

Head Impulse Test

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ABSTRACT

In 1988, Michael Halmagyi and Ian Curthoys described a simple yet reliable indicator of unilateral peripheral vestibular deficits at the bedside. This 'head impulse test' (HIT) as is has been known since, has stood the test of being a reliable indicator of vestibular deficit, especially for the horizontal semicircular canal.

The test has been described using various techniques, such as the scleral search coil, the video HIT, etc. but even a simple bedside clinical evaluation with a little amount of experience usually sufficed to detect overt vestibular deficits.

Numerous studies have compared the efficacy of the HIT as compared with other tests of vestibular deficit, the caloric test (CT) being the most commonly compared one. A look at the physiology behind the two tests reveals the differing pathways tested by the two tests in mention, and hence it would be wise to say that the two are complimentary, rather than exclusive of the other.

The HIT today forms a vital part of the armamentarium of the otologist to arrive at rapid understanding of the nature of the vestibular deficit, the laterality of the lesion, and the probable prognostic significance.

Keywords: Vestibulo-ocular reflex, Head impulse test, Sclera search coil.

How to cite this article: Khattar VS, Hathiram BT. Head Impulse Test. *Otorhinolaryngol Clin Int J* 2012;4(2):106-111.

Source of support: Nil

Conflict of interest: None declared

INTRODUCTION

Broadly, vestibular pathologies can be classified into three main categories, namely:

1. Unilateral vestibular weakness/failure
2. Bilateral vestibular weakness/failure
3. Inadequate paroxysmal stimulation or inhibition of the vestibular system.

Most conditions that fall under category (3) are easily diagnosed by their characteristic symptomatology and presentation, such as benign paroxysmal positional vertigo (BPPV). The head impulse test (HIT) plays an important role in the diagnosis of the presence of categories (1) and (2).

Although the test has been described with the 'scleral search coil technique' as its gold standard, the video HIT and even the bedside HIT has been proven to be an effective and comparable alternative. While the former technique would employ the use of a magnetic frame, one search coil mounted in the sclera and another on the forehead, and prove to be expensive, time consuming and impossible at the bedside, the video HIT has been shown to have a reasonably good sensitivity and specificity, comparable to the former

technique.¹ For that matter, even the bedside or clinical HIT has been shown to be a useful clinical tool to assess patients with acute spontaneous vertigo, and differentiate between acute vestibular neuritis which would show a positive test, and an acute cerebellar lesion (where the test would be negative). However, there have been reports of a 9 to 39% false-positive rate for the bedside HIT in acute cerebellar or brainstem strokes.^{2,3} It shall nevertheless remain a useful screening test at the bedside, which may be validated as soon as possible by the video HIT.

TECHNIQUE

A small, rapid, passive, unpredictable horizontal head rotation or 'head impulse' is given to the patient by the clinician standing behind the patient, while the patient fixates on a target in front of him/her. In patients with a deficient vestibulo-ocular reflex (VOR), the slow phase eye velocity is reduced. Thus, when the head is thrust in a certain direction, the eyes move along with the head, and then after a time lag, they perform a corrective saccade to return to their initial position to fixate on the target. This corrective saccade is detected by the video camera, and is reported as a positive HIT in the same direction as that of the head thrust, indicating an ipsilateral defective VOR.

Despite being reasonably comparable to the scleral coil technique, there are two main drawbacks with the video HIT:

Firstly, there is no quantitative estimation of the extent of VOR gain, and it is a purely subjective estimation of the clinician. In fact, in a study by Jorns-Haderli et al⁴ in 2007, they demonstrated that there existed a significant difference between the interpretation of the video HIT between 'experts' (those who had a minimum training of at least 6 months in neuro-otology) and 'nonexperts' (those who had no training). But this drawback could be reduced by showing the 'non-experts' introductory video clips (termed by these authors as 'minimal instructions') so as to train them in the interpretation of the HIT.

Secondly, different clinicians have differing techniques as regards the amount of thrust, the degree of lateral excursion of the head, the velocity of thrust, the distance of the target from the patient's eyes, etc. These variables have the potential to alter the VOR gain and thus the clinical interpretation. In general it has been seen, however, that the greater the thrust, the more robust the VOR gain achieved; also, the shorter the distance between the eyes

and target, the greater the VOR gain. This latter aspect may explain the reason why the bedside HIT test has become popular, as the viewing target distance is usually 0.5 meters, thus amplifying the VOR gain.⁵⁻⁷ Nevertheless, Jorns-Haderli et al (2007)⁴ have shown that between two target distances of 0.5 meters (for the bedside HIT) and 1.5 meters (for the video HIT), there was a difference between the VOR gains of only about 10%; thus this factor did not probably have a large role to play, as long as the distances were not kept too large.

Comparison with the Caloric Test

Most studies have compared the effectiveness of the HIT based on its comparison with the caloric test. While both tests undoubtedly test the degree of horizontal canal paresis, their comparison would be lopsided on physiological grounds.

Numerous studies that compared the effectiveness of the HIT vis-a-vis the caloric test found very low sensitivities for the former, thus rendering it a useless tool for the assessment of peripheral vestibular disease.^{9,10}

Caloric irrigation testing has been used for a long time to determine the amount of canal paresis. This test assesses the VOR at very low-frequencies of stimulation, i.e. 0.003 Hz. The HIT on the other hand is a high-frequency stimulation test, with stimulation frequencies of about 5 Hz (Halmagyi et al 1990; ranging between 3 to 6 Hz). Thus, both these tests are physiologically exclusive and evaluate different aspects of the VOR.

In addition, after a vestibular insult, for example vestibular neuritis, the central compensatory mechanisms that come into play, can accommodate for the low-frequency VOR recovery, but are inadequate for high-frequency VOR recovery.^{8,11-14} Thus, in such situations, comparing the two tests would also depend upon the stage of evolution of the pathology.

Also, in patients suffering from bilateral canal paresis (bilateral vestibulopathy; Figs 1A to G), especially in the symmetrical cases, the canal paresis factor as calculated by the caloric test would be absent (in the normal range) as compared to the HIT which would show a bilateral decreased VOR.

Thus, the HIT can prove to be a very useful tool in cases that may be missed on caloric testing.

Canal Testing in Different Planes: Testing all Semicircular Canals Independently

Although the horizontal semicircular canals are the most frequently tested (and reported) canals for testing canal paresis with the HIT, the other semicircular canals can also be tested. In fact, this is a distinct advantage of the HIT over the caloric test.



Fig. 1A: A patient with bilateral vestibulopathy, being prepared for the HIT. The patient is asked to fixate at a target in front, and keep looking at the target while the head is rotated in an unpredictable fashion, passively by the examiner, standing behind the patient



Fig. 1B: On rapid rotation of the head to the left, the eyes move with the head to the left. This indicates a defective VOR to the left. This is the left horizontal semicircular canal being tested



Fig. 1C: The corrective saccade that brings the eyes back to the center after a small time lag. This saccade indicates the 'central' corrective measure to combat the defective VOR to the left

The fact that the normal response to a high frequency head movement has a latency of about 10 ms, as compared to other eye movement control mechanisms which come into play only after 70 ms.¹⁶ Thus, these responses are too



Fig. 1D: On rapid rotation of the head to the right, the eyes move with the head to the right



Fig. 1G: On performing the slow passive movements to the left, the eyes again move equal and opposite to the direction of the head and continue to fixate on the target. Thus, we understand that the HIT is a 'high-frequency' test



Fig. 1E: The corrective saccade that brings the eyes back to the center after a small time lag. This indicates that the patient has a bilateral defective VOR, consistent with the diagnosis of bilateral vestibulopathy



Fig. 1F: On performing slow passive movements to the right, the patient's eyes move equal and opposite to the head

'early' for other eye movement control mechanisms to participate, and only the semicircular canals can accommodate these quick responses. This is why the HIT becomes an indirect test of semicircular canal function.

By making head rotations in the directions of semicircular canal pairs, it can detect a deficit in the canal being tested.¹⁵ As per Ewald's laws, a deficit in the horizontal canal can be unveiled when rotation in the direction of the concerned canal detects a corrective saccade (*vide supra*). For the vertical canals, the deficiencies are unveiled when the canal is stimulated in the opposite direction of their ampulla. Notwithstanding the differences between the canals, the ability to test each canal individually allows for a more accurate representation of the exact nature of the vestibular loss (Figs 2A to C, 3A to J).

The test may thus be able to differentiate between superior and inferior vestibular neuritis, Lindsay Hemenway syndrome, etc.

SUMMARY

The HIT forms a valuable test, which may be performed on all patients who present with vertigo. It helps to broadly classify the patient into either a unilateral or bilateral vestibular loss, and thus may even help in targeting the investigative protocol for the patient. It is a simple, safe yet valuable test which can easily be performed at the bedside as well as in the outpatient clinic, and is bereft of side effects or complications. It is easily reproducible, and reliable, and despite interinvestigator variability, it has a large range of scope for error, and thus a comparable sensitivity and specificity. It should always be interpreted with other tests, such as the caloric test (along with the entire nystagmography protocol), and audiological and radiological tests. Nevertheless, it gives an impression of the probable diagnosis, and coupled with a good history taking, can help arrive at a probable diagnosis, which might prove to be accurate, even after the entire vertigo test battery. It is also reliable in cases of partial vestibular weakness, as well as in partially recovered weakness.



Figs 2A to C: Clinical photograph of a patient with a granulomatous lesion in the right internal auditory canal, as seen on MRI. The patient has right infranuclear facial palsy, as seen from the images



Fig. 3A: The patient being prepared for the HIT



Fig. 3B: Rapid rotation of the head to the right showing the eyes moving with the head to the right



Fig. 3C: The corrective action taken by the eyes to revert to the central position, while the head is still turned toward the right. This indicates a defective right VOR



Fig. 3D: On turning the head toward the left, the eyes make an equal and opposite excursion to the right, thus maintaining their central position fixated on the target. This is a normal response, indicating that the left VOR is normal



Fig. 3E: The head is now turned to the left, and then rapid rotations are given in the pitch plane. Turning the head to the left and down anteriorly, as seen here, stimulates the right posterior semicircular canal (see Alexander's law). We see here that the eyes move with the head initially



Fig. 3F: The eyes now perform a corrective action to refixate on the target. Thus, the right posterior canal is parietic



Fig. 3G: With the head turned to the left, and rotated rapidly upwards and posteriorly, the eyes move equal and opposite—thus the left anterior/superior canal is normal



Fig. 3H: Turning the head to the right, and then rapidly rotating downward, tests the left posterior canal which is seen to be normal (for interpretation *vide supra*)



Fig. 3I: On turning the head to the right and rapidly rotating upward posteriorly the right anterior canal is being tested. The eyes again move with the head initially



Fig. 3J: The corrective action taken by the eyes. Thus, we see that all three semicircular canals on the right are involved/hypoactive/parietic, which is consistent with the diagnosis

ACKNOWLEDGMENT

The authors would like to thank the Dean, TN Medical College and BYL Nair Charitable Hospital, Mumbai for his permission to publish this article.

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