Optometric Education Consultants

Challenging and Controversial Topics in Glaucoma

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DISCLOSURE

***Joseph Sowka, OD* is/ has been a Consultant/ Speaker Bureau/ Advisory Board member for Novartis, Ocular Therapeutix, Allergan, Glaukos, Zeiss, and B+L. Dr. Sowka has no direct financial interest in any of the diseases, products or instrumentation mentioned in this presentation. He is a co-owner of Optometric Education Consultants**

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The ideas, concepts, conclusions and perspectives presented herein reflect the opinions of the speaker; he has not been paid, coerced, extorted or otherwise influenced by any third party individual or entity to present information that conflicts with his professional viewpoints.

Disclosures- Greg Caldwell, OD, FAAO

- Will mention many products, instruments and companies during our discussion
  - I don't have any financial interest in any of these products, instruments or companies
- Pennsylvania Optometric Association –President 2010
  - POA Board of Directors 2006-2011
- American Optometric Association, Trustee 2013-2016
  - Thank you to the members and those who join
- I never used or will use my volunteer positions to further my lecturing career
- Lectured for: Shire, BioTissue, Optovue
- Advisory Board: Allergan
- Envolve: PA Medical Director, Credential Committee
- He is a co-owner of Optometric Education Consultants

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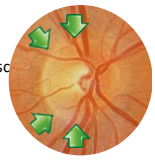
What do you do when you see a disc hemorrhage?

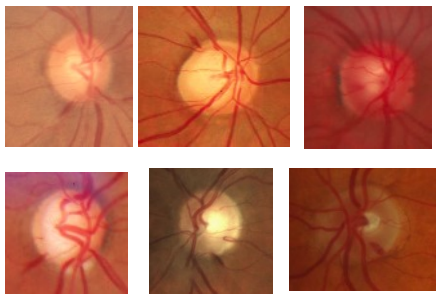
Not all hemorrhages of the disc are disc hemorrhages.

Disc Hemorrhages

- Inferior, inferior temporal, superior, and superior temporal regions of the disc are most susceptible and account for virtually all true glaucomatous disc hemorrhages
- Typically occurs where notches and RNFL defects occur- likely a mechanical shifting of tissues.

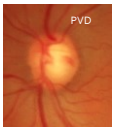


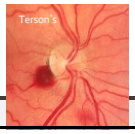
Hemorrhages at other areas of the disc (nasal and temporal) tend to not be associated with glaucoma.



Other Causes of 'Disc' Hemorrhages

- PVD
- HTN
- Anemia
- Diabetes
- Vascular occlusion
- Subarachnoid bleed
  - Terson's syndrome
    - Subretinal and intraretinal
    - May be juxtapapillary





Not all hemorrhages of the disc are disc hemorrhages. Make sure that the glaucomatous characteristics are there.

Are disc hemorrhages a risk factor for progression or actual progression?

Early Manifest Glaucoma Trial

- Disc hemorrhages- predictive of progression
- Treatment was unrelated to the presence or frequency of disc hemorrhages.
  - Disc hemorrhages were equally common in both the treated and untreated groups of patients.
  - Disc hemorrhages don't occur in all glaucoma pts.
- Disc hemorrhages cannot be considered an indication of insufficient IOP-lowering treatment,
  - Glaucoma progression in eyes with disc hemorrhages cannot be totally halted by IOP reduction.

Ocular Hypertension Treatment Study

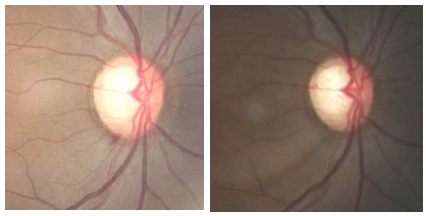
- The occurrence of a disc hemorrhage increased the risk of developing POAG 6-fold in a univariate analysis and 3.7-fold in a multivariate analysis that included baseline factors predictive of POAG
- Occurrence of an optic disc hemorrhage was associated with an increased risk of developing a POAG end point in participants in the OHTS
  - However, most eyes (86.7%) in which a disc hemorrhage developed have not experienced a POAG end point to date

Ocular Hypertension Treatment Study

- Stereophotography-confirmed glaucomatous optic disc hemorrhages were detected in 128 eyes of 123 participants before the POAG end point
- Twenty-one cases (16%) were detected by both clinical examination and review of photographs, and 107 cases (84%) were detected *only by review of photographs*

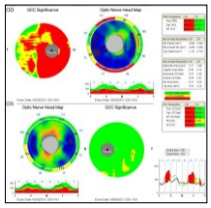
Ocular Hypertension Treatment Study

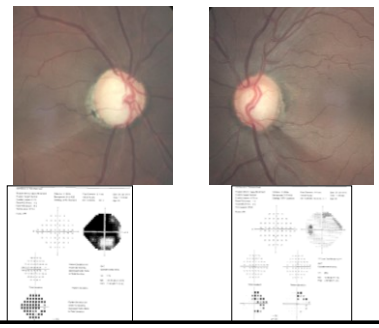
- Review of stereophotographs was more sensitive at detecting optic disc hemorrhage than clinical examination.
- Occurrence of an optic disc hemorrhage was associated with an increased risk of developing a POAG end point in participants in the OHTS
  - However, most eyes (86.7%) in which a disc hemorrhage developed have not experienced a POAG end point to date



55 YOM

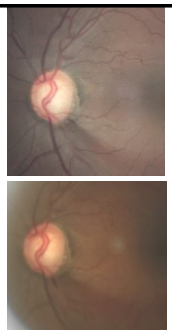
- 2012 presents without complaints
- BCVA 20/20 OD, OS
- IOP:
  - OD: 27 mm; 30 mm
  - OS: 15mm; 15 mm
- CCT: 536; 531



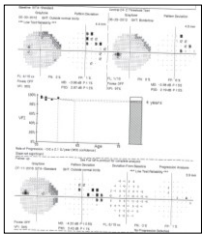


55 YOM

- Treatment initiated
  - IOP drops to mid teens OU
- Optic disc change OS noted 4/14
- Therapy amplified
- 7/15: latanoprost and dorzolamide/timolol FC OU
- IOP: 10 mm OU
- CCT: 536; 531



2012



2016

So what do I do when I see a disc hemorrhage?

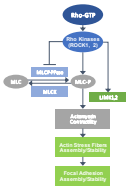
- (Treated) IOP high teens:
  - Progression documented- increase therapy
  - Risk of visual disability- increase therapy
  - None of the above: increase therapy or monitor for progression then increase therapy
- (Treated) IOP low teens
  - Monitor for progression (if safe)- no change
  - Progression documented or risk visual disability
    - ? Therapy increase
    - Equal risk of blindness from disease or treatment

What are the new medications and where do they fit?

Rho Kinase (ROCK) Inhibition

New Development in IOP Reduction

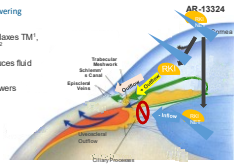
- Rho activation increases contractility of TM cells
  - Reduces outflow of aqueous humor
- Rho kinase inhibition relaxes TM cells
  - Reduces actin stress fibers/focal adhesions
  - Increases outflow of aqueous humor
- Rho kinase inhibition may also:
  - Increase ocular blood flow
  - Increase retinal ganglion cell survival



Uehata M, et al. Nature 1997;389:990-994
Honjo A, et al. Graefes Arch Clin Exp Ophthalmol. 2018;256(1):51-59
Wang RK, Chang RT. Clin Ophthalmol 2018;12:869-880

Netarsudil ophthalmic solution 0.02% (Rhopressa- ROCK-NET Inhibitor) Triple-Action

- 3 Identified IOP-Lowering Mechanisms
- ROCK inhibition relaxes TM[1], increases outflow[1,2]
- NET inhibition reduces fluid production[2]
- ROCK inhibition lowers Episcleral Venous Pressure (EVP)[3]



1. Wang RK, Chang RT. An emerging treatment option for glaucoma: Rho kinase inhibitors. Clin Ophthal 2016;8:899-905.
2. Wang RF, Williamson JE, Kopczynski C, Serle JB. Effect of 0.04% AR-13324, a ROCK and creatopyrimine transporter inhibitor, on aqueous humor dynamics in normotensive monkey eyes. J Glaucoma 2015; 24:532-6.
3. Kiel JW, Kopczynski C. Effect of AR-13324 on episcleral venous pressure (EVP) in Dutch-Belted rabbits. ARVO 2016. Abstract 2900

Netarsudil ophthalmic solution 0.02% Rhopressa ™

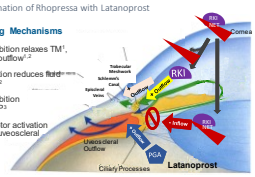
- In two phase III studies, more than half of patients experienced conjunctival hyperemia compared to 8% to 10% of timolol patients.
  - **More complaints of eye redness with Rhopressa.**
- 9% and 5% of Rhopressa once-daily patients reported corneal deposits (vortex keratopathy) across the two phase III studies compared to 0.4% and 0% of the timolol patients.
- Blurry vision was reported by 7% and 5% of Rhopressa patients compared to 3% and 0.5% of timolol patients in the studies.

Rocklatan™ (netarsudil/latanoprost ophthalmic solution) 0.02%/0.005%

Fixed Combination of Rhopressa with Latanoprost

4 Identified IOP-Lowering Mechanisms

- ROCK inhibition relaxes TM[1], increases outflow[1,2]
- NET inhibition reduces fluid production[2]
- ROCK inhibition lowers EVP[3]
- PGA receptor activation increases uveoscleral outflow[4]



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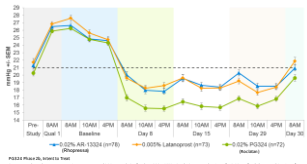
2. Wang RF, Williamson JE, Kopczynski C, Serle JB. Effect of 0.04% AR-13324, a ROCK and norepinephrine transporter inhibitor, on aqueous humor dynamics in normotensive monkey eyes. J Glaucoma 2015; 24(3):214-9.
3. Kiel JW, Kopczynski C. Effect of AR-13324 on episcleral venous pressure (EVP) in Dutch-Belted rabbits. IMVS 2016. Abstract 2982
4. Latanoprost prescribing information

Rocklatan Achieved Statistical Superiority Over Individual Components at All Time Points (p<0.001)

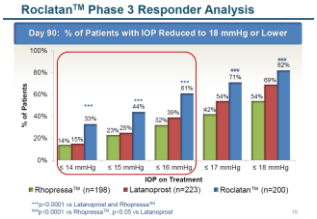
- Mean IOP at Each Time Point (Primary Efficacy Measure)



PG324 Phase 2b, Intent to Treat

Source: Bacharach J, Lewy R, Ramirez N, Kopczynski CC, Novack GD for the PG324-CS201 Study Group. Evaluation of PG-324, a fixed dose combination of AR-13324 and latanoprost, in patients with elevated intraocular pressure in a double-masked, randomized, controlled study. American Glaucoma Society 2015 (in press).

Rocklatan™ Phase 3 Responder Analysis



Rocklatan 12-Month Safety and Efficacy Highlights

- Safety results for Rocklatan for the 12-month period were consistent with those observed for the 90-day efficacy period in the trial.
- Rocklatan IOP lowering exceeded that of both latanoprost and Rhopressa in a range from 1 to 3 mmHg.
  - Levels of IOP lowering were consistent with those observed in the Mercury 1 and Mercury 2 90-day efficacy results for all arms of the study.
  - Rocklatan also demonstrated consistent levels of IOP lowering across the 12-month study period.
- Most common adverse event for Rocklatan was conjunctival hyperemia, (60 percent of patients-considered mild), petechiael conjunctival hemorrhages (often not noticed by patients), and vortex keratopathy (reversible).

VYZULTA™ (latanoprostene bunod ophthalmic solution, 0.024%)

- First prostaglandin analog with one of its metabolites being nitric oxide (NO)
- QD dosing
- Dual mechanism of action
  - metabolizes into two moieties, latanoprost acid, which primarily works within the uveoscleral pathway to increase aqueous humor outflow, and butanediol mononitrate, which releases NO to increase outflow through the trabecular meshwork and Schlemm's canal.

latanoprost bunod (LBN) Vyzulta™

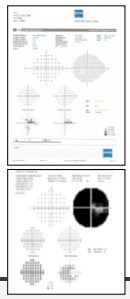
- Nitric oxide is an important physiological signaling molecule, which plays a key role in IOP regulation in healthy eyes.
- LBN/Vyzulta is thought to increase aqueous humor outflow by acting on both the uveoscleral (non-conventional) pathway via latanoprost acid, and trabecular meshwork and Schlemm's canal (conventional pathway) via nitric oxide signaling.
- LBN showed a reduction in mean IOP of 7.5 to 9.1 mmHg from baseline between 2 and 12 weeks through Phase 3 studies and 1.2 mm greater decrease on average than latanoprost.

Pearls on Static Visual Fields

- Most visual fields test 0-51 decibels
  - 41-51 decibels is outside human vision
- 1 diopter of refractive blur in undilated patient
  - A little more than 1 decibel of depression of the hill of vision
    - With Goldmann III stimulus
- Leave cylindrical errors of less than 2 diopters uncorrected
  - Adjusted with spherical equivalent
  - Above 2 diopters correct the astigmatism with trial lens
- Background of a visual field illuminated (31.5 apostilbs)
  - Minimum brightness for photopic or daylight
  - Cones are isolated, test photopic system
    - More on contrast, less on absolute brightness
  - Changes in pupil size, crystalline lens color and transparency have less effect on result

24-2 vs 30-2 vs 10-2 Static Visual Field

- 30-2 tests 76 locations
- 24-2 tests 54 locations
  - Tests 30 degrees nasal
  - Little diagnostic information lost in 24-2
  - Time is saved; Fewer trial lens and lid artifacts
- 24-2 has become the VF for glaucoma
  - Only a small percentage of glaucomatous defects occur in the peripheral visual field alone
  - Only down side, 30-2 can sometimes find progression earlier due to more test points
- 10-2: Measures 10 degrees temporally and nasally and tests 68 points. Used for macula, retinal and neuro-ophthalmic conditions and advanced glaucoma
- At any sign of paracentral depression on a 24-2 field, 10-2 fields should be done additionally.
- Clinical Pearl: Once Mean Deviation (MD) drops below -14 dB, the patient is not likely safe to drive. Once MD drops below -22 dB, the patient meets SSA criteria for visual disability.



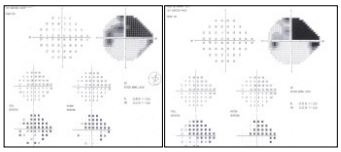
10-degree visual fields in advanced glaucoma: How to interpret and how to judge progression

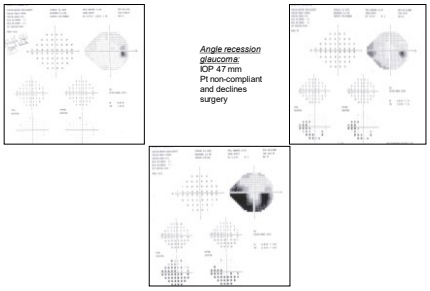
- Paracentral scotomas (5-15^0)
  - A relatively small visual field abnormality (a cluster or a single point) in the nerve fiber bundle region that is generally not contiguous with the blind spot or the nasal meridian. In particular, it does not involve points outside 15 degrees that are adjacent to the nasal meridian.
- Central scotomas
  - Visual field loss that is predominantly in the macular region. The foveal threshold must have a p < 5% value. Can be associated with a single hemifield and paired with another defect.

Visual Field Progression: Three ways fields get worse

- Deepening of an existing defect
- Extension of an existing defect
- Development of a new defect in previously normal area

Visual Field Progression





Glaucoma Progression

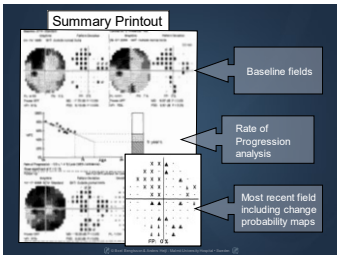
- Progression can be categorized as event analysis or trend analysis
  - Event analysis- compares baseline to most recent data; change as dictated by criteria has occurred or not.
  - Trend analysis- looks at the significance of rate of change over time.
    - Identifies progression by looking at patient behavior over time. Uses all data points and a linear regression formula
      - Weakness- progression is not necessarily linear
- Event analysis and trend analysis are complementary. Without event analysis, we have no early detection of glaucoma or early detection of progression in patients having glaucoma diagnoses. Without rate analysis, we have no ability to decide if detected changes are clinically significant.

Glaucoma Progression

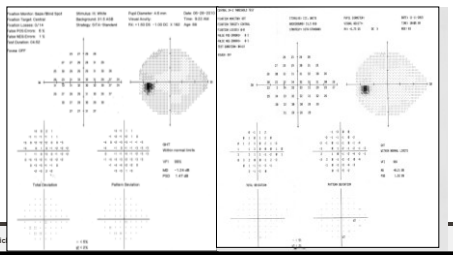
- Event analysis- identify statistically significant events.
  - Event analyses can be used to detect glaucoma before the patient is outside the normal range on RNFL thickness or perimetry
- Trend analysis try to quantify the rate of progression with the goal of determining if that rate is clinically significant
  - The rate can be statistically significant but not be clinically significant.
  - Conversely, we can see rates that clearly are clinically significant, even clinically threatening, and not yet have enough data to say today that we have statistical significance.
-

Guided Progression Analysis: GPA

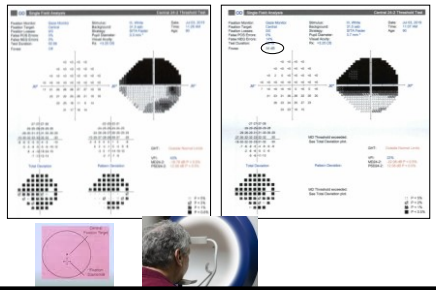
- Designed to help identify clinically significant progression of visual field loss in patients with glaucoma
- Highlights changes from selected baseline examinations that are larger than typical clinical variability in patients with similar degrees of glaucoma.
- Identifies consistent and repeated patterns of loss

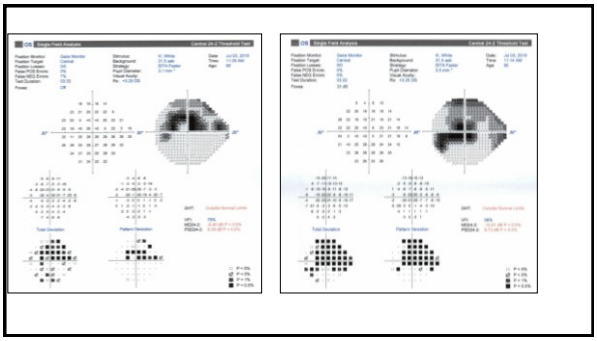


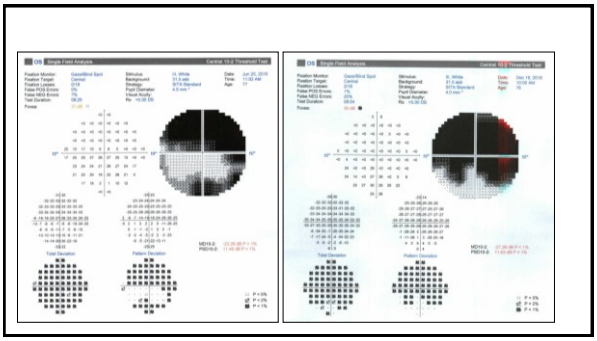
Is This Field Progressing?



Is this patient getting worse?







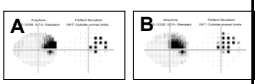
What is more important? Target IOP or Peak IOP?

Target IOP

- A clinical GPS
- How do you get there if you don´t know where you are going?
- Is it important? Yes
- How do I figure it out? That's not so easy
- To get where you're going, don't forget where you have been.
  - Peak IOP
- Should we do it? Do we do it?

Target IOP

- Practice guidelines recommend target IOP
  - Should be written in chart
  - Rarely done so
- Determining initial target pressure
  - Existing disc damage
  - Extent of field damage
  - Risk of imminent/ future functional disability
    - Rate of change impacts target IOP
  - Patient age and life expectancy
  - *Peak IOP*



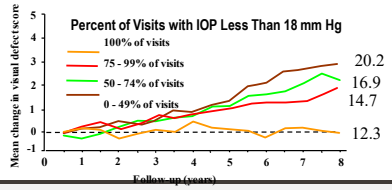
"B" should have lower target IOP than "A"

Setting a Target Pressure

Two Methods

- Absolute Value
  - Risk of over-treating or under-treating patients
  - What pressure is ideal for everyone?
  - *One size fits all… doesn't*
- Percentage decrease from baseline
  - What constitutes an acceptable decrease?

AGIS 7: Sustained IOP below 18 mm Hg: Positive Correlation with Stability of Visual Field



AGIS Investigators. 2000. Am. J. Ophthalmol., 130, 429-440

Percentage Decrease from Baseline

- Can the studies guide us?
- Clinical trials provide Evidence and Guidelines
  - Take care to apply the correct study to the correct population
- Understand how and why target pressures are chosen for clinical studies

Are we setting target pressures too high?

Information from Major Studies

Study	IOP reduction	Progression
• OHTS	20%	Yes
• EMGT	25%	Yes
• CNTGS	30%	Yes
• CIGTS (med)	35%	No
• CIGTS (Surg)	48%	No
• AGIS	< 18 all visits	No

- A target pressure is that pressure at which the sum of the impact of the glaucomatous vision loss upon the patient and the impact of treatment upon the patient is minimized.
- Once treatment is started, the goal is not to make the IOP 'normal', but safe for the patient.
- Demand greater reductions than before
  - 40-50% vs 20-30%, especially for advanced disease/ risk of visual disability.
- Am I at medicolegal risk if I don't have target in chart? No
- Am I at medicolegal risk if I have target in chart and I don't reach it? No

Target IOP: A best guess based upon:

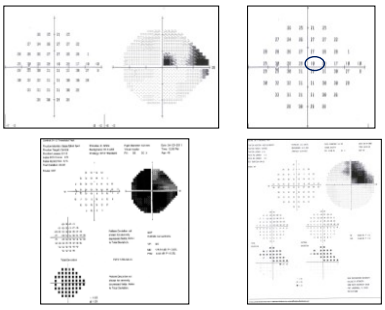
- Age and longevity
  - Don't be age-prejudiced
- Degree of optic nerve damage
- Degree of visual field loss
- Threat to fixation and risk of disability
- IOP at which damage occurred
- Corneal thickness
- Family history of glaucoma blindness

Conclusions

- Don't over-treat those at minimal risk of vision loss
- Don't under-treat those at high risk of vision loss
- Don't focus on the IOP to the exclusion of other factors
- Remember to treat the patient, not a number

What is "Advanced" Glaucoma?

- Subjective terms- advanced/ end stage
- Central and paracentral defects
- 10-2 fields
- Loss of fixation
- Loss of Snellen acuity
- Loss of unassisted ambulatory ability
- Severe/ advanced:
  - MD < -12.01 db
  - Any point inside 5° degrees with threshold 0 db
- End stage: HVF not possible- attributable to central scotoma; acuity of 20/200 or worse attributable to glaucoma



Re-thinking Advanced Glaucoma Treatment

- Published guidance from National Institute for Health and Clinical Excellence (NICE) has recommended primary surgical intervention for patients presenting with advanced disease.
- No studies comparing modern medical against modern surgical interventions in patients presenting with advanced disease

Re-thinking Advanced Glaucoma Treatment

- Surgery appears to offer better IOP and visual field outcomes than medical treatment for advanced glaucoma
- Non-adherent to their medication
- Some patients will not respond adequately despite adherence
  - Result in a delay in successful IOP reduction and may result in further visual field progression

Br J Ophthalmol. 2011 Sep;95(9):1185-92

Re-thinking Advanced Glaucoma Treatment

- Surgical intervention is associated with risk and possible catastrophic loss of vision due to either early or late complications
  - Vision 'wipeout'
  - *"Doctor, I can't see your face anymore"*
- There is no evidence for clinicians to adopt the primary surgical intervention suggested by NICE. Clinicians should continue to manage patients according to their current practice until more decisive evidence becomes available.

Br J Ophthalmol. 2011 Sep;95(9):1185-92

Re-thinking Advanced Glaucoma Treatment

- *"Not all patients with advanced glaucoma will progress within the period of time that is still allotted to them to live"*
- *"Not taking into account the rate of change of the condition, and the estimated years remaining is an inappropriate approach to patient care."*
  - George Spaeth, MD

Re-thinking Advanced Glaucoma Treatment

- *"There is insufficient evidence to suggest that primary surgery would result in better clinically relevant outcomes in patients with severe glaucoma compared to standard medical therapy."*
  - Felipe Medeiros, MD

Re-thinking Advanced Glaucoma Treatment

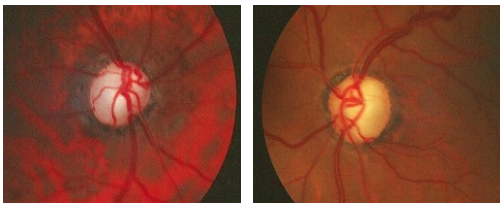
- *"In eyes with advanced glaucoma at presentation, a low IOP should be sought rapidly and efficiently, with the intent of seeking and maintaining a low target IOP."*
- *"Treatment for eyes with advanced glaucoma can be extremely effective, but only if IOP reduction is significant and long-lasting. This should dispel the notion that these patients are doomed to blindness."*
  - Jeffrey Liebmann, MD

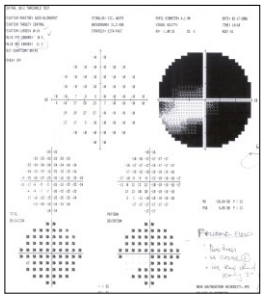
Why is the Glaucoma Advanced?

- Poor/ no access to care
- Not diagnosed
- Not aware of disease- no care
- Poor compliance/ adherence/ persistence with medications
- Poor understanding of glaucoma
- Nature of the Recession/ pandemic/ loss of income and insurance
  - Risks for the future- projections

Doug

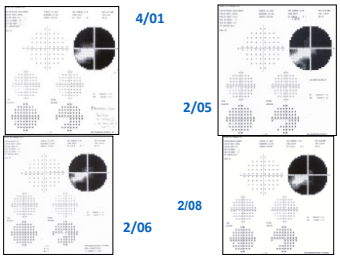
- 63 YOBM
- Knows he has POAG – doesn't follow through with treatment
  - Poor care in Caribbean
- IOP 43 mm Hg OD; 60 mm Hg OS
- Angles open by gonio OU
- Hand Motion OD, 20/40 OS
  - Small temporal island of vision OS

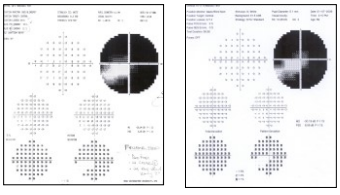




Doug

- 63 YOBM - POAG
- Medications:
  - Timolol/brimonidine FC, brinzolamide, travoprost OS; travoprost OD
- IOP: 29-34 mm Hg OD, 10-13 mm Hg OS
- Never misses appointment
- Thankful things are as goods as they are





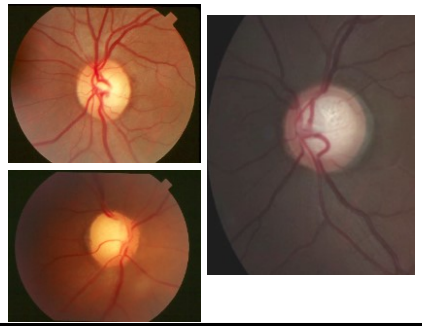
How do you manage glaucoma suspects?

Who is a Glaucoma Suspect?

- Elevated IOP/ OHTN
- Suspicious disc appearance
  - Thin rim tissue; Disc asymmetry
- Suspicious RNFL/ OCT
- Disc hemorrhage
- Suspicious visual field loss
- Family history of glaucoma
- Age
- Race
- Phakic hyperopia- angle closure suspect

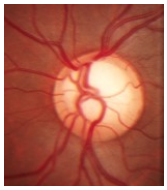
Disc Evaluation

- Size
- Rim color
- Focal rim defects (notching)
- Hemorrhages
- RNFL defects
- Parapapillary atrophy



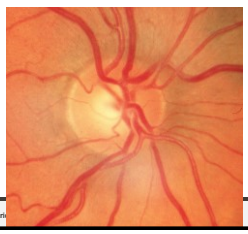
Which of these 3 patients do you most suspect has glaucoma?

Patient 1: 28 YOF



- **IOP: 11 mm**
- **CCT: 610**

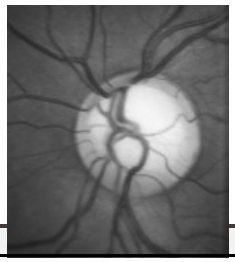
Patient 2: 56 YOM



- **IOP: 22 mm**
- **CCT: 598**

Patient 3: 64 YOF

- **IOP: 31 mm**
- **CCT: 490**



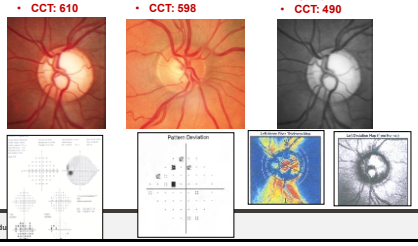
Which patient has glaucoma? 1? 2? 3?

Which patient has glaucoma? 1? 2? 3?

- **IOP: 11 mm**
- **CCT: 610**

- **IOP: 22 mm**
- **CCT: 598**

- **IOP: 31 mm**
- **CCT: 490**



Rule: When *diagnosing* glaucoma, take IOP out of the equation

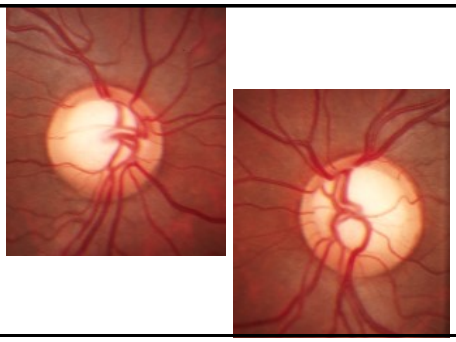
(When managing glaucoma, put IOP back into the equation…but that's another lecture.)

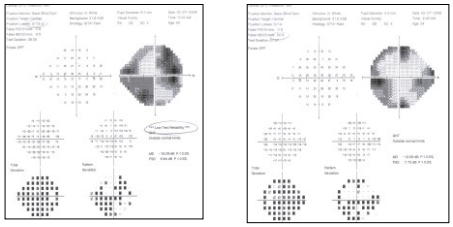
Who are the glaucoma suspects?

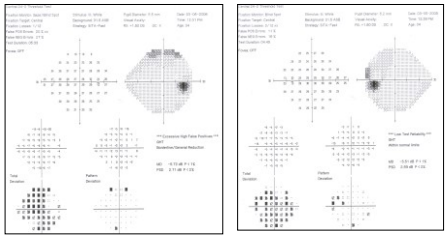
- Large cupping- normal IOP
- Large cupping- high IOP
- Normal cupping- high IOP

Is This Glaucoma?

- 34 YOHF
- "Highly suspicious" ONH OU
- IOP statistically normal
  - 13 mm Hg OU
- Average CCT
- Previously treated for NTG



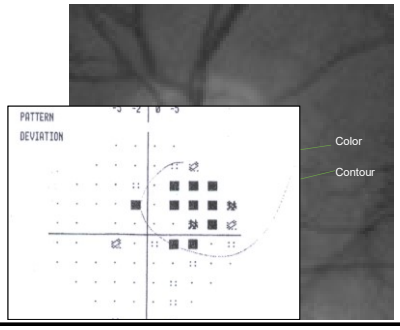




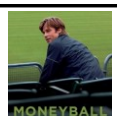
My advice to patients: If you insist on having a suspicious optic disc, you had better be a good field taker.

Is This Glaucoma?

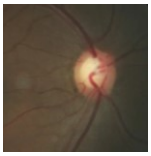
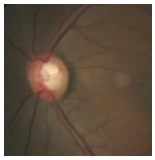
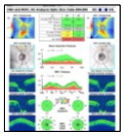
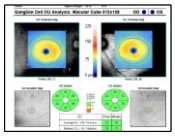
- 78 YOWM
- Annual exams with multiple doctors
- IOP ranges from 17 – 21 mm Hg
- CCT 570
- Ocular health always "normal"
- Small discs with indistinguishable cupping
  - 0.2/0.2 – 0.3/0.3



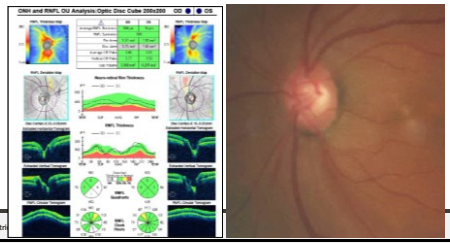
over-



"When you get the answer you want, hang up"

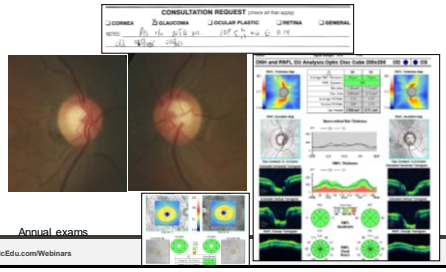
But don't under-test, either



Who are the glaucoma suspects and what do I do?

- Large cupping- normal IOP
  - Does the nerve look glaucomatous?
    - Yes- photos, fields, pachymetry, gonio, OCT
    - No- OCT- if normal-done; if abnormal- fields- if normal- done, if abnormal- monitor
- Large cupping- high IOP
  - Does the nerve look glaucomatous?
    - Yes- photos, fields, pachymetry, gonio, OCT
    - No- OCT, photos, pachymetry, fields, gonio
- Normal cupping- high IOP
  - OCT, photos, pachymetry, fields, gonio

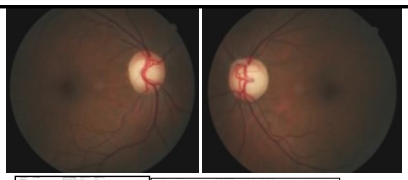
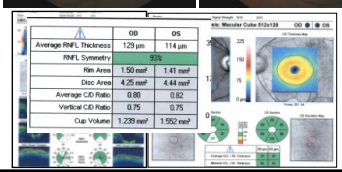
Large cupping- normal IOP



Annual exams

Large cupping- unknown IOP- diagnosed with glaucoma

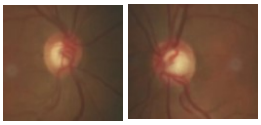
- 46 YOF
- Diagnosed and treated for glaucoma in Jamaica
- Brimonidine 0.1%; latanoprost/timolol FC OU
- IOP: 14 mm OD, 16 mm OS
- CCT: 530; 528
- 0.75/0.75 OU
- Fields unreliable- high FP

	OD	OS
Average RNFL Thickness	129 µm	114 µm
RNFL Symmetry	93%	
Rim Area	1.50 mm²	1.41 mm²
Disc Area	4.25 mm²	4.44 mm²
Average C/D Ratio	0.80	0.82
Vertical C/D Ratio	0.75	0.75
Cup Volume	1.239 mm³	1.552 mm³

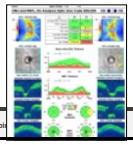
D/C all meds:
IOP: 17 mm OD, 18 mm OS

Large cupping- high IOP

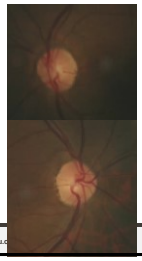


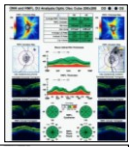
56 YOF
IOP: 24 mm OH
CCT: 550 OD, 539 OS

RTC 6 mos fields
Follow w/o treatment Q 6 mos

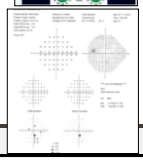


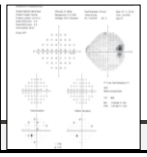
normal cupping- high IOP

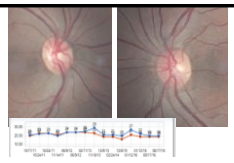




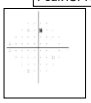
IOP: 30 mm OD, 32 mm OS
Mother + glaucoma (10-2 field)
Rx: Latanoprost OU

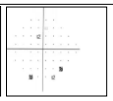


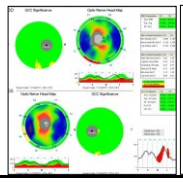


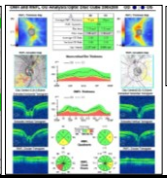


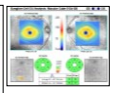
56 YOM: mother had glaucoma
CCT: 548 OU
Peak IOP: 29 mm











Make a decision! Patients shouldn't be 'glaucoma suspects' for ten years. Either they have the disease or they don't.