







#### MISTAKES TO AVOID IN GLAUCOMA MANAGEMENT

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## DISCLOSURE



Dr. Joseph Sowka is/ has been in the past 24 months a consultant or member of the advisory or speaker boards for Zeiss, Visus, and B&L. All relevant relationships have been mitigated. 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. <a href="https://www.optometricedu.com">www.optometricedu.com</a>



#### **MISTAKE TO AVOID**

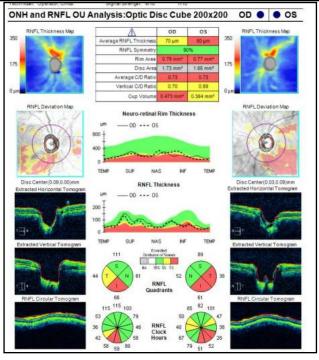
- Not recognizing a neurologic field
- Thinking glaucoma causes optic disc pallor
- Diagnosing NAAION in glaucoma patients
- Not recognizing when the OCT is wrong
- Treating red disease
- Not treating real disease
- Changing therapy based upon one bad IOP or field
- Not getting enough pre-treatment...and posttreatment IOPs
- Not recognizing patients who will likely do well
- Not identifying patients who likely will not do well.

### **MISTAKE TO AVOID**

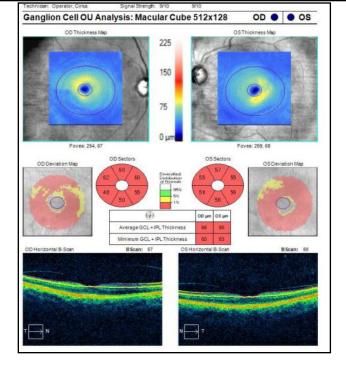
Not recognizing a neurologic field

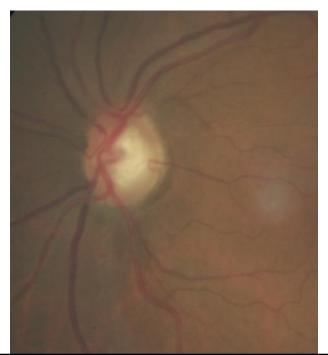
### **74 YOF**

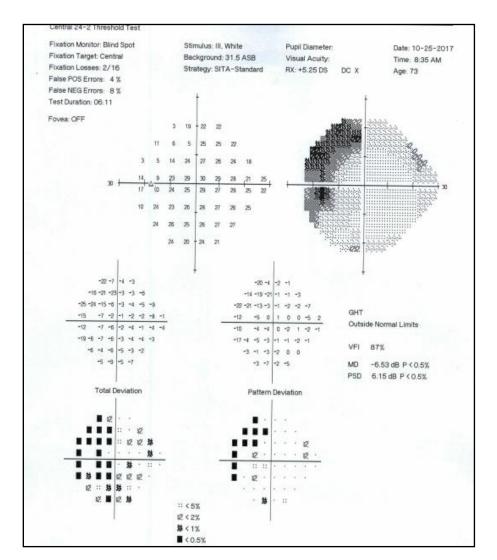
- Diagnosed with glaucoma in Jamaica
- Ran out of meds: IOP 20 mm OU
- 20/50 OD, 20/40 OS
- NS 2+
- PERRL(-)RAPD

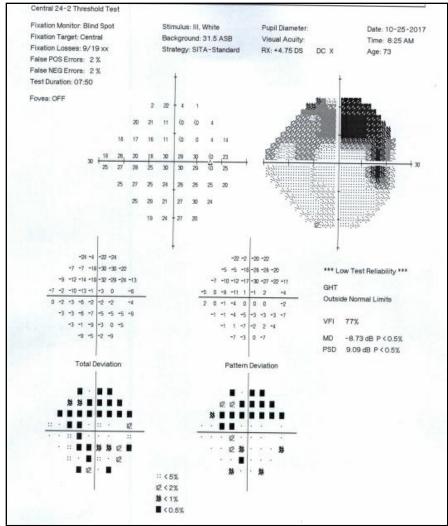


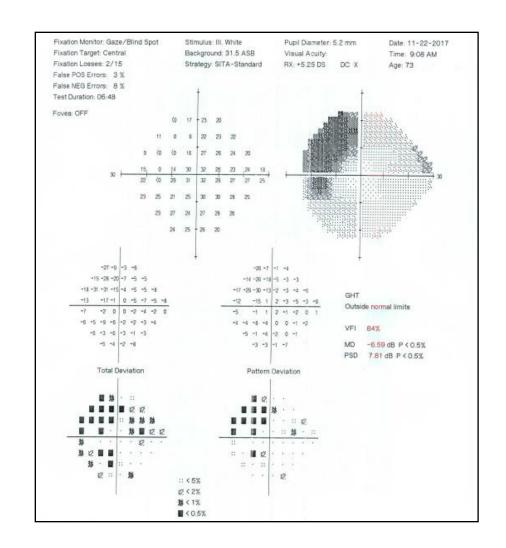


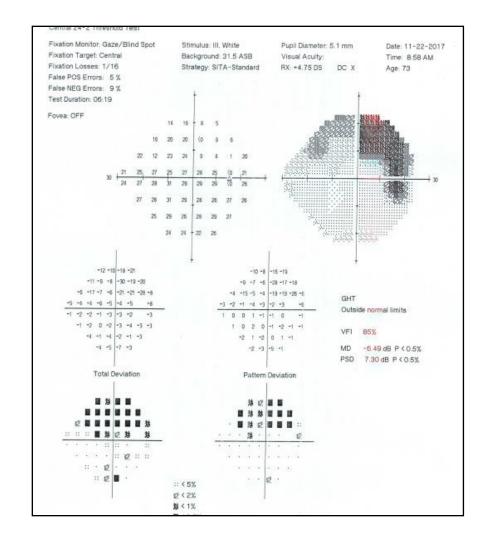












**FINDINGS:** There is a large T1 hypointense and T2 iso- to hyperintense lesion extending between the sella into the suprasellar region showing heterogeneous enhancement on the post-contrast images measuring 2.7 cm craniocaudal x 2.1 cm AP x 2 cm transverse. Findings are compatible with a pituitary macroadenoma. It is resulting in compression of the optic chiasm and slightly compressing upon the hippocampus. There is preservation of the signal void of the cavernous carotids. There is possible extension into the cavernous sinus medially. There is slanting of the floor of the sella.

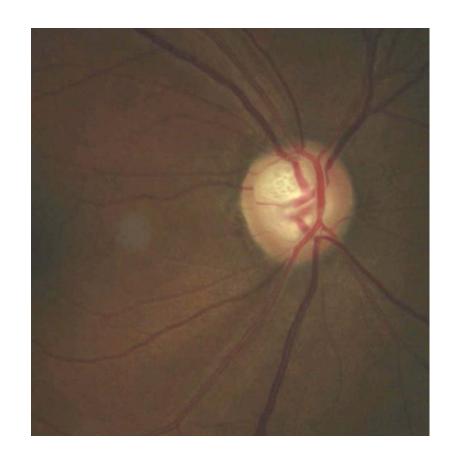
The ventricles are in midline. There are multiple bilateral periventricular and subcortical T2 hyperintensities most commonly representing chronic small vessel ischemia in this age group.

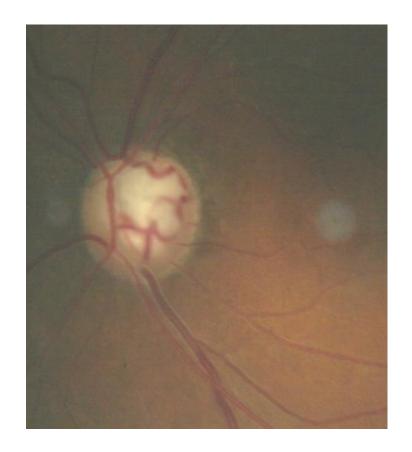
The globes are symmetric. There is no lens dislocation. The post-septal soft tissues are preserved with no definite intra- or extraconal mass. The optic nerves are symmetric at the orbital level showing no abnormal enhancement.

#### IMPRESSION:

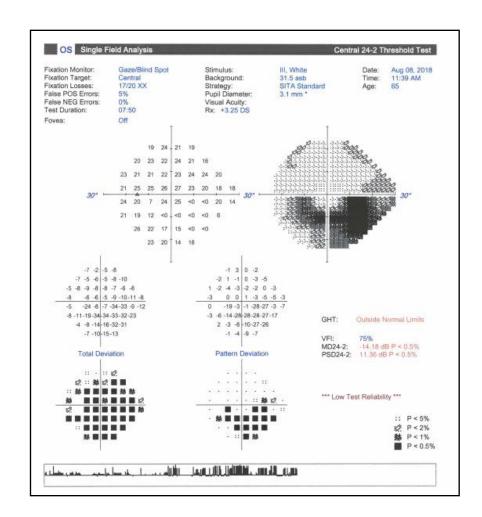
- 1. Large heterogeneous enhancing sella/suprasellar mass resulting in compression of the optic chiasm compatible with a pituitary macroadenoma.
- 2. Bilateral periventricular and subcortical T2 hyperintensities compatible with chronic small vessel ischemia.

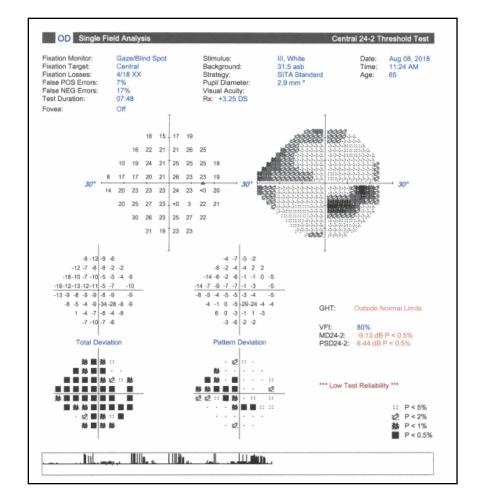
#### 65 YOF- POAG OU





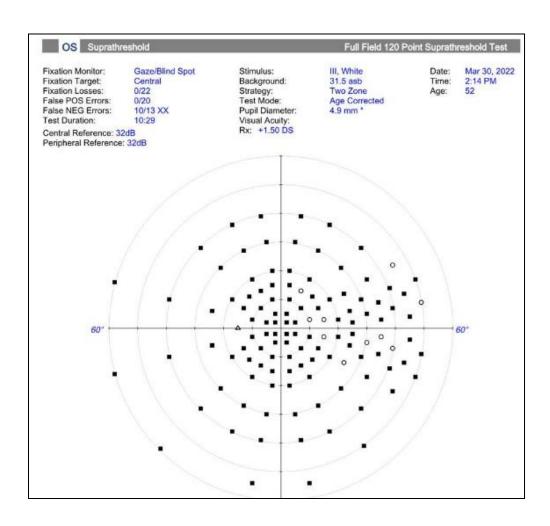
Peak IOP unknown; s/p SLT OU and on latanoprost at first visit.

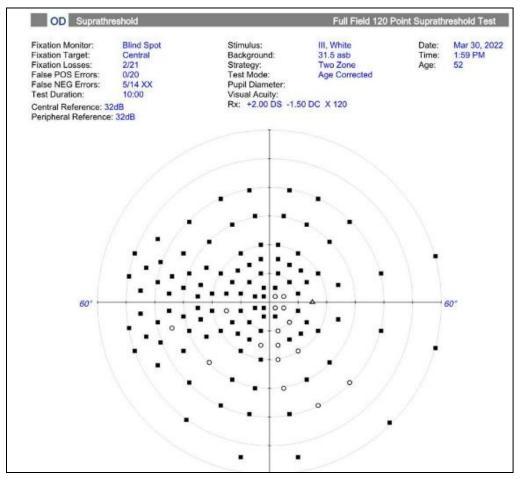


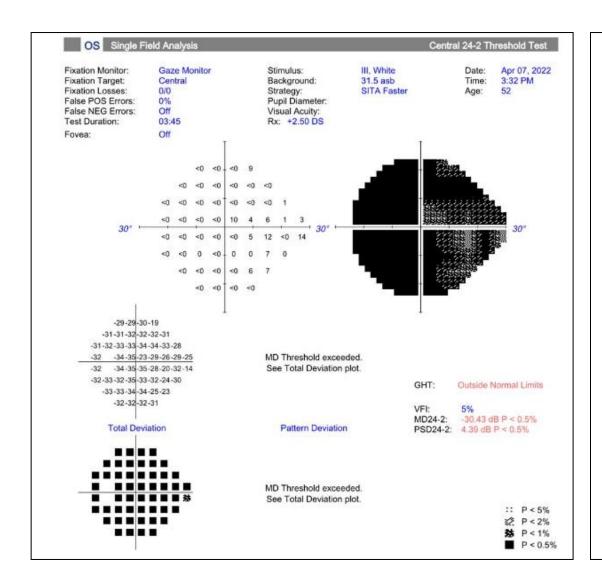


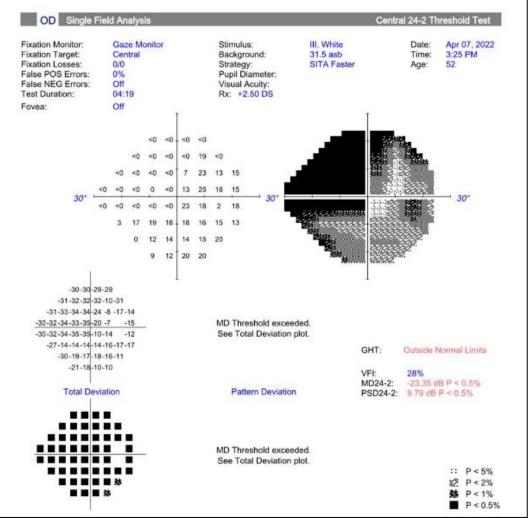
Oh, by the way, she remembered waking up 10 years ago unable to speak for several hours.

# 53 YOM COMPLAINS OF BLIND LEFT SPOT WHILE DRIVING









#### **MISTAKE TO AVOID**

Thinking glaucoma causes optic disc pallor

#### **RULE**

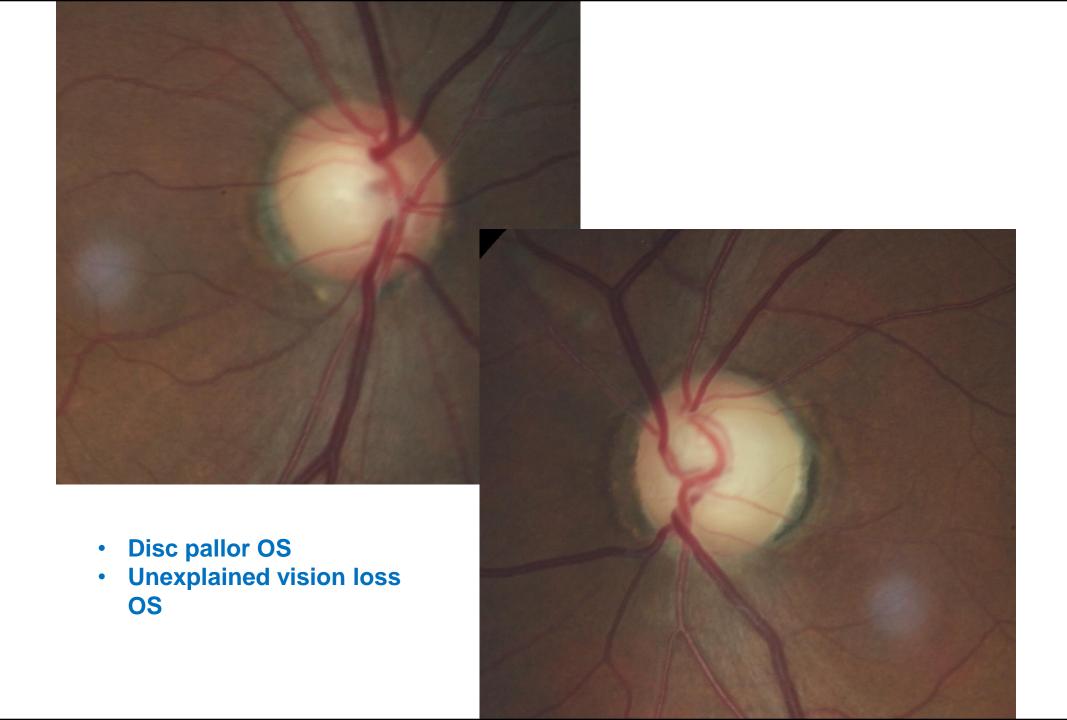
Pallor in excess of cupping indicates something other than, or in addition to, glaucoma

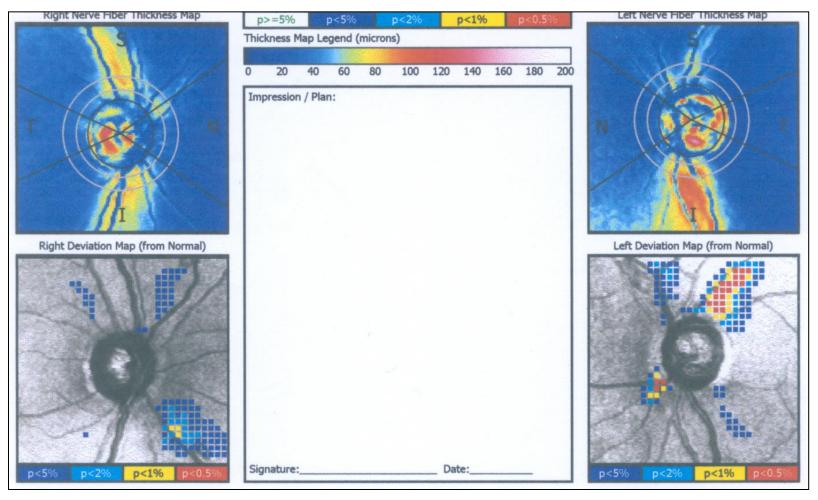
#### **RULE**

# Nothing notches a nerve like glaucoma

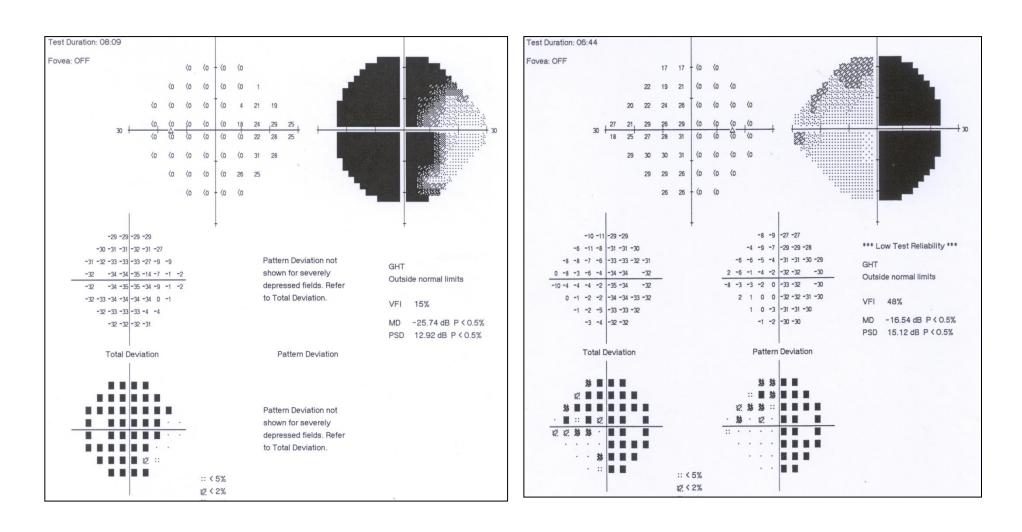
# IN THE AGE OF IMAGING, DO WE REALLY NEED FIELDS?

- 54 YO Nigerian man
- Referred for glaucoma management
- Told he had glaucoma 6 years earlier- no Tx
- 6/9 OD; HM OS
  - Vision loss from glaucoma- not coming back
- 30 mm Hg OD; 23 mm Hg OS
  - Lumigan- 17 mm Hg OD, 15 mm Hg OS





Do we really need fields in this case?



Yes, we still need to do fields in the age of imaging.

Sometimes its not glaucoma

#### **ODE TO A CUPPED DISC**

Oh, to have a cupped disc pink. That my friend hath a glaucomatous stink. But to have a cupped disc pale, Call this glaucoma and you shall fail. Disc and field damage that is one-sided Simply cannot be abided. It might be trauma, infarct or meningioma. But if the rim is cut always remember, Nothing notches a nerve like glaucoma

#### **MISTAKE TO AVOID**

Diagnosing non-arteritic anterior ischemic optic neuropathy in glaucoma patients

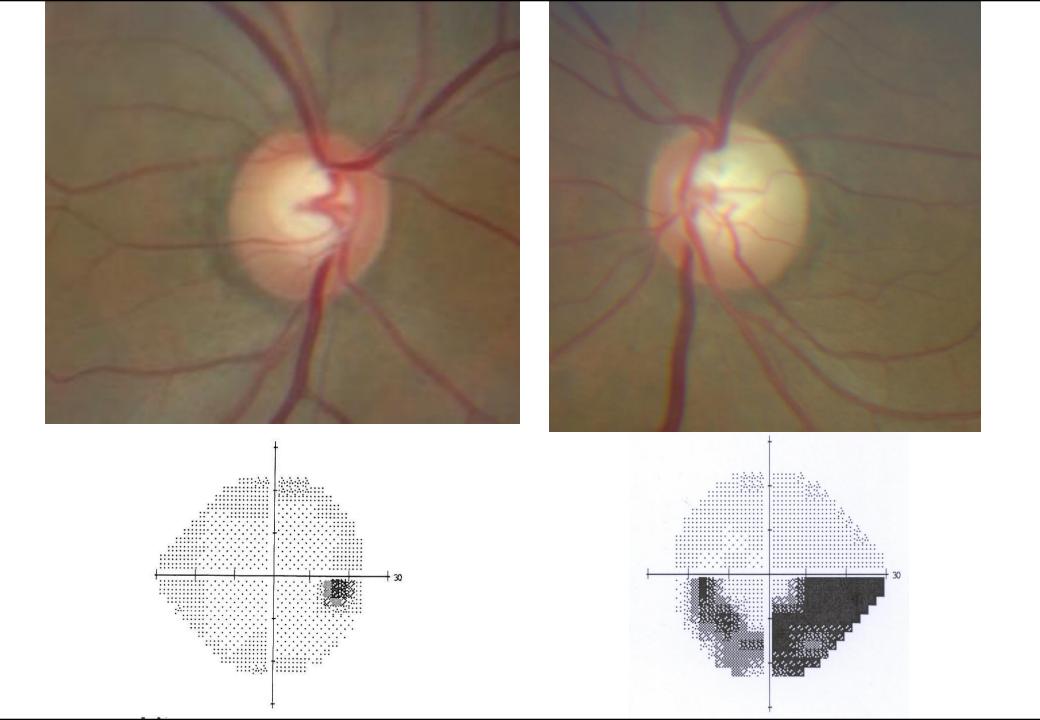
# NAAION IS A GREAT DIAGNOSIS OF CONVENIENCE

- There is no test to conclusively diagnose it
- There is no treatment so nothing that you need to do for it
- It's a great explanation for pallor in a glaucoma patient
- But... 97% of NAAION patients have c/d of 0.2/0.2 or less.
- NAAION is a disease of non-cupping and glaucoma is a disease of cupping.



NAAION OS Disc at risk OD





#### **MISTAKE TO AVOID**

Not recognizing when the OCT is wrong

## **ISSUES IN IMAGING**

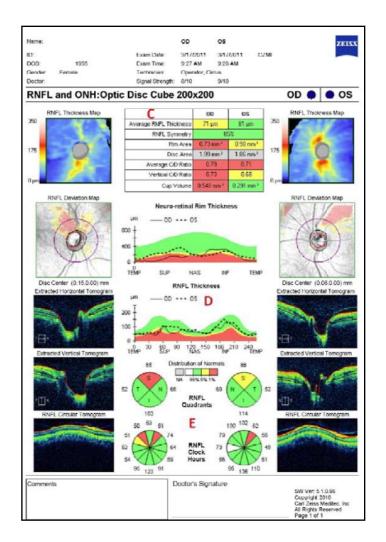
- OCT is not a Silicon Valley Rumplestilskin.
   You cannot put in straw and get out gold
- The use and overemphasis of imaging technology to the exclusion of additional clinical findings and assessment of risk will put patients in peril.
- Exactly how much confidence should an OCT give you as to whether or not a patient has glaucoma?
  - Depends how much confidence you had before you imaged the patient.

#### **ISSUES IN IMAGING**

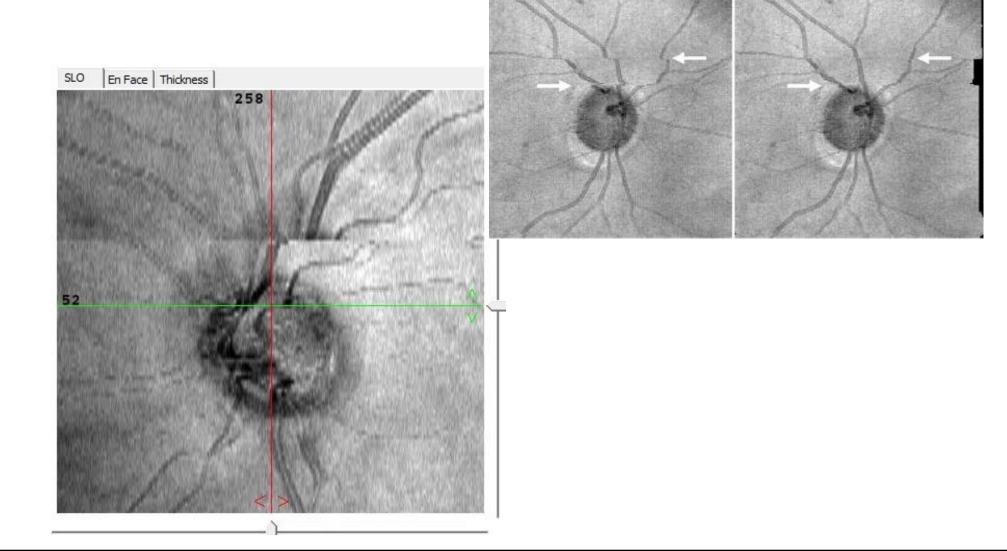
- Normative Database
- Signal Quality
- Blink/Saccades
- Segmentation Errors
- Media Opacities
- Axial Length

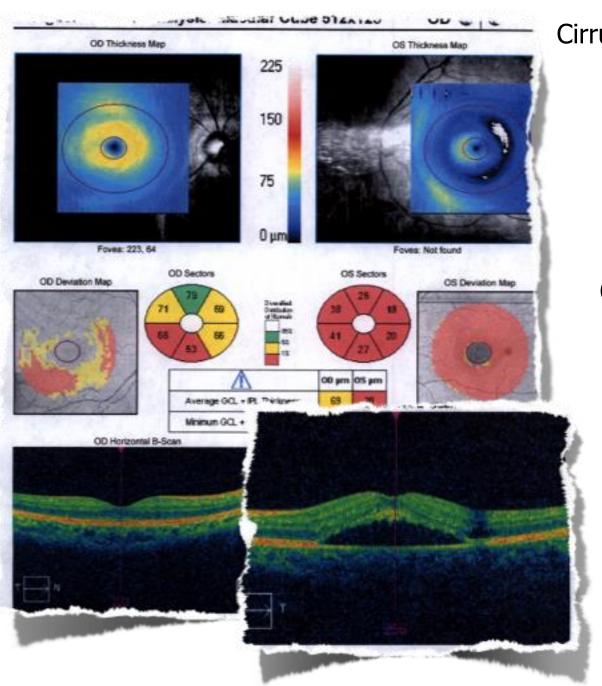
# WHAT TO LOOK FOR WHEN INTERPRETING OCT SCANS

- Quality score
- Illumination
- Focus clarity
- Image centered
- Any signs of eye movement
- Segmentation accuracy
- B Scan Centration
- Missing data
- Media issues
- Maculopathy for GCC scans



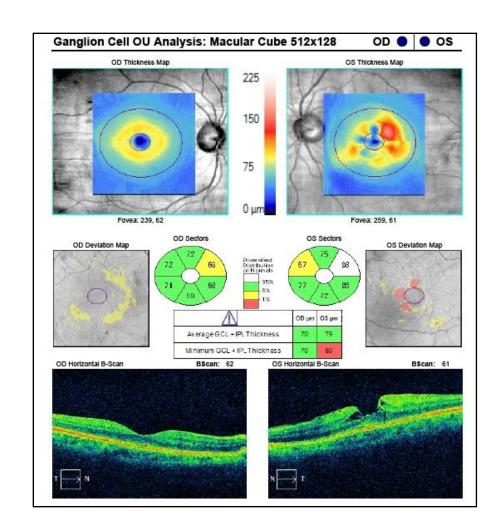
## **EYE MOVEMENT**

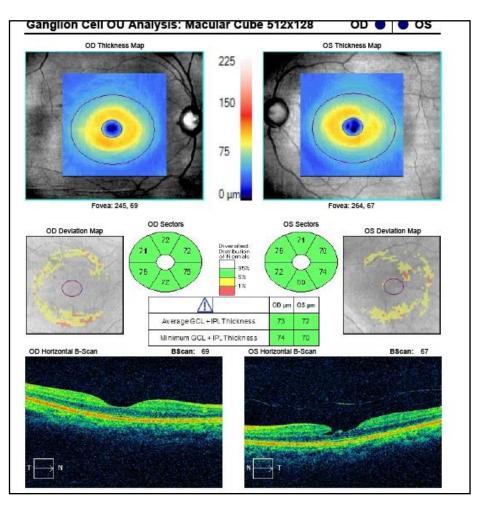




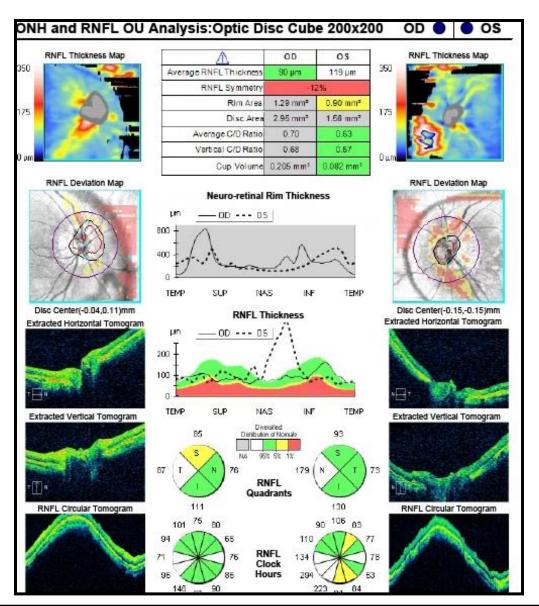
Cirrus

Accidentally find CSC when looking for glaucoma

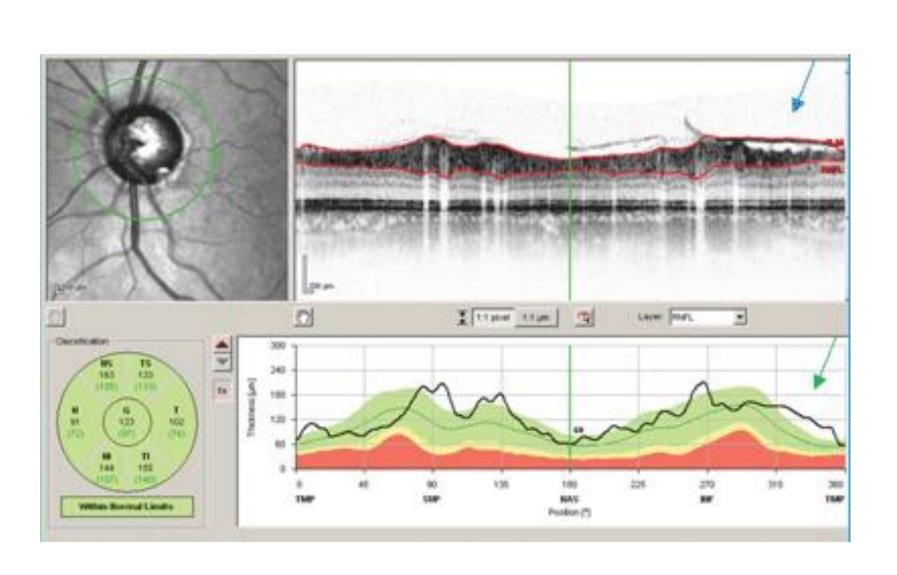




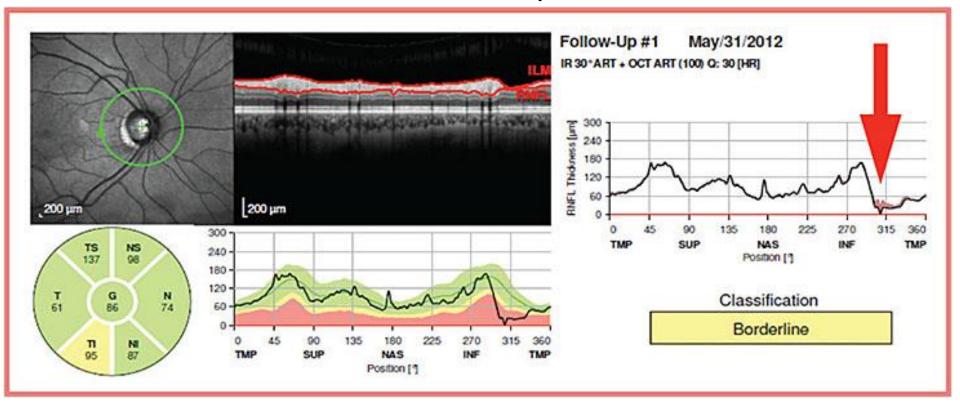
# IF YOU THINK DEVICES MEASURE TISSUE ACCURATELY EVERY TIME...

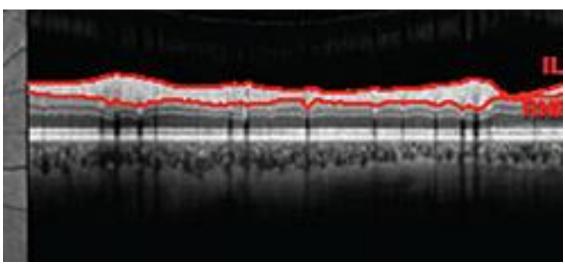


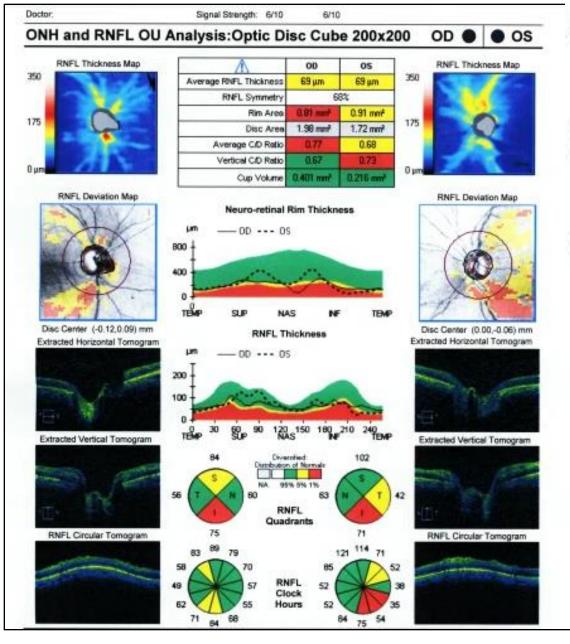
#### Spectralis

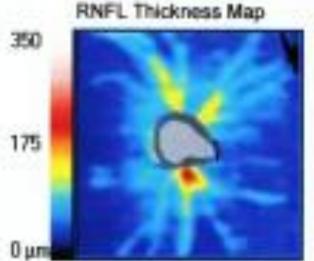


#### **Spectralis**



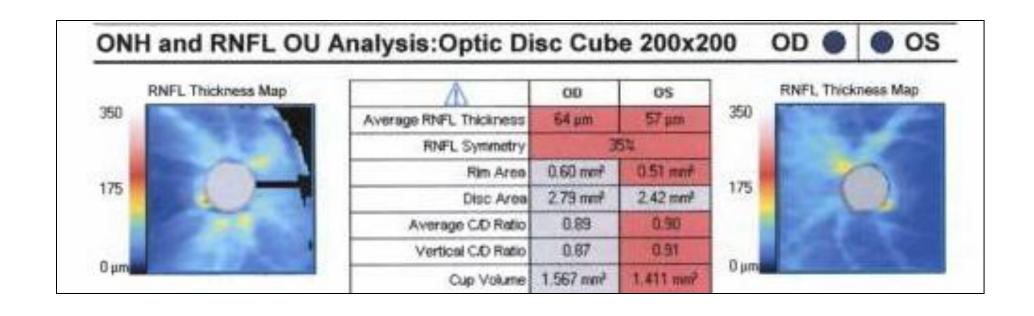


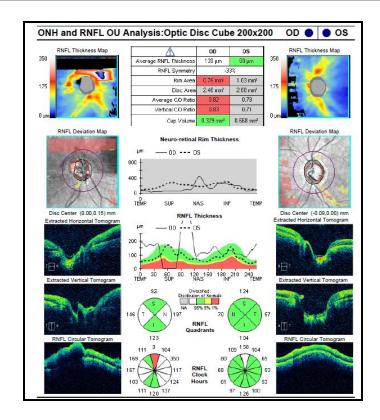


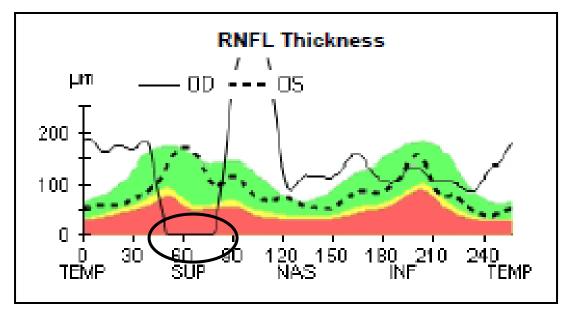




#### **FLOOR EFFECT**







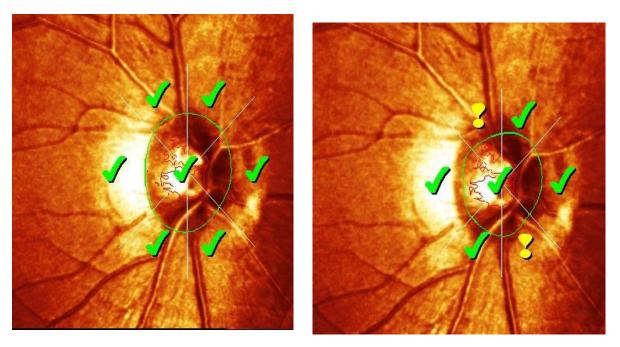
### Don't make clinical decisions based upon bad data

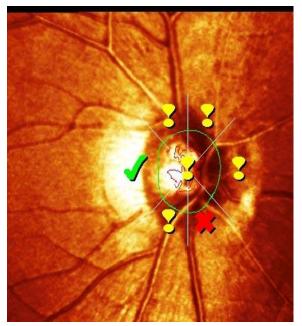
Treating red disease

#### RED DISEASE – A NEW CLINICAL NON-ENTITY

- A supratentorial, non-glaucomatous masquerade disease
- Afflicts the educated patient (especially with Internet access) with good health care plans and/or wealth
- Debilitating to the patient and painful for the visual care provider to treat

### SCANNING LASER OPHTHALMOSCOPY EXAMPLE OF RED DISEASE





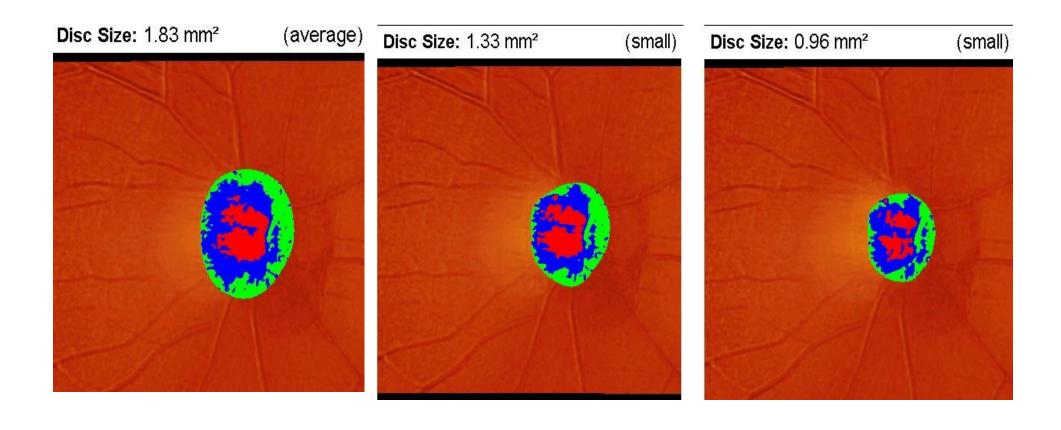
First Visit

Follow up visit #1

Follow up visit #2

HRT3 Optic Nerve Head Changes How long did this change take?

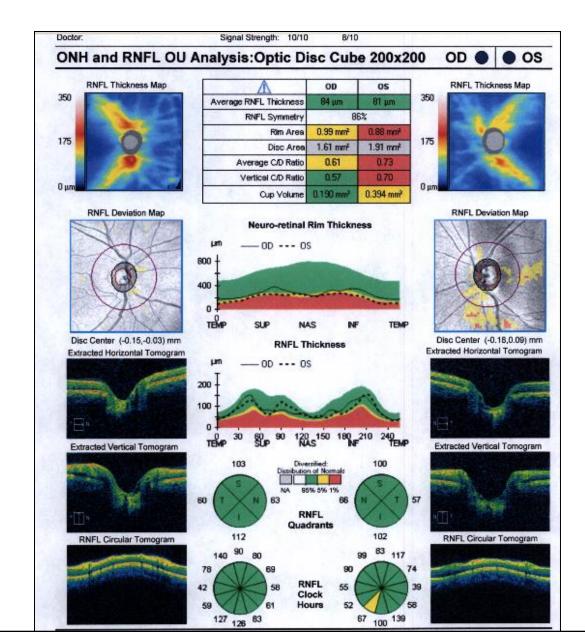
## WITHIN 15 MINUTES! HRT DISC SIZING ARTIFACT

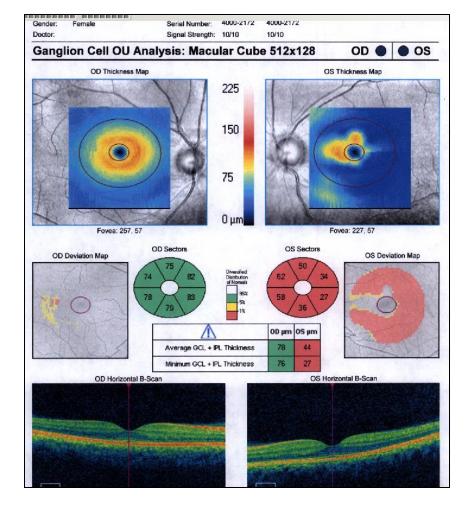


### HELP! THE DIAGNOSTIC IMAGING DOESN'T AGREE WITH MY DIAGNOSIS!

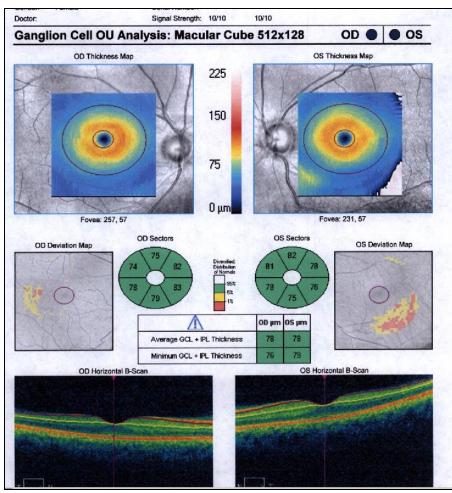
- Low risk OHTN
- Local OD wants imaging for baseline

#### **OCT RNFL NORMAL...**



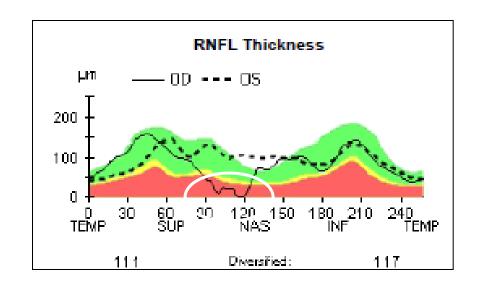


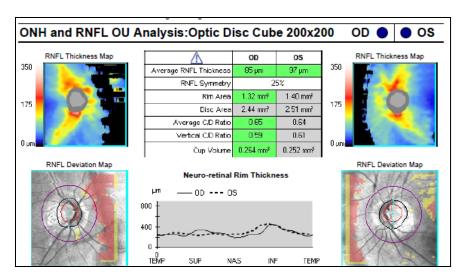
...but markedly abnormal GCC OS

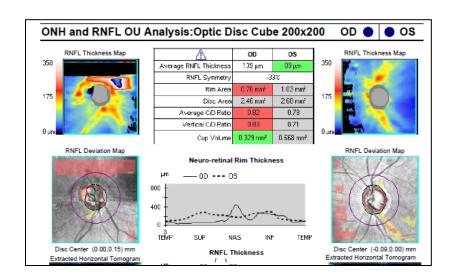


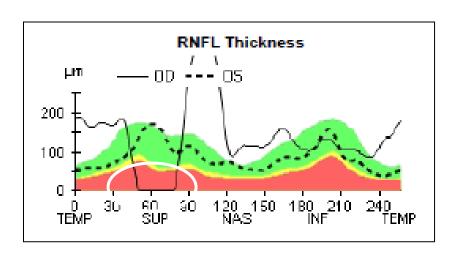
Same patient, same day, same quality, GCC now normal

Signal strength: 10/10 OD, OS on both images





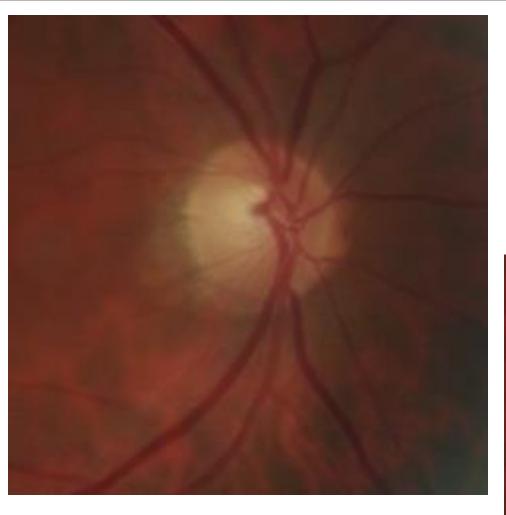


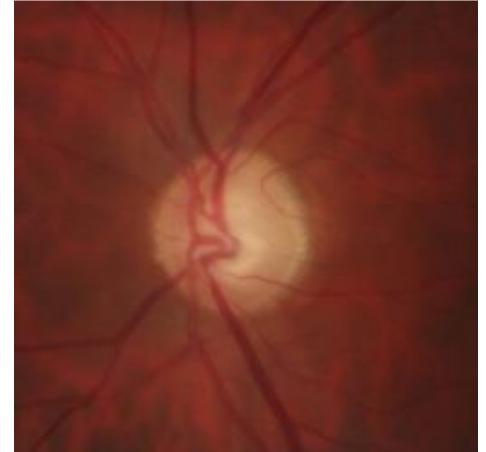


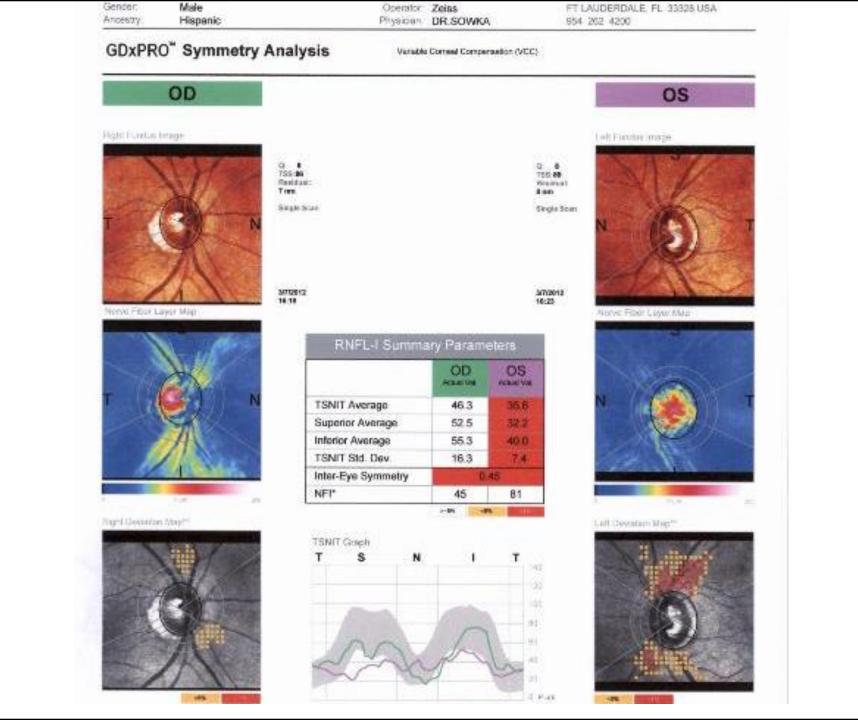
Don't make clinical decisions based upon bad data

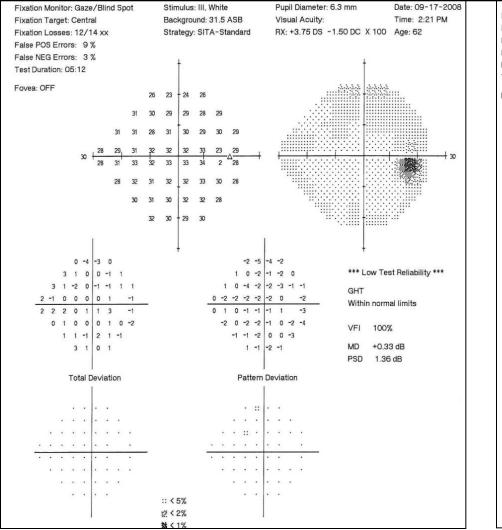
#### **CASE: 62 YOHM**

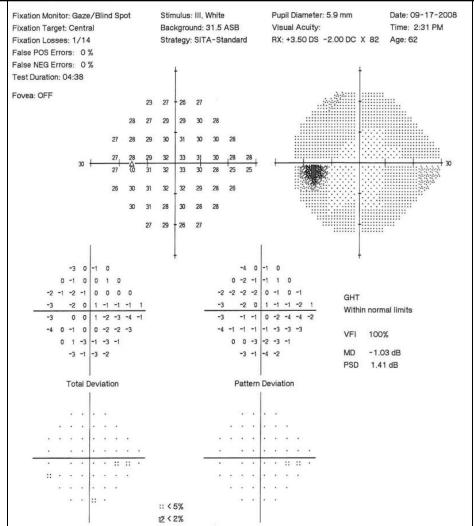
- Asymptomatic; 20/20 OD; OS
- PERRL (-) RAPD
- TA 30 mm OD, 28 mm OS
  - Isolated measurement
  - 12-17 mm OD, 13-17 mm OS
    - 11 visits
- Gonio: open OU w/o abnormalities
- CCT: 597 OU











Not treating green disease

### GREEN DISEASE- AN INSIDIOUS CLINICAL *ENTITY*

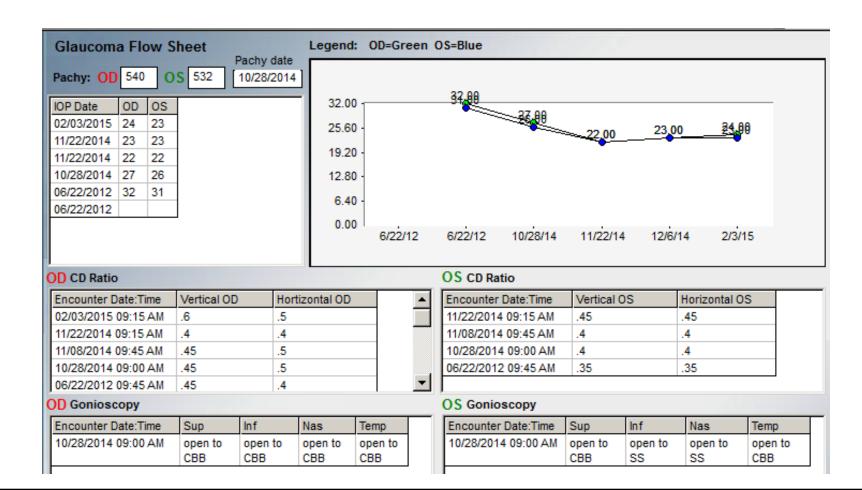
A glaucomatous process masquerading as nondisease

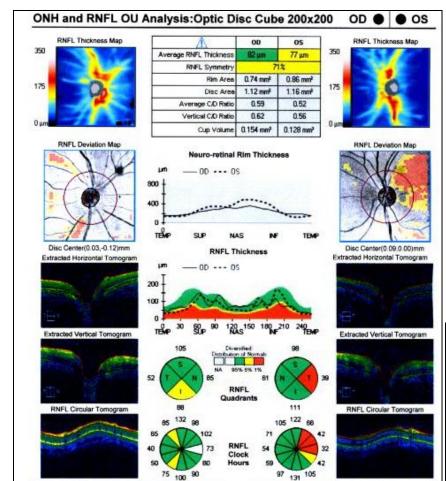
Afflicts inexperienced, poorly-educated doctors who simply want a machine to make all clinical decisions for them

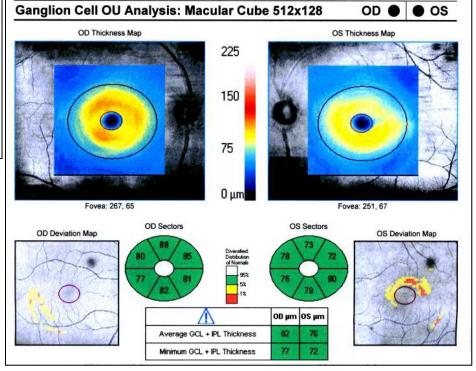
Debilitating to the patient and painful for the visual care provider, but a boon for malpractice attorneys

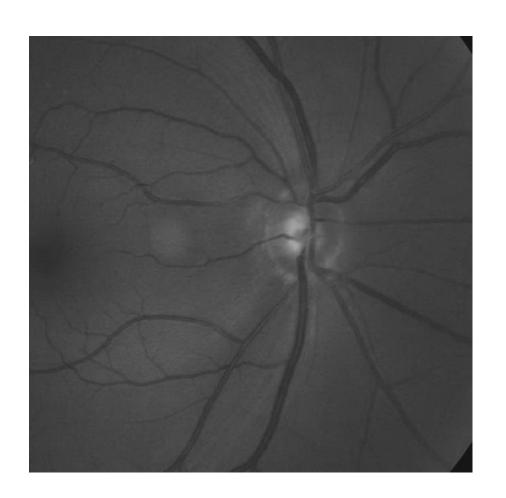
# HELP! THE DIAGNOSTIC IMAGING DOESN'T AGREE WITH MY DIAGNOSIS!

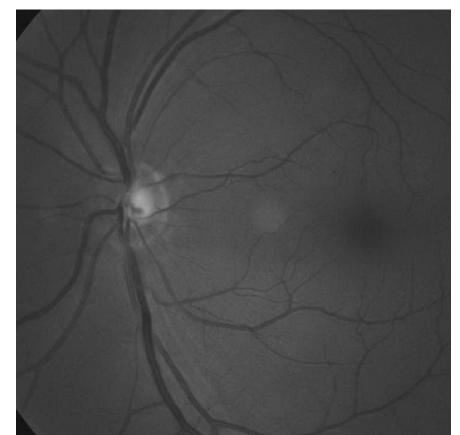
56 YOM- Glaucoma suspect since 2012

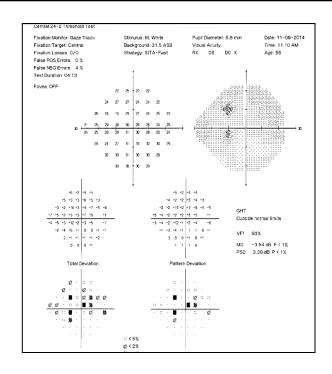


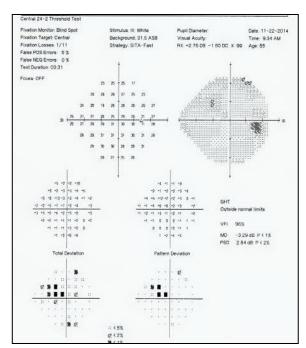


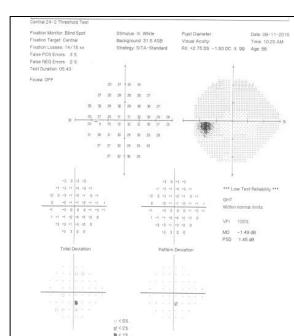


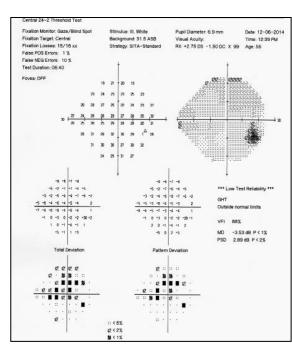


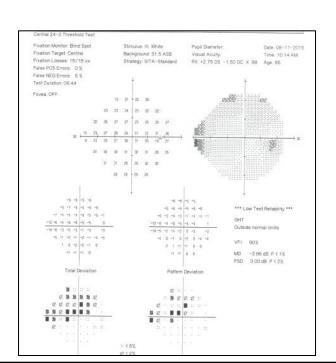








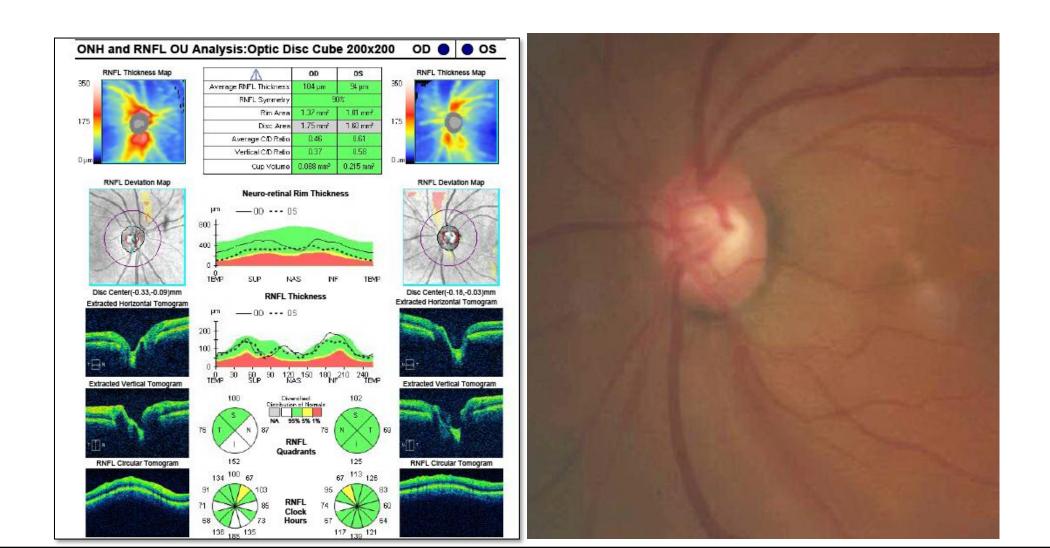




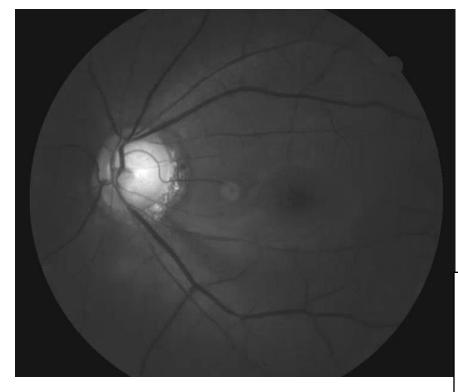
Is this person really a glaucoma 'suspect'?

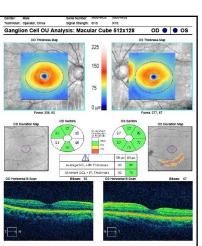
An example of Red Disease OS and Green Disease OD

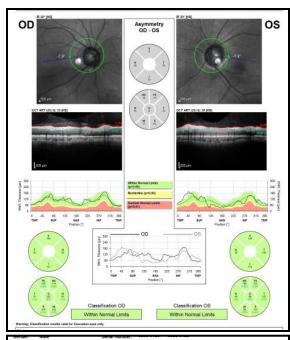
#### **GREEN DISEASE**

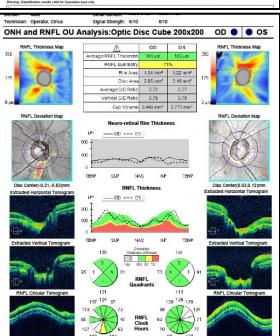


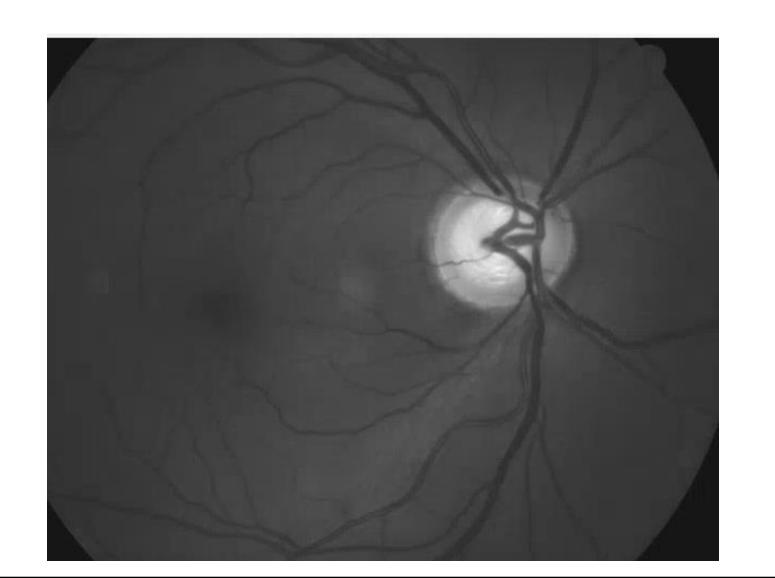
#### **GREEN DISEASE**

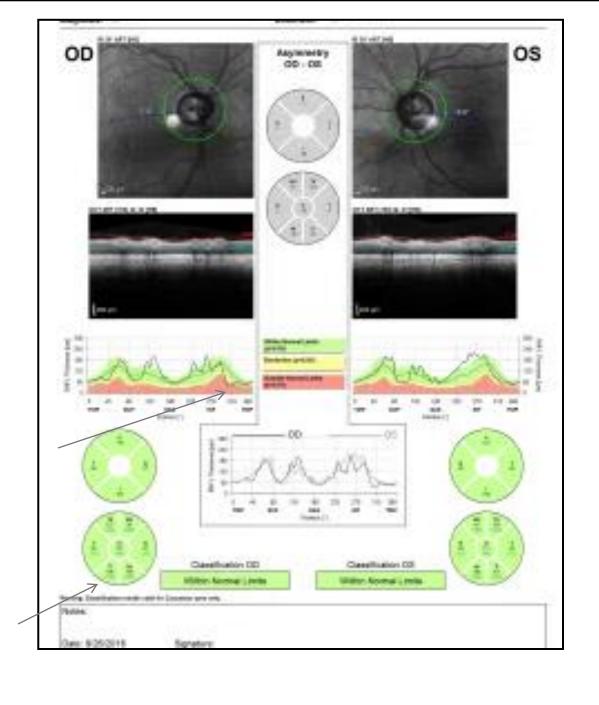


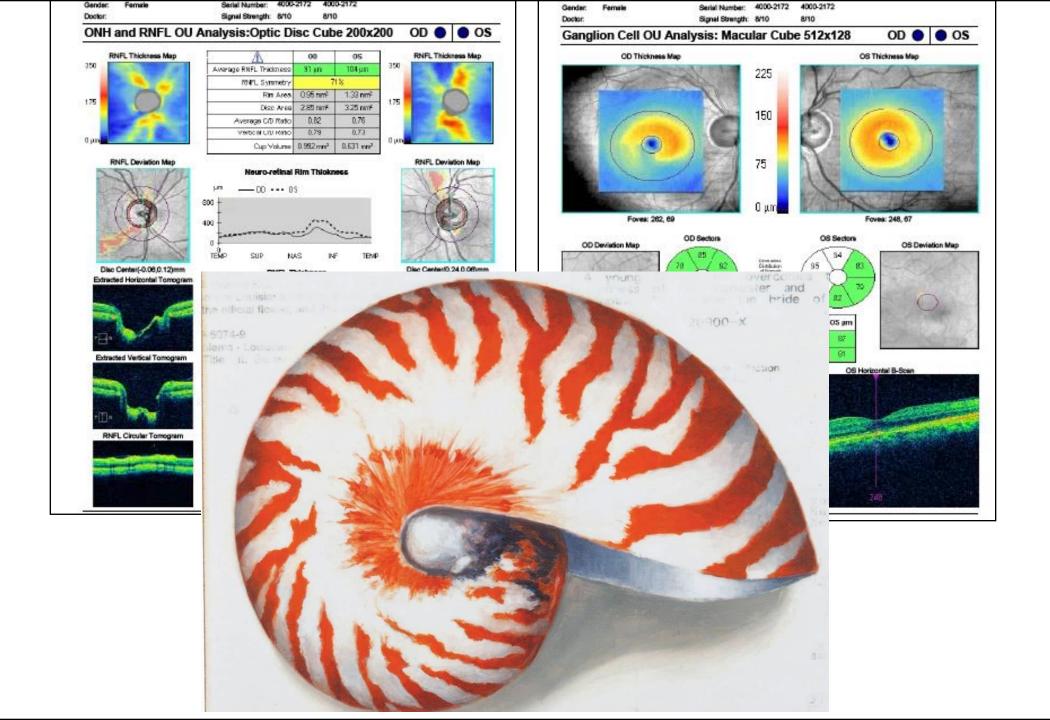


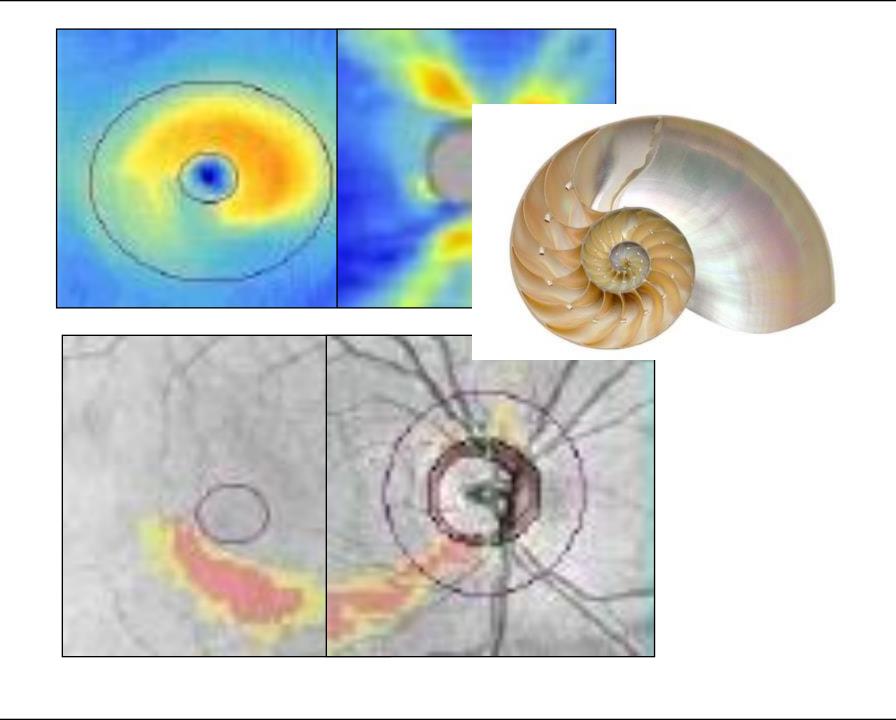












#### **OCT IMAGING TAKE HOME POINTS**

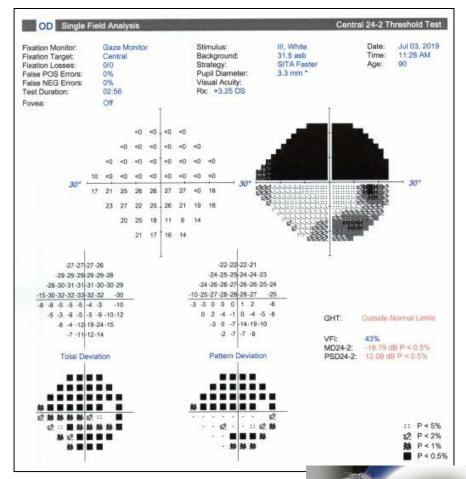
- Serial overlays/imaging to determine baseline (intra-session) noise
- Good signal strength
- Good segmentation without errors
- Optic nerve head exam for disc hemorrhage, pallor, myopic, and tilted nerve heads
- Determine structure-function correlation
- Follow all ancillary tests visual fields and optic nerve head photos for progression

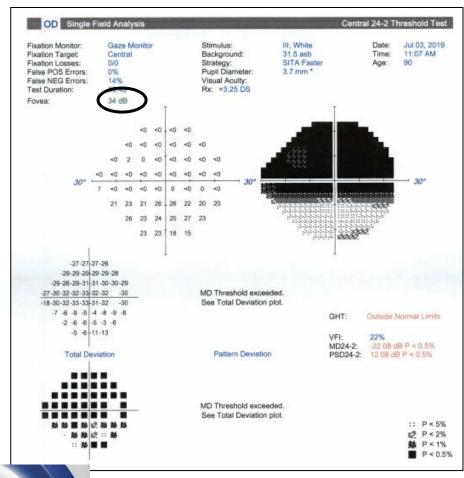
#### **CAUTIONS ABOUT IMAGING**

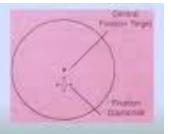
- No current technology is better than the human eye and common sense
- Beware of "Red Disease"
- Treat Real Disease and not Red Disease
- Don't miss Green Disease
- Know the limitations of the technology: normative database, reproducibility, resolution, quality of imaging
- Technologies come and go

Changing therapy based upon one bad IOP or field

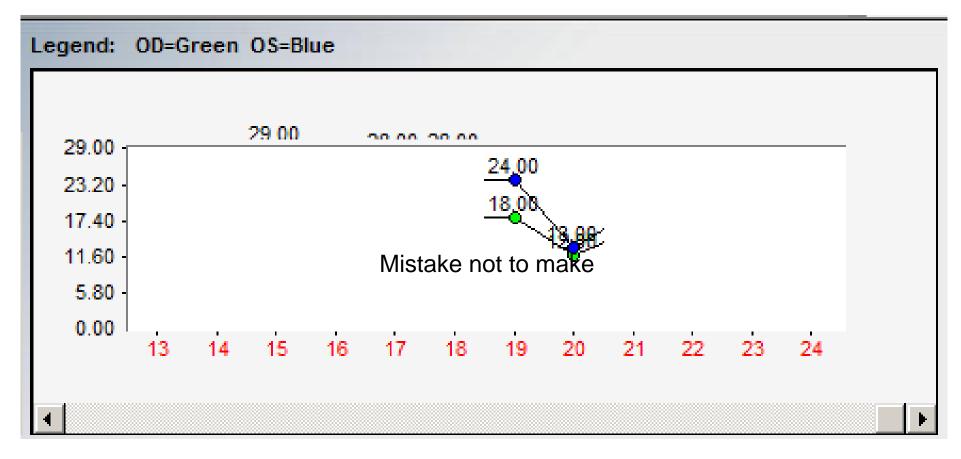
#### Is this patient getting worse?



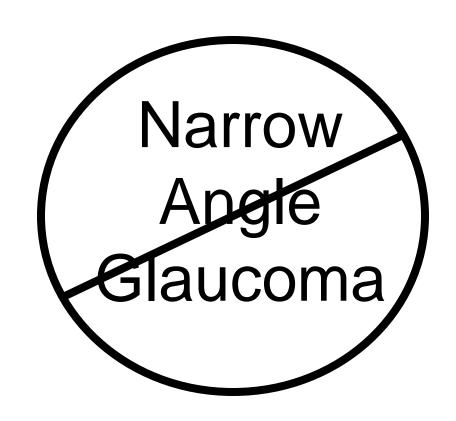




 Not getting enough pre-treatment...and posttreatment IOPs



 Not getting enough pre-treatment...and posttreatment IOPs



#### **CURRENT TERMINOLOGY**

- Primary angle closure suspect
- Primary angle closure
- Primary angle closure glaucoma
- Primary angle closure attack

#### PRIMARY ANGLE CLOSURE SUSPECT

- Pigmented trabecular meshwork blocked by iris
  - Extent of blockage not clear- about 180 degrees
- No PAS
- Disc and IOP normal
- Probe for symptoms of intermittent closure
- Not clear if LPI or observation is better

#### **PRIMARY ANGLE CLOSURE**

- Pigmented TM is blocked by iris for 180<sup>o</sup>
- Have either PAS or elevated IOP
- No disc damage or field loss
- Considered pathologic
- LPI recommended

### PRIMARY ANGLE CLOSURE GLAUCOMA

- Pigmented TM is blocked by iris for 180<sup>o</sup>
- Have either PAS or elevated IOP
- Glaucomatous neuropathy and field loss
- LPI recommended

### PRIMARY ANGLE CLOSURE ATTACK

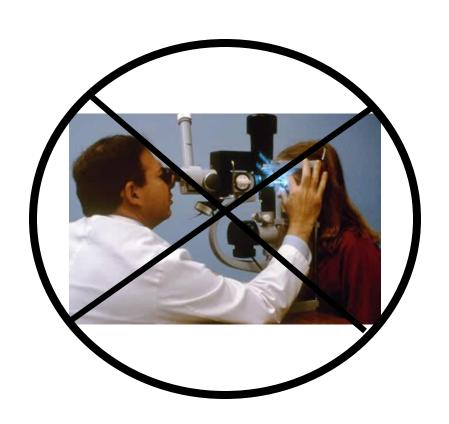
- Near complete apposition of iris to pigmented TM
- Classic signs and symptoms
  - Injection, vision loss, nausea, emesis, halos, corneal edema, elevated IOP, inflammation, mid-dilated fixed pupil
- Medical therapy, iridotomy, iridoplasty, trabeculectomy
  - Lens extraction?

Name	Irido- trabecular contact (> 180)	Increased IOP	PAS	GON	Acute Attack
PACS	+	-	-	-	-
PAC (CAC)	+	+/-	+/-		-
PACG (CACG)	+	+/-	+/-	+	-
AAC	+	+	+/-	+/-	+



## **MISTAKE TO AVOID**

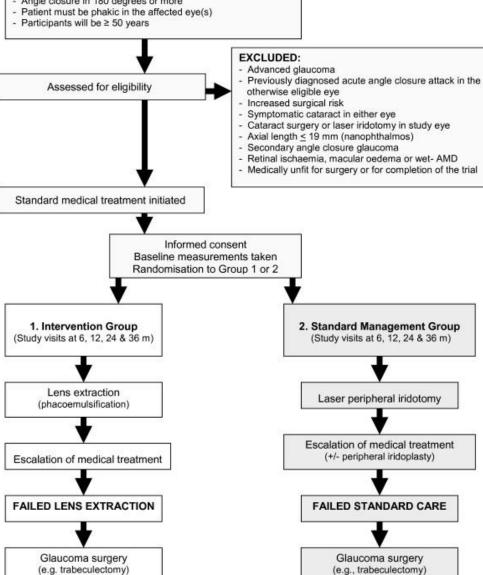
 Thinking LPI is the best management for angle closure glaucoma



#### INCLUSION CRITERIA:

- Diagnosis: (1) PACG or (2) PAC with IOP ≥ 30 mmHg at diagnosis
- Newly diagnosed, (i.e. either (i) untreated or (ii) under medical treatment for six months or less)
- Angle closure in 180 degrees or more

### **EAGLE** study



### **EAGLE STUDY**

- Removal of clear lenses in eyes with PACG with IOP > 21 mm or eyes with PAC (without glaucoma) and IOP > 30 mm. 419 patients. Findings included:
- Patients undergoing phaco lens extraction had far fewer IOP controlling meds compared to LPI
- Only 1 patient needed trabeculectomy after phaco whereas 24 patients in the LPI group needed trabeculectomy

### **ACUTE ATTACK MANAGEMENT**

- Lens removal has been found to be a more effective treatment for an attack of acute primary angle closure (APAC) than laser iridotomy.
- Compared with the eyes that underwent iridotomy, those treated with phacoemulsification experienced dramatically fewer IOP elevations, had lower mean IOPs, required fewer medications, and had deeper angles following lens removal.
- In APAC eyes presenting with an IOP greater than 55 mm Hg, phacoemulsification was a "definitive treatment" for preventing subsequent IOP elevations

# YOU ARE DOING IT CORRECTLY IF YOU RECOGNIZE THE IMPORTANCE OF LENS REMOVAL

- EAGLE study clearly shows that clear lens extraction is preferred management of chronic angle closure.
- Acute angle closure attack: break the attack medically and get the lens removed within a month.

# TO ZAP OR NOT TO ZAP...THAT IS THE QUESTION

### **ZAP STUDY**

Ophthalmic Epidemiol. 2010 Oct;17(5):321-32. doi: 10.3109/09286586.2010.508353.

Design and methodology of a randomized controlled trial of laser iridotomy for the prevention of angle closure in southern China: the Zhongshan angle Closure Prevention trial.

Jiang Y1, Friedman DS, He M, Huang S, Kong X, Foster PJ.

Author information

#### Abstract

PURPOSE: To summarize the design and methodology of a large-scale trial in southern China, the Zhongshan Angle Closure Prevention (ZAP) trial. This trial will determine if laser iridotomy (LI) is superior to no treatment for managing Chinese people who are Primary Angle Closure Suspects (PACS). In this trial, PACS was defined as having 6 or more clock hours of angle circumference in which the pigmented trabecular meshwork was not visible under static gonioscopy in both eyes without elevated intraocular pressure, peripheral anterior synechiae or glaucomatous neuropathy.

METHODS: Subjects were recruited from an urban district in Guangzhou. The target sample size was 870. Persons 50 years of age and older with 20/40 or better vision in both eyes identified as having 6 or more clock hours of angle circumference in which the pigmented trabecular meshwork was not visible under static gonioscopy in both eyes were enrolled. Each subject was randomized to undergo LI in one eye with the fellow eye left untreated. Follow up is planned for a minimum period of 3 years. Baseline examination included tonometry, limbal chamber depth grading, gonioscopy, fundus photography, anterior segment coherence tomography, ultrasound A scan, ultrasound biomicroscopy, specular microscopy and dark room provocative testing. Endpoints for the study include developing elevated intraocular pressure, peripheral anterior synechiae or experiencing acute primary angle closure.

**CONCLUSION:** The ZAP trial will determine if LI is safe and effective at preventing pathological angle closure in asymptomatic eyes with narrow angle configurations on gonioscopy. It will also provide data on what happens to untreated eyes in PACSs. Data collected at baseline will also help identify those at high risk for developing primary angle closure and primary angle closure glaucoma.

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

Mingguang 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

### **ZAP RESULTS**

### 889 angle closure suspects

- One eye received LPI and the other observation

### Outcomes at 72 months:

- IOP > 24 mm; development of at least 1 clock hour of PAS, or acute attack.

### Results:

- Outcome in 4.19 per 1000 eyes/yr in treated and 7.97 per 1000 eyes/yr (19 treated eyes and 36 untreated eyes)
  - Acute angle closure: 5 patients untreated, 1 treated (3 control eyes and one LPI eye were after dilation)
- Prophylactic LPI statistically significantly reduced incidence of ACG, but the actual event was very infrequent and hard to justify widespread use.
- Very low rate of angle closure in suspect eyes (<1%/yr); prophylactic LPI did confer 47% risk reduction
- Authors determined that laser peripheral iridotomy was not justified

> Am J Ophthalmol. 2021 Jun 28;S0002-9394(21)00343-3. doi: 10.1016/j.ajo.2021.06.018. Online ahead of print.

# The impact of pharmacological dilation on intraocular pressure in primary angle closure suspects

Lanhua Wang <sup>1</sup>, Wenyong Huang <sup>1</sup>, Xiaotong Han <sup>1</sup>, Chimei Liao <sup>1</sup>, Ling Jin <sup>1</sup>, Mingguang He <sup>2</sup>

Affiliations + expand

PMID: 34197780 DOI: 10.1016/j.ajo.2021.06.018

**Conclusions:** Post-dilation IOP elevation is similar among treated and untreated eyes, and the risk of developing AAC is very low even among PACS. Routine LPI before pupil dilation for PACS people is not recommended.

to 70 years with LPI in one randomly selected eye and a fellow untreated eye were included. All participants underwent comprehensive examinations before and at 2 weeks, 6 m, 18 m, 36 m, 54 m, and 72 m after LPI. IOP was measured using Goldmann applanation tonometry before and 1 hour after pharmacological dilation.

**Results:** The mean pre-dilation IOP in the untreated eyes was 14.8±2.7 mmHg, which increased to 16.4±2.7 mmHg after pharmacological dilation (p<0.001). The treated and untreated eyes had similar pre-dilation and post-dilation IOP (all p>0.05). The average post-dilation IOP elevation was 1.5 mmHg in the treated eyes and 1.6 mmHg in the untreated eye without significant differences (p=0.802). Lower pre-dilation IOP (p<0.001), smaller AOD500 (p=0.001), smaller ARA500 (p=0.030), smaller TISA500 (p=0.043), and larger larea(p<0.001) were associated with post-dilation IOP elevation 5 mmHg and greater. Three untreated (1.04 per 1000 pupil dilation) and one treated eye (0.34 per 1000 pupil dilation) developed acute angle closure (AAC) after dilation during the 72 m follow-up.

**Conclusions:** Post-dilation IOP elevation is similar among treated and untreated eyes, and the risk of developing AAC is very low even among PACS. Routine LPI before pupil dilation for PACS people is not recommended.



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

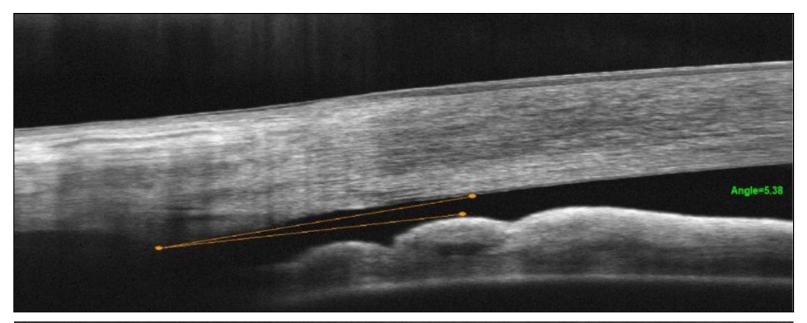
The Zhongshan Angle Closure Prevention Trial

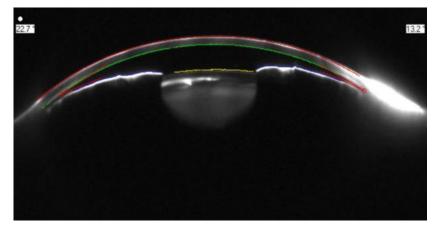
Benjamin Y. Xu, MD, PhD,<sup>1</sup> David S. Friedman, MD, PhD,<sup>2</sup> Paul J. Foster, FRCS(Ed), PhD,<sup>3</sup> Yu Jiang, MD,<sup>4</sup> Anmol A. Pardeshi, MS,<sup>1</sup> Yuzhen Jiang, MD, PhD,<sup>4</sup> Beatriz Munoz, MS,<sup>5</sup> Tin Aung, FRCS(Ed), PhD,<sup>6</sup> Mingguang He, MD,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

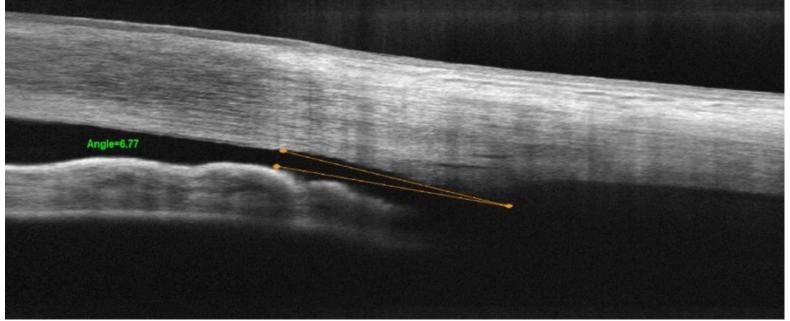
**Conclusions:** Superior LPI location results in significantly greater angle widening compared with temporal or nasal locations in a Chinese population with PACS. This supports consideration of superior LPI locations to optimize anatomic changes after LPI. *Ophthalmology 2021;* ■:1−8 © 2021 by the American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

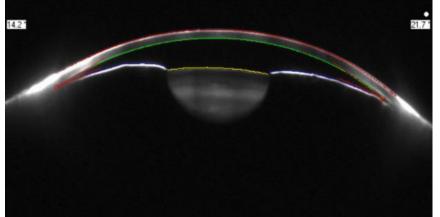
### **74 YOF**

- CC: Blurred vision OU
- BVA: +5.25-1.75x145 20/60; +5.50-0.25x45 20/20
- PERRL(-)RAPD
- Nuclear sclerotic cataracts OD>OS
- IOP 30 mm OD, 25 mm OS
- Narrow angles
- Gonio: No structures OD; ATM nasal and temporal OSotherwise no structures seen
- Fundus: no view undilated









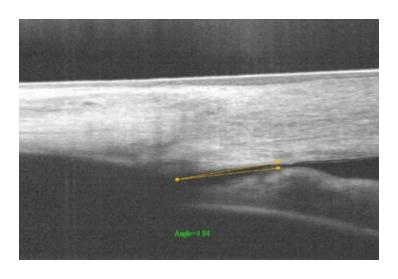
Assessment and Plan?

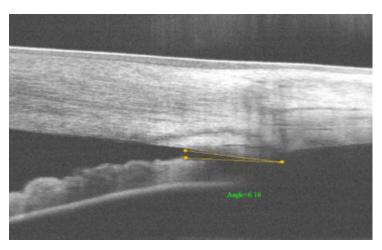
### **74 YOF**

- Diagnosis: Primary chronic angle closure (glaucoma?)
- Plan: sampled PGA and set for cataract consult
- IOP at consult: 17 mm OD, OS
- Surgical measurements made (no dilation)- planned cataract extraction basic emme OD, then OS; CPM
- Pt cancelled surgery twice- reasons unknown.

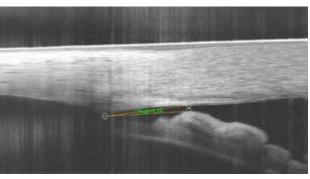
# YOU CAN LEAD AN ANGLE CLOSURE TO OSMOGLYN, BUT YOU CAN'T MAKE HIM DRINK

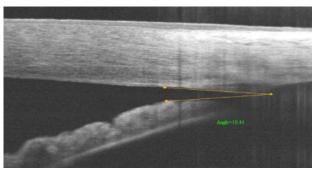
- 30 YOF
- 2018: Referred for narrow angles
- BVA: +2.00 DS 20/20; +1.25 DS 20/20
- Gonio: "slit OU" Grade 1 OU
- IOP 18 mm OU
- Dx: PACS OU
- Plan LPI OU





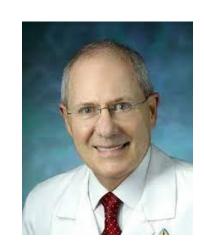
- Follow up (2018)
- No appreciable change after LPI
- Gonio: grade 1; no PAS, double hump sign
- Dx: plateau iris syndrome
- Plan: Discussion iridoplasty, pilocarpine, lens extraction
- Observation recommended
- Other glaucoma specialists may have different approach
  - welcome to second opinion
- Do not start any new medication without clearance
  - Cold and allergy meds





- 2022: Emergently presents with migraine aura
- Records reviewed
- No resolution to issue
- Forgot about the medication admonition
- Has been told that she can never be dilated
- She is worried and doesn't know what to do
- So, what do we do?





- Can this 30 YO go the rest of her life without dilation?
- Really no great options (Pilo? Iridoplasty? Lens extraction at 30 years old?)
- Hasn't had an attack yet
- Harry Quigley, MD, "You just don't know, so sometimes you gotta bite the bullet, dilate, and see what happens. But you don't do it on Friday at 4 pm. You do it Friday at 9 am and tell them that they will be here until lunch time"

- Returns 8:30 am Tuesday
- IOP: 22 mm OD, 22 mm at 8:30 am; pt informed of risks; dilated 0.5% tropicamide
  - Diamox and Combigan ready
    - It works- trust me
- IOP: 22 mm OD, 22 mm OS at 9:30 am
- IOP: 22 mm OD, 23 mm OS at 1:15 pm; pupil in mid-dilated state
- Fundus normal OU; C/D 0.2 OU
- Pt educated si/sx AACG
- Will follow annually

### **MISTAKE TO AVOID**

Thinking that glaucoma causes collateral disc vascularization

### **COLLATERAL VESSELS**

- Historically and often incorrectly called "Optociliary shunt vessels"
  - They are not opto, not ciliary, and not a shunt
- Collateral (not shunt)
- May be on the optic disc or in the retina
- Pre-existing anastamotic communications involving deep capillary beds through which blood flows in response to vascular occlusion
- Retinochoroidal: Typically venule to venule in retina or retinal venule to choroidal venule
- Highly indicative of retinal vascular occlusion





### **COLLATERAL VESSELS**

- Non-fenestrated and non-leaking
- Common after vein occlusion
- Occurs from nerve sheath meningioma
  - Collaterals, optic atrophy, vision loss
- Has been said to occur secondary to glaucoma. Urban legend/ error.
- Acquired collateral vessels occur in association with ophthalmic conditions that produce impaired retinal venous outflow
  - Where is the venous outflow stagnation in glaucoma?
- Glaucoma and vein occlusion are commonly occurring co-morbidities.
- Glaucoma patients who have collaterals likely have had a previous vein occlusion.

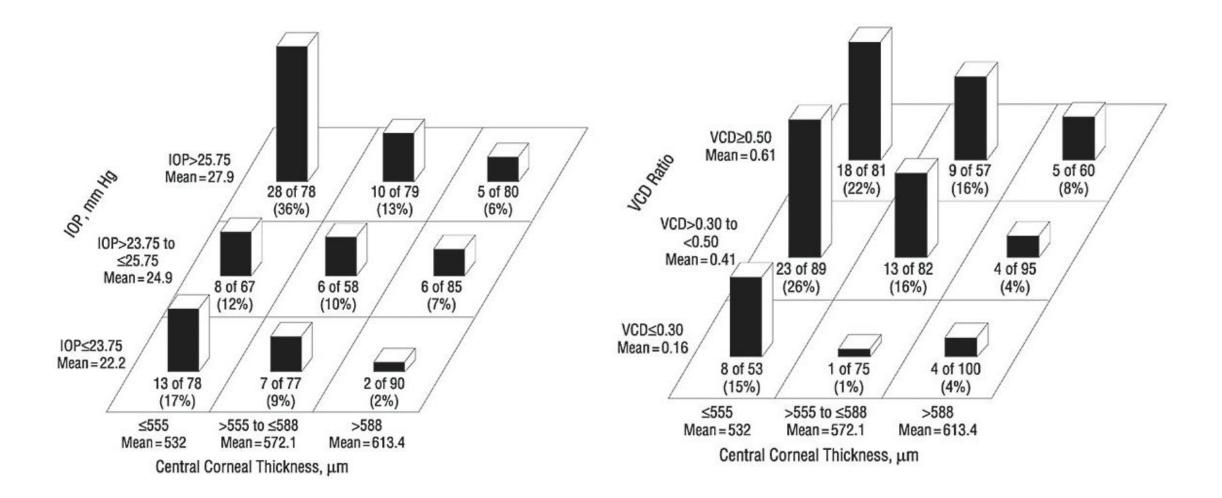






## **MISTAKE TO AVOID**

Correcting IOP based upon pachymetry



Central Corneal Thickness (Microns)	Adjustment in IOP (mm Hg)		
445	+7		
455	+6		
465	+6		
475	+5		
485	+4		
495	+4		
505	+3		
515	+2		
525	+		
535	+		
545	0		
555	-1		
565	-1		
575	-2		
585	-3		
595	-4		
605	-4		
615	-5		
625	-6		
635	-6		
645	-7		

## Why CCT-based IOP correction is flawed

### Can't we calculate "true IOP" using pachymetry (CCT)?

NO! Central Corneal Thickness based IOP adjustment algorithms

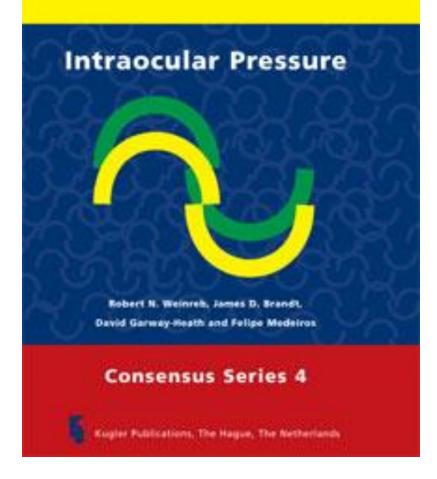
DO NOT WORK. These formulas have been scientifically discredited and the glaucoma opinion leaders of the world are cautioning clinicians against using them.

As such, CCT correction tables and pachymeters and tonometers that provide CCT-based "corrected" IOP values are OBSOLETE.

## "Correction nomograms that adjust GAT IOP based solely on CCT are neither valid nor useful in individual patients"

- Pg 18. Robert N. Weinreb, James D. Brandt, David Garway-Heath and Felipe Medeiros World Glaucoma Association on Intraocular Pressure; Consensus Series 4; May 5, 2007

### **World Glaucoma Association**



- 6. Correction nomograms that adjust GAT IOP based solely on CCT are neither valid nor useful in individual patients.
  Comment: A thick cornea gives rise to a greater probability of an IOP being over-estimated (and a thin cornea of an IOP being under-estimated), but the extent of measurement error in individual patients cannot be ascertained
- 7. Measurement of CCT is important in assessing risk for incident glaucoma among ocular hypertensives in the clinical setting, though the association between CCT and glaucoma risk may be less strong in the population at large.

from the CCT alone.

### **MISTAKE TO AVOID**

Not recognizing patients who will likely do well

### **CLINICAL PEARL**

- You can only call a glaucoma patient "well controlled" in retrospect
- Some patients progress slowly without treatment and some progress rapidly, even with treatment
  - You don't know who is who until you follow up over time

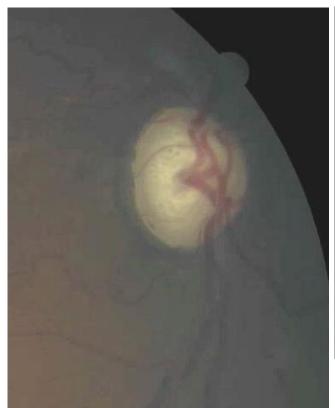


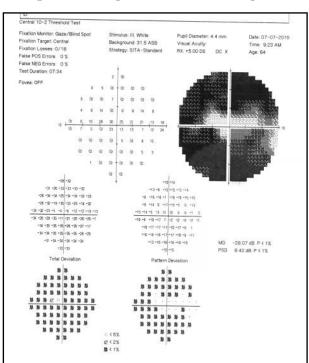


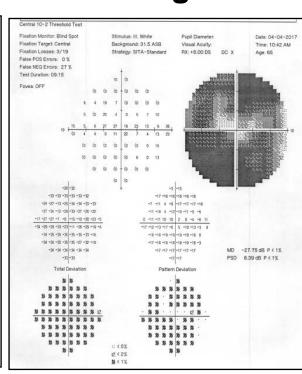
# PATIENTS I WORRY LESS ABOUT

## **66 YOM**

- Starting IOP 58 mm Hg; CCT 536
  - 20/30 OD; OS not seeing due to vascular occlusion
- Stepped regimen: Brimonidine, dorzolamide/timolol, latanoprost, pilocarpine (GlaucAll)- IOP 14 mm Hg







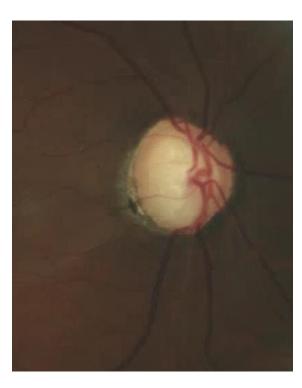
Baseline

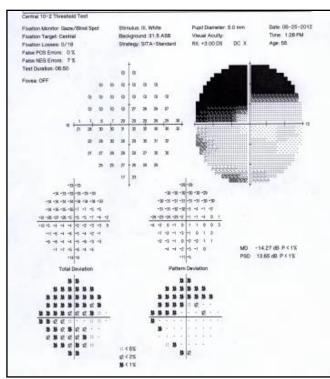
- Very high peak IOP
- Exceptional IOP reduction (75%) ©

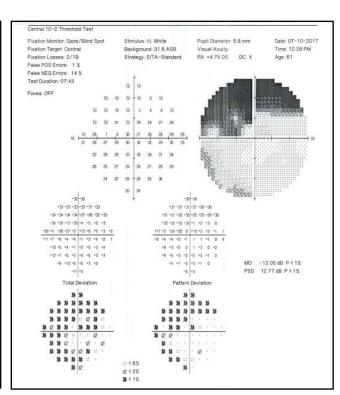
2 years

## **61YOM**

- IOP 30 mm; CCT 545
- Latanoprost, dorzolamide/timolol 12 mm







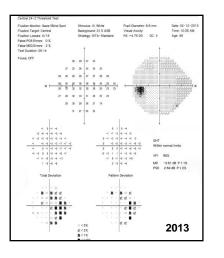
- High peak IOP
- Excellent IOP reduction (60%) ☺

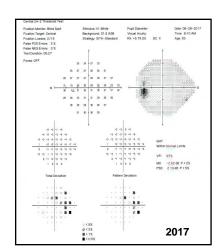
Baseline

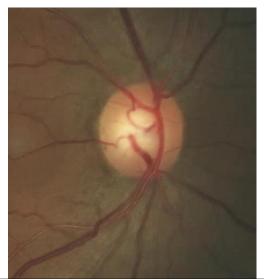
5 years

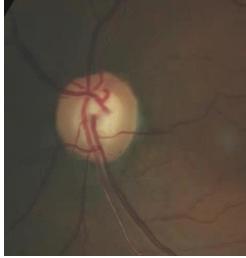
### **53 YOM**

- Peak IOP: 32 mm OD, 43 mm OS; CCT 453
   OD, 446 OS
- Latanoprost: 15-18 mm OD, 18-22 mm OS
  - Recently added dorzolamide

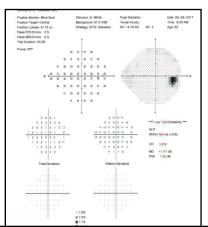








- High peak IOP
- Significant initial IOP reduction with 1 med
- Low med load

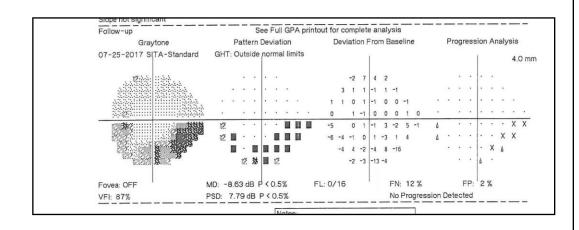


# **63 YOF: GLAUCOMA OS X 5 YEARS**

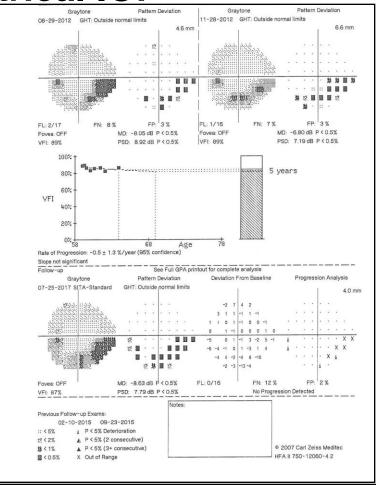
IOP typical range: 14-18 OD; 15-18 OS; CCT: 556 OD; 543 OS

Unilateral disease; symmetrical IOP

Pt chooses observation.



- True "normotensive" range
- Moderate disease not threatening fixation
- Stable ©



# **MISTAKE TO AVOID**

 Not identifying patients who likely will not do well.

# WHICH PATIENTS REPRESENT UNSUSPECTING DANGER?



## **65 YOM**

- Peak IOP 22 mm OD, 29 mm OS; CCT 560
  - Followed 7 yrs
- Latanoprost, dorzolamide/timolol, brimonidine- 15 mm OD, 14 mm OS

■ Time to MMT: 3 ½ years





to MMT

• High med load for modest reduction

Peak IOP

terribly high

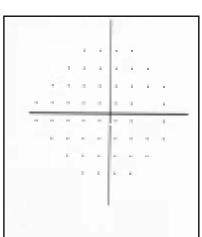
Short duration

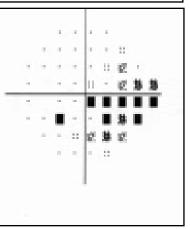
OS

not

Fields unchanged; possible disc change OS

OD





## **55 YOF**

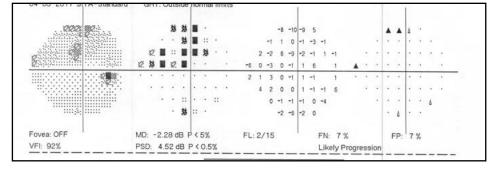
#### Initial: Peak IOP??- treated since age 35

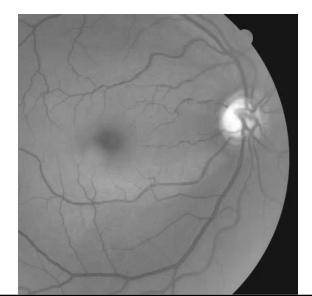
- Timolol; CCT 472 OD, 497 OS; Disc change OD 2010

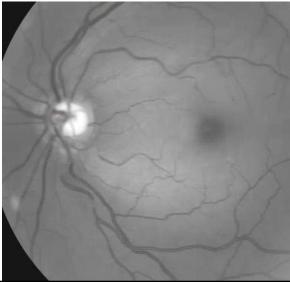
- Currently: latanoprost, dorzolamide/timolol, brimonidine;

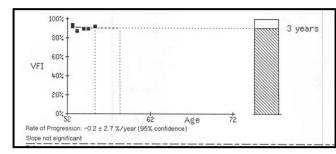
15 mm OU

Followed 15 yrs









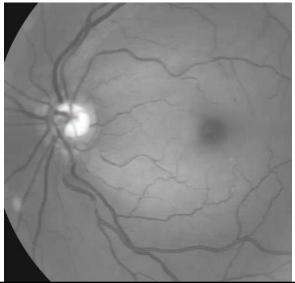
- Young age
- Long duration of treatment
- MMT
- Thin cornea 🟻

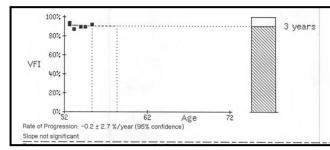
# **55 YOF**

#### Shows late progression on field OU at IOP of 15 mm OU

- Difficulty arranging surgery due to insurance
- Pt had to leave country 4 months
- Switched latanoprost with Rocklatan (other meds continued)
- IOP now 09 mm OD, 10 mm OS





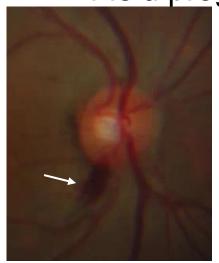


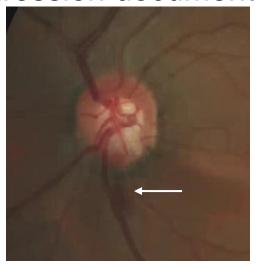
- Young age
- Long duration of treatment
- MM1
- Thin cornea 8

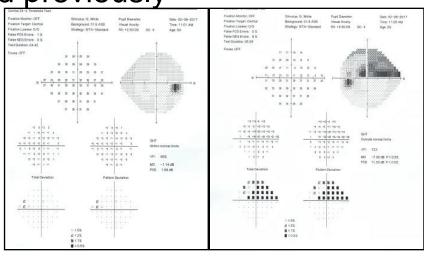
# **53 YOF**

- Peak IOP: 20 mm OD, 22 mm OS; CCT: 510
   OD, 508 OS
- Treated IOP: 12-15 mm OD, 12-16 mm OS
  - Brimonidine, latanoprost, dorzolamide/timolol

- Field progression documented previously







Low baseline IOP (low 20s), MMT to achieve 'modest IOP reduction, bilateral recurrent disc hemorrhages 😢

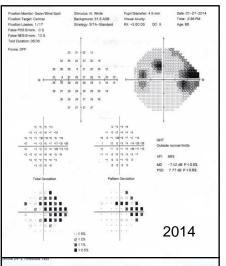
# 69 YOF: POAG OU X 11 YEARS

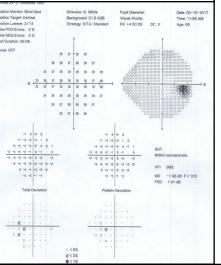
Peak IOP: 20 mm OU; CCT: 540 OD, 532 OS

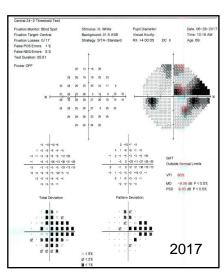
- Dorzolamide/timolol; latanoprost OU; IOP- 15 mm OD,

14 mm OS









- Bilateral disc hemorrhages
- Low peak/baseline IOP
- Modest pressure reduction
- High med load
- Field/ disc change ⊗

# THINGS THAT GIVE ME COMFORT

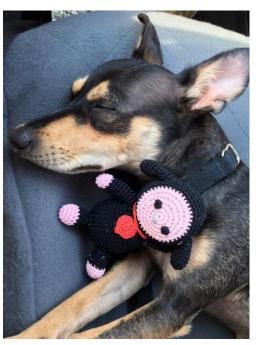












## OTHER THINGS THAT GIVE ME COMFORT

- High initial peak IOP
  - 30s and 40s better than low 20s
- Significant IOP reduction
  - Regardless of disc/ field status
- Good initial response to one medication
- Minimal medications
- High peak IOP and significant medical

response

# THINGS THAT MAKE ME UNCOMFORTABLE









# OTHER THINGS THAT MAKE ME UNCOMFORTABLE

- Exfoliation
- A STATE OF THE PARTY OF THE PAR
- Disc hemorrhages



- Rapid escalation in therapy
  - Adding 2 meds w/i 3 years
- Low peak IOP
  - Low to mid 20s bad

Portends worse prognosis

- Mid teens- not so bad
- Poor initial IOP reduction
- Low peak IOP and poor initial IOP reduction





### **ODE TO GLAUCOMA TREATMENT**

When the pressure starts high and the treated drop great,

Likely a good outcome is to be the fate.

Compliance, exfoliation and disc hemorrhage must be watched,

So the case doesn't get botched.

Most patients can be predicted,

And your Zen won't be afflicted

But some patients will surprise,

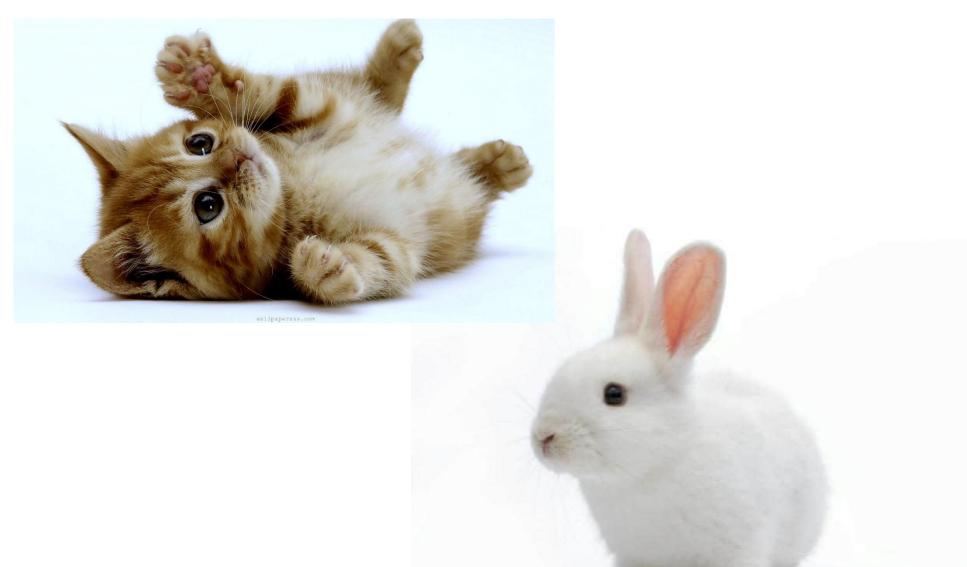
And cause your blood pressure to rise.

Lowering 22 down to 18 is not enough,

Go for 50% so they don't snuff.



# ...MAY YOU HAVE NOTHING BUT KITTENS AND BUNNIES



#### **STAY SAFE EVERYONE**

