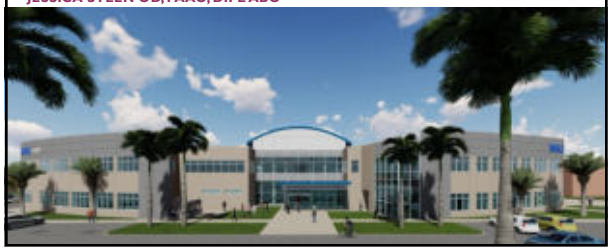
PROBLEM SOLVING AND GLAUCOMA MANAGEMENT

JESSICA STEEN OD, FAAO, DIPL ABO



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FINANCIAL DISCLOSURES

- Speaker-Carl Zeiss Meditec, Bausch and Lomb, Oyster Point Pharma, Thea Pharma, Alcon, Allergan, Iveric Bio
- Advisory Board-Bausch and Lomb, Santen, Peripherex, Ocuphire, OcuTerra, Oyster Point Pharma, Allergan, Iveric Bio, Radius XR
- Shareholder-Clearside Biomedical (<0.01% ownership)
- *All relevant relationships have been mitigated*

2

61 YEAR OLD HISPANIC FEMALE

- Primary open angle glaucoma OU diagnosed in 1998
  - At the age of 36
  - Treated with timolol 0.5% BID OU
    - IOP 18-20mmHg OD and OS; peak untreated IOP not known
- CCT 477µm OD 495µm OS

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- Hypothyroidism managed with levothyroxine
  - Multivitamin, Omega-3
- ***Not*** hypertensive

Date	Time	Temp F	Temp C	BP	Site	Cuff Size	Pulse
11/12/2020	10:01 AM			90 / 60	wrist	adult	72
06/22/2020	3:22 PM			111 / 78			84
10/28/2019	2:15 PM			104 / 66	wrist	adult	65
03/30/2016	4:05 PM			110 / 62		adult	62
05/19/2016	12:40 PM			102 / 68			66
07/14/2015				110/60			

- No family history of glaucoma
  - Mother-Alzheimer's disease

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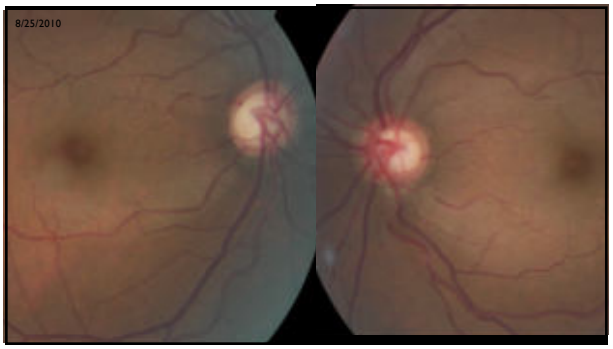

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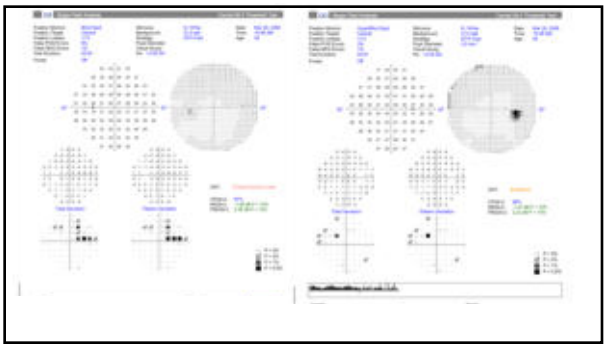

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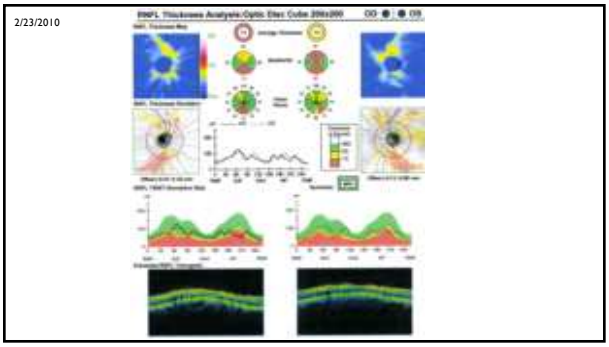

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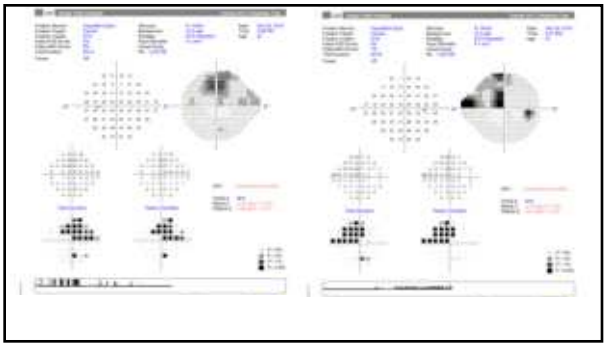

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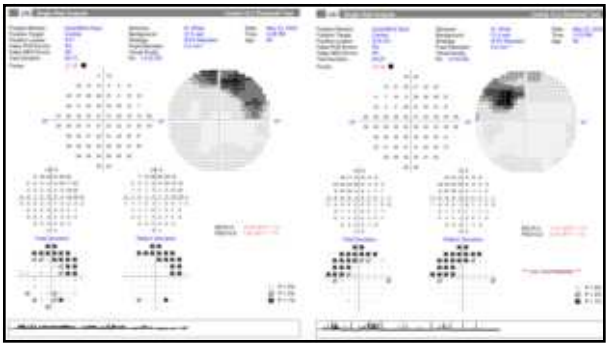

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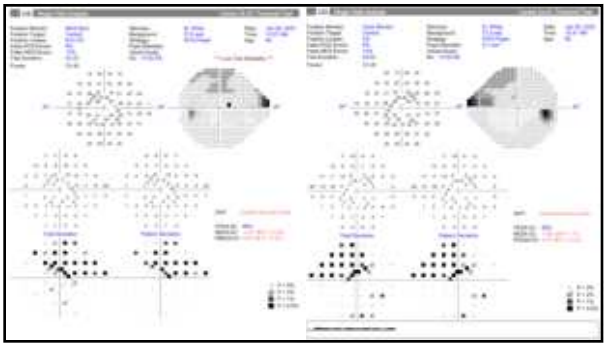

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A Comparison of the Visual Field Parameters of SITA Faster and SITA Standard Strategies in Glaucoma

- Removes 'dead time' during the test
- No blind spot, no false negatives
  - Gaze monitoring and false positives
    - Unless you manually adjust settings
- Slightly increased overall threshold sensitivity (is this bad?!)
- More difficult testing situation vs. 'positive start bias' of SITA Standard
  - No 'easy' answers
- *Clinically* equivalent to SITA Standard(?)

Lavanya R et al. A Comparison of the Visual Field Parameters of SITA Faster and SITA Standard Strategies in Glaucoma. J Glaucoma. 2020 Sep;29(9):783-788.

Thulasidas M, Patyal S. Comparison of 24-2 Faster, Fast, and Standard Programs of Swedish Interactive Threshold Algorithm of Humphrey Field Analyzer for Perimetry in Patients With Manifest and Suspect Glaucoma. J Glaucoma. 2020 Nov;29(11):1070-1076.

24-2C Testing pattern: an additional 10 points in the paracentral area overlaid on the the 24-2 pattern

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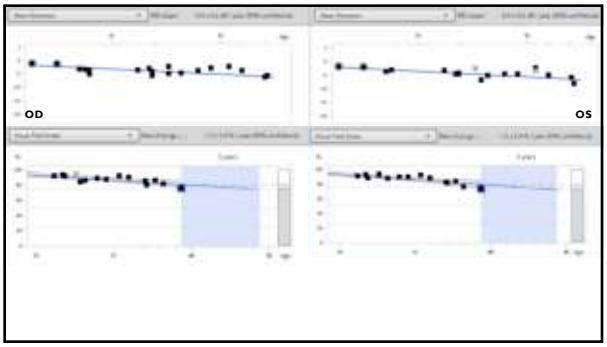

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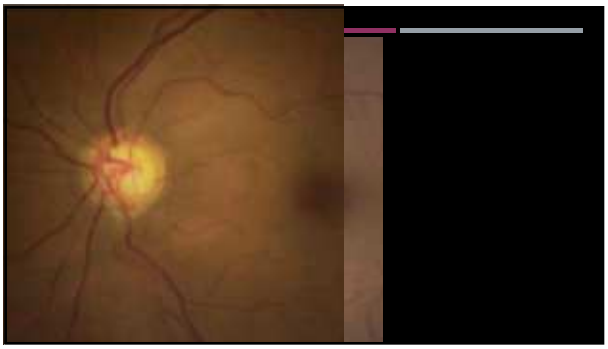

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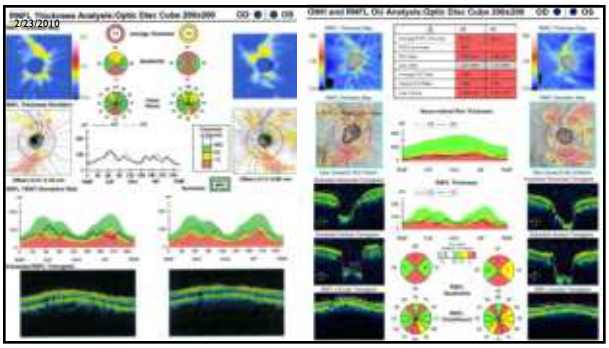

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HAS THIS PATIENT'S DISEASE PROGRESSED?

YES...

Therapy was escalated appropriately over the last 20 years.

But. There is evidence of progression with IOP 8-10mmHg

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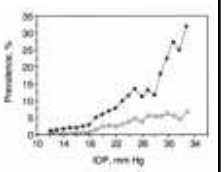
Intraocular Pressure

- This is the most significant risk factor overall
- IOP which is statistically abnormal is not necessary physiologically abnormal for an individual eye
- Conversely, IOP that is statistically normal is not necessarily physiologically normal for an individual eye
- **There is no clinically useful level of IOP to differentiate all normal from all people with glaucoma**



African American subjects, n = 4674 (closed circles; Caucasian subjects, n = 5700 (open circles)

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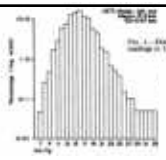
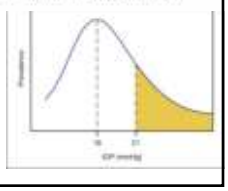


INTRA-OCULAR PRESSURE, GLAUCOMA, AND GLAUCOMA SUSPECTS IN A DEFINED POPULATION*

- "Normal tension glaucoma" "Primary open angle glaucoma with statistically normal pressure"
- "Average" intraocular pressure is 15-16mmHg (SD = 2.5mmHg)
- "Normal" range 11-21mmHg
  - Based on a population-based study in England of nearly 2000 white males over 40 years of age



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BUT—THE PRESSURE IS LOW

- But, the cornea is thin.
- Central corneal thickness impacts applanation tonometry measurement
  - Can lead to misdiagnosis or treatment changes
- Thin corneas are a risk factor for development of glaucoma in patients with ocular hypertension (OHTS)

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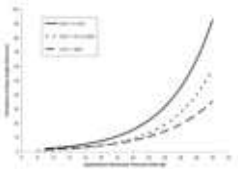
CENTRAL CORNEAL THICKNESS

- Persons with thin CCT had a significantly higher prevalence of OAG than did those with normal or thick CCTs at all levels of IOP
- **CCT is an important independent risk factor for the prevalence of glaucoma**
  - Los Angeles Latino Eye Study Group



Los Angeles Latino Eye Study, n = 5970

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Do not adjust IOP based on CCT measurements

Pachymetry measurement and conversion models may themselves be error sources

It's not that simple

No validated algorithm to correct IOP based on CCT

No proven association of CCT and any other structural abnormality

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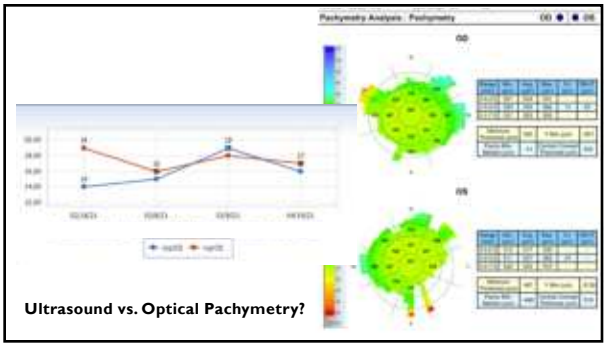

Ultrasound vs. Optical Pachymetry?

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The Cupped Disc

Who Needs Neuroimaging?

Conclusions: Anterior visual pathway compression is an uncommon finding in the neuroradiologic evaluation of patients with a presumptive diagnosis of normal-tension glaucoma. Younger age, lower levels of visual acuity, vertically aligned visual field defects, and neuroretinal rim pallor may increase the likelihood of identifying an intracranial mass lesion. *Ophthalmology* 1998;105:1866–1874

- "Nothing notches a nerve like glaucoma"
- Disc hemorrhage, vertical cup elongation

I appreciate the opportunity to discuss this article because I feel so passionately about its conclusion. I agree with the authors: if it looks like normal-tension glaucoma, you do not have to do neuroimaging to sleep at night.

Richard Mills MD, MPH

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THOUGHTS?

- #1 **Adherence.**
- What is the impact of:
  - Central corneal thickness
    - Corneal hysteresis
    - Corneal biomechanics
    - Laminar biomechanics
  - Disease mechanism
    - Mechanical
    - Vascular dysfunction or IOP-independent factors
      - *Glaucoma is a neurodegenerative disease*

PGAs are associated with the best adherence at FDA approved dosing

Corneal Biomechanics and Visual Field Progression in Eyes with Seemingly Well-Controlled Intraocular Pressure

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What other risk factors exist?

Elevated IOP
Older age
Black or African race or Latino or Hispanic ethnicity
Family history of glaucoma
Thin central corneal thickness
Low ocular perfusion pressure
Myopia
Type 2 diabetes mellitus
Low systolic and diastolic blood pressure
Hypothyroidism
Migraine
Sleep apnea
Peripheral vasospasm (Raynaud's syndrome)
Cardiovascular disease
Low corneal hysteresis
Systemic hypertension
Low cerebral spinal fluid pressure
Genetics

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WHAT ELSE CAN WE DO?

- Are we missing true peak IOP?
  - Home tonometry
  - Needs to be accurate, portable, painless, relatively inexpensive, continuous, supported by software

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Glaucoma and genetics

Currently, about 296 loci have been identified (Han et al. 2023)

In most patients, complex genetics are involved

Each gene contributes a small amount of risk, but none of which cause disease on their own

- ***Direct contribution to disease development***
- ***Influence biological pathways***
- ***Contribute to other risk factors (IOP)***

Polygenic risk score; one more parameter to consider (not yet)

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Glaucoma and genetics

Polygenic risk score development using GWAS data

Diagnostic holy grail

Predict outcomes of disease

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JUVENILE OPEN ANGLE GLAUCOMA

- Developmental immaturity of the trabecular meshwork
- Essentially normal appearance by gonioscopy
  - Open anterior chamber angle without significant abnormality
- **There is no such thing as 'normal tension' JOAG**
- Often considered to be inherited as an autosomal dominant trait
- IOP rises sometime between about 2 and 16 years of age
  - Diagnosed before about 40 years of age

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GENETICS IN JOAG

- Multiple myocilin gene mutations implicated in development
  - Myocilin is found in trabecular meshwork cells, beams, and in juxtacanalicular tissue
- Myocilin-associated glaucoma: mutant protein aggregates within TM cells→leads to cell death→TM damage→high IOP→glaucoma
  - Increases resistance to outflow
- *Not all patients with SNPs in the myocilin gene develop JOAG.*
- **Family history matters**
  - Especially when it's real and close
  - **Evaluate family members: siblings, children**

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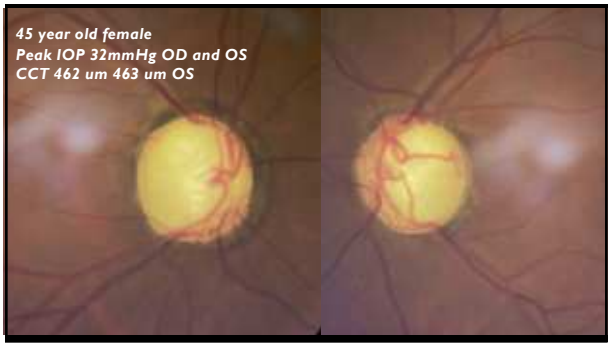

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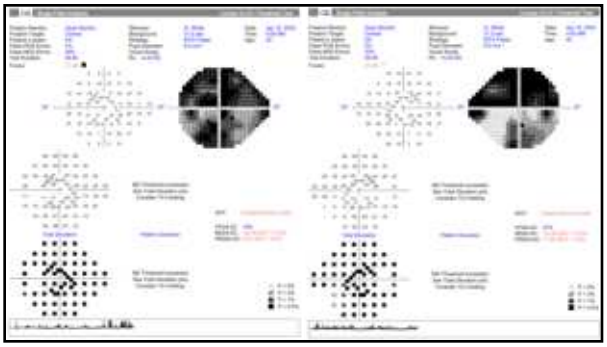

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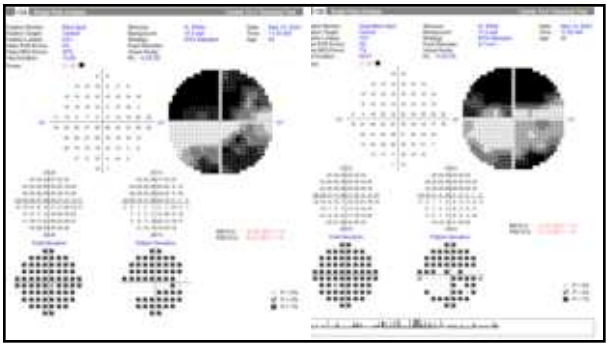

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Myocilin gene-autosomal dominant

- *90% penetrance*
- *What is the impact on the emotional aspect with genetic testing?*
- *Low vision consultation-most effective early in the disease course*

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WHAT IS 'MAX MEDICAL THERAPY?'

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What is maximum medical therapy?

It depends on what the patient can comfortably manage (tolerate)

Zero medications…6 medications…or somewhere in between

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What is maximum medical therapy?

What is the tolerability—and long-term feasibility of treatment?

Next steps?
In what time frame?

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Ocular surface disease is common

Around 60% of glaucoma patients are reported to have ocular surface disease…

Really…that's it?

It matters, but does not impact target IOP

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OCULAR SURFACE DISEASE AND GLAUCOMA

- Manage the ocular surface early
  - If patients are asymptomatic when clinical signs are apparent prior to initiation of therapy-expect symptoms to develop with therapy
- Long-term impact of benzalkonium chloride
  - Decreased density of goblet cells
    - Related to concentration of BAK

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ADDITIONAL OPTIONS

- Medication options
  - Non-BAK formulations
    - Travoprost 0.004% (Travatan Z) sofZia-teal colored cap
    - Latanoprost 0.005% ophthalmic emulsion (Xelpros) potassium sorbate
  - Preservative-free formulations
    - Tafluprost 0.0015% (Zioptan)-prostaglandin analog
    - Dorzolamide-timolol (Cosopt PF)
    - Timolol 0.25% and 0.5% (Timoptic in Ocudose)
    - Latanoprost 0.005% (Iyuzeh)

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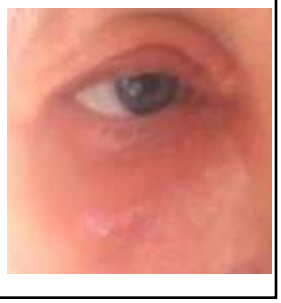
Dorzolamide-timolol BID OS x 6 years (IOP 20mmHg OD)

Peak IOP 27mmHg OD, 17mmHg OS

Variation between generic manufacturers?

Delayed hypersensitivity?



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"NEW" MEDICATION CLASSES AND EXPECTED EFFECTS

- Rho kinase family includes proteins which regulate cell shape, motility, proliferation, and apoptosis
  - **Regulate smooth muscle contraction in the trabecular meshwork and ciliary body**
- Rho kinase **inhibitors**
  - Relax trabecular meshwork cells to increase trabecular outflow
- *May also affect ocular blood blow and retinal ganglion cell survival*
  - *Role in cardiovascular procedures, corneal procedures*
  - *Role in development of fibrosis*

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RHO KINASE INHIBITOR/NOREPINEPHRINE TRANSPORT INHIBITOR

- **Increase trabecular outflow**
- **Lower episcleral venous pressure**

- Netarsudil 0.02% (Rhopressa)
  - QHS dosing
- Netarsudil/latanoprost 0.02%/0.005% (Rocklatan)

- Hyperemia-most common
  - Typically improves over time
    - *When do you see your patients back after altering medical therapy?*
- Subconjunctival hemorrhage
- Corneal verticillata

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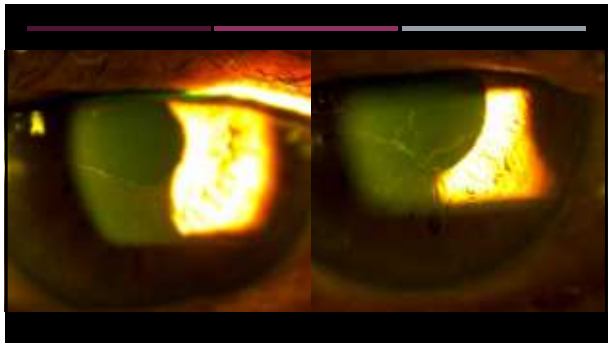

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WHERE DO RHOPRESSA & ROCKLATAN FIT IN?

- Efficacy is similar to timolol 0.5% (BID)
  - **In clinical trials
- Ideally a second line treatment
  - Seems to work better with low/moderate IOP (<25mmHg)
- Advantage of once daily dosing vs. other typical second line medication
- Cost?

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STREAMLINING MEDICAL THERAPY

- 51 year old male
- Diagnosis of pigmentary glaucoma left eye
- Presents for second opinion; he is cautious about SLT—but wishes to reduce medication load
  - Significant dryness-failed on an immunomodulator and serum tears
- Non-Hodgkin's lymphoma (2017), CMML (2023)
- History of bilateral LASIK
- Latanoprost QHS OU, dorzolamide-timolol BID OU, brimonidine BID OS
  - IOP 17mmHg OD, 21mmHg OS

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51 YEAR OLD MALE

- Gonioscopy: open to CBB 360 degrees OD and OS
- 3+ dense Sampaolesi line right eye; 4+ dense Sampaolesi line left eye
- Flat iris approach
- Peak IOP 27mmHg OD 33mmHg OS

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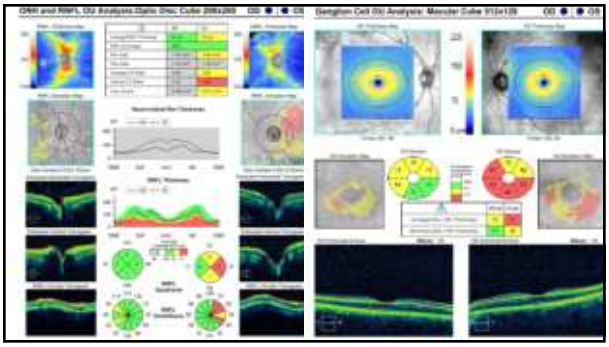

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MORE phase 4 trial

Multicenter, prospective, open-label study
No comparator; treated IOP =>20mmHg

Latanoprost, latanoprost + 1, latanoprost +2
Switch to netarsudil/latanoprost

Latanoprost → -4.9mmHg
Latanoprost + 1 → -3.6mmHg
Latanoprost +2 → -3.7mmHg

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51 year old male

Leaving South Florida for the summer in 2.5 weeks

IOP check on netarsudil/latanoprost QHS OU for 16 days OU

IOP 15mmHg OD 21mmHg OS

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"NEW" MEDICATION CLASSES

- Latanoprostene bunod 0.024% (Vyzulta)
  - Latanoprost acid + butanediol mononitrate
    - Butanediol monohydrate releases NO which increases outflow through the trabecular meshwork and Schlemm's canal
      - Relaxes trabecular beams

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The real world isn't always perfect

- In the past year, you must have failed all of the formulary alternatives for your condition AND your doctor must provide reason(s) for failure. You may not have to try these drugs if you have a Food and Drug Administration (FDA) labeled contraindication (a health condition or risk factor that may cause harm if you take a drug) that would prevent you from using them. Formulary alternatives include betaxolol ophthalmic solution, carteolol ophthalmic solution, metipranolol ophthalmic solution, levobunolol ophthalmic solution, timolol ophthalmic solution and gel, Lumigan ophthalmic drops, and bravoprost 0.004% ophthalmic drops and may require prior authorization review. ******Please note, your doctor has provided a reason you have failed or cannot take an alternative in the past year. The alternative you have tried are latanoprost ophthalmic solution, brimonidine ophthalmic solution, and dorzolamide-timolol ophthalmic solution.******.

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ALTERNATIVES

- Procedure-based options
  - SLT
  - Sustained-delivery devices
  - Surgical options

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Efficacy of Repeat Selective Laser Trabeculoplasty in Medication-Naïve Open-Angle Glaucoma and Ocular Hypertension during the LiGHT Trial

Selective laser trabeculoplasty versus eye drops for first-line treatment of ocular hypertension and glaucoma (LiGHT): a multicentre randomised controlled trial

No game-changing data

But did provide good quality evidence for what was already known

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Laser in Glaucoma and Ocular Hypertension (LIGHT) Trial

Six-Year Results of Primary Selective Laser Trabeculoplasty versus Eye Drops for the Treatment of Glaucoma and Ocular Hypertension

Conclusions: Selective laser trabeculoplasty is a safe treatment for OAG and OHT, providing better long-term disease control than initial drop therapy, with reduced need for incisional glaucoma and cataract surgery over 6 years. *Ophthalmology 2023;130:139-151* © 2022 by the American Academy of Ophthalmology. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

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What's next?

Low-energy Selective Laser Trabeculoplasty Repeated Annually: Rationale for the COAST Trial

Tony Realini, MD, MPH, Gus Gazzard, MD,†‡ Mark Latina, MD,§ and Michael Ross, MD§‖*

Estimated primary completion date: June 2027

Aims to determine optimal energy level and frequency of SLT

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Direct SLT-no lens used!

120 shots, 3ns duration, 400 micron spot size, 2 seconds (GLAUrious trial)

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Sustained release devices

Bimatoprost implant 10mcg (Durysta)

Sustained release bimatoprost
Equivalent to about 2-3 drops
Drug release complete in 3-4 months
Lasts about 6 months (may be longer)…extension of the ARTEMIS trial
Implant on day 1, week 16, week 32
Eyelash growth, redness, iris color change?

Travoprost titanium implant (iDose TR)
FDA approved December 14, 2023
Not refillable

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SURGICAL OPTIONS

- Symptoms of ocular surface disease will likely worsen after cataract surgery with or without MIGS (minimally invasive glaucoma surgery)-based procedures
  - *MIGS procedures are currently primarily approved for individuals with mild-moderate open angle glaucoma*
  - Exacerbation of inflammation
  - Epithelial disruption
  - Corneal nerve transection
  - Additional topical medications

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WHAT'S ON THE MIGS MENU?

- Non-bleb forming
  - Inflow
    - Transscleral cyclophotocoagulation
  - Outflow
    - Implant (stent)-iStent inject, iStent inject W
    - Excision of tissue-Trabectome, GATT, Kahook dual blade
    - Dilation of tissue-canaloplasty
  - Bleb-forming (*ab interno* implants)-e.g. Xen Gel Stent

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REMOVING MEDICATIONS WILL NOT ELIMINATE OCULAR SURFACE DISEASE

Cost and access are real concerns to alternative medications and procedures

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HOW DO WE TREAT THE OCULAR SURFACE?

- More topical ocular medications
  - *Is there another route of administration that may be useful?!*
- Oral medications
- In-office therapies

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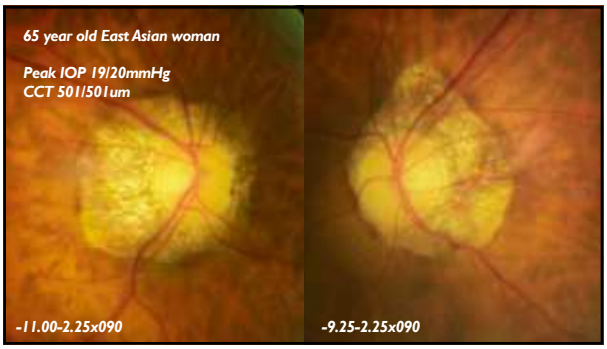

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Peripapillary atrophy or "halo"

Nerve fibers are susceptible to damage when they are passing bare choroid

These eyes may be more sensitive to pressure changes--and this halo can enlarge and change over time

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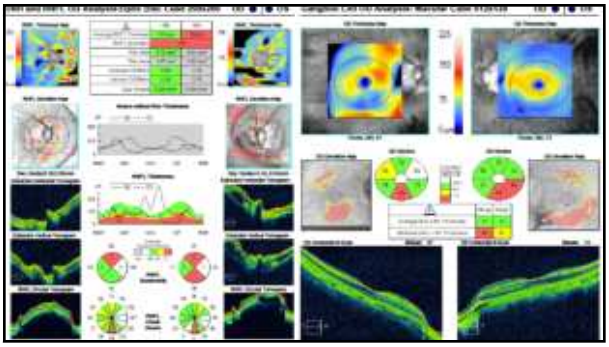

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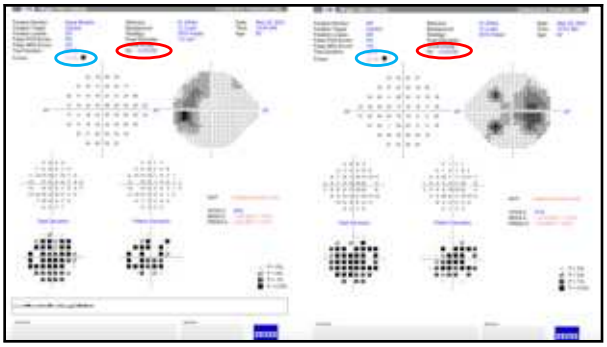

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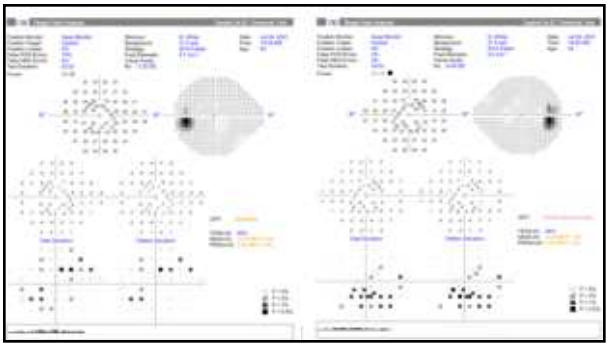

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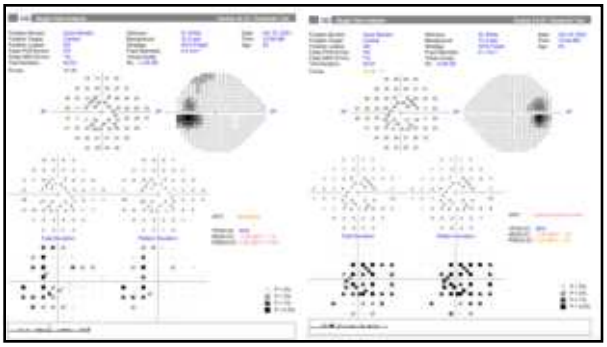

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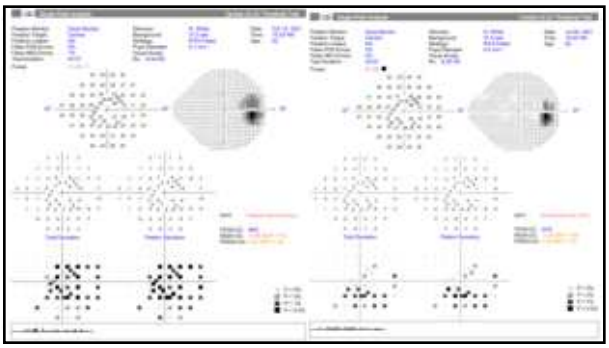

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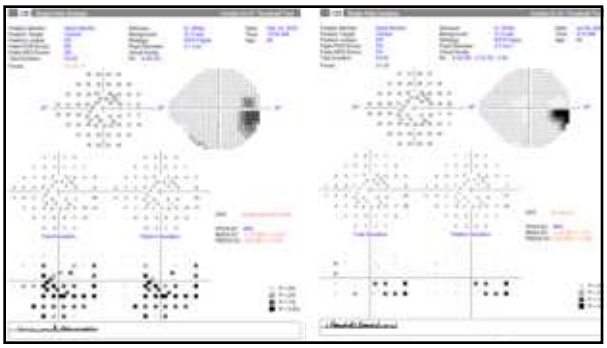

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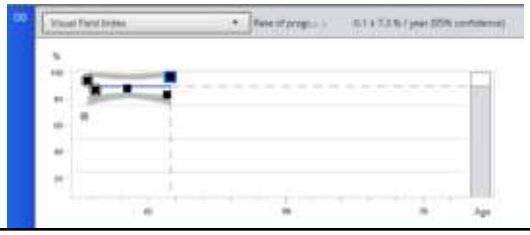
Have her fields changed?!



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*When **diagnosing** glaucoma, take IOP out of the equation*
*When **managing** glaucoma, bring IOP back in*

Ideally, 3 IOP readings prior to making or changing a treatment

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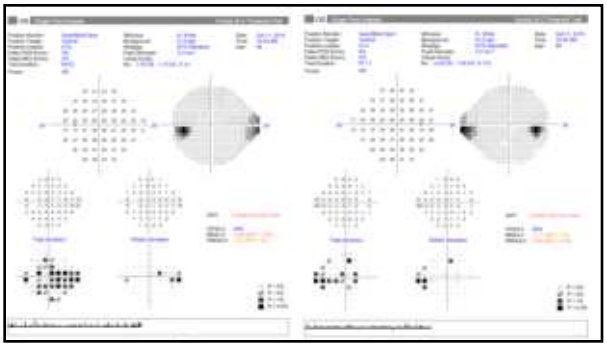

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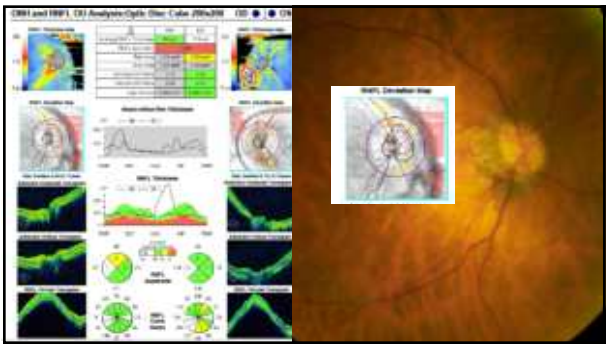

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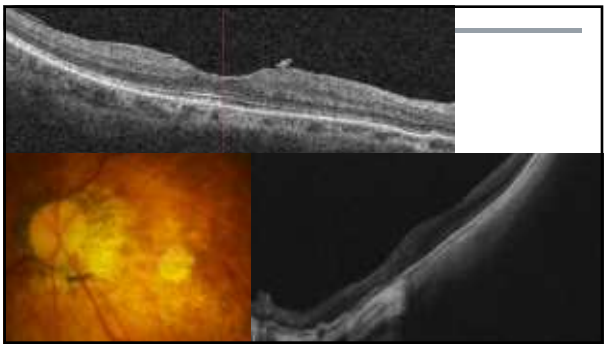

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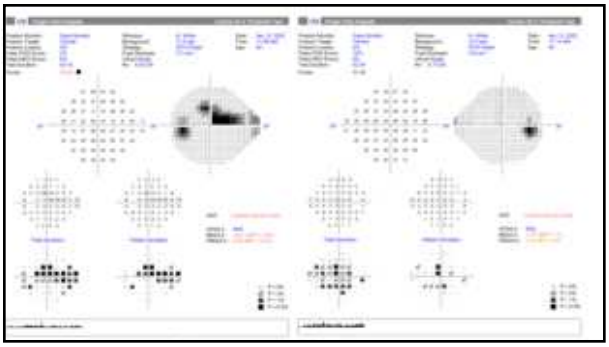

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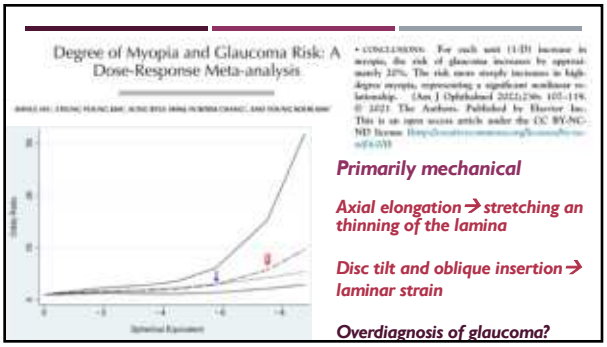

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67 YEAR OLD FEMALE; GLAUCOMA SUSPECT

- **What changed?**
  - Multiple IVI (approximately 6-9 per year) between 2014 and 2016 for macular edema secondary to CRVO
    - Avastin, Ozurdex

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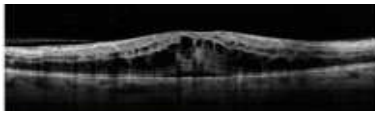
HOW *COULD* IVI AND VITRECTOMY CAUSE ELEVATED IOP?!

- Widening indications for vitrectomy
- Anti-VEGF injections are the cornerstone of medical retinal treatment
- **Long and short term IOP rise possible**
- Development of OAG & progression of OAG



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SHORT TERM

- We tend to think about the greatest risk of IVI to be endophthalmitis (1/2659)
- Immediately after injection: IOP rise to up to **87mmHg**
  - Most patients increase approximately 20mmHg-35mmHg
  - *Do most surgeons measure IOP after injections?*
- How does this happen?!
  - Increased intravitreal volume
    - 4-4.4mL average volume; most injections 0.05mL

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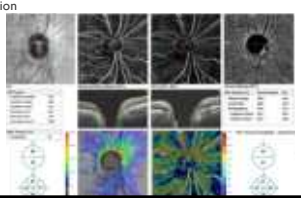
WELL THAT CAN'T BE GOOD

- Risk of retinal artery occlusion (as high as 1/1389 Gao et al 2019)
- Repeated, sudden, **significant** IOP spike and temporary loss of perfusion



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HOW CAN WE FIX THIS?

- **Treatment for elevated IOP vs. IOP spike-prevention**
  - Role of pre-procedure IOP lowering medication
  - Paracentesis
    - 32 gauge needle
    - Fluid balance

Anterior chamber paracentesis during intravitreal injections in observational trials: effectiveness and safety and effects

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ALL ABOUT OUTFLOW

- Reduced trabecular outflow:
  - ~~1) Direct toxicity of medication~~
  - 2) Inflammation
    - Trabeculitis
  - 3) Aggregation of particles
    - Silicone, protein in the TM
  - 4) Nitric oxide reduction

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SILICONE?

- Medical grade silicone oil droplets
  - Barrel of the syringe
  - Hub of the needle
  - Tip of the plunger
  - Stopper of the medication vial
- *Silicone oil has the potential to be pro-inflammatory*

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NITRIC OXIDE

- Nitric oxide is involved in the signaling pathway which leads to relaxation of trabecular beams
  - Leads to increased trabecular outflow
    - Latanoprostene bunod
      - Latanoprost acid + butanediol monohydrate
        - NO is a gas, so must be attached to another molecule
- VEGF upregulates nitric oxide synthase = increased nitric oxide
- *Effect of **anti-VEGF** medications?*

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SO WHO IS MOST AT RISK?

- Greater number of injections (20+)
- Higher frequency of injections (7/year +)
  - Eadie et al 2017
- Younger patients
- Patients with shorter axial length

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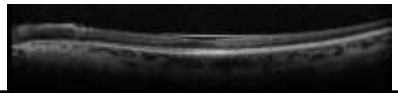
VITRECTOMY & TAMPONADE AGENTS

- Long term potential for IOP rise
  - Oxidative stress-fluid/air exchange
- Tamponade agents
  - Sulfur hexafluoride (SF_6)
  - Perfluoropropane (C_3F_8)
  - Silicone oil-greatest risk of IOP elevation-as high as 40%



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BOTTOM LINE

- **Monitor intraocular pressure in patients undergoing IVI or who have a history of PPV**

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48 YEAR OLD FEMALE

- Recently relocated and presented to establish ongoing glaucoma care
  - POAG OU (diagnosed about 15 years ago)
    - Latanoprost QHS OU
    - Dorzolamide-timolol BID OU
    - Brimonidine BID OU
- IOP 10mmHg OD and OS
  - CCT 477um/487um
- Gonioscopy
  - Open to ciliary body 360 degrees and unremarkable
    - Best repeated every 1-2 years—or with an unexpected IOP measurement

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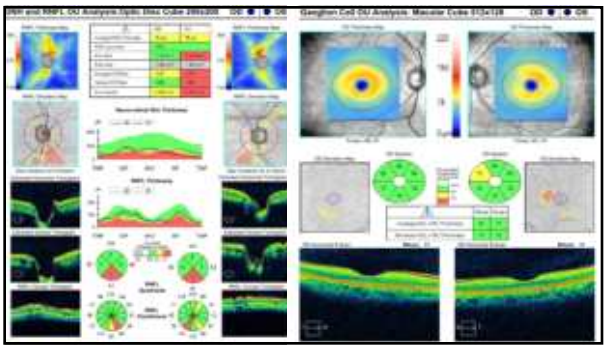

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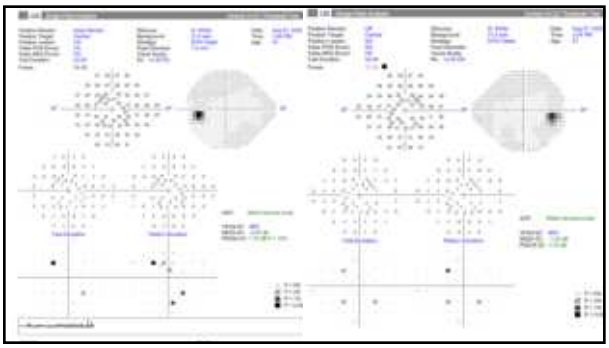

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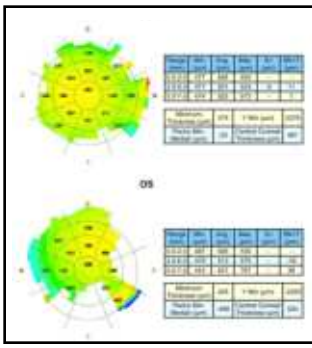

Is this glaucoma?

What are this patient's risk factors for development of glaucoma?

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48 YEAR OLD FEMALE

- Now what?
  - Discontinue medication?
  - What is the risk of continuing therapy and carrying over the previous diagnosis?

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Discontinuation of therapy

Step-wise, logical approach

1. Stop dorzolamide-timolol
IOP 15/15mmHg
2. Stop brimonidine
IOP 17/18mmHg
3. Stop latanoprost
IOP 29/28mmHg

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Discontinuation of therapy

4. Diagnose ocular hypertension

5. Restart latanoprost → switch to latanoprostene bunod 0.024%
14mmHg OD 13mmHg OS

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Sometimes the best action is <u>seemingly</u> "inaction"

Taking the time you need you need to evaluate a treatment, repeat a test, or observe an individual over time will clarify unexpected or equivocal findings

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41 YEAR OLD FEMALE

- Referred for evaluation of suspicion of glaucoma due to optic disc appearance and narrow angles
- Comprehensive eye examination:
  - HPI:
    - 1) Blurred vision
    - 2) Halos at night
    - 3) Redness (bilateral, relatively constant)
    - 4) Headache (2-3 times per month)
  - +0.75-1.00x170
  - +0.25-0.75x015
  - IOP 18/19mmHg

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41 YEAR OLD FEMALE

- Pinhole VA 20/20 OD and OS
- IOP 18/19mmHg
- Gonioscopy
  - OD: No structures seen superior and temporal, anterior trabecular meshwork nasal and inferior
  - OS: Anterior trabecular meshwork 360
  - ***Convex iris approach, no PAS, NVA, AR 360 OD and OS (with compression)***

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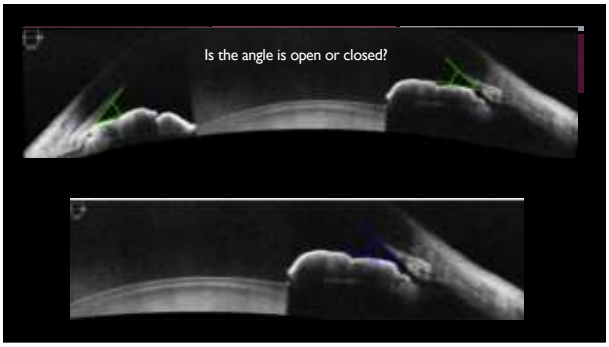

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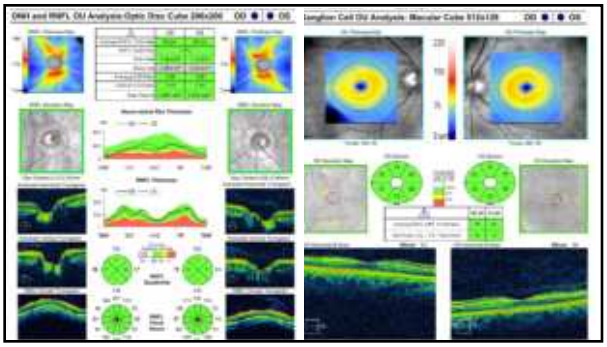

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TERMINOLOGY

- **1) Primary angle closure suspect**
- 2) Primary angle closure
- 3) Primary angle closure glaucoma
- 4) Acute angle closure crisis

Either open or closed
There is no such thing as "narrow angle glaucoma"

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PRIMARY ANGLE CLOSURE SUSPECT

- AKA "anatomical narrow angle"
- The pigmented trabecular meshwork is blocked by the iris 180 degrees or more by ***gonioscopy***
  - Without compression
    - No peripheral anterior synechiae
- **Disc is normal; IOP is normal**
- Ask the patient about symptoms of intermittent closure
  - Especially when the pupil is dilated (i.e. at night)
- ***LPI or observation?***
  - Stop going to movies, stop going to restaurants at night, stop using anti-allergy or cold medications…

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Laser peripheral iridotomy for the prevention of angle closure: a single-centre, randomised controlled trial

- Zhongshan Angle Closure Prevention (ZAP) trial
- Purpose: to determine if laser iridotomy is superior to observation in primary angle closure suspects in China over a 6 year period
  - PACS = 6 or more clock hours where posterior trabecular meshwork was not visible
    - Without elevated IOP, disc change, or peripheral anterior synechiae
- Endpoint: elevated IOP--used dark-room prone provocative testing (compared pre-test IOP to IOP measured after 15 minutes in a dark room in prone position), PAC, acute angle closure
- Outcome: 889 eyes treated. 50% reduction in risk for development of primary angle closure over 6 years, but only 4% of untreated eyes progressed to primary angle closure
  - Acute angle closure: 5 patients untreated, 1 treated (3 control eyes and one LPI eye were after dilation)
  - Authors determined that laser peripheral iridotomy was not justified in smaller populations

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14-Year Outcome of Angle-Closure Prevention with Laser Iridotomy in the Zhongshan Angle Closure Prevention Study: Extended Follow-Up of a Randomized Controlled Trial

Endpoint: PAC, PAS, IOP>24mmHg or AAC

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Progression of Primary Angle Closure Suspect to Primary Angle Closure and Associated Risk Factors: The Handan Eye Study

526 patients (111 male, 415 female)
32 progressed to angle closure (31 PAC, 1 PACG) in 5 years = 6%

CLINICAL SCIENCE

Five year risk of progression of primary angle closure suspects to primary angle closure: a population based study

Southern India: ¼ PACS subjects developed PAC

132

Anatomic Changes and Predictors of Angle Widening after Laser Peripheral Iridotomy

The Zhongshan Angle Closure Prevention Trial

Conclusions: Superior LPI location results in significantly greater angle widening compared with temporal or nasal locations in a Chinese population with PACS. This supports consideration of superior LPI locations to optimize anatomic changes after LPI. *Ophthalmology 2021;■:1–8 © 2021 by the American Academy of Ophthalmology*

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What does LPI do?!

Prevents or reverses pupil block

134

Do we feel comfortable dilating this patient?!

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BOTTOM LINE

- Challenging clinical circumstances arise.
- When they do: stick to first principles
  - No device is better than a skilled and experienced clinician
- New medications and procedure-based therapies are excellent options when cost and access allow
- Collaboration is central to person-centered glaucoma care

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Jessicaa.steen@gmail.com
480.289.0613

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