

The Virtual Ocular Motor Exam:

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The Virtual Vestibular Exam:

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A TELEMEDICINE APPROACH TO THE OCULAR MOTOR & VESTIBULAR EXAMINATION

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1. Ocular Motor Examination

- **Range of movements** ([VIDEO](#)) <30 seconds to examine
 - *When should you evaluate?*
 - All patients, but especially when diplopia is a complaint.
 - *What technology is needed to examine?*
 - Any video platform will do for synchronous patient to provider examination. For asynchronous recordings, 30 frames/second and 720p HD or better are preferable.
 - *What is the ideal placement of the patient's camera in the home?*
 - While it is ideal for the examiner to see the fixation target and the patient's eyes simultaneously, a more close-up view of the eyes may be needed. In this case, the patient's face is 1 foot away from the smartphone/computer camera with the fixation target being held behind the camera (so the examiner cannot visualize it).
 - *What equipment/household items are needed to examine?*
 - Good lighting is essential for adequate visualization of the eyes and face. A small fixation target like the tip of a pen or pencil. Or, the patient can use their finger.
 - *Is assistance in the patient's home (e.g., family, caregiver) necessary?*
 - Possibly to stabilize the camera if a portable/smartphone camera is being used.
 - *What are the instructions for synchronous examination (e.g., examining the patient via video platform in real time)?*
 - Have the patient hold the small fixation target at arm's length. After evaluating fixation in primary gaze, have the patient move the target far to the right, left, up and down (and hold it in each position) so the range of movements can be evaluated in all planes. When looking down, the patient should lift both of their eyelids with one hand. Also, evaluate for the presence of gaze-evoked nystagmus in eccentric gaze. In some situations, having the patient look up/right, up/left and down/right, down/left can be helpful – e.g., a right superior oblique palsy with mild motility deficit noted in the paretic right eye with down/left gaze; a patient with a Chiari malformation may only have mild downbeat nystagmus in far down/lateral gaze. If double vision is present or a motility deficit is suspected, the range of movements of each eye individually should be evaluated – e.g., have the patient cover the right eye and evaluation the range of movements of the left eye in 9 cardinal positional of gaze, then cover the left eye and repeat.
 - *What are the instructions for asynchronous examination (e.g., having the patient or a provider [in the ED] record for the purposes of store and forward)?*
 - Turn on the video camera and switch on the light for the entirety of the video. Figure out how to stabilize the camera (mini-tripod, using 2 hands, etc) so that it's about arm's length from the patient's eyes, or place your left hand on the patient's head while holding the camera in your right hand while stabilizing the left side of the camera against your left arm. Make sure the patient's eyes are centered in the video. Either have the patient look as far as they can to the right, left, up and down, or have the patient hold a small fixation target (or their hand/finger) at arm's length in each direction of gaze. When looking down, the patient should lift both of their eyelids with one hand. Versions (both eyes viewing) and ductions (each eye individually viewing as the patient covers one of their eyes and then the other) can both be checked in this way.
 - *What are the limitations?*
 - A small motility deficit can be missed. If the patient has a complaint of double vision or if there is a concern for a mild motility deficit, the ocular alignment exam is particularly important.

- **Convergence** (VIDEO) – may bring out or cause reversal of vertical nystagmus (e.g., bring out DBN in a cerebellopathy, transition from UBN to DBN in Wernicke’s encephalopathy), or may exaggerate some acquired forms of nystagmus or damp congenital or infantile nystagmus. If the patient complains of binocular symptoms or double vision while reading and near viewing and the patient has a near point of convergence >10 cm, think about convergence insufficiency (particularly with parkinsonism [VIDEO] or TBI/concussion).
- **Alignment** (VIDEO) – start with alternate cover testing where one eye is occluded, and then the occluder (or examiner’s hand) is moved to the fellow eye, and then back and forth as the patient continues viewing the same (usually distant) target. Look for a horizontal (when the eye under cover is crossed in so that it has to move outward when uncovered to take up fixation – ESO [VIDEO]; when the eye under cover is deviated outward so that it has to move inward when uncovered to take up fixation – EXO [VIDEO]) or vertical (generally named after the side of the higher eye – e.g., if the right eye is uncovered and has to come down to fixate on the target, this is a right hyperdeviation – see HINTS table below under skew) movements of the uncovered eye. The eso-, exo-, hyper is further classified as a *tropia* (misalignment present with both eyes open – use a cover-uncover technique on each eye individually) or *phoria* (misalignment present when binocular vision is broken with alternate cover testing but no misalignment with cover-uncover).
- **Saccades** (VIDEO) – have the patient rapidly look back and forth between 2 visual targets, noting the speed, conjugacy, latency, and accuracy. First have the patient look between an eccentric target and the examiner’s nose horizontally and vertically, making assessment of accuracy easier – e.g., overshooting the nose (hypermetria) or overshooting the nose (hypometria). Then have the patient make larger amplitude saccades horizontally and vertically, which makes assessment of speed and conjugacy (e.g., adduction lag suggest an internuclear ophthalmoplegia [INO]) easier. Saccadic dysmetria is seen in cerebellar disease (or brainstem connections w/ cerebellum). Ipsilateral hypermetria and contralateral hypometria occurs in Wallenberg syndrome (VIDEO).
- **Smooth pursuit** (VIDEO) – have the patient slowly track a target and note saccadic (where saccades substitute for subnormal smooth pursuit gain to catch-up to the target) or “choppy” pursuit (VIDEO). Impaired pursuit horizontally and vertically is typically seen in cerebellar disease (or its connections). If impairment of pursuit is asymmetric, think about an ipsilesional process – e.g., saccadic or choppy pursuit to the right due to a right hemispheric lesion (VIDEO).
- **Vestibulo-Ocular Reflex Suppression (VORS)** (VIDEO) – the VOR will need to periodically be suppressed or cancelled in certain situations – e.g., sitting on a bus and reading a newspaper while the bus turns. The VOR is stimulated by the turning of the bus, but the VOR is suppressed and the eyes remain stable so the reader can continue to foveate the words on the page. VORS will generally be saccadic when pursuit is saccadic and vice versa (VIDEO). However, when pursuit is impaired and the VOR is lost (bilateral vestibular loss), VORS can look better than pursuit since there is no VOR to suppress (VIDEO).
- **Optokinetic nystagmus** (VIDEO) – at the bedside, using an optokinetic stimulus can assist in the evaluation of smooth pursuit and saccades. The slow phases represent smooth pursuit while the fast phases represent saccades. Since the bedside optokinetic stimulus used (optokinetic tape/flag, examiner’s fingertips, or any alternating patterns/lines, optokinetic drum) does not involve full visual field stimulation like looking out the window at passing scenery from a moving train, the examiner is not really isolating the optokinetic system in this way.

2. Involuntary Eye Movements

- **Saccadic intrusions** – saccades are the culprit
 - **With an intersaccadic interval** (VIDEO)
 - Square wave jerks are most common, mainly seen with basal ganglia and/or cerebellar pathology
 - **Without an intersaccadic interval** (VIDEO)
 - Ocular flutter (horizontal plane) and opsoclonus (horizontal, vertical and torsional planes)
- **Nystagmus** – slow phases are the culprit
 - **Pendular** – back-to-back slow phases, giving a pendular appearance. Most commonly seen in multiple sclerosis (VIDEO) or with oculopalatal tremor (VIDEO).
 - **Jerk** – alternating slow and fast phases, where the slow drift is the pathological phase, although nystagmus is named for the direction the fast phase. Can be further localized by the slow phase velocity waveform (see figure below). Vestibular nystagmus tends to have a linear appearance; gaze-evoked nystagmus (due to impaired neural integrators) tends to have a velocity decreasing waveform; infantile nystagmus (and occasionally an conditions causing an unstable neural integrator) tends to have a velocity increasing waveform.
 - **Vestibular**
 - Spontaneous vestibular nystagmus implies imbalance of semicircular canal (SCC) afferents, either peripherally or centrally.

- Fixation suppresses peripheral nystagmus, but occasionally central nystagmus too. Visual fixation can be removed by occlusive ophthalmoscopy, pen-light cover test, or using Frenzel lenses. Note that the actual direction of any horizontal or vertical spontaneous nystagmus is opposite of that seen with the ophthalmoscope (you are viewing posterior to the axis of rotation for horizontal and vertical movements), but is the same for torsional movements.
- Peripheral nystagmus should be unidirectional, follow Alexander's law (intensity of the nystagmus increases in the direction of the fast phase) and acutely, has a mixed horizontal-torsional appearance ([VIDEO](#)). Central vestibular nystagmus can be indistinguishable from peripheral ([VIDEO](#)).
- **Gaze-holding**
 - Have the patient maintain eccentric fixation in each of the other cardinal positions of gaze. If the eyes are unable to maintain eccentric fixation and instead drift back towards center (slow phase) and then quickly move back toward the intended direction of gaze, then this is referred to as gaze-evoked nystagmus (GEN). When GEN is present horizontally and vertically, this generally implies a disorder in the vestibulocerebellum (flocculus/paraflocculus, or its connections [VIDEO](#)). If only vertical GEN is observed, an INC (or medial longitudinal fasciculus or adjacent paramedian tract lesion(s) as these structures play a role in relaying vertical gaze position signals) should be suspected. If only horizontal GEN is observed, damage to the NPH-MVN complex should be suspected. Patients with GEN commonly have rebound nystagmus – e.g., the patient with GEN will have left-beating nystagmus (LBN) in left gaze and right-beating nystagmus (RBN) in right gaze, and when the patient is asked to look to the right (where there is RBN) and then back to primary gaze, the appearance of LBN will suggest rebound nystagmus. Again, this generally indicates flocculus/paraflocculus (or its connections) pathology.
 - Commonly, patients without posterior fossa pathology have a small amplitude, fatigable, physiologic end point nystagmus (EPN) in far lateral gaze, although this should abate when bringing the fixation target back to so that it can be viewed by both eyes, or at about ¾ lateral position. Rebound nystagmus should be absent.

3. Vestibulo-Ocular Reflex (VOR)

VESTIBULO-OCULAR REFLEX (VOR) – THE VOR ALLOWS FOR RETINAL STABILITY DURING HEAD MOVEMENTS. EX) A PERSON WITH A NORMAL VOR CAN WALK DOWN THE STREET AND CLEARLY READ A SIGN IN FRONT OF THEM SINCE THE EYES ADJUST FOR EACH HEAD MOVEMENT AND THE FOVEA REMAIN ON THE TARGET. A PERSON WITH BILATERAL VESTIBULAR LOSS WILL WALK DOWN THE STREET AND THE SIGN WILL JUMP UP AND DOWN BECAUSE THE HEAD AND EYES MOVE TOGETHER – I.E., THE FOVEA CANNOT BE HELD ON THE STREET DUE TO IMPAIRED VOR, SO THE IMAGE APPEARS TO JUMP UP AND DOWN (SO-CALLED *WALKING OSCILLOPSIA* [HTTPS://COLLECTIONS.LIB.UTAH.EDU/DETAILS?ID=1213442](https://collections.lib.utah.edu/details?id=1213442)). THE FOLLOWING ARE WAYS TO EVALUATE VOR FUNCTION AT THE BEDSIDE.

- **Dynamic Visual Acuity** ([VIDEO](#)): Passive rotation of head (horizontally to evaluate the horizontal SCC and vertically to evaluate the anterior and posterior SCC function) at 2 Hz while viewing a distance (preferred) or near eye chart. A decrease in best-corrected vision of 2 lines or more from baseline is considered abnormal – patients with unilateral vestibular loss may lose 2-3 lines prior to compensation, while patients with bilateral vestibular loss will lose 4 or more lines.
- **Visually enhanced VOR (vVOR)**: Passive rotation through entire horizontal or vertical ocular motor range at 0.5 Hz while fixating on the examiner's nose. This combines smooth pursuit and VOR. If pursuit is impaired and the VOR is hypoactive (e.g., cerebellopathy and bilateral vestibular loss due to cerebellar ataxia, neuropathy, vestibular areflexia syndrome, CANVAS [VIDEO](#)), the vVOR will be impaired and will look choppy or saccadic. If either system is functional, this will be smooth.
- **Head impulse test (HIT)** ([VIDEO](#)): With the patient fixating on the examiner's nose, perform a brief, rapid head rotation of 15-20°. In the case of an acute right peripheral vestibulopathy due to vestibular neuritis, a rightward HIT will result in the eyes moving to the right with the head initially, so that a corrective re-fixation saccade will be needed to move the eyes back to the target, or to the left. This is considered an abnormal or positive HIT and generally suggests a peripheral process (although there are exceptions).
- **Vibration** ([VIDEO](#)): Vibration of the mastoids and vertex will induce an ipsilesional slow phase with unilateral vestibular loss, more so acutely than chronically.
- **Head-shaking** ([VIDEO](#)): Sustained, rapid, back and forth, horizontal head shaking for ~15 secs may produce a spontaneous nystagmus that slowly dies out. With peripheral lesions, the slow phase is toward the affected ear. With central

lesions, the slow phase may be vertical or the nystagmus may change direction from the baseline spontaneous nystagmus. If there's strong HSN without clear unilateral vestibular loss ([VIDEO](#)), think about a central process.

4. Other Audiovestibular Tests and Special Situations

- **Pressure-induced** ([VIDEO](#)): Valsalva against closed glottis, pinched-nose Valsalva, pressure in the external auditory canal causing nystagmus (Hennebert's Sign) mainly in SCDS ([VIDEO](#)), may see Valsalva-induced symptoms with cervicomedullary lesions such as a Chiari; or **Sound-induced nystagmus** (Tullio Phenomenon) mainly in SCDS.
- **Hyperventilation** ([VIDEO](#)): Alkalosis and changes in iCa from 30-60 sec hyperventilation may improve conduction through an affected segment of 8th cranial nerve due to vestibular schwannoma ([VIDEO](#)) or neurovascular compression, usually causing excitatory nystagmus with a contralesional-directed slow phase. When a chronic vestibular imbalance has been compensated for by central mechanisms, hyperventilation can cause a transient decompensation and bring out nystagmus with an ipsilesional slow phase. Hyperventilation can enhance/produce downbeat nystagmus in cerebellar disease.
- **Dix-Hallpike Maneuver**: Used to test for posterior canal (PC) BPPV.
 - Example: when *right* posterior BPPV is suspected, turn the head 45° to the *right*, and rapidly move en bloc straight back so that the head is slightly hyperextended (~20 degrees) while hanging over the edge of the examination table with the head still turned 45° to the *right*. This maximally stimulates the right PC SCC. In *right* PC-BPPV, the *right* Dix-Hallpike will provoke upbeat-torsional nystagmus towards the *right* (lowermost) ear, which is due to otoconial debris falling through the canal (causing endolymph movement and cupular deflection in an excitatory direction). The nystagmus **1**) typically begins with a short latency (sometimes as long as 30 secs) after change in head position, **2**) lasts less than 1 min, **3**) fatigues with repeated testing, and **4**) often reverses direction (downbeat-torsional towards the left ear with right PC-BPPV) when the patient sits up again.

CHARACTERISTICS OF POSITIONAL NYSTAGMUS

Site	Peripheral	Central
Direction	<ul style="list-style-type: none"> • Posterior Canal – mixed upbeat-torsional with (torsional with top poles beating toward the dependent ear VIDEO); reverses on sitting up (VIDEO); treated with Epley or Semont (VIDEO) • Horizontal Canal – either beats toward (geotropic VIDEO) or away from (apogeotropic VIDEO) the ground. Tested with supine roll testing, and when the side is identified to which nystagmus is more intense (right or left ear down), nystagmus will beat toward the affected ear – e.g., more RBN with right ear down, diagnosis is right geotropic HC-BPPV; more RBN with left ear down, diagnosis is right apogeotropic HC-BPPV: treated with BBQ roll (VIDEO) or Gufoni (VIDEO) among others • Anterior Canal – given its parasagittal orientation, nystagmus can be pure downbeat (VIDEO), but more often downbeat-torsional (top poles towards affected ear), and can be brought out with right or left Hallpike, or straight head-hanging – central mimics must be excluded; treated with deep head hanging (VIDEO) among others 	<p>Can be pure vertical (down VIDEO >up), pure torsional, or can have vertical and torsional components</p> <p>Can be horizontal, more commonly apogeotropic as compared to geotropic</p> <p>Central positional nystagmus is usually associated with other abnormal ocular motor findings</p>
Latency	Typically after a few seconds but may be longer	May or may not have a latency
Duration	<1 minute with canalithiasis, can be longer with cupulolithiasis (otoconia stuck to cupula)	Can be <1 minute or sustained
Fatigability	Yes	Generally does not fatigue, but it may
Vegetative Symptoms	Yes	Usually less pronounced

- **Auditory Testing**:
 - **Bedside** – Rinne and Weber ([VIDEO](#))
 - The Rinne test is an assessment of auditory thresholds to air and bone conduction of sound. The Weber test is a comparison of bone conducted sound of either ear. Conductive hearing loss results in a loss of air conducted greater than bone conducted sound, whereas sensorineural hearing loss results in the loss of both air and bone conducted sound. Peripheral vestibular disease affecting the labyrinth or the 8th cranial nerve can be associated with sensorineural hearing loss. In these cases, the sensitivity to air conduction will remain greater than to bone conduction. Weber will lateralize away from the side of sensorineural hearing loss. As an example, destruction of the right labyrinth (e.g., bacterial labyrinthitis) will cause decreased hearing in the right ear, and air conduction will be greater than bone conduction in the right (affected) and left (unaffected) ears. Weber will lateralize to the left (unaffected) ear. In the case of superior semicircular canal dehiscence (SCDS), there may be increased sensitivity to bony transmission of sound through a (third mobile window)

as well as conductive hearing loss, with bone conduction greater than air conduction and Weber lateralizing to the side of the dehiscence.

- **Audiometry** ([ADDITIONAL READING ON AUDIOMETRY](#))

5. ‘HINTS’ testing in the acute vestibular syndrome

	Peripheral Pattern	Central Pattern	Comments
Head Impulse Test (HIT)	Abnormal (VIDEO)	Normal more often than abnormal (VIDEO)	-Can have normal HIT with the rare inferior division vestibular neuritis, which spares the horizontal canal -Can have abnormal HIT with lesions involving the root entry zone of CN8; vestibular nucleus; labyrinthine ischemia among other ‘dangerous’ etiologies (VIDEO)
Nystagmus (spontaneous)	Mixed horizontal-torsional; unidirectional; follows Alexander’s law; suppresses with fixation (VIDEO)	Can be pure horizontal, horizontal-torsional, pure torsional (VIDEO), vertical or torsional-vertical (e.g., medullary, MLF VIDEO); often changes direction with gaze (gaze-evoked VIDEO) but can be unidirectional and follow Alexander’s law; may or may not suppress with fixation	-‘Central’ and ‘peripheral’ spontaneous nystagmus in the acute vestibular syndrome can be indistinguishable
Test of Skew	Normal (VIDEO)	Normal or Abnormal (VIDEO)	-A skew deviation on a peripheral basis (labyrinth or CN8) is possible, but rare, and should be small and short-lived -Presence of a skew should be considered central until proven otherwise -Rarely, a congenital (unrelated) 4 th nerve palsy can lead to a false positive ‘test of skew’