

# PHYSICAL EXAMINATION

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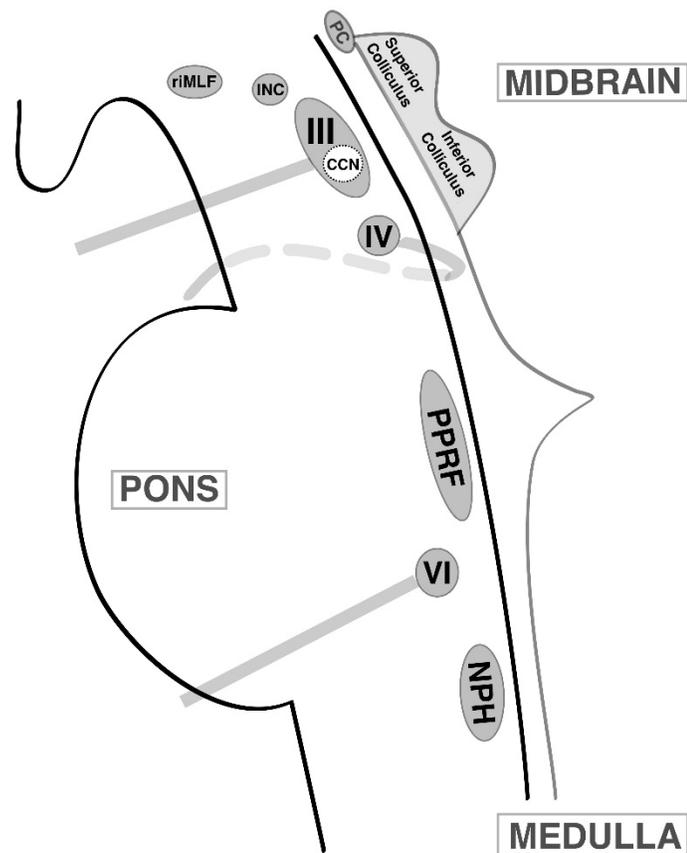
## 4. Other Audiovestibular Tests and Special Situations

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- Audiometry

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# 1. Ocular Motor Examination

- Important brainstem anatomy (see Figure)
  - **Midbrain:** vertical/torsional burst neurons for saccades (rostral interstitial medial longitudinal fasciculus [riMLF]); vertical/torsional gaze-holding machinery (interstitial nucleus of Cajal [INC]); nuclei of IIIrd (including central caudal nucleus [CCN]) and IVth nerves; posterior commissure (PC) which plays a role in upward gaze.



- **Pons:** horizontal burst neurons for saccades (paramedian pontine reticular formation [PPRF]); nucleus of VIth nerve.
- **Medulla:** horizontal gaze-holding machinery (nucleus prepositus hypoglossi [NPH] & medial vestibular nucleus [MVN]).

### Complete ocular motor examination should consist of:

- **Range of movements** (VIDEO) – in 9 cardinal positions of gaze with both eyes viewing (versions). Check the range of each individual eye (ductions) if there is diplopia or if a motility deficit is suspected.
- **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). Any change in deviation or lack thereof helps in the localization. See examples below.
  - **3<sup>rd</sup> (left)** – due to left medial rectus paresis, exotropia in right gaze. Due to left superior rectus paresis, right hypertropia in up gaze. Due to left inferior rectus paresis, left hypertropia in down gaze.
  - **6<sup>th</sup> (left)** – an esotropia worse in left gaze suggests that there is an abduction deficit on the left; in the example of a left 6<sup>th</sup> nerve palsy, there is less left lateral rectus tone with normal medial rectus tone thereby resulting in crossing of the eyes (esotropia), and this will be maximal in the direction of the paresis (to the left).
  - **4<sup>th</sup> (left)** – due to left superior oblique (SO) paresis and given the actions of this muscle – primary – incycloduction; secondary – depression; tertiary – abduction – there will be left excycloduction, hypertropia, and a V-pattern esotropia with bilateral 4ths (the tertiary action of abduction is generally not so clinically relevant with unilateral palsies, but when bilateral, an esotropia greatest in down gaze is seen), respectively. A left 4<sup>th</sup> nerve palsy is diagnosed using the 3-step test where the examiner identifies that there is a 1) left hypertropia that 2) increases in (contralateral) right gaze (the oblique muscles have greatest vertical action in adduction), and 3) increases in (ipsilateral) left head tilt. Because the SO is also a depressor, the left hypertropia will tend to increase in down gaze (at least with new-onset acquired palsies), and torsional measurements (e.g., double Maddox rod, bucket test, measuring the angle between the fovea and optic nerve) will demonstrate (ipsilateral) left excycloduction.
  - **Skew deviation** (VIDEO) – a vertical, non-paralytic misalignment of the eyes from utricle-ocular motor pathway imbalance, which should be assumed to be central until proven otherwise. The vertical deviation (and vertical diplopia) is generally the same or similar in different directions of gaze.
    - **Low/Low** – the hypotropic (**lower**) eye will be ipsilesional if the injury occurs caudal to (**lower than**) the decussation of the utricle-ocular motor pathway. Accompanied by an *ipsiversive* ocular tilt reaction (OTR) – e.g., left Wallenberg causes left head tilt, ocular counterroll with top poles toward left ear

- Examples: labyrinth and 8<sup>th</sup> cranial nerve (both are rare causes of a skew), vestibular nucleus (common cause)
    - **High/High** – the hypertropic (**higher**) eye will be ipsilesional if the injury occurs rostral to (**higher than**) the decussation of the utricle-ocular motor pathway. Accompanied by a *contraversive* OTR – e.g., left MLF lesion causes right head tilt, ocular counterroll with top poles toward right ear
      - Examples: MLF, INC
  - **4<sup>th</sup> vs Skew**
    - **Head tilt** – patients with a 4<sup>th</sup> nerve palsy have a compensatory head tilt to the opposite side – e.g., a left 4<sup>th</sup> nerve palsy will cause a compensatory rightward head tilt. The reason is that by tilting the head to the right, the patient excites the right utricle which initiates the utricle-ocular motor reflex, resulting in minute elevation of the right eye and depression of the left eye and very slight ocular counterroll with top poles toward the left ear. This will help to minimize retinal image disparity resulting from the paretic left SO and improves diplopia. A patient with a left MLF lesion will have a contraversive OTR with right head tilt – in this case, the head tilt is pathologic, or an attempt to correct for an abnormal internal representation of where Earth vertical is located. In other words, with a left MLF lesion (where the utricle pathways that are injured originated in the right labyrinth, and the utricle pathways that originated in the left labyrinth are intact and are relatively hyperactive), the brain thinks that the head/body is tilting to the left (when in fact it is not) and attempts to compensate and bring the head to Earth vertical by tilting the head to the right.
    - **Cycloduction** – a patient with a left 4<sup>th</sup> will have left hypertropia and left excycloduction with no abnormal cycloduction in the right eye. A patient with a left MLF will have a left hypertropia and left incycloduction in addition to right excycloduction – this results from the ocular counterroll portion of the OTR.
- **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).
  - Slow saccades of normal amplitude occur in brainstem disease, typically involving burst neurons in the PPRF for horizontal saccades (e.g., SCA 1, 2, 3, 7 among others) or riMILF for vertical saccades (e.g., progressive supranuclear palsy, PSP VIDEO).
  - Slow saccades of restricted amplitude occur in motor nerve paresis or muscle weakness.
  - Slow adducting saccades are seen with an INO (lesion involving the MLF, commonly an INO is seen with an OTR/skew acutely – e.g., left MLF lesion with a left INO and left hypertropia, contraversive OTR), even when no adduction deficit is present (although at least an exodeviation in contralateral gaze is almost always noted). This is accompanied by abducting nystagmus. INO may be due to MS, stroke, or structural and metabolic injuries (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.
  - Situations in which bedside OKN can be helpful
    - Rapid assessment of symmetry and presence/absence of pursuit/saccades in an uncooperative or difficult to examine patient
    - It can help to bring out a subtle adduction lag in INO
    - One of the first ocular motor signs of PSP is loss of the downward fast phase to an optokinetic stimulus directed upward (goes along with downward saccades being slightly slower than upward saccades initially, and downgaze being more affected than upgaze)
    - If nystagmus is seen in a patient with functional monocular (when the good eye is occluded) or binocular blindness, this suggests that the patient has at least some vision
    - Since upward saccades are often affected in dorsal midbrain (Parinaud’s syndrome), vertical OKN can demonstrate this and convergence retraction nystagmus (when stimulus is directed downward).

## 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.

<b>NYSTAGMUS</b> (slow phase is culprit)			
<b>Pendular</b>	<b>Jerk (slow phase velocities)</b>		
	<b>Linear (constant)</b>	<b>Decreasing</b>	<b>Increasing</b>
			
<b>INTRUSIONS/OSCILLATIONS</b> (saccade is culprit)			
<b>Square Wave Jerks</b>	<b>Macrosaccadic Oscillations</b>	<b>Opsoclonus &amp; Flutter</b>	
			

○ **Jerk nystagmus**

▪ **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  $\frac{3}{4}$  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>). 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 8<sup>th</sup> 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.
- **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](#))
- **HINTS** (see table below)

### CHARACTERISTICS OF POSITIONAL NYSTAGMUS

Site	Peripheral	Central
Direction	<ul style="list-style-type: none"> <li>• Posterior Canal – mixed upbeat-torsional with (torsional with top poles beating toward the dependent ear <a href="#">VIDEO</a>); reverses on sitting up (<a href="#">VIDEO</a>); treated with Epley or Semont (<a href="#">VIDEO</a>)</li> <li>• Horizontal Canal – either beats toward (geotropic <a href="#">VIDEO</a>) or away from (apogeotropic <a href="#">VIDEO</a>) 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 (<a href="#">VIDEO</a>) or Gufoni (<a href="#">VIDEO</a>) among others</li> <li>• Anterior Canal – given its parasagittal orientation, nystagmus can be pure downbeat (<a href="#">VIDEO</a>), but more often downbeat-torsional (top poles towards affected ear), and can be</li> </ul>	<p>Can be pure vertical (down <a href="#">VIDEO</a> &gt;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</p>

	brought out with right or left Hallpike, or straight head-hanging – central mimics must be excluded; treated with deep head hanging ( <a href="#">VIDEO</a> ) among others	abnormal ocular motor findings
Latency	<ul style="list-style-type: none"> <li>Typically after a few seconds but may be longer</li> </ul>	May or may not have a latency
Duration	<ul style="list-style-type: none"> <li>&lt;1 minute with canalithiasis, can be longer with cupulolithiasis (otoconia stuck to cupula)</li> </ul>	Can be <1 minute or sustained
Fatigability	<ul style="list-style-type: none"> <li>Yes</li> </ul>	Generally does not fatigue, but it may
Vegetative Symptoms	Yes	Usually less pronounced

## 5. ‘HINTS’ testing in the acute vestibular syndrome

	Peripheral Pattern	Central Pattern	Comments
Head Impulse Test (HIT)	Abnormal ( <a href="#">VIDEO</a> )	Normal more often than abnormal ( <a href="#">VIDEO</a> )	-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 ( <a href="#">VIDEO</a> )
Nystagmus (spontaneous)	Mixed horizontal-torsional; unidirectional; follows Alexander’s law; suppresses with fixation ( <a href="#">VIDEO</a> )	Can be pure horizontal, horizontal-torsional, pure torsional ( <a href="#">VIDEO</a> ), vertical or torsional-vertical (e.g., medullary, MLF <a href="#">VIDEO</a> ); often changes direction with gaze (gaze-evoked <a href="#">VIDEO</a> ) 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 ( <a href="#">VIDEO</a> )	Normal or Abnormal ( <a href="#">VIDEO</a> )	-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 <sup>th</sup> nerve palsy can lead to a false positive ‘test of skew’