**On behalf of Vision Expo, we sincerely thank you for being with us this year.**

**Vision Expo Has Gone Green!**

We have eliminated all paper session evaluation forms. Please be sure to complete your electronic session evaluations online when you login to request your CE Letter for each course you attended! Your feedback is important to us as our Conference Advisory Board considers content and speakers for future meetings to provide you with the best education possible.

VISION EXPO

1

# PROBLEM SOLVING IN GLAUCOMA MANAGEMENT

JESSICA STEEN OD, FAAO, DIPL ABO

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## FINANCIAL DISCLOSURES

- Speaker-Carl Zeiss Meditec, Bausch and Lomb
- Advisory Board-Bausch and Lomb, Santen, Peripherex, Ocuphire, Oyster Point, Ocuterra
- *All relevant relationships have been mitigated*

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## 60 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 | | | 64 |
| 10/28/2019 | 2:15 PM | | | 104 / 66 | wrist | adult | 65 |
| 03/30/2018 | 4:05 PM | | | 110 / 60 | | adult | 62 |
| 05/18/2016 | 12:40 PM | | | 102 / 68 | | | 60 |
| 07/14/2015 | | | | 110/60 | | | |

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

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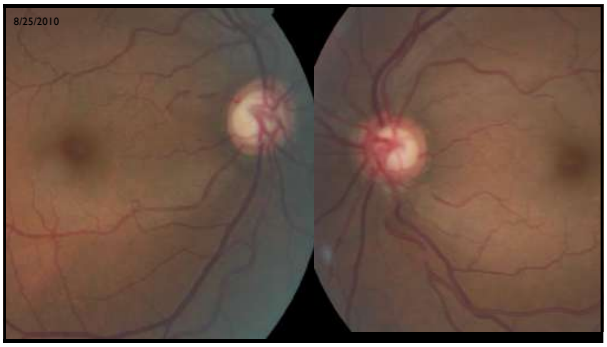


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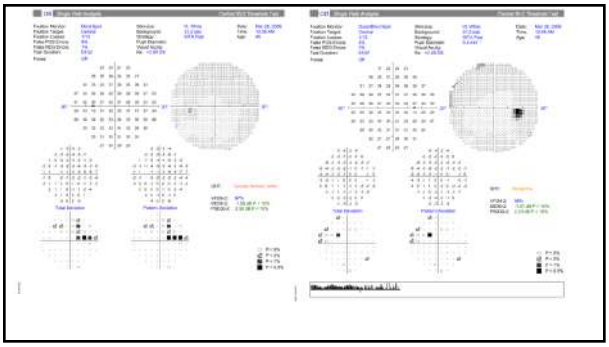

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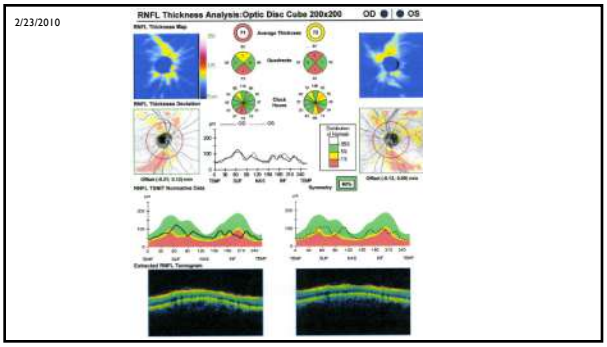
2/23/2010
RNFL Thickness Analysis:Optic Disc Cube 200x200
OD
OS

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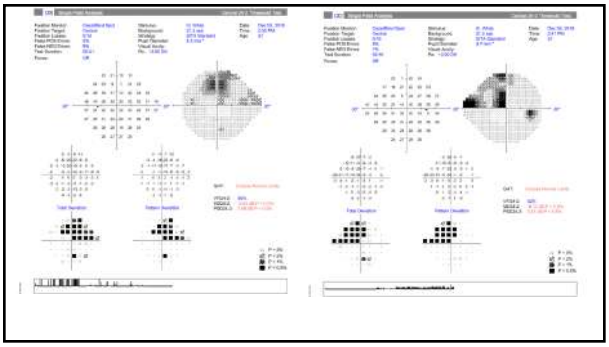

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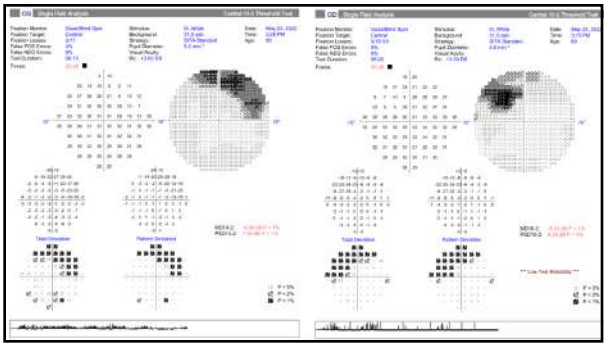

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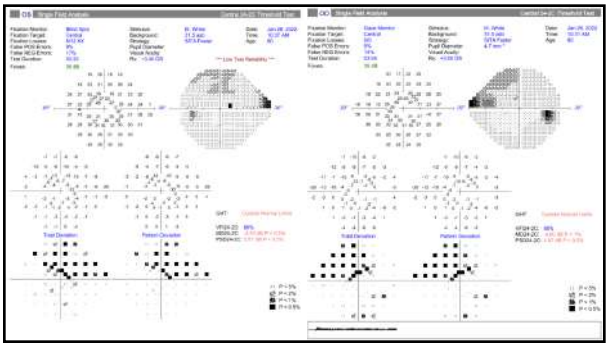

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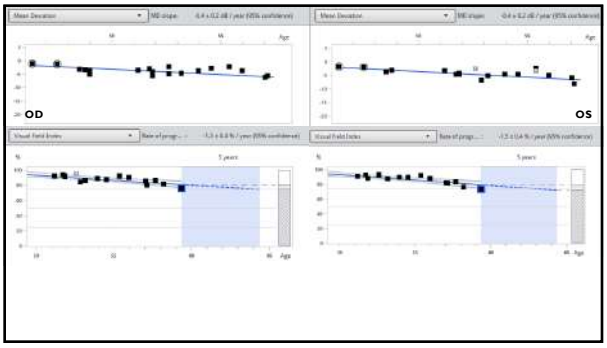
OD
OS

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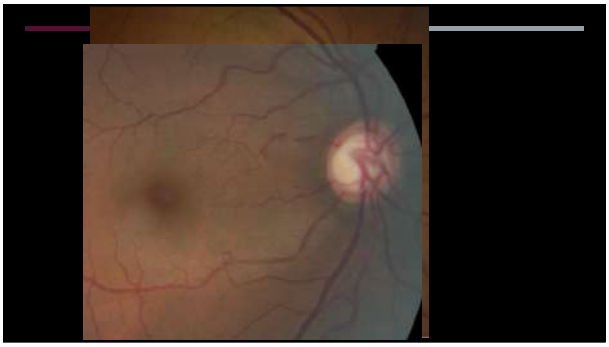

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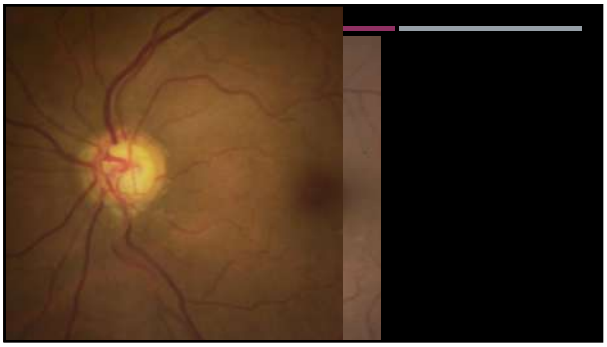

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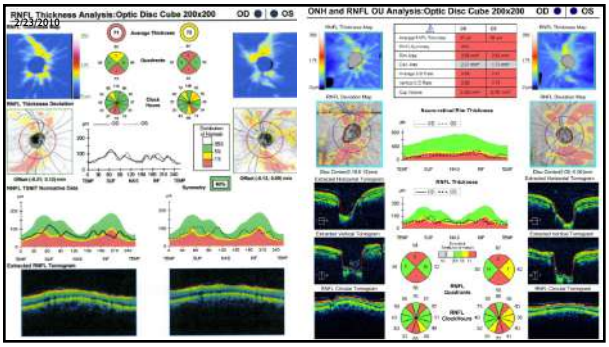


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***HAS THIS PATIENT'S DISEASE PROGRESSED?***
***YES.***

**But. The pressure is 8-10mmHg**

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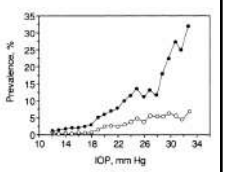
## Intraocular Pressure

- 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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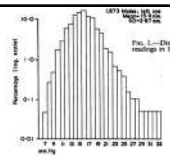
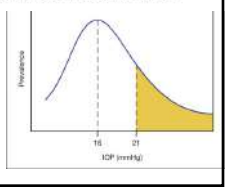


INTRA-OCULAR PRESSURE, GLAUCOMA, AND GLAUCOMA SUSPECTS IN A DEFINED POPULATION*

F. C. HOLLOWS† AND P. A. GRAHAM

- "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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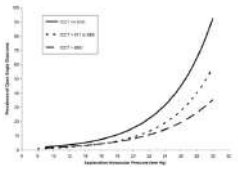
## CENTRAL CORNEAL THICKNESS

- 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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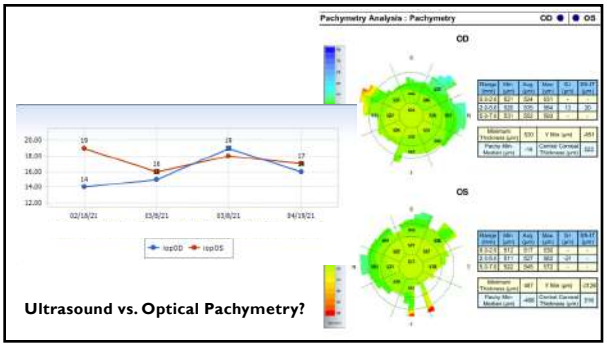


**Ultrasound vs. Optical Pachymetry?**

23

## The Cupped Disc

*Who Needs Neuroimaging?*

David S. Greenfield, MD,[1] R. Michael Siatkowski, MD,[1] Joel S. Glaser, MD,[1,2] Norman J. Schatz, MD,[1,2] Richard K. Parrish II, MD[1]

***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

Published in final edited form as:
*Ophthalmology*. 2019 December ; 126(12): 1640–1646. doi:10.1016/j.ophtha.2019.07.023.

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

Bianca N. Susanna, MD[1,2], Nara G. Ogata, MD[1], Alessandro A. Jammal, MD[1], Carolina N. Susanna, MD[1,2], Samuel I. Berchuck, PhD[1,3], Felipe A. Medeiros, MD, PhD[1]

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## *What else can we blame glaucoma on?*

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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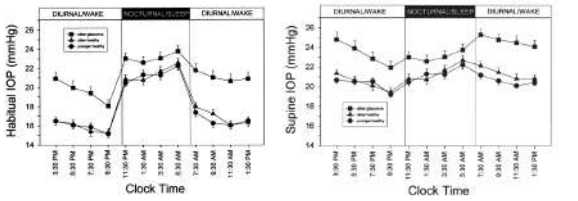
## INTRAOCULAR PRESSURE VARIATION




Mosaed S, Liu JHK, Weinreb J. Am J Ophthalmol 2005

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## *Impact of head position on IOP*

**Measure supine IOP in office?**

**Have patients sleep with their head elevated?**

28

## 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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## *WHAT IS 'MAX MEDICAL THERAPY?'*

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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
- Less common-corneal verticillata

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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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## "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, cartelol ophthalmic solution, metipranolol ophthalmic solution, levobunolol ophthalmic solution, timolol ophthalmic solution and gel, Lumigan ophthalmic drops, and travoprost 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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## *Ocular surface disease is common*

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

***Really…that's it?***

40

## 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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## ALTERNATIVES

- Medication options
  - Non-BAK formulations
    - Travoprost 0.004% (Travatan Z) sofZia
    - 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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## ALTERNATIVES

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

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**Efficacy of Repeat Selective Laser Trabeculoplasty in Medication-Naive 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**

Gus Gazzard, Evgenia Konstantakopoulou, David Garway-Heath, Anurag Garg, Victoria Vickerstaff, Rachael Hunter, Gareth Ambler, Catey Bunce, Richard Wormald, Neil Nathwani, Keith Barton, Gary Rubin, Marta Buszewicz, on behalf of the LiGHT Trial Study Group*

**No game-changing data**

***But did provide good quality evidence for what was already known***

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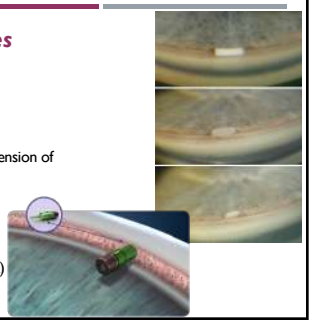
## *Sustained release devices*

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)
NDA Accepted by the FDA
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)

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## REMOVING MEDICATIONS WILL NOT ELIMINATE OCULAR SURFACE DISEASE

***Cost and access are real concerns to alternative medications and procedures***

48

## 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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## 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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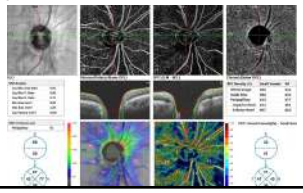
## WELL THAT CAN'T BE GOOD

- 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

International Journal of Retina and Vitreous

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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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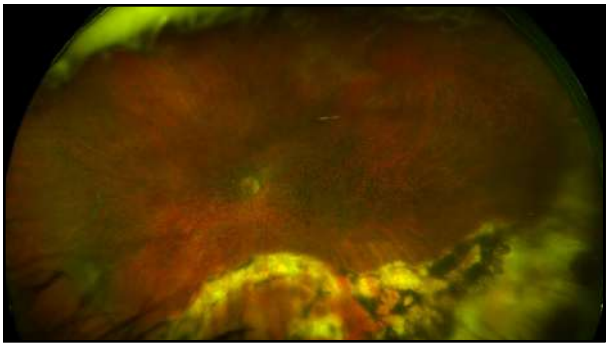

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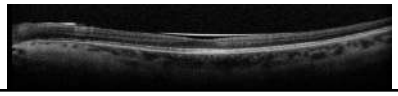
## VITRECTOMY & TAMPONADE AGENTS

- 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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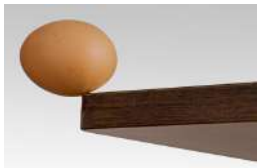
## BOTTOM LINE

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



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## CASE

- 27 year old black male presents for evaluation of blurred vision in the right eye for about a year; he reports 'good' vision in the left eye
  - Thinks it has been a gradual change the visual reduction in the right eye
- First eye examination
- 3+ APD OD
- BCVA:
  - 20/400 OD
  - 20/30 OS
- Unremarkable anterior segment
- IOP 41/36mmHg
- **Now what?!**

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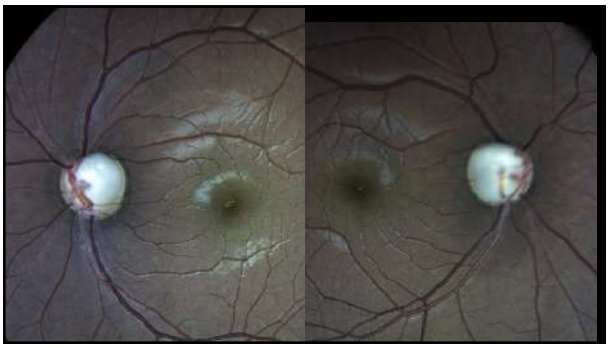

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## JUVENILE OPEN ANGLE GLAUCOMA

- 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

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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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## *Glaucoma and Genetics*

***Currently, about 127 loci have been identified***

***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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***ROUTINE GENETIC TESTING FOR GLAUCOMA RISK ALLELES IS NOT RECOMMENDED FOR PATIENTS WITH POAG***

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## JUVENILE OPEN ANGLE GLAUCOMA

- IOP rises sometime between about 2 and 16 years of age
  - Diagnosed before about 40 years of age

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## MANAGEMENT OF JOAG?

- Most patients respond very well to topical medications
  - The challenge is adherence to therapy through a patient's life
- Medication options-depends on the the patient's age
  - Brimonidine
    - Crosses the blood brain barrier
    - Contraindicated in children less than 2; causes **significant** somnolence and decreased alertness in children 2-7 years of age
  - Prostaglandin analog-best adherence at FDA approved
    - Safe in pediatric populations; but just don't work well in many children

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## MANAGEMENT?

- Surgical options need to last many, many years
  - No rush towards incisional surgery for most patients with JOAG
- Robust healing response
- Don't want to run out of options too soon
  - Canaloplasty and trabeculotomy being explored in kids with JOAG
    - Durability is the key
- Low vision consultation?
  - Most effective early in the course of disease

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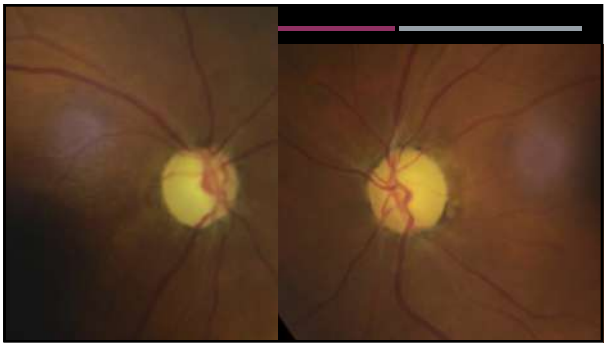
## 68 YEAR OLD MALE

- Referred by NSU primary care service due to advanced disease
- BCVA 20/60 OD 20/40 OS
- NO APD
- IOP 32mmHg OD and OS
  - CCT 482µm OD 476 µm OS

71



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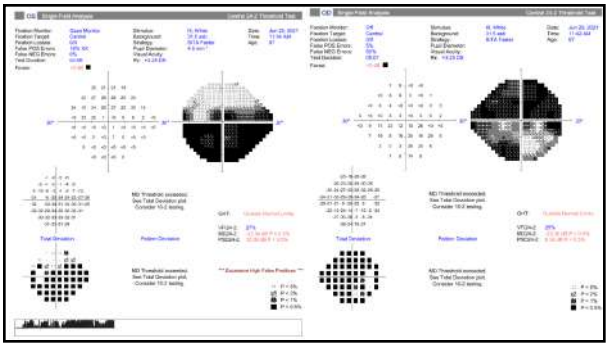

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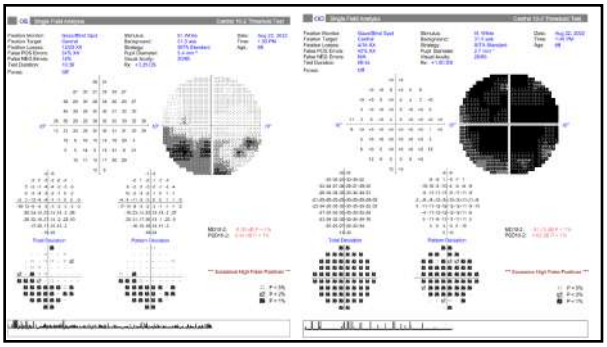

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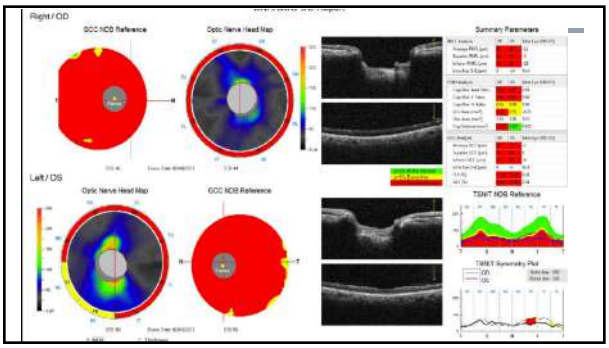


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***Now what?!***

***Medication?***
***Laser?***
***Intracameral drug delivery?***
***Surgery?***

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## *TAGS*

***Primary trabeculectomy for advanced glaucoma (2021) United Kingdom***

***Primary trabeculectomy (12.4mmHg)***
***Primary medication (15.1mmHg)***

***Similar quality of life, similar safety outcomes***

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## *CIGTS*

***United States (1993-1997)***

***Medication arm (35% IOP reduction)***
***Stepped topical therapy (timolol, pilocarpine, dipivefrin), oral medications***

***Surgical arm (48% IOP reduction)***
***Trabeculectomy (with or without 5-FU)***

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## *PTVT*

***3 year results (2020)***

***Trabeculectomy with MMC: lower IOP with fewer topical ocular medications vs. tube shunt***

***No significant difference in the rate of surgical failure at 3 years***

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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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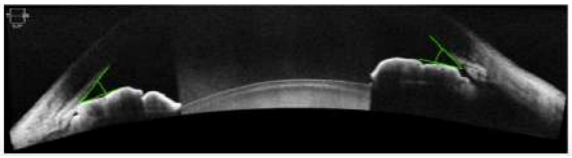
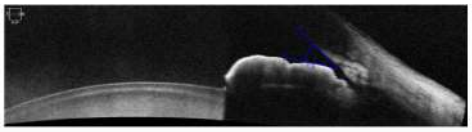
## 41 YEAR OLD FEMALE

- 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)***

81





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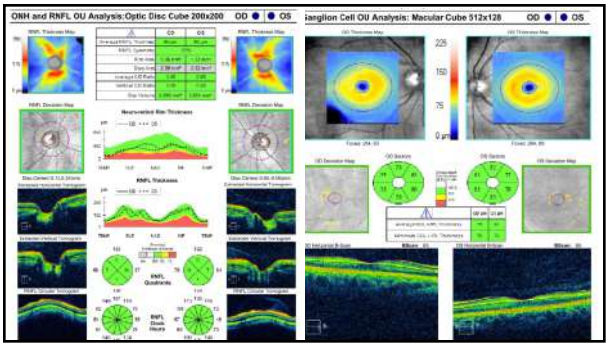


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## TERMINOLOGY

- **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

Minggaang He, Yuzhen Jiang, Shengsong Huang, Dolly S Chang, Beatriz Munoz, Tin Aung, Paul J Foster*, David S Friedman*

- 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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Glaucoma

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

Ye Zhang,[1] Ravi Thomas,[2] Qing Zhang,[1] Si Zhen Li,[1] and Ning Li Wang[1,3]

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

R Thomas, R George, R Parikh, J Muliyil, A Jacob

Southern India: ¼ PACS subjects developed PAC

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***Do we feel comfortable dilating this patient?!***

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AMERICAN ACADEMY OF OPHTHALMOLOGY

## Anatomic Changes and Predictors of Angle Widening after Laser Peripheral Iridotomy

*The Zhongshan Angle Closure Prevention Trial*

Benjamin Y. Xu, MD, PhD,[1] David S. Friedman, MD, PhD,[2] Paul J. Foster, FRCS(Ed), PhD,[3] Yu Jiang, MD,[4] Anmol A. Pardeshi, MS,[1] Yuzhen Jiang, MD, PhD,[4] Beatriz Munoz, MS,[5] Tin Aung, FRCS(Ed), PhD,[6] Mingguang He, MD, PhD[4]

***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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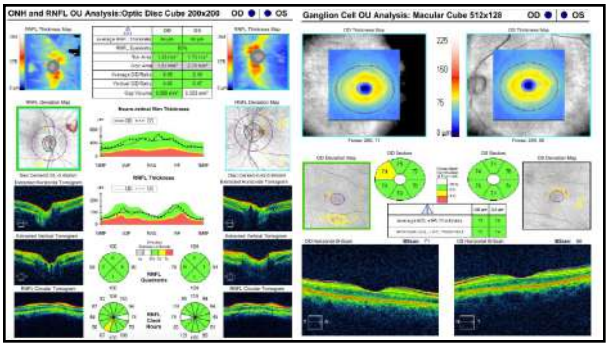
## 64 YEAR OLD HISPANIC FEMALE

- History of bilateral retinoschisis (monitored without treatment)
- +1.00-0.50x090 OD and OS; 20/20 OD, 20/50 OS
- IOP 19mmHg OD, **33mmHg OS**


90

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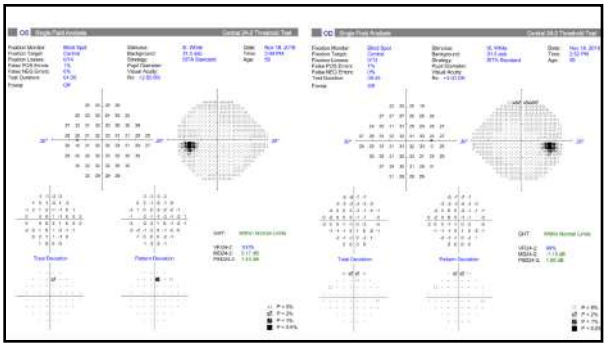

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## Effectiveness of early lens extraction for the treatment of primary angle-closure glaucoma (EAGLE): a randomised controlled trial

- Removal of clear lenses in eyes with PACG with IOP > 21 mmHg or eyes with PAC (without glaucoma) and IOP >30mmHg vs. LPI (and medications); greater than 50 years of age
  - Clear lens extraction patients had greater IOP control and improved quality of life
  - Patients who underwent lens extraction had fewer IOP lowering medications
    - Only 1 needed trabeculectomy after phaco whereas 24 patients in the LPI group needed trabeculectomy
  - Cost-effective at 3 years; savings by 10 years
    - Fewer procedures, fewer office visits
- Clear lens extraction can be considered—good luck having it covered by insurance

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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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