

ED EXTRA EYE MOVEMENTS DIAGNOSIS & INTERPRETATION: OSCILLOPSIA, NYSTAGMUS, SACCADIC INTRUSIONS ET AL

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Introduction

Oscillopsia is the illusion of movement of the environment, and is most often due to nystagmus or some other unwanted intrusive eye movements. Although not always a true emergency, oscillopsia or the associated symptoms often trigger ED presentation; because most ED personnel are not well versed in abnormal eye movements, this frequently prompts urgent neurologic consultation.

While not all the disorders discussed in this section are true emergencies, some are included because they are quite common, and while others appear because of the useful information they provide to the clinician.

Definitions

Oscillopsia – the illusion of visual motion of the environment. This may be due to several areas of dysfunction, all of which are involved in stabilization of the visual world on the fovea under differing conditions of head, eye, and environmental movement. The most common cause of oscillopsia is nystagmus.

Differential Diagnosis of Oscillopsia

1. Nystagmus or nystagmoid eye movements
 - a. These movements take the fovea off the object of regard, creating oscillopsia and/or a sense of blur as the object image slides across the fovea thus eluding focus. This category encompasses both nystagmus and saccadic intrusions. The accompanying symptoms depend upon the root cause of the eye movement disorder and associated areas of dysfunction.
2. Vestibular ocular reflex (VOR) dysfunction
 - a. A hypoactive or impaired VOR fails to keep the visual object of regard on the fovea as the head accelerates. With unilateral vestibular nerve damage, head acceleration toward the damaged side does not produce a normal compensatory eye movement to keep the visual object on the fovea, and oscillopsia ensues. If both vestibular nerves are hypofunctioning, then head acceleration to either side produces this effect; because the head is almost always in a state of acceleration (e.g., heartbeat induced vertical head movements), patients with vestibular hypofunction AU experience an almost constant sense of environmental movement. Bilateral symmetric VOR hypofunction does not produce nystagmus because the vestibular input remains symmetric (albeit impaired), thus no slow phase is generated. VOR hypofunction often occurs acutely in association with vertigo; while vertigo and acute vestibular nystagmus resolve, the VOR gain may remain abnormal leaving oscillopsia as the sole symptom.
3. Ophthalmoplegia
 - a. With impaired eye movements, the normal components of the VOR may work fine, but the eye muscle's ability to generate the appropriately directed compensatory movement is faulty, resulting in oscillopsia. Of course, these patients often also have diplopia and obvious impairment of extra-ocular movements.

Nystagmus – this is an abnormal eye movement that **has at least 1 slow phase**; jerk nystagmus is comprised of 1 slow phase followed by a corrective fast phase, while pendular nystagmus is made up of 2 back-to-back slow phases (no fast phase). Several different systems can produce nystagmus when they are malfunctioning, including the vestibular system and the gaze holding systems.

Saccadic intrusion – this is an abnormal eye movement comprised of JUST fast phases; saccadic intrusions do not have a slow phase. The most common saccadic intrusion is a square wave jerk, and these are observed in lower frequencies (less than 20/min) even in the healthy elderly patient.

Intersaccadic interval – following a normal saccade, there is typically a ~180-200 mSec pause before another saccade can be generated (the normal intersaccadic interval). Some saccadic intrusions demonstrate a normal intersaccadic interval, while others appear as back-to-back saccades sans an intersaccadic interval; this information in addition to the plane of the eye movement help classify saccadic intrusions.

Specific abnormal eye movements

Although several disorders may produce oscillopsia, we will focus on those abnormalities which are either common, or provide unique localizing or prognostic information.

1. Peripheral vestibular nystagmus:

Peripheral vestibular nystagmus is extremely common, and typically accompanied by vertigo. Additionally, the reverse is also true: if a patient has vertigo from peripheral vestibular dysfunction during the exam, the patient **MUST** have nystagmus (NB, this is a real time phenomena – the nystagmus accompanies the vertigo, and resolves with resolution of the vertigo – i.e., if you have symptoms now, you have to have signs now)! Nonetheless, it is common for a clinician to miss the nystagmus because many physicians do not understand how to look for this type of eye movement. A patient with peripheral vestibular nystagmus can suppress or ‘hide’ the nystagmus by fixing on an object; therefore, if the clinician fails to ‘block fixation’ the nystagmus can go undetected. There are several ways to block fixation allowing observation of peripheral vestibular nystagmus including the use of Frenzel lens, use of ophthalmoscopy, or just a bright fixation light. Frenzel lens are high plus lenses that blur vision for the patient while magnifying the eyes for the clinician. The ophthalmoscope is an excellent tool to detect subtle or peripheral vestibular nystagmus. The clinician views the optic disc, then covers the fellow eye; if the disc moves to the patient’s left, be aware that the front of the eye is moving to the patient’s right. Alternatively, shine a bright light into the patient’s eye while covering or closing the fellow eye, or placing a blank featureless paper close enough to the eye to fill the visual field can provide similar information.

Nystagmus with peripheral vestibular disorders typically has a mixed torsional-horizontal, or torsional-vertical jerk waveform (NOT pure vertical – e.g., downbeat – which indicates CENTRAL vestibular dysfunction).

“Mixed/no fix” – this refers to the characteristic of peripheral vestibular nystagmus to be suppressed by patient visual fixation, and maintain a mixed torsion-horizontal or torsion-vertical slow phase.

Benign paroxysmal positional nystagmus (BPPV) – this is the most common acute peripheral vestibulopathy, and is accompanied by typical torsional-vertical nystagmus during episodes. Most BPPV involves the posterior semicircular canal; the Dix-Hallpike procedure is performed by turning the head 45 degrees to right or left (placing the head in the plane of the posterior canal). The head is then rotated back by laying the patient down, causing any debris in the posterior canal to move with gravity. A burst of nystagmus helps make the diagnosis; for right posterior semicircular canal BPPV, the head is turned to the right, and nystagmus provoked upon laying down has a **slow phase which is down and counter-clockwise (from the patient perspective)**. Conversely, posterior semicircular canal BPPV AS produces a **slow phase** clockwise and down. This is not only the most common peripheral vestibulopathy, but also the easiest to treat. A repositioning maneuver, often performed from the Dix-Hallpike head hanging position, is ~70% effective at curing the disorder. Additional treatment (and teaching patients to perform the maneuver at home) often resolves all but the most refractory cases.

Acute vestibular syndrome (combination of vertigo, nystagmus, nausea/vomiting, head-motion intolerance, and unsteady gait) – the acute vestibular syndrome (AVS) can result from any of several different pathophysiologies, including viral (vestibular neuritis), endolymphatic hydrops (Meniere), trauma, or ischemia (cerebellar infarct). As a peripheral form of vestibular nystagmus, this can be suppressed by the patient with visual fixation. A lesion involving all 3 semicircular canals (or the vestibular nerve) on one side produces a typical pattern of nystagmus; right vestibular nerve dysfunction: slow phase clockwise and right, while left vestibular nerve dysfunction causes slow phases left and counter-clockwise. The historical and exam details assist the clinician in diagnosing the pathophysiology. Cerebellar infarcts can mimic peripheral vestibulopathies; 3 “HINTS” strongly suggest cerebellar infarction (HINTS = Head impulse, Nystagmus, Test of Skew):

1. Head Impulse (HI) - A NORMAL VOR gain per horizontal head impulse test,
2. Nystagmus (N) - direction-changing nystagmus in eccentric gaze (implying gaze evoked mechanisms)
3. Test of Skew (TS) - skew deviation (vertical misalignment of the eyes) is suggestive of a central origin.

Ocular misalignment such as skew is detected by alternate cover or red Maddox rod testing.

More benign peripheral vestibulopathies such as vestibular neuritis demonstrate an ABNORMAL VOR gain, fixed direction nystagmus, and no vertical misalignment of the eyes.

2. Central Vestibular Nystagmus: downbeat

Central vestibular nystagmus is easier to detect than peripheral vestibular nystagmus: while peripheral vestibular nystagmus can be suppressed by the patient with visual fixation, central forms of vestibular nystagmus are not suppressible by the patient. The most common forms of central vestibular nystagmus include downbeat, upbeat, and pendular. Among these types, downbeat is by far the most common. Downbeat is best detected by having the patient look to the side and down (e.g., right and slightly down) where a slow phase up with down beat is most evident. Some patients ONLY have downbeat in down and lateral gaze, so unless patients are observed in this position of gaze, the diagnosis may go undetected. Downbeat indicates posterior fossa disease (brainstem or cerebellum), and does not occur in peripheral or 'ear' disease.

3. Gaze evoked & rebound

Gaze evoked nystagmus is the most common form of nystagmus clinicians encounter. This is provoked by having the patient look in eccentric gaze (horizontal gaze evoked nystagmus is more common than vertical, thus, right or left gaze are the more common positions to observe gaze evoked nystagmus), and the slow phase is always back toward primary position. The slow phase is related to the physical elastic properties of the orbit – rectus and oblique muscles plus other orbital tissue tend to bring the eye into primary position. The neural network that is responsible for overcoming these forces is known collectively as the neural integrator. When directing gaze to the side, a 'pulse' of innervation is delivered to move the eyes to the desired position of gaze, and is then followed by a 'step' of innervation to keep the eyes in this position (the step amplitude is the mathematical integral of the pulse, thus the network name neural integrator). A few unsustained beats of gaze evoked nystagmus may be seen in isolation in normal subjects, so separating pathologic from physiologic gaze evoked is always a concern. There are 3 features to help this differentiation:

1. Is the gaze evoked nystagmus sustained?
2. Is the gaze evoked nystagmus asymmetric (same in right and left gaze)?
3. Are other features of posterior fossa or cerebellar dysfunction such as rebound nystagmus present?

If any of these statements are true, pathology that requires investigation and explanation exists. Generally, greater than 3-4 beats of gaze evoked nystagmus is considered sustained. Other features of posterior fossa dysfunction include limb ataxia, diplopia, dysarthria, weakness, numb, gait dysfunction, etc. Rebound nystagmus is provoked by maintaining eccentric gaze for several seconds, then redirecting the patient's gaze back to primary position. Rebound nystagmus appears as a few beats with opposite directed slow phases to the prior gaze evoked direction (e.g., sustained right gaze with slow phase left is noted; the patient then returns to primary position, where slow phases to the right are present for a few beats). This is thought to represent the brain's attempt to dampen sustained gaze evoked nystagmus to one side. Asymmetric gaze evoked nystagmus militates for ipsilateral structural cerebellar system dysfunction.

Gaze evoked nystagmus is useful for localization, but does not yield information concerning pathophysiology; for this, the clinician must incorporate additional features of the history and exam in addition to ancillary testing (e.g., MRI, labs). Drugs such as anticonvulsants, anxiolytics, and alcohol commonly produce dose-related horizontal gaze evoked nystagmus.

4. Miscellaneous

Bobbing

Ocular bobbing is a rare and distinct ocular motility pattern that conveys important localization and prognostic information. Typical ocular bobbing implies usually non-rhythmic fast phase downward followed by a slower return to primary position in a coma patient in combination with absent spontaneous or reflex-induced horizontal eye movements (bilateral horizontal gaze palsy). This typically indicates a destructive lesion of the pons, and portends a poor prognosis.

Internuclear Ophthalmoplegia (INO)

Internuclear Ophthalmoplegia (INO) is included here because of its commonality. Abducting nystagmus is commonly present with INO and is often the first clue to the diagnosis (observed with pursuit movements when testing ductions even before saccades are tested). Many neurologists fail to recognize INO because they don't

routinely check saccades – most INOs are ONLY present with saccades! Vertical gaze evoked nystagmus is also typically present (slow phase back toward primary position in upgaze). Myasthenia gravis may mimic an INO; variability and associated features are the clues that assist with this differentiation.

Parinaud Dorsal Midbrain Syndrome

Parinaud dorsal midbrain syndrome is a distinct ocular motor syndrome with valuable localization information. There are 7 features of the syndrome; however, most patients do not have all the features:

1. Impaired vertical gaze
2. Square wave jerks
3. Vergence dysfunction
4. Skew deviation
5. Convergence retraction 'nystagmus'
6. Lid retraction
7. Light-near pupil dissociation

Among these features, convergence-retraction nystagmus is the most specific: this reliably localizes to the dorsal midbrain. Several different pathophysiologies may produce Parinaud syndrome including hydrocephalus (including shunt dysfunction), MS/demyelination, ischemia, or neoplasm (dysgerminoma).

Conclusions

Nystagmus and other causes of oscillopsia are frequently encountered in the ED. Working knowledge of peripheral vestibular nystagmus (mixed torsion/horizontal or torsion/vertical), central vestibular nystagmus (e.g., downbeat), and gaze evoked nystagmus (slow phase back to primary position) facilitate efficient localization in such patients.

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