

**RESIDENT / FELLOW  
RESEARCH DAY**

**Department of Ophthalmology  
and Visual Sciences**

~

**Roy J. and Lucille A. Carver  
College of Medicine**

**University of Iowa Hospitals & Clinics**

**Iowa City, Iowa**



**Braley Auditorium, 01136 Lower Level, Pomerantz Family Pavilion  
Friday, May 15, 2015**

# RESIDENT/FELLOW RESEARCH DAY - 2015

## FELLOWS

### CORNEA

Matthew E. Raecker, M.D.

### GLAUCOMA

Megan Chambers, M.D.

### NEURO-OPHTHALMOLOGY

Enrique J. Rivera, M.D.

### OCULOPLASTIC SURGERY

Meredith A. Baker, M.D.

### OPHTHALMIC GENETICS

Matthew C. Weed, M.D.

### PEDIATRIC OPHTHALMOLOGY

Ashley Ko, M.D.

### VITREORETINAL DISEASE

David R. Almeida, M.D., MBA, Ph.D., FRCSC

Eric K. Chin, M.D.

Yasser Elshatory, M.D., Ph.D.

Rachel M. Huckfeldt, M.D., Ph.D. (Medical Retina)

Christopher N. Roybal, M.D., Ph.D.

# RESIDENT/FELLOW RESEARCH DAY - 2015

## RESIDENTS

### THIRD-YEAR RESIDENTS

Jonathan L. Hager, M.D.  
C. Blake Perry, M.D.  
Bradley A. Sacher, M.D.  
Jesse M. Vislisel, M.D.  
Jeffrey D. Welder, M.D.

### SECOND-YEAR RESIDENTS

P. Christi Carter, M.D.  
Johanna M. (Dijkstal) Bebee, M.D.  
Chris A. Kirkpatrick, M.D.  
Philip I. Niles, M.D., M.B.A.  
David L. Phillips, M.D.

### FIRST-YEAR RESIDENTS

Stephen M. Christiansen, M.D.  
William E. Flanary II, M.D.  
Jaclyn M. Haugsdal, M.D.  
Lucas T. Lenci, M.D.  
Prashant K. Parekh, M.D., M.B.A.

### TRANSITIONAL INTERNS

Thomas T. J. Clark, M.D.  
Lindsay K. McConnell, M.D.  
Matthew A. Miller, M.D.  
Lorraine A. Myers, M.D.  
Tyler B. Risma, M.D.

## RESIDENT / FELLOW RESEARCH DAY – 2015

### GUEST FACULTY

#### **David Gamm, M.D., Ph.D.**

David M. Gamm, M.D., Ph.D., is the Emmett A. Humble Distinguished Director of the McPherson Eye Research Institute, an Associate Professor of Ophthalmology and Visual Sciences at the University of Wisconsin-Madison, and the Sandra Lemke Trout Chair in Eye Research. He is also a member of the Waisman Center Stem Cell Research Program, the UW Stem Cell and Regenerative Medicine Center, and the American Society for Clinical Investigation. Dr. Gamm earned his medical and doctoral degrees from the University of Michigan-Ann Arbor and completed his residency and pediatric ophthalmology fellowship at the University of Wisconsin. In his clinical practice, Dr. Gamm diagnoses and manages a wide range of pediatric eye and vision disorders, including inherited retinal diseases; however, the majority of his effort is directed toward basic and translational retinal stem cell research. The aims of his laboratory are 1) to investigate the cellular and molecular events that occur during human retinal differentiation and 2) to generate cells for use in retinal disease modeling and cell replacement therapies. To meet these goals, his lab developed a novel 3-dimensional optic vesicle culture system using human ES and iPS cells, which has the capacity to model retinal development and disease, as well as to delineate the genetic "checkpoints" necessary to produce particular retinal cell types. By understanding the behavior of these cell types in vitro and in vivo, they hope to optimize strategies to delay or reverse the effects of blinding disorders such as retinitis pigmentosa and age-related macular degeneration.



**OPHTHALMOLOGY RESIDENT/FELLOW RESEARCH DAY  
SCHEDULE OF EVENTS**

**Friday, May 15, 2015, 8:00 AM - 3:20 PM**

- 8:00 Coffee
- 8:30 Welcoming Statements

**8:35 – 10:15 Scientific Papers, Residents, Session I  
Braley Auditorium**

8:35	<b>David Phillips</b> , sponsor, Michael D. Wagoner ..... Boston Type II Keratoprosthesis for Iridocorneal Endothelial Syndromes	1
8:45	<b>William E Flanary, II</b> , sponsor, Mark A. Greiner..... Incidence of Cystoid Macular Edema Following Descemet Membrane Endothelial Keratoplasty	2
8:55	<b>Jaelyn M. Haugsdal</b> , sponsor, Michael D. Wagoner..... Boston Type I Keratoprosthesis for Congenital Glaucoma	3
9:05	<b>Lucas Lenci</b> , sponsor, Richard Allen ..... Dermoid Cysts: Imaging in the Prevention of Complications	4
9:15	<b>Prashant K. Parekh</b> , sponsor, Michael D. Abramoff..... Is fluorescein angiography effective for management of suspected choroidal neovascularization? FFA plus OT versus OCT alone in the diagnosis of choroidal neovascularization	5
9:25	<b>Johanna M. Beebe</b> , sponsors, Randy H. Kardon, Matthew J. Thurtell..... The Yield of Diagnostic Imaging in Patients with Isolated Horner’s Syndrome	6
9:35	<b>P. Christi Carter</b> , sponsors, James C. Folk, Michael D. Abramoff..... Comparison of Early Changes in the Neuroretina to those in the Brain in Diabetes	7
9:45	<b>Christopher A. Kirkpatrick</b> , sponsor, Randy H. Kardon..... Revisiting the definition of a positive apraclonidine test in the diagnosis of an oculosympathetic palsy: an objective assessment of the pupil reaction to apraclonidine in controls, physiologic anisocoria, and Horner syndrome populations	8
9:55	<b>Philip Niles</b> , sponsors, Ali R. Cohen, Scott A. Larson, Richard J. Olson, William E. Scott..... Cost Difference between Optometrists and Pediatric Ophthalmologist for Treating Pediatric Hyperopia: Over \$1 Billion	9
10:05	<b>Steven M. Christiansen</b> , sponsor, Thomas A. Oetting ..... Twitter at the 2014 American Academy of Ophthalmology Annual Meeting	10

**OPHTHALMOLOGY RESIDENT/FELLOW RESEARCH DAY  
SCHEDULE OF EVENTS**

**Friday, May 15, 2015, 8:00 AM - 3:20 PM**

**10:15 – 10:45 Morning Break**

**10:45 – 11:15 Fellow Scientific Papers, Fellows, Session I  
Braley Auditorium**

- 10:45 **Megan Chambers**, sponsor, John H. Fingert ..... 11  
Clinical and Genetic Characterization of a Pedigree with  
Autosomal Dominant Primary Open Angle Glaucoma
- 10:55 **Matthew Weed**, sponsor, Arlene V. Drack ..... 12  
Genetic Testing for Congenital Cataract
- 11:05 **Matthew E. Raecker**, sponsors, Gregory Schmidt, Kenneth M. Goins, Anna  
S. Kitzmann, Michael D. Wagoner, Mark A. Greiner ..... 13  
The exclusion of diabetic tissue from DMEK preparation  
results in decreased rates of tissue preparation failure

**11:15 – 12:00 Keynote Speaker  
David Gamm - Braley Auditorium**

**12:00 – 1:30 Buffet Luncheon and Posters  
Pappajohn Gym 1701 JPP**

**1:30 – 2:20 Fellow Scientific Papers, Fellows, Session II  
Braley Auditorium**

- 1:30 **David R. P. Almeida**, sponsors, Stephen R. Russell, Michael D. Abramoff ..... 14  
Comparison of retinal and choriocapillaris thickness following  
sitting to supine transition in healthy individuals and patients  
with age-related macular degeneration
- 1:40 **Ashley Ko**, sponsors, Arlene Drack, Wanda Pfeifer, Richard Smith ..... 15  
“Is it Usher Syndrome? Misdiagnosis in children with hearing  
loss and ocular findings.”
- 1:50 **Rachel Huckfeldt**, sponsor, Edwin M. Stone ..... 16  
Retinal microarchitecture in Bardet-Biedl syndrome
- 2:00 **Enrique J. Rivera**, sponsor, Randy H. Kardon ..... 17  
Chronic Optic Neuropathy Causes Decreases in both Inner  
Retinal Blood Flow and Prelaminar Optic Nerve Blood Flow

**OPHTHALMOLOGY RESIDENT/FELLOW RESEARCH DAY  
SCHEDULE OF EVENTS**

**Friday, May 15, 2015, 8:00 AM - 3:20 PM**

2:10      **Eric K. Chin** sponsor, Vinit B. Mahajan..... 18  
Elevated Intraocular Pressure after Intravitreal Dexamethasone  
(Ozurdex) Sustained-Release Implant

**2:20 – 2:50**

**Keynotes Speaker- David Gamm**

Career Development for a practicing clinician Scientist

**3:00 – 3:20**

**Faculty Meeting/Judging For Research Day Awards**

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 1

### **Boston Type 1 Keratoprosthesis for Iridocorneal Endothelial Syndromes**

**David L. Phillips, M.D.**

**Primary Supervisor:** Michael D. Wagoner, M.D., Ph.D.

**Other Supervisors:** Kenneth M. Goins, M.D., Anna S. Kitzmann, M.D., Mark A. Greiner, M.D.

**Background/Purpose:** To evaluate the outcome of the Boston type 1 keratoprosthesis (Kpro-1) in eyes with iridocorneal endothelial syndromes.

**Patients and Methods:** A retrospective review was performed of every eye with a history of iridocorneal endothelial syndrome that was treated with a Kpro-1 at a tertiary eye care center between January 1, 2008 and July 1, 2014. The main outcome measures were visual outcome, prosthesis retention, and postoperative complications.

**Results:** Four eyes of 4 patients with met the inclusion criteria. Two eyes had Chandler's syndrome and 2 eyes had essential iris atrophy. All 4 eyes had failed corneal transplants. All 4 eyes had had previous glaucoma shunts and satisfactory intraocular pressure control at the time of surgery. Preoperatively, the BCVA in the operative eye was worse than 20/250 in all 4 eyes, including 2 eyes that were hand motions (HM). The mean patient age at the time of Kpro-1 was 68.3 years (range, 60 to 80 years). After Kpro-1, all 4 (100%) eyes initially experienced improved visual acuity, with BOVA of 20/20, 20/25, 20/50, and 20/70. After a mean follow-up of 47 months (range, 27-69 months), the BCVA was still improved compared to the preoperative vision in 3 eyes, with final acuities of 20/25, 20/63, and 20/100. The Kpro-1 was retained in all 4 (100%) eyes, with no cases of wound dehiscence, sterile ulceration, or microbial keratitis. One (25%) eye developed absolute glaucoma, became no light perception (NLP), and required enucleation due to chronic pain.

**Conclusion:** Kpro-1 offers a reasonable prognosis for graft retention and visual rehabilitation in eyes iridocorneal endothelial syndrome and one or more previous failed keratoplasties.



## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 2

### **Incidence of Cystoid Macular Edema Following Descemet Membrane Endothelial Keratoplasty**

**William E. Flanary, M.D.**

**Supervisor:** Mark A. Greiner, M.D.

**Background/Purpose:** Descemet membrane endothelial keratoplasty (DMEK) is a relatively new surgical technique used to treat disorders of the corneal endothelium including Fuchs dystrophy and pseudophakic bullous keratopathy. Cystoid macular edema (CME) is a known complication of intraocular surgery, particularly following phacoemulsification in which the incidence of postoperative CME ranges from 0.1-2.4%. Few studies have evaluated CME associated with DMEK. The purpose of our study was to evaluate the incidence of postoperative CME following uncomplicated DMEK surgery.

**Patients and Methods:** A retrospective review was performed of eyes that underwent DMEK at the University of Iowa between October 2012 and November 2014. For purposes of statistical analysis, eyes were divided into three groups based on the timing of DMEK surgery in relation to cataract surgery: eyes that underwent DMEK  $\geq 6$  months after cataract surgery (Group 1), between 2 weeks and 6 months after cataract surgery (Group 2), or combined with cataract surgery (Group 3). A minimum follow-up of 6 months after DMEK surgery was required for inclusion in the statistical analysis.

**Outcome Measures:** The main outcome measure was the incidence of visually significant postoperative CME, which was defined as the presence of characteristic cystic changes on macular OCT. Macular OCT imaging was obtained when the BCVA was  $\leq 20/30$  in the presence of a clear cornea  $\geq 1$  month after DMEK and without other reason for visual compromise.

**Results:** Of the 196 eyes that met criteria for inclusion in this study, 14 (7.1%) were found to have visually significant CME after DMEK. Eight of 94 eyes (4.1%) in Group 1 developed CME after DMEK performed  $\geq 6$  months after cataract surgery. Five of 86 eyes (2.5%) in Group 2 developed CME after DMEK was performed  $< 6$  months after cataract surgery. One of 16 eyes (0.5%) in Group 3 was diagnosed with CME after combined phacoemulsification and DMEK surgery. There were no significant statistical differences between groups.

**Conclusion:** The incidence of CME following DMEK may be higher than the reported incidence following phacoemulsification. Performing a staged surgical procedure does not appear to increase the risk of CME after DMEK.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 3

### **Boston Type I Keratoprosthesis for Congenital Glaucoma**

**Jaclyn M. Haugsdal, M.D.**

**Supervisor:** Michael D. Wagoner, M.D. Ph.D.

**Co-authors:** Kenneth M. Goins M.D.; Mark A. Greiner M.D.; Young Kwon, M.D. Ph.D; Wallace L.M. Alward M.D.

**Background/Purpose:** To evaluate the outcome of the Boston type 1 keratoprosthesis (Kpro-1) in eyes with a loss of corneal clarity in association with congenital glaucoma.

**Patients and Methods:** A retrospective review was performed of every eye with a history of congenital glaucoma that was treated with a Kpro-1 at a tertiary eye care center between January 1, 2008 and July 1, 2014. The main outcome measures were visual outcome, prosthesis retention, and postoperative complications.

**Results:** Eight eyes of 8 patients met the inclusion criteria. The mean patient age at the time of Kpro-1 was 26.3 years (range, 0.5 to 50 years). Preoperatively, the best corrected visual acuity (BCVA) in the operative eye was  $\geq 20/400$  in only 1 (12.5%) eye. After a mean follow-up period of 38.1 months (range, 16 to 52 months), 3 (37.5%) eyes had a BCVA that was  $\geq 20/400$ , although no eyes were better than 20/300. Two eyes were hand motions, 2 eyes were light perception, and 1 eye was no light perception. The Kpro-1 was retained in 7 (87.5%) eyes. Sight-threatening complications were common and included 3 (37.5%) cases of wound dehiscence secondary to sterile ulceration, 3 (37.5%) retro-prosthetic membranes, 2 (25.0%) cases of progressive optic neuropathy, 1 (12.5%) epiretinal membrane, and 1 (12.5%) total retinal detachment.

**Conclusion:** The Boston Kpro-1 has an excellent prognosis for retention in eyes with a loss of corneal clarity due to congenital glaucoma, but has a very guarded prognosis for successful visual rehabilitation due to a high incidence of sight-threatening postoperative complications, including progressive optic neuropathy.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 4

### Dermoid Cysts: Imaging in the Prevention of Complications

Lucas T. Lenci, M.D.

Supervisors: Richard Allen M.D., Ph.D.

Other Supervisors: Meredith Baker, M.D.

**Background/Purpose:** Dermoid Cysts are very common lesions, particularly in children, in the periorcular region. Dermoids account for around 5% of all orbital lesions and up to 46% in children (1,2). The goals of surgical excision include complete removal without rupture of the cyst. These lesions can sometimes present a diagnostic conundrum as they can have a deep orbital component (dumbbell shape), intracranial extension, or mask an alternate diagnosis such as encephalocele. Imaging dermoid cysts, particularly in children, presents quite a burden to the patient, families and the healthcare system. To our knowledge, there are no guidelines on when to image these patients. Some authors have suggested medially located masses, immobility of the lesion, and inability to palpate around the masses as reasons to image these patients (3,4).

The purpose of this research is to evaluate the role of pre-operative imaging of dermoid cysts and the predictors of complications, such as cyst rupture.

**Methods:** A retrospective chart review was performed on our patient database at the University of Iowa over the last 15 years. Charts were found using clinical diagnosis codes as well as pathologically confirmed dermoid cysts. Only patients who underwent surgery were analyzed.

**Results:** 100 consecutive dermoid patients that underwent surgical excision were analyzed. Mean age was 5.8 years with 75% of lesions located superotemporally. Imaging was obtained in 42% of patients. 3 patients were found to have dumbbell shaped dermoids with large intraorbital extension. 1 unusual dermoid was nonpalpable and located in the posterior orbit. These 4 patients, unlike the others, presented with orbital signs such as hypoglobus, strabismus, or proptosis. Cyst rupture during surgery was more common in medial cysts (39%). Immobility was present in all complex dermoids.

Gender	Location	Immobility (%)	Imaging (%)	Cyst Rupture (%)	
38 Female 62 Male	54 Right 46 Left	75 Superotemporal 17 Superomedial 8 Other	54 mobile 21 immobile* <small>All complex lesions were immobile or nonpalpable</small>	11 MRI 22 CT 6 US 3 multiple	10 Superotemporal (13%) 7 Superomedial (39%) 1 nonpalpable

**Conclusion:** No study, to our knowledge, has looked at how often imaging is performed in these patients, nor when obtaining imaging is appropriate. According to our data, imaging is performed too frequently. Orbital signs alone should be the main factor which guides the clinician to obtain imaging. Forty two percent of our cohort were imaged, but intra-operative and clinical management was altered in only 9% of those imaged patients. No imaging needed in medial lesions or immobile lesions. Interestingly, there is a higher risk of rupture in medial lesions (39%).

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 5

**Is fluorescein angiography effective for management of suspected choroidal neovascularization? FFA plus OCT versus OCT alone in the diagnosis of choroidal neovascularization.**

**Prashant K. Parekh, M.D., M.B.A.**

**Supervisor:** Michael D. Abramoff, M.D., Ph.D.

**Other Supervisors:** David RP Almeida, B.S.c(Hon), M.D., M.B.A., Ph.D, F.R.C.S.C., Priya Gupta, M.D.

**Background/Purpose:** Fluorescein angiography (FA) has long been the standard modality to diagnose and manage choroidal neovascularization (CNV), and is a requirement

for Medicare reimbursement for managing this condition. However, FA is costly, has a mortality of 1 per 220000, and considerable morbidity from allergic reactions. Since the advent

of anti-VEGF therapy for CNV, optical coherence tomography (OCT), a non-invasive imaging method free of these disadvantages, is used extensively to manage CNV, while FA is

primarily used to make the initial diagnosis. A recent study found the sensitivity and specificity of OCT compared to FFA in diagnosis of CNV to be 100 and 80.8%, respectively. We hypothesize that FA changes the management of patients that are initially suspected of having CNV in less than 5% of cases. If this hypothesis is confirmed, it would cast doubt on the clinical as well as cost-effectiveness of FA for diagnosing CNV, except in treatment failures and non-standard cases.

**Methods:** We will retrospectively review the FA, OCT, and clinical histories of 200 initial visits from 200 patients (200 eyes) who had an initial presentation of confirmed CNV. After

de-identification, three retinal specialists masked to each other will review, in randomized order, the standardized brief clinical history, the posterior pole color fundus image, and

complete OCT scan of the initial visit, and choose whether they will manage each case by three consecutive injections, further imaging, or other (FA- arm). After re-randomization, corresponding early, mid, and late phase FA images will be added to each patient's case data, and the three experts will again choose from these 3 management options (FA+). We will determine for each expert, the case discordance (i.e., the percentage of cases where they differed between FA- and FA+) and inter-observer discordance (i.e., percentage

of cases where all 3 experts differed). We will also devise a random model of mixed effects to look at interaction with patient age and fellow eye history.

**Results:** Pending

**Conclusion:** Pending

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 6

### The Yield of Diagnostic Imaging in Patients with Isolated Horner's Syndrome

Johanna D. Beebe, M.D.

**Supervisors:** Randy H. Kardon, M.D., Matthew J. Thurtell, M.D.

**BACKGROUND:** Acute Horner's syndrome is a neuro-ophthalmic emergency that is caused by a lesion affecting the oculosympathetic pathway. There are a large number of potential etiologies, including internal carotid artery dissection, stroke, and malignancy. The presence of associated symptoms or signs sometimes helps to localize the lesion and determine the etiology. However, Horner's syndrome is often isolated with no localizing symptoms or signs. Imaging of the entire oculosympathetic pathway (magnetic resonance imaging and magnetic resonance angiography) is often pursued in patients with isolated Horner's syndrome, but the yield of diagnostic imaging has not been evaluated in a large series of patients.

**PURPOSE:** To determine the yield of diagnostic imaging (MRI/MRA) evaluating the entire oculosympathetic pathway in a large series of patients with isolated Horner's syndrome.

**METHODS:** We conducted a retrospective chart review of 123 patients seen by our service between 2000 and 2014, who were coded as having Horner's syndrome with clinical and/or pharmacologic confirmation of the diagnosis. We excluded infants and patients with congenital Horner's syndrome.

**RESULTS:** Patient age ranged from 6 to 87 years old, with an average age of 50 years old. There were 123 cases of clinically- or pharmacologically-confirmed Horner's syndrome, with 63 male and 60 female patients. On initial presentation, 19 patients had a known cause or constellation of findings that indicated the likely etiology of their Horner's syndrome. Imaging of the oculosympathetic pathway was pursued in 94 patients (76.4%). Prior to imaging, 5 of the 94 patients (5%) had a known etiology for their Horner's syndrome. There were 89 patients with an isolated Horner's syndrome who underwent imaging in search of a causative lesion. Of these, 17 (19%) were found to have a lesion on imaging that was causative for the Horner's syndrome. The most common finding was carotid artery dissection in 7 patients (8%), with all but one having an acute-onset painful Horner's syndrome. None of the imaged patients were found to have a primary malignancy, but one patient with known metastatic disease was found to have a new metastatic lung lesion. Seven of the patients (7%) had incidental findings on imaging that were unrelated to the oculosympathetic defect.

**CONCLUSIONS:** To our knowledge, this is the largest series evaluating the diagnostic yield of neuroimaging for isolated Horner's syndrome. Imaging identified a causative lesion in 19% of patients with an isolated Horner's syndrome. About 7% of patients had an incidental finding on neuroimaging that was unrelated to the oculosympathetic defect.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 7

### Comparison of Early Changes in the Neuroretina to those in the Brain in Diabetes

**P. Christi Carter, M.D.**

**Supervisors:** James Folk, M.D.; Michael Abramoff, M.D., Ph.D.

**Other Supervisors:** Elliott Sohn, M.D.

**Background/Purpose:** Diabetes mellitus is associated with vision loss. Recent evidence indicates that diabetes mellitus is also associated with cognitive decline and cerebral atrophy. For years, the pathogenesis of diabetes mellitus has been attributed to microvascular changes. However, recent evidence suggests that neurodegeneration may precede these vascular changes in the pathogenesis. This study seeks to determine if there is a correlation between neuronal degeneration in the brain to that in the retina. It is possible to measure retinal neuronal thickness (specifically in the ganglion cell layer and nerve fiber layer) through optical coherence tomography (OCT) imaging of the retina. Advances in computer analysis of magnetic resonance imaging (MRI) of the brain also allow for measurement of cerebral thickness in specific Brodmann areas. The retina is an embryologic extension of the brain; thus if a correlation is found between retinal neuronal thickness and cerebral thickness in specific Brodmann areas in the brain, the retina could serve as an easily accessible structure to further study the cause and natural history of neuronal degeneration in the brain in patients with diabetes. Furthermore, OCT of the retina could potentially be used as a fast, cost-effective, and patient-friendly screening tool for cognitive decline in the future.

**Methods:** A cross-sectional pilot study will be performed in 10 people with Type I diabetes mellitus of at least 15 years duration, with plans to extend the study to 100 subjects subsequently. Subjects who have no or minimal diabetic retinopathy will be preferentially selected. After informed consent, each subject will undergo OCT of the retina, fundus photography, and MRI of the brain. Fully-automated segmentation algorithms (that have been previously validated) will be applied to the images obtained from OCT and MRI to quantify the thickness of specific layers in the retina and specific Brodmann areas in the brain, respectively. The brain and retinal measurements will be analyzed using a mixed model to determine if there is early neuronal degeneration and whether the changes in the eye and brain are correlated.

**Results:** *Pending* - The study has IRB approval. The team of study investigators, including ophthalmologists, an endocrinologist, and electrical engineers, has been assembled. One subject has completed the study and there is ongoing recruitment. An application to obtain MRIs at no cost to the first subjects has also been approved.

**Conclusion:** Pending

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 8

**Revisiting the definition of a positive apraclonidine test in the diagnosis of an oculosympathetic palsy: an objective assessment of the pupil reaction to apraclonidine in controls, physiologic anisocoria, and Horner syndrome populations**

**Christopher A. Kirkpatrick, M.D.**

**Supervisor:** Randy H. Kardon, M.D., Ph.D.

**Background:** The clinical diagnosis of Horner Syndrome (HS) is based on miosis, ptosis and anhidrosis and can be equivocal, requiring confirmation with pharmacological eye drop testing. In recent years, apraclonidine, an alpha-2 agonist with weak alpha-1 properties, has gained acceptance over cocaine testing for the diagnosis of HS. However, criteria for a positive apraclonidine test for diagnosing HS have been lacking. A positive test, currently defined as reversal of anisocoria, is influenced by the development of alpha-1 adrenergic supersensitivity and its magnitude, which depends on the duration of the oculosympathetic deficit and the amount of deficit causing it.

**Purpose:** Objective criteria for a positive apraclonidine test for diagnosing HS were developed based on ocular responses of normal subjects to topical apraclonidine which were compared to patients.

**Methods:** Pupillary measurements in 100 normal healthy control subjects were obtained from digital infrared video frames before and 30 minutes after one drop of 0.5% apraclonidine OU in dim light. Pupillary responses were analyzed as a ratio of pupil/limbus diameter (P/I ratio) and the normal range of pupil response to apraclonidine was objectively defined. A retrospective chart review of patients receiving apraclonidine testing – carrying either the diagnosis of physiologic anisocoria or HS (based on cocaine testing, imaging or clinical objective criteria other than a positive apraclonidine test) – were evaluated against these results to critically assess the sensitivity and specificity of the apraclonidine test and to propose objective criteria for its use to diagnose HS.

**Results:** In the normal population (N=100), after apraclonidine, the mean change in pupil diameter was  $-0.044 \pm 0.033$  (range -0.142 to 0.088) using the P/I ratio. The mean change in anisocoria was  $0.019 \pm 0.016$  (range 0.00004 to 0.076) using the P/I ratio. In the HS group (N=32), the mean change in anisocoria was  $0.133 \pm 0.051$  (range 0.053 to 0.233). When compared to the control population, 29 of 32 patients (90.63%) fell outside of the 5<sup>th</sup>-95<sup>th</sup> predictive limits of a normal apraclonidine response. In the physiologic anisocoria group (N=38), the mean change in anisocoria was  $0.029 \pm 0.025$  (range -0.090 to 0.076). When compared to the control population, 1 of 38 patients (2.63%) fell outside of the 5<sup>th</sup>-95<sup>th</sup> predictive limits of a normal apraclonidine response. Comparing the HS group and the physiologic anisocoria group (N=70) to the 5<sup>th</sup>-95<sup>th</sup> predictive limits of a normal apraclonidine response results in a sensitivity of 90.62% (95% CI: 74.95% - 97.91%) and a specificity of 97.37% (95% CI: 86.14% - 99.56%).

**Conclusion:** Pupil responses to apraclonidine in normal subjects and in patients can be utilized to develop objective criteria for the diagnosis of HS. Some patients with Horner syndrome show a decrease in anisocoria with apraclonidine, indicating a positive test, even though the change is not great enough to result in a reversal of anisocoria.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 9

### **Cost Differences between Optometrists and Pediatric Ophthalmologist for Treating Pediatric Hyperopia: Over \$1 Billion**

**Philip Niles, M.D.**

**Supervisors:** Ali R. Cohen, M.D., Scott A. Larson, M.D., Richard J. Olson, M.D., William E. Scott, M.D.

**Introduction:** Optometrists and pediatric ophthalmologists have different practice patterns when prescribing hyperopic correction for children. This study examines the difference in costs between the optometric and pediatric ophthalmologic practice patterns.

**Methods:** A literature review was conducted to determine the prescription recommendations of optometrists and ophthalmologists. Race-specific population data was retrieved from 2010 US Census Bureau records for children ages 2- to 17-years-old. The Baltimore Pediatric Eye Disease Study provided data on the incidence of hyperopia in children ages 2- to 6-years-old. A sensitivity analysis was employed to determine the effect of the incidence of hyperopia in children ages 7- to 17-years-old. The number of children who would meet optometric and ophthalmologic prescription recommendations was determined. The total one-time cost of each group's recommendations was then determined by multiplying the number of children who would receive spectacle correction by the median cost of pediatric spectacles (\$245), which was determined from a 2013 industry survey of 171 independent optical retailers. The cost of glasses wear was conservatively limited to the initial purchase price of one pair of spectacles. Further costs, such as a child's second pair of glasses, refraction, time off of work or glasses repairs were conservatively excluded.

**Results:** There were 69,420,516 American children between the ages of 2- and 17-years-old, inclusively. There are 1.23 million children between the ages of 2- and 6-years-old who have a refraction that would receive glasses from an optometrist but not from a pediatric ophthalmologist for a cost difference of \$300.1 million. The cost difference for children ages 7- to 17-years-old was between \$666 million and \$1.76 billion, depending on the incidence of hyperopia in school-age children that would be treated by optometrists but not pediatric ophthalmologists. The total cost difference between the practice patterns of optometrists and pediatric ophthalmologists for children ages 2- to 17-years old is between \$966 million and \$2.06 billion.

**Discussion:** The difference in practice patterns for pediatric hyperopic correction between optometrists and pediatric ophthalmologists leads to significantly different costs. This difference would be even larger if the need for subsequent glasses and spectacle repair were included.

**Conclusion:** There is a large cost difference between the hyperopic correction recommendations followed by optometrists and ophthalmologists. The cost of healthcare recommendations should be taken into consideration when making healthcare policy.



## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 10

### Twitter at the 2014 American Academy of Ophthalmology Annual Meeting

Steven M. Christiansen, M.D.

Supervisor: Thomas Oetting, M.D

**Background:** The use of social media within medicine is rapidly expanding. In recent years, many physicians, academic journals, professional societies and industry groups have begun using Twitter to share information. Twitter is a popular microblogging service, which allows users to publicly share messages (tweets) of up to 140 characters. Perhaps the best demonstration of how Twitter is being used within medicine is at medical conferences, wherein participants use a designated hashtag (e.g. #AAO2014) within the text of their tweet to enable other Twitter users to follow the dialogue of tweets containing the same searchable hashtag. The medical literature contains multiple peer-reviewed articles within several specialties documenting the use of Twitter at medical conferences. At the time of this study, however, the use of Twitter to document conference proceedings within ophthalmology remains unknown.

The aim of this study is threefold: 1) to document the number of tweets published using the #AAO2014 hashtag, 2) to provide a clear method by which future studies may categorize tweets at medical conferences, and 3) to analyze the demographics of Twitter users who shared original content at the 2014 American Academy of Ophthalmology Annual Meeting.

**Methods:** All tweets published during the five days of the 2014 American Academy of Ophthalmology (AAO) Annual Meeting with the hashtag #AAO2014 included in the tweet were collected using commercially available services and aggregated for data analysis. Tweets were categorized as original tweets (original content), retweets (forwarding of a previously published tweet to one's followers), or modified retweets (modification of a previously-published tweet followed by forwarding the tweet to one's followers.) The content of each original tweet was analyzed according to carefully determined criteria and categorized as being primarily meeting-related, logistical, social, or advertising. The public profiles of Twitter users who published original tweets were analyzed based on self-identification as an ophthalmologist or as representing an ophthalmology practice or academic ophthalmology department.

**Results:** A total of 1,067 Twitter users published a total of 4,539 tweets using the #AAO2014 hashtag during the five days of the 2014 AAO Annual Meeting, of which 2,193 were original tweets (48% of total), 2,299 were retweets (51% of total), and 47 were modified retweets (1% of total). A total of 345 Twitter users from 25 countries published original tweets, 41% of which contained content categorized as primarily meeting-related, 33% advertising, 16% social, and 10% logistical. One out of every four Twitter accounts that published original content was registered to an ophthalmologist (18%), ophthalmology practice (5%), or US academic ophthalmology department (2%).

**Conclusion:** Twitter represents a new forum for the sharing of information at ophthalmology conferences, providing meeting participants a medium with which to engage in scholarly discussion and to share ophthalmology-related information with a digital audience worldwide.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 11

### Clinical and Genetic Characterization of a Pedigree with Autosomal Dominant Primary Open Angle Glaucoma

**Megan Chambers, M.D.**

**Supervisor:** John H. Fingert, M.D., Ph.D.

**Other Supervisors:** Ben R. Roos, Ph.D., Emily Greenlee, M.D., Edwin M. Stone, M.D., Ph.D., Wallace L.M. Alward, M.D.

**Background:** Glaucoma is a group of diseases that cause optic nerve damage. It is characterized by progressive optic nerve cupping, which can lead to vision loss. This disease is often insidious and affected patients are frequently asymptomatic. Prompt recognition of the disease and its risk factors is important in order to provide timely treatment and prevent visual disability. Many genes have been identified to carry a significant role in the pathogenesis of glaucoma and several of these genes were discovered through pedigree-based and population-based studies.

**Purpose:** To clinically and genetically characterize a large Caucasian pedigree with autosomal dominant primary open angle glaucoma (POAG).

**Methods:** Twenty-one members of a large Caucasian pedigree (Pedigree GGA-882) had complete eye examinations and were assessed for the presence of POAG. DNA samples were collected from all available family members and were studied with linkage analysis and DNA sequencing to search for the gene that causes POAG in this pedigree.

**Results:** Ten members of Pedigree GGA-882 were diagnosed with POAG that is transmitted as an autosomal dominant trait. Members of the family with POAG had a mean age at diagnosis of 56 years, a mean CCT of 550 microns (n=12 eyes), a mean maximum intraocular pressure (IOP) of 26 mm Hg (n=14 eyes). Testing for glaucoma-causing mutations in known genes (*MYOC*, *OPTN*, *TBK1*) was negative. Linkage analysis of Pedigree GGA-882 identified the *GLC1R* locus on chromosome 4 and that is co-inherited with disease in this family.

**Discussion:** Here we report identification and characterization of a large pedigree with POAG that is inherited as an autosomal dominant trait. No glaucoma-causing mutations were identified in known glaucoma-causing genes, suggesting that Pedigree GGA-882 has disease caused by an as yet undiscovered glaucoma gene. Linkage analysis of this family identified a novel glaucoma locus (*GLC1R*) on chromosome 4. These studies represent the first steps towards discovering the glaucoma-causing gene in this locus.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 12

### Genetic Testing for Congenital Cataract

**Matthew C. Weed, M.D.**

**Supervisor:** Arlene V. Drack, M.D.

**Other Supervisors:** Scott R. Lambert M.D., Scott A. Larson M.D., Richard J. Olson M.D., Susannah Q. Longmuir M.D., Adam P. DeLuca Ph.D., Jeremy M. Hoffmann B.A., Edwin M. Stone M.D., Ph.D.

**Background/Purpose:** Congenital cataracts are a major cause of treatable blindness in children worldwide. The many causative genes and inheritance patterns make genetic testing complex. We present a tiered strategy for genetic testing of congenital cataracts.

**Methods:** Congenital cataract patients from two pediatric ophthalmology services were offered research-based genetic testing. Literature review of causative mutations published at least twice was used to develop a Sanger sequencing pre-screen of 24 exons in 11 genes (*BFSP2*, *CRYAA*, *CRYBA1*, *CRYBB2*, *CRYGD*, *EPHA2*, *FAM126A*, *FYCO1*, *NHS*, *PAX6* and *VSX2*). Individuals with negative pre-screens received whole exome sequencing with analysis focused on lens-related genes. Putative disease-causing variants were verified by Sanger sequencing. Allele frequencies, familial segregation and predicted effects on protein structure were used to infer pathogenicity.

**Results:** Thirty-one families were studied. Pre-screening identified likely causative variants in three. Two had mutations in *CRYGD* and one in *CRYBA1*. Two additional families harbored possible disease-causing variants, one in *CRYAA* and one in *NHS*. The remaining 26 probands had whole exome sequencing performed. 23 were found to have protein-altering mutations in one or more lens-related genes. Four families with previously identified cataract-causing mutations served as a comparison group.

**Conclusion:** Exome sequencing can be used to simultaneously screen multiple genes for disease-causing mutations and is useful for genetically heterogeneous diseases like congenital cataract. However, non-disease causing variants are common in humans making it difficult to determine which if any of the observed variants is responsible for a patient's disease unless multiple affected individuals are available for segregation analysis. A Sanger-sequencing-based pre-screen for known mutations decreases cost and increases testing efficiency.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Morning Session – Paper 13

### **The exclusion of diabetic tissue from DMEK preparation results in decreased rates of tissue preparation failure**

**Matthew E. Raecker, M.D.**

**Other Supervisors:** Gregory A. Schmidt, B.S., CEBT; Kenneth M. Goins, MD; Anna S. Kitzmann, M.D., Michael D. Wagoner, M.D., Ph.D., Mark A. Greiner, M.D.

**Purpose:** To compare the rate of tissue loss during preparation of Descemet's membrane endothelial keratoplasty (DMEK) grafts before and after the exclusion of diabetic donors at a single eye bank.

**Methods:** 418 consecutive donor corneas were pre-stripped for DMEK by experienced technicians at Iowa Lions Eye Bank. After July 19, 2013, corneal tissues from donors with a medical history of diabetes mellitus were excluded from DMEK preparation. The main outcome measure was the rate of unsuccessful (failed) DMEK graft preparation, defined as tears through the graft area preventing tissue use.

**Results:** 73 corneas from diabetic and non-diabetic donors were prepared before exclusion of diabetic donors on July 19, 2013, and 345 corneas from non-diabetic donors were prepared between July 20, 2013 and August 19 2014. DMEK graft preparation failure occurred in 6 cases (6/73, 8.22%) prior to the exclusion of diabetic donors, and in 8 cases (9/421, 2.1%;  $p < 0.05$ ) after exclusion of diabetic donors. All cases of graft preparation failure prior to exclusion of diabetic donors occurred in tissues from diabetic donors.

**Conclusion:** This study suggests that exclusion of diabetic donors from DMEK preparation results in a decreased rate of tissue loss during preparation. Findings support our previous study demonstrating that tissue preparation may be more likely to fail when the donor has a history of diabetes mellitus.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Afternoon Session – Paper 14

### **Comparison of retinal and choriocapillaris thicknesses following sitting to supine transition in healthy individuals and patients with age-related macular degeneration**

**David R.P. Almeida, M.D., MBA, Ph.D.**

**Primary Supervisors:** Stephen R Russell MD. & Michael D. Abramoff M.D., Ph.D.

**Other Supervisors:** Li Zhang Ph.D., Eric K. Chin M.D., Robert F. Mullins Ph.D., Murat Kucukcilioglu M.D., D. Brice Critser, Milo Sonka Ph.D., Edwin M. Stone M.D., Ph.D., James C. Folk M.D.

**Background:** The effects of position on retinal and choroidal structure are absent from the literature yet may provide insights into disease states such as age-related macular degeneration (AMD).

**Purpose:** To evaluate the effect of postural change on retinal and choroidal structures in healthy volunteers and patients with non-neovascular AMD.

**Methods:** Prospective observational case series at an academic tertiary care retina service from September 2013 to April 2014 involving 4 unaffected volunteers (8 eyes) and 7 patients (8 eyes) with intermediate AMD. Healthy volunteers selected for the study had no evidence of ocular disease. Patients with AMD were required to have at least 10 intermediate-sized drusen. Spectral-domain optical coherence tomography with enhanced depth imaging was performed in the upright (sitting) and supine positions. Stable imaging was achieved using a rotating adjustable mechanical arm that we constructed to allow the optical coherence tomography transducer to rotate 90 degrees. The Iowa Reference Algorithms were used to quantify choroid and choriocapillaris thicknesses. Main outcome measures were defined as changes in sitting and supine position central macular thickness (micrometers), total macular volume (cubic millimeters), choroidal thickness (micrometers), and choriocapillaris-equivalent thickness (CCET, micrometers).

**Results:** Choriocapillaris-equivalent thickness was thinner in healthy participants (9.89  $\mu\text{m}$ ; range, 7.15-12.5  $\mu\text{m}$ ) compared with patients with intermediate AMD (16.73  $\mu\text{m}$ ; range, 10.31-27.38  $\mu\text{m}$ ) ( $P = .02$ ); there was no difference in overall choroidal thickness between the 2 groups ( $P = .38$ ). There was a 15% CCET reduction among healthy participants when transitioning from a sitting (9.89  $\mu\text{m}$ ) to supine (8.4  $\mu\text{m}$ ; range, 6.92-10.7  $\mu\text{m}$ ) position ( $P = .02$ ) vs a CCET reduction of 11.1% from sitting (16.73  $\mu\text{m}$ ) to supine (14.88  $\mu\text{m}$ ; range, 8.76-20.8  $\mu\text{m}$ ) positioning ( $P = .10$ ) in patients with intermediate AMD.

**Conclusion:** Intermediate AMD appears to be associated with an increase in CCET and with a lack of positional responses that are observed in the CCET of normal eyes. Our results suggest that although outer portions of the choroid do not appear to be responsive to modest positional or hydrostatic pressure, the choriocapillaris capacity is, and this is measurable in vivo. Whether this physiologic deviation that occurs in AMD is related to atrophy, inflammation, or changes in autoregulatory factors or growth factors remains to be determined.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Afternoon Session – Paper 15

**Is it Usher Syndrome? Misdiagnosis in children with hearing loss and ocular findings.**

**Ashley Ko, M.D., FRCSC**

**Supervisor:** Arlene Drack, M.D.

**Other Supervisors:** Wanda Pfeifer, COMT, OC(C) and Richard Smith, M.D., Ph.D.

**Background:** Children with combined hearing and vision loss often are referred to the Paediatric Genetic Eye Disease service. We created a combined Genetic Eye/Ear clinic to reduce the need for multiple visits and allow collaboration between Genetic Eye and Ear departments at the University of Iowa. Because Usher syndrome is one of the most commonly recognized genetic disorders presenting with both vision and hearing loss, these patients are often given an initial diagnosis of Usher syndrome before complete workup is performed, sometimes incorrectly. Other conditions should also be considered for these patients because a misdiagnosis may be as difficult to disprove as the correct diagnosis is to confirm.

**Purpose:** To improve diagnostic accuracy in the work-up of patients with combined deafness and decreased vision by analyzing patient data from the Genetic Eye/Ear clinic.

**Design and Method:** This study single centre study is a retrospective chart review. We looked at all patients referred to the Paediatric Genetic Eye Disease services for evaluation of an ocular disorder associated with sensorineural hearing loss. Genotype-phenotype correlation was made for all included subjects.

**Results:** Seventeen patients were identified and included in the study [11 (64.7%) males, 6 (35.3%) females]. Age at referral averaged 5.9 years (range 8 months to 30 years). Referring diagnoses included Usher syndrome (9), nonspecific nystagmus (3), dominant optic atrophy (1), optic nerve atrophy (1), cone dystrophy (1), fourth nerve palsy/esotropia (1), and ocular motor apraxia (1). Of the 9 patients with suspected Usher syndrome, 7 were confirmed to truly have the condition. Other final diagnoses included Baraitser-Winter syndrome, Waardenburg syndrome, Harboyan syndrome, Cowchock syndrome and a complex deletion-duplication syndrome.

**Conclusion:** Etiology of visual complaints in children with hearing loss may be rare and atypical. ERG is helpful but often not diagnostic. Molecular genetic testing provides the most accurate diagnosis, but requires parental blood samples and detailed analysis of DNA variants for accuracy. Great care must be taken in the interpretation of the genetic results, taking into account pathogenicity of variants, the fact that Usher syndrome is a recessive disease and its genes are polymorphic.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Afternoon Session – Paper 16

### Retinal microarchitecture in Bardet-Biedl syndrome

Rachel Huckfeldt, M.D., Ph.D.

**Supervisor:** Edwin Stone, M.D., Ph.D.

**Other Supervisors:** Scott Whitmore, Ph.D; Li Zhang, Ph.D.; Robert Mullins, Ph.D; Michael Abramoff, M.D., Ph.D.

**Background/Purpose:** Optical coherence tomography (OCT) allows detailed visualization of retinal microarchitecture that can provide unique insights into inherited retinal diseases. This information can have relevance toward understanding mechanisms of disease and may influence future therapeutic choices. In addition, awareness of the association of particular genes with specific OCT phenotypes allows strong clinical hypotheses to guide genetic testing.

Bardet Biedl syndrome (BBS), which consists of retinitis pigmentosa (RP) in association with systemic features including polydactyly and truncal obesity, is genetically diverse with at least 15 causative genes identified. Moreover, RP can occur in the absence of clinically apparent syndromic features. Identifying a characteristic OCT phenotype for BBS may speed clinical diagnosis in non-syndromic individuals as well as provide insights into pathophysiology.

**Methods:** In this retrospective study, OCT images were analyzed from patients with clinical diagnoses of BBS as well as non-syndromic autosomal recessive RP secondary to BBS mutations. OCT from individuals without known retinal disease served as positive controls. Retinal thickness in the two populations was measured at multiple distances from the fovea, and segmentation of retinal layers was also performed.

**Results:** Preliminary data demonstrates decreased central macular thickness in four BBS patients compared to normal individuals. In contrast, non-foveal retinal thickness was similar in the two populations despite the decreased thickness of the outer retina in BBS patients. Segmentation of individual retinal layers revealed decreased thickness of several cellular layers but increased thickness of layers including the nerve fiber layer and Henle fiber layer in BBS patients.

**Conclusion:** Patients with BBS appear to have unique features on OCT that may distinguish them from other forms of RP. Additional genotype-specific analysis may identify factors contributing to these features. In addition, longitudinal analysis of individual patients will provide additional insight into how ubiquitous this phenotype is among BBS patients and how it relates to visual function.

## RESIDENT / FELLOW RESEARCH DAY – 2015

Afternoon Session – Paper 17

### **Chronic Optic Neuropathy Causes Decreases in both Inner Retinal Blood Flow and Prelaminar Optic Nerve Blood Flow**

**Enrique J. Rivera, M.D.**

**Supervisor:** Randy Kardon, M.D., Ph.D.

**Other Supervisors:** Susan Anderson, B.S., Matthew Thurtell, M.B.B.S., M.S.c., Michael Wall, M.D., Robert Mallery, M.D.

**Background/Purpose:** Our purpose was to determine if optic neuropathy results in an obligatory decrease in blood flow to the inner retina and optic nerve due to decreased metabolic demand from non-functioning neurons. Visual stimulation causes transient hyperemia, but it is unknown if decreases in neuron activity cause reduced blood flow.

**Methods:** Laser speckle blood flowgraphy imaging (LSFG-NAVI; Softcare Ltd, Fukuoka, Japan) was performed on a 25x20 degree area of the fundus incorporating the optic nerve head. Blurring of the laser speckle pattern by moving particles in the image plane was used to simultaneously measure blood flow in the major circumpapillary retinal arteries and veins, and below the surface of the optic nerve head. Blood flow was determined in each eye of 19 patients with unilateral optic neuropathy (ischemic=13, compressive=5, inflammatory=1) in the chronic state. Inner retinal blood flow and optic nerve head blood flow were compared between affected and unaffected eyes. Retinal nerve fiber layer (RNFL) and retinal ganglion cell layer complex (GCL) were compared to retinal and optic nerve blood flow.

**Results:** There was a significant decrease in inner retinal blood flow in eyes with optic neuropathy compared to the fellow normal eye ( $P=0.002$ ; mean= $69\pm 17\%$  of fellow eye). A significant decrease in optic nerve head blood flow deep to the superficial retinal capillaries was also present ( $P<0.001$ ; mean= $65\pm 22\%$  of fellow eye). There was also significant decrease in both RNFL ( $P=0.02$ ; mean= $83\pm 32\%$  of fellow eye) and GCL thickness ( $P<0.001$ ; mean= $71\pm 11\%$  of fellow eye), but no significant correlation between the inter-ocular asymmetry of retinal or optic nerve blood flow and RNFL or GCL thickness.

**Conclusion:** Chronic optic neuropathy results in decreased retinal and optic nerve head blood flow, likely due to reduced metabolic demand. Laser speckle blood flowgraphy allows non-invasive simultaneous measurement of retinal, choroidal, and optic nerve head blood flow.

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Randy Kardon, M.D., Ph.D., Grants:

Funding (grants) from NEI R009040554; R01 EY018853

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Novartis steering committee OCTiMS

Co-Founder of Med-Face

Michael Wall, M.D.

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## RESIDENT / FELLOW RESEARCH DAY – 2015

Afternoon Session – Paper 18

### Elevated Intraocular Pressure after Intravitreal Dexamethasone (Ozurdex) Sustained-Release Implant

Eric K. Chin, M.D.

**Supervisor:** Vinit B. Mahajan, M.D. Ph.D.

**Background/Purpose:** To evaluate the likelihood of elevated intraocular pressure (IOP) following intravitreal dexamethasone (Ozurdex) biodegradable implantation. The incidence is compared between those with and without a glaucoma history, and across those who had one or more sequential Ozurdex implants across various vitreoretinal disease.

**Design:** Retrospective, observational, consecutive clinical case series.

**Participants:** Any patient who received Ozurdex injection with at least one month of follow-up: a total of 105 Ozurdex injections across 58 patients were administered.

**Methods:** Retrospective review of medical records of all patients receiving Ozurdex injection(s) between October 2009 and April 2014 was performed. Any patients with less than one month of follow-up were excluded. Data was reviewed from the time of Ozurdex injection to the time of subsequent injection or surgery.

**Main Outcome Measures:** Intraocular pressure (IOP) following Ozurdex injection, number of sequential Ozurdex injections, and diagnosis/indication for Ozurdex injection. The frequency of IOP elevation  $\geq 10$  mmHg from baseline IOP,  $\geq 30$  mmHg at any visit, number of patients started on IOP lowering drops, and number of patients requiring invasive glaucoma surgery were evaluated.

**Results:** Ninety-four injections from 52 patients (60 eyes) met our inclusion criterion. Eight patients had bilateral disease. Forty eyes had one, 9 eyes had two, 3 eyes had three, 3 eyes had four, and 3 eyes had five Ozurdex injections. The greatest IOP elevation occurred during the 1.5 to 2.5 month follow-up interval. Thirteen patients (25%; 14 eyes) had a known history of glaucoma risk or glaucoma. There were 14 instances (14.9%; 13 patients) with an IOP elevation of 10 mmHg or greater from baseline, of which five patients (38.5%) had glaucoma or glaucoma risk ( $P = 0.5443$ ). There were 10 instances (10.6%; 7 patients) of an IOP elevation to 30 mmHg or greater, of which three patients (42.9%) had glaucoma or glaucoma risk ( $P = 0.0696$ ). Glaucoma eye drops were initiated following 13 injections (13.8%; 11 patients), and only two patients (18.2%) had glaucoma or glaucoma risk. Invasive glaucoma surgery was required following 3 injections (3.2%; 3 patients), of which all three patients had a history of glaucoma or glaucoma risk.

**Conclusion:** A majority of patients who receive Ozurdex do not result in an IOP elevation. Among those who have an IOP increase, most do not have a history of glaucoma or glaucoma risk and responded to topical therapy alone; however, this history was present in all of the patients who ultimately required invasive glaucoma surgery following Ozurdex injection.

**RESIDENTS AND FELLOWS THAT HAVE COMPLETED RESEARCH  
PROJECTS AND SUBMITTED THEM FOR PUBLICATION IN PEER-  
REVIEWED JOURNALS**

**Residents:**

Blake Perry

Repair of 50-75% full-thickness lower eyelid defects: lateral stabilization as a guiding principle

Orbit (manuscript submitted)

Jesse M. Vislisel

Graft Survival of Diabetic Versus Non-Diabetic Donor Tissue after Initial Keratoplasty

Cornea. 2015 Apr;34(4):370-4.

Bradley Sacher

Treatment of Acanthamoeba keratitis with intravenous pentamidine prior to therapeutic keratoplasty.

Cornea. 2015 Jan;34(1)

Jeffrey Welder

Prophylactic Povidone-Iodine and Amphotericin B After Boston Keratoprsthesis. British Journal of Ophthalmology (manuscript submitted)

Jonathan L. Hager

Boston type 1 keratoprosthesis for failed keratoplasty

Cornea (manuscript in press)

**Fellows:**

Meredith A. Baker

The Quantitated Internal Suture Browpexy: Comparison of Two Brow-Lifting Techniques in Patients Undergoing Upper Blepharoplasty.

Ophthal Plast Reconstr Surg. 2015 Apr 7. [Epub ahead of print]