The American Ophthalmological Society

ONE HUNDRED AND FIFTIETH ANNUAL MEETING

Hans E. Grossniklaus ..... **PRESIDENT**
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MAY 15–18, 2014
THE RITZ-CARLTON NEW YORK, BATTERY PARK
NEW YORK, NEW YORK

The American Ophthalmological Society

Office of the Executive Vice President
Jacksonville, Florida
May 2014

THE ONE HUNDRED AND FIFTIETH ANNUAL MEETING
of the Society will be held at

The Ritz-Carlton New York, Battery Park
Thursday through Sunday
May 15–18, 2014

COMMITTEE ON PROGRAMS CHAIR
Carol L. Shields

TARGET AUDIENCE

Ophthalmologists involved in academic practice, private practice, training programs, research, or administrative health care.

MEETING OBJECTIVES

The objectives of the 2014 Annual Meeting are to:

1. Discuss historical and important advances in the prevention, cause, diagnosis, and treatment of eye diseases over the past 25 years in all subspecialties in ophthalmology.
2. Identify future basic and clinical vision research that might be transformed into improved clinical care.
3. Assess the role of past, current and future technologies in the evaluation and treatment of eye diseases.
4. Describe the historical 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 in the past, present and possible future.

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-IX 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

The entire 2014 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 15: 1:30 PM – 5:00 PM

Friday, May 16: 6:30 AM – 12:00 PM

Saturday, May 17: 6:00 AM – 12:00 PM

Sunday, May 18: 6:30 AM – 10:00 AM

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 2013 MEETING

Anthony C. Arnold	Los Angeles, California
Sophie J. Bakri	Rochester, Minnesota
Michael F. Chiang	Portland, Oregon
Christina J. Flaxel	Portland, Oregon
Karl C. Golnik	Cincinnati, Ohio
David Huang	Portland, Oregon
L. Jay Katz	Philadelphia, Pennsylvania
Steven L. Mansberger	Portland, Oregon
Timothy J. McCulley	Baltimore, Maryland
Cameron F. Parsa	Madison, Wisconsin
Louis R. Pasquale	Boston, Massachusetts
Jonathan E. Sears	Cleveland, Ohio
David R. Stager, Jr.	Plano, Texas
Joshua D. Stein	Ann Arbor, Michigan

MEMBERS ATTENDING THEIR FIRST AOS ANNUAL MEETING

Peter J. Francis (2011)	Portland, Oregon
Judy E. Kim (2012)	Milwaukee, Wisconsin
Shigeru Kinoshita (2012)	Kyoto, Japan

IN MEMORIAM

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

Bernard Becker	Saint Louis, Missouri	Joined 1960
Melvin L. Rubin	Gainesville, Florida	Joined 1975
Ronald E. Smith	Los Angeles, California	Joined 1982
Fred M. Wilson, II	Carmel, Indiana	Joined 1983

FUTURE ANNUAL MEETINGS

2015 AOS ANNUAL MEETING
The Hotel Viking
Newport, Rhode Island
May 14–17, 2015

FINANCIAL DISCLOSURES

The following are the healthcare-related financial disclosures of those involved in the preparation or presentation of this AOS event. The AOS Committee on Programs gathered this information to plan the program and has attempted to manage relevant financial disclosures 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.

Category	Code	Description
Consultant/ Advisor	C	Consultant fee, paid advisory boards or fees for attending a meeting (for the past 1 year)
Employee	E	Employed by a commercial entity
Lecture Fees	L	Lecture fees (honoraria), travel fees or reimbursements when speaking at the invitation of a commercial entity (for the past 1 year)
Equity Owner	O	Equity ownership/stock options of publicly or privately traded firms (excluding mutual funds) with manufacturers of commercial ophthalmic products or commercial ophthalmic services
Patents/Royalty	P	Patents and/or royalties that might be viewed as creating a potential conflict of interest
Grant Support	S	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

ALBINI, Thomas
C – Allergan; Bausch + Lomb; Eleven Biotherapeutics; ThromboGenics
S – Genentech

ALLOJU, Shashi
S – Research to Prevent Blindness

AREF, Ahmad
L – Alcon

ARNOLD, Anthony C.
C – Pfizer, Inc.

BEAVER, Hilary
L – Genzyme Corporation

BROWN, Gary C.
O – Center for Value-Based Medicine

BROWN, Melissa M.
O – Center for Value-Based Medicine

BROWNING, David J.
C – Alimera
S – Aerpio; Novartis; DRCR Network; Regeneron

BRUSIE, Steven
P – Steven R. Brusie, MD

CAMPOCHIARO, Peter A.
C – Advanced Cell Technology; Aerpio Therapeutics; Alimera; Applied Genetic Technologies; Gene Signal; Genentech; Kala Pharmaceuticals; Regeneron
S – Aerpio Therapeutics; Genentech; Allergan; Genzyme; GlaxoSmithKline; Oxford Biomedia

CHEN, Teresa C.
S – American Glaucoma Society Mid-Career Award; Massachusetts Lions Eye Fund; Harvard Catalyst Grant, NIH UL1 RR 025758; Agency for Healthcare Research and Quality

COLEMAN, Anne L.
S – NEI; AHRQ

COOK, Paul F.
C – Takeda, Inc.; University of Colorado Health; Covance Market Access, Inc.; Medical Simulation Corp. Inc.; Academic Impressions Inc.; Credentialing & Competency Institute, Inc.
E – University of Colorado
L – National Food Program Sponsors Association
S – Merck & Co., Inc.; National Institutes of Health; Health Resources & Services Administration; Colorado Health Foundation; Substance Abuse & Mental Health Administration

CULP, JR., W. David
C – Powered Research
E – Affinergy, LLC

DE BOER, Johannes F.
C – Harvard Medical School – CBORT Scientific Advisory Board Chair
P – Harvard Medical School – Licenses to NIDEK, Inc, Terumo, Ninepoint Medical

EAGLE, JR., Ralph C
O – Merck

EDWARD, Deepak P.
S – United States Air Force Grant, Department of Defense

ELMAN, Michael J.
C – Genentech/Roche
O – Ohr Pharmaceuticals
S – Emmes Corp; Jaeb Center; NEI; Genentech/ Roche; iCo

FREUND, K. Bailey
C – Regeneron; Genentech; Bayer; Heidelberg; Optos

GALLEGO–PINAZO, Roberto
C – Alcon Laboratories, Inc.; Bayer Healthcare Pharmaceuticals; Novartis Pharmaceuticals Corporation
L – Alcon Laboratories, Inc.; Bayer Healthcare Pharmaceuticals; Heidelberg Engineering; Novartis Pharmaceuticals Corporation; Topcon Medical Systems Inc.
S – Alcon Laboratories, Inc.; Allergan; Bayer Healthcare Pharmaceuticals; Carl Zeiss Meditec; Heidelberg Engineering; Novartis Pharmaceuticals Corporation; Sensimed; Topcon Medical Systems Inc.

GARDNER, Thomas W.
C – Kalvista; Aerpio

GAUDRIC, Alain
C – Novartis Pharmaceuticals; ThromboGenics
S – Bayer

HAN, Dennis P.
C – FlowOne LLC
P – RAVI–Guide

HARTNETT, M. Elizabeth
C – National Institutes of Health
S – National Eye Institute/ National Institutes of Health; March of Dimes

HUANG, David
O – Optovue
P – Optovue
S – Optovue

JACKSON, Gregory R.
E – MacuLogix
O – MacuLogix
P – MacuLogix

JAMPOL, Lee M.
C – Janssen/Quintiles
L – Novartis

JIA, Yali
P – Optovue Inc.
S – Optovue Inc.; NIH

KAHOOK, Malik Y.
S – Merck

KAMMER, Jeffrey A.
C – Allergan; Alcon; Iridex
L – Allergan
S – Merck; Transcend; Aquesys

KANG–MIELER, Jennifer J.
C – Genentech

KOCH, Douglas D.
C – Abbott Medical Optics; Alcon; Ziemer; Revision Optics
O – Optimedica
S – iOptics; Ziemer

LAUER, Andreas K.
S – Allergan; NIH; Oxford BioMedica

LEE, Andrew G.
O – Credential Protection

MANSBERGER, Steven L.
C – Alcon; Allergan; Santen; Glaukos
L – Merck
S – Merck; Allergan

MIELER, William F.
C – Genentech

PASQUALE, Louis R.
P – Louis R. Pasquale, MD; Steven M. Brusie, MD; Massachusetts Eye and Ear Infirmary

ROSEN, Richard B.
C – Optovue; Clarity
L – OD–OS
S – Optovue

SADUN, Alfredo A.
S – Edison Pharmaceuticals; Stealth Peptides; International Foundation of Optic Nerve Diseases

SEBAG, Jerry
C – ThromboGenics
L – Alcon; ThromboGenics
O – ThromboGenics

SIMAVLI, Huseyin
S – The Scientific and Technological Research Council of Turkey (TUBITAK) 2219

SINGH, Ravi S. J.
P – RAVI Guide

SPAETH, George L.
L – Merck; Allergan; Alcon
S – Merck; Allergan

SRINIVASAN, Vivek J.
P – MIT, license to Optovue Inc.

STEM, Maxwell S.
C – Kalvista

STOUT, J. Timothy
C – Oxford Biomedica; AGTC
P – Oxford Biomedica
S – Oxford Biomedica; AGTC

THOMPSON, John T.
C – Genentech (Roche)
S – Genentech (Roche); Regeneron Pharmaceuticals

TRABOULSI, Elias I.
C – Oxford Biomedica

TSE, David T.
O – Innovia
P – Innovia

WILSON, David J.
S – Oxford Biomedica; AGTC; Foundation Fighting Blindness; Research to Prevent Blindness; National Institutes of Health

YUE, Beatrice Y.
S – National Eye Institute Core Grant EU001792; Research to Prevent Blindness, Inc.

ZARBIN, Marco A.
C – Calhoun Vision, Inc.; Imagen Biotech, Inc.; Iridex; Novartis; Pfizer
P – UMDNJ–NJMS (RPE Transplant Treatment)

NO FINANCIAL RELATIONSHIPS TO DISCLOSE

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AL JASTANEIAH, Sabah
AL KATAN, Hind
ALPER, Stephen
AL ZOBIDI, Mohammed
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BARB, Scott M.
BARKER, Gordon T.
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BERINGER, Joseph
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BOYNTON, Grace E.
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CHEW, Emily Y.
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COSTA, Roberta M. S.
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CUNHA DE SOUZA, Eduardo
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GUTHRIE, Micah J.
GUTMAN, Froncie A.
HAMMOND, Scott
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KAFRI, Tal
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TANNA, Angelo P.
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TSIKATA, Edem
VAJARANANAT, Thasarat
VAN METER, Woodford S.
WALSH, Joseph B.
WANG, Haibo
WILENSKY, Jacob
WILSON, M. Edward
YANG, Zhihong
YANNUZZI, Lawrence A.
YU, Suqin

American Ophthalmological Society
Annual Meeting

Event Schedule

THURSDAY, MAY 15

12:00 PM – 1:30 PM	New Member Luncheon (by invitation)	Rise I and II (14th Floor)
1:30 PM – 5:00 PM	Registration	Harbor Vista Alcove
1:45 PM – 3:45 PM	New Member Spotlight Presentations	Manhattan Ballroom
6:00 PM – 7:30 PM	Reception for New Members (black tie optional)	Manhattan Ballroom/ Skyline Vista

FRIDAY, MAY 16

6:30 AM – 12:00 PM	Registration	Harbor Vista Alcove
6:30 AM – 8:00 AM	Continental Breakfast	Harbor Vista
7:30 AM – 9:30 AM	Spouse/Personal Guest Hospitality Lounge	Manhattan Ballroom
7:30 AM – 12:00 PM	AOS-Knapp Symposium	Salons II and III
10:00 AM – 10:30 AM	Coffee Break & Poster Viewing Session	Salon I
6:00 PM – 7:30 PM	Reception (casual)	Rise I-III (14th Floor)

American Ophthalmological Society Annual Meeting

Event Schedule

SATURDAY, MAY 17

6:00 AM – 12:00 PM	Registration	Harbor Vista Alcove
6:00 AM – 8:00 AM	Continental Breakfast	Harbor Vista
6:30 AM – 7:15 AM	Executive Session (members only)	Salons II and III
7:30 AM – 9:30 AM	Spouse/Personal Guest Hospitality Lounge	Rise Ballroom (14th Floor)
7:30 AM – 10:10 AM	Ophthalmic Subspecialty Presentations	Salons II and III
10:10 AM – 10:40 AM	Coffee Break & Poster Viewing Session	Salon I
10:40 AM – 12:00 PM	AOS History Presentations*	Salons II and III
12:00 PM – 1:30 PM	Emeritus Luncheon (by invitation)	Rise Ballroom (14th Floor)
6:30 PM – 7:30 PM	Reception (black-tie preferred)	Harbor Vista
7:30 PM – 10:30 PM	Banquet (black-tie preferred)	Ritz Carlton Ballroom

* Spouses and guests are encouraged to attend these special presentations on the history of the Society

SUNDAY, MAY 18

6:30 AM – 10:00 AM	Registration	Harbor Vista Alcove
6:30 AM – 8:00 AM	Continental Breakfast (Spouses & Personal Guests welcome)	Harbor Vista
7:30 AM – 10:00 AM	Ophthalmic Subspecialty Presentations	Salons II and III

Scientific Program

SYMPOSIUM AGENDAS AND POSTER ABSTRACTS

The following abstracts of posters selected to be presented at the meeting have been printed as received. Scientific sessions will be held in Salons II, III.

Presentations are limited to the time indicated in the program. If time permits, brief discussions or comments will be permitted.

PLEASE NOTE THE FOLLOWING PROGRAM KEY

Bold = AOS Member
♦ = Financial Disclosure

(Presenters will indicate their financial disclosure verbally and in the first slide.)

Knapp Symposium

Friday, May 16

OCULAR GENE THERAPY

7:30 AM	*Welcome to the 150th Anniversary Meeting* **Hans E. Grossniklaus**
7:35 AM	*Introduction of Verhoeff Lecture* – **David J. Wilson♦**
7:45 AM	*Frederick Verhoeff Lecture* *The Rocky Road to Successful Human Gene Therapy* **J. Timothy Stout♦** Baylor College of Medicine Houston, Texas
8:25 AM	*Introduction of AOS-Knapp Symposium* – **Edward G. Buckley**
8:27 AM	*Brief History of the Knapps* **Froncie A. Gutman**
8:30 AM	*Introduction of Genetic Eye Disease* **Edwin M. Stone** University of Iowa Hospitals & Clinics Iowa City, Iowa
8:50 AM	*Gene Replacement Therapy* Albert M. Maguire University of Pennsylvania School of Medicine Philadelphia, Pennsylvania
9:10 AM	*Pharmacologic Delivery with Gene Therapy* Peter A. Campochiaro♦ Johns Hopkins University School of Medicine Baltimore, Maryland
9:30 AM	*Discussion*
10:00 AM	Break and Poster Viewing
10:30 AM	*Gene Therapy vs. Stem Cell Therapy* **Marco A. Zarbin♦** Rutgers, New Jersey Medical School Newark, New Jersey
10:50 AM	*Risks of Gene Therapy* **Paul A. Sieving** National Eye Institute Bethesda, Maryland
11:10 AM	*Ethical Considerations in Gene Therapy* **Alex V. Levin** Wills Eye Hospital Philadelphia, Pennsylvania
11:30 AM	*Discussion*

Scientific Presentations

Saturday, May 17

THE HISTORY & EVOLUTION OF OPHTHALMIC SUBSPECIALTIES: PAST, PRESENT & FUTURE

6:30 AM	*Executive Session* (Members Only)
7:30 AM	*Welcome* – **Hans E. Grossniklaus**
7:32 AM	*Introduction* – **Carol L. Shields**

Moderators: **Hans Grossniklaus, Carol Shields, David Tse**

7:35 AM	*Presentation of Member Interview Videos* – **Jay C. Erie**
7:40 AM	*AOS Leadership Video:* **Hugh Taylor**
7:43 AM	*Glaucoma* – **George L. Spaeth**
8:10 AM	*AOS Leadership Video:* **Al Sommer**
8:13 AM	*Retina* – **Lee M. Jampol**
8:40 AM	*AOS Leadership Video:* **Susan H. Day**
8:43 AM	*Neuro-Ophthalmology* – **Alfredo A. Sadun**
9:10 AM	*AOS Leadership Video:* **Richard K. Parrish, II**
9:13 AM	*Oculoplastics* – **George B. Bartley**
9:40 AM	*AOS Leadership Video:* **John G. Clarkson**
9:43 AM	*Ocular Oncology* – **Carol L. Shields**
10:10 AM	Break and Poster Viewing

Moderators: **Hans Grossniklaus, Edward Buckley**

10:40 AM	*AOS Leadership Video:* **Dan B. Jones**
10:43 AM	*Introduction of 150th Anniversary Lecturer* – **Hans E. Grossniklaus**
10:48 AM	*150th Anniversary Lecture: History of the American Ophthalmological Society* – **Daniel M. Albert**
11:18 AM	*Pictoral History of the American Ophthalmological Society* – **Ralph C. Eagle, Jr.**

Scientific Presentations

Sunday, May 18

THE HISTORY & EVOLUTION OF OPHTHALMIC SUBSPECIALTIES: PAST, PRESENT & FUTURE

Moderators: **Hans Grossniklaus, Jerry Sebag**

7:30 AM	*AOS Leadership Video:* **Lee M. Jampol**
7:33 AM	*Ophthalmic Pathology* – **Hans E. Grossniklaus**
8:00 AM	*AOS Leadership Video:* **William S. Tasman**
8:03 AM	*Pediatric Ophthalmology & Strabismus* – **Marilyn T. Miller**
8:30 AM	*AOS Leadership Video:* **Bruce E. Spivey**
8:33 AM	*Cornea* – **Ivan R. Schwab**
9:00 AM	*AOS Leadership Video:* **Paul R. Lichter**
9:03 AM	*Cataract & Refractive Surgery* – **Douglas D. Koch**
9:30 AM	*Closing Remarks* – **Hans E. Grossniklaus**

Scientific Program

POSTER ABSTRACTS

PLEASE NOTE THE FOLLOWING PROGRAM KEY

Bold = AOS Member
♦ = Financial Disclosure

Posters will be displayed on Friday, May 16 and Saturday, May 17. Poster authors will be available to discuss their work on Saturday, May 17 from 10:10 AM – 10:40 AM.

1

AMERICAN JOINT COMMITTEE ON CANCER (AJCC) CLASSIFICATION OF POSTERIOR UVEAL MELANOMA (ANATOMIC STAGE) PREDICTS PROGNOSIS IN 7731 PATIENTS

Carol L. Shields, Swathi Kaliki, Minoru Furuta, Enzo Fulco, Carolina Alarcon, **Jerry A. Shields**

Purpose: To analyze prognosis of posterior uveal melanoma based on the American Joint Committee on Cancer (AJCC) (7th edition) tumor staging.

Methods: Retrospective series of 7731 patients for outcomes of melanoma-related metastasis and death.

Results: The AJCC tumor staging was stage I in 2767 (36%), stage II in 3735 (48%), stage III in 1220 (16%), and stage IV in 9 (<1%). Based on tumor staging I, II, III, and IV respectively, features that showed significant ($p<0.05$) increase with tumor staging included age at presentation (years) (57, 58, 60, 60), tumor base (mm) (8, 12, 17, 17), tumor thickness (mm) (2.9, 6.0, 10.1, 10.2), distance to optic disc (mm) (3, 5, 5, 5), distance to foveola (mm) (3, 5, 5, 5), mushroom configuration (6%, 24%, 34%, 33%), plateau configuration (3%, 4%, 7%, 11%), tumor pigmentation (48%, 53%, 69%, 78%), and extraocular extension (0%, 1%, 11%, 22%). Following therapy, Kaplan-Meier estimates of metastasis at 5, 10, and 20 years were 5%, 12%, and 20% for stage I, 17%, 29%, and 44% for stage II, 44%, 61%, and 73% for stage III, and 100% by 1 year for stage IV. Kaplan-Meier estimates of death at 5, 10, and 20 years were 3%, 6%, and 8% for stage I, 9%, 15%, and 24% for stage II, 27%, 39%, and 53% for stage III, and 100% by 1 year for stage IV. Compared to stage I, the hazard ratio for metastasis/death for stage II was 3.1/3.1, and for stage III was 9.3/10.1.

Conclusions: Based on the AJCC tumor stage I, the rate of metastasis/death was 3 times greater for stage II, 9 to 10 times greater for stage III, and further greater for stage IV. The risk for metastasis and death increased three-fold with each increasing melanoma staging.

2

INNER RETINAL SENSORY NEUROPATHY IN TYPE 1 DIABETES MELLITUS

Maxwell S. Stem♦, Grace E. Boynton, Ashley Thompson, Naheed W. Khan, Gregory R. Jackson♦, Rodica Pop-Busui, **Thomas W. Gardner♦**

Purpose: To quantify differences in inner and outer retinal function between patients with type 1 diabetes mellitus (T1DM) with no to mild diabetic retinopathy (DR) and healthy controls

Methods: We examined 29 adults with T1DM and no to minimal DR and 25 age-matched healthy controls. Participants underwent a complete ocular examination, measurement of best-corrected visual acuity, and fundus photography for retinopathy grading. Contrast sensitivity testing, frequency doubling technology (FDT) perimetry, and the amplitude of the scotopic threshold response (STR) on electroretinography (ERG) were used to assess inner retinal function. Outer retinal function was assessed with dark adaptation testing. Parametric tests were used to analyze normally distributed variables, and non-parametric tests were used to evaluate non-normally distributed variables. Subgroup analyses were performed for T1DM participants with > and ≥ 15 years duration of diabetes.

Results: 15 T1DM participants (52%) had no retinopathy, 11 (38%) had mild retinopathy, and 3 (10%) had moderate retinopathy. FDT mean deviation (MD) was significantly reduced in T1DM patients (MD ± SD = -1.49 ± 3.09 decibels (dB)) relative to controls (0.11 ± 3.44 dB) ($p=0.026$). Similarly, patients with T1DM exhibited a significant reduction in log contrast sensitivity (1.65 ± 0.08) compared to healthy controls (1.79 ± 0.13) ($p<0.001$). Individuals with T1DM for > 15 years (n=11) had significantly reduced STR amplitudes (3.8 ± 3.9 microvolts) vs. controls (9.2 ± 4.8 microvolts) ($p=0.036$), while STR amplitudes were similar between individuals with T1DM ≤ 15 and controls ($p=0.37$). There were no differences in dark adaptation between controls and the patients with T1DM ($p=0.42$).

Conclusions: Patients with T1DM exhibit inner retinal sensory neuropathy in spite of having no to minimal DR. Further studies are ongoing to understand how such retinal dysfunction might progress over time and to identify new means to prevent vision loss in patients with T1DM.

3

COMPARING THE PREVALENCE OF PERIPAPILLARY RETINOSCHISIS ON OCT IN GLAUCOMA AND GLAUCOMA SUSPECTS AND NORMAL SUBJECTS

Dilraj S. Grewal, Dan Merlau, Amani A. Fawzi, **Lee M. Jampol♦**, Angelo P. Tanna

Purpose: To compare the prevalence of peripapillary retinoschisis between patients with glaucoma or glaucoma suspects and normal subjects.

Methods: In this institutional cross-sectional study, 800 consecutive patients examined in the glaucoma clinic were reviewed. 495 patients (990 eyes) who had undergone spectral-domain OCT (Spectralis HRA-OCT, Heidelberg Engineering) optic nerve head (ONH) raster OCT and did not have optic nerve pits, pseudopits or coloboma were included. 278 eyes, from 144 participants (81 females, 63 males) with a mean age of 37.6 years (range 18-74; SD=15.5) were used as controls and were imaged with the 3DOCT-1000 (Topcon Corp., Tokyo, Japan) using the raster scan protocol. OCT scans for both groups were reviewed by a single observer. The main outcome measure was the presence of peripapillary retinoschisis identified on OCT raster scans. Four cases with uncertain findings on the initial evaluation were adjudicated by a team of two retina specialists and one glaucoma specialist.

Results: None of the 278 eyes of control subjects had peripapillary retinoschisis. 11 eyes (1.1%) of 7 patients (2 females, 5 males, mean age 64.5±9.2 years) had peripapillary retinoschisis, 2/11 eyes had extension of the retinoschisis into the macula. Of these 7 patients, 2 (28.6%) had primary open-angle glaucoma, 3 (42.9%) were glaucoma suspects, 1 (14.3%) had chronic narrow-angle glaucoma and 1 (14.3%) had pigmentary glaucoma. Mean IOP at the time of imaging was 15.4±5.1 mmHg. The mean Humphrey visual-field mean-deviation was -3.48 dB and PSD was 3.45 dB. 7/11 (63.6%) eyes had vitreous traction visualized on OCT and 6/11 eyes (54.5%) had beta-zone peripapillary atrophy.

Conclusions: An increased prevalence of peripapillary retinoschisis was seen in glaucoma and glaucoma-suspects compared to controls. Evidence of adherent vitreous with traction and peripapillary atrophy was found in a majority of the eyes with retinoschisis.

4

FOCAL VENOUS HYPERTENSION AS A PATHOPHYSIOLOGIC MECHANISM FOR TISSUE HYPERTROPHY, PORTWINE STAINS, THE STURGE-WEBER SYNDROME, AND RELATED DISORDERS: PROOF OF CONCEPT WITH NOVEL HYPOTHESIS FOR UNDERLYING ETIOLOGICAL CAUSE

Cameron F. Parsa

Purpose: To provide an in-depth re-examination of assumed causes of tissue hypertrophy, port-wine stains, and the Sturge-Weber, Cobb, Klippel-Trenaunay, and related syndromes to support an alternative unifying pathophysiologic mechanism of venous dysplasia producing focal venous hypertension with attendant tissue responses; to provide proof of concept with new patient data; to propose a novel etiological hypothesis for the venous dysplasia in these syndromes and find supportive evidence.

Methods: Data from twenty patients with port-wine stains and corneal pachymetry readings was collected prospectively by the author in an institutional referral-based practice. The literature was searched using Medline, and articles and textbooks were obtained from the bibliographies of these publications.

Results: Newly obtained dermatologic, corneal pachymetry, fundus ophthalmoscopic, ocular and orbital venous Doppler ultrasonography, and magnetic resonance imaging findings in patients with the Sturge-Weber syndrome or isolated port-wine stains, along with published data, reveal diffusely thickened tissues and neural atrophy in all areas associated with venous congestion.

Conclusions: Contrary to traditional understanding [1,2], signs and symptoms in the Sturge-Weber and related syndromes, including both congenital and acquired port-wine stains are shown to arise from effects of localized primary venous dysplasia [3,4] or acquired venous obstruction rather than neural dysfunction, differentiating these syndromes from actual phacomatoses with no somatic mutations responsible.[4] Effects of focal venous hypertension are transmitted to nearby areas via compensatory collateral venous channels in the above conditions, as in the Parkes Weber syndrome. A novel underlying etiology, prenatal venous thrombo-occlusion, may be responsible for the absence of veins with persistence and enlargement of collateral circulatory pathways with data in the literature backing this offshoot hypothesis.[4] The mechanism for isolated pathologic tissue hypertrophy in these syndromes clarifies physiologic mechanisms for exercise-induced muscle hypertrophy to occur via venous compression and transudation.

5

A CROSS SECTIONAL EXAMINATION OF VISUAL ACUITY BY SPECIFIC TYPE OF ALBINISM

Caitlin Nosanov, Ann M. Holleschau, **C. Gail Summers**

Purpose: Reports of best-corrected visual acuity (BCVA) in albinism are often based on overlapping clinical phenotypes. Also, BCVA in albinism improves with age. The purpose of this study is to report a large cross sectional investigation to determine if BCVA differs by specific type of albinism when age-corrected.

Methods: A retrospective chart review of 538 patients with albinism identified 164 with a definite diagnosis of albinism, defined by mutations on a gene known to cause albinism [for OCA1, OCA2, and Hermansky Pudlak syndrome (HPS]) or a specific phenotype (white hair and no melanin pigment in OCA 1A; pigmentary mosaicism in the obligate carrier for a male with OA1). We recorded age at last visit and binocular optotype BCVA for the 164 patients. Patients were grouped by age (2-5 years, 6-12 years, and ≥ 13 years) and type of albinism.

Results: Mean BCVA at 2-5 years (n=36) was 20/181, 20/108, 20/72, 20/108, and 20/100 for OCA1A, OCA1B, OCA2, OA1, and HPS, respectively. At 6-12 years (n=48), mean BCVA was 20/119, 20/72, 20/61, 10/109, and 20/80 for OCA1A, OCA1B, OCA2, OA1, and HPS, respectively. At ≥13 years (n=80), mean BCVA was 20/123, 20/54, 20/77, 20/70, and 20/130 for OCA1A, OCA1B, OCA2, OA1, and HPS, respectively.

Conclusions: This study is unique because of the large sample size used and inclusion of only those with a known specific type of albinism. Our study shows that BCVA varies by type of albinism, with worse BCVA for OCA1A for all ages, and best BCVA for OCA2 < age 13 and OCA1B ≥ age 13, although there is some overlap in BCVA. These results will assist in counseling those with the specific types of albinism studied here.

6

OUTCOMES OF SURGERY FOR REMOVAL OF VISUALLY SIGNIFICANT HYPERPLASTIC PERSISTENT PUPILLARY MEMBRANES

Gregg T. Lueder, Courtney Kraus

Purpose: Infants with hyperplastic persistent pupillary membranes (PPM) may be at risk for deprivation amblyopia due to obstructions of the visual axis. We describe a surgical technique for removal of PPMs, and the long-term visual and anatomic outcomes following the procedure.

Methods: This is a retrospective review of 6 patients who underwent surgical removal of PPMs. Each PPM was judged to be visually significant based on poor visual acuity, poor retinoscopic reflex, or inability to visualize the fundus. The surgical technique included injection of a viscoelastic agent beneath the pupillary strands to bow them anteriorly, careful peeling of residual adherent strands from the anterior lens capsule, and lysis of the strands at the pupillary margin with intraocular scissors. Pre- and postoperative visual and anatomic results were recorded.

Results: The PPMs were bilateral in 4 patients and unilateral in 2 patients. The patient age at time of surgery ranged from 2.5 months to 2.5 years (mean 14 months). Mean postoperative follow-up was 4.4 years (range 2-8 years). Visual acuity improved in all patients. One patient was treated for anisometropic amblyopia. No operative complications occurred.

Conclusions: Our patients had excellent visual and structural outcomes, with no significant complications. The described surgical technique is a good option for treating patients with visually significant PPMs.

7

INHIBITION OF VASCULAR ENDOTHELIAL GROWTH FACTOR SPLICE VARIANT INHIBITS PATHOLOGIC NEOVASCULARIZATION SAFELY IN MODEL OF RETINOPATHY OF PREMATURITY

Yanchao Jiang, Haibo Wang, W. David Culp♦, Zhihong Yang, Lori Fotheringham, John Flannery, Scott Hammond, Tal Kafri, **M. Elizabeth Hartnett♦**

Purpose: Inhibition of vascular endothelial growth factor (VEGF) has improved outcomes in retinovascular diseases associated with intravitreal neovascularization (IVNV). However, concerns have arisen regarding long-term effects from anti-VEGF agents and especially in developing preterm infants. We developed a gene therapy approach to test strategies to inhibit pathologic VEGF-induced IVNV in a model representative of human retinopathy of prematurity (ROP). We determined effects on safety and efficacy.

Methods: We use a well-accepted rat model that causes extrauterine growth restriction and zone II, stage 3 ROP. We created short hairpin RNAs within a microRNA30 context to silence VEGFA (shRNA-VEGF), splice variant VEGF164 (shRNA-VEGF164) or control luciferase (shRNA-luc). We cloned each shRNA into a plasmid with a CD44 promoter to specifically target Mueller cells. Subretinal injections of 1 uL of each lentivector were performed at postnatal day (p)8. GFP of transduced cells was visualized in vivo with the Micron fundus camera. At p18 and p25, measurements were made for: IVNV and percent avascular retina (AVA) in lectin-stained retinal flat mounts; TUNEL positivity and retinal thickness in retinal cryosections; and protein in fresh retinas.

Results: shRNA-VEGFA and shRNA-VEGF164 reduced retinal VEGF at p18 and p25 compared to shRNA-luc. IVNV, and not AVA, was reduced by each lentivector at p18, but only by shRNA-VEGF164 at p25. shRNA-VEGFA increased TUNEL positive cells at p18 and reduced inner and outer layer thickness at p25, whereas shRNA-VEGF164 maintained outer and inner retinal layer thicknesses.

Conclusions: Targeted reduction in Mueller cell-VEGFA or VEGF164 effectively reduced IVNV at p18 without affecting physiologic AVA, but only shRNA-VEGF164 inhibited IVNV at p25, reduced retinal cell death and maintained retinal layer thickness compared to control. Further studies are needed to determine the effect of Mueller cell knockdown of VEGFA and VEGF164 on retinal function.

8

DEVELOPMENT OF A NEW VALID, RELIABLE, AND INTERNATIONALLY APPLICABLE ASSESSMENT TOOL OF RESIDENTS' COMPETENCE IN OPHTHALMIC SURGERY

Karl Golnik, Hilary Beaver♦, Vinod Gauba, Andrew Lee♦, Eduardo Mayorga, Gabriela Palis, George Saleh

Purpose: To test the validity and reliability of a new tool for assessing residents' competence in ophthalmic surgery. Changing paradigms of ophthalmic education in the United States have influenced worldwide ophthalmic education and necessitated new methods of assessing resident competence. Accordingly, a new tool for assessing residents' competence in ophthalmic surgery (phacoemulsification) that could be applicable internationally was developed. We hypothesize that this instrument is valid and reliable.

Methods: A panel of six international content experts adapted a previously published tool for assessing phacoemulsification.1 The tool (called the International Council of Ophthalmology's Ophthalmic Surgical Competency Assessment Rubric -ICO-OSCAR:phaco), was reviewed by 12 international content experts for their constructive comments which were incorporated to ensure content validity. Ten expert cataract surgery teachers then graded six recorded phacoemulsification surgeries with the ICO-OSCAR:phaco to investigate inter-rater reliability.

Results: The coefficient alpha statistic (a measure of reliability/internal consistency) for the ICO-OSCAR:phaco as a whole was 0.92, and 17 of its 20 dimensions had alpha coefficients greater than 0.70.

Conclusions: The ICO-OSCAR:phaco is a valid and reliable assessment tool that could be applied internationally to satisfy the global need of new instruments to comply with emerging trends in ophthalmic education. A toolbox of similar surgical competency assessment tools is being developed.

9

LONG-TERM FOLLOW-UP OF INTRAOCULAR PRESSURE AFTER VITRECTOMY

Cindy W. Mi, **John T. Thompson♦**

Purpose: To identify whether vitrectomy is associated with an increased risk of elevated intraocular pressure (IOP) and to report the incidence of open angle glaucoma (OAG) following vitrectomy. Vitrectomy has been associated with increased risk of elevated intraocular pressure and glaucoma in some published studies, but not in others.

Methods: Retrospective case series of 234 consecutive patients undergoing vitrectomy in one eye for idiopathic epiretinal membrane or macular hole. Patients with less than 2 years follow-up, with previous vitrectomy in either eye, glaucoma, diabetes, or reason for secondary glaucoma such as intravitreal steroids were excluded. Main outcome measures include mean IOP (measured with Tonopen or applanation if ≥25 mmHg) of the operative and fellow eye at baseline and multiple postoperative time points. Operative and fellow eyes were also assessed for new development of OAG.

Results: Mean baseline IOP was 14.91 mmHg in the operative eyes and 15.21 mmHg in the fellow eyes ($P<0.05$). Mean final IOP was 14.6 mmHg in the operative eyes and 14.74 mmHg in the fellow eyes ($P=0.45$). There was a significant difference in mean IOP between the two eyes at baseline but no significant difference at the final exam (mean of 4.3 years). Linear regression analysis of IOP in operative eyes from baseline to final visit found an increase of 0.000047 mmHg/year compared to -0.00027 mmHg/year in fellow eyes with no significant difference in the slope of the two regression lines ($P= 0.27$). An IOP increase of >4 mmHg from baseline to final exam was found in 15/211 (7.1%) nonphakic vitrectomy eyes compared to 8/67 (11.9%) nonphakic fellow eyes ($P=0.21$). Three (1.3%) study eyes and four (1.4%) fellow eyes were diagnosed with new onset OAG.

Conclusions: Vitrectomy eyes do not appear to be correlated with increased risk of IOP elevation or glaucoma development in comparison to fellow control eyes.

10

OPTICAL COHERENCE TOMOGRAPHY ANGIOGRAPHY OF MACULAR DISEASES

David Huang♦, Yali Jia•, Steven T. Bailey, Andreas K. Lauer♦, **David J. Wilson**♦

Purpose: To determine the features of common macular diseases using the novel imaging modality of optical coherence tomography (OCT) angiography.

Methods: Healthy control subjects and subjects with a variety of macula pathologies underwent OCT angiography in prospective observational studies. Fluorescein angiography (FA) was obtained when clinically indicated. OCT angiography scans were obtained with a high-speed (100,000 A-scans/sec) 1050 nm wavelength swept-source OCT. The scans spanned 3.5X3.5 mm and was acquired in 4.0 sec. Flow was detected using the split-spectrum amplitude-decorrelation angiography algorithm. The volumetric angiography was segmented into four layers: inner retina, outer retina, inner choroid and outer choroid. En face maximum flow projection was used to obtain 2D angiograms of the 4 layers.

Results: In all 5 healthy subjects, retinal capillary networks were visualized in the inner retinal layer, confluent choriocapillaris could be visualized in the inner choroid, and no flow was detected in the outer retina. In all 8 cases of neovascular age-related macular degeneration (AMD), OCT angiography demonstrated choroidal neovascularization (CNV) in the outer retina layer. The depth of CNV relative to the retinal pigment epithelium (RPE) could be determined and the area and flow index could be quantified. The CNV area corresponded well to FA except in 3 cases where fluorescence was blockage by hemorrhage or RPE. In 2 cases of dry AMD, OCT angiography showed loss of choriocapillaris from the inner choroid in areas of geographic atrophy and some areas of large drusen. In 2 cases of nonproliferative diabetic retinopathy (DR), regions of retinal capillary dropout shown by OCT angiography agreed with FA.

Conclusions: OCT angiography was able to detect the presence of abnormal vessels (CNV) by their location in the normally avascular out retinal layer. Capillary dropout could be mapped in DR and choriocapillaris loss could be detected in AMD.

11

MULTIPLE EVANESCENT WHITE DOT SYNDROME: MULTIMODAL IMAGING

Lawrence A. Yannuzzi, Marcela Marsiglia, Eduardo Cunha de Souza, Sarah Mrejen-Uretsky, Suqin Yu, Roberto Gallego-Pinazo♦, Thomas Albini•, Alain Gaudric♦, K. Bailey Freund♦

Purpose: Background: Multiple Evanescent White Dot Syndrome (MEWDS) has been a perplexing, idiopathic chorioretinal disease since its first description by Jampol and co-authors more than 25 years ago. It is believed to be an immune-mediated inflammatory disease seen in genetically predisposed individuals, a vague description at best. The typical presentation is in young, female adults with a mild degree of myopia and a sudden disturbance in the central vision of one eye. The clinical presentation varies, usually with a few to numerous 'white spots' noted in the fundus, enlargement of the blind spot and a granular disturbance of the foveal area. There is a gradual, spontaneous recovery period of a few to several weeks and a few lasting chorioretinal spots may persist, along with foveal abnormality. Occasionally, there may be a recurrence in the same eye.

Methods: Retrospective Case Series with Multimodal Imaging of 18 newly diagnosed patients examined with multimodal imaging.

Results: New observations on the nature and distribution of the spots and the clinical course were observed clinically with Fundus Autofluorescence (FAF), SD-Optic Coherence Tomography (OCT) Imaging and Indocyanine Green (ICG) angiography.

Conclusions: With the advent of new technological imaging systems, particularly OCT, FAF, and ICG angiography, it is easier to make a specific diagnosis, since these diagnostic adjuncts show specific manifestations in MEWDS, but not always. In some patients, there may be difficulty in differentiating MEWDS from other 'white spot syndromes', most specifically idiopathic multifocal choroiditis. This presentation describes new observations in MEWDS that will assist in its diagnosis and also add to its clinical spectrum in quest of further knowledge on its pathogenesis, and hopefully its treatment.

12

THE IMPACT OF DIFFERENT ALGORITHMS FOR IDEAL BODY WEIGHT ON SCREENING FOR HYDROXYCHLOROQUINE RETINOPATHY

David J. Browning♦, Chong Lee

Purpose: To determine how algorithms for ideal body weight (IBW) affect decisions about hydroxychloroquine dosing.

Methods: This was a retrospective, observational cohort study from a private practice of 29 doctors. Four hundred patients were screened for hydroxychloroquine retinopathy and their charts were reviewed for gender, height, weight, and daily dose of hydroxychloroquine. The main outcome measures were ranges of IBW across algorithms; rates of potentially toxic dosing for different algorithms; height thresholds below which 400 mg/d dosing is potentially toxic; and rates for which actual body weight (ABW) was less than IBW.

Results: Women made up 368 (92%) of the patients screened. The range of IBW for a given height varied from 20-31 pounds. For a dose of 400 mg/d, the threshold height below which toxic dosing occurred varied from 62 to 68 inches. The percentages of patients with heights less than or equal to the thresholds of 62, 63, 64, 66, 67, and 68 inches were 13.9%, 26.1%, 36.7%, 66.7%, and 77.2%, respectively. The algorithms placed 16-92% of women in the toxic dosing range. The proportion of patients for whom dosing should have been based on ABW rather than IBW ranged from 8-28% across algorithms.

Conclusions: Although hydroxychloroquine dosing should be based on the lesser of ABW and IBW, there is no consensus about the definition of IBW. The algorithm chosen will affect the frequency of calls to prescribing doctors to reduce dosing. The Broca algorithm is associated with the most frequent need to adjust dosing; the Metropolitan Life Insurance, Medium Build, Upper Bound Table with the least frequent need. No published evidence indicates that a particular algorithm is preferred. The clinician can benefit from understanding the trade-offs made in choosing one algorithm over another.

13

ALGORITHMS FOR APPLICATION OF RESULTS OF MOLECULAR PROGNOSTIC TESTING IN POSTERIOR UVEAL MELANOMA

James J. Augsburger, Zélia Corrêa

Purpose: Chromosomal or molecular prognostic (gene expression profile, GEP) testing of clinically diagnosed posterior uveal melanomas prior to or at the time of initial treatment is fast becoming the standard of care in ocular oncology practices in North America and Europe. Unfortunately, there are no current consensus guidelines about how the results of this prognostic testing should be used.

Methods: The authors prepared algorithms explaining how the authors apply the results of prognostic GEP testing of tumor cells obtained by fine needle aspiration biopsy (FNAB) from patients with posterior uveal melanoma encountered in their clinical practice. In this series, patients were classified as having either a GEP class 1 (low metastatic risk) or class 2 (high metastatic risk) tumor. Separate algorithms were developed for patients with clinically unequivocal posterior uveal melanoma and those with "nevus versus melanoma."

Results: Between September 2007 and August 2013, the authors obtained GEP test results on 376 patients with a melanocytic posterior uveal tumor. These tumors were categorized as class 1 in 266 cases (70.7%) and class 2 in 110 cases (29.3%). In patients with an unequivocal melanoma, the GEP test result is used to advise patients about their metastatic risk and identify those "high risk" persons who would be appropriate candidates for enrollment in adjuvant therapy clinical trials. In patients with a "nevus versus melanoma," the GEP test result is used primarily to determine the urgency of intraocular tumor treatment and secondarily to advise patients about their metastatic risk and potential eligibility for adjuvant therapy trials.

Conclusions: Algorithms for application of the results of prognostic GEP testing of clinically diagnosed posterior uveal melanoma will be presented for information and discussion. The authors do not currently recommend differential surveillance testing for patients in the two GEP groups except in the context of prospective clinical trials.

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LEAN TRANSFORMATION OF AN INTRAVITREAL INJECTION CLINIC: INCREASING ACCESS AND ENHANCING PATIENT EXPERIENCE

Dennis P. Han♦, Ravi S.J. Singh♦, Kay Mareno, Kay Kastner, Joseph Beringer, Stephen Alper, Aneesh Suneja

Purpose: Increased adoption of anti-VEGF therapy for retinal disease requires that we become more efficient in providing intravitreal injections. We determined if application of Lean manufacturing principles (1,2) could improve access and reduce patient visit length in a retina outpatient clinic structured to provide intravitreal injection therapy.

Methods: Resources consisted of a core team of a receptionist, one physician, two technician-scribes and a photographer (1.0 FTE of each of above). We used three multifunctional exam rooms and an OCT room. We mapped the flow of 30 patients. Current state value stream mapping quantified patient wait time, technician cycle time, walking distance and imaging cycle time. A future state value stream map was then formulated that predicted a 40-50% reduction in visit length. Primary outcome measure was total visit length reduction; secondary outcome measure was capacity for injection visits per 3.5 hour clinic interval.

Results: Process interventions included reduced (1) handoffs, (2) walking of patients and staff, (3) dependency of process steps, (4) technician and physician cycle time, and (5) more rapid access to OCT instrumentation. A multifunctional device, (the RAVI-Guide), was designed to increase the speed and comfort of the injection procedure. After intervention, we observed mean total visit length reduction from 88 minutes to 44 minutes (50%) for patients undergoing OCT and from 45 minutes to 29 minutes (35%) for patients without OCT. Capacity increased from 16 to 21 injections per 3.5 hour clinic session with preservation of physician-patient face-to-face counseling time. We received overall Avatar satisfaction top-box ratings of over 90%, exceeding our institutional average.

Conclusions: Application of Lean in an intravitreal injection clinic improved patient access, reduced visit length, preserved value-added time, and created a high level of patient satisfaction, without increased resource expenditure. The forms of waste we identified are universal, indicating likely benefits of Lean in a variety of ophthalmic practice settings.

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MOLECULAR DIAGNOSTIC PRECISION IN THE RETINAL DYSTROPHIES

Elias I. Traboulsi♦, Meghan J. Marino

Purpose: To investigate the ability of a team composed of an ophthalmic geneticist and a genetic counselor, running a specialized retinal dystrophy clinic to reach a precise diagnosis in patients with retinal dystrophies.

Methods: This study was approved by the Cleveland Clinic IRB. We conducted a retrospective analysis of clinical records and results of molecular genetic testing conducted in CLIA-certified laboratories in a cohort of 120 patients with retinal dystrophies evaluated between January 2011 and October 2013 in a specialized retinal dystrophy clinic.

Results: A molecular diagnosis was made in 76/120 patients. There were 32 patients with Stargardt disease, 29 with retinitis pigmentosa, 24 with Leber congenital amaurosis (LCA) and the rest had a variety of other retinal dystrophies. Highest negative rate was in those with retinitis pigmentosa (14/29). 9/32 patients with a clinical diagnosis of Stargardt disease were not found to have mutations in ABCA4, and 1 had a mutation in PRPH2. One patient with blue cone monochromacy and 2 with Alström disease were misdiagnosed as having LCA.

Conclusions: A precise molecular diagnosis of retinal dystrophies can be reached in two-thirds of patients with retinal dystrophies using a combination of clinical examinations, imaging studies and molecular genetic testing. Genetic testing methodologies continue to evolve and will allow a higher degree of diagnostic precision. This will facilitate the provision of better clinical and genetic counseling and the identification of candidates for treatment trials.

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PATHOLOGIC AND IMMUNOHISTOCHEMICAL FEATURES OF PELLUCID MARGINAL DEGENERATION

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Purpose: Pellucid Marginal Degeneration (PMD) has pathologic features similar to keratoconus (KCN). In KCN, the corneal immunophenotype suggests that enzymatic degradation may play a role in corneal thinning; however the immunophenotype of the PMD cornea remains unknown. We hypothesized that the immunophenotype of the cornea in PMD would be similar to that previously reported in the KCN cornea.

Methods: The clinical data of PMD patients (n=8) were retrieved. Archived corneal tissues of patients with PMD, KCN (n=8) and normal control corneas (n=6) were immunolabeled with antibodies against Sp1, α1-proteinase inhibitor (α1-PI), and cathepsin B in duplicate. The labeling pattern was examined and the intensity graded. Immunohistochemical (IHC) staining intensity was compared using nonparametric tests.

Results: By light microscopy, stromal thinning and breaks in Bowman's layer were noted in all PMD and KCN specimens. Compared to normal controls, increased Sp1 staining (1.42 and 1.39 fold; P=0.112) was observed in basal and wing cell nuclei of corneal epithelium of KCN and PMD respectively. α1-PI labeling was decreased in the corneal epithelial cytoplasm in KCN (1.36 fold; P=0.006) and PMD corneas (1.60 fold; P=0.002) when compared with the normal control. Cathepsin B labeling slight but insignificant increase in labeling in the epithelial cytoplasm in KCN and PMD (1.06 and 1.20 fold respectively; P=0.29) when compared to normal controls. IHC staining for all antibodies was similar in corneas with PMD and KCN (Sp1, P=0.112; α1-PI, P=0.21; and cathepsin B, P=0.209).

Conclusions: The immunophenotype in PMD was comparable to what was previously reported in KCN. This suggests that enzyme related tissue degradation may play a role in the pathogenesis of PMD as well. PMD and KCN may share a common pathway as to the underlying mechanisms involved in stromal thinning.

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LIQUID NITROGEN CRYOTHERAPY TREATMENT OF HERPES SIMPLEX EPITHELIAL KERATITIS

Shashi Alloju♦, Michelle Patel, **Rick Fraunfelder**

Purpose: To evaluate the efficacy of liquid nitrogen cryotherapy as an primary treatment for herpes simplex epithelial keratitis.

Methods: A retrospective case series was performed in a university setting for all patients with herpes simplex epithelial keratitis who underwent liquid nitrogen cryotherapy from 2011-2013. This study reviewed 6 eyes of 6 patients (age 30-79). Patients underwent double freeze-thaw cryotherapy to the epithelial lesion with a Brymill spray tip. Patients were also placed on prophylactic dose acyclovir 400 mg BID dosing as tolerated. Outcome measure included clinical evidence of resolution of epithelial keratitis in the cornea with re-epithelialization.

Results: All cases of epithelial keratitis showed partial and complete resolution at day 1 and week 1 respectively. There were no observed cases of endothelial decompensation or scarring. There were no other adverse outcomes.

Conclusions: Liquid nitrogen cryotherapy is a safe and effective treatment for herpes epithelial keratitis comparable to other published studies. In patients that may not tolerate topical or oral anti-viral therapy, this procedure may be a viable option.

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OUTCOMES OF GLAUCOMA DRAINAGE IMPLANT SURGERY IN UVEITIC GLAUCOMA

Eun Sara Huh, Ahmad Aref♦, Thasarat Vajaranant, **Jacob Wilensky**

Purpose: This study reports the long term efficacy of glaucoma drainage implant (GDI) in the management of uveitic glaucoma.

Methods: A retrospective review of patients with uveitic glaucoma who underwent GDI surgery from January 1, 2002 to December 31, 2012 was performed. Outcome parameters collected include visual acuity (VA), intraocular pressure (IOP), type of glaucoma implant utilized, number of glaucoma medications, and postoperative complications.

Results: Sixty-six eyes of 56 patients underwent GDI for the management of uveitic glaucoma refractory to medical management. The average age was 35 years with 19 males and 37 females. The average VA preoperatively was logMAR 0.76 with an IOP on average measuring 32mmHg (±11.1 mmHG) on an average of 4 (±0.9) IOP lowering medications. Forty-three eyes underwent Ahmed glaucoma implant (AGI) and 23 eyes underwent Baerveldt glaucoma implant (BGI) placement. The average follow up was 32 months (range 2 - 129 months). Postoperatively, the average VA was logMAR 0.973 ($p=0.19$) with an average IOP of 11mmHG (±4.6mmHG, $p<0.0001$) on an average of 1 (±1.3, $p<0.0001$) IOP lowering medication. Pre-operative VA of the AGI and BGI groups were logMAR 0.81 and logMAR 0.68, respectively ($p=0.53$); pre-operative IOP were 32 mmHG and 31mmHG ($p=0.64$) managed by an average of 4 medications for both groups ($p=0.47$). Post-operative VA of the AGI and BGI groups were logMAR 0.93 and logMAR 1.05, respectively ($p=0.65$); post-operative IOP were 12 mmHG and 11 mmHG ($p=0.81$) managed by an average of 2 and 1 medications ($p=0.67$). Four eyes needed additional glaucoma procedures to manage intraocular pressure and all underwent transcleral cyclophotocoagulation. Complications included: three eyes with corneal decompensation, two cases of endophthalmitis, one eye with suprachoroidal hemorrhage and two eyes with hypotony.

Conclusions: GDIs effectively control intraocular pressure in refractory uveitic glaucoma in the long term with a relatively low risk of postoperative complications.

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THE SPECTRUM OF OPTIC DISC ISCHEMIA IN PATIENTS YOUNGER THAN 50 YEARS

Anthony C. Arnold♦, Roberta M. S. Costa, Oana M. Dumitrascu

Purpose: To identify the spectrum of clinical and fluorescein angiographic features of optic disc ischemia in patients younger than 50 years

Methods: This retrospective comparative case series from a university consultative neuro-ophthalmology practice consisted of two phases. The first compared 108 cases of nonarteritic anterior ischemic optic neuropathy in patients younger than 50 years (NAIONy) to a cohort of 108 cases in patients older than 50 years (NAIONo). Predisposing risk factors, fluorescein angiographic features, and clinical course were compared. In the second phase, 12 cases of diabetic papillopathy under age 50 were assessed by fluorescein angiographic criteria for evidence of optic disc ischemia and compared to patients with NAIONy.

Results: NAIONy comprised 108 (12.7%) of 848 NAION cases reviewed. Chronic renal failure with dialysis (CRF) and migraine were more common in NAIONy. Fellow eye involvement rate was significantly higher for NAIONy (46/108, 42.6%) patients, than for NAIONo (32/108, 29.6%) [Chi square test, P = .047]. Fluorescein angiographic features of ischemia were documented in 44/54 (81.5%) of eyes studied. In one case, these features were documented in pre-NAION edema. Diabetic papillopathy demonstrated delayed filling consistent with ischemia in 7/10 (70.0%), without significant visual field loss.

Conclusions: Ischemic optic neuropathy in patients younger than 50 years is not rare. Fellow eye involvement is more frequent in younger patients. Fluorescein angiography confirmation of impaired perfusion in multiple syndromes of optic neuropathy corroborates a spectrum of optic disc ischemia ranging from perfusion delay without visual loss to severely impaired perfusion and visual loss and incorporates optic neuropathies previously considered nonischemic.

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ARTISAN APHAKIA INTRAOCULAR LENS FOR CHILDREN: MULTICENTER, PROSPECTIVE STUDY

M. Edward Wilson, Rupal H. Trivedi

Purpose: To study the early outcomes of the ARTISAN aphakia intraocular lens (IOL) in children

Methods: Prospective, open-label, multicenter, ongoing FDA Investigational Device Exemption (IDE) study in children between 2 to 21 years of age.

Results: 77 eyes of 48 children have been implanted at the time of this submission. In 44 subjects, the IOL was implanted into aphakic eyes as a secondary procedure. In 4 subjects the IOL was implanted primarily, at the time of lensectomy. Age at implantation was 10.0±4.5 (3-18) years. Intraoperative complications included, anterior segment bleeding (1), difficult enclavation (2) and iris damage (1). Follow-up (subjects): 1 month: 41; 3 months: 36; 6 months: 33; 1 year: 13. Postoperative complications requiring reoperation have occurred in 3 subjects (4 eyes). 1 eye had a revision of the peripheral iridectomy for high intraocular pressure (2 weeks post-op). 1 eye had a re-enclavation after post-operative trauma (11 weeks post-op). 1 eye had a wound leak repair (2 days post-op). 1 eye had pupillary block glaucoma requiring 4 re-operations before stabilization (2 weeks post-op). Best Corrected Distant Visual Acuity (BCDVA) improved after implantation (paired T test) at 1 month post-op: logmar 0.37 pre-op to 0.30 post-op (P=0.03,n=47). Two eyes reported a reduction of >2 lines of BCDVA at 1 month postoperative visit, but vision improved at latest follow-up without further intervention. At one year follow-up (13 subjects) BCDVA improved from a mean of 20/88 before implantation to 20/40 after implantation (P=0.05)

Conclusions: Implantation of the ARTISAN aphakic IOL in children under an FDA IDE reveals acceptable safety and visual outcomes. Adverse events have occurred but without permanent visual consequences. Monitoring is ongoing.

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THE HOME STUDY: ANALYSES OF FINDINGS FROM THE HOME STUDY – HOME MONITORING OF INTERMEDIATE AMD PATIENTS

Michael J. Elman♦

Purpose: To determine whether home monitoring with the ForeseeHome device (Notal Vision, Israel), and tele-monitoring, results in earlier detection of age-related macular degeneration associated choroidal neovascularization (CNV), when compared with standard care.

Methods: Of the 1,970 screened participants at high risk of developing CNV, 1520 were enrolled in the HOME study at 44 AREDS2 clinical centers and randomized to two arms. Instructions were provided to participants in both arms for self-monitoring vision function at home followed by report of new symptoms to the clinic with subsequent examinations. Participants in the device arm were also asked to perform daily device testing. When changes in device test results were identified by the monitoring center, participants were contacted for in clinic examination. The primary outcome was the change in best-corrected visual acuity (VA) between baseline and the visit in which the study ophthalmologist detected CNV.

Results: The DSMC recommended early study termination. The DSMC report included data up to April 2013 and is the basis for this presentation. Mean follow-up was 1.4 years. 82 participants progressed to CNV, 51 in the device arm and 31 in the standard care arm. The baseline VA was similar in the two treatment groups with a mean of 20/25. Participants in the device arm had a smaller decline in VA from baseline to CNV detection (median, -4 letters; interquartile range [IQR], -11.0 to -1.0 letters) compared with standard care (median, -9 letters; IQR, -14.0 to -4.0 letters; P = 0.021).

Conclusions: Persons at high risk for AMD associated CNV benefit from this home monitoring strategy for earlier detection of CNV. The smaller decline in visual acuity at the time of CNV detection in the home monitoring arm is more likely to result in optimal visual acuity outcomes following therapy.

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MUTATION IDENTIFICATION IN THE FAMILY WITH AUTOSOMAL RECESSIVE ENCEPHALOCELE AND RETINAL DETACHMENTS ORIGINALLY DESCRIBED BY KNOBLOCH

Behrad Y.Milani, **C. Gail Summers, Irene H. Maumenee**

Purpose: The ocular findings in the Knobloch syndrome, in addition to retinal detachments, are characterized by incomplete macular development and congenital high myopia. Mutations in COL18 had been previously identified in this disease. Menzel et al described evidence for exclusion of the chr: 21 locus in one family with clinically indistinguishable Knobloch syndrome. No causative mutations had previously been identified in the original family with autosomal recessive encephalocele and retinal detachments described by Knobloch and Layer, 1971 and Cook and Knobloch, 1982. The search for a second causative gene for Knobloch syndrome beside COL18 had been ongoing in this as well as additional Knobloch families. The purpose of this study is to detect causative gene mutations in the large family originally described.

Methods: Peripheral blood samples for DNA extraction as well as clinical information of all living affected and potentially informative family members were obtained after informed consent was secured and the study had been approved by the Internal Review Board. DNA was extracted from one sample of the large original Knobloch family and submitted for exon sequencing of the following genes: COL18A1, ADAMTS18 and COL15.

Results: DNA analysis showed compound heterozygosity of mutations in exon 41 of COL18A1 in the original family described by Knobloch in which meningocele, ateliotic macula and retinal detachment are described as an autosomal recessive disease.

Conclusions: The Knobloch syndrome is classified as heritable disorders of connective tissue and combines ocular, CNS and skeletal findings. It is a complex autosomal recessive syndrome, involving intrauterine developmental anomalies of the brain (meningoencephaloceles), the macula (ateliotic macula) and the face. Ocular complications consist of irregular astigmatism, juvenile cataracts, dislocated lenses and retinal detachments. To date, no definitive mutations beyond those in COL18 have been identified in patients with the Knobloch phenotype.

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SPHENOID SINUS EXPANSION: A RADIOGRAPHIC SIGN OF INTRACRANIAL HYPOTENSION AND THE SAGGING BRAIN, SUNKEN EYES SYNDROME

Timothy J. McCulley

Purpose: "Sunken eyes, sagging brain syndrome" refers to a newly recognized syndrome characterized by intracranial hypotension and consequent enophthalmos from orbital volume expansion. To test the hypothesis that bone remodeling is not limited to the orbits, in this study volumetric analysis of the sphenoid sinus is performed.

Methods: In this university based retrospective case controlled study the dimensions of the sphenoid sinus were measured in four patients (2 males, 2 females, mean age 26.3 years, range 16 to 38 years) out of five individuals identified with sagging brain, sunken eyes syndrome. Three measurements were taken: the distance between the orbital apices, the posterior extension of the sphenoid sinus posterior to the orbital apices and the maximal horizontal width. The mean of each was determined and compared to that of the control group (5 males, 5 females, mean age 35.6 years old, range 23 to 45 years).

Results: Posterior extension and width of the sphenoid sinus were markedly larger in the enophthalmic than the control group: posterior extension (26.3+/-4.1mm vs. 13.4+/-6.3mm, p=0.0015), width (39.2+/-8.7mm vs. 25.1+/-6.9mm, p=0.0035). Mean distance between the orbital apices was slightly greater (36.3+/-1.7mm vs. 34.1+/-2.1mm, p=0.047).

Conclusions: Skull remodeling occurring in association with intracranial hypotension after VPS is not limited to the orbits. In this study we have demonstrated expansion of the sphenoid sinus. This finding adds to our knowledge and understanding of the scope of the sagging brain, sunken eyes syndrome and elucidates a clinically useful radiographic sign.

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A VALUE-BASED MEDICINE COMPARATIVE EFFECTIVENESS ANALYSIS OF TREATMENT OF RETINAL VEIN OCCLUSION THERAPIES

Gary C. Brown♦, Melissa M. Brown♦, **George Beauchamp**

Purpose: To ascertain which therapies for central and branch retinal vein occlusion are most beneficial for patients, taking into account: 1) intravitreal VEGF-inhibitors, 2) laser photocoagulation, 3) surgical therapies and 4) medical therapies. Since treatment cohorts have different baseline visions, different vision outcomes and different associated adverse events and their incidences, a methodology should be utilized which integrates all into a common therapeutic metric.

Methods: Patient preference-based, quality-of-life instruments (utilities) have distinct advantages over function-based quality-of-life instruments. Though the latter are important within specialties, they generally are unable to compare interventions across medical specialties or be used in economic analyses. Validated, reliable, ophthalmic, time tradeoff utilities from over 1,000 patients were utilized to quantify the patient value (improvement in quality-of-life and/or length-of-life) gain conferred by Level 1 interventions to treat central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO). Adverse events associated with the therapies were taken from a validated, 50,000+ time tradeoff utility database. Outcome metrics included patient value (predominantly quality-of-life) gain and QALY (quality-adjusted life-year) gain.

Results: Therapeutic value gains are shown in the Table.

Central Retinal Vein Occlusion		Branch retinal vein occlusion	
*Aflibercept, 2mg, intravitreal	15.1%	*Ranibizumab, 0.5mg, intravitreal	18.0%
*Bevacizumab, 1.25mg, intravitreal	13.5%	*Aflibercept, 2mg, intravitreal	16.3%
*Ranibizumab, 0.5mg, intravitreal	11.6%	*Bevacizumab, 1.25mg, intravitreal	10.2%
*Triamcinolone, 1mg, intravitreal	4.8%	*Grid laser	6.1%
*Dexamethasone, 0.7mg, intravitreal	4.1%	*Dexamethasone, 0.7mg, intravitreal	4.4%
*Grid laser	-1.5%	*Triamcinolone, 1mg, intravitreal	-0.2%
Radial optic neurotomy	13.2%	Arteriovenous sheathotomy	9.9%
Retinal vein cannulation, Tissue plasminogen activator (TPA) injection	10.9%		
Vein cannulation, endovascular	9.7%		
Chorioretinal laser anastomosis	5.0%		
Intravenous tissue plasminogen activator (TPA)	3.0%		

* = level 1 interventional evidence from randomized clinical trials with an $\alpha < 0.05$ and $\beta < 0.20$

Conclusions: Integrating all benefits and adverse events, interventions for CRVO and BRVO evaluated in Level 1 randomized clinical trials demonstrate that VEGF-inhibitors provide the greatest patient value.

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AMERICAN OPHTHALMOLOGICAL SOCIETY EARLY PAPERS OF VISUAL FIELD ANALYSIS

Steven A. Newman

Purpose: In 1824 Wollaston, probably suffering migraine episodes, recognized the implications of fibers crossing at the chiasm. In the 1850s, von Graefe introduced preliminary attempts at visual field analysis, and in 1862, Förster introduced a practical means of assessing visual fields with an arc perimeter.

Methods: A retrospective analysis of twelve early papers from the first 25 years of the American Ophthalmological Society concerning visual field techniques and localization, published between 1870 and 1889.

Results: Three papers described modifications of European developed visual field machines including progression from tangent screen testing to arcuate, to hemispheric bowls. Other papers recognized wedge defects, ring scotomas, homonymous hemianopsia, and central scotomas. One paper probably described junctional syndromes before Wilbrand and Traquair.

Conclusions: Increasing understanding of the visual pathways permitted recognition of homonymous hemianopsia following gunshot wounds or tumors. Early description of congenital absence of the chiasm produced bitemporal visual field defects as predicted by MacKenzie. Ring scotomas were appreciated secondary to retinitis pigmentosa, but also to choroiditis. An early victim of gunshot wound at Antietam provided even earlier demonstration of cortical localization preceding Inoue in the Russo-Japanese War, Gordon Holmes during World War I, and follow up studies during World War II which helped localize the visual pathways by studying gunshot wounds.

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DIAGNOSTIC CAPABALITY OF A SPECTRAL DOMAIN OPTICAL COHERENCE TOMOGRAPHY THREE-DIMENSIONAL NEURORETINAL RIM PARAMETER FOR GLAUCOMA

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Purpose: To evaluate the diagnostic capability of a spectral domain optical coherence tomography (OCT) three-dimensional (3D) neuroretinal rim parameter for glaucoma.

Methods: A cross-sectional study was conducted in a university hospital population of patients who had 3D spectral domain OCT imaging between 2009 and 2013. Optic nerve 3D scans were acquired using the Spectralis (Heidelberg Engineering, Heidelberg, Germany). Of patients who met inclusion criteria, only eye of each patient was selected. From these images, the optic nerve boundaries were automatically detected, and the neuroretinal rim minimum distance band was computed as the shortest distance between the optic nerve boundary and the optic nerve surface. For each eye, the thickness of the 3D neuroretinal rim minimum distance band was calculated globally, for four quadrants (superior, nasal, inferior, temporal), and for four sectors (superior-temporal, superior-nasal, inferior-temporal, inferior-nasal). To evaluate the diagnostic performance of this 3D neuroretinal rim parameter, calculations included areas under the receiver operating characteristic curve (AROC), sensitivity, specificity, positive predictive value, negative predictive value, positive likelihood ratio, and negative likelihood ratio.

Results: One eye each of 44 normal subjects and 47 open angle glaucoma subjects [with average visual field mean deviation (MD) of -9.97 +/- 7.76 dB] was included in the study. Global or overall 3D neuroretinal rim band thickness had the highest AROC value (0.976 for all subjects). For all subjects, the highest sensitivity values were seen in the global area, the inferior quadrant, and the inferior-temporal sector [95.7%, 95% confidence interval (CI), 85.5% to 99.5%], with specificity values of 88.6% (CI, 75.4% to 96.2%), 84.1% (CI, 69.9% to 93.4%), and 81.8% (CI, 67.3% to 91.8%), respectively.

Conclusions: A 3D spectral domain OCT neuroretinal rim parameter demonstrated statistically significant differences between glaucomatous and normal eyes. This 3D parameter may have future potential for glaucoma diagnosis, monitoring and clinical trial endpoints.

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EFFECT OF AGE AT INTRAOCULAR LENS (IOL) IMPLANTATION ON REFRACTIVE GROWTH IN VERY YOUNG CHILDREN

Scott Barb, Mary Ellen Hoehn, **Natalie Kerr**, T. Amerson Pegram, Kyle Cox, Joshua Little

Purpose: Understanding refractive growth (RRG3) of the pseudophakic eye during childhood is important to determining the best intraocular lens (IOL) power at the time of surgery. Few studies have analyzed the effect of IOL implantation on RRG3 in infants.

Methods: A retrospective chart review was performed for children undergoing primary IOL implantation at the time of cataract surgery by two ophthalmologists (MEH and NCK) at Hamilton Eye Institute and St. Jude Children's Research Hospital between 1999 and 2010. One eye of 31 patients with implantation before 6 years of age was included. Refractions were measured within 3 months and annually thereafter for 3+ years. RRG3 was calculated using first and last post-operative refractions, A-constant, and IOL power. RRG3 values for those pseudophakes implanted by 6 months of age were compared with values for the remaining patients (divided into two age groups) and with published values for similarly aged aphakes.

Results: There was no statistically significant difference in RRG3 values among the pseudophakic groups (P = 0.10-0.57). The mean RRG3 value (diopters/year) was -17.90+/-4.72 in the 0-6 month age group and -14.50+/-8.66 and -12.24+/-8.50 in the two later age groups. Although not statistically significant, there was a slight increase in RRG3 seen in younger pseudophakes (age 0-6 months) as compared to similarly aged aphakes.

Conclusions: Our findings suggest age at implantation does not significantly effect RRG3 in pseudophakic children, including patients 0-6 months of age at implantation, although RRG3 in pseudophakic infants may be higher than in similarly aged aphakes. Though RRG3 differences among our groups were not statistically significant, standard deviations were large and values trended higher in the younger children. These findings benefit infants with congenital cataracts undergoing primary IOL implantation by allowing the surgeon to use certain assumptions about refractive growth rate in the youngest children.

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THE GLAUCOMA TREATMENT COMPLIANCE ASSESSMENT TOOL (GTCAT) HAS AN ORGANIZATIONAL STRUCTURE CONSISTENT WITH THE HEALTH BELIEF MODEL

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Purpose: To determine the psychometric properties of a new version of the Glaucoma Treatment Compliance Assessment Tool (GTCAT), a survey evaluating health behavior and glaucoma adherence using constructs of the Health Belief Model.

Methods: We modified the GTCAT into a 5-point Likert scale with standard anchoring definitions and added questions related to mood and satisfaction with your eye doctor. We administered the 47-item GTCAT to participants using a single bottle of an ocular hypotensive agent, and objectively measured adherence with Medication Event Monitoring System (MEMS) devices over 60 days. Adherence was the number of bottle openings divided by the number of openings expected. We used Principal Components Analysis (PCA) to determine construct validity, Cronbach's alpha (α) for internal consistency reliability, frequency analysis for floor and ceiling effects, and Spearman's Rho for re-test reliability. We determined predictive validity using univariate and multivariate regression using adherence as the dependent variable.

Results: We included 201 open angle glaucoma or glaucoma suspect patients from three tertiary glaucoma centers. The mean (+/-SD, range) adherence was 79.9% (+/- 18.5%, 20.3% to 100.0%). PCA explained 38.6% of the variance and loaded 24 questions into 6 components. Five of the extracted components were consistent with the Health Belief Model while the sixth component included knowledge questions. All six components had reasonable reliability (α = .601-.797). No items had floor or ceiling effects, and all items had good test re-test reliability. Regression analysis showed 10 questions to be associated with adherence in univariate analysis, and 6 questions in a multivariate analysis ($P < .05$). White race, older age, and married had higher adherence in a multivariate model. A final predictive multivariate model had an adjusted R-square of .27.

Conclusions: The newest version of the GTCAT showed good psychometric properties. Further evaluations will compare a shorter questionnaire in a clinical setting.

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THE NEW YORK EYE AND EAR INFIRMARY: INCUBATOR OF AMERICAN OPHTHALMOLOGY AND BIRTHPLACE OF THE AMERICAN OPHTHALMOLOGIC SOCIETY

Richard B. Rosen♦, Joseph B. Walsh, **James Ravin**

Purpose: To describe the origins of the New York Eye and Ear Infirmary and discuss its pivotal role in the establishment of organized ophthalmology in the new world and the foundation of the American Ophthalmologic Society

Methods: An extensive review of historical documents, including institutional and community sources as well as ophthalmic literature were used to reconstruct this critical kernel of our professional heritage.

Results: The Infirmary as the first specialty hospital in the new nation traces its origins to the London Dispensary for Curing Diseases of the Eye and Ear founded to battle the scurge of British veterans returning from the Egyptian campaign against Napolean. Edward Delafield and John Kearny Rogers, disciples of the now venerable Moorfields Eye Hospital, initiated this charitable institution to bring relief to the segment of New York citizens at risk for blindness and not served by the local health facilities. Its success encouraged the subsequent creation of similar centers in other cities and fostered the emergence of the specialty hospitals in general. With the advancements of our field such as the inventions of ophthalmoscopy and anesthesia over the next 44 years, a growing community of physicians committed to maintaining the high standards of the specialty came together to form the American Ophthalmologic Society in 1864 lead by many of the Infirmary's pillars, including Delafield, Freeman Bumstead, Henry Noyes, and George Wilkes. The first two meetings of the society took place within the institution and the emblem of the society was adopted from the Infirmary's original seal commissioned in 1824.

Conclusions: As the birthplace of both organized American ophthalmology and the American Ophthalmologic Society, the Infirmary remains as a treasured reminder of our roots and continues as a vibrant resource of the world community dedicated patient care, physician training and advancement of our specialty.

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THE BLUE ARC ENTOPTIC PHENOMENON IN GLAUCOMA

Louis R. Pasquale♦, Steven Brusie♦

Purpose: To determine whether the blue arc entoptic phenomenon, a positive visual response originating from the retina with a shape that conforms to the topology of the nerve fiber layer, is depressed in glaucoma.

Methods: We recruited a cross-sectional, nonconsecutive sample of 202 patients from a single institution in a prospective manner. Subjects underwent full ophthalmic exam including standard automated perimetry (Humphrey Visual Field 24-2) or frequency doubling technology (Screening C 20-5) perimetry. Eligible patients viewed computer-generated stimuli under conditions chosen to optimize perception of the blue arcs. Unmasked testers instructed patients to report whether they were able to perceive blue arcs but did not reveal what response was expected. We created multivariable logistic regression models to ascertain the demographic and clinical parameters associated with perceiving the blue arcs.

Results: In multivariable analyses, each 0.1 unit increase in cup-disc ratio was associated with 36% reduced likelihood of perceiving the blue arcs (Odds Ratio (OR) = 0.66 [95% confidence interval (CI): 0.53-0.83], P<0.001). A smaller mean defect was associated with an increased likelihood of perceiving the blue arcs (OR=1.79 [95% CI: 1.40-2.28]); P<0.001), while larger pattern standard deviation (OR=0.72 [95% CI: 0.57-0.91]; P=0.005) and abnormal glaucoma hemifield test (OR=0.25 [0.10-0.65]; P=0.006) was associated with a reduced likelihood of perceiving them. Older age and media opacity were also associated with an inability to perceive the blue arcs.

Conclusions: In this study, the inability to perceive the blue arcs correlated with structural and functional features associated with glaucoma, although older age and media opacity were also predictors of this entoptic response.

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DIRECT MEASUREMENT OF INTRARETINAL NITRIC OXIDE IN EARLY DIABETIC RETINOPATHY

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Purpose: Nitric oxide (NO) levels are altered in diabetic retinopathy. We developed a NO sensor that can measure the intraretinal NO concentration. The purpose of this study is to measure NO levels in early stages of diabetic retinopathy.

Methods: Long-Evans rats received an intraperitoneal injection of streptozotocin and NO measurements were made at three weeks. Diabetic rats were divided into moderate blood glucose (MBG) or high blood glucose (HBG) groups. Intraretinal NO measurements were made using a double-barreled microelectrode. Diabetic NO measurements were compared to untreated controls and to healthy rats that received an intravitreal injection of nitric oxide synthase (NOS) inhibitor L-NG-nitroarginine methyl ester (L-NAME). HBG diabetics also received intravenous injections of L-arginine (50-500 mg/kg BW) after control profiles were recorded.

Results: NO concentration at the choroid/retina boundary was 2.32 ± 0.27 μM for controls, while MBG diabetics had significantly higher NO concentration at 3.73 ± 0.39 μM ($p=0.007$). HBG diabetics and L-NAME profiles had significantly lower NO concentration at 0.924 ± 0.12 μM ($p=0.005$) and 0.83 ± 0.15 μM for L-NAME ($p=0.014$), respectively. NO concentration at the retina/vitreous boundary was 1.18 ± 0.11 μM. MBG diabetics were not significantly different from controls. while HBG and L-NAME profiles were significantly lower. L-arginine injection did not alter NO levels of HBG diabetics ($p=0.32$).

Conclusions: This study is the first to measure intraretinal NO levels in control and diabetic rats. High levels of NO in MBG diabetics and low levels in HBG diabetics suggest a non-linear relationship of NO and blood glucose level. The similarity of HBG diabetic and L-NAME profiles and the unresponsiveness of NO levels to L-arginine suggest inhibition or decreased expression of NOS in HBG diabetics. The differences between MBG and HBG diabetics may explain the conflicting reports in literature regarding the NO levels in early diabetic retinopathy.

Notes