STATE BOARD OF OPTOMETRY
2450 DEL PASO ROAD, SUITE 105, SACRAMENTO, CA 95834
P (916) 575-7170 F (916) 575-7292 www.optometry .ca.gov

Continuing Education Course Approval Checklist

Title: Optic Nerve Cupping

Provider Name: USC Roski Eye Institute c/o Ms. Marithel Tesoro

☑Completed Application
Open to all Optometrists? ☑Yes ☐No
Maintain Record Agreement? ☑Yes ☐No

☑Correct Application Fee

☑Detailed Course Summary

☑Detailed Course Outline

☑PowerPoint and/or other Presentation Materials

☑Advertising (optional)

☑CV for EACH Course Instructor

☑License Verification for Each Course Instructor
Disciplinary History? ☐Yes ☑No

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CONTINUING EDUCATION COURSE APPROVAL APPLICATION

$50 Mandatory Fee

Pursuant to California Code of Regulations (CCR) § 1536, the Board will approve continuing education (CE) courses after receiving the applicable fee, the requested information below and it has been determined that the course meets criteria specified in CCR § 1536(g).

In addition to the information requested below, please attach a copy of the course schedule, a detailed course outline and presentation materials (e.g., PowerPoint presentation). Applications must be submitted 45 days prior to the course presentation date.

Please type or print clearly.

Course Title	**Course Presentation Date**
Optic Nerve Cupping	02/13/2017

Course Provider Contact Information

Provider Name

Lina (First) Poyzner (Last) _____ (Middle)

Provider Mailing Address

Street 1450 San Pablo St **City** Los Angeles **State** CA **Zip** 90033

Provider Email Address lina.poyzner@med.usc.edu

Will the proposed course be open to all California licensed optometrists?	☑ YES ☐ NO
Do you agree to maintain and furnish to the Board and/or attending licensee such records of course content and attendance as the Board requires, for a period of at least three years from the date of course presentation?	☑ YES ☐ NO

Course Instructor Information

Please provide the information below and attach the curriculum vitae for each instructor or lecturer involved in the course. If there are more instructors in the course, please provide the requested information on a separate sheet of paper.

Instructor Name

Alena (First) Reznik (Last) _____ (Middle)

License Number 113775	**License Type** MD
Phone Number (323) 442-6383	**Email Address** lina.poyzner@med.usc.edu

I declare under penalty of perjury under the laws of the State of California that all the information submitted on this form and on any accompanying attachments submitted is true and correct.

Poyzner 02/01/2017

Signature of Course Provider ***Date***

Optic Nerve Cupping

Alena Reznik, MD

Summary

The goal of the lecture is to describe the signs and features of optic nerve cupping and provide differential diagnosis. I will review definitions of glaucoma (primary open angle, closed angle and inflammatory), optic nerve atrophy due to compression, due to toxic optic neuropathy and pre-natal injury. Rare syndromes will be described such as morning glory and hypoplasia of optic nerves. Diagnostic criteria and approach will be outlines for each possible condition (difference in physical exam, visual field and OCT). Images will be reviewed with case presentation for each condition. Special attention will be paid to the need for central nervous system imaging (CT scan vs MRI) and pediatric patient population. I will conclude with an outline of clinical criteria for glaucomatous optic neuropathy vs optic neuropathy of other etiologies.

Optometry CME outline (Alena Reznik MD) – 1 hour lecture

February 13, 2017 6pm-7pm

Optic Nerve Cupping

1. Optic nerve cupping- definition. Adults vs pediatric patients.
2. Non-glaucomatous optic nerve cupping- definition.
3. Cup to disk ratio in normal subjects (adult): review of data
4. Cup to disk ratio in normal subjects (pediatric): review of data
5. Review of etiologies of cupping in absence of high IOP: physiologic, normal tension glaucoma, congenital abnormalities, prematurity, hereditary optic neuropathy, optic nerve compression, toxic and nutritional optic neuropathies, ischemic optic neuropathies.
6. Physiologic cupping- definition, examples.
7. Normal tension glaucoma- definition, examples
8. Congenital abnormalities- definition, examples
9. Prematurity- definition, examples
10. Hereditary optic neuropathy- definition, examples
11. Optic nerve compression - definition, examples
12. Toxic and nutritional optic neuropathies- definition, examples
13. Ischemic optic neuropathies- NAION vs pre-operative, definition, examples
14. Conclusion: how to differentiate and work up non-glaucomatous cupping.

Optic Nerve Cupping

Alena Reznik, MD

Assistant Professor of Ophthalmology

Glaucoma Service

Cell 310-980-6038

alenarez@med.usc.edu

Children's Hospital LOS ANGELES
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LAC+USC HEALTHCARE NETWORK

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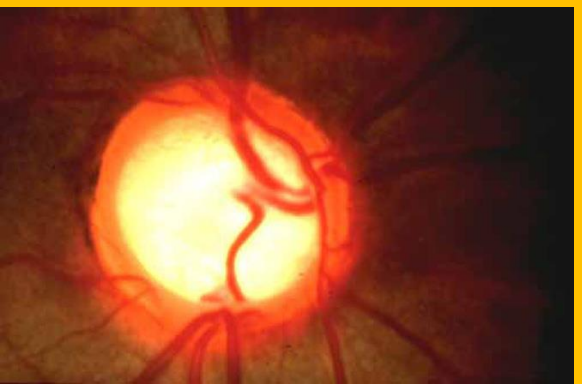
Optic Nerve Cupping

- R/o glaucoma
- Special population: children with large C:D
    - IOP measurements
    - Ability to perform VF tests
- Childhood glaucoma- rare

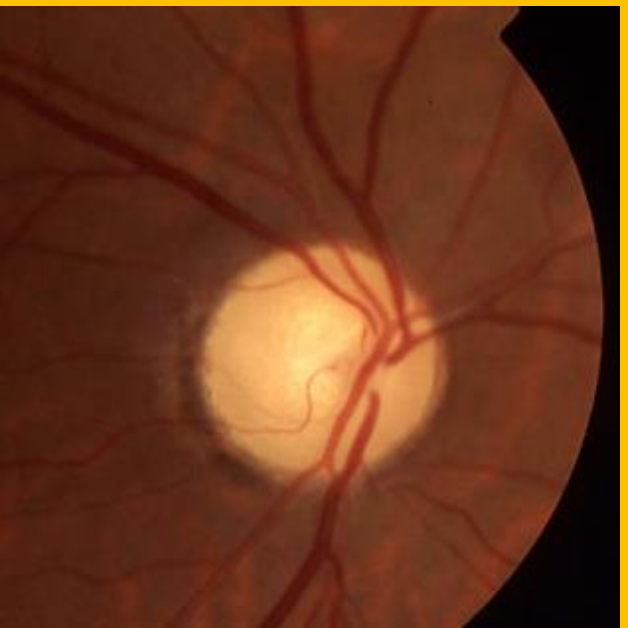
What is Optic Nerve Cupping?

- Due to nerve fiber loss
- Cupping is a sign of irreversible damage in adults
- Cupping is also seen in infantile and childhood forms of glaucoma and may be reversible



What is Non-Glaucomatous Optic Nerve Cupping?

- Increased C:D from another disease
- Increased C:D is harder to notice in a smaller disk and is often harder to see in children



Vitamin B12 deficiency

C:D in Normal Subjects

- Adults
  - Median C:D ratio is 0.25-0.3
  - Normal amount of C:D asymmetry is <0.2
- Children
  - Mean C:D ratio
    - 0.22±0.13 (Sydney Childhood Eye Study: 6 y.o., OCT)
      - By race: European White 0.2, East Asian 0.3, Middle Eastern 0.2
      - Absent cup present only in European White subjects
    - 0.2 (Duke Study: OCT)
      - By race: Af Am 0.23, Caucasian 0.16

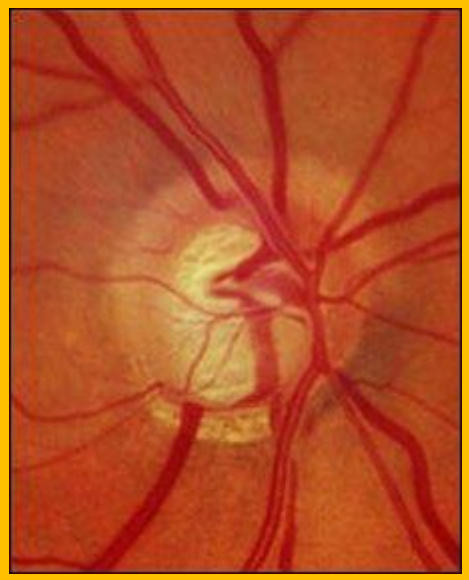
What is a normal optic nerve in children?

- C:D ratio increases over time in children
- Mean C:D:
  - <0.3 in infants <1 year old
  - 0.37 in 3 year olds
  - 0.42 in 17 year olds[1]

[1]El-Dairi et al. OCT in the eyes of normal children. Arch Ophthalmol 2009.

Glaucomatous Cupping

- Loss of neuroretinal rim area
- Increase in the absolute size of the cup
- Vertical elongation of the cup
- Excavation of the lamina cribosa
- Peripapillary atrophy
- Asymmetry
- Disc hemorrhage



Cupping in the Absence of High IOP

- Physiologic
- Normal tension glaucoma (NTG)
- Congenital anomalies
  - Coloboma, pit, hypoplasia, tilting, morning glory
- Hereditary optic neuropathies
  - Leber's, dominant optic atrophy
- Optic Nerve Compression
- Toxic and nutritional optic neuropathies
  - B12, methanol, medications
- Ischemic optic neuropathies
  - NAION, shock, peri-operative
- Prematurity/Low Birth Weight

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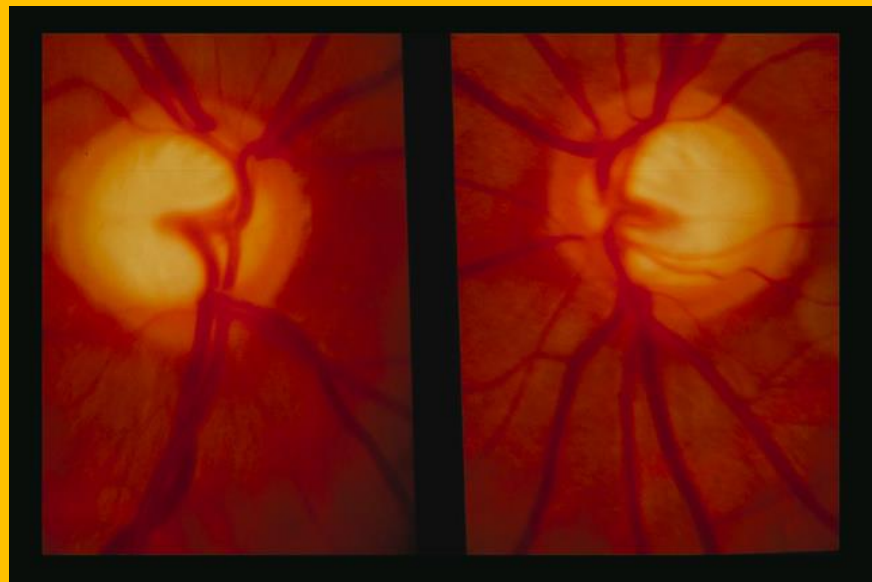
Physiologic Cupping

- Congenital variant
- Symmetric, NON-PROGRESSIVE
- Large cups
- No visual effects



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Congenital Anomalies

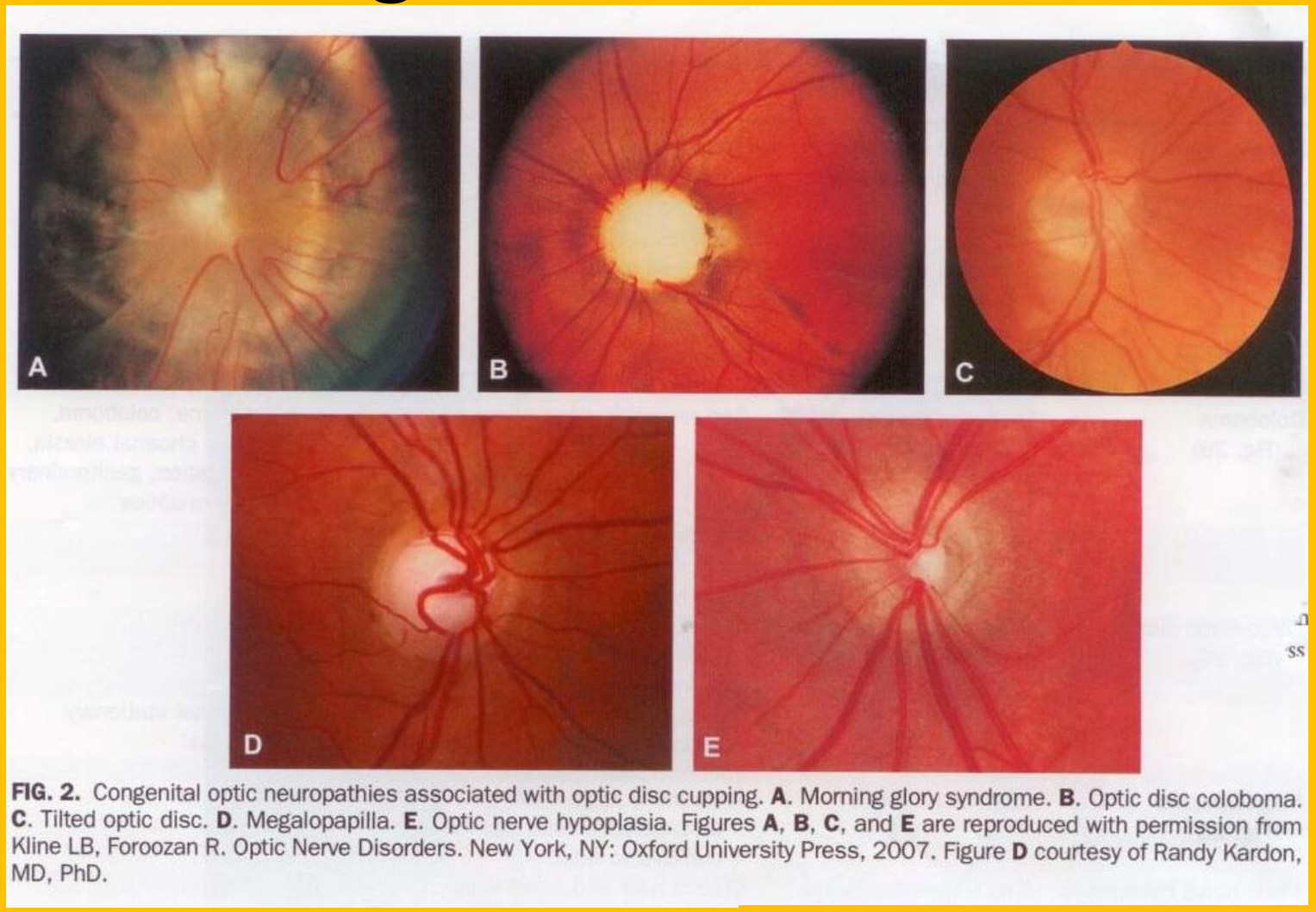

FIG. 2. Congenital optic neuropathies associated with optic disc cupping. **A.** Morning glory syndrome. **B.** Optic disc coloboma. **C.** Tilted optic disc. **D.** Megalopapilla. **E.** Optic nerve hypoplasia. Figures **A**, **B**, **C**, and **E** are reproduced with permission from Kline LB, Foroozan R. Optic Nerve Disorders. New York, NY: Oxford University Press, 2007. Figure **D** courtesy of Randy Kardon, MD, PhD.

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Coloboma

- Irregular inheritance, variable expressivity and penetrance
- Abnormality in the distal extremity of the embryonic fissure
- Progressive cupping has been described in autosomal-dominant coloboma[1]
- Association with CHARGE syndrome
  - Coloboma, heart, choanal atresia, mental retardation, GU, ear

[1]Moore et al. Progressive optic nerve cupping and neural rim decrease in a patient with bilateral autosomal dominant optic nerve colobomas. AJO 2000.

Morning Glory Disk

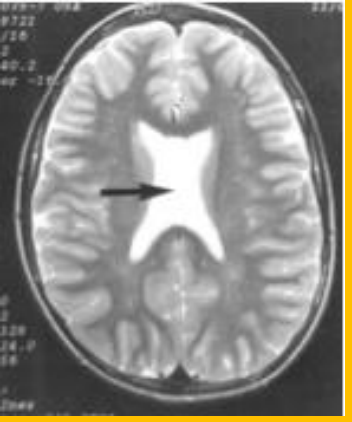
- Funnel-shaped excavation of disc,

With disc enlargement, pigmentary changes, retinal vessel radial distribution, glial tuft

- Vision usually sub-normal
- Associations:
  - Moyamoya disease (progressive bilateral stensosis of distal ICA)
  - Transsphenoidal basal encephalocele (chiasm, hypothalamus, pituitary, ACA herniation via anterior skull base) – pulsatile nasal mass

Optic Nerve Pits

- Congenital, frequency 1:11K
- Temporal location, 15% bilateral
- ~50% have visual field defects
- Serous macular detachments possible: 25-75%
- No CNS malformations



Optic Nerve Hypoplasia

- Small, surrounded by a yellow halo (double ring sign)
- May be associated with cupping
  - Loss of (or lack of formation) of nerve fiber tissue after scleral canal has formed results in more cupping than decrease in overall size
- Associated with midline or hemispheric brain defects (De Morsier Syndrome: ON hypoplasia, pituitary abnormalities, absent septum pellucidum)

Tilted Discs

- May appear cupped
- Displacement of the optic disc peripherally
- Oblique vessel insertions

- Associations
  - High myopia
  - Amblyopia
  - VF defects

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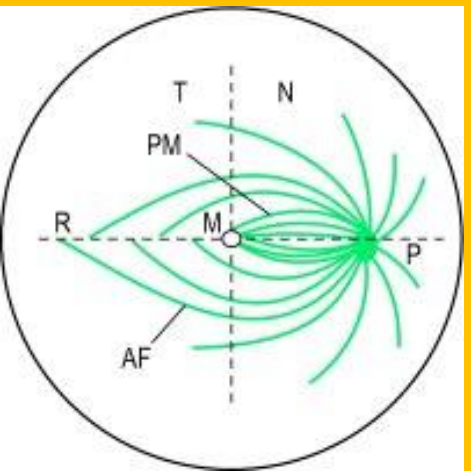
Nutritional, Toxic and Genetic Causes of Optic Neuropathies

- Most etiologies in these categories are related to mitochondrial dysfunction
- Papillomacular bundle is the most metabolically active, and therefore often affected first
  - Cupping and/or temporal pallor



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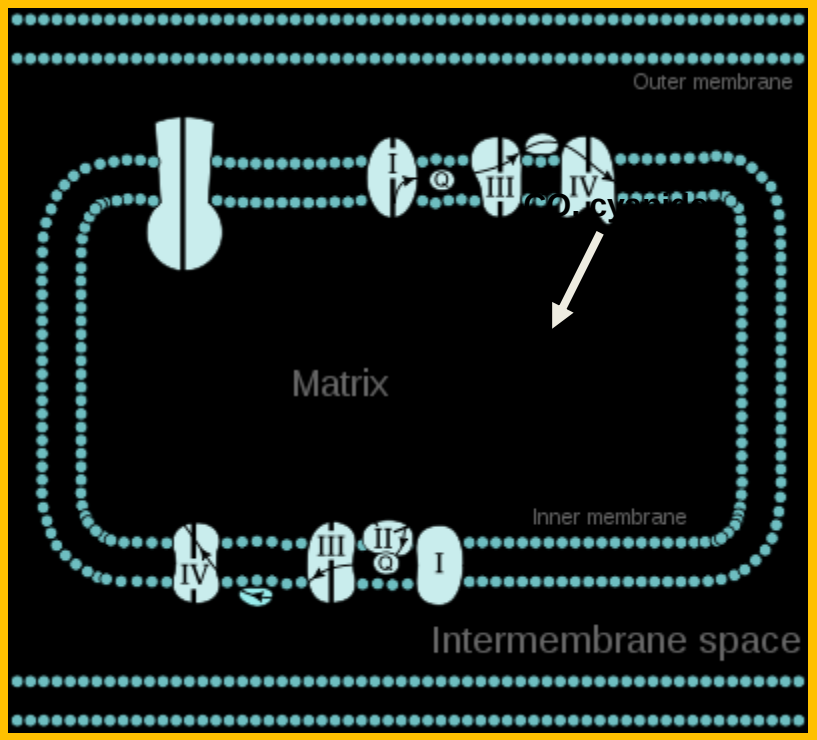
Mitochondrial Optic Neuropathies

Nutritional, Toxic, Genetic

- Pathogenesis not completely understood
- Final common pathway – disruption of mitochondrial oxidative phosphorylation, increased oxidative stress



Copper, ethambutol

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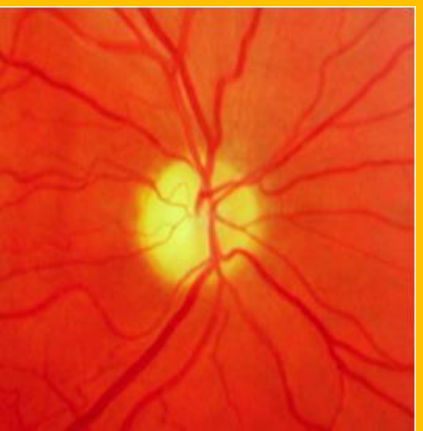
Differentiating Mitochondrial ON from Glaucoma

- Visual acuity and color vision
  - Late loss in glaucoma, early loss in ON
- Optic disk
  - Focal loss of rim in glaucoma
    - Also in glaucoma: normal rim color, vertical elongation, splinter hemorrhage
  - Rim pallor in ON (pallor>cupping)
- Visual field
  - Arcuate, nasal step, generalied depresson in glaucoma
  - Central loss in ON

Hereditary Optic Neuropathies

- Leber's hereditary optic neuropathy (LHON)
  - Mitochondrial inheritance
  - 80-90% males, 10-30 yrs old
  - Central vision loss, color loss
  - Initially disc elevation and peripapillary thickening and telangectasia
  - Eventual loss of unmyelinated axons (which have more mitochondria) leads to cupping



Hereditary Optic Neuropathies

- Dominant Optic Atrophy
  - OPA1 gene mutation
  - Insidious onset in first 2 decades
  - Mild to moderate symmetric central vision and color vision loss
  - Temporal disc pallor and wedge-shaped temporal excavation

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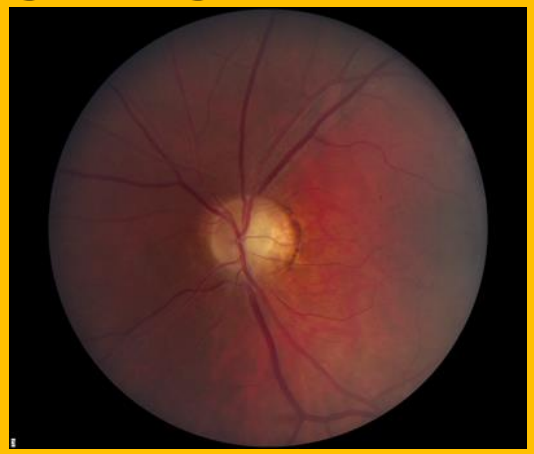
Nutritional Optic Neuropathies

- Slowly progressive optic nerve atrophy and/or cupping
- Central vision loss, color vision loss
- Co-existent neurologic and hematologic signs and symptoms
- GI disease or extreme diet



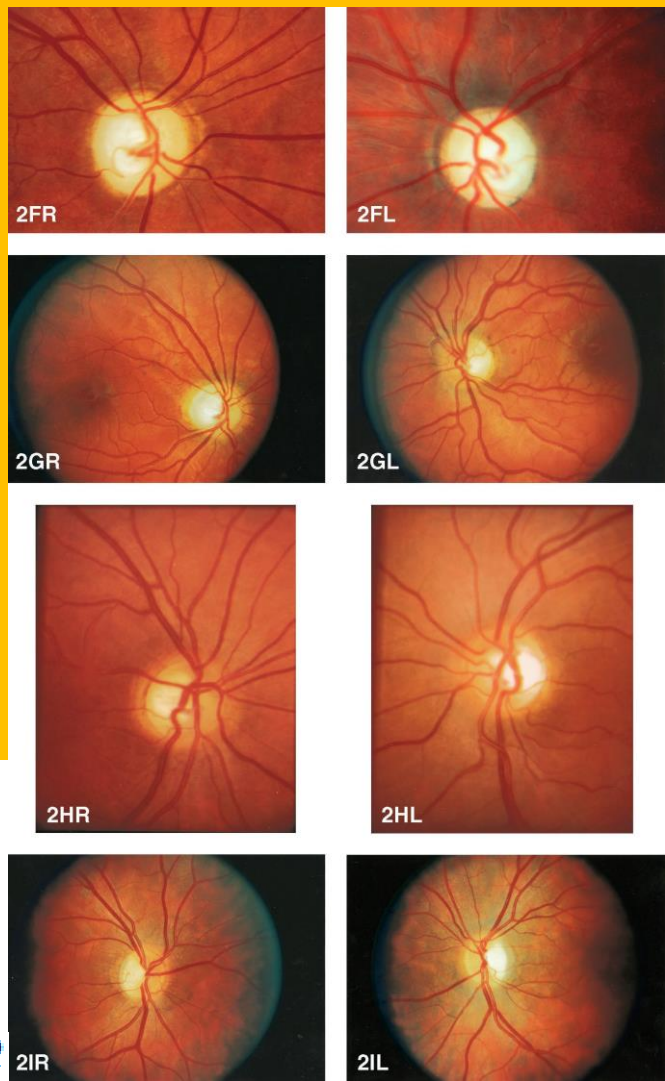
- Vitamin B12
  - Copper, Vitamin A, Folate

Toxic Optic Neuropathies

- Methanol toxicity
  - Disc and peripapillary retinal edema followed by atrophy and disc excavation within days to weeks
- Other toxins
  - Medications (ethambutol, chemotherapy)
  - Lead, heavy metals, arsenic
  - Cyanide
  - Ethylene glycol

Comparing Hereditary/Toxic/Nutritional ON and NTG

- Differentiation mainly by:
  - Family history
  - Central visual acuity
  - Color vision
  - RIM PALLOR
  - Early age of onset



Disc Excavation in Dominant Optic Atrophy

Differentiation from Normal Tension Glaucoma

Annick V. Fournier, MD, FRCS(C),[1] Karim F. Damji, MD, FRCS(C),[1] David I. Epstein, MD,[2] Stephen C. Pollock, MD[2]

Ophthalmology 2001;108:1595–1602

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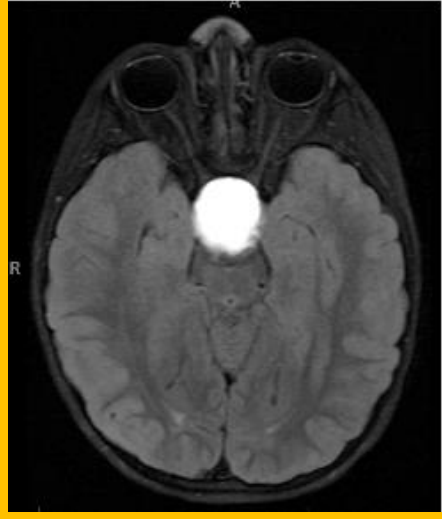
Compressive Lesions



- Suprasellar lesions
- Several case series in <u>adults</u> describing glaucoma-like cupping[1]
  - 50% pituitary adenoma
  - Less commonly pituitary apoplexy, meningioma, craniopharyngioma, other cystic lesions[2]

[1]Piette and Sergott. Pathological optic disc cupping. Curr Op Ophthalmol 2006.
[2]Greenfield et al. The cupped disc: Who needs neuroimaging? Ophthalmology 1998.

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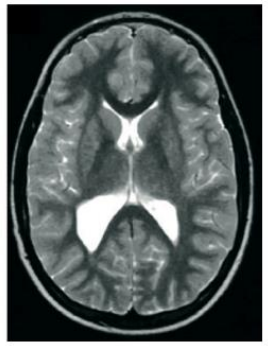
Cerebral Injury in Newborns

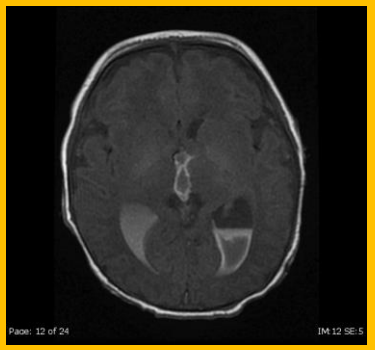
- Damage to the immature brain (<34 wks) primarily affects the periventricular region
- Birth weight <1750 gm and GA <30 weeks
- Periventricular leukomalacia (PVL)
  - Reduced BP and impaired autoregulation leads to decreased cerebral perfusion
  - Watershed zone exists at posterior horns (less commonly anteriorly) as immature blood supply converts to adult vascular supply
  - Corticospinal tract and optic radiations most commonly affected



Dilation of right ventricle

Cerebral Injury in Prematurity

- Intraventricular hemorrhage (IVH)
  - Preterm infants have limited ability to autoregulate cerebral blood flow; abrupt ⇧BP can results in development of IVH
  - 15% of children with IVH can have resultant parenchymal damage to the periventricular white matter including optic radiations



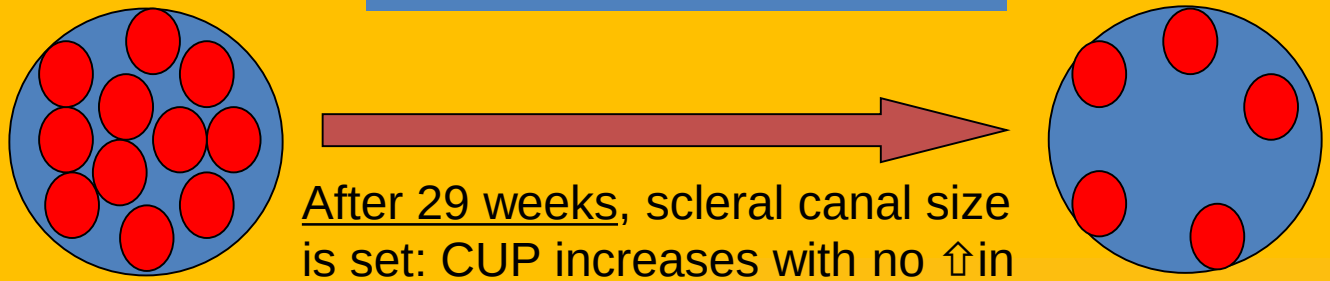
Perinatal Ischemic Damage

- Preterm babies with anoxic brain injury (ischemic or hemorrhagic)
- Axonal disruption in peri-ventricular optic radiations leads to transsynaptic retrograde degeneration across the lateral geniculate body
- Insult <29 weeks: hypoplasia, scleral plasticity will lead to a diffusely hypoplastic nerve
- Insult >30 weeks: cupping, scleral canal fully formed, rim diameter set

Timing of Ischemic Insult Plays a Role in Whether ON is Hypoplastic or Cupped

Before 29 weeks, scleral canal plasticity results in decreased ON diameter

Ischemic Insult Results in Degeneration of 50% of Optic Nerve Fibers



After 29 weeks, scleral canal size is set: CUP increases with no ⇧in disk diameter

EXTENDED REPORT

Optic disc morphology may reveal timing of insult in children with periventricular leucomalacia and/or periventricular haemorrhage

L Jacobson, A-L Hård, E Svensson, O Flodmark, A Hellström

Br J Ophthalmol 2003;**87**:1345–1349

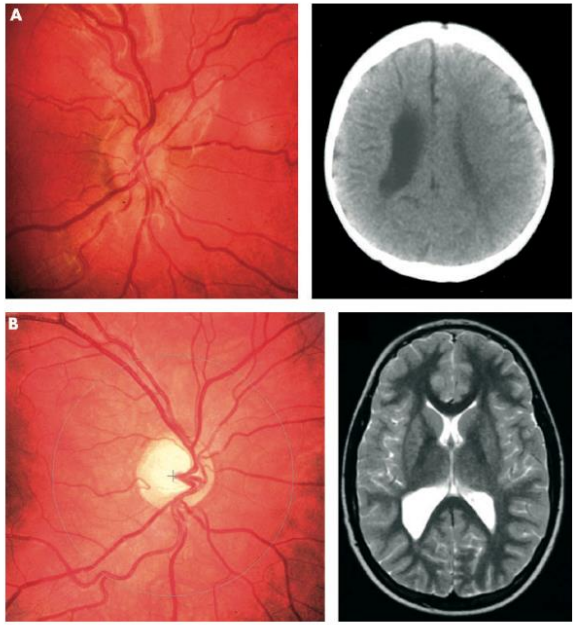


Figure 1 (A) Fundus photograph and CT of a 5 year old girl with gestational age at birth of 34 weeks, esotropia, and visual acuity 20/100. The optic disc has a small area. The CT scan shows extensive loss of periventricular white matter including almost all white matter in the right cerebral hemisphere. This image represents the end stage following a periventricular haemorrhagic infarction, indicating an early lesion. (B) Fundus photograph and MRI of a 10 year old boy with gestational age 31 weeks at birth and perinatal asphyxia. He is ortophoric, with visual acuity right eye 20/20, 20/30 left eye, and small bilateral defects in the inferior fields, normal intraocular pressure. The optic disc has a large cup in a normal sized optic disc. The T2 weighted MR scan demonstrates focal dilatation of the occipital horns, more pronounced in the right cerebral hemisphere, reflecting loss of peritrigonal white matter. White matter is preserved anteriorly as well as in centrum semiovale (not shown), indicating a late lesion.

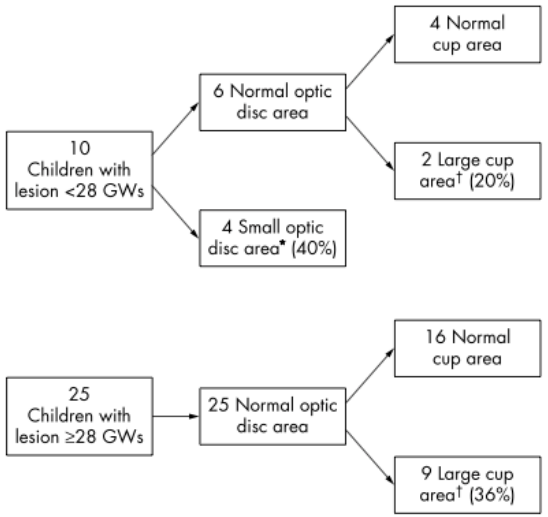


Figure 3 Schematic illustration of number of children and their optic disc morphology in relation to estimated timing of brain lesion. *Below the 2.5th centile of the reference group. †Above the 97.5th centile of the reference group.

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Prematurity and Low Birth Weight

In the absence of cerebral damage from PVL or IVH

- Sydney Childhood Eye Study
  - Low birth weight (<2.5 kg) associated with increased C:D at 12 years of age
    - Adjusted for age, gender, ethnicity, height, AL, BMI
  - Smaller length and head circumference similarly associated with larger C:D
  - Relationships persisted when premature babies <33 wks removed from analysis
- Authors speculate that intrauterine influences result in interruption of the maturation of ganglion cells

Samarawickrama, et al. Birth weight and optic nerve head parameters. Ophthalmology 2009.

Now that we know the other causes of optic nerve cupping, how can we distinguish them from glaucoma??

Glaucoma	Non glaucomatous cupping
Lack of Rim Pallor	Pallor>Cupping (94% specific)[1]
Excavation of cup	Focal excavation (temporally)
Rim notching or obliteration (87% specific)[1]	
Peripapillary atrophy	Color vision loss
Disc hemorrhages (specific but not sensitive)	rAPD
Spares papillomacular bundle; thinnest rim is inferior	Involves papillomacular bundle; thinnest rim is usually temporal

[1]Trobe et al. Nonglaucomatous excavation of the optic disc. Arch Ophthalmol 1980.

What's so important about the rim?

- Rim pallor warrants a search for an alternative etiology other than glaucoma in any patient with ON cupping, especially in children
- Rim color is usually normal in glaucoma
- Rim color is usually pale in non-glaucomatous cupping

Management

- History
  - Birth History crucial
  - Diet, GI system, other exposures, medications
  - Family History
- Exam
  - Rim pallor
  - Evaluate for signs of glaucoma (vertical notching, disk hemorrhages)
  - Evaluate for signs of non-glaucoma (color vision loss, rAPD, decreased visual acuity, spared peripheral fields)

Management

- Further work-up based on history and physical examination
- Consider
  - Blood work
    - Vitamin B12, Vitamin A, Folate
    - Lead, heavy metals, arsenic
    - LHON and Dominant OA genetic testing
  - Neuroimaging
    - MRI brain with and without IV contrast
      - Evaluate for perinatal damage
      - Evaluate for compressive mass
      - Signs of ON hypoplasia

Thank you!!!

- Questions?
- Cell 310-980-6038
- alenarez@med.usc.edu

Pasadena/Downtown/Arcadia/Beverly Hills

USC Eye Institute

RetCam image analysis of optic disc morphology in premature infants and its relation to ischaemic brain injury

E McLoone, M O'Keefe, V Donoghue, S McLoone, N Horgan, B Lanigan

Br J Ophthalmol 2006;**90**:465–471. doi: 10.1136/bjo.2005.078519

Table 2 Optic disc parameters in premature infants with and without periventricular white matter (PVWM) damage

Variable (median values)	No PVWM damage (n=80)	PVWM damage (n=24)	p Value
Gestational age (weeks)	28.0	27.0	0.20
Body weight (g)	1050.0	1008.0	0.68
Optic disc diameter (mm)	1.05	0.95	0.002
Optic disc area (mm^2)	1.13	1.06	0.03
Optic cup area (mm^2)	0.09	0.08	0.07
Optic rim area (mm^2)	1.03	0.95	0.02

Inc C:D ratio

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Cortical Visual Impairment and Optic Nerve Cupping

Changes in the Optic Disc Excavation of Children Affected by Cerebral Visual Impairment: A Tomographic Analysis

Giulio Ruberto,[1] Roberto Salati,[2] Giovanni Milano,[1] Chiara Bertone,[1] Carmine Tinelli,[3] Elisa Fazzi,[4] Rosanna Guagliano,[1] Sabrina Signorini,[3] Renato Borgatti,[2] Alessandro Bianchi,[1] and Paolo Emilio Bianchi[1]

(*Invest Ophthalmol Vis Sci.* 2006;47:484–488)

TABLE 1. Optic Disc Parameters and Statistical Significance in CVI-Affected Subjects Compared with Healthy Controls

	CVI Subjects (n = 24)				Control Subjects (n = 88)				P	
	Mean		SD		Mean		SD			
	OD	OS	OD	OS	OD	OS	OD	OS	OD	OS
Disc area	2.05	1.97	0.48	0.38	2.48	2.42	0.46	0.50	0.000075	0.000163
Cup area	0.64	0.60	0.48	0.42	0.41	0.41	0.35	0.38	0.016224	0.005800
Rim area	1.40	1.30	0.49	0.41	2.03	2.01	0.47	0.44	0.000001	0.000000
Cup volume	0.23	0.13	0.50	0.12	0.09	0.10	0.13	0.15	0.045874	0.080435
Rim volume	0.42	0.29	0.28	0.18	0.52	0.52	0.23	0.32	0.032521	0.000140
Cup/disc ratio	0.30	0.33	0.21	0.19	0.16	0.16	0.11	0.12	0.001874	0.000147
Mean cup depth	0.25	0.17	0.26	0.09	0.16	0.17	0.10	0.12	0.009064	0.413672
Maximum cup depth	0.69	0.48	0.66	0.22	0.50	0.53	0.30	0.36	0.081090	0.962876
Cup shape measure	−0.15	−0.16	0.11	0.08	−0.22	−0.23	0.10	0.08	0.001681	0.000711
Mean RNFL thickness	0.15	0.10	0.16	0.11	0.21	0.24	0.08	0.13	0.003430	0.000022

Periventricular White Matter Damage and the Optic Disc

- PVL and Ischemia from IVH:
  - Most commonly involves optic radiations passing adjacent to lateral ventricles
  - Associated in several studies with smaller ON area, larger cup area, smaller rim area, increased C:D
  - Most significant in babies with grade 4 IVH
  - Periventricular white matter damage from grade 4 IVH or PVL can result in retrograde trans-synaptic damage
    - Before 28 weeks: small nerve (scleral canal plasticity)
    - After 29 weeks: cupped nerve (scleral canal size set)

Vision is our Mission—Preserve, Protect, Restore

USC Roski Eye Institute

Keck Medicine of USC

Nationally top ranked ophthalmology program — 22 consecutive years and counting!

J. Bradley Randleman, MD

Professor of Ophthalmology, Director, Cornea, External Disease, and Refractive Surgery Service

J. Bradley Randleman, MD is one of the nation's top corneal researchers and surgeons and an expert on everything from LASIK to the latest FDA approved treatments for corneal cross-linking surgery.

Alena Reznik, MD

Assistant Professor of Clinical Ophthalmology

Dr. Reznik specializes in early detection and treatment of glaucoma and cataracts as well as novel surgical techniques for advanced cases. Her research interests are minimally invasive glaucoma surgery and new approaches to eye emergencies. She is a principal investigator on clinical trials for glaucoma medications and surgical devices.

Damien C. Rodger, MD, PhD

Assistant Professor of Clinical Ophthalmology

Dr. Rodger's clinical interests include diabetic retinopathy, macular degeneration, medical retina, retinal detachment, uveitis and vitreoretinal surgery.He has conducted research on the design, fabrication, and testing of high-density microtechnologies for retinal and spinal cord prostheses, and has been instrumental in the development of other novel bioMEMS.

PLEASE JOIN US FOR

AN EDUCATIONAL EVENING WITH FRIENDS & NEIGHBORS

Private 2 Hour CME in Beverly Hills

Date: Monday, Februay 13, 2017
Time: 7:00pm
Location:
Maggiano's Little Italy at The Grove
189 The Grove Dr. Suite Z80
Los Angeles, CA 9036
RSVP: Lina Poyzner at
lina.poyzner@med.usc.edu

Program:

Glaucoma (Optic Nerve Cupping), presented by Dr. Reznik — 1 Hour

Retina (OCT Reading and OCT Enigmas), presented by Dr. Rodger — 30 min.

Cornea (Cross Linking), presented by Dr. Randleman — 30 min.

USC Roski Eye Institute • 323-442-6335 • www.usceye.org • Clinics conveniently located at:

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1450 San Pablo Street, 4th Floor
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Pasadena, CA 91105
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Arcadia Clinic
USC Roski Eye Institute
65 N. First Avenue, Suite 101
Arcadia, CA 91006
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CURRICULUM VITAE
ALENA REZNIK,MD
SEPTEMBER 19, 2016

PERSONAL INFORMATION:

Work

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EDUCATION AND PROFESSIONAL APPOINTMENTS

EDUCATION:

2005 *BS, Summa Cum Laude,Microbiology/Immunology/Molecular Genetics, UCLA, LA*
2009 *MD, Johns Hopkins University School Of Medicine, Baltimore*

POST-GRADUATE TRAINING:

2009-2010 *Internship in Internal Medicine, UC Davis Medical Center, Sacramento*

2010-2013 *Ophthalmology Residency, UC Davis Medical Center, Sacramento*

2013-2014 *Glaucoma Fellowship, Jules Stein Eye Institute, UCLA, LA*

HONORS, AWARDS:

2016	*Top Doctor 2016*	*Pasadena Magazine*
2016	*Rising Star 2016*	*Top Doctors*
2012	*ARVO Travel Grant*	*ARVO, Ft.Lauderdale, FL*
2007	*AFAR Research Fellow*	*Johns Hopkins Hospital, Baltimore, MD*
2008-2009	*The Roothbert Fund Fellow*	*Johns Hopkins Hosptal, Baltimore, MD*
2008-2009	*Marilyn and Marshall Butler Scholarship*	*Johns Hopkins Hospital, Baltimore, MD*
2007-2008	*William W.More, Ph.D. Memori¿ Scholarship[*	*Johns Hopkins Hospital, Baltimore, MD*
2007-2009	*HIAS Scholarship*	*NY*
2007-2009	*Ruth G White PEO Scholarship*	*Johns Hopkins Hospital, Baltimore, MD*
2007-2009	*Ethel O. Gardner PEOP Scholarship*	*Johns Hopkins Hospital, Baltimore, MD*
2007	*Travel Grant AGS*	*WA*

ACADEMIC APPOINTMENTS:

2014-current	*Assistant Professor of Ophthalmology*	*USC Eye Institute, USC, LA, CA*

TEACHING

DIDACTIC TEACHING:

Institution USC

2014	*Femtosecond Laser in Glaucoma Surgery*	*1Hr*	*Lecturer*
2015	*Novel Techniques in Glaucoma Surgery*	*2Hrs*	*Lecturer*
2015	*New Treatments in Advanced Glaucoma*	*2Hrs*	*Lecturer*
2015	*Glaucoma Curriculum (USC LAC Residency)*	*12Hrs*	*Lecturer*
10/2015	*Wet Lab "Minimally Invasive Glaucoma Surgery/Trabectome"*	*3Hrs*	*Lecturer*
11/2015	*Wet Lab "iStent and Angle Surgery"*	*3Hrs*	*Lecturer*
11/2015	*Wet Lab "Trabeculectomy"*	*2Hrs*	*Lecturer*
3/2016	*OKAP Review Lectures*	*4 Hrs*	*Lecturer*
3/2016	*Journal Club Glaucoma*	*2Hrs*	*Lecturer*

POSTGRADUATE MENTORSHIP:

2015-2016	*Benjamin Xu, MD, PhD*	*PGY4 2016*	*Career Guidance*
2014-2015	*Yohko Murakami, MD*	*PGY4 2015*	*Career Guidance*

SERVICE

DEPARTMENT SERVICE:

2014-current	*Complaince Committee*	*USC Eye Institute*
12/2015-current	*Residency Selection*	*USC Eye Institute*
07/2014-current	*Glaucoma Fellowship Selection*	*USC Eye Institute*
07/2014-current	*Postgraduate Education Committee*	*USC Eye Institute*

PROFESSIONAL SOCIETY MEMBERSHIPS:

2010-current	*American Academy of Ophthalmology*
2013-current	*American Glaucoma Society*
2010-current	*Women In Ophthalmology*
2010-current	*American Society of Cataract and Refractive Surgeons*

RESEARCH AND SCHOLARSHIP

EDITORSHIPS AND EDITORIAL BOARDS:

03/2015-current Editor Elsevier Editorial System

MAJOR AREAS OF RESEARCH INTEREST

Research Areas

1. Minimally Invassive Glaucoma Surgery
2. Novel Surgical Techniques in Glaucoma

PUBLICATIONS:

REFEREED JOURNAL ARTICLES:

Klimava, A, Akpek, E. Evaluation of Patients with Dry Eye Syndrome for Associated Medical Conditions. ARVO 2007. Lecture presentation, May 2007. Published in Cornea September 2010:29(9):1072.

Reznik J, Salz, J, **Klimava A**. Late Unilateral Corneal Ectasia After PRK With Preoperative Topography Suggestive of FFK. AAO Refractive Subspecialty Day, November 2006. Lecture presentation; Published in J Refract Surg. 2008 Oct;24(8):843-7.

Nagai N, **Klimava A**, Wen-Hsiang L, Handa J. CTGF is increased in Basal Deposits and Regulates Matrix Production through the ERK (p42/p44^{mapk}) MAPK and the p38 ^{mapk} signaling pathways. Published in Invest Ophthalmol Vis Sci. 2009 Apr;50(4):1903-10.

REFEREED JOURNAL ARTICLES IN PRESS:

Format: Authors, Title. *Journal.* Volume #(Suppl ##):Page-Page, Year. PMID#, PMCID#, *Narrative describing personal contribution.*

REFEREED REVIEWS, CHAPTERS, AND EDITORIALS:

Reznik, A, Varma, R. (12/2015). Ab-Interno Subconjunctival Glaucoma Implant for Advanced Open-Angle Glaucoma

CLINICAL COMMUNICATION: (CASE REPORTS, LETTERS)

Authors. Title. *Journal* Volume(Suppl ##):Page-Page, Year. PMID#, PMCID#

ON-LINE PUBLICATIONS:

Reznik A, Mukundum G, Sonu R, Lin L. Imaging in immunohistologically proven orbital tumors. Submitted for a publication in Radiographics, May 2012

BOOKS, MONOGRAPHS, AND TEXT BOOKS:

Authors. Title. *Publication* Volume(Suppl ##):Page-Page, Year.

LETTERS TO THE EDITOR:

Authors. Title. *Publication* Volume(Suppl ##):Page-Page, Year. PMID#, PMCID#

ABSTRACTS AND PRESENTATIONS:

Reznik A, Keltner J. Emergency department direct ophthalmoscopy and non-mydriatic funduscopic camera as a training tool. UC Davis research Symposium 2012. Lecture presentation.

Reznik A, Weber C, Telander D, Morse L, Thirkill C. Inflammatory reactions complicating exudative age-related macular degeneration. ARVO 2012. Poster presentation.

Akpek E, **Klimava A**, Thorne J, Martin D, Lekhanont K, Ostrovsky A. Evaluation of Dry Eye Patients for Presence of Underlying Sjogren's Syndrome. AAO 2007 meeting. Lecture presentation.

Gupta A, Sadeghi P, **Klimava A**, Akpek E. Occult thyroid eye disease in patients presenting with dry eye symptoms. Tear Film and Ocular Surface Society Annual Meeting, Taormina, Sicily, September 2007. Lecture presentation.

Reznik J, Salz, J, **Klimava A**. Late Unilateral Corneal Ectasia After PRK With Preoperative Topography Suggestive of FFK. AAO Refractive Subspecialty Day, November 2006. Lecture presentation; Published in J Refract Surg. 2008 Oct;24(8):843-7.

Klimava A, Handa J. Increased Connective Tissue Growth Factor in Basal Deposits of Bruch's Membrane of Human Maculae. American Geriatric Society Annual Meeting, May, 2007. Poster presentation.

Klimava A, Handa J. Connective Tissue Growth Factor Expression in ARMD. Johns Hopkins Summer Activities Symposium, October 2006. Poster presentation.

Reznik J, Kim A, **Klimava A**, Akpek E, Gatifloxacin 0.3% in treatment of bacterial keratitis; ARVO 2009. Poster presentation.