Fundamentals of Video Head Impulse Test: Lesson 3 – Interpretation and Clinical applications

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Learning objective

VHITS REPORTING

CLINICAL APPLICATIONS

vHITs reporting parameters

1) High velocity impulses

2) Gain in all the canals

3) Covert saccades

4) Overt saccades

5) Horizontal dispersion of latency

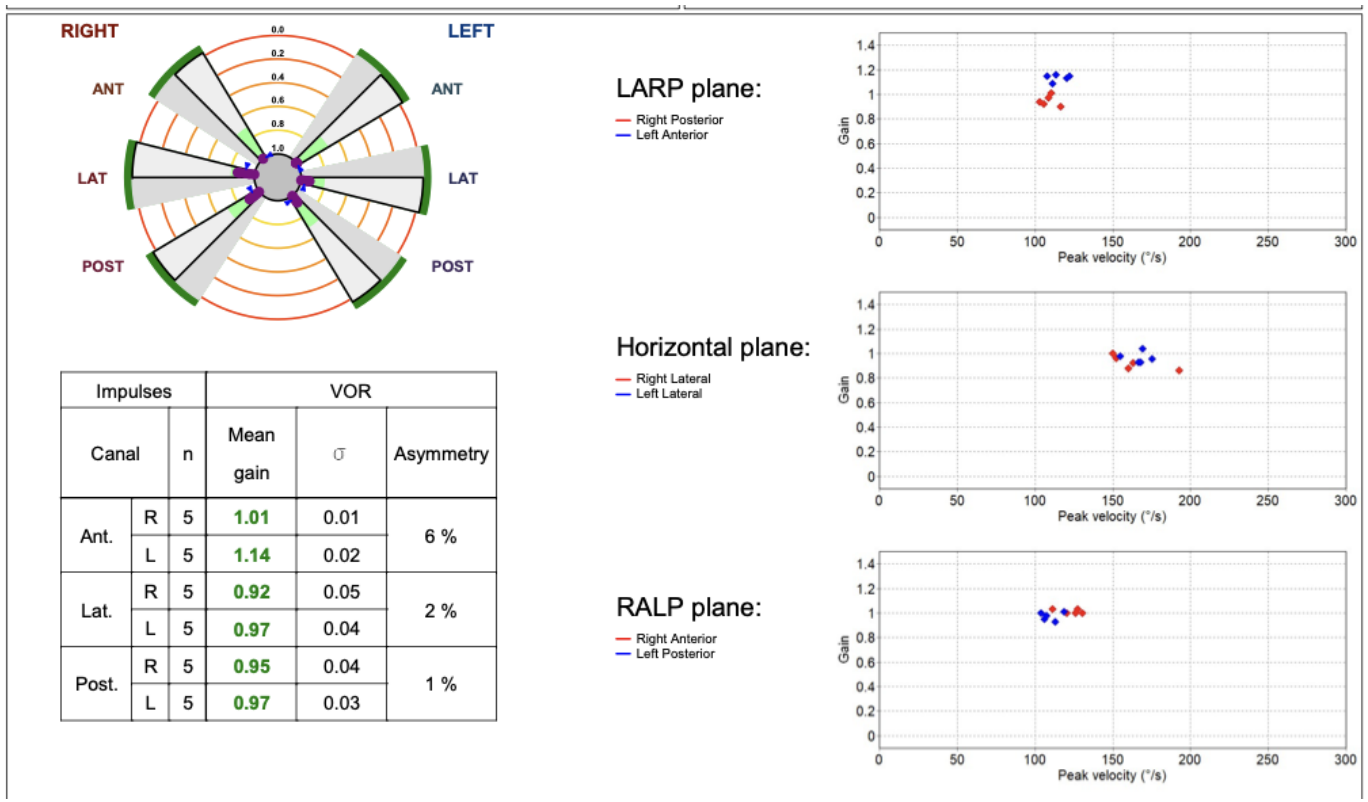
6) Vertical dispersion of gain

7) Anti-compensatory Quick eye movements (AQEM)

8) Artifacts

Mention the Head impulse velocity range during the test

High velocity impulses



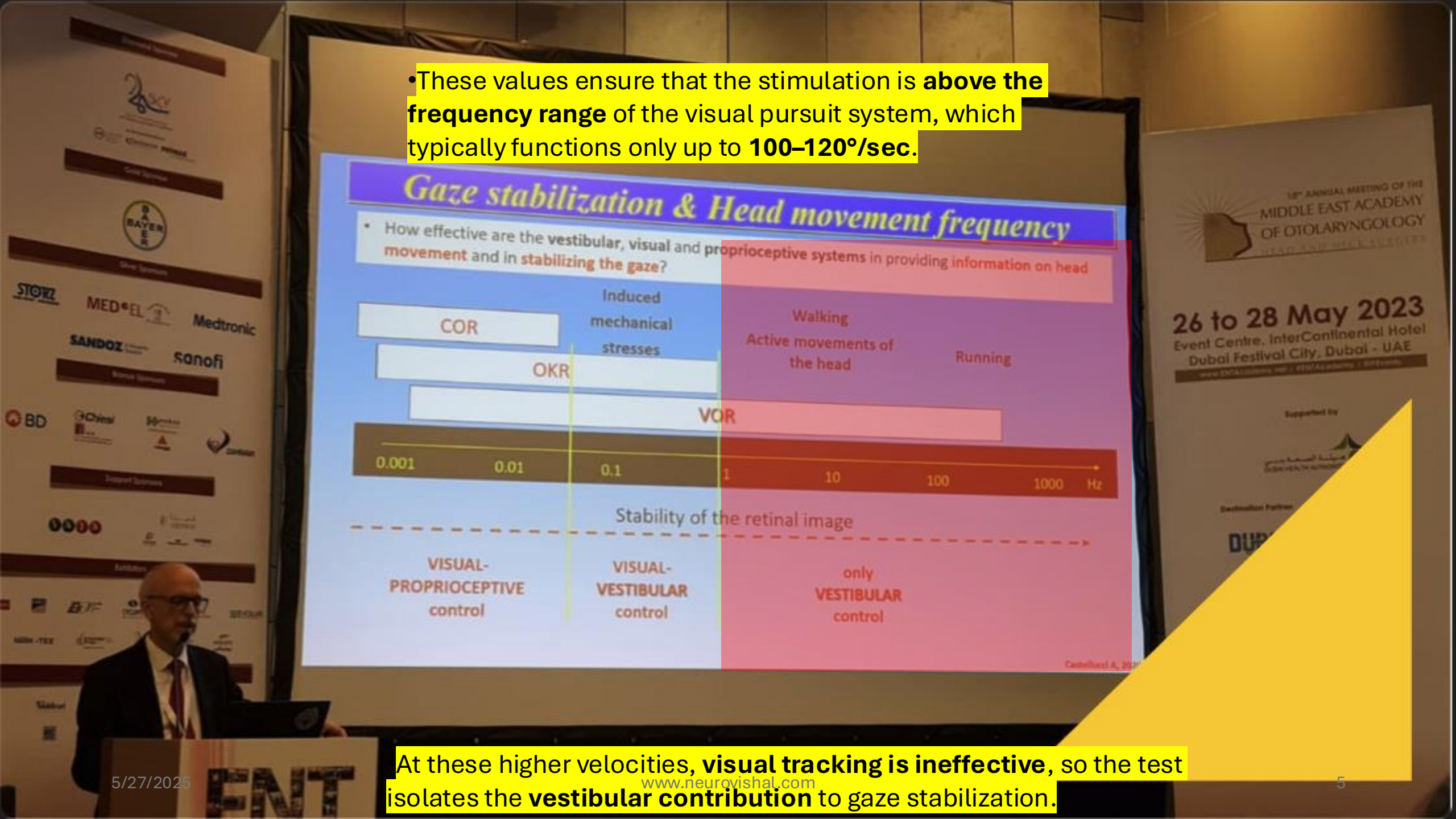
Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	1.01	0.01	6 %
	L	5	1.14	0.02	
Lat.	R	5	0.92	0.05	2 %
	L	5	0.97	0.04	
Post.	R	5	0.95	0.04	1 %
	L	5	0.97	0.03	

Horizontal Canals:

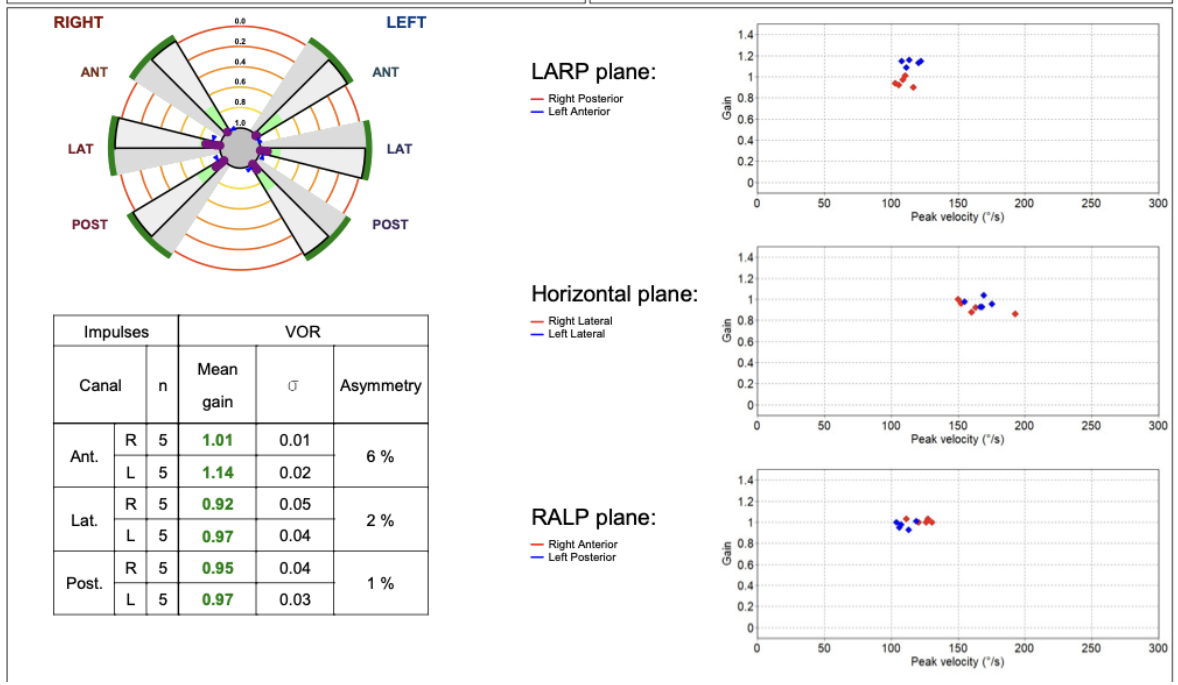
≥ 150°/sec head velocity

Vertical Canals (Anterior and Posterior):

≥ 100°/sec head velocity


•These values ensure that the stimulation is **above the frequency range** of the visual pursuit system, which typically functions only up to **100–120°/sec**.
Gaze stabilization & Head movement frequency
How effective are the vestibular, visual and proprioceptive systems in providing information on head movement and in stabilizing the gaze?
COR
OKR
VOR
Induced mechanical stresses
Walking
Active movements of the head
Running
0.001
0.01
0.1
1
10
100
1000
Hz
Stability of the retinal image
VISUAL-PROPRIOCEPTIVE control
VISUAL-VESTIBULAR control
only VESTIBULAR control
At these higher velocities, **visual tracking is ineffective**, so the test isolates the **vestibular contribution** to gaze stabilization.

1. Impulse Quality



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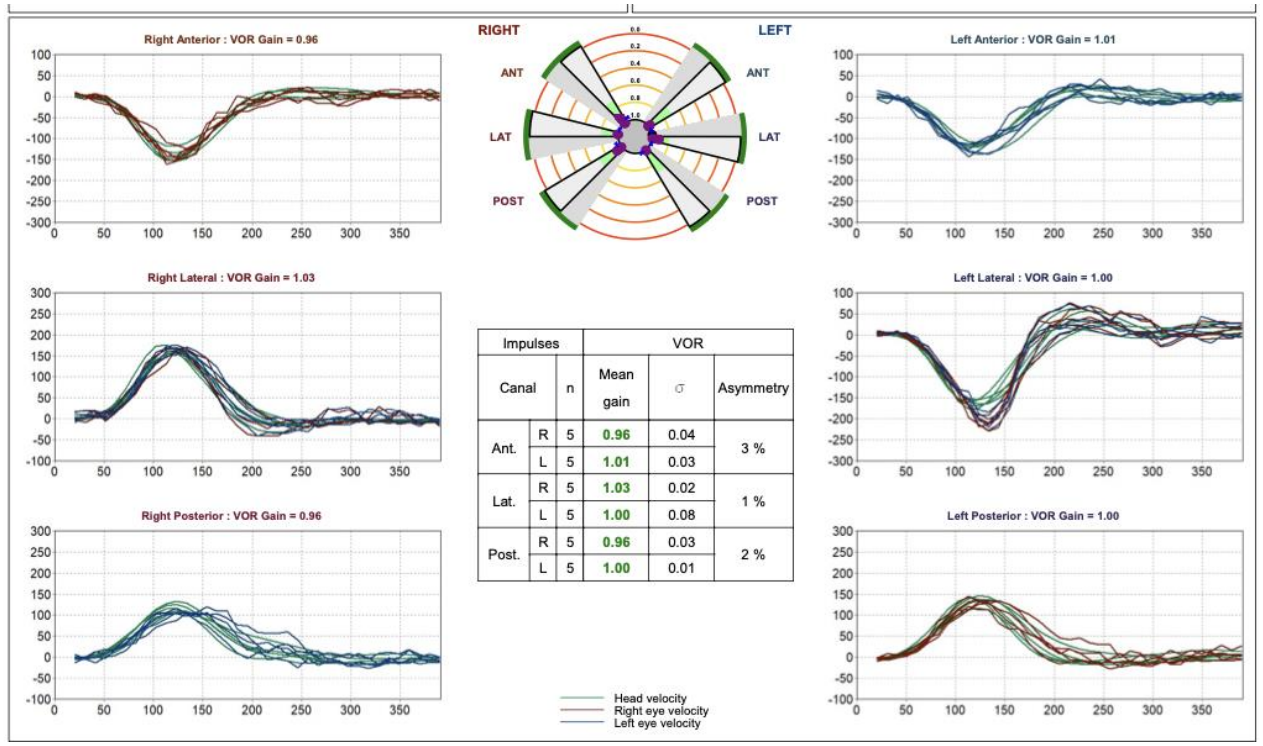
- **Number of valid impulses per canal:**
  - Horizontal (R/L): _____ / _____
  - Anterior (R/L): _____ / _____
  - Posterior (R/L): _____ / _____
- **Peak head velocity:**
  - Horizontal: _____°/s
  - Vertical: _____°/s
- ☐ All impulses exceeded minimum velocity threshold (≥100-150°/s)

Canals Evaluated



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	L	5	1.01	0.03	
Lat.	R	5	1.03	0.02	1 %
	L	5	1.00	0.08	
Post.	R	5	0.96	0.03	2 %
	L	5	1.00	0.01	

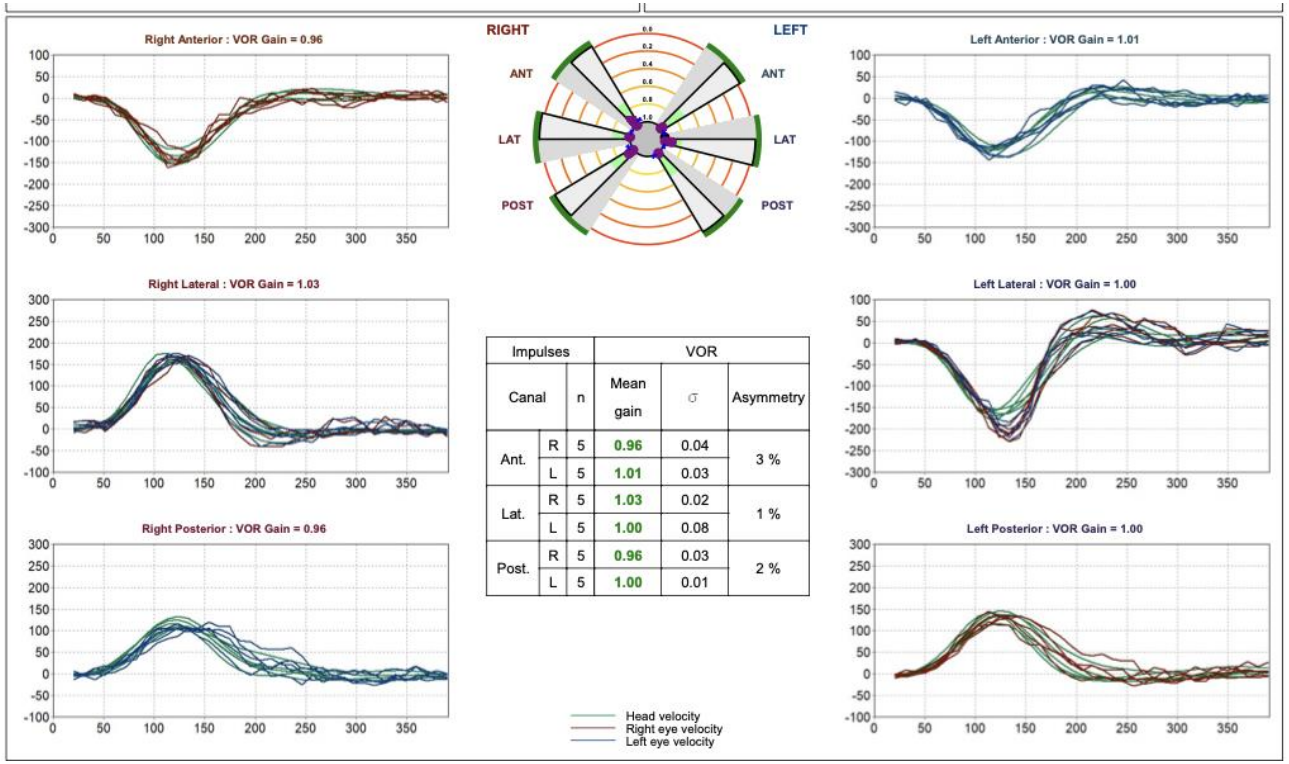
Horizontal:

- Right & Left Lateral

Vertical Pairs:

- Right Anterior / Left Posterior (RA/LP)
- Left Anterior / Right Posterior (LA/RP)

Gain Definition



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	L	5	1.00	0.01	

VOR Gain = Eye Velocity / Head Velocity

Normal gain ≈ 0.8 to 1.1 Horizontal 0.7 to 1 for vertical

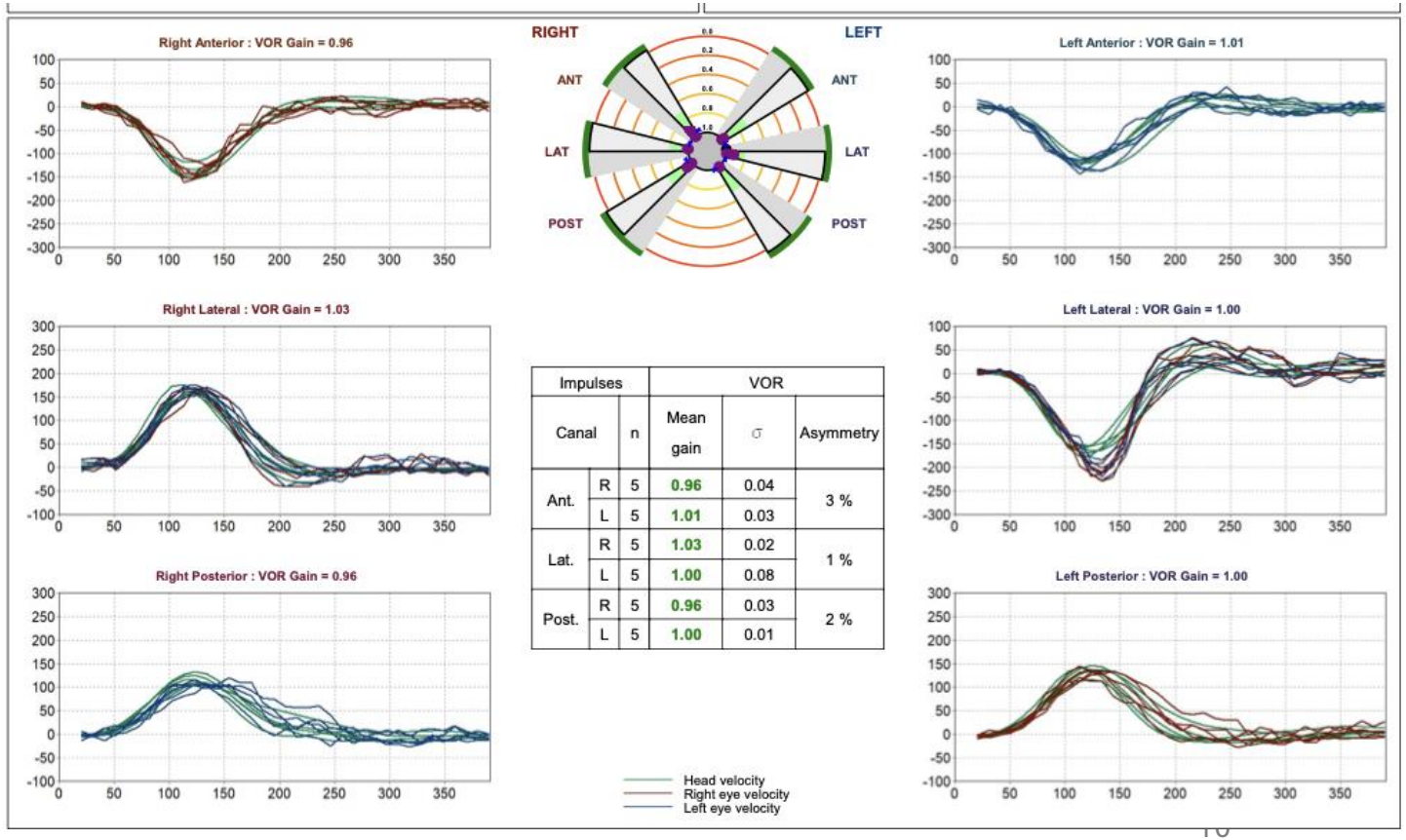
Remote Camera Advantages

- No headgear (reduced slippage/artifacts)
- **Binocular recording for horizontal canals**
- Automatic canal/eye detection
- **Fewer impulses needed** (~5/canal)

Reporting Parameters

- Individual **gain values per canal**
- **Gain asymmetry**
- **Morphology of VOR trace**

Canal	Gain (Right)	Gain (Left)	Normative Range
Horizontal			≥0.8
Anterior (Superior)			≥0.7
Posterior			≥0.7



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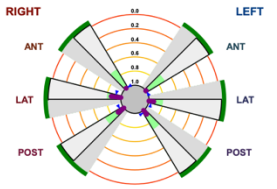
vHIT overview



Impulses			VOR			Early saccades		
Canal		n	Mean gain	σ	Asymmetry	Ratio	Mean latency	Mean apparent gain
Ant.	R	5	1.01	0.01	6 %	0 %		
	L	5	1.14	0.02		0 %		
Lat.	R	5	0.92	0.05	2 %	0 %		
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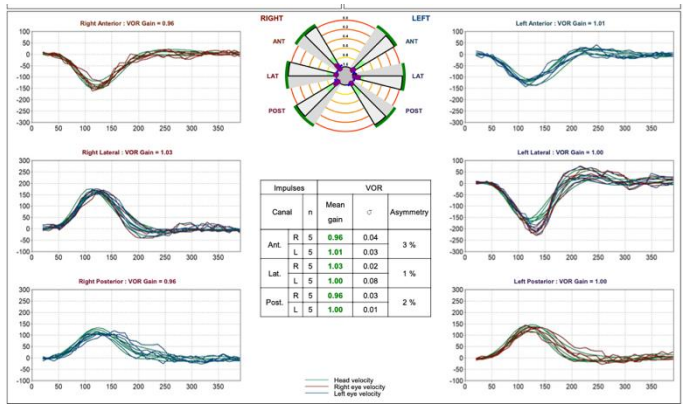
Velocities graph



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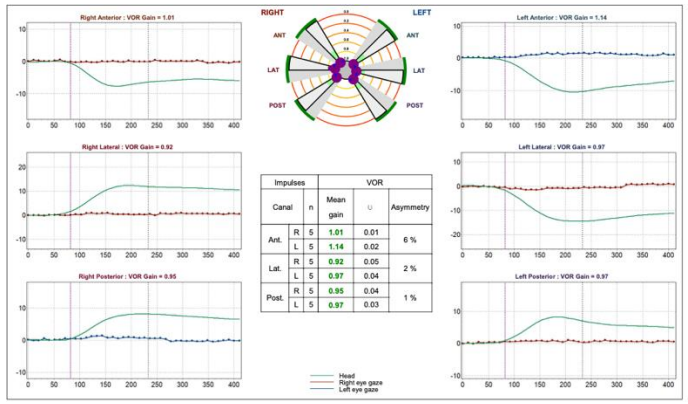
Position graph



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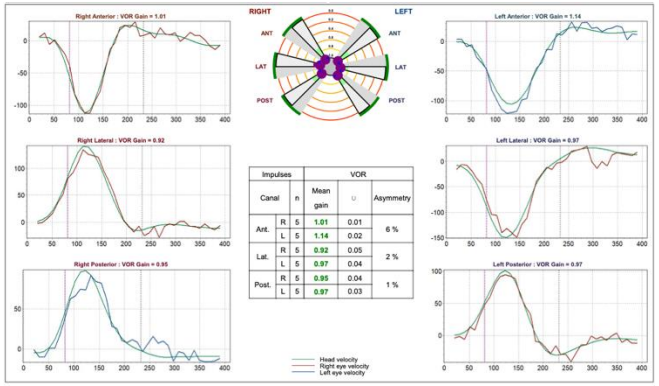
Single impulse velocities graph



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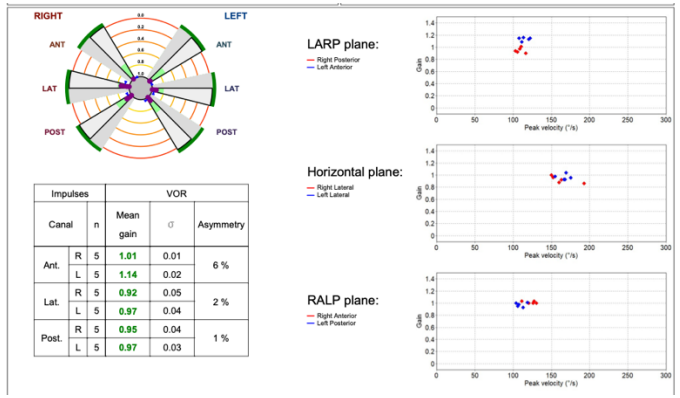
vHIT gain velocity graph



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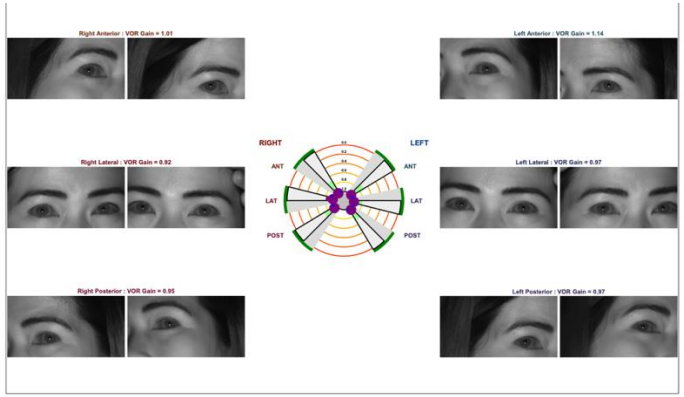
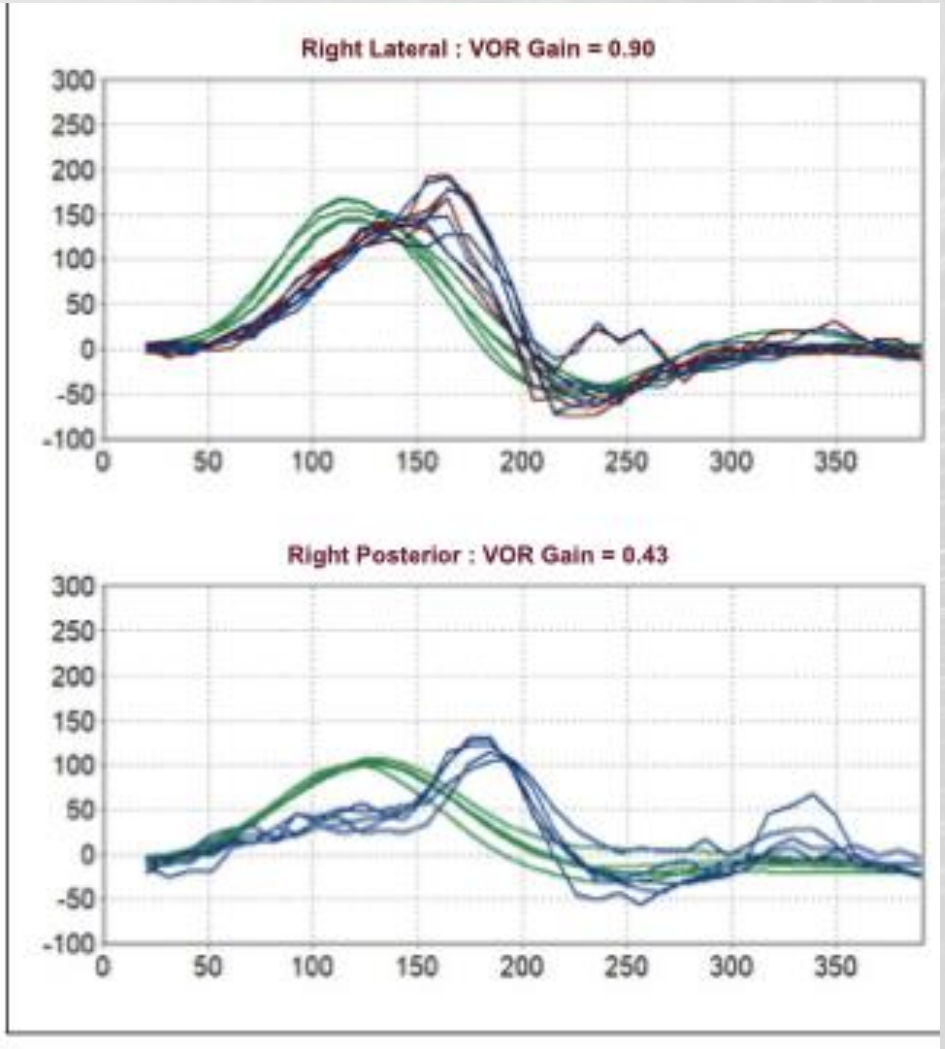
vHIT pictures



Covert saccades



What Are Covert Saccades?

- Occur **during** head impulse
- **Invisible** to the naked eye
- Compensate for **VOR deficits** to stabilize gaze

Diagnostic Value

- **Marker of vestibular hypofunction**, even with normal gain
- Improve detection over bedside tests (cHIT)
- Help localize **peripheral lesions**

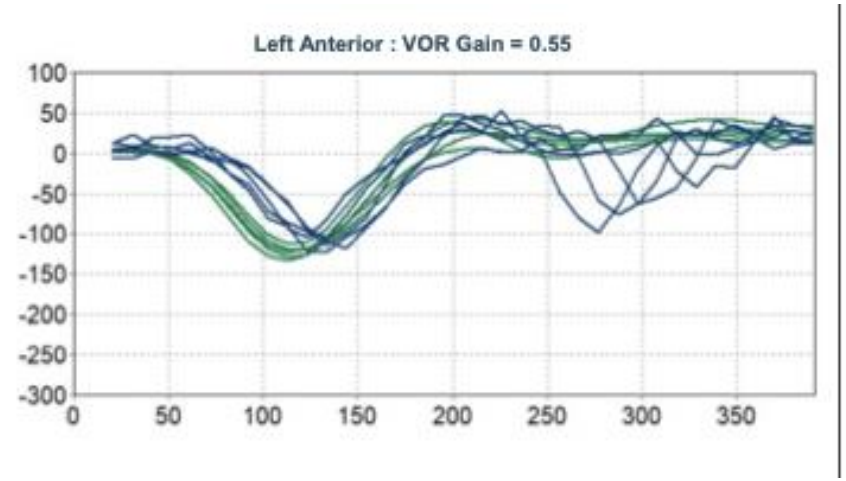
Reporting Criteria

- **Latency > 70 ms**
- **Amplitude/Velocity** >100°/sec or >½ head speed
- **Consistency** = recurrent across impulses
- **Direction** = same as VOR (except in hyper-gain)

Clinical Relevance

- Covert saccades with normal gain = **still pathological**
- Key for identifying **compensated vestibular loss**

Reporting Overt Saccades in Remote Camera vHIT



🔍 What Are Overt Saccades?

- **Corrective eye movements** occurring **after** head impulse
- **Visible** in clinical exams (cHIT) and detected in vHIT
- Indicate **VOR failure** to maintain gaze stability

🧠 Diagnostic Significance

- Signal **vestibular hypofunction**, especially with low gain
- Common in **acute vestibular disorders** (e.g., neuritis)
- **Repeatable overt saccades** = pathological, even if gain borderline

📏 Reporting Criteria

- **Latency**: ~200 ms post-impulse
- **Velocity**: >100°/sec or >½ of head speed
- **Amplitude**: Large enough to restore fixation
- **Consistency**: Seen in most impulses to affected side

🧩 Interpretation Tips

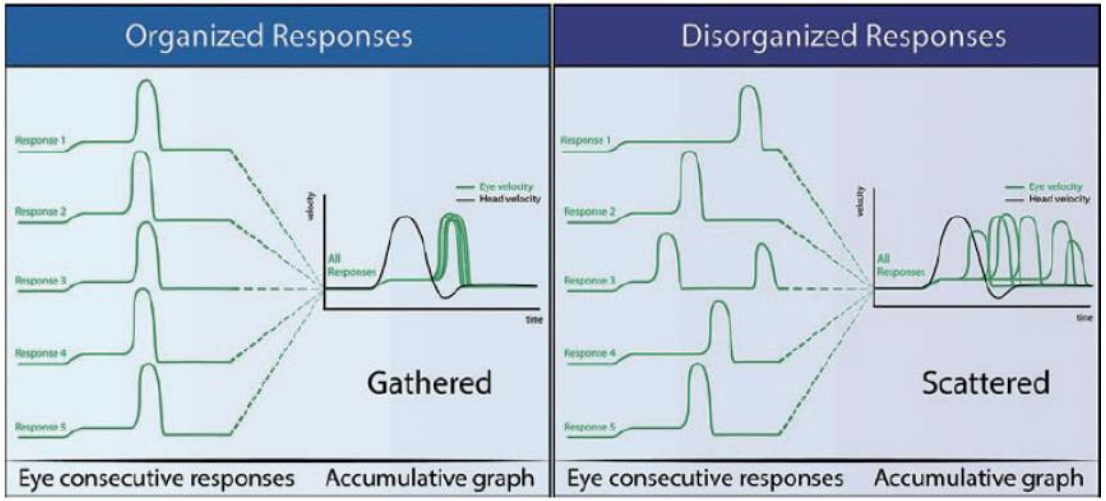
- Overt saccades + ↓ gain = **clear vestibular deficit**
- May appear with **covert saccades** in chronic cases
- ↓ gain but no saccades? → **Suspect artifacts or bilateral loss**

- **Presence**:
  - ☐ Covert
  - ☐ Overt
  - ☐ Both
  - ☐ None
- **Latency**:
  - Covert: ~_____ ms
  - Overt: ~_____ ms
- **Amplitude / Velocity**:
  - Covert: _____ ° / _____°/s
  - Overt: _____ ° / _____°/s

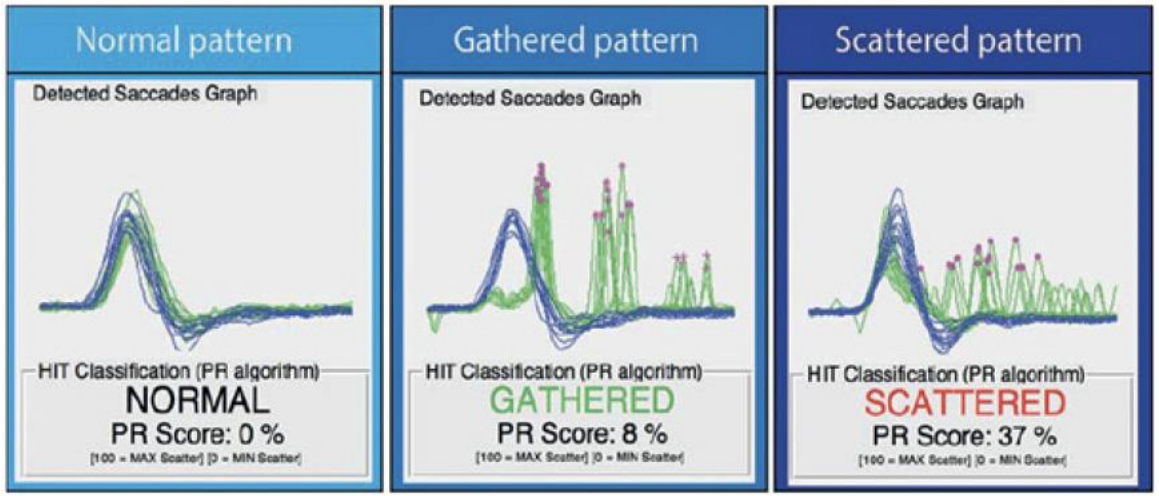
Horizontal dispersion of saccades

- **Definition**: Variability in gain values across impulses
- **Healthy**: Tight clustering
- **Deficit**: Wide-spread → **unstable or partial VOR compensation**
- Often linked to **inconsistent saccades**

Prediction and refixation score: Temporal scatter



Compensated Uncompensated



(Rey-Martinez J, 2015).

Gain Decline with Increasing Head Velocity

VOR Gain Decline with Head Velocity

- Slight decline is normal; **marked drop = pathology**
- Exposed by **high-velocity impulses** (≥220°/sec)
- Suggests **canal hypofunction**, especially with corrective saccades

Clinical Insight

- Analyze **both gain & dispersion**
- Even moderate gain with high dispersion = **possible dysfunction**

•**Observation**:
•☐ Low dispersion (predictable timing)
•☐ High dispersion (irregular timing)
•**PR Score** (if available): _____
•☐ Low (gathered)
•☐ High (scattered)

Reporting Vertical Dispersion in Remote Camera vHIT

What Is Vertical Dispersion?

- Variability in **eye velocity** across impulses
- Seen as **spread in superimposed traces** (gain or velocity plots)
- Indicates **inconsistent VOR response** despite uniform stimuli

Causes

- **Central compensation** instability
- **Variable covert saccades**
- **Inconsistent head impulses**
- Reflects **partial recovery** or neurological involvement

Clinical Relevance

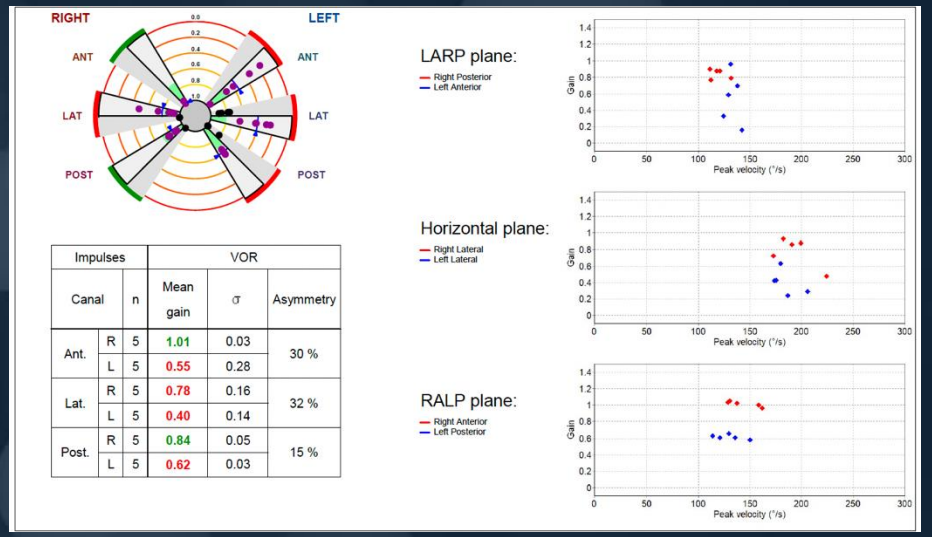
- **Healthy = tight clustering; pathology = wide dispersion**
- Suggests **unstable VOR or test artifacts**
- Combine with **gain and saccade metrics** for accurate interpretation

Reporting Tips

- Note **presence, symmetry**, and **relationship to gain**
- **High dispersion + low gain = likely dysfunction**
- Recommend **repeat testing** or monitor during compensation

Summary

- Vertical dispersion = **trace variability**
- Relevant in identifying **hidden vestibular deficits**
- Enhances assessment of **test quality and clinical accuracy**



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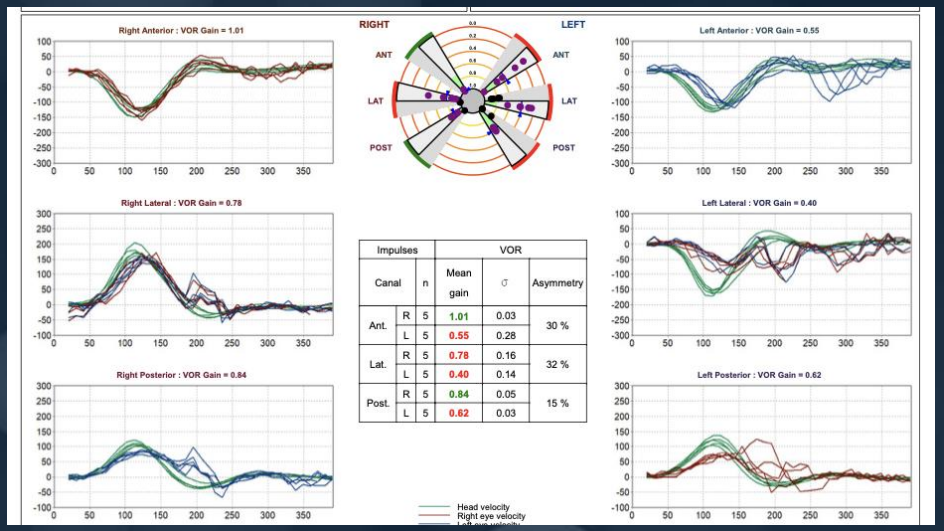

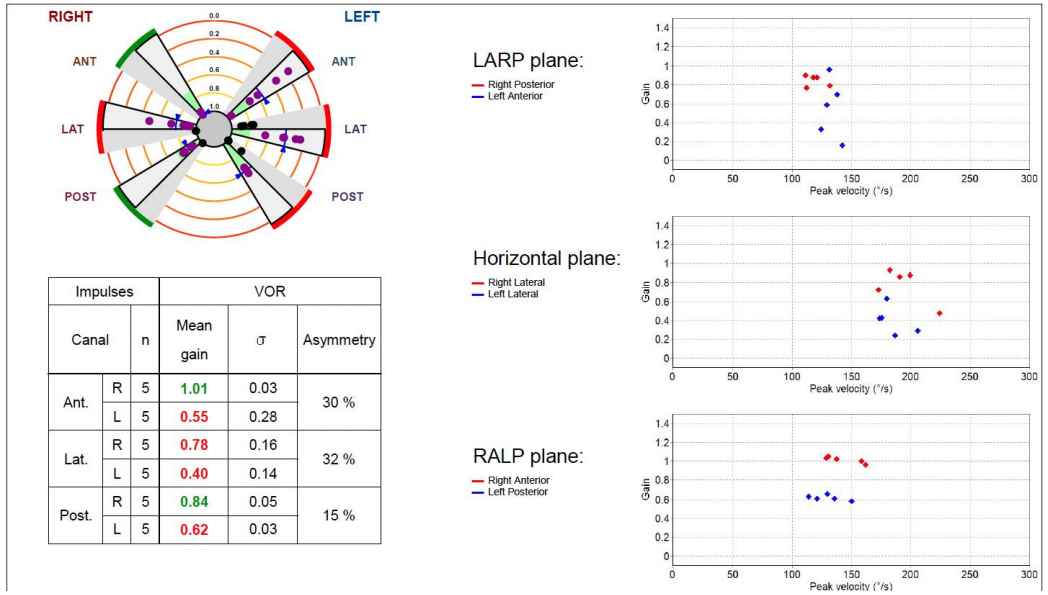

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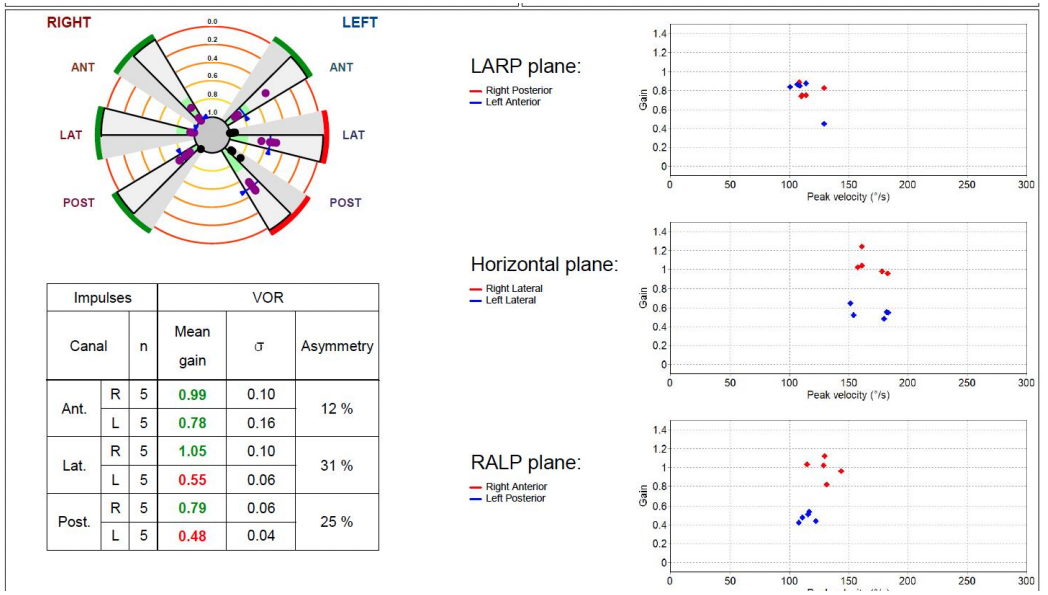

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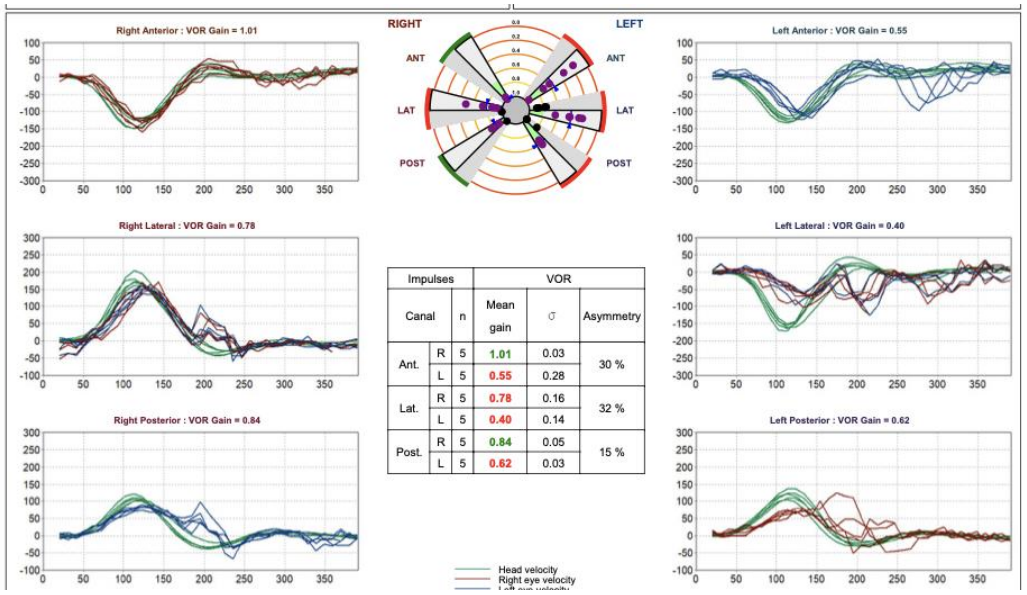

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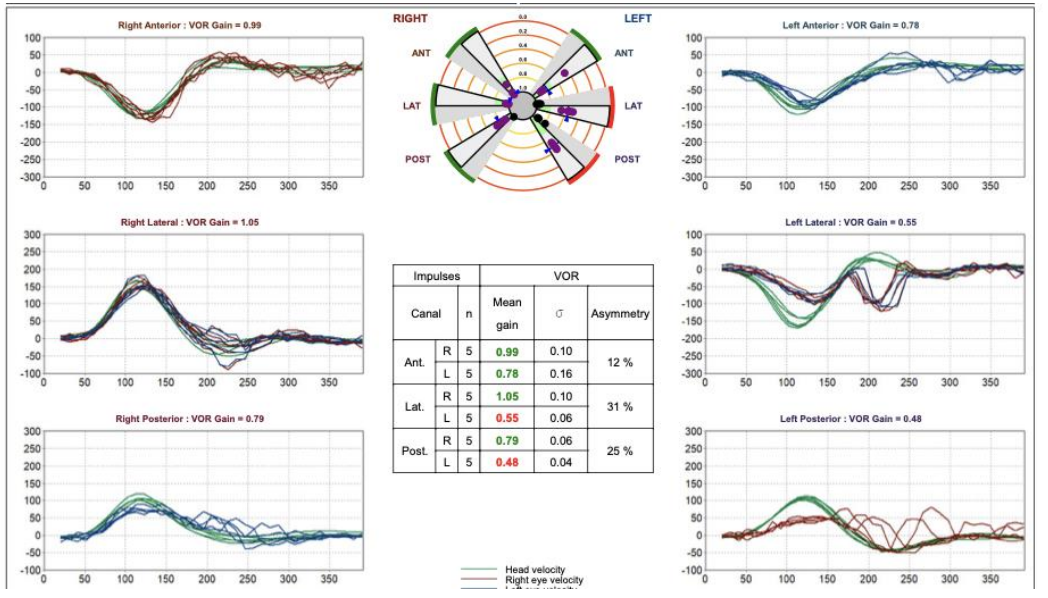

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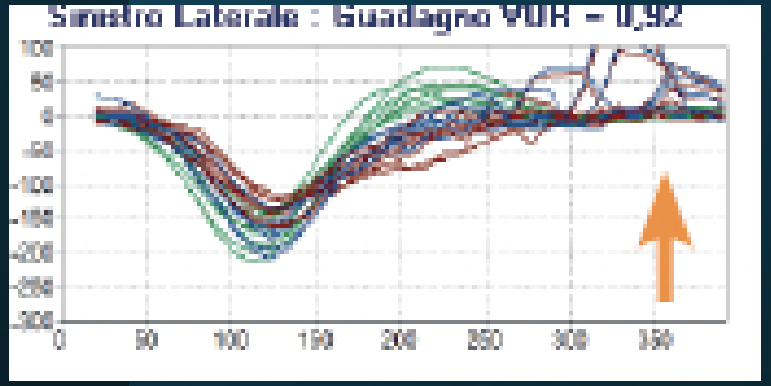
Anti-compensatory quick eye movements



Definition:

- AQEM are **quick eye movements in the same direction as the head impulse,** opposite to expected compensatory saccades.

Clinical Utility:

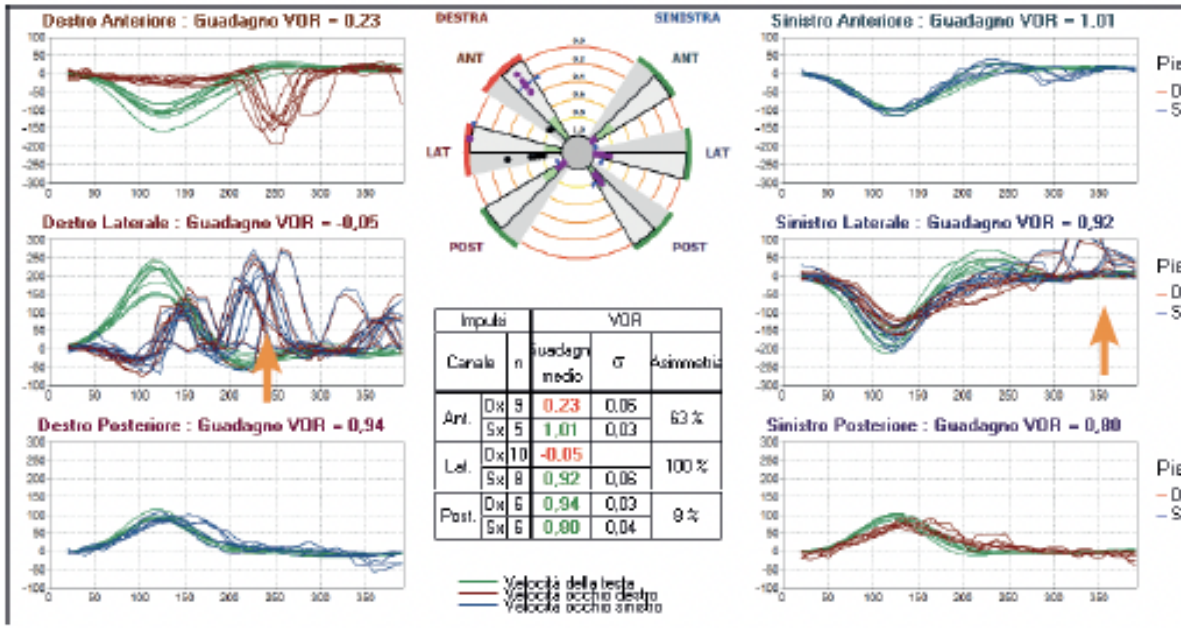
- Seen in **vestibular migraine** and **Ménière's disease**, not typically in central lesions .
- Help differentiate **peripheral vs. central causes**

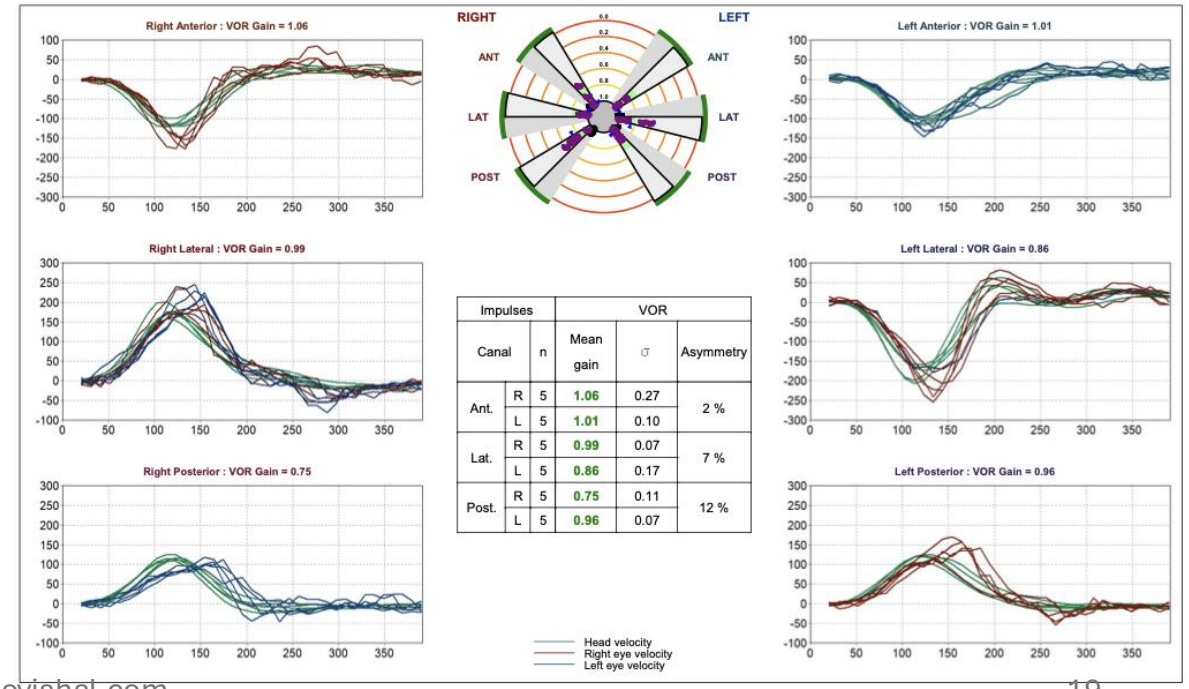
Interpretation Tip:

- AQEM are **not artifacts or saccades due to VOR hyper-gain.**
- They reflect **delayed deceleration of the VOR and eye overshoot.**

Types:

- **Covert AQEM**:
  - May reflect **gain asymmetry** and subtle vestibular imbalance.
- **Overt AQEM**:
  - Often a sign of **peripheral vestibular lesion** during contra-lesional impulses





Artifacts in Remote Camera vHIT: Recognition & Avoidance

🎯 What Are Artifacts?

False signals in eye/head velocity traces

Caused by **technical issues, patient behavior**, or **environmental interference**

May **mimic pathology** or obscure real deficits

⚠️ Common Types

Blinks → spikes/interruption

Corneal reflex loss → dropout

Poor tracking → jagged, noisy traces

Fixation errors / facial obstruction (e.g., glasses, eyelashes)

🧠 How to Recognize

Flat traces = fixation loss

Spikes = blink or drift

Out-of-phase motion = slippage

Inconsistent gain = poor stimuli or frame drops

🩹 How to Prevent

Instruct patient to **minimize blinking**

Ensure **stable corneal tracking & good lighting**

Avoid reflections or shadows

Perform **≥5 clean impulses per canal**

Review video + traces for confirmation

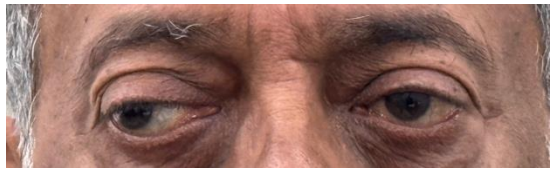
📌 Red Flags

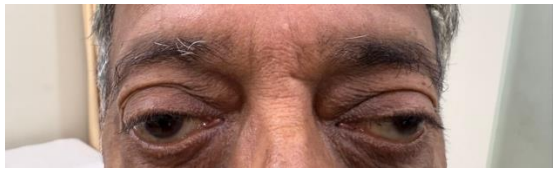
Sudden gain drop **without saccades**

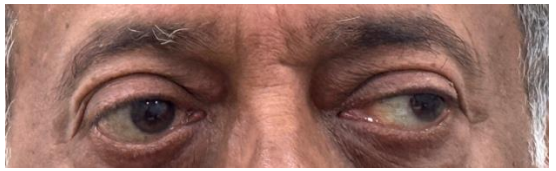
Eye faster than head

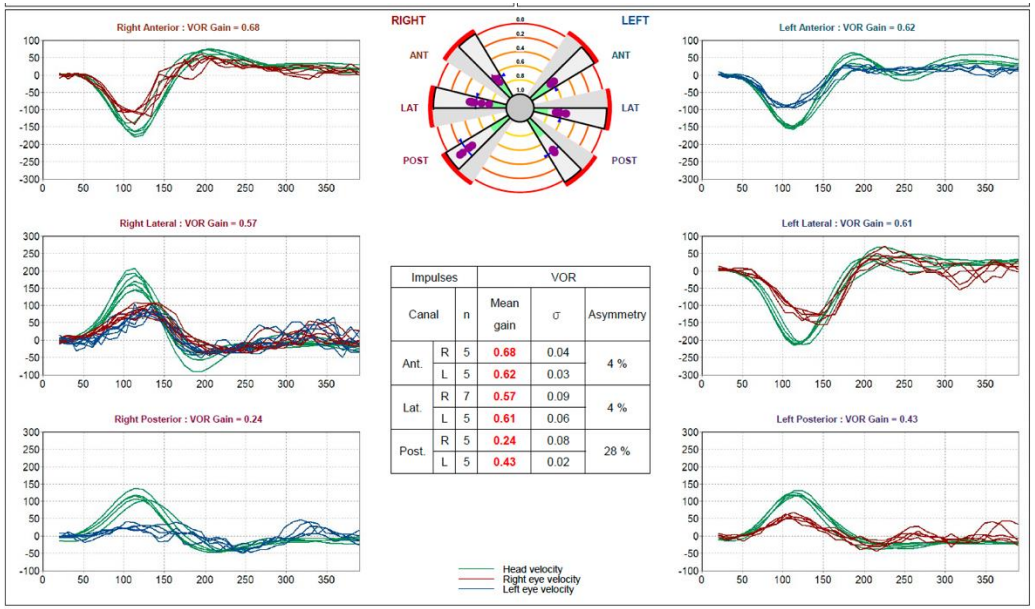
High trace **variability across impulses**

Video dropout or reflex misalignment





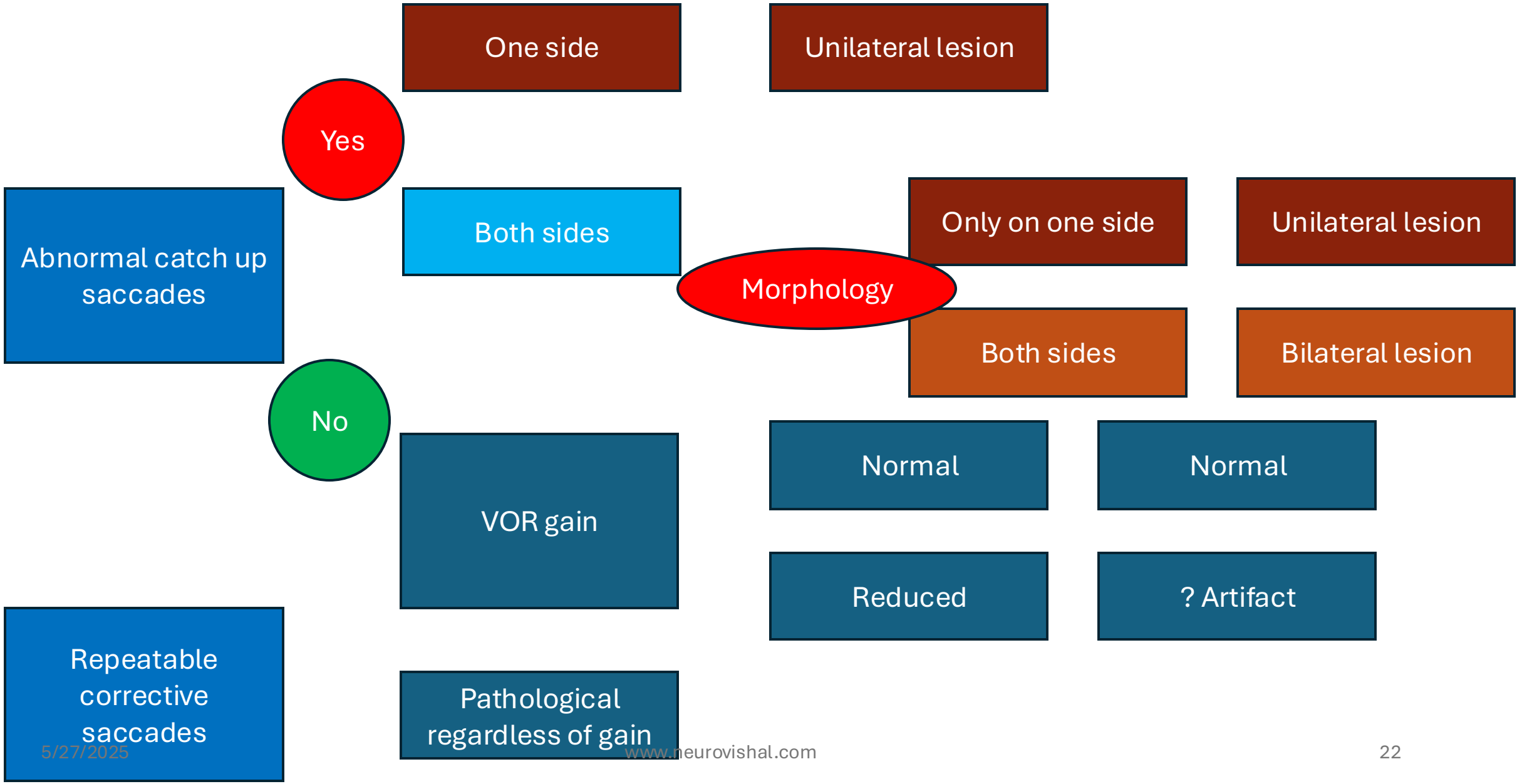




Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	0.68	0.04	4 %
	L	5	0.62	0.03	
Lat.	R	7	0.57	0.09	4 %
	L	5	0.61	0.06	
Post.	R	5	0.24	0.08	28 %
	L	5	0.43	0.02	

vHIT in Squint Post Eye Surgery

- **Challenge**:
  - Strabismus and eye muscle surgery can cause **ocular misalignment**, making vHIT interpretation unreliable.
- **Observation**:
  - In squint or skew deviation, **both eyes fail to converge** on the central target—leading to **asymmetric position and velocity curves** during horizontal impulses .
- **Clinical insight**:
  - In cases with ocular misalignment, **interpret results cautiously**—prioritize clinical history and alternate vestibular tests.


One side
Unilateral lesion
Yes
Abnormal catch up saccades
Both sides
Only on one side
Unilateral lesion
Morphology
Both sides
Bilateral lesion
No
VOR gain
Normal
Normal
Reduced
? Artifact
Repeatable corrective saccades
Pathological regardless of gain

Interpretation

☐ Normal vHIT

☐ Unilateral vestibular hypofunction – Side……

☐ Bilateral vestibular hypofunction

☐ Signs of compensation (e.g., covert saccades)

☐ AQEM noted – suggests peripheral imbalance

☐ Artifacts present – interpret with caution

Recommend correlation with clinical findings and other vestibular tests

vHITs applications

PERIPHERAL VESTIBULAR DISORDERS

CENTRAL VESTIBULAR DISORDERS

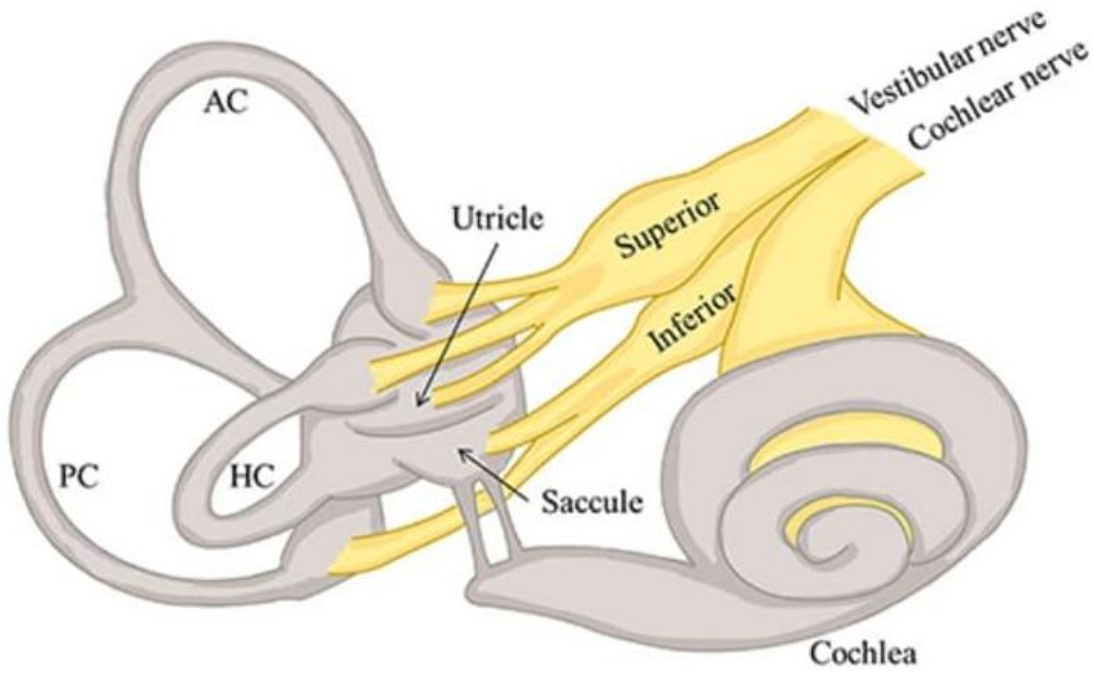
OTHER CONDITIONS

Peripheral vestibular disorders

- **Acute Unilateral Vestibulopathy (AUV)**
- **Ménière's Disease**
- **Benign Paroxysmal Positional Vertigo (BPPV)**
- **Bilateral Vestibular Hypofunction (BVH)**
- Presbyvestibulopathy
- **Ototoxic Vestibulopathy (e.g., Gentamicin)**
- **Post-Cochlear Implantation Vestibular Dysfunction**
- **Vestibular Schwannoma**
- **Suspected Canal Dehiscence / Fistulae**
- **Post-Traumatic Vestibular Hypofunction**
- **Pediatric Peripheral Vestibular Disorders**
- Other peripheral disorders

Acute unilateral vestibulopathy

- Acute, unilateral dysfunction of the vestibular nerve
- Presents with **sudden vertigo, imbalance, and nausea**
- **No hearing loss** (differentiates from labyrinthitis)
- Most commonly affects the **superior division** (lateral and anterior canals)



Role of vHITs in acute unilateral vestibulopathy

1. Rapid Bedside Confirmation of Peripheral Lesion

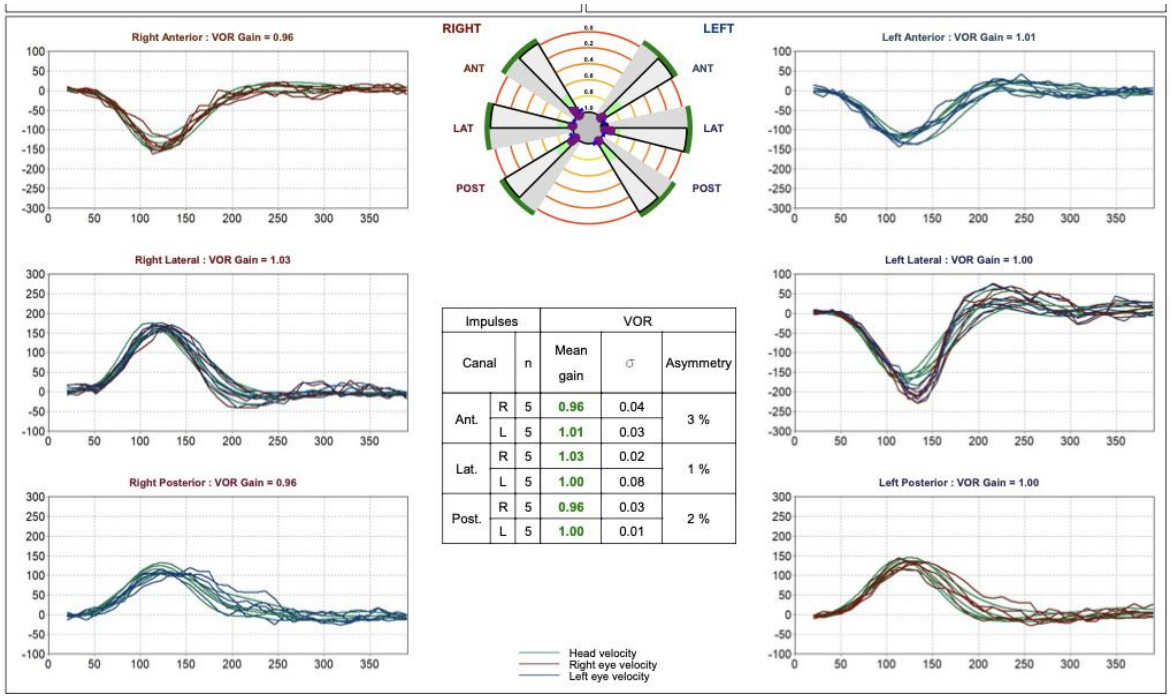
2. Identifies Canal-Specific Dysfunction

3. Detects Compensation Strategies

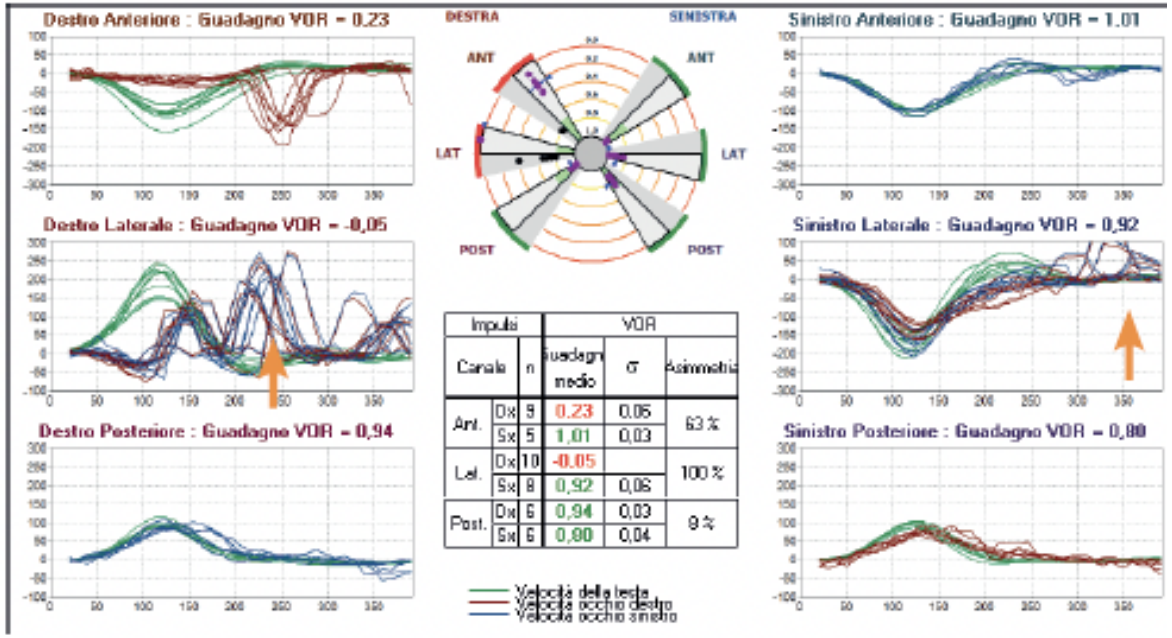
4. Essential for Monitoring Recovery

Rapid Bedside Confirmation of Peripheral Lesion

Acute vestibular syndrome

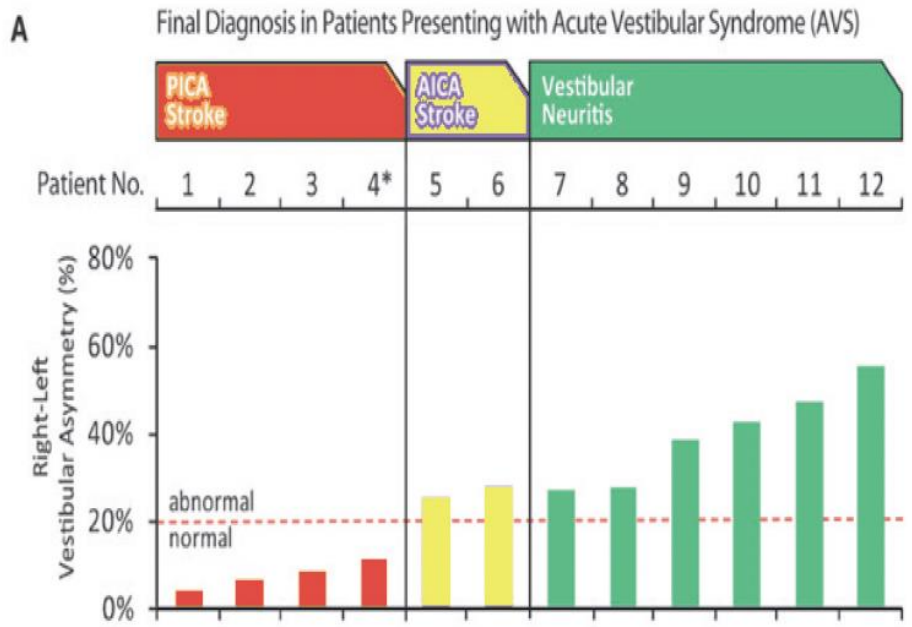


Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	0.96	0.04	3 %
	L	5	1.01	0.03	
Lat.	R	5	1.03	0.02	1 %
	L	5	1.00	0.08	
Post.	R	5	0.96	0.03	2 %
	L	5	1.00	0.01	



Impulsi			VOR		
Canale		n	Guadagno medio	σ	Asimmetria
Ant.	Dx	9	0,23	0,05	63 %
	Sx	5	1,01	0,03	
Lat.	Dx	10	-0,05		100 %
	Sx	8	0,92	0,05	
Post.	Dx	6	0,94	0,03	9 %
	Sx	6	0,80	0,04	

vHITs in acute vestibular syndrome

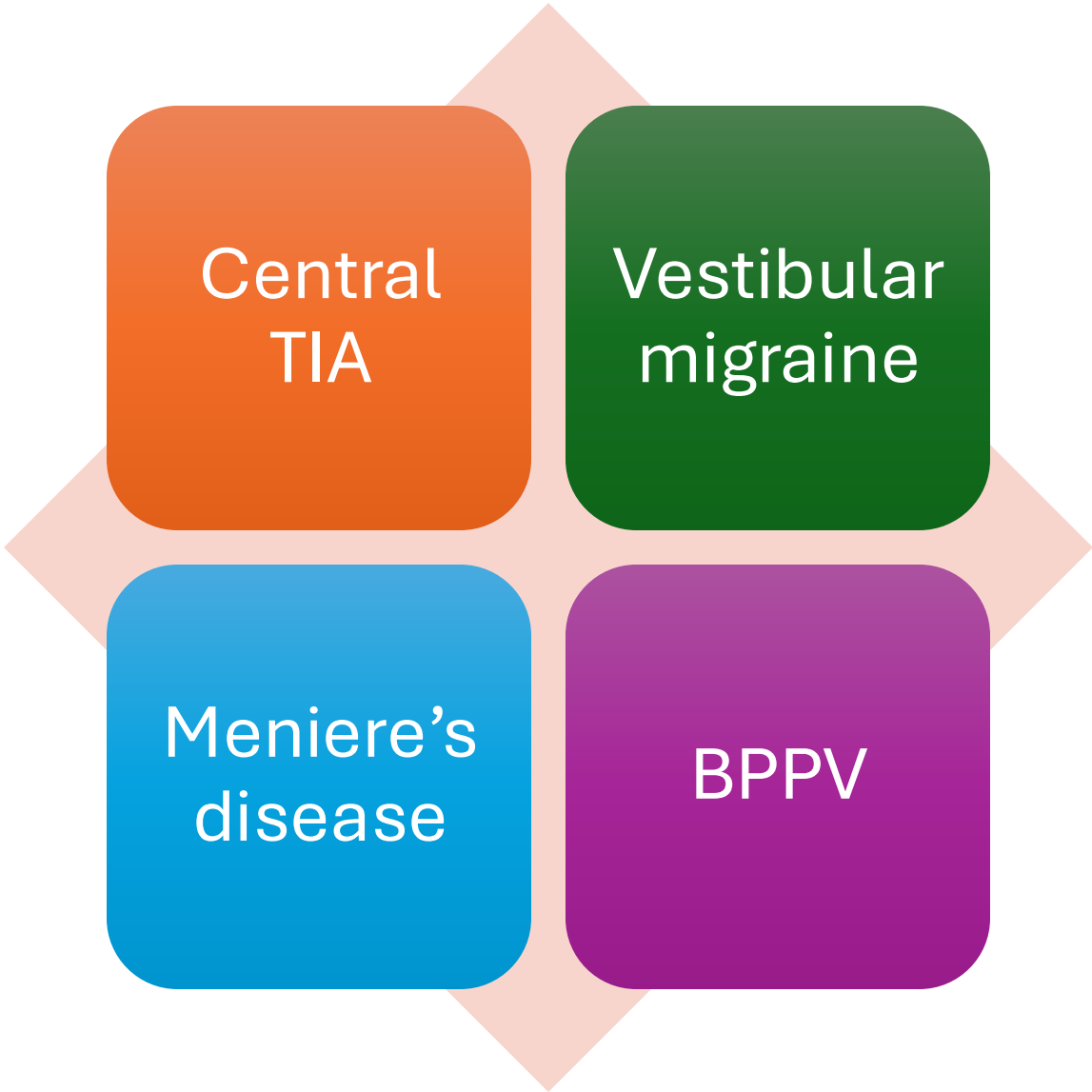


Newman-Toker DE, 2013.

AICA infarct Vs AUV

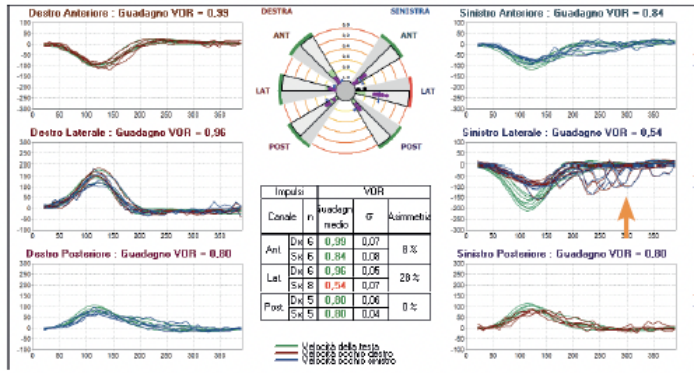
Feature	AICA Infarct	Vestibular Neuritis
vHIT VOR gain	↓ Unilateral or mixed	↓ Unilateral
Saccades	Present, possibly disorganized	Present, organized
Hearing loss	Common	Rare
Central signs	Often present	Absent
VOR suppression	May be impaired	Usually normal

Normal vHITs in episodic vertigo

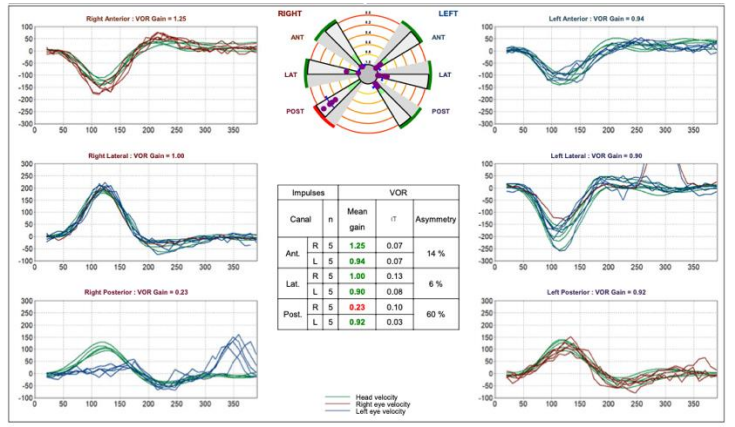


Identifies Canal-Specific Dysfunction

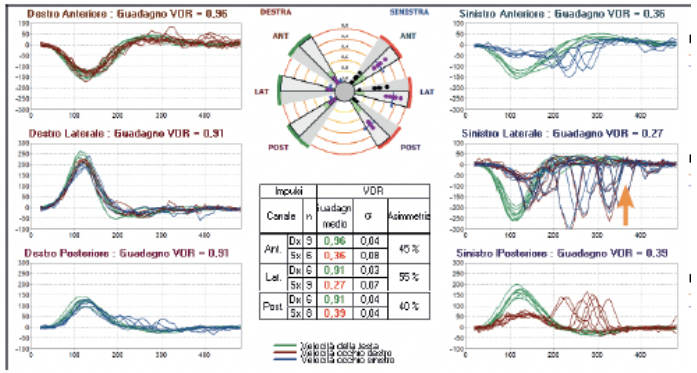
Left Lateral canal



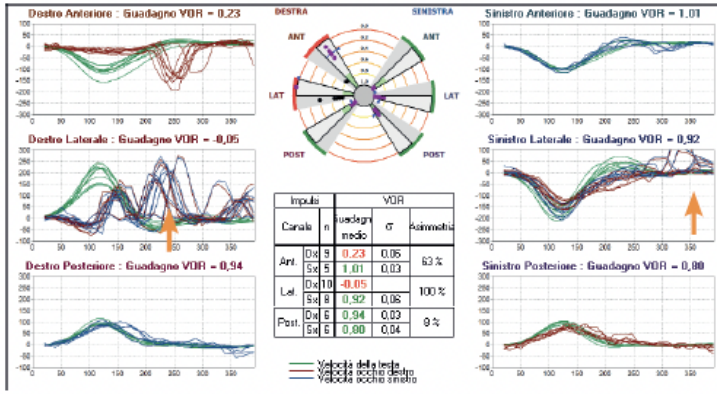
Right posterior canal



Left total vestibular loss

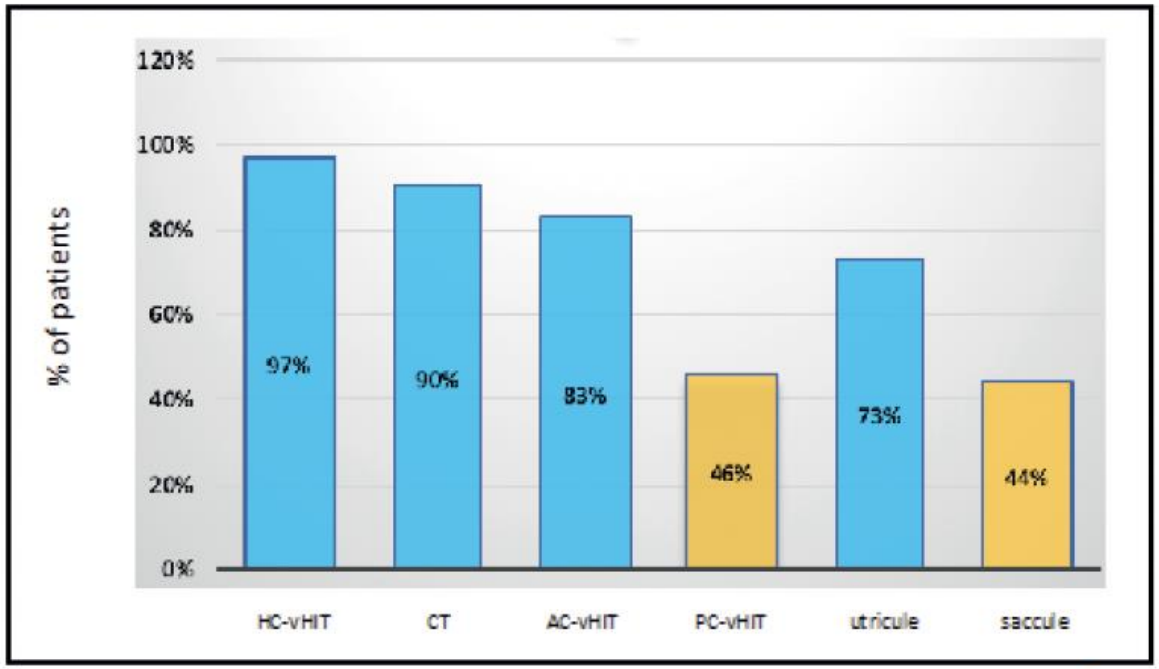


Right Anterior and Lateral canal

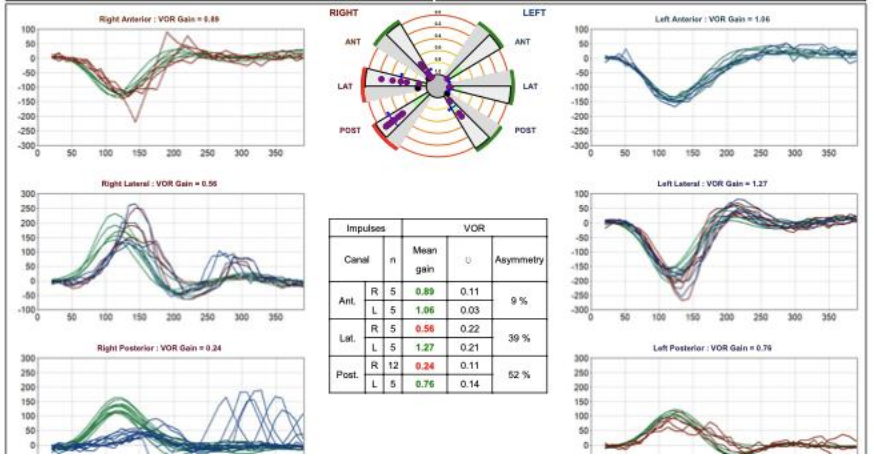


oVEMP	Utricle	73%
vHITs	Anterior canal	83%
	Lateral canal	92%
	Posterior canal	46%
cVEMP	Saccule	44%

Casani AP, Navari E.
Lesion Patterns and Possible Implications for Recovery in Acute Unilateral Vestibulopathy.
In: vHIT Enrico Book, Chapter contribution.
Otol Neurotol. 2020;41:250–255



	Normal	Superior vestibular loss	Inferior vestibular loss	Total vestibular loss	Partial superior	Partial superior + Inferior
oVEMP	Utricle	Utricle	Utricle	Utricle	Utricle	Utricle
vHITs	Anterior canal	Anterior canal	Anterior canal	Anterior canal	Anterior canal	Anterior canal
	Lateral canal	Lateral canal	Lateral canal	Lateral canal	Lateral canal	Lateral canal
	Posterior canal	Posterior canal	Posterior canal	Posterior canal	Posterior canal	Posterior canal
cVEMP	Saccule	Saccule	Saccule	Saccule	Saccule	Saccule
		22%		32%	22%	24%



Uncompensated vestibulopathy

Detects Compensation Strategies

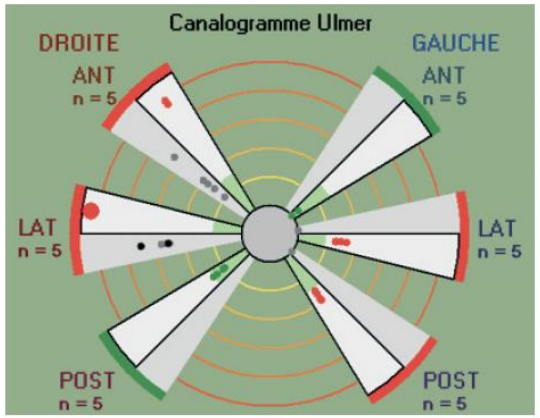
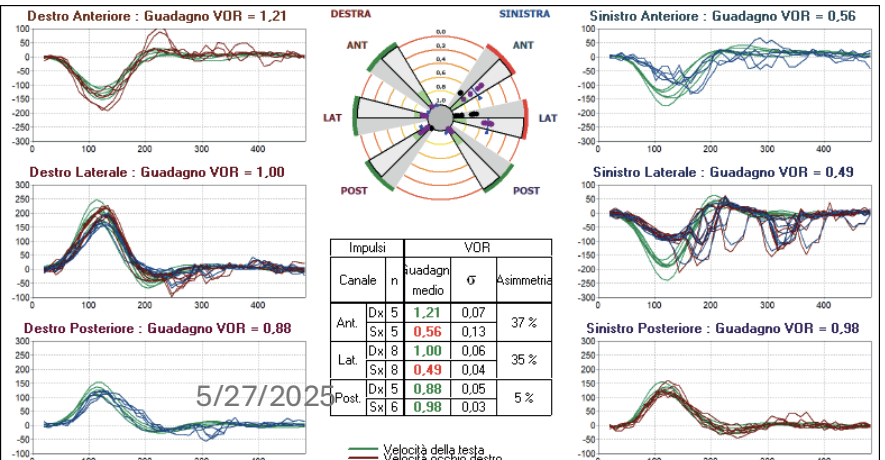
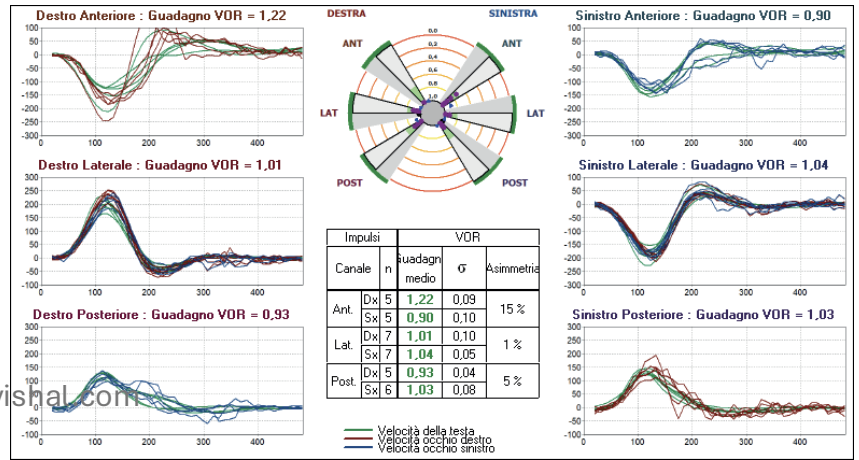


Figure 12.3 Neuritis of the right superior vestibular nerve. Dumas. 0. 2020.

Compensatory gain loss on healthy side to reduce VOR gain asymmetry

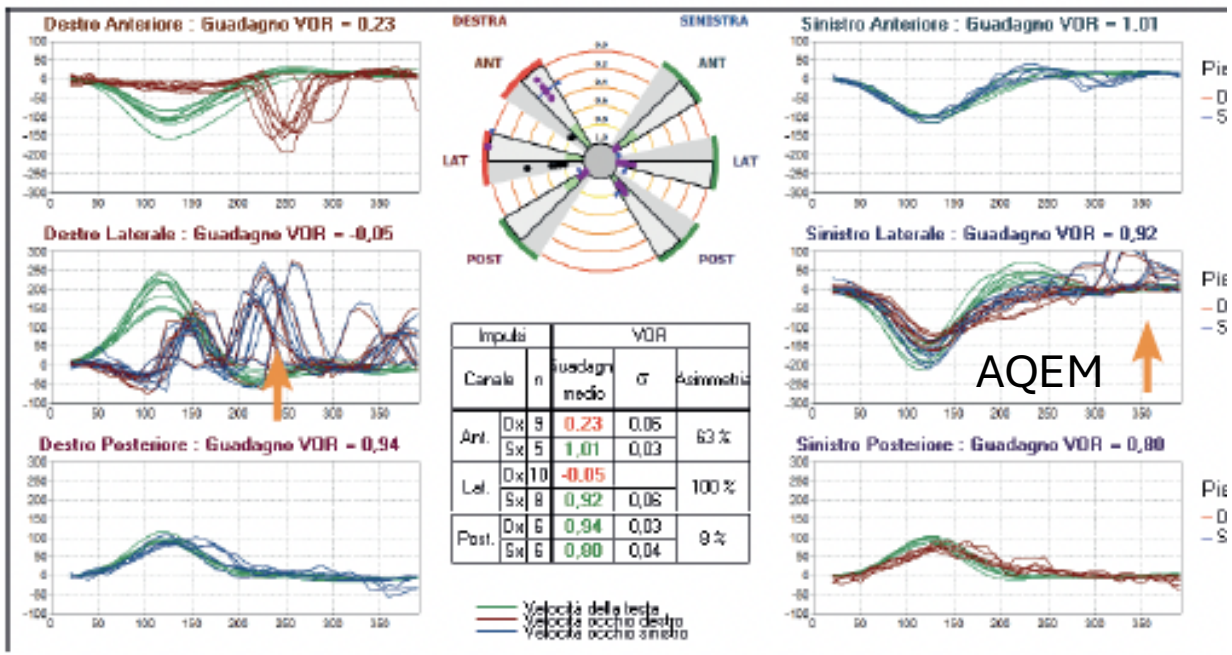


9 days after VRT

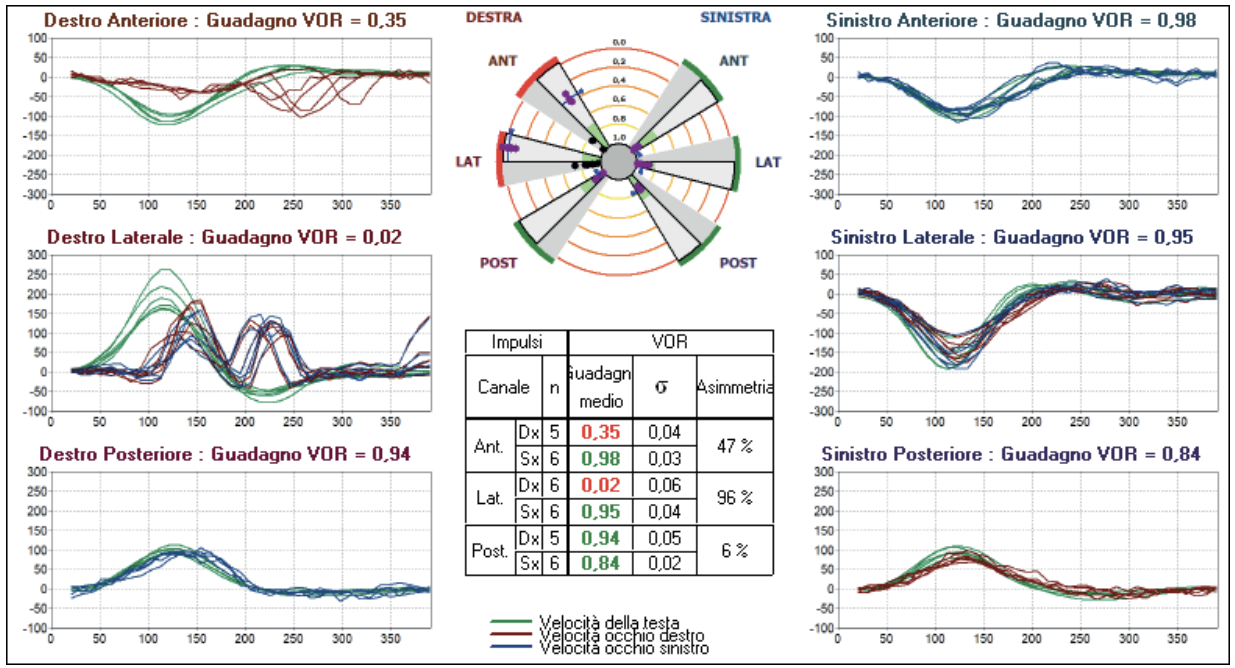


Essential for Monitoring Recovery

Scattered Overt saccades



Gathered overt saccades



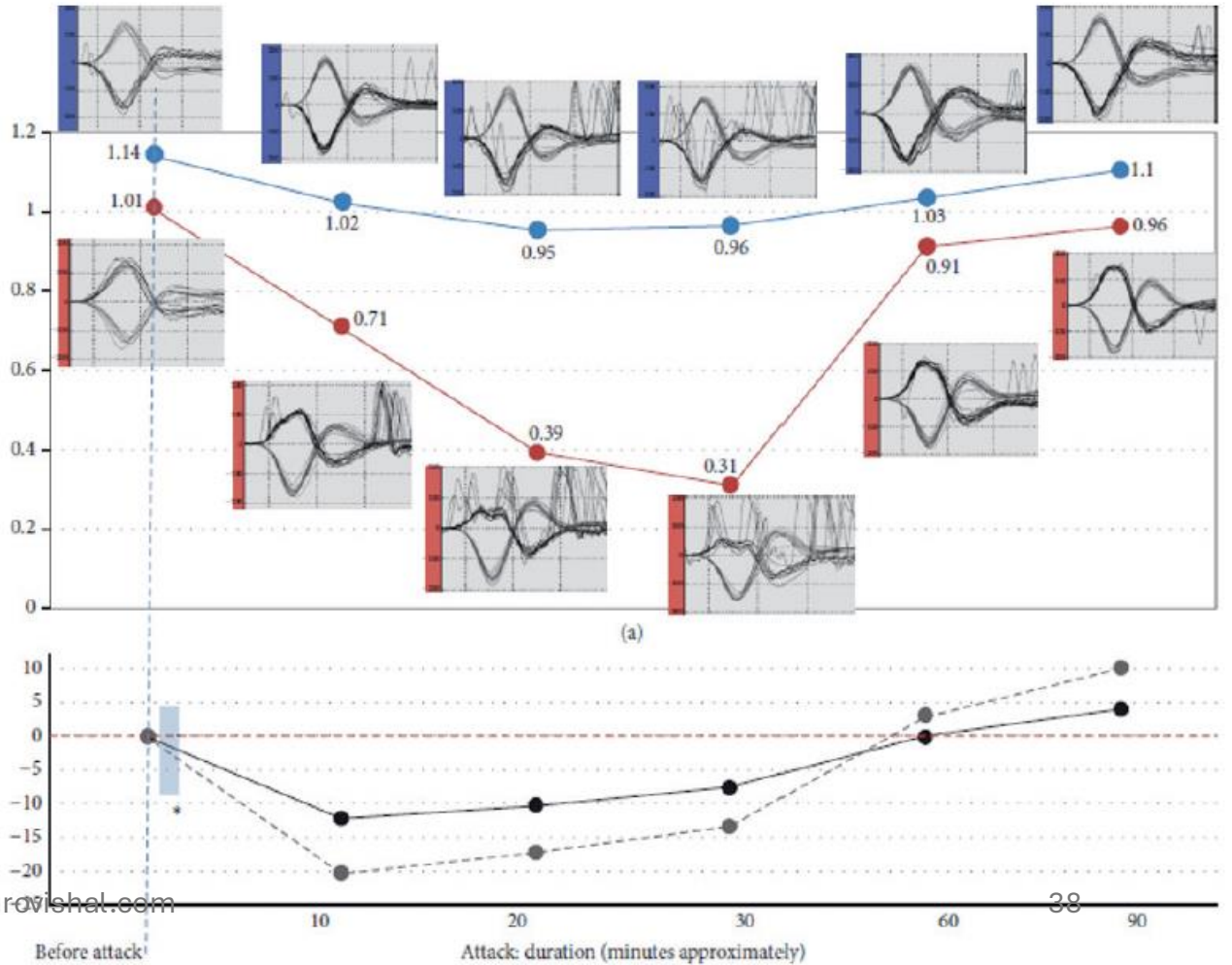
Findings in Menieres disease depend on stage

- During the attack
- Repeated attacks
- After repeated attacks in late stages
- After rehab
- After gentamicin injection



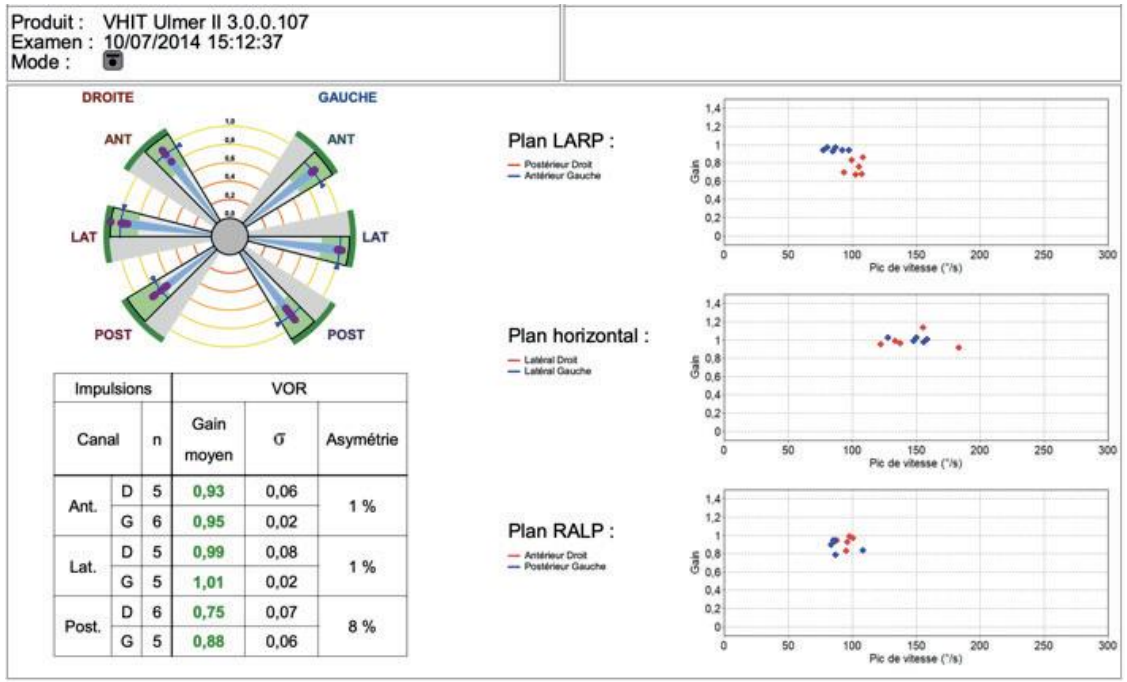


During the attack



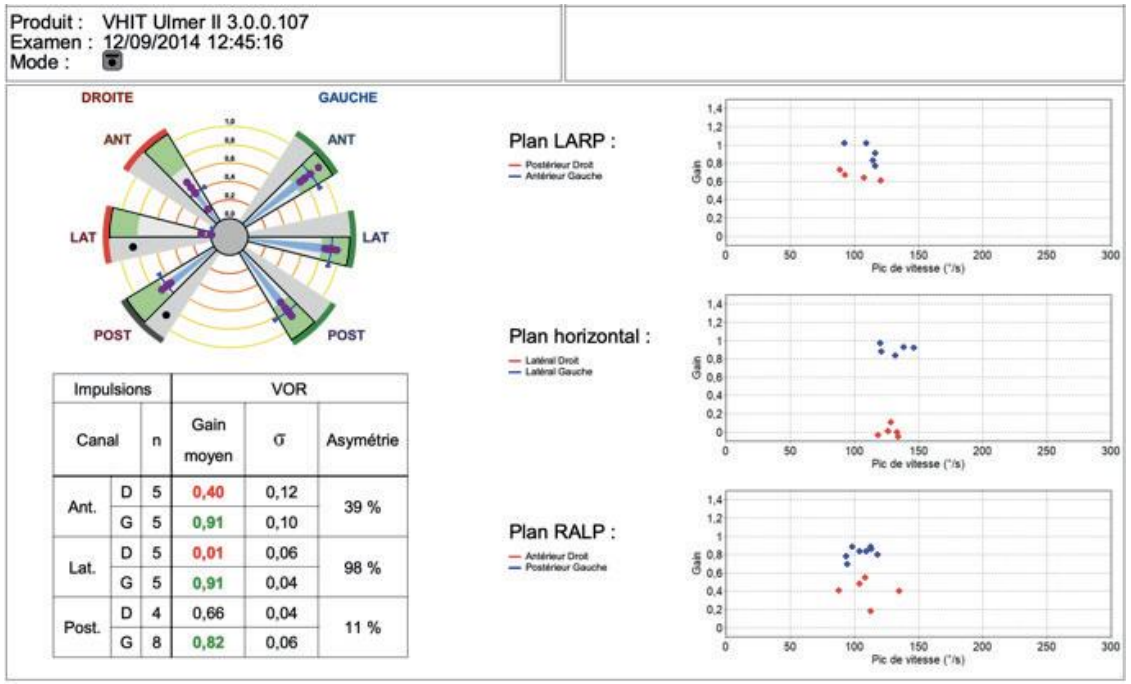
Yacovino DA.
Chapter: *vHIT in Ménière's Disease*.
In: vHIT Enrico Book, Chapter 6, pp. 119–121.
Referenced study: *Intra-Attack Vestibuloocular Reflex Changes in Ménière's Disease* (2016)

vHIT allows **individual canal assessment**, aiding in a **topographic diagnosis** of vestibular injury.



Impulsions			VOR		
Canal		n	Gain moyen	σ	Asymétrie
Ant.	D	5	0,93	0,06	1 %
	G	6	0,95	0,02	
Lat.	D	5	0,99	0,08	1 %
	G	5	1,01	0,02	
Post.	D	6	0,75	0,07	8 %
	G	5	0,88	0,06	

Between the attacks

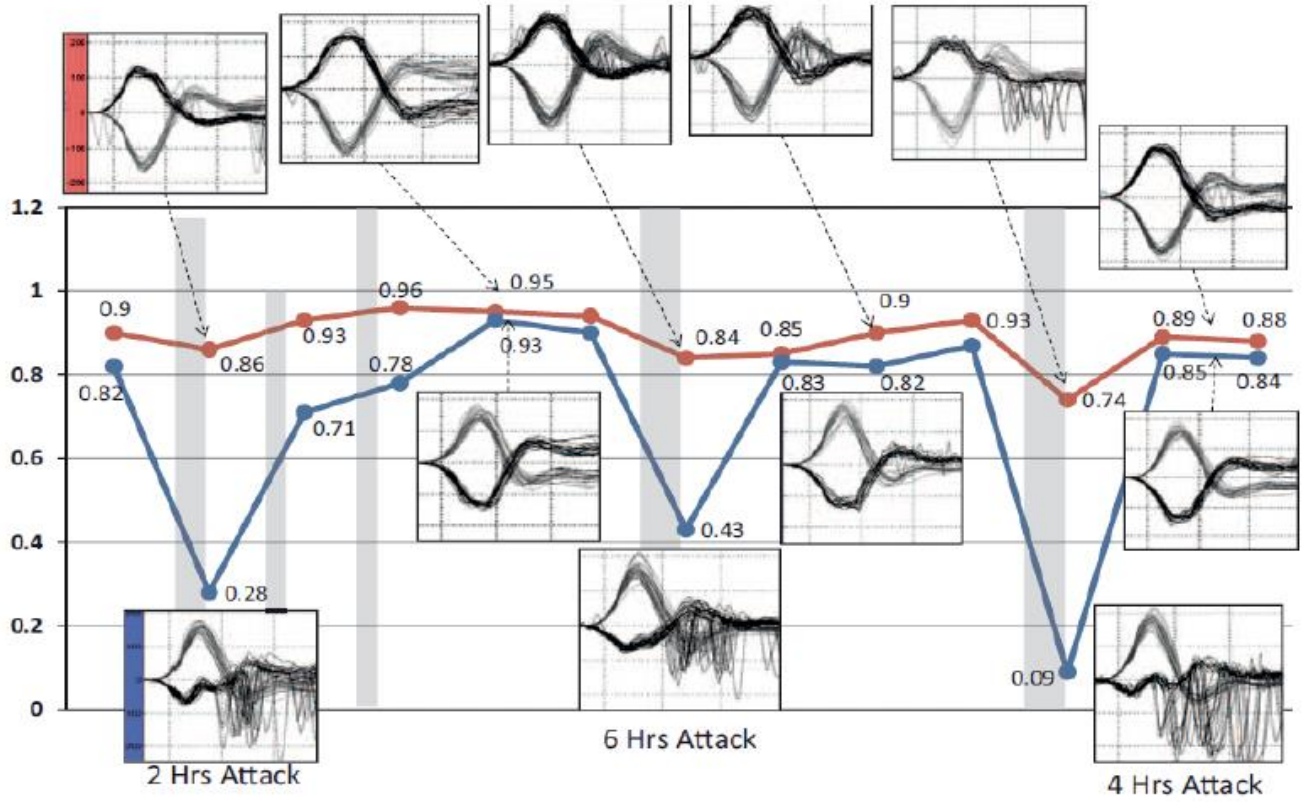


Impulsions			VOR		
Canal		n	Gain moyen	σ	Asymétrie
Ant.	D	5	0,40	0,12	39 %
	G	5	0,91	0,10	
Lat.	D	5	0,01	0,06	98 %
	G	5	0,91	0,04	
Post.	D	4	0,66	0,04	11 %
	G	8	0,82	0,06	

During the second attack

Tardivet L. 2020.

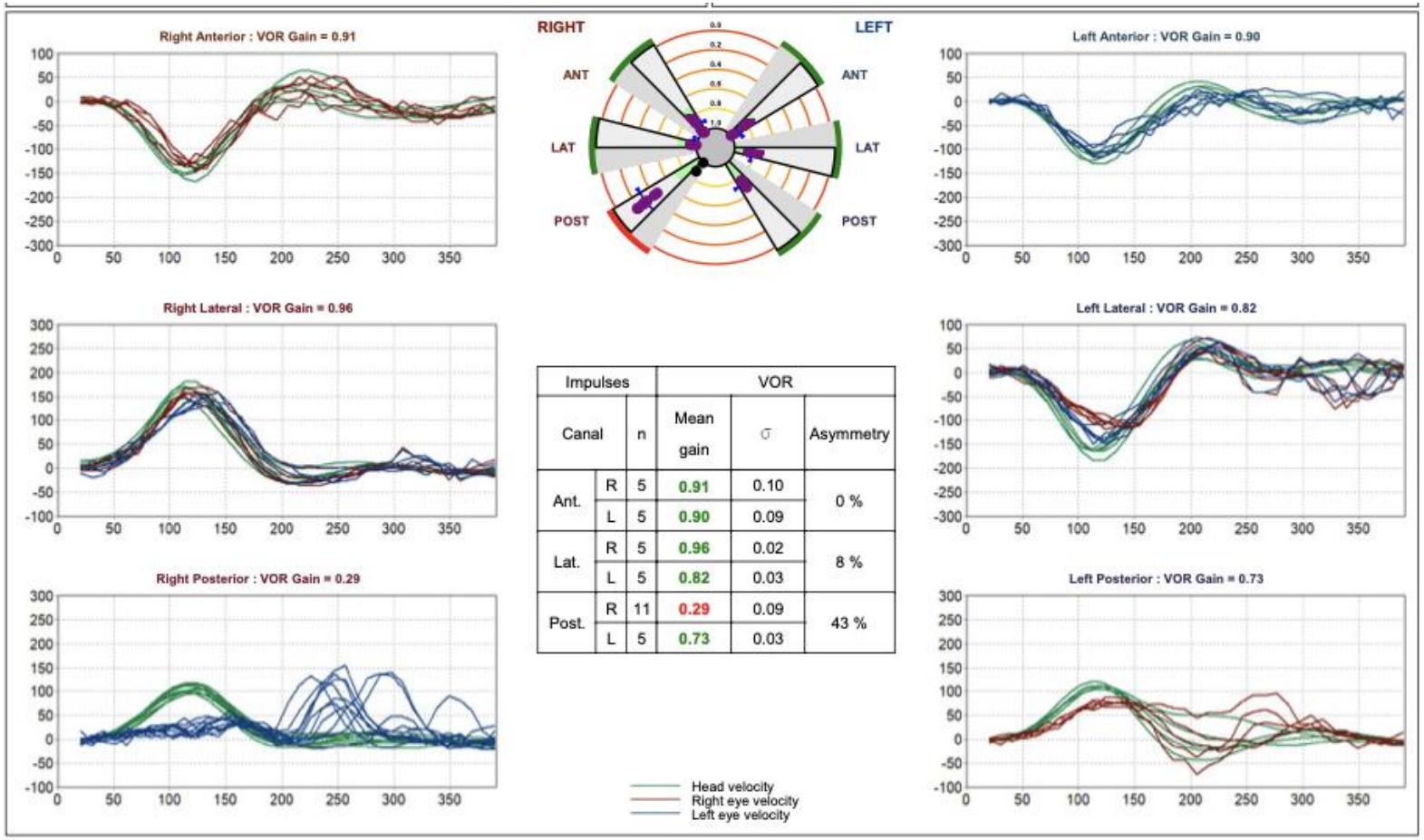
Monitoring the patient with repeated attacks



The v-HIT in Ménière disease

Yacovino DA, 2017.

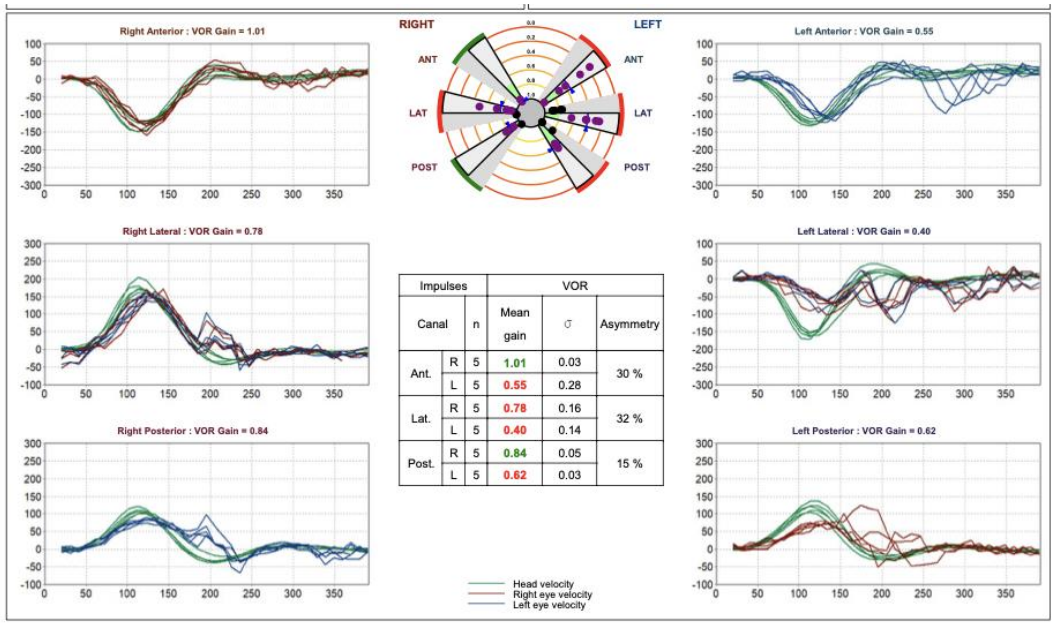
Late stages



Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	0.91	0.10	0 %
	L	5	0.90	0.09	
Lat.	R	5	0.96	0.02	8 %
	L	5	0.82	0.03	
Post.	R	11	0.29	0.09	43 %
	L	5	0.73	0.03	

Monitoring rehabilitation

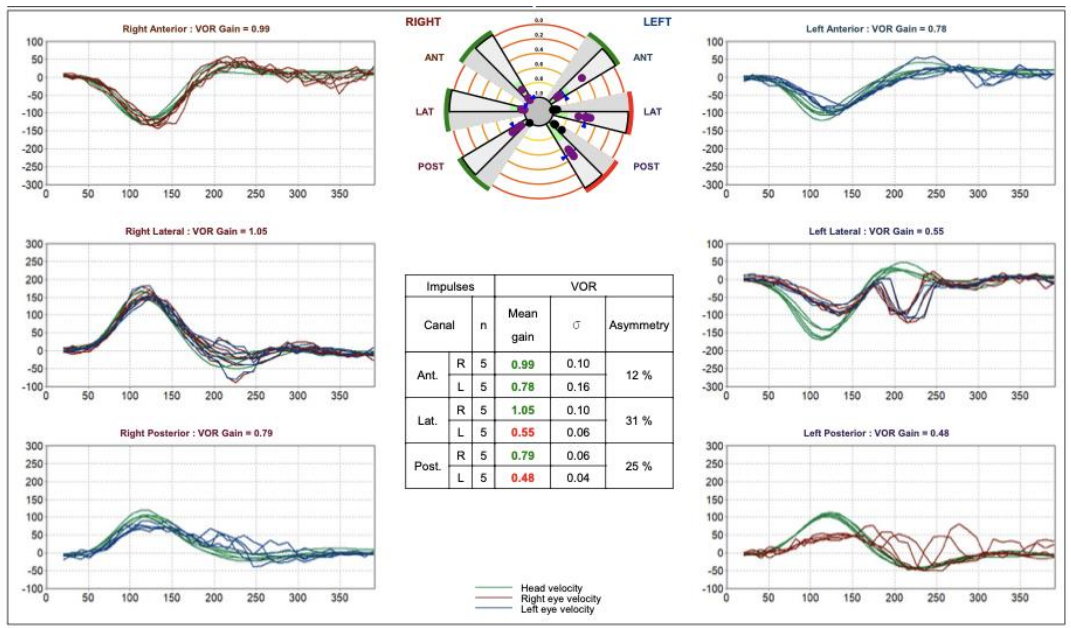
4 out of 6 canals – Low gain



Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	1.01	0.03	30 %
	L	5	0.55	0.28	
Lat.	R	5	0.78	0.16	32 %
	L	5	0.40	0.14	
Post.	R	5	0.84	0.05	15 %
	L	5	0.62	0.03	

4 sessions of vestibular physiotherapy

2 out of 6 canals – Low gain



Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	0.99	0.10	12 %
	L	5	0.78	0.16	
Lat.	R	5	1.05	0.10	31 %
	L	5	0.55	0.06	
Post.	R	5	0.79	0.06	25 %
	L	5	0.48	0.04	

Dissociation between vHITs and Caloric test

vHIT and calorics test different frequency ranges:

vHIT: High-frequency (5–7 Hz) stimulation

Caloric test: Low-frequency (~0.003 Hz) stimulation

Dissociation occurs when one test is normal and the other abnormal:

Reflects frequency-specific vestibular dysfunction:

type I – Vestibular neuritis (vHITs)

type II- Meniere's (Caloric)

Clinical relevance in diagnosis:

Helps distinguish **Ménière's disease** from **vestibular neuritis**.

Identifies **high-velocity deficits missed by calorics**.

Impacts clinical decision-making:

Supports **complementary testing** approach—neither test alone is sufficient.

Abnormal vHITs with normal calorics

Scenario	vHIT	Caloric
High-frequency loss	Abnormal	Normal
Selective vertical canal loss	Abnormal	Normal
Central compensation/saccades	Abnormal pattern	Normal
Early or partial vestibular loss	Abnormal	Borderline/Normal

Normal vHITs with abnormal calorics

Condition	Explanation
Early Ménière's Disease	Low-frequency VOR affected first
Compensated Vestibular Neuritis	High-frequency VOR recovers before low-frequency
Aging (Presbyvestibulopathy)	Age-related low-frequency decline

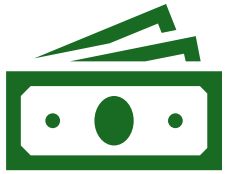
Post intratympanic gentamicin therapy (chemical labyrinthectomy)

Confirm

Confirm successful vestibular ablation in treatment-resistant MD.

Monitor

Monitor extent of canal ablation.



Track

Track compensation via gain recovery or saccadic reduction.

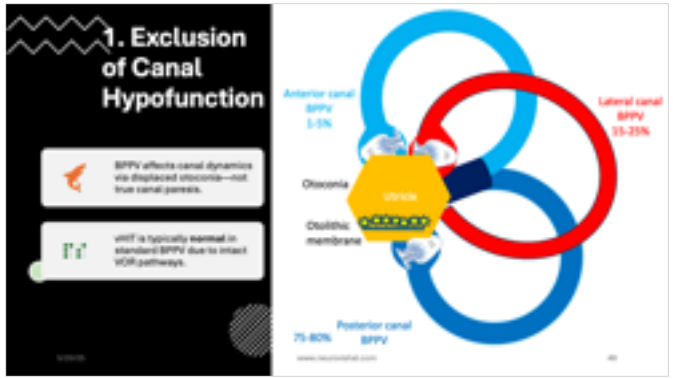
Integration with Other Vestibular Tests

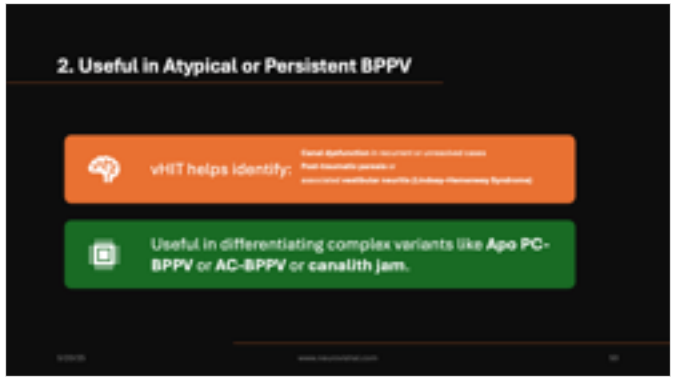
vHIT is most informative when combined with:

- Calorics (for low-frequency function)
- VEMPs (for otolith involvement)
- Audiometry (to track cochlear progression)

Together, they provide a **comprehensive view** of disease staging and prognosis.

Role of vHITs in BPPV

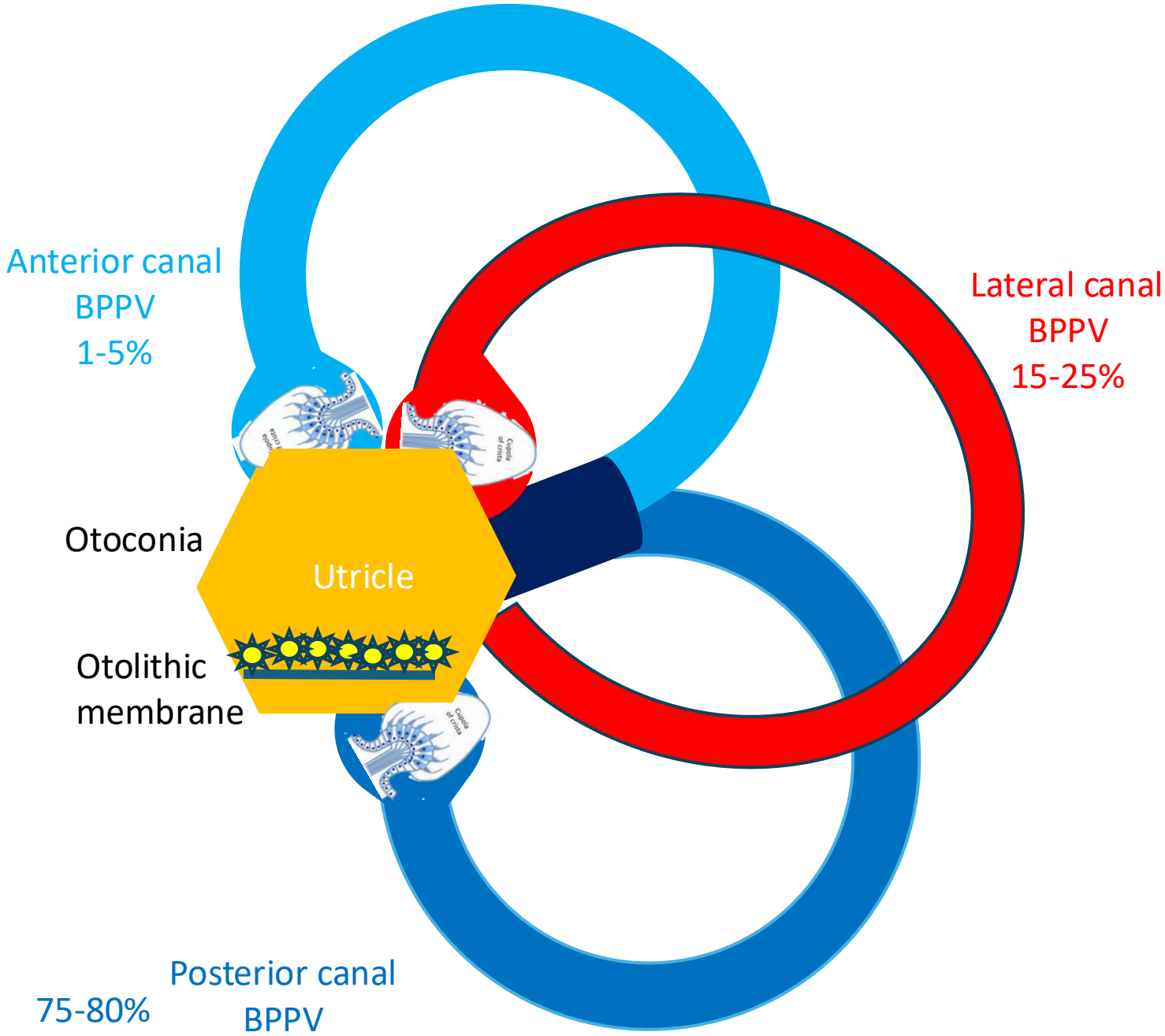




1. Exclusion of Canal Hypofunction

BPPV affects canal dynamics via displaced otoconia—not true canal paresis.

vHIT is typically **normal** in standard BPPV due to intact VOR pathways.



2. Useful in Atypical or Persistent BPPV

vHIT helps identify:

Canal dysfunction in recurrent or unresolved cases
Post-traumatic paresis or
associated **vestibular neuritis (Lindsay–Hemenway Syndrome)**

Useful in differentiating complex variants like **Apo PC-BPPV** or **AC-BPPV** or **canalith jam.**

3. Evaluation Post-Treatment

Confirms **canal function integrity** after maneuvers

Detects **canalith jam**

Temporary reduction in VOR gain before maneuver	normalization after liberatory maneuvers	supports reversible cupular-endolymph dysfunction

4. Differentiating Central Positional Vertigo

Persistent or **positional down-beating nystagmus (DBN)** can be challenging to interpret.

Abnormal vHIT:

- Suggests **peripheral** involvement **saccades**, especially in the context of DBN, it supports a **peripheral origin** such as **AC-BPPV** or **apogeotropic PC-BPPV**.

Normal vHIT with **DBN:**

- Raises suspicion of **central causes** (e.g., cerebellar lesions).

5. Complementary to Positional Testing

Not diagnostic alone, but enhances:

- **Dix–Hallpike**, **Roll**, and **Supine tests**

Offers **canal-specific function** insights in ambiguous cases.

Diagnostic criteria for bilateral vestibulopathy

A. Chronic vestibular syndrome with the following symptoms

- 1. Unsteadiness when walking or standing[1] plus at least one of 2 or 3
- 2.Movement-induced blurred vision or oscillopsia during walking or quick head/body movements[2] and/or
- 3.Worsening of unsteadiness in darkness and/or on uneven ground[3]

B. No symptoms while sitting or lying down under static conditions[4]

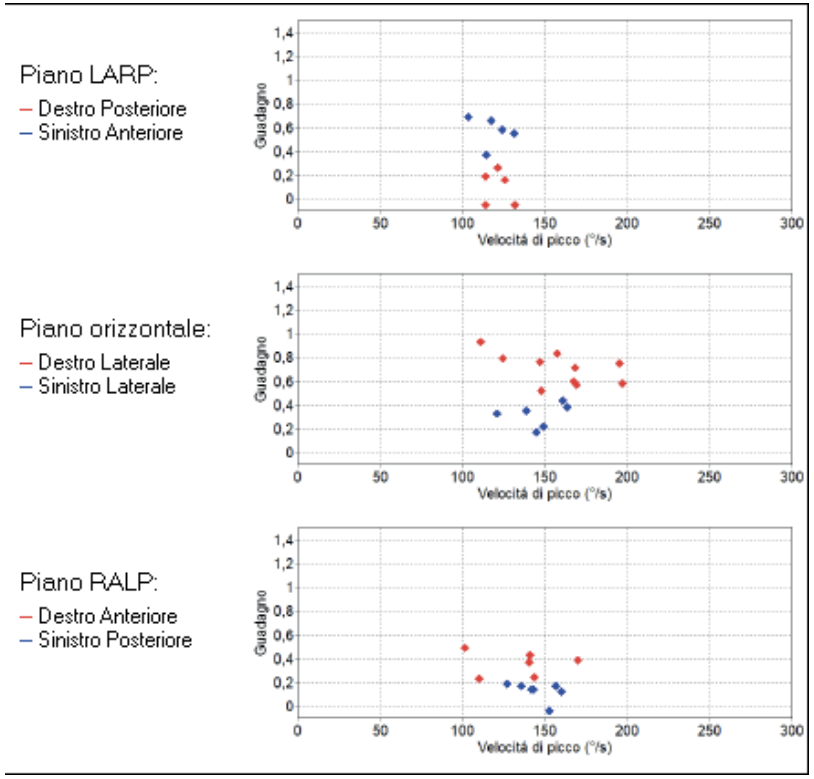
C. Bilaterally reduced or absent angular VOR function documented by

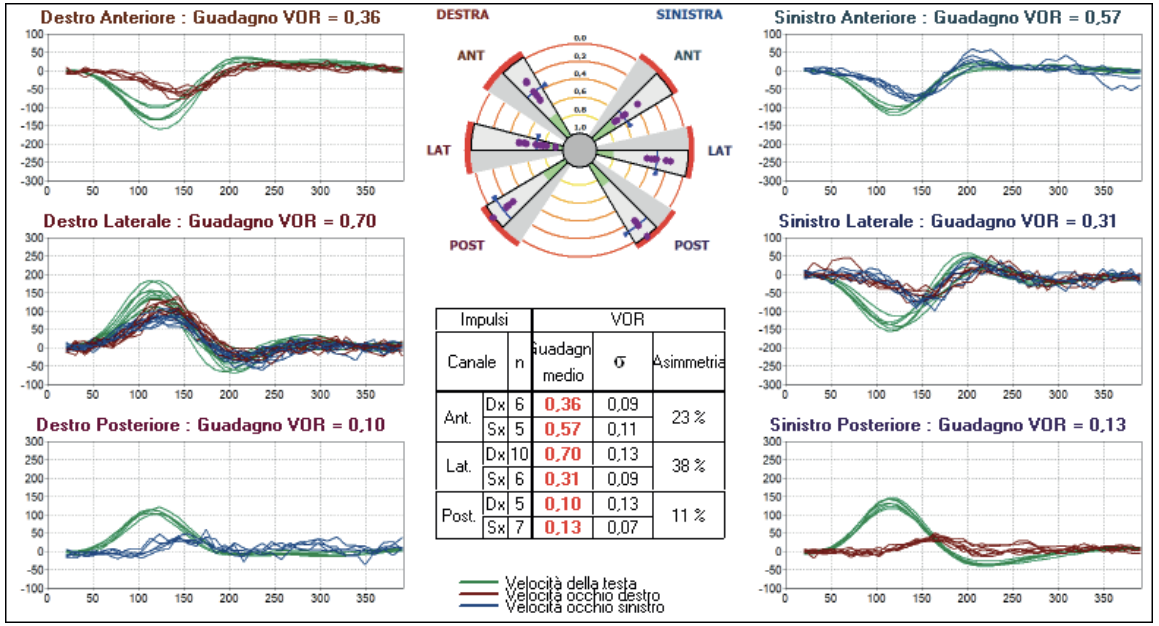
- •Bilaterally pathological horizontal angular VOR gain <0.6, measured by the video-hit[5] or scleral-coil technique and/or
- •Reduced caloric response[6] (sum of bithermal max. Peak SPV on each side <6°/sec[7]) and/or
- •Reduced horizontal angular VOR gain <0.1 upon sinusoidal stimulation on a rotatory chair (0.1 hz, vmax=50°/sec) and a phase lead >68 degrees (time constant <5 sec).

D. Not better accounted for by another disease

Key Diagnostic Features on vHIT

- **Bilateral reduced VOR gain** across horizontal and vertical canals.
- Lack of asymmetry helps differentiate BVH from unilateral deficits.
- Gain reduction correlates with **head velocity increase**
- Detects whether loss is **partial vs. complete**, based on residual gain.
- Guides **rehabilitation planning** by mapping preserved canal function





Diagnostic criteria for presbyvestibulopathy (PVP)

Each of the criteria A through D have to be fulfilled

A. Chronic vestibular syndrome (at least 3 months duration) with at least 2 of the following symptoms:[1]

1. Postural imbalance or unsteadiness
2. Gait disturbance
3. Chronic dizziness
4. Recurrent falls

B. Mild[2] bilateral peripheral vestibular hypofunction documented by at least 1 of the following:

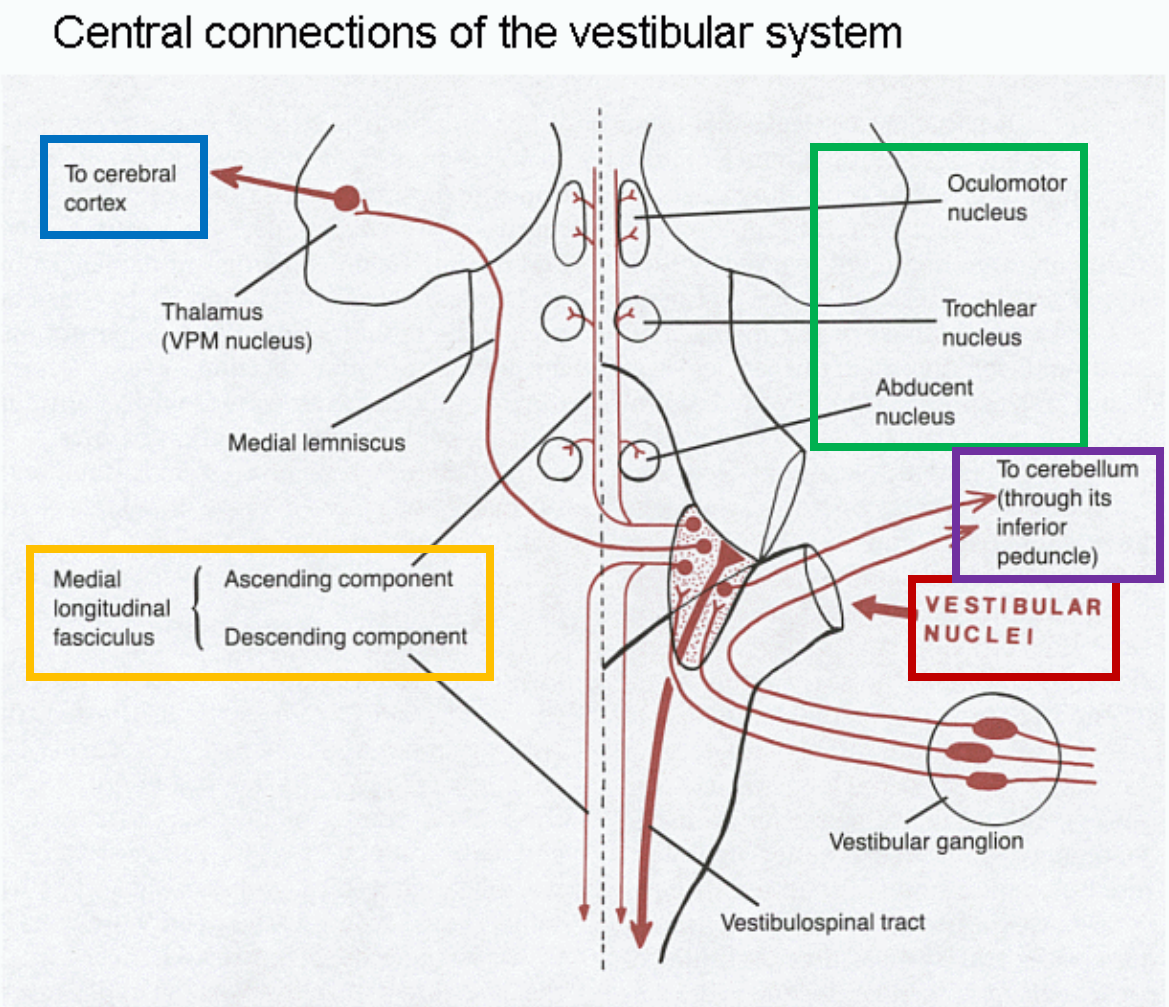
1. VOR gain measured by video-HIT between 0.6 and 0.8[3] bilaterally
2. VOR gain between 0.1 and 0.3 upon sinusoidal stimulation on a rotatory chair (0.1 Hz, Vmax = 50–60°/sec)[4]
3. Reduced caloric response (sum of bithermal maximum peak SPV on each side between 6 and 25°/sec)[5,6], [5,6]

C. Age≥60 years[7]

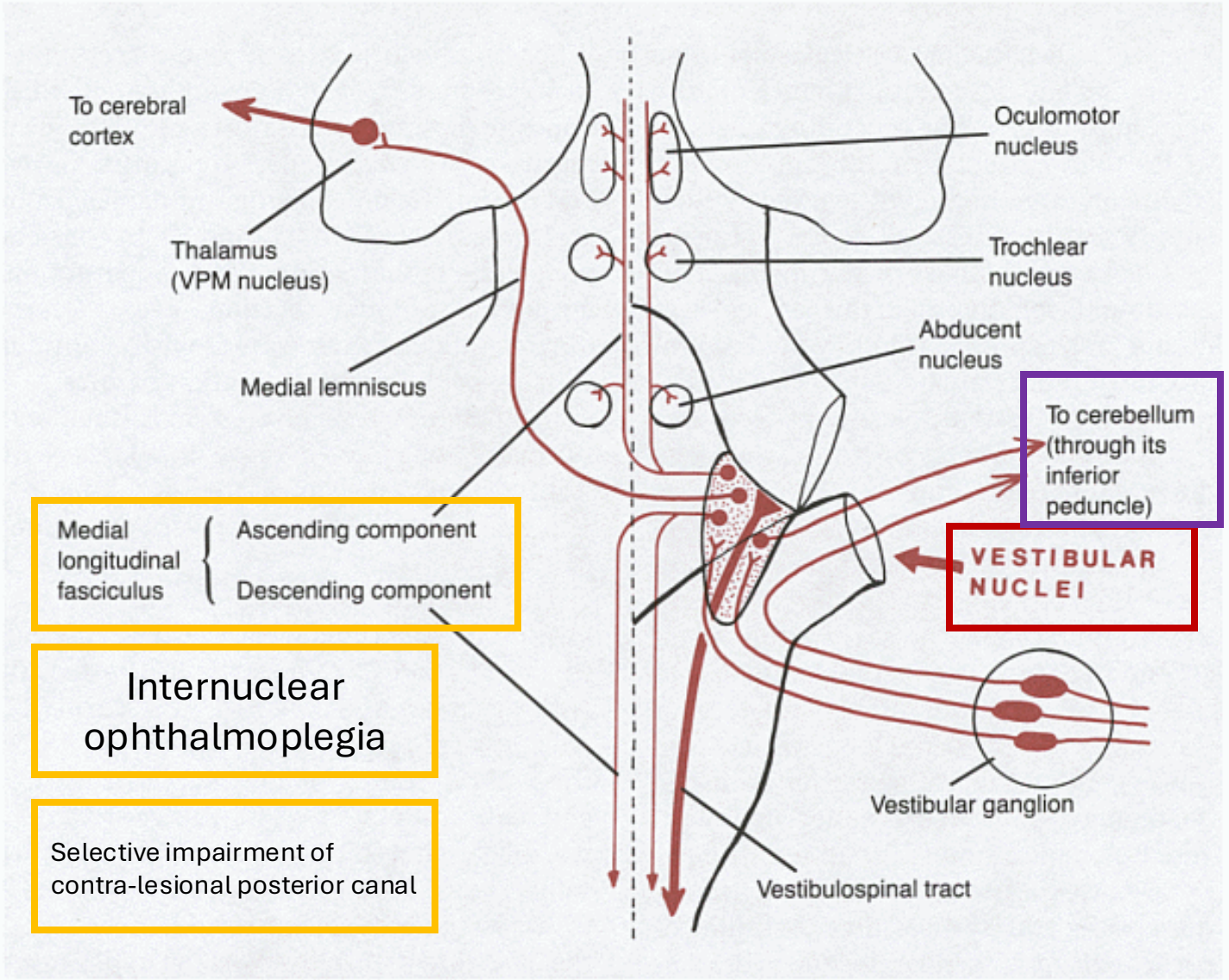
D. Not better accounted for by another disease or disorder[8]

Role of central structures in VOR

Structure	Role in VOR
Vestibular nuclei	First central relay; processes input from semicircular canals
Ocular motor nuclei (III, IV, VI)	Generate eye movements
Medial longitudinal fasciculus	Coordinates conjugate gaze
Flocculus and para-flocculus	Regulate and adapt VOR gain
Nodulus and uvula	Handle prolonged motion and otolith signals
Cortex (parietal, frontal areas)	Modulate voluntary and perceptual aspects of gaze



Central connections of the vestibular system



Internuclear ophthalmoplegia

Selective impairment of contra-lesional posterior canal

Dis-conjugate horizontal VOR: severe loss in adducting eye without saccades

CANVAS, SCA, Cerebellar degeneration

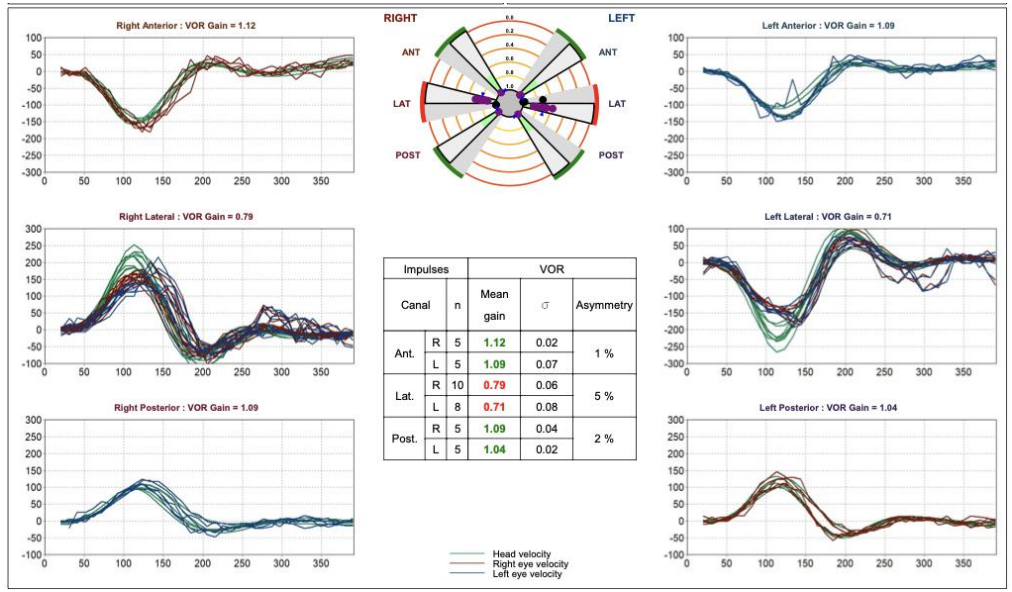
Pontine/Lateral medullary stroke

Impaired VOR on cHITs/vHITs

Condition	Observed VOR Gain Abnormality	Proposed Mechanism
Parkinson's Disease (early stages)	Elevated VOR gain (~1.20–1.23 vs ~0.98 in controls)	Central compensation or altered gain control due to basal ganglia dysfunction
Vestibular Migraine (occasionally)	Mildly increased VOR gain	Altered central processing of vestibular signals
Fixation on Near Targets	Increased VOR gain as target distance decreases	Physiological enhancement of VOR to stabilize gaze on closer objects
Dance Training	Higher VOR gain, especially in left-head impulses	Adaptive modulation of eye-head coordination due to repetitive vestibular stimulation
Goggle Slippage Artifact	Artificially high VOR gain values	Loose vHIT goggle strap causes slingshot effect mimicking high gain

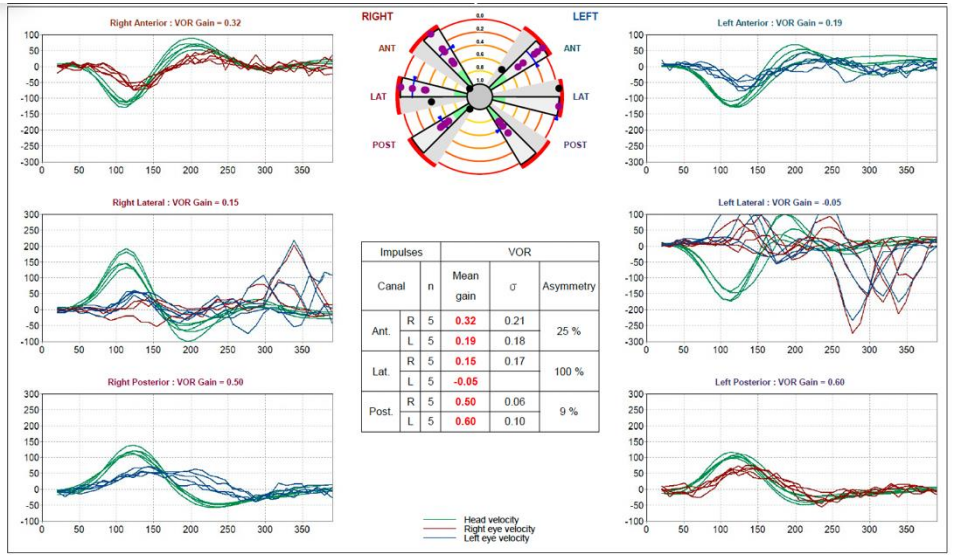
vHIT in Thiamine Deficiency (Wernicke's Encephalopathy)

Primary finding:	Selective **bilateral horizontal semicircular canal (HSC) dysfunction** with **preserved vertical canal function** is a hallmark pattern .
Mechanism:	Involvement of **medial vestibular nuclei (MVN)** due to thiamine depletion—especially vulnerable in early Wernicke's encephalopathy .
Associated signs:	Horizontal VOR gain loss, spontaneous/gaze-evoked nystagmus, ataxia, and ophthalmoparesis .
Clinical Utility:	Detects **pre-encephalopathy vestibular dysfunction** even with a normal MRI. Assists in **early diagnosis and monitoring response to thiamine replacement**. Offers **objective, quantitative insight** into central vestibular involvement.



Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	1.12	0.02	1 %
	L	5	1.09	0.07	
Lat.	R	10	0.79	0.06	5 %
	L	8	0.71	0.08	
Post.	R	5	1.09	0.04	2 %
	L	5	1.04	0.02	

CANVAS



Impulses			VOR		
Canal		n	Mean gain	σ	Asymmetry
Ant.	R	5	0.32	0.21	25 %
	L	5	0.19	0.18	
Lat.	R	5	0.15	0.17	100 %
	L	5	-0.05		
Post.	R	5	0.50	0.06	9 %
	L	5	0.60	0.10	

- **Classic triad**:
  - Cerebellar ataxia, sensory neuropathy, and bilateral vestibular areflexia.
- **vHIT findings**:
  - Marked bilateral **low VOR gain** across all six semicircular canals—especially horizontal and anterior canals .
- **Early marker**:
  - Horizontal vHIT loss may **precede cerebellar signs**, aiding early diagnosis .
- **Progressive loss**:
  - Longitudinal vHIT shows **declining gain over time**, correlating with disease duration and male sex .
- **Diagnostic value**:
  - Combined with tuning fork apallesthesia and MRI (vermal atrophy), vHIT supports **functional confirmation** of CANVAS .

Central signs in vHITs and localisation

Central Sign on vHIT	Possible Location
Normal vHIT with severe symptoms	Cortical or cerebellar lesion

vHIT Applications in Specialized Vestibular Disorders

🧪 Ototoxic Vestibulopathy (e.g., Gentamicin)

- Detects **bilateral high-frequency canal loss**
- Useful for **monitoring progression** and **treatment impact**

🦻 Post-Cochlear Implantation

- Assesses **iatrogenic damage** to canals, especially **posterior canal**
- Pre-/post-op comparison helps determine **vestibular preservation**

🧠 Vestibular Schwannoma

- Identifies **asymmetrical canal hypofunction**
- Supports **lesion lateralization** and **surgical planning**

🔍 Canal Dehiscence / Fistulae (Suspected)

- vHIT may be **normal** in these cases
- Helps rule out **true canal paresis**, guiding toward **third-window diagnosis**

💥 Post-Traumatic Vestibulopathy

- Reveals **selective canal deficits** (e.g., posterior canal)
- Useful for evaluating **injury extent and recovery**

👶 Pediatric Vestibular Disorders

- Non-invasive, tolerable test for children
- Detects **congenital or acquired canal dysfunction** without sedation

Some signature abnormalities

vHIT Abnormality	Description	What to Suspect
Low VOR Gain (<0.8 lateral, <0.7 vertical)	Indicates reduced semicircular canal function	Vestibular neuritis, bilateral vestibulopathy, vestibular schwannoma, MD

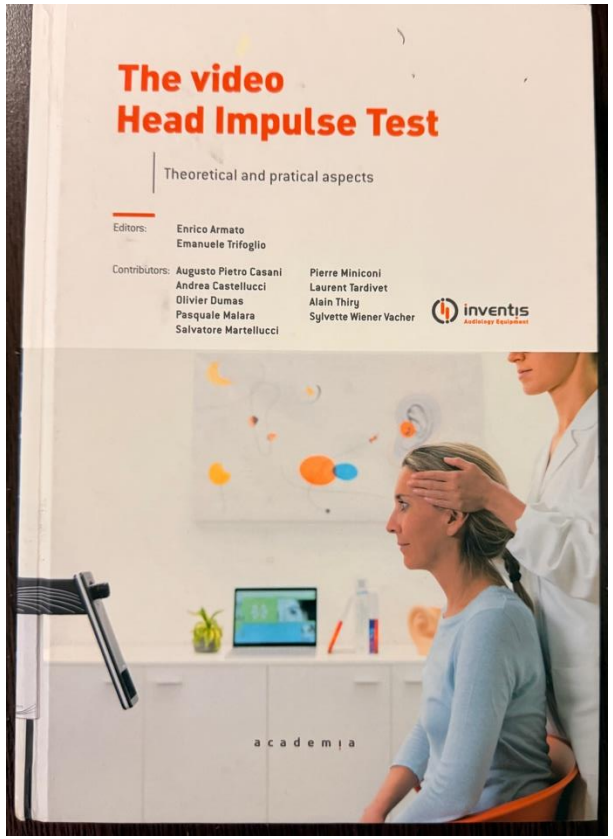
Take home message

Diagnosis of Vestibular Disorders:

- Primary tool for identifying peripheral vestibular hypofunction.

References

- International journal of vestibular research
- 1111 articles from pubmed



Thanks to

- Anna
- Mario
- Ganesh

Thank you

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The frequency spect

Frequency Range	Test Type	Gain	Phase
Very Low (0.003 Hz)	Caloric	Low (~0.3)	High lag (60–90°)
Low–Mid (0.1–0.5 Hz)	Rotatory Chair	Moderate–High (~0.6–1)	Reducing lag
Mid–High (1–6 Hz)	vHIT / HST / VAT	High (~1)	Near 0° (optimal)
Very High (>100 Hz)	VEMPs (cVEMP/oVEMP)	Not gain-based	Not measured (EMG)