Glaucoma 2015
Winning Strategies—Glaucoma in the 21st Century

Under Pressure®

Program Directors
James D Brandt MD and Joel S Schuman MD

In conjunction with the American Glaucoma Society

Sands Expo/Venetian
Las Vegas, Nevada
Saturday, Nov. 14, 2015

Presented by:
The American Academy of Ophthalmology
2015 Glaucoma Subspecialty Day Planning Group

David S Greenfield MD
Alcon Laboratories, Inc.: C
Allergan: C
Bausch + Lomb: C
Biometric Imaging: C
Carl Zeiss Meditec: S
Heidelberg Engineering: S
National Eye Institute: S
Optovue: S
Senju: C

Cynthia Mattox MD FACS
Allergan, Inc.: S
National Eye Institute: S
Transcend: S

Lucy Q Shen MD
None

Jody R Piltz-Seymour MD
Alcon Laboratories, Inc.: L
Allergan, Inc.: C
Forsight: C

Angelo P Tanna MD
Alcon Laboratories, Inc.: C,L
Apothex, Inc.: C
Sandoz: C
Watson Laboratories, Inc.: C

Shan C Lin MD
None

Anthony D Realini MD
Alcon Laboratories, Inc.: C
Lumenis, Inc.: S
Optovue: S
Reichert: C
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CME Credit

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.

2015 Glaucoma Subspecialty Day Meeting Learning Objectives
Upon completion of this activity, participants should be able to:
• Describe innovations in the diagnosis and management of glaucoma within their historical context
• Manage complex cases of glaucoma when other eye diseases are present
• Evaluate the current status of optic disc and retinal nerve fiber layer imaging and their role in diagnosing and managing glaucoma
• Demonstrate familiarity with current issues in medical and surgical therapy for glaucoma, both open-angle and angle-closure variants
• Understand the unique challenges and treatment options for infants and children with glaucoma
• Identify and manage glaucoma surgical complications

2015 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.

2015 Glaucoma Subspecialty Day Meeting CME Credit
The American Academy of Ophthalmology is accredited by the Accreditation Council for Continuing Medical Education to provide CME for physicians.

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

ABO Self-Assessment Credit
This activity meets the Self-Assessment CME requirements defined by the American Board of Ophthalmology (ABO). Please be advised that the ABO is not an accrediting body for purposes of any CME program. The ABO does not sponsor this or any outside activity, and the ABO does not endorse any particular CME activity. Complete information regarding the ABO Self-Assessment CME Maintenance of Certification requirements is available at http://abop.org/maintain-certification/part-2-lifelong-learning-self-assessment/cme/

NOTE: Credit designated as “self-assessment” is AMA PRA Category 1 Credit™ and is also preapproved by the ABO for the Maintenance of Certification (MOC) Part II CME requirements.

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

Scientific Integrity and Disclosure of Financial Interest
The American Academy of Ophthalmology is committed to ensuring that all 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 interest. The Academy has mechanisms in place to resolve all conflicts of interest prior to an educational activity being delivered to the learners.

The Academy requires all presenters to disclose on their first slide whether they have any financial interests from the past 12 months. Presenters are required to verbally disclose any financial interests that specifically pertain to their presentation.

Attendance Verification for CME Reporting
Before processing your requests for CME credit, the Academy must verify your attendance at Subspecialty Day and/or AAO 2015. In order to be verified for CME or auditing purposes, you must either:
• Register in advance, receive materials in the mail, and turn in the Final Program and/or Subspecialty Day Syllabus exchange voucher(s) onsite;
• Register in advance and pick up your badge onsite if materials did not arrive before you traveled to the meeting;
• Register onsite; or
• Scan the barcode on your badge as you enter an AAO 2015 course or session room.

CME Credit Reporting
Level 2 and Academy Resource Center, Hall B – Booth 2632 Attendees whose attendance has been verified (see above) at AAO 2015 can claim their CME credit online during the meet-
ing. Registrants will receive an email during the meeting with the link and instructions on how to claim credit.

Onsite, you may report credits earned during Subspecialty Day and/or AAO 2015 at the CME Credit Reporting booth.

**Academy Members:** The CME credit reporting receipt is not a CME transcript. CME transcripts that include AAO 2015 credits entered onsite will be available to Academy members on the Academy’s website beginning Dec. 10, 2015.

**NOTE:** CME credits must be reported by Jan. 13, 2016. After AAO 2015, credits can be claimed at www.aao.org.

The Academy transcript cannot list individual course attendance. It will list only the overall credits spent in educational activities at Subspecialty Day and/or AAO 2015.

**Nonmembers:** The Academy will provide nonmembers with verification of credits earned and reported for a single Academy-sponsored CME activity, but it does not provide CME credit transcripts. To obtain a printed record of your credits, you must report your CME credits onsite at the CME Credit Reporting booths.

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**Proof of Attendance**

The following types of attendance verification will be available during AAO 2015 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

Visit www.aao.org for detailed CME reporting information.
The American Glaucoma Society (AGS)
Subspecialty Day Lecture
OHTS at 20 Years: What We Have Learned
and What We Hope to Learn

Saturday, Nov. 14, 2015
11:49 AM – 12:19 PM

Michael A Kass MD

After earning his B.S. degree from the University of Michigan (1962) and his M.D. degree from Northwestern University Medical School in 1966, Dr. Kass completed an internship at Passavant Memorial Hospital in Chicago. He then completed a residency in ophthalmology and a fellowship in glaucoma in the Department of Ophthalmology at Washington University School of Medicine in St. Louis, working under the guidance of Dr. Bernard Becker. Dr. Kass served as an Assistant Professor of Ophthalmology and Director of the Glaucoma Service at Yale University School of Medicine. The returned to Washington University in 1975 as an assistant professor of Ophthalmology and was promoted through the ranks. He is currently the Bernard Becker Professor of Ophthalmology and Visual Sciences. Dr. Kass was the Chairman of the Department of the Department of Ophthalmology and Visual Sciences at Washington University 1999-2013, as well as the Ophthalmologist-in-Chief at Barnes-Jewish Hospital. Dr. Kass also serves as Senior Associate Dean for Human Research Protection.

Dr. Kass’ research has focused on Glaucoma, aqueous humor dynamics and clinical trials. His publications include original papers in major journals, including Journal of the American Medical Association (JAMA), Journal of Glaucoma, American Journal of Ophthalmology, Archives of Ophthalmology, and the Journal of Glaucoma. Dr. Kass’ work has been funded by the National Institutes of Health (NEI) for the past 30 years. He has served as an editor for several journals, including American Journal of Ophthalmology, The United States Pharmacopeia and The National Formulary, Investigative Ophthalmology and Visual Science, and the Journal of Glaucoma. He is currently Deputy Editor JAMA Ophthalmology and serves as a member in the National Eye Institute Intramural Clinical Trial Data and Safety Monitoring Committee.

Dr. Kass is a member of several professional and scientific societies Ophthalmology. He is also a member of the American Academy of Ophthalmology, American Board of Ophthalmology Associate for Research in Vision and Ophthalmology, and the American Glaucoma Society.
Faculty

Iqbal K Ahmed MD
Mississauga, ON, Canada
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Emory University Department of Ophthalmology

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Harkness Eye Institute
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Professor of Epidemiology  
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MD, Massachusetts Eye and Ear Infirmary

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Director, Shiley Eye Institute
Director, Hamilton Glaucoma Center
University of California, San Diego
# Glaucoma Subspecialty Day 2015:
## Winning Strategies—Glaucoma in the 21st Century

*In conjunction with the American Glaucoma Society*

**SATURDAY, NOV. 14**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Presenter</th>
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<tbody>
<tr>
<td>7:00 AM</td>
<td>CONTINENTAL BREAKFAST</td>
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<tr>
<td>8:00 AM</td>
<td>Welcome and Introductions</td>
<td>James D Brandt MD*</td>
</tr>
<tr>
<td>8:02 AM</td>
<td>American Glaucoma Society Introduction</td>
<td>David S Greenfield MD*</td>
</tr>
<tr>
<td>8:04 AM</td>
<td>Announcements</td>
<td>Joel S Schuman MD*</td>
</tr>
<tr>
<td>8:06 AM</td>
<td><strong>Section I: OCT Reboot</strong></td>
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</tr>
<tr>
<td>8:08 AM</td>
<td>Introduction</td>
<td>Jeffrey M Liebmann MD*</td>
</tr>
<tr>
<td>8:10 AM</td>
<td>Principles of OCT: Assessment of Scans and Scan Parameters</td>
<td>Donald L Budenz MD MPH* 1</td>
</tr>
<tr>
<td>8:16 AM</td>
<td>Clinical Case Study I: Early Glaucoma—Spin the Wheel</td>
<td>Kyung Rim Sung MD PhD 3</td>
</tr>
<tr>
<td>8:19 AM</td>
<td>Imaging for Glaucoma Diagnosis: Increasing Diagnostic Certainty With Structure-Function Correspondence</td>
<td>Robert N Weinreb MD* 4</td>
</tr>
<tr>
<td>8:27 AM</td>
<td>Clinical Case Study II: Artifact, Anomaly, or Disease—Betting on Red</td>
<td>Jullia A Rosdahl MD PhD 3</td>
</tr>
<tr>
<td>8:30 AM</td>
<td>Panel Discussion</td>
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<tr>
<td>8:35 AM</td>
<td>There’s Red on That Scan—Is It Real? Identifying Artifacts and Anomalies</td>
<td>Yao Liu MD 6</td>
</tr>
<tr>
<td>8:43 AM</td>
<td>Clinical Case Showing Progression on OCT</td>
<td>Gustavo DeMoraes MD*</td>
</tr>
<tr>
<td>8:46 AM</td>
<td>Place Your Bet: Progression by Imaging or Perimetry</td>
<td>Joel S Schuman MD*</td>
</tr>
<tr>
<td>8:54 AM</td>
<td>Panel Discussion</td>
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<tr>
<td>9:01 AM</td>
<td><strong>Section II: Glaucoma in Children</strong></td>
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<tr>
<td>9:03 AM</td>
<td>Introduction</td>
<td>Sharon F Freedman MD*</td>
</tr>
<tr>
<td>9:05 AM</td>
<td>Case Presentation: Infant Presenting With Buphthalmos</td>
<td>Teresa C Chen MD 11</td>
</tr>
<tr>
<td>9:10 AM</td>
<td>EUA and Beyond: Classification, Evaluation, and Treatment Planning</td>
<td>Sharon F Freedman MD* 12</td>
</tr>
<tr>
<td>9:13 AM</td>
<td>Fooled You! Mimics of Pediatric Glaucoma</td>
<td>Arif O Khan MD* 14</td>
</tr>
<tr>
<td>9:19 AM</td>
<td>Intervention Round One: Old and New in Angle Surgery</td>
<td>Allen Dale Beck MD* 15</td>
</tr>
<tr>
<td>9:25 AM</td>
<td>Case Presentation: Failed Angle Surgery</td>
<td>Lorna E Edmunds MBCHB 11</td>
</tr>
<tr>
<td>9:33 AM</td>
<td>Case Presentation: Aphakic or Pseudophakic Pediatric Glaucoma</td>
<td>David S Walton MD 11</td>
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<tr>
<td>9:35 AM</td>
<td>Glaucoma After Pediatric Cataract Extraction</td>
<td>Scott R Lambert MD* 18</td>
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<tr>
<td>9:47 AM</td>
<td>Case Presentation: Teen or Young Adult With Old Pediatric Glaucoma</td>
<td>Maria Papadopoulos MBBS 11</td>
</tr>
<tr>
<td>9:49 AM</td>
<td>All Grown Up: Teens and Adults Who Had Childhood Glaucoma</td>
<td>Alana L Grajewski MD 21</td>
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</tbody>
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* Indicates that the presenter has financial interest.
No asterisk indicates that the presenter has no financial interest.
<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Presenter(s)</th>
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<tbody>
<tr>
<td>9:55 AM</td>
<td>Conclusion and Discussion</td>
<td>Sharon F Freedman MD*</td>
</tr>
<tr>
<td>9:57 AM</td>
<td>Advocating for Patients</td>
<td>Thomas A Graul MD 22</td>
</tr>
<tr>
<td>10:02 AM</td>
<td>REFRESHMENT BREAK and AAO 2015 EXHIBITS</td>
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</tbody>
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**Section III: Playing Your Hand With Early Glaucoma—Current Dilemmas, Future Tools**
Moderators: Anthony D Realini MD*, Anjali M Bhorade MD

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<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Presenter(s)</th>
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<tr>
<td>10:40 AM</td>
<td>Introduction</td>
<td>Anthony D Realini MD*</td>
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<tr>
<td>10:41 AM</td>
<td>Case Presentation</td>
<td>Robert D Fechtner MD FACS* 24</td>
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<tr>
<td>10:43 AM</td>
<td>ARS Question – Audience Vote</td>
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<tr>
<td>10:44 AM</td>
<td>Case Presentation</td>
<td>Gail F Schwartz MD* 24</td>
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<td>10:46 AM</td>
<td>ARS Question – Audience Vote</td>
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<tr>
<td>10:47 AM</td>
<td>Case Presentation</td>
<td>Thasarat S Vajaranant MD* 24</td>
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<tr>
<td>10:49 AM</td>
<td>ARS Question – Audience Vote</td>
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<tr>
<td>10:50 AM</td>
<td>Case Presentation</td>
<td>Steven L Mansberger MD MPH* 24</td>
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<td>10:52 AM</td>
<td>ARS Question – Audience Vote</td>
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<tr>
<td>10:53 AM</td>
<td>Case Presentation</td>
<td>Carla J Siegfried MD* 24</td>
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<tr>
<td>10:55 AM</td>
<td>ARS Question – Audience Vote</td>
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<tr>
<td>10:56 AM</td>
<td>Surveying Your Cards:</td>
<td>Joshua D Stein MD MS 25</td>
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<td></td>
<td>Optimal Strategies to Monitor Glaucoma Suspects</td>
<td></td>
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<tr>
<td>11:03 AM</td>
<td>Doubling Down on Prostaglandin Analog Therapy:</td>
<td>Sanjay G Asrani MD* 28</td>
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<td>Single Agent, Fixed Combination, or SLT?</td>
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<tr>
<td>11:10 AM</td>
<td>The Disease That Never Sleeps:</td>
<td>Kaweh Mansouri MD* 30</td>
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<tr>
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<td>24-Hour IOP Monitoring Devices</td>
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<tr>
<td>11:17 AM</td>
<td>New Strategies to Beat the Odds:</td>
<td>Gary D Novack PhD* 31</td>
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<tr>
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<td>Controlled Delivery of IOP Medications</td>
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<tr>
<td>11:24 AM</td>
<td>Provider Roulette: New Models of Care</td>
<td>David S Friedman MD MPH PhD* 32</td>
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<tr>
<td>11:31 AM</td>
<td>ARS Question – Audience Vote</td>
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<tr>
<td>11:32 AM</td>
<td>Point – Counterpoint:</td>
<td>Ivan Goldberg MBBS FRANZCO* 34</td>
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<tr>
<td></td>
<td>Cashing Out Too Soon: Which Form of Noncompliance Poses a Higher Risk for Glaucoma Progression?</td>
<td></td>
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<tr>
<td></td>
<td>Noncompliance With Therapy</td>
<td>Kuldev Singh MD MPH* 35</td>
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<td>11:39 AM</td>
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<td>Cashing Out Too Soon: Which Form of Noncompliance Poses a Higher Risk for Glaucoma Progression?</td>
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**The American Glaucoma Society Subspecialty Day Lecture**

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<td>Introduction of the Lecturer</td>
<td>David S Greenfield MD*</td>
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<td>11:49 AM</td>
<td>OHTS at 20 Years: What We Have Learned and What We Hope to Learn</td>
<td>Michael A Kass MD 37</td>
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<td>David S Greenfield MD*</td>
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* Indicates that the presenter has financial interest.
No asterisk indicates that the presenter has no financial interest.
## Section IV: Spotlight on Angle-Closure Glaucoma

**Moderator:** Shan C Lin MD

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<td>Shan C Lin MD</td>
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<td>Setting the Stage: Laser Iridotomy—When, Where, and How Large?</td>
<td>Shan C Lin MD</td>
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<td>Point – Counterpoint: Gonioscopy Is the Gold Standard and Management Should Be Based on This Procedure</td>
<td>Tanuj Dada MD</td>
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<td>Point – Counterpoint: Anterior Segment Imaging Is More Precise and Represents the “True Angle”</td>
<td>Hiroshi Ishikawa MD</td>
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<td>Point – Counterpoint: Clear Lens Extraction Is Effective and Safe for Angle-Closure Glaucoma (and Insurance Should Pay for It as Primary Treatment)</td>
<td>Clement C Y Tham FRCS MBBS FCOphthHK*</td>
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<td>Point – Counterpoint: Clear Lens Extraction Has Not Been Sufficiently Demonstrated to Be Safe and Effective (and Insurance Shouldn’t Pay for It as Primary Treatment)</td>
<td>Tin Aung FRCS PhD*</td>
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<td>Point – Counterpoint: Laser Iridoplasty Is Effective for Treating Angle-Closure Glaucoma</td>
<td>Robert Ritch MD FACS*</td>
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<td>Point – Counterpoint: Laser Iridoplasty Is Not Effective for Treating or Preventing Angle-Closure Glaucoma</td>
<td>Paul J Foster FRCS*</td>
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<td>Audience Vote, Summary, and Panel Discussion</td>
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## Section V: Video Surgical Nightmares—Managing the Cards You Are Dealt

**Moderator:** Yvonne M Buys MD*

**Panelists:** Iqbal K Ahmed MD*, Lama A Al-Aswad MD, Keith Barton MD*, Marlene R Moster MD*, Thomas W Samuelson MD*

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<td>Yvonne M Buys MD*</td>
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<td>Scleral Flap Dehiscence</td>
<td>Marlene R Moster MD*</td>
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<td>Bleb Revision for Hypotony</td>
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<td>Lama A Al-Aswad MD</td>
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<td>Complications With iStent Insertion</td>
<td>Steven R Sarkisian MD*</td>
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<td>Ab Interno Ex-Press Shunt Removal</td>
<td>Davinder S Grover MD*</td>
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<td>Aqueous Misdirection During Trabectome</td>
<td>Douglas J Rhee MD*</td>
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<td>Management of Intraoperative Suprachoroidal Hemorrhage</td>
<td>Steven D Vold MD*</td>
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<td>4:13 PM</td>
<td>Conclusion</td>
<td>Yvonne M Buys MD*</td>
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### Section VI: Counting All of the Cards—Surgical Management of Glaucoma in Comorbidities

**Moderator:** Lucy Q Shen MD

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<td>Case Presentation: Glaucoma After Descemet-Stripping Endothelial Keratoplasty</td>
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<td>Special Considerations for Glaucoma Surgery in Patients After Corneal Transplantation: Options and Potential Pitfalls</td>
<td>Peter Andreas Netland MD PhD*</td>
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<td>Comanaging Corneal Transplant Patients After Glaucoma Surgery: Preserving Corneal Endothelium and Vision Rehabilitation</td>
<td>Samir A Melki MD PhD*</td>
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<td>Lucy Q Shen MD</td>
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<td>Anti-VEGF Agents and Their Effect on IOP</td>
<td>K Bailey Freund MD*</td>
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<td>Glaucoma Surgeries and Potential Complications in Patients Undergoing Frequent Anti-VEGF Injections</td>
<td>Malik Y Kahook MD*</td>
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<td>Case Presentation: Glaucoma in a Child With Chronic Anterior Uveitis</td>
<td>Gary N Holland MD*</td>
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<td>Inflammation and IOP: When Is Steroid Therapy Beneficial in Uveitic Glaucoma?</td>
<td>Gary N Holland MD*</td>
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<td>Surgical Options for Uveitic Glaucoma: Pros and Cons of Tube vs. Trabeculectomy</td>
<td>Anne Louise Coleman MD PhD*</td>
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<td>4:58 PM</td>
<td>ARS Question: What Surgery Would You Choose for This Patient?</td>
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<td>Closing Remarks</td>
<td>James D Brandt MD*</td>
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Principles of OCT: Assessment of Scans and Scan Parameters

Donald L Budenz MD MPH

Scan Quality
As with visual field testing for glaucoma, obtaining a test of good quality significantly affects the ability to diagnose glaucoma and glaucoma progression. Signal strength is the easiest quality measure to assess. One must know the acceptable range of signal strength for the OCT instrument that one is using. The signal strength or quality scores for the 3 major instruments in use in the United States are as follows: Signal strength ≥ 6 for Cirrus (Carl Zeiss Meditec, Inc.; Dublin, California, USA), quality score ≥ 20 for Spectralis (Heidelberg Engineering; Heidelberg, Germany), and signal strength index ≥ 30 for RTVue (Optovue, Inc.; Fremont, California, USA). One important pearl is that decreasing signal strength due to cataract, for instance, causes artifactual thinning of the retinal nerve fiber layer (RNFL) measurements.

However, it is critically important to look beyond just the signal strength or quality score to make sure a scan is of acceptable quality. One must also be on the lookout for algorithm segmentation failure, blocked signal, and eye movement or blinking artifacts. In addition to assessing scan quality, one must also be aware of patient or operator-related characteristics that can cause abnormalities on OCT that are unassociated with glaucoma, like incorrect scanning distance,1 head tilt,2 media opacities,3,4 drying cornea, non-glaucomatous optic neuropathies,5 moderate to high myopia,6,7 large and small optic nerves, optic nerve tilt,8 peripapillary atrophy,9 posterior vitreous detachment,10 and anatomic variations in the position of the superior and inferior nerve fiber bundles. These can cause “red disease,” or false positive results, or “green disease,” or false negative results.

Scan Parameters
There are currently 4 different structural parameters that may be useful in diagnosing and following glaucoma: RNFL thickness, ganglion cell layer (GCL) thickness, optic nerve head (ONH) parameters, and total macular retinal thickness (TRT). Each of these is measured differently with each instrument, so comparison of measurements across manufacturers in the same patient is not possible. RNFL thickness was the first parameter developed and validated for glaucoma diagnosis and for following glaucoma. Peripapillary RNFL cross-sectional thickness and area maps11 should both be evaluated to assess RNFL loss. Optic disc parameters are intuitive and familiar but do not replace in vivo and photographic assessment of the optic nerve head since things like optic disc hemorrhages and pallor cannot be assessed with OCT. Measurement of the GCL in the macula is a more recent parameter used in glaucoma assessment. Approximately 54% of the ganglion cell bodies reside in the macula, so measuring this layer perhaps does not provide as much information as RNFL or ONH measurements since the latter measure 100% of their structures. The boundary between the GCL and the underlying inner plexiform layer (IPL) is difficult to segment, so these two layers are measured together even though the inner plexiform layer is not affected in glaucoma. The instrument from Optovue includes the RNFL, GCL, and IPL in its Ganglion Cell Complex (GCC) measurement,12 while the instrument from Zeiss does not include the RNFL in its Ganglion Cell/Inner Plexiform Layer (GCIPL) measurements.13 As long as one is not comparing across OCT manufacturers, this has no impact on clinical judgment. Finally, the Spectralis OCT provides measurements of total retinal thickness (TRT) in the macula and relies on symmetry analysis comparing TRT between the right and left eyes of a patient and between upper and lower hemifields in the same eye.14 Good sensitivity and specificity have been reported for these OCT parameters.15,17

One of the challenges of having so many parameters that correlate with glaucoma and glaucoma progression is that they may not always agree, making interpretation difficult. If one adds up all of the available parameters on a Cirrus OCT glaucoma scan, for instance, there are over 30 to evaluate for each eye! Many of these correlate so highly with each other that they essentially say the same thing. Also, it is not uncommon in clinical practice to find eyes with thinning of ganglion cell parameters but normal RNFL parameters, or vice versa. New statistical algorithms are being developed to provide the clinician with a single probability score, as with older imaging technologies, to aid in interpretation of the large amount of data that is presented.18 It is likely that a similar probability parameter will be needed for progression as well.

Conclusions
The use of OCT has become common in the assessment of glaucoma and glaucoma progression. There is danger, however, in interpreting the OCT uncritically. The clinician must always assess the quality of the scan and look out for artifacts and clinical features of the patients that cause false positives and false negatives. Also, the OCT should not be interpreted in isolation; assessment of visual field and optic nerve visualization and/or stereo photography are essential. While accurate and precise measurements of structural loss in glaucoma are now possible with OCT, improved sensitivity to early glaucoma necessarily results in increased false positives. Overtreatment of patients can result, and clinicians must always keep in mind the causes of “red disease.”19

References


Clinical Case Study I: Early Glaucoma—Spin the Wheel

*Kyung Rim Sung MD PhD*

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Clinical Case Study II: Artifact, Anomaly, or Disease—Betting on Red

*Jullia A Rosdahl MD PhD*

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Clinical Case Showing Progression on OCT

*Gustavo DeMoraes MD*
Imaging for Glaucoma Diagnosis: Increasing Diagnostic Certainty With Structure-Function Correspondence

Robert N Weinreb MD

I. Early Glaucoma
   A. Visual fields perform poorly at detecting glaucoma damage.
      1. There can be substantial loss of optic nerve tissue while visual fields remain within statistically normal limits.
      2. Because they underestimate the amount of optic nerve loss early in the disease, visual fields also can underestimate the rate of progression.
   B. OCT performs well at detecting glaucoma damage.
      1. Progression (rates of change) can be better assessed by measuring retinal nerve fiber layer thickness, for example, than with visual fields.
      2. Rates of change have clinical relevance and are important to the patient as they are predictive of visual field loss, patient-reported disability, and difficulty doing everyday tasks.

II. Advanced Glaucoma
   A. Visual fields perform better than OCT for detecting progression.
   B. OCT generally is ineffective for detecting progression due to a floor effect.

Do Not Forget Optic Nerve Photographs

Limitations
- Need for a skilled examiner to assess change
- Poor agreement among examiners
- Do not provide a quantitative assessment of rates of change over time

Advantages
- Hemorrhages are better detected with photos than with any other examination
- Photographic technology is relatively stable over time. This is in contrast to OCT technology, which, despite its relatively short time of adoption, has already changed platforms several times and has not always been back-compatible and thus able to compare current tests with previous ones.

C. Combining structural and functional testing, rather than using either alone, improves monitoring of progression and measuring rates of change through all stages of disease.

Structural and Functional Testing Are Complementary.

Structural imaging (OCT) and functional testing (standard automated perimetry) are complementary.

They perform better and provide more useful information at different stages of the disease (ie, each can compensate for weakness in the other).

Combining structural and functional testing improves diagnosis and monitoring of progression.

IV. Using OCT and Visual Field Testing in Clinical Practice

Progression is key.
   A. OCT now has progression software that should be used, particularly in early stages of disease, by repeatedly testing over time.
      1. Fast progressors need frequent follow-up examinations and adjustments in therapy.
      2. In contrast, slow progressors do not need follow-up examinations as frequently and may not have loss of vision during their lifetime.
   B. Estimation of risk assessment is improved by incorporation of longitudinal testing (ie, do not rely on single-visit information for assessing risk).
   C. OCT and visual field measurements often disagree. In addition to their limitations and capabilities, they use different measurement scales (visual fields are logarithmic and OCT is linear) and also have different variability.

Rely more on OCT when visual field is normal. Once there is a repeatable visual field defect, both OCT and visual field are important.

D. Repeat testing as often as feasible, keeping in mind stage of disease, risk of progression, life expectancy, patient convenience, and payer rules.

More frequent testing provides better estimates of the rate of progression.

E. Beware of false positives.
   1. OCT provides so much information that there is a good likelihood of at least one of the parameters being a false positive (so-called “red disease”), particularly when looking at small areas to detect localized changes.
2. In contrast, global average thickness has very high reproducibility and accuracy for detecting progression with high specificity.\(^5\) With glaucoma average thickness, however, there is a trade-off as small, localized changes may be missed.

3. A change in average thickness of more than 5 µm should be considered very suspicious for indicating true glaucomatous progression, and confirmation should be sought with repeated testing.\(^9\)

References


There’s Red on That Scan—Is It Real? 
Identifying Artifacts and Anomalies

Yao Liu MD

I. Overview
A. Artifacts in retinal nerve fiber layer (RNFL) OCTs occur frequently.
   1. At least 1 artifact is reported to be present in 19.9%-46.3% of all scans.1,2
   2. Artifacts affect all OCT systems.3,4 Not unique or limited to any single OCT type or device.
B. Anomalies: Characteristics that may cause deviations from normative values, thereby limiting the ability of the normative database to provide useful comparisons
   1. Avoid mistakenly treating “red disease” rather than actual glaucoma5
   2. Areas flagged in red on the OCT report represent thin RNFL measurements relative to the normative database, but do not always represent glaucoma.
C. Imperative for clinicians to recognize artifacts and anomalies.
   1. Avoid mistakenly treating “red disease” rather than actual glaucoma5
   2. Areas flagged in red on the OCT report represent thin RNFL measurements relative to the normative database, but do not always represent glaucoma.

II. Types of Artifacts and Anomalies
A. Technical artifacts
   1. Scan centration: Most common artifact (reported in up to 30% of all scans)2,6,7
   2. Motion and blink artifacts8
   3. Software-related segmentation errors4 (ie, misidentification of RNFL borders)
   4. Truncation/missing scan areas
   5. Patient head tilt9
B. Patient characteristics
   1. Poor scan quality/low signal strength
      a. Media opacity or cataract10
      b. Dry or irregular ocular surface1
      c. Pupil size
         i. Does not significantly affect spectral-domain OCT (SD-OCT) measurements, but does affect those of time-domain OCT (TD-OCT)11
         ii. Dilation not required for SD-OCT systems.
   2. Limitations in normative database12
      a. Myopia with longer axial length
      b. Age
      c. Ethnicity
   3. Retinal characteristics
      a. Vitreomacular interface abnormality
         i. Partial posterior vitreous detachment (reported in 14.4% of all scans)2
         ii. Epiretinal membrane (reported in 9.4% of all scans)1
      b. Blockage of OCT signal
         i. Posterior vitreous detachment13
         ii. Retinal vasculature14
      c. Retinal atrophy (ie, prior retinal detachment, dystrophy, vascular occlusion)
      d. Prior panretinal laser photocoagulation15
      e. Large optic nerve head area16
      f. Peripapillary atrophy
      g. Cystoid macular edema
      h. Optic nerve drusen
      i. Optic nerve edema
      j. Non-glaucomatous optic nerve atrophy (ie, ischemic optic neuropathy, etc.)17
   4. Disc characteristics
      a. Large optic nerve head area16
      b. Peripapillary atrophy
      c. Staphyloma
      d. Myelinated nerve fiber layer
      e. Optic nerve drusen
      f. Optic nerve edema
      g. Non-glaucomatous optic nerve atrophy (ie, ischemic optic neuropathy, etc.)17
   5. Post-cataract surgery
      a. Artifactual increase in thickness may occur due to improved signal strength.10
      b. Multifocal lens implants may cause scan artifacts but have not been shown to cause significant differences in retinal thickness measurements in the macula.18
   6. Risk factors for increased prevalence of artifacts2
      a. Moderate to severe cataract (reduced signal strength)
      b. Visual acuity worse than 20/40 (may reduce ability to cooperate with scan)
      c. Advanced-stage glaucoma (lower signal-to-noise ratio)
      d. Diagnosis of primary open-angle glaucoma

III. Methods for Reducing Artifacts and Anomalies
A. Operator factors1
   1. Instruct patient on when to blink and use artificial tears to optimize ocular surface.
2. Ensure patient’s forehead is completely forward and stabilized in headrest.
3. Dim lighting to increase pupil size if needed.
4. Carefully adjust machine focus dials/knobs to optimize image quality.

B. Technological advancements
1. Eye tracking to reduce motion and blink artifacts
2. Automated scan registration for consistent scan circle placement in follow-up studies
3. Manual correction of software errors can be performed.
4. Future areas of development
   a. Automated scan circle placement
   b. Customization of scan circle diameter to compensate for large disc areas
   c. Improved segmentation algorithms
   d. Compensation for retinal vascular density

IV. Strategies for Recognizing Artifacts and Anomalies
A. Assessment of scan quality
   1. Signal strength recommendations (Scale/range is unique to each instrument.)
      a. 7 or higher: Cirrus or Stratus, Carl Zeiss Meditech
      b. 15 or higher: Spectralis, Heidelberg Engineering
      c. 40 or higher: RTVue, Optovue
      d. 50 or higher: Topcon 3D, Topcon
   2. Careful inspection of images included in the RNFL OCT report
      a. Scan circle centration assessment
      b. Individual B-scan evaluation to visually confirm appropriate segmentation

B. Consideration of possible retinal, optic nerve, and post-cataract surgery confounders
C. Evaluating RNFL OCT in eyes with myopia/high axial-length and in adults over age 80 years
   1. Recognition of limitations in normative database comparisons
   2. Software to overlay and compare changes in RNFL thickness scans over time: Guided progression analysis (Cirrus; Carl Zeiss Meditech)
   3. Comparison of right and left eyes for asymmetry
   4. Consider prioritizing visual field test results for glaucoma assessment.

References


I. Function Follows Structure
A. 40% to 50% of the optic nerve needs to be lost before there is a functional deficit (Sommer and Quigley).

B. 30% of the optic nerve needs to be lost with SAP (Quigley). Less with SWAP.

C. 17% to 20% needs to be lost before a visual field defect when retinal nerve fiber layer (RNFL) is measured with OCT (Wollstein).

II. Or Does It?
A. Corresponding coincident loss (Quigley & Harwerth; Harwerth; Harwerth & Anderson)
B. Short wavelength automated perimetry (SWAP) shows earlier loss than standard achromatic perimetry.
C. Pattern electroretinography (PERG) earlier loss than standard automated perimetry (SAP)
D. Quality of life is reduced even in patients with very early visual field loss.

III. What Is Happening?
A. There must be something that we’re missing, as an artifact of how we measure function in glaucoma.
B. There is essentially always an abnormality in the optic nerve and/or nerve fiber layer corresponding to visual field defects.

IV. Hypothesis
A. Structure and function change at similar times.
B. Current measurement technology limits our ability to detect functional abnormalities and change early in glaucoma.
C. Similarly, it is difficult to measure structural change late in the disease. In the region where both structure and function are changing, both change at similar times.

V. When RNFL is thick, we can measure structural loss only and no functional loss.
Past the tipping point we can measure both structural and functional loss, and once the RNFL measurement floor is reached we measure primarily functional loss.

Selected Readings


Case Presentations

Infant Presenting With Buphthalmos
*Teresa C Chen MD*

Failed Angle Surgery
*Lorna E Edmunds MBCHB*

Aphakic or Pseudophakic Pediatric Glaucoma
*David S Walton MD*

Teen or Young Adult With Old Pediatric Glaucoma
*Maria Papadopoulos MBBS*
EUA and Beyond: Classification, Evaluation, and Treatment Planning

Sharon F Freedman MD, Allen D Beck MD, and Ta Chen P Chang MD

I. Why do we need a new classification?
   A. Confusing and overlapping nomenclature such as “primary congenital” or “primary infantile glaucoma,” “developmental glaucoma,” and “juvenile glaucoma”
   B. Numerous rare conditions and syndromes associated with childhood glaucoma
   C. Lack of consensus on study definitions of glaucoma and glaucoma suspect

II. How do we make the diagnosis of glaucoma or glaucoma suspect in a child? Definitions (International Consensus)
   A. Childhood: Based on national criteria: < 18 years of age (United States); ≤ 16 years of age (U.K., Europe, UNICEF)
   B. Glaucoma: IOP-related damage to the eye (2 or more of the following required for diagnosis):
      1. IOP > 21 mmHg (discretion if examination under anesthesia data alone)
      2. Optic disc damage: Progressive increase in cup-disc ratio, cup-disc asymmetry of ≥ 0.2 when the optic discs are similar in size, or focal rim thinning
      3. Corneal findings: Haab striae or diameter ≥ 11 mm in newborn, > 12 mm in child under 12 months of age, > 13 mm any age
      4. Progressive myopia or myopic shift coupled with an increase in ocular dimensions out of keeping with normal growth
      5. Reproducible visual field defect consistent with glaucomatous optic neuropathy without other observable cause
   C. Glaucoma suspect: No IOP-related damage and at least one of the following:
      1. IOP > 21 mmHg on 2 separate occasions, or
      2. Suspicious optic disc appearance for glaucoma (ie, increased cup-disc ratio for size of optic disc), or
      3. Suspicious visual field for glaucoma, or
      4. Increased corneal diameter or axial length in setting of normal IOP

III. International Classification of Childhood Glaucoma (International Consensus)
   A. Primary childhood glaucoma
      1. Primary congenital glaucoma (PCG)
         a. Isolated angle anomalies (± mild congenital iris anomalies)
         b. Meets glaucoma definition (usually with ocular enlargement)
         c. Subcategories based on age of onset
            i. Neonatal or newborn onset (0-1 month)
            ii. Infantile onset (> 1-24 months)
            iii. Late onset or late-recognized (> 2 years)
         d. Spontaneously arrested cases with normal IOP but typical signs of PCG may be classified as PCG.
   2. Juvenile open-angle glaucoma (JOAG)
      a. No ocular enlargement
      b. No congenital ocular anomalies or syndromes
      c. Open angle (normal appearance)
      d. Meets glaucoma definition
   B. Secondary childhood glaucoma
      1. Glaucoma associated with nonacquired ocular anomalies
         a. Includes conditions of predominantly ocular anomalies present at birth that may or may not be associated with systemic signs
         b. Meets glaucoma definition
         c. List common ocular anomalies (eg, Axenfeld-Rieger anomaly/syndrome, Peters anomaly/syndrome, aniridia, ectopia lentis without systemic association, etc.)
      2. Glaucoma associated with nonacquired systemic anomalies
         a. Includes conditions of predominantly systemic disease present at birth that may be associated with ocular signs
         b. Meets glaucoma definition
         c. List common systemic syndrome or disease (eg, Down syndrome, Marfan and other connective tissue disorders, Lowe syndrome and other metabolic disorders, neurofibromatosis, Sturge-Weber, and other phacomatoses, etc.)
      3. Glaucoma associated with acquired condition
         a. Meets glaucoma definition after the acquired condition is recognized. An acquired condition is one that is not inherited or present at birth but that develops after birth.
b. Glaucoma developing after cataract surgery is excluded from this category to highlight its frequency and differences from other conditions in the acquired condition category.

c. List common acquired conditions (eg, uveitis, trauma, steroid-induced, tumors, ROP, post non-cataract surgery)

d. Based on gonioscopy results:
   i. Open-angle glaucoma (≥ 50% open) or
   ii. Angle-closure glaucoma (< 50% open or acute angle closure)

4. Glaucoma following cataract surgery
   a. Meets glaucoma definition after cataract surgery is performed and subdivided into three categories:
      i. Congenital idiopathic cataract
      ii. Congenital cataract associated with ocular anomalies / systemic disease (no previous glaucoma)
      iii. Acquired cataract (no previous glaucoma)

b. Based on gonioscopy results:
   i. Open angle glaucoma (≥ 50% open) or
   ii. Angle-closure glaucoma (< 50% open or acute angle closure)

IV. Evaluation of the Child With Known or Suspected Glaucoma

A. Consider the whole picture: ocular and systemic features, family history, etc.

B. Don’t forget the office visit (visual behavior, anterior/posterior segment …)
   1. Tonometry in the office: Plethora of instruments; special role of rebound?
   2. Imaging and visual fields can help in the cooperative child.
   3. Extra tools: CCT, diurnal curve and home tonometry(?)

C. When to proceed with examination under anesthesia?
   1. The uncooperative child
   2. When surgery will definitely be needed
      a. Surgical planning: IOP, central corneal thickness (CCT), gonioscopy, slitlamp anterior segment examination
      b. Special considerations: Is the view adequate for goniosurgery? Are there obstacles to surgery in the angle (eg, adhesions, peripheral anterior synechiae, etc.)?
      c. Special considerations: Is the eye amenable to glaucoma drainage implant? Consider axial length, scarring / exposure / anterior chamber adequacy, etc.

Selected Readings


I. Introduction

Primary congenital glaucoma (isolated goniodysgenesis) is often a straightforward diagnosis. Classic phenotypic features include increased measured IOP, corneal haze/scarring with associated astigmatism, corneal enlargement (megalocornea) with globe enlargement (buphthalmos) and associated myopia, Descemet membrane breaks (Haab striae), iris atrophy, and optic nerve cupping. These findings also occur in early developmental glaucomas. However, although the diagnosis of primary congenital or early developmental glaucoma is often straightforward, similar phenotypic features can occur in other pediatric conditions, which are thus sometimes mistaken as early childhood glaucoma. Ophthalmologists who care for children with glaucoma must always keep in mind such potential mimics of pediatric glaucoma.

II. Increased Measured IOP

A. Cooperation issues
B. Corneal issues

III. Corneal Haze

A. Mucopolysaccharidoses: multiple genetic causes
B. Congenital hereditary endothelial dystrophy: recessive mutations in
   SLC4A11 (Online Mendelian Inheritance in Man [MIM] *610206)

IV. Megalocornea

A. Primary megalocornea: X-linked form from mutation in
   CHRDL1 (MIM *300350) and other forms
B. Primary megalocornea with zonular weakness and secondary lens-related glaucoma: recessive mutations in LTBP2 (MIM *602091)

V. Posterior Corneal Breaks

A. Birth trauma
B. Primary corneal ectasia/fragility (brittle cornea syndrome): recessive mutations in ZNF469 (MIM *612078) or PRDM5 (MIM *614161)

VI. Optic Nerve Head Cupping

A. Physiologic
B. Congenital malformations

Selected Readings

Intervention Round One: Old and New in Angle Surgery

Allen Beck MD

I. Old Angle Surgeries
   A. Goniotomy: Developed in the 1930s by Barkan
   B. Trabeculotomy: Standard technique with metal probes developed in the 1960s (Harms most commonly used)

II. New Angle Surgeries
   B. Circumferential microcatheter trabeculotomy: iTack microcatheter developed for canaloplasty used for trabeculotomy in the last 10 years.

III. Old Angle Surgeries
   A. Advantages: Familiar techniques, very long follow-up reported
   B. Disadvantages: Repeat surgery necessary for 15%-40% of cases as only 1/3 to 1/2 of the angle is treated.

IV. New Angle Surgeries
   A. Advantages: Can treat the entire angle with one procedure with better reported outcomes than old angle surgeries.
   B. Disadvantages: More time consuming and technically challenging than old angle surgeries. Microcatheter approach is more expensive than the suture technique. Subretinal suture misdirection has been reported with the suture technique. Follow-up duration is not as long as old angle surgeries.

References
Children with primary congenital / infantile glaucoma in phakic eyes in whom goniotomy and/or trabeculotomy have been unsuccessful pose a difficult management dilemma. None of the commercially available, minimally invasive approaches are options; although in the future, translimbal filtering and/or suprachoroidal implants may be considerations for these patients. Currently, most surgeons managing pediatric glaucomas will choose either trabeculotomy with mitomycin C (MMC) or aqueous shunting procedures as their next intervention (with occasional exceptions in which a cyclodestructive procedure might be chosen). Both approaches have success rates in the range of 60% to 80% at about 2 years, with lower and higher outliers reported in the literature. However, most studies include a mixture of glaucomas, making it difficult to ascertain the likelihood of success with primary congenital / infantile glaucoma in phakic eyes.

Trabeculotomy with MMC in patients under age 1 or possibly 2 years has been noted to have higher failure rates. Trabeculotomy in the setting of younger children is complicated by the inherent limitations with postoperative manipulations that would otherwise reduce immediate postoperative complications and increase long-term success. These would typically include early postoperative wound manipulation with digital pressure, suture lysis or removal, and wound-healing modulations with supplemental 5-fluorouracil injections; although one series incorporated both adjunctive measures. When successful, some series found that children with filtering blebs may be at greater risk of blebitis and bleb-related endophthalmitis than adults.

Aqueous shunts frequently necessitate subsequent procedures to manage tube-related complications, such as tube-cornea touch, tube erosion, and tube retraction. Pupillary distortion can occur even with tubes that are apparently well positioned in the anterior chamber angle. Although presumptively less frequent than after trabeculotomy with MMC, endophthalmitis has been reported after aqueous shunt procedures, especially in the context of tube erosion.

Trabeculotomy with MMC is a reasonable first option in phakic children older than age 1 or possibly 2 years whose families grasp the long-term risk of bleb-related infection and are capable of presenting immediately upon recognition of suggestive symptoms. Broader MMC application with fornix-based conjunctival flaps should be considered, based on the clinical impression that this approach produces thicker, lower-profile blebs, which are presumably less prone to late-onset leaks and infection.

In younger children and/or in those lacking sufficient family / social support to ensure a timely response to possible blebitis, aqueous shunt surgery should be the initial procedure after goniotomy and/or trabeculotomy failure. With this approach, families and surgeons must be prepared for the likely need for additional interventions to deal with tube-related complications.


Glaucoma After Pediatric Cataract Extraction

Scott R Lambert MD

I. Incidence
15%-30% after cataract surgery during infancy; rare after cataract surgery in children > 6 months of age

II. Proposed Etiology
A. Cataract surgery interferes with the maturation of the trabecular meshwork.
B. “Collapse” of the immature angle after cataract surgery

III. Proposed Risk Factors
A. Cataract surgery during infancy
B. Reoperations
C. Nuclear cataracts
D. Persistent fetal vasculature
E. Microphthalmia
F. Chronic inflammation and retained lens material
G. Bilateral cataracts

IV. Proposed Protective Factors
IOL implantation

V. Randomized Clinical Trials: Infant Aphakia Treatment Study
114 infants (1-6 months of age) randomized to undergo unilateral cataract surgery with or without IOL implantation. Children followed to age 5 years.
A. The risk for glaucoma 4.8 years after cataract surgery was 17%.
B. The risk for glaucoma + glaucoma suspect 4.8 years after cataract surgery was 31%.
C. Risk of glaucoma was the same with or without IOL (hazard ratio [HR], 0.8, $P = .62$)
D. Younger age at surgery increased the risk for glaucoma (HR, 3.2).
E. Smaller corneal diameter increased the risk for glaucoma + glaucoma suspect (HR, 2.5). However, corneal diameter and age were highly correlated ($r = 0.65$).
F. Glaucoma was predominantly open angle (19 of 20 cases; 95%).

VI. Observational Cohort Studies: IOLunder2 Study
221 children (< 2 years) underwent cataract surgery (131 bilateral, 90 unilateral) in the UK/Ireland (2009-2010). Median age at cataract surgery was 2.0 months. Postoperative follow-up: 1 year. IOL implantation was performed in 104 children.
A. Cumulative incidence of glaucoma: 13%
B. All patients who developed glaucoma underwent cataract surgery < 6 months of age.
C. Microphthalmia was an independent predictor of glaucoma with unilateral (OR, 12.1), but not bilateral cataracts.

VII. Retrospective Studies
A. International Meta-analysis
470 infants underwent cataract surgery at a median age of 3.0 months (pooled data from 7 centers). Median postoperative follow-up: 6 years.
1. Cumulative incidence of glaucoma: 17%
2. Mean time to glaucoma diagnosis after cataract surgery: 4.3 years (interquartile range [IQR], 1.2-6.3 years)
3. Primary IOL implantation was protective against glaucoma (HR, 0.10). However, pseudophakic children had surgery at older age (3.7 vs. 2.8 months, $P < .001$).
4. Additional intraocular procedures increased risk of glaucoma (HR, 2.52).
5. Cataract surgery ≤ 4 weeks increased risk of glaucoma (HR, 2.10).
6. No difference in risk of glaucoma after unilateral or bilateral cataract surgery

B. Melbourne Study
1. Cumulative incidence of glaucoma: 32%
2. Mean time to glaucoma diagnosis: 7.2 years (IQR, 2.0-9.5 years)
3. Age at cataract surgery was the only factor independently associated with increased risk of glaucoma.
4. Risk of glaucoma: 60% after cataract surgery during first month of life

5. Risk of glaucoma was higher in children undergoing cataract surgery ≤ 3 months (HR, 1.88).

6. No child developed glaucoma when > 7 months of age at time of cataract surgery.

C. Emory Longitudinal Study

37 infants (< 7 months) underwent cataract surgery (26 bilateral, 11 unilateral) by the same surgeon (1988-2010). Median age of cataract surgery was 2.0 months. Median postoperative follow-up: 7.9 years (range: 3.2-23.5 years).

1. Cumulative incidence of glaucoma: 14.5%
2. Estimated probability of an eye developing glaucoma by age 10 years: 19.5%
3. Estimated probability of an eye developing glaucoma or glaucoma suspect by age 10 years: 63%.

VIII. Conclusions

A. Cataract surgery should be deferred to at least 4 weeks of age.

B. The risk of glaucoma is lower if cataract surgery is delayed to age 2-3 months; however, this may compromise visual outcome.

C. The optimal time to perform cataract surgery to minimize risk of glaucoma without compromising visual outcome has not been established.

References


Available Tests for Disease-Causing Glaucoma Genes in 2015: From genetests.org

<table>
<thead>
<tr>
<th>Disease</th>
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<tr>
<td>Primary open-angle glaucoma (POAG)</td>
<td>Myocilin (MYOC)</td>
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<td>WDR36</td>
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<td>POAG with nail-patella syndrome</td>
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<td>Peters plus</td>
<td>CYP1B1</td>
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There are anterior segment dysgenesis panels that include glaucoma genes: B3GALTL, BCOR, BMP4, COL4A1, CYP1B1, FOXC1, FOXE3, FRAS1, FREM2, GRIP1, HCCS, MFRP, OTX2, PAX6, PITX2, PITX3, SMOC1, SOX2, STRA6, VAX1, VSX2.

Or, one can screen for known eye-related genes: ABCA4, ABHD12, ADAM9, AH1, AIP1, ALMS1, ARL13B, ARL6, ATP13A2, B3GALTL, BBS1, BBS10, BBS2, BBS4, BBS5, BBS7, BBS9, BCOR, BEST1, BMP4, C1QTNF5, C2orf71, C5orf42, C8orf37, CA4, CAVP4, CACNA1F, CACNA2D4, CC2D2A, CDH23, CDH3, CDHR1, CEP290, CEP41, CERKL, CHM, CIB2, CLN3, CLN5, CLN6, CLN8, CLRN1, CNGA1, CNGA3, CNGB1, CNGB3, CNNM4, COL11A1, COL11A2, COL2A1, COL4A1, COL9A1, COL9A2, CRB1, CRX, CTSD, CYP1B1, DFN3B31, DHDDS, EFEMP1, ELOVL4, EYS, FAM161A, FLVCR1, FOXC1, FOXE3, FRAS1, FREM1, FREM2, FSCN2, FZD4, GNAT1, GNAT2, GPR143, GPR179, GPR98, GRIP1, GMR6, GRN, GUA1A, GUA1B, GUCY2D, HARS, HCCS, IDH3B, IMPDH1, IMPG2, INV3, IQC1B1, KCN1J3, KCNV2, KCDT7, KIF7, KLH17, LCA5, LRA, LRT13, LRP5, LTF1L1, MAK, MERTK, MRN2, MFRP, MFS5D, MK5S, MKN1, MYO7A, MYOC, ND, NPHP1, CYP1B1, NPHP4, NR2E3, NRI, NYX, OCA2, ODF1, OPA1, OPA3, OTX2, PAX6, PCDH15, PDE6A, PDE6B, PDE6C, PDE6G, PDE6H, PDZD7, PEX7, PHYH, PTPNM3, PITX2, PITX3, PLA2G5, PPT1, PRCD, PROM1, PRP2, PRP6, PRPE8, PRPH2, RAX2, RB3P3, RBP4, RD3, RDH12, RDRH5, RGR, RGRG, RGS9B, RHO, RIMS1, RLBP1, ROM1, RP1, RP2, RP9, RPE65, RPR, GRPRIP1, RPRIP2, RS1, SAG, SDCCAG8, SEMA4A, SLC2A4, SLC45A2, SMOC1, SNRNP200, SOX2, SPATA7, STRA6, TCTN1, TCTN2, TCTN3, TIMM8A, TIMP3, TMEM126A, TMEM216, TMEM237, TMEM67, TOPORS, TPI1, TRIM32, TRPM1, TSPAN12, TTC21B, TCT8, TULP1, TYR, TYPRL1, UNC119, USH1C, USH1G, USH2A, VAX1, VCAN, VSX2, WDP, WT1, ZNF423, ZNF513.
All Grown Up: Teens and Adults Who Had Childhood Glaucoma

Alana L Grajewski MD

I. Original Diagnosis: The Baby Album
   A. Primary congenital glaucoma
   B. Glaucoma associated with non-acquired systemic disease or syndrome
   C. Glaucoma associated with non-acquired ocular anomalies

II. Mom Says I Am Special
   A. Monocular/amblyopia: Unilateral glaucoma
   B. Consequence of initial treatment: Corneal changes and more
   C. Puberty: More than a hormonal storm

III. The Constant Reminders
   A. Monocular/amblyopia: Unilateral glaucoma
   B. Consequence of initial treatment: Corneal changes and more
   C. Puberty: More than a hormonal storm

IV. Life Goes On
   A. When glaucoma reoccurs: Management and monitoring
   B. Genetic consideration and counselling

Selected Readings

**2015 Advocating for Patients**

*Thomas A Graul MD*

Ophthalmology’s goal in protecting quality patient eye care remains a key priority for the Academy. All ophthalmologists should consider their contributions to the following three funds as (a) part of their costs of doing business and (b) their individual responsibility in *advocating for patients and their profession*:

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

Your ophthalmologist colleagues serving on Academy committees—the Surgical Scope Fund Committee and the Secretariat for State Affairs and the OPHTHPAC Committee—are committing many hours on *your* behalf. The Secretariat for State Affairs is collaborating closely with state ophthalmology society leaders to protect *Surgery by Surgeons* at the state level. Meanwhile, the OPHTHPAC Committee is hard at work identifying congressional advocates in each state to maintain close relationships with federal legislators in order to advance ophthalmology and patient causes. Both groups’ ultimate goals are to ensure robust funds for both the SSF and the OPHTHPAC Fund so that they are able to (a) protect quality patient eye care, (b) protect ophthalmology practices from payment cuts, (c) reduce burdensome regulations, and (d) advance the profession by promoting funding for vision research and expanded inclusion of ophthalmology in public and private programs.

These committed ophthalmologists serving on your behalf have a simple message to convey:

> “We also need you”!

- We need you to contribute to each of these 3 funds.
- We need you to establish relationships with state and federal legislators.
- We need you to help us protect quality patient eye care and the profession.

**Surgical Scope Fund**

The Surgical Scope Fund (SSF) provides grants to state ophthalmology societies to support their legislative, regulatory, and public education efforts to derail optometric surgery proposals that pose a threat to patient safety, quality of surgical care, and surgical standards. Since its inception, the *Surgery by Surgeons* campaign—in partnership with state ophthalmology societies and with support from the SSF—has helped 32 state/territorial ophthalmology societies reject optometric surgery proposals.

As of July 1, 2015, the Secretariat for State Affairs, in collaboration with the California Academy of Eye Physicians and Surgeons (CAEPS) and the California Medical Society, continues to battle an onerous optometric surgery scope of practice bill (SB 622) in the Golden State. The Secretariat has reached out to all ophthalmology subspecialty society partners to help in this effort, and several have stepped up to the plate. In addition, ophthalmology leaders at California academic institutions have played a critical role by voicing their concerns about the California surgery bill and the impact it would have on quality eye care for patients. A June 24 op-ed in the *San Francisco Examiner* aptly focused on these leaders’ concerns with its headline, “Quality surgical eye care ensured through training.” CAEPS has benefitted from contributions to the SSF, having received significant support from the fund.

Other state ophthalmology societies have also benefitted from SSF distributions in 2015 and were able to successfully implement patient safety advocacy campaigns to defeat attempts by optometry to expand its scope of practice to include surgery. The Texas Ophthalmological Association was successful in its patient advocacy and public education efforts to defeat three different optometric-backed surgical scope expansion bills in the Texas state legislature.

In addition, the Academy supported the Alaska Society of Eye Physicians and Surgeons in opposing optometric surgery scope legislation that posed a threat to patient surgical care. If enacted, the optometric surgery bill would have authorized optometrists in Alaska to perform surgery with lasers, scalpels, and needles, and to perform other surgical procedures. The legislation would also have allowed optometrists to perform all injections except intravitreal and to prescribe any controlled substances. Thanks to an effective *Surgery by Surgeons* advocacy campaign, with support from the SSF, this legislation died in committee. The Alaska state legislature adjourned for the year on April 27.

The Academy relies not only on the financial contributions to the SSF from individual ophthalmologists and their business practices, but also on the contributions made by ophthalmic state, subspecialty, and specialized interest societies. The *American Glaucoma Society* has been an ongoing contributor to the Surgical Scope Fund, and the Academy thanks the society leadership for its support.

**OPHTHPAC® Fund**

OPHTHPAC is a crucial part of the Academy’s strategy to protect and advance ophthalmology’s interests in key areas including physician payments from Medicare as well as protecting ophthalmology from *federal* scope of practice threats. Established in 1985, OPHTHPAC is one of the oldest, largest, and most successful political action committees in the physician community and is very successful in representing your profession to the U.S. Congress. As one election cycle ends, a new one starts. OPHTHPAC is always under financial pressure to support our incumbent friends as well as to make new friends with candidates. These relationships allow us to have a seat at the table and legislators willing to work on issues important to us and our patients. Among the significant achievements of OPHTHPAC are the following:

- Repealed the flawed Sustainable Growth Rate (SGR) formula
- Blocked the unbundling of the Medicare global surgery fee period
- Removed a provision in fraud and abuse legislation that targeted eyelid surgery
- Protected your ability to perform in-office ancillary services
• Working to reduce the burdens from Medicare’s existing quality improvement programs such as the Electronic Health Record Meaningful Use program
• Working in collaboration with subspecialty societies to preserve access to compounded and repackaged drugs such as bevacizumab

Leaders of the American Glaucoma Society (AGS) are part of the American Academy of Ophthalmology’s Ophthalmic Advocacy Leadership Group (OALG), which has met for the past eight years in January in the Washington D.C. area to provide critical input and to discuss and collaborate on the Academy’s advocacy agenda. The topics discussed at the 2015 OALG meeting included collaborative efforts on the IRIS Registry and quality reporting under Medicare. As a 2015 Congressional Advocacy Day (CAD) partner, the AGS ensured a strong presence of glaucoma specialists to support ophthalmology’s priorities as nearly 400 Eye M.D.s had scheduled CAD visits to members of Congress in conjunction with the Academy’s 2015 Mid-Year Forum in Washington, D.C. The AGS remains a crucial partner with the Academy in its ongoing federal and state advocacy initiatives.

**Surgical Scope Fund**

To derail optometric surgical scope of practice initiatives that threaten patient eye safety and quality of surgical care

Political grassroots activities, lobbyists and media

No funds may be used for candidates or PACs.

Contributions: Unlimited

Individual, practice, and organization

Contributions are 100% confidential.

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**OPHTHPAC® Fund**

Ophthalmology’s interests at the federal level

Support for candidates for U.S. Congress

Campaign contributions, legislative education

Campaign contributions, legislative education

Contributions are Limited to $5,000

Contributions above $200 are on the public record.

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**State Eye PAC**

Support for candidates for State House and Senate

Campaign contributions, legislative education

Contributions are on the public record depending upon state regulations.

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Please respond to your Academy colleagues who are volunteering their time on your behalf to serve on the OPHTHPAC® and Surgical Scope Fund** Committees, as well as your state ophthalmology society leaders, when they call on you and your subspecialty society to contribute. Advocate for your patients now!

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Surveying Your Cards: Optimal Strategies to Monitor Glaucoma Suspects

Joshua D Stein MD MS

I. Who Is a Glaucoma Suspect?
   A. Definition: A patient with risk factors and/or clinical findings that increase the likelihood of developing open-angle glaucoma (OAG) in the setting of open anterior chamber angles by gonioscopy.
   B. Structural changes that are suspicious for glaucoma
      1. Increased cup-to-disc (C/D) ratio
      2. C/D asymmetry
      3. Notching or thinning of the neuroretinal rim
      4. Focal or diffuse retinal nerve fiber layer dropout
      5. Presence of a disc hemorrhage
   C. Visual field (VF) changes that are suspicious for glaucoma in the absence of other clinical signs
   D. Consistently elevated IOP with no evidence of structural damage or functional loss from glaucoma
   E. Coding for Glaucoma Suspects
      1. ICD-9-CM code 365.01: Open angle with borderline finding, low risk (1-2 risk factors)
      2. ICD-9-CM code 365.05: Open angle with borderline findings, high risk (3 or more risk factors)
      3. Risk factors: + family history, non-white race, elevated IOP, optic disc appearance, and thin central corneal thickness

II. How Often Should These Patients Be Seen?
   A. Monitoring of glaucoma suspects varies from patient to patient, depending on the unique characteristics of the patient (ie, age, overall health), the number of risk factors, whether the patient is receiving treatment, and the length of time they have been monitored in the past with no signs of progression.
   B. The higher the IOP, the greater the risk of developing glaucoma and thus the more closely patients need to be monitored.
   C. Patients who exhibit large fluctuations in IOP also require more close monitoring.
   D. Importance of regular follow-up
      Since OAG is often asymptomatic, we need to reinforce to patients that even though all the test results are “normal” at the present time, it is still essential to keep checking periodically to make sure no damage develops.

III. What Tests Should Be Performed?
   A. Perimetry
      1. Standard automated perimetry
      a. Key test to perform to look for signs of functional vision loss that are consistent with glaucoma
      b. As many as 35%-50% of retinal ganglion cells can be lost before a VF defect is detected on white-on-white standard automated perimetry. For this reason, it is often useful to perform additional perimetric testing beyond white-on-white perimetry (as described below) to look for early signs of glaucoma.
      2. Frequency doubling technology (FDT) perimetry
         a. When an achromatic sinusoidal grating of low spatial frequency undergoes counterphased flickering at a high temporal frequency, the apparent spatial frequency of the grating appears to be doubled.
         b. The FDT stimulus predominately stimulates the magnocellular ganglion cell pathway, which is primarily involved in motion detection and flicker detection.
         c. High sensitivity and specificity in detecting early glaucoma
         d. Capable of detecting glaucoma earlier than when defects become detectable on white-on-white perimetry
         e. Rates of FDT pattern standard deviation (PSD) change have been shown to be highly predictive of the development of future white-on-white VF loss in glaucoma suspects.
      3. Short wavelength automated perimetry (SWAP)
         a. SWAP utilizes a blue stimulus to preferentially stimulate the blue cones and a high luminance yellow background to adapt the green and red cones and to saturate, simultaneously, the activity of the rods.
         b. SWAP can be difficult to perform, especially on patients who have media opacities. The increased examination duration and additional learning effect can make this test challenging for many patients. Use of SITA SWAP can cut down lengthy testing durations.
         c. SWAP can be difficult to perform, especially on patients who have media opacities. The increased examination duration and additional learning effect can make this test challenging for many patients.
   4. Central 10-2 VF testing
      a. Hood and colleagues have demonstrated that testing of the central 10 degrees for VF loss
can be useful in patients with early glaucoma (those with mean deviations better than -6 dB) at detecting field loss that may be missed on the 24-2 test.\textsuperscript{10}

b. Testing the central 10 degrees can be particularly useful in monitoring patients with normal-tension glaucoma as these patients often develop field loss that is close to central fixation.

B. Tonometry

1. Elevated IOP is the only known modifiable risk factor at reducing the risk of development of glaucoma.

2. Studies have demonstrated that 6\%-12\% of patients with IOPs in the 20-30 mmHg range on initial exam go on to develop VF loss. When the IOP is > 30 mmHg, the proportion of patients developing VF loss rises to 30\%.\textsuperscript{11}

3. ADAGES Study (2015)
   a. 336 eyes of glaucoma suspects were monitored for progression to glaucoma.
   b. Findings: At higher mean IOP levels, race was predictive of the development of VF damage even after adjusting for potentially confounding factors. At mean IOPs during follow-up of 22-26 mmHg, multivariable hazard ratios for the development of VF damage in African-descent compared to European-descent persons were 2- to 4-fold higher. However, at mean IOP levels < 22 mmHg during follow-up, African descent was not predictive of the development of VF damage.\textsuperscript{12}

C. Pachymetry

1. The Ocular Hypertension Treatment Study established that central corneal thickness is an important risk factor for glaucoma.

2. Patients with lower than normal central corneal thickness are at increased risk for developing OAG, as the true level of IOP is higher than the level that is measured by tonometry.\textsuperscript{13}

3. It is thought that beyond underestimating the true level of IOP, patients with reduced central corneal thickness may also have increased susceptibility to developing OAG.\textsuperscript{14}

D. Ocular imaging

1. OCT of the nerve fiber layer
   a. OCT is a useful adjunctive test to look for early signs of structural damage from glaucoma.
   b. OCT has been shown to have good sensitivity and specificity at distinguishing patients with early glaucoma from normal controls.\textsuperscript{15}
   c. Technological advances have led to the development of software that can identify progressive loss of nerve fiber layer tissue, which can indicate disease progression.\textsuperscript{16}
   d. OCT is a good complimentary test to check for glaucoma, but it is not a substitute for perimetric testing.\textsuperscript{17}

2. OCT of the macula: Asymmetry of the macular hemifield thickness may be an early sign of glaucoma.\textsuperscript{18,19}

3. Other ocular imaging tests
   a. Confocal scanning laser ophthalmoscopy
   b. Scanning laser polarimetry

E. Stereoscopic optic disc photography

1. Excellent means of documenting the appearance of the optic nerve and peripapillary nerve fiber layer tissue to look for signs of thinning of the neuroretinal rim tissue, notching, disc hemorrhages, and other findings suggestive of glaucoma.

2. Can be particularly useful with the transition to EHR, since most EHR systems do not have good capabilities of documenting the appearance of the nerve.

3. Multicolor photography can be very useful in delineating nerve fiber layer defects

F. Importance of correlating structural / functional damage

1. Artifacts on structural and functional testing can lead to erroneously concluding that glaucoma is present when it is not.\textsuperscript{20}

2. Presence of corroborating evidence of early signs of glaucomatous damage that is present on both structural and functional testing can be useful in distinguishing real damage from artifact.

3. Studies show nice correlation between damage on FDT and changes on OCT,\textsuperscript{21} damage on SWAP and OCT,\textsuperscript{22} changes in macular thickness on OCT and HVF loss.\textsuperscript{23}

IV. Predicting Who Will Progress From Glaucoma Suspect / Ocular Hypertension to Glaucoma

A. Risk factors for progressing from OHTN to OAG in the OHTS Study\textsuperscript{24}

   Every 10-year increase in age confers a 22\% increased risk; every 1-mmHg increase in baseline IOP confers a 10\% increased risk, every 40-μm decrease in CCT confers a 71\% increased risk, every increase in horizontal C/D of 0.1 confers a 27\% increased risk, every increase in vertical C/D ratio of 0.1 confers a 32\% increased risk, every increase in PSD of 0.2 dB confers a 27\% increased risk.

B. Risk calculator (OHTS / EGPS)

   Considers: Patient age, vertical C/D ratio, IOP, CCT, PSD on perimetry
V. The Future

Researchers at the University of Michigan are developing a glaucoma forecasting tool that can generate personalized, real-time, dynamically updated forecasts of glaucoma progression trajectory dynamics.\(^{25,26}\) The forecasting tool uses Kalman filtering, a powerful engineering technique that can reduce noise associated with perimetry and IOP measurements to improve the accuracy of forecasts.

The tool generates personalized forecasts of the likelihood of experiencing disease progression, estimates of the optimal time to perform additional testing to monitor for disease progression, and assistance with setting target IOPs for each patient. The tool performs well on patients with moderate and advanced glaucoma. We are presently exploring use of the tool in patients with ocular hypertension.

References


Doubling Down on Prostaglandin Analog Therapy: Single Agent, Fixed Combination, or SLT?

Sanjay Asrani MD

Prostaglandins (PGAs) remain the preferred first-line therapy for most patients with glaucoma. However, there remains little consensus on the optimal first adjunct to a PGA. Does one add a second drug, or a combination medication, or SLT?

One needs to add a second-line treatment when target IOP is not achieved or when the IOP lowering, which was thought to be enough with monotherapy, is accompanied by progression per visual field or OCT. In such cases, one needs to reset the target IOP.

How does one determine target IOP? The Canadian Society guidelines are relatively easy to follow: Mild glaucoma = high teens; moderate glaucoma = mid-teens; advanced glaucoma = low teens (with caveats for pachymetry and fluctuations).

Effectivity, allergic reaction, compliance are the issues that one grapples with.

How does one decide? Adding a second agent is in effect expecting patients to dose themselves twice a day. Even if you add a daily dose of beta-blocker, patients typically will use their prostaglandins at night (rather than wait 5 minutes to instill the prostaglandin after the beta-blocker in the morning). If you are considering a beta-blocker (which works better during the day than at night), you need to confirm the absence of asthma, emphysema, or a cardiac bundle branch block. Topical carbonic anhydrase inhibitors (CAIs) are shown to be most effective at IOP lowering when combined with a prostaglandin. They also have a few more advantages, such as good nocturnal IOP lowering and avoiding a decrease of ocular perfusion pressure due to absence of effects on systemic blood pressure. Unfortunately, though brimonidine reduces maximum IOP with t.i.d. dosing, at that dose it also reduces ocular perfusion pressure.

Approximately 30% of patients starting glaucoma therapy will require adjunctive therapy within 1 year, and many receive a fixed-combination product as initial adjunctive therapy shortly after starting glaucoma therapy. This suggests a prescribing trend toward earlier, more aggressive drug therapy to control pressure and minimize disease progression. Since topical CAIs and alpha agonists are used typically twice a day in conjunction with PGAs, some practitioners feel that adding a combination drug may provide a better IOP-lowering profile (such as a beta-blocker + topical CAI or beta blocker + alpha agonist or a topical CAI + alpha agonist). Additionally, using a combination drug, a significant IOP lowering of 5 mm or more is achievable. Since the patient goes through the extra trouble of instilling a set of drops twice a day in addition to a PGA, compliance may be improved due to positive reinforcement with the realization that there was significant IOP lowering. Pressure reduction is also maximized by adding a combination eye drop (since all combination drops are aqueous suppressants) to a PGA (a great outflow enhancer). In addition, combination therapy eliminates washout effects and reduces preservative load.

The rate of allergy to brimonidine is approximately 25%-28%. The allergic response to brimonidine can typically be delayed by 6-8 months if it is in combination with a beta-blocker. Ocular congestion due to allergy to brimonidine is commonly mistaken as dry eye and thus inappropriately treated. The allergic reaction to timolol is rare but possible and presents a picture similar to brimonidine allergy (follicular conjunctivitis). The allergic reaction to a topical CAI is different in that it typically involves periocular redness and lid margin erythema. Topical CAIs are commonly shied away from since the most common EMR-listed allergy is to sulfa drugs. However, the possibility of an allergic cross-reaction to topical CAI is extremely rare.

One of the common criticisms of adding a combination drug is the inability to check effectivity of the individual components and/or the inability to identify the agent that is causing the allergic reaction if one occurs. However, since the copay of patient visits is now typically $50 to $75, progressively adding one agent at a time significantly adds to the cost and inconvenience to the patient, besides the cost of the individual medications. Since a majority of patients are not allergic to ocular medications, one can consider replacing the combination medication with individual components if an allergic reaction were to occur.

In patients with low-tension glaucoma, lowering IOP and controlling fluctuations are equally vital. Thus in such cases, adding selective laser trabeculoplasty (SLT) as a second-line agent may be extra beneficial. This is because it reduces issues such as fluctuations due to missed doses or delayed dosing. Of course, it also reduces issues of allergic reactions. SLT effectivity is less on non-naïve eyes than eyes naïve to any treatment, but that is the case for any second-line treatment. SLT is also an excellent choice as a second-line agent in primary open-angle glaucoma since it allows the patient to remain on monotherapy. Compliance with therapy (especially if it involves more than one bottle) is especially difficult in younger patients, who are likely to be on no systemic medications. Older patients also find it difficult to comply in cases of arthritis or memory issues. In both the above instances, SLT can be a good second-line option. SLT can also be added as a second-line agent in patients with narrow angles (especially if the lens is clear) using compression gonioscopy.

References


The Disease That Never Sleeps: 24-Hour IOP Monitoring Devices

Kaweh Mansouri MD

Clinical Pearls

- Current tonometry techniques provide a mere snapshot of a true IOP.
- IOP is highest during sleep in most glaucoma patients.
- Nocturnal effect of many hypotensive drops is limited.
- Effect of nocturnal IOP on glaucoma progression is unknown.
- 24-hour IOP monitoring is around the corner.

Background

Intraocular pressure (IOP) is the only modifiable risk factor for glaucoma.1 It is a highly dynamic parameter with a circadian rhythm. The most widely used method to measure IOP is Goldmann applanation tonometry (GAT), a technique that was developed in the 1950s and has remained unchanged ever since.2 Its main limitation is the isolated nature of its measurements during office visits, which do not reflect the full range of IOP changes over the 24-hour period.

IOP is highest during the nocturnal sleep period in a majority of healthy individuals and glaucoma patients.3,4 Peak IOP can be undetected if nocturnal measurements are not obtained. As a consequence, IOP remains poorly characterized and peak values are missed in two-thirds of patients, possibly leading to undertreatment and glaucoma progression.5 In addition, the role of other IOP parameters such as short- and long-term variations and fluctuations remains understudied and controversial.6

Purpose of Presentation

To discuss 24-hour IOP dynamics and provide an overview of current status and future of ambulatory 24-hour IOP monitoring.

Recent Findings

There has been considerable progress recently with the prototype and commercial introduction of continuous 24-hour IOP monitoring devices. Recent advances in microelectromechanical systems (MEMS) and nanoelectromechanical systems (NEMS) have enabled the development of these devices. Implantable IOP sensors have the advantage of directly measuring IOP over many months and years, while temporary (contact lens-based) approaches provide a noninvasive alternative for repeated 24-hour periods. Once these technologies have shown their safety and efficacy, larger questions as to the data interpretation and handling will arise. It is likely that the use of 24-hour IOP monitoring will herald fundamental changes in our understanding and management of glaucoma.

References

New Strategies to Beat the Odds: Controlled Delivery of IOP Medications

Gary D Novack PhD

I. What Is the Problem?
   A. Elevated IOP puts patient at risk for visual field loss.
   B. Lowering IOP is needed to decrease risk.
   C. Low trust
   D. Patients do not adequately adhere or perform.
   E. Are there effective drug delivery systems?
   F. All medications (and delivery systems) have risks.
   G. Primum non nocere: Earlier stage patients need a higher benefit/risk ratio.

II. What Can You Do Today?
   A. Watch your patient use drops – and offer education (Academy pamphlet)
   B. Help convince health-care payers that improvements to patient compliance and performance are of value (and thus worth the premium).
   C. Sign up as a clinical investigator for new delivery systems.

Selected Readings

Provider Roulette: New Models of Care
Or How the Unstoppable Force of Demographics Will Change Our Approaches to Patient Care

David S Friedman MD MPH PhD

I. Introduction
Demographics require innovative management approaches.
A. Populations in developing countries are aging.
B. Life expectancy is increasing as well.
C. Glaucoma increases dramatically with increasing age.
D. The number of people requiring care for glaucoma will be much larger in the coming decades.
E. Currently glaucoma is expensive to treat, requiring frequent visits and monitoring.
F. The number of people trained as ophthalmologists will not keep up with the demand for services using current approaches.

II. Glaucoma is highly prevalent in older populations.
A. Over the age of 80 nearly 1 in 10 white individuals has glaucoma, and this rate is higher among Hispanic and Afro-Caribbean populations.
B. By 2040 there will be approximately 100 million people with glaucoma.
C. Nearly 10 million people will be blind from glaucoma.

III. Current systems are failing.
A. In developed countries 50%-90% of those with glaucoma are not diagnosed.
B. In developing countries very few are diagnosed.

IV. Glaucoma care is expensive.
A. Case detection
B. Monitoring with testing
C. Pharmaceuticals
D. Surgeries and other procedures

V. The population requiring treatment is even larger than estimated by prevalence.
A. Those with high IOP are frequently treated.
B. Many with pre-perimetric glaucoma are not included in population estimates of disease burden.
C. Likely twice as many people need care than have definite confirmed glaucoma.

VI. Failure to detect and adequately treat glaucoma has a negative impact on population health.
A. Blindness
B. Disability
   1. Less mobility and greater risk of falls
   2. Less independent
   3. Less able to read

VII. Care process is inefficient at present.
A. Most people being seen do not require face-to-face encounter with MD.
B. Most visits do not require a change in care.
C. Counseling about how to adhere to treatment programs is limited, and many fail to take medications as directed.

VIII. Role of the Ophthalmologist
A. Diagnosis (but imperfect)
B. Care plan developer
C. Surgeon
D. Educator (unlikely spending enough time)

IX. Much of this can be done remotely or by other personnel.
A. Imaging of the fundus, OCT of optic nerve, and visual field testing all can be done anywhere.
B. Newer devices constantly in development can easily be included in remote management.
C. Others can provide educational information.

X. Already Common in Ophthalmology
Diabetic retinopathy screening programs are common.
A. Rely on trained personnel to do much of the work
B. Ophthalmologists only involved when care is needed
C. Far more efficient and often better than routine eye exams

XI. Other fields also have remote care, such as radiology and dermatology.

XII. Improved Model of Care
An improved model of care could involve:
A. Centralized testing sites with constant upgrading of equipment
B. Review of tests in a systematic way to provide integration of all data (which could include computer-assisted interpretation)
C. Education of the patient, mostly by trained personnel

D. Ophthalmologists spending more time with patients who need changes in care or surgery

Selected Readings


Point – Counterpoint: Cashing Out Too Soon: Which Form of Noncompliance Poses a Higher Risk for Glaucoma Progression? Noncompliance With Therapy

Ivan Goldberg MBBS FRANZCO

The Case for Therapy (as Opposed to Keeping Appointments)

- Glaucoma progression is linked with higher IOP and mean, peak, fluctuations.1-4
- Reduction in IOP decreases risk of onset and of progression of glaucoma visual loss.1-4
- Medical therapy remains the mainstay of IOP-reduction strategies.5
- For this to be effective, a patient needs continuous activation / suppression of relevant receptors to increase outflow / decrease inflow.
- In turn, this requires continuous access of active molecule(s) to the receptors.
- This depends on pharmacokinetically determined frequency of instillation.
- This requires patient adherence to and persistence with the regimen in the space-time continuum.

Keeping appointments has nothing to contribute to meeting these requirements.

In fact: if a patient is nonadherent, often drops will be instilled for a few days just before an appointment, leading to an apparently controlled IOP at the visit; if nothing untoward is observed or measured at the visit, reassurance from the ophthalmologist reinforces nonadherent behavior. In this context, visits could be dangerous!

References

Point – Counterpoint: Cashing Out Too Soon: Which Form of Noncompliance Poses a Higher Risk for Glaucoma Progression? Noncompliance With Follow-up

Kuldev Singh MD MPH

Primary open-angle glaucoma is a complex disease, the management of which is often more art than science. The evidence that guides glaucoma therapy is generally based upon average results from large clinical trials, yet the seasoned practitioner knows that the stage of disease at presentation and the rate of future progression are unique to each patient. While there is no doubt that the use of effective and safe IOP-lowering therapy improves the odds of good visual health, the impact of such therapy varies considerably between glaucoma patients. There are certainly patients who never miss their medications and sometimes get worse while others who are noncompliant surprisingly do not show substantial disease progression. There is no compelling evidence that rigid compliance ensures a good outcome and that noncompliance with prescribed medications definitively results in vision loss. Given that at least half of all Americans with primary open-angle glaucoma are unaware that they have the disease, that some who know that they have the disease are not regularly receiving treatment, and that many among the majority who are not receiving glaucoma medications live and die without ever knowing that they have the disease, much less are going blind, suggests that instillation of glaucoma medications is just one of many important factors in determining glaucoma outcomes.

The issue of medication noncompliance has become big business in all areas of medicine, and ophthalmology is no exception. Legions of glaucoma speakers get up on the podium at sponsored dinner meetings and talk about how noncompliance can cause blindness, with the implied presumption that compliance with medications generally prevents vision loss from the disease. Seasoned practitioners, however, know that it is not quite so simple.

The treatment choices for individuals showing disease worsening despite taking effective glaucoma medications are not much different than the options for individuals who are noncompliant and show such progression. Practically speaking, failure to instill medications to adequately slow disease results in the same consequences as disease progression related to failure to take such medications as prescribed. Both types of patients will likely require laser or surgical intervention to further lower IOP. Practitioners may also acknowledge that there exist glaucoma patients who clearly have very poor compliance with their medications, sometimes admitting to such noncompliance, but remarkably do not demonstrate measurable progression of disease, and these individuals, just as those who are compliant with medical therapy and are not progressing, may not require additional treatment. The practical consequences of relative stability of disease also does not substantially differ between those who do or do not regularly take their medications. Further, the practitioner cannot prospectively determine whether or not noncompliance with medical therapy will definitively result in vision loss and that compliance will ensure good vision for an individual glaucoma patient. To further put matters in context, the Collaborative Normal Tension Glaucoma Study showed that approximately half of untreated glaucoma subjects showed no measurable disease progression for almost a decade, and there were others who were randomized to receive treatment, including medications, laser trabeculoplasty, and filtration surgery, who continued to worsen.

Nothing in the above argument should lead the practitioner to feel hopeless with regard to glaucoma care, as there is ample evidence that both medical and surgical treatment of the disease does, on average, improve outcomes. Encouraging compliance with prescribed medications is good medical practice that should not be abandoned. Yet there is another form of often overlooked noncompliance that may overshadow, in significance, all efforts related to improving patient instillation of glaucoma medications. Compliance with follow-up visits is critical for successfully treating glaucoma patients because such follow-up allows the practitioner to gauge the rate of disease progression and modify therapy as needed.

Patients with glaucoma, particularly those who are diagnosed at the mild to moderate stages of the disease, do not commonly go blind simply because they don’t take their glaucoma medications, although noncompliance may clearly contribute to such an outcome. One can postulate that a more important predictor of poor outcomes among glaucoma patients may be the failure to keep ophthalmic appointments at appropriate intervals, which denies the practitioner an opportunity to determine whether or not advancement of therapy, including possible surgical intervention, is needed to decrease the likelihood of vision loss over the course of the patient’s lifetime. All glaucoma patients, regardless of whether or not they are taking their glaucoma medications correctly, are at risk for such vision loss. While those who are compliant with medications, all other things being equal, are at less risk than those who are not, the inter-individual variability in the risk of progression arguably makes appropriate surveillance of all patients at least as important as compliance with medications. Such surveillance allows those who need more aggressive IOP lowering, in some cases surgical intervention, to be treated at the ideal juncture.

Among those who may be at highest risk are patients who falsely believe that they don’t need to see their ophthalmologist at recommended intervals because, by virtue of taking their medications as prescribed, they are safe from disease worsening. Such individuals may conceivably be at greater risk of substantial disease worsening and severe vision loss than patients who sometimes miss their medications but come to see their ophthalmologist regularly at appropriate intervals. One cannot offer surgery to a progressing patient who doesn’t show up for regular visits, and while it is tempting to assume that the patients who don’t take their medications correctly are also the ones who are likely to miss their appointments, a recent study by Ung and colleagues did not reveal such a correlation.

Why is it, then, that there is a paucity of lectures at continuing medical education meetings regarding the importance of regular surveillance and the perils of noncompliance with follow-up vis-
its? The obvious answer is that unlike the case of noncompliance with medications, there is no convergence of special interests that have led to an emphasis on timely follow-up. The majority of glaucoma patients in developed countries are cared for by ophthalmic practitioners who also treat other ocular diseases, and given that glaucoma is a lifelong disease, often requiring visits every 3-6 months for decades, practitioners can be inundated with glaucoma follow-up visits, preventing them from having openings for other types of patients. The anticipated labor shortages for providing glaucoma care in coming generations will likely make matters worse unless models are developed for efficient glaucoma surveillance.

Seasoned practitioners spend time and effort in educating glaucoma patients regarding the risks of long absences from the clinic. There has not been, however, a national or international campaign with significant commitment of resources to educate patients about the dangers of insufficient follow-up. Glaucoma lectures commonly discuss the perils of noncompliance with medications and even show videos of patients incorrectly instilling their glaucoma medications, yet there is rarely even mention of patients who went blind from glaucoma primarily as a result of insufficient disease surveillance, sometimes with the false sense of security that taking their glaucoma medications or having previously undergone a laser or surgical procedure would arrest their disease, making such follow-up less important. It is fortunate that glaucomatous disease does not always result in severe visual disability among those who are afflicted. The most important factor in distinguishing those who are destined for poor outcomes from the many who are not is to make sure that all those with the disease are seen at recommended intervals, regardless of whether or not they are taking their medications as prescribed.

References


OHTS at Twenty Years: What We Have Learned and What We Hope to Learn

Michael A Kass MD

I. Ocular Hypertension Treatment Study, Phase 1
   A. Study goals
      1. Evaluate the safety and efficacy of topical ocular hypotensive medication in delaying or preventing the development of primary open-angle glaucoma (POAG) in individuals with elevated IOP
      2. Identify baseline demographic and clinical factors that predict which participants will develop POAG
      3. Why did we do the study? Didn’t everyone already know that preventative treatment is effective?
      4. Majority of the published studies failed to demonstrate that early treatment is effective in delaying or preventing POAG in patients with ocular hypertension (OHT). Many of these studies had methodologic limitations.
   B. Entry criteria
      1. Age: 40-80
      2. Normal visual fields
      3. Normal optic discs
      4. Untreated IOP 24-32 mmHg in 1 eye and 21-32 mmHg in fellow eye
   C. Study design
      1. Participants have 2 visits per year with visual fields.
      2. Stereoscopic optic disc photographs once a year
      3. Participants randomized to close observation or topical ocular hypotensive treatment
      4. Treatment goal: IOP reduction of 20% from baseline and IOP ≤ 24 mmHg
      5. Photos and fields judged by masked readers in reading centers
      6. Final decision on POAG by masked endpoint committee
   D. Results
      1. At 5 years POAG developed in 9.5% of observation participants and 4.4% of medication participants; hazard ratio, 0.40
      2. Treatment differential for both structural and functional measures
      3. Most endpoints noted first in the optic disc
      4. Treatment effective in African Americans
      5. No safety concerns detected
   E. Prediction model
      1. Model of baseline age, IOP, central corneal thickness (CCT), cup/disc (C/D) ratio and visual field pattern standard deviation (PSD) effectively separates participants at low, medium, and high risk (C statistic 0.75)
      2. Race drops out of the model when C/D ratio and CCT included
      3. Model confirmed by European Glaucoma Prevention Study
      4. Model widely accepted and incorporated into preferred practice plans
      5. Allows evidence-based decisions about frequency of visits and benefit of early treatment

II. OHTS Phase 2
   A. Rationale
      1. OHTS Phase 1 provided proof of concept: Lowering IOP reduces the incidence of POAG.
      2. OHTS Phase 1 does not indicate when treatment should begin or whether all OHT patients should receive medication.
      3. In large part this depends on whether there is a penalty for delaying treatment in OHT.
   B. Study design
      1. Participants in medication group stay on medication.
      2. Participants in observation group offered medication (ie, create an early treatment group and a delayed treatment group)
   C. Results at 13 years
      1. Incidence of POAG in OHTS Phase 2 essentially identical in the two groups: relative risk, 1.0. Incidence of POAG in the observation group dropped to the level of the medication group even though medications were started much later.
      2. Participants in the observation group do have a higher burden of disease: 22% vs. 16% at 13 years. The difference is greatest in the highest risk group.
      3. African Americans have a higher incidence of disease at 13 years: 0.28 vs. 0.16 for other participants.

III. What We Have Learned So Far
   A. Early medical treatment reduces the cumulative incidence of POAG.
The absolute effect is greatest in highest-risk OHT individuals.

There is little absolute benefit of early treatment in low-risk individuals.

There are safe and effective medications for most OHT patients.

The risk of developing POAG continues over at least a 15-year period.

African Americans develop POAG at a higher rate despite similar treatment and similar baseline and follow-up IOPs. The higher incidence is related to baseline factors, especially larger C/D ratio and thinner CCT.

IV. Personalized Medicine

A. Across medicine there is an attempt to increase the value and appropriateness of care and to diminish wasteful care. This approach has been given different names, including personalized medicine, precision medicine, patient-centered care, parsimonious medicine, and evidence-based care.

B. These terms are not synonymous but share several key features:

1. Risk stratification of patients
2. Estimation of potential treatment benefit
3. Estimation of the frequency and severity of potential adverse events
4. Inclusion of shared patient-clinician decision making
5. Inclusion of patient-reported outcomes as a key measure of treatment success

V. OHTS Phase 3

A. Rationale

While developing POAG is an important clinical landmark, the true goal of managing patients with OHT is to prevent the development of functional limitations from POAG. As recently as 2013, the U.S. Preventative Services Task Force (USPSTF) concluded that “there was inadequate evidence that treatment of increased IOP or early asymptomatic POAG reduces the number of persons who will develop impaired vision and quality of life....Treatments that are effective in reducing intraocular pressure have potential harms, and their effectiveness in reducing patient perceived impairments in vision related functions is uncertain.” Additionally the task force concluded that “more evidence is needed on the link between the intermediate glaucoma outcome of optic nerve damage and visual field loss and the health outcomes of visual disability and patient reported outcome.” The 20-year follow-up study we propose will address some of the gaps in our knowledge identified by the USPSTF.

B. Specific aims

1. Determine the 20-year incidence and severity of POAG in the OHTS cohort. Twenty years approaches median life expectancy for patients in their 60s and 70s, 50% of median life expectancy for patients in their 40s and 50s.

2. Develop a 20-year prediction model for stratifying OHT patients by their risk for developing POAG

3. Develop a prediction model for the rate of visual field loss. OHTS has a large inception cohort and a control group that did not develop POAG, which should control for the effects of aging.

4. Determine the frequency and severity of self-reported limitations associated with POAG and correlate these with clinical findings.

C. Study design

1. Re-examine all living participants in original OHTS cohort.

2. Same assessment of tests.

D. Goal

Collect information that will allow patients and clinicians to make more informed, evidence-based decisions about management of OHT and early open-angle glaucoma.

References


Primary angle-closure glaucoma (PACG) is estimated to be responsible for approximately half of binocular glaucoma blindness worldwide, and by 2020 over half of PACG cases will be found in East Asia. Approximately 10% of the Chinese population of 50 years and older are classified as asymptomatic primary angle-closure suspect (PACS). Laser iridotomy (LI) has been recommended by American Academy of Ophthalmology as the preferred initial treatment for both PACG and primary angle closure (PAC).

When?
The effectiveness of LI in preventing acute attacks of angle closure in the fellow eyes of patients who have suffered from unilateral acute angle closure has been demonstrated in previous studies. Although it is also recommended in the Academy’s preferred practice pattern that “iridotomy may be considered to reduce the risk of developing angle-closure,” no conclusive evidence has demonstrated that persons with asymptomatic narrow angles on gonioscopy benefit from prophylactic LI. One of our previous studies assessing immediate effects of LI in urban citizens of South China has shown that LI could open the drainage angle in a majority of PACS cases, whereas angles in a significant minority of eyes remained closed after the treatment. Thus, the timing and the identification of the high-risk populations are of crucial importance in the prophylactic LI treatment of angle closure.

Where?
LI is a procedure of generally high safety profile. One of its most clinically manifest complications is visual disturbance, including transient blurred vision, halos, shadows, glare, ghost images, and crescents and lines. Incidence of these visual symptoms was found to be closely associated with positions of the iridotomy. Significantly higher proportion of patients with iridotomies partially covered by the upper eyelid margin had visual disturbance following LI. Hence it has been suggested that the 11 to 1 o’clock range would be the preferred position, providing relatively greater chance for the iridotomy to be fully covered. However, recent evidence has not shown such association between straylight and PL. Other location-sensitive complications are also to be considered, such as dysphotopsia and corneal decompensation. More recently, data have suggested that a temporal LI has less risk for dysphotopsias than a superior one.

How Large?
The success and efficacy of the procedure greatly rely on patency of the iridotomy, and long-term patency is associated with the size of the iridotomy. Back in the 1990s, Flegg and colleagues advocated a theory that an iridotomy of 10-15 μm should be theoretically adequate for preventing acute angle closure in an eye with a peripheral iris thickness of 50 μm. Based on the current understanding of peripheral iris anatomy, it was proposed that a minimum iridotomy size of 150 μm should be recommended to ensure the efficacy of LI in the elimination of pupillary block and to consistently provide adequate bypass aqueous flow.

There is also evidence from a study utilizing ultrasound biomicroscopy that enlargement of small LIs helps deepen the angle further.

Zhongshan Angle-closure Prevention (ZAP) Study
In the context of limited health-care resources and budgets, the efficacy and potential risk for using LI as a prophylactic treatment in PAC suspects need to be demonstrated by well-designed clinical trials with long-term follow-up. In September 2008, we started a randomized controlled trial in the South China city of Guangzhou, aiming to assess the effects of LI in preventing the development of acute or chronic angle closure in PACS.

Out of the 11,991 urban Chinese aged 30 to 70 examined in the screening survey, 1113 were identified as PAC suspects, of which 889 gave consent to be randomized as study participants. Each participant was treated by LI in a randomly selected eye, with the fellow eye serving as a control. In this ongoing trial, PAC suspects will be followed up for a period of 36 to 60 months for comparing incidence of acute or chronic angle closure in treated and untreated eyes. Midterm data analysis has shown that at 18 months after LI, when 94.3% of the study participants still remained in the trial, there was a trend for significant decrease in angle width in both treated and untreated eyes despite the remarkably widened drainage angle immediately following LI in treated eyes. We believe outcomes of this trial will provide important information to help clinicians determine the indication and optimal timing of applying laser iridotomy.

This lecture will review existing evidence, introduce selective ongoing studies, and discuss the timing, location, size, and scale of LI in high-risk populations.

References


Point – Counterpoint: Gonioscopy Is the Gold Standard, and Management Should Be Based on This Procedure

Tanuj Dada MD

Gonioscopy is the gold standard technique to view the anterior chamber angle and is critical for both diagnosis and laser/surgical interventions. The diagnosis of angle closure is based on the visibility of the pigmented trabecular meshwork, peripheral anterior synechiae, pigment clumps, and the effect of indentation/manipulation on these structures—all clinical findings that are diagnosed with gonioscopy. Additionally, gonioscopy can pick up early neovascularization of the angle, pseudoexfoliation, and blood in the Schlemm canal. It can also be used for laser trabeculoplasty and intraoperative procedures such as goniosynechialysis. Despite recent advances in anterior segment imaging using ultrasound biomicroscopy and anterior segment OCT, gonioscopy is the only cost-effective method and can be universally taught to ophthalmologists/optometrists and adopted worldwide for diagnosing primary angle-closure disease.
Anterior segment imaging using either OCT or ultrasound biomicroscopy provides an objective cross-sectional view of the anterior chamber angle, which can be used to evaluate the angle condition (eg, open or closed, relative pupillary block or plateau iris, etc.). Semiautomated software allows us to measure various anterior chamber angle-related parameters (eg, angle opening distance, trabecular-iris space area, etc.) in an objective and highly reproducible fashion. However, the agreement of angle assessment between gonioscopy and anterior segment imaging is still controversial.

The current discussion will cover information on quantitative glaucoma angle assessment using anterior segment imaging modalities and the potential pitfalls when interpreting such analyses, as well as practical suggestions on integrating anterior segment imaging into the conventional glaucoma practice setting.

References
Point – Counterpoint: Clear Lens Extraction Is Effective and Safe for Angle-Closure Glaucoma (and Insurance Should Pay for It as Primary Treatment)

Clement C Y Tham FRCS MBBS FCOphthHK

Summary

The thickness and anterior positioning of the lens is an important mechanism leading to angle-closure glaucoma. Whether there is visually significant cataract or not, such lenses are pathological and are the direct causes of anterior chamber shallowing and angle closure. Extraction of such lenses is the most effective way to reverse the anatomical predisposition to angle closure in these eyes. Lens extraction in such situations significantly reduces IOP and the reliance on glaucoma drugs, and such effects have been shown to persist for at least 6 years after surgery. Lens extraction also eliminates pupillary block and the risk of acute angle closure.

If these lenses are not extracted, cataract will eventually develop. The development of cataract may be accelerated by the treatments for angle-closure glaucoma and also possibly by major IOP fluctuations. Delaying lens extraction until there is visually significant cataract may mean more intraoperative surgical risks, including irreversible damage to corneal endothelium when the anterior chamber is shallow. Delaying lens extraction may mean more expenditure on glaucoma drugs, as well as the possible cost of other glaucoma surgery, in addition to the phacoemulsification that will eventually be needed anyway.

For the above reasons, whenever surgery is contemplated in an eye with angle-closure glaucoma, lens extraction should be one procedure within the first surgical intervention, whether performed alone or in combination with other glaucoma surgery. Considering its central position in the surgical management algorithm for angle-closure glaucoma, there is no reason why insurance should not pay for lens extraction.
Point – Counterpoint: Clear Lens Extraction Has Not Been Sufficiently Demonstrated to Be Safe and Effective (and Insurance Shouldn’t Pay for It as Primary Treatment)

Tin Aung FRCS PhD

This is a controversial topic: the role of clear lens extraction (CLE) for primary angle-closure glaucoma (PACG). The arguments against CLE are summarized as follows:

1. Multiple Mechanisms of Angle Closure

Angle closure occurs due to obstruction of the trabecular meshwork by the iris, resulting in impaired aqueous outflow and causing an increase in IOP. Patients with PACG tend to have shallower anterior chamber depth, with thicker and more anteriorly placed lenses. Pupillary block is considered to be the primary mechanism for angle closure, and laser peripheral iridotomy is the initial treatment option. Recent advances in imaging techniques such as anterior segment OCT (AS-OCT) and ultrasound biomicroscopy (UBM) have highlighted the role of non-pupil block factors in PACG. These are plateau iris, a thick peripheral roll of the iris, and dynamic factors such as increase in iris volume with dilation and choroidal expansion. Novel anatomical risk factors have also been identified through imaging, including smaller anterior chamber width (ACW), area, and volume, thicker iris with greater curvature and area, and an increased lens vault (LV).

To better elucidate their individual contribution toward PACG pathogenesis and to identify the predominant mechanism involved, we evaluated these anatomical risk factors in a recent study. Using hierarchical clustering and Gaussian mixture model methods, three subgroups of angle closure patients were identified. The predominant phenotypes were (1) greater iris area (30%-33.9%), (2) large LV (4.2%-10.7%), and (3) a mixture of iris and LV components (59.3%-61.8%).

These findings suggest that angle closure is not a homogeneous disease but that there are subtypes according to the presence of one or a combination of several anatomic risk factors. The lens may thus play a predominant role in only a subset of eyes with angle closure. Tran et al also found persistent angle apposition following lens extraction in PACG eyes with plateau iris. This suggests that not all angle closure eyes respond similarly after lens extraction.

2. Lack of Standardized Definition of “Clear Lens”

There are currently no standardized criteria for the definition of “clear lens”: do we mean “visually insignificant” or “symptomatically insignificant” cataract? Or absolutely clear lenses? Tham and associates, in their randomized controlled trial of phacoemulsification versus trabeculectomy in PACG, defined “no cataract” as an eye with BCVA of 20/40 or better and not affecting activities of daily living whereas in the ongoing multicentered RCT of Lens Extraction (EAGLE), the presence of symptomatic cataract was considered an exclusion criteria. Therefore, the use of “clear lens” as a term to describe visually / symptomatically insignificant cataract is misleading. Advocating CLE in all patients with PACG may lead to needless lens extraction procedures among nonglaucoma specialists, who may not have the expertise to handle lens extraction-related complications in eyes with PACG.

3. Complications/Risk of Surgery

Lens extraction in angle closure eyes is often technically challenging, with risk of complications. There is a greater possibility for posterior capsule rupture, endothelial cell loss, and iris prolapse, as these eyes tend to have shallower anterior chambers. Also, eyes with previous acute angle closure may have weakened zonules and compromised endothelium. In some cases, pupil dilation may be poor. Additionally, there are the small but potential risks that are associated with lens extraction in general such as endophthalmitis, retinal detachment, macular edema, and posterior capsule opacity. Such possibilities should be considered when contemplating CLE in PACG.

4. Insufficient Evidence on the Benefits of CLE in PACG

The IOP-lowering ability of lens extraction alone in medically uncontrolled PACG eyes with no significant cataract was evaluated in an RCT comparing phacoemulsification with trabeculectomy. The trial recruited 50 eyes, of which 26 were randomized to phacoemulsification. Although the magnitude of IOP reduction was comparable, the trabeculectomy group required on average 1.1 fewer medications than the phacoemulsification-treated eyes. There was no significant difference in terms of visual acuity or the proportion of eyes requiring additional surgical interventions between the groups at 2 years. Data from this trial suggest that trabeculectomy is more effective in reducing IOP and dependence on glaucoma drops in medically uncontrolled PACG eyes with no cataract, but is associated with more complications. The authors surmised that in medically uncontrolled PACG with no cataract, lens extraction alone can be an alternative to trabeculectomy as an initial approach. In a case series of 5 primary angle closure / PACG eyes with uncontrolled IOP, CLE was performed for the treatment of the elevated IOP. Four of the 5 cases demonstrated significant IOP lowering and angle widening; while in 1 eye the IOP remained unchanged, and this was attributed to the presence of widespread peripheral anterior synechiae (PAS) preoperatively.

Some eyes with PACG may benefit from CLE; however, the results from a single RCT and case series are not sufficient to justify a paradigm shift in the management approaches in PACG. More studies and multicentered clinical trials are needed to provide convincing data that would necessitate modification of the
existing angle closure management strategy / guidelines and to aid in deciding the appropriate therapy for an individual patient.

5. Cost-Effectiveness of CLE in PACG

Presently, there are no published data that have assessed the cost-effectiveness and cost benefits of CLE vs. alternative treatment options for PACG. Unless evidence clearly suggests that CLE is not only effective but also cost-effective and safe compared to the current standard of care, it is unlikely that insurance will pay for it as a primary treatment in angle closure.

Conclusions

There is evidence to suggest that lens removal results in lowering of IOP in some PACG eyes. However, in the absence of cataract, the benefits of lens extraction should be weighed against the underlying angle closure mechanism, the potential risks of complications, efficacy of IOP reduction, and overall cost-effectiveness.

References

Point – Counterpoint: Laser Iridoplasty Is Effective for Treating Angle-Closure Glaucoma

Robert Ritch MD FACS

The title of this talk was given to me as such, and because of the short time for presentation, I did not request that it be changed. However, a general discussion of argon laser peripheral iridoplasty will be presented first.

I. Argon Laser Peripheral Iridoplasty

ALPI is a simple and effective method of opening an appositionally closed angle when laser peripheral iridotomy (LPI) either cannot be performed (acute angle closure, or AAC) or does not eliminate appositional angle closure because mechanisms other than or in addition to pupillary block are present (plateau iris syndrome or lens-related angle closure).

A. Long-duration, low-power, and large spot size contraction burns are placed in the extreme iris periphery to contract and compact the iris stroma at the site of the burn, physically pulling open the angle (see Figure 1).

B. Indications

1. Breaking an attack of AAC

   a. ALPI is a safe and effective alternative to anti-glaucoma medications for initial treatment of AAC.

   b. No longer necessary to treat medically prior to performing ALPI
c. Circumferential treatment of the iris opens the angle in those areas in which there are no peripheral anterior synechiae (PAS).

d. Appositional closure is eliminated immediately and IOP is almost always normalized within one hour in eyes without extensive PAS. Even when extensive PAS are present, the IOP is usually normalized within an hour or two.

e. All published series have reported virtually 100% success.

2. Eliminating continued appositional closure if elimination of pupillary block by LPI is insufficient to open the angle

a. Plateau iris
b. Lens-related (phacomorphic) angle closure
c. Malignant glaucoma

C. Contraindications

1. Severe and extensive corneal edema or opacification
2. Flat anterior chamber
3. Total PAS
   a. ALPI is ineffective at breaking PAS despite two publications claiming this.
   b. However, even in eyes with extensive PAS, ALPI can open the angle in areas where it is appositionally closed and result in lowering of IOP.
   c. It is ineffective in eyes with angle closure originating anterior to the iris ("pull" mechanisms), such as in iridocorneal endothelial syndrome.

II. Important Considerations for Success in Performing ALPI

A. The diagnostic indications for ALPI require the ability to differentiate subtle gonioscopic findings.

B. The examiner must be familiar with indentation gonioscopy, which is necessary for differentiating appositional from synechial angle closure.

C. Gonioscopy should be performed in a completely darkened room using a small square beam of light so as not to cause pupillary constriction, while the beam should be focused on the superior angle, which is usually the narrowest.

D. Since ALPI does not eliminate pupillary block, a peripheral iridotomy is still required in eyes with AAC once IOP is controlled and the cornea has cleared sufficiently.

E. The optimum laser burn parameters consist of 0.5-0.7 second, 500 microns, starting at 240 mW and increasing the power until contraction of the peripheral iris stroma is noted.
   1. An Abraham lens is used.
   2. Burns must be placed in the far periphery of the iris in order to achieve successful opening of the angle. Burns placed more centrally will have no effect (see Figures 2 and 3).
Figure 2.

Figure 3.
III. Treatment of Angle-Closure Glaucoma

Few studies have been published on the effect of ALPI in eyes with angle closure and glaucomatous damage.

A. Eyes with chronic angle-closure glaucoma and a combination of PAS and appositional closure can respond to ALPI with opening of the appositionally closed portions of the angle.

B. Chew et al reported 11 eyes with IOP > 20 mmHg despite maximal medical therapy, of which all responded with initial lowering of IOP; 7 remained controlled at 6 months, while 4 required trabeculotomoy.

C. Study comparing long-term course of eyes with glaucomatous damage in New York and Singapore

1. 31.3% of the New York eyes went on to filtering surgery compared to 53.0% of the Singapore eyes.

2. Seven eyes in the New York group underwent ALPI, after which IOPs were controlled and surgery was not required, while ALPI was not performed in the Singapore patients.

D. ALPI may open sufficient portions of the angle for laser trabeculoplasty to be performed.

E. If extensive PAS are present after ALPI, goniosynechialysis (GSL) may be performed, surgically stripping PAS from the angle wall to restore aqueous access to the trabecular meshwork.

1. GSL is thought to be useful only if the PAS have been present for < 1 year.

2. Combined cataract extraction and GSL was reported more effective than GSL alone.

Selected Readings


Point – Counterpoint: Laser Iridoplasty Is Not Effective for Treating or Preventing Angle-Closure Glaucoma

Paul J Foster FRCS

Since its inception in the late 1960s, argon laser peripheral iridoplasty (ALPI) has been promoted as an effective treatment for primary angle-closure disease, both in the acute and chronic manifestations of the disease. In this review, I summarize data on the effectiveness of ALPI. Using PubMed, I used the following search terms to identify randomized controlled trials (RCTs) reporting the performance of ALPI: “iridoplasty trial,” “iridoplasty randomized controlled trial,” “plateau iris,” “plateau iris trial,” and “plateau iris management.” Reports were generally small case-series, with only 2 RCTs. A Cochrane review was also identified.

In 2002, Lam et al examined the role of ALPI in treatment of acute primary angle-closure (APAC) when immediate laser peripheral iridotomy is not considered possible. Seventy-three eyes of 64 consecutive patients with their first presentation of APAC, with IOP levels of ≥ 40 mmHg, were recruited. Each affected eye was treated with topical pilocarpine (4%) and timolol (0.5%). The patients were then randomized to receive either immediate ALPI or systemic medication (500 mg of intravenous acetazolamide, followed by oral acetazolamide 250 mg 4 times daily) until IOP levels normalized. Both groups continued to receive topical pilocarpine (1%) until peripheral iridotomy could be performed. Thirty-three eyes of 32 patients were randomized to receive immediate ALPI. Forty eyes of 32 patients had conventional systemic medical therapy. Both treatment groups were matched for age, duration of attack, and IOP at presentation.

In a subsequent report, the same team reported on the intermediate term outcome in the two groups, although the number of patients (N = 71) was slightly larger, with more patients having been enrolled after the study period in the 2002 report finished. With a minimum of 6 months follow-up, there were no statistically significant differences in mean IOP and requirement for glaucoma drugs between APAC eyes treated with ALPI and those treated with systemic medications.

The other RCT was carried out to examine the efficacy of ALPI in conjunction with laser peripheral iridotomy in the management of eyes with synchial primary angle-closure (PAC) or primary angle-closure glaucoma (PACG). The study recruited consecutive patients; 77 eyes of 77 people underwent iridotomy alone, and 81 eyes of 81 patients were treated by iridotomy and iridoplasty. Of these, 61 patients in the iridotomy group (79%) and 65 patients from the iridotomy plus iridoplasty group (80%) completed 1 year of follow-up. There were no significant differences between the groups in the baseline data. There was no significant difference in IOP, medications, need for surgery, or visual function between groups at the 1-year visit. The authors concluded that in eyes with synchial PAC or PACG, iridotomy and iridotomy combined with iridoplasty both provided a significant and equivalent reduction in IOP.

Neither RCT identified any significant safety concerns with ALPI, although it is not clear that they were adequately powered to examine the safety of the procedure. While one must recognize that conventional medical treatment of raised IOP has some safety concerns, subsequent reports have identified adverse effects of ALPI. A report from New York and London identified persistent pupil dilation developing after ALPI in 12 eyes of 8 patients. This was unresponsive to pilocarpine, but generally resolved by 1 year.

Evidence-based reviews of management have not identified any hard data supporting a role for ALPI. The American Academy of Ophthalmology’s 2005 Preferred Practice Pattern for Primary Angle-Closure (PAC) carried out an evidence-based review of management of the disease and recommended only laser iridotomy as an initial intervention, surgical iridectomy as an alternative if laser iridotomy is not possible, and consideration of the use of surgical lensectomy in chronic primary angle-closure, with or without glaucoma. Laser iridoplasty was not recommended, due to lack of evidence. A Cochrane review carried out in 2008 to assess the role of iridoplasty in management of PAC in the nonacute setting found that at the time there were no RCTs that examined the performance of this treatment. The authors concluded that “there is currently no strong evidence for laser peripheral iridoplasty’s use in treating angle-closure.”

In summary, ALPI is used as a method of managing APAC, PAC, and PACG, and it may have benefits in specific individual cases. However, the case for widespread use of ALPI in a routine care pathway remains unproven.

References
Scleral Flap Dehiscence

Marlene R Moster MD
Bleb Revision for Hypotony

Keith Barton MD

The Effects of Hypotony and When to Revise

Hypotony is usually diagnosed when the IOP is less than 3 standard deviations (SD) below the population mean. Less than 6 mmHg is typically used as a cutoff. Ideally, hypotony should not be diagnosed unless the IOP is consistently below this cutoff (eg, measured below 6 mmHg on at least 2 consecutive clinic visits), unless the IOP level is very low (eg, less than 3 mmHg).

The effects of hypotony may be mild and reversible, such as astigmatism (typically with the rule), intermittent blurring due to compression of the globe on blinking, and mild shallowing of the anterior chamber or axial length reduction. While reversible, the visual effects can be frustrating for patients as they often fluctuate and therefore cannot be corrected easily by refractive methods alone. On the other hand, a proportion of patients with IOP levels chronically around 4 or 5 mmHg will remain completely asymptomatic and may require no intervention at all.

Of greatest concern is the group who either have severe hypotony or sequelae of severe hypotony. These include those with consistently very low IOP of < 3 mmHg, hypotony maculopathy, marked anterior chamber shallowing, or choroidal effusions. Aphakic eyes, high myopes, and those with sudden decompression can also be at risk of suprachoroidal hemorrhage, which is the most devastating complication of hypotony. In eyes with hypotony sufficient to cause significant sequelae or to confer a high risk of sequelae, prompt revision is generally indicated.

Choroidal effusions will generally resolve without draining if the IOP is restored to levels above 6 mmHg, though occasionally IOP levels in double figures are required to achieve resolution. Hypotony maculopathy should be suspected if the BCVA (with pinhole) is less than 20/30. Clinically, wrinkling of choroid and retina may be seen and demonstrated using OCT. Hypotony maculopathy is more common in myopes and requires prompt correction of the IOP in order to restore the correct choroidal and retinal morphology. Failure to correct severe hypotony maculopathy promptly may occasionally result in a chronic irreversible macular pucker with marked visual loss.

Conservative Treatment of Hypotony After Trabeculectomy

Conservative treatment of hypotony is often useful in the early postoperative period after trabeculectomy. The frequency of postoperative topical corticosteroids may be reduced in an attempt to accelerate healing.

In the early postoperative period, one or more anterior chamber injections of viscoelastic may provide a temporary solution to protect against the sequelae of hypotony while waiting for healing to occur. Viscoelastic may be injected into the anterior chamber at the slit lamp if a temporal corneal paracentesis has been made at the time of the original surgery. In my own practice, I typically transfer the viscoelastic to an insulin syringe with a fixed 29-gauge needle and inject transcorneally after topical anesthetic and 5% povidone iodine. This must be performed very carefully in phakic eyes, and I usually inject horizontally from a corneal entry site at 11 or 1 o’clock, rather than radially, so that the lens is not traumatized if the patient moves inadvertently.

In contrast to the early postoperative period, hypotony may occur later, secondary to changes in either the scleral flap or conjunctival filtration bleb.

How to Revise

Semiconservative methods

There are a number of semiconservative methods of bleb revision for hypotony. These include autologous blood injection into or around the drainage bleb, bleb delimitation or compression suturing, and cautery to shrink the bleb.

In occasional cases, late hypotony may develop many years after a trabeculectomy in the presence of an unimpressive drainage bleb, and occasionally no visible drainage bleb at all. In that situation, it can be unclear if the hypotony is related to the prior trabeculectomy or whether another cause should be sought. In pseudophakic eyes, a useful diagnostic test in this situation is the intracameral injection of a nonexpansile concentration of a gas such as 20% SF6 or 12% C3F8. Later appearance of gas bubbles under the conjunctiva over the subsequent 24 hours will confirm the cause of the hypotony to be an over-draining bleb, while also temporarily correcting the hypotony. Care must be taken not to overfill, as a temporary dramatic elevation in IOP may occur if gas completely obstructs the trabeculectomy site.

Formal bleb revision

The biggest fear on the part of the surgeon and perhaps also the patient when revising a bleb for hypotony is that excessive wound healing will supervene and the IOP will elevate with loss of IOP control. In my personal experience, in eyes where hypotony has developed because of insufficient scleral flap healing, the greatest risk is often the opposite, namely, that the IOP will be temporarily restored and then become low again.

The nature of formal bleb revision will depend on the cause of the hypotony. When hypotony is due to scleral flap insufficiency alone (ie, a loose flap, a very thin flap, or a flap that has degenerated over time), either flap resuture or reinforcement with a donor scleral or commercially available pericardial patch graft may be all that is required. Hypotony in association with an unsatisfactory bleb morphology or a leaking bleb may require bleb excision and conjunctival advancement or replacement in addition to scleral flap resuture / reinforcement.

Scleral flap insufficiency

In general, the primary flow restrictor in a trabeculectomy procedure should be the scleral flap, rather than the conjunctiva. Early hypotony, especially in younger patients, is therefore often due to insufficiently tight suturing of the scleral flap, or a scleral flap that is compromised in some other way (eg, it is very thin so that suture bites create buttonholes in the flap). In that situation, I would open the conjunctiva at the limbus, taking care to avoid traumatizing the scleral flap when reflecting the conjunctiva, and gently dissect the Tenon away from the area of the flap.
The flap integrity should then be examined. If the eye is very soft, first BSS should be injected via a temporal corneal paracentesis to restore the IOP. Application of 2% fluorescein drops to the scleral flap will then identify areas of drainage that can be closed with 10-0 nylon sutures to eliminate drainage. In eyes where there has been hypotony, it is almost impossible to gauge how much flow is adequate, and therefore I recommend eliminating flow with sutures, either releasable or fixed, and removing or lasering them later, in combination with massage, to adjust the IOP downward if too high postoperatively.

The conjunctiva and Tenon can then be undermined, mobilized, and closed at the limbus.

Scleral flap insufficiency in combination with a dysmorphic or dysfunctional conjunctival drainage bleb

If the bleb morphology is unsatisfactory or if the bleb is leaking, then the control of hypotony is still effected by examining and closing the scleral flap in the manner described above, but in addition, the abnormal bleb is excised. More extensive conjunctival and Tenon mobilization is then likely to be required, with conjunctival closure as before.

Occasionally, there may be insufficient conjunctiva to close without excessive tension. This can be remedied by taking a conjunctival autograft from the inferior conjunctiva, which is then used to provide extra conjunctiva superiorly for closure.
Tube Exposure

Lama A Al-Aswad MD

Tube shunt exposure is an infrequent complication. In the literature there are only few papers discussing risk factors and management of this complication.

In my presentation I will discuss the following:

1. Definition
2. Types of patch grafts
3. Risk factors for exposure
4. Surgical management
5. Prevention

I will cite a few articles, including the following:


Complications With iStent Insertion

Steven R Sarkisian MD

Trabecular micro-bypass has a steep learning curve of approximately 5-20 cases, depending on a surgeon’s familiarity with gonioscopic surgery. This is a video presentation of some of the common complications I encountered during my first 5 cases with the iStent. Now, after several hundred cases, these complications no longer occur or are extremely rare, and my focus is no longer on implantation woes but on finding the premium location for implantation that maximizes outflow due to collector channel visualization. IOP-lowering results are therefore improved in a fashion directly proportional to the cumulative case volume of the surgeon.

This video demonstrates the following complications:

- Hyphema in the anterior chamber (AC) during implantation of the iStent, affecting visualization
- Chasing after a “loose” iStent in the AC after incomplete implantation
- Dislodging a well implanted iStent with a viscoelastic cannula
- Poor visualization of the AC due to red blood cells on the cornea and under the gonioprism due to having too posterior of a clear cornea wound causing bleeding of peripheral corneal vessels
- Poor visualization due to corneal stria from an underfilled AC or by excessive pushing on the eye with the gonioprism
- The stent having to be placed back in the inserter in the AC or on the cornea
- The stent getting trapped in the cornea wound while pulling the stent out of the eye with the inserter
- Difficulty implanting the stent due to steep an angle of entry, causing the stent to hit the scleral wall and causing the eye to move and prohibiting implantation

Other complications not included in the video but that can occur include:

- Grabbing the iris with the inserter and causing traumatic hyphema or an iridodialysis
- The patient under topical anesthesia moves unexpectedly, causing iridodialysis or stent dislodging or trabecular damage and hyphema or a Descemet tear or IOL dislocation.
- Implanting the stent in the ciliary body in a patient with an overfilled AC with poor angle landmarks due to no trabecular pigment
- A underfilled AC, causing the iris to obstruct the view of the angle anatomy
Ab Interno Ex-Press Shunt Removal

Davinder S Grover MD

Outline

I. Case Presentation

II. Description of Problem

III. Introduction of Ab Interno Technique for Ex-Press Shunt Removal
   A. Direct view
   B. Indirect view

IV. Possible Indication for Ex-Press Shunt Removal

V. Discussion
Aqueous Misdirection During Trabectome

Douglas J Rhee MD
Management of Intraoperative Suprachoroidal Hemorrhage

Steven D Vold MD
Case Presentations

Glaucoma After Descemet-Stripping Endothelial Keratoplasty
Lucy Q Shen MD

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Glaucoma and AMD With Anti-VEGF Treatment
Lucy Q Shen MD

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Special Consideration for Glaucoma Surgery in Patients After Corneal Transplantation: Options and Potential Pitfalls

Peter A Netland MD PhD

I. Key Features of Clinical Case
   A. Patient has pre-existing glaucoma.
   B. Patient has loss of vision due to steroid-induced increase of IOP after Descemet-stripping endothelial keratoplasty (DSEK) in fellow eye.
   C. Patient likely needs both glaucoma and corneal surgery.

II. Management Issues
   A. Patients with corneal surgery (PK and DSEK) have a high frequency of secondary glaucoma.
      1. IOP is elevated in approximately 9% to 35% of patients without pre-existing glaucoma.
      2. In patients with pre-existing glaucoma, approximately 29% to 80% develop elevated IOP.
   B. Elevated IOP after PK and DSEK may be due to multiple mechanisms.
      1. Steroid response is the major cause of IOP elevation after DSEK.
      2. DSEK surgeons may extend steroid use (less wound issues after DSEK compared with PK).
      3. IOP increase usually occurs 1-3 months after DSEK.
   C. Pre-existing glaucoma and increased IOP are risk factors for poor vision outcome and graft failure.
      1. Pre-existing glaucoma and increased IOP are risk factors after PK.
      2. After DSEK, mixed results of graft failure and poor vision in the literature
   D. Prior glaucoma surgery in patients treated with DSEK
      1. IOP controlled during postoperative period in reported studies
      2. Trabeculectomy and drainage implants have been reported as prior glaucoma surgery.

III. Surgical Options
   A. Staged surgery
      1. DSEK followed by glaucoma surgery
         a. High risk of IOP elevation in this patient
         b. This was the previous approach associated with vision loss in this patient.
      2. Glaucoma surgery followed by DSEK (or PK)
         a. Trabeculectomy or EX-Press under scleral flap are options.
         b. Glaucoma drainage implant surgery is an option (especially if patient needs contact lenses).
         c. Little information about microinvasive glaucoma surgery (MIGS) in the literature
         d. Little or no increased risk of intraoperative and early postoperative complications such as donor dislocation and graft failure after DSEK with previous glaucoma surgery
   B. Combined glaucoma and cornea surgery
      1. Reported for deep sclerectomy with DSEK
      2. Perioperative issues related to air bubble in DSEK
      3. Combined outcomes described with PK (good compared with staged), but not DSEK

IV. Conclusions
   A. Clinical case management
   B. Follow-up for clinical case
   C. Management is individualized, depending on corneal procedure, stage of disease, visual potential, and other factors.

Selected Readings


Comanaging Corneal Transplant Patients After Glaucoma Surgery: Preserving Corneal Endothelium and Vision Rehabilitation

Samir Melki MD PhD, Ali Fadlallah MD

I. Penetrating Keratoplasty (PK) in Patients With Prior Trabeculectomy or Tube Shunt

A. PK is the most common transplant procedure in the United States, with > 42,000 corneal transplants performed in 2010 alone.1

B. Patients with corneal transplants are at increased risk for glaucoma because of preoperative (ocular comorbidities) and postoperative (surgical trauma, inflammation, and chronic steroid therapy) risk factors.

C. In an eye with a functional trabeculectomy, the most frequent complication after PK is IOP spike (IOP ≥ 25 mmHg within the first 3 postoperative months in > 50% of eyes). Retained viscoelastic is cited as one cause for early postoperative IOP spikes.2

D. There is no significant difference in graft rejection or failure rates for PK between eyes with prior trabeculectomy and those with no prior glaucoma surgery at 4 years’ follow-up.3 There is no study on long-term graft survival combining trabeculectomy with Ex-Press shunt. However, the Descemet membrane may interfere with aqueous humor drainage through an Ex-Press shunt.4

E. Graft survival is lower in eyes with tube shunts than in eyes without glaucoma or with glaucoma that is managed medically.5,6

1. Graft survival in the setting of PK and tube shunt ranges from 58.5% to 96% at 1 year.

2. Endothelial cell loss is proposed to occur intraoperatively and postoperatively from blinking, eye rubbing, or eye movements.

3. The anterior chamber may also be shallow, with tube-corneal touch, which increases the risk of endothelial touch.

4. Graft survival may be lower if a tube shunt is placed after PK than if it is placed concurrent with or prior to PK.

5. Several reports propose to splint the anterior chamber tube away from the cornea or to implant the tube shunts into the ciliary sulcus to distance the tube from the cornea.7,8

II. Endothelial Keratoplasty (EK) in Patients With Prior Trabeculectomy or Tube Shunt

A. EK allows for selective replacement of damaged endothelial cells, using stromal layer with endothelium and Descemet membrane for Descemet-stripping endothelial keratoplasty (DSEK) or only donor Descemet membrane with endothelium for Descemet manual endothelial keratoplasty (DMEK). No corneal sutures are needed, and an air bubble is used to attach the partial donor graft.

1. EK remains the preferred technique for replacing endothelium in corneal endothelial disease, mainly in Fuchs endothelial dystrophy.9

2. EK has come into favor compared to PK, offering a faster visual recovery with minimal refractive change, a reduction in intraoperative complications, and a preserved ocular surface.10

3. There is no controlled, randomized clinical trial showing that graft rejection occurs more frequently after PK than after EK.10

4. Prednisolone remains the treatment of choice for management and treatment of graft rejection.10

5. There are no differences in prophylactic steroid treatment for PK and EK.10

B. The most common postoperative complication in eyes with functional trabeculectomy undergoing DSEK or DMEK remains IOP spike. Pupillary block may develop from the anterior chamber air bubble, and air posterior to the iris may lead to angle closure.

C. Other common complications include graft detachment/dislocation, at a rate around 25%.

1. Dislocations may be strongly correlated with postoperative hypotony.11

2. In patients with prior trabeculectomy or tube shunt, air may escape to the subconjunctival space postoperatively, which may result in graft dislocation.12

3. At the end of DSEK surgery, leaving a larger-than-usual air bubble in eyes with prior trabeculectomy and low preoperative IOPs should be considered.

D. Studies report higher rates of secondary graft failure in eyes with a tube shunt or trabeculectomy but comparable rates of primary graft failure and graft dislocations to patients without glaucoma.13

1. Rate of DSEK failure in eyes with glaucoma ranges from 6% to 60%.13

2. This increases further with multiple tube shunts or the presence of uveitis.14

3. Other groups found higher rates of primary graft failure and/or dislocation in eyes with trabeculectomy or tube shunt.15
E. DMEK has also been performed in the setting of prior glaucoma surgery.
1. DMEK may be advantageous over DSEK.
2. Additional stroma from DSEK would crowd the anterior chamber and increase endothelial touch in eyes with existing tube shunts.16-17

III. Vision Rehabilitation After Corneal Transplantation in Eyes With Existing Trabeculectomy / Tube Shunts
A. Vision rehabilitation after corneal transplantation depends mainly on corneal clarity, astigmatism management, presence of concomitant cataract, macular integrity, and optic nerve status.
B. There is good evidence that glaucoma is a risk factor for both DSEK and PK failure; this may have influence on postoperative corneal clarity at short- and long-term follow-up.18
C. With careful fitting, the presence of a filtering bleb need not be a contraindication to contact lens wear, particularly when a smooth-edged, rigid gas permeable (RGP) daily wear lens is used to treat resultant astigmatism.19
D. There is no clear evidence that concomitant cataract should be treated during or after the corneal transplant.20
E. Cystoid macular edema remains a major complication (9.6%) after corneal transplantation and is mainly associated with combined surgery.20
F. Special care for IOP spike following corneal transplantation should be undertaken to prevent additional damage of the optic nerve head.

References
Anti-VEGF Agents and Their Effect on IOP

*K Bailey Freund MD*

I. Acute

**Factors influencing post-injection IOP spikes**

1. Pre-injection IOP
2. Injection volume
3. Phakic vs. pseudophakic state (higher in phakic eyes)
4. History of glaucoma
5. Axial length and patient age are poor predictors of IOP spike.
6. Needle bore size (higher with smaller needle gauge, reduced reflux)

**Management of post-injection IOP spikes**

1. Observation (IOP usually < 25 mmHg within 30-60 minutes)
2. Pre- and postoperative medical therapies
3. Paracentesis (rarely needed)

II. Chronic

**Sustained IOP elevations related to intravitreal anti-VEGF therapy**

1. The incidence of IOP elevation after anti-VEGF therapy
2. Was also observed in the pivotal trials of ranibizumab for neovascular AMD
3. Max IOP 23 mmHg to 70 mmHg in these reports
4. Incisional filtration procedures performed in 7 eyes
5. No consistent definition of sustained IOP elevation
6. Some reports have not found an association. (These tend to be smaller cohorts with eyes having shorter follow-up and receiving fewer injections.)

**Risk factors for sustained IOP elevation related to intravitreal anti-VEGF therapy**

1. Higher number of intravitreal injections
2. Shorter interval between injections
3. Pre-existing glaucoma
4. Family history of glaucoma
5. History of intravitreal or topical steroids
6. Possibly higher with compounded bevacizumab
7. Possibly lower with aflibercept

**Potential mechanisms for sustained IOP elevation related to intravitreal anti-VEGF therapy**

1. Impaired outflow due to microparticles (silicone, protein aggregates)
2. Mechanical trauma: Impaired outflow due to repeated IOP spikes
3. Pharmacologic effect of VEGF blockade on:
   a. Trabeculocytes
   b. Trabecular meshwork extracellular matrix
   c. Episcleral venous pressure
   d. Uveoscleral outflow
4. Inhibition of nitric oxide synthase (decreased outflow)
5. Inflammatory mechanism / trabeculitis
6. Idiopathic

III. Possible Ways to Prevent IOP Elevation in Patients With Pre-existing Glaucomatous Optic Nerve Damage

A. Manage pre-injection IOP
B. Reduce injection volume
C. Use lower needle gauge (more reflux)
D. Give fewer injections
E. Lengthen interval between injections
F. Choice of anti-VEGF agent

**Selected Readings**


I. Is the Visual Field Defect From Glaucoma, or AMD?
II. What Are the Treatment Options for This Patient?
   A. Optimizing medical management
   B. Laser therapy
   C. Surgical therapy
III. Should Anti-VEGF Treatment Continue?
IV. Advice for My Retina Colleagues When Using Anti-VEGF Agents in Glaucoma Patients

Selected Readings
Uveitic Glaucoma in a Child With Juvenile Idiopathic Arthritis

Gary N Holland MD and Anne L Coleman MD PhD

Case for Discussion

FP is currently a 6.5-year-old white girl who was diagnosed with pauciarticular juvenile idiopathic arthritis (JIA) at 2 years of age because of pain and swelling in one knee. Her arthritis came under good control initially with use of oral naproxen, and since that time occasional exacerbations of joint symptoms have responded well to courses of NSAIDs. She currently has no joint symptoms and is an active child.

Just before her third birthday, the patient’s parents had noticed that her left pupil was not round and took her to a pediatric ophthalmologist for evaluation. She was found to have cells in both anterior chambers, and she had posterior synechiae in the left eye, as the cause of her irregular pupil. According to the parents, her anterior uveitis improved at that time with a course of topical prednisolone acetate 1%, and the drops were tapered and discontinued after a few weeks, but she has had repeated “recurrences” that required additional courses of topical corticosteroids. During the past year, she has been left on prednisolone acetate 1% three times daily, and they have been told that her uveitis is “quiet.”

According to the patient’s parents, at her last eye examination, IOP in the left eye had increased to 27 mmHg, and the pediatric ophthalmologist recommended switching from prednisolone acetate 1% eye drops to loteprednol eye drops because it is less likely to cause elevated pressure as a side-effect. The parents are now requesting a second opinion about treatment.

On your examination, visual acuity without correction is 20/20 O.U. IOPs are 21 mmHg O.D. and 27 mmHg O.S., as reported to the parents by the pediatric ophthalmologist. On slitlamp biomicroscopic examination, there are 1+ cells in both anterior chambers. In addition, examination of the left eye reveals band keratopathy near the limbus at the 3:00 and 9:00 positions and the aforementioned posterior synechiae, without pupillary seclusion. The patient is uncooperative for indirect ophthalmoscopy.

Initial Questions for Drs. Coleman and Holland

- What are possible mechanisms for pressure elevation in the left eye?
- How should the patient be evaluated at this point?
- What should be done in terms of anti-inflammatory treatment initially?

Glaucoma Associated with Chronic Anterior Uveitis in Children: Additional Issues for Discussion

Glaucoma is a common complication of chronic anterior uveitis in children, as seen in those with JIA (incidence, 7.4/100 eye-years of follow-up1). It appears to be more common in children with early onset of uveitis and in eyes with more severe inflammation and with other complications of uveitis.
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