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Continuing Education Course Approval Checklist

Title: Biomechanical Glaucoma Damage

Provider Name: USC Roski Eye Institute c/o Ms. Amy Atondo

☑Completed Application
Open to all Optometrists? ☑Yes ☐No
Maintain Record Agreement? ☑Yes ☐No

☑Correct Application Fee

☑Detailed Course Summary

☑Detailed Course Outline

☑PowerPoint and/or other Presentation Materials

☐Advertising (optional)

☑CV for EACH Course Instructor

☑License Verification for Each Course Instructor
Disciplinary History? ☐Yes ☑No

CALIFORNIA STATE BOARD OF OPTOMETRY

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CONTINUING EDUCATION COURSE APPROVAL APPLICATION

$50 Mandatory Fee

Pursuant to California Code of Regulations (CCR) § 1536, the Board will approve continuing education (CE) courses after receiving the applicable fee, the requested information below and it has been determined that the course meets criteria specified in CCR § 1536(g).

In addition to the information requested below, please attach a copy of the course schedule, a detailed course outline and presentation materials (e.g., PowerPoint presentation). Applications must be submitted 45 days prior to the course presentation date.

Please type or print clearly.

Course Title	**Course Presentation Date**
Biomechanical Glaucoma Damage	04/08/2017

Course Provider Contact Information

Provider Name

Amy (First) Atondo (Last) (Middle)

Provider Mailing Address

Street 1450 San Pablo St. **City** Los Angeles, **State** CA **Zip** 90033

Provider Email Address amy.atondo@med.usc.edu

Will the proposed course be open to all California licensed optometrists?	☑ YES ☐ NO
Do you agree to maintain and furnish to the Board and/or attending licensee such records of course content and attendance as the Board requires, for a period of at least three years from the date of course presentation?	☑ YES ☐ NO

Course Instructor Information

Please provide the information below and attach the curriculum vitae for each instructor or lecturer involved in the course. If there are more instructors in the course, please provide the requested information on a separate sheet of paper.

Instructor Name

Sahar (First) Bedrood (Last) (Middle)

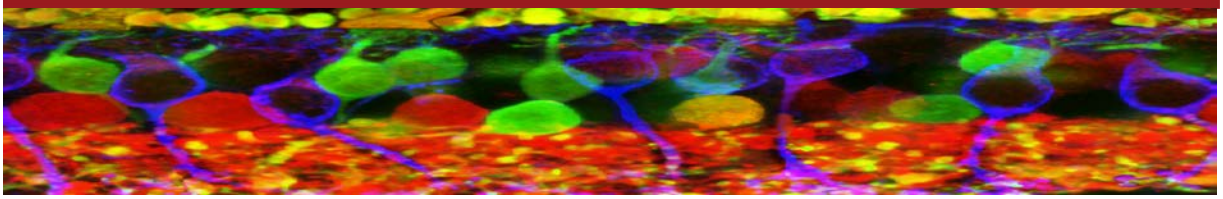
License Number A124192	**License Type** MD
Phone Number (323) 442-6372	**Email Address** sahar.bedrood@med.usc.edu

I declare under penalty of perjury under the laws of the State of California that all the information submitted on this form and on any accompanying attachments submitted is true and correct.

Signature of Course Provider *Date* 3/6/2017

USC Roski Eye Institute

Keck Medicine of USC



Dear Colleague:

Please join us for our FREE 3 HOUR Glaucoma CE by Drs. Sahar Bedrood, MD, PhD, Alena Reznik, MD and Grace Richter, MD, MPH on April 8, 2017. We invite you to participate in this specialty 3 hour continuing education (CE) lecture. We will have a Q & A after each Lecture. Lectures include:

08:00am – 08:30am	Registration and Continental Breakfast
08:30am – 08:40am	Welcom and Introduction by Dr. Alena Reznik
08:40am – 09:40am	OCT in Glaucoma by Dr. Grace Richter
09:40am – 10:40am	Biomechanical Glaucoma Damage By Dr. Sahar Bedrood
10:40am – 10:55am	Break
10:55am – 11:55am	Suprachoroidal Space by Dr. Alena Reznik

USC Roski Eye Institute
1450 San Pablo St. 3rd Floor Conference Room
Los Angeles, CA 90033

Free registration is provided for your convenience.

RSVP acceptance or regrets to Amy Atondo at amy.atondo@med.usc.edu Should you have any questions or require additional information**,** please call 323-442-6372.

The Keck School of Medicine of the University of Southern California designates this lecture for a maximum of 3 CE credits. Registrar and Administrator for the Optometry Portion is California State Board of Optometry.

Grace Richter, MD, MPH

Sahar Bedrood, MD, PhD

Alena Reznik, MD

Department of Ophthalmology * USC Eye Institute
Keck School of Medicine * University of Southern California

USC University of Southern California

Sahar Bedrood, MD, PhD.
Assistant Professor of Ophthalmology
Glaucoma Division
USC Roski Eye Institute

Summary:

There are many theories about the causes of glaucoma and the reason for the optic nerve damage in glaucomatous optic neuropathy. One of the causes of glaucoma includes the biomechanical theory of damage, in which the sclera and lamina cribrosa sustains a certain level of stress from intraocular pressure which then leads to retinal ganglion cell apoptosis and glaucoma. Studies are being done on how pressure along the lamina cribrosa affects its position and relationship to glaucomatous disease. Additionally, many analyses have been done in regards to how the structural changes along the optic nerve can affect its function. This talk will serve to review all of these concepts and discuss the structure-function relationship in glaucoma.

Outline:

Presentation 1: "Biomechanical Glaucoma Damage and the Structure-Function Relationship"
Presentation 2: "Interesting and Challenging Glaucoma Cases"

Optic Nerve Damage in Glaucoma
&
The Structure-Function Perspective

Sahar Bedrood, MD, PhD
Assistant Professor of Ophthalmology
Glaucoma Division
USC Roski Eye Institute

Disclosures

- none

Glaucoma

- Second cause of blindness worldwide
- IOP is a risk factor related to the presence and degree of optic nerve damage
- Tolerance of IOP varies greatly
- There are multiple theories of the causes of glaucoma, this lecture will discuss the structure and behavior of the connective tissue and the role it plays in the progression of glaucomatous optic neuropathy

Theories of Optic Nerve Damage

- Blood Flow theory
- Neurodegenerative theory
- Immune mediated damage
- Connective tissue/mechanical theory

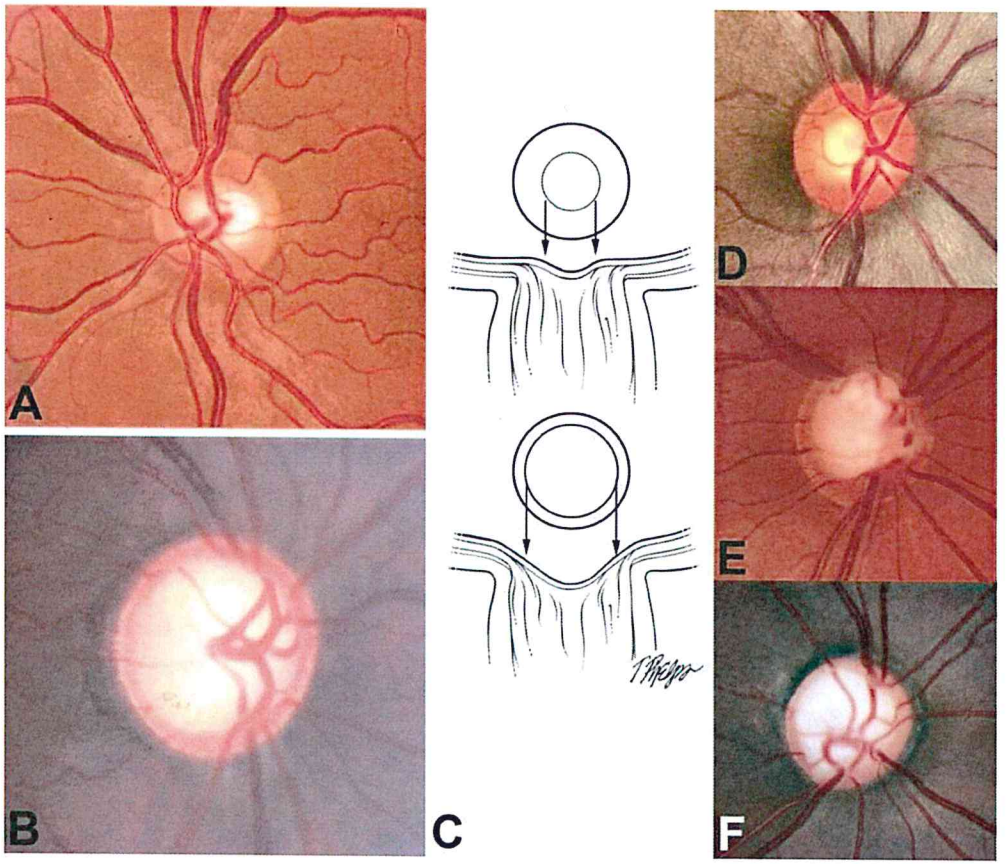
Glaucoma damage

- Glaucomatous optic nerve damage results in *deepening* and *widening* of the optic nerve head canal

Loss of RGC's

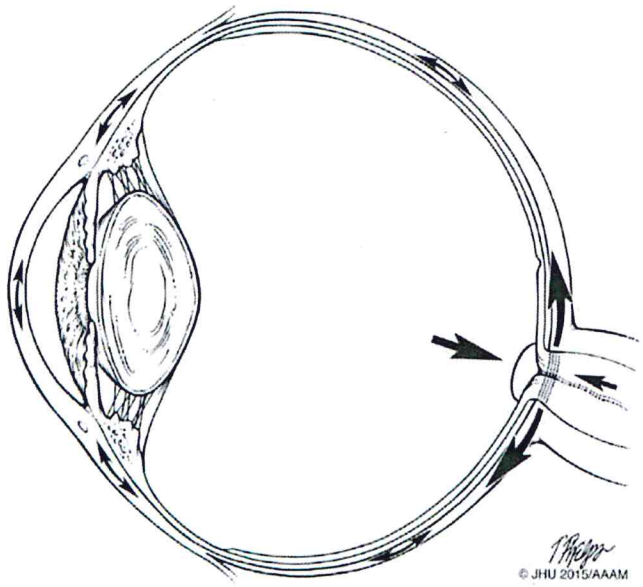
↓

Remodeling of connective tissue within ONH and surrounding peripapillary sclera



IOP, RGC's and the Optic nerve head

- Intraocular pressure induces circumferential stress in the sclera AND peripapillary (translaminar stress) region
- Increased stress pushes the connective tissue posteriorly and causes excavation or "cupping"
- Properties of the sclera related to age, disease or connective tissue disorders can play a role in its pliability



RGC death

RGC axon compression

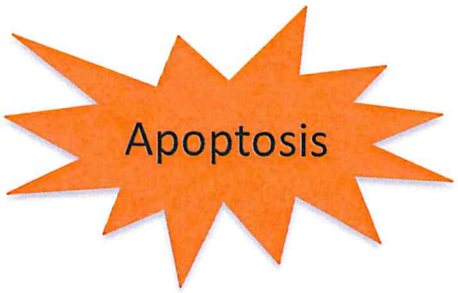
↓

Withdrawal of neurotrophic support and neurotrophic factors

↓

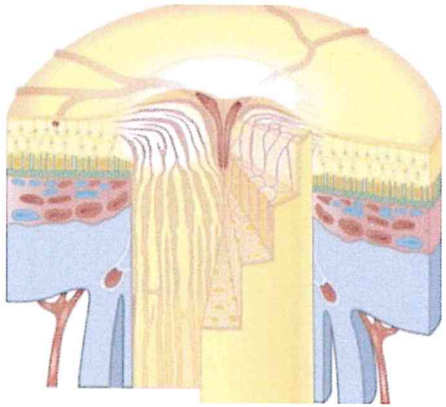
Anterograde and retrograde axonal transport disrupted in RGC

↓



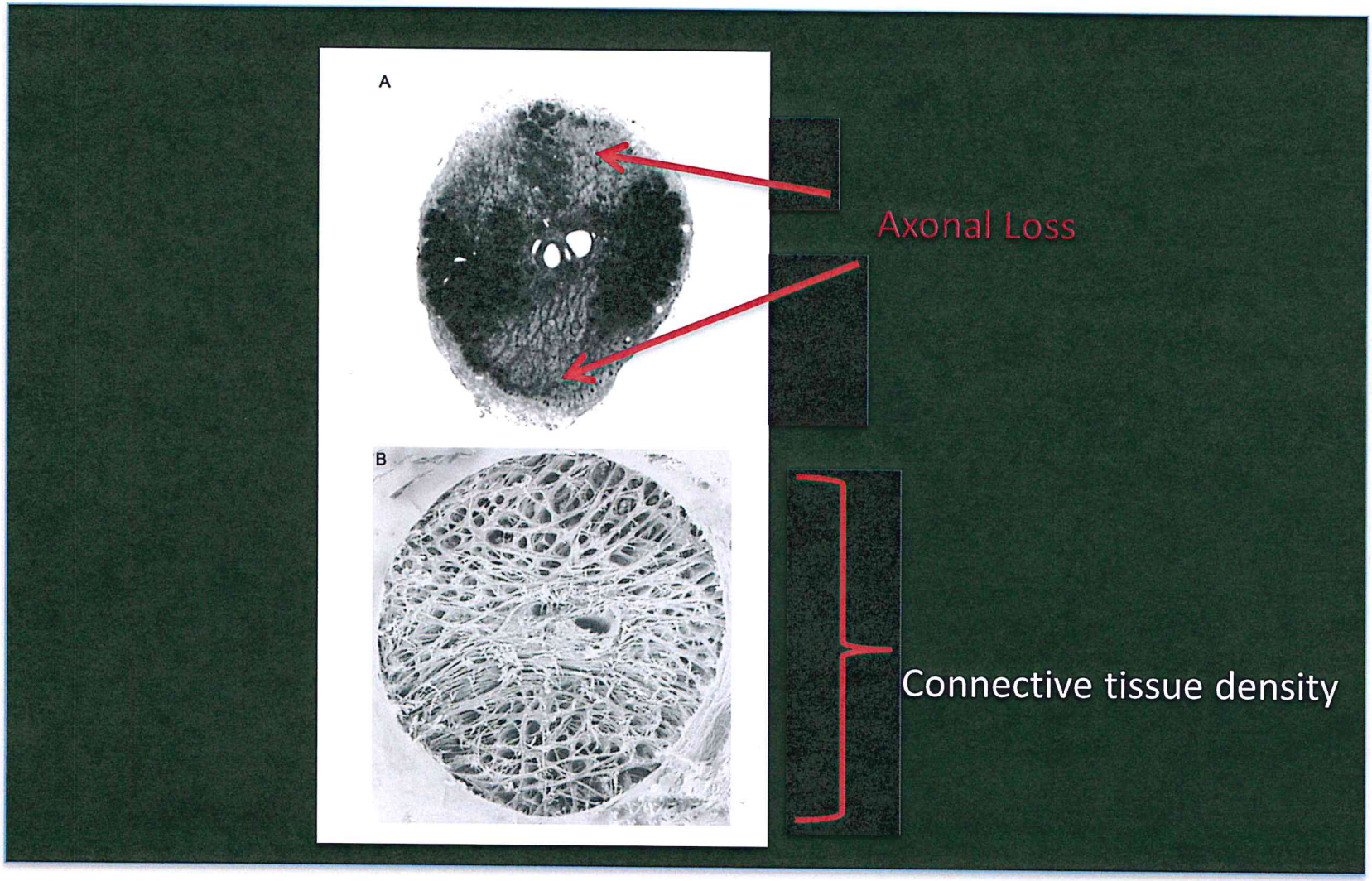
RGC apoptosis

- Studies have found that brain-derived neurotrophic factor (BDNF) and ciliary-derived neurotrophic factor are BLOCKED from reaching the RGC soma at the optic nerve head in acute and chronic IOP elevation
- Viral vector-derived overexpression of BNDF or CNTF is neuroprotective in rat glaucoma



Pease ME, McKinnon SJ, Quigley HA, Kerrigan-Baumrind LA, Zack DJ.
2000. Invest Ophthalmol Vis Sci. 41:764-74.

Pathological changes to the optic nerve head



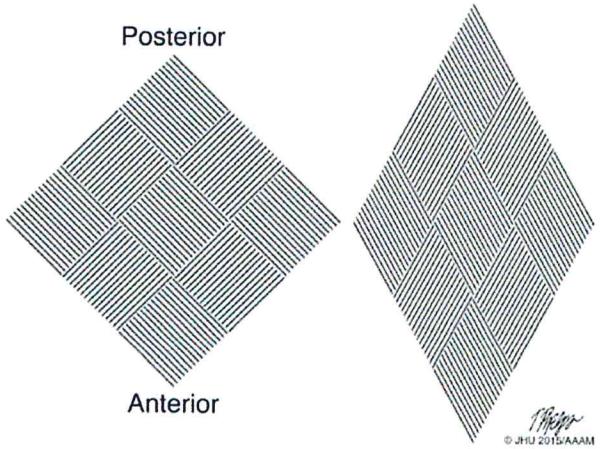
Quigley H.A. 2015. Progress in Brain research Volume 220, 2015, 59-86

Pathological changes to optic nerve head

- Deformation of the connective tissue is greatest along the areas of the optic nerve damage
- Deformative changes in
  - Myopia
  - Connective tissue disorder
  - Stiff sclera due to aging, crosslinking and genetic predisposition
- Susceptibility of OAG patients to glaucoma is from weakness in the baseline sclera or ONH state
- Fluctuations in the IOP further produce more stress and cause RGC

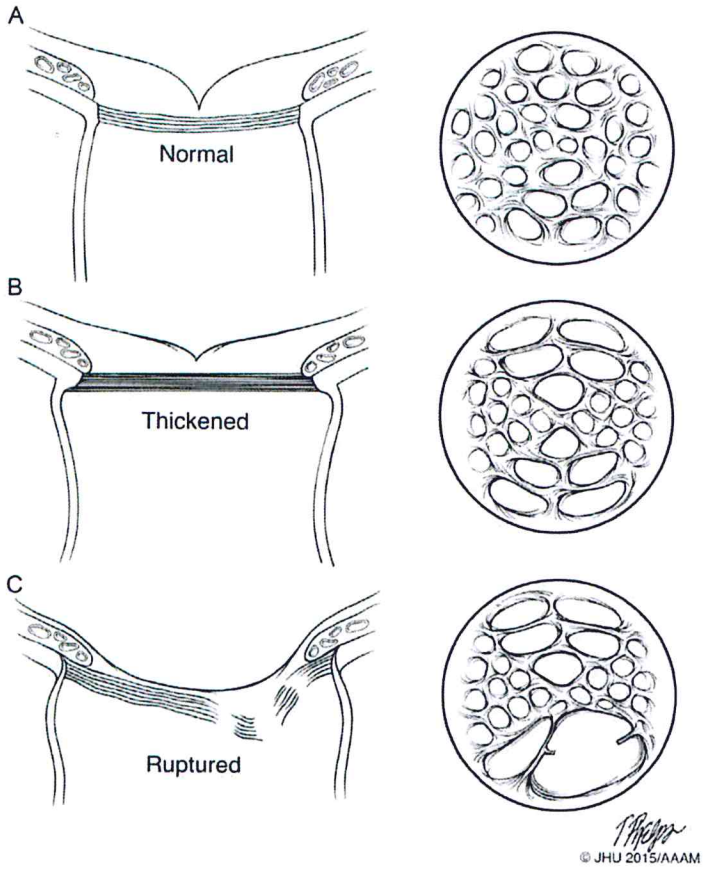
Changes to the Sclera in glaucoma

- The sclera consist of multiple layers of lamellae that consist of fibroblast that are arranged oblique or perpendicular to each other
- In experimental glaucoma eyes, the fibrils are arranged in a more antero-posterior orientation allowing for elongation of the sclera. This thinning does not happen in older human eyes with glaucoma due to crosslinking of the sclera
- Proteomic studies show up-regulation of proteins involved in scleral remodeling



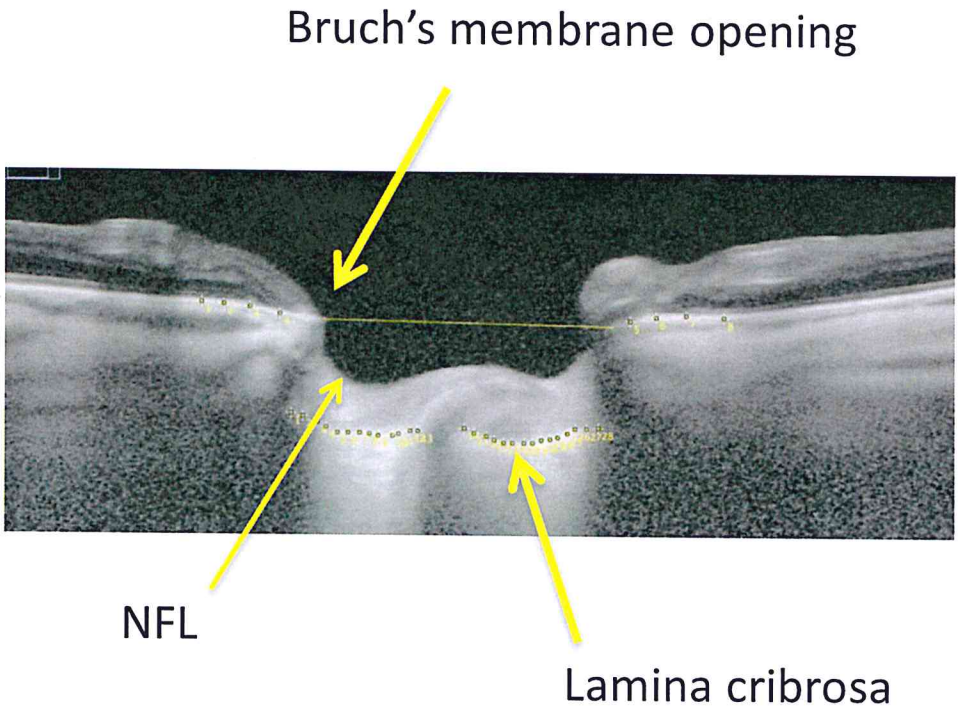
Remodeling of optic nerve head

- Glaucoma lamina showed remodeling of laminar beams with increased production of collagens 1, 3, 4
- These changes did not occur with optic atrophy from transection
- Rat model of glaucoma shows up-regulation of skeletal proteins



Changes to the lamina cribrosa

- Pressure changes can change the antero-posterior position of the lamina cribrosa
- The direction and degree of these changes can reflect the severity of disease
- Can these parameters be diagnostic of disease progression?



Connective tissue and cellular based therapy

- How does the pliability of the sclera contribute to the death of RGC's?
  - Too pliable leads to loss of structure and support of cells?
    - Crosslinking would make the sclera more stiff
  - Too stiff leads to strain on RGC's?
    - Digestion of collagen and matrix glycosaminoglycans
- Can cellular changes to astrocytes and fibroblasts change remodeling?

Structure-Function relationship

- Structural changes = neuroretinal rim loss, RNFL changes
- Functional changes = visual field changes
- "Structure before function"
- Often information from one domain us used to support the diagnostic findings seen in the other domain
- Each domain can vary in severity and its relationship to the severity of POAG

Mapping the Visual Field to the Optic Disc

Structural damage

↓

Decrease in neuroretinal rim

↓

Reduction in axon number histologically

↓

What is the correspondence of these structural deficits to the visual field ?

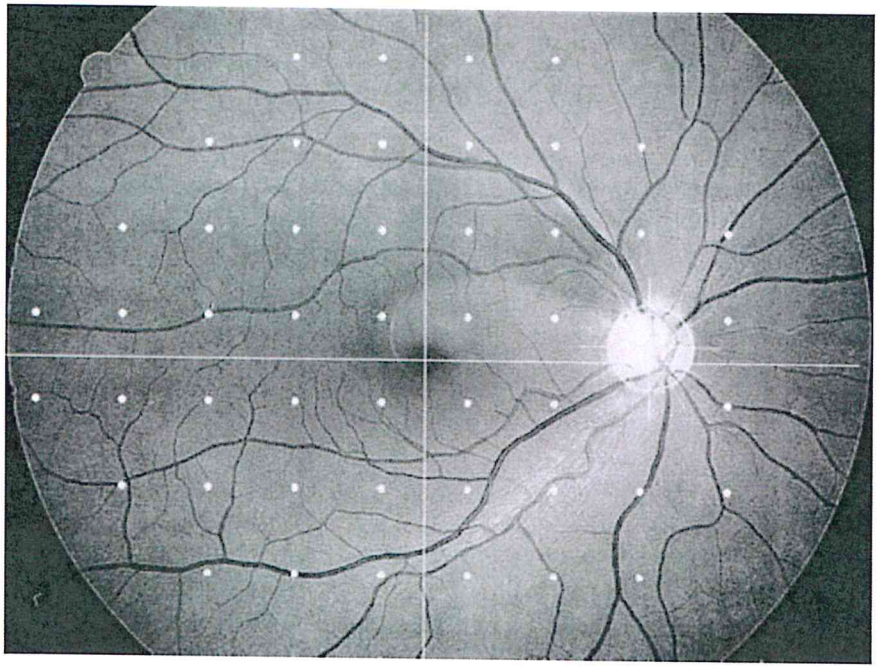


Figure 2. Example of a retinal nerve fiber layer photograph with the Humphrey 24-2 visual field test pattern and optic nerve head reference circle superimposed.

Garway-Heath D et al 2000 Mapping the Visual Field to the Optic Disc in Normal Tension Glaucoma Eyes *Ophthalmology 2000;107: 1809–1815*

Models of Structure-Function Relationship

- Hood-Kardon model
- Harwerth et al model
- Drasdo et al model
- The Hockey Stick model

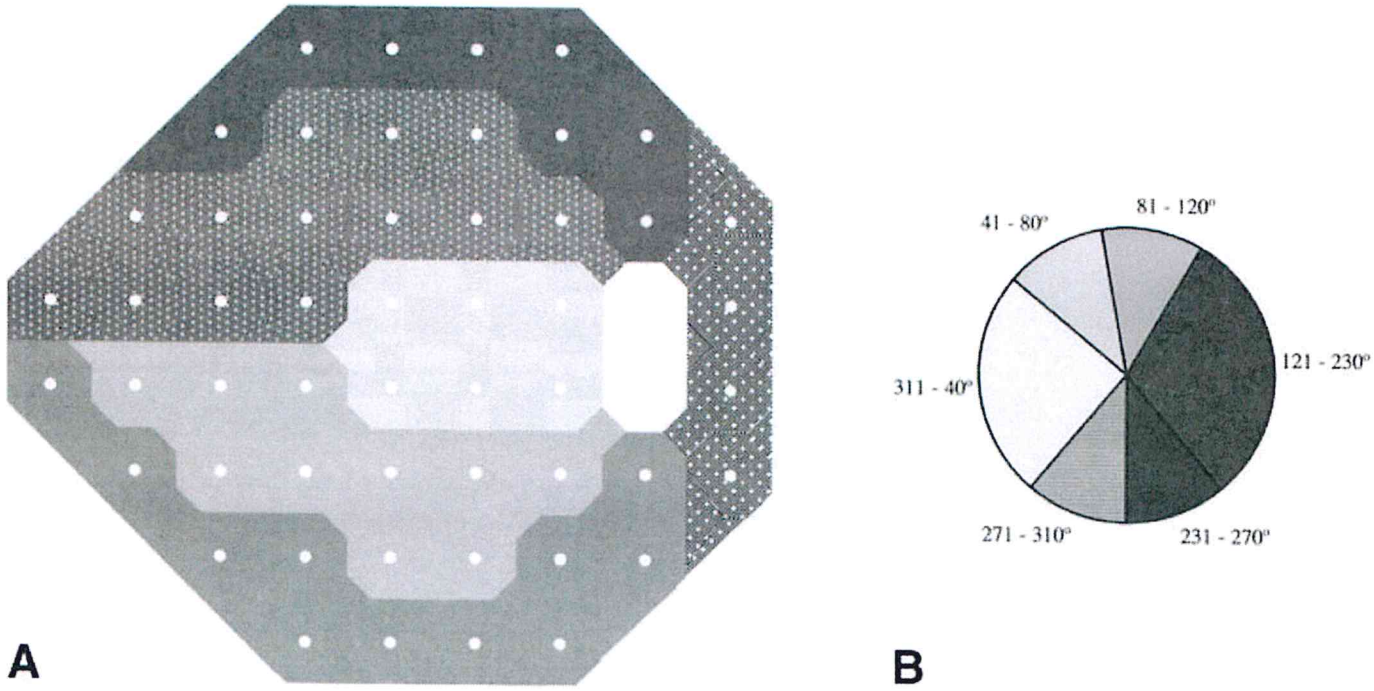
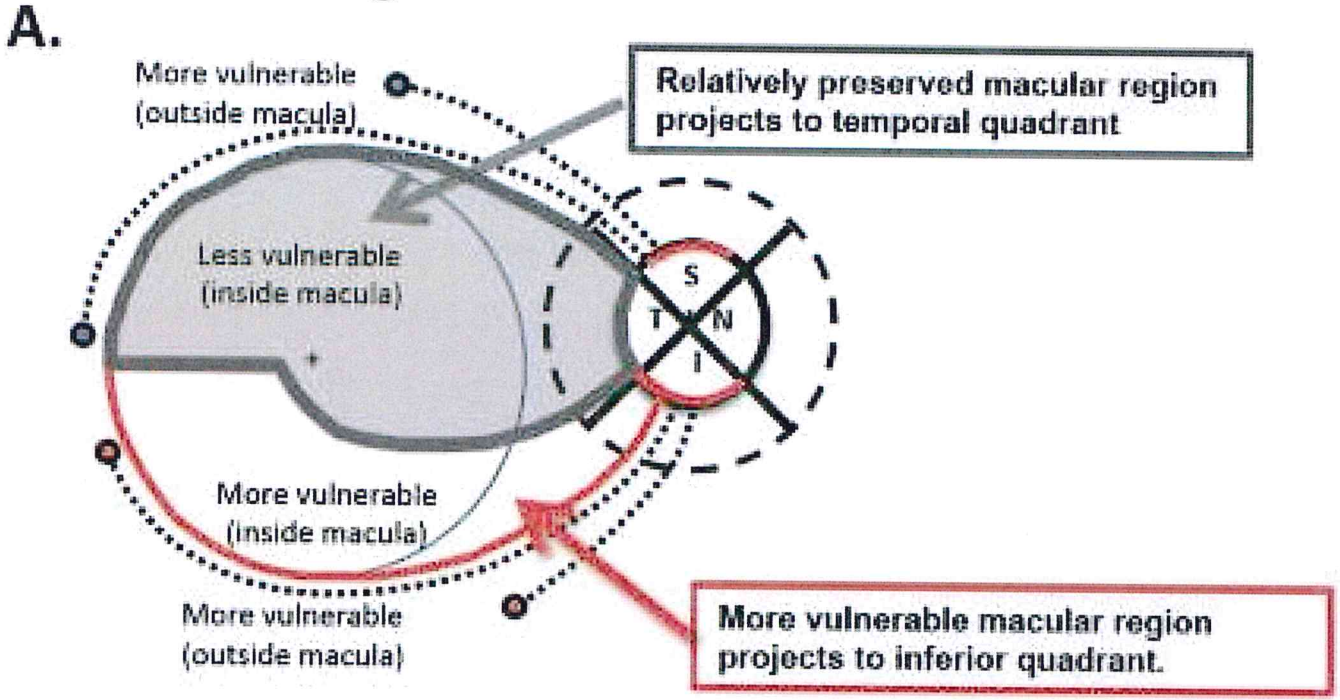


Figure 7. A division of the visual field **(A)** and optic nerve head **(B)** into sectors according to the results of this study.

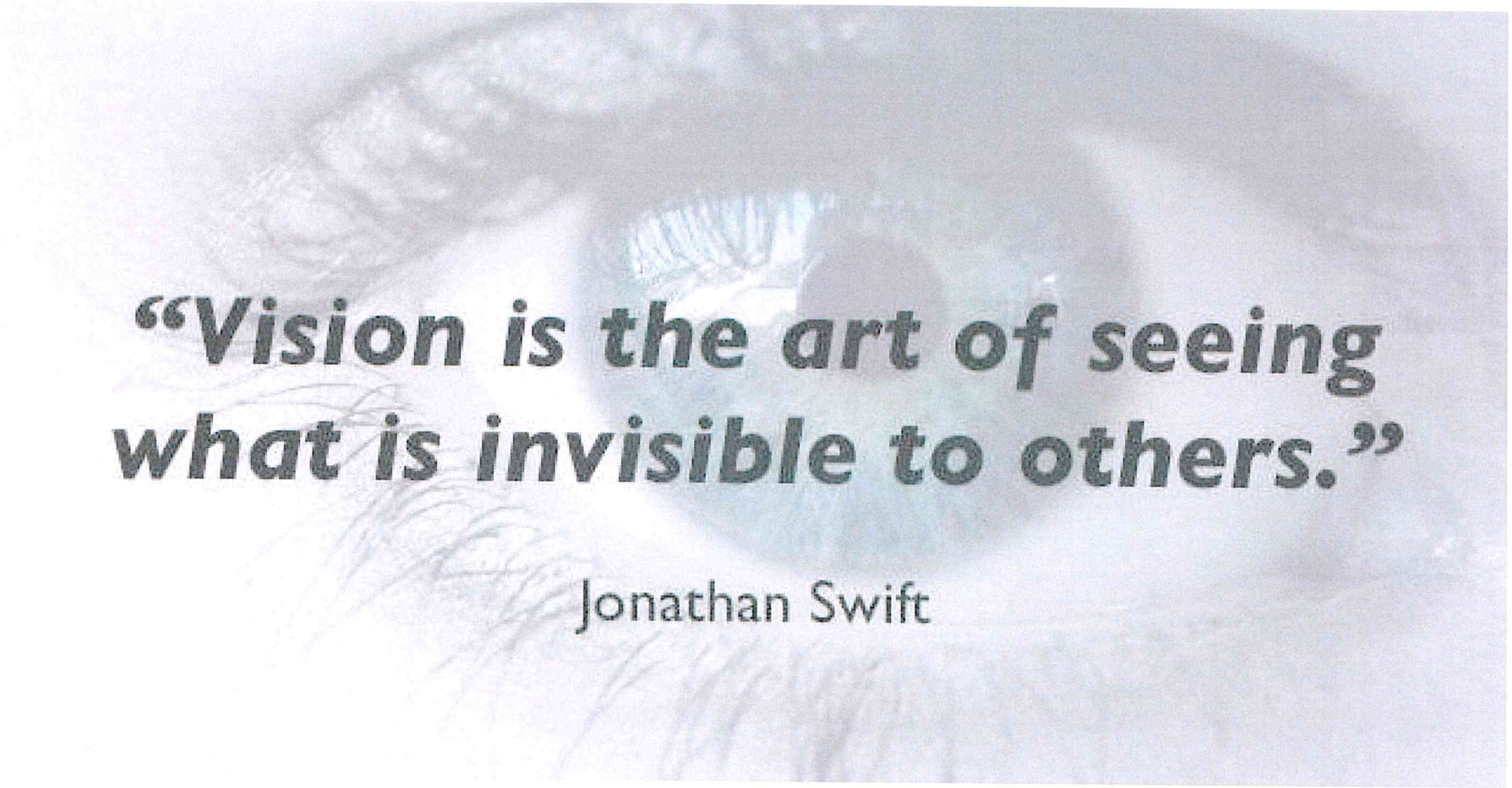
Vulnerability of the ganglion cells in glaucoma



Summary

- The mechanical properties of the lamina cribrosa and sclera play a pivotal role in glaucoma progression
- Variability of an individuals scleral properties can contribute to their tolerance of intraocular pressure
- Studies are currently being done on the movement and importance of the lamina cribrosa in glaucoma
- The structure-function model describes the relationship between RNFL changes and visual changes

Thank You


"Vision is the art of seeing what is invisible to others."
Jonathan Swift

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- Pease ME, Zack DJ, Berlinicke CA, Bloom KM, Cone FE, et al. 2009. CNTF over-expression leads to increased retinal ganglion cell survival in experimental glaucoma. Invest Ophthalmol Vis Sci. 50:2194- 200.
- Garway-Heath D, Poinoosawmy D, Fitzke FW, Hitchings RA 2000 Mapping the Visual Field to the Optic Disc in Normal Tension Glaucoma Eyes *Ophthalmology 2000;107: 1809–1815*

Other Models of Structure-Function Relationship

- Hood-Kardon model
  - Predicts RNFL thickness from dB visual field measurements
  - Linear relationship between RNFL thickness and visual field sensitivity
- Harwerth et al model
  - Comparison of perimetric data and histological data found a liner relationship between visual field sensitivity and ganglion cell number
- Drasdo et al model
  - Used a map of normal ganglion cell density within the central visual field to develop a model of ganglion cell density to perimetric sensitivity
- The Hockey Stick model
  - the relationship between sensitivity and ganglion cell receptive field density (measured from grating acuity) was linear with a slope of 1 at greater eccentricities and with a slope of 0.16 in the macula

Interesting Cases

Sahar Bedrood, MD, PhD

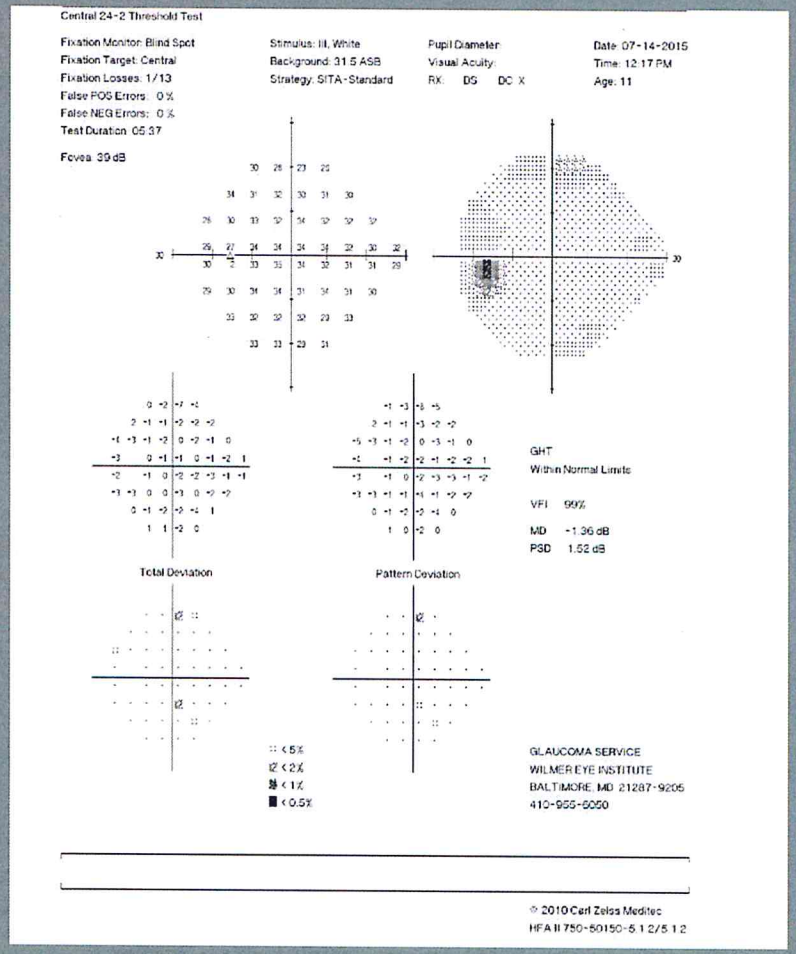
Case 1:

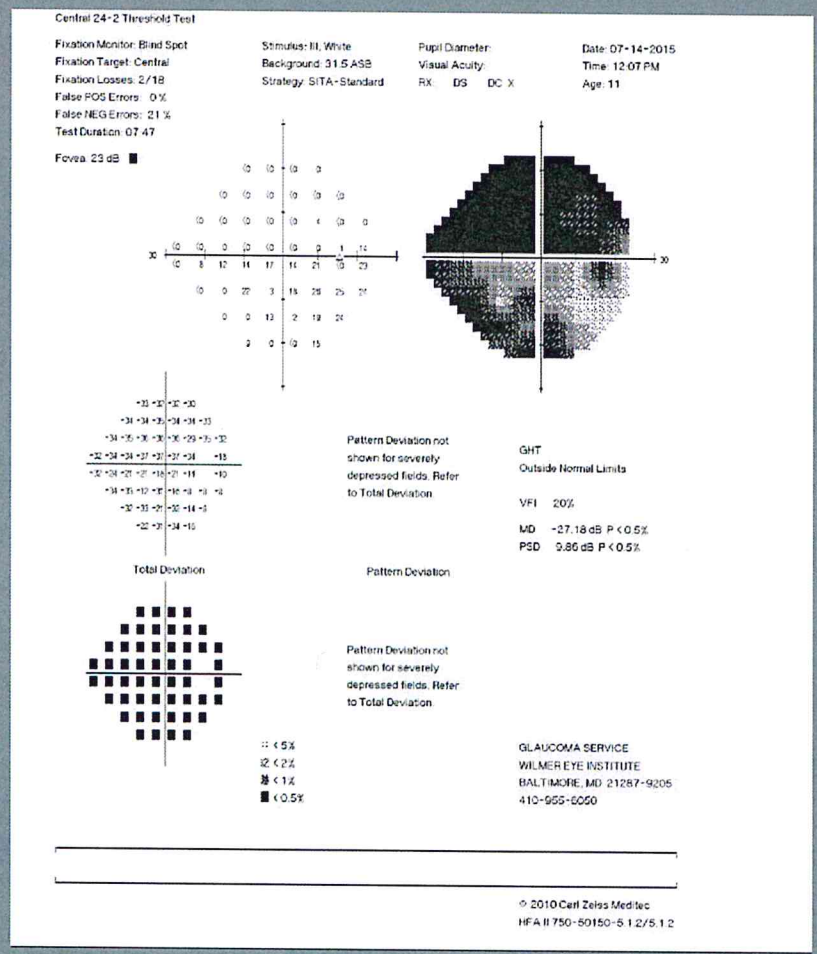
- 11 yo Female with no PMH seen by optometrist for refraction and sent urgently for high IOP
- PMH: none, no systemic symptoms
- Family history: grandparents with glaucoma
- Va 20/40 ou
- T(iCare): 43/30
- Pachymetry: 575/560
- Gonioscopy:

SLE and DFE

- SLE
  - LLL – wnl ou
  - S/C – White ou
  - K – clear ou
  - AC – Deep and Quiet ou
  - I- round and reactive ou
  - Lens- clear ou
  - AV – wnl ou
- DFE
  - c/d right eye: 0.95 , deeply excavated
  - c/d left eye: 0.75
  - Macula – normal
  - Vessels - + venous pulsations right eye
  - Periphery – wnl

Humphrey Visual Fields



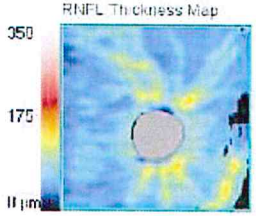


ID:
DOB:
Gender:
Technician: Operator, Cirrus

Exam Date:	6/2/2015	6/2/2015	WEI - Glaucoma
Exam Time:	3:38 PM	3:39 PM	
Serial Number:	4000-9229	4000-9229	
Signal Strength	8/10	10/10	

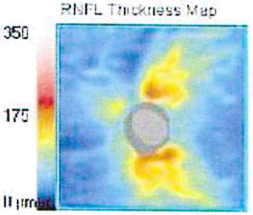
ONH and RNFL OU Analysis:Optic Disc Cube 200x200 OD ● | ● OS

RNFL Thickness Map



	OD	OS
Average RNFL Thickness	68 µm	85 µm
RNFL Symmetry	37%	
Rim Area	0.33 mm²	0.99 mm²
Disc Area	2.19 mm²	1.97 mm²
Average C/D Ratio	0.93	0.71
Vertical C/D Ratio	0.93	0.66
Cup Volume	1.030 mm³	0.619 mm³

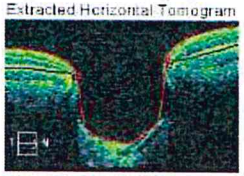
RNFL Thickness Map



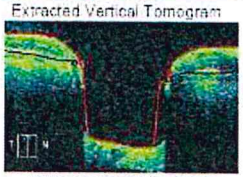
RNFL Deviation Map

Disc Center(0.03,-0.57)mm

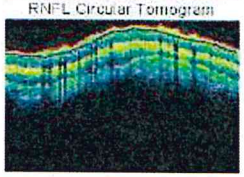
Extracted Horizontal Tomogram



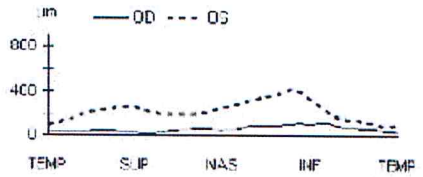
Extracted Vertical Tomogram



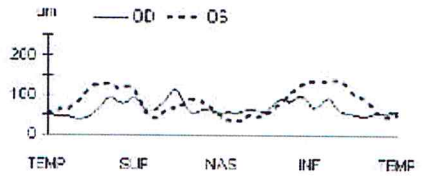
RNFL Circular Tomogram



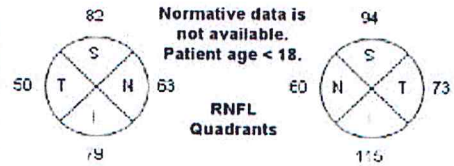
Neuro-retinal Rim Thickness



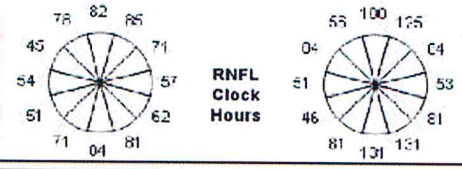
RNFL Thickness



Normative data is not available. Patient age < 18.

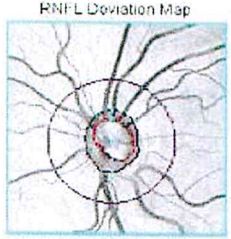


RNFL Quadrants



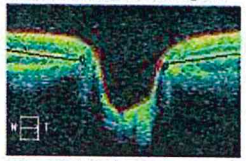
RNFL Clock Hours

RNFL Deviation Map

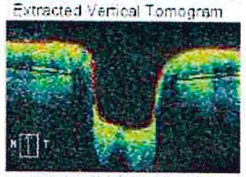


Disc Center(-0.18,-0.33)mm

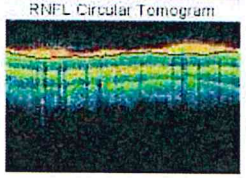
Extracted Horizontal Tomogram



Extracted Vertical Tomogram



RNFL Circular Tomogram



Comments	Doctor's Signature

CirrusG1
SW Ver: 7.0.2.5

Diagnosis & Course of treatment

- Juvenile Open angle glaucoma
  - 6/02/15: Started on latanoprost, dorzolamide-timolol and brimonidine in the right eye and continued to have IOP of 23
    - 8/3/15 Baerveldt glaucoma implant with rip cord suture
    - 9/8/15 The tube was noted to have moved and be abutted against the limbus and nonpatent with an IOP 48
    - Latina suture removed, IOP still high
    - 9/28/15: revision with tube extender
    - 11/15/15: IOP in the right eye 23, left eye 17 with normal fields in the left eye

Learning points for this case

- Juvenile sclera are different
- Rip cord sutures can provide another safeguard against hypotony
- Tube extenders
- When revising a tube:
  - insert in same spot or take out?
  - Insert and suture into a different entry site?

Case 2

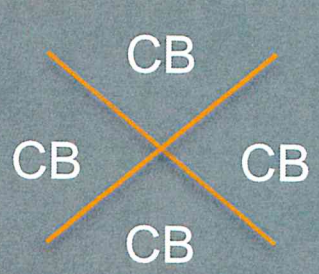
- 70 yo Male presented with high IOP after cataract surgery in the left eye and wanted a second opinion regarding surgery
- Family history: none

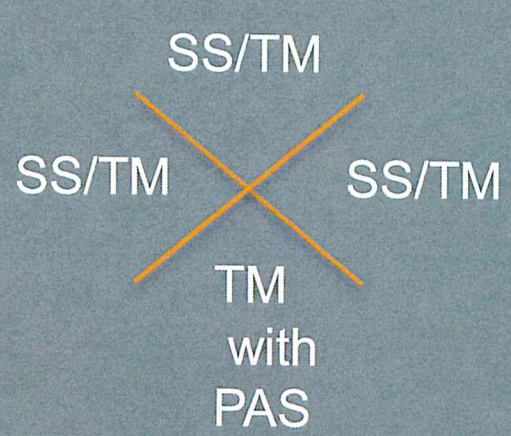
- Ocular history: Primary open angle glaucoma s/p complicated cataract surgery with nucleus drop, vitrectomy
- POD#1 – developed retinal detachment, underwent repair with buckle
- IOP remained high (IOP spikes to 40) during first 3 postoperative months and underwent trabeculectomy in the left eye
- 1.5 years later he is having high IOP in 30's and wants a second opinion
- Currently on: PF BID OS only, dorzolamide-timolol BID OU, brimonidine BID OU, methazolamide 50 mg BID

Exam

- Va 20/25, 20/40
- IOP: 17, 37
- CCT 520/510

- Gonio

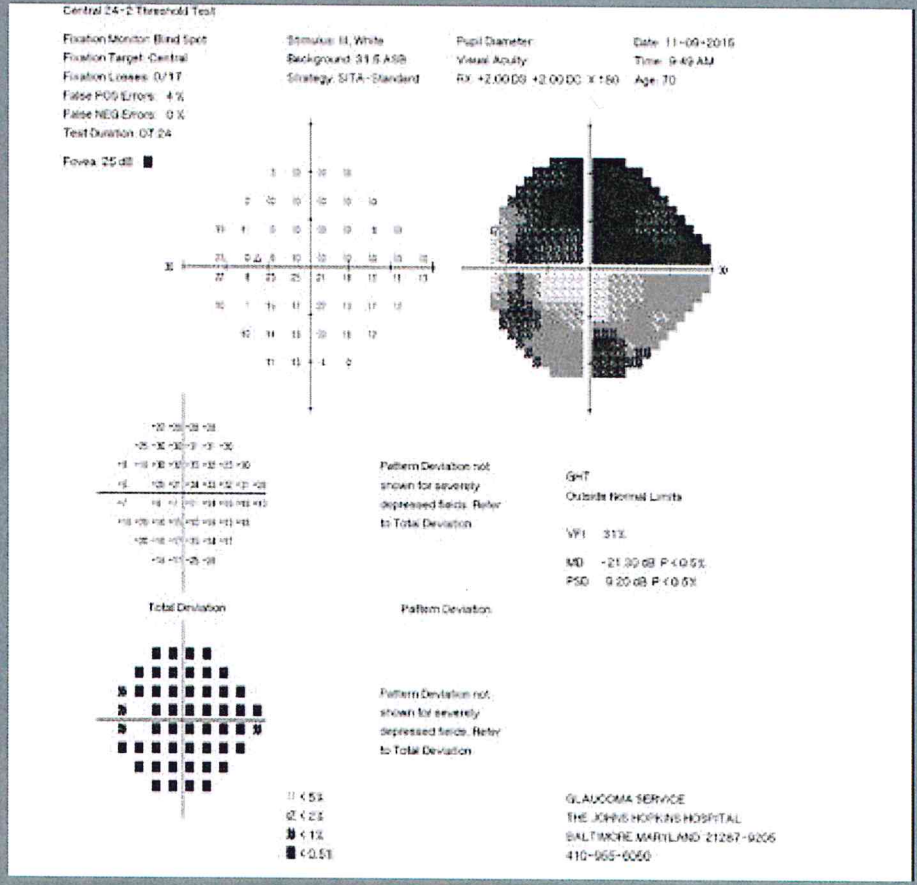


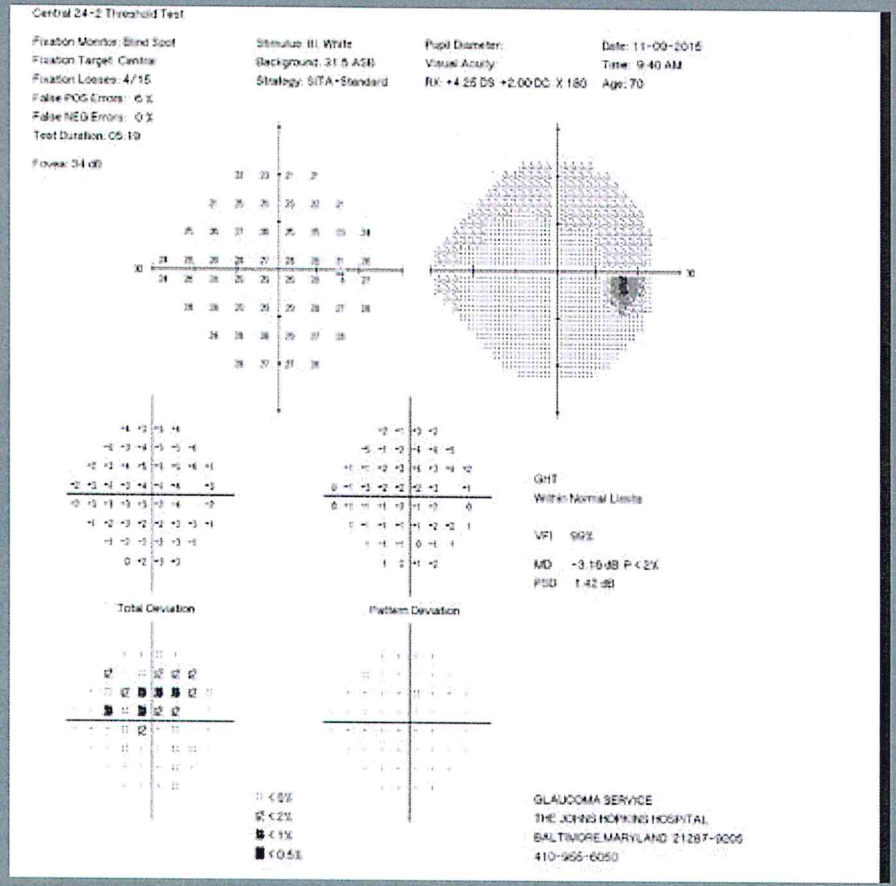


SLE and DFE

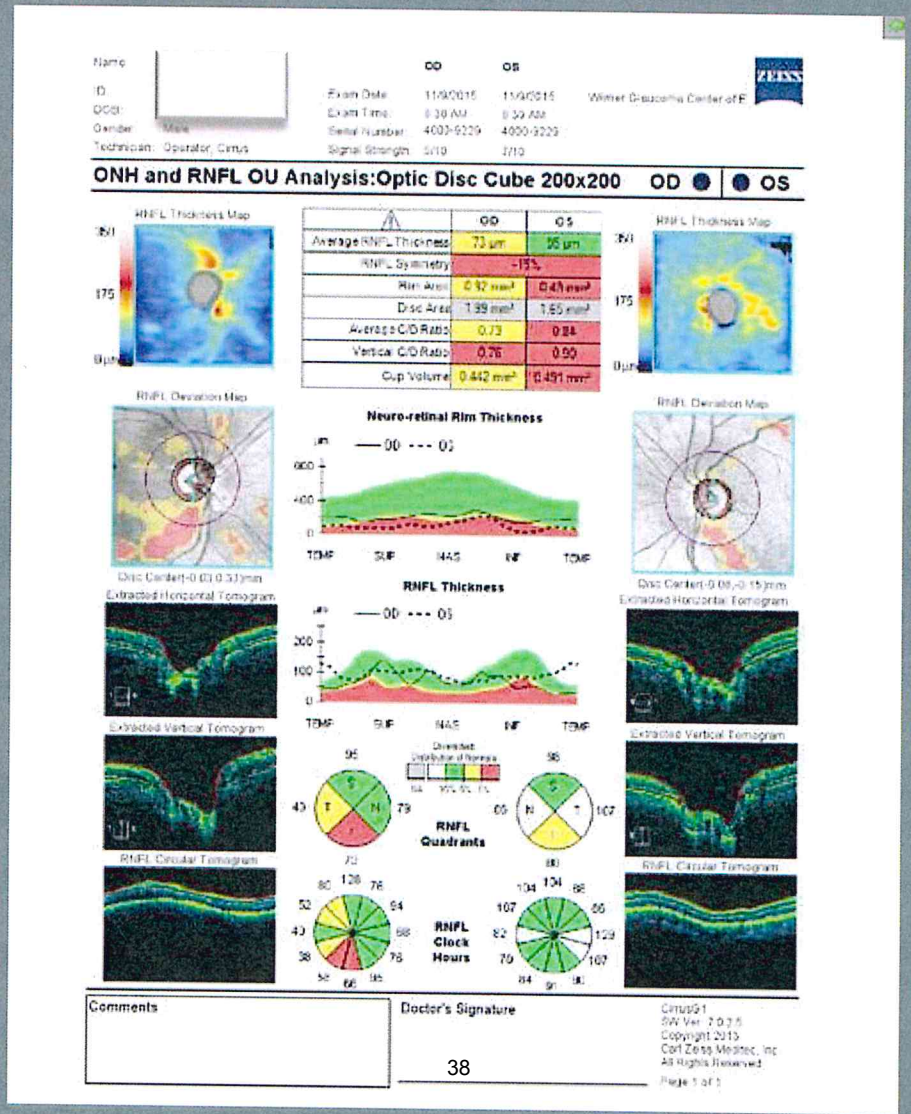
- SLE
  - LLL – blepharitis ou
  - S/C – White OD, vascular scarred bleb superiorly OS, scleral buckle posteriorly placed
  - K – clear ou with suture temporally
  - AC – Deep and Quiet ou
  - I- round and reactive ou
  - Lens- 1+ NSC OD, PCIOL OS in sulcus with open capsule
  - AV – wnl ou
- DFE
  - c/d right eye: 0.6
  - c/d left eye: 0.9
  - Macula – normal
  - Vessels - normal
  - Periphery – good buckle height, laser scars in periphery

HVF


Central 24-2 Threshold Test
Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 0/17
False POS Errors: 4 %
False NEG Errors: 0 %
Test Duration: 07:24
Fovea: 25 dB
Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA-Standard
Pupil Diameter:
Visual Acuity:
RX: +2.00 DS +2.00 DC X 180
Date: 11-09-2015
Time: 9:49 AM
Age: 70
Total Deviation
Pattern Deviation
Pattern Deviation not shown for severely depressed fields. Refer to Total Deviation
GHT
Outside Normal Limits
VFI 31%
MD -21.30 dB P<0.5%
PSD 9.20 dB P<0.5%
Pattern Deviation not shown for severely depressed fields. Refer to Total Deviation
< 5%
< 2%
< 1%
< 0.5%
GLAUCOMA SERVICE
THE JOHNS HOPKINS HOSPITAL
BALTIMORE, MARYLAND 21287-9205
410-955-6050


Central 24-2 Threshold Test
Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 4/15
False POS Errors: 6 %
False NEG Errors: 0 %
Test Duration: 05:19
Fovea: 34 dB
Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA-Standard
Pupil Diameter:
Visual Acuity:
RX: +4.25 DS +2.00 DC X 180
Date: 11-09-2015
Time: 9:40 AM
Age: 70
Total Deviation
Pattern Deviation
GHT
Within Normal Limits
VFI 99%
MD -3.16 dB P<2%
PSD 1.42 dB
< 5%
< 2%
< 1%
< 0.5%
GLAUCOMA SERVICE
THE JOHNS HOPKINS HOSPITAL
BALTIMORE, MARYLAND 21287-9205
410-955-6050

OCT



Name
ID
DOB
Gender
Technician: Operator, Cirrus

	OD	OS
Exam Date	11/9/2015	11/9/2015
Exam Time	8:38 AM	8:39 AM
Serial Number	4000-9229	4000-9229
Signal Strength	5/10	7/10

Winter Glaucoma Center of E

ZEISS

ONH and RNFL OU Analysis:Optic Disc Cube 200x200 OD ● ● OS

RNFL Thickness Map

	OD	OS
Average RNFL Thickness	73 µm	96 µm
RNFL Symmetry	-15%	
Rim Area	0.92 mm²	0.47 mm²
Disc Area	1.99 mm²	1.65 mm²
Average C/D Ratio	0.73	0.84
Vertical C/D Ratio	0.76	0.90
Cup Volume	0.442 mm³	0.491 mm³

RNFL Deviation Map

Neuro-retinal Rim Thickness

RNFL Thickness

Extracted Horizontal Tomogram

Extracted Vertical Tomogram

RNFL Circular Tomogram

RNFL Quadrants

RNFL Clock Hours

Comments

Doctor's Signature

Cirrus/91
SW Ver: 7.0.2.5
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Page 1 of 1

Questions to consider

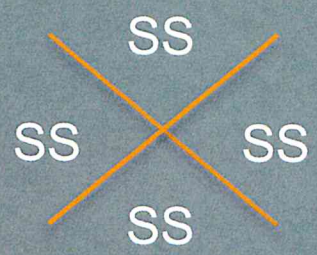
- What are the surgical options?
  - second trabeculectomy
  - needling the bleb
  - implant a Ahmed or Baerveldt tube
- Can one place a tube in an eye with a scleral buckle?

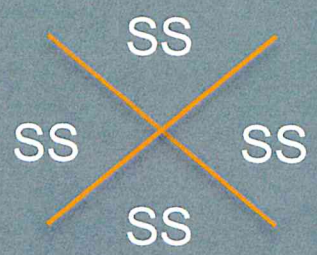
Case 3

- 42 yo M with Grave's disease referred to glaucoma to evaluate visual field defects
  - POHx: Graves disease s/p upper and lower eyelid retraction repair, was told by airforce that he may have early visual field loss in early 2000's
  - Currently has proptosis and is considering orbital decompression surgery
  - Recent and first episode of mild iritis and photosensitivity
  - Family history: maternal grandmother used glaucoma drops, no vision loss

Exam

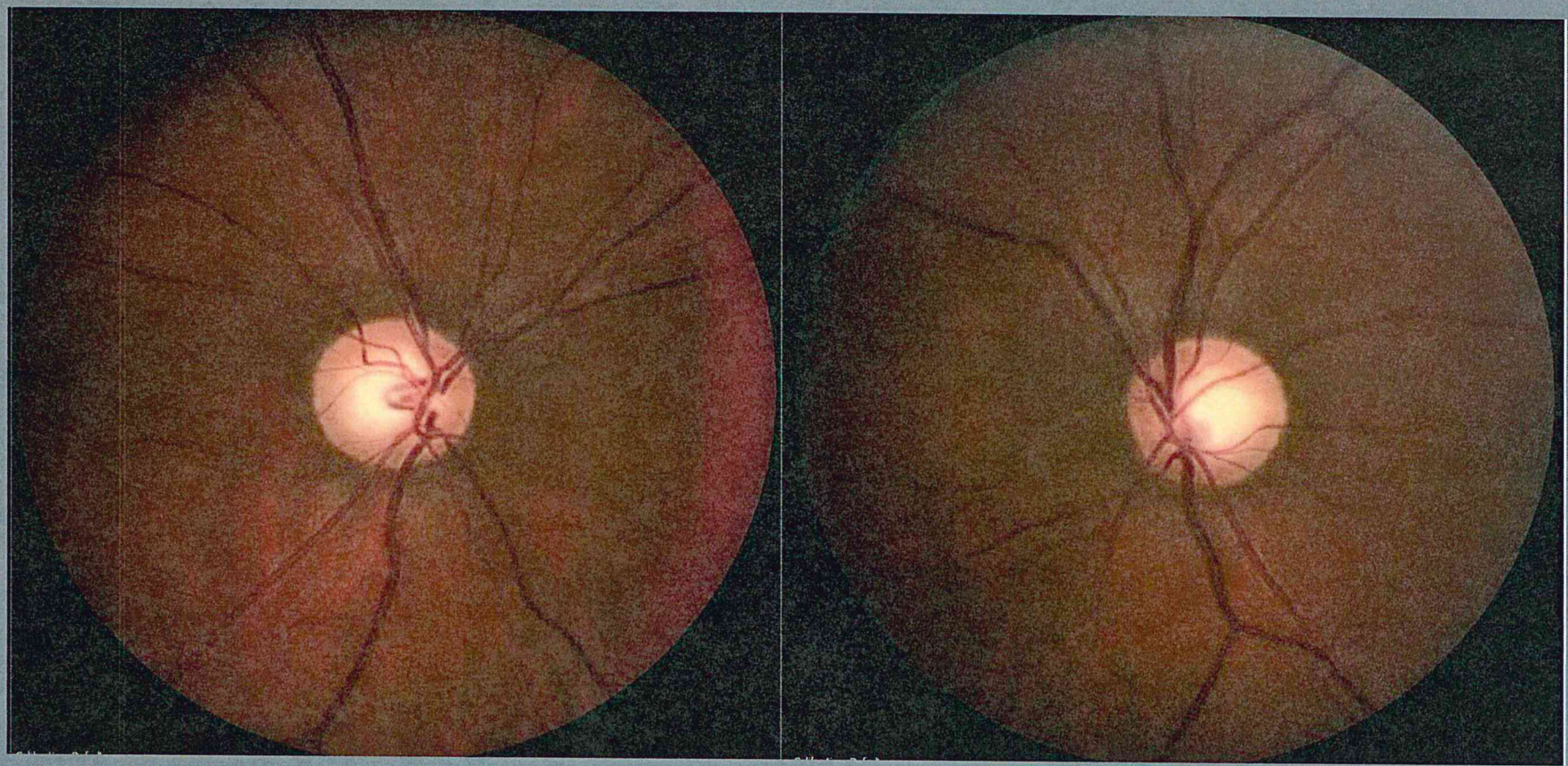
- Va 20/20 OU
- IOP: 19/18
- Pachymetry: 561/551
- PERRL and no APD



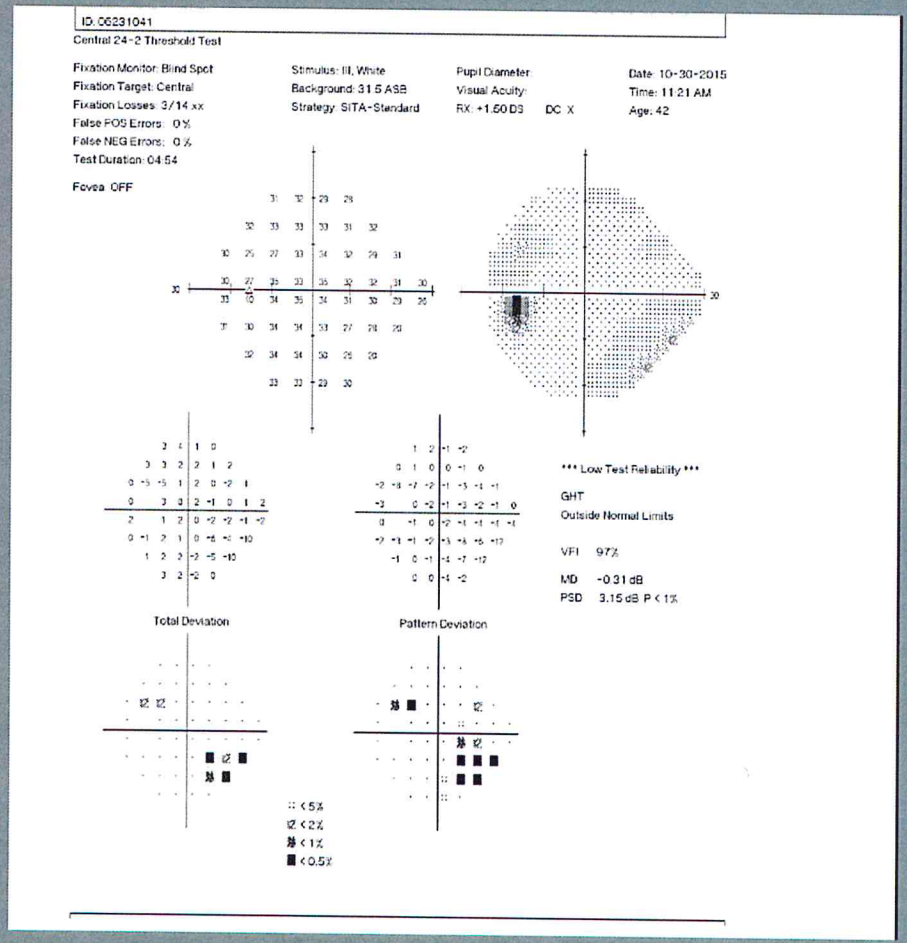


SLE and DFE

- SLE
  - LLL – mild edema of upper and lower eyelid OD, ptosis OS
  - S/C – White od, 1+ injection OS
  - K – clear ou
  - AC – Deep and Quiet ou
  - I- round and reactive ou
  - Lens- clear ou
  - AV – wnl ou
- DFE
  - c/d right eye: 0.7
  - c/d left eye: 0.7
  - Macula – normal
  - Vessels - normal
  - Periphery – wnl



HVF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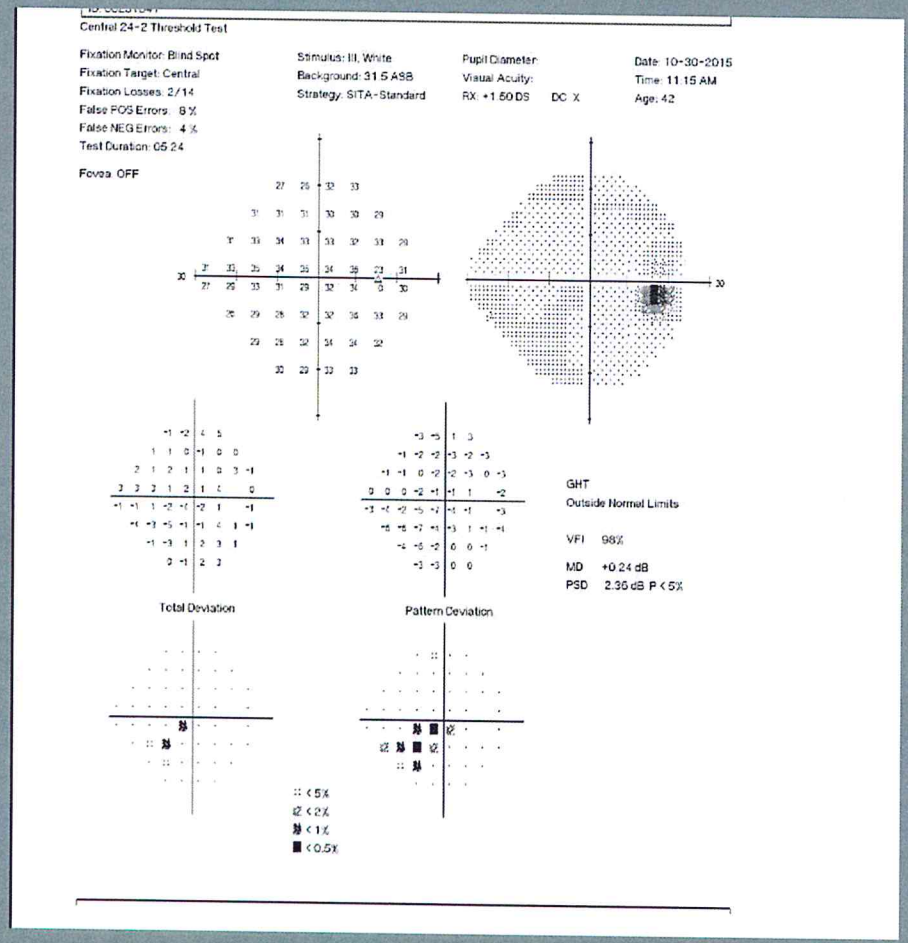
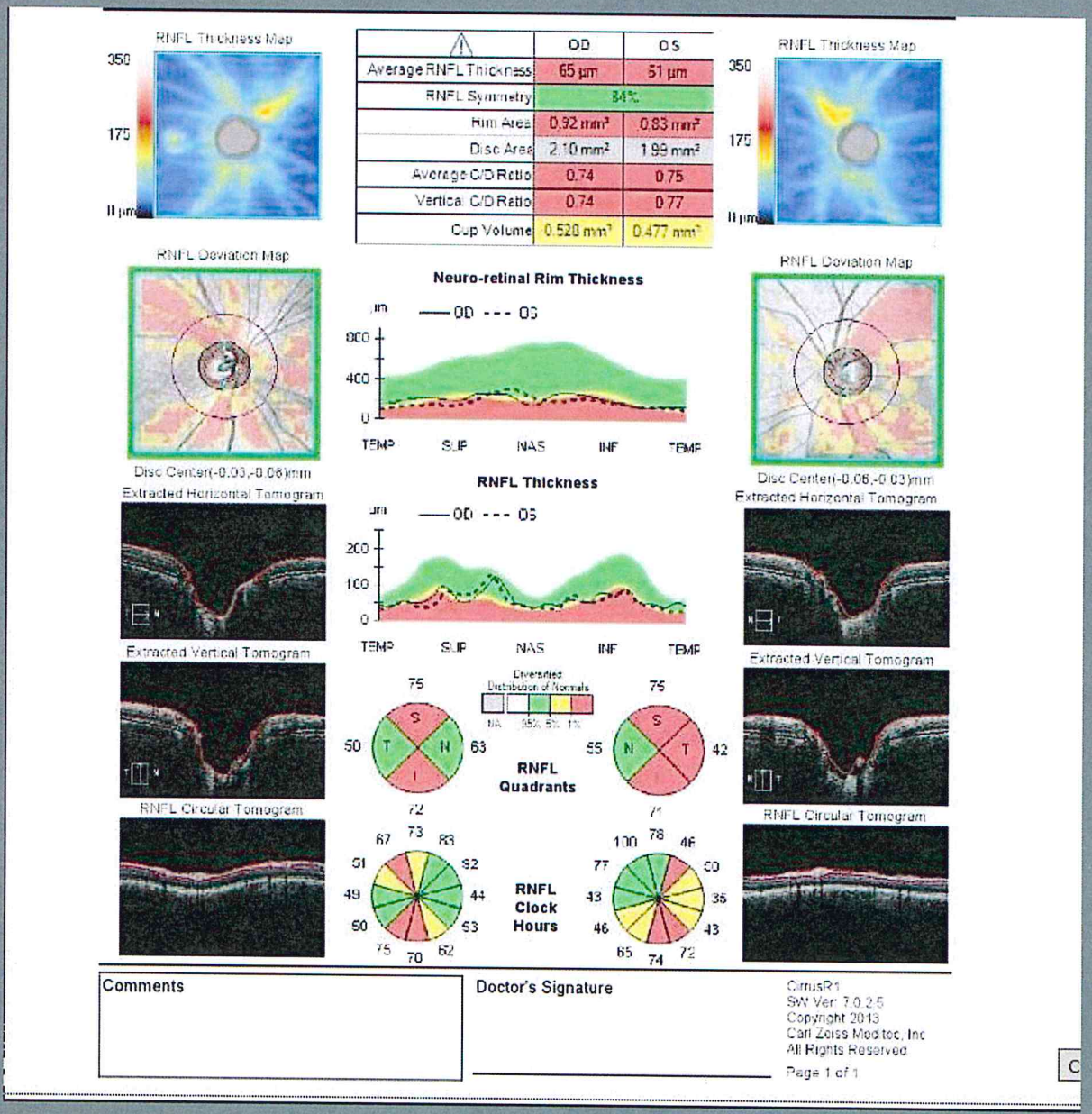
Central 24-2 Threshold Test
Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 3/14 xx
False POS Errors: 0 %
False NEG Errors: 0 %
Test Duration: 04:54
Fovea: OFF
Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA-Standard
Pupil Diameter:
Visual Acuity:
RX: +1.50 DS
DC: X
Date: 10-30-2015
Time: 11:21 AM
Age: 42
*** Low Test Reliability ***
GHT
Outside Normal Limits
VFI 97%
MD -0.31 dB
PSD 3.15 dB P < 1%
Total Deviation
Pattern Deviation
:: < 5%
Ø < 2%
▒ < 1%
■ < 0.5%


Central 24-2 Threshold Test
Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 2/14
False POS Errors: 6 %
False NEG Errors: 4 %
Test Duration: 05:24
Fovea: OFF
Stimulus: III, White
Background: 31.5 ASB
Strategy: SITA-Standard
Pupil Diameter:
Visual Acuity:
RX: +1.50 DS
DC: X
Date: 10-30-2015
Time: 11:15 AM
Age: 42
GHT
Outside Normal Limits
VFI 98%
MD +0.24 dB
PSD 2.36 dB P < 5%
Total Deviation
Pattern Deviation
:: < 5%
Ø < 2%
▒ < 1%
■ < 0.5%

OCT


RNFL Thickness Map
350
175
0 µm
OD
OS
Average RNFL Thickness
65 µm
51 µm
RNFL Symmetry
84%
Rim Area
0.92 mm²
0.83 mm²
Disc Area
2.10 mm²
1.99 mm²
Average C/D Ratio
0.74
0.75
Vertical C/D Ratio
0.74
0.77
Cup Volume
0.528 mm³
0.477 mm³
RNFL Deviation Map
Neuro-retinal Rim Thickness
µm
OD
OS
800
400
0
TEMP
SUP
NAS
INF
TEMP
Disc Center(-0.03,-0.06)mm
Extracted Horizontal Tomogram
RNFL Thickness
200
100
Extracted Vertical Tomogram
RNFL Circular Tomogram
Diversified Distribution of Normals
RNFL Quadrants
S
T
N
I
RNFL Clock Hours
Comments
Doctor's Signature
CirrusR1
SW Ver 7.0.2.5
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Page 1 of 1

Discussion

- Is this glaucoma?
- Contribution from steroids a cause?
- Would you initiate treatment at his pressure of 18/19?
- How often would you monitor him?

Course:

- Strong suspicion for glaucoma given HVF, rnfl loss and family Hx.
- Patient was started on timolol with plan for repeat HVF

Case 4

- 64 yo F with longstanding OAG with
  - s/p CEIOL OU 2014
  - Endolaser to CB OU
  - Tmax 20
  - Trabeculectomy 8/6/15 with 1/3 lysis of sutures
- Presents after husband accidentally hit her in the eye while sleeping and stated noticing a change in the position of the bleb

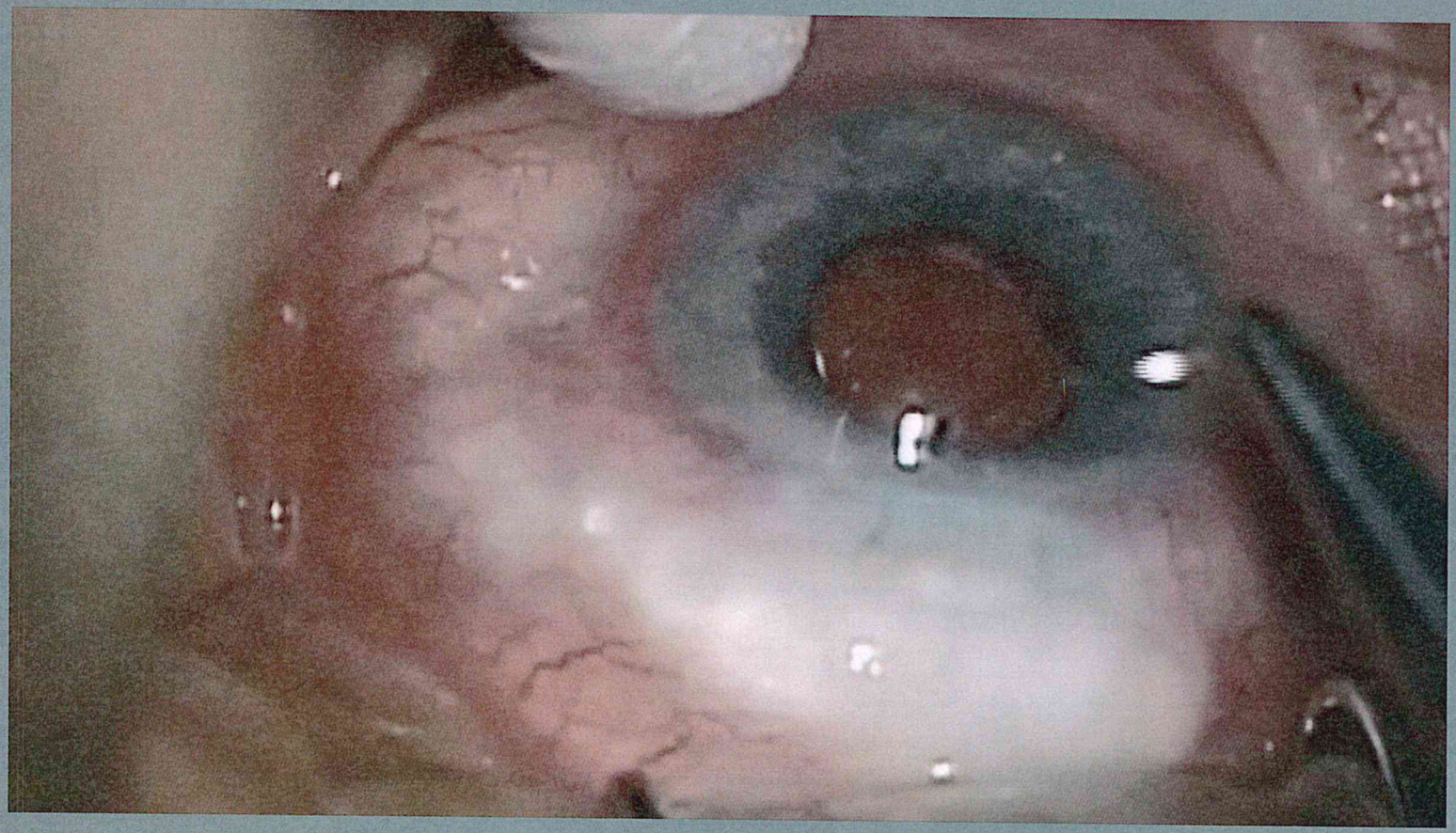
Exam

- Va 20/50, 20/40
- IOP: 8/12

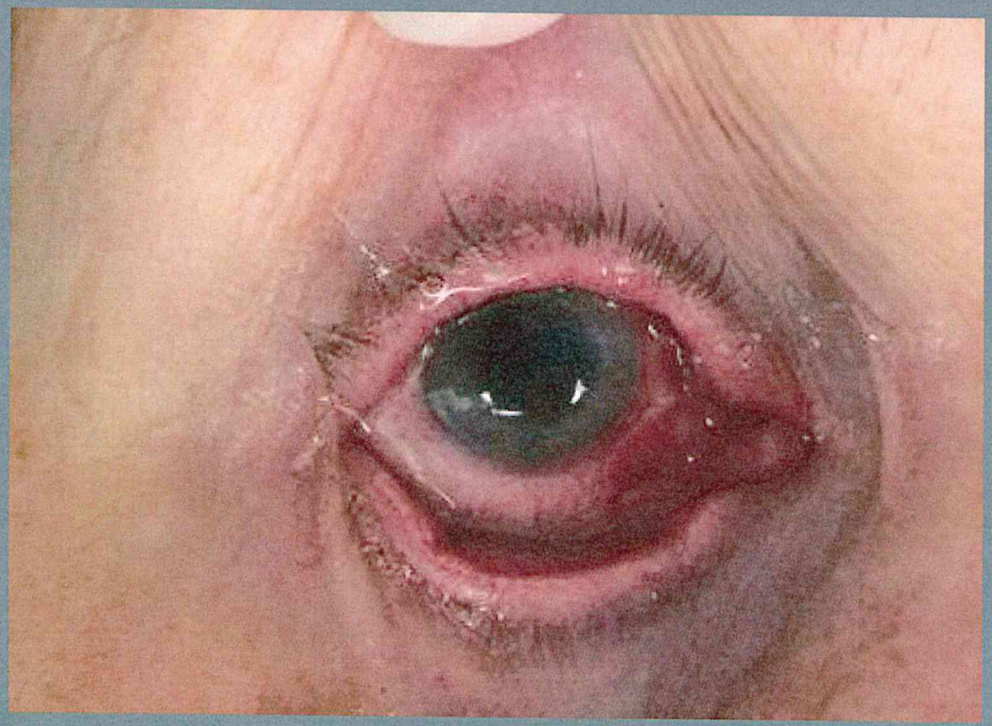
SLE and DFE

- SLE
  - LLL – wnl ou
  - S/C – large bleb OD, spreads nasally
  - K – clear ou
  - AC – Deep and Quiet ou
  - I- iridectomy OD superiorly with transillumination defects OD
  - Lens- sulcus IOL piggyback on IOL in the bag
  - AV – wnl ou

- DFE
  - c/d right eye: 0.9, notched inferior rim
  - c/d left eye: 0.6
  - Macula – epiretinal membrance with pseudohole OD + drusen OU
  - Vessels - wnl
  - Periphery – wnl



Preop



Immediate Postop

Learning points

- Nasal blebs are cosmetically unpleasing to patients
- Multiple methods have been used to revise a bleb
- Be cautious around the flap and the medial rectus

Thank You

- USC Roski glaucoma department
  - Dr. Rohit Varma
  - Dr. Grace Richter
  - Dr. Alena Reznik

- Wilmer Eye Institute Glaucoma Center for Excellence
  - Dr's Quigley, Jampel, Ramulu,

Sahar Bedrood, M.D., Ph.D.

4463 Chevy Chase Drive La Canada, CA 91011
818-744-5545 ◆ saharbedrood@gmail.com

Education/Training

2015-2016	Glaucoma Fellowship Wilmer Eye Institute, Johns Hopkins Hospital, Baltimore MD
2012- 2015	Ophthalmology Residency Training USC Roski Eye Institute, University of Southern California, Los Angeles, CA
2011-2012	Internship in Internal Medicine Huntington Memorial Hospital, Pasadena, CA
2003-2011	Doctor of Medicine/Doctor of Philosophy, Biochemistry and Molecular Biology Keck School of Medicine of the University of Southern California, Los Angeles, CA
1998-2003	Bachelor of Science, Biochemistry and Chemistry Minor- English Literature University of California Los Angeles, Los Angeles, CA

Research Experience

2015- 2016 Dr. Harry Quigley, Professor of Ophthalmology
Wilmer Eye Institute, Glaucoma Center of Excellence

- *Fellowship research comparing the three-dimensional topography of the anterior lamina cribrosa in glaucoma versus normal patients using SD-OCT*

2010- 2011 Dr. Alfredo Sadun, Professor of Ophthalmology
Doheny Eye Institute/UCLA
Keck School of Medicine, University of Southern California

- *Post-doctoral research, studying the morphological changes in the optic nerve of mouse models of optic neuropathies*

2005- 2009 Dr. Ralf Langen, Professor of Biochemistry
Zilkha Neurogenetic Institute
Keck School of Medicine, University of Southern California

- *Doctoral degree in Biochemistry and Molecular Biology, studying structural features and biological modifiers of islet amyloid polypeptide protein found in Type II diabetes patients*

1999-2002 Dr. Louis Ignarro, Nobel Laureate in Physiology or Medicine 1998, Professor
Department of Pharmacology and Medicine, University of California Los Angeles

- *Studied the physiological and pharmacological applications of nitric oxide as it relates to cardiovascular medicine*

Awards and Honors

2015-2016	Society of Heed Fellows Fellowship Award
2011	National Eye Institute 2011 ARVO Travel Grant Award

2011	Doheny Eye Institute ARVO 2011 Basic Science Award
2009	*P.E.O. Scholar Award*- recipient of $15,000 international award for women pursuing doctoral degrees
2009	*Order of the Areté* – USC's highest honor accorded to graduate students
2008	*Remarkable Women of USC Award* – for significant contributions to USC, commitment to students, women's issues and community involvement

Leadership and Service

2007-2009	Health Science Campus Chair and Executive Board Member of Graduate and Professional Student Senate (GPSS)
2004-2005	Co-Director, Bravo High School Mentorship Program Co-Director of Mentorship Program, Salerni Collegium
2005-2006	Participant, *Healing Hearts Across Borders* - medical missions to Mexico

Publications, Patents and Presentations

Provisional Patent, USC-File 09-369, **Bedrood, S.**, Langen, R., Haworth, I. Three-dimensional structure of Islet Amyloid Polypeptide Fibrils, issued April 2009

Quigley, H., Arora, K, Idrees,S., Solano, F., **Bedrood, S.,** Lee, C., Jefferys, J, Nguyen T Biomechanical Responses of Lamina Cribrosa to Intraocular Pressure Change Assessed by Optical Coherence Tomography in Glaucoma Eyes. *IOVS,* [in review]

Bedrood, S., Chopra, V., Alasil, T., Lin, C., Dustin, L., Varma, R., Francis, B., Comparison of Non-staged (complete) versus Two-Stage Baerveldt Aqueous Shunt Implantation in Patients with Advanced Glaucoma. *J Clin Exp Ophthalmol, 2014 Nov 29;* 5: 372

Bedrood, S., Li, Y., Isas, J.M., Hegde, B.G., Baxa, U., Li, Y., Haworth, I.S., Langen, R., Fibril Structure of Human Islet Amyloid Polypeptide. *The Journal of Biological Chemistry,* 2012 Feb 17; 287(8):5235-5241

Daval, M.*, **Bedrood, S.***, Gurlo T., Huang, C.J, Costes S., Butler, PC., Langen, R. The Effect of Curcumin on Human Islet Amyloid Polypeptide Misfolding and Toxicity. *Amyloid.* 2010 Sep;17(3-4):118-28

Bedrood, S. Jayasinghe, S., Sieburth, D., Chen, M., Erbel S., Butler PC, Langen R., Ritzel RA., Annexin A5 directly interacts with amyloidogenic proteins and reduces their toxicity. *Biochemistry.* 2009 Nov 10;48(44):10568-76

Ignarro, L., Sisodia, M., Trinh K., **Bedrood, S.**, Wu, G., Hua W., Buga, G., Nebivolol inhibits vascular smooth muscle cell proliferation by mechanisms involving nitric oxide but not cyclic GMP, *Nitric Oxide: Biology and Chemistry.* vol 7, issue 2, September 2002, pp 83-90

Bedrood, S. Jayasinghe, S., Langen, R. Identifying Structural Features of Islet Amyloid Polypeptide Using Site-Directed Spin Labeling. *Journal of Investigative Medicine.* 2006 Jan; 54(1):S90

Comparison of corneal specular microscope features in pediatric glaucomatous eyes versus non-glaucomatous eyes. ARVO 2015 Denver, CO.

Effect of Early Aqueous Tube Shunt Placement in the Management of Neovascular Glaucoma. ARVO May 2015 Denver, CO

Clinical Outcomes of Anterior Chamber vs. Scleral fixated intraocular lenses in complicated cataract surgeries performed by Ophthalmology residents. ARVO May 2015 Denver, CO

Combined Trabectome and Cataract Surgery versus Combined Trabeculectomy and Cataract Surgery in Open-Angle Glaucoma. AAO 2014 Chicago, Il

Characteristics of epiretinal membranes which influence post-surgical visual outcomes. ARVO 2013 Seattle, WA

A Morphological Study of the Optic Nerve in a Mito-Mouse Model Carrying an ND6 Point Mutation. ARVO 2011 Paper Presentation Fort Lauderdale, FL

Languages
Farsi, Spanish

Activities/Hobbies
Indoor cycling (spinning), yoga, spending quality time with my family