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: Glaucoma Studies and Their Impact on Clinical Management

Provider Name: Veteran's Administration c/o Dr. Sally Dang

☑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

I-767/383129/439708/SD

BUSINESS, CONSUMER SERVICES, AND HOUSING AGENCY — GOVERNOR EDMUND G. BROWN JR.

OPTOMETRY

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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**
Glaucoma Studies and their Impact on Clinical Management	10/10/2016

Course Provider Contact Information

Provider Name

Sally (First) Dang (Last) H. (Middle)

Provider Mailing Address

Street 5901 E. 7th St. **City** Long Beach **State** CA **Zip** 90822

Provider Email Address Sally.Dang@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

Edward (First) Chu (Last) (Middle)

License Number CA 13585	**License Type** TLG
Phone Number (844) 808.2020	**Email Address** Edward.Chu@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.

Sally Dang — *Signature of Course Provider*

9/1/16 — *Date*

Form CE-01, Rev. 5/16

MAJOR ROB SOLTES

MEMORIAL GOLF TOURNAMENT

February 2017

Dear Board of Optometry,

Please see below for the requested supplemental information to the initial course submission for the annual Major Rob Soltes Memorial Golf Tournament.

Application was submitted on 9/1/16 for 10/10/16 CE event. We will adjust our process to ensure 45 day processing time for future submissions.

Summary of Course Topic:

Landmark glaucoma studies and their impact on clinical management including Ocular hypertension treatment study (OHTS), Early Manifest Glaucoma Trial, Collaborative Normotension Glaucoma Study. Attention to study design, risk factors for progression, and implication of results treatment and management.

Ocular Hypertension Treatment Study: Linking IOP and Onset of Glaucoma

Key Findings: Treatment delays onset of glaucoma. Treating abnormally elevated intraocular pressure (IOP) with topical medications delays or prevents the onset of glaucomatous damage. A second goal of the study was to identify baseline demographic and clinical risk factors for developing primary open-angle glaucoma (POAG).

-Clinical implication: It is possible to separate ocular hypertensive patients into categories of high, medium and low risk.

Early Manifest Glaucoma Trial: Treat IOP Early, Follow Progress Closely

Key Findings: Treatment effect validated. The goals of the EMGT twofold: to compare the effect of IOP-lowering treatment versus observation on the progression of early, newly detected untreated glaucoma and to assess the magnitude of any treatment effect.

- **Clinical implication:** Follow progression closely; reset target as needed.

MAJOR ROB SOLTES

MEMORIAL GOLF TOURNAMENT

September 2016

Dear Board of Optometry,

The annual Major Rob Soltes Memorial Golf Tournament will take place on Monday, October 10, 2016. As in the past 2 years we had continuing education offered for Optometrists.

Last year we had a morning lecture series session, then the golf CE scramble on the golf course (for 7 hrs of CE total). This year, due to time constraints, the format will be similar without morning lecture series.

We are requesting CE approval for 4 hours. The reading material and outline with CV of the speaker is enclosed.

Thank you for your consideration,

Sincerely,

Thomas J. Clarke
Blinded Veterans Association Representative
Operation Peer Support External Advisor
Golf Tournament Chairman

Sally H. Dang, O.D.
Veterans Services Liaison
Operation Peer Support External Advisor
Golf Tournament Board of Directors

Collaborative Normal-Tension Glaucoma Study: IOP Reduction Important Even for Normotensives

Key Findings: IOP plays a role in NTG. Glaucoma progression was slower in the treated group than in the untreated group. This answered the primary question of the trial: Is IOP involved in normal-tension glaucoma?

- **Clinical implication:** Distinguish between progressive and nonprogressive disease.

A 30-item questionnaire will be completed at the end of the course and must be submitted for course credit.

The course topic of "Glaucoma Studies and Their Impact on Clinical Management" reviews landmark studies which have greatly deepened the knowledge of glaucoma. The studies have asked and addressed diagnostic and treatment questions clinicians face each day. The course will highlight the clinical implications and practical usage of each study. Education on pivotal studies that have driven clinical decisions on glaucoma management are fundamental to the practice of optometry.

We are requesting CE approval for 4 hours. The reading material and outline with CV of the speaker is enclosed.

Thank you for your consideration,

Sincerely,

Sally H. Dang, O.D.

Glaucoma Studies and their Impact on Clinical Management
Edward Chu, OD, FAAO

I. Ocular Hypertension Treatment Study (OHTS)

A. Study Design

a. IOP 24-32 mmHg in 1 eye, 21-32 mmHg in other eye
b. Normal visual fields, open angles
c. Treatment goal 20% vs Observation
d. Endpoint of VF defects or optic nerve damage
e. Visual field performed q 6 months, photos q 12 months

B. Results

a. Treatment Group = 4.4% Observation = 9.5%
b. Topical ocular meds delay/prevent glaucoma with IOP between 24-32 mmHg
c. Patients with CCT < 555 um had 3 fold greater risk vs CCT > 588 um
d. Relative risk reduction of treatment was 54%
e. Absolute risk reduction ONLY 5.1%
f. Number need to treat was 20 people vs EMGT (5 people), CNTGS (4 people)

C. Risk Factors for Development of POAG

a. Older age
b. Larger vertical C/D
c. Greater PSD
d. Higher IOP
e. Thin Corneal Thickness – highest risk of conversion was IOP > 26 AND corneas thinner than 555 um (36% of patients converted from ocular hypertension to glaucoma)

D. Treatment/Management

a. Patients with IOP greater than 32 were excluded from observation, need Tx
b. Thin corneas and pressures above 26 mmHg
c. Adjusting IOP for corneal thickness does NOT improve prediction models

II. Early Manifest Glaucoma Trial

A. Study Design

a. Reproducible visual field defects in 1 eye
b. 129 patients w/ 360 laser trabeculoplasty and betaxolol BID
c. 126 patients observed w/o treatment
d. Patient moved to Tx group if progression occurred or IOP > 35, mean IOP > 30

B. Results

a. Average IOP lowered by 5 mmHg (25%) in treatment group
b. VF defects or ON damage at 6 years was 45% in treatment group, 62% observation
c. Progression less frequent in Tx group, occur significantly later, 18 months on average

d. Progression risk cut in half with treatment
e. Number needed to treat to prevent 1 patient from developing glaucoma progression = 5 people

C. Risk Factors for Progression
a. Older age
b. Worse mean deviation on Visual Field
c. Higher baseline IOP
d. Pseudoexfoliation
e. Disc Hemorrhages
f. Bilateral Disease

D. Treatment/Management
a. Natural History, median untreated, normal to blindness in 70 years
b. Natural History, mean untreated, normal to blindness in 25 years
c. Time to progression varies, no standard treatment, take it patient by patient!
d. 50% of patients had IOP below 20 mmHg

III. Normal Tension Glaucoma

A. Study Design
a. 10 baseline IOP measurements, median IOP less than 20 mmHg
b. No IOP readings over 24 mmHg
c. 140 patients randomized: 61 treatment, 79 controls
d. Target 30% reduction in IOP via surgery/medications (CAI, Latanoprost)

B. Results
a. VF progression noted in 12% treatment group, 35% controls
b. Survival time to progression, 7.36 years Tx group vs 4.64 years for controls

C. Risk Factors for Progression
a. Women – link to shorter exposure to estrogen levels
i. 2.6 fold increased risk glaucoma if menopause before 45 vs after 50
ii. > 5 years contraceptive use increase risk 25%
iii. Age of first period > 13 years increase risk glaucoma by 47%
b. H/O Migraines – vasospasm, poor autoregulation of blood flow to nerve
i. Higher association NTG w/ consistent unilateral presentation, accompanying nausea, migraine aura
c. Disc Hemorrhages
d. African American > Caucasian > Asians
e. Low Pressure Glaucoma Treatment Study
i. Systemic Beta Blockers – Perfusion pressure overnight
f. Obstructive Sleep Apnea (OSA)
i. Higher prevalence glaucoma with moderate/severe OSA

D. Treatment/Management
a. Natural History: Broad Spectrum of deterioration, most cases progress slowly
b. 50% eyes show progressive deterioration by years 5-7

c. Only 50% met target IOP reduction of 30%, unreasonable?

IV. Optic Nerve Hemorrhages

A. Background

a. Splinter or flame shaped hemorrhages at optic nerve border

b. Radially oriented and perpendicular to disk margin

c. Most common in low tension glaucoma, but also seen in POAG, Ocular Hypertension

d. Warning sign that eye at risk for developing glaucoma or having progression of glaucomatous damage

B. Pathophysiology

a. Vasculopathic event leading to NFL loss

b. Degeneration of tissue from stress on microvasculature

C. Natural History

a. Resolve in 2-3 months

b. Tend to recur in same region with corresponding visual field defect

c. Common in early and moderate glaucoma

D. Clinical Management

a. Ocular Hypertension Treatment Study:

i. 6 times more likely convert glaucoma over 1 year

ii. However, 87% did not convert from ocular hypertension to glaucoma over 3 years

iii. 84% photographed disk hemes missed during dilated exam by OMD

b. Early Manifest Glaucoma Trial

i. Disc hemes cannot be considered indication of insufficient IOP lowering

ii. Occur equally between treated and non-treated

c. Collaborative Normal Tension Glaucoma Study

d. Visual field progression with heme vs without heme

e. Recurrent heme vs single occurrence

V. Auxiliary Testing

A. Visual Field: Diagnosis of Glaucoma

a. Glaucoma Hemifield Test (GHT) outside normal limits on 2 consecutive fields (Early Manifest Glaucoma Trial, Hodapp/Anderson/Parrish)

b. GHT outside normal limits on 3 consecutive fields (Ocular Hypertension Treatment Study)

c. 3 visual fields x 3 times in 1 month (Collaborative Normal Tension Glaucoma Study)

B. Visual Field: Progression of Glaucoma

a. 3 same progressing points x 3 consecutive fields (EMGT)

b. OHTS – 86% 2nd visual field tests failed to confirm abnormality on baseline VF, need to retest before treatment!

C. OCT: Database
    a. Stratus 328 subjects, only 8% African American, mean age 47 years
    b. Cirrus 284 subjects, only 18% African American, only 3 patients older than 80
D. OCT: Diagnosis of Glaucoma
    a. Interocular difference of average RNFL thickness > 9 um indicative of early glaucoma damage

Glaucoma Studies and their Impact on Clinical Management

1. Inclusion criteria for the **Early Manifest Glaucoma Trial** included which of the following?
    a. Those with previously treated glaucoma
    b. Patients with advanced visual field defects
    c. Men and women age 50-80
    d. Acute angle closure patients

2. Which of the following is true regarding the **Early Manifest Glaucoma Trial**?
    a. Progression risk decreased by half in treated patients vs control patients
    b. Progression decreased with higher baseline IOP
    c. A single glaucomatous visual field was necessary for glaucoma diagnosis
    d. Previously treated glaucoma patients were eligible for the study

3. Results of the **Early Manifest Glaucoma Trial** revealed:
    a. Average IOP lowered by 5mmHg in treatment group
    b. Glaucoma progression did not differ between treatment and control group
    c. Treatment effects present only in patients with IOP over 25mmHg
    d. IOP reduction failed to maintain through post treatment follow up

4. The **Ocular Hypertension Treatment Study** concluded:
    a. Having thinner corneal hysteresis increases the risk of developing primary open-angle glaucoma
    b. Lowering IOP in ocular hypertensive patients did not effectively delay glaucoma onset
    c. All participants with IOP over 25mmHg will eventually develop glaucomatous damage if left untreated
    d. During the five-year study, IOP reduction similar in the medication group and the observation group

5. What percentage of the glaucoma patient population have normal tension glaucoma?
    a. 3%
    b. 10%
    c. 30%
    d. 50%

6. Appropriate treatment for normal tension glaucoma include:
    a. Topical ocular hypotensive medication
    b. Oral carbonic anhydrase inhibitor
    c. Oral beta blocker
    d. Topical steroid

7. Which of the following describes the **Collaborative Normal-Tension Glaucoma Study**?
    a. By decreasing IOP by 30%, glaucoma progression could be reduced by 50%
    b. Increased cataract development was observed in patients treated with glaucoma surgery
    c. The main outcome measure was visual field progression from baseline
    d. All of the above

8. Which of the following is/are potential contributors to normal tension glaucoma?
    a. **Low optic nerve perfusion pressure**
    b. High blood pressure
    c. High cerebrospinal fluid pressure
    d. All of the above

9. The **Collaborative Normal Tension Study** Group revealed:
    a. IOP reduction did not decrease disease progression
    b. **Females with history of migraines have great risk of developing rapid visual field deterioration**
    c. Achieving 30% IOP reduction prevented further progression of glaucomatous damage
    d. Timolol is more effective than Brimonidine at preventing visual field progression

10. All of the following increase risk factors for normal tension glaucoma EXCEPT:
    a. Migraine
    b. Sleep apnea
    c. Disc hemorrhage
    d. **Hypertension**

11. What was the target IOP lowering in the **Collaborative Normal Tension Glaucoma** study?
    a. 20%
    b. 25%
    c. **30%**
    d. 35%

12. Which is true for patients with normal tension glaucoma?
    a. They should always be treated with IOP lowering agents
    b. Visual field loss will not progress if treated early
    c. **Treatment should be individualized according to stage of disease**
    d. Always consider trabeculoplasty in conjunction with topical IOP lowering agents to maximize IOP reduction

13. Which of the following is seen more often in normal tension glaucoma vs primary open angle glaucoma?
    a. **Optic disc hemorrhages**
    b. Superior/inferior arcuate defects
    c. Narrow angles
    d. Higher ocular perfusion pressure

14. Faster rate of normal tension glaucoma progression occurs in
    a. **Women**
    b. Patients over 50 years of age
    c. Patients with systemic hypertension
    d. All of the above

15. Glaucomatous visual field damage can be made using all of the following measures EXCEPT:
    a. Glaucoma hemifield test
    b. Pattern standard deviation
    c. Mean deviation
    d. Point-wise analysis of the pattern deviation plot

16. A modifiable risk factor for glaucoma can include:
    a. Intraocular pressure
    b. Diet
    c. Blood glucose control
    d. Blood pressure control

17. When would a detailed workup including neuroimaging be indicated?
    a. visual field defects that respects the vertical rather than horizontal midline
    b. optic nerve pallor greater than cupping
    c. decreased central visual acuity (< 20/40)
    d. all of the above

18. Target intraocular pressures in patients with primary open angle glaucoma is determined by:
    a. Pretreatment pressure levels associated with optic nerve damage
    b. Life expectancy
    c. Risk factors for progression
    d. All of the above

19. Which should NOT be used in patients with a sulfa allergy?
    a. Latanoprost
    b. Brimonidine
    c. Dorzolamide
    d. Timolol

20. The **Early Manifest Glaucoma Trial** concluded:
    a. Elevated IOP is the primary cause of glaucoma
    b. IOP reduction slows glaucoma progression
    c. Target IOP should always be set at 30%
    d. Glaucoma damage occurs equally between treated and untreated patients

21. When choosing a second- line treatment to add to a prostaglandin it is important to consider:
    a. How much it will affect the IOP
    b. Patients willingness to adhere to the medication regimen
    c. Cost to the patient
    d. All of the above

22. Benefits of Alpha Agonists include:
    a. Low allergic response rate

b. **Possibly neuroprotective**
c. Increases perfusion pressure
d. Once-daily dosing

23. Which is true regarding early stages of glaucoma?
    a. Visual fields are the best detector of damage
    b. **OCT gives good assessment of structural damage**
    c. Combination therapy should be considered first line
    d. Visual field defects appear in the temporal quadrants

24. According to the **Ocular Hypertension Treatment Study** a good predictor for onset of primary open angle glaucoma is:
    a. Family history
    b. Race
    c. **Central corneal thickness**
    d. Positive history of ocular trauma

25. According to the **Ocular Hypertension Treatment Study** which of the following patients should have treatment initiated?
    a. Two consecutive IOP readings over 25mmHg
    b. **Ocular hypertension with moderate or high risk for developing glaucoma**
    c. Evident retinal nerve fiber layer thinning on OCT
    d. Ocular hypertension with central corneal thickness greater than 550 microns

26. The **Ocular Hypertension Treatment Study** revealed:
    a. **There is little absolute benefit of early treatment in low risk individuals**
    b. Caucasians develop POAG at a higher rate despite similar treatment
    c. Incidence of POAG is 30% lower in the treatment group
    d. Positive family history was the greatest predictive factor for glaucoma developement

27. Risk factors for primary open angle glaucoma include:
    a. Asian race
    b. Pressures over 18mmHg
    c. Small C/D ratio
    d. **Elevated IOP fluctuation**

28. Which of the following antiglaucoma agents work by increasing uveoscleral outflow?
    a. **Latanoprost**
    b. Cosopt
    c. Timolol
    d. Dorzolamide

29. Goals of primary open angle glaucoma management include all of the following except:
    a. Control IOP in target range
    b. Stable optic nerve/ retinal nerve fiber layer status
    c. Keep IOP below 15 in both eyes
    d. Stable visual fields

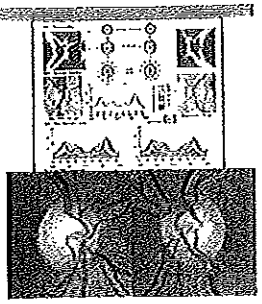
30. Which of the following is TRUE with visual field evaluation and primary open angle glaucoma?
    a. Manual kinetic perimetery is the preferred technique
    b. Complete arcuate defects are signs of early visual field changes
    c. The central 20 degree test on Frequency doubling technology (FDT) can be used to screen for visual field defects
    d. Changing test protocols when repeating visual fields can be useful

Glaucoma Studies and their Impact on Clinical Management

Dr. Edward Chiu, OD, FAAO
Long Beach VA - Residency Coordinator
MBKU - Assistant Professor
Treatment and Management of Ocular Ds

Case

- 70 yo caucasian male
- IOP 26/28 @ 0932
- Pachymetry 520 um OU
- Gonioscopy: CB 360 OU w/ 1+ pigment, no PAS/NVA/AR
- Humphrey VF – no glaucomatous clusters



With IOPs of 26/28 and thin corneas, what is your treatment plan?

1. Monitor without treatment, RTC 3 months IOP check
2. Start IOP lowering medication and follow in your clinic
3. Start IOP lowering medication and refer to OMD
4. Consult with a colleague and follow their recommendation
5. Refer to Ophthalmology
6. Flip a coin

Would your plan change....

- Patient was monocular?
- Pachymetry of 600 um OU?
- Patient was 55 years old? 85 years old?
- Positive Family H/O Glaucoma?
- African American?
  - Higher IOP, larger C/D, thinner corneas

The Ocular Hypertension Treatment Study

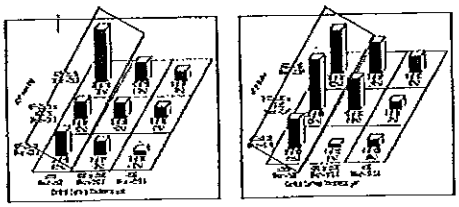
- Question: Should patients be treated? Is it safe and effective to treat them with IOP lowering meds?
- Criteria
  - Normal visual fields, Open Angles
  - IOP
    - 24-32 mmHg in 1 eye, 21-32 mmHg in other eye
  - 1636 patients randomized to Tx (goal to reduce IOP by 20% or more) vs observation
  - Endpoint of VF defects or optic disc deterioration
    - VF performed q6months, photos q12 months

OHTS Findings

- Chances of developing glaucoma
  - Treatment group = 4.4%
  - Observation = 9.5%
- Topical ocular medications effective in delaying or preventing onset POAG w/ IOP between 24 and 32
- Patients w/ CCT < 555 had 3 fold greater risk of POAG vs patients with CCT > 588

OHTS

- Risk factors for development of POAG
  - Older Age
  - Larger vertical C/D
  - Greater PSD
  - High IOP
  - Thin Cornea



Glaucoma risk highest among participants w/ THINNEST central corneal thickness

OHTS 5 year Risk of POAG

	A < 555 um	555 < A < 588 um	A > 588 um
IOP > 25.75	36%	13%	6%
IOP between 23.75 and 25.75	12%	10%	7%
IOP < 23.75	17%	9%	2%

To treat or not to treat....

- What amount of damage greatly compromises the patient's life?
- Is it better to watch an ocular hypertensive patient without treatment and only treat when patient converts to definite glaucoma?
- Remember to consider
  - Cost of drops
  - Side effects
  - Inconvenience of drops every day for the rest of your life

Treat Ocular HTN?

- Treatment group = 4.4%
- Observation = 9.5%
- Relative Risk Ratio = .44/.95 = .46
- Relative Risk Reduction w/ Tx = 100-46% = 54%
- Absolute risk reduction (9.5% - 4.4% = 5.1%)
- Number needed to treat is inverse of AR reduction
  - 1/.05 = 20 people

What does that mean?

- Therefore, 20 ocular hypertensive patients need to be treated as they were in the OHTS study to prevent 1 OHTS patient from developing glaucoma over 5 years
- Compare
  - Early Manifest Glaucoma Trial (3 people)
  - Collaborative Normal Tension Glaucoma Trial (4 people)
- OHTS: 87% drance hemes DO NOT convert from ocular hypertension to glaucoma over 31 months

Treatment Criteria

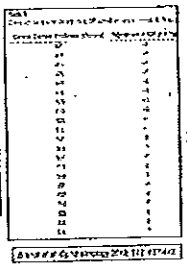
- Pressures above 30
  - Patients with IOP greater than 30 were excluded from Early Manifest Glaucoma Study
  - IOP greater then 32 excluded from the OHTS study
- Thin corneas and pressures above 26
  - 26% chance of developing glaucoma

Adjusting Intraocular Pressure for Central Corneal Thickness Does Not Improve Prediction Models for Primary Open-Angle Glaucoma

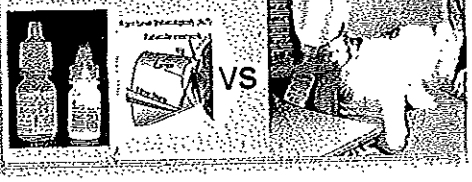
1433 patients from OHTS

Analysis of predictive value of 6 different correction formulas for unadjusted IOP

The calculation of risk for POAG in OH is simpler and equally accurate using IOP and CCT AS MEASURED



Early Manifest Glaucoma Trial



Reduction of Intraocular Pressure and Glaucoma Progression

Results from the Early Manifest Glaucoma Trial

- Question: Does Immediate lowering of IOP affect the progression of NEWLY detected POAG?
- Criteria: reproducible glaucomatous VF defects in at least 1 eye
  - 255 patients
    - 129 Patients 360 laser trabeculoplasty and betaxolol BID
    - 126 observed with no treatment
    - Observe → Tx Group if significant progression occurred or pressure > 35 mmHg, mean IOP > 30

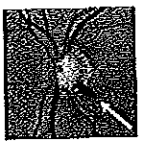
EMGT Results

- Average IOP lowered by 5.1 mmHg (25%) in Tx
- Development VF defects or ON progression at 6 years: Treatment 30% vs Observation 49%
- Progression *less frequent* in Tx group, occur *significantly later (18 months)*
  - 53% patients progressed during study, progression risk cut in half with treatment
  - Progression linked to magnitude initial IOP reduction

Factors for Glaucoma Progression and the Effect of Treatment

The Early Manifest Glaucoma Trial

- Treatment groups 1 mmHg reduction in IOP resulted in a 10% reduction of progression
- Factors that predicted progression
  - Older age
  - Worse MD on VF
  - Higher baseline IOP
  - Pseudoexfoliation
  - Disc hemorrhages
  - Bilateral Disease



Natural History of Open-Angle Glaucoma

Followed UNTREATED patients from EMGT for 6 years
- 45 High Tension Glaucoma
- 57 Normal Tension Glaucoma
- 15 PEX Glaucoma

Progression vs Non-Progression, Median Time to Progression

HTG	21%	41.8 months
NTG	56%	61.8 months
PEXG	93%	19.5 months

68% overall showed definite VF progression
- MEDIAN untreated progression = normal to blindness in 70 years
- MEAN untreated progression = normal to blindness in 23 years

EMGT

- Number needed to treat 5
  - We need to treat 5 newly diagnosed glaucoma patients to reduce the risk of 1 of them continuing to lose VF over 4 years of follow-up
- Endpoint OHTS and EMGT was VF loss, not blindness and not interference w/ activities of daily living
- Study took place in Sweden
  - No African Americans, Asians, or Hispanics
- 50% patients had IOPs below 20 mm Hg

EMGT Pearls

- Time to progression varied greatly among treated and untreated patients
- "Standardized treatment was insufficient in many rapidly progressing patients"
- **Take it patient by patient!**
  - LTG progresses slower
  - PEX progresses faster

Factors for Glaucoma Progression and the Effect of Treatment

Progression of Glaucoma Increased
- Higher baseline IOP
- Bilateral Disease
- Worse NO
- Older Age
- Frequent Disk Hemes on FU
- PSEUDOEXFOLIATION

PEX protect < 10% EMGT patients ... in Sweden?

83% patients w/ PEX at baseline progressed

Risk of Glaucoma in Ocular Hypertension with and without Pseudoexfoliation

Cohort study: 32,918 participants from Sweden (EMGT)

All w/ Ocular Hypertension: IOP 24-32 mmHg
98 patients w/ PEX, 98 patients w/o PEX

65.1% PEX vs 27.6% w/o PEX → Glaucoma
Risk ratio 2.0 over 9 years

Recommend treatment of PEX-OHT

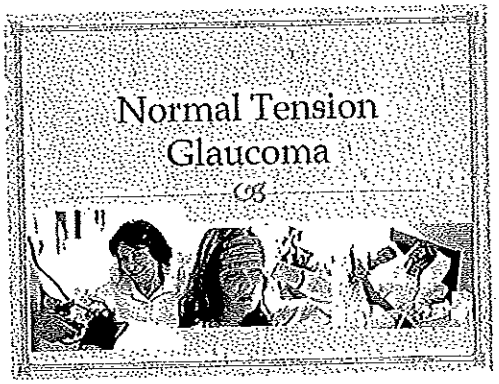
Natural History of Intraocular Pressure in the Early Manifest Glaucoma Trial

Median IOP 20.8 mmHg vs 24.0 mmHg PEX patients

IOP stable -0.01 mmHg/year vs 0.96 mmHg/year PEX patients
1 mmHg → 13% increased risk progression

ONLY factor related to IOP change → Pseudoexfoliation more serious course, worse prognosis

Normal Tension Glaucoma



Background

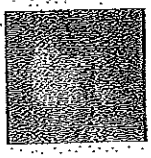
- 1984 – Glaucoma research foundation of SF
  - Can glaucoma occur with a pressure that was not above the statistically normal range?
- Conventional wisdom – normal IOP should not be harmful
  - Was this an optic neuropathy that looks like glaucoma but is unrelated to IOP?
- Practical question – If it looks like glaucoma but the pressures are normal, should we treat? Is it even beneficial?

What IOP qualifies as "Normal" or "Low" when you diagnose NTG/LTG?

1. 24 mmHg and below
2. 22 mmHg and below
3. 20 mmHg and below
4. 18 mmHg and below
5. 16 mmHg and below
6. 14 mmHg and below

CNTGS, LPGTS

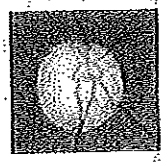
- 10 baseline IOP measurements
  - Median IOP of 20 mmHg or less
  - No readings > 24
  - IOP always under 21



Comparison of Glaucomatous Progression Between Untreated Patients With Normal-Tension Glaucoma and Patients With Therapeutically Reduced Intraocular Pressures

COLLABORATIVE NORMAL-TENSION GLAUCOMA STUDY GROUP

- Objective: role 30% IOP reduction progression of visual changes in NTG
- 140 patients
  - 61 treatment, 79 observed
  - Treatment (Target 30% reduction via surgery/meds)
    - CAI, latanoprost; No B blockers or Alpha Agonists



Am J Ophthalmol 1998; 126: 487-497

Comparison of Glaucomatous Progression Between Untreated Patients With Normal-Tension Glaucoma and Patients With Therapeutically Reduced Intraocular Pressures

COLLABORATIVE NORMAL-TENSION GLAUCOMA STUDY GROUP

VF progression	12%	35%
Survival time to endpoint	7.36 years	4.64 years
Cataract Development	35%	14%

Am J Ophthalmol 1998; 126: 487-497

Natural History of Normal-Tension Glaucoma

Collaborative Normal-Tension Glaucoma Study Group

About 50% eyes show progressive deterioration 5-7 years
MOST cases progress slowly, small change

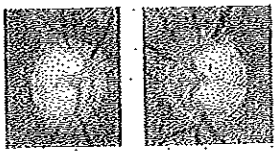
BROAD SPECTRUM rates of deterioration

Immediacy + Aggressiveness of NTG therapy, guide by
1) Stage of disease at presentation
2) Expected rate of natural decline w/o treatment

Ophthalmology 2001; 108: 1767-1776

CNTGS

- Drance et al
- *Might be prudent NOT to Tx most patients with NTG until rate of disease in particular individual has been established over period of observation.*



CNTGS

Conclusions:
IOP lowering 30% beneficial in NTG

Only 50% met target IOP (30% reduction) in study → unreasonable target?

Factors for faster progression of NTG
- Women
- Migraines
- Disc Hemorrhages
- Race (Blacks > Whites > Asians)

Why Women?

- Estrogen stimulates and enhances blood flow
- Postmenopausal women lower blood velocity, higher vascular resistance
- Cycles of hormonal change

Increased risk of POAG in women with *early* menopause
2.6 fold increased risk if occurred before age 45 vs after 50

Shorter estrogen exposure might be associated with POAG

Nurses' Health Study

- 79,410 women followed 1980-2006
- > 5 years of oral contraceptive (25% increase)
  - Maintain steady estrogen/progesterone
  - Inhibit FSH and LH
  - Prevent secondary surge of estrogen/progesterone
- Age of first period > 13 years old (47% increase)

- Theory: circulating estrogen contributes to glaucomatous process

Why Migraines?

- Migraines – associated w/ vasospasm
- Transient cerebral vasospastic episodes
  - H/O silent cerebral infarct in migraine and low pressure glaucoma patients
- Autoregulation *reduced or absent*
- Impairment blood supply when PP low
  - Aura coincides w/constriction of BV
  - Decreased blood flow during aura
  - After aura, blood vessels then dilate

Migraines

- Higher association with LTG
  - Consistent unilateral presentation
  - Accompanying nausea
  - Migraine Aura
- 44% low pressure glaucoma (+) migraines
- Ocular vasospasm can cause VF defects


Risk Factors for Optic Disc Hemorrhage in the Low-Pressure Glaucoma Treatment Study

Patients all with untreated IOP always 21 or less
Randomized to Alphagan 0.2% or Timolol 0.5%

History of Migraine	HR = 5.73
Narrow Neuroretinal Rim	HR = 2.91
Systemic Beta blockers	HR = 5.58

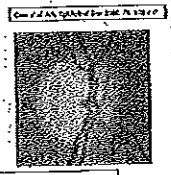
Obstructive Sleep Apnea

- Pauses in breathing during sleep
- Characterized by:
  - Snoring
  - Restless sleep
  - Daytime sleepiness
- 40% obese individuals suffer from OSA
  - fat tissue in neck
  - large tonsils or tongue constrict airway

OSA and Glaucoma

212 POAG, prevalence sleep-disordered breathing 47.6%

69 patients with OSA, 7.2% prevalence of glaucoma compared to 2% in general population



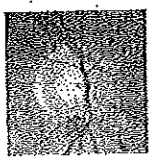
209 OSA patients, 7.1% prevalence of NTG
-higher prevalence moderate/severe cases OSA

OSA and Glaucoma: Vascular

- Disrupted autoregulation of blood flow – inability to change flow w/ demand
  - Hypoxia
  - Hypercapnia - excessive CO_2
- Disrupted blood flow – LESS blood
  - Hypotension during apneas
- Direct hypoxic injury – LESS O_2 in blood
  - O_2 desaturation due to apneas
- All cause ischemic damage to the nerve

Continuous Positive Airway Pressure (CPAP) used for moderate to severe sleep apnea
-keeps airway open during sleep
-prevents apneas, reduces snoring

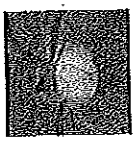
Does CPAP therapy have any effect on IOP and glaucoma?



Continuous Positive Airway Pressure Therapy Is Associated with an Increase in Intraocular Pressure in Obstructive Sleep Apnea

CPAP additional IOP increase, especially w/ mask on

24-hour IOP fluctuations
6.7 mmHg at baseline vs 9.0 mmHg during CPAP



Statistically significant drop in mean IOP found just 30 minutes after CPAP withdrawal (20.8 mmHg to 18.6 mmHg)

OSA in your exam chair

- Glaucoma patients or suspects, perform thorough medical history to illicit history of OSA and CPAP treatment
- Floppy Eyelids
- Ask appropriate questions
  - Sleeping problems?
  - Daytime drowsiness?
  - H/O Snoring?

- CPAP safest and most effective Tx, not a cure

Diastolic Perfusion Pressure (DPP)

- Low perfusion of optic nerve as cause of glaucomatous changes
- Combination of drop in BP and increase IOP in PM
- Critical value for DPP is around 50-55 mmHg
  - Diastolic – IOP < 50, higher risk for low perfusion
  - Diastolic – IOP < 30, definite ischemia

Risk Factors for Incident Open-angle Glaucoma

The Barbados Eye Studies

Low DPP (< 53 mm Hg) 3 times increased risk developing OAG
Mean DPP: 63.2 mmHg healthy vs 53.8 mmHg glaucoma

Hypertension, Perfusion Pressure, and Primary Open-angle Glaucoma

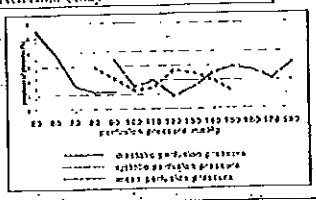
A Population-Based Assessment

Diastolic Perfusion Pressure ≤ 30 mm Hg
Risk of POAG 6 times higher vs DPP > 50 mm Hg

Vascular Risk Factors for Primary Open Angle Glaucoma

The Egna-Neumarkt Study

4297 patients rural Italy

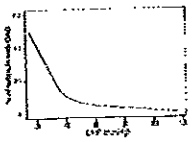


Lower DPP (< 68 mm Hg) associated marked, progressive increase frequency glaucoma

Distribution of Ocular Perfusion Pressure and Its Relationship with Open-Angle Glaucoma: The Singapore Malay Eye Study

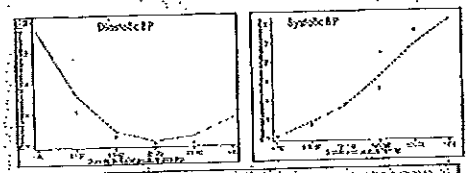
3261 Ethnic Malays
2.5% OAG Malays 40 - 80 years old
Mean IOP 15.3 mm Hg
ONLY 17% IOP > 21

Low DBP and DPP (<56 mmHG) OAG risk factors



Vascular Mechanism in Glaucoma

Los Angeles Latino Eye Study



Both LOW DIASTOLIC and HIGH SYSTOLIC BP increased prevalence OAG

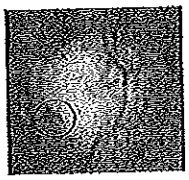
Patients at both BP extremes spectrum greater risk

Perfusion Pressure

- Ask blood pressure medication(s), time taken
- Evening BP medications + drop in BP during sleep
  - IOP increase supine position (Diastolic - IOP = DPP)
  - Consult PCP if patient on multiple BP medications, has low DPP and Glaucoma
- Diurnal IOP HIGHEST after awakening

Case

- 75 year old Caucasian male w/ low tension glaucoma
- Travatan Z qhs OU, good compliance
- Pre Tx- average 18 mm Hg
- Post Tx- average 12 mm Hg
- Last VF 2 months prior
- Today's examination
  - IOP 13/13 @ 9:00 AM

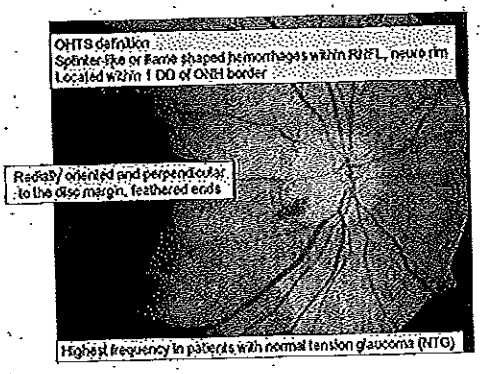


Management plan?

A. Add Alphagan TID
B. Add Timolol BID or Timoptic XE qAM
C. Add Dorzolamide BID
D. Add Cosopt BID
E. Add Simbrinza TID
F. Add Combigan BID
G. No change to treatment, update VF

Drance Hemes

- Warning sign for either developing glaucoma or progression of glaucomatous damage
- Infero-temporal and supero-temporal, areas most susceptible to damage
- VF deterioration corresponding to heme
- Shorter time to visual field progression compared to individuals w/o disc heme

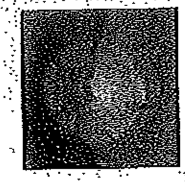


Reported Rates

- Normal patients 0-0.4%
- Ocular Hypertension 0.4-10%
- Primary Open Angle Glaucoma 2-37%
- Normal Tension Glaucoma 11-12%
  - Bilateral, more likely recur
- More common early/moderate glaucoma
- Less common in advanced disease

Pathophysiology

- Vascular event → NFL loss
- Degeneration of tissue → stress on microvasculature
- Poor vascular autoregulation
- 100% of eyes w/ subsequent DH first developed focal rim notch in area future DH

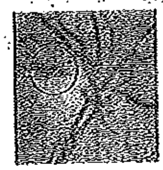


Optic disc hemorrhages in glaucoma and ocular hypertension: implications and recommendations

Most disc hemorrhages disappear/resolve within 2 months

Associated with RNFL thinning, rim notching, peripapillary atrophy

Despite Tx and lower IOP, eyes that bleed tend to re-bleed



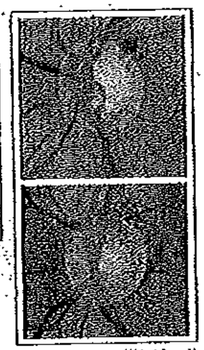
In the Ocular Hypertension Treatment Study, what % of photographed drance hemorrhages were actually detected by OMDs during the live dilated exam?

- 0%
- 15%
- 30%
- 50%
- 75%
- 95%

OHTS Group

-16% photographically documented disc hemorrhages detected with DFE

84% photographically documented disc hemorrhages MISSED during DFE

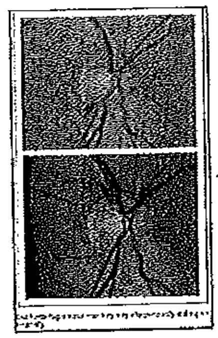


OHTS Group

Develop POAG endpoint 6 times more likely patients with DH
-median 13 months

86.7% eyes w/ DH did not convert to POAG at end of study over 31 months

To treat or not to treat....No glaucoma in short term



OHTS: Drance Hemes

- Incidence 0.5% per year PRIOR to POAG
- Incidence 2.5% per year AFTER develop POAG
- *POAG patients more DHs vs Ocular HTN*
- Frequency DH similar between treated vs untreated
- Don't treat "Disk Hemes", treat Glaucoma!

Disc Hemorrhages and Treatment in the Early Manifest Glaucoma Trial

Disc hemorrhages EQUALLY common treated vs non-treated

Frequency of disc hemes no differ between treated vs non-treated

IOP-reduction UNRELATED to presence/frequency of disc hemes

Disc Hemorrhages CANNOT be considered indication insufficient IOP lowering

EMGT Disk Hemes

- Confirmed that disc hemes sign of glaucoma damage
- But *"offered with CAVEAT that our report is based on clinical assessment of disc hemorrhages"*
- Subject to inter-observer variation
- Lead to "considerable underascertainment" vs standardized photos
- Frequent DHs at f/u conferred worse prognosis

Recurrent disc hemorrhage does not increase the rate of visual field progression

[redacted]

117 patients divided into 2 groups, followed 4.6 +/- 2.2 years
A: single DH over course of study
B: At least one recurrence of DH

Mean number of visual fields after initial DH was 8 +/- 3

Recurrent DH does NOT result in faster rate VF progression vs single detected DH

Once optic nerve experiences bleeding → greater risk VF progression regardless of whether or not the hemorrhage recurs

[redacted]

Factors Affecting Rates of Visual Field Progression in Glaucoma Patients with Optic Disc Hemorrhage

76 eyes enrolled, photos q 6 months, mean f/u 4 years
- Baseline IOP when DH detected: 16.6 mmHg
- Baseline VF MD when DH detected: -5.6 dB

Ipsilateral DH recurrence observed 21% eyes, fellow eye 23%

Baseline worse than -4.0 dB ~ 2.9X increased risk fast rate progression vs better than -4.0 dB

Presence DH older subjects with worse VF, predicted future greater VF global MD deterioration

[redacted]

Management of DHs

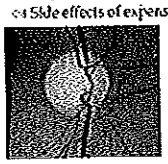
- Louis R. Pasquale, Harvard Medical School, Glaucoma Service Mass Eye and Ear
- IOP of 16 mmHg
  - Photos available?
  - If previous hemorrhage seen, look at VF over time
  - If no progression, level of concern lower
  - If VF progression, change target IOP
- IOP of 12?
  - No good answers
  - Treatment may be worse than disease

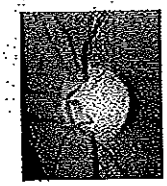
Glaucoma Update

- Louis R. Pasquale, Harvard Medical School, Glaucoma Service Mass Eye and Ear
- Overall goal of glaucoma management
  - Keep patients functional (perform ADL)
  - NOT to save every ganglion cell
- Natural History of Glaucoma
- If patient symptomatic (VF loss) → disease is fairly advanced, treat aggressively

Treatment Pearl

- If patient's risk unknown, consider observation
- Pre-emptive treatment
  - Not cost effective
  - May not help
  - Side effects of expensive therapy





Baseline IOP

- OHTS, CNGTS, EMGT, AGIS all with same finding
  - Only Tx for Glaucoma is lower IOP
- Baltimore Eye Survey
- 50% glaucoma patients IOP < 21mmHg 1st visit
  - Need multiple measurements
  - At least 3 baseline readings
    - Mix morning and afternoon readings if possible

Staging each eye for glaucoma damage

Suspect	One or two of the following: IOP >21 mm Hg; suspicious disc (or C/D asymmetry of >0.2; suspicious 24-2 (or similar) VF defect
Early	Early glaucomatous disc features (e.g., C/D* <0.65) and (or) mild VF defect not within 10° of fixation (e.g., MD better than -6 dB on HVF 24-2)
Moderate	Moderate glaucomatous disc features (e.g., vertical C/D* [0.7-0.85) and (or) moderate VF defect not within 10° of fixation (e.g., MD from -6 to -12 dB on HVF 24-2)
Advanced	Advanced glaucomatous disc features (e.g., C/D* >0.9) and (or) VF defect within 10° of fixation† (e.g., MD worse than -12 dB on HVF 24-2)

[redacted]

Canadian Ophthalmological Society evidence-based clinical practice guidelines for the management of glaucoma in the adult eye. Can J Ophthalmol 2009;44(Suppl 1):S1-S33.

TARGET IOP: Suggested *upper limit of initial* target IOP for each eye

Stage	Suggested upper limit of target IOP. Modify based on longevity, QOL and risk factors for progression	Evidence
Suspect (no/min clinical decision to treat)	21 mm Hg with at least 20% reduction from baseline	OHTS, EGPS
Early	18 mm Hg with at least 25% reduction from baseline	EMGTS, CNGTS
Moderate	17 mm Hg with at least 30% reduction from baseline	CNTGS, AGIS
Advanced	14 mm Hg with at least 30% reduction from baseline	AGIS, Opinion

[redacted]

Canadian Ophthalmological Society evidence-based clinical practice guidelines for the management of glaucoma in the adult eye. Can J Ophthalmol 2009;44(Suppl 1):S1-S33.

VF testing confirm glaucoma vs progression

- Determining earliest glaucomatous VF defects

GHT outside normal limits on 2 consecutive fields
- Hodapp, Anderson, Parrish
- Early Manifest Glaucoma Trial

GHT outside normal limits on 3 consecutive fields
- Ocular Hypertension Study

Glaucoma confirmed 3 VF 3 times w/in 1 month
- Collaborative NTG Study

Progression: 3 same progressing points x 3 consecutive VFs
- Early Manifest Glaucoma Trial

Confirmation of Visual Field Abnormalities in the Ocular Hypertension Treatment Study

85.9% 2nd VF test FAILED to confirm abnormality from baseline VF

Reliability: 33% False Positive/False Negative/Fixation Loss

Considerable variability Ocular HTN patients w/ early glaucoma VF loss

High % of first abnormal VFs found normal on retest. Recommend 3 VFs to confirm same defect

Glaucoma: OCT

- Age-matched norms
- Superior/Inf. Thinning
- Reduced average thickness
- Correlate to optic nerve
- Symmetry

White = above average

Yellow = borderline

How many "norms" in database? Race?

How many "norms" in OCT database?

- A. 100
- B. 300
- C. 1000
- D. 5000
- E. 10,000
- F. 50,000

Stratus Database

- 328 subjects
- 48% male, 52% female
- Mean age 47.4 +/- 15.8 yrs, range 18-85
- Rx: -11.75 to +6.75, mean -0.54
- 63% Caucasian, 24% Hispanic, 8% African American, 11% Asian
- No eye surgery except cataract (9 pts), no ocular disease, IOP <22, normal and reliable VF, normal ONH, BCVA >20/32

Slide courtesy of Dr. David Hicks

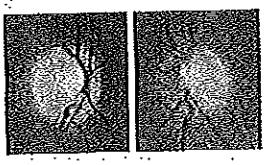
Cirrus Database

- 284 subjects
- 47% male, 53% female
- Age range 19-84
  - Only 3 pts >80 and 28 pts between 70-79
- Rx: -12 to +8
- 43% Caucasian, 18% African American, 12% Hispanic, 1% Indian, 6% mixed
- All normal subjects

Slide courtesy of Dr. David Hicks

Interocular Asymmetry

- C/D Ratio and/or IOP asymmetry
  - Early signs glaucoma
  - Predictor of future damage in ocular HTN
  - Related to glaucomatous VF loss



The Value of Intraocular Pressure Asymmetry in Diagnosing Glaucoma

326 controls, 326 glaucoma patients
-ethnicity diverse, age and sex matched

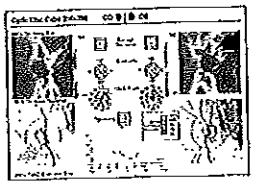
Absence of IOP asymmetry	1% probability develop glaucoma
Difference of > 3 mmHg	6% probability develop glaucoma
Difference of > 6 mmHg	57% probability develop glaucoma

Likelihood of having POAG increases as intereye IOP asymmetry increases

Interocular Symmetry in Peripapillary Retinal Nerve Fiber Layer Thickness Measured With the Cirrus HD-OCT in Healthy Eyes

RNFL early GLC damage, may occur before structural change and VF loss

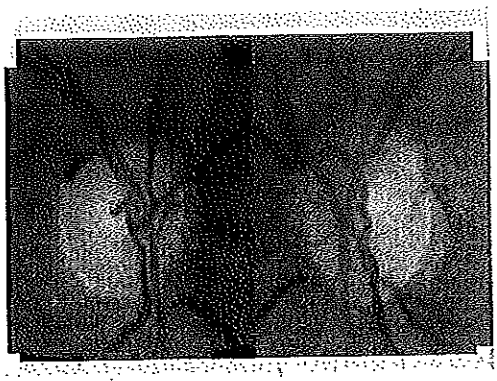
Interocular difference ave RNFL thickness exceeded 9 um

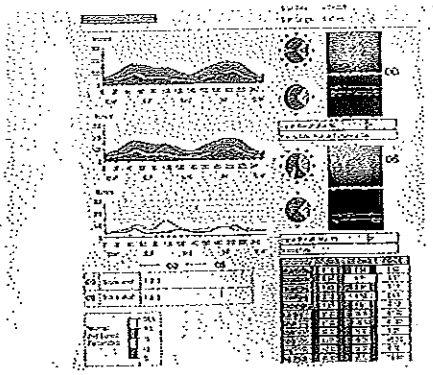


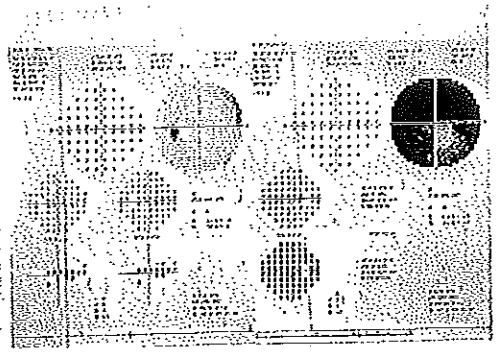
Statistically significant asymmetry → indicative early glaucomatous damage

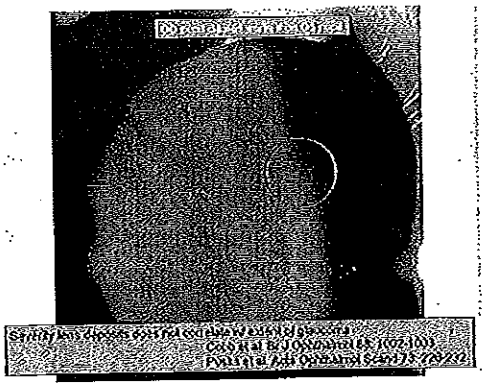
Case

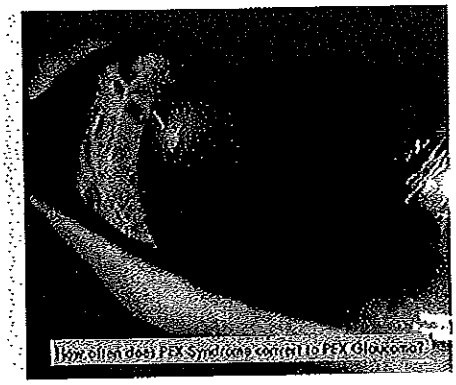
- 75 year old Caucasian male
- CC: 6 month F/U asymmetric IOPs (24/21)
- VA: OD 20/25 OS 20/25
- Pupils: + APD OD (not detected 6 months ago)
- IOP: 38/25 @ 0850
- Cornea: No K spindle, no steamy K
- A/C: No cells/flare
- Iris: No TIDs, no NVI
- Angle: Deep, 4+
  - Gonioscopy: CB NR, trace pigment, no PAS/NVA/AR











Approximately how often does PEX Syndrome convert to PEX Glaucoma?

1. 0%
2. 5%
3. 20%
4. ★ 40%
5. 60%
6. 75%

The Risk of Glaucoma in Pseudoexfoliation Syndrome

253 patients Olmsted County, Minnesota x 15 years

Treated PEX-Glaucoma and PEX- OHT (ave: 32 mmHg)
51% OHT patients nerve damage despite Tx

Risk Factors: IOP initial visit, bilateral involvement

53% patients treated within 15 years

Unilateral Exfoliation Syndrome: Conversion to Bilateral Exfoliation and to Glaucoma: A Prospective 10-Year Follow-up Study

63 Non-Glaucomatous Subjects w/ Unilateral PEX

32% PEX Syndrome → PEX Glaucoma
Fellow eye, 38% Non-Clinical PEX → PEX Glaucoma
100% conversion by year 7

PEX Glaucoma
- -Highest risk conversion once PEX seen
- -Higher initial IOP (20 vs 16)
- -Greater IOP asymmetry
- -Poor pupillary dilation

The Relationship Between Glaucoma and Pseudoexfoliation

The Rhine-Westphalia Eye Study

3554 patients, aged 49-97, most white w/ northern Europe origin

Ocular Hypertension (>21 mmHg) 3 times more frequent w/ PEX: 9.3% vs 3.1%

Glaucoma damage 14.2% PEX vs 1.7% w/o PEX
OR = 5.0, PEX 8 times as frequent!!!

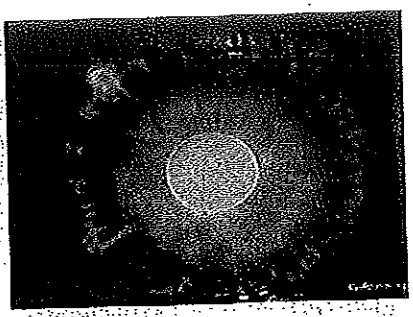
Visual field progression outcomes in glaucoma subtypes

Angle Closure Glaucoma	76 eyes	
Normal Tension Glaucoma	81 eyes	
Pigmentary Glaucoma	34 eyes	Ave 10.4 VFs
Primary Open Angle Glaucoma	275 eyes	FAU 6.4 years
Exfoliative Glaucoma	84 eyes	
Juvenile Glaucoma	37 eyes	

Exfoliative glaucoma
- Fastest rate of global change (-0.65 dB/year)
- Highest mean IOP, fluctuation, peak IOP during f/u
- Highest rate of progression (40%)

PEX Glaucoma Management

- Most common secondary glaucoma
- Relative to POAG, *more difficult to manage*
  - Higher mean IOPs
  - Greater diurnal fluctuation
  - Marked pressure spikes
  - Worse ONH/VF damage
  - Poor response to medications
  - More frequent necessity for surgery
  - Greater proportion blindness



Pigmentary Dispersion Syndrome

PDS Triad

- TRIAD
  - 1) Iris Transillumination Defects
  - 2) Krukenberg Spindle on K endothelium
  - 3) Increased pigmentation of anterior TM

How many have DXT

How many parts of the classic "Triad" for Pigmentary Dispersion do you need to see clinically to make the diagnosis?

1. 1
2. 2
3. 3

Only approximately 42% will show all 3 signs

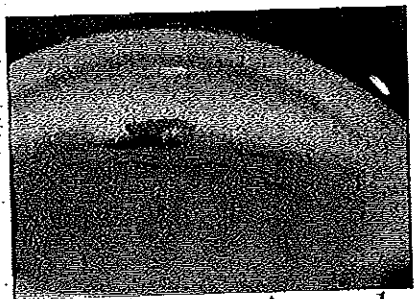
What is the risk of pigmentary glaucoma in PDS?

1. 0%
2. ★ 15%
3. 25%
4. 40%
5. 55%
6. 75%

What Is the Risk of Developing Pigmentary Glaucoma From Pigment Dispersion Syndrome?

113 patients: Risk of PDS → PG
- 10% at 5 years
- 15% at 15 years

IOP > 21 mm Hg initial exam associated with increased risk conversion (46% vs 2%)



Angle Recession and Traumatic Glaucoma

Approximately what percentage of individuals with angle recession develop glaucoma?

1. 2%
2. ★ 6%
3. 15%
4. 25%
5. 35%
6. 55%

Eye Trauma and Glaucoma

- 2 peak incidences of Glaucoma
- 1st peak- first few weeks after trauma, < 1 year
  - Self limited, medications
  - RBC/inflammatory cells plug TM
- 2nd peak – more than 10 years after injury
  - More difficult management, surgery
  - 2 reports glaucoma > 50 years after initial injury
- Approximately 6-7% w/ AR eventually develop glaucoma, 10% at 10 years

How many degrees of angle recession are typically needed for glaucoma to develop?

1. 45 degrees
2. 90 degrees
3. 180 degrees ★
4. 270 degrees
5. 360 degrees

Early Predictors of Traumatic Glaucoma After Closed Globe Injury

40 ejes with closed globe injury w/ glaucoma, 62 w/o glaucoma

3+ pigmentation of angle (80% vs 13% , RR 20.8)
Hyphema (93% vs 42%)
Angle recession > 180 degrees (68% vs 13%)
Elevated baseline IOP (mean 35 vs 17 mmHg)
Phacodonesis (35% vs 15%)
Wider angle
Absence of cyclodialysis

Thank you!

Major Charles Robert Soltes, Jr., O.D.

March 15, 1968 - October 13, 2004

Rob served our nation as a Public Health Commander with the 426th Civil Affairs Battalion, United States Army. On October 13, 2004, while serving in Mosul, Iraq, his convoy was attacked. Major Soltes was the first optometrist killed in action while on active duty in the United States Army.

Today Rob's charitable donations are going to the Blinded Veterans Association to aid Operation Peer Support, a supportive program for war blinded veterans. Donations will also support his alma mater, Norwich University, The Rob Soltes Memorial Scholarship.

Since 2004, the Major Rob Soltes Memorial Tournament has grown each year, raising funds to support eye care services in the name of Dr. Rob Soltes.

The Blinded Veterans Association

The Blinded Veterans Association (BVA) is the only congressionally chartered non-profit veterans service organization that exclusively dedicated to serving the needs of our nation's blinded veterans and their families for 66 years.

The BVA is an organization of blinded veterans helping blinded veterans. Through its service programs, regional groups, resources and advocacy before legislative and executive branches of government, the BVA hopes to make life better for blinded veterans.

Operation Peer Support Testimonial

"The BVA Operation Peer Support program was vital and very important during my recovery and rehabilitation process early on. During military operations in May of 2008, I suffered traumatic injuries to my head which resulted in complete loss of sight. The BVA provided me with the ability to meet other veterans with similar injuries, which in turn helped me build a support network. I would not be where I am today without Operation Peer Support."

SPC Steven C. Baskis (Medic Ret.)
United States Army

Schedule of Events

Oak Creek Golf Club
One Golf Club Drive, Irvine, CA 92618
949-653-5300

10:00 AM
Golf Registration
11:00 AM
Range, and Silent Auction Opens
Golf Contests Begin
11:30 – 12:30 PM
Lunch Served
12:30 PM
Golf Tournament
Helicopter Bail Drop
Shotgun Start - Scramble Format
5:30 – 6:30 PM
Cocktail Hour
6:30 PM
Dinner & Evening Program Begins
Live Auction & Awards

REGISTER EARLY ONLINE FOR DISCOUNTED RATES.

www.soltesmemorial.com

"like" us on facebook

We thank you for your support !

www.bva.org www.norwich.edu

12th Annual
Major Rob Soltes Memorial Golf Tournament

Proceeds Benefiting

Columbus Day
Monday, October 10, 2016

Oak Creek GOLF CLUB®

Sponsorship Opportunities

PATRIOT - Main Sponsor (please inquire)

- Visible identification with The Blinded Veterans Association (BVA)
- Sponsor name attached to event name:
  "The 12th Annual Major Rob Soltes Memorial Golf Tournament presented by: "Your Name"
- Sponsor spokesperson may speak at the dinner program
- Full page ad placement of choice within the Event Program ($200 value)
- Logo displayed prominently with the BVA on the event website
- Sponsor will be provided banner signage at the event and on hole flags
- Two Foursomes (8 players, $1450 value)
- Eight addt'l tickets & reserved seating at the Dinner Program ($400 value)
- Sponsor name mentioned in all media promotions
- Recognition in BVA's newsletter, website, & each document sent to golfers and other sponsors
- Placement of company logo on participant gift bag & promo item placement within the participant gift bag
- Tee Box sponsorship of choice ($300 value)
- Recognition during lunch, throughout the day's announcements, and within the evening program.
- First option for presenting sponsorship next year

Elite Sponsor - $5,000

- Elite Sponsors can choose an exclusive corporate promotion opportunity. Tailored opportunities include:
  (Lunch, Golf Awards Sponsor, Cocktail Hour, Helicopter Ball Drop, Hole in One Opportunity, or Golf Contests: Putting Contest, Par 3 Contests, Driving Contests.)
- Sponsor will be provided signage at the event
- One Foursome (4 players, $725 value)
- Four addt'l tickets & reserved seating at the Dinner Program ($200 value)
- Tee Box sponsorship of choice ($300 value)
- Placement of company promo item within the participant golf bag
- Logo displayed on the event website
- Full page ad placement within the Event Program ($200 value)
- Sponsor will be provided banner signage at the event
- Recognition during the Dinner Program

Sponsor a Veteran Participant - $185

- Host one of our veterans for our event
- Green, Cart & Range Fees, Lunch, Dinner & Program

Gold Sponsor - $2,500

- Corporate Banner on website
- Corporate Banner at Oak Creek GC
- Corporate Sign on Hole (signs provided)
- Full Page Ad in the Event Program ($200 value)
- One Foursome (4 players, $725 value)
- Reserved Seating (evening program)

Silver Sponsor - $1,750

- Corporate Banner on website
- Corporate Sign on Hole (signs provided)
- Full Page Ad in the Event Program ($200 value)
- One Foursome (4 players, $725 value)

Green Sponsor - $1,250

- Corporate Sign on Hole (signs provided)
- Half Page Ad in the Event Program ($150 value)
- One Foursome (4 players, $725 value)

Tee Box Sponsor - $300

- Corporate Sign on Hole (signs provided)
- Half Page Ad in Event Book ($150 value)

Advertise in Our Event Program

Business Card - $40 Half Page - $150
Quarter Page - $75 Full Page - $200

The Blinded Veterans Association is a
Charitable & Educational Non-Profit Organization (501c3).
Federal Tax ID #530214281

Attend

Golf & Dinner Program

Individual Golf Participant

$185 - Early Bird Registration Online or by Mail
Green, Cart & Range Fees
Lunch, Dinner & Evening Program
$200 - Paid on Tournament Day

Golf Foursome

$725 - Early Bird Registration Online or by Mail
Green, Cart & Range Fees
Lunch, Dinner & Evening Program for Four
$800 - Paid on Tournament Day

Dinner & Evening Program Only - $50

Dinner, Beverages, Program, Auction, Awards

Helicopter Ball Drop

Golf Ball Lottery!

Purchase as many opportunities as you would like, if your ball drops from the helicopter and falls into the hole – you win & s will the veterans! (10% UP $2500)

$10 ea. @ soltesmemorial.com

Participate

Volunteer Opportunities • Donate Raffle Items
Silent Auction Donations • Monetary Contributions
FOR MORE INFORMATION PLEASE VISIT
SOLTESMEMORIAL.COM OR EMAIL
INFO@SOLTESMEMORIAL.COM
(949) 438–0140

DR. EDWARD CHU, O.D., F.A.A.O.

Long Beach VAMC
5901 E. 7th Street
Long Beach, CA 90822
Cell (408) 992-5789
Edward.Chu@va.gov

EDUCATION

Berkeley Optometry - *Doctor of Optometry* **May 2008**
GPA: 3.837

University of California, Berkeley -*Bachelor of Arts in Molecular Cell Biology* **December 2003**
Emphasis in Cell and Developmental Biology
Minor: Business Administration
Graduated with Academic Honors, GPA: 3.677

EMPLOYMENT

Long Beach VAMC, Long Beach, California – *Staff Optometrist, Co-Residency Coordinator* **Apr 2014 – Current**
-Supervise and teach optometry residents and fourth year optometric externs
-Weekly extern/resident journal club, grand rounds, and education
-Western University College of Optometry – Adjunct Faculty
-Marshall B. Ketchum University – Adjunct Faculty
-New England College of Optometry – Adjunct Faculty
-Externship Coordinator: SALUS, Western

Salisbury VAMC, Salisbury, North Carolina – *Staff Optometrist* **Sept. 2009- Apr 2014**
-Supervise and teach optometry residents and fourth year optometric externs
-Certified Teleretinal Imaging (TRI) Program Reader
-Salisbury VAMC Residency Interview Committee (2011, 2014)
-Optometry Service Reusable Medical Equipment Committee Liaison (2010-Present)
-The Ohio State University School of Optometry – Adjunct Faculty (2009 – 2014)
-Started and lead extern journal club (Jan. 2013 – Apr. 2014)
-Developed online library of journals for residents/students (Over 700 total)
-Developed 80 "Case of the Day" presentations with accompanying mini-lecture

San Francisco VAMC, San Francisco, California – *Fee Basis Optometrist* **Aug. 2009 – Sept. 2009**

RESIDENCY

San Francisco VAMC, San Francisco, California **July 2008 – July 2009**
-Primary Care Residency with emphasis on Ocular Disease
-Supervised and taught fourth year optometric externs
-4 case presentations and 1 written report
-Ambulatory Low Vision rotation
-Specialty Contact Lens rotation
-Attended weekly UCSF Ophthalmology Grand Rounds and FA Conference
-Research project on Corneal Biomechanics and Glaucoma

CLINICAL EXPERIENCE/ROTATIONS

Meredith W. Morgan University Eye Center, Berkeley, CA	May 2006- July 2007
Tacoma VAMC, Tacoma, Washington	Aug. – Dec. 2007
Fresno VAMC, Fresno, California	Jan. - Mar. 2008
San Diego State University Student Health Services Clinic, San Diego, CA	Mar. – May 2008

LEADERSHIP AND AFFILIATIONS

American Academy of Optometry Press Conference – *Chair*	2016-Present
American Academy of Optometry Website Special Website Task Force - *Member*	2016-Present
Accreditation Council on Optometric Education – *Consultant*	2014-Present
American Academy of Optometry North Carolina Chapter – *President*	2013-2014
American Academy of Optometry Communications Committee – *Member*	2013-Present
American Academy of Optometry Education Quality Assurance Committee – *Member*	2009-Present
Veterans Affairs FAC Recruitment & Retention Subcommittee – *Member*	Feb. 2013-Present
Optometry and Vision Science – *Peer Reviewer*	June 2012–Present
Review of Optometry – *Peer Reviewer*	June 2011-Present
National Association of VA Optometrists – *Member*	2009-Present
American Academy of Optometry – *Member*	2009-Present
American Optometric Association – *Member*	2014-Present
Armed Forces Optometric Society – *Member*	2014-Present
Berkeley Optometry Student Government – *Class of 2008 President*	2007-2008
Berkeley Optometry Chapter of (VOSH) – *Vice President*	2006-2007
Berkeley Optometry Intramural Sports Commissioner	2004-2008
Berkeley Optometry Softball Team Captain (The Fighting Scleras)	2004-2008
UC Berkeley Undergraduate Student Instructor, Anatomy Lab	2003

LECTURES AND PRESENTATIONS

American Academy of Optometry Meeting Anaheim 2016 — Nov 2016
- Lecture: "Nocturnal Considerations in Glaucoma Management

Marshall B Ketchum VA Faculty Program — Sep 2016
- Lecture: "Glaucoma Studies and their Impact on Clinical Management"

Marshall B Ketchum: Ocular Disease Part II — July 2016
- Lecture: "Optic Nerves that Pale in Comparison"

Long Beach VA Grand Rounds — May 2016
- Lecture: "A Day at the VA: Diabetic Retinopathy, Glaucoma, and ARMD"

Greater LA VA Seminars — Jan. 2016
- Lecture: "Strokes and Ocular Manifestations in Your Patients: Prevention and Management"
- Lecture: "Optic Nerves that Pale in Comparison"

American Academy of Optometry Meeting New Orleans 2015 — Oct. 2015
- Lecture: "Evidence Based Management of Secondary Glaucoma"
- Poster: "Kjellin Syndrome: Various Diagnostic Testing for Multifocal Pattern Dystrophy Associated with Hereditary Spastic Parapiegia
  "VA Optometric Service Opportunities"
- Resident Posters: "TBI w/ Loss of Eye and Traumatic Optic Neuropathy in Fellow Eye Following Cannon Blast"
  "Chorioretinitis Sclopetaria w/ Rupture of the Lateral Rectus due to Gunshot"

"Functional loss in Traumatic Brain Injury"

Marshall B. Ketchum Treatment and Management of Ocular Disease — Sep. 2015
Lecture: Strokes and Ocular Manifestations in Your Patients: Prevention and Management

Marshall B. Ketchum Ocular Disease Part II — Jul. 2015
Lecture: Nocturnal IOP in Glaucoma Management

Greater LA VA Seminars — Apr. 2015
Lecture: "Strokes and Ocular Manifestations in Your Patients: Prevention and Management"
Lecture: "Optic Nerves that Pale in Comparison"

Inland Empire Optometric Society — Feb. 2015
Lecture: "Evidence Based Management of Secondary Glaucoma"

American Academy of Optometry Meeting Denver 2014 — Nov. 2014
Lecture: "Optic Nerves that Pale in Comparison"
Lecture: "Evidence Based Management of Secondary Glaucoma"
Lecture: Residents Education Event: "Clinical Problem Solving and the Study of Diagnostic Expertise: 3rd Nerve Palsies"
Resident Posters: "Hemodialysis and the Optic Nerve"
"Central Retinal Artery Occlusion with Large Disc Hemorrhage"
"Chorioretinal Folds: Wrinkles that Warrant Investigation"

Marshall B. Ketchum Treatment and Management of Ocular Disease Event — Sept. 2014
Lecture: "Optic Nerves that Pale in Comparison"

Southeastern Conference of Optometry (SECO) Meeting 2014 – Atlanta — Mar. 2014
Poster: "A Tale of Two Diseases: Mixed Mechanism Macular Edema"
Poster: "Blurred Lines: Weiss Ring, Swollen Disk, or Vitreopapillary Traction?"

Berkeley Practicum Continuing Education Program — Jan. 2014
Lecture: "Strokes and Ocular Manifestations in Your Patients: Prevention and Management"

American Academy of Optometry Meeting Seattle 2013 — Oct. 2013
Lecture: "Optic Nerves that Pale in Comparison"
Poster: "North Carolina Chapter of the American Academy of Optometry"

VISN 6 Diabetes TRI Meeting — Jan. 2013
Lectures: "Flashes and Floaters"

American Academy of Optometry Meeting Phoenix 2012 — Oct. 2012
Lecture: "Under Pressure: Ocular Perfusion, Nocturnal IOP, and Eye Disease"
Lecture: "Preventing Stroke in Your Patients"

North Carolina Armed Forces Optometric Society (AFOS) — Mar. 2012
Lecture: "Evidence Based Management of the 'Other' Glaucomas: An Interactive Discussion"

Southeastern Conference of Optometry (SECO) Meeting 2012- Atlanta — Mar. 2012
Poster: "Ocular Manifestations of Erectile Dysfunction Medication: Waking up with more than you bargained for"

American Academy of Optometry Meeting Boston 2011 — Nov. 2011
Lecture: "Under Pressure: Ocular Perfusion, Nocturnal IOP, and Eye Disease"
Ellerbrock Grand Rounds II: Vitreous Wick Syndrome

American Academy of Optometry Meeting San Francisco 2010 — Nov. 2011
Ellerbrock Grand Rounds I: Topless Optic Disk Syndrome
Workshop Speaker: Obtaining Fellowship in the Academy

North Carolina Piedmont Optometric Society — Oct. 2011
Topless Optic Disk Syndrome
Vitreous Wick Syndrome

American Academy of Optometry North Carolina Chapter — Oct. 2011
Ocular Anomalies from Posterior to Anterior

VISN 6 Diabetes TRI Conference — Mar. 2010
Diabetic Retinopathy

Age Related Macular Degeneration

San Francisco VA Residency Conferences/Presentations — Jul 2008 – Jun 2009

Corneal Biomechanics and Glaucoma
Topless Optic Disk Syndrome
Cystoid Macular Edema and Vitreous Wick Syndrome
Persistent Fetal Vasculature

Berkeley Optometry Student Presentations — Aug 2007 – May 2008

Retinal Arterial Macroaneurysm
Swollen Disks: Differential Diagnoses
Allergic Conjunctivitis

PUBLICATIONS

Chu, E. (2016). Ocular Manifestations of Acute Pancreatitis: Purtscher's Retinopathy. New York NY. NOVA Science Publishers. *Book in press*

Hamp A, Chu E, Slagle S, Hamp R, Joy J, Morris R. Purtscher's Retinopathy Secondary to Acute Pancreatitis. Optometry and Vision Science. 2014 Feb; 91(2): e43-51.

Chu E. Stroke Awareness: The Role of the Optometrist. California Optometry. Sept/Oct 2013: 32-34.

Chu E. Vitreous Wick Syndrome and Cystoid Macular Edema: Advanced Ocular Care. July/August 2013: 63-66.

Chu E. Eye on Stroke Prevention. Review of Optometry. June 2013. 40, 43-44, 46, 48-49.

Chu E. The Topless Optic Disk Syndrome. Advanced Ocular Care. March/April 2013: 57-58.

Chu E, Hamp A. Branded vs Generic Medications. Review of Optometry. February 2012. 68-75.

Chu E, Hamp A. IOP Goes 'Bump' in the Night. Review of Optometry. March 2011. 45-53.

HONORS AND AWARDS

American Academy of Optometry – *Fellow*	2009-Present
Beta Sigma Kappa Optometric Honor Society	2005-2008
Berkeley Optometry Clinical Honors Awards	2006
Berkeley Optometry George L. Schneider Professional Student Support Scholarship	2005
Berkeley Optometry Early Admission Scholarship	2004

RESEARCH EXPERIENCE

Corneal Biomechanics and Glaucoma - *Investigator* — 2008-2009

Supervised by Dr. Andrew Mick, staff optometrist at SFVA. Our prospective study examined biomechanical markers as indicators of optic nerve compliance in glaucoma and normal subjects. We used the Reichert Ocular Response Analyzer to measure biomechanical markers, corneal hysteresis, and corneal resistance factor. Optic nerve cup volume was measured using proprietary software from Zeiss on Cirrus High-Definition 3D-OCT.

Eye Strain in Myopes and the Stiles-Crawford Effect - *Research Assistant* — 2002-2004

Supervised by Professor Jay Enoch, Berkeley Optometry. Our study examined the effects of forces and resultant strain occurring in the posterior pole of the retina with a focus on manifestations found in middle and higher myopes. My contributions to the study included running experiments, participating in the study as a test subject, and processing data. Subjects were assessed by testing the Stiles-Crawford Effect. An OKN drum was used to determine whether eye movements contribute to these retinal strains affecting higher myopic eyes. Findings revealed that strain on the retina in myopia resulted in alterations in retinal receptor orientations that reduced visual function in sampled retinal locations.

VOLUNTEER WORK

San Francisco Veterans – Connect Day **Aug. 2008**
Provided free screenings to veterans of San Francisco. Facilitated enrollment into hospital programs to ensure access to medical care

Berkeley Optometry Interview Day Committee **2006-2007**
Served as co-interviewer with Berkeley Optometry Staff Member. Selected to participate on panel of current Berkeley Optometry students for question and answer session

VOSH, Koror, Republic of Palau **Dec. 2006**
Participated in inaugural VOSH trip to Palau. Provided free eye screenings to over 2500 Palauan citizens

Berkeley Optometry "Opto-Camp" Counselor **Jul. 2006**
Aided in mentoring undergraduate students interested in optometric profession

Suitcase Clinic **Sep. 2005**
Provided free vision screenings to homeless population at First Presbyterian Church of Berkeley

Habitat for Humanity **Oct. 2005**
Participated in building homes for underprivileged families in Bay Area

Anatomy Enrichment Program **Nov. 2003**
Student-teacher in UC Berkeley outreach program to Oceanside Elementary School

Molecular Cell Biology Mentor Program **2002-2003**
Mentored students at Arrowsmith High School Academy. Tutored math and provided college preparation advice

CERTIFICATIONS

EYEPACS Diabetic Retinopathy Screening Program **Mar 2013-Present**

WORK EXPERIENCE

Silicon Valley Eyecare Optometry and Contact Lenses, Santa Clara, CA – *Intern* **2002-2005**
Pre-testing, Humphrey Visual Fields, Pachymetry, Optomap, Contact Lens insertion/removal training, progress calls, confirmed appointments

Impax Pharmaceuticals, Incorporated – *Intern* **Jun. 2000-Aug. 2000**
Worked in Quality Assurance Department testing raw materials prior to production

SPECIAL SKILLS

Technology
Cirrus OCT, Stratus OCT, Non-Mydriatic Fundus Camera, Digital Anterior Segment Slit Lamp Camera, B-Scan Ultrasound, Computerized Patient Record System (CPRS)

Languages
Clinical Chinese and Spanish

HOBBIES AND INTERESTS

Golf (4 Handicap), Basketball, Football, Baseball, Tennis, Softball