The Head Impulse Test:
Technique and Pitfalls for Emergency Clinicians

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How to Use This Review

The head impulse test (HIT), first described by Halmagyi and Curthoys in 1988, is a bedside examination of vestibulo-ocular reflex (VOR) function. It is rapid, requires no equipment, and has high sensitivity and specificity for peripheral vestibular lesions. However, pitfalls—particularly the "dangerous negative" HIT—require careful understanding [14] [3] [6] [8] [10].

The document follows a structured clinical format with numbered sections, integrated callout boxes for rapid reference, summary tables, and a references section. It is designed both as a learning resource and a quick-reference tool for practising clinicians [20].

 Key Point: *Foundational concepts and summary statements that anchor the core scientific content of each section.*

 Clinical Insight: Clinically relevant observations derived directly from the evidence — for direct application in assessment and diagnosis.

 Clinical Pearl: High-yield, memorable clinical points — the take-home messages most likely to influence management or examination performance.

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I. Introduction and Historical Context

The head impulse test (HIT) was first formally described by Halmagyi and Curthoys in 1988 as a rapid bedside assessment of the horizontal vestibulo-ocular reflex (VOR) and semicircular canal function [1,2]. A brief, unpredictable, high-acceleration head rotation is applied while the patient fixates on a target — loss of compensatory VOR drive is unmasked by a visible corrective (catch-up) saccade back to the target [1,3] [18].

The test has since become a cornerstone of acute vestibular assessment because it directly probes peripheral vestibular function and differentiates peripheral from central causes of acute vestibular syndrome (AVS) in seconds, at the bedside [2,4,5]. Its negative predictive value for peripheral pathology — when positive — is what gives HIT its clinical weight within the HINTS protocol [5,8].

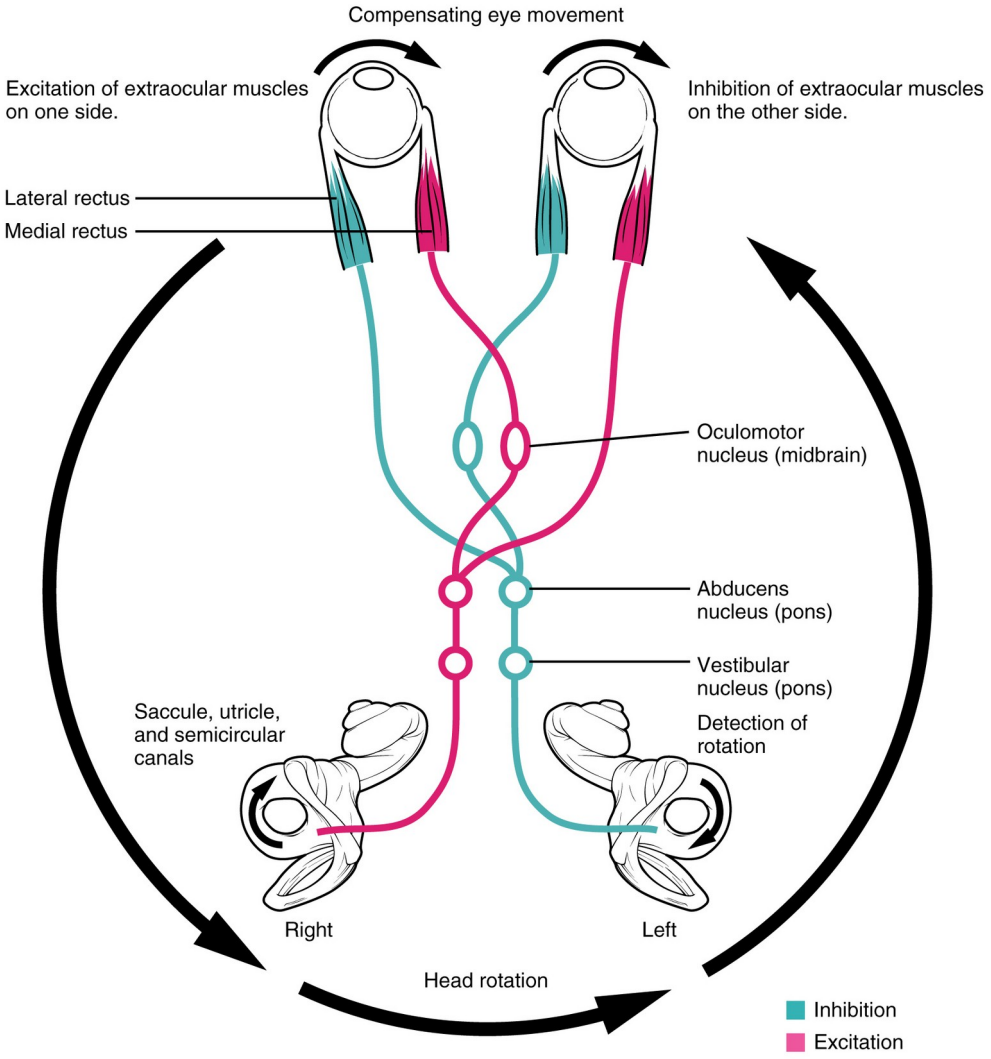
HIT remains highly valuable in the ED, but clinicians must understand its physiological basis, proper technique and limitations to avoid the catastrophic error of a ‘dangerous negative HIT’ in posterior-circulation stroke [3,5,11] [14].

The clinical utility of HIT derives from its direct probe of the three-neuron VOR arc: semicircular canal afferents, vestibular nuclei and oculomotor nuclei [6,7]. Because the arc is short and fast, a normal VOR implies peripheral and brainstem integrity along that pathway — which is precisely why a negative HIT in a patient with AVS is a red flag for a lesion outside the arc, typically a cerebellar or brainstem stroke [5,11,12].

Accurate interpretation depends as much on technique as it does on pathology recognition [3,13]. Slow thrusts, poor fixation, blinking, neck rigidity and anticipation all degrade the test [13]. ED clinicians should rehearse the technique deliberately — HIT is a skill, not a glance [3,13] [19].

 Key Point: *HIT is a test of VOR function, not a test of vertigo. A positive HIT indicates peripheral vestibular involvement; a negative HIT does not exclude central disease.*

II. VOR Physiology and the Three-Neuron Arc



Anatomical Plate 1. Overview of the vestibulo-ocular reflex — head rotation is detected by the semicircular canals, transduced by hair cells, and drives contralateral eye rotation via the three-neuron arc.

Source: OpenStax College, Anatomy & Physiology. Wikimedia Commons, CC BY 3.0.

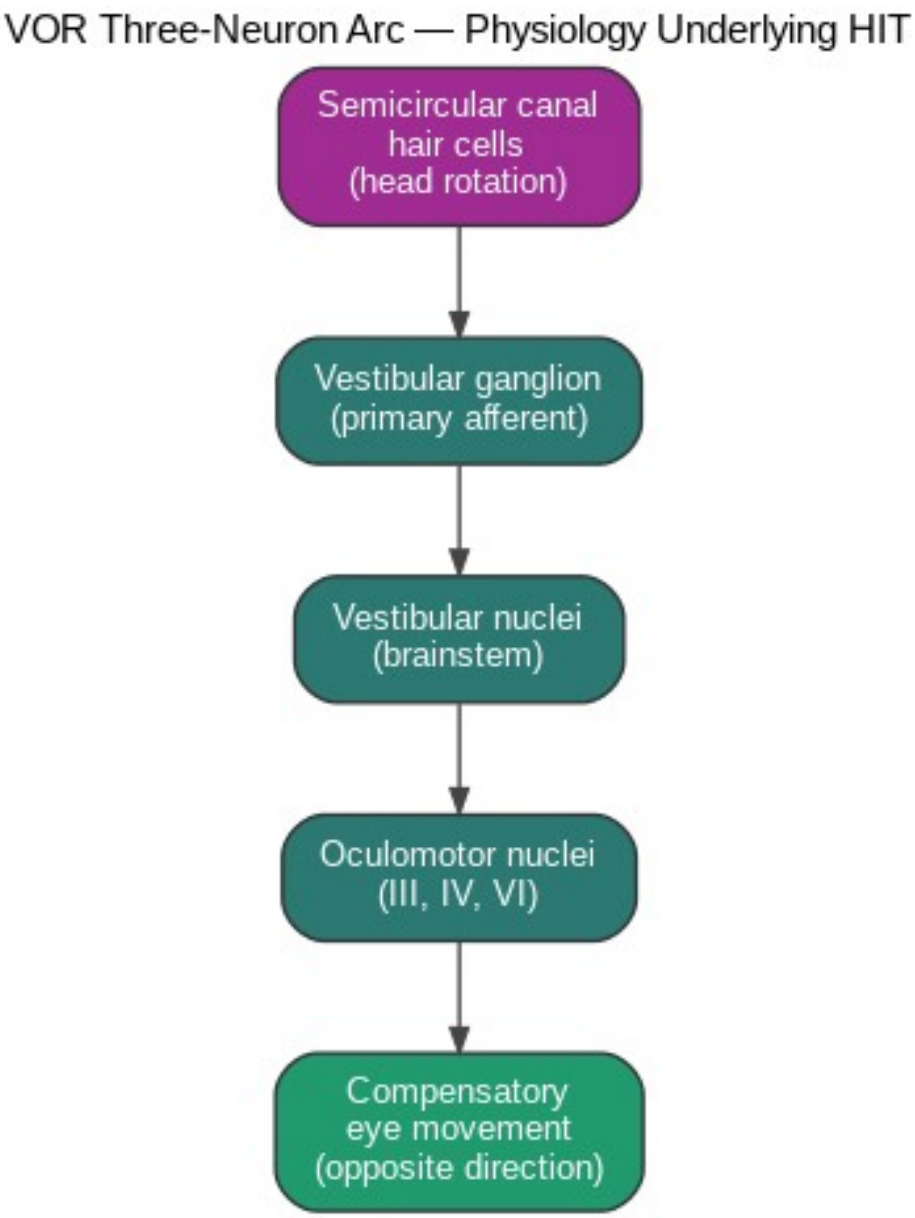
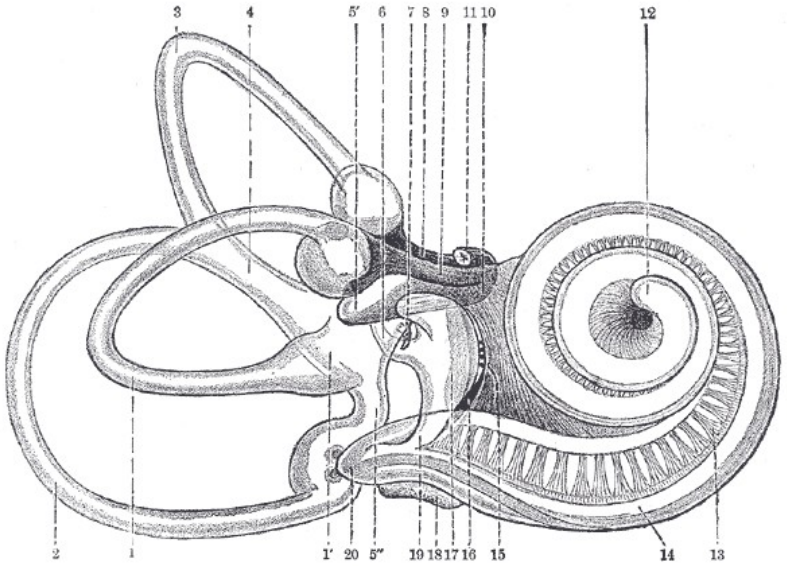


Figure 1. VOR three-neuron arc — the physiological basis of HIT.

The Vestibulo-Ocular Reflex



Anatomical Plate 2. The right membranous labyrinth — three semicircular canals (horizontal, anterior, posterior) and the otolith organs (utricle and saccule), each with sensory hair cells within a cupula or macula.

Source: Henry Vandyke Carter, Gray's Anatomy (1918). Public Domain.

The vestibulo-ocular reflex stabilises gaze during head movement via a three-neuron arc [6,7]. When the head rotates right, canal afferents on the right side increase their firing rate while left-sided afferents decrease; the differential signal drives conjugate eye rotation to the left at equal velocity — keeping the visual world stable on the retina [6,7].

VOR latency is ~10 ms — faster than any visually driven tracking system — which is why head-motion-induced visual blur is normally imperceptible [7]. Loss of this rapid drive manifests as oscillopsia and the characteristic corrective saccade that HIT exploits [3,7].

The three neurons are: (1) the primary vestibular afferent in Scarpa's ganglion, (2) the second-order neuron in the vestibular nuclei of the medulla and pons, and (3) the ocular motor neuron in the abducens, oculomotor or trochlear nuclei [6,7]. Damage anywhere along this short arc — or in the labyrinth feeding into it — produces an abnormal HIT; lesions outside it generally do not [5,6,11].

Push-Pull Mechanism

The VOR works on a push-pull principle: each horizontal canal increases firing for ipsilateral rotation and decreases firing for contralateral rotation [6,7]. A unilateral peripheral lesion removes the push from that side, so during a rapid thrust toward the lesioned side the brain receives no adequate VOR signal and the eyes lag — generating the corrective catch-up saccade that defines a positive HIT [3,7].

High-Frequency Response and Velocity Storage

The VOR is tuned to high-frequency, high-acceleration head movements — the exact stimulus produced by walking and running [7]. Low-frequency, slow head movements are supplemented by smooth pursuit and optokinetic reflexes, which is why asking the patient to follow your finger does NOT test the VOR and cannot substitute for HIT [3,7,13].

Peripheral vestibular lesions selectively impair the high-frequency VOR, and this is precisely the frequency range HIT samples [7,13]. Caloric testing, by contrast, stimulates low-frequency VOR and may be normal when HIT is grossly abnormal, and vice versa — the two are complementary, not redundant [7,13].

 Clinical Insight: The VOR is the most primitive and fastest eye movement system. Peripheral lesions reliably disrupt it; central lesions may spare it if the three-neuron arc is intact.

III. Bedside HIT Technique

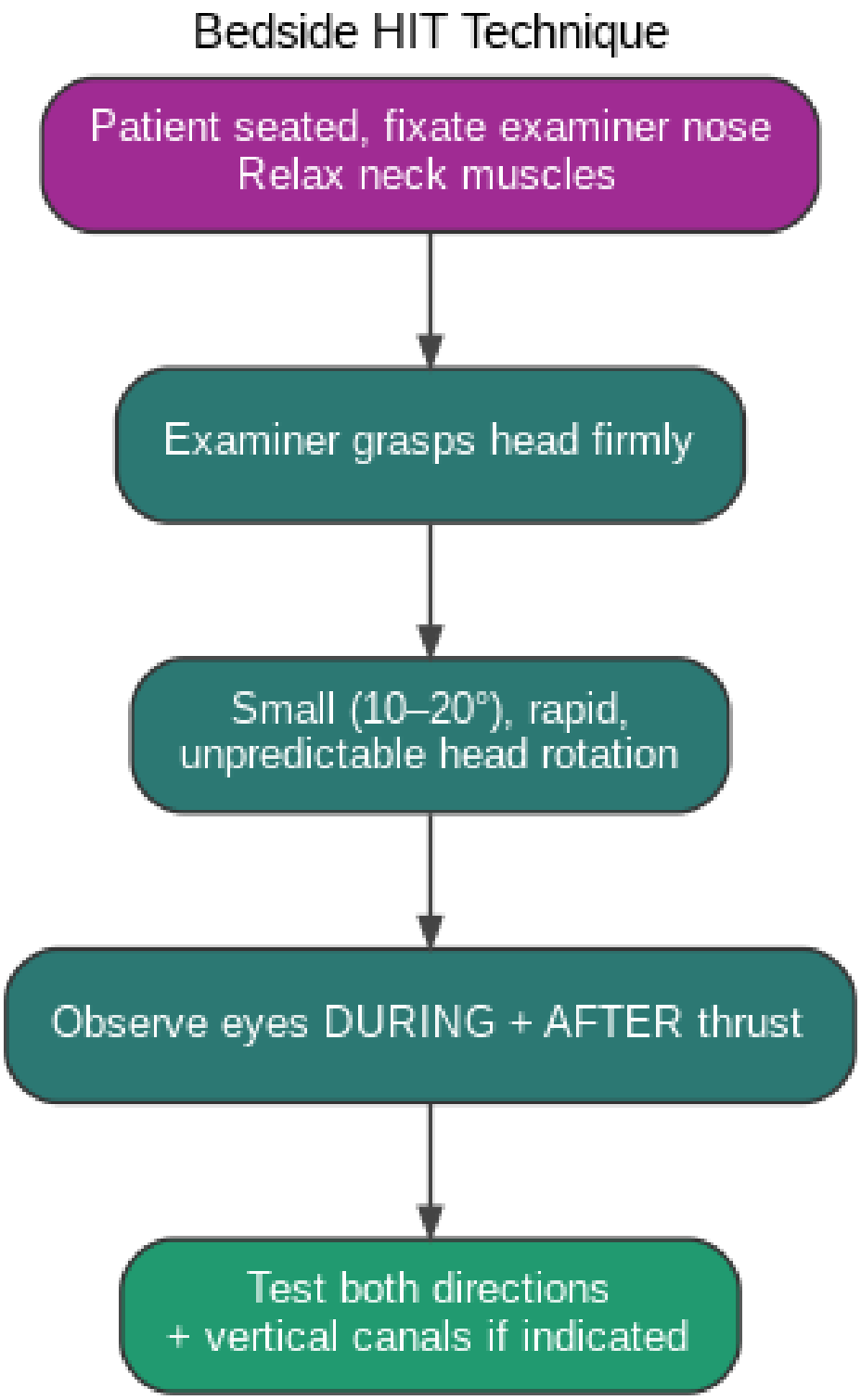


Figure 2. Bedside HIT technique — small amplitude, high acceleration, unpredictable timing.

Patient Positioning and Instruction

Seat the patient upright, facing the examiner at arm's length [3,13]. Give clear instructions: 'Keep your eyes locked on my nose no matter what I do with your head.' Confirm the patient can fixate and is not dizzy from positioning before starting [3,13].

Cooperation is critical [3,13]. Children under five, patients with impaired consciousness, severe anxiety, or very stiff necks may not tolerate the test — record this rather than over-interpret equivocal findings [13]. Neck pathology (significant cervical spondylosis, recent trauma, suspected vertebral dissection) is a contraindication to rapid passive head movement [11,13].

Head Rotation Speed and Amplitude

Grasp the patient's head firmly with both hands on the mastoid processes or the angle of the jaw [3,13]. Deliver a small-amplitude (10–20°), high-acceleration, unpredictable horizontal rotation — the unpredictability is essential, because a patient who anticipates the thrust can generate a covert saccade that masks a positive result [3,13].

The commonest technical error is a thrust that is too slow [13]. Slow head movements engage smooth pursuit, which will keep the eyes on target even in a peripheral lesion, producing a false-negative result [3,13]. The thrust should feel brisk; think 'flick,' not 'push' [13].

Observation During the Test

Watch the patient's eyes during AND immediately after the thrust [3,13]. A positive HIT is a visible corrective saccade bringing the eyes back onto the target; a negative HIT is eyes that remain locked on the target throughout [3,5]. Do not watch the head — watch the eyes [13].

Perform HIT in both directions, several times each, and avoid rhythmic timing so the patient cannot anticipate [3,13]. Asymmetric catch-up saccades — positive on one side, negative on the other — indicate unilateral peripheral hypofunction and are the classic pattern of vestibular neuritis and labyrinthitis [5,13].

Systematic Testing

Test horizontal canals first, then angle the head ~30° forward and test the vertical canals when indicated (superior and posterior canals) [7,13]. Vertical HIT is more technically demanding and less widely used at the bedside, but vHIT makes it routine and is particularly useful in AICA-territory stroke and in Ménière's disease [4,13] [10].

 Key Point: *Proper HIT technique requires: brisk head movement (~180 °/sec), patient fixation, and careful observation.*

IV. Interpreting HIT Results

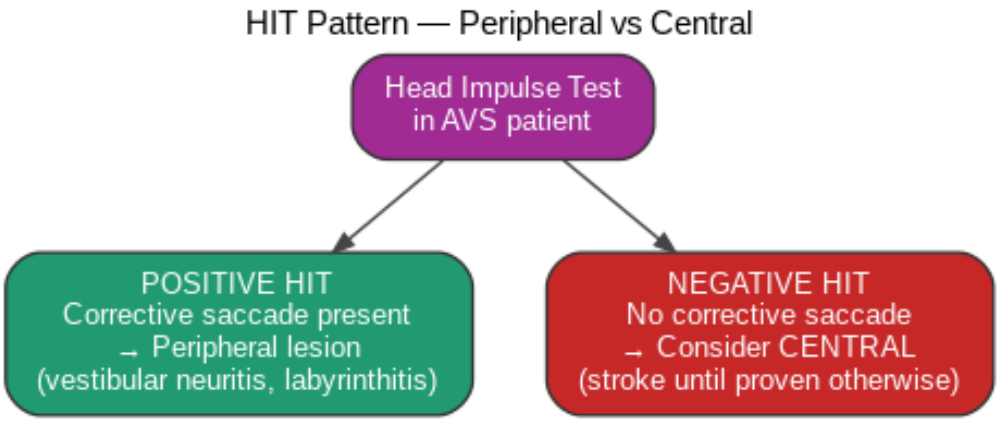


Figure 3. Interpreting HIT — peripheral (positive) vs central (negative) patterns.

Positive HIT (Abnormal VOR)

A positive HIT is defined by a corrective catch-up saccade occurring during or immediately after the head thrust, indicating that the VOR failed to maintain gaze stability [3,5]. The saccade is always directed opposite to the head thrust — if the head is thrust to the right and the VOR is impaired, the eyes are dragged to the right with the head and must saccade back left to refixate [3,13].

Corrective saccades are classified as overt (visible to the naked eye after the thrust) or covert (occurring during the thrust, typically <100 ms, often invisible at the bedside) [13,14]. Video HIT captures both; bedside HIT captures only the overt saccade, which is one of the reasons naked-eye HIT has imperfect sensitivity for peripheral vestibular hypofunction [13,14].

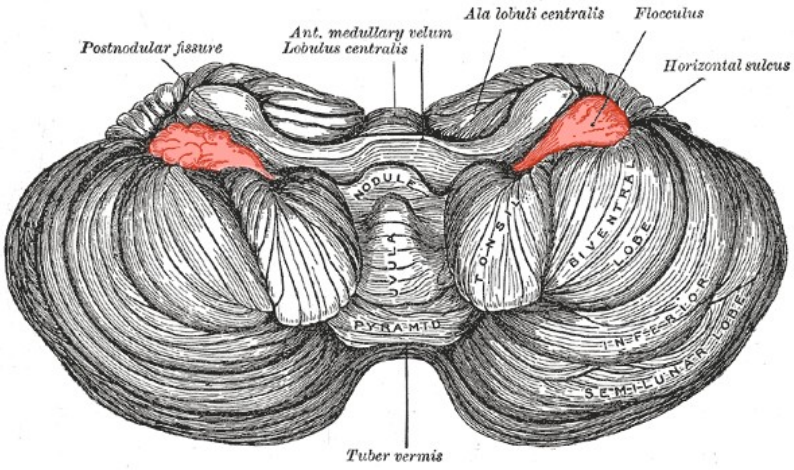
A positive HIT in a patient with AVS strongly favours peripheral pathology — most often vestibular neuritis, labyrinthitis or Ménière's disease [5,15]. However, 'positive HIT' is not synonymous with 'benign' — AICA-territory stroke can mimic vestibular neuritis with a positive HIT when the infarct includes the labyrinth or root entry zone [4,11,16].

Negative HIT (Normal VOR)

A negative HIT means the eyes remain locked on the target throughout and after the thrust, implying an intact VOR arc on both sides [3,5]. In a patient with ongoing AVS, a negative HIT is dangerous: it signals that the peripheral vestibular system is working normally, and the dizziness must therefore be generated centrally [5,11,12].

A negative HIT carries three possible meanings: (1) the patient has a normal peripheral vestibular system and no acute vestibular disease, (2) the patient has a central lesion that spares the VOR arc — cerebellar or small brainstem stroke being the dominant concern, or (3) the test was technically inadequate [3,5,11]. In the AVS context, option (2) dominates until proven otherwise [5,11,12].

V. The Dangerous Negative HIT



Anatomical Plate 4. The cerebellar flocculus and vestibulocerebellum — the site of central VOR modulation. A stroke here can spare the peripheral VOR (negative bedside HIT) while producing severe vertigo and nystagmus.

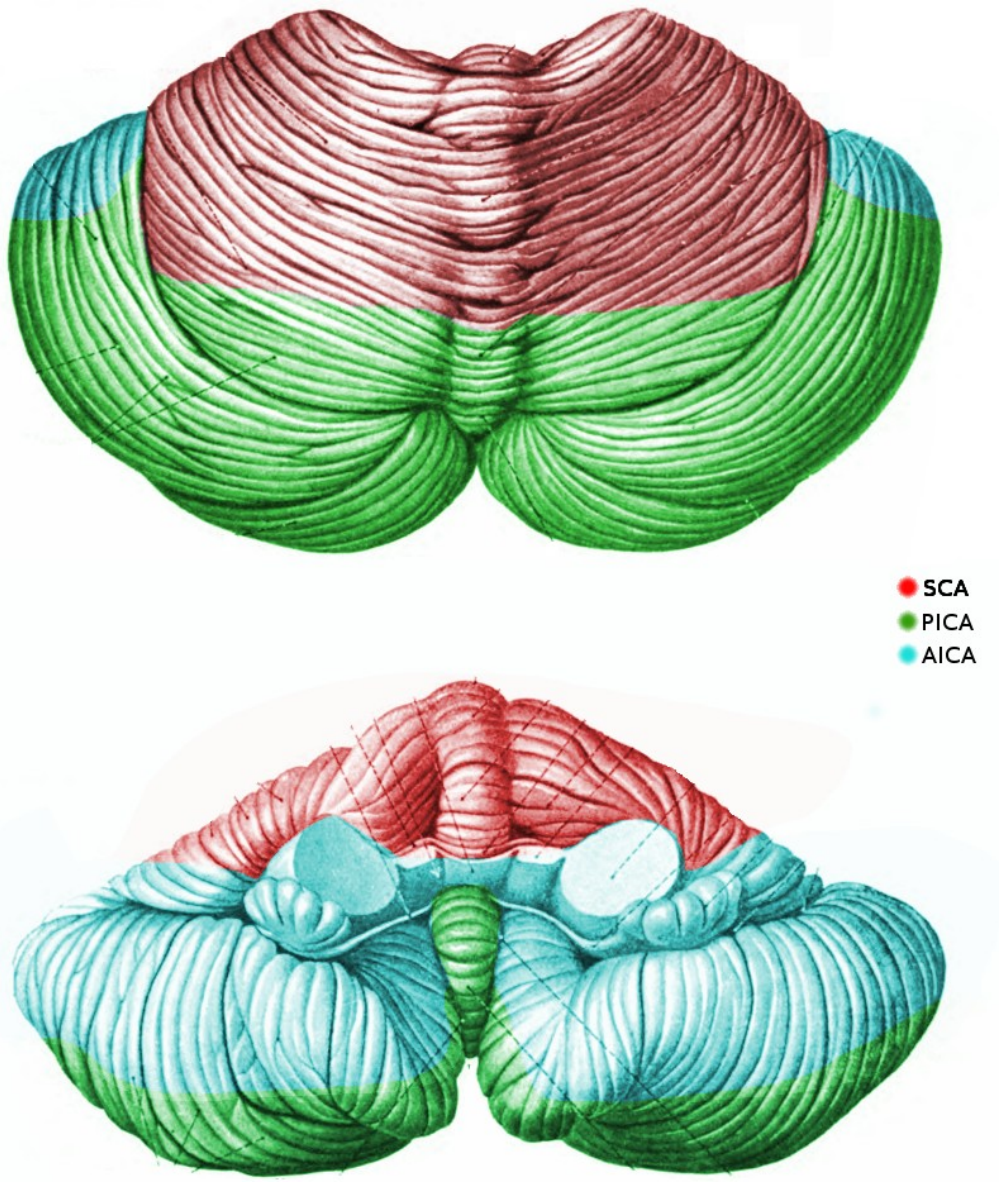
Source: Henry Vandyke Carter, Gray's Anatomy (1918); retouched by Hellerhoff. Public Domain.

When Normal VOR Coexists with Central Pathology

The ‘dangerous negative HIT’ is the single most important pitfall in acute vestibular assessment [5,11,12]. A negative HIT in a patient with spontaneous nystagmus and AVS should prompt urgent central workup — not reassurance — because cerebellar and brainstem strokes frequently produce a normal bedside HIT [5,11,17] [14].

This pattern occurs because the VOR arc is short and anatomically compact; strokes in the cerebellum, lateral medulla or distal vertebrobasilar territory commonly spare it while producing florid vertigo and nystagmus [11,17,18]. HIT therefore cannot rule out stroke — it can only rule in peripheral disease when paired with the rest of HINTS [5,8,12].

Examples of Central Disease with Negative HIT



Anatomical Plate 5. Cerebellar arterial territories — AICA, PICA, and SCA. AICA supplies the inner ear as well as the lateral pons and anterolateral cerebellum; PICA supplies the inferior cerebellum and is the classic territory for the dangerous normal HIT.

Source: CFCF, Wikimedia Commons, CC BY-SA 3.0. Based on Sobotta's anatomy plates.

Classical examples of central disease with a negative HIT include lateral medullary (Wallenberg) stroke, PICA-territory cerebellar infarcts, and small pontine infarcts that spare the VOR arc [11,17,18]. In each, focal neurological findings (Horner syndrome, crossed sensory loss, dysphagia, truncal ataxia) are the clue — if you look for them [11,17].

Prevention: Always Examine the Whole Patient

Never rely on HIT in isolation [5,8,12]. The examination must include nystagmus characterisation, test of skew, gait testing, cranial nerve and cerebellar assessment [5,8,11]. A full HINTS+ (HINTS plus bedside hearing and gait) adds additional sensitivity for posterior-circulation stroke in AVS [8,12].

If HIT is negative in a patient with AVS — or if any other finding suggests central disease — proceed with urgent neuroimaging [5,12,19]. MRI-DWI is the standard, accepting that sensitivity in the first 24–48 h

can be 80–85% in small cerebellar and brainstem infarcts, so a negative scan early does not fully exclude stroke [19,20].

⚠ **Important:** A negative HIT does NOT rule out brainstem or cerebellar stroke. Always examine the entire patient.

VI. Video Head Impulse Test

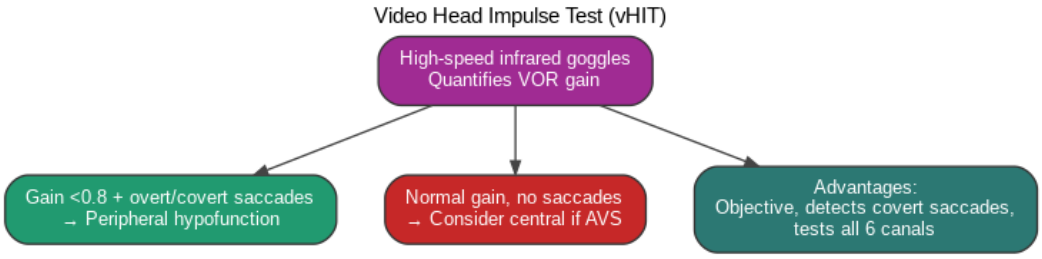


Figure 4. Video HIT — quantitative VOR gain and covert saccade detection [16].

Technology and Advantages

Video HIT (vHIT) uses high-speed infrared cameras in a lightweight goggle to track eye position at 250 Hz and simultaneously record head velocity, allowing quantitative measurement of VOR gain (eye velocity / head velocity) and automated detection of overt and covert corrective saccades [13,14]. Gain <0.8 with replicable corrective saccades is the standard criterion for peripheral hypofunction [14] [10].

The principal advantages of vHIT over bedside HIT are: (1) quantification of gain, (2) detection of covert saccades, (3) testing of all six semicircular canals, and (4) objective documentation for follow-up [13,14]. Limitations include cost, the need for a technically adequate recording, and artefacts from goggle slip, blinks and poor fixation [13,14] [10].

Clinical Application

vHIT is increasingly available in neuro-otology and stroke centres and is being integrated into ED evaluation of AVS [14,16]. It is particularly useful for detecting AICA-territory strokes masquerading as vestibular neuritis, and for identifying subtle bilateral vestibular hypofunction missed at the bedside [14,16] [10].

 Clinical Pearl: vHIT detects covert saccades and quantifies mild VOR deficits better than bedside HIT, but does not change fundamental interpretation.

VII. Common Pitfalls and False Results

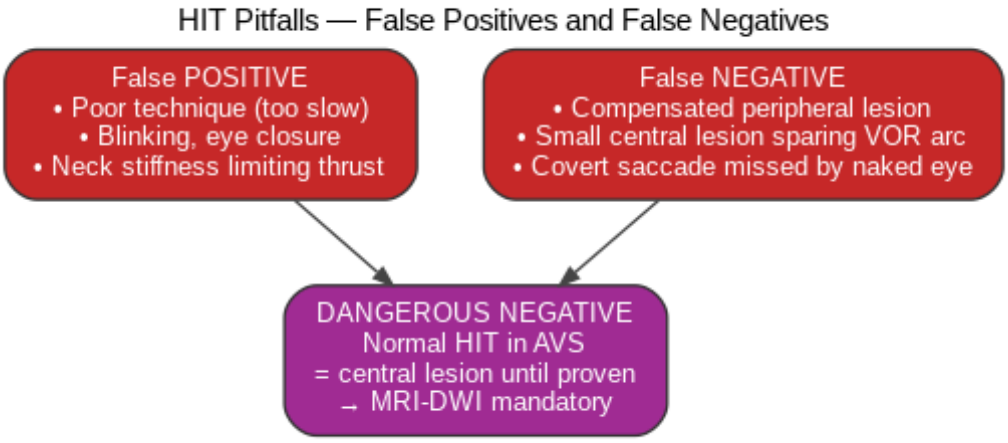


Figure 5. HIT pitfalls — false positives, false negatives, and the dangerous negative [14].

False Positives

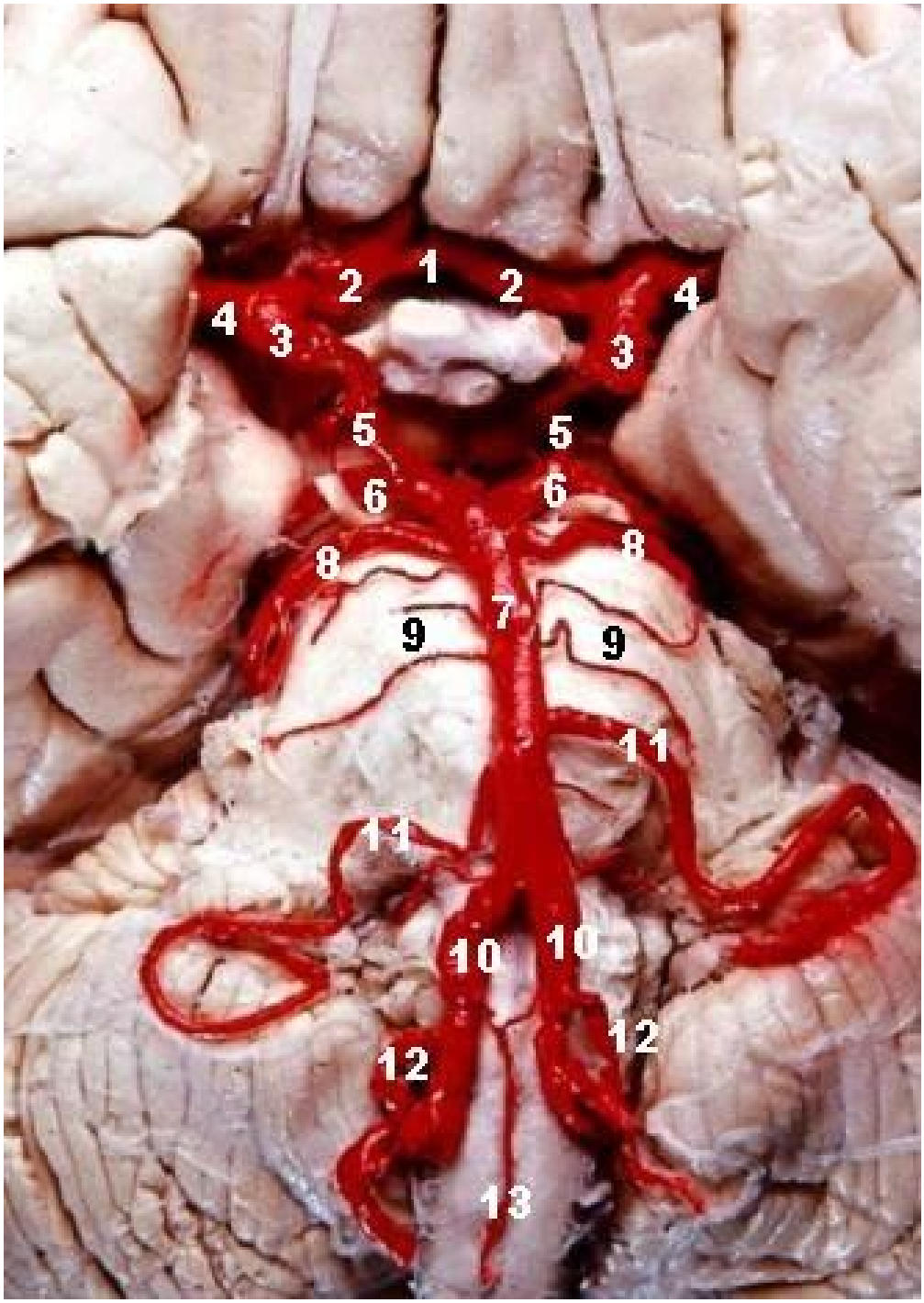
False-positive HIT can occur in BPPV during an active paroxysm, in patients with poor fixation or eye pathology, and when technique is slow or predictable [3,13]. Careful history (episodic vs sustained vertigo) and Dix–Hallpike testing usually clarify BPPV; repeating HIT in a quiescent interval reduces the false-positive rate [13,15].

False Negatives

False-negative HIT is most often seen in well-compensated chronic peripheral hypofunction, where covert saccades occur during the thrust and are invisible to the naked eye [13,14]. Small central lesions that spare the VOR arc produce a genuinely negative HIT — the reflex arc remains intact and no corrective saccade appears — even in the presence of florid posterior fossa pathology; this is the anatomical basis of the ‘dangerous negative’ [5,11,12].

 Key Point: *Covert saccades are common in mild peripheral lesions and easily missed at bedside. If suspicion is high and bedside HIT appears normal, vHIT may reveal the defect.*

VIII. HIT in Clinical Context: HINTS



Anatomical Plate 6. Blood supply of the human brainstem — the vertebrobasilar arterial system. HIT interrogates a functional pathway that spans peripheral labyrinth, cerebellum, and brainstem, all of which can be compromised by posterior circulation stroke.

Source: John A. Beal, PhD, LSU Health Sciences Center Shreveport. Wikimedia Commons, CC BY 2.5.

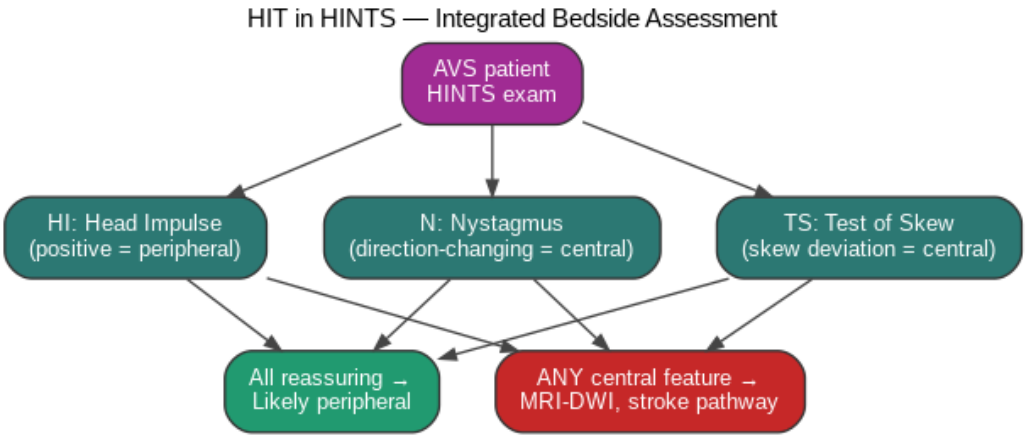


Figure 6. HIT within HINTS — integrated bedside assessment for AVS.

HINTS: Head Impulse, Nystagmus, Test of Skew

HINTS (Head Impulse, Nystagmus, Test of Skew) is a three-step bedside protocol validated for acute vestibular syndrome [5,8]. In experienced hands, a 'peripheral' HINTS pattern — positive HIT, direction-fixed horizontal nystagmus, no skew — has reported sensitivity approaching 100% and specificity of 95–96% for peripheral disease, outperforming early MRI for ruling out stroke in AVS [5,8,12].

A 'central' HINTS pattern — any one of negative HIT, direction-changing nystagmus, or skew deviation — has high sensitivity for posterior-circulation stroke and mandates urgent MRI and stroke pathway activation [5,8]. Adding bedside finger-rub hearing and gait assessment (HINTS+) further increases sensitivity for AICA and cerebellar infarcts [8,12].

A critical caveat: HINTS is validated only in patients with an ongoing acute vestibular syndrome — continuous vertigo, nystagmus, and head-motion intolerance — NOT in episodic vertigo, positional vertigo or in patients whose symptoms have resolved [5,8]. Applying HINTS outside this population generates misleading results and has caused real clinical harm [8,11].

 Clinical Insight: HINTS is powerful but is one component of evaluation. Never substitute HINTS for full history, complete neuro exam, and imaging when stroke is suspected.

Table 1 summarises HIT interpretation alongside the other HINTS components; clinicians should treat the pattern as a whole rather than any single element in isolation [5,8,12].

HIT Result	VOR Status	Primary Implication	Critical Note
Positive (corrective saccade)	Impaired	Peripheral vestibular hypofunction	Does NOT exclude concurrent central disease
Negative (no saccade, eyes fixed)	Normal	Intact VOR arc	Does NOT exclude brainstem/cerebellar stroke
Covert saccades only (vHIT)	Mild impairment	Mild peripheral loss or early compensation	Can be missed at bedside
Asymmetric (positive one side)	Asymmetric impairment	Unilateral peripheral lesion	Side of positive HIT = affected apparatus

IX. Conclusions

The head impulse test is a rapid, physiologically grounded bedside probe of peripheral vestibular function and an essential component of HINTS for acute vestibular syndrome [1,5,8]. Performed well, it substantially improves diagnostic accuracy at the bedside; performed poorly or interpreted in isolation, it misleads [3,5,13].

The critical pitfall remains over-reliance on HIT in isolation, especially the misinterpretation of a negative HIT as reassurance in a patient with genuine AVS and central features [5,11,12]. A negative HIT in AVS should raise, not lower, clinical concern for posterior-circulation stroke [5,11,12].

Proper technique — small amplitude, high acceleration, unpredictable timing, watching the eyes not the head — is essential for accurate results [3,13]. Video HIT adds objectivity and sensitivity and is increasingly available in ED-adjacent services [13,14]. The bedside examination, however, remains the test that every ED clinician must master [3,5].

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