What’s New in Glaucoma

George W. Comer, OD, MBA
SCCO at M.B. Ketchum University

Disclosures
George Comer, OD, MBA
SCCO at M.B. Ketchum University

Research funding from Topcon
Research funding from Optovue
Research funding from Heidelberg Instruments

TOPICS

New glau meds & classes of meds
Tonometry/IOP measurement/home tonometry
VF evaluation and progression analysis
ONH/RNFL assessment – assessment for glaucomatous structural damage and progression

What our current glaucoma meds do to lower IOP?

Cholinergics (pilocarpine)
Causes ciliary spasm – increase aqueous outflow through TM
Sympathomimetics (bromidine and apraclonidine)
Stimulate alpha 2 sites in ciliary body – decrease aqueous formation; also increases uveoscleral outflow
Sympatholytics (beta blockers – timolol, levobunolol, etc.)
Block beta 2 sites in ciliary body – decrease aqueous formation
CAIs (dorzolamide, brinzolamide)
Block carbonic anhydrase in ciliary body – decrease aqueous formation via active secretion
Prostaglandin analogs (latanoprost, travaprost, bimatoprost)
Loosen the extracellular matrix in ciliary muscle – increases uveoscleral outflow rate

Glaucoma: Too much aqueous produced vs too slow aqueous outflow??

Most high IOP and open angle glaucoma, particularly primary open angle glaucoma, is caused by resistance to aqueous outflow through the trabecular meshwork.

AQUEOUS OUTFLOW PATHWAYS

Trabecular outflow (70 - 90%)
Uveoscleral outflow (10 - 30%)

Yet currently our most effective IOP-lowering meds are PGs??!
Where is the resistance to aqueous outflow in the TM?

- Juxtacanalicular tissue
- Wall of Schlemm’s canal

We need meds that work on the TM endothelium, the TM cytoskeleton and extracellular matrix and wall of Schlemm’s Canal.

New Meds are Coming with the Following Potential Advantages

- New target tissues and mechanisms of action
- Some have multiple mechanisms of action
- May achieve equal to or greater IOP lowering than the PGs

May be able now to switch from an intolerable or ineffective PG to another single med with similar IOP-lowering – this should yield better adherence than adding a med to the PG.

What new classes of meds for glaucoma are in the FDA pipeline?

- Rock inhibitors
- Rock inhibitor + prostaglandin fixed combo – Rocklatan™
- Nitric oxide donors
- Selective adenosine receptor agonists

Rho-kinase (ROCK) inhibitors

Four are in development at this time

Act to inhibit Rho-kinases (RhoA, RhoB, RhoC) which regulate cell shape, motility, proliferation and cellular apoptosis.

Relax the TM tissues stimulating increased outflow through TM pathway

Farthest along in the FDA process is Rhopressa (AR 13324) by Aerie Pharmaceuticals

Rhopressa™

Multiple mechanisms of action

Three mechanisms of action of Rhopressa:

- Enhancement of TM outflow by relaxing the TM tissues
- Reduced aqueous production by norepinephrine transport (NET) inhibition
- Decreased episcleral venous plexus pressure

http://www.eyeworld.org/images/New_Articles/2015/11/35_b.jpg
Rhopressa™
Other potential actions

Animal studies suggest other potential positive effects of ROCK in the eye:
- Enhanced corneal endothelium function
- Enhanced blood flow to the optic nerve
- Improved RGC survival
- Reduced bleb scarring after trabeculectomy

Rhopressa™ Early FDA Phase 3 Trials – IOP Results

Rocket 1 trial
Rhopressa did NOT meet the primary endpoint of non-inferiority to timolol bid in patients with a baseline of 20 to 27 mmHg.

Rocket 2 trial
Rhopressa qd or bid DID meet the primary endpoint of non-inferiority to timolol bid in patients with a baseline of 20 to 25 mmHg.
Secondary endpoint (non-inferiority to timolol bid for baseline IOP 20 to 27) was NOT achieved (at 2 of the 9 time points)

Rhopressa™ Early FDA Phase 3 Trials – Adverse Events

Most common adverse event was conj hyperemia; 35% with qd dosing, 50% with bid dosing.
About 80% of the conj hyperemic was classified as mild.

Roclutan™

Rhopressa + latanoprost
Four IOP-lowering mechanisms: increased uveoscleral outflow due to latanoprost in addition to the 3 mechanisms from Rhopressa
The combination of Rhopressa and travatan produced IOP-lowering that was clinically and statistically greater than travatan alone.

Roclutan™ FDA Phase 2b Trial

297 patients; mean IOP 25.1
Roclutan lowered IOP by a mean of 34% to mean of treated IOP of 16.5
Roclutan IOP lowering effect was a mean of almost 2 mmHg greater than that of latanoprost alone
Roclutan Phase III trial (Mercury 1) is in progress
**Latanoprostene bunod**

**Pharmaceuticals by Nicox**

**Mechanisms of action**

Increased uveoscleral outflow

Nitric oxide donation causes relaxation of the TM causing increased TM outflow – causes TM endothelial cell shape changes and relaxation of the cytoskeleton resulting in increased TM outflow

**FDA Phase 2 (Voyager) Study:**

Latanoprostene bunod 0.024% gave best IOP lowering among 4 concentrations with qd dosing in a 28 day trial

Lowered IOP to statistically greater amount than latanoprost 0.005% at all time points.

Side effects were comparable to those related to latanoprost 0.005%. Most common side effect was conjunctival hyperemia.


**FDA Phase 3 Trials (Apollo, Lunar)**

IOP Lowering

Latanoprostene bunod 0.024% qd gave greater IOP-lowering than 0.5% timolol at bid dosing.

IOP lowering of mean IOP was 7.5 to 9.1 mmHg.


**Adenosine agonists**

There are 4 adenosine receptors found in the TM.

Three have the ability to lower IOP: A1, A2A and A3 though A3 has the potential to raise IOP.

Inotek Inc. is in Phase 3 trials with an A1 agonist, trabodenoson.

Acucela and Otsuka Pharmaceuticals are working in a A2A receptor agonist and completed a Phase1/2 trial in 2012.

Santen Pharmaceuticals also has an A2A receptor agonist in development.

**Trabodenoson**

Selective adenosine type 1 receptor agonist.

Increases metabolic activity within the TM which upregulates proteases specifically MMP-2 within the TM. These proteases remove accumulated material that slows TM aqueous outflow.

**Trabodenoson Phase 1/2 Trial: IOP Lowering and Adverse Events**

- 144 patients treated for 28 days
- Compared to placebo
- Lowered IOP by average of about 7 mmHg

Adverse events:
- No systemic side effects
- Less rate of conj hyperemia than the current PGs

**Trabodenoson Phase 3 Trial**

- Phase 3 trial currently ongoing
- Results expected in late 2016.

**Other Glaucoma Medical Treatments in Development**

- Fixed combination of PG + beta blocker Taptiqom (tafluprost 0.0015% + timolol 0.5%)
  Santen Pharmaceuticals
  A 6-month prospective randomized, double-masked, parallel group Phase III study showed significant IOP reduction with Taptiqom beyond the IOP reduction of the individual components.

- Biromatrost SR implant (Allergan)
  Biodegradable, preservative-free implant which after placing in the AC lasts several months
  In Phase 3 trials now.

- Sustained release Travatan punctal plug (Ocular Therapeutics)
  Intracanicular depot of polyethylene glycol hydrogel + travaprost containing microparticles
  Phase 2 trials showed significant IOP reduction for up to 3 months
  Advantage of these: not dependent on patient adherence

**Tonometry/IOP Assessment**

**Newer (Better??) Tonometers**

- iCare tonometer
- Pascal Dynamic Contour Tonometer (DCT)
- Reichert Ocular Response Analyser (ORA)/Reichert 7CR/Reichert G3
- Oculus Corvis ST
What about GAT?

“Gold standard” has been GAT
Most commonly used in clinical practice – “standard of care”
Relatively inexpensive compared to NCTs, Tonopen, Pascal DCT etc.
Relatively mechanically reliable
   Calibration should be checked q3 mos

Is GAT the standard of care?
Is GAT the gold standard (of accuracy)?

Limitations of GAT

Not objective – judging the alignment of fluorescein semicircles is subjective
Many cases where GAT accuracy is compromised: thick CCT, thin CCT, scarred corneas, edematous corneas etc.
Should check calibration q3mos
Not self recording; no hard copy printout
Relative to NCTs: must instill anesthetic and touch cornea

CLINICAL SIGNIFICANCE

CORNEAL THICKNESS & IOP

GAT is most accurate for CCT of 540µ
Thinner (than 540) cornea → possible false low IOP on GAT
Thicker (than 540) cornea → possible false high IOP on GAT

CORNEAL THICKNESS FINDINGS

Clinical Issues

Ocular hypertensives - may have false high GAT readings due to thicker cornea
LTG suspect - may have false low GAT readings due to thinner cornea
Post LASIK → false low IOP readings on GAT

Ocular Hypertension Treatment Study (OHTS)

All ocular hypertensives - average corneal center thickness (CCT): 573 microns → thicker than normal patients
White ocular hypertensives - average corneal center thickness (CCT): 579 microns
African-American ocular hypertensives average corneal center thickness (CCT): 555 microns

SHOULD I ADJUST IOP FOR CCT TO IMPROVE THE ACCURACY OF GAT?

**No!** No adjustment was found to be appropriate in OHTS using several different algorithms. CCT is one of several corneal biomechanical factors affecting IOP readings, so adjusting for one single factor is really not helpful.

---

**iCare Tonometer**  
Rebound tonometry

- Handheld, portable tonometer
- Anesthetic not needed
- Momentary touch with small probe
- **Advantages:**
  - Useful for screening IOP
  - Useful in children
- **iCare Home tonometer**
- **Disadvantages:**
  - Tends to read false high
  - Degree of false high increases with CCT

---

**I-Care Tonometer**  
(Rebound tonometry)

**iCare Home Tonometer**

- Self administered tonometer for home use by patients
- Some form of home tonometry is badly needed in glaucoma diagnosis and management.

---

**iCare Tonometer Summary**

- Portable, easy to use
- No anesthetic needed
- Very good for screening situations and kids

However, influenced by corneal factors such as CCT (higher CCT gives higher readings). Also better to take multiple readings than just single reading.
Contact Lens IOP Monitor
Sensimed Triggerfish - FDA Approved 3/2016

The FDA Approves Marketing of the Triggerfish "Smart" Contact Lens Sensor to Monitor Glaucoma Eye Pressure

Pascal Dynamic Contour Tonometer

Not an applanation tonometer – concave surface
Surface (flexible membrane) conforms to the curvature of the cornea → effect of corneal rigidity and thickness minimized
IOP microsensor built into the tip
Obtains 100 IOP readings/sec. → averages the IOP changes over the ocular pulse
Gives ocular pulse amp as well as IOP
Generally reads ~1-2 mmHg higher than GAT

Pascal DCT

Does not applanate but contours to the cornea
Minimizes the effect of corneal biomechanical properties like CCT
Almost no effect of Lasik, keratoconus, edema, scarring etc.
Close correlation to manometry

GAT vs Pascal DCT

GAT - visual monitoring of IOP, manual measurement
Pascal DCT – automated IOP measurement, 100/second

IOP Printout from Pascal DCT
Continuous IOP measurement across 6 cardiac cycles
Note the change of about 3 mmHg across each cardiac cycle
The average across the 6 cycles is 21.1 mmHg. OPA = 3.1 and Quality = 1 (excellent)
Summary – Pascal DCT

Not significantly influenced by corneal biomechanical properties – more accurate than GAT in cases where cornea is thick, thin, edematous, scarred etc.
However, not real good mechanical reliability; those fine, Swiss-made plastic parts break!

OCULAR RESPONSE ANALYZER (Reichert Instruments)

NCT that is designed to improve the accuracy of the IOP measurement by accounting for some corneal biomechanical factors
Monitors response of cornea to airpuff
Adjusts out the error caused by CCT, corneal rigidity, keratoconus, changes in biomechanical properties caused by laser refractive procedures etc.
Measures corneal hysteresis, CPT 92145

OCULAR RESPONSE ANALYZER (Reichert Instruments)

Measures corneal hysteresis (CH) and corneal resistance factor (CRF)
Gives Goldmann-correlated IOP (IOPg) = closely correlated to GAT
Gives corneal compensated IOP (IOPcc) = IOP not influenced by CCT. Similar to IOPs determined by the Pascal DCT

CORNEAL HYSTERESIS

What is it?
An index of the visco-elastic properties (viscous dampening) of the cornea
Not a measure of stiffness/rigidity of the cornea but rather how the cornea absorbs and dissipates energy during deformation and return
On air puff the 1st applanation point of the cornea occurs at a higher IOP than the 2nd - the difference is CH and is the result of viscous dampening effect of the cornea

CORNEAL HYSTERESIS

Why might it be important?
Increased dampening capability (high CH) of the cornea may allow it to effectively buffer IOP variations & protect the ONH from them
Corneal hysteresis may be a biomarker for biomechanical properties of the scleral shell including the lamina cribosa
**What is Measured by the Reichert Ocular Response Analyzer (ORA)?**

Hamilton K. Clinical Decision-making V: IOP & Tonometry, Optometry Today 11/18/2008

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>IOP&lt;sub&gt;c&lt;/sub&gt;</td>
<td>Gelatinous correlated IOP, this measurement is directly comparable with any conventional contact or non-contact tonometer</td>
</tr>
<tr>
<td>IOP&lt;sub&gt;p&lt;/sub&gt;</td>
<td>Cornea compensated IOP, an IOP measurement that is less influenced by corneal properties such as elasticity and thickness</td>
</tr>
<tr>
<td>CH</td>
<td>Corneal hysteresis, this is calculated as the difference between application pressures 1 and 2, as shown in Figure 4. It is an indicator of a particular aspect of corneal behavior called viscous damping, and this number is used in further analysis</td>
</tr>
<tr>
<td>GTF</td>
<td>Corneal resistance factor, a measurement of the total viscoelastic response of the cornea, which includes elasticity and thickness</td>
</tr>
</tbody>
</table>

**CORNEAL HYSTERESIS**

**What is the significance?**

Low corneal hysteresis (CH) = low viscous dampening capability of the cornea

- Low CH suggests increased risk of corneal disorders (k-conus, post-Lasik ectasia etc) & glaucoma
- Low CH has been shown to be an independent risk factor for glaucoma progression (Congdon NG Am J Ophthal, 2006)
- CH is also low in low tension glaucoma
- May reflect properties of the lamina cribosa
- CH has been found to correlate to lamina cribosa deformation & was the only factor to correlate (Wells IOVS 2008)

**Low CH was shown to be an independent risk factor for more rapid glaucoma progression** (Congdon NG Am J Ophthal, 2006)

- In POAG patients with asymmetric VF damage the eye with the lower CH had the greater VF loss (Anad A, DeMoraes C. Invest Ophthalmol 2010)
- CH was associated with more rapid progression of glau VF loss; CCT was not associated (DeMoraes CG et al. Journal of Glaucoma, 2011)

**CH has been shown to:**

- Be lower in glau eyes with acquired pits of the ONH (APON) than in glau eyes without APON
- Be associated with more rapid progression of glau VF loss; CCT was not associated
  - DeMoraes CG et al. Journal of Glaucoma, 2011

**CH was associated with more rapid progression of glau VF loss:**

- Be modestly correlated to CCT
- GAT readings are significantly influenced by CCT: CH was more (3x) strongly associated with the rate of progression – DIGS, prospective study

**REICHERT ORA SUMMARY**

ORA is an NCT – no anesthetic, no corneal touch

- Gives IOPs largely free of the influence of corneal biomechanical properties, IOP<sub>c</sub>
- Measures corneal hysteresis (CH) which has clinical significance – low CH → faster glau progression rate

**CAN I IMPROVE THE ACCURACY OF GAT BY ADJUSTING GAT IOP FOR CCT?**

No!! No adjustment was found to be appropriate in OHTS

- CCT is one of several corneal biomechanical factors affecting IOP readings so adjusting for one single factor is really not helpful; it may “adjust” IOPs the wrong way!!

- Use Pascal DCT or Reichert ORA (or 7CR) instead of trying to adjust IOP for CCT
When might it be important to get DCT or ORA IOPs?

- High or low CCT
- Scarred cornea
- Corneal edema
- Keratoconus
- Corneal refractive surgeries
- Progressive glau despite having reached the target IOP

VF Testing in Glaucoma - Questions

- What's new?
- Is there a perimeter that is much faster than current perimeters?
- How about less boring?!
- Is perimetry necessary? It is so time-consuming with lots of artifact.
- Can OCT be substituted for perimetry?
- How critical is perimetry in monitoring glaucoma?
- How to detect progression of glau VF loss?

Common Forms of Perimetry

Today

- Standard automated perimetry (SAP)
  - White stimulus on white background
  - Stimulates all retinal ganglion cells
- Alternative perimetry
  - Stimulus designed to stimulate a subset of all retinal ganglion cells
  - SWAP (blue on yellow) perimetry
  - Frequency doubling: FDT, Matrix
  - Heidelberg Edge Perimetry (HEP)
  - Quasar perimetry on the Octopus 600

Advantages of Alternative Perimetry

- More rapid screening programs
  - N30 on FDT/Matrix is about 45 seconds if normal – very rapid
- Better ability than SAP to detect early glaucoma due to the stimulus used
- Often smaller, more compact perimeter
STAGES OF GLAUCOMA
In ICD9 and ICD10 by American Glaucoma Society

Mild
- Optic Nerve abnormalities consistent with glaucoma
- But NO visual field abnormalities on standard automated perimetry (white on white perimetry)
- Abnormalities may be present on alternative perimetry such as FDT, SWAP etc.

Moderate
- Optic nerve abnormalities consistent with glaucoma
- AND glaucomatous visual field abnormalities in ONE hemifield and
- NOT within 5 degrees of fixation (note: 5 degrees is involvement of test points nearest fixation)

Severe, advanced, late stage
- Optic nerve abnormalities consistent with glaucoma
- AND glaucomatous visual field abnormalities in BOTH hemifields
- AND/OR loss within 5 degrees of fixation in at least one hemifield.

What can alternative perimetry be used for?

- All levels of perimetry including threshold perimetry (92083)
- Exception is SWAP – no screening
- FDT/Matrix is very good for screening
- Screening is a very efficient way to check the VF and may be the only reliable, artifact-free way in some patients

AC 9/30/05 Glaucoma F/U

49 y/o Persian female
- Get 2nd opinion on her COAG diagnosis and treatment.
- Discontinued Txe 12 mos ago without consulting her OMD.
- Seen at ECC 3x over last 10 mos as glau suspect. Tried Alphagan P – pt. self discontinued it though no side effects.
- PEHx: Initiated Timoptic XE 1 gt OU HS in 1/1999. Compliance has not been good.
- PMHx: Good health

SLE: Normal, deep AC, no sign of 2° glau
- GAT: 20/21 10:30 AM
- No glau meds within past 3 mos
- Gonio: Angles wide, no 2° glau
- Pachymetry: OD: 570 µ  OS: 575 µ (ave of 8)
- ONH/RNFL eval: C/D .25-.3 rd OU, small ONHs
- RNFL: normal OD, OS

GDx VCC: Normal OU
- HRT: Normal stereometric parameters and normal Moorfields Regression Analysis
- HFA 24-2 SS: See VFs
Second HRT – normal MRA but small ONH – common cause of false negative on MRA

Two nonadjacent depressed points at <2% in inf nasal step region. Difficult to call it a VF defect!

Definite inf nasal step.

Progression superior temporal.

Superior temporal RNFL OD is normal on Stratus.

Reliability not good high FPs which may conceal a VF defect.
AC – What does this case illustrate?

Alternative perimetry (FDT), even the screening tests, can detect VF loss before SAP (but not always)

VFs can show damage before structural tests e.g. clinical exam & imaging of ONH &/or RNFL

Do not replace your VFs with imaging; use both

HRT may pick up structural damage not evident on OCT and vice versa

Can alternative perimetry such as FDT/Matrix be used for glaucoma management/progression analysis?

Very good for detection of early glaucoma

Very good for following into moderate glaucoma

However, may show VF loss that is much worse (even absolute VF loss) when there is little or no VF loss on SAP

Glau VF loss progresses primarily by increasing depth (dB values within VF defect decline) but cannot do progression analysis if the dB values are already 0 dB or close to 0dB

There is increased variability/fluctuation

Mary Mad or P Ford

HFA finds scotoma – confirmed by Matrix N-30 screening

MM
HFA 24-2
1/29/2016

Very deep (0 dB) one point inf nasal step not seen on previous VFs

GPA
4/2012 to 1/29/2016

Very deep (0 db) one point inf nasal step not seen on the 3 previous VFs
What about alternative perimetry for glaucoma management?

Practical recommendations for measuring rates of visual field change in glaucoma


• Use SAP (white-on-white perimetry)
• May use FDT/Matrix or SWAP in addition to SAP to confirm but not in place of SAP
• Use imaging to complement SAP but not in place of SAP
• Use same strategy (SITA Std on HFA and TOP on Octopus etc) and test point pattern at all exams; 24-2 is most common on HFA
• Must obtain good quality VFs - minimize false responses & fixation losses

CLINICAL STRATEGIES FOR DETERMINING RATE OF VF PROGRESSION


• Use SAP (white-on-white perimetry)
• May use FDT/Matrix or SWAP in addition to SAP to confirm but not in place of SAP
• Use imaging to complement SAP but not in place of SAP
• Use same strategy (SITA Std on HFA and TOP on Octopus etc) and test point pattern at all exams; 24-2 is most common on HFA
• Must obtain good quality VFs - minimize false responses & fixation losses
CLINICAL STRATEGIES FOR DETERMINING RATE OF VF PROGRESSION

- Do not blindly trust reliability indicators - especially false negatives & fixation losses
- Watch for prolonged (beyond 2nd threshold test) learning effect
- IMPORTANT: Perform VFs at an adequate frequency to detect VF loss

KEY FACTORS IMPACTING THE DETECTION OF VF PROGRESSION

Frequency of VF testing
Best to test at least 2 VFs/year
Better is 3/year in first 2 years OR if progression is suspected

Variability of results
Minimize sources of variability - consistent patient ed, pupil size, trial lens used etc. – need best quality, least variable VFs in order to best detect progression

FACTORS IMPACTING THE DETECTION OF VF PROGRESSION
Examples
With moderate variability and 3 exams/year a 3 dB (1 dB/year) decline in MD can be detected in 3 years.
With low variability and 3 exams/year a 2 dB (1 dB/year) decline in MD can be detected in 2 years
With moderate variability and 1 exam/year a 6 dB (1 dB/year) decline in MD would take 6 years to detect!

JP
66 y/o wm with h/o RD OD with unsuccessful repair x 3
Also k-conus OU
All past IOPs have been <10 mmHg OS. Vertical cup on clinical exam. Normal OCT. Recent VF screening showed inf paracentral scotoma OS
Today CCT is 420µ OS; all tonometers read <10 except Pascal DCT
Later (after Trav Z was intitiated) ORA showed: 14 mmHg (DCT: 12, GAT 11, 12) CH: 3.9 very low

How is this “adjustment” possible?

Very low CCT
STRATEGIES FOR DETECTING VF PROGRESSION

**Frequency of VF Exams**

Perform 5 or 6 VF exams in the first two years in order to:

- Identify rapid progressors ASAP (≥1 dB/yr decline) (Heijl A, Bengtson B et al. EMGT Ophthalmology 1997)
- Establish a very solid baseline from which change can be more quickly recognized

Once the baseline has been established and the rate of progression has been determined to be slow then go to 1 or 2 VFs/year

---

**VF PROGRESSION SUMMARY POINTS**

- Can imaging be used instead of VFs for VF progression detection? No, must get both.
- What perimeter should I use? Easiest, most automated is an SAP (white-on-white) instrument: HFA, Octopus, Oculus etc.
- Can I use FDT/Matrix? Can use FDT but must use "eyeball or seat of your pants" techniques. Best to use specific criteria, not "seat of pants" judgment, for recognition of VF progression
- But doesn't FDT detect progression prior to SAP? In many cases in early glau but progression analysis software for alternative perimetry is not refined.

---

**VF PROGRESSION ANALYSIS THE NEXT BIG ADVANCE**

- Combined structure-function analysis
- Why? Better sensitivity and specificity in determining true progression

---

**HEIDELBERG STRUCTURE/FUNCTION MAP**

- OS has inf notch with inf RNFL defect. Sup arcuate VF defect corresponds exactly to it.
ONH/RNFL Analysis for Glaucomatous Damage and Progression

Ophthalmoscopy
Slit lamp and fundus lens
ONH photos
OCT and other imaging techniques

Advantages of Imaging in Glaucoma

Better detection of structural damage
Ability to target three areas of glaucoma damage:
ONH
RNFL
GCC – not possible to visualize in your clinical exam
Better ability to detect change
Better ability to determine rate of change

Why is the clinical ONH/RNFL exam still very important?

ONH damage is very specific to glaucoma & OCTs are not great at ONH analysis
Certain structural changes cannot be seen on OCT e.g. Drance heme, PPA etc
OCT does miss RNFL defects
OCT compares to a database of normal eyes. This generally works but variations of normal, congenital anomalies (ONH drusen, hypoplasia etc) and acquired disorders other than glaucoma (AION, OA etc) will fool the OCT

The Future of OCT

What is already here?
Higher scanning speeds (up to 70,000 A-scans/sec)
Better progression analysis software
GPA on Cirrus, RNFL/GCC Change on Optovue Avanti
Wide field scans (temporal to macula across the ONH) – peripheral retinal disease more accessible: schisis vs RD, peripheral CSR, nevus vs. melanoma etc. - iVue, Avanti
Structure-function correlation
Viewing software (Zeiss Forum) to allow visual correlation of VFs, imaging, photos etc.
Analysis software to correlate structure to function – Heidelberg
The ability to identify glaucoma is better when two or more parameters are analyzed and correlated e.g. VFs and OCT

NOTE: GCC loss at sup temporal horizontal raphe – corresponds to RNFL defect on clinical exam and photos
VFI trend is flat but the point-to-point comparison (event analysis) suggests likely progression at 2 points over 3 consecutive VFs in the inferior arcuate region of VF.

See the Trend Analysis for Ave RNFL and Inf RNFL Thickness. Inf RNFL Thickness is statistically thinner on last 3 scans than on the baseline. The more scans and the tighter (less variable) the data the better the ability to detect small changes in RNFL.

The Future of OCT
What is coming in posterior segment OCT?
- Better imaging of the choroid
  - EDI (Enhanced Depth Imaging)
  - Oversampling to minimize speckle
- Swept source OCT
  - Long wavelength (1050 µm) and very high speed scanning (600,000 A-scans/sec)
  - However, the trade-off for speed is resolution
  - Switchable OCTs – Nidek RS3000 Advance
- OCT angiography (Virtual angiography) – here now!! Both Cirrus and Optovue Avanti are FDA approved!
- Polarization sensitive OCT

Optovue Avanti
HD line scan through sup arcuate OS with 120 scans! Note the greatly enhanced detail in the choroid.