Clinical Application of Vestibular Function Test

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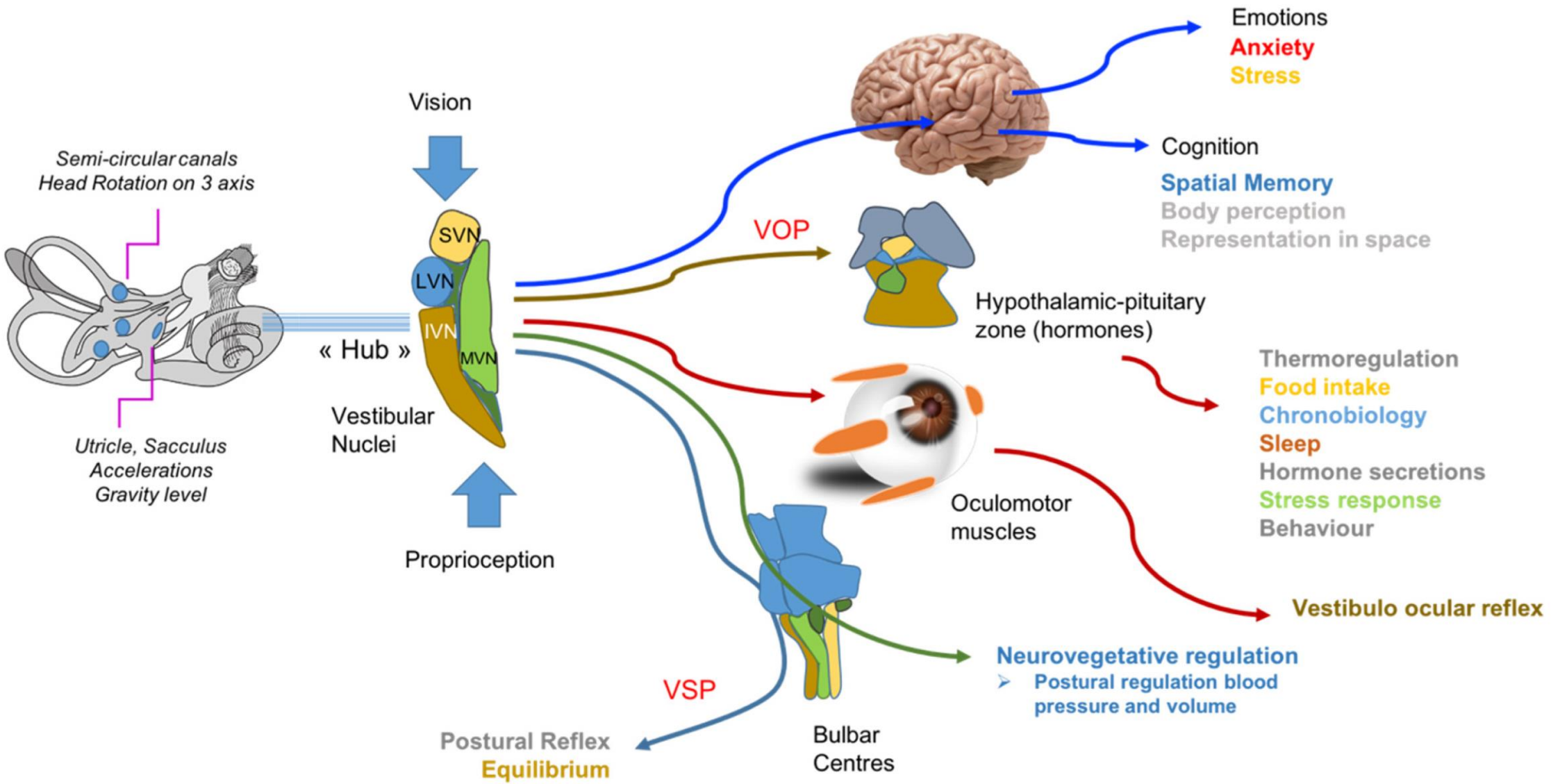
outline

- Overview of vestibular function test (VFT)
- Role of VFT in TITRATE approach to diagnosing dizziness/vertigo
- Selection of VFT in daily practice

overview of VFT

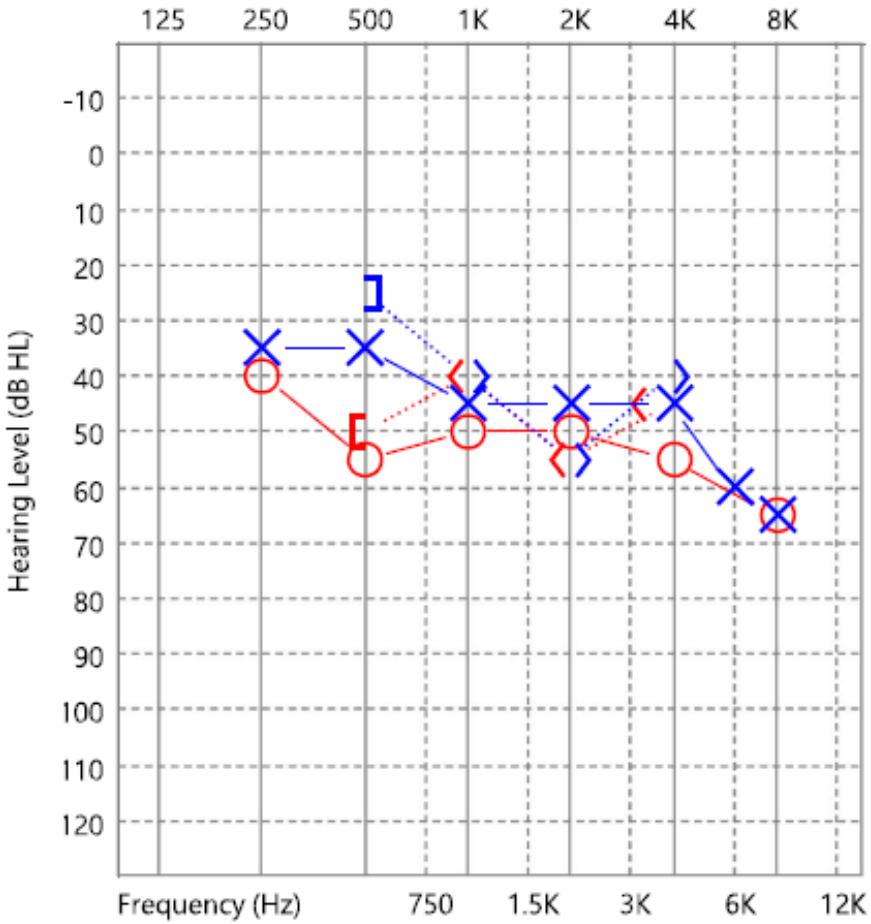
The vestibular system contributes to the perception of self-motion and orientation, ensuring stable gaze and posture



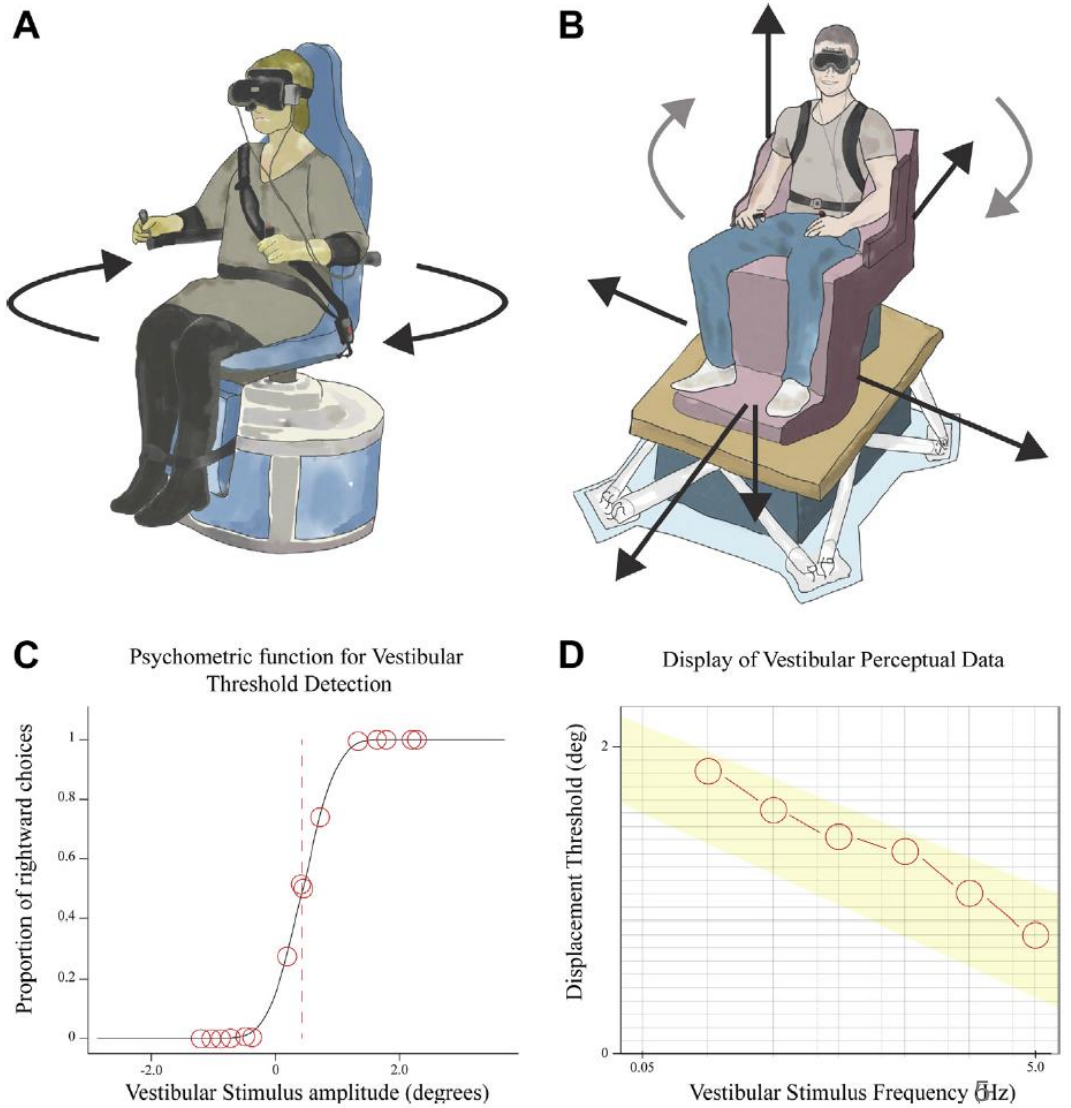
Brain Sci. 2022, 12, 592.

Perceptual threshold testing

pure tone audiometry (PTA)



vestibulometry ?



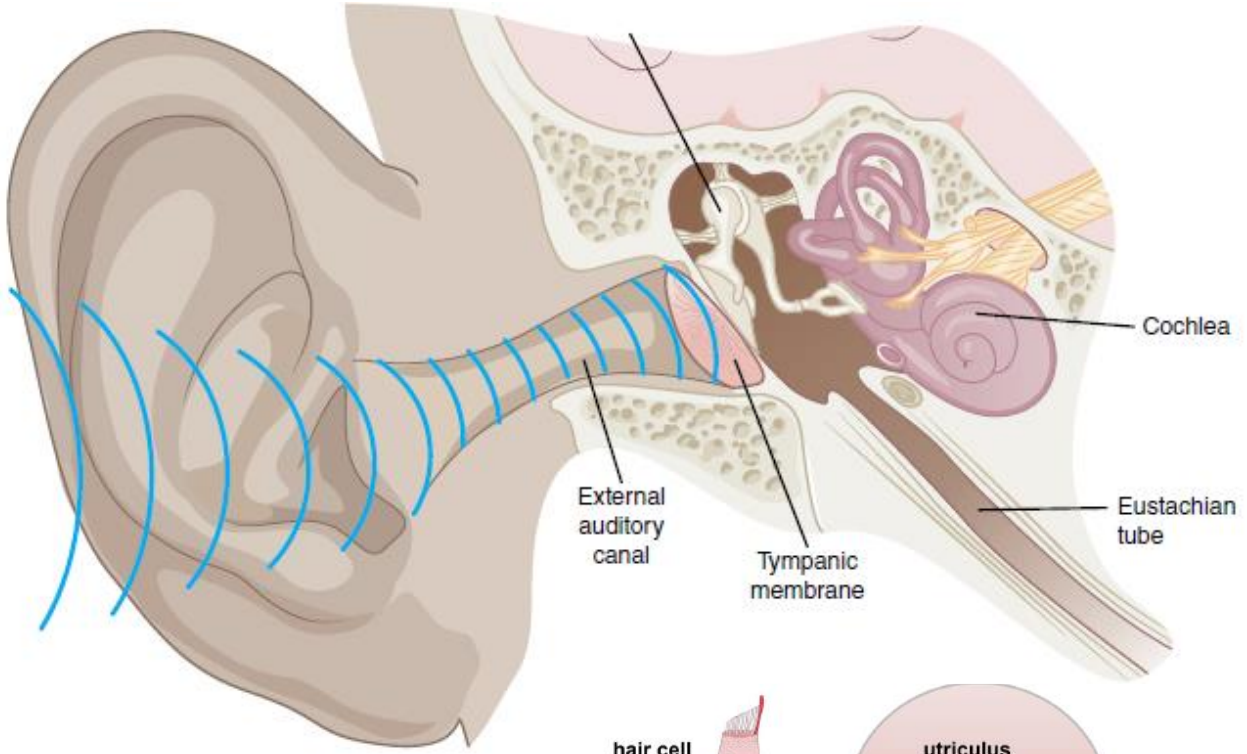
Otolaryngol Clin N Am 54 (2021) 1069–1080

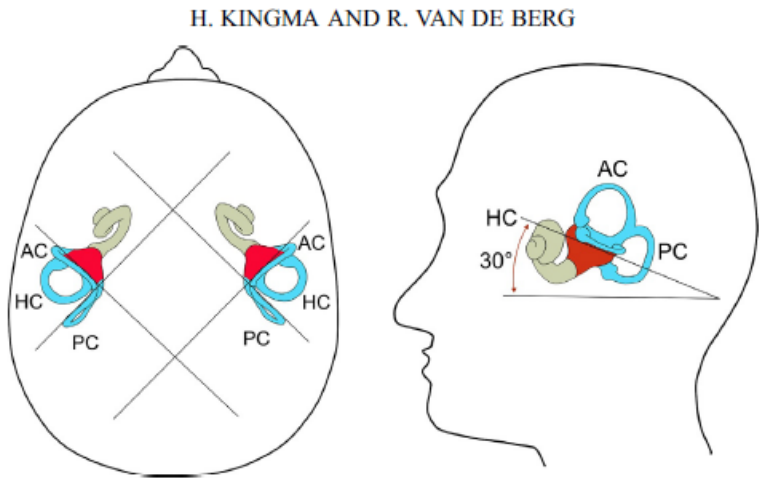
Testing of vestibular function

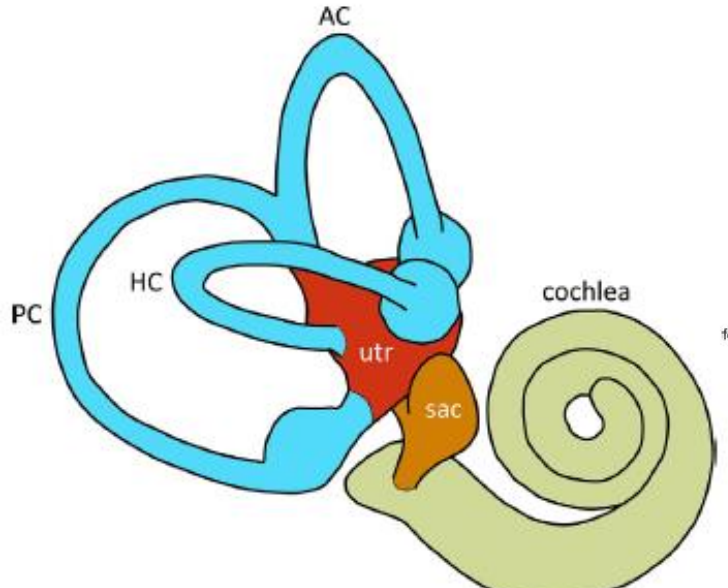
- The vestibulo-ocular reflex (VOR)
  - Caloric test , rotational chair, video head impulse test (vHIT)
  - Ocular vestibular evoked myogenic potentials (oVEMP)
  - Ocular counterrolling (OCR)
- The vestibulocollic reflex (VCR)
  - Cervical vestibular evoked myogenic potentials (cVEMP)
- The vestibulospinal reflex (VSR)
  - Romberg test
  - Posturography

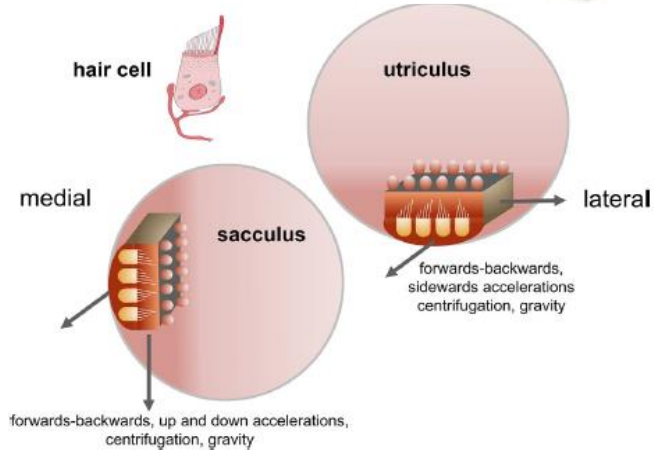
Membranous labyrinth

- Otolith organ
  - Utricle & saccule
  - Response to liner head motion and static head tilt with respect to the gravitational axis
- Semicircular canals
  - Anterior, horizontal, and posterior
  - Head rotational rate sensors







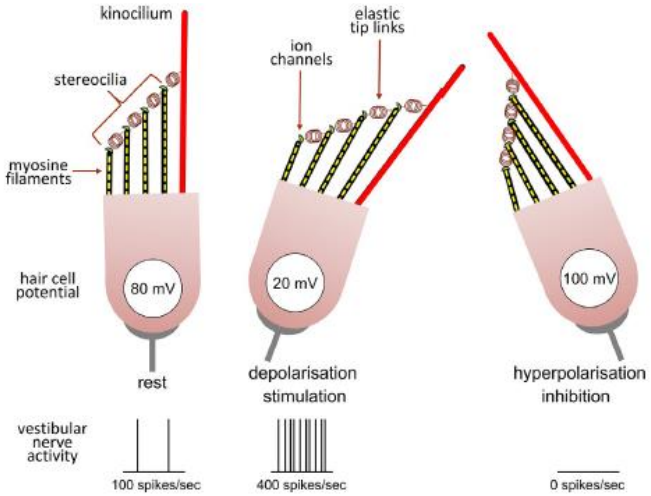


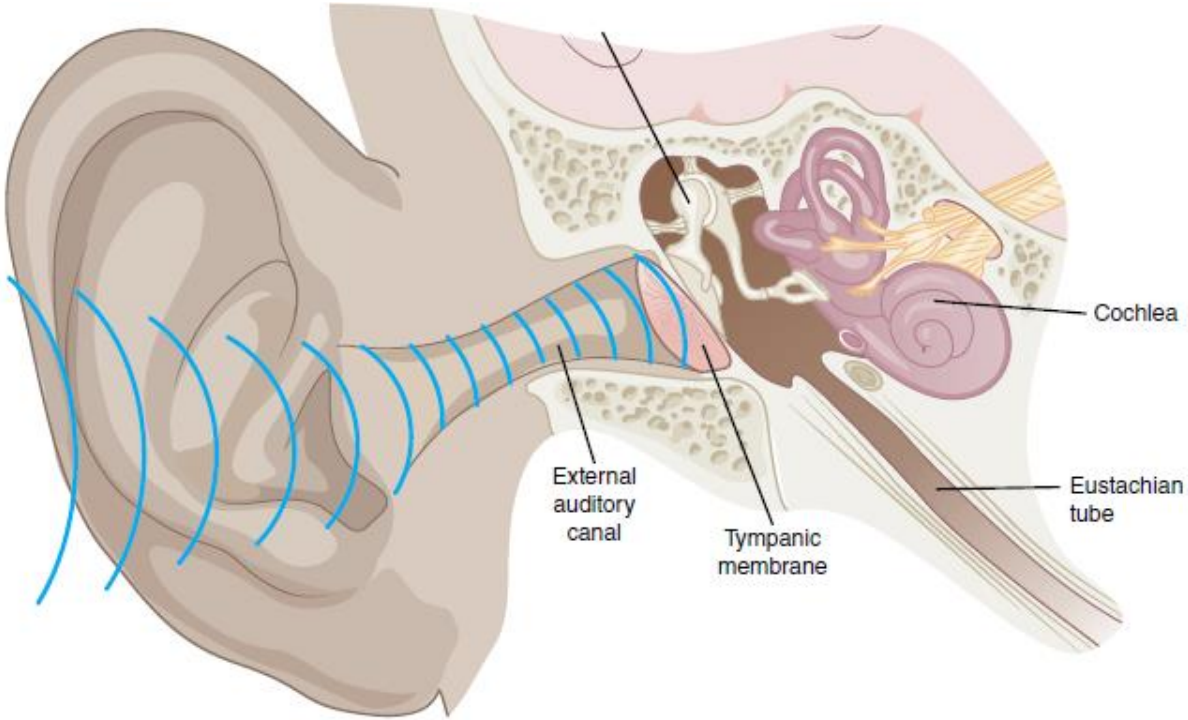
Handbook of Clinical Neurology, Vol. 137 (3rd series) *Neuro-Otology*

Title: Fundamentals of audiology for the speech-language pathologist/ Deborah Welling and Carol Ukstins.

Vestibular hair cells

- Primary motor sensors – transform a mechanic displacement into electric energy
- Asymmetric sensitive mechanoreceptor cell (2nd law of Ewald)
- Semicircular canals
  - Angular acceleration detect (>0.5 °/s²)
- Otolith organs
  - Liner acceleration detect (>2cm /s²)
  - Angular acceleration detect (>3.0 °/s²)
  - Head tilt accuracy about 0.5 °
  - Response to sound





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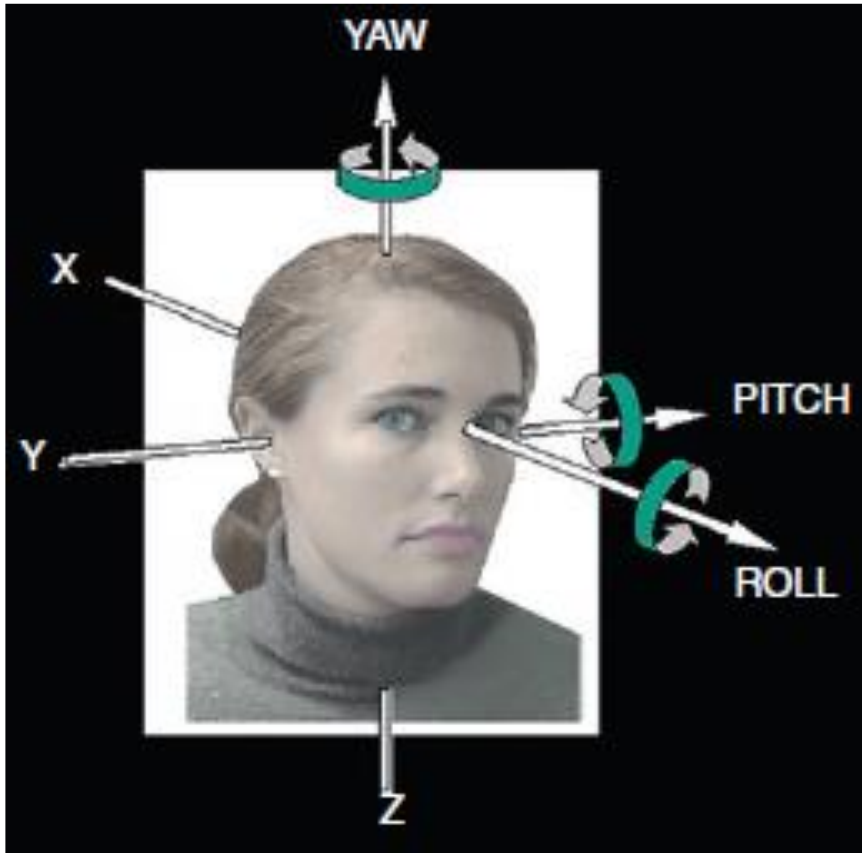
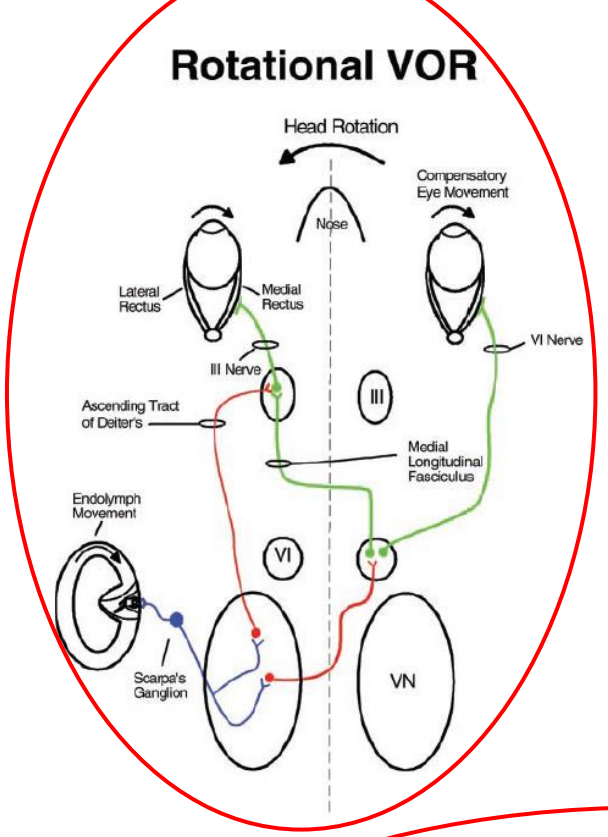
Deborah Welling and Carol Ukstins.

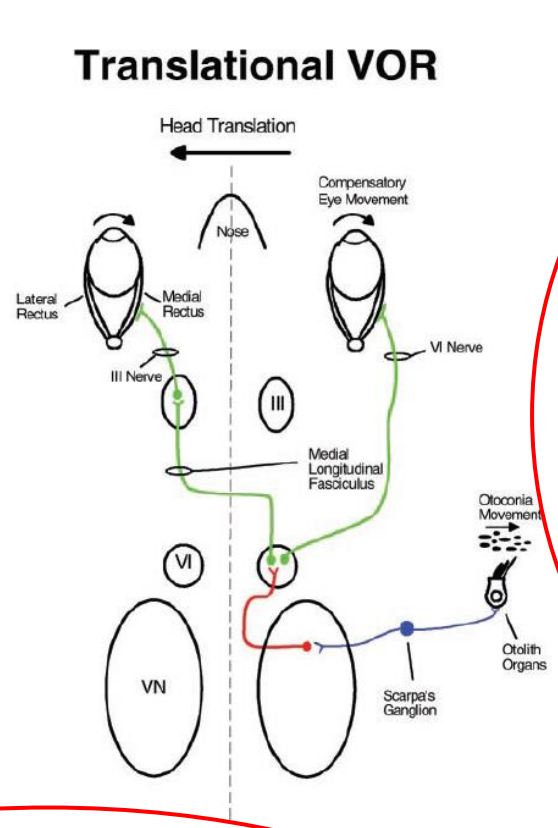
VOR

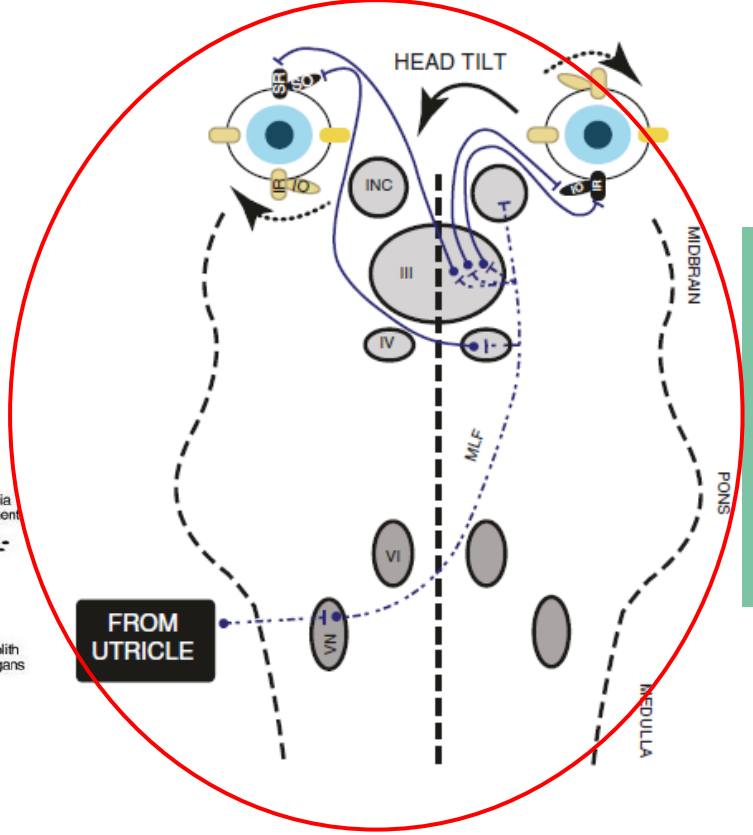
- Keep the image of outside world still when head moves
  - Angular VOR (aVOR) - rotation
  - Translational VOR (tVOR) – linear
  - Tortional VOR (OCR)– head static tilt in roll plane
- aVOR
  - One of the fastest reflex in the human body
  - Produce equal and opposite eye movement to head movement

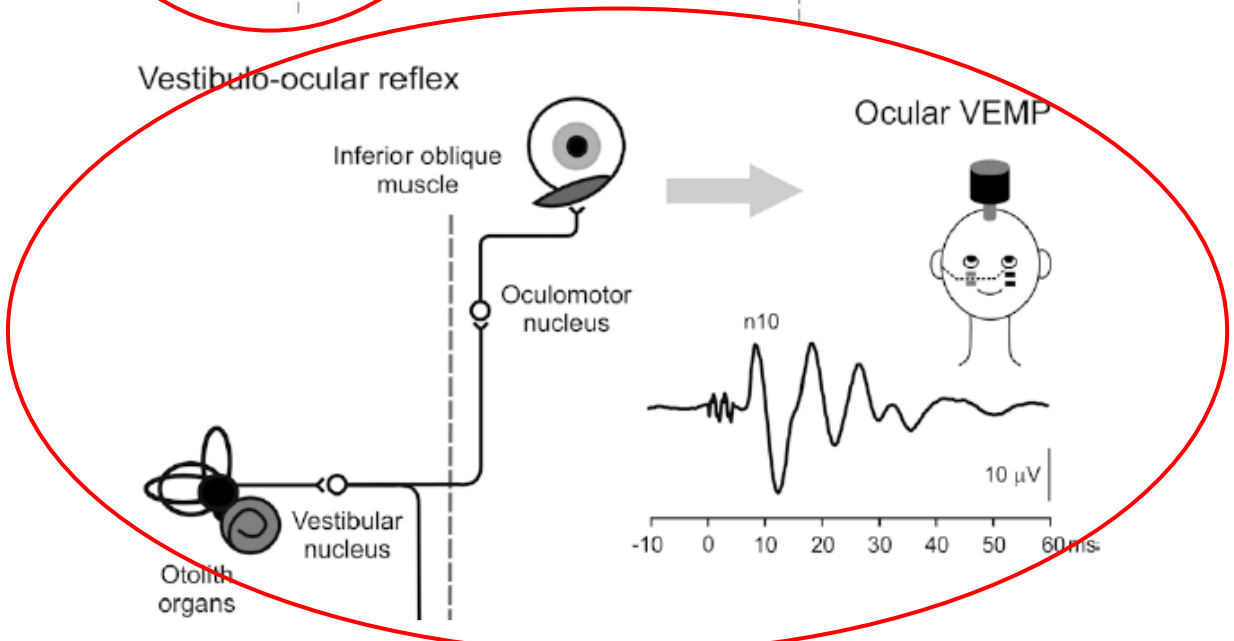


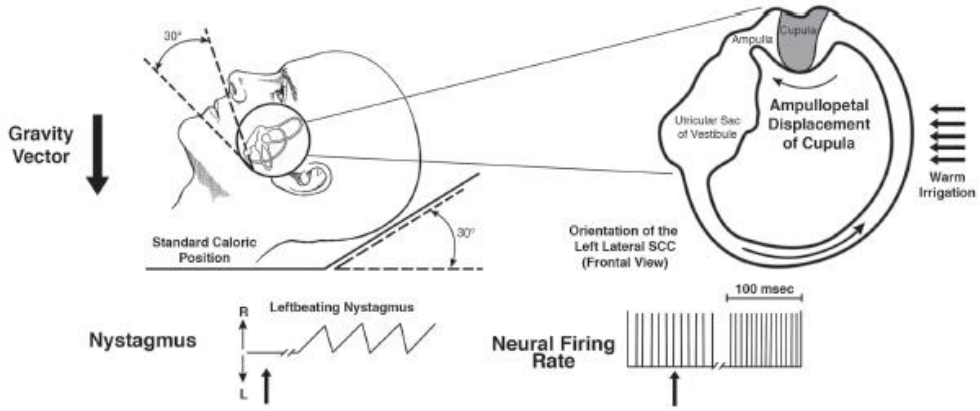
Vestibular rehabilitation Susan J. Herdman Richard A. Cledenial











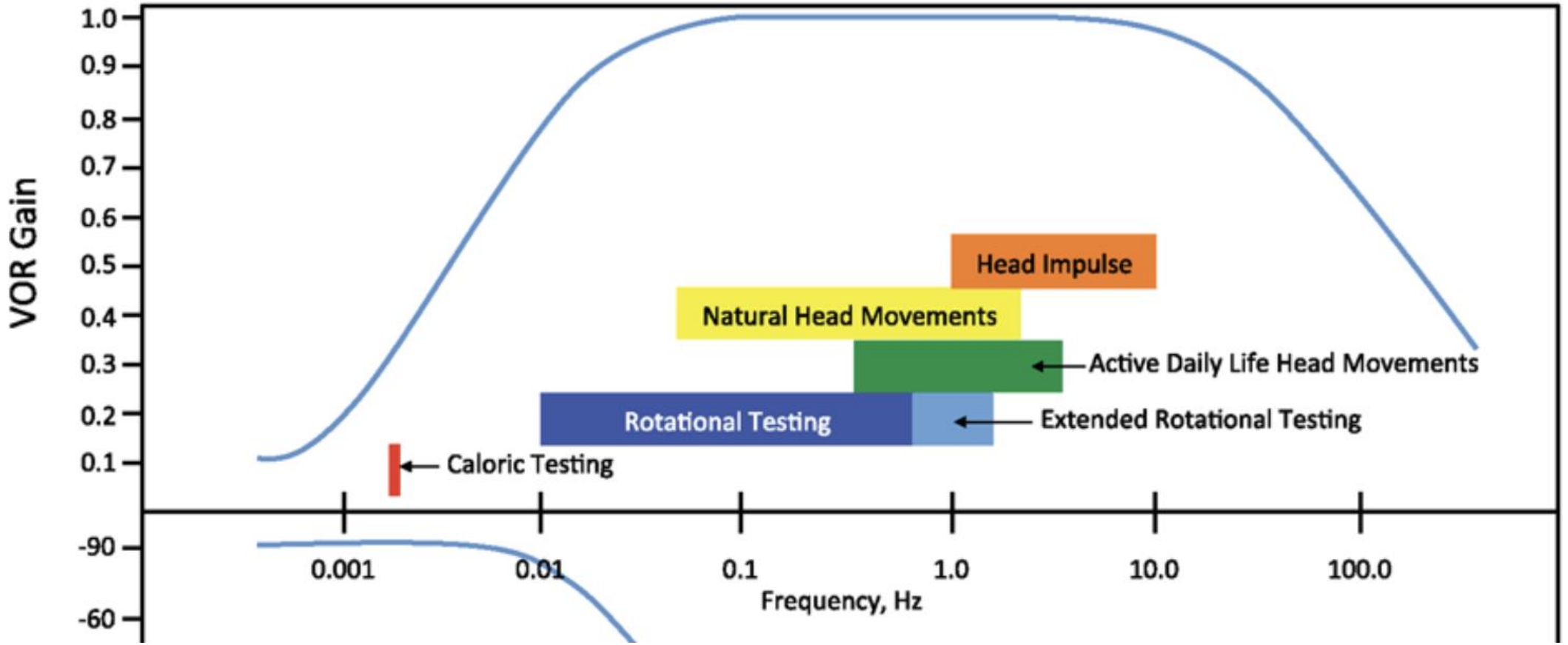
Vertigo and Dizziness

Michael Strupp • Thomas Brandt • Marianne Dieterich

Title: Electronystagmography/videonystagmography (ENG/VNG) / Devin L. McCaslin.

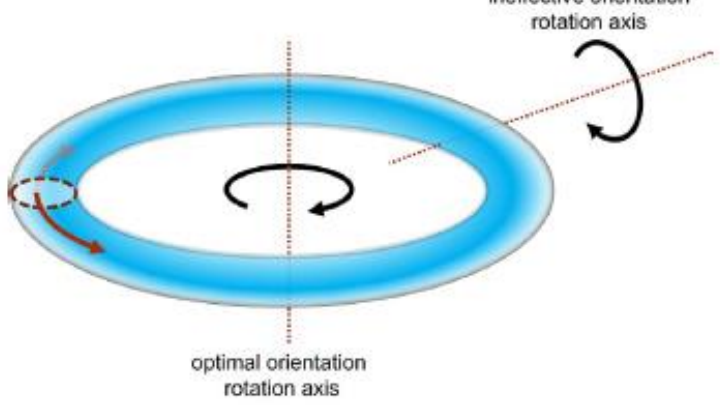
J Neurophysiol 92: 20–35, 2004.

Frequency spectrum of angular VOR

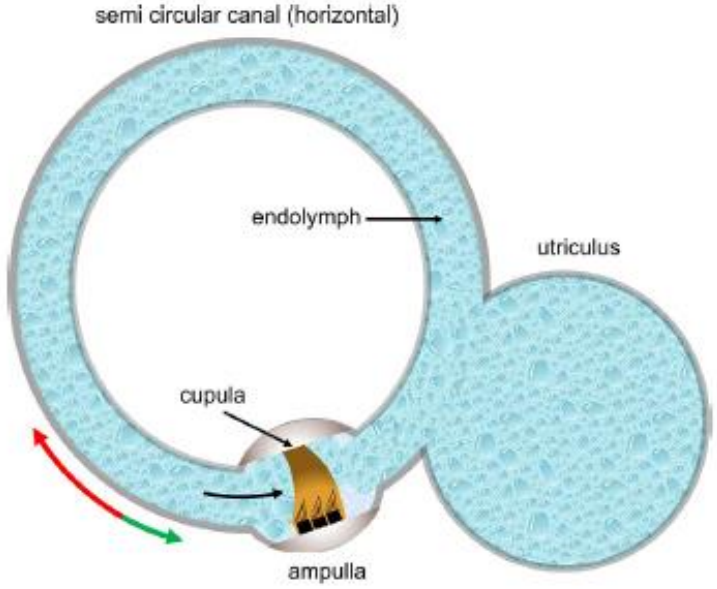


Ewald’s law

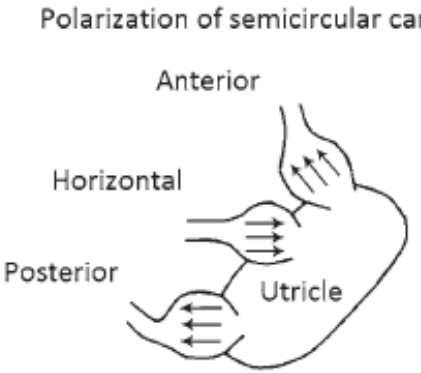
- First law
  - Maximal stimulation of eye movements always occur in the plane of the canal being stimulated
- Second / Third law
  - Vestibular excitation provokes a stronger neural response than inhibition
  - Ampullopetal flow in the horizontal canal causes stronger response than ampullofugal flow
  - Ampullofugal flow in the vertical canals causes stronger response then ampullopetal



Ewald’s 1st Law



Ewald’s 2nd Law: asymmetry



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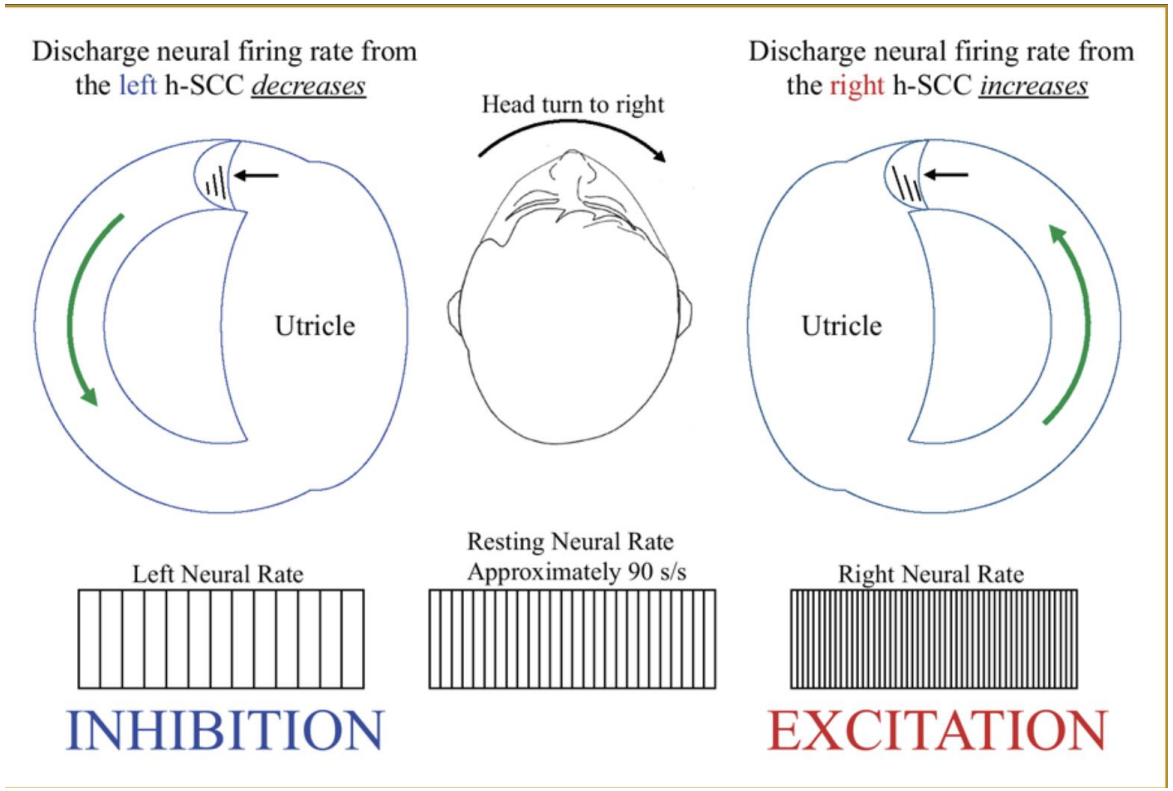
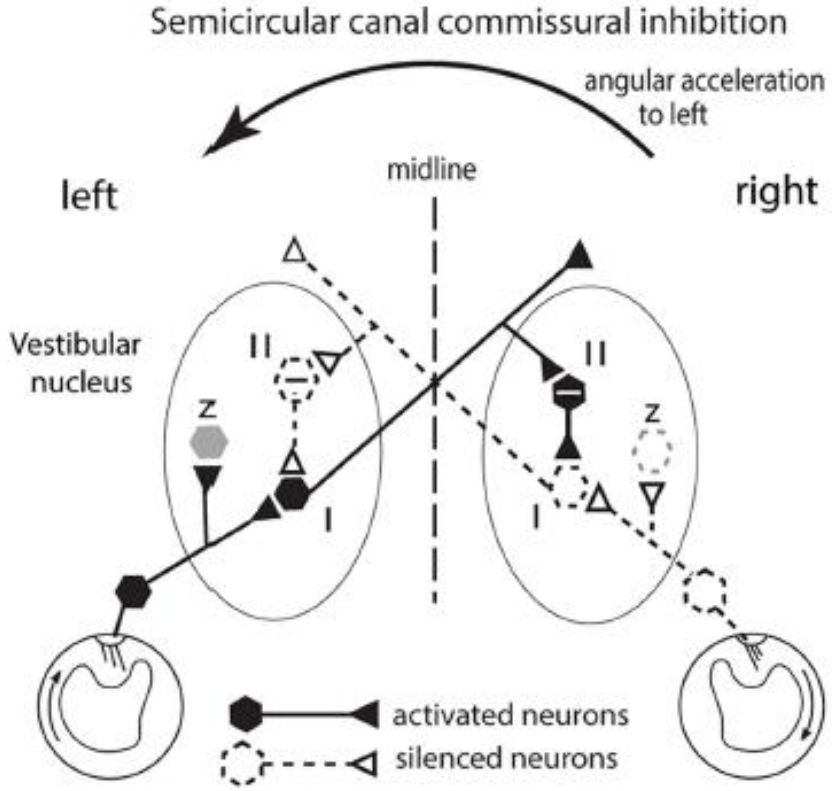
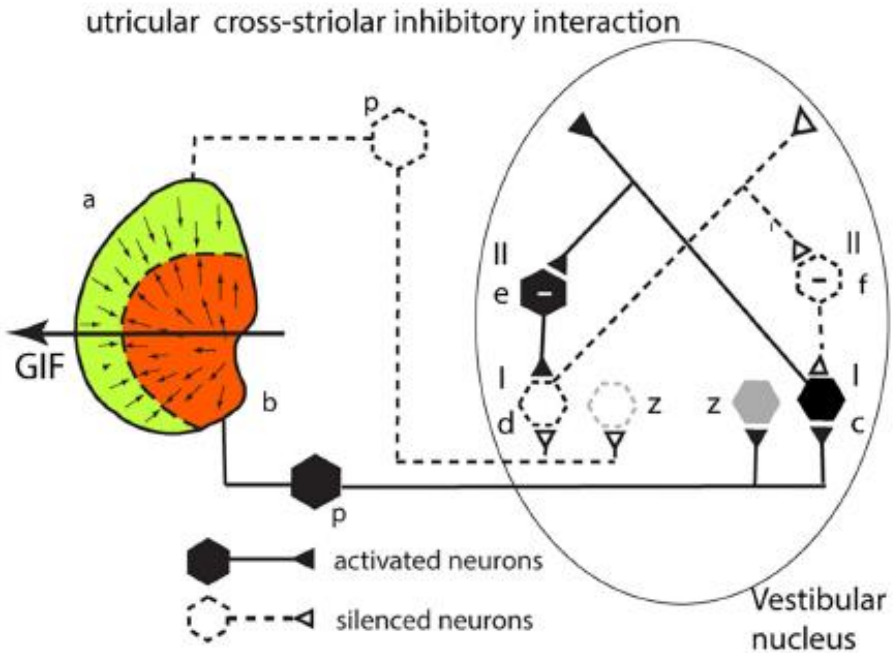
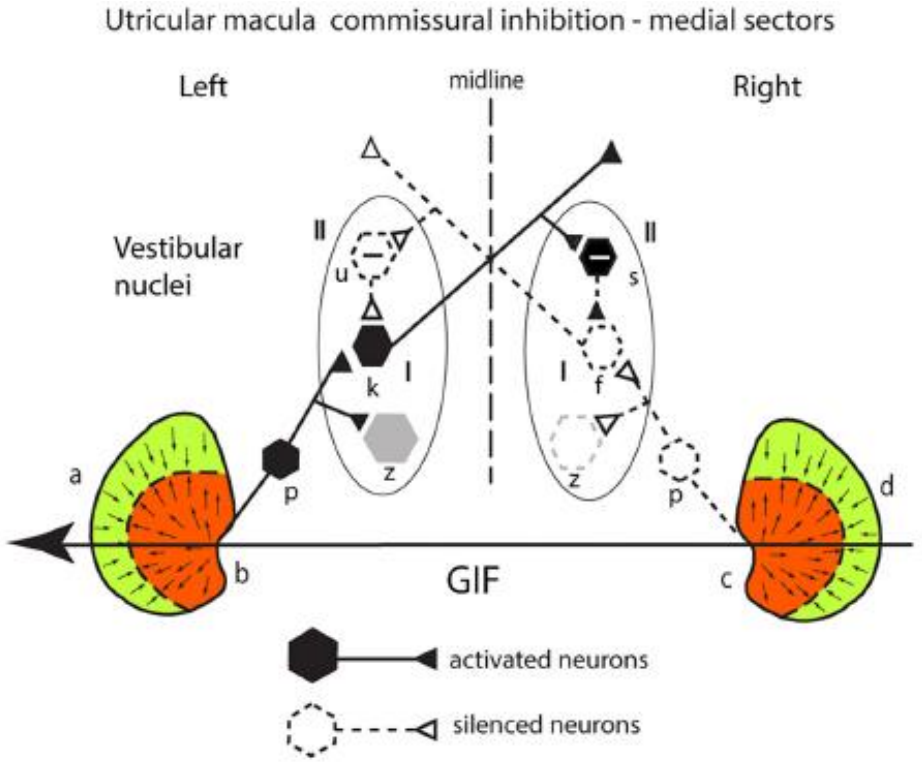
Functional pairs of canals “push – pull”





Utricular system – two inhibitory interactions





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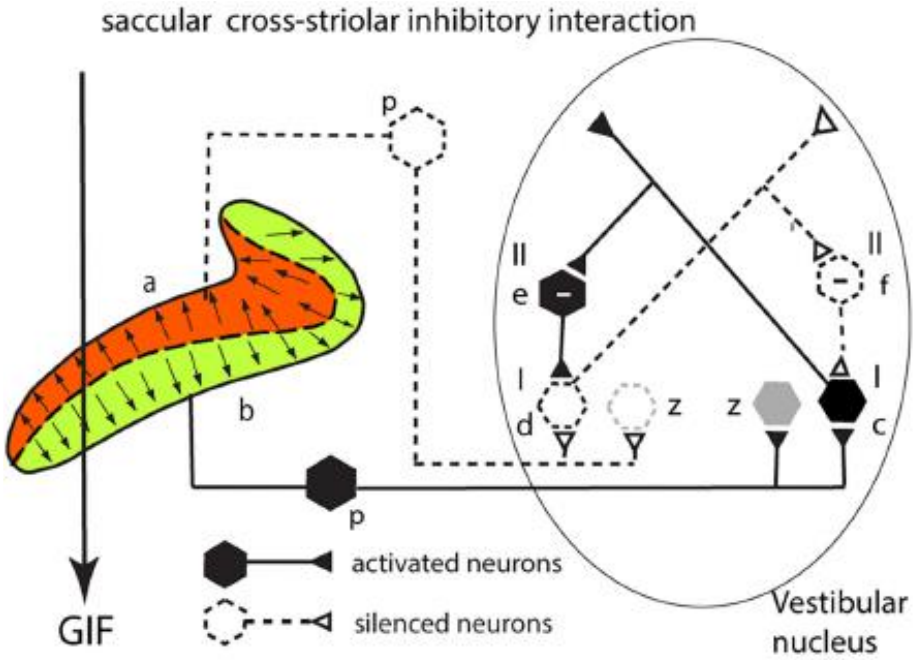
Saccular system – no commissural inhibition

- Losing unilateral saccular macula
  - no measurable postural or oculomotor change in Guinea pig
  - sudden sensorineural hearing loss without vertigo



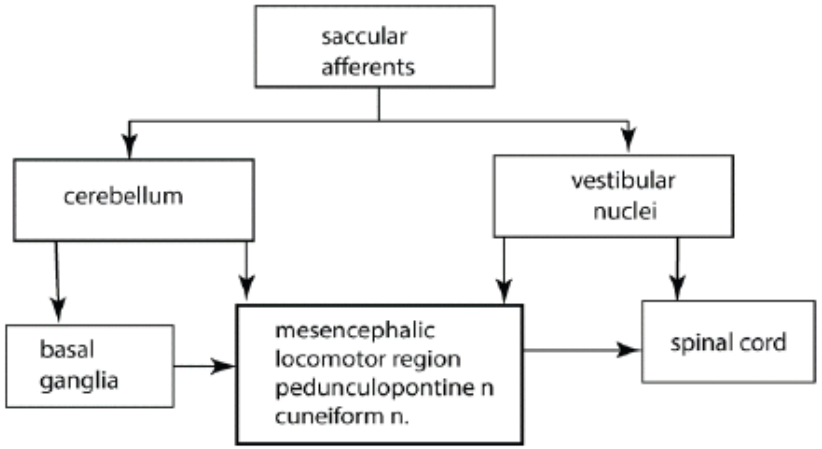


Table 1. Demographics of Patients and Clinical Data

	SSNHL (N = 152)	SSNHL_V (N = 73)	SSNHL_N (N = 79)
Sex			
Male/female	70 (46.1%)/ 82 (53.9%)	35 (47.9%)/ 38 (52.1%)	35 (44.3%)/ 44 (55.6%)
Age	53.6 ± 15.9	56.7 ± 14.4	50.7 ± 16.8
Site			
Right/left	78 (51.3%)/ 74 (48.7%),	39 (53.4%)/ 34 (46.6%)	39 (49.4%)/ 40 (50.6%)
Pure-tone average, dB	68.8 ± 27.1	78.1 ± 26.9	58.0 ± 20.5
Impaired vestibular organs			
ASCC	5 (3.3%)	4 (5.5%)	1 (1.3%)
LSCC	18 (11.8%)	15 (20.5%)	3 (3.8%)
PSCC	48 (31.6%)	41 (56.2%)	7 (8.9%)
Utricle	31 (20.4%)	26 (35.6%)	5 (6.3%)
Saccule	41 (27.0%)	25 (34.2%)	16 (20.3%)
Number of impaired organs	0.94 ± 0.8	1.52 ± 1.01	0.41 ± 0.38
Hearing recovery			
Complete	46 (30.3%)	16 (21.9%)	30 (38.0%)
Partial	69 (45.4%)	40 (54.8%)	29 (36.7%)
No	37 (24.3%)	17 (23.3%)	20 (25.3%)
Salvage ITS	114 (75.0%)	61 (83.6%)	53 (67.1%)

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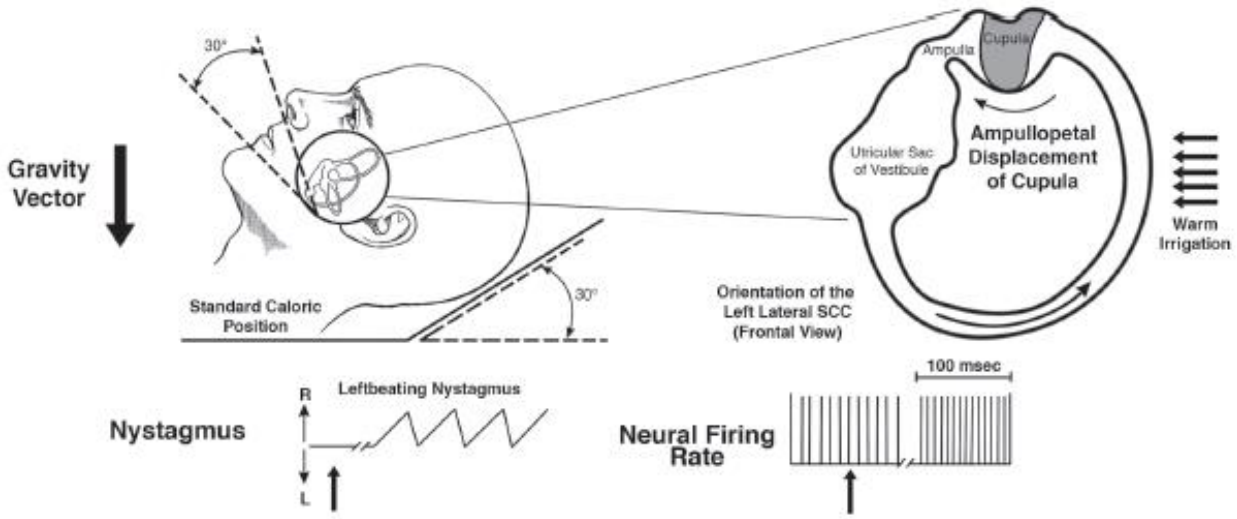
Calorics

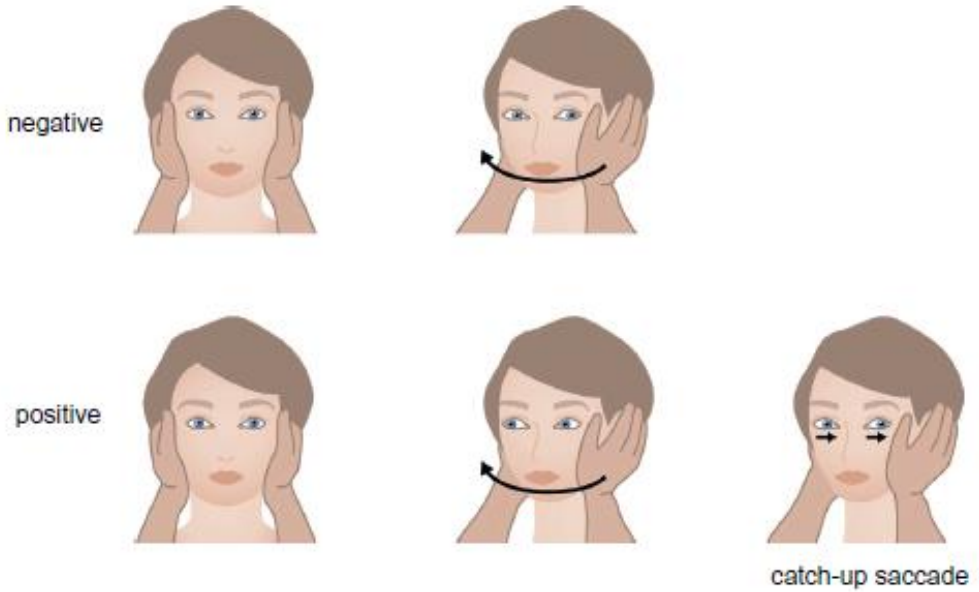


Measurement Parameters	Questions
Unilateral weakness	Are there interaural (left versus right ear) differences in slow-phase velocity nystagmus?
Directional preponderance	Is there a bias in the direction of the responses (e.g., is right-beating nystagmus always larger than left-beating nystagmus)?
Fixation index	Can the central nervous system appropriately exert control over the vestibular nuclei and reduce amplitude of the caloric nystagmus with visual fixation?
Hypofunction	Is the total of all four caloric responses abnormally low?
Hyperfunction	Is the total of all four caloric responses abnormally high?

Title: Electronystagmography/videonystagmography (ENG/VNG) / Devin L. McCaslin.

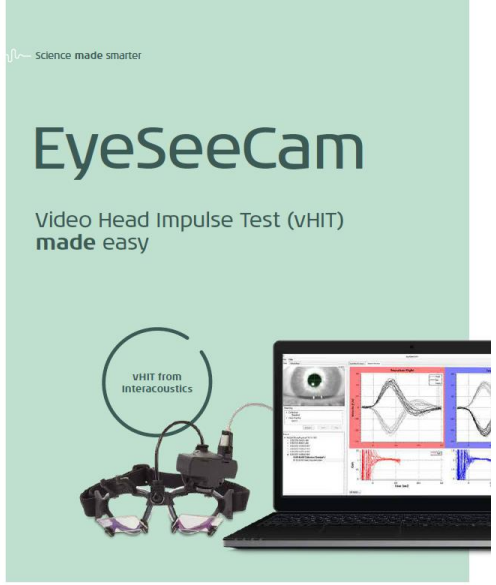
Clinical head impulse test (cHIT)

- First introduction of horizontal cHIT in 1988 by Halmagyi & Curthoys
- The patient fixates on a stationary target while the head is turned right or left
- Abrupt rotate the head 10-20 degrees to one side, the aVOR will produce equal and opposite eye movement
- Impaired aVOR can't produce equal eye movement – a catch-up saccade (overt saccade) is noted
- Vertical canal cHIT introduced in 1998

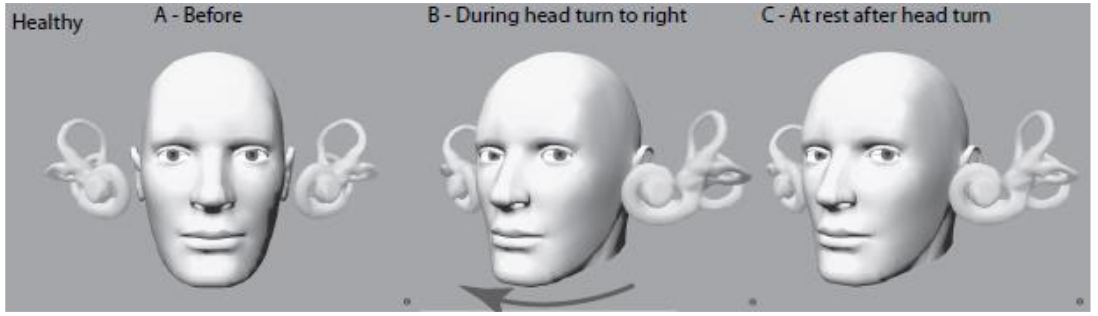


Video HIT (vHIT)

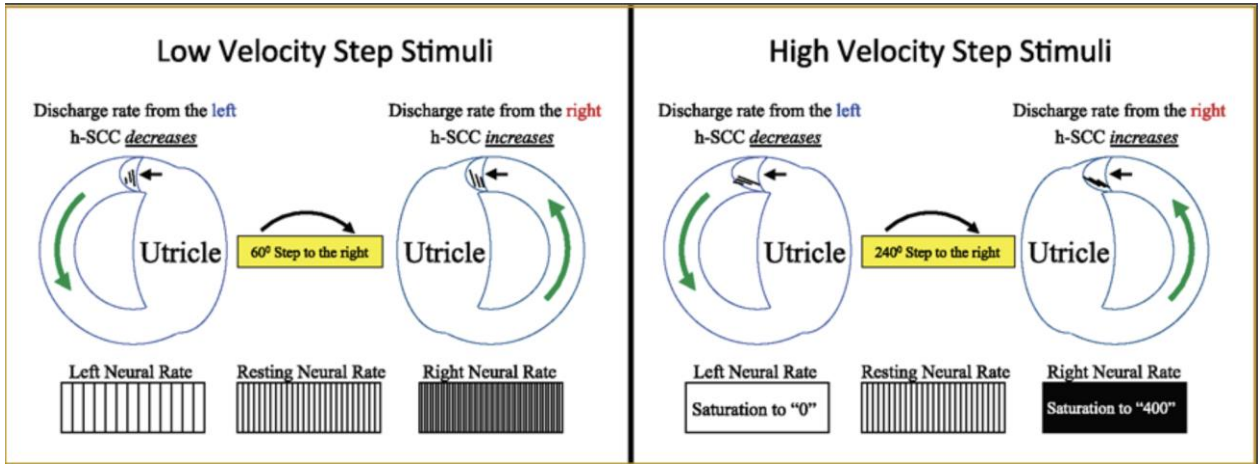
- Quantitative eye movement recording
  - scleral search coil : gold standard in academic research
  - high-frequency video oculography : commercialized for clinical use
- vHIT is now an important physiologic evaluation tool for aVOR

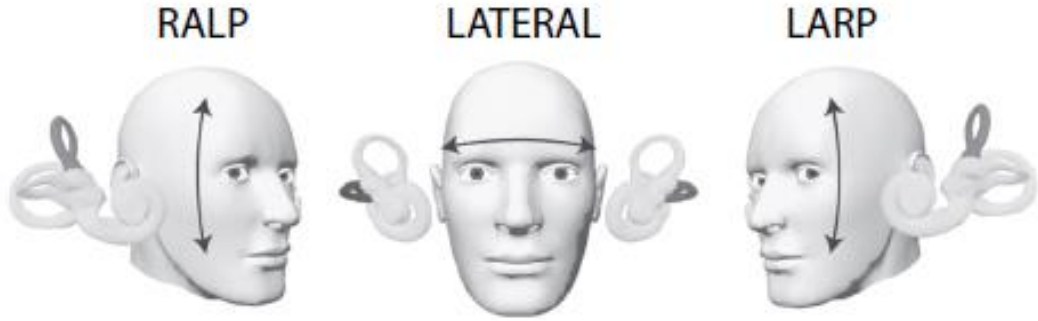


Video HIT (vHIT)



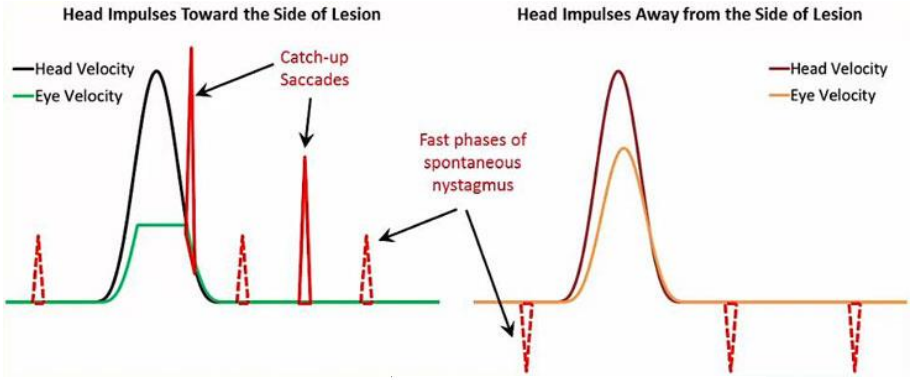
- A valid head impulse test stimulus
  - Passive , unpredictable
  - Abrupt start and stop
  - Displacement = 20° - 30°
  - Peak head velocity = 200°/s - 400°/s
  - Peak head acceleration = 2000°/s²- 4000°/s²
- Patient cooperation
  - Gaze remained on the earth-fixed target
  - Relax neck muscle
- Covert saccade
  - Catch up saccade occurring during the head movement , undetectable in cHIT
  - Latency usually < 200ms

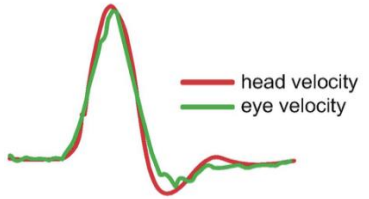




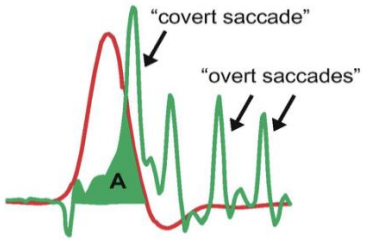
vHIT analysis

- Gain
  - Ratio of the eye movement velocity to the head movement velocity
    - Calculation method: velocity/position/regression
    - Relatively stable in ageing process
  - Norms :
    - 0.8-1.2 in lateral canal, 0.7-1.2 in vertical canals
    - Value > 1.2 : slippage of goggles, patient too close to the fixation dot, anticipated, or possible Meniere's disease
  - Asymmetry (lateral , LARP or RALP)
    - %=(1-lower gain/higher gain) x100
    - Gain asymmetry ratio (gain difference of horizontal HIT) : 0-13.3%
- Saccade
  - A fast eye movement (100°/s to 700°/s during excursion of 0.5 to 50 degrees) to shift our gaze to interesting objects and explore our visual environments
  - The quick phases of nystagmus during vestibular and optokinetic stimulation
  - The fastest eye movement in velocity but not the quickest in response (average 80-250 ms and last less than 100 ms)

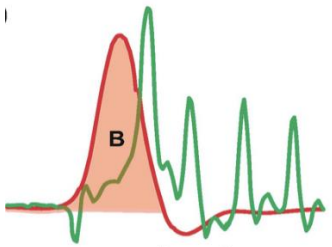




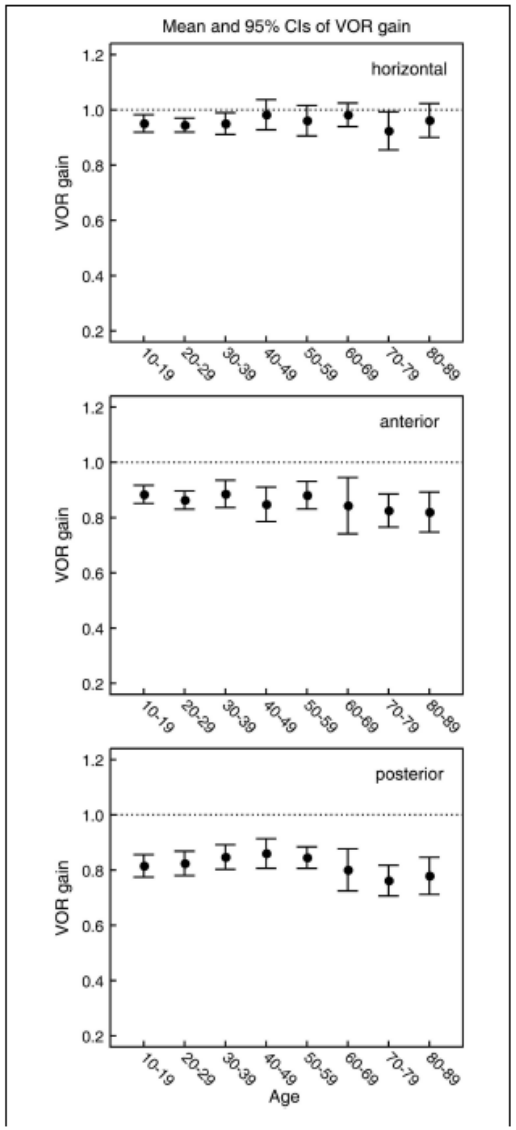
normal vHIT



abnormal vHIT



$$\text{Gain} = \frac{\text{Area A}}{\text{Area B}}$$



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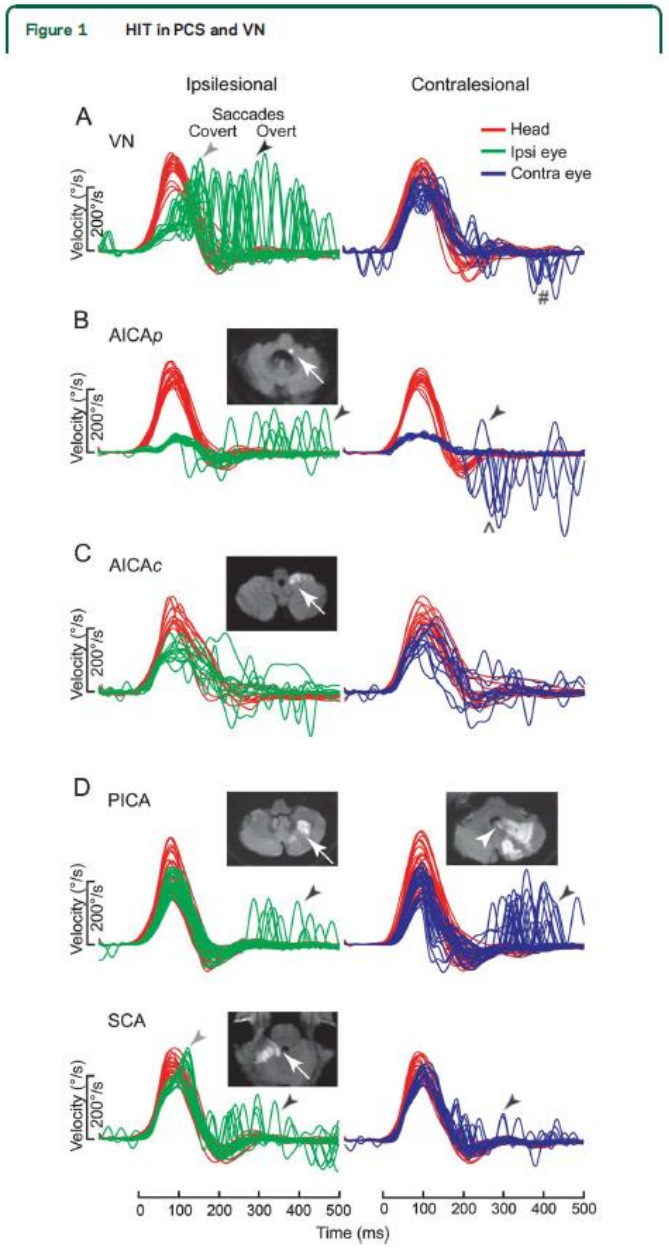

Gain

Normal or central type as in posterior circulation stroke (PCS)

Abnormal or peripheral type as in vestibular neuritis (VN)

Table 1. Differential diagnosis using vHIT in acute vestibular syndrome

Disease	vHIT saccade	vHIT gain	Accompanied symptoms and signs
VN	Large overt and covert saccades in lesion side/ small overt saccades in contralateral side	Markedly decreased in lesion side and mild decrease in contralateral side	Nausea, vomiting
AICA infarction	Overt saccades in both sides (less asymmetry in saccade amplitude than VN)	Bilateral gain reduction (lesser asymmetry than VN)	Hearing loss, facial palsy, sensory deficit, dysphagia/perverted nystagmus
PICA/SCA infarction	Small overt saccades in both sides (saccades are more frequent and/or larger in contralateral side than VN)	Symmetric [normal or mild (up to 20%) reduction]	Truncal ataxia, headache, facial pain/perverted nystagmus

vHIT results of lateral semicircular gain. vHIT: video head impulse test, AICA: anterior inferior cerebellar artery, PICA: posterior inferior cerebellar artery, SCA: superior cerebellar artery, VN: vestibular neuritis

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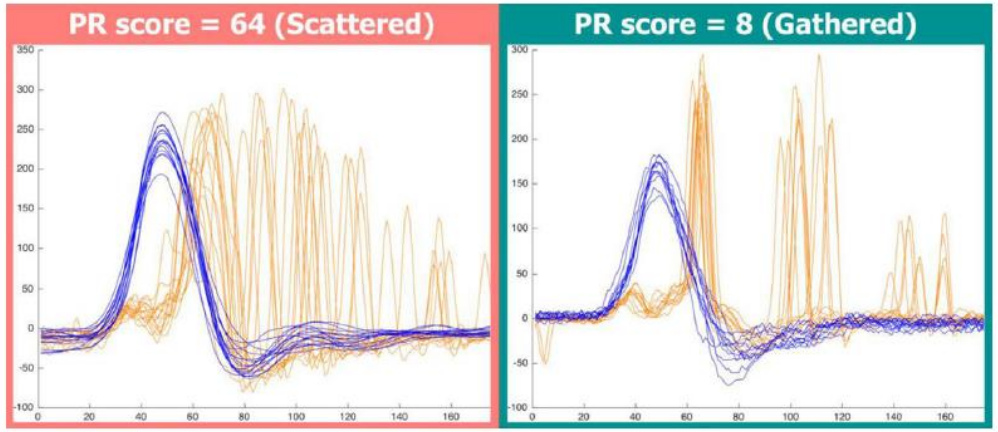
Neurology 83 October 21, 2014

PR score is a quantitative variable that ranges between 0 and 100; when PR is close to 0, saccadic responses are described as gathered, and when it is nearing 100, they are described as scattered.

Saccade

Gathered vs scattered

Related to central compensation



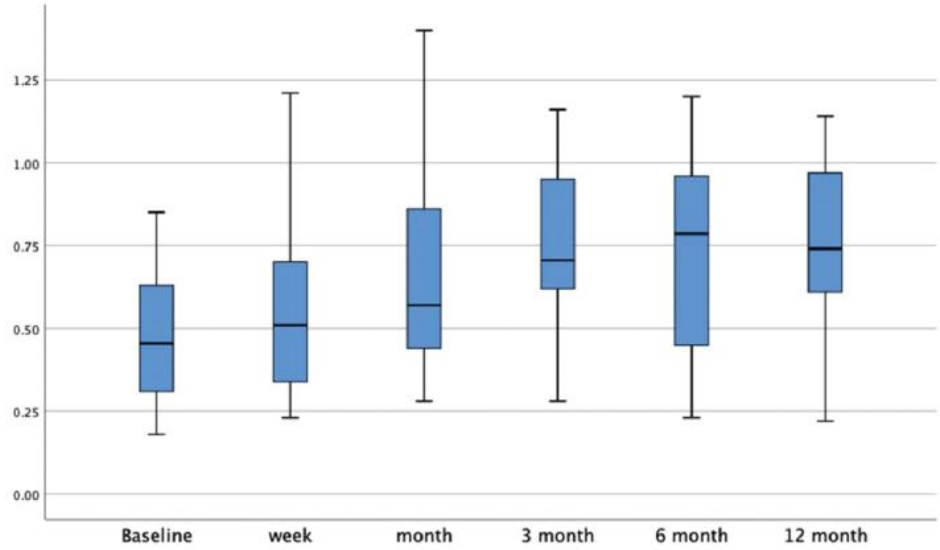
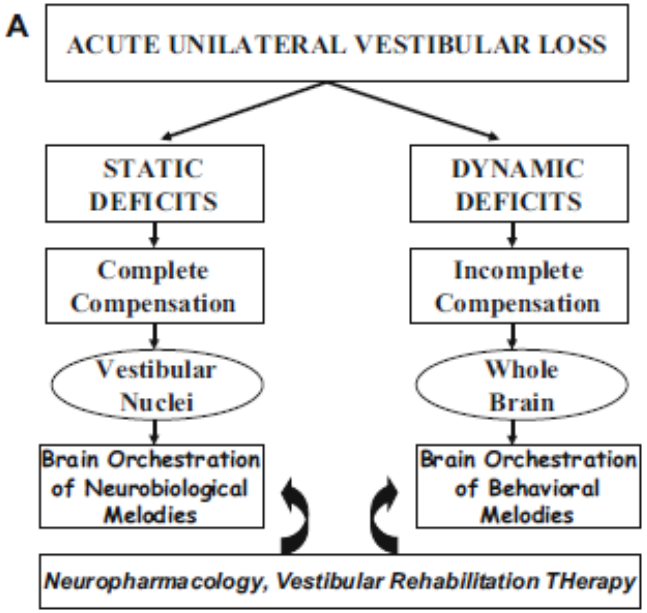
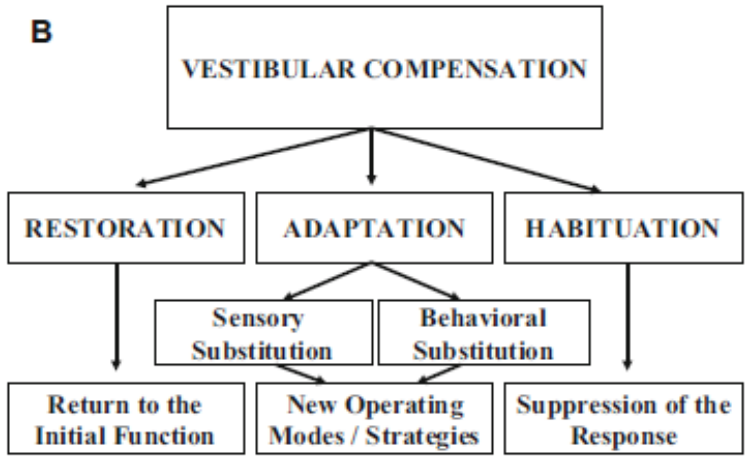


Figure 1. Bar graphic showing the HC gain values at every follow up.





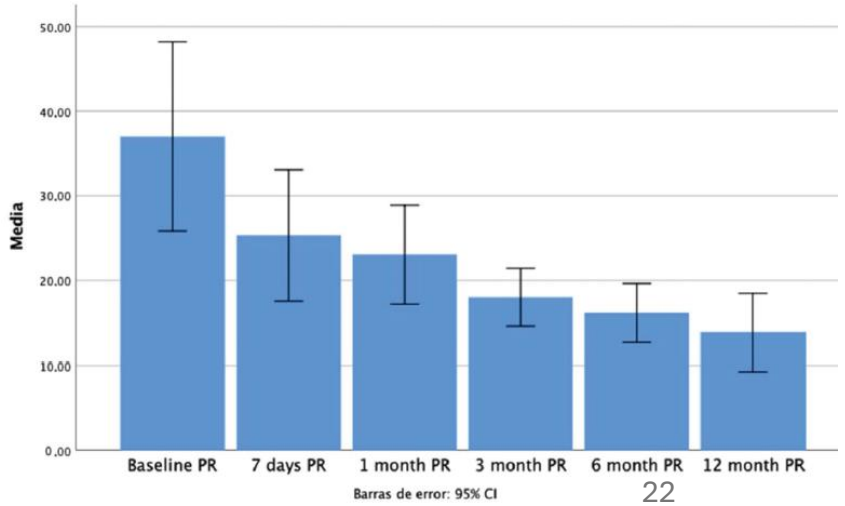
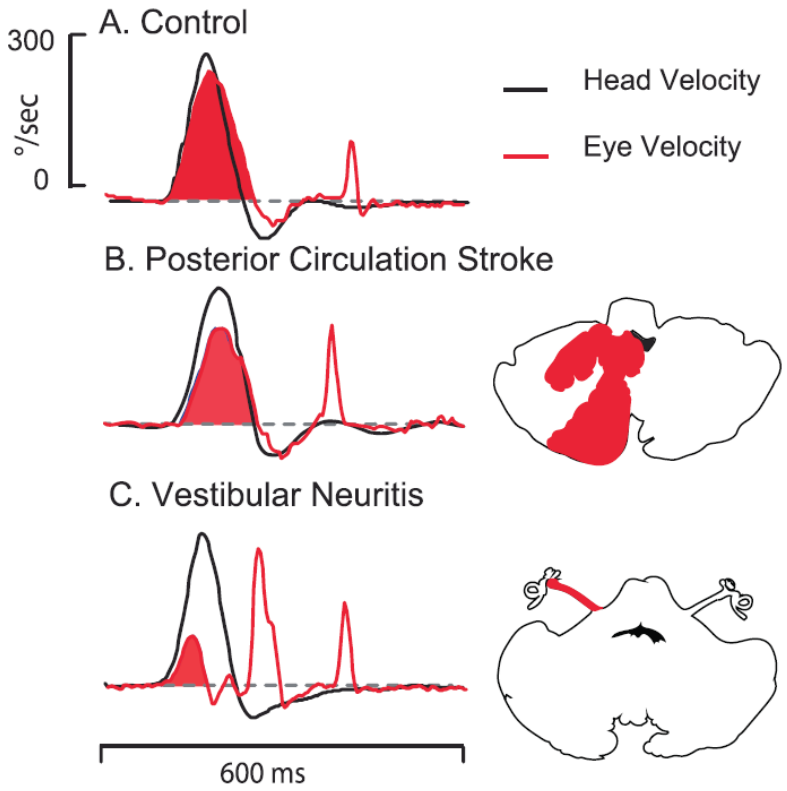


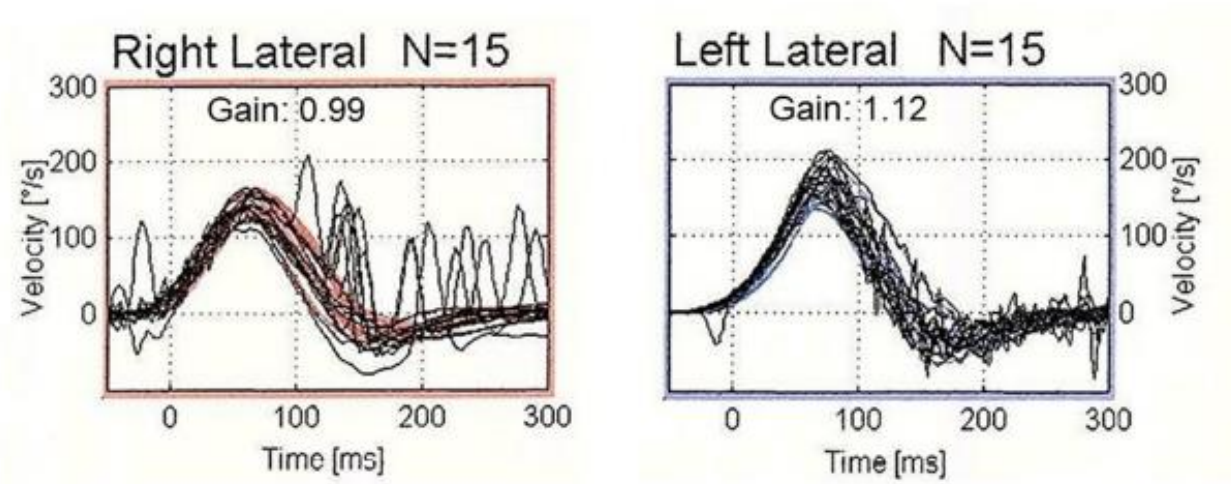
Figure 2. Bar chart for PR values at each follow up.

Timing of test



Presence of corrective saccades in patients with normal vestibulo-ocular reflex gain in video head impulse test

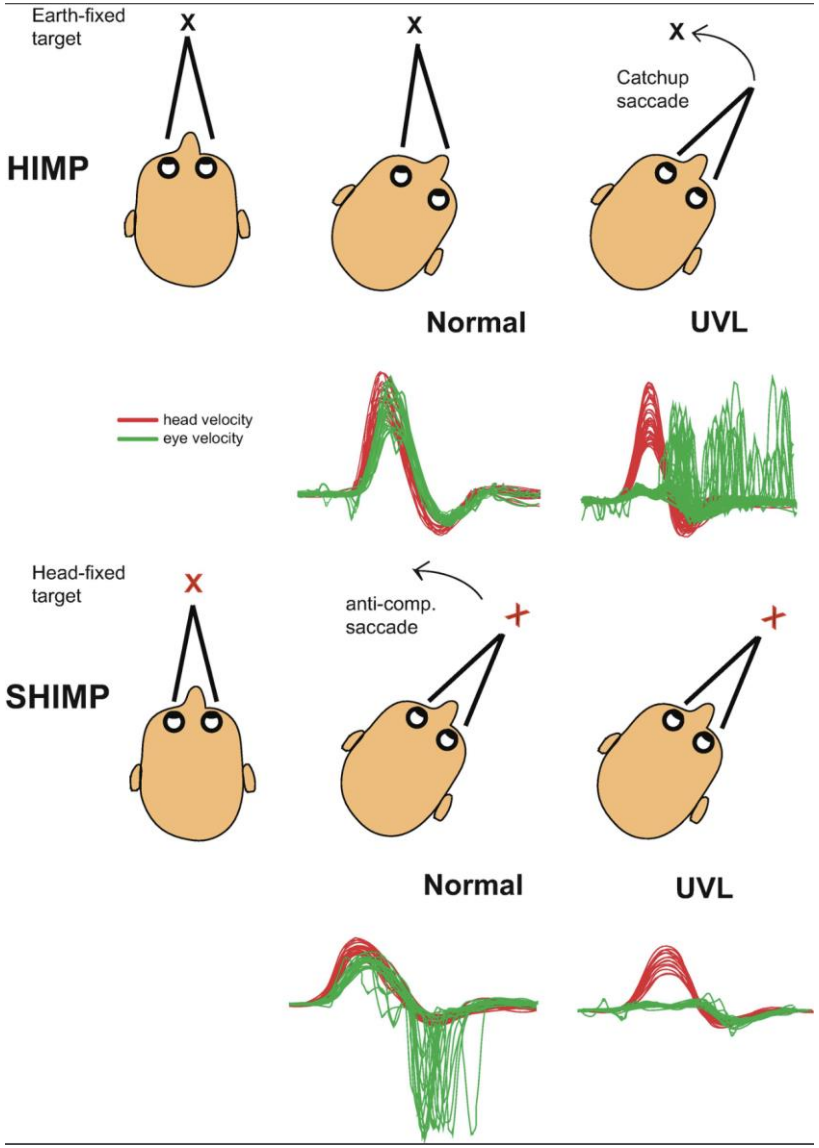
Kayoko Kabaya*, Akina Fukushima, Sachiyo Katsumi, Toshiya Minakata and Shinichi Iwasaki



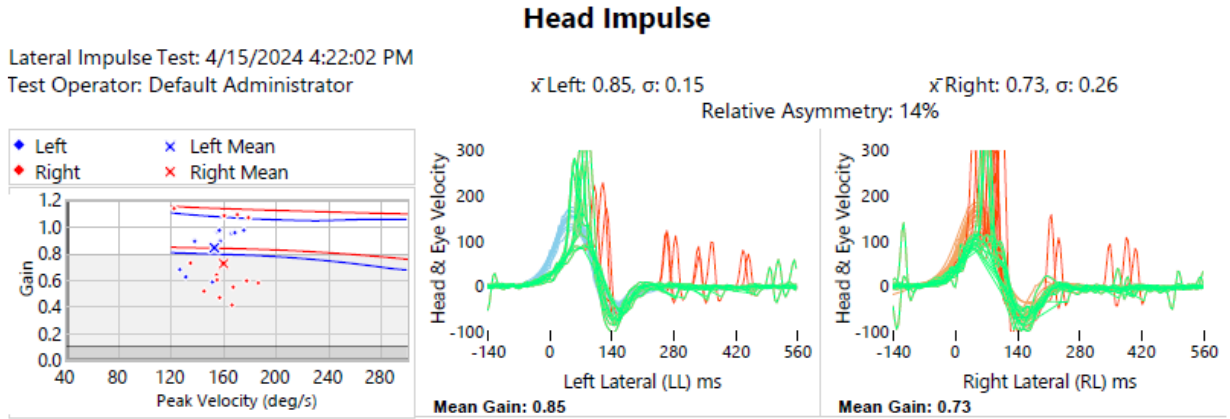
Clinical Neurophysiology 131 (2020) 2047–2055

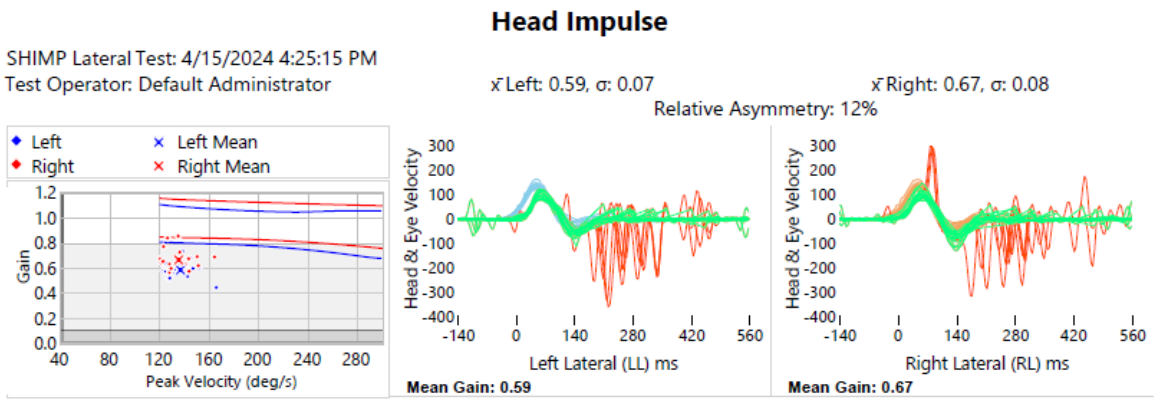
10.3389/fneur.2023.1152052

HIMP (head impulse) vs SHIMP (suppression head impulse)

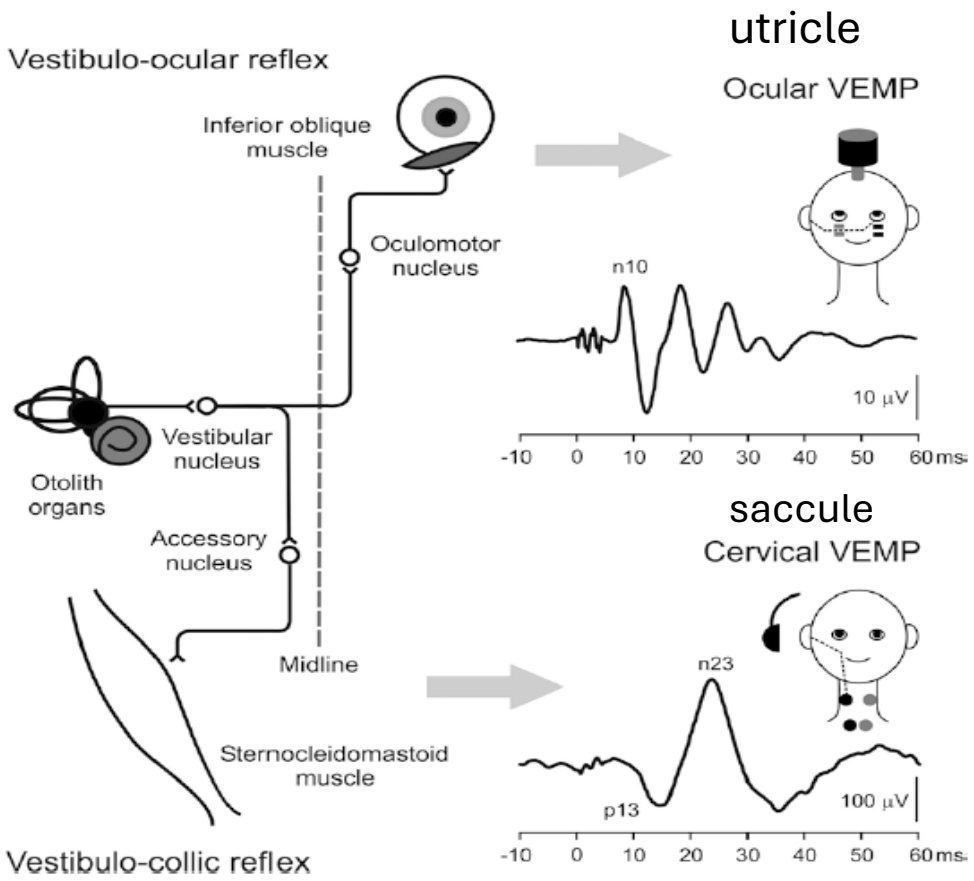


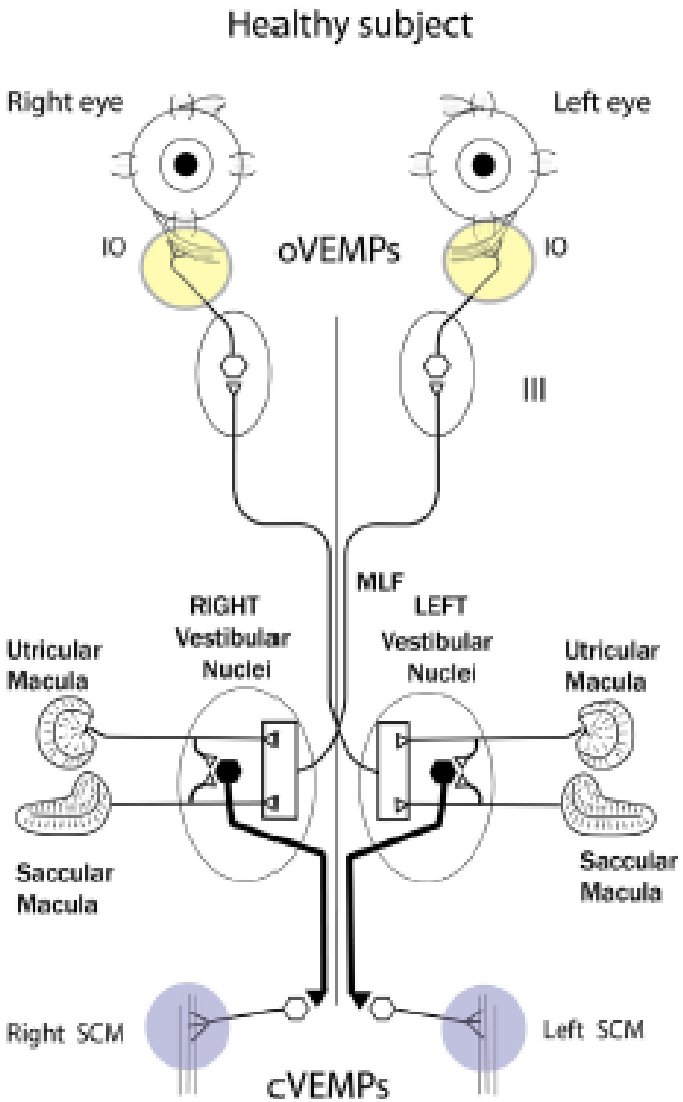
- The size of anti-compensatory saccades correlate with VOR gain
- An absent VOR will result in absent saccades
- SHIMP helps with gain calculation with HIMP paradigm can be difficult when covert saccades are present
- Some kids like SHIMP

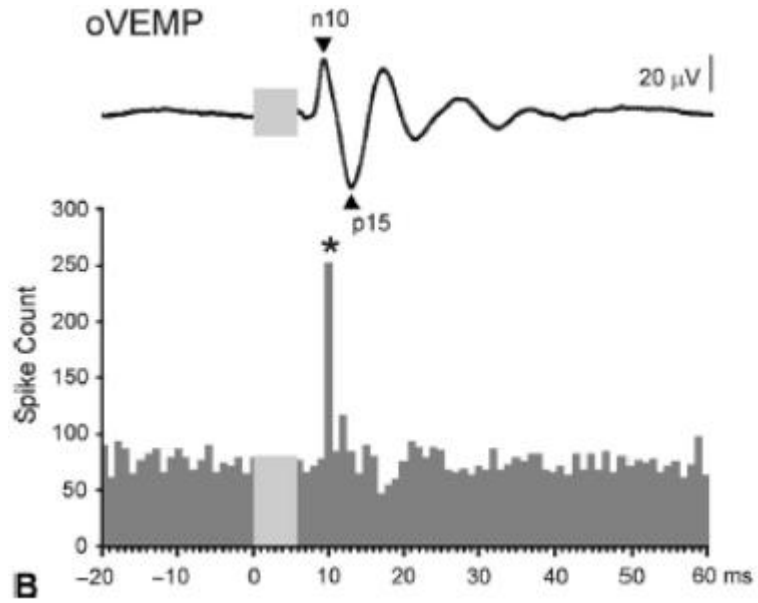


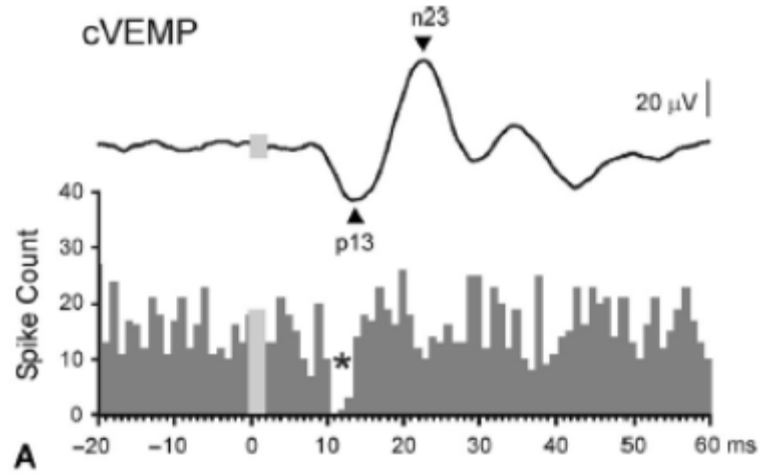


oVEMP(VOR) & cVEMP(VCR)





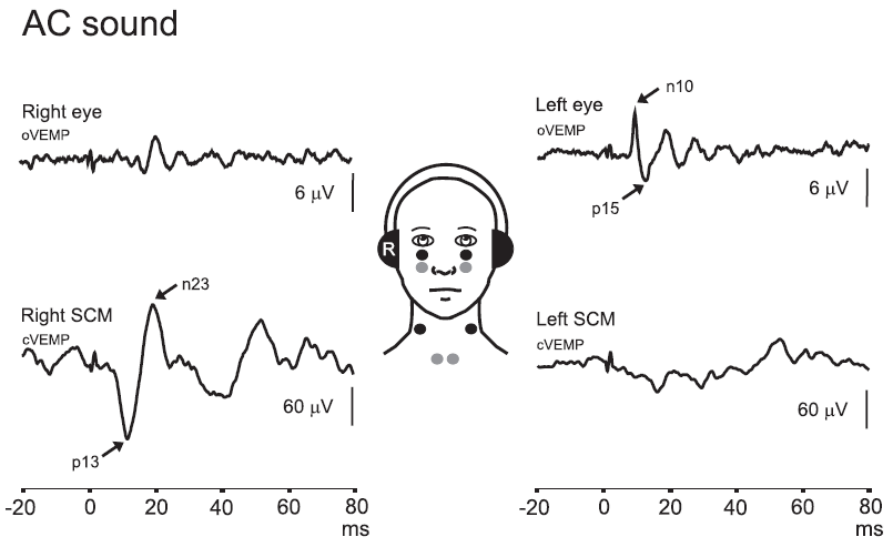


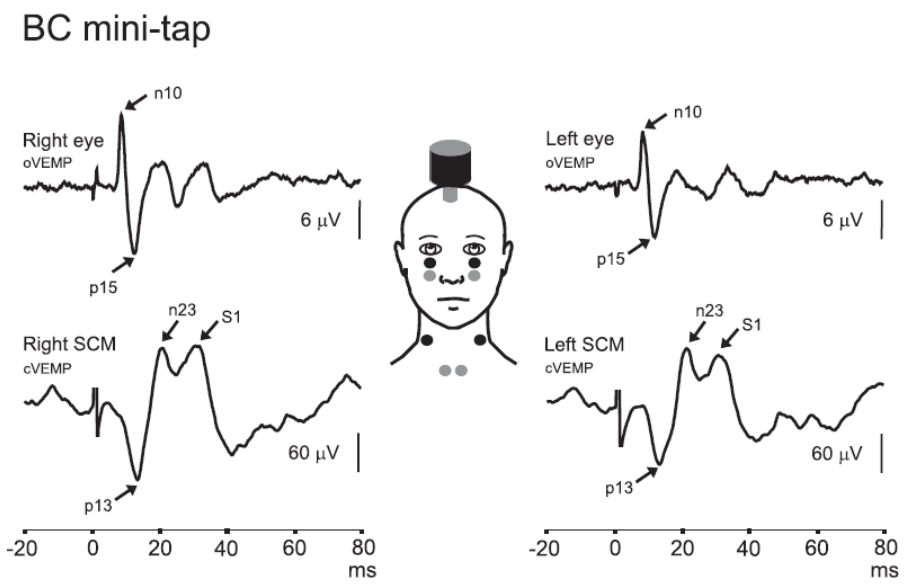


published: 30 March 2017
doi: 10.3389/fneur.2017.00117

VEMPs

- cVEMP first described by Colebatch in 1994, oVEMP first described by Rosengren in 2005
- Lack of consistency in recording procedures
- The rate of absent AC cVEMP is typically about 5-15% in subjects older than 60 years, the rate can be higher in AC oVEMP --Caution in interpreting with abnormal AC VEMPs
- Spontaneous horizontal nystagmus may impact oVEMP recording
- Asymmetries of amplitudes are readily interpretable, except in third mobile window disorders and early Meniere's disease - Have your own norms in peak latencies, interaural asymmetric ratio (IAR) and lower limit of amplitude at the 5th percentile





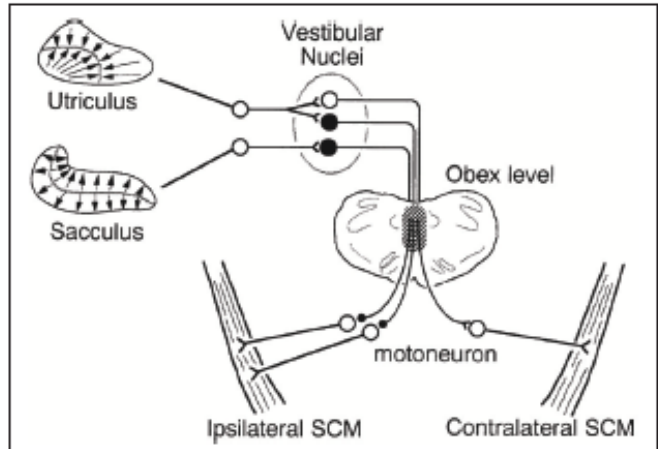
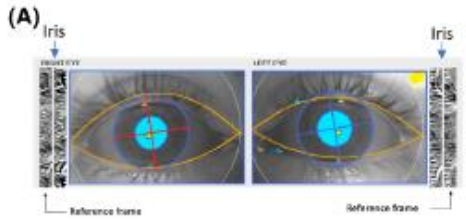
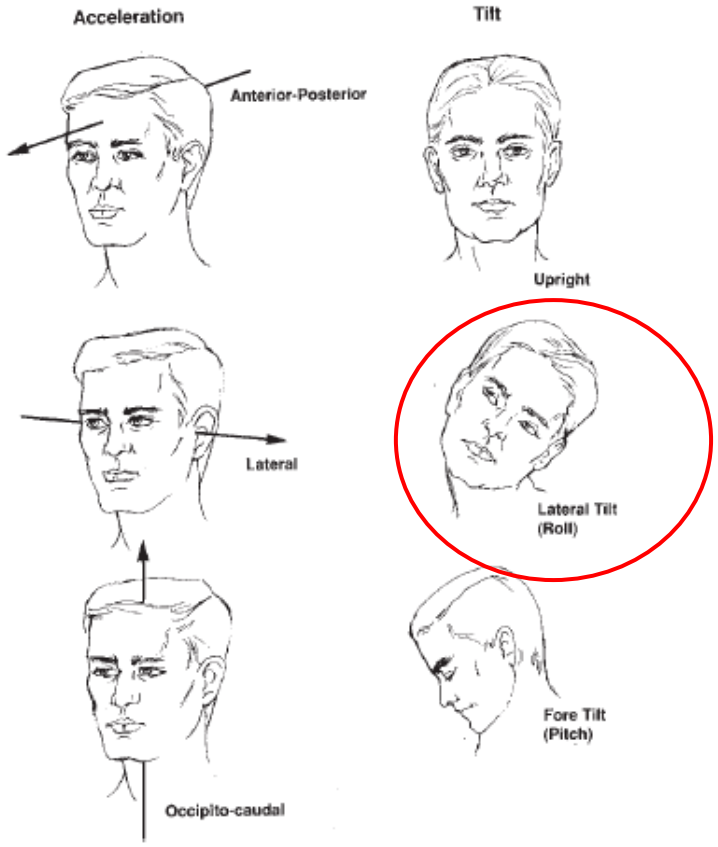


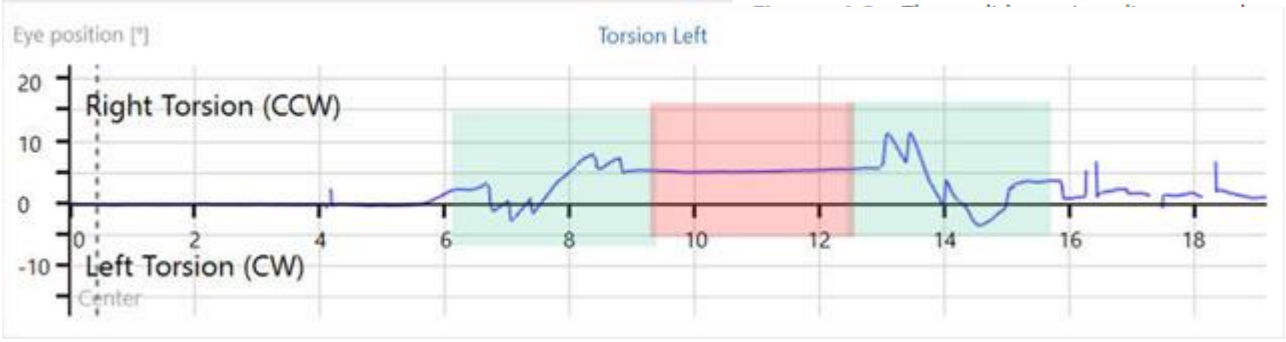
FIGURE 3 | Schematic diagram of saccular and utricular projections to the SCM muscles in cats. Kushiro et al. (35) determined the projections to the SCM muscles from the otoliths for the first time. The saccule has an inhibitory projection to the ipsilateral SCM, while the utricle has a similar ipsilateral projection and an additional excitatory projection to the contralateral side. Filled neurons are inhibitory, and the open ones are excitatory. The hatched portion indicates the main pathway of the otolith-SCM connection. Reprinted from Kushiro et al. (35).

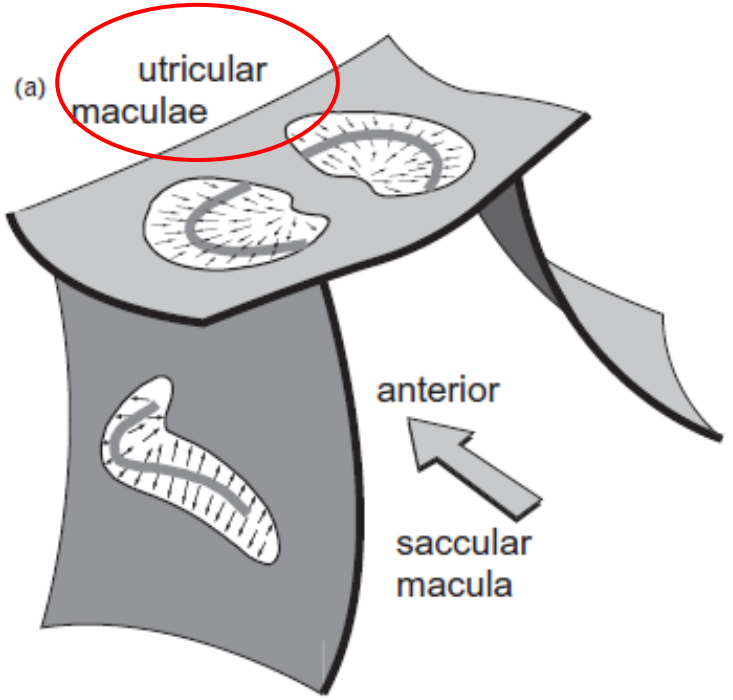
Clinical Neurophysiology Practice 4 (2019) 47–68
published: 29 June 2018
doi: 10.3389/fneur.2018.00481

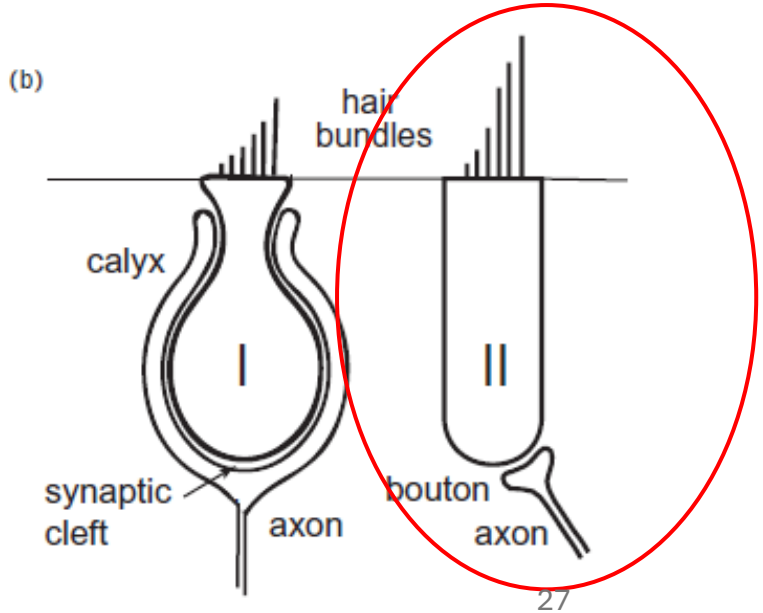
OCR (VOR)





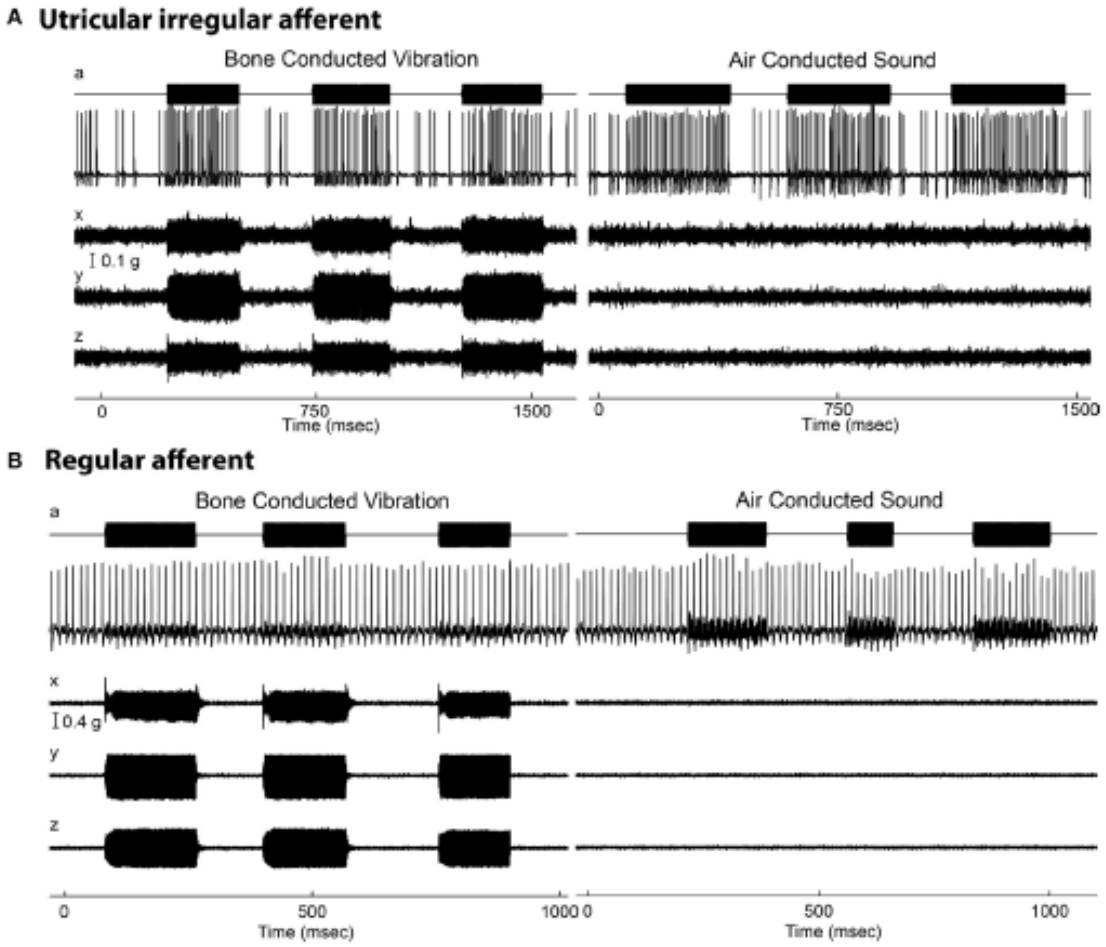




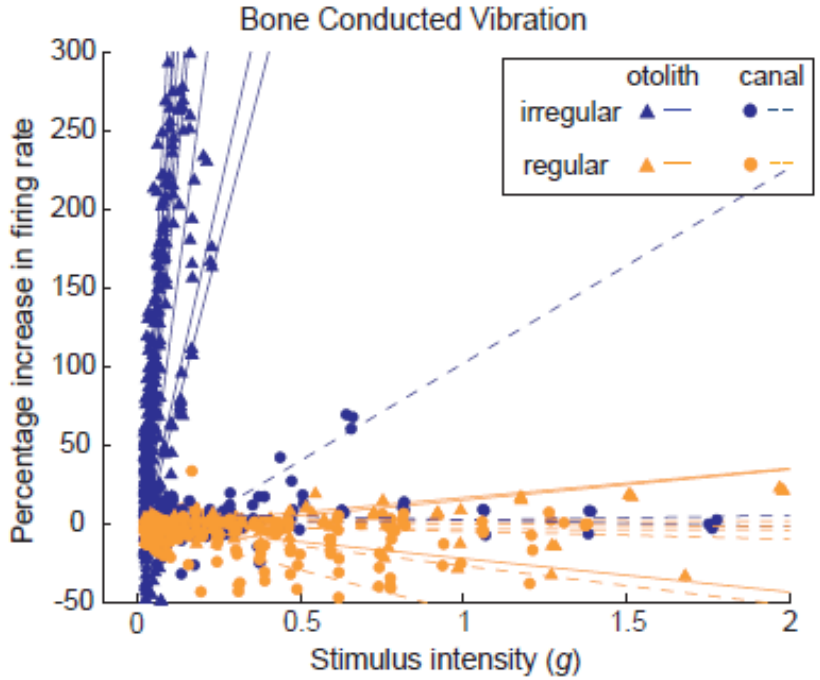


Handbook of Clinical Neurology, Vol. 137 (3rd series)
Neuro-Otology

Irregular/ Regular vestibular afferents

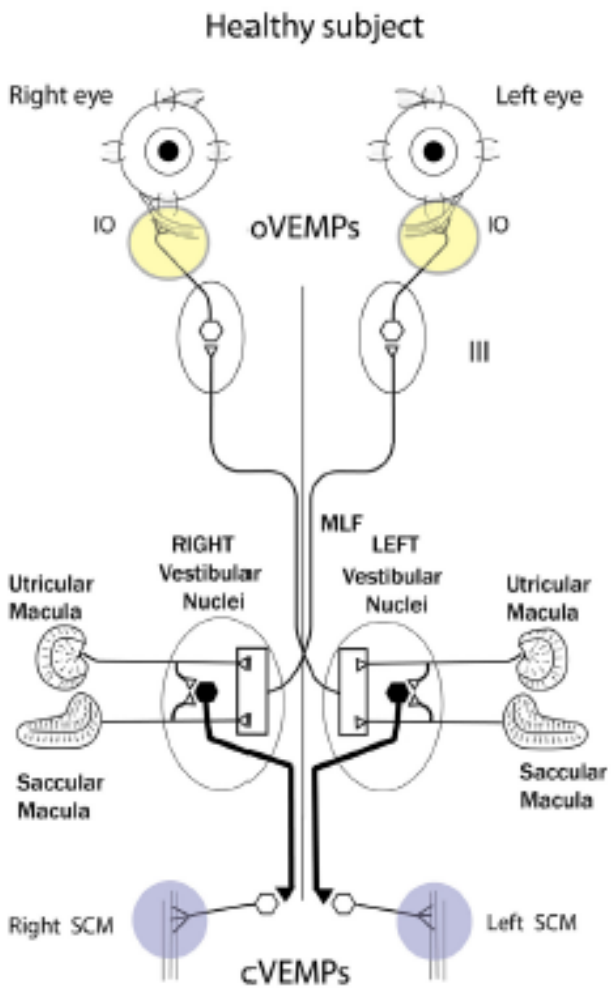


- Irregular afferents synapse on type-1 receptors at the striolar zone and central crista
- Transient (kinetic) fast conducting otolithic afferents
- Highly sensitive to linear and angular acceleration, galvanic stimulation and ototoxicity of gentamicin
- Can be activated by sound and vibration
- Less neurons than regular afferent neurons



published: 30 March 2017
doi: 10.3389/fneur.2017.00117

VEMPs Transient (kinetic) vs OCR Sustained (tonic)



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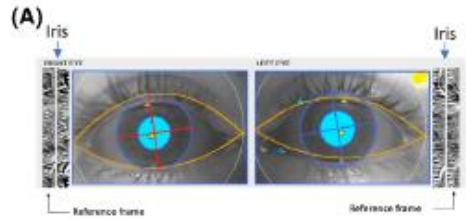
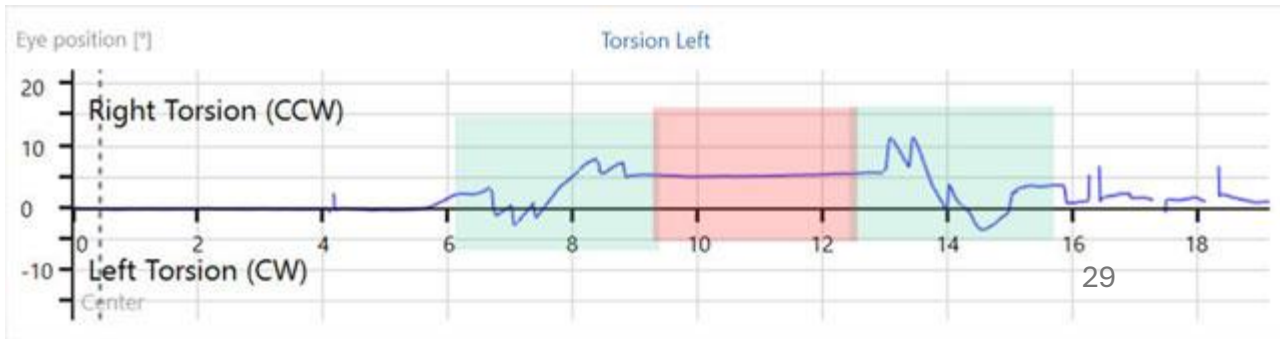


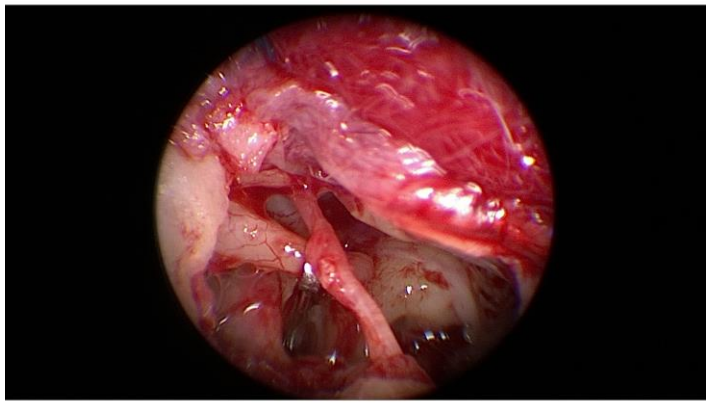


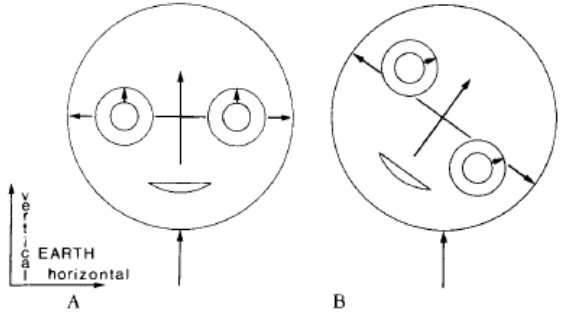
Ocular Tilt Reaction with Peripheral Vestibular Lesion

G. Michael Halmagyi, FRACP,
Michael A. Gresty, PhD,
and William P. R. Gibson, FRCS

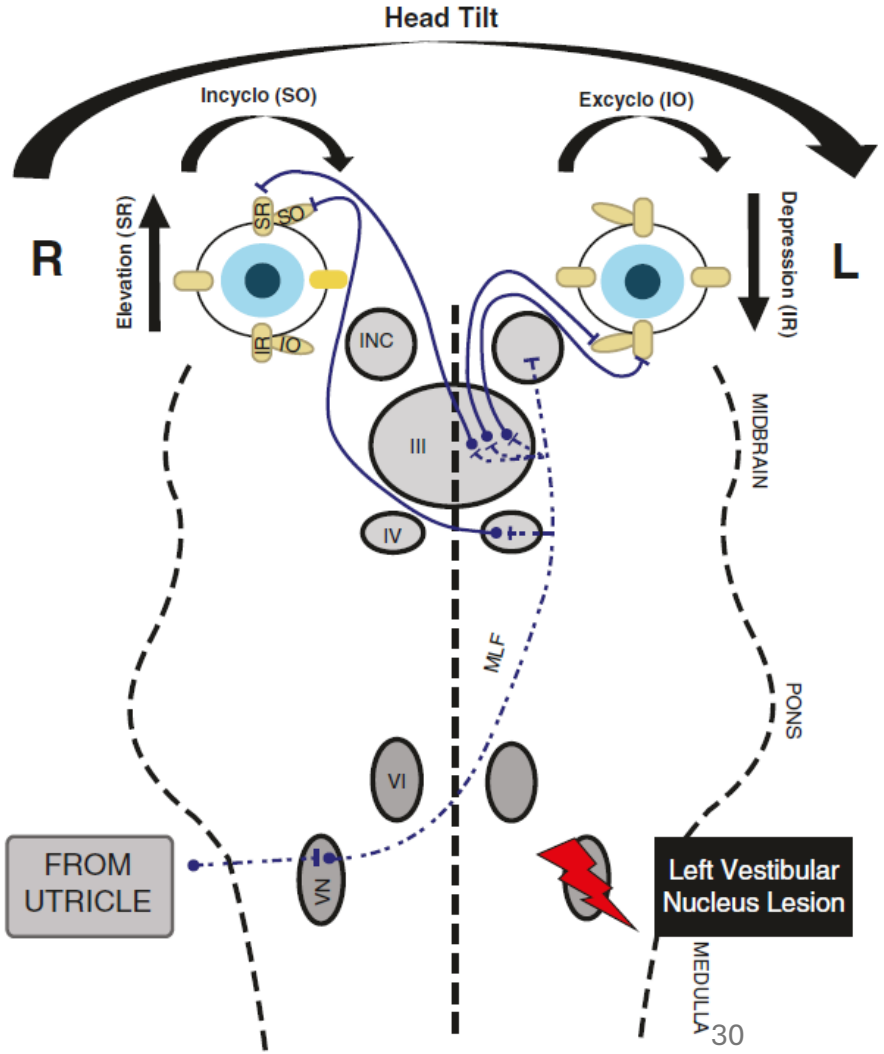
Following inadvertent destruction of the left vestibular labyrinth during stapedectomy, a patient developed a transient abnormality of posture consisting of leftward ocular counterrolling, leftward head tilting, and a right-over-left skew deviation. This postural pattern, known as the "ocular tilt reaction," is the normal compensatory response of the dependent utricle to tilting. In this patient, the unopposed action of the intact right utricle was presumably responsible for the appearance of a normal leftward ocular tilt reaction.

Halmagyi GM, Gresty MA, Gibson WPR: Ocular tilt reaction with peripheral vestibular lesion.
Ann Neurol 6:80–83, 1979





pathologic ocular tilt reaction(OTR)
head tilt , skew deviation , OCR
tilt of vestibular perception [subjective visual vertical (SVV)]



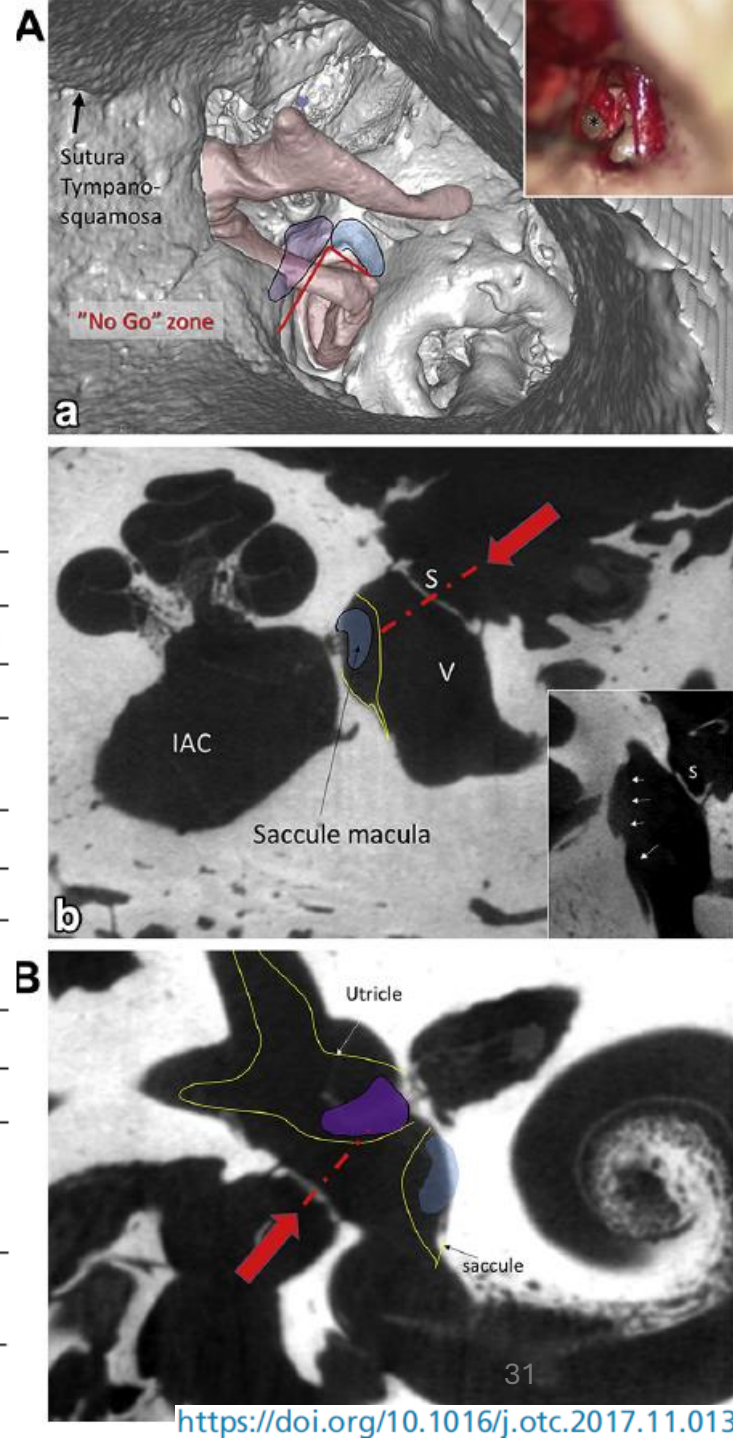
vertiginous attacks with low tone AB gap

J Int Adv Otol 2020; 16(1): 111-6

Table 1. Diagnostic strategy to differentiate middle-ear involvement, third-window lesions, and endolymphatic hydrops conditions

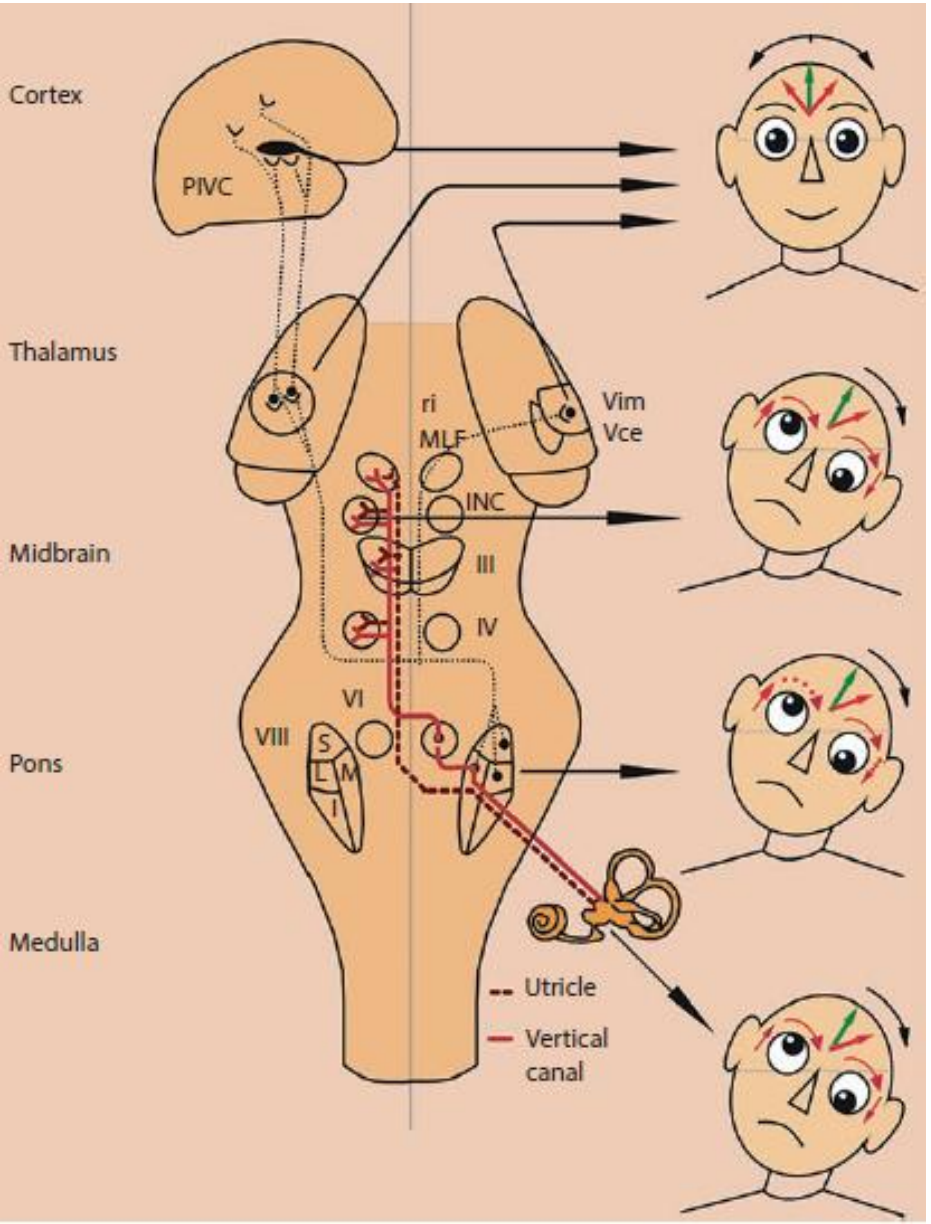
Diagnostic measures	Middle ear involvement	Third-window lesion	Endolymphatic hydrops conditions
Type of hearing loss	Conductive hearing loss	Conductive or mixed hearing loss	Generally mixed or sensorineural hearing loss
Air-bone gap	0–60 dB, may involve all frequencies	0–60 dB, greatest at frequencies <2000 Hz	0–50 dB, greatest at frequencies <1000 Hz
Bone conduction thresholds	Rarely < 0 dB	Sometimes negative (–5 to –25 dB for low frequencies	Rarely < 0 dB
Tympanometry	Type A or B or C tympanogram	Type A tympanogram	Type A tympanogram
Acoustic reflex	Absent	Present	Generally present
Tullio phenomenon and/or Hennebert sign	Absent	May be present	Absent
Otoacoustic emissions	Absent	May be present	Generally absent
ECochG	Normal	Generally elevated SP/AP ratio	SP/AP ratio generally > 0.37
Cervical VEMP	Absent	Low threshold and large amplitude	Generally absent or lower peak-to-peak amplitude at 500 Hz that 1000 Hz (tuning shift)
CT/MRI scan	Middle ear abnormality	Third-window lesion	Distension of the structures filled with endolymph at MRI

ECochG: Electrocochleography; VEMP: vestibular evoked myogenic potentials; CT: computed tomography; MRI: magnetic resonance imaging; SP: Summating Potentials, AP: Action Potentials.



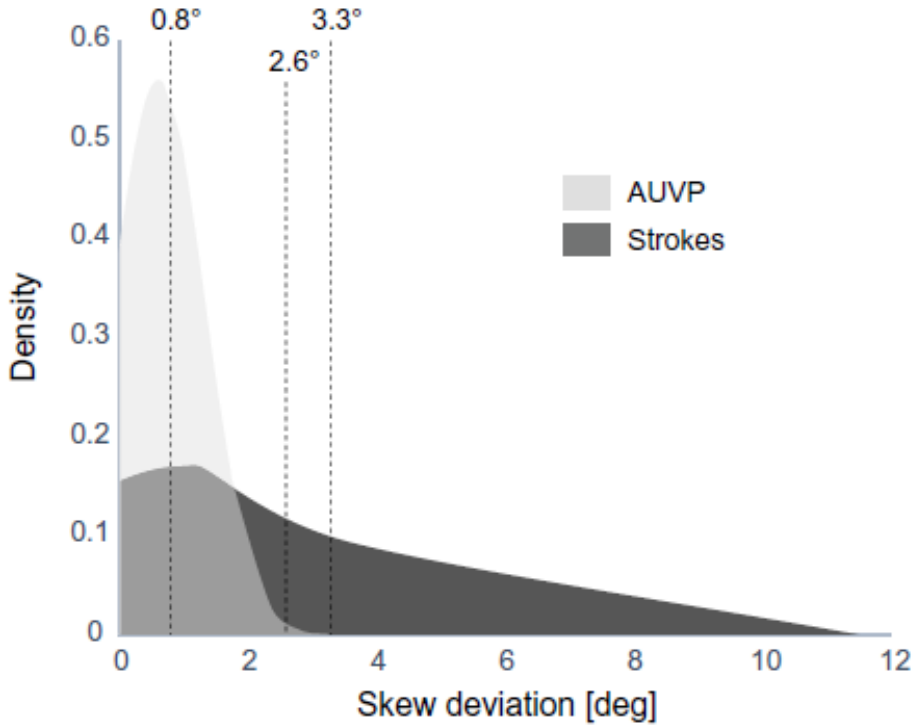
https://doi.org/10.1016/j.otc.2017.11.013

OTR : central or peripheral ?



Skew deviation

¼ patients with acute unilateral vestibulopathy have skew deviation
Large skew deviation (> 3.3°) seen in stroke patients
Low sensitivity but high specificity for posterior circulation stroke



Vertigo and Dizziness
Michael Strupp • Thomas Brandt • Marianne Dieterich

Journal of Neurology (2022) 269:1396–1403

SVV

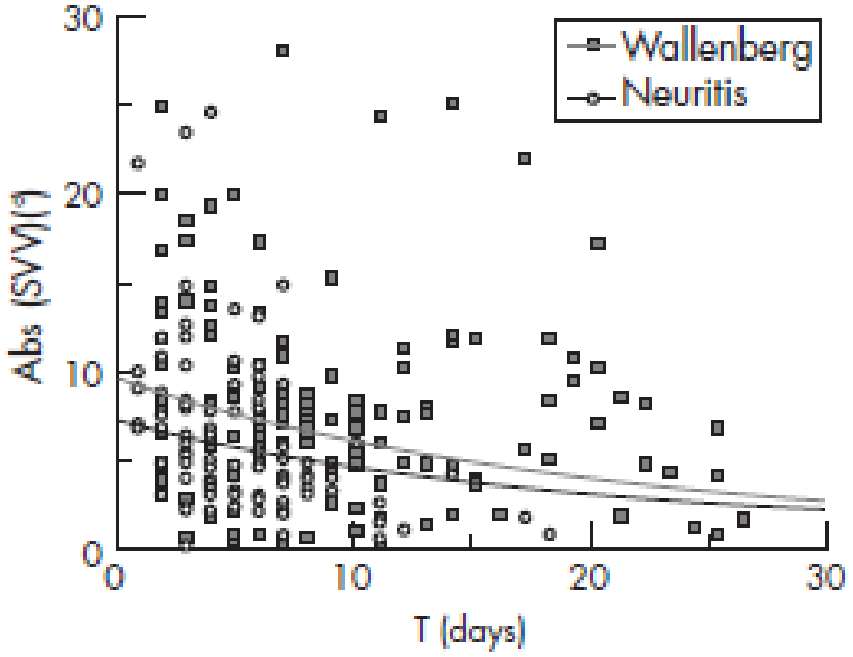


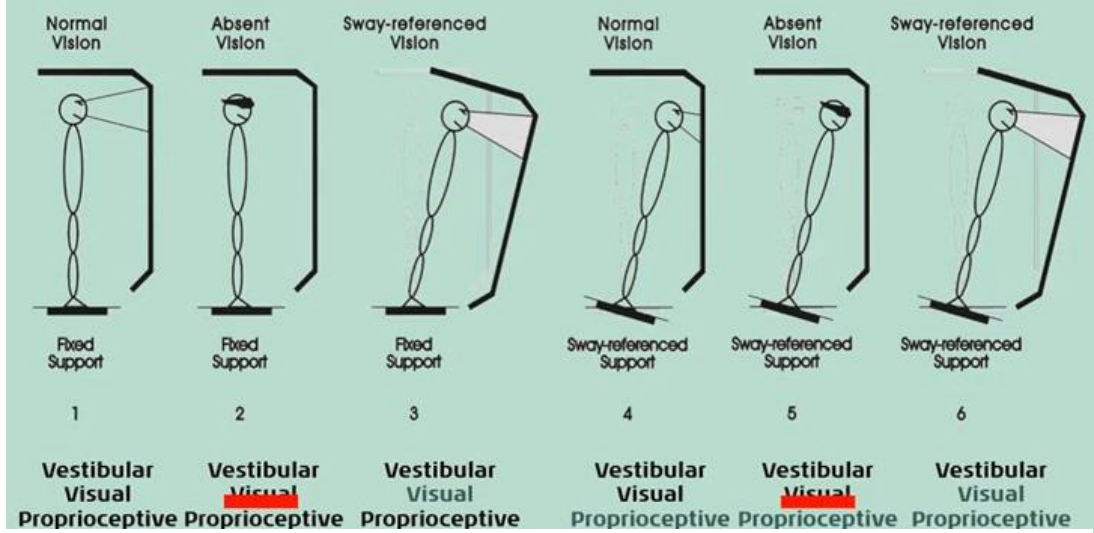
Figure 1 Subjective visual vertical (SVV) displacement (deviations to the left are flipped to the right side) in the course of time, and regression curves for 50 patients with Wallenberg's syndrome (Wallenberg) and 50 patients with vestibular neuritis (neuritis).

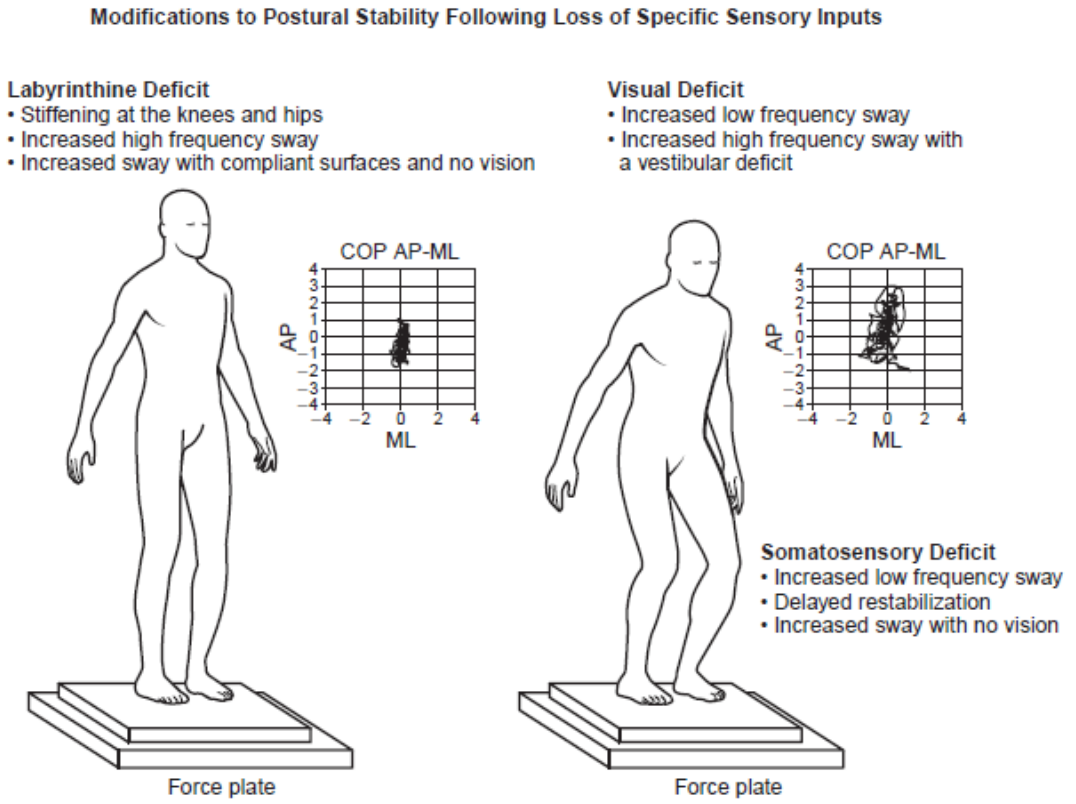
Dai *et al. Journal of Otolaryngology - Head and Neck Surgery*
https://doi.org/10.1186/s40463-020-0402-3

J Neurol Neurosurg Psychiatry 2007;**78**:527–528.

VSR

- Head acceleration
  - Limbs in the direction of acceleration extended
  - Limbs in the opposite contracted
- Lateral head tilt
  - Muscles of trunk and or lower limb on the side of tilt extended
  - On the opposite side flexed
- Maintain the center of pressure over the base of support –prevent falling
- Posturography – controversial role in diagnosis
  - Measurement of body sway under different conditions
  - Considered sensitive but not specific
  - New automatic analysis of the sway patterns and neural networks
    - Peripheral vestibular deficits
    - Cerebellar syndromes
    - Orthostatic tremor
    - Functional dizziness





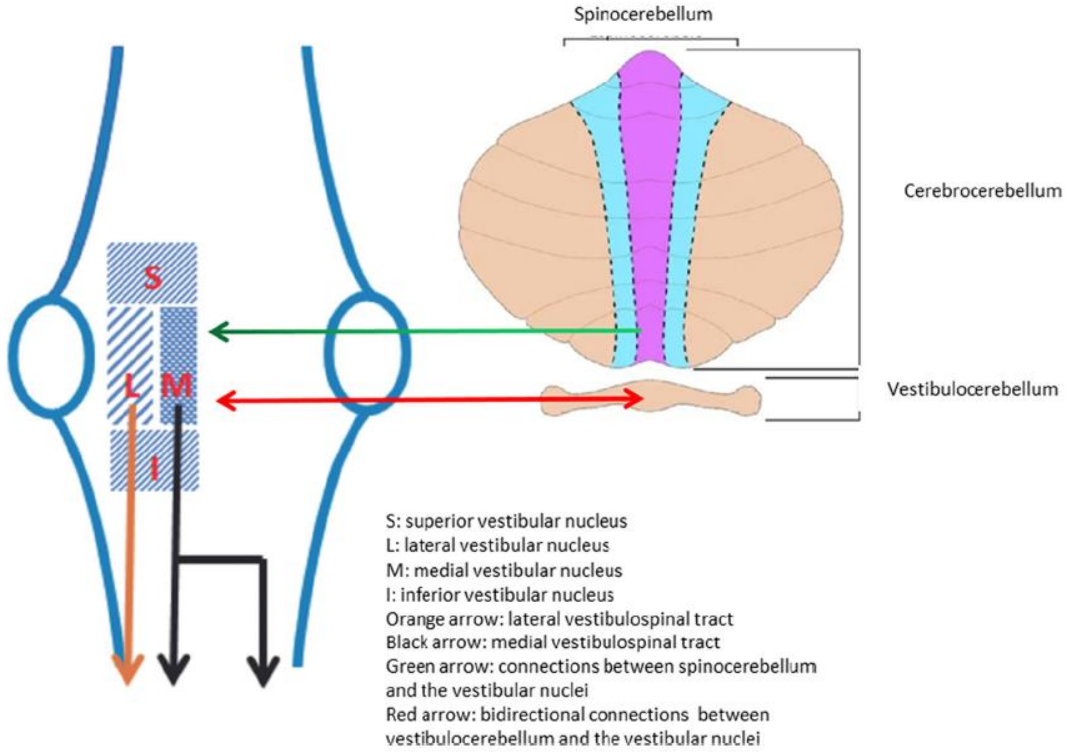


Fig. 1. Main Cerebellar and Vestibular nuclei, and descending vestibulospinal and vestibulocollic projections [modified on 1, 4].

Truncal ataxia in acute vestibular syndrome (AVS)

AVS

- sudden-onset, with spontaneous nystagmus
- Continuous vertigo lasting longer than 24 hours
- with associated nausea and vomiting, worsened with head movement

Lee (2006) classification truncal ataxia in acute vertigo patients

- Grade 1 : able to ambulate independently
- Grade 2 : unable to stand without support
- Grade 3 : unable to stand

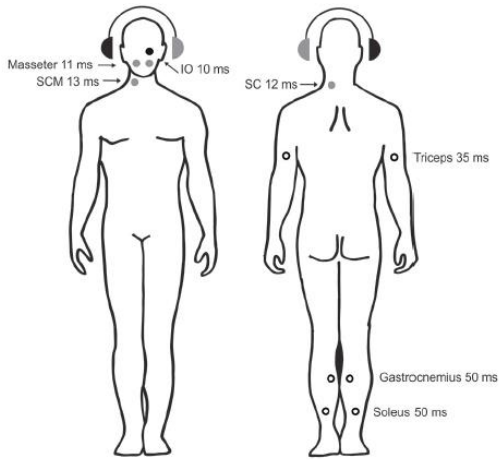
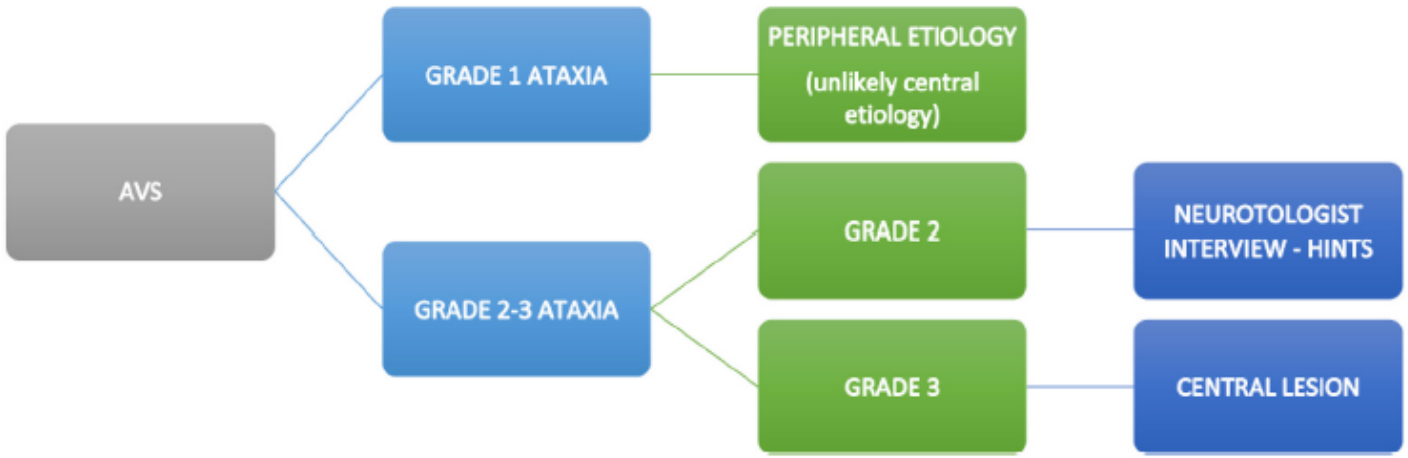


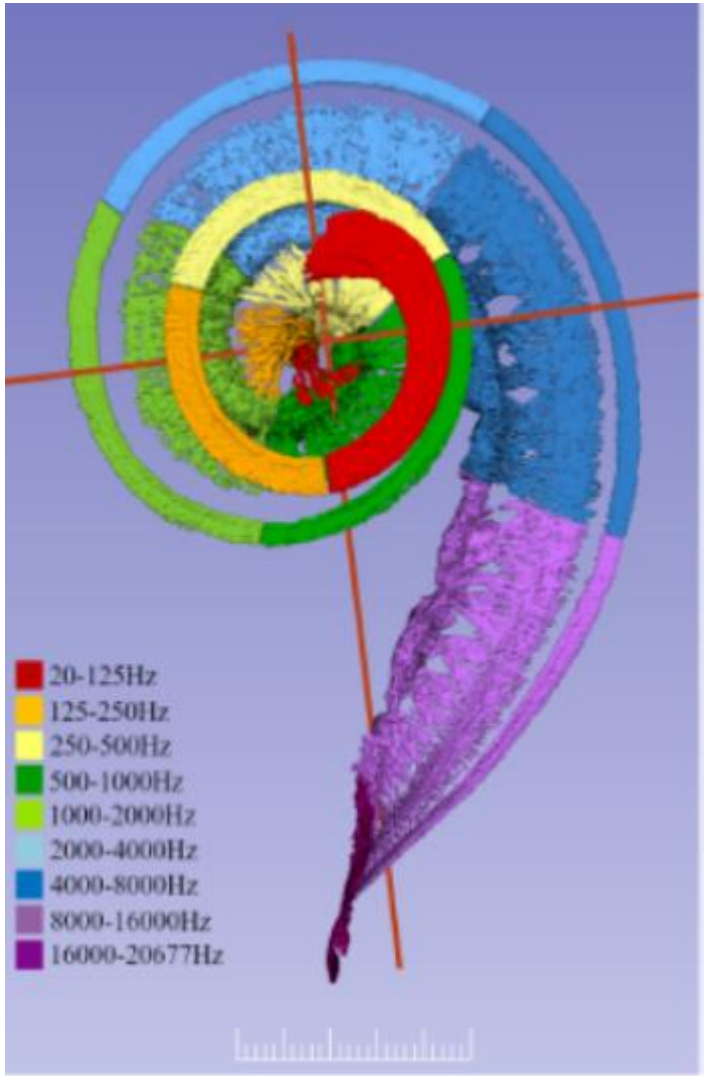
FIGURE 4 | Sound-evoked reflexes in postural muscles. The circles show the



published: 29 June 2018
doi: 10.3389/fneur.2018.00481

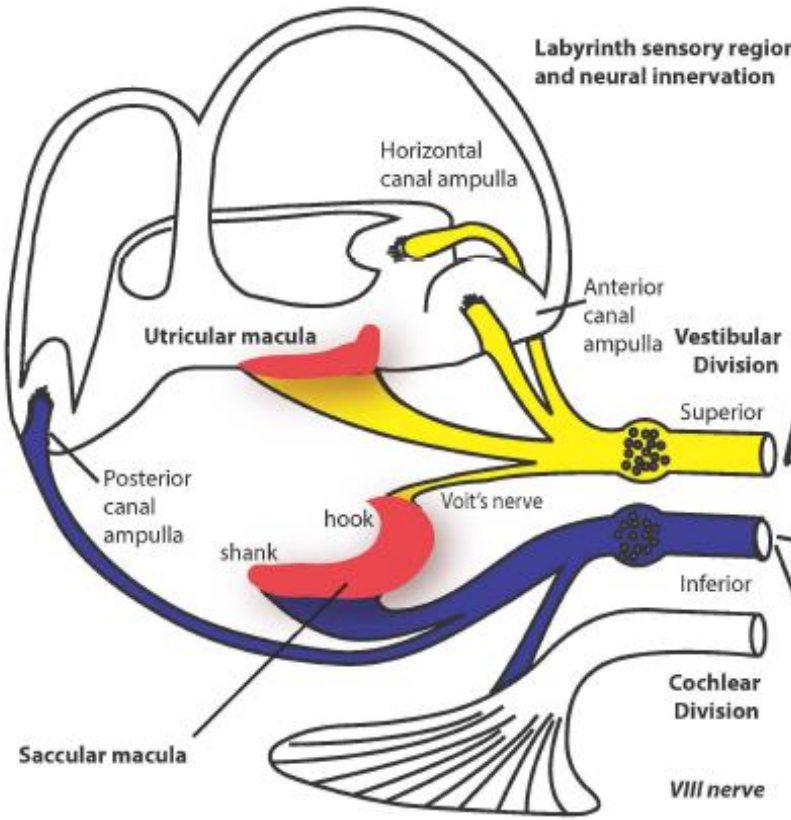
PTA and VFT(vHIT + VEMPs) – inner ear mapping

Rarely pathognomonic
Timing is important
Pattern recognition
No redundancy



| https://doi.org/10.1038/s41598-021-83225-w

I.S. Curthoys et al.



Clinical Test	Healthy Subject	Superior Vestibular Neuritis	Inferior Vestibular Neuritis	Unilateral Vestibular Loss
Horizontal canal - horizontal head impulse to the ipsilateral side	✓	✗	✓	✗
Anterior canal - pitch head impulse in the plane of the ipsilateral anterior canal, nose down	✓	✗	✓	✗
Utricular macula - oVEMP n10 beneath the ***contralateral*** eye to 500Hz bone conducted vibration at Fz, or 500Hz air-conducted sound of the ipsilateral ear	✓	✗	✓	✗
Saccular macula - cVEMP p13-n23 over the tensed ***ipsilateral*** sternocleidomastoid (SCM) muscle to 500Hz bone conducted vibration at Fz, or 500Hz air-conducted sound of the ipsilateral ear	✓	✓	✗	✗
Posterior canal - pitch head impulse in the plane of the ipsilateral posterior canal, nose up	✓	✓	✗	✗

✓ = Normal Response ✗ = Abnormal Response

VFT in TITRATE

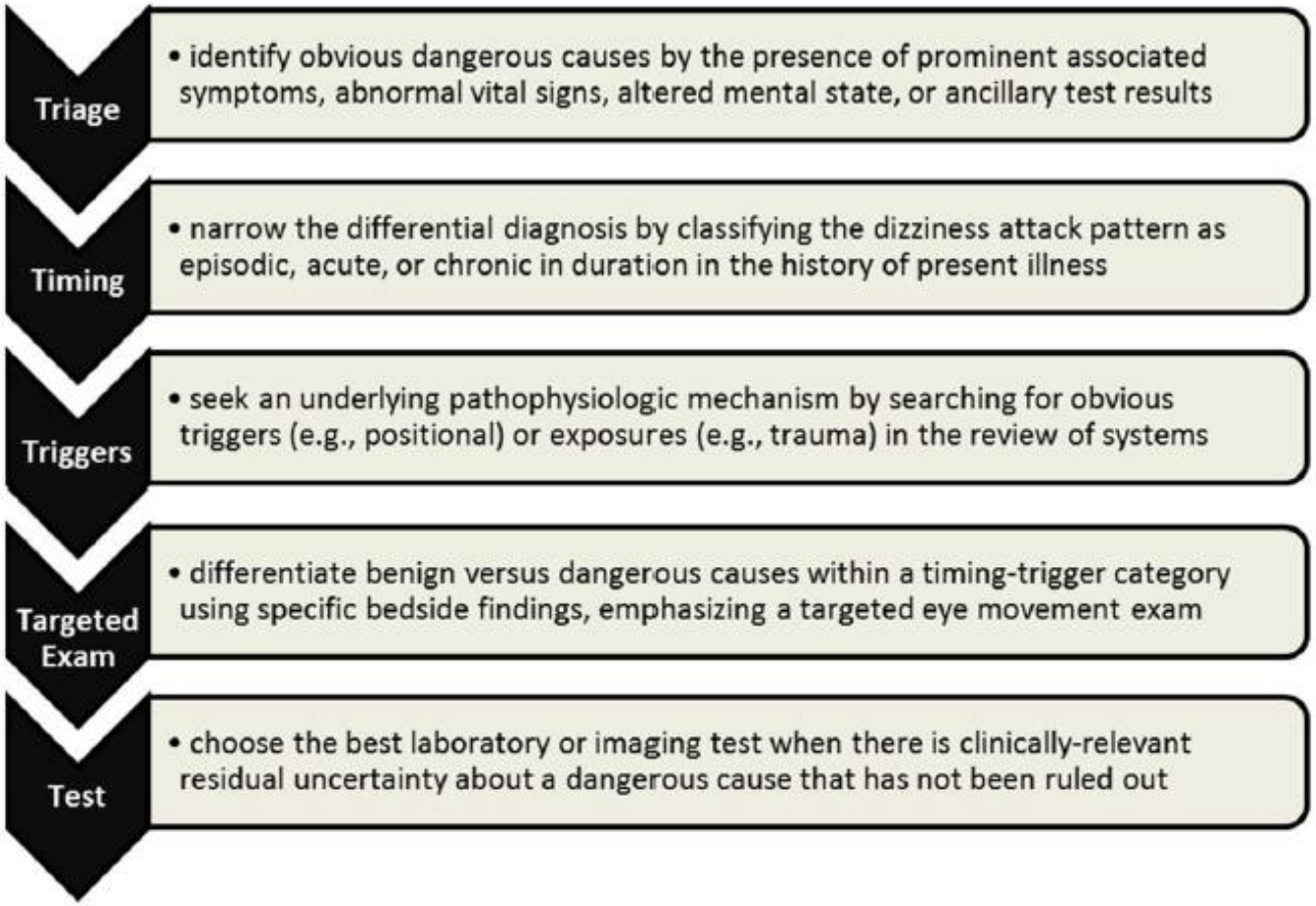


Figure 1. The "Triage – TITRATE – Test" approach to diagnosing dizziness and vertigo
The 'TI.TR.A.T.E.' acronym stands for **TI**ming, **TR**iggers, **A**nd **T**argeted **E**xams.

Table 1

Timing-and-trigger-based 'vestibular* syndromes'

Timing	Triggers† Present	No Triggers
New, episodic	**Triggered episodic vestibular syndrome (t-EVS)** (e.g., positional vertigo from BPPV)	**Spontaneous episodic vestibular syndrome (s-EVS)** (e.g., cardiac arrhythmia)
New, continuous	**Post-exposure acute vestibular syndrome (t-AVS)** (e.g., post gentamicin)	**Spontaneous acute vestibular syndrome (s-AVS)** (e.g., posterior fossa stroke)
Chronic, persistent	**Context-specific chronic vestibular syndrome (t-CVS)** (e.g., uncompensated unilateral vestibular loss, present only with head movement)	**Spontaneous chronic vestibular syndrome (s-CVS)** (e.g., chronic, persistent dizziness associated with cerebellar degeneration)

* Note that the use of the word 'vestibular' here connotes vestibular *symptoms* (dizziness or vertigo or imbalance or lightheadedness, etc.), rather than underlying vestibular *causes* (e.g., benign paroxysmal positional vertigo, vestibular neuritis).

† 'Triggers' here for non-spontaneous forms refer to obligate triggers (EVS), exposures (AVS), and contexts (CVS) that sharply distinguish these forms from their spontaneous counterparts. Spontaneous causes, as defined here, sometimes have underlying predispositions or precipitants, but these are not 'only-and-always.'

s-AVS

Syndrome	Description	Common Benign Causes	Common Serious Causes
AVS	Acute, continuous dizziness lasting days, accompanied by nausea, vomiting, nystagmus, head motion intolerance, and gait unsteadiness	Vestibular neuritis, Labyrinthitis	Posterior circulation ischemic stroke

Use of the Physical Examination to Diagnose Patients With Acute Vestibular Syndrome

Exam Component	Peripheral (All Must Be Present to Diagnose Vestibular Neuritis)	Central (Any One of These Findings Suggests Posterior Fossa Stroke)
Nystagmus (straight-ahead gaze and rightward and leftward gaze)	Dominantly horizontal, direction-fixed, beating away from the affected side *	Dominantly vertical or torsional or dominantly horizontal, direction-changing on left/right gaze †
Test of Skew (alternate cover test)	Normal vertical eye alignment and no corrective vertical movement (i.e., no skew deviation)	Skew deviation (small vertical correction on uncovering the eye) ‡
Head Impulse Test	Unilaterally abnormal with head moving towards the affected side (presence of a corrective re-fixation saccade towards the normal side) §	Usually bilaterally normal (no corrective saccade)

Stroke. 2009 November

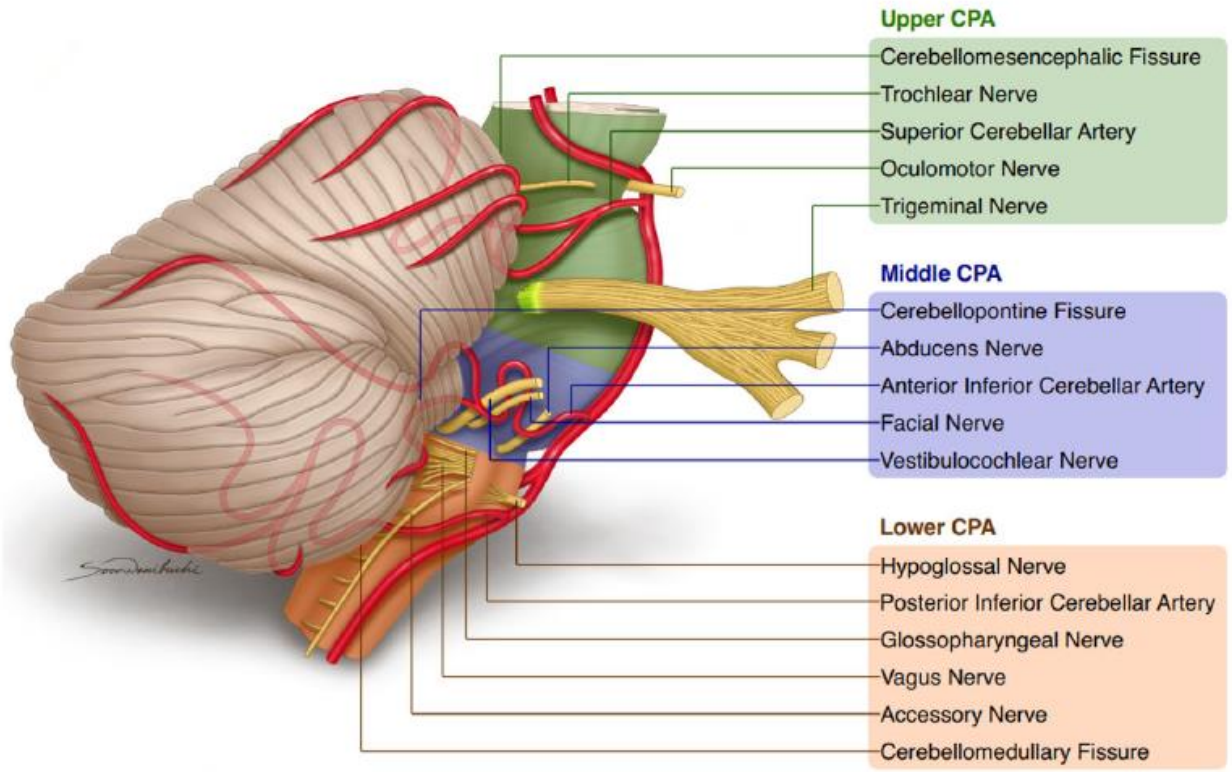


Figure 1. Summary of basic knowledge about superior cerebellar artery. Legend: CPA—cerebellopontine angle.

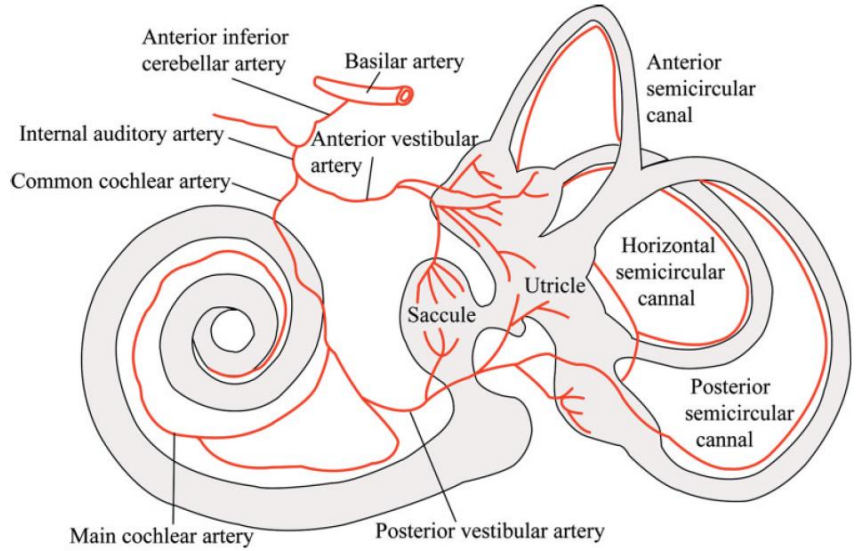


Figure 2 The arterial supply to the inner ear.

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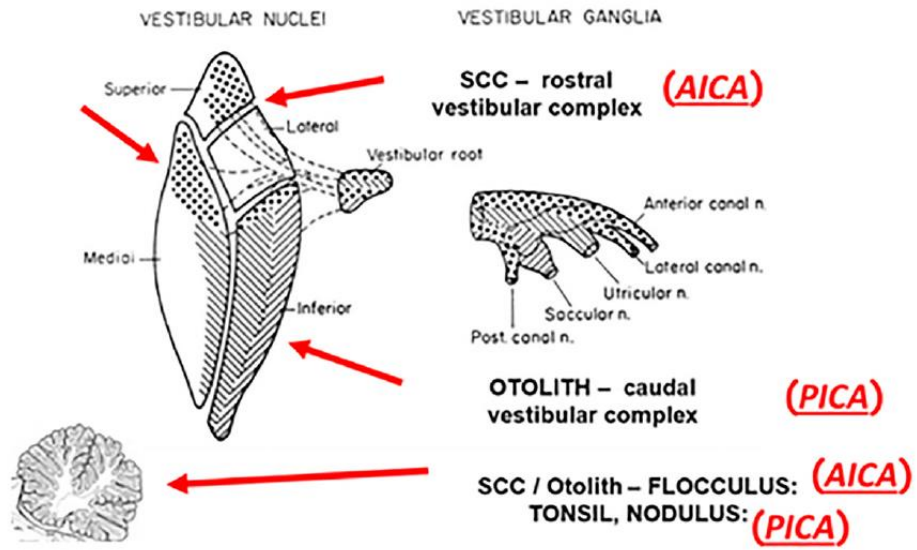


Table 4: Pooled analysis of key bedside diagnostic predictors of stroke in patients with acute vestibular syndrome*

Bedside diagnostic predictor*	No. of studies reporting data on total/peripheral/central causes	No. of patients, with peripheral/central causes	Sensitivity (95% CI†)	Specificity (95% CI†)	Negative likelihood ratio (95% CI†)	Positive likelihood ratio (95% CI†)
Normal result of horizontal head impulse test						
All central causes[6,10,11,33]	4/2/4	65/152§	0.85 (0.79–0.91)	0.95 (0.90–1.00)	0.16 (0.11–0.23)	18.39 (6.08–55.64)
PICA or SCA stroke[6,10,33]	3/1/3	25/72 (68 PICA)	0.99 (0.96–1.00)	–**	0.01 (0.00–0.10)	–**
AICA stroke[6,10]	2/1/2	25/13	0.62 (0.35–0.88)	–**	0.40 (0.20–0.80)	–**
Direction-changing nystagmus‡[6,9–11,27,28]	6/3/6	83/239§	0.38 (0.32–0.44)	0.92 (0.86–0.98)	0.68 (0.60–0.76)	4.51 (2.18–9.34)
Skew deviation[6,11]	2/2/2	65/119§	0.30 (0.22–0.39)	0.98 (0.95–1.00)	0.71 (0.63–0.80)	19.66 (2.76–140.15)

Note: AICA = anterior inferior cerebellar artery, CI = confidence interval, PICA = posterior inferior cerebellar artery, SCA = superior cerebellar artery.
*Only studies with an adequate (medium-quality) or superior (high-quality) reference standard for stroke diagnosis were considered. This table includes all bedside tests used in at least two studies that provided data for either peripheral or central causes of acute vestibular syndrome. Bedside examination of smooth-pursuit eye movements, optokinetic nystagmus, saccades, positional nystagmus, head-shaking nystagmus, vibration-induced nystagmus and hearing loss are not shown because of insufficient study numbers (< 2) to offer pooled results.
†Calculated by the method described by Simel et al.[25] Upper-bound values of sensitivity or specificity that were calculated to be greater than 100% using this method were assigned a value of 1.0.
‡Data based on bedside clinical findings and electro-oculography were pooled for these estimates because not all of the studies reported separate results for the two methods of determining nystagmus.
§Includes 27 patients with central causes other than ischemic stroke (14 with demyelination; 5 with brainstem or cerebellar hemorrhage; 8 with other causes).
**Pooled specificity is not calculated for this subset of patients with stroke because only one study reporting stroke location also included patients with peripheral causes. Specificity of a normal result of the head impulse test for predicting stroke should not differ based on stroke location.

Table 1.

Ocular motor and auditory features of acute peripheral and central vestibulopathies

Clinical Disorder	Head Impulse	Nystagmus with Right Lesion	Skew Deviation	Hearing Loss
Vestibular neuritis (VN) Labyrinthitis or cochlea-vestibular neuritis (CVN)	Unilateral decrease	Contralesional-beating, direction-fixed, obeys Alexander's law	Rare [22,60] *	VN: absent † CVN: present † (mild to severe [34])
VN or CVN complete (AC, HC, PC, utricle, saccule)	AC, HC, PC	H: left (major) V: none T: left ear beating (minor)	As above	As above
VN or CVN superior branch (AC, HC, utricle)	AC, HC	H: left (major) V: upbeat (minor) T: left ear beating (minor)	As above	As above
VN or CVN inferior branch (PC, saccule)	PC	H: none V: downbeat (major) T: left-ear beat (minor)	None [35]	As above (n=3/9 [35])
AICA Stroke	Unilateral decrease ~50% [22,34]; occasional bilateral asymmetric decrease [25,36]	50% direction-changing, gaze-evoked nystagmus [34]	28% [34]	Present in 56% [34] (mild to severe [34])
PICA or SCA Stroke	99% clinically normal bilaterally [22]	38% direction-changing, gaze-evoked nystagmus [22]	30% [22]	Rare [37,38]

* Skew deviation is well known in vestibular neurectomy, but rarely found in vestibular neuritis or labyrinthitis (2%, n=2/91[22,34,35]). This may be because small skew deviations (<2–4 prism diopters) are not easily identified at the bedside by alternate cover testing.

† Standard terminology suggests that, by definition, hearing loss is absent in vestibular neuritis and present in labyrinthitis.[39] However, these terms are used inconsistently in clinical practice and the medical literature. As yet, there is no way to reliably determine whether a lesion is located in the vestibular nerve or the labyrinth in the majority of acute cases.

Abbreviations: AC – anterior canal; AICA – anterior inferior cerebellar artery; CVN – cochleo-vestibular neuritis (i.e., labyrinthitis); H – horizontal; HC – horizontal canal; PC – posterior canal; PICA – posterior inferior cerebellar artery; SCA – superior cerebellar artery; T – torsional; V – vertical; VN – vestibular neuritis

Table 4
ABCD2 ≥ 4 Versus HIT, HINTS, and HINTS "plus" for Stroke or Central Cause in AVS

Test Properties	ABCD2 ≥ 4 (Five-item Rule*)	HIT (One-step Rule*)	HINTS (Three-step Rule*)	HINTS "Plus" (Four-step Rule*)
Stroke only (n = 113 stroke, n = 77 nonstroke)				
Sensitivity for stroke	61.1 (51.8–69.7)	90.3 (83.7–94.8)	96.5 (91.7–98.9)	99.1 (95.7–100.0)
Specificity for stroke	62.3 (51.2–72.6)	87.0 (78.1–93.2)	84.4 (75.0–91.3)	83.1 (73.5–90.3)
LR+ stroke	1.62 (1.17–2.24)	6.95 (3.89–12.43)	6.19 (3.68–10.42)	5.87 (3.58–9.64)
LR– stroke	0.62 (0.47–0.83)	0.11 (0.06–0.20)	0.04 (0.02–0.11)	0.01 (0.00–0.08)
Reduction missed stroke†	Reference case	75.0	90.9	97.7
Any central cause (n = 124 central, n = 66 peripheral)				
Sensitivity for central	58.1 (49.2–66.5)	91.1 (85.1–95.3)	96.8 (92.4–99.0)	99.2 (96.1–100.0)
Specificity for central	60.6 (48.5–71.8)	100.0 (95.6–100.0)	98.5 (92.8–99.9)	97.0 (90.4–99.5)
LR+ any central cause	1.47 (1.05–2.06)	>91.1‡ (NC)	63.9 (9.13–446.85)	32.7 (8.36–128.16)
LR– any central cause	0.69 (0.52–0.92)	0.09 (0.05–0.16)	0.03 (0.01–0.09)	0.01 (0.00–0.06)
Reduction missed central†	Reference Case	78.8	92.3	98.1

Data are reported as percentages, except LRs, with (95% CI)
ABCD2 = age, blood pressure, clinical features, duration of symptoms, diabetes; AVS = acute vestibular syndrome; LR+ = positive likelihood ratio; LR– = negative likelihood ratio; HINTS = head impulse, nystagmus type, test of skew; HINTS "plus" = HINTS plus new hearing loss detected by finger rubbing; HIT = head impulse test.
*The ABCD2 rule requires five historical elements. The standard HINTS approach has three physical examination elements, the most predictive of which is the HIT. HINTS "plus" adds the presence of new hearing loss by bedside finger rub as a predictor of a stroke syndrome.
†These values represent the reduction in missed stroke or central causes relative to ABCD2 that would be projected if HIT, HINTS, or HINTS "plus" were used to determine the diagnosis instead of ABCD2.
‡The LR+ for HIT alone was calculated using a specificity of 99.0% and listed as ">" since the LR+ associated with 100% specificity (measured in this sample) is infinite.

Peripheral Vestibulopathy Presenting as Acute Vertigo and Spontaneous Nystagmus with Negative Video Head Impulse Test

Jung-Yup Lee, MD[1], Chang-Hee Kim, MD, PhD[2], Jin Su Park, MD[1] and Min-Beom Kim, MD, PhD[1]

Table 2. Number of Vertigo Episode and Pure Tone Average of Threshold Values at 0.5, 1, 2, and 4 kHz of 17 Patients with Ménière's disease.

No.	Vertigo Episode	Threshold, dB
1	First	23
2	First	20
3	First	16
4	First	17
5	Third	15
6	Second	46
7	First	15
8	Third	23
9	First	29
10	Second	52
11	First	18
12	Second	50
13	First	23
14	Second	10
15	First	42
16	First	16
17	First	15

Table 1. Final Diagnosis and Initial Bedside Examination Findings of 30 Patients with Acute Vertigo Showing Spontaneous Nystagmus with Negative Video Head Impulse Test.

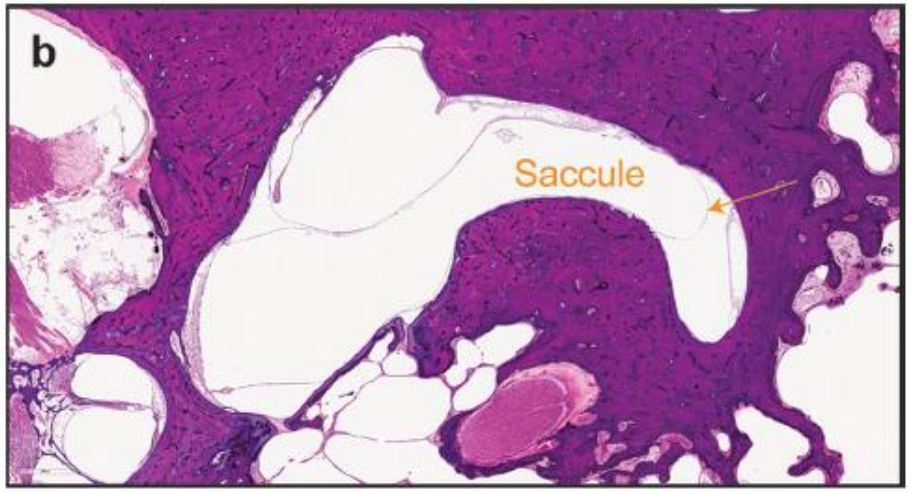
No.	Sex	Age, y	Onset, d	Underlying Disease	Diagnosis[a]
1	F	67	<1		MD
2	M	71	<1		MD
3	F	58	<1		MD
4	M	40	1		MD
5	F	25	1		MD
6	F	61	<1		MD
7	M	49	1		MD
8	M	49	<1		MD
9	F	20	<1		MD
10	M	60	2		MD
11	F	51	2		MD
12	F	78	<1	HTN	MD
13	F	55	2		MD
14	M	42	1		MD
15	M	62	1		MD
16	F	35	1		MD
17	F	64	2		MD
18	M	67	1		SSNHL_V
19	F	57	3		SSNHL_V
20	M	67	<1	DM	SSNHL_V
21	F	19	3		SSNHL_V
22	F	72	2	HTN, DM	SSNHL_V
23	M	55	<1		SSNHL_V
24	F	79	1		SSNHL_V
25	M	63	2	HTN, DM	Medullary infarction
26	M	72	2	HTN	Cerebellar infarctionb
27	M	64	2	HTN	Cerebellar infarction
28	F	58	1		Vestibular migraine
29	M	79	2		Vestibular migraine
30	F	57	2		Inferior vestibular neuritis

Abbreviations: DM, diabetes mellitus; F, female; HTN, hypertension; M, male; MD, Ménière's disease; SSNHL_V, sudden sensorineural hearing loss with vertigo.

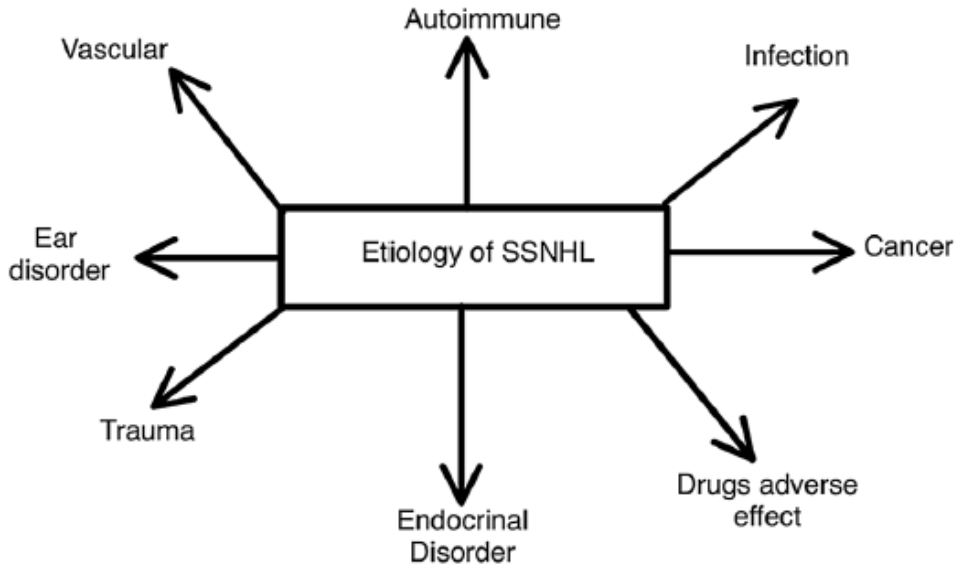
[a]Results for direction-changing gaze-evoked nystagmus, head impulse test, and skew deviation were negative for all patients unless noted otherwise.

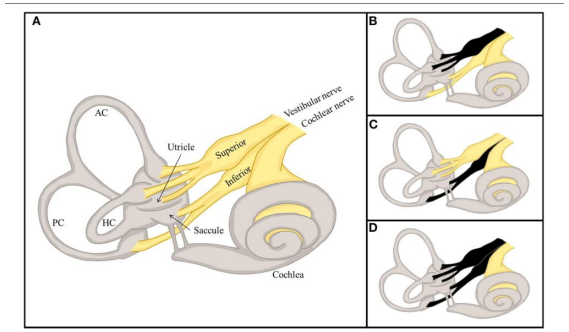
[b]This patient tested positive for direction-changing gaze-evoked nystagmus.

Meniere's disease (MD)



Sudden sensorineural hearing loss (SSNHL)





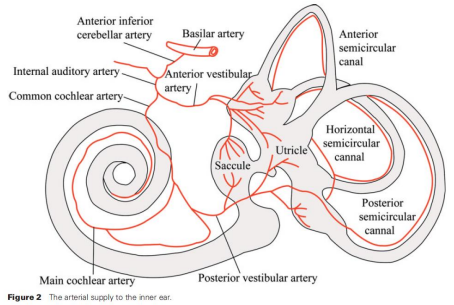


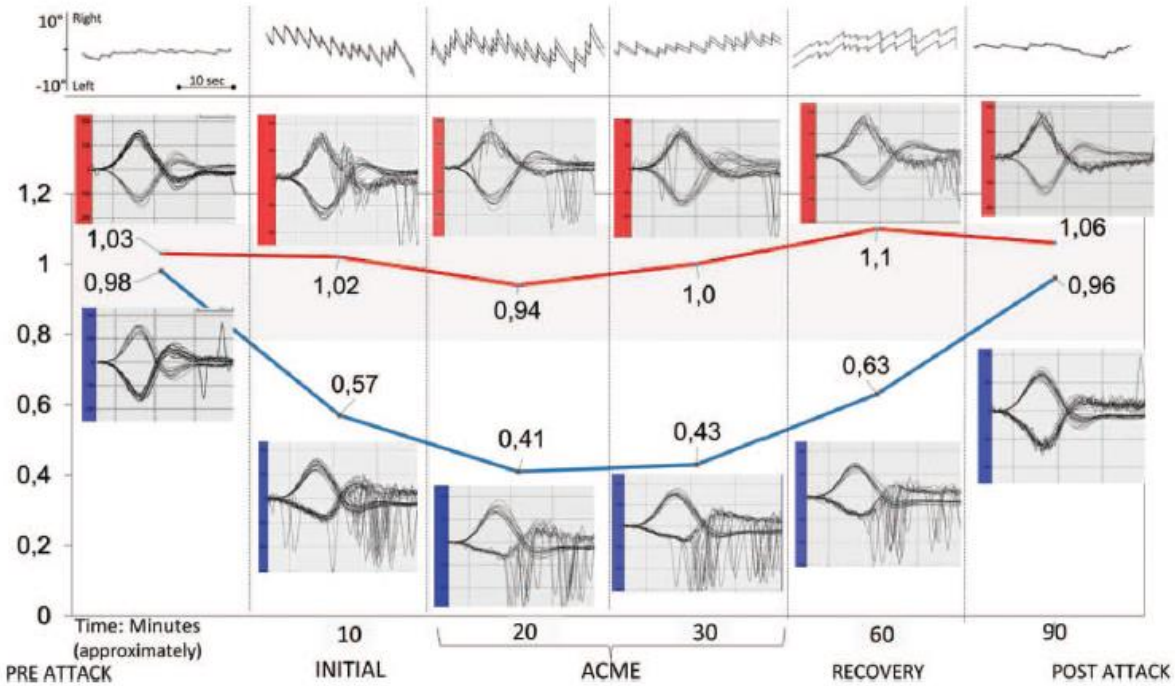
Figure 2 The arterial supply to the inner ear.

Cureus 14(9): e29458. DOI 10.7759/cureus.29458 March 2020 | Volume 11 | Article 157

TABLE 1. *Individual demographic data and results of the vestibular function testing*

			Quiescent Stage			Acute Episode with Vertigo (Attack) VHIT Gain (Affected Side)				
Patient (Attack)	Age (yr)	Gender	Time of Disease (yr)	Affected Side	UW-Caloric Test (% Asymmetry)	Pre-attack	Initial	Acme	Recovery	Post Attack
1	80	M	5	Right	49	1.01	0.71	0.31	0.91	0.96
2	66	F	6	Left	39	0.98	0.57	0.41	0.63	0.96
3	65	F	3	Right	NP	0.95	0.56	0.54	0.70	0.99
4	62	F	5	Right	32	0.92	–	0.78	0.81	0.91
5	47	F	1	Right	24	0.83	–	0.55	0.66	0.94
6	90	F	10	Right	NP	0.86	–	–	0.68	0.82
7	5	M	5	Left	NP	0.89	–	0.61	0.86	0.88
8	53	F	3	Left	43	0.84	–	0.68	0.75	0.82
9a	63	M	3	Left	40	0.93	0.3	0.1	0.44	0.84
9b	64	M	4	Left	40	0.9	–	–	0.42	0.85

Only the ipsilesional hVOR gain of the lowest magnitude during the attack are included. 9a/9b indicates two attacks recorded within a 1-year interval from the same patient; NP, not performed; UW, unilateral vestibular weakness of the affected ear.



Otolith Membrane Herniation, not Semicircular Canal Duct Dilation, Is Associated with Decreased Caloric Responses in Ménière's Disease

Leo L. Shen[1] · Nicholas S. Andresen[1] · Divya A. Chari[2] · Jacob M. Pogson[1,3] · Amanda M. Lauer[1,4] · Richard D. Rabbitt[5] · John P. Carey[1] · Felipe Santos[2] · Bryan K. Ward[1]

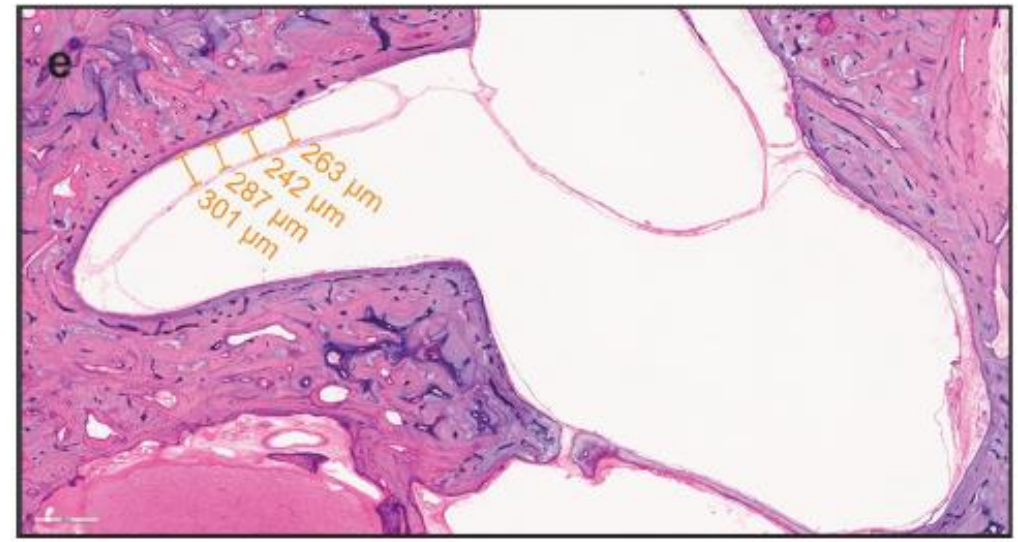


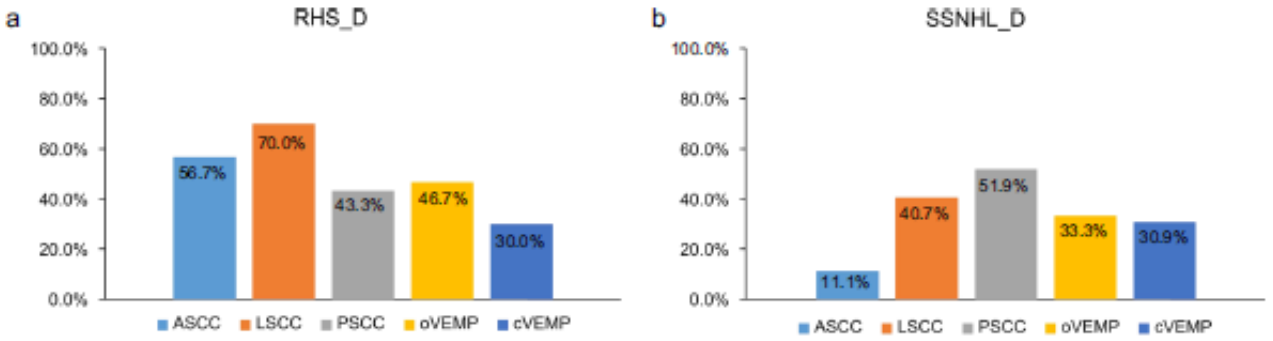
Table 2 Ocular Motor Findings of the Patients With Unilateral Meniere Disease During and Between the Attacks

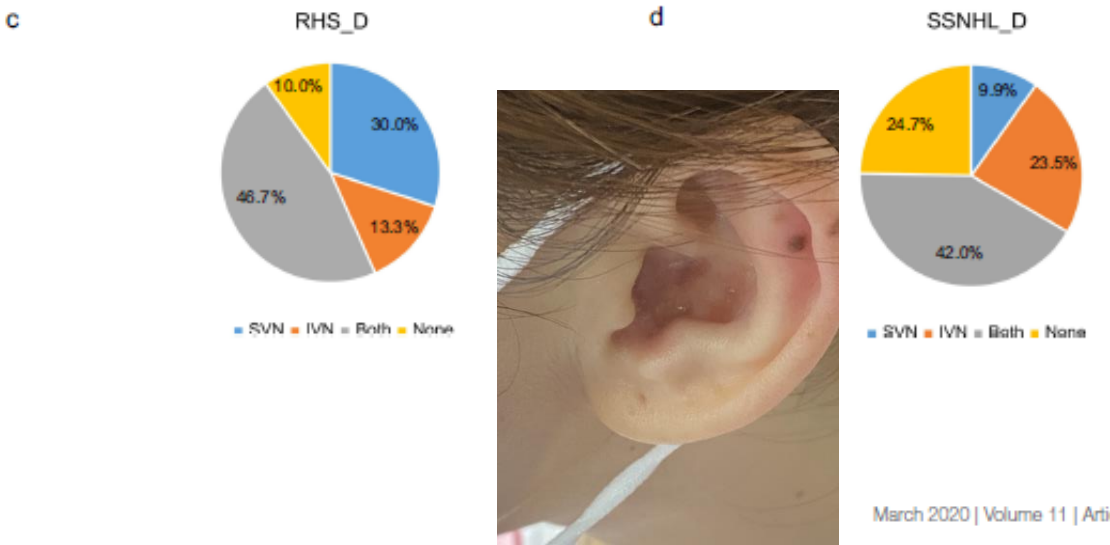
	Irritative/recovery	Paretic	Interictal phase
Spontaneous nystagmus	I	C	None or only subtle nystagmus
	Downbeat, discordant, aperiodic alternating nystagmus		
Video head-impulse tests	Mostly normal	Either decreased or normal VOR gain, frequent covert and overt saccades	Mostly normal
	Can be impaired for the PCs in case of downbeat nystagmus		
Canal paresis	I or C	I	I
Head-shaking nystagmus	I	C	—
Vibration-induced nystagmus	C	C	C
Positional nystagmus	Geotropic/apogeotropic or direction-fixed nystagmus		
Cervical VEMPs	Decreased response in the affected ear		
Ocular VEMPs	Augmented response in the affected ear		Variable

Abbreviations: C = contralesional, I = ipsilesional, PC = posterior canal, VEMPs = vestibular-evoked myogenic potentials, VOR = vestibulo-ocular reflex.

s-AVS with new hearing loss – SSNHL with vertigo viral ?

Ramsay Hunt syndrome vs SSNHL





 DOI 10.3389/fnins.2023.1102512

VN vs labyrinthitis

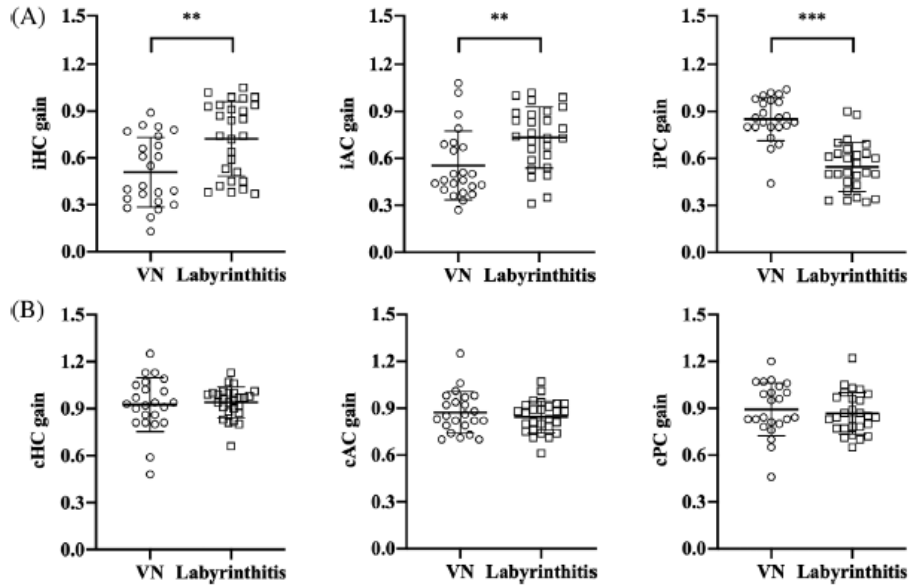
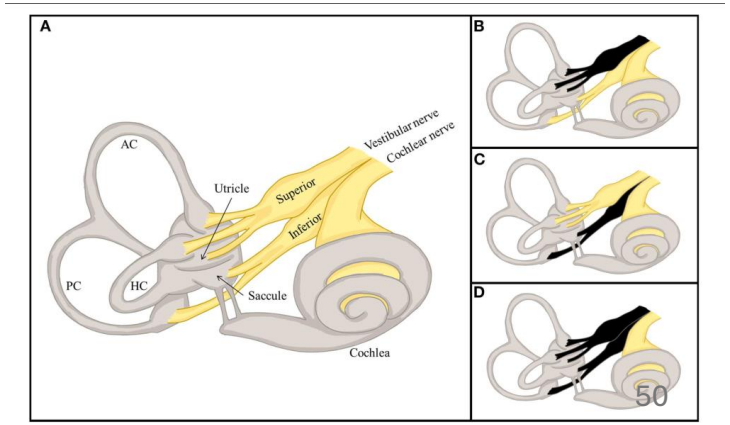


FIGURE 1 VOR gain of the (A) ipsilesional (i) and (B) contralesional (c) sides of three semicircular canals in patients with VN and labyrinthiti Compared with the VN group, the VOR gain for labyrinthitis patients was reduced in the iPC, but relatively preserved in the iHC and iAC. The VOR gain of the contralateral sides showed no statistically significant differences. ***p-value <.001, **p-value <.01, *p-value <.05. AC, anterior semicircular canal; HC, horizontal semicircular canal; PC, posterior semicircular canal; VN, vestibular neuritis; VOR, vestibulo-ocular reflex.



s-AVS with new hearing loss – SSNHL with vertigo vascular ?

TABLE 1. *Summary of clinical and histopathologic data*[a]

Case	Age at onset (yr)	Sex	Ear	Hearing loss	Vertigo	Died at age (yr)	Organ of corti: Hair cells	Organ of corti: Supporting cells	Tectorial membrane	Stria vascularis	Cochlear neurons	Spiral limbus	New bone in cochlea	URI
1	63	M	L	++++	++++	67	++++	++		+++				
2	57	M	R	++++		57	++++	++++	++++	+		+++		+
3	39	F	R	+++		59		+++	++++	++				+
4[b]	65	F	R	++++	++	85	+++	++						
5	43	F	L	+++	++	45	++	++						+
6	63	M	R	++++		64	+++	+++						+
7[b]	52	F	L	++++		85	++++	++		++++	+	++		
8	68	M	L	++++		87	++	++	++++	++++		+		+
9	63	M	R	++	+	67					+++			+
10	43	F	L	++++	+	48	+++	++						
11	25	M	L	++++	+++	47	++++	+++	++++	++	++	+		
12	46	M	R	++++	+	46	All structures appear normal (hearing recovered)							
13	40	F	R	++++		83	++++	++++	++++	+++	++	+++	+++	
14[b]	10	F	R	++++		94	++++	+++		+++	++	++		
15[b]	65	F	L	+++		94	+++	++		++				
16[b]	78	M	R	++		80	No histologic correlates (hearing recovered)							+
17[b]	78	M	L	++++	++	80	++++	++		+	+			+

[a]Cases 1–12 were previously reported from our laboratory (4–6). Cases 13–17 are described in detail in this report.
[b]Bilateral cases.
M, male; F, female; URI, upper respiratory infection; +, indicates presence of symptom or presence of abnormality; ++, +++, and ++++ indicate increasing severity of symptom or abnormality.

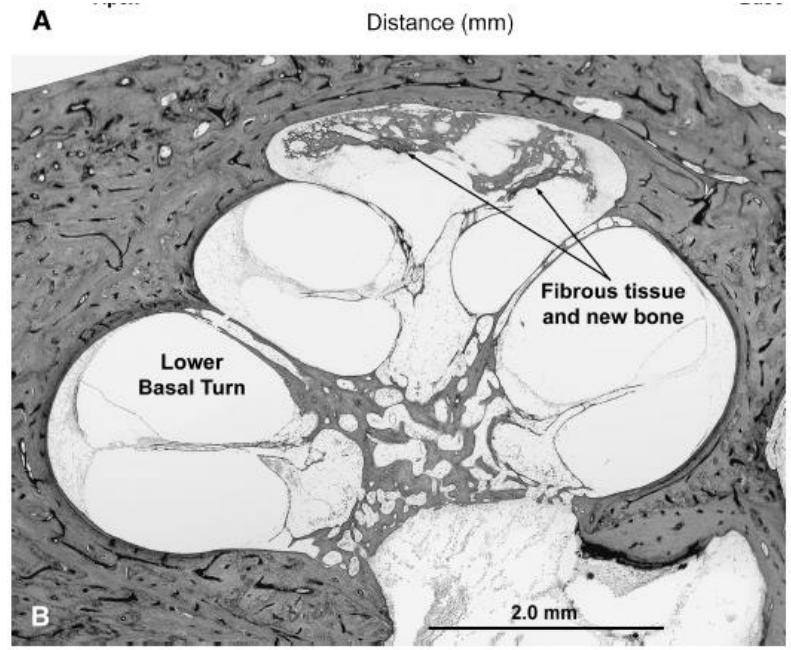


FIG. 1. Case 13, right ear, age 83 years. (*A*) Audiogram and cytocochleogram. Areas marked by an X in the cochleogram charts represent those regions of the inner ear where accurate assessments of the organ of Corti could not be made because the plane of section was tangential to the structure of interest. (*B*) Midmodiolar section showing complete degeneration of sensory and neural structures within the apical and middle turns, along with replacement by fibrous tissue and new bone. The cochlear duct is not affected in the lower basal turn, where hair cells are present with innervating dendrites.

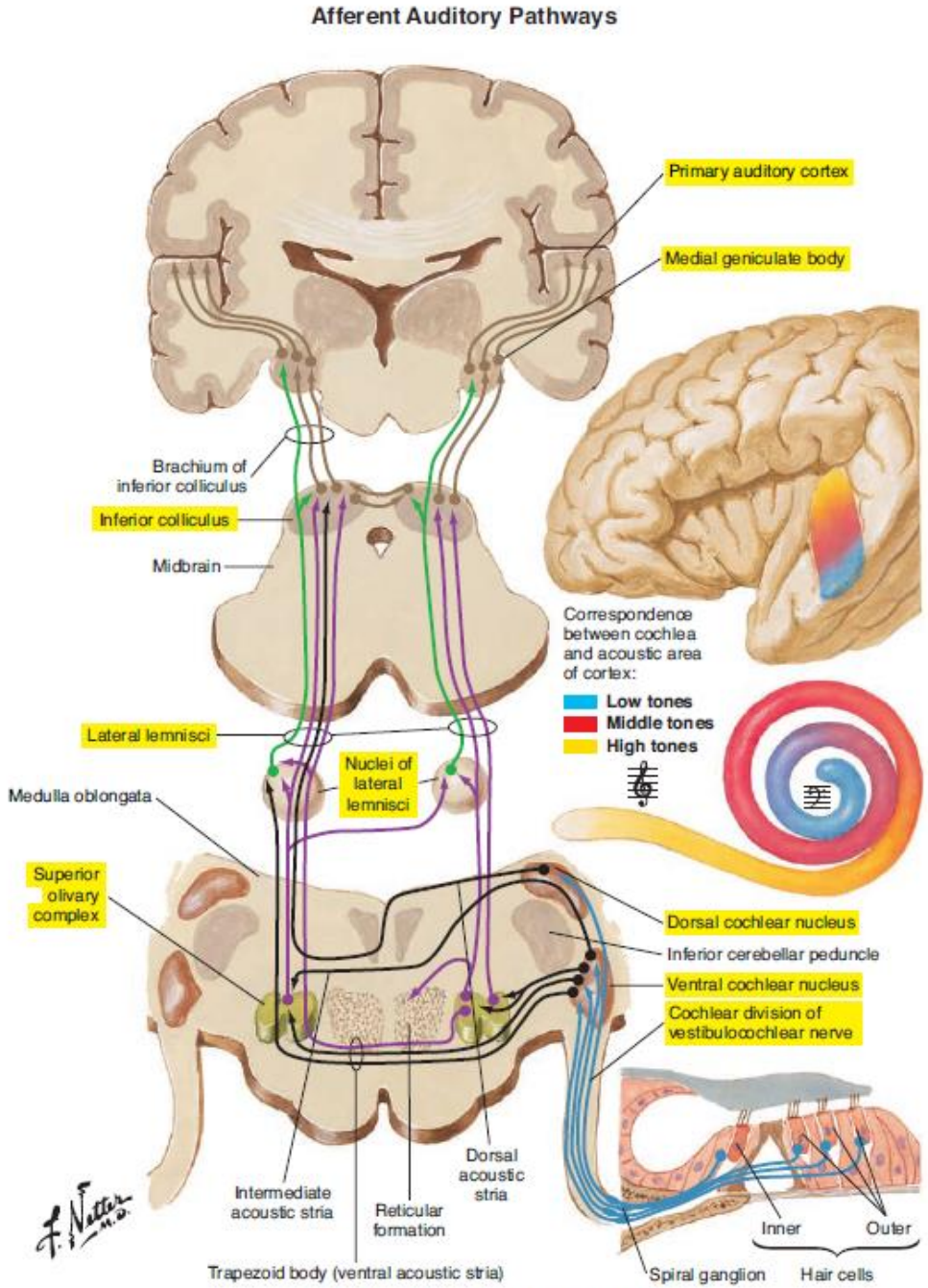
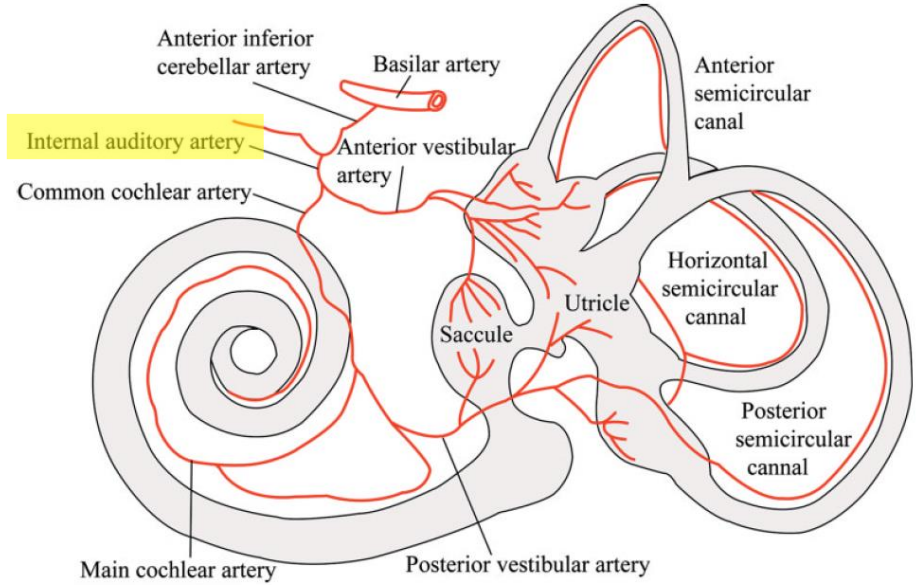


Figure 4-14. Afferent auditory pathways.



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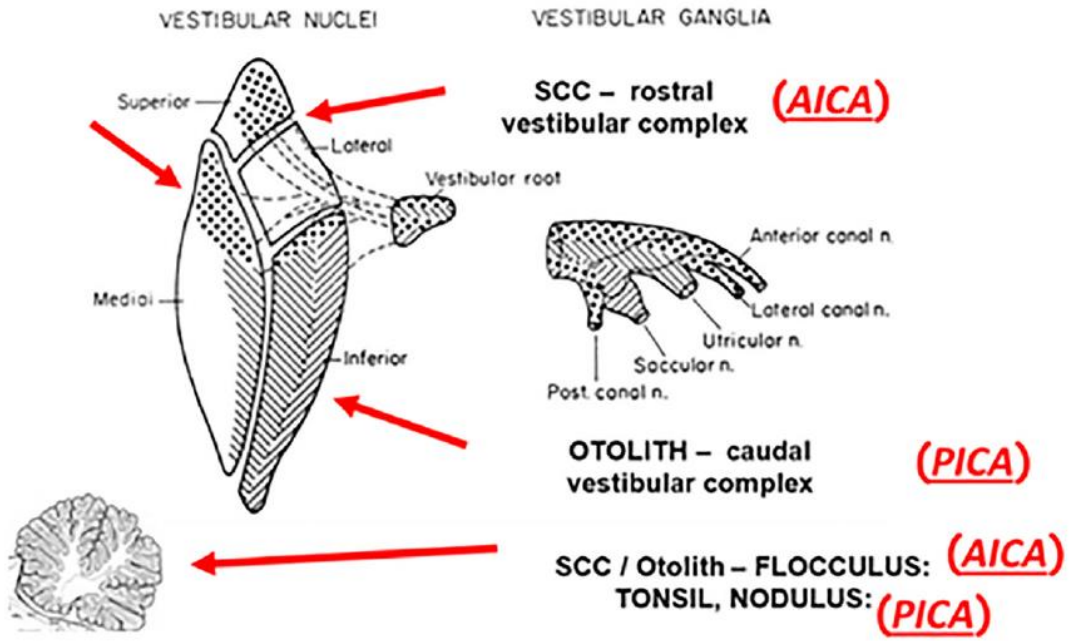

Journal of the Neurological Sciences 441 (2022) 120374

central vascular territory

Head-Shaking Aids in the Diagnosis of Acute Audiovestibular Loss due to Anterior Inferior Cerebellar Artery Infarction

Young Eun Huh[a] Ja-Won Koo[b] Hyung Lee[c] Ji-Soo Kim[a]

- 17 AICA infarction
  - 16 with vertigo
  - 10 without hearing loss
    - 6 normal gain of lateral HIT
    - 4 abnormal gain of ipsi lateral HIT
  - 7 with hearing loss
    - abnormal gain of ipsi lateral HIT
    - 3 profound HL, 3 moderate HL , 1 mild HL
- 21 labyrinthitis
  - vertigo and HL
  - abnormal gain of ipsi lateral HIT

Table I. *Auditory symptoms reported in strokes*

Symptom	Location of ischemia	
Hearing loss—unilateral	Ipsilateral	Cochlea Auditory nerve Cochlear nucleus Acoustic striae
Hearing loss—bilateral	Bilateral	Cochleae Auditory nerves Cochlear nuclei Acoustic striae Superior olivary complexes Lateral lemnisci Inferior colliculi Brachia of inferior colliculus Medial geniculate bodies Auditory radiations Primary auditory cortices and/or subcortical white matter
Tinnitus—unilateral	Ipsilateral	Cochlea Auditory nerve Cochlea nucleus Acoustic striae
Tinnitus—bilateral	Bilateral	Cochleae Auditory nerves Cochlear nuclei Acoustic striae
	Unilateral	Inferior colliculus (transient)
Hallucinations	Pontine tegmentum Midbrain ("peduncular") Non-dominant temporal lobe (musical)	
Hyperacusis	Unilateral inferior colliculus	

Positive horizontal-canal head impulse test is not a benign sign for acute vestibular syndrome with hearing loss

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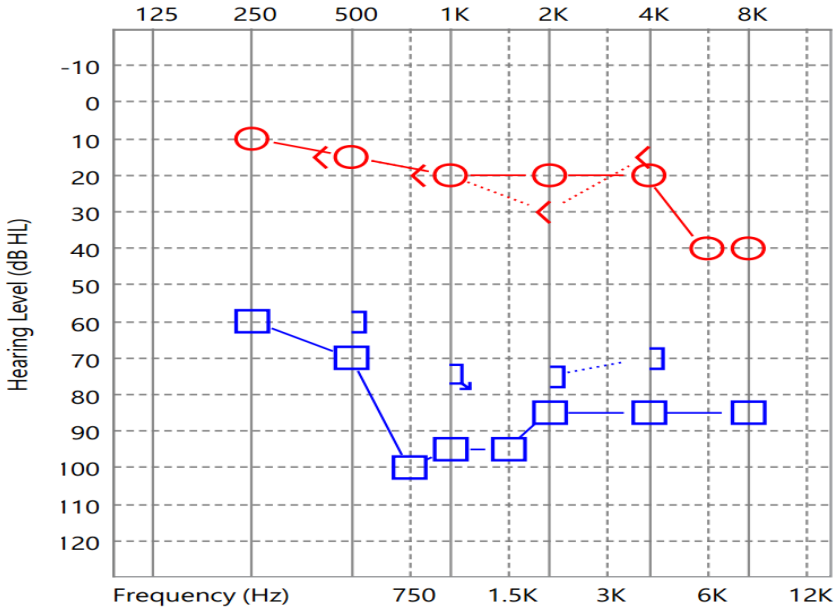
[1]Division of Neurology, Department of Medicine, University of Ottawa, Ottawa, ON, Canada, [2]Department of Neurology/Neuro-Medical Scientific Center, Taichung Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taichung, Taiwan, [3]Department of Neurology, School of Medicine, Tzu Chi University, Hualien, Taiwan

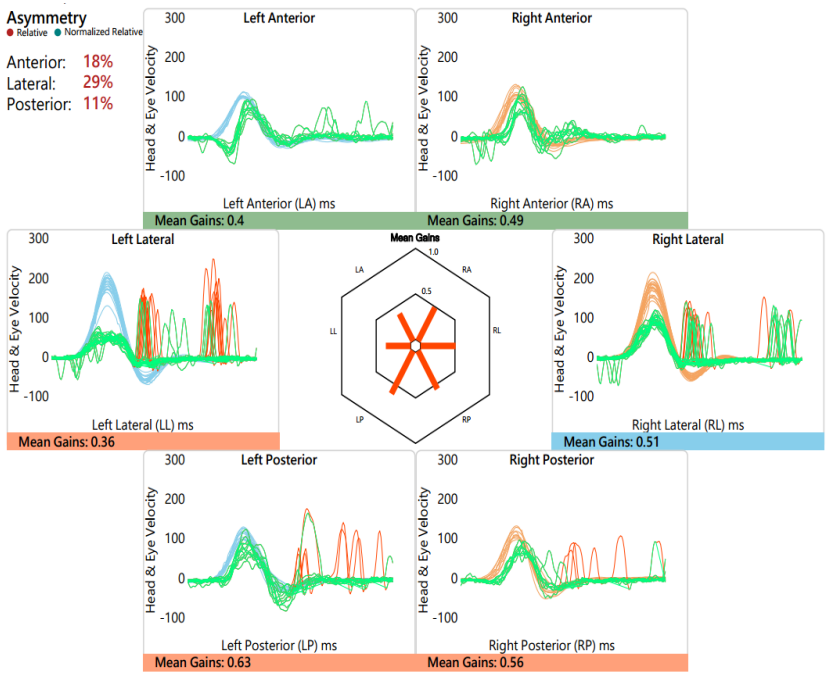
TABLE 1 Clinical and neuro-otologic findings in the patients with acute vestibular syndrome plus hearing loss.

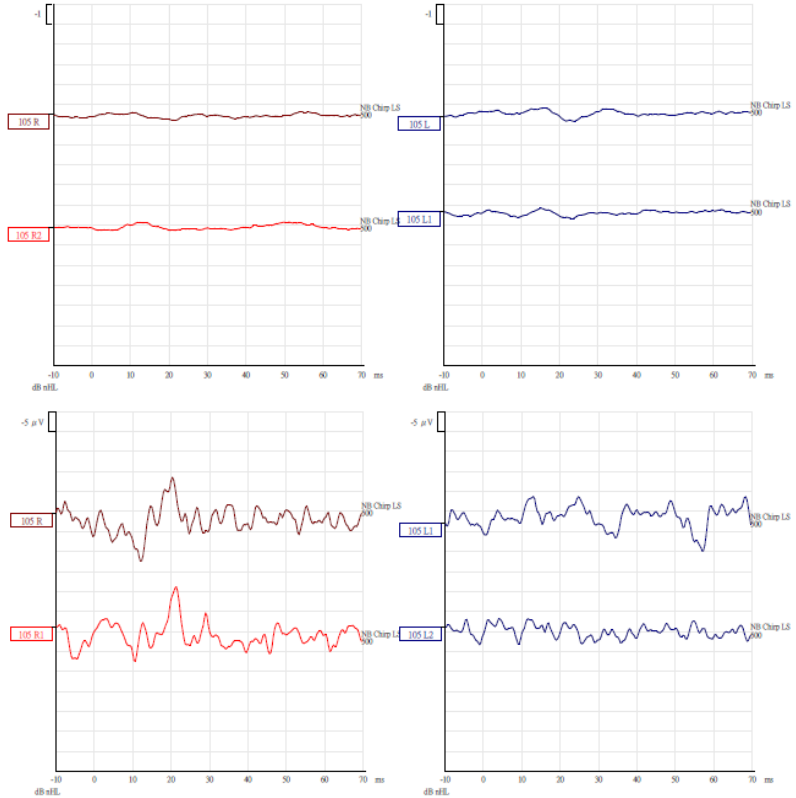
Pt	Age/Sex	HL	PTA (dB)	H-bHIT	H-vHIT		Fixation		Fixation block		Skew	SVV (°)	Other signs	MRI
					Ipsi-lesional	Contra-lesional	SN	GEN	SN	PN				
1	68/F	L	29	L	0.45	1.26	RBN	–	RBN	RBN	–	L, 6	–	–
2	48/M	L	65	L	0.64	1.11	RBN	+	RBN	RBN	+	L, 5	Diplopia	Left lateral pontine infarction
3	41/M	L	25	L	0.56	0.61	RBN	–	RBN	RBN	+	L, 5	Left facial palsy	Left lateral pontine demyelinating lesion
4	46/M	R	49	R	0.49	0.78	LBN	–	LBN	LBN	–	R, 10	–	Right lateral pontine infarction
5	77/M	R	90	R	0.68	0.68	LBN	–	LBN	LBN	–	R, 15	–	Right lateral pontine infarction
6	69/M	R	24	R	0.42	1.17	LBN	–	LBN	LBN	–	R, 1	Right facial palsy	Right lateral pontine infarction
7	62/M	R	90	Normal	1.14	0.92	–	–	–	RBN in right lying	–	L, 1	Right PC impaired at bHIT and vHIT (0.57)*	–
8	69/M	R	104	Normal	ND	ND	–	–	LBN	LBN	–	0	Right PC impaired at bHIT	–
9	60/F	R	79	Normal	1.14	1.15	–	–	LBN	LBN	–	R, 2	Right PC impaired at bHIT and vHIT (0.29)*	–
10	58/F	R	113	Normal	0.77	0.69	RBN	–	RBN	Geotropic	–	L, 3	–	–
11	62/M	L	115	Normal	0.88	0.88	LBN	–	LBN	Geotropic	–	ND	–	–
12	53/M	R	109	R	0.87	0.86	LBN	–	LBN	Apogeotropic	–	R, 3	–	–
13	59/F	L	79	L	0.82	0.98	RBN	–	RBN	Geotropic	–	L, 1	–	–

HL, hearing loss; PTA, pure-tone average (the average of pure tone thresholds at 500, 1,000, and 2,000 Hz in the lesion ear); H-bHIT, horizontal-canal bedside head impulse test; H-vHIT, horizontal-canal video head impulse test; SN, spontaneous nystagmus; GEN, gaze-evoked nystagmus; PN, positional nystagmus; SVV, subjective visual vertical; F, female; M, male; L, left; R, right; B, bilateral; RBN, right-beating nystagmus; LBN, left-beating nystagmus; PC, posterior semicircular canal; ND, not documented.

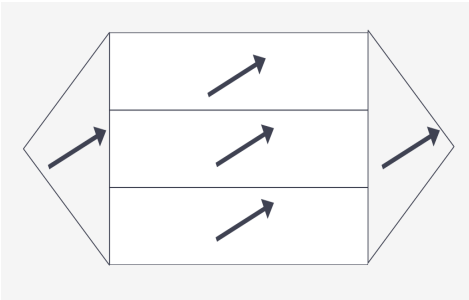
*The numbers in the parentheses are the VOR gains of vertical-canal vHIT.

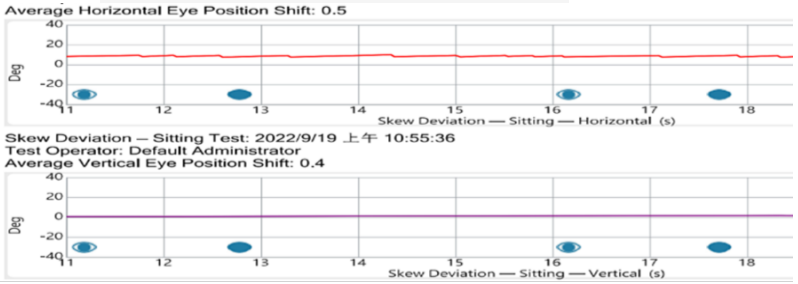

125
250
500
1K
2K
4K
8K
-10
0
10
20
30
40
50
60
70
80
90
100
110
120
Hearing Level (dB HL)
Frequency (Hz)
750
1.5K
3K
6K
12K

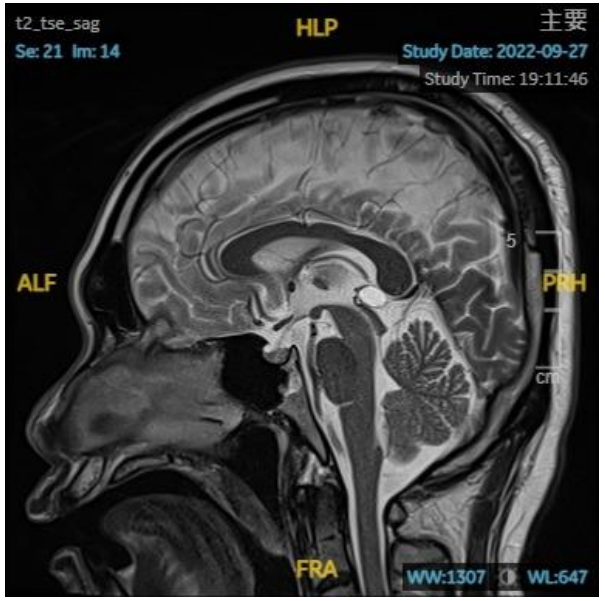

Asymmetry
Relative
Normalized Relative
Anterior: 18%
Lateral: 29%
Posterior: 11%
Left Anterior
Right Anterior
Left Lateral
Right Lateral
Left Posterior
Right Posterior
Head & Eye Velocity
Left Anterior (LA) ms
Right Anterior (RA) ms
Left Lateral (LL) ms
Right Lateral (RL) ms
Left Posterior (LP) ms
Right Posterior (RP) ms
Mean Gains: 0.4
Mean Gains: 0.49
Mean Gains: 0.36
Mean Gains: 0.51
Mean Gains: 0.63
Mean Gains: 0.56
Mean Gains
LA
RA
LL
RL
LP
RP

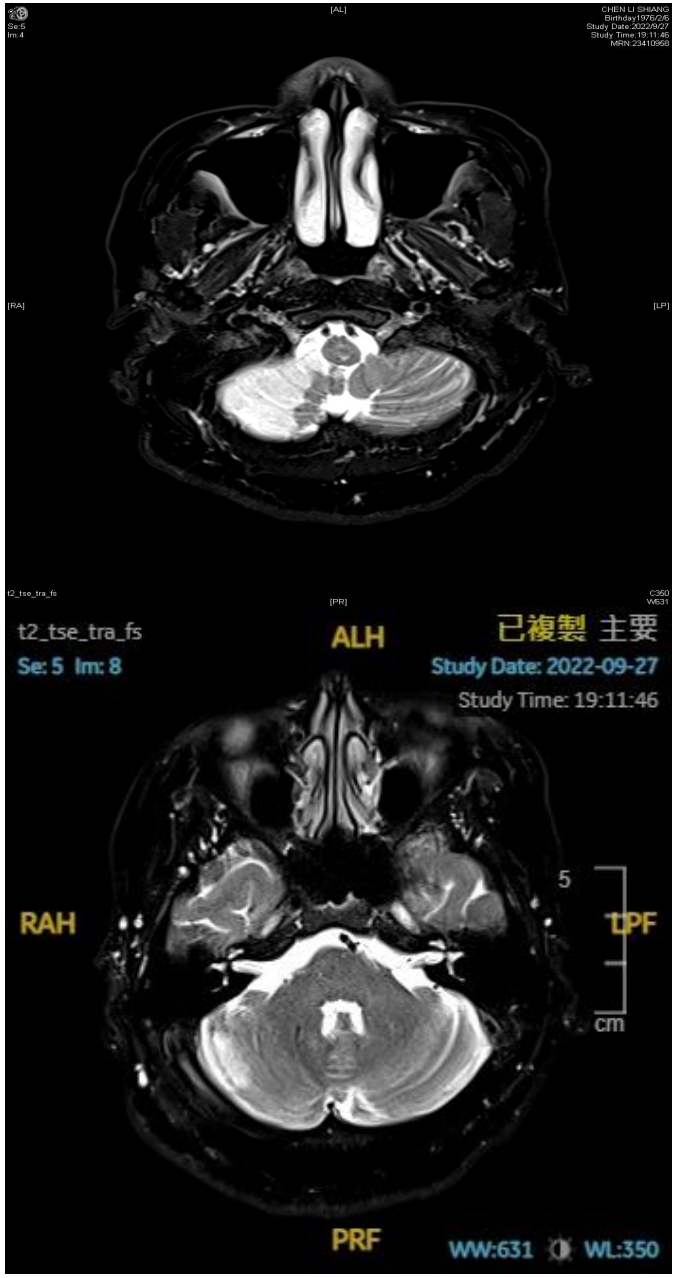

105 R
105 R2
105 L
105 L1
105 R1
105 L2
NB Chirp LS

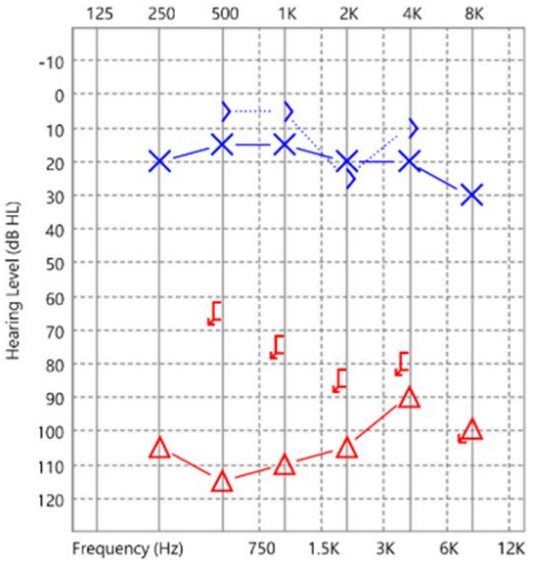

ARH
PLF

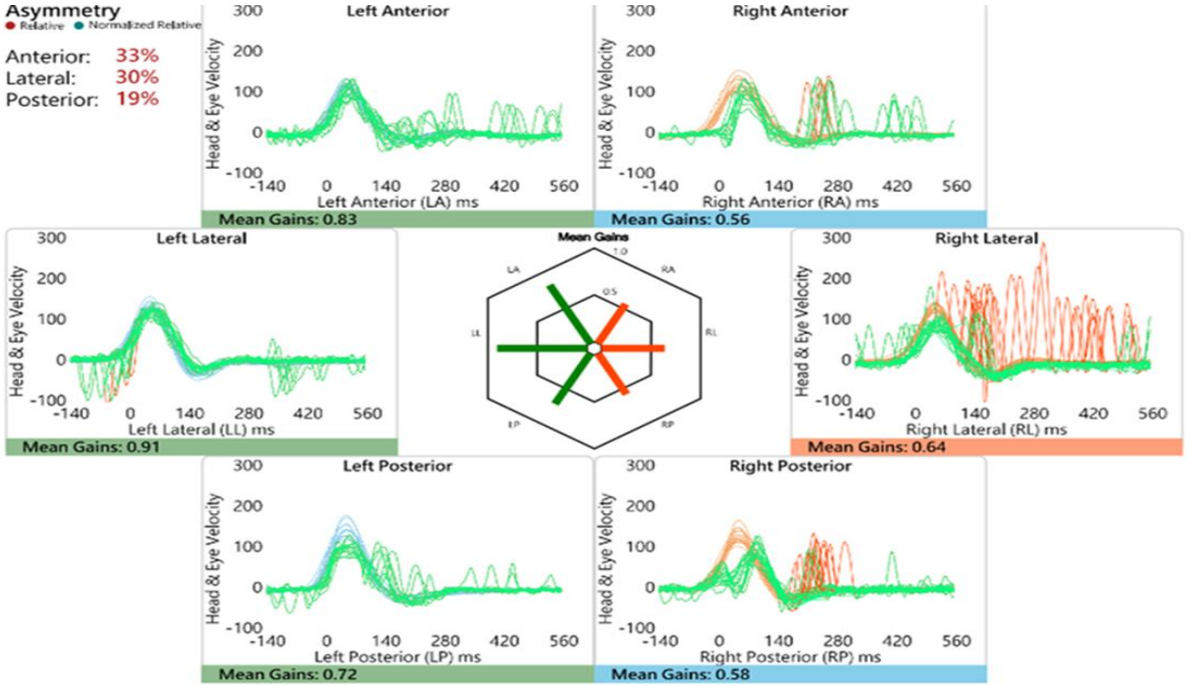



Average Horizontal Eye Position Shift: 0.5
Deg
Skew Deviation — Sitting — Horizontal (s)
Skew Deviation – Sitting Test: 2022/9/19 上午 10:55:36
Test Operator: Default Administrator
Average Vertical Eye Position Shift: 0.4
Deg
Skew Deviation — Sitting — Vertical (s)


t2_tse_sag
Se: 21 Im: 14
HLP
主要
Study Date: 2022-09-27
Study Time: 19:11:46
ALF
PRH
FRA
WW:1307
WL:647


t2_tse_tra_fs
Se: 5 Im: 8
ALH
已複製 主要
Study Date: 2022-09-27
Study Time: 19:11:46
RAH
LPF
PRF
WW:631
WL:350


Hearing Level (dB HL)
Frequency (Hz)


Asymmetry
Relative
Normalized Relative
Anterior: 33%
Lateral: 30%
Posterior: 19%
Left Anterior
Left Anterior (LA) ms
Mean Gains: 0.83
Right Anterior
Right Anterior (RA) ms
Mean Gains: 0.56
Left Lateral
Left Lateral (LL) ms
Mean Gains: 0.91
Mean Gains
Right Lateral
Right Lateral (RL) ms
Mean Gains: 0.64
Left Posterior
Left Posterior (LP) ms
Mean Gains: 0.72
Right Posterior
Right Posterior (RP) ms
Mean Gains: 0.58
Head & Eye Velocity

inner ear vascular territory

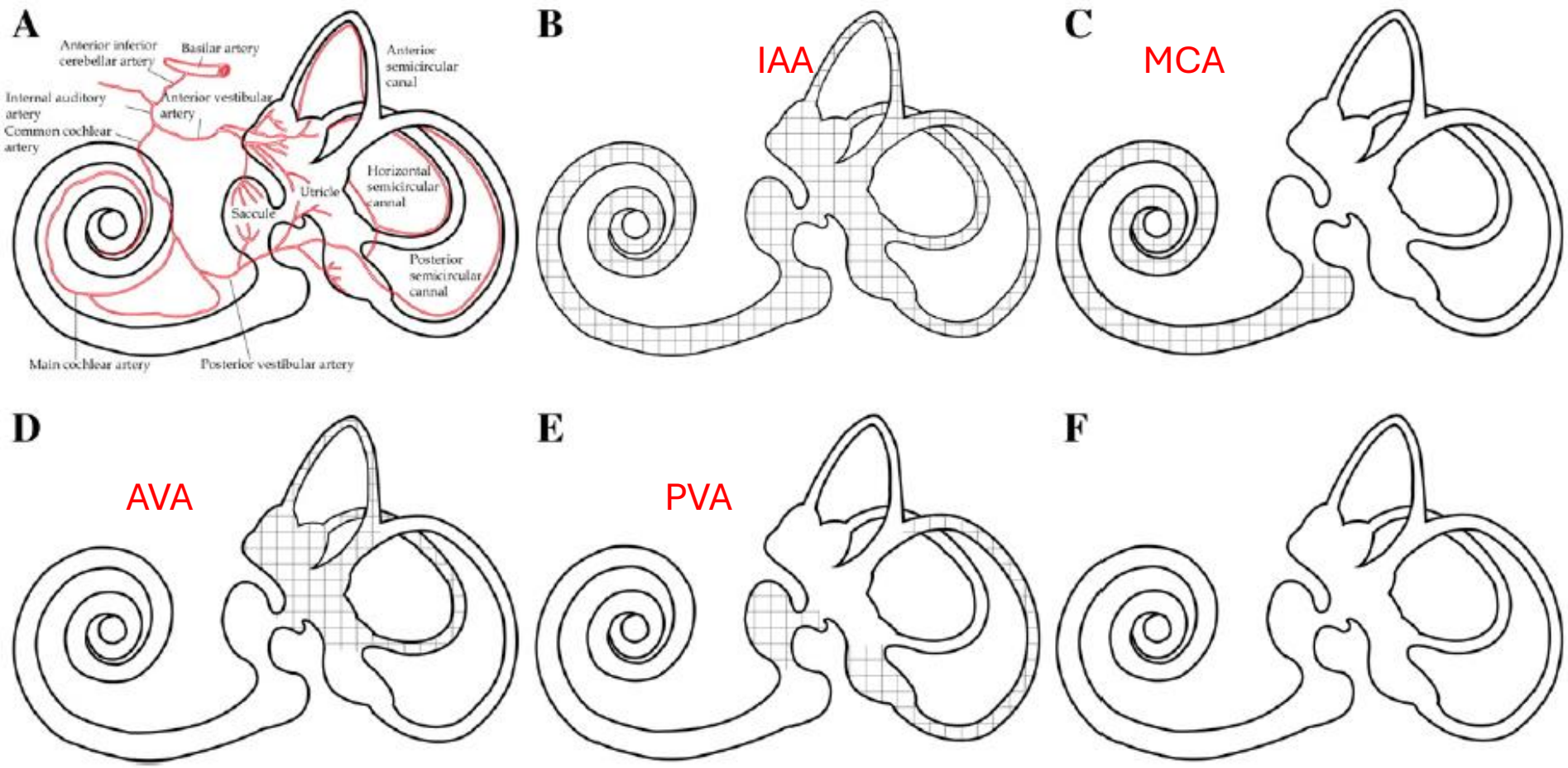
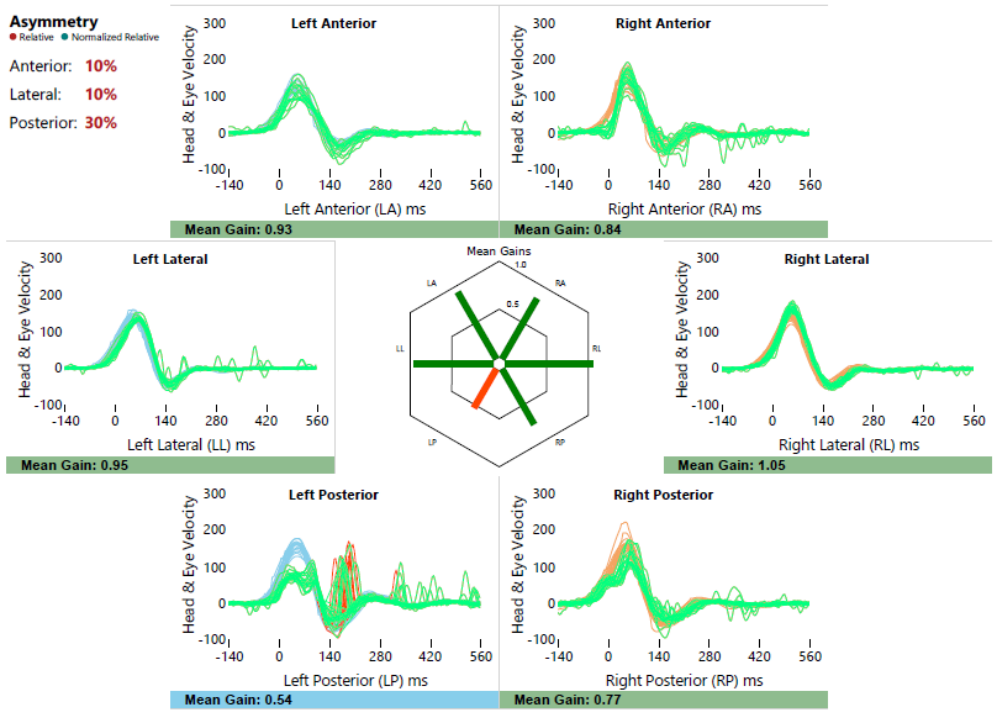
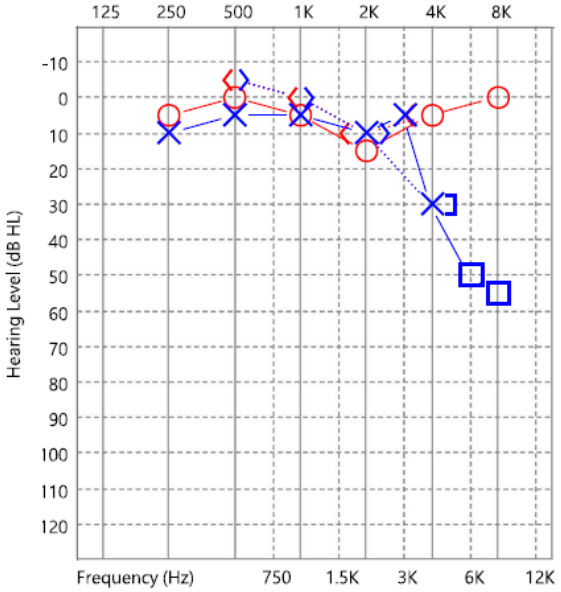
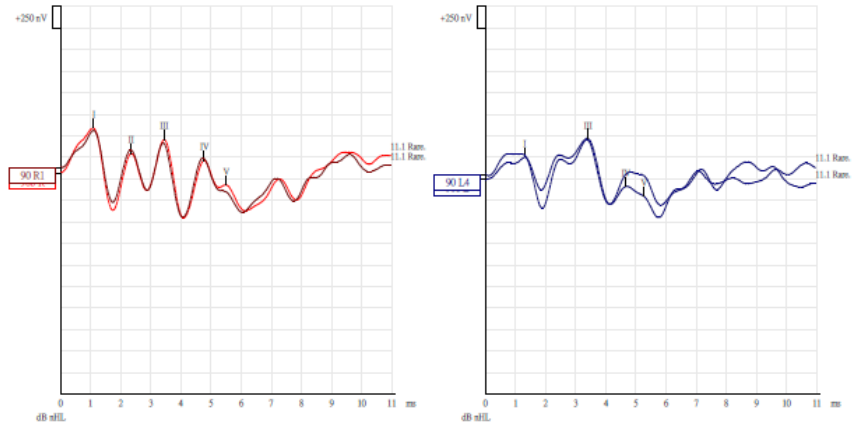
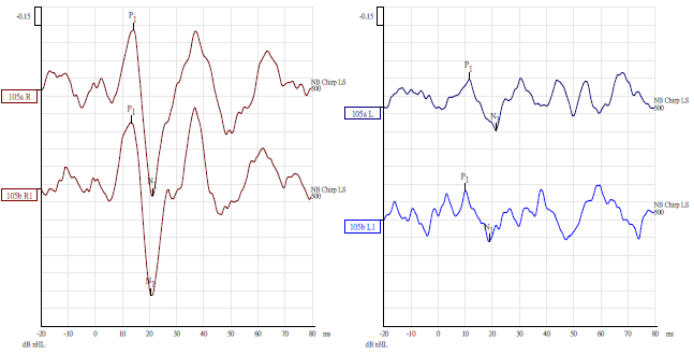


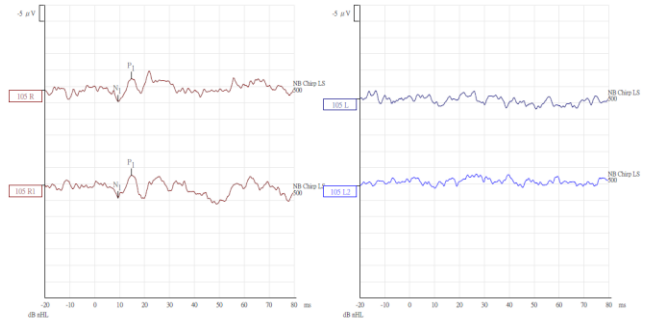
Fig. 3. Schematic diagram of the patterns of inner ear involvement in AICA territory infarction according to type of involved artery. A. The arterial supply to the inner ear. B. Internal auditory artery occlusion results in combined loss of cochlear and vestibular functions (i.e., SNHL, CP, and abnormal VEMP). C. Main cochlear artery occlusion causes selective loss of cochlear function (isolated SNHL). D. Anterior vestibular artery occlusion causes selective loss of superior vestibular function (isolated CP only). E. Posterior vestibular artery occlusion causes selective loss of inferior vestibular function (abnormal VEMP only). F. No involvement of the internal auditory artery results in normal cochlear and vestibular functions. Hatched region indicates area of lesion. SNHL: sensorineural hearing loss, CP: canal paresis, VEMP: vestibular evoked myogenic potentials.

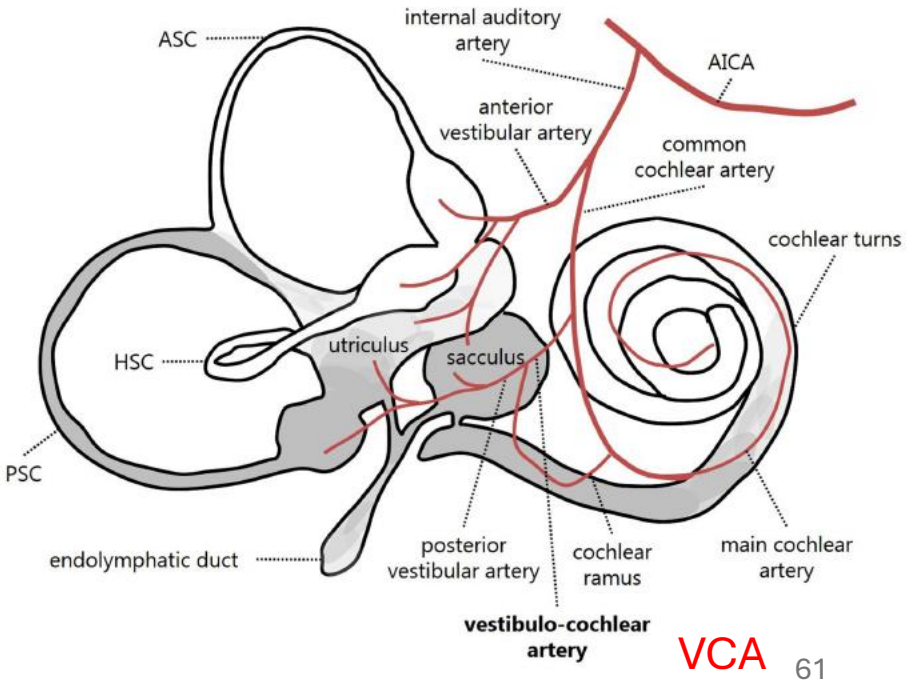

Asymmetry
Relative
Normalized Relative
Anterior: 10%
Lateral: 10%
Posterior: 30%
Left Anterior
Right Anterior
Left Lateral
Right Lateral
Left Posterior
Right Posterior
Head & Eye Velocity
Left Anterior (LA) ms
Right Anterior (RA) ms
Left Lateral (LL) ms
Right Lateral (RL) ms
Left Posterior (LP) ms
Right Posterior (RP) ms
Mean Gain: 0.93
Mean Gain: 0.84
Mean Gain: 0.95
Mean Gain: 1.05
Mean Gain: 0.54
Mean Gain: 0.77
Mean Gains

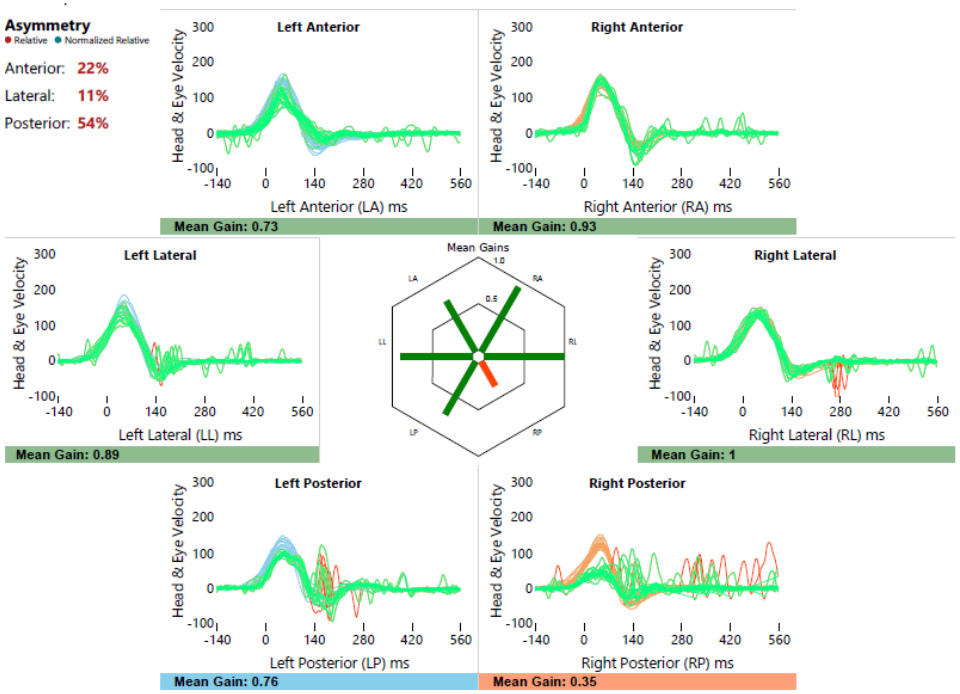

125
250
500
1K
2K
4K
8K
Hearing Level (dB HL)
Frequency (Hz)
750
1.5K
3K
6K
12K

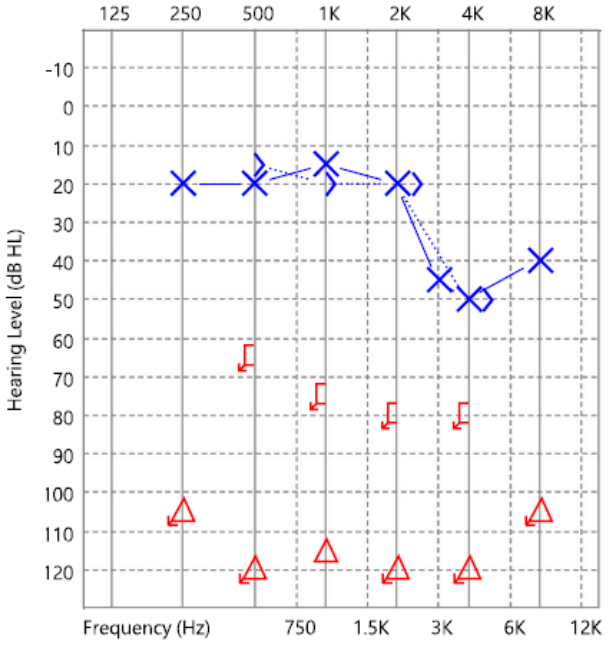


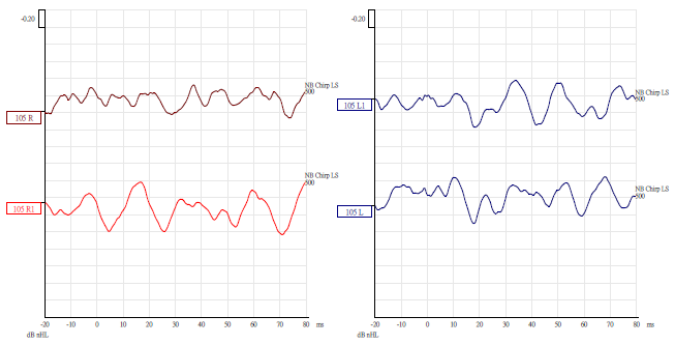


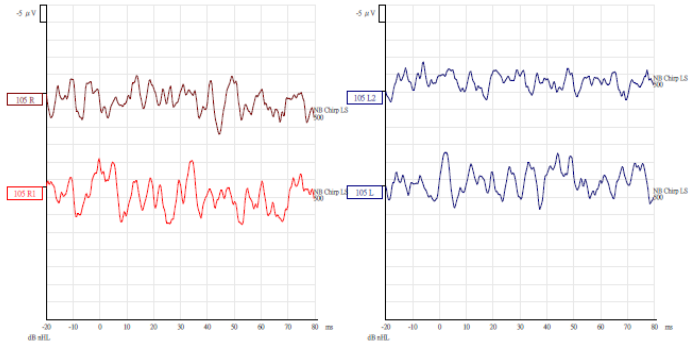


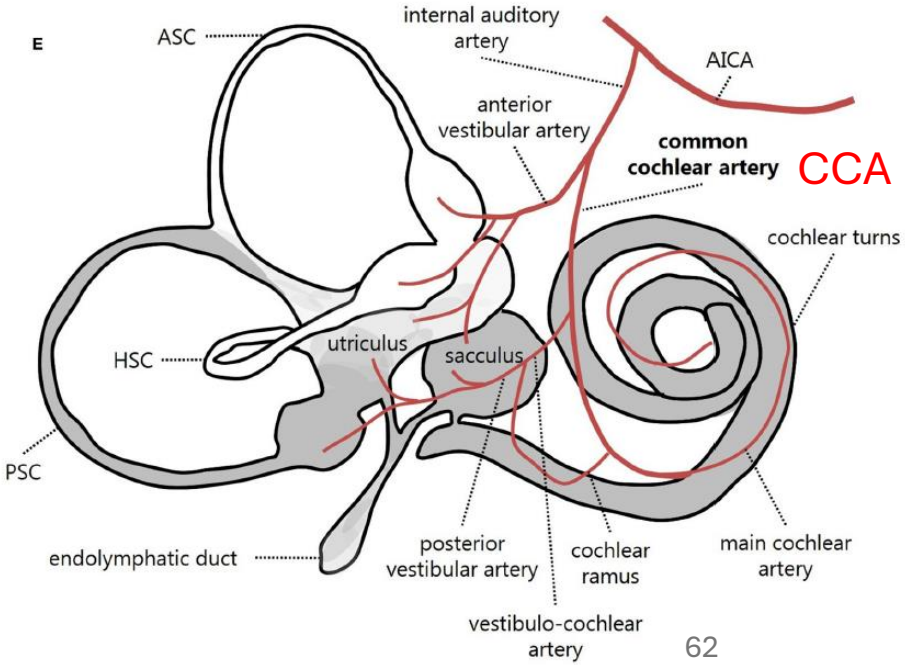

ASC
internal auditory artery
AICA
anterior vestibular artery
common cochlear artery
cochlear turns
HSC
utriculus
sacculus
PSC
endolymphatic duct
posterior vestibular artery
cochlear ramus
main cochlear artery
vestibulo-cochlear artery

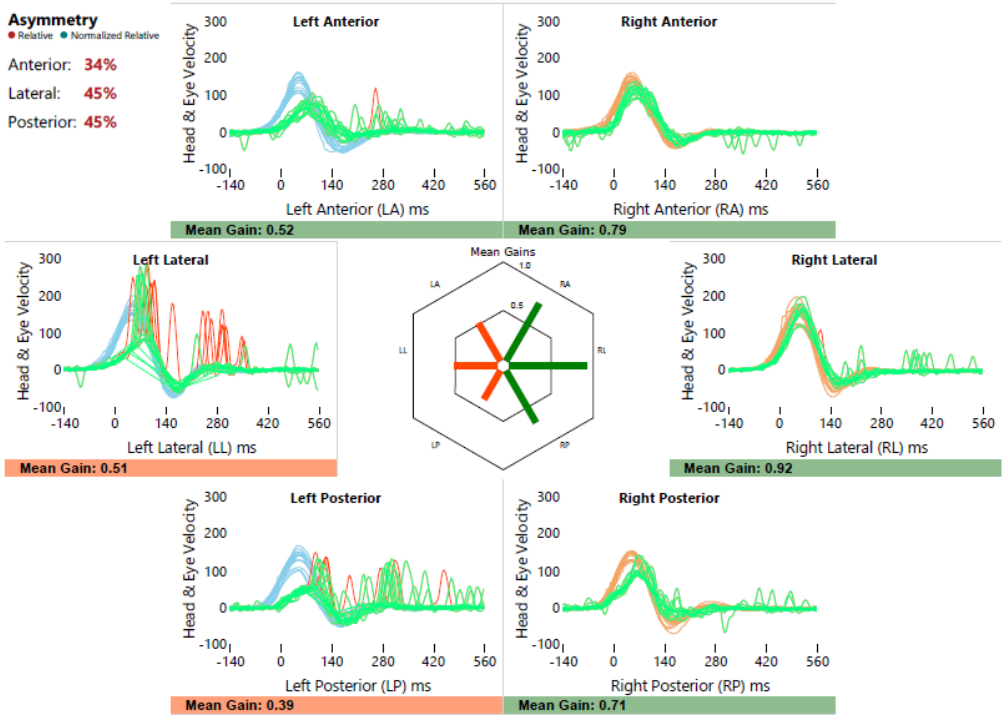

Asymmetry
Relative
Normalized Relative
Anterior: 22%
Lateral: 11%
Posterior: 54%
Left Anterior
Right Anterior
Left Lateral
Right Lateral
Left Posterior
Right Posterior
Head & Eye Velocity
Left Anterior (LA) ms
Right Anterior (RA) ms
Left Lateral (LL) ms
Right Lateral (RL) ms
Left Posterior (LP) ms
Right Posterior (RP) ms
Mean Gains
Mean Gain: 0.73
Mean Gain: 0.93
Mean Gain: 0.89
Mean Gain: 1
Mean Gain: 0.76
Mean Gain: 0.35

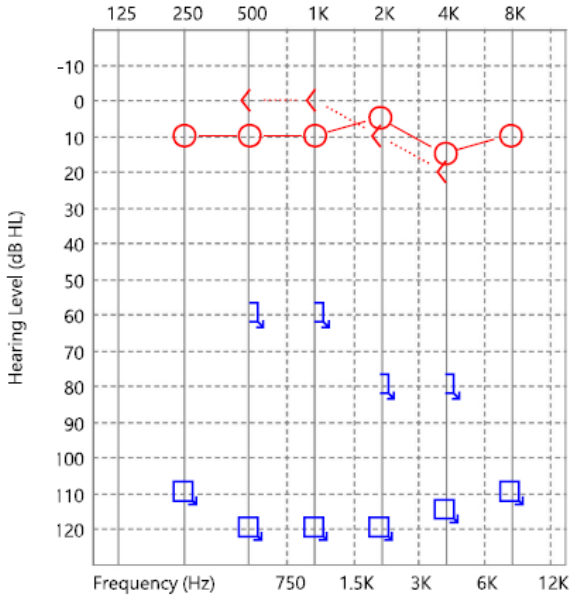

125
250
500
1K
2K
4K
8K
Hearing Level (dB HL)
Frequency (Hz)
750
1.5K
3K
6K
12K





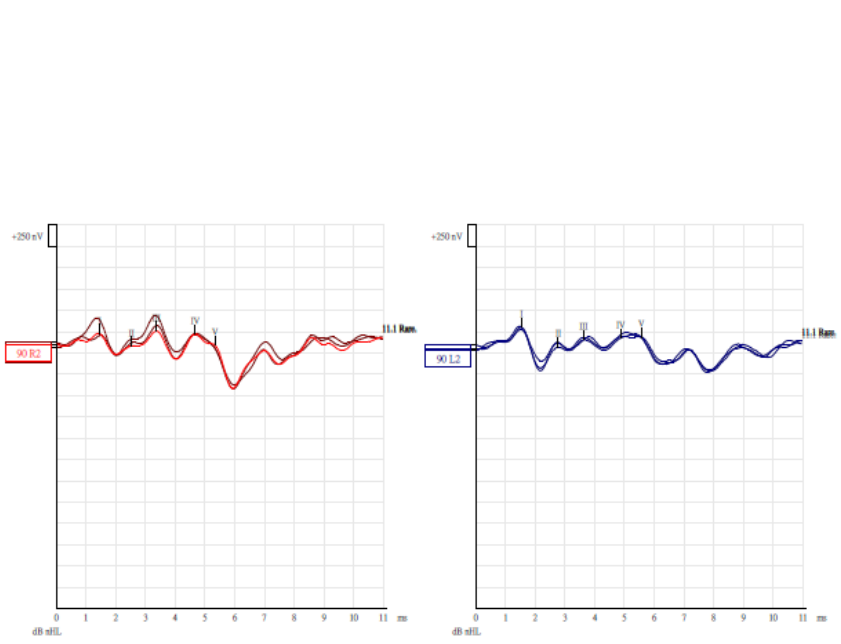

E
ASC
internal auditory artery
AICA
anterior vestibular artery
common cochlear artery
CCA
cochlear turns
HSC
utriculus
sacculus
PSC
endolymphatic duct
posterior vestibular artery
cochlear ramus
main cochlear artery
vestibulo-cochlear artery

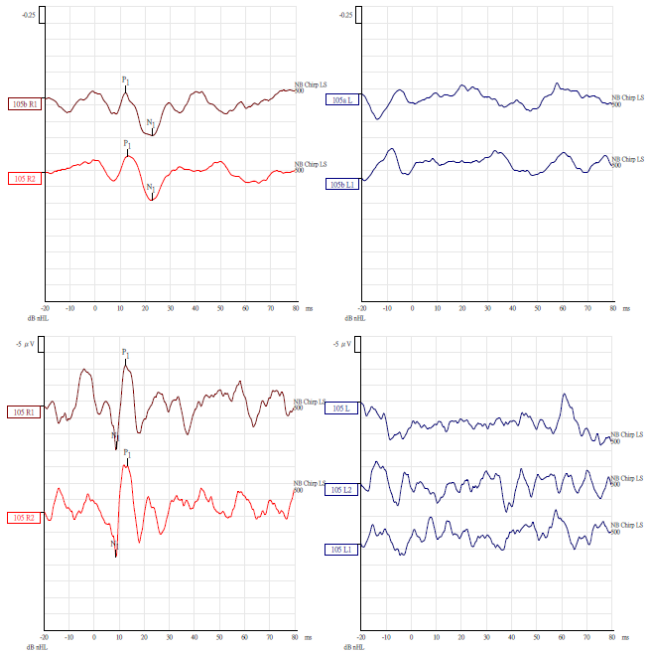

Asymmetry
Relative
Normalized Relative
Anterior: 34%
Lateral: 45%
Posterior: 45%
Left Anterior
Right Anterior
Left Lateral
Right Lateral
Left Posterior
Right Posterior
Head & Eye Velocity
Left Anterior (LA) ms
Right Anterior (RA) ms
Left Lateral (LL) ms
Right Lateral (RL) ms
Left Posterior (LP) ms
Right Posterior (RP) ms
Mean Gains
Mean Gain: 0.52
Mean Gain: 0.79
Mean Gain: 0.51
Mean Gain: 0.92
Mean Gain: 0.39
Mean Gain: 0.71

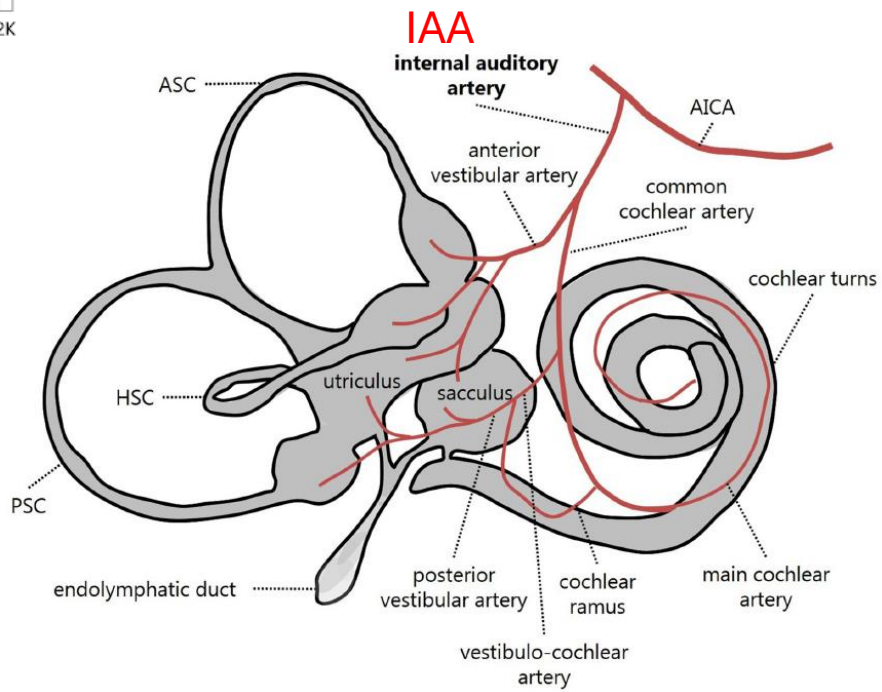

125
250
500
1K
2K
4K
8K
Hearing Level (dB HL)
Frequency (Hz)
750
1.5K
3K
6K
12K

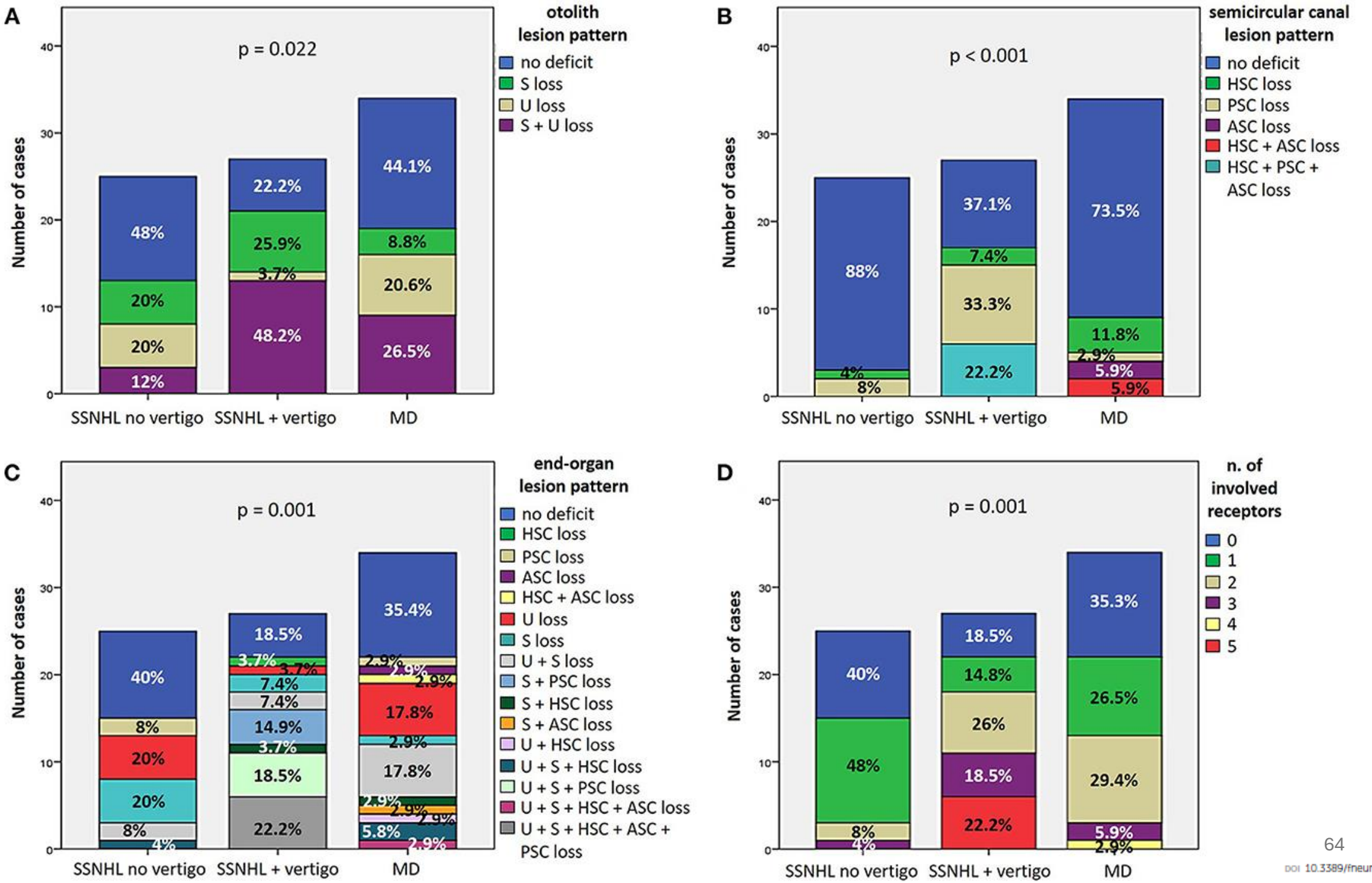
E


ARH
RPH
PLF
LAF
t2_tse_tra_fs
Se: 5 , Im: 7
Institution: Changhua Christian hospital
MRI of Cerebello-pontine angle with contrast
Study Date: 2024-04-14
Study Time: 17:20:05
400 x 400
ST: 4 mm SP: 5.2 mm
WW:914 - WL:400
1.32






IAA
internal auditory artery
ASC
AICA
anterior vestibular artery
common cochlear artery
cochlear turns
HSC
utriculus
sacculus
PSC
endolymphatic duct
posterior vestibular artery
cochlear ramus
main cochlear artery
vestibulo-cochlear artery



DOI 10.3389/fneur.2023.1127008

s-AVS in ENT Clinics – HINT Plus

- No hearing loss
  - HINT
    - Normal HSC vHIT
- Acute hearing loss
  - HINT
    - Abnormal vHIT in ipsilateral HSC
    - Abnormal vHIT in ipsilateral PSC–check vascular risk factors
    - Normal ipsilateral vHIT –check VEMPs and/or vascular risk factors

Response: Proposed Diagnostic Criteria for Definite Isolated Otolith Dysfunction

Myung-Whan Suh[1] and Toshihisa Murofushi[2]

Definite Isolated Otolith Dysfunction

Table 1. Proposed diagnostic criteria for isolated otolith dysfunction (iOD)

Definite iOD

A. Laboratory findings that indicate OD but normal semicircular canal function.
1. OD proven using the cVEMP and/or oVEMP tests.
2. Normal caloric and vHIT test results (for all vertical and horizontal canals).

B. Symptoms that indicate OD.
- Non-spinning, translation, tilt, floating, or flipping-over.

C. Cannot be explained by another disease or disorder.

Probable iOD

Laboratory-based probable iOD

A. Laboratory findings that indicate OD but normal semicircular canal function.
1. OD proven by cVEMP and/or oVEMP and/or SVV testing.
2. Normal caloric and vHIT test results.

B. Cannot be explained by another disease or disorder.

Symptom-based probable iOD

A. Symptoms that indicate OD.
- Non-spinning, translation, tilt, floating, or flipping-over.

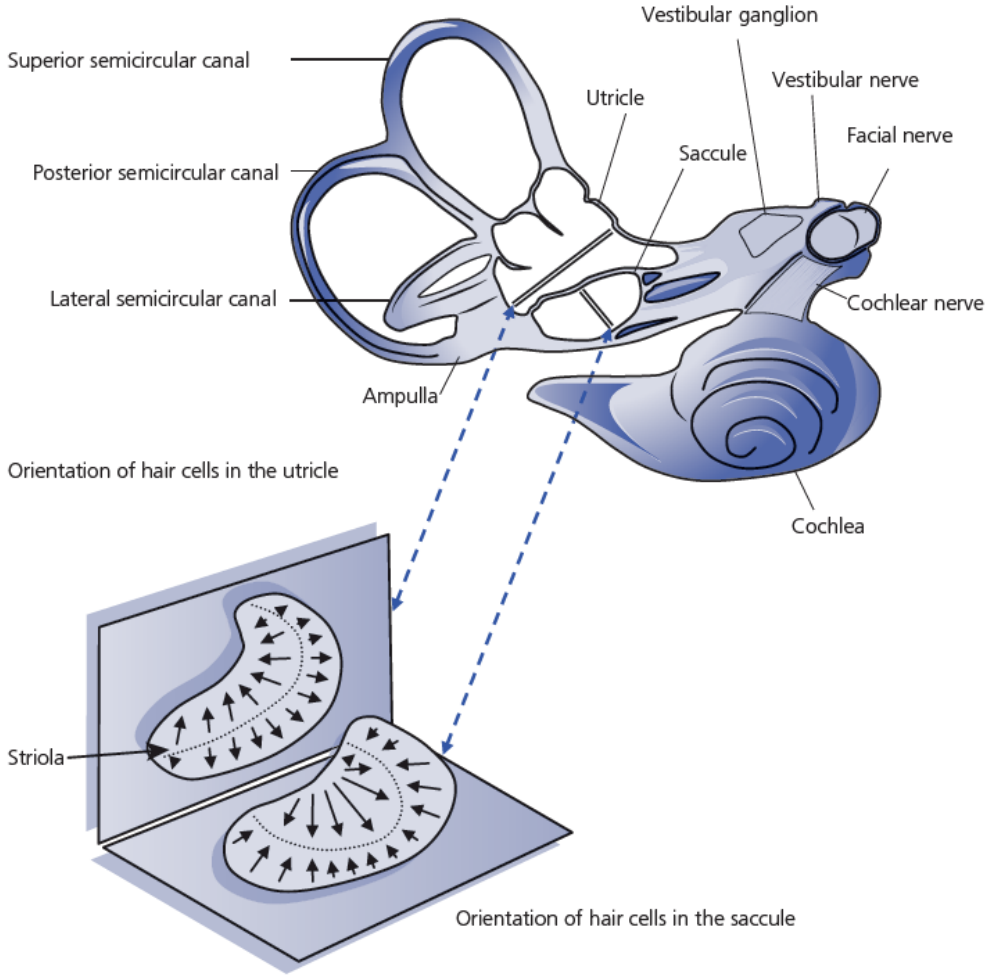
B. Cannot be explained by another disease or disorder.

Additional descriptions (**not mandatory**)

Idiopathic definite/probable iOD.
- When the etiology of iOD cannot be identified.

Secondary definite/probable iOD.
- When a causal relationship with another disorder is identified.

cVEMP: cervical vestibular myogenic potential, oVEMP: ocular vestibular myogenic potential, vHIT: video head impulse test, SVV: subjective visual vertical



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Description: Second edition. | Boca Raton, FL : CRC Press, 2024. | Includes bibliographical references and index.

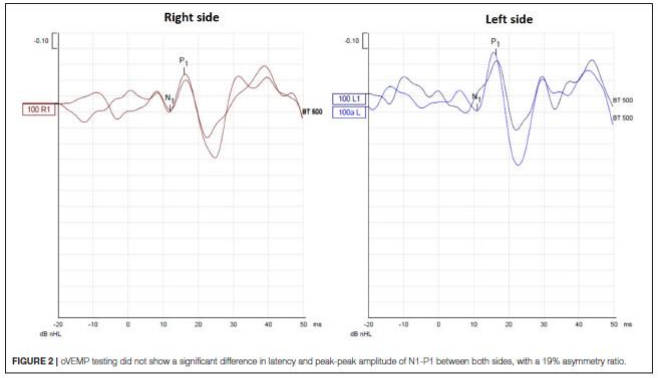


FIGURE 2 | cVEMP testing did not show a significant difference in latency and peak-peak amplitude of N1-P1 between both sides, with a 19% asymmetry ratio.

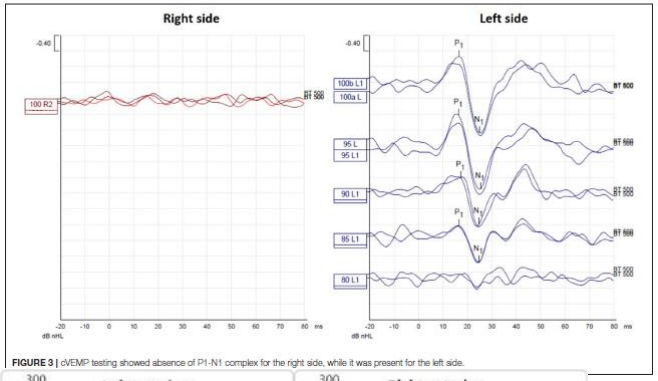
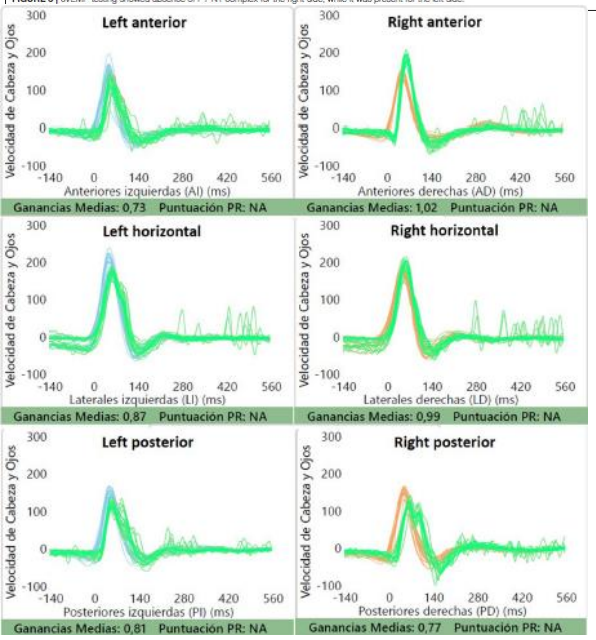
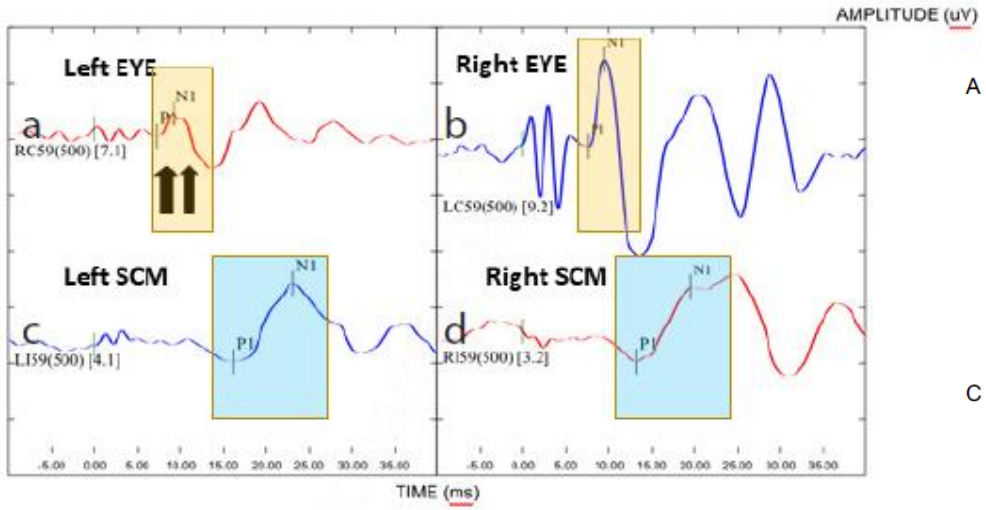


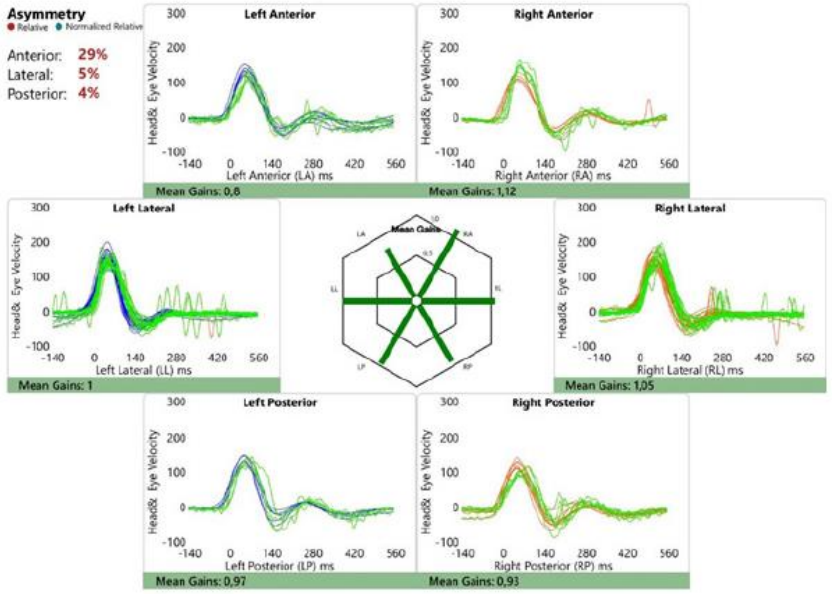
FIGURE 3 | cVEMP testing showed absence of P1-N1 complex for the right side, while it was present for the left side.



Manzari et al. Selective Utricular Dysfunction

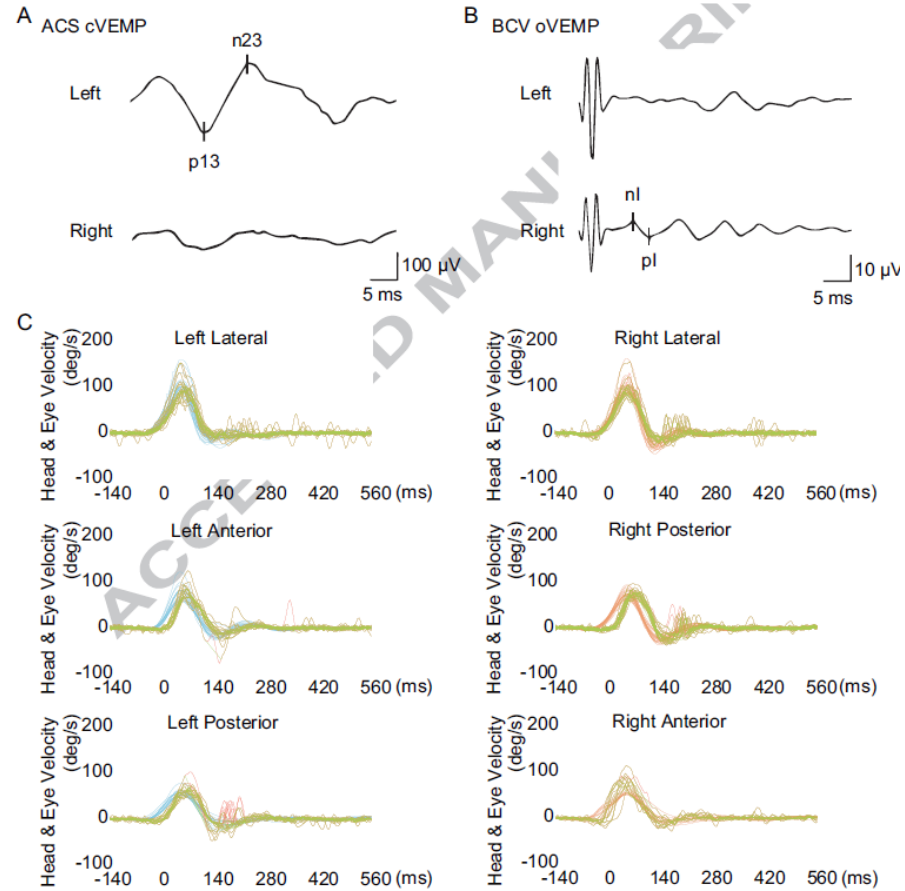
BCV VEMPs





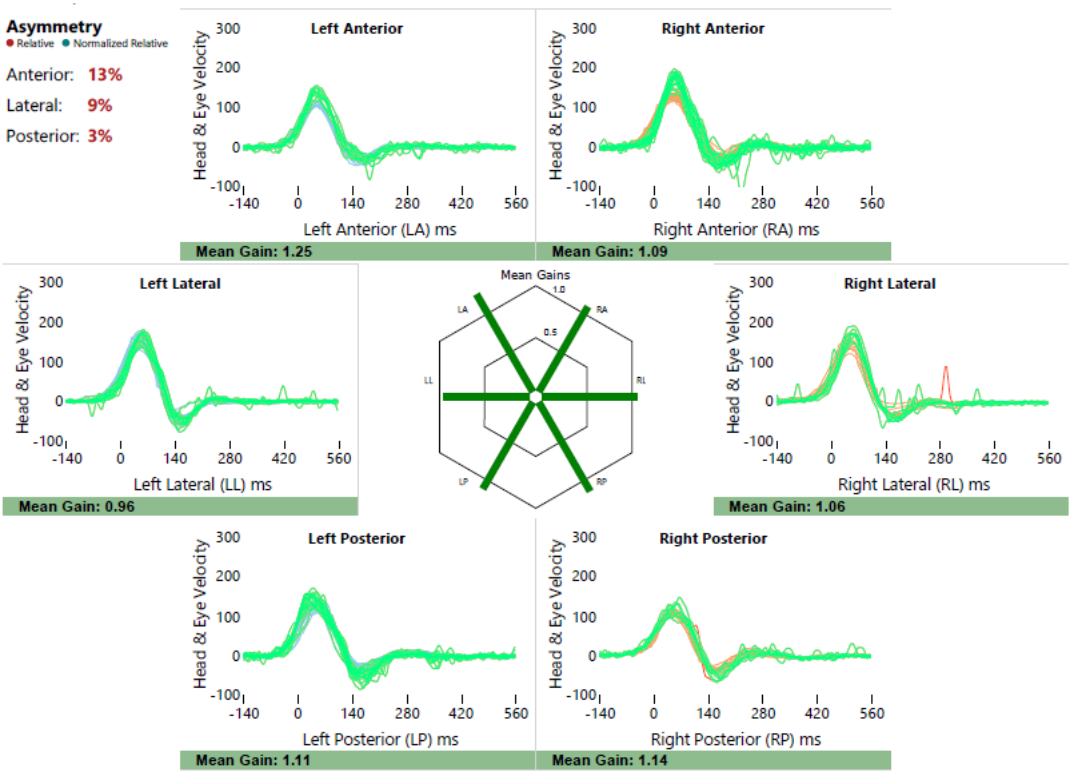
Case Report: Isolated Idiopathic Saccular Dysfunction

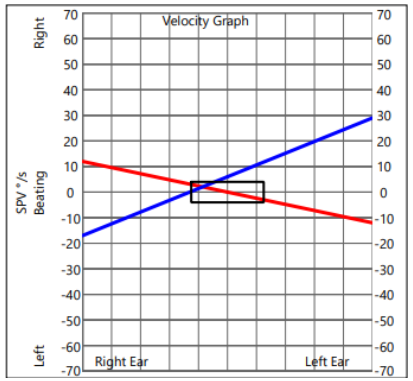
Sofia Waissbluth and Javier Oyarzún*

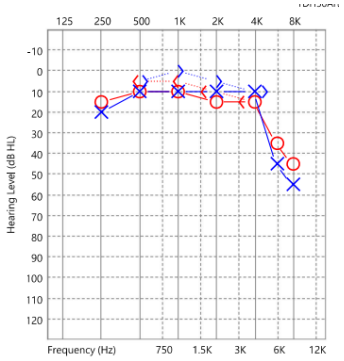


Clinical features of otolith organ-specific vestibular dysfunction

Chisato Fujimoto, Sayaka Suzuki, Makoto Kinoshita, Naoya Egami, Keiko Sugasawa, Shinichi Iwasaki

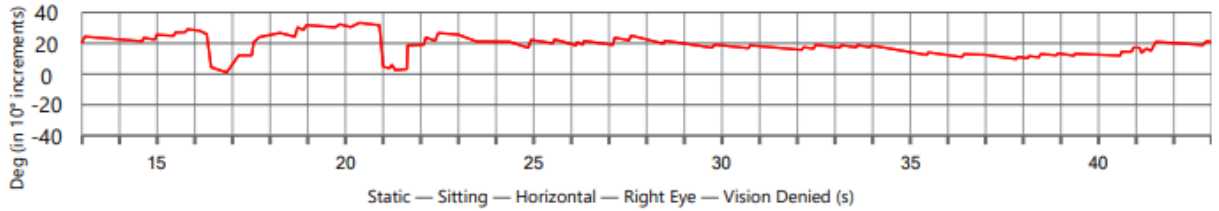


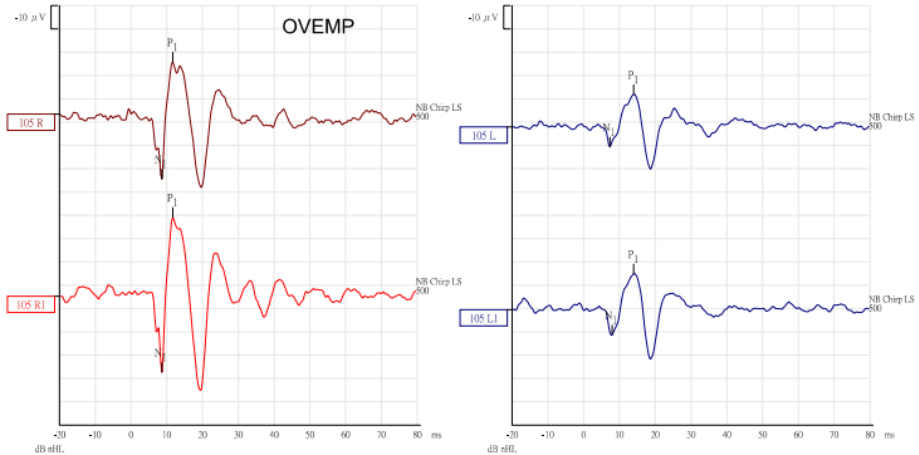


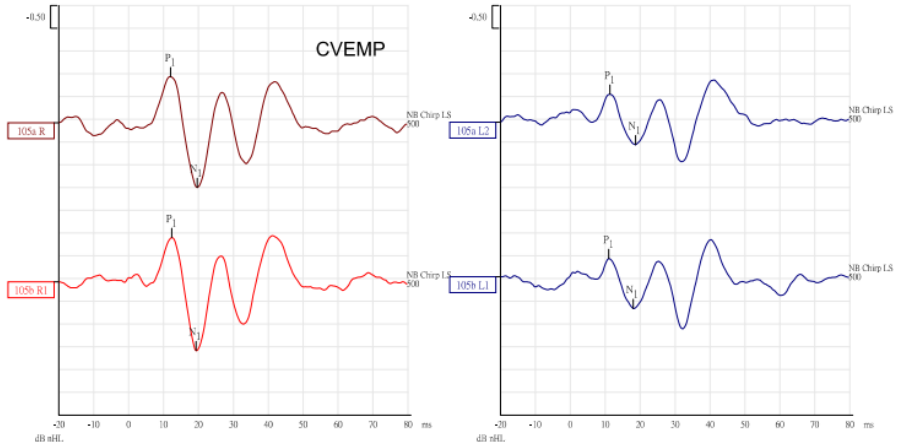


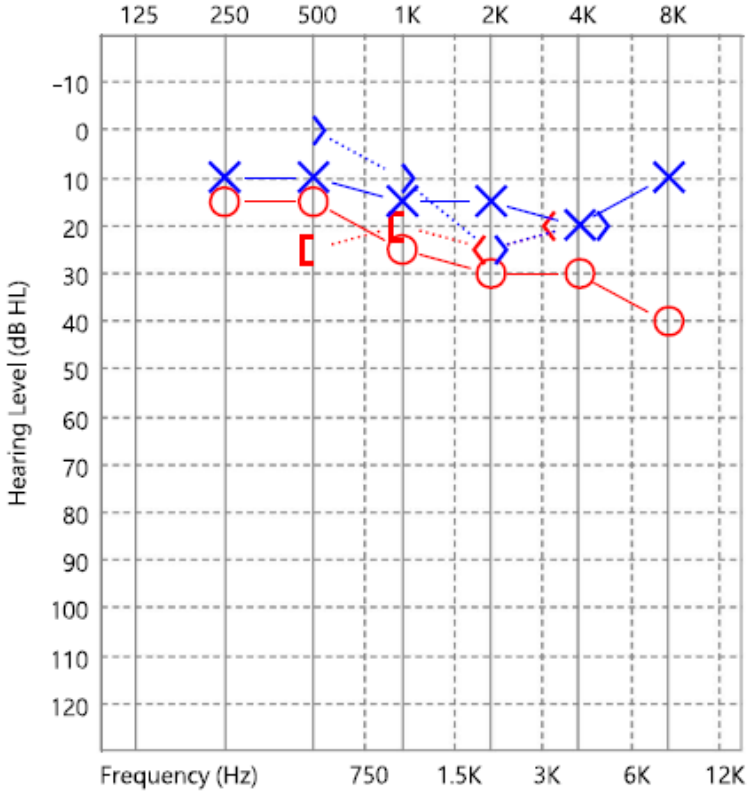
Positional - Static

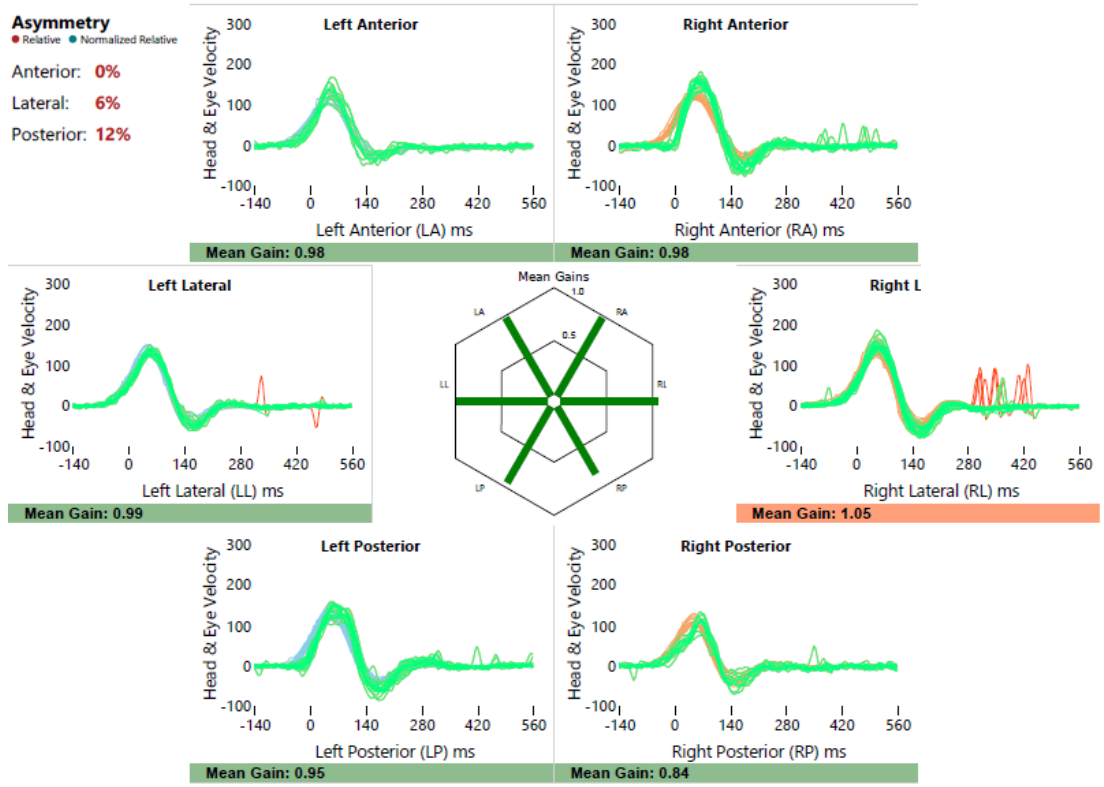
Static — Sitting Test: 6/3/2024 11:54:01 AM
Test Operator: Default Administrator
Right Eye
SPV Horizontal: Peak -6 °/s at 24.2 s

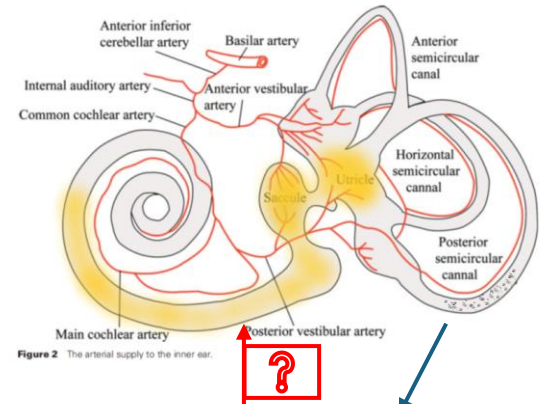


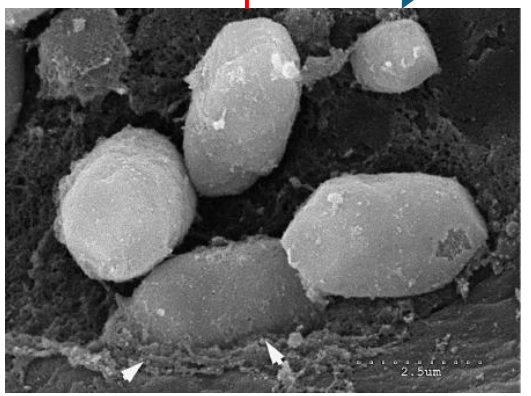




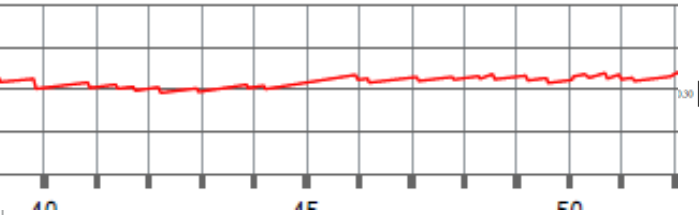


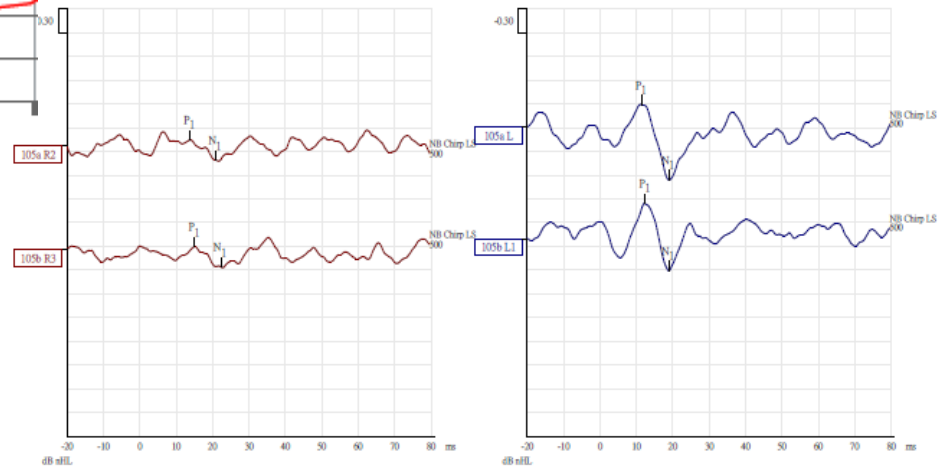


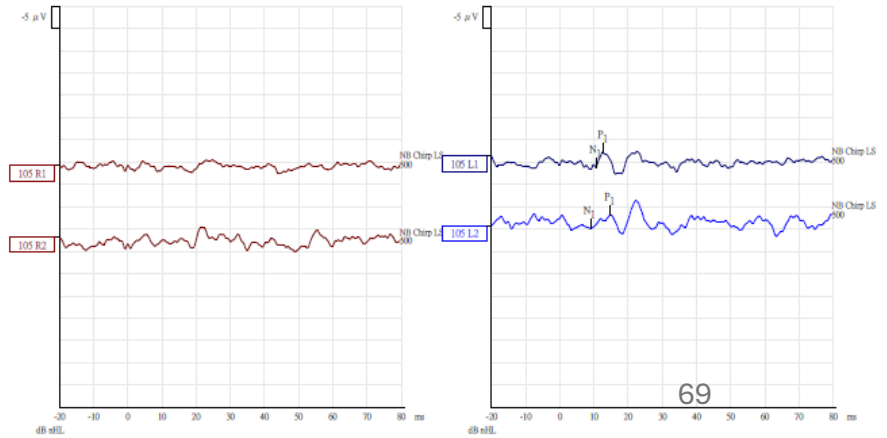




Laryngoscope. 2017 March







Comments

O. A. E. (Otoacoustic Emission) Report
RE: Normal OHC function
LE: Normal OHC function

s-AVS in ENT Clinics – HINT Plus

- No hearing loss
  - HINT
    - Normal HSC vHIT
    - Optional PSC vHIT for IVN
- Acute hearing loss
  - HINT
    - Abnormal vHIT in ipsilateral HSC
    - Abnormal vHIT in ipsilateral PSC–check vascular risk factors
    - Normal ipsilateral vHIT –check VEMPs and/or vascular risk factors
- Subsequent VEMPs accordingly

s-EVS : MD vs VM

Diagnostic criteria : MD vs VM

Tab. I. Comparison of AAO 1995 diagnostic criteria (10) and joined Equilibrium Committee AAO-HNS, Japan Society for Equilibrium Research, EAONO, Korean Balance Society and Barany Society 2016 criteria [11].

AAO 1995	EQUILIBRIUM COMMITTEE AAO-HNS, JAPAN SOCIETY FOR EQUILIBRIUM RESEARCH, EAONO, KOREAN BALANCE SOCIETY, BARANY SOCIETY 2015
CLINICALLY DEFINED MD	**DEFINED MD**
At lest 2 vertigo spells (lasting more than 20 minutes)	At least two vertigo spells (20 min – 12 h)
Hearing loss (0.5, 1, 2, 3 kHz) documented at least once	Hearing loss at low or medium tones (at least 30 dB, < 2 kHz, at least 35 dB, < 2 kHz) documented at least once
Tinnitus or feeling of fullness in the affected ear	Fluctuating symptoms (hearing loss, tinnitus, ear fullness)
Other causes excluded	Other causes excluded
PROBABLE MD	**PROBABLE MD**
One vertigo spell	At least two vertigo or dizzy spells (20 min – 24 h)
Hearing loss documented at least once	
Tinnitus or feeling of fullness in the affected ear	Fluctuating symptoms (hearing loss, tinnitus, ear fullness)
	Other causes excluded
	DELAYED MD
	auditory symptoms may precede vertigo spells by months or years
	vertigo spells can precede hearing loss by weeks or months

1. Vestibular migraine

A. At least 5 episodes with vestibular symptoms[1] of moderate or severe intensity[2], lasting 5 min to 72 hours[3]
B. Current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD-3)[4]
C. One or more migraine features with at least 50% of the vestibular episodes[5]:
   – headache with at least two of the following characteristics: one sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity
   – photophobia and phonophobia[6],
   – visual aura[7]
D. Not better accounted for by another vestibular or ICHD diagnosis[8]

2. Probable vestibular migraine

A. At least 5 episodes with vestibular symptoms[1] of moderate or severe intensity[2], lasting 5 min to 72 hours[3]
B. Only one of the criteria B and C for vestibular migraine is fulfilled (migraine history *or* migraine features during the episode)
C. Not better accounted for by another vestibular or ICHD diagnosis[8]

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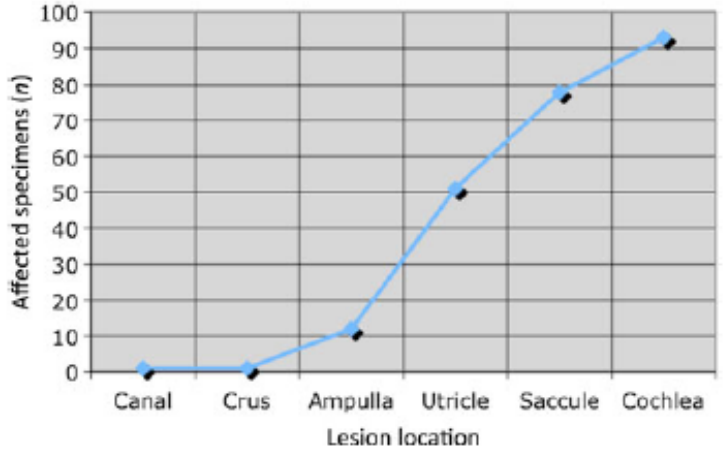
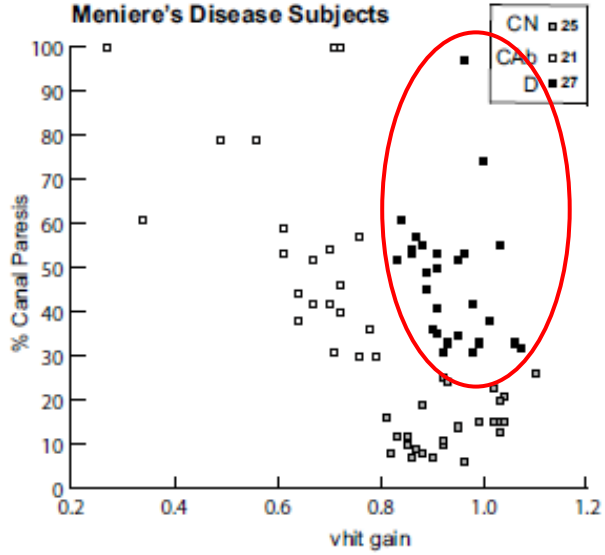
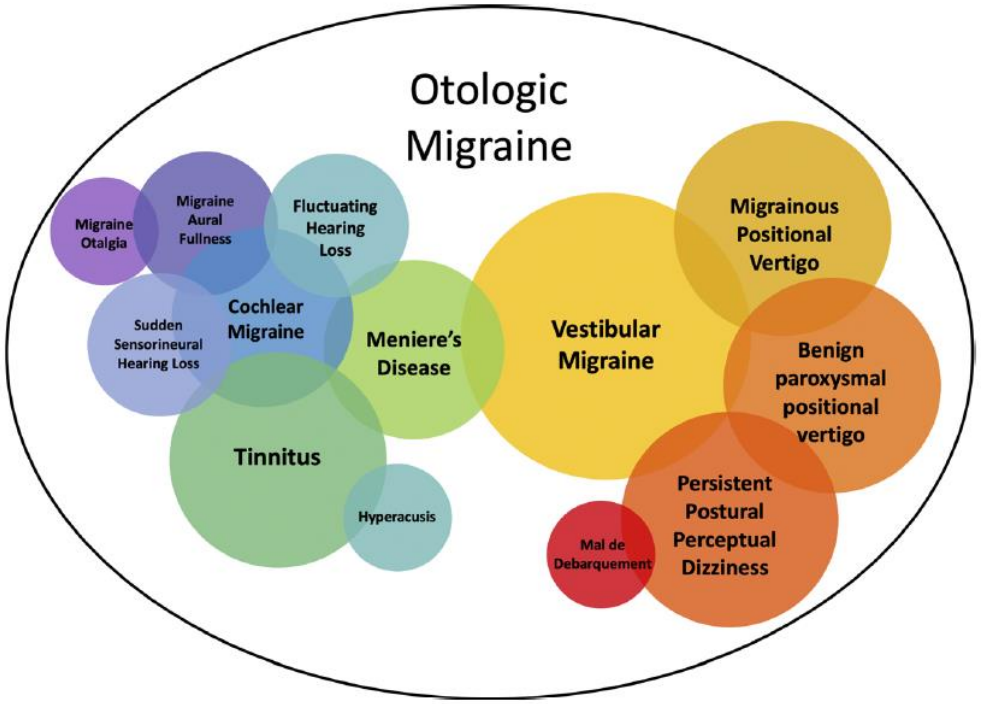
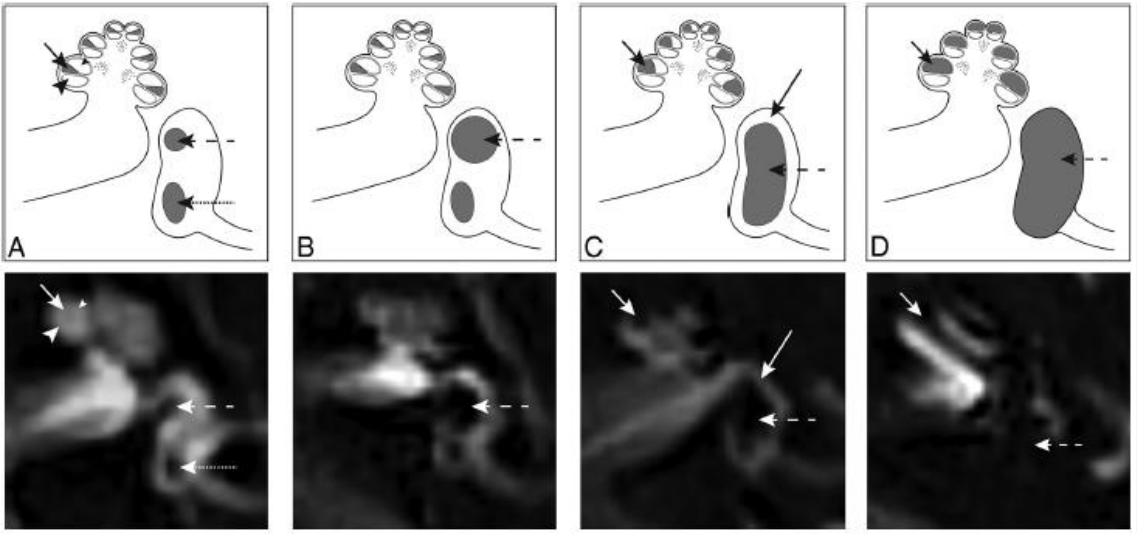
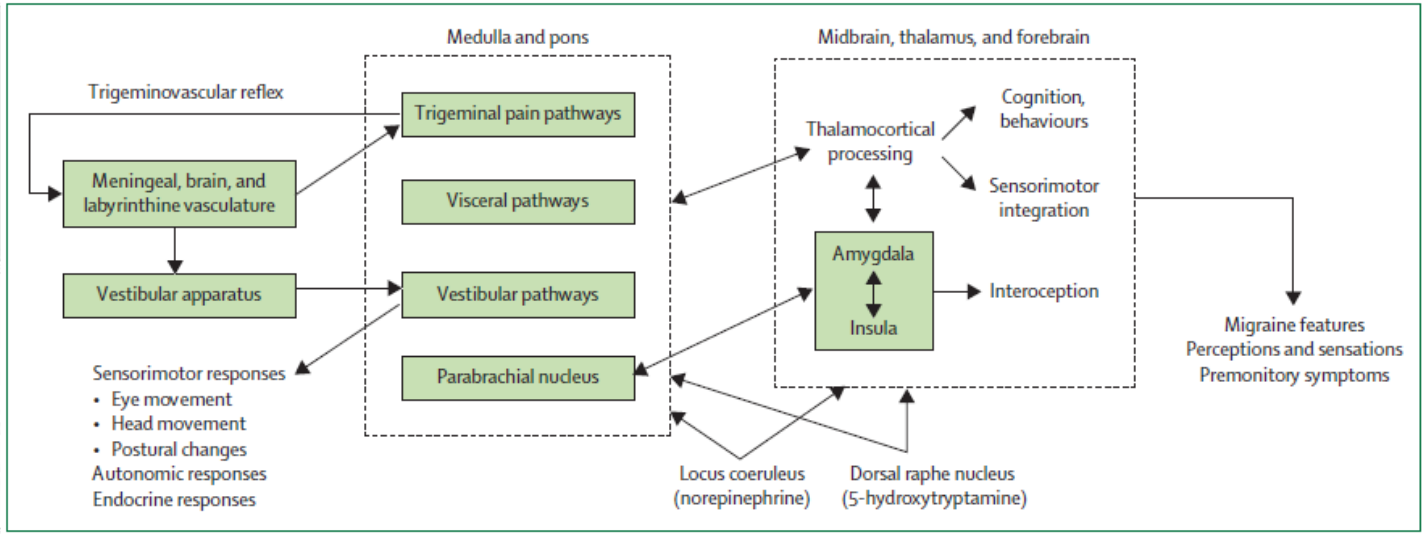
Suspected Peripheral (MD) vs central or mixed (VM)





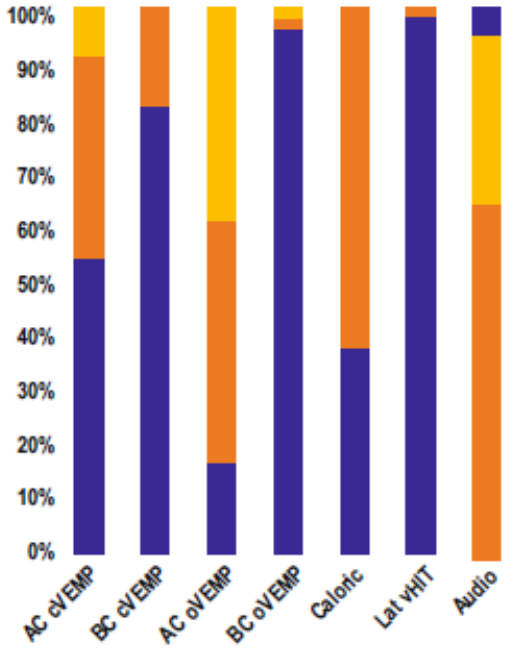






The Journal of Laryngology & Otology (2014), 128, 859–865. Journal of Neurology (2021) 268:431–439 AJNR Am J Neuroradiol 2020, 41 (3) www.thelancet.com/neurology Vol 12 July 2013

Vestibular mapping : MD vs VM



AC cVEMP (n=58)
- Normal (55.2%)
- Asymmetric (37.9%)
- Absent bilat. (6.9%)

Caloric (n=57)
- Normal (38.6%)
- Asymmetric CP (61.4%)

BC cVEMP (n=62)
- Normal (82.3%)
- Asymmetric (17.7%)

Lateral vHIT (n=66)
- Normal (98.5%)
- Reduced gain (1.5%)

AC oVEMP (n=36)
- Normal (19.4)
- Asymmetric (44.5%)
- Absent bilat. (36.1%)

Audiogram (n=70)
- Flat asym. (62.9%)
- Low-frequency asym. (31.4%)
- High-frequency asym. (5.7%)

BC oVEMP (n=52)
- Normal (96.2%)
- Asymmetric (1.9%)
- Absent bilat. (1.9%)

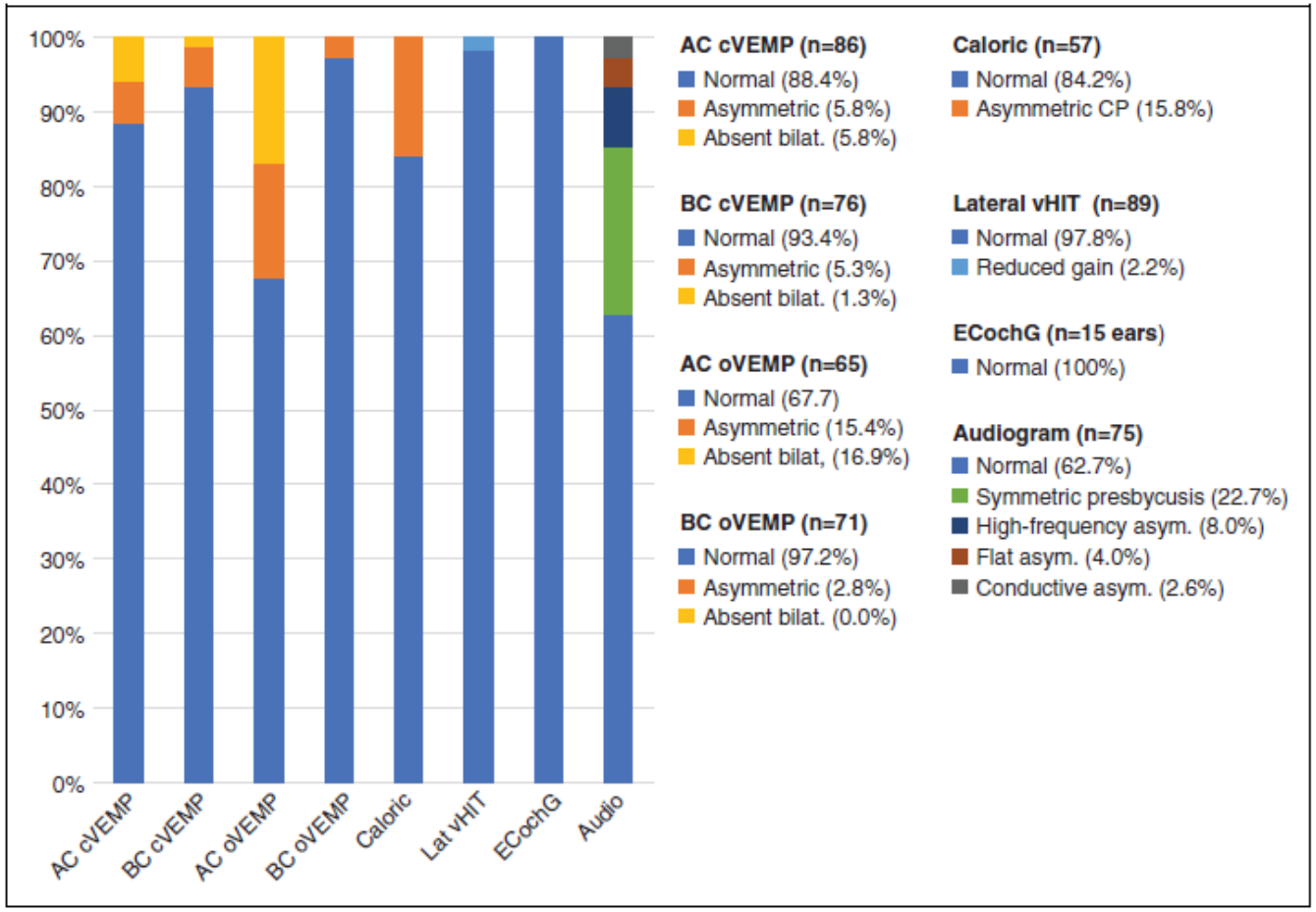


Figure 4. Audiovestibular test results in vestibular migraine patients.
AC: air-conducted; asym: asymmetry; BC: bone-conducted; bilat: bilaterally; c: cervical; ECochG: electrocochleography (trans-tympanic); o: ocular; VEMP: vestibular evoked myogenic potential; CP: canal paresis.

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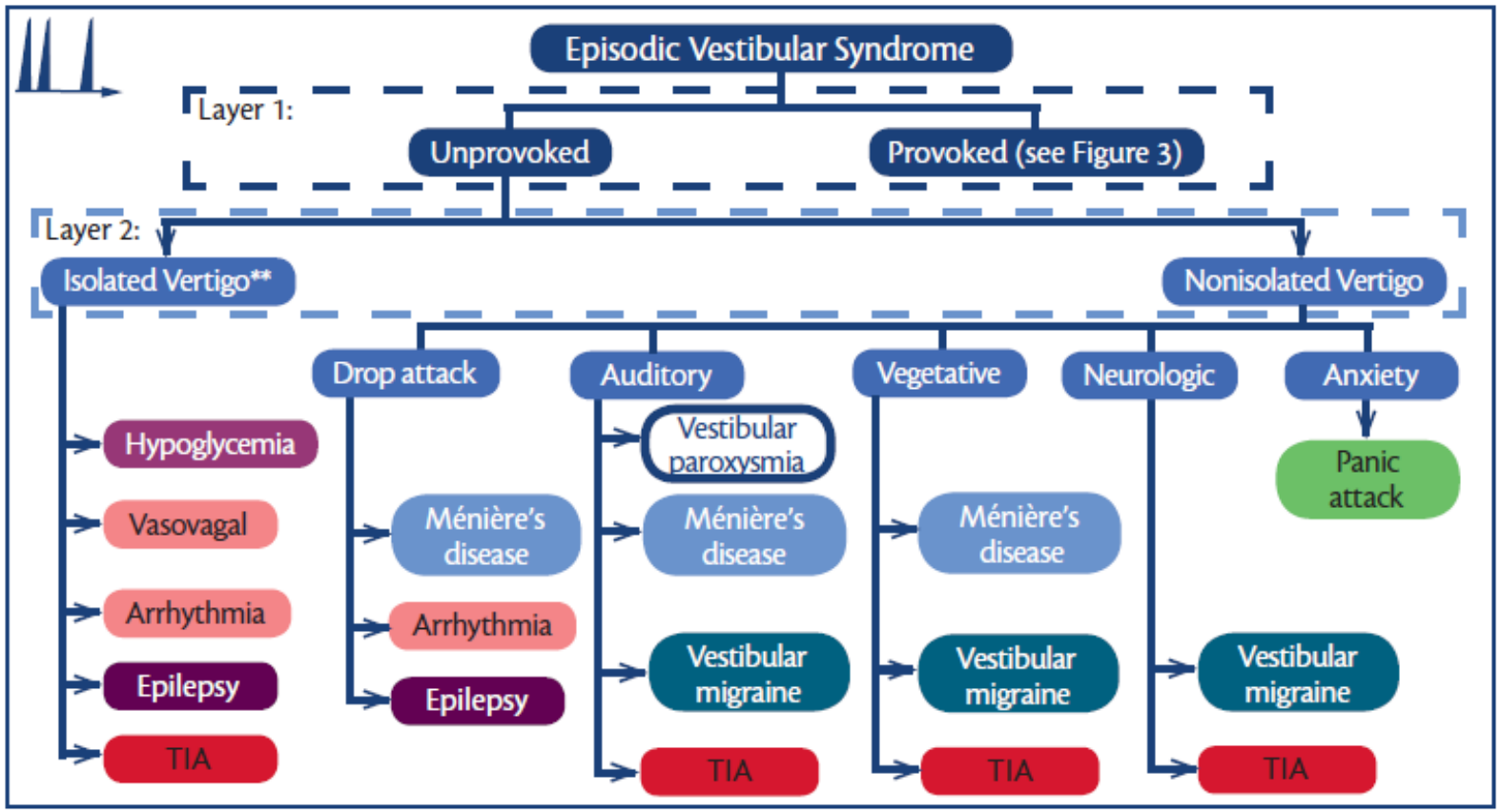
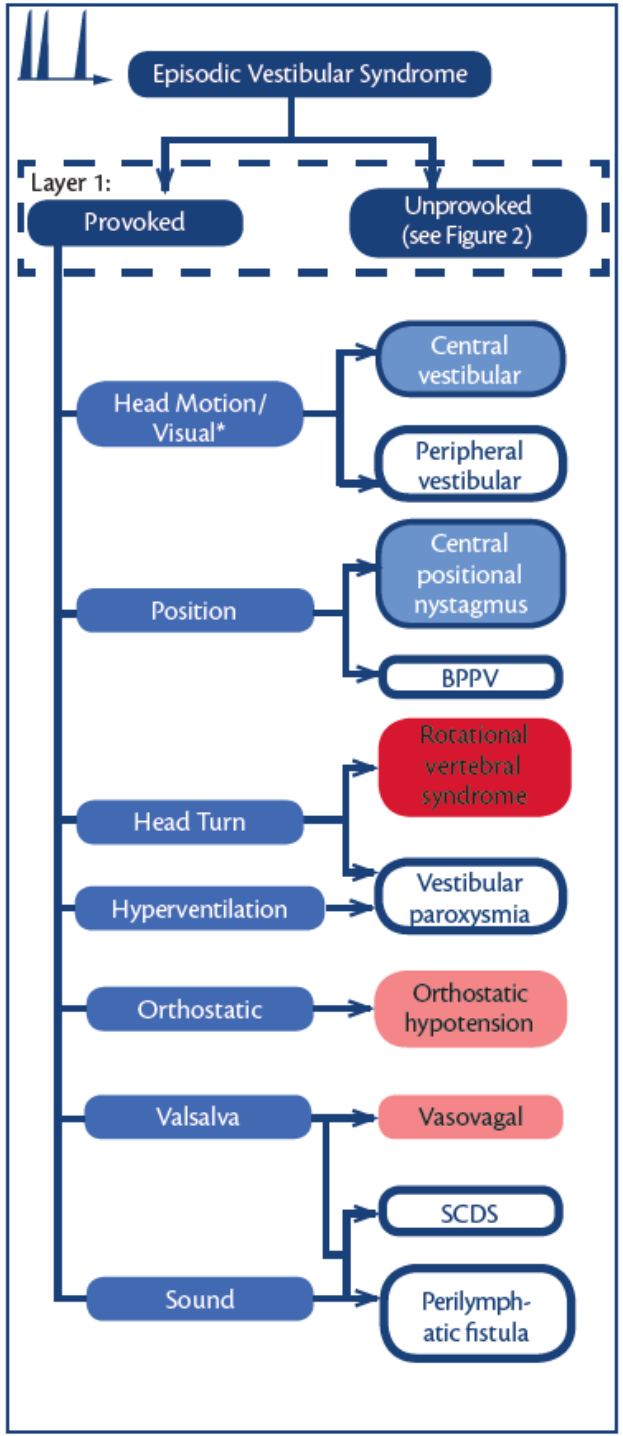
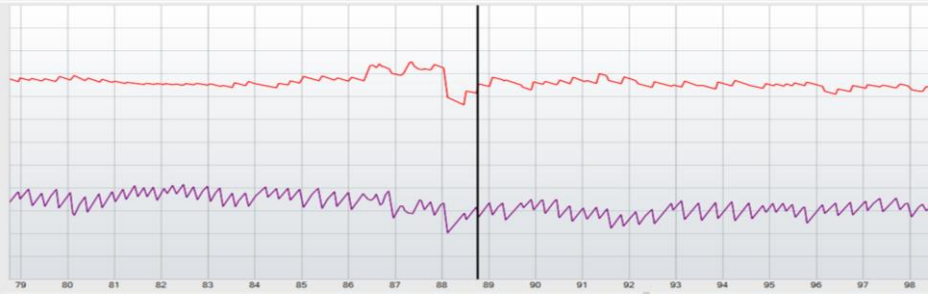

Episodic Vestibular Syndrome
Layer 1:
Unprovoked
Provoked (see Figure 3)
Layer 2:
Isolated Vertigo**
Nonisolated Vertigo
Drop attack
Auditory
Vegetative
Neurologic
Anxiety
Hypoglycemia
Vasovagal
Arrhythmia
Epilepsy
TIA
Ménière's disease
Arrhythmia
Epilepsy
Vestibular paroxysmia
Ménière's disease
Vestibular migraine
TIA
Ménière's disease
Vestibular migraine
TIA
Vestibular migraine
TIA
Panic attack

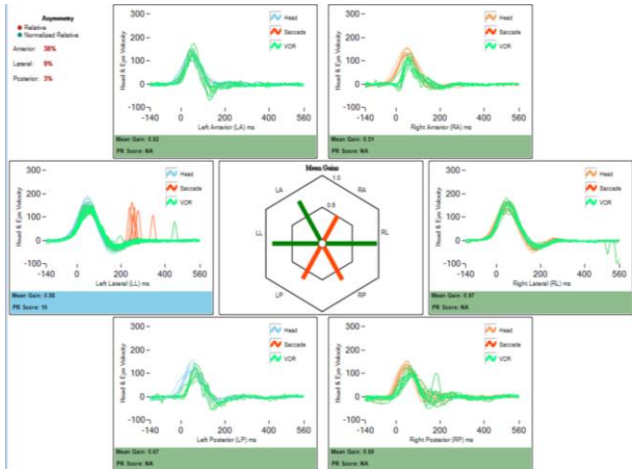

Episodic Vestibular Syndrome
Layer 1:
Provoked
Unprovoked (see Figure 2)
Head Motion/ Visual*
Central vestibular
Peripheral vestibular
Position
Central positional nystagmus
BPPV
Head Turn
Rotational vertebral syndrome
Hyperventilation
Vestibular paroxysmia
Orthostatic
Orthostatic hypotension
Valsalva
Vasovagal
SCDS
Sound
Perilymph-atic fistula

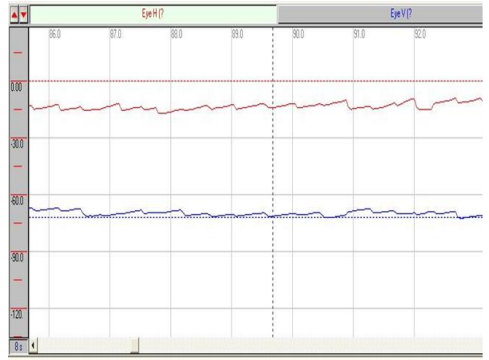
t-EVS : CPPV vs BPPV

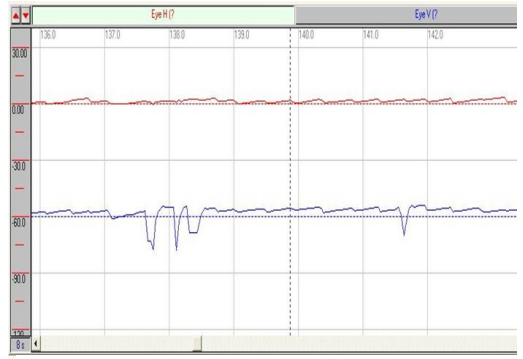
(Central paroxysmal positional vertigo)

CPPV mimicking BPPV of superior canal or non-ampullary arm posterior canal

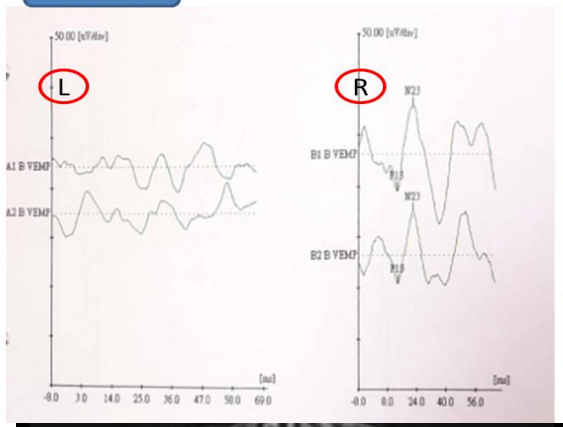




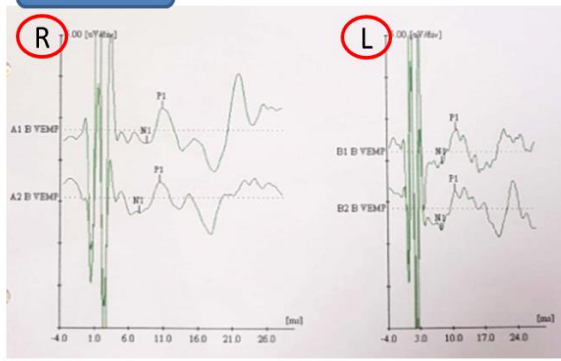


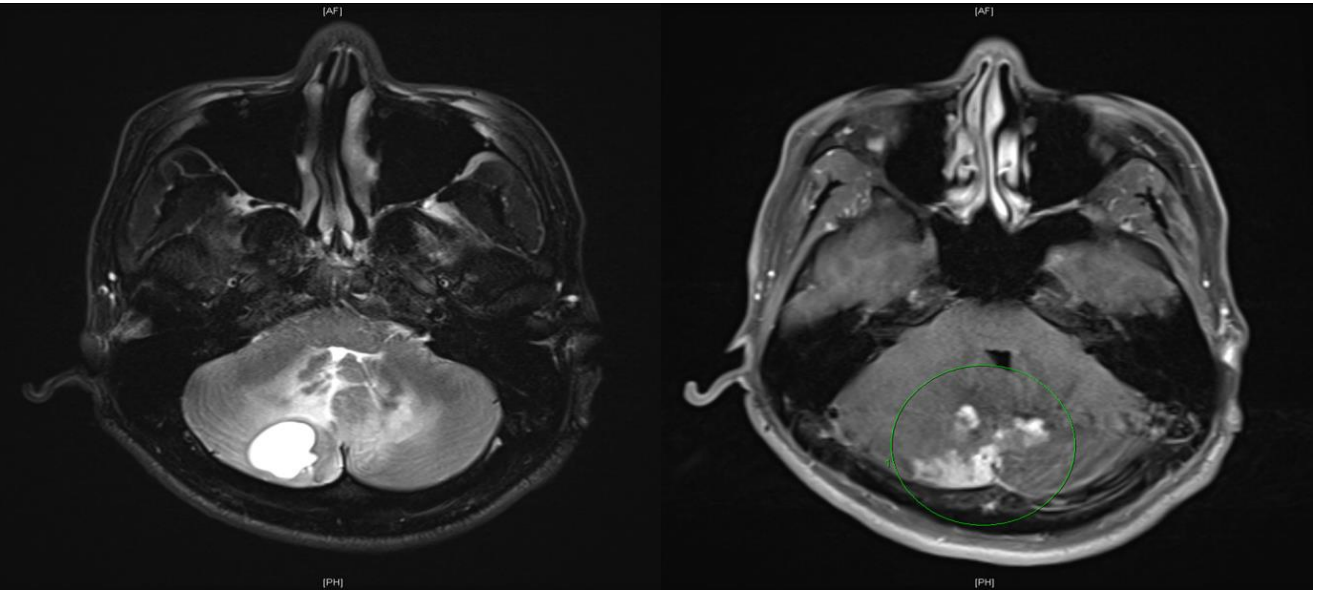


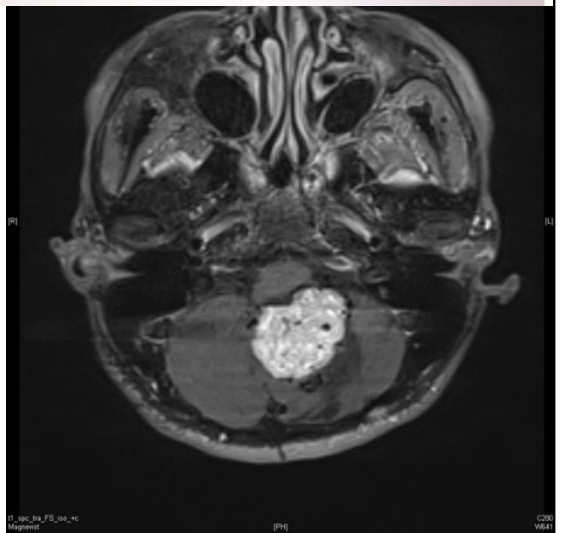
c-VEMP

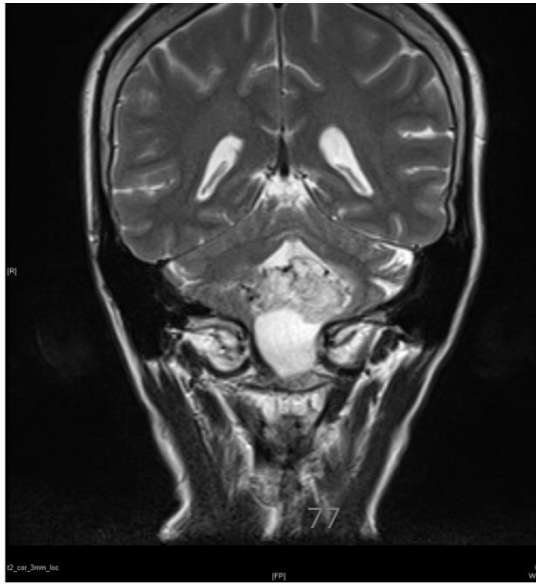


o-VEMP





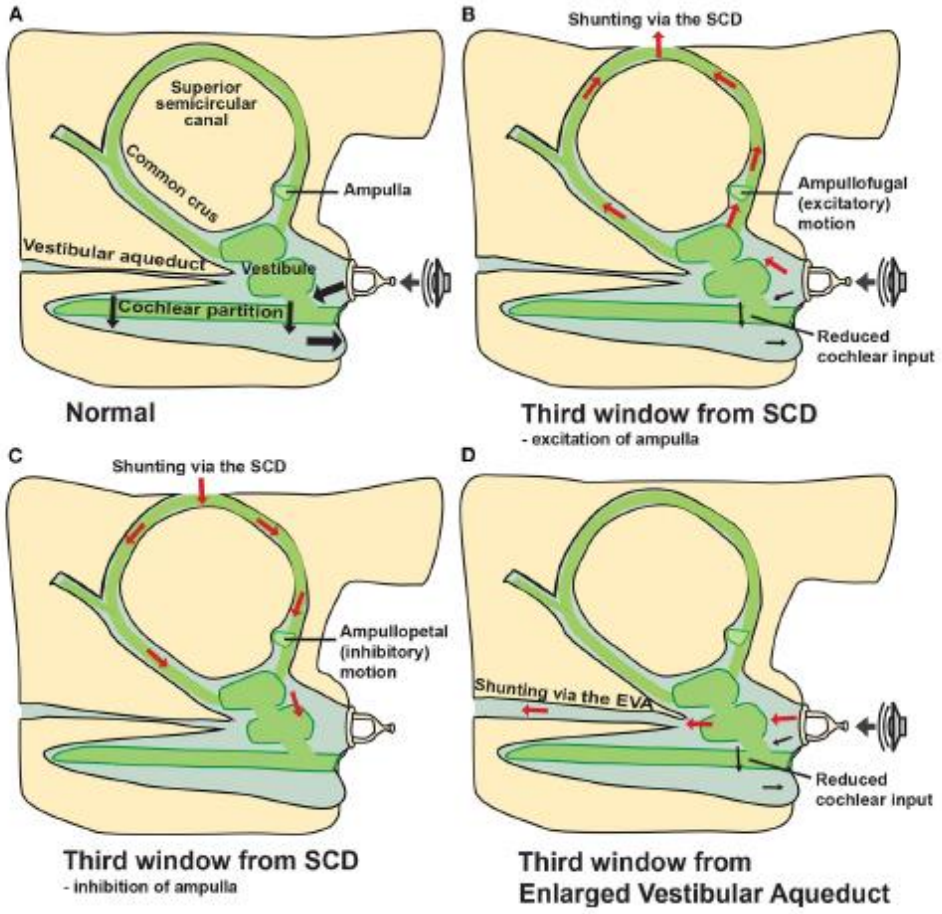


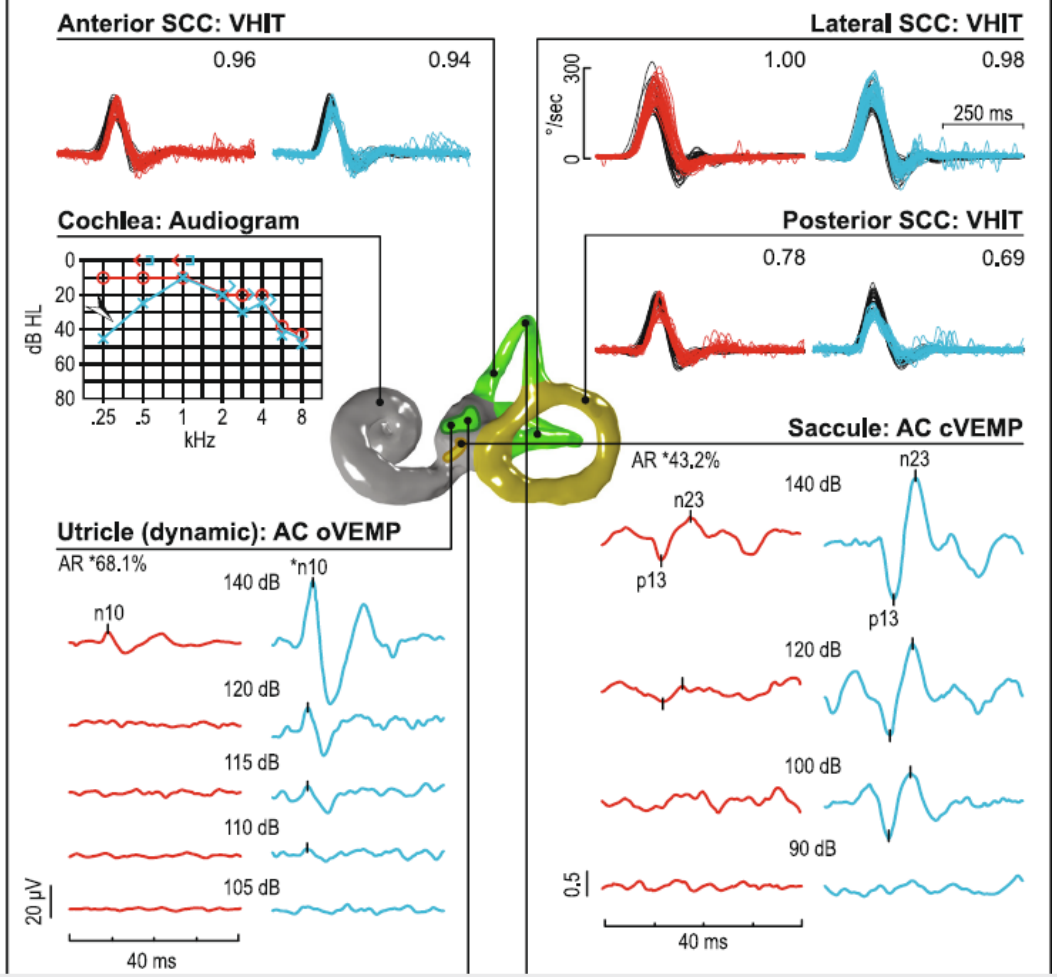


t-EVS : third window disease superior semicircular dehiscence (SSCD)

VEMPs in Third Window Diseases

- Both AC VEMP testing are augmented in third window diseases (superior semicircular dehiscence) with high amplitude and low threshold





▶ **Table 2** Differential diagnosis of middle-ear and inner-ear conductive hearing loss (CHL) [131] (see Chapter 3.2.1)

Audio-vestibular test	Middle-Ear Conductive Hearing Loss	Inner-Ear Conductive Hearing Loss
tympanogram	type A, B or C	type A
stapedial reflex	often absent	present
otoacoustic emissions	absent	present
bone-conduction thresholds	rarely <0 dB nHL	frequently <0 dB nHL for frequencies <2 kHz
ACS VEMPs	reduced or absent amplitudes (for CHL ≥ 10 dB)	increased amplitudes, reduced thresholds despite CHL

Abbreviations: ACS VEMPs = vestibular evoked myogenic potentials (VEMPs) evoked by air-conducted sound (ACS); nHL = normal hearing level

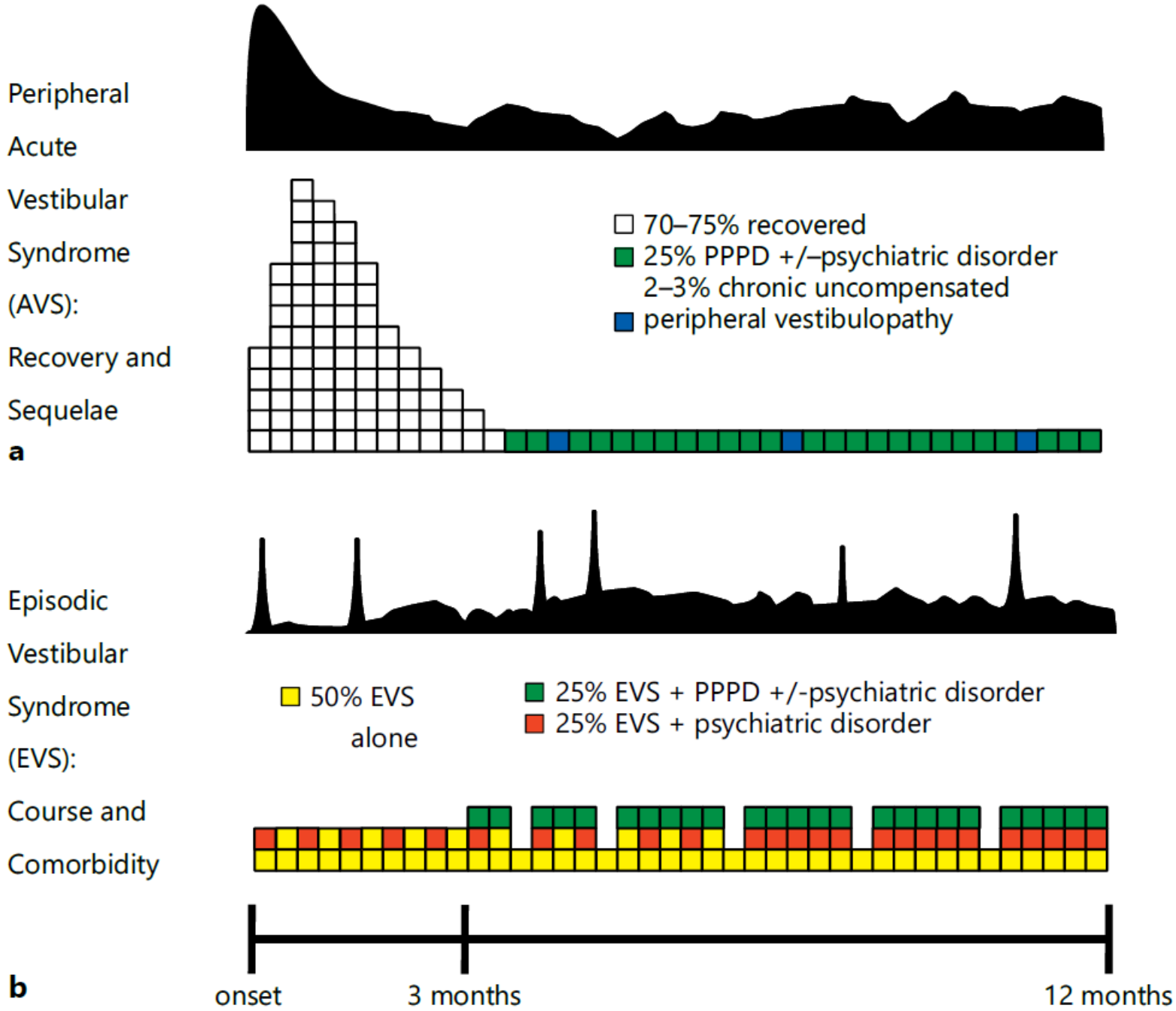
CVS

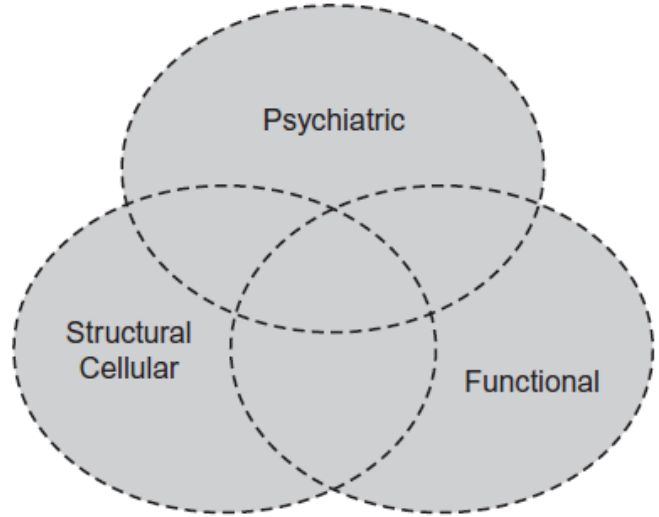
Table 1 International Classification of Vestibular Disorders diagnostic criteria for PPPD

All five criteria, A–E, must be fulfilled to make the diagnosis
A. One or more symptoms of dizziness, unsteadiness, or nonspinning vertigo are present on most days for 3 mo or more
1. Symptoms last for prolonged (hours-long) periods of time but may wax and wane in severity
2. Symptoms need not be present continuously throughout the entire day
B. Persistent symptoms occur without specific provocation, but are exacerbated by three factors:
1. Upright posture
2. Active or passive motion without regard to direction or position
3. Exposure to moving visual stimuli or complex visual patterns
C. The disorder is precipitated by conditions that cause vertigo, unsteadiness, dizziness, or problems with balance including acute, episodic, or chronic vestibular syndromes; other neurologic or medical illnesses; or psychological distress
1. When the precipitant is an acute or episodic condition, symptoms settle into the pattern of criterion A as the precipitant resolves, but they may occur intermittently at first, and then consolidate into a persistent course
2. When the precipitant is a chronic syndrome, symptoms may develop slowly at first and worsen gradually
D. Symptoms cause significant distress or functional impairment
E. Symptoms are not better accounted for by another disease or disorder

Abbreviation: PPPD, persistent postural-perceptual dizziness.

Source: Reprinted with permission from Staab JP, Eckhardt-Henn A, Horii A, et al. Diagnostic criteria for persistent postural-perceptual dizziness (PPPD): consensus document of the Committee for the Classification of Vestibular Disorders of the Barany Society. J Vestib Res 2017;27(4):191–208.





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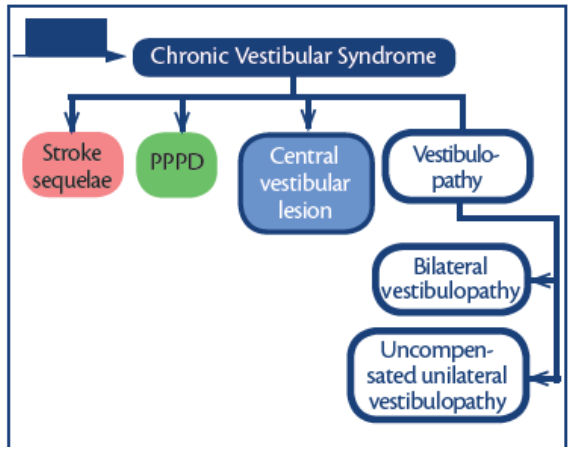
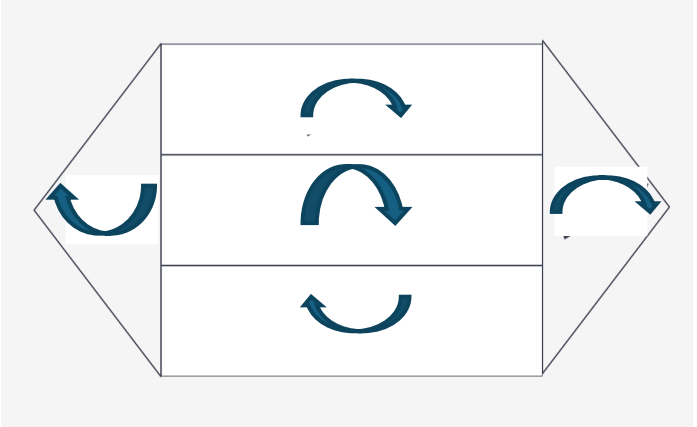
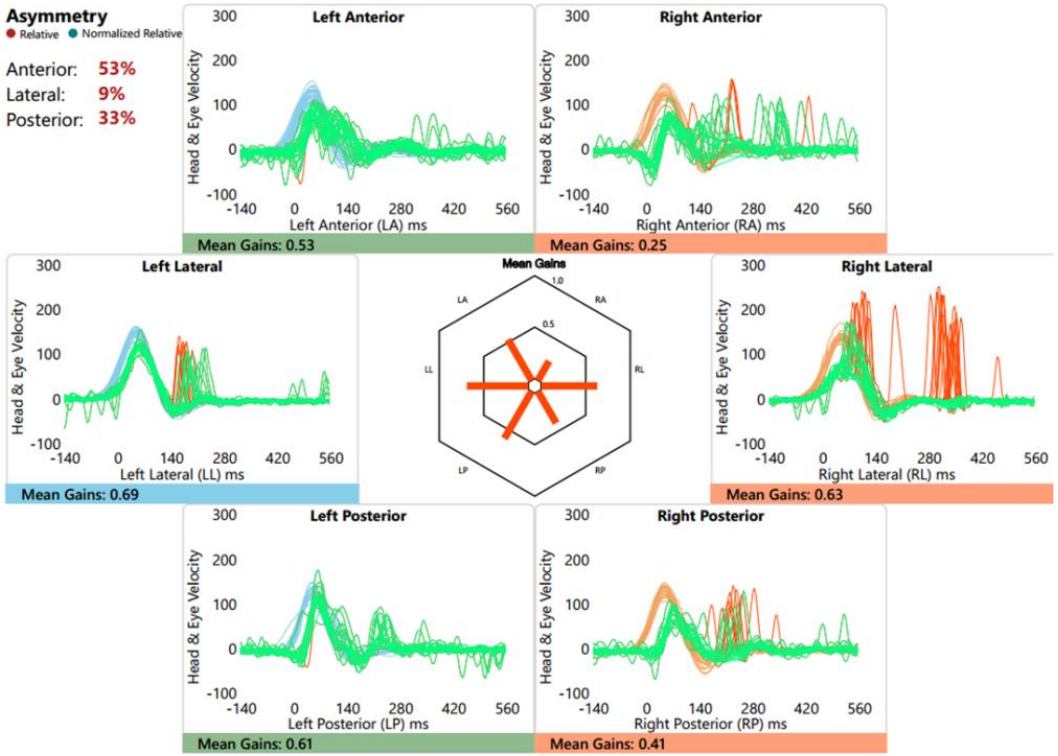
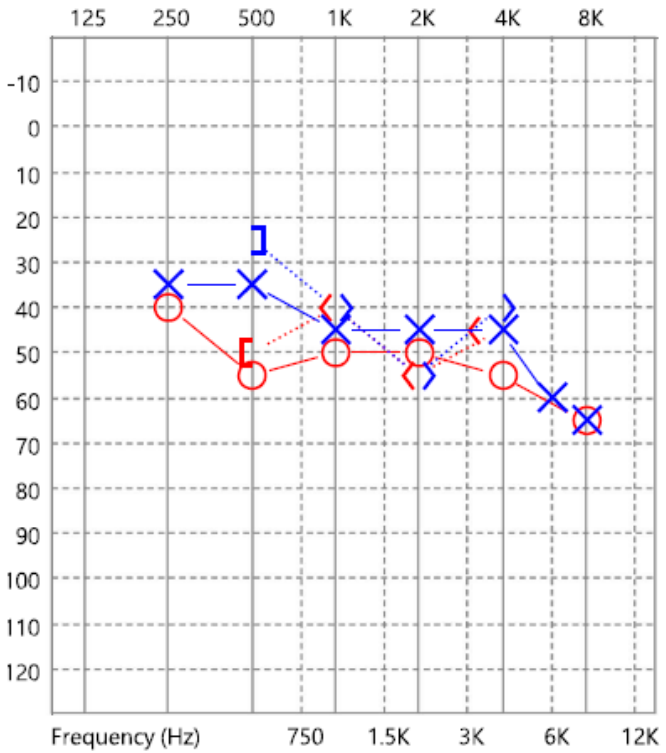
Functional Neurologic Disorders
M. Hallett, J. Stone, and A. Carson, Editors

Adv Otorhinolaryngol. Basel, Karger, 2019

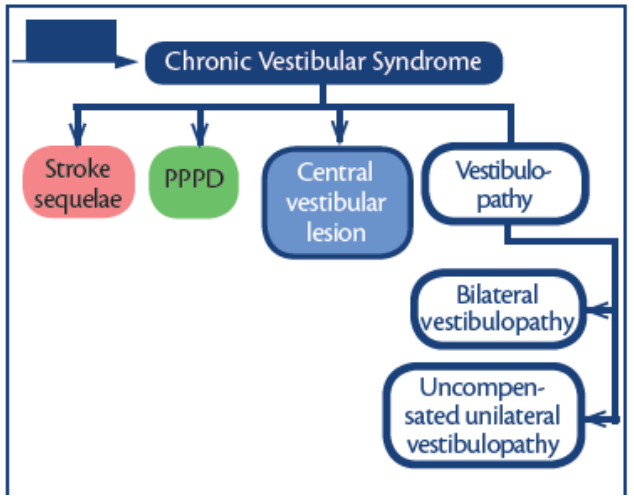

Chronic Vestibular Syndrome
Stroke sequelae
PPPD
Central vestibular lesion
Vestibulo-pathy
Bilateral vestibulopathy
Uncompen-sated unilateral vestibulopathy

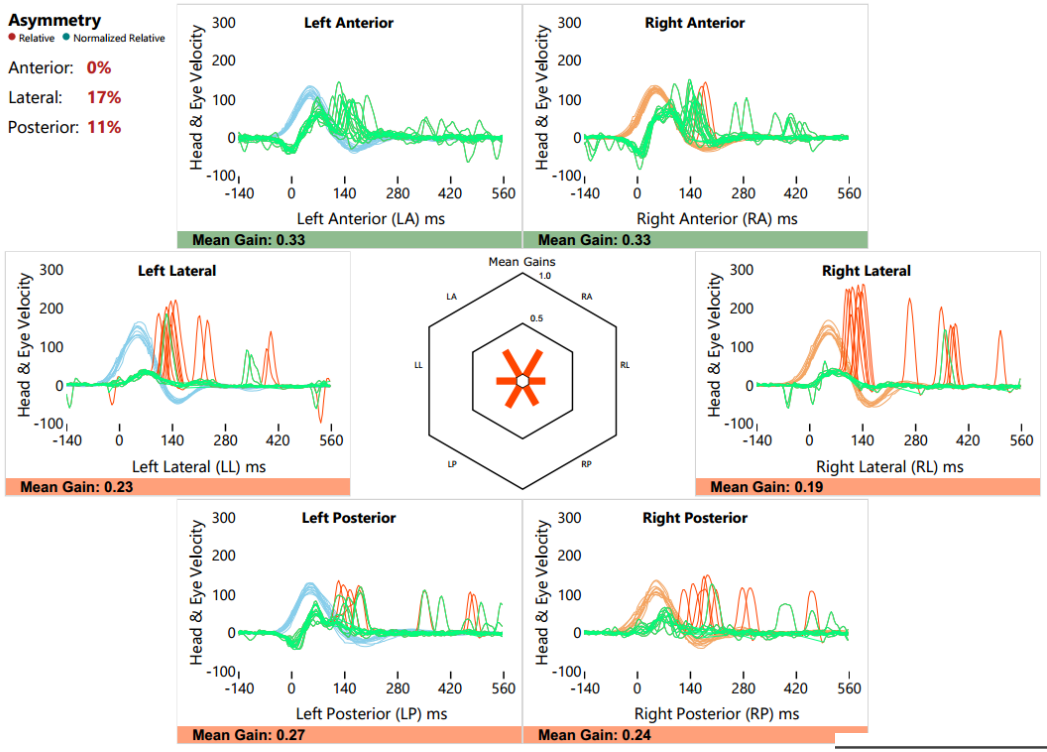


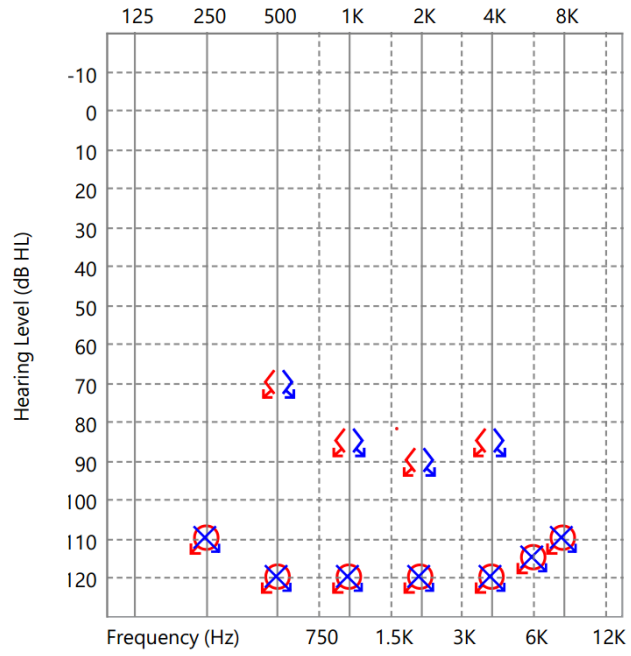

Asymmetry
Relative
Normalized Relative
Anterior: 53%
Lateral: 9%
Posterior: 33%
Left Anterior
Left Anterior (LA) ms
Mean Gains: 0.53
Right Anterior
Right Anterior (RA) ms
Mean Gains: 0.25
Left Lateral
Left Lateral (LL) ms
Mean Gains: 0.69
Mean Gains
Right Lateral
Right Lateral (RL) ms
Mean Gains: 0.63
Left Posterior
Left Posterior (LP) ms
Mean Gains: 0.61
Right Posterior
Right Posterior (RP) ms
Mean Gains: 0.41
Head & Eye Velocity

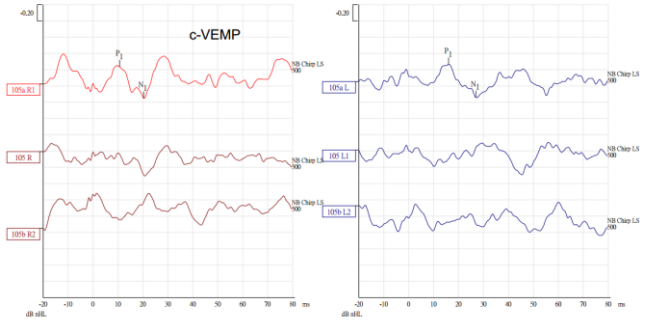

125
250
500
1K
2K
4K
8K
Frequency (Hz)
750
1.5K
3K
6K
12K

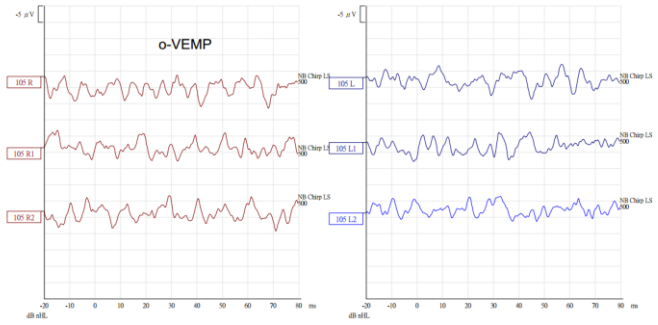

ALH
Institution: CHANGHUA
C.T. Scan head ro
PRF

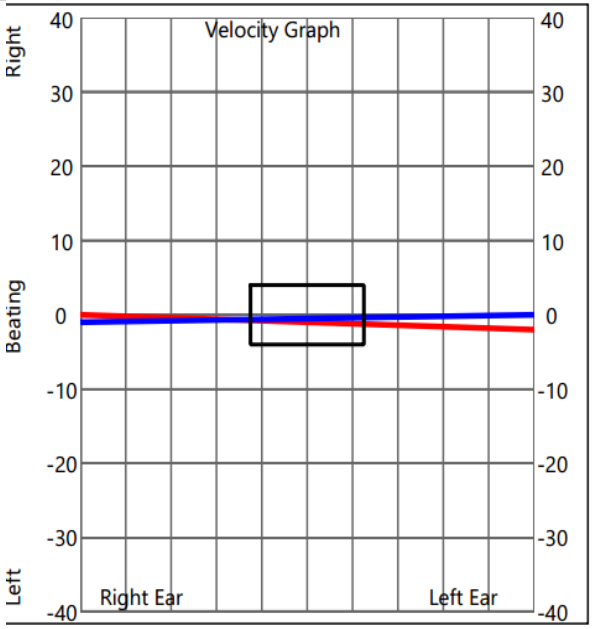




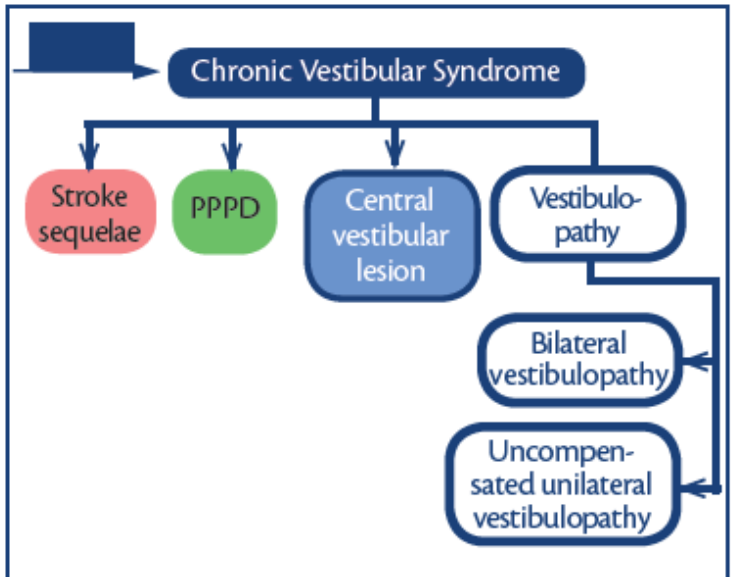








Pods Analysis	
Total Right:	1°/s
Total Left:	2°/s
Spontaneous Nystagmus:	
Unilateral Weakness:	
Gain Asymmetry:	
SPV Peak (deg/sec)	
Left Warm:	2 at 35.5 s
Right Warm:	0 at 26.5 s
Left Cool:	0 at 24.5 s
Right Cool:	1 at 21.5 s

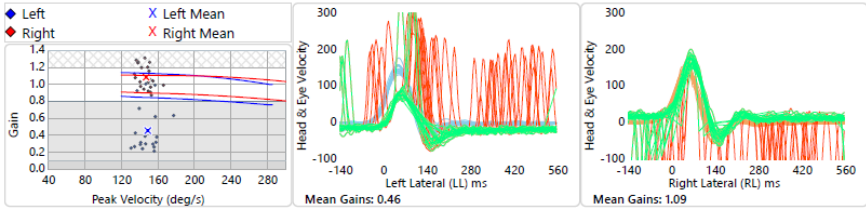


Patient ID: 20210624
DOB: 4/1/1952
Gender: Male

Head Impulse

Lateral Impulse Test: 6/24/2021 5:01:13 PM
Test Operator: Default Administrator

x̄ Left: 0.46, σ: 0.26 x̄ Right: 1.09, σ: 0.14
Relative Asymmetry: 58%



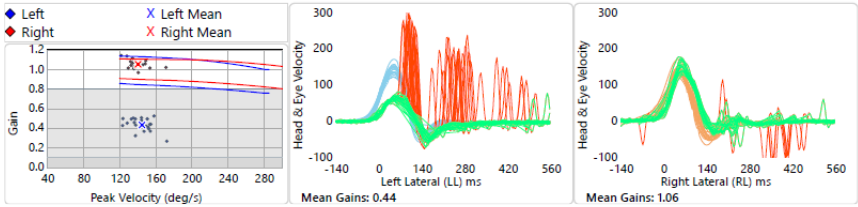
Patient ID: 20210712
DOB: 4/1/1952
Gender: Male

Report Date: 7/12/2021 Report Operator: Default Administrator

Head Impulse

Lateral Impulse Test: 7/12/2021 9:08:55 AM
Test Operator: Default Administrator

x̄ Left: 0.44, σ: 0.06 x̄ Right: 1.06, σ: 0.05
Relative Asymmetry: 58%



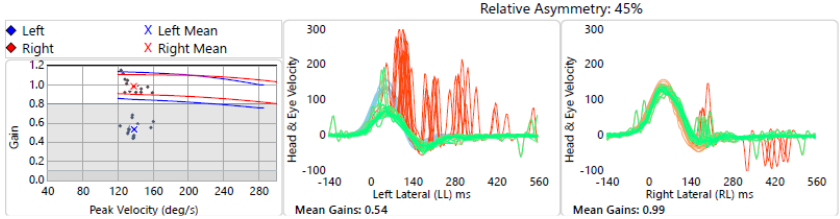
Patient ID: 20210924
DOB: 4/1/1952
Gender: Male

Report Date: 9/27/2021 Report Operator: Default Administrator

Head Impulse

Lateral Impulse Test: 9/24/2021 9:41:26 AM
Test Operator: Default Administrator

x̄ Left: 0.54, σ: 0.07 x̄ Right: 0.99, σ: 0.07
Relative Asymmetry: 45%

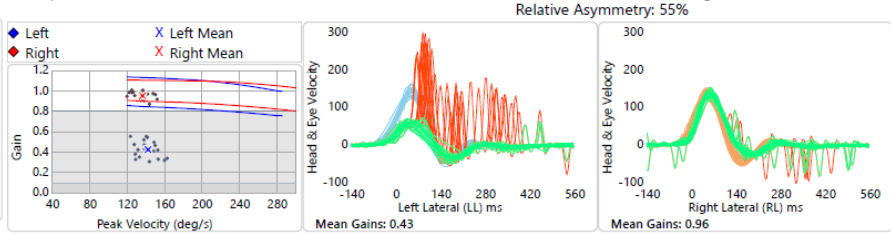


Patient ID: 20211215
DOB: 4/1/1952
Gender: Male

Head Impulse

Lateral Impulse Test: 12/15/2021 3:05:18 PM
Test Operator: Default Administrator

x̄ Left: 0.43, σ: 0.08 x̄ Right: 0.96, σ: 0.04
Relative Asymmetry: 55%



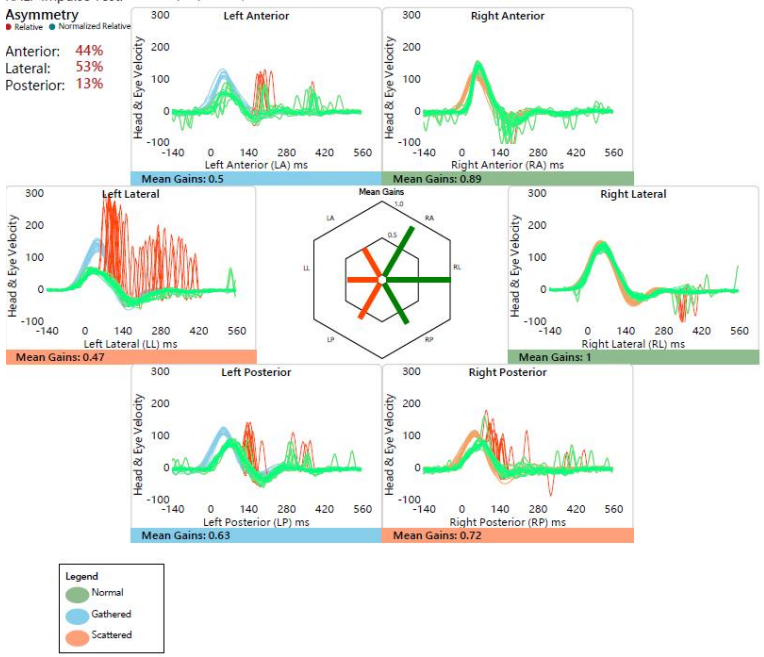
Patient ID: 20221020
DOB: 1952/4/1
Gender: Male

Head Impulse

Lateral Impulse Test: 2022/10/20 下午 03:59:39
LARP Impulse Test: 2022/10/20 下午 04:01:42
RALP Impulse Test: 2022/10/20 下午 04:06:08

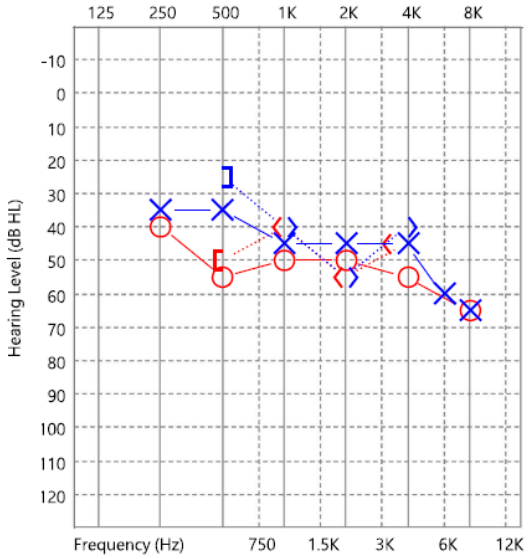
Asymmetry
Relative Normalized Relative

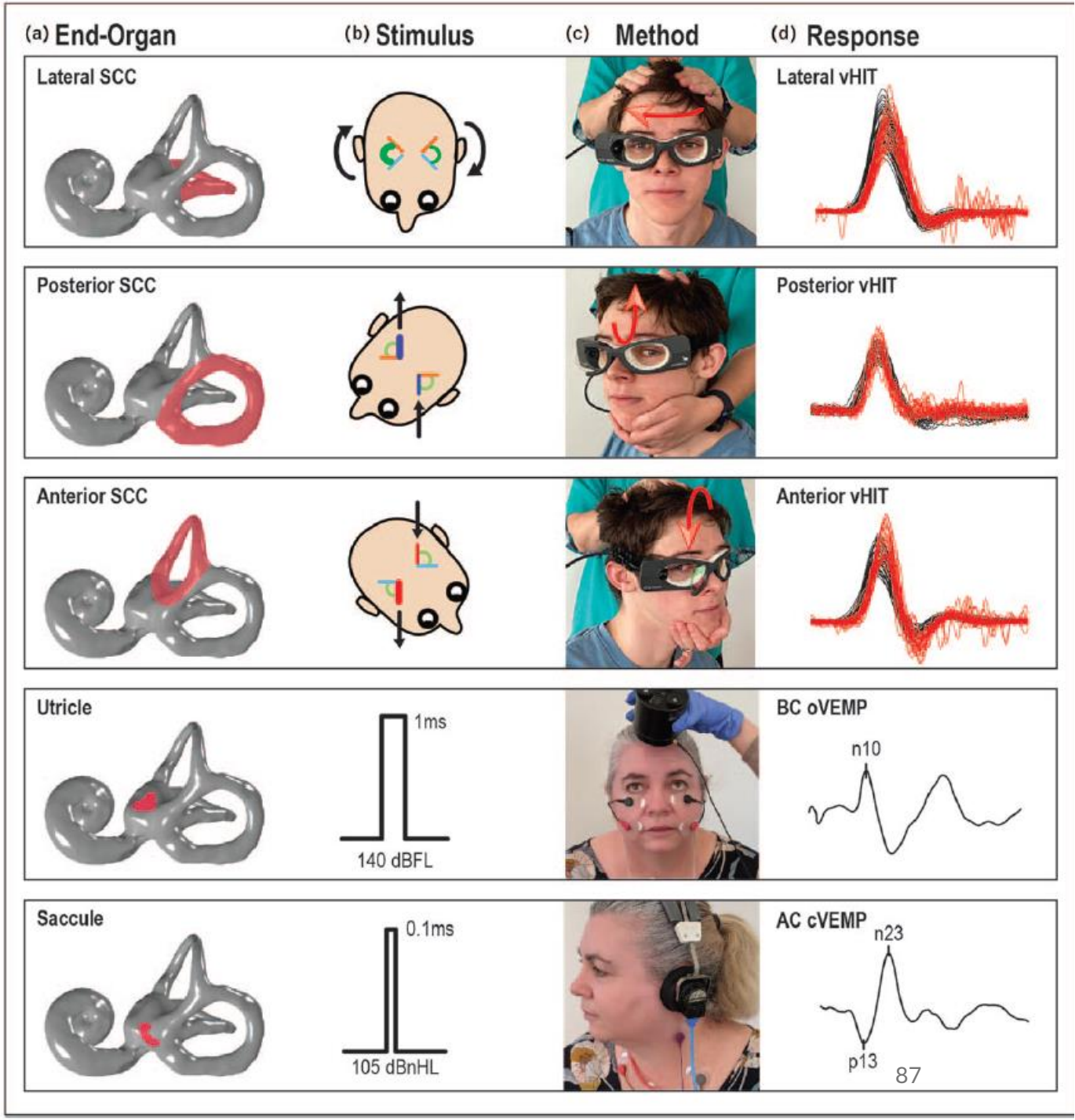
Anterior: 44%
Lateral: 53%
Posterior: 13%



VFT selection in daily practice

- s-AVS
- s-EVS
- t-AVS
- t-EVS
- CVS





Vestibular function testing in the 21st century Rosengren *et al.*

	goggle	PTA	vHIT	HINT Plus	VEMP
s-AVS	+	+	+	+	+/-
CVS	+	+	+	+/-	+/-
s-EVS	+	+	+/-	+/-	+/-
t-AVS t-EVS	+/-	+/-	+/-	-	+/-

Thanks