Cornea 2014
Restocking the Toolbox: Concepts and Techniques for the Toughest Jobs

Program Directors
William Barry Lee MD, Elmer Y Tu MD and Stephen C Kaufman MD PhD

In conjunction with the Cornea Society
McCormick Place
Chicago, Illinois
Saturday, Oct. 18, 2014

Presented by:
The American Academy of Ophthalmology
2014 Cornea Subspecialty Day Planning Group

On behalf of the American Academy of Ophthalmology and the Cornea Society, it is our pleasure to welcome you to Chicago and Cornea 2014: Restocking the Toolbox: Concepts and Techniques for the Toughest Jobs.
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CME Credit

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

2014 Cornea Subspecialty Day Meeting Learning Objectives
Upon completion of this activity, participants should be able to:
- Describe the evidence-based approach to managing medical diseases of the cornea and ocular surface, including inflammatory, infectious, and degenerative conditions
- Compare the advantages and disadvantages of recently popularized techniques of corneal transplantation (Descemet membrane endothelial keratoplasty, deep anterior lamellar keratoplasty, and keratoprosthesis) and understand strategies to incorporate these techniques into their practices
- Identify patient groups for whom collagen crosslinking alone or in combination with additional procedures is a promising treatment and understand the advantages and disadvantages of different crosslinking techniques
- Identify indications and surgical techniques for corneal disease in the presence of cataracts, glaucoma, ocular surface disease, and complex anterior segment anatomy

2014 Cornea Subspecialty Day Meeting Target Audience
The intended audience for this program is cornea surgeons, comprehensive ophthalmologists with an interest in anterior segment and allied health personnel who are performing or assisting with cornea surgery.

2014 Cornea Subspecialty Day CME Credit
The American Academy of Ophthalmology is accredited by the Accreditation Council for Continuing Medical Education to provide CME for physicians.

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

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

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

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

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

Attendance Verification for CME Reporting
Before processing your requests for CME credit, the Academy must verify your attendance at Subspecialty Day and/or at AAO 2014. In order to be verified for CME or auditing purposes, you must either:
- Register in advance, receive materials in the mail and turn in the Final Program and/or Subspecialty Day Syllabus exchange voucher(s) onsite;
- Register in advance and pick up your badge onsite if materials did not arrive before you traveled to the meeting; or
- Register onsite.

CME Credit Reporting
South, Level 2.5; Academy Resource Center, Booth 508
Attendees whose attendance has been verified (see above) at AAO 2014 can claim their CME credit online during the meeting. Registrants will receive an email during the meeting with the link and instructions on how to claim credit.

Onsite, you may report credits earned during Subspecialty Day and/or AAO 2014 at the CME Credit Reporting booth.
Academy Members: The CME credit reporting receipt is not a CME transcript. CME transcripts that include AAO 2014 credits entered onsite will be available to Academy members on the Academy’s website beginning Nov. 13, 2014.

NOTE: CME credits must be reported by Jan. 15, 2015. After AAO 2014, credits can be claimed at www.aao.org.

The Academy transcript cannot list individual course attendance. It will list only the overall credits spent in educational activities at Subspecialty Day and/or AAO 2014.

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

Proof of Attendance

The following types of attendance verification will be available during AAO 2014 and Subspecialty Day for those who need it for reimbursement or hospital privileges, or for nonmembers who need it to report CME credit:

- CME credit reporting/proof-of-attendance letters
- Onsite Registration Form
- Instruction Course Verification Form

Visit the Academy’s website for detailed CME reporting information.
Faculty

**Esen K Akpek MD**
Baltimore, MD
Professor of Ophthalmology and Rheumatology
Johns Hopkins University School of Medicine
Director, Ocular Surface Diseases and Dry Eye Clinic
Division of Cornea and External Diseases
The Wilmer Eye Institute

**Reay H Brown MD**
Atlanta, GA
Founder, Atlanta Ophthalmology Associates

**Vincent P de Luise MD FACS**
Woodbury, CT
Assistant Clinical Professor of Ophthalmology
Yale University School of Medicine
Adjunct Clinical Assistant Professor of Ophthalmology
Weill Cornell Medical College

**Juan F Batlle MD**
Santo Domingo, Dominican Republic
Chief Ophthalmology
Elias Santana Hospital
President of Laser Center, Chairman of Blindness Prevention
International Agency for the Prevention of Blindness VISION 2020
Laser Center

**Clara C Chan MD**
Toronto, ON, Canada
Lecturer, Department of Ophthalmology
University of Toronto

**Minas T Coroneo MD MS**
Randwick, NSW, Australia
Professor of Ophthalmology
University of New South Wales

**Deepinder K Dhaliwal MD**
Pittsburgh, PA
Associate Professor of Ophthalmology
University of Pittsburgh School of Medicine
Director of Cornea and Refractive Surgery
UPMC Eye Center
University of Pittsburgh
William J Dupps MD PhD
Bay Village, OH
Staff in Ophthalmology, Biomedical Engineering and Transplantation
Cleveland Clinic Cole Eye Institute
Adjunct Associate Professor
Biomedical Engineering
Case Western Reserve University

Jose Gomes MD
São Paulo, SP, Brazil
Professor of Ophthalmology and Director of the Advanced Ocular Surface Center
Federal University of São Paulo

Sadeer B Hannush MD
Langhorne, PA
Attending Surgeon
Cornea Service, Wills Eye Hospital
Department of Ophthalmology
Jefferson Medical College

Anat Galor MD
Miami, FL
Associate Professor of Ophthalmology
Bascom Palmer Eye Institute, University of Miami
Staff Physician
Miami VAMC

Mark S Gorovoy MD
Fort Myers, FL
Founder, Gorovoy MD Eye Specialists

Ana Luisa Höfling-Lima MD MBA
São Paulo, SP, Brazil
Head Professor of Ophthalmology
Federal University of São Paulo
President, Pan American Association of Ophthalmology

José L Güell MD PhD
Barcelona, Spain
Associate Professor of Ophthalmology
Universidad Autónoma de Barcelona
Director of Cornea and Refractive Surgery Unit
Instituto de Microcirugía Ocular de Barcelona

Edward J Holland MD
Union, KY
Director, Cornea Services
Cincinnati Eye Institute
Clinical Professor of Ophthalmology
University of Cincinnati

Dasa Gangadhar MD
Wichita, KS
Partner, Director of Corneal Services
Grene Vision Group
Clinical Assistant Professor of Surgery
University of Kansas School of Medicine
Bennie H Jeng MD  
Baltimore, MD  
Professor of Ophthalmology  
University of California, San Francisco

Carol L Karp MD  
Miami, FL  
Professor of Ophthalmology  
Bascom Palmer Eye Institute  
University of Miami Miller School of Medicine

Bennie H Jeng MD  
Baltimore, MD  
Professor of Ophthalmology  
University of California, San Francisco

Carol L Karp MD  
Miami, FL  
Professor of Ophthalmology  
Bascom Palmer Eye Institute  
University of Miami Miller School of Medicine

Friedrich E Kruse MD  
Erlangen, Germany  
Professor of Ophthalmology  
University of Erlangen

William Barry Lee MD  
Atlanta, GA  
Attending Surgeon  
Piedmont Hospital / Eye Consultants of Atlanta  
Co-Medical Director  
Georgia Eye Bank

Friedrich E Kruse MD  
Erlangen, Germany  
Professor of Ophthalmology  
University of Erlangen

William Barry Lee MD  
Atlanta, GA  
Attending Surgeon  
Piedmont Hospital / Eye Consultants of Atlanta  
Co-Medical Director  
Georgia Eye Bank

Thomas M Lietman MD  
San Francisco, CA

Jennifer Y Li MD  
Sacramento, CA

Marian Sue Macsai-Kaplan MD  
Glenview, IL  
Professor of Ophthalmology  
University of Chicago Pritzker School of Medicine  
Chief of Ophthalmology  
NorthShore University HealthSystem

Francis S Mah MD  
La Jolla, CA  
Director, Corneal and External Disease  
Scripps Clinic  
Codirector, Refractive Surgery  
Scripps Clinic
Boris Malyugin MD PhD
Moscow, Russian Federation
Professor of Ophthalmology
S Fyodorov Eye Microsurgery Complex
State Institution

Shahzad I Mian MD
Ann Arbor, MI
Associate Professor
University of Michigan

Stephen C Pflugfelder MD
Houston, TX
Professor of Ophthalmology
Baylor College of Medicine

Mark J Mannis MD
Sacramento, CA
Professor of Ophthalmology
Department of Ophthalmology & Vision Science
University of California, Davis

Ramana S Moorthy MD
Indianapolis, IN
Clinical Associate Professor of Ophthalmology
Indiana University School of Medicine
CEO and Founding Partner
Associated Vitreoretinal and Uveitis Consultants

Francis W Price Jr MD
Indianapolis, IN
Medical Director
Price Vision Group
President of the Board
Cornea Research Foundation of America

Stephanie Jones Marioneaux MD
Chesapeake, VA
American Academy of Ophthalmology
Assistant Professor of Ophthalmology
Eastern Medical of Virginia

Venkatesh Prajna
Namperumalsamy MBBS
Madurai, Tamilnadu, India
Academic Director
Aravind Eye Hospital, India

Shigeto Shimmura MD
Tokyo, Japan
Associate Professor of Ophthalmology
Keio University School of Medicine
Christopher E Starr MD
New York, NY
Associate Professor of Ophthalmology
and Director of Refractive Surgery
Weill Cornell Medical College

Donald Tan MD FRCS FRCOphth
Singapore, Singapore
Medical Director
Singapore National Eye Centre
Professor of Ophthalmology
Department of Ophthalmology
National University of Singapore

Radhika Tandon MD
New Delhi, India
Professor of Ophthalmology and Officer in Charge, National Eye Bank
Dr Rajendra Prasad Centre for Ophthalmic Sciences
All India Institute of Medical Sciences (AIIMS)

Joseph Tauber MD
Kansas City, MO
Medical Director
Tauber Eye Center

Elmer Y Tu MD
Glenview, IL
Professor of Clinical Ophthalmology
University of Illinois Eye and Ear Infirmary
## Cornea 2014

**Restocking the Toolbox: Concepts and Techniques for the Toughest Jobs**

In conjunction with the Cornea Society

**SATURDAY, OCT. 18**

<table>
<thead>
<tr>
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<th>Event</th>
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<tr>
<td>7:00 AM</td>
<td>CONTINENTAL BREAKFAST</td>
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<td>Welcome and Introductions</td>
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<td>8:02 AM</td>
<td>Introduction and Self-assessment</td>
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<tr>
<td>8:04 AM</td>
<td>Point of Care Dry Eye Tests: What Are They Worth?</td>
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<td>How to Manage the Problem Dry Eye Patient</td>
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<td>Shedding Blood for a Tear: Blood Products for Dry Eye</td>
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<td>Pain Without Stain</td>
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<td>8:44 AM</td>
<td>Persistent Epithelial Defects: Current Management Strategies</td>
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<td>8:54 AM</td>
<td>Anti-angiogenesis Therapy</td>
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<td>9:04 AM</td>
<td>Presentation of Case</td>
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<td>Discussion</td>
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<td>Conclusion and Self-assessment</td>
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<tr>
<td>9:25 AM</td>
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<tr>
<td>9:27 AM</td>
<td>Peripheral Ulcerative Keratitis—Herpes Simplex Virus + Autoimmune</td>
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<td>9:37 AM</td>
<td>Scleritis and Episcleritis: Update on Diagnosis and Management</td>
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<td>Fungal, and TB—Care With Steroids</td>
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<td>Surgical Techniques of Peripheral Corneal Disease</td>
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<td>Modern Immunosuppressant Agents for Corneoscleral Disease</td>
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<td>Conclusion and Self-assessment</td>
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### Section I: Smoothing Out the Rough Surfaces

Moderator: William Barry Lee MD*

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<td>Marian Sue Macsai-Kaplan MD*</td>
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### Section II: Inflammatory Disorders—The Old and New

Moderator: Stephen C Kaufman MD PhD*

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<td>Introduction and Self-assessment</td>
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<td>Radhika Tandon MD</td>
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<td>Donald Tan MD FRCS FRCOphth*</td>
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<td>Modern Immunosuppressant Agents for Corneoscleral Disease</td>
<td>Ramana S Moorthy MD</td>
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<td>Stephen C Kaufman MD PhD*</td>
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* Indicates that the presenter has financial interest.
No asterisk indicates that the presenter has no financial interest.
### Section III: Spotlight Session—Cornea Controversies

**Moderator:** Elmer Y Tu MD  

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<td>Perioperative Antibiotics: Pretreating with Antibiotics/Intracameral Antibiotics</td>
<td>Francis S Mah MD*</td>
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<td>11:23 AM</td>
<td>Steroids in Ulcers</td>
<td>Thomas M Lietman MD</td>
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<td>11:31 AM</td>
<td>Fungal Keratitis Management: Best Drugs; Mycotic Ulcer Trial Review</td>
<td>Venkatesh Prajna</td>
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<td>11:47 AM</td>
<td>Which Is Better: Epi-on or Epi-off Corneal Crosslinking?</td>
<td>William J Dupps MD PhD*</td>
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<td>11:55 AM</td>
<td>Advanced Combination Crosslinking</td>
<td>José L Güell MD PhD*</td>
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<td>12:03 PM</td>
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### Section IV: Combination Cornea—My Top 5 Tips

**Moderator:** Stephen C Kaufman MD PhD*  

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<td>Introduction and Self-assessment</td>
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<td>1:39 PM</td>
<td>Combined Glaucoma and Cataract Surgery: Microinvasive Glaucoma Surgery—Top 5 Pearls</td>
<td>Reay H Brown MD*</td>
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<td>1:48 PM</td>
<td>Five Times You Really Need a Femtosecond Laser</td>
<td>Juan F Batlle MD*</td>
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<td>Cataract Surgery in Corneal Disease: Five Pearls</td>
<td>Dasa Gangadhar MD</td>
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<td>Cataract Case With Zonular Insufficiency</td>
<td>Boris Maluygin MD PhD*</td>
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<td>Dislocated IOL Case</td>
<td>Sadeer B Hannush MD</td>
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<td>Toric IOL in Cornea Disease</td>
<td>Christopher E Starr MD*</td>
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<td>Stephen C Kaufman MD PhD*</td>
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### Section V: Walking the Tightrope—Corneal Surgery Quandaries

**Moderator:** William Barry Lee MD*  

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<td>Introduction and Self-assessment</td>
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<td>Endothelial Disease: Why Descemet-Stripping Automated Endothelial Keratoplasty Is Better</td>
<td>Mark S Gorovoy MD</td>
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<td>Endothelial Disease: Why Descemet Membrane Endothelial Keratoplasty Is Better</td>
<td>Friedrich E Kruse MD*</td>
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<td>Anterior Corneal Disease: Why Penetrating Keratoplasty Is Better</td>
<td>Mark J Mannis MD</td>
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<td>3:43 PM</td>
<td>Anterior Corneal Disease: Why Deep Anterior Lamellar Keratoplasty Is Better</td>
<td>Shigeto Shimmura MD*</td>
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</table>

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### 3:53 PM
**Limbal Stem Cell Disease: When Is Ocular Surface Transplantation Better?**
Edward J Holland MD*

### 4:03 PM
**Limbal Stem Cell Disease: When Is Keratoprosthesis Better?**
Clara C Chan MD*

### 4:13 PM
Discussion

### 4:25 PM
Conclusion and Self-assessment
William Barry Lee MD*

### Section VI: The Best Tool From the Toolbox? Challenging Cases
Moderator: Elmer Y Tu MD

<table>
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<td>Elmer Y Tu MD</td>
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<td>4:29 PM</td>
<td>Case #1</td>
<td>Shahzad I Mian MD*</td>
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<td>Case #2</td>
<td>Carol L Karp MD</td>
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<td>Case #3</td>
<td>Jennifer Y Li MD</td>
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<td>Case #4</td>
<td>José Gomes MD*</td>
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<td>Closing Remarks</td>
<td>William Barry Lee MD*</td>
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<td>Stephen C Kaufman MD PhD*</td>
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</table>
Point of Care Dry Eye Tests: What Are They Worth?

Joseph Tauber MD

I. What should we evaluate in a patient complaining of dry eye?
   A. History
   B. General physical examination
   C. Eyelid closure and anatomy
   D. Eyelid margin, meibomian gland orifices and secretions, follicles
   E. Tear film: volume, gross physical characteristics
   F. Tear film chemical characteristics
   G. Tear production, clearance
   H. Corneal epithelial appearance, function, healing response
   I. Corneal topographic abnormalities

II. Screening for Sjögren’s Syndrome
   A. Historic methods: clinical, laboratory, biopsy
   B. Currently available methods
   C. Interpretation of test results
   D. Correlations with other measures of ocular health
   E. Correlation with symptoms
   F. Predictive value
   G. Clinical use of testing

III. Tear Osmolarity Testing
   A. Historic methods
   B. Currently available methods
   C. Interpretation of test results
   D. Correlations with other measures of ocular surface disease
   E. Correlation with symptoms
   F. Predictive value
   G. Clinical use of testing

IV. Tear MMP-9 Testing
   A. Currently available methods
   B. Interpretation of test results
   C. Correlations with other measures of ocular health
   D. Correlation with symptoms
   E. Predictive value
   F. Clinical use of testing

V. Tear Lactoferrin Testing
   A. Historic methods
   B. Currently available methods
   C. Interpretation of test results
   D. Correlations with other measures of ocular health
   E. Correlation with symptoms
   F. Predictive value
   G. Clinical use of testing

VI. Tear Film Stability
   A. Clinical methods: tear breakup time (TBUT)
   B. Currently available methods: noninvasive tear break-up time (NITBUT), Tear film Stability Analysis System
   C. Interpretation of test results
   D. Clinical use of testing

VII. Tear Lipid Layer Analysis
   A. Historic methods
   B. Currently available methods: K5 Keratograph, Lipiview
   C. Interpretation of test results
   D. Clinical use of testing

VIII. Summary: Potential Benefits of Testing
   A. Improved accuracy of diagnosis
   B. Improved communication with patients
   C. Metric for evaluation of therapeutic intervention
   D. Practice development
How to Manage the Problem Dry Eye Patient

Deepinder K Dhaliwal MD

We all commonly face these difficult patients in our practices. It is important to have strategies to tackle these challenging patients, and I would like to share with you some things that help me in my practice.

History
First, get a good history. Don’t ask, “What seems to the problem?” because you will get a litany of complaints. Instead ask, “What are the top 3 things that bother you about your eyes?” Then address those things.

Important questions to ask:
- Are symptoms worse in the morning?
  - If yes, then think lagophthalmos or floppy lid syndrome. Typical dry eye patients have worse symptoms later in the day.
- Do you sleep with a fan on?
  - This often exacerbates symptoms in patients with lagophthalmos. Instruct the patient to improve their sleeping environment by eliminating any drafts on their face, or by wearing night goggles.
- Does anything make you feel better (even for a minute)?
  - Place a drop of artificial tear in the patient’s eye and assess response. Then place a drop of topical anesthetic; if nothing helps, you will likely not help them with standard treatment.
- Do you have any mucus discharge?
  - Mucus fishing syndrome is underdiagnosed. It is very important to ask the patient to demonstrate how they remove the mucus from their eyes. I have been shocked by what patients have shown me. Also, mucus can be a sign of associated allergy.

Testing
- Tear osmolarity
- Zone quick, Schirmer
- Corneal sensation

Exam
The exam actually started while you were taking the history. Note blink rate, amount of eyelid closure per blink, eye rubbing, etc. Next perform a good external exam. Pull lower lids down, have patient look up. Any chronic conjunctivitis can be diagnosed with this view. Lower lid snap should also be assessed. Pull upper lids up and have patient look down. Floppy lid syndrome and SLK can be diagnosed at this time. Then have patients look left and right.

Slitlamp Exam
- To stain the ocular surface, use a fluorescein strip, not Fluress. Assess staining of conjunctiva, cornea, and lid margin.
- Assess tear film break-up time.
- Press on lid margins to assess degree of meibomian gland inspissation.
- Look for Demodex.
- Watch patient blink and more closely assess lid closure.

Treatment
Depending on findings, the following treatment options can be considered:
- Topical steroids
- Restasis
- Preservative-free tears: Not q 1 hour, to avoid “dish-pan eyes”
- Autologous serum
- Lid hygiene: LipiFlow, Meiboflow, rice in a sock
- Scrape lid margins
- Oral omega 3, GLA
- Low-dose doxycycline
- Tea tree oil if Demodex
- No plugs until inflammation improves
- Treat associated allergy “pollen vortex”
- Acupuncture
- Topical testosterone cream

Patient Discussion
- Empathize, give hope. Stress that we are striving for a long-term solution, and that results will not be seen overnight.
- Use analogies: Blepharitis/chronic dry eye is like dandruff. You can control it, but it really never goes away.
Shedding Blood for a Tear: Blood Products for Dry Eye

Minas T Coroneo MD MS
Pain Without Stain

Stephen C Pflugfelder MD

I. Symptomatic Patients With Minimal or No Signs
   A. Symptoms typical of dry eye (burning, scratching, photophobia)
   B. Often exquisitely sensitive to air drafts
   C. Report minimal or no relief from conventional dry eye treatments
II. Patients may have rapid tear breakup time (TBUT) as only sign.
III. Disconnect between signs and symptoms may be discounted or dismissed.
IV. Patients are often depressed and feel they are less productive.
V. Pain without stain suggests neuropathic pain, also termed “keratoneuralgia.”
   A. Cornea is vulnerable to developing neuropathic pain.
      1. Most exposed mucosal tissue in the body
      2. Susceptible to environmental stress/trauma
      3. Has the highest density of sensory receptors (nociceptors) of any tissue in the body
VI. Diagnosis is primarily clinical.
   A. Pain symptoms are out of proportion to clinical signs.
   B. Persistent pain after topical anesthetics suggests central sensitization.
   C. Confocal microscopy may show abnormal nerves.
      1. Beaded nerves in the sub-basal plexus
      2. Decreased number and increased tortuosity of sub-basal nerves
      3. Short nerves lacking connections between nerve bundles
      4. Excessive sprouting
      5. Dendritic cell infiltration
      6. History of poor response to conventional dry eye therapy
VII. Therapy
   A. Conventional dry eye therapy often ineffective
   B. Consider prosthetic replacement of the ocular surface ecosystem (PROSE), but not as effective as conventional dry eye and patients may complain of lens awareness
   C. Autologous serum/plasma; may promote normal nerve regeneration
   D. Gabapentin/pregabalin
   E. Counseling to cope with pain

Selected Readings


Persistent Epithelial Defects: Current Management Strategies

Bennie H Jeng MD

Introduction

In the presence of certain risk factors, including corneal hypoperfusion, diabetic keratopathy, limbal stem cell deficiency, dry eye disease, exposure keratopathy, and neurotrophic keratopathy from herpetic infections or previous corneal transplantation, epithelial defects can persist beyond the usual treatment period despite standard therapies. A persistent epithelial defect (PED) can be loosely defined as an epithelial defect that has not healed in the expected period of time. However, in accordance with the literature, when a defect has been treated for approximately 2 weeks without resolution, the cornea is said to have a PED.

PEDs have been reported to occur in 16.4% of eyes after penetrating keratoplasty and in 22.8% of eyes after corneal epithelial debridement during diabetic vitrectomy surgery. Given that improvements in techniques of vitrectomy surgery have dramatically reduced surgical time, the need to debride the corneal epithelium has decreased, and the number of eyes developing PED in this situation has decreased as well. However, the rates of PED occurring after other events, such as ocular surface burns, post-photorapeutic keratotomy, and postinfectious keratitis, are unknown. A conservative estimate may place the figure for the number of PEDs in the United States annually at 40,000.

Potential Complications

Although PED is uncommon, management of patients with PED can be quite challenging and requires intensive and sometimes extensive follow-up to ensure resolution. Complications from PED include infection (usually bacterial, but it can be fungal in etiology), corneal melting, and perforation. Less commonly thought of, but just as important, is the development of subepithelial haze and scarring from long-standing defects that can ultimately limit final visual outcome. Evidence in the literature suggests that the longer an epithelial defect is left open, the longer it will take to heal, and therefore the higher the chance of complications. As such, it is advisable to treat PED aggressively, and potentially even earlier in the course of the disease process if it is anticipated that difficulty with healing may be the case.

Etiologies

There are numerous possible etiologies for PED, and the more common ones include dry eye, exposure keratopathy, diabetic keratopathy, herpetic keratopathy, neurotrophic keratopathy (for example, after keratoplasty or keratorefractive surgery), and limbal stem cell deficiency (for example, after chemical burns). It is imperative to identify the possible causative etiologies for PED because for some of these situations, treatment of the PED should be first aimed at the underlying cause; the definitive management of PED secondary to exposure keratopathy from thyroid eye disease, for example, may be vastly different from that for a patient with limbal stem cell deficiency (LSCD) from an alkali burn. In the former condition, a tarsorrhaphy or even orbital decompression surgery may be warranted, whereas in the latter condition, limbal stem cell transplantation or Boston keratoprosthesis implantation may be the indicated treatment. In eyes with herpetic keratopathy, sometimes a PED is present because inflammation or persistent viral infection persists, precluding closure of the epithelial defect. Treatment targeted toward the inflammation (with corticosteroids) or toward the persistent viral infection (with antivirals) can sometimes cure the PED. A careful examination of the eyelids, tear film, and ocular surface are required in order to make the correct diagnosis.

Medical management strategies

- Aggressive lubrication
- Discontinuation of medications
- Punctual occlusion
- Bandage soft contact lens placement
- Pressure patching
- Autologous serum
- Scleral contact lenses

Surgical management strategies

- Epithelial debridement
- Tarsorrhaphy
- Amniotic membrane grafting
- Conjunctival flap
- Limbal stem cell grafting
- Boston keratoprosthesis

Experimental therapies

- Novel compounds
- Mesenchymal stem cells
- Other whole blood-derived products

Conclusions

Managing the PED can be both an arduous task for the ophthalmologist and a burden on the patient. Conventional therapies are often times ineffective, and this can result in complications such as infection and perforation. Having a PED also requires frequent clinic follow-up, at a cost to society and worker productivity. As one study has shown that the length of time a defect is left open and unhealed is directly proportional to the time it will take for the defect to be fully repaired, it can be argued for prophylactically managing patients at high risk for developing PED: it may be prudent to be more aggressive at the onset of epithelial compromise in certain patient populations. When standard medical therapies fail, there are several surgical options that exist for treatment of PED. In the event that even surgical treatment fails, there are other more experimental modalities that can be employed. Such alternative treatment modalities have been suggested to be beneficial and efficacious in early studies. Other promising newer therapies are under investigation, and hopefully these will add to our armamentarium of options for treating PED in the very near future.
References


Anti-angiogenesis Therapy and the Cornea/Ocular Surface

Anat Galor MD

I. What are medication options?
   A. Bevacizumab
      1. Mechanism of action
      2. Cost
   B. Ranibizumab
      1. Mechanism of action
      2. Cost
   C. Aflibercept
      1. Mechanism of action
      2. Cost

II. What are the indications?
   A. Corneal neovascularization
      1. Review of data: Does therapy lead to regression of vessels?
         Several clinical studies. Overall, a significant reduction of corneal neovascularization seen in humans.
      2. Review of data: Does therapy improve clinical outcomes?
         No conclusive data
   B. Pterygium
      1. Anti-angiogenesis therapy as adjuvant therapy at time of surgical removal
         a. Review of data: Does it improve clinical outcomes?
      b. No conclusive data
      2. Anti-angiogenesis therapy as primary therapy for early recurrent pterygium
         Review of data: Does it improve clinical outcomes?
         a. Transiently improves hyperemia
         b. Mixed results regarding reduction in neovascular area
   C. Are there safety concerns?
      1. Epitheliopathy
      2. Corneal melt after penetrating keratoplasty
      3. Wound dehiscence after pterygium removal with conjunctival autograft
Presentation of Case

Marian Sue Macsai-Kaplan MD

NOTES
Peripheral Ulcerative Keratitis—Herpes Simplex Virus + Autoimmune

Radhika Tandon MD

Overview

Peripheral ulcerative keratitis is a complex clinical entity of varied etiology that epitomizes the link between the eye and the rest of the human body. Though considered a rare disease, with a reported incidence of 3 cases per million population per year in a localized region in England, it is nevertheless an illness that draws attention due to its propensity for relentless progression and sight-threatening complications affecting vulnerable populations.

Etiopathogenesis

Peripheral ulcerative keratitis is essentially an inflammatory disorder involving the peripheral cornea adjacent to the limbus that spreads circumferentially along the limbus and that also can extend centripetally toward the central cornea. The illness is triggered by an immunological reaction involving the activation of the complement system by immune complexes leading to the chemotactic migration of inflammatory cells—predominantly neutrophils and macrophages. The inflammatory cellular infiltration is accompanied by the release of keratolytic enzymes such as protease and collagenase, and inflammatory cytokines induce the stromal keratocytes to release matrix metalloproteases, which have the combined effect of leading to corneal stromal destruction and breakdown of its normal architecture with loss of transparency and strength, eventually resulting in severe thinning and even perforation.

Autoimmune diseases are by far the most common associated with peripheral ulcerative keratitis, of which rheumatoid arthritis is the most frequent. However, infectious agents such as herpes virus and staphylococcal infectious are also responsible, and idiopathic Mooren ulcers with no identifiable causative agent or systemic disease are well established.

Diagnosis

Patients with peripheral ulcerative keratitis may present with a variety of symptoms and signs. Pain, redness, foreign body sensation, watering and blurring of vision are commonly seen. Patients may or may not give a history of past or current active systemic disease, and it is important to carefully elicit these details by direct questioning. In the active stage of the disease there are signs of inflammation, with peripheral ulceration represented by an epithelial defect with stromal lysis (see Figure 1). Differential diagnosis includes Terrien marginal degeneration, superior limbic keratoconjunctivitis, phtyctenular keratoconjunctivitis, acne rosacea-associated keratitis, and staphylococcal blepharitis with exotoxin-induced marginal keratitis.

Investigations

Based on history and examination, the diagnosis is established with the help of investigations to determine underlying etiology, systemic associations, and extent of ocular involvement with prognosis.

Ophthalmic investigations

- Tear film analysis
- Corneal scrapings for microbiological screening by culture and microscopy
- Corneal topography
- Ultrasonography for posterior segment and sclera involvement

Systemic evaluation for underlying autoimmune diseases or infectious agents may include:

- Complete blood count
- Complete biochemical analysis for metabolic profile including blood glucose levels and kidney and liver function
- Urinalysis with microscopy
- Antinuclear antibody
- Anti-neutrophil cytoplasmic antibody
- Rheumatoid factor
- Anti-cyclic citrullinated peptide
- Rapid plasma reagin
- Fluorescent treponemal antibody (FTA-Abs)
- Chest x-ray
- Purified protein derivative
- X-ray of sacroiliac joints
CT scan of sinuses
Screening for systemic infection with hepatitis B virus and hepatitis C virus
Screening for tuberculosis
Ultrasound of the eye

Treatment

Medical
Topical preservative-free lubricants help by washing away inflammatory mediators and diluting their toxic effect in the tear film, apart from promoting epithelialization. Application of cyanoacrylate or fibrin glue on the ulcer bed with a bandage contact lens helps by limiting the influx of inflammatory cells from the tear film and saving off further thinning in case of impending perforation. Topical broad spectrum antibiotics are used to fend off secondary bacterial superinfection. Judicious use of topical steroid eye drops 3-4 times daily for brief durations is recommended to control inflammation without aggravating the corneal melting response.

Systemic corticosteroids are considered the mainstay of treatment, with adjunctive prescription of steroid-sparing agents such as cyclosporine and other immunosuppressive agents as and when required.18,19,22-25

Surgical
 Conjunctival resection and amniotic membrane inlay and onlay grafts can be helpful if medications fail to halt the progressive ulceration and thinning. Impending perforation or actual perforation are indications for further surgical measures—including patch grafts, which can be challenging to perform as the defects are often large and crescent shaped, with irregular margins, and a lot of skill is involved in manually fashioning a best-fit donor tissue. Depending on the nature of the defect, the surgeon can use a lamellar or full-thickness graft to achieve a best possible anatomic restoration, followed by a lamellar or full-thickness keratoplasty for optical and visual rehabilitation after the inflammation settles.20,21

References
Scleritis and Episcleritis: Update on Diagnosis and Management
The Good, The Bad, The Ugly: How to Solve the Conundrums

Vincent P de Luise MD FACS

I. Episcleritis
   A. Definition
   B. Clinical manifestations and etiology
   C. Pathophysiology
   D. Nosology
      1. Nodular episcleritis
      2. Diffuse episcleritis
      3. “Pingueculitis”
   E. Treatment strategies
   F. Clinical examples

II. Scleritis
   A. Definition
   B. Clinical manifestations and etiology
   C. Pathophysiology
   D. Nosology
      1. Anterior scleritis (98%)
         a. Non-necrotizing anterior scleritis
            i. anterior diffuse scleritis
            ii. anterior nodular scleritis
         b. Necrotizing scleritis with inflammation
         c. Necrotizing scleritis without inflammation (“scleromalacia perforans”)
      2. Posterior scleritis (2%)

E. Laboratory and imaging studies
F. Treatment paradigms
   1. NSAIDs
   2. Oral vs. periocular corticosteroids
   3. Cytotoxic immunosuppressives (oral, IM, or IV: when and why)
   4. Immunomodulatory drugs / biologic agents
   5. Role of rheumatologist / immunologist
   6. Role of surgery
G. Clinical examples
Scleritis is a state of ocular inflammation with a wide spectrum of clinical presentations and etiologic factors. There are specific etiologies of scleritis, varying from idiopathic to autoimmune to infectious, with a variable disease severity and outcome. Infection is an important but rare cause of scleritis, occurring in about 5%–10% of all scleritis cases. However, due to the similarity of its presentation, infectious scleritis is often initially managed as autoimmune, potentially worsening its outcome. The overall visual outcome in infectious scleritis is generally worse than in its autoimmune counterparts, perhaps because of this delay in diagnosis or because of the aggressive nature of the infection.

Risk factors for infectious scleritis are typically surgery, most commonly pterygium surgery but also excisions of conjunctival neoplasms, cataract surgery, vitreoretinal surgeries, and glaucoma surgeries. The use of concomitant radiation or mitomycin C in some of those surgeries can also be a risk factor. Trauma, especially with introduction of organic material to the ocular surface or self-inoculation from a distant site on the body is also a significant inciting factor. In isolated cases, immunosuppression due to human immunodeficiency virus or chemotherapy may be a risk factor for spontaneous cases of infectious scleritis. Interestingly, Meyer et al reported an individual case of spontaneous infectious scleritis without any such prior history that revealed *Pseudomonas aeruginosa* resistant to fourth-generation fluoroquinolones, indicating the importance of considering infectious etiologies despite the lack of leading history.

The following are the most important etiological agents related with infectious scleritis: herpes (simplex and zoster), fungi (filamentous and yeast), *Acanthamoeba*, and bacteria (mycobacteria, Gram-positive cocci and Gram-negative bacilli).

### Clinical and Laboratory Diagnosis

The clinical diagnosis can vary, depending of the risk factors and the etiological agent. It is common to have a painful red eye that clinically is difficult to differentiate from the noninfectious cases. Ultrasound biomicroscopy can provide information for early detection of infectious scleritis that can be associated with retinal and choroidal detachments. OCT can also be used in those cases and allows the visualization of small vitreoid opacities and abnormal subretinal deposits.

A complete diagnostic workup is recommended of infectious etiologies and includes scleral scrapings and culture on blood and chocolate agar, brain-heart infusion and thioglycolate broth, non-nutrient agar with *E. coli* overlay, and Sabouraud dextrose agar. Diagnosis of viral etiologies like herpes-related scleritis can be confirmed by scleral biopsy and the use of immunofluorescence, as well as positive titers or signs of chronic herpetic infection like corneal hypoesthesia. Scleral scrapings in every case of suspected infectious scleritis are recommended for the purpose of diagnosis but also to debulk necrotic tissue and improve antimicrobial penetration.

### Herpes Scleritis

Herpes infections of the eye, both herpes simplex virus and varicella zoster virus, have predominantly been thought of in relation to the cornea and ocular adnexa, but they have also been reported in cases of infectious scleritis alone. Gonzalez-Gonzalez et al conducted a large retrospective study of patients with herpetic scleritis and found a predominance of middle-aged females with unilateral findings and moderate to severe pain. This disease can be acute, but chronic herpes-associated inflammation is also described. Herpetic scleritis is more likely to be of the diffuse anterior type than of the nodular or necrotizing type. Perilimbal devascularization with peripheral corneal thinning along with a significant amount of associated uveitis are findings associated with herpetic scleritis, with significantly greater vision loss noted in herpes than in idiopathic cases of scleritis. In general, herpes infections are not associated with posterior scleritis, vitreoretinal involvement, or scleromalacia perforans.

Herpetic scleritis typically responds quickly to acyclovir treatment, within 3–8 weeks, with the inflammation generally lasting from 5 to 32 months. The initial dosage of acyclovir used in these studies was 800 mg, 5 times daily, with most patients requiring a lower maintenance dose of acyclovir to prevent recurrence.

Previous treatment with immunosuppressive therapy can prolong and worsen herpetic scleral inflammation. Steroids if used have to be concomitant with oral antiviral drugs.

### Mycotic Scleritis

Fungal scleritis usually occurs as an exogenous infection. Occasionally it may result from hematogenous spread of systemic fungal disease, and unlike bacterial and viral scleritis, it has worse overall outcomes. Fungal scleritis is more commonly encountered in hot, humid climates, generally following surgical or accidental trauma. In cases of fungal scleritis, full-thickness corneal inflammation contiguous with scleral lesions is quite common. In many situations, patients with fungal scleritis progress despite treatment to develop rapidly progressive cataracts, serous retinal or choroidal detachments, phthisis, or endophthalmitis. Jain et al further report that *Aspergillus* and *Nocardia* are the most common fungal agents in infectious scleritis, but rare fungal agents can also be isolated from scleral samples.

Thus, it is evident that overall visual prognosis in fungal scleritis is generally poor, possibly because of delayed diagnosis, poor penetration of antifungals into avascular sclera, no availability of fungicidal agents, or the ability of organisms to persist in avascular scleral tissue for long periods of time without inciting inflammatory response, allowing progressive worsening.

### Bacterial Scleritis

Infectious scleritis due to bacteria contamination can be caused by exogenous contamination and *P. aeruginosa*, the most common agent. Infectious scleritis has to be treated with systemic and
topical ciprofloxacin, and even with the specific treatment the evolution can be very poor. *Mycobacterium chelonae*, abscessus, and tuberculosis can also be a cause of a severe bacterial scleritis.

**Acanthamoeba**

*Acanthamoeba* keratitis is a rare but potentially devastating ocular infection usually affecting healthy soft contact lens wearers. It is caused by a ubiquitous free-living protozoan that exists in the form of cysts or infective trophozoites. *Acanthamoeba* sclerokeratitis is an uncommon but severe complication that potentially can result in blindness. Mannis et al reported that 14% of *Acanthamoeba* keratitis cases have an associated scleritis.

The pathogenesis of scleritis is poorly understood and can be anterior, diffuse or nodular necrotizing. It has been already described as scleral invasion by *Acanthamoeba*, but an immune mediated response to dead and dying ameba within the limbal area is responsible for the most cases of sclerokeratitis.

The classical treatment with polyhexamethylene biguanide (PHMB) and topical propamidine (Brolene) for the cornea may not achieve therapeutic levels in the sclera.

Immunosuppression may be important for the cases that have no activity of *Acanthamoeba*. Systemic treatment with miltefosine or voriconazole may be considered. Cryotherapy can be used for the treatment of this disease in combination with antibiotics. Enucleation may be required.

**Infectious Scleritis: Treatment and Considerations**

Prevention of infectious scleritis is most important for patient outcome. The challenge for treatment is that traditional antibiotic regimens, both topical and systemic, remain inadequate due to the avascularity of the sclera and the dense structure of collagen fibers, limiting tissue penetration.

Avoiding overuse of cautery and adjunctive therapy during surgical procedures may spare the episcleral blood flow and allow better wound healing and resistance to infection. Most studies advocate the avoidance of bare sclera techniques that leave the ocular surface vulnerable to infection. The use of amniotic membrane over an area of debridement can be discouraged, suggesting that it provides maximum exposure for topical antibiotics and prevents the incubated microbes from staying at the site, with adequate re-epithelialization occurring shortly after debridement.

One large study of patients with infectious scleritis by Hodson et al employed various antimicrobial treatment regimens: 95% topical, 77% oral, and 11% intravitreal therapy. Despite a mean treatment duration of 50 days, medical therapy was adequate as the sole treatment in only 18% of patients, with most requiring surgical debridement.

Cryotherapy, lamellar or penetrating corneoscleral grafts, or removal of hardware in addition to intensive antibiotics improves overall outcomes in patients. Most significantly, it has been shown in multiple studies that the inflamed area is often found intraoperatively to be much larger than clinically judged by slitlamp exam, so careful surgical exploration has to be done. And a scleral patch graft has to be prepared.

The use of fascia lata grafts in combination with an amniotic membrane graft after surgical debridement for infectious scleritis was used successfully with *Pseudomonas* and with fungal scleritis. Fascia lata was thought to be ideal because of its biocompatibility in size and thickness as well as its good cosmetic appearance. Not unlike fascia lata grafts, tectonic scleral reinforcement with preserved pericardium and donor corneal tissue was used successfully in a patient with nocardial scleritis that progressed to scleral perforation and uveal prolapse.

A variety of medical modalities have been suggested as good adjunctive treatments to surgical debridement. These include systemic and topical antibiotics, subconjunctival injections of antibiotic at both ends of the scleral lesion, and wound irrigation with antibiotic solution 1 to 2 times a day followed by normal saline after improvement. Intraocular antibiotics should be used in all cases of endophthalmitis, and topical steroids should always be initiated only when antibiotics have been reliably used for several days.

**Selected Readings**


Surgical Techniques of Peripheral Corneal Disease

Donald Tan MD FRCS FRCOphth

I. Surgical Principles for Managing Peripheral Ulcerative Keratitis (PUK)

A. Maximal medical management before and after surgical intervention
   1. Infection: Pretreat with antibiotics, both topical and systemic
   2. Autoimmune disease: Topical and/or systemic immunosuppression (treat primary disease)
   3. Ocular surface instability – limbal stem cell deficiency, dry eye, exposure, etc.

B. Tectonic indications: failing medical management, increase in melting process, prevention of descemetostome formation or perforation

C. Optical indications: Secondary goal to reduce / correct irregular or regular astigmatism

D. Postoperative care involves continued management of infection or inflammatory processes.

II. Peripheral Corneal Disease Conditions That May Require Surgical Intervention

A. Infectious keratitis / limbitis / scleritis
B. Rheumatoid and other autoimmune disorders (eg, Wegener granulomatosis)
C. Mooren ulcer
D. Ocular surface melts (eg, chemical / thermal burns)
E. Other peripheral lesions: pterygium melts, epibulbar dermoids, Terrien degeneration

III. Types of Surgical Intervention

A. Peripheral thinning without perforation: lamellar patch graft
B. Peripheral thinning with perforation: lamellar or full-thickness patch graft
C. Role of corneal glue – acrylic vs. fibrin
D. Role of amniotic membrane transplantation
E. Conjunctival resection – Mooren ulcer

IV. Concept of Surgical Technique for PUK

A. Tectonic lamellar procedure: avoid endothelial rejection, AC complications
B. Minimal tissue replacement: Avoid replacing normal corneal tissue – shape will be similar to the melt – peripheral crescenteric graft – “match-patch” grafts (see Figures 1a, 1b)

C. Correct ectasia, reduce astigmatism induced by the graft shape/site – peripheral crescenteric graft
D. Recurrent melts may occur. either over the original graft or over another area. Repeat patch grafting may be necessary (see Figure 2).
V. Surgical Technique of Tectonic Lamellar Semilunar (Crescentric) Patch Graft

A. Conjunctival peritomy/resection

B. Recipient bed preparation
   1. Marking trephines and measurement of recipient bed outlines (see Figure 3)
   2. Regularization of edges: straight, vertical edges, regular lamellar bed depth
   3. Shape of graft: crescentric or “banana” shape to follow melt profile and peripheral corneal profile; less involvement of central cornea, less irregular astigmatism

C. Donor preparation
   1. Glass ball mount as an artificial chamber maintainer; donor sutured onto cloth-covered ball (see Figure 4)
   2. Replication of recipient bed shaped onto donor tissue – marking trephines, (corneal and dermatological), caliper measurements
   3. Lamellar dissection or full-thickness donor; if full thickness, strip Descemet membrane for better graft adhesion (see Figure 5).

4. Use diamond knife for freehand cutting out of donor shape, together with various trephines.
D. Graft suturing
1. Compressive suturing; overcorrect astigmatism to allow postoperative selective suture removal.
2. To greatly reduce severe ectasia, utilize a donor that has a width of 0.25 mm narrower than the recipient bed, and use tight 9/0 nylon sutures to compress the recipient bed, which reduces the ectasia. Aim for significant overcorrection for subsequent selective suture removal (see Figure 6).

E. Descemetocles or small perforations: similar lamellar technique can be employed:
1. Descemetocle or near descemetocle depth of melt – use full-thickness donor
2. Small perforation: During lamellar bed dissection, leave area of perforation till last; fibrin glue may be useful, but usually unnecessary.
3. Perforation with flat or shallow AC: Perform lamellar dissection first, then reform chamber and sweep iris away from perforation site.

VI. Postoperative Care
A. Topical steroids and antibiotics
B. Bandage contact lens removal
C. Continue / modify / tail-off medical / systemic management
D. Selective suture removal to reduce the overcorrection of astigmatism
E. Long-term follow-up to be emphasized for cases of PUK with recurrent potential

References
Modern Immunosuppressant Agents for Corneoscleral Disease

Ramana S Moorthy MD

I. Treatment Paradigm for Immunomodulatory Therapy

Treatment guidelines for noninfectious ocular inflammatory disease

A. Indications
   1. Noninfectious ocular inflammatory disease
   2. Other diseases in which an inflammatory component exists, if therapy directed against the primary etiology is also used (eg, infectious disease)

B. Goal of therapy
   1. Control of the inflammation
   2. Eliminate the risk to vision from the structural and functional complications resulting from uncontrolled inflammation

C. Choice of agent based on a careful consideration of:
   1. Specific diagnosis
   2. Concurrent ocular or systemic disease
   3. Existing level of ocular function compromise
   4. Patient desires

D. Initial therapy goal: rapid control of inflammation; “douse the fire”
   Corticosteroids: most effective agent
   1. Topically, regionally, and systemically
   2. The Multicenter Uveitis Steroid Treatment Trial (MUST), a randomized, controlled, superiority trial, comparing systemic anti-inflammatory therapy with fluocinolone acetonide implant for intermediate, posterior, and panuveitis, was conducted and recently published the following results:
      a. In each treatment group, mean visual acuity improved over 24 months, with neither approach superior, to a degree detectable with the study’s power.
      b. The specific advantages and disadvantages identified based on individual patients’ particular circumstances should dictate selection between these two alternatives.
      c. Systemic therapy with aggressive use of corticosteroid-sparing immunosuppression was well tolerated.
      d. For certain conditions such as mild scleritis, nonsteroidal anti-inflammatory agents may be used instead of corticosteroids.

E. Certain conditions indications for the early initiation of immunomodulatory therapy: Strongly consider early initiation if:
   1. Ocular cicatricial pemphigoid
   2. Necrotizing scleritis with systemic association
   3. Peripheral ulcerative keratitis
   4. High-risk corneal transplantation
   5. Limbal stem cell transplantation

F. Tapering of initial therapy
   1. If disease activity recurs with taper, then the dose of corticosteroid at which the flare occurred determines whether long-term corticosteroid therapy or second-line therapy is used.
   2. If control is not achieved with initial therapy, then transition to second-line therapy.

II. Paradigm: Second-Line Therapy

A. In chronic disease not controlled at a safe dose of corticosteroid (actually, there is no chronic dose of systemic corticosteroid considered by bone specialists to be safe for chronic use)

B. Acute or limited duration disease in which initial corticosteroid therapy failed to achieve control

C. Individuals unable to tolerate doses of initial therapy

D. Multiple drug classes and agents: data from randomized controlled trials generally lacking

E. Selection of agent is thus based on a consideration of an individual patient’s comorbidities.

III. Drug Classes

(As new agents are continually being developed and released, this list may be incomplete.)

A. Antimetabolites
   1. Methotrexate
      a. Mechanism of action
      b. Dosing
      c. Side effects and complications
      d. Laboratory screening
   2. Azathioprine
   3. Mycophenolate mofetil or mycophenolic acid

B. Calcineurin inhibitors
   1. Cyclosporine
   2. Tacrolimus
C. Alkylating agents
   1. Cyclophosphamide
   2. Chlorambucil

D. Biological response modifiers
   1. Infliximab
   2. Adalimumab
   3. Etanercept
   4. Rituximab
   5. Intravenous immunoglobulin

IV. Beyond Second-Line Therapy
A. If use of initial and second-line therapy are ineffective in controlling inflammation
B. Options may include:
   1. Medical combination immunomodulatory therapy: multiple drugs from more than one class of drug
   2. Surgical therapy in specific entities

V. Special Considerations
A. Pregnancy
   1. Pregnancy testing in all cases
   2. Prevention / contraception – 2 forms
B. Vaccine recommendations
   Patients receiving anti-TNF therapy should not have live vaccines, including, but not limited to varicella zoster, oral polio, or rabies vaccination, and the influenza vaccine made with a live virus.

VI. Evidence-Based Guidelines for Immunomodulatory Therapy in Specific Corneal and External Disease Inflammatory Entities
A. Ocular cicatricial pemphigoid
   1. Corticosteroids
   2. Mycophenolate mofetil: milder disease
   3. Methotrexate: milder disease
   4. Cyclophosphamide IV; 6 months; rapidly progressive disease
   5. Dapsone (1 mg/kg/d, max dose 200 mg/day); recurrence with taper within 6 months
B. Necrotizing scleritis and peripheral ulcerative keratitis, Mooren ulcer
   1. Corticosteroids
   2. Alkylating agents: early use especially of associated systemic vasculitis
   3. Rituximab: Granulomatosis with polyangiitis, polyarteritis nodosa, relapsing polychondritis
C. High-risk corneal transplantation
   1. Should there be a role for HLA typing?
   2. Role of stromal anti-VEGF therapy
   3. Immunomodulatory therapy: Evidence?
      a. Cyclosporine
      b. Tacrolimus
      c. Azathioprine
      d. Mycophenolate
      e. Rapamycin
      f. Monoclonal antibodies: Campath -1H
D. Limbal stem cell transplantation
   1. Cyclosporine
   2. Tacrolimus
   3. Azathioprine
   4. Mycophenolate
   5. Rapamycin
   6. Biologics
Presentation of Case

Esen K Akpek MD

Diagnosis and Treatment of Necrotizing Scleritis

In a series of 243 patients with scleritis, we noted that 44.0% had an associated medical condition: 7.0%, an infection, and 37.0%, a rheumatic disease. The most frequent rheumatic disease was rheumatoid arthritis (15.2% of patients). Of the patients with an underlying disease, 77.6% had a previously diagnosed disease, 14.0% had their conditions diagnosed as a result of the initial evaluation, and 8.4% developed a systemic disease during the follow-up. Systemic vasculitis was less likely to have been previously diagnosed than other rheumatic diseases (59.1% vs. 83.8%, \( P = .015 \)) and more likely to be diagnosed by the initial diagnostic evaluation (27.3% vs. 8.8%, \( P = .027 \)). 4.1% of the patients had a positive antineutrophil cytoplasmic antibody (ANCA) test result without clinical evidence of a systemic vasculitis. Among patients with no evident systemic disease after the initial diagnostic evaluation, the rate of occurrence of a rheumatic disease was 4% per person-year.

REPORT OF A CASE

Patient SW is a 49-year-old Asian woman whose symptoms began in 2001 when she developed left ear pain. She had copious drainage as well and was found to have an eardrum rupture. She also developed mastoiditis and polyarthralgias, mostly manifested by bilateral ankle pain and swelling. The patient was having headaches that were quite severe.

When she was admitted to have a mastoidectomy she became hypoxic. A chest x-ray and computed tomography were ordered and noted to have abnormal pneumonia, infectious vs. inflammatory, was raised. Bronchoscopy showed no infectious etiology. An extensive diagnostic workup was performed, including serology. Open lung biopsy was pursued and showed “multiple foci of neutrophilic capillaritis with wisps and plugs of organizing pneumonia.” Some of the larger blood vessels were also involved, with granulomatous inflammation and giant cells. Eosinophils were appreciated as part of the inflammatory infiltrate. These findings were interpreted as alveolar capillaritis.

During her hospital stay, the patient developed left eye pain and redness. Slitlamp examination revealed an area of conjunctival and deep episcleral hyperemia, and scleral edema. No corneal or anterior segment inflammation was noted. Dilated fundus examination was unremarkable. Despite aggressive medical treatment using systemic rituximab and high-dose corticoste-roids, the scleral inflammation worsened and progressed to a large area of necrosis with uveal show (see Figure 1). A tectonic patch graft using irradiated bovine pericardium had to be performed to reinforce the sclera. At the same time multiple biopsy specimens harvested from the conjunctiva and necrotic scleral bed were submitted for histopathological evaluations and did not demonstrate any infectious etiology but chronic nongranulomatous inflammation.

The rituximab treatment was discontinued, and the patient was placed on oral cyclophosphamide. Systemic corticosteroids were continued. Over a period of 3 weeks the scleral inflammation became stable with frank improvement in the systemic symptoms as well. The plan was to keep the patient on the cyclophosphamide treatment for a period of 1 year.

Figure 1. Slitlamp appearance of the scleral necrosis and uveal show in a patient with multiple systemic complaints and granulomatous angiitis and alveolar capillaritis of the lungs.

Selected Readings


First and foremost in the discussion of antibiotic prophylaxis for cataract surgery, it must be stressed that the use of the antiseptic povidone-iodine 5% solution in the conjunctival cul-de-sac prior to surgery is the cornerstone of endophthalmitis prophylaxis. There is no consensus on which antibiotic to use or the method of application surrounding cataract surgery; however, there is general agreement that perioperative antibiotics are standard of care. Since there are no prospective, randomized clinical trials regarding when to start antibiotic prophylaxis, we will defer to the plethora of studies done by our general surgery colleagues. The studies in general surgery have shown that the most efficacious time to use antibiotics is starting no more than 1 hour prior to surgery, 30 minutes prior to incision being the optimal time to begin intravenous antibiotic prophylaxis. Many ophthalmic surgeons point to studies done by Frank Bucci and Christopher Ta, showing a decrease in periocular bacterial flora as the rationale for using preoperative topical antibiotics 1 to 7 days prior to cataract surgery. Although this is surrogate evidence that there may be prophylactic efficacy to this strategy, other reasons may make this strategy useful—for example, teaching patients to use medications from a reusable dropperette. Since there is no extra cost to the health-care system (one bottle typically lasts more than enough time for effective cataract surgery prophylaxis), and since there most likely is no harm being done as long as the patient is using the medication in the manner accepted by the FDA, the strategy of using preoperative antibiotics day(s) prior to cataract surgery can be an acceptable means of prophylaxis, if not yet proven.

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The reason I don’t use antibiotics day(s) prior to surgery stems from my fear that the patient may not follow instructions, since they are typically given week(s) before surgery. Were patients to use the antibiotic for weeks prior to surgery, or use varying doses, resistant bacteria may be selected prior to surgery. Another issue may be that if the patient did not use the antibiotics prior to surgery, does one cancel the surgery because the medication was not used, and if not, the patient may question why it was recommended to be used in the first place. Furthermore, a patient may lose the bottle or use the entire bottle prior to surgery, adding to the cost of surgery.

Again, since the ophthalmic peer-reviewed literature is of no help in terms of the optimal time to end postoperative use of antibiotics for the purpose of prophylaxis, we will turn to the general surgery literature, which shows that the benefits of postoperative intravenous antibiotics lasts for the first 12 hours, 24 hours maximum. The general feeling for ophthalmic surgery is to use topical antibiotics until the epithelium is healed, roughly 3 to 10 days. Due to the use of topical steroids following cataract surgery, which are not typically used following general surgery procedures, I think it is prudent to use topical antibiotics until the epithelium is intact following cataract surgery. Although it is typical to taper steroids and other anti-inflammatory medications, it is important not to taper antibiotics due to the real risk of developing and selecting for bacteria that are resistant.

So the recommendation is to use topical antibiotics starting at least 30 minutes to 1 hour prior to surgery and continuing the medication at full FDA dosages until the epithelium is healed, roughly 3 to 10 days, without tapering the antibiotic.

Which antibiotic to use is even less clearly elucidated. Several caveats to use while deciding which antibiotic include peak concentrations (Cmax) in the ocular tissues, and the minimum inhibitory concentration (MIC) of the key bacteria that cause endophthalmitis. Other characteristics may be considered, such as spectrum of coverage, cidal vs. static, biocompatibility, cost, and so on, but the main characteristics that define antibiotic efficacy are Cmax and MIC. Today in ophthalmic surgery, fluoroquinolones have the combination of generally the lowest MICs and the highest concentrations in ocular tissues when used topically. Currently, among the fluoroquinolone class, besifloxacin, gatifloxacin, and moxifloxacin are the most potent (lowest MICs) and have the highest concentrations in the cornea and the anterior chamber (highest Cmax). It is my opinion that cataract surgeons should be utilizing one of these two agents for perioperative cataract surgery prophylaxis. The choice may also vary depending on the resistance patterns in the area and the medical history of the patient, for example, if there is a history of methicillin-resistant Staphylococcus aureus (MRSA) colonization. The surgeon may wish to consult with the local infectious disease specialist to aid in choosing the best prophylactic antibiotic.

Recently, many retrospective and prospective studies evaluating intracameral antibiotics injected at the conclusion of surgery have created earnest discussion among cataract surgeons regarding the future of prophylaxis. The studies are reporting significant reduction in the rates of endophthalmitis when using intracameral antibiotics at the conclusion of surgery. Intracameral antibiotics may be the future of cataract surgery endophthalmitis prophylaxis, but questions must be addressed before they become the standard of care, including questions about optimal drug, optimal dose, and potential short-term and long-term adverse effects of injecting such medications intracameraly.
Steroids in Ulcers

Thomas M Lietman MD
Fungal Keratitis Management: Best Drugs; Mycotic Ulcer Trial Review

Venkatesh Prajna Namperumalsamy MBBS

Introduction

Fungal keratitis is an important cause of corneal suppuration. In many developing countries around the world, fungi have replaced bacteria as the most common cause of ulceration. The visual outcome following this condition is poorer than that of bacterial keratitis. Therapeutic options for this condition are limited. Natamycin, discovered in the 1960s, still remains as the gold standard. In fact, it is the only topical antifungal approved by the U.S. Food and Drug Administration for topical ophthalmic use. In recent times, voriconazole has emerged as an effective alternative, with isolated case reports discussing its efficacy. A survey performed among chosen cornea specialists throughout the world suggested that they preferred voriconazole (79%) over natamycin (55%) in the treatment of fungal keratitis. In vitro microbiological studies have demonstrated good in vitro susceptibility patterns for voriconazole. In order to compare the efficacy of voriconazole and natamycin in the treatment of filamentous fungal keratitis, we performed a double-masked multicenter randomized clinical trial.

Methods

In this study, 368 fungal keratitis patients (smear positive for fungus) were randomized to receive either topical 1% voriconazole or 5% natamycin. The drugs were applied topically every hour during waking hours till re-epithelialization and then 4 times per day for 3 weeks. The primary outcome was BSCVA 3 months from enrollment. Secondary outcomes that were measured were infiltrate or scar size at 3 weeks and 3 months, time to re-epithelialization, microbiological positivity at 6 days, and corneal perforation and/or therapeutic penetrating keratoplasty. Double-masking was achieved through repackaging both the natamycin suspension (5% Natacyn, preserved with benzalkonium chloride 0.01% ) and voriconazole 1% (Vfend IV, reconstituted in sterile water for injection with benzalkonium chloride, 0.01%) in identical opaque containers of 3 ml each. Prior to each examination, the eye was irrigated gently to avoid the failure of masking.

Results

A total of 323 patients were enrolled into the study. One hundred and sixty-one patients were allocated to the voriconazole group and 162 patients to the natamycin group. Fusarium was the most common organism isolated, accounting for 40% of the patients, followed by Aspergillus (17%). The median duration of treatment was 31 days in the natamycin-treated arm and 39 days in the voriconazole-treated arm. Eyes that received natamycin therapy had a significantly better 3-month BSCVA (P = .006). They were also less likely to have perforation or require penetrating therapeutic keratoplasty (P = .009). On subgroup analysis, it was found that the difference was more pronounced in Fusarium cases.

A higher fraction of individuals randomized to voriconazole (69 out of 144, constituting 48%) tested culture positive at 6 days than individuals randomized to natamycin (23 out of 155, constituting 15%).

Conclusion

Natamycin treatment had a better outcome than voriconazole in patients will filamentous fungal keratitis. This difference was more pronounced in keratitis caused by Fusarium. Gram-positive and Gram-negative bacteria have been known to have different clinical susceptibility patterns. Our study demonstrates that this phenomenon of differential susceptibility exists among fungal organisms as well. Therapeutic options should be tailored according to the individual fungal organisms. Monotherapy with topical voriconazole cannot be recommended for filamentary fungal keratitis.

References

Glaucoma Surgery and Corneal Surgery: What’s the Best Procedure for the Penetrating Keratoplasty Eye, and the Best Timing?

Francis W Price Jr MD

What are the objectives?
- Control the IOP reliably
- Minimize surgical damage to the corneal endothelium and transplant
- Minimize long-term problems
- Consider costs

What are the options when pressure can no longer be controlled with eye drops?
- Laser: selective laser trabeculoplasty (SLT); argon
- Minimally invasive glaucoma surgery (MIGS)
- Standard filtration surgeries
- Nonpenetrating surgeries
- Tubes/setons

What do we know?
- Prolonged elevated IOP damages the optic nerve and the graft endothelium.¹
- Long-term use of topical corticosteroids to prevent graft rejection increases IOP in many patients.²
- Intracorneal and immediate postoperative surgical trauma can damage the endothelial cells from flat anterior chambers, suprachoroidal hemorrhages, bleeding, and manipulation.
- Filtration surgeries, both trabeculectomies and tubes, are the most significant risk factor for graft failure in endothelial keratoplasty (EK) eyes, and they are also a risk factor for penetrating keratoplasty (PK) failure.³ ⁴

What are the issues?
- Laser: SLT, argon
  - Excellent option, especially SLT, because the treatment causes limited damage to the angle structures
  - However, either of these laser procedures may be difficult or impossible in post-PK eyes because of disruption of the view through gonioscopy from a PK or Descemet-stripping automated endothelial keratoplasty (DSEA)wound.
- Descemet membrane endothelial keratoplasty (DMEK) wounds have the lowest profile and are least likely to cause focusing problems.
- MIGS
  - Excellent option but currently only approved for placement of 1 implant at the time of cataract surgery.⁵
    - May require 2 stents to adequately control IOP
    - With new regulations, no reimbursement for placement of MIGS outside of cataract surgery or as multiple implants. Therefore it has limited application in the United States unless in a privileged group.
  - No information yet on effect on graft or endothelium
- Standard filtration surgeries
  - Trabeculectomies: typically done with antimetabolites like mitomycin C
  - Metal shunts
- Nonpenetrating surgeries
  - Stegmen’s innovations
  - Implants with blebs
  - Sutures in Schlemm canal without blebs
  - Essentially avoids flat anterior chambers, overfiltration, and suprachoroidal effusions and hemorrhages
- Tubes/setons
  - Valve or no valve
  - Most “reliable” IOP control in complicated cases
  - Most long-term trauma to endothelium
- How to minimize complications
  - Use topical anesthesia to prevent “central snuff” from injection of fluid and anesthetics in area of orbital apex in eyes with compromised nerves (still required in eyes with nystagmus).
  - Use low-profile implantation technique to avoid chronic irritation to tube from lid (Alvarado)
  - Nonpenetrating techniques vs. standard filters
  - Limit tube length in the eye to 1 to 2 mm
- Timing
  - As long as a patient is on topical steroids there is a chance the IOP can become elevated, so routine follow-up is required.
  - Intervention depends on status of cup, visual field, and IOP. Operate when it is obvious that topical medications are not working.
  - My opinion: Oral carbonic anhydrase inhibitors should be used only for temporary IOP control, not long-term, due to serious systemic side effects.
  - Because of the risk of accelerated endothelial loss with filters and tubes, these surgeries should be done if the optic nerve is in jeopardy, but the patient should be told that the graft may need to be replaced within 5 years. We can replace grafts, but not optic nerves.

References


Which Is Better: Epi-on or Epi-off Corneal Crosslinking?

William J Dupps MD PhD
Advanced Combination Crosslinking

José L Güell MD PhD

The most common indication for corneal collagen crosslinking (CXL) has been on primary or secondary keratoconic corneas with the goal of strengthening the corneal biomechanical structure. The conventional CXL strategy (c.CXL) with riboflavin and ultraviolet light (UVA) consists first of corneal wide-area de-epithelialization to allow an adequate riboflavin penetration into the stroma. Not only discomfort but also most of the described complications are related to this step. This has been the main reason for the proposal of some transepithelial techniques. While preserving the epithelium, those techniques pretend to ensure the same efficacy concerning postoperative corneal strength.

To allow the riboflavin to penetrate through the epithelial barrier, several chemical modifications of riboflavin have been applied, such as the addition of enhancers (EDTA, benzalkonium chloride, or 20% alcohol) or osmolar modifications. Between them, the most commonly used has been Ricrolin TE, which combines amino alcohol and EDTA. Neither with it, nor with other modifications, has efficacy been demonstrated to be as good as with the conventional approach (see Baozch, JCRS, 2009; Filippello, JCRS, 2012; Caporossi, EJO, 2012, and JCRS, 2013; Zhang, JCRS, 2012; Stojoanovic, Ophthalmology, 2012; Leccisotti, JCRS, 2012; Spadea, Clin Ophthalmol, 2012; Koppen, JCRS, 2012; Rechichi, JCRS, 2013; Salmon, JCRS, 2013; Arbaleda, IOVS, 2014; Wollensak, Cornea, 2014; Kocak, J Fr Ophthalmol, 2014).

On the other hand, the iontophoresis technique, a noninvasive procedure during which a low-intensity electric current is applied to enhance the penetration of riboflavin into the stroma, looks to be as efficient as the conventional application of riboflavin, at least in preclinical studies (Cassagne, IOVS, 2014) and in the initial clinical cases where it is being tested, such as in our group at Instituto Microcirugía Ocular in Barcelona.

Another field that is significantly changing is the modification of the UVA indication profile, as well as the shortening of the UVA irradiation time, while increasing the irradiation power. Of course, longer follow-up and more comparative studies are needed to define the future transepithelial strategies, as well as more convenient times in this rapidly evolving field.

At the same time, and considering that the main indication for CXL in keratoconic eyes is the stabilization of the corneal changes (new investigators seem to suggest the refractive capabilities of this technology, although the trials are still on their early phase), several combination strategies have been proposed since 2008.

In between the different strategic combinations, we should highlight the following: (1) photorefractive keratectomy (PRK) and CXL, (2) intracorneal ring segments (ICRS) and CXL, and (3) phakic IOLs (P-IOLs) and CXL.

Despite the conceptual contraindications in using PRK on keratoconic eyes, the combination with CXL is becoming a powerful tool, well accepted internationally, to improve high- and low-order aberrations (PRK) and stabilize the situation (CXL). In most circumstances both surgeries may be performed at the same surgical session, and most investigators consider topo-guided transepithelial PRK the refractive procedure of choice (Kymionis, JCRS, 2010; Kremer, JCRS, 2012; Yeung, JCRS, 2013; Kanellopoulos, JRS, 2011; Alessio, Am J Ophthalmol, 2013). If the results are consistently good (as they are in the published preliminary studies) over a mid- and long-term follow-up time, this combination will probably substitute the use of ICRS because with the excimer laser we may correct low and mid levels of ametropia (low order) but also most of the irregular astigmatism component (high order).

The combination of ICRS and CXL (with or without additional PRK) has also been extensively used worldwide in this group of patients from 2008. The goal of ICRS is to improve the irregular astigmatism component and some degree of low ametropia (although some published work seems to give to ICRS the capability to stop the progression of the cone, this has not been accepted by all of us). There have been defenders of all the possible steps in such a strategy, first CXL and some weeks or months later the ICRS, the opposite or both at the same time (Kambouroglu, JRS, 2008; Coskunseven, JCRS, 2009; Vicente, BJO, 2010; Kymionis, JCRS, 2010; El-Raggal, JCRS, 2011; Kremer, JCRS, 2012; Kilic, JCRS, 2012; Yeung, JCRS, 2013; Legare, Can J Ophthalmol, 2013; Yildirim, AJO, 2014). In clinical practice, most of us prefer the implantation of the ICRS first and some months later, the CXL procedure to stabilize the situation, both from a biomechanical and a refractive point of view.

In those situations where the cone is stable (usually after CXL) and, ideally, when a low degree of irregular astigmatism is present (with or without previous topo-guided PRK and/or ICRS implantation), we might consider the implantation of a P-IOL to correct the residual ametropia. This strategy has been especially useful in those young patients with stable cone after CXL and with high ametropia, intolerant to contact lenses. In general, one of the main advantages of such a group of patients is that they used to have deep anterior chambers, being the most important concern, in the long term, after P-IOL implantation (endothelial protection; see Figure 1). Regarding this particular combination, not too many papers have been published (case report Kymionis, OSLI, 2011; Dirani, EJO, 2013; Coskunseven, JCRS, 2013; Fadlallah, JRS, 2013) although, possibly, the strategy, including the

Figure 1. 27-year-old woman KC O.U. with c.CXL + ICRS implantation O.U.; UCVA O.D. 20/25; O.S. 20/20.
additional possible use of ICRS will be incorporated in the near future by a significant number of surgeons (see Figure 2).

We will show some examples of our experience with this particular combination using c.CXL and Artiflex P-IOL mostly Toric (Guell, JCRS, 2012).

**Take-home message:** The combination of stabilization techniques (CXL in any of its forms) with transepithelial PRK and/or ICRS and/or P-IOLs is a robust strategy that probably will help keratoconic eyes to avoid the need for keratoplasty throughout all their lives, enjoying a good quality of uncorrected vision.
2014 Advocating for Patients

Stephanie J. Marioneaux MD

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

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

Your Eye M.D. colleagues serving on the Academy’s Secretariat for State Affairs commit many hours on your behalf while strategizing and collaborating with state ophthalmology society leaders to ensure the success of Surgery by Surgeons. Their ultimate goal—protecting quality patient eye care in the states—requires a robust Surgical Scope Fund, and we need every single Eye M.D. to step up to the plate and deliver with their checkbooks.

The Academy’s federal advocacy arm works to protect ophthalmology practices from payment cuts, burdensome regulations, scope of practice threats, as well as to advance the profession by promoting funding for vision research and expanded inclusion of ophthalmology in public and private programs. It is critical for our OPHTHPAC Fund to also be strong.

Surgical Scope Fund

The Surgical Scope Fund (SSF) provides grants to state ophthalmology societies to support their legislative, regulatory and public education efforts. Since its inception, the Surgery by Surgeons campaign, in partnership with state ophthalmology societies and with support from the SSF, has helped 31 state/territorial ophthalmology societies reject optometric surgery proposals.

2014 has proved to be a challenging year, with several battleground states facing major optometric surgery initiatives. A number of state ophthalmic societies benefited from SSF disbursements and were able to successfully implement patient safety advocacy campaigns to defeat attempts by optometry to expand its scope of practice to include surgery. The Nebraska Academy of Eye Physicians and Surgeons was successful in its patient advocacy and public education efforts to derail legislation that would have granted optometrists the authority to perform eyelid surgery and injections. Additionally, the Arizona Ophthalmological Society succeeded in protecting patients by stopping legislation that would have allowed optometrists to gain authority to perform injections. The SSF is also at work assisting ophthalmic societies with their efforts to protect patients in California, Delaware and Massachusetts.

Proactively, the Georgia Society of Ophthalmology introduced a bill that would establish a formal definition of “surgery” into state law. While the legislative session expired before the bill could advance, Georgia ophthalmologists will be back in 2015 in an effort to pass this important safeguard for their patients.

2014 was certainly not without its challenges. Despite a vigorous battle for patient safety on the part of the Tennessee Academy of Ophthalmology, the Tennessee Medical Association and the Academy, the legislature passed a bill allowing optometrists to inject anesthesia into the eyelids. Previously, optometrists were authorized to perform only therapeutic injections and any surgical procedure that required no more than a topical anesthetic.

And in Louisiana, the Academy, the Louisiana Ophthalmology Association, and the Louisiana State Medical Society vigorously opposed legislation that would authorize optometrists to perform certain scalpel and laser surgeries and injections. On June 1, 2014, Louisiana Governor Bobby Jindal signed into law a laser surgery bill that will allow optometrists to perform scanning laser trabeculoplasty and argon laser trabeculoplasty glaucoma surgery procedures, as well as YAG capsulotomy surgery procedures, with the completion of as little as 32 hours coursework. The Academy’s Secretariat for State Affairs knows from past experience that with this success in Louisiana, organized optometry will push hard in 2015 to see if they can gain additional surgery states. This is why everyone must “advocate for patients,” engage in the state political process, and aggressively support the SSF.

California, Delaware, and Massachusetts remain “in play” and are still faced with active O.D. surgery legislation. When it comes to state legislation of any kind, California and Massachusetts are often considered bellwether states for the rest of the nation. Now more than ever, your contribution to the SSF is needed as a critical tool of the Surgery by Surgeons campaign to protect quality surgical care for our patients. The Academy relies not only on the financial contributions to the SSF from individual Eye M.D.s and their business practices, but also on the contributions made by ophthalmic state, subspecialty and specialized interest societies. The Cornea Society contributed to the Surgical Scope Fund in 2013, and the Academy counts on its contributions in 2014.

OPHTHPAC® Fund

OPHTHPAC is a crucial part of the Academy’s strategy to protect and advance ophthalmology’s interests in key areas, including physician payments from Medicare as well as protecting ophthalmology from federal scope-of-practice threats. Established in 1985, today OPHTHPAC is one of the largest and most successful political action committees in the physician community.

In the past, Politico highlighted OPHTHPAC as one of the most successful health PACs in strategic giving. By making strategic election campaign contributions and independent expenditures, OPHTHPAC helps us elect friends of ophthalmology to federal leadership positions, ultimately resulting in beneficial outcomes for all Eye M.D.s. For example, in the 2012 election cycle, OPHTHPAC was able to help retain 20 physicians in Congress.

Among the significant impacts made by OPHTHPAC are the following:

- Prevented onerous national patient prescription requirements for compounded drugs and preserved access to most ophthalmic compounded drugs for office use
- Averted significant cuts to Medicare payments due to the Sustainable Growth Rate (SGR) formula
### Surgical Scope Fund
- To derail optometric surgical scope-of-practice initiatives that threaten patient eye safety and quality of surgical care
- Political grassroots activities, lobbyists and media; no funds may be used for candidates or PACs
- Contributions: Unlimited.
- Contributions are 100% confidential.

### OPHTHPAC® Fund
- Ophthalmology’s interests at the federal level – Support for candidates for U.S. Congress
- Campaign contributions, legislative education
- Contributions: Limited to $5,000
- Contributions above $200 are on the public record.

### State EyePAC
- Support for candidates for State House and Senate
- Campaign contributions, legislative education
- Contribution limits vary based on state regulations.
- Contributions are on the public record depending upon state statutes.

- Protected practice expense increases for ophthalmology when other specialties sought legislative carve-outs
- Protected ophthalmologists’ ability to provide in-office diagnostic testing without triggering self-referral violation
- Prompted congressional action that helped reduce ophthalmology’s multiple procedure payment reduction
- Secured appointment of full-time ophthalmology national program director in the U.S. Department of Veterans Affairs
- Provided further exemptions from both the Electronic Prescribing and Meaningful Use EHR penalties

Leaders of the Cornea Society are part of the Academy’s Ophthalmic Advocacy Leadership Group (OALG), which has met for the past seven years in January in the Washington, D.C., area to provide critical input and to discuss and collaborate on the Academy’s advocacy agenda. The topics discussed at the 2014 OALG meeting included a focus on the collaboration needed among the Academy and its OALG partners on the issue of compounding. As a 2014 Congressional Advocacy Day (CAD) partner, the Cornea Society ensured a strong presence of cornea specialists to support ophthalmology’s priorities as nearly 400 Eye M.D.s had scheduled CAD visits to members of Congress in conjunction with the Academy’s 2014 Mid-Year Forum in Washington, D.C. The Cornea Society remains a crucial partner with the Academy in its ongoing federal and state advocacy initiatives.

### State Eye PAC
We all must also support our respective State Eye PACs, because state ophthalmology societies cannot count on the Academy’s SSF alone. 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 also critical. The Secretariat for State Affairs strategizes with state ophthalmology societies on target goals for state eye PAC levels.

### ACTION REQUESTED: Advocate for your patients!!
Academy Surgical Scope Fund contributions are used to support the infrastructure necessary in state legislative / regulatory battles and for public education. PAC contributions are necessary at the state and federal level to help elect officials who will support the interests of our patients. Contributions to each of these three funds are necessary and should be considered the costs of doing business. Surgical Scope Fund contributions are completely confidential and may be made with corporate checks or credit cards, unlike PAC contributions, which must be made by individuals and are subject to reporting requirements.

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

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Combined Glaucoma and Cataract Surgery: Microinvasive Glaucoma Surgery—Top 5 Pearls

Reay H Brown MD

I. Introduction: Why should cornea and refractive surgeons be interested in glaucoma surgery?

Why should you make a commitment to microinvasive glaucoma surgery (MIGS)?

A. MIGS allows you to help your cataract patients with glaucoma (within the skill set of cornea/refractive surgeons).

B. Glaucoma is common in patients having cataract surgery.

1. 22.6% of Medicare patients having cataract surgery had coexistent diagnosis of glaucoma!

2. Study was looking at risk of fractures after cataract surgery.

II. MIGS

A. Microinvasive glaucoma surgery: Definition

1. Ab interno incision

2. Minimal trauma: by strict definition, not destructive or ablative

   a. Strict MIGS definition would not include endocyclophotocoagulation (ECP) or Trabectome.

   b. MIGS does not include trabeculectomy or tube shunts.

3. Very safe

4. Rapid recovery

5. Effective

B. MIGS devices currently available: iStent approved 2012

C. Other MIGS devices in FDA studies

1. Cypass (Transcend)

2. Hydrus (Ivantis)

3. iStent Inject (Glaukos)

4. iStent Supra (Glaukos)

III. Pearl #1: MIGS is a new way of thinking about glaucoma surgery.

A. Eye drops have been mainstay of treatment for glaucoma. But many problems with medical therapy:

   1. Compliance is terrible.

   2. Recurring expense

   3. Side effects

      a. Redness

IV. Pearl #2: Pick the Right Patients

A. Patients with mild to moderate glaucoma undergoing cataract surgery

   1. Controlled IOP with early VF loss

   2. Generally 2 or fewer drops; 3 drops possible if good IOP and minimal VF loss

   3. Open angles

B. Avoid patients with advanced VF loss and uncontrolled IOP. MIGS is not a replacement for trabeculectomy or tube shunt.

V. Pearl #3: Set Proper Expectations

A. Goal: Lower IOP

   1. Perhaps reduce drops—if possible and if VF loss is minimal.

   2. Do not overpromise.

B. Especially in early cases, leave yourself an out. Tell patient you may not implant iStent if . . .

   1. Any concerns about the cataract surgery

   2. Angle is not sufficiently open

b. Irritation

c. Eyelash growth

d. Discolored skin

e. Sunken orbit

B. Distribution of medical treatment in glaucoma 2013

1. 77% of glaucoma patients on 2 or fewer drops

2. Fewer than 5% on 4 medications

   a. Pre-MIGS: This is the group where surgery was considered.

   b. But MIGS works best in the 77% on 2 or fewer drops.

C. MIGS changes attitudes toward surgery: no longer the last resort, when all else has failed.

1. Indication for surgery not visual field (VF) progression, uncontrolled IOP

2. Goal is lowering IOP/reducing medical burden.

   a. Reduce problems of noncompliance

   b. Reduce cost

   c. Reduce side effects
VI. Pearl #4: Learn the Technique

A. Adopting a new surgical procedure is challenging.
   1. Earlier is “easier.”
      a. Fewer surgeons have extensive experience.
      b. Smaller gap between you and early adopters
   2. Fear holds us back: “A year from now you will wish you had started today.”

B. Use all resources to prepare.
   1. There is a learning curve—expect challenges.
   2. Review videos.
   3. Company offers teaching: Reps and wet labs
   4. Get a nonsterile device and practice delivery outside actual surgery.
      a. Deliver into Weck-Cel sponge at the slit lamp
      b. Become familiar with release mechanism.
         Grasp and regrasp—“Letting go” is critical.
   C. Video all cases—review.
   D. Surgical technique
      1. Get best gonio view possible.
         Gonio lens: Hill vs. Vold TVG
      2. Implantation technique
         a. Approach meshwork at 15-degree angle.
         b. Engage meshwork with sharp tip.
         c. Elevate inserter and flatten out iStent as you slide it along Schlemm canal.
         d. Continue advancing until snorkel contacts meshwork.
         e. Release snorkel and tap snorkel forward until fully seated.
      3. Expect bleeding through snorkel and around it.
   D. Common implantation problems
      1. Poor gonio view
         a. Need to stop and fix
         b. More viscoelastic, less pressure on eye
      2. Approach too angled, too deep
         a. Device impaled in posterior wall, can’t advance
         b. Pull back and flatten out approach.
      3. Approach too shallow, not fully in canal
         a. Pull back and deepen approach.
         b. If device already released, regrasp and advance more deeply.
   4. Expect bleeding—may obscure view
      a. Viscoelastic to push blood away
      b. Irrigation/aspiration to clear blood if viscoelastic not enough

VII. Pearl #5: This is only the beginning! Think of RK or early phaco.

A. New MIGS devices will be approved.
   1. Cypass (Transcend)
   2. Hydrus (Ivantis)
   3. iStent Inject (Glaukos)
   4. iStent Supra (Glaukos)

B. New techniques and strategies will improve efficacy.
   1. Targeting pigmented meshwork
   2. Implanting multiple iStents
   3. Implanting multiple devices utilizing different outflow pathways. Canal-based device with suprachoroidal device.

VIII. Summary: MIGS is an opportunity to help your glaucoma patients.

A. Glaucoma is common among cataract patients; cornea/refractive surgeons do most of the cataract surgery.
B. Your innovative spirit will lead to new and improved glaucoma surgical technologies.
C. MIGS can help make glaucoma become more of a surgical disease.

References
Five Times You Really Need a Femtosecond Laser

Juan F Batlle MD

I. Four Steps to Success in Femtosecond Laser-Assisted Cataract Surgery (FLACS)
   A. Plan
   B. Dock and engage: Liquid optics interface
   C. Visualize and customize: Integral guidance OCT-safety
   D. Treat and release: Femtosecond laser high precision

Figure 1.

Figure 2.

Figure 3.
II. White Cataracts
   A. Trypan blue
   B. Avoid Argentinian flag
   C. Soft vs. hard
   D. Milky cloud of cortex
   E. Visualization of posterior capsule
   F. Nucleus is not always soft.

III. Black Cataracts / Cataracta Nigra
   A. Rock-hard nucleus
   B. Larger capsulorrhexis
   C. Identifying safety margins and posterior capsule via OCT
   D. Lotus flower phenomenon: leathery posterior nucleus
   E. Small vs. large cubes for nucleus softening
   F. Reduce phaco time and energy

IV. Zonular Weakness and Pseudoexfoliation
   A. Less trauma with femtosecond capsulorrhexis
   B. Less manipulation after softening
   C. Better centration of capsulorrhexis
   D. Capsular tension rings and segments
   E. Phaco aspiration vs. phacoemulsification
V. Fuchs Endothelial Dystrophy
A. Low endothelial cell count
B. Pachymetry near 600 microns
C. Reduced ultrasound energy
D. Less trauma to the endothelium
E. Phacoaspiration vs. phacoemulsification
F. Venturi vs. peristaltic pumps

VI. Congenital Cataracts
A. Sterile technique for femto and phaco
B. Common operating theater
C. Anterior capsulorhexis and posterior capsulorhexis
D. Lens-in-the-bag technique by Dr. Burkhard Dick
E. Effective lens position

Selected Readings
Cataract Surgery in Corneal Disease: Five Pearls

*Dasa V Gangadhar MD*

**Introduction**

Cataract surgery is frequently necessary in patients with pre-existing corneal disease. A cookie-cutter approach to cataract surgery is best avoided in the presence of complex corneal diseases. Often, special techniques are necessary in these situations to obtain the best possible outcomes. We will describe situations where a modification in standard cataract technique is necessary and offer pearls for such cases.

I. Basement Membrane Dystrophy, Salzmann Nodules, Pterygia, and Ocular Surface Disease
   A. Can create subtle or overt irregular astigmatism
   B. IOL calculation errors
   C. Dissatisfied patients postop due to “imprecise vision”
   D. Train staff to spot irregular keratometry before dilation. Surgeons should view such corneas before dilation to identify ocular surface disease and corneal pathology.
   E. Remove pathology before cataract extraction (CE).
   F. Aggressively treat dry eye and ocular surface diseases to allow for more accurate keratometry and safer surgery.
   G. If a keratectomy is performed to remove corneal pathology, allow a minimum of 6 weeks to then remeasure keratometry for IOL calculations.

II. Fuchs Dystrophy and Endothelial Compromise
   A. Scleral incision is not an arcane concept. More protective of the endothelium than standard clear cornea incisions.
   B. Use dispersive viscoelastics.
   C. When in doubt, opt for CE alone. The cornea can be fixed later if needed.
   D. Preop pachymetry and cell counts can be helpful for creating a risk-analysis for corneal decompensation, counseling patients, and for medicolegal protection.
   E. Use low-energy, low-flow, and in-the-bag emulsification techniques.
   F. If moderate or high risk for corneal decompensation, aim −1.00 to −1.50 more myopic than final intended refraction as Descemet-stripping endothelial keratoplasty (DSEK) creates a hyperopic shift.

III. Keratoconus
   A. Toric IOLs can be of great value if reasonable congruity of axis is established via manual keratometry, automated keratometry, and topography.
   B. Must use scleral incisions. Clear cornea incisions can act as a limbal relaxing incision “on steroids” and create significant shifts in astigmatism axis and magnitude.
   C. Do not use toric IOLs if rigid gas permeable contact lenses are a consideration postop.

IV. Penetrating Keratoplasty (PK)
   A. Triple procedure (PK, extracapsular cataract extraction, IOL) is becoming much less common.
   B. Perform PK alone in the majority of cases, even if cataract is present.
   C. After sutures are out in 12 to 18 months, CE with toric IOLs is a reliable way of reducing astigmatism and fine-tuning the final refractive error.
   D. Maximally protect the endothelium with a scleral incision and dispersive viscoelastic.
   E. Preop specular microscopy is of great value to identify grafts at risk for corneal decompensation.
   F. If decompensation of the PK is a likely possibility, plan −1.00 to −1.50 more myopic with CE than the final intended refraction in case DSEK over the PK is needed.

V. CE Post-Refractive Surgery (RK/LASIK)
   A. Challenging IOL calculations
   B. For RK, use a scleral incision to minimize the risk of intersecting with an RK incision (especially in 16-incision RK, where there is “no room” between incisions). Intersecting with an RK incision can create unpredictable, large astigmatic swings and irregular astigmatism.
   C. For LASIK, obtain historical data if at all possible.
   D. Use the American Society of Cataract and Refractive Surgery online calculator to facilitate IOL calculations.
   E. Consider utilizing multiple methods to predict corneal refractive power (manual keratometry, automated keratometry, topography: Nidek, Pentacam, HAZ).
   F. Carefully advise patients on the difficulties with IOL calculations, the potential for refractive surprises, and the possible need for IOL exchange, piggyback IOL, or laser refractive surgery after CE. Document!

**Selected Readings**

Cataract Case With Zonular Insufficiency

Boris Malyugin MD PhD

Introduction

Patients with zonular pathology present significant challenges for the anterior segment surgeon. Increased risk of capsular tears, vitreous prolapse, and IOL instability is expected in these cases. The cases prone to the above-mentioned complications include trauma, pseudoexfoliation syndrome, glaucoma, high myopia, hereditary systemic diseases (Marfan syndrome, homocystinuria, Weill-Marchesani syndrome, etc.), and some others.

There are several types of device designed to facilitate cataract surgery in patients with zonular dialysis. For instance, flexible iris hooks can be used to support the capsular bag in the presence of extremely loose zonules. Nevertheless, as a result of having relatively short curved portions, iris retractors tend to slip off and in some cases tear the anterior capsulorrhexis.

Specially designed capsule retractors (MicroSurgical Technology, Inc.; USA) have hooked ends, which are elongated in order to support the peripheral capsular fornix and prevent damaging the capsulorrhexis margin. These devices function like temporary artificial zonules, fixating the capsular bag to the limbal area.

Capsular tension segments (I. Ahmed, 2001) Assia Anchor (E. Assia, 2005) and suture with the T-shaped ending (R. Yamaguchi, 2008) both have the advantage of minimizing the surgical trauma and providing the permanent zonular replacement not only during the surgery but also in the long term after it. The main disadvantage of the above-mentioned devices is that they can cause only focal support of the capsular bag and do not totally restore its equator shape. That is why in many cases their combination with conventional or modified capsular tension rings is mandatory. Assia Anchor and Yamaguchi device are not FDA approved.

Capsular tension rings (CTRs) are extremely useful in stabilizing the lens during cataract surgery and reducing the likelihood of intraoperative complications. Conventional CTRs are helpful in generalized zonular weakness or localized zonular defects not exceeding 2-3 clock hours. These devices are used to maintain the circular contour of the capsular equator during surgery through stretching of the capsular bag, preventing its collapse and capsule aspiration, distributing forces equally over all zonules and preventing the vitreous prolapse into the anterior chamber.

To address moderate and profound cases of zonular weakness Dr. Cionni modified the standard CTR, adding the fixation eyelet attached to the central portion of the ring. This eyelet allows the ring to be sutured to the sclera and to provide intraoperative support during phacoemulsification. Cionni modified CTR (Morcher GmbH; Germany) is a useful tool, which can be recommended for patients with zonular dialysis exceeding the area of 3 clock hours. The ring has different embodiments, having 1 or 2 fixation elements. Most surgeons implant the device manually through the main cataract incision using forceps. The use of the injectors is very uncommon.

We introduce the Malyugin modified CTR produced by Morcher GmbH (Stuttgart, Germany), FDA approved and distributed in United States by FCI Ophthalmics (Pembroke, MA). This is a new endocapsular supporting device, developed to address the microincisional cataract surgery difficulties in patients with zonular weakness and/or zonular dialysis and to improve the issues surgeons face while implanting the Cionni modified CTR. It is designed to center the subluxated lens capsule by securing it to the scleral wall (see Figure 1).

![Malyugin modified CTR](Image)

Figure 1. Malyugin modified CTR.

The idea of the Malyugin modified CTR is based on moving the fixation element to the very tip of the ring. This makes the device completely retractable into the injector tube, subsequently allowing it to be inserted into the eye in a very controlled manner. The second advantage deriving from the design is that the curved portion of the CTR slides along the equator of the capsular bag during its injection. Thus the risk of perforating the capsular fornix with the tip of the CTR is eliminated.

Video Description

After creating clear corneal incision, capsulotomy is initiated with the sharp bent needle. Microcapsulorrhexis forceps (Seidel Rhexis Ruler, MST) are used to grasp the flap and to tear the capsule in the circular manner. Capsular folds and lens instability during anterior curvilinear capsulorrhexis are observed, which presents additional evidence of significant zonular pathology. During capsulotomy, the lens is temporarily stabilized with capsular hooks (MST) introduced through corneal paracentesis, and catching the capsulorrhexis edge.

The Malyugin CTR is partially retracted into the injector cartridge. Needle is passed through the eyelet in order to fixate it with the 90 polypropylene suture. To avoid damaging the residual zonular apparatus, the Malyugin CTR is injected in the direction of the zonular defect. During insertion the injector is positioned in the center of the anterior chamber. By pushing the plunger, the surgeon slowly guides the ring, at the same time carefully watching that it slides below the anterior capsule edge directly into the capsular bag.
The curved fixation element safely slides along the capsular bag equator without any risk of damaging it. The trailing end of the device is guided under the fixation element and released from the injector plunger with the help of the side port instrument. Using the reversed Sinskey hook, the CTR is rotated to position the fixation element at the very center of the zonular defect. The fixation element is guided out of the bag through the capsulorhexis opening and positioned on the anterior surface of the sclera.

A fornix-based conjunctival flap is created with scissors, followed by gentle cautery of the episcleral vessels. Ophthalmic viscosurgical device (OVD) is injected beneath the iris to lift it and to expand the ciliary sulcus area. The needle is passed through the ciliary sulcus with the ab interno approach. After needle externalization, it is then fixated to the superficial scleral layers in the zig-zag fashion with several bites, followed by tying a double knot. The conjunctival flap is repositioned and sutured with two 8/0 vicryl sutures placed in the limbal area.

Multiple-quadrant hydrodissection is then carefully performed. It is followed by phacoemulsification with the Stellaris system (Bausch + Lomb) utilizing the quick-chop technique of nucleus disassembly. Ultrasound energy (30% max, 160 pps) is used in the linear mode with the dual linear foot pedal control. The latter helps very much in stabilizing the anterior chamber and increasing the procedure safety margin. Vacuum is adjusted to 400 mmHg.

After the last fragment of nucleus is removed, cortical material is aspirated from the capsular bag with bimanual irrigation-aspiration (I/A) system. This step is more difficult as compared to I/A in standard cases, because the cortex is partially compressed to the equator of the capsular bag with CTR.

After cleaning the bag, a foldable acrylic IOL is injected and positioned in place with the sideport instrument. Stable and central position of the “capsular bag–IOL” complex is verified. Corneal wounds are closed with stromal hydration.

Conclusions

Cataract surgery techniques have significantly progressed during the last decades. However, compromised zonules still present significant challenges and raise the risk of intra- and postoperative complications. One of these is the long-term stability of the “IOL–capsular bag” complex. The ongoing development of endcapsular devices allows for removal of cataract in complicated cases with weak or absent zonules, as well as providing fixation of the capsular bag to the sclera. Conventional and modified CTRs provide numerous benefits to surgeons managing challenging cataracts with weakened zonules. One of the benefits of a CTR in the postoperative period is that it counters progressive contractile centripetal forces and resists capsulorrhexis shrinkage as the capsular bag contracts. This is a very likely scenario in pseudoexfoliation syndrome in which the entire capsular bag dislocates years following the initial surgery.

The Malyugin modified CTR is the novel capsular expansion device designed to be injected through the microincision and to center the subluxated capsule by securing it to the scleral wall. The modified CTR allows the loose eccentric capsules to be recentered and secured, achieving safe endcapsular implantation and long-term stability of the capsular bag and providing favorable functional and anatomical results in patients with acquired and hereditary zonular pathology.

References

Dislocated IOL Case

Sadeer B Hannush MD

Management of the dislocated IOL in 2014 remains controversial. Treatment options include observation, repositioning and fixation, removal and replacement with an anterior chamber IOL (AC-IOL) or a posterior chamber IOL (PC-IOL), fixated to the iris or the sclera, with or without sutures.

My 5 tips for managing the case of a dislocated IOL are as follows:

1. Evaluate the corneal endothelium to determine the need for endothelial keratoplasty.
2. Rule out macular edema. The presence of macular edema may change the treatment strategy and final location of the IOL.
3. Familiarize yourself with anterior vitrectomy techniques, including a transconjunctival 23- or 25-gauge pars plana approach.
4. Choose a go-to technique for PC-IOL fixation in the absence of capsular support (suture or glue).
5. Carefully plan a perioperative strategy for visual rehabilitation: intravitreal triamcinolone, postoperative corticosteroids, NSAIDs, avoidance and management of ametropia/astigmatism.

Case

We present the case of a dislocated PC-IOL with early corneal edema (low endothelial cell count) and no cystoid macular edema. Management includes a pars plana anterior vitrectomy, explantation of the IOL with residual lens material, scleral fixation of a PC-IOL, and endothelial keratoplasty.
Toric IOL in Cornea Disease
Five (or More) Pearls for the Use of Toric IOLs in Patients With Abnormal Corneas

Christopher E Starr MD

Introduction
Advanced technology IOLs (AT-IOLs), both presbyopia-correcting and astigmatism-correcting (toric), have had a major positive impact on our field, for patients and surgeons alike. For many patients who desire a reduced dependence on glasses and contact lenses and who have normal stable corneas with regular astigmatism, the added cost and complexity of an AT-IOL makes a lot of sense. But for patients with unstable, abnormal corneas and/or with irregular astigmatism, the picture is not as clear, both figuratively and literally. Some doctors argue that AT-IOLs should never be used in patients with diseased corneas, while others employ them liberally.

I sit somewhere in the middle of those extremes. Every patient, every cornea, and every surgeon is unique, and thus what works well in one scenario may not work at all in another. For almost every “rule of thumb” there is a caveat. What lies below is my personal approach to some of these complex surgical challenges.

I. No cornea is static; all corneas change with time.
A. However, normal corneas change much more slowly and more predictably than abnormal corneas.
B. Normal corneas gradually drift toward against-the-rule astigmatism (ATR) with age and time.
   1. Some surgeons advocate undercorrecting with-the-rule astigmatism (WTR) and overcorrecting ATR by about 0.5-0.75 D with toric IOLs to account for this drift, while others advocate for fully correcting all astigmatism in all cases, regardless of age.
   2. The role of the posterior corneal astigmatism and patient age should be factored into the surgical strategy. Modern tomographic diagnostic tools can help evaluate the magnitude and contribution of the posterior corneal surface to total corneal astigmatism.
   3. Abnormal corneas typically change their shape much faster and less predictably than normal corneas, making IOL selection like shooting at a moving target. This can be a risky proposition when out-of-pocket expenses and higher expectations are in play, as with AT-IOLs.
   4. Abnormal corneas typically have irregular astigmatism, which cannot fully be neutralized with spectacles or toric IOLs.
      a. Extra care and time in appropriately managing patient expectations is of paramount importance in these situations, especially if an AT-IOL is to be used.

b. As a general rule of thumb: If a rigid-gas-permeable contact lens (RGP) is to be used postoperatively to address the irregular astigmatism, then a toric IOL should not be implanted.

c. Another general rule of thumb: If a procedure (ie, superficial keratectomy [SK]) can be done before cataract surgery to normalize the corneal surface and improve astigmatism, it should be done.
   i. This is a reasonable rule to follow when the pathology is significant and in the central 3-5 mm of the cornea; however, when subtle and peripheral, the gains from a preemptive SK may be outweighed by long surgical delays, pain, healing issues, etc.
   ii. If SK is done prior to cataract surgery, another general rule of thumb is to allow at least 6 weeks of healing before calculating IOLs.
      a) In many cases, however, this is not long enough for corneal stability to occur, and thus surgical delays can be quite lengthy.
      b) Many patients lead active lives, require good vision for their jobs, and with significant cataracts in addition to corneal problems, many simply can't wait so long to have cataract surgery. A careful and thorough consent process is needed before undertaking multiple procedures.

II. Dry Eye Disease (DED)
A. By far the most common hurdle in my practice when planning cataract surgery
B. Prevalence of DED is much higher in the cataract population than previously thought.
C. Early identification and aggressive treatment preoperatively is critical to successful outcomes.
D. Novel diagnostics can help facilitate both early detection and treatment efficacy monitoring (osmolality, MMP-9, interferometry, etc.).
E. Rule of thumb: Delay surgery, and more importantly, IOL calculations, until the tear film is optimized and stable, corneal staining is absent, and corneal topography is normalized.
   Caveat: This can sometimes take a very long time, and some patients simply can't delay surgery that
long . . . but yet they still expect perfect uncorrected vision after surgery (case presentation).

F. Once the ocular surface is stabilized, AT-IOLs can be used successfully.
1. Cataract surgery incisions, pre- and postoperative drops (NSAID, preservatives, etc.), and, very significantly, limbal relaxing incisions (LRIs)\(^3\) can worsen DED after surgery.
2. Similar to laser vision correction surgery (LVC), aggressive DED management should be continued after cataract surgery for best visual outcomes with AT-IOLs. (case presentation of glare and halos with multifocal IOL and LRI that completely resolved after DED management)

III. Epithelial Basement Membrane Dystrophy, Salzmann Nodules, Subepithelial Fibrosis, Pterygia
A. To scrape or not to scrape? That is the question.
B. All of these conditions can cause significant irregular astigmatism, can fluctuate widely over time, and can recur after removal.
C. Rule of thumb: In patients who want the best chance of spectacle-free vision after cataract surgery, then “to scrape” is the answer.
1. Caveat: Patients must be told that this will delay surgery for a minimum of several months and that there is no guarantee that the irregular astigmatism will be completely resolved or that the problem won’t recur.
2. I allow a minimum of 6-8 weeks after SK to repeat keratometry and topography. I then repeat 3-4 weeks later, and only if stable will I consider proceeding with cataract surgery and AT-IOL selection.
D. In patients with subtle, stable peripheral lesions, fairly regular astigmatism in the central cornea, normal scotopic pupil sizes, and decent spectacle-corrected vision preoperatively, it may be fine to proceed with cataract surgery without performing a SK.
E. Note: Pterygia should obviously not be “scraped” but excised. Large pterygia that extend onto the cornea and induce significant astigmatism, whether regular or irregular, should be removed before cataract surgery (never concurrently).

IV. Post-Refractive Corneal Surgery: LASIK, PRK, CK, RK
A. Most commonly patients have had prior myopic LASIK or PRK, many with intentional monovision. (In my experience it is usually the near vision eye that has the more advanced cataract—unclear why. Has anyone else noticed this?)
1. These patients are generally more interested in AT-IOLs and spectacle-free vision after cataract surgery (for obvious reasons).
2. IOL calculation errors, typically hyperopic surprises, are common.
3. I always use the ASCRS Post-Refractive IOL Calculator (http://iolcalc.org). Always try to get pre-refractive surgery data, but this is not always possible. Accuracy of “no prior data” calculations has improved over the years.
4. Due to induced spherical aberration following myopic LVC, an aspheric IOL with negative spherical aberration (SA) is preferred. The opposite is true for post-hyperopic LVC in which an IOL with positive or zero SA is best.
5. Most patients following successful LVC do not have high amounts of corneal astigmatism (>1.0 D) and thus may not need a toric IOL. If there are higher levels of astigmatism, especially if irregular, then ectasia should be ruled out (see section V.)
6. If the patient wants monovision I start with the nondominant eye and enter a target of -.200 D into the ASCRS Calculator. Using the refractive outcome of that eye I can be more aggressive with targeting plano to -0.5 D in the dominant eye.
7. If postop astigmatism is predicted to be >0.75 D, I recommend a toric IOL.
8. Corneal pachymetry, surgical records, flap diameter and thickness, dry eye disease, topography, etc. should be carefully assessed prior to cataract surgery with an AT-IOL in case a postop LVC enhancement is needed. This possibility and added costs, if any, should be discussed with patients prior to cataract surgery.
B. Conductive keratoplasty (CK)
1. Treatment spots are typically peripheral, leaving central cornea fairly regular.
2. Treatment effect often regresses over time.
3. If pupil not overly large and patient sees well with glasses, then an AT-IOL is a reasonable option.
C. Radial keratotomy (RK)
1. These eyes are typically extremely complicated for a number of reasons: severe irregular astigmatism, unstable corneal biomechanics, wide refractive shifts throughout the day, unpredictable IOL calculations, complex higher-order aberrations, higher risk of surgical complications such as corneal perforation, zonular insufficiency, and capsule violation, etc.
2. Because of the above, in the precorneal collagen crosslinking (CXL) and pre-topography-guided era in the United States, I rarely, if ever, recommend toric IOLs, and never multifocal IOLs, in post-RK patients.
V. Highly Irregular/Aberrated Corneas: Keratoectasias (Keratoconus), Pellucid Marginal Degeneration), Post-LASIK Ectasia), and Post-Penetrating Keratoplasty (PK)

A. Rule of thumb common to all: If patient is a successful RGP/scleral lens wearer, then do not place a toric IOL.

B. Toric IOLs can be considered in the following instances:
   1. RGP/scleral lens intolerance
   2. Long-term corneal stability (2-3 years) in an older patient or following CXL
   3. Minimal irregular astigmatism in central 3-5 mm and/or patient has acceptable vision with spectacles
   4. Post-PK if all sutures removed, low-risk category for rejection, non-steroid responder, healthy endothelium, stable refraction, keratometry & topography, fairly regular astigmatism
      a. Toric IOLs alone in general can treat only about 4-5 D of astigmatism. Thus, astigmatic keratotomy (AK), LRI, piggyback IOL, and/or LVC can be used in addition to neutralize the higher levels of astigmatism commonly seen after PK.
      b. In keratoectasia, extreme caution should be used if planning AK, LRI, or LVC, as these procedures can further destabilize and weaken the cornea.

C. IOL calculations are very difficult and prone to error in these eyes due to a number of confounding factors, but in very select patients there can be successful outcomes.4

D. In general a standard IOL with neutral or positive spherical aberration is preferred with a slightly myopic refractive target, as hyperopic surprises are common.

E. Personal observation: Now that I get routine topography on all preop cataract patients it seems that subclinical ectasia is more common than previously thought (case presentation).

VI. Corneal Endothelial Dysfunction

A. Most commonly Fuchs dystrophy in cataract age patients

B. IOL strategies have changed in the transition from Descemet-stripping endothelial keratoplasty (DSEK) to Descemet membrane endothelial keratoplasty (DMEK).

C. In patients with mild-moderate guttata but without morning blur or Descemet folds and a central corneal thickness (CCT) less than about 630 μm, it is reasonable to proceed with cataract surgery alone.
   1. Care should be taken to protect the endothelium during surgery.
   2. If DSEK is planned in future, then IOL target should be between −1.00 and −1.50 D, depending on thickness of graft. (The thicker the tissue, the more hyperopic shift; I use 90-100 μ grafts and aim for −1.00 D in most cases.)
   3. If DMEK is planned in future, then IOL target should be −0.25 to −0.5 D.
   4. Interface interactions can limit BCVA in both DSEK/DMEK but typically less so in DMEK.
   5. Neither DSEK or DMEK should significantly change the anterior corneal astigmatism; however, both can alter posterior corneal astigmatism (less so for DMEK) and this should be factored in when planning toric IOL implantation.5

If there is still refractive error after AT-IOL and DSEK/DMEK, these eyes are generally well suited to enhancement procedures with LASIK, PRK, LRI, etc.

References
Endothelial Disease: Why Descemet-Stripping Automated Endothelial Keratoplasty Is Better

Mark S Gorovoy MD

I. Risk / Benefit
   A. Photo of penetrating keratoplasty (PKP)
   B. Another PKP photo
   C. Specific to procedure
   D. Specific to surgeon
   E. How to quantify and assign a numerical value?
      1. Risk: Is the risk double if the primary failure rate doubles from 2% to 4%?
      2. Benefit: Is 20/20 twice as good as 20/40?
      3. Is plano = +5-10 D if both 20/20?
   F. It is not numeric, but just a gestalt value that helps
guides clinical decisions.
   G. Visual level that initiates surgical decision may be
best comparative index.
   H. Example: 20/25 cataract surgery

II. Risk: Operative
   A. Technical difficulty: DLEK never popularized
   B. Eye anatomy; difficult eyes (ie, no iris)
   C. Eye anatomy (show difficult case slides)
   D. Risk: surgeon
      1. Learning curve
      2. Volume – catch 22
      3. Skill level: free throws

III. Risk / Benefit
   A. Despite conclusions of Coster et al, DSAEK is so
superior to PK in terms of risk/benefit that it has
become the standard for endo disease.
   B. “The visual results and safety are so superior,
repeating DSAEK every year is better than PK”
(Terry, Gorovoy, Price, 2005; DC Cornea Congress;
private conversation).
   C. Proof
      1. PK: 20/70
      2. DSAEK: 20/25

IV. DSAEK vs. DMEK

V. DMEK Benefit
   A. BSCVA
   B. Refractive shift
   C. Smaller incision
   D. Aberrations
   E. Rejection
   F. Cell loss

VI. BSCVA
   A. 20/20: 40% vs. 20%
   B. Difference less over time
   C. Average: 20/25 vs. 20/30
   D. Time: 2-4 weeks vs. 6-8 weeks
   E. Conclusion: Slight advantage DMEK

VII. Refractive Shift
   A. 75 D vs. 1.25 D
   B. Tighter range
   C. Conclusion: slight advantage DMEK

VIII. DMEK Advantages
   A. Smaller incision: 2.4 vs/ 3.2
   B. Less aberrations: ??? most are anterior
   C. Less rejection: 1% vs. 3%
   D. Cell loss: 35% vs. 20%
   E. Conclusion: slight advantage DMEK

IX. Risks
   A. Learning curve – 50 cases
   B. Primary failure and rebubbles
   C. Volume to maintain skill
   D. Donor issues

X. Donor Peeling
   A. Difficult!!!
   B. Time to peel
   C. Greater loss of tissue
   D. Older donors
   E. Eye bank costs: loss of tissue and increased charges?
   F. Conclusion: significant advantage DSAEK

XI. Primary Failure and Rebubbling
   A. 5% (10%*) vs. 2% failure
   B. 5% (33%*) vs. 4% rebubble
   C. Conclusion: slight advantage DSAEK after learning
curve (*huge before)
XII. Specific Eye Risks
   A. DMEK requires normal ant. seg
      1. Miotic pupil
      2. No prior vitrectomy
      3. No aphakia
      4. No AC IOL
   B. Conclusion: large advantage DSAEK

XIII. Risk/Benefit Conclusion
   A. Risks greater until surgery is mastered.
   B. Low-volume surgeons may never overcome learning curve.
   C. This volume number is further reduced by the eyes that are not good candidates.
   D. Donor peel risks mostly solved by eye bank for a cost.
   E. Benefits of improved visual acuity are real, but only incrementally.
   F. Perioperative risks > DMEK
   G. Long term - to be determined

XIV. Final Conclusion
   A. Equal surgical bar 20/25
   B. Clear advantage in certain eyes for DSAEK
   C. Goal = 20/happy
Endothelial Disease: Why Descemet Membrane Endothelial Keratoplasty Is Better

Friedrich E Kruse MD

Penetrating keratoplasty has been the gold standard for corneal surgery for than 100 years. More recently it became possible to perform a layer-specific substitution for corneal diseases: several techniques have been developed allowing selective replacement of diseased structures of the cornea.

In North America and Europe disorders of the endothelium make up for more than one-third of all corneal grafts, and the outcome of penetrating grafts was often unsatisfactory. Several new techniques have been described for selective replacement of diseased endothelial cells together with the Descemet membrane (DM) (see Figure 1). In principle, Descemet-stripping automated endothelial keratoplasty (DSAEK) (A)1-3 replaces the host’s diseased endothelium and DM by a thin piece of corneal stroma with healthy endothelium and DM measuring between 150 and 50 µm in thickness. In contrast, Descemet membrane endothelial keratoplasty (DMEK) (B)4 or DMEK with stromal support (C),5,6 as well as Descemet membrane endothelial transfer,7 only replaces the host’s diseased tissue with healthy DM and endothelium.

Figure 1. Endothelial keratoplasty techniques: (A) Descemet-stripping automated endothelial keratoplasty (DSAEK). (B) Descemet membrane endothelial keratoplasty (DMEK). (C) Descemet membrane endothelial keratoplasty with a stromal rim (DMEK-S).

DMEK is better than DSAEK.

In the recent past several studies have been performed that clearly show that DMEK is superior to DSAEK. Several aspects such as safety, visual acuity, and optical properties have been investigated:

DMEK is safer than DSAEK.

Among the many arguments for DMEK the issue of safety seems to be most convincing: In a landmark study Price and coworkers have shown that the risk of immunologic graft rejection is almost negligible after DMEK in comparison to both PK and DSAEK.16,17

DMEK renders better visual acuity.

Although it is known that functional results of DSAEK are superior to PK, it soon became apparent that graft thickness has an impact on visual acuity and that thicker grafts do not perform as well as thinner grafts.18-20 We have recently performed a direct comparison between DSAEK and DMEK that clearly shows the superiority of thinner grafts in DMEK in regard to visual acuity.21 As shown in Figure 2, patients achieved significantly better visual acuity 3 and 6 months after DMEK surgery when compared to DSAEK.

Figure 2. Comparison of visual acuity (logMAR) 3 and 6 months after DSAEK (open bars) and DMEK (grey bars) (n = 40 in each group). Patients after DMEK showed statistically significant better visual acuity(s).
DMEK renders better optical quality of the inner surface of the cornea.

There are several possible explanations for the fact that DMEK renders better functional results than does DSAEK. One possibility is that DMEK renders an optimal physiological posterior corneal curvature with low higher-order aberrations, as shown by us in a recent study (see Figure 3). Also a disparity of the corneal curvature of the donor and the recipient in DSAEK could cause light scattering.

The Use of DMEK

In the light of the superiority of DMEK, this type of surgery gains more widespread attention. It is not only applicable to simple situations such as Fuchs dystrophy but also in complicated situations such as failed penetrating grafts, or eyes with anterior chamber, scleral-sutured, or iris-sutured IOLs. A triple procedure (DMEK combined with cataract surgery) represents an effective and safe treatment choice for patients with endothelial dystrophy and cataract.

At present the significant advantages of DMEK seem to be balanced by disadvantages regarding preparation, surgical manipulation, and early postoperative performance. The latter is exemplified by a higher need for postoperative air injections caused by partial detachment of the DM. In order to allow a more widespread use of DMEK it is necessary to standardize the technique, which requires a systematic analysis of the variables associated with the surgical technique.

References


Anterior Corneal Disease: Why Penetrating Keratoplasty Is Better

Mark J Mannis MD

I. The New Surgical Paradigm
   A. Endothelial disease + normal stroma = endothelial keratoplasty
   B. Anterior corneal/stromal disease + normal endothelium = deep anterior lamellar keratoplasty
   C. Anterior/stromal disease + endothelial dysfunction = penetrating keratoplasty
   D. Corneal trauma = either penetrating keratoplasty or deep anterior lamellar keratoplasty

II. Effective Decision Making
   A. Where is the disease?
   B. What procedure will provide the patient with the appropriately rapid recovery?
   C. What are the patient’s visual needs?

III. What are the comparative outcomes in the literature?
   A. Are the visual outcomes comparable?
   B. Is graft longevity comparable?

IV. What are the technical challenges?
   A. Knowing when to convert
   B. Knowing when not to convert
Anterior Corneal Disease: Why Deep Anterior Lamellar Keratoplasty Is Better

Shigeto Shimmura MD

Introduction

Deep anterior lamellar keratoplasty (DALK) is a surgical technique that replaces diseased stromal tissue in eyes with healthy corneal endothelium. The lack of endothelial rejection and a lower risk of endophthalmitis are some of the advantages of DALK over penetrating keratoplasty (PK), and an increasing number of corneal surgeons are reverting to DALK for the treatment of stromal disease. Yet despite the clear benefits of DALK, it is still not the first choice of surgery for many surgeons due to the difficult skills and longer surgery time required compared to PK. Furthermore, a recent report by Coster et al showed that DALK was associated with worse graft survival and visual acuity compared to PK. Therefore, there are still controversies concerning the evidence of the benefits of DALK over PK.

Why Choose Deep Anterior Lamellar Keratoplasty?

Many publications have shown that rates of graft survival and visual acuity are similar between DALK and PK, while long-term results for DALK may still be lacking. PK also has a long and successful track record for common diseases such as keratoconus. Our own experience also shows that the only difference between the two procedures is the significantly greater loss of endothelial cells after PK over the long term, while there was no difference in graft survival. Therefore, the strongest support for choosing DALK would be for cases that are relatively contraindicated for PK. These include cases with anterior surface disease with or without stem cell dysfunction, or high-risk cases with vascularized corneas. Another reason to choose DALK over PK would be in cases where compliance to eye drop use is expected to be poor, since steroid drops can be tapered earlier in DALK.

Selected Readings

Limbal Stem Cell Disease: When Is Ocular Surface Transplantation Better?

Edward J Holland MD

I. KPro: Caution in Severe Ocular Surface Disease (OSD)
   A. Inability to measure IOP accurately
   B. Complication rate higher in severe OSD: Corneal melts, infectious keratitis, endophthalmitis
   C. Complications can lead to loss of eye.

II. Retention of KPro: Worse in OSD
   A. Of 28 KPros implanted in 23 eyes with limbal stem cell disease (LSCD)
   B. Explant rate of 25%
   C. 5 in Stevens-Johnson syndrome (SJS), out of 6 SJS patients
   D. 1 in chemical injury
   E. 1 in ocular cicatricial pemphigoid (OCP)

III. Ocular Surface Transplant Advantages in OSD
   A. Achieves good results
   B. Can be used for severe conjunctival disease (symblepharon, conjunctival inflammation)
   C. Accurate IOP monitoring
   D. Complications result in loss of surface only, not loss of the eye
   E. Can always default to a KPro

IV. Why do cornea specialists avoid ocular surface transplantation (OSTx)?
   A. Main barrier is the fear of use of systemic immunosuppression (IS).
      1. Lack of knowledge of how to manage IS
      2. Fear of IS side effects

V. OSTx with systemic immunosuppression is safe.
   A. 136 patients (225 eyes) received systemic IS from 1997 to 2007 for OSTx.
   B. Mean follow-up: 4.6 years
   C. 75% of patients on tacrolimus, mycophenolate mofetil, prednisone sparing
   D. Adverse events
      1. No deaths, no secondary tumors
      2. Three severe events in 2 patients (1.5%; 2 MI, 1 PE)
   E. Previous studies of adverse events attributed to IS have only been in organ transplant patient.

VI. The “Cincinnati Procedure”: Combined Living-Related Conjunctival Limbal Allografts and Keratolimbal Allografts (LR-CLAL/KLAL) for Severe Conjunctival and Limbal Disease
   A. 24 eyes: preop BCVA < 20/400 in 87.5%
   B. 19 SJS, 2 OCP, 2 chemical injuries, 1 atopic keratoconjunctivitis
   C. 80% had staged keratoplasty after OSTx.
   D. 75% had stable surface mean.
   E. 71% achieved 20/125 or better.
   F. Mean follow-up: 3.5 years

VII. Conclusion: OCT vs. KPro for OSD
   A. In the management of OSD: Surgeons should become accomplished with both OSTx and KPro procedures for the best outcomes.
   B. Preference is OSTx for the primary procedure in the majority of patients.

VIII. Why I Prefer OSTx Over KPro for OSD
   A. With KPro
      1. Follow-up is frequent forever.
      2. Inability to accurately monitor IOP
      3. High complication rate with severe OSD
      4. Complications can lead to permanent loss of vision.
         a. Glaucoma
         b. Endophthalmitis
   B. With OSTx
   C. Good success rate
   D. Systemic immunosuppression is well tolerated and safe.
   E. When stable, the follow-up is not frequent.
   F. Able to monitor IOP
   G. If ocular surface stem cell transplantation fails:
      1. Loss of surface, not permanent loss of vision
      2. Can always perform subsequent KPro
Limbal Stem Cell Disease: When Is Keratoprosthesis Better?

Clara C Chan MD

I. Types of Keratoprotheses
   A. Boston Type 1 Keratoprosthesis
   B. Boston Type 2 Keratoprosthesis
   C. Osteo-odonto-keratoprosthesis
   D. Alphacor keratoprosthesis
   E. KeraKlear keratoprosthesis (not FDA approved)

II. Advancements in Design and Innovations Related to the Boston Type 1 Keratoprosthesis
   A. Contact lens use to improve hydration of the corneal tissue
   B. Backplate holes to improve aqueous contact and to decrease tissue necrosis
   C. Postoperative topical vancomycin (14 mg/ml with benzalkonium chloride) to reduce Gram-positive endophthalmitis
   D. Titanium blackplate to reduce retroprosthetic and retro-backplate membrane formation (and corneal melt)
   E. Blue and brown coloring of the titanium backplate
   F. Role of corneal crosslinking for the vehicle cornea to reduce corneal melt and infection?
   G. Role of antifungal prophylaxis?

III. Treatment Algorithms in Limbal Stem Cell Disease
    Summary of discussions from the Cornea Society Limbal Stem Cell Disease Working Group

IV. Limbal Stem Disease and the Boston Keratoprosthesis Type 1
    A. Issues regarding status of the ocular surface
       1. Severe dry eye
       2. Keratinized ocular surface
       3. Symblepharon or lid abnormalities
       4. Ability to tolerate contact lens wear
    B. Decision-making process
       1. Older patients
       2. Binocular legally blind
       3. Unable to tolerate systemic immunosuppression
       4. Non-candidate for limbal stem cell transplantation procedure
       5. Presence of active ocular issues such as lid abnormalities or glaucoma
    C. Case examples
       1. Aniridia
       2. Aniridia fibrosis syndrome
       3. Hypotony
       4. Failed corneal graft
       5. Failed limbal stem cell transplant
       6. Stevens-Johnson syndrome
       7. Chemical burn
       8. Ocular cicatricial pemphigoid / mucous membrane pemphigoid
       9. Graft versus host disease

V. General Complications of the Boston Keratoprosthesis Type 1
   A. Glaucoma
   B. Retroprosthetic membrane
   C. Retinal detachment
   D. Infectious keratitis
   E. Infectious endophthalmitis
   F. Stromal necrosis and extrusion
   G. Sterile vitritis
   H. Wound rupture

VI. Complications of the Boston Keratoprosthesis Type 1 Related to Limbal Stem Cell Disease
   A. Fungal infectious keratitis and endophthalmitis
   B. Stromal necrosis and extrusion
   C. Role of contact lens intolerance
   D. Glaucoma
   E. Retroprosthetic membrane (Aniridia is independent risk factor.)

VII. Summary: When is KPro better?
    A. Prior corneal graft failure and high risk for repeat graft failure
    B. Older patient
    C. No severe dry eye
    D. No chronic conjunctival inflammation or autoimmune disease
    E. Intact fornix and able to tolerate contact lens wear
    F. Able to comply with lifelong follow-ups and topical antibiotic use
References


3. Greiner MA, Li JY, Mannis MJ. Longer-term vision outcomes and complications with the Boston type 1 keratoprosthesis at the University of California, Davis. *Ophthalmology* 2011; 118:1543-1550.


Case #1

*Shahzad I Mian MD*

A 76-year-old male presented with gradual decline in vision in the right eye more than the left, with difficulty with glare at night while driving. He also noted increased difficulty with reading until noon. He has a history of cataract surgery in both eyes, dry AMD, and Fuchs dystrophy diagnosed several years ago. He has tried sodium chloride 2.5% drops without significant improvement.

On examination, the BCVA was 20/60 in the right eye and 20/50 in the left eye. There were diffuse confluent corneal guttae with mild corneal edema and posterior chamber IOLs. The patient elected to undergo Descemet-stripping automated endothelial keratoplasty in the right eye. A precut tissue was obtained from the eye bank for the surgery and placed using a tissue injector. The patient tolerated the procedure well and the vision gradually improved to 20/25 by 1 month. At this visit, an 1-mm peripheral interface opacity was noted. The donor rim culture was positive for *Candida glabrata*. Over the next 3 weeks, the patient developed FBS and the vision decreased to 20/50. The peripheral interface opacity increased to 2 mm with new KP and an anterior chamber cellular reaction. Topical amphotericin and oral voriconazole was started. The posterior corneal disc was removed, and intracameral amphotericin was given. The cultures from the posterior corneal disc and the infiltrate grew *Candida glabrata*. After treatment for 2 months, a posterior corneal disc was placed. The vision improved to 20/25 over 1 year of follow-up.

Fungal keratitis is uncommon after keratoplasty but maybe more common after endothelial keratoplasty. Interface infections can be difficult to treat and often require surgical intervention. Treatment options include removal of posterior corneal disc, repeat EK, or penetrating keratoplasty. Vision can recover especially with early treatment.
Case #2

Carol L Karp MD

A conjunctival lesion will be presented, with emphasis on new imaging techniques and management options.

NOTES
Case #4
Making It Easy: Approaches to Treating Pediatric Limbal Dermoids

Jennifer Y Li MD

Case Report
A 10-year-old male was referred from pediatric ophthalmology for evaluation of a limbal dermoid in the right eye. According to his parents, the lesion has been present since birth, but it appears to be causing increasing amounts of cylinder in his spectacles. Moreover, the appearance of the lesion is beginning to cause some social distress for the child. They are interested in exploring options for excision that will be as minimally disruptive as possible for this active child.

Discussion
I. Epibulbar Dermoids
A. Congenital choristoma
1. Develops at 5-10 weeks gestational age1
2. Collagenous connective tissue with overlying stratified squamous epithelium2
May have associated hair, dermal appendages, fat, nerves, lacrimal tissue
B. Variable inheritance pattern
1. Associated with:
   a. Goldenhar syndrome
   b. Epidermal nevus syndrome
C. Incidence between 1/10,000 to 3/10,0003
II. Clinical Findings
A. Unilateral or bilateral
B. Majority (~80%) in inferotemporal quadrant of the eye1,3
C. Solid tumor
1. Hard, rubbery, or soft
2. Color: white, yellow, pink
3. Smooth or rough, ± fine hair
D. Grading scale
1. Grade I: superficial; < 5 mm; limbal location
2. Grade II: deep, but not involving Descemet membrane; extending over cornea
3. Grade III: involves entire cornea, extends into anterior chamber (least common)
E. Ocular associations: staphyloma, microcornea, ptosis, microphthalmia, corneal anesthesia, strabismus, ocular colobomas2
III. Medical Management (Grade I Lesions Only)
A. Spectacle correction
B. Close observation
C. Management of amblyopia
IV. Indications for Surgery
A. Grade II or Grade III lesions1,3
   1. Grade II may require penetrating keratoplasty.3
   2. Unilateral Grade III are frequently enucleated or eviscerated.3
B. Amblyopia refractory to medical management
C. Chronic irritation
D. Recurrent conjunctivitis
E. Growth into pupillary or visual axis
F. High astigmatism or irregular astigmatism
G. Progressive dellen
H. Cosmesis
I. Inadequate lid closure
V. Surgical Management
A. Simple excision4-6
   1. Complications may include persistent epithelial defect, corneal vascularization, scarring.4
   2. Corneal tattooing may improve cosmetic appearance.5
   3. Perforations can be associated with deep dissections.7
B. Lamellar keratoplasty
   1. Effective with good cosmetic appearance
   2. ± reduction of astigmatism7,9 (Astigmatism may increase in some cases.6)
   3. May not improve visual acuity7,8
   4. Requires subsequent suture removal
C. Amniotic membrane transplantation
   1. Multilayer application with fibrin glue ± suture10,11
      a. Rapid recovery with minimal patient discomfort
      b. Remodeling allows for good cosmetic appearance.
   2. Adjuvant use of MMC12; possible prevention of pseudopterygium
References


Case #4  
Candida parapsilosis: The Enemy Within?  
José Alvaro Pereira Gomes MD PhD, Rodrigo Antonio Brant Fernandes MD, Ana Luisa Höfling-Lima MD PhD  

CASE REPORT  

First visit: 06/20/2011  

Identification/Main Complaint  
A female patient, 52 years old, from Rio de Janeiro, Brazil, came complaining of decreased visual acuity (VA) O.U. for the last year.  

Previous History  
Open-angle glaucoma for 10 years. Underwent multiple surgeries: 3 PK O.D. and 2 PK O.S. One glaucoma drainage device implantation O.U. (Ahmed); oculoplastic surgeries OU for lagophthalmos.  

Ophthalmic Exam  
BCVA: O.D. = 20/400 and O.S. = CF 2 m, with high IOP O.U. (O.D. = 35 mmHg and O.S. = 24 mmHg). At the slitlamp exam we observed bilateral lagophthalmos, conjunctival hyperemia, and corneal grafts presenting mild edema and inferior corneal keratinization O.U., with corneal melting; shallow AC with anterior synechiae O.D.; tube shunt exposure through the conjunctiva superiorly O.U.  

Management and Follow-up  
Started oral and topical antiglaucomatous medication, oral doxycycline, preservative-free lubricants and topical antibiotic / steroid combination. Additionally, the patient underwent oculoplastic surgery for lagophthalmos correction O.U.; corneal patch O.D.; and pars plana vitrectomy (PPV) with scleral patch and tube shunt reposition from the anterior chamber (AC) to the pars plana O.U. The patient developed persistent epithelial defect on the corneal patch O.D., which was handled with the use of bandage contact lens and topical prophylactic antibiotic.  

On May 5, 2012, IOP O.U. was 26 mmHg, even with maximum antiglaucomatous medication; it was decided to perform endocyclophotocoagulation 180° O.U. followed by a new PK and pupilloplasty, which were performed with a good outcome. During the procedure on O.D., a suspicious corneal infiltrate was noticed, which was scraped and sent for culture. After 7 days, it came back positive for Candida orthopsilosis, a species of the Candida parapsilosis complex. Patient was treated topically with amphotericin-B 0.15% and systemically with itraconazole.  

After 1 month, vision was 20/400p O.D. and 20/200 O.S.; IOP was 7 mmHg O.D. and 13 mmHg O.S. Funduscopy revealed macular edema O.D. and cup-to-disc ratios of 0.6 O.U. In the following months, the patient complained of decreased VA O.D.; slitlamp exam revealed clear corneal graft O.U. with the presence of large pigmented keratic precipitates (KP) and AC reaction ++/+++ O.D.; IOP was 4 mmHg O.D. and 13 mmHg O.S. OCT demonstrated macular edema and epiretinal membrane O.D. It was decided to perform PPV and epiretinal membrane removal O.D.  

Figure 1. O.D. presenting a suspicious corneal infiltrate on the corneal patch, which was scraped and sent for culture. Returned positive for Candida orthopsilosis.  

Figure 2. Four months postop of repeated PK, presenting a clear corneal graft with the presence of large pigmented KP and AC reaction in O.D.
During the PPV procedure, the presence of 2 small white/yellow elevated infiltrates at the periphery of the retina, together with lesions at the pars plana similar to snowballs, was noted. A vitreous tap and biopsy were performed and sent for microbiological analysis.

The result was positive for Candida orthopsilosis (sensitive to amphotericin-B, fluconazole, and voriconazole) and Bacillus circulans. It was decided to treat the patient with systemic and IV voriconazole 50 µg/0.1 ml, maintaining the use of topical amphotericin q.i.d. Patient was followed with ultrasound, which revealed small peripheral choroidal detachment, and funduscopy, which showed improvement of the white lesions. VA was CF 1m O.D. and 20/200 O.S.; IOP was 2 mmHg O.D. and 12 mmHg O.S.

The same treatment was repeated twice, with microbiological evidence of Candida orthopsilosis in both vitreous taps. It was then decided to remove the tube shunt and IOL together with capsular bag O.D., perform another PPV with epiretinal/intimal limiting membrane removal and silicone oil tamponade, and vitrectomy (IOL, capsular bag, and vitrectomy) and for Streptococcus viridans (tube shunt and Tenon filtering capsule) sensitive to azithromycin, which was administered orally.

Patient developed worsening of the graft transparency, with calcium deposit and epithelium defect inferiorly, but without signs of intraocular inflammation O.D. On June 18, 2014, a new vitreous tap with IV voriconazole injection was performed. Microbiological analysis came back negative this time. VA was HM O.D. and 20/100 O.S. OCT presented macular edema O.D.

![Figure 3](image)

**Figure 3.** Last visit follow-up: Four months postop of VVP, IV voriconazole and IOL/tube shunt removal O.D. Note worsening of the graft transparency, with calcium deposit and epithelium defect inferiorly, but without signs of intraocular inflammation.

**Discussion**

- Be careful in reintroducing exposed tube shunts from a glaucoma drainage device implanted previously from the AC into the vitreous cavity.
- Candida parapsilosis complex can produce biofilm and cause endophthalmitis resistant to conventional treatment. When associated with bacterial infection, it can be protected by a mixed biofilm, which may make treatment even more difficult.
- Management includes removal of all intraocular biomaterials/IOL, tube shunts, etc.) and the use of intravitreous and systemic antifungal agents as voriconazole.
- And now: Would you recommend another surgical procedure for this patient? Another PK vs. KPro? Secondary IOL implantation? PPV + membrane peeling?
- What about intraocular use of steroid?

**Selected Readings**


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Clara C Chan MD  
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Minas T Coroneo MD MS  
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Ana Luisa Höfling-Lima MD MBA  
None  

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Marian Sue Macsai-Kaplan MD  
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Ramana S Moorthy MD  
None  

Venkatesh Prajna  
Namperumalsamy MBBS  
None  

Stephen C Pflugfelder MD  
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Francis W Price Jr MD
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Christopher E Starr MD
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Donald Tan MD FRCS FRCOphth
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