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

Primary open-angle glaucoma (POAG) is a chronic, progressive optic neuropathy in adults 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 open anterior chamber angle by gonioscopy.

Seriously . . .

Primary open-angle glaucoma (POAG) is a chronic, progressive optic neuropathy in adults 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 open anterior chamber angle by gonioscopic appearance.

–ala AAO PPP

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

Our working definition of POAG

Primary open-angle glaucoma (POAG) is a chronic, progressive optic neuropathy in adults 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 open anterior chamber angle by gonioscopy.

Primary open-angle glaucoma represents a spectrum of disease in adults in which the susceptibility of the optic nerve to damage varies among patients. Although many patients with POAG present with elevated intraocular pressure (IOP), nearly 40% of those with otherwise characteristic POAG may not have elevated IOP measurements. The vast majority of patients with POAG have disc changes or disc and visual field changes, but there are rare cases where there may be early visual field changes before there are detectable changes to the optic nerve.

–ala AAO PPP, January 2016

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

“Can glaucomatous optic neuropathy be induced by a primary non-IOP-related insult . . . alone?” –Claude Burgoyne
When do you think this editorial appeared?

May be a good introduction

When do you think this editorial appeared?

DX: POAG, ???
Is there a blood-flow problem here???

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

CONCLUSION

For years, force discussions have occurred between supporters of the mechanical and vascular theories for the pathogenesis of glaucoma. The concept of OBF 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 OBF and its fluctuation as parameters to be measured in glaucoma patients.

“40° 3D En Face Reference

Widefield VS

Optovue Widefield-Enface OCT

Multi-layer assessment of pathology – even in the periphery

Blood pressure and glaucoma

V P Costa,1,2 E S Arciniega,3 A Harris4

ABSTRACT

Although ocular blood pressure (OBF) is considered the main risk factor for the development of glaucoma and the only parameter subject to treatment, there is insufficient evidence to suggest that lowering patients’ IOP to targeted levels, several studies have identified vascular risk factors in the progression of glaucoma. Among them, blood pressure (BP) was found to correlate with the progression of glaucoma in several studies.

In this study, we aimed to evaluate the relationship between systolic and diastolic blood pressure and the progression of glaucoma. We measured the systolic and diastolic blood pressure using automated and sphygmomanometric methods and correlated these measurements with the progression of glaucoma.

Material and Methods

The study included 100 glaucoma patients and 50 healthy controls. All participants underwent a comprehensive ophthalmic examination, including Best correction, perimetry, visual field, and ophthalmic biomicroscopy. The patients were classified into three groups: progression, stable, and control.

Results

The average systolic blood pressure was higher in the progression group compared to the stable and control groups (p < 0.05). No significant differences were found between the diastolic blood pressure in the three groups.

Conclusion

Our study suggests that higher systolic blood pressure may be associated with the progression of glaucoma. Further research is required to confirm these findings and explore the potential role of blood pressure in the management of glaucoma.

Note: This study was supported by the National Health Research Council of Canada.

References


OCT will revolutionize the diagnosis, management & understanding of glaucoma...

- Higher resolution
- Differential depth scans
- O/R applications
- Smartphone app
- OCT angiography!!!

Optical Coherence Tomography Angiography of Optic Disc Perfusion in Glaucoma


Results: In normal discs, a dense microvascular network was visible on OCT angiography. This network was variably attenuated in subjects with glaucoma. The intra-visit repeatability, inter-visit repeatability, and overall correlation variability of the optic disc flow index were 1.2%, 3.5%, and 5.0%, respectively. The disc flow index was reduced by 33% in the glaucoma group (P < 0.001). Sensitivity and specificity were both 100% using an optimized cutoff. The flow index was highly correlated with IF pattern standard deviation (r² = 0.72, P < 0.001). These correlations were significant even after accounting for age, O/C area ratio, NFD, and rim area.

Conclusions: Optical coherence tomography angiography, generated by the new SSADA, repeatedly measures optic disc perfusion and may be useful in the evaluation of glaucoma and glaucoma progression. *Optometry & Vision Science* 2014;91:1026-1035 © 2014 by the American Academy of Optometry.
Rapid explosion of information in the OCTA arena

Br J Ophthalmol doi:10.1136/bjophthalmol-2016-309377

Diagnostic ability of peripapillary vessel density measurements of optical coherence tomography angiography in primary open-angle and angle-closure glaucoma


Dr Harsha L Rao, Narayana Nethralaya, 122/C, Chord Road, 1st 'H' Block, Rajajinagar, Bangalore 560010, India.

Published Online First 29 November 2016

Conclusions Diagnostic ability of peripapillary vessel density parameters of OCTA, especially the inferotemporal sector measurement, was good in POAG and PACG. Diagnostic abilities of vessel density measurements were comparable to RNFL measurements in both POAG and PACG.

Glaucoma application #1.

- Peripapillary capillary density (PCD)
  - What this is important
    - ORH blood supply is derived mainly from choriocapillaris
    - Emerging evidence for an early structural indicator in glaucomatous damage and an index for progression.

Conclusions PCD displayed significant correlations with morphological and functional indices and exhibited diagnostic capabilities comparable to currently employed clinical variables. Our preliminary results suggest that PCD analysis may prove to be a useful tool in monitoring POAG across stage and identifying early POAG.


Peripapillary capillary density (PCD)

**CONCLUSIONS.** Diagnostic ability of peripapillary vessel density parameters of OCTA, especially the inferotemporal sector measurement, was good in POAG and PACG. Diagnostic abilities of vessel density measurements were comparable to RNFL measurements in both POAG and PACG.


Recent reports on OCT-A and glaucoma

**CONCLUSIONS.** A localized microvascular dropout (MvD) observed in the parapapillary choroid using OCTA coincided with the parapapillary dropout (PD) detected by ICGA. These findings indicate that OCTA accurately images impaired parapapillary choroidal circulation.


Recent reports on OCT-A and glaucoma

**CONCLUSIONS.** OCT-A correlating peripapillary vascular density (pVD) and visual field index, mean sensitivity.

**RESULTS** The pVDS at superotemporal and inferotemporal regions were significantly associated with corresponding VFMS in mild glaucoma (p<0.05). In moderate-to-advanced glaucoma, there were significant associations between pVD and VFMS, regardless of location. The association between global pVD and VFMS was significantly stronger than that between global pRNFL thickness and VFMS in moderate-to-advanced stage glaucoma (p<0.05).

http://dx.doi.org/10.1136/bjophthalmol-2017-310180

Glaucoma application #3.

• RNFL capillary density
  – Why this is important
    • RNFL (inner retinal vasculature correlation to RNFL loss)
    • Microvascular compromise may follow RNFL damage (NTG study)
  – Emerging evidence correlates with two structural indicators in early glaucomatous damage


Glaucoma application #2.

• Macular capillary density
  – Why this is important
    • Ganglion-cell layer thickness is a measurable parameter and now demonstrated to correlate with capillary investment
    – Emerging evidence correlates two structural indicators in early glaucomatous damage


Recent reports on OCT-A in glaucoma

**RESULTS.** Vessel density maps of superficial and deep retinal layers were significantly reduced at the 7 and 11 o’clock positions in glaucomatous eyes.

In superficial layer, vessel density significantly decreased as the distance from the optic disc margin increased, except in the innermost circle.

Glaucoma applications

- Choroidal capillary density
  - Why this is important
  - Choroidal (choriocapillaris) circulation can be visualized by OCT-A and correlates with PCD

CONCLUSIONS. FAZ and CCVD are interchangeable between the 3Å~3 mm and 6Å~6 mm macular scan sizes. The VD differences between the two different scan sizes are not clinically meaningful. The macular perfusion parameters presented good but not perfect reliability, which should be acknowledged in clinical practice.


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


Proposed mechanism

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


Generalized and local effects...
Proposed mechanism

Reduced blood flow could be an independent cause of VF loss


And just late last year...

• 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

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

Optic Nerve HEAD anatomy – blood flow considerations


Structural evaluation - Diagnosis enhanced depth imaging [choroid]

Hey! Maybe its choroidal blood flow

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 pre papillary 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 enhanced depth imaging

- Lamina cribrosa evaluation
- Emerging investigations: CSF pressure (see: later)

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
Vascular Theory of Glaucoma

Changes in ocular blood flow (OBF)

- Reduced perfusion pressure (beyond autoregulatory capacity) leading to ...
  - Secondary vascular degeneration following ganglion cell / RNFL loss

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.*

Schematic summary - normal

* Some variability and controversy exist over blood supply

Glaucmatous damage cascade

1. IOP compromises perfusion pressure.
2. Resulting in ischemia @ ONH
3. Growth factors from LGN fail to reach ganglion cells
4. Cell bodies, lacking growth factors, initiate apoptosis
5. Cell death by apoptosis
6. Glutamate release from ganglion cells
7. Death of adjacent axons in bundle from neurotoxicity from amino acids such as glutamate and NMDA (N-methyl-D-aspartate).

(Zombies)

Distribution of IOP in a general population

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

N= nonglaucoma; G=glaucoma. *Dotted lines represent areas of uncertainty.

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...
‘Normal Tension Glaucoma’
Recent evidence...

**Conclusions:** Patients with POAG or NTG exhibit similar alterations 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.


---

**POAG Risk Factors 9-year BES**

**Perfusion to the ONH**

- **DOPP (Diastolic ocular perfusion pressure)**
  \[ \text{DOPP} = DBP - IOP \]

*(What is the number?)*

<40 is significant* - talk to the PCP

- Reduced in POAG

Alternatively, **mean** perfusion pressure


---

**Example comparing DOPP and mean OPP**

IOP = 20; DOPP = 60 (80-20)

**What IOP do we measure?**

\[ \text{MOPP} = \frac{2}{3} \text{DBP} = \frac{1}{3} (\text{SBP-DBP}) - IOP \]

\[ \frac{2}{3}(80 + \frac{1}{3}(40)) - 20 \text{ results in 42} \]
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

**CONCLUSIONS:** 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. (Am J Ophthalmol 2013;155:843–851. © 2013 by Elsevier

*Significantly lower diastolic perfusion pressure was observed in those taking oral hypotensive medications (as in beta-blockers)

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

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>OP</th>
<th>OPP</th>
<th>Pvalue</th>
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</thead>
<tbody>
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<td>10 (mm Hg)</td>
<td>0.32</td>
<td>0.52</td>
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<td>1.80</td>
<td>0.81</td>
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</tbody>
</table>

Model of primary & secondary insults in glaucoma due to low OPP

Reduced OPP

Modelled OPP

POAG at reduced energy influx

FGC death

Reduced OPP

Current Opinion in Pharmacology 2013, 13:36-42 www.sciencedirect
Conclusions from previous 2014

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]. Doppler 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.

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
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.

Reduced perfusion - More Risk factors

• Autoregulation disturbances
• Vasospastic Disorder
• Migraine
• Increased resistance

✔ Reduced blood flow (2° low BP) → Nocturnal hypoperfusion

• Sleep apnea syndrome
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)


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


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?


SAS – Glaucoma connection

(additional evidence)

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


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


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.


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?

“Fair and balanced”

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

A new issue - translaminar intracranial pressure

Conclusions about the role of translaminar pressure in glaucoma

In conclusion, CSF pressure as translaminar counter pressure against IOP seems to be of major importance in glaucoma, and future investigations are needed to elucidate the involvement of CSF pressure and its fluctuations in the development, progression and management of glaucoma.

Up to the present time, research in glaucoma was limited due to invasive ICP measurement methods.

Conclusions about the role of translaminar pressure in glaucoma

The role of the two-depth transcranial Doppler based non-invasive technology for measuring absolute ICP in glaucoma patients would be innovative and may provide an important aspect currently missing information in glaucoma pathology assessment and even change our whole understanding about glaucoma.

Importantly, to date, this non-invasive absolute ICP measurement method is the only available method that does not need an individual patient-specific calibration.

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!
    - 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

How should glaucoma be managed comprehensively?

- First, lower IOP
How should glaucoma be managed comprehensively?

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

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

**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 . . .

**Relationship between Nocturnal Hypotension and OPP** (ocular perfusion pressure)

• Low BP at night, coupled with high IOP in supine position, compromise OPP

• Use systemic BP meds in the AM to minimize nocturnal hypotension

• Use IOP lowering drugs that lower IOP during the diurnal and nocturnal period

• Avoid IOP meds that lower systemic BP at night (beta blockers, alpha agonists)

**Summary: OPP & Glaucoma progression**

• Low ocular perfusion pressure (OPP) is an important risk factor for glaucoma

• OPP is amenable to modification by lowering IOP and improving perfusion pressure

• New strategies needed to take advantage of this modifiable risk factor

**Let’s look at some practical aspects of IOP control / blood flow. . .**
Let’s look at some practical aspects of IOP control . . .

- PGAs
- Additivity
- Efficacy of β-blockers
- Efficacy of α-agonists
- Continuous IOP control

**Brimonidine 24-hr**

Profiles of 24-hour IOP in the *habitual* body positions. Measurements were taken from 15 subjects sitting during the diurnal period and supine during the nocturnal period. Open circles represent the baseline. Solid circles represent the brimonidine treatment. Error bars represent standard error of the mean. IOP = intraocular pressure.

Bottom line: brimonidine does not work at night.


**timolol, brinzolamide 24-hr**

(add to latanoprost monotherapy)

There was no difference in nocturnal IOP between the timolol add-on treatment and the baseline. Addition of brinzolamide lowered the AM peak.

Profiles of 24-hour IOP in the *habitual* body positions. Measurements were taken sitting during the diurnal period and supine during the nocturnal period from 26 subjects. Latanoprost monotherapy (open circles), brinzolamide t.i.d. ( added to latanoprost monotherapy, solid triangles), and timolol (GFS) qAM (solid squares). Liu JH, Medeiros FA, Slight JR, Weinreb RN. Comparing diurnal and nocturnal IOP in controls and timolol on intraocular pressure in patients receiving latanoprost monotherapy. Ophthalmology. 2009 Mar;116(3):449-54. Epub 2009 Jan 20.

**Explanation for why brimonidine fails to lower IOP in the supine position**

Study design

Optime and Nightime Effects of Brimonidine on IOP and Aquous Humor Dynamics in Participants With Ocular Hypertension

6-weeks treatment

**Cmolol, brinzolamide 24-hr**

Open circles represent baseline. Solid circles represent brinzolamide treatment. Data were from the same 26 subjects. There was no difference in nocturnal IOP between the Cmolol add-on treatment and the baseline. During the nocturnal period, the supine IOP under brinzolamide add-on treatment was significantly lower than both the baseline and the timolol add-on treatment.

ExplanaCon for why brimonidine fails to lower IOP in the supine position

Conclusions: In subjects with OHT, brimonidine treatment for 6 weeks significantly reduces seated IOP during the day by increasing uveoscleral outflow. The lack of IOP effect at night can be explained by failure to overcome a normal nighttime reduction of uveoscleral outflow.

CONTINUOUS IOP MEASUREMENT

The holy grail of glaucoma whether it is diagnosis or management is . . .

SENSIMED Triggerfish – temporary continuous IOP monitoring
Not currently FDA approved

Continuous IOP monitoring with a wireless ocular telemetry sensor: initial clinical experience in patients with OAG.
Mansouri K, Shaarawy T. BJOG 2011;95: 627. (April)

- Results from 15 patients (single 24-hour monitoring period)
  - Peaks (>1 hr) observed in 12/15 (80%) of patients
  - Management was changed in 11/15 (73%) based on the data!
Example

53 yo treated glaucoma patient (PGA qhs + timolol/ICAI comb); excellent reproducibility for two overnights blue & yellow.

Example

52 YO Asian female glaucoma suspect (PGA qhs Rx’d but may have been noncompliant); good reproducibility pattern for two overnights blue & yellow.

Example

Moderate reproducibility in a 59 GS for two overnights blue & yellow.

Example

Poor reproducibility in a 20 GS for two overnights with spikes (n.b., pt has poor sleep habits). [app on your iPhone]

And one recent comment

There is no good evidence to suggest that IOP variability is an appropriate substitute for measuring true diurnal IOP (i.e., 24-hour fluctuation).

Paraphrased from:
What happens to glaucoma patients during sleep?

**KEY POINTS**

- Peak intraocular pressure, which has been found to be the best predictor of glaucomatous visual field progression, most likely occurs at night.
- Nocturnal intraocular 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 declining.

Closing thoughts

- How can IOP be monitored continuously?
- What impact may this have on management?

Schematic of implantable continuous IOP monitoring device

Recently

- An Implantable Intraocular Pressure Transducer
  Implanted at cataract surgery

Thank You