Neuro-eye for Primary Eyecare: From Clinical Concepts to What's New in Ocular Management

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Abstract:
Using a combination of clinical patient cases and imaging studies this course presents the latest advancements in diagnostic testing and management options in patients with neuro-eye conditions. The presentation will be in a clinical rounds format.

Course Objectives:
1. To review the various clinical entities associated neuro-eye conditions that may be encountered in a primary eyecare setting.
2. Understand how to clinically differentiate the different types of conditions, and based upon clinical findings determine the underlying etiology and.
3. Determine what constitutes a medical emergency and the proper referral channels.
4. How to manage neuro-eye patients during the initial workup including in-office imaging technology, when referring for advanced testing, and when returning the primary eyecare setting.

CASE STUDY 1
Clinical Presentation:

What’s your DDx:

Tests to Order:

Management:

What’s New/Pearls
CASE STUDY 2
Clinical Presentation:

What's your DDx:

Tests to Order:

Management:

What's New/Pearls

CASE STUDY 3
Clinical Presentation:

What's your DDx:

Tests to Order:

Management:

What's New/Pearls
Neurogenic Diplopia

Is it true diplopia?
A. What questions do you ask (most cases diagnosed during the history)?

B. R/O non-neurogenic etiologies of diplopia

C. Measuring diplopia
   - Prisms
   - Evaluation in all gazes
   - Park’s three, head tilt test

D. Evaluation of diplopia
   1. Is the deviation incomitant or comitant
      a. If the deviation incomitant: Forced duction (+) \rightarrow restrictive ophthalmopathy (TED, trauma, myositis)
                     Forced duction (-) & tensilon (+) \rightarrow myasthenia gravis,
                     Forced duction (-) & tensilon (-) \rightarrow cranial nerve palsies 3\textsuperscript{rd}, 4\textsuperscript{th}, 6\textsuperscript{th},
                     internuclear ophthalmoplegia, supranuclear disorders
                     Ductions (both eyes) are better than versions \rightarrow neurogenic
      b. If the deviation comitant: decompensated phorias, accommodative esotropia, vergence paresis

   2. If the deviation is incomitant then what to do next?
      a. Forced duction test
         (+) Think restrictive disease: Thyroid ophthalmopathy (can be neg. in early stage), orbital
         myositis/orbital pseudotumor (IOIS), orbital mass lesions, trauma, orbital fracture,
         entrapment, Browns tendon sheath syndrome
         (-) Think myasthenia, CN palsies, INO/BINO
      b. Exophthalmometry \rightarrow proptosis present?
         Rule out contralateral endophthalmos (blow-out fracture, etc.)
         Proptosis possible causes: Orbital mass, Inflammatory, Enlargement of muscles. Extraorbital
         process (sinus, CNS, bone disease)
      c. Resistance on retropulsion?
         - Orbital mass
         - TED (Thyroid Eye Disease)
      d. Orbital pain?
         - Orbit: Inflammatory pseudotumor (IOIS), sinusitis, DM - mucormycosis, metastatic tumor
         - Superior orbital fissure, anterior cav. sinus, Tolosa-Hunt Syndrome, carotid
         - fistula, cavernous sinus thrombosis, herpes zoster, metastatic, lymphoma
      e. Check lids: Is ptosis present or retraction?
         - Does ptosis alternate (pathognomonic for myasthenia)
         - Lid retraction: (pathognomonic for thyroid)
      f. Pupil abnormalities
         - Dilated pupil: third nerve palsy
         - Migraine
      g. Visual loss (optic nerve compromise)
         - Optic nerve tumors, compressive, inflammatory (may need STAT imaging/referral)
         - Combination of exophthalmos, optic atrophy, optociliary shunt vessels, and
         - decreased acuity \rightarrow pathognomonic of optic nerve sheath meningioma
         - Thyroid optic neuropathy
      h. Chemosis, injection (indicates inflammation or venous stasis)
         - Thyroid orbitopathy
         - Orbital pseudotumor (i.e. IOIS)
         - Cavernous-sinus fistula
      i. Pulsations
         - Sphenoidal dysplasia, vascular lesions, carotid-cavernous fistulas
      j. Fusional amplitudes \rightarrow high amplitudes indicates long standing
      k. Tensilon test \rightarrow if indicated
E. **Common diplopia disorders**: Web site for more general info: [http://www.thamburaj.com/ocular_palsies.htm](http://www.thamburaj.com/ocular_palsies.htm)

**Cranial nerve palsies**

**Third nerve palsy**


A. Anatomy

B. Localization:

* Nuclear lesions: Rare, caused by focal ischemia, metastatic, inflammation Must have contralateral SR paresis and bilateral or absent ptosis

  * **Fascicular: (most cases go to neurology first)**

    Nothnagel's syndrome Ipsilateral 3rd nerve paresis/cerebellar ataxia

    Benedikt's syndrome Ipsilateral third nerve palsy/hemi tremor

    Weber's syndrome Ipsilateral third nerve palsy/contralateral hemiparesis

    Claude's syndrome Ipsilateral third nerve palsy/contralateral ataxia

* Subarachnoid space:

  Uncal herniation: supratentorial tumor causes downward displacement & herniation with CN3 compression: look for pupil involvement

  PCom aneurysm: Pupil affected first or within days → STAT ER/imaging (CT Angiography/spiral CT/MRI)

* Cavernous sinus

  Look for other signs: Combination of CN3, 4, 5, 6, Horner's

  Partial or complete

  R/O tumors, inflammation, aneurysms

* Orbit

  Proptosis, chemosis

  R/O tumors, infections, inflammation, trauma

  Other cranial nerves: CN 2, 3, 4, 5, 6

* Aberrant Regeneration (Primary vs. Secondary Aberrant Regeneration)

  Pseudo-von Graefe's sign: Lid retraction on downgaze (IR → levator)

  Reverse Duane's syndrome:lid retraction on adduction (MR → levator)

  Pupillary changes w motility or levator function (MR/IR/levator → pupil)

  ** NEVER OCCURS IN ISCHEMIC MICROVASCULAR CN3 PALSY**

  Occurs secondary to aneurysm, tumor, trauma MUST IMAGE in no h/o trauma

  * Ophthalmoplegic migraine

    occurs in children, residual findings as adult including possible pupil involvement

C. Etiology

**ADULT:**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Undetermined</td>
<td>24%</td>
</tr>
<tr>
<td>Microvascular</td>
<td>20%</td>
</tr>
<tr>
<td>Trauma</td>
<td>15%</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>15% (up to 21%)</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>12%</td>
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<tr>
<td>Other</td>
<td>13%</td>
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**CHILD:**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Congenital</td>
<td>47%</td>
</tr>
<tr>
<td>Trauma</td>
<td>21%</td>
</tr>
<tr>
<td>Inflammation</td>
<td>11%</td>
</tr>
<tr>
<td>Other</td>
<td>11%</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>11%</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>3%</td>
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</table>
D. Management (nontraumatic) with age consideration:

<table>
<thead>
<tr>
<th>Pearl: if no improvement in 90 days for microvas.</th>
<th>MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Under 50 without pupil involved: MRI, med eval., possible CTA/MRA</td>
<td></td>
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<tr>
<td>* Under 50 with pupil involvement: MRI/MRA/CTA/angio, if neg, thorough med eval (primary to r/o ischemic vascular)</td>
<td></td>
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<tr>
<td>* Over 50 without pupil involvement: Observe pupil, MRI, ESR/CRP (if no h/o DM, HTN do med eval) <strong>VIEW ONH \rightarrow DO NOT DILATE!</strong></td>
<td></td>
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<tr>
<td>* Over 55 with polymyalgia rheumatica or sx of GCA: ESR, CRP, CBC, possible TA bx</td>
<td></td>
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<tr>
<td>* Aberrant regeneration: Image (MRI/MRA) if no old h/o trauma</td>
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**Fourth nerve palsy**


A. Anatomy

B. Localization

  - Nuclear/fascicular: Cannot separate clinically, frequently seen with contralateral Horner’s
  - Subarachnoid space: Trauma is frequent, anterior medullary tumors are cause of bilateral CN4 palsies
  - cavernous sinus: Look for CN 3, 4, 5, 6 & Horner’s

C. Congenital Paresis

  - Common! \rightarrow decompensate and manifests in adulthood

D. Clinical Signs

  - Presents with vertical and/or torsional diplopia with head tilt toward the opposite shoulder to improve fusion
  - Acquired: low vertical fusional amplitudes (may develop larger amplitudes weeks or months after the insult)
  - Longstanding: large vertical fusional amplitudes \rightarrow test with prism bar

E. Clinical diagnosis - Parks three step

  **The 3 Cardinal Questions:**

  1. Which eye is higher in primary gaze?
  2. Does the hyper deviation get worse in R or L gaze?
  3. Does the hyper deviation get worse on R or L head tilt?

![Right SO palsy](image)

F. Etiology

  **ADULT:** 40-30-20-10 rule

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<tr>
<td>Head trauma</td>
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<tr>
<td>Undetermined</td>
<td>30-36% (30)</td>
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<tr>
<td>Microvascular</td>
<td>17-20% (20)</td>
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<tr>
<td>Other (MS)</td>
<td>15%</td>
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<tr>
<td>Neoplasm</td>
<td>4-10% (10)</td>
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**CHILDREN:**

most are Congenital or Trauma
H. Management isolated 4th Nerve Palsy:

- All ages with large fusional amplitudes: Watch; decompensated congenital 4th
- Nonvasculopathic age (<40yo), without large fusional amplitudes, if motility disorder not classic or + fatigue of EOMs → Tension, if normal or h/o trauma → MRI
- Vasculopathic age (>40): Evaluate for HTN, DM, image not necessary unless below items noted
- Progression or addition neurological sx: MRI

**Six nerve palsy** (called **ABduction Deficit** until entrapment or MG r/o'ed)


A. Anatomy

B. Localization

* **Nucleus**: Gaze palsy to ipsilateral side
* **Fasciculus**:
  - *Foville's syndrome*: Ipsilat 6th + gaze palsy (PPRF), 7th facial palsy, 5th palsy, 8th nerve damage
  - *Millard-Gubler Syndrome*: Ipsilat 6th palsy/7th palsy, and contralat hemiplegia
* **Subarachnoid space**:
  - Basilar skull tumors or trauma
  - Increased Intracranial pressure: papilledema, vomiting
* **Petrous portion**
  - *Gradenigo’s syndrome*: Ipsilateral 6th palsy/ear pain/trigeminal pain
* **Cavernous sinus**: CN 3, 4, 6 with possible Horner's

C. Etiology:                      ADULT

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<tbody>
<tr>
<td>1. Undetermined</td>
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<tr>
<td>2. Neoplasm</td>
<td>22%</td>
</tr>
<tr>
<td>3. Vascular</td>
<td>12-17%</td>
</tr>
<tr>
<td>4. Other (MS)</td>
<td>16-22%</td>
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<tr>
<td>5. Head trauma</td>
<td>14%</td>
</tr>
<tr>
<td>6. Aneurysm</td>
<td>3.6%</td>
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<table>
<thead>
<tr>
<th></th>
<th>CHILDREN</th>
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</thead>
<tbody>
<tr>
<td>1. Trauma</td>
<td>32%</td>
</tr>
<tr>
<td>2. Neoplasm</td>
<td>30%</td>
</tr>
<tr>
<td>3. Inflammatory</td>
<td>12%</td>
</tr>
<tr>
<td>4. Vascular</td>
<td>&lt;5%</td>
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D. Management (non-trauma) isolated 6th nerve palsy

- **Age < 14**: If p-viral, watch 2 wks then at 4 wks if other neurological sx or no imp. → MRI
- **Age 15-40**: MRI, if neg. Include HTN/DM, collagen dz, syphilis, Lyme, consider LP
- **Age over 40**: Usually vascular: blood glucose/HTN, if no imp by 3 mos → MRI
- **Over 60**: Vascular, ESR/CRP to rule out GCA if suspected

**ALWAYS IMAGE 6th if H/O CANCER, PAIN, NEURO-SI's!**
Myasthenia Gravis

MG MUST always be in your DDx

In general: found in • young females or older males (but note minor peaks below)
  • Is a disorder of neuromuscular transmission recognized clinically by varying muscle weakness which is characterized by worsening with fatigue.
  • ACh receptors are being destroyed; thus less receptor sites at the neuro-muscle junct
  • Neuro-ophthalmic significance

50% present w/ Ocular MG
10-30% of all MG remain ocular
90% of all MG have ocular involvement (usually diplopia is CC)
It is RARE to generalize (develop systemic MG) after 2 yrs. of isolated ocular involvement

Neuro-Ophthalmic Findings – (all should be noted in videos)

Ptosis
  Lid fatigue: Does ptosis becomes exaggerated after the pt is asked to repeatedly blink or after they look in upgaze for a couple of minutes

Herring’s Law: Look for the Curtain Si • hold upper lid up against the frontal bone, does the other (contralateral) lid drop down?

Cogan’s lid twitch Have pt look down for 20-30 sec, then have them look in ¼ gaze… the upper lids will over shoot & then come back to the normal position.
Sometimes this can occur spontaneously in primary gaze.

Peak sign – orbicularis weakness
  Ask pt to gently close eyes, look for one lid opening up slightly looking for asymmetry between the 2 lids.

Ophthalmoparesis
  Any pattern is possible (can mimic CN3,4,6, etc.)
  Mimics a BINO (“pseudo-BINO”)

How to DDx? • In MG, can induce an OKN nystagmus in the adduction deficit eye. • In True BINO, you will NOT be able to induce an OKN nystagmus in the Adduction deficit eye.

Look for diplopia worse in the PM
Look for lid involvement

Lightening eye movements
  Saccadic-like movement to the eyes (but of limited range)

NORMAL pupils
  Normal light & near rxns, normal pupils sizes (RARE, RARE, RARE …cases with pupil abnorm.)

**VARIABILITY IS THE HALLMARK**

Diagnosis
1. Ice Pack Test
   Procedure: Induce a ptosis & measure it --then apply ice pack on lids for a few mins --remove ice-pack & immediately (w/n 10 secs) measure ptosis. If ptosis improves probably MG.
   Mechanism: cold slows down acetylcholinesterase, allowing more [AcH] w/n the neuromuscle junction EOM improvement controversial
   Can do in-office, but can’t make Dx of MG solely on this test.

2. Tensilon Test (Gold Standard)
   A relatively simple test; however, it should always be done in a hospital setting (i.e. not in your private office!) b/c it may cause respiratory distress or cardiac failure in some pt's. Must have IV Atropine on stand by if you do this test.
Best to send pt to a **Neuro-ophthalmologist** for this test if referring out

**There must be a clear endpoint for accurate interpretation of the Tensilon test:**
In other words, **careful** measurements of the inter-palpebral aperture & EOM motility must be done **PRIOR** to administering the test. After the Tensilon is given does the ptosis & EOM paresis get better? If it does, then it is MG.

**Procedure:** Inject 0.2 cc, does pt get better? If not, inject another 0.2cc, does pt get better? If not, inject the remainder 0.6cc.

**Note:** Tensilon test will often **NOT** eliminate the pt's diplopia, just improves it.

3. **Acetylcholine Receptor (AChR) Antibodies** - (not used as a routine test with all MG's)
   - **When to order?**
     - Used on questionable pt’s when you can’t make Dx from tensilon test, and as gMG risk
   - **Results are (+) in:**
     - up to 90% of Generalized MG (gMG)
     - 40-50% of Ocular MG
   - **TYPES:**
     - Binding Antibody
     - Blocking Antibody
     - Modulating Antibody

**Striated Muscle Antibody**
- >80% of thymomatous MG, some nonthymomatous MG patients
- can occur in the absence of AChR antibody in patients with MG
- rare in ocular MG
- presence in early onset disease → ≥95% likelihood of an underlying thymoma

**Anti-MuSK Antibodies**
- MuSK – Muscle-Specific receptor tyrosine Kinase on the muscle side of neuromuscular junction
- mediates clustering of AChRs during synapse formation
- positive in up to 70% AChR antibody “seronegative” MG patients
- neck, shoulder, respiratory muscle weakness, less or delayed ocular involvement

4. **When to image muscles to r/o TED?**
   - Used on pt’s that have a partial (poor) rxn w/ the Tensilon test but are put on MG meds & they are not responding to medical therapy. So, order CT/MRI/Ultrasound to r/o TED.
   - There is approx. 10% association between TED & Ocular MG. (remember both are auto-immune dz’s)

5. **EMG (Electromyography)**
   - **RNS** – Repetitive nerve stimulation
   - **SFEMG** – Single-fiber electromyography

6. **Eye movement recordings**
**MG Treatment**

Not all MG are treated w/ drugs – 20% resolve spontaneously.

**Systemic Tx:**

In MOST cases that receive Tx, the prognosis **systemically** is very good.

Some pts can have a **Myasthenia Crisis** → pt can die of respiratory failure 2¼ respiratory muscle involvement.

Even though Myasthenia Crisis is a RARE occurrence, you must ask ALL MG pt's “Are you having any problems with your breathing?” If they say yes, then you must refer out immediately.

**Mestinon** (pyridostigmine bromide)

Anticholinesterase medication

Most common drug Rx’ed for **systemic** aspect of MG → or diabetic MG

~30% of pt’s **do not** respond to Tx.

**Dosing:** 60 mg BID-TID up to 120 mg QID (max amount)

**Prostigmin** (neostigmine chloride)

Anticholinesterase medication

15 mg tab – up to QID

**Corticosteroids**

Number **ONE** medication for treatment of **ocular** aspect of MG, reduces oMG generalizing to gMG

Given by itself or concurrently w/ Mestinon/Prostigmin

**Slow initial dosing** or risk of myasthenic crisis

In long-term therapy, watch for 2¼ Glc & PSC

**Immunosuppressives**

Use when pt not responding to Corticosteroids

Azathioprine, cyclosporine, mycophenolate, cyclophosphamide

In long-term therapy, watch for 2¼ infections & carcinomas.

**Plasmapheresis**

Short-term for impending crisis, or surg. Tx that make MG worse

**Thymectomy**

Removal of thymus gland w/ or w/o a thyoma – always done with thyoma, considered in young MG patients even without a thyoma present.

**Ocular Tx:**

Even w/ systemic Tx, pt’s do poor w diplopia

Prism & EOM surgery do NOT work well here b/c diplopia is **variable.** (usually diplopia worse the 1 yr., but tends to improve w/time.)

Often have to **patch diplopia pts.**

For the ptosis can use a “**Ptosis Crutch**” – a wire that is placed on the back of glasses that holds the lid up but still allows pt to blink.

**Stem Cell Therapy** – where are we in the clinical trials?

**Orbital**

**Etiologies of Orbital Disease**

Grave’s Disease 47%  Neoplasms 22%  Inflammations 10% (“orbital pseudotumor”)  Vascular 7%  Trauma 5%

**PROPTOSIS & DIPLOPIA**

Axial masses with slow progression • no diplopia until late Non-axial masses • diplopia with gaze toward mass

Inflammatory pseudotumor • diplopia Grave’s disease • diplopia
PROPTOSIS

- average  
  upper normal range

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<tr>
<td>White female</td>
<td>15.4mm</td>
<td>20.1mm</td>
</tr>
<tr>
<td>White male</td>
<td>16.5mm</td>
<td>21.7mm</td>
</tr>
<tr>
<td>Black female</td>
<td>17.9mm</td>
<td>23.0mm</td>
</tr>
<tr>
<td>Black male</td>
<td>18.5mm</td>
<td>24.7mm</td>
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No normal with more than 2mm of asymmetry between the OD and OS!
(681 normal adults, Hertel exophthalmometer)

GLOBE PULSATION

Biomicroscope, Goldmann, Cotton-tipped applicators to view

Etiology

- Congenital sphenoidal dysplasia (Neurofibromatosis)
- Transmission ICP via roof defect (Trauma/Surgical)
- AV fistulas
- Congenital AV malformations

Thyroid Eye Disease (TED)

Grave’s disease restrictive myopathy is the most common cause of spontaneous diplopia in the adult patient ...

Female:Male, 3:1 Fourth/Fifth decades Autoimmune, can be unrelated thyroid status IgG orbital/extraorbital antigen
Increased serum IgE Increased edema and orbital volume Lymphocytic/plasmacytic infiltration EOM fibrosis

Correlation not 100% between ophthalmopathy & thyroid hormone status! Grave’s Ophthalmopathy:

- Pt can be → Hyper/Hypo/Euthyroid
- Hyperthyroid >50% pts Hyperthyroid at Dx (Grave’s Disease)
- Euthyroid (most of remaining pts)
  - many go on to develop hyperthyroidism existing subtle thyroid dysfunction
- Hypothyroid (MUST ASK ABOUT PRIOR TX!)
  - most had radio-I tx for hyperthyroid rare cases of primary hypothyroid

Exposure Keratitis

Dry eye very common complaint: Eyelid dysfunction, Proptosis, Loss of Bell’s phenomenon with restrictive myopathy
Untreated can lead to ulceration and perforation

EOM Involvement

- Commonest cause of DV in middle age
- Present in up to 80% of patients
- IR>MR>LR>SR (can vary!)
- Mimics CN4 or 6, double elevator palsy
- Forced duction + (may be neg. early in disease)
- Rare cases of muscle paresis

Optic Neuropathy

-5% Grave’s patients
- Most do not have marked proptosis or optic nerve head changes
- Insidious onset
- Worse/more progressive in smokers
- Decreased VA, color vision, APD(<50% pts)
- Visual field defects:
  - central scotomas
  - arcuate/altitudinal
  - paracentral scotomas
  - generalized constriction
**Laboratory Testing** *(Don’t need for ocular dx…do to r/o systemic involvement)*

- TSH - single best test
  - **Hyperthyroidism** → low TSH
  - **Hypothyroidism** → high TSH
- T4 - most is bound to proteins
- T3 - more active hormone
- RT3 – consider for hypothyroid patients

When to order antibody studies?

**Imaging Studies**

- **CT imaging**, study of choice in most places
  - EOM enlargement sparing tendinous insertions
  - Orbital fat increased some patients
  - Occasional lacrimal gland enlargement
  - Optic nerve compression at apex

- **Ultrasonography** — no ionizing radiation — measurement of enlarged EOM’s
- **MRI** — when indicated, can this pick up early changes missed by CT?

**EOM Treatment**

- Prisms
- Surgery after stable for 6 months
  - Recessions only
  - Adjustable sutures
  - Late over correction common

Prednisone if congestion/early

Radiation tx: if congestion

**Optic Neuropathy Treatment**

**Vision loss can be severe to (NLP)**

- Prednisone 100 mg po/dy immediately
  - If improved surgery may not be need

- Posterior orbital decompression
  - Must be done prior to muscle surg.

- Radiation tx
  - If not at fibrotic stage and without sig. visual loss

**Use of OCT in Neuro-eye Disease**

OCT compared to other forms of RNFL assessment

OCT can provide:

1. Diagnostic information/ long term findings usually show thinning in most clinical cases
   - DDx of functional visual loss, optic neuropathy vs. maculopathy, true disc swelling vs. pseudopapilledema

   - Optic neuritis w MS, RNFL thinning and/or macula thinning:

<table>
<thead>
<tr>
<th>RRMS</th>
<th>SPMS</th>
<th>PPMS</th>
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<td>Pulicken et al. [37]</td>
<td>94.4 (14.6)</td>
<td>81.8 (15.6)</td>
<td>88.9 (13.3)</td>
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</table>
• NAION
• LHON
• Retrogeniculate lesions
• Alzheimer’s disease
• Parkinson’s disease

2. Disease monitoring for progression or resolution
3. Prognosis

Clinical cases
1. MS with primary finding of RNFL thinning
2. MS with primary finding of macula thickness thinning
3. Case of questionable optic nerve swelling in early IIH
4. Case of RNFL loss with a parachiasmal mass

Pearl: If OCT findings unremarkable or questionable, consider ordering a multifocal ERG (mfERG) along with a pattern VEP (pVEP) to rule-out an occult maculopathy or optic nerve dysfunction.

Excellent Website for additional neuro-eye information:  http://novel.utah.edu/