

Gary G. Berger DO Memorial Lecture

2022 Annual Meeting of the Brain Injury Association of
Kansas and Greater Kansas City

Rehabilitation of Patients with Homonymous Hemianopia from Stroke

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Financial Disclosures

- Nothing to disclose
- The opinions expressed here are mine alone, and do not necessarily represent the opinions of the University of Kansas School of Medicine, its departments and faculty, nor The University of Kansas Health System.

So what are we here about?

We're here to honor the memory of
Gary G. Berger DO.

Who is Gary G. Berger DO?

A brief history of Gary—A family man from Philly

His family—wife Randi; children Joel, Melissa, and Hayley

His education--

- B.A. Temple University 1979;
- D.O. Philadelphia College of Osteopathic Medicine 1983;
- Residency PM&R, Temple University Hospital 1987;
- Certification, American Board of Physical Medicine and Rehabilitation 1988

His career--

Gary initially practiced PM&R in the Philadelphia and New Jersey area. In 1992 Gary moved to this area. He practiced primarily at Menorah Medical Center/Health Midwest (later Physician Services of HCA), but also at The Rehabilitation Institute of Kansas City (later Ability KC), Park Lane Medical Center, and Baptist Medical Center.

At Menorah, Gary served as:

- Medical Director, Rehab Services
- Menorah Medical Staff: President, Secretary, Treasurer, Immediate Past President
- Utilization Review Committee
- Hospital Peer Review Committee

Gary was our beloved colleague, friend, and mentor until his untimely passing June 25, 2014

Who am I? Well, I'm a local guy--

- Grew up here, St Peter's grade school, Rockhurst high school
- Education—
 - B.A., Rice Univ 1977;
 - M.S., Biomed Engineering, Marquette Univ 1979;
 - J.D. with distinction, UMKC School of Law 1982;
 - M.D., Univ Kansas School of Medicine 1990; Internship/residency UMKC School of Medicine 1994;
 - Fellowship, neuro-ophthalmology, Bradley K. Farris MD, Dean McGee Eye Institute (David W. Parke II MD, Chairman), Univ Oklahoma Health Sciences 1996
 - Certification, American Board of Ophthalmology 1996; maintenance of certification 2006, 2016
- Practice—Law and Medicine
 - Trial Law Practice, Shughart Thomson & Kilroy, 1981-1990
 - Private ophthalmology practice, 1994-96 (*when I met Gary!*)
 - Neuro-ophthalmology, University of Kansas School of Medicine, Departments of Ophthalmology and Neurology, 1997 to present
- My family--
 - Wife, Lissa; Kids, Leah, Jon (Brenna), Andrew (Alicia), Mary Pat (Eric), and Sarah (Dominic); Grandkids, Benjamin, Paige, Aiden, and Charlie
 - Father, C. Keith Whittaker MD
 - Grandfather, Charles E. Whittaker Esq
 - My cousin, some of you may remember—
Amy Thompson



This is my professional family--

Bradley K. Farris MD

NeuroOphthalmology, Dean McGee Eye Institute, University of Oklahoma Health Sciences

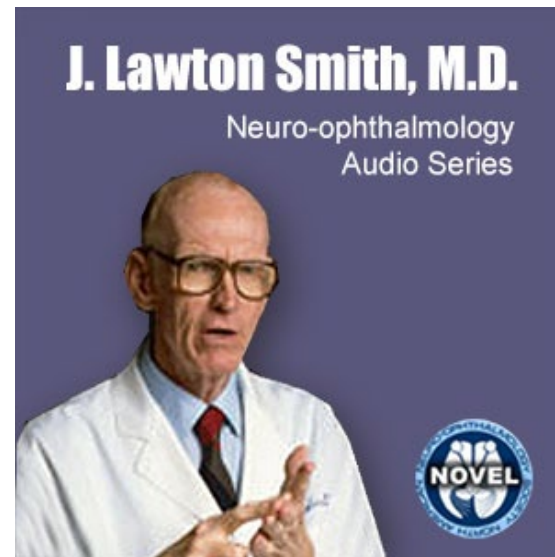
- Trained by Dr. J. Lawton Smith at Bascom Palmer Eye Institute



J. Lawton Smith MD

NeuroOphthalmology, Bascom Palmer Eye Institute, University of Miami

- Trained by Dr. David Cogan at Massachusetts Eye and Ear Infirmary



So let's get started--

People with stroke (20% to 57% of them) lose part of their visual field, often one complete half—a homonymous hemianopia. These are people we see and care for.

Now a question--Does rehab for our patients with homonymous field defects make a difference?

Obviously, we all think it helps because patients tell us it does, and we see them improve.

But what is the evidence our work does what we say it does?

That's the goal today:

1. Review the therapies for hemianopia from stroke. (And yes, I'm limiting this to only hemianopia from stroke. You'll understand why later.)
2. Ask the question, "What is the **evidence** that what we do really works?"

Pertinent “Gary-ism” here to guide us:
"Help the patient! Everything else comes second."

Agenda

- Review anatomy and corresponding visual field defects with clinical examples
- Review current therapy options
- Consider other new therapies and evidence
- Review evidence for effectiveness

Caution: this is not intended as an exhaustive literature review, but instead a summary of the evidence from the literature.

Anatomy Review

- Damage to tissue along the visual pathways causes vision loss in predictable patterns corresponding to the anatomic location of the damage.
- Knowing the location of tissue damage can predict the type and pattern of visual loss and other deficits.
- These rules hold true for damage from any cause.

Anatomy Review

The Visual Pathway:

Retina and optic nerve

Optic chiasm

Optic tracts

Lateral geniculate nucleus

Optic radiations: temporal and parietal

Occipital (visual) cortex

Testing for visual field defects can--

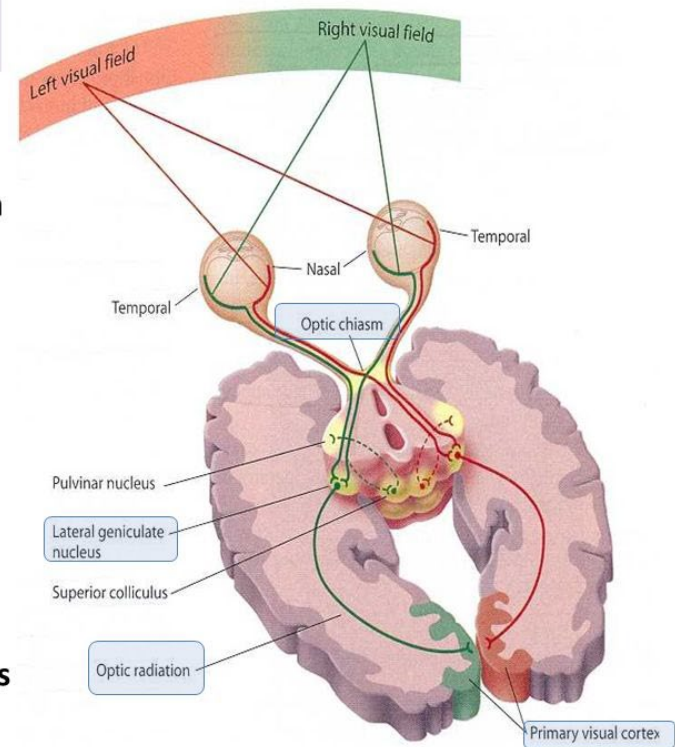
--localize the damage,

--predict or guide neuroimaging, and

--guide therapy and rehabilitation

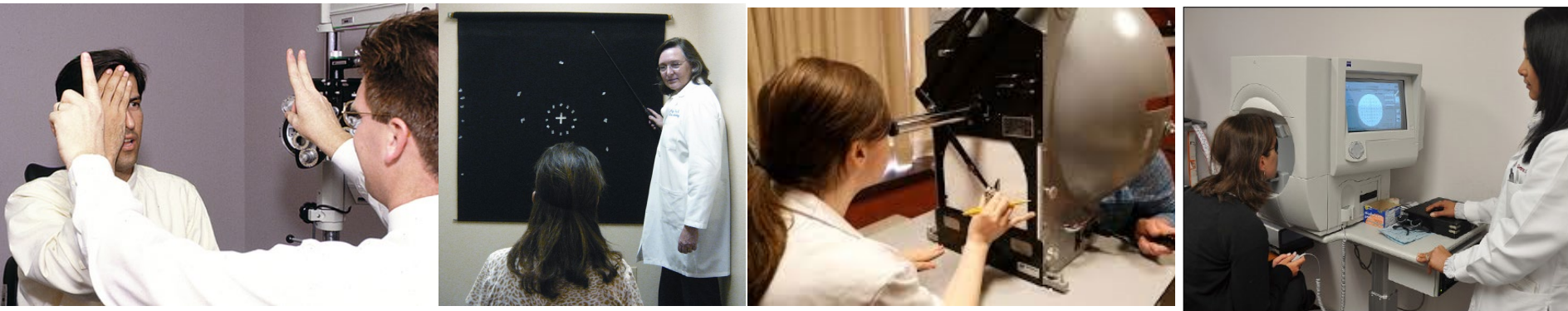
Visual Pathway

1. **Cones**
2. **Bipolar neurons**
3. **Ganglion cell's axon forms the optic nerve**
4. **Optic nerve to the Optic Chiasm**
5. **Optic tract**
6. **Lateral geniculate nuclei of the thalamus**
7. **Optic Radiations**
8. **Primary visual areas of the occipital lobes**

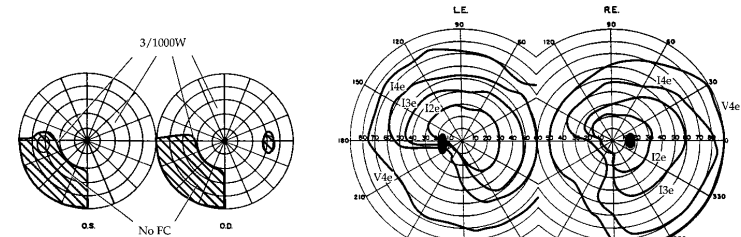
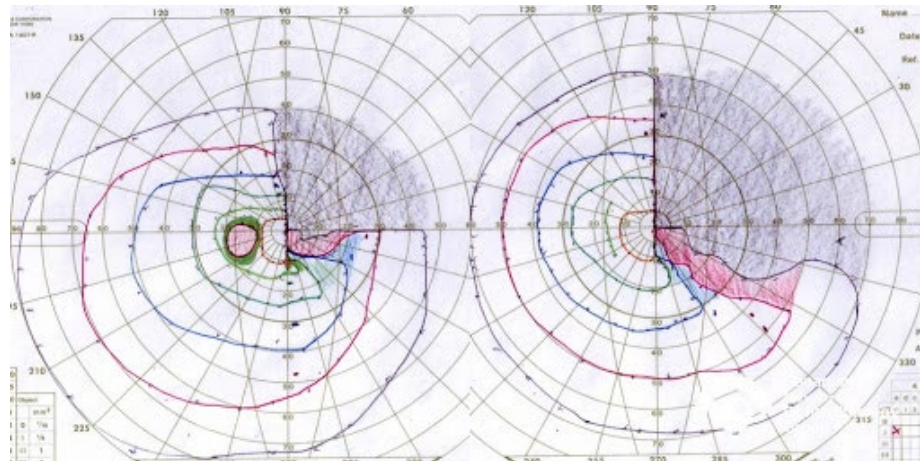
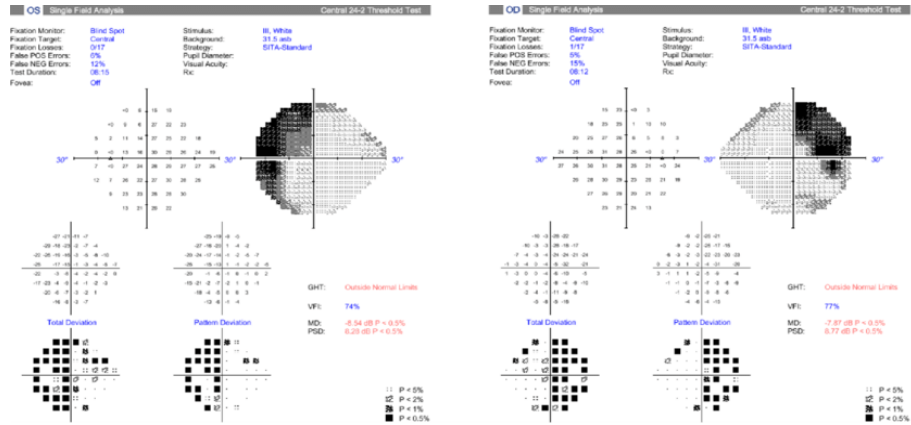


Visual Loss and Stroke: testing the visual field

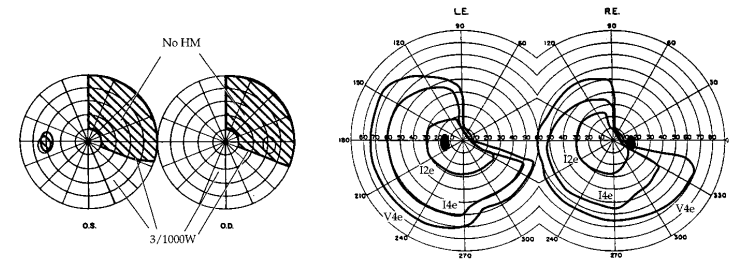
- Types of visual field tests:
 - Confrontation visual field testing
 - Kinetic perimetry—tangent screen, Goldmann, automated perimetry
 - Static perimetry—automated perimetry (Humphrey, Octopus, Zeiss, and many others, including apps like MRF Neural Lite for iPad)



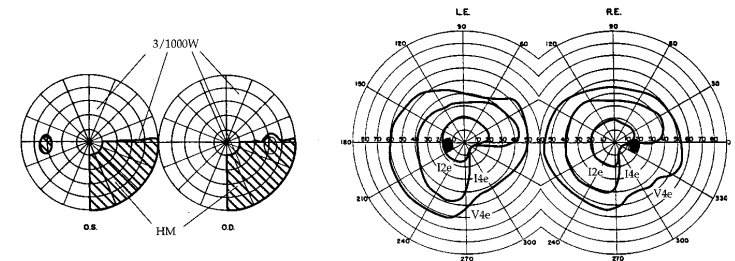
Testing the visual field



Patient 2



Patient 3



Types of visual field defects

Monocular field defects (present in one eye only)

--Monocular field defects must localize anterior to the chiasm, the optic nerve or retina (one exception).

--Field defects may cross vertical midline

Heteronymous field defects (present in both eyes but with different laterality)

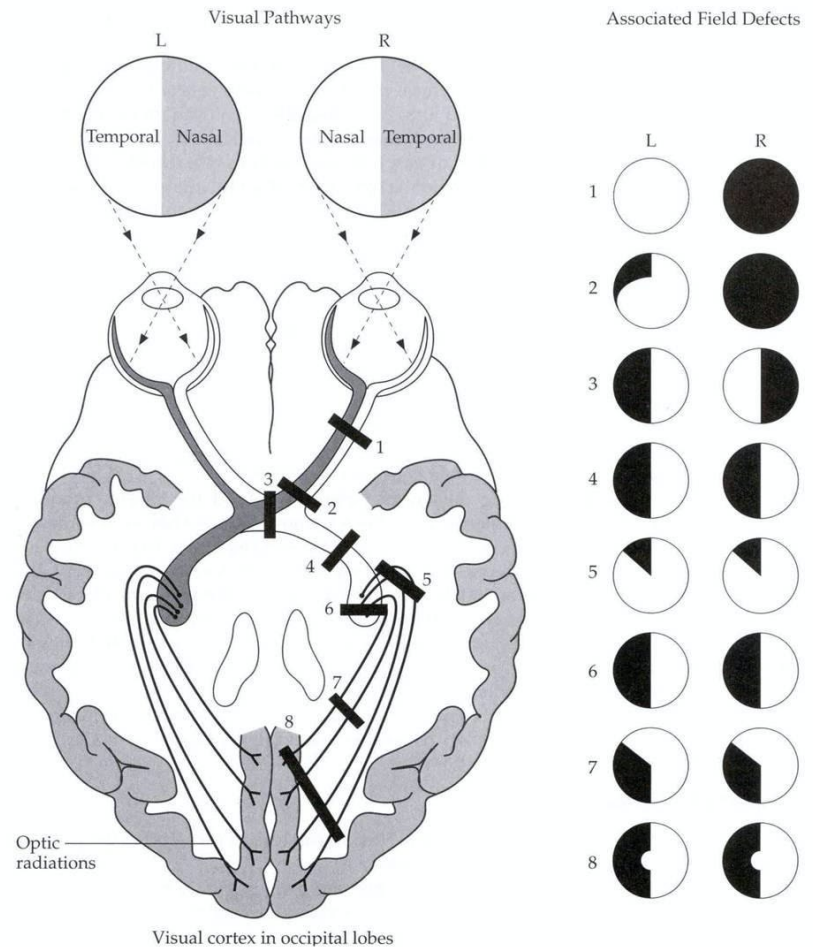
--Heteronymous field defects must localize to the chiasm, typically bitemporal

--Field defects do not cross vertical midline.

Homonymous field defects (present in both eyes with same laterality)

--Homonymous field defects must localize posterior to the chiasm.

--Field defects do not cross vertical midline



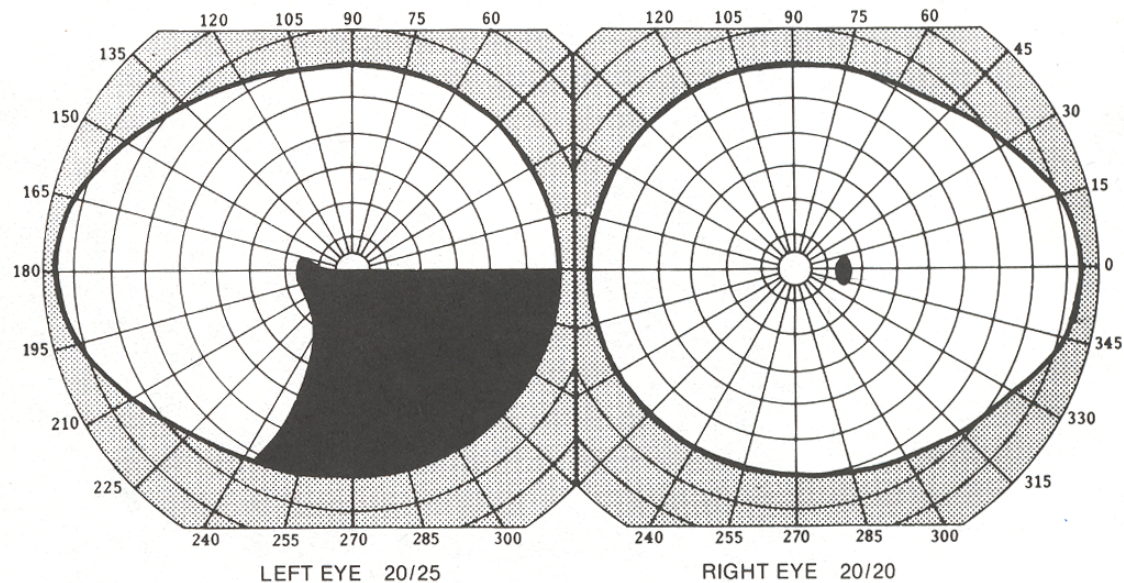
Clinical examples

45 yo male w/ sudden loss of vision left eye on awakening this morning.
History of increased intraocular pressure, HTN, elevated cholesterol, and obstructive sleep apnea.

Where's the field defect? Monocular, left eye only.

Does it cross the vertical midline? Yes, so it has to be in the retina or optic nerve.

Diagnosis: Inferior arcuate/altitudinal defect OS, suspect non-arteritic ischemic optic neuropathy (NAION), a stroke of the optic nerve.



Clinical examples

72 yo male with history of HTN, DM and elevated cholesterol reports acute onset of decreased vision in the left eye.

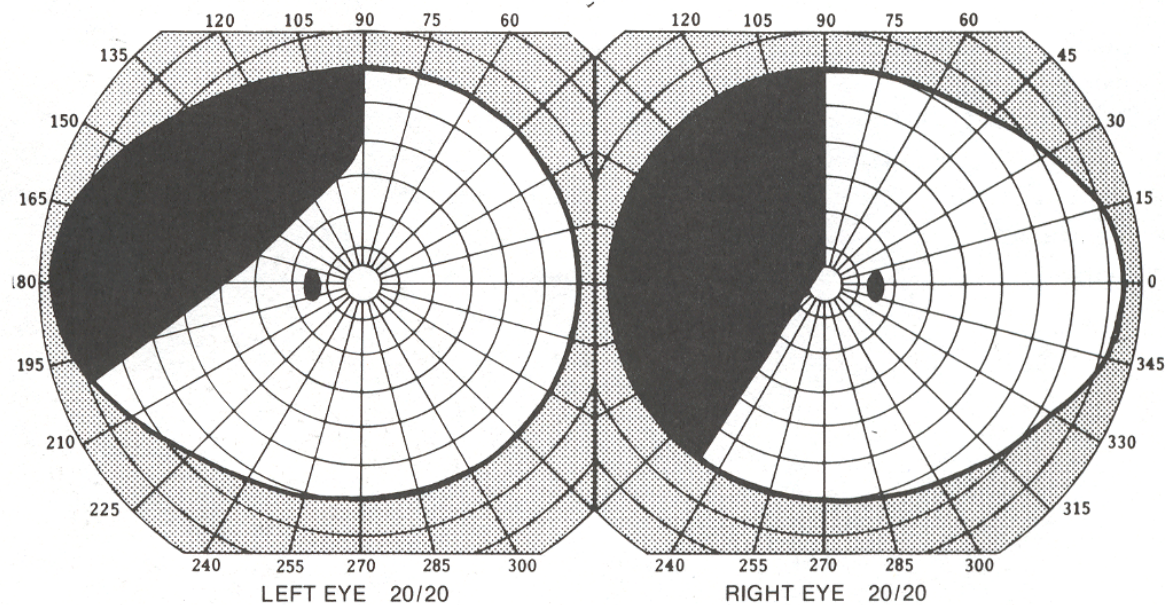
Where's the field defect? In both eyes.

Does it cross the vertical midline? No.

Is the field defect homonymous? Yes, but it's not congruous.

Diagnosis: left homonymous hemianopia, incongruous and denser superiorly. Suspect right temporal lobe infarct.

Clinical concern: visual agnosia, inability to recognize familiar objects despite normal acuity.



Clinical examples

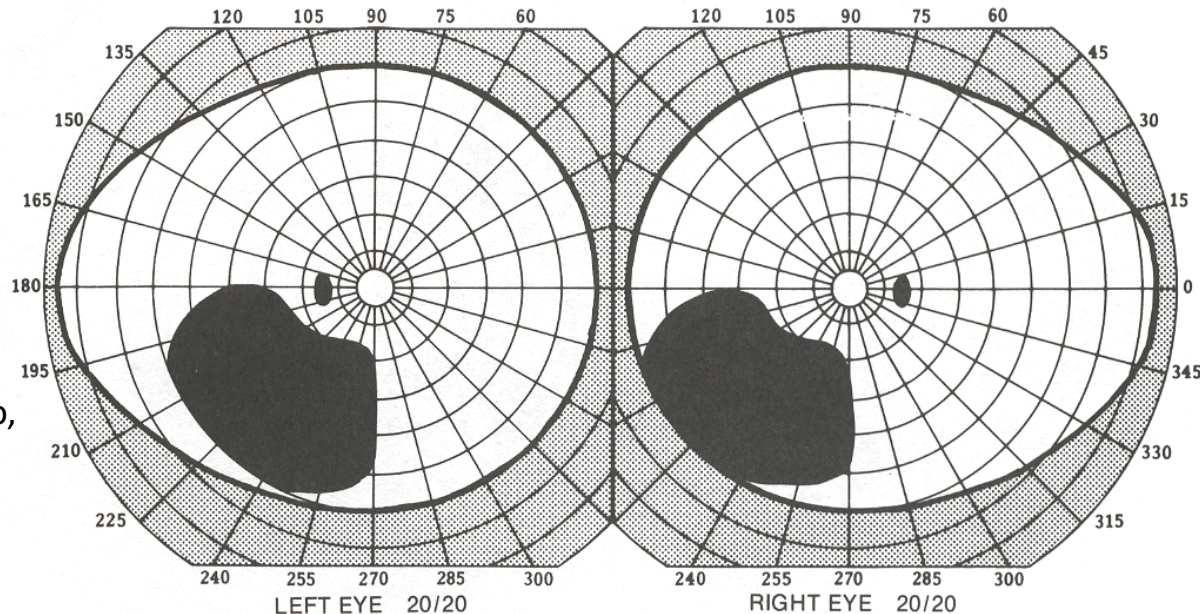
81 yo male with instability, and visual neglect. History of DM, elevated cholesterol, and atrial fibrillation.

Where's the field defect? In both eyes.

Does it cross the vertical midline? No, it's homonymous on left.

Diagnosis: left homonymous hemianopia, denser inferiorly, and seems congruous. Suspect right parietal lobe with neglect, possible right occipital due to congruity.

Clinical concern: spatial processing deficits, visual neglect



Clinical examples

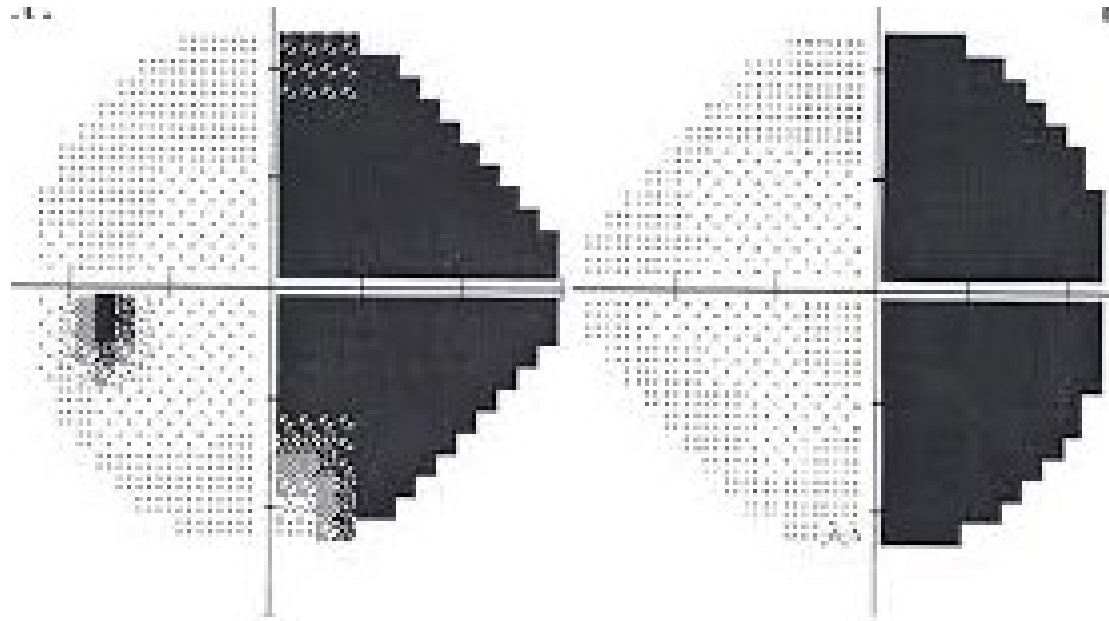
71 yo male with poorly controlled HTN, AFib with drop attacks, and DM II reports decreased vision to right 5 days prior.

Where's the field defect? In both eyes.

Does it cross the vertical midline? No, it's right homonymous.

Diagnosis: Right homonymous hemianopia. Suspect left occipital CVA.

Clinical concern: source either cardiac arrhythmia or posterior circulation.



Clinical examples

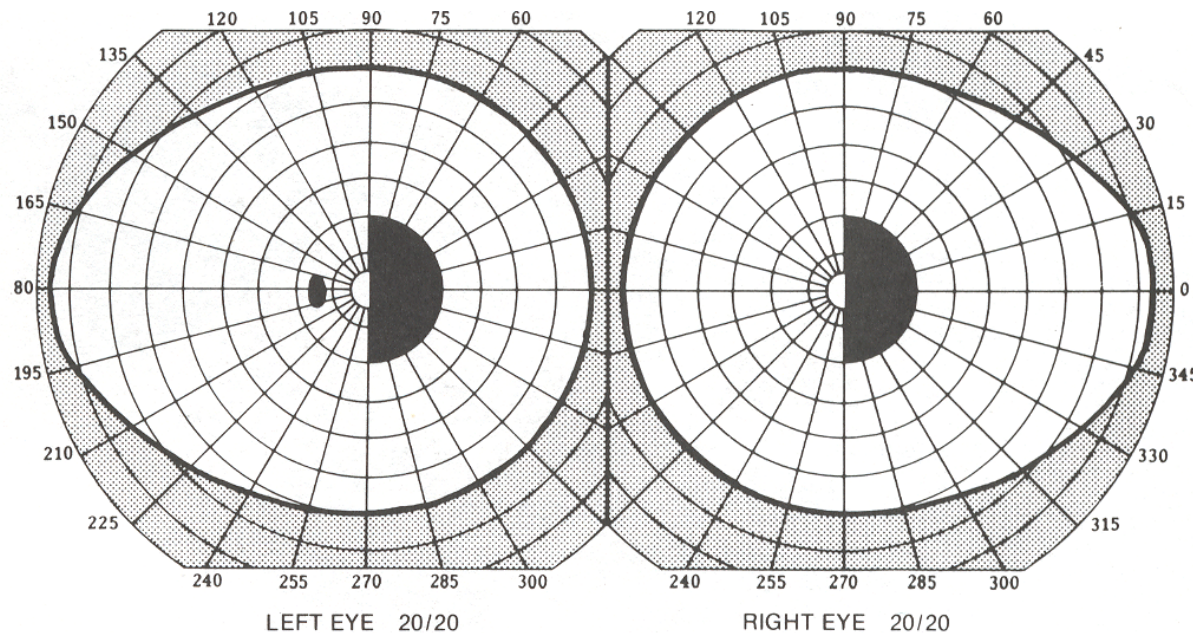
54 yo female complains of missing words when reading; PMHx: HTN, elevated cholesterol; smoker 1 ppd for 30 years.

Where's the field defect? In both eyes.

Does it cross the vertical midline? No, it's right homonymous.

Diagnosis: right central homonymous hemianopia, suspect left occipital tip CVA.

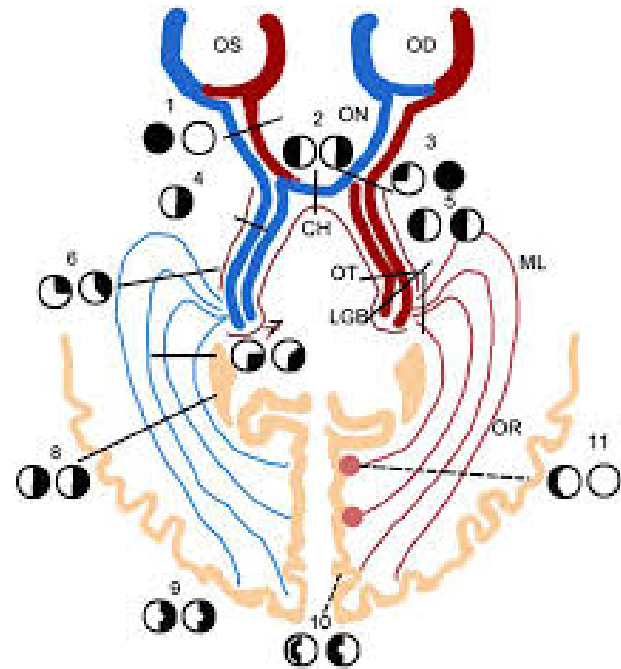
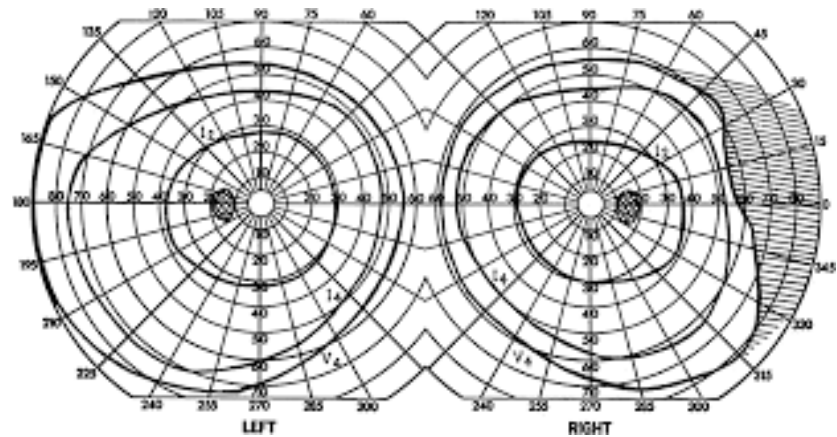
Clinical concern: left occipital tip CVA vs mass, possible metastatic disease.



Unusual field defect

Temporal crescent syndrome:

Monocular visual field defect caused by lesion in contralateral anterior mesial occipital lobe—exception to rule that retrochiasmal lesions must cause homonymous field defects.



Impact of hemianopia

- Social/emotional impact due to loss of independence, anxiety in new places, loss of ability to drive
- Impaired visual search and awareness leading to collisions, falls, and further injury
- Reading, accommodative and oculomotor/scanning difficulties required for modern life/self-sufficiency
- Comorbidities from damage beyond visual cortex
 - Visual neglect
 - Vestibular and sensory-motor deficits
 - Cognitive, language and memory deficits

Damage to other cortical areas can cause higher order visual processing deficits

- Parietal—neglect/spatial processing deficits
- Temporal—object recognition deficits, visual agnosia
 - Inability to recognize familiar objects in setting of preserved visual function/normal acuity)
- Frontal—defective visual search, tracking, attention
 - Planning deficits, Decision making, Visual search and tracking

Our discussion will be limited to visual field loss without higher order deficits

Strategies for therapy/treatment

1. Spontaneous recovery

- Partial or complete recovery possible, typically within first 2-6 months
- Wide variation in spontaneous recovery: 7%-85% of stroke patients

2. Compensatory

- Use of remaining field through scanning/training

3. Optical

- Prism to displace/expand field from blind to sighted field

4. Restoration

- Reduce field loss through repetitive training or regrowth
- Other new technologies

Interventions for visual field defects in people with stroke, Cochrane Database Syst Rev, 2019 May; 2019 (5)

Howard C, Rowe F, *Adaptation to poststroke visual field loss: A systematic review, Brain and Behavior, 2018 June;8:e01041, <https://doi.org/10.1002/brb3.1041>*

Hemianopia rehab strategies

Spontaneous recovery

- Dependent on location, cause and extent of damage
- Wide variability reported in literature; data largely retrospective
- Spontaneous recovery is early and likely complete by 2 months

Pambakian A, Currie J, Kennard C. *Rehabilitation strategies for patients with homonymous visual field defects*. J Neuroophthalmol 2005;25:136–42.

Goodwin D. *Homonymous hemianopia: challenges and solutions*. Clin Ophthalmol 2014;8:1919–27.

Zhang X, Kedar S, Lynn MJ, et al. *Homonymous hemianopias: clinical-anatomic correlations in 904 cases*. Neurology 2006;66:906–10.

Zhang X, Kedar S, Lynn MJ, et al. *Natural history of homonymous hemianopia*. Neurology 2006;66:901–5.

Hemianopia rehab strategies

Compensatory

- Increase sensory awareness through scanning/training
 - Encourage scanning to affected hemifield,
 - Improve reading/saccade accuracy,
 - Use strategic scene exploration
- Does scanning training work? Yes, it seems to help compensate for the missing field
 - improved search fields of hemianopic patients by 10 degrees
 - reduced the time to find objects by up to 50%
 - improved patients' sense of impairment

Zihl J. *Recovery of visual functions in patients with cerebral blindness: Effect of specific practice with saccadic localization.* Exp Brain Res 1981;44:159–69.

Kerkhoff G. *Neurovisual rehabilitation: recent developments and future directions.*

J Neurol Neurosurg Psychiatr 2000;68:691–706.

Hemianopia rehab strategies

Compensatory, cont'd

- Does scanning address reading deficits?
 - Right hemianopia: decreased fluency, inefficient saccades
 - Left hemianopia: decreased comprehension, impaired “carriage return”
- Conclusion: There is some evidence supporting compensatory scanning for patients with hemianopia

Pollock A, et al. *Interventions for visual field defects in people with stroke*, Cochrane Database Syst Rev. 2019 May; 2019 (5)

Schuett S, Heywood c, et al, *Rehabilitation of reading and visual exploration in visual field disorders: transfer or specificity?* Brain, 135(3): 912-921 March 2012.

Hemianopia rehab strategies

Optical

Spectacle-mounted prisms transpose portions of non-seeing field onto seeing field

- Partial or complete coverage
- One or both lenses
- Temporary or ground in prism
- Designs: Gottlieb, Chadwick, Peli



Hemianopia rehab strategies

Optical, cont'd

- Yoked (bilateral) prism effectiveness suggested by several studies, but
 - Criticized as potentially confusing or not beneficial, and potentially dangerous
- Monocular prism
 - Peli group: randomized, masked, crossover trial: about 50% patients found prism helpful but study criticized for subjective outcomes

Bowers A, Keeney K, Peli E. *Randomized Crossover Clinical Trial of Real and Sham Peripheral Prism Glasses for Hemianopia*, *JAMA Ophthalmol.* 2014; 132(2):214-222

Peli E, *Fitting Peripheral Prisms for Hemianopia*, NANOS Annual Meeting 2017.

<https://collections.lib.utah.edu/ark:/87278/s65t7fbv>

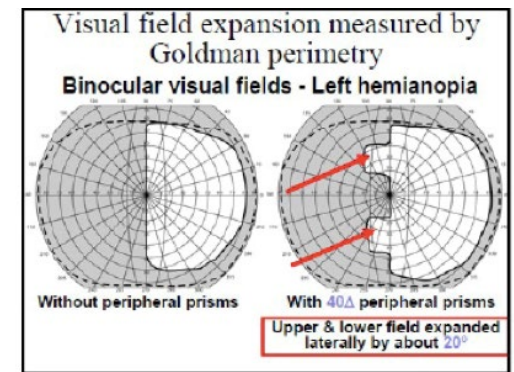


Illustration: Expansion of field of view with ML Peli Prism 40 PRD in 2 segments

Hemianopia rehab strategies

- **Restitutive/restorative**
 - Historically many attempts with various techniques
 - Recent review documented 23 different trials for restorative techniques, but only 5 for compensatory

State-of-the-Art Review

Section Editors: Valérie Blousse, MD
Steven Galetta, MD

Rehabilitation of Visual Loss: Where We Are and Where We Need to Be

Behzad Mansouri, MD, PhD, FRCPC, Marinya Roznik, BSc, Joseph F. Rizzo III, MD,
Sashank Prasad, MD

Journal of Neuro-Ophthalmology 2018;38:223–229

Hemianopia rehab strategies

Restorative

- Vision restoration therapy (VRT)--promising but not proven
 - One example, in 2004-5, *NovaVision*, a proprietary, computer-driven, home-based therapy for postchiasmal homonymous visual field loss
 - Repeated visual stimulation of transition zone, 1 hour sessions, 6 days/week, 6 months. Average 5 degree visual field gain
 - Criticisms:
 - Benefits not clinically meaningful (objective/subjective mismatch; no appreciable transfer to ADLs, small magnitude of visual field effect)
 - Extraneous factors/artifacts not controlled (eye movements/crossing, method of perimetry measurement)
 - Validity of home-based therapy (unsupervised, lack of controlled environment)
 - Ongoing dispute in literature as to effectiveness.

Frolov A, Feuerstein J, Subramanian P, *Homonymous Hemianopia and Vision Restoration Therapy*, Neurol Clin 35 (2017) 29–43.

Horton JC. *Disappointing results from nova vision's visual restoration therapy*. Br J Ophthalmol 2005;89:1–2.

Sabel BA, Kenkel S, Kasten E. *Vision restoration therapy*. Br J Ophthalmol 2005; 89:522–4.

Hemianopia rehab strategies

Restorative

- Vision restoration therapy—some interesting options
 - Noninvasive brain stimulation of occipital cortex (transcranial DC or AC current stimulation)
 - Magnetic field stimulation of optic pathway neurons

Gall C, Schmidt S, Schittkowski MP, Antal A, Ambrus GG, Paulus W, et al. *Alternating Current Stimulation for Vision Restoration after Optic Nerve Damage: A Randomized Clinical Trial*. (2016) PLoS ONE 11(6): e0156134.

B. Laha, B. Stafford, A. Huberman, *Regenerating Optic Pathways from the Eye to the Brain*, Science 2017 June 09, 356 (6342): 1031-1034.

Hemianopia rehab strategies

Restorative

"In sum, there are theoretic underpinnings, animal data, and human experience to suggest that there is a large amount of potential for plasticity in the visual system, despite the gestalt to the contrary. . . . Although multiple studies have showed [sic] statistically significant effects after various training protocols, controversy remains as to their validity and applicability in clinical practice. ***Neither VRT nor other researched methods of visual system training have led to standardized approaches to visual rehabilitation in patients with visual field defects, indicating that larger and more targeted studies are still needed to determine the best approach to the patient with a new visual field defect in clinical practice.***"

--Frolov A, Feuerstein J, Subramanian P, *Homonymous Hemianopia and Vision Restoration Therapy*, Neurol Clin 35 (2017) pp 41, *emphasis added*.

Effectiveness of interventions for homonymous hemianopia from 2019 Cochrane Review

Authors' conclusions in "plain language"

"There is a lack of evidence relating to the effect of interventions on our primary outcome of functional ability in activities of daily living.

There is limited low-quality evidence that compensatory scanning technique may be more beneficial than placebo or control at improving quality of life, but not other outcomes.

There is insufficient evidence to reach any generalised conclusions about the effect of restitutive interventions or substitutive interventions (prisms) as compared to placebo, control, or no treatment.

There is low-quality evidence that prisms may cause minor adverse events."

Pollock A, Hazelton C, Rowe FJ, Jonuscheit S, Kernohan A, Angilley J, Henderson CA, Langhorne P, Campbell P. *Interventions for visual field defects in people with stroke*. Cochrane Database of Systematic Reviews, 2019, Issue 5, Art. No.: CD008388. DOI: 10.1002/14651858.CD008388.pub3.

2019 Cochrane Review Findings

Summary of findings

- The quality of evidence summarised in this review is judged to be low to very low.
- Methodological quality of studies is, in general, poor or poorly reported, providing insufficient high-quality evidence on which to reach generalisable conclusions.
- Limited low-quality evidence suggests compensatory interventions may improve an important outcome (quality of life) in patients with visual field defects following stroke, but further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate. There is insufficient evidence to reach a conclusion about the impact of compensatory scanning training on functional activities of daily living, or other outcomes.
- There is insufficient evidence to reach generalised conclusions about the benefits of vision restoration therapy for patients with visual field defects after stroke.
- There is insufficient evidence to reach generalised conclusions about the benefits of prisms for patients with visual field defects after stroke; there is some low-quality evidence that prisms may cause adverse events.
- High-quality RCTs are needed to compare compensatory, restitutive, substitutive, and assessment interventions with placebo, control, no treatment, or usual care.

2019 Cochrane Review: Implications for practice

- There is limited low-quality evidence that compensatory scanning training may improve an important outcome (quality of life) in patients with visual field defects following stroke, but further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate. There is insufficient evidence to reach a conclusion about the impact of compensatory scanning training on other outcomes.
- There is insufficient evidence to reach generalised conclusions about the benefits of visual restitution therapy (restitutive intervention), prisms (substitutive intervention), or assessment or screening interventions for patients with visual field defects after stroke. Prisms may cause a range of adverse events, particularly headache.

2019 Cochrane Review: Implications for research

Randomized clinical trials (RCTs) are required to determine the effect of:

- compensatory scanning training compared to no treatment, placebo, or usual care;
- restitutive interventions compared to no treatment, control, or placebo;
- substitutive interventions compared to no treatment, control, or placebo;
- assessment or screening interventions compared to standard care.

Such RCTs must:

- have adequate power (i.e. with an appropriate power calculation undertaken based on existing trial evidence);
- have adequate allocation concealment, blinding of outcome assessor, and intention-to-treat analysis;
- clearly define trial participants, with particular care relating to the diagnosis and inclusion of patients with visual field defects or visual neglect, or both;
- consider the severity of the visual field defect and plan subgroup analyses, where appropriate;
- include measures of functional ability in activities of daily living;
- collect and report data relating to adverse events;
- report clear and usable data.

Pretty depressing, isn't it?

- The only evidence that what we do matters is "low quality" and offers scant support for scanning
- So now what? Should we all give up and go to law school? Learn to be plumbers? What would Gary do?
- I think he'd say, "There's a lot of smart people in here. Why can't we do something about this?"
- We have three medical schools here, multiple PM&R physicians, residents, therapists, and lots of patients.
- We come together for this meeting, so why can't we work together to do "high grade" research on this one problem we all see and treat?

Closing comments

- So that's where I'm going to leave this.
- We can fix the problem, and produce good data that means something.

Is there a better way to honor Gary?