Fernando Vaz Garcia, ||Josep Valls-Solé, ¶Thomas Brandt, and ¶#Erich Schneider

Isolated horizontal canal hypofunction differentiating a canalith jam from an acute peripheral vestibular loss

Andrea Castellucci[a], Pasquale Malara[b], Cristina Brandolini[c], Valeria Del Vecchio[c], Davide Giordano[a], Angelo Ghidini[a], Gian Gaetano Ferri[c,*], Antonio Pirodda[c]

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Horizontal semicircular canal jam: Two new cases and possible mechanisms

Michael C. Schubert[1,2] | Janet Helminski[3] | David S. Zee[1,4,5,6] | Elisabetta Cristiano[7] | Antonio Giannone[7] | Giuseppe Tortoriello[8] | Vincenzo Marcelli[8]

Spontaneous Jamming of Horizontal Semicircular Canal Combined with Canalolithiasis of Contralateral Posterior Semicircular Canal

Salvatore Martellucci[1], Andrea Castellucci[2], Pasquale Malara[3], Giulio Pagliuca[1], Veronica Clemenzi[1,4], Andrea Stolfa[1,4], Andrea Gallo[1,4], and Giacinto Asprella Libonati[5]

HSC canalith jam

- **CASE 1**

o 74-year-old woman

o Abrupt onset of **acute vertigo** with nausea and vomiting since one day prior to presentation

o **Neither auditory symptoms** nor previous vestibular symptoms

o **Brain CT scan**: negative

o **Neurological examination:** unremarkable

o **Bedside HIT: positive on the right side** with re-fixation saccades

→ Diagnosis of right AVL

Isolated horizontal canal hypofunction differentiating a canalith jam from an acute peripheral vestibular loss

Andrea Castellucci[a], Pasquale Malara[b], Cristina Brandolini[c], Valeria Del Vecchio[c], Davide Giordano[a], Angelo Ghidini[a], Gian Gaetano Ferri[c,*], Antonio Pirodda[c]

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[b] *Audiology & Vestibology Service, Centromedico, Viale Officina 7, 6500 Bellinzona, Switzerland*
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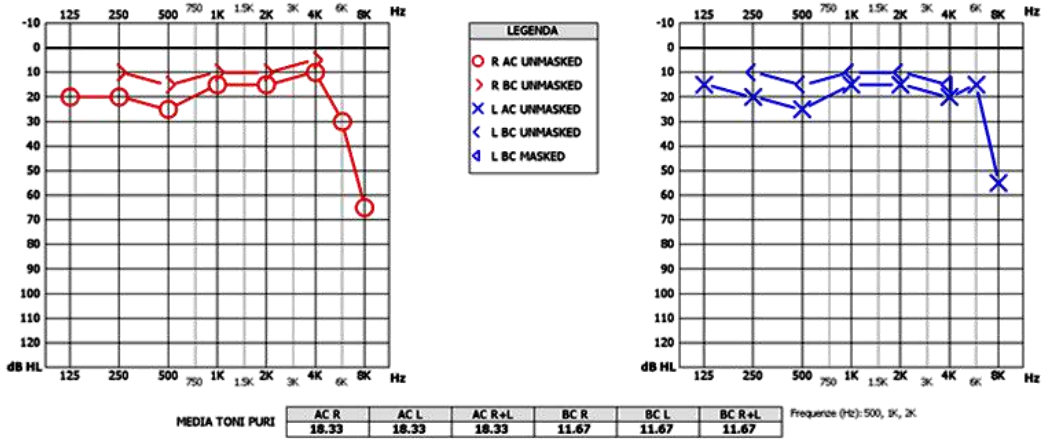
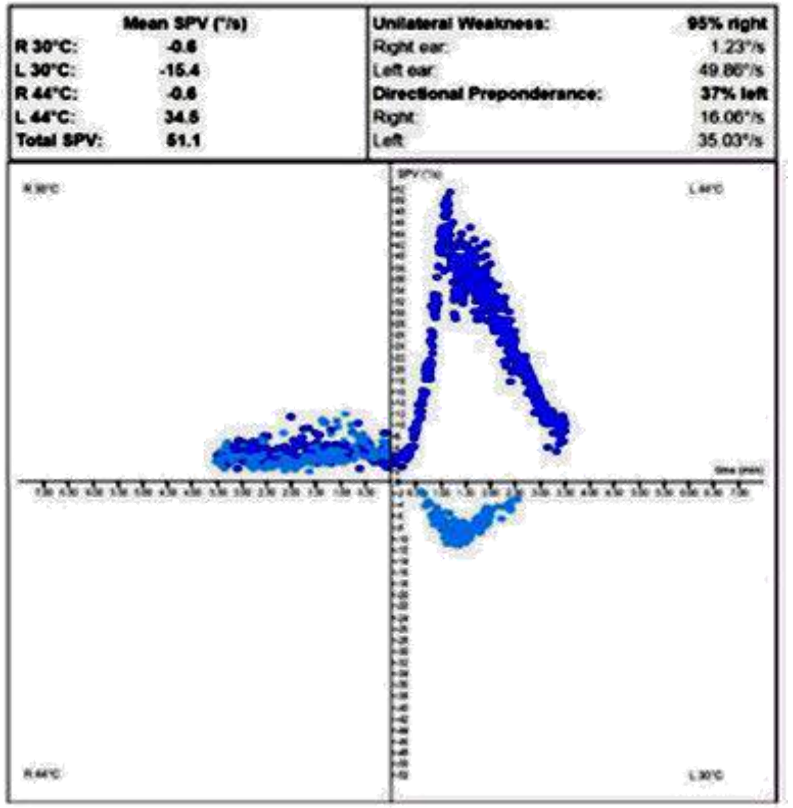
HSC canalith jam

- **INSTRUMENTAL WORK-UP IN ACUTE STAGE**

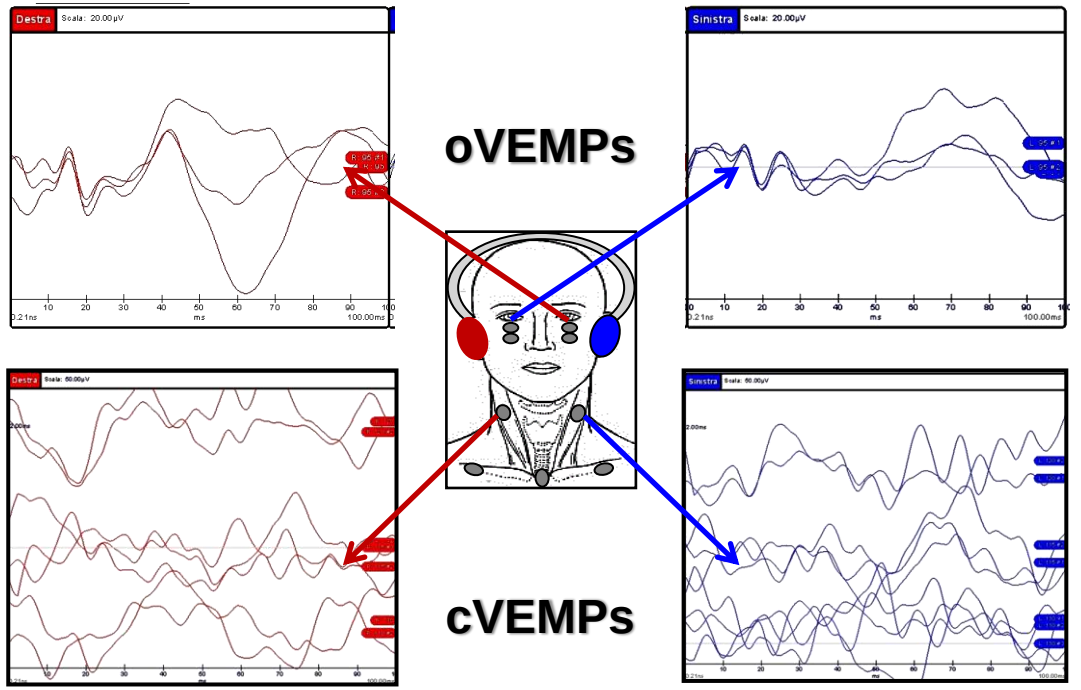
o **Pure Tone Audiometry:** mild and symmetrical HF-SNHL **consistent with the patient's age**





o **cVEMPs: symmetrical** amplitudes (AR 10%, R = L)

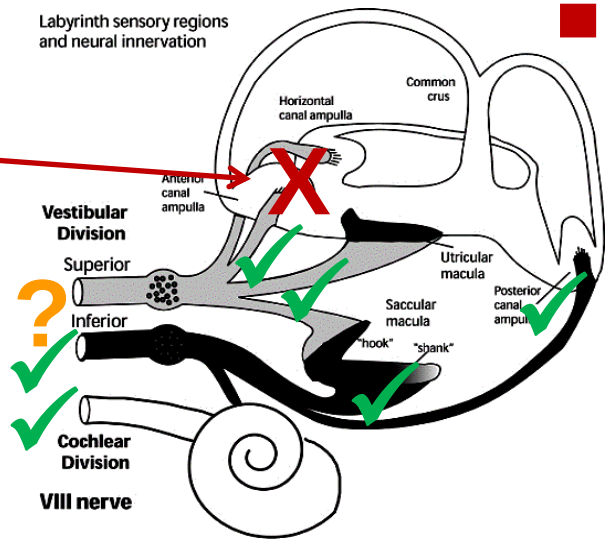
o **oVEMPs: symmetrical** amplitudes (AR 12%, R = L)

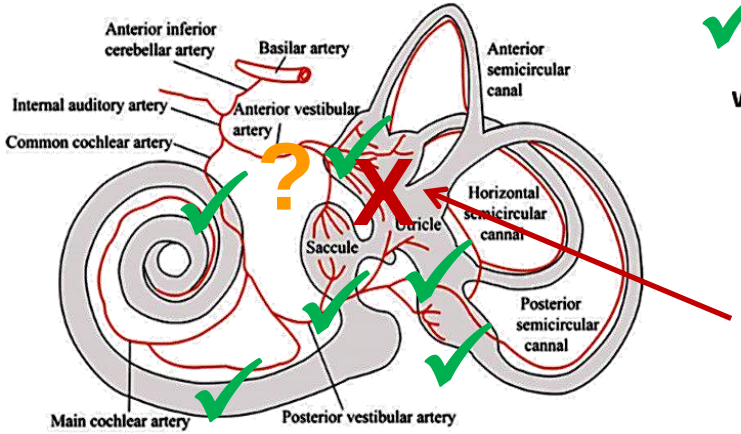


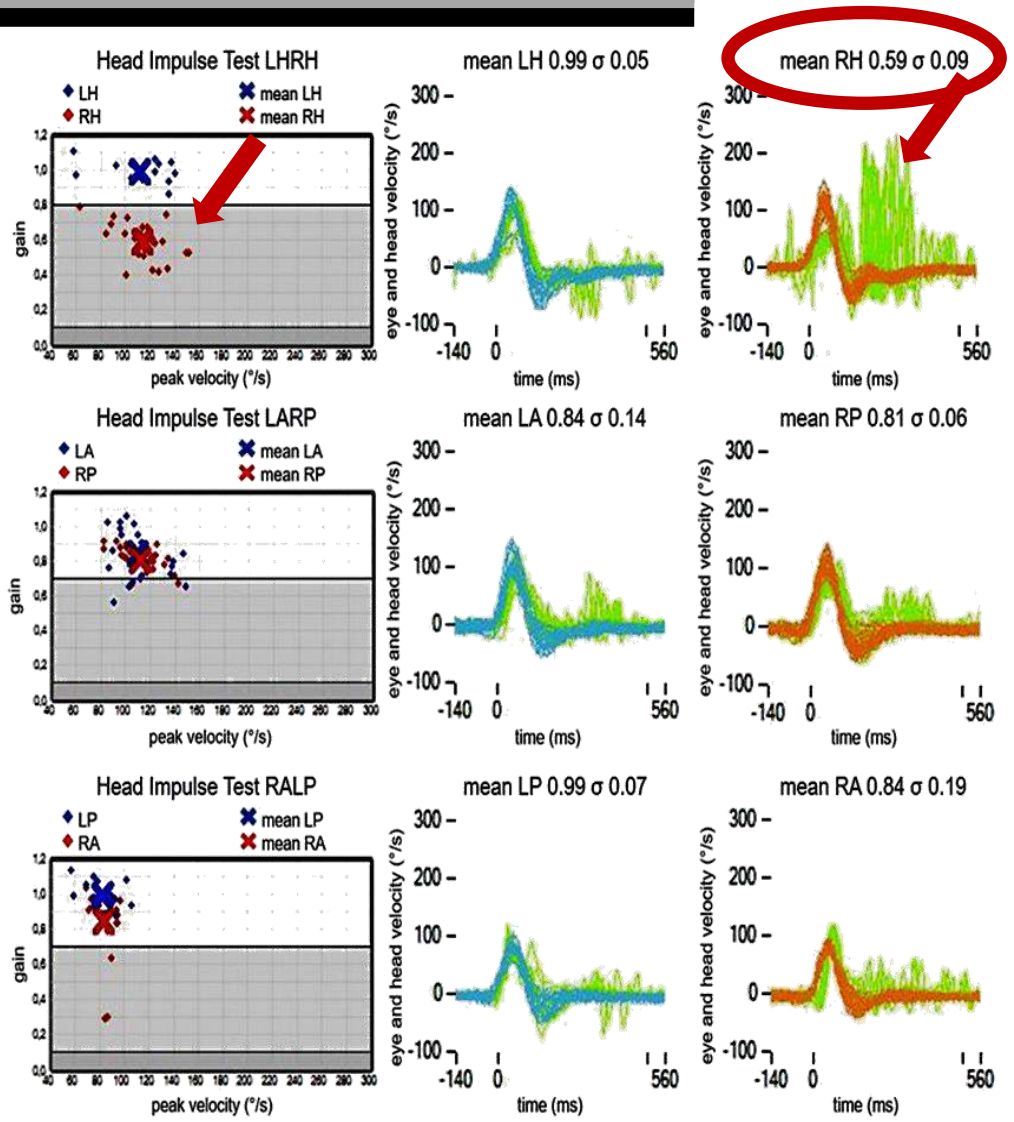
o **Caloric Test: severe right canal paresis** (UW 95%)

HSC canalith jam

- **INSTRUMENTAL WORK-UP IN ACUTE STAGE**
  - **Video HIT: Selective right HSC hypofunction** (gain 0.59)
- **HYPOTHETICAL PATHOMECHANISM**
  - **Selective neuritis** involving right HSC ampullary nerve?
  - **Selective ischemia** of the arterial terminal branch supplying right HSC?





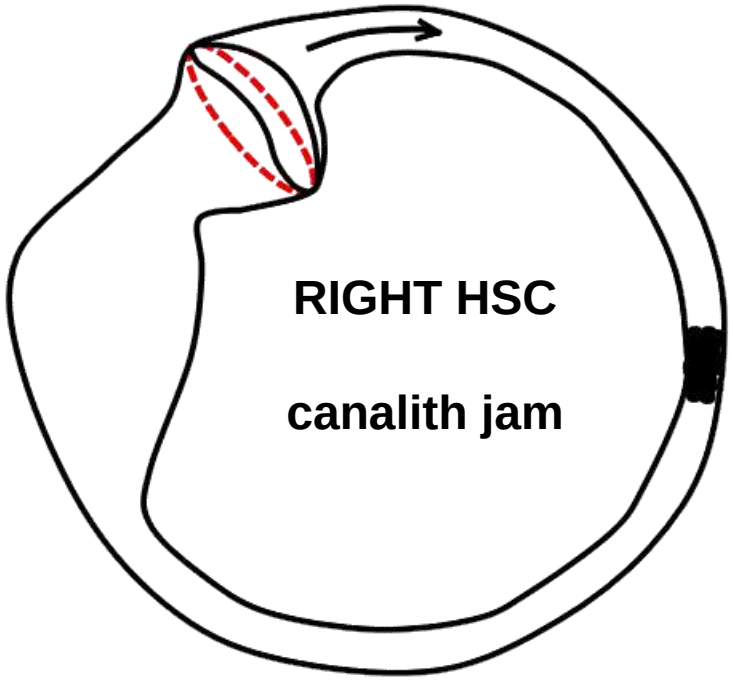


HSC canalith jam

- **HYPOTHETICAL PATHOMECHANISM**

  - **Right HSC pseudo-hyporeflexia** due to a **spontaneous canalith jam** inducing a **selective plug of the canal lumen**

  - An **otoliths clot** entrapped within the membranous duct could likely exert a **continuous negative pressure** on the cupula, leading to a **persistent ampullofugal deflection of the cupula**, **preventing dynamic responses of the right HSC** by blocking endolymphatic flow.



- This condition could likely result in:

  - **Paretic spontaneous nystagmus** regardless of head position
  - **HSC VOR gain impairment** for both **low- (caloric) and high-frequency (video HIT)** domains

HSC canalith jam

- **Video-Frenzel examination (2 days later)**

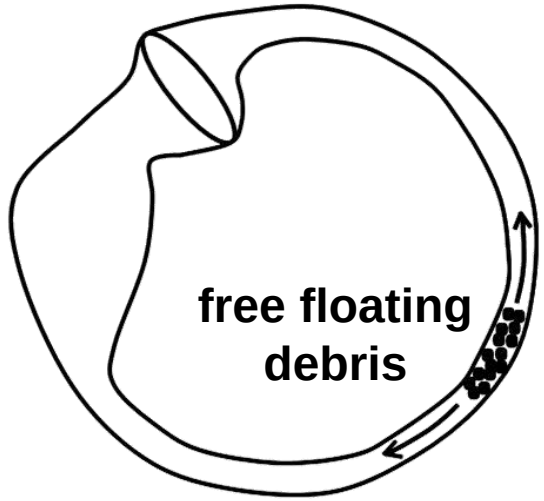
o After repeated head shakings, the supine roll test elicited bilaterally a **positional paroxysmal direction-changing geotropic horizontal nystagmus (R > L)** suggesting a **BPPV involving the non-ampullary arm of the right HSC**

o Otoconia likely **fell in the non-ampullary arm of the right HSC**

- **TRATMENT AND FOLLOW-UP**

o The patient was then submitted to **CRM for HSC-BPPV** (Gufoni) and was instructed to lay with the head on the healthy side for two days

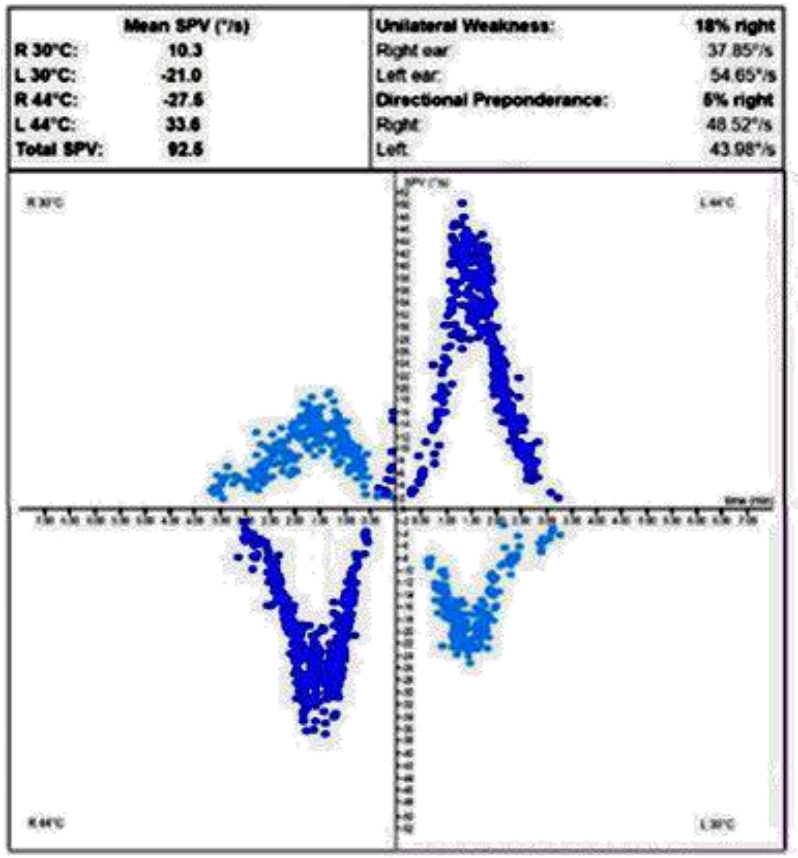
o When the patient returned 2 days later, **her symptoms had subsided**



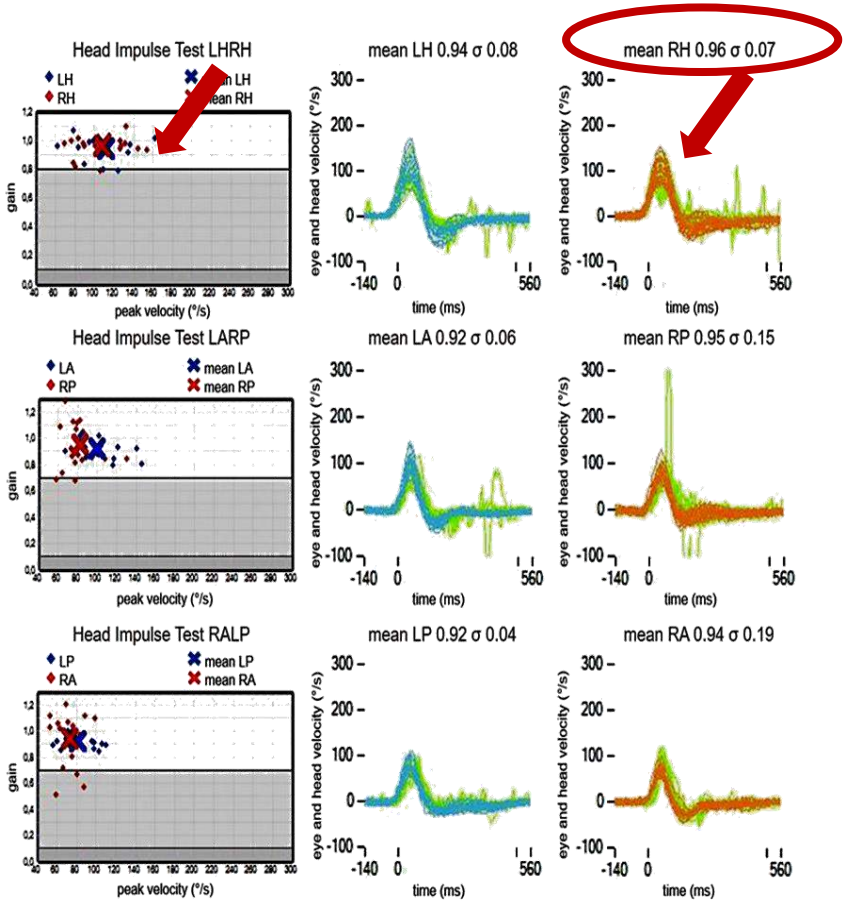
HSC canalith jam

- **INSTRUMENTAL WORK-UP AFTER CRM (2 days later)**

o **Caloric test: complete recovery of the right canal paresis** (UW 18%)



o **Video HIT: functional recovery of the right HSC** (gain 0.96)

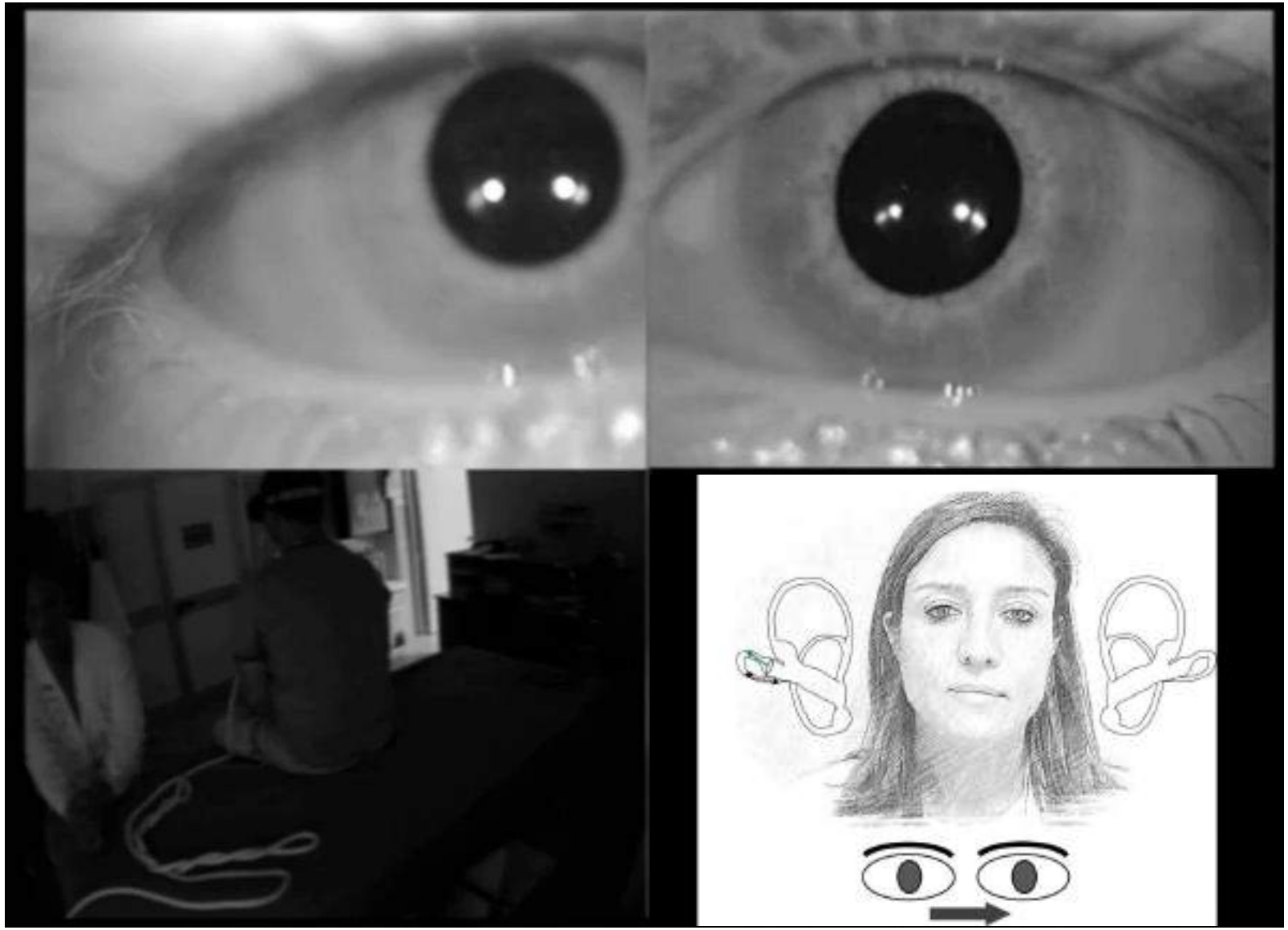


HSC canalith jam

- **CASE 2**

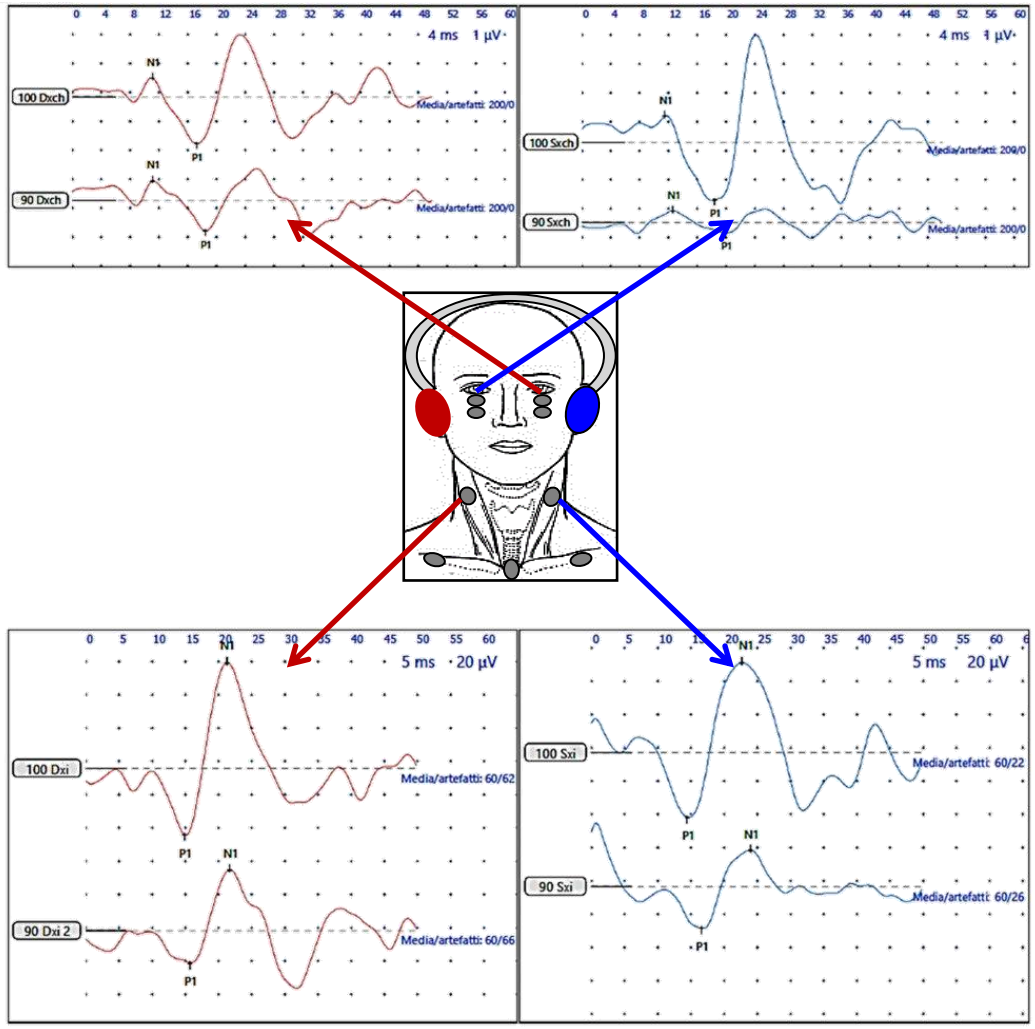
- 49-year-old man
- Onset of **acute vertigo** in the morning
- Recent history consistent with positional vertio
- **Neither auditory symptoms** nor previous vestibular symptoms
- **Brain CT scan**: negative
- **Neurological examination:** unremarkable

- **h-Bedside HIT: positive on the right** with re-fixation saccades



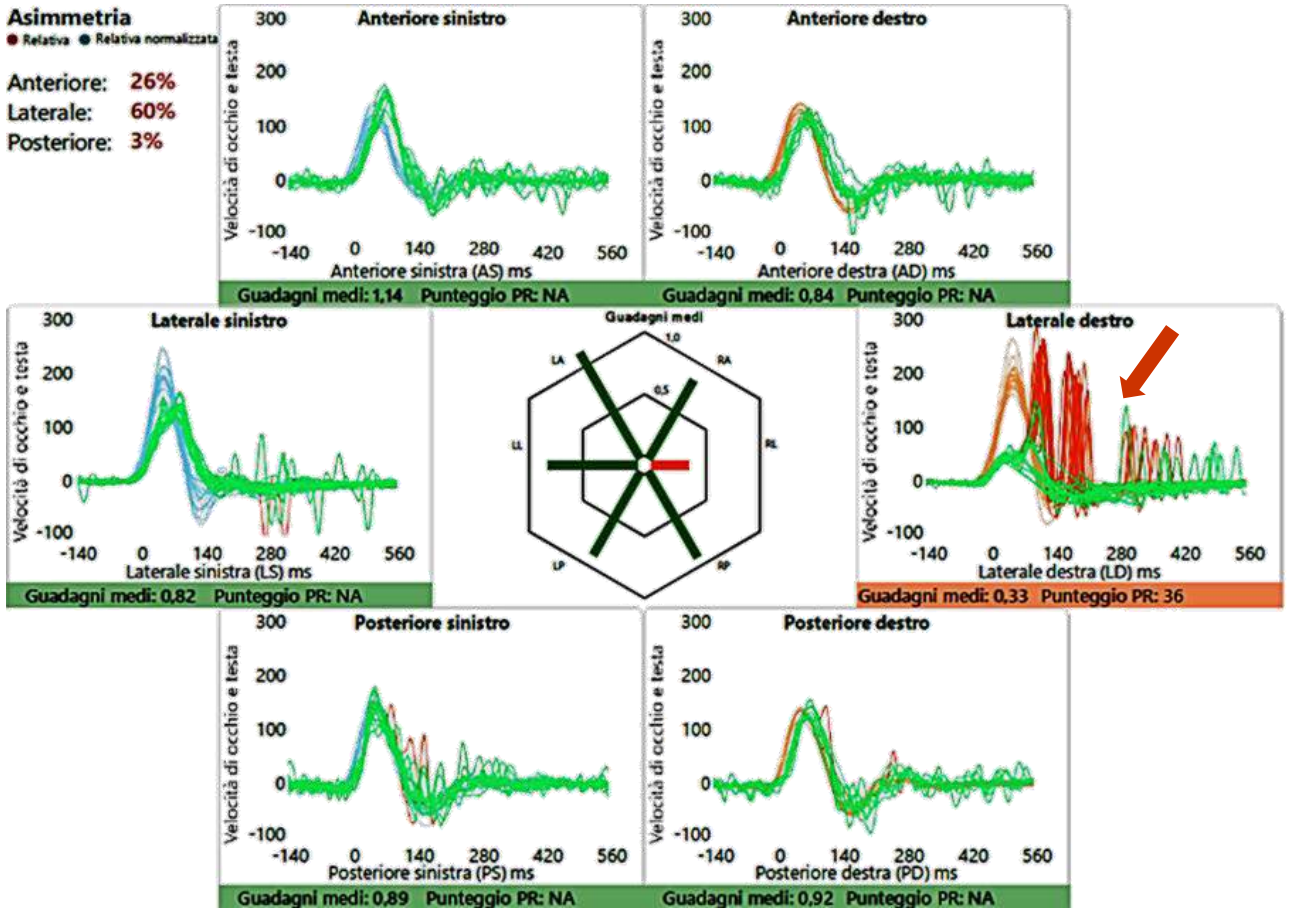
HSC canalith jam

oVEMPs



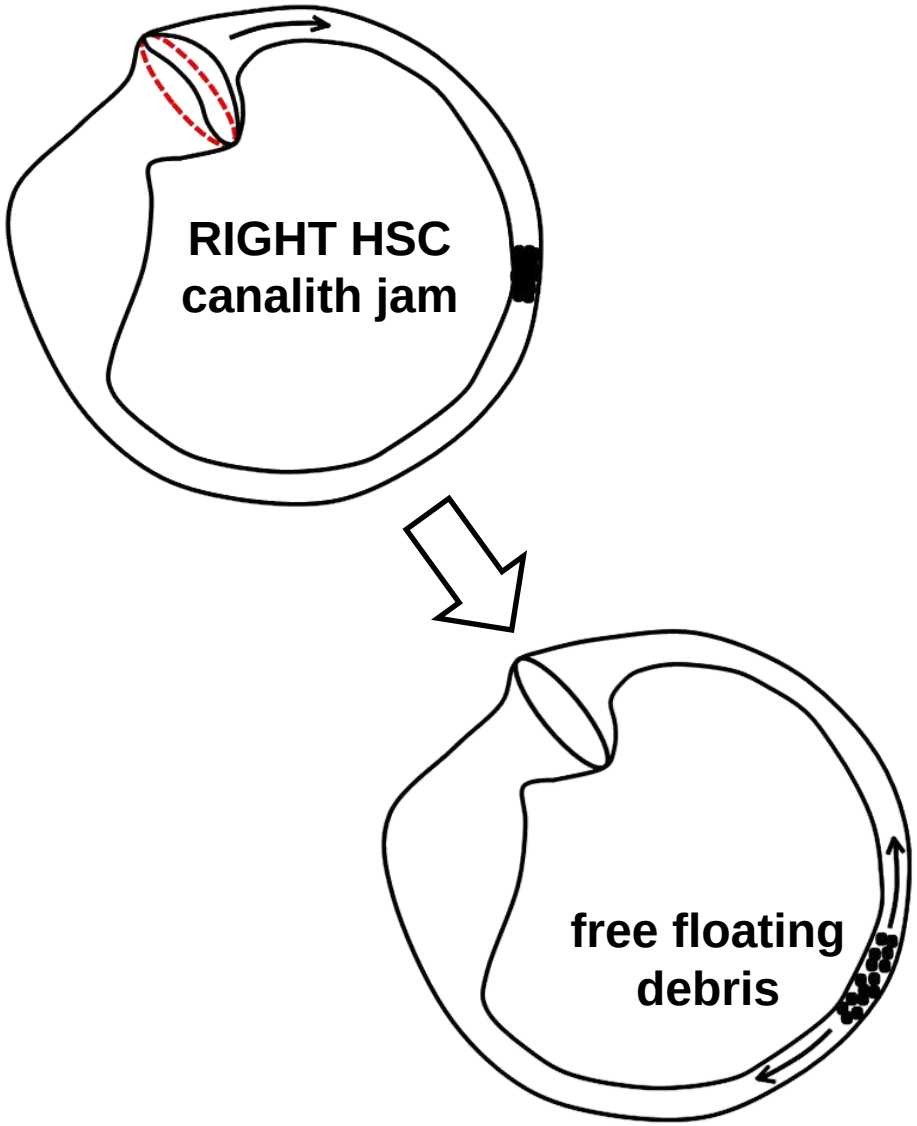
cVEMPs

Video HIT: Selective right HSC hypofunction (gain 0.33)

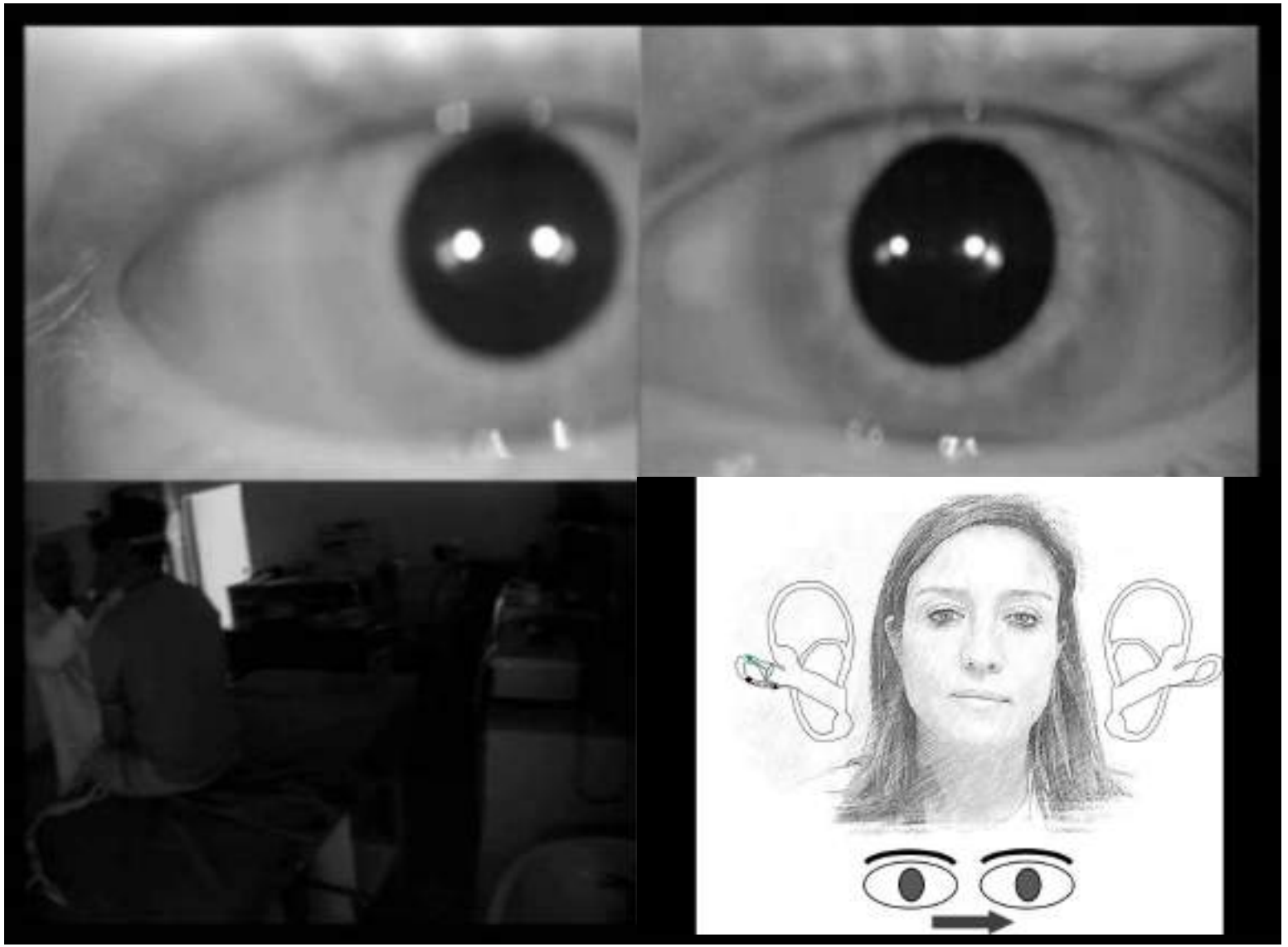


HSC canalith jam

- **HYPOTHETICAL PATHOMECHANISM**



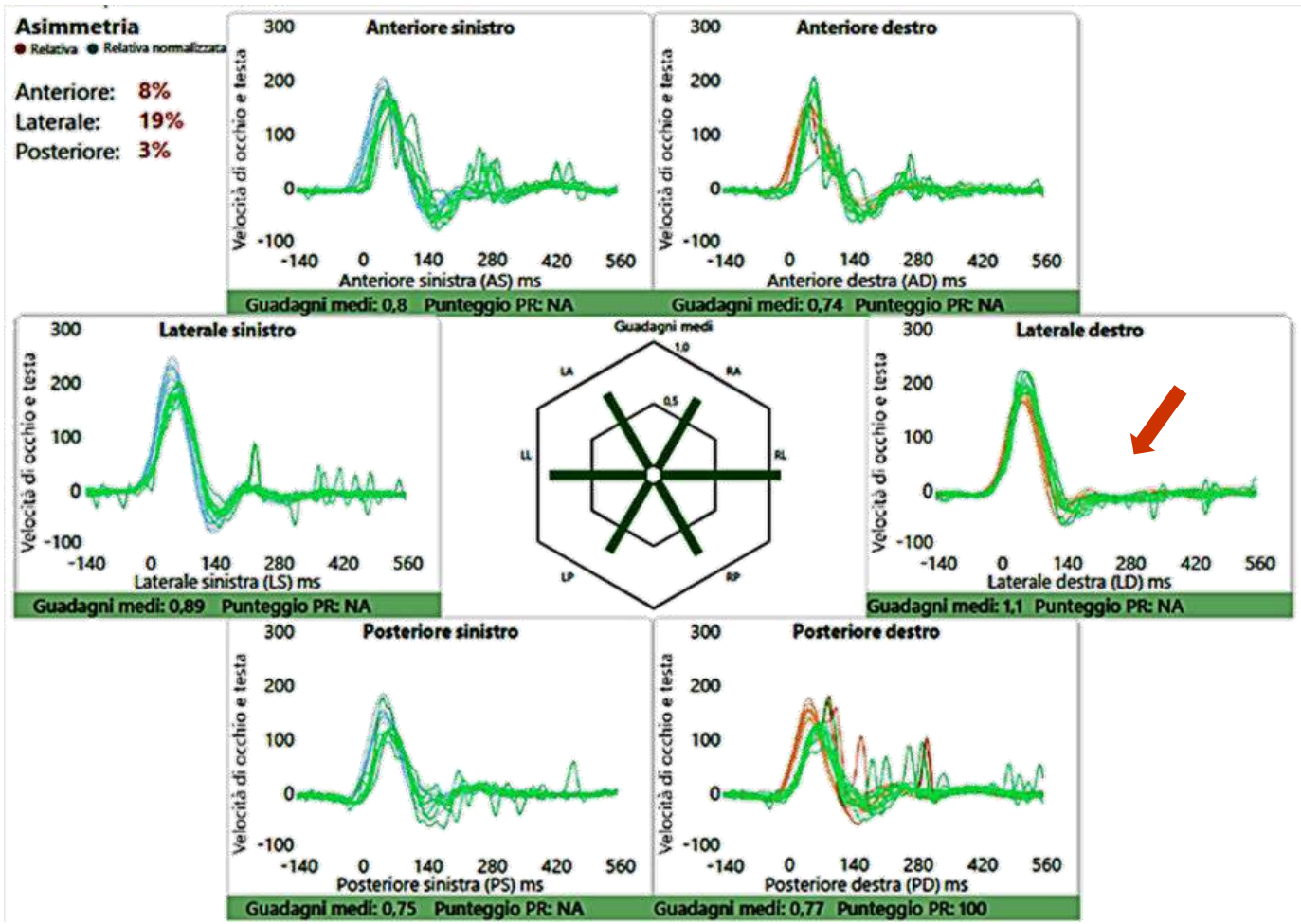
- **Video-Frenzel examination** (after head shakings)



HSC canalith jam

- **INSTRUMENTAL WORK-UP FOLLOWING CRM for HSC-BPPV**

o **Video HIT: normalization of right HSC VOR-gain** (gain 1.1)

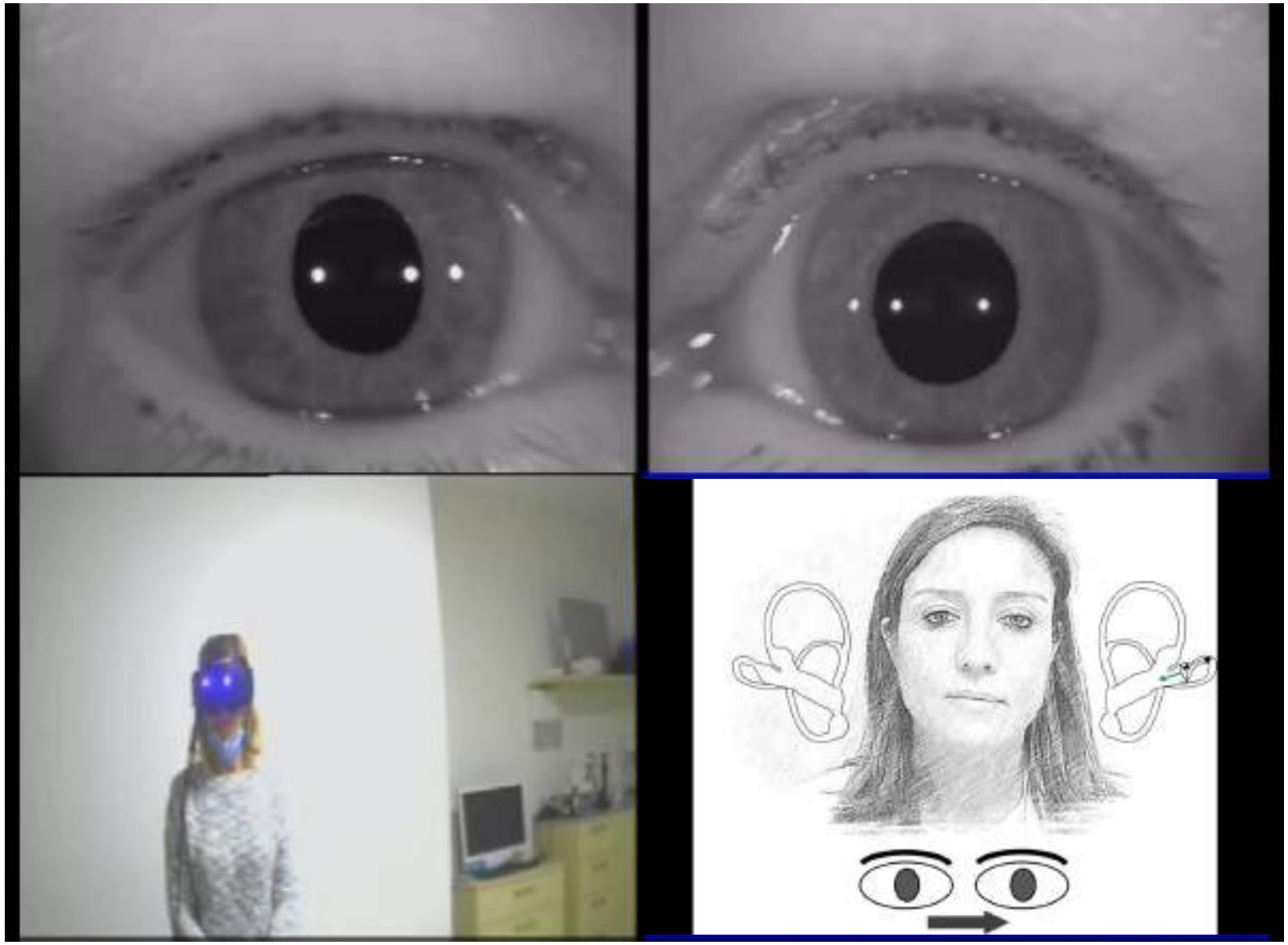


HSC canalith jam

- **CASE 3**

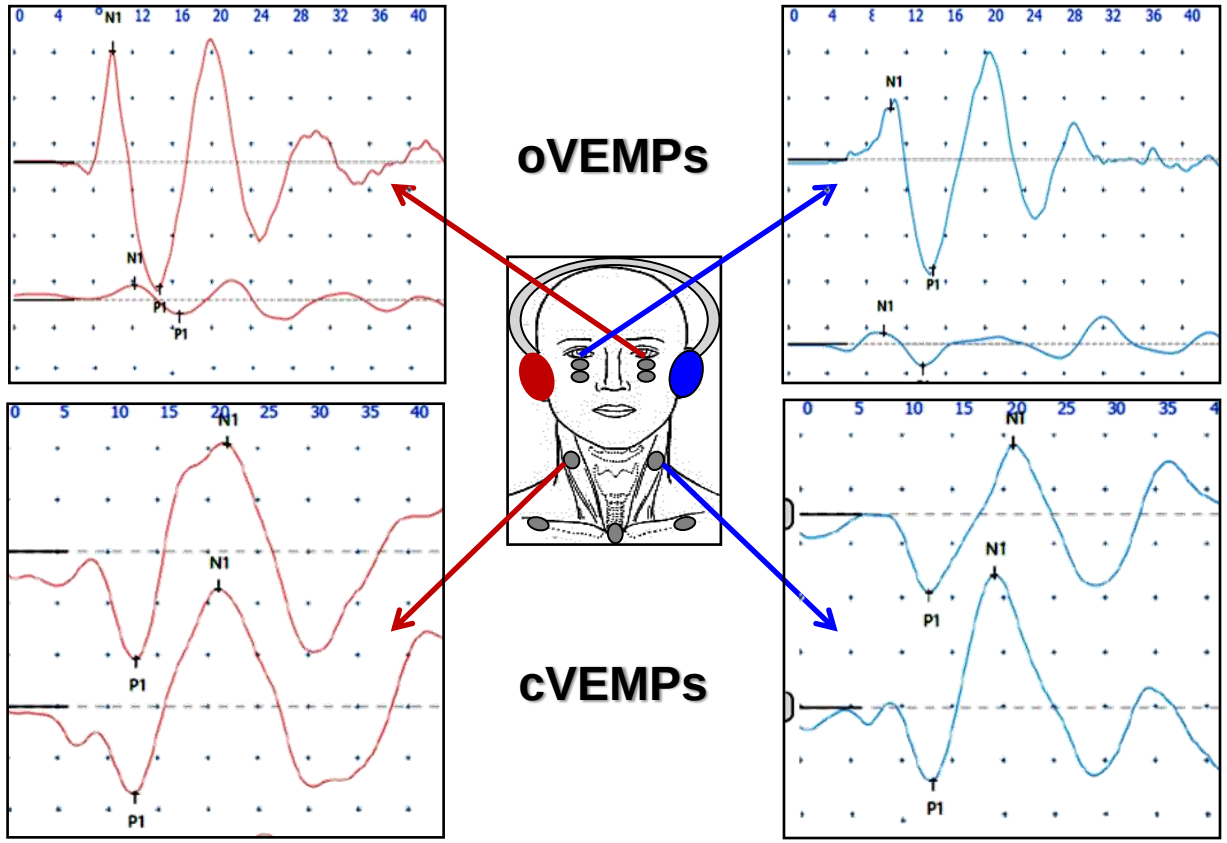
- 47-year-old woman
- Abrupt onset of **acute vertigo** with nausea and vomiting
- **Neither auditory symptoms** nor previous vestibular symptoms
- No migraine
- **Brain CT scan**: negative
- **Neurological examination:** unremarkable

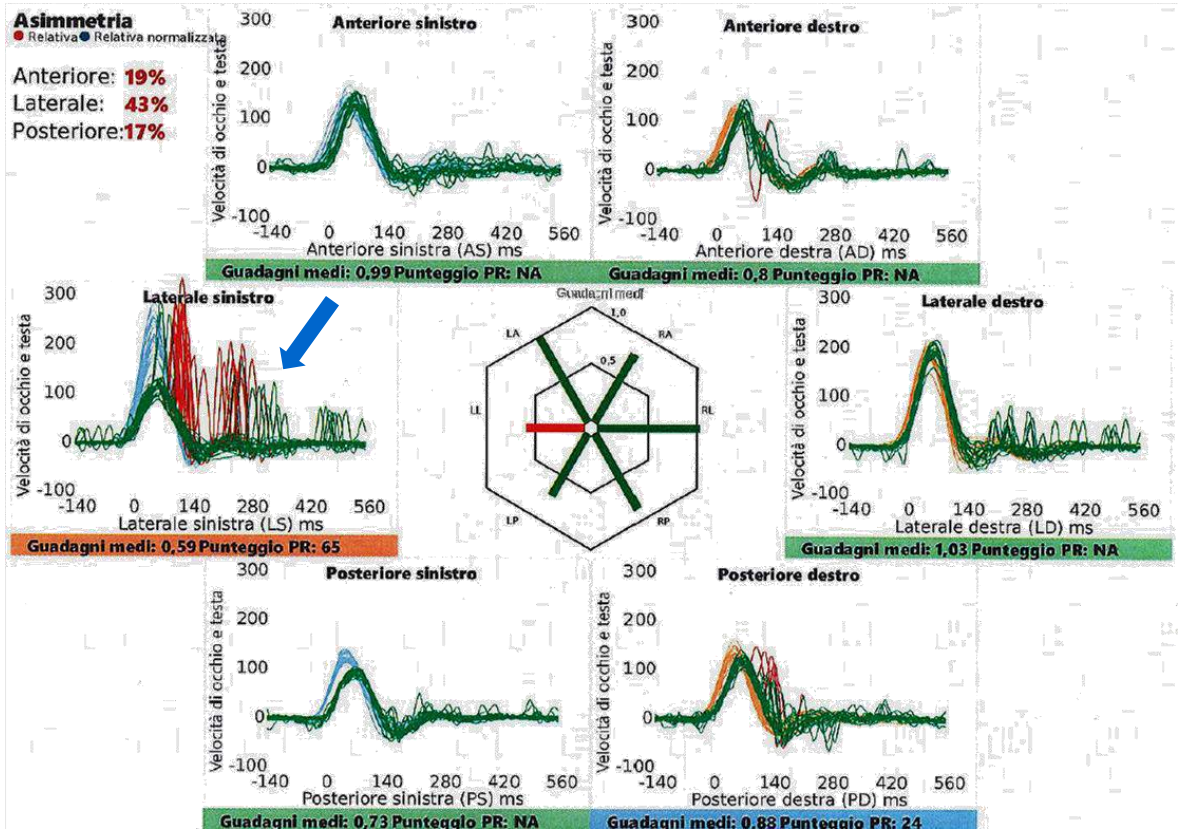
- **h-Bedside HIT: positive on the left** with re-fixation saccades



HSC canalith jam

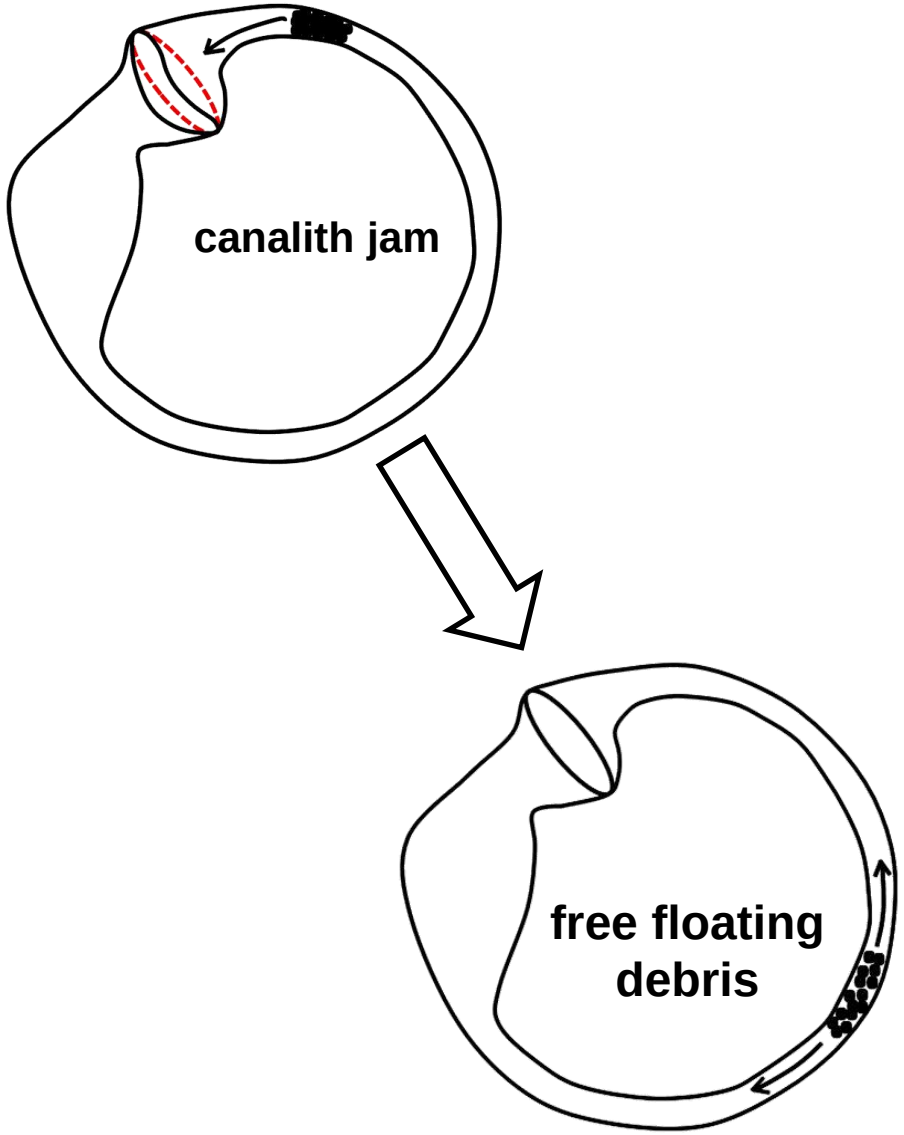
- **INSTRUMENTAL WORK-UP IN ACUTE STAGE**
  - **cVEMPs: symmetrical** amplitudes
  - **oVEMPs: symmetrical** amplitudes
  - **Video HIT: Selective left HSC hypofunction** (gain 0.59)



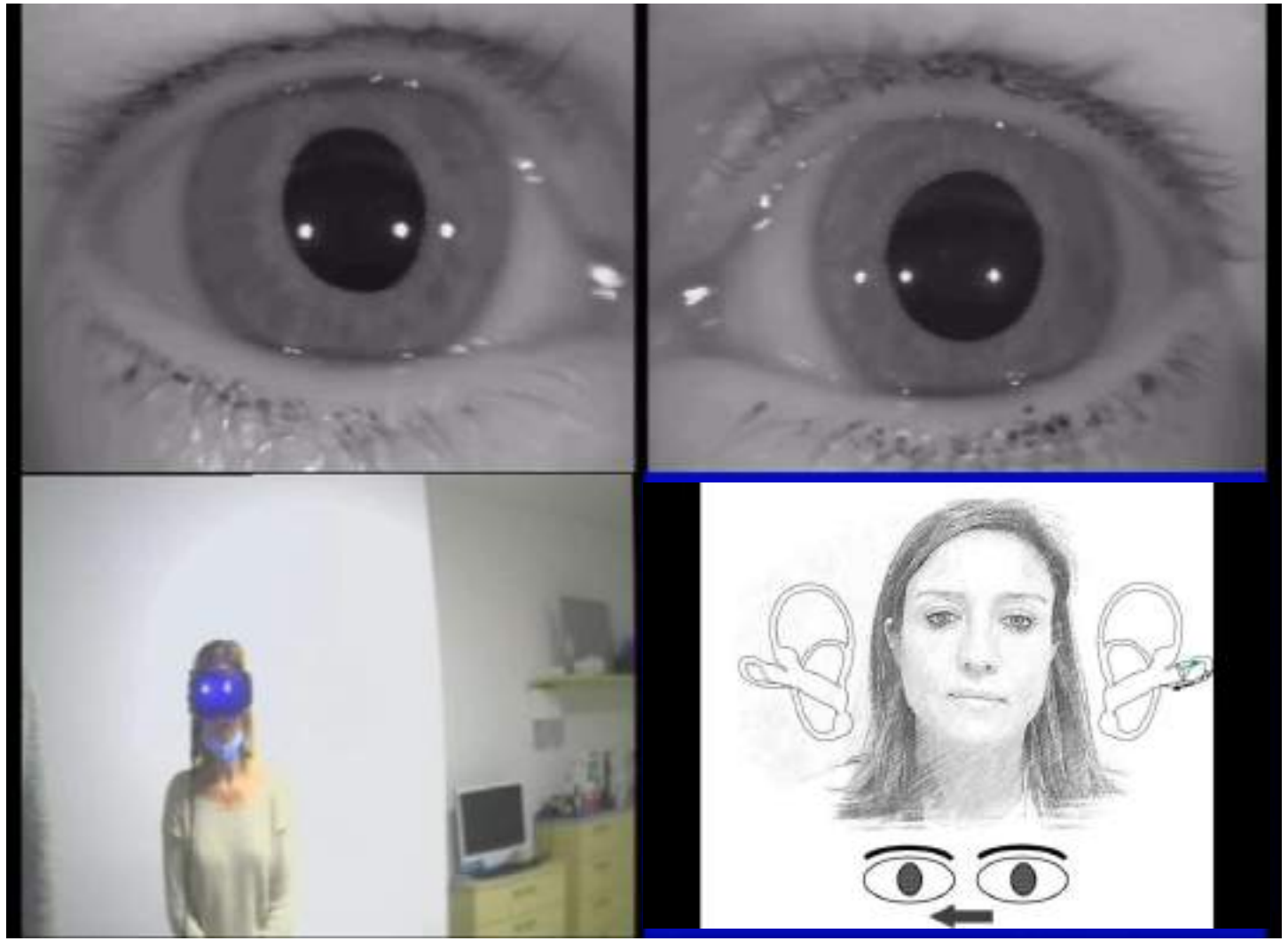


HSC canalith jam

- **HYPOTHETICAL PATHOMECHANISM**



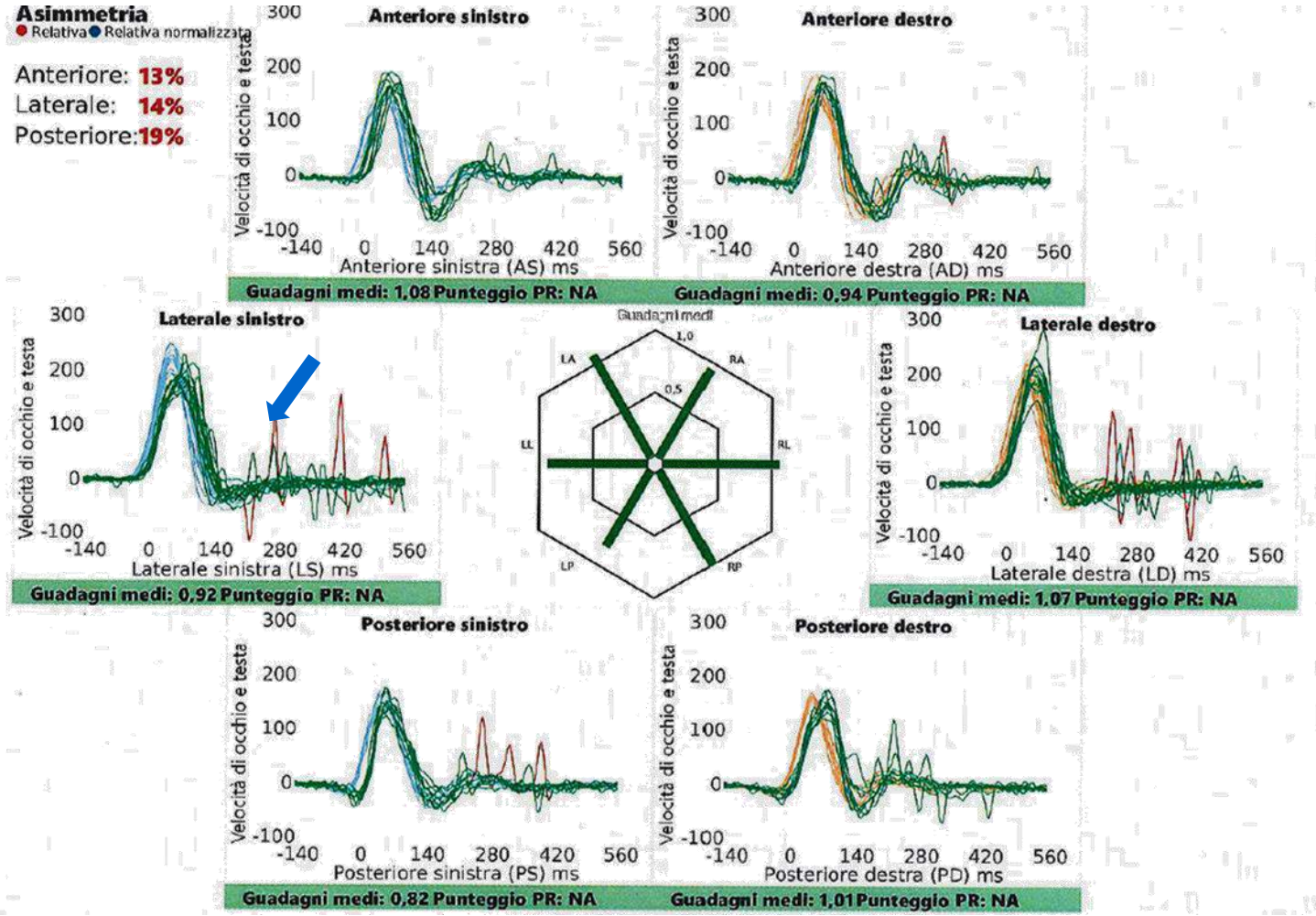
- **Video-Frenzel examination** (after 48 hours)



HSC canalith jam

- **INSTRUMENTAL WORK-UP FOLLOWING CRM for HSC-BPPV**

o **Video HIT: normalization of left HSC VOR-gain** (gain 0.92)



PSC canalith jam

- **CASE 1**

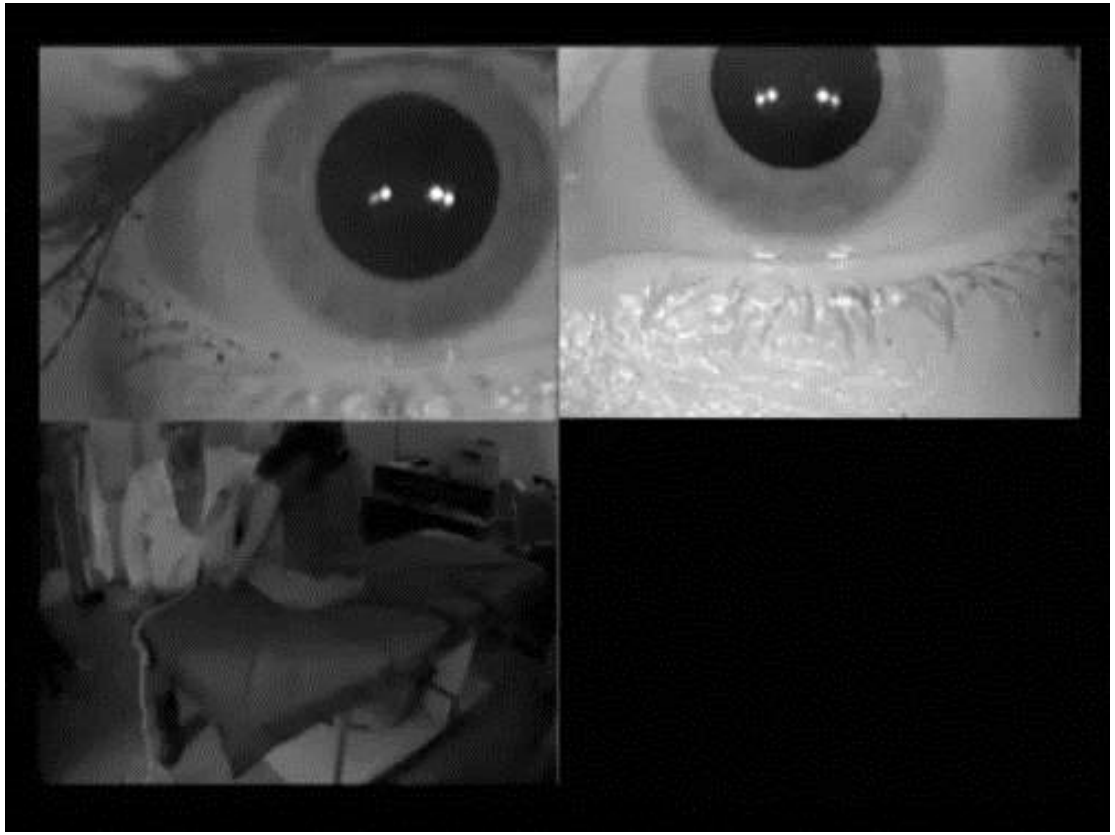
o 41-year-old woman

o **positional vertigo** without headache or auditory symptoms

o initially seen in the ED → diagnosis of **right PSC-BPPV**

o brain CT scan normal

o 3 days of **self-administered physical therapy** at home led to **worsening of symptoms with the onset of disabling dizziness**

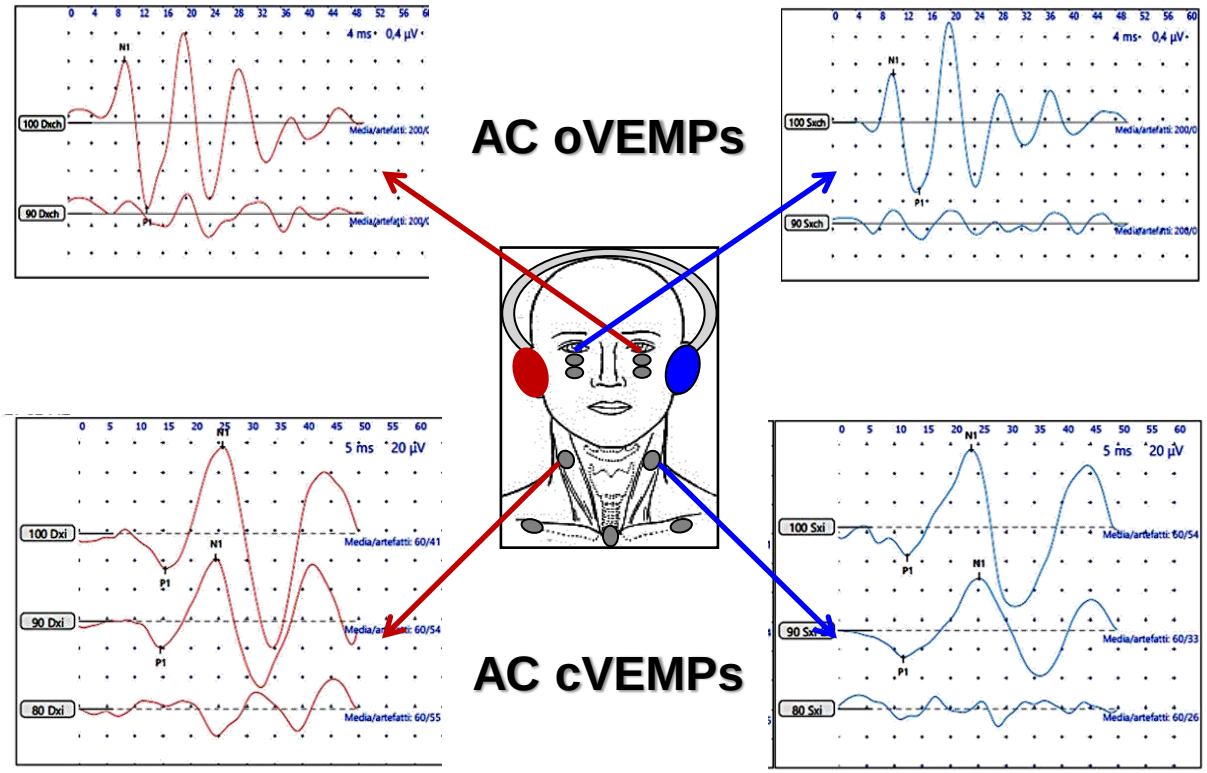


→ Central DBN? Peripheral DBN?

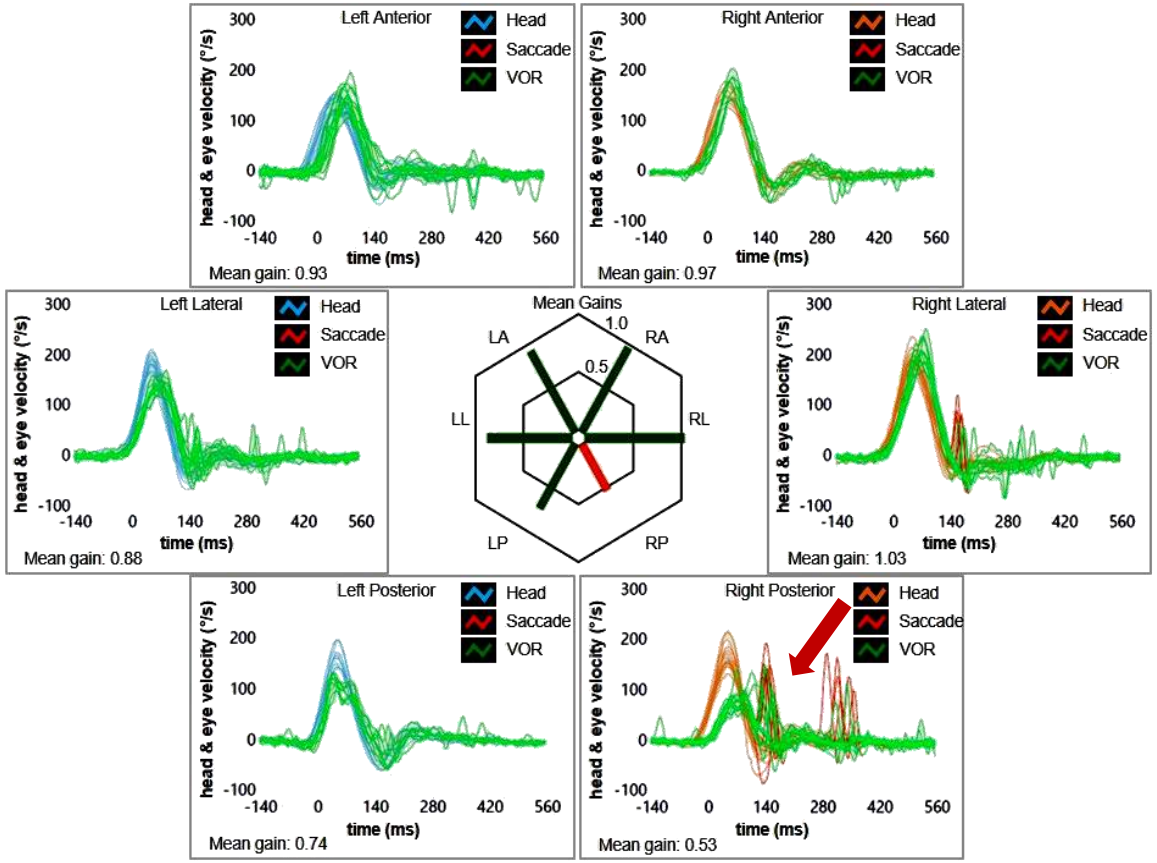
PSC canalith jam

- **INSTRUMENTAL WORK-UP**

  - **cVEMPs: symmetrical** amplitudes
  - **oVEMPs: symmetrical** amplitudes



  - **Video HIT: Selective right PSC hypofunction** (gain 0.53)

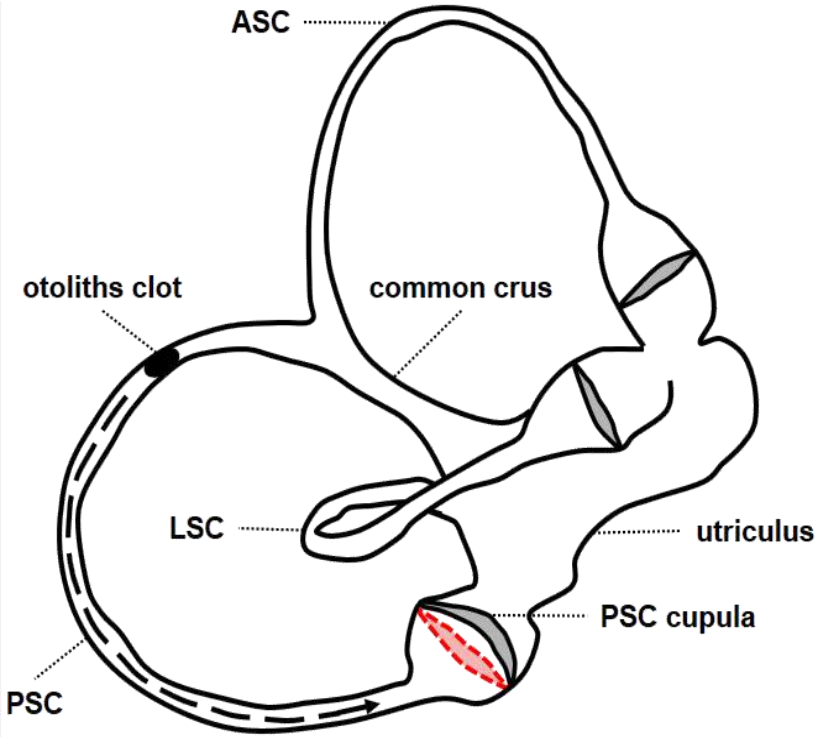


PSC canalith jam

- **HYPOTHETICAL PATHOMECHANISM**

o **Right PSC pseudo-hyporeflexia** induced by a **canalith jam** resulting in a **selective and complete canal deficit.**

o An **otolith clot completely entrapped** within the membranous duct could likely exert a **continuous positive pressure on the cupula**, resulting a **persistent utricolopetal (inhibitory) deflection, preventing right PSC dynamics by blocking endolymphatic flows and ampullary responses**



Neurological Sciences
https://doi.org/10.1007/s10072-020-04529-9

LETTER TO THE EDITOR

Spontaneous downbeat nystagmus in posterior semicircular canal benign paroxysmal positional vertigo: a canalith jam?

Andrea Castellucci[1] · Pasquale Malara[2] · Angelo Ghidini[1]

- This condition could likely result in:

o **Spontaneous downbeat/left-torsional nystagmus** regardless of head position

o **PSC VOR deficit** for both low- (?) and high-frequency (vHIT) domains

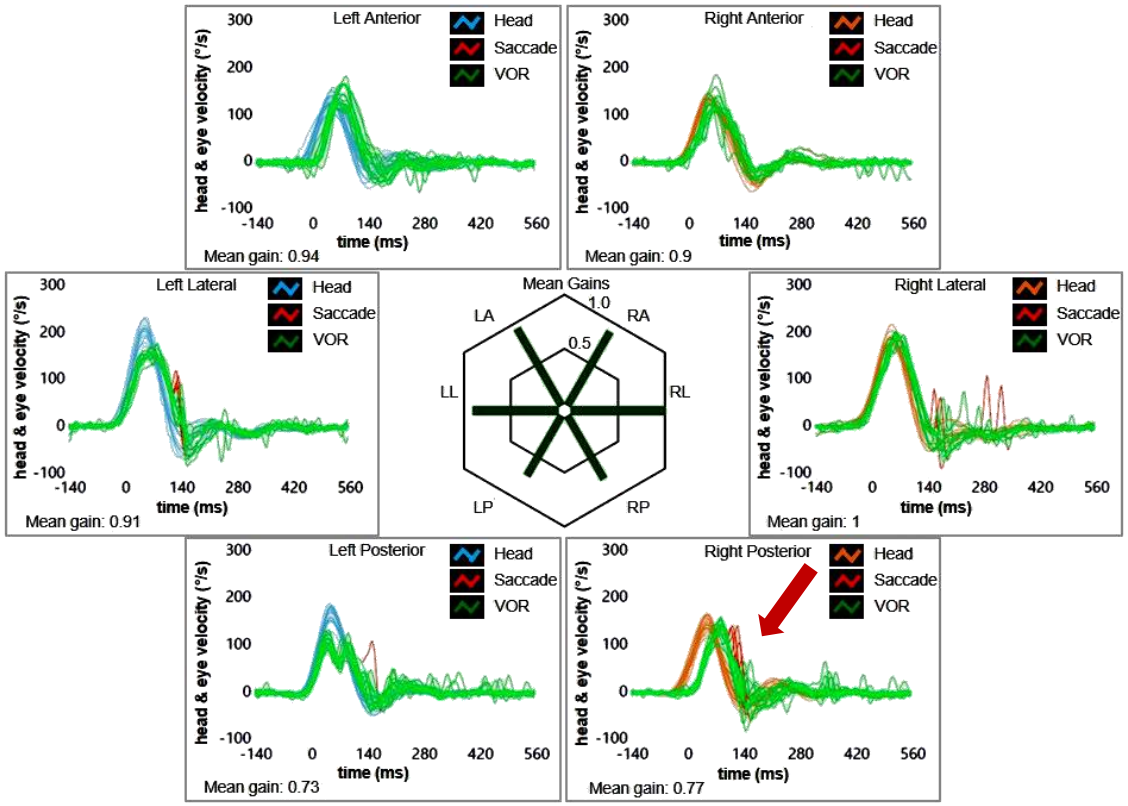
PSC canalith jam

- **Video-Frenzel evaluation**

o Demi Semont maneuvers and **resolution of nystagmus** following CRM

- **INSTRUMENTAL WORK-UP FOLLOWING CRM**

o **Video HIT: normalization of PSC VOR-gain** (gain 0.77)



PSC canalith jam

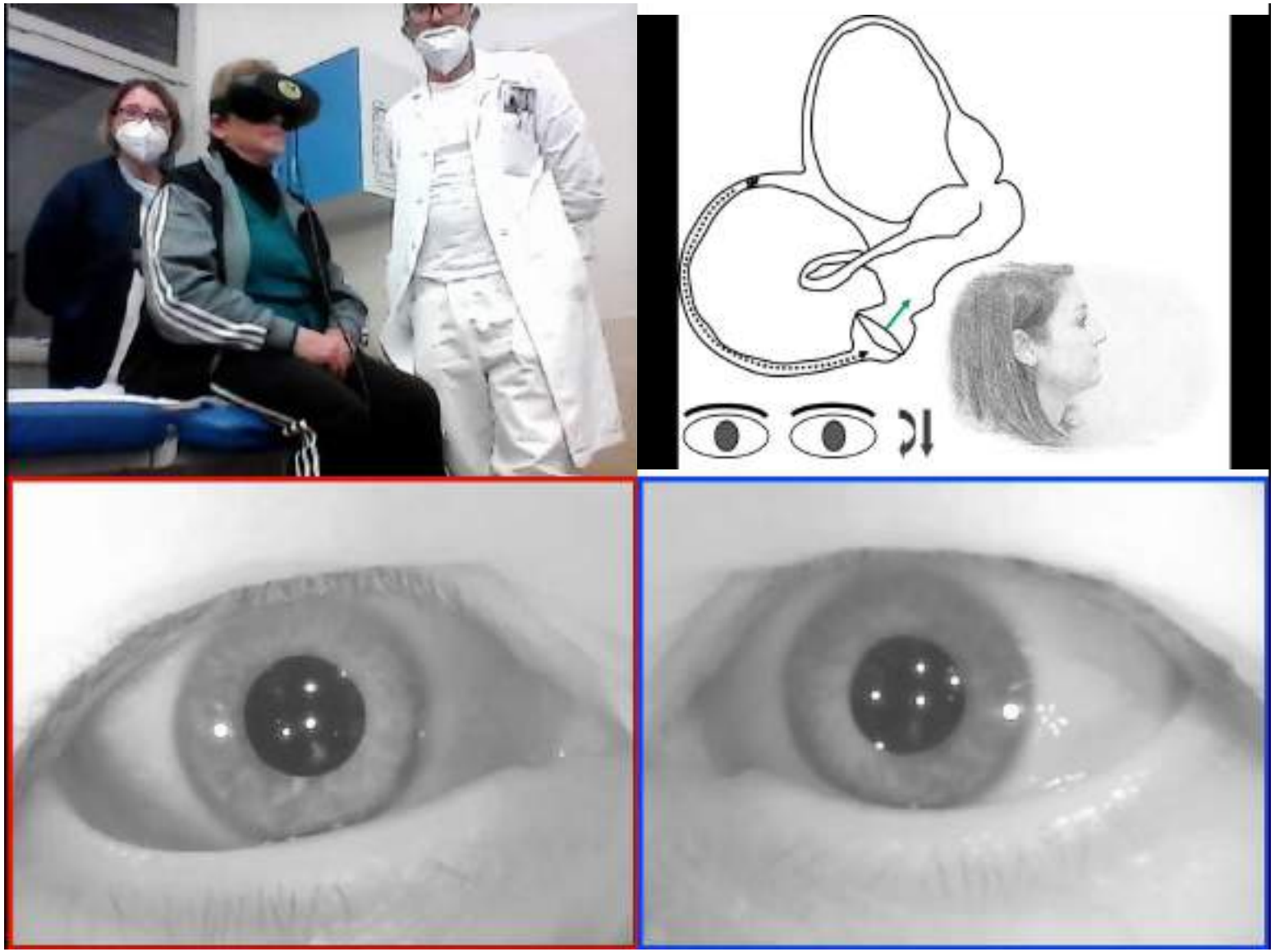
- **CASE 2**

o 69-year-old woman

o **Acute onset of unsteadiness** without headache or auditory symptoms

o brain CT scan normal

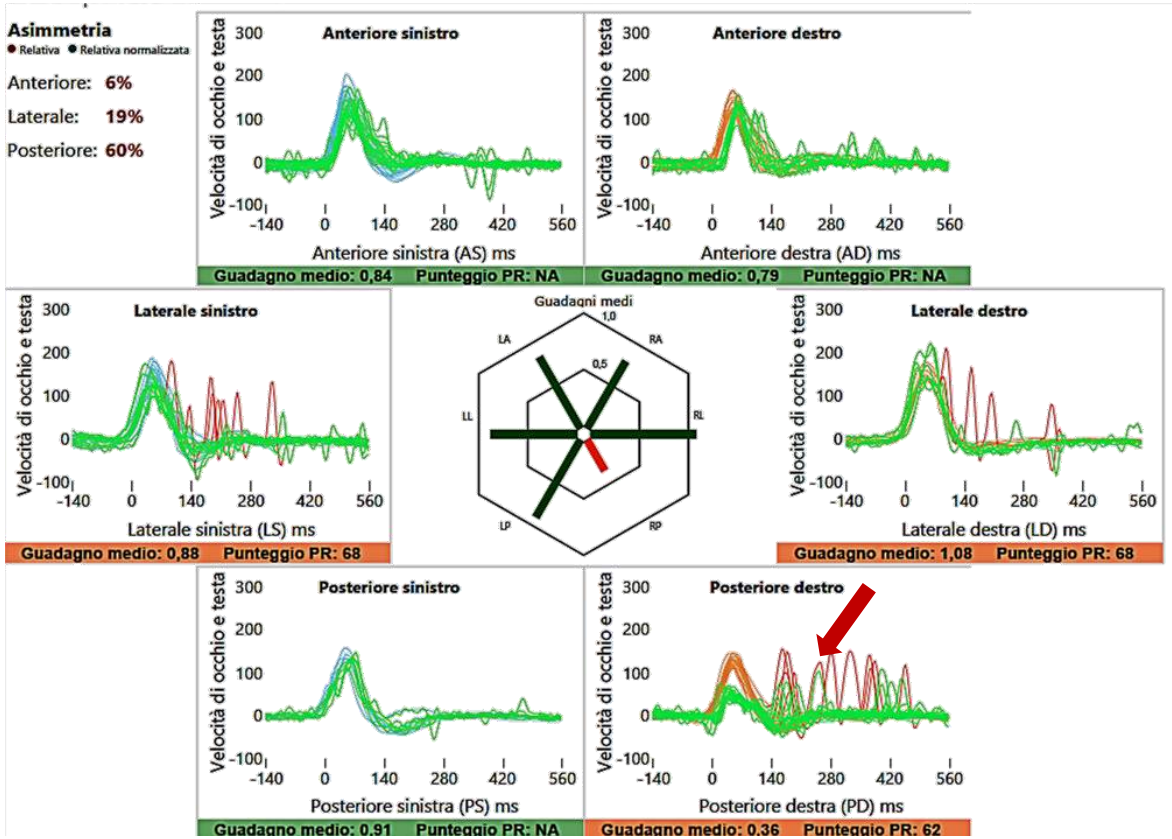
o Arterial hypertension, dislipidemia



PSC canalith jam

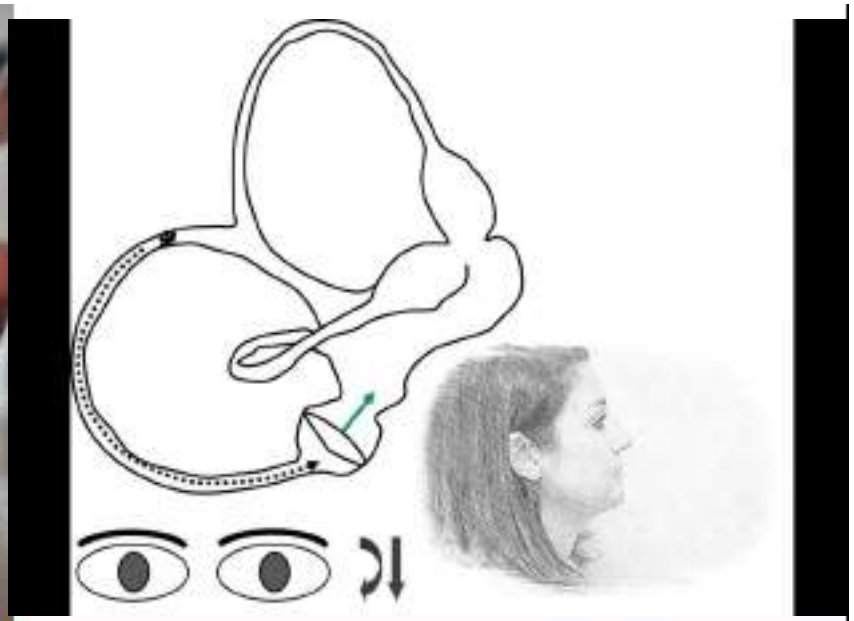
- **Bedside HIT**

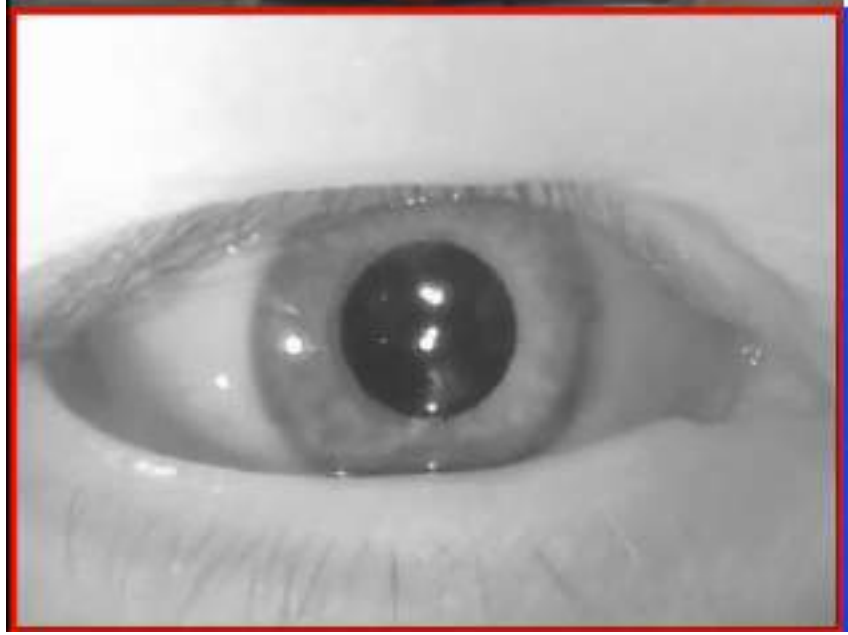
- **Video HIT: Selective right PSC hypofunction** (gain 0.36)

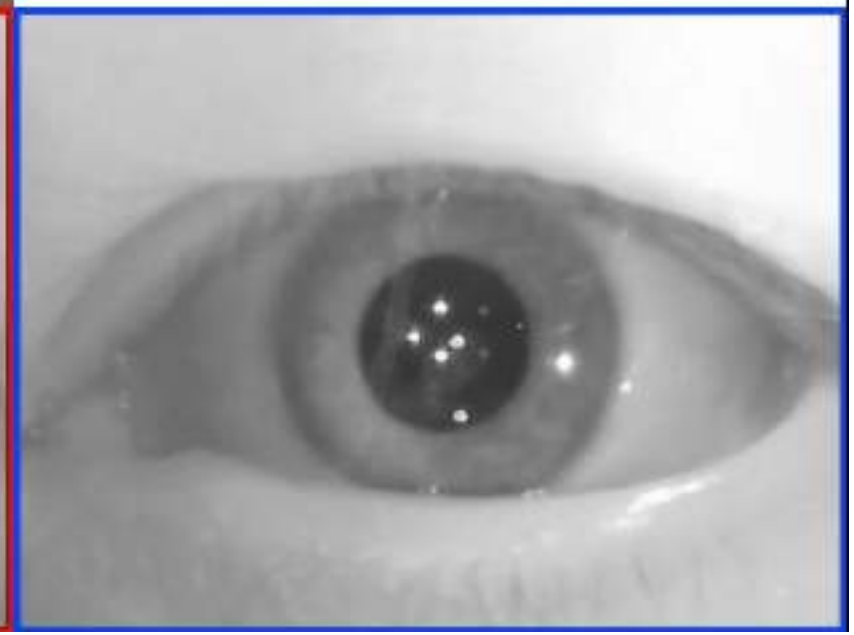


PSC canalith jam

- **1 week later**

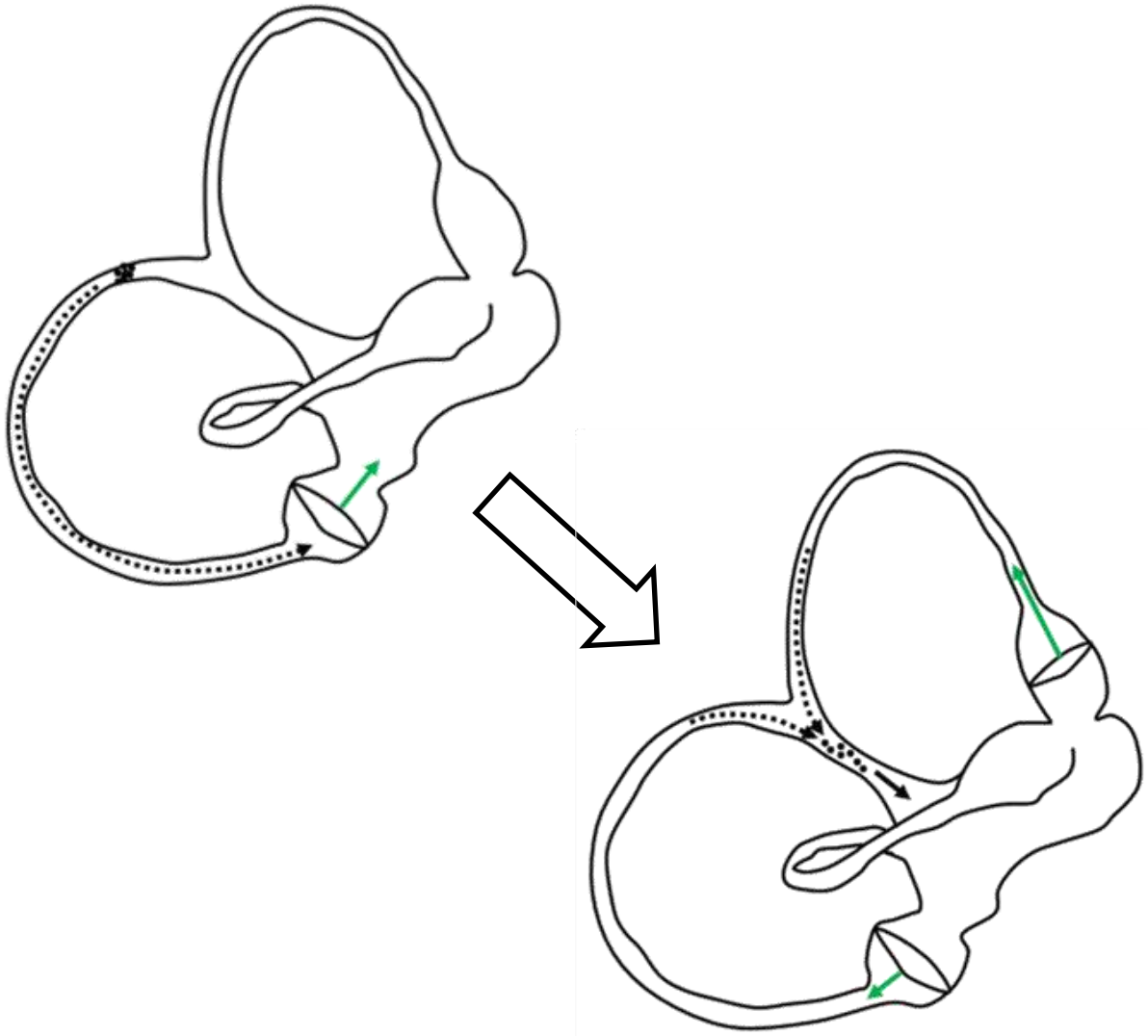




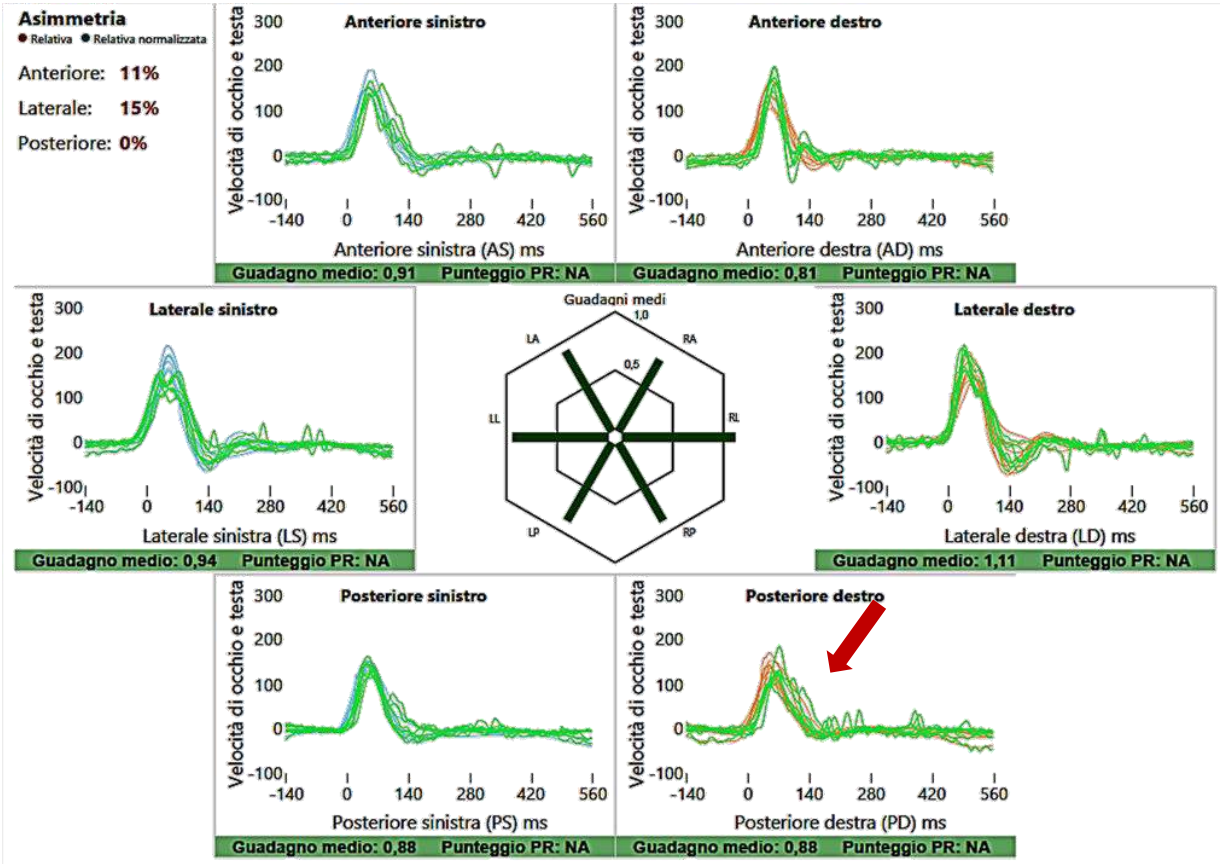


PSC canalith jam

- **HYPOTHETICAL PATHOMECHANISM**



- **Video HIT: functional restoration of the right PSC** (gain 0.88)



PSC canalith jam

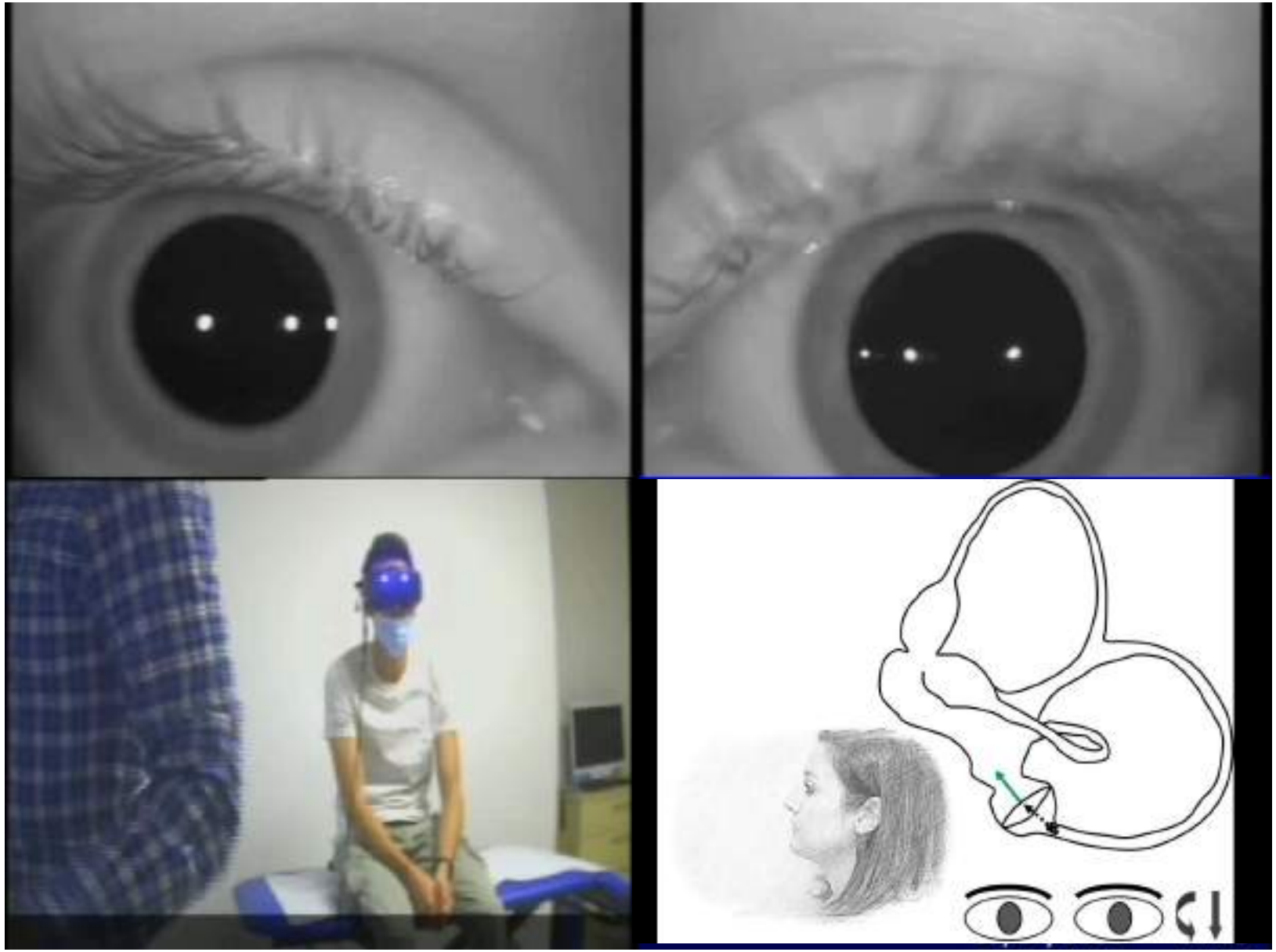
- **CASE 3**

o 42-year-old man

o **Recent onset of positional vertigo**

o No headache

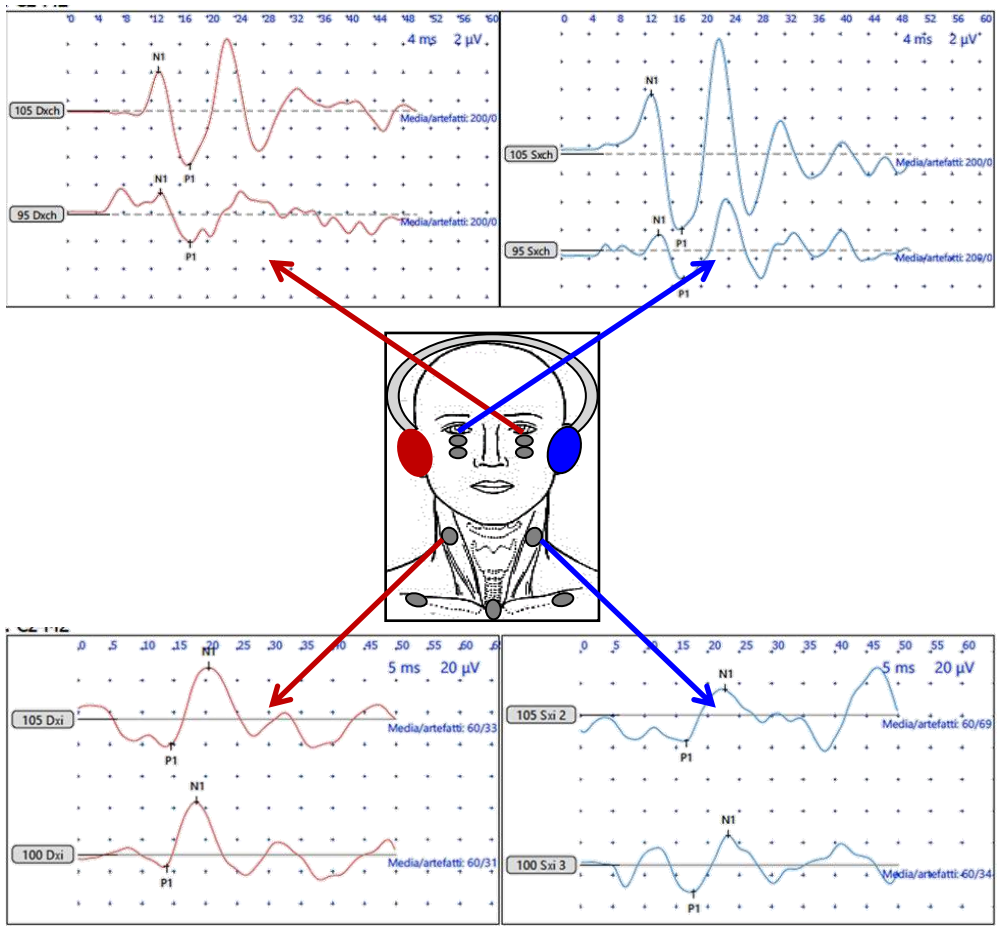
o No auditory symptoms



PSC canalith jam

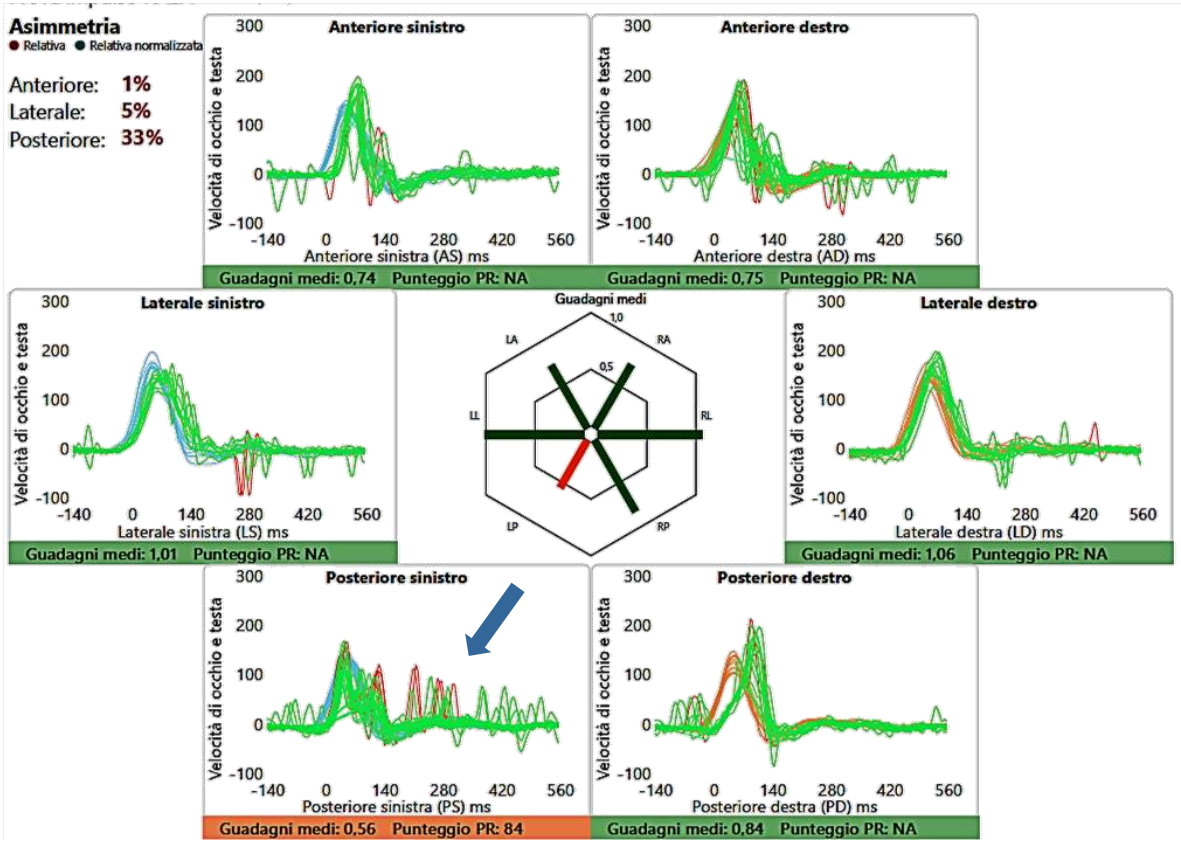
- **INSTRUMENTAL WORK-UP**

oVEMPs



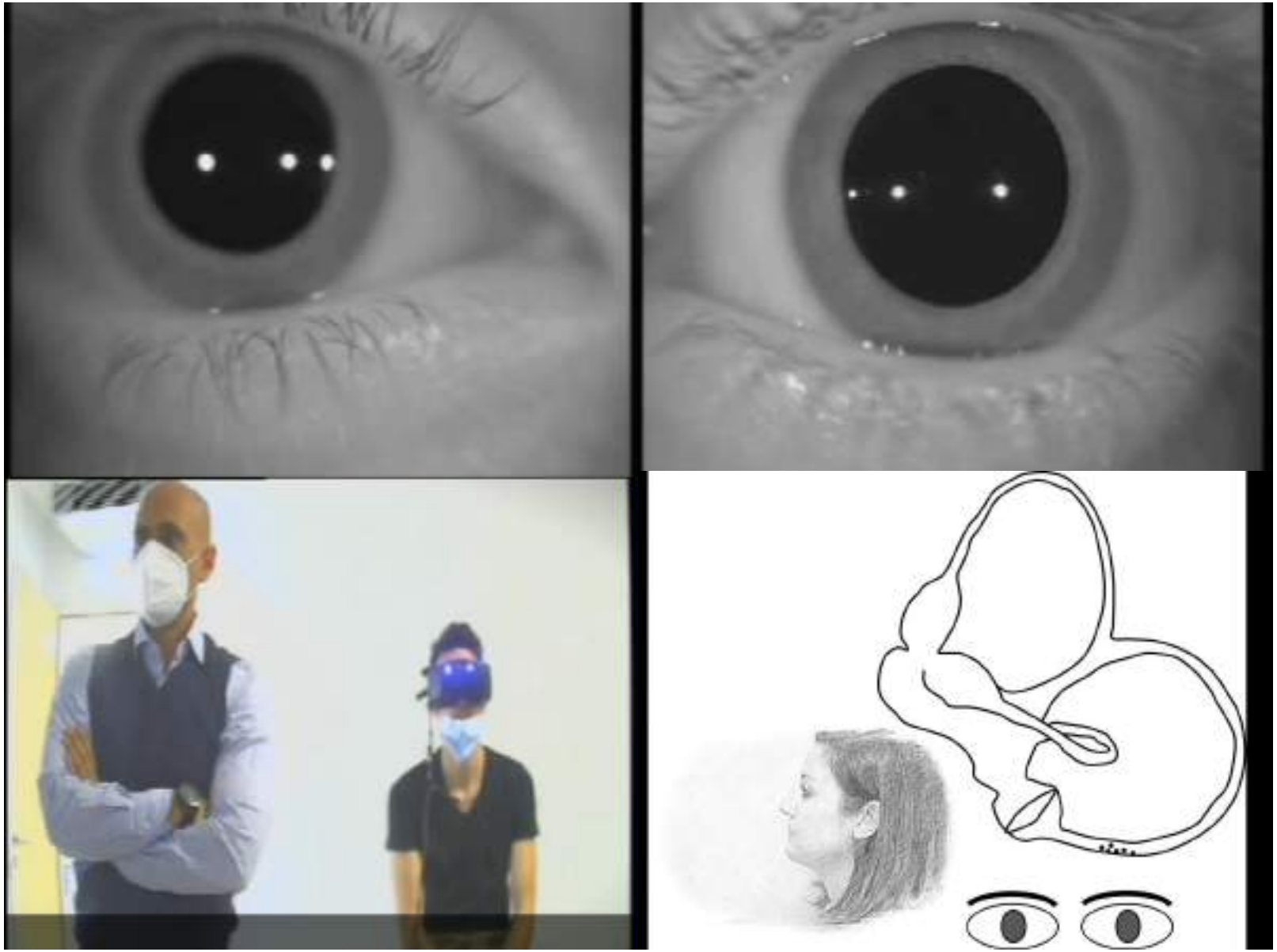
cVEMPs

- **Video HIT: Selective right PSC hypofunction** (gain 0.56)



PSC canalith jam

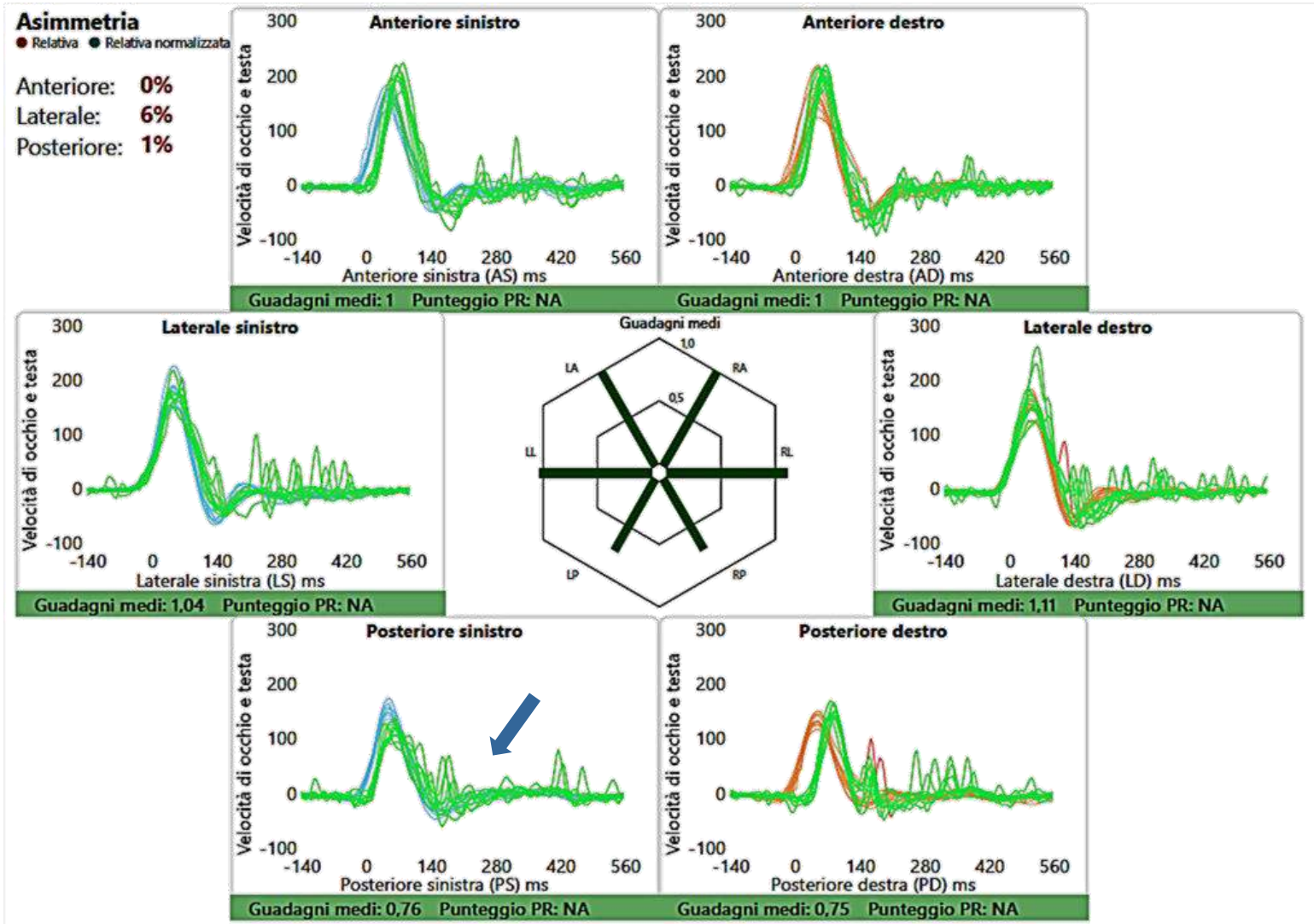
- **1 week later following CRM for refractory PSC-BPPV**



PSC canalith jam

- **INSTRUMENTAL WORK-UP FOLLOWING CRM for refractory PSC-BPPV**

o **Video HIT: normalization of left HSC VOR-gain** (gain 0.76)



ASC canalith jam

- **CASE 1**

o 56-year-old woman

o Recently received repeated **CRM for refractory left PSC-BPPV**

o Symptoms progressively turned into **continuous unsteadiness.**

o **Neither concurrent auditory symptoms, nor migraine**

o Neurological evaluation and **brain MRI**: no CNS sings

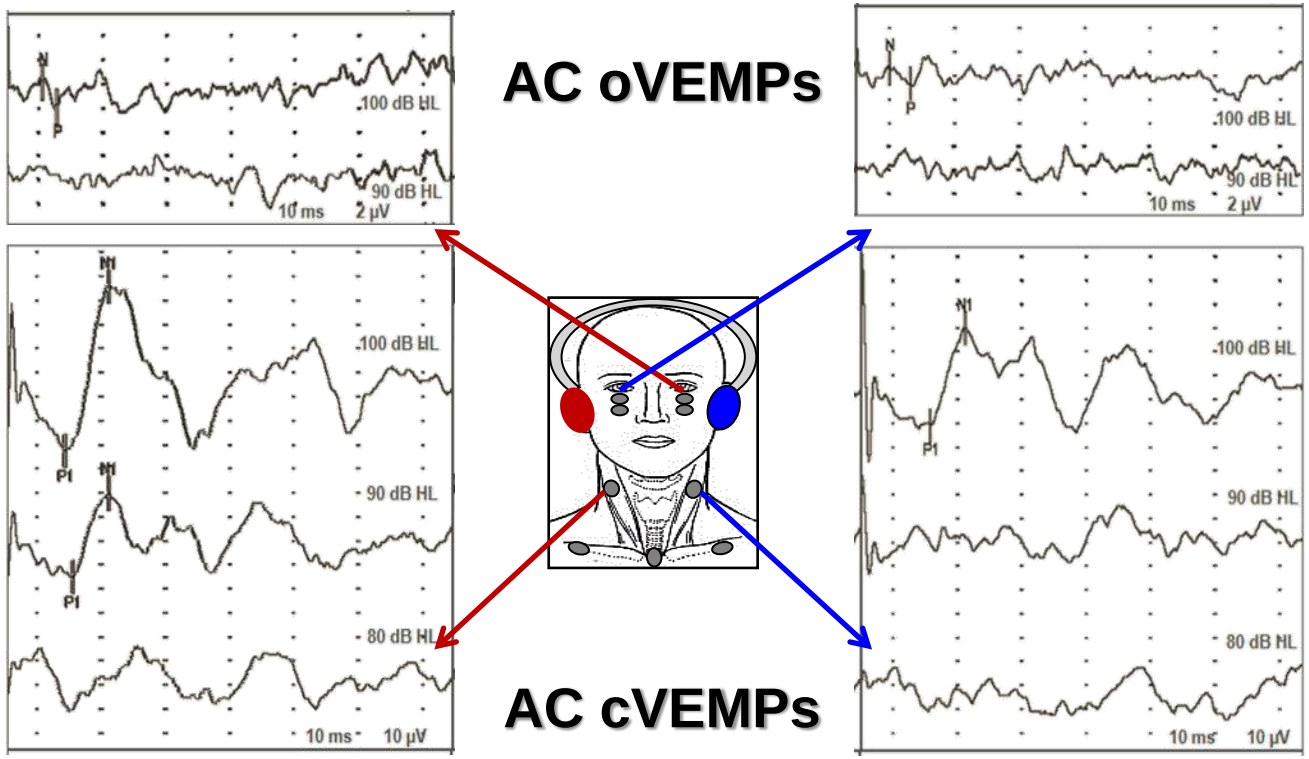
o Audiometry: slight bilateral HFSNHL (L > R)

→ Central UBN? Peripheral UBN?

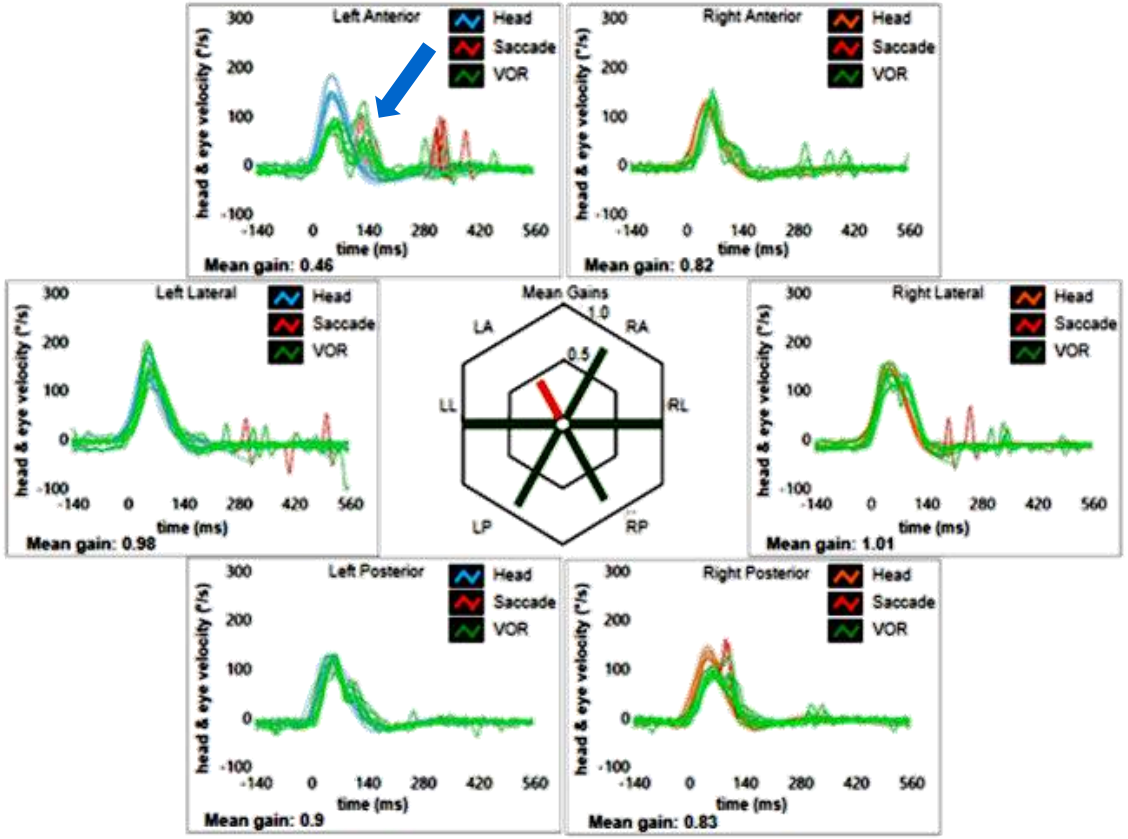
ASC canalith jam

- **INSTRUMENTAL WORK-UP**

  - **cVEMPs:** amplitudes R > L (within normal AR: 25%)
  - **oVEMPs:** amplitudes R > L (within normal AR: 19%)

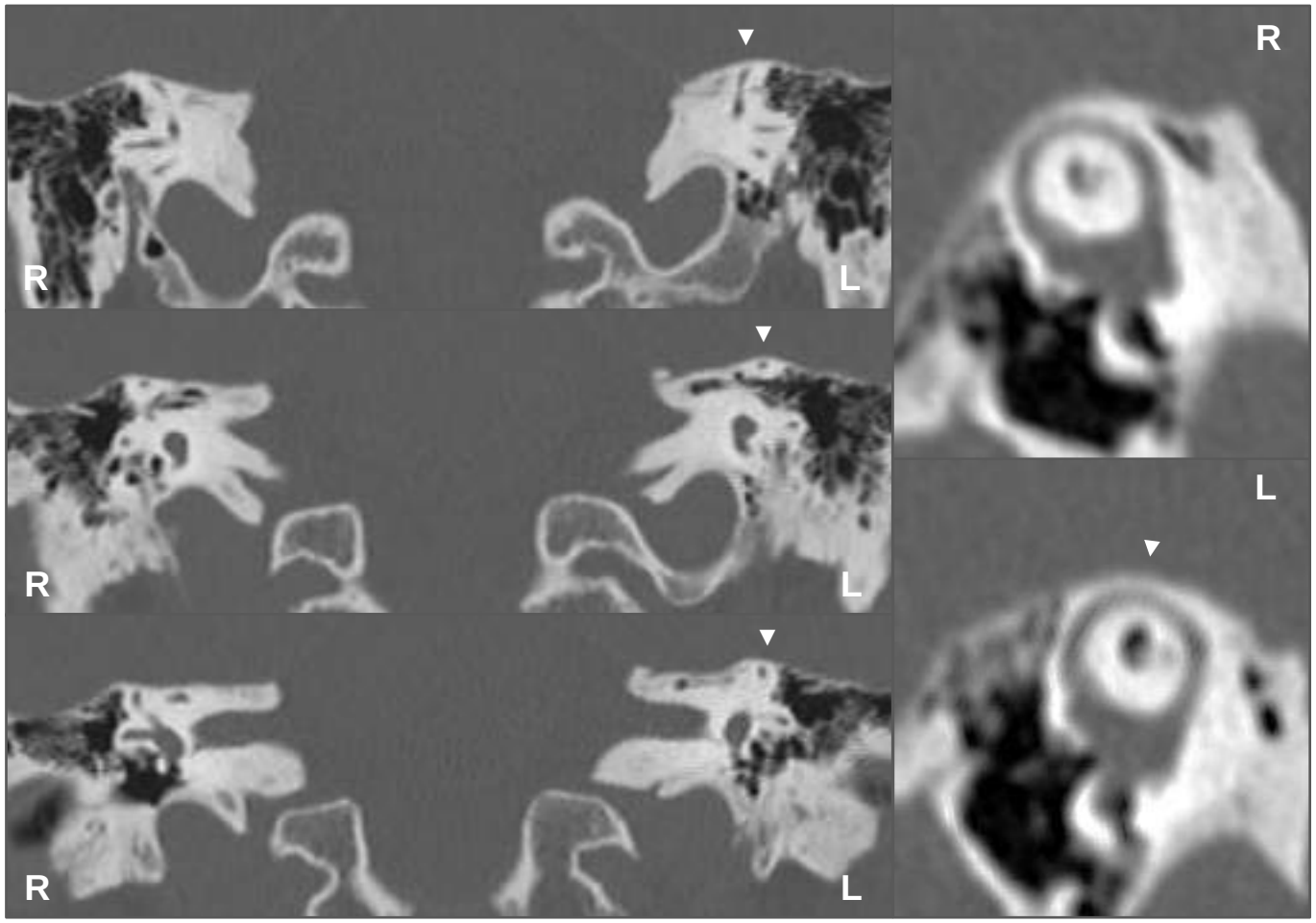


  - **Video HIT:** **Selective left ASC hypofunction** (gain 0.46)



ASC canalith jam

- **IMAGING**
  - **HRCT scans: no signs of SCD** on the left



→ Possible canalith jam of the left ASC?

ASC canalith jam

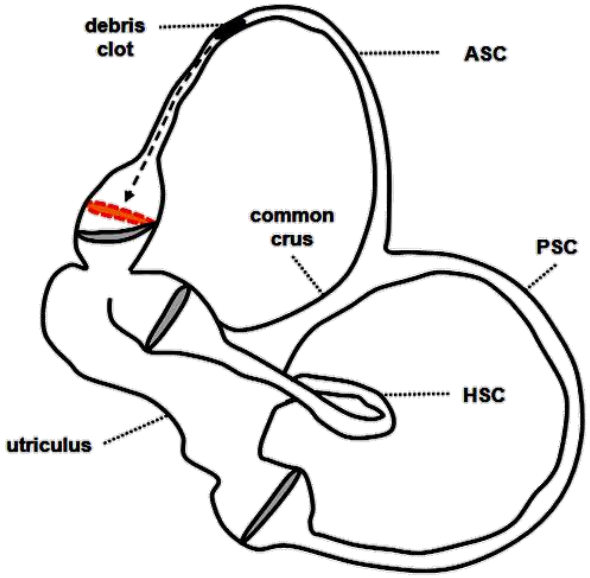
CASE REPORT
J Audiol Otol 2021 Nov 23 [Epub ahead of print]
pISSN 2384-1621 / eISSN 2384-1710
https://doi.org/10.7874/jao.2021.00297

Spontaneous Upbeat Nystagmus and Selective Anterior Semicircular Canal Hypofunction on Video Head Impulse Test: A New Variant of Canalith Jam?

Andrea Castellucci[1], Cecilia Botti[1,2], Salvatore Martellucci[3], Pasquale Malara[4], Silvia Delmonte[1], Francesca Lusetti[1], and Angelo Ghidini[1]

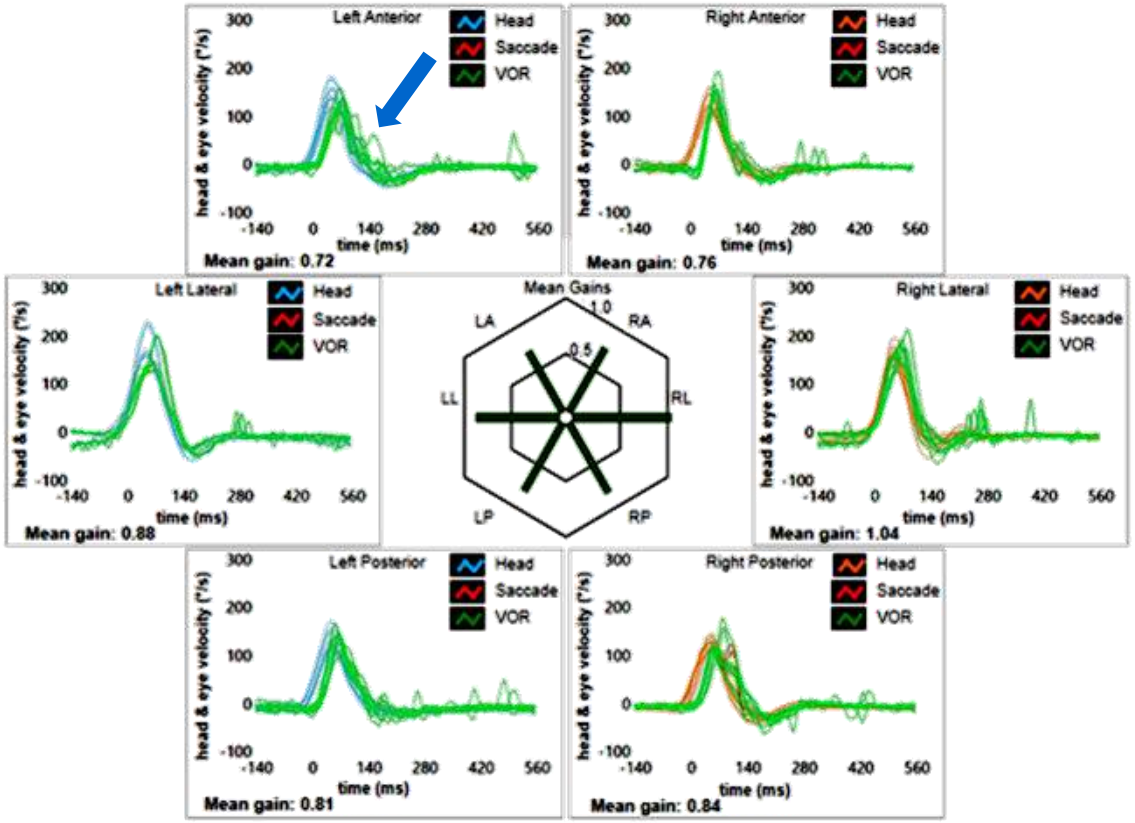
- **HYPOTHETICAL PATHOMECHANISM**

o **Left ASC pseudo-hyporeflexia** due to a **canalith jam** inducing a **selective plug of the canal lumen** and a **continuous positive (inhibitory) pressure on the cupula**



- **Video-Frenzel evaluation after Brandt-Daroff exercises → conversion in typical left PSC-BPPV**

- **Video HIT: Normalization of left ASC VOR-gain** (gain 0.72)

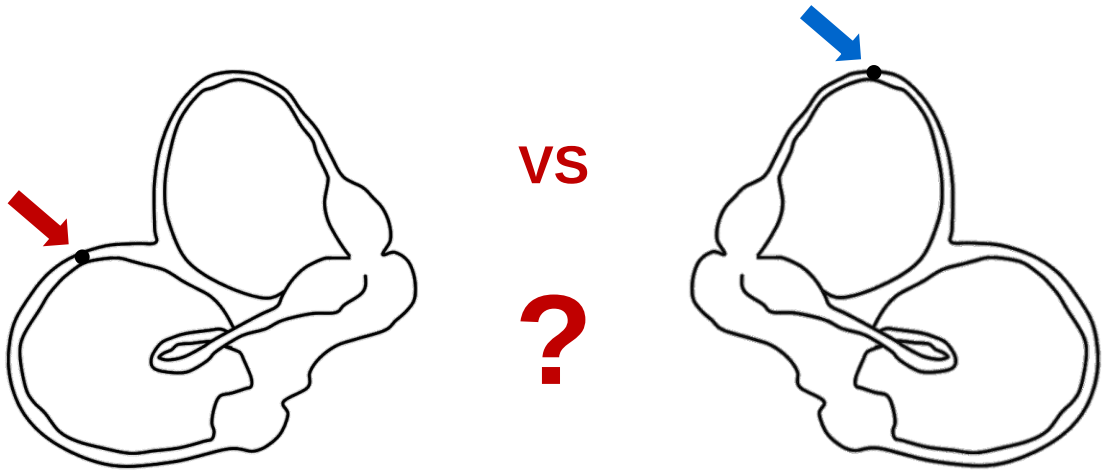


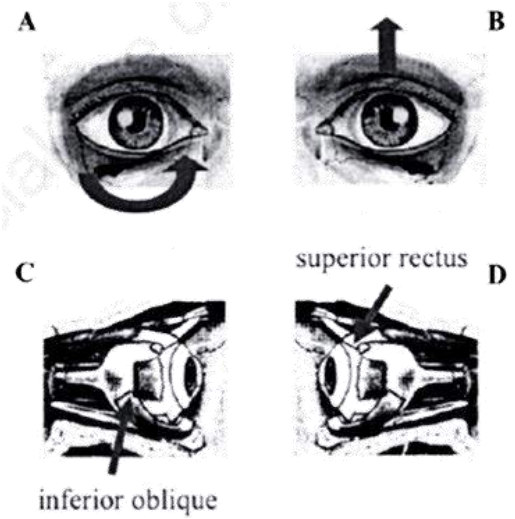
Incomplete canalith jam

- **BPPV involving either the ASC** or **the non-ampullary arm of the PSC** lead to **positional downbeat nystagmus (pDBN)**

- In some cases, pDBN could **not have a clear torsional component** and could **exhibit a persistent course, mimicking central pDBN**

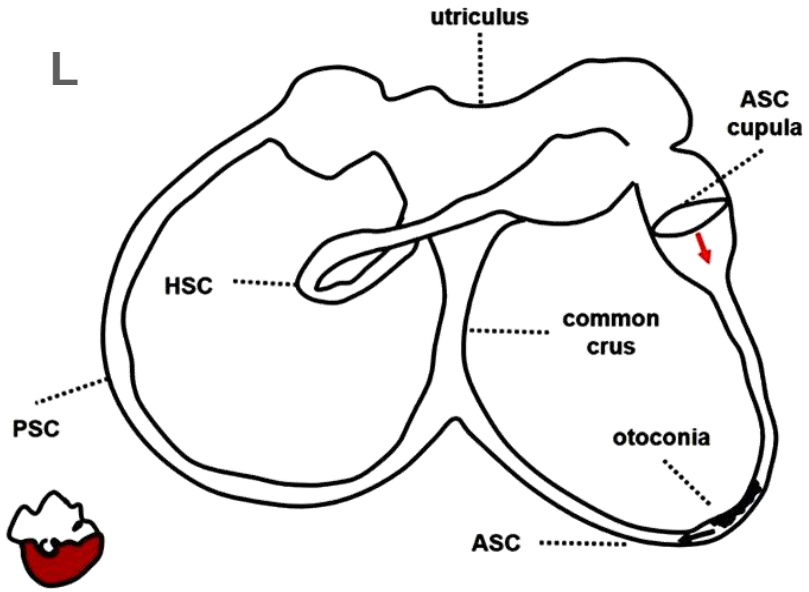
- **Differential diagnosis could be challenging:**
  - **Central** vs **peripheral pDBN**
  - among peripheral forms, **apogeotropic PSC–BPPV** vs **contralateral ASC-BPPV**





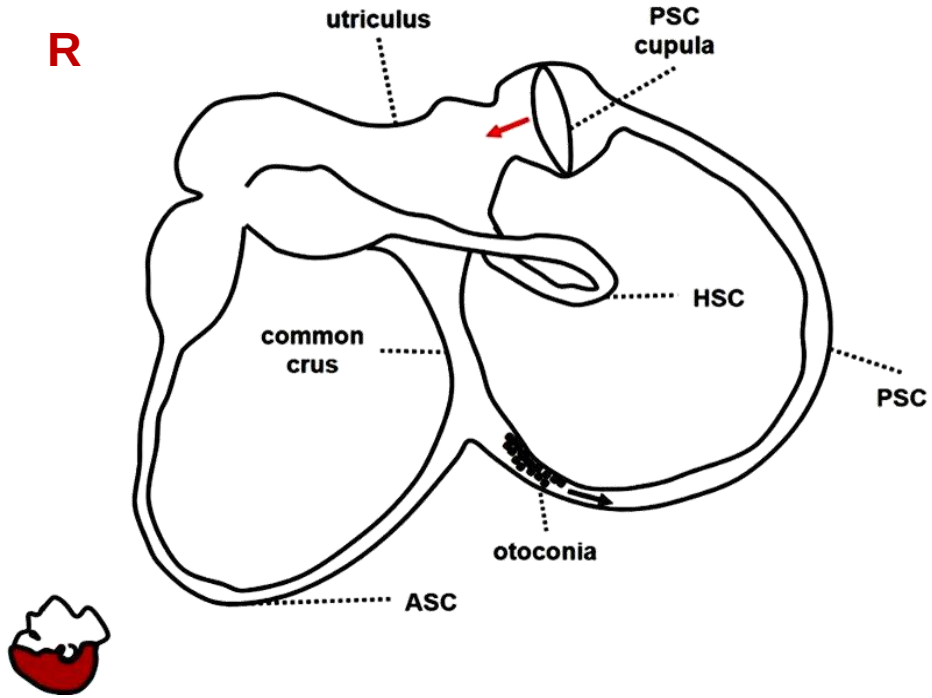
- **pDBN due to inhibition of PSC** is **hardly distinguishable** from **pDBN due to excitation of contralateral ASC** as nystagmus is generated from the **contraction of the same paired ocular muscles**

BPPV with pDBN



➢ **Left ASC-BPPV**

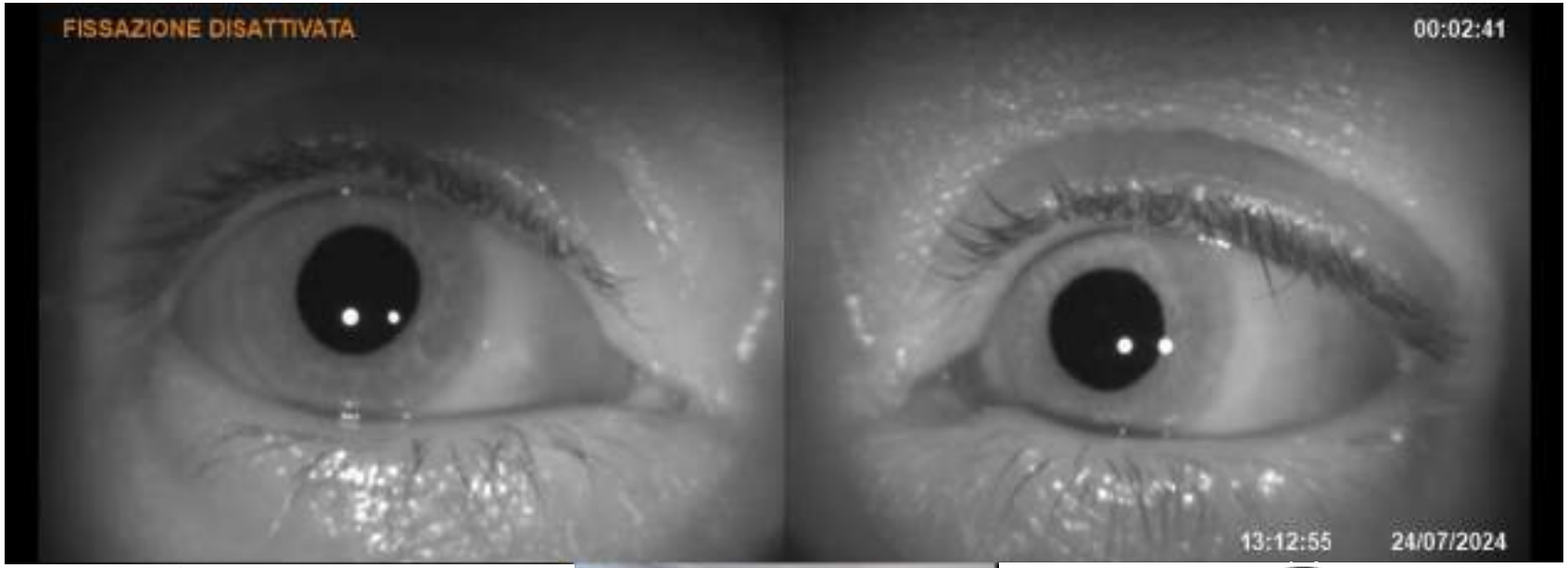
- Otoconia shift towards the common crus
- **Ampullofugal deflection (excitation) of ASC cupula**
- **pDBN with variable left-torsional component**

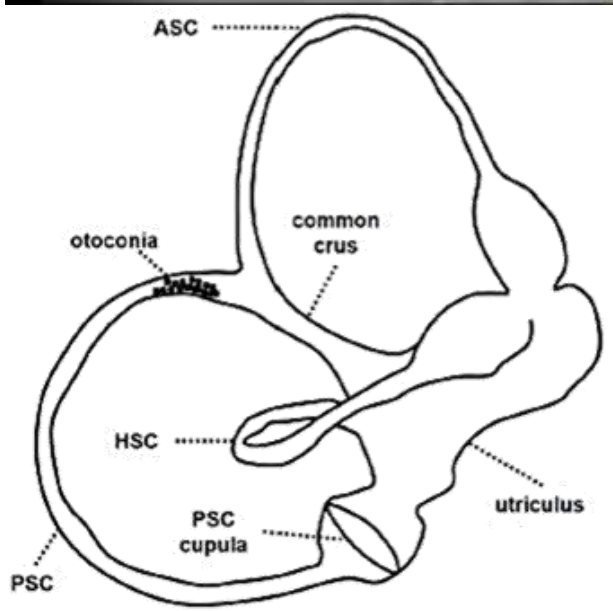


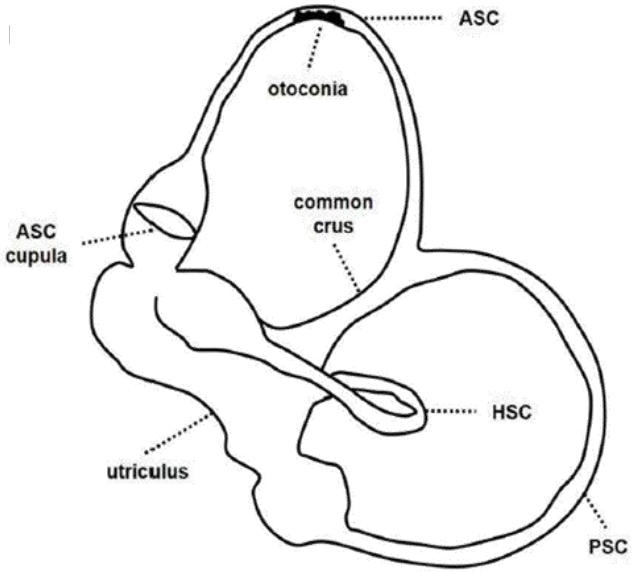
➢ **Right apogeotropic PSC-BPPV**

- Otoconia shift towards the ampullary arm
- **Ampullopetal deflection (inhibition) of PSC cupula**
- **pDBN with variable left-torsional component**

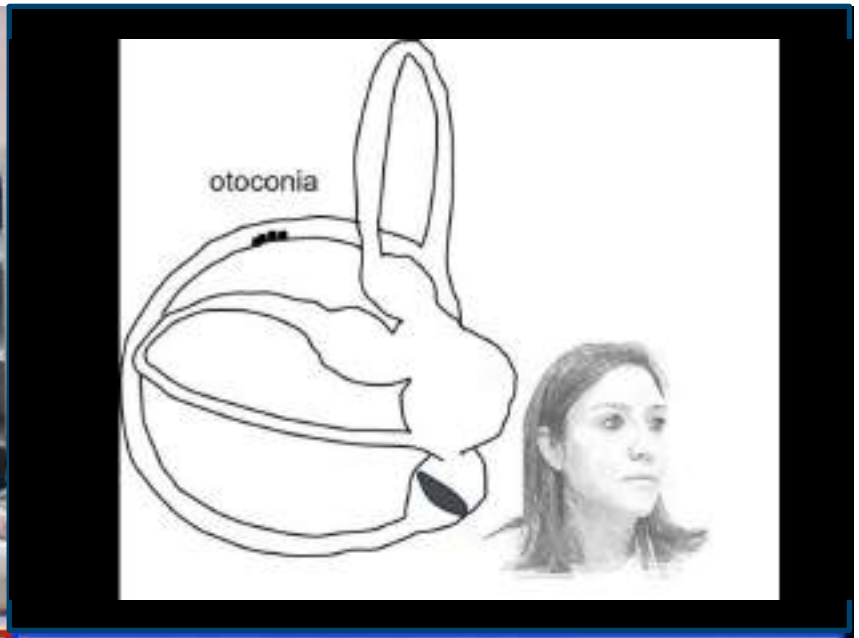
BPPV with pDBN

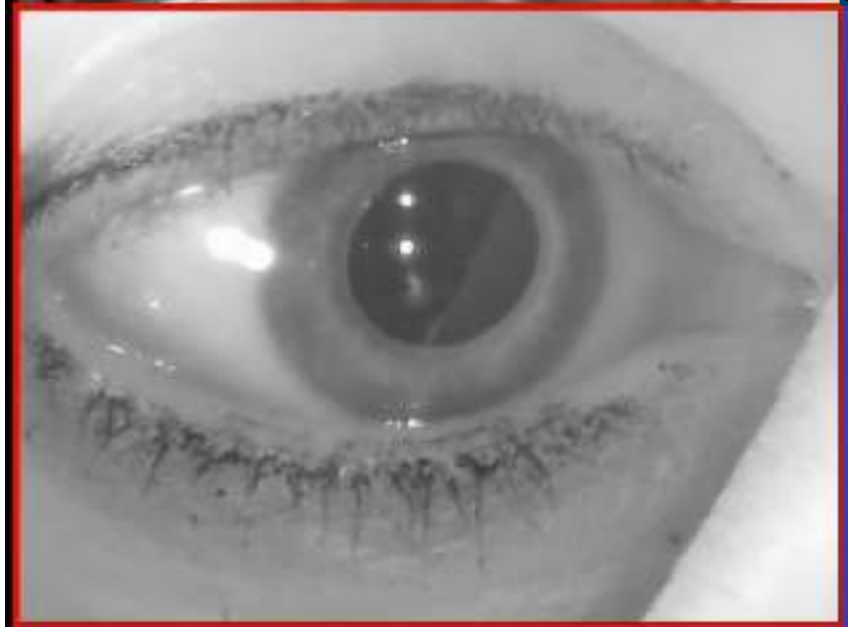






BPPV with pDBN





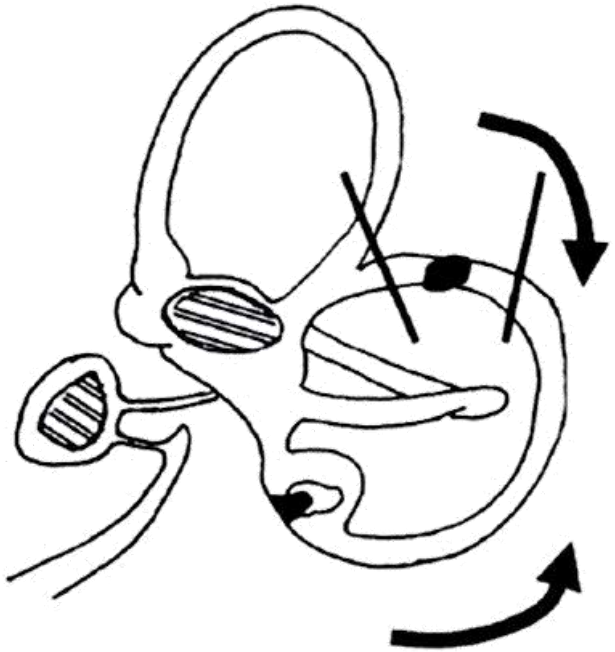
BPPV with pDBN

Audiology Research 2015; volume 5:130

Apogeotropic posterior semicircular canal benign paroxysmal positional vertigo: some clinical and therapeutic considerations

Paolo Vannucchi, Rudi Pecci, Beatrice Giannoni, Fabio Di Giustino, Rossana Santimone, Arianna Mengucci

Department of Surgical Sciences and Translational Medicine, Unit of Audiology, Careggi Hospital, University of Florence, Italy



- An **"incomplete canalith jam"** (also called **"positional jam"** or **"transient jam")** has been hypothesized

- **If vHIT** can precisely measure **high-frequency responses of ampullary afferents for each single canal**

o **vHIT** could **provide useful clues to detect the involved canal** in BPPV with pDBN (**non-ampullary arm PSC vs ASC**)

o It could potentially provide a **precise distinction between peripheral and central pDBN**

vHIT and typical (ampullary arm) PSC BPPV

Original Article
Iranian Journal of Otorhinolaryngology, Vol.29(5), Serial No.94, Sep 2017

Vestibulo-Ocular Reflex Abnormalities in Posterior Semicircular Canal Benign Paroxysmal Positional Vertigo: A Pilot Study

Tayyebe Fallahnezhad[1], *Mansoureh Adel Ghahraman[1], Saeid Farahani[1], Reza Hoseinabadi[1], Shohreh Jalaie[2]

Turkish Archives of Otorhinolaryngology
Türk Otorinolarengoloji Arşivi
Turk Arch Otorhinolaryngol 2018; 56(2): 81-4

Analyses with the Video Head Impulse Test During the Canalith Repositioning Maneuver in Patients with Isolated Posterior Semicircular Canal Benign Paroxysmal Positional Vertigo

Yusuf Çınar[1], Ali Bayram[1], Ramazan Culfa[2], Cemil Mutlu[1]
[1]Department of Otorhinolaryngology, Kayseri Training and Research Hospital, Kayseri, Turkey
[2]Department of Otorhinolaryngology, Sorgun State Hospital, Yozgat, Turkey

European Archives of Oto-Rhino-Laryngology
https://doi.org/10.1007/s00405-021-06832-3

REVIEW ARTICLE

Clinical significance of video head impulse test in benign paroxysmal positional vertigo: a meta-analysis

Mayada Elsherif[1] · Dalia Eldeeb[2] · Mirhan Eldeeb[1]

ACTA OTO-LARYNGOLOGICA
https://doi.org/10.1080/00016489.2020.1805123

RESEARCH ARTICLE

Video head impulse testing in patients with benign paroxysmal positional vertigo

Ziya Saltürk and Sertaç Yetişer
ENT Department, Anadolu Health Center

ACTA OTORHINOLARYNGOLOGICA ITALICA 2021;41:69-76; doi: 10.14639/0392-100X-N1033

VESTIBOLOGY

The Video Head Impulse Test in the acute stage of posterior canal benign paroxysmal positional vertigo

Il Video Head Impulse Test nello stadio acuto della BPPV da canalolitiasi posteriore

Luigi Califano, Raffaella Iannella, Salvatore Mazzone, Francesca Salafia, Maria Grazia Melillo
San Pio Hospital, Department of Audiology and Phoniatrics, Benevento, Italy

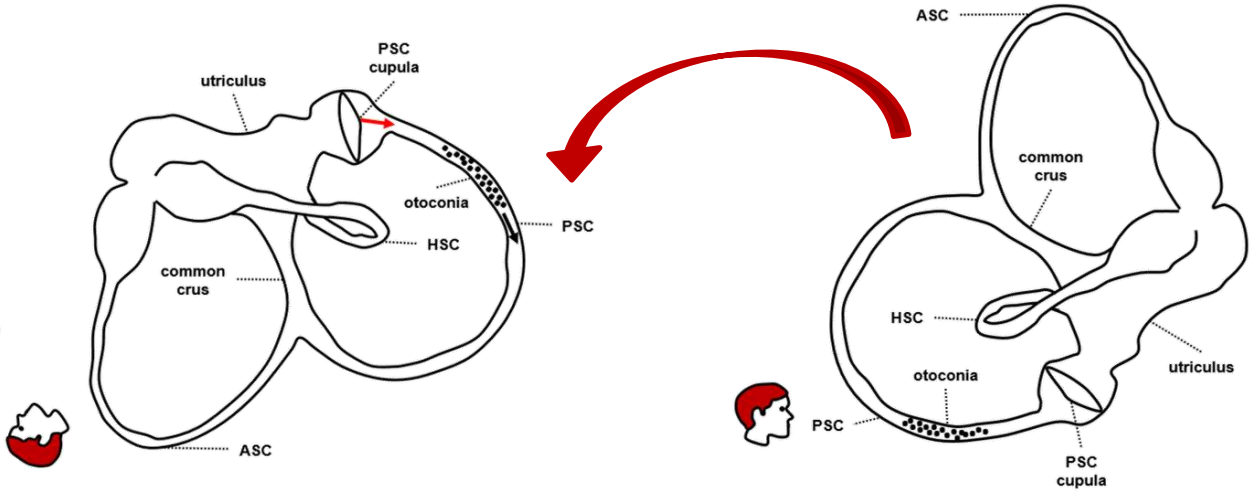
Indian J Otolaryngol Head Neck Surg
(August 2022) 74(Suppl 1):S506–S510; https://doi.org/10.1007/s12070-020-02351-5

ORIGINAL ARTICLE

The Role of Video Head Impulse Test (Vhit) in Diagnosing Benign Paroxysmal Positional Vertigo (BPPV)

Razna Abdulrahim[1] · B. Satheesh Kumar Bhandary[1] · A. Rajeshwary[1] · M. K. Goutham[1] · Vadisha Bhat[1] · Marina Saldanha[1]

- **In patients with typical PSC-BPPV,** affected canal **VOR gain is rarely impaired**
  - **No significant alterations** for the PSC involved at the vHIT in most cases as **free-floating particles do not alter high-frequency responses**



vHIT and BPPV with pDBN

Clinical Capsule Report

A Possible Role of Video-Head Impulse Test in Detecting Canal Involvement in Benign Paroxysmal Positional Vertigo Presenting With Positional Downbeat Nystagmus

*Andrea Castellucci, †Pasquale Malara, *Silvia Delmonte, and *Angelo Ghidini

*ENT Unit, Department of Surgery, Arcispedale Santa Maria Nuova, Azienda USL - Center for Clinical and Basic Research (IRCCS), Reggio Emilia, Italy; and †Audiology & Vestibology Service, Centromedico, Bellinzona, Switzerland

Original Study

Which Inner Ear Disorders Lie Behind a Selective Posterior Semicircular Canal Hypofunction on Video Head Impulse Test?

*†Andrea Castellucci, *‡Gianluca Piras, *§Valeria Del Vecchio, *Gian Gaetano Ferri, †Angelo Ghidini, and *Cristina Brandolini

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Clinical Capsule Report

Fluctuating Posterior Canal Function in Benign Paroxysmal Positional Vertigo Depending on How and Where Otoconia Are Disposed

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LETTER TO THE EDITOR

Check for updates

Possible pathomechanism behind the transient hypofunction of the affected canal in BPPV. Letter to the editor regarding "Clinical significance of video head impulse test in benign paroxysmal positional vertigo: a meta-analysis" by Elsherif M et al. European Archives of Oto-Rhino-Laryngology (2021);278(12):4645–4651

Andrea Castellucci[1] · Pasquale Malara[2] · Salvatore Martellucci[3] · Enrico Armato[4] · Luigi Califano[5]

- As already theorized, this condition could likely result in an **incomplete canalith jam (positional jam)** behaving as a **"low-pass filter":**
  - allowing the cupula to be activated by **low frequency stimuli** (**otoconial shifts**)
  - impeding the ampullary receptor to respond to **high-frequency inputs** (**head impulses**)

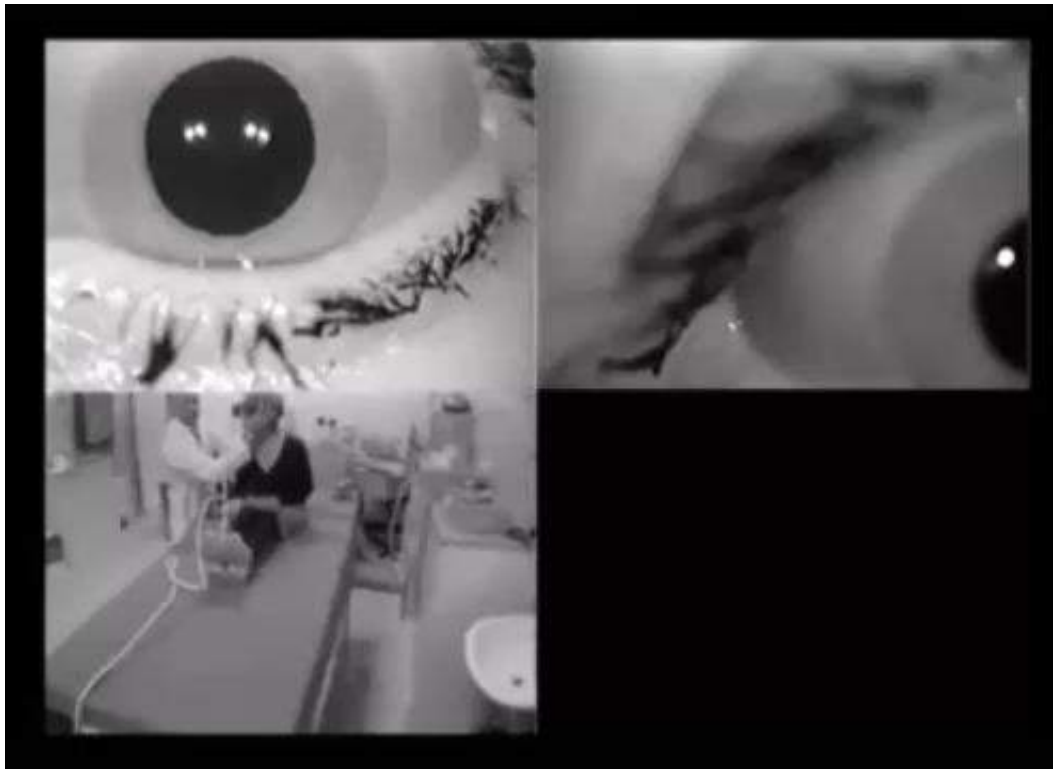
vHIT and BPPV with pDBN

- **Video-Frenzel evaluation**

o persistent pDBN with slight right torsional components

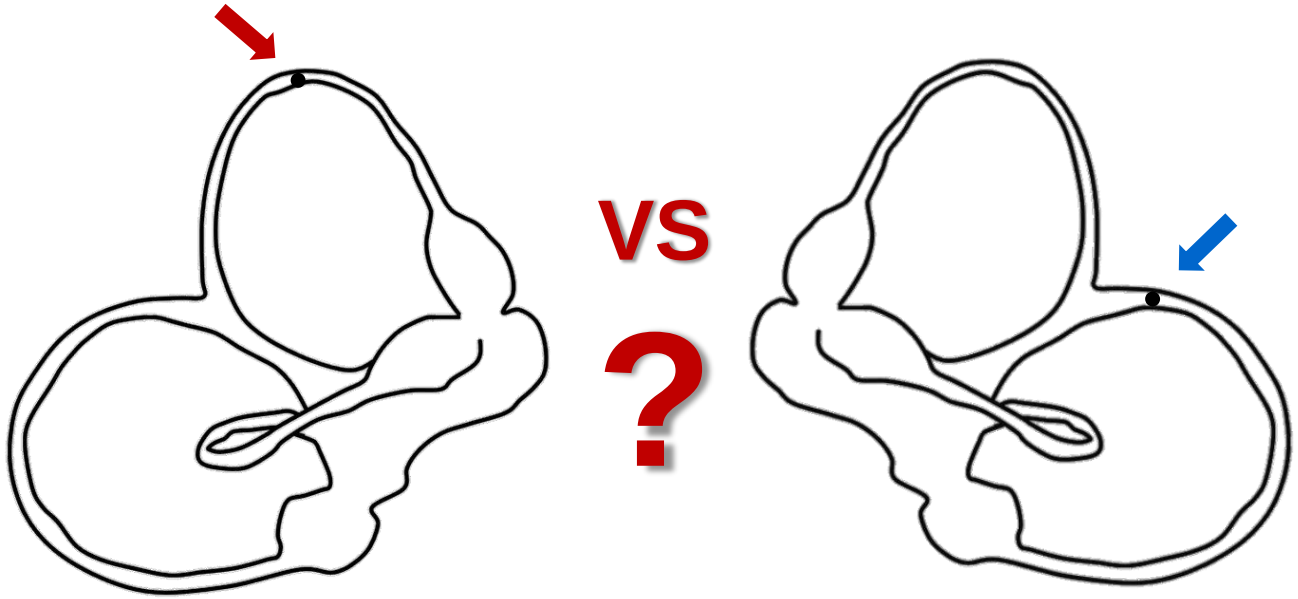
→ **Central pDBN?**

→ **Peripheral pDBN?**



Right ASC- BPPV

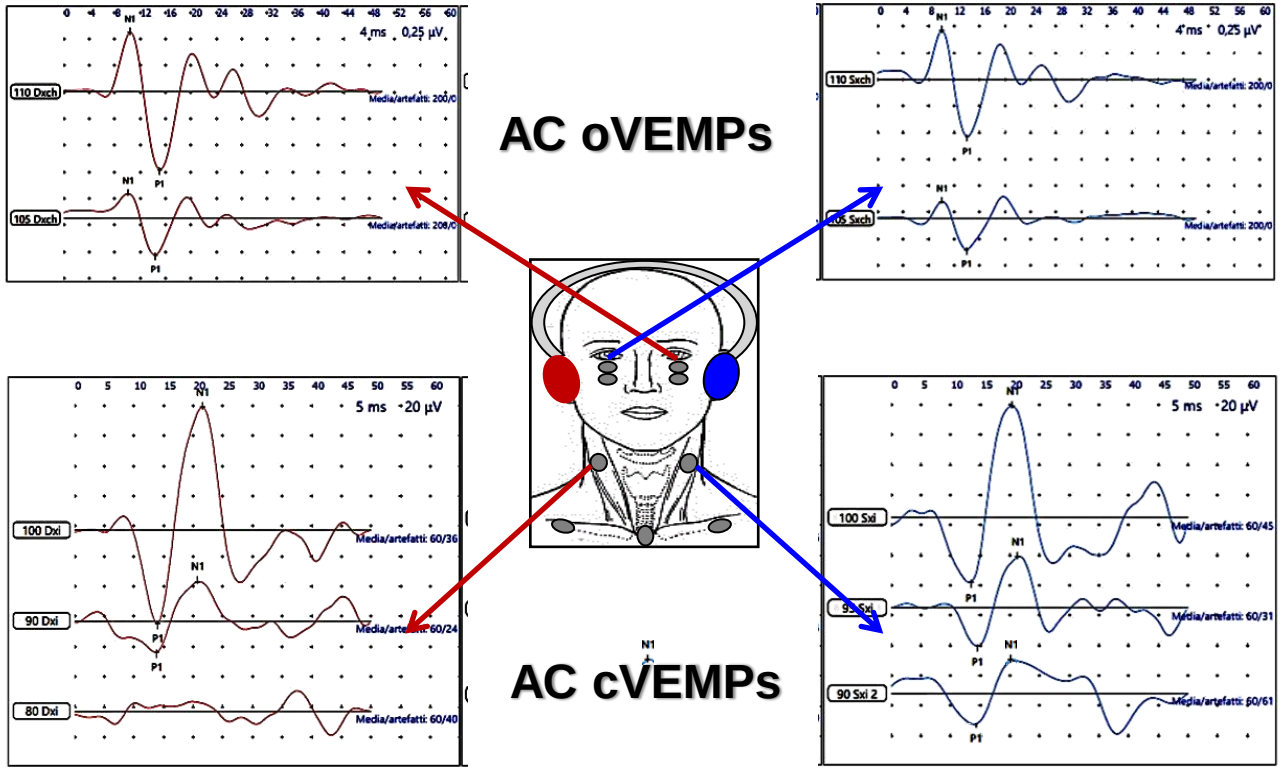
Left apogeotropic PSC-BPPV



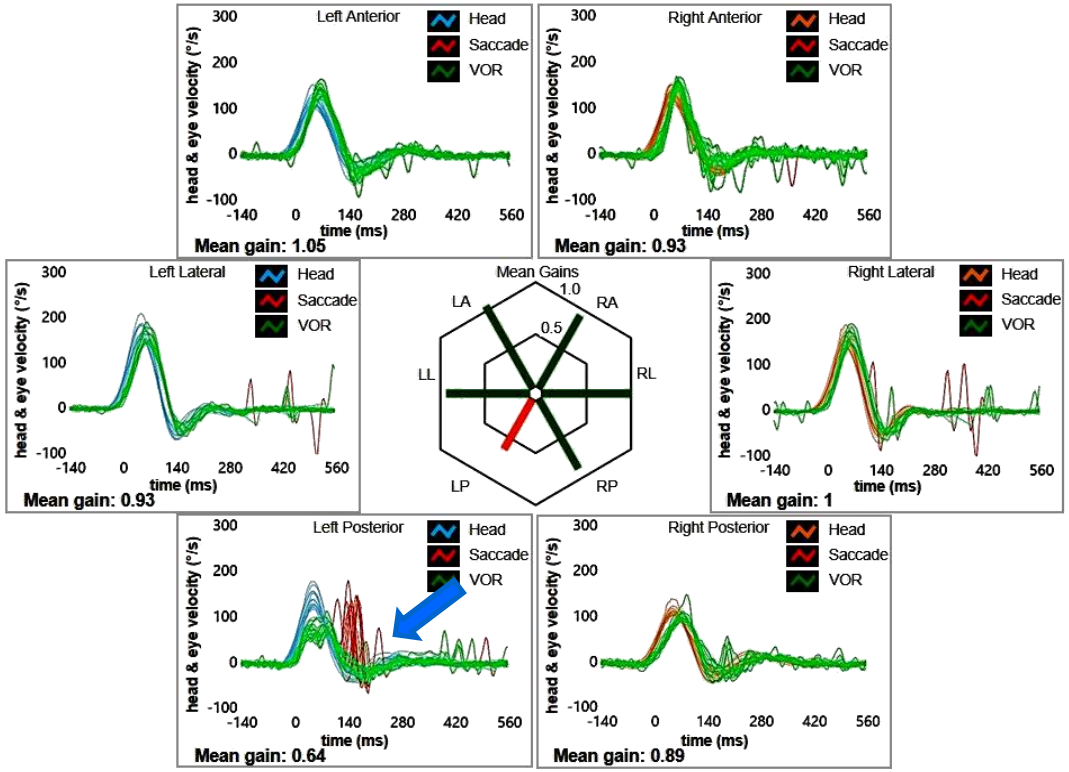
vHIT and BPPV with pDBN

- **INSTRUMENTAL ASSESSMENT**

o **AC cVEMPs: symmetrical** amplitudes
o **AC oVEMPs: symmetrical** amplitudes

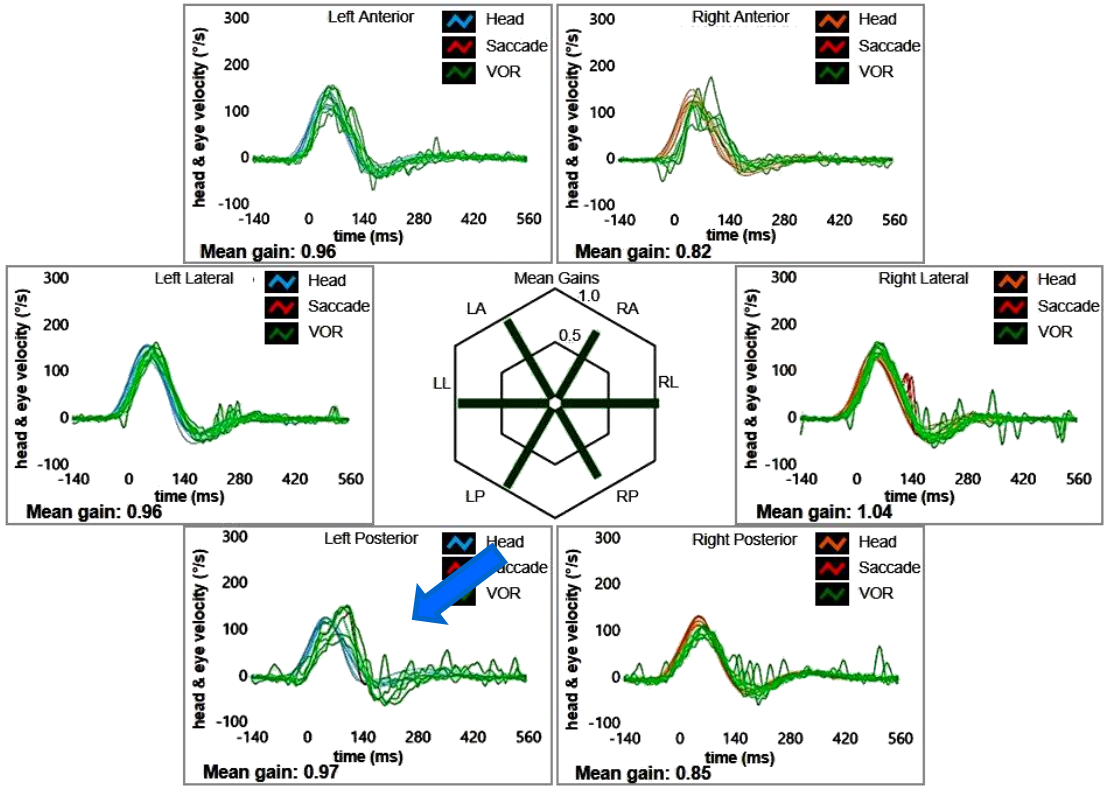


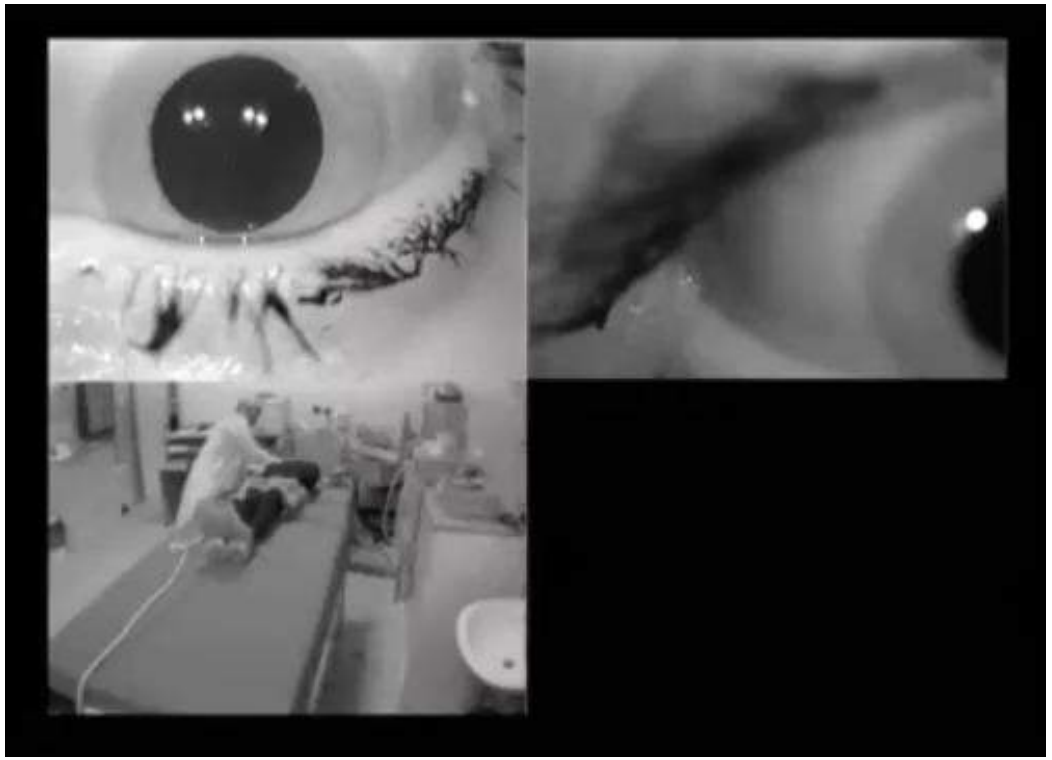
o **Video HIT: Selective left PSC hypofunction** (gain 0.64)



vHIT and BPPV with pDBN

- **INSTRUMENTAL ASSESSMENT after conversion** in **paroxysmal positional upbeat/left torsional nystagmus** during reversed Epley's CRM due to involvement of **left PSC ampullary arm**





- **Video HIT: Normalization of left PSC VOR-gain** (gain 0.97)

vHIT and BPPV with pDBN

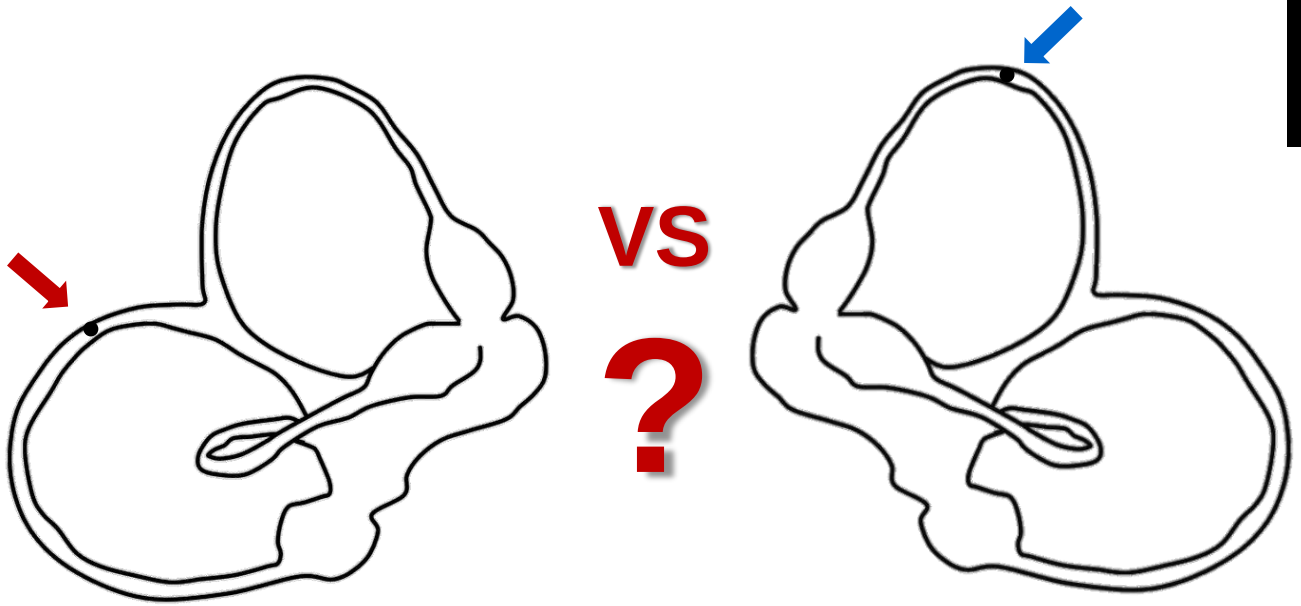
- **Video-Frenzel evaluation**

o persistent pDBN with slight left-torsional components

→ **Central pDBN?**

→ **Peripheral pDBN?**

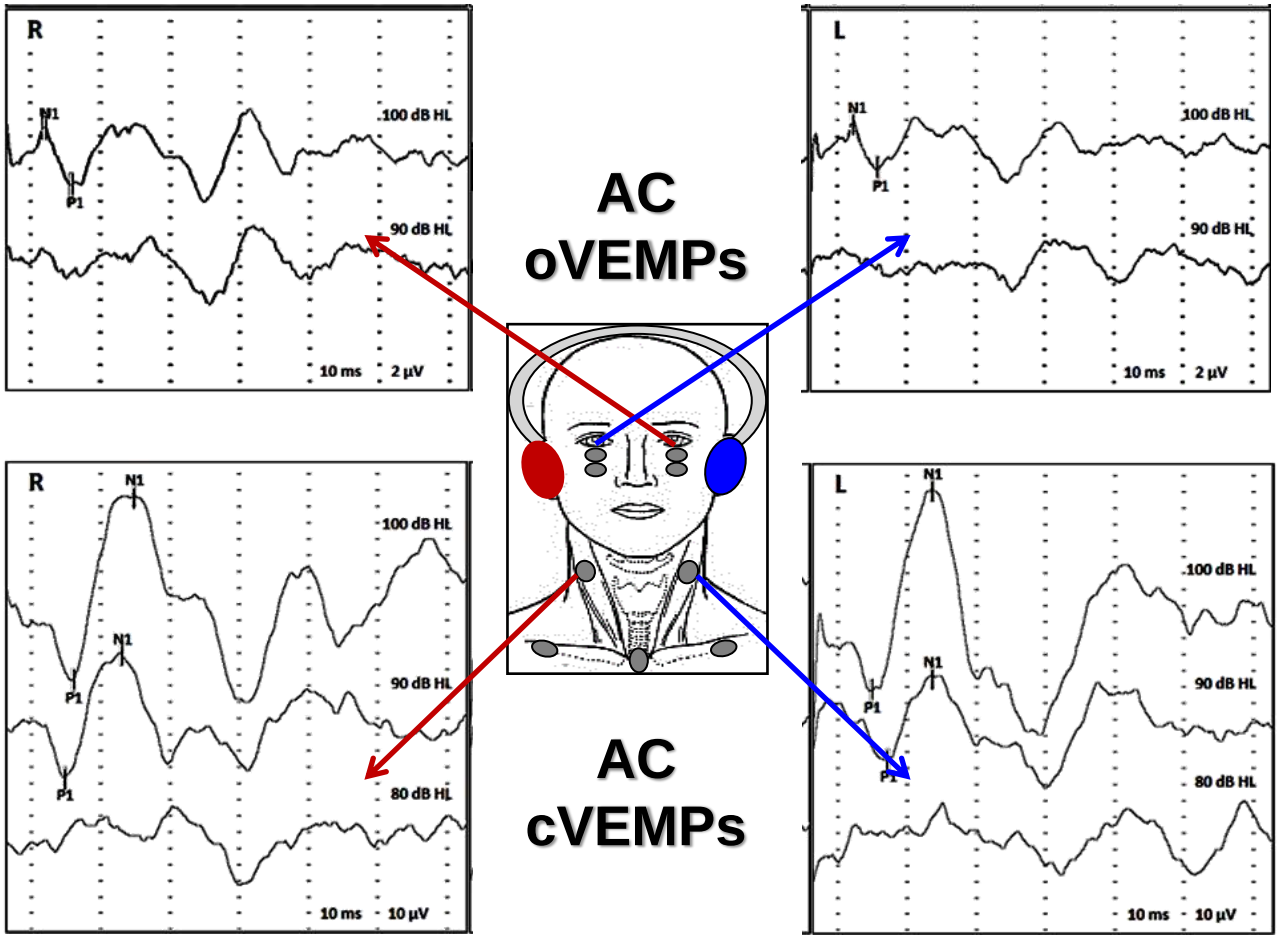
Right apogeotropic PSC- BPPV **Left ASC-BPPV**



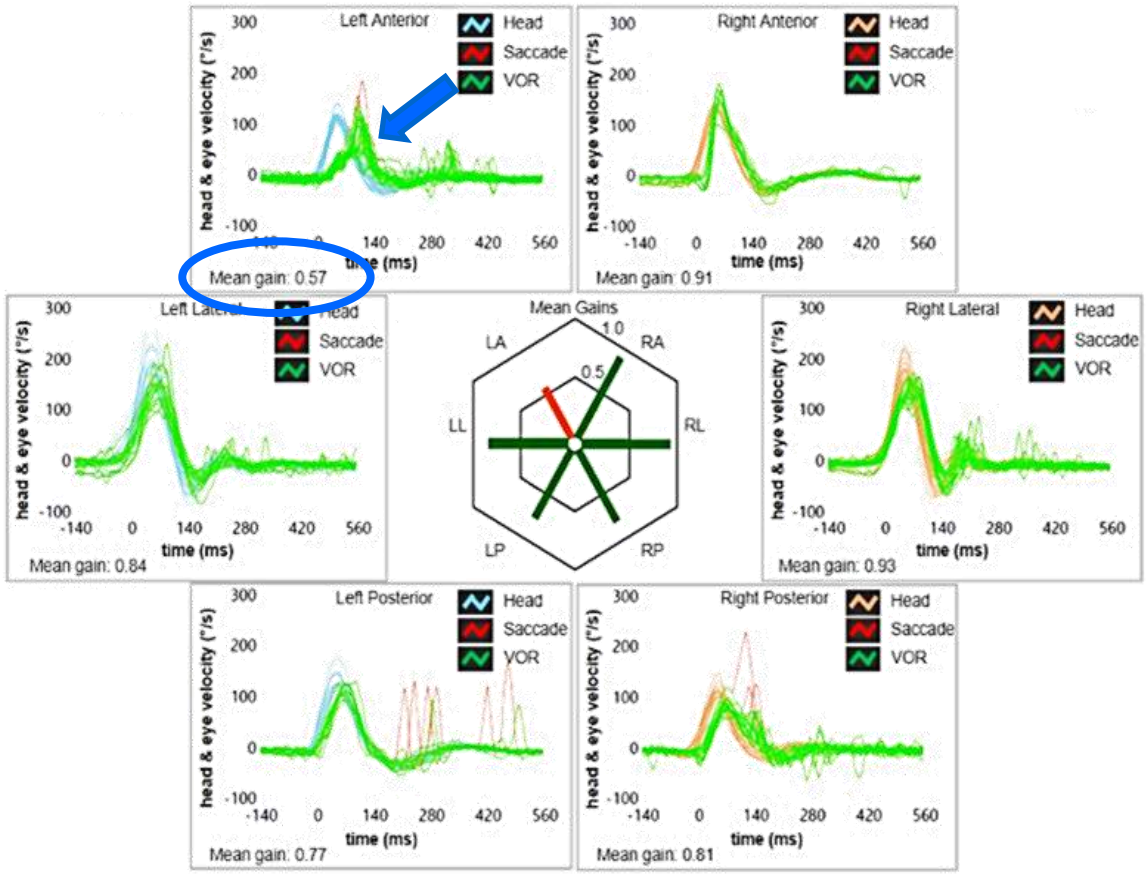
vHIT and BPPV with pDBN

- **INSTRUMENTAL ASSESSMENT**

  - **AC cVEMPs:** symmetrical amplitudes and thresholds
  - **AC oVEMPs:** symmetrical amplitudes and thresholds

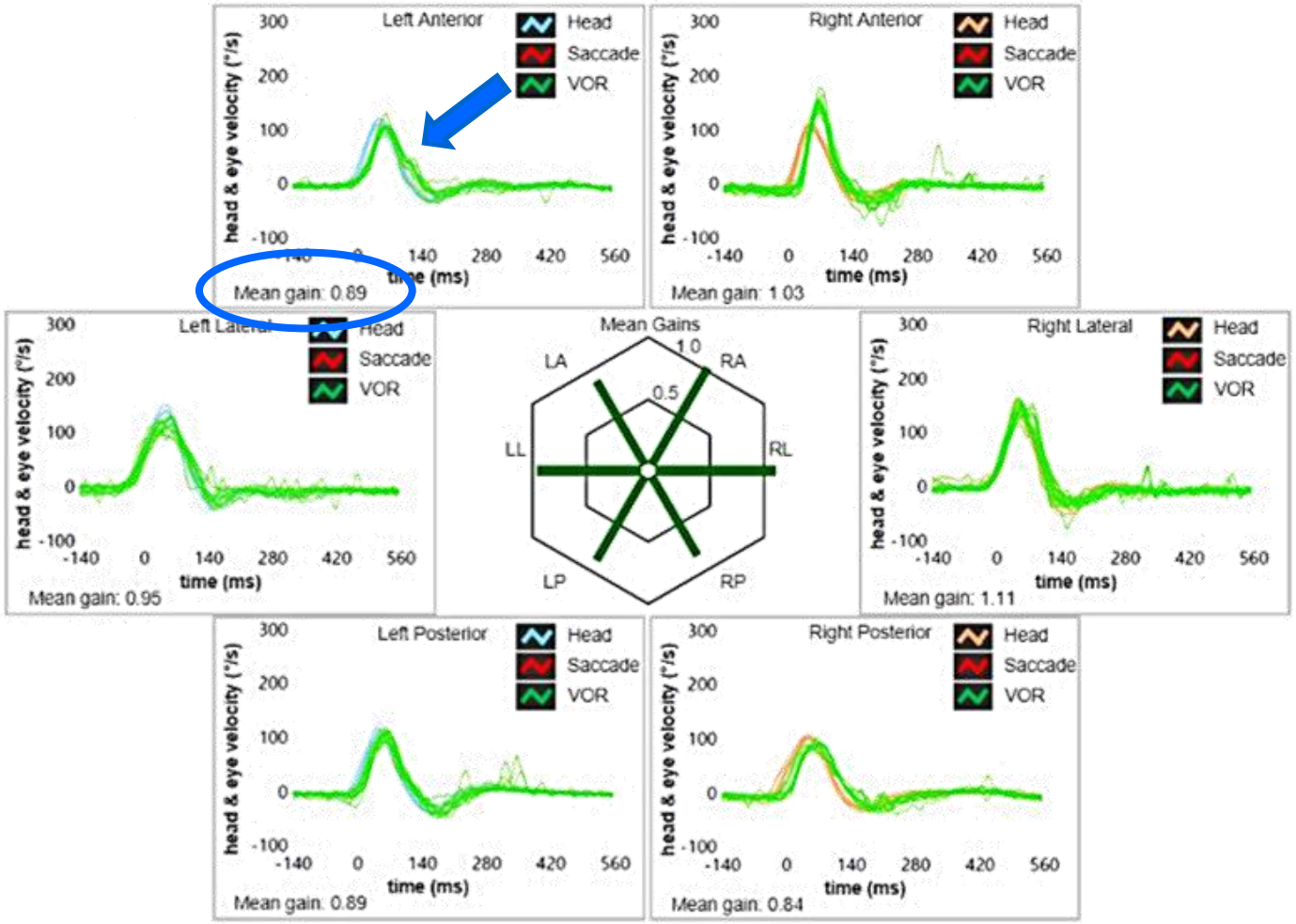


- **Video HIT: selective left ASC loss** (VOR-gain 0.57)



vHIT and BPPV with pDBN

- **INSTRUMENTAL ASSESSMENT after CRM** (Yacovino)
  - **Video HIT: normalization of left ASC VOR-gain** (gain 0.89)



vHIT and BPPV with pDBN

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informa
healthcare

ORIGINAL ARTICLE

Vestibulo-ocular reflex in patients with superior semicircular canal benign paroxysmal positional vertigo (BPPV)

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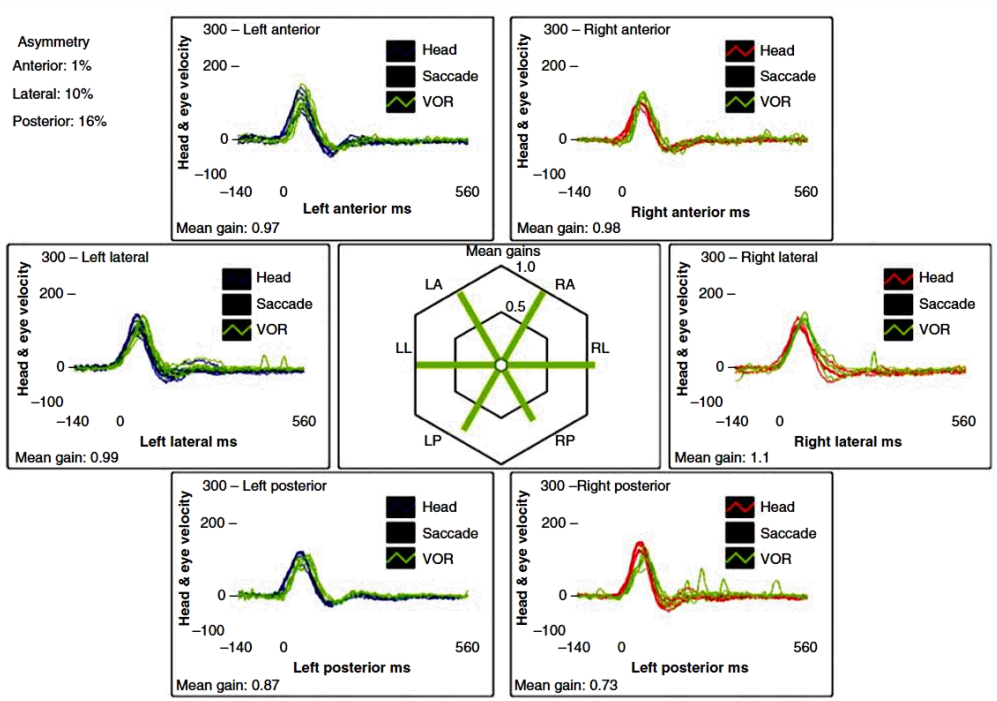
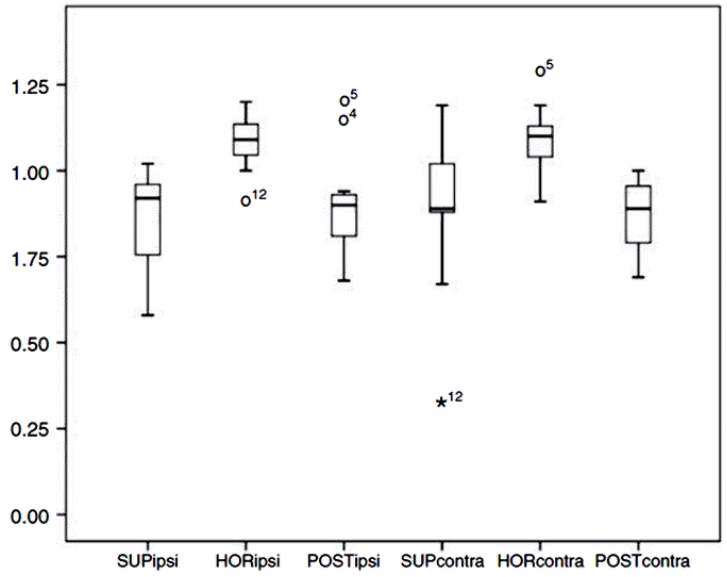


- **12 patients with ASC-BPPV**
- All exhibited **pDBN with torsional component** in provoking positions
- The **mean VOR-gain was within normal limits for the 6 semicircular canal** including the involved ASC
- In each case **nystagmus exhibited a crescendo-decrescendo pattern**
- **mean duration of nystagmus: 51 ± 28 s**



vHIT and BPPV with pDBN

frontiers in Neurology

ORIGINAL RESEARCH
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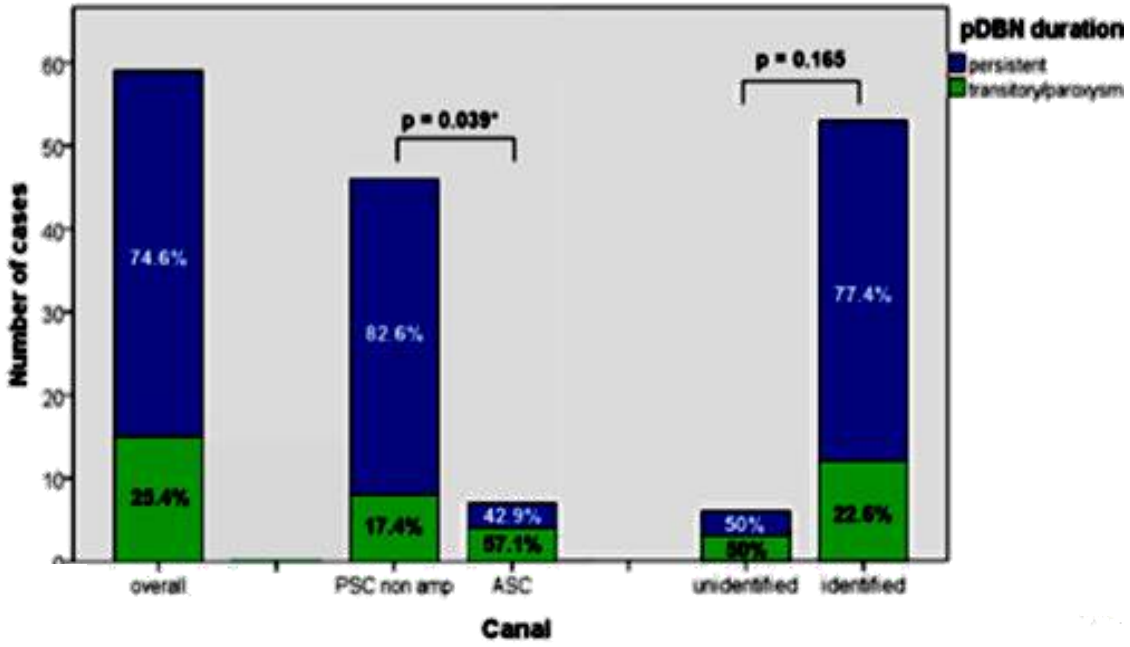
Feasibility of Using the Video-Head Impulse Test to Detect the Involved Canal in Benign Paroxysmal Positional Vertigo Presenting With Positional Downbeat Nystagmus

Andrea Castellucci[1†], Pasquale Malara[2†], Salvatore Martellucci[3†], Cecilia Botti[1,4], Silvia Delmonte[1], Silvia Quaglieri[5], Elisabetta Rebecchi[6], Enrico Armato[7], Massimo Ralli[8,9], Marco Lucio Manfrin[5], Angelo Ghidini[1] and Giacinto Asprella Libonati[10]*

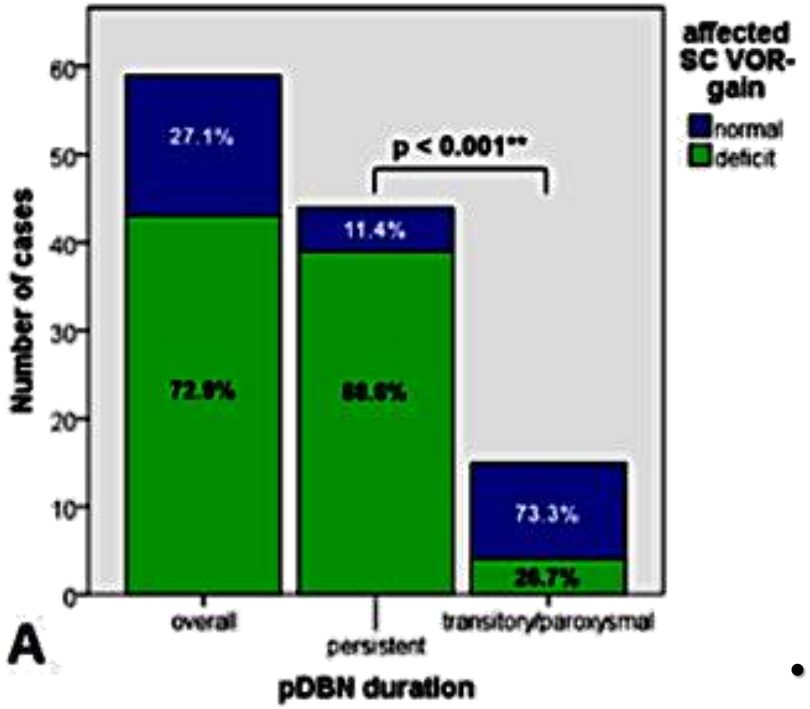


- **59 patients with peripheral pDBN** submitted to **vHIT before and after CRM** resulting either in **symptoms receding or transformation in a typical BPPV variant.**

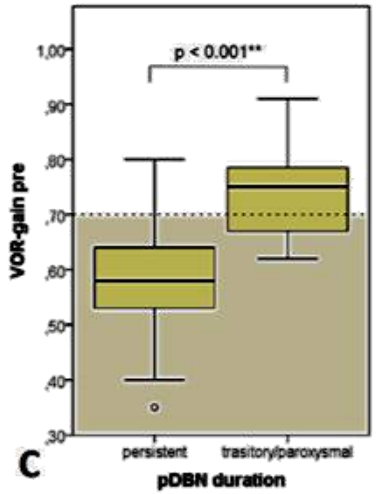
  - **Apogeotropic PSC-BPPV**: 46 cases (78%)
  - **ASC-BPPV**: 7 cases (11.9%)
  - Unidentified canal: 6 cases (10.1%)

- In most cases **nystagmus was persistent (> 2 min)** with a significant prevalence among apogeotropic PSC-BPPV

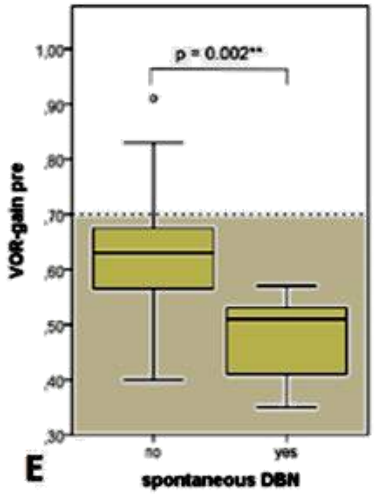
vHIT and BPPV with pDBN



- **VOR-gain of the affected canal** was **impaired** in 43/59 cases **(vHIT sensitivity: 72.9%)**
- **Considering only patients with <u>persistent</u> pDBN**, VOR-gain of the affected canal was impaired in 39/44 cases **(vHIT sensitivity: 88.6%)**
- **Considering patients with <u>paroxysmal</u> pDBN (vHIT sensitivity: 26.7%)**

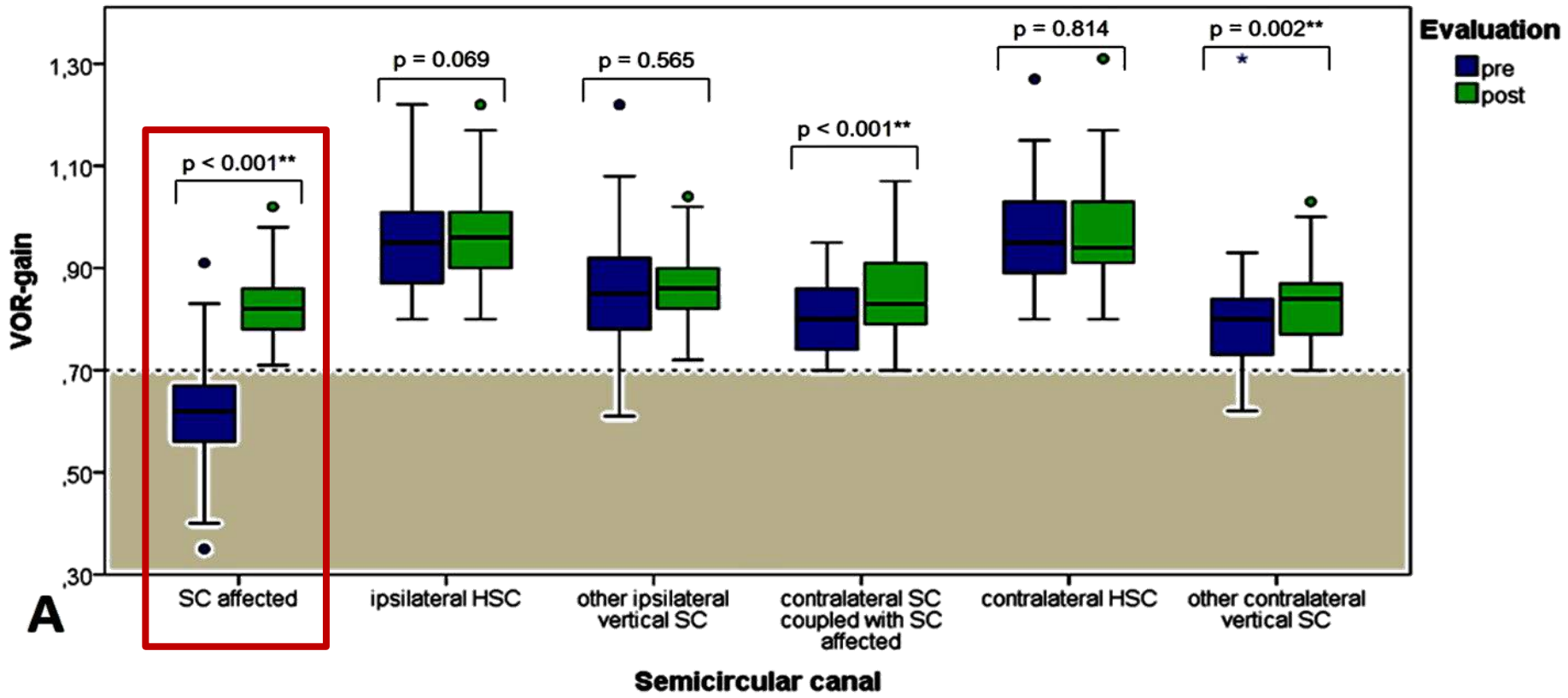


- **VOR-gain of the affected canal** was significantly **<u>more impaired</u>** in case with **<u>persistent</u> pDBN** compared to paroxysmal pDBN



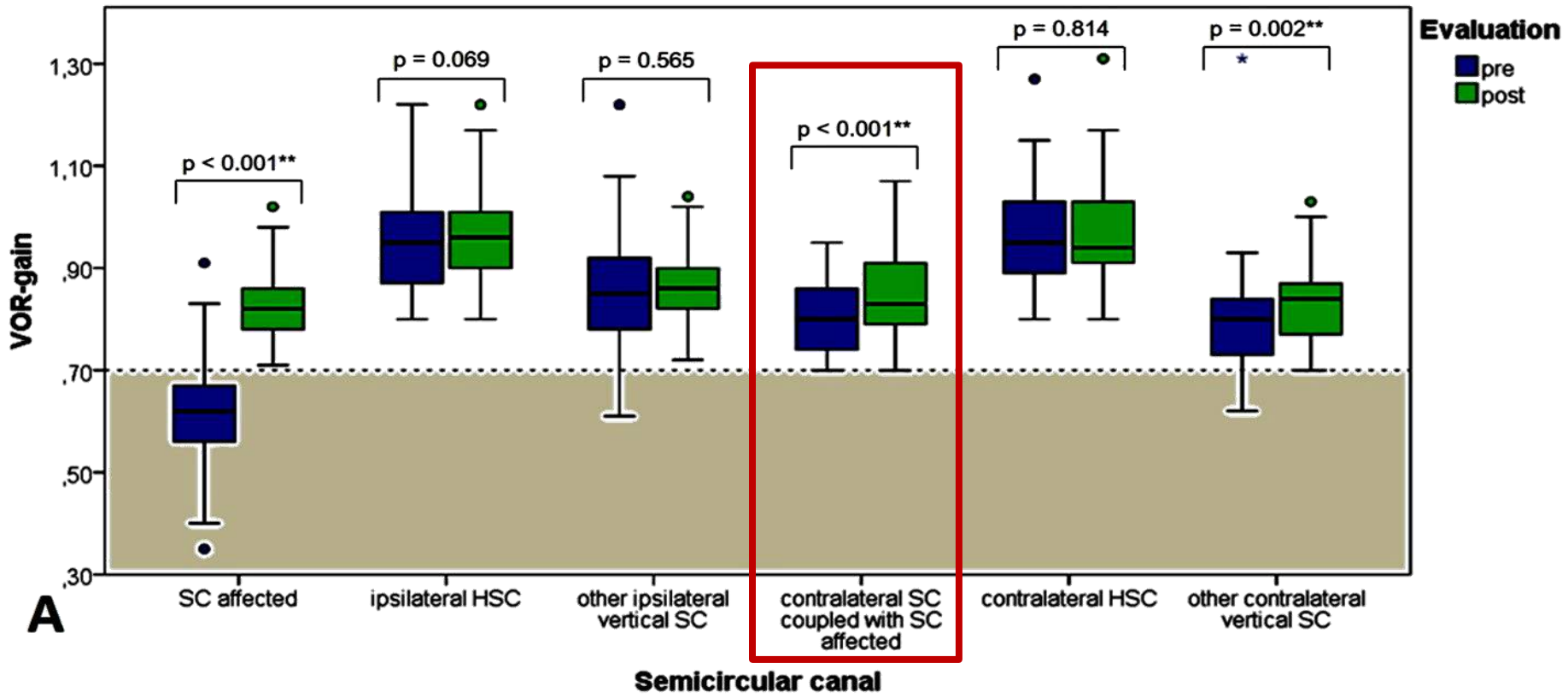
- **VOR-gain of the affected canal** was significantly more impaired **in cases of <u>spontaneous</u> DBN** where it is possible to assume a **complete canalith jam entirely plugging the canal lumen**

vHIT and BPPV with pDBN



- **Presenting impaired VOR-gain values for the affected canal normalized** after symptoms receding or conversion into a typical BPPV-form, confirming a **strong relationship between lithiasis and altered cupular dynamics**.

vHIT and BPPV with pDBN



- Also **VOR-gain values for the contralateral vertical canal functionally coupled with the involved canal significantly improve after CRM**, despite presenting VOR-gain was within normal ranges.
  - **peripheral phenomena** (functional **loss of the "push-pull" mechanism**) → could likely explain this phenomena among cases with **recent onset** of BPPV
  - **central compensation** (**cerebellar "shut-down"**) → may likely explain reduced VOR-gain values in those patients with **long-lasting symptoms**

VOR fluctuations in BPPV

- **54-year-old female**
- Recurrence of **positional vertigo with nausea**, **progressively turned in chronic disequilibrium**
- Arterial hypertension
- Menopause for 3 years
- **Not-disabling pulsatile tinnitus on the right side**
- **History of recurrent BPPV treated elsewhere**
- Bedside oculomotor test: negative
- Neurologic evaluation: negative

Clinical Capsule Report

Fluctuating Posterior Canal Function in Benign Paroxysmal Positional Vertigo Depending on How and Where Otoconia Are Disposed

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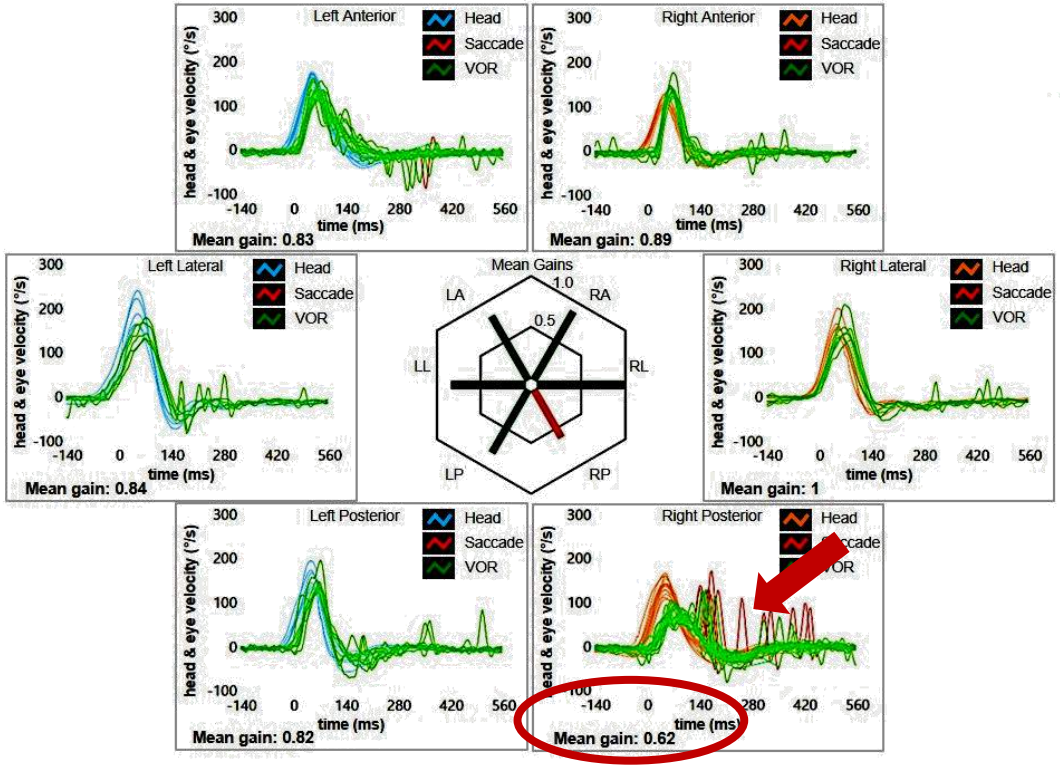
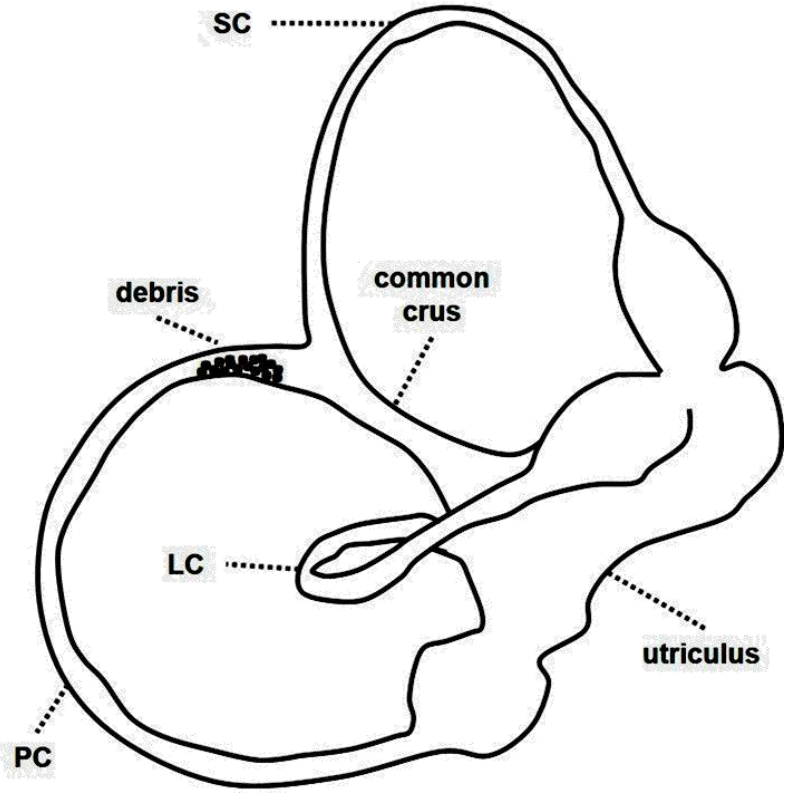
VOR fluctuations in BPPV

- **Video HIT: selective right PSC loss** (gain 0.62)





- We assumed that debris were **partially entrapped in the non-ampullary arm** of the right PSC resulting in **persistent pDBN** and **slight high-frequency VOR impairment**

VOR fluctuations in BPPV

- **Video-Frenzel evaluation** → Demi Semont maneuver

VOR fluctuations in BPPV

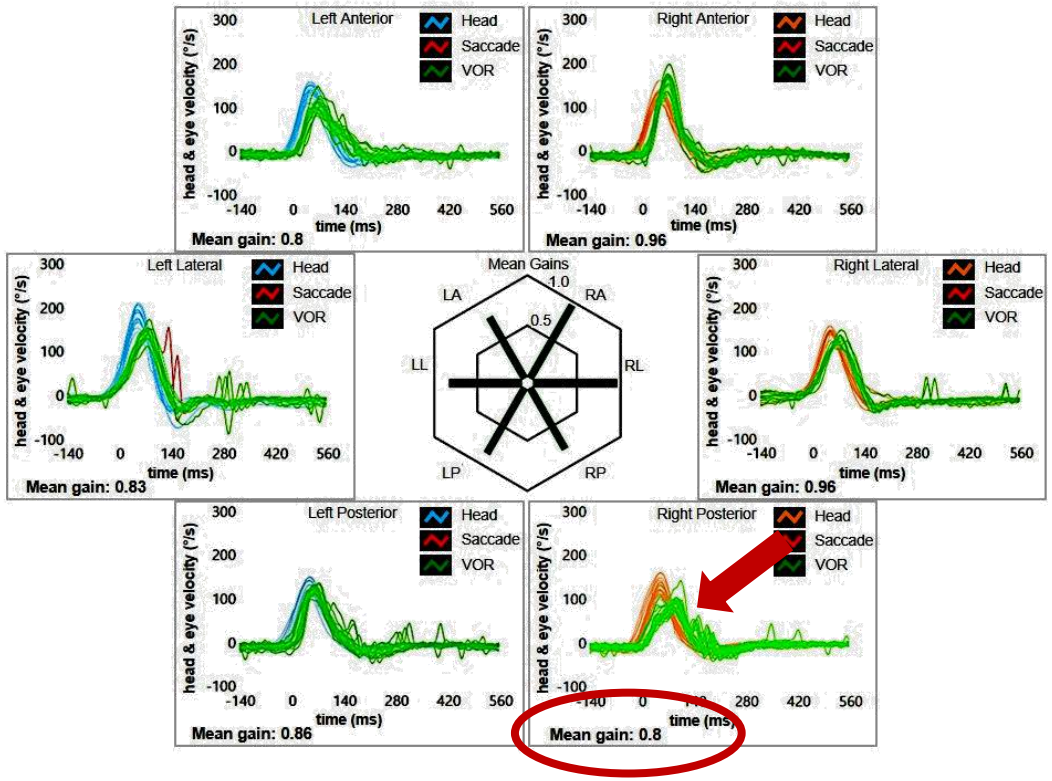
- **Video-Frenzel evaluation** → Demi Semont maneuver

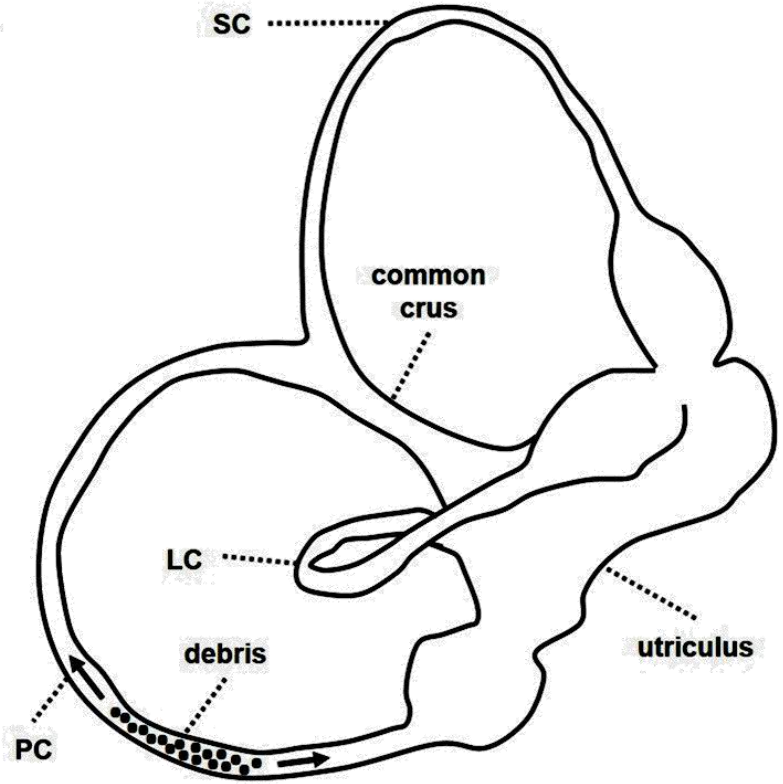
- **Video-Frenzel evaluation at 3 days:** persistence of symptoms

VOR fluctuations in BPPV

- **Conversion in typical geotropic PSC-BPPV**

o **Video HIT: normalization of right PSC VOR gain** (gain 0.8)





o Debris were **free to move back and forth along the ampullary arm** of the canal **without impacting on high-frequency VOR dynamics**

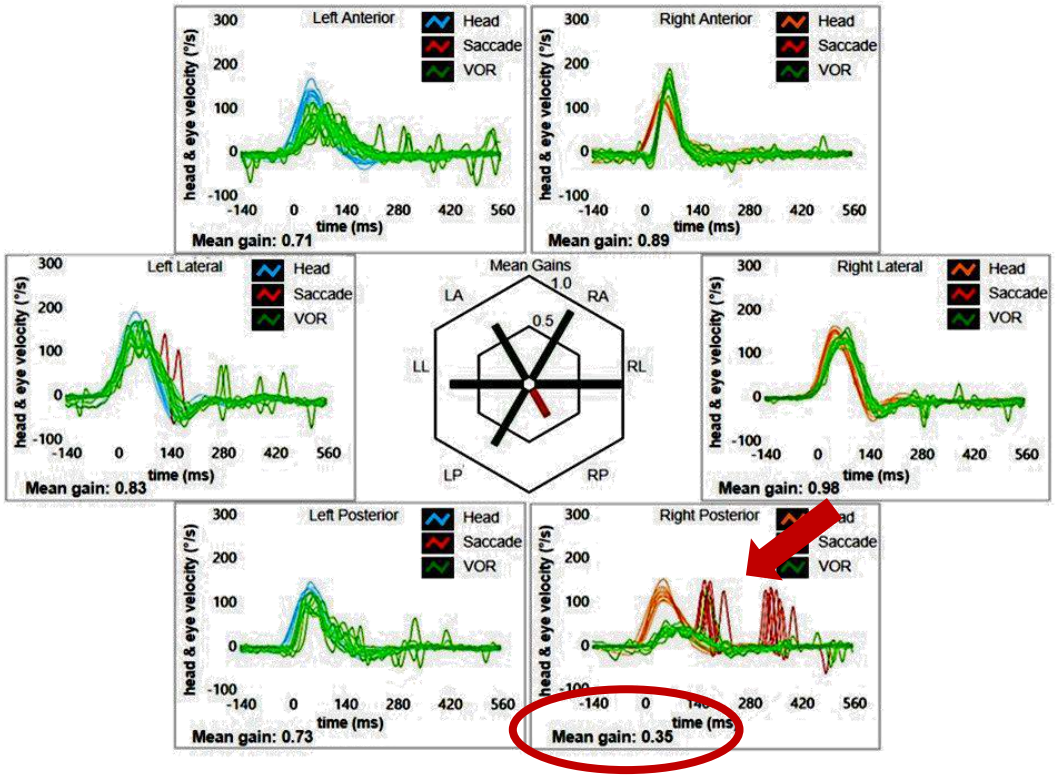
VOR fluctuations in BPPV

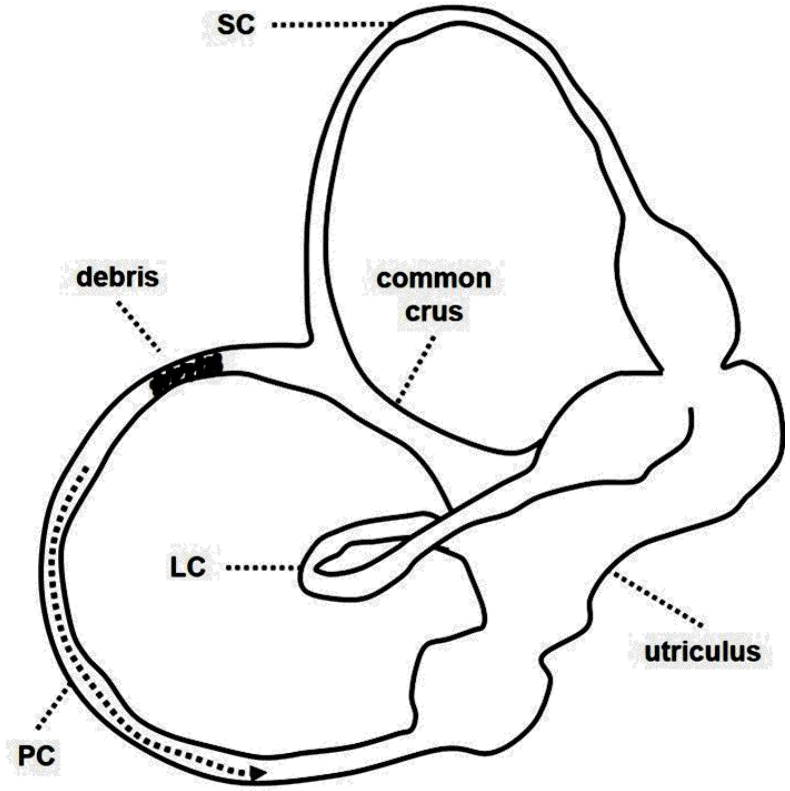
- **Video-Frenzel evaluation 5 days later:** severe worsening of symptoms with the onset of disabling dizziness

VOR fluctuations in BPPV

- **PSC canalith jam**

o **Video HIT: new severe impairment of PSC VOR gain** (gain 0.35)





o Debris failed to return to the utricle and were **completely blocked within a narrow portion of PSC non-ampullary arm plugging the canal** → **severely impairing VOR dynamics by exerting a continuous positive pressure on the cupula**

VOR fluctuations in BPPV

- **Video-Frenzel evaluation** → Demi Semont maneuver

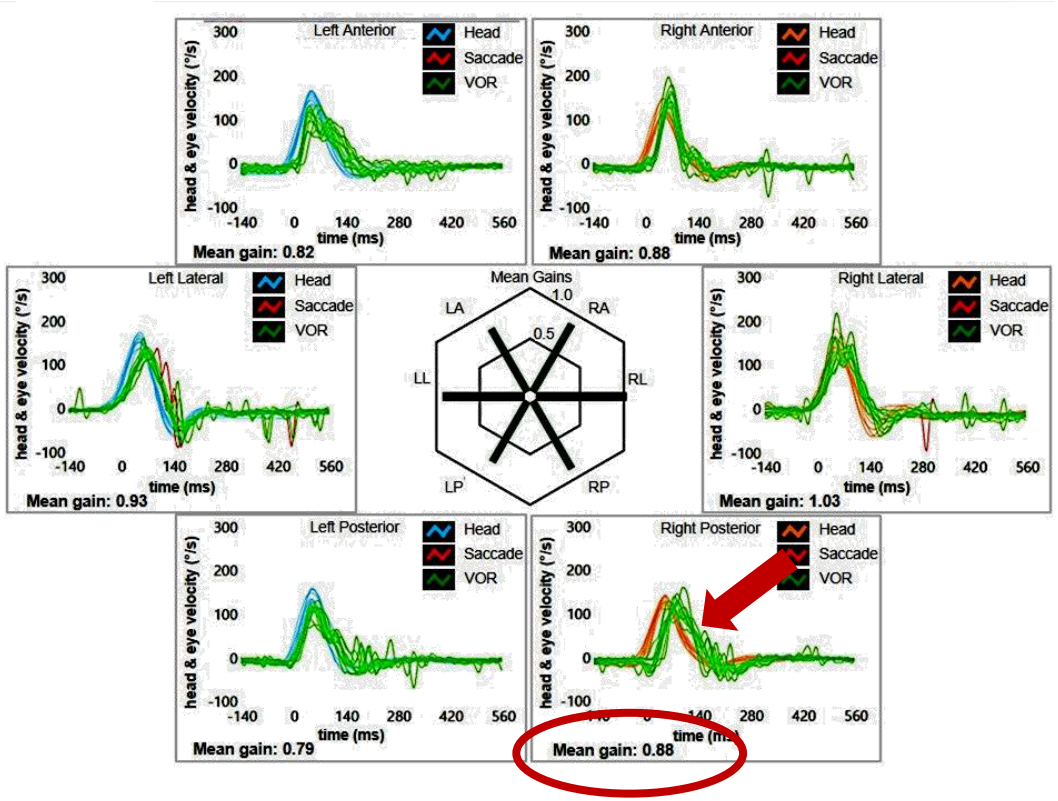
VOR fluctuations in BPPV

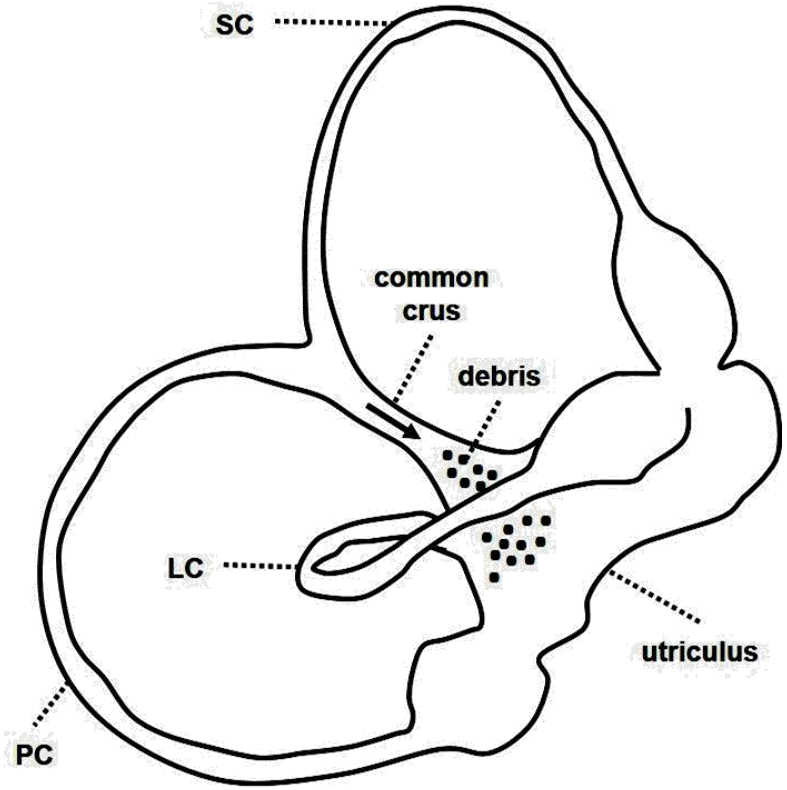
- **Video-Frenzel evaluation 4 days later:** symptoms resolution

VOR fluctuations in BPPV

- **Debris are back to the utricle**

o **Video HIT: normalization of PSC VOR gain** (gain 0.88)





o Debris were **finally back to the utricle** after CRM, **VOR dynamics are newly restored**

vHIT and BPPV with spontaneous reversal ny

- **HISTORY**

o 57-year-old female

o **Multiple Sclerosis** in follow up since 15 years

o Recurrent PSC - BPPV

o **Recurrence of positional vertigo since few weeks**

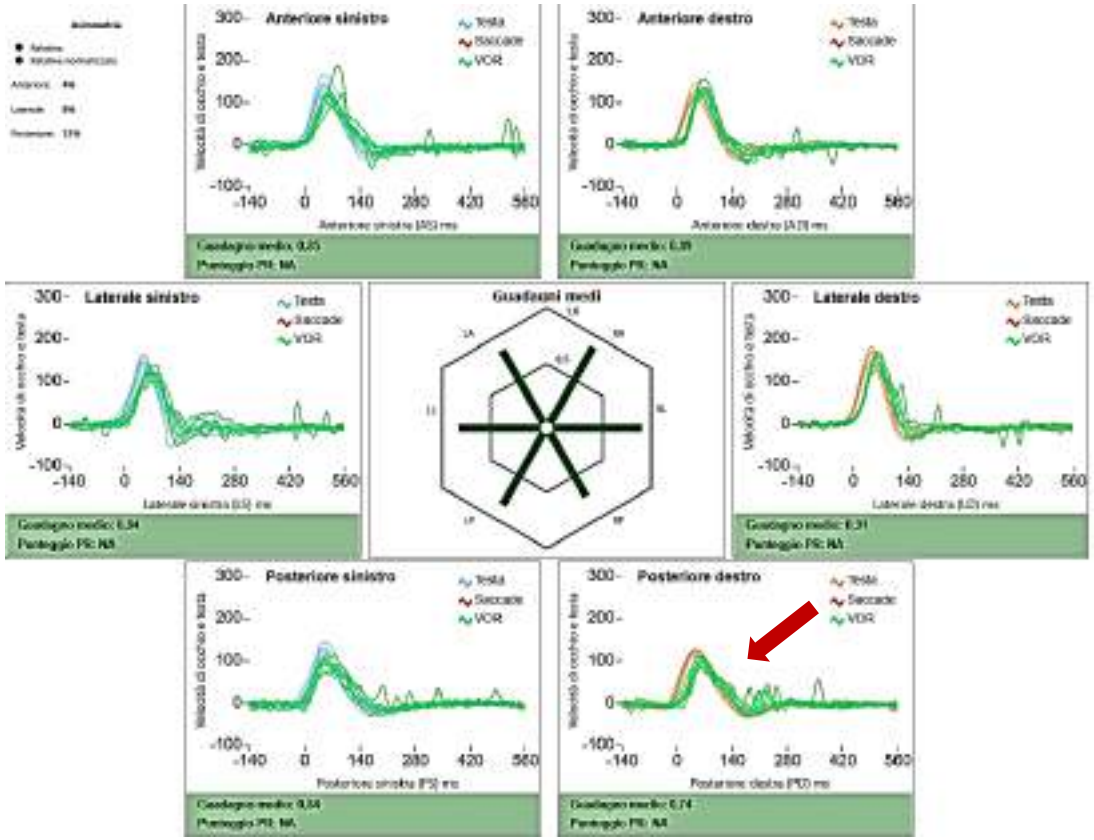
o No auditory symptoms

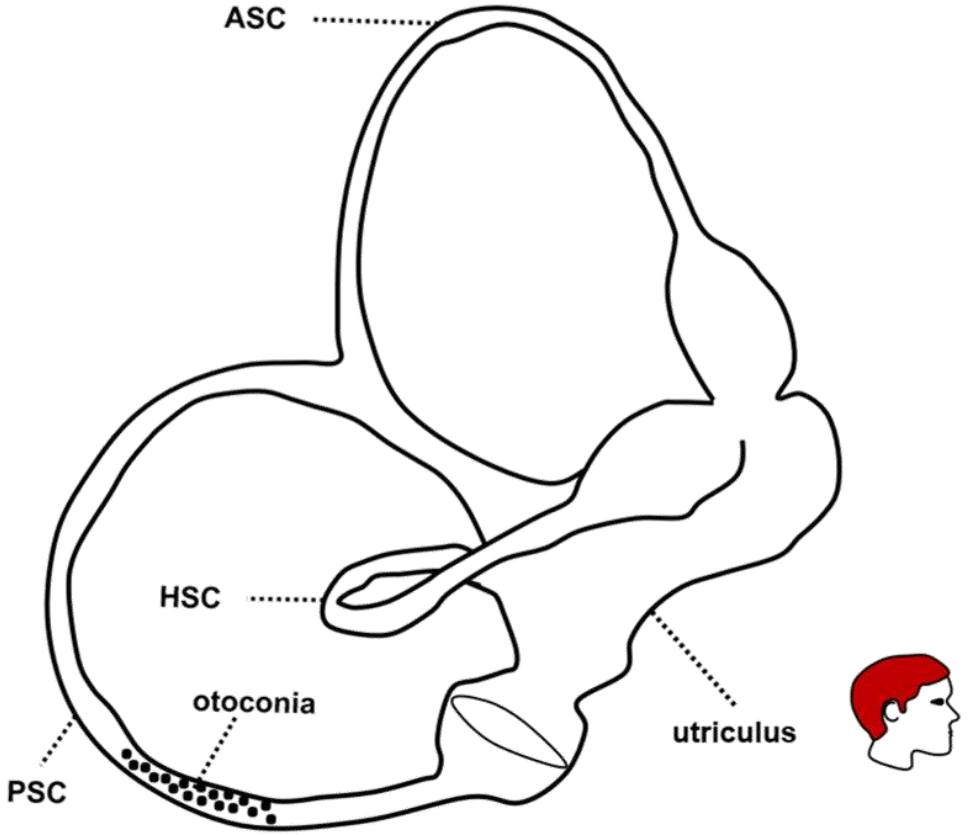
o **MRI: posterior fossa free from MS lesions**

o Bedside oculomotor test: negative

vHIT and BPPV with spontaneous reversal ny

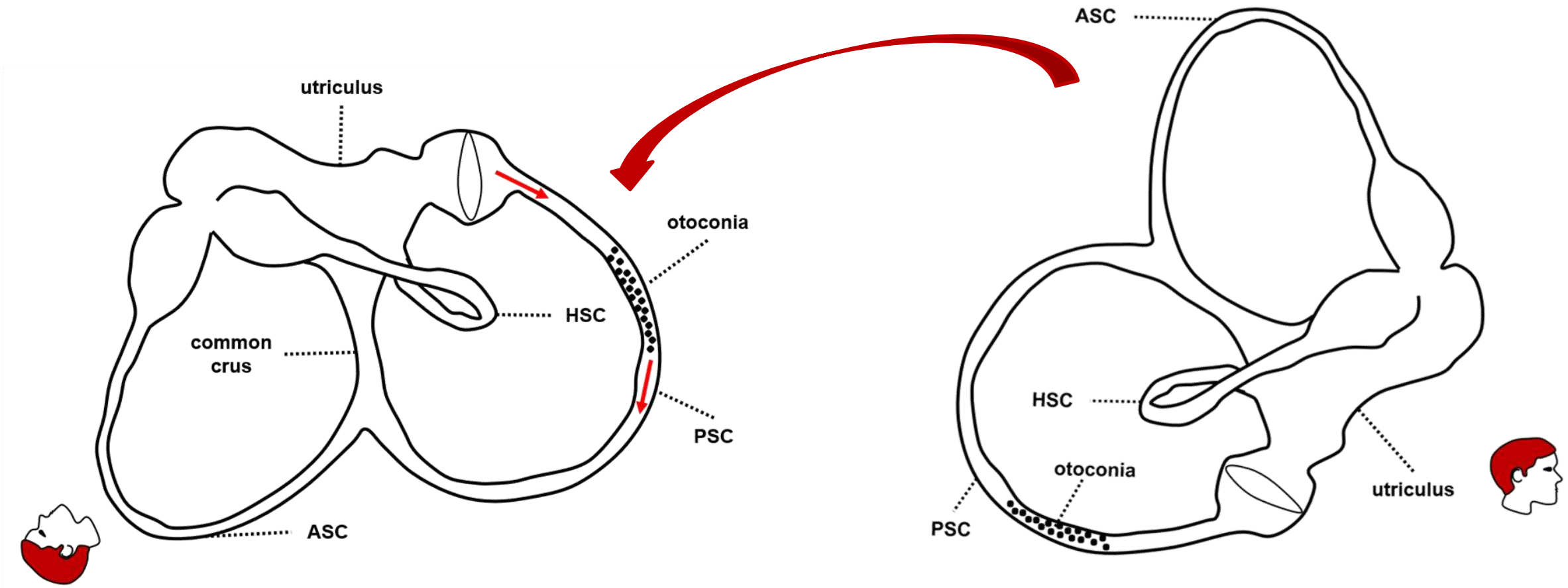
- **Video HIT: normal right PSC VOR gain** (gain 0.74)





o debris are **free to move back and forth along the ampullary arm** of the right PSC, **without impacting on high-frequency VOR dynamics**

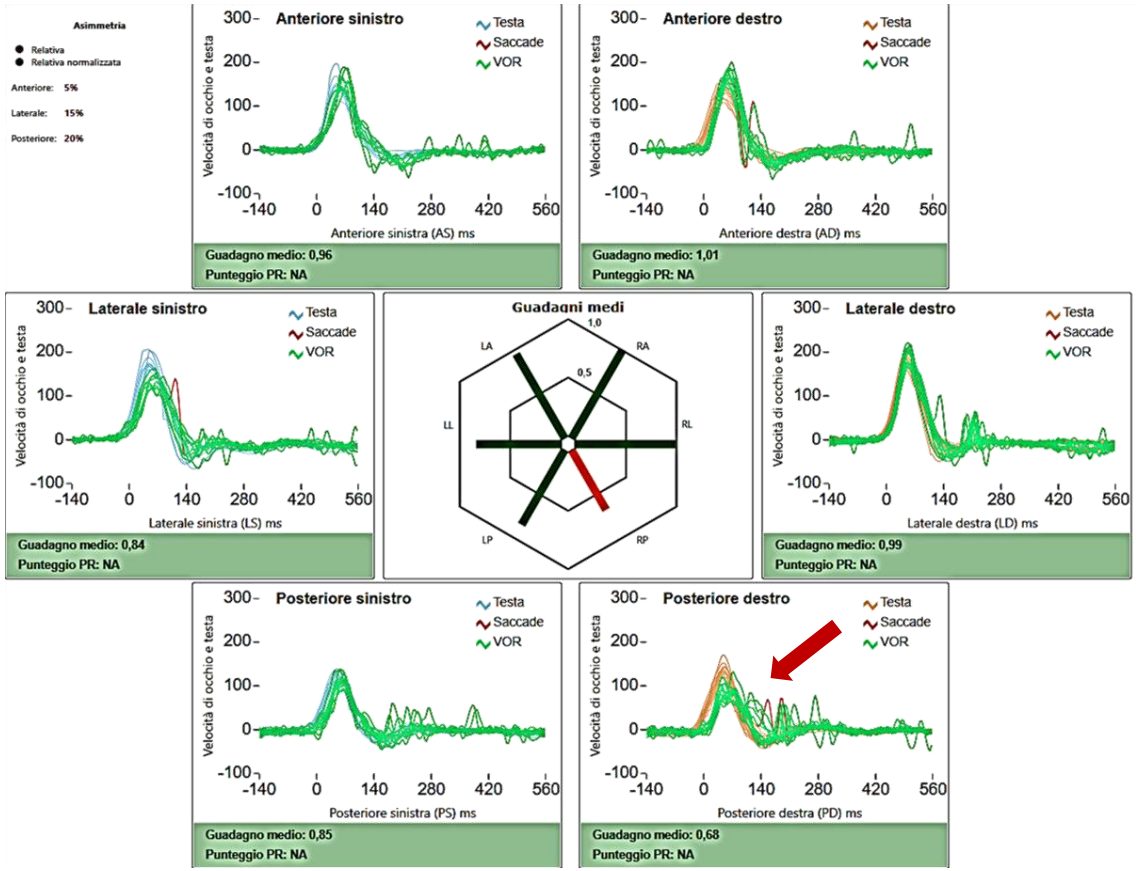
vHIT and BPPV with spontaneous reversal ny



- debris are **free to move back and forth along the ampullary arm** of the right PSC, **without impacting on high-frequency VOR dynamics**
- **free-floating debris in the ampullary arm** of the canal **move away from the ampulla**, resulting in **transient excitation of the PSC afferents and in paroxysmal upbeat/right-torsional nystagmus**

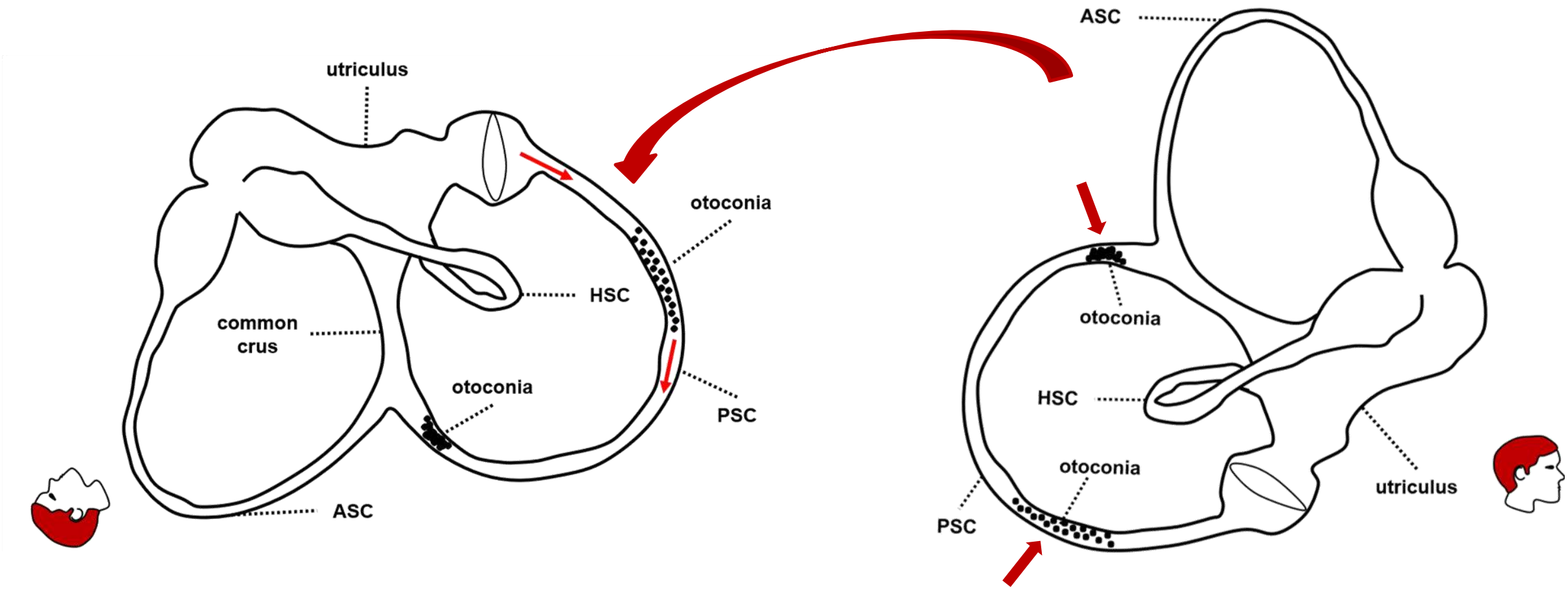
vHIT and BPPV with spontaneous reversal ny

- **Evaluation after 5 days:** persistence of symptoms
- **Video HIT:** **reduced PSC VOR gain** (gain 0.68)



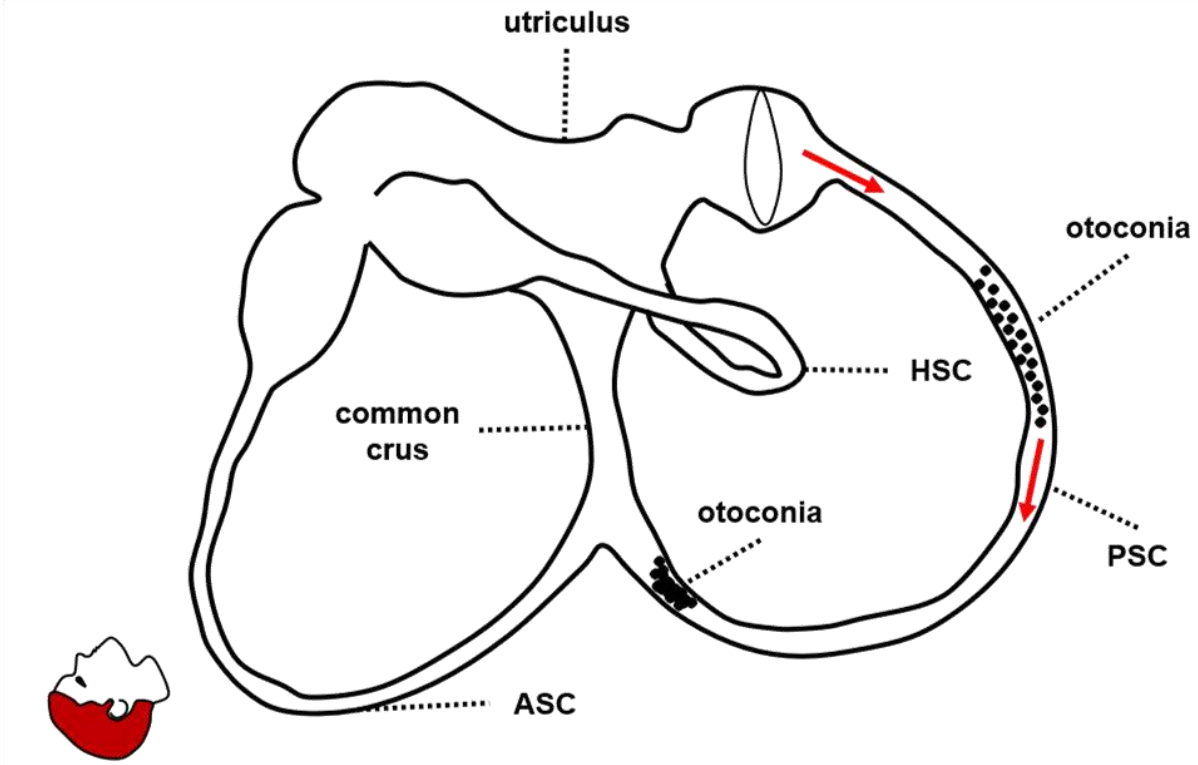
- **First:** **transient excitation of the PSC afferents** with paroxysmal upbeat/right-torsional nystagmus
- **Then:** **spontaneous reversal nystagmus** with persistent positional DBN and left-torsional components

vHIT and BPPV with spontaneous reversal ny



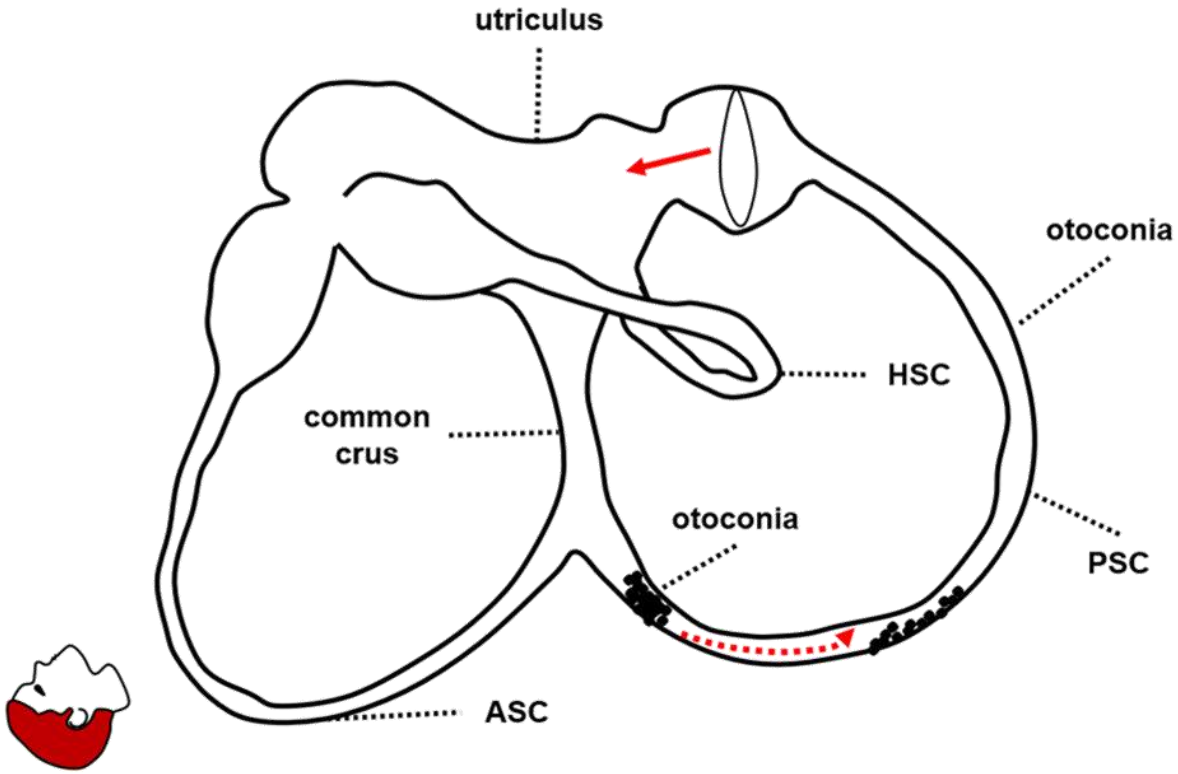
- 2 separate groups of otoconia:
  - **ampullary arm**: debris are **free to move back and forth along the ampullary arm** of the canal, **without impacting on high-frequency VOR dynamics**
  - **non-ampullary arm:** debris were **partially entrapped in the non-ampullary arm** of the right PSC resulting in **slight high-frequency VOR impairment**

vHIT and BPPV with spontaneous reversal ny



- In provoking position:
  - First, **free-floating debris in the ampullary arm** of the canal **move away from the ampulla**, resulting in **transient excitation of the PSC afferents and in paroxysmal upbeat/right-torsional nystagmus**

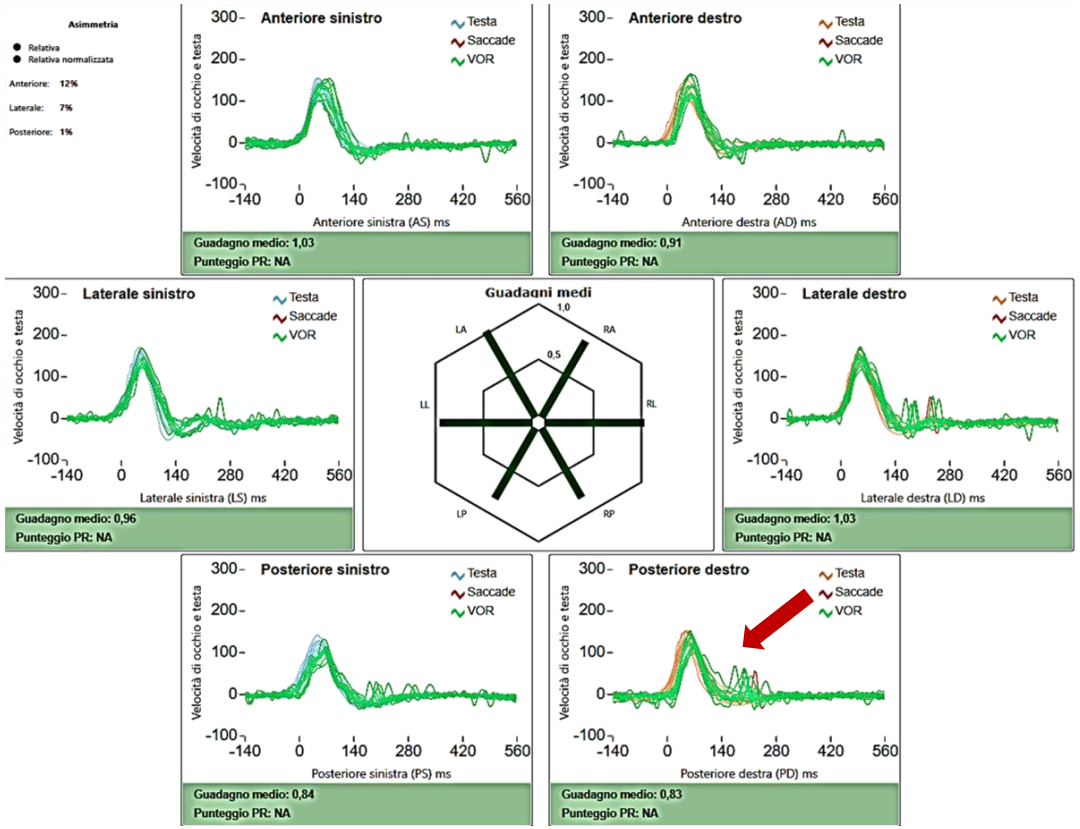
vHIT and BPPV with spontaneous reversal ny



- In provoking position:
  - First, **free-floating debris in the ampullary arm** of the canal **move away from the ampulla**, resulting in **transient excitation of the PSC afferents and in paroxysmal upbeat/right-torsional nystagmus**
  - Then, **partially entrapped debris in the non-ampullary arm** of the canal move toward the ampulla and **block the PSC lumen resulting in an incomplete/positional canalith jam and in persistent positional DBN with left-torsional components**
- **Result: spontaneous reversal of positional nystagmus**

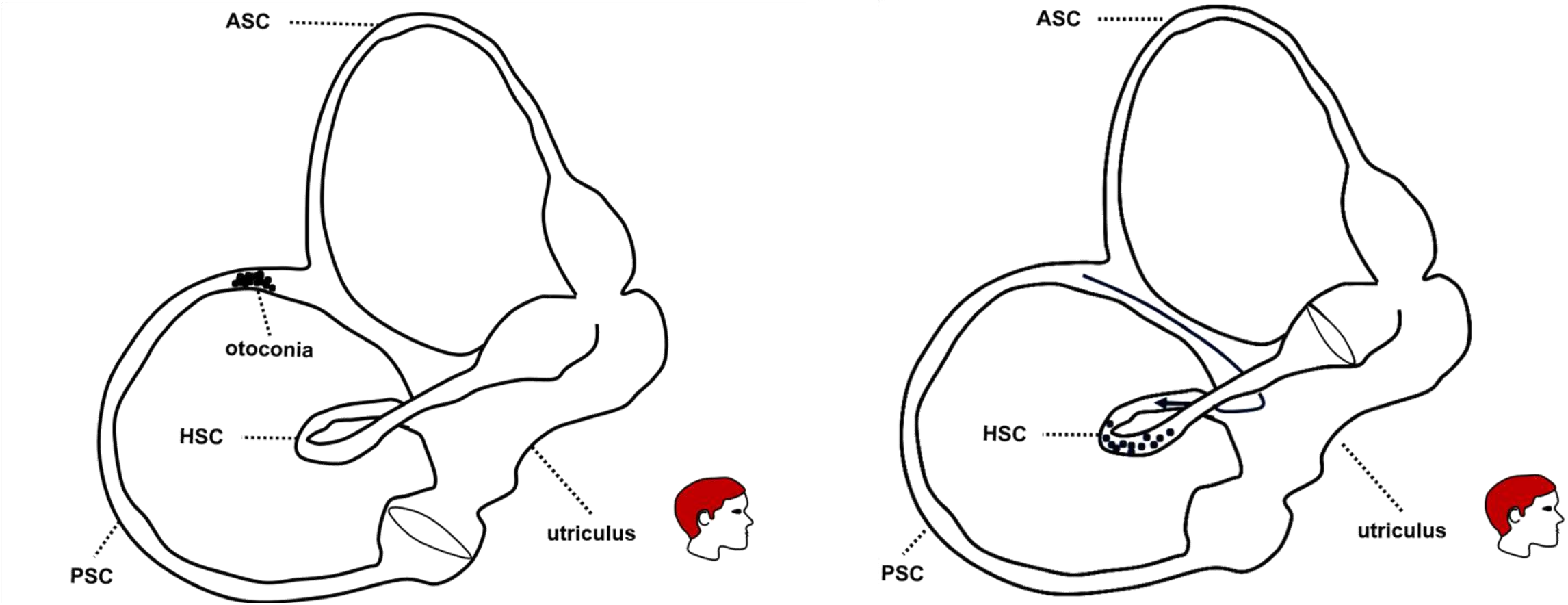
vHIT and BPPV with spontaneous reversal ny

- **Re-evaluation after 3 days**
- **Video HIT: normalization of PSC VOR gain** (gain 0.83)



- **Horizontal paroxysmal direction-changing geotropic nystagmus** on supine head roll maneuver
- **free-floating debris in the non-ampullary arm** of the **right HSC** **move away from the ampulla**, resulting in **transient avtivation of the HSC afferents and in paroxysmal geotropic nystagmus**

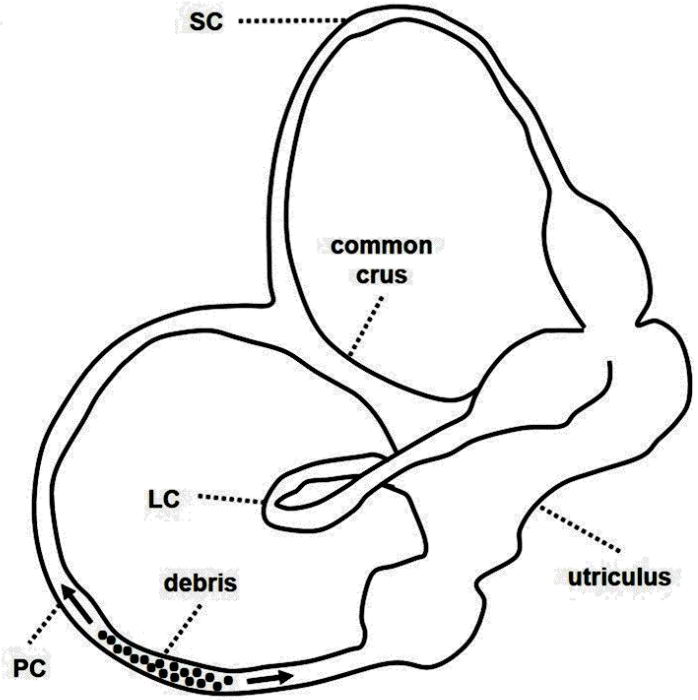
vHIT and BPPV with spontaneous reversal ny

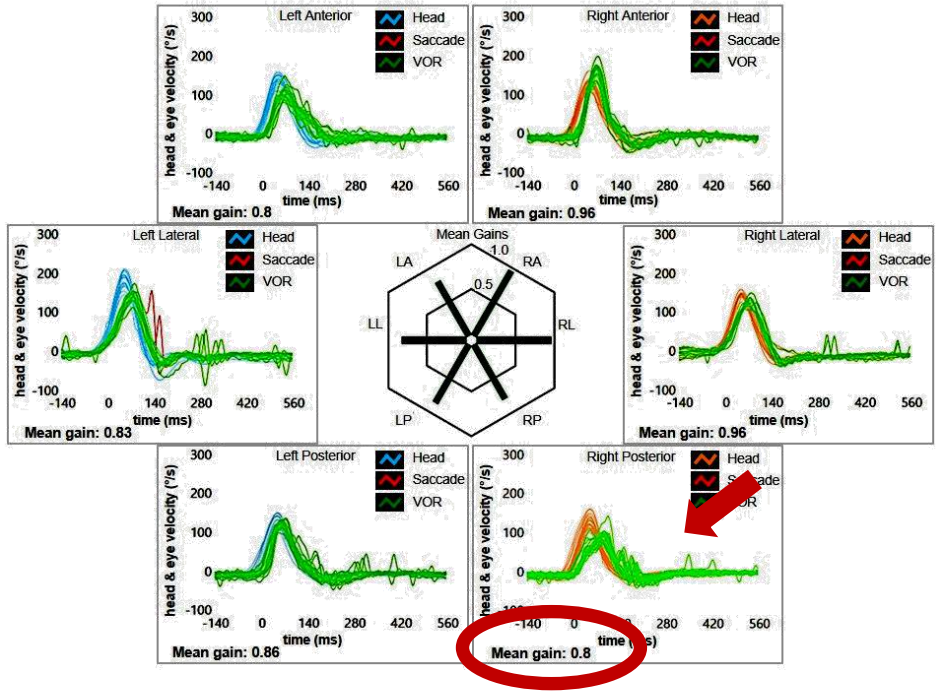


- **Canalith switch** from **right PSC** to **non-ampullary arm of the right HSC**
- Debris are **free to move back and forth along the non-ampullary arm** of the canal, **without impacting on high-frequency VOR dynamics**

Conclusions

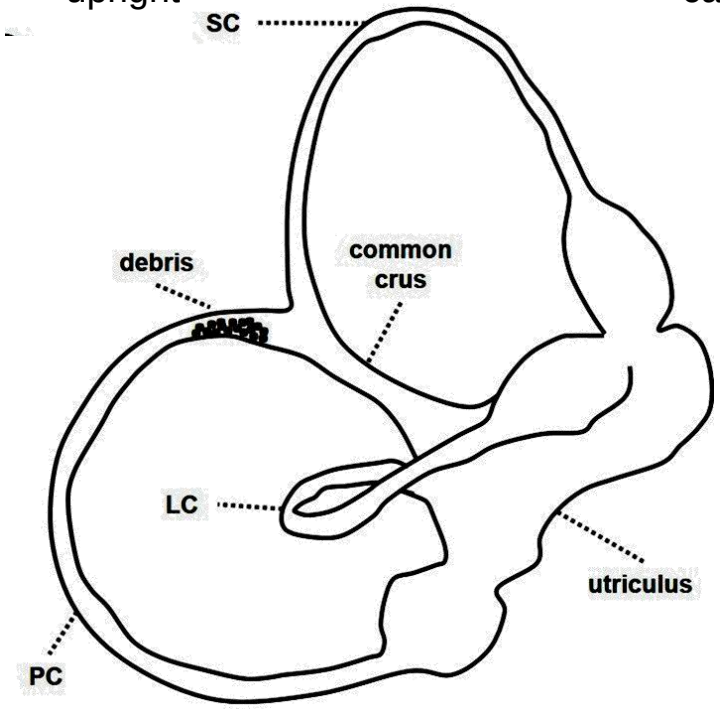
1	eye movements	affected canal VOR-gain on vHIT	proposed pathomechanism	endolymphatic flows	
				low frequency	high frequency
canalolithiasis	transient paroxysmal nystagmus in positioning test, reversing with gravity	generally normal	free-floating debris moving back and forth within the membranous canal	preserved as debris are free to move in both directions along the membranous canal	preserved as debris neither aggregate nor occlude the membranous canal, thus they do not alter ampullary responses

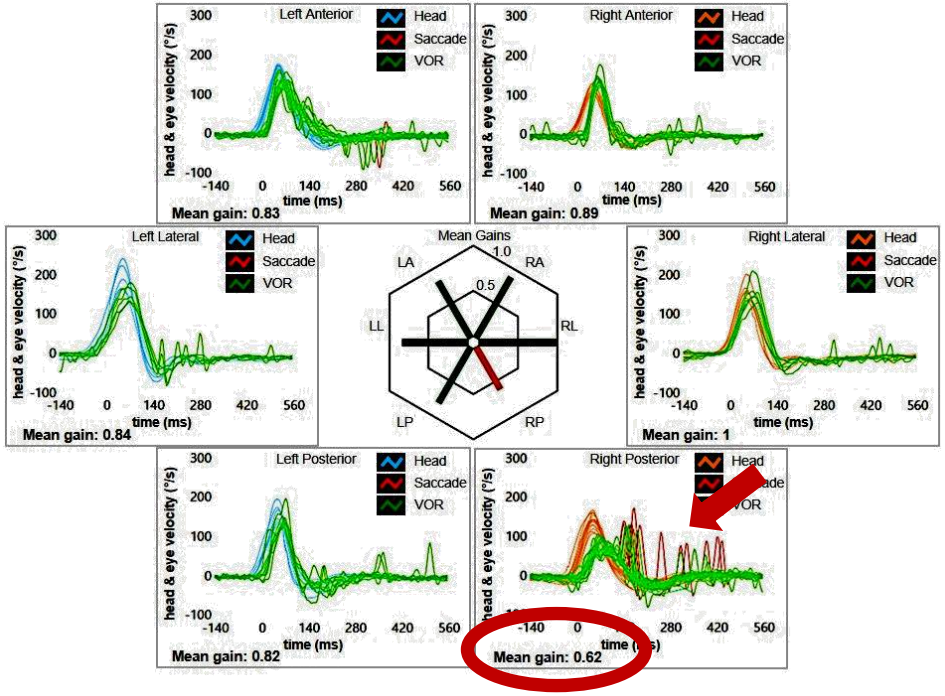




Conclusions

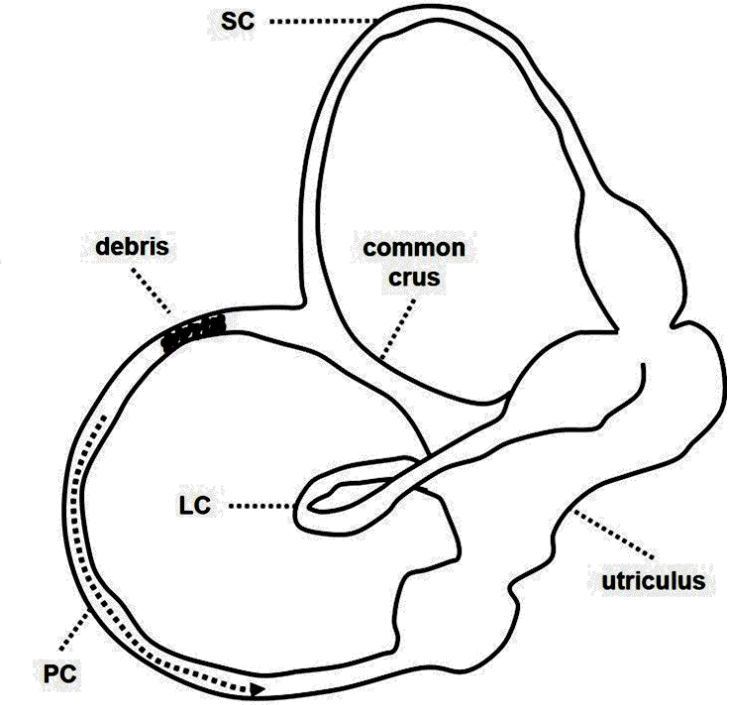
2	eye movements	affected canal VOR-gain on vHIT	proposed pathomechanism	endolymphatic flows	
				low frequency	high frequency
incomplete (or "transient" or "positional") canalith jam	persistent pDBN in both Dix Hallpike test and in SHHP, rarely reversing while upright	slightly reduced	debris are partially entrapped in a narrow tract of the membranous canal, partially plugging the canal lumen	likely preserved as debris, even if partly blocked, are allowed to slowly shift towards the cupula or in opposite direction in Dix Hallpike test or in SHHP	reduced as debris likely impede high-frequency flows dampening responses to head impulses (behaving as a "low-pass filter")

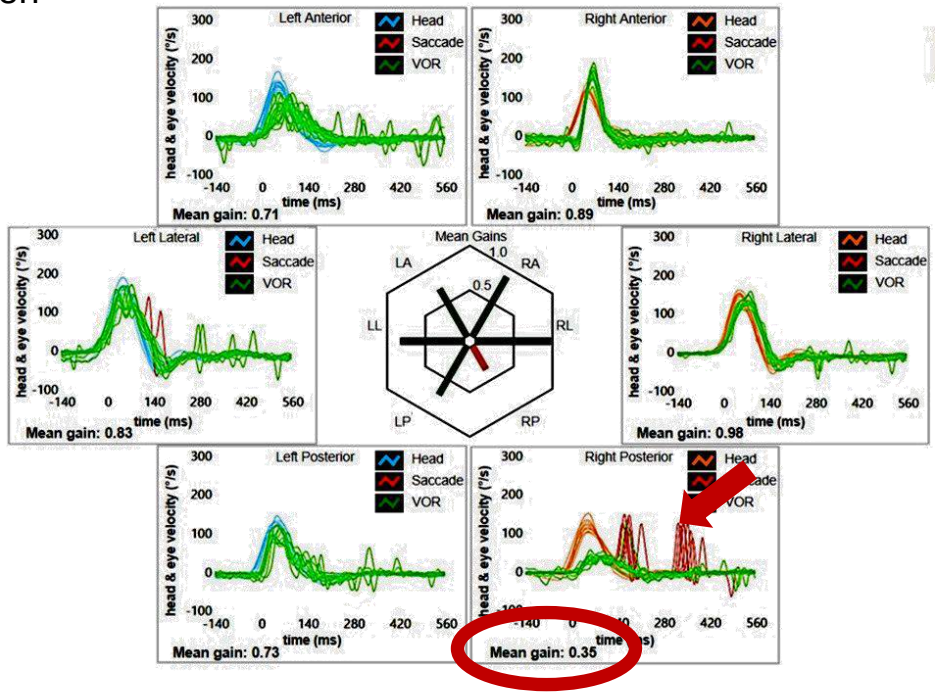




Conclusions

3	eye movements	affected canal VOR-gain on vHIT	proposed pathomechanism	endolymphatic flows	
				low frequency	high frequency
complete canalith jam	spontaneous nystagmus, slightly increasing in positioning tests	markedly reduced	an otolith clot is completely entrapped within a narrow tract of the membranous canal, entirely plugging the canal lumen	prevented due to the continuous endolymphatic pressure generated, leading to a constant cupular deflection	prevented due to the continuous endolymphatic pressure generated, leading to a constant cupular deflection





Thank you

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