The American Ophthalmological Society

ONE HUNDRED AND FIFTY-FIRST ANNUAL MEETING

Richard P. Mills .............................................................. PRESIDENT
Thomas J. Liesegang.................................................. EXECUTIVE VICE PRESIDENT
Emily Y. Chew ............................................................ EDITOR OF THE TRANSACTIONS

COUNCIL
Jay C. Erie, Chair
M. Edward Wilson, Jr.
Anne L. Coleman
Woodford S. Van Meter
Marco A. Zarbin

MAY 14–17, 2015
THE HOTEL VIKING
NEWPORT, RHODE ISLAND
THE ONE HUNDRED AND FIFTY-FIRST ANNUAL MEETING

of the Society will be held at

The Hotel Viking
Newport, Rhode Island
Thursday through Sunday
May 14–17, 2015

COMMITTEE ON PROGRAMS
Edward G. Buckley, Chair
J. Sebag
David T. Tse
Eduardo C. Alfonso
TARGET AUDIENCE
Ophthalmologists involved in academic practice, private practice, training programs, research, or administrative health care.

MEETING OBJECTIVES
The objectives of the 2015 Annual Meeting are to:

1. Discuss important new advances in the prevention, cause, diagnosis, and treatment of eye diseases.
2. Identify basic and clinical vision research that can be transformed into improved clinical care.
3. Assess the role of new technologies in the evaluation and treatment of eye diseases.
4. Describe factors that impact the effective delivery of the highest quality eye care for the public.
5. Identify clinical, scientific, and ethical issues confronting the profession.
6. Demonstrate new educational approaches to training ophthalmologists.
7. Describe the economic impact of governmental initiatives on the delivery of ophthalmic care.

FDA STATUS DISCLAIMER
Some material on recent developments may include information on drug or device applications that are not considered community standard, that reflect indications not included in approved FDA labeling, or that are approved for use only in restricted research settings. This information is provided as education only so physicians may be aware of alternative methods of the practice of medicine, and should not be considered endorsement, promotion, or in any way encouragement to use such applications. The FDA has stated that it is the responsibility of the physician to determine the FDA status of each drug or device he or she wishes to use in clinical practice, and to use these products with appropriate patient consent and in compliance with applicable laws.

The Society provides the opportunity for material to be presented for educational purposes only. The material represents the approach, ideas, statement, or opinion of the presenter and/or author(s), not necessarily the only or best methods or procedure in every case, nor the position of the Society. The material is not intended to replace the physician’s own judgment or give specific advice for case management. The Society specifically disclaims any and all liability for injury or other damages of any kind for any and all claims that may arise out of the use of any technique demonstrated or described in any material by any presenter and/or author(s), whether such claims are asserted by a physician or any other person.

FINANCIAL DISCLOSURE
The financial disclosures of all presenters, authors, Council members, and members of the Committee on Programs are listed on pages VII-XI in the program book. Conflicts, if noted, were managed prior to the presenter being included in the program. If the presenter has a financial disclosure related to the specific presentation, the disclosure will be stated verbally and presented on the first slide of their presentation. Audience participants are required to state their financial disclosure before they join a discussion of a paper or poster.
PARTICIPATION AND CONSENT TO BE RECORDED
Parts of the 2015 Annual Meeting will be recorded for subsequent posting on the Society’s website, including discussion. Approaching the microphone to discuss a presentation is considered implicit consent to the participant’s discussion being included in this recording. Attendees who do not wish to be recorded should refrain from approaching the microphone.

REGISTRATION
Members and guests should check in at the AOS registration desk on the first day of the meeting to receive meeting information. Those who have not pre-registered and paid their registration fees or dues may do so at this time. Registration will be available at the following times:

- Thursday, May 14: 1:30 PM – 5:00 PM
- Friday, May 15: 6:30 AM – 12:00 PM
- Saturday, May 16: 6:00 AM – 12:00 PM
- Sunday, May 17: 6:30 AM – 10:00 AM

ACCREDITATION STATEMENT
This activity has been planned and implemented in accordance with the Essential Areas and policies of the Accreditation Council for Continuing Medical Education through the joint providership of the American Academy of Ophthalmology and The American Ophthalmological Society. The American Academy of Ophthalmology is accredited by the ACCME to provide continuing medical education for physicians.

CREDIT DESIGNATION STATEMENT
The American Academy of Ophthalmology designates this live activity for a maximum of 10.75 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

BYLAWS
The following Bylaws are published each year in the program as a reminder to the members of the Society:

ARTICLE IX, Section 3 – Any member who shall be absent from meetings for three consecutive years without acceptable excuse shall be dropped from the roll, except for Honorary Members, Emeritus Members, Members of twenty years standing or those then serving in the armed forces. An excuse for absence is acceptable only when a member is ill, or when there is illness of a member of his or her immediate family, and may not be considered approved until received in written form and acted upon by the Council. The Council shall have the authority to approve other excuses only upon a finding of exceptional circumstances. This Bylaw shall be printed in every call for the Annual Meeting.
MEMBERS ELECTED AT THE 2014 MEETING

Massimo Busin*  
J. Douglas Cameron  
Clement Chan  
Raymond Douglas  
Harminder Singh Dua  
Tamara Fountain  
Jost Jonas*  
Gregg Kokame  
Baruch Kuppermann  
Alex Levin**  
David Sarraf  
Donald Tan*  
Stephen Tsang  
Janey Wiggs

*Provisional Members  
**Attended as Provisional Member in 2014

IN MEMORIAM
The Executive Vice President has received notice of the deaths of the following members during the past year:

William H. Annesley, Jr.  
Jose A. Berrocal  
George O. Waring, III  
Barbara W. Streeten  
Ronald M. Burde

Bryn Mawr, Pennsylvania  
Santurce, Puerto Rico  
Atlanta, Georgia  
Syracuse, New York  
Longboat Key, Florida  
Joined 1980  
Joined 1980  
Joined 1989  
Joined 1982  
Joined 1983

FUTURE ANNUAL MEETINGS
2016 AOS Annual Meeting
The Broadmoor Hotel
Colorado Springs, Colorado
May 19–22, 2016
FINANCIAL DISCLOSURES

The following are the financial disclosures of those involved in the preparation or presentation of this AOS event, some of which are commercial interests. A commercial interest is any entity producing, marketing, re-selling, or distributing health care goods or services consumed by, or used on, patients. The AOS Committee on Programs gathered this information to plan the program and has attempted to manage relevant conflicts of interest to present a balanced program. The presenter will indicate on the first slide and verbally at the beginning of the talk, if any of the financial disclosures listed has a relationship to the specific presentation. Participants that might speak from the floor are requested to state their financial disclosures before they speak.

<table>
<thead>
<tr>
<th>Category</th>
<th>Code</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Consultant/Advisor</td>
<td>C</td>
<td>Consultant fee, paid advisory boards or fees for attending a meeting (for the past 1 year)</td>
</tr>
<tr>
<td>Employee</td>
<td>E</td>
<td>Employed by a commercial entity</td>
</tr>
<tr>
<td>Lecture Fees</td>
<td>L</td>
<td>Lecture fees (honoraria), travel fees or reimbursements when speaking at the invitation of a commercial entity (for the past 1 year)</td>
</tr>
<tr>
<td>Equity Owner</td>
<td>O</td>
<td>Equity ownership/stock options of publicly or privately traded firms (excluding mutual funds) with manufacturers of commercial ophthalmic products or commercial ophthalmic services</td>
</tr>
<tr>
<td>Patents/Royalty</td>
<td>P</td>
<td>Patents and/or royalties that might be viewed as creating a potential conflict of interest</td>
</tr>
<tr>
<td>Grant Support</td>
<td>S</td>
<td>Grant support for the past 1 year (all sources) and all sources used for this project if this form is an update for a specific talk or manuscript with no time limitation</td>
</tr>
</tbody>
</table>

ALBUQUERQUE, Romulo
P - University of Kentucky

ALFONSO, Eduardo C.
C - Bio Tissue*; Foresight*

AMBATI, Jayakrishna
P - University of Kentucky

ASBELL, Penny A.
C - Alcon; Contact Lens Association of Ophthalmologists, Inc.; Paragon; Senju; Valeant
S - Senju
L - Oasis; Oculus; R-Tech Ueno; UPenn

AZAR, Dimitri T.
C - Forsight Labs
E - Novartis
O - Novartis

BAKRI, Sophie
C - Allergan; Genentech

BERNSTEIN, Audrey
S - Research to Prevent Blindness; The Moses Y. Safra Family U.S. Charitable Foundation, Inc.

CAPRIOLI, Joseph
C - Allergan
S - Allergan; Alcon Laboratories, Inc.; New World Medical, Inc.; National Institute of Health/National Eye Institute; Research to Prevent Blindness

CHAN, Clement
C - Allergan
S - Acucela; Genentech-Roche; National Eye Institute; Regeneron
CHEN, Teresa  
S - Agency for Healthcare Research and Quality; American Glaucoma Society Mid-Career Award; Fidelity Charitable Fund; Harvard Catalyst Grant, National Institutes of Health Award UL 1RR 025758; Massachusetts Lions Eye Fund

CHIANG, Michael  
C - Clarity Medical Systems (unpaid member of Scientific Advisory Board)  
S - National Institutes of Health; Research to Prevent Blindness

COLEMAN, Anne L.  
L - Allergan

ELNER, Susan G.  
S - National Eye Institute; Santen Pharmaceutical  
P - Ocuscience

GARDNER, Thomas W.  
C - Aerpio; BetaStem; Johnson & Johnson; Kalvista; Novo Nordisk

GILLESPIE, Stephanie R.  
S - The Moses Y. Safra Family U.S. Charitable Foundation, Inc. and Research to Prevent Blindness

HARTNETT, Mary Elizabeth  
C - Knights Templar Eye Foundation; National Eye Institute  
S - National Eye Institute; March of Dimes  
L - Kresge Eye Institute; Medical College of Wisconsin, Milwaukee; University of Oklahoma Department of Ophthalmology; Women in Ophthalmology  
P - Lippincott Wolters Kluwer

HAUSER, Michael A.  
S - National Institutes of Health/ National Eye Institute

HRIBAR, Michelle R.  
S - National Library of Medicine

HUANG, David  
O - Optovue, Inc.*  
P - Optovue, Inc.; Carl Zeiss Meditec*  
S - Optovue, Inc.*

JAIN, Atul  
C - Allergan; Regeneron  
L - Regeneron

JAMPEL, Henry D.  
C - Endo Optiks; ForSight; Ivantis; Transcend  
O - Allergan

JIA, Yali  
P - Optovue, Inc.*  
S - Optovue, Inc.*

JOHNSON, Chris A.  
C - Centervue; Ivantis

JOHNSON, Mark W.  
C – GlaxoSmithKline; Oraya

JONAS, Jost  
C - Alimera; Allergan; Boehringer Ingelheim; Merck, Sharpe & Dohme; Pfizer; Sanofi  
P - CellMed Alzenau

KANG-MIELER, Jennifer J.  
C - Genentech

KETTERLING, Jeffrey  
P - Riverside Research  
S - Riverside Research

KOKAME, Gregg  
C - Allergan; Santen; Zeiss  
L - Thrombogenics; Regeneron  
S - Regeneron; Genentech*

KRUEGER, Ronald  
C - Alcon; Clarity; LensAR  
O - LensAR

KRUEGER, Ronald  
C - Alcon; Clarity; LensAR  
O - LensAR
KUPPERMANN, Baruch
C - AcuFocus; Alcon; Allegro; Ampio; Aqua Therapeutics; Bausch & Lomb; Glaukos; Neurotech; Novagali; Novartis; SecondSight; Staar Surgical; Teva; Allergan; Genentech; Ophtotech; Regeneron
S - Allergan; Genentech; Ophtotech; Regeneron; ThromboGenics

ROBIN, Alan
C - Aerie Pharmaceuticals; Bio-light; Glauk; TEVA Pharmaceuticals

SADDA, Srinivas R.
C - Alcon; Novartis; Roche; Allergan; Carl Zeiss Meditec; Genentech; Optos, plc.
S - Allergan; Carl Zeiss Meditec; Genentech; Optos, plc.

LOMBARDI, Lorinna H.
E - Husband is employee of Genentech-Roche (not related to the study)

SADUN, Alfredo A.
S - Edison Pharmaceutical; Stealth Peptides (Pharmaceutical)

MIELER, William F.
C - Genentech

SARRAF, David
C - Genentech
S - Regeneron

MORRISON, John C.
S - National Institutes of Health

SEBAG, J.
C - Alcon*; ThromboGenics*
L - Alcon*; ThromboGenics*
P - Riverside Research, New York

NOURI-MAHDAVI, Kouroos
C - New World Medical; Allergan
L - Allergan
S - Heidelberg Engineering

SIATKOWSKI, R. Michael
C - American Board of Ophthalmology
S - National Eye Institute

OLSEN, Timothy W.
S - Abraham J. and Phyllis Katz Foundation; Emtech Biotechnology Grant; Fraser Parker Foundation; Genentech-Roche; Georgia Research Alliance; Minnesota Lions; National Institute of Health/National Eye Institute; National Institute of Health/National Institute on Aging; R. Howard Dobbs Jr. Foundation; Research to Prevent Blindness
P - Scleral Depressor; Tissue Support Structure; Postop Pain Formulation

SPAETH, Eric L.
P - Spaeth-Richman Contrast Sensitivity Test

SPAETH, George L.
C - Pfizer
O - Spaeth-Richman Contrast Sensitivity Test
S - Allergan; Glaucoma Service Foundation; Merck

RICHMAN, Jesse
P - Spaeth / Richman Contrast Sensitivity Test

STAMPER, Robert L
C - SightSciences; Transcend
O - SightSciences

RITCH, Robert
C - Aeon Astron; iSonic Medical; Mobius Therapeutics; Sensimed
L - Santen
P - Ocular Instruments

TAN, Donald
P - Network Medical Products
S - Alcon Labs; Bausch & Lomb; Carl Zeiss Meditec; Santen
Financial Disclosures

TERRY, Mark
P - Bausch & Lomb Surgical
S - Bausch & Lomb Surgical; Moria

TOMITA, Minoru
C - AcuFocus, Inc.; Avellino; Schwind Eye Tech Solutions; VSY Biotechnology; Ziemer Ophthalmic

TSAI, James C.
C - Aerie Pharmaceuticals; Amakem

VARSHNEY, Neeta
C - Applied Biomedical; Regeneron
L - Regeneron; Allergan
S - Regeneron

WAISBOURD, Michael
C - Pfizer
P - Spaeth-Richman Contrast Sensitivity Test
S - Allergan; Glaucoma Service Foundation; Merck

WANT, Andrew D.
S - Moses Y. Safra Family U.S. Charitable Foundation Inc.; Research to Prevent Blindness

WEIKERT, Mitchell P.
C - Ziemer, Inc.*

WIGGS, Janey
C - Genentech
S - Alcon; March of Dimes Foundation; National Institute of Health/National Eye Institute

WILSON, David
C - American Board of Ophthalmology

WILSON, M. Edward
P - Springer Books
S - National Eye Institute - Infant Aphakia Treatment Study; Omeros; Ophtec

WILSON, Steven
C - Cambium; Allergan; Amekam Corporation; Auven Therapeutics; Symic Corporation
L - Allergan
S - Amekam Corporation; Auven Therapeutics; Symic Corporation

WOLOSIN, J. Mario
S - The Moise and Chella Safra Award for Exfoliation Syndrome Glaucoma Research; National Eye Institute EY 014878; Research to Prevent Blindness, Inc.

YANNUZZI, Lawrence A.
L - Genentech*

ZARBIN, Marco A.
C - EyEngineering, Inc.; Genentech, Inc.; Helios, KK, Inc.; Makindus, Inc.; Novartis Pharma AG; Foundation Fighting Blindness, Inc.
L - Novartis Pharma AG
S - Foundation Fighting Blindness, Inc.; National Eye Institute
P - Rutgers University

*Relevant financial disclosures that underwent a conflict resolution review process by Edward G. Buckley, Chair of the Committee on Programs.
NO FINANCIAL RELATIONSHIPS TO DISCLOSE

ACHIM, Catherine
AFIFI, Abdelmonem
ALLEN, Keri F.
ALLINGHAM, Rand
ALMAN, Benjamin A.
ANDREWS, Chris A.
ANG, Marcus H.
ANSHU, Arundhati
ARCHER, Steven
ARNOLD, Anthony C.
AUGSBURGER, James J.
AUNG, Tin
AZARBOD, Parham
BALASUBRAMANIAM, Saranya C.
BARTLEY, George
BENAGE, Matthew J.
BOTITINI, Alexander
BROWN, Lewis M.
BROWN, Lisa
BUCKLEY, Edward G.
BULLOCK, John
CAMERON, John Douglas
CHAN, Lawrence
CHEN, Andrew
CHENG, Ching-Yu
CHOI, Catherine S.
CLARKSON, John G.
COATS, David K.
COLEMAN, D. Jackson
CORREA, Zelia M.
DAI, Yang
DAVIS, Ellen J.
DE LEON, Mark S.
EBERHART, Charles G.
EDMUNDS, Beth
ERIE, Jay C.
FRAUNFELDER, Frederick
GILLESPIE, Stephanie R.
GIOVINGO, Michael C.
GOGTE, Priyanka
GRAND, Gil
GRANNIS, Charity H.
GROSSNIKLAUS, Hans E.
HANYUDA, Akiko
HARK, Lisa
HARRINGTON, Kathleen
HERMAN, William H.
HONG, Jason
HORKAYNE-SZAKALY, Iren
HOYT, William F.
HSU, Chun-Wei
JAMPOL, Lee
JOSEPH, Anthony
KANG, Jae H.
KANG-MIELER, Jennifer J.
KENNEY, Cristina M.
KERR, Natalie C.
KHAMIS, Harry J.
KHOR, Chiea C.
KIRCH, Darrell G.
KNEPPER, Paul
KONG, Lingkun
LAKHANPAL, Vinod
LI, Yao
LIESEGANG, Thomas J.
LIU, Liang
MAMOU, Jonathan
MCCULLEY, Tim
MENDEZ, Amber
MORALES, Esteban J.
NANGIA, Vinay
NEWMAN, Steven
NGUYEN, Huy V.
PACKER, Samuel
PARSA, Cameron
PASQUALE, Louis
PAULOS, Michael
PAYSSE, Evelyn
RAHIMY, Ehsan
READ-BROWN, Sarah
REN, Ai
REPKA, Michael X.
REZNICK, Leah G.
RIDENHOUR, Nick C.
RIZZO, Jennifer L.
RODRIGUEZ, Fausto J.
RUSHING, Elisabeth J.
SCHECHET, Sid
SILVERMAN, Ronald H.
SINGH, Jasleen K.
SPARROW, Janet R.
SPIVEY, Bruce E.
STEIN, Joshua D.
STEINERT, Roger F.
TAKUSAGAWA, Hana L.
TAPIA, Ket
THAM, Yih-Chung
THAPA, Suman S.
THIESSEN, Craig
TIMIMI, Farris K.
TSAI, Yi-Ting
TSANG, Stephen
TSE, David T.
TYCHSEN, Lawrence
VAN METER, Woodford S.
VENABLE, Garrett T.
VITHANA, Eranga N.
WA, Christianne A.
WAISBOURD, Michael
WALA, Harpreet S.
WANG, Haibo
WANG, Ningli
WANG, Sophia Y.
WANG, Daniel Y.
WARWAR, Ronald E.
WHITLOW, Bryan T.
WILENSKY, Jacob
WIZOV, Sheryl S.
WONG, Tien
WU, Wen-Hsuan
YACKEL, Thomas R.
YANG, H. Kell
YANG, Jin
YEE, Kenneth M.P.
ZACHARIAS, Leandro C.
# Event Schedule

**THURSDAY, MAY 14**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>12:00 PM – 1:30 PM</td>
<td>New Member Luncheon (by invitation)</td>
<td>Colonnade Room</td>
</tr>
<tr>
<td>1:30 PM – 5:00 PM</td>
<td>Registration</td>
<td>Viking Ballroom Foyer</td>
</tr>
<tr>
<td>2:00 PM – 3:30 PM</td>
<td>New Member Spotlight Presentations</td>
<td>Salon DE</td>
</tr>
<tr>
<td>6:00 PM – 7:30 PM</td>
<td>Reception for New Members (black tie optional)</td>
<td>Bellevue Ballroom</td>
</tr>
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**FRIDAY, MAY 15**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>6:30 AM – 12:00 PM</td>
<td>Registration</td>
<td>Viking Ballroom Foyer</td>
</tr>
<tr>
<td>6:30 AM – 8:00 AM</td>
<td>Continental Breakfast</td>
<td>Viking Ballroom Foyer</td>
</tr>
<tr>
<td>7:20 AM – 9:30 AM</td>
<td>AOS-Knapp Symposium</td>
<td>Salon ABC</td>
</tr>
<tr>
<td>7:30 AM – 9:30 AM</td>
<td>Spouse/Personal Guest Hospitality Lounge</td>
<td>Colonnade Room</td>
</tr>
<tr>
<td>9:30 AM – 10:00 AM</td>
<td>Coffee Break and Poster Viewing</td>
<td>Salon DE</td>
</tr>
<tr>
<td>10:00 AM – 12:00 PM</td>
<td>Scientific Program (Moderator: David T. Tse)</td>
<td>Salon ABC</td>
</tr>
<tr>
<td>1:00 PM – 4:30 PM</td>
<td>Tennis Tournament</td>
<td>Pop Flack Tennis Court</td>
</tr>
<tr>
<td>1:00 PM – 4:30 PM</td>
<td>Golf Tournament</td>
<td>Newport National Golf Course</td>
</tr>
<tr>
<td>5:45 PM – 6:15 PM</td>
<td>Artistic Performances by Members &amp; Guests</td>
<td>Bellevue Ballroom</td>
</tr>
<tr>
<td>6:00 PM – 7:30 PM</td>
<td>Reception (business casual)</td>
<td>Bellevue Ballroom</td>
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</table>
# American Ophthalmological Society
## Annual Meeting

### Event Schedule

#### SATURDAY, MAY 16

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<td>6:00 AM – 12:00 PM</td>
<td>Registration</td>
<td>Viking Ballroom Foyer</td>
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<tr>
<td>6:00 AM – 8:00 AM</td>
<td>Continental Breakfast</td>
<td>Viking Ballroom Foyer</td>
</tr>
<tr>
<td>6:30 AM – 7:15 AM</td>
<td>Executive Session (members only)</td>
<td>Salon ABC</td>
</tr>
<tr>
<td>7:30 AM – 8:15 AM</td>
<td>Frederick C. Blodi Inaugural Lecture</td>
<td>Salon ABC</td>
</tr>
<tr>
<td>7:30 AM – 9:30 AM</td>
<td>Spouse/Personal Guest Hospitality Lounge</td>
<td>Colonnade Room</td>
</tr>
<tr>
<td>8:15 AM – 9:15 AM</td>
<td>Symposium: Health Care Reform in 2015</td>
<td>Salon ABC</td>
</tr>
<tr>
<td>9:15 AM – 10:00 AM</td>
<td>Coffee Break and Poster Session</td>
<td>Salon DE</td>
</tr>
<tr>
<td>10:00 AM – 12:00 PM</td>
<td>Scientific Program (Moderator: J. Sebag)</td>
<td>Salon ABC</td>
</tr>
<tr>
<td>12:00 PM – 1:30 PM</td>
<td>Emeritus Luncheon (by invitation)</td>
<td>Touro Room</td>
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<tr>
<td>12:30 PM – 3:30 PM</td>
<td>America’s Cup Sail Race</td>
<td>Newport Harbor</td>
</tr>
<tr>
<td>6:30 PM – 7:30 PM</td>
<td>Reception (black tie optional)</td>
<td>Bellevue Ballroom</td>
</tr>
<tr>
<td>7:30 PM – 10:30 PM</td>
<td>Banquet (black tie optional)</td>
<td>Salon ABC</td>
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#### SUNDAY, MAY 17

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>6:30 AM – 10:00 AM</td>
<td>Registration</td>
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</tr>
<tr>
<td>7:30 AM – 10:00 AM</td>
<td>Scientific Program (Moderator: Edward G. Buckley)</td>
<td>Salon ABC</td>
</tr>
</tbody>
</table>
Scientific Program

SYMPOSIUM AGENDAS AND PAPER ABSTRACTS

The following abstracts of papers selected to be presented at the meeting are printed in presentation order. Scientific sessions will be held in Salon ABC.

Papers presented at this meeting may be published in other medical journals after this meeting provided the authors adhere to the strict guidelines in the author instructions listed at aosonline.org and consult with the editor of the transactions.

Papers are limited to 10 minutes and the first discussant to 3 minutes. General discussion will be limited to 6 minutes.

PLEASE NOTE THE FOLLOWING PROGRAM KEY

**Bold** = AOS Member
* = Presenter
* = Financial Disclosure

(Presenters will indicate their financial disclosure verbally and in the first slide.)
2015 Knapp Symposium
INNOVATIONS IN MEDICAL EDUCATION

FRIDAY, MAY 15

7:20 AM  
Introduction  
**J. Sebag, MD**  
Professor of Clinical Ophthalmology  
Doheny Eye Institute  
VMR Institute for Vitreous Macula Retina  
Huntington Beach, CA

7:25 AM  
What Happened to my Medical School?  
**Edward G. Buckley, MD**  
Vice Dean for Education  
Chair, Department of Ophthalmology  
Duke University Medical Center  
Durham, NC

7:45 AM  
Creating Future Leaders  
Darrell G. Kirch, MD  
President and CEO  
Association of American Medical Colleges  
Washington, DC

8:05 AM  
Transforming Resident Education  
Benjamin A. Alman, MD  
Chair, Department of Orthopedics  
Duke University Medical Center  
Durham, NC

8:25 AM  
Health Care Social Media and Digital Identity for Ophthalmology: Opportunity, Risk and Reward  
Farris K. Timimi, MD  
Medical Director of Social Media  
Mayo Clinic  
Rochester, MN

8:45 AM  
Educating the World’s Ophthalmologists  
**Bruce E. Spivey, MD**  
Immediate Past President  
International Council of Ophthalmology  
San Francisco, CA

9:05 AM  
Audience Q and A
TNF-ALPHA INDUCED CHOROIDAL NEOVASCULARIZATION INHIBITED BY ACTIVE RAP1 GTPASE

Haibo Wang, Mary Elizabeth Hartnett*

Purpose: When activated choroidal endothelial cells (CECs) migrate into the retina in neovascular age-related macular degeneration (AMD), vision loss invariably occurs. We explored crosstalk between inflammatory and oxidative mechanisms involved in CEC activation and tested the hypothesis that tumor necrosis factor alpha (TNF alpha)-mediated CEC migration and choroidal neovascularization (CNV) were inhibited by activation of guanosine triphosphatase (GTPase), Rap1.

Methods: CECs were isolated from de-identified donor human eyes in accordance with University of Utah Human Studies, expanded and cultured through passage 5 for experimentation. CECs were stimulated with TNF alpha, vascular endothelial growth factor (VEGF), or phosphate-buffered saline (PBS) control, and cells or lysates analyzed for reactive oxygen species (ROS), active Rac1, or CEC migration. In some experiments, CECs were 1) treated with antioxidant, apocynin, the Rap1 activator, 2′-O-Me-cAMP (8CPT), or PBS; 2) transfected with small interfering RNA (siRNA) to nicotinamide adenine dinucleotide phosphate-(NADPH) oxidase subunit, p22phox, or control siRNA; or 3) infected with adenoviral-activated Rap1a (adRap1a) or adenoviral-green fluorescent protein (adGFP) control. Six-week old C57Bl/6 mice underwent laser (MicronIV, Phoenix) and were treated with TNFalpha antibodies, 8CPT, or controls. Lectin-stained choroidal flat mounts were analyzed for volume of CNV using confocal microscopy. Statistics were analyzed by ANOVA.

Results: Compared to PBS, CECs stimulated with TNFalpha or VEGF had significantly greater migration (p<0.01). Compared to respective controls, p22phox siRNA reduced TNF alpha-induced ROS, active Rac1, and apocynin reduced CEC migration (all p<0.05). Compared to PBS or adGFP, 8CPT or active Rap1 inhibited ROS, active Rac1, and CEC migration induced by TNF alpha. Either TNF alpha antibody or 8CPT inhibited laser-induced CNV compared to controls.

Conclusion: These results support the hypothesis that TNF alpha-induced ROS mediate CEC migration through Rac1 and that activation of Rap1 by chemical or gene therapy inhibits TNFalpha-induced CEC migration. These results support additional investigation into Rap1 as a potential therapeutic in CNV.

Discussant: Susan Elner*
**OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY OF THE PERIPAPILLARY RETINAL CIRCULATION IN GLAUCOMA**

David Huang*, Yali Jia*, Liang Liu, Beth Edmunds, Lorinna Lombardi*, Ellen Davis, Hana Takusagawa, John C. Morrison*

**Purpose**: To evaluate the peripapillary retinal circulation in glaucoma.

**Methods**: Glaucoma and normal control participants are enrolled in a prospective observational study at the Casey Eye Institute according to criteria defined by visual field (VF) and optic disc appearance. One eye of each participant was imaged using a high-speed (70 kHz) 840 nm wavelength spectral optical coherence tomography (OCT) system (RTVue-XR, Optovue). The optic disc region was scanned twice using a 3x3 mm volumetric angiography scan. The split-spectrum amplitude decorrelation angiography (SSADA) algorithm was used to compute 3D angiograms. En face retinal angiogram was obtained by maximum flow projection. The peripapillary flow index was defined as the average decorrelation value in the peripapillary region, defined as a 700-micron wide elliptical annulus around the disc. The peripapillary vessel density was the percentage area occupied by vessels.

**Results**: The study included 12 glaucoma and 12 age-matched normal participants. The retinal vascular network around the disc was visibly attenuated in glaucomatous eyes and focal capillary dropout could be detected. The flow index in the glaucoma group was 0.066 ± 0.012 (mean ± SD), which was lower (P=0.001, Mann-Whitney U test) than normal (0.082 ± 0.007). The vessel density in the glaucoma group was 80.6% ± 11.1%, which was lower (P<0.001) than normal (93.0% ± 2.8%). Both flow index and vessel density were highly correlated (Pearson’s R = -0.808 and -0.835, p<0.001) with VF pattern standard deviation in the glaucoma group. The area under the receiver operating curve for differentiating healthy and glaucoma participants was 0.892 for flow index and 0.938 for vessel density.

**Conclusion**: Using OCT angiography, glaucomatous reduction in peripapillary retinal perfusion could be visualized as focal defects and quantified as flow index and vessel density with high diagnostic accuracy. Quantitative OCT angiography is potentially useful in glaucoma evaluation.

**Discussant**: James Tsai*
OCT EVALUATION OF SUBRETINAL VESSEL LOCATION IN POLYPOIDAL CHOROIDAL VASCULOPATHY (PCV) AND RESPONSE OF HEMORRHAGIC AND EXUDATIVE PCV TO HIGH DOSE ANTIANGIOGENIC THERAPY

Gregg T. Kokame

Purpose: The purpose was to test two primary hypotheses: 1) Is polypoidal choroidal vasculopathy (PCV) a subretinal neovascular process, rather than a choroidal vascular anomaly? 2) Is a higher dose of ranibizumab (2.0 mg/0.05ml) more effective in PCV than the current dose (0.5 mg/.05 ml) approved for age-related macular degeneration (AMD)?

Methods: Prospective evaluation of PCV in 104 eyes of 86 patients with ICG+OCT localizing the branching vascular network and the polyps. 19 eyes of 19 patients with active leaking and exudation underwent a prospective open-label trial of monthly high-dose intravitreal ranibizumab (2.0 mg per 0.5 ml). The primary outcomes were prevention of major vision loss (> or = 15 ETDRS letters). Secondary outcomes included adverse events, improved vision, and changes in subretinal hemorrhage, subretinal fluid, macular edema, and polypoidal complexes at 6 months.

Results: The PCV vessels were localized beneath the retinal pigment epithelium (RPE) and above Bruch’s membrane in 103 of 104 eyes (99%). In the high dose ranibizumab trial at 6 months none of the patients lost > or =15 letters in visual acuity, and 26% (5/19 eyes) gained > or = 15 letters. Decreases were noted in subretinal fluid in 14/17 eyes (82%), subretinal hemorrhage in 12/12 eyes (100%), RPE detachment in 14/16 eyes (88%), macular edema in 11/12 eyes (92%), and polyps in 15/19 eyes (79%).

Conclusion: PCV vessels are a subtype of subretinal neovascularization located above Bruch’s membrane and below RPE. High dose ranibizumab (2.0 mg/0.05ml) decreased exudation and hemorrhage, and resulted in significant polyp regression, although branching vascular networks persisted.

Discussant: Lawrence Yannuzzi
NAILFOLD MICROVASCULAR ABNORMALITIES IN PRIMARY OPEN-ANGLE GLAUCOMA

Louis R. Pasquale*, Aiai Ren, Akiko Hanyuda, Jae Hee Kang, Michael Giovingo, Paul Knepper

Purpose: There is considerable evidence for systemic vascular dysfunction in primary open angle glaucoma (POAG). Since the pre-capillary arteriole-to-venous connection is more readily visible in the nailfold compared to the optic nerve head, we performed nail fold capillary video microscopy to directly observe the nature of vascular dysfunction in POAG.

Methods: We performed nailfold capillary video microscopy on the fourth and fifth digit of the non-dominant hand using a JH=1004 capillaroscope. We enrolled 209 POAG patients (including 28 with normal tension glaucoma) and 165 control subjects from four sites. Videos were placed in cloud storage for viewing by masked observers, who graded them for hemorrhages, dilated capillary loops >50 microns and avascular zones > 200 microns. Multivariable odds ratio (ORs) of POAG and glaucoma severity (based on a visual field score from 1 to 4) with associated 95% confidence intervals were obtained from logistic and ordinal regression analysis, respectively.

Results: After controlling for demographic factors, family history of glaucoma, systemic disease and use of anticoagulants, for each 100 nailfold capillaries sampled, avascular zones > 200 microns (OR = 4.20 (1.35-13.11); p=0.01) and hemorrhages (OR = 1.53 (1.31-1.83); p<0.001) were associated with POAG. Furthermore nailfold hemorrhages were also associated with incremental glaucoma severity based on visual field scoring (OR = 1.23 (1.11-1.36); p<0.001). Avascular zones > 200 microns were not associated with glaucoma severity (OR=1.36 (0.95-1.93); p=0.09). The number of dilated capillary loops >50 microns was only weakly associated with POAG (OR=1.12 (0.99-1.27); p=0.08) and not associated with glaucoma severity ( OR=1.04 (0.93-1.17); p=0.47).

Conclusion: These data provide insight into the nature of non-ocular capillary bed abnormalities in POAG. Whether similar abnormalities occur in relevant ocular tissues needs to be determined.

Discussant: Henry Jampel*
SPATIAL DISTRIBUTION OF VISUAL FIELD LOSS FOR DIABETIC RETINOPATHY AND GLAUCOMA USING AN IPAD VISUAL FIELD SCREENING TEST

Alan L. Robin*, Chris A. Johnson†, Suman Thapa

Purpose: To determine the spatial characteristics and frequency of visual field (VF) deficits using a free iPad application, Visual Fields Easy (VFE), in screening normal and glaucomatous Nepalese at the Tilganga Eye Hospital, Kathmandu.

Methods: The VFE iPad app, presents 88 Goldmann size V targets (22 per visual field quadrant) at a16 dB intensity on a 31.5 apostilb (10 cd/m2) background. A red fixation point is presented at one corner of the display (located 33 cm in front of the observer) and test locations are presented (200 msec duration) at various locations in the quadrant and then the red fixation point moves to another corner of the display (the next quadrant). SITA 24-2 Standard tests were used for comparison. We evaluated 210 normal control, 183 glaucoma, and 18 eyes with diabetic retinopathy. We compared the number of missed points on screening with the number of locations outside normal limits for the SITA Total Deviation (TD) and Pattern Deviation (PD).

Results: The number of missed test locations for the VFE demonstrated a good correlation (r=0.79) with the SITA Standard Mean Deviation (MD) and Pattern Standard Deviation (PSD) values (r=0.60). In all tested, VFE found no difference in the frequency of VF deficits in different quadrants, while in glaucoma eyes, SITA found a slightly greater proportion of deficits in the nasal visual field. The average testing time for VFE was 3.3 minutes. There were approximately twice as many locations outside normal limits for TD compared to the screening test, but PD abnormalities were similar to the screening results, indicating that mild deficits were not detected by the screening procedure.

Conclusion: VFE is a relatively effective procedure for perimetric population screening. These findings provide a basis for developing platforms and probability values that can be used for refined adaptive screening.

Discussant: Robert L. Stamper*
2015 Saturday Symposia

SATURDAY, MAY 16

FREDERICK C. BLODI INAUGURAL LECTURE

7:30 AM  Introduction
Hans E. Grossniklaus, MD
Calhoun Jr. Professor of Ophthalmology
Emory Eye Center
Atlanta, GA

7:35 AM  The Macular Degeneration Complex
Timothy W. Olsen, MD
Chair, Department of Ophthalmology
Emory Eye Center
Atlanta, GA

8:00 AM  Audience Q and A

HEALTH CARE REFORM IN 2015

8:15 AM  Introduction
Jay C. Erie, MD
Professor of Ophthalmology
Mayo Clinic
Rochester, MN

8:20 AM  Navigating the Changing Healthcare Policy and Political Landscape
Kathleen Harrington
Chair of Government Relations
Mayo Clinic
Rochester, MN

8:40 AM  Health Care Reform and Ophthalmology: A View at 5 Years
Michael X. Repka, MD
Medical Director of Government Affairs
American Academy of Ophthalmology
Johns Hopkins University School of Medicine
Baltimore, MD

9:00 AM  Audience Q and A
TRABECULECTOMY SLOWS OR REVERSES THE RATE OF VISUAL FIELD DECAY FROM GLAUCOMA

Joseph Caprioli*, John Mark De Leon, Parham Azarbod, Esteban Morales, Andrew Chen, Kouros Nouri-Mahdavi*, Abdelmonem Afifi, Anne L. Coleman*

Purpose: To investigate alterations in the magnitude and direction of visual field (VF) rates of decay in glaucoma patients after trabeculectomy.

Methods: This is a retrospective study of open-angle glaucoma patients who underwent trabeculectomy with mitomycin-C. Inclusion criteria included ≥ 4 reliable VFs before and after trabeculectomy and a minimum of 2 years follow-up prior to and after trabeculectomy. A pointwise exponential regression model was used to measure VF decay rates at every test location before and after surgery; these were assigned to either a fast or slow component of VF decay for each eye. Fast and slow component rates were calculated before and after trabeculectomy. An algorithm determined whether a VF location decayed or improved after trabeculectomy and corresponding decay or improvement exponential regression models were used to calculate rates. The rates of change for mean deviation (MD) and visual field index (VFI) were also calculated with linear regression.

Results: Seventy three eyes (64 subjects) met the inclusion criteria and were followed (mean ± SD) for 5.1 ± 2.1 years before and 5.4 ± 2.3 years after surgery, with 8.9 ± 4.7 VFs before and 9.0 ± 4.4 VFs after surgery. The mean intraocular pressures (IOP) were 14.7 ± 3.3 and 10.0 ± 3.2 mmHg before and after surgery, respectively (p<0.001). The mean rate of the fast component of VF decay changed from -8.3 ± 12.8 %/year before to -0.5 ± 8.3 %/year after surgery (p<0.001). The slower component mean rate changed from +4.4 ± 8.7 %/year before to -0.1 ± 8.6 %/year after surgery (p=0.002). For test locations belonging to the fast component, there were more improving VF locations after surgery (53%) compared to before surgery (13%, p<0.001). Compared to a glaucomatous non-operated comparison group (with a “mock” surgery date in the middle of follow-up), there were significantly more locations that decayed pre-operatively and improved post-operatively after trabeculectomy (p<0.001).

Conclusions: Trabeculectomy significantly decreases the rate of VF decay in open-angle glaucoma. This slowing is particularly robust for the fast VF decay component. The changes in the fast component represent reduced rates of glaucoma deterioration while the changes in the slower component represent mostly non-glaucomatous deterioration such as caused by media, aging, and other effects. There is evidence of significant and sustained improvement of visual sensitivities after trabeculectomy.

Discussant: George Spaeth*
LONG-TERM DEVELOPMENT IMPROVEMENT IN CHILDREN WITH NEUROBEHAVIORAL DISORDERS FOLLOWING PHOTOREFRACTIVE KERATECTOMY FOR ISOAMETROPIC AMBLYOPIA


Purpose: To assess the long-term impact of photorefractive keratectomy (PRK) correction of severe isoametropia on the development of children with neurobehavioral problems.

Methods: This is a prospective, interventional case series. Children with neurobehavioral disorders and severe isoametropia unwilling or unable to tolerate using refractive correction underwent PRK. Developmental status was evaluated preoperatively and at 6, 12, 24 and 36 months postoperatively. The main outcome measure was the developmental quotient (DQ). Secondary outcome measures were uncorrected visual acuity, refractive error, and cycloplegic refraction.

Results: Fifteen children aged 4-11 years were included. Twelve were myopic (-9.8 +/- 3.9D), two hyperopic (+5.8 +/- 0.4D) and one astigmatic (+3.5D). Significant DQ improvement was found in receptive, expressive and written communication (p=0.001, 0.05, 0.04 respectively), domestic daily living skills (p=0.03) and interpersonal socialization skills (p=0.02) for the first 12 months, which then plateaued. Improvement in visual perception and motor coordination occurred at 36 months postoperatively. Uncorrected visual acuity improved after PRK (logMAR +1.25 +/- 0.6 to +0.55 +/- 0.4). Mean spherical equivalent refractive error was significantly improved at 6 and 36 months at -0.6 +/- 1.5D and -1.7 +/- 2.2D for the myopic group, +1.4 +/- 1.1D and +2.0 +/- 1.1D for hyperopic group and +1.6 +/- 0.5D and +2.4 +/- 0.2D for the astigmatic patient.

Conclusion: In addition to improvement in visual acuity and refractive error, PRK in children with neurobehavioral disorders and severe isoametropia results in long-term improvement in development, social skills and communication. This translates into an improvement in the quality of life of these severely impaired children.

Discussant: Lawrence Tychsen
QUANTITATIVE ULTRASONOGRAPHY OF VITREOUS CORRELATES WITH CONTRAST SENSITIVITY AND VFQ VISUAL QUALITY OF LIFE ASSESSMENT IN PATIENTS WITH FLOATERS


Purpose: The clinical evaluation of floaters lacks quantitative assessment of vitreous structure. This study developed quantitative ultrasonography (QUS) to measure vitreous echodensities in patients with floaters. Since floaters reduce contrast sensitivity (CS) and visual quality of life (VFQ), it is hypothesized that QUS will correlate with CS and VFQ in patients with floaters.

Methods: 22 eyes of 22 subjects (age = 57+/−19 years) with floaters were evaluated with Freiburg Acuity Contrast Testing (CS; %Weber) and Visual Function Questionnaire (VFQ). Ultrasonography used a customized probe (15MHz, 20mm focal length, 7mm aperture; Quantel Medical) with longitudinal and transverse scans taken in primary gaze and a horizontal longitudinal scan through the premacular vitreous in temporal gaze. Each scan set had 100 frames of log-compressed envelope data. Within each frame, two regions of interest (ROIs) were analyzed (whole-central and posterior vitreous) to yield parameters (energy, E; mean amplitude, M; and percentage of vitreous filled by echodensities, P50) averaged over the 100-frames. Spearmans R evaluated E, M, and P50 correlations with CS and VFQ.

Results: 10 eyes from 10 additional subjects showed good reproducibility (R>0.828) for all QUS indices. In the 22 eyes with floaters, CS ranged from 1.19% Weber to 5.59% Weber. All QUS parameters (E, M, P50) correlated with CS (R>0.67, p<0.001), and notably, P50 had R=0.867 (p<0.001). Correlations of QUS with VFQ ranged from R=0.52 (p<0.013) to R=0.65 (p<0.001) for the different QUS parameters and vitreous ROIs.

Conclusion: Quantitative ultrasonography (QUS) in patients with floaters provides objective, reproducible measures of vitreous echodensity that correlate with contrast sensitivity and quality of life, providing objective assessment of vitreous structure underlying the functional disturbances induced by floaters. By quantifying disease severity, this approach should facilitate surgery case selection and have great utility in quantifying the response to surgery as well as new forms of therapy, such as pharmacologic vitreolysis.

Discussant: Mark W. Johnson*
THE CECOCENTRAL SCOTOMA: A NEURO-OPHTHALMIC UPDATE

Steven A. Newman*

Purpose: Central scotoma is the typical defect in neuro-ophthalmology. Lawton Smith studied 65 patients with cecocentral scotomas. Optic neuritis was followed by toxic, and genetic. Since 1979 VF (kinetic perimetry and tangent screen) is now done with automated static perimetry. Macula pathology is a frequent cause of central scotomas. When large enough to involve the blind spot they may look cecocentral. We have retrospectively analyzed central and cecocentral scotomas seen at the UVA studied with automated static perimetry.

Methods: A retrospective study of 193 patients referred to the Neuro-Ophthalmology Division at UVA and coded for central and cecocentral scotomas. Patterns were analyzed. Foveal sensitivity was available on all fields. Most patients were studied with a 10-2 program (+ 24-2) and some patients had V size as well as III size test object.

Results: Twenty-nine charts were excluded for transient central scotomas, arcuate, or paracentral defects (14 were unknown). Of remaining 150 patients, 80 macular pathology, (67 macula and 13 with retinovascular disease). Of primary optic nerve pathology, optic neuritis and compressive optic neuropathies were the most common, followed by AION, neuroretinitis, neuromyelitis optica, and traumatic optic neuropathy. There were 2 cases of Leber’s, 1 case of dominant optic atrophy, 2 cases of nutritional optic neuropathy, and 1 case of toxic.

Conclusion: In spite of a selective referral bias (often retina to neuro-op) retinal disease was still the most common cause of central and cecocentral scotomas. OCT has helped distinguish macula pathology from optic nerve pathology. Even when the macula looked normal, functional studies could demonstrate underlying physiologic pathology. The use of the 10-2 program (+/-a V size test object) was often necessary to recognize the pattern. Even the classic toxic, metabolic, and hereditary optic neuropathies may involve central fixation without extension to the blind spot, calling into question the classical teaching underlying cecocentral scotoma.

Discussant: Anthony C. Arnold
USE OF THE AMERICAN BOARD OF OPHTHALMOLOGY'S MAINTENANCE OF CERTIFICATION PROGRAM TO MEET REGULATORY AND QUALITY REQUIREMENTS

David Wilson*, Michael Siatkowski*, John Clarkson

Purpose: To evaluate if a modification of the American Board of Ophthalmology's (ABO) Maintenance of Certification (MOC) process is suitable to meet The Joint Commission’s (TJC) Ongoing Professional Provider Evaluation (OPPE) requirement and the OHSU institutional requirement for a quality monitoring and improvement program.

Methods: For the past year clinical faculty at the Casey Eye Institute were provided a list of charts conforming to the requirements of the ABO’s practice improvement module (PIM), specific for their area of practice. This length of time corresponds to two OPPE cycles, and four cycles for OHSU quality reporting cycles. The faculty abstracted the information from the assigned charts to complete the requirements of the PIM. The abstracted data was compared to predetermined metrics to assess for gaps in care that would be relevant to the OPPE process and the institutional quality program.

Results: During the first OPPE cycle faculty performance fell below the predetermined metric in the following areas: 1) informed consent for ptosis surgery, 2) graft clarity in keratoplasty for corneal edema, 3) preoperative eye position measurement in surgical treatment of esotropia, 4) counseling for vitamin usage in age related macular degeneration, and 5) performance of neuro-imaging in optic neuritis. All of these gaps in performance were remedied in the second OPPE cycle, but other performance deficiencies were noted.

Conclusion: The modified ABO PIMs were very useful as a program to meet OPPE and Institutional Quality Requirements. The PIMs have the advantage of having been rigorously and professionally developed by the standard setting organization for Ophthalmology.

Discussant: George Bartley
DEFECTIVE EPITHELIAL BASEMENT MEMBRANE REGENERATION, MYOFIBROBLASTS, AND SCARRING IN THE CORNEA AFTER PRK IN RABBITS

Steven E. Wilson*

**Purpose:** To examine mechanisms related to defective regeneration of the epithelial basement membrane (EBM) that has been shown to underlie the development of corneal scarring (haze) after photorefractive keratectomy (PRK).

**Methods:** Transmission electron microscopy (TEM) was used to monitor regeneration of the lamina lucida (LL) and lamina densa (LD) of the EBM in rabbits after moderate PRK (-4.5D) that does not produce haze and high PRK (-9D) that produces haze in 100% of rabbit corneas. Laser capture-reverse transcription polymerase chain reaction (RT-PCR) was used to measure mRNA production for the EBM components nidogen-1 and perlecan in the anterior stroma of corneas that had -4.5D PRK or -9D PRK.

**Results:** TEM showed that LL/LD regenerated on average at 9.5 days after -4.5D PRK, but had not regenerated by 3 months after -9D PRK. Laser capture RT-PCR showed that nidogen-1 and perlecan mRNAs were produced by anterior stromal cells (primarily corneal fibroblasts and keratocytes) during the days leading up to and after regeneration of the LD/LL. Conversely, in corneas that had -9D PRK, that develop haze, little nidogen-1 or perlecan mRNA was produced by anterior stromal cells (primarily myofibroblasts and precursors associated with the haze) up to one month after surgery.

**Conclusion:** The EBM, the critical regulator of epithelium-derived TGFβ that drives myofibroblast development in the stroma, does not regenerate fully in corneas that develop haze after PRK. This study suggests that the anterior stromal cells in corneas that develop haze (primarily myofibroblasts and their precursor cells) do not produce sufficient nidogen-1 or perlecan needed to regenerate LL/LD of the EBM. We hypothesize that the larger wave of stromal keratocyte apoptosis after higher PRK corrections leads to diminished anterior keratocytes to provide critical EBM components and, therefore, allows ongoing penetration of TGFβ from the epithelium to drive generation and persistence of the haze-associated myofibroblasts.

**Discussant:** Dimitri Azar*
THE POWER OF SAMPLE SIZE IN UNDERSTANDING FLAP STRIAE AS A RISK FACTOR OF LOW INCIDENCE IN REFRACTIVE SURGERY

Ronald R. Krueger*, Minoru Tomita*

Purpose: To correlate climatic change to the monthly incidence of flap striae requiring flap lift after LASIK performed at a single high volume center.

Methods: Data on all LASIK cases performed at the Shinagawa LASIK Center in Tokyo between June 2007 and April 2012 was reviewed by month for total number of LASIK cases and striae requiring flap realignment. Statistical analyses were then performed to determine any significant differences in incidence by month, season, and year. Using data from the Japan Meteorological Agency (http://www.jma.go.jp/jma/indexe.html), average monthly humidities and temperatures for the same time were obtained and compared to monthly realignment rates.

Results: For the period reviewed, 614,340 eyes had LASIK surgery at Shinagawa LASIK Center of Tokyo. Of these, a total of 5,244 developed striae requiring realignment, a cumulative incidence of 0.85%. Averaged for all years, the monthly incidence ranged from 0.657% to 1.006%. The lowest monthly incidences were noted in the summer months (June-August), which was statistically significant for the summer season (p<.05). Comparison of the average monthly humidity and temperatures to the average monthly incidence of macrostriae for the years 2008 to 2011 revealed a strong inverse correlation for each (R = -.902, R = -.888).

Conclusion: Due to the very high sample size, the 0.85% cumulative incidence represents a number that experienced surgeons can use as a metric to assess their own flap striae rates. The reported lower incidence during the summer months is the first time that climatic change has been statistically correlated to flap striae rate, which although different is too low to change practice patterns. The strong inverse correlation with seasonal temperature and humidity may point to air moisture providing a protective effect against post-LASIK dryness and subsequent eye rubbing.

Discussant: Roger Steinert
A COMMON POAG RISK VARIANT OF THE GENE SIX6 IS ASSOCIATED WITH REDUCED SUPERIOR AND INFERIOR RETINAL NERVE FIBER LAYER (RNFL) THICKNESS IN NON-GLAUCOMATOUS ASIAN SUBJECTS


Purpose: POAG is a complex inherited trait. Recently, a common genetic variant of the gene SIX6, rs33912345 (Asn141His), has been identified that is highly associated with POAG-risk. This variant affects optic nerve and eye development in the zebrafish model. It is also associated with reduced RNFL thickness in POAG cases. We examined the effect of this common, functional genetic variant on RNFL thickness in the Singapore Chinese population.

Methods: Study subjects were enrolled in the IRB-approved Singapore Chinese Eye Study (SCES), a population-based survey of Singaporean Chinese aged 40 years or older. Subjects underwent a comprehensive ocular examination according to a standardized protocol. SD-OCT was used to measure RNFL thicknesses. Genotyping of SIX6 rs33912345 (Asn141His) was performed using the Illumina exome array.

Results: A total of 1,222 subjects without glaucoma (mean age: 55.0 +/- 7.4 years) with genotype data and SD-OCT images were analyzed. The allele frequency of the risk variant was 80%. Each rs33912345 risk allele was associated with a 1.34 um decrease in mean RNFL thickness, after adjusting for age, sex, and axial length (P=0.003). The strongest association was observed in the superior and inferior RNFL quadrants (P < 0.001 and 0.003, respectively). There was no significant difference in RNFL thickness in the nasal and temporal quadrants.

Conclusion: The very common, functional SIX6 POAG-risk variant, rs33912345, is associated with a global reduction in RNFL thickness that is confined to the superior and inferior quadrants in the Asian population without glaucoma, regions usually affected early in glaucomatous optic neuropathy. This suggests that this variant increases risk of POAG but also reduces RNFL thickness in a large number of persons that will never develop glaucoma. Further studies are needed to determine if this effect on RNFL thickness occurs in other populations and how this confers increased risk for POAG.

Discussant: Louis Pasquale
PARTIAL MUSCLE RECESSION FOR SMALL-ANGLE VERTICAL STRABISMUS

Steven M. Archer*, Catherine S. Choi, Jasleen K. Singh

Purpose: To evaluate vertical rectus muscle partial tendon recession for treatment of small vertical deviations.

Methods: This is an institutional retrospective consecutive series of 56 recessions of one pole of one or two vertical rectus muscles in 47 patients performed by one surgeon; 4 patients/procedures were excluded for lack of follow-up data. Preoperative deviation, change and residual deviation and the PD/mm surgery were evaluated. Separate analyses compared outcomes in patients with Graves eye disease and those in whom the operated muscle had previous surgery.

Results: The mean vertical deviation preoperatively was 4.6 PD (SD 2.0 PD) and postoperatively was 0.0 PD (SD 2.4 PD), p < 0.0001. The distribution of observed surgical responses in PD/mm was not Gaussian, but instead was sharply peaked at the mean of 1.5 PD/mm. With regard to vertical deviation, 64% were orthophoric post-operatively and only 7/43 patients required prism or additional surgery after their initial surgery. 60% of Graves patients were orthophoric post-operatively versus 65% of non-Graves patients. 29% of patients who had previous surgery on the operated muscle were orthophoric versus 69% of those who did not.

Conclusion: For patients with small vertical deviations who reject prism spectacles, partial tendon recession is an alternative to previously described partial tenotomy, mini-tenotomy and mini-plication procedures. There is no significant difference in outcomes between patients with or without Graves eye disease; however, muscles with previous surgery are less predictable.

Discussant: M. Edward Wilson†
TRANSFORMATION OF BENIGN CHOROIDAL NEVI TO MALIGNANT MELANOMAS: AUTHORITATIVE PRONOUNCEMENTS VERSUS SCIENTIFIC EVIDENCE

James J. Augsburger*, Zelia M. Correa

Purpose: Some small melanocytic choroidal tumors diagnosed as nevi by experienced clinicians enlarge during post-baseline follow-up. Many clinicians advise such patients that their previously documented benign choroidal nevus has transformed into a malignant melanoma. The purpose of this paper is to show that growth of a clinically diagnosed choroidal nevus is unreliable evidence of malignant transformation of that tumor.

Methods: Retrospective analysis of 8 patients with a clinically diagnosed choroidal nevus versus melanoma [tumors most clinicians would classify either as a suspicious choroidal nevus or small choroidal melanoma] whose tumor was biopsied at baseline, shown to be a nevus or paucicellular spindle cell tumor by cytology and class 1 tumor by gene expression profile testing, monitored periodically without treatment following the biopsy, documented to enlarge following the biopsy, and then re-biopsied. The tumors increased in size by an average of 1.0 mm in largest basal diameter (extremes 0 to 3.5 mm) and 0.8 mm in thickness (extremes 0 to 2.3 mm) during a median follow-up of 7.5 months (extremes 5.5 to 39 months).

Results: Re-biopsy showed each tumor in this series to have similar cytopathologic features after growth and a persistent class 1 gene expression profile.

Conclusion: Many benign choroidal nevi that enlarge after initial documentation are still benign nevi after that growth, and gene expression profile transformation from class 1 to class 2 appears to be uncommon in such tumors. Based on this evidence, it is inappropriate to equate enlargement of a clinically diagnosed choroidal nevus with malignant transformation of that lesion.

Discussant: Samuel Packer
MODELING AND OPTIMIZATION OF CLINICAL WORKFLOW USING COMPUTER BASED SIMULATIONS

Michelle R. Hribar*, Sarah Read-Brown, Leah G. Reznick, Thomas R. Yackel, Michael F. Chiang*t

Purpose: Although electronic health records (EHRs) have potential to improve the quality and cost of health care, there are concerns that they negatively impact clinical efficiency. Ophthalmologists typically attempt to improve efficiency by scheduling, and by multiple ancillary staff members and exam rooms. However, there are no methods for optimizing this process. This study validates methods for automated time-motion data collection using EHR timestamps, and proposes using these data for computer simulation models to optimize efficiency.

Methods: Two authors (MRH, SRB) observed a provider (LGR) clinic for 3 days, using time-motion methods to manually record times that patients spent during each part of their exam ("encounter") with the provider and each ancillary staff member. Observed times were compared with timestamps automatically recorded in the EHR (Epic; Verona, WI). Simulation models (Arena; Rockwell Automation, Milwaukee, WI) were run using these data to optimize scheduling strategies, staff usage, and number of exam rooms for minimizing patient wait time.

Results: 33 patient visits were observed. This involved 28 (85%) encounters with ancillary staff and 27 (82%) encounters with the attending provider. Overall, 55/82 (67%) of encounter times from EHR timestamps fell within 3 minutes of observed times. Discrete simulation models showed impact on mean patient wait times: (1) improved when scheduling patients alternating dilated and not (8 minutes) vs. scheduling without regard to dilation (10.3 minutes); (2) improved when using 1 ancillary staff (12 mins/patient) compared to no ancillary staff (44 mins/patient), and reduced more (8 mins/patient) when using 2 ancillary staff; (3) improved when using 3 exam rooms (13 mins/patient) vs. 2 exam rooms (16 mins/patient), with less improvement (12 mins/patient) when using 4 exam rooms.

Conclusion: Automated EHR timestamp data can be used to estimate exam times accurately. This creates potential for creating computer simulation models to evaluate and improve efficiency and workflow.

Discussant: Timothy Olsen*
COMPARISON OF DALK VS PK OUTCOMES FOR KERATOCONUS, STROMAL DYSTROPHIES AND HSV KERATITIS

Donald Tan**, Marcus Ang, Arundhati Anshu

Purpose: To compare graft outcomes between Deep Anterior Lamellar Keratoplasty (DALK) and Penetrating Keratoplasty (PK) for various indications, including keratoconus, stromal dystrophies, and herpes simplex keratitis (HSV).

Methods: Data was obtained from the Singapore Cornea Transplant Study (SCTS), a prospective transplantation registry in Asian eyes spanning 4,700 grafts over 23 years. Chi-square and Fisher's Exact tests were conducted for comparison of disease groups, survival rates were determined using Kaplan-Meier method, and Mantel-Cox Log Rank test used to compare survival rates.

Results: Overall 1 and 5-year survival rates for 1,242 first time grafts performed between 2000 to 2011 was highest in DALK (96.3%, 90.5%) compared to PK (94.2%, 71%, p<.001), and EK (96%, 77.3%). Glaucoma was highest among PK (n=106, 17.8%) and EK (n=68, 19.4%), and lowest in the DALK group (n=22, 7.4%); graft rejection was also highest in the PK group (n=59, 9.9%), followed by EK (n=15, 4.2%) and DALK (n=3, 1.0%). In keratoconus (n=125), logMAR visual outcomes of descemets baring DALK and PK were not statistically different (0.15, 0.27 (p=.26) but were lower in the predescemet DALK cases (0.41, p=.013). Long term (10 year) survival analysis of 110 grafts (DALK=63, PK=47) for stromal dystrophies confirmed enhanced 10-year graft survival in the DALK group (log rank p=0.013) and similar rates of primary disease recurrence (PK=10.6%, DALK=12.7%, p=0.74). Analysis of 324 grafts for HSV keratitis (PK=224, DALK=100) showed better 6-year graft survival in the DALK group (PK=68.8%, DALK=85%)(log rank p=0.024) with lower incidence of HSV reactivation and reduced number of recurrences in DALK cases.

Conclusion: Our analysis of DALK outcomes as compared to PK for a variety of indications including keratoconus, stromal dystrophies and HSV keratitis confirms better long-term outcomes, in terms of reduced complications, and enhanced graft survival. Surgeons should consider performing DALK as an alternative to conventional PK for these conditions.

Discussant: Penny Asbell*
AFLIBERCEPT, BEVACIZUMAB, OR RANIBIZUMAB FOR DIABETIC MACULAR EDEMA

Lee M. Jampol*

Purpose: To evaluate the relative visual acuity and OCT efficacy of intravitreous injections of vascular endothelial growth factor inhibitors aflibercept, bevacizumab, and ranibizumab for treating diabetic macular edema (DME) involving the center of the macula.

Methods: At 89 sites, one eye of 660 adults with decreased visual acuity from DME involving the macular center was assigned randomly to a standardized treatment protocol of aflibercept, bevacizumab, or ranibizumab. Follow up visits occurred every 4 weeks. The primary outcome was change in visual acuity at 1 year. Secondary outcomes included change in central subfield thickness on optical coherence tomography.

Results: The mean change in VA letter score at one year was greater with aflibercept (+13.3) than bevacizumab (+9.7) or ranibizumab (+11.2). The greater overall effect was driven by eyes with initial VA 20/50 or worse (50% of the cohort). Mean VA letter score improvement in this subgroup was +18.9 aflibercept, +11.8 bevacizumab, +14.2 ranibizumab (P-values: aflibercept-bevacizumab <0.001, aflibercept-ranibizumab =0.003, Ranibizumab-bevacizumab=0.21). The mean letter score difference between aflibercept and bevacizumab of +6.5 equates on a patient level to 63% relatively more aflibercept than bevacizumab-treated eyes improving ≥ 15 letters (improvement 67% versus 41%); +4.7 letter mean difference between aflibercept and ranibizumab equates to 36% relatively more aflibercept than ranibizumab-treated eyes (improvement 68% vs. 50%). For eyes with initial VA, 20/32 to 20/40 mean change in visual acuity was the same for all three drugs.

Conclusion: In eyes with decreased VA from DME, all three agents on average substantially improve VA. However, the relative effect depends on initial visual acuity. When initial visual acuity loss is mild, on average, there were no apparent differences between the three treatment groups. However, the worse the initial VA, the greater the relative advantage of aflibercept over the two agents.

Discussant: David Sarraf*
Scientific Program

POSTER ABSTRACTS

PLEASE NOTE THE FOLLOWING PROGRAM KEY

**Bold** = AOS Member

* = Financial Disclosure

Posters will be displayed on Friday, May 15 and Saturday, May 16. Poster authors will be available to discuss their work on Saturday, May 16 from 9:15 AM – 10:00 AM.
THE ROLE OF LYMPHATIC VESSELS IN CORNEAL ALLOGRAFT REJECTION

Romulo Albuquerque*, Woodford S. Van Meter, Jayakrishna Ambati*

Purpose: Successful corneal transplantation results in part from the avascularity of the cornea. Clinical studies and animal models of corneal allografts have linked both hemangiogenesis (blood vessels) and lymphangiogenesis (lymphatics) to increased rejection. The precise contribution of each of these two vasculature systems to allograft rejection is unclear. A variant of VEGF receptor-2, soluble VEGFR-2 (sVEGFR-2), has been described which specifically blocks lymphangiogenesis without affecting blood vessels. We evaluated the ability of sVEGFR-2 to block lymphatic vessels and the effect of inhibiting lymphangiogenesis on allograft rejection.

Methods: PCR was used to detect sVEGFR-2 mRNA and protein in the cornea of mouse, humans and other mammals. A tissue-specific genetic ablation system was used to delete sVEGFR-2 in the mouse cornea. The effects of this deletion were studied in murine models of suture injury and corneal transplantation. Rejected human allografts were studied for the presence of sVEGFR-2 and lymphatic vessels.

Results: Elimination of sVEGFR-2 genetic ablation resulted in spontaneous invasion of lymphatic vessels, but not blood vessels, into the mouse cornea at birth. Increased sVEGFR-2 by overexpression in mice reduced suture-induced corneal lymphangiogenesis by 70% with no effect on hemangiogenesis (P<0.05). sVEGFR-2 administration inhibited the invasion of lymphatic but not blood vessels into the donor bed and resulted in doubled allograft survival time (P<0.05). Rejected human corneal allografts that had ingrowth of lymphatic vessels lacked sVEGFR-2.

Conclusion: Endogenous sVEGFR-2 is a pure lymphangiogenesis inhibitor. sVEGFR-2 is required for the development of an alymphatic cornea and it is evolutionarily conserved in mammals. Uncoupling the two circulatory systems suggests that specific inhibition of lymphangiogenesis ALONE reduces allograft rejection. Corneas treated with sVEGFR-2 remained clear without inflammation despite the presence of blood vessels. sVEGFR-2 can be a therapeutic modality for reducing corneal allograft rejection and has potential use as a biomarker of early allograft rejection.
IS THERE A NEED FOR INTERVAL ULTRASOUND SCANNING TO DETECT INTRAOCULAR TUMORS IN EYES WITH OPAQUE MEDIA?

Sophie J. Bakri*, Saranya Balasubramanian

Purpose: To study the prevalence of intraocular tumors detected by screening ultrasonography in eyes with opaque media.

Methods: Retrospective review of ultrasounds done in 119 eyes with opaque media and the diagnosis of blindness or phthisis between January 1, 1994 and December 31, 2013. Data were extracted on visual acuity, IOP, presence or absence of ocular pain, etiology of opaque media, number of ultrasounds received during study time period, and ultrasound findings. Follow up was defined as the time range for which an eye was followed from initial documentation of opaque media to last visit with opaque media. In addition, ultrasounds obtained for screening prior to evisceration or enucleation was noted along with pathology findings.

Results: A total of 173 ultrasounds corresponding to 119 eyes were reviewed. No intraocular tumors were detected. Mean age of patients was 59 years. Visual acuity was hand motions or worse in 89 eyes (74.8%), elevated IOP was found in 23 eyes (19.3%) and ocular pain was noted in 30 eyes (25.4%). 69 eyes with opaque media (58%) had at least one year follow up from initial visit where opaque media was noted. The mean follow up was 65 months (median 56 months; range 12-129). Of these, 2 eyes (2.9%) had an annual ultrasound, 43 eyes (62%), had an ultrasound done every 13-60 months, and 19 eyes (27.5%) had an ultrasound every 61-120 months. In addition, 16 eyes with opaque media for at least 6 years only received an ultrasound at presentation (11 eyes had 6-8 years follow up; 5 eyes had >8 years of follow up). 6 eyes had screening ultrasonography prior to evisceration or enucleation, with pathology clear of intraocular tumors.

Conclusion: In this series of eyes with opaque media, no intraocular tumors were detected by screening ultrasonography.
IMPAIRED LYSOSOMAL AND MITOCHONDRIAL FUNCTION IN EXFOLIATION GLAUCOMA

Audrey Bernstein*, Andrew Want*, Stephanie Gillespie*, J. Mario Wolosin*, Robert Ritch*

Purpose: In the eye, exfoliation syndrome (XFS) is characterized by the aggregation of disorganized microfibrils (exfoliation material, XFM). Deposition of XFM and pigment in the aqueous outflow pathway leads to chronic intraocular pressure elevation leading in turn to glaucoma. Similar to other age-related diseases in which protein aggregates cause disease, we hypothesize that lysosomal and mitochondrial dysfunction lead to XFS pathology.

Methods: Tenon fibroblasts (TFs) were explanted from tissue discards obtained from older, age-matched XFS and primary open-angle glaucoma (POAG) patients who underwent trabeculectomy surgery and from young healthy donors who underwent strabismus surgery. Cell size and mitochondrial membrane potential (JC1 dye) were quantified by flow cytometry. Lysosomes and microtubules were immunodetected with Lamp-1 and β-tubulin antibody, respectively. Culturing TFs in media with stabilized vitamin C for 1 month generated self-synthesizing 3D gels.

Results: Normally, under conditions of nutrient deprivation, lysosomes become perinuclear, where they fuse with autophagosomes, clearing the cells of waste. In XFS TFs compared to POAG TFs and healthy TFs, lysosomes did not relocalize in response to changes in nutrient conditions, suggesting that lysosomal degradation is impaired in these cells. In 3D culture, XFS TFs demonstrated a disorganized morphology with elevated protein expression of XFM-containing proteins LOXL1 and Fibulin-5. Consistent with impaired lysosomal degradation a) the percent of cells displaying depolarized mitochondria was 10x higher in XFS than in POAG TFs (26 % vs. 2%, p < 0.01) and b) the build up of intracellular organelles led to a 1.7-fold increase in XFS cell size.

Conclusion: Our findings suggest that lysosomes and mitochondria are compromised in XFS TFs, leading to a toxic environment. This may lead to reduced degradation and increased secretion of XFM aggregates. This represents the first intracellular pathologic findings reported in XFS.
MATHEMATICAL ANALYSIS OF ALEXIDINE ABSORPTION BY HIGH DENSITY POLYETHYLENE PLASTIC BOTTLES AND THE WORLDWIDE RENU-RELATED FUSARIUM KERATITIS EVENT OF 2004-2006

John D. Bullock, Harry J. Khamis, Ronald E. Warwar

Purpose: In May 2006, Bausch & Lomb was cited by the Food and Drug Administration for improper storage/transport temperatures of ReNu with MoistureLoc (RML) multi-purpose contact lens solution [1]. The Centers for Disease Control and Prevention suggested disinfection failure as the cause of this event [2]. RML contained the antimicrobial agent, alexidine (0.00045% = 4.5 parts per million [PPM]). In our previous studies: heating (56oC) RML in its bottle resulted in its decreased ability to inhibit Fusarium organisms [3]; and, Fourier transform infrared (FTIR) spectroscopy showed that alexidine absorbed into the wall of the RML polyethylene bottle [4]. The purposes of the present study were to measure alexidine concentrations over time and mathematically correlate them with our previous FTIR spectroscopic and microbiological studies.

Methods: Triplicate alexidine levels (initially, 4.5 PPM) were measured by liquid chromatography/mass spectroscopy in heated (56oC)/unheated RML bottles stored for six hours to four weeks. Using a Gauss-Newton iterative least squares nonlinear regression estimation procedure (Statistical Analysis System [SAS]), alexidine loss, L, was fit to an exponential saturation curve, $L = S(1-e^{-kt})$, where S is the alexidine saturation level, k is a function of storage temperature, and t is time.

Results: The ratio of heated:unheated alexidine loss, calculated by integrating the exponential functions, was 3.0, equivalent to that previously determined by FTIR spectroscopy (3.1). Over 95% of the alexidine was lost from the heated solution at one week. When the alexidine concentration decreases to < 0.8 PPM, the solution fails to inhibit Fusarium organisms.

Conclusion: These studies signify that temperature-enhanced alexidine-polyethylene interaction was the pharmaceutical failure mechanism of the Fusarium keratitis event of 2004-2006.
EVALUATION OF OPTIC NERVE GLIOMA SERIES AT THE ARMED FORCES INSTITUTE OF PATHOLOGY SUGGESTS POSSIBLE INTERVENTIONS IN CELLULAR SENESCENCE AND MICROGLIAL PATHWAYS (AN AOS THESIS)

J. Douglas Cameron, Fausto Rodrigues, Elisebeth Rushing, Iren Horkayne-Szakaly, Charles Eberhart

**Purpose:** To describe the demographic and clinical characteristics of an optic nerve glioma case series; to describe the historical context of tissue evaluation techniques from museum to molecular at the AFIP; and identify molecular factors in senescence and microglial pathways with treatment potential.

**Methods:** Cases were retrieved from the Armed Forces Institute of Pathology Registry of Ophthalmic Pathology. Clinical information was tabulated. In specimens with sufficient tissue, a tissue microarray (TMA) was constructed to conduct molecular studies.

**Results:** Ninety-two cases were included: gender distribution was M:F - 1.6 (2 months to 50 years) (average 10.8 years). NF1 was identified in 10 (10.8%) cases. The majority presented with decreased vision and exophthalmos. Forty-eight cases were studied by a tissue microarray construction. Glial fibrillary acidic protein (GFAP), a control for immunoreactivity, was positive in 46 (96%) cases. Immunoreactivity for p16 protein was seen in 36 (75%) cases and CD68 positive cells in 34 (71%). Limitations include referral bias, limited clinical information, limited amount of tissue, and extended period of tissue preservation.

**Conclusion:** ONG is a tumor of the visual axis in young individuals, which is generally indolent but with a variable clinical course. Traditional histopathologic techniques have not been reliably predictive of clinical course. This microarray provides representative demographic, clinical and histologic characteristics for ONG. Immunoreactivity to P16 protein and CD68 are positive in the majority. These findings suggest a possible explanation for the variable clinical course and identify therapeutic targets in the cell senescence and microglial pathways.
OCT AND VISUAL RESULTS AT SIX MONTHS AFTER TRANSITIONING TO AFLIBERCEPT FOR PATIENTS ON PRIOR RANIBIZUMAB OR BEVACIZUMAB TREATMENT FOR EXUDATIVE AMD (AN AOS THESIS)

Clement Chan*, Atul Jain*, Srinivas Sadda*, Neeta Varshney*

Purpose: To study the OCT and vision outcomes and complications at 6 months (mo) after transitioning from intravitreal ranibizumab or bevacizumab, or both to aflibercept for eyes with exudative age-related macular degeneration.

Methods: This retrospective study adhered to strict inclusion and exclusion criteria, and all conditions that could confound results were excluded. Single masked investigator performed all OCT measurements by Simplified Method per Heussen et al.1 All adverse events were recorded.

Outcome measures included the following: Macular Volume; central-1 and 3-mm subfields; subretinal fluid (SRF), cystoid macular edema (CME) and pigment epithelial detachment (PED) heights (Ht) and volumetrics (Vol); best spectacle and pinhole VA for each visit.

Results: From 11/11 to 2/13, 189 eyes (E) in 172 patients (mean age:83.4; 66 men) receiving ranibizumab (84E), bevacizumab (95E), or Mixed Group (both drugs) (10E) were transitioned (Tx) to aflibercept and followed for 6 mo. Overall mean pre-Tx and post-Tx injection frequencies in 6 mo were 6.5 and 5.4, respectively. Baseline characteristics were comparable among all groups. For entire cohort, significant decreases were noted for post-Tx vs pre-Tx SRF/CME/PED Ht and Vol (all p<.001). Post-Tx vs pre-Tx VA were (20/48 vs 20/58, p<.001). Sub-group analysis showed no differences between bevacizumab and ranibizumab in improved post-Tx SRF/CME/PED Ht and Vol (all p≤.001). Post-Tx VA, SRF/CME/PED Ht and Vol were improved for Nonresponders (suboptimal response to bevacizumab or ranibizumab), (p≤.001), but not for Responders (good response to same) at 6 mo. Only adverse event was RPE tear in 1E.

Conclusion: Study eyes showed significant improvements in all OCT measures and vision at 6 mo after transitioning from bevacizumab or ranibizumab to aflibercept. VA and OCT metrics were improved for Nonresponders and maintained for Responders. Post-Tx adverse events were uncommon.
CHALAZIA ASSOCIATED WITH INTRAVENOUS BORTEZOMIB FOR TREATMENT OF MULTIPLE MYELOMA

Frederick W. Fraunfelder, Matthew Benage, Kell Yang

Purpose: To report an association between chalazia and intravenous bortezomib treatment for multiple myeloma.

Methods: Spontaneous reports from World Health Organization (WHO) (Uppsala Monitoring Centre, Uppsala, Sweden) as well as Medline literature search using the keywords Chalazia, Bortezomib, and Myeloma.

Results: A total of 24 cases are reported from the WHO Monitoring Centre and two case series. 14 cases of chalazia were reported to the WHO monitoring Centre, with 5 female cases, 8 male cases, and 1 of unknown gender. 5 cases reported positive re-challenge and 2 cases reported positive de-challenge. Grob et al, reported 6 cases of chalazia following bortezomib (4 females and 2 males) with an average onset of 3.3 months. 5 cases reported positive de-challenge. Furthermore, Mundia, et al, reported 4 cases of chalazia with an 11-22 month time course.

Conclusion: Because of the large number of cases amassed among all three groups and the striking finding that the WHO study and the Mass Eye and Ear study both produced a large number of positive de-challenge cases, we conclude that there is a likely association between chalazia and intravenous bortezomib for treatment of multiple myeloma.
HEMORRHAGIC RISK OF VITREORETINAL SURGERY IN PATIENTS MAINTAINED ON NOVEL ORAL ANTICOAGULANT THERAPY (NOACS)

M. Gilbert Grand, Harpreet S. Walia

Purpose: To evaluate the frequency and type of perioperative hemorrhagic complications associated with vitreoretinal surgery in patients undergoing systemic treatment with the newer anticoagulant and antiplatelet agents (NOACS) including rivaroxaban, apixaban, dabigatran and prasugrel.

Methods: Retrospective review of a cohort of patients being treated with anticoagulant and antiplatelet drugs who underwent any vitreoretinal surgical procedure over a two year period.

Results: Thirty-six eyes of 33 patients on these medications underwent vitreoretinal surgical operations. No eyes suffered perioperative complications of retrobulbar hemorrhage, suprachoroidal hemorrhage, subretinal hemorrhage or intraoperative bleeding. Four eyes (11.1%) experienced postoperative vitreous cavity hemorrhage; two of these eyes (5.5%) required repeat surgical intervention and two eyes (5.5%) cleared the hemorrhage spontaneously.

Conclusion: This is the first report describing the frequency and type of hemorrhagic complications occurring in patients undergoing vitreoretinal surgery while on therapy with NOACS drugs. None of our patients experienced intraoperative hemorrhagic complications. The postoperative vitreous hemorrhage rate was consistent with rates reported in patients undergoing similar surgery while anticoagulated with warfarin. Our findings suggest that patients may safely undergo vitreoretinal surgery while maintaining therapy with rivaroxaban, apixaban, dabigatran and prasugrel (NOACS). Decisions to modify anticoagulation may have serious implications and should be made on an individualized basis. Patients need to be informed of hemorrhagic risks associated with vitreoretinal surgery.
OCULAR PERFUSION PRESSURE VERSUS ESTIMATED TRANS-LAMINA CRIBROSA PRESSURE DIFFERENCE IN GLAUCOMA. THE CENTRAL INDIA EYE AND MEDICAL STUDY (AN AOS THESIS)

Jost B. Jonas, Ningli Wang, Vinay Nangia

Purpose: To test the hypothesis whether taking trans-lamina pressure difference into the consideration changes associations between ocular perfusion pressure and glaucomatous optic neuropathy.

Methods: The population-based Central India Eye and Medical Study included 4711 subjects. Ocular perfusion pressure was calculated as $2/3 \times (\text{diastolic blood pressure} + \frac{1}{3} \times (\text{systolic blood pressure} - \text{diastolic blood pressure}) - \text{Intraocular pressure (IOP)}$. Cerebrospinal fluid pressure [mmHg] was estimated as $0.44 \times \text{Body Mass Index [kg/m}^2]+0.16 \times \text{Diastolic Blood Pressure [mmHg]}-0.18 \times \text{Age [Years]}-1.91$. Trans-lamina pressure difference was $\text{IOP} - \text{cerebrospinal fluid pressure}$.

Results: In multivariate analysis, higher open-angle glaucoma prevalence was associated with higher IOP ($P<.001; \text{odds ratio (OR):1.19}; 95\% \text{ confidence interval (CI):1.15,1.24}$) or with higher trans-lamina pressure difference ($P<.001; \text{OR:1.15}; 95\% \text{CI:1.10,1.19}$), but not with ocular perfusion pressure ($P<.37$). A smaller neuroretinal rim area was correlated with higher IOP ($P<.001; \text{standardized coefficient beta:-0.09}$) or larger trans-lamina pressure difference ($P<.001; \text{beta:-0.10}$), but not with ocular perfusion pressure ($P=.26$). Greater prevalence of angle-closure glaucoma was associated with higher IOP ($P<.001; \text{OR:1.22}; 95\% \text{ CI:1.15,1.28}$) or higher trans-lamina pressure difference ($P<.001; \text{OR:1.19}; 95\% \text{CI:1.13,1.25}$) or lower ocular perfusion pressure ($P<.04; \text{OR:0.95}; 95\% \text{CI:0.90,0.996}$). Correlation coefficients were highest for the association with IOP and lowest for ocular perfusion pressure. A smaller rim area was correlated with higher IOP ($P<.001; \text{beta:-0.08}$) and higher trans-lamina pressure difference ($P<.001; \text{beta:-0.08}$); rim area and ocular perfusion pressure were not significantly associated ($P=.25$).

Conclusion: The present study provides information of the relationship of trans-lamina pressure difference to the development of optic nerve damage in what is presently called glaucoma. It does not provide support of the idea that ocular perfusion pressure plays a major role in the pathogenesis of optic neuropathy.
ROLE OF INTRARETINAL NITRIC OXIDE IN THE DEVELOPMENT OF DIABETIC RETINOPATHY

Jennifer J. Kang-Mieler*, William F. Mieler*

Purpose: The goal of this study was to directly measure in vivo retinal nitric oxide (NO) concentration in experimental early diabetic retinopathy and to determine how intraretinal NO changes with severity of diabetes.

Methods: Long-Evans rats were made diabetic with streptozotocin (STZ). Three weeks post-STZ injection, intraretinal NO concentration profiles were recorded using a dual NO/electroretinogram microelectrode. Diabetic profiles were compared to profiles from healthy controls, healthy rats injected with the NO synthase inhibitor L-NG-nitroarginine methyl ester (L-NAME), and healthy rats that received acute glucose injections (acute hyperglycemia). NO values at the retina/RPE boundary (100% retinal depth) and retinal surface (0% depth) were analyzed for correlation with blood glucose.

Results: The average NO concentrations in the outer retina, inner retina, and vitreous humor of mild diabetics (250-400 mg/dL) were significantly higher than control by 73%, 47%, and 70%, respectively. The average NO concentrations in the outer retina, inner retina, and vitreous humor of severe diabetics (500-600 mg/dL) were lower than control with NO at 41%, 36%, and 36% of control, respectively. Severe diabetic NO profiles were also similar to L-NAME treated eyes. NO levels in moderate diabetics (400-500 mg/dL) and acute hyperglycemia rats were similar to control. NO was significantly and inversely correlated with blood glucose for diabetic rats at 100% depth ($R=-0.91$) and 0% depth ($R=-0.79$) but not for acute hyperglycemia rats.

Conclusion: The higher-than-control level of NO in mild diabetics and lower-than-control level in severe diabetics show that severity of diabetes may be an important factor in the development of early stages of diabetic retinopathy.
STEROID DIFFERENTIATION: THE SAFETY PROFILE OF VARIOUS STEROIDS ON RETINAL CELLS IN VITRO AND THEIR IMPLICATIONS FOR CLINICAL USE (AN AOS THESIS)

Baruch D. Kuppermann, Leandro Zacharias, Cristina M. Kenney

Purpose: To determine if potentially viable alternatives to the clinical use of intravitreal triamcinolone acetonide should be considered based on a comparative assessment of the in vitro effects of five commercially available corticosteroids. We hypothesized that dexamethasone, betamethasone, methylprednisolone, loteprednol etabonate, and fluocinolone acetonide, at clinically relevant doses, may show different levels of in vitro cytotoxicity to retinal cells.

Methods: Cultures of human retinal pigment epithelial cells (ARPE-19) and rat embryonal neurosensory precursor retinal cells (R28) were treated with dexamethasone, betamethasone, methylprednisolone, loteprednol, or fluocinolone acetonide. Cell viability as a measure of cell death was determined by trypan blue dye exclusion assay. The mechanical effect of drug crystals was evaluated by solubilizing the steroid formulations. Mitochondrial dehydrogenase and membrane potential were assessed to measure cell damage.

Results: Betamethasone, loteprednol, and methylprednisolone, in commercially available forms, caused significant cytotoxic changes to retinal cells in vitro at clinically relevant doses. This effect was less pronounced with solubilized betamethasone. Dexamethasone at concentrations up to 5 times the clinical dose of free drug injections and 1000 times greater than a drug implant did not cause decreased cell viability. Fluocinolone acetonide at doses 1000 times higher than observed with drug delivery systems showed no cytotoxic effect.

Conclusion: Betamethasone, loteprednol, and methylprednisolone exhibited cytotoxicity at clinically relevant doses and do not appear to be good therapeutic options for intravitreal use. In comparison, dexamethasone and fluocinolone acetonide, which exhibited fewer cytotoxic effects than other steroids, may be potentially viable alternatives to triamcinolone acetonide for clinical use.
**ENDOGENOUS ENDOPHTHALMITIS – ONE EYE FOLLOWED BY THE OTHER**

Sid Schechet, Jason Hong, **Vinod Lakhanpal**

**Purpose:** To describe an unusual case of culture-proven bilateral endogenous endophthalmitis secondary to an underlying psoas abscess. The patient first was found to have endophthalmitis in the left eye, and, in spite of being on the appropriate intravenous antibiotics, he subsequently developed endophthalmitis in the right eye.

**Methods:** A 54 year-old male with no ocular history presented to the emergency room with lower abdominal pain. He was found to have an ST-elevation myocardial infarction. After undergoing coronary stenting, he developed Methicillin- sensitive staph aureus (MSSA) sepsis. While on appropriate intravenous antibiotics he developed endophthalmitis in the left eye (VA count fingers) followed two days later by endophthalmitis in the right eye (VA 20/200).

**Results:** Within 24 hours of reported visual symptoms, and presumed left endophthalmitis, he underwent vitreous tap and vitrectomy with intravitreal Vancomycin and Amikacin. Vitreous cultures grew MSSA. Two days later he developed MSSA culture-positive endophthalmitis in the right eye despite being on IV antibiotics, and he was treated with intravitreal antibiotics. CT scan of the abdomen revealed a psoas abscess which was drained and also found to be MSSA. Three weeks later his VA improved to 20/40 OD and 20/25 OS.

**Conclusion:** This case demonstrates that despite being on the appropriate intravenous antibiotics, patients can still develop endophthalmitis due to poor ocular penetration of the antibiotics. Early recognition of this disease with aggressive management of vitreous tap and antibiotic injection with or without vitrectomy should be considered to ensure a successful visual outcome. Close follow up and communication with the primary team is vital in terms of locating and treating the underlying pathology.
AN ANALYTICAL REPORT OF PUBLICATION PRODUCTIVITY FOR 748 ACADEMIC OPHTHALMOLOGISTS AND 37 DEPARTMENTS IN THE SOUTHERN REGION OF THE UNITED STATES

Craig R. Thiessen, Garrett T. Venable, Nick C. Ridenhour, Natalie C. Kerr

**Purpose**: Bibliometrics, a statistical method to analyze scientific literature, has yet to be applied to academic ophthalmology departments. While many benchmarking methods have been proposed, the h-index has been most widely accepted. The h-index samples a researcher’s publication quantity while controlling for a measure of quality. The m-quotient adjusts the h-index according to the number of years elapsed in the field. We measured the publication productivity of academic ophthalmology departments in the Southern region of the United States.

**Methods**: Bibliometric profiles were created for 748 ophthalmologists from 37 (of 39) nonmilitary departments in the Southern United States. Profiles included the h-index and m-quotient, which were calculated from the citation database, Scopus. Comparisons between academic rank (i.e. chairman, professor, associate, assistant, and instructor), subspecialty, and gender were also performed. Departments were ranked by the summation of h-indices for each member in a department and also by mean h-index for the whole department.

**Results**: The median h-index and m-quotient were 10.16 and 0.53 respectively. Both of these values exhibit a positive relationship with increasing academic rank (p < 0.001). Ophthalmologists with subspecialties in pathology, neurology, vitreoretina, cornea and external disease, and glaucoma had higher median h-indices than those in uveitis, pediatrics, oculoplastics, comprehensive, and oncology. Males demonstrated a significantly higher mean h-index (11.55, n=523) than females (mean = 6.91, n=225) after correction for academic rank (p = 0.001). However, there was no significant difference in m-quotients between genders. Ranked by summed h-indices, the top 5 programs for publication productivity in the Southern region of the U.S. in descending order were University of Miami, Johns Hopkins University, Duke University, Baylor College of Medicine, and Emory University.

**Conclusion**: This report presents the first detailed publication analysis utilizing bibliometrics to assess academic ophthalmology. These results provide academic benchmarks that may be used for further analysis and program development.
STEM CELL LINES FROM PATIENTS WITH THE MACULAR DEGENERATION COMPLEX

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Purpose: Genome-wide association studies (GWAS) identified DNA variants that are strong risk factors for age-related macular degeneration (AMD). One single-nucleotide polymorphism (SNP) lies in the 402H allele in the CFH gene and the three others are tightly linked and lie in the neighboring HTRA1 and ARMS2 genes. These SNPs confer the most significant genetic risk factors in the history of GWAS studies in human genetics. How these mutations might cause sight to deteriorate is unclear because the underlying molecular mechanisms of AMD are unknown.

Methods: Induced pluripotent stem (iPS) cell-derived RPE from patients provides us with earlier stage AMD patient-specific cells and allows us to analyze the underlying mechanisms at this critical time point.

Results: An unbiased proteome screen of A2E-aged patient-specific iPS-derived RPE cell lines identified SOD2-mediated antioxidative defense in the genetic allele’s susceptibility of AMD. The AMD-associated risk haplotype (T-in/del-A) impairs the ability of the RPE to defend against aging-related oxidative stress. SOD2 defense is impaired in RPE homozygous for the risk haplotype (T-in/del-A; T-in/del-A), while the effect was less pronounced in RPE homozygous for the protective haplotype (G-Wt-G; G-Wt-G). ARMS2/HTRA1 risk alleles decrease SOD2 defense, making RPE more susceptible to oxidative damage and thereby contributing to AMD pathogenesis.

Conclusion: iPS cells can be differentiated and "aged" to generate a virtually unlimited supply of RPE that models early-stage AMD (or an aged control) which risk allele drives risk for AMD can be determined by monitoring SOD2 activities as a surrogate for increased risk.
COMPARATIVE RESULTS WITH REGARDS TO HUMPHREY VISUAL FIELDS AND THE SPARCS CONTRAST SENSITIVITY TEST IN PATIENTS WITH GLAUCOMA

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Purpose: To compare visual field clusters obtained by the Humphrey visual field (HVF, 24-2 SITA Standard perimeter, Carl Zeiss Meditec, Inc., Dublin, CA) analyzer with contrast sensitivity clusters obtained by the Spaeth-Richman Contrast Sensitivity (SPARCS) test. SPARCS is a novel computerized-base test, which measures contrast sensitivity threshold of patients’ central vision and peripheral vision.1

Methods: Central, superior and inferior HVF clusters were compared with contrast sensitivity clusters obtained by SPARCS in the same regions. For each HVF and SPARCS cluster, the mean deviation (MD) or contrast sensitivity scores were averaged. Pearson correlation coefficient was calculated for each cluster.

Results: One hundred and sixty-one patients with moderate-stage glaucoma were included in the study. The mean age was 64.6 years (rang: 30-83), predominantly female (n=86, 53%). The mean MD score significantly correlated with the mean SPARCS score (HVF MD=-9.79 dB, SPARCS=11.30; r=0.62, P<0.0001). The superior and inferior clusters showed stronger correlations compared with the central cluster (inferior cluster: r=0.72, P<0.0001; superior cluster: r=0.67, P<0.0001; central cluster: r=0.46, P<0.0001).

Conclusion: Visual field MD scores significantly correlated with SPARCS scores in all tested clusters. The strongest correlations were in the superior and inferior clusters. This investigation supports our previous study, 1 showing that contrast sensitivity measured by SPARCS is a potentially useful tool in the overall assessment of patients with glaucoma.
INCIDENCE AND RISK FACTORS FOR DEVELOPING DIABETIC RETINOPATHY AMONG YOUTH WITH TYPE 1 AND TYPE 2 DIABETES THROUGHOUT THE UNITED STATES

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Purpose: Despite the rise of Type 2 diabetes mellitus (T2DM) among children and adolescents in the United States, little is known about the incidence of diabetic retinopathy (DR) among children with T2DM compared to those with Type 1 DM (T1DM) and risk factors associated with DR in youth with T2DM.

Methods: We reviewed data from a large U.S. managed care network to identify all children and adolescents age ≤21 years who were newly diagnosed with T1DM or T2DM and underwent ≥1 examination by an ophthalmologist or optometrist. Youth who developed DR were identified by ICD-9-CM diagnosis codes. Kaplan-Meier survival curves were created to depict the time from first DM diagnosis to first record of DR. Multivariable Cox regression modelling was performed to identify sociodemographic factors associated with DR development.

Results: Among the 2457 eligible youth with newly-diagnosed T1DM and 1673 with T2DM, 275 (6.7%) developed DR. The proportion of youth with T1DM and T2DM who developed DR was 9.2%, and 2.9%, respectively. The incidence rates of DR were 25.8 and 8.9 per 1000 person-years among youth with T1DM and T2DM, respectively. Youth with T1DM developed DR faster than youth with T2DM (P<0.0001, Log-Rank test). Youth with T1DM had a 322% increased hazard rate of developing DR compared to those with T2DM (HR 4.22, CI 2.98-5.99). Males had a 29% increased hazard rate of developing DR compared to females (HR 1.29, CI 1.00-1.65). For each one year age increase at time of first DM diagnosis, the hazard rate for developing DR increased by 7.7% (HR 1.08, CI 1.05-1.10).

Conclusion: Youth with T1DM and T2DM exhibit significant risk of retinopathy and should undergo regular screenings by eye care professionals to check for DR. These results will help formulate clinical practice guidelines to advise clinicians when to screen children with T2DM for DR.
CARRIER FREQUENCY OF CYP1B1 MUTATIONS IN THE UNITED STATES (AN AOS THESIS)

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*Purpose: CYP1B1 mutations cause autosomal recessive congenital glaucoma. Disease risk assessment for families with CYP1B1 mutations requires knowledge of the population mutation carrier frequency. The purpose of this study is to determine the CYP1B1 mutation carrier frequency in clinically normal individuals residing in the United States. Because CYP1B1 mutations can exhibit variable expressivity, we hypothesize that the mutation carrier frequency is higher than expected.

*Methods: Two hundred fifty individuals without glaucoma or a family history of glaucoma were enrolled. CYP1B1 mutations were identified by DNA sequencing, and pathogenicity was estimated by PolyPhen-2 or a previous report of disease causality.

*Results: Based on the disease frequency (1 in 10,000) and prevalence of CYP1B1-related congenital glaucoma (15% to 20%), the frequency of CYP1B1-related congenital glaucoma in the United States is approximately 1 in 50,000. Assuming Hardy-Weinberg equilibrium, the expected CYP1B1 mutation carrier frequency would be 1 in 112, or 0.89%. Among the 250 study participants, 11 (4.4%) are carriers of a single pathogenic mutation, representing a carrier frequency of 1 in 22, which is 5.1 times the expected frequency. A higher-than-expected carrier frequency (1 in 33, 3.0%) was also observed in 4300 white individuals sequenced by the National Heart Lung and Blood Institute Exome Sequencing Project.

*Conclusion: Our results show that the CYP1B1 mutation carrier frequency in the US population is between 1 in 22 and 1 in 33, which is 5.1 to 3.4 times the expected frequency. These results suggest that more individuals than expected are carriers of a deleterious CYP1B1 mutation, and that the prevalence of CYP1B1-related disease may be higher than expected.
CAN BENZALKONIUM CHLORIDE BE DETECTED IN THE AQUEOUS OF GLAUCOMA PATIENTS

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Purpose: Benzalkonium chloride (BAK) is used as a preservative in many glaucoma medications. In vitro studies have shown that BAK is toxic to trabecular meshwork cells. Theoretically such toxicity could lead to worsening of the glaucoma, in which case these medications should be avoided. We are unaware of any studies indicating whether BAK can be found in aqueous humor. This study was performed to determine whether BAK could be detected in the aqueous of glaucoma patients after administration of BAK containing medications.

Methods: Aqueous samples were obtained from 10 glaucoma patients who were undergoing a glaucoma drainage operation. All had been treated chronically with BAK containing medications. Additionally, a drop of a BAK containing antibiotic was instilled in the surgical eye one hour prior to surgery. After the eye was anesthetized a paracentesis was performed with a 30 gauge needle and .1 cc of aqueous was aspirated. The aqueous samples were tested with mass spectrometry to determine the presence of BAK. We determined the sensitivity of the test using serial dilutions of BAK containing eye drops.

Results: We were able to detect BAK down to a concentration of 0.1 micrograms per milliliter. The test could also detect BAK when a BAK containing eye drop was mixed with an aqueous sample. We were unable to detect BAK in any of the 10 aqueous sample obtained from the glaucoma patients.

Conclusion: Our results indicate that BAK is not present in the aqueous of glaucoma patients chronically using BAK containing medications, and, therefore, is unlikely to cause additional damage to the trabecular meshwork and worsen the glaucoma.