

Glaucoma Unplugged...and Seated

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Incorporating New Technologies such as OCT in glaucoma practice

- Imaging devices are not Silicon Valley Rumpelstilskins. You cannot put in straw and expect to get out gold. Garbage in – garbage out. No amount of technology will replace your clinical acumen.
- Interpretation of any diagnostic laser is a three part process. 1. Understand that the printout identifies how the patient's measured data differs from the normative data base and to what statistical degree, 2. Use personal clinical experience to determine if the results are consistent with normal or abnormal anatomy and, 3. Incorporate everything into the entire clinical picture.
- GDx solely measures RNFL thickness. HRT solely measures optic disc topography. OCT has analysis of RNFL thickness, optic disc topography, and ganglion cell complex as well as retinal and macular thickness analyses. OCT is multifaceted while the other technologies have much more limited functions.
- Be aware of issues of Red Disease and Green Disease. Red Disease occurs when a normal patient's measurements fall outside normal limits (i.e. too much red on the printout), leading you to think that there is disease present when it is not. Conversely, Green Disease occurs when a patient with true disease has measurements fall within the normative data base (i.e. everything is green), leading you to think that the patient is normal but the clinical findings indicate true disease.

Corneal Hysteresis (CH)

- Characterizes the response to an application and removal of force
- Difference in the inward and outward pressure values
 - A result of viscous dampening in the cornea
 - Hysteresis is an inherent biomechanical property of the cornea which measures the cornea's ability to dampen a force when applied. Although central corneal thickness (CCT) plays a role in this ability, CH may be a better indicator of how the cornea, as well as other ocular tissue including the lamina cribrosa, respond to short and long term pressure fluctuations. CH is a measure of tissue function, rather than just a structural parameter like CCT
- Corneal Hysteresis and Glaucoma
 - Glaucoma subjects have lower corneal hysteresis than normal
 - CH has been identified as a risk factor in glaucoma progression independent of CCT.
 - Eyes with a higher CH, or a greater ability to dampen intraocular pressure fluctuations may be less susceptible to the development of glaucoma and eyes with low CH may increase the risk of glaucomatous optic neuropathy.

- Eyes with low corneal hysteresis at initiation of treatment with topical prostaglandin analogs showed a greater reduction in IOP than those patients with higher baseline CH.
- CH may increase following the initiation of topical therapy.
- Ocular Response Analyzer (ORA)
 - Non-contact ‘tonometer’- only FDA approved device
 - Corneal Hysteresis measurement
 - An air impulse applies a force to the cornea in a similar fashion to a non-contact tonometer. Corneal applanation is measured at two moments in time: at an ‘inward’ bending (P1) and an ‘outward’ bending point (P2) over a total period of 20 milliseconds. The difference between the two endpoints, the inward and outward applanation pressure, is the corneal hysteresis, measured in mmHg.
 - A result of viscous dampening of the cornea
 - Inward applanation pressure (P1) minus the outward applanation pressure (P2)

Target Pressures

- Based upon diagnostic evaluation, weighing risk factors, and considering risk-to-benefit ratio of treatment, the decision to initiate medical therapy is made. Once the decision to treat is made, a goal of therapy is set. In some cases, a target pressure is chosen. This pressure (or range) is the one that is felt to be a safe level for a given patient.
- This must be periodically re-evaluated with considerations as to severity of disease, risk of visual impairment and disability, general health of the patient, and life expectancy.
- The target pressure may be adjusted either up or down depending upon factors considered.
- There is no guarantee that IOP less than 21 will preserve a patient’s vision.
- The greater the degree of damage, the lower the IOP needs to be due to fragility of the already damaged nerve.

Monocular Trials

- Monocular trials do not give valuable information. That is, historically medication has been 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. However, this concept was based upon a number of assumptions which have been subsequently proven to be incorrect:
 - 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
 - These assumptions have all been proven to be erroneous
- 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.

Gonioscopy Facts

- Must be done on every glaucoma patient and suspect (at least at some point in the evaluation). You cannot accurately diagnose any glaucoma until you know the anatomic status of the angle.
- Repeat as indicated. Do not assume that the configuration of the angle remains the same. As the patient ages, cataracts develop, the lens thickens, the apposition between the anterior lens capsule and posterior iris the pupil becomes firmer (pupil block), aqueous egress from the posterior to the anterior chamber becomes blocked, and the angle becomes shallower (or closes). There are patients that slowly convert from open angle glaucoma to chronic (or even acute) angle closure.
- Intervals to repeat are debatable- every 1-3 years
- 2 years seems reasonable.
- Any patient that has had any form of angle closure or been prophylactically treated to prevent angle closure should have gonio done every 6-12 months.

When do you recommend LPI?

- Primary angle closure suspect
 - Pigmented trabecular meshwork blocked by iris
 - Extent of blockage not clear (typically 180⁰)
 - No peripheral anterior synechiae (PAS)
 - Disc and IOP normal
 - Probe for symptoms of intermittent closure
 - Not clear if laser peripheral iridotomy (LPI) or observation is better for these patients
 - Primary angle closure
 - Pigmented TM is blocked by iris for 180⁰
 - Have either PAS or elevated IOP as well
 - No disc damage or field loss
 - Considered pathologic
 - LPI recommended
- Primary angle closure glaucoma
 - Pigmented TM is blocked by iris for 180⁰
 - Have either PAS or elevated IOP
 - Glaucomatous neuropathy and field loss also present
 - 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, lensectomy, trabeculectomy (as dictated by severity)

Where does SLT Belong?

- Selective Laser Trabeculoplasty (SLT)
- 532 nm frequency doubled, Q-switched, Nd:YAG Laser
 - Laser seems to stimulate the cells in the trabecular meshwork that have not been cleaning out the debris and dividing into new cells.
 - New cells are much more vigorous about cleaning out the meshwork.
 - Wavelength that targets just the pigment in the trabecular meshwork. It provides the effect of the argon laser with less injury to the inside of the eye.
 - SLT & ALT are equivalent in their capacity to decrease the IOP in glaucoma patients
 - Produces different effects at the treatment site
 - ALT induces mechanical alterations, due to collateral thermal effects
 - SLT induces no apparent tissue alterations, due to the lack of such thermal effects
 - It is believed that SLT can be performed more than once, but this is unproven. Most will only repeat one time.
 - Lack of coagulative necrosis produced by SLT is related to the nanosecond duration of each pulse and the selective targeting of melanin chromophores
 - Application of SLT obliterated macrophages, leaving the non-pigmented lining TM cells intact
 - SLT Efficacy
 - 360° SLT treatment is equal latanoprost QHS in IOP lowering
 - 90° or 180° SLT is not as good in lowering IOP as latanoprost QHS

Disc hemorrhages

- Patients with normal tension glaucoma, primary open angle glaucoma, ocular hypertension
 - Anemia, posterior vitreous detachment, vascular occlusion can cause hemorrhages of the disc that are mistaken for glaucomatous disc hemorrhages
- Ischemic or mechanical
- Probable infarction of the blood supply to the ONH
- Inferior, inferior temporal, superior, superior temporal regions of the disc most susceptible and account for virtually all true disc hemorrhages
 - Hemorrhages at other areas of the disc (nasal and temporal) tend to not be associated with glaucoma
- Typically occurs where notches occur
- Resides in the retinal nerve fiber layer
 - Not in the cup!
- Small and contiguous with the neuroretinal rim
- Can be recurrent and, if it recurs, it typically is in the same place on the disc each time
- Precedes notching, NFL defect, field loss. Perhaps the earliest change in glaucoma (if it happens)
- More common in patients with large IOP variations

- Meaning is unclear – possibly indicates poor control of IOP?
 - EMGT study showed that lowering IOP cannot prevent disc hemorrhages from occurring
- Disc hemorrhages do not constitute a diagnosis of glaucoma nor a progression or conversion to glaucoma or an endpoint for any major glaucoma studies

IOP and CCT: Correct or not?

I. Raised intraocular pressure (IOP) is the only causal risk factor for glaucoma that can be therapeutically manipulated to change the course of the disease process.

- A. Goldman applanation tonometry (GAT) is the “gold standard” for IOP measurement; readings of IOP with GAT are affected by central corneal thickness (CCT).
- B. Formulae to correct IOP based on CCT are flawed
- C. IOP is a measurement that has too much variability for and many of those variables we don't understand well

II. Risk Factors for Developing Glaucoma?

- A. Strong association
 1. Age
 2. IOP
 3. Central corneal thickness
 4. CDR
 5. Race
 6. Moderate association
 7. Family history
- B. Weak association
 1. Refractive error
 2. Systemic diseases-mixed

III. Central Corneal Thickness

- A. 500 microns CCT and 21 mm Hg with Goldmann
 1. Discussion on the true IOP
 2. Avoid adjusting the IOP as we don't know the patients corneal rigidity
- B. 600 microns CCT and 21 mm Hg with Goldmann
 1. Discussion on the true IOP
 2. Avoid adjusting the IOP as we don't know the patients corneal rigidity
- C. Discussion on factors that influence applanation tonometry
 1. Corneal curvature
 2. Corneal thickness
 3. Corneal rigidity