STATE BOARD OF OPTOMETRY
2450 DEL PASO ROAD, SUITE 105, SACRAMENTO, CA 95834
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Continuing Education Course Approval Checklist

Title: Is This Glaucoma?

Provider Name: UCSF c/o Dr. Jane Kuo

☑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

CALIFORNIA STATE BOARD OF OPTOMETRY

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CONTINUING EDUCATION COURSE APPROVAL APPLICATION

$50 Mandatory Fee

FEES PAID

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**
Is this glaucoma?	02/05/2017

Course Provider Contact Information

Provider Name

Andrew (First) Mick (Last) Boyd (Middle)

Provider Mailing Address

Street 4150 Clement St **City** San Francisco **State** CA **Zip** 94121

Provider Email Address andrew.mick@va.gov

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

Andrew (First) Mick (Last) Boyd (Middle)

License Number 11996TPLG	**License Type** Optometrist
Phone Number (415) 221-4810 x 4606	**Email Address** andrew.mick@va.gov

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.

Signature of Course Provider *Date* 12/29/2016

Title: Is This Glaucoma?

Presenter: Andrew Mick, OD FAAO

Summary:
The initial sign of a potential diagnosis of glaucoma is often enlarged nerve cupping. This sign is not specific and many of the confirmatory tests are also abnormal with compressive optic neuropathy. The lecture will review the patient profiles, systemic symptoms and ocular signs to help differentiate between glaucoma and compressive neuropathy.

Is this Glaucoma?

Andrew B Mick, OD, FAAO
Associate Clinical Professor
UC Berkeley School of Optometry
UCSF Department of Ophthalmology

I. Glaucomatous and compressive optic neuropathy: Why we can be fooled

A. Enlargement of the optic cup is nonspecific and found in more optic neuropathies than just glaucoma, specifically compressive optic neuropathy
B. Loss of nerve fiber layer, as seen clinically or by OCT imaging, also is nonspecific
C. Visual fields, used heavily in glaucoma diagnosis, are prone to fluctuation and unreliability
D. There is overlapping patient demographics for glaucoma and chiasmal compressive optic neuropathy
E. Glaucoma is diagnosed more commonly than compressive optic neuropathy
F. Non-secreting pituitary adenomas often have no overt systemic signs/symptoms
G. The cost and logistics of getting brain imaging leads to resistance in ordering

II. Brief anatomical review of the optic nerve and anterior visual pathway

A. The retinal ganglion cell axons and the optic nerve
    1. The primary neural tissue of the optic nerve is composed of the retinal ganglion cell axons
    2. The ganglion cell axons originating nasal to the optic nerve within the retina take a direct course to the optic nerve
    3. The ganglion cell axons originating temporal to the optic nerve within the retina do not all course directly to the optic nerve
    4. The ganglion cell axons originating in the central macula course directly to the temporal optic nerve (papillomacular bundle)
    5. Ganglion cell axons from other portions of the temporal retina must take an arcuate course around the papillomacular bundle and therefore enter the optic nerve at the superior and inferior poles
B. The optic nerve and the visual field
    1. The fovea sits in the center of the vertical meridian that separates the visual field into nasal and temporal hemifields
    2. The ganglion cell axons corresponding to the temporal field project to the ipsilateral optic tract
    3. The ganglion cell axons corresponding to the nasal retina cross over to the contralateral optic tract at the chiasm
    4. The retinotopic organization of the ganglion cell axons is generally preserved within the optic nerve, but as the nerve approaches the chiasm, the axons from the nasal retina begin to occupy more nasal portions of the optic nerve in preceding their chiasmal crossing
    5. After the chiasm, some of the crossed nasal fibers briefly loop into the opposite nerve before coursing back in the tract (Wilbrand's knee)

III. Demographic overlap between glaucoma and pituitary adenoma

A. Demographics of glaucoma
    1. Primary open angle glaucoma is more common in African American and Latino populations compared to Caucasians
    2. Low tension glaucoma more common in Asian (specifically Japanese) populations compared to Caucasians
    3. In all ethnic groups, glaucoma becomes more common with increasing age
A. Demographics of pituitary adenoma
    1. Incidence of adenomas is more common in women than men until age 30 then reverses into older age groups

2. Adenomas have been found in as high as ~15% of autopsy studies and 23% of radiographic studies
3. Adenomas have been found in up to 6-8% of patients diagnosed with low tension glaucoma
4. Pituitary adenomas are more common in African American compared to Asian and Caucasian populations
5. Pituitary adenomas become more common with increasing age with peak incidence when glaucoma is also most common

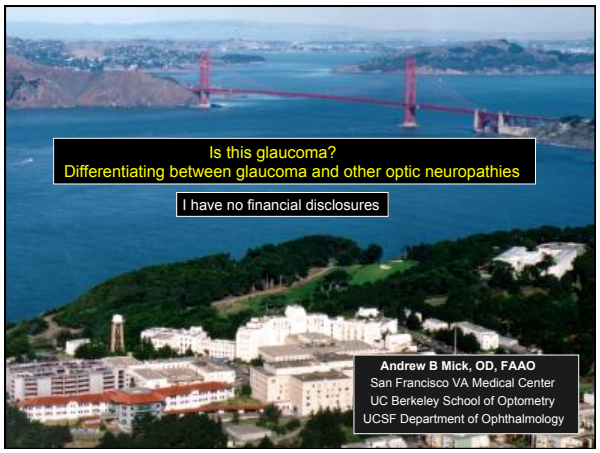
IV. Clinical feature overlap between glaucoma and compressive optic neuropathy from pituitary adenoma
- A. Seen in patients with with normal ranges of intraocular pressure
- B. Increased optic nerve cupping
- C. More pallor of the optic disc relative compared to a normal population
- D. Loss of retinal nerve fiber layer seen clinically or measured by ocular coherence tomography
- E. Visual field loss

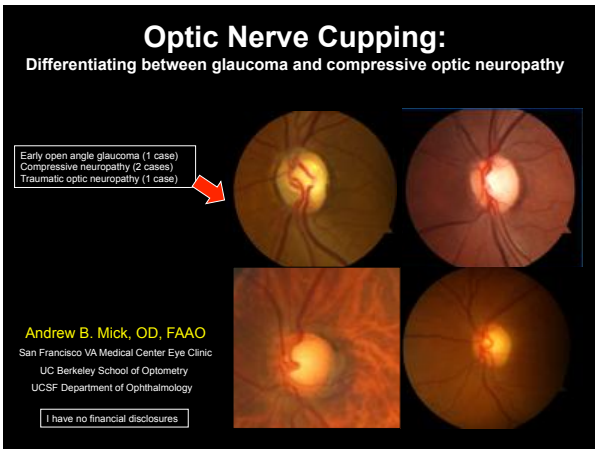
V. How we can better differentiate between glaucoma and compressive optic neuropathy and determine who should be scanned

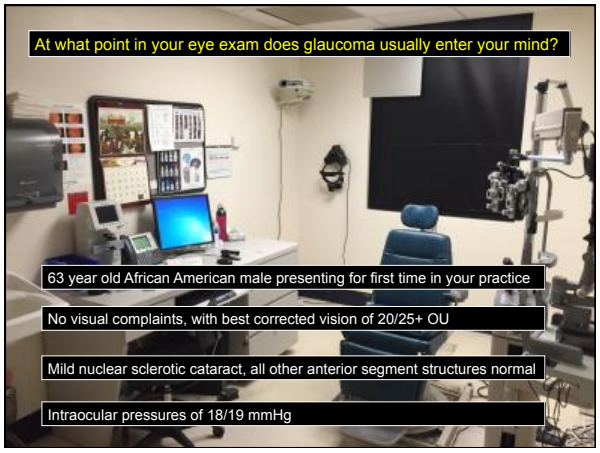
- A. More likely to be glaucoma
  1. Retention of good central vision until late in the disease
  2. Vertical enlargement of optic nerve cupping
  3. Sectoral loss of the neuroretinal rim especially at vertical poles
  3. Presence of progressive laminar remodeling
  4. Presence of disc hemorrhage
  5. Presence large areas and progressive peripapillary atrophy
  6. Visual fields relatively respecting of horizontal midline
- B. More likely to be chiasmal compressive optic neuropathy
  1. Occuring in young age groups (less than 40)
  1. Reduction in central visual acuity relatively early in the disease
  2. Color vision deficits early relatively early in the disease
  3. Optic nerve pallor clinically obvious and greater than the extent of cupping
  4. Pallor and preferential nerve fiber layer loss of the horizontal poles of the nerve
  5. Visual fields relatively respecting of vertical midline
  6. Presence of systemic signs consistent with pituitary dysfunction

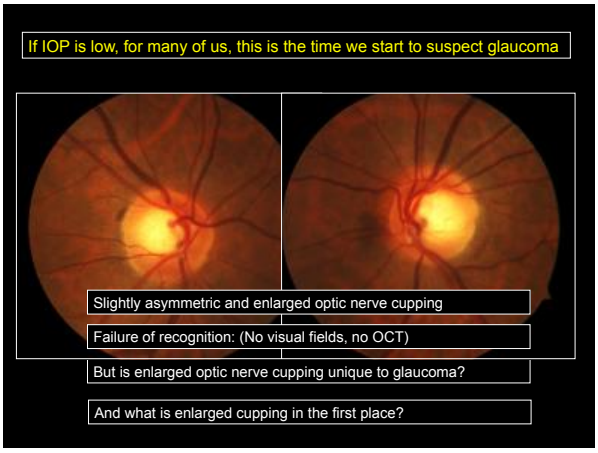
VI. Systemic symptoms/signs of pituitary dysfunction

- A. Non-secreting adenomas usually only produce signs/symptoms associated with mass effect
  1. Headache
  2. Visual dysfunction
  3. Seizures
- B. Secreting adenomas can produce symptoms specific to overproduced hormone
  1. Prolactinoma (Prolactin)
     a) Men: erectile dysfunction, infertility, galactorrhea
     b) Women: galactorrhea, amenorrhea, infertility
  2. Somatotrophic adenoma (Growth hormone)
     a) Gigantism in children
     b) Acromegaly in adults
  3. Corticotrophic adenoma (ACTH)
     a) Cushing's disease
  4. Gonadotrophic adenoma(Follicle-stimulating hormone)
     a) Men: decreased libido, erectile dysfunction


Is this glaucoma?
Differentiating between glaucoma and other optic neuropathies
I have no financial disclosures
Andrew B Mick, OD, FAAO
San Francisco VA Medical Center
UC Berkeley School of Optometry
UCSF Department of Ophthalmology


Optic Nerve Cupping:
Differentiating between glaucoma and compressive optic neuropathy
Early open angle glaucoma (1 case)
Compressive neuropathy (2 cases)
Traumatic optic neuropathy (1 case)
Andrew B. Mick, OD, FAAO
San Francisco VA Medical Center Eye Clinic
UC Berkeley School of Optometry
UCSF Department of Ophthalmology
I have no financial disclosures


At what point in your eye exam does glaucoma usually enter your mind?
63 year old African American male presenting for first time in your practice
No visual complaints, with best corrected vision of 20/25+ OU
Mild nuclear sclerotic cataract, all other anterior segment structures normal
Intraocular pressures of 18/19 mmHg


If IOP is low, for many of us, this is the time we start to suspect glaucoma
Slightly asymmetric and enlarged optic nerve cupping
Failure of recognition: (No visual fields, no OCT)
But is enlarged optic nerve cupping unique to glaucoma?
And what is enlarged cupping in the first place?

First of all, what is enlarged optic nerve cupping?

What is the average cup-disc ratio?

Investigative Ophthalmology & Visual Science, Vol. 29, No. 7, July 1988

Optic Disc, Cup and Neuroretinal Rim Size, Configuration and Correlations in Normal Eyes

Jost Bruno Jonas, Gabriele Charlotte Gusek, and Gottfried Otto Helmut Naumann

457 eyes of 319 subjects
51% men, 49% female
No racial information reported
Mean age of 42.7 years
Mean refractive error -0.13 D

Number	457
c/d Ratio	
Horizontal	0.39 ± 0.28
Vertical	0.34 ± 0.25

Jonas. Invest Ophthalmol Vis Sci 1988;29(7):1151-1158

The Normal Optic Nerve Head

NOELANI M. TAM SING, OD, SHEILA F. ANDERSON, OD, FAAO, and JOHN C. TOWNSEND, OD, FAAO

TABLE 4.
Ethnic differences in normal optic nerve head parameters

Study	Ethnicity	Number of patients studied	Age	Method of measurement	Mean cup-to-disc ratio Vert.	Mean cup-to-disc ratio Horiz.
Chi et al[17]	White	31	18 to 35	RODA	0.41	
	African-American	30			0.62	
Varma et al[18]	White	1853	40 & older	Topcon Imagenet	0.49	
	African-American	1534			0.56	
Mansour[23]	White	51	21 to 54	Zeiss Fundus Camera		
	Hispanic[a]	24				
	Non-American Indian	15				
	Asian[b]	14				
	African-American	21				
Tsai et al[24,6]	White	44	18 to 40	HRT	0.27	0.52
	Hispanic[c]	48			0.33	0.53
	Asian[d]	45			0.29	0.56
	African-American	43			0.41	0.57
Tsai et al[6,7]	White	41	18 to 35	RODA	0.42	
	White	40	>55		0.43	

White
↓
Hispanic / Asian
↓
African American

Optom Vis Sci 2000;77(6):293-301

What cup disc ratios should raise suspicion?

Based on the published data, consensus seems to be:

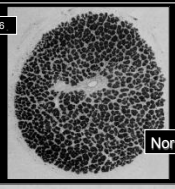
White	~0.40
Asian / Hispanic	~0.55
Black	~0.60

I get suspicious when I see significant increase in c/d ratio compared to these average values

But that just tells you the cupping IS larger than average, not WHY the disc is cupped!

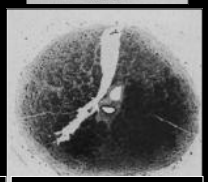
Loss of neural tissue of the optic nerve is not unique to glaucoma!

Quigley. Arch Ophthalmol 1982;100:135-146

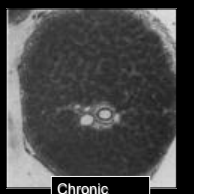


Normal optic nerve

Nutritional optic neuropathy in chronic alcoholic



Non-arteritic anterior ischemic optic neuropathy



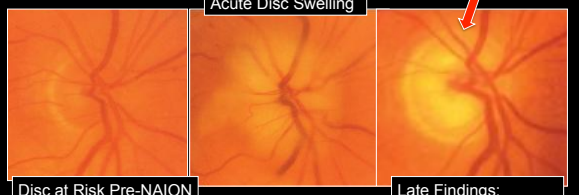
Chronic Papilledema

Prime Example: Anterior Ischemic Optic Neuropathy

Hayreh. Ind J Ophthalmol 2011;59(2):126-136

Looks a lot like glaucoma

Acute Disc Swelling



Disc at Risk Pre-NAION

Late Findings:
- Pallor
- Nerve fiber loss
- Increased cupping

But all those neuropathies look different, right?

We can tell clinically what is glaucoma and what is not!

70 eyes with different reasons for increased cupping:
- Eyes with open angle glaucoma
- Eyes with no ocular disease
- Eyes with hereditary optic neuropathy
- Eyes with compressive optic neuropathy
- Eyes with traumatic optic neuropathy
- Eyes with old CRAO
- Eyes with retrobulbar optic neuritis

Could you group them into one of three groups based just on the nerve?
-Glaucomatous cupping
Non-glaucomatous cupping (A neuropathy that isn't glaucoma)
Normal (No pathologic cupping)

We can tell clinically what is glaucoma and what is not, right?

Nonglaucomatous Excavation of the Optic Disc

Jonathan D. Trobe, MD; Joel S. Glaser, MD; Janet Cassady, MS; Jonathan Herschler, MD; Douglas R. Anderson, MD

Cohort had 29 eyes with non-glaucomatous optic neuropathy:
- 10 eyes with hereditary optic neuropathy
- 11 eyes with compressive optic neuropathy
- 3 eyes with traumatic optic neuropathy
- 3 eyes with old central retinal artery occlusion
- 2 eyes with retrobulbar optic neuritis

Cohort had 32 eyes with open-angle glaucoma

Cohort had 8 eyes with no ocular pathology

Arch Ophthalmol 1980;98:1046-1050

Not as easy as you might think!

Glaucomatous cupping?
Non-glaucomatous cupping? (other neuropathy)
Normal (No pathologic cupping)?

Nonglaucomatous Excavation of the Optic Disc

Jonathan D. Trobe, MD; Joel S. Glaser, MD; Janet Cassady, MS; Jonathan Herschler, MD; Douglas R. Anderson, MD

Of the 29 eyes with non-glaucomatous neuropathies, 48% were graded as not showing any pathology by two or more observers

Of the 29 eyes with non-glaucomatous neuropathies, 21% were graded as having glaucomatous optic neuropathy by two or more observers

Overall, 44% of the 29 non-glaucomatous optic neuropathies were misdiagnosed by at east one observer

Of these misdiagnosed, 77% were the compressive optic neuropathies

Why we can be fooled, especially with compressive neuropathies

Enlargement of the optic cup is nonspecific and found in numerous optic neuropathies, not just glaucoma

Loss of nerve fiber layer, as seen clinically or by OCT measurement, also is nonspecific

Visual fields, used heavily in glaucoma diagnosis, are prone to fluctuation and unreliability

Glaucoma is DIAGNOSED more commonly than compressive optic neuropathy

Compressive lesions (especially non-secreting pituitary adenomas) often have no overt systemic signs/symptoms

The cost / logistics of getting brain imaging leads to resistance to ordering

There is overlapping patient demographics for glaucoma and the most common chiasmal compressive lesions (pituitary adenomas)

The most common cause of compressive optic neuropathy:

Pituitary Tumors: Pituitary Basics

Endocrine gland located at the base of the hypothalamus

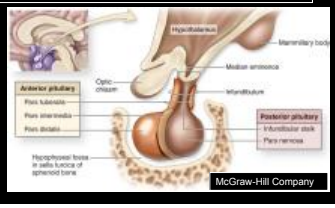
Sets in a bony cavity within the sphenoid bone (sella turcica) within the middle cranial fossa

Connected to the hypothalamus by the pituitary stalk

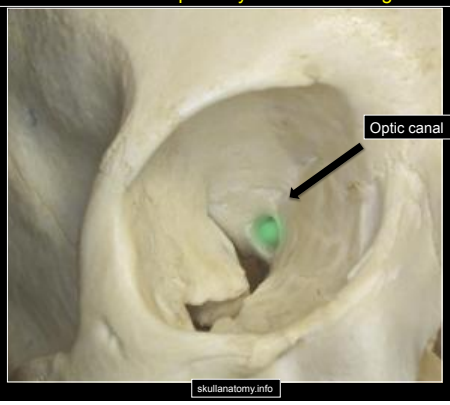
Secretes nine different hormones involved in body homeostasis

Sits just below the optic chiasm.

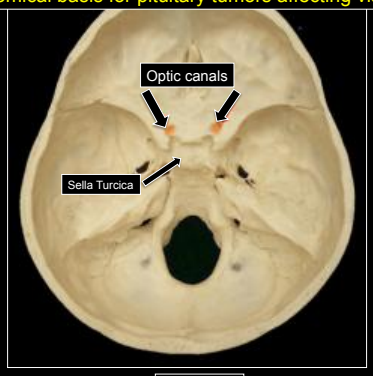
This anatomical relationship is why these tumors are so relevant to an optometric audience!!!



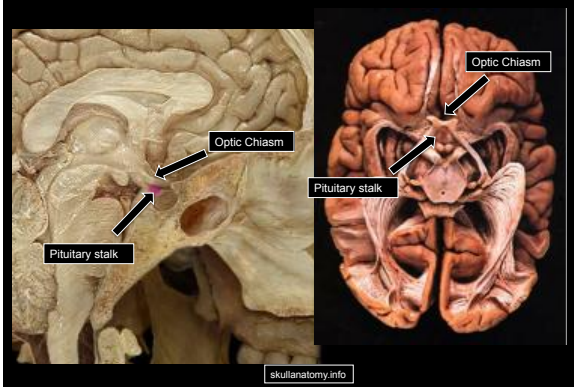
Anatomical basis for pituitary tumors affecting vision



Anatomical basis for pituitary tumors affecting vision

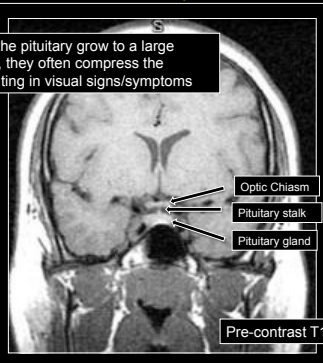


Anatomical basis for pituitary tumors affecting vision



Anatomical basis for pituitary tumors affecting vision

If tumors of the pituitary grow to a large enough size, they often compress the chiasm resulting in visual signs/symptoms



When we talk chiasmal tumors, we're primarily talking about adenomas

- Pituitary adenomas account for 7% -15% of all CNS tumors
- Pituitary adenomas account for ~90% of tumors involving the sella
- Adenomas by definition are cancers of glandular tissue
- Although in generally they are benign, they can result in pathology from local mass effect, altered secretion of normal hormone, and rarely bleeds (Apoplexy)
- Categorized by size:
  - Microadenoma (< 1 cm) – Vast majority of adenomas
  - Macroadenoma (> 1 cm)
- Categorized by function (Secreting vs. non-secreting):
  - Secreting often diagnosed early in life due to hormonal effects
  - Non-secreting often diagnosed in later life with mass effects

Ezzat. Cancer 2004;101:613-9

Pickett. Prim Care Clin Office Pract 2003;30:765-789

Pituitary adenoma basics

Table 1
Classification, prevalence and clinical manifestations of pituitary adenomas

Adenoma type	Prevalence (% of adenomas)	Pituitary hormone secreted (secondary hormone produced)	Tumor hormone staining	Female/male ratio (age related prevalence)	Most frequent clinical manifestations
Prolactinoma	40-45	PRL	PRL	10:1 (2nd to 3rd decade) 1:1 (after 5th decade)	Amenorrhea, galactorrhea. Impotence, decreased libido. Mass effects.
Somatotroph	20	GH (IGF-1)	GH ± PRL	1:1 (4th to 5th decade)	Acromegaly and/or gigantism: soft tissue swelling, prognathism and frontal bossing, deep voice, excess sweating, carpal tunnel, DM or impaired GT, cardiomegaly, HTN. Mass effects.
Corticotroph	10-12	ACTH (Cortisol)	ACTH	8:1 (3rd to 4th decade)	Cushing's disease/hypercortisolism: central adiposity, DM or impaired GT, HTN, depression/anxiety, hirsutism, fragile skin, and osteoporosis/porosis.
Gonadotroph	15	Usually none. LH, FSH, or α subunit. (Rarely testosterone, estrogen)	LH, FSH and/or β-subunit or α-subunit	1:1.5 (after 5th decade)	Mass effects. Hypopituitarism.
Null-cell	5-10	None	None	1.7:1	Mass effects. Hypopituitarism.
Thyrotroph	< 5	TSH (Thyroxine)	TSH	1:1 (3rd to 4th decade)	Mild hyperthyroidism, goiter. Mass effects.

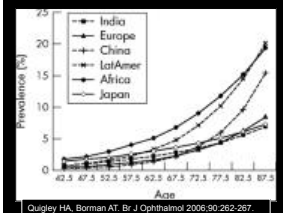
PRL: Prolactin
GH: Growth Hormone
ACTH: Adrenocorticotropic hormone
LH: Luteinizing hormone
FSH: Follicle stimulating hormone
TSH: Thyroid stimulating hormone

Pickett. Prim Care Clin Office Pract 2003;30:765-789

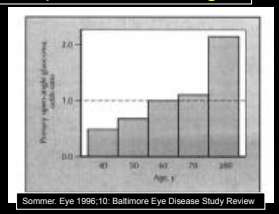
Why we can be fooled, especially with compressive neuropathies

There is overlapping patient demographics for glaucoma and one of the most common chiasmal compressive lesions (Pituitary adenoma)

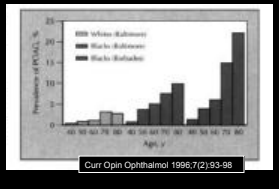
Glaucoma becomes more prevalent with age!



Quigley HA, Borman AT. Br J Ophthalmol 2006;90:262-267.



Sommer. Eye 1996;10: Baltimore Eye Disease Study Review



Curr Opin Ophthalmol 1996;7(2):93-98

Glaucoma prevalence increases with age

Rate of increase greatest in African American Individuals

Glaucoma becomes more prevalent with age!

Study	Racial/Ethnic Group	Age-Specific Prevalence Age Groups (yrs)					
		40–49	*50–59*	*60–69*	*70–79*	*≥80*	*Total*
Baltimore Eye Study*	*Blacks*	1.27	4.15	6.19	8.88	12.87	4.97
Barbados Eye Study†	*Blacks*	1.4	4.1	6.7	14.8	23.2	6.8
LALES	**Latinos**	**1.32**	**2.92**	**7.36**	**14.72**	**21.76**	**4.74**
Proyecto VER‡	*Latinos*	0.50	0.59	1.73	5.66	12.63	1.97
Baltimore Eye Study*	*NHW*	0.18	0.32	1.53	3.33	1.94	1.44
Blue Mountains Eye Study§	*NHW*	0.4‖		1.3	4.7	11.4	3.0
Visual Impairment Project¶	*NHW*	0.5	1.5	4.5	8.6	9.9	3.4
Rotterdam#	*NHW*		0.72	1.76	3.2	3.05	1.88

Across all age groups, African American population affected at a higher rate

LALES. Ophthalmology 2004;111(8):1439-1448.

Guess what else becomes more common with age and in black patients

Demographic differences in incidence for pituitary adenoma

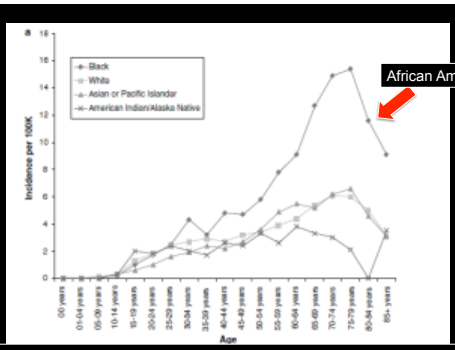
Bradley D. McDowell · Robert B. Wallace · Ryan M. Carnahan · Elizabeth A. Chrischilles · Charles F. Lynch · Janet A. Schlechte

Data from 17 surveillance, epidemiology and end-result (SEER) programs in the United States

From 2004 – 2007, programs recorded 8276 pituitary adenomas

Of those, 8118 had known sex and race data recorded

Pituitary 2011;14:23-40.



African Americans

In the study, the mean tumor size was 23 mm for men and 15 mm for women

Size likely to result in compression of the overlying chiasm

Pituitary 2011;14:23-40.

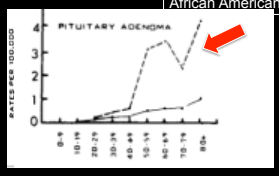
NEOPLASMS OF THE CENTRAL NERVOUS SYSTEM

Incidence and Population Selectivity in the Washington DC, Metropolitan Area

M. Y. Heshmat, MD, DrPH,* J. Kovi, MD,† C. Simpson, MPH,‡ J. Kennedy, MS,§ and K. J. Fan, MD‖

Cancer 1976;38:2135-2142

African Americans



Overall, 990 primary tumors of the central nervous system were identified in the community

Compared to white population, pituitary adenoma rates were 4x greater for black men and 3x for black women

ETHNIC DISTRIBUTION OF PRIMARY CENTRAL NERVOUS SYSTEM TUMORS IN WASHINGTON, DC, 1971 TO 1985

J Natl Med Assoc 1992;84:858

Armed Forces Institute of Pathology identified 8947 cases of CNS tumor

Pituitary adenoma reported at a 4.2:1 ratio in black population compared to white population

Why we can be fooled

The optic nerves can look strikingly similar

There is overlapping demographics between glaucoma and CON

Glaucoma is DIAGNOSED more commonly than compressive optic neuropathy

Implication that pituitary adenoma are rare
But how rare are they?

Pituitary adenomas are rare……right?

The Prevalence of Pituitary Adenomas

A Systematic Review

Pituitary adenoma prevalence studies utilize either autopsy or radiographic evidence

Meta-analysis of all published studies on pituitary adenoma prevalence prior to the year 2000

Identified 10 studies: 3 radiographic and 7 autopsy evidence based

Ezzat. Cancer 2004;101:613-9

The Prevalence of Pituitary Adenomas

A Systematic Review

Estimated prevalence of pituitary adenoma across all autopsy studies was 14.4%

Estimated prevalence of pituitary adenoma across all radiographic studies was 22.5%

Only two of the studies segregated tumor by size with prevalence of macroadenoma only being 0.16-0.20% (~1/500 individuals)

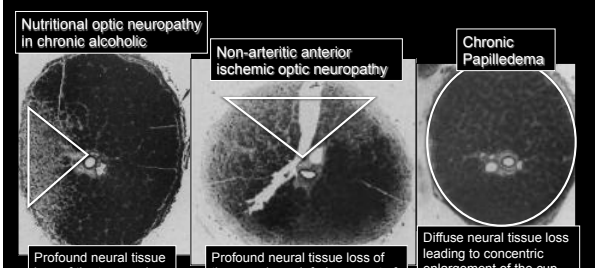
Authors did note that study population was over-represented by adults over age 50 (But so is your glaucoma suspect population!)

Ezzat. Cancer 2004;101:613-9

So what are clinicians to do to differentiate between glaucoma and compressive optic neuropathy?

To start, look for the signs besides increased cupping that are characteristic of glaucoma……

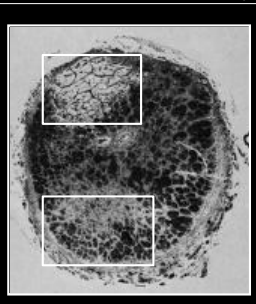
Loss of neural tissue of the optic nerve is not unique to glaucoma, but the PATTERNS can help us determine underlying cause!



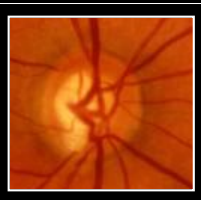
Quigley. Arch Ophthalmol 1982;100:135-146

Glaucomatous optic neuropathy also has characteristics patterns

In glaucoma there is preferential loss of retinal nerve fiber layer at the inferior and superior poles of the nerve (Especially inferior and superior temporal sections)



Therefore, cupping is more likely to manifested with increased vertical c/d ratios and focal loss of neuroretinal rim at inferotemporal and superotemporal poles



Quigley. Am J Ophthlamol 1983;95:673

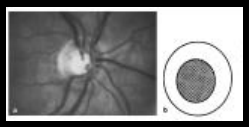
Glaucomatous optic neuropathy has characteristics patterns

Optic disc morphometry in chronic primary open-angle glaucoma
I. Morphometric intrapapillary characteristics*

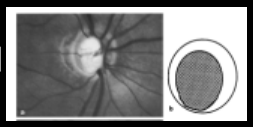
Jonas. Graefes Arch Clin Exp Ophthalmol 1988;226(6):522-530.

233 open angle glaucoma nerves vs. 253 normal nerves

Morphologic differences between early OAG and normal nerves (p<0.001)
- Decreased quotient of horizontal to vertical c/d ratio
- Greater thinning of the inferior rim relative to the superior rim



vs



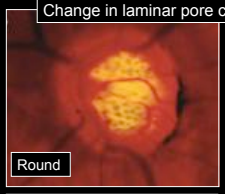
Glaucomatous optic neuropathy is more than just increased cupping

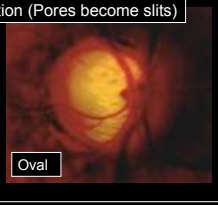
Vertical elongation of the cup or notching of vertical pole

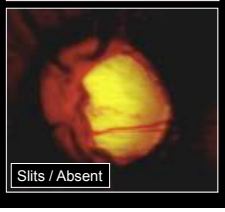
Connective tissue remodeling leads to change in cup depth and shape plus altered laminar pore conformation

Remodeling of the laminar tissues is essentially unique to glaucoma!

Change in laminar pore conformation (Pores become slits)







Number of visible pores increases with loss of neural tissue

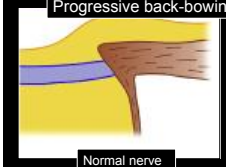
Shape of visible pores change in shape from round, to oval, to slits with connective tissue remodeling

In advanced glaucoma, pores may be completely absent due to scarring

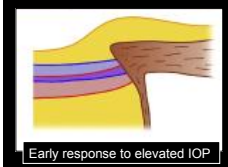
Tezel. Br J Ophthalmol 2004;88:251-256.

Remodeling of the laminar tissues is essentially unique to glaucoma!

Progressive back-bowing of the laminar floor (Cup gets deeper)



Normal nerve



Early response to elevated IOP

Laminar changes early in glaucoma:

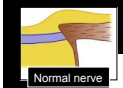
Lamina begins to bow backward

Insertion of the lamina begins to move more posterior in the sclera and even the pia mater

Downs. Exp Eye Res 2011;93:133-140.

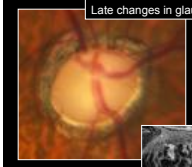
Remodeling of the laminar tissues is essentially unique to glaucoma!

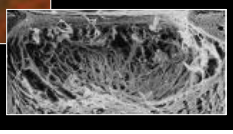
Remodeling leads to change in cup depth and shape



Normal nerve

Late changes in glaucoma





Changes late in glaucoma:

The floor of the lamina progressively moves posteriorly

As remodeling continues, overall laminar thickness decreases as sheets become compressed together.

The lateral walls bow into the adjacent sclera while floor progressively deepens giving W shape

Downs. Exp Eye Res 2011;93:133-140.

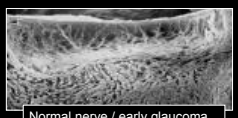
Glaucomatous optic neuropathy is more than just increased cupping

Vertical elongation of the cup or notching of vertical pole

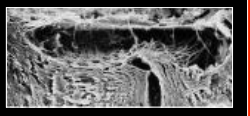
Connective tissue remodeling leads to change in cup depth and shape plus altered laminar pore conformation

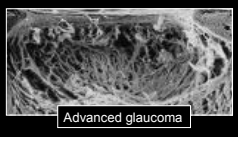
Vascular tissues are also affected in progressive glaucomatous optic neuropathy

Vascular tissues are also affected in glaucomatous optic neuropathy

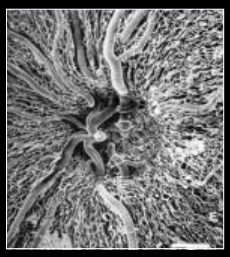


Normal nerve / early glaucoma





Advanced glaucoma

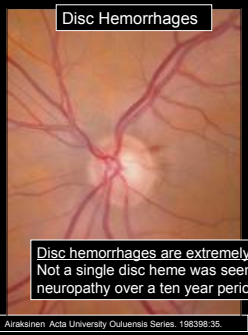


Quigley Am J Ophthlamol 1983;95:673

Prog Ret Eye Res 2002;21(4):359-393

Vascular tissues are also affected in this progressive optic neuropathy

Disc Hemorrhages



Found in ~0.5% of healthy individuals over the age of 55 years

Found in ~5-7% of open angle glaucoma patients

Found in 20-40% of low tension glaucoma patients

Disc hemorrhages are extremely rare in compressive optic neuropathy:
Not a single disc heme was seen in 44 eyes with known compressive optic neuropathy over a ten year period

Airaksinen Acta University Ouluensis Series. 198398:35.

Bengtsson Acta Ophthalmol 1981;59:1-14.

Bengtsson Acta Ophthalmol 1986;64:152-156

Drance Surv Ophthalmol 1989;33(5):331-337

Greenfield. Ophthalmology 1998;105:1866-1874

Glaucomatous optic neuropathy is more than just increased cupping

Vertical elongation of the cup or notching of vertical pole

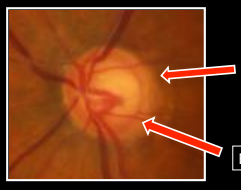
Connective tissue remodeling leads to change in cup depth and shape plus altered laminar pore conformation

Vascular tissues are also affected in progressive glaucomatous optic neuropathy resulting in disc hemorrhages

Peripapillary tissues are also affected in progressive glaucomatous optic neuropathy

Glaucomatous changes to peripapillary tissues

Chorioretinal atrophy surrounding the optic nerve: Peripaillary atrophy



Alpha Zone

Beta zone

Alpha Zone:	Peripheral zone characterized by an irregular hypopigmentation and hyperpigmentation of the RPE.
Beta Zone:	Inner zone with marked atrophy of the choriocapillaris, RPE, and photoreceptor outer segments allowing for visualization of the large choroidal vessels. Always inside of alpha zone and outside a scleral ring.

Curcio. Ophthalmology2000;107:334-343.

Glaucomatous changes to peripapillary tissues

Alpha zone PPA is present in almost all normal eyes most commonly temporally

Beta zone PPA is found in only 15-20% of normal eyes

Both alpha and beta zone PPA is significantly larger in eyes with glaucoma compared to in normals

Size of both alpha and beta zones are correlated with severity of glaucoma

14-37% of eyes with open angle glaucoma show progression of PPA area over long-term follow-up

Progressive PPA is not seen in non-glaucomatous optic neuropathies such as compressive optic neuropathy

Hayreh Ophthalmolog 2001;108:1586-94.

Uchida. Ophthalmolog 1998;105:1541-1545.

Kwon. J Glaucoma 2003;12:409-416.

Rath Ey 2003;17:1019-1024

So what are clinicians to do to differentiate between glaucoma and compressive optic neuropathy?

You have already looked for changes to the nerve that are relatively specific to glaucomatous optic neuropathy:

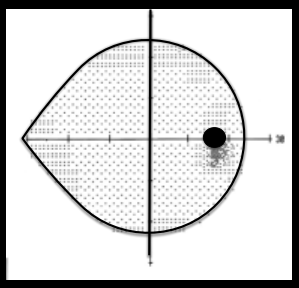
- Vertical elongation of cup or vertical quadrant notch
- Laminar changes
- Disc hemorrhages
- Enlarged areas of or progressive peripapillary atrophy

Next, make sure there is not evidence of the changes to the nerve characteristic of compressive optic neuropathy?

Let go back to the anatomy!

Let's discuss the visual fields first: Remembering the anatomy!

The center of the visual field, bisected by the vertical meridian, corresponds to the fovea and separates the nasal and temporal field



We all remember that chiasmal compression results in bi-temporal field defects

Therefore, chiasmal compression results from compression of nerve fibers originating nasal to the fovea

Anatomy of the retinal nerve fiber layer

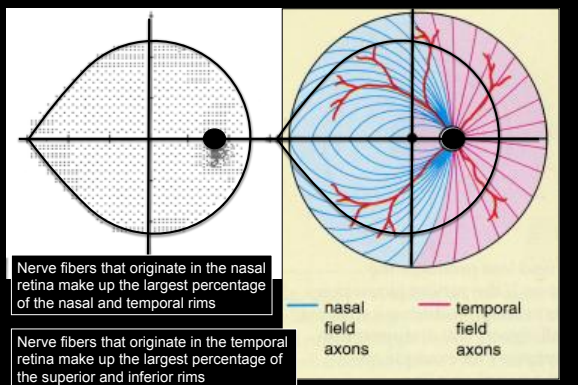
All ganglion cell axons originating nasal to the optic nerve within the retina essentially take a direct course to the nasal, superior-nasal, and inferior-nasal optic nerve

Ganglion cell axons originating temporal to the optic disc within the retina also take a direct course to the superior-nasal, nasal, and inferior-nasal nerve

The large number of ganglion cell axons originating from the fovea course directly to the temporal optic nerve creating the papillomacular bundle

Ganglion cell axons originating from portions of the retina temporal to the fovea must take an arcuate course around the papillomacular bundle entering the superior and superior-temporal, inferior and inferior-temporal nerve

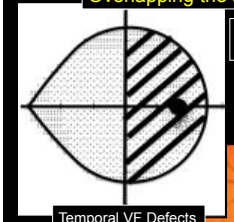
Overlapping the anatomy with the visual field



Nerve fibers that originate in the nasal retina make up the largest percentage of the nasal and temporal rims

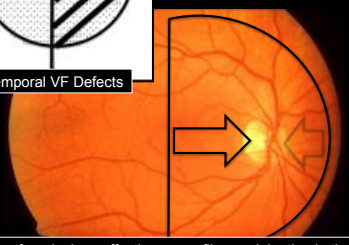
Nerve fibers that originate in the temporal retina make up the largest percentage of the superior and inferior rims

Overlapping the anatomy with the visual field



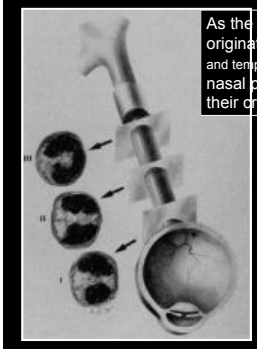
Result from retinal nerve fiber layer loss primarily at the nasal and temporal rims of the nerve

Temporal VF Defects

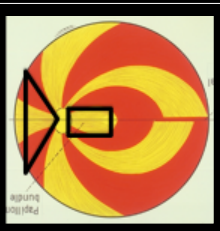


Arise from lesions affecting nerve fibers originating in the nasal retina

Following the nasal and temporal rim nerve fibers back toward chiasm

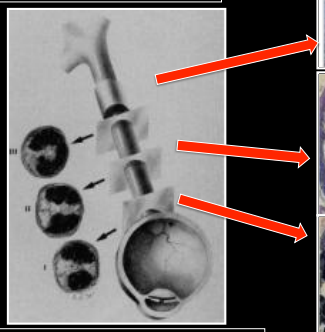


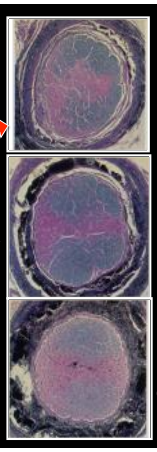
As the nerve approaches the chiasm, the axons originating in the retina nasal to the fovea (nasal and temporal rims of OHN) begin to occupy more nasal portions of the optic nerve in anticipation of their crossover at the chiasm



Anatomy of the optic nerve behind the globe

Unsold and Hoyt. Arch Ophthalmol 1980;98:1637-1638.





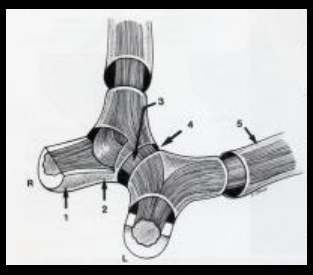
Chiasm

Globe

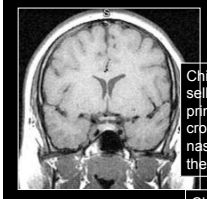
Left optic nerve after chiasmal compression

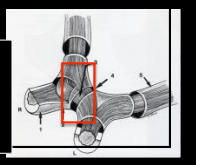
Anatomy of the retro-orbital optic nerve

At the chiasm, axons from the nasal retina cross over to the contralateral optic tract.

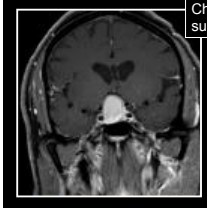


Remember the spatia relationship between the chiasm and the pituitary

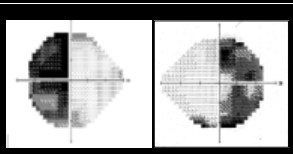




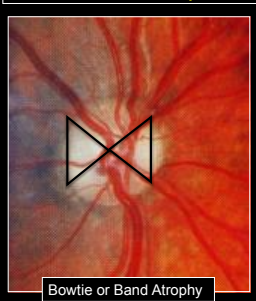
Chiasmal compression from sellar masses results in damage primarily to the fibers that crossover and originated at the nasal and temporal aspects of the optic nerve



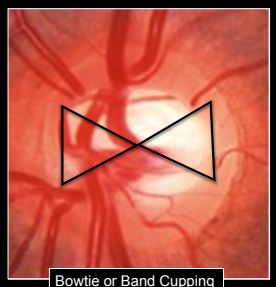
Chiasmal compression therefore initially results in superior > inferior bitemporal visual field loss



Chiasmal compression results in damage to the fibers that enter the nasal and temporal aspects of the optic nerve at the lamina and then cross over in the chiasm just above the sella tursica



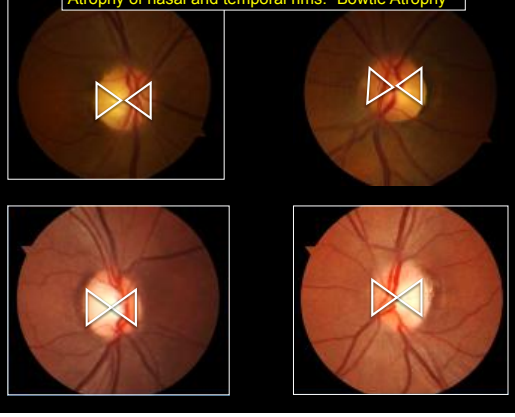
Bowtie or Band Atrophy



Bowtie or Band Cupping

Hildebrand. Arch Ophthalmol 2010;128(12):1625-6.

Atrophy of nasal and temporal rims: “Bowtie Atrophy”



So what are clinicians to do to differentiate between glaucoma and compressive optic neuropathy?

You have already looked for changes to the nerve that are relatively specific to glaucomatous optic neuropathy:

- Vertical elongation of cup or vertical quadrant notch
- Laminar changes
- Disc hemorrhages
- Enlarged areas of or progressive peripapillary atrophy

Chiasmal compressive optic neuropathy preferentially causes atrophy and NFL loss to the fibers that represents the temporal VF and enter the nasal and temporal rims of the optic nerve that later crossover at the chiasm

How does compressive optic neuropathy show up on OCT imaging

Do we see preferential loss of nasal and temporal nerve fiber layer as measured by ocular coherence tomography?

Comparison of retinal nerve fiber layer measurements using Stratus OCT fast and regular scan protocols in eyes with band atrophy of the optic nerve and normal controls

Monteiro Arq Bras Oftamol 2008;71(4):534-9

Optical Coherence Tomography Detects Characteristic Retinal Nerve Fiber Layer Thickness Corresponding to Band Atrophy of the Optic Discs

Kanamori. Ophthalmology 2004;111:2278

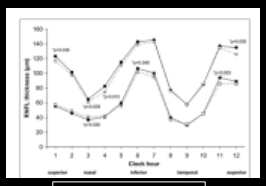
SCIENTIFIC REPORT

Optical coherence tomography analysis of axonal loss in band atrophy of the optic nerve

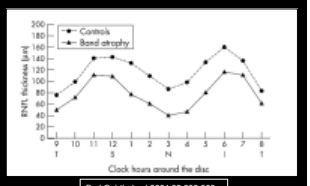
M L R Monteiro, B C Leal, A A M Rosa, M D Bronstein

Monteiro. Br J Ophthalmol2004;88:868-899

The literature primarily reports advanced cases of compressive neuropathy and therefore extensive diffuse NFL atrophy in all quadrants



Arq Bras Oftamol 2008;71(4):534-9



Br J Ophthalmol 2004;88:868-899

Hidden in data:
Greater temporal and nasal RNFL loss

Parameter	Band Atrophy: Within Normal Limits	Borderline	Outside Normal Limits
Average thickness	1 (3%)	6 (16%)	30 (81%)
Superior thickness	10 (27%)	4 (11%)	23 (62%)
Temporal thickness	6 (16%)	6 (16%)	25 (68%)
Inferior thickness	9 (24%)	11 (30%)	17 (46%)
Nasal thickness	2 (5%)	21 (57%)	14 (38%)

Monteiro. Am J Ophthalmol 2007;896-899.

Optical Coherence Tomography Detects Characteristic Retinal Nerve Fiber Layer Thickness Corresponding to Band Atrophy of the Optic Discs

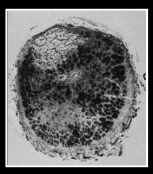
Kanamori. Ophthalmology 2004;111:2278

Measurement (Mean ± SD) (μm)	Normal (n = 160)	Band atrophy (n = 34)	P Value*	Reduction Rate (%)
Average	120.8 ± 12.9	80.1 ± 22.3	<0.001	33.7
Quadrants				
Superior	145.5 ± 19.6	101.2 ± 32.3	<0.001	30.4
Temporal	98.7 ± 20.8	57.2 ± 25.6	<0.001	42.0
Inferior	143.1 ± 19.5	107.5 ± 27.9	<0.001	24.9
Nasal	92.6 ± 20.4	50.7 ± 21.6	<0.001	45.2

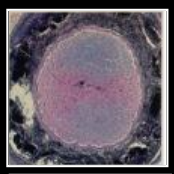
Higher percentage reduction in nerve fiber layer thickness in the nasal/temporal quadrants compared to superior/inferior of compressive neuropathy patients compared to normal

This is opposite from studies comparing normal subjects to early glaucoma where superior/inferior quadrants show greater percentage of thinning

Regarding OCT Measurements in Compressive Optic Neuropathy



Glaucomatous Neuropathy



Compressive Neuropathy

In advanced OAG and compressive optic neuropathy there is significant thinning in all quadrants of the optic nerve

But in early glaucoma, thinning of the superior and inferior quadrants predominates and nasal/temporal thinning is less likey

In early compressive optic neuropathy, thinning of the nasal and temporal quadrants predominates

So what are clinicians to do to differentiate between glaucoma and compressive optic neuropathy?

You have already looked for changes to the nerve that are relatively specific to glaucomatous optic neuropathy:
- Laminar changes
- Disc hemorrhages
- Enlarged areas of or progressive peripapillary atrophy

Chiasmal compressive optic neuropathy preferentially affects nerve fibers that represent the temporal VF, enter the nasal and temporal rims of the optic nerve, and later crossover at the chiasm

Although both neuropathies show OCT NFL thinning in all quadrants in advanced cases, early in glaucoma vertical poles are affected and early in compressive horizontal poles are affected

Glaucoma should be isolated to the nerve (No orbital, hormonal or mass-effect signs!)

How to differentiate between glaucoma and orbital compressive optic neuropathy!

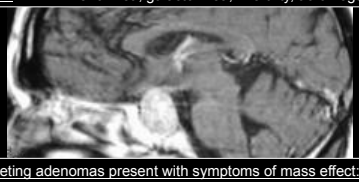
Look for optic nerve, visual field and OCT signs consistent with glaucoma besides increased cupping

Additional signs of orbital compressive:
- Proptosis
- Conjunctival injection
- Optic nerve head collaterals
- Extraocular muscle abnormalities from other cranial neuropathies

Non-eye symptoms of pituitary adenoma

Secreting adenomas: depends on which over-produced hormone:

Men:	Reduced libido, erectile dysfunction, galactorrhea, acromegaly, gigantism
Women:	Amenorrhea, galactorrhea, infertility, acromegaly/gigantism



Non-secreting adenomas present with symptoms of mass effect:
- Headache
- Seizures
- Reduced vision
- Visual field deficits (Usually start temporal, but can expand)
- Rarely cranial neuropathies

The Cupped Disc

Who Needs Neuroimaging?

Daniel S. Greenfield, MD,[1] R. Michael Siatkowski, MD,[2] Joel S. Glaser, MD,[1,3] Norman J. Schatz, MD,[1,3] Richard K. Parrish II, MD[1]

Ophthalmology: 1998;105(10):1866-74

Retrospective case-control study

Fifty-two eyes of 29 patients with OAG and no IOP over 21 mmHg and clear brain imaging

Forty-four eyes of 28 patients with compressive lesions resulting in visual field defects and cup-disc ratios of 0.4 or greater

Compressive patients should not have any neurologic symptoms suggesting a non-glaucomatous etiology

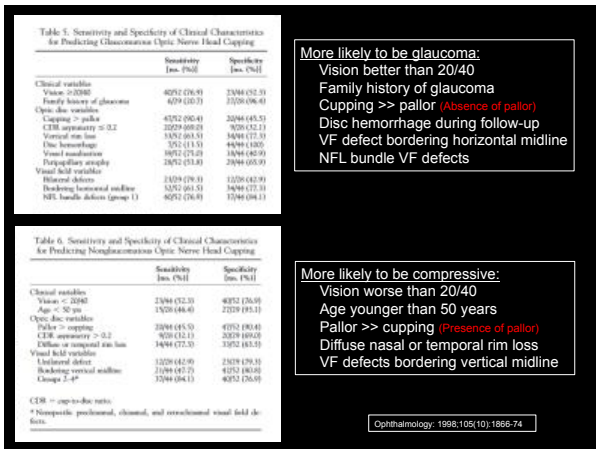
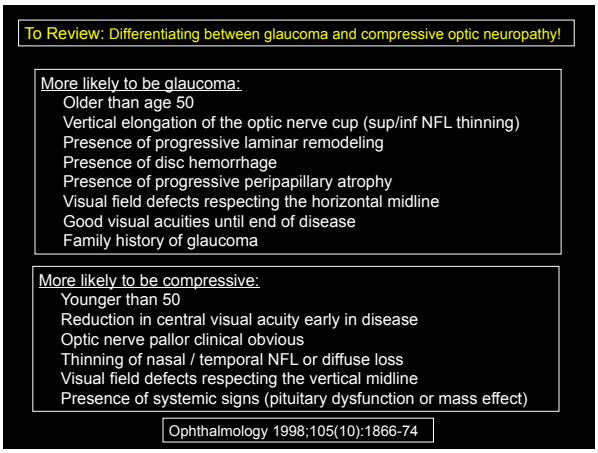


Table 5. Sensitivity and Specificity of Clinical Characteristics for Predicting Glaucomatous Optic Nerve Head Cupping
Table 6. Sensitivity and Specificity of Clinical Characteristics for Predicting Nonglaucomatous Optic Nerve Head Cupping
More likely to be glaucoma:
Vision better than 20/40
Family history of glaucoma
Cupping >> pallor (Absence of pallor)
Disc hemorrhage during follow-up
VF defect bordering horizontal midline
NFL bundle VF defects
More likely to be compressive:
Vision worse than 20/40
Age younger than 50 years
Pallor >> cupping (Presence of pallor)
Diffuse nasal or temporal rim loss
VF defects bordering vertical midline
Ophthalmology. 1998;105(10):1866-74


To Review: Differentiating between glaucoma and compressive optic neuropathy!
More likely to be glaucoma:
Older than age 50
Vertical elongation of the optic nerve cup (sup/inf NFL thinning)
Presence of progressive laminar remodeling
Presence of disc hemorrhage
Presence of progressive peripapillary atrophy
Visual field defects respecting the horizontal midline
Good visual acuities until end of disease
Family history of glaucoma
More likely to be compressive:
Younger than 50
Reduction in central visual acuity early in disease
Optic nerve pallor clinical obvious
Thinning of nasal / temporal NFL or diffuse loss
Visual field defects respecting the vertical midline
Presence of systemic signs (pituitary dysfunction or mass effect)
Ophthalmology 1998;105(10):1866-74


Is this glaucoma?
Differentiating between glaucoma and other optic neuropathies
Thank you for your attention!
Andrew B Mick, OD, FAAO
San Francisco VA Medical Center
UC Berkeley School of Optometry
UCSF Department of Ophthalmology

Andrew B Mick, OD, FAAO

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Eye Clinic (112-A)
4150 Clement St
San Francisco, California, CA 94121
415-221-4810 x 4606 (Office), 415-378-0028 (Cell)
andrew.mick@va.gov

EDUCATION

1993-1997	**University of Michigan,** Ann Arbor. Bachelors of Science in Biology
1997-2001	**University of California,** Berkeley. Doctorate of Optometry
2001-2002	**Bascom Palmer Eye Institute** **University of Miami, Department of Ophthalmology** Optometric Residency in Ocular Disease

EMPLOYMENT

1995-1997	**Kellogg Eye Center, University of Michigan, Department of Ophthalmology** Glaucoma/Molecular Biology Research Assistant Principle Investigator: Julia E. Richards, Ph.D.
2002-2004	**Meredith Morgan Eye Center, University of California Berkeley** Clinical Faculty, School of Optometry
2002-Present	**San Francisco VA Medical Center** Staff Optometrist (2002-Present) Optometry Student Externship Coordinator (2002-2012) Optometric Residency Coordinator (2012-Present)

FACULTY APPOINTMENTS

2002-Present	**University of California, Berkeley, School of Optometry** Associate Clinical Professor
2007-Present	**University of California, San Francisco, Department of Ophthalmology** Associate Clinical Professor

HONORS AND AWARDS

2000	Harris Family Scholarship
2000	California Optometric Association Junior Leadership Award
2001	Thal/VSP Excellence in Primary Care Award
2001	Vision West Annual Scholarship

2001	Vistakon Award of Contact Lens Excellence
2001	Robert Gordon and Andrea Silvers Award
2001	William Feinbloom Low Vision Award
2001	Medical Eye Services Award
2001	University of California, Berkeley, Gold Retinoscope Award
2003	American Academy of Optometry Fellowship
2004	San Francisco VA Medical Center, Service and Patient Care Award
2012	Bernard Dolan Residency Mentor of the Year Award

BOOK CHAPTERS

1. Mick AB. Lacrimal disorders. In Onofrey B, Skorin L, Holdeman N (Editors). Ocular Therapeutics Handbook: A Clinical Manual 2rd Edition 2005. Philadelphia: Loppincott, Williams, Wilkins.

2. Mick AB. Ocular Trauma. In Onofrey B, Skorin L, Holdeman N (Editors). Ocular Therapeutics Handbook: A Clinical Manual 2rd Edition 2005. Philadelphia: Loppincott, Williams, Wilkins.

3. Mick AB. Lacrimal disorders. In Onofrey B (Editor). Ocular Therapeutics Handbook: A Clinical Manual 3rd Edition 2011. Philadelphia: Loppincott, Williams, Wilkins.

4. Mick AB. Ocular Trauma. In Onofrey B (Editor). Ocular Therapeutics Handbook: A Clinical Manual 3rd Edition 2011. Philadelphia: Loppincott, Williams, Wilkins.

PEER REVIEWED PUBLICATIONS

1. Othman MI, Sullivan SA, Skuta GL, Cockrell DA, Stringham HM, Downs CA, Fomes A, Mick AB, Boehnke M, Vollrath D, Richards JE. Autosomal dominant nanophthalmous (NN01) with high hyperopia and angle closure glaucoma maps to chromosome 11. *Am J Hum Genet* 1998;63:1411-1417.

2. Mick AB, Gonzalez S, Dunbar MT, McSoley JJ. A cost analysis of the prostaglandin analogs. Optometry 2002;73(10):614-619.

3. Tsou-Chong J, Mick AB. Choroidal metastasis: Case reports and review of the literature. *Optometry* 2005;76(5):293-301.

4. Hicks D, Mick AB. Recurrent conjunctival hemorrhage leading to the discovery of ocular adnexal lymphoma. *Optometry* 2010;81(10):528-32.

5. Harrison WW, Bearse MA, Schneck ME, Wolfe BE, Jewell NP, Barez S, Mick AB, Dolan BJ, Adams AJ. Prediction by retinal location of the onset of diabetic macular edema in patients with nonproliferative diabetic retinopathy. *Invest Ophthalmol Vis Sci* 2011;52(9):6825-6831.

6. Guan H, Mick A, Porco T, Dolan BJ. Preoperative factors associated with IOP reduction after cataract surgery. *Optom Vis Sci* 2013;90(2):179-184.

PEER REVIEWED POSTERS

1. Carlson PE, Mick AB, McNamara NA, Fleiszig SMJ. Hypoxia protects human corneal epithelial cells from killing by cytotoxic P. Aeruginosa. ARVO, 2000.

2. Tran T, Mick A, Dolan B. Posterior segment complications of interferon therapy for chronic hepatitis C. American Academy of Optometry; Dallas 2003.

3. Fong C, Chen M, Mick A. Ocular side effects with reduced vision from high dose, long term chlorpromazine treatment. American Academy of Optometry; San Diego 2005.

4. Yoshiyama K, Mick A, Dolan B. Corneal crystal deposits secondary to multiple myeloma. American Academy of Optometry; Denver 2006.

5. Wong A, Dolan B, Mick A. Visual loss as the only presenting symptom in a patient with AIDS-associated progressive multifocal leukoenchephalopathy. American Academy of Optometry; Tampa 2007.

6. Tobin L, Dolan B, Mick A. Idiopathic intracranial hypertension presenting as symptomless unilateral optic disc edema. American Academy of Optometry; Tampa 2007.

7. Hicks D, Mick A. Ocular adnexal lymphoma presenting as recurrent subconjunctival hemorrhage. American Academy of Optometry; Orlando 2009.

8. Bedwell A, Mick A. Spectral domain OCT in four patients with adult onset foveomacular vitelliform dystrophy. American Academy of Optometry; Boston, MA 2011.

9. Jones H, Mick A. Expanding the differential diagnosis of papilloedema: Ruling out cerebral venous thrombosis. American Academy of Optometry; Boston, MA 2011

10. Flettner J, Mick A, Dolan B. Federal aviation (FAA) vision requirements: What are your responsibilities when a pilot develops a disqualifying visual condition? American Academy of Optometry; Phoenix, AZ 2012

11. Meadows J, Bahn M, Mick A. Antibiotic therapy in anticoagulated patients with risk factors for community associated methicillin-resistant Staphylococcus aureus. American Academy of Optometry; Seattle, WA 2013.

NON-PEER REVIEWED PUBLICATIONS

1. Mick AB. A revolution at Berkeley. *California Optometry* 1999;26(6):21.

2. Mick AB. A cancer patient's vision declines. *Review of Optometry* 2002;139(2):101-102

3. Mick AB. Book Review: Imaging the eye from front to back with RTVue fourier domain optical coherence tomography. *Optom Vis Sci* 2011;88:781.

4. Mick AB. Book Review: Cataracts: A patient's guide to treatment. *Optom Vis Sci* 2012;89(10).

5. Chen-Lynch M, Mick AB. Nonnecrotizing anterior scleritis mimicking orbital inflammatory disease. *Clin Optom* 2013;5:29-37.

NATIONAL PROFFESSIONAL APPOINTMENTS

1999	**American Optometric Association** House of Delegates, Student Delegate
2004-2006	**American Academy of Optometry** Membership Committee
2005-2008	**National Board of Examiners in Optometry** Part III Examiner
2006-2010	**Accreditation Council on Optometric Education** Consultant (2006-2008) Team Chair (2009-2010)
2006-2016	**American Academy of Optometry, Scientific Program Committee** Member (2006-2012) Vice Chair (2012-2014) Chair (2014-2016)
2014-2016	**Optometric Glaucoma Foundation** Chief Financial Officer
2015-2016	**American Academy of Optometry, Awards Committee** Member
2015-Present	**American Academy of Optometry, Glaucoma Diplomate Program** Candidate Mentor

VETERANS AFFAIRS COMMITTEE APPOINTMENTS

2004-2006	**Advanced Clinic Access Committee** Eye Clinic Representative
2005-Present	**Veterans Integrated Service Network 21** Co-Consultant to National Optometry Service
2009-Present	**Reusable Medical Equipment Disinfection Committee** Eye Clinic Representative
2016 – Present	**Direct Scheduling Committee** Eye Clinic Representative

ACADEMIC COMMITTEE APPOINTMENTS

1999-2000	**University of California, Berkeley, School of Optometry** Optometry Student Association President
2000	**University of California, Berkeley, School of Optometry** ACOE Self Study Committee: Student Education
2000	**University of California, Berkeley, School of Optometry** Admissions Committee
2002-2006	**University of California, Berkeley, Optometry Alumni Association** Vice President
2003-2004	**University of California, Berkeley, School of Optometry** Clinic Advisory Committee
2002-2005	**University of California, Berkeley, School of Optometry** Faculty Glaucoma Certification Program Instructor
2006	**University of California, Berkeley, School of Optometry** ACOE Self Study Committee: Resident Education
2006-2008	**University of California, Berkeley, School of Optometry** Clinical Curriculum Committee
2008	**University of California, Berkeley, School of Optometry** California State TPA Glaucoma Course Curriculum Committee
2008-2009	**University of California, Berkeley, School of Optometry** Curriculum Committee
2011-2012	**University of California, Berkeley, School of Optometry** California State Optometry Glaucoma Certification Course Beta II Course Reviewer Beta III Course Reviewer Examination Question Writer Grand Rounds Facilitator
2012	**University of California, San Francisco** **Department of Ophthalmology** Staff Optometrist Search Committee
2014	**University of California, San Francisco** **Department of Ophthalmology** San Francisco General Hospital Staff Optometrist Search Committee
2016	**University of California, San Francisco** **Department of Ophthalmology** Staff Optometrist Search Committee

EXPERT WITNESS CONSULTING

2012	**Montana Fourth Judicial District Court**
2012 - Present	**Superior Court of the State of California**
2016 – Present	**Superior Court of the State of Illinois**

JOURNALS EDITED

2011-Present	**Optometry and Vision Science** **Journal of the American Academy of Optometry** Associate Topical Editor (2011-2014) Editorial Board (2014-Present)

JOURNALS REVIEWED

2004-Present	**Optometry and Vision Science** Journal of the American Academy of Optometry
2007-2011	**Optometry** Journal of the American Optometric Association
2013-Present	**Journal of General Internal Medicine**

INVITED PROFESSIONAL LECTURES

1. **American Academy of Optometry, Dallas, TX, 2003**
   Recent large multi-center clinical trials and how they have shaped optometric glaucoma management

2. **University of California, Berkeley, 2003**
   Optometry Alumni Association Reunion
   The ocular ischemic syndrome

3. **Clinical Educators in Eyecare, San Jose, CA, 2003**
   Glaucoma treatment: A study driven philosophy

4. **University of California, Berkeley, 2003**
   Meredith Morgan Symposium
   Glaucoma management in optometric practice

5. **Sacramento Optometric Society, 2003**
   Integrating recent glaucoma clinical trials into patient management

6. **San Mateo Optometric Society, 2003**
   Uveitic glaucoma

7. **American Academy of Optometry, Tampa, FL, 20004**
   Seeing the whole picture: Ocular clues to systemic disease

8. **San Francisco Optometric Society, 2004**
   Anterior uveitis and the judicious use of steroids

9. **University of California, Berkeley, 2004**
   Optometry Alumni Association Reunion
   Diabetes and the eye: Diagnosis, management strategies, and potential future therapies
10. **American Academy of Optometry, San Diego, CA, 2005**
    Evidenced based medicine

11. **Tri-County Optometric Society, Santa Barbara, CA, 2005**
    Central corneal thickness: Its relationship to IOP and glaucoma

12. **VISN 21 Nurse Practitioners Conference, San Francisco, CA 2005**
    Ocular emergencies

13. **American Academy of Optometry, Denver, CO, 2006**
    Transient ischemic attack

14. **Kentucky Optometric Association, Louisville, KY, 2006**
    Current and future AMD treatments
    Ocular manifestations of systemic disease

15. **Asian American Optometry Study Group, San Francisco, CA, 2006**
    Corneal thickness: What is it telling us?

16. **Vision Expo West, Las Vegas, NV, 2007**
    Evidenced based medicine
    A review of the glaucoma medications
    Central corneal thickness and glaucoma

17. **American Academy of Optometry, Tampa, FL, 2007**
    The dilemma of early glaucoma diagnosis
    Transient ischemic attack

18. **University of California, Berkeley, 2007**
    Meredith Morgan Symposium
    Early glaucoma diagnosis dilemma: Should early diagnosis be followed by treatment?

19. **Northern California Optometric Society, Chico, CA 2007**
    Transient ischemic attack
    Early diagnosis dilemma: Should early diagnosis be followed by treatment?

20. **American Academy of Optometry, Anaheim, CA, 2008**
    Vitreous: Friend or Foe?
    The dilemma of early glaucoma diagnosis

21. **Santa Clara County Optometry Society, 2008**
    Transient ischemic attack

22. **Asian American Optometric Study Group, Berkeley, CA, 2008**
    Transient ischemic attack

23. **University of Alabama, Birmingham, 2009**
    Primary Eye Care Update
    Vitreous: Friend or Foe?
    The dilemma of early glaucoma diagnosis
    Ocular manifestations of systemic disease

24. **American Academy of Optometry, Orlando, FL, 2009**
    Vitreous: Friend or Foe?
    Angle Closure Glaucoma

25. **Kaiser Foundation Optometric Symposium, Anaheim, CA, 2009**
    Transient ischemic attack
    Early glaucoma diagnosis dilemma

26. **Santa Clara County Optometric Society, 2009**
    Ocular manifestations of systemic disease

27. **Northern California Optometric Society, Chico, CA, 2009**
    Vitreous: Friend or Foe?
    Ocular manifestations of systemic disease

28. **American Academy of Optometry, San Francisco, CA, 2010**
    Angle closure glaucoma
    The art of writing scientific abstracts
    The Viagra anterior ischemic optic neuropathy link

29. **Alameda Contra Costa County Optometric Society, 2010**
    Ocular manifestations of systemic disease

30. **Alameda Contra Costa County Optometric Society, 2010**
    Transient ischemic attack

31. **Santa Clara County Optometric Society, 2010**
    Early glaucoma diagnosis dilemma

32. **American Academy of Optometry, Boston, MA, 2011**
    The trabecular meshwork
    The art of writing scientific abstracts

33. **Wyoming Optometric Association, Cheyenne, WY, 2011**
    Angle closure glaucoma
    The vitreous: Friend or Foe
    Ocular manifestations of systemic disease

34. **San Francisco Optometric Society, 2011**
    Challenging cases from SFVA

35. **Bay Area Optometric Societies, San Jose, CA, 2011**
    Tales from the trenches

36. **Southeastern Council of Optometrists (SECO), Atlanta, GA, 2012**
    Talking TIA
    The other glaucoma: Angle closure glaucoma
    Tales from the trenches

37. **American Academy of Optometry, Phoenix, AZ, 2012**
    The trabecular meshwork
    The art of writing scientific abstracts
    Identifying glaucoma progression clinically

38. **Santa Clara County Optometric Society, 2012**
    SFVA grand rounds

39. **Alameda Contra Costa County Optometric Society, 2012**
    Angle closure glaucoma

40. **American Academy of Optometry, Seattle, WA, 2013**
    The cupped disc: Differentiating between glaucoma and compressive optic neuropathy

41. **Vision Expo East, New York, NY, 2013**
    Talking TIA
    The vitreous: Friend or Foe?
    Ocular manifestations of systemic disease

42. **Southeastern Council of Optometrists (SECO), Atlanta, GA, 2013**
    VA eye clinic grand rounds
    Current and future trends in AMD
    Ocular manifestations of systemic disease

43. **Santa Clara County Optometric Society, 2013**
    Lessons learned as a malpractice consultant

44. **Maine Optometric Association, Freeport, ME, 2013**
    The trabecular meshwork
    Lessons learned as a malpractice consultant
    Ocular manifestations of systemic disease
    Talking TIA
    The cupped disc: Differentiating between glaucoma and compressive optic neuropathy

45. **Broward County Optometric Association, Ft. Lauderdale, FL, 2014**
    Ocular manifestations of systemic disease
    VA eye clinic grand rounds

46. **Vision Expo East, New York, NY, 2014**
    Retinal manifestations of systemic disease and drugs
    Talking TIA
    The other glaucoma: Angle closure

47. **San Francisco Optometric Society, 2014**
    Lessons learned as a malpractice consultant

48. **American Academy of Optometry, Denver, CO, 2014**
    Ocular Herpes Management: Beyond HEDS
    OVS author workshop: Preparing a manuscript
    Glaucoma Special Interest Group Roundtable: Angle closure glaucoma

49. **Santa Clara County Optometric Society, 2014**
    Ocular herpes management: Beyond HEDS

50. **Redwood Empire Optometric Society, Petaluma, CA, 2015**
    Ocular herpes management: Beyond HEDS

51. **Southeastern Council of Optometrists (SECO), Atlanta, GA, 2015**
    Talking about TIAs
    The other glaucoma: A closer look at angle closure
    How to avoid a lawsuit
    Breakfast with the experts

52. **Vision Expo East, New York, NY, 2015**
    Enlarged optic nerve cupping: Differentiating glaucoma from compressive optic neuropathy
    Lessons learned as a malpractice consultant
    The other glaucoma: A closer look at angle closure

53. **Vision Expo West, Las Vegas, NV, 2015**
    Enlarged optic nerve cupping: Differentiating glaucoma from compressive optic neuropathy
    Lessons learned as a malpractice consultant
    The other glaucoma: A closer look at angle closure

54. **American Academy of Optometry, New Orleans, LA, 2015**
    Methicillin Resistant Staph Aureus: Ocular manifestations and clinical management

55. **Association of Lease-Holding Lenscrafters Doctors Meeting, Cancun, Mexico, 2015**
    Methicillin resistant Staph aureus: Ocular manifestations and clinical management
    Ocular herpes management: Beyond HEDS

56. **UC Berkeley Optometry Alumni: 65th Annual Alumni CE Program, Berkeley, CA 2015**
    Update on the optometric management of angle closure

57. **Maine Optometric Association, Freeport, ME, 2015**
    Methicillin resistant Staph aureus: Ocular manifestations and clinical management
    Ocular herpes management: Beyond HEDS
    VA Eye Clinic Grand Rounds
    Retinal manifestations of system disease and drugs

58. **San Mateo County Optometric Association, San Mateo, CA 2015**
    Methicillin resistant Staph aureus: Ocular manifestations and clinical management

59. **Santa Clara County Optometric Society, 2016**
    Methicillin resistant Staph aureus: Ocular manifestations and clinical management

60. **San Francisco Optometric Society, 2016**
    Methicillin resistant Staph aureus: Ocular manifestations and clinical management

61. **UC Berkeley School of Optometry: Sheldon M. Golden Conference, Berkeley, CA**
    The use of imaging in the diagnosis and management of glaucoma: Where are we?
    The use of visual fields in the diagnosis and management of glaucoma: Where are we?
    The surgical management of glaucoma: Where are we?
    Glaucoma panel discussion

62. **East West Eye Conference, Cleveland, OH, 2016**
    The early glaucoma diagnosis dilemma
    Enlarged optic nerve cupping: Differentiating glaucoma from compressive optic neuropathy
    The trabecular meshwork: Its role in glaucoma pathogenesis and as a target of therapy
    The other glaucoma: A closer look at angle closure glaucoma
    Methicillin resistant Staph aureus: Ocular manifestations and clinical management
    Ocular herpes management: Beyond HEDS

63. **American Academy of Optometry, Anaheim, CA, 2016**
    Headache disorders that affect the visual system
    Essentials of peer-review and constructive criticism
    Best practices for getting published

64. **Maine Optometric Association, Portland, ME 2016**
    Headache disorders that affect the visual system
    The early glaucoma diagnosis dilemma
    VA Eye Clinic Grand Rounds
    Retinal manifestations of system disease and drugs

INVITED ACADEMIC LECTURES

1. **University of California, Berkeley, 2000**
   Course: Optometry 106B
   Problem based learning facilitator

2. **University of California, San Francisco, 2002-Present (Recurring)**
   Department of Medicine
   Differential diagnosis of the acute red eye
   Differential diagnosis of painless loss of vision
   Slit lamp and direct ophthalmoscopy techniques

3. **University of California, Berkeley, 2002-2005**
   Course: 430
   Glaucoma clinical trials: What they tell us
   Glaucoma management: A literature driven philosophy
   Common and uncommon retinal vascular diseases
   The pupil: Important clinical indicator
   Anterior ischemic optic neuropathy
   Macular degeneration basics
   Glaucoma medication review
   Diabetic retinopathy basics

4. **University of California, San Francisco, 2008**
   Department of Ophthalmology Grand Rounds
   Progressive multifocal leukoenchephalopathy

5. **University of California, San Francisco, 2012**
   Department of Ophthalmology Grand Rounds
   FAA guidelines on reporting visual dysfunction

6. **University of California, San Francisco, 2013**
   Department of Ophthalmology Grand Rounds
   Brimonidine associated uveitis

7. **University of California, San Francisco, 2008-Present (Recurring)**
   Department of Ophthalmology
   Fundamentals of Ophthalmology Course
   Basic refraction and lensometry
   The optics of refraction and retinoscopy
   Introduction to rigid gas permeable contact lenses
   Introduction to hydrogel contact lenses
   Ophthalmic Knowledge Assessment Program (OKAP) Examination Optics Review

8. **University of California, Berkeley 2011-Present (Recurring)**
   Course: 256
   Retinal vascular occlusive disease

9. **University of California, Berkeley, 2014-Present (Recurring)**
   Old Week 2014 Graduating Class Final Review
   Clinical Advice to Avoid Malpractice

10. **University of California, San Francisco, 2014**
    School of Nursing
    Ocular disorders: The red eye

11. **University of California, San Francisco, 2016**
    Department of Ophthalmology Grand Rounds
    Topiramate associated ciliochoroidal effusion angle closure

12. **University of California, Berkeley**
    School of Optometry Grand Rounds Program
    Management of bacterial keratitis in the era of antibiotic resistance

13. **University of Alabama, Birmingham**
    School of Optometry
    Course: 316
    Secondary open angle glaucoma

PROFESSIONAL ORGANIZATIONS

American Academy of Optometry, Fellow, 2003-Present
National Association of VA Optometrists, 2003-Present
American Optometric Association; 2001-2009
Optometric Glaucoma Society, 2013-Present

VOLUNTEER ORGANIZATIONS

Project Homeless Veteran Connect, 2008-2010
Volunteer Optometric Service to Humanity, Costa Rica, Brazil, 2000-2003
Oakland Public Schools, Eyeball dissections in high school science curriculum , 1999-2000

OPTOMETRIC LICENSURE

State of Florida, 2001-2015 (#OPC 3605)
State of California, 2002-Present (#11996TPLG)
State of Idaho, 2015-Present (#ODP-100330)