Review

DOI: 10.5152/NSN.2018.0001

The video head impulse test in clinical practice

G. Michael Halmagyi[1*], Ian S. Curthoys[2*]

[1]Department of Neurology, Royal Prince Alfred Hospital, University of Sydney Medical School, Sydney, Australia
[2]School of Psychology, University of Sydney, Sydney, Australia

Abstract

Over the last 10 years the video Head Impulse Test has become the generally accepted initial test vestibular function to be done in patients presenting with vertigo or imbalance. vHIT can give reliable reproducible measurements of the function of each of the 6 semicircular canals individually, in any reasonably cooperative adult or child in about 15 minutes. There are published normative values and the test can be particularly useful in diagnosing: (1) acute unilateral peripheral vestibulopathy (i.e. vestibular neuritis) and differentiating it from cerebellar infarction; and (2) chronic bilateral vestibular vestibulopathy - and differentiating if from the many other causes of persistent imbalance. Here we review the basic physiological principles behind the Head Impulse test and some of the benefits and potential practical problems in video Head Impulse Testing.

Keywords: Vestibular, vertigo, imbalance, semicircular-canal

PHYSIOLOGICAL BASIS OF THE HEAD IMPULSE TEST

Think of the vestibulo-ocular reflex (VOR) as the tendon reflex of the brainstem. A short, fast head rotation (a head impulse) tests semicircular canal (SCC) afferents and the brainstem in response to a synchronized acceleration in much the same way as a patellar tap tests 1a muscle afferents and the spinal cord. The VOR is hard-wired into the neurophysiology of the SCCs and the brainstem; it depends on the resting rate and on-off asymmetry of SCC primary afferents and their robust projections via the vestibular nuclei to the oculomotor nuclei. To understand why and how the Head Impulse Test (HIT) can detect impairment of any single SCC it is necessary to review the basics of SCC function (1).

Primary SCC afferents, the neurons in Scarpa's ganglion, fire at rest signaling the fact that the head is not moving. This resting rate is about 80 spikes/s in the squirrel monkey lateral SCC primary afferents (2). Primary afferents synapse with vestibular nucleus neurons which also fire at rest and then they synapse with abducens motoneurons and interneurons destined to innervate medial rectus motoneurons. Resting rate is the basis of bidirectional responses of single SCCs. Angular acceleration in one direction (the "on") direction will increase the resting rate whereas angular acceleration in the opposite ("off") direction will decrease it. Up to about 120deg/s head velocity the "on" and the "off', in other words left and right responses of primary afferents are symmetrical.

Therefore with angular accelerations in the horizontal plane, smooth symmetrical compensatory eye movements to both leftward and rightward accelerations are due to the two labyrinths working seamlessly together. For natural rapid accelerations the ipsilateral labyrinth predominantly determines the response as shown by neural recordings of irregular primary afferents; specifically, single neurons show a strong response for ipsilateral angular accelerations, but a weak or absent response for contralateral angular accelerations (2). That asymmetrical response of primary afferents for symmetrical accelerations appears to be amplified along the vestibulo-ocular pathway where at each succeeding stage (vestibular nucleus, and abduces nucleus) there is, on average, in-

You may cite this article as: Halmagyi GM, Curthoys IS. The video head impulse test in clinical practice. Neurol Sci Neurophysiol 2018; 35: 1-5.

*The authors contributed equally to this work.

Corresponding Author: Michael G. Halmagyi **E-mail:** gmh@icn.usyd.edu.au **Submitted:** 8 April 2018 **Accepted:** 11 April 2018

creased neural gain and reduced resting discharge (3). Both of these act to enhance the asymmetrical response of the irregular primary afferents to symmetrical angular accelerations. The result is that when one labyrinth is destroyed, the remaining labyrinth generates a strong response for rapid ipsilateral angular accelerations but a weak or absent response for rapid contralateral angular accelerations.

So, after unilateral vestibular destruction in patients and in monkeys, even after vestibular compensation is complete the VOR in response to symmetrical rapid head rotations will forever remain markedly asymmetrical: it will be nearly normal in response to head rotations towards the sole remaining SCC, but markedly impaired in response to head rotations away from the sole remaining SCC (1, 4, 5). This is the physiological basis of the head impulse test. The example above is for the horizontal semicircular canal, but similar neural mechanisms operate for the paired vertical canals (6).

Principles of the Head Impulse Test

Consider a seated patient staring at a target on a wall. If the patient's head is rotated to the left by 30deg, her eyes will counter-rotate by exactly the same amount, at exactly the same speed about exactly the same axis, and will exactly compensate for the head rotation and so keep visual fixation on-target. If the head is rotated slowly then this counter-rotation of the eyes will be the result of visual+vestibular+cervical reflexes. If however the head is rotated quickly then visual and cervical reflexes will be unable to respond quickly enough, so that the compensatory counter-rotation of the eyes will be produced exclusively by vestibular stimulation – i.e. the VOR. Furthermore if the head rotation is very quick then vestibular afferents from the right lateral SCC will be driven into inhibitory saturation and the VOR will be produced predominantly by afferents from the left lateral SCC. If the left lateral SCC if impaired (low VOR gain), then rapid leftward head rotations will produce inadequate compensatory counter-rotation of the eyes the patient will lose fixation of the target and will need to make compensatory ("catch-up") saccades in the opposite direction to the head rotation (i.e. in the same direction is the deficient VOR). If these compensatory saccades occur immediately after the head rotation they will be observable ("overt") and constitute the clinical sign of canal paresis (7). If they occur during the head rotation they will be invisible ("covert") but measurable with the video Head Inpulse Test (vHIT). With vHIT it is possible to measure VOR gain from all 6 SCCs less than 15 minutes (1).

Clinical Applications of the vHIT

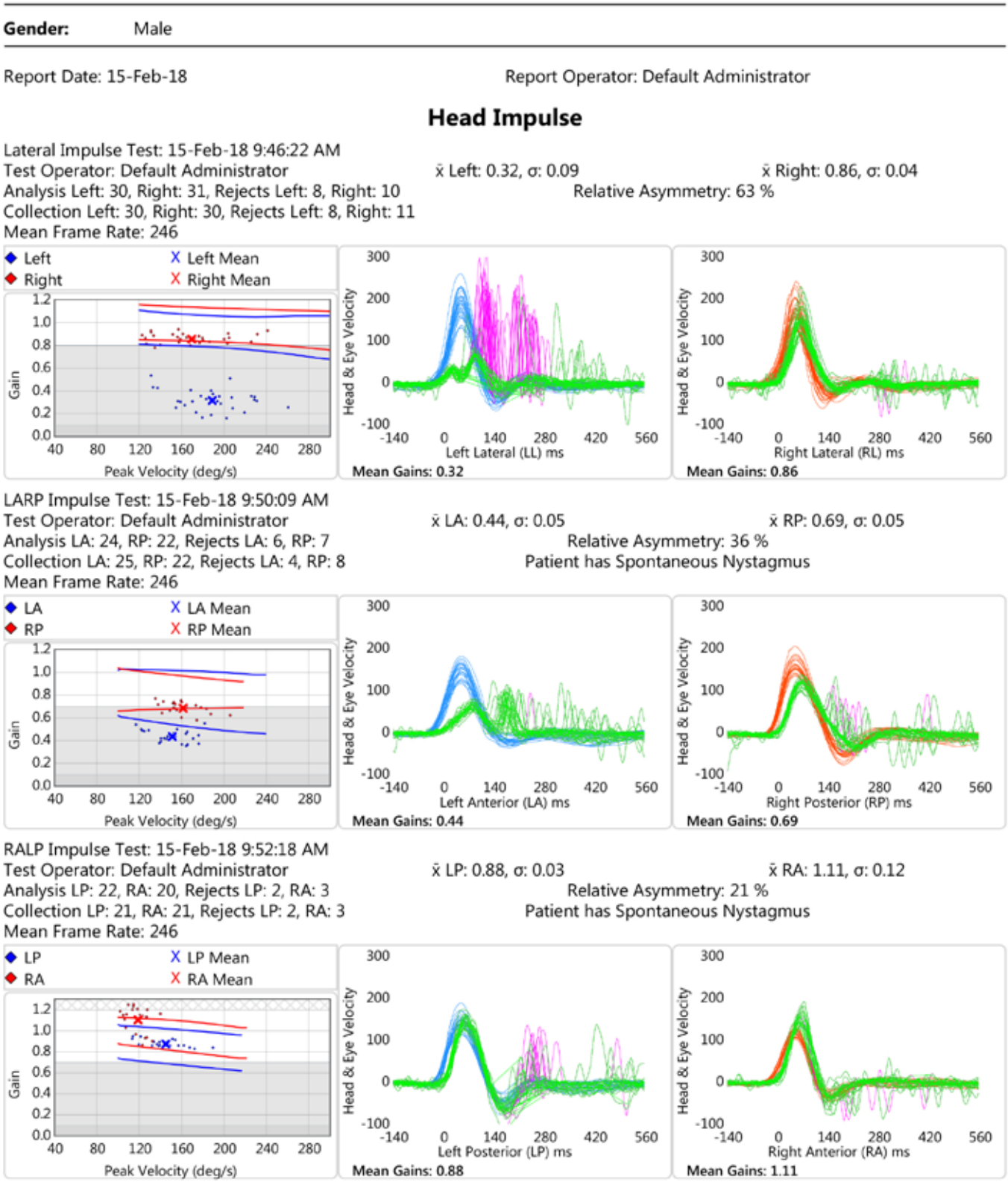
(A) *In a patient seen during an acute vestibular syndrome:* In a patient having her first attack of acute, spontaneous, isolated vertigo the main differential diagnosis is vestibular neuritis and cerebellar infarction. In acute vestibular neuritis there has to be loss of function in 1, 2, or all 3 SCCs – that is a positive HIT. Loss of anterior plus lateral SCC function indicates superior vestibular neuritis. Patients with acute superior vestibular neuritis will also have 3rd degree horizontal-torsional spontaneous nystagmus beating away from the side with reduced VOR gain (a positive HIT). However the clinical HIT, even in capable hands, will be negative if the compensatory saccades are covert. The vHIT in contrast can show and measure any unilateral impairment of SCC function (Figure). Vestibular neuritis, affecting just the posterior SCC can only be diagnosed with vHIT (8). In contrast to acute vestibular neuritis, a cerebellar infarct rarely impairs the VOR so the patient will have a normal vHIT (9-11). In a patient seen in the emergency room with a first-ever acute vestibular syndrome a normal vHIT, indicates a potentially serious condition – cerebellar infarction - about 20% of patients need urgent posterior fossa decompression to prevent death, whereas an abnormal vHIT, indicates a safe-to-discharge condition – vestibular neuritis (12). HINTS is a standardized clinical protocol to distinguish vestibular neuritis from cerebellar infarction (13).

(B) *In a patient seen after an acute vestibular syndrome:* The patient is seen weeks after an acute vestibular syndrome, asymptomatic, or complaining of residual imbalance. The question is: has the patient had acute vestibular neuritis but has not recovered peripheral vestibular function, and now has chronic vestibular insufficiency (or has the patient actually had cerebellar infarct (14) ? If the vHIT is still impaired on one side the diagnosis of vestibular neuritis can be made retrospectively. If the vHIT is now normal the patient might have had vestibular neuritis and SCC recovered but it is also possible that SCC was never impaired since the patient actually had a cerebellar infarct and now needs MRI.

(C) *In a patient with recurrent vertigo attacks:* In a patient with recurrent vertigo attacks the differential diagnosis is migraine, Meniere's disease and benign positional vertigo. Most patients who start to have isolated vertigo attacks from vertebrobasilar TIAs will have a stroke with a week (15). In Meniere's disease vHIT can be normal while the caloric test shows a canal paresis (16). Occasionally BPV is secondary to an inner ear disease, and then might be abnormal (17).

(D) *In a patient with chronic imbalance:* There are many possible causes of (MR negative) chronic imbalance, especially in the elderly: neurological (sensory neuropathy, extrapyramidal disorders, orthostatic tremor, normal pressure hydrocephalus), psychological, musculoskeletal and chronic vestibular insufficiency due to severe unilateral or moderate, symmetrical or asymmetrical, bilateral vestibular impairment (18-21). The patient with chronic vestibular insufficiency has no symptoms while sitting or lying but feels off-balance as soon as she stands and more so when she walks (22). vHIT has a normal range, and can measure function in all 6 SCCs and help decide whether vestibular function is impaired sufficiently to produce imbalance

Figure 1. Video Head Impulse Test (measured with a GN Otometrics Impulse system) in a male (52y) with severe acute left vestibular neuritis. All 3 SCCs are affected with VOR gains of 0.2 or less (N>0.8). Overt compensatory saccades are present with posterior (top) and lateral SCC impulses more than with anterior SCC impulses (middle).



(23, 24). A common cause of an isolated severe unilateral vestibular loss presenting with chronic vestibular insufficiency is an unrecognized previous attack of acute vestibular neuritis (25). If there is no history of a vertigo attack, then chronic progressive cause of unilateral vestibular loss such as a schwannoma needs to be considered (26). Selective bilateral vestibular loss (vestibular loss with normal hearing can be due to gentamicin toxicity (27). If there are also proprioceptive and cerebellar impairments then SCA3 and CANVAS need to be considered (28, 29).

vHIT: Potential Practical Problems

Although vHIT can be quick and easy to do, like any other physiological test it requires training, practice, and attention to detail and learning to recognize artifacts (30, 31). The skill level required to be confident of producing reproducible results is about that required for recording sensory nerve action potentials. For example it is important that the patient is continually encouraged to keep looking at the visual target, not to blink, and not to resist or assist with the passive head turning. Head impulse stimuli should be given at a range of speeds up to 300deg/s. During vertical SCC canal testing the patient must maintain eccentric horizontal eye position (32).

vHIT versus Caloric Tests

Caloric testing still has a place in patients with a normal vHIT, if Meniere's disease is a possible diagnosis since the caloric can be impaired with the vHIT normal (33, 34). Also in patients with recovered vestibular neuritis, the recovery might be less obvious on caloric testing than on vHIT. This means that a patient seen some time after an acute vestibular syndrome who now has a normal vHIT should have a caloric test – which might still show a canal paresis, indicating that it really was vestibular neuritis and there is no need for MRI.

Conflict of Interest: The authors declared that they act as unpaid consultants to GN Otometrics for the development of video Head Impulse Testing and have received reimbursement for conference travel and sundry expenses.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Halmagyi GM, Chen L, MacDougall HG, Weber KP, McGarvie LA, Curthoys IS. The video head impulse test. Front Neurol 2017; 8: 258. [CrossRef]
2. Sadeghi SG, Minor LB, Cullen KE. Response of vestibular-nerve afferents to active and passive rotations under normal conditions and after unilateral labyrinthectomy. J Neurophysiol 2007; 97: 1503-1514. [CrossRef]
3. Shinoda Y, Yoshida K. Dynamic characteristics of responses to horizontal head angular acceleration in vestibuloocular pathway in the cat. J Neurophysiol 1974; 37: 653-673. [CrossRef]
4. Sadeghi SG, Minor LB, Cullen KE. Dynamics of the horizontal vestibuloocular reflex after unilateral labyrinthectomy: response to high frequency, high acceleration, and high velocity rotations. Exp Brain Res 2006; 175: 471-484. [CrossRef]
5. Curthoys IS, Halmagyi GM. Vestibular compensation: a review of the oculomotor,neural, and clinical consequences of unilateral vestibular loss. J Vestib Res 1995; 5: 67-107. [CrossRef]
6. Markham CH. Midbrain and contralateral labyrinth influences on brain stem vestibular neurons in the cat. Brain Res 1968; 9: 312-333. [CrossRef]
7. Weber KP, Aw ST, Todd MJ, et al. Head impulse test in unilateral vestibular loss: vestibulo-ocular reflex and catch-up saccades. Neurology 2008; 70: 454-463. [CrossRef]
8. Taylor RL, McGarvie LA, Reid N, et al. Vestibular neuritis affects both superior and inferior vestibular nerves. Neurology 2016; 87: 1704-1712. [CrossRef]
9. Mantokoudis G, Tehrani AS, Wozniak A, et al. VOR gain by head impulse video-oculography differentiates acute vestibular neuritis from stroke. Otol Neurotol 2015; 36: 457-465. [CrossRef]
10. Chen L, Todd M, Halmagyi GM, Aw S. Head impulse gain and saccade analysis in pontine-cerebellar stroke and vestibular neuritis. Neurology 2014; 83: 1513-1522. [CrossRef]
11. Kim HA, Yi HA, Lee H. Recent advances in cerebellar ischemic stroke syndromes causing vertigo and hearing loss. Cerebellum 2016; 15: 781-788. [CrossRef]
12. Neugebauer H, Witsch J, Zweckberger K, Jüttler E. Space-occupying cerebellar infarction: complications, treatment, and outcome. Neurosurg Focus 2013; 34: E8. [CrossRef]
13. Newman-Toker DE, Curthoys IS, Halmagyi GM. Diagnosing stroke in acute vertigo: The HINTS family of eye movement tests and the future of the "Eye ECG". Semin Neurol 2015; 35: 506-521. [CrossRef]
14. Adamec I, Krbot Skorić M, Ozretić D, Habek M. Predictors of development of chronic vestibular insufficiency after vestibular neuritis. J Neurol Sci 2014; 347: 224-228. [CrossRef]
15. Halmagyi GM. Brainstem stroke preceded by transient isolated vertigo attacks. J Neurol 2017; 264: 2170-2172. [CrossRef]
16. Cordero-Yanza JA, Arrieta Vázquez EV, Hernaiz Leonardo JC, et al. Comparative study between the caloric vestibular and the video-head impulse tests in unilateral Menière's disease. Acta Otolaryngol 2017; 137: 1178-1182. [CrossRef]
17. Riga M, Bibas A, Xenellis J, Korres S. Inner ear disease and benign paroxysmal positional vertigo: a critical review of incidence, clinical characteristics, and management. Int J Otolaryngol 2011; 2011: 709469. [CrossRef]
18. Popkirov S, Staab JP, Stone J. Persistent postural-perceptual dizziness (PPPD): a common, characteristic and treatable cause of chronic dizziness. Pract Neurol 2018; 18: 5-13. [CrossRef]
19. Hillier S, McDonnell M. Is vestibular rehabilitation effective in improving dizziness and function after unilateral peripheral vestibular hypofunction? An abridged version of a Cochrane Review. Eur J Phys Rehabil Med 2016; 52: 541-556.
20. Moon M, Chang SO, Kim MB. Diverse clinical and laboratory manifestations of bilateral vestibulopathy. Laryngoscope 2017; 127: E42-E49. [CrossRef]
21. Strupp M, Kim JS, Murofushi T, et al. Diagnostic criteria consensus document of the classification committee of the Bárány Society. J Vestib Res 2017; 27: 177-189. [CrossRef]
22. Petersen JA, Straumann D, Weber KP. Clinical diagnosis of bilateral vestibular loss: three simple bedside tests. Ther Adv Neurol Disord 2013; 6: 41-45. [CrossRef]

23. McGarvie LA, MacDougall HG, Halmagyi GM, et al. The Video Head Impulse Test (vHIT) of semicircular canal function - age-dependent normative values of VOR gain in healthy subjects. Front Neurol 2015; 6: 154. [CrossRef]
24. Agrawal Y, Davalos-Bichara M, Zuniga MG, Carey JP. Head impulse test abnormalities and influence on gait speed and falls in older individuals. Otol Neurotol 2013; 34: 1729-1735. [CrossRef]
25. Patel M, Arshad Q, Roberts RE, Ahmad H, Bronstein AM. Chronic symptoms after vestibular neuritis and the high-velocity vestibulo-ocular reflex. Otol Neurotol 2016; 37: 179-184. [CrossRef]
26. Taylor RL, Kong J, Flanagan S, et al. Prevalence of vestibular dysfunction in patients with vestibular schwannoma using video head-impulses and vestibular-evoked potentials. J Neurol 2015; 262: 1228-1237. [CrossRef]
27. Ahmed RM, Hannigan IP, MacDougall HG, Chan RC, Halmagyi GM. Gentamicin otoxicity: a 23-year selected case series of 103 patients. Med J Aust 2012; 196: 701-704. [CrossRef]
28. Gordon CR, Zivotofsky AZ, Caspi A. Impaired vestibulo-ocular reflex (VOR) in spinocerebellar ataxia type 3 (SCA3): bedside and search coil evaluation. J Vestib Res 2014; 24: 351-355.
29. Szmulewicz DJ, Roberts L, McLean CA, et al. Proposed diagnostic criteria for cerebellar ataxia with neuropathy and vestibular areflexia syndrome (CANVAS). Neurol Clin Pract 2016; 6: 61-68. [CrossRef]
30. Curthoys IS, MacDougall HG, McGarvie LA, et al. The video head impulse test (vHIT). In: Jacobson GP, Shepard NT, eds. Balance Function Assessment and Management. 2nd ed. San Diego, CA: Plural Publishing; 2014: 391-430.
31. Mantokoudis G, Saber Tehrani AS, Kattah JC, et al. Quantifying the vestibulo-ocular reflex with video-oculography: nature and frequency of artifacts. Audiol Neurootol 2015; 20: 39-50. [CrossRef]
32. McGarvie LA, Martinez-Lopez M, Burgess AM, MacDougall HG, Curthoys IS. Horizontal eye position affects measured vertical VOR Gain on the video head impulse test. Front Neurol 2015; 6: 58. [CrossRef]
33. van Esch BF, Nobel-Hoff GE, van Benthem PP, van der Zaag-Loonen HJ, Bruintjes TD. Determining vestibular hypofunction: start with the video-head impulse test. Eur Arch Otorhinolaryngol 2016; 273: 3733-3739. [CrossRef]
34. McGarvie LA, Curthoys IS, MacDougall HG, Halmagyi GM. What does the dissociation between the results of video head impulse versus caloric testing reveal about the vestibular dysfunction in Ménière's disease? Acta Otolaryngol 2015; 135: 859-865. [CrossRef]