Electrophysiology in Vision

How VEP and ERG Testing Can Impact Your Treatment Decisions

J. James Thimons, OD, FAAO, ABO
Medical Director / Glaucoma Institute
Ophthalmic Consultants of CT
jimthimons@gmail.com
Caveat for all Imaging devices

CAUTION
THIS MACHINE HAS NO BRAIN
USE YOUR OWN
What do you think of when you think of electrophysiology?
How about when you think of EKG?
Electrophysiology in Eye Care
Electrophysiology Of Vision

ERG
Electroretinogram
Electrical activity of the retina

VEP
Visual Evoked Potential
Electrical activity of the visual cortex
Electrophysiology of Vision

ERG (Electroretinogram)
- Electrical activity of the retina

VEP (Visual Evoked Potential)
- Electrical activity of the visual cortex
Electrophysiology of Vision

ERG

Electroretinogram

pERG
Pattern-Electroretinogram

fERG
Flash-Electroretinogram
Electrophysiology Of Vision

ERG (Electroretinogram)

- Inner retinal layers (Pattern-Electroretinogram)
- Outer retinal layers (Flash-Electroretinogram)
NEURO-PHYSIOLOGY

Phototransduction

Conversion of light into electricity
Ganglion cell (action potential)

Photoreceptor (phototransduction)

Light

Electricity

Bipolar

Electricity

Ganglion cell (action potential)

Electricity

Ganglion cell axon

Electricity

Relay neuron

Electricity

Relay neuron axon

Electricity

Visual cortex neuron
Electrophysiology objectively measures **strength** and **speed** of the visual signal to the brain (VEP) or retina (PERG).
Clinical Applications in Eye Care

- Inherited retinal dystrophies
- Vascular diseases including diabetes
- Opaque media or trauma
- Retrobulbar neuritis
- Unexplained visual loss
- Infant with questionable vision
- Toxic and nutritional eye disease
- Glaucoma
- Suspected intracranial lesion
PREVIOUS LIMITATIONS

- Test time was approximately 45 minutes
- Required highly trained operators
- Limited to large research institutions
- Required highly trained neurophysiologists
Time, Space, Cost

Continuum

• Actual test time is considerably shorter
• Does not require highly trained operators
• Easy to use, intuitive software
• Comfortable for the patient, convenient for doctor and staff
**VEP MADE SIMPLE**

Visual Evoked Potential (VEP)

Visual – patient observes a visual stimulus  
Evoked – generates electrical energy at the retina  
Potential – measure the electrical activity in the visual cortex

Objective measurement of the function of entire vision system;  
No verbal response or “button pushing” like visual field tests
International VEP Standard

- Time (latency) is measured in milliseconds (ms)
- Amplitude is measured in microvolts (μV)
- N75-P100-N135 Complex
  - N75: Negative Pulse around 75ms
  - P100: Positive pulse around 100ms
  - N135: Negative pulse around 135ms

*Figure 2. A normal pattern reversal VEP.*
Clinical Applications

- Clarify Differential Diagnosis – Is it Systemic, Trauma or Ocular?

- When Standard Tests are Unattainable or Unreliable – Visual Field Can’t be Performed or Results are Unreliable

- Other Tests are Inconsistent or Borderline – Patient Symptom and/or Test Results are Equivocal

- Monitor Subclinical Ophthalmic Disease – Functional Changes
VEP and PERG stimuli:

Check or line size
Contrast
Speed of reversal pattern

VEP – entire visual pathway

ERG – Retinal function
Why VEP?

- Many optic nerve diseases are asymptomatic because central vision is not affected until late in the disease¹

- Diagnosis and management of optic nerve disorders are often based on structural or subjective visual field tests²

VEP is an objective, functional test that can help discriminate between healthy and glaucomatous eyes²

Visual Evoked Potential (VEP)

MAIN INDICATIONS

• Glaucoma
• Multiple Sclerosis
• Ischemic Optic Neuropathy
• Traumatic Brain Injury
• Amblyopia
• Other Neuropathies
“Visual evoked potentials (VEPs) can provide important diagnostic information regarding the functional integrity of the visual system.”
Where Does Glaucoma Begin?

Researchers at UCSD, Vanderbilt University, and the University of Toronto claim glaucoma begins in the LGN and is later seen as progression measured by visual field and structural changes measured by OCT, GDX, HRT, etc.
Glaucoma & the Brain

- Led by Robert N. Weinreb, M.D., a team of scientists at UCSD’s Glaucoma Center recently reported that glaucoma is not a disease restricted only to the eye. Their study, "Loss of LGN Neurons in Glaucoma," appeared in the March 2000 issue of Archives of Ophthalmology.

- Though commonly misunderstood as a disease of "increased eye pressure" the hallmark of glaucoma is the optic nerve fiber loss, regardless of the pressure.

- Ninety percent of the optic nerve fibers from the eye terminate in the lateral geniculate nucleus. The visual cortex detects the electrical signals, processes them, and provides us with our sense of sight.

- Dr. Weinreb’s group, including lead author Yeni Yucel, M.D., Ph.D. (a neuropathologist and UCSD Postdoctoral Glaucoma Fellow at the time, presently the Director of Ophthalmic Pathology at the University of Toronto), found in a primate model that there was extensive loss of nerve cells in the lateral geniculate nucleus with progressive glaucoma, a process known as transneuronal degeneration. According to Dr. Weinreb, by studying changes in the brainstem we may better understand what causes vision loss in glaucoma. This information could be critical in helping researchers determine how to prevent vision loss in patients with known glaucoma or at risk for delaying it.
VEP and Glaucoma: Well Defined Science

The Visual Evoked Potential in Glaucoma and Ocular Hypertension: Effects of Check Size, Field Size, and Stimulation Rate

Increased pattern VEP latency was significantly correlated with both the severity and location of visual field defects and the degree of cupping and pallor of the optic disc.
The Visual Evoked Potential in Glaucoma and Ocular Hypertension: Effects of Check Size, Field Size, and Stimulation Rate

Vernon L. Towle,∗ Anne Masiakowicz, Samuel Soiok † and Bernard Schwartz‡

In order to determine the optimum stimulus conditions for the detection of optic nerve damage due to glaucoma and ocular hypertension, checkerboard pattern reversal visual evoked potentials (VEPs) were recorded from 20 glaucoma patients, 20 ocular hypertensive patients, and 20 age-matched normals. Two check sizes (12′ and 48′), two field sizes (14′ and 28′), and two alternation rates (1.9 and 7.5 alt/sec) were used. All subjects had visual acuities of 20/40 or better in each eye and equal pupils of 2 to 5 mm diameter. The largest number of VEP abnormalities were found with large checks (48′) reversing at a fast rate (7.5 alt/sec). After correcting for the effects of age, visual acuity, and pupil size, 16 of 30 eyes with glaucomatous visual field defects had abnormally long VEP latencies under this condition (beyond the 99% confidence limit of the normal subjects). Nine of 40 ocular hypertensive eyes also had abnormally long latencies. Increased pattern VEP latency was significantly correlated with both the severity and location of visual field defects and the degree of cupping and pallor of the optic disc. VEP latency was not significantly related to intraocular pressure. Invest Ophthalmol Vis Sci 24:175–183, 1983

The pattern visual evoked potential (VEP) has been shown to be sensitive to optic nerve lesions caused by demyelination, sclerosis, and compression of the anterior visual pathway.1 Glaucoma has also been reported to affect the VEP by causing both reductions in amplitude2–11 and increases in latency.12–13 In optic disc cupping and the presence of visual field loss.14–19 In ocular hypertension the pattern VEP has been normal21 unless eccentric viewing22 or provocative techniques have been employed.23–25

In those nonprovocative studies in which abnormally long VEP latencies were obtained it is not clear whether the results were due, in part, to the confounding effects of miosis, pupils,26,27 advanced age,28 or reduced visual acuity.29 All three of these factors can cause VEP latency increases. The one study that carefully controlled for the effects of these three variables30 reported a small group difference in relative interocular VEP latency for glaucoma patients and normal control subjects.

The purpose of the present study was to obtain VEP latencies for various stimulus conditions in carefully selected groups of ocular hypertensive and glaucoma patients and visually normal controls while controlling for the confounding effects of pupil size, age, and visual acuity.

Materials and Methods

Subjects

All subjects were free from neurologic disease, had clear media, visual acuities of 20/40 or better in each eye, and equal pupils of 2 to 5 mm diameter. The 60 subjects formed three groups of 20 subjects each, as described below.

Group 1: Normal controls. This group consisted of ten volunteers (five men and five women) less than 50 years of age (3 ≤ 30 years) and ten volunteers (six men and four women) older than 50 (≥ 63 years). All of these subjects had normal fundi and discs, full and normal visual fields as measured on the Goldmann perimeter by static and kinetic methods, and ocular pressures less than 21 mmHg as measured by the Goldmann applanation tonometer. Stereoscopic fundus photographs were taken with the Donaldson stereoscopic fundus camera from six of these subjects.

From the Department of Ophthalmology, New England Medical Center, Boston, Massachusetts.


Reprint requests: Vernon L. Towle, PhD, Department of Neurology, University of Chicago, Box 425, 150 East 59th Street, Chicago, IL 60637.
Under this stimulus condition, 16 of the 30 eyes with glaucomatous field defects had abnormally long VEP latencies. None of the VEPs from the normal subjects had abnormally long latencies. It is of particular interest that 9 out of 40 of the eyes of ocular hypertensive patients had abnormally long VEP latencies. These nine eyes were from five patients.

**VEP Latency and Field Defects**

VEP latency for the 48 checks presented at the fast alternation rate in the small field was correlated positively with the severity of the field defect \( r = -0.48, P < 0.0001, df = 58 \) as shown in Figure 5. The results of this categorical breakdown were supported by a quantitative analysis of the visual fields: VEP latency was negatively correlated with the distance to the O-4c isopter \( r = -0.46, P < 0.01, df = 28 \) and the V-4c isopter \( r = -0.35, P < 0.01, df = 29 \). VEP latency was also correlated negatively with the area of the O-4c isopter \( r = -0.43, P < 0.01, df = 28 \). All of these correlations indicate that the size and location of a visual field defect can influence VEP latency.

In spite of these significant correlations, it should be noted that nearly half of the eyes with glaucomatous field defects (14 of 30 eyes) generated normal VEPs even though many of these defects clearly en-
In spite of these significant correlations, it should be noted that nearly half of the eyes with glaucomatous field defects (14 of 30 eyes) generated normal VEPs even though many of these defects clearly encroached upon the macula. We performed additional tests on 9 of these patients in an attempt to understand why these patients generated normal VEPs.
Reducing the intensity of the stimulus display by as much as 1.5 log units—to the range of the targets used to map the visual fields—caused abnormal VEPs (either abnormally long in latency or unrecordable) in five of these nine patients.
The finding that is of clinical importance is the presence of abnormally long VEP latencies in some patients with ocular hypertension. The abnormal prolongation of VEP latency in these eyes may reflect subclinical optic nerve lesions that have not been uncovered with other techniques.

VEP Latency and Ocular Hypertension

The finding that is of clinical importance is the presence of abnormally long VEP latencies in some patients with ocular hypertension. The abnormal prolongation of VEP latency in these eyes may reflect subclinical optic nerve lesions that have not been uncovered with other techniques.

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Additional Clinical Papers


Glaucmatous Brain Damage

VEP (Function)

Brain LGN

Eye

Stress (OHT)

OCT
HRT (Structure)
GDX

After Treatment

Understanding VEP Wave:

NORMAL

ABNORMAL
Reading Results:
Normal
Reading Results: Abnormal
Case Study 1 – unreliable visual field

**Introduction**
- 70 year old white female
- Glaucoma suspect for elevated IOP’s
- General health history unremarkable; mother has Open Angle Glaucoma

**Findings**
- Best corrected acuities 20/25 OD OS
- Pupils and ocular motilities normal
- Slit lamp exam unremarkable and angles were open to grade IV OU
- Intraocular pressures at 4:30 PM were OD 23mmHg and OS 21mmHg
- Low reliability on visual field
- Central corneal thicknesses 506 and 507
Case Study 1

- Humphrey 24-2 Visual Field Testing showed a slight depression in the inferior Bjerrum area, and borderline GHT findings although this field was somewhat unreliable.
- OS visual field was without defect.
- 2nd visual field equally unreliable.
Case Study 1

- Optic nerve cupping was .50 OD and .35 OS
- HRT scans normal
Case Study 1: VEP Aids in Decision to Treat Early POAG

- VEP studies revealed
  - increased latency at 15% contrast level OD
  - 85% contrast level normal OD
  - both 15% and 85% normal OS

**Diagnosis and Treatment**

The ability to obtain a new objective measurement of optic nerve function by performing a VEP is significant in the decision to treat patients at the earliest level of POAG.”

- Tx: Lumigan 0.01 % 1 gtt QHS OU
- Follow up visit one month
- Repeat at 6 months with 10-2 VF
Case Study 2 – borderline for treatment

Introduction
- 45 year old white male
- General health, eye health and family health histories unremarkable
- Routine eye exam without significant complaints

Findings
- Best corrected visual acuities with a small compound myopic correction of 20/20 OD OS
- Pupils, color vision, motilities, and muscle balance normal
- Intraocular pressures at 3:30 pm were 15mmHg in each eye
- Asymmetric cupping on Fundoscopic exam as determined with a 78 D lens of .65 OD and .40 OS
- Intraocular pressures at 10:30 in the morning were 15mmHg OU
- Central corneal thickness was 550 OD and 550 OS
- Gonioscopic view of the angles was open to the ciliary body OU with a 30 degree approach angle and grade II pigmentation
• Threshold Humphrey 24-2 visual fields full without defects OD OS
Case Study 2 (cont.)

- HRT optic nerve scans confirmed asymmetric cupping and findings were abnormal OD and borderline OS
Case Study 2: VEP Aids in Decision Not to Treat a Glaucoma Suspect

- VEP studies revealed
  - both 15% and 85% normal OD
  - both 15% and 85% normal OS

Typically most clinicians would have considered treating a patient of this type.

VEP allows for an objective assessment in addition to anatomy that supports monitoring instead of Tx.
Case Study 3

Introduction

- 56-year-old black female
- O.D. is -5.75 - 0.50 x 015 with 20/20- acuity
- O.S. is -5.75 - 0.25 x 180 with 20/20- acuity
- Mild cortical cataracts
- No apparent retinal pathology
- No significant medical or ocular history
- Patient states her vision in decreasing and she “just can’t see right”
Case Study 3

Normal optic nerve appearance
Case Study 3

Cirrus OCT retinal nerve fiber layer scan
Abnormal sector plot analysis on right eye
Superior RNFL defect on right eye
No clinically significant asymmetry
Case Study 3

Abnormal VEP waveforms in each eye
Delayed P100 peak times in both high-contrast and low-contrast VEP responses
N75-P100-N135 complex is abolished in the low-contrast response in the left eye
Clinically significant asymmetry
Diagnosis and Treatment
Patient was referred out for an MRI
MRI results found a cavernous hemangioma

Medical Decision Making:
In patients with subjective visual disturbances VEP can be a valued adjunct to traditional imaging.
Normal VEP test results may suggest a conservative approach such as monitoring (i.e., temporary cessation of the diagnostic program).
Abnormal VEP test results suggests a continuation of the diagnostic program with one or more of the following treatment options:
   - Confirm all abnormal findings within 1-2 weeks
   - Brain, optic nerve, orbital imaging and angiography
   - Referral as appropriate
Misinterpretation artifacts

- Localized losses of RNFL or macular thickness classified as normal due to averaging of thickness values by quadrant, sector or hemisphere
- Misinterpretation of shadow artifacts
“Green Disease”
Warning: Classification results valid for Caucasian eyes only.
There is a higher percentage of abnormal diagnostic classification since the RNFL normative databases typically do not include moderate and high myopes.

Myopic eyes are also associated with many other artifacts such as difficulty in acquiring a good image due to excessively long axial length or myopic retinal schisis affecting peripapillary RNFL thickness.
VEP - Summary for Use

VEP is an **objective, functional** test that can help discriminate between healthy and diseased eyes.

- Differentiate ocular from systemic, trauma or other conditions for co-management.

- Diagnosis and management of ophthalmic concerns:
  - Alternative to VF or VA (need reliable results for diagnosis and treatment)
    - Visual Field limitations 368.40
    - Subjective Visual Disturbance 368.10
  - Questionable vision or diagnostic inconsistencies
    - Conversion disorder (malingering) 300.11
    - Visual disturbances 368.xx
    - Optic Nerve and Pathway disorders 377.xx
  - Subclinical vision disorders for diagnosis and management
    - Disorders affecting optic nerve 377.xx
    - MS/Optic neuritis 340
    - Optic neuropathies 377.xx
    - Unexplained vision loss 368.11
    - Transient vision loss 368.12
    - Visual field defects 368.xx
    - Amblyopia/Strabismus 368.0x
    - Traumatic brain injury 850-853.xx
How Does pERG Work?

Pattern electroretinogram (pERG) is an electrical recording of retinal function in the macula and ganglion cells stimulated by contrast-reversing patterns, usually black and white.
Pattern Electroretinogram (pERG)

- pERGs are electrical signals that are a measure of the electrophysiological activity in the ganglion cells in the retina.

- Can help improve sensitivity and specificity in diagnosing neuropathies and maculopathies like macular degeneration and glaucoma when used in conjunction with other tests.

- Can also help the clinician differentiate between retinal and optic nerve disorders when used in conjunction with Visual Evoked Potential (VEP).
Clinically, pERGs can be used in patients with abnormal pattern VEPs to establish if a central retinal disorder is present and thus differentiate between retinal and optic nerve dysfunction as a cause for the VEP abnormality.

It can also be used to detect and monitor dysfunction of retinal ganglion cells caused by conditions such as glaucoma, optic neuropathies and primary ganglion cell diseases.

Thus, the pERG has clinical value in both neurological and neurological conditions.
Progressive loss of retinal ganglion cell function precedes structural loss by several years in glaucoma suspects.

pERG is influenced by the IOP

**Question**
Can IOP be inferred by the pERG result?
ERG Clinical Applications

- Differentiate retinal and optic nerve disorders

- Inconsistent or borderline test results (Patient symptoms or test results are equivocal)

  - Diagnose and manage treatment efficacy of subclinical ophthalmic disorders:
    - glaucoma
    - age-related macular degeneration (AMD)
    - diabetic edema, diabetic retinopathy
    - Toxicity/plaquenil
The pERG arises largely in the ganglion cells, driven by the photoreceptors and corresponding retinal cells. Since the pERG (in contrast to the flash ERG) is a local response from the area covered by the retinal stimulus image, it can be used as a sensitive indicator of dysfunction within the macular region and it reflects the integrity of the optics, photoreceptors, bipolar cells and retinal ganglion cells."
Steady-state pERG

“At higher temporal frequencies, that is, above 10 rps (5 Hz), the successive waveforms overlap and a “steady-state” PERG is evoked.”
• Per NIH and Bascom-Palmer:
  
  • In patients who are glaucoma suspects, PERG signal anticipates an equivalent loss of OCT signal by several years (as many as 8 years).

  DOI: 10.1167/iovs.12-11026
Pattern ERG - Steady State

- **Concentric Stimulus Fields** - Protocol driven test designed for objective, functional study of focal disease (drug toxicity, diabetic edema, AMD)

- **Contrast Sensitivity** - Protocol driven test designed for objective, functional study of diffuse disease (glaucoma, diabetic retinopathy, etc)
Concentric Stimulus Field pERG

Information affecting the central or paracentral area of the macula and ganglion cells: AMD, Plaquesnil, Diabetic Edemas

Waveforms in Phase = normal
Contrast Sensitivity PERG Test

High and Low contrast

Information affecting the retina in a diffuse pattern: Chronic Open Angle Glaucoma and Diabetic Retinopathy

High Contrast

Low Contrast
Testing Protocols: Concentric Stimulus Fields

- Stimulus delivered at 15 flips/second
- BCVA
  - Patient should be properly refracted for 24”.
- 24” testing distance
- 85% contrast
- Right Eye (OD) then Left Eye (OS)
  - 25 seconds at 24°
  - 25 seconds at 16°
Testing Protocols: Contrast Sensitivity

- Stimulus presented at 15 flips/second

- BC VA
  - Patient should be properly refracted for 24”.

- 24” testing distance

- Right Eye (OD) then Left Eye (OS)
  - 25 seconds at High Contrast (Hc)
  - 25 seconds at Low Contrast (Lc)
Normal PERG Response

3 Quick Steps To Report Interpretation

Signal Quality – Look for a green signal

Sinusoidal Peaks – Look for 3 humps

Magnitude, MagnitudeD and MagD/Mag Ratio are colorized.

Green indicates within normal limits
Yellow indicates values are borderline
Red indicates outside normal limits
PERG Report – Data Table

Magnitude (μV) is defined as the strength of the patient’s response at a reversal rate of 15 reversals per second.

Larger magnitudes are typically generated from normal eyes. Smaller magnitudes typically indicate pathology.

As the contrast level drops or the stimulus size decreases, the magnitude will typically decrease.
MagnitudeD averages the signal within the 25 second test time and takes into account the magnitude strength and the phase variability throughout the test.

In a healthy patient, the phase response tends to be consistent throughout the test. In this case, MagD is close in value to Mag.

In a patient with disease, the phase response tends to be inconsistent throughout the test - MagD will be significantly reduced in comparison with Mag.
MagD/Mag Ratio is the most repeatable measurement test-over-test. The closer the ratio is to 1.0, the lower the phase variability throughout the test, and the healthier the patient’s response. Variability in phase may indicate pathology.

MagD/Mag ratio can be used to monitor patients over time.
SNR - Signal to Noise Ratio shows how strong the signal is at 15Hz compared to noise at 15Hz. Larger numbers indicate stronger PERG signals compared to the noise.

SNR values like 5, 15, >20 show strong PERG response. Numbers less than 2 are typical of a weak response.
Artifacts are caused by blinking or patient movement. They are detected and counted. A high number of artifacts will effect the amount of data that can be analyzed.

The goal is to have a low number of artifacts. The patient should be comfortable and blink when necessary, but not excessively. The goal is less than 10. If tests results show Artifacts greater than 10, the test should be repeated.
Abnormal PERG

Missing 3 humps

Yellow indicates values compared to normal are borderline

Red indicates values are outside normal limits
PERG is an **objective, functional** test on the retina that can help discriminate between healthy and diseased eyes

- Differentiate retinal and optic nerve disorders
  - After an abnormal VEP to isolate dysfunction 794.13
  - Retinal 362.xx and Optic Nerve 377.xx concerns
- Questionable, inconsistent or borderline test results (Patient symptoms or test results are equivocal)
  - Visual Disturbances 368.xx
  - Retinal Disorders 362.xx
  - Optic Nerve function 377.xx
- Diagnose and manage treatment efficacy of subclinical ophthalmic disorders:
  - Glaucoma 365.xx
  - Age-related macular degeneration (AMD) 362.50
  - Diabetic edema and retinopathy 250.xx and 362.0x
  - Toxicity from drug use/plaquenil V58.69
The Case of the Missing Exam
The Case of the Missing Exam

WC, a 33-year-old Caucasian male was seen for a second opinion consultation regarding a diagnosis of advanced glaucoma in one eye.

History was negative for medical treatment in fact until the initial eye exam, the patient had never seen a doctor since early high school.

Patient was asymptomatic other than distance blur which precipitated the original eye exam.

Denies trauma, Fam Hx, medications.

Currently on TZ 1/0.
Case of the Missing Exam

Clinical Assessment:
- Vacc: 20/20 (OD/OS)
- Ta: 9/14 @3:30
- HFA II: As shown
- Ext: 3/3/ 2+/ +MG OD
- SLE: Unremarkable
- Pach: 567/572
- Tora: 8.4/ 14.7
- DFE: As shown
The Case of the Missing Exam
The Case of the Missing Exam

- Clinical decision?
  - What do you recommend?
  - Same drug?
  - Change drug?
  - No Tx!
Ganglion Cell OU Analysis: Macular Cube 512x128

DOB: 3/22/1982
Gender: Male
Doctor:
Exam Time: 7:24 PM
7:26 PM
Serial Number: 4000-3809
4000-3809
Signal Strength: 7/10
8/10

OD Thickness Map
OS Thickness Map

Fovea: 246, 63
Fovea: 259, 62

OD Deviation Map
OD Sectors

OS Deviation Map
OS Sectors

OD Average GCL + IPL Thickness: 80
OS Average GCL + IPL Thickness: 90

OD Minimum GCL + IPL Thickness: 62
OS Minimum GCL + IPL Thickness: 88

OD Horizontal B-Scan
OS Horizontal B-Scan
ONH and RNFL OU Analysis: Optic Disc Cube 200x200

**OD**

- Average RNFL Thickness: 86 μm
- RNFL Symmetry: 42%
- Rim Area: 1.05 mm²
- Disc Area: 3.69 mm²
- Average CD Ratio: 0.65
- Vertical CD Ratio: 0.79
- Cup Volume: 1.504 mm³

**OS**

- Average RNFL Thickness: 100 μm
- RNFL Symmetry: 42%
- Rim Area: 1.58 mm²
- Disc Area: 1.63 mm²
- Average CD Ratio: 0.20
- Vertical CD Ratio: 0.23
- Cup Volume: 0.013 mm³

**RNFL Deviation Map**

- Disc Center (0.06, 0.06) mm

**RNFL Thickness**

- Extracted Horizontal Tomogram
- Extracted Vertical Tomogram
- RNFL Circular Tomogram

**Neuro-retinal Rim Thickness**
CENTRAL 24-2 THRESHOLD TEST

FIXATION MONITOR: CAGE/BLIND SPOT
FIXATION TARGET: CENTRAL
FIXATION LOSSES: 0/16
FALSE POS ERRORS: 0%
FALSE NEG ERRORS: 2%
TEST DURATION: 00:153

STIMULUS: III, WHITE
BACKGROUND: 31.5 HSB
STRATEGY: GITA-STANDARD
PUPIL DIAMETER: 6.1 MM
VISUAL ACUITY:
RX: +1.00 DS
DC X
DATE: 05-08-2015
TIME: 2:17 PM
AGE: 33

TOTAL DEVIATION

PATTERN DEVIATION

OPHTHALMIC CONSULTANTS OF CONNECTICUT
1375 KING'S HIGHWAY
FAIRFIELD, CT 06430
(203) 366-8800
The Case of the Missing Exam

MRI report:

Clinical History: Optic atrophy and visual field loss of right eye.

Technique: An MRI of the orbits was performed. The study consisted dedicated axial T1-weighted, coronal T2 fat-saturated, and axial and coronal postgadolinium fat-saturated T1-weighted scans of the orbits. Additional survey images of the brain were obtained using sagittal and axial FLAIR, and axial diffusion-weighted, and postgadolinium T1-weighted images to evaluate for residual defect. Contrast images were obtained with 10 cc Gadavist.

Findings: The optic nerves and chiasm are normal in caliber and in signal intensity and there is no abnormal enhancement of these structures. Retrobulbar orbital fat is preserved. Extraocular muscles appear normal. The cavernous sinuses and paracavernous regions appear normal.

The ventricles and sulci are within normal limits in size and in configuration. There are a few punctate white matter hyperintensities on the FLAIR series within the cerebral white matter bilaterally and there is a small focus of abnormal hyperintense signal in the genu of the corpus callosum.

No mass is identified. No acute hematomas are present. There is no restricted diffusion to suggest an acute infarction. The brainstem and cerebellum appear normal.

There are normal vascular flow-voids.

The paranasal sinuses and mastoid air cells, as visualized, are clear.

Impression: Normal-appearing optic nerves and chiasm. A few white matter hyperintensities and a focus of abnormal signal intensity in the corpus callosum are nonspecific. Consider small foci of demyelination, gliosis, microvascular ischemia. The involvement of the corpus callosum would be somewhat unusual for microvascular ischemia.
Case 1: Glaucoma Suspect - inconsistent baseline test results

- **Banitt et al.** Progressive Loss of Retinal Ganglion Cell Function Precedes Structural Loss by Several Years in Glaucoma Suspects. *IOVS*, March 2013, Vol. 54, No. 3 (From the Bascom Palmer Eye Institute, supported by Grant National Institutes Health–National Eye Institute (NIH-NEI), NIH Center Grant, and Research to Prevent Blindness)
Case 1: Glaucoma Suspect

Visual fields are full, HRT normal
Case 1: Glaucoma Suspect

Patient has Kruckenbg spindles and IOP in 20’s, therefore, is a glaucoma suspect.

PERG shows borderline results. This defines a higher level of risk over time and the patient should be followed on a more intensive basis.
Case 2: Diabetic Retinopathy – subclinical management required for patient compliance

<table>
<thead>
<tr>
<th>Patient Work-Up</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Gender</td>
<td>Male</td>
</tr>
<tr>
<td>Age</td>
<td>62</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>White</td>
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<tr>
<td>Complaints/Symptoms</td>
<td>Blurred vision, flashes of light x 30 seconds</td>
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<tr>
<td>Personal History</td>
<td>Diabetes Mellitus II (Dx 2001), Hypertension, No allergies</td>
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<td>Family History</td>
<td>Grandmother: Diabetes Mellitus II</td>
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<td>Height / Weight</td>
<td>6f / 242Lb</td>
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<td>BS (6 month avg.)</td>
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<tr>
<td>BP</td>
<td>123/84</td>
</tr>
<tr>
<td>Pulse</td>
<td>101</td>
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<tr>
<td>IOP (mmHg) OD</td>
<td>16</td>
</tr>
<tr>
<td>IOP (mmHg) OS</td>
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</tr>
<tr>
<td>Refraction OD</td>
<td>+1.75 -0.75 x 130 +2.50</td>
</tr>
<tr>
<td>Refraction OS</td>
<td>+1.00</td>
</tr>
<tr>
<td>VCVA OD</td>
<td>20/50</td>
</tr>
<tr>
<td>VCVA OS</td>
<td>20/60</td>
</tr>
<tr>
<td>Preliminary Diagnosis</td>
<td>Diabetes Mellitus II, Hypertension, Diabetic Retinopathy</td>
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Case 2: Diabetic Retinopathy
Fundus OD  Fundus OS
Case 2: Diabetic Retinopathy

OCT OD

OCT OS
Case 2: Diabetic Retinopathy

OD VF OS
Case 2: Diabetic Retinopathy – ERG
Case 3: ERM – subclinical management for appropriate treatment

<table>
<thead>
<tr>
<th>Patient Work-Up</th>
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<tbody>
<tr>
<td>Gender</td>
<td>Female</td>
</tr>
<tr>
<td>Age</td>
<td>60</td>
</tr>
<tr>
<td>Ethnicity</td>
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<tr>
<td>Complaints/Symptoms</td>
<td>Floaters</td>
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<td>Personal History</td>
<td>Hypertension</td>
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<tr>
<td>Family History</td>
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</tr>
<tr>
<td>Height / Weight</td>
<td>5f / 175Lb</td>
</tr>
<tr>
<td>BP</td>
<td>174/91</td>
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<tr>
<td>Pulse</td>
<td>68</td>
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<td>IOP (mmHg) OD</td>
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</tr>
<tr>
<td>IOP (mmHg) OS</td>
<td>16</td>
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<tr>
<td>Refraction OD</td>
<td>+2.00 -1.00 x 155 +2.75</td>
</tr>
<tr>
<td>Refraction OS</td>
<td>+2.25 -2.00 x 180 +2.75</td>
</tr>
<tr>
<td>VCVA OD</td>
<td>20/25</td>
</tr>
<tr>
<td>VCVA OS</td>
<td>20/30</td>
</tr>
<tr>
<td>Preliminary Diagnosis</td>
<td>Hypertension, Cataract, Epiretinal Membrane (ERM)</td>
</tr>
</tbody>
</table>
Case 3: ERM

OCT OD

OCT OS
Case 3: ERM

VF OD

VF OS
Case 3: ERM - ERG
When used judiciously to improve visual function, medically necessary

Where Medical Necessity and Coverage Meet

VEP CPT 95930
• For subclinical optic nerve concerns (beneath the surface of clinical detection)
  • 377 (optic nerve/pathway disorders)
  • 368 (questionable vision)
• Systemic or Traumatic manifestations that affect vision
  • 368 (visual disturbances) or other signs and symptoms or concerns from
    • Neurological, TBI, Infectious, Infiltrative, Degenerative,

ERG CPT 92275
• For subclinical retinal concerns (beneath the surface of clinical detection)
  • 365.0X Glaucoma suspects
  • 377 (optic nerve/pathway disorders)
  • 365.1X and greater, confirmed glaucoma (mild to moderate stage)
  • 362 Retina (DR), Macula (AMD) and toxicity concerns
Can I use both tests on the same day?

• Both tests seen used to locate dysfunction – is it retinal (ERG) or retrobulbar (VEP) - optic nerve to visual cortex?

• Always requires documentation of medical necessity and impact on care

• May be performed same day as other tests, NO Correct Coding Initiatives

• Select the most appropriate ICD for the chief reason for the test – different reasons for different tests
Visual Electrophysiology is accepted as an additional testing method when more in-depth measures of visual function are required for diagnosis.

Clinical Considerations for Visual Electrophysiology

Visual Electrophysiology is accepted as an additional, alternative testing method when more in-depth measures of visual function are required for diagnosis, treatment, and management of disorders that affect vision.

- Subclinical disease (below the surface of clinical detection)
- Equivocal due to inconsistent clinical findings (e.g., unreliable) or patient testing limitations
- Locate area of dysfunction
- Progressive disease management

This is not an instruction of whom to use VEP and ERG or a guarantee of reimbursement. Coverage guidelines vary by payer policy. Testing frequency varies by patient medical need, disease type, severity and/or age. Always document medical rationale for testing and the impact on patient care on an Interpretation and Report in the chart. See below key for policy mapping and evidence for use.

Key with Hypertext:
A. Bolded codes refer to National VIP LCD 14072 (12/27/15)
B. ** codes refer to Aetna VIP policy 0183 (4/16/15)
C. *** Asterisked codes refer to Aetna ERG policy 0854 (12/22/14)

Key with Hypertext:
1. American Board of Ophthalmology Board
2. National Board of Examiners in Optometry
3. AAO Preferred Practice Pattern Comprehensive Adult Eye Exam
4. AAO Basic Clinical Science Course - The Visual Field
5. AAO Care of the Patient with Visual Impairment
6. CCGS 2012 Phys Port II Medicare Claims Data
7. PCCY (International Society of Clinical Electrophysiology for Vision)

Description ICD-9-CM ICD-10-CM VEP ERG

A. Asymptomatic Vision Disorders: eg. for subclinical disorders, below the surface of clinical detection

- Glaucoma
  - Open-Angle Glaucoma - 635.0
  - Primary Open-Angle Glaucoma - 365.00-365.08

- Glaucoma
  - Open-Angle Glaucoma - 635.10-635.19

- Diabetes mellitus with ophthalmic complications/diabetic retinopathy
  - Diabetic Retinopathy - 249.25-250.01-250.02

- Disorders of choroid and retinal

- Cataract

B. Optic Nerve & Visual Pathway - to objectively measure function vs. structure

- Optic neuropathy

- Disorders of optic nerve

- Other disorders of optic (2nd) nerve and visual pathways

- Glaucomatous optic atrophy

- Other disorders of optic nerve

- Disorders of visual pathways

- Amblyopia

- Psychophysical visual disturbances

- Other subjective visual disturbances

- Transient vision loss

- Subacute visual loss

- Visual Field Defects

- Impaired contrast sensitivity/other visual disturbances

- Conversion Disorders

C. Visual Disturbances - eg. patients who cannot produce a reliable field or acuity test (elderly, add, handicapped) or incomplete findings

- Amblyopia

- Psychophysical visual disturbances

- Subjective visual disturbances

- Transient vision loss

- Subacute visual loss

- Visual Field Defects

- Impaired contrast sensitivity/other visual disturbances

D. Trauma - eg. for opemagement (refer) to and from other specialists for care of patient care

- Other transient cerebral ischemic attacks and related syndromes

- Injury to optic nerve and visual pathways

- Intracranial injury, TBI

- Other trauma

- Trauma - eg. nutrition and drug toxities, there is no use test for placement of the recommendations

- Other long term (current) drug therapy

- Inflammatory and toxic neuropathy

- Neurological (Degenerative) - eg. long accepted for optic nerve and retinal degenerations including signs and symptoms

- Multiple sclerosis

- Neuromyelitis optica (Devic)-Other specified acute disseminated encephalomyelitis

- Hamipapiea and hemiparesis

- Dizziness and giddiness

- Infantile Cerebral Palsy

- Benign intracranial hypertension

- Cerebral palsy

E. Infections - eg. inflammation in the visual pathway from bacterial, viral, fungal

- Meningitis - subarachnoid hemorrhage

- Meningitis -.getCurrentUser().getMeningitis

- Lyme Disease

F. Inflammatory - eg. neoplasms, primary or metastatic, retinoblastoma, pituitary tumors

- Malignant neoplasm of cerebral meninges/epithelial meningitis

- Malignant neoplasm of optic nerve

- Benign neoplasm of brain, supratentorial-other specified parts of (CNS)

- Neoplasms of uncertain behavior of pituitary gland-adenoma gland

- Abnormal Visual Evoked Potential (VEP)

- Abnormal Electroencephalogram (ERG)

- Abnormal pupillary function, unspecified

- Abnormal vertical gazes, unspecified

- Abnormal visual evoked potentials, unspecified

- Abnormal visual fields, unspecified

- Abnormal visual evoked potentials, unspecified

- Abnormal visual evoked potentials, unspecified
A SUMMARY FOR USE AND DOCUMENTATION OF ELECTROPHYSIOLOGY

Electrophysiology, (VEP and ERG) is accepted as additional testing when standard tests, patient limitations and/or subclinical disease processes require objective, functional data to aid in patient management of diseases that cause vision loss. The example below provides essential components to help determine and document need for the test(s). While completion of all sections on an Interpretation and Report (I&R) support compliant documentation, the arrows identify important areas that document medical need for the test(s). Blank examples for each test are also provided for your review.

REMEMBER:
1. Test selection is always determined by physician based on the patient’s documented medical need.
2. All diagnostic tests require a SEPARATE I&R. This also applies to VEP and ERG.

### Electrophysiology Interpretation and Report

(Use separate sheet for each test)

<table>
<thead>
<tr>
<th>Date:</th>
<th>Ordering doctor:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Patient Name:

<table>
<thead>
<tr>
<th>MEDICAL NECESSITY FOR FUNCTIONAL EVALUATION TO DIAGNOSE AND TREAT CONDITION THAT IS:</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Subclinical - absent of signs/symptoms or differential dx/systemic manifestation</td>
</tr>
<tr>
<td>☐ Location need site of functional abnormality</td>
</tr>
<tr>
<td>☐ Equivocal-inconclusive based on evidence or patient/test capabilities</td>
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</tbody>
</table>

#### TEST SELECTION/OFFER FOR:

| VEP CPT 95980 for Vision Function | ERG CPT 92275 for Retinal Function |

<table>
<thead>
<tr>
<th>PATIENT NEED FOR VEP DUE TO:</th>
<th>PATIENT NEED FOR ERG DUE TO:</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Change in condition</td>
<td>☐ Change in condition</td>
</tr>
<tr>
<td>☐ Re-evaluation of condition</td>
<td>☐ Re-evaluation of condition</td>
</tr>
<tr>
<td>☐ Abnormal function test</td>
<td>☐ Abnormal function test</td>
</tr>
<tr>
<td>☐ Optic Nerve or Visual pathway disorder</td>
<td>☐ Optic Nerve or Visual pathway disorder</td>
</tr>
<tr>
<td>☐ Unexplained loss of vision</td>
<td>☐ Unexplained loss of vision</td>
</tr>
<tr>
<td>☐ Visual disturbance</td>
<td>☐ Visual disturbance</td>
</tr>
<tr>
<td>☐ Toxicity and nutritional eye disease</td>
<td>☐ Toxicity and nutritional eye disease</td>
</tr>
<tr>
<td>☐ Suspected intracranial lesion</td>
<td>☐ Suspected intracranial lesion</td>
</tr>
<tr>
<td>☐ Infant/newborn with questionable vision</td>
<td>☐ Retinal Disorders</td>
</tr>
<tr>
<td>☐ Injury to the eye or orbit</td>
<td>☐ Glaucoma</td>
</tr>
<tr>
<td>☐ Other patient limitations</td>
<td>☐ Other</td>
</tr>
<tr>
<td>☐ Malingerage</td>
<td>☐ Other</td>
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</tbody>
</table>

**Patient unable to perform visual field**

### TECHNICIAN’S NOTES: (If any, may be on test result)

<table>
<thead>
<tr>
<th>Electrodes in standard configuration</th>
<th>Electrode placement:</th>
</tr>
</thead>
<tbody>
<tr>
<td>[ ] Normal</td>
<td>☐ Reliable</td>
</tr>
<tr>
<td>☐ Abnormal</td>
<td>☐ Unreliable</td>
</tr>
<tr>
<td>Notes:</td>
<td>see FAQ reference # 15</td>
</tr>
</tbody>
</table>

### RESULTS:

| ☐ Normal                          | ☐ Abnormal                        |
| ☐ Reliable                        | ☐ Unreliable                       |

### IMPRESSION:

| ☐ Worsen with follow up or frequency of visit | ☐ No change |
| ☐ Referral Required - confirms ocular condition w/ referral for co-management | ☐ No change |
| ☐ Additional testing - e.g. ERG after abnormal VEP to localize dysfunction | ☐ No change |
| ☐ Counseling - discussion of risk for patient management and compliance | ☐ No change |
| ☐ Treatment - validates existing treatment plan or need to change due to disease progression | ☐ No change |

### IMPACT ON TREATMENT AND COORDINATION OF CARE:

| ☐ Monitor for progression of functional loss for treatment | ☐ Change follow up from 1 year to 6 months |
| ☐ Monitor for progression of functional loss for treatment | ☐ Change follow up from 1 year to 6 months |

### SIGNATURE:

[Signature]

[Date: 11/05/2014]