C18

The Five Common Neuro-ophthalmic Symptoms You Should Not Disregard

12 June 2017

08:00 - 09:30hrs

Room 118/119

HAND-OUTS
Intermittent Diplopia

Patients whose symptoms occur intermittently often pose a particular diagnostic challenge. We will start by looking at the various mechanisms that can produce such intermittent visual symptoms and then, based on this list, will offer a diagnostic approach to the problem.

MECHANISM

A. Misalignment present only in a particular field of gaze

Some patients with incomitant misalignment are aware that their diplopia occurs only on gaze in a certain direction but others are not, reporting instead that their double vision comes and goes. Examples of this mechanism include the following:

1. Internuclear ophthalmoplegia (INO)
   Most patients with an INO are orthophoric in primary position, even in the face of a marked adduction deficit. The key to the diagnosis of an INO is the characteristic slowing of medial rectus saccades, the most sensitive sign of the condition. Abduction nystagmus, while characteristic, is actually non-specific.

2. Restrictive orbitopathy
   Examples include Graves disease and orbital fracture. Similar to INO, such eyes are often aligned in primary, the misalignment only becoming manifest on upgaze (for a floor fracture) or on abduction (for medial wall entrapment). In contrast to INO, saccades in the direction of gaze that is limited are of normal velocity.

3. Mild 6th nerve palsy (NP)
   In cases of partial 6th NP, the misalignment may be sufficiently small in positions of gaze away from the action of the muscle that it can be kept in check by fusion. Diplopia is thus present only on lateral gaze or on far distance viewing.

4. Vergence disorders
   Convergence or divergence insufficiency may be similarly present just in certain fields of gaze. Divergence insufficiency is characterized by horizontal diplopia just at distance, most typically when driving and especially at night. Convergence insufficiency produces horizontal diplopia at near, often accompanied by blur due to associated accommodative insufficiency.

B. Underlying heterophoria that intermittently escapes fusion

Having some degree of latent misalignment (heterophoria) is quite common in normal individuals but usually asymptomatic because it is kept in check by fusion. Fusion however can be impaired for a number of reasons, thus producing diplopia. It is important to be familiar with these precipitating factors.
1. Fusional amplitudes may be reduced with normal aging, often exacerbated by intercurrent illness.
2. Loss of fusion is sometimes brought on by a change in optical status such as first bifocals, or after refractive surgery.
3. Fusional capacity is particularly sensitive to the effects of medications that have central nervous system depressant action. Medications in this category include: sedative hypnotics, anti-convulsants, opiates and muscle relaxants.
4. Certain neurologic degenerative diseases, particularly Parkinsonian syndromes, cause early and prominent loss of fusional capacity. One form, Progressive Supranuclear Palsy (PSP), also causes early loss of vergence (both divergence and convergence), adding to difficulty with fusion.
5. In addition to the above ocular motor factors, it is important to realize that afferent visual loss can also diminish fusion. The basis for fusion is the brain receiving a slightly different version of the same object in each eye. The temporal fields are especially important in this regard. The optical challenge for someone with a dense bi-temporal hemianopia is to line up the two nasal hemi-fields to provide a coherent and complete image. Failure to do so produces intermittent diplopia, which can be horizontal or vertical, depending on the direction of an underlying phoria.

C. Truly intermittent disorders

After the above mechanisms are ruled out, we then move on to those ocular motor disorders that are genuinely episodic. In most cases the clinical features enable diagnosis; sometimes there are clues from the examination, even when the patients is between episodes. In some cases additional ancillary testing is needed.

1. Myasthenia  
   The hallmark of myasthenia is actually fatigability rather than weakness. Improvement upon awakening is more helpful than worsening later in the day.

2. Vertebro-basilar transient ischemic attacks (TIA’s)  
   Most V-B TIA’s have other focal deficits besides diplopia.

3. Spasm of the near reflex (convergence spasm)  
   Look for associated miosis and myopia.

4. Increased intracranial pressure (ICP)  
   When increased ICP causes diplopia, it is usually due to 6th nerve palsy, which is often intermittent. Occasional cases are due to 4th nerve weakness.

5. EOM ischemia  
   There are not many conditions that cause ischemia of the eye muscles. In older individuals, think about giant cell arteritis. In those with vascular risk factors, consider ocular ischemic syndrome. EOM ischemia is usually accompanied by pain, which should help distinguish it from myasthenia.
6. Irritable EOM disorders
These are disorders in which the eye muscle, or the nerve innervating it, becomes intermittently overactive rather than weak. The most common of these disorders is superior oblique myokymia (SOM) and ocular neuromyotonia (ONM). SOM is characterized by brief episodes of monocular oscillopsia and vertical diplopia, lasting just a few seconds but often recurring multiple times per day. Episodes are sometimes precipitated by looking in the field of action of the superior oblique muscle.

ONM occurs most often after radiation for skull base tumor. The diplopia in this disorder is due to delayed relaxation of the involved eye muscle. For example, if the left medial rectus is affected, the eyes may be orthophoric in primary position initially but, following conjugate right gaze then returning to primary, continued contraction of the MR produces esotropia. This situation thus mimics a left 6th NP.

7. Periodic or paroxysmal skew deviation
This rare disorder is associated with other midbrain signs.

8. Cyclic esotropia
This rare condition is more properly termed congenital 3rd nerve palsy with cyclic spasm. This condition has also been reported in acquired 3rd NP.

B. DIAGNOSTIC APPROACH

1. Establish that the diplopia is in fact binocular
Monocular diplopia is due to aberrations in the ocular media, never neurologic. When due to refractive error or lenticular change, diplopia is usually constant; when intermittent, more likely due to keratopathy. Accommodative insufficiency may cause intermittent monocular diplopia at near. If the patient is certain about this question, it is sometimes appropriate to give them a ‘homework assignment” – to check with each eye covered the next time they have a spell.

2. Is the diplopia gaze-evoked?
Convergence insufficiency/paresis produces horizontal diplopia at near. Divergence paresis or 6th nerve paresis causes horizontal diplopia at distance. Fourth nerve palsy causes vertical diplopia on down gaze and with head tilt. Ocular neuromyotonia and superior oblique myotonia are often precipitated by activation of the involved EOM (e.g. gaze down-in for SOM).

3. Is there an underlying phoria that is intermittently decompensating?
The examination should include cross-cover testing and Maddox rod for small vertical deviations. Fusional amplitudes may be reduced by medication effect so ask about recent additions or changes in dose.

If a small phoria is present, consider measuring fusional amplitudes (using a prism bar) and then comparing the phoria with the amplitude to judge its significance. Normally we wouldn’t expect a 1 diopter vertical heterophoria to be symptomatic, but if the fusional amplitude is <1 diopter then it may be.
Assess the visual field, at least by confrontation, and consider perimetry if intermittent diplopia is still unexplained.

4. Are there signs/symptoms of increased intracranial pressure?  
Ask about headaches, pulsatile tinnitus, look for papilledema.

5. Are there brainstem symptoms?  
Most vertebro-basilar TIA’s are not monosymptomatic. Ask about other brainstem symptoms such as vertigo (most common), peri-oral numbness, dysphagia, dysarthria, and weakness of face or limbs. TIAs are sometimes precipitated by head/neck movements.

6. Are there signs or symptoms of myasthenia?  
Is the diplopia present upon awakening? Is it worse with prolonged effort?  
Look for myasthenic features on examination. These include: ptosis/lid fatigability/lid twitches, Cogan’s sign, EOM fatigability/variability/quiver movements, and orbicularis weakness or fatigability. A modified sleep test (eyes closed for about 20 minutes) is very useful, especially for ptosis: look for brief improvement immediately upon re-opening the eyes.

7. Are there signs or symptoms of giant cell arteritis?  
Intermittent diplopia is a common symptom of GCA, probably due to eye muscle ischemia rather than cranial nerve weakness. As such, it can have any pattern. In any elderly patient with diplopia, persistent or intermittent, ask about scalp tenderness, jaw pain, weight loss, fever and other systemic manifestations.

C. ANCILLARY TESTING

Additional diagnostic testing depends on results of the historical and exam findings as noted above. If there are provocative maneuvers that bring on symptoms, efforts should be made to reproduce this in the office (e.g. prolonged reading, physical exertion). In cases with a completely normal exam it may be appropriate to obtain an MRI scan, acetylcholine receptor antibodies and (in older individuals) an ESR and CRP for giant cell arteritis. It should be noted that antibody tests are not as sensitive for ocular myasthenia as for the generalized form of the disease. Negative antibody testing does not rule out the disease.

REFERENCES


Transient Visual Loss

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Transient visual loss (TVL) lasts from seconds to minutes in one eye or both. By convention, the definition includes abrupt onset. The visual loss does not clear with blinking (as would an abnormal tear film). TVL is a challenging clinical problem because the symptom is gone by the time you examine the patient, and most often there are no helpful traces. But you have to start somewhere.

Begin by dividing TVL into transient MONOCULAR visual loss (TMVL) and transient BINOCULAR visual loss (TBVL) because they have different causes and demand different investigations. But here again you run into a problem: it is often hard to determine whether the visual loss was MONOCULAR or BINOCULAR! Most patients will attribute homonymous binocular visual loss to the eye that suffered temporal visual field loss. Four historical clues tell you that the TVL was probably binocular and homonymous: 1) It interfered with visual clarity. (If the patient has intact vision in both eyes, monocular TVL does not disturb visual clarity.) 2) It occurred in a temporal field. (Monocular TVL may be nasal, superior, or inferior, but rarely temporal.) 3) It included sparkles or bright spots. (The retina and optic rarely do not often produce positive visual phenomenon when ischemic.) 4) The scotoma migrated across the visual field. (Retinal or optic nerve ischemia never produces a migrating scotoma, whereas migrainous occipital events often do.)

Let us consider TMVL first. It is always caused by a failure of blood flow (perfusion) in the affected eye. There are 7 main causes: 1) low blood pressure; 2) high blood pressure; 3) ipsilateral cervical carotid occlusive disease; 4) distal (ophthalmic, ciliary, retinal artery) occlusive disease; 5) cardioembolic or carotid embolic disease; 6) hypercoagulable states and 7) papilledema. (Low blood pressure is an underestimated cause of TMVL, especially if it is provoked by assuming the upright posture, by an increase in blood pressure-lowering medication, or by dehydration.)

To evaluate TMVL, start by asking if it is provoked, in which case systemic hypotension is a common cause. Measure systemic blood pressure! (To be fancy, measure it in lying and standing positions, looking for orthostatic hypotension if TMVL is provoked by those circumstances.) Perform ophthammoscopy to exclude papilledema, hypertensive retinopathy, and venous stasis retinopathy. Stethoscope auscultation of the neck is a quaint but insensitive exercise. You will have to rely on neck ultrasound or CT/MR/digital angiography to rule out carotid or aortic arch stenosis. You would investigate cardioembolic disease, a rare cause of TMVL, by echocardiography and heart rhythm monitoring, which has low yield unless the patient has known heart disease or reports palpitations. Vascular occlusive disease in the ophthalmic or ciliary circulations is undetectable and can only be surmised. Hypercoagulable states usually occur within known systemic disorders, which you can sometimes elicit by history, but otherwise you may have to order a complete blood count and protein electrophoresis. In elderly adults, TMVL can be a warning sign of occlusive disease in giant cell arteritis, so ask about appropriate systemic symptoms and order a sedimentation rate and C-reactive protein.
Because TMVL is a transient ischemic attack (TIA), your evaluation must occur quickly if the event has been recent. Some writers suggest that the patient be sent to an emergency room for expedited evaluation. If critical ipsilateral carotid stenosis is found, the patient will be considered for carotid endarterectomy or carotid stenting. There is little evidence to support the efficacy of that procedure for TMVL alone—but welcome to controversy!

We all know that the evaluation of TMVL is often completely negative. In that case, you can presume the cause to be occlusive vascular disease in older adults and perhaps vasospasm in younger adults. Treat older adults with low-dose aspirin. No one knows what to do with younger adults.

Transient binocular visual loss (TBVL) has 4 main causes: 1) migraine; 2) low perfusion of visual cortex (TIA); 3) visual cortex seizure, or 4) papilledema.

Migraine, the most common cause, usually signals itself with a sparkling visual disturbance ("scintillating scotoma") that migrates across the hemifield in about 20 minutes. Headache may develop afterwards. The first episode usually occurs before age 40. But there are exceptions to this classic pattern: no scintillations, no migration, no confinement to a hemifield, onset after age 40. The less classic the pattern, the more you must consider TIA or seizure.

TIA typically lasts less than 1 minute, may be homonymous or total, and usually occurs without scintillations. If there are scintillations, the patient had a stroke or a seizure. TBVL from visual cortex TIA may be accompanied by other brain symptoms of low perfusion, such as presyncope, disequilibrium, diplopia, dysphagia, limb weakness, or limb numbness, but do not except those symptoms. Visual loss often occurs alone.

Visual cortex seizures, an uncommon but important cause of TBVL, typically have sparkling—often colored—scintillations that do not migrate and last from minutes to hours. The scintillations may be formed if they involve adjacent temporal cortex. Most visual cortex seizures have isolated visual manifestations, but rarely they spread to adjacent parietal or frontal cortex to cause head and gaze deviation, jerking limb movements, and even temporary unconsciousness.

To evaluate TBVL, begin by trying to elicit the classic symptoms of migraine. If you find them, you can probably stop the evaluation there. Anything less than a classic pattern of migraine, however, demands that you do more. Ophthalmoscopy will rule out ocular causes such as papilledema (and rarely binocular low-flow states). If that is negative, undertake formal visual field testing to rule out homonymous hemianopia, which would indicate a lesion in the posterior cerebral hemisphere. (Caution: a normal visual field does not entirely exclude such a lesion, which may be just outside the visual pathway!) If you cannot blame migraine or the eyes, you must proceed with brain imaging, especially if visual fields suggest a homonymous hemianopia. MRI will detect all but the most unusual causes: status migrainosus, focal status epilepticus, Creutzfeldt-Jacob disease, Alzheimer disease, and nonketotic hyperglycemia. If MRI is negative, the patient must undergo electroencephalography.

If this evaluation of TBVL is negative, and you believe the symptoms to be organic, presume TIA and treat with aspirin.
Photopsias

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Migraine Aura

- Homonymous
- Colored or black and white
- Expands from near fixation
- 5 min ≥ aura ≥ 60 min
- Subsequent headache

Migraine Aura

- Symptoms less than 5 min
- Photopsias without form
- Highly stereotyped symptoms
- Constant laterality
- Absence of headache
  - No history of migraine
  - Older age of onset

Visual Snow

- Continuous dots in vision
- Variant of migraine aura?
  - Constant
  - Uniformed
  - Throughout the visual field
- Young patients
  - May be present lifelong
  - Avg age at onset: 23 yrs old

“Migraine” Aura

- 63 year old man
- Trouble driving at night
- Bumps into objects
- Sees flashes with eyes closed

Case

- Va 6/6 in both eyes
- Slow pupil reactions
**Cancer-Associated Retinopathy**

- Paraneoplastic syndrome
- Symptoms before cancer is found?
- Autoimmune
  - Anti-recoverin
  - Anti-enolase
- Treat the malignancy!

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**Case**

- 43 year old woman
- Past history of Lyme disease (treated)
- Several days of photopsia OS
- Missing objects in OS
- Va 6/6 OD, 6/12 OS, RAPD OS

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**AZOOR/AIBSE**

- Spectrum of disease with photoreceptor dysfunction
- Unilateral or bilateral
- Cause unknown
- ?Systemic associations
- Other white dot syndromes
- Treatment unclear
Neuro-ophthalmic symptoms you should not disregard: Vision Blur with a “Normal Examination”

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Possibilities

- Symptom is transient
- Condition or exam findings are transient
- Exam is not really normal
- Too soon for the exam to be abnormal
- Pathogen is too “small” and condition insidious
- Exam is normal but “imaging” is not
  - OCT
  - MRI
- Non Organic overlay confuses situation
- Subtle Motility problem is causing symptom
- Higher cortical function is causing symptom

Symptom is transient
Condition or Exam findings are transient

- Transient monocular blindness
- Giant cell arteritis

Exam is not really normal

- “occult” maculopathy
- You misses history or setting of amblyopia
- You did not test visual fields
- You missed cornea issue or subtle cataract
- You missed APD
- Choroidal ischemia (giant cell arteritis)

Exam is normal but “imaging” is not

- Neuro-ophthalmic exam is not complete until VFs, OCT, (sometimes FA) and MRI are done
  - OCT
  - Occult maculopathies
  - VR interface disorders
- MRI
- Tumor
- Stroke

CASE PRESENTATION

- 41 yo woman with vision loss OS > OD for 3 days. (“blindspots”) One day prior to vision loss, pt took Nyquil for flu.
  - Va sc: 20/25 OD, 7/200 OS
  - Color and Brightness: WNL OU, NO APD
  - SLE: WNL OU
  - What test?
ACUTE MACULAR NEURORETINOPATHY

15 year old boy failed school vision screen
20/100 OD, 20/80 OS
Pupils: normal
Color: normal

Case
- 58 year old man
- Routine Exam
- Abnl Screening VF
- Unaware of the defect
- 20/20 OU
- Color: nl
- Pupils: nl

Maculopathies in NO
- Should be easy based on History and Exam, OCT:
  - cystoid macula edema
  - Macula hole
  - Some V-R interface cases
  - central serous chorioretinopathy
  - Stargardt’s Disease
  - ischemic maculopathy in diabetes
OCT should be used to screen all central vision loss

V-R interface Abnormalities
Small scotoma reduced acuity
- VR attachments, cysts, holes
- OCT

Too soon for the exam to be abnormal
- Compressive lesion without pallor
- Optic neuritis
  - You missed APD
  - VFs not done or unhelpful
- Neuroimaging early after ischemia

MRI and Optic Neuritis
- 32 ON patients
  - 31 enhancement and STIR signal
  - 18 entire length of intraorbital ON
  - 19 intracranial ON

OCT
“Outer” Retinopathies”

- Photopsia
  - Women > men
  - Blind spot enlargement
  - Careful ophthalmoscopy Late ophthalmoscopic abnormalities
- OCT
  - Autofluorescence
- Acute macular neuroretinopathy
- MEWDS
- AZOOR
- Paraneoplastic

MEWDS

Cone Dystrophy

- Heterogenous group of disorders
- Day blindness, glare
- Dyschromatopsia
- Central scotoma
- B wave decrease on ERG
Case

- 58 y.o. decreased vision and flickering
- First difficulty adjusting to light
- Progressing over six weeks
- 60 pack year smoker
- Abnormal CXR
- Small cell ca of lung
- Exam 20/80 OU, dyschromatopsia, some vitreous cells

Cancer Associated Retinopathy

- photopsia and progressive vision loss
- mid peripheral field defects
- small cancer of lung
- any tumor-uterine and lymphoma
- often present before tumor
- paucity of fundus findings
- narrowed vessels and vitritis
- diagnose with ERG
- Treatment?

Cancer Associated Retinopathy

- Autoimmune process
- antibody poisons recoverin energy production in photoreceptors
- commercial test for recoverin 23kD antibody
- Small cell cancer express recoverin
- Over 20 other antigens described in CAR syndrome
- Anti-recoverin retinopathy in non cancer patients-Heckenlively-1998

Melanoma Associated Retinoapthy

- Late in disease with metastases
- Flickering, vision and VF loss, difficulty with night vision
- Many maintain good acuity
- Male predominance
- ERG similar to CSNB
- Reduced b wave (bipolar cell)
- normal dark adapted a wave
Paraneoplastic Optic Neuropathy

- Painless, subacute bilateral vision loss
- Disc swelling
- Must rule out DIRECT affect of cancer
- Lymphocyte vasculitis and demyelination
- Cranial n. palsies, cerebellar signs, nystagmus, polyneuropathy

CRMP-5

- Specific form of PON
- Subacute cerebellar syndrome
- 62 kD neuronal antigen - Collapsin Response Mediating Protein-5
- Neuronal cytoplasmic protein
- Yu et al Ann Neurol 1998
- As common as anti Yo < anti Hu
- 7% of 116 pts had optic neuropathy
- Small cell ca lung, thymoma

CRMP 5

- Cross et al, Ann Neurol 2003
- 172 patients, 15 with optic neuritis, 5 had retinitis
- All smokers, 10 small cell ca lung
- Subacute vision loss and VF defects
- Vitreous cells and abnormal ERG
- Numerous other neurologic symptoms
  - Subacute cerebellar ataxia, dementia, cranial n., movement disorders, peripheral neuropathy
Pathogen is too “small” and/or condition insidious

- Toxic optic neuropathies
- Nutritional optic neuropathies
- LHON
- Prion Disease

Plaenil Screening: AAO guidelines 2009

- Dosing according to ideal body weight
- SD-OCT
- Fundus Autofluorescence
- Psychophysical
  - Amsler grid (Threshold, Red)
  - HVF-10-2 (white or Red target)
  - Ishihara plates
  - Multifocal ERG

Toxic Retinopathies

- Digoxin
- yellow vision
- canthaxanthine
- (hydroxy)chloroquine
- Bull’s eye maculopathy
- niacin
- CME
- tamoxifen
- crystals deposit in

Hydroxychloroquine (2628 mg, 18 yrs)

- Plaquenil Screening: AAO guidelines 2009
- Toxic Retinopathies
  - Digoxin
  - yellow vision
  - canthaxanthine
  - (hydroxy)chloroquine
  - Bull’s eye maculopathy
  - niacin
  - CME
  - tamoxifen
  - crystals deposit in

Clinical Indications for mERG

- Focal visual field defects
  - AIBSE
  - MEWDS
  - Previous RAO
- Unexplained visual loss
- Functional vision loss
- Optic nerve vs. Retina
- Hydroxychloroquine toxicity--Early detection
- Hereditary Maculopathy and retinal degenerations

Hydroxychloroquine OCT

- “flying saucer” appearance, with loss of our segments sparing fovea
- Progression to RPE atrophy centrally and enlargement of zone FAF involvement
- Re-elaboration of IS/OS

Clinical Indications for mERG
Non Organic overlay confuses situation

- Functional features can confuse and frustrate the examiner
- Can make VFs impossible to interpret
- Are most commonly seen in patients with organic disease

Organic vs. Functional Vision Loss

- Most common setting for functional is superimposed on organic
- Inconsistent fields are not always functional
- Cover your self with consults and testing
- All unexplained vision loss should be imaged

Non Organic Vision Loss

- Most important in the differential diagnosis of unexplained vision loss
- It is all about the story
- No rush to “secure diagnosis”
- Examination
  - Almost never central scotoma
  - Almost always non organic fields
  - “Reverse” acuity testing
  - stereopsis

Flash and Pattern VEP

- Halliday reported (Lancet 1972) delayed response in optic neuritis
  - pVEP 15 deg diameter, misses local defects
  - Dominated by lower field responses
- Clinical Indications
  - Functional vision loss
  - Demyelinating disease
  - Default for many non experts
Subtle Motility problem is causing symptom

- If subtle misalignment, symptom will improve with monocular occlusion
- Occasional nystagmoid movement with fixation instability

Higher cortical dysfunction is causing symptom

- JC disease could have normal MRI
- Visual variant of Alzheimer's Disease

Summary

- Blur with normal exam
- Careful history sets stage
- Repeat steps if necessary (APD, fields)
- Consider transient and intermittent conditions
- OCT necessary
- MRI recommended
- When in doubt
  - Order another test
  - Bring the patient back
  - Get a second opinion
  - Treat empirically for worst case if necessary

• Blur with normal exam
Course Handout Approval Form

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