On the Road Again
Driving with Low Vision

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Rosenberg School of Optometry
Overview

- Driving Requirements
- Tests to perform on driving patients
- Current and future visual accommodations for driving
- Identifying Low Vision driving patients in your practice
Driving Requirements

- Determined by STATE laws, not federally required
- International Counsel of Ophthalmology, published *Vision Requirements for Driving Safety* recommend unrestricted driving be;
  - visual acuity of 20/40
  - horizontal field of 120°
  - vertical field of 40°
- Assistive Technology is allowed in a state by state manner
  - Texas ALLOWS bioptics
Driving Requirements

- Assistive technology by states
  - States that do NOT allow bioptics
    - Alabama, Arizona, Connecticut, Florida, Iowa, Louisiana, Minnesota, New Mexico, North Carolina, Oklahoma, West Virginia
  - Can patients drive with bioptics in these states?
    - As long as requirements of license are being met, it is legal to drive with a biopic or other accommodative device
Texas Requirements

Unrestricted
Better eye better than 20/40

Unrestricted
Better eye is 20/50

Restricted
Better eye is 20/60 or 20/70

Unrestricted
Monocularly 20/25
Texas Requirements

- Visual Field Requirement
  - Not Required
  - 140° horizontally
  - No vertical recommendations

[Diagram showing visual field]

https://eyeternus.wordpress.com/tag/visual-field/
Texas Requirements as an Optometrist

- Do not have a duty to report
- Duty to inform
- Texas Vision Form
- Visual Acuity testing
- Field of Vision Tests
A Clinician's Approach

Does the patient meet;

1. Vision Requirements
2. Motor capabilities to handle driving the car
3. Cognition to make decisions, navigate a route, pay attention to obstacles
Optometric Testing

- Visual Acuity*
- Visual Fields*
- Contrast Sensitivity
- Visual Processing Skills

*Required
Optometric Testing - Visual Acuity

Visual Acuity

- High contrast targets
- Visual acuity has a correlation of <1% of motor vehicle accidents
- Functional acuity measures lower
Optometric Testing - Visual Acuity

- **Functional acuity**
  - Acuity that is possible during normal daily tasks
  - Functional visual acuity of normally 20/20 patient decreases to 20/40 when driving at nighttime, going >55MPH, with high beams on

[Image: https://www.consumerreports.org/car-repair-maintenance/old-headlights-can-be-dangerously-dim/]
Optometric Testing

- Visual Acuity*
- Visual Fields*
  - Contrast Sensitivity
- Visual Processing Skills

*Required
Optometric Testing

Functional Visual Field Assessment

Binocular Esterman

Goldman Perimeter

Arc Perimetry

https://www.west-op.com/perimeterpaper.html

vision2020lvrc.org.hk
Optometric Testing

- Visual Acuity*
- Visual Fields*
- Contrast Sensitivity
- Visual Processing Skills

*Required

Tests that optometrists should consider

Hold a truer correlation between good results and safer more alert driving
Optometric Testing
Contrast Sensitivity

- More predictive of patients having a difficulty time driving
  - Greater than VA
  - Greater than field loss
- Even if just one eye is affected
- Contrast sensitivity of less than 1.25 log units (moderate contrast impairment) were 8x more likely to have been involved in a car accident
Optometric Testing
Visually Processing Skills

- Impaired Visual Processing speed was strong predictor of driving performance
- Predict driving safety patients with systemic conditions
  - Stroke, Parkinson’s, and Dementia
Optometric Testing
Visual Processing Skills

- Useful Field of View (UFOV)
  - Measures “functional field of view”
  - Higher order visually possessing skills
    1. Quickly detect and localize targets
    2. Divide visual attention in both central and peripheral visual field
    3. Detect relevant targets amongst “visual clutter”
  - Stronger predictor of driving ability, safety, and crash risk than JUST visual acuity and fields

http://www.biopticdrivingusa.com/ufov-useful-field-of-vision/
Visual Accommodations

- Present
- Future
  - Self-driving cars
Bioptic Driving

- Bioptic is mounted over “good” eye
- Maximum visual acuity of 20/200
  - Maximum bioptic telescope is 4x
- Still need to maintain appropriate 140° of uninterrupted horizontal field of vision
  - Create an additional ring scotoma when being used
- Bioptic is used less than <1% of time
A Driver’s Approach to Autonomous Driving

Stage 1: Visual stimuli is registered

Stage 2: Stimuli needs to be localized and recognized

Stage 3: Driver decides action

Stage 4: Driver executes motor response to complete decision

https://www.prescouter.com/2013/01/intelligent-steering-wheel-tells-you-to-go-left-or-right/
Bioptic Training

Hand Held Telescope Skills

- Localizing
- Focusing
- Spotting
- Tracking
- Tracing

Real Life Bioptic Skills

- Nearness illusion
- Reduced Field
- Movement of objects

Passenger Skills → Driving Skills

- Receive and remember directions
- Detect and react to obstacles
- Intersections
- Glare/light reactions
Driving with a Bioptic-Perspectives

- Safety study looked at motor collision rates with bioptic drivers
- A Self assessment of the safety of bioptic drivers
  - Not overconfident
  - Maintain safe driving skill
Identifying Low Vision Drivers in your practice

New drivers with stable conditions and permanent vision loss

Experienced drivers with age related and likely progressive visual impairments and/or vision loss


Aging Population and the Implications of driving

- Loss of independence
- Non-Visual disorders that affect driving
  - Neurological issues
  - Stroke/Cerebrovascular Event
- Texas Medical Advisory board these patients need comprehensive driving tests before being allowed back on the road
  - In a survey of 290 stroke survivors
    - 30% resumed driving
    - 48% received NO ADVICE about returning to driving
    - 87% reported that NO driving evaluation was done on them before they started driving
Texas Resources

• State Agencies
  • Texas School for the Blind Visually Impaired (Austin)
  • Texas Workforce Agency

• Federal Agencies
  • Veterans Affairs Blind Rehab Centers

• Non-Profits
  • Criss Cole Center (Austin)
  • OWL radio

• Private resources
  • Hospitals with Rehabilitation
  • Private Occupational Therapists
  • Private Low Vision Clinics


Questions?

“I’m sure he’s well-trained, but seeing-eye dogs just aren’t allowed to drive.”
Non Organic Vision Loss

Doctor Bailey Peterson
Nothing to Disclose
Objectives

To learn what non-organic vision loss means to patients and providers
To categorize non-organic vision loss to a specific cause to ensure proper pt care
To learn what exam elements can be used to test for NOVL
To effectively and efficiently treat patients with NOVL
To become more comfortable with a NOVL patient
What is Non Organic Vision Loss?

Disturbance of vision not supported by an organic etiology upon examination

Commonly associated with the following:

- Stress
- Pediatric Population

Important to remember: not a diagnosis of exclusion
Malingering or Simulation

Intentionally counterfeiting a disease with the intent to benefit either monetarily or non-monetarily

Reasoning: Escape work, reduction of court sentence, collect social security, attract sympathy

Positive Simulation: Simulating an ophthalmic disease

Negative Simulation: Denial of an ophthalmic disease
Factitious Disorder

Mental disorder in which someone deceives others by appearing sick by purposely getting sick or by self injury

Mild to severe (munchausen)
Somatoform Disorders

Conversion Disorder

Neurological symptoms specifically

- Weakness/paralysis
- Abnormal Movements (tremor)
- Blindness
- Hearing Loss
- Numbness
## What Does the Patient Look Like?

<table>
<thead>
<tr>
<th></th>
<th>Malingering</th>
<th>Factitious</th>
<th>Conversion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt Background</td>
<td>Financial benefit: transferred from court, local military draft office, health insurance or other gov org</td>
<td>Extensive health knowledge, eager to have testing, many doctors, don’t talk to family</td>
<td>Anxiety, trauma, grief, depression, stress, guilt, or anger</td>
</tr>
<tr>
<td></td>
<td>Child</td>
<td></td>
<td>Presence of other neurologic complaints</td>
</tr>
<tr>
<td>Chief Complaints/symptoms</td>
<td>Vague: vision loss, field loss, decreased vision; monocular or binocular</td>
<td>Vague: vision loss, field loss, decreased vision; monocular or binocular</td>
<td>Double vision, blindness, field loss</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Highly suggestible or exaggerated</td>
<td>Get worse without apparent reason</td>
<td>Vary year to year and are rarely ever absent</td>
</tr>
<tr>
<td>Ocular health</td>
<td>Unremarkable</td>
<td>Unremarkable</td>
<td>Unremarkable</td>
</tr>
</tbody>
</table>
How to Test for Non Organic Vision Loss

Visual Acuity

Monocular vs Binocular

Visual Field Loss

Electrodiagnostics
### Table 15.3. Examination strategies for alleged blindness or loss of vision

<table>
<thead>
<tr>
<th>Bilateral blindness</th>
<th>Monocular blindness or visual impairment</th>
<th>Bilateral visual impairment</th>
</tr>
</thead>
</table>
| 1. Testing of reflexes  
  - Observation of behavior  
  - Blink reflex  
  - Prisms: fixation movements  
  - OKN  
  - Mirror-induced pursuit movements  
  - Fixation star of direct ophthalmoscope  
  - Pupillary light reactions | 1. Testing of reflexes  
  - Pupillary reactions: relative afferent pupillary defect  
  - Convergence test  
  - Refixation movement when covering the healthy eye | 1. Deception regarding the sizes of optotypes  
  - Use of constant angular sizes of optotypes at varying distances  
  - Testing with single optotypes, allowing no comparisons to other characters  
  - Near acuities with optical magnification |
| 2. Deception  
  - Tasks of coordination that require no visual function  
  - Saccades on command combined with index finger movements | 2. Binocular Tests  
  - Prisms  
  - Stereoaucity  
  - Confusion test (polarized tests, red–green glasses, etc.) | 2. Tests of central vision by other methods  
  - Central threshold sensitivity at the perimeter  
  - Preferential-looking acuity  
  - Fixation  
  - Laser interferometry  
  - Probability of seeing |
| 3. Objective testing methods  
  - VEP, ERG | 3. Testing central visual functions by other methods  
  - Central thresholds at the perimeter  
  - Preferential looking acuity  
  - Fixation  
  - Laser interferometry | 3. Objective methods  
  - Acuity VEP  
  - Multifocal ERG  
  - OKN (quantitative)  
  - Psychogalvanic reflexes |
<table>
<thead>
<tr>
<th>Visual Acuity</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Fog lens</td>
<td>Proprioception</td>
</tr>
<tr>
<td>Mirror Test</td>
<td>Stereo</td>
</tr>
<tr>
<td>Colored Lenses</td>
<td>PAM</td>
</tr>
<tr>
<td>OKN</td>
<td>Menance test</td>
</tr>
<tr>
<td>Prism Test</td>
<td>Others</td>
</tr>
</tbody>
</table>
Fog Lens

Asymmetric vision loss

Easiest with a trial frame

Good eye: Place +6 and -6 cyl lenses both axis 180 to effectively make plano lens
Bad eye: plano or similar set up; Dr’s preference

Have the pt read a chart full of BCVA of good eye and while they are reading move the axis of one lens so it blurs the good eye and the pt is reading the chart with the bad eye

Second version:
Check near acuity of good eye with high plus lenses, then suddenly switch to distance chart and have pt read chart with “bad eye”
VA: Mirror test

Test for gross vision

Move a mirror in front of a patient’s face and their eyes will have a horizontal movement (pursuits)

VA: OKN

Gross vision corresponds to at least 20/400

Works on the same principle as the mirror test
VA: Prism Test

4 BO over the weak eye (typically)

Vertical 4 BU

   Half way over pupil of the good eye; bad eye covered; monocular diplopia

   Ask if clarity of two lines is the same

Uncover bad eye and move prism down and completely cover good eye

   Pt will say they still see double if they have good vision

Duane Test: 10 BU while reading paragraph text over bad eye; look for hesitation
VA: Proprioception

When a patient reports blindness

Finger to nose

Index fingers end to end
VA: Menace, Provocative test, Signature

Menace: reflex testing

Provocative: make someone read something funny, inappropriate, or shocking and watch for response

Signature: blind pts can still write their signatures; not scribbles
Visual Fields

Confrontations

Goldmann

Tangent
Fig. 15.3. The differential diagnosis of concentric constriction of the visual field. Top Typical configurations of the visual field in feigned loss of peripheral vision. a Symmetrical constriction of the more peripheral isopters (tunneling of the visual field). b Spiraling of isopters. c Crossing of adjacent isopters. Bottom Constriction of the visual field in cases of organic disease. d Symmetrical narrowing predominantly of the central isopters, caused by loss of media clarity. e Asymmetric constriction of the peripheral isopters in heredofamilial retinal degenerations. f Sharp discontinuities of isopters at the vertical midline and macular sparing in visual field loss caused by bilateral occipital lobe disease.
Fig. 20.1: Neurobehavioral visual field examination on tangent screen. The patient's visual field is initially tested at the indicated distances.
Electrodiagnostics/Psychophysical testing

VEP

ERG

Dark Adaptometry
Neuro-imaging consult

CT

MRI
Psychology/Psychiatrist Consult

Patient dependent
Treatment

Reassurance

Therapy
1. 48 WM

Hx of TBI and complex PTSD
  2006 NBR LOC <10 mins
  2009 NBR -LOC
Chief complaint blur dist and near; glare; headaches from focusing near; dryness;
UIW (4/19) checked him for glaucoma
Lasik 2004
## Entrance Testing

<table>
<thead>
<tr>
<th>DVA sc</th>
<th>NVA sc</th>
</tr>
</thead>
<tbody>
<tr>
<td>OD 20/30</td>
<td>OD 20/60</td>
</tr>
<tr>
<td>OS 20/30</td>
<td>OS 20/80</td>
</tr>
<tr>
<td>OU 20/25</td>
<td>OU 20/60</td>
</tr>
</tbody>
</table>
Entrance Testing

CT Distance: ortho
Near: XP

Maddox Rod sc
Vertical: Rhyper
Horizontal: eso

EOMS: FROM OD, OS; erratice saccadic movements; had to keep reminding pt to look at the target

CVF
OD: inf nasal sup nasal defect
OS: inf nasal sup nasal defect

Midline shift: pt reports double vision; mild misalignment to pt’s right

Pupils normal
Refraction

OD: plano+0.50x005 20/20
OS: plano+0.50x020 20/20
Add:+1.25

Initial horizontal diplopia when both eyes opened and pt wouldn’t accept any prism to make it single

Worth 4 fused
BV

Von grafe

**Dist V:** 1 BD OS  
**Bi:** x/6/4

**Dist H:** ortho  
**Bo:** x/4/2

Von grafe: pt reports near diplopia fused with 9 Bi

**Near V:** ortho  
**Bi:** unable

**Near H:** 10 exo  
**Bo:** unable

**Stereo:** 100 arc seconds
Ocular Health

IOP: 17 mmHG OD/OS

Anterior Seg: unremarkable

Posterior Seg:

.60 nerves OD/OS; healthy rim tissue

+FLR

Pavingstone inf temp OU
Assessment/Plan
1. **Hx of TBI: 2006 NBR (+)LOC <10 min; 2009 NBR (-) LOC**
   a. (+) inconsistent finding: EOMS, midline, CVF, diplopia reporting; repeat BV 2 months after glasses adapt
   b. (+) accommodative dysfunction: presbyopia
   c. (+) Ocular path: glc suspect (low risk) and possible binasal hemi vf defect

2. **Refractive error**

3. **Photosensitivity**
   a. Send for tint eval

4. **Headaches**
   a. Could be from uncorrected ametropia vs diplopia

5. **Glcs suspect secondary to large C/D; doubt glc at this time**
   a. Large C/D
   b. ONH ok for iop
   c. Monitor

6. **Possible binasal hemianopsia**
   a. Abnormal confrontations
   b. Pt asymptomatic
   c. 1 month VF
1 month VF 10/31/19
Patient 2
57HM

Seen at DPC since 2009 corrected 20/20- and 20/25

Glc Suspect secondary to .5/.55 nerves and HVF

Always been observing annually

Seen 3/2019 for TBI exam and subsequently sent to UIW for tangent screen visual fields
Visual Fields 2017
2019 fields
UIW Electrodiagnostics 6/5/19

VEP- “normal, robust amplitudes and normal latencies, providing no evidence of central visual pathway dysfunction”

ERG- “photopic negative responses were within normal limits, as were multi luminance flicker ERG and high-luminance flicker ERGs”

But…. “Measurements had to be repeated (OS only), due to lead and electrode issues but no evidence of retinal or optic nerve dysfunction”

Tangent Screening OU- “pt presents with tubular fields consistent with non-organic psychogenic origin for vision loss”
UIW repeat Testing 10/16/19

Tangent Screen: “tubular response 7 inches @ 1 meter and 9 inches @ 2m”

ERG: “full field flash ERG show largely normal amplitudes and latencies though patient cooperation was limited making several repetitions of ERG recording necessary”
PRESENTING SPECTACLE Rx: (#1)
OD: -0.50DS  Add: +1.25  DVA: 20/200
OS: -0.50DS  Add: +1.25  DVA: 20/70
OU: NVA 12pt@30cm


HIRSCHBERG: eyes grossly aligned

FINAL SPECTACLE Rx:
OD: -0.50 -0.50 x 180  Add: +1.00  DVA: 20/25-
OS: Plano  -1.00 x 015  Add: +1.00  DVA: 20/20-2
Assessment/Plan

Possible visual motion sensitivity syndrome- increased peripheral clutter causing him to “shut down”

- Binasal occlusion
- Applied to lenses and pt likes them
- RTC 1 month

VIST- giving him voice output tech because was an avid reader before injury

Neuropsychology consult
Take Aways!
Patient 2
2. 43 AAM

2008 BR (+) LOC 5 min; 2010 NBR (-) LOC
Migraine, TBI, PTSD, anger/irritability
First exam 9/27/18

   CC: trouble focusing small print, photophobia

   Hx retinal hole repair and glaucoma; diplopia; words jump on the page

   Nerves: .65 and .7 IOP on two meds with IOP’s of 15 OD/OS

   Send for field to establish glc care with VA

   RX: -0.75 OD 20/20 -1.25 OS 20/20

Pt very upset he was dilated and yelled at the doctors
Second exam 11/1/18

CC: wants dark window tint for his vehicle because sunglasses block his vision; has not received glasses and isn’t sure why he is here

Wearing dark sunglasses and hood; insistent that provider would give him a letter for window tint; when the provider mentioned the tints will help him and became aggravated and was asked to leave

The wife wanted to talk to the provider but the husband would not her talk

** pt was called and talked to later; came to understanding to try tints and then window tint if the sunglasses don’t help
1 year later

Pt did not take visual fields

At my visit 9/23/19

CC: vision worse, depth perception is bad, stopped driving, eyes twitching,

“Hasn’t seen his glc doctor in a while and his PCP changed his glc drops”

VERY DARK TINTS: 20/25; 20/40 PH 20/30 OU 20/25

RX -1.00+1.00x180 20/20 and -1.75 20/20 but prefers habitual rx

IOP 30/28 and 32/34

Now a .55 and .80 with vessel baring inf rim and excavated

Send to datapoint to lower pressures: refuses to let doctors take his pressures
Visual Fields
Plan/Assessment
1. TBI; BR (+) LOC <30 min 2008

2. RE- prescribe new tints

3. POAG OU

   Currently on unknown hypotensive; send to DPC to establish glc care

   DPC glc specialist/co-resident started him on: simbrinza TID and Travatan Z
   QHS OU

   Discontinued- alphagan P and lumigan
New Patient
Diagnosis of Low Vision and Fundus Flavimaculatus

ALEX CHRISTENSEN, O.D.
Case Report - Exam #1

- 49 y/o AA female
- June 17, 2019
- CC: Blurry vision and that she would like bifocals
- Admitted to the Substance Abuse Treatment Program (SATP)
  - June 13, 2019
- Pt was calm and appropriately oriented
- LEE: 04/10/19 - Shreveport Ophthalmology clinic
  - Pending appointment: 06/21/19 - Retina specialist per pt
Patient History - Exam #1

- Ocular Hx
  - Recent ocular trauma of black eyes
  - No current medical history
  - No known allergies.
  - Smoking Hx: current/working to quit

- Medications taken:
  - Amlodipine
  - Aripiprazole
  - Cetirizine
  - Diclofenac
  - Duloxetine
  - Fluticasone
  - Gabapentin
  - Trazodone
VA distance sc:
   20/150 OD/OS, PH NI
VA near sc:
   20/200 OU
Refraction:
   +0.25 sph OD  20/100
   -0.25 sph OS  20/80
   Near OU: +1.75  20/40

EOM: Smooth, accurate, full and extensive OU
Confrontational visual fields: Full to counting OD/OS
Pupils: round, equal, reactive to light, (-) afferent pupillary defect
## Anterior Segment Examination

<table>
<thead>
<tr>
<th>Item</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lids and lashes</td>
<td>Clear OU</td>
</tr>
<tr>
<td>Conjunctiva</td>
<td>Clear OU</td>
</tr>
<tr>
<td>Cornea</td>
<td>Clear OU</td>
</tr>
<tr>
<td>Tear break up time</td>
<td>&gt;8 seconds OU</td>
</tr>
<tr>
<td>Iris</td>
<td>Flat, no masses, no rubeosis, no synechia OU</td>
</tr>
<tr>
<td>Angle</td>
<td>4x4 OD/OS</td>
</tr>
<tr>
<td>Anterior chamber</td>
<td>Deep and quiet OU</td>
</tr>
<tr>
<td>Lens</td>
<td>Clear OU</td>
</tr>
<tr>
<td>Intraocular pressure</td>
<td>15mmHg OD, 14mmHg OS</td>
</tr>
<tr>
<td><strong>Fundoscopic Examination</strong></td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Optic nerve head</td>
<td>Pink rims, flat, distinct margins OU</td>
</tr>
<tr>
<td>C/D ratio</td>
<td>0.20 OD/OS</td>
</tr>
<tr>
<td>Macula</td>
<td>OD: clear</td>
</tr>
<tr>
<td></td>
<td>OS: <em>Grade 1</em> epiretinal membrane</td>
</tr>
<tr>
<td>Vitreous</td>
<td>OU: <em>Vitreal syneresis</em></td>
</tr>
<tr>
<td>Posterior pole</td>
<td>Unremarkable OU</td>
</tr>
<tr>
<td>Vessels</td>
<td>Normal caliber and crossings OU</td>
</tr>
<tr>
<td>Periphery</td>
<td>No retinal tears/holes/detachments OU</td>
</tr>
</tbody>
</table>
Assessment/Plan – Exam #1

- **Assessment:**
  - Refractive Error
    - Subjective compliant not consistent with objective findings
    - Suspicious of malingering
    - Macular OCT showed macular thickness volume loss
    - Recent h/o blunt force trauma/black eyes may be cause of decreased vision or to another yet specified retina disorder

- **Plan:**
  - RTC 5 weeks for repeat refraction
  - Ordered new glasses with photochromic tint
July 24, 2019
RFV: 1 month follow up, full exam with dilation, OCT imaging and retinal photos
CC: She could not see and everything was blurry
- Reports near > distance blur with and without correction
- Slowly worsening over the past 1-2 years
- Reports vision not being as good as peers when in the Service
Smoking 1/2 pack per day of cigarettes
Denies ocular pain
Exam #2

- **VA distance cc:**
  - 20/400 OD/OS PH NI

- **Optokinetic Drum:**
  - (+) nystagmus OD/OS
    - 20/400 or better

- **Refraction:**
  - Plano +0.25 x180 OD 20/400
  - -0.25 +0.75 x180 OS 20/100
  - Near VA: +2.00 20/40

- **EOMs, CVF, and Pupils:**
  - Within normal limits

- **Anterior segment findings:** stable to LEE
<table>
<thead>
<tr>
<th>Fundoscopic Examination</th>
<th>OU: Grade 2 epiretinal membrane</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macula</td>
<td>OU: Vitreal syneresis</td>
</tr>
<tr>
<td>Vitreous</td>
<td>OU: Diffuse hypoflourescent flecking throughout posterior pole and periphery</td>
</tr>
<tr>
<td>Posterior pole</td>
<td>OD: CHRPE 3DD size temporal OS: Unremarkable</td>
</tr>
</tbody>
</table>
Assessment/Plan – Exam #2

Assessment:
- Fundus Flavimacularus

Plan:
- Educate patient fulltime wear of glasses for safety
- Educate no driving
- Referred patient to Visual Impairment Service Team (VIST) services
- Given handout for vision impaired support groups
- Co-signed Chief of Eye Clinics of CTX, and Temple Low vision specialist for remote second opinion
Follow Up #3

- July 25, 2019
- Reason for visit: Visual field and to fully educate patient on condition
Assessment/Plan #3

- Education on condition
- Reaffirmed importance to set up appointment with the Shreveport VA eye clinic when she gets discharged
- Counseled to avoid smoking
- Seek opinion of eye care provider prior to starting certain medications such as retinoids, plaquenil, tamoxifen, etc
- Tell children the diagnosis of fundus flavimaculatus and to ask them to get eye exams soon.
Differential Diagnoses

- Retinitis Punctata Albescens
- Familial Drusen
- Stargardt Disease
- Fundus Flavimaculatus
Differential Diagnoses

- Retinitis Punctata Albescens
  - Childhood onset night blindness
  - White retinal deposits
  - Decreased vision of around 20/40
  - Peripheral retinal atrophy

![Image of Retinal Image]
Differential Diagnoses

- **Familial Drusen**
  - Confluent soft drusen near the macula
  - Drusen temporal to the optic nerve head that are arranged in radial lines that converge towards the fovea
  - Large drusen nasal to the optic nerve head
Fundus Flavimaculatus vs Stargardt Disease

- Genetically the same
- Autosomal recessive
- Difference between the two
  - Fundus Flavimaculatus tends to have a later disease onset
  - Slower visual deterioration
- Fundus Flavimaculatus can be seen as a subset of Stargardt disease
Fundus Flavimaculatus

- Early stages:
  - Asymptomatic
  - Visual loss without clinical signs
  - Malingering
  - Misdiagnosed as amblyopia
Fundus Flavimaculatus

- **Symptoms:**
  - Central vision loss
  - Photophobia
  - Abnormal color vision
  - Slow dark adaptations

- **Fundus:**
  - Yellow-whitish “fishlike” (pisciform) shaped flecks

- **Later stages:**
  - Macular atrophy showing a “beaten bronze” appearance

- **Rare complication:**
  - Choroidal neovascular membranes (CNVM) or subretinal bleeds
Mutation of the ABCA4 gene
ABCA4 gene encodes for a photoreceptor rim protein, ABCR
When ABCR is defected, it causes the accumulation of protonated N-retinylidene-PE in the rod outer segment
N-retinylidene-N-retinylethanolamine (A2E), a byproduct of N-retinylidene-PE then accumulates in the RPE and is toxic
Photoreceptors then die due to the loss of RPE support function
Specialty Testing

- Visual fields
  - Early stage: normal
  - Later stage: relative central scotoma → absolute central scotoma

- Fundus Autofluorescence
  - Hyperfluorescence at lipofuscin deposits (flecks)
  - Hypofluorescence at sites of atrophy
Specialty Testing

- Fluorescein Angiography
  - “Dark-choroid” due to the lipofuscin accumulation in RPE
  - Hyperfluorescent flecks
  - Hypofluorescence at fovea depending on level of atrophy

- Electrophysiology Testing
  - EOG: subnormal
  - ERG: normal
Treatment

- **Prognosis**
  - The earlier the onset of the disease, the more likely it is that both eyes will eventually have vision of 20/200 or worse

- **No current treatment**
  - Refrain from having a Vitamin A rich diet
  - Avoid direct sunlight exposure
  - Use ultraviolet blocking sunglasses and brimmed hats

- **Experimental Treatment**
  - Isotretino in
Low Vision

- Optical devices
  - Magnifiers for near vision
  - Telescopes for distance vision
- Non-optical devices
  - Tints
- Educating patients on eccentric viewing
Clinical Pearls

- Perform a visual field, fundus autofluorescence, fluorescein angiography, and electrophysiologic testing as needed to make a diagnosis.
- Do genetic testing on the ABCA4 gene to help find the exact etiology.
- Educate the patient on low vision services and aids such as magnifying devices.
Conclusion

- FFM can be easily misdiagnosed
- Remember what diagnostic examinations are available, fundus autofluorescence, fundus angiography, electrophysiologic testing, and genetic testing
- Educate the patient on the realistic prognosis but have empathy for how it will change the patient’s life
- Low vision services can be very impactful to help the patient have a functional life
References


Questions?
INCORPORATING ELECTRODIAGNOSTIC TESTING IN THE MODERN OPTOMETRIST’S PRACTICE

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ROSENBERG SCHOOL OF OPTOMETRY PRIMARY CARE RESIDENT
DISCLOSURES

• No conflicts of interest or financial disclosures to report.
VISUAL EVOKED POTENTIAL (VEP)

• 1\textsuperscript{st} mentioned description of VEP was in the year 1934.

• “The VEP is an evaluation of the entire visual system from the retina through the primary visual cortex. It measures the conduction time of neuronal activity from the retina to the occipital cortex and is used clinically as a measure of the functional integrity of the visual system.”

• Historically VEPs have been used to study the function of the afferent visual pathway objectively.
VEP REFRESHER

- Types of VEP
  - Flash VEP
  - Pattern VEP
  - Multifocal VEP

- VEP wave components:
  - N1 (N70)- Visual cortex
  - P1 (P100)- Dorsal extrastriate cortex
ELECTRORETINOGRAM (ERG)

• 1st recorded electroretinogram (ERG) in humans was performed in the year 1877.
• ERG works by recording retinae responses to a light stimulus of known luminance.
• Historically ERGs have been used to monitor/study retinal function objectively.
  • A wave- photoreceptor response
  • B-wave bipolar/muller cells response
ERG REFRESHER

- **Full Field ERG**
  - Measures a global retinal response.
  - May mask smaller retinal lesions.

- **Multifocal ERG**
  - Measures multiple local macular responses.
  - Capable of revealing localized macular dysfunction.
ERG

- Pattern ERG
  - Measures ganglion cell function and correlates to optic nerve integrity (Mohsen)
  - P50 may be used to check macular function.
  - N95 used for ganglion function.
Figure 2: Mean visual evoked potential P100 latency, N75-P100 amplitude and interocular latency difference (mean of both the eyes) compared in cases and controls.
Reproducibility of Fixed-luminance and Multi-luminance Flicker

Electrodiagnostic testing shows that the fixed-luminance phase value, a frequency domain analog of the time domain B-wave implicit time, was the most highly reproducible across test-retest comparisons. Our preliminary findings suggest that office-based flicker ERG, especially the fixed-luminance phase parameter, is highly reproducible and could be reliably used as a biomarker to initially characterize and quantify global retinal cone and bipolar cell dysfunction in diabetes and potentially be employed to follow eyes with mild-to-moderate NPDR over time. Additional potential applications include serving as an adjuvant to fluorescein angiography and supporting the management of anti-VEGF therapy.

Keywords: diabetic retinopathy, diagnostic testing, flicker electrotetinography retinal dysfunction, ultra-widelfield fluorescein angiography
FIXED LUMINENCE ERG
Reduced amplitude and delayed latency in foveal response of multifocal electroretinogram in early age related macular degeneration

Methods
In this study, early AMD changes were defined by a significant delay in the P1 amplitude as well as a significant delay in the N1 latency of foveal responses from pre-AMD or early AMD eyes and their asymptomatic fellow eyes when compared with normal control eyes. These findings suggest that both foveal P1 amplitude and foveal N1 latency in MERG measurements may be sensitive means to detect early foveal AMD changes.

Table 4  Summary of P1 amplitudes and N1 latencies with configuration C

<table>
<thead>
<tr>
<th>Group</th>
<th>R1 Amplitude (μV/deg)</th>
<th>R1 Latency (ms)</th>
<th>R2 Amplitude (μV/deg)</th>
<th>R2 Latency (ms)</th>
<th>R3 Amplitude (μV/deg)</th>
<th>R3 Latency (ms)</th>
<th>R4 Amplitude (μV/deg)</th>
<th>R4 Latency (ms)</th>
<th>R5 Amplitude (μV/deg)</th>
<th>R5 Latency (ms)</th>
<th>R6 Amplitude (μV/deg)</th>
<th>R6 Latency (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (n=20)</td>
<td>60.1 (15.5)</td>
<td>14.2 (1.3)</td>
<td>32.5 (5.7)</td>
<td>14.6 (1.0)</td>
<td>21.5 (3.2)</td>
<td>14.5 (0.6)</td>
<td>16.2 (2.6)</td>
<td>14.5 (0.8)</td>
<td>13.7 (2.5)</td>
<td>14.8 (0.7)</td>
<td>12.2 (2.4)</td>
<td>15.1 (0.8)</td>
</tr>
<tr>
<td>AMD (n=15)</td>
<td>44.5 (13.7)</td>
<td>17.1 (1.0)</td>
<td>25.6 (7.1)</td>
<td>15.6 (1.5)</td>
<td>19.7 (4.9)</td>
<td>14.4 (1.0)</td>
<td>15.1 (3.6)</td>
<td>15.2 (1.0)</td>
<td>12.8 (3.7)</td>
<td>15.8 (1.0)</td>
<td>11.4 (3.6)</td>
<td>16.0 (0.7)</td>
</tr>
<tr>
<td>Fellow eye (n=15)</td>
<td>49.2 (13.4)</td>
<td>15.8 (1.1)</td>
<td>27.2 (6.8)</td>
<td>15.0 (1.0)</td>
<td>20.0 (5.0)</td>
<td>14.7 (1.2)</td>
<td>15.0 (3.9)</td>
<td>14.8 (0.9)</td>
<td>12.8 (3.9)</td>
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</tr>
</tbody>
</table>

six concentric rings centred on the fovea.
AMD AND MF-ERG
PLAQUENIL AND MF-ERG
Steady-state pattern electroretinogram and short-duration transient visual evoked potentials in glaucomatous and healthy eyes

MagnitudeD was the most accurate ss-PERG parameter for discerning glaucomatous dysfunction across all stimuli. MagnitudeD represents the amplitude of the ss-PERG signal as well but is also heavily influenced by the phase consistency of the signal. This phase is calculated by averaging measurements taken during each testing sequence. Phase consistency describes the SD of these measurements. A low phase consistency indicates a failure of the RGCs to adapt to the large energy demand of an ss-PERG stimulus, leading to different phases as testing time goes on. This difference in phase consistency is demonstrated in Figures 1c,d. A low ss-PERG phase consistency and decreased amplitude have been previously seen in patients with glaucoma. Accordingly, we found MagnitudeD to be significantly
GLAUCOMA AND PERG

Glaucoma
SUMMARY

- Electrodiagnostic testing is a clinically relevant tool that is underutilized in the modern everyday optometric practice.

- As the years advance, electrodiagnostic testing will continue to do the same.

- I believe that with the increasing amount of studies on the clinical use of these electrodiagnostic tests it is only a matter of time until they become a part of the standard of care for these common ocular conditions.
REFERENCES


