Glaucoma 2023

Glaucoma Care at the Golden Gate and Beyond

Under Pressure®

Program Directors
Teresa C Chen MD and Steven L Mansberger MD MPH

In conjunction with the American Glaucoma Society

Moscone Center
San Francisco, California
Friday, Nov. 3, 2023

Presented by the American Academy of Ophthalmology

Supported by an unrestricted educational grant from Sight Sciences

Glaucoma 2023 Planning Group
Teresa C Chen MD
Program Director
Steven L Mansberger MD MPH
Program Director
Robert T Chang MD
Anna T Do MD
Christopher A Girkin MD MSPH
Leon W Herndon Jr MD
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Mary Qiu MD
Manjool M Shah MD
Ramya N Swamy MD MPH
Richard Dwight Ten Hulzen MD
Luis E Vazquez MD PhD

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An Ongoing Commitment to Education

Covering all subspecialties, the BCSC books are an invaluable resource not just for residents but also for practicing ophthalmologists.

The BCSC books are authored and revised by expert ophthalmic subspecialists, ensuring that the information presented is accurate, up-to-date, and authoritative. Please join us in thanking these volunteers for their hard work and commitment to education.

Faculty for BCSC Section 10 Glaucoma

Michael V. Boland, MD, PhD 
Chair  
Boston, Massachusetts

JoAnn A. Giaconi, MD 
Los Angeles, California

Richard K. Lee, MD, PhD 
Miami, Florida

Shan C. Lin, MD  
San Francisco, California

Shalini Sood, MD  
Cleveland, Ohio

Kelly Walton Muir, MD, MHSc  
Durham, North Carolina

Derek S. Welsbie, MD  
La Jolla, California

All Ophthalmologists are Invited to Help

The BCSC is created by ophthalmologists for ophthalmologists. As such, the writing committees are always looking for and considering new members. No previous experience necessary. As part of BCSC’s commitment to diversity, we seek individuals who are good at writing and editing, and represent all aspects of the AAO’s diverse membership, including gender, ethnicity, geography, and private versus academic practice. If you are interested in volunteering for a BCSC writing committee, please submit a CV and indicate your area of interest to: aao志愿者@aao.org.
On behalf of the American Academy of Ophthalmology and the American Glaucoma Society (AGS), it is our pleasure to welcome you to San Francisco and Glaucoma 2023: Glaucoma Care at the Golden Gate and Beyond.
Leon W Herndon Jr MD
Aerie Pharmaceuticals, Inc.: C
Alcon Laboratories, Inc.: C
Allergan, Inc.: C,L
Elios: C
Equinox: C
Genentech: C
Glaukos Corp.: C,L
Graybug: C
New World Medical, Inc.: C,L
Ocular Therapeutix: C,S
Sight Sciences, Inc.: C

Mary Qiu MD
None

Ramya N Swamy MD MPH
None

Manjool M Shah MD
Alcon Laboratories, Inc.: C
Allergan/AbbVie: C
Elios: C
Glaukos Corp.: C
Katena Products, Inc.: C
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ONL Therapeutics: C

Robert J Noecker MD MBA
AbbVie: C,L
Alcon Laboratories, Inc.: C,L
Bausch + Lomb: C,L
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Glaukos Corp.: C,S
Iridex: C,L
New World Medical, Inc.: C,L
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Santen, Inc.: C
Sight Sciences, Inc.: C,SO,L
Thea: C

Luis E Vazquez MD PhD
New World Medical, Inc.: L

Richard Dwight Ten Hulzen MD
None
Subspecialty Day 2023 Advisory Committee

R Michael Siatkowski MD, Associate Secretary (Pediatric Ophthalmology)
None

Bennie H Jeng MD (Secretary for Annual Meeting)
GlaxoSmithKline: C
Kiora: US

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NGM: S
Novartis Pharma AG: C
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Regeneron Pharmaceuticals, Inc.: C,S
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Spring Vision: S
Stealth Biotherapeutics: S
Taylor & Francis (CRC Press): P
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Viridian: C

Shahzad I Mian MD (Cornea)
Kowa American Corp.: S
Novartis: S
VisionCare, Inc.: S

Jody R Piltz MD (Glaucoma)
Aerie Pharmaceuticals, Inc.: C,L
Alcon Laboratories, Inc.: C,L
Nanoscope Therapeutics: C

Sonia H Yoo MD (Refractive Surgery)
Carl Zeiss Meditec: C
Dermavant: C
Oyster Point Pharma: C

AAO Staff

Mecca Boutte
None

Ann L’Estrange
None

Melanie Rafaty
None

Debra Rosencrance
None

Beth Wilson
None
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CME Credit

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

Glaucoma Subspecialty Day 2023 Learning Objectives
Upon completion of this activity, participants should be able to:
■ Demonstrate familiarity with controversial management issues and current gaps in evidence-based glaucoma care
■ Evaluate the status of glaucoma imaging and test interpretation, as well as their role in diagnosing and managing glaucoma
■ Demonstrate familiarity with current issues in medical, laser, and surgical therapy for glaucoma and how these therapies affect other eye diseases
■ Recognize factors that complicate the care of the adult and pediatric glaucoma patient

Glaucoma Subspecialty Day 2023 Target Audience
This activity has been designed to meet the educational needs of general ophthalmologists, glaucoma specialists and other ophthalmologic subspecialists, and allied health personnel who are involved in the management of glaucoma patients.

Teaching at a Live Activity
Teaching an instruction course 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. To obtain an application form, please contact the AMA at www.ama-assn.org.

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

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Subspecialty Day 2023 CME Credit
The American Academy of Ophthalmology is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide CME for physicians.

Friday Subspecialty Day Activity: Glaucoma, Neuro-Ophthalmology, Ocular Oncology and Pathology, Refractive Surgery, and Retina (Day 1)
The Academy designates this Other (blended live and enduring material) activity for a maximum of 12 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Saturday Subspecialty Day Activity: Cornea, Oculofacial Plastic Surgery, and Retina (Day 2)
The Academy designates this Other (blended live and enduring material) activity for a maximum of 12 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Physicians registered as In Person and Virtual are eligible to claim the above CME credit.

Attendance Verification for CME Reporting
Before processing your requests for CME credit, the Academy must verify your attendance at AAO 2023 and/or Subspecialty Day. Badges are no longer mailed before the meeting. Picking up your badge onsite will verify your attendance.

How to Claim CME
Attendees can claim credits online.
For AAO 2023, you can claim CME credit multiple times, up to the 50-credit maximum, through March 29, 2024. You can claim some in 2023 and some in 2024, or all in the same year.
For Subspecialty Day, you can claim CME credit multiple times, up to the 12-credit maximum per day, through March 29, 2024. You can claim some in 2023 and some in 2024, or all in the same year.
You do not need to track which sessions you attend, just the total number of hours you spend in sessions for each claim. You can view content in the virtual meeting through March 1, 2024.

Academy Members
CME transcripts that include AAOE Half-Day Coding Sessions, Subspecialty Day, and/or AAO 2023 credits will be available to Academy members through the Academy’s CME Central web page.

The Academy transcript cannot list individual course attendance. It will list only the overall credits claimed for educational activities at AAOE Half-Day Coding Sessions, Subspecialty Day, and/or AAO 2023.

Nonmembers
The Academy provides nonmembers with verification of credits earned and reported for a single Academy-sponsored CME activity.

Proof of Attendance
You will be able to obtain a CME credit reporting/proof-of-attendance letter for reimbursement or hospital privileges, or for nonmembers who need it to report CME credit:

Academy Members
When you claim CME credits and complete the evaluation, you will be able to print a certificate/proof-of-attendance letter from your transcript page. Your certificate will also be emailed to you.

Nonmembers
When you claim CME credits and complete the evaluation, a new browser window will open with a PDF of your certificate. Please disable your pop-up blocker. Your certificate will also be emailed to you.

CME Questions
Send your questions about CME credit reporting to cme@aao.org. For Continuing Certification questions, contact the American Board of Ophthalmology at MOC@abpo.org.
The American Glaucoma Society (AGS) Subspecialty Day Lecture
It Began in San Francisco—How Treatment of Childhood Glaucoma Continues to Evolve

James D Brandt MD

FRIDAY, NOV. 3, 2023
11:51 AM – 12:21 PM

James D Brandt MD is Professor of Ophthalmology & Vision Science, Director of the Glaucoma Service, and Vice-Chair for International Programs & New Technologies at the University of California, Davis in Sacramento, California.

After receiving a BS from Yale University and an MD from Harvard Medical School, he pursued a two-year postdoctoral research fellowship in glaucoma-related pharmacology and cell biology at the Schepens Eye Research Institute in Boston before his residency at the University of Southern California/LA County/Doheny Eye Institute. He completed a glaucoma fellowship at the Wills Eye Hospital before joining the UC Davis faculty in 1989.

Dr. Brandt’s clinical practice is limited to glaucoma, with a particular focus on infantile and pediatric glaucoma. He is a frequent volunteer faculty member with ORBIS International (www.orbis.org), traveling worldwide to train glaucoma specialists in the management of pediatric glaucoma.

Dr. Brandt’s research interests focus primarily on the material properties of the eye as they affect the measurement of IOP and in the physiology of outflow resistance. Dr. Brandt has served as the principal investigator of numerous clinical trials, including the Ocular Hypertension Treatment Study (OHTS). As an OHTS principal investigator, Dr. Brandt initiated the effort to measure central corneal thickness (CCT) as a potential risk factor for glaucoma and has continued to investigate the role of CCT in glaucoma diagnosis and pathogenesis.

Dr. Brandt has served in several leadership roles for the American Glaucoma Society, and in 2012 he was selected to give the 13th Clinician-Scientist Lecture at the annual meeting of the AGS. In 2018 he was presented with the AGS Humanitarian Award for his work in the developing world on childhood glaucoma.
Faculty

Inas F Aboobakar MD
Boston, MA

Michael V Boland MD PhD
Boston, MA

Teresa C Chen MD
Boston, MA

Iqbal K Ahmed MD
Mississauga, Canada

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Sacramento, CA

Anna T Do MD
La Jolla, CA

Sally Liu Baxter MD
FPO, AP

Robert T Chang MD
Los Altos, CA

Beth Edmunds MD PhD
Portland, OR

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Atlanta, GA

Ta Chen Peter Chang MD
Miami, FL

Brian A Francis MD
Pasadena, CA
Steven J Gedde MD  
Miami, FL

Ying Han MD PhD  
San Francisco, CA

Karen M Joos MD PhD  
Nashville, TN

Christopher A Girkin MD  
Birmingham, AL

Leon W Herndon Jr MD  
Durham, NC

Jessica M Kang MD  
Chicago, IL

Sylvia L Groth MD  
Nashville, TN

Donald C Hood PhD  
New York, NY

Courtney L Kraus MD  
N Bethesda, MD

Davinder S Grover MD  
Dallas, TX

Murray A Johnstone MD  
Bainbridge Island, WA

Rachel H Lee MD MPH  
New York, NY
Richard Lee MD  
Miami, FL

Felipe A Medeiros MD  
South Miami, FL

Yvonne Ou MD  
San Francisco, CA

Michael Lin MD  
Brookline, MA

Paula Anne Newman-Casey MD MS  
Ann Arbor, MI

Mary Qiu MD  
Chicago, IL

Steven L Mansberger MD MPH  
Portland, OR

Kanwal K Nischal MBBS  
Pittsburgh, PA

Anthony D Realini MD  
Morgantown, WV

Catherine M Marando MD  
Concord, NH

Robert J Noecker MD  
Easton, CT

Jullia A Rosdahl MD PhD  
Chapel Hill, NC
Ahmara G Ross MD  
Philadelphia, PA

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Toronto, ON, Canada

Arsham Sheybani MD  
Saint Louis, MO

Osamah J Saeedi MD  
Baltimore, MD

Leonard K Seibold MD  
Aurora, CO

Aakriti Garg Shukla MD  
New York, NY

Thomas W Samuelson MD  
Minneapolis, MN

Manjool M Shah MD  
Ann Arbor, CT

Erin G Sieck MD  
Saint Louis, MO

Steven R Sarkisian MD  
Oklahoma City, OK

Lucy Q Shen MD  
Boston, MA

Arthur J Sit MD  
Rochester, MN
Catherine Q Sun MD  
San Francisco, CA

Ramya N Swamy MD  
North Potomac, MD

Richard D Ten Hulzen MD  
Jacksonville Beach, FL

Luis E Vazquez MD PhD  
Miami, FL

Sarah Wellik MD  
Miami, FL
Ask a Question or Respond to a Poll During the Meeting Using the Mobile Meeting Guide

To ask the moderator a question or respond to a poll, follow the directions below.

- Access at www.aao.org/mobile
- Select “Polls/Q&A”
- Select “Current Session”
- Select “Interact with this session (live)” to open a new window
- Choose “Ask a Question”
- Choose “Answer Poll”
# Glaucoma Subspecialty Day 2023: Glaucoma Care at the Golden Gate and Beyond

## FRIDAY, NOV. 3, 2023

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Speaker(s)</th>
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<tbody>
<tr>
<td>7:00 AM</td>
<td>Continental Breakfast</td>
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</tr>
<tr>
<td>8:00 AM</td>
<td>Welcome and Introductions</td>
<td>Teresa C Chen MD</td>
</tr>
<tr>
<td>8:02 AM</td>
<td>American Glaucoma Society Introduction</td>
<td>Leon W Herndon Jr MD</td>
</tr>
<tr>
<td>8:04 AM</td>
<td>AGS Cares</td>
<td>Leon W Herndon Jr MD</td>
</tr>
<tr>
<td>8:09 AM</td>
<td>Announcements</td>
<td>Steven L Mansberger MD MPH</td>
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</tbody>
</table>

### Section I: Diagnostics #HVF #OCT @Goldengate

Moderators: Anna T Do MD and Steven L. Mansberger MD MPH

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Speaker(s)</th>
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</thead>
<tbody>
<tr>
<td>8:11 AM</td>
<td>Should Virtual Reality Visual Fields Be Prime Time?</td>
<td>Steven R Sarkisian MD</td>
</tr>
<tr>
<td>8:18 AM</td>
<td>Genetics Now or Later?</td>
<td>Inas F Aboobakar MD</td>
</tr>
<tr>
<td>8:25 AM</td>
<td>AI Can Do Anything . . . Use It for Glaucoma?</td>
<td>Michael V Boland MD PhD</td>
</tr>
<tr>
<td>8:32 AM</td>
<td>What Does OCT Angiography Add to Our Glaucoma Toolbox?</td>
<td>Lucy Q Shen MD</td>
</tr>
<tr>
<td>8:39 AM</td>
<td>Pearls and Pitfalls of OCT Testing</td>
<td>Richard K Lee MD</td>
</tr>
<tr>
<td>8:46 AM</td>
<td>Glaucoma and Driving Safety</td>
<td>Felipe A Medeiros MD</td>
</tr>
<tr>
<td>8:53 AM</td>
<td>Audience Questions and Interactive Presentation of Diagnostic Cases</td>
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### Section II: Escaping From OR Alcatraz—Update on Medications and Lasers

Moderators: Richard D Ten Hulzen MD and Luis E Vazquez MD

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Speaker(s)</th>
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<tbody>
<tr>
<td>9:08 AM</td>
<td>Pathophysiology of Aqueous Outflow Regulation: Evolving Concepts</td>
<td>Murray A Johnstone MD</td>
</tr>
<tr>
<td>9:15 AM</td>
<td>Emerging Glaucoma Meds</td>
<td>Arthur J Sit MD</td>
</tr>
<tr>
<td>9:22 AM</td>
<td>Medication Adherence</td>
<td>Paula Anne Newman-Casey MD MS</td>
</tr>
<tr>
<td>9:29 AM</td>
<td>Selective Laser Trabeculoplasty Outcomes as Primary Glaucoma Treatment</td>
<td>Sylvia L Groth MD</td>
</tr>
<tr>
<td>9:36 AM</td>
<td>Cold Feet? Why Selective Laser Trabeculoplasty Should Be a First-line Treatment</td>
<td>Anthony D Realini MD</td>
</tr>
<tr>
<td>9:43 AM</td>
<td>How Selective Laser Trabeculoplasty and Meds Reduce the Diurnal Fluctuations in IOP of Glaucoma Patients</td>
<td>Leonard K Seibold MD</td>
</tr>
<tr>
<td>9:50 AM</td>
<td>Glaucoma Medical Management Before and After Surgery</td>
<td>Rachel H Lee MD MPH</td>
</tr>
<tr>
<td>9:57 AM</td>
<td>Q&amp;A</td>
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<tr>
<td>10:08 AM</td>
<td>United for Sight: A Vision for Effective Advocacy</td>
<td>Sarah Wellik MD</td>
</tr>
<tr>
<td>10:13 AM</td>
<td>REFRESHMENT BREAK</td>
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</table>

### Section III: Fisherman’s Wharf—Catching Up on New Ideas

Moderators: Robert T Chang MD and Christopher A Girkin MD

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Speaker(s)</th>
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<tbody>
<tr>
<td>10:43 AM</td>
<td>Why We Should Participate in Glaucoma Registries</td>
<td>Sally Liu Baxter MD</td>
</tr>
<tr>
<td>10:50 AM</td>
<td>New Frontier in Neovascular Glaucoma</td>
<td>Catherine Q Sun MD</td>
</tr>
<tr>
<td>Time</td>
<td>Session</td>
<td>Speaker</td>
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<tr>
<td>10:57 AM</td>
<td>Emerging Functional Outcomes With Minimally Invasive Glaucoma Surgery Studies</td>
<td>Iqbal K Ahmed MD</td>
</tr>
<tr>
<td>11:04 AM</td>
<td>Can OCT Predict Field Loss? When Should We Test the Central Field?</td>
<td>Donald C Hood PhD</td>
</tr>
<tr>
<td>11:11 AM</td>
<td>Fine Tuning Ciliary Body Ablation and Other Outflow: Cyclophotocoagulation (CPC), Endoscopic CPC vs. MicroPulse P3 CPC</td>
<td>Osamah J Saeedi MD</td>
</tr>
<tr>
<td>11:18 AM</td>
<td>Helping Glaucoma Patients at Home With Tools</td>
<td>Yvonne Ou MD</td>
</tr>
<tr>
<td>11:25 AM</td>
<td>How Direct Selective Laser Trabeculoplasty May Change Glaucoma Care</td>
<td>Thomas W Samuelson MD</td>
</tr>
<tr>
<td>11:32 AM</td>
<td>Cutting-Edge Glaucomatology: Top Discoveries From ARVO 2023</td>
<td>Ahmara G Ross MD</td>
</tr>
<tr>
<td>11:39 AM</td>
<td>Discussion</td>
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**The American Glaucoma Society Subspecialty Day Lecture**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
<th>Speaker</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>11:49 AM</td>
<td>Introduction of the Lecturer</td>
<td>Leon W Herndon Jr MD</td>
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<tr>
<td>11:51 AM</td>
<td>It Began In San Francisco—How Treatment of Childhood Glaucoma Continues to Evolve</td>
<td>James D Brandt MD</td>
<td>29</td>
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<tr>
<td>12:21 PM</td>
<td>Presentation of the Award</td>
<td>Leon W Herndon Jr MD</td>
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<tr>
<td>12:22 PM</td>
<td>LUNCH</td>
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**Section IV: Reaching New Pacific Heights in Surgery—How to Do It Right**

Moderators: Manjool M Shah MD and Ramya N Swamy MD

<table>
<thead>
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<th>Time</th>
<th>Session</th>
<th>Speaker</th>
<th>Page</th>
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<tbody>
<tr>
<td>1:42 PM</td>
<td>Anesthesia-Perioperative Considerations—How to Optimize Surgical Preparation</td>
<td>Catherine M Marando MD</td>
<td>31</td>
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<tr>
<td>1:50 PM</td>
<td>Trabeculectomy + Xen + Blebs—How to Do It Right</td>
<td>Erin G Sieck MD</td>
<td>34</td>
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<tr>
<td>1:58 PM</td>
<td>Glaucoma Drainage Device, Part 1: Nonvalved Implants—How to Do It Right</td>
<td>Ying Han MD PhD</td>
<td>35</td>
</tr>
<tr>
<td>2:06 PM</td>
<td>Glaucoma Drainage Device, Part 2: Valved Implants + Other Special Considerations—How to Do It Right</td>
<td>Brian A Francis MD</td>
<td>36</td>
</tr>
<tr>
<td>2:14 PM</td>
<td>Angle-Based Surgery—Best Practices</td>
<td>Arsham Sheybani MD</td>
<td>37</td>
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<tr>
<td>2:22 PM</td>
<td>Newer Devices in the Pipeline and Available Outside the United States</td>
<td>Matthew Bryan Schlenker MD</td>
<td>38</td>
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<tr>
<td>2:30 PM</td>
<td>Discussion</td>
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</table>

**Section V: Pediatric Glaucoma—The Earthquake in Our Practice**

Moderators: Teresa C Chen MD and Robert J Noecker MD

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<th>Time</th>
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<tbody>
<tr>
<td>2:40 PM</td>
<td>Congenital Glaucoma: Goniotomy Alternatives; and What to Do When Goniotomy Fails</td>
<td>Allen Dale Beck MD</td>
<td>39</td>
</tr>
<tr>
<td>2:47 PM</td>
<td>Aphakic Glaucoma, a Primer: Infant Aphakia Treatment Study, Best Practices</td>
<td>Courtney L Kraus MD</td>
<td>40</td>
</tr>
<tr>
<td>2:54 PM</td>
<td>Uveitic Glaucoma: Goniotomy’s Role, Steroid Response</td>
<td>Kanwal K Nischal MBBS</td>
<td>42</td>
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<tr>
<td>3:01 PM</td>
<td>Juvenile Open-Angle Glaucoma: How Is Treatment Different From That of Adult OAG?</td>
<td>Beth Edmunds MD PhD</td>
<td>43</td>
</tr>
<tr>
<td>3:08 PM</td>
<td>Exam/EUA/IOP: How-to, Anesthesia, CCT, Tonometer Types</td>
<td>Karen M Joos MD PhD</td>
<td>45</td>
</tr>
<tr>
<td>3:15 PM</td>
<td>How Are Trabs and Tubes in Kids Different Than Those in Adults?</td>
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### Section VI: Winding Through Lombard Street’s Difficult Turns—What to Do When Surgery Goes Wrong

Moderators: Leon W Herndon Jr MD and Mary Qiu MD

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<td>Davinder S Grover MD</td>
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<td>Jullia A Rosdahl MD PhD</td>
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Should Virtual Reality Visual Fields Be Prime Time?

*Steven R Sarkisian Jr MD*

**Introduction**

Visual field testing is a central aspect of glaucoma diagnosis and management. The “gold standard” has been the Humphrey Field Analyzer, or HFA (Carl Zeiss Meditech, Inc.). However, virtual modalities have been growing in use in the last several years and offer distinct advantages to conventional automated perimetry.1-11

This presentation will discuss advantages and disadvantages of virtual field testing in a real-world setting, with practical applications for how they might best fit into your practice.

**References**

Genetics Now or Later?

Inas F Aboobakar MD

I. Introduction

A. Genetic factors play an important role in both early-onset (age ≤ 40) and adult-onset (age > 40) forms of glaucoma.1

1. Early-onset: Mendelian inheritance pattern (typically autosomal dominant or autosomal recessive), where a single gene mutation is sufficient for disease development.


B. Genetics will likely play an important role in clinical glaucoma practice in the years to come, so it is critical we all become familiar with the basics!

1. Gene-based panels are already available for early-onset glaucoma.

2. Polygenic risk scores (PRSs) will likely aid in disease diagnosis and risk stratification for adult-onset glaucoma.

3. Gene-based therapies may provide targeted treatment options in the future.

II. Early-Onset Glaucoma

A. Causal gene mutations have been identified for various forms of early-onset glaucoma, and additional genes will likely be identified in the near future:1

1. Primary congenital glaucoma: CYP1B1, LTBP2, TEK, ANGPT1

2. Axenfeld-Rieger syndrome: PITX2, FOXC1

3. Aniridia: PAX6

4. Juvenile open-angle glaucoma: MYOC, EFEMP1, THBS1

5. Pigment dispersion syndrome/glaucoma: PMEL, GSAP, GRM5/TYR

B. Translational studies in mouse models and human eyes ex vivo demonstrate that gene editing of mutant Myocilin (MYOC) lowers IOP and prevents further glaucoma damage.2 Clinical trials in human patients have not yet been performed. Similar gene-editing approaches can likely be applied to other early-onset glaucoma genes as well.

C. Existing gene panels test for mutations in early-onset glaucoma genes, which is useful for screening first-degree relatives. (Carriers of a causal mutation warrant close surveillance, while the risk for non-carriers is similar to that of the general population.)

It is crucial to ensure a genetic testing lab is CLIA-certified prior to ordering testing. Providers who are not familiar with interpretation of genetic testing results can consider referring patients to a genetic counselor.

III. Adult-Onset Glaucoma

A. Unlike early-onset glaucoma, where 1 mutation is sufficient for disease development, adult-onset glaucoma is a complex inherited disease, where multiple gene variants and environmental factors interact to influence disease development.

B. PRSs enable evaluation of the cumulative effect of all glaucoma-associated gene variants in aggregate.

1. Primary open-angle glaucoma (POAG): at least 127 genetic loci identified to date

   a. A multitrait PRS for POAG is associated with higher odds of developing POAG, earlier age at disease diagnosis, lower retinal nerve fiber layer thickness, and greater need for trabeculectomy.3

   b. Prospective studies in an Australian cohort of suspect and early glaucoma demonstrate that higher POAG PRSs are associated with earlier initiation/escalation of glaucoma treatment4 and faster visual field progression.5

2. Primary angle-closure glaucoma (PACG)

   a. 8 genetic loci identified to date

   b. A PRS for PACG is associated with more severe disease in a cross-sectional cohort study of individuals of Chinese ancestry.6

IV. Conclusions and Future Directions

A. Genetics has the potential to revolutionize clinical glaucoma care, paving the way for personalized approaches to disease diagnosis, risk stratification, monitoring, and therapy.

B. Important next steps and unmet needs for making precision glaucoma care a reality:

1. Genetic studies to date have largely included white European populations; ongoing efforts to recruit ethnically diverse populations for genetic studies will enable identification of novel loci and ensure that PRSs perform equally well in patients of all ethnic backgrounds.
2. Further prospective evaluation of PRSs are needed in population-based cohorts to fully characterize their predictive power.

3. Ongoing translational work will facilitate targeted gene therapies, particularly for early-onset forms of glaucoma, where a single gene defect causes disease development.

4. Ethical, legal, and social ramifications of genetic testing and gene therapy warrant consideration.

References


AI Can Do Anything . . . Use It for Glaucoma?

Michael V Boland MD PhD

Artificial intelligence (AI) re-emerged as a potentially transformative technology over the past several years. Ophthalmology has played an important role in the emergence of AI in medicine, with the first FDA-approved, autonomous AI system for screening fundus photographs for diabetic retinopathy.

Where is glaucoma in this AI resurgence, what challenges do we (and other subspecialties) face, and where will we likely be regarding AI in the next several years?

Historical Perspective on AI in Glaucoma (We Have Been Here Before.)

Current work represents at least the second time AI has been applied to diagnosing glaucoma and identifying its worsening. We saw an initial application of AI in the late 1990s, when artificial neural networks became available. The results of those systems were not dramatically better than traditional statistical methods and so did not end up transforming glaucoma care. We did have our first experience with the challenge of lacking a clear computable definition of glaucoma even then, and that problem plagues us still.

What Glaucoma-Related Problems Are Being Addressed Now?

As before, AI is being applied to the diagnosis of glaucoma, though we now are able to use more complex data like OCT images and optic disc photographs in addition to visual fields and global summary measures of computerized imaging. It is also possible to use multiple modalities as inputs, which more closely reflects clinical glaucoma care.

What Limitations Have We Identified So Far?

As with prior work, we are finding it “easy” to differentiate normal subjects from those with clear glaucoma. Given that this is not a huge diagnostic dilemma in most cases, the ultimate applicability of AI to address this particular scenario is unclear. It remains to be seen whether AI can help with more subtle distinctions or identify glaucoma earlier in its course so that treatment might be initiated sooner.

In addition to diagnostic issues, we are also quickly learning that when AI is trained on existing biased data, it will recapitulate those same biases. In other areas, issues of racial and gender bias have been found. It will be an important ethical imperative to avoid this systematized inequity.

Part of the issue with AI bias is the relatively small data sets used to train the latest, most complicated networks. While some data sets seem large to us, they are vanishingly small compared to the Internet-size data sets used to train AI in nonmedical domains. A key challenge to overcoming this barrier is the need to share data between organizations. Because fundus images (photos, OCT) constitute personally identifiable information, we will need new frameworks in place to share them with greater ease.

An exciting area of AI development has been generative AI, which allows users to generate images and large bodies of text using relatively simple prompts. As we have started using these tools, we have discovered they are also subject to the biases incorporated into their training data. There are also common examples now of the text-based generative systems “hallucinating” and making up facts and sources to support false statements.

What Should We Do From Here?

- Work on more challenging clinical problems:
  - Less defined diagnoses
  - Multimodal data
- Develop explainable (justifiable?) systems.
- Be aware of coverage bias.
- Don’t “teach” systems health disparities.
- Develop ethical and legal frameworks.
OCT angiography (OCT-A) is a noninvasive imaging technology that uses the blood flow as an intrinsic contrast agent to generate high-resolution images of the ophthalmic vasculature. Furthermore, OCT-A is coupled with structural OCT, so that the microvasculature of specific retinal layers can be imaged and quantified. Although OCT-A has not been commonly used in the clinical care of glaucoma patients, much research has been done with OCT-A and glaucoma to show its clinical utility and diagnostic capability. The 3 main regions of interest for glaucoma are the superficial peripapillary microvasculature, the superficial macular microvasculature, and the choroidal microvasculature surrounding the optic nerve.

In primary open-angle glaucoma, vessel density of the superficial peripapillary and macular regions has been shown to have diagnostic capability similar to that of structural OCT of the retinal nerve fiber layer (RNFL) to differentiate patients with mild to moderate glaucoma from healthy subjects. Additionally, peripapillary and macular vessel density both decrease as the glaucoma progresses from mild to moderate to severe stages of glaucoma. OCT-A measurements have also been correlated moderately or strongly with visual field (VF) mean deviation and RNFL measurements.

Glaucoma progression is associated with lower baseline macular and peripapillary vessel densities in patients with mild to moderate glaucoma. Similarly, microvascular dropout in the peripapillary choroid is frequently found in locations of prior disc hemorrhages and is associated with higher likelihood of glaucoma progression.

OCT-A may be particularly useful in glaucoma subtypes that are likely to be of vascular etiology. For example, peripapillary vessel density is decreased in normal-tension glaucoma compared to high-tension glaucoma. Similarly, peripapillary vessel density is lower in eyes with paracentral VF loss compared to eyes with peripheral VF loss. Furthermore, choroidal microvascular dropout has been identified in eyes with paracentral VF loss.

In advanced glaucoma, where VF mean deviation is worse than −12 dB, floor effect is less a concern for OCT-A, in contrast to RNFL. Furthermore, in moderate-to-advanced stage glaucoma, the relationship between peripapillary vessel density and VF mean deviation is much stronger than in early glaucoma. Hence, OCT-A may be useful to monitor progression in patients with advanced disease.

OCT-A measurements are available on several commercial devices: Cirrus AngioPlex, AngioVue RTVue, DRI Triton Topcon, PLEX Elite swept source OCT, and Spectralis OCT. Quantification and customization vary on each device, and normative database is lacking. Artifacts are common in OCT-A, further limiting its clinical utility.

In conclusion, OCT-A can play a complementary role to structural OCT imaging and VF testing in the diagnosis and monitoring of glaucoma. It has the potential to differentiate subtypes of glaucoma based on disease etiology. Current clinical use is limited by artifacts, insufficient longitudinal data, and lack of normative database. Active research efforts are under way to improve the clinical utility of OCT-A for glaucoma patients and to enhance our understanding of the vascular etiology of glaucoma.

References

Pearls and Pitfalls of OCT Testing

Richard K Lee MD
Glaucoma and Driving Safety

Felipe A Medeiros MD
Pathophysiology of Aqueous Outflow Regulation: Evolving Concepts

Murray A Johnstone MD

I. What Do We Know From Direct Observation of the Aqueous Veins?
A. Aqueous flows. Aqueous outflow is pulsatile!
B. Pulsatile aqueous outflow stops in glaucoma.
C. Pilocarpine restores pulsatile aqueous outflow.

II. What Do We Know From Cardiovascular Physiology?
A. Like veins and lymphatics, the aqueous outflow system returns fluid to the heart.
B. Venous blood and lymph flow by pulsatile mechanisms.
C. Veins and lymphatics use displacement pumps to move fluid.
D. Displacement pumps have unique requirements:
   1. A chamber, inlet valves, and outlet valves
   2. Segments between the vein and lymphatic valves act as miniventricles.
   3. Walls of the miniventricle compartments must move to propel fluid forward.
   4. A driving force is present, resulting from the cyclic cardiac pulse and transient tissue motion.
   5. The outflow system has Prox 1, a marker for lymphatic valves.
   6. Defective Prox 1, necessary for lymphatic valve development, causes glaucoma.

III. What Have We Known Before Recent OCT Imaging Advances?
A. The trabecular meshwork (TM) is the wall of a vessel called the Schlemm canal (SC).
B. The SC is a chamber, and SC chamber volume changes with IOP.
C. Aqueous-containing endothelial-lined conduits arise from the SC inner wall.
D. The conduits attain a tube-like shape, cross the SC, and attach to the external wall.
E. The conduits act as SC inlet valves (SIV). They allow aqueous flow and prevent blood reflux.
   1. Light, scanning, and transmission electron microscopy document SIV structure.
   2. Microsphere and red blood cell tracer studies document SIV function as conduits.

III. What Do We Know From Direct Observation of the Aqueous Veins?
A. Aqueous flows. Aqueous outflow is pulsatile!
B. Pulsatile aqueous outflow stops in glaucoma.
C. Pilocarpine restores pulsatile aqueous outflow.

IV. What Insights Does Ex Vivo High-Resolution OCT Provide?
A. Collector channel (CC) valves
   1. CC entrances have collagen flaps attached only at 1 end.
   2. The CC-hinged flaps undergo pressure-dependent changes in position.
   3. Position changes allow the hinged flaps to open and close the CC.
   4. Position changes enable the flaps to act as SC outlet valves (SOV).
   5. SIV provides connections between the TM and the SOV hinged flaps.
   6. SIVs elongate as SC pressure increases, placing tension on the hinged flaps.
B. Circumferentially oriented deep scleral plexus (CDSP) channels are adjacent to the SC.
   1. Thin septa separate the CDSP from SC and move by pressure-dependent mechanisms.
   2. CDSPs open and close like a second pressure-dependent chamber.
C. Evidence from real-time imaging of tissue motion
   1. The TM beams, SIV, SOV, and CDSP all undergo rapid cyclic pulsatile movement.
   2. The amplitude and motion velocity can account for all of the aqueous outflow.
   3. The motion of TM, SIV, and SOV and the SC and CDSP volume changes are all synchronous.
   4. Cellular attachments between the structures can explain the synchrony of motion.

V. What Insights Does Human In Vivo Phase OCT (PhS-OCT) Provide?
A. OCT in human subjects is challenging because of motion and light scattering.
B. Commercial spectral domain OCT systems using an 810-nm wavelength have limited sensitivity.
C. A purpose-built 1310-nm PhS-OCT system resolves motion of ~20 nm.
D. The PhS-OCT system quantitates TM velocity and displacement.
E. Recent reports find significant motion differences between normal and glaucoma eyes.

VI. What Pump Function Behavior May Explain Selective Laser Trabeculoplasty (SLT) Effects?
A. Pilocarpine temporarily restores pulsatile flow in glaucoma by increasing ciliary muscle tension.
B. Transscleral micropulse laser (TML) simulates pilocarpine’s effect on the outflow system.
C. TML heat tightens the ciliary muscle and opens proximal and distal outflow pathways.
D. SLT and argon laser trabeculoplasty (ALT) both use heat to cause changes in outflow pathways.
E. Heat anywhere along the TM beam/ciliary muscle system will cause collagen shrinkage.
F. Collagen shrinkage persists, in contrast to the short-term effects of cytokine release.
G. Both SLT and ALT may act like pilocarpine, increasing TM tensions to restore pulsatile outflow.

VII. What Behavior May Explain Minimally Invasive Glaucoma Surgery (MIGS) Effects? Priming the Pump?
A. Real-time OCT imaging of motion following insertion of a MIGS-like cannula into the SC.
B. Pressure introduced into SC simulates pressures and transients experienced after MIGS.
1. A pressure increase in SC causes TM movement and SC dilation ≥5 mm beyond the device.
2. SC pressure changes cause CCs to open and close distal to the insertion area.
3. SC and CC dilation is sufficiently rapid to permit pulsatile aqueous flow.
4. CDSPs in the deep scleral plexus open and close with SC pulsatile pressure.

VIII. Can Pump Function Improvement Explain IOP Reduction After Cataract Extraction?
A. Pilocarpine briefly restores pump function by improving scleral spur (SS) traction.
B. High-resolution MRI demonstrates improved spur vectors and traction after cataract surgery.
C. Reports propose that cataract surgery improves pump function by improving SS traction.

Selected Readings
Emerging Glaucoma Meds

Arthur J Sit MD

Introduction
Reduction of IOP is currently the only known effective treatment for glaucoma. Topical medications are the first-line therapies for most glaucoma patients. Although there are numerous available treatments, and increased utilization of selective laser trabecuoplasty (SLT) and minimally invasive glaucoma surgeries (MIGS) have reduced medication burden in many patients, eyedrops remain a critical option for IOP reduction. There continues to be a need for the development of new medical therapies due to variable response, intolerable side-effect profiles in some patients, and elevated IOP refractory to other treatments.

Background
IOP is determined by the rate of aqueous humor outflow (Q), outflow facility (c), the rate of aqueous humor outflow through the uveoscleral pathway (U), and episcleral venous pressure (EVP), as described by the modified Goldmann equation:

\[ IOP = \left( \frac{Q - U}{c} \right) + EVP. \]

The conventional outflow pathway, which accounts for the majority of aqueous humor outflow, involves passage from the anterior chamber, across the trabecular meshwork (TM) into the Schlemm canal, and drainage into the collector channels and episcleral veins. Episcleral venous pressure is the back pressure against which aqueous humor must drain. The uveoscleral pathway involves aqueous humor outflow through interstitial spaces in the ciliary muscle and suprachoroidal space. All glaucoma medications target one or more of these parameters to reduce IOP. However, while glaucoma medications have historically often been discovered serendipitously, novel compounds in development are more targeted in their mechanism of action and have an increased focus on minimizing adverse side-effects.

Emerging Medications
Numerous ocular hypertensive medications with novel therapeutic targets are currently in or have recently completed Phase 2 and 3 clinical trials or were recently approved.

Selective, nonprostaglandin, prostanoid EP2 receptor agonists are a new class of medications that have effects on both IOP and episcleral venous pressure (EVP). In particular, there appears to be a marked reduction of prostaglandin-associated periorbitopathy, which manifests as a deepening of the upper eyelid sulcus secondary to orbital fat atrophy. OMDI was FDA approved on September 22, 2022.

NCX 470 is a bimatoprost-containing compound with nitrous oxide (NO)-donating group developed by Nicox Ophthalmics (Durham, NC). NCX 470 is dosed daily and is expected to have a mechanism of action similar to latanoprost (trade name Vyzulta), which is a NO-donating derivative of latanoprost, and was FDA approved in November 2017. Bimatoprost is a prostaglandin analog that increases uveoscleral outflow, while NO reduces IOP through activation of soluble guanylyl cyclase, leading to increased cyclic guanosine monophosphate (cGMP) formation in ocular tissues. This alters aqueous humor formation at the ciliary epithelium and improves conventional outflow facility through relaxation of the TM and Schlemm canal, which leads to IOP reduction. In a Phase 2 clinical trial, NCX 470 0.065% demonstrated superior IOP reduction compared to latanoprost at all time points (8 am, 10 am, and 4 pm) at 28 days, and a mean diurnal decrease of 8.7 mmHg. Phase 3 clinical trials are ongoing.

AKB-9778 or razuprotafib is a tyrosine kinase receptor 2 activator, developed by Aerpio Pharmaceuticals, that reduces IOP by improving aqueous humor outflow through the conventional pathway. It inhibits vascular endothelial protein tyrosine phosphatase (VE-PTP) which leads to activation of tyrosine kinase with immunoglobulin-like and epidermal growth factor-like 2 domain (Tie2). Tie2 activation in the Schlemm canal inner wall endothelium decreases outflow resistance and leads to IOP reduction. Clinical trial data for razuprotafib have focused on its use as an adjuvant therapy. A Phase 2 clinical trial found that in patients with primary open-angle glaucoma or ocular hypertension, razuprotafib given twice per day in addition to latanoprost results in a modest but statistically significant reduction in IOP when compared to latanoprost alone (7.95 ± 0.26 mmHg vs. 7.04 ± 0.26 mmHg). The efficacy of razuprotafib as monotherapy has not been reported.

SYL (bamosiran) is a double-stranded siRNA being developed by Sylentis (Madrid, Spain). Dosed once daily, it silences β-2 adrenergic receptors (ADRB2). Reduction of ADRB2 expression results in a decrease of aqueous humor production. Unlike traditional aqueous humor suppressants, the effects of bamosiran remain until ADBR2 receptor synthesis increases after cessation of treatment. Also, bamosiran appears to have limited systemic side effects since naked siRNA molecules are degraded by the kidneys shortly after reaching the blood stream. Syltag, a multicentered, double masked, randomized Phase 2b study for patients with primary open-angle glaucoma and ocular hypertension, found statistically significant IOP reduction for each concentration of the compound at 28 days compared with vehicle. However, it did not demonstrate noninferiority to timolol, except in patients with IOP greater than 25 mmHg.
of cells in the iris, ciliary, body, TM, and sclera. However, the primary site of action for IOP reduction is in the conventional outflow system distal to the Schlemm canal, which appears to reduce EVP. Preclinical studies of CKLP1 have reported IOP reduction in both wild type and ocular hypertensive mouse models, with greater than 20% decrease in IOP and a reduction in EVP by at least 29%. CLKP1 also lowered IOP in dog and nonhuman primate models by 18.9% ± 1.1% and 16.7% ± 6.7%, respectively.21 Consistent with a primary effect on EVP, there were no histological changes to TM or Schlemm canal cells in human, Dutch-belted pigmented rabbit, or mouse models, although there was a reduction in extracellular matrix accumulation in the TM of a steroid-induced ocular hypertension mouse model.18,22 QLS-101 appears to be the first medication approved have demonstrated significant and clinically relevant reduction of IOP. Differentiating factors compared with existing medications include improved side-effect profile, novel mechanisms of action, and superior reduction of IOP. Even if there are clinical advantages, however, the adoption of new medications can be uncertain due to the widespread availability of generic prostaglandin analogs. This may limit acceptance of these novel compounds as first-line agents, except for certain subgroups of glaucoma patients. Use as adjuvant or second-line therapy appears more likely for the majority of glaucoma patients.

Summary and Conclusions

Numerous compounds currently in development or recently approved have demonstrated significant and clinically relevant reduction of IOP. Differentiating factors compared with existing medications include improved side-effect profile, novel mechanisms of action, and superior reduction of IOP. Even if there are clinical advantages, however, the adoption of new medications can be uncertain due to the widespread availability of generic prostaglandin analogs. This may limit acceptance of these novel compounds as first-line agents, except for certain subgroups of glaucoma patients. Use as adjuvant or second-line therapy appears more likely for the majority of glaucoma patients.

References


12. Walters TR, Kothe AC, Boyer JL, et al. A randomized, controlled comparison of NCX 470 (0.021%, 0.042%, and 0.065%) and latanoprost 0.005% in patients with open-angle glaucoma or ocular hypertension: the Dolomites Study. J Glaucoma. 2022; 31(6):382-391.


Medication Adherence

Paula Anne Newman-Casey MD MS

I. Glaucoma Medication Adherence—Why Is It Important?
   A. Association with glaucoma severity
   B. Impact on field loss over time

II. Glaucoma Medication Adherence—Is It Really a Problem?
   A. Persistence with different classes of glaucoma medications
   B. Who persists with glaucoma medications over time?

III. Glaucoma Medication Adherence—Why Is It So Hard?

IV. Glaucoma Medication Adherence—How Can We Improve?
   A. Expanding the glaucoma care team
      1. MAGIC Trial
      2. SEE Trial
      3. Lumata Health
   B. Helping patients feel heard
      1. Asking open-ended questions
      2. Reflecting
      3. Asking permission to provide advice
   C. How can physicians help?
      1. Assessing adherence on all patients
      2. Addressing cost/side effects/complex drop regimens
      3. Assessing ability to instill drops
      4. Organized ophthalmology can support expansion of the glaucoma care team.

Selected Readings


Selective Laser Trabeculoplasty Outcomes as Primary Glaucoma Treatment

*Sylvia L Groth MD*
Cold Feet? Why Selective Laser Trabeculoplasty Should Be a First-Line Treatment

Anthony D Realini MD

I. The current glaucoma treatment paradigm starts with topical medications.
   A. This made sense in the era of argon laser trabeculoplasty and trabeculectomy.
   B. Does it still make sense in the era of selective laser trabeculoplasty (SLT) and minimally invasive glaucoma surgery?

II. The meds-first approach has many important limitations.
   A. Poor adherence
   B. Side effects
   C. Cost
   D. Difficult to administer

III. An SLT-first approach makes more sense.
   A. Overcomes nonadherence
   B. Safer
   C. Better outcomes (Laser in Glaucoma and Ocular Hypertension Trial)
   D. Cost-effective

IV. Talking to Patients About SLT
   A. Describing it as an alternative to medications suggests deviating from the standard of care. Patients don’t care how we used to treat glaucoma; they care about what’s best now.
   B. If you (the doctor) would opt for primary SLT if you were diagnosed with glaucoma, then clearly you believe it is the best treatment—and how can your patients make an informed decision without that information?
How Selective Laser Trabeculoplasty and Meds Reduce the Diurnal Fluctuations in IOP of Glaucoma Patients

Leonard K Seibold MD

I. The Importance of IOP
   A. Only modifiable risk factor
   B. All treatments focused on this single metric

II. IOP is dynamic.
   A. Single office IOP readings are limited.
   B. Every patient has a maximum, minimum, and overall range of IOP.
   C. Fluctuations of IOP occur from many factors:
      1. Short term: blinking, heart rate
      2. Intermediate term: medication effect, activity, positional changes
      3. Long term: disease progression, treatment changes

III. Diurnal Rhythm of IOP
   A. Present in normal and glaucoma patients
   B. Typically highest in early morning; declines to nadir in afternoon/evening
   C. Some patients peak in afternoon.
   D. Sustained overnight increase
   E. Relates to catecholamines, aqueous humor production, positional changes

IV. What happens at night?
   A. Nadir of aqueous flow (production)
   B. Decrease in outflow facility
   C. Increase in episcleral venous pressure due to supine positioning
   D. Overall increase in IOP in habitual position

V. Why Variability Matters
   Studies have shown that progression is associated with greater IOP fluctuation and range.
   A. Advanced Glaucoma Intervention Study
   B. Collaborative Initial Glaucoma Treatment Study

VI. How can we measure IOP fluctuations?
   A. Diurnal curve: Serial IOP measures in clinic every 2 hours during office hours
   B. Home tonometry
      1. iCare HOME
   C. 24-hour sleep lab: Admit to overnight sleep lab with IOP measurement every 2 hours
   D. Temporary continuous IOP monitors
      1. Contact lens sensors (Triggerfish)
      2. More complete assessment of IOP fluctuation over 24-hour period
      3. Does not measure true IOP
   E. Permanent continuous IOP monitors
      1. Several in development, none FDA approved
      2. Eyemate from Implantdata, CE marked

VII. Medication Effect on Diurnal IOP Fluctuations
   A. Prostaglandin analogues (PGA)
      1. Reduction in overall IOP fluctuation
      2. IOP lowering at night
      3. Sustained effect even with missed doses
   B. Beta blockers
      1. Reduction in daytime IOP fluctuation
      2. No overnight effect on IOP
      3. Sustained reduction in heart rate
   C. Carbonic anhydrase inhibitors: Reduction in daytime IOP and overnight when used as adjunct to PGA
   D. Alpha agonists
      1. Daytime IOP reduction, but negligible overnight effect
      2. Reduction in blood pressure
   E. Miotic agents
      1. Daytime IOP reduction but short-term effect
      2. Overnight IOP reduction when used as adjunct to PGA
   F. Combination agents
      1. Timolol/dorzolamide, timolol/brimonidine, brinzolamide/brimonidine
      2. All can reduce IOP during day and night.
   G. Rho-kinase inhibitors: Reduction in IOP during day and night
VIII. Selective Laser Trabeculoplasty (SLT) Effect on Diurnal Fluctuations

A. Lowers mean IOP during daytime
B. Additional effect in reduction of IOP peak, minimum, and overall fluctuation
C. Overnight reduction in IOP
D. Laser in Glaucoma and Ocular Hypertension Trial: Less disease progression compared to medications despite similar IOP
E. Reduced fluctuations compared to drops due to noncompliance, variable timing of drops

Selected Readings


Glaucoma Medical Management Before and After Surgery

Management of Hypertensive Phase

Rachel Han Lee MD MPH

I. What is the hypertensive phase?
   Pattern of IOPs after insertion of (valved) glaucoma drainage device
   A. Hypotensive phase: Lasts 1+ weeks
   B. Hypertensive phase: Occurs 1-6 weeks after tube patent, lasts up to 6 months

II. Importance of Managing Hypertensive Phase
   A. May exceed preoperative levels
   B. May be predictive of surgical failure
   C. May not always resolve

III. Causes of Hypertensive Phase
   A. Likely due to bleb encapsulation
      1. Early mechanical force
      2. Early exposure to cytokines
   B. Early use of IOP-lowering medications should address both.

IV. Does early use of IOP-lowering medications work?
   A. Randomized clinical trial of patients receiving:
      1. Dorzolamide-timolol if IOP >10 mmHg
      2. “Conventional therapy” when IOP > target pressures
   B. Early pressure lowering resulted in lower pressures up to 54 weeks following surgery without significantly different medication usage.
   C. Early pressure lowering resulted in lower rates of hypertensive phase, without an increase in rate of choroidal effusions.

V. What target pressures should we target?
   A. Randomized clinical trial of patients receiving:
      1. IOP-lowering medications when IOP >10 mmHg
      2. IOP-lowering medications when IOP >17 mmHg
   B. IOPs and medication usage were similar between the 2 groups 6-24 months after surgery.
   C. Overall, ~40% of eyes underwent hypertensive phase, at approximately 1 month post-surgery, lasting 15 days with IOP peaks of 30 mmHg, on average.
      1. There were no significant differences in rates, onset, duration, or peaks of hypertensive phase between the 2 groups.
      2. Eyes with hypertensive phase were more likely to have higher IOPs and require more glaucoma surgery than eyes without hypertensive phase.
United for Sight: A Vision for Effective Advocacy

Glaucoma Subspecialty Day 2023

Sarah Wellik MD

Action Requested: Donate to strengthen ophthalmology’s legislative voice and protect patients and your profession

Please respond to your Academy colleagues and join the community that advocating for ophthalmology: OPHTHPAC, the Surgical Scope Fund, and your State Eye PAC. Ensure you and your patients are heard by our nation’s lawmakers by giving to each of these funds.

Where and How to Contribute

During AAO 2023 in San Francisco, please contribute to OPHTHPAC® and Surgical Scope Fund at one of our two convention center booths or online. You may also donate via phone to both funds by sending two texts:

- Text MDEYE to 41444 for OPHTHPAC
- Text GIVESSF to same number (41444) for the Surgical Scope Fund

We also encourage you to support our congressional champions by making a personal investment via OPHTHPAC Direct, a unique and award-winning program that lets you decide who receives your political support.

Surgical Scope Fund contributions are completely confidential and may be made with corporate checks or credit cards. PAC contributions may be subject to reporting requirements.

Why Should You Contribute?

Member support of the Academy’s advocacy funds—OPHTHPAC and the Surgical Scope Fund—powers our advocacy efforts at the federal and state levels. When you give to OPHTHPAC, you give ophthalmology a voice on Capitol Hill on critical issues like Medicare payment, optometry’s scope expansion efforts in the VA, and prior authorization and step therapy burdens. When you give to the Surgical Scope Fund, you’re funding our efforts to fight dangerous optometric surgery initiatives at the state level, whenever and wherever they arise. And finally, when you give to your state Eye PAC, you help elect officials in your state who will support the interests of you and your patients. Giving to each of these three funds is essential to helping protect sight and empower lives.

Protecting quality patient eye care and high surgical standards is a “must” for everybody. Our mission of “protecting sight and empowering lives” requires robust funding of both OPHTHPAC and the Surgical Scope Fund. Each of us has a responsibility to ensure that these funds are strong so that ophthalmology continues to thrive and patients receive optimal care.

OPHTHPAC for Federal Advocacy

OPHTHPAC is the Academy’s award-winning, non-partisan political action committee representing ophthalmology on Capitol Hill. OPHTHPAC works to build invaluable relationships with our federal lawmakers to garner their support on issues such as:

- Improving the Medicare payment system, so ophthalmologists are fairly compensated for their services, and working to prevent impending payment cuts of 3.36% scheduled to take effect in 2024
- Securing payment equity for postoperative visits, which will increase global surgical payments
- Stopping optometry from obtaining surgical laser privileges in the Veterans’ Health-care System
- Increasing patient access to treatment and care by reducing prior authorization and step therapy burdens

Academy member support of OPHTHPAC makes all this possible. Your support provides OPHTHPAC with the resources needed to engage and educate Congress on our issues, helping advance ophthalmology’s federal priorities. Your support also ensures that we have a voice in helping shape the policies and regulations governing the care we provide. Academy member support of OPHTHPAC is the driving factor behind our advocacy push, and we ask that you get engaged to help strengthen our efforts and make sure that the ophthalmology specialty has a seat at the table for the critical decisions being made that affect our ability to care for our patients.

At the Academy’s annual Mid-Year Forum, the Academy and the American Glaucoma Society (AGS) ensure a strong presence of glaucoma specialists to support ophthalmology’s priorities. As part of this year’s meeting, the AGS supported participation of fellowship trainees via the Academy’s Advocacy Ambassador Program. During Congressional Advocacy Day, they visited Members of Congress and their key health care staff to discuss glaucoma priorities. The AGS remains a crucial partner with the Academy in its ongoing federal and state advocacy initiatives.

Surgical Scope Fund (SSF) for State Advocacy

The Surgical Scope Fund works in partnership with state ophthalmic societies to protect patient safety from dangerous optometric surgery proposals through advocacy. The Fund’s mission is to ensure surgery by surgeons, and since its inception, it has helped 43 state/territorial ophthalmology societies reject optometric scope-of-practice expansions into surgery.

Support for the Surgical Scope Fund from ophthalmic interest societies like the American Glaucoma Society (AGS) makes our advocacy efforts possible. These efforts include research, lobbyists, political organization, polling, advertising, social media, digital communications, and grassroots mobilization.
However, the number of states facing aggressive optometric surgery legislation each year has grown exponentially. And with organized optometry’s vast wealth of resources, these advocacy initiatives are becoming more intense—and more expensive. That’s why ophthalmologists must join together and donate to the Surgical Scope Fund to fight for patient safety.

The Academy’s Secretariat for State Affairs thanks the AGS for its past support of the Surgical Scope Fund and looks forward to its 2023 contribution. The AGS’ support for the Surgical Scope Fund is essential to fighting for patient safety and quality eye care!

**State Eye PAC**

The presence of a strong state Eye PAC providing financial support for campaign contributions and legislative education to elect ophthalmology-friendly candidates to the state legislature is critical as scope of practice battles and many regulatory issues are all fought on the state level.

**Support Your Colleagues Who Are Working on Your Behalf**

Two Academy committees made up of your ophthalmology colleagues are working hard on your behalf. The OPHTHPAC Committee continues to identify Congressional Advocates in each state to maintain close relationships with federal legislators to advance ophthalmology and patient causes. The Surgical Scope Fund Committee is raising funds used to protect Surgery by Surgeons during scope battles at the state level.

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Why We Should Participate in Glaucoma Registries

Sally L Baxter MD

I. Introduction

In this talk, I will provide a brief overview of databases and registries that have been used to advance glaucoma knowledge. I will provide examples of studies that have utilized these data sources to advance glaucoma knowledge with regard to pathophysiology, treatment outcomes, and health disparities. This information will hopefully help motivate increased participation in registries among attendees, by contributing data and/or by analyzing data.

II. Types of Data Sources

A. Secondary use of data from routine clinical care
   (This will be the focus of subsequent components of the talk.)
   1. Claims data
   2. Electronic health record (EHR) data
   3. Clinical registries

B. Data generated from research studies
   1. Clinical trials
   2. Observational studies

III. Benefits of Databases/Registries

A. Understanding real-world practice patterns and outcomes
B. Increasing sample sizes/power (vs. siloed datasets): enhances rigor, enables study of rare diseases
C. Enhancing diverse representation, for patients and researchers

IV. Local/Institutional EHR Data Warehouses and EHR-Based Registries

A. Overview
   1. Typically EHR data maintained by individual institution
   2. Includes systemic data as well (in addition to ophthalmic data)
   3. May have limited generalizability
   4. May also have limited structured data, depending on how the ophthalmic data are formatted and how they are entered by the clinicians at that institution
   5. But can inform local interventions and quality improvement activities
      a. EHR-based registries can enable bulk orders, bulk messaging.

B. Examples of research studies
   1. Impact of COVID-19 on missed ophthalmology visits
   2. Claims/EHR registry from a single practice

C. How to contribute data
   1. Typically no additional effort needed from clinicians to contribute to EHR data warehouse
   2. Construction of EHR-based registries requires substantial effort and investment
      a. Clear inclusion/exclusion criteria need to be established.
      b. Close collaboration with IT teams to build the registries and integrate with EHR systems
      c. Can be difficult to find resources in the absence of quality metrics/incentive payments

D. How to access data
   1. Access to data governed by institutional policies
   2. May have user-facing tools for quick queries and reports
   3. Larger-scale data extraction may require going through centralized services.

V. American Academy of Ophthalmology Intelligent Research in Sight (IRIS®) Registry

A. Overview
   1. Largest medical specialty database in the world
   2. Wide range of sites from across the United States
   3. Does not include substantial systemic/nonophthalmic data
   4. Image integration in progress

B. Examples of research studies
   1. Demographic and clinical characteristics associated with minimally invasive glaucoma surgery
   2. Risk factors for glaucoma drainage device revision or removal
   3. Primary open-angle glaucoma practice patterns in academic vs. nonacademic settings
C. How to contribute data
The Academy is partnering with Verana Health, which manages EHR data ingestion, harmonization, and deidentification.

D. How to access data
1. Academic Consortium partners
2. Research awards (eg, American Glaucoma Society)

VI. National Institutes of Health (NIH) All of Us Research Program
A. Overview
1. Nationwide prospective cohort study with an emphasis on diverse enrollment
2. Wide range of data types: EHR data, surveys, physical measurements, wearables, biospecimens, genomic data
3. Includes substantial systemic data
4. No imaging/testing data at this time, although efforts to incorporate ophthalmic imaging are under way

B. Examples of research studies
1. Predictive analytics/machine learning models of glaucoma
2. Association between blood pressure, BP meds, and glaucoma

C. How to contribute data: Encourage patients to enroll at an All of Us enrollment site

D. How to access data
1. “Data Passport Model” to democratize access
2. Publicly available data browser (for aggregate counts)
3. Minimal barriers to accessing data and analytic tools (need ID verification, data use agreement, basic responsible conduct of research training, and data use attestation)

VII. Sight Outcomes Research Collaborative (SOURCE)
A. Overview
1. Consortium of academic ophthalmology departments using the Epic EHR
2. Wide range of data types available, including both ophthalmic data and systemic data (including some free-text notes)
3. Some sites contributing imaging/testing data

B. Example research study: Phenotyping of exfoliation syndrome using both structured and unstructured data

C. How to contribute data
1. Need clinical champion/site lead, need to be using Epic

D. How to access data
1. Coordinate with Josh Stein MD (PI of SOURCE)

VIII. Conclusions
A. Increasing opportunities to study glaucoma using registries/databases, including several of nationwide scope
B. Multiple benefits of registry participation, including rigor/reproducibility and enhancing diverse representation
C. Many challenges remain, including data standardization, ensuring data quality, etc.
D. The more contributions there are from glaucoma specialists, the more useful these data sources will be to the glaucoma community.

References
New Frontiers in Neovascular Glaucoma

Catherine Sun MD

I. Common Causes of Neovascular Glaucoma (NVG)
   A. Proliferative diabetic retinopathy
   B. Central retinal vein occlusion
   C. Ocular ischemic syndrome

II. Treatment Goals for NVG
   A. Lower the IOP
      1. Medications: topical and/or oral
      2. Surgery
         a. Trabeculectomy or Ex-Press shunt
         b. Glaucoma drainage devices
      3. Laser: Cyclophotocoagulation
   B. Target VEGF production
      1. Panretinal photocoagulation (PRP)
      2. Intravitreal anti-VEGF injections
   C. Treat the underlying disease etiology

III. Evidence From Clinical Studies
   A. Role of anti-VEGF vs. PRP before NVG has developed
   B. Role of anti-VEGF vs. PRP after NVG has developed
   C. Optimal IOP-lowering procedure for NVG

IV. Future Goals to Improve NVG Management
   A. Need a multidisciplinary treatment approach
   B. Earlier detection of anterior segment neovascularization
   C. Improved patient engagement and education about NVG
   D. Standardize the definition and staging of NVG
   E. Need more high-quality evidence-based research in NVG

Selected Readings
Emerging Functional Outcomes With Minimally Invasive Glaucoma Surgery Studies

Iqbal K Ahmed MD
Can OCT Predict Field Loss? When Should We Test the Central Field?

Donald C Hood PhD

I. Introduction
I have been asked to address the two questions:
Q1 When should we test the central field?
Q2 Can OCT predict field loss?

II. Q1: When should we test the central field?
I will treat this as two separate questions.
A. Q1a: When do you perform a 10-2 visual field test?
   1. Answer: All glaucoma patients or suspects, or at least anyone for whom you would do, or have done, a 24-2/30-2 visual field.1
   2. Why3,6
      a. The 24-2 or 30-2 test pattern can miss and/or underestimate central (macular) damage.
      b. Central/macular damage is very common even in the earliest stages of glaucoma.
   B. Q1b: When do you perform an OCT scan of the macula?
   1. Answer: All glaucoma patients or suspects should have an OCT scan that includes the macula.1
   2. Why?2,3,6
      a. Central/macular damage is very common.
      b. OCT scans of the disc can miss and/or underestimate damage seen on a cube scan of the macula and ganglion cell thickness.

III. Q2: Can OCT predict field loss?
A. A simple answer is Yes! In fact, not only can OCT predict field loss, but loss on 24-2 and 10-2 fields can predict OCT.
B. Why?7-10
   1. There is a common misconception that structure (eg, OCT) and function (visual field) do not agree. This is not true!8
   2. The OCT is typically a good predictor of damage on the visual field.
   3. You will get good abnormal structure (OCT) and abnormal function (visual field) agreement if you:
      a. Perform both 10-2 and 24-2 visual fields
      b. Use both retinal nerve fiber layer (RNFL) and ganglion cell deviation and thickness maps
      c. Compare local regions of visual field and OCT deviation maps.

IV. Notes
a. If you compare 24-2 metrics (mean deviation, pattern standard deviation, glaucoma hemifield test) and OCT disc scan metrics (global RNFL thickness) you will not get best agreement.
   b. Agreement between visual field and OCT can be objectively determined.10

References
Fine Tuning Ciliary Body Ablation and Other Outflow: Cyclophotocoagulation (CPC), Endoscopic CPC vs. MicroPulse P3 CPC

Osamah Saeedi MD

I. Case Presentation
   CPC vs. endoscopic CPC (ECP) vs. MicroPulse

II. Pros and Cons of ECP
   A. Technique
      1. Anterior ECP
      2. Posterior ECP (ECP Plus)
   B. Complications
   C. Efficacy

III. Pros and Cons of Conventional CPC
   A. Technique
   B. Complications
   C. Efficacy

IV. Pros and Cons of MicroPulse P3
   A. Technique
   B. Complications
   C. Efficacy

V. Comparative Studies

Selected Readings


Helping Glaucoma Patients at Home With Tools

Yvonne Ou MD

I. Motivation for Helping Glaucoma Patients at Home With Tools
   A. Patient autonomy and engagement
   B. Remote areas/access to care
   C. A new form of teleglaucoma
   D. Future pandemics

II. Home Visual Acuity Measurements

III. Nanodropper
   A. Efficacy
   B. Potential benefit

IV. Home Tonometry
   A. Use cases for devices such as iCare HOME2
      1. Decision-making regarding advancement of treatment
      2. Checking IOP after an intervention (eg, response to treatment)
      3. Diurnal IOP measurements
   B. Comparison of home tonometers with in-clinic measurements

V. Home Visual Field
   A. Tablet perimeters
   B. Virtual reality visual field headsets or goggles
   C. Feasibility and adherence to home visual field testing
   D. Correlation with standard automated perimetry

VI. Beware the Digital Divide

VII. What the Present and Future Hold
   A. Home optic nerve imaging
   B. Wearable devices
   C. Generation of large amounts of data
   D. Home monitoring of stable and low risk patients?
How Direct Selective Laser Trabeculoplasty May Change Glaucoma Care

Thomas W Samuelson MD

Background: Selective Laser Trabeculoplasty (SLT)

While laser trabeculoplasty has been long considered an effective treatment for open-angle glaucoma (OAG) and is offered as initial therapy by many providers, the landmark Laser in Glaucoma and Ocular Hypertension Trial (LiGHT) has significantly changed the initial treatment schema for OAG, elevating the status of SLT for use as first-line therapy. Indeed, the European Glaucoma Society and the HOPE Public Healthcare network in the United Kingdom now advocate for SLT as the recommended initial treatment for OAG, reserving eyedrop therapy for those patients who either decline laser therapy or for whom laser therapy is not suitable. Based on recent data, a very strong case can be made that SLT should be the recommended initial treatment for most patients with OAG rather than passively offered as an equal option to medical therapy.

The standard SLT procedure is performed using a gonioscopy lens, which allows the physician to aim the laser beam directly on the trabecular meshwork (TM). Visualization of the angle with a gonioscopy lens requires sufficient angle width, and the procedure requires expert training and experience. In addition, the gonioscopy lens is in direct contact with the corneal surface, which can lead to patient discomfort and, while uncommon, may pose a risk of infection or mechanical damage of the cornea. These procedural limitations, while relatively minor, likely reduce the adoption of SLT despite ample data that supports its use. This may be especially true in areas underserved by eye care providers.

Background: Direct SLT

Direct SLT (Belkin Vision) is a delivery system in which the laser beam is applied to the TM through the limbus “directly,” thus eliminating the need to use a gonioprism during the procedure. To simplify the procedure for the eye care professional, the Eagle DSLT system automatically locates the limbal region of the eye to be treated. After the treatment location has been optionally adjusted and confirmed by the laser surgeon, the system then automatically applies the laser pulse treatment sequence to the periligmal region, usually the entire circumference (360 degrees) or any portion thereof. This procedure is referred to as direct SLT (DSLT).

Other than the way in which the laser beam is delivered to the target tissue, the DSLT shares many of the same design specifications as a traditional SLT laser. Its therapeutic laser engine employs a Q-switched, frequency-doubled Nd:YAG laser that generates pulses of 532-nm wavelength and 3-nanosecond duration, the same as most commercially available SLT systems. Furthermore, the system is designed to deliver a similar amount of total energy to the target TM tissue as is commonly used in traditional SLT procedures.

Procedural Description

Video demonstration.

Comparative Data: DSLT vs. SLT

The GLAUrious Trial is a prospective randomized, multicenter, multinational trial comparing DSLT to SLT. The primary study hypothesis was noninferiority of DSLT to SLT at 6 months for unmedicated IOP change from baseline, evaluated on the modified-per-protocol population. At 6 months, the mean reduction in IOP from washout baseline was 5.46 mmHg for DSLT compared to 6.16 mmHg for SLT. There were no meaningful safety issues in either group. Medication reduction and percent of patients remaining medication free at 12 months were similar in each group.

Clinical Implications and Conclusions

DSLT has safety and efficacy similar to SLT and offers several advantages to both patients and physicians that may increase adoption of this important technology in U.S. and global populations, especially in underserved communities lacking specialized glaucoma care. The noncontact and “direct” approach allow for treatment without the need for specialized gonioscopic techniques while offering a more patient-friendly experience and clinical efficiencies for physicians and clinics.

Selected Readings

4. GLAUrious Trial: Data on file with Belkin Vision.
The Association for Research in Vision and Ophthalmology (ARVO) met for the 2023 meeting in New Orleans from Sunday, April 23, to Thursday, April 27. The program was subdivided into poster sessions, symposia, keynote addresses, mini-symposia, special interest sessions, award ceremonies, competitions, and social events. There were approximately 160 post-sessions, 16 symposia and mini-symposia, 139 paper sessions, 7 special interest group sessions, and 2 diversity, equity, inclusion, and accessibility–specific research sessions. This was a large and comprehensive meeting featuring key advancements in glaucoma, such as:

**Sunday April 23, 2023**
- Glaucoma Data Science
- Glaucoma: Molecular, Biochemical, and Biomechanical Mechanisms

**Monday April 24, 2023**
- Aqueous Humor Dynamics
- Neuroregeneration and Neuroprotective Mechanisms
- Glaucoma Structure and Function Relationships

**Tuesday April 25, 2023**
- Neuroprotective Strategies
- Drug Delivery to the Iris and Ciliary Body
- Neuroregeneration

**Wednesday April 26, 2023**
- Glaucoma Surgery and Wound Healing
- Retinal Ganglion Cells and Beyond
- The Role of Immune Cells in Ocular Disease

**Thursday April 27, 2023**
- Glaucoma Lasers
- Visual Fields and Psychophysics
- Glaucoma Surgery and Lasers
- New Machine Learning for Imaging Analysis

This talk will summarize cutting-edge research in both the clinical and basic science space, highlighting the following talks identified by the ARVO Selection Committee as “Hot Topics”:
- Associations between glaucoma prevalence and body mass index, waist circumference, and metabolic syndrome using the NIH “All of Us” database.
  *First Author*: Jennifer E Lee (UCLA David Geffen School of Medicine)
- Development of a microfluidic culture platform to explore RGC compartmentalization and glial orientation in a human pluripotent stem cell model of neurodegeneration and neuroinflammation.
  *First Author*: Jason S Meyer (Indiana University School of Medicine)
It Began in San Francisco—How Treatment of Childhood Glaucoma Continues to Evolve

James D Brandt MD

Congenital glaucoma or hydrophthalmos is perhaps the most hopeless and certainly the most pathetic of ocular conditions requiring surgery. The end result, with or without operation, is frequently blindness, and more often than not, enucleation of one or both eyeballs is required.

J Ringland Anderson MD (London, 1939)

Prior to the mid-20th century, childhood glaucoma was uniformly blinding; it was the leading cause of blindness among children enrolled in schools for the blind. The treatment of primary congenital glaucoma (PCG) was revolutionized midcentury with the introduction of what is collectively called “angle surgery”—first goniotomy ab interno by Barkan1 in the 1940s and the subsequent introduction of trabeculotomy ab externo developed independently by Harms2 and Smith3 in the 1960s. Multiple studies of trabeculotomy and goniotomy among patients with PCG report success rates of 75% to 90%. The high success rate of angle surgery in PCG has led to the consensus that childhood glaucoma is primarily a surgical disease,4 not only because surgery on the angle is highly effective but also because long-term, compulsive adherence to a multidrug medical regimen in young children is frequently impossible.

Conventional goniotomy and trabeculotomy each treat approximately 120° of the anterior chamber angle. If the response to surgery is insufficient, a return to the operating room may be necessary to treat the remaining angle. Current preferred practice is to perform circumferential surgery, cannulating the canal of Schlemm and opening the entire canal; both ab externo and ab interno options exist. Circumferential treatment is advantageous as the entire angle is treated, allowing the surgeon to move on quickly to alternative treatments if the angle surgery fails. Transcorneal ab interno approaches such as goniectomy-assisted transluminal trabeculotomy (GATT)5 have the added benefit in children of preserving conjunctival real estate for subsequent fistulizing procedures should the angle surgery fail.

Unfortunately, even in the best of hands some 15%-25% of primary angle surgery performed in PCG eventually fails, due to disease severity, delay in diagnosis, age at which surgery is performed, and even specific disease phenotype. For example, certain genetic variants only recently identified result in congenital absence of the canal of Schlemm and downstream collector channels, something that cannot yet be determined clinically. Secondary forms of childhood glaucoma, such as aniridia, Sturge-Weber syndrome, anterior segment dysgenesis syndromes, and glaucoma following cataract surgery (GFCS) may respond poorly, if at all, to primary angle surgery. Despite the lower success rate of angle surgery in these secondary forms of childhood glaucoma, angle surgery is still generally attempted first due to its significant safety advantage compared to the alternatives.

If we define eyes that have failed angle surgery as having “refractory childhood glaucoma,” it is worth recognizing that none of the options currently employed in this setting have been evaluated prospectively prior to widespread adoptions. We are witnessing a revolution of surgical innovation in adult glaucoma, but sadly, childhood glaucoma remains an orphan disease. Some of the new techniques for circumferential ab interno trabeculotomy, such as GATT, clearly advance how we do angle surgery.

Should minimally invasive glaucoma surgery (MIGS) be considered? In general, no. The stakes are too high in children, and angle-based MIGS implants offer no known advantage over goniotomy/trabeculotomy/GATT and have unknown long-term consequences. The CyPass suprachoroidal stent (Alcon; Fort Worth, TX) was removed from the global market in 2018 out of concern for endothelial damage in the adult pivotal trial, and there is no data for the device in children. In at least 1 European case in a buphthalmic eye, the device migrated into the suprachoroidal space, never to be seen again.

Bleb-forming MIGS may be an attractive interim step for refractory childhood glaucoma before moving on to the more extensive dissection and risk associated with trabeculectomy with mitomycin C (MMC) or plate-based glaucoma drainage devices. Small-lumen tubes that shunt aqueous humor to a subconjunctival space treated with MMC are marketed or in development. We have limited data about the Xen (Allergan; Irvine, CA) implant in children, but the observation that the device may degrade over time5 should give pause when considering its use in children. In my lecture I’ll review my recent experience with the PreserFlo Microshunt (Santen USA; Emeryville, CA), which I was able to implant in children under a compassionate use exemption from the FDA.7 As we move forward with new approaches and devices for adult glaucoma that trickle down to children, we must insist on the same level of evidence in children (eg, prospective trials, surgical and/or device registries, etc.) that we take for granted in adults.

Our pediatrician colleagues constantly remind us that children are not just little adults. The same goes for eyes with a history of childhood glaucoma. Unlike adult-onset glaucoma, childhood glaucoma is a panophthalmic disease, affecting all structures of the eye. The eye of an adult who had childhood glaucoma is not the same as other adult eyes. The general ophthalmologist or the adult glaucoma specialist must be prepared to deal with the consequences in a buphthalmic eye (eg, loose lens, very thin sclera, leaking corneal incisions, etc.).

Having focused my practice on childhood glaucoma for more than 3 decades, I’m now operating on the babies of children I operated on years ago. Caring for infants and children is not for the fainthearted. The stakes are high and long term. These patients are best cared for at centers with deep experience and a team in place covering all aspects of their special ophthalmic needs, including amblyopia management, contact lens management for aphakia, etc. Children and their families are poorly served when care is fragmented. We can do better!
References


Anesthesia-Perioperative Considerations—How to Optimize Surgical Preparation

Catherine Marando MD

Introduction

By failing to prepare, you are preparing to fail
—Benjamin Franklin

Perioperative Management of Antithrombotic Therapy (ATT)

There are no formal evidence-based guidelines for the management of perioperative ATT for glaucoma surgery. The decision to continue or temporarily discontinue ATT should be made on a case-by-case basis, where the individual patient’s risk of intraoperative bleeding is weighed against the risk of stroke or cardiac event. Numerous recent studies have suggested that rates of hyphema following minimally invasive glaucoma surgeries (MIGS) are not affected by the use of ATT. This has been mainly demonstrated with the iStent inject (Glaukos Corp.; Laguna Hills, CA, USA), Kahook Dual Blade (KDB, New World Medical; Rancho Cucamonga, CA), Trabectome (Microsurgical Technology; Redmond, WA), and Hydrus Microstent (Ivantis, Inc.; Irvine, CA). However, for more extensive angle-based procedures, such as a 180- or 360-degree gonioscopy-assisted transcleral trabeculotomy (GATT), rates of hyphema at postoperative Week 1 are higher (~30%), and therefore an inability to discontinue ATT is thought to be an absolute contraindication to surgery.

For filtering surgeries, such as trabeculectomy or glaucoma drainage implant (GDI) surgery, practice patterns vary widely. In a 2021 survey of American Glaucoma Society (AGS) members, 64% of respondents routinely stop warfarin prior to trabeculectomy and 47% routinely stop aspirin (ASA). For GDI surgeries, 54% stop warfarin and 42% stop ASA perioperatively. This variation in practice patterns is reflective not only of the lack of standardized guidelines but also of the uniqueness of each patient’s clinical presentation. One study of 347 patients on ATT suggests that use of ATT (whether continued or discontinued) increases the risk of hemorrhagic complications in filtering surgery as compared to controls who are not on ATT (10.1% vs. 3.7% respectively, P = .002). Temporarily discontinuing ATT appeared to reduce the risk of hemorrhagic complications by approximately half; however, this was not statistically significant, likely due to the small sample size and relatively low frequency of these complications. A case control study of 2285 filtering surgeries showed that perioperative use of anticoagulation significantly increased the risk of delayed suprachoroidal hemorrhage (odds ratio: 2.70). A retrospective review of 367 trabeculectomies demonstrated that 50% of those on ASA and 100% of those on warfarin developed postoperative hyphemas as compared to 28% of controls. Given the increased risks of hemorrhagic complications with use of ATT at the time of filtering surgery, it is important to discuss with your patient the risks and benefits of holding ATT, taking into account their total risk profile. For example, aphakia, prior vitrectomy, uncontrolled hypertension and bleeding history have also been associated with an increased risk for suprachoroidal hemorrhage; therefore, in patients with multiple coexisting risk factors, ATT should be discontinued prior to surgery if possible with clearance from the primary care physician (PCP) or cardiologist. If the cardiologist is hesitant to discontinue ATT, a compromise may be a possible solution (eg, stopping warfarin and bridging with enoxaparin). Additionally, anticoagulation (eg, warfarin, heparin) may confer a higher risk for intraocular bleeding than antiplatelet therapy (eg, ASA, clopidogrel) followed by filtering surgery (23% vs. 8%, respectively), so surgeons should consider the type of ATT when weighing risks and benefits. Finally, much of the existing cardiac and medical literature stratifies all eye surgery as low risk. In reality, glaucoma surgery has a much higher risk for severe hemorrhagic complications than cataract surgery. Plus, unlike cataract surgery, excess perioperative bleeding may decrease the success of certain glaucoma procedures. Therefore, the glaucoma community could benefit from more tailored guidelines for higher-risk filtering surgeries in the medically complex patient.

Optimizing Systemic Comorbidities

According to the American Heart Association and the American Association of Cardiology, ophthalmic procedures are the lowest risk and are rarely associated with excess morbidity and mortality (<1%). If a patient has unstable coronary symptoms, decompenated heart failure, significant arrhythmias (eg, high grade atrioventricular block, symptomatic bradycardia, or symptomatic ventricular arrhythmias), or severe valvular disease, then these conditions should be treated prior to elective surgery. In the case of urgent glaucoma surgery, a careful discussion with the cardiologist and anesthesiology team is imperative to optimize medical management and best understand the risks of anesthesia prior to proceeding with surgery if any of these active cardiac conditions are present. Cardiac medications, such as beta-blockers, statins, alpha-2 agonists, ace-inhibitors, and calcium channel blockers, should be continued perioperatively. For stage 3 hypertension (systolic ≥ 180 and diastolic ≥ 110), rapidly acting antihypertensive medications should be given in the preoperative area prior to proceeding with surgery, as these agents act within hours and reduce morbidity as well as intraoperative bleeding risk, the most extreme of which is suprachoroidal hemorrhage.

Patients with diabetes are at increased risk of surgical complications, such as endophthalmitis, cardiovascular events, and poor wound healing. When possible, it is advisable to schedule these patients early in the day to avoid hypoglycemia. For nonurgent surgeries, work with the PCP to optimize glycemic control perioperatively. There are no randomized controlled trials to support a target perioperative hemoglobin A1c; however, one large interdisciplinary organization led by the Royal College of Anaesthetists has suggested that a level of 8.5% or less should be achieved prior to surgery when safe and reasonable to do

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Section IV: Reaching New Pacific Heights in Surgery
so. Consider working with the PCP to reduce the long-acting insulin by half or more the night before and morning of surgery to minimize the risk of hypoglycemia. Oral diabetic medications can generally be continued in patients with normal renal function, with the exception of SGLT2 inhibitors (ie, gliflozins), which may need to be stopped the day before glaucoma surgery to reduce the risk of diabetic ketoacidosis. 

Extremely High Perioperative IOP and Associated Suprachoroidal Hemorrhage

It is now well known that a rapid, large-magnitude reduction in IOP at the time of surgery increases the risk of suprachoroidal hemorrhage. Specifically, the Fluorouracil Filtering Surgery Study Group found no cases of suprachoroidal hemorrhage with preoperative IOPs of 29 mmHg or less but an incrementally increased risk with preoperative IOPs from 30 mmHg to 70 mmHg, with the risk being as high as 25% in the 60-70 mmHg group. In the preoperative area, consider giving intravenous mannitol an hour before the start of surgery to reduce the magnitude of IOP reduction when the eye is entered. Alternatively, at the start of surgery and prior to the conjunctival peritomy, one can create a small paracentesis to allow for a more gradual egress of fluid from the eye. Then, by the time the eye is entered to create a sclerostomy or insert a tube, the magnitude of rapid IOP change is reduced.

Local Anesthesia

There are no standardized guidelines for when to use retrobulbar, peribulbar, subtenon, subconjunctival, or topical anesthesia for glaucoma surgery. Given the numerous potentially devastating consequences of retrobulbar blocks, including optic nerve trauma, globe perforation, and retinal detachment, less invasive techniques have become more common.

General Anesthesia (GA) vs. Monitored Anesthesia Care (MAC)

MAC is preferred to GA for all suitable cases since it reduces risks for systemic morbidity, reduces total case time, and allows for a quicker recovery. Examples of situations in which to consider GA include patients with dementia and patients with severe anxiety who refuse MAC.

Reinforcing Patient Expectations

Most patient counseling occurs preoperatively; however, it is also important to re-enforce expectations immediately prior to surgery. It is often difficult for patients to understand why they would have a surgery that will not improve their vision and may in fact induce more refractive error or blur. Despite your best efforts, they may convince themselves that this surgery will reverse glaucoma damage and be upset with the postoperative result, regardless of how technically perfect the surgery may have been. Taking a few extra moments in the preoperative area to reinforce patient expectations can be beneficial. For example, prior to filtering surgery in a patient with uncontrolled IOPs, one can say, “As we have discussed, the purpose of this surgery is to lower the eye pressure and slow down vision loss in the future. We cannot restore the vision that has already been lost from glaucoma.” Every surgeon will have a different approach in counseling, but a simple reinforcement of expectations in the perioperative setting can be the difference between a happy and an unhappy patient after surgery.

Take-away Points

- Continuing ATT may not significantly increase the risk of perioperative bleeding in some limited, minimally invasive angle-based procedures (eg, iStent, KDB, Trabectome, Hydrus), but ATT may be an issue for more extensive angle-based surgery (eg, 180- or 360-degree GATT).
- Multiple studies suggest that the use of ATT increases the risk of hemorrhagic complications in filtering surgery; therefore, the risks and benefits of temporary discontinuation of ATT must be considered on a case-by-case basis.
- Anticoagulation (eg, warfarin, heparin) confers a higher risk for intraocular bleeding than antiplatelet therapy (eg, aspirin, clopidogrel) following filtering surgery (23% vs. 8%, respectively).
- Stage 3 hypertension (systolic ≥ 180 and diastolic ≥ 110) should be treated with rapid-acting medications if present at the time of surgery in order to minimize the risk of intraoperative bleeding and suprachoroidal hemorrhage.
- Diabetic patients should be counseled on their increased risk for postoperative infection and delayed wound healing. Target a hemoglobin a1c ≤8.5% before elective procedures when reasonable and safe to do so. With input from the PCP, consider halving the long-acting insulin dosage prior to surgery and stopping SGLT2 inhibitors 1 day prior to surgery.
- Rapid, large-magnitude reductions in IOP intraoperatively increase the risk for suprachoroidal hemorrhage and vision loss. Use techniques to lower IOP gradually whenever possible.
- Confirm that patient expectations align with surgeon expectations prior to surgery.

References

Trabeculectomies and bleb-based procedures are here to stay. In the Primary Tube vs. Trabeculectomy study (PTVT), the trabeculectomy had a lower IOP compared to tube shunts for the first 3 years. For Years 4 and 5, the IOPs equalized but the trabeculectomy obtained this pressure on fewer medications. The Xen is a newer bleb-forming procedure that has been shown to be effective in reducing IOP and noninferior to trabeculectomy. Blebs work, and if done well, they can be long-term solutions for patients with glaucoma.

The key to success with bleb-based procedures is like a good concert—you need to create a strong opening act, give a solid performance, and nail the encore.

The opening act for bleb-based procedures is patient selection. Based on the PTVT, patients with starting IOP less than 21 did better with trabeculectomy. For this reason and fewer medications at 5 years, I choose to do a trabeculectomy in a patient who has a lower starting IOP and needs a lower long-term IOP and has an intolerance to medications. PTVT did not show any differences in race and/or age, so I feel comfortable performing trabeculectomies in these cases. For Xen patients we still lack long-term data, but we know needling rates are high and I worry about scarring in African American patients. Finally, a bleb needs babysitting. You must pick a reliable patient for successful bleb formation.

Surgical approach is the main act and performance for successful trabeculectomy surgery and Xen placement. The key to a functioning bleb is adequate dissection. I do this on my trabeculectomy with a Blumenthal Conjunctival Dissector. Xen was found to be noninferior to trabeculectomy with an ab interno approach, but oftentimes you might need an ab externo approach to ensure flow. If you do decide for a solely ab interno, closed conjunctival approach, I recommend primary needling on the table. Mitomycin C (MMC) is a must for all bleb-based procedures. The consensus is still out, but there might be early data that injected MMC is safe and might have a superior bleb morphology when compared to sponges.

Finally, the encore is postoperative management. Aggressive topical steroids are needed for an extended length of time. Any increase in IOP or flattening of the bleb needs to be treated with a laser suture lysis for a trabeculectomy or in-office needling for a Xen. I warn patients about the high-maintenance postoperative period, but if done right it can lead to long-term success.

References
Glaucoma Drainage Device, Part 1: Nonvalved Implants—How to Do It Right

Ying Han MD PhD

I. Introduction
A. History of tube shunt
1. Molteno implant was first invented in the 1970s.
2. Other implants, such as Krupin, Ahmed glaucoma valve, Baerveldt glaucoma implant, and Paul glaucoma implant, were then developed.
B. Trabeculectomy remains the most common surgical intervention, but use of glaucoma drainage devices (GDDs) is on the rise.
1. Secondary glaucoma
2. High-risk populations
C. How does tube shunt work?
1. Silicone tube to equatorial plate
2. IOP reduction is a function of plate surface area.
   a. Larger plate = lower IOP.
   b. Thickness of plate capsule affects outflow of the tube shunt.
D. Type of tube shunt
1. Valved tube shunt: Ahmed valve
2. Nonvalved tube shunt: Baerveldt, Molteno, or Paul tube shunt
3. Brief summary of Ahmed Baerveldt Comparison and Ahmed vs. Baerveldt studies

II. Surgery Video
Video of nonvalved tube shunt to show key elements of placing nonvalved tube shunt, including placing ripcord suture and tying the tube

III. Plate Placement
A. Location of plate: between 2 rectus muscles
B. Underneath Tenon (video of Tenon dissection) vs. supratenon

IV. Tube Placement
A. Anterior chamber tube placement: American Glaucoma Society survey shows 90% of surgeons place tube at anterior chamber.

B. Sulcus tube placement: Between iris and IOL
1. Surgery video to show how to place sulcus tube
2. Intervention study: Compared to anterior segment placement, ciliary sulcus tube implantation may be a preferred surgery approach to reduce endothelial cell loss in pseudophakic patients. A clinical trial is under way to compare endothelial cell loss between anterior chamber tube vs. sulcus tube.

C. Pars plana tube placement
1. Requires a concurrent or antecedent vitrectomy procedure
2. Surgery video to show pars plana tube placement

V. Postoperative Management
A. Use of aqueous suppressant to modulate capsular formation
B. Use of postoperative topical steroid: A retrospective study showed use of topical difluprednate may be associated with better surgical outcome than prednisolone.
C. May consider injection of antifibrotic agents round the plate when there is hypertensive phase: Video of postoperative mitomycin C injection in clinic

VI. Summary of How to Correctly Place Nonvalved Tube Shunt
A. Nonvalved tube shunt may be chosen for patients who need lower IOP while surgeons need to be aware of the risk of hypotony.
B. Meticulous conjunctiva and tenon dissection is important.
C. Tying tube and/or placing ripcord suture is to avoid hypotony and justify outflow postoperatively.
D. Avoid interfering with rectus muscles.
E. Sulcus tube placement may be associated with less endothelial cell loss than anterior chamber tube placement.
F. Postoperative management is important to avoid hypertensive phase and improve surgery outcome.
Glaucoma Drainage Device, Part 2: Valved Implants + Other Special Considerations—How to Do It Right

Brian A Francis MD

I. Aqueous Tube Shunts
   II. Nonvalved (Baerveldt) Implant
      Valved (Ahmed) implant
   III. Ahmed vs. Baerveldt Studies (ABC, AVB)
   IV. Reducing Complications Specific to Valved Shunts
   V. Aqueous Tube Shunt Surgery: 5 Pearls
      A. Tube erosion
      B. Capsule patch graft
      C. Episceral dissection
      D. Tube exchange
      E. What to do when a tube fails
   VI. Tube Shunt Erosion
   VII. Capsule Patch Graft
   VIII. Aqueous Tube Shunt—Episceral Dissection
   IX. Aqueous Tube Shunt—Tube Shunt Exchange
   X. When a Tube Shunt Fails
      A. Aqueous inflow surgeries
      1. Micropulse transscleral laser therapy
      2. Endoscopic cyclophotocoagulation (ECP)
      B. Tube exchange valved for nonvalved
      C. Second tube
      1. Inferior temporal quadrant preferred
      2. But can use superior nasal quadrant if diplopia is not a consideration
   XI. ECP in the Management of Uncontrolled Glaucoma With Prior Aqueous Tube Shunt
   XII. Outcomes of MicroPulse Transscleral Laser Therapy in Eyes With Prior Glaucoma Aqueous Tube Shunt
Angle-Based Surgery—Best Practices

*Arsham Sheybani MD*

I. Angle Anatomy
   A. Identification of trabecular meshwork in clinic
   B. Identification of trabecular meshwork in the operating room

II. How to Achieve the Best View of the Angle in Surgery

III. The Factors that Go Into Device or Surgical Instrument Selection

IV. How MIGS Trial Data Can Be Integrated in the Informed Decision-Making of Physician and Patient

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**Figure 1**

**Perspective**

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<th>What we expect to see</th>
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**Perspective**

<table>
<thead>
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<th>What we do see</th>
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Figure 1
Newer Devices in the Pipeline and Available Outside the United States

Matt Schlenker MD

I. Overview of the Options
   A. Implants
   B. Surgical device/instruments
   C. Laser

II. In Use Elsewhere Than the United States
   A. Xen 63
   B. PreserFlo

III. Promising in the Pipeline
   A. Drainage to ocular surface
   B. Aqueous outflow at meshwork
   C. Laser trabeculoplasty
   D. Schlemm canal
   E. Aqueous production
   F. Supraciliary drainage
   G. Subconjunctival

IV. Reflections and Key Takeaways
Congenital Glaucoma: Goniotomy Alternatives; and What to Do When Goniotomy Fails

Allen Beck MD

I. Goniotomy Alternatives
   A. Conventional trabeculotomy
   B. Circumferential trabeculotomy
   C. Gonioscopy-assisted transluminal trabeculotomy (GATT)
   D. Combined trabeculotomy/trabeculectomy ± mitomycin C
   E. Trab 360/Omni Surgical System (Sight Sciences)

II. What to Do When Angle-Based Surgery Fails
   A. Glaucoma drainage device
   B. Trabeculectomy with mitomycin
   C. Cyclophotocoagulation
   D. Novel techniques such as PreserFlo ab externo microshunt (Santen)

Selected Readings
Aphakic Glaucoma, a Primer: Infant Aphakia Treatment Study, Best Practices

Courtney L Kraus MD

I. Definition of Glaucoma Following Cataract Surgery
   A. Congenital Glaucoma Research Network (CGRN) criteria for glaucoma
   B. CGRN for glaucoma following cataract surgery
      1. Supersedes conflicting classifications
      2. Includes all cataract entities (acquired and congenital)

II. Early Theories of “Aphakic” Glaucoma
   The IOL as “protective”

III. Infant Aphakia Treatment Study (IATS)
   A. Methods: multicenter (12), randomized trial
      1. Infants 4 weeks to <7 months of age with unilateral cataract
      2. Exclusion criteria included corneal diameter (KDM) <9 mm, IOP >25, persistent fetal vasculature syndrome (PFV), stretching ciliary processes, retinal or optic nerve disease
      3. Randomized to IOL implantation with hyperopia vs. intentional aphakia and contact lens management
      4. 10 years of follow-up on VA, IOP, nerve assessment
   B. Study question: Was IOL implantation superior in achieving visual outcomes to intentional aphakia?
   C. Results
      1. VA results: There was no significant difference between the median VA of operated eyes in children that underwent primary IOL implantation and those left aphakic.
      2. Glaucoma results: Data on cases of glaucoma and glaucoma suspects available at age 10.5 following randomization in IATS
         a. Risk of glaucoma after cataract removal rose from 9% at Year 1 to 17% at 5 years to 22% at 10 years.
         b. Risk of glaucoma + glaucoma suspect rose from 12% at Year 1 to 31% at 5 years to 40% at 10 years.
         c. Risk of glaucoma and risk of glaucoma + glaucoma suspect did not differ between groups.
   d. Associations with treatment group (IOL vs. contact lens), age at surgery (<48 days vs. >48 days), PFV, corneal diameter (<10 mm vs. >10 mm), and IOP (<12 mmHg vs. >12 mmHg)
   e. After controlling for age, all other terms were not significant.

IV. Similar Studies
   Unilateral/Bilateral Toddler Aphakia and Pseudophakia Treatment Study (TAPS), IOLunder2, Pediatric Eye Disease Investigator Group (PEDIG) Cataract Outcomes Study (CO2)
   A. IOLunder2: Prospective observational cohort study at 31 sites in the UK and Ireland
      1. Bilateral and unilateral cataract
      2. Primary IOL implantation was not associated with increased risk of glaucoma; a reduction in glaucoma risk was seen with each additional week of age at surgery.
      3. Median time to glaucoma development was 3.6 months in bilateral cases, 1.8 months in unilateral.
   B. PEDIG CO2
      1. Cohort longitudinal registry from 45 institutions and 16 community sites in the United States
      2. Cataract extraction prior to age 13
      3. Glaucoma results at 5 years
         a. Risk of glaucoma + glaucoma suspect at 5 years was 29% for children initially managed with aphakia.
         b. Risk of glaucoma + glaucoma suspect at 5 years was 7% for children with primary IOL placement.
         c. Development of glaucoma in aphakes was associated with (1) age <3 months, (2) abnormal anterior segment, (3) intraoperative complications during lensectomy, and (4) bilateral cataracts.
C. TAPS

1. Same study sites/surgeons as IATS, but studied bilateral children 4 weeks to <7 months and unilateral and bilaterally affected children 7-24 months, retrospectively during same study period as IATS.

2. IOL implantation was at discretion of surgeon.

3. Glaucoma results: Glaucoma suspect much lower among 7-24 month cohort; no bilateral child was diagnosed with glaucoma.

4. Glaucoma rates were similar to IATS at 5 years (30%) in the young cohort of bilateral cases.

References


Uveitic Glaucoma: Goniotomy’s Role, Steroid Response

Ken K Nischal MBBS

Introduction
Uveitic glaucoma is one of the most serious complications of intraocular inflammation, but its pathogenesis is not completely understood. The type of inflammation, steroid response, and anterior chamber anatomical relationships likely play an interactive role, with both acute and chronic types occurring in closed or open angles.

In eyes with closed-angle uveitic glaucoma, there are 3 mechanisms: seclusion pupillae, peripheral anterior synechiae, and forward rotation of the ciliary body.

Increased pressure in uveitic eyes is associated with poorer outcomes. We are going to discuss the role of steroid response in the development of uveitic glaucoma and the role of goniotomy for treating uveitic glaucoma.

Steroid Response
An increase in IOP related to topical steroid use has been reported in 18%-36% of patients. The response is expected to present 2-6 weeks after therapy initiation but can develop in a few days, especially in children. Studies have shown that timely intervention with medical therapy can avert the need for surgical intervention but usually only if the steroids can be reduced or stopped. Continued steroid use topically makes glaucoma control even more difficult. The use of agents that have been designed to reduce steroid response, such as loteprednol etabonate, is well described, but whether their use helps control IOP better once a steroid response has occurred or been initiated is unclear.

Goniotomy
While filtration surgery and glaucoma drainage devices have been used in control of uveitic glaucoma, evidence for the use of angle surgery has become more available recently. Both goniotomy and 360-degree trabeculotomy have shown promising results. This increased interest in angle surgery for uveitic glaucoma has also opened the door for various minimally invasive glaucoma surgery (MIGS) techniques to be considered.

Summary
Management of uveitic glaucoma is complex and invariably needs a multidisciplinary approach between uveitis and glaucoma experts to attain the most favorable outcome for the patients.

Selected Readings
Juvenile Open-Angle Glaucoma: How Is Treatment Different From That of Adult OAG?

Beth Edmunds MD PhD

I. A Childhood Glaucoma Research Network (CGRN)
Classification of Childhood Glaucoma1: Primary Glaucomas
A. Primary congenital glaucoma (PCG): onset before 3 years of age
B. Juvenile open-angle glaucoma (JOAG): onset after age of 3 and up to age of 40 years

II. Variable Phenotypes Within JOAG
A. The “buphthalmic spectrum”: Observations from clinical practice, the spillover of “buphthalmos” into JOAG, and why this is important in the treatment of JOAG

As with adult-onset primary open-angle glaucoma (POAG), JOAG is defined as a disease in which raised IOP results in characteristic optic nerve damage and visual field (VF) defects (the definitions of both also include a gonioscopically normal-appearing angle). Unlike PCG, where the angle is visibly immature on gonioscopy and the onset of raised IOP and tissue damage occurs before the age of 3 years, JOAG encompasses those POAGs where the onset of raised IOP and tissue damage occur after the age of 3 years. PCG is also defined by the secondary effects of raised IOP on other globe tissues, with a characteristically enlarged eye, described as buphthalmos. This panophthalmitic manifestation of raised IOP is due to the effects of raised IOP on the outer coats of the globe (cornea and sclera), which are at their most soft, distensible, and stretchable in the very young eye, becoming less susceptible as the eye matures.

Thus, even though the classification system creates a watershed between PCG and JOAG at 3 years of age, with apparently clear distinctions in the anatomy of the angle and effects of IOP on the globe, there is some spillover of buphthalmos-type features to be found in some eyes in the JOAG category. Biological tissue properties tend to behave on a continuum, and so they manifest on a “buphthalmic spectrum” beyond the cut-off of 3 years. Apart from Haab striae, which reliably chronicle a high-IOP insult prior to the age of 2 years, the other features of buphthalmos can either develop and/or continue to progress beyond this, though with diminishing effect.

This stretching and thinning response to IOP wanes first for the cornea (tailing off around 3 years of age) and then for the sclera (may last into late childhood in a few patients, which likely explains the myopia found in many JOAG eyes). The manifestations of raised IOP in a JOAG eye will depend on individual susceptibility factors, which are not well studied and likely multifactorial, including genetic. Their interplay with the IOP insult—determined by age of onset and “dose” of IOP—is also likely multifactorial, including genetic; the best characterized of these thus far being mutations of the myocilin gene. This produces considerable phenotypic variation within the category of JOAG, ranging from POAG-like (optic nerve damage only) to PCG-like (panophthalmitic damage). So this is an alert to all ophthalmologists, that JOAG is not necessarily younger-onset POAG, and its treatment benefits from the input and skills of clinicians and surgeons versed in PCG as well as POAG.

B. Genetic testing in JOAG

As for POAG, this is an evolving area. However, the stakes are higher in juvenile-onset disease as there is a greater lifetime of glaucoma risk ahead. Identifying the presence (or absence) of genetic mutations implicated in glaucoma in the individual (and discovering new ones) can allow more tailored surveillance and treatments. Currently, approximately 30% of JOAG can be explained by one of the known genetic mutations; there are several genes that have been identified with pathogenic mutations, with variable inheritance patterns.2 The AD-inherited-myocilin-mutation phenotype is relatively well known, and CYP1B1 is another of the more common genes whose mutations are associated with JOAG. For both, the presence of a pathogenic mutation is associated with a more aggressive and early-onset form of JOAG.

There are many reasons why genetic testing is not routinely practiced, and this will hopefully change as payers recognize its health value, as the costs of testing lessen, and as there are better testing panels/ algorithms to produce usable information. Interpreting the results of these tests remains the province of experienced geneticists. See a useful article from AAO’s EyeNet Magazine that covers this in some detail.3
III. Three Important Reasons You Should Know About JOAG

A. All practitioners treat patients with JOAG, sometimes unknowingly.

An enlarged eye resulting from the effects of IOP on youthful globe tissue (“buphthalmos”) is found in adults too, because children, teens, and younger adults with early-onset glaucoma grow older and age, with their eyes showing the anatomical stigmata and behaviors of that original early-onset IOP insult, as well as bearing the scars of its subsequent course and treatment(s).

B. Young patients with JOAG are not mini POAG baby boomers.

The behavior and impact of IOP on the eye in JOAG has many similarities to, but also many differences from, those of an adult-onset POAG eye. The general function of other body systems (which support the eye) and metabolism will differ between the young and the old. This is relevant when making treatment decisions. Response to medications may be different (see Selvan’s paper2 for more detail), and ocular tissues may behave differently at surgery. Further, patients with younger-onset disease may warrant more aggressive IOP lowering and prompter inflection points for initiating or escalating treatment, with the goal of preserving a sighted lifetime. Younger people’s lifestyle priorities, employment requirements, and attitudes to treatment may be different from those of the older generations. JOAG patients fall within the child-bearing age, which can have profound implications on treatment options (see AAO website on glaucoma drugs in pregnancy/nursing4). Most teens will still have family members involved in their care, whose engagement must also be sought.

Managing and “seeing” the whole patient is crucial to treatment success in chronic diseases with burdensome treatments, such as glaucoma. In JOAG, ensuring that the whole care team’s attitudes and approaches accommodate a younger person’s developmental stage, as well as a generational difference in world perspectives, may need to be intentional, as these patients are a minority in the typical glaucoma clinic.

C. JOAG and the happy surgeon

1. Beware the surgeon’s Bermuda triangle of (1) distorted anatomical and surgical landmarks, (2) thinned floppy tissues that predispose to leaks and wound gape if not appropriately sutured, and (3) hypotony with its many complications—often more profound and difficult to treat in children, teens, and young adults with myopic eyes on the “buphthalmic spectrum.”

2. Consider the following:
   a. Careful preop exam to recognize occult buphthalmos and/or myopia
   b. Adjusting surgical wound site to ensure correct location relative to limbus and ciliary body, using a safe technique for filtering surgery (see Moorfields Safer Surgical System5,6)
   c. Using intraoperative anterior chamber (AC) maintainer (cannula attached to bottle of BSS) in tube and trabeculectomy (and other) surgeries, both to avoid intraoperative hypotony and also to assess fluid dynamics, AC stability, and adequacy of wound closure (using Weck-Cel sponges to test the integrity of incision closure; viscoelastic in the AC can disguise a leaky wound, oozing suture track, or floppy tissue).

References

Exam/EUA/IOP: How-to, Anesthesia, CCT, Tonometer Types

Karen M Joos MD PhD

I. Childhood Glaucoma Research Network (CGRN)
   Glaucoma Classification
   Requires at least 2 criteria
   A. IOP >21 mmHg: examination under anesthesia (EUA) - anesthesia discretion variable effects on all IOP assessment methods
   B. Optic disc cupping: progressive increase in cup-to-disc ratio, cup-disc asymmetry of ≥0.2 when the optic discs are similar size, or focal rim thinning presence
   C. Cornea: Haab striae or diameter ≥11 mm in newborn, >12 mm in child <1 year, or >13 mm any age
   D. Progressive myopia, myopic shift, or an increase in axial length greater than normal growth
   E. Reproducible visual field defect consistent with glaucomatous optic neuropathy without another observable cause

II. Glaucoma Suspect
   No IOP-related damage and one of the following criteria:
   A. IOP >21 mmHg on 2 separate occasions
   B. Suspicious optic disc appearance for glaucoma (ie, increased cup-to-disc ratio for size of optic disc)
   C. Suspicious visual field for glaucoma
   D. Increased corneal diameter or axial length in setting of normal IOP

III. Attempt Office Exam
   The key is a pleasant experience. Avoid fear. It’s helpful if parents are encouraging.
   A. < 2 months old: Arrive hungry to place drops, then fall asleep during feeding, screening and postoperative exams
      1. Tonometry
      2. Portable slit-lamp exam
   B. About 3 months old to about 3 years old: Limited office exam, usually EUA
   C. ≥3 years old: Playful and bribable (stickers, tattoos, parental treats) unless developmentally delayed
      1. Use favorable terminology
         a. The slit lamp is “a motorcycle with handlebars and a head helmet.” Make motorcycle noises during the exam.

IV. EUA Components
   A. Mask gas anesthesia (rarely preoperative sedative): initial tonometry when anesthesiologist permits
   B. Laryngeal mask airway (LMA)/endotracheal tube
      1. Additional tonometry readings throughout the examination
      2. Corneal pachymetry (central corneal thickness, CCT): normal = 537-558 microns
      3. Corneal diameters: Abnormal diameter = ≥11 mm in newborn, >12 mm in child <1 year, or >13 mm any age
      4. Examine anterior segment; look carefully for Haab striae
         a. Surgical microscope
         b. Portable slit lamp
         c. High-resolution anterior ultrasound
      5. Gonioscopy: direct gonioprism
      6. Axial length: initial and monitor growth over time
         a. Premature normal mean
            i. 24 weeks: 11.47 mm
            ii. 28 weeks: 13.39 mm
            iii. 32 weeks: 15.51 mm
            iv. 36 weeks: 16.20 mm
b. Term normal mean
   i. Full-term: 16.83 mm
   ii. 1 month: 17.22 mm
   iii. 3 months: 18.41 mm
   iv. 6 months: 19.53 mm
   v. 1 year: 20.74 mm
   vi. 2 years: 21.71 mm
   vii. 3 years: 21.84 mm

7. B-scan ultrasound if dilation deferred for surgery

8. Dilation to evaluate cup-to-disc ratio and retina; surgical microscope stereo view; a retina flat lens is valuable.

V. Anesthesia

A. Agents reported to lower IOP: halothane, propofol, sevoflurane, isoflurane, enflurane

B. Agents reported to raise IOP: ketamine, succinylcholine, suxamethonium, endotracheal tube intubation

C. Agents reported to minimally affect IOP: nitrous oxide, midazolam, chloral hydrate, LMA

VI. Tonometer Types

Pediatric IOP meta-analysis: mean 16.22 mmHg; ranges: 12.02-17.38 mmHg

A. Palpation

B. Appplanation: fixed-area tonometers, fixed-force tonometers

C. Noncontact tonometers

D. Indentation tonometers

E. Appplanation-indentation tonometers

F. Dynamic contour tonometers

G. Rebound tonometers

VII. CCT

Normal: 537-558 microns

A. Instruments: ultrasound pachymetry, Scheimpflug camera, optical coherence pachymetry, OCT, confocal microscopy

B. Thicker with aphakia: 651 ± 64 microns

C. Thicker in presence of corneal edema

D. Careful differential: Corneal entities with normal IOP and cup-to-disc ratio such as congenital hereditary endothelial dystrophy (CHED) or Harboyan syndrome—do genetic testing.

References


How Are Trabs and Tubes in Kids Different Than Those in Adults?

Ta Chen Peter Chang MD

I. Before Surgery
   A. Set expectations and communicate them
   B. Choose your poison (anesthesia)
      1. General
      2. Local

II. Day of Surgery: Choose Your Own Adventure
   A. Tube or trab: How to decide?
   B. Tube: Tips and pearls
   C. Trab: Tips and pearls

III. After Surgery
   A. Medications
   B. Timing of follow-up
   C. How to gauge outcome and monitor complications?

IV. Summary
Trabeculectomy Complications

Aakriti Garg Shukla MD

I. Introduction
Trabeculectomy remains the gold standard management option for advanced glaucoma. The literature details its efficacy in lowering IOP and halting glaucoma progression. In this presentation, we will detail its intraoperative and postoperative complications and their management options.

II. Intraoperative Complications
A. Thin scleral flap and/or poor reapproximation
B. Excessive subconjunctival hemorrhage adjacent to scleral flap
C. Presentation of vitreous or iris bleed during iridectomy
D. Suprachoroidal hemorrhage
E. Malignant glaucoma
F. Difficulty with wound closure

III. Postoperative Complications
A. Associated with elevated IOP
1. Bleb encapsulation
2. Malignant glaucoma
B. Associated with low IOP
1. Wound/bleb leak
2. Shallow or flat anterior chamber
3. Hypotony maculopathy
4. Choroidal effusion
5. Suprachoroidal hemorrhage
C. Other
1. Hyphema
2. Persistent corneal edema
3. Cystoid maculopathy
4. Anterior migration of bleb, irregular astigmatism
5. Bleb dysesthesia
6. Early or late blebitis/endophthalmitis

IV. Management
Options for each situation will be discussed. For instance, in cases of postoperative malignant glaucoma, a combination of intensive aqueous suppression, cycloplegia, and anti-inflammatory agents is used as a first-line option. Viscoelastic agents can be used to reform the anterior chamber. In pseudophakic or aphakic patients, YAG laser can be used to perform an iridotomy, capsulotomy, and anterior hyaloidotomy. If this does not lead to resolution of malignant glaucoma or if the eye is aphakic, surgical irido-zonulohyaloido-vitrectomy is the next step.

References
Early Tube Complications

Steven J Gedde MD

Introduction

Tube shunts are being used with increasing frequency as an alternative to trabeculectomy. Medicare claims data show a 70% decrease in the number of trabeculectomies and a concurrent 493% increase in tube shunt implantation between 1994 and 2016.1,2 Anonymous surveys of the American Glaucoma Society membership have also demonstrated a rise in the selection of tube shunts and a decline in the popularity of trabeculectomy in a variety of clinical settings.3 Similar postoperative complications can develop after trabeculectomy and tube shunt placement. Additionally, unique complications may occur with tube shunt surgery, associated with implantation of a device. Randomized clinical trials have found a higher rate of early postoperative complications following trabeculectomy with mitomycin C compared with tube shunt surgery.4,5 Table 1 shows the frequency of early postoperative complications in several prospective trials involving tube shunts.4-6 Prompt diagnosis and proper management of these complications can optimize patient outcomes.

Hypotony

Hypotony should not be considered a complication of glaucoma surgery, but low IOP does increase the risk of certain postoperative complications. Hypotony maculopathy, choroidal effusion, anterior chamber shallowing, corneal edema, cystoid macular edema, and suprachoroidal hemorrhage are surgical complications associated with hypotony. Valved implants have a flow restrictor designed to prevent hypotony, and these devices should be used in eyes in which aqueous hyposecretion may be present (eg, uveitic glaucoma, previous cyclodestruction). Valve malfunction can still result in early postoperative hypotony. I routinely fill the anterior chamber with a cohesive viscoelastic during valved implant surgery to allow a more gradual reduction of IOP postoperatively. Early postoperative hypotony may develop following placement of a nonvalved implant because of incomplete tube ligation, excess flow through fenestrations, or ligature release. Leakage around the tube can produce early hypotony with both valved and nonvalved implants, but use of a 23-gauge needle creates a tight tube track that reduces the possibility of leakage at the tube entry site.

Select Complications

Tube obstruction

Obstruction of the distal tube tip may occur with iris, fibrin, blood, or vitreous. If tube patency is uncertain, B-scan ultrasonography can be used to evaluate for fluid surrounding the end plate. Tube obstruction is more common when tubes are placed in the ciliary sulcus or through the pars plana compared with anterior chamber tube insertion. Beveling the tube anteriorly for anterior chamber tube insertion and posteriorly for sulcus placement can reduce the risk of tube obstruction. When inserting the tube through the pars plana, a complete pars plana

Table 1. Early Postoperative Complications After Tube Shunt Surgery in Randomized Clinical Trials

<table>
<thead>
<tr>
<th>Complication</th>
<th>PTVT Studya</th>
<th>TVT Studya</th>
<th>ABC Studyb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shallow or flat anterior chamber</td>
<td>10%</td>
<td>10%</td>
<td>20%</td>
</tr>
<tr>
<td>Choroidal effusion</td>
<td>7%</td>
<td>14%</td>
<td>10%</td>
</tr>
<tr>
<td>Hyphema</td>
<td>6%</td>
<td>2%</td>
<td>17%</td>
</tr>
<tr>
<td>Tube obstruction</td>
<td>0%</td>
<td>0%</td>
<td>9%</td>
</tr>
<tr>
<td>Diplopia</td>
<td>–</td>
<td>–</td>
<td>5%</td>
</tr>
<tr>
<td>Cystoid macular edema</td>
<td>1%</td>
<td>0%</td>
<td>2%</td>
</tr>
<tr>
<td>Hypotony maculopathy</td>
<td>1%</td>
<td>0%</td>
<td>2%</td>
</tr>
<tr>
<td>Vitreous hemorrhage</td>
<td>0%</td>
<td>1%</td>
<td>2%</td>
</tr>
<tr>
<td>Suprachoroidal hemorrhage</td>
<td>0%</td>
<td>2%</td>
<td>2%</td>
</tr>
<tr>
<td>Malignant glaucoma</td>
<td>0%</td>
<td>3%</td>
<td>0%</td>
</tr>
<tr>
<td>Endophthalmitis</td>
<td>0%</td>
<td>0%</td>
<td>1%</td>
</tr>
<tr>
<td>Overall rate of early postoperative complications</td>
<td>19%</td>
<td>21%</td>
<td>58%</td>
</tr>
</tbody>
</table>

Abbreviations: ABC, Ahmed Baerveldt Comparison; PTVT, Primary Tube Versus Trabeculectomy; TVT, Tube Versus Trabeculectomy.

aOnset ≤1 month after surgery
bOnset ≤3 months after surgery
cTVT and PTVT studies only evaluated for diplopia after 3 months postoperatively.
vitrectomy, with trimming of the vitreous base in the respective quadrant, should be performed to minimize the chance of vitreous obstruction of the tube. Intracameral tissue plasminogen activator can dissolve fibrin or blood obstructing a tube. Laser treatment can be used to open a tube occluded by iris or vitreous. Vitreous incarceration within a tube is difficult to resolve with laser, and vitrectomy is frequently required. Trypan blue dye can be used to confirm tube patency after intraoperative management to relieve tube obstruction.

Suprachoroidal hemorrhage
Suprachoroidal hemorrhage is a rare but sight-threatening complication of intraocular surgery. Hypotony appears to be the major precipitating factor, resulting in rupture of the posterior ciliary artery and bleeding into the suprachoroidal space. A suprachoroidal hemorrhage typically presents with severe ocular pain, anterior chamber shallowing, increased IOP, and choroidal elevation. The occurrence of suprachoroidal hemorrhage after nonvalved tube shunt surgery has a bimodal distribution, corresponding to the 2 times when IOP is usually lowest—that is, immediately postoperatively and following tube opening.

The risk of this complication is higher in patients with advanced age, anticoagulant therapy, uncontrolled hypertension, high myopia, prior intraocular surgery, choroidal hemangioma, markedly elevated preoperative IOP, and hypotony and/or large drops in IOP. Primary prevention is aimed at mitigating modifiable risk factors. Blood pressure should be adequately treated, anti-platelet and anticoagulant therapy suspended when possible, and IOP maximally lowered preoperatively. Rapid decompression of the globe should be avoided intraoperatively, and patients should be advised against Valsalva maneuvers postoperatively. Medical management of a suprachoroidal hemorrhage includes topical steroids, cycloplegics, aqueous suppressants, and oral analgesics as needed. Surgical drainage is indicated in the presence of uncontrolled IOP, flat anterior chamber, appositional (“kissing”) choroidals, and retinal detachment. It is desirable to delay surgical intervention for 7-14 days to allow clot lysis. Ultrasonography can help diagnose and direct treatment of a suprachoroidal hemorrhage by determining the extent of the hemorrhage, status of the retina, and degree of clot liquefaction.

Malignant glaucoma
Malignant glaucoma is characterized by axial shallowing of the anterior chamber, patent iridectomy, and absence of suprachoroidal blood or fluid. The IOP is often markedly elevated but may be normal. Angle-closure glaucoma, axial hyperopia, and a history of malignant glaucoma in the contralateral eye are risk factors for malignant glaucoma. In a case series of malignant glaucoma after tube shunt implantation, the median time to diagnosis was 33.5 days, and 80% of eyes developed the disorder within 2 months of surgery. The close temporal relationship between the development of malignant glaucoma and tube ligature release suggests that patients should be carefully monitored during the time of tube opening, especially those predisposed to this complication. Medical treatment consists of topical steroids, cycloplegia, and aqueous suppressants. Disruption of the anterior hyaloid with the Nd:YAG laser may resolve malignant glaucoma in some aphakic and pseudophakic eyes. When medical and laser therapy fails, pars plana vitrectomy or iridozonulohyaloidectomy should be performed to establish a direct communication between the posterior and anterior segments.

Conclusions
Early postoperative complications are common after tube shunt surgery, but most are transient and self-limited. It is not surprising that glaucoma procedures that produce greater IOP reduction also have a higher risk of hypotony-related sequelae. Unfortunately, a trade-off exists between efficacy and safety with currently available glaucoma operations. Patients with advanced and/or progressive glaucoma benefit from aggressive IOP reduction with traditional glaucoma surgery, as glaucoma procedures that produce greater IOP reduction are more likely to stabilize the disease. Early recognition and appropriate treatment of early tube complications can optimize patient outcomes.

References
Late Tube Complications

Jessica M Kang MD

Late Tube Complications in Clinical Trials

I. Primary Tube vs. Trabeculectomy Study (PTVT) 3-Year Outcomes
A. 33% of patients in the tube group had late postoperative complications.1
B. Most common (11%): encapsulated bleb
C. Other complications (1%-2%): persistent diplopia, shallow/flat anterior chamber, dysesthesia, hypotony maculopathy, iritis, choroidal effusion, cystoid macular edema, conjunctival cyst, plate erosion, tube retraction

II. Ahmed vs. Baerveldt (AVB) Study 5-Year Outcomes
A. Did not specifically separate out early/late complications (Ahmed/Baerveldt %)2
B. Most common long-term complication: corneal edema (11/12%)
C. Persistent iritis (7%/12%), motility disorder or persistent diplopia (5%/2%)

III. Ahmed Baerveldt Comparison (ABC) Study 5-Year Outcomes
A. Late complications (after 3 months) (Ahmed/Baerveldt %)3
B. Most common late complication in both groups: corneal edema (20.1%/20.4%)
C. Other common complications: diplopia (12.7%/11.8%), cystoid macular edema (6.2%/7.2%), cornea graft rejection (7.1%/7.0%), recurrent/persistent iritis (6.2%/5.5%)

Different Surgical Techniques to Address Late Tube Complications

I. Covering the Tube
A. When would we do this?
   1. Tube erosion: This can occur in 1%-8% of cases.1,4
      a. Risk factors: Some reported risk factors include prior intraocular surgery, concomitant surgery, and an inferior location.4-8 Other risk factors such as age, gender, or race have varying results across different studies.5-11 The data is limited due to the retrospective nature and smaller sample size of these studies.
      b. Presence of a tube/plate erosion is a significant risk for endophthalmitis.12-15

B. How do we do this?
   1. Re-exposure rates can be high (18%-44%).16,17
   2. Not using a patch graft can increase risk of subsequent re-exposure.18
   3. Many different patch graft materials and methods of tube exposure revision have been described.11 Some studies suggest scleral patch grafts may be protective against risk of exposure or re-exposure.5,17 However, other retrospective studies have not found a difference between patch graft materials.4,10 If using a pericardium patch graft, a double layer may be more protective against exposure than a single layer.19
   4. Other considerations
      a. Posterior repositioning of the tube to the ciliary sulcus or pars plana at the time of the repair can reduce the extraocular portion of the tube that is subject to desiccation and mechanical trauma.20,21
      b. Rerouting tube closer to 12 or 6 o’clock can allow for better lid coverage.
      c. In cases of limited conjunctiva, a conjunctival pedicle flap22 or buccal mucous membrane graft23 may be considered.

II. Repositioning the Tube Posteriorly
A. When would we do this?
   1. Tube erosion (see above)
   2. Corneal decompensation: Damage to corneal endothelial cells may be due to mechanical damage from the tube, high fluid flow, and/or postoperative inflammation.24 A more posterior tube placement may help mitigate these factors.
      a. Insertion of the tube in the vicinity of or anterior to the Schwalbe line, as well as a shorter tip length, was associated with more endothelial cell density (ECD) loss in Baerveldts.25
      b. A nonrandomized study comparing Ahmed tube placement in anterior chamber (106 eyes) vs. ciliary sulcus (105 eyes) demonstrated higher mean monthly ECD loss in the anterior chamber group compared with the sulcus group.26 The length of the tube in the anterior chamber was not associated with worse ECD. Two other smaller studies had similar results of greater ECD loss in anterior chamber tubes compared to sulcus tubes.27,28
B. How do we do this?

1. When repositioning to the sulcus, rebevel the tube from bevel-up to bevel-down to avoid occlusion from iris.20

2. Repositioning to the pars plana may require concurrent pars plana vitrectomy (PPV) to avoid occlusion from vitreous, unless a thorough vitrectomy has previously been performed.21

3. If the tube tip is too short to visualize after repositioning posteriorly, an endoscopic cyclophotocoagulation probe can be used to visualize the tip.

III. Removing the Tube

A. When would we do this?

1. Endophthalmitis: Rates of tube-related endophthalmitis are low (1%-2%) but more common in the pediatric population.29
   a. Risk factors: Exposure of the device is a major risk factor for tube-related endophthalmitis,12-15 and exposure over an inferior implant is more likely to be associated with infection than a superior one.7
   b. Treatment: Same-day injection of intravitreal antibiotics is first-line therapy.15 Decisions to consider include whether to remove vs. retain the device and whether to perform early PPV. In a literature review of 88 cases, the device was explanted in 70.5% of cases, and explantation was associated with a lower rate of evisceration/enucleation.29 Visual outcomes between the 2 groups (explant vs. retain) were similar. PPV was performed in 37.8% of cases and was associated with a lower rate of evisceration/enucleation.29

2. Dioplia
   a. Decreased motility and ocular misalignment may not always result in dioplia.30
   b. While dioplia can often be managed with prisms or strabismus surgery, surgical treatment may include trimming or removing the fibrous capsule and/or the tube shunt.31-33

3. Tube exchange: Can consider a valved to nonvalved exchange if the IOP is too high or a nonvalved to valved exchange if the IOP is too low.34

B. How do we do this?

1. If the sclerostomy is leaking or not well closed with sutures alone, it can be plugged with pericardium.35,36

2. All fibrous stalks through fixation holes of the plate must be identified and amputated.

3. Depending on the indication, the capsule of the tube may also be removed; this can be used as a patch graft if implanting another tube.34

References


Angle Surgery Complications: Failure to Launch

Davinder S Grover MD

I. Introduction of Goals
   A. This talk will focus on initiating angle surgery.
   B. Subsequent talk will discuss postop issues.

II. First Necessary Goal of Angle Surgery: Visualization
   A. Angle anatomy
   B. Intraoperative gonioscopy
   C. Pearls for optimal view

III. Failure to Launch Stents
   A. Pearls for success with iStent
   B. Pearls for success with Hydrus

IV. Failure to Launch Goniotomy
   Pearls for success with managing Kahook Dual Blade/ Tanito goniotomy

V. Failure to Launch Circumferential Trabeculotomy
   Pearls for success cannulating canal with suture/catheter

VI. Backup Plan if Initial Surgery Cannot Be Completed
   Strategy and how to discuss issue with patient

VII. Conclusion
Angle Surgery Complications: Hyphema and Hypotony

Michael Lin MD

I. Preventing Complications of Angle Surgery
   A. Understand why hyphema and hypotony happen
      1. Tissue bleeding
      2. Blood reflux from angle
      3. Patient factors
         a. Anticoagulation
         b. Valsalva maneuvers
         c. Increased episcleral venous pressure
   4. Angle surgery in the absence of suprachoroidal space entry usually should not cause IOP to go below episcleral venous pressure (EVP), but hypotony can develop if cyclodialysis cleft occurs.

B. Preoperative approaches
   1. Procedure selection
      a. Consider stenting or endoscopic cyclophotocoagulation instead of goniotomy if anticoagulated.
      b. Stenting and goniotomy are not mandatory, and sometimes the best course of action may be to do stand-alone cataract surgery.
   2. Patient selection and counseling
      a. Discuss postoperative activity restrictions and head-up positioning.
      b. Acetaminophen instead of ibuprofen for pain control after surgery
      c. Stool softeners to avoid bearing down due to constipation
   3. Coordination with other physicians
      a. Anesthesiologist and primary care physician can help with blood pressure control.
      b. Primary care physician and cardiologist can help with perioperative anticoagulation management.
      c. Retina specialist can help with anti-VEGF injections or panretinal photocoagulation.

C. Intraoperative approaches
   1. Good gonioscopic view
   2. Consider mild reverse Trendelenburg positioning.
   3. Point angle instruments anteriorly during initial approach to trabecular meshwork.
   4. Be vigilant for red flags during surgery.
      a. Resistance to advancement of device
      b. Device diving posteriorly
      c. Patient discomfort during device advancement
   5. Prevent anterior chamber collapse when removing irrigation/aspiration handpiece.
      a. Prehydrate main corneal incision.
      b. Inject BSS through paracentesis while removing irrigation/aspiration handpiece.
   6. If there is hyphema, keep eye pressurized and wait. Can be done with BSS, air bubble, or viscoelastic

II. Recovering After Hyphema
   A. Expectant management
      1. Activity restrictions
   2. Head elevation
   3. IOP and inflammation control
   B. Anterior chamber washout
   C. Anterior chamber air/gas injection
   D. Argon laser to focal bleeding source

III. Recovering After Cyclodialysis Cleft
   A. For small clefts, medical management may be appropriate.
      1. Rapid steroid taper, atropine, laser to promote cleft closure
      2. Counsel about postoperative IOP spike, especially if cleft has been present for weeks or months.
      3. Consider surgical repair if cleft does not close within 3 months.
B. For large clefts, surgical repair likely is required.
   1. Ab interno approaches
      a. 9-0/10-0 polypropylene on double-armed STC-6 straight needle to reapproximate tissue
      b. Can dock with 25/27-gauge needle to help with externalizing needle
      c. Sewing machine technique¹
   2. Ab externo approaches
      a. Create full-thickness scleral flap and suture uveal tissue directly to sclera.
      b. Transconjunctival cryotherapy

Reference and Selected Readings
How to Not Let Those Flat Chambers Get You Down

Julia A Rosdahl MD PhD

I. Burnout and Resilience in Medicine/Ophthalmology/Glaucoma
   A. In 2022, self-reported burnout was found in 38% of ophthalmologists in the United States.
      1. 36% of glaucoma specialists (44 of the 121 who filled out the survey)
      2. There was more burnout in women compared with men, and more in academic compared with private groups.
      3. Burnout was associated with low work control, low time for documentation, and misalignment with leaders.
   B. Resilience levels for physicians is high compared to the general working population in the United States.
      1. Ophthalmology is in the most resilient quartile.
      2. Resilience was inversely related to burnout symptoms.
      3. Burnout still occurred even in the most resilient physicians.
      4. Organizational solutions are needed to reduce burnout and promote well-being.

II. ABC’s for Self-Care
   Creating a culture of wellness in medicine/ophthalmology/glaucoma
   A. Mindfulness
   B. Boundaries
   C. Community/connection

III. Take Action
   A. SMART goal
      1. Choose area of focus and set a SMART goal.
      2. Specific, Measurable, Achievable, Relevant/Realistic, Time-bound
   B. WISER
      1. Randomized controlled trial showed that web-based wellness intervention reduced burnout in health care workers.
   C. Organizational considerations
      1. Target inefficiencies in work environments.
      2. Cultivate community at work.
      3. Consider unintended consequences of incentives.
      4. Promote work-life integration.

Selected Readings
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<thead>
<tr>
<th>Code</th>
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</thead>
</table>
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LayerBio: C
Leica Microsystems: C
Life Long Vision: C,SO
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Glaukos Corp.: US
Santen, Inc.: S
Théa: C

Robert T Chang MD
1800 Contacts: C
Alcon Laboratories, Inc.: C
Belkin Laser: C
Genentech: C,SO
Ocular Therapeutix: C
Optomed Corp.: C
Sight Sciences, Inc.: C
Smartlens: C
Verana Health: C
XP Health: C

Ta Chen Peter Chang MD
None

Teresa C Chen MD
Alcon Laboratories, Inc.: S

Anna T Do MD
AbbVie: L

Beth Edmunds MD PhD
None

Brian A Francis MD
Aerie Pharmaceuticals, Inc.: C,L
Alcon Laboratories, Inc.: C,SO
Allergan, Inc.: C,SO
Beaver-Visitec International, Inc.: C
Greybug: C
Iridex: C
iStar Medical: C
MicroSurgical Technology: C
New World Medical, Inc.: S
Théa: C

Steven J Gedde MD
None

Christopher A Girkin MD
Amylin: SO
Heidelberg Engineering: C,SO
Topcon Medical Systems, Inc.: C,SO

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