BLOOD FLOW IN GLAUCOMA – INSIGHTS AND PERSPECTIVES

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David Sackett, MD [1934-2015]

• Widely regarded as the father of evidence-based medicine.

Half of what you’ll learn during training will be shown to be either dead wrong or out-of-date within 5 years . . . ;

...the trouble is that nobody can tell you which half.

How many states have only four letters?

• Clue: Ohio is not one

Ocular Blood Flow and glaucoma?
State of the science 2009

“At the present time, no single blood flow imaging device is capable of evaluating ocular blood flow relevant to glaucoma.

“A comprehensive approach, utilizing multiple imaging technologies is required for meaningful insight into the multiple vascular beds of the eye.”

Consensus statement of the WGA 2009

Seriously . . .

POAG is a progressive, chronic optic neuropathy in adults in which intraocular pressure (IOP) and other currently unknown factors contribute to damage and in which there is a characteristic acquired atrophy of the optic nerve and loss of retinal ganglion cells and their axons. This condition is associated with an anterior chamber angle that is open by gonioscopic appearance.

—ala AAO PPP
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-ala AAO PPP

“Can glaucomatous optic neuropathy be induced by a primary non-IOP-related insult...alone?” - Claude Burgoyne

CONCLUSION
For years, fierce discussions have occurred between supporters of the mechanical and vascular theories for the pathogenesis of glaucoma. The concept of IOP and the identification of this as an important risk factor for the development and progression of glaucoma brought together the vascular and mechanical components of glaucoma. We believe that it is the balance between IOP and BP, influenced by the autoregulatory capacity of the eye, that determines whether an individual will develop optic nerve damage. However, further research is required to evaluate the importance of IOP and its fluctuation as parameters to be measured in glaucoma patients.

At the present time, no SINGLE blood flow imaging device is capable of evaluating ocular blood flow relevant to glaucoma.

“A comprehensive approach, utilizing multiple imaging technologies is required for meaningful insight into the multiple vascular beds of the eye.”

Consensus statement of the WGA 2009
OCT will revolutionize the diagnosis, management & understanding of glaucoma...

- Higher resolution
- Differential depth scans
- O/R applications
- Smartphone app
- OCT angiography!!!
note area above ONH with demonstrable RPE loss and prominence of choroidal vasculature. AND, absence of superficial as well as deep retinal capillaryplexuses.

Compare with OCT X-section showing thinned retina.

Optical Coherence Tomography Angiography of Optic Disc Perfusion in Glaucoma

Yuli Ku, MD, PhD, Fei Wu, BS, Xiaogang Kong, MD, Zehua Zhang, MD, and Julio C. Merida, MD

Purpose: To compare optic disc perfusion between normal subjects and subjects with glaucoma using optical coherence tomography angiography (OCTA) angiography and to detect optic disc perfusion changes in glaucoma.

Design: Observational, cross-sectional study.

Participants: Twenty-four normal subjects and 11 patients with glaucoma were included.

Methods: High-definition OCTA imaging was performed on a high-definition 1024x1024 mm2-sweep rate source OCT system. The anti-skyrmion amplitude-decorrelation angiography (SSDA) algorithm was used to compute 3-dimensional optical disc angiography. A disc flow index was calculated from a region-of-interest (ROI) centered on the optic disc and defined by the optic disc margin on OCT B-scans. The flow index was used to measure retinal nerve fiber layer (RNFL) thickness, RNFL tissue perfusion, and compared with peripapillary RNFL thickness. The Spearman’s rank correlation test was used to assess the sensitivity and specificity. Comparisons between glaucoma and normal groups were analyzed by Wilcoxon rank-sum test. Correlations among disc flow index, structural assessments, and visual field parameters were determined by linear regression.

Results: A normal disc, a dense microvascular network was visible on OCT angiography. This network was visible attenuated in subjects with glaucoma. The normal-to-dysfunction ratio, standard deviation, and normal population variability of the optic disc flow index were 5.2%, 4.5%, and 5.0% OU, respectively. The disc flow index was reduced by 25% in the glaucoma group (P < 0.001). Sensitivity and specificity were both 100% using an optimized cutoff. The flow index was highly correlated with VF loss (R^2 = 0.752, P < 0.001). These correlations were significant even after accounting for age, CDR area ratio, MFL, and rim area.

Conclusions: Optical coherence tomography angiography, generated by the new SSDA, repeatedly measures optic disc perfusion and may be useful in the evaluation of glaucoma and glaucoma progression. Ophthalmology 2014;121:1222-1232 © 2014 by the American Academy of Ophthalmology.
Generalized and local effects...

**Capillary Density and RNFL Thickness Comparison**

33 POAG pts. with VF depressions unilaterally (MD = -3.91 +/- 3.09 and normal fellow eye (MD = -0.2 +/- 0.9)

- whole image vessel density (wiVD)
- circumpapillary vessel density (cpVD)
- parafoveal vessel density (pfVD)

Mean wiVD in unaffected eyes of patients with POAG was higher than in their fellow affected eyes but lower than in healthy eyes (P < 0.001).

Mean circumpapillary RNFL (cpRNFL) thickness, mGCC thickness were also higher than those measurements in fellow eyes.

"OCT-A measures detect changes in retinal microvasculature before VF damage is detectable in patients with POAG, and these changes may reflect damage to tissues relevant to the pathophysiology of glaucoma."


**Choroidal Microvascular Dropout**

(Focal sectoral capillary dropout with no visible microvascular network identified in the choroidal layer)

32 Patients with an initial parafoveal scotoma (IPFS) within a 10° radius in 1 hemifield and, 42 POAG patients with an initial nasal step (INS) within the nasal periphery outside 10° of fixation in 1 hemifield.

Microvasculature dropout (MvD) was observed in 25 of 32 eyes (78.1%) in the IPFS group, but in only 1 of 42 eyes (2.4%) in the INS group (P < 0.001).

"The presence of MvD in the parapapillary choroid was a strong predictor for IPFS."


**Retinal Blood Flow in Glaucomatous Eyes with Single-Hemifield Damage**

Conclusions: In glaucomatous eyes with single-hemifield damage, the RBF is significantly reduced in the hemispheres associated with the affected hemifield. Reduced RBF is associated with thinned RNFL and GCC in the corresponding affected hemispheres. Reduced RBF and RNFL, and GCC have also been observed in the peripherally normal hemispheres of glaucomatous eyes.

Maybe this helps explain the asymmetry that is so prevalent in glaucoma.

Think: VF, rim tissue, PPA...
Proposed mechanisms

**A**

- Elevated IOP
- Loss of retinal ganglion cells & nerve fibers
- Loss of visual field
- Decreased blood flow

Reduced blood flow could be a consequence of neural tissue loss arising from elevated IOP


**B**

- Elevated IOP
- Decreased blood flow
- Loss of retinal ganglion cells & nerve fibers
- Loss of visual field

Reduced blood flow and elevated IOP could both lead to neural structure loss


**C**

- Elevated IOP
- Decreased blood flow
- Loss of retinal ganglion cells & nerve fibers
- Loss of visual field

Reduced blood flow could be an independent cause of VF loss


**Proposed mechanism**

And more evidence...

- association between glaucoma* and vascular dementia* but not between glaucoma and Alzheimer disease*.
- [*Alzheimer and vascular dementia are both neurodegenerative diseases and glaucoma is now being lumped into that bucket, too.]


Ocular Perfusion Pressure & Glaucoma Progression – emerging paradigms

- Increased IOP
- Sustained Impact Perfusion Pressure

Hayreh SS. Trans Am Acad Ophthalmol 1974;78:240-54

Dx: POAG, ???

Is there a blood-flow problem here???
Optic Nerve HEAD anatomy – blood flow considerations


Structural evaluation - Diagnosis enhanced depth imaging [choroid]

- Choroidal thickness and perfusion/flow evaluation

- Age, axial length, CCT, and diastolic ocular perfusion pressure are significantly associated with choroidal thickness in glaucoma suspects and glaucoma patients.

- Degree of glaucoma damage was not consistently associated with choroidal thickness.


Hey! Maybe its choroidal blood flow

Choroidal blood flow (arbitrary units)

Cellular and physiological mechanisms underlying blood flow regulation in the retina and choroid in health and disease

Journal of the Retinal and Eye Research


Hey! Maybe its choroidal blood flow. After all that seems to be the case in AMD

Implications of BF alterations with ↑↑ IOP

Note: increased IOP induces

- posterior rotation of the peripapillary sclera
- flattening of the cup floor
- thinning of the lamina cribrosa and the preppapillary neural tissue and
- anterior movement of the central optic nerve relative to the LC

Which may be complementary to reduced blood flow OR a result of same

**Structural evaluation – diagnosis**

- *Lamina cribrosa evaluation*

- **Emerging investigations:** CSF pressure (see: later)

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**Analyzing the flow**

- *Ophthalmology*

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**Blood supply summary**

- Interindividual variation
- Retinal nerve fiber layer
  - CRA / CRV
- Optic nerve head
  - SPCaa
  - choroidal plexus
- blood supply is segmental

- Ultimate blood supply to RNFL and ONH is from the ophthalmic artery, a branch of the internal carotid artery

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**Vascular Theory of Glaucoma**

**Changes in ocular blood flow (OBF)**

- Reduced perfusion pressure (beyond autoregulatory capacity)

  - Secondary vascular degeneration following ganglion cell / RNFL loss

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**Vascular Theory of Glaucoma**

**Changes in ocular blood flow (OBF)**

- Peripheral vascular dysregulation - PVD

  - which can result in reperfusion injury (RI)

- All can be IOP independent and may involve both the retinal and choroidal circulatory systems.

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**Distribution of IOP in a general population**

- Implying an IOP-independent component in glaucoma ("NTG"???)

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**What are the possibilities in the absence of elevated IOP?**

- Primary / Peripheral vascular dysregulation

- Inadequate ONH perfusion

  - Let’s try and connect the dots
Relationship of perfusion to glaucoma

- Low diastolic ocular perfusion pressure may be associated with increased risk for POAG.
- This association was confirmed in subjects treated for systemic hypertension in subgroup analysis. This may support the hypothesis that the concept of ocular perfusion pressure status may be more relevant to glaucoma pathogenesis than ocular perfusion pressure alone.

Consult the patient’s beta-blocker prescriber in the context of progressive glaucoma damage with “good” IOP control.

Primary OBF component

- Risk factors (RF) for atherosclerosis are largely parallel to increased IOP
  - age
  - smoking
  - dyslipidemia
  - systemic hypertension
  - male sex
  - obesity

Therefore reducing these RF reduces IOP (slightly)
  - physical exercise
  - weight loss
  - treatment of dyslipidemia

- And may increase blood flow and aqueous outflow through the TM

‘Normal Tension Glaucoma?’

- Glaucomatous disc and field changes with IOP consistently < 22

  20% of newly diagnosed glaucoma patients have IOP < 21 mm Hg at presentation

- CAUSE ?? Decreased perfusion of disc (arteriosclerosis, low BP)

‘Normal Tension Glaucoma’

Recent evidence . . .

Primary Open-Angle Glaucoma vs Normal-Tension Glaucoma

Conclusions: Patients with POAG or NTG exhibit similar alternations in ocular and systemic circulation in the early stages of their disease process. This finding highlights the importance of considering vascular risk factors in both conditions and raises questions about the current separation of the two conditions into distinct clinical entities.

Published online September 10, 2012.
doi:10.1001/jamaophthalmol.1
Ocular Perfusion Pressure & Glaucoma Progression

Perfusion to the ONH

• DOPP (Diastolic ocular perfusion pressure) = DBP – IOP

(What is the number?)<40 is significant* – talk to the PCP)

– Reduced in POAG

Alternatively, mean perfusion pressure


Recent association between BP/OPP and structural glaucoma progression

• Two greatest risk factors
  – Older age
  – Lower diastolic BP

• Structural elements assessed – ONH (rim tissue), RNFL thickness.

Emerging importance of diastolic BP

- Low mean diastolic BP is consistently associated with structural glaucoma progression (Rim tissue, RNFL)


Association of Open-angle Glaucoma With Perfusion Pressure Status in the Thessaloniki Eye Study

2013,


*Significantly lower diastolic perfusion pressure was observed in those taking oral hypotensive medications (as in beta-blockers)
Contributing factors to abnormal neurovascular coupling in glaucoma

Conclusions from previous

Conclusions and future directions

One of the reasons why our understanding of the relation between OPP and glaucoma is still limited lies in the difficulties to measure retinal and ONH BF [55–58]. Dopppler optical coherence tomography may become a technique capable of measuring BF in a valid and reproducible way [59–61,62]. This improvement in technology is associated with the hope of gaining more insight into ocular BF regulation.

So, which is more important, lowered BP or elevated IOP?

Conclusions

• The results show that optic nerve head blood flow is more susceptible to an ocular perfusion pressure decrease induced by lowering the blood pressure compared with that induced by increasing the intraocular pressure.

• This blood flow autoregulation capacity vulnerability to low blood pressure may provide experimental evidence related to the hemodynamic pathophysiology in glaucoma.

NOCTURNAL HYPOPERFUSION AS A GLAUCOMA RISK FACTOR

Objective: The objective of this prospective, longitudinal study of patients with normal-tension glaucoma (NTG) was to determine whether patients with nocturnal hypotension are at greater risk for visual field (VF) loss over 12 months than those without reproducible hypotension.

Methods: The baseline evaluation assessed demographic and clinical characteristics, covering endocrine somnolent conditions, including systemic hypertension. All and oral antihypertensive medications were recorded. A complete ophthalmological examination was performed at baseline and follow-up. Patients had their blood pressure (BP) monitored every 20 minutes for 4 hours with an ambulatory recording device both at baseline and 2 and 12 months.

Outcome Measures: Primary outcome was based on the global sites of VF progression by linear regression of the mean VF threshold sensitivity over time (skewed-distributed).

Results: Eighty-nine patients with NTG (199 eyes) were mean age, 68 years; 97% were women included. Of the 88 patients, 20% had progressed in the 0.5 dB cutoff before study enrollment. The nocturnal mean arterial pressure (MAP) was controlled with the use of OMS. Multivariate analysis showed that the only time that mean arterial pressure (MAP) was associated with visual field progression was at 12 months, where a lower MAP was associated with a lower rate of visual field progression.

Conclusions: Nocturnal hypotension predicted VF loss in this cohort. Our data suggest that the duration and magnitude of decrease in nocturnal blood pressure below the diastolic MAP, nocturnal pressure that are 10 mmHg lower than diastolic MAP, predict progression of NTG. Low nocturnal blood pressure, whether measured spontaneously or as a result of medication, may lead to worsening of VF damage (OCT).
Conclusions and guidance

• In conclusion, the magnitude and duration of nocturnal hypotension identify patients with NTG who have VF progression.

• Ambulatory monitoring of systemic BP should become part of routine assessment of patients with NTG, particularly among those who continue to progress despite IOP lowering.

Conclusions and guidance

• Nocturnal BP should be considered a modifiable risk factor in NTG.

• Randomized trials will be required to assess the efficacy of different interventions designed to avoid nocturnal hypotension to prevent VF loss in patients with NTG, as well as to test the effect of more aggressive IOP-lowering therapy in these cases.

Conclusions and Guidance

• Blood flow measurements could guide changes in treatment protocol with emphasis on normalization of circulatory alteration rather than just IOP.

*Recent association between nocturnal BP dips and ODH in NTG

A reduction of nocturnal blood pressure (BP) in the range of 10%-20% relative to daytime BP levels is usually observed in normotensive subjects and in the majority of hypertensive patients.223 This dip is termed “physiologic,” while BPs that exhibit excessive (>20%) or minimal (<10%) dips at night are termed “nonphysiologic” dippers. over-dippers = progressors

*Recent association between nocturnal BP dips and ODH in NTG

over-dippers = progressors
Reduced perfusion - More Risk factors

- Autoregulation disturbances
- Vasospastic Disorder
- Migraine
- Increased resistance

- ✔ Reduced blood flow (20 low BP) →
  **Nocturnal hypoperfusion**

- **Sleep apnea syndrome**

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**SAS and Normal Tension Glaucoma**

- 50 sleep apnea patients were compared with 40 normals
- Prevalence of NTG among SAS pts was 5.9% (and 0% among the controls)
- Severity of SAS was correlated positively with [structural and functional elements]
  - IOP
  - MD
  - C/D
  - mean NFL thickness (HRTII)

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**SAS – Glaucoma connection (additional evidence)**

- The prevalence of glaucoma in patients with obstructive sleep apnea is an estimated 27%!

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**SAS – Glaucoma connection (further evidence)**

- In patients with OSAS, a high prevalence of glaucoma was found.
- Visual field defects may be due to optic nerve perfusion defects and these field defects also increase as the RI (resistance index) increases.

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**Ocular blood flow and Obstructive Sleep Apnea Syndrome (OSAS)**

- 31 patients with proven OSAS / 25 controls
- 12.4% of OSAS and none of the controls were diagnosed with glaucoma
- No differences in retinal circulation measures or IOP (implying IOP-independent risks)
- Positive correlation between MD and LV & retinal circulatory measures

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**And, more recently raised questions...**

- Should OSAHS be included in the DDx of glaucoma?
- Is OSAHS another glaucoma or a contributor?
- Does lowering IOP in OSAHS arrest the progression of optic neuropathy?

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Limitations and confounders


**Fair and balanced**

- Found that there IS a relationship between IIH and AION and those using a C-PAP but **not** between glaucoma and C-PAP use.


**Obstructive Sleep Apnea and Increased Risk of Glaucoma**

A Population-Based Matched-Cohort Study 2013,

Chen CH, et al.

**Obstructive Sleep Apnea and Increased Risk of Glaucoma**

A Population-Based Matched-Cohort Study

In conclusion, our results suggest that OSA is associated with an increased risk of subsequent OAG diagnosis during the first 5 years after OSA diagnosis. We found that the hazard of receiving an OAG diagnosis during the 5-year follow-up period was 1.67 times greater in patients with OSA than in gender- and age-matched comparison subjects, after adjusting for socioeconomic characteristics and medical comorbidities. The authors hope that this study encourages clinicians to alert OSA patients of the association between OSA and OAG as a means of raising the awareness and encouraging treatment of those who need it.

And, the very latest!

Prevalence and risk factors of eye diseases in adult patients with obstructive sleep apnoea: results from the SLE.E.P.Y cohort study

Prevalence and risk factors of eye diseases in adult patients with obstructive sleep apnoea: results from the SLE.E.P.Y cohort study

And, the very latest!
And, the very latest!

**BMJ Open** Prevalence and risk factors of eye diseases in adult patients with m in general population.

The fourth most frequently observed ED was glaucoma.

First, lower IOP

- Systemically (regulating blood pressure and monitoring perfusion pressure)
- Locally – endothelial-cell activity by modulating Nitric Oxide (NO) This is the NEXT BIG THING!
  - Regulation of aqueous dynamics at the trabecular meshwork by vascular modulation
  - In addition, the application of NO-donating compounds for the lowering of IOP directly

New directions in glaucoma treatment

- Yes, treatment
- Beyond IOP reduction, regulation of blood flow . . .
  - Systemically (regulating blood pressure and monitoring perfusion pressure)
  - Locally – endothelial-cell activity by modulating Nitric Oxide (NO) This is the NEXT BIG THING!

Future options for medical management – targeting the site of glaucoma, the TM

- latanoprostene bunod (a nitric oxide-donating compound, NO) Vyzulta (Valeant)
- FDA-approved November 2017
- **MOAs:**
  - Relax the cellular matrix of the TM (and perhaps more distally)
  - May also act at the endothelium of TM blood vessels to constrict and therefore further open drainage pathways.

Future options for medical management – targeting the site of glaucoma, the TM

- Rho-kinase inhibitors (Rhopressa and Rocklatan, (netarsudil/latanoprost ophthalmic solution, 0.02%/0.005%, Aerie)
- FDA-approved December 2017
- **MOAs:**
  - Increase fluid outflow through the trabecular meshwork, (1° drainage)
  - Increase fluid outflow through the uveoscleral pathway, (2° drainage)
  - Reduce fluid production in the eye, and
  - Reduce episcleral venous pressure (EVP).
How should glaucoma be managed comprehensively?

- **Second**, consider increasing perfusion (may be a consequence of lowered IOP)
  - Topical treatments? (betaxolol, brimonidine, brinzolamide, 
  - Gingko Biloba and other CAM interventions
  - Exercise, weight loss
  - Lower cholesterol, blood sugar levels
  - Treat underlying vascular disorders (HT, SAS, CVD)
  - Etc.

The effects of antioxidants on ocular blood flow in patients with glaucoma


**Study design**

- 45 patients with confirmed glaucoma on IOP-lowering treatment (placebo controlled, X-over)
- Baseline and post-administration (@ 1 month) measurements
  - IOP
  - OPP
  - Retrobulbar (ultrasound) and retinal capillary (Doppler) blood flow

**Results**

- Increased peak systolic and/or end diastolic velocities among the active group (but not placebo)
- Reduced vascular resistance in central retinal and short posterior ciliary arteries
- Increased superior and inferior temporal retinal artery mean blood flow
- Enhanced retinal capillary density

**SO, what were they given?**

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin C (ascorbic acid)</td>
<td>250 mg</td>
</tr>
<tr>
<td>Vitamin E (a-tocopherol, mixed tocopherols)</td>
<td>30 IU</td>
</tr>
<tr>
<td>Vitamin B6 (pyridoxine hydrochloride)</td>
<td>10 mg</td>
</tr>
<tr>
<td>Folate (50% folic acid, 50% calcium folinate)</td>
<td>400 mcg</td>
</tr>
<tr>
<td>Vitamin B2 (riboflavin)</td>
<td>300 mcg</td>
</tr>
<tr>
<td>Magnesium (magnesium oxide, aspartate)</td>
<td>120 mg</td>
</tr>
<tr>
<td>Taurine</td>
<td>250 mg</td>
</tr>
<tr>
<td>N-Acetylcysteine (NAC)</td>
<td>300 mg</td>
</tr>
<tr>
<td>Alpha Lipoic Acid</td>
<td>200 mg</td>
</tr>
<tr>
<td>Gingko Biloba Extract (15% girken flavone glycosides)</td>
<td>150 mg</td>
</tr>
<tr>
<td>Omega-3 Fatty Acid (Docosahexaenoic acid 100 mg, Eicosapentaenoic acid 20 mg)</td>
<td>120 mg</td>
</tr>
<tr>
<td>Bilberry fruit extract (25% anthocyanidins)</td>
<td>115 mg</td>
</tr>
<tr>
<td>Coenzyme Q10 (CoQ10)</td>
<td>50 mg</td>
</tr>
<tr>
<td>Grape seed extract (95% proanthocyanidins)</td>
<td>50 mg</td>
</tr>
<tr>
<td>Quercetin</td>
<td>50 mg</td>
</tr>
</tbody>
</table>
| Flax seed oil (400 mg omega-3), gelatine, glycerine, 
  - water, benzyl, linitol (from soya beans), lemon 
  - oil flavouring, caramel colour, and titanium dioxide |
How should glaucoma be managed comprehensively?

• Third, reduce oxidative stress (Ca++ blockade [BUT, not systemic β-blockers], supplements) AND enhance blood flow!

NON-SELECTIVE Beta-blockers: Significant additional precaution

Topical β-blockers administered at night to those taking systemic β-blockers may reduce perfusion to the ONH plus β-blocker therapy to reduce IOP is ineffective at night.

Which brings us to . . .

Consider this:

• Is glaucoma AION that happens over a lifetime?
• OR
• Is AION glaucoma that happens overnight?

What happens to glaucoma patients during sleep?

2013, Ahmed A. Ann.

Summary
Several nighttime events including increased IOP, decreased OPP, and possibly OSA contribute to the development and progression of glaucomatous optic neuropathy. These events may explain the occurrence and progression of glaucomatous disease in the setting of seemingly controlled IOP.

KEY POINTS
• Peak intracocular pressure, which has been found to be the best predictor of glaucomatous visual field progression, most likely occurs at night.
• Nocturnal intracocular pressure is dependent on the body position and may be significantly lowered in a 30° head-up position during sleep.
• A decrease or fluctuation in nocturnal ocular perfusion pressure increases the risk of glaucomatous visual field progression.
• The relationship between obstructive sleep apnea and glaucoma remains unclear, with smaller prospective studies reporting a positive association and larger retrospective cohort studies declaring no association.

62 WM

- Complained of vision loss superiorly in the left eye (May, 2018)
- VA 20/20 OD, OS; (L)RAPD 2+; IOP 11,9 mmHg.
- Seen by primary-care OD – Dx = NTG, initiated on latanoprost qhs.

- Sent for consultation/SLT due to significant VF depressions. (June 2018)
The holy grail of glaucoma whether it is diagnosis or management is . . .

CONTINUOUS IOP MEASUREMENT

Closing thoughts

• How can IOP (and BP) be monitored continuously?

• What impact may this have on management?

States with only four letters

• Iowa
• Utah
• Mississippi
• Tennessee
• Alaska
• Hawaii
• Indiana
• Kansas

Thank You

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