Clinical decision making following a diagnosis of POAG

COPE Course ID: 53663-GL

Derek MacDonald, OD, FAAO
Waterloo, ON, Canada

Please silence all mobile devices and remove items from chairs so others can sit. Unauthorized recording of this session is prohibited.
Disclosure Statement:

Speakers Bureau:  
Alcon  
Allergan  
Carl Zeiss Meditec
Glaucoma is a **progressive optic neuropathy** 
*(resulting in)*** **cupping**, a characteristic appearance of the **optic nerve** *(due to)* the **loss of retinal ganglion cell axons** ...
... supporting glia and vasculature ...
**with or without correlating visual field changes**¹⁻⁴

‘Although IOP is no longer part of the definition ... it is the only modifiable factor proven to decrease both the risk of disease onset and its progression.’⁵

**IOP is a risk factor** and **VF loss is a symptom**

---

- 55 y/o Caucasian female; good GH (no systemic medications)
- **ophthalmic history of OH (IOPs low 20s)**: healthy ONH; no VF Δ
- BCVA 6/6 (-4.00); normal BV, pupils, CVF, and anterior segment
- **IOPs 16/19**: CCT 541/539

superior-temporal wedge RNFL defect, O.S.

normal average RNFLT, but thinning at 1:00, O.S.

repeatable inferior nasal step, O.S.
So, you’ve diagnosed glaucoma ... now what?

1. Establishing solid baseline data
2. Staging disease severity
3. Establishing a target IOP
4. Initiating treatment
5. Detecting disease progression
6. Altering treatment
Establishing solid baseline data

Without knowing the starting point, detecting and quantifying progression is impossible. However, baseline data is often poorly acquired.

i. IOP

Regardless of whether you use peak or mean IOP, multiple measurements at different times of the day are necessary to establish a pre-treatment baseline\(^1\)

Glaucoma is a 24-hour disease that we try to monitor over (the wrong) 8 hours\(^2\)

---

Establishing solid baseline data

ii. Structure

Ensure that you have a **quality** (in-focus; high signal strength) baseline assessment of ONH/RNFL/RGC structure\(^1,2\)

---

Establishing solid baseline data

iii. Function

Although structural damage defines glaucoma, functional loss is what impacts the patient
• white-on-white standard automated perimetry (SAP) remains the gold standard for automated visual field (AVF) analysis

Establish a reliable and repeatable pre-treatment baseline:
• 2 AVFs within a time frame too short for progression
• fixation losses <20% and steady gaze tracking
• false negatives <25% and false positives <15%

Central 24- and 30-2 SITA strategies (stimulus size III) are commonly used in diagnosing and monitoring glaucoma

24-2 versus 10-2 visual field assessment

A 24-2 grid has 54 points (only 4 in the central 8°) separated by 6°
A 10-2 grid has 68 points (all in the central 10°) separated by 2°

A paracentral scotoma can ‘fall between the cracks’ of the 24-2

Glaucmatous damage of the macula

Most axons from the inferior-temporal macula project to the **macular vulnerability zone**, while those from the superior macula project to the less vulnerable temporal quadrant\(^1,2\)

Glucomatous damage of the macula

‘Macular damage, as seen on 10-2 VF s, appears to occur almost as frequently as peripheral defects in patients with ... early glaucoma.’

Which is better, one (OCT) or two (AVF)?

OCT may be more valuable in detecting the presence and progression of early disease (robust RNFL)$^1$
• functional (AVF) change is likely present but not detectable (PPG)

AVF may be more valuable in detecting the progression of advanced disease, and early disease threatening fixation$^2$
• OCT has a floor effect at ~50μm: little remaining viable RNFL (measuring residual or remodeled glial/vascular tissue)$^3$

Establish a reliable baseline for both structure (OCT) and function (24-2 and 10-2 AVF) as early as possible: leverage OCT in early disease and AVF in advanced disease$^4,5$

Staging disease severity

Disease severity may be staged into relatively broad categories using structural or functional criteria, or a combination\textsuperscript{1-3}

Staging enhances patient management by:

- encouraging careful assessment and documentation of clinical damage
- determining initial treatment (target IOP)
- facilitating monitoring for progression
- informing adjustment of treatment
- allowing shared management or transfer of care with a common and more objective understanding of disease severity

Staging disease severity

In this Canadian paradigm, staging is based upon both the amount of structural damage and functional loss\(^1,2\)

<table>
<thead>
<tr>
<th>Disc Features</th>
<th>Early OAG</th>
<th>Moderate OAG</th>
<th>Severe OAG</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Structure</strong></td>
<td>Glaucomatous ONH: vertical C/D 0.65 or less</td>
<td>Glaucomatous ONH: vertical C/D 0.7 to 0.85</td>
<td>Glaucomatous ONH: vertical C/D 0.9 or greater</td>
</tr>
<tr>
<td><strong>Function</strong></td>
<td>Mild VF defect: MD &lt;-6dB, not within 10 degrees of fixation</td>
<td>Moderate VF defect: MD -6 to -12dB, not within 10 degrees of fixation</td>
<td>Severe VF defect: MD &gt;-12dB, and/or within 10 degrees of fixation (utilize 10-2)</td>
</tr>
</tbody>
</table>

Our patient had a vertical C/D <0.65, a MD <-6.0dB, and fixation was not threatened: **staged as having early OAG**

---

Establishing a target IOP

A target IOP can be defined as:

The estimated upper limit of a dynamic IOP range that is expected to slow disease progression in an individual eye of an individual patient by reducing the rate of ganglion cell loss to be no greater than the age-related loss¹⁻³

A target IOP:

• is impossible to predict with absolute certainty
• must be aggressive, yet tolerable for the patient
• is a moving target⁴

References:

Establishing a target IOP

<table>
<thead>
<tr>
<th>Disease Severity</th>
<th>Target IOP</th>
<th>Minimum IOP Reduction</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated suspect (OH)</td>
<td>&lt;25</td>
<td>20%</td>
<td>OHTS</td>
</tr>
<tr>
<td>Early</td>
<td>&lt;21</td>
<td>25%</td>
<td>EMGT CIGTS</td>
</tr>
<tr>
<td>Moderate</td>
<td>&lt;18</td>
<td>30%</td>
<td>CNTGS AGIS</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt;15</td>
<td>&gt;30%</td>
<td>AGIS Shirakashi (^1) Odberg (^2)</td>
</tr>
</tbody>
</table>

Initiating treatment

Unless contraindicated, topical medications are the most common initial intervention\(^1,2\)

Prostaglandin analogs (PGA)
- \(\Delta\) IOP ~30% by enhancing uveo-scleral outflow
- the most favorable instillation schedule (once daily)
- effective 24-hour (including nocturnal) IOP reduction\(^3\)

**IOP lowering:**
\[\text{bimatoprost} \geq \text{travoprost} \geq \text{latanoprost}\] \(^4,5\)

---

Initiating treatment

Prostaglandin analogs (PGA)

- ocular side effects: conjunctival hyperemia (less with latanoprost); darkening of mixed-color irides; lash growth; exacerbation of HSK/uveitis/post-CE CME
  - prostaglandin-associated periorbitopathy (PAP)

Initiating treatment

Beta blockers (BB)
- Δ IOP ~25% by decreasing aqueous production
- QD dosing (morning) may be as efficacious as BID
  - relative cardiopulmonary contraindications ($\beta_2 > \beta_1$)$^1$
  - IOP lowering: $\text{levobunolol} \geq \text{timolol} > \text{betaxolol}$ $^2$

Alpha agonists (AA)
- Δ IOP ~20 to 25% by decreasing aqueous production
- TID dosing as stand-alone treatment
  - allergy (~20%); decreased BP; fatigue; dry mouth; HA$^3$

### Initiating treatment

#### Beta blockers (BB)
- Δ IOP ~25% by decreasing aqueous production
- QD dosing (morning) may be as efficacious as BID
  - relative cardiopulmonary contraindications ($\beta_2 > \beta_1$)
- IOP lowering: **levobunolol ≥ timolol > betaxolol**

#### Alpha agonists (AA)
- Δ IOP ~20 to 25% by decreasing aqueous production
- TID dosing as stand-alone treatment
  - allergy (~20%); decreased BP; fatigue; dry mouth; HA

---

**Neuroprotection?**
Although timolol lowers IOP more than betaxolol and brimonidine, the latter two may preserve VF more effectively$^{1,2}$

---

Initiating treatment

Topical carbonic anhydrase inhibitors (CAI)
- Δ IOP ~20% by decreasing aqueous production
- TID dosing as stand-alone treatment
  - burning on instillation; metallic taste (~7%)
  - systemic complications far more common with oral CAI
    - sulfonamide allergy; anemia; acidosis

CAI improve ONH blood flow
and reduce nocturnal IOP as effectively as PGA

Nocturnal IOP reduction: CAI = PGA > BB = AA

Initiating treatment

Our patient was staged as having early disease

Peak IOP in the left was 19mmHg

Target IOP was a 25% reduction (to less than 21mmHg):

14mmHg

Treatment was initiated with latanoprost HS

Post-treatment IOPs have remained 13 to 15mmHg over nearly 5 years of follow-up
Initiating treatment

What about the monocular trial?

• proposed to differentiate therapeutic from spontaneous change in IOP by comparing a treated to an untreated eye
  • (incorrectly) assumes a repeatable diurnal IOP variation that is consistently symmetric between eyes\textsuperscript{1-3}
  • the response of the first-treated eye may be a poor predictor of the response of the second-treated eye\textsuperscript{4}

\textbf{The monocular trial may not be as valuable as we thought} \textsuperscript{5}

‘Measuring IOP on multiple occasions before and after the decision to start treatment will always remain the gold standard for assessing ... IOP-lowering effect ...’ \textsuperscript{6}

Initiating treatment

What about laser trabeculoplasty?

Argon laser trabeculoplasty (ALT) is ‘... at least as efficacious as initial treatment with topical medication ...’ ¹

Selective laser trabeculoplasty (SLT) is ‘... a safe and effective initial therapy ... equally efficacious as latanoprost ... as primary or adjunctive therapy ... in open-angle glaucoma or ocular hypertension ...’ ²⁻⁵

• maintenance of target IOP can vary widely
  • from 30% to 90% efficacy at 3 to 5+ years⁶

---

Initiating treatment

What about laser trabeculoplasty?

SLT:
• is equivalent to treatment with a PGA (25-30% IOP reduction)\(^1\)
• reduces diurnal IOP fluctuation\(^2\)
• has an excellent safety profile
  • transient AC inflammation and IOP elevation
• is ‘non-preserved’
• guarantees adherence to treatment
  • can be repeated\(^3\)

SLT may be more effective as primary therapy, and less effective in patients with advanced disease on multiple medications\(^4\)

Initiating treatment

What about cataract surgery?

In open-angle glaucoma:
• **stand-alone CE modestly reduces IOP**
  • on average 13 to 17%\(^1,2\)
  • greater IOP reduction with higher pre-CE IOP
  • 34% drop from mid-20s\(^3\)

CE is invaluable in the management of angle-closure glaucoma\(^4\)

Initiating treatment

What about micro-invasive glaucoma surgery (MIGS)?

- *ab interno* implantation of a biocompatible drainage device, often (but not always) paired with CE, targeting Schlemm’s canal, the suprachoroidal space, or the subconjunctival space\(^1,2\)
- little trauma; rapid recovery; safe; effective; improved adherence
- currently positioned between medications/SLT and filtration surgery in the treatment of **mild to moderate** glaucoma\(^3\)

---

Detecting disease progression

Non-modifiable risk factors for progression

- older patient age at baseline
- family history (first-degree relative)
  - 3- to 9-fold increase
- ethnicity
  - black or Hispanic > Caucasian
- female gender (in NTG)
- concurrent cardiovascular disease
  - 2-fold increase, irrespective of IOP

Detecting disease progression

Non-modifiable risk factors for progression

- severe or rapidly progressing disease at baseline\(^1-3\)
  - 7- to 9-fold increase with significant RNFL loss
- exfoliation (in high-pressure OAG)\(^4\)
- disc hemorrhages/parapapillary atrophy (in NTG)\(^5\)

Detecting disease progression

Non-modifiable risk factors for progression

- corneal biomechanical properties\textsuperscript{1,2}
  - thin central corneal thickness (CCT)
  - low corneal hysteresis (CH < 9 mmHg)

Modifiable risk factors for progression (other than IOP itself)

- short- and long-term IOP fluctuations\textsuperscript{3-5}
  - particularly at low IOPs (relative > absolute change)?

Ocular perfusion pressure (OPP)?
Cerebrospinal fluid pressure (CSFP)?

---

Detecting structural progression

**Clinical examination: review of ONH/RNFL photos**
- **pros:** ‘backward compatible’; reveal DH and NRR pallor
- **cons:** qualitative; subjective; requires expertise\(^1\)

The most common clinical ONH changes are **rim thinning** and/or **inferior-temporal excavation**\(^2\)

---
Detecting structural progression

**Objective imaging:**

<table>
<thead>
<tr>
<th>In early (including pre-perimetric) to moderate glaucoma:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• ONH, RNFL, and macular analyses all detect structural change, often in advance and predictive of VF loss (3 to 8x risk)(^1,2)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>In moderate to advanced glaucoma:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• with extensive RNFL loss (floor effect at ~50μm), macular analysis may be more valuable to detect change(^3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>With anomalous ONH and parapapillary retina:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• macular analysis may be more valuable than ONH/RNFL analyses(^4)</td>
</tr>
</tbody>
</table>

Detecting structural progression

Guided Progression Analysis

Event-based analysis:
- highlights change from baseline that exceeds normal variability
  - change on one occasion: possible loss
  - change on two or more successive occasions: likely loss

Detecting structural progression

**Guided Progression Analysis**

Trend-based analysis:
- plots thickness over time to quantify rate of change in:
  - average, superior, and inferior RNFL thickness
  - average C/D ratio
  - average, superior, and inferior GCIPL thickness

---

Detecting structural progression

**Guided Progression Analysis**

**GPA: RNFL**
- look for widening of an existing inferior-temporal RNFL defect
- inter-visit changes in average RNFLT ≥5μm are suspicious
- a rate of average RNFL thinning ≥2 to 3μm/yr. is suspicious

Detecting structural progression

**Guided Progression Analysis**

**GPA: GCIPL**

- GCIPL loss doubles in progressive disease of any severity\(^1\)
- inter-visit changes in average GCIPLT ≥4μm are suspicious\(^2\)
- a rate of average GCIPL thinning ≥1 to 1.5μm/yr. is suspicious\(^3\)

---

RNFL versus GCIPL in advanced glaucoma

Quantitative structural progression: summary

**Early (including pre-perimetric) to moderate glaucoma:**
- an inter-visit change in average RNFLT ≥5μm
- a rate of average RNFL thinning ≥1.5 to 2μm/year

**Moderate to advanced glaucoma:**
- an inter-visit change in average GCIPLT ≥4μm
- a rate of average GCIPL thinning ≥1 to 1.5μm/year

**Remember:**
- smaller changes are more significant in advanced disease
- OCT cannot detect DH or NRR pallor
- allow for age-related loss of 0.4 to 0.6μm/year
- rates of change are correlated: ‘unilateral’ change is rare

---

Detecting functional progression

**Visual field analysis**
- usually deepening or expansion of an existing VF defect
- less commonly the appearance of a new defect

Detecting functional progression

Look for:

- an **increase in pattern standard deviation (PSD)**: focal VF loss\(^1\)
- a **glaucoma hemifield test (GHT)** that is **outside normal limits**\(^2\)

Event-based analysis: detects **statistically significant change** in **individual VF points** of a follow-up test compared to baseline

- baseline tests must be reliable
- early detection of change, albeit with more variability\(^3\)

Trend-based analysis: based upon the Visual Field Index (VFI), a center-weighted measure of **global residual visual function**\(^4\)

- allows rate of change to be quantified and extrapolated\(^5\)
- requires more tests; less sensitive to **diffuse loss (15%)**\(^6\)

---

Detecting functional progression

Event-based Humphrey AVF analysis

<table>
<thead>
<tr>
<th>Graytone</th>
<th>Pattern Deviation</th>
<th>Deviation From Baseline</th>
<th>Progression Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feb 05, 2016 SITA-Fast</td>
<td>GHT: Outside Normal Limits</td>
<td>3 5</td>
<td>Visual Acuity:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>-11 -2 -2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>-4 -3 -1 2 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>-10 -3 0 1 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>-18 -4 -1 2</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>-4 1 3</td>
<td></td>
</tr>
<tr>
<td>Fovea: 32 dB MD: -7.20 dB P &lt; 0.5% PSD: 4.48 dB P &lt; 0.5%</td>
<td>0 0 2</td>
<td>0 0 3 2</td>
<td>4 0</td>
</tr>
<tr>
<td>VFI: 86% FL: 0/12</td>
<td>-1 -3</td>
<td>-1</td>
<td>-1 -3</td>
</tr>
</tbody>
</table>

Statistically significant change may not always be clinically significant change

---

Detecting functional progression

Progression requires confirmation\(^1,2\)

In OHTS, 86% of patients with one abnormal AVF reverted to normal on the next assessment

66% did so with two consecutive abnormal AVFs

Only 12% did so with three consecutive abnormal AVFs


Detecting functional progression

At least 6 reliable AVF assessments within the first 24 months are essential to identify potentially catastrophic progression of ≥ -1.5dB (or -5% VFI) per year

In real life, AVFs are performed far too infrequently (~0.7/year)

Study the event analysis:
up to 50% of patients with OAG show exponential local progression that may go undetected by global indices (VFI trend analysis) that assume linear rates of change

Trend-based analysis may underestimate risk

Although the trend analysis appears relatively stable (flat) ...

... the event analysis shows localized progression that is beginning to encroach on fixation
Which is better, one (OCT) or two (AVF)?

RNFL assessment is linear; AVF assessment is logarithmic

35 to 50% of RGC may be lost before VF loss is detected

There is a tipping point at an average RNFLT of ~75μm (~17% loss of total RNFLT, but ~40% loss of functional RNFLT) at which detectable AVF progression increases

Which is better, one (OCT) or two (AVF)?

Utilize all the tools at your disposal:
progression is usually detected by only one method\(^1\)

<table>
<thead>
<tr>
<th>Early (including pre-perimetric) glaucoma:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• clinical exam and OCT (RNFL ≥ GCIPL &gt; ONH)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Moderate glaucoma:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• clinical exam and OCT (RNFL ≥ GCIPL &gt; ONH)</td>
</tr>
<tr>
<td>• AVF (24-2, and 10-2 guided by GCIPL abnormalities)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Advanced glaucoma:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• 10-2 &gt; 24-2 AVF (or 24-2 with stimulus size (V))</td>
</tr>
<tr>
<td>• clinical exam and OCT (GCIPL &gt; RNFL &gt; ONH)</td>
</tr>
</tbody>
</table>

Leverage OCT in early and AVF in advanced disease\(^2\)

---

Altering treatment

Due to exceeding target IOP or documenting progression

- with increasing disease severity, up to 80% of patients will require more than one medication to reach target IOP\(^1\)
- consider switching before adding medications
  - ... including switching from one PGA to another\(^2\)

Be aware of generic substitution\(^3\)
- IOP control: branded > generic?

*Even a small IOP drift may increase progression (EMGT/CGS)*\(^4\)

Be aware of the law of diminishing returns\(^5\)
- IOP reduction: primary > adjunctive (by \(\sim 2\) to 3mmHg)

---

### Initiating and altering treatment

1. PGA QD (pm) monotherapy
   - bimatoprost ≥ travoprost ≥ latanoprost?¹

<table>
<thead>
<tr>
<th>Xalatan</th>
<th>Travatan Z/Izba</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lumigan/Vistitan</td>
<td>Saflutan/Zioptan</td>
</tr>
</tbody>
</table>

2. a) unfixed combinations (add to PGA)
   - add CAI ≥ add BB > add AA²,³
   - CAI and AA BID; BB QD (am)

<table>
<thead>
<tr>
<th>Trusopt/Azopt</th>
<th>Timoptic/Betoptic/Betagan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alphagan (P)</td>
<td></td>
</tr>
</tbody>
</table>

2. b) PGA/BB fixed combination (FC)⁴
   - QD am or pm

<table>
<thead>
<tr>
<th>DuoTrav</th>
<th>Xalacom</th>
</tr>
</thead>
</table>

#### SLT may be considered at any point as primary or adjunctive therapy

---

2. Reis R, et al. A randomized, investigator-masked, 4-week study comparing timolol maleate 0.5%, brinzolamide 1%, and brimonidine tartrate 0.2% as adjunctive therapies to travoprost 0.004% in adults with primary open-angle glaucoma or ocular hypertension. *Clin Ther* 2008;28:352-9.
4. Topouzis F, et al. A 1-year study to compare the efficacy and safety of once-daily travoprost 0.004%/timolol 0.5% to once-daily latanoprost 0.005%/timolol 0.5% in patients with open-angle glaucoma or ocular hypertension. *Eur J Ophthalmol* 2007;17:183-90.
### Initiating and altering treatment

<table>
<thead>
<tr>
<th>3. a) PGA/BB FC + stand alone CAI or AA</th>
<th>3. b) stand alone PGA + BB FC</th>
<th>4. PGA/BB FC + non-BB FC</th>
</tr>
</thead>
<tbody>
<tr>
<td>add CAI = add AA, both BID</td>
<td>add CAI/BB = add AA/BB, both BID</td>
<td>add CAI/AA TID$^2$</td>
</tr>
</tbody>
</table>

**Cosopt/Azarga Combigan**

**Simbrinza**

---

**SLT may be considered at any point as primary or adjunctive therapy**

---


2. Whitson JT, et al. Six-month results from a Phase III randomized trial of fixed-combination brinzolamide 1% + brimonidine 0.2% versus brinzolamide or brimonidine monotherapy in glaucoma or ocular hypertension. *Clin Ophthalmol* 2013;7:1053-60.
Initiating and altering treatment

Although there is no cookbook, treatment may resemble:

1. Initiate treatment with Lumigan RC HS
   - 1 medication, 1 bottle, 1 drop
2. Alter treatment by changing to DuoTrav QD am
   - 2 medications, 1 bottle, 1 drop
3. Alter treatment by continuing DuoTrav QD am and adding Azopt BID
   - 3 medications, 2 bottles, 3 drops
4. Alter treatment by continuing DuoTrav QD am, discontinuing Azopt, and adding Simbrinza TID (perhaps BID)
   - 4 medications, 2 bottles, 4 (perhaps 3) drops

**SLT may be considered at any point as primary or adjunctive therapy**
Altering treatment

Lowering IOP an additional 10% halves the rate of progression\textsuperscript{1,2}

\textbf{1.} Aptel F, et al. Change in visual field progression following treatment escalation in primary open-angle glaucoma. \textit{J Glaucoma} 2017; doi:10.1097/IJG.748.

Altering treatment

Due to treatment intolerance or adverse effects

- 50% of patients report ocular surface disease (OSD) that is often induced or exacerbated by preservatives, most commonly benzalkonium chloride (BAK)\(^1\)
  - BAK is toxic to the conjunctiva, cornea, and TM\(^2-4\)
  - BAK increases the risk of failure of filtration surgery\(^5\)

- non-BAK preserved or preservative-free medications:
  - Timoptic XE; Alphagan P; Travatan Z; DuoTrav PQ
  - Timoptic Ocudose; Cosopt PF; Saflutan/Zioptan (tafluprost)
    - *SLT and/or MIGS?*

Treat the patient, not the pressure

The goal of treatment is **not** to simply lower IOP:

- treatment aims ‘... to enhance ... quality of life by preserving visual function ... at a sustainable cost ... without causing untoward side effects from treatment’

50% of patients discontinue treatment within 6 months

‘Drugs don’t work for patients who don’t take them’

‘More than half of the progression in treated OAG may be attributable to poor adherence with treatment’ (WGC 2013)

Treatment of an asymptomatic disease must be convenient, affordable, and tolerable

---

Altering treatment

Adherence decreases as number of daily doses increases\(^1\)

Fixed combinations decrease and simplify dosing:
- increasing convenience and patient adherence\(^2\)
- reducing AA allergy (due to BID versus TID dosing?)\(^3\)
- reducing or completely eliminating BAK exposure
- reducing washout of first drop by instillation of second\(^4\)

However, FC have some restrictions:
- drug concentration (0.25% BB not available)
- frequency and timing of dosing (BB QD vs. BID; am vs. pm)

For the most part, the upsides of FC outweigh the downsides

---

3. Sherwood MB, et al. Twice-daily 0.2% brimonidine-0.5% timolol fixed-combination therapy vs monotherapy with timolol or brimonidine in patients with glaucoma or ocular hypertension. *Arch Ophthalmol* 2006;124:1230-8.
So, you’ve diagnosed glaucoma ... now what?

1. Establishing solid baseline data
2. Staging disease severity
3. Establishing a target IOP
4. Initiating treatment
5. Detecting disease progression
6. Altering treatment
Please remember to complete your session evaluations online
Tweet about this session using the official meeting hashtag
#academy17