

# Vision Science III

## Assessment of the Extraocular Muscles

Reading Chapter 10 in Borish's

When evaluating for saccades, pursuits and vestibular movements, ability to: fixate and change fixation (saccade), follow a target (pursuit), fixate a target while head position is changed (vestibular) gives information on the ocular motility system.

### Definitions:

**Concomitancy:** (concomitant) angle of deviation is the same in all positions of gaze.

**Nonconcomitancy:** (incomitant) angle of deviation does change in different fields of gaze.

**Paralysis/Paralytic/palsy:** nerve damage is complete and no innervation flows to the affected eye muscle.

**Paresis/paretic:** disruption of innervation is partial, not total. This occurs most often and can be of any degree from mild to severe.

### Testing Methods:

One of the most important tools in assessing ocular motility is observation: during history, as the patient walks and looks around the room...look for signs of strabismus, head position in tilts, tips or turns. Head position help compensate for muscle dysfunction. Anatomical structure can fool you into thinking you have a strabismus. Check bridge width on children, observe for facial asymmetry.

Clues to onset: Recent diplopia, noncomitancy and abnormal head posture help to separate acquired problems from congenital. Is a new problem more likely to have diplopia?, Which would be more noncomitant? When does head position change?

### Testing of Versions and Ductions:

- **Broad H test:** This test is done binocularly. If the deviation measured is concomitant when the strabismus is nonparetic. If the deviation measured is incomitant suspect one or more paretic EOMS. If you test with both eyes, you can have the patient inform you of diplopia and if a penlight is used for the testing the corneal reflex can also give you clues to the alignment. If over or under actions noted, recheck monocularly. The monocular test identifies which eye has the paretic muscle. If binocular test is normal, monocular testing is not done. See fig. 10-9

### **Further testing that helps identify incomitancy:**

- **Maddox Rod:** can be used to evaluate phorias and also comitancy in various fields of gaze. Prism can be used to align the vertical red line and the light to measure the phoria. Be aware that glasses may induce prism that may not be present normally.
- **Red Lens test:** subjective test of deviations in nine positions of gaze. With a red lens over one eye, ask for separation between white and red light in the nine positions of gaze.
- **Parks 3 step test:** Used with vertical deviations to isolate the paretic muscle.

Step 1....in primary gaze which eye is hypertropic.

Step 2....in which gaze, right or left does the hypertropia increase

Step 3....when the head is tilted left or right which has the greatest hypertropia

Use lecture handouts and table 10-3

### **Neuromuscular muscle anomalies....infranuclear**

Lead to eye fatigue and discomfort, compensating head position can occur. Diplopia would be present early on, but with time improve as the visual system compensates. See overhead.

**6<sup>th</sup> nerve palsy:** this nerve is easily damaged due to the length of the nerve and its course. Palsy results in inability to abduct the eye, esotropia in primary gaze. To compensate the face is turned towards the affected side to decrease diplopia. This is the most prevalent acquired noncomitant deviation and rarely congenital. In young patients consider trauma. In older patients, 6<sup>th</sup> nerve palsies are from ischemic causes, related to hypertension or diabetes. Under the age of 40 consider MS. Other considerations: nystagmus blocking, thyroid myopathy, myasthenia gravis, orbital inflammatory pseudotumor, orbital blowout trauma.

### **Syndromes:**

Duane's retraction syndrome: globe is retracted and fissure narrowed when adduction is attempted, unable to abduct the eye. Left eye most often effected. 15-20% bilateral, females have a greater incidence, 4:1 ratio.

**Etiology of Duane's:** Tradition explanation is that lateral rectus is fibrotic. Alternate theory is miswiring of innervation to the lateral rectus. Innervation goes to both MR and LR by the 3<sup>rd</sup> nerve causing contract of both muscles resulting in retraction of the globe, partial limitation of adduction and narrowing of the palpebral fissure.

## **Types of Duanes:**

### **Duane's Type I**

Very limited abduction in the affected eye  
Lids may widen on abduction  
Slightly restricted adduction  
Upshoot or downshoot on adduction  
Retraction of the globe and narrowing of the palpebral fissure on adduction  
Binocular in primary gaze usually, but may be esotropic  
Observe for head turn in direction of affected eye., limited binocularity

### **Duane's type II**

Very limited adduction  
Relatively normal abduction  
Retraction on abduction with lid narrowing  
AKA Inverse Duane

### **Duane's type III**

Limited or absent adduction and abduction  
Retraction of globe and narrowed fissures on adduction  
Rarest of the 3

**Rule out other causes:** MR palsy, LR palsy, thyroid myopathy, spasm of near reflex, esotropia

**Treatment...** find cause and RO other causes, give RX, monitor, VT, Prism, patching. Most patients learn to move their head. With strabismus the suppression helps prevent the diplopia. Consider sx if cosmesis is a factor.

**Mobius Syndrome;** LR palsy that is bilateral. Also have lack of facial musculature, decreased bulk of one side of the tongue, mental retardation, congenital heart defects, limb and chest deformities, hearing, speech and swallowing problems.

### **Fourth nerve palsy:**

The thinness, position of the IV makes it vulnerable to injury. Traumatic closed head injury from a frontal blow is a very common cause. Even minor head injury can cause nerve damage. Most common cause of vertical deviation (90%) Paresis of the superior oblique results in hypertropia that increases in inferonasal gaze. Torsional diplopia in other positions that increases temporally. Parks test will show this.

If the lesion is in the nucleus, the defect is contralateral, if the lesion is prior to decussation, the defect is contralateral, and if the lesion is at the site of decussation the result is bilateral paresis. If post decussation, ipsilateral paresis. The third nerve may often be involved resulting in a down and out eye that can not be moved into the correct field of gaze to assess the 4<sup>th</sup> nerve. Watch for torsion of the eye to see if the 4<sup>th</sup> nerve is intact.

### **Position of eye and head;**

The affected eye is hypertropic in primary gaze. There may excyclotorsion and small eso in primary. No depression when adducted. The chin is depressed allowing for up gaze. Head is tilted away from the affected side towards the opposite shoulder. Falling eye sign, as the yoke muscle in the other eye is the inferior rectus, when fixating in the affected eyes field of gaze, the other eye may drop due to increased innervation to the inferior rectus.

### **Causes of IV palsy:**

Trauma...

60% congenital in children/ 35% traumatic

Vascular infarct from DM or HTN

Idiopathic

MS

Tumor

Aneurysm.

21% of IV nerve palsies are bilateral and caused by head trauma...where? The patient sees tilting and diplopia. To see bilateral involvement do an alternate cover test in right and left gaze.

### **Differential diagnosis:**

Myasthenia gravis...fatigue in up gaze

Thyroid myopathy

Orbital inflammatory pseudotumor

Treatment: Rx with prism, Vt, spot patching, monitor, find cause

**3<sup>rd</sup> Nerve palsy:** This is the thickest nerve and lesions may not interfere with the entire function of the nerve. Compressive and traumatic lesions of less impact may disrupt a portion of the nerve. Pupil function may be spared. With the nerve splitting location of the lesion may affect which functions are lost. Total block prior to division leads to

ipsilateral paralysis of the MR, IR, IO, SR, Levator, Pupillary sphincter, and Ciliary muscle. The eye is dilated, unable to focus, has ptosis, and divergent strabismus. The eye is down and out. Incomplete paresis results in combinations of these signs and symptoms. Superior branch lesions will take out the superior rectus and levator, inferior branch lesions will take out the inferior rectus, medial rectus and motor root to the ciliary ganglion. Lesions in the oculomotor complex can affect some nuclei and save others. Many variations are possible.

### **Etiology of III nerve can be shown by:**

**Pupil sparing:** Often vascular and deeper parts of nerve affected resulting from possibly DM and Cavernous sinus syndrome

**Pupil involved:** indicates a more compressive lesion such as aneurysm, tumor, trauma, rarely herpes zoster. When the pupil is blown and fixed there is internal ophthalmoplegia which is the pupil and total IIN.

**Appearance of patient;** Eye will be down and out with only temporally movement and slight inferior movement as only the 6<sup>th</sup> and 4<sup>th</sup> nerve are operating. The lid may be closed, the pupil blown, and diplopia present.

Differential diagnosis:

Myasthenia Gravis  
Thyroid myopathy  
Chronic progressive external ophthalmoplegia  
Orbital pseudo tumor  
Midbrain lesion  
Severe hypertension

Treatment:

Find the cause, immediate CT and or MRI if

1. Pupil involved
2. Pupil spared but the patient is less than 50, no history of DM no improvement after 2 to 3 months, other nerves affected

Give RX, Fresnel prisms, possible spot patching, vision therapy for calisthenics and sensorimotor fusion training.

### **SR palsy:**

Hypotropia and ptosis of involved eye why?

Hypotropia increases in abduction with head tilted to involved side on 3 step test. Why does it increase with abduction?

Patient cannot elevate eye during abduction

May have extorsion of eye if acquired as an adult.

Chin is usually elevated to keep eyes in down gaze.

Isolated SR is very rare, usually congenital

### **Rule out**

Mechanical causes for limitation:

**Thyroid myopathy**, proptosis, inflamed EOM's, lid retraction, With thyroid problems the IR is most often affected and then the  $MR < SR < LR$ . Obliques are rarely affected by thyroid disorders.

Trauma such as a blow out fracture may trap the IR and led to poor elevation

Ocular myasthenia

Inhibitional palsy of contralateral antagonist incases of superior oblique palsy.

TX: find cause, give RX, VT, prism if needed spot patching

### **MR palsy:**

Exotropia of involved eye, cannot adduct the eye, face turns away from the affected side to place the affected eye in temporal gaze.

Isolated MR extremely rare

May occur from blowout fracture:

### **DDx:**

INO(Internuclear Ophthalmoplegia) where the affected eye cannot adduct to hook horizontally. INO's caused by lesions in the MLF, a unilateral INO suggests an infarct in branch of the Basilar artery, bilateral INO(BINO) is a red flag to MS.

Duane's syndrome, type II

Thyroid myopathy

Ocular Myasthenia

**Treatment:** find cause, give Rx, VT, prisms, spot patching

### **IR palsy;**

Hypertropia of involved eye

Hyper increases in abduction and with head tilt to opposite side

Intorsion if acquired as adult

Cannot depress the eye during abduction

Chin is depressed slightly  
Most frequent muscle to become fibrotic

**DDX**

Trauma  
Thyroid myopathy  
Ocular myasthenia

**Treatment**

Find cause, give RX, VT, prisms, spot patching, and monitor

**IO palsy:**

Hypotropia of involved eye with some intorsion.  
Cannot elevate eye during adduction  
Chin is elevated, with head tilt towards involved side, face towards noninvolved side  
Hypotropia increases in adduction and on head tilt to unaffected side on park's test  
A pattern exo in down gaze  
IO least likely muscle to be affected, usually congenital.

**Syndrome:**

Brown's Syndrome;  
Eye will not elevate when adducted  
Patient may have chin elevation  
Minimal vertical deviation in primary gaze, poor park's 3 step test  
Normal to slight limitation of elevation in primary gaze and abduction  
Patient is often binocular in primary gaze,  
Monocular but can be binocular: 10%  
Congenital, sometimes trauma  
Widening of the lids on adduction  
Divergence in up gaze, V pattern exo  
Caused by mechanical anomaly of the SO, the tendon sheath is short and fixed at the pulley and will become a barrier as the eye tries to adduct. The globe will slip under the stretched sheath and you will hear a click as the patient tries to elevate during adduction..

**DDX** of Brown's Syndrome: IO paresis, forced duction test will R/O this.

Treatment: find cause, give RX, monitor, VT, prism, spot patching.

Surgery not recommended unless there is a significant vertical strabismus. Many folks with Brown's have normal BV.

**Remember:** lesions in the cortex and supranuclear connections can cause conjugate dysfunction in saccades, pursuits, or vestibular movements. Lesions in the midbrain that interfere with the gaze centers and internuclear connections can result in the inability to produce conjugate eye movements. It is only at the motor nuclei level that damage will be present in one eye or the other.

**Disconjugate eye movements (Vergence):** function to maintain bifoveal fixation of targets at various distances.

**Evaluation:**

**Angle Lambda (or Kappa)**...the angle between the pupillary axis and the line of sight. Normally this measures +5 degrees, the plus noting that line of sight is nasal to the pupillary axis, - noting that the line of sight is temporal. The average position of the corneal reflex will be 0.4mm nasal to the center. A difference between the two angle Lambdas suggests eccentric fixation.

**Hirschberg test:** With both eyes unoccluded and the patient fixating a penlight at 50cm, compare the positions of the corneal reflexes in both eyes under binocular conditions. Then compare these measurements with the position of the reflex under monocular conditions. This test reveals the presence or absence of a strabismus and is very useful in children, infants and those with poor cooperation. 1 mm of deviation indicates 22prism diopters of strabismic deviation. Compare the reflex difference starting from the position of the fixating eye. Do not start from the center of the pupil, so; If the reflex is 0.5mm nasal on the right eye and 1mm temporal on the left eye, how much of a difference is between the two. The total difference is 1.5mm leading to 30prism diopters of exotropia. These reflexes can be recorded with photos to help in assessment.

**Krimsky reflex prism test:** similar to the Hirshberg, this test uses prism before the fixating eye to equalize the reflexes.

**Bruckner test;** with the ophthalmoscope, sit 50cm to 1m from the patient having them look directly at the light. Focus on the anterior surface and watch both eyes. Judge the color and symmetry of the red fundus reflex. The whiter, brighter eye is the one that is turning.

**Unilateral and alternate cover tests**

The unilateral portion reveals the presence of strabismus or phoria. Size of tropia is measured on the alternate cover test using prism to neutralize movement or estimating 7diopters per mm of movement.