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: Vitreo-Retinal Disorders

Provider Name: Dr. Joseph Pruitt

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

☑Correct Application Fee

☐Detailed Course Summary

☑Detailed Course Outline

☑PowerPoint and/or other Presentation Materials

☐Advertising (optional)

☑CV for EACH Course Instructor

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

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1-3323	439594	439594	50

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**
Vitreo-Retinal Disorders	06/23/2017

Course Provider Contact Information

Provider Name

Joseph (First) Pruitt (Last) Allan (Middle)

Provider Mailing Address

Street 11980 Mt Vernon Ave. **City** Grand Terrace **State** CA **Zip** 92313

Provider Email Address pruitt.joseph@gmail.com

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

Joseph (First) Pruitt (Last) Allan (Middle)

License Number 13429	**License Type** TLG
Phone Number (909) 721-7751	**Email Address** pruitt.joseph@gmail.com

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.

Joseph
Signature of Course Provider

3/13/2017
Date

Form CE-01, Rev. 5/16

1 **Vitreo-Retinal Disorders**

Joseph A. Pruitt, O.D., M.B.A., FAAO
Riverside-San Bernardino County Indian Health, Inc.

2 **Anatomy and Landmarks**

- 9 Retinal Neurosensory Layers
  - Internal limiting membrane (ILM)
  - Nerve fiber layer (NFL)
  - Ganglion cells
  - Inner plexiform
  - Inner nuclear
  - Outer plexiform
  - Outer nuclear
  - External limiting membrane
  - Photoreceptors

3 **Retina**

4 **Bonds Between Layers**

- Attaching bond varies
  - RPE to retinal photoreceptors...? (tight or weak)
    - Weak
    - Easily separated by fluid
  - RPE to Bruch's membrane...? (tight or weak)
    - Tight
  - RPE cell to RPE cell...? (tight or weak)
    - Tight

5 **Coloration**

- RPE:
  - has melanin
    - Causes varying shades of black with hypertrophy
  - Has lipofuscin
    - Released by degenerated RPE cells ("wear and tear")
    - Autofluorescent

- Orange→Yellow→Golden→Brown
-
- Whites > Blacks
-
- Known to be a by-product of light exposure

6 **Lipofuscin**

- Clinical Exam
-
  - FDA biomicroscope guidelines
    -
    -
    - "Because prolonged intense light exposure can damage the retina, the use of the device for ocular examination should not be unnecessarily prolonged, and the brightness setting should not exceed what is needed to provide clear visualization of the target structures. This device should be used with filters that eliminate UV radiation (< 400 nm) and, whenever possible, filters that eliminate short-wavelength blue light (< 420 nm)."
  -

7 **Coloration**

- Choriocapillaris
  - Acts as a red filter (uniform; independent of race)
  -
- Choroidal Vessels (larger and deeper)
  - Uniformly red
  - Do not filter color
  -
- Choroid
  - Contains varying amounts melanocytes
    - Thus, variable brown/black color

8 **Coloration**

- Retina
-
  - Pale orange → orange to red → gray/brown
    - Dependent upon:
      - Hemoglobin in choriocapillaris (constant)
      - Melanin in RPE (variable)
      - Lipofuscin in RPE (variable)
      - Melanin in choroid (variable)
      -

9 **Thickness**

- Sensory Retina
-
  - Quite thin in the peripheral (normal)
  -
  - Subject to full thickness breaks from one or more:
    - Atrophy (degenerative)
    - Traction (vitreal-retinal)

10 **Approximate Distances**

- Retinal Periphery

- Equator is marked by...?
  - Vortex veins' ampullas
  -
- Vitreous base's posterior edge is usually ~2 DD posterior to ora
-
- Distance from ora to equator is ~ 4 DD
-
- Vitreous base overlying the retina starts about halfway between the ampullae and the ora

11 **Vitreous Base**

- Normally invisible
- May have pigment at its border or appear white and elevated from traction

12 **Vitreous Base**

- A PVD will not advance farther anteriorly than the posterior vitreous base
-
-
- The vitreous base may advance posteriorly with increasing age

13 **Aging Changes in the Vitreous**

- Liquefaction
  - Manifested by formation of lacunae
    - Lacunae = optically empty cavities filled with fluid, and surrounded by walls of condensed vitreous fibers
    -

- Shrinkage
  - aka "Syneresis"
    - Drawing together of fibers
    - Fibers are drawn away from the liquid (separation of liquid and solid)

14 **Age Changes in the Vitreous**

- Early Shrinkage
  - Condensation only, but readily visible
  -
- Late Shrinkage
  - Increasingly dense
  - Highly visible fibers
  - PVD
  - Traction
  - With symptoms

15 **Vitreous Shrinkage**

- Symptoms:
  - Floaters: spiders, flies, cobwebs, worms seen against light, high-contrast, backgrounds

  - Photopsia
    - Due to mechanical stimulation of retina in areas of traction
    - Varying shapes: light rays, arcuate bands, straight lines
    - Color is of no significance
  -

16 **Vitreous Shrinkage**

- Symptoms (cont.)
  - Metamorphopsia
    - Rather rare
    - Due to macular edema secondary to traction
  - Blur
    - Secondary to:
      - Macular edema
      - Vitreous hemorrhage
      - Transient obscuration from floaters

17 **Vitreous Shrinkage**

- Signs:
  - Opacities
  - Vitreous hemorrhage
    - Actually very common, but very transient and escapes notice
  - Retinal hemorrhage
    - Due to traction on blood vessels

18 **Posterior Vitreous Detachment**

- Complete PVD
  - Detachment extends to the posterior border of the vitreous base, and attachment at the optic nerve is lost

19 **Posterior Vitreous Detachment**

- Incomplete PVD
  - Not a total separation of retina and vitreous
  - Usually occurs superiorly
  - Attachment at optic nerve remains

20 **Course of PVD**

- Usually acute
- Becomes complete in several hours
- Particles from resolving heme disappear in a few days
- Vitreous contracts over a period of ~2 years
- Stable thereafter

21 **PVD Etiology**

- PVD without collapse results from syneresis but no liquefaction
- PVD with collapse results from syneresis with liquefication

22 ☐ **Vitreous Traction**
- 2 different directions of movement
  - Centripetal
    - Away from retina toward vitreous center
      - Edema
      - Hemes
      - Tears
    - *Intermittent traction is often centripetal*
  - Tangential
    - Moving parallel to retina
      - Thinning
      - Wrinkling
      - Horseshoe tear

23 ☐ **Development Anomalies**
- Retinal tufts (aka granular tissue)
  - Located between equator and ora
  - Can be elevated
  - 3 types (2 developmental + 1 circumstantial)
    - Non-cystic tufts:
      - Small
      - Irregularly shaped
      - Internal projections of retina
    - Cystic tufts:
      - Larger
      - Broader bases
      - Nodules of degenerated tissue
    - *Traction tufts:
      - Project more anteriorly into vitreous cavity
      - Develops close to ora (most common nasally)

24 ☐ **Retinal Tufts**

25 ☐ **Congenital Hypertrophy of RPE**
- CHRPE
  - Benign
  - Rarely enlarges over time
  - Sharp borders
  - Usually had depigmented "halo" or internal lucunae
  - CHRPEs in FAP (familial adenomatous polyposis) are irregularly shaped

26 ☐ **Congenital Hypertrophy of RPE**
- Bear Tracks
  - Variety of CHRPE
  - Usually multiple and smaller

- aka "congenital grouped pigmentation"
-
- Multiple (or solitary), small, flat black/brown spots

27 **Congenital Hypertrophy of RPE**

28 **Congenital Hypertrophy of RPE**

29 **Congenital Hypertrophy of RPE**

30 **Choroidal Nevus**

- Benign accumulation of melanocytes in choroid
-
- Usually slate gray
  - Variable color due to overlying RPE
  -
- Feathery borders
  - Melanocytes are randomly gathered at the border
  -
- Mottled appearance due to overlying degenerated RPE
-
- Drusen occurs in response to "abnormality" underneath the RPE

31 **Choroidal Nevus**

32 **Choroidal Nevus**

- Choroidal Nevus vs. Melanoma
-
  - Clinical diagnostic skill/test...?
  -
    - Red-free filter
    -
  - How/why does it work?
  -
    - Green light is reflected and absorbed by melanin granules in RPE; thus structures deeper are absent of light (i.e. disappear)
  -
    -

33 **Choroidal Nevus**

- To Find Small Ocular Melanoma Using Helpful Hints Daily
  - T: Thickness
    - > 2 mm
  - F: Fluid
    - Sub-retinal fluid (suggestive of serous retinal detachment)
  - S: Symptoms
    - Photopsia
    - Vision loss
  - O: Orange Pigment overlying the lesion
    - Lipofuscin
  - M: Margin
    - < 3 mm from optic nerve head
  - U: Ultrasonographic Hollowness
  - H:
  - D: Drusen Absence

34 **TFSOM UHH D Pnemonic**

- Risk Scale:
  - 0 factors = <1% - 3%* risk of nevus converting to melanoma in 5 years
  - 1 factor = 8-38%* risk of nevus converting to melanoma in 5 years
  - 2-3* or more factors: 50% risk of nevus converting to melanoma in 5 years

35 **TFSOM UHH D Pnemonic**

- Good Clinical Tool
  - Relatively straight forward with the possible exception of:
    - Lipofuscin vs. Drusen
    -

36 **Choroidal Nevus**

- Choroidal Nevus vs. Melanoma
  - S.P.O.T.S
    - S: Symptoms
    - P: Position
    - O: Orange Pigment
    - T: Thickness
    - S: Sub-retinal Fluid

37 **Choroidal Nevus**

- Choroidal Nevus vs. Melanoma
    - *Dr. Pruitt's MO:*
      - *0 factors = annual comprehensive exams*
      - *1-2 factors = follow-up every 4-6 months; photo-documentation*
      - *3 or more factors = automatic referral to ocular oncology*

38 **Bonus....**

- Cutaneous nevi vs. melanoma
  - ABCD (est. 1985) – EFG (recently)
    - A: Asymmetric
    - B: Borders
      - irregular
    - C: Color
      - 1 color = good
      - Multi-colored = bad
    - D: Diameter
      - > 6 mm (weakest of the system)
    - E: Enlarging or Evolving
      - Some advocate for "Elevated" instead
    - F: Family History
    - G: Great numbers of nevi

39 **Bonus...numero dos**
- Iris nevi vs. melanoma
  - ABCDEF
    - A:
      - Age (young)
    - B:
      - Blood (hyphema)
    - C:
      - Clock hour (inferior greater risk)
    - D:
      - Diffuse configuration
    - E:
      - Ectropion uveae
        - eversion of the pigmented posterior epithelium of the iris at the pupillary margin.
    - F:
      - Feathery tumor margin

40 **Degenerative Conditions**
- Cystoid Degeneration
  - Intra-retinal cysts in the outer plexiform and inner nuclear layers
  - Cysts are separated by photoreceptor axons and Mueller cells
  - Separating elements break down; cysts enlarge and become confluent
  - Retinal thickness is 3x that of the usual thickness
  - Translucent gray, white or red dots with a stippled surface

41 **Cystoid Degeneration**
- Outer cyst wall is intact, so no risk of penetration of liquefied vitreous
- "Typical Cystoid" is universal condition; not always readily visible
- "Reticular cystoid" occurs at the posterior border of typical cystoid
  - Net-like appearance (hence its name); often bordered by retinal vessels

42 **Cystoid Degeneration**

43 **Degenerative Conditions**
- Equatorial Drusen
  - Same composition and subretinal location as in the posterior pole
  - Very often have pigment surrounding the base
    - Leads to reticular degeneration
  - Extremely low (but possible) risk of developing SRNVM

44 **Equatorial Drusen**

45 **Degenerative Conditions**
- Reticular Degeneration of the RPE

- aka "Peripheral Senile Pigmentary Degeneration"
- aka "Peripheral Tapetochoroidal Degeneration"
- aka "Peripheral Chorioretinal Degeneration"
- aka "Honeycomb Degeneration"
  - Hyper and hypo-pigmentation
    - Most common appearance is light area with variable overlying pigment
    - Reticular = net-like or lacy-appearing

- When located at the equator
  - Histology:
    - Pigment surrounding bases of large equatorial drusen
    - Variable hyper and hypo-pigmentation of RPE cells
    - Pigmented venous cuffing (macrophages try to remove pigment)

46 ☐ **Reticular Degeneration**

Due to a loss of perfusion of choriocapillaris from arteriosclerosis

Loss of both RPE melanin granules as well as photoreceptors

May have irregular lines of pigment or a "honeycomb" appearance

47 ☐ **Reticular Degeneration**

48 ☐ **Degenerative Conditions**

Cobblestone/Pavingstone Degeneration

- aka "Chorioretinal Atrophy"
- Depigmented round or oval areas where sclera and large choroidal vessels are visable
- Pigment varies within the area itself and its border
- Arranged parallel to the ora
- Increases with age
- Most commonly observed inferiorly
  - ~50% between 5 and 7 o'clock

49 ☐ **Cobblestone/Pavingstone Degeneration**

Percentage of increased risk of Retinal Detachment when present...?

50 ☐ **Degenerative Conditions**

Acquired (Adult) Retinoschisis

- Retinal split between inner nuclear and outer plexiform layers
- Same location within retina as with cystoid degeneration
- Most often occurs inferior-temporally
- Absolute visual field defect

- Often goes unnoticed by patient until schisis progresses past the equator

51 **Retinoschisis**

52 **Retinoschisis**

53 **Degenerative Conditions**

- White with/and Without Pressure
  - WWP = with scleral depression
  - WOP = without scleral depression
  -
  - Mechanisms:
    - Related to vitreo-retinal traction
    - Interface between vitreous and retina is altered
    -

54 **White Without Pressure**

- Younger patients
  - Possibly due to increased vitreous contraction since typically too young for PVD
  -
- Associated risks/cause for concern:
  - When located along posterior border of lattice
  -
  - When posterior border is irregular or scalloped
  -
  - When any vitreous membrane or bands are attached
  -
  - When present in the fellow eye of a patient with retinal tear
  -

55 **White Without Pressure**

56 **Degenerative Conditions**

- Lattice Degeneration
  - Epidemiology
    - Young patients; first appears in 10-20 year age group
    - Refractive error is <u>not</u> associated
    - Temporal retina more affected than nasal retina
    - Most common 11 to 1 o'clock and 5 to 7 o'clock
    -
  - Appearances
    - Early: loss of retinal transparency; mimics WOP
    - Later: sclerosed vessels + increase RPE changes

57 **Lattice Degeneration**

- Typical Features:
  - Ragged, dull, roughened retinal
  - Oval, elongated or round in shape
  - Typically parallel to ora
  -
- Pathogenesis most likely due to vitreous degeneration plus traction leading to loss of inner retinal layers

58 **Lattice Degeneration**

- Retina is thinned down to outer nuclear layer and external limiting membrane (i.e. inner retinal degeneration)
  - Possibly to loss of entire sensory retina

- Signs of progression
  - Enlargement
  - Increase pigmentation
  - WoP
  - Hemes
  - Holes

59 **Lattice Degeneration**

- Holes with Lattice Degeneration
  - Occur up to ~30% of the time
  - Infrequent relation to RD; ~14%
- Tears associated with Lattice Degeneration
  - Usually linear in orientation when along posterior border
  - Much higher likeliness for RD due to liquid vitreous' easy access

60 **Lattice Degeneration**

- Follow-up
  - Yearly if asymptomatic
  - Every 6 months if symptomatic
  - Asymptomatic holes should be treated if other risks are present
  - ALL tears and breaks should be treated
  - Treat in the presence of cataract that precludes laser treatment
  - Treat in monocular patient

61 **Lattice Degeneration**

62 **Degenerative Conditions**

- Snailtrack Degeneration
  - Appears like "frost" on the retina
    - Very similar to WOP
    - Shaped similarly as lattice
  - ~80% occur between ora and 2 DD anterior to the equator
  - Speculation eventually becomes lattice
    - Unsubstantiated

63 **Snailtrack Degeneration**

64 **Retinal Holes and Breaks**

- Atrophic retinal holes
  - NOT caused by traction
  - Occur in atrophic retina
    - Possibly related to underlying vascular insufficiency

- Small, round and red
  - *Although, can appear gray-ish against darker backgrounds (e.g. tigroid fundi)*
- Non-operculated (since no traction)

65 **Atrophic Retinal Holes**

- Percentage of increased risk of Retinal Detachment when present...?

66 **Atrophic Retinal Holes**

67 **Atrophic Retinal Holes**

68 **Retinal Holes and Breaks**

- Operculated Retinal Breaks
  - Round, red hole with operculum attached to vitreous
  - Operculum looks smaller than hole due to degeneration of tissue
  - Locations are typically between equator and ora
    - Occurs temporally more so than nasally

69 **Operculated Retinal Breaks**

- Should treat an operculated break with presence of other risk factors:
  - High myopia
  - Aphakia
  - Extensive vitreoretinal degeneration
  - History of RD in fellow eye

70 **Operculated Retinal Breaks**

71 **Retinal Holes and Breaks**

- Horseshoe Retinal Tear (Flapped Tear)
  - Characteristics
    - Horseshoe-shaped, with apex
    - Flap looks white/gray from edema and degeneration
  - Occurs more often with increasing age, myopia and aphakia
  - The leading cause of RD

72 **Horseshoe Retinal Tear**

- Up to 30% of symptomatic tears go on to a retinal detachment
  - Thus ALL symptomatic tears are treated
  - AND most all asymptomatic tears are treated

73 **Horseshoe Retinal Tear**

74 **Horseshoe Retinal Tear**

75 **Retinal Detachment**

- Contributing factors
  - Weak bonds between RPE and retina
  - Vitreous loses its shock-absorbing capacity with aging
  - Lattice, chorioretinal scars, pigments clumps all have increased traction
  - Vitreous liquefaction

76 **Retinal Detachment**

- Symptoms
  - Photopsia
  - Floaters
  - Veiling (i.e. "curtains falling")

77 **Retinal Detachment**

- Appearance
  - Grey-white retina
  - NO choroidal details are visible
  - Billowing folds due to subretinal fluid
  - Undulating surface
  - Shafer's sign ("tobacco dust")
    - Can also be present with retinal tears

78 **Retinal Detachment**

- Rhegmatogenous RD
  - Arising from a retinal break
    - Non-traumatic
      - Most common
      - Older patients with equatorial retinal break
    - Traumatic
      - Less common
      - Typically in the far periphery
      - Delayed appearance (up to 2 years)

79 **Retinal Detachment**

- Non-rhegmatogenous RD
  - NOT arising from a retinal break
    - Accumulation of exudate or transudate in subretinal space
    - Tumors, choroiditis and retinal angiomatosis
    - Traction upon adhesion bands

80 **Retinal Detachment**

- Risk Factors
  - Risk of an RD with a retinal break is 1 in 70
  - Risk increases with family history of RD
  - Risk increases with high myopia, vitreoretinal traction, retinal degeneration
  - Superior RDs progress faster due to gravity pulling subretinal fluid down
  - Superior-temporal location is worst
    - Macula most vulnerable
  - Superior-nasal area has less risk
    - Optic nerve blocks progression
  - Greater risk with non-operculated retinal breaks
    - Traction is continuous

81 **Retinal Detachment**

- Histopathology
  - Serous fluid enters through retinal break
  - Passes underneath retina
  - Photoreceptors degenerate
  - Outer layers become edematous and atrophic 2-3 months later
  - Cysts and glial tissue proliferate

82 **Retinal Detachment**

- With longstanding RD
  - Extensive and degeneration of outer retinal layers
  - Glial tissue proliferates extensively
  - Retina contracts and stretches tightly
  - Subretinal bands of tissue adhere the retina to itself
  - Glial tissue may seal retinal breaks and trap retinal debris and fluid
  - Trapped irritants cause uveitis, secondary glaucoma, cataract, phthisis
  - Enucleation is quite likely

83 **Retinal Detachment**

84 **Retinal Detachment**

- Treatment
  - Scleral buckle
    - Adhere retina to RPE with cryo or laser
    - Then encircling silicone sponge/band placed adjacent to tear (relieving traction on retina)
    - Then drain subretinal fluid

- Finally tighten sutures to permanently indent globe

85 **Scleral Buckle**

86 **Retinal Detachment**

- Treatment
  - Pneumoretinopexy
    - Cryo seals the tear
    - Gas is then injected to "splint" the retina against the eye wall
    - Works best for small breaks located superiorly

87 **Retinal Detachment**

- Treatment
  - Vitrectomy
    - Remove vitreous
    -
    - Replacement fluid instilled
    -
    - Then peel vitreous and debris from retina
    -
    - Then exchange air for fluid
    -
    - Then endolaser to adhere retina to RPE
    -
    - Lastly, long-acting gas to replace the air

88 **Vitrectomy**

89 **Differentiating Retinal Detachment from Retinoschisis**

Vitreo-Retinal Disorders

Anatomy and Landmarks

- 9 Retinal Neurosensory Layers
  - Internal limiting membrane (ILM)
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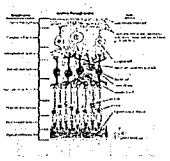
Retina



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  - RPE to Bruch's membrane…? (tight or weak)
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  - RPE cell to RPE cell…? (tight or weak)
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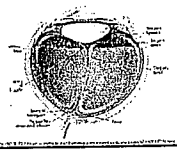

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- Late Shrinkage
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Vitreous Shrinkage

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Vitreous Shrinkage

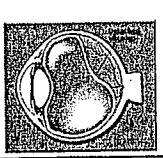
- Symptoms (cont.)
  - Metamorphopsia
    - Rather rare
    - Due to macular edema secondary to traction
  - Blur
    - Secondary to:
      - Macular edema
      - Vitreous hemorrhage
      - Transient obscuration from floaters

Vitreous Shrinkage

- Signs:
  - Opacities
  - Vitreous hemorrhage
    - Actually very common, but very transient and escapes notice
  - Retinal hemorrhage
    - Due to traction on blood vessels

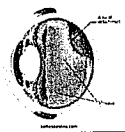
Posterior Vitreous Detachment

- Complete PVD
  - Detachment extends to the posterior border of the vitreous base, and attachment at the optic nerve is lost



Posterior Vitreous Detachment

- Incomplete PVD
  - Not a total separation of retina and vitreous
  - Usually occurs superiorly
  - Attachment at optic nerve remains



Course of PVD

- Usually acute
- Becomes complete in several hours
- Particles from resolving heme disappear in a few days
- Vitreous contracts over a period of ~2 years
- Stable thereafter

PVD Etiology

- PVD without collapse results from syneresis but no liquefaction
- PVD with collapse results from syneresis with liquefication

Vitreous Traction

- 2 different directions of movement
  - Centripetal
    - Away from retina toward vitreous center
      - Edema
      - Hemes
      - Tears
    - *Intermittent traction is often centripetal*
  - Tangential
    - Moving parallel to retina
      - Thinning
      - Wrinkling
      - Horseshoe tear

Development Anomalies

- Retinal tufts (aka granular tissue)
  - Located between equator and ora
  - Can be elevated
  - 3 types (2 developmental + 1 circumstantial)
    - Non-cystic tufts:
      - Small
      - Irregularly shaped
      - Internal projections of retina
    - Cystic tufts:
      - Larger
      - Broader bases
      - Nodules of degenerated tissue
    - "Traction tufts:
      - Project more anteriorly into vitreous cavity
      - Develops close to ora (most common nasally)

Retinal Tufts

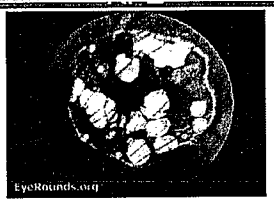
Congenital Hypertrophy of RPE

- CHRPE
  - Benign
  - Rarely enlarges over time
  - Sharp borders
  - Usually had depigmented "halo" or internal lucunae
  - CHRPEs in FAP (familial adenomatous polyposis) are irregularly shaped

Congenital Hypertrophy of RPE

- Bear Tracks
  - Variety of CHRPE
  - Usually multiple and smaller
  - aka "congenital grouped pigmentation"
  - Multiple (or solitary), small, flat black/brown spots

Congenital Hypertrophy of RPE



Congenital Hypertrophy of RPE

Congenital Hypertrophy of RPE

Choroidal Nevus

- Benign accumulation of melanocytes in choroid
- Usually slate gray
  - Variable color due to overlying RPE
- Feathery borders
  - Melanocytes are randomly gathered at the border
- Mottled appearance due to overlying degenerated RPE
- Drusen occurs in response to "abnormality" underneath the RPE

Choroidal Nevus

Choroidal Nevus

- Choroidal Nevus vs. Melanoma

Clinical diagnostic skill/test…?

- Red-free filter

How/why does it work?

- Green light is reflected and absorbed by melanin granules in RPE, thus structures deeper are absent of light (i.e. disappear)

Choroidal Nevus

To Find Small Ocular Melanoma Using Helpful Hints Daily

- T: Thickness
  - > 2 mm
- F: Fluid
  - Sub-retinal fluid (suggestive of serous retinal detachment)
- S: Symptoms
  - Prolapse
  - Vision loss
- O: Orange Pigment overlying the lesion
  - Lipofuscin
- M: Margin
  - < 3 mm from optic nerve head
- U: Ultrasonographic Hollowness
- H:
- D: Drusen Absence

TFSOMUHHD Phemonic

- Risk Scale:

  0 factors = <1% - 3%* risk of nevus converting to melanoma in 5 years

  1 factor = 8-38%* risk of nevus converting to melanoma in 5 years

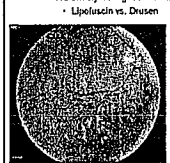
  2-3* or more factors: 50% risk of nevus converting to melanoma in 5 years

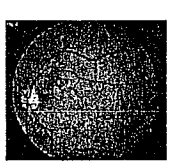
* Figures vary in literature

TFSOMUHHD Phemonic

- Good Clinical Tool

  Relatively straight forward with the possible exception of:
  - Lipofuscin vs. Drusen





Choroidal Nevus

- Choroidal Nevus vs. Melanoma

S.P.O.T.S

- S: Symptoms
- P: Position
- O: Orange Pigment
- T: Thickness
- S: Sub-retinal Fluid

Choroidal Nevus

- Choroidal Nevus vs. Melanoma
  - *Dr. Pruitt's MO:*
    - *0 factors = annual comprehensive exams*
    - *1-2 factors = follow-up every 4-6 months; photo-documentation*
    - *3 or more factors = automatic referral to ocular oncology*

Bonus…

- Cutaneous nevi vs. melanoma
  - ABCD (est. 1985) – EFG (recently)
    - A: Asymmetric
    - B: Borders
      - Irregular
    - C: Color
      - 1 color = good
      - Multi-colored = bad
    - D: Diameter
      - > 6 mm (weakest of the system)
    - E: Enlarging or Evolving
      - Some advocate for "Elevated" instead
    - F: Family History
    - G: Great numbers of nevi

Bonus…numero dos

- Iris nevi vs. melanoma
  - ABCDEF
    - A:
      - Age (young)
    - B:
      - Blood (hyphema)
    - C:
      - Clockhour (inferior greater risk)
    - D:
      - Diffuse configuration
    - E:
      - Ectropion uveae
        - eversion of the pigmented posterior epithelium of the iris at the pupillary margin
    - F:
      - Feathery tumor margin

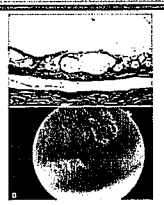
Degenerative Conditions

- Cystoid Degeneration
  - Intra-retinal cysts in the outer plexiform and inner nuclear layers
  - Cysts are separated by photoreceptor axons and Mueller cells
  - Separating elements break down; cysts enlarge and become confluent
  - Retinal thickness is 3x that of the usual thickness
  - Translucent gray, white or red dots with a stippled surface

Cystoid Degeneration

- Outer cyst wall is intact, so no risk of penetration of liquefied vitreous
- "Typical Cystoid" is universal condition; not always readily visible
- "Reticular cystoid" occurs at the posterior border of typical cystoid
  - Net-like appearance (hence its name); often bordered by retinal vessels

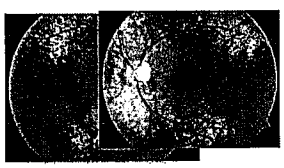
Cystoid Degeneration



Degenerative Conditions

- Equatorial Drusen
  - Same composition and subretinal location as in the posterior pole
  - Very often have pigment surrounding the base
    - Leads to reticular degeneration
  - Extremely low (but possible) risk of developing SRNVM

Equatorial Drusen



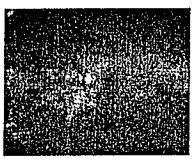
Degenerative Conditions

- Reticular Degeneration of the RPE
  - aka "Peripheral Senile Pigmentary Degeneration"
  - aka "Peripheral Tapetochoroidal Degeneration"
  - aka "Peripheral Chorioretinal Degeneration"
  - aka "Honeycomb Degeneration"
    - Hyper and hypo-pigmentation
      - Most common appearance is light area with variable overlying pigment
      - Reticular = net-like or lacy-appearing
  - When located at the equator
    - Histology:
      - Pigment surrounding bases of large equatorial drusen
      - Variable hyper and hypo-pigmentation of RPE cells
      - Pigmented venous cuffing (macrophages try to remove pigment)

Reticular Degeneration

- Due to a loss of perfusion of choriocapillaris from arteriosclerosis
- Loss of both RPE melanin granules as well as photoreceptors
- May have irregular lines of pigment or a "honeycomb" appearance

Reticular Degeneration



Degenerative Conditions

- Cobblestone/Pavingstone Degeneration
  aka "Chorioretinal Atrophy"

  Depigmented round or oval areas where sclera and large choroidal vessels are visable

  Pigment varies within the area itself and its border

  Arranged parallel to the ora

  Increases with age

  Most commonly observed inferiorly
  - ~50% between 5 and 7 o'clock

Cobblestone/Pavingstone Degeneration

- Percentage of increased risk of Retinal Detachment when present...?

0%

Degenerative Conditions

- Acquired (Adult) Retinoschisis
  Retinal split between inner nuclear and outer plexiform layers

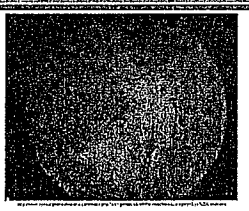
  Same location within retina as with cystoid degeneration

  Most often occurs inferior-temporally

  Absolute visual field defect

  Often goes unnoticed by patient until schisis progresses past the equator

Retinoschisis



Retinoschisis

Degenerative Conditions

- White with/and Without Pressure
  - WWP = with scleral depression
  - WOP = without scleral depression

  Mechanisms:
  - Related to vitreo-retinal traction
  - Interface between vitreous and retina is altered

White Without Pressure

- Younger patients
  Possibly due to increased vitreous contraction since typically too young for PVD
- Associated risks/cause for concern:
  When located along posterior border of lattice

  When posterior border is irregular or scalloped

  When any vitreous membrane or bands are attached

  When present in the fellow eye of a patient with retinal tear

White Without Pressure

Degenerative Conditions

- Lattice Degeneration
  - Epidemiology
    - Young patients; first appears in 10-20 year age group
    - Refractive error is not associated
    - Temporal retina more affected than nasal retina
    - Most common 11 to 1 o'clock and 5 to 7 o'clock
  - Appearances
    - Early: loss of retinal transparency; mimics WOP
    - Later: sclerosed vessels + increase RPE changes

Lattice Degeneration

- Typical Features:
  - Ragged, dull, roughened retinal
  - Oval, elongated or round in shape
  - Typically parallel to ora
- Pathogenesis most likely due to vitreous degeneration plus traction leading to loss of inner retinal layers

Lattice Degeneration

- Retina is thinned down to outer nuclear layer and external limiting membrane (i.e. inner retinal degeneration)
  - Possibly to loss of entire sensory retina
- Signs of progression
  - Enlargement
  - Increase pigmentation
  - WoP
  - Hemes
  - Holes

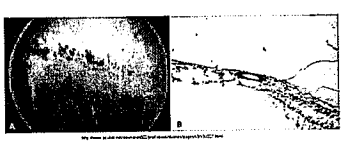
Lattice Degeneration

- Holes with Lattice Degeneration
  - Occur up to ~30% of the time
  - Infrequent relation to RD; ~14%
- Tears associated with Lattice Degeneration
  - Usually linear in orientation when along posterior border
  - Much higher likeliness for RD due to liquid vitreous' easy access

Lattice Degeneration

- Follow-up
  - Yearly if asymptomatic
  - Every 6 months if symptomatic
  - Asymptomatic holes should be treated if other risks are present
  - ALL tears and breaks should be treated
  - Treat in the presence of cataract that precludes laser treatment
  - Treat in monocular patient

Lattice Degeneration



Degenerative Conditions

- Snailtrack Degeneration
  - Appears like "frost" on the retina
    - Very similar to WOP
    - Shaped similarly as lattice
  - ~80% occur between ora and 2 DD anterior to the equator
  - Speculation eventually becomes lattice
    - Unsubstantiated

Snailtrack Degeneration

Retinal Holes and Breaks

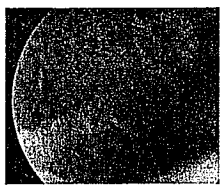
- Atrophic retinal holes
  - NOT caused by traction
  - Occur in atrophic retina
    - Possibly related to underlying vascular insufficiency
  - Small, round and red
    - Although can appear gray-ish against darker background (e.g. tigroid fundi)
  - Non-operculated (since no traction)

Atrophic Retinal Holes

- Percentage of increased risk of Retinal Detachment when present…?

Atrophic Retinal Holes

Atrophic Retinal Holes



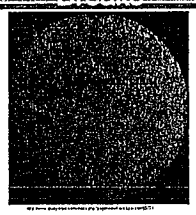
Retinal Holes and Breaks

- Operculated Retinal Breaks
  - Round, red hole with operculum attached to vitreous
  - Operculum looks smaller than hole due to degeneration of tissue
  - Locations are typically between equator and ora
    - Occurs temporally more so than nasally

Operculated Retinal Breaks

- Should treat an operculated break with presence of other risk factors:
  - High myopia
  - Aphakia
  - Extensive vitreoretinal degeneration
  - History of RD in fellow eye

Operculated Retinal Breaks



Retinal Holes and Breaks

- Horseshoe Retinal Tear (Flapped Tear)
  - Characteristics
    - Horseshoe-shaped, with apex
    - Flap looks white/gray from edema and degeneration
  - Occurs more often with increasing age, myopia and aphakia
  - <u>The leading cause of RD</u>

Horseshoe Retinal Tear

- Up to 30% of symptomatic tears go on to a retinal detachment
  - Thus ALL symptomatic tears are treated
  - AND most all asymptomatic tears are treated

Horseshoe Retinal Tear

Horseshoe Retinal Tear

Retinal Detachment

- Contributing factors
  - Weak bonds between RPE and retina
  - Vitreous loses its shock-absorbing capacity with aging
  - Lattice, chorioretinal scars, pigments clumps all have increased traction
  - Vitreous liquefaction

Retinal Detachment

- Symptoms
  - Photopsia
  - Floaters
  - Veiling (i.e. "curtains falling")

Retinal Detachment

- Appearance
  - Grey-white retina
  - NO choroidal details are visible
  - Billowing folds due to subretinal fluid
  - Undulating surface
  - Shafer's sign ("tobacco dust")
    - Can also be present with retinal tears

Retinal Detachment

- Rhegmatogenous RD
  - Arising from a retinal break
    - Non-traumatic
      - Most common
      - Older patients with equatorial retinal break
    - Traumatic
      - Less common
      - Typically in the far periphery
      - Delayed appearance (up to 2 years)

Retinal Detachment

- Non-rhegmatogenous RD
  - NOT arising from a retinal break
    - Accumulation of exudate or transudate in subretinal space
    - Tumors, choroiditis and retinal angiomatosis
    - Traction upon adhesion bands

Retinal Detachment

- Risk Factors
  - Risk of an RD with a retinal break is 1 in 70
  - Risk increases with family history of RD
  - Risk increases with high myopia, vitreoretinal traction, retinal degeneration
  - Superior RDs progress faster due to gravity pulling subretinal fluid down
  - Superior-temporal location is worst
    - Macula most vulnerable
  - Superior-nasal area has less risk
    - Optic nerve blocks progression
  - Greater risk with non-operculated retinal breaks
    - Traction is continuous

Retinal Detachment

- Histopathology
  - Serous fluid enters through retinal break
  - Passes underneath retina
  - Photoreceptors degenerate
  - Outer layers become edematous and atrophic 2-3 months later
  - Cysts and glial tissue proliferate

Retinal Detachment

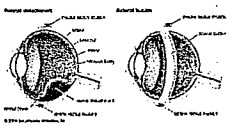
- With longstanding RD
  - Extensive and degeneration of outer retinal layers
  - Glial tissue proliferates extensively
  - Retina contracts and stretches tightly
  - Subretinal bands of tissue adhere the retina to itself
  - Glial tissue may seal retinal breaks and trap retinal debris and fluid
  - Trapped irritants cause uveitis, secondary glaucoma, cataract, phthisis
  - Enucleation is quite likely

Retinal Detachment

Retinal Detachment

- Treatment
  - Scleral buckle
    - Adhere retina to RPE with cryo or laser
    - Then encircling silicone sponge/band placed adjacent to tear (relieving traction on retina)
    - Then drain subretinal fluid
    - Finally tighten sutures to permanently indent globe

Scleral Buckle



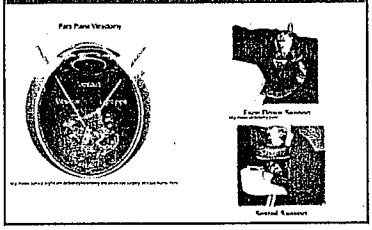
Retinal Detachment

- Treatment
  - Pneumoretinopexy
    - Cryo seals the tear
    - Gas is then injected to "splint" the retina against the eye wall
    - Works best for small breaks located superiorly

Retinal Detachment

- Treatment
  - Vitrectomy
    - Remove vitreous
    - Replacement fluid instilled
    - Then peel vitreous and debris from retina
    - Then exchange air for fluid
    - Then endolaser to adhere retina to RPE
    - Lastly, long-acting gas to replace the air

Vitrectomy



Detachment from

Characteristic	Retinal Detachment	Retinal [illegible]
Transparency	Little	Common
Mobility	Mobile	Immobile
Surface	Folds	Smooth
Fluid Shift	Often	Absent
Tear	Common	Rare
Field Defect	Relative	Absolute

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Joseph A. Pruitt, O.D., M.B.A., FAAO

Objective:

Education:

Nova Southeastern University, Fort Lauderdale-Davie, Florida 2008-2011
Master of Business Administration, 2011

West Los Angeles Veteran Affairs Healthcare Center, Los Angeles, California 2007-2008
Residency Certificate, Geriatric/Primary Care, 2008

Illinois College of Optometry, Chicago, Illinois 2003-2007
Doctor of Optometry, 2007

California State Polytechnic University, Pomona, California 2000-2003
Bachelor of Science, Biology, 2003

University of Memphis, Memphis, Tennessee 1999-2000
Major in Biology

Licenses:

Tennessee #2753 Date of Issue: July 10, 2007
- Active
- Injectible Certification
- Therapeutic Certification

California #13429T Date of Issue: Sept. 28, 2007
- Active
- Therapeutic and Pharmaceutical Agent + Lacrimal Irrigation and Dilation + Glaucoma (TLG) Certified

Georgia #OPT002454 Date of Issue: June 12, 2008
- Active
- Diagnostic and Therapeutic Pharmaceutical Agent Certified

Minnesota #3130 Date of Issue: June 17, 2008
- Active
- Diagnostic Pharmaceutical Agent (DPA) Certified
- Therapeutic Pharmaceutical Agent (TPA) Certified

Board Certification:

American Board of Certification in Medical Optometry Date of recertification: Feb 2018
- Board certified

Certifications:

Drug Enforcement Agency (DEA) Certified Date of Expiration: Mar 2020

Cardiopulmonary Resuscitation (CPR) &
Automated External Defibrillator (AED) Recommended Renewal: Mar 2017

Bausch & Lomb Overnight Orthokeratology Date of Issue/Completion: April 6, 2006
- Certification Number: 20060406002

Paragon Corneal Refractive Therapy (CRT) — Date of Issue/Completion: Dec. 28, 2007
- Certification Number: 161000

Advance Competence in Medical Optometry (ACMO) — Date Taken: June 13, 2008
- Administered by the National Board of Examiners in Optometry (NBEO)
- Examination only made available to candidates meeting specific clinical experience requirements/pre-requisites
- Passed examination

Employment:

Riverside San Bernardino County Indian Health, Inc (RSBCIHI) — Oct. 2014- present
- Director of Eye Care
- Staff Optometrist

Riverside San Bernardino County Indian Health, Inc (RSBCIHI) — July 2014- Oct. 2014
- Staff Optometrist

Minneapolis Veteran Affairs Health Care System — Nov 2008- June 2014
- Low Vision/Staff Optometrist
- Optometric Residency Coordinator
  - Spearheaded and implemented program
- Student Externship Coordinator
  - Spearheaded and implemented program

Wal-Mart Vision Center (Red Wing & Rochester, MN) — Jul 2008- Nov 2008
- Associate Optometrist

EyExam of California — Oct 2007- June 2008
- On-call/Fill-in Optometrist

Faculty Appointments:

Western University of Health Science / College of Optometry, Pomona, California — Jan 2015 - present
- Clinical Assistant Professor of Optometry
- RSBCIHI Externship Site Program Director
  - As part of being RSBCIHI Eye Care Director

University of the Incarnate Word-Rosenberg School of Optometry, San Antonio, Texas — May 2012- June 2014
- Clinical Assistant Professor
- Minneapolis VA HCS Externship Site Program Director

Midwestern University-Arizona College of Optometry, Glendale, Arizona — May 2012- June 2014
- Adjunct Clinical Assistant Professor
- Minneapolis VA HCS Externship Site Program Director

Southern College of Optometry, Memphis, Tennessee — Dec 2010- June 2014
- Adjunct Faculty
- Minneapolis VA HCS Externship Site Program Director

University of Missouri, St. Louis College of Optometry, St. Louis, Missouri — Jul 2009- June 2014
- Adjunct Assistant Professor
- Minneapolis VA HCS Externship Site Program Director

Experience:

Riverside-San Bernardino Indian Health, Inc — Oct 2014 - present
- Director of Eye Care
  - Oversee all organizational Eye Care activities

- Staff Optometrist

Riverside-San Bernardino Indian Health, Inc — Jul 2014 – Oct 2014
- Staff Optometrist

Minneapolis Veteran Affairs Medical Center — Nov 2008- June 2014
- Staff Optometrist
  - Primary Eye Care
  - Low Vision
    - Sole low vision eye care provider
  - Polytrauma/Traumatic Brain Injury (TBI) Ocular Health & Vision Assessments

- VISN 23 Low Vision Continuum of Care Conference (May 2009)
  - Faculty
  - Planning committee

- Established Associated Health Education Affiliation Agreement with University of Missouri, St. Louis College of Optometry, Ferris State University Michigan College of Optometry, & Southern College of Optometry for the optometric externship program
  - Externship program director

- Established Associated Health Education Affiliation Agreement with the Illinois College of Optometry for the optometry residency program
  - Residency in Primary Care/Brain Injury and Vision Rehabilitation
  - Residency program director
    - Designed the program's curriculum
    - Secured all necessary approvals and funding
    - After the initial site visit, program received full ACOE accreditation

Wal-Mart Vision Center (Red Wing & Rochester, MN) — Jul 2008- Nov 2008
- Associate Optometrist

Residency:

West Los Angeles Veteran Affairs Healthcare Center — Jul 2007- June 2008
- Geriatrics/Primary Care
  - Primary Care including Diabetic exams
  - Low Vision evaluations/exams
  - Nursing home/in-patient exams
  - Medically justified specialty contact lenses exams/fittings
  - Lecture Internal Medicine's and Endocrinology's Residents & Interns on Diabetic Retinopathy
    - Given during Chief Resident rotation
  - Precept Southern California College of Optometry's interns

Optometric Externships:

Atlantic Eye Institute, Jacksonville Beach, FL — Feb-May 2007
- OD/MD private practice with an emphasis on Contact Lenses and Primary Care
- Observed multiple surgical procedures:
  - Cataract Extraction
  - Blepharoplasty
  - Strabismus recession and resection

Memphis Veterans Affairs Medical Center (VAMC), Memphis, TN — Nov 2006-Feb 2007
- Emphasis on Primary Care
- Assisted in direct care in a high patient volume

medical optometric eye clinic
- Assisted in optometric injections and fluorescence angiographies procedures

Illinois Eye Institute (IEI), Chicago, IL — Aug-Nov 2006
- Emphasis on Pediatrics/Binocular Vision, Advance Care, and Low Vision
- Performed comprehensive eye exams on pediatric patients (infants-11yrs of age)
- Performed comprehensive eye exams on "at risk/2nd chance" children one day a week at Maryville Academy
- Constructed, tailored and performed successful binocular vision/vision therapy treatments to 4 children over a 10 week period
- Assisted in the treatment of advance glaucoma with attending University of Chicago ophthalmologist
- Performed problem specific examinations one day per week in IEI's Emergency/Urgent Care/Walk-in clinic
- Performed full Low Vision examinations including Low Vision device selection and training

Body of Christ Optometry Clinic, Tegucigalpa, Honduras — May-Aug 2006
- Emphasis on Primary and Advance Care
- Performed full-scope optometric care in a high patient volume medical clinic geared towards the underprivileged
- Also worked closely with a local ophthalmologist
  - Observed and assisted in Cataract Extraction and Incision and Curettage procedures
  - Provided pre and post-surgical care

Primary Care Clinical Education
Illinois Eye Institute, Chicago, IL — Aug 2005-May 2006

Volunteer Optometric Assistant
Body of Christ Optometry Clinic, Tegucigalpa, Honduras — Jun-Aug 2004
- Assisted staff optometrist in direct patient care in the clinic and multiple remote satellite outreach locations

Professional Affiliations/Memberships:
- Accreditation Council on Optometric Education
  - Consultant, 2014-present
- American Academy of Optometry (AAO)
  - Fellow; Class of 2009
- American Optometric Association (AOA)
- Armed Forces Optometric Society (AFOS)
- European Academy of Optometry and Optics (EAOO)
  - Candidate for Fellowship
- Fellowship of Christian Optometrists (FCO)
- Minneapolis VAMC Medical Staff Association
  - Steering Committee, member 2010-2014
- National Association of Veteran Affairs Optometrists (NAVAO)
  - Newsletter Committee, member 2010-2014
- National Optometric Association (NOA)
  - Minnesota's NOA State Representative 2010-2012
  - National Optometric Student Association (NOSA)
    - NOSA National Vice-President: 2006-2007
    - NOSA-ICO President: 2005-2006
    - NOSA-ICO Vice-President: 2004-2005

- Volunteer Optometric Service to Humanity (VOSH)
- Journal of Rehabilitation Research and Development
  - Peer Reviewer, 2013-2014

Activities:

- VOSH Medical Mission Trip, Bamenda, Cameroon (May 2010)
- Mayo Medical School/Brighter Tomorrow's Winter Warmth Festival (Jan 2009 & Jan 2010)
  - Fun day of activities for children battling cancer and their families
  - Volunteer
- Veteran Affairs Disaster Emergency Medical Personnel System (DEMPS)
  - Volunteer (Aug 2009-present)
- FCO Optometry Mission Trip, Port Au Prince, Haiti (Feb 2007)
- SVOSH Medical Mission Trip, Addis Addaba, Ethiopia (Mar-Apr 2006)
- FCO Optometry Mission Trip, Tegucigalpa, Honduras (Apr 2003 & Nov 2004)

Honors/Rewards:

- Recognition of Excellence in Teaching as Clinical Assistant Professor, Western University Health Sciences/College of Optometry (2015-2016 Academic Year)
- Nomination for Medical Staff Clinical Excellence Award (2012 & 2013)
- Recognition for Outstanding Dedication and Service as Adjunct Assistant Professor, University of Missouri – St. Louis (2010-2011 Academic Year)
- Journal of the American Optometric Association: Optometry's Eagle Award (Nov 2010)
- Certificate of Appreciation (July 2009)
  - Department of Veterans Affairs – VISN 23
    - Awarded for participation in VISN 23 Blind and Low Vision Continuum of Care Conference
- Recognition for Clinical Excellence (May 2007)
- Derald Taylor Low Vision Award (May 2007)
- Clinical Dean's List (summer 2005; summer & fall 2006, winter & spring 2007)
- Academic Dean's List (fall 2004)
- Wildermuth Leadership Award/Scholarship (Aug 2006)
- Vistakon Acuvue Eye Health Advisor Citizenship Scholarship (Jan 2006)
- NOSA Service Award/Scholarship (Aug 2004)

Publications:

Pruitt JA. The Management of Homonymous Hemianopsia Secondary to Hemispheric Ischemic Cerebral Vascular Accident. Accepted for publication by Review Optometry (July 2010)

Rittenbach TL, Pruitt JA. A Roundup of Recently Approved Ophthalmic Drugs (and their Use in Practice.) Rev Optom. 2014. 151(2):22-28.

Pruitt JA. Management strategies for patients with AION. Rev Optom. 2011. 148(6):57-65.

Pruitt JA. Neuro-Optometric Rehabilitation Association Program Summary. Optimum VA: The Official Newsletter of the National Association of VA Optometrists Summer 2010.

Pruitt JA, Ilsen P. On the frontline: What an optometrist needs to know about myasthenia gravis. Optometry 81(9): 454-460.

Pruitt JA, Sokol T, Maino D. Fragile X Syndrome and the Fragile X-associated Tremor/Ataxia Syndrome. Eye Care Review: Ophthalmology, Optometry, Opticianry 4(2): 17-23

Posters/Presentations

Pruitt JA. The Curious Case of the Functionally Legally Blind Patient with 20/25 (6/7.5) Visual Acuity. *Accepted into American Optometric Association Annual Meeting: Optometry's Meeting (2012) Poster Session.*

Pruitt JA, Prussing N. Successfully Treated Horizontal Diplopia Returns with Subsequent Traumatic Brain Injury. *Accepted into American Optometric Association Annual Meeting: Optometry's Meeting (2012) Poster Session.*

Pruitt JA, Prussing N. The Curious Case of the Functionally Legally Blind Patient with 20/25 (6/7.5) Visual Acuity. European Academy of Optometry and Optics Annual Meeting (2012) Poster Session.

Pruitt JA, Prussing N. Successfully Treated Horizontal Diplopia Returns with Subsequent Traumatic Brain Injury. European Academy of Optometry and Optics Annual Meeting (2012) Case Presentation Session.

Pruitt JA, Prussing N. Traumatic Brain Injury Resulting in Horizontal Diplopia Resolved 5 Years Later with 12 Weeks of Vision Therapy. Minnesota Optometric Association Annual Meeting (2012) Poster Session.

Pruitt JA, Wiley LM. Overcoming Mental Barriers in Visual Rehabilitation. American Optometric Association Annual Meeting: Optometry's Meeting (2011) Poster Session.

Pruitt JA, Prussing N. Traumatic Brain Injury Resulting in Horizontal Diplopia Resolved 5 Years Later with 12 Weeks of Vision Therapy. European Academy of Optometry and Optics Annual Meeting (2011) Poster Session.

Pruitt JA. Overcoming Mental Barriers in Visual Rehabilitation. European Academy of Optometry and Optics Annual Meeting (2011) Case Presentation Session.

Pruitt JA, Wiley LM. Overcoming Mental Barriers in Visual Rehabilitation. Minnesota Optometric Association Annual Meeting's (2011) Poster Session

Pruitt JA, Ilsen P, Yeung C. Ptosis Crutch: Success Treating Myogenic Ptosis Secondary to Myasthenia Gravis. American Optometric Association (AOA) 2008 Optometry Meeting Poster Session

Pruitt JA, Ilsen P. Ptosis Crutch: Success Treating Myogenic Ptosis Secondary To Myasthenia Gravis. Southeastern Congress of Optometry (SECO) 2008 Multimedia Poster Session

Lectures and Other:

Riverside-San Bernardino County Indian Health, Inc.: Eye Care Rounds (Nov 2016)
- Ptosis Crutch: Success Treating Myogenic Ptosis Secndary to Myasthenia Gravis
- CA Board of Optometry-approved CE

Riverside-San Bernardino County Indian Health, Inc.: Eye Care Rounds (Sept 2016)
- Visual Fields
- CA Board of Optometry-approved CE

Riverside-San Bernardino County Indian Health, Inc.: Eye Care Rounds (July 2016)
- Ethical Concerns with Short-term Mission Trips
- CA Board of Optometry-approved CE

Riverside-San Bernardino County Indian Health, Inc.: Eye Care Rounds (July 2016)
- Systemic Urgencies and Emergencies
- CA Board of Optometry-approved CE

Riverside-San Bernardino County Indian Health, Inc.: Eye Care Rounds (Mar 2016)
- Episcleritis, Scleritis, and Iritis
- CA Board of Optometry-approved CE

Illinois College of Optometry: Practice Opportunities Symposium (Mar 2011)
- Represented and presented on VA Optometry
- Participated in panel discussion on "Residency-trained Optometrists"

University of Minnesota: Pre-Optometry Club (Oct. 2010)

- Presentation on the profession of Optometry
- Presented and represented VA Optometry and NOA

Illinois College of Optometry: Capstone Ceremony (May 2010)

- Represented and presented on VA Optometry

Illinois College of Optometry: Practice Opportunities Symposium (Mar 2010)

- Participant in Residency-trained Speaker's Panel
- Represented and presented on VA Optometry

Illinois College of Optometry: White Coat Ceremony/Smart Business Program (Sept 2009)

- Participant on Recent Graduate Speaker's Panel