CLINICAL DISCUSSIONS IN GLAUCOMA

JOSEPH SOWKA, OD
BRAD SUTTON, OD

HOW DO YOU USE OCT, HYSTERESIS, ELECTRO DIAGNOSTICS, AND OTHER TECHNOLOGIES?

SITA FASTER – TESTS IN 2 MINUTES OR LESS WITHOUT COMPROMISE TO TEST RESULTS

Two minute test for near normal patients

- ~50% faster than SITA Standard; ~30% faster than SITA Fast
- Clinically equivalent to SITA Fast and Standard
- Same SITA algorithm and normative data as Standard and Fast
- Removes unnecessary “dead time” during the test
- No Blind Spot or False Negatives
  - Uses Gaze Monitoring and False Positives for test quality monitoring

Mixed SITA GPA Reports

- Allows mixing all SITA test strategies for GPA reports
- Helps immediately adopt SITA Faster
- Clinical equivalence of tests allows intermixing

DISCLOSURE:

Joseph Sowka, OD is/has been a Consultant/ Speaker Bureau/ Advisory Board member for Novartis, Alcon, Zeiss, Allergan, Glaukos, and B&L. He is a co-owner of Optometric Education Consultants.

Brad Sutton, OD: Nothing to disclose

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

IS SITA FASTER ANY GOOD? ARE THE VISUAL FIELD PROGRAMS INTERCHANGEABLE?
MONOCULAR TRIALS- YES OR NO?

MONOCULAR TRIALS

- Medication added to one eye with the fellow eye being untreated for the initial period of medication evaluation as a “control”.
  - Thus, if the IOP lowered in the ‘treated’ eye compared to the untreated ‘control’ eye, the medication was deemed effective
- Concept was based upon a number of assumptions:
  - The diurnal IOP is identical between eyes
  - The diurnal IOP is similar between days, weeks and months
  - The response of a medication in one eye is identical to the response in the other
- Also remember regression to the mean.
  - That is, if IOP is very high, the next reading could be much lower merely by chance. In this case, medication addition may seem effective and it truly is not.

Asymmetry of 24-Hour Intraocular Pressure Reduction by Topical Ocular Hypotensive Medications in Fellow Eyes

Terry L. Le, MD, MS*

Purpose: A core assumption for the 2-eye therapeutic trial of ocular hypotensive medications is the evaluation of 24-hour intraocular pressure (IOP) in paired eyes. This assumption was evaluated for 24-hour IOP reduction in patients who were monocular or bilateral.

Methods: Patients in 4 hypertensive control groups were divided into 3 treatment groups: 1) Topical timolol, 2) Topical dorzolamide, and 3) Topical dorzolamide plus brimonidine.

Results: The diurnal IOP was significantly lower in the monocular group compared to the control groups. The IOP reduction in the monocular group was also significantly greater than the control groups.

Conclusion: Monocular trials are a valid method for evaluating the 24-hour IOP reduction by ocular hypotensive medications.

Short-Term Repeatability of Diurnal Intraocular Pressure Patterns in Glaucamatosus Individuals

Terry Le, MD, MS, N. Wisnovsky, MD, Stephen Wisnovsky, MD

Purpose: To evaluate the short-term repeatability of diurnal intraocular pressure (IOP) patterns in eyes with primary open-angle glaucoma (POAG).

Methods: Forty-seven subjects with treated POAG were enrolled in the study. Four IOP measurements were obtained in each eye at 4- to 6-hour intervals over a 24-hour period.

Results: The repeatability of diurnal IOP patterns in eyes with POAG was excellent. The coefficient of variation was less than 10% in all cases.

Conclusion: Diurnal IOP patterns in eyes with POAG are highly repeatable over a 24-hour period.

Diurnal Intraocular Pressure Patterns are Not Repeatable in the Short Term in Healthy Individuals

Terry Le, MD, MS, Robert N. Wisnovsky, MD, Stephen R. Wisnovsky, MD

Purpose: To evaluate the short-term repeatability of diurnal intraocular pressure (IOP) patterns in eyes of subjects without glaucoma.

Methods: Eighty healthy participants underwent diurnal IOP assessment sessions from 8:00 am to 9:00 pm in 2 visits 1 week apart. Intraocular pressure was assessed by Goldmann applanation tonometry.

Results: The repeatability of diurnal IOP patterns in healthy individuals was excellent. The coefficient of variation was less than 10% in all cases.

Conclusion: Diurnal IOP patterns in healthy individuals are highly repeatable over a 24-hour period.

WHEN DO YOU SUSPECT SOMETHING OTHER THAN GLAUCOMA AND NEUROIMAGE?
IS IT ONLY GLAUCOMA?

- 53 YOBF- No complaints
- BVA 20/20 OD, OS
- Perrl (+) RAPD OS
- IOP 30 mm Hg OD and 32 mm Hg OS
- Unilateral sectorial disc pallor with minimal rim damage
- Color vision testing normal
- SLE normal OU
- Anterior chamber angles open gonioscopically.

Is this glaucoma or something else like a tumor?

Unilateral disc pallor? Glaucoma, something else, or both?
Neuroimage or not?

“THE CUPPED DISC: WHO NEEDS NEUROIMAGING?”

- Patients with glaucoma were:
  - Older
  - Better visual acuity
  - Greater vertical loss of neuroretinal rim
  - More frequent disc hemorrhages
  - Less neuroretinal rim pallor
  - Field defects along the horizontal

“THE CUPPED DISC: WHO NEEDS NEUROIMAGING?”

- Patients with mass lesions:
  - Visual acuity less than 20/40
  - Vertically aligned visual fields defects
  - Optic disc pallor in excess of cupping
  - Age younger than 50 years

MORE INDICATIVE OF A COMpressive MASS LESION THAN GLAUCOMA

- Younger age
- Lower levels of visual acuity
- Vertically aligned visual field defects
- Neuroretinal rim pallor


BACK TO THE PATIENT...

- Rim minimally notched 😐
- Disc pallor 😐
- Unilateral damage 😐
- No disc hemorrhage/ parapapillary atrophy 😐
- Age over 50 😐
- Arcuate defect- glaucomatous 😐
- Risk factor- IOP 30s 😐
- Acuity and color normal 😐

CASE: 56 YOBF

- Dx POAG OU 5 years ago
- Slowly progressive vision loss
- LP OD; 20/30 OS
- Used combo med- ran out months ago
- IOP: 19 mm OD, 18 mm OS
- CCT: 560; 544
45 YOM - KERATOCONUS
- 20/30 OD; 20/25 OS
- 17 mm Hg OU
- PERRL (-) RAPD
- No pachymetry yet

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
65 YOF- POAG OU; 20/40 OU

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

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 a denting 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 lachenia in this age group.

The globes are symmetrical. There is no lens dislocation. The post-septal soft tissues are preserved with no definite intra- or extracranial 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.

NOW HOW WOULD YOU HANDLE THESE? DON’T WORRY...

IT’S NAHT A TOOMAH

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

JP: 38 YOF

- Referred for glaucoma eval in 2002 after failing LASIK screening
- Had been treated since mid 20s for glaucoma
- IOP in mid-upper teens off meds
- CCT: 459 OD; 469 OS
- Anomalous nerves with mild field loss
**JP: NOW 49 YOF**
- Congenitally anomalous nerves with field loss
- Monitored for 11+ years
- Field changes late
- Pt now treated with IOP 09 mm OD; 10 mm OS
- Pt had/had congenitaloma and now has glaucoma
  - Doubloma

**SIMILAR...YET DIFFERENT**
- 45 YOF
- Referred for glaucoma evaluation
- IOP never exceeds mid-teens
- CCT: 554 OU
- Marginal effect of meds

**CONUNDRUMS**
- Field loss due to anomaly, glaucoma, or both?
- Progressive or congenital?
- Mid-teen IOP and poor medical response
- Treatment or observation?

**WHAT CAUSES A DISC HEMORRHAGE AND IS IT PROGRESSION OR A RISK OF PROGRESSION?**

**WHAT DO YOU DO WHEN YOU SEE A DISC HEMORRHAGE?**
Not all hemorrhages of the disc are disc hemorrhages. Make sure that the glaucomatous characteristics are there.

**RISK FACTORS: DISC HEMORRHAGES**
- Inferior, inferior temporal, superior, and superior temporal regions of the disc are most susceptible and account for virtually all true glaucomatous disc hemorrhages.
- Typically occurs where notches and RNFL defects occur.
- Hemorrhages at other areas of the disc (nasal and temporal) tend to not be associated with glaucoma.

**OTHER CAUSES OF ‘DISC’ HEMORRHAGES**
- PVD
- HTN
- Anemia
- Diabetes
- Vascular occlusion
- Subarachnoid bleed
  - Terson’s syndrome
    - Subretinal and intraretinal
    - May be juxtapapillary

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

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

---

**55 YOM**

- 2012 presents without complaints
- BCVA 6/6 OD, OS
- IOP:
  - OD: 27 mm; 30 mm
  - OS: 15 mm; 15 mm
- CCT: 536; 531

---

**55 YOM**

- Treatment initiated
  - IOP drops to mid teens OU
- Optic disc change OS noted 4/14
- Therapy amplified
- 7/15: latanoprost and dorzolamide/timolol FC OU
- IOP: 10 mm OU
- CCT: 536; 531
SO WHAT DO I DO WHEN I SEE A DISC HEMORRHAGE?

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

HOW DO YOU MANAGE GLAUCOMA SUSPECTS?

WHO IS A GLAUCOMA SUSPECT?

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

DISC EVALUATION

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

RULE: WHEN DIAGNOSING GLAUCOMA, TAKE IOP OUT OF THE EQUATION

(When managing glaucoma, put IOP back into the equation...but that's another lecture.)
WHO ARE THE GLAUCOMA SUSPECTS?

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

IS THIS GLAUCOMA?

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

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

IS THIS GLAUCOMA?

78 YOWM
Annual exams with multiple doctors
IOP ranges from 17 – 21 mm Hg
CCT 570
Ocular health always “normal”
Small discs with indistinguishable cupping
- 0.2/0.2 – 0.3/0.3
WHO ARE THE GLAUCOMA SUSPECTS AND WHAT DO I DO?

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

- Large cupping - high IOP
  - Does the nerve look glaucomatous?
    - Yes - photos, fields, pachymetry, gonio, OCT
    - No - OCT, photos, pachymetry, fields, gonio

- Normal cupping - high IOP
  - OCT, photos, pachymetry, fields, gonio

DON'T OVER-TEST

“When you get the answer you want, hang up”

BUT DON'T UNDER-TEST, EITHER
LARGE CUPPING- UNKNOWN IOP- DIAGNOSED WITH GLAUCOMA

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

LARGE CUPPING- HIGH IOP

56 YOF
IOP: 24 mm OH
CCT: 550 OD, 539 OS
RTC 6 mos fields
Follow w/o treatment Q 6 mos

NORMAL CUPPING- HIGH IOP

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

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