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SHORT REPORTS

An Algorithm for the Diagnosis of Vestibular, Cerebellar, and Oculomotor Disorders Using a Systematized Clinical Bedside Examination

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Abstract
The bedside examination associated with their clinical history remains the most critical means to accurately diagnose the cause for most of the signs and symptoms related to pathology of the cerebellum and vestibular system in patients presenting with dizziness and imbalance. This paper focuses on those critical bedside examinations, suggests when laboratory testing might be useful to confirm the clinical suspicion, and considers the shared neural circuitry within the visual and vestibular systems to offer an algorithmic approach in conducting the clinical bedside examination.

Keywords Eye movements · Vestibulo-ocular reflex · Pathologic nystagmus · Ocular motility disorders · Vestibule · Labyrinth · Cerebellum

Introduction

In recent years, there have been many advances in the bedside examination that have improved the accuracy in evaluating patients with vestibular symptoms. Concurrently, laboratory tests of vestibular function including videonystagmography, the video head impulse test, and improved resolution in imaging studies have also developed and can assist confirming the bedside examination.

To assist the clinical reasoning for these patients, it is essential to have a working knowledge of the anatomy and neurophysiology of the vestibulo-ocular reflex (VOR) as well as the brainstem and cerebellar regions that trigger and control eye movements.

The VOR stabilizes retinal images during head motion by moving the eyes at the same speed as the head but in the opposite direction. However, when the head moves, the visual system is also stimulated. Thus, the brain must integrate sensory afference from each of these systems, which have unique latency and velocity profiles. The labyrinths are very sensitive sensors of head acceleration and tilt; with its sensory afference requiring less than 10 ms to travel from the peripheral vestibular hair cells, through the brainstem and onto the oculomotor neurons that mediate the eye movements [1]. This short

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latency enables compensatory eye movements during brisk head oscillations. In contrast, visual information takes about 100 ms to travel from the visual cortex to the oculomotor neurons. This delay is much too long for the eyes to keep up with a briskly moving object [1]. For example, if one oscillates a piece of paper back and forth horizontally at a rate of about two cycles per second, it becomes apparent that the text is blurred. However, if the page is held still and the head instead oscillates at that same rate, one can readily read the text clearly.

The purpose of this review is to describe an algorithmic approach using clinical bedside examinations of the critical eye movements that indicate vestibular, brainstem, and/or cerebellar regions dedicated to the generation and mediation of eye movements.

Anatomical and Neurophysiological Clinical Correlates

1. Rapid horizontal eye movements are generated and controlled in the pontine region, namely the paramedian pontine reticular formation (PPRF). The primary neural integrators for horizontal eye rotation include are the nucleus prepositus hypoglossi and the medial vestibular nucleus (NPH-MVN).
2. Rapid vertical and torsional eye movements are generated and controlled in the midbrain and are mediated by the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) and interstitial nucleus of Cajal (INC).
3. The cerebellum provides immediate "online" mediation and long-term adaptive mechanisms that optimize ocular motor performance to meet the demands of the visual system [2]. One common oculomotor finding of spinocerebellar pathology is slow vertical saccades [3]. However, distinct regions within the cerebellum are associated with specific oculomotor pathologies:

a. The flocculus and paraflocculus (cerebellar tonsil) are implicated in downbeat nystagmus, rebound nystagmus, gaze evoked nystagmus, pathologic smooth pursuit, and pathologic VOR suppression.
b. The nodulus and ventral uvula are implicated with periodic alternating nystagmus (PAN), central positional nystagmus, and head shaking-induced nystagmus.
c. Dorsal vermis lesions cause hypometric saccades.
d. Caudal fastigial nuclei lesions cause hypermetric saccades [4].

Bedside Relevance: The visual and vestibular system share similar neural circuitry responsible for eye movements [5]. Therefore, to accurately localize the site of lesion, clinicians must perform separate and combined evaluations of the visual and vestibular systems.

Static Evaluation of Vestibular and Visual Systems (Fig. 1)

Head Tilt

A tilt of the head to one side (cervical lateral flexion) in a patient may suggest a trochlear nerve palsy or a pathologic ocular tilt reaction (OTR). Patients with trochlear nerve palsy tilt their head in the direction of the healthy (lower) eye given the superior oblique muscle no longer maintains the ipsilesional eye in a centered position. Therefore, the contralesional head tilt engages the utricular ocular reflex to elevate the lowered eye. A pathologic OTR occurs when there is an imbalance in the otolith-ocular and otolith-spinal pathways. In addition to the head tilt, two oculomotor signs may also present and occur on the side ipsilateral with the tilted head: skew deviation and roll of the eyes [6]. In peripheral vestibular damage or lesions within the vestibular nuclei, there will be a head tilt (and if present, the oculomotor signs) toward the affected side. In contrast, lesions within the rostral pons and midbrain usually present with a head tilt (and oculomotor signs) away from the affected side [7].

Subjective Vertical Visual (SVV)

A tilt of the subjective vertical visual is usually caused by a disturbance in the otolith-ocular pathway and often in the same direction as the OTR [8]. It can be measured at the bedside using the bucket method [9].

Spontaneous Nystagmus

Spontaneous jerk nystagmus is a clinical sign of a tonic imbalance at the level of the vestibular nuclei between the afferent inputs derived from the semicircular canals of each labyrinth. Peripheral (i.e., 8th cranial nerve) causes for spontaneous nystagmus typically increase or are only present when visual fixation is blocked, with the fast phase beating toward the healthy labyrinth. In contrast, central causes for spontaneous nystagmus often has its fast phase beating toward the side of the lesion, is typically present in room light (without visual fixation), and often demonstrate a corrective saccade returning the eyes to their primary position during visual fixation testing (i.e., saccade testing) [6].

Ocular Lateropulsion

Occasionally, it is possible to identify pathology by having the patient close and then open their eyes. For example, in Wallenberg's syndrome, the eyes may deviate toward the side of the lesion under closed lids, which when the eyes are opened signals the pons to generate a horizontal corrective saccade back to primary position.

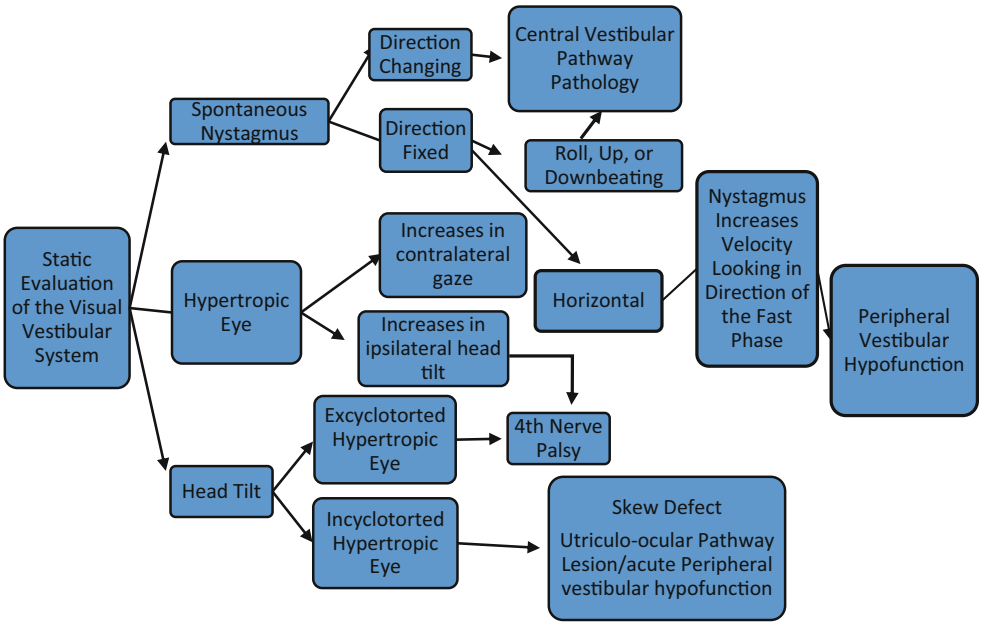

Fig. 1 Algorithm for the bedside static evaluation of the visual vestibular system. This examination should be conducted in part, in room light and with fixation blocked (i.e., Frenzel lens)

Examination of the Visual System in Isolation

Ocular Alignment

The cover test is very important in the evaluation of patients with diplopia or ocular misalignment. The patient is asked to fix his gaze on a small visual target and the eyes are covered alternately. The examiner looks for movements from the eye that has just been uncovered. When there is a vertical misalignment, the differential diagnosis typically will include a trochlear nerve palsy (i.e., superior oblique palsy) or a skew deviation (utriculo-ocular pathway lesion) [6]. To help distinguish these two causes, the clinician should consider that in the fourth nerve palsy when the patient close his eyes, the magnitude of head tilt may diminish, though not always [10]. Additionally, clinicians may use the cover test combined with the Parks-Bielschowsky three-step. The first step in the three-step test is to determine which eye is elevated (hypertropic) in primary position. Next step is to determine whether the elevated eye increases on right or left gaze. The last step is to determine if the elevated eye increases upon left or right head tilt. In the case of a right hypertropia due to superior oblique palsy, the magnitude of the right eye elevation will increase in left gaze and right head tilt. Clinicians may use prisms to quantify the hypertropia in each of the steps [11].

Examining for reduced torsion during the head tilt test alone may also help, though caution to the clinician using the head tilt exclusively as it can be misleading [12]. During right head tilt, the right eye normally incyclotorts (Superior Oblique and Superior Rectus), while the left eye excyclotorts (Inferior Oblique and Inferior Rectus). In 4th nerve palsy, the right eye elevation increases during right head tilt. Additionally, a fourth nerve palsy will create an excyclotorsion of the hypertropic eye, where a skew deviation may be accompanied with incyclotorsion of the hypertropic eye [13]. One final clinical suggestion that can help distinguish a fourth-nerve palsy from skew deviation (i.e., utriculo-ocular pathway lesion) is to repeat the cover/uncover test while the patient is supine. In the case of a skew identified in sitting due to utriculo-ocular asymmetry, this may reduce when positioned supine [14].

Spontaneous Nystagmus Pattern With and Without Fixation and Gaze-Evoked Nystagmus

The effect of removing visual fixation on nystagmus can be appreciated using Frenzel lenses, or during occlusive ophthalmoscopy. For examination of the gaze-evoked nystagmus, the patient is asked to fix his gaze on a small target 20 degrees to the left, right, up, and down always returning to the central position, typically held for ~20 s. When a horizontal nystagmus appears in only one direction of eccentric gaze, the cause is typically a unilateral peripheral vestibular lesion. The pathologic gaze-evoked nystagmus due to brainstem or cerebellar lesions is usually direction changing in the horizontal plane (i.e., when the patient looks to the right, the fast phase beats to the right; when the patient looks to the left, the fast phase beats to the left) [15].

Characteristics of Peripheral Vestibular Lesions

a. Nystagmus increases or only appears with the removal of fixation.
b. Nystagmus increases in intensity when the patient looks in the direction of the quick phase (Alexander's Law).

c. Nystagmus will primarily be horizontal though may include a torsional component, suggesting a more severe unilateral loss of function [14].
d. An eye motion induced from a head motion will obey Ewald's first law (the eye will always rotate in a plane parallel to the stimulated canal), independently of the position of the eye in the orbit.

Characteristics of Central Lesions

a. The suppression of the nystagmus by fixation is usually impaired in patients with unilateral brainstem or cerebellar lesion.
b. Persistent nystagmus that is purely vertical or torsional during eccentric gaze is always central.
c. Central nystagmus is often modulated by vergence.
d. Central nystagmus has smaller amplitude when the eyes move in the direction of the quick phase.
e. VOR gain may be elevated above normal in early stages of cerebellar disease (i.e., SCA6, often easiest to document using video head impulse testing) [16].

Saccades

Saccades are best examined at the bedside by having a patient look alternately at two targets held apart horizontally or vertically, such as the examiner's finger and nose. The velocity, accuracy, trajectory, and ability to conjugate should be noted [1, 15].

For Horizontal Saccades

a. A discrete lesion of the PPRF causes a conjugate horizontal saccadic palsy to the same side.
b. A lesion of the medial longitudinal fasciculus causes ipsilateral adduction palsy; the cardinal manifestation of internuclear ophthalmoplegia.
c. A lesion of the NPH-MVN (often termed a leaky integrator) causes horizontal gazed-evoked nystagmus.

For Vertical Saccades

a. A lesion of the riMLF causes slowing of downward saccades.
b. A lesion of INC (often termed a leaky integrator) causes torsional nystagmus.
c. A lesion of the fastigial nucleus will cause hypermetric saccades.
d. A lesion in the cerebellar vermis will cause hypometric saccades [17].

Smooth Pursuit

The bedside exam for smooth pursuit tasks the patient to visually track a small target with the head still. Smooth pursuit is generated from many cortical regions, making it difficult to localize sites of pathology. Generally, catch-up saccades are seen when the gain is low, while backup saccades are observed when the gain is high.

Vergence

The patient is asked to fixate on a small target as it is brought toward the bridge of the nose or asked to shift gaze between a near and far object. The vergence system is the first oculomotor system to be affected by fatigue, alcohol, and other drugs, and defective vergence is a common cause of strabismus and diplopia [1].

Optokinetic Nystagmus

Optokinetic nystagmus (OKN) is induced reflexively by motion of a larger visual scene, which causes an illusionary sensation of self-rotation in the opposite direction [1]. It is a simple test that evaluates smooth pursuit and saccades in the horizontal and vertical planes. This test can be useful to evaluate non-collaborative patients and children.

Examination of the Vestibular System in Isolation

Positional Tests

Positional testing is an essential part of the vestibular examination, particularly for patients with reports of positional vertigo. Two maneuvers must be performed, and the patient should wear Frenzel lenses to block fixation:

a. The Dix-Hallpike maneuver, to detect otoconia displaced (canalithiasis or cupulolithiasis subtypes) into the vertical semicircular canals. The patient's head is 1st rotated 45° right (or left) relative to the trunk and then moved from upright to the supine head-hanging position.
b. The supine-roll maneuver to detect otoconia displaced into the lateral semicircular canals (canalolithiasis or cupulolithiasis). The patient is position in supine with head elevated ~ 20 deg, then the head is rotated right and then left.

Accurate diagnosis of the affected semicircular canal and type of positional vertigo depends on the characteristics of the positional evoked nystagmus and not on the maneuver that is performed.

Vibration Induced Nystagmus

Low frequency vibration (60 or 100 Hz) applied to the mastoid tip or vertex may induce nystagmus in patients with unilateral loss of vestibular function and occasionally in other conditions, such as anterior canal dehiscence. In the case of a unilateral loss of function, the slow phase of the nystagmus usually drifts toward the side of the paretic ear [18, 19]. When vibration elicits a vertical nystagmus, a central lesion should be suspected. A vibration-induced torsional or vertical nystagmus may also occur with an anterior canal dehiscence [20].

Head Shaking Nystagmus (HSN) and Head Shaking Tilt Suppression (HSTS)

The head-shaking nystagmus (HSN) test is considered a useful clinical tool for detecting asymmetries between the vestibular labyrinths and provides some insight into the integrity of the velocity storage system [21]. Shaking the head with an oscillation about 2 Hz for approximately 20 s in the horizontal plane may cause a horizontal nystagmus where the fast phase beats toward the unaffected labyrinth [22]. This finding suggests a peripheral vestibular hypofunction. On the other hand, a vertical nystagmus after a horizontal head shaking typically suggests pathology affecting the central vestibular pathways [23].

For the HSTS test, the patient is instructed to tilt his head forward until the chin rests on the upper thorax, immediately after the passive horizontal head shaking. Maia et al. [24] suggest that tilting of the head forward after first applying horizontal head-shaking (HSTS test) is much less effective at suppressing the induced nystagmus in patients with a central cause for their vestibular-like symptoms.

Hyperventilation-Induced Nystagmus

Hyperventilation is performed in a sitting position through quick and deep respiratory cycles for 60 s. The evoked nystagmus is due to metabolic changes, including extracellular alkalosis, hypoxia, and hypocapnia [25], and can beat toward the affected ear in demyelinating lesions [26].

Hyperventilation can also induces nystagmus in patients with compression of the vestibular nerve by a tumor (e.g., vestibular schwannoma) or small blood vessel loops (microvascular compression) or with demyelinating diseases affecting the central pathways (e.g., multiple sclerosis) [6].

Valsalva-Induced Nystagmus

The Valsalva maneuver can induce nystagmus either by increasing intracranial pressure (straining against closed glottis (as in lifting a heavy weight) or by increasing pressure in the middle ear (blowing out against pinched nostrils). The nystagmus may be induced in patients with cranio-cervical junction anomalies, such as Chiari malformation, with perilymphatic fistulas, or anterior canal dehiscence. Jugular venous compression may also increase the intracranial pressure and induce a nystagmus similar to that of the Valsalva maneuver [6, 27].

Tullio's Phenomenon

Tullio's phenomenon (noise-induced nystagmus and oscillopsia) often occurs in patients who have Valsalva-induced nystagmus and is commonly associated with superior canal dehiscence [5] and perilymphatic fistula.

Dynamic Examination of the Visual System and Vestibular Systems (Fig. 2)

Dynamic Visual Acuity

Dynamic visual acuity is a functional measure of the VOR and is assessed by asking the patient to read while the head is oscillated horizontally, vertically, or in the roll plane (from ear to shoulder) at a relatively high frequency (~2 Hz). Normal individuals may lose one or two lines of acuity with head rotation, whereas patients with vestibular abnormalities often lose three or more [6]. In cases where the clinician suspects the patient is exaggerating the amount of gaze instability, dynamic visual acuity can be measured during passive head roll. During roll, the axis of eye rotation remains nearly perpendicular to the fovea, resulting in minimal displacement between the fovea and fixation target image projected onto the fovea [28]. Because DVA is a functional test known to convey evidence of compensation after a peripheral vestibular hypofunction, false negatives limit its diagnostic acumen [29, 30].

Visually Enhanced Vestibulo-Ocular Reflex (VVOR)

The bedside examination of the VVOR test requires a low frequency head stimulation. The subject is asked to stare at an earth-fixed target (i.e., dot on the wall or the examiners nose). Next, the head is slowly moved at about 0.5 Hz and with an amplitude of 10° in the horizontal plane. The patient is asked to maintain the fixation on the dot during the whole test. An abnormal test is noted when the patient is unable to keep the eyes focused on the earth fixed target, and instead generates reflexive saccades to catch up with the target. For clinicians with access to vestibulo-oculography equipment (VOG), patients with unilateral vestibular hypofunction exhibit corrective saccades beating to the healthy side during head rotation to the affected side, while patients with bilateral vestibular hypofunction exhibit corrective saccades beating to the opposite side of the head movement, for both sides

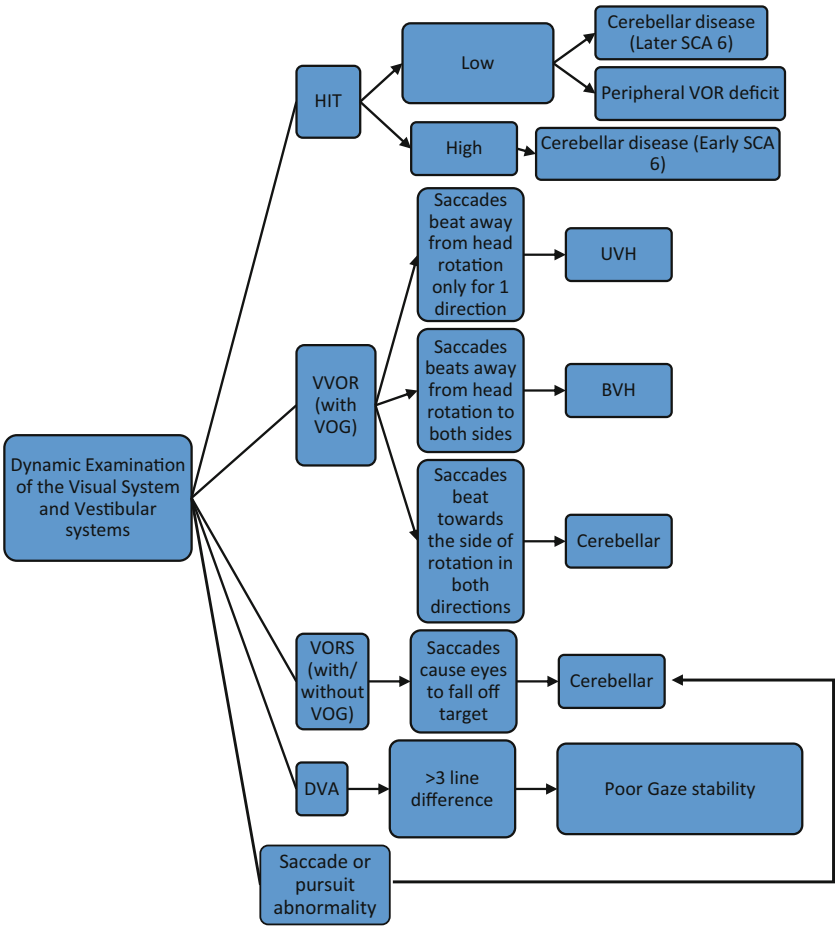

Fig. 2 Algorithm for the bedside dynamic examination of the visual and vestibular systems, considering laboratory equipment if available to the clinician. *HIT* head impulse test; *DVA* dynamic visual acuity; *VOR* vestibular ocular reflex; *VOG* video oculography; *VVOR* visual vestibular ocular reflex; *VORS* vestibular ocular reflex suppression; *UVH* unilateral vestibular hypofunction; *BVH* bilateral vestibular hypofunction; *SCA* spinal cerebellar atrophy

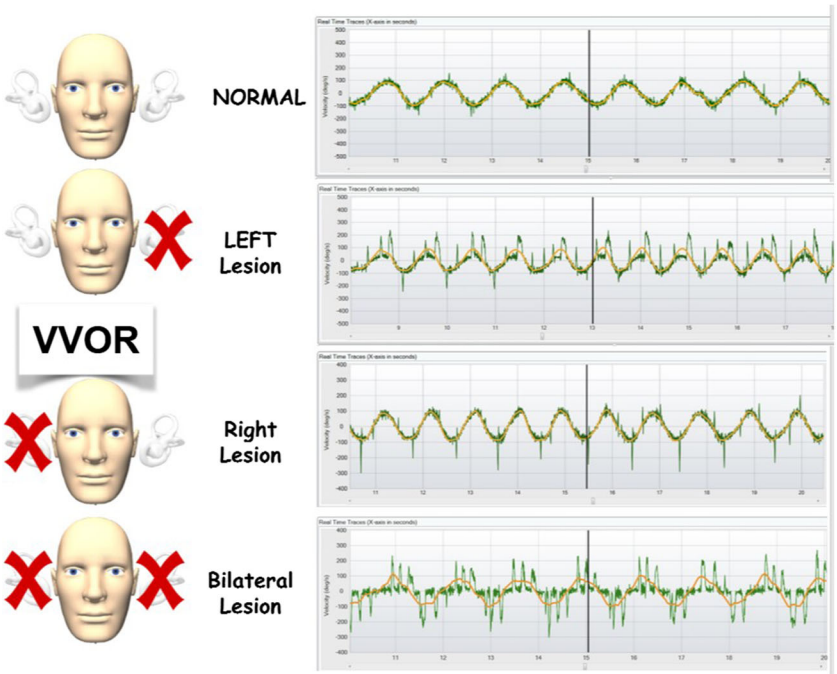

Fig. 3 Visual vestibulo-ocular reflex with video-oculography (head moving and target fixed) in controls, patients with unilateral vestibular hypofunction affecting the right and left sides, and patients with bilateral vestibular hypofunction. The red X denotes the lesion side. By convention, the top half of the 4 plots reflect head rotation to the right (yellow) and saccades beating to the right (green) and the bottom half denotes head rotation to the left (yellow) and saccades beating to the left (green) [31]

Fig. 4 Vestibulo-ocular reflex suppression with video-oculography (target and head moving) in controls, patients with unilateral vestibular hypofunction affecting the right and left sides, and patients with bilateral vestibular hypofunction. The red X denotes the lesion side. By convention, the top half of the 4 plots reflect head rotation to the right (yellow) and saccades beating to the right (green) and the bottom half denotes head rotation to the left (yellow) and saccades beating to the left (green) [31]

(Fig. 3) [31]. Patients with cerebellar lesion exhibit catch-up saccades to the same side of head movement for both sides [15].

Vestibulo-Ocular Reflex Suppression (VORS)

For the VORS, the patient looks at a finger of his extended arm while he turns smoothly at the waist, from side to side with arm, trunk, and head en bloc [32]. An abnormal test is noted when the patient is unable to keep the eyes focused on the moving target, and instead generates reflexive saccades to catch up with the target. For clinicians with access to VOG, a distinct pattern emerges: patients with unilateral vestibular hypofunction exhibit corrective saccades to the healthy side when the head is moved to this side, while patients with bilateral vestibular hypofunction do not show corrective saccades during head movement to either side (Fig. 4) [31]. Failure of VORS in the absence of spontaneous nystagmus indicates a lesion within the basal ganglia, otherwise we would expect a concurrent spontaneous nystagmus as occurs for lesions impairing the posterior fossa or supratentorial cortex [33].

Head Impulse Test (HIT)

The patient is asked to maintain fixation on a fixed target (i.e., the examiner's nose), then the patient's head is rapidly rotated to one each side, separately. This is a high-acceleration head rotation. Patients with healthy function are able to maintain gaze fixation on the target. In patients with vestibular hypofunction, when the head is rotated to the affected side, the eyes move with the head and a corrective saccade is then generated to bring the eyes back to the target. This catch-up saccade indicates peripheral vestibular hypofunction on the side toward which the head was rotated [34].

Conclusion

A systematized clinical bedside examination that includes a static evaluation of the patient's posture, evaluations of the eyes alone, of the vestibular system alone, and of the combined visual and vestibular system is of great help to diagnose vestibular, cerebellar, and oculomotor disorders, due in part to the fact that the visual and vestibular systems share neural circuitry and neural output.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

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