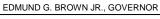


STATE BOARD OF OPTOMETRY

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:

Provider Name:

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

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

1-419/4272064/4425508/50

	AGENCY	GOVERNORE	DMUND G, BROWN JR.
OPTOMETRY AND L3 P(916) 575-7170 F (916) 5	75-7292 www.optometry.ca.gov	
CONTINU	ING EDUCATI	ON COURSE APPROVAL	<u></u>
\$50 Mandatory Fee	APPLI	CATION	
Pursuant to California Code of Regulati receiving the applicable fee, the reques specified in CCR § 1536(g).	ions (CCR) § <u>1536,</u> th sted information below	ne Board will approve continuing education and it has been determined that the contract the cont	on (CE) courses afte urse meets criteria
n addition to the information requested presentation materials (e.g., PowerPoir presentation date. Please type or print clearly.	below, please attach t presentation). Appl	a copy of the course schedule, a detaile lications must be submitted 45 days prio	ed course outline an r to the course
Course Title	,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,	Course Presentation Date	
Common Subclinical Retinal Diseases		09/08/2016	
	Course Provider (Contact Information	
Provider Name			
Margarette	Recalde, (DD R.	
(First) Provider Mailing Address	(Last) (Mide	lle)
Provider Email Address mrecalde@	eyeqvc.com		
Will the proposed course be open to	all California licens	ed optometrists?	VIYES 🗆 NO
of course content and attendance as	the Board requires	or attending licensee such records , for a period of at least three years	n ves ⊡ no
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Central California Optometric Society

December 30, 2016

Dear California Board of Optometry,

This letter is in response to your request regarding "Common Subclinical Retinal Diseases: Diagnosis and Management Utilizing OCT". As you requested, this letter addresses the reason why the application was not submitted within the 45-day deadline prior to the course date.

There was a miscommunication between the sponsor for the dinner program and the Central California Optometric Society. Each party thought the other party had communicated to the speaker, Dr. Ken Ekelund that the lecture needed to be submitted for CE approval ahead of time. In reality, the speaker wasn't contacted until later. By the time the speaker was informed, it was already very close to the 45-day deadline. The speaker did his best to submit the outline as soon as possible.

Thank you again for your patience. If you have any questions, please feel free to contact me.

Respectfully,

llarge Recalde 180

Margie R. Recalde, OD, FAAO CCOS President

Title:

Common Subclinical Retinal Diseases: Diagnosis and Management Utilizing OCT

Speaker:

Kenneth E Ekelund, O.D. Ekelund Vision Center 2190 Larkspur Lane, Suite 200 Redding, CA 96002

Summary of Presentation:

Optical Coherence Tomography (OCT) is an extremely powerful tool in the identification and assessment of retinal abnormalities. With Spectral Domain OCT, this technology provides quantitative and repeatable measurements to identify diseases of the retina, monitor progression, and evaluate for pharmacological/surgical interventions. The goal is to advance the optometrist's knowledge and clinical/professional skills on the OCT and to aid their decision-making skills for retinal diseases.

1 Common Subclinical Retinal Diseases: Diagnosis and Management Utilizing OCT

Kenneth E Ekelund, O.D. Ekelund Vision Center 2190 Larkspur Lane, Suite 200 Redding, CA 96002

2 OCT History/Paradigm Shift

- •OCT first introduced in 1991
- •OCT provides cross-sectional images of the retina using the reflectivity of light waves to obtain a profile of the retinal layers.
- •Specialized anterior segment OCT machines became available in 2005 and the introduction of Spectral (Fourier) Domain OCT (SD-OCT, FD-OCT) technology now provides greater tissue resolving power, significantly higher scan density, and faster data acquisition than original Time Domain OCT.

3 Review of Retinal Anatomy with gray scale retinal layers

4 OCT Importance in daily practice

- •OCT is an extremely powerful tool in the identification and assessment of retina abnormalities
- •The high resolving power (10um Time Domain, 5um Spectral Domain) provides excellent detail for evaluating the vitreo-retinal interface, neurosensory retinal morphology, and the RPE-choroid complex.
- •The ability to perform volumetric and retinal thickness analysis also provides a quantitative and repeatable method to evaluate surgical and pharmacological interventions.

5 🔲 Review of Retinal History

• Time domain systems acquire approximately 400 A-scans per second using 6 radial slices oriented 30 degrees apart. Because the slices are 30 degrees apart, care must be taken to avoid missing pathology between the slices.

- Spectral domain technology, on the other hand, scans approximately 20,000-40,000 scans per second. This increased scan rate and number diminishes the likelihood of motion artifact, enhances the resolution and decreases the chance of missing lesions. 6 Choroid Complex review The choroid is a major vascular layer of the eye and provides oxygen and nourishment to the outer layers of the retina. •Changes in the choroid, in particular its thickness, have been hypothesized to be of critical importance in the pathophysiology of a number of retinal diseases. 7 Mueller glia cell review and function Müller glia are the major glial component of the retina. They are one of the last retinal cell types to be born during development and they function to maintain retinal homeostasis and integrity. 8 3 4 Categories of Retinal Disease with OCT Macular Degeneration ERM/Vitreo-Macular Traction Macular Edema Macular Hole 9 Diagram review of macula 10 Comparison of fundus photo vs cross section OCT view of posterior pole
- 11 Outer layer retina view of cross section OCT IS/OS junction and its importance
- 12 Review of PVD Partial PVD (APVD) development and stats
 - •According to autopsy studies, PVD is present in fewer than 10% of persons younger than 50 years
 - •Has been found in at least one eye in 27% of individuals aged 60 to 69 and in 63% of subjects aged 70 and older.
 - •Clinical studies also reveal a low incidence of PVD in

2

individuals younger than 50.

13 Review of PVD Partial PVD (APVD) development and stats

- Posterior vitreous detachment is believed to develop after liquefied vitreous passes abruptly into the subhyaloid space and separates the posterior hyaloid from the retina.
- •However, the actual process in older persons with healthy eyes remains unknown, because of the difficulty in identifying its initial stage.
- •With SD-OCT, we can improve our understanding of the process of PVD.

14 Review of PVD Partial PVD (APVD) development and stats

- •Gel liquefaction that exceeds the degree of vitro-retinal dehiscence results in anomalous PVD (APVD).
- APVD varies in its clinical manifestations depending upon where in the fundus vitreo-retinal adhesion is strongest.

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- At the periphery, APVD results in retinal tears and detachments.
- In the macula, APVD causes vitreo-macular traction syndrome, results in vitreoschisis with macular pucker or macular holes, or contributes to some cases of diabetic macular edema.
- •At the optic disc and retina, APVD causes vitreo-papillary traction and promotes retinal and optic disc neovascularization.

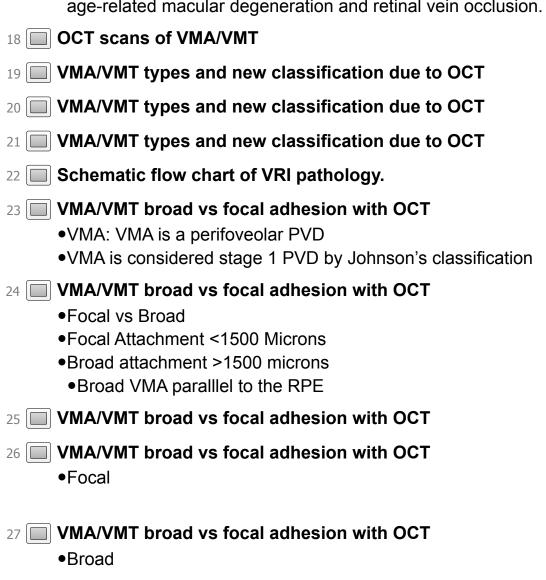
16 Review of PVD Partial PVD (APVD) development and stats

17 APVD creating multiple VRI maculopathies

- •The physics of anomalous PVD has the potential to generate forces which split the posterior vitreous cortex causing vitreoschesis.
- •When this phenomenon occurs in the periphery, tractional

forces increase the risk of retinal tears and detachments.

- •When it occurs in or adjacent to the macula, it has the potential to induce wrinkling of the neurosensory retina referred to as macular pucker.
- •Vitreoschisis may contribute to the process of macular hole formation and increase the risk diabetic macular edema.
- •Other associations appearing concurrently with VMT include age-related macular degeneration and retinal vein occlusion.



28 New Classification of macular holes due to OCT

7

29 Pre op OCT to create better post op outcomes and expectation with OCT
30 OCT thickness maps of macula importance for managing pre and post op retinal pathology
31 D What makes a good candidate for VMT treatment with Jetrea
 FDA Approval of Jetra (Ocriplasmin) 2012 Is the first and only FDA-approved nonsurgical treatment for symptomatic VMA.
•Jetrea has proteolytic activity against protein components of the vitreous body and the vitreoretinal interface (VRI) (e.g. laminin, fibronectin and collagen), thereby dissolving the protein matrix responsible for the vitreomacular adhesion (VMA).
 Success rates for release of traction within 28 days was 26.1% as compared to 10.1% in the placebo group
What makes a good candidate for VMT treatment with Jetrea
 Also patients who have mild to moderate symptomatic VMA, and also have good visual acuity. Patients may be experiencing metamorphopsia, but test 20/40 or better on a Snellen visual acuity chart.
33 Measurement for macula hole to determine Jetrea vs PPV
What makes a good candidate for VMT treatment with Jetrea
³⁵ Types of Lamellar Holes interpretation and/management with OCT
³⁶ Types of Lamellar Holes interpretation and/management with OCT
37 Descudohole interpretation and management with OCT

tissue loss.

- •Clinical features: Altered light reflex and darker appearance of the fovea
- •Optical coherent tomography (OCT) demonstrates:
 - Steepening of foveal contour
 - •Full thickness retinal tissue is present
 - •Reflective epiretinal membrane layer is present on the surface of the retina

38 Seudohole interpretation and management with OCT

- •Fluorescein angiography often reveals normal fluorescence except if traction-induced retinal vascular disruption present.
- •Management:
 - Treatment of the underlying causes of epiretinal membrane.
 - •Observation of progressive contraction, which may lead to macular edema.
 - Surgical vitrectomy to peel the epiretinal membrane may be indicated in patients with worsening vision of 20/80 or less.

39 Seudohole interpretation and management with OCT

40 Dest op macular hole interpretation with OCT

- •The length of time a patient has experienced symptoms is key to predicting likelihood of visual improvement. Less than 6 months is an ideal duration.
- To facilitate macular hole closure, all tractional forces surrounding the open hole should be removed.
- •Vitrectomy for macular hole repair has proven highly successful through a number of techniques. Pharmacologic vitreolysis with ocriplasmin is another treatment option, although the effects of ocriplasmin on visual acuity may be unpredictable.
- 41 Dost op macular hole interpretation with OCT

42 Dost op macular hole interpretation with OCT

43 ERM/Macular Pucker interpretation and management with

7

macular thickness maps with OCT

- •Epiretinal membrane (ERM) is a disorder of the vitreomacular interface characterized by symptoms of decreased visual acuity and metamorphopsia.
- •OCT has proven to be more sensitive than clinical examination for the diagnosis of ERM.
- •It has been estimated that 30 million people of advanced age in the US have an ERM in at least one eye.
- •Most ERMs occur in individuals older than 50 years, and the prevalence of ERM increases as age increases

44 ERM/Macular Pucker interpretation and management with macular thickness maps with OCT

- Management
 - Vitrectomy
 - Epiretinal Membrane Peel
 - •Internal Limiting Membrane Peel
- •Complications
 - The most common complication, occurring in 12% to 68% of cases, is accelerated nuclear sclerosis of crystalline lens.
 - •Recurrent ERM occurs in 3% to 12% of patients
 - •Retinal detachment occurs in 3% to 14%. This may develop within 1 month postoperatively and may be related to intraoperative retinal breaks.
- 45 ERM/Macular Pucker interpretation and management with macular thickness maps with OCT
- 46 ERM/Macular Pucker interpretation and management with macular thickness maps with OCT
- 47 ERM/Macular Pucker interpretation and management with macular thickness maps with OCT
- 48 ERM/Macular Pucker interpretation and management with macular thickness maps with OCT

49 Middle layer retina showing CME interpretation/ management with OCT

- •Diabetic Macular Edema (DME) versus Pseudophakic Cystoid Macular Edema (PCME)
- •DME: More ONL cysts, no subretinal fluid, presence of hard exudates, microaneurysms, and ganglion cell layer and/or retinal nerve fiber layer cysts
- PCME: No ERM, solely INL cysts
- •DME: PCME:

50 Middle layer retina BVO interpretation/management with OCT

- Vision loss from retinal vein occlusions is secondary to macular edema and if ischemic, has the risk to develop neovascularization and neovascular glaucoma.
- •OCT can assist in detecting macular edema secondary to retinal vein occlusions and track response to treatment.
- Macular edema in retinal vein occlusions causes diffuse leakage, especially in CRVO.
- The macular edema is driven by ischemia and release of vascular endothelial growth factors (VEGF) that increases the inner retinal blood-brain barrier causing leakage of intraretinal fluid.

51 Middle layer retina BVO interpretation/management with OCT

- Macular edema secondary to branch retinal vein occlusion will respond to argon grid laser and anti-VEGF therapies (Lucentis, Eylea, avastin).
- Macular edema secondary to central retinal vein occlusion will respond to anti-VEGF therapies (Lucentis, Eyelea, and avastin) and intravitreal dexamethasone implant (Ozurdex).

52 Middle layer retina BVO interpretation/management with OCT

53 Middle layer retina BVO interpretation/management with OCT
54 Review of the choroid complex anatomy.
 55 Early stage dry ARMD interpretation/management One in 10 people over the age of 60 have some form of AMD. That ratio increases to one in four people over 70. AMD is the leading cause of legal blindness for people over age 55 in the Western world — more than 5 million new cases a year reported in Europe and North America alone.
 56 Early stage dry ARMD interpretation/management As defined by the AREDS, early AMD is characterized by small drusen (<63 μm), few medium drusen (63–125 μm), and/or minimally detected or no pigment epithelial abnormalities in the macula. Patients in this category have a low risk of progressing to advanced AMD after 5 years in either eye.
57 🔲 Early stage dry ARMD interpretation/management
 58 Early stage dry ARMD interpretation/management Intermediate Dry ARMD Defined by the AREDS as having extensive medium drusen (63–124 μm) or one or more large drusen (³125 μm in diameter) in one or both eyes. The progression to advanced AMD at 5 years in this group is approximately 18% according to the original AREDS. However, for patients with large drusen in one eye, the rate of development of advanced AMD at 5 years is 6.3%, whereas the rate for patients with multiple bilateral large
drusen increases to 26% at 5 years <u>.</u>
59 Early stage dry ARMD interpretation/management
60 Late stage geographic degeneration OCT interpretation/ management

- Advanced ARMD
 - •Geographic atrophy of the RPE involving the foveal center
 - •Neovascular maculopathy that includes the following:
 - •Choroidal neovascularization (CNV) defined as pathologic angiogenesis originating from the choroidal vasculature that extends through a defect in Bruch's membrane.
 - •Serous and/or hemorrhagic detachment of the neurosensory retina or RPE
 - •Retinal hard exudates (a secondary phenomenon resulting from chronic intravascular leakage)
 - •Subretinal and sub-RPE fibrovascular proliferation
 - •Disciform scar (subretinal fibrosis)
- 61 Late stage geographic degeneration OCT interpretation/ management
- 62 Late stage geographic degeneration OCT interpretation/ management
- 63 Late stage geographic degeneration OCT interpretation/ management
 - •Treating the wet form of macular degeneration may involve the use of anti-VEGF treatment, thermal laser treatment or photodynamic therapy (PDT).
 - •Treatment of wet macular degeneration generally reduces but does not eliminate-- the risk of severe vision loss.
 - •It is important to remember that only about 10 percent of all macular degeneration cases are exudative, or wet form, and about 75 percent of these cases cannot be treated.

64 Review histology of Wet ARMD (CNV)

- •New capillaries and fibroblasts originate from the choroid and grow through a defect in the Bruch membrane into the subretinal space or the sub-RPE space. Reactive hyperplastic RPE is present at the advancing edge of CNV.
- •Specimens obtained from surgical excision of CNV reveal that the most common cellular components are vascular

endothelium and RPE. These were present in more than 85% of samples.

•Fibrocytes and macrophages also have been identified in more than 50% of specimens. Extracellular components include collagen and fibrin. VEGF has been identified in the specimens obtained during submacular surgery.

65 Wet ARMD interpretation/management with OCT

- Targeting VEGF allows a two-hit strategy: antiangiogenesis +antipermeability
 - Avastin (bevacizumab)
 - •Lucentis (ranibizumab)
 - •Eylea (aflibercept)
 - Macugen (pegaptanib)

66 Wet ARMD interpretation/management with OCT

- •The Macular Photocoagulation Study (MPS) proved the efficacy of laser photocoagulation in the treatment of CNV secondary to ARMD.
- •PDT uses light-activated drugs and nonthermal light to achieve selective destruction of CNV with minimal effects on the surrounding normal tissues.
- •Uncontrolled studies have recommended surgical excision of subfoveal CNV via pars plana vitrectomy. The goal is to remove CNV but to leave the underlying RPE and choriocapillaris intact.

67 Wet ARMD interpretation/management with OCT

68 CSR Interpretation/management with OCT

- •The disease classically affects men between the ages of 20 and 50 and has been associated with corticosteroid exposure, phosphodiesterase inhibitor use, obstructive sleep apnea and "type A" personality traits.
- •The characteristic finding is a posterior neurosensory retinal detachment caused by leakage of fluid from the level of the retinal pigment epithelium.

69 Retinal Detachment with OCT
 Macula On RD with VA 20/20. Emergency surgery within 24 hours.
70 Retinal Detachment with OCT
 Anterior seg angles with OCT Gonioscopy: If the posterior trabecular meshwork can't be seen for 180 degrees or more, that's considered to be an occludable angle and the patient should receive prophylactic laser.
72 Pachymetry with OCT
73 🔲 Keratoconus using the OCT pachymetry
74 D Normal RNFL interpretation with OCT
75 OHT interpretation/management with OCT
 Ocular hypertension is a condition in which the following criteria are met:
 An intraocular pressure greater than 21 mm Hg in one or both eyes, as measured by applanation tonometry on 2 or more occasions
 Absence of glaucomatous defects on visual-field testing Normal appearance of the optic disc and nerve fiber layer Anatomically normal, open angles on gonioscopy
 Absence of ocular conditions contributing to the elevation of pressure, such as narrow angles, neovascular conditions, and uveitis
76 🔲 Early glaucoma interpretation/management with OCT
 The advantage of OCT in early glaucoma is that it can detect damage that is not measurable with any other technology. Need to lose approximately 20 percent of her retinal nerve fiber layer before a visual field defect is likely to be present. Helpful to compare one eye to the other and look for asymmetry. Asymmetry, especially in the supero- or infero-

1/1/17

temporal regions, provides a clue that it could be early glaucoma.

- 77 Late stage glaucoma interpretation/management with OCT
 - •In the advanced stages of glaucoma, the standard 24-2 visual fields may no longer be very sensitive to subtle progression.
 - Imaging technologies such as SD-OCT also encounter problems with advanced optic nerve damage. On the SD-OCT measurements of the RNFL, the so-called floor effect becomes relevant in eyes with severe thinning. RNFL thinning levels off at approximately 40 to 50 µm, perhaps due to residual glial tissue, blood vessels, or other nonneural tissue.
- 78 MS interpretation/management with OCT
- 79 Brain trauma interpretation/management with OCT

80 Detecting PSC with OCT

- •Nuclear cataracts have less influence on OCT image quality relative posterior cataracts.
- 81 My gold standard to practice with OCT

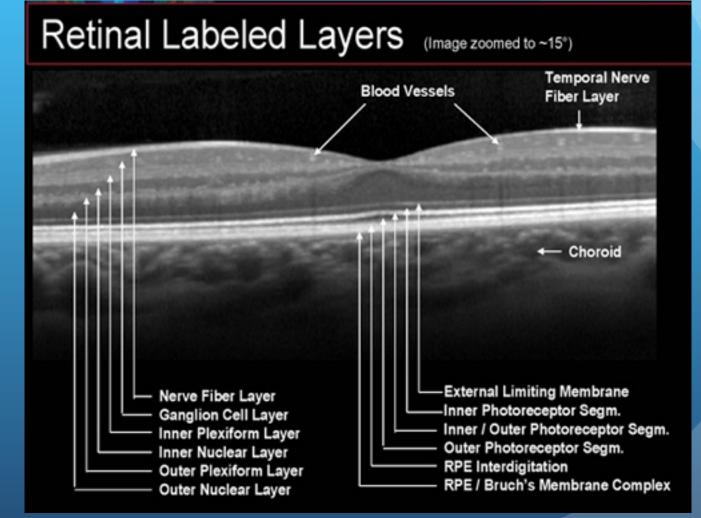
Common Subclinical Retinal Diseases: Diagnosis and Management Utilizing OCT

> Kenneth E Ekelund, O.D. Ekelund Vision Center 2190 Larkspur Lane, Suite 200 Redding, CA 96002

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Review of Retinal Anatomy with gray scale retinal layers

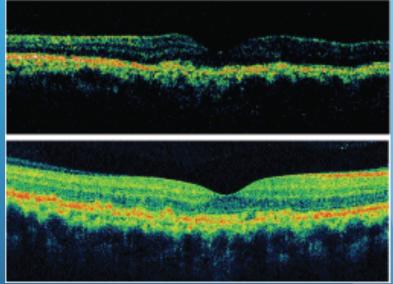


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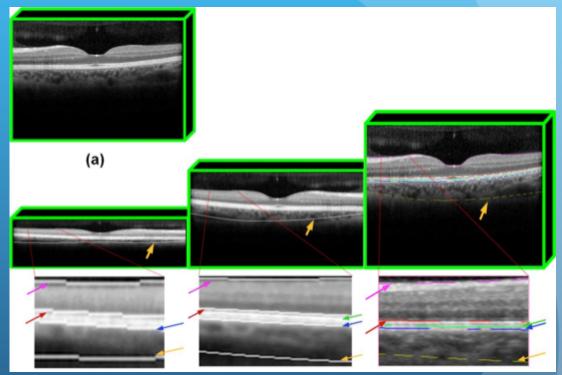
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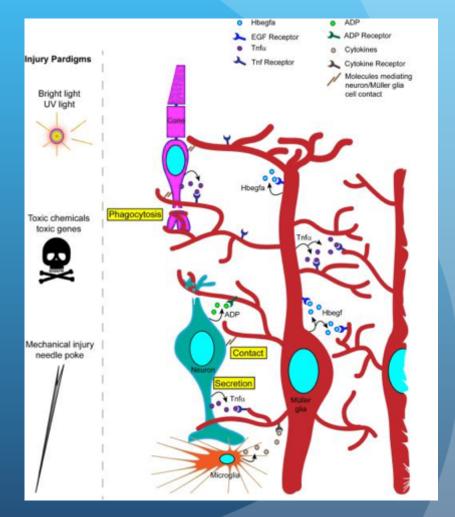
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Mueller glia cell review and function

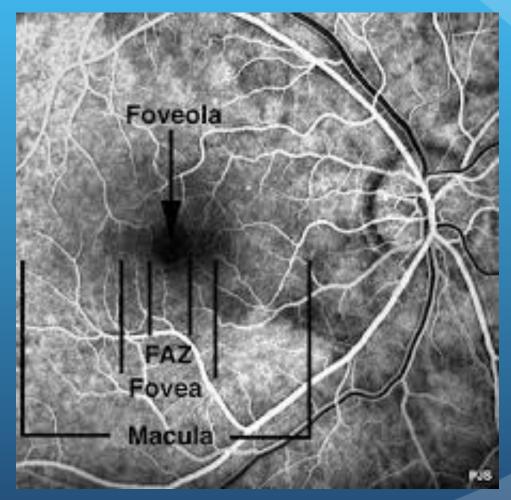
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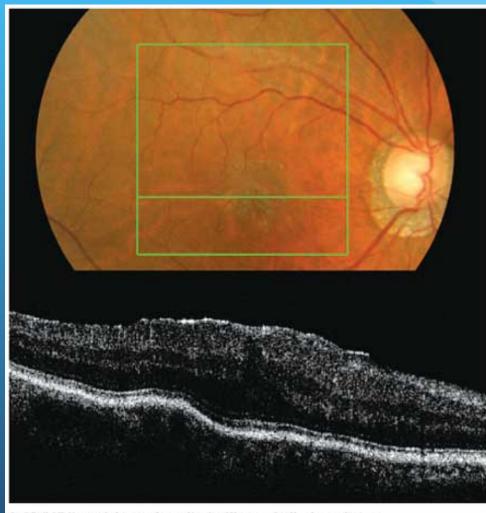
4 Categories of Retinal Disease with OCT

Macular Degeneration
ERM/Vitreo-Macular Traction
Macular Edema
Macular Hole

Diagram review of macula

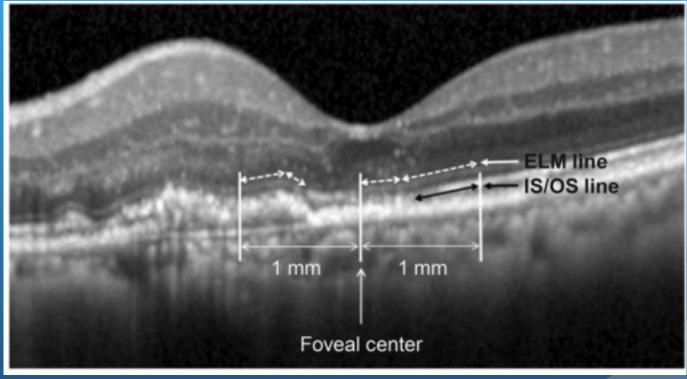


Comparison of fundus photo vs cross section OCT view of posterior pole



Outer layer retina view of cross section OCT IS/OS junction and its importance

OCT IS/OS Junction: important indicator of photoreceptor integrity



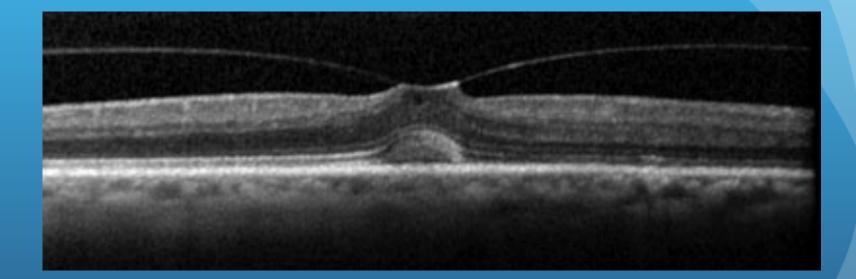
- According to autopsy studies, PVD is present in fewer than 10% of persons younger than 50 years
- Has been found in at least one eye in 27% of individuals aged 60 to 69 and in 63% of subjects aged 70 and older.
- Clinical studies also reveal a low incidence of PVD in individuals younger than 50.

- Posterior vitreous detachment is believed to develop after liquefied vitreous passes abruptly into the subhyaloid space and separates the posterior hyaloid from the retina.
- However, the actual process in older persons with healthy eyes remains unknown, because of the difficulty in identifying its initial stage.
- With SD-OCT, we can improve our understanding of the process of PVD.

 Gel liquefaction that exceeds the degree of vitro-retinal dehiscence results in anomalous PVD (APVD).

• APVD varies in its clinical manifestations depending upon where in the fundus vitreo-retinal adhesion is strongest.

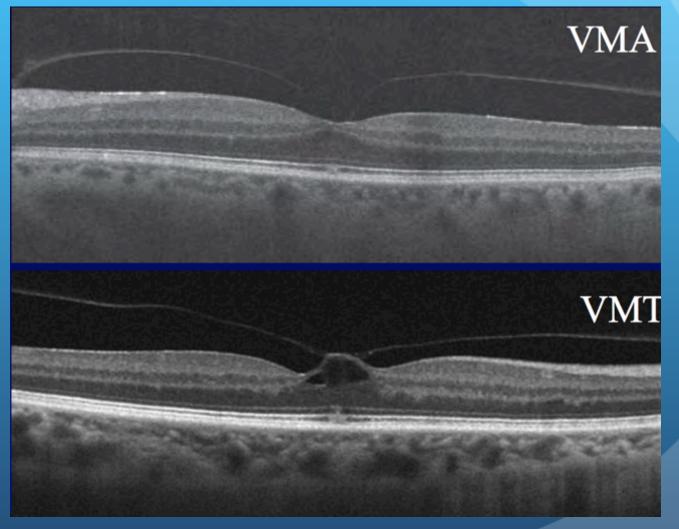
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APVD creating multiple VRI maculopathies

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- Other associations appearing concurrently with VMT include agerelated macular degeneration and retinal vein occlusion.

OCT scans of VMA/VMT



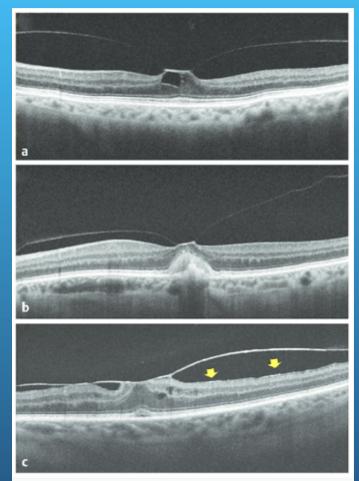
VMA/VMT types and new classification due to OCT

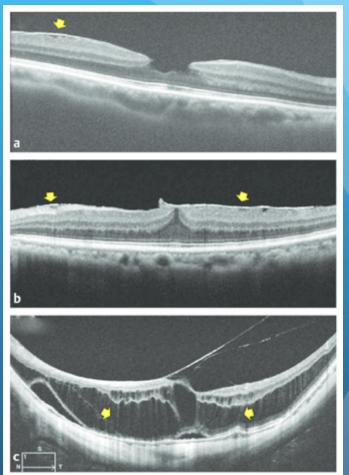
Anatomic State	Classification	Definition	Gass Classification
VMA	Size of attachment area Focal (≤ 1500 µm) Broad (> 1500 µm) Presence of associated macular abnormalities Isolated Concurrent	No detectable change in foveal contour	Stage 0
VMT	Size of attachment area Focal (≤ 1500 µm) Focal (≤ 1500 µm) Presence of associated macular abnormalities Isolated Concurrent	 Absence of full-thickness interruption of all retinal layers and vitreous attachment associated with: Distortion of foveal surface Intraretinal structural changes Elevation of the fovea above the RPE ³⁵ 	Stage 1

VMA/VMT types and new classification due to OCT

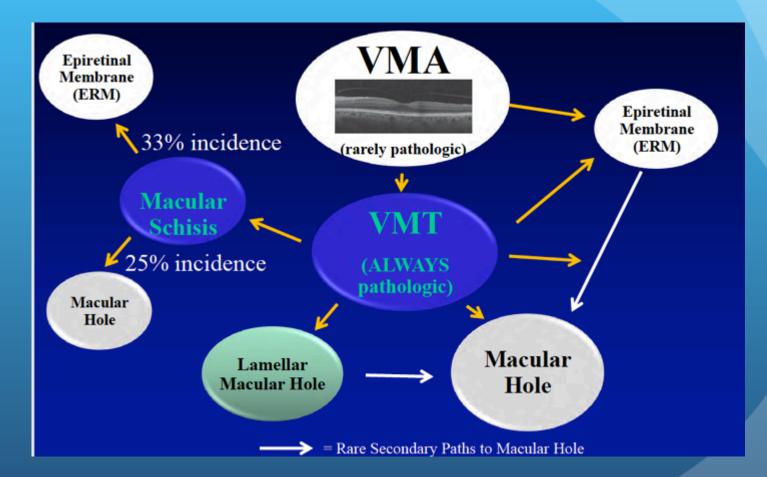
Full- thickness macular hole	Size horizontal diameter at narrowest point Small (≤ 250 µm) Medium (250-400 µm) Large (> 400 µm) Cause Primary Secondary* Presence or absence of VMT	Full-thickness foveal lesion from the ILM to the RPE	Stage 2: small or medium hole with VMT Stage 3: medium or large hole with VMT Stage 4: any size hole without VMT
Lamellar macular hole	—	Irregular foveal contour with defect in the inner fovea and an intact photoreceptor layer	
Macular pseudohole	—	Concomitant ERM with central opening and invaginated or heaped foveal edges without loss of retinal tissue	

VMA/VMT types and new classification due to OCT





Schematic flow chart of VRI pathology.



- VMA: VMA is a perifoveolar PVD
- VMA is considered stage 1 PVD by Johnson's classification

TABLE 1 STAGES* OF POSTERIOR VITREOUS DETACHMENT

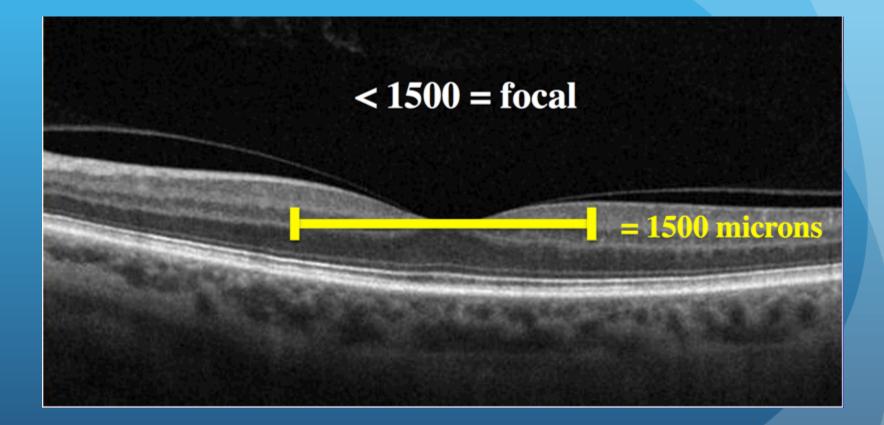
Stage 1	Perifoveal separation with adhesion of vitreous to the fovea	
Stage 2	Complete separation of vitreous from the macula	
Stage 3	Extensive vitreous separation with adhesion of vitreous to the disc	
Stage 4	Complete posterior vitreous detachment	

These stages can be studied with optical coherence tomography.^{4,17}

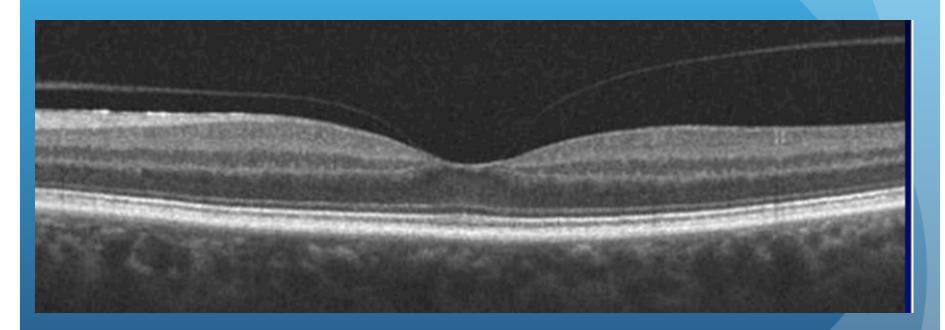
* The proposed staging levels may not imply a linear, staged progression of a posterior vitreous detachment.

Focal vs Broad
Focal Attachment <1500 Microns

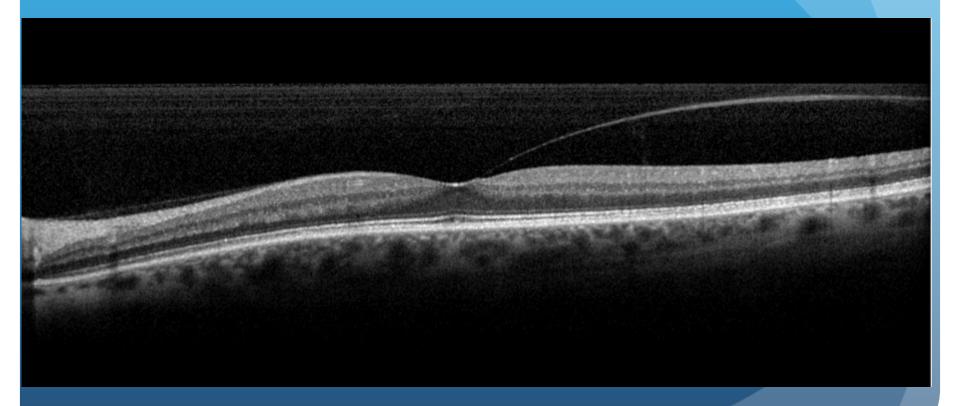
Broad attachment >1500 microns
Broad VMA parallel to the RPE



• Focal



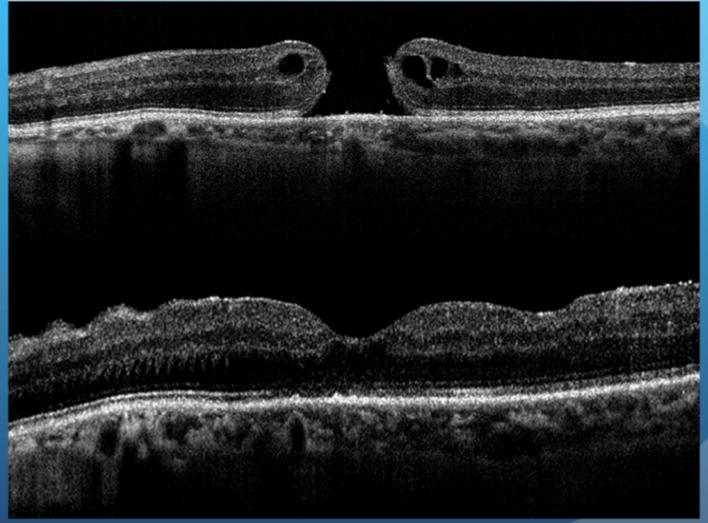
• Broad



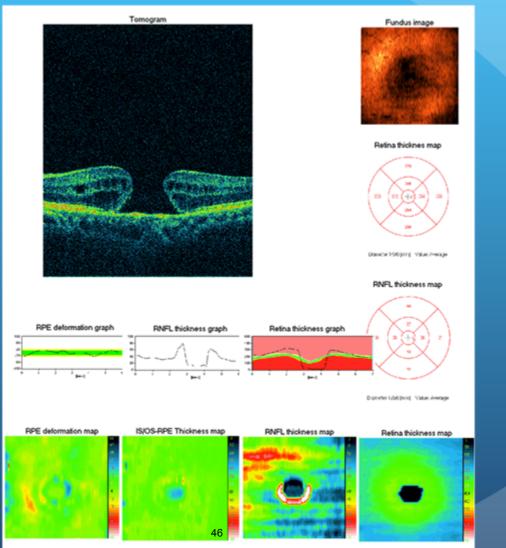
New Classification of macular holes due to OCT

Stage	Biomicroscopy (Gass)12	Interpretation (Gass) ¹²	Optical coherence tomography ^{25,43,44,47}	
Stage 0: MH			Perifoveolar detachment of posterior hyaloid with normal fovea or minor changes in the foveal contour and reflectivity ^{25,26}	
Stage 1A: Impending MH	Central yellow spot, loss of foveolar depression, no vitreofoveal separation	Early serous detachment of foveolar retina	Perifoveolar detachment of posterior hyaloid. Foveal cyst in the inner foveola, and/or foveolar detachment of the cone outer segment tip line ²⁶	
Stage 1B: Impending MH	Yellow ring with bridging interface, loss of foveolar depression, no vitreofoveal separation	Serous foveolar detachment with lateral displacement of xanthophyll	Perifoveolar detachment of posterior hyaloid. Foveal cyst extending in the outer retina, causing a break in the photoreceptor layer. "Occult Macular Hole"	
		or		
		Central occult foveolar hole with bridging contracted prefoveolar cortex		
Stage 2: MH	Eccentric oval, crescent, or horseshoe retinal defect inside edge of yellow ring	Hole (tear) in contracted prefoveolar vitreous bridging round retinal hole, no loss of foveolar retina	Hole of various size. Partial opening of the roof of the cyst, the operculum staying still attached to the edge of the	
	Central round retinal defect with rim of elevated retina, with or without prefoveolar opacity	Hole with pseudo-operculum, rim of retinal detachment	hole. Partial detachment of the posterior hyaloid, which is still attached at the operculum. The operculum contains retinal elements	
Stage 3: MH	Central round ≥400 µm diameter retinal defect, no Weiss's ring, rim of elevated retina, with or without prefoveolar opacity	Hole with pseudo-operculum, no posterior vitreous detachment	Hole of various size. Posterior hyaloid detached from the macular surface, but still attached to the optic disc, most often containing an operculum	
Stage 4: MH	Central round defect, rim of elevated retina. Weiss's ring with prefoveolar opacity	Hole with pseudo-operculum and posterior vitreous detachment from optic disc and macula	Hole of various size, with complete posterior vitreous detachment on biomicroscopy. The posterior hyaloid is not visible on OCT	

Pre op OCT to create better post op outcomes and expectation with OCT



OCT thickness maps of macula importance for managing pre and post op retinal pathology



What makes a good candidate for VMT treatment with Jetrea

- FDA Approval of Jetra (Ocriplasmin) 2012
- Is the first and only FDA-approved nonsurgical treatment for symptomatic VMA.
- Jetrea has proteolytic activity against protein components of the vitreous body and the vitreoretinal interface (VRI) (e.g. laminin, fibronectin and collagen), thereby dissolving the protein matrix responsible for the vitreomacular adhesion (VMA).
- Success rates for release of traction within 28 days was 26.1% as compared to 10.1% in the placebo group

What makes a good candidate for VMT treatment with Jetrea

Best candidates for	r ocriplasmin injection
---------------------	-------------------------

1	Phakic eyes
2	Age ≤65 years
3	No previous surgeries
4	No diabetic retinopathy
5	No ERM
6	VMA <1,500 μm
7	No macular pucker

8 FTMH <250 μm

 Also patients who have mild to moderate symptomatic VMA, and also have good visual acuity. Patients may be experiencing metamorphopsia, but test 20/40 or better on a Snellen visual acuity chart.

Measurement for macula hole to determine Jetrea vs PPV



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What makes a good candidate for VMT treatment with Jetrea



- Remove vial from -4 °F (-20 °C) freezer
- Thaw to room temperature—takes a few minutes



 Remove protective polypropylene filp-off cap from vial after thawing is completed



 Disinfect top of vial with alcohol wipe



- Use aseptic technique and included sterile #19-gauge needle
- Add 0.2 mL of 0.9% w/v sodium chloride injection (in supply pack)



 Swirl vial gently until solutions are mixed



- Inspect vial for particulate matter
- Solution must be clear and colorless without visible particles



- Employ aseptic technique to withdraw all diluted solution using included sterile #19-gauge needle
- Tilt vial slightly to ease withdrawal of JETREA
 Discard needle after
- withdrawal of JETREA
 Do not use this needle for intravitreal injection



8

- Replace needle with included sterile #30-gauge injection needle
- Carefully expel air bubbles and excess drug from syringe
- Adjust dose to 0.1 mL mark on syringe (corresponding to 0.125 mg ocriplasmin)



- The solution should be used IMMEDIATELY, as it contains no preservatives
- Discard vial and unused portion of diluted solution after single use

ADMINISTRATION

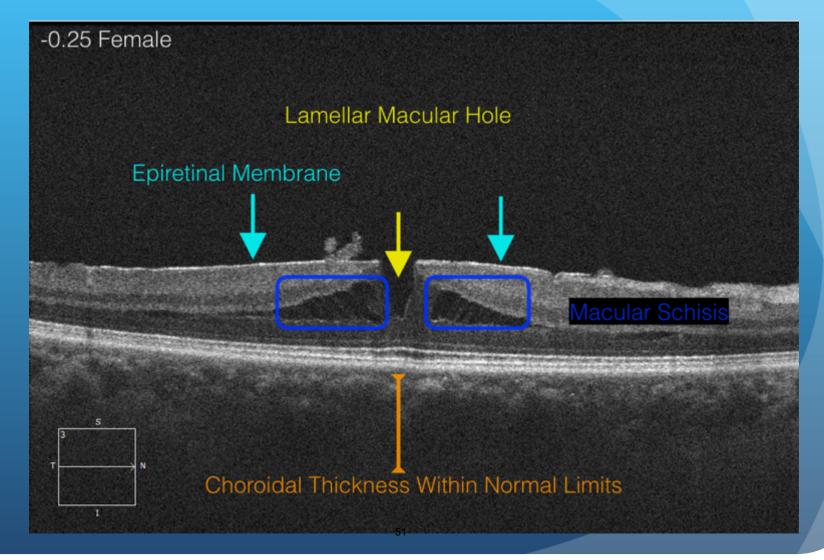
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 Carry out intravitreal injection under controlled aseptic conditions 11 • Administer adequate anesthesia and a broad-spectrum microbiocide according to standard medical practice

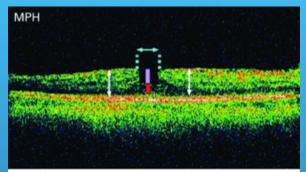
- 12 Insert injection needle 3.5 mm to 4.0 mm posterior to limbus
 - Aim toward center of vitreous cavity; avoid horizontal meridian
 - Injection volume of 0.1 mL is delivered into mid-vitreous
- Discard any unused product after injection

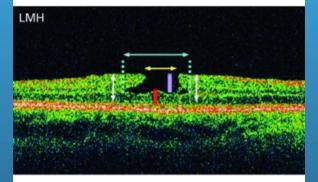
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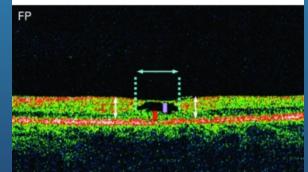
Types of Lamellar Holes interpretation and/management with OCT



Types of Lamellar Holes interpretation and/management with OCT







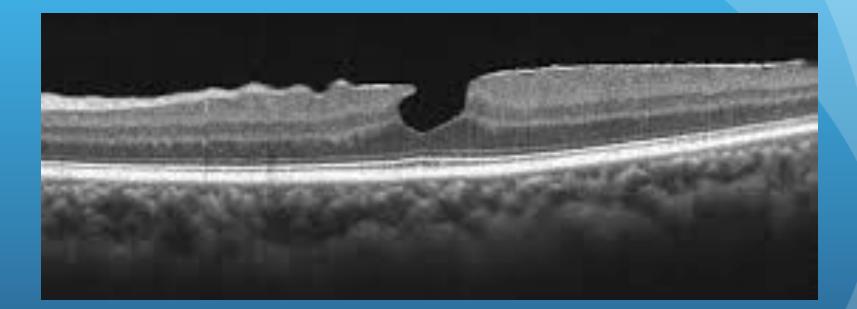
Pseudohole interpretation and management with OCT

- Retinal lesion that simulates macular hole without actual tissue loss.
- Clinical features: Altered light reflex and darker appearance of the fovea
- Optical coherent tomography (OCT) demonstrates:
 - Steepening of foveal contour
 - Full thickness retinal tissue is present
 - Reflective epiretinal membrane layer is present on the surface of the retina

Pseudohole interpretation and management with OCT

- Fluorescein angiography often reveals normal fluorescence except if traction-induced retinal vascular disruption present.
- Management:
 - Treatment of the underlying causes of epiretinal membrane.
 - Observation of progressive contraction, which may lead to macular edema.
 - Surgical vitrectomy to peel the epiretinal membrane may be indicated in patients with worsening vision of 20/80 or less.

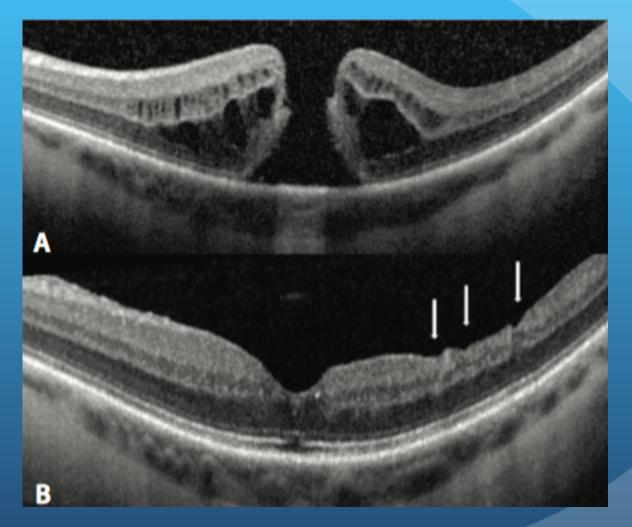
Pseudohole interpretation and management with OCT



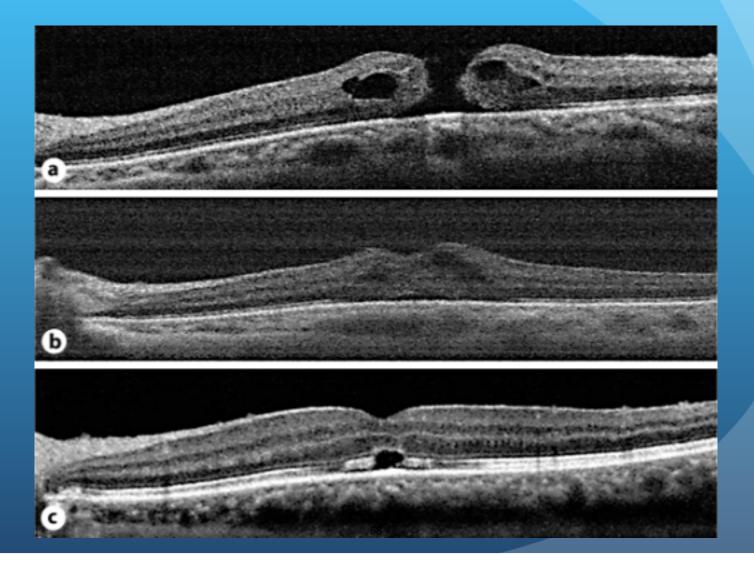
Post op macular hole interpretation with OCT

- The length of time a patient has experienced symptoms is key to predicting likelihood of visual improvement. Less than 6 months is an ideal duration.
- To facilitate macular hole closure, all tractional forces surrounding the open hole should be removed.
- Vitrectomy for macular hole repair has proven highly successful through a number of techniques.
 Pharmacologic vitreolysis with ocriplasmin is another treatment option, although the effects of ocriplasmin on visual acuity may be unpredictable.

Post op macular hole interpretation with OCT



Post op macular hole interpretation with OCT



- Epiretinal membrane (ERM) is a disorder of the vitreomacular interface characterized by symptoms of decreased visual acuity and metamorphopsia.
- OCT has proven to be more sensitive than clinical examination for the diagnosis of ERM.
- It has been estimated that 30 million people of advanced age in the US have an ERM in at least one eye.
- Most ERMs occur in individuals older than 50 years, and the prevalence of ERM increases as age increases

• Management

- Vitrectomy
- Epiretinal Membrane Peel
- Internal Limiting Membrane Peel

Complications

- The most common complication, occurring in 12% to 68% of cases, is accelerated nuclear sclerosis of crystalline lens.
- Recurrent ERM occurs in 3% to 12% of patients
- Retinal detachment occurs in 3% to 14%. This may develop within 1 month postoperatively and may be related to intra-operative retinal breaks.

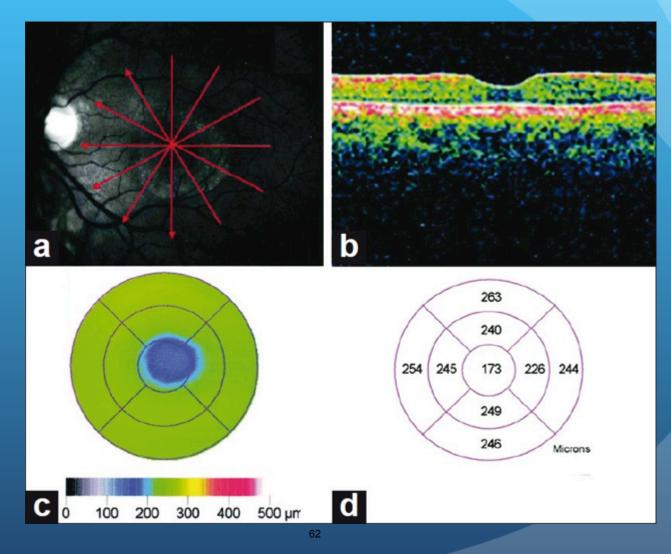
OCT-based morphologic classification of ERMs

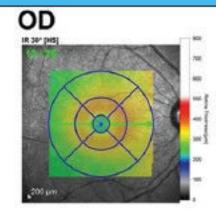
Group A: with posterior vitreous detachment

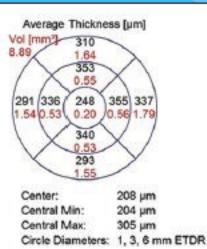
- A1 No contraction
- A2 Contraction
 - A2.1 With retinal folding
 - A2.2 With edema
 - A2.3 With cystoid macular edema
 - A2.4 With lamellar macular hole

Group B: with vitreous attachment

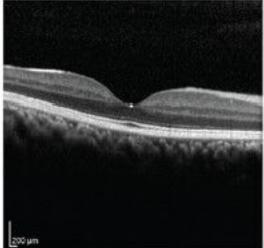
- B1 No traction
- B2 Vitreomacular traction
 - B2.1 With edema

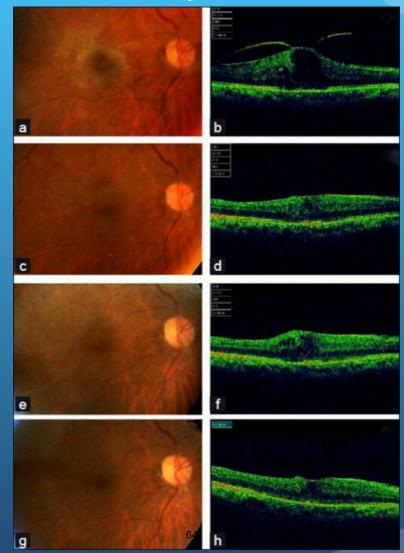






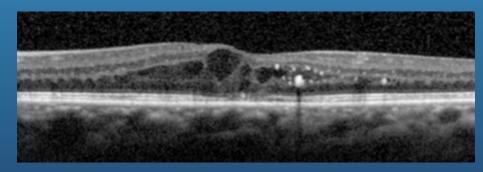
OCT 20" (6.0 mm) ART (49) Q: 25 [HS]



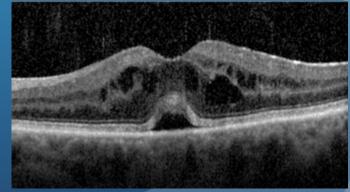


Middle layer retina showing CME interpretation/management with OCT

- Diabetic Macular Edema (DME) versus Pseudophakic Cystoid Macular Edema (PCME)
- DME: More ONL cysts, no subretinal fluid, presence of hard exudates, microaneurysms, and ganglion cell layer and/or retinal nerve fiber layer cysts
- PCME: No ERM, solely INL cysts
- DME:

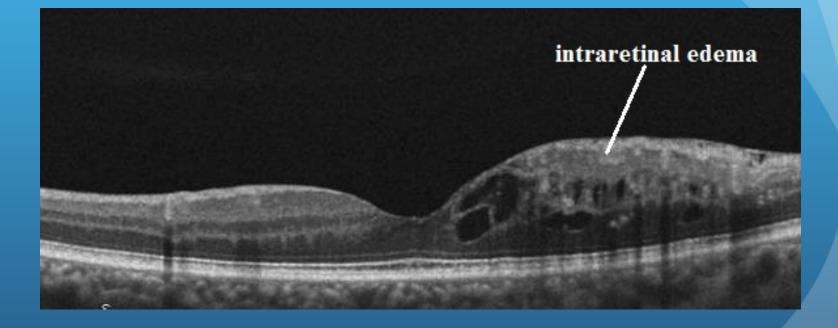


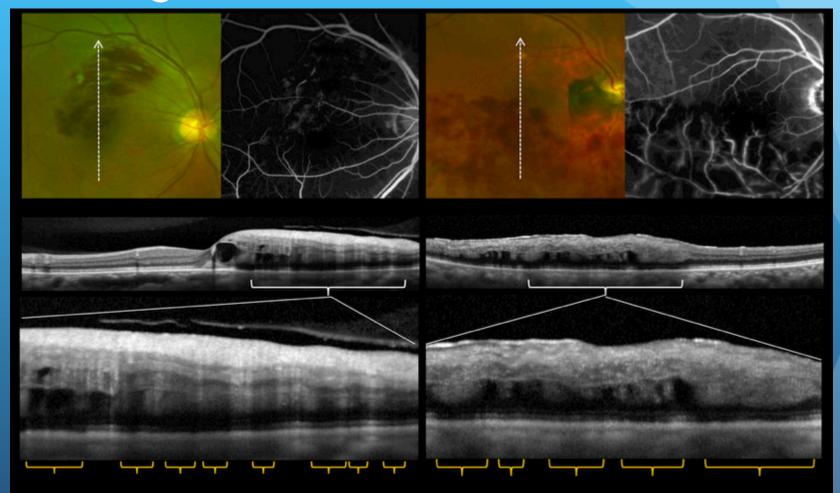
PCME:



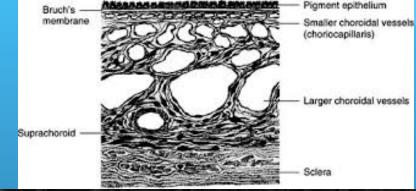
- Vision loss from retinal vein occlusions is secondary to macular edema and if ischemic, has the risk to develop neovascularization and neovascular glaucoma.
- OCT can assist in detecting macular edema secondary to retinal vein occlusions and track response to treatment.
- Macular edema in retinal vein occlusions causes diffuse leakage, especially in CRVO.
- The macular edema is driven by ischemia and release of vascular endothelial growth factors (VEGF) that increases the inner retinal blood-brain barrier causing leakage of intraretinal fluid.

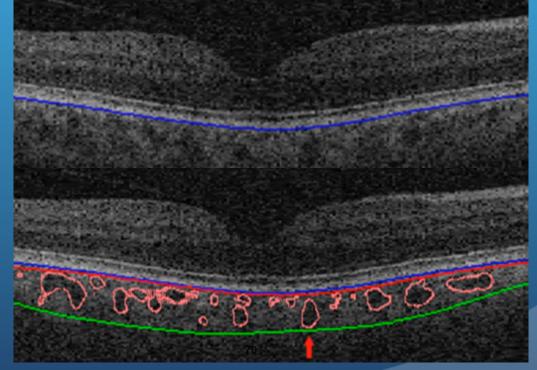
- Macular edema secondary to branch retinal vein occlusion will respond to argon grid laser and anti-VEGF therapies (Lucentis, Eylea, avastin).
- Macular edema secondary to central retinal vein occlusion will respond to anti-VEGF therapies (Lucentis, Eyelea, and avastin) and intravitreal dexamethasone implant (Ozurdex).





Review of the choroid complex anatomy.





Early stage dry ARMD interpretation/management

- One in 10 people over the age of 60 have some form of AMD.
- That ratio increases to one in four people over 70.
- AMD is the leading cause of legal blindness for people over age 55 in the Western world more than 5 million new cases a year reported in Europe and North America alone.

- As defined by the AREDS, early AMD is characterized by small drusen (<63 µm), few medium drusen (63-125 µm), and/or minimally detected or no pigment epithelial abnormalities in the macula.
- Patients in this category have a low risk of progressing to advanced AMD after 5 years in either eye.

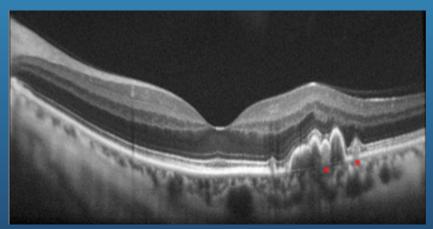


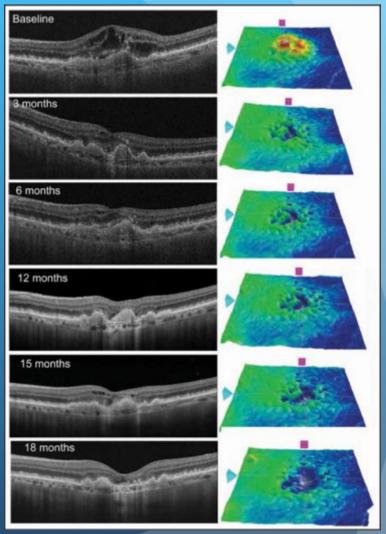
Table 1. Recommendations for AMD

Type of AMD	Characteristics	Vitamin Recommendation(s)
Early dry	A few small/medium-sized drusen; normal vision, no symptoms	Not recommended
Intermediate dry	Many medium-sized drusen; drusen under the retina; possible visual blurring	Recommended
Advanced dry	Drusen as in intermediate stage, but also breakdown of light-sensitive cells and macular tissues; blurred central vision	Recommended if in one eye only
Wet	Abnormal vessels behind the retina; macula displacement; wavy appearance to straight lines	Recommended if in one eye only
AMD: age-related macular degeneration. Source: References 5, 26.		

Intermediate Dry ARMD

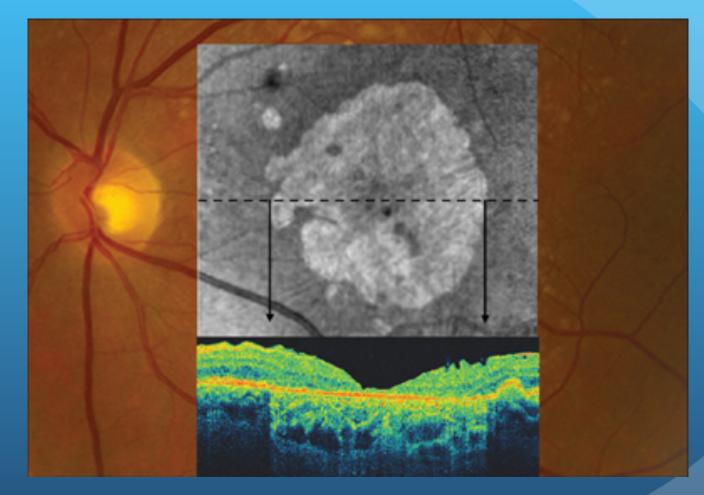
- Defined by the AREDS as having extensive medium drusen (63-124 µm) or one or more large drusen (³125 µm in diameter) in one or both eyes.
- The progression to advanced AMD at 5 years in this group is approximately 18% according to the original AREDS.
- However, for patients with large drusen in one eye, the rate of development of advanced AMD at 5 years is 6.3%, whereas the rate for patients with multiple bilateral large drusen increases to 26% at 5 years.

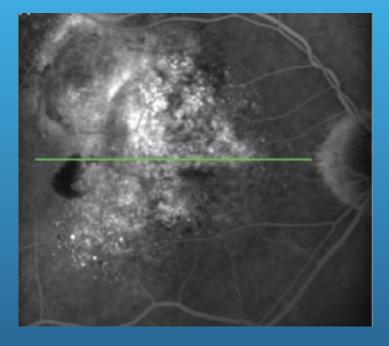


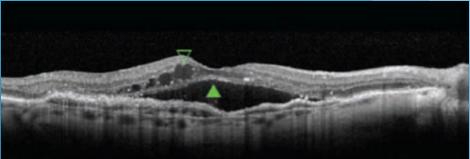


Advanced ARMD

- Geographic atrophy of the RPE involving the foveal center
- Neovascular maculopathy that includes the following:
 - Choroidal neovascularization (CNV) defined as pathologic angiogenesis originating from the choroidal vasculature that extends through a defect in Bruch's membrane.
 - Serous and/or hemorrhagic detachment of the neurosensory retina or RPE
 - Retinal hard exudates (a secondary phenomenon resulting from chronic intravascular leakage)
 - Subretinal and sub-RPE fibrovascular proliferation
 - Disciform scar (subretinal fibrosis)







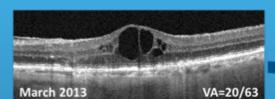
- Treating the wet form of macular degeneration may involve the use of anti-VEGF treatment, thermal laser treatment or photodynamic therapy (PDT).
- Treatment of wet macular degeneration generally reduces—but does not eliminate-- the risk of severe vision loss.
- It is important to remember that only about 10 percent of all macular degeneration cases are exudative, or wet form, and about 75 percent of these cases cannot be treated.

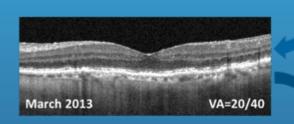
Review histology of Wet ARMD (CNV)

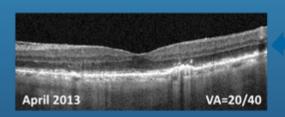
- New capillaries and fibroblasts originate from the choroid and grow through a defect in the Bruch membrane into the subretinal space or the sub-RPE space. Reactive hyperplastic RPE is present at the advancing edge of CNV.
- Specimens obtained from surgical excision of CNV reveal that the most common cellular components are vascular endothelium and RPE. These were present in more than 85% of samples.
- Fibrocytes and macrophages also have been identified in more than 50% of specimens. Extracellular components include collagen and fibrin. VEGF has been identified in the specimens obtained during submacular surgery.

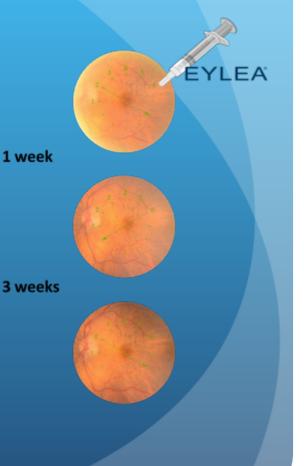
Wet ARMD interpretation/ management with OCT

- Targeting VEGF allows a two-hit strategy: antiangiogenesis +antipermeability
 - Avastin (bevacizumab)
 - Lucentis (ranibizumab)
 - Eylea (aflibercept)
 - Macugen (pegaptanib)





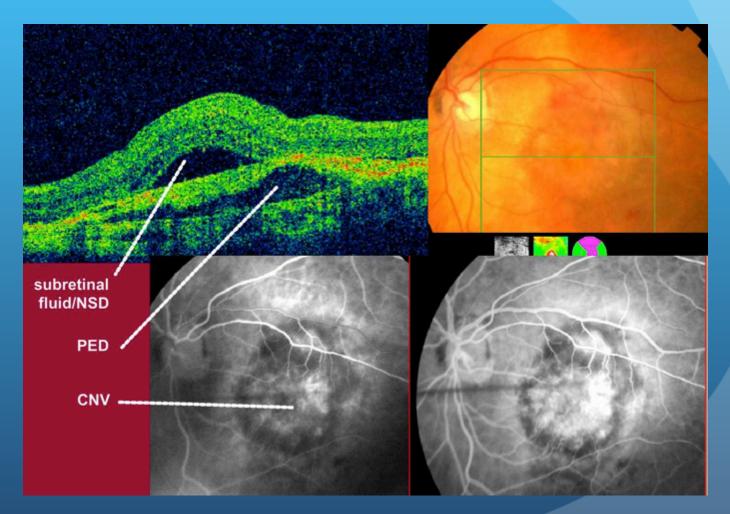




Wet ARMD interpretation/ management with OCT

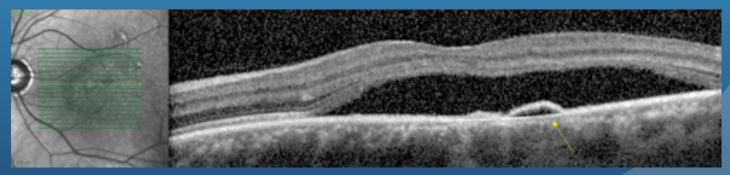
- The Macular Photocoagulation Study (MPS) proved the efficacy of laser photocoagulation in the treatment of CNV secondary to ARMD.
- PDT uses light-activated drugs and nonthermal light to achieve selective destruction of CNV with minimal effects on the surrounding normal tissues.
- Uncontrolled studies have recommended surgical excision of subfoveal CNV via pars plana vitrectomy. The goal is to remove CNV but to leave the underlying RPE and choriocapillaris intact.

Wet ARMD interpretation/ management with OCT



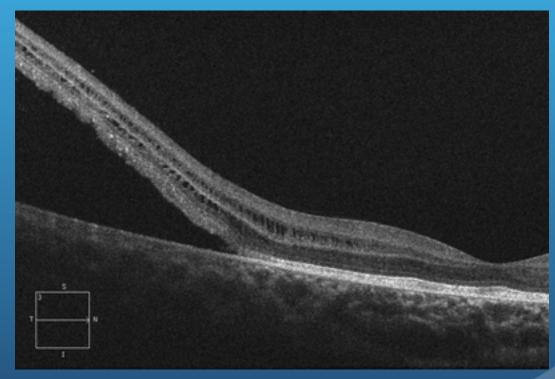
CSR Interpretation/management with OCT

- The disease classically affects men between the ages of 20 and 50 and has been associated with corticosteroid exposure, phosphodiesterase inhibitor use, obstructive sleep apnea and "type A" personality traits.
- The characteristic finding is a posterior neurosensory retinal detachment caused by leakage of fluid from the level of the retinal pigment epithelium.



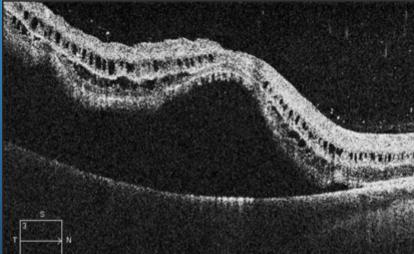
Retinal Detachment with OCT

• Macula On RD with VA 20/20. Emergency surgery within 24 hours.



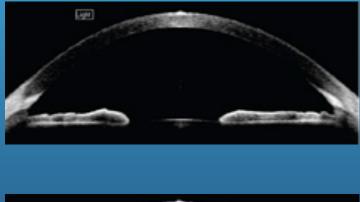
Retinal Detachment with OCT

If the macula has detached it is referred to as a "macoff" retinal detachment and prognosis for recovery of central acuity is worse, and thus urgent treatment is less critical. Nonetheless, studies show visual recovery is best if the procedure is performed within 7-10 days.



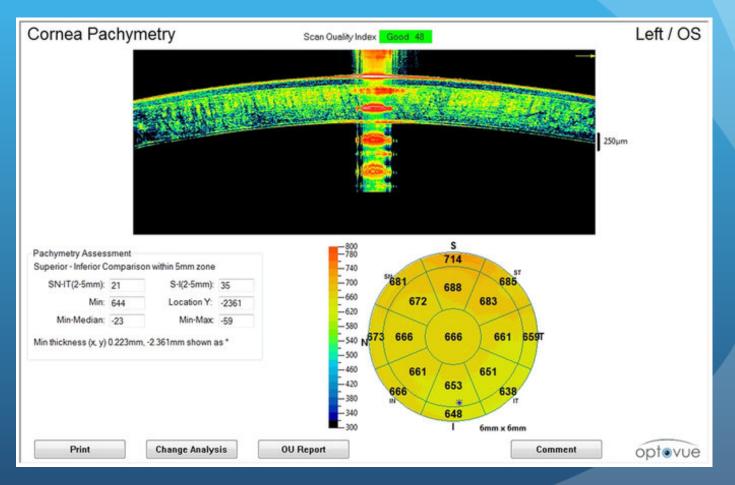
Anterior seg angles with OCT

 Gonioscopy: If the posterior trabecular meshwork can't be seen for 180 degrees or more, that's considered to be an occludable angle and the patient should receive prophylactic laser.

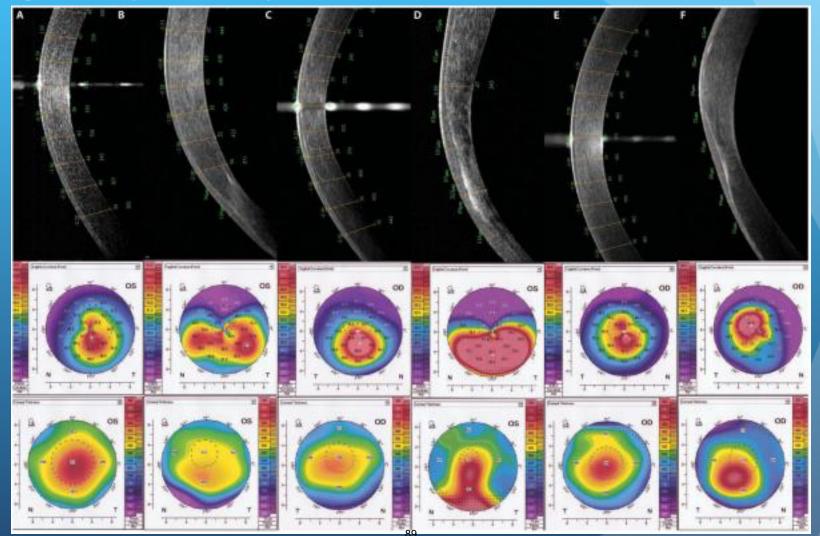




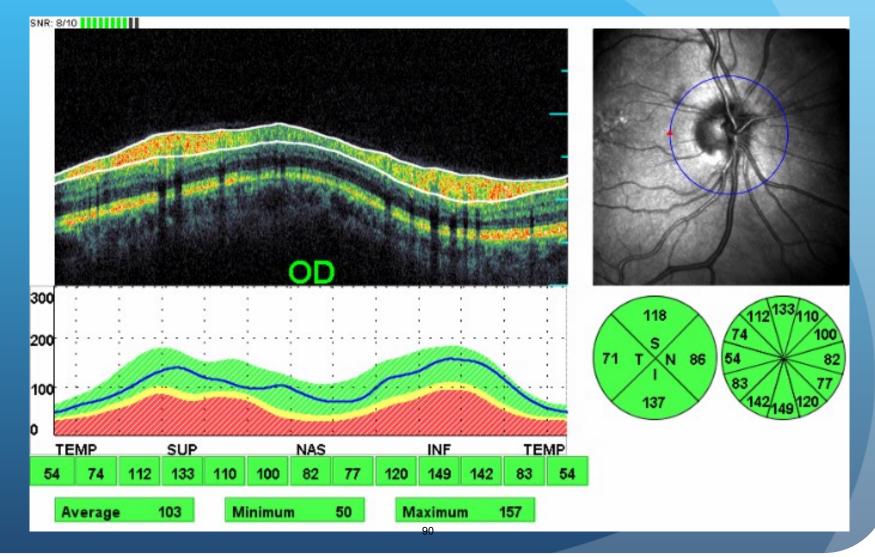
Pachymetry with OCT



Keratoconus using the OCT pachymetry



Normal RNFL interpretation with OCT

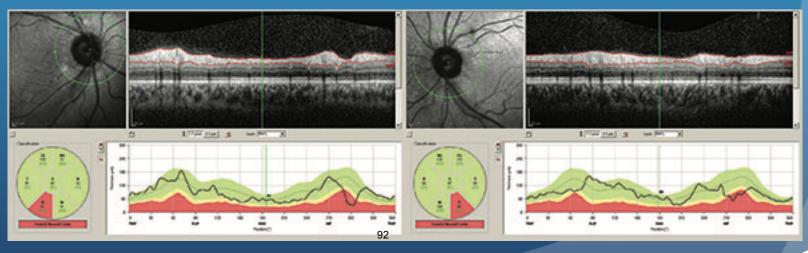


OHT interpretation/management with OCT

- Ocular hypertension is a condition in which the following criteria are met:
 - An intraocular pressure greater than 21 mm Hg in one or both eyes, as measured by applanation tonometry on 2 or more occasions
 - Absence of glaucomatous defects on visual-field testing
 - Normal appearance of the optic disc and nerve fiber layer
 - Anatomically normal, open angles on gonioscopy
 - Absence of ocular conditions contributing to the elevation of pressure, such as narrow angles, neovascular conditions, and uveitis

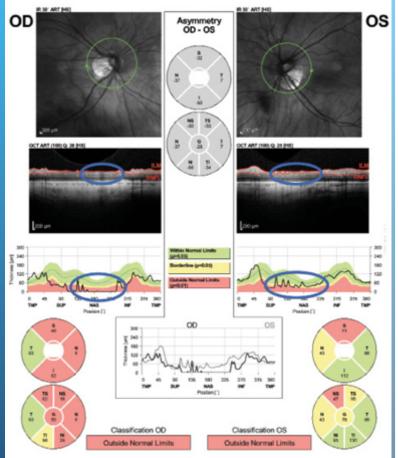
Early glaucoma interpretation/ management with OCT

- The advantage of OCT in early glaucoma is that it can detect damage that is not measurable with any other technology.
- Need to lose approximately 20 percent of her retinal nerve fiber layer before a visual field defect is likely to be present.
- Helpful to compare one eye to the other and look for asymmetry. Asymmetry, especially in the supero- or infero-temporal regions, provides a clue that it could be early glaucoma.

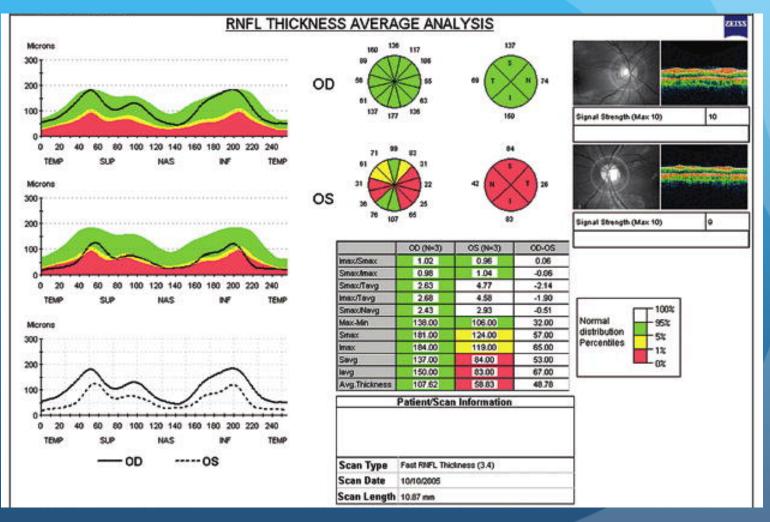


Late stage glaucoma interpretation/ management with OCT

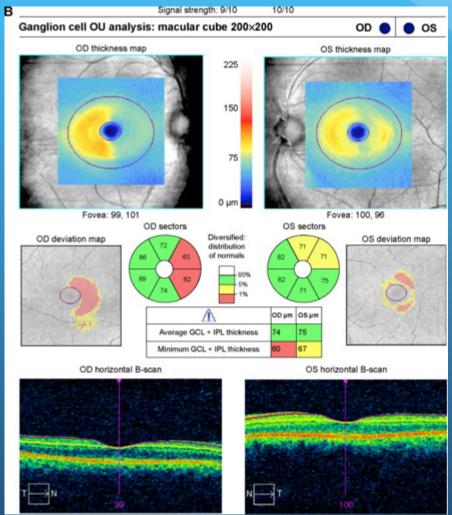
- In the advanced stages of glaucoma, the standard 24-2 visual fields may no longer be very sensitive to subtle progression.
- Imaging technologies such as SD-OCT also encounter problems with advanced optic nerve damage. On the SD-OCT measurements of the RNFL, the socalled floor effect becomes relevant in eyes with severe thinning. RNFL thinning levels off at approximately 40 to 50 µm, perhaps due to residual glial tissue, blood vessels, or other nonneural tissue.



MS interpretation/management with OCT

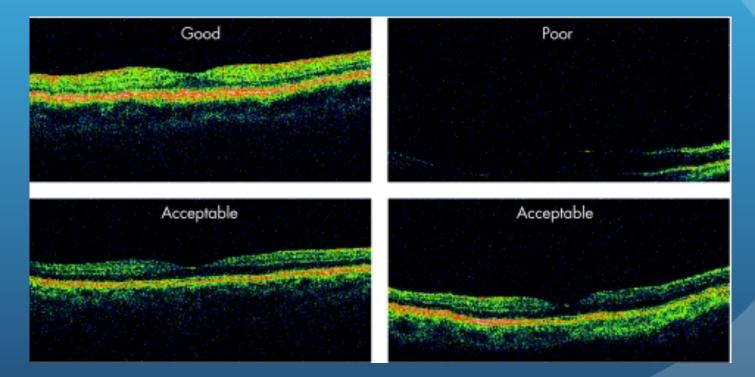


Brain trauma interpretation/ management with OCT



Detecting PSC with OCT

 Nuclear cataracts have less influence on OCT image quality relative posterior cataracts.



My gold standard to practice with OCT



Central Calífornía Optometríc Society

Tulare-Kings Optometric Society Cordially Invite You To Attend

The Inaugural Joint Society CE Program

"Common Subclinical Retinal Diseases. Diagnosis & Management Utilizing OCT" 2-Hour CE Approval Pending

> **Guest Speaker:** Dr. Kenneth E. Ekelund, OD Ekelund Vision Center Redding, California

Dinner Program Sponsored by



Thursday, September 8, 2016 6:00 PM – 8:30 PM Muskat Conference Room Holiday Inn-Selma Swancourt 2950 Pea Soup Anderson Blvd. Selma, CA 93662

We welcome all optometrists to attend. This CE Dinner Program is FREE for CCOS and TKCOS Members. For Non-Members, cost is \$50. This event has a cash/no-host bar.

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CCOS Members and Non-Members email your RSVP to <u>ccosociety@gmail.com</u> TKCOS Members email your RSVP to <u>TSuorsa@msn.com</u> Kenneth E Ekelund, O.D. Ekelund Vision Center 2190 Larkspur Lane, Suite 200 Redding, CA 96002 (530) 221-0726 Office (530) 221-1377 Fax

07/23/2015

Resume: Kenneth Errol Ekelund, O.D.

1975 Graduate of SCCO

- 1975-1987: Associate/Clinic Director with Carl Ebersole M.D.
- 1984-1997: Associate with Lee Nordan M.D., in refractive surgery (MKM, RK, ALK, PRK, Lasik)

1987-Present: Established solo private practice: General Optometry

1999-2008: Partnership/ clinic director, with Tom Tooma M.D., established lasik center in my private practice creating TLC Redding, Calif.

2013-2014: Optometry liaison with NVision/Tom Tooma M.D. NPB, Calif.

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Lecture Title: Common Subclinical Retinal Diseases. Diagnosis and Management Ulitizing OCT

Course Catagory: Ocular Disease

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Slide 1: OCT History / 4 Paradigm Shift.

Slide 2: Review of Retinal Anatomy with gray scale retinal layers

Slide 3: OCT Importance in daily practice:

Slide 4: Review of Retinal History

Slide 5: Choroid Complex review:

Slide 6: Mueller/glia cell review and function

Slide 7: 4 Categories of Retinal Disease with OCT

Slide 8: Diagram review of macula fovea faz foveola dimensions

Slide 9: Comparsion of fundus photo vs cross section OCT view of posterior pole

Slide 10: Outer layer retina view of cross section OCT is/os junction and its importance

Slide 11/12/13/14/15/: Review of PVD Partial PVD (APVD) development and stats

Slide 16: APVD creating multiple VRI maculopathies

Slide 17: OCT scans of VMA/VMT

Slide 18/19/20/: VMA/VMT types and new classification due to OCT

Slide 21: Schematic flow chart of VRI pathology.

Slide 22/22/23/24/25/: VMA/VMT broad vs focal adhesion with OCT

Slide 26: New Classification of macular holes due to OCT

Slide 27: Pre op OCT to create better post op outcomes and expection with OCT

Slide 28: OCT thickness maps of macula importance for managing pre and post op retinal pathology

Slide 29/30/31/: What makes a good candidate for VMT treatment with Jetrea

Slide 32: Measurement for macula hole to determine Jetrea vs PPV

Slide 33/34: Types of Lamellar Holes interpretation and/management with OCT

Slide 3536/37/: Pseudohole interpretation and management with OCT

Slide38/39/40: Post op macular hole interpretation with OCT

Slide 41/42/43/44/45/: ERM/Macular Pucker interpretation and management with macular thickness maps with OCT

Slide 46: Middle layer retina showing CME interpretation/management with OCT

Slide 47/48/49/50: Middle lalyer retina BVO interpretation/mangement with OCT

Slide: Review of the choroid complex anatomy.

Slide 49/50/51/52/53/: Early stage dry ARMD interpretation/management

Slide 54/55/56/57/: Late stage geographic degeneration OCT interpretation/management

Slide 58: Review histology of Wet ARMD (CNV)

Slide 59/60/61/: Wet ARMD interpretation/management with OCT

Slide 62: CSR Interpretation/management with OCT

Slide 63/64 Retinal Detachment with OCT

Slide:65: Anterior seg angles with OCT

Slide 66: Pachymetry with OCT

Slide 67: Keratoconus using the OCT pachymetry

Slide 68: Normal RNFL interpretation with OCT

Slide 69: IOHT interpretation/management with OCT

Slide 70: early glucoma interpretation/management with OCT

Slide 71: Late stage glaucoma interpretation/management with OCT

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Slide 72: MS interpretation/management with OCT Slide 73: Brain trauma interpretation/management with OCT Slide 74: Detecting PSC with OCT Slide75: My 4 th upgrade OCT and my gold standard to practice with OCT