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vHIT interpretation

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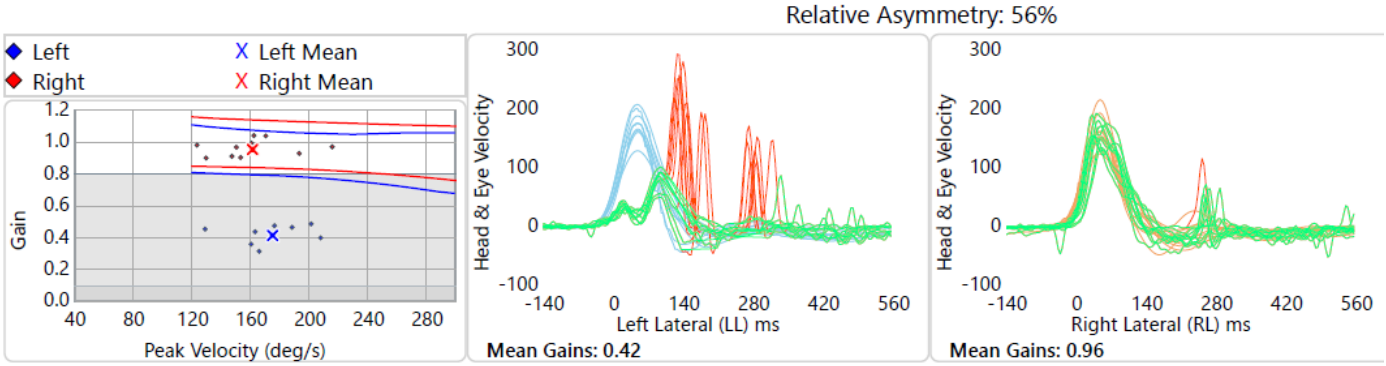
Video head Impulse Test (vHIT)

- Still no evidence-based recommended procedures or guidelines for interpretation.
- The following is based on a review of the current literature and clinical experience
- New papers being published all the time
- Still ***a lot*** we don't understand about this test paradigm

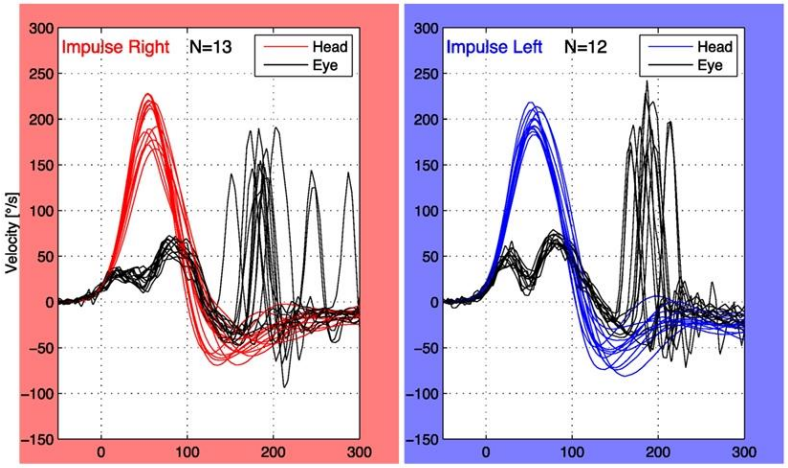
Unequivocal vHIT results

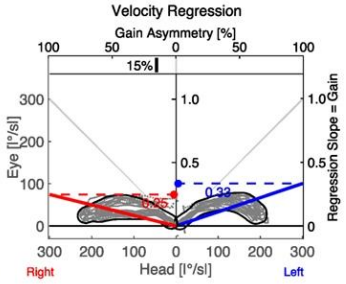
- Classic triad for dysfunction of the high-frequency VOR
  1. Abnormal morphology of eye curve (shallow)
  2. VOR gain below the normal range
  3. Large saccades



Dysfunction of the left lateral semi-circular canal and/or superior vestibular nerve

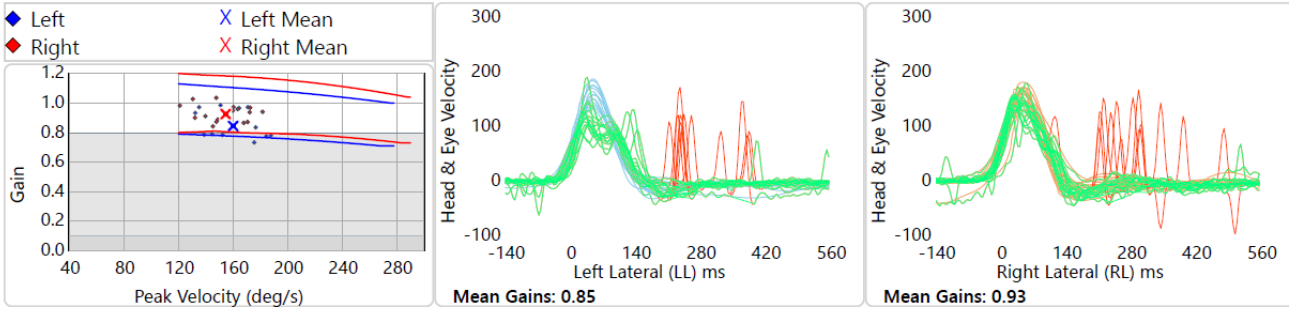
Bilateral dysfunction of the lateral semi-circular canals and/or superior vestibular nerves

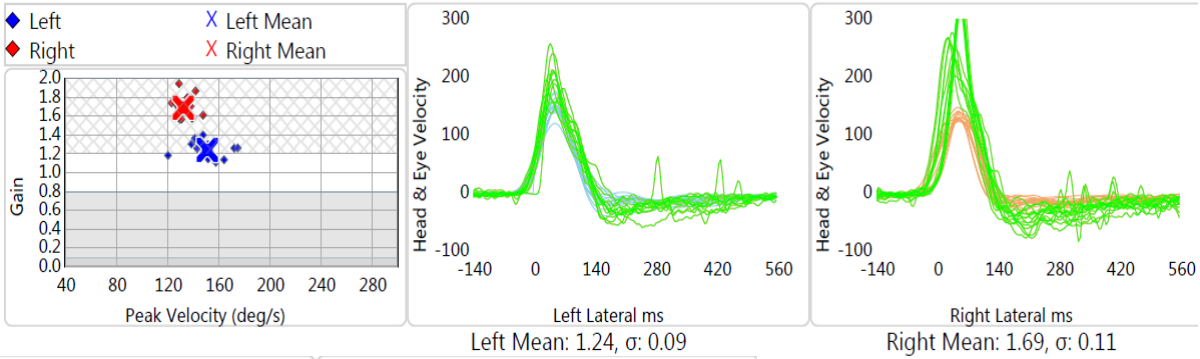


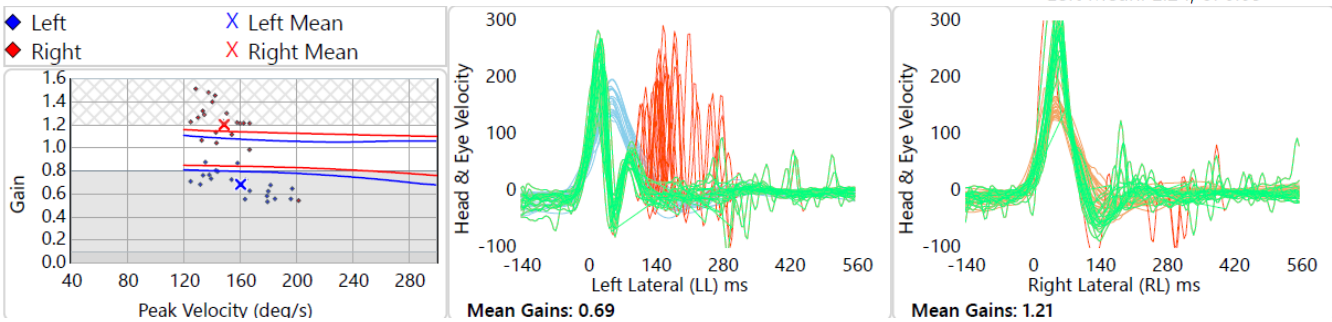


Equivocal vHIT results

- But what about these?







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Four vHIT systems

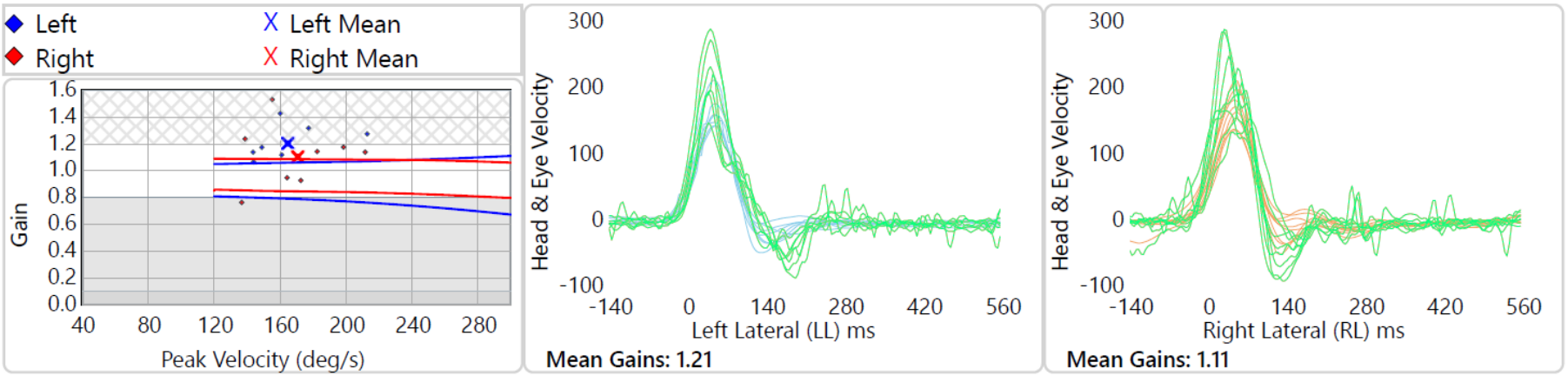
	Device	Sold by	Validated against scleral search coils?		Peer-reviewed original research articles
			Horizontals	Verticals	
	ICS Impulse	GN Otometrics	✓	✓	>300
	Eyeseecam	Interacoustics	✓	✗	approx. 20
	vHIT Ulmer	Synapsys	✗	✗	approx. 15
	VORTEQ	Micromedical	✗	✗	approx. 5

VOR gain

Measuring gain

- Essentially: **VOR gain =** $\frac{\textbf{Eye velocity}}{\textbf{Head velocity}}$
- Normal range for gain is 0.8 – 1.2 (lateral canals)
  0.7 - 1.2 (vertical canals)
- Gain is affected by the device used (Cleworth *et al.*, 2017; Janky *et al.*, 2017). , head velocity, and the eye that is being recorded – but all within the normal range
- Asymmetry is not a focus of the vHIT literature, and has limited clinical value compared with absolute gain values and saccade metrics.
  - Compared with calorics, the size of the vHIT normal range is much tighter, therefore comparisons between ears become less relevant (Curthoys *et al.*, 2008).

Goggle slippage



- Most commonly occurs in those with lots of hair or loose skin on the head
- Particularly problematic in non-caucasian subjects, when goggles can float over the nose, and tightening the strap can result in excessive pressure on the lateral eye rims.

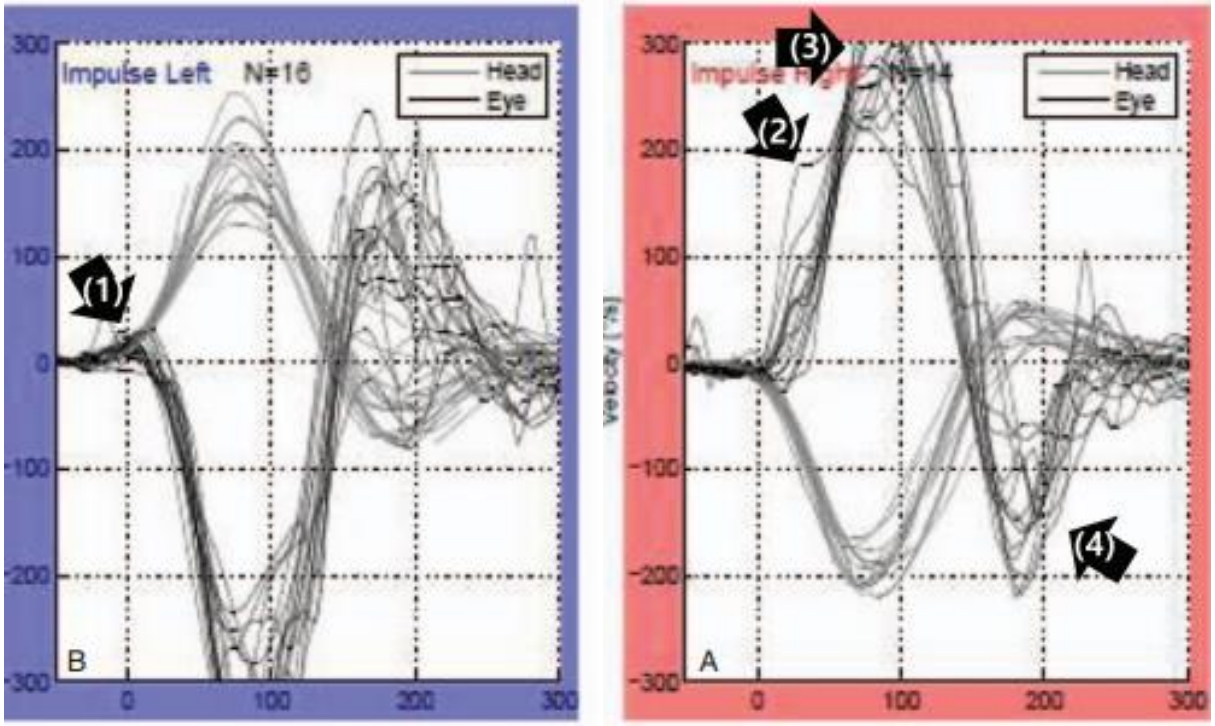
- Tighten goggle strap as much as possible
- Locate strap over occiput
- Consider alternative hand technique (hands on top vs hands on jaw)
- Can use putty on bridge of nose (Versino *et al.,* 2013)

Goggle slippage

Various artefacts can be seen

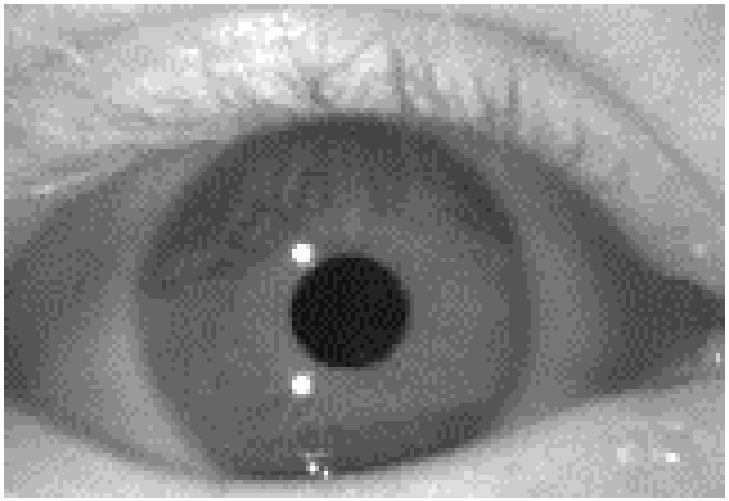
1. Initial backwards eye movement toward the head movement near 0 ms
2. Eye leading head, and acceleration bump
3. **High gain** near 80ms
4. Deceleration bump

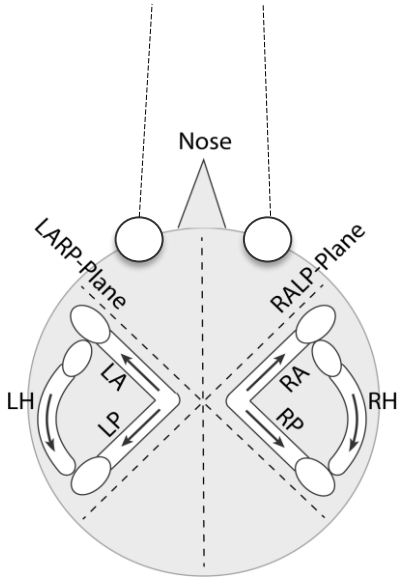
See Suh *et al., (*2017) for discussion of mechanics of goggle slip.



Vertical canals – plane of movement

- Movement of head in LARP or in RALP plane combination of pitch and roll
- Corresponding VOR eye movement for a target at 0° is combination of vertical and torsional movement, involving superior/inferior recti and superior/inferior oblique muscles



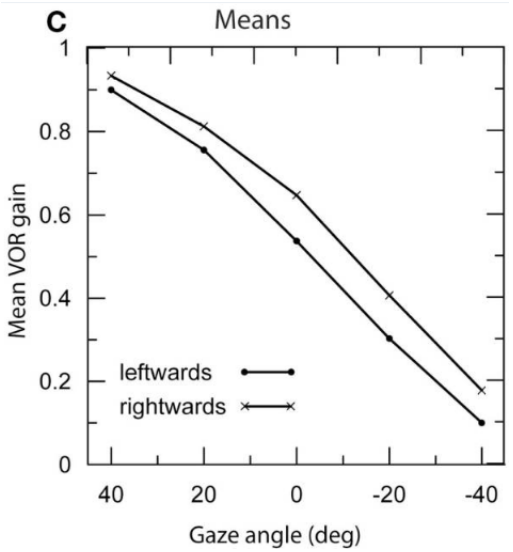


Target at 0°

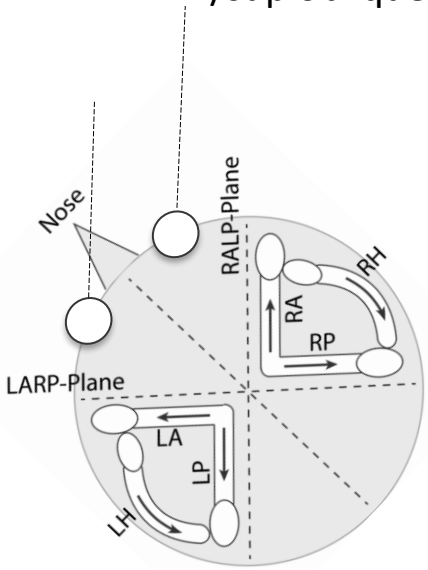
- Torsion cannot be detected with any current software, so torsional component of gain cannot be captured and gain will reflect only vertical component

Vertical canals – plane of movement

- This has been well demonstrated in McGarvie *et al., (*2015)
- So need to turn head (or body) away from target to make the eye movement purely vertical and only engage two extraocular muscles
  - Sup/inf reci when recording eye is abducting
  - Inf/sup obliques when recording eye is adducting

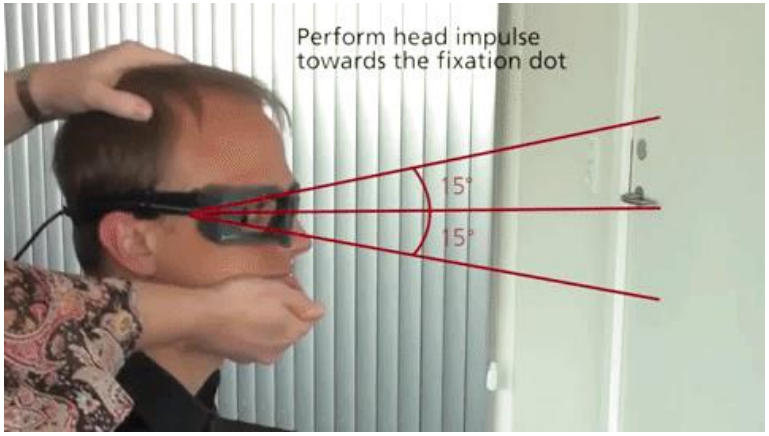


From McGarvie *et al.,* 2015



Target at 45 ° for RALPS

- Eyes in same plane as canals
- Subject fixates target out of eccentric gaze
- Tip head towards and away from target

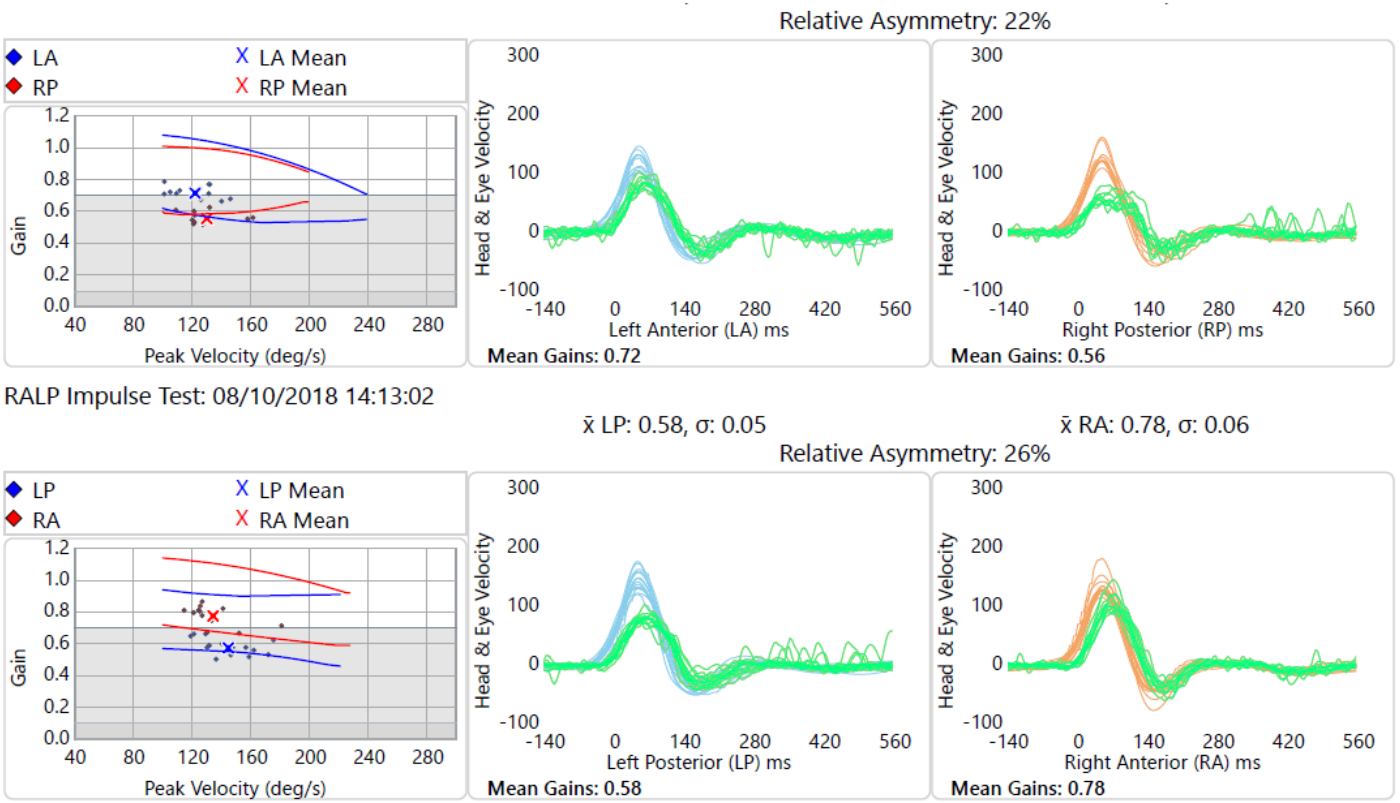


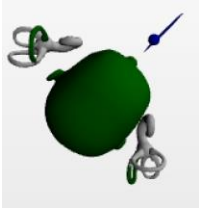
Vertical canals – plane of movement

- Manufacturer of eyeseecam does dispute this
- Recent paper shows that eyeseecam *does* give comparable gain to ICS impulse even when done with head at 0° (Patterson *et al.,* 2020)
  - Only LARPs tested
  - Head velocities were on the slow side
  - Large standard deviations
  - This device has been shown to have poor inter-rater reliability for vertical canals (Abrhamsen *et al.,* 2018)
  - No discussion of torsion – how could it possibly be fair measure of gain without torsion?
- Doing LARPS/RALPS with head at 0° is very difficult for the clinician and the patient
- No guarantee you are getting the right plane
- Turning head (or body on swivel chair) lets you consistently stimulate in the appropriate plane

Vertical canals – plane of movement

Low gain with ‘phase shift’ is sign that canal pair was not perfectly stimulated

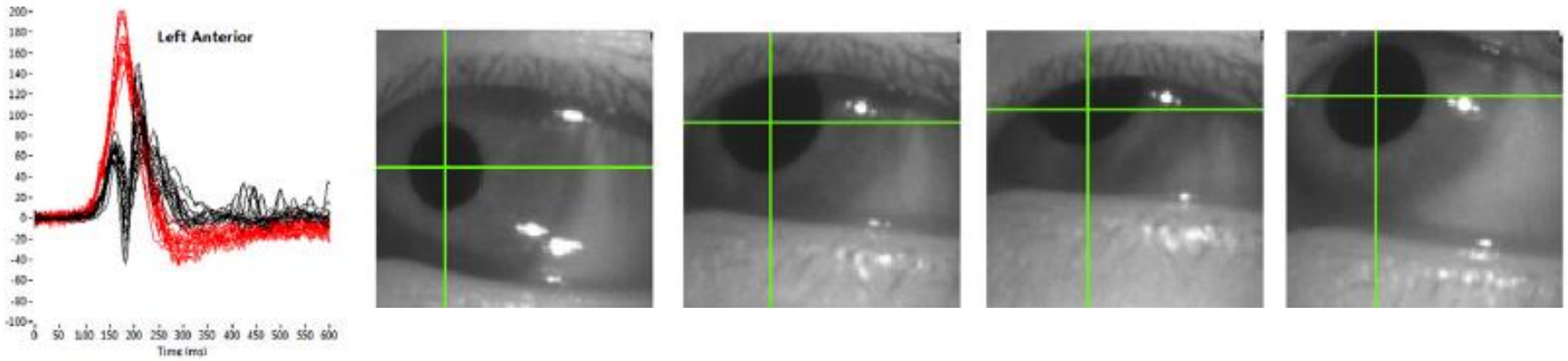




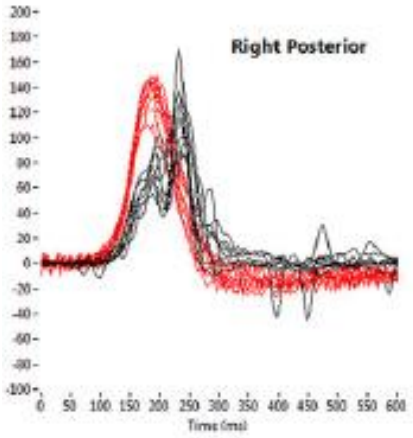
- Can occur without and with saccades (saccades are the true sign of vestibular dysfunction)
- Use green/yellow head
- Remember the large inter-subject variation in semi-circular canal orientation (Blanks *et al.,* 1975, Curthoys *et al.,* 1977, Bradshaw *et al.,* 2010)

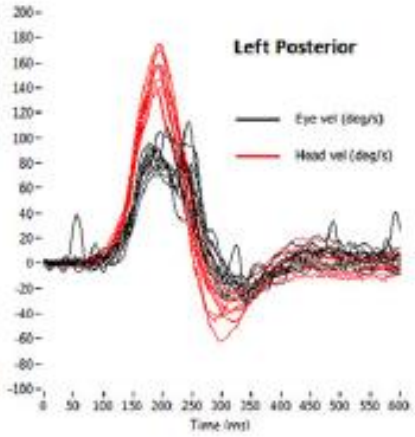
Eyelid artefacts

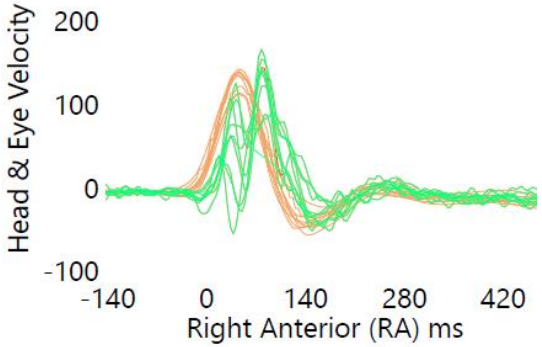
It is vital to ensure the head and eye are positioned in such a way that either eyelid and/or the corneal reflection of an infra-red LED does not interfere with the pupil-tracking crosshair



Halmagyi *et al.,* (2017) discuss this occurring in LA, RP and LP impulses, but can also be seen in RA.

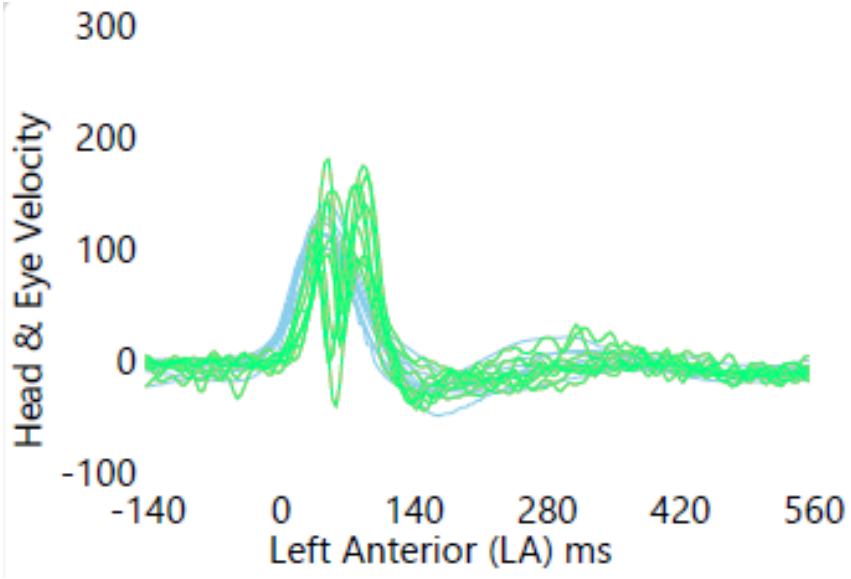


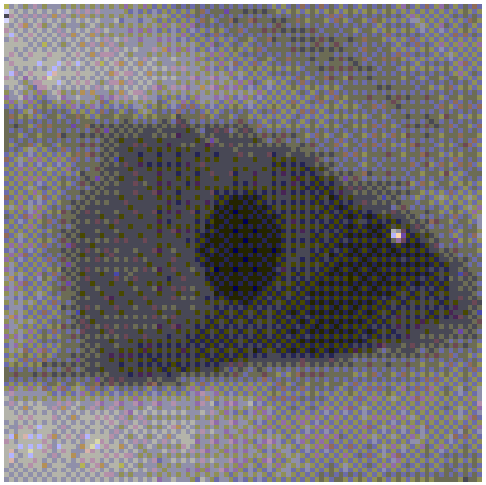




<u>Need to reinstruct on keeping eyes open and potentially use micropore tape to open eyelids</u>

Vertical canals – mini blinks

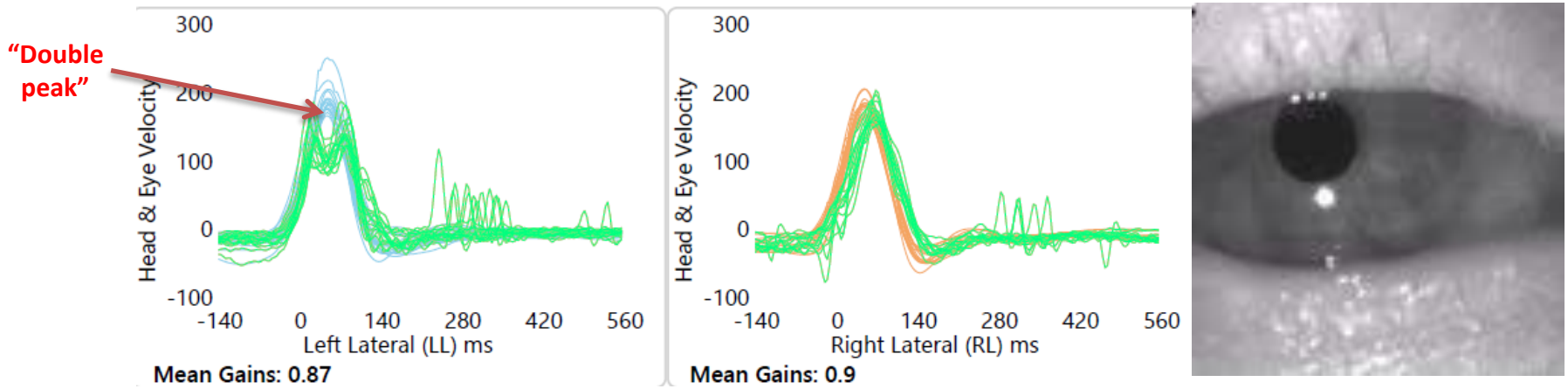




Need to reinstruct on trying not to blink, sometimes this will be impossible as it can be reflexive.

Technical factors that may artificially alter gain

Lateral canals - Eyelid / LED reflection artefact



In laterals you can sometimes see a squashing of the pupil 'blob' affecting velocity curve and causing a double peak.

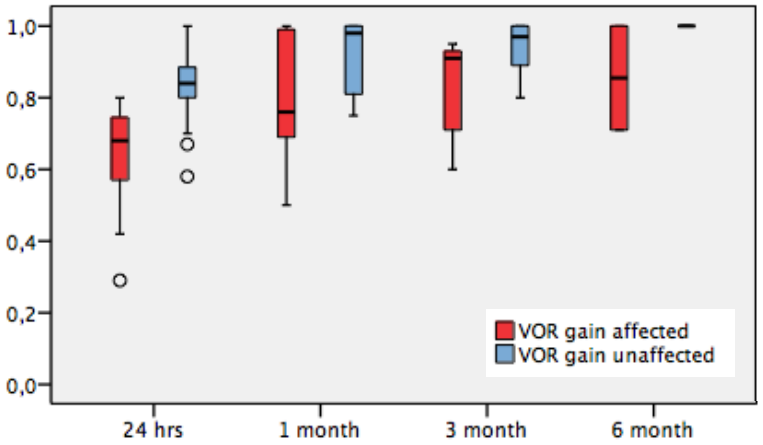
Alternate between 'grayscale' and 'pupil location' image to help see when this is happening

Other technical factors that may artificially alter gain

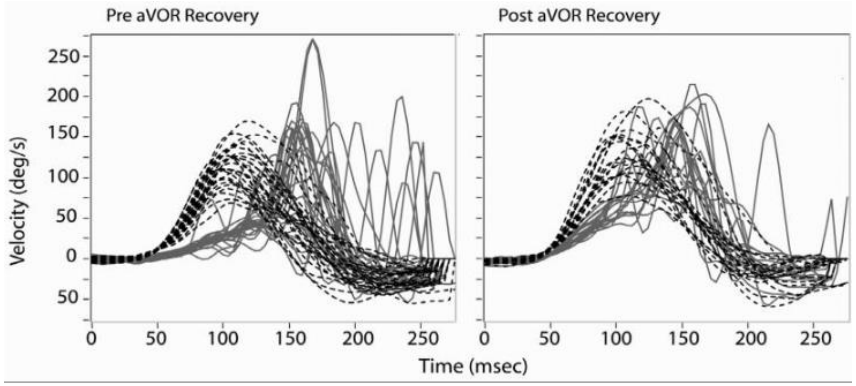
Calibration	If incorrectly performed, can have significant effect on *how far the software thinks the eye moved* during VOR (Mantokoudis *et al.*, 2015) Very high gain *or* low gain without saccades: • Repeat calibration and check for repeatability of Δ
Convergence	If subject too close to target, eyes have to converge slightly, raising gain (Judge *et al.,* 2018) • Seat subject at least 1m from target
Low velocity head impulses	Can mask truly weak VOR gain when head moved too slowly because 1. Ewald's 2nd law: Need to drive contralateral ear into 'inhibitory cutoff' 2. Contribution of smooth pursuit system at lower velocities Move head quickly (laterals150 -250°/s, verticals 100-200°/s) (Curthoys and Manzari 2017)

The bad news about gain

- Gain and the morphology of the VOR eye curve can RECOVER!
  - vHIT gain returned to normal in all 18 vestibular neuritis patients (Martin-Sanz *et al.,* 2017)
  - VHIT gain returned to normal in 15 out of 29 cases of vestibular neuritis (Bartolomeo *et al.,* 2014)
  - vHIT positive in 63% of acute patients (<5 days since onset), but only 33% of non-acute patients (>5 days) (n=172) (Mahringer and Rambold, 2014)



From Martin-Sanz *et al.,* (2017)



In a 42 year old vestibular neuritis patient, VOR gain recovered from 0.25 0.62 after three weeks of vestibular rehabilitation (Schubert *et al.,* 2006).

- High frequency VOR gain had previously been thought of as fairly stable until the advent of vHIT allowed relatively easy longitudinal testing.
- The jury is still out on how this recovery of VOR gain is achieved in these patients

Saccades

vHIT saccades are an exciting area of vestibular research

- Positive results have previously been thought of as having to contain the triad:
  1. Abnormal morphology of eye curve (shallow)
  2. VOR gain below the normal range
  3. Large saccades
- But we are beginning to understand how unreliable (1) and (2) are
- No wonder vHIT is often so insensitive
- Saccades can often be more useful than gain and morphology (Korsager *et al.,* 2016 ; Korsager *et al.,* 2017; Janky *et al.,* 2018)
- Saccades can remain in the vHIT trace for many years after a vestibular event

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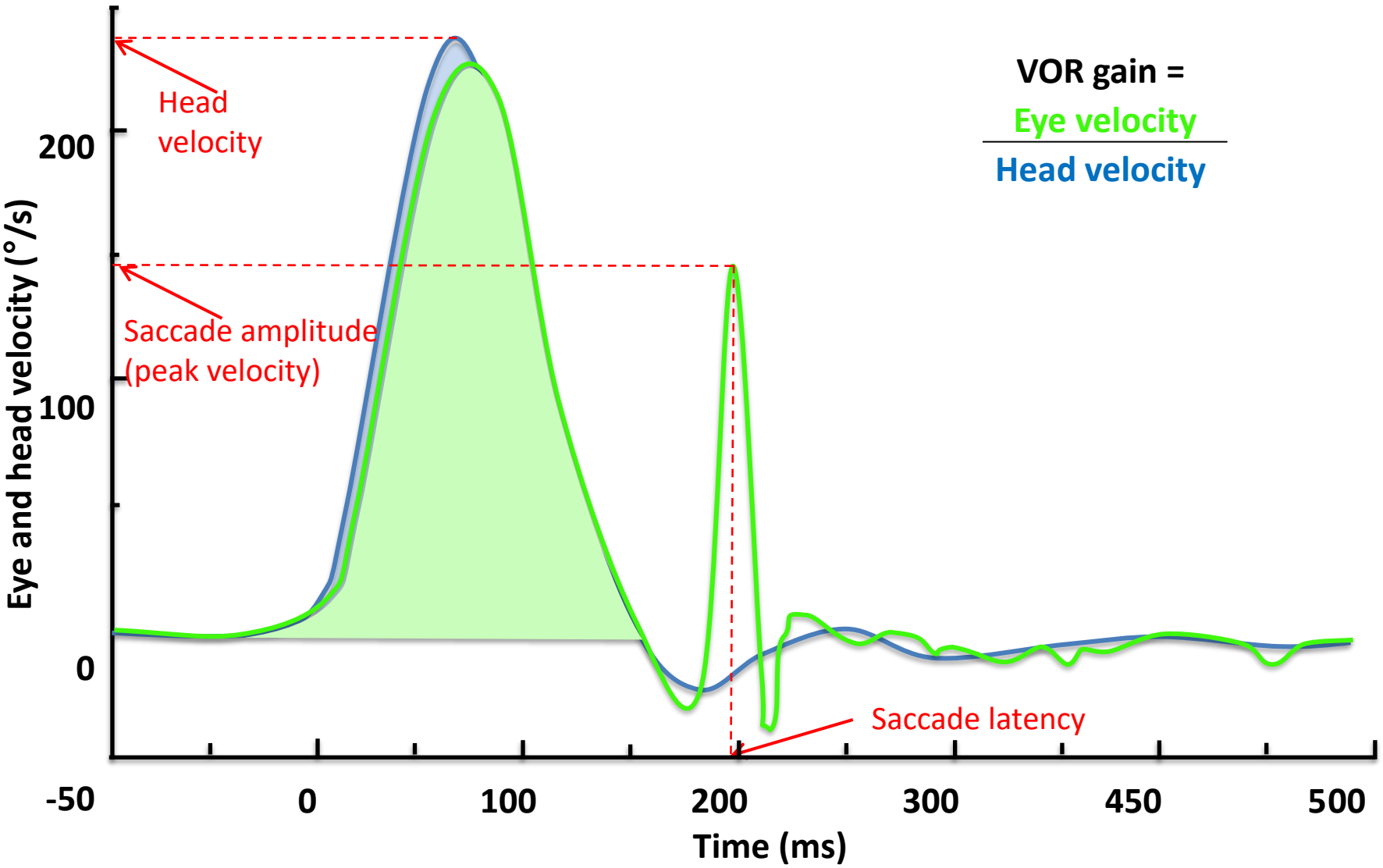
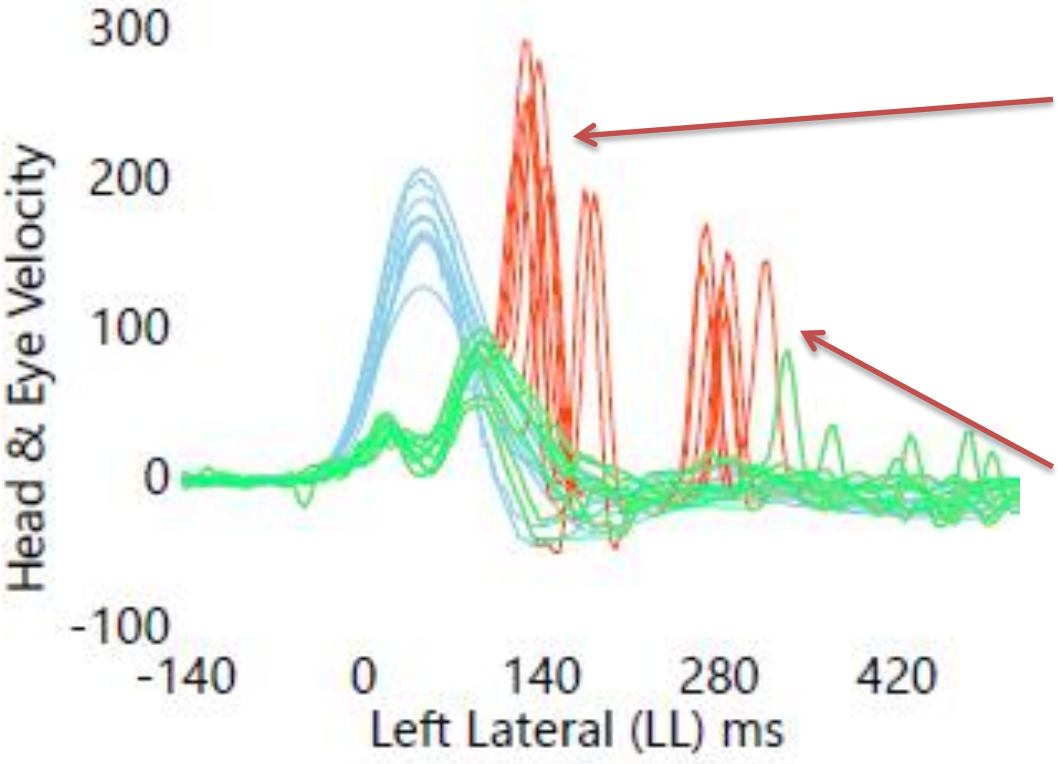
Typical vHIT trace



True “catch-up” saccades



“Covert” saccades

- Not visible with the naked eye
- Occur while head is still moving (before head trace crosses the x-axis)
- Non-visual, occur before retinal error signal has been processed by visual cortex
- Pre-programmed by cerebellum

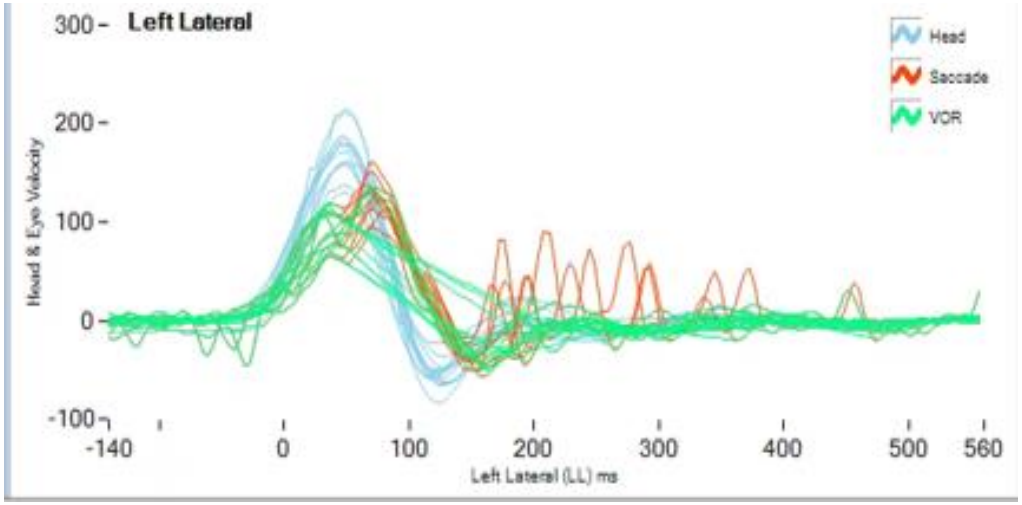
“Overt” saccades

- Visible with the naked eye
- Occur after head trace crosses the x-axis
- Visually driven

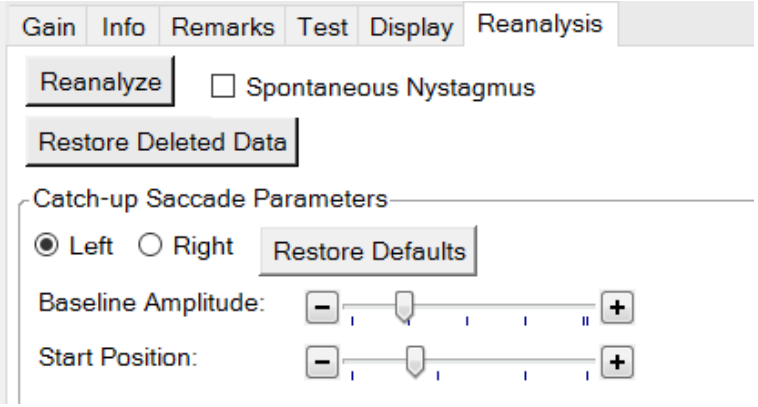
- Guidance around what constitutes genuine saccades has been fairly vague in the past, i.e. must be *“large’”* or *“at least as big as the head velocity”* or *“must occur on the majority of traces”*
- Rambold (2016) found that the borderline between non-pathological saccades seen in normal individuals and pathological saccades seen in vestibular patients is an **amplitude** (peak velocity) **of 110°/s**

Editing saccades

In otosuiteV, saccade detection algorithm good but does make mistakes



Use reanalysis tab to edit what is classed as a saccade



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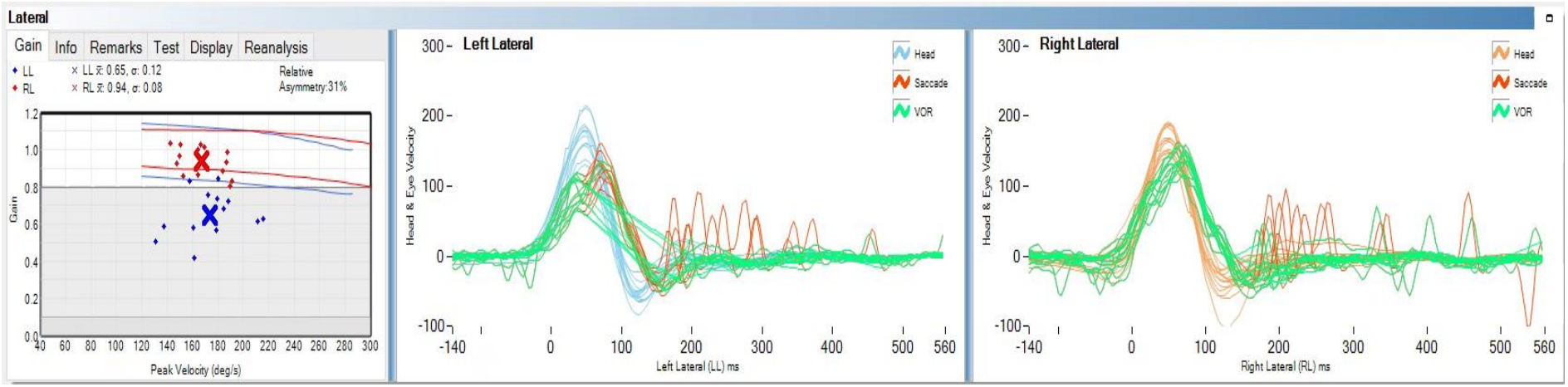
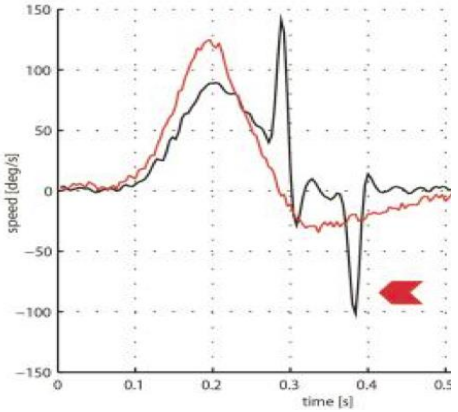
Editing saccades



False generators of saccades

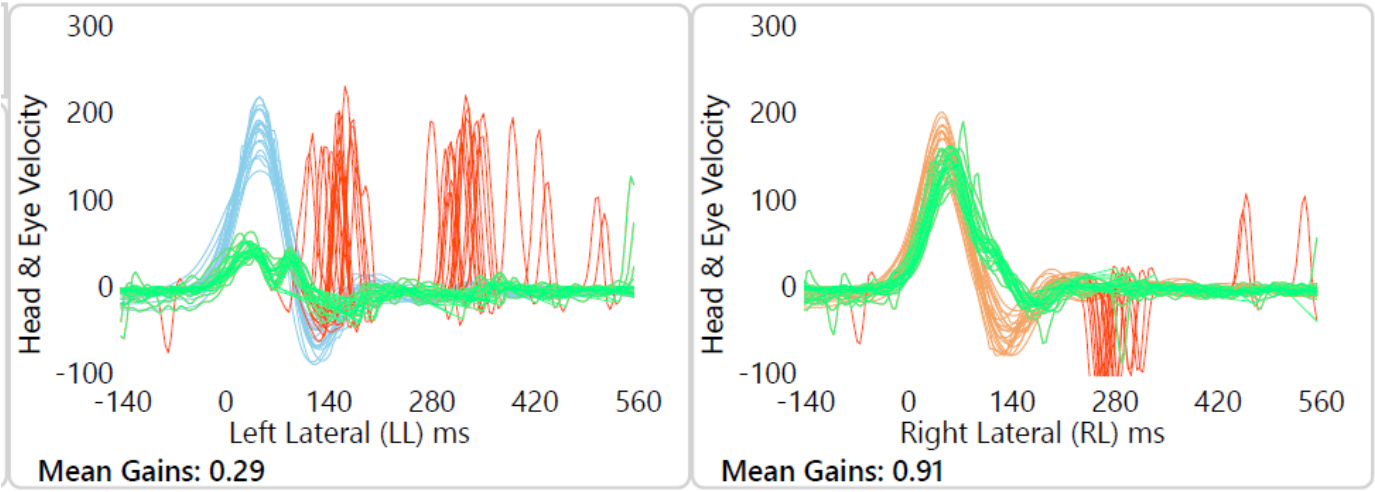


Refixations

- When patients eyes are moving around inside the target or close to it, these tend to bi-directional – Reinstruct to keep eyes on target!

Excessive rebound

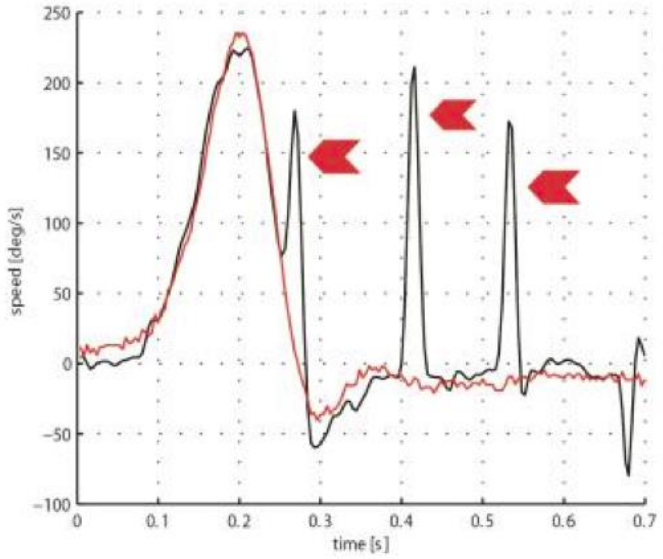
- If head does not come to a sudden stop, and there is rebound in the opposite direction, this is akin to an impulse to the contralateral side
- If the contralateral side has dysfunction, you may see some saccades

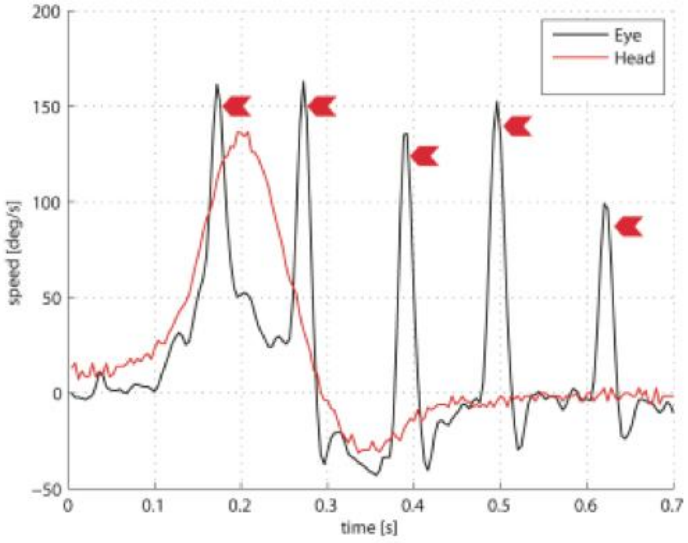


False generators of saccades

Spontaneous nystagmus

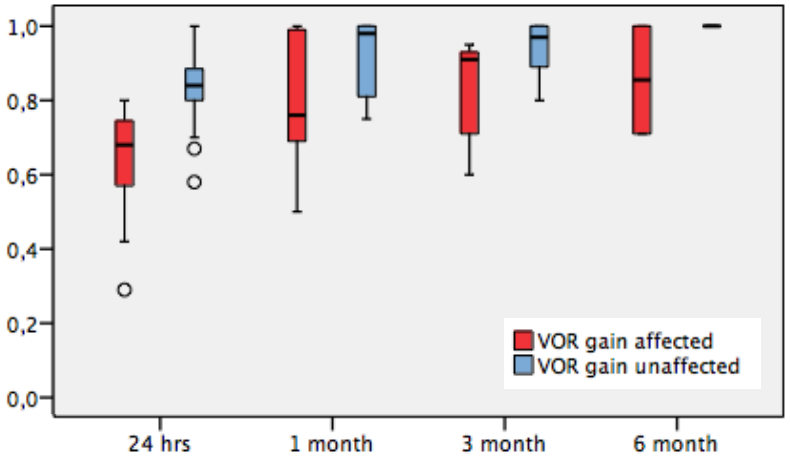
- Can look just like saccades, could be in either direction
- Will appear at regularly spaced intervals, with consistent amplitude
- If present in central gaze (i.e. congenital nystagmus)
  - Will appear in the live trace
  - May appear in vHIT trace before start of head movement
- If present in eccentric gaze only (i.e. sustained, gaze-evoked central nystagmus or large end-point nystagmus)
  - May not appear before start of head movement
- Always do bedside eye exam before vHIT!



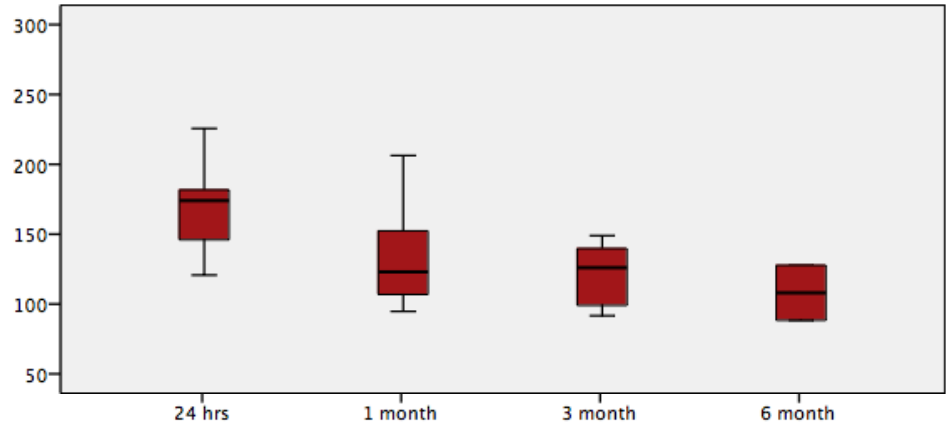


Effects of central compensation on saccades

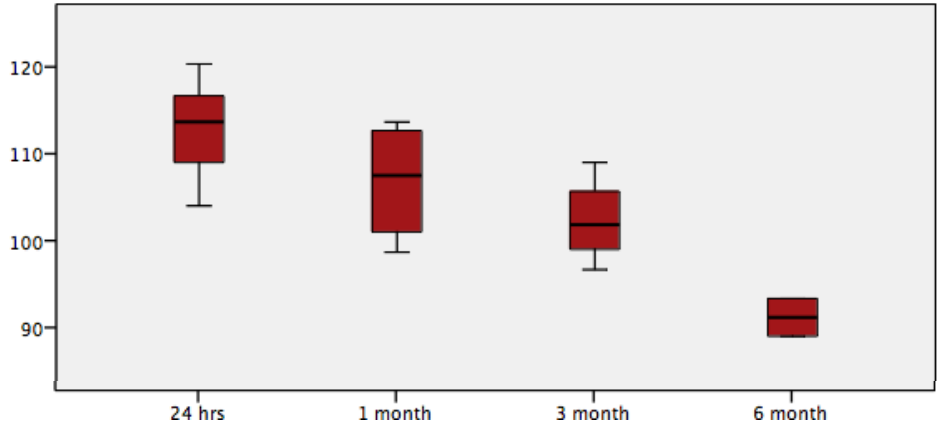
18 vestibular neuritis patients (Martin-Sanz *et al.,* 2017)



Gain over time



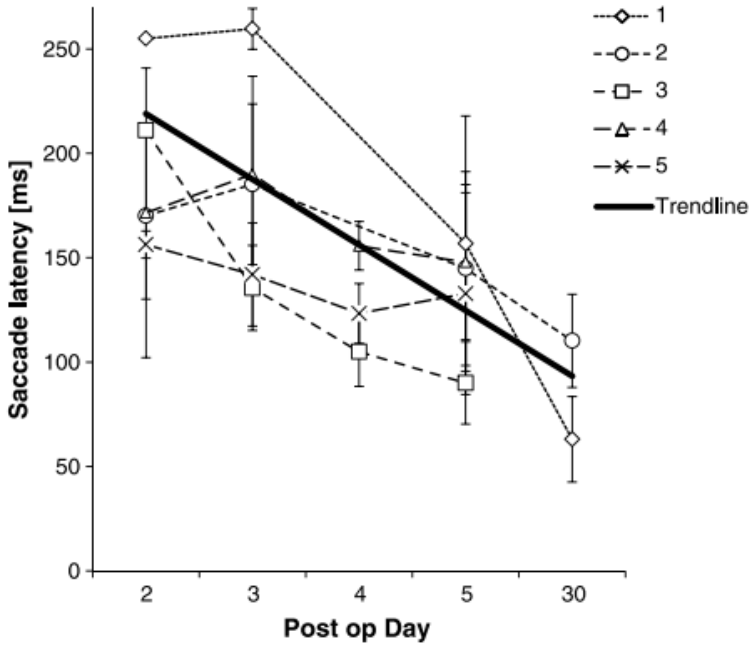
Saccade amplitude (peak velocity) over time

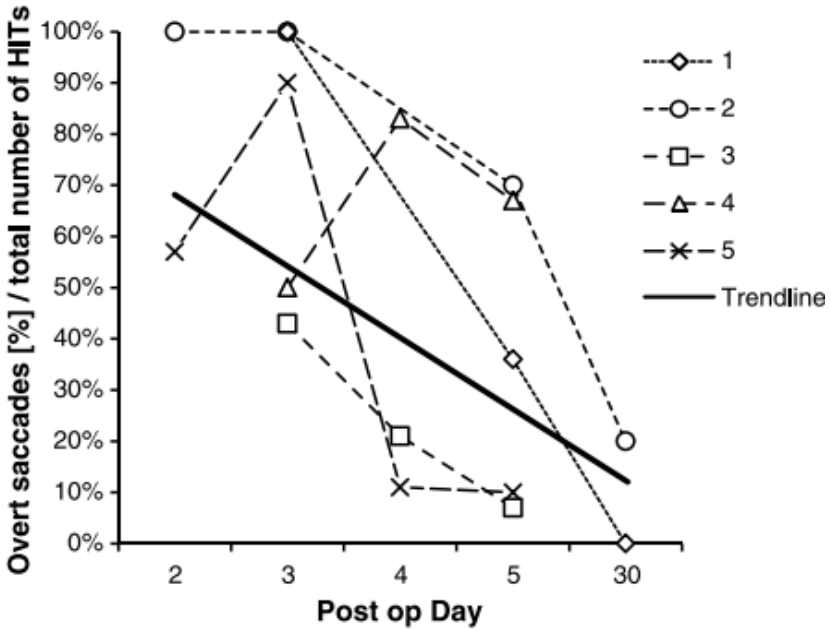


Saccade latency over time

Effects of central compensation on saccades

Five patients with unilateral vestibular deafferentation after vestibular schwannoma resection (Mantokoudis *et al.*, 2013)





"Compensatory saccade latency shortens over time, and corrective eye movements may only be overt (visible by the naked eye) during the first 3 days after deafferentation"

Similar findings in (Mantokoudis et al., 2016). 'Adaptation and Compensation of Vestibular Responses Following Superior Canal Dehiscence Surgery'.

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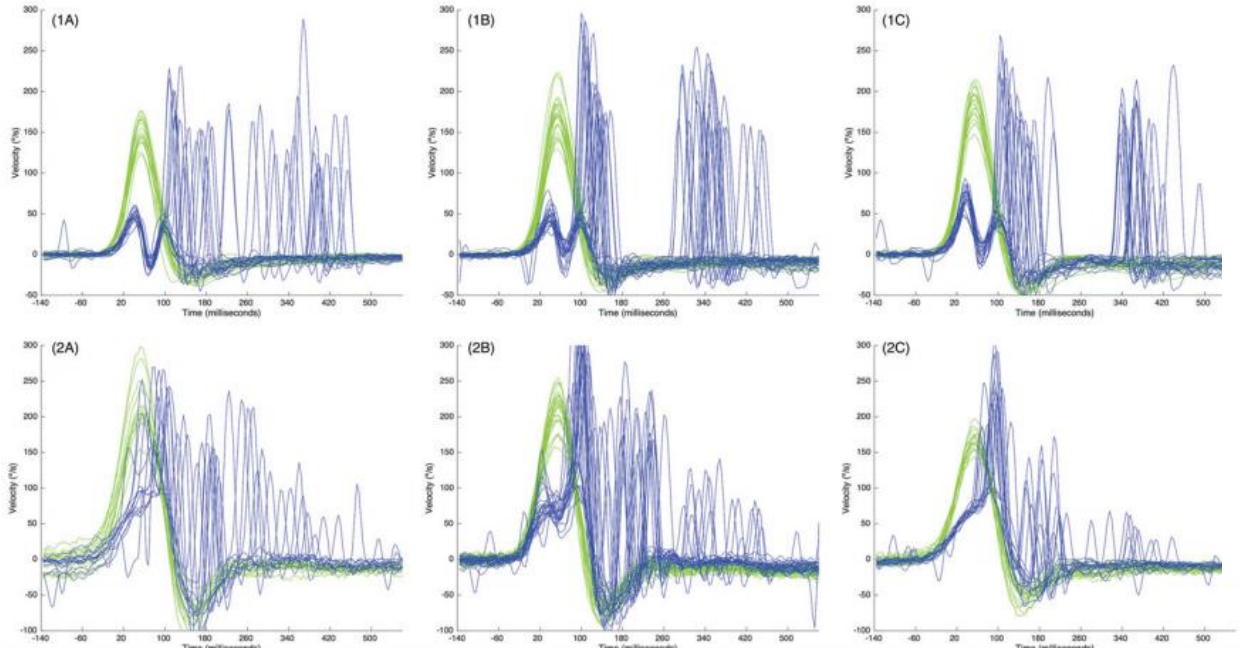
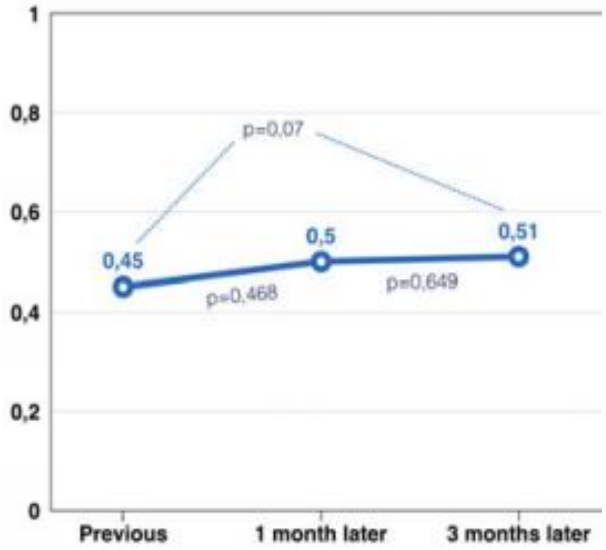
Effects of central compensation on saccades

16 patients with unilateral vestibular loss before vestibular rehabilitation (A), at 1 month (B) and 3 months (C) (Matiñó-Soler *et al.,* 2016)

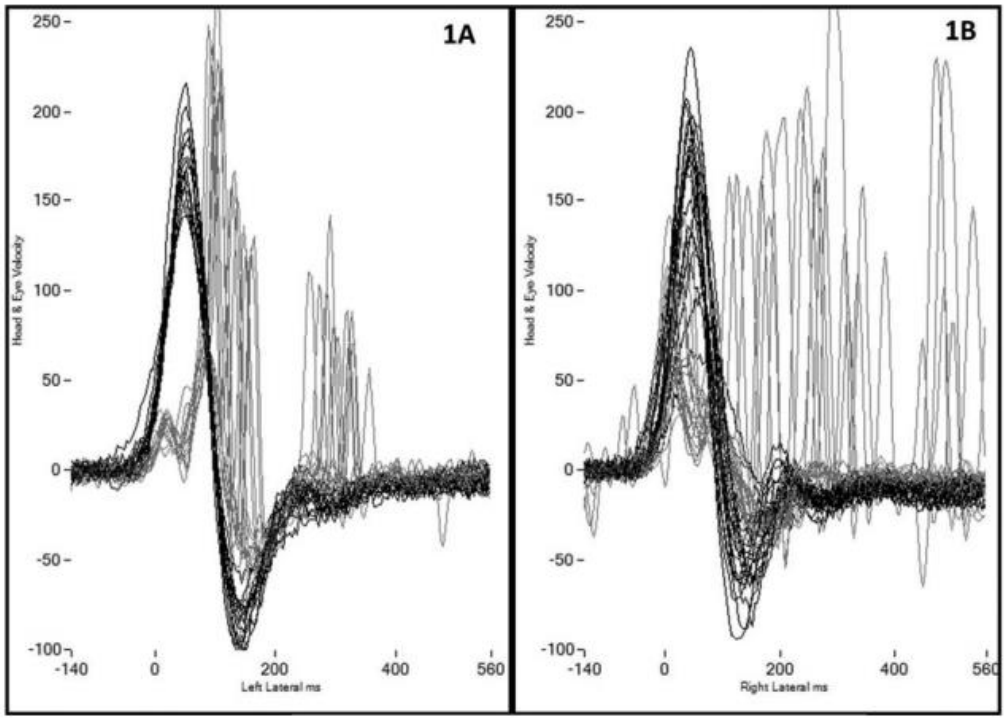




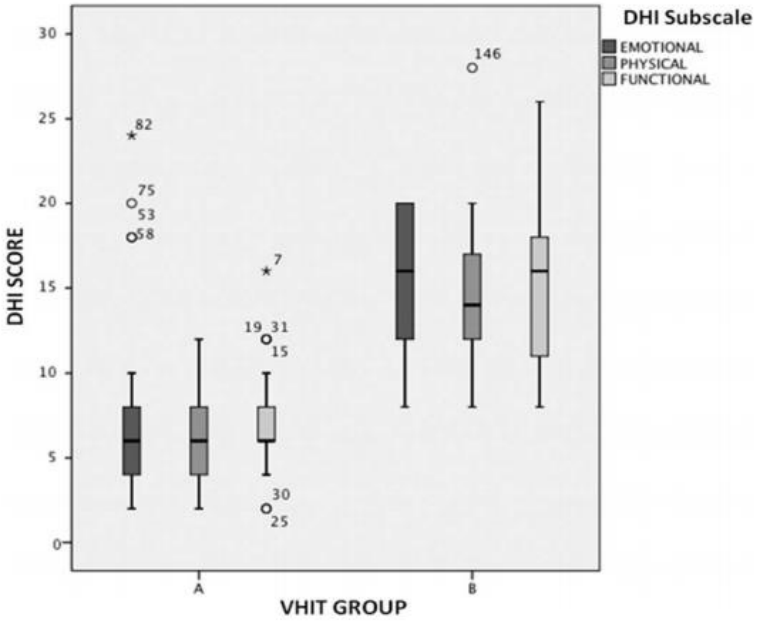
While gain did not significantly recover, overt-catch up saccades decreased in number and amplitude, <u>became more clustered</u>, and progressively reduced in latency becoming covert

Saccades and subjective symptoms

- 49 patients with complete unilateral deafferentation one year after surgery
- Significant correlations between scattering of saccades and subjective symptoms as measured by the DHI. (Batuecas-Caletrio *et al.,* 2013).



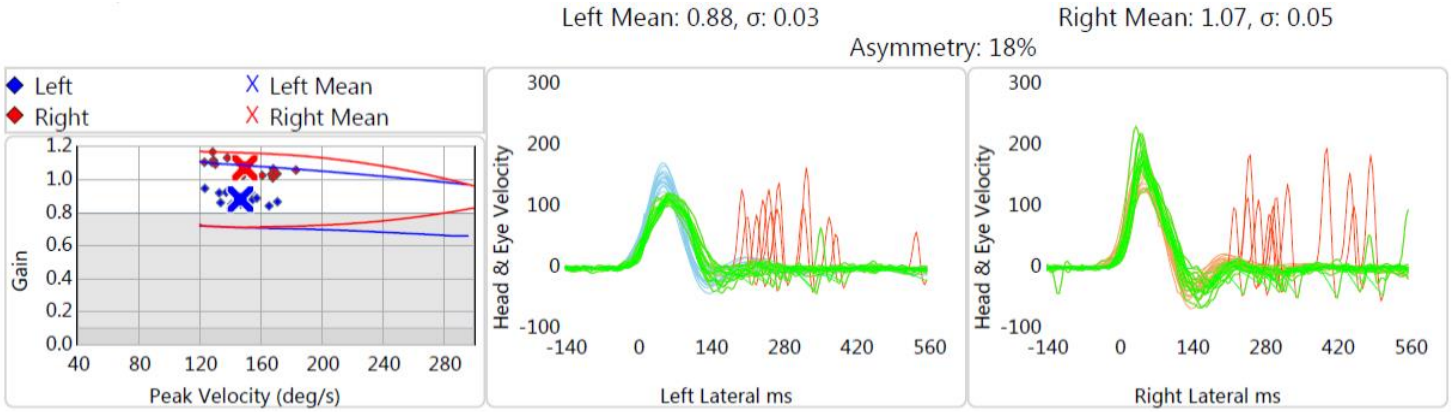
Group A = More gathered/clustered/organised
Group B = Less gathered/clustered/organised



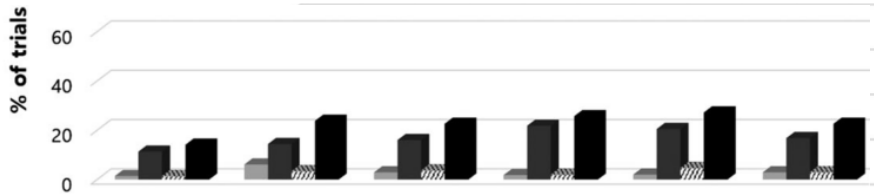
A greater degree of scatter correlated with worse subjective report

Think about covert saccades as **clues** to an <u>old, probably well compensated injury</u>

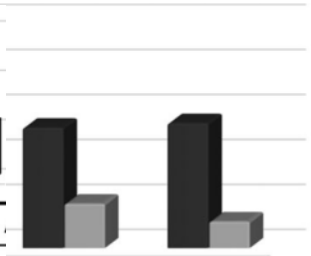
Non-pathological / age-related saccades



- Smaller, non-pathological saccades are a normal finding in heathy individuals with no relevant clinical history and vHIT gains within the normal range (Matino-Soler *et al.*, 2015; Yang *et al.*, 2015; Anson *et al.*, 2016; Anson *et al.*, 2016; Rambold, 2016).
- There have been significant correlations shown between age and saccade incidence, and between age and mean saccade amplitude in all of these studies



	20's	30's	40's	50's	60's	Total
Isolated covert CS	1.4	6.1	2.9	1.8	2.0	**2.8**
Isolated overt CS	11.3	14.4	16.0	21.8	20.5	**16.8**
Combined covert & overt CS	1.4	3.4	3.7	2.0	4.7	**3.0**
Total	**14.1**	**23.8**	**22.6**	**25.7**	**27.2**	**22.6**



60's	Total	*P*-value*
53.5±22.0 (25–100)	**55.5±16.9 (20–100)**	0.392
19.7±13.4 (5–40)	**11.8±10.7 (0–40)**	0.275

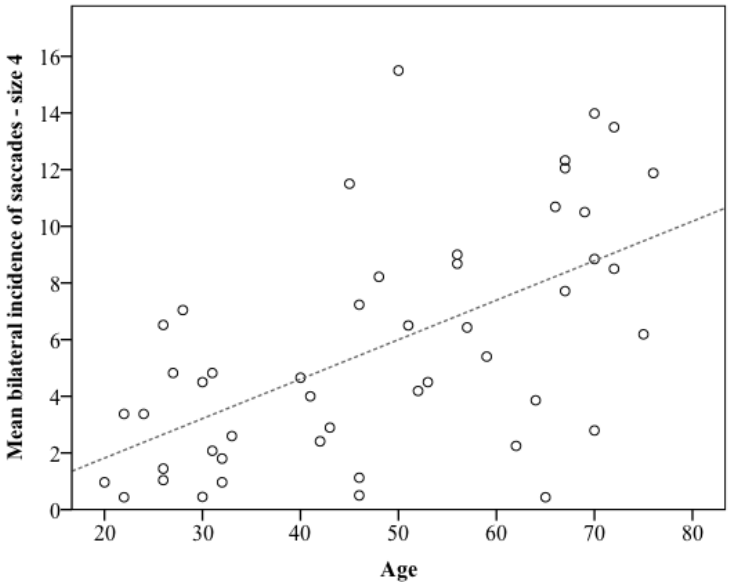
From Yang *et al.*, (2015)

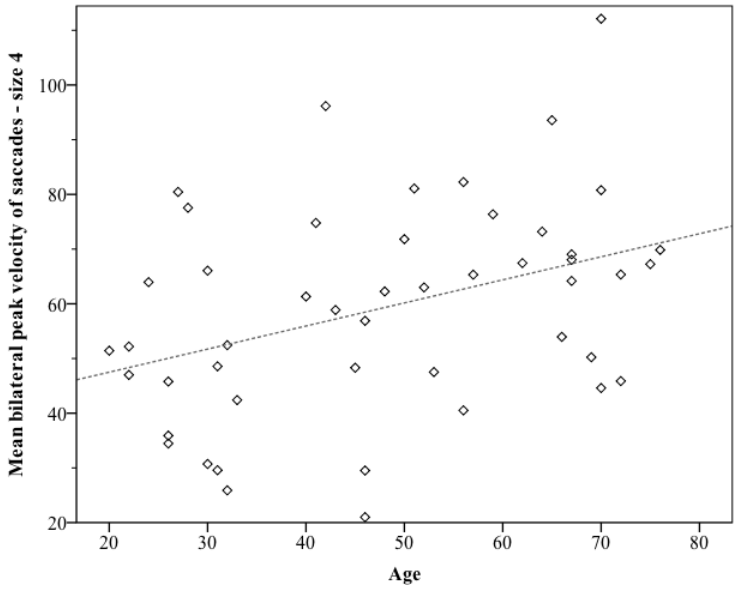
Non-pathological / age-related saccades

Same trends found in our study (Jay, Cane and Howe, 2018).

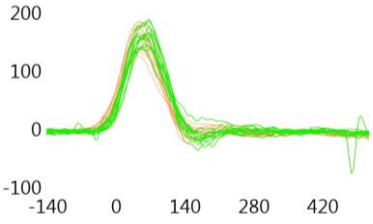
As participants became older, the amount of saccades produced and their size in the vHIT trace increased.

(No correlation between age and saccadic latency).



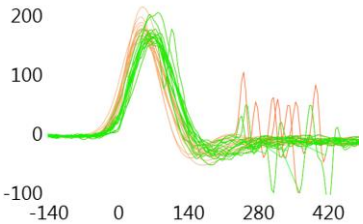


Gain (SD): 1.02 (0.03)



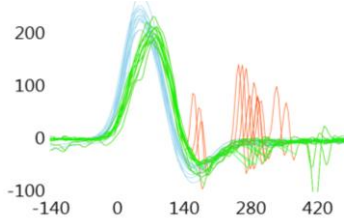
Age: 20

Gain (SD): 0.99 (0.03)



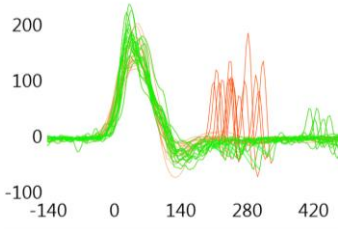
Age: 42

Gain (SD): 0.88 (0.03)



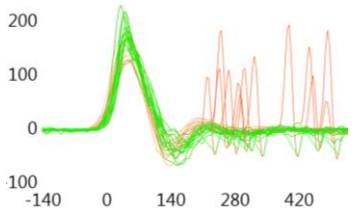
Age: 50

Gain (SD): 1.04 (0.05)



Age: 70

Gain (SD): 1.07 (0.05)



Age: 71

Non-pathological / age-related saccades

- Mean bilateral saccade amplitude across cohort was 59.31°/s (SD 19.24). Two standard deviations from this value gives us an upper limit for the normal range of 97.79°/s.
- However, as older participants made saccades with larger amplitudes, an upper limit of the normal range for those aged between 68 and 77 years of age is actually 110.51°/s, agreeing with the findings of Rambold (2016).
- Non-pathological saccades are common in vHIT, especially in older individuals, and care should be taken during interpretation of vHIT saccade profiles so as to avoid false positive results, especially when non-pathological saccades are seen in patients prone to sources of error which may lower gain.
- If such saccades are common in normal individuals, they may also be seen in individuals with vestibular dysfunction *as well as* larger 'catch-up' saccades

Non-pathological / age-related saccades

What causes these and why are they more common in older people?

- Increased 'fidgeting' of eyes in older people, though fixation stability has been shown to be unaffected by age (Gottlob, Fillmore and Abroms, 2007; Ridderinkhof and Wijnen, 2011).
- A declining ability to suppress reflexive saccades to the goggle frame or nose entering the visual periphery (Rambold , 2016)
- A non-vestibular effect of aging on the central nervous system that is not currently understood by the literature
- They are more frequent and of greater amplitude with lower gains *within the normal range* so might represent the effect of aging on the vestibular system (Presbystais) (Anson *et al.,* 2016)
    - If the literature is beginning to establish that gain can recover; these saccades might represent old 'scars 'of the gradual bilateral aging process of the vestibular system.

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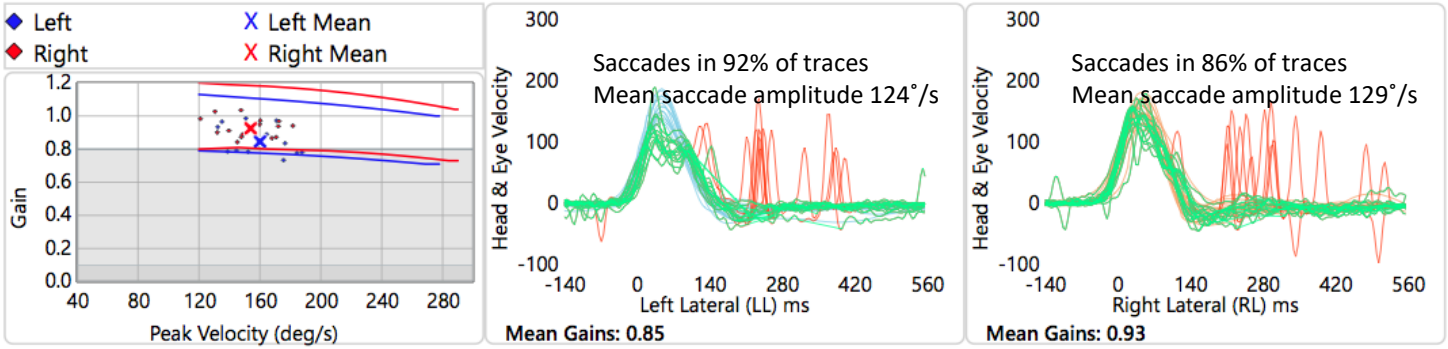
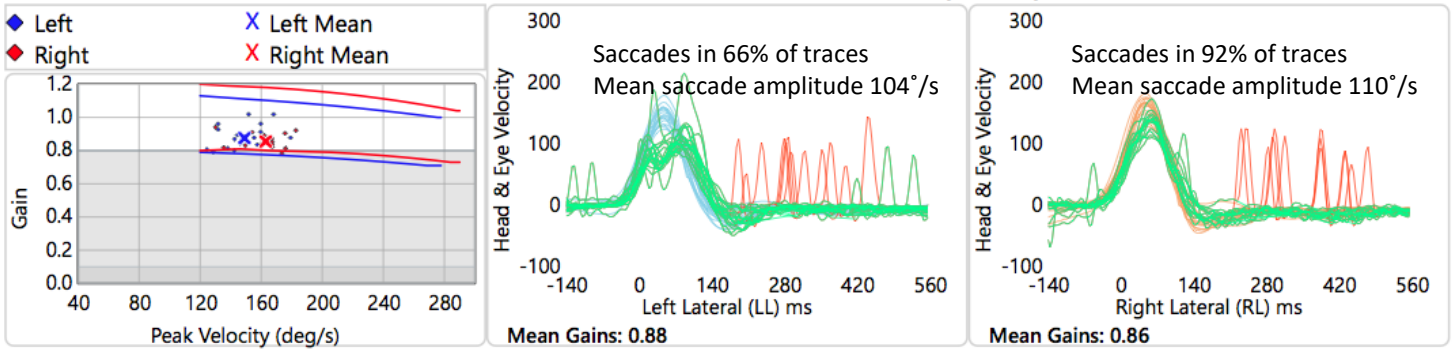
Saccades with normal gain

Case: 81 year old woman with non-specific generalised imbalance, no history of TRV, no BPPV, no other remarkable medical history. No VFT performed. Tailored vestibular rehabilitation.

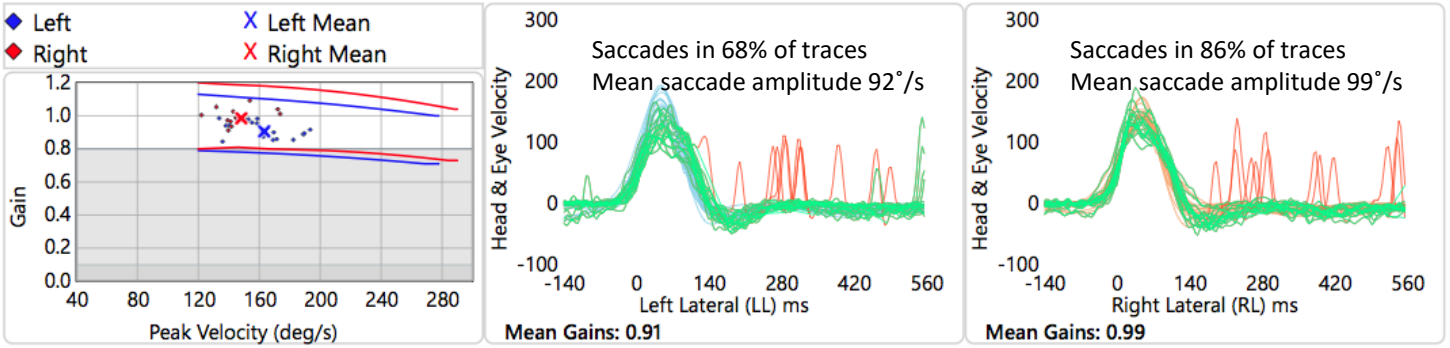
Initial assessment



+11 weeks



+16 weeks



Questions still to answer

- What is restoring VOR gain and curve morphology in some people?
  - Neck cues / COR?
  - Regeneration of sensory hair cells?
- What is causing the saccades seen in older people?
  - If not vestibular, can we eliminate these somehow by changing the methodology?
- What can we use SHIMP for?
- Anterior canal sparing?
- Bilateral posterior canal deficits?

Anterior canal sparing?

› Clin Neurophysiol. 2016 Aug;127(8):2791-2801. doi: 10.1016/j.clinph.2016.05.005. Epub 2016 May 24.

Disease-specific sparing of the anterior semicircular canals in bilateral vestibulopathy

Alexander A Tarnutzer [1], Christopher J Bockisch [2], Elena Buffone [3], Stefan Weiler [4], Lucas M Bachmann [5], Konrad P Weber [6]

- Retrospective case series
- Seen in patients with:
  - Aminoglycoside-associated BVH
  - Idiopathic BVH
  - Ménière's disease

› Clin Neurophysiol. 2016 Aug;127(8):2791-2801. doi: 10.1016/j.clinph.2016.05.005.
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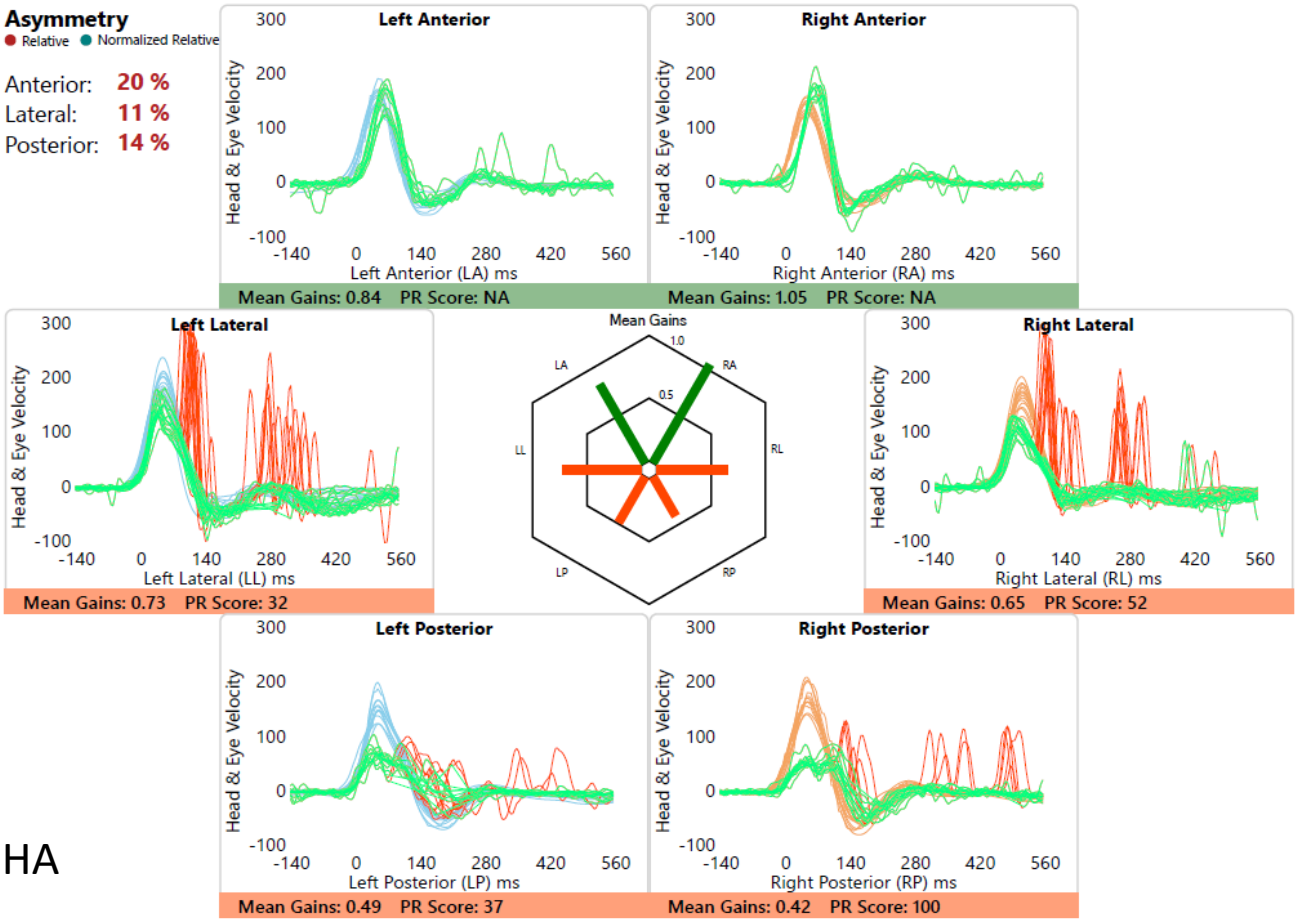

Idiopathic BVH confirmed by SHA

› Clin Neurophysiol. 2016 Aug;127(8):2791-2801. doi: 10.1016/j.clinph.2016.05.005.
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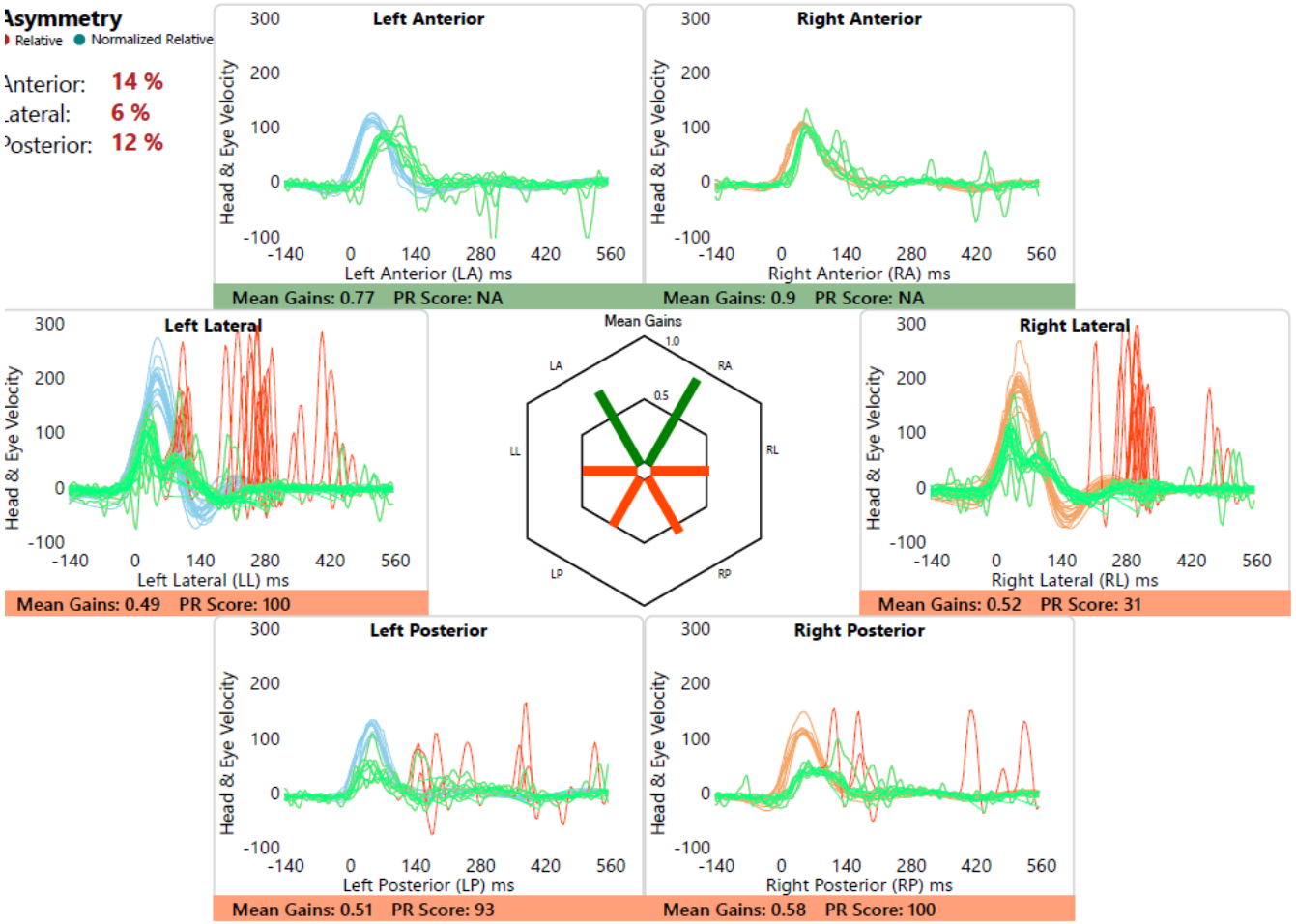

Post-gentamicin

Journal of Neurology (2020) 267:2347–2352
https://doi.org/10.1007/s00415-020-09793-5

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ORIGINAL COMMUNICATION

Bilateral posterior semicircular canal dysfunction: a new finding with video head impulse test

Florencia Lerchundi[1] · Alfredo Hernan Laffue[1] · Marina Olivier[1] · Francisco Jose Gualtieri[1]

- Isolated deficits of the posterior canals (or inferior vestibular nerves?)
- Recent retrospective case series (n=41)
- Mostly chronic gait instability and disequilibrium, not TRV
- Peak prevalence at 71–80 years.
- Mostly idiopathic, often presenting together with presbycusis and positional DBN.
- Related to“presbyastasis” or disequilibrium of aging?

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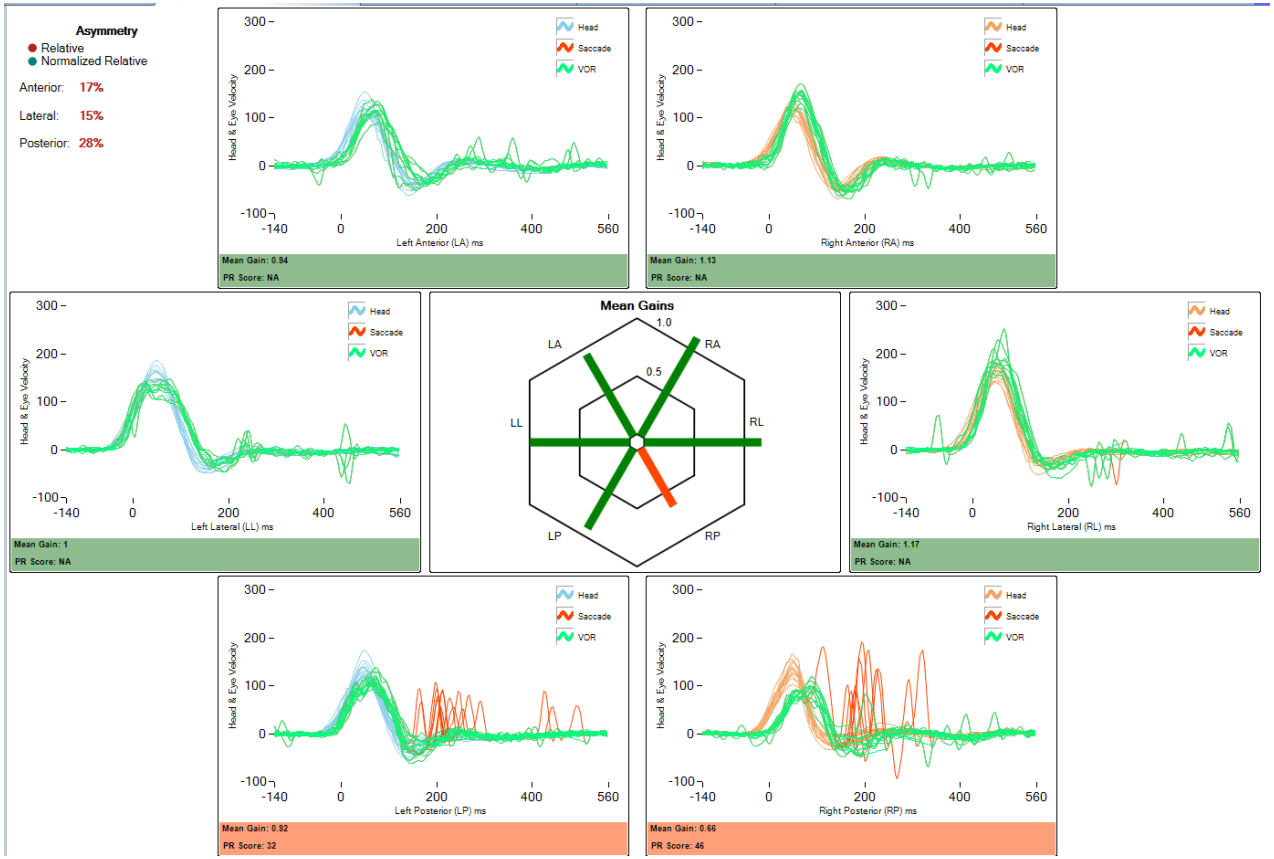

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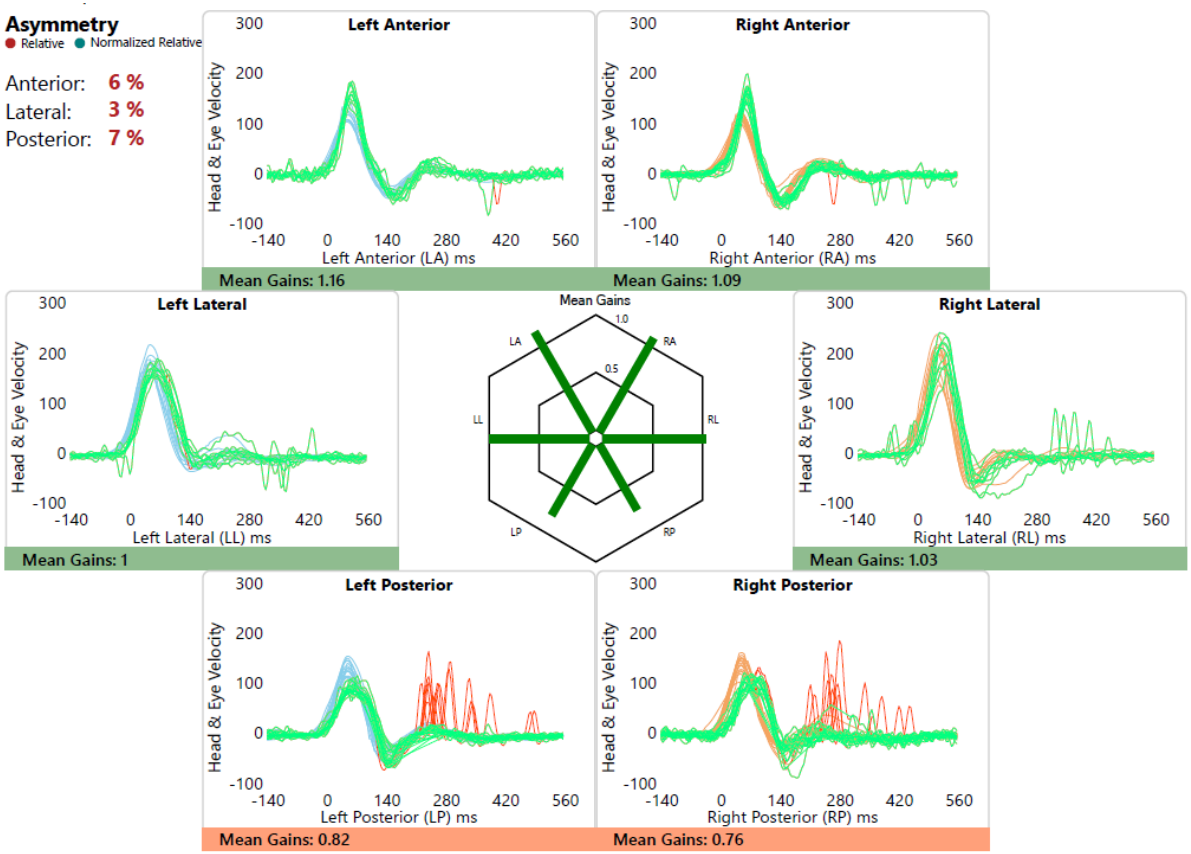


Questions?

More detail

Measurement of gain

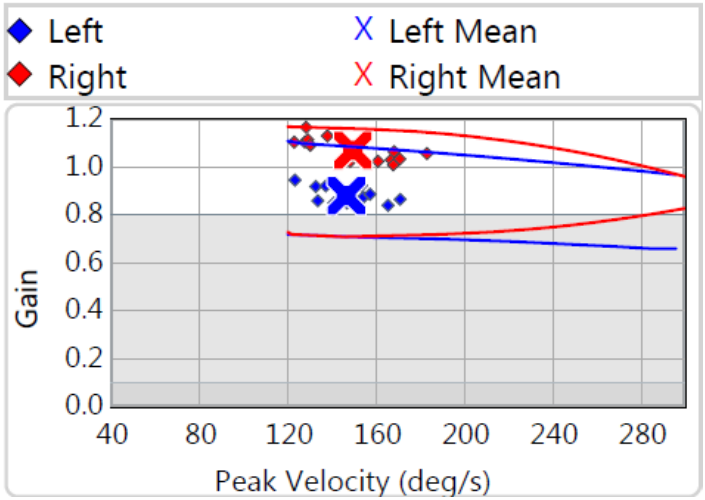
- There are significant differences in intra-subject VOR gain when measured by different devices (Cleworth *et al.*, 2017; Janky *et al.*, 2017).

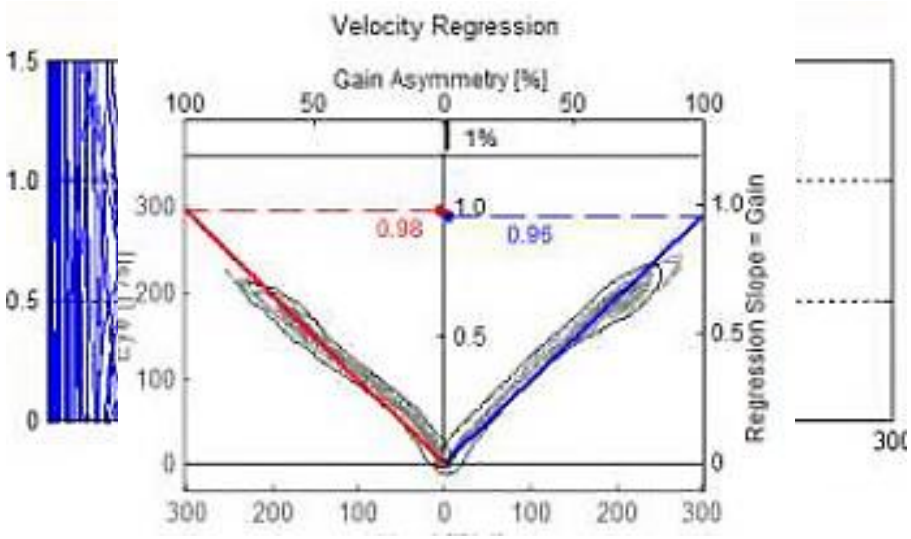
ICS Impulse

- 'Area under the curve' method
- Bilateral average normal gain range of 0.8 – 1.2
- Ear-specific, age-stratified, head-velocity-specific normative ranges also shown (ages 10-99)

Eyeseecam

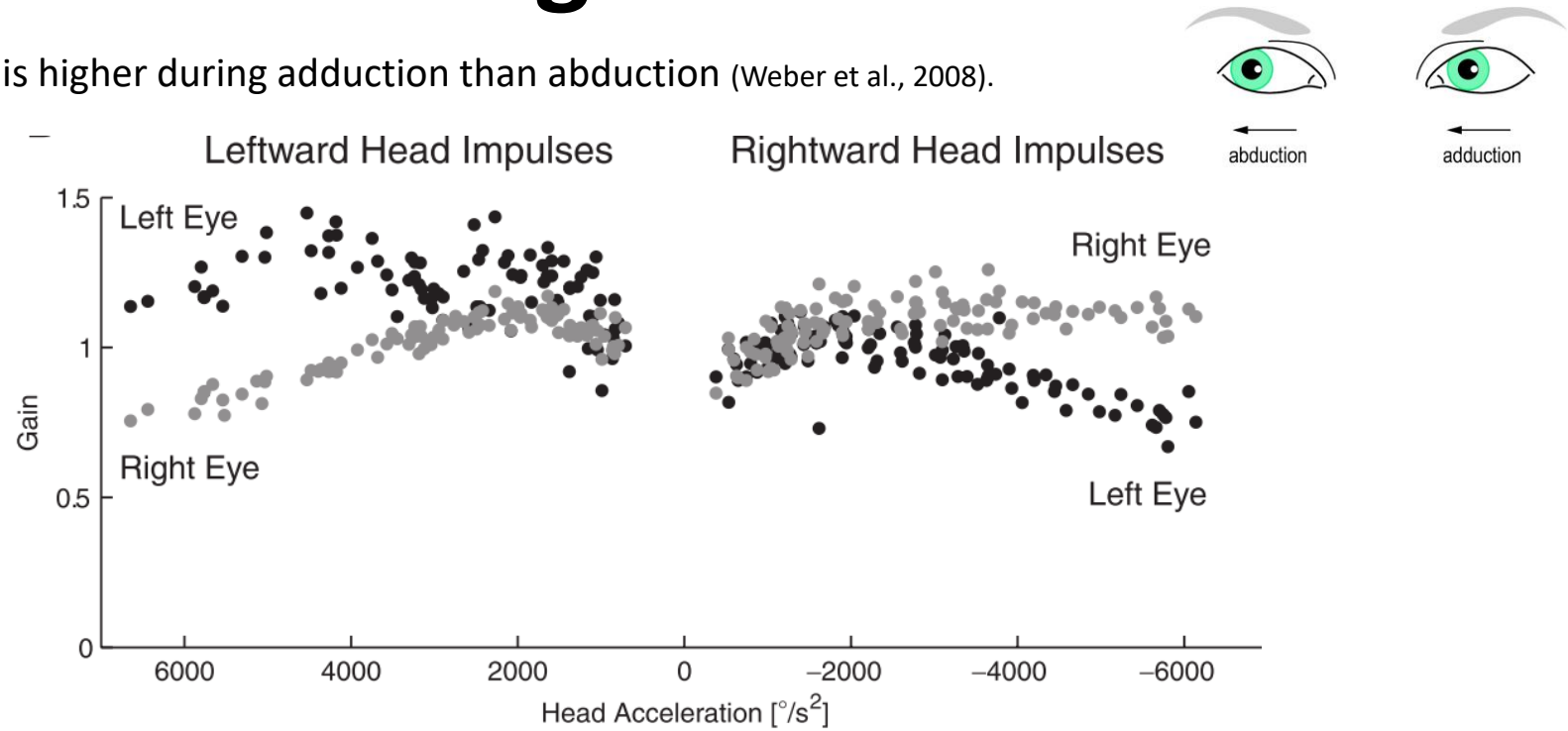
- 3 measures of instantaneous gain (40, 60 and 80ms)
- Easily skewed by artefact at moment of gain calculation (Macdougall *et al.*, 2013).
- Bilateral average normal gain range is 0.74-0.14 for 60ms and 0.79-1.15 for 80ms (Mossman *et al.*, 2015) (ages 20–80)
- Regression line less likely to be skewed by artefact or covert saccades, though no data for normative ranges.





Inter-ocular gain differences

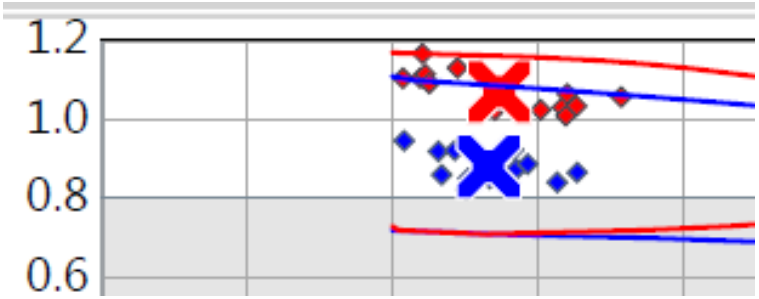
- VOR gain is higher during adduction than abduction (Weber et al., 2008).



- This has been suggested to be due to:
  - Geometry: The globe is not located along the axis of rotation of the head and rotates freely to one side of this axis. (McGarvie et al., 2015)
  - Differences in the neural pathways and dynamics of the yoked medial and lateral rectus eye muscles (Weber et al., 2008)
- When recording monocularly, the recorded eye adducts on impulses towards that side, meaning gains of the recorded eye will always be higher than the eye which is not recorded.
- Recording from right eye: **right gains tend to exceed left gains.**

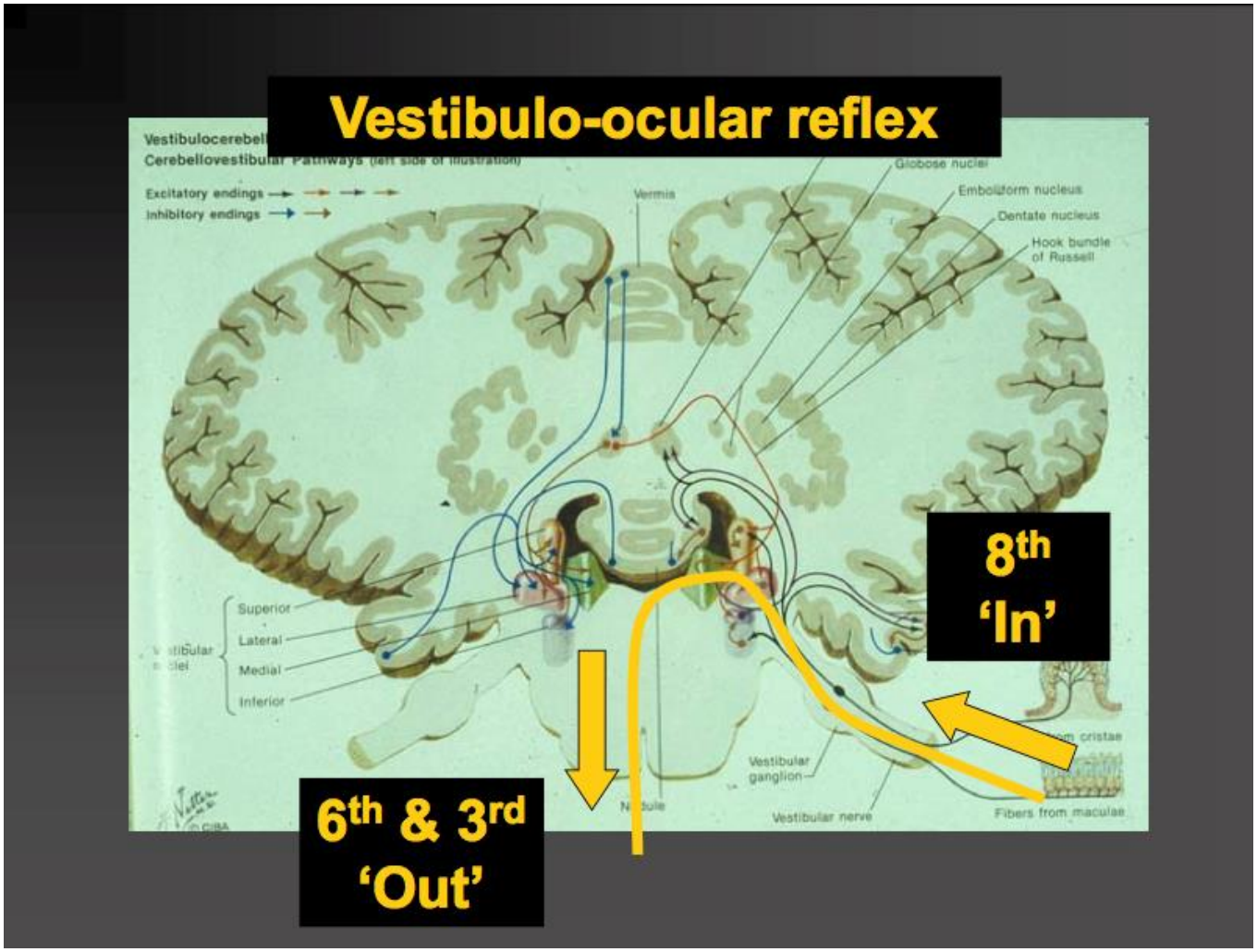
Asymmetry

- Normal percentage difference between horizontal gain values could be between 9-15%
  (Curthoys *et al.*, 2008; Weber *et al.*, 2008; Matiño-Soler *et al.*, 2015).



- Various methods of calculating this
- This metric is not a focus of the vHIT literature, and has limited clinical value compared with absolute gain values and saccade metrics.
- Compared with calorics, the size of the vHIT normal range is much tighter, therefore comparisons between ears become less relevant than absolute gain values and saccade characteristics (Curthoys *et al.*, 2008).

vHIT tests the high frequency VOR



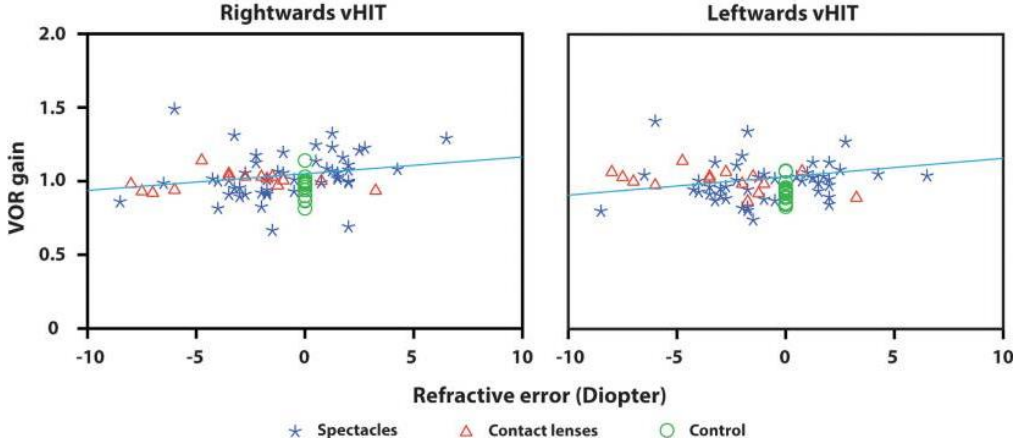
Patient-specific factors that may artificially alter gain

Visual acuity

- During the time period during which gain is calculated (<150ms), VOR is not visually driven and therefore the gain value should not be affected by poorer visual acuity, though saccades might be if vision is very poor.

Refraction

- Does adaptation of the VOR to strong glasses prescriptions artificially raise gain, even when the glasses are not on?
- Older rotatory chair studies suggested that VOR adapts to move the eye further underneath the prismatic effect of refractive glasses (Gonshor and Jones, 1976; Cannon *et al.*, 1985).
- A more recent study suggested that re-adaptation can occur very quickly after removal of glasses and that vHIT gain should not be affected (Van Dooren *et al.*, 2018).



Patient-specific factors that may artificially alter gain

Drug intake

- Pre-vHIT studies:
  - Betahisthine reduces VOR gain in ?episodic patients (n=12) (Kingma et al., 1997)
  - Prochlorperazine unlikely to affect vestibular testing in normal subjects (n=12) (Patel et al., 2014)
- No vHIT study on Betahisthine or Prochlorperazine.
- Alcohol intake can lower horizontal vHIT gain and increase incidence and size of catch-up saccades (n=8) (Roth *et al.*, 2014)
- Effect of common antivertiginous agents on VOR gain in young, normal subjects (n=12) (Anagnostou et al., 2017).
  - An antihistamine (dimenhydrinate), a benzodiazepine (diazepam), and a calcium channel antagonist (cinnarizine).
  - None was found to significantly affect horizontal VOR gain
  - Saccades not considered
- Can findings in normal subjects be applied to patients with vestibular dysfunction?

Patient-specific factors that may artificially alter gain

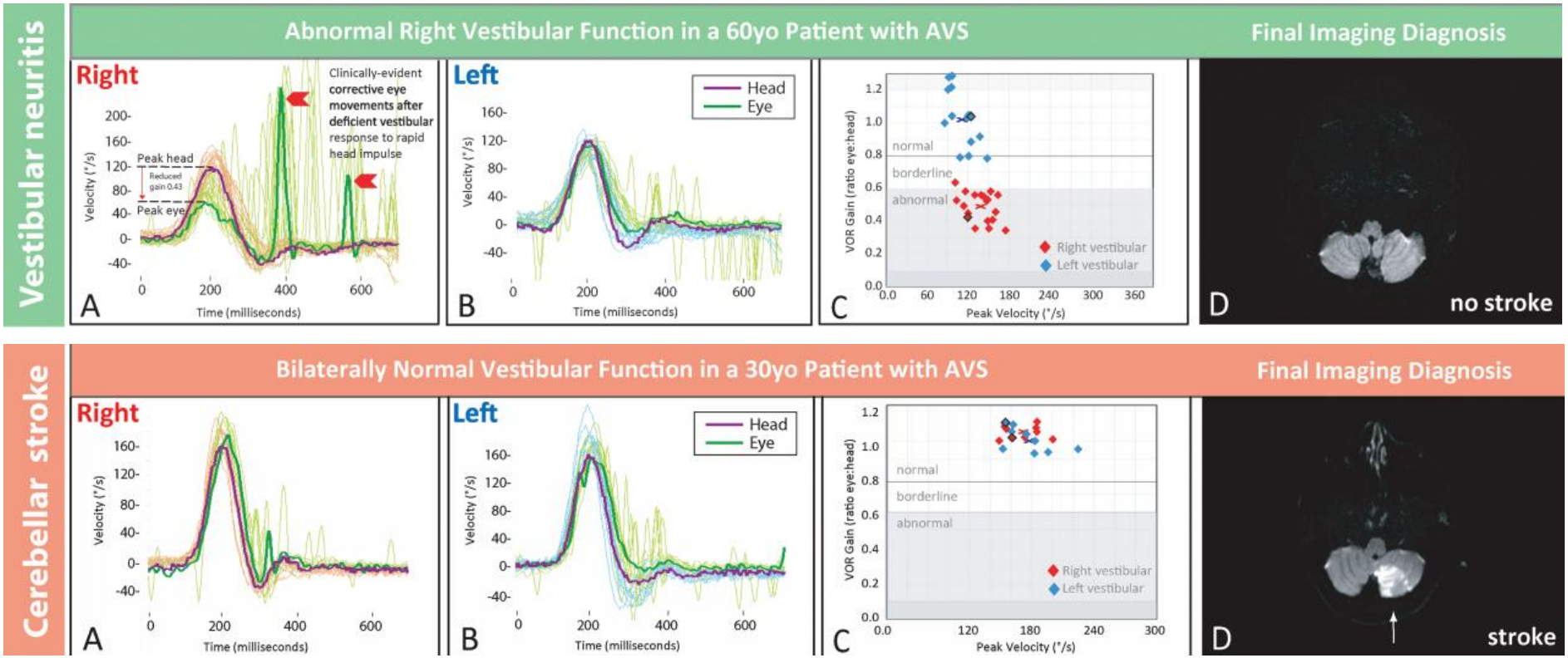
Ocular misalignment / strabismus

- No studies have looked at this
- We patch one eye to avoid saccades caused by changing fixation
  - Unilateral squints: Ideally the squinting eye should be patched
  - If bilateral squint, or squinting eye is only one that can be recorded, patch the other eye: important thing is to keep one eye fixing
- When it is only possible to record from a strabismic eye
  - Make sure the pupil is located in the centre of the orbit.
  - If eye is allowed to drift into eccentric gaze where movement is limited, it may be receiving VOR commands but is not able to move through the full VOR curve. This might theoretically lower the gain.
  - Similar effects might be seen in paralytic squints due to nerve palsies in CN III, IV and VI.
- For these reasons it is essential to perform bedside eye examination before vHIT, including cover/uncover and assessment of ocular range of movement.

Patient-specific factors that may artificially alter gain

Central vestibular dysfunction - Acute

- Growing body of work on use of head impulse in acutely dizzy patients to differentiate peripheral from central vestibular dizziness (acute vestibular syndrome / AVS) using HINTS protocol (Newman-Toker *et al.,* 2015)



Essentially, vHIT will be normal in acutely dizzy patient having a stroke in the posterior fossa, because VOR does not pass through the cerebellum. However, VOR can rarely be damaged by small strokes affecting the brainstem and blood supply to the labyrinth.

Patient-specific factors that may artificially alter gain

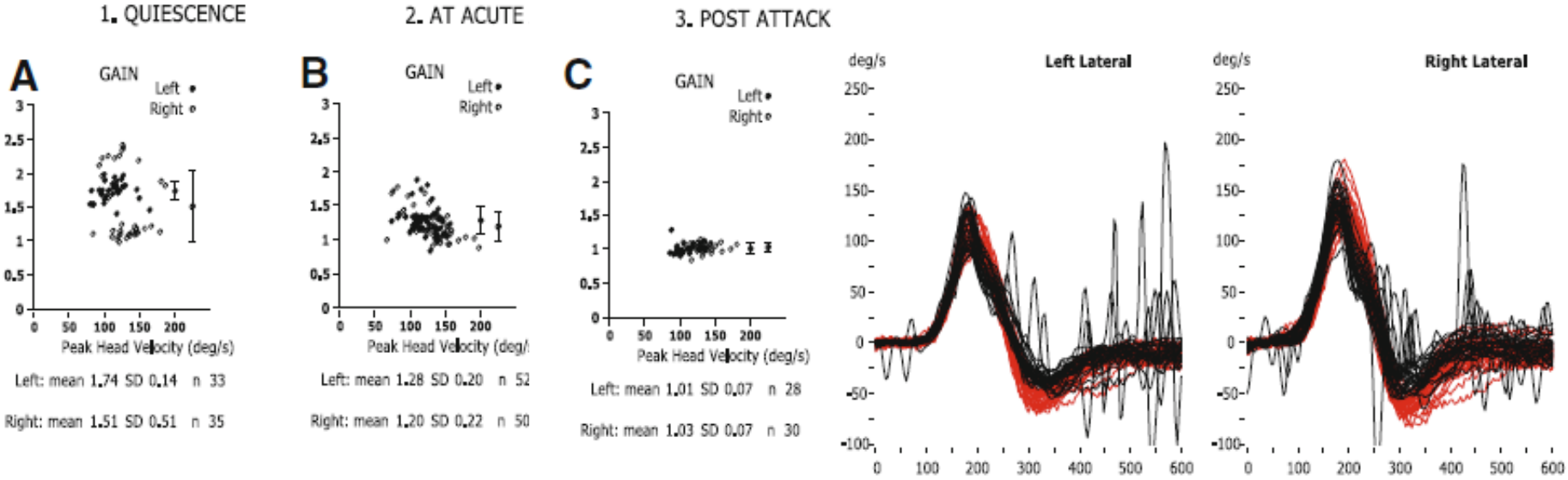
Central vestibular dysfunction - chronic

- You may see in textbooks that cerebellar disorders might increase *or* decrease VOR gain
- This appears to be based on rotatory chair studies:
  - Healthy individuals exposed to cerebellar-inhibiting drugs (n=6) (Shaikh *et al.*, 2013).
  - Animals studies when bilateral ablation of the flocculus and ventral paraflocculus can increase, decrease or have no effect at all on VOR gain (Takemori and Cohen, 1974; Zee et al., 1981; Lisberger et al., 1984; Rambold et al., 2002).
- There are a few more recent small studies using high frequency head impulses
  - Two case studies showing slight reductions in VOR gain on the opposite side of small, isolated infarctions of the cerebellar flocculus (Park *et al.*, 2013; Yacovino *et al.*, 2018)
  - Isolated VOR deficits in cerebellar ataxia patients (n=8) where all other vestibular testing was normal. Due to lesions in the flocculus? (Kremmyda *et al.*, 2012)
- Evidence is scanty for changes in high frequency VOR gain due to central lesions. Much more evidence from the HINTS literature that VOR tends to be is unaffected, even in the acute stage, in the majority of cases.

Patient-specific factors that may artificially alter gain

Meniere's disease?

- Suggestion that Meniere's disease might cause fluctuations in VOR gain.
  - Letter to the editor detailing six Meniere's patients with fluctuations in vHIT gain; hyperactive (≈1.5) during an inactive period, dropping during an attack (≈ 1.2), and returning to normal just after attack (≈ 1.0) (Manzari et al., 2011)
  - Two Meniere's patients with fluctuating and sometimes hyperactive VOR gain (Rey- Martinez *et al.,* 2018)
  - One patient with fluctuations in gain before and during attacks (Yacovino *et al.,* 2017)



"Increased endolymphatic volume could cause an increased effective pressure on the cupula of [a] hydropic horizontal semi-circular canal during a horizontal head impulse. This would produce an increased afferent vestibular signal depending directly on the endolymphatic hydrops magnitude"

But why no large saccades in the opposite direction?

Patient-specific factors that may alter gain

Central vestibular dysfunction - Acute

- Growing body of work on use of head impulse in acutely dizzy patients to differentiate peripheral from central vestibular dizziness using HINTS protocol (Newman-Toker *et al.,* 2015)
- Essentially, vHIT will be normal in acutely dizzy patient having a stroke in the posterior fossa, because VOR does **not pass through the cerebellum**. However, VOR can rarely be damaged by small strokes affecting the brainstem and blood supply to the labyrinth.

Central vestibular dysfunction - chronic

- You may see in textbooks that cerebellar disorders might increase *or* decrease VOR gain chronically, but this is based on old rotatory chair studies and not borne out in vHIT studies
- Evidence is scanty for changes in high frequency VOR gain due to central lesions. Much more evidence from the HINTS literature that VOR tends to be unaffected, even in the acute stage, in the majority of cases.
- Some peripheral vestibular weakness might have central source at level of vestibular nuclei?

Patient-specific factors that may alter gain

Meniere's disease?

- Fluctuating, sometimes hyperactive VOR gain right before an attack (Manzari et al., 2011; Rey- Martinez *et al.,* 2018; Yacovino *et al.,* 2017)
- Small case series, no compelling evidence yet – difficult to study
- In clinical experience Meniere's patients tend to have normal VHIT until well into the third stage

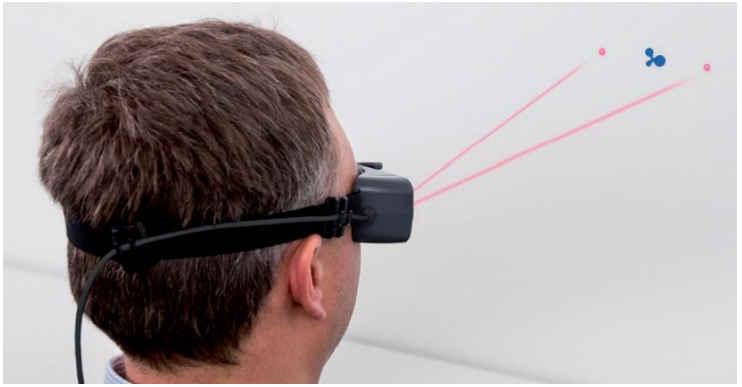
Patient-specific factors that may artificially alter gain

Visual acuity	Does not affect VOR curve or gain, but might affect saccades
Refraction	Recent study suggests that strong glasses prescription do not alter the VOR gain after glasses taken off (this was previously suggested)
Drug intake	Only small studies exist, suggesting: • Recent alcohol intake can lower horizontal vHIT gain and increase incidence and size of catch-up saccades (Roth *et al.*, 2014; Martelluci *et al.* 2021) • Dimenhydrinate, Diazepam and cinnarizine have no effect on VOR gain in healthy individuals, saccades not considered (n=12) (Anagnostou et al., 2017) • No vHIT study on Betahisthine (previously suggested to affect VOR gain) or Prochlorperazine (previously found not to)
Squint	In theory, squint, oculomotor nerve palsies or ophthalmoplegia could affect VOR gain. • Always do bedside eye exam first • Patch the eye not being recorded to stop re-fixation • Move head to keep pupil in centre of orbit if having to record from a strabismic eye

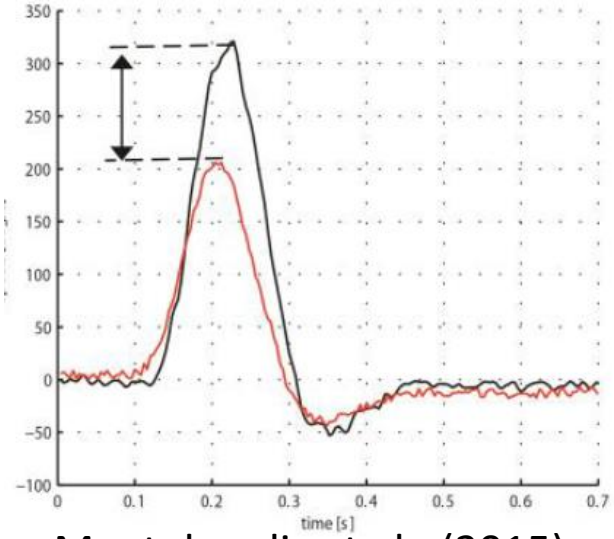
Technical factors that may artificially alter gain

Calibration

- How many pixels in video image equal a known visual angle 15° (on the back of the eye)
- Varies with anatomy



- If incorrectly performed, can have a significant effect on *how far the software thinks the eye moved* during VOR



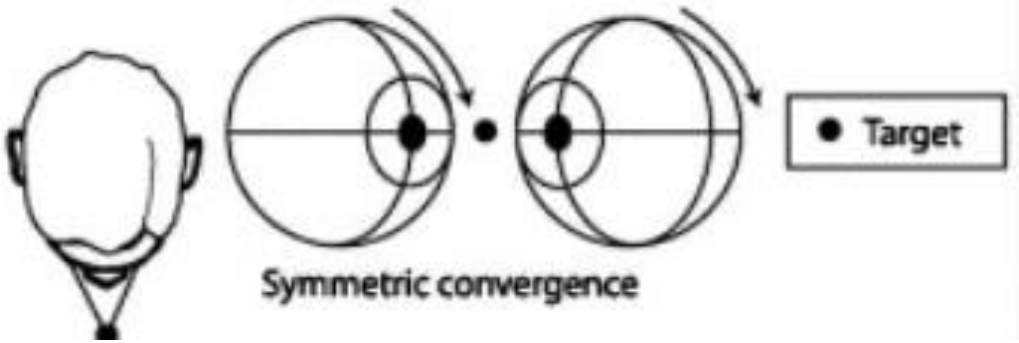
Mantokoudis et al., (2015)

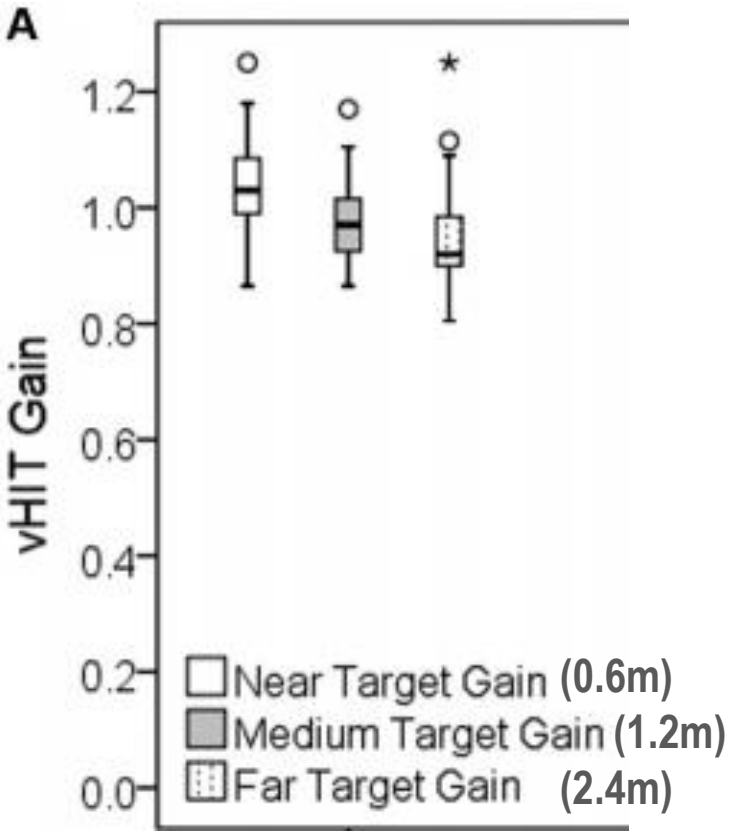
- Poor calibration is most likely cause of very high gain (>1.2) or low gain without saccades
- If you have very high gain or low gain without saccades, **repeat calibration** and check for repeatability of Δ value

Technical factors that may artificially alter gain

Convergence

- If the subject is too close to the target, the eyes will have to converge slightly, giving false VOR gain
- Typically this raises gain





From Judge *et al., (*2018)

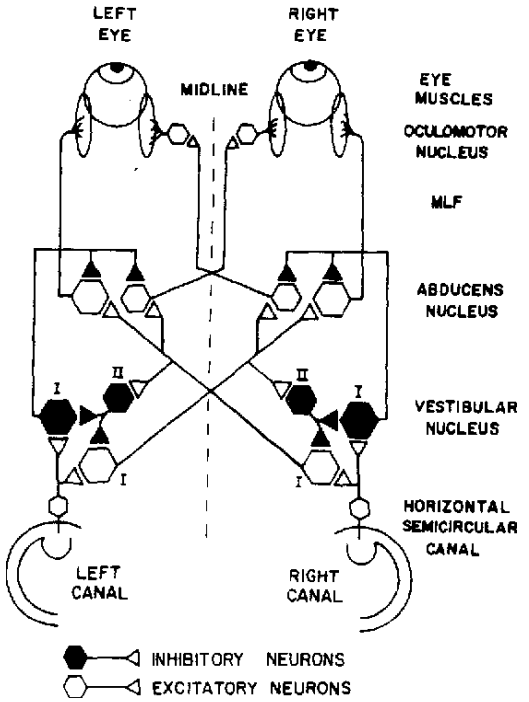
- Seat subject at least 1m from target

Technical factors that may artificially alter gain

Low velocity head impulses

Recommended head velocities are high (lateral impulses 150 -250 °/s, verticals 100-100-200 °/s) (Curthoys and Manzari 2017), for two reasons:

1. To drive the contralateral side into 'inhibitory cutoff' (Ewald's 2nd law)
   - A single impulse causes complementary stimulation of canals on both sides of the head
   - Neural input from both ears contributes to the response at low head velocities
   - In patients with unilateral loss the contribution from the healthy ear can be ruled out by using high velocity head impulses which silence the input from the opposite side.
   - If low velocity head impulses are used, the remaining healthy ear can quite effectively drive the eye movement response for head turns to the affected ear, so that the affected ear appears to have a normal VOR gain.

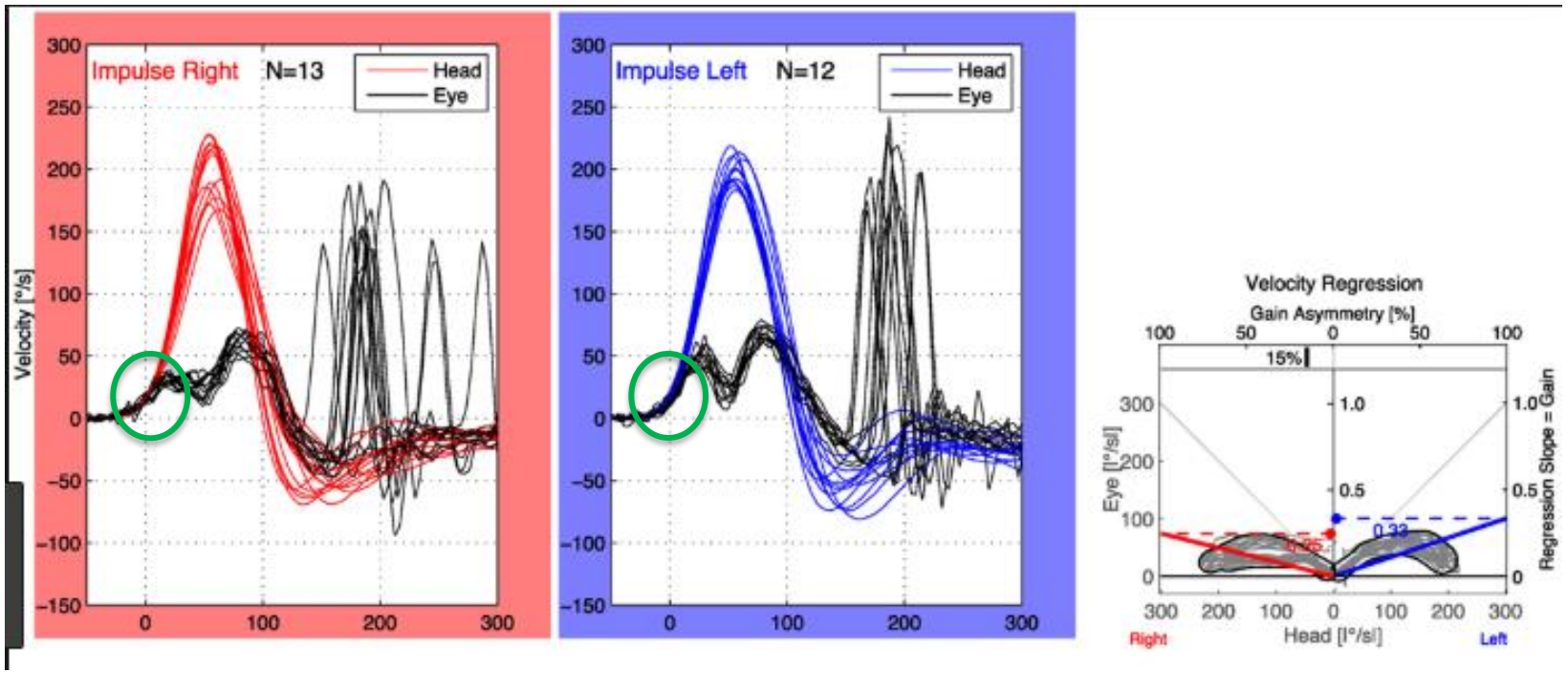


Technical factors that may artificially alter gain

Low velocity head impulses

Recommended head velocities are high (lateral impulses 150 -250 °/s, verticals 100-100-200 °/s) (Curthoys and Manzari 2017), for two reasons:

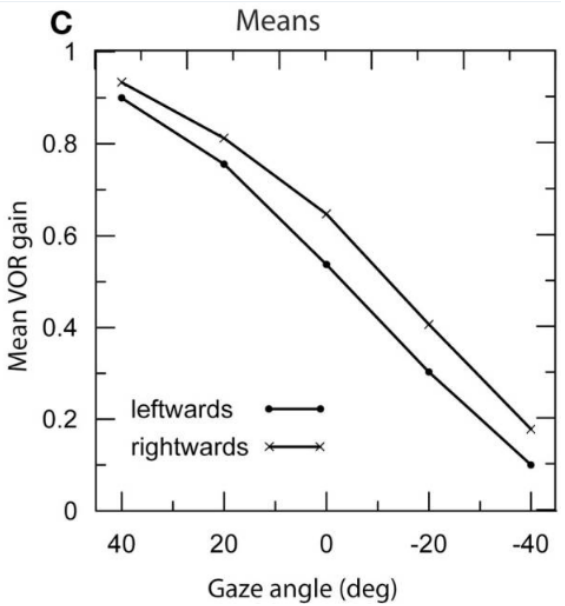
2. The smooth pursuit system contributes to head rotations with fixation below approximately 100 °/s. You can sometimes see this at the start of a head impulse in a patient with bilateral vestibular loss



Technical factors that may artificially alter gain

Vertical canals – torsional eye movements

- LARP and RALP impulses performed with eyes in central gaze produce torsional eye movements
- These cannot be detected with current software
- Gain is artificially reduced
- Need to turn head (or body) 45° away from target
- The subject fixates target out of eccentric gaze
- Tip head towards and away from target
- Resulting eye movement should be purely vertical



From McGarvie *et al.,* 2015