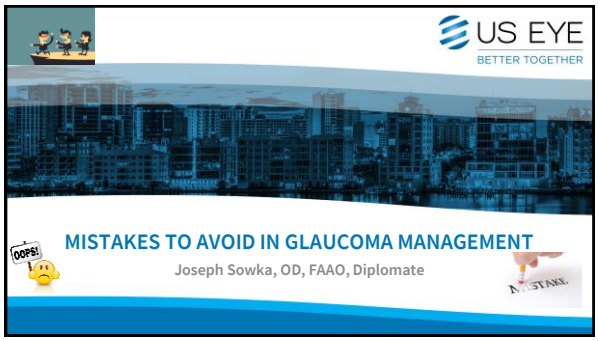
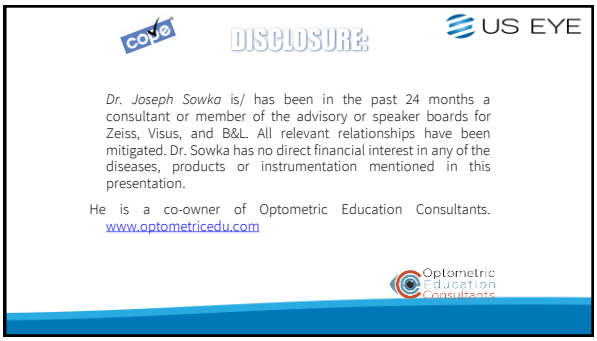




MISTAKE TO AVOID

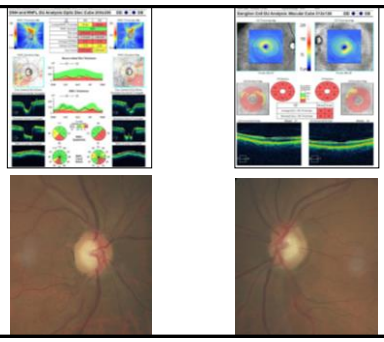
- Not recognizing a neurologic field
- Thinking glaucoma causes optic disc pallor
- Diagnosing NAAION in glaucoma patients
- Not recognizing when the OCT is wrong
- Treating red disease
- Not treating real disease
- Changing therapy based upon one bad IOP or field
- Not getting enough pre-treatment…and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

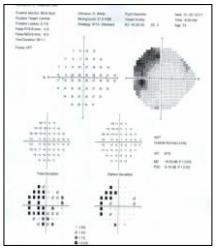
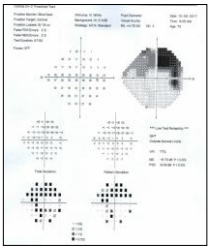
MISTAKE TO AVOID

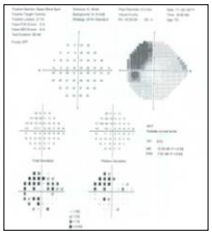
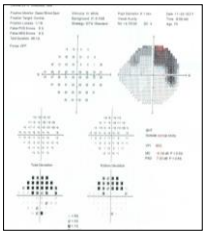
- Not recognizing a neurologic field

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



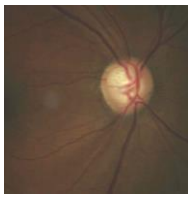
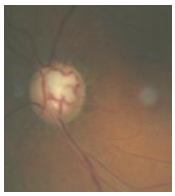
FINDINGS: There is a large T1 hypointense and T2 iso- to hyperintense lesion extending between the sella into the suprasellar region showing heterogeneous enhancement on the post-contrast images measuring 2.7 cm craniocaudal x 2.1 cm AP x 2 cm transverse. Findings are compatible with a pituitary macroadenoma. 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.

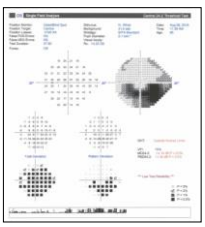
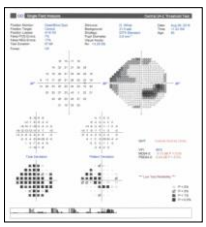
The globes are symmetric. There is no lens dislocation. The post-septal soft tissues are preserved with no definite intra- or extraconal mass. The optic nerves are symmetric at the orbital level showing no abnormal enhancement.

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

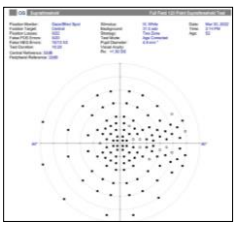
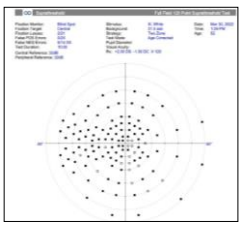



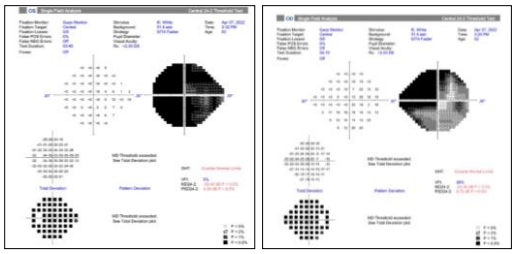
Peak IOP unknown; s/p SLT OU and on latanoprost at first visit.

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

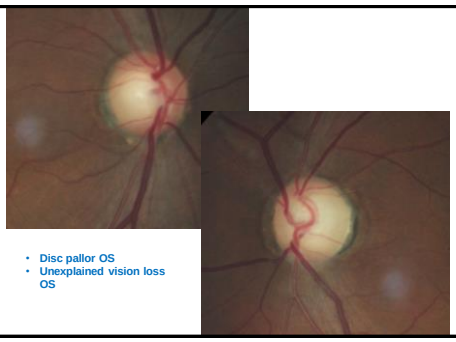
Pallor in excess of cupping indicates something other than, or in addition to, glaucoma

RULE

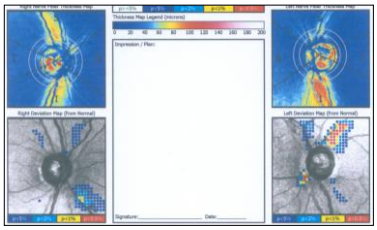
Nothing notches a nerve like glaucoma

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

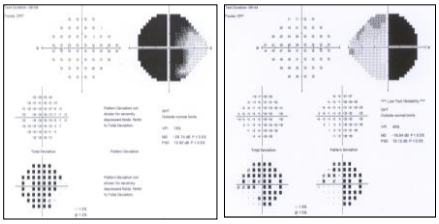
- **54 YO Nigerian man**
- **Referred for glaucoma management**
- **Told he had glaucoma 6 years earlier- no Tx**
- **6/9 OD; HM OS**
  - Vision loss from glaucoma- not coming back
- **30 mm Hg OD; 23 mm Hg OS**
  - Lumigan- 17 mm Hg OD, 15 mm Hg OS



- **Disc pallor OS**
- **Unexplained vision loss 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

Joseph Sowka, OD

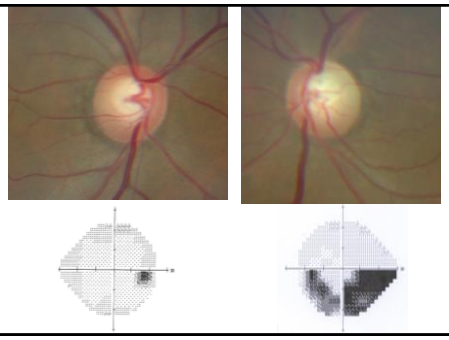
MISTAKE TO AVOID

- **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.

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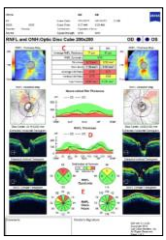
ISSUES IN IMAGING

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

33

WHAT TO LOOK FOR WHEN INTERPRETING OCT SCANS

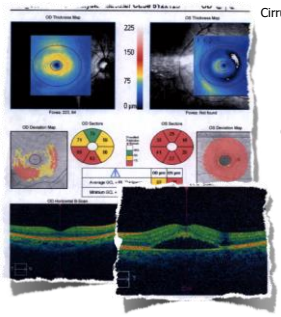
- Quality score
- Illumination
- Focus clarity
- Image centered
- Any signs of eye movement
- Segmentation accuracy
- B Scan Centration
- Missing data
- Media issues
- Maculopathy for GCC scans



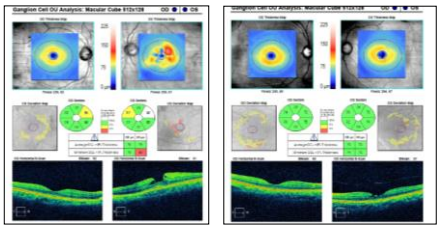
34

EYE MOVEMENT

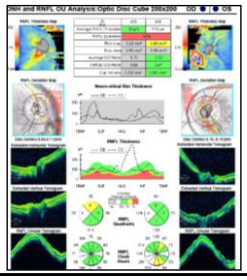
Cirrus



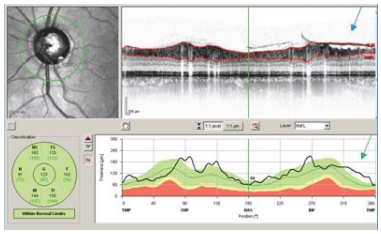
Accidentally find CSC when looking for glaucoma



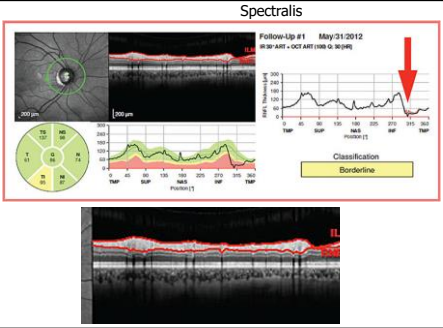
IF YOU THINK DEVICES MEASURE TISSUE ACCURATELY EVERY TIME…

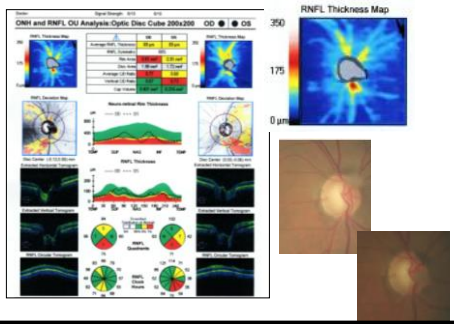


Spectralis

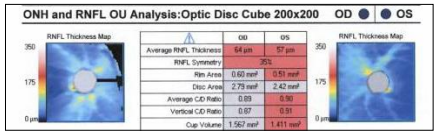


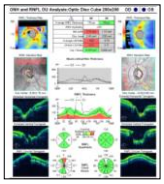
Spectralis

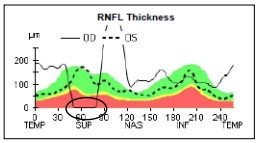




FLOOR EFFECT







Don't make clinical decisions based upon bad data

MISTAKE TO AVOID

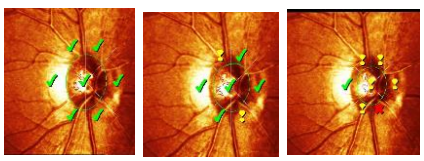
- **Treating red disease**

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**

Sherlock, NS. 2005. *Journal of Irreproducible Results and Senseless Studies*

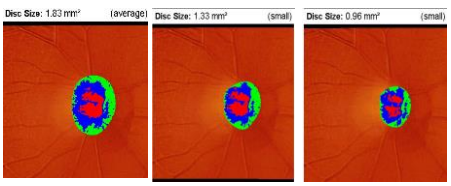
SCANNING LASER OPHTHALMOSCOPY EXAMPLE OF RED DISEASE



First Visit | Follow up visit #1 | Follow up visit #2

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

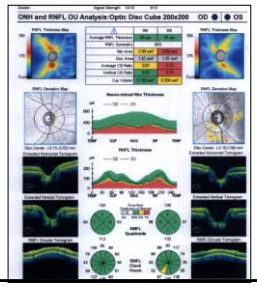
WITHIN 15 MINUTES! HRT DISC SIZING ARTIFACT

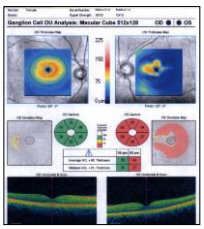


HELP! THE DIAGNOSTIC IMAGING DOESN'T AGREE WITH MY DIAGNOSIS!

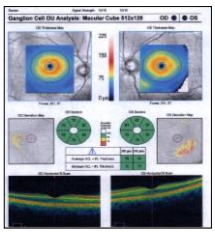
- **Low risk OHTN**
- **Local OD wants imaging for baseline**

OCT RNFL NORMAL…



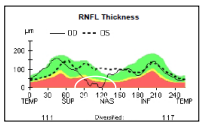


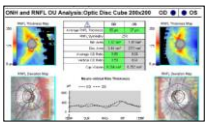
…but markedly abnormal GCC OS

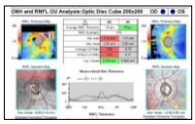


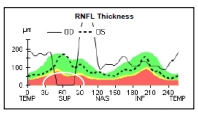
Same patient, same day, same quality, GCC now normal

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





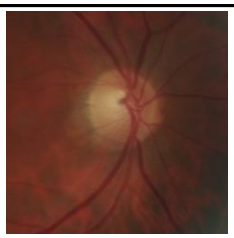


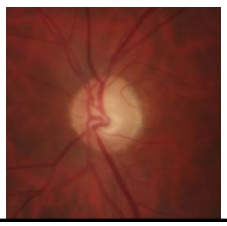


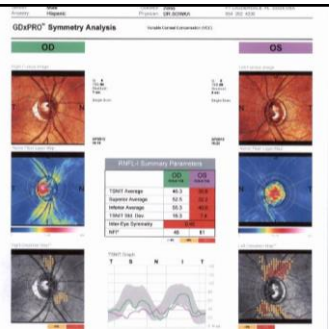
Don't make clinical decisions based upon bad data

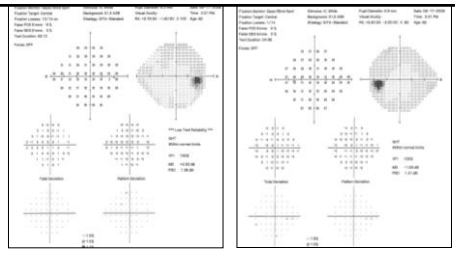
CASE: 62 YOHM

- **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**









MISTAKE TO AVOID

- Not treating green disease

GREEN DISEASE– AN INSIDIOUS CLINICAL *ENTITY*

A glaucomatous process masquerading as non-disease

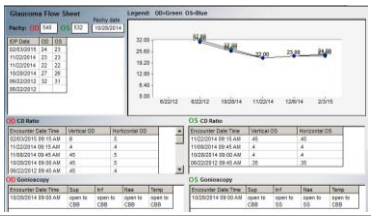
Afflicts inexperienced, poorly-educated doctors who simply want a machine to make all clinical decisions for them

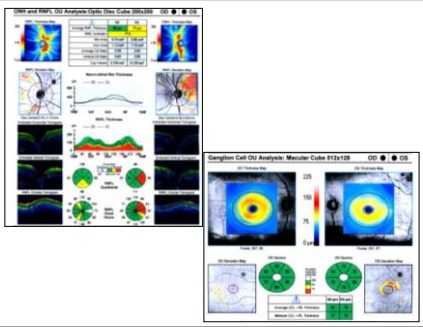
Debilitating to the patient and painful for the visual care provider, but a boon for malpractice attorneys

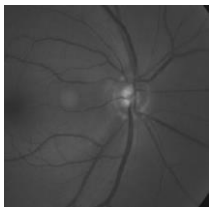
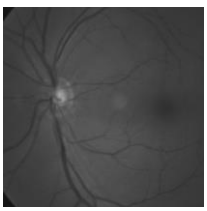
Sherlock NS. 2015. *Journal of Irreproducible Results and Senseless Studies*

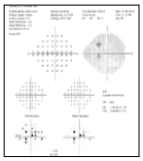
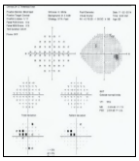
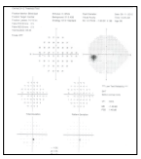
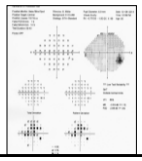
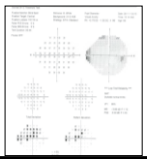
HELP! THE DIAGNOSTIC IMAGING DOESN'T AGREE WITH MY DIAGNOSIS!

- 56 YOM- Glaucoma suspect since 2012





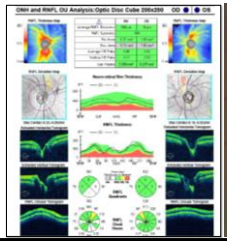
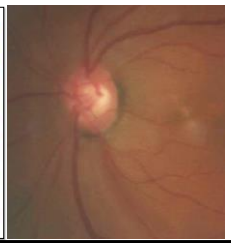



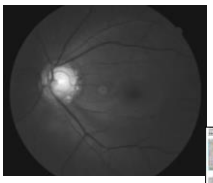
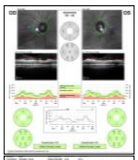
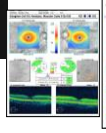
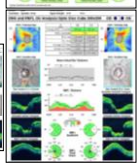
Is this person really a glaucoma 'suspect'?

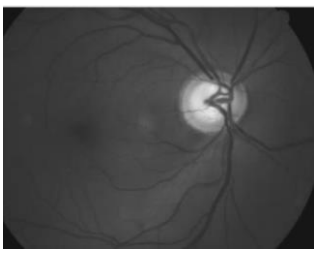
An example of Red Disease OS and Green Disease OD

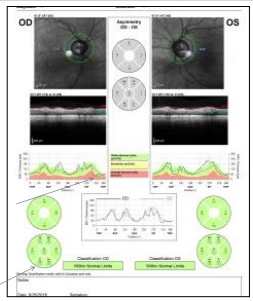
GREEN DISEASE

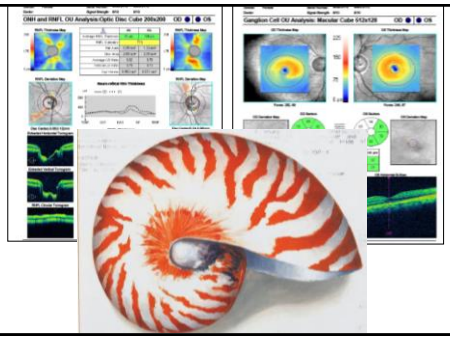



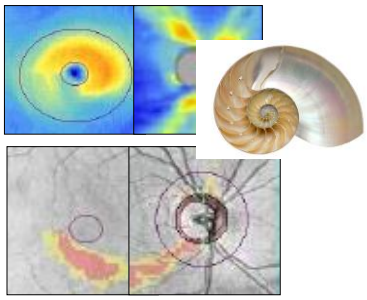
GREEN DISEASE









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

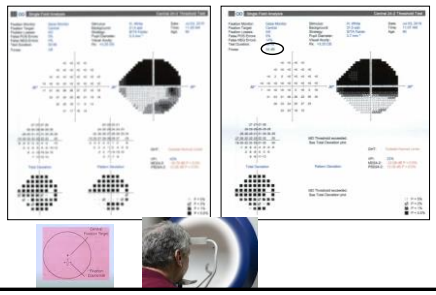
CAUTIONS ABOUT IMAGING

- 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

MISTAKE TO AVOID

- Changing therapy based upon one bad IOP or field

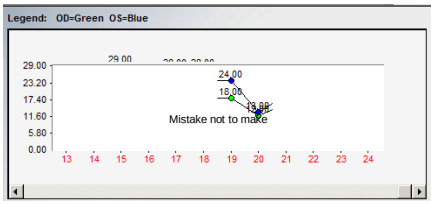
Is this patient getting worse?



MISTAKE TO AVOID

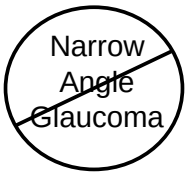
- Not getting enough pre-treatment…and post-treatment IOPs

MISTAKE TO AVOID



- Not getting enough pre-treatment…and post-treatment IOPs

MISTAKE TO AVOID



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 180^0
- 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 180^0
- 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?

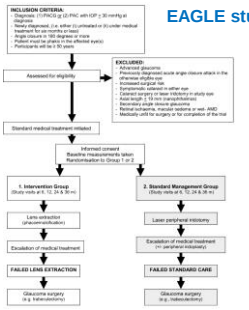
Name	Irido-trabecular contact (> 180)	Increased IOP	PAS	GON	Acute Attack
PACS	+	-	-	-	-
PAC (CAC)	+	+/-	+/-		-
PACG (CACG)	+	+/-	+/-	+	-
AAC	+	+	+/-	+/-	+

UCLA Stein Eye Institute | DOHENY EYE INSTITUTE Emanual, Parrish, Gedde; 2014

MISTAKE TO AVOID

- Thinking LPI is the best management for angle closure glaucoma

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

Azuara-Blanco A, Burr JM, Cochran C, et al. Effectiveness in Angle-closure Glaucoma of Lens Extraction (EAGLE) Study Group. The effectiveness of early lens extraction with intraocular lens implantation for the treatment of primary angle-closure glaucoma (EAGLE):The Lancet. Volume 388, No. 10052, p1389–1397, 1 October 2016.

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

Lam DS, Leung DY, Leung DY, et al. Randomized trial of early phacoemulsification versus peripheral iridotomy to prevent intraocular pressure rise after acute primary angle closure. Ophthalmology. 2008;115:1134-40.

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

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.

Lavanya R, Friedman DS, Hu SJ, Huang B, Kumar A, 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

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

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.

AMERICAN ACADEMY OF OPHTHALMOLOGY

Anatomic Changes and Predictors of Angle Widening after Laser Peripheral Iridotomy

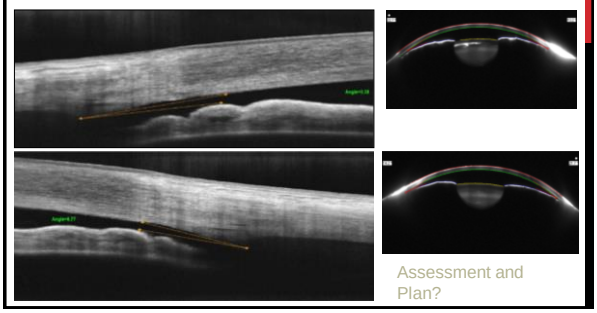
The Zhongshan Angle Closure Prevention Trial

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[7]

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 OS- otherwise no structures seen**
- **Fundus: no view undilated**



Assessment and Plan?

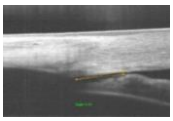
74 YOF

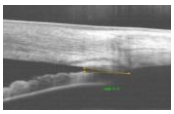
- **Diagnosis: Primary chronic angle closure (glaucoma?)**
- **Plan: sampled PGA and set for cataract consult**
- **IOP at consult: 17 mm OD, OS**
- **Surgical measurements made (no dilation)- planned cataract extraction basic emme OD, then OS; CPM**
- **Pt cancelled surgery twice- reasons unknown.**

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

BACKED INTO A CLOSURE CORNER

- 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





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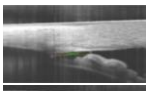
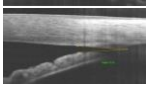




- Follow up (2018)
- No appreciable change after LPI
- Gonio: grade 1; no PAS, double hump sign
- Dx: plateau iris syndrome
- Plan: Discussion iridoplasty, pilocarpine, lens extraction
- Observation recommended
- Other glaucoma specialists may have different approach
  - welcome to second opinion
- Do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

BACKED INTO A CLOSURE CORNER

- 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?

BACKED INTO A CLOSURE CORNER

- 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*"

BACKED INTO A CLOSURE CORNER

- 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

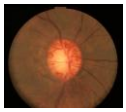
MISTAKE TO AVOID

- 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**



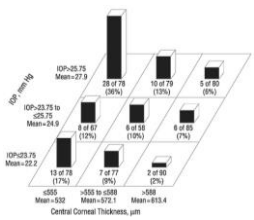


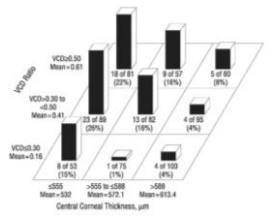
COLLATERAL VESSELS

- **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	+1
535	+1
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

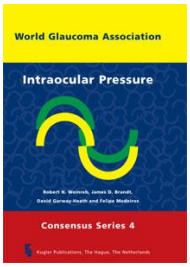
Can't we calculate "true IOP" using pachymetry (CCT)?

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



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 from the CCT alone.
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.

MISTAKE TO AVOID

- **Not recognizing patients who will likely do well**

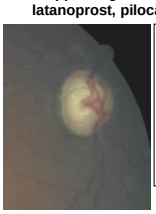
CLINICAL PEARL

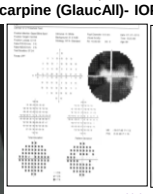
- **You can only call a glaucoma patient "well controlled" in retrospect**
- **Some patients progress slowly without treatment and some progress rapidly, even with treatment**
  - You don't know who is who until you follow up over time

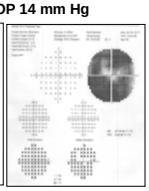
PATIENTS I WORRY LESS ABOUT

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**







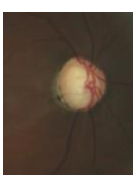
Baseline

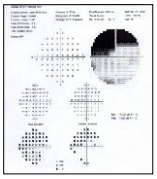
- **Very high peak IOP**
- **Exceptional IOP reduction (75%)**

2 years

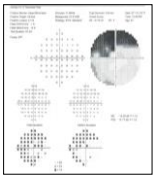
61YOM

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





Baseline

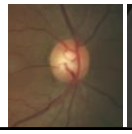


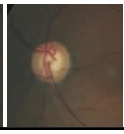
5 years

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

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

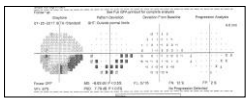


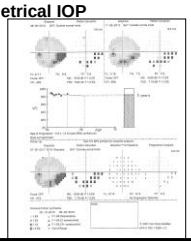


- High peak IOP
- Significant initial IOP reduction with 1 med
- Low med load

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

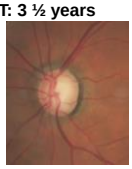
MISTAKE TO AVOID

- Not identifying patients who likely will not do well.

WHICH PATIENTS REPRESENT UNSUSPECTING DANGER?

65 YOM

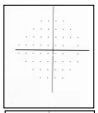
- Peak IOP 22 mm OD, 29 mm OS; CCT 560
  - Followed 7 yrs
- Latanoprost, dorzolamide/timolol, brimonidine- 15 mm OD, 14 mm OS
- Time to MMT: 3 ½ years

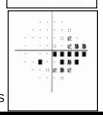


- Peak IOP not terribly high
- Short duration to MMT
- High med load for modest reduction

Fields unchanged; possible disc change OS

OD

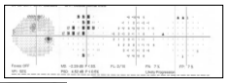


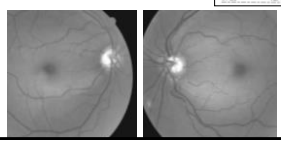


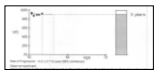
OS

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



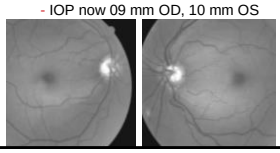


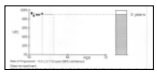


- Young age
- Long duration of treatment
- MMT
- Thin cornea ☹

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

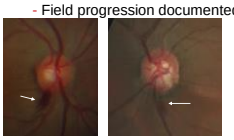


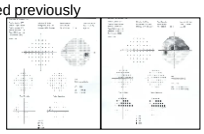


- Young age
- Long duration of treatment
- MMT
- Thin cornea ☹

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

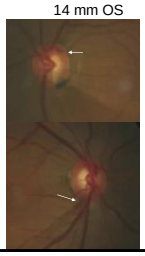


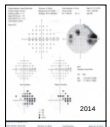


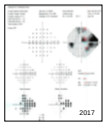
Low baseline IOP (low 20s), MMT to achieve 'modest IOP reduction, bilateral recurrent disc hemorrhages ☹

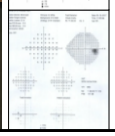
69 YOF: POAG OU X 11 YEARS

- **Peak IOP: 20 mm OU; CCT: 540 OD, 532 OS**
  - Dorzolamide/timolol; latanoprost OU; IOP- 15 mm OD, 14 mm OS



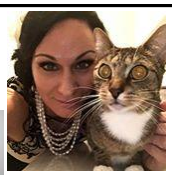






- Bilateral disc hemorrhages
- Low peak/baseline IOP
- Modest pressure reduction
- High med load
- Field/ disc change ☹

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 
- Disc hemorrhages 
- Rapid escalation in therapy
  - Adding 2 meds w/i 3 years
- Low peak IOP
  - Low to mid 20s bad
  - Mid teens- not so bad
- Poor initial IOP reduction
- Low peak IOP and poor initial IOP reduction


Portends worse prognosis

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.

Joseph Sowka, OD

BE AWARE OF THE GRAY AREA WHERE DANGER LURKS…

…MAY YOU HAVE NOTHING BUT KITTENS AND BUNNIES


STAY SAFE EVERYONE