Glaucoma 2019 Crossing the Golden Gate to Exceptional Glaucoma Care

Under Pressure®

Program Directors

JoAnn Giaconi MD and Eydie G Miller-Ellis MD

In conjunction with the American Glaucoma Society

Moscone Convention Center San Francisco, California Saturday, Oct. 12, 2019

Presented by: The American Academy of Ophthalmology

Supported by an unrestricted educational grant from Aerie Pharmaceuticals, Inc.

Cover Photo courtesy of JoAnn Giaconi MD

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	James D Brandt MD

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	David S Friedman MD MPH PhD
2012	Wallace L M Alward MD
	Thomas W Samuelson MD
2011	Leon W Herndon MD
	Wallace LM Alward MD
2010	Rohit Varma MD MPH
	Leon W Herndon MD
2009	Donald L Budenz MD MPH
	Rohit Varma MD MPH
2008	Henry D Jampel MD MHS
	Donald L Budenz MD MPH
2007	Anne Louise Coleman MD PhD
	Henry D Jampel MD MHS
2006	Christopher A Girkin MD
	Anne Louise Coleman MD PhD
2005	Claude F Burgoyne MD
	Christopher A Girkin MD
2004	David S Greenfield MD
	Claude F Burgoyne MD
2003	Kuldev Singh MD MPH
	David S Greenfield MD
2002	Theodore Krupin MD
	Kuldev Singh MD MPH
2001	Robert D Fechtner MD
	Theodore Krupin MD
2000	Jeffrey M Liebmann MD
	Robert D Fechtner MD
1999	Robert N Weinreb MD
	Jeffrey M Liebmann MD
1998	George A Cioffi MD
	Robert N Weinreb MD





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2019 Glaucoma Subspecialty Day Planning Group

On behalf of the American Academy of Ophthalmology and the American Glaucoma Society (AGS), it is our pleasure to welcome you to San Francisco and Glaucoma 2019: Crossing the Golden Gate to Exceptional Glaucoma Care.



JoAnn Giaconi MD Program Director Allergan, Inc.: L



Eydie G Miller-Ellis MD Program Director

Aerie Pharmaceuticals, Inc.: C Allergan: S National Eye Institue: S

Program Planning Group



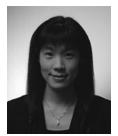
Lama Al-Aswad MD MPH Aerie Pharmaceuticals, Inc.: C GlobeChek: O New World Medical, Inc.: S Save Vision Foundation: S Topcon Medical Systems, Inc.: S Zeiss: S



Edward Barnett MD PhD None



Donald L Budenz MD MPH Bausch + Lomb: C Carl Zeiss Meditec: P Ivantis: C iView: C New World Medical, Inc.: C Nicox: C



Teresa C Chen MD Department of Defense: S Harvard Foundation Grant (Fidelity Charitable Fund): S



Vikas Chopra MD Allergan: C,S



lan Patrick Conner MD PhD Ocugenix: C,O,P

No photo available

Annette L Giangiacomo MD None



Davinder S Grover MD MPH Alcon Laboratories, Inc.: S Allergan: S,L,C Bausch + Lomb: L Glaukos Corp.: L MicroOptx: C New World Medical, Inc.: C,L Reichert, Inc.: C,L



Leon W Herndon Jr MD Aerie Pharmaceuticals: C Alcon Laboratories, Inc.: C Glaukos Corp.: L,S New World Medical, Inc.: C Sight Sciences: C



Dale K Heuer MD InnFocus: C National Eye Institute: S



Albert S Khouri MD Aerie Pharmaceuticals: C,L Allergan: L,S Glaukos Corp.: C New Jersey Health Foundation: S



Christine LeeAnn Larsen MD Aerie Pharmaceuticals, Inc.: L



John T Lind MD Aerie Pharmaceuticals, Inc.: C,L Allergan: C,L Perrigo: S

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None

Melanie Rafaty None

Debra Rosencrance None

Beth Wilson None

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CME Credit

The Academy's CME Mission Statement

The purpose of the American Academy of Ophthalmology's Continuing Medical Education (CME) program is to present ophthalmologists with the highest quality lifelong learning opportunities that promote improvement and change in physician practices, performance, or competence, thus enabling such physicians to maintain or improve the competence and professional performance needed to provide the best possible eye care for their patients.

2019 Glaucoma Subspecialty Day Meeting Learning Objectives

Upon completion of this activity, participants should be able to:

- Demonstrate familiarity with controversial management issues and current gaps in evidence-based glaucoma care
- Evaluate the current status of optic disc and retinal nerve fiber layer imaging and interpretation, as well as its role in diagnosing and managing glaucoma
- Demonstrate familiarity with current issues in medical and surgical therapy for glaucoma and how it affects other eye disease
- Recognize factors that complicate care of the glaucoma patient

2019 Glaucoma Subspecialty Day Meeting Target Audience

This activity has been designed to meet the educational needs of general ophthalmologists, glaucoma specialists and other ophthalmologic subspecialists, and allied health personnel who are involved in the management of glaucoma patients.

Teaching at a Live Activity

Teaching instruction courses or delivering a scientific paper or poster is not an AMA PRA Category 1 CreditTM activity and should not be included when calculating your total AMA PRA Category 1 CreditsTM. Presenters may claim AMA PRA Category 1 CreditsTM through the American Medical Association. To obtain an application form please contact the AMA at www.ama-assn.org.

Scientific Integrity and Disclosure of Conflicts of Interest

The American Academy of Ophthalmology is committed to ensuring that all continuing medical education (CME) information is based on the application of research findings and the implementation of evidence-based medicine. It seeks to promote balance, objectivity, and absence of commercial bias in its content. All persons in a position to control the content of this activity must disclose any and all financial interests. The Academy has mechanisms in place to resolve all conflicts of interest prior to an educational activity being delivered to the learners.

Control of Content

The American Academy of Ophthalmology considers presenting authors, not coauthors, to be in control of the educational content. It is Academy policy and traditional scientific publishing and professional courtesy to acknowledge all people contributing to the research, regardless of CME control of the live presentation of that content. This acknowledgement is made in a similar way in other Academy CME activities. Though they are acknowledged, coauthors do not have control of the CME content and their disclosures are not published or resolved.

2019 Glaucoma Subspecialty Day CME Credit

The American Academy of Ophthalmology is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide CME for physicians.

The Academy designates this live activity for a maximum of 7 AMA PRA Category 1 CreditsTM. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Attendance Verification for CME Reporting

Before processing your requests for CME credit, the Academy must verify your attendance at AAO 2019 and/or Subspecialty Day. Badges are no longer mailed before the meeting. Picking up your badge onsite will verify your attendance.

Badge Scanning and CME

Getting your badge scanned does not automatically grant CME credit. You still need to record your own educational activities.

NOTE: You should claim only the credit commensurate with the extent of your participation in the activity.

CME Credit Reporting

Onsite, report credits earned during Subspecialty Day and/or AAO 2019 at CME Credit Reporting kiosks located in South Lobby, West Lobby, and in the Academy Resource Center, West, Booth 7337.

Registrants whose attendance is verified at AAO 2019 receive an email on **Monday**, **Oct. 14** with a link and instructions on how to claim credit online. Attendees can use this link to report credits until **Wednesday**, **Oct. 30**.

Starting **Thursday**, Nov. 14, attendees can claim credits online through the Academy's CME web page, aao.org/ cme-central.

Academy Members

The CME credit reporting receipt is not a CME transcript. CME transcripts that include AAO 2019 credits entered at the American Academy of Ophthalmology's annual meeting will be available to Academy members through the Academy's CME web page beginning **Thursday**, **Nov. 14**.

The Academy transcript cannot list individual course attendance. It will list only the overall credits claimed for educational activities at Subspecialty Day and/or AAO 2019.

Nonmembers

The Academy provides nonmembers with verification of credits earned and reported for a single Academy-sponsored CME activity. To obtain a printed record of your credits, claim CME credits onsite at the CME Credit Reporting kiosks. Nonmembers choosing to claim credits online through the Academy's CME web page after Nov. 14 will have one opportunity to print a certificate.

Proof of Attendance

The following types of attendance verification are available during AAO 2019 and Subspecialty Day for those who need it for reimbursement or hospital privileges, or for nonmembers who need it to report CME credit:

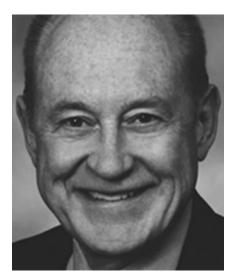
- CME credit reporting/proof-of-attendance letters
- Onsite registration receipt
- Instruction course and session verification

You must have obtained your proof of attendance at the CME Credit Reporting kiosks onsite located in South Lobby, West Lobby, and in the Academy Resource Center, West, Booth 7337.

The American Glaucoma Society (AGS) Subspecialty Day Lecture

A Lymphatic-like Pump Controls Aqueous Outflow: POAG Management and MIGS Implications

Saturday, Oct. 12, 2019 11:48 AM - 12:18 PM



Murray A Johnstone MD

Murray Johnstone MD is currently a clinical professor at the Eye Institute of the University of Washington Department of Ophthalmology. Many years spent interacting with patients suffering from glaucoma has made him very aware of the need to better understand the IOP problem in glaucoma. This experience led him to transition recently to a research-oriented environment.

Dr. Johnstone's laboratory research focuses on understanding how the aqueous outflow system normally regulates pressure, how pressure regulation becomes abnormal in glaucoma, and how normal function can be restored. The laboratory addresses the problem with microdissections; confocal, light, transmission, and scanning electron microscopy; perfusion studies; microvascular casting; and next-generation OCT imaging. In addition, clinical studies with new OCT imaging technologies provide direct observation and measurement of trabecular meshwork and collector channel motion in response to the ocular pulse. Translational studies identify the effects of viscoelastic injection into the Schlemm canal, the effects of pulsatile flow on the distal outflow pathways, and the effects of a micropulse laser on outflow structures.

Dr. Johnstone spent 36 years in a full-time clinical practice limited to glaucoma, during which time he has organized and participated in numerous national and international clinical and surgical courses and symposia. He has authored over 50 peer-reviewed publications and eight book chapters, has given 21 university invited lectures and 8 named national honorary lectures, and has been listed in Best Doctors in America continuously for many years.

Faculty



Victoria M Addis MD Philadelphia, PA



Lama A Al-Aswad MD MPH New York, NY



Edward M Barnett MD PhD Milwaukee, WI



Keith Barton MBBCh London, England



Sanjay G Asrani MD Durham, NC

No photo available

Michael R Banitt MD Mercer Island, WA



John P Berdahl MD Sioux Falls, SD



Lauren S Blieden MD Houston, TX



Donald L Budenz MD MPH Chapel Hill, NC



Claude F Burgoyne MD Portland, OR



Joseph Caprioli MD FACS Los Angeles, CA



Pratap Challa MD Durham, NC



Ta Chen Chang MD Miami, FL



Teresa C Chen MD Boston, MA



Vikas Chopra MD Santa Monica, CA



George A Cioffi MD New York, NY



Ian P Conner MD PhD Pittsburgh, PA



Gustavo De Moraes MD New York, NY



William J Flynn MD San Antonio, TX



Brian A Francis MD Pasadena, CA



David S Friedman MD MPH PhD Baltimore, MD



JoAnn A Giaconi MD Los Angeles, CA



Christopher A Girkin MD Birmingham, AL



Jeffrey L Goldberg MD PhD Palo Alto, CA



David S Greenfield MD Palm Beach Gardens, FL



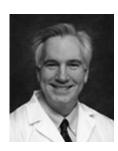
Davinder S Grover MD Dallas, TX



Paul J Harasymowycz MD Westmount, QC, Canada



Leon W Herndon Jr MD Durham, NC



Dale K Heuer MD Sierra Madre, CA



Alex Ansun Huang MD Pasadena, CA



Bret A Hughes MD Detroit, MI



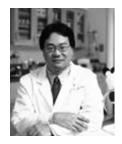
Murray A Johnstone MD Seattle, WA



Albert S Khouri MD Staten Island, NY



Christine L Larsen MD Eden Prairie, MN



Richard K Lee MD Miami, FL



Michele C Lim MD Sacramento, CA



John T Lind MD Saint Louis, MO



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Mildred M G Olivier MD Hoffman Estates, IL



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John D Shepherd MD Omaha, NE



Oluwatosin U Smith MD Colleyville, TX



Thasarat S Vajaranant MD Chicago, IL



Scott M Walsman MD Jersey City, NJ

Ask a Question and Respond to Polls Live During the Meeting Using the Mobile Meeting Guide

To submit an answer to a poll or ask the moderator a question during the meeting, follow the directions below.

- Access at www.aao.org/mobile
- Select Program, Handouts & Evals
- Filter by Meeting Glaucoma Meeting
- Select Current Session
- Select "Interact with this session (live)" link to open a new window
- Choose "Answer Poll" or "Ask a Question"



Glaucoma 2019: Crossing the Golden Gate to Exceptional Glaucoma Care

In conjunction with the American Glaucoma Society

SATURDAY, OCT. 12, 2019

7:00 AM	CONTINENTAL BREAKFAST	
8:00 AM	Welcome and Introductions	JoAnn A Giaconi MD*
8:01 AM	American Glaucoma Society Introduction	Dale K Heuer MD*
8:02 AM	American Glaucoma Society Cares	William J Flynn MD
8:05 AM	Announcements	Eydie G Miller-Ellis MD*

Section I: Escape From OCT-a-Traz—Knowing the Ins and Outs of Imaging

Moderators: Sanjay G Asrani MD* and Teresa C Chen MD*		
RNFL Imaging With Different Devices	Joel S Schuman MD*	1
Optic Nerve Imaging With Different Devices	Claude F Burgoyne MD*	6
Macular Imaging With Different Devices	Jullia A Rosdahl MD PhD*	8
Audience Q&A		
OCT Artifacts and Pitfalls	Richard K Lee MD	9
Determining OCT Progression	Felipe A Medeiros MD*	10
What to Do When the HVF and OCT Don't Match	David S Greenfield MD*	12
Audience Q&A		
	RNFL Imaging With Different Devices Optic Nerve Imaging With Different Devices Macular Imaging With Different Devices Audience Q&A OCT Artifacts and Pitfalls Determining OCT Progression What to Do When the HVF and OCT Don't Match	RNFL Imaging With Different DevicesJoel S Schuman MD*Optic Nerve Imaging With Different DevicesClaude F Burgoyne MD*Macular Imaging With Different DevicesJullia A Rosdahl MD PhD*Audience Q&AOCT Artifacts and PitfallsRichard K Lee MDDetermining OCT ProgressionFelipe A Medeiros MD*What to Do When the HVF and OCT Don't MatchDavid S Greenfield MD*

Section II: The Great Quake-Everyday Topics Rethought

Moderators: Joseph Caprioli MD FACS* and Albert S Khouri MD*

9:10 AM	Meds After MIGS	Alex Ansun Huang MD*	14
9:17 AM	Moving Glaucoma Neuroprotection Into Clinical Trials	Jeffrey L Goldberg MD PhD*	16
9:24 AM	Is There a Role for Marijuana?	Bret A Hughes MD*	17
9:31 AM	Discussion		
9:40 AM	How to Handle Dissonance Between OCT and Gonioscopy	David S Friedman MD MPH PhD*	19
9:47 AM	Does the Use of Third, Fourth, and Fifth Drugs Delay Surgery? How Much Medication Is Too Much?*	Janet B Serle MD*	20
9:54 AM	How to Protect Glaucoma Drainage Devices From Erosion?	Victoria M Addis MD	22
10:01 AM	Discussion		
10:10 AM	There Is Something You Can Do	John D Shepherd MD	23
10:14 AM	REFRESHMENT BREAK and AAO 2019 EXHIBITS		

* Indicates that the presenter has financial interest. No asterisk indicates that the presenter has no financial interest.

Section III: Casting Your Net for the Best Glaucoma Surgery at Fisherman's Wharf

Moderators: Lama A Al-Aswad MD MPH* and Davinder S Grover MD*

Panelists: George A Cioffi MD, Joseph F Panarelli MD*, Pradeep Y Ramulu MD PhD*, Oluwatosin U Smith MD*, and Scott M Walsman MD*

10:44 AM	Case Presentation 1	Lama A Al-Aswad MD MPH*	24
10:54 AM	Case Presentation 2	Davinder S Grover MD*	24
11:04 AM	Case Presentation 3	Lama A Al-Aswad MD MPH*	24
11:15 AM	Case Presentation 4	Davinder S Grover MD*	24
11:26 AM	AGS-IRIS® Registry Project	Mildred M G Olivier MD*	25
11:31 AM	Ocular Hypertension Treatment: 20-Year Incidence and Severity of Primary Open-Angle Glaucoma (POAG)	Richard K Parrish II MD*	26
11:41 AM	Are You AT the Table or ON the Menu?	Donald L Budenz MD*	27

The American Glaucoma Society Subspecialty Day Lecture

11:46 AM	Introduction of the Lecturer	Dale K Heuer MD*	
11:48 AM	A Lymphatic-like Pump Controls Aqueous Outflow: POAG Management and MIGS Implications	Murray A Johnstone MD	29
12:18 PM	Presentation of the Award	Dale K Heuer MD*	
12:19 PM	LUNCH and AAO 2019 EXHIBITS		

Section IV: Cable Car Next Stop—Open-Angle Glaucoma With Low Pressures

	Moderators: Edward M Barnett MD PhD and Christine L Larsen MD'	÷	
1:29 PM	Normal-Pressure Glaucoma Masqueraders	Ahmara V Ross MD PhD	31
1:36 PM	Addressing the Normal-Pressure Glaucoma Patient With Systemic Hypotension	Jody R Piltz MD*	32
1:43 PM	Is Testing for Sleep Apnea Indicated?	Christopher A Girkin MD	35
1:50 PM	A Disc Hemorrhage: What Does It Mean? What Should I Do?	Gustavo De Moraes MD*	36
1:57 PM	Considerations for Medical Management	Ki Ho Park MD PhD*	37
2:04 PM	Surgery for Normal-Pressure Glaucoma: Is There a Role for Minimally Invasive Glaucoma Surgery Before Filtering Surgery?	Leonard K Seibold MD*	39
2:11 PM	Discussion		

Section V: Lombard Street—Winding Your Way Through Glaucoma and Anterior Segment Disease From Birth Until Death

	Moderators: Ian P Conner MD PhD* and John T Lind MD*		
2:29 PM	Congenital Anomalies Presenting With Anterior Segment and Glaucoma Findings	Ta Chen Chang MD	40
2:36 PM	Iridocorneal Endothelial Syndrome (ICE)	Lauren S Blieden MD	41
2:43 PM	Medication and Laser Therapy in Glaucoma Patients With Corneal Disease	Keith Barton MB BCh*	42
2:50 PM	Minimally Invasive Glaucoma Surgery Consideration in Patients With Diseases of the Cornea	John P Berdahl MD*	44
2:57 PM	Why Does My Glaucoma Surgery Make the Cornea Fail, and Why Does My Cornea Surgery Make Glaucoma Worse?	Michael R Banitt MD	45

* Indicates that the presenter has financial interest. No asterisk indicates that the presenter has no financial interest.

3:04 PM	Measuring IOP After Cornea Surgery	Thasarat S Vajaranant MD	46
3:11 PM	Discussion		
3:29 PM	REFRESHMENT BREAK and AAO 2019 EXHIBITS		
Section VI:	Pacific Heights—Reaching 20/20 (or Near That) With Catara	act Surgery and Glaucoma	
	Moderators: Vikas Chopra MD* and Leon W Herndon Jr MD*		
	Virtual Moderator: Douglas J Rhee MD*		
3:59 PM	Cataract Surgery Pearls in Eyes With Pre-existing Trab or Tube Shunt	Thomas W Samuelson MD* Constance O Okeke MD*	48
4:14 PM	Cataract Surgery Combined With Minimally Invasive Glaucoma Surgery in Eyes With Controlled Glaucoma on Meds and History of Prior Incisional Glaucoma Surgery	Paul J Harasymowycz MD* Sameh Mosaed MD*	50
4:29 PM	Cataract Surgery Concerns in Short Eyes With Narrow Angles or Angle Closure	Michele C Lim MD* Brian A Francis MD*	51
4:44 PM	Managing Intraoperative Cataract Surgery Complications in Eyes With Glaucoma	Shakeel R Shareef MD Pratap Challa MD*	53
4:59 PM	Case Discussions With Panel and Audience Q&A		
5:09 PM	Closing Remarks	JoAnn A Giaconi MD* Eydie G Miller-Ellis MD*	
5:11 PM	Adjourn		

RNFL Imaging With Different Devices

Joel S Schuman MD

Acknowledgment: Portions of this outline are adapted from Schuman JS, Puliafito CA, Fujimoto JG, Duker JS, Ishikawa H, Wollstein G (editors). Everyday OCT: A Handbook for Clinicians and Technicians, 2nd ed. Thorofare, NJ: Slack Incorporated; 2017.

- I. Commercial OCT Devices
 - A. Spectralis (Heidelberg Engineering; Heidelberg, Germany)
 - B. RTVue, Avanti (Optovue; CA), introduced in USA in 2006
 - C. Cirrus, Plex Elite (Carl Zeiss Meditec, Inc.; Dublin, CA), introduced in USA in 2007
- II. Cirrus (Carl Zeiss Meditec, Inc.; Dublin, CA), introduced in USA in 2007
 - A. 3-D (volumetric) scans
 - 1. Macular cube, 512x128
 - 2. Macular cube, 200x200
 - 3. Optic disc cube, 200x200
 - B. Raster scans
 - 1. HD 1 line 100x
 - 2. HD 5-line raster and HD 1 line 20x
 - 3. 5-line raster
 - 4. HD 21 line
 - 5. HD cross
 - 6. HD radial
 - C. Anterior segment scans: anterior chamber
 - 1. Wide-field single HD B-scan (20 frames averaged) with 15.5-mm scan length (when oriented horizontally) at a scan depth of 5.8 mm
 - 2. Anterior segment cube, 512x128
 - 3. Anterior segment 5-line raster
 - 4. HD angle
 - 5. HD cornea
 - 6. Wide angle to angle
 - D. FastTrac
 - E. Macular analysis
 - 1. Macular thickness analysis
 - 2. Macular change analysis
 - 3. Macular thickness OU analysis

- 4. Advanced retinal pigment epithelium (RPE) analysis
- 5. Ganglion cell OU analysis
- F. Optic disc analysis
 - 1. Optic nerve head (ONH) and retinal nerve fiber layer (RNFL) OU analysis
 - 2. RNFL thickness analysis
 - 3. Guided progression analysis
- G. Comprehensive analysis
 - 1. Single eye summary
 - 2. PanoMap
- H. Advanced Visualization: Provides a powerful exploratory tool for any cube scan types
- I. AngioPlex
 - 1. Visualization of retinal vasculature, depth resolved
 - 2. Quantitation of vascular density
- J. PlexElite
 - 1. Swept source OCT
 - 2. 100,000 A-scans/second
 - 3. Widefield OCT scanning is possible.
 - 4. Both structural and vascular measurements can be performed.
 - 5. Excellent visualization of choroidal structures due to longer scanning wavelength
- III. Optovue
 - A. 26,000 A-scans/second systems
 - B. iScan is designed as a cost-effective, fully automated OCT imaging of the basic retina, nerve fiber, and anterior chamber structures with minimal operator input.
 - C. iVue is designed for cost-effective ease of use in those same areas in a single practice or multiple offices. The iStand can be used to hold the iVue for use on supine patients.
 - D. RTVue was the first commercially available spectral domain OCT system in the United States (2006).
 - E. RTVue XR Avanti utilizes full-time 70k spectral domain imaging for all scans.

F. Line scan collection

The line scan is a single straight line that can be adjusted in position, angle, and length from 2 mm to 12 mm and can be oversampled (scanned over and over) between 1 and 500 times. The collection of obtained images are aligned and averaged. This provides extremely high-resolution scan detail and depth resolution.

- 1. HD line scan: Like the line scan, this can be adjusted in position, angle, length, and over-samples, but it is optimized to display both the vitreous and retina.
- 2. Cross line scan: This is a pair of lines that are offset by 90 degrees, and like the line scan, it is fully adjustable.
- 3. Raster scan: This is a series of 17 parallel line scans that can be fully adjusted for position, length, spacing, angle, and oversamples (averaging).
- 4. Grid scan: Five lines in 2 groups offset by 90 degrees
- 5. Radial scan: 16 radial oversampled lines offset at 22.5 degree intervals
- 6. Cornea line and cross line scans: Similar to the retinal scans but optimized for the corneal visualization and measurements
- 7. Corneal angle scan: Placed over the angle at any location, this can be used to measure the angle opening in degrees or area (mm).
- G. Pattern scan collection

The advantage of pattern scans is that they allow for quick representative sampling of large areas for mapping analysis.

- 1. Retina map: A fast central retinal thickness map covering 5x5 mm centered over the fovea
- 2. GCC map: A fast thickness map of the ganglion cell layer thickness covering 7x7 mm near the fovea
- 3. ONH map: A fast RNFL thickness map covering the 5 mm around and through the optic nerve head
- 4. Corneal pachymetry map: A fast thickness map covering 6 mm centered on the central cornea (pupil)
- 5. Epithelial/stromal map: A fast thickness map covering 9 mm centered on the pupil
- 6. iWellness scan: Does a map of the central retina and a ganglion cell analysis used to evaluate for retinal and nerve fiber issues (This scan is available as an option on the iScan and iVue only.)

- H. The RTVue XR Avanti utilizes full-time 70k spectral domain imaging for all scans.
- I. Volume scan collection

Volume scans provide dense coverage of areas that can be reconstructed into 3-dimensional images.

- 1. Retina 3D scan: This is an adjustable 3-D scan that can be positioned on the retina over pathology.
- 2. Widefield retina scan: A 12x9-mm 3-D scan covering the entire macular and optic nerve areas
- 3. Optic nerve 3D scan: A 6x6-mm scan covering the area around the optic nerve
- J. Angio scan collection

These exciting new scans allow for completely noninvasive display of perfusion within the back of the eye. They show the pattern of blood movement within the blood vessels (angiography) and show areas where there is nonperfusion.

- 1. Retina angiography scan: Available in 2-, 3-, 6-, or 8-mm area centered over the fovea that shows the movement of the blood within the various retina and choroidal layers
- 2. Optic nerve angiography scan: Available in 3- or 4.5-mm area centered over the optic nerve that shows the movement of the blood within the vasculature of various retina, optic nerve, and choroidal layers
- IV. Spectralis OCT (Heidelberg Engineering; Heidelberg, Germany)
 - A. Multimodality imaging

The HRA+OCT is a multimodality imaging device. It combines spectral domain OCT with the following fundus imaging modalities:

- 1. Blue reflectance imaging (BR)
- 2. Infrared reflectance imaging (IR)
- 3. BluePeak blue laser autofluorescence imaging (BAF)
- 4. Fluorescein angiography (FA)
- 5. Indocyanine green angiography (ICGA)
- 6. MultiColor imaging (option)
- B. TruTrack Active Eye Tracking
- C. Image registration
- D. Heidelberg noise reduction
- E. AutoRescan
- F. Fovea-to-Disc (FoDi) Alignment Technology
- G. Anatomic Positioning System (APS)
- H. Enhanced depth imaging
- I. ART mean

V. Topcon

- A. Triton
 - 1. Posterior scans
 - 2. Radial scan: The radial scans are available in 6.0-mm and 9.0-mm diameters with 12 lines at a resolution of 1024 per line, oversampled (averaged) up to 16 times.
 - 3. Line scan: The line scan can be placed horizontally, vertically, or in any orientation and is useful for obtaining highly detailed images of a particular area of interest. The line scan can be 12.0 mm, 9.0 mm, or 6.0 mm in length with a resolution of 1024. The Triton is capable of oversampling the line scan up to 128 times.
 - 4. 5-line cross scan: 10 horizontal and vertical lines arranged in a cross pattern (5 horizontal and 5 vertical). Available in 9.0-mm or 6.0-mm lengths and oversampled up to 16 times per line.
 - 5. 3D wide (H) scan: A 12.0-mm by 9.0-mm volume scan with a resolution of 512x256, capable of acquiring information about the macula and optic disc in a single scan. A Normative or Reference database comparison is available for 3D wide scans.
 - 6. 3D macula scan: Has a resolution of 512x256 performed over a 7.0x7.0-mm scan area. It can also be performed over a 3.0x3.0-mm scan area to increase the sampling density. This scan also allows comparison with the Reference Database.
 - 3D disc scan: Uses a resolution of 512x256 performed in a 6.0x6.0-mm scan area. For additional detail of the optic nerve, a 3.0x3.0-mm scan option is also available. This scan also provides a comparison to the Reference Database.
 - 8. Combination scan: line scan, 5-line cross scan, and radial scan

The combination scans are a series of scans that combine the 3D Wide (H) scan with various other high-resolution line scans (line, 5-line cross, and radial). This is particularly useful for obtaining the necessary volume information to display the grid data while maintaining high-resolution B scans for detailed views of the pathology. This scan also allows comparison with the Reference Database.

- 9. FGA mode
 - a. "FGA" stands for Fundus Guided Acquisition. This mode allows you to select an area of interest on the fundus image and position the OCT scan directly through the area of interest.
 - b. The instrument then uses its alignment and tracking abilities to scan the exact area specified.

10. Anterior scans

All anterior Triton scans are captured using an additional lens that attaches to the front lens of the device. This lens improves image quality for the anterior scans by making the optics telecentric, greatly improving the signal and detail obtained.

- a. Line scan: The line scan size can be 3.0 mm, 6.0 mm, or 16.0 mm in length and oriented in any direction. The maximum oversampling rate is 64.
- b. Radial scan: 12 lines evenly spaced in a radial pattern. You can choose between 6.0-mm and 16.0-mm lengths for the line scans. They can be oversampled up to 4 times.
- c. 3D anterior (3.0x3.0 mm): This anterior 3D scan is meant to help visualize the cornea in 3 dimensions. It is available only in 3.0x3.0 mm.
- B. Maestro
 - 1. Posterior scans
 - a. Line scan: The line scan can be placed horizontally, vertically, or at any orientation and is useful for obtaining highly detailed images of a particular area of interest. The line scan can be 9.0 mm or 6.0 mm in length with a resolution of 1024. The Maestro is capable of oversampling the line scan up to 50 times.
 - b. 5-line cross scan: The 5 line cross scan consists of 10 lines arranged in a cross pattern (5 horizontal and 5 vertical). Available in 9.0mm or 6.0mm lengths and oversampled up to 16 times per line.
 - c. 3D wide scan: The 3D wide scan is a 12.0mm by 9.0-mm volume scan with a resolution of 512x128, capable of acquiring information about the macula and optic disc in a single scan. This scan provides a comparison with the Reference Database.
 - d. 3D macula scan: The 3D macula scan has a resolution of 512x128 performed in a 6.0x6.0-mm scan area. This scan can be compared with the Reference Database.
 - B disc scan: The 3D disc scan also uses a resolution of 512x128 performed in a 6.0x6.0-mm scan area. This scan also allows comparison with the Reference Database.
 - 2. Anterior scans

Anterior scans on the Maestro do not use the additional lens. Instead there is an attachment to the headrest that increases the distance of the OCT from the eye, essentially changing the focal plane from the back of the eye to the front.

a. Radial scan: Radial scans are 12 lines evenly spaced in a radial pattern, 6.0 mm in length. They can be oversampled up to 4 times for

higher contrast/detail. Anterior radial line scans are typically used for assessment of the cornea including thickness determination.

- b. Line scan: The line scan size can be 3.0 mm and 6.0 mm in length and oriented in any direction. The maximum oversampling rate is 50. This line scan is typically used to visualize the angle of the cornea at the iris.
- C. Image acquisition procedure

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The following steps are general image acquisition steps for both the Maestro and the Triton.

- 1. Make sure the patient is seated comfortably in front of the OCT. Adjust the table height until they can comfortably place their chin in the chinrest with their forehead against the forehead rest.
- 2. Have them sit back and relax while you enter the patient information into the PC. For new patients, click on the Register Patient button on the PC and enter the appropriate patient demographics. For pre-existing patients, pull up their name from the database list and their information will be revealed.
- 3. On the instrument touchscreen, select the eye and/or scan pattern to be taken.
- 4. Adjust the chinrest height until the patient's eyes are level with the canthus mark on the side of the headrest. For the Maestro, the operator needs to be sure the pupil is in the center of the view window (external view of pupil). If it is not, the operator should press on the center of the pupil and the device will align itself to the center of the pupil.
- 5. On the Maestro touch screen, press Capture Start to initiate the automated capture process; this can be overridden to operate the instrument manually via the "digital joystick" where desired. On the Triton, simply drive the instrument using the traditional joystick.
- 6. It is important to coach the patient to look at the fixation target and to blink normally until instructed otherwise. The operator should ask the patient to blink once prior to capturing the actual scan. For the Maestro, this should be at the point when the countdown timer has reached 2, with the scan beginning at 0. After the scan the operator should tell the patient to relax. These small coaching tips are important and can help maximize image quality.
- 7. Review the scan for quality (see below). If the scan is not of good quality, it should be retaken while the patient is still in the chair.

VI. Canon OCT-HS100

The Canon OCT-HS100 is the first fully automated spectral domain OCT.

- A. Scan modes: Canon OCT-HS100 provides a variety of scanning types. Here each scan type is briefly described for its application and characteristics.
 - 1. Posterior segment scans:

The following scans can be performed at any location on the retina visible through the scanner, but they are usually used for evaluating macular or disc structures. All scan types except for Disc 3D provides B-scan images with 1024 A-scans, which is the highest transverse sampling density using Canon OCT-HS100. Scan length can be set to 3-10 mm.

- a. 3D scans
 - i. Macula 3D: This mode is used for evaluating macular diseases quantitatively, such as AMD, diabetic macular edema, and macular edema associated with retinal vein occlusion. Scan is performed centering on the macula, for an evaluation of the retina and the choroid. Scan size: 10x10 mm.
 - ii. Glaucoma 3D: This mode is used for evaluating glaucoma in the macular area. The main scan direction is vertical. Normal retinal layers should be symmetric with respect to a horizontal line through the center of the fovea. Early stage glaucoma can be diagnosed based on the asymmetry of inner retinal layers on vertical B-scan. Scan is performed centering on the macula, for an evaluation of the nerve fiber layer (NFL) + ganglion cell layer (GCL) + inner plexiform layer (IPL). Scan size: 10x10 mm
 - iii. Disc 3D: This mode is used for evaluating morphology of the optic disc. Scan size: 6x6 mm
 - iv. Custom 3D: This mode is for general usage. This mode does not support analysis reports for any specific diseases. Scan size: 3x3 to 10x10 mm.
- b. Raster scan
- c. Cross scan: This mode produces highestquality images by multiple B-scan averaging (up to 50 B-scan images), composed of 1 horizontal and vertical line scans. Scan size: 3x3 to 10x10 mm.

- d. Multi cross: This mode is suitable for daily clinical observation, composed of 5 horizontal and vertical line scans. Averaging can be up to 10 B-scans. Scan size: 3x3 to 10x10 mm. If the patient's fixation is unstable, this mode may be suitable. Intervals of line width (vertical and horizontal) can be changed independently.
- e. Radial: This mode is used for daily clinical observation, composed of 12 radial scans (each angle: 15 degrees). Scan size: 3 mm to 10 mm (phi). Averaging can be up to 10 B-scans.
- 2. Anterior segment
 - a. Anterior segment scans: This mode is suitable for daily clinical observation, composed of 12 radial scan lines (each angle: 15 degrees). Scan size: 6 mm (phi). Corneal thickness analysis is possible using this mode.
 - b. Anterior cross: This mode produces high-resolution cross scan images, for evaluating cornea and angle. Averaging can be up to 50 B-scan images. Manual measurement functions such as the depth of anterior chamber and the width of angle are supported in this mode. Scan size: 3x3 to 10x10 mm.
 - c. Anterior 3D: This mode is used for 3D volume scan in the anterior segment. Scan size: 6x6 mm length at cornea.
- B. Analysis reports
 - 1. Macula 3D for macula disease:

This mode is a macula analysis report for diagnosing and evaluating macula diseases. The report includes information about retinal thickness in each sector, significance map, thickness map from IS/OS (ellipsoid zone) to RPE, and morphology of RPE surface.

2. Macula 3D for glaucoma (Glaucoma 3D):

This is a macula analysis report for glaucoma diagnosis. The report focuses on the thickness of NFL, NFL+GCL+IPL, and GCL+IPL, comparison between superior sector and inferior sectors.

3. Disc analysis for glaucoma:

This is a disc analysis report for glaucoma diagnosis. This report focuses on the thickness of NFL and disc measurements such as cup volume.

4. Corneal analysis for corneal disease:

This is a corneal analysis report for evaluating corneal diseases. This report includes Report Form. Canon OCT-HS100 has 4 report forms: Single, Both eyes, Comparison, and Progression.

5. Data management:

Canon OCT-HS100 manages the patient data with database. If you prepare the hard disk and a server, you can take the backup by export. If you save it to a server, it is possible for you to read the data from multiple viewers.

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Optic Nerve Imaging With Different Devices

Claude F Burgoyne MD

I. Disclosures

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- A. Unrestricted research support from and an unpaid consultant to Heidelberg Engineering: occasional travel support, no honorarium, no patents and no personal income related to this consultancy
- B. NIH funded to build OCT strategies for phenotyping the optic nerve head (ONH) peripapillary retinal and macular tissues of healthy and glaucomatous human eyes
- II. Definitions
 - A. In this talk I will focus on the ONH tissues.
 - B. We define the ONH to include the tissues that are contained within the scleral canal and those immediately adjacent to it (ie, the peripapillary sclera, choroid, and retina as well as the immediate retrolaminar optic nerve).
- III. Context/Working Hypotheses/Biases

OCT anatomy is to the clinical "disc" examination what the introduction of chest x-rays was to the clinical examination of the lungs—it is newly accessible clinical anatomy that requires effort to understand, assimilate, and integrate into your own clinical examination of these tissues.

- A. OCT anatomy must be learned.
- B. OCT anatomy (not just a 1-page printout of its quantification) should be integrated into your clinical examination.
- C. While OCT anatomy underlies and explains clinical disc appearance, it is often clinically invisible.
- D. OCT anatomy is 3-D; its parameterization should not mimic the 2-D clinical disc examination.
- E. Understanding OCT anatomy is part of the professional responsibility of ordering and interpreting an OCT scan.
- IV. Current Status of OCT ONH Image Acquisition and Parameterization
 - A. Image acquisition
 - 1. Radial, horizontal, or vertical B-scans
 - 2. High-resolution isotropic grid scans
 - 3. A-scans acquired relative to ONH and retinal anatomy
 - a. Axis between the center of the Bruch membrane opening (BMO) and the center of the fovea (the FoBMO axis)

- b. Consistent in each eye on each imaging day if accurate eye tracking algorithm employed
- c. Consistent among all human eyes
- d. Challenging where BMO and macula anatomy is difficult to identify (ie, in highly myopic eyes)
- 4. A-scans acquired relative to the acquired image frame without regard to ONH and retinal anatomy
 - a. Different in the same eye on different days due to inter-imaging session differences in head tilt and cyclotorsion
 - b. Different among all human eyes due to differences in head tilt and cyclotorsion
- B. Parameterization/automated segmentation, FDA Normative Data Bases: available now
 - 1. 3-D: BMO area, BMO minimum rim width (MRW) and minimum rim area (MRA) parameters
 - 2. 2-D: conventional "disc" parameters (cup-todisc ratio, cup volume, cup depth, rim area, disc size)
- C. Regionalization
 - Data regionalized into Garway-Heath sectors, 30° (clock-hour) sectors or quadrants
 - 2. Data regionalized relative to ONH /retinal anatomy to employ anatomic definitions of superior, inferior, nasal, temporal
 - The FoBMO axis defines the nasal-temporal axis.
 - b. The superior-inferior axis passes through the center of BMO perpendicular to the nasal-temporal axis.
 - c. Sectoral data are consistent in each eye on each imaging day.
 - d. Sectoral data are consistent among all human eyes.
 - 3. Data regionalized relative to the acquired image frame without regard to ONH and retinal anatomy
 - a. Sectors are different in the same eye on different days due to inter-imaging session differences in head tilt/cyclotorsion.
 - b. Sectors are different among all human eyes due to differences in head tilt/cyclotorsion.

- V. The Future: What's in the Literature but Is Not Yet Automatically Segmented
 - A. Laminar depth, curvature, and thickness
 - B. Peripapillary choroidal thickness
 - C. Anterior scleral canal opening (ASCO) size, shape, and offset from BMO
 - D. Neural canal direction, obliqueness, and minimum cross-sectional area (NCMCA)
 - E. ASCO tilt and rotation relative to BMO
 - F. Peripapillary scleral bowing
- VI. Current Status Update
 - A. Disease and progression detection
 - B. ONH OCT angiography
- VII. Integration, Quantification, and Comparison

Clinically intuitive integration of OCT ONH / RNFLT/macula anatomy and angiography, its quantification and its comparison to normative databases is what the field requires, and this is what the field must fight to achieve.

- A. Presenting OCT anatomy and its quantification relative to normative databases in a clinically intuitive manner should be the responsibility of each instrument company.
- B. Presenting OCT anatomy and its quantification relative to normative databases in a clinically intuitive manner within an EMR system should be the responsibility of each EMR provider.
- C. Neither (A) nor (B) will happen if clinicians do not demand this from instrument manufacturers and EMR providers.

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Macular Imaging With Different Devices

Jullia A Rosdahl MD PhD

- I. Illustrative Case Example
- II. Background on the Use of Macular OCT for Glaucoma
 - A. Review of retinal ganglion cell anatomy
 - B. Structure-function correlation of macular OCT and visual field
 - C. Macular OCT for clinical glaucoma care
- III. Macular Protocols on Common Devices
 - A. Total retinal thickness vs. segmentation of layers
 - B. Diagnostic use and monitoring of progression
 - C. Common devices with protocols
- IV. Examples of Glaucomatous Defects on Macular OCT, on Different Devices
 - A. Arcuate defect
 - B. Paracentral defect
 - C. Advanced glaucoma
- V. Examples of Nonglaucomatous Defects, on Difference Devices
 - A. Epiretinal membranes
 - B. Retinal vascular disease
 - C. CNS degenerations

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OCT Artifacts and Pitfalls

Richard K Lee MD

NOTES

Determining OCT Progression

Felipe A Medeiros MD

Assessing rates of progression is an indispensable step in glaucoma management as it provides a means to identify rapidly progressing patients who are at high risk of visual disability and who may require escalation in treatment. However, although OCT has been widely adopted in clinical practice, there is still uncertainty regarding how OCT should be best used to detect glaucoma progression.

The ideal parameter for measuring glaucoma progression should be highly reproducible and useful at all stages of disease. Although the majority of studies have focused on OCT measurements of rates of change using the circumpapillary retinal nerve fiber layer (cpRNFL) thickness, recent studies have shown that additional information can be obtained by examining changes in other parameters and areas-for example, by examining the topography of RNFL loss across a 6x6 mm² optic disc cube scan RNFL map. OCT devices also provide the ability to quantify changes to the macula using measurements such as ganglion cell-inner plexiform layer (GC-IPL) and ganglion cell complex (GCC) thickness. Macular measures are of special interest because of the density of retinal ganglion cells located in this region. Some OCT devices also include the ability to obtain optic nerve head metrics, such as Bruch membrane openingminimum rim width and Bruch membrane opening-minimum rim area (BMO-MRW, BMO-MRA), which use the BMO as an anatomical point of reference landmark for measurements.

Several studies have investigated the role of cpRNFL, optic disc topography, and macular measurements for assessing glaucoma progression. However, it has been difficult to determine whether one parameter is better than another due to the lack of a gold standard, and although all glaucomatous changes reflect loss of retinal ganglion cells, there is still poor understanding of the temporal relationship between changes to the optic nerve head, RNFL, and macula. One might suppose that measurements taken relative to BMO would perform better than conventional structural measures in detecting glaucoma progression, given the relatively stability of the BMO as a point of reference for repeat scans. However, recent studies have raised concerns related to lower longitudinal signal-to-noise ratio compared to cpRNFL and possible changes in the location of the BMO over time.

Regardless of which parameter might be best, there is now a large body of evidence that progressive changes on OCT are clinically relevant. Faster rates of cpRNFL loss on OCT are associated with higher risk of future development of visual field defects, faster decline in quality of life, and worse performance on driving simulation, with information from OCT offering additional predictive value compared to information from visual field testing alone.

Importantly, glaucoma progression must be differentiated from normal age-related changes to cpRNFL and macula. A suggestion has been made that trend-based analysis of change should at least involve testing the statistical significance of its change relative to the mean estimate of age-related changes. This would be analogous to evaluating visual field progression using mean deviation (MD) instead of mean sensitivity (with the former being an age-adjusted parameter) and could be described as an RNFL "mean deviation" trend analysis. Although OCT has a valuable role in assessing glaucoma progression, visual field testing remains the primary method of assessing glaucomatous damage, and some patients may have functional changes in the absence of detectable structural changes. The ability to detect progression by perimetry vs. OCT is significantly influenced by the stage of disease, with eyes with less severe disease at baseline having a higher chance of being detected as progressing by OCT but not SAP, and eyes with more advanced disease having a higher chance of being detected as progressing by SAP but not OCT. This raises the question of how OCT should be best used to complement assessment of visual function. Several approaches for combining structure and function have been described, including subjective ones, which may be limited by varying degrees of examiner expertise, as well as objective measurements based on Bayesian analysis and combined structurefunction indices.

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What to Do When the HVF and OCT Don't Match

David S Greenfield MD

Glaucoma is a multifactorial optic neuropathy characterized by progressive neurodegeneration of retinal ganglion cells (RGCs) and their axons, resulting in retinal nerve fiber layer (RNFL) attenuation, a specific pattern of damage to the optic nerve head, and visual field loss. Since glaucoma leads to irreversible loss of vision, the early identification of disease progression is essential to clinical management.

Two fundamental components of monitoring glaucoma progression include documentation and longitudinal monitoring of relevant structural and functional measures. It is widely recognized that both structure and function are useful for detecting glaucoma progression, given discordance in the timing of detecting longitudinal changes in the optic nerve and visual field (VF).¹⁻⁷ The relationship between signal-to-noise,⁸ stage of glaucomatous damage,⁹ and the technique and region of VF studied have a significant impact on the comparison of structure and function for the progression of glaucoma.³

VF assessment using standard automated perimetry (SAP) in glaucoma is an established method of monitoring functional disease progression. It is statistically correlated with visionrelated quality of life,¹⁰ it represents the reference standard used in major landmark glaucoma treatment trials, and algorithms exist that enable clinicians to measure change over time. As with all technologies, there are limitations. High test-retest variability exists, necessitating repeated exams to confirm suspect progression, and poor sensitivity exists in eyes with early glaucoma as substantial RGC loss can occur before perimetric loss becomes manifest.¹¹

Imaging technologies have been developed that are capable of quantifying early glaucomatous damage at the micron level using structural measures. Spectral domain OCT (SD-OCT) has been widely adopted in clinical practice, provides quantitative assessments of the optic nerve head, RNFL, and macula, and can detect longitudinal structural loss in glaucomatous eyes over time.¹²⁻¹³ As with perimetry, computerized algorithms exist to facilitate longitudinal change detection using repeated measures and quantify rates of progression.¹⁴

Although agreement between VF and OCT measures are common in glaucomatous eyes, there are many circumstances in which they are not well correlated. A recent study by Nguyen and colleagues¹⁵ compared progression with VF and OCT using Guided Progression Analysis (Carl Zeiss Meditec; Dublin, CA) in 147 eyes monitored over a mean 69 months of follow-up. Approximately 25% showed RNFL progression, 24% of eyes showed GC-IPL progression, and 14% of eyes showed VF progression. However, progression by all 3 methods was noted in only 7.0%.

Various mechanisms contribute to structure-function discordance in glaucomatous eyes. Poor quality data such as unreliable VF performance or imaging artifact, which may occur in 20%-30% of eyes,¹⁶⁻¹⁷ often contributes to poor agreement. Glaucoma severity stage will impact the sensitivity of testing strategies, and there is a nonlinear relationship between visual function and estimates of RGC counts.¹⁸ For example, SAP has poor sensitivity in eyes with early glaucoma in which substantial RGC loss may occur prior to detectable changes in visual function.^{1,19-20} Conversely, detecting change in eyes with advanced glaucoma is difficult with OCT, given increased measurement variability, reduced signal-to-noise ratio, potential for algorithm failure, and a measurement floor effect²¹⁻²² below which further loss in visual function and structure no longer can be detected. Lastly, change analysis methods differ. Regions of interest selected for measurements using OCT and VF are not in direct correspondence,²³ and testing strategies may differ based upon linear or logarithmic scaling.

There are several important points for clinicians to consider during serial glaucoma monitoring when assessing disagreement between structure and function. Repeat testing is essential to confirm suspected change, particularly in eyes with poor quality imaging or unreliable SAP. Concordance between structure and function may improve over time; therefore, observation and more frequent testing may be warranted, particularly in eyes with controlled IOP. Clinicians should incorporate change detection algorithms to measure change and assess rates of progression. To demonstrate localized changes, OCT deviation maps are more useful than summary parameters that may be insensitive. Incorporating adjunctive testing methodologies such as central VF testing24-25 and macular RGC thickness15,26-27 assessments is useful in predicting and identifying progression and may help to resolve disagreement among discordant test results. Agreement between structural tests, such as macular RGC and parapapillary RNFL progression, has been shown to be mutually predictive of VF progression and to facilitate detection of disease deterioration.²⁸ Finally, optic disc hemorrhage²⁹ has been consistently shown in clinical trials and longitudinal studies to be a strong predictor of progression. Disc hemorrhages should be documented with fundus photography and considered as a biomarker for progression when assessing disagreement between SAP and OCT measures.

Summary: VF assessment and OCT imaging of the RNFL and macula are essential methodologies for glaucoma monitoring and detecting progression. Given that the relationship between structure and function is nonlinear and highly dependent upon the severity of glaucoma, disagreement between assessments may be observed. Other factors that contribute to discordance between VF and OCT imaging results include measurements that may not be in direct topographic correspondence, imaging artifact and algorithm failure, measurement variability, and a floor effect in eyes with severe damage below which further loss can no longer be detected. In order to improve agreement between structure and function, clinicians should repeat poor quality tests, perform more frequent monitoring, incorporate ancillary diagnostic tests such as central 10-2 VF testing and macular imaging, assess localized change to enhance detection sensitivity, and document optic disc hemorrhage when present.

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Meds After MIGS

Alex Ansun Huang MD

- I. Spectrum of Glaucoma and Treatment
 - A. Types and severity
 - B. Treatments (drops to laser to MIGS to surgeries)
- II. Combination Therapies
 - A. Different meds
 - B. Meds + specific treatments (plus laser/surgery)
- III. Meds + MIGS
 - A. We do have some current concepts, divided into 3 categories: trabecular meshwork (TM), distal outflow, and steroid-response concepts.
 - B. The problem is the lack of data.
- IV. TM Level Concepts
 - A. Histology with picrosirius red slide demonstrating TM/scleral spur/angle anatomy
 - B. Pilocarpine, ciliary muscle, scleral spur, lever concept, and rationale for muscarinic IOP control
 - C. Aqueous angiography shows that pilocarpine increases aqueous humor outflow (AHO).
 - D. Recommendations of pilocarpine use started with goniotomy.^{1,2}
 - E. Trabectome
 - 1. Without pilocarpine, peripheral synechiae can form.³
 - 2. However, limited data says pilocarpine does not help Trabectome results.⁴
- V. Distal Outflow Pathway Concepts
 - A. AHO is segmental, as shown by aqueous angiography.^{5,6}
 - B. AHO is nonstatic (dynamic AHO and rescuable AHO). ^{5,7-9}
 - C. Teleological argument for stable vision: Vision requires stable optics, which requires stable IOP; thus there have to be regulatory points. (See TM/ pilo above and here introduce distal control.)
 - D. Post-trabeculotomy, outflow resistance still exists (see ET1 and DETA-nitric oxide) in experimental models.¹¹
 - E. New drugs (cytoskeletal relaxing agents) can impact distal outflow (eg, lower episcleral venous pressure [netarsudil]).¹⁰
 - F. Evidence of drug-induced structural change at episcleral vein and collector channels (ET1 and DETA-nitric oxide) in experimental models.

- VI. Steroid-Response Concept
 - A. What is steroid-response and how to distinguish it from surgical failure?
 - B. Steroid-response still occurs following trabecular MIGS.^{12,13}
 - C. Steroids can induce steroid response-type changes in scleral fibroblasts surrounding the distal outflow pathways, providing a possible mechanism.
- VII. Recommendations
 - A. Pilocarpine after trabecular ablation: Conflicting, thus minimal strength. Needs further study to just decide if this has no role.
 - B. Cytoskeletal relaxing agents after trabecular bypass: Theoretical, thus minimal strength. Needs further study. Has potential.
 - C. Quick steroid response after MIGS: Limited data, thus minimal strength.
 - D. Conclusion: Many points of synergy may exist with MIGS and pharmacological therapy, but more data are needed.

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Moving Glaucoma Neuroprotection Into Clinical Trials

Jeffrey L Goldberg MD PhD

- 1. Unmet need for neuroprotection and other therapies to supplement IOP lowering
- 2. Candidate therapies with strong preclinical evidence
- 3. Considerations in clinical trial design and exploratory vs. pivotal endpoints
- 4. Clinical data from early phase trials in progress

Is There a Role for Marijuana? Up in Smoke: Cannabis and Glaucoma

Bret A Hughes MD

- I. The Pathogenesis of Glaucoma Is Complex and Multifactorial
 - A. IOP factor most closely associated with onset and progression; reduced axoplasmic flow and blood flow
 - B. Lower IOP prevents structural damage and functional vision loss.
- II. Glaucoma Is the Second Leading Cause of Blindness
 - A. 66 million diagnosed
 - B. 10% will develop blindness.
 - C. Third most common reason patients >65 years visit MD
- III. Traditional Glaucoma Therapy
 - A. Prostaglandins
 - B. Beta blockers
 - C. Alpha agonists
 - D. Carbonic anhydrase inhibitors
 - E. Cholinergics
- IV. Complementary Treatments
 - A. Alternative medicine is not taught in medical school and not covered by medical insurance.
 - B. 50% of patients believe in complementary medicine.
 - C. 5% of glaucoma patients use complementary medicine.
 - 1. Herbal
 - a. Ginkgo biloba
 - b. Bilberry
 - c. Marijuana/Cannabis
 - 2. Vitamins
 - 3. Diet
 - 4. Exercise
- V. Ancient Societies With Documented Medicinal Use, 500 AD
 - A. Asia
 - B. Africa
 - C. Middle East
- VI. Cannabis
 - A. Plant contains >100 individual (phyto)cannabinoids in addition to tars, teratogens, carcinogens (greater amounts than tobacco), pesticides?

- B. Predominant cannabinoids
 - 1. Tetrahydrocannabinol (THC)
 - 2. Cannabidiol (CBD)
- VII. United States of Hemp
 - A. Hemp derived from seeds or stalk of cannabis plant
 - 1. Used to make rope, sails, clothing, textiles ...
 - 2. Lower in THC
 - B. Marijuana is the flowering bud, higher in THC.
 - C. 1691 Virginia passed law requiring farms/colonists to grow hemp.
 - D. Cannabis hemp is one of history's most widely used plants; seeds used for oils, paints, and varnishes.
 - E. Tincture of Cannabis was the basis for most patented medicines prior to discovery of aspirin.
- VIII. Federal Law: Controlled Substance Act of 1970
 - A. Control all stages of manufacture, distribution, supply chain, handlers, users
 - B. Classification I-V
 - 1. Based on medical effectiveness
 - 2. Potential for abuse
- IX. Amendment to Controlled Substance Act
 - A. DEA developed 5 criteria
 - 1. Drug chemistry should be known and reproducible.
 - 2. Safety studies
 - 3. Efficacy studies
 - 4. Accepted by medical experts
 - 5. Scientific evidence widely available; anecdotal studies would not satisfy these criteria for commercial consideration.
- X. Compassion Use Act of 1996
 - A. Federal law allows medicinal use of Cannabis despite Controlled Substance Act.
 - B. California was the first state to institute medical use.
 - C. Currently 30 states, D.C., and Puerto Rico allow medicinal use.
 - D. Currently 10 states allow recreational use.

- XI. Cannabis and Glaucoma
 - A. Merritt et al and Helper, Frank, et al evaluated cannabis in normal and glaucoma patients; IOP reduction 20%-30%.
 - B. Inhalation, intravenous, oral are effective.
 - C. Topical not effective (lipophilic); poorly crosses corneal epithelium
- XII. Cannabis Inhalation
 - A. Heat/vaporization needed for activation
 - B. Quickest onset (10 minutes)
 - C. Shortest duration (3-4 hours)
 - D. Processed by liver
 - E. Stored in brain, vascularized organs, and fatty tissue
- XIII. Cannabis Effects
 - A. Hypotension/orthostatic changes
 - B. Tachycardia
 - C. Emphysematous lung changes
 - D. Motor coordination
 - E. Abuse
 - F. Psychotropic effects
 - 1. Altered mood
 - 2. Paranoia
 - 3. Hallucinations
 - 4. Difficulty thinking, problem solving
 - 5. Memory
- XIV. Cannabis Dosing
 - A. 6-8 cigarettes daily
 - B. 2920 cigarettes annually
 - C. \$10 cigarette
 - D. \$80 day/\$29,200 annual
- XV. American Glaucoma Society Position Statement

Cannot recommend smoking marijuana as treatment based on side effects and risks involved, compared to benefit in IOP reduction

- XVI. Cannabis Mechanism of Action
 - A. Central vs. peripheral
 - B. Sympathetic vs. parasympathetic
 - C. Aqueous suppressant vs. enhanced outflow
 - D. Vascular component?
- XVII. Endocannabinoid System
 - A. CB1: psychoactive receptor, primarily stimulated by THC
 - B. CB2: immune modulation, primarily stimulated by CBD

- XVIII. Endocannabinoid System
 - A. AEA (arachidonoylethanolamine)
 - B. 2-AG (2-arachydonoylglycerol)
 - 1. Both are naturally occurring endocannabinoids.
 - 2. Mimic effects of phytocannabinoids
 - C. GI, appetite, metabolism, pain, memory, movement, immune system
- XIX. Endocannabinoid System in the Eye
 - A. CB1 receptors in various parts of the eye; anterior segment
 - B. CB2 receptors in the retina and trabecular mesh-work
 - C. Natural and synthetic molecules can interact with endocannabinoid system.
 - 1. IOP reduction
 - 2. Neuroprotection
- XX. Endocannabinoid System and Neuroprotection
 - A. Glutamate excitatory neurotransmitter
 - B. Elevated levels of glutamate found in vitreous of glaucoma patients
 - C. Elevated level triggers excitotoxic cascade, leading to retinal ganglion cell (RGC) death.
 - D. Glutamate receptor antagonists conferred neuroprotection (including diminished RGC apoptosis).
- XXI. Endocannabinoid System of Neuroprotection
 - A. Studies show CB receptor activation inhibits glutamate release.
 - B. Maintaining agonists of CB important neuroprotective effect in preventing RGC death

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How to Handle the Dissonance Between OCT and Gonioscopy

David S Friedman MD MPH PhD

I. Introduction

Glaucoma is the second leading cause of blindness globally, and although primary open-angle glaucoma (POAG) is present in nearly 60% of cases, primary angle-closure glaucoma (PACG) is more severe and more frequently results in blindness. Determining who has angle-closure glaucoma remains problematic. One study from the United States reported that nearly half the charts of patients with "POAG" had no evidence of gonioscopy in the chart. Without assessment of the angle, may cases of PACG will be missed.

- II. Overview of Gonioscopy for Angle Closure
 - A. Widely used, various approaches, Goldmann-style lenses vs. 4-mirror lenses
 - B. Advantages of gonioscopy
 - 1. Allows visualization of peripheral anterior synechiae
 - 2. Allows pigment to be seen
 - 3. Compression is fairly straightforward.
 - C. Disadvantages of gonioscopy
 - 1. Patient discomfort
 - 2. Possible infection, corneal abrasion
 - 3. Subjective, significant variation between observers and possibly intraobserver variation
 - 4. Difficult to document in the medical record photographically
 - 5. Need for illumination and eye contact
 - a. Light falling on the pupil can result in opening of the angle.
 - b. Touching the eye can compress the angle open, even with Goldmann lenses.

- III. Overview of OCT for Angle Closure
 - A. Many devices, no clear standard imaging approach
 - 1. Spectral domain OCT
 - 2. Swept source OCT
 - B. Advantages
 - 1. Minimal illumination required, so angle maximally closed
 - 2. No compression required
 - 3. No drops needed
 - 4. Potentially automated assessment of angle structures
 - 5. Allows for categorization of principle causes of angle closure
 - a. Pupil block
 - b. Peripheral iris crowding
 - c. Lens vault
 - d. Others
 - 6. Highly reproducible
 - 7. Images are able to be stored; could be integrated into artificial intelligence algorithms
 - C. Disadvantages
 - 1. Difficult to image superior angle
 - 2. Costly
 - 3. Currently seems to over diagnose
- IV. Conclusion

Currently, gonioscopy remains a reference standard for angle closure diagnosis. The field is evolving, and the future looks promising for OCT-based angle assessment.

Does the Use of Third, Fourth, and Fifth Drugs Delay Surgery? How Much Medication Is Too Much?

Janet B Serle MD

With the recent introduction of 2 new classes of ocular hypotensive medications (nitric oxide donating prostaglandin analog [PGA] and rho kinase inhibitor), the standard of care for maximum medical management of glaucoma needs to be defined. In 1994, when fewer classes of ocular hypotensive medications were available, the mean number of maximum tolerated medications of patients at baseline enrolled in the Advanced Glaucoma Intervention Study was 2.7 for both the argon laser trabeculoplasty (ALT)-trabeculectomy-trabeculectomy and trabeculectomy-ALT-trabeculectomy treatment groups. After the introduction of prostaglandins in 1996, the mean number of maximum tolerated medications increased. For patients enrolled in the Tube Versus Trabeculectomy Study, the mean number of medications at baseline was 3.2 ± 1.1 in the tube group and 3.0 ± 1.2 . in the trabeculectomy group; in the Ahmed vs. Baerveldt Study it was 3.1 ± 1.0 in both treatment groups; and in the Ahmed Baerveldt Comparison Study it was 3.4 ± 1.1 in the Ahmed group and 3.5 ± 1.1 in the Baerveldt group.

The number of classes of medications and available combinations for lowering IOP continues to expand. If a patient was prescribed all classes of ocular hypotensive medications currently available, they could conceivably be receiving 7 different classes of medications and as many as 16 drops a day (beta blocker q.d. or b.i.d., PGA q.d., topical carbonic anhydrase inhibitor b.i.d. or t.i.d., selective alpha adrenergic agonist b.i.d. or t.i.d., nitric oxide donating PGA q.d., rho kinase inhibitor q.d., miotic t.i.d. or q.i.d., nonselective adrenergic agonist b.i.d.). This particular regimen is not advised, and presumably rarely if ever prescribed. Fixed-dose combination products do reduce the frequency of daily instillation of drops, addressing the issues of compliance and potential ocular surface disease, and allowing 2 or more classes of medications to be administered simultaneously (brinzolamide/brimonidine, dorzolamide/timolol, latanoprost/ nitric oxide donor, netarsudil/latanoprost). Other commercially available combinations of 2 drugs are approved outside of the U.S.A. (latanoprost/timolol, travoprost/timolol, bimatoprost/ timolol, brinzolamide/timolol). Combinations of 3 drugs are available through compounding pharmacies in the U.S.A. and approved as commercial products outside of the U.S.A. (triple fixed-combination bimatoprost/brimonidine/timolol, timolol/ brimonidine/dorzolamide). Patients are often receiving 3 or 4 different classes of medications a day, as has been described in clinical reports and many CME publications that include case presentations, and as can be found when practitioners review their patient files. The efficacy of the various ocular hypotensive agents has been most commonly defined when used as solo therapy. Additivity studies typically include 2 medications, and rarely more. The additive effects on IOP of additional medications has rarely been described in prospective studies and has mostly been observed in retrospective and compassionate case studies, which involve complicated regimens and limited opportunity to assess compliance, and through anecdotal experience in the office.

Medical therapy is associated with many limitations and potential complications. In addition to the specific ocular and systemic side effects associated with each class of compound, there are many patient-specific factors. These include patient compliance, particularly with more complicated dosing regimens, patient age, infirmity, facility with administration, medication cost, ocular surface disease, level of education, and health literacy. Adequate control of intra-day and inter-day IOP fluctuations may not be achieved with tolerated medications. Limited nocturnal efficacy is associated with some classes of compounds typically in use (beta antagonists, selective alpha₂ adrenergic agonists). Changes in the conjunctiva from chronic medication use may lower the success rate of incisional glaucoma surgery.

When assessing the maximum number of medications that may be reasonable and sufficiently effective, all of the above factors are considered. The physician needs to determine if the individual patient regimen may achieve the target IOP, basing this determination on many factors, including stage of disease and rate of progression. Laser trabeculoplasty has been recommended and is being utilized earlier in the treatment scheme, both as first-line treatment and as an adjunct to medications. Some retrospective evaluations suggest reduced efficacy of selective laser trabeculoplasty (SLT) in patients on multiple IOPlowering medications, other evaluations suggest similar efficacy. It is unclear if the range of effects of SLT may be due to effects of medications on trabecular tissues or the responsiveness of the meshwork at various stages of glaucoma.

Newer surgical techniques for lowering IOP (minimally invasive glaucoma surgery [MIGS]) have reduced the postoperative recovery time and the postoperative complication rate, and thus are being considered and may be indicated for treatment earlier in the disease. Particularly in patients being treated for elevated IOP who require cataract surgery, combining cataract with MIGS is rapidly becoming the standard of care. This combined treatment often reduces the postoperative medication burden, may reduce postoperative IOP elevations, and may reduce 24-hour IOP fluctuations.

Some studies have suggested a cost benefit to proceeding with surgery compared to the costs of maximum medical therapy. A large prospective evaluation of patients newly presenting with advanced glaucoma is being conducted in the UK. Patients are being randomized to medications vs. trabeculectomy with mitomycin, and outcomes include vison-related quality of life, efficacy, and cost of the treatments.

The maximum number of medications that are effective, tolerated, and complied with varies markedly. The most efficacious and best-tolerated combinations of medications is unlikely to be uniform for all patients. Some patients may be well controlled with 3 or more medications; for others this is an unrealistic burden. Attempts should be made to educate patients about the benefits and risks of both medical and surgical options, and treatment decisions should be made in concert with the patient.

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How to Protect Glaucoma Drainage Devices From Erosion

Victoria M Addis MD

- I. Background
 - A. Glaucoma drainage devices (GDDs) are increasingly being used as a primary surgery to lower IOP.
 - B. Results of Tube versus Trabeculectomy (TVT) Study¹
- II. Late Complications of GDD Implants
 - A. Tube erosion/extrusion²
 - 1. Seen in 2%-7% of eyes after GDD implantation
 - 2. Several mechanisms proposed
 - a. Mechanical rubbing of conjunctivae over tube
 - b. Tension of conjunctivae over tube
 - c. Patch graft melting
 - d. Abnormal positioning of the tube
 - 3. Increased risk of ocular inflammation, endophthalmitis, hypotony, and phthisis
 - B. Elevated IOP or hypotony
 - C. Diplopia
 - D. Endophthalmitis
 - E. Corneal decompensation
 - F. Cataract
- III. Risk Factors for GDD Exposure
 - A. Implant location: Inferior location more likely to become exposed than superior location³
 - B. Type of patch graft⁴
 - 1. Sclera associated with lowest risk of exposure in some studies
 - 2. Implants covered with cornea and pericardium associated with highest exposure rates
 - C. Patient age and demographics
 - D. Preoperative patient characteristics⁵
 - E. Risk factors for GDD re-exposure⁶

- IV. Techniques for GDD Tube Coverage
 - A. Patch graft⁷
 - B. Scleral tunnel⁸
- V. Management of GDD Exposure9
 - A. Tube revision
 - B. Tube removal
 - C. Outcomes¹⁰

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There Is Something You Can Do

John D Shepherd MD

The patient presented to my low vision practice with advanced glaucoma. A review of her ophthalmologist's notes demonstrated stable IOPs in the target range and stable visual fields. Her increasing tendency to trip and fall was not noted or discussed. Her primary care provider completed a thorough medical workup to evaluate this tendency to trip and fall, but no cause was found. The primary care provider referred the patient to me to determine if the vision loss from the glaucoma might be the problem. It was.

As ophthalmologists, we know that despite our best efforts, glaucoma can cause irreversible vision loss. This can create challenges for our patients with reading, taking a walk at the mall, or even driving. Safety and ability to live independently can be significantly compromised. Vision rehabilitation addresses these very challenges. Although the potential benefits of vision rehabilitation are known, many patients never benefit from these services because they are not referred by their ophthalmologist.

The Vision Rehabilitation Committee (VRC) of the American Academy of Ophthalmology (the Academy) is very grateful to Dr. David W. Parke III, chief executive officer of the Academy, for introducing the video entitled "There Is Something You Can Do." In this video, which demonstrates the benefits of vision rehabilitation in the lives of patients with central and/or peripheral vision loss, Dr. Parke states, "One of the things we can do as ophthalmologists is to realize the importance of referral for vision rehabilitation for any patient who is starting to lose their vision. And it's most effective when we do this early ... at a time when they can really begin to involve themselves in the vision rehabilitation process. Vision rehabilitation is now the standard of care for patients who are losing their vision."

One of the challenges we face as ophthalmologists is identifying the patient who may benefit from vision rehabilitation. The Academy's Vision Rehabilitation Preferred Practice Pattern (PPP) guidelines suggest offering referral to vision rehabilitation for patients with BCVA less than 20/40, visual field loss, a scotoma, or contrast sensitivity loss. A screening questionnaire may be helpful. For example, the Glaucoma Activity Limitation questionnaire consists of 9 questions and can be administered by office staff in a glaucoma practice. Perhaps the simplest way to identify an appropriate patient is to ask any patient with irreversible vision loss this one question: "Does your vision loss make it difficult for you to participate in your favorite activities?" A "yes" response should prompt a referral. Vision rehabilitation providers need to be seen as partners with comprehensive ophthalmologists and glaucoma specialists in the holistic care of glaucoma patients. Truly comprehensive patient care aims to protect the remaining vision through consistent care, restore what is lost through medical and surgical treatments, *and* build on what remains through rehabilitation. The Academy's mission and motto is "Protecting sight. Empowering lives." While it is important to protect the sight of our patients through appropriate medical and surgical treatments, it is equally important to empower their lives by providing the tools, counseling, and training to live as well as they can despite irreversible vision loss.

For more information on empowering the lives of patients through vision rehabilitation, consider attending SYM12 "Vision Rehabilitation for Glaucoma" at 2:15 PM on Saturday, Oct. 12, at the annual meeting. In case you are unable to attend, stay tuned; an attempt is being made to video record the symposium. You may also find out more by going to the Academy website, www.aao.org, and entering "vision rehabilitation" in the search bar.

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Casting Your Net for the Best Glaucoma Surgery at Fisherman's Wharf

Lama A Al-Aswad MD MPH and Davinder S Grover MD

Panelists: George A Cioffi MD, Joseph F Panarelli MD, Pradeep Y Ramulu MD PhD, Oluwatosin U Smith MD, and Scott M Walsman MD

NOTES

AGS-IRIS[®] Registry Project

Glaucoma Characteristics for MIGS Utilization: IRIS® Registry Analysis

Mildred MG Olivier MD, Eydie Miller Ellis MD, Maureen Maguire PhD, Tosin Smith MD, Brian VanderBeek MD MPH, and Clarisse Croteau-Chonka PhD

- I. Demographics
 - A. Ethnicity data
 - B. Stratified procedures
- II. Results
 - A. Number of medications
 - B. IOP
 - C. Cup-to-disc ratios
 - D. Reoperation rates
 - E. Complications
- III. Limitations
 - A. Visual fields
 - B. Imaging studies
 - C. EMR

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2. Nguyen AH, Fatehi N, Romero P, et al. Observational outcomes of initial trabeculectomy with mitomycin C in patients of African descent vs patients of European descent: five-year results. *JAMA Ophthalmol.* 2018; 136(10):1106-1113.

OHTS III Data Ocular Hypertension Treatment Study: 20-Year Incidence and Severity of POAG

Richard K Parrish MD for the Ocular Hypertension Treatment Study Group

From 1994 to 1996, 1636 participants with ocular hypertension were randomized to treatment with hypotensive medication (MED) or observation (OBS). The cumulative incidence of primary open-angle glaucoma (POAG) at 60 months was reduced in the OBS group from 9.5% to 4.4% (hazard ratio, 0.40; 95% CI, 0.27-0.59; *P* < .0001). In Phase 2, OBS participants were started on meds to determine if there was a penalty for delaying treatment. After 5 years, the cumulative proportion of participants who developed POAG in Phase 2 was virtually identical in the OBS and MED groups, 11% and 12%, respectively. From 2016 to 2019, participants (median age: 75) were recalled to assess functional status. As of May 2019, functional status was ascertained for 74% (845 of 1143) of survivors. We report 20-year cumulative POAG incidence overall, by randomization group and by race. We report POAG severity by mean deviation, contrast sensitivity, visual acuity, and bilaterality of visual field loss.

Are You AT the Table or ON the Menu?

Donald L Budenz MD MPH

Ophthalmology's goal to protect sight and empower lives requires active participation and commitment to advocacy from every ophthalmologist. Contributions to the following three critical funds are a part of that commitment:

- OPHTHPAC[®]
- Surgical Scope Fund (SSF)
- State Eye PAC

Please join the dedicated community of ophthalmologists who are contributing to protect quality patient eye care for everybody.

The OPHTHPAC Committee is identifying Congressional Advocates in each state to maintain close relationships with federal legislators to advance ophthalmology and patient causes. At Mid-Year Forum 2019, we honored three of those legislators with the Academy's Visionary Award. This served to recognize them for addressing issues important to us and to our patients. The Academy's Secretariat for State Affairs is collaborating closely with state ophthalmology society leaders to protect Surgery by Surgeons at the state level.

Our mission of "protecting sight and empowering lives" requires robust funding of both the Surgical Scope Fund and OPHTHPAC. Each of us has a responsibility to ensure that these funds are strong so that ophthalmology can be represented "at the table."

OPHTHPAC®

OPHTHPAC represents the profession of ophthalmology to the U.S. Congress and operates to protect you and your fellow ophthalmologists from payment cuts, burdensome regulations, scope-of-practice threats, and much more. OPHTHPAC also works to advance our profession by promoting funding for vision research and expanded inclusion of vision in public and private programs—all of which provide better health-care options for your patients. OPHTHPAC is your federal voice in Washington, D.C., and we are very successful in representing your professional needs to the U.S. Congress.

Among OPHTHPAC's most recent victories are the following:

- Securing greater flexibility in the new Medicare Payment System
- Ensuring proper reimbursement of Medicare Part B drugs
- Blocking onerous administrative burdens on contact lens prescribers
- Preserving access to compounded drugs
- Preventing additional cuts to Medicare

However, ophthalmology's federal issues are a continuous battle, and OPHTHPAC is always under pressure to ensure we have strong political connections in place to help protect ophthalmology, its members, and their patients.

The support OPHTHPAC receives from invested U.S. Academy members helps build the federal relationships that advance ophthalmology's agenda on Capitol Hill. These relationships allow us to have a seat at the table with legislators willing to work on issues important to us and our patients. We also use these congressional relationships to help shape the rules and regulations being developed by federal agencies. Help strengthen these bonds and ophthalmology's legislative support.

Right now, major transformations are taking place in health care. To ensure that our federal fight and our PAC remain strong, we need the support of every ophthalmologist to better our profession and ensure quality eye care for our patients. Invest with confidence in the strongest PAC working to ensure your success as an ophthalmologist.

Contributions to OPHTHPAC can be made here at AAO 2019, online at www.aao.org/ophthpac, or by texting MDEYE to 41444.

At Mid-Year Forum 2019, the Academy and the American Glaucoma Society (AGS) ensured a strong presence of glaucoma specialists to support ophthalmology's priorities. Ophthalmologists visited members of Congress and their key health staff to discuss ophthalmology priorities as part of Congressional Advocacy Day. The AGS remains a crucial partner with the Academy in its ongoing federal and state advocacy initiatives.

Surgical Scope Fund

The Surgical Scope Fund (SSF) provides grants to state ophthalmology societies to support their efforts to protect patient safety from dangerous optometric surgery proposals. Since its inception, the Surgery by Surgeons campaign and the SSF, in partnership with state ophthalmology societies, have helped 40 state/ territorial ophthalmology societies reject optometric scope-ofpractice expansions into surgery.

Thanks to the 2019 SSF contributions from ophthalmologists just like you, SSF has had a successful year, preserving patient safety and surgical standards in state legislatures across the country, including six critical wins in Alabama, Texas, Vermont, Wyoming, Maryland, and Iowa. The 2019 battle is far from over, though. For example, Pennsylvania and Massachusetts are under attack, and California and Illinois are facing threats.

If you have not yet made a 2019 SSF contribution, *contributions can be made at our booth at AAO 2019 or online at www.aao.org/ssf.* If you already have made that 2019 contribution, please go to www.safesurgerycoalition.org to see the impact of your gift.

Dollars from the SSF are critical to building complete cutting-edge political campaigns, including media (TV, radio, and social media), educating and building relationships with legislators, and educating the voting public to contact their legislators. This work helps to secure success in protecting patient safety by defeating optometry's surgical initiatives.

Each of these endeavors is very expensive, and no one state has the critical resources to fight big optometry on their own. Ophthalmologists must join together and donate to the SSF at www.aao.org/ssf to fight for patient safety.

Surgical Scope Fund	OPHTHPAC [®] Fund	State EyePAC
To protect patient safety by defeating opto- metric scope-of-practice initiatives that threaten patient safety and quality surgical care	Ophthalmology's interests at the federal level Support for candidates for U.S. Congress	Support for candidates for state House, Senate, and governor
Political grassroots activities, government relations, PR and media campaigns	Campaign contributions, legislative education	Campaign contributions, legislative education
No funds may be used for campaign contribu- tions or PACs.		
Contributions: Unlimited	Contributions: Limited to \$5,000	Contribution limits vary based on state regu-
Individual, practice, and organization		lations.
Contributions are 100% confidential.	Contributions above \$200 are on the public record.	Contributions are on the public record depending upon state statutes.

The Secretariat for State Affairs thanks the AGS, which in the past has joined state ophthalmology societies in contributing to the SSF, and it looks forward to the society's 2019 contribution. These ophthalmic organizations complete the necessary SSF support structure for the protection of our patients' sight.

State Eye PAC

It is increasingly important for all ophthalmologists to support their respective State Eye PACs because campaign contributions to legislators at the state level must come from individual ophthalmologists and cannot come from the Academy, OPH-THPAC, or the SSF. The presence of a strong State Eye PAC providing financial support for campaign contributions and legislative education to elect ophthalmology-friendly candidates to the state legislature is critical, as scope-of-practice battles and many regulatory issues are all fought on the state level.

ACTION REQUESTED: Help Ophthalmology Ensure a "Seat at the Table"

Academy SSF contributions are used to support the infrastructure necessary for state legislative/regulatory battles and for public education. State PAC and OPHTHPAC contributions are necessary at the state and federal levels, respectively, to help elect officials who will support the interests of our patients. Contributions to *each* of these three funds are necessary and help us protect sight and empower lives. SSF contributions are completely confidential and may be made with corporate checks or credit cards, unlike PAC contributions, which must be made by individuals and are subject to reporting requirements.

Please respond to your Academy colleagues and be part of the community that contributes to OPHTHPAC, the SSF, and your State Eye PAC. Please be part of the community that ensures ophthalmology has a strong voice in advocating for patients.

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A Lymphatic-like Pump Controls Aqueous Outflow: POAG Management and MIGS Implications

Murray A Johnstone MD

- I. What Do We Know From Direct Observation of the Aqueous Veins?
 - A. Aqueous flows. Aqueous outflow is pulsatile!
 - B. Pulsatile aqueous outflow stops in glaucoma.
 - C. Pilocarpine restores pulsatile aqueous outflow.
- II. What Do We Know From Cardiovascular Physiology?
 - A. Like veins and lymphatics, the aqueous outflow system returns fluid to the heart.
 - B. Venous blood and lymph flow by pulsatile mechanisms.
 - C. Veins and lymphatics use displacement pumps to move fluid.
 - D. Displacement pumps have unique requirements:
 - 1. A chamber, inlet valves, and outlet valves
 - 2. Segments between the vein and lymphatic valves act as miniventricles.
 - 3. Walls of the miniventricle compartments must move to propel fluid forward.
 - 4. A driving force is present resulting from the cyclic cardiac pulse and transient tissue motion.
 - 5. The outflow system has Prox 1, known to be a marker for lymphatic valves.
 - 6. Defective Prox 1, necessary for lymphatic valve development, causes a glaucoma.
- III. What Have We Known Before Recent OCT Imaging Advances?
 - A. The trabecular meshwork (TM) is the wall of a vessel called the Schlemm canal (SC).
 - B. The SC is a chamber. SC chamber volume changes with IOP.
 - C. Aqueous-containing endothelial-lined conduits arise from the SC inner wall.
 - D. The conduits attain a tube-like shape, cross the SC, and attach to the external wall.
 - E. The conduits act as SC inlet valves (SIVs), allowing flow and preventing blood reflux.
 - 1. Light, scanning, and transmission electron microscopy document SIV structure.
 - 2. Microsphere and RBC tracer studies document SIV function as conduits.
 - 3. During SC unroofing, SIVs break and discharge aqueous.

- 4. During gonioscopy, SIVs discharge oscillating waves of aqueous into the SC.
- IV. What Insights Does Ex Vivo High-Resolution OCT Provide?
 - A. Collector channel (CC) valves
 - 1. CC entrances have collagen flaps attached only at one end.
 - 2. The CC hinged flaps undergo pressure-dependent changes in position.
 - 3. Position changes allow the hinged flaps to open and close CC.
 - 4. Position changes enable the flaps to act as SC outlet valves (SOV).
 - 5. SIV provides connections between the TM and the SOV hinged flaps.
 - 6. SIV elongate as SC pressure increases, placing tension on the hinged flaps.
 - B. Circumferentially oriented deep scleral plexus (CDSP) channels are adjacent to the SC.
 - 1. Thin septa separate the CDSP from the SC and move by pressure-dependent mechanisms.
 - 2. The CDSP opens and closes like a second pressure-dependent chamber.
 - C. Evidence from real-time imaging of tissue motion
 - 1. The TM beams, SIV, SOV, and CDSP all undergo rapid cyclic pulsatile movement.
 - 2. The amplitude and speed of motion can account for all of the aqueous outflow.
 - 3. Motion of TM, SIV, and SOV, as well as the SC and CDSP volume Δ s, are all synchronous.
 - 4. Cellular attachments between the structures can explain the synchrony of motion.
- V. What Insights Does Human In Vivo Phase Sensitive OCT (PhS-OCT) Provide?
 - A. Trans-scleral OCT in human subjects is challenging because of motion and light scattering.
 - B. Commercial spectral domain OCT systems using an 810-nm wavelength have limited sensitivity.
 - C. A purpose-built 1310-nm PhS-OCT system resolves motion of ~20 nm.
 - D. The PhS-OCT system quantitates TM velocity and displacement.
 - E. A recent report finds significant motion differences between normal and glaucoma eyes.

- VI. What Benefits May Motion Monitoring by PhS-OCT Provide for Medical Management?
 - A. Infrequent office IOP measurements are of poor predictive value for IOP peaks and fluctuation.
 - B. IOP is measured 3 to 4 times a year, thus sampling IOP for ~12 seconds/year.
 - C. Patients look straight with no blinking or eye movement, preventing the capture of transients.
 - D. As a pump, the outflow system requires movement to function properly.
 - E. Poor TM movement may predict early aqueous outflow abnormalities.
 - F. PhS-OCT measurement of TM motion may be an alternative to brief, infrequent IOP sampling.
 - G. TM movement deterioration may be a sensitive indicator of a need for medication escalation.
 - H. Improved TM movement following outflow drugs may assess their effectiveness.
- VII. What Aqueous Outflow Pump Behavior May Explain MIGS Effects? Priming the Pump?
 - A. Real-time OCT imaging of motion following insertion of a MIGS-like cannula into the SC
 - B. Pressure introduced into SC simulates AC pressures and transients after MIGS.
 - 1. A SC pressure increase at device end causes TM movement and SC dilation ≥5 mm distally.
 - 2. SC pressure changes cause collector channels to open and close distal to the insertion area.
 - 3. SC and CC dilation is sufficiently rapid to permit pulsatile aqueous flow.
 - 4. CDSP channels in the deep scleral plexus open and close with SC pulsatile pressure.
- VIII. Can Pump Function Improvement Explain IOP Reduction After Cataract Extraction?
 - A. Pilocarpine briefly restores pump function by improving scleral spur (SS) traction.
 - B. High-resolution MRI demonstrates improved spur vectors and traction after cataract surgery.
 - C. Reports propose that cataract surgery improves pump function by improved SS traction.
- IX. Can Awareness of the Pump Function Suggest New Therapies?

An ideal goal: Restoration of pump function without frequent meds or invasive surgery.

A. Pilocarpine temporarily restores pulsatile flow but with many side effects.

- B. Trans-scleral micropulse laser (TMP) simulates pilocarpine effect on outflow system.
- C. Can optimization of TMP delivery systems and parameters provide a persistent effect?

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- Microscope real-time video (MRTV), high-resolution OCT (HR-OCT) & histopathology (HP) to assess how transscleral micropulse laser (TML) affects the sclera, ciliary body (CB), muscle (CM), secretory epithelium (CBSE), suprachoroidal space (SCS) & aqueous outflow system. ARVO Abstract 2825, p. 182, 2019.

Normal Pressure Glaucoma Masqueraders

Ahmara Ross MD PhD

Introduction

Glaucoma is the most common optic neuropathy, but its presentation includes signs and symptoms that are similar to those of other neuro-ophthalmic conditions, such as damage to the optic nerve and gradual loss of vision. Although glaucoma has been generally viewed as a separate disease entity, with potential treatment aimed at decreasing IOP, a concerning subset of this disease is the normal-tension glaucoma (NTG) variant. Laboratory research continues to provide a more detailed understanding of the etiology and pathophysiology of optic nerve death in glaucoma, leading to a true paradigm shift to our understanding of glaucoma as another neurodegenerative disease rather than a separate diagnosis. With this shift comes a more complex outlook on the diagnosis of normal-tension glaucoma with routine tools used in the clinic.

Various initial clinical presentations of normal-tension glaucoma usually raise "red flags" and suspicion when the patient's vision continues to decline despite adequate treatment. With this discovery, misdiagnosis and delay in treatment can leave uncertainty regarding the diagnosis initially. In this presentation, I will discuss various case scenarios of neuro-degenerative diseases with key points that could be used to eliminate normaltension glaucoma from the differential diagnosis.

Background Observations

The most common normal-tension variant mimicking neurodegenerative disorders that lead to overdiagnosis include compressive optic neuropathy and toxic optic neuropathy. Additionally, the conditions resembling normal-tension glaucoma that result in overdiagnosis are patients with concurrent neurodegenerative diagnosis or retinal degenerative diseases.

In this presentation, I will discuss pertinent history, examination findings, and imaging modalities as clues to avoid missing these entities in the clinical setting.

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Compressive Optic Neuropathies	Toxic Optic Neuropathies	Neurodegenerative Diseases	Retinal Degenerative Diseases
Midline tumors (craniopharyn- gioma, pituitary adenomas)	Alcohol-induced amblyopia	Parkinson disease	Pathogenic myopia
Optic nerve sheath tumors (meningioma)	Drug-induced amblyopia (amiodarone, ethambutol)	Parkinson-like diseases	
Infiltrative optic nerve disease (gliomas)	Radiation	Multiple sclerosis	
		Leber/hereditary optic neuropathy	

Table 1. Common Normal Tension Variant Mimickers

Addressing the Normal-Pressure Glaucoma Patient With Systemic Hypotension

Jody Piltz-Seymour MD

Why Is Low BP Important?

- I. Increased Prevalence of Glaucoma With Low Diastolic Perfusion Pressure
 - A. Low ocular perfusion pressure (OPP) is associated with an increased prevalence of glaucoma in population-based studies.
 - 1. Barbados Eye Study¹: Low diastolic OPP (<55 mmHg) was a risk factor for the development of glaucoma (relative risk: 3.2).
 - 2. Baltimore Eye Survey²: Subjects with low diastolic OPP (<30 mmHg) had a 6x increased open-angle glaucoma (OAG) prevalence.
 - 3. Rotterdam Study³
 - 4. Proyecto VER⁴
 - Singapore Malay Eye Study⁵: Low diastolic blood pressure (BP), low mean OPP, and low diastolic OPP are independent risk factors for OAG development.
 - B. Glaucoma prevalence decreases as diastolic perfusion pressure increases (Egna-Neumarkt study⁶).
 - C. Low diastolic and high systolic BP are associated with an increased prevalence of primary OAG (POAG) (J-shaped curve) in the Los Angeles Latino Eye Study.⁷
- II. Increased Incidence of Glaucoma With Low Perfusion Pressure
 - A. Barbados Eye Study
 - 1. Longitudinal population-based study
 - 2. 3222 African origin participants monitored for 9 years
 - 3. OAG incidence: 4.4%
 - 4. Higher incidence of OAG with lower systolic, diastolic, and mean perfusion pressure
 - B. Not verified by the Rotterdam 20-year follow-up study
- III. Increased Progression of Glaucoma Low OPP
 - A. Early Manifest Glaucoma Trial⁸: Low OPP (<125 mm Hg) significantly increased the risk of progression of glaucoma, and higher OPP (>160 mmHg) was protective. Follow-up: 11+ years.
 - B. Barbados

- C. Ramin et al⁹ study of 65 normal-tension glaucoma (NTG) patients studied for 5 years with baseline 24-hour BP and IOP measurements.
 - 1. Twenty-three eyes (35.4%) reached the progression endpoint.
 - 2. Progressors vs. nonprogressors: Low nocturnal diastolic OPP is an independent predictor of glaucomatous visual field (VF) progression in NTG patients.
 - a. No difference in comorbidities such as systemic hypertension, diabetes, or medication class of hypertension treatment
 - b. The progressing group had significantly lower diastolic BP and diastolic perfusion pressure (day, evening, and night).
 - c. No significant difference detected in 24-hour IOP
 - d. No difference in the nocturnal dip percentage in the progressing group
- IV. Controversies With NTG
 - A. Rotterdam¹⁰: Low perfusion pressure was positively associated with high-tension glaucoma but inversely associated with NTG.
 - B. Egna-Neumarkt Study: Did not find an association with low diastolic PP and increased prevalence of NTG
- V. Nocturnal Hypotension and NTG: The Problem of the Overdippers
 - A. Nocturnal dips of BP are a result of diminished sympathetic nervous system activity during sleep and are physiologic. They are protective against cardiovascular mortality (MI, CVA, CHF).
 - B. Approximately 10% of people have <10% decrease in BP at night and are labeled "nondippers." Nondippers have an increased risk of cardiovascular mortality, particularly if they are hypertensive.
 - C. Even though IOP increases with supine position, true OPP increases by approximately 15 mmHg when changing from standing to supine position as the eye aligns horizontally with the heart. Typical nocturnal dips of BP of 10-20 mmHg should not result in ischemic injury in normal eyes.

- D. Data are variable, but most data suggest greater dips in nocturnal BP may be related to glaucoma progression.
 - 1. NTG and HTG patients with VF progression had lower nocturnal BP and greater nocturnal dips of BP.¹¹ Ambulatory BP analyzed in 70 patients, with 5 years of progression data available.
 - 2. Nondippers and extreme dippers were more likely to progress than those with a normal dipping pattern.¹²
 - 3. Nocturnal overdipping was more likely to result in VF progression in patients with normal BP rather than in hypertensives.¹³ Meta-analysis of 5 papers demonstrated that the odds ratio for deteriorating VF was 3.32 for systolic nocturnal BP dips >10% and 2.09 for diastolic nocturnal BP dips >10%.¹⁴
 - 4. POAG patients with nocturnal BP dips demonstrated reduced retrobulbar blood flow parameters.¹⁵
- E. Nocturnal OPP and diastolic and systolic BP were lower in NTG vs. controls. 16
- VI. Disc Hemes and BP

Nocturnal overdippers have an increased risk of optic disc hemorrhages. $^{17}\,$

- VII. Impaired Autoregulation
 - A. Longstanding hypertension may lead to atherosclerosis, increased vascular resistance, and impaired vascular autoregulation.
 - B. Impaired autoregulation may also develop in many situations including medication use, diabetes, and glaucoma.
 - C. The Baltimore Eye Survey demonstrated that young hypertensives had a lower prevalence of POAG than nonhypertensives, while older hypertensives with assumed impaired autoregulation had higher prevalence compared to nonhypertensives.
- VIII. Treatment of Systemic Hypertension May Increase Glaucoma Progression
 - A. Aggressive BP lowering in glaucoma patients may cause a drop in OPP and ischemic injury, which was also found to be a significant risk factor for glaucoma in large epidemiologic studies.
 - B. Treatment may lower BP below a level that can autoregulate, resulting in a drop in OPP and ischemic injury.
 - 1. The Thessaloniki Eye Study demonstrated an increase risk of enlarged cup-to-disc ratio with aggressive antihypertensive therapy.
 - 2. May divert blood from the vasculature supplying important capillary beds. While calcium channel blockers may increase OBF, studies have shown an increased incidence of glaucoma

with their use. Rotterdam follow-up¹⁹ study: 1.8 -fold higher risk of developing OAG if using calcium channel blockers.

- IX. How to Assess
 - A. Why does the patient have systemic hypotension?
 - 1. Overtreatment of systemic hypertension
 - 2. Poor hydration
 - 3. Impaired autoregulation: orthostasis, labile BP
 - 4. Natural phenotype: cold hands and feet, migraine, thin, young to middle-aged women
 - B. Measure circadian OPP
 - 1. To assess OPP over a 24-hour period, you need an ophthalmic sleep lab, which is available only in research settings.
 - 2. 24-hour IOP measurements
 - a. Ideally we would be able to calculate perfusion pressure throughout the 24-hour cycle. There is no currently available method of determining circadian variations in IOP in a routine clinical setting.
 - b. The triggerfish device, a contact lens device, measures fluctuations in ocular dimensions. It is cumbersome and expensive, creates extensive data that requires interpretation, and provides relative measures, but not actual IOP measures.
 - c. We can use surrogates for peaks of IOP not typically measured in the office by:
 - i. assessing supine IOP and/or
 - ii. performing a water drinking test.

The water drinking test was first introduced as a method to diagnose glaucoma and was abandoned since it was highly inaccurate. While the water drinking test is currently rarely performed, there has been recent interest in its ability to act like a stress test for the eye; it can provide useful information regarding outflow reserve, IOP instability, and peak IOP.

- 3. Ambulatory BP measurements: Most important part of the assessment
 - a. Work with a cardiologist or
 - b. Do it yourself: Medtronics or other reasonably priced unit
 - i. Measure mean diurnal and nocturnal BP
 - ii. Measure nocturnal dips
 - iii. Assess variability of BP
- 4. OCT angiography: No clear role
- 5. The usual suspects: OCT (nerve fiber layer and ganglion cell complex) and VF

- X. Can OPP Be Managed?
 - Lowering IOP is our best-studied mechanism for increasing OPP.
 - B. Work with primary care/cardiologist to coordinate and understand each other's concerns.
 - 1. Avoid extreme nocturnal dips.
 - Avoid nocturnal peak response of antihypertensives.
 - a. Avoid nighttime antihypertensives.
 - b. Prevent overtreatment of systemic hypertension.
 - c. Lower BP cautiously after prolonged hypertension.
 - d. Try to modulate extreme fluctuations of BP.
 - 3. Consider alternatives to calcium channel blockers in the treatment of hypertension.
 - C. Encourage patients to stay hydrated.
 - D. Consider alternatives to topical beta blockers in patients with systemic hypotension, encourage punctal occlusion and eyelid closure for 3 minutes after dosing topical beta blockers.
 - E. Unclear how interventions such as salt supplementation affect the various ocular beds

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Is Testing for Sleep Apnea Indicated?

Christopher Girkin MD

- I. Overview of Sleep Apnea
 - A. Types
 - 1. Obstructive
 - a. Most common
 - b. Caused by a blockage of the airway when soft tissue in the back of the throat collapses during sleep
 - 2. Central
 - a. Much less common
 - b. Unstable respiratory control center
 - B. Prevalence
 - 1. 18 million adults
 - 2. Adult prevalence: 3%-7% have OSA with daytime somnolence, but over 1/4 of individuals have multiple episodes of apnea on sleep studies.
 - 3. 2%-3% of kids (10%-20% with chronic snoring)
 - C. Risk factors
 - 1. Male
 - 2. Over 40
 - 3. Overweight
 - 4. Large neck size
 - a. 17 inches in men
 - b. 16 inches in women
 - 5. Large tongue or tonsil relative to jaw
 - 6. Family history of sleep apnea
 - 7. Nasal obstruction (deviated septum, allergies, sinus disease)

- D. Sociologic impact of sleep apnea
 - 1. Academic underachievement
 - 2. Poor work performance
 - 3. Increase in motor vehicle accidents
- E. Impact of sleep apnea on general health
 - 1. High blood pressure
 - 2. Stroke
 - 3. Heart failure, arrhythmia, infarction
 - 4. Diabetes
 - 5. Depression
 - 6. Exacerbation of ADHD
 - 7. Headaches
 - 8. Glaucoma
- II. Sleep Apnea and Glaucoma
 - A. Moderate association seen in the clinical studies and administrative databases
 - B. Mechanism unclear
 - 1. IOP: Evidence suggests IOP drops during apneic episodes.
 - 2. Hypoperfusion
 - 3. Hypoxia
 - C. No evidence that treating sleep apnea retards glaucoma progression
- III. Whom to Test for Sleep Apnea in the Glaucoma Clinic
 - A. All patients with signs and symptoms consistent with sleep apnea?
 - B. All patients who progress at low pressures?
 - C. All patients with normal-tension glaucoma?

A Disc Hemorrhage: What Does It Mean? What Should I Do?

C Gustavo De Moraes MD

- I. What Does the Literature Tell Us About Disc Hemorrhages?
 - A. Population-based studies
 - B. Clinical trials
 - C. Longitudinal studies
- II. What Are the Current Theories on the Pathogenesis of Disc Hemorrhages?
 - A. Vascular theory
 - B. Biomechanical theory
- III. What Is New in Terms of Imaging and Disc Hemorrhages?
 - A. Disc photography
 - B. OCT
 - C. OCT angiography

- IV. What Is the Relationship Between Disc Hemorrhages and IOP?
 - A. Does IOP lowering decrease the risk of disc hemorrhage?
 - B. Does IOP lowering decrease the rate of progression in eyes with disc hemorrhage?

Selected Readings

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Considerations for Medical Management

Ki Ho Park MD PhD

- I. Medical Management to Control IOP in Normal-Tension Glaucoma (NTG)
 - A. Evidence for IOP-lowering treatment in NTG
 - 1. Clinical trials
 - a. Collaborative Normal-Tension Glaucoma Study^{1,2} (CNTGS; 30% IOP reduction by medication, laser trabeculoplasty, or surgery): 80% survival in the treated arm and 40% survival in the control arm at 5 years
 - i. Beta-blockers and adrenergic agonists were not allowed to be used because of their potential cardiovascular effect.
 - ii. Prostaglandins were not available at the time of the study.
 - b. Early Manifest Glaucoma Trial³ (EMGT; 25% IOP reduction by laser trabeculoplasty plus betaxolol): an effective reduction in progression to about 50% in low-baseline-IOP group
 - 2. Retrospective long-term follow-up studies in Korea and Japan
 - a. 12-year follow-up study: Lower percentage reduction in IOP was a risk factor for progression in NTG. Upper tertile percentage IOP reduction (>22% reduction from baseline) group showed a greater cumulative probability of nonprogression than lower tertile percentage IOP reduction (<13% reduction from baseline) group.⁴
 - b. A pressure-dependent maintenance effect of the visual field was confirmed in progressive NTG patients followed up for over 15 years.⁵
 - B. Effect of IOP fluctuation on NTG progression
 - 1. Evidence for long-term fluctuation of IOP in prospective 5-year study⁶
 - a. In Japanese NTG patients with mean baseline IOP of 12.3 mmHg without treatment, estimated mean MD slope for 5 years was -0.33 dB/year.
 - b. Probability of glaucoma progression based on visual field or disc/peripapillary end points at 5 years was 66%.
 - c. Presence or history of disc hemorrhage, longterm IOP fluctuation, and greater vertical cup-to-disc ratio significantly contributed to progression.
 - 2. Habitual IOP peaks or 24-hour fluctuation⁷⁻⁹

- a. The mean peak IOP was significantly higher during nighttime phasing at home (15.8 ± 4.8 mmHg) compared with daytime phasing (12.8 ± 2.7mmHg, P = .0018) and clinic IOP measurements (11.8 ± 1.6 mmHg, P < .0001) by rebound tonometer. Following IOP phasing, a change in management occurred in 10 of 18 patients (56%).⁸
- b. The 24-hour range of IOP-related profile fluctuations in the NTG group was significantly larger than that of the nonglaucoma group (measured by contact lens sensor).⁹
- C. Choice of medication
 - 1. Prostaglandin analogue or prostamide¹⁰
 - 2. Brimonidine¹¹
 - 3. Others: topical carbonic anhydrase inhibitor, selective beta-blocker, nonselective beta-blocker, rho kinase inhibitor
 - 4. Results of survey in Canada and USA including glaucoma specialists (Canadian Glaucoma Society, American Glaucoma Society) and general ophthalmologists (Canadian Ophthalmological Society)¹⁰
 - a. Response rate: 19%-23%
 - b. 95% and 64% of the respondents were familiar with the CNTGS and the Low-pressure Glaucoma Treatment Study (LoGTS), respectively.
 - c. 68% responded that they would initiate treatment in mild-to-moderate NTG without waiting for documented disease progression.
 - d. 61% of the total surveyed and 50% of the glaucoma specialists felt that the LoGTS results had no impact on their usual clinical practice.
 - e. The first-choice topical drug for NTG: prostaglandin analogue (88% of respondents), brimonidine (10% of respondents), or betablocker (1%).
 - 5. Results of survey in Korean glaucoma specialists (Korean Glaucoma Society; unpublished data)
 - a. Response rate: 55% (117/212) in glaucoma specialists; 65% (17/26) in glaucoma fellows
 - b. 88% and 77% of the respondents were familiar with the CNTGS and the LoGTS, respectively.
 - c. 91% responded that they would initiate treatment in mild-to-moderate NTG without waiting for documented disease progression.

- d. 52% of the total surveyed and 51% of the glaucoma subspecialists felt that the LoGTS results had no impact on their usual clinical practice.
- e. The first-choice topical drug for NTG: prostaglandin analogue (76% of respondents), brimonidine (9%), beta-blocker (8%), or topical carbonic anhydrase inhibitor (6%).
- II. Considerations for Effect of Lifestyle on IOP
 - A. Exercise and yoga¹²
 - B. Sleeping habit and body posture^{7,13,14}
 - C. Playing wind instruments¹⁵
 - D. Meditation¹⁶
- III. Considerations for IOP-Independent Factors
 - A. Systemic hypotension and hypertension^{17,18}
 - B. Vascular and autonomic dysregulation¹⁹
 - C. Diet or dietary supplements^{20,21}
 - D. Excitotoxicity²²
 - E. Aberrant immunity²³
 - F. Low BMI¹⁸ and low CSF pressure²⁴

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Surgery for Normal-Pressure Glaucoma: Is There a Role for Minimally Invasive Glaucoma Surgery Before Filtering Surgery?

Leonard K Seibold MD

- I. Minimally Invasive Glaucoma Surgery (MIGS)
 - A. Advantages
 - 1. Microinsional, ab interno approach
 - 2. Augment physiologic outflow/inflow pathways
 - 3. At least modest efficacy
 - 4. Very high safety profile
 - 5. Rapid patient recovery
 - B. Variety of approaches
 - 1. Angle based
 - a. Goniotomy/trabeculotomy: Kahook dual blade, Trabectome, gonioscopy-assisted transluminal trabeculotomy, Trab360
 - b. Bypass stents: iStent, Hydrus
 - c. Canaloplasty: ab interno canaloplasty
 - Cyclophotocoagulation: Endoscopic cyclophotocoagulation (ECP), micropulse cyclophotocoagulation (MP-CPC)
 - 3. Subconjunctival: Xen gel stent
- II. Normal-Tension Glaucoma (NTG)
 - A. Progressive optic neuropathy with optic nerve damage and visual field loss with IOP consistently <21 mmHg
 - B. In the Collaborative Normal-Tension Glaucoma Study, IOP reduction of 30% or more stopped visual field progression in 80% of eyes.
 - C. With low pretreatment IOP, treatment goals are often <12 and sometimes below episcleral venous pressure (EVP).
- III. Filtering Surgery
 - A. Trabeculectomy is traditionally used to achieve ultra-low IOP targets in NTG.
 - B. Significant risks of trabeculectomy are even higher when targeting single-digit IOP.
 - 1. Hypotony maculopathy
 - 2. Serous/hemorrhagic choroidal effusions
 - 3. Flat anterior chamber
 - 4. Cataract progression
- IV. MIGS as First-line Surgical Treatment in NTG
 - A. Very limited published data on outcomes in this specific population

- B. Use of MIGS is very dependent on goal IOP.
- C. Angle-based procedures are unlikely to lower IOP below EVP. Mean postoperative IOP ranges: 13-16 mmHg.

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- D. ECP/MP-CPC are also capable of achieving IOP less than EVP, with risk of overtreatment.
- E. Xen gel stent is capable of achieving single-digit IOP, but more likely 12-15 mmHg.
- V. Phakic/Cataract Patients
 - A. Reasonable to consider MIGS procedure in combination with phacoemulsification if visually significant cataract is present
 - B. IOP reduction can be maximized by combining MIGS approaches—typically with inflow and outflow procedure.
 - C. Cataract is likely to progress after filtering surgery, requiring removal, which may lead to fibrosis/failure of bleb.
 - D. Some concern for decreased trabeculectomy success after cataract surgery
 - E. Depending on IOP goal, option for micropulse, Xen, or goniotomy/trabeculotomy
- VI. Pseudophakic/Clear Lens Patients
 - A. Some MIGS devices are not approved as standalone procedures.
 - B. Goniotomy/trabeculotomy, ECP, or combined approach can be considered.
- VII. When to Consider MIGS in NTG
 - A. IOP goal > 10 mmHg
 - B. High risk for filtration surgery (high myope, monocular, conjunctival scarring, prior complications)
 - C. Mild to moderate disease
 - D. Coexisting cataract
 - E. Treatment goal is medication replacement.
- VIII. When Not to Consider MIGS in NTG
 - A. IOP goal < 10 mmHg
 - B. Advanced disease approaching fixation
 - C. Elevated EVP

Congenital Anomalies Presenting With Anterior Segment and Glaucoma Findings

Ta Chen Peter Chang MD

- I. Evaluation of Congenital Anterior Segment Anomalies
 - A. Opaque cornea
 - 1. Congenital glaucoma
 - a. Primary
 - b. Secondary: ReNTALS (Rubinstein-Taybi, NF1, trisomy 18, Axenfeld-Rieger, Lowes, Sturge-Weber)
 - 2. Peters anomaly
 - 3. Congenital hereditary endothelial dystrophy
 - B. Unusual iris, chamber, angle
 - 1. Aniridia
 - 2. Pachyphakia, microcornea, angle closure (PMAC)
 - 3. Axenfeld-Rieger syndrome
 - 4. Ectropion uveae
 - C. Lens-related issues: aphakia/pseudophakia
 - D. Whole globe issue
 - 1. Microphthalmia
 - a. Simple: less common, 1/3 of microphthalmia cases, 50% associated with developmental anomalies
 - b. Complex: more common, 2/3 of microphthalmia cases, lack of secondary vitreous production
 - 2. Early-onset high myopia

- II. Is Glaucoma Present?
 - A. Childhood Glaucoma Research Network criteria
 - B. Role of OCT in diagnosing glaucoma
 - C. Role of ultrasound in diagnosing and monitoring glaucoma
- III. Management
 - A. Medications
 - B. Surgical
 - 1. Cornea clear, angle open: goniotomy and related ab interno angle surgery
 - 2. Cornea cloudy, angle open: ab externo trabeculotomy
 - 3. Cornea clear, angle closed: lensectomy, goniosynechialysis, ± endocyclophotocoagulation
 - 4. Cornea cloudy, angle closed: many options
- IV. Follow-up
 - A. Amblyopia
 - B. Genetic testing
 - C. Social support

Iridocorneal Endothelial Syndrome (ICE)

Lauren S Blieden MD

I. Pathogenesis

What we know (or don't know)

- II. Clinical Presentation
 - A. Demographics: How young can it present?
 - B. Cornea or iris or angle
 - 1. Corneal findings
 - 2. Iris findings
 - 3. Angle findings
- III. Management and Outcomes
 - A. Cornea or iris or angle
 - B. Corneal disease: Role of DSAEK vs. PKP
 - C. Glaucoma: Traditionally surgical management
 - 1. Trabeculectomy
 - 2. Tube shunt
 - 3. "Hybrid" procedures such as Ex-Press, Xen or PreserFlo
 - 4. Laser
 - 5. Angle surgery (MIGS)

Medication and Laser Therapy in Glaucoma Patients With Corneal Disease

Keith Barton MB BCh

The interaction between glaucoma and the cornea is complex:

- 1. Glaucoma medication can thin the cornea, causing a slight underestimation of IOP.
- 2. Ocular surface disease is common in the age group that develops glaucoma.
- 3. Glaucoma medication exacerbates ocular surface disease.
- 4. The preservatives in glaucoma medication exacerbate ocular surface disease.
- 5. Glaucoma medication can cause ocular surface disease (drug allergies and pseudopemphigoid).
- 6. Elevated IOP reduces the corneal endothelial cell count.
- 7. Glaucoma surgery may exacerbate corneal endothelial cell loss.
- 8. Early laser may protect the ocular surface and endothelium against the long-term effects of medical therapy.
- 9. In recalcitrant cases, laser may be an alternative to incisional surgery.

1. Glaucoma medication thins the cornea.

There is some evidence that glaucoma medication and especially prostaglandin agonists (PGAs) result in a small amount of corneal thinning. Harasymowycz et al reported that 6 weeks of travoprost treatment was associated with a 6.9- μ m reduction in central corneal thickness in a large prospective interventional case series.¹ The following year, Brandt et al reported that in the Ocular Hypertension Treatment study, the rate of central corneal thickness thinning was greater in patients who took PGA monotherapy, at 1.3 μ m per year, than in those who took beta-blocker therapy or were randomized to observation.² In neither study was the change sufficiently great to impact clinical decision making.^{1,2}

2. Ocular surface disease is common in the age group that develops glaucoma.

Dry eye syndrome and ocular surface disease are common with advancing age. In the Beaver Dam Eye Study, 14.4% of patients had dry eye symptoms, more commonly in women (17.0%) than men (11.1%) and increasing in frequency with age up to 70 years.³

3. The drugs in glaucoma medication exacerbate ocular surface disease.

Ocular surface disease is more common in patients with increasing glaucoma severity.⁴ Beta-blockers (timolol and levobunolol) reduce goblet cell density (GCD) more than do pilocarpine, brimonidine, or PGAs. Brimonidine seems more harmful than PGAs. Pilocarpine is more harmful than brimonidine but less than timolol. However, as pilocarpine is a sialogogue, it improves dry eye symptoms. PGAs, on the other hand, seem to increase GCD.⁵

4. The preservatives in glaucoma medication exacerbate ocular surface disease.

Benzalkonium chloride (BAK) is the preservative used in more than 70% of commercially available formulations used to treat glaucoma. BAK disrupts the lipid layer of the tear film, reduces GCD, and promotes inflammatory mediators.⁶ The GCDlowering drugs mentioned above lower GCD even more when given with BAK.⁵

5. Glaucoma medication can cause corneal and ocular surface disease.

Allergy to topical carbonic anhydrase inhibitors may develop after years on treatment. This typically causes eczema below the lower lid, rather than actual ocular surface disease, whereas brimonidine allergy may cause both. Typically, brimonidine allergy begins as chronic itching and erythema, often with a bulbar follicular reaction. When more advanced, lower lid ectropion may develop, with delayed tear drainage and a sticky eye, sometimes mistaken for infection. In advanced stages an impressive chronic granulomatous uveitis may occur. Levobunolol allergy may result in a red itchy eye with a papillary conjunctivitis.

Toxicity from excessive medication, both drugs and BAK, may result in dystrophic corneal epithelium and conjunctival fibrosis. In one large study, 28% of patients with biopsy-negative mucous membrane pemphigoid (pseudopemphigoid) were chronically medicated with multiple glaucoma drugs, to the extent that it was difficult to identify one particular culprit.⁷

6. Elevated IOP reduces the corneal endothelial cell count.

Glaucoma was the single biggest risk factor for corneal transplant failure in the Australian corneal graft registry,⁸ and elevated eye pressure results in a reduction in corneal endothelial cell count.

7. Glaucoma surgery may exacerbate corneal endothelial cell loss.

Typically, aqueous shunts, if improperly positioned, may exacerbate corneal endothelial cell loss, but endothelial cell loss is also associated with other types of glaucoma surgery,⁹ as evidenced by the recent market withdrawal of CyPass.

8. Early laser may protect the ocular surface and endothelium against the long-term effects of medical therapy.

The recent Laser in Glaucoma and Ocular Hypertension (LiGHT) Trial demonstrated clear benefits of selective laser trabeculoplasty over medical therapy in newly diagnosed, medication-naïve ocular hypertensive and glaucoma patients.¹⁰

9. In recalcitrant cases, laser may be an alternative to incisional surgery.

Cyclophotocoagulation (CPC) is not new, but there are now more options, either under development, or approved, than have previously been available, such as high frequency focused ultrasound, micropulse diode laser CPC, conventional transscleral diode laser CPC, and endoscopic diode laser CPC.

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Minimally Invasive Glaucoma Surgery Consideration in Patients With Diseases of the Cornea

John P Berdahl MD

- I. Concomitant Corneal Disease and Glaucoma Are Relatively Common
 - A. Incidence of glaucoma and corneal diseases
- II. Common Concomitant Diseases
 - A. Ocular surface disease: effects of glaucoma drops on the ocular surface
 - B. Fuchs dystrophy: concurrent vs. sequential surgery
 - 1. Risk of bleeding
 - 2. Risk of long-term steroid use
 - C. Full-thickness corneal disease
 - 1. Visualization challenges
 - 2. Long-term steroid use
- III. Summary and Conclusions

Why Does My Glaucoma Surgery Make the Cornea Fail, and Why Does My Cornea Surgery Make Glaucoma Worse?

Michael R Banitt MD

- I. Corneal Problems After Glaucoma
 - A. Delle
 - B. Infections: keratitis from sutures
 - C. Astigmatism
 - D. Corneal endothelial decompensation
- II. Corneal Edema After Glaucoma Surgery
 - A. Rates
 - 1. Trabeculectomy
 - 2. Tube shunts
 - B. Causes / theories
 - 1. Surgical insult
 - 2. Hypotony
 - 3. Disrupting natural flow inside anterior chamber
 - 4. Damage from tube
 - a. Direct damage from tube entry site
 - b. Damage near tube tip
 - c. Local jet/flow of fluid in and out of tube tip
- III. Corneal Edema After Glaucoma Surgery: What Can Be Done?
 - A. Trabeculectomy: Nothing specific. Avoid hypotony and shallowing of the chamber.
 - B. Tube shunts
 - 1. Same as above: Avoid hypotony and shallowing of the chamber.
 - 2. Perform gonioscopy. Tube should enter through the trabecular meshwork, not anterior to Schwalbe line.
 - 3. Insert the tube in the posterior chamber if pseudophakic.
- IV. Glaucoma Worse After Cornea Surgery?
 - A. Medically treated glaucoma
 - 1. Angle closure or reduced outflow
 - 2. Steroid response

- B. Surgically treated glaucoma, trab/tube
 - 1. Occlusion of outflow: Iris covering ostium of trab/tube tip
 - 2. Harder to control pressure if angle closure or reduced outflow
 - 3. Steroid response?
- C. Penetrating keratoplasty (PKP)
 - 1. Significantly distorted angle structures
 - 2. Angle closure that occurs at time of surgery or slowly increases over months
- D. Deep anterior lamellar keratoplasty (DALK): Distorted angle structures, but it should not develop peripheral anterior synechiae since no graft-host junction or shallowing of chamber
- E. Endothelial keratoplasty (EK)
 - 1. Pupillary block from air bubble on day of surgery
 - 2. Angle closure at time of surgery or slowly increases over months
 - 3. Steroid responses
- F. Keratoprosthesis
 - 1. Angle closure
 - 2. Steroid response
- G. What can be done if glaucoma is worse?
 - 1. Coordinate with cornea specialist regarding topical steroids
 - 2. Angle closure is difficult to treat; avoidance is best.
 - 3. Iris plugging trab or tube is often amenable to vitrectomy to open. Consider repositioning tube to posterior chamber or vitreous cavity.
- V. Cornea and Glaucoma Dilemma
 - A. When to trim a tube?
 - B. Too anterior
 - C. Too long
 - D. Affect a PKP
 - E. Affect an EK

Measuring IOP After Corneal Surgery

Thasarat Sutabutr Vajaranant MD

Corneal thickness and corneal pathologies such as corneal edema and scarring can influence all tonometry techniques. Specifically, Goldmann tonometry, the gold standard, is the most affected, whereas dynamic contour tonometer is the least affected by corneal properties. Awareness of the limitations of each tonometry technique used in surgically altered corneas is key to monitoring IOP and glaucoma in eyes after corneal surgery.

Table 1.

Pros and Cons	Corneal Considerations	
Pros	• Not best suited for most corneal diseases	
 Readily available Cons Sensitive to cornea changes: thickness, astigmatism, edema, or surface 	 For astigmatism, average vertical and horizontal readings Formula for CCT corrected not recommended ↑ CCT in DSEK does not cause false ↑ reading 	
Pros	• Affected by corneal thickness	
 Upright or supine position Objective measurements of variability and quality of readings (deviation <0.5) Cons Not readily available 	 Not affected by surface diseases Corneal edema and scar? Alternative method in KPro (on sclera) 	
Pros	• Affected by corneal thickness	
• Handheld	• Less affected by corneal edema	
 Disposable sleeve Cons Low accuracy outside normal IOP 	• Avoid diseased areas, but stay in the center for more accurate reading	
Pros	• Measures biomechanical properties	
• Noncontact	• Corneal hysteresis (viscous dampening)	
 Measures biomechanical properties Cons Not readily available 2-3 seconds to measure Affected by ocular pulse; multiple readings 	 Corneal resistance factor (corneal elastic response) Goldmann (IOPG) and corneal compensated (IOPCC) 	
	Pros • Readily available Cons • Sensitive to cornea changes: thickness, astigmatism, edema, or surface Pros • Upright or supine position • Objective measurements of variability and quality of readings (deviation <0.5)	

are recommended

Table 1. (continued)

Tonometry	Pros and Cons	Corneal Considerations					
Dynamic contour tonometer	Pros	• Least affected by corneal properties					
Principle: Contour matched	• Least affected by corneal properties						
• Close to manometry	 Measures ocular pulse amplitude (OPA) Measures reliability (Q >3) Cons Not readily available Time consuming 						
				• Needs cooperative patients			
				ICare	Pros	Affected by corneal thickness	
				Principle: ballastic/rebounding probe	• No anesthetic	 Less affected by corneal edema 	
				Higher reading compared to Goldmann	Disposable probe	• Avoid diseased areas, but stay in the cente	
	right reading compared to command	 Handheld Home tonometer Well tolerated in children 					
	Cons						
	• Not readily available						
Diaton	Pros	• No corneal contact					
Principle: Ballastic/rebounding probe	• Anesthetic not required	Affected by corneal thickness					
	• Handheld	 Measures scleral rigidity? 					
	• Well tolerated	Alternative method in KPro					
	Cons						
	Positioning technique						
	• Accuracy?						

Abbreviations: CCT, central corneal thickness; DSEK, Descemet-stripping endothelial keratoplasty; KPro, keratoprosthesis

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Cataract Surgery Pearls in Eyes With Pre-existing Trab or Tube Shunt

Thomas W Samuelson MD, Constance O Okeke MD

Thomas W Samuelson MD

I. Introduction

Phacoemulsification in eyes with pre-existing blebs requires attention to several important considerations, some related to cataract management, and others more specific to the glaucoma and/or the filtration bleb.

- II. Glaucoma-Specific Considerations
 - A. Intraocular pressure

Numerous studies have shown that phacoemulsification lowers IOP in most patients when the preoperative IOP is higher than the typical physiological range. However, it is important to realize that such studies do not apply to eyes with pre-existing blebs. Indeed, while phacoemulsification generally lowers IOP in eyes dependent on trabecular outflow, phaco often *increases* IOP in eyes with pre-existing blebs because of fibrosis and subsequent contraction of the bleb. This is one situation in which the favorable effect of cataract surgery on IOP is far less certain; indeed, it is more likely that IOP may increase postoperatively.

It is instructive to consider 3 preoperative scenarios in eyes with pre-existing filtration blebs, based on whether the IOP is in the desirable range.

- 1. Eyes with preoperative IOP within target range
 - a. Consider supplemental antimetabolite to help maximize the chance that the bleb will retain its favorable function and reduce the chance that bleb fibrosis will occur perioperatively.
 - b. If antimetabolite is considered, care should be exercised to avoid intraocular exposure to the antimetabolite.
- 2. Eyes with preoperative IOP that is higher than desired range
 - a. Again, consider supplemental antimetabolite.
 - b. Consider bleb needling or other filtration enhancement procedure.
 - c. Consider alternative procedure such as angle or additional trans-scleral glaucoma surgery.
- 3. Eyes in which preoperative IOP is lower than desired
 - a. In eyes with overfiltering blebs, phacoemulsification may result in favorable bleb contraction, resulting in more favorable IOP postoperatively. However, if IOP is low enough to cause a reduction in axial length or chorioretinal folds, there may be important implications for IOL calculations.

- b. Make cautious assessment of IOL calculations, especially with regard to axial length.
- c. Consider trab revision prior to phaco if necessary to obtain adequate IOL calculations in the setting of hypotonous maculopathy and clinically important axial length changes.
- B. Eyes with far advanced glaucoma

Consideration of a same-day pressure check several hours after cataract surgery should be considered in very advanced glaucoma or eyes with very high preoperative IOP.

- III. Phacoemulsification-Specific Considerations
 - A. Exfoliation: The implications of exfoliation are well known. The most important considerations include pupillary dilation and stability of the zonular-capsular complex.
 - B. Posterior synechias are common in patients with glaucoma, especially those with prior iridotomy or inflammation.
 - C. The fluidics of phacoemulsification are especially important in the presence of a thin-walled bleb. The pressure of the infusion/bottle height should be adjusted to avoid stressing or rupturing the bleb wall. Likewise, caution should be exercised with the eyelid speculum to avoid damaging the bleb. This includes avoiding the use of a suction speculum, which can create holes within the bleb wall.
 - D. Viscoelastic should be evacuated thoroughly at the end of the case.
 - E. Miotics may be considered to help control perioperative IOP and prevent pressure spikes.
- IV. Phacoemulsification in Eyes With Previous Tube Shunt
 - A. Corneal endothelium considerations
 - B. Tube positioning
 - C. Fluidics

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Constance Okeke MD

Introduction

Cataract surgeons should realize that cataract surgery for patients with prior glaucoma surgery can present challenges and points to consider that need to be addressed prior to, during, and after surgery. However, with the advent of newer technological advances, these patients have more options for achieving great visual outcomes, with improvements to aid in IOP control.

Background

Several factors can pose potential problems when previous trabeculectomy or tube shunts are present.¹ There are several anatomical concerns and reasons that surgeons should not approach these cases as your typical cataract surgery, especially relating to issues of uncontrolled IOP and failed prior glaucoma surgery.²

Preoperative Evaluation and IOP Options

In the preoperative evaluation, surgeons should understand that they will need to take extra time and steps with these patients, above the routine cataract evaluation. There are key points to review in the diagnostic testing. Also, there should be additional emphasis on the discussion with the patient to make sure proper expectations are set.

Several IOL choices are available for these glaucoma patients, but it does depend on the stage and extent of visual dysfunction.³

MIGS Options

The availability of various minimally invasive glaucoma surgery (MIGS) options has revolutionized my approach to these patients. MIGS in combination with cataract surgery in these patients should be considered if IOP is not controlled, if the patient still requires drops, and if the angle anatomy is amendable to it. There are several benefits, including aiding in reducing postoperative IOP spikes⁵ and further IOP reduction when the prior glaucoma surgery has failed.⁶ MIGS options that I have personally found beneficial in this situation include goniotomy, bypass stents, and canaloplasty.

Video case presentations of MIGS surgery performed in prior trabeculectomy and tube shunt patients will be offered.

Intraoperative Efforts, Recommendations, and Pearls

There are intraoperative techniques and suggestions that over the years have helped to improve outcomes for cataract surgery patients who have had prior glaucoma surgery. These suggestions involve the steps of anesthesia, the incision site around blebs, tube assessment and management, pupillary expansion, endothelial protection, anterior chamber depth management, hydrodissection pearls, cortical cleanup, viscoelastic removal, and wound closure.

I will present surgical videos highlighting alterations in the incision site to avoid bleb, tube management when too long, pupillary expansion with Malyugin ring, hydrodissection pearls, and wound closure.

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Cataract Surgery Combined With Minimally Invasive Glaucoma Surgery in Eyes With Controlled Glaucoma on Meds and History of Prior Incisional Glaucoma Surgery

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Paul J Harasymowycz MD

The presenter will review published literature and present personal data and cases on the topic of performing MIGS combined with phacoemulsification after failed glaucoma filtration surgery, including the beneficial effects on IOP and reduction of topical medication burden.

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Recent experience and literature investigating the MIGS procedures have had the interesting side effect of promoting cataract surgery alone as a "glaucoma procedure," consistently effective in lowering IOP. These studies have confirmed that cataract surgery by itself lowers IOP to a significant extent, while avoiding the risks and costs associated with minimally effective MIGS procedures. All available prospective and retrospective data on MIGS indicate that the long-term IOP from most of these procedures is typically in the mid-teens at best, often with the need for additional medications. Furthermore, long-standing experience has demonstrated that CE/IOL can cause scarring and failure of a pre-existing trabeculectomy. In the patient with moderate to advanced glaucoma in whom a trabeculectomy has already been required, adding a MIGS to the CE/IOL is a careful balance of weighing the risks to the benefits.

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Cataract Surgery Concerns in Short Eyes With Narrow Angles or Angle Closure

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Michele C Lim MD

Introduction

Cataract surgery in the short eye can be challenging because of anatomical issues such as short axial length and a crowded anterior chamber. However, we may find ourselves offering cataract surgery to this patient population with increasing frequency based on two factors. First, phacoemulsification in eyes with primary angle closure can open up the anterior chamber angle, and cataract surgery has been shown to lead to lower IOP in multiple studies.¹⁻⁴ Imaging studies using OCT demonstrate an increase of the angle-opening distance and trabecular iris space area, which are both objective measurements of the "openness" of angle structures, and these measurements may be linearly correlated with postoperative IOP lowering.⁴ Based on these studies, modern-day cataract surgery can be offered as a treatment option to our patients with primary angle closure (PAC) or primary angle-closure glaucoma (PACG).

Second, recent studies suggest that phacoemulsification has advantages in treating PAC,⁵ PACG, and acute angle closure³ in comparison to laser peripheral iridotomy. The EAGLE study (Effectiveness, in <u>Angle Closure Glaucoma</u>, of Lens Extraction)⁶ was a large multicenter randomized clinical trial in which patients with PAC and PACG were assigned to receive either cataract surgery or laser peripheral iridotomy. The study reported a significantly lower IOP, a higher self-reported health status score, and greater cost-effectiveness in the cataract surgery group than in the LPI group, suggesting that phacoemulsification may be a better treatment option.

With cataract surgery becoming a treatment choice that occurs earlier in the trajectory of care for a person with PAC, awareness of the challenges of an eye with narrow angles can help the surgeon prepare for a successful outcome.

Preoperative Considerations

- IOL calculation considerations: The effective lens position is harder to determine in short eyes because of a shallow anterior chamber, potentially steeper corneas, and the requirement for higher IOL powers that can cause bigger discrepancies, even with small misses in the predicted power of the lens.⁷ The Haigis, Holladay 2, Hoffer Q, and Barrett Universal II formulae may be considered for calculation of IOL power in short eyes.
- Ask about a history of trauma and look for signs of lens instability. Eyes that have suffered an acute angle closure (AAC) attack could have zonular instability. Kwon and Sung⁸ compared eyes post-AAC attack and subcategorized eyes by whether zonular instability was present or not during cataract surgery. Eyes with zonular instability had a smaller anterior chamber depth and greater lens vault mean measurement than eyes without zonular instability. In addition, the spherical equivalent was less hyper-

opic and axial length was longer in the former group. The conclusion was that AAC attack could be secondary to zonular instability, which would allow the crystalline lens to move forward, leading to pupillary block.

Intraoperative Considerations

- Due to shallow anterior chamber and/or a possible previous AAC attack, the corneal endothelium may be at risk for failure during phacoemulsification. The choice of a dispersive viscoelastic may be beneficial in protecting the endothelial cells. A narrow angle eye is at higher risk for iris prolapse during the case, and making the main corneal incision approximately 0.5 to 1 mm anterior to the limbus and creating a 2-step incision may help prevent it. Likewise, at the end of the case, suturing the incision closed can help avoid iris prolapse overnight should the patient inadvertently press on the eye. When making the capsulorrhexis, placing the cystotome needle (if you use one) on a viscoelastic syringe may be handy if the anterior chamber shallows during handling of the anterior capsule. If posterior pressure is suspected, IV acetazolamide or IV mannitol may be indicated.
- Goniosynechialysis combined with phacoemulsification may be considered for patients with peripheral anterior synechiae; however, the literature suggests that this technique does not lead to lower IOP in the postoperative period.^{9,10}

Postoperative Considerations

- Eyes with short axial length are at higher risk for malignant glaucoma. In a retrospective study of 20 eyes of 18 patients that developed malignant glaucoma after phacoemulsification, Varma et al¹¹ reported a mean axial length of 21.30 mm ± 1.40 mm and a mean refractive error of +3.11 ± 2.89 (SD). The authors point out that glaucoma in this setting may present after surgery with a myopic surprise, shallow anterior chamber, and high IOP.
- Eyes with nanophthalmos (≤20 mm) are at high risk for malignant glaucoma as well as other complications, such as uveal effusion, corneal edema, cystoid macular degeneration, and retinal detachment.¹²

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Surgical Pearls (with accompanying surgical videos)

- 1. Intraoperative flat anterior chamber: Techniques including single port, "dry" pars plana vitrectomy
- 2. Nanophthalmos or relative nanophthalmos: Tips for managing these very short axial length eyes, including intraoperative scleral windows, and performing various glaucoma procedures
- 3. Postoperative management of choroidal effusions and hemorrhage in eyes with short axial length
- 4. The role of endoscopic cycloplasty (ECPL) in the management of glaucoma with shallow anterior chamber
- 5. Goniosynechialysis and angle-based MIGS in angleclosure glaucoma

Managing Intraoperative Cataract Surgery Complications in Eyes With Glaucoma

Shakeel R Shareef MD and Pratap Challa MD

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I. Introduction

Cataract surgery complications in patents with glaucoma parallel complications seen in nonglaucoma patients, but they can occur in higher frequencies due to underlying pathology. Careful diagnosis and management techniques can improve outcomes.

- II. Preoperative Considerations
 - A. High-risk groups for complications: pseudoexfoliation syndrome, uveitic glaucoma, nanophthalmos, prior glaucoma surgery, traumatic glaucoma
 - B. Careful preop examination to evaluate for phacodonesis, zonular compromise, anterior or posterior synechiae, short axial length predisposing to uveal effusion or malignant glaucoma, and pupil dilation
- III. Intraoperative Considerations/Complications
 - A. Prior glaucoma surgery
 - B. Poor dilation
 - C. Zonular weakness
 - D. Phacodonesis
 - E. Iris prolapse
 - F. Posterior capsular rupture
 - G. Suprachoroidal hemorrhage
 - H. Pupillary block
 - I. Malignant glaucoma
 - J. Uveal effusion syndrome

- IV. Useful Intraoperative Techniques
 - A. Iris expansion devices to improve visualization
 - B. Capsule support (ie, hooks to stabilize bag support intraoperatively)
 - C. Capsule tension rings and segments to support lens placement and centration. In general, if 4 or more clock hours of poor zonular support is present, then you need to scleral fixate the ring or segment.
 - D. Intraoperative flat or shallow anterior chamber (AC)
 - 1. Low IOP: Check for wound leak or pinched tubing
 - 2. High IOP
 - a. Viscoelastic behind lens: Tilt lens gently to relieve posterior pressure.
 - b. Pupillary block
 - c. Suprachoroidal hemorrhage
 - d. Malignant glaucoma
 - e. Can address with intraoperatively with Chandler 3-step technique
 - E. Intraoperative hyperdeep AC
 - 1. Myopia
 - 2. Prior PPV
 - 3. Reverse pupillary block

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