# Electroretinogram



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## Definition

The electroretinogram (ERG) is a diagnostic test that measures the electrical activity of the retina in response to a light stimulus. The ERG arises from currents generated directly by retinal neurons in combination with contributions from retinal glia. Importantly, the ERG is an objective measure of retinal function that can be recorded noninvasively under physiologic conditions. ERGs are often recorded using a thin fiber electrode that is placed in contact with the cornea or an electrode that is embedded within a corneal contact lens. These electrodes permit the electrical activity generated by the retina to be recorded at the corneal surface. The ERG can be elicited by diffuse flashes or patterned stimuli.

The International Society for Clinical Electrophysiology of Vision (ISCEV) has introduced standards for the different forms of ERG recordings. The ERG has important clinical utility, in that it provides diagnostic information concerning a variety of inherited and acquired retinal disorders. Moreover, the ERG can be used to monitor disease progression and evaluate retinal toxicity due to various drugs or retained intraocular foreign bodies.

## History

The first known ERG was recorded from amphibian retina in 1865 by the Swedish physiologist Alarik Frithiof Holmgren. James Dewar of Scotland subsequently recorded an ERG in humans in 1877. In 1908, Einthoven and Jolly separated the ERG response into 3 components: a-wave, b-wave, and c-wave, which are further described below. Despite the early discovery of the ERG, widespread application did not begin until 1941, when American psychologist Lorin Riggs introduced a contact-lens electrode for ERG recording.

Many of the observations that serve as the basis for our understanding of the ERG were conducted by Ragnar Granit, for which he won the Nobel Prize for Physiology and Medicine in 1967. Granit's studies were primarily conducted on dark-adapted, rod-dominated cat retina. Using this model, he was able to demonstrate the physiology underlying different ERG sources by altering the level of anesthesia and observing the loss of different ERG components. Modern pharmacologic manipulations in various animal models have confirmed Granit's findings and have extended our understanding of the cellular sources of the ERG.

## Preparing the Patient

The ISCEV 2015 provide these guidelines for conducting a full-field ERG:

- Avoid fundus photography, fundus autofluorescence, fluorescein angiography, and other intense illumination before ERG recording. If this is unavoidable, allow at least 30 minutes of recovery time in ordinary room illumination
- Maximally dilate the pupils (note pupil size before testing). There is no need to correct refractive error
- Before dark-adapted protocols: 20 minutes of dark adaptation
- Before light-adapted protocols: 10 minutes of light-= adaptation.
- If corneal contact-lens electrodes are inserted after dark adaptation, this should be performed under dim red light. Allow 5 additional minutes of dark adaptation after insertion of contact-lens electrodes
- Present low-strength flashes before stronger flashes to avoid partial light adaptation from strong flashes

- Request the patient to fixate steadily and not move his/her eyes. Ocular movements introduce large electrical artifacts, change electrode position, and may cause blockage of light by the eyelids/electrode
- Young children and infants can be placed in the supine position, lying on their parent's legs, with their head under the stimulator[1]

# Types of Recording Electrodes

- **Burian-Allen (BA)**: Annular ring of stainless steel surrounding a polymethylmethacrylate contact-lens core. BA electrodes incorporate a lid speculum, which helps to minimize eye blinks/closure. BA lenses are reusable and are available in sizes ranging from pediatric to adult
- **Dawson-Trick-Litzkow (DTL)**: Low-mass conductive silver/nylon thread. DTL electrodes are disposable and are typically more comfortable for the patient than other corneal electrodes
- **Jet**: Disposable plastic lens with a gold-plated peripheral circumference
- **Skin Electrode**: May be used as a replacement for corneal electrodes. An electrode is placed on the skin over the infraorbital ridge near the lower eyelid. ERG amplitudes tend to be small and noisy, but skin electrodes are better tolerated by children
- **Mylar Electrode**: Aluminized or gold-coated Mylar (not in common use)
- **Cotton-Wick**: BA electrode shell fitted with a cotton wick, which is useful for minimizing light-induced artifacts (not in common use)
- **Hawlina-Konec Electrode**: Teflon-insulated thin metal wire (silver, gold, or platinum) with 3 central windows, 3 mm in length, molded to fit into the lower conjunctival sac (not in common use)

# Placement of Electrodes

## Recording Electrodes (in contact with the cornea, bulbar conjunctiva, or skin below lower eyelid)

- Protect the corneal surface with non-irritating ionic conductive solution (artificial tears or contact-lens solutions containing sodium chloride and no more viscous than 0.5% methylcellulose) Improper installation of contact-lens electrodes can cause corneal abrasions
- Topical anesthesia is used for contact-lens electrodes but may not be necessary for DTL electrodes.

## Reference and Ground Electrodes

- Electrical activity from the corneal electrode is comparable to that of a reference electrode placed at a distant site (e.g., ear, forehead, temple)
- A differential amplifier is typically used to amplify the difference between 2 inputs (corneal electrode and reference electrode) and reject signals that are common to both inputs (relative to a ground electrode placed at a third site)
- Reference and ground electrodes are commonly made of a highly conductive material that is fixed to the patient with paste. Gold cup electrodes are common because they can be reused; disposable adhesive skin electrodes are also available.
- Some corneal electrodes contain a reference, which obviates the need for a reference to be placed elsewhere (e.g. BA bipolar electrodes and some skin electrodes)

# Full-Field ERG (ffERG)

The ffERG is a mass response of the retina that has contributions from several retinal sources. This is useful in detecting diseases that have widespread retinal dysfunction: e.g., rod/cone dystrophies, cancer-associated retinopathy, and toxic retinopathies. Importantly, the ffERG is not useful for detecting small retinal lesions.

The ffERG waveform components and their underlying sources depend on both the strength of the stimulus flash and the state of adaptation. That is, scotopic measurements that target rod-pathway function are made from the dark-adapted eye, whereas photopic measurements that target cone-pathway function are made from the light-adapted eye. A minimum set of responses that should be obtained was defined by the ISCEV in 1989 and updated in 2015. Examples of the minimum ISCEV-specified ffERG set of responses under dark- and light-adapted conditions are shown in **Figure 1.**

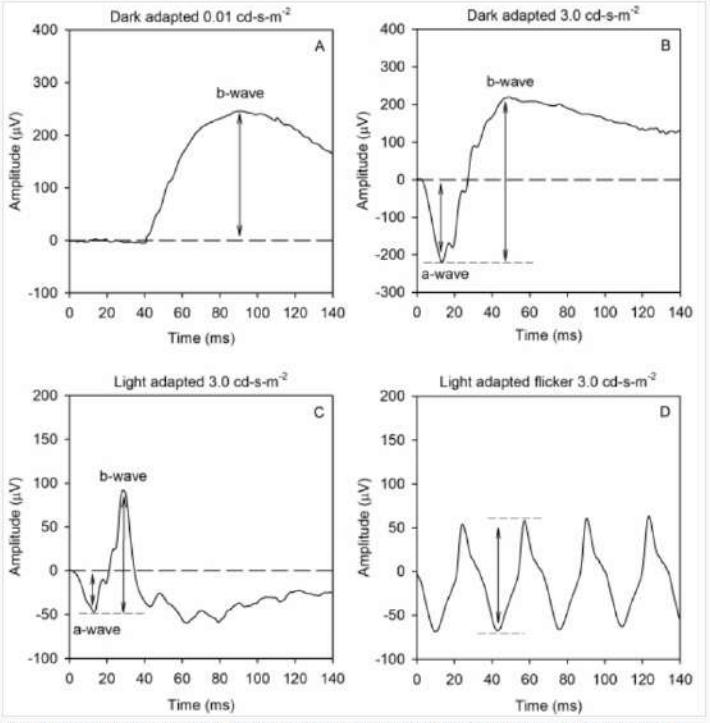



**Figure 1.** Examples of the minimum ISCEV-specified ffERG set of responses under dark- and light-adapted conditions (Courtesy of J. Jason McAnany, PhD.)

Panel A shows the ffERG recorded under dark-adapted conditions in response to a weak, diffuse, full-field flash of light. This stimulus elicits a slow cornea-positive potential, termed the **b-wave**, that is primarily generated by ON-type bipolar cells. The response is quantified by measuring the amplitude of the b-wave from the pre-stimulus baseline voltage (0 μV) to the peak of the response. Timing of the response is also measured; the implicit time of the b-wave is defined as the time between the flash and the peak of the response.

Panel B shows the ffERG recorded under dark-adapted conditions in response to a stronger flash of light. This stimulus elicits a rapid cornea-negative potential, termed the **a-wave**, and a subsequent positive b-wave. The amplitude of the a-wave is typically measured from the pre-stimulus baseline (0 μV) to the trough of the a-wave. The implicit time of the a-wave is measured from the time of the flash to the trough of the a-wave. The amplitude of the b-wave is measured from the trough of the a-wave to the peak of the b-wave, and the implicit time of the b-wave is measured from the time of the flash to the peak of the b-wave. This response is often referred to as the "mixed rod-cone response," as there are contributions from both rods and cones to the a-wave

from the time of the flash to the peak of the b-wave. This response is often referred to as the "mixed rod-cone response," as there are contributions from both rods and cones to the a-wave. However, the rod contribution exceeds the cone contribution, given the rod/cone distribution of the human retina. The b-wave is generated by ON- and OFF-type bipolar cells. Certain conditions, including complete congenital stationary night blindness, melanoma-associated retinopathy, and juvenile X-linked retinoschisis, produce a characteristic abnormality of this response that has been termed "electronegative." Specifically, the a-wave has a normal (or nearly normal) amplitude, whereas the b-wave is markedly attenuated. Thus, an electronegative response can have diagnostic value. Of note, a series of wavelets can be seen on the ascending portion of the b-wave. These wavelets are termed oscillatory potentials and are thought to be generated primarily by amacrine cells, but details of their source are presently debated. Oscillary potential that are reduced in amplitude and/or delayed in time often indicate disorders of the retinal blood supply.

Panel C shows the ffERG recorded under light-adapted conditions in response to a strong flash presented against a light background. The intent of the light background is to suppress the rod response, allowing for assessment of the cone pathway. This stimulus elicits a negative a-wave and a positive b-wave, much like that shown in panel B. The amplitude and implicit times of the a- and b-waves are quantified in the same manner as for the dark-adapted responses shown in panel B. Given that this response is recorded under photopic conditions, the a-wave is generated by cone photoreceptors, with additional contributions from OFF-type bipolar cells. The b-wave is generated by a combination of ON- and OFF-type bipolar cells.

Panel D shows the ffERG elicited by a 31-Hz flicker train. Rapid flicker is a useful stimulus for assessing cone-pathway function, because rod photoreceptors generally cannot follow rapid flicker. Each stimulus flash of the flicker train generates a response that has a peak and a trough. The amplitude of the flicker ERG is typically defined as the trough-to-peak amplitude, whereas the timing of the flicker response is typically defined as the time between a stimulus flash and the corresponding response peak.

## Other Waveform Components

**Photopic negative response (PhNR)**: The PhNR is a slow negative potential that follows the b-wave recorded under light adapted conditions (panel C, above). The PhNR has gained interest because it is primarily driven by retinal ganglion cells (RGCs); thus, it is one of the few ffERG components that provides insight into RGC function. The most effective measure of the PhNR and the optimal recording conditions are debated, but it is often measured from the pre-stimulus baseline to the trough of the response, or at a fixed time following the stimulus flash. In 2018, the ISCEV published guidelines for measuring and reporting the PhNR.

**c-wave**: The c-wave is a slow positive component that follows the b-wave and is generated from the retinal pigment epithelium and photoreceptors. Conventional ISCEV recordings do not provide assessment of the c-wave.

**d-wave**: The d-wave is a rapid positive potential that follows light offset and is generated by OFF-type bipolar cells. Conventional ISCEV recordings do not provide assessment of the d-wave.

## Reporting ffERG According to ISCEV Standards

Reports should include the following:

- At least 20 ms of baseline recording before the stimulus for single-flash ERGs
- Stimulus onset time
- At least 2 responses from each stimulus condition, for validation of consistency/assessment of variability
- Time-integrated luminance of the stimulus (cd-s-m-2) and background luminance (cd/m2)
- Reference values and range
- Any deviations from the standard ISCEV protocol (if applicable)
- Time of testing
- Pupil diameter
- Type and position of electrodes
- Any sedation/anesthesia
- Level of compliance

## Factors Affecting the ffERG

- Duration of stimulus
- Size of retinal area illuminated (amplitude can be reduced if the stimulus is not full field, because the patient would then be positioned too far from the stimulus source)
- Interval between stimuli
- Size of pupil
- Systemic circulation and drugs
- Development of retina
- Clarity of ocular media (note that mild cataract has minimal effects on the ffERG)
- Age
- Reduced ERG amplitude due to high myopia
- Anesthesia

# Other Types of ERG Measurement

## Focal ERG (fERG)

The focal ERG (fERG) is used primarily to measure the functional integrity of the central macula and is therefore useful in providing information in diseases limited to the macula. At present, this technique is not in common use, in part due to a lack of commercially available instruments. In addition, the multifocal ERG (discussed below) can be used to assess macular function. The electrode types and placement discussed for the ffERG can also be applied for fERG measurement.

A variety of approaches have been described in the literature for recording fERGs. Differing field sizes, varying from 3 degrees to 18 degrees, and stimulus temporal frequencies are used. However, each technique must address the challenge of limiting the amount of light scattered outside the focal test area. Focal ERG is useful for assessing macular function in conditions such as age-related macular degeneration; however, good fixation is required.

## Multifocal ERG (mfERG)

The multifocal ERG (mfERG) assesses many local ERG responses, typically 61 or 103 within the central 30 degrees. This provides important spatial information that is lacking in the ffERG, allowing dysfunction within the macula that might be missed by ffERG to be assessed. Multifocal ERG responses are recorded under light-adapted conditions from the cone pathway. It is important to note that mfERG is not a replacement for the ffERG: if pan-retinal damage or rod pathway dysfunction is suspected, then the ffERG should also be performed. The mfERG is becoming more commonly used for both research and clinical purposes, and the ISCEV provided the first standards for mfERG in 2007 (later updated in 2011).

Clarity of the ocular media and proper refraction are important for mfERG measurement. Electrodes and their placement can be the same as those described for the ffERG. A scaled hexagonal pattern, like that shown in **Figure 2**, is commonly used to elicit the mfERG. Each of the hexagons in the stimulus has a 50% chance of being illuminated at a given time. Although random in appearance, the same on/off sequence is used for each hexagon (an "m-sequence"). This permits a response to be recovered for each stimulus hexagon. The resulting mfERG waveforms (**Figure 2**) are similar in shape to those of the light-adapted ffERG: there is an initial negative deflection (termed N1), followed by a positive deflection (termed P1), and a second negative deflection (termed N2). Research indicates that N1 has generators similar to those of the a-wave of the light-adapted ffERG, whereas the P1 and N2 have generators that are similar to the light-adapted b-wave and OPs. However, the manner in which the mfERG is elicited and processed differs considerably from the ffERG; therefore, the mfERG response is not necessarily a miniature ffERG.

This approach produces a wealth of information and there are several ways in which the information can be condensed for display. Example mfERG responses from a normal eye are shown in **Figure**

This approach produces a wealth of information and there are several ways in which the information can be condensed for display. Example mfERG responses from a normal eye are shown in **Figure 2**. The same mfERG data are displayed in 3 different ways. The array of traces in the top row shows the mfERG response obtained from each hexagon. The middle panel of traces shows "ring averages," or average mfERG traces within rings of different eccentricity. For example, the red trace is the mfERG response obtained from the fovea, whereas the orange trace is the average of the ring of hexagons immediately surrounding the fovea. The other traces represent averages of rings of increasing eccentricity, as shown in the schematic to the right. Often, the ratio of amplitudes within rings is compared (i.e., the "ring ratios"). The lower image is a 3-dimensional mfERG amplitude plot. This topography plot shows the largest amplitude at the fovea, with a generally uniform decline in amplitude moving towards more eccentric locations. Another useful approach to visualizing the data is to plot the standard deviation of the amplitude (or implicit time) relative to visually normal controls within each hexagon. Thus, there are a number of ways in which the responses can be summarized for display; the optimal visualization is guided by the question that is being pursued.

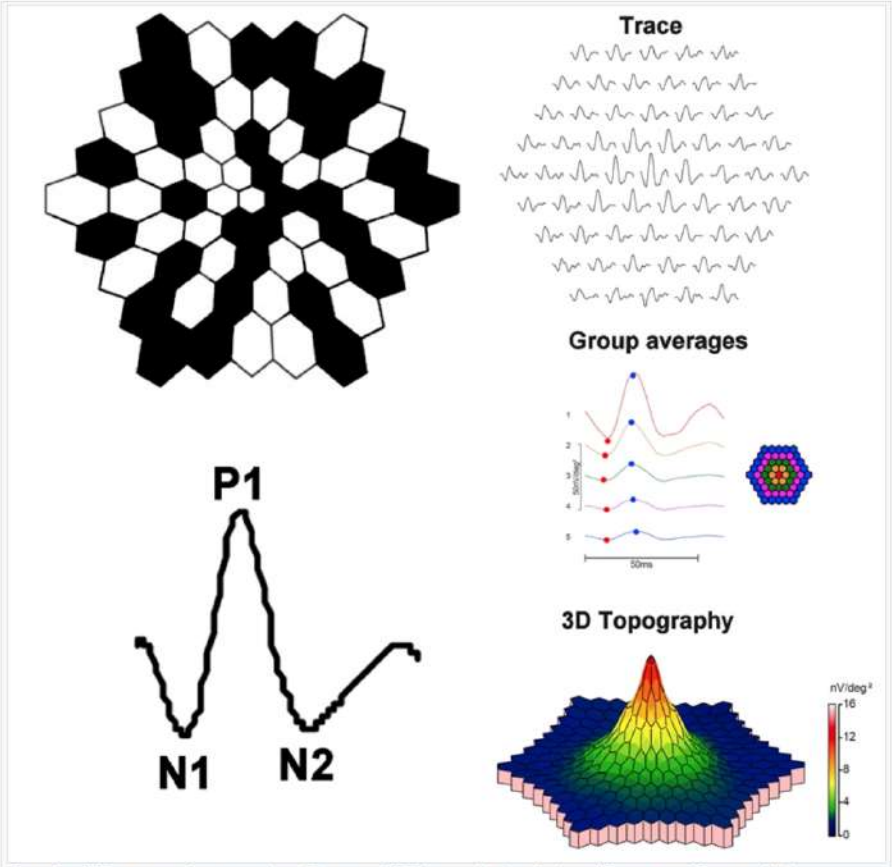



**Figure 2.** mfERG responses from a normal eye. The same mfERG data are displayed in three different ways. (Courtesy of J. Jason McAnany, PhD.)

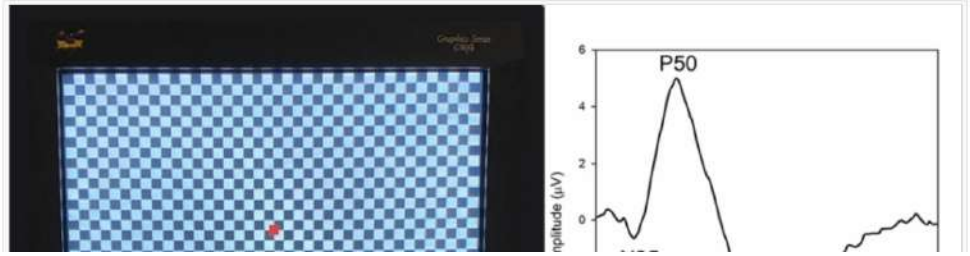
Given that mfERGs are useful for detecting localized abnormalities within the macula, a common application has been in assessing retinal dysfunction in hydroxychloroquine toxicity. The mfERG abnormality observed in these patients is often a decrease in the second ring amplitude, relative to the central ring. The mfERG has also been recorded in conditions such as retinitis pigmentosa, branch retinal artery occlusion, and Stargardt disease.

## Pattern ERG (pERG)

The pattern ERG (pERG) uses contrast reversing pattern stimuli (sinewave gratings or checkerboards) to assess macular RGC activity. Electrodes and their placement may be the same as those for the ffERG; however, contact-lens electrodes are often avoided to maintain optimal optical quality of the stimulus. Clarity of the ocular media and proper refraction are important for pERG measurement. The pERG is typically recorded with natural pupils. ISCEV has provided a standard for recording the pERG that was most recently updated in 2012. An example of a common pERG stimulus is shown below (**Figure 3, left**). Over time, the dark checks become light, and the light checks become dark (typically at a rate of 4 reversals per second). It is important that there is no net change in luminance during the dark-to-light transition of the checks (i.e., the average luminance of the screen must be constant over time), or a luminance artifact will be introduced into the response.

Given that the pERG responses have relatively small amplitude, many repetitions are obtained in clinical practice. The trace below (**Figure 3, right**) shows the pERG from a visually normal individual (average of 150 responses). The pERG waveform consists of a small negative deflection near 35 ms (the N35 component), a positive deflection near 50 ms (the P50 component), and a negative deflection near 95 ms (the N95 component). The amplitude and implicit time of each of these components can be measured. Of note, this waveform is characteristic of the "transient pERG" obtained with a stimulus that reverses 4 times per second, so that the response is essentially complete before the next contrast reversal begins. For higher reversal rates (e.g. ,16 reversals per second) a "steady-state" pERG is produced, which has different characteristics.

The N95 component is markedly reduced or eliminated in experimental glaucoma or by blocking action potentials using tetrodotoxin. Thus, the N95 component is likely generated by action potentials from RGCs. The source of the P50 is debated, but there is some evidence suggesting that it is generated by RGCs with additional contributions from more distal sites. The P50 and N95 components are dependent on macular cone function, as the photoreceptors provide input into the RGCs. Macular cone dysfunction can reduce the amplitude of the P50 and delay the response. Selective reduction of the N95 amplitude, with preservation of the P50 component, suggests RGC dysfunction. The pERG can be useful for assessing RGC function in conditions such as glaucoma and ischemic optic neuropathy. The pERG has also been shown to be abnormal in diabetic retinopathy and idiopathic intracranial hypertension.



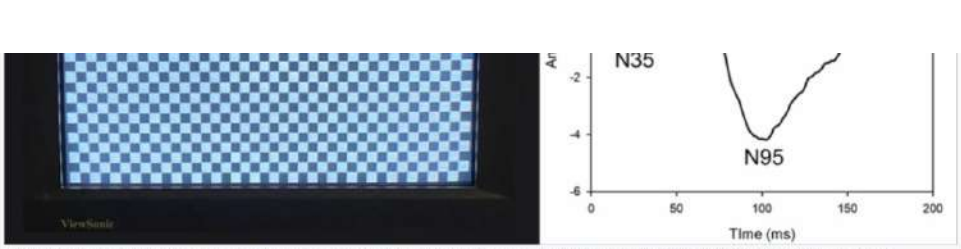



Figure 3. The pattern ERG (pERG) contrast reversing pattern stimuli and trace from a visually normal individual (average of 150 responses). (Courtesy of J. Jason McAnany, PhD.)

## ERG Abnormalities in Various Disease States

| Disease entity | Full-field ERG findings | Multifocal ERG findings |
|---|---|---|
| Abusive head trauma | Reduction of the b-wave; preservation of the a-wave[2] | Reduction of the b-wave; preservation of the a-wave[2] |
| Anesthesia | Amplitude of scotopic responses; implicit time prolongation of all components[1] | Abnormal |
| Achromatopsia (rod monochromacy) | Scotopic responses are normal/nearly normal; photopic responses are undetectable | Abnormal |
| Batten disease | Abnormal scotopic responses; strong flash response can be electronegative; photopic responses are abnormal | Abnormal |
| Best vitelliform macular dystrophy | Normal ffERG (abnormal electro-oculogram) | Possible mfERG abnormalities that localize to lesion location |
| Birdshot chorioretinopathy | Variable depending on disease state; photopic flicker response is commonly delayed; responses may be super-normal in early stages and reduced/delayed in late stages | Can be reduced/delayed; few reports are available in the literature |
| Cancer-associated retinopathy | Often severely abnormal or undetectable; photopic responses often more abnormal than scotopic | Often significantly abnormal |
| Central retinal artery and vein occlusions | Often significantly abnormal; reduced scotopic b-wave amplitude; OP abnormalities | Variable |
| Chloroquine/hydroxychloroquine | Scotopic and photopic responses are variable in mild cases; more likely to be abnormal in severe | Parafoveal abnormality in early stages with later fovea/central involvement |
| Choroideremia | Often severely abnormal; scotopic responses often worse than photopic responses | Typically abnormal, particularly with late macular involvement |
| Cone dystrophy | Abnormal photopic responses with normal/nearly normal scotopic responses | Often shows early and severe abnormalities |
| Congenital red-green color deficiency | Normal | Normal |
| Cone-rod dystrophy | Cone and rod abnormalities; photopic responses are more affected than scotopic responses | Often shows early and severe abnormalities |
| Congenital stationary night blindness (complete; Schubert-Bornschein type) | Dark-adapted weak flash response is absent; strong flash response is electronegative; photopic responses are usually abnormal | Abnormal |
| Congenital stationary night blindness (incomplete; Schubert-Bornschein type) | Dark-adapted weak flash response is abnormal; strong flash response is electronegative; photopic responses are substantially abnormal | Abnormal |
| Congenital stationary night blindness (Riggs type) | Scotopic responses are absent; photopic responses are typically normal | Normal |
| Diabetic retinopathy | Variable depending on disease stage; oscillatory potentials can be abnormal in early stages; flicker responses can be reduced and delayed; PhNR can be reduced | Patchy abnormalities; location of timing delays may correlate with present or future microaneurysms |
| Enhanced S-cone syndrome | Undetectable/significantly abnormal scotopic responses; significantly abnormal photopic responses | Abnormal |
| Fundus albipunctatus | Abnormal scotopic responses; variable photopic responses; scotopic responses improve after prolonged dark adaption | Variable |
| Leber congenital amaurosis | Severely abnormal or undetectable scotopic and photopic responses; abnormalities often present in infancy | Abnormal |
| Melanoma-associated retinopathy | Dark adapted weak flash response is absent; strong flash response is electronegative; photopic responses are variable, but can be abnormal | Abnormal |
| Multiple evanescent white dot syndrome | Scotopic/photopic abnormalities that resolve following the acute phase | Variable; abnormalities can be observed that resolve following the acute phase |
| North Carolina macular dystrophy | Typically normal | Abnormal in central macula |
| Oguchi disease | Dark adapted weak flash response is absent; strong flash response is electronegative; photopic responses are normal; scotopic responses improve after prolonged dark adaption | Normal |
| Pattern dystrophy | Normal | Normal |
| Quinine toxicity | Abnormal scotopic responses; strong flash response can be electronegative; abnormal photopic responses | Abnormal |
| Retinitis pigmentosa | Severely abnormal or undetectable scotopic responses; photopic responses are variable, but usually abnormal; scotopic/photopic are undetectable in late-stage | Variable |
| Retinopathy of prematurity (ROP) | Lower sensitivity of rods, which corresponds with severity of ROP[3] | May be abnormal |
| Siderosis | Usually abnormal; scotopic responses are usually more affected than photopic responses; initially can produce supernormal responses followed by amplitude loss over time | Can be abnormal |
| Stargardt disease | Variable: can find normal scotopic and photopic responses; normal scotopic and abnormal photopic responses; and abnormal scotopic and photopic responses | Abnormal |
| Vitamin A deficiency | Abnormal scotopic responses; normal photopic responses (but can vary) | Normal |
| X-linked retinoschisis | Dark adapted weak flash response is significantly reduced/absent; strong flash | Abnormal |

| | | |
|---|---|---|
| | response is often electronegative; photopic responses are abnormal | |

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# How to read OCTs: 8 fundamental diseases

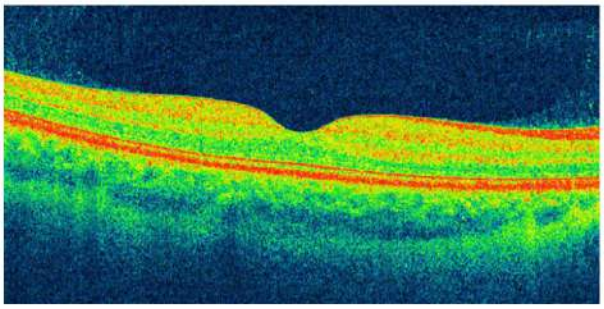
Want to accelerate your learning curve? Train your brain with our OCT Mastery Trainer.

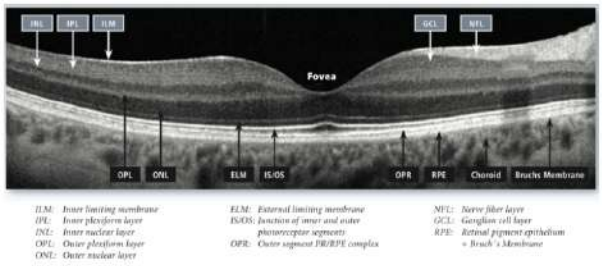
Listed here are 80-90% of the OCTs that you are going to be seeing. Most OCT is used for imaging the retina, so that's what we'll focus on. It's revolutionized the field of retina, helped us revise the pathophysiology of multiple diseases based on OCT evidence, and is a standard for the treatment of multiple macular diseases.

## About OCT

Optical coherence tomography is a non-contact, high-resolution, in vivo imaging modality. It produces cross-sectional tomographic images just like ultrasound. Decreased OCT image quality can be attributable to cataracts which block light, patient motion artifact, or any other media opacity.

## View of the normal retina with OCT





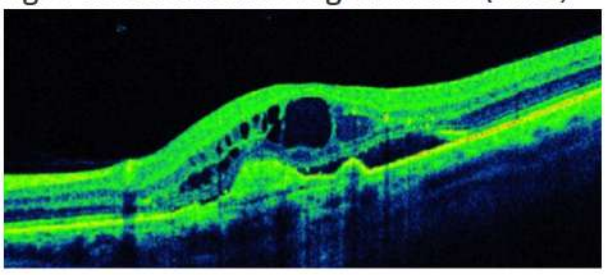

ILM: Inner limiting membrane
IPL: Inner plexiform layer
INL: Inner nuclear layer
OPL: Outer plexiform layer
ONL: Outer nuclear layer
ELM: External limiting membrane
IS/OS: Junction of inner and outer photoreceptor segments
OPR: Outer segment PR/RPE complex
NFL: Nerve fiber layer
GCL: Ganglion cell layer
RPE: Retinal pigment epithelium + Bruch's Membrane

The macular OCT can be used to evaluate the premacular vitreous, macula, and choroid. We'll look at the OCT through a number of common diseases.

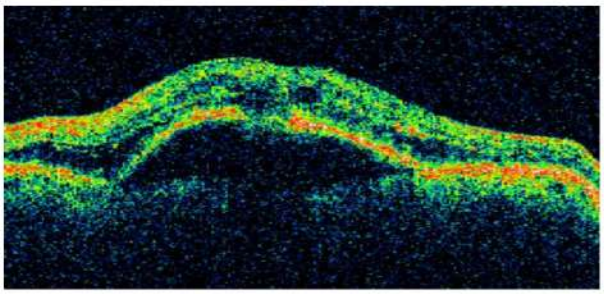
Below, we've highlighted a few diseases with their common OCT findings:

## View of the retinal pathologies with OCT

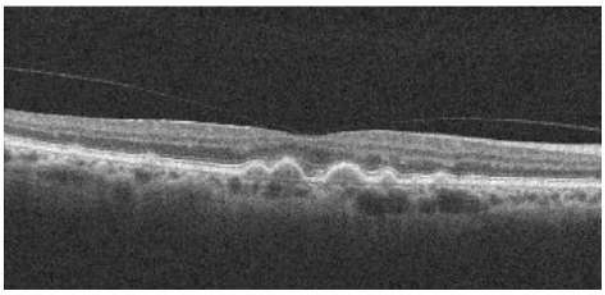
### Age-related macular degeneration (AMD)



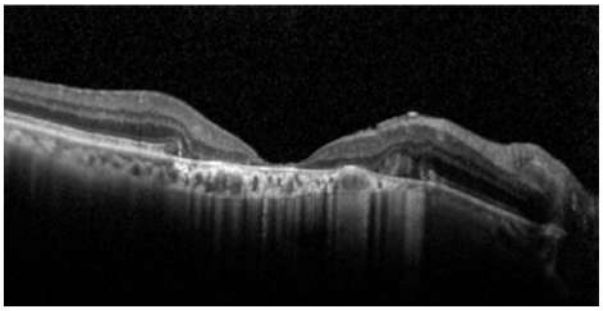
Wet AMD leads to intraretinal fluid (IRF) and subretinal fluid (SRF) accumulation. The choroidal neovascular membrane (CNV) can be visualized.



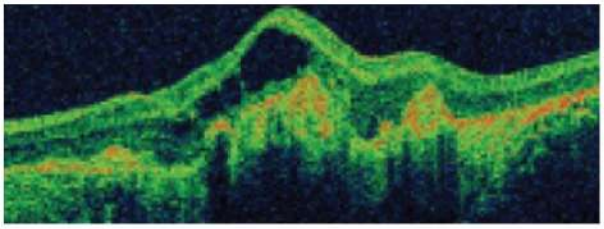
This is an OCT of a pigment epithelial detachment (PED) in wet AMD with some adjacent subretinal fluid (SRF) and an overlying area of focal intraretinal fluid (IRF).



Drusen: Lumps of deposits under the RPE.

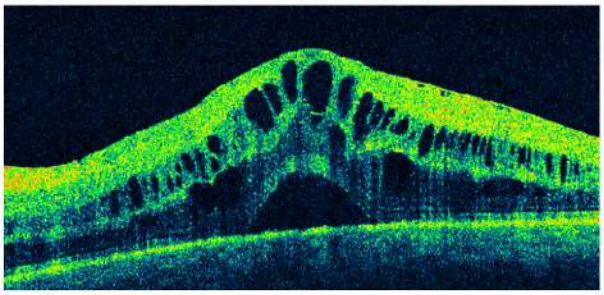


Geographic atrophy: Atrophy of the outer retinal layers with OCT signal penetrating deeper into the choroid.



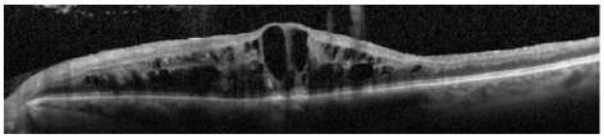
Another example of wet AMD: Eyes show fibrovascular pigment epithelial detachments (PEDs), neovascular membranes and subretinal fibrosis along with fluid.

## Diabetic macular edema (DME)

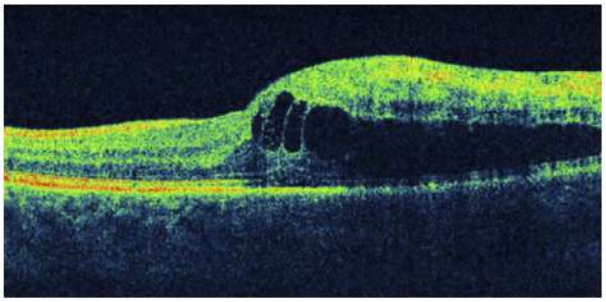


This is  classic DME with cystoid intraretinal fluid pockets in the outer plexiform layer (OPL). Subretinal fluid (SRF), which is present in severe DME, is also seen here.

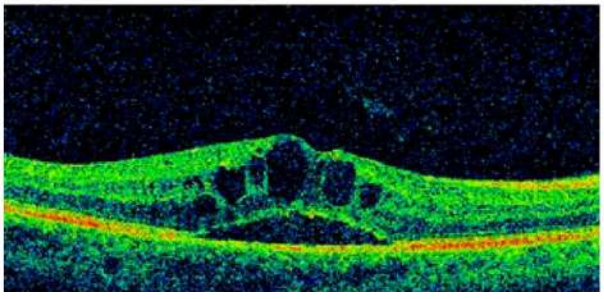
## Retinal vein occlusions (RVOs)



This central retinal vein occlusion (CRVO) causes severe cystoid macular edema (CME). There isn't a clear differentiation between CME from CRVOs and CME from diabetic retinopathy on OCT, but the fundus appearance is obvious.

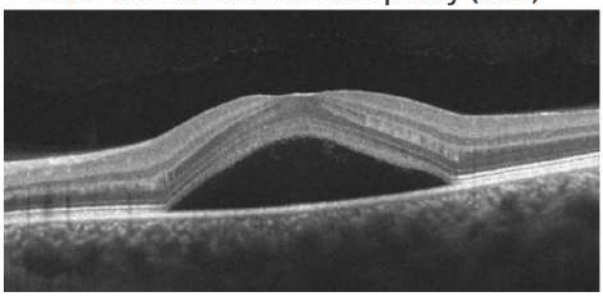


In comparison to CRVO, this branch retinal vein occlusion (BRVO) shows retinal edema on the temporal side of the macula, which is a more common finding in BRVO. You know it's the temporal side because the nasal side of the OCT has a thicker retinal nerve fiber layer (RNFL).

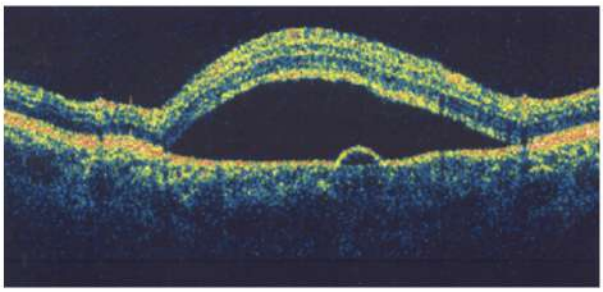


Here is a BRVO with central macular edema. Chronic RVOs lead to inner retinal atrophy, which is also characteristic of the disease.

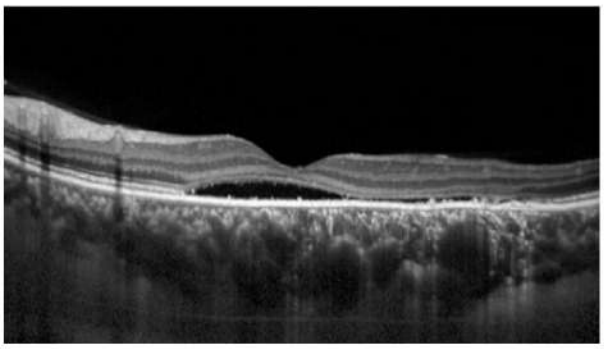
## Central serous chorioretinopathy (CSR)



CSR has a central SRF collection, no intraretinal fluid (IRF), and a thickened choroid.

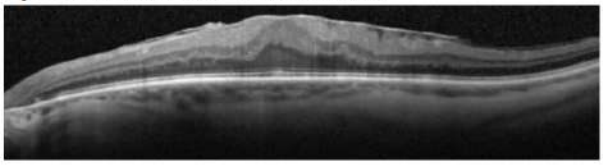


With CSR, there can often be a component of pigment epithelial detachment (PED) inside the area of serous detachment. These PEDs can be quite large.

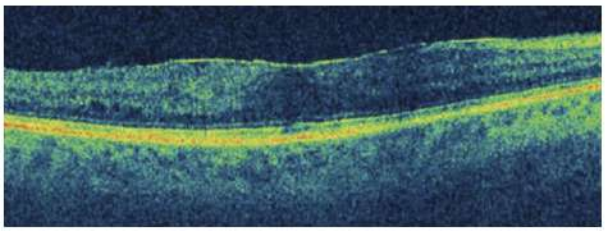


This example of CSR displays a very thick choroid.

## Epiretinal membrane (ERM)

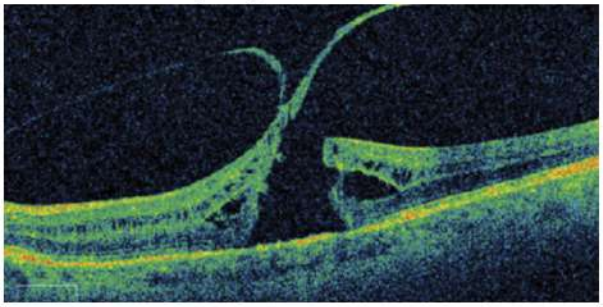


A dense epiretinal membrane (ERM) can be seen here leading to inner retinal wrinkling and distortion of the foveal contour. A severe ERM can also be associated with cystoid macular edema.

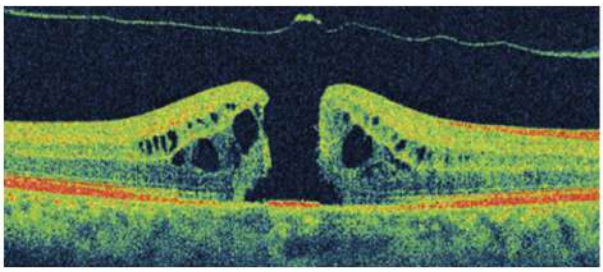


A mild-moderate ERM.

## Macular Hole (MH)

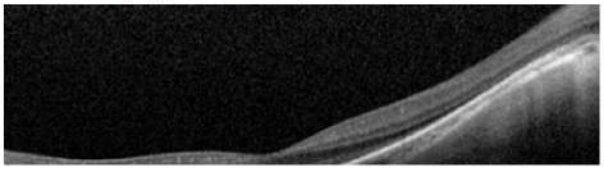


Full-thickness macular holes (FTMH) are very easy to diagnose with OCT. They are always a foveal, full-thickness defect that can have associated cystoid macular edema. Here, there is traction from the posterior hyaloid membrane that opens the hole in a “can opener” effect.



Here, the posterior hyaloid has separated, leaving a central operculum and a full thickness defect.

## Retinitis pigmentosa (RP)

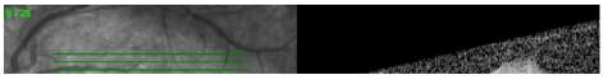


RP is a rod-cone dystrophy. The photoreceptor layer is completely lost except for a central island.

Along with it comes thinning of the outer nuclear layer (ONL), which is where the cell bodies of the photoreceptor cells reside.

Vision loss from RP can also come from CME (cystoid macular edema) which is something to keep in mind for your RP patients. You can treat this with topical dorzolamide.

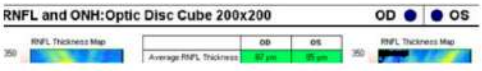
### Retinal detachment



A retinal detachment is usually diagnosed clinically and with exam, but shallow macular detachments are sometimes hard to appreciate early on. If any doubt, a retinal OCT can demonstrate a detachment easily.

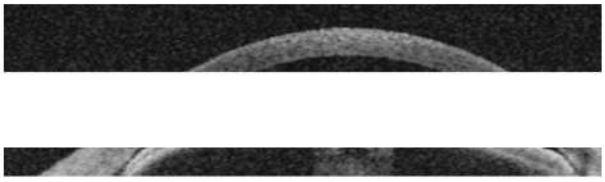
## Optic nerve OCT

Optic nerve and nerve fiber layer OCT helps in the management of glaucoma. The OCT machines provide automated, serial analysis of the nerve fiber layer thickness, cup-to-disc ratio, and other measurements. They can compare the patient's optic nerve and nerve fiber measurements against age-matched normal patients to show areas of loss. These analyses have become an important adjunct to visual field testing in the treatment of glaucoma. It can also be used to track optic nerve edema.



## Anterior segment OCT (AS-OCT)

Anterior segment OCT is most commonly used to evaluate the iridocorneal angle, such as for patients with narrow angles. It can also be used for corneal biometry to measure the thickness and steepness of the cornea.



AS-OCT of an eye with narrow angles.

## Conclusion

1. OCT is a non-contact, cross-sectional imaging modality providing high-resolution images of the macula.
2. Summary of the diseases in this article:
    1. Wet age-related macular degeneration (AMD)
        1. Intraretinal fluid (IRF) and subretinal fluid (SRF) accumulation
        2. Pigment epithelial detachments (PEDs)
    2. Diabetic macular edema (DME)
        1. Cystoid macular edema (CME), intraretinal fluid pockets in the outer plexiform layer
        2. SRF (subretinal fluid) if severe
    3. Central retinal vein occlusions (CRVO)
        1. Severe CME
    4. Branched retinal vein occlusions (BRVO)
        1. Retinal edema on temporal side of macula
        2. Chronic RVOs lead to inner retinal atrophy, which is characteristic of the disease
    5. Central serous chorioretinopathy (CSR)
        1. Central SRF (subretinal fluid) collection, no IRF (intraretinal fluid), and a thickened choroid
        2. Can have PED (pigment epithelial detachment) inside the area of SRF (subretinal

fluid) accumulation

6. Epiretinal membrane (ERM)
    1. Inner wrinkling and distortion of foveal contour
    2. Cystoid macular edema if severe
7. Macular hole
    1. Foveal, full-thickness defect
    2. Can have associated
8. Epiretinal membrane (ERM)
    1. Inner retinal wrinkling and distortion of foveal contour
    2. Cystoid macular edema if severe
9. Macular hole
    1. Foveal, full-thickness defect
    2. Can have associated CME (cystoid macular edema)
10. Retinitis Pigmentosa
    1. Loss of photoreceptor layer, with sparing of a central island
    2. Thinning of outer nuclear layer (ONL)
    3. CME can be present (cystoid macular edema)
11. Retinal detachment
    1. Usually diagnosed clinically and with exam, but OCT can be used to check shallow macular detachments

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### Share Your Comments & Feedback

You must be logged in to post a comment.

## 15 Responses...

*Vignesh Rengappa* says:
March 22, 2018 at 12:38 am
For a beginner to interpret it is important to know what schitic and cystic spaces look like ( just an example) … So it'll be helpful if they are arrowd down or numbered

LOG IN TO REPLY

*Christine Morrison* says:
October 4, 2019 at 9:06 am
This was very interesting. I work with a consultant ophthalmologist a half day a week in a medical retina clinic, and despite seeing these images regularly, this was still very helpful.

LOG IN TO REPLY

*영현 김* says:
May 15, 2020 at 1:05 am
thank you for your kindness

LOG IN TO REPLY

*ANGABEEN AHMAD* says:
September 21, 2021 at 9:05 am
its very helpful for beginners

LOG IN TO REPLY

*Tahir Shaukat* says:
October 21, 2021 at 3:27 am
overall article is good and knowledge able but at the end ERM and macular hole are repeated in summery of the article.

LOG IN TO REPLY

*Taha Elftisi* says:
October 27, 2021 at 3:01 am
Highly appreciated, very helpful and simplified

LOG IN TO REPLY

*AMATER TRAYLOR* says:
June 20, 2022 at 2:59 pm
i will return to this site for more practice, since i find this module most helpful

LOG IN TO REPLY

*Shsul Sh* says:
October 4, 2022 at 2:46 pm
So helpful.
But at the conclusion ERM and macular hole are repeated 2 times.

LOG IN TO REPLY

*MOHAMAD ALRUBY* says:
February 13, 2024 at 12:19 pm
very good account of the OCT

LOG IN TO REPLY

*mahesh* says:
June 4, 2024 at 9:23 am
Interesting but representation with arrow mark would be more helpful for learning like pointing/highlighting SRF , IRF , CNVM etc .

LOG IN TO REPLY

*joseph bundi* says:
March 18, 2025 at 8:17 am
quite helpful, thank you for the updates

LOG IN TO REPLY

*Ali Azizi* says:
July 21, 2025 at 9:01 am
Thank you✊

LOG IN TO REPLY

S

*Shifaa* says:
August 26, 2025 at 10:53 am
Thank you so Much

LOG IN TO REPLY

*M Wasim Sayed Issa* says:
September 14, 2025 at 2:15 am
I higly appreciate this simple explanation.

LOG IN TO REPLY

*Robert Deacon* says:
December 23, 2025 at 8:14 am

Very useful

LOG IN TO REPLY

# Electroretinogram (ERG) and Electrooculogram (EOG)

Change Language German



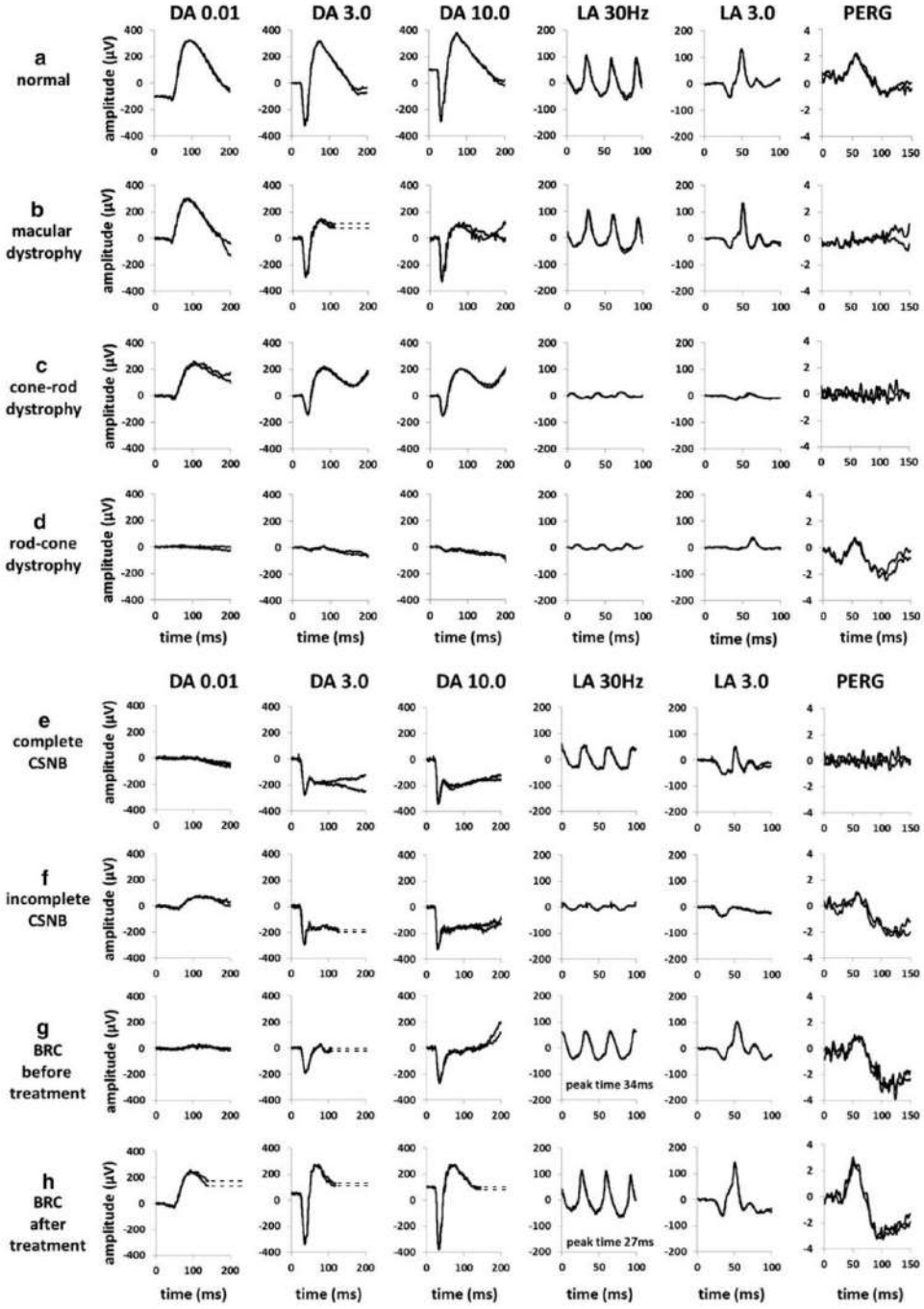
## Introduction

- 3 main types of electroretinograms (ERGs)
  - Full-field ERG (ffERG): Illuminates the entire retina uniformly
  - Pattern ERG (PERG): Utilizes a contrast stimulus, typically an alternating checkerboard pattern
  - Multifocal ERG (mfERG): Provides a topographic map of cone system function over approximately 50° of the retina
- Characteristics of Full-field ERG
  - DA = Dark Adaptation
  - LA = Light Adaptation
  - 0.01 / 3.0 / 10.0 = Flash intensities, indicating the strength of the light flash
  - a-wave: Response of photoreceptors (negative wave)
  - b-wave: Response of Müller cells and bipolar cells (positive wave)
  - c-wave: Response of the retinal pigment epithelium (RPE), 2-4 seconds delayed, (positive wave)
    - occurs only in dark adaptation
  - Implicit time: Time from light stimulus to the peak of the b-wave (in milliseconds)

## Overview ERG und PERG[1]

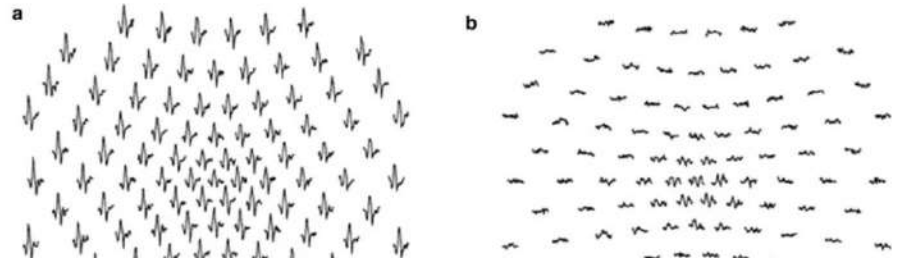
- DA 0.01: Rod-specific; b-wave, cannot differentiate between photoreceptors and inner retinal layers.
- DA 3.0: Mixed rod-cone response; includes both a- and b-waves.
- DA 10.0: a-Wave indicates photoreceptor function; differentiates between photoreceptor dysfunction and inner retinal dysfunction.
  - If DA 0.01 is reduced:
    - DA 10.0 a-wave reduced? -> photoreceptor dysfunction
    - DA 10.0 b-wave reduced? -> inner retinal dysfunction
- LA 30 Hz: "photopic flicker" tests cone function
- LA 3.0: "photopic single-flash" a-wave corresponding to cone photoreceptors and off-bipolar cells, b-wave with on/off-bipolar cells
- **PERG**: Pattern ERG: contrast stimulus, typically an alternating checkerboard pattern at consistent brightness
  - Must be focused on the macula
  - e.g. LHON
  - Main components
    - P50: Amplitude allows objective assessment of macular function

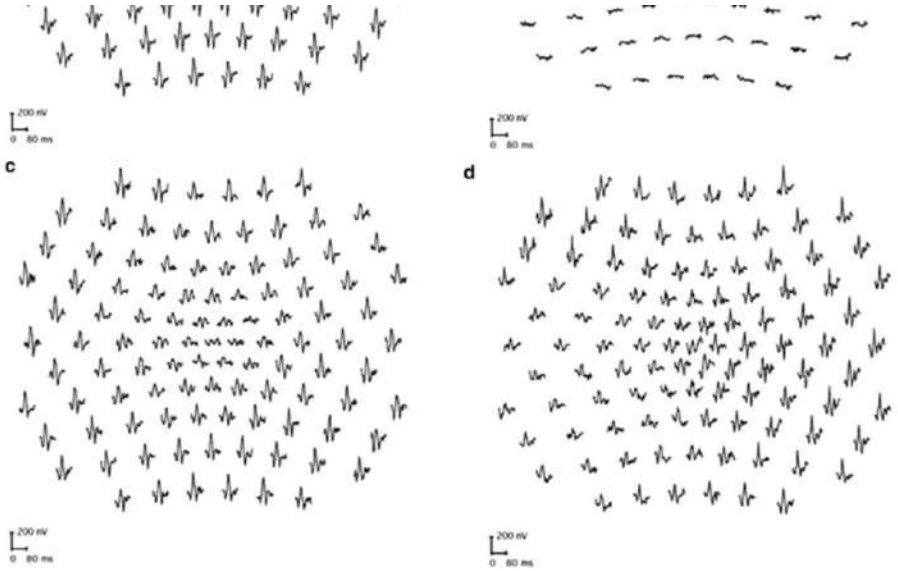
- N95: Measures central retinal ganglion cell function (RGCs)
  - Additional component: N35




Multifocal ERG[1]

- Topographic representation of the cone system over approximately 50°
- Good fixation is important
- e.g. for early detection of hydroxychloroquine retinopathy
- a) Normal finding; b) Retinitis pigmentosa; c) Macular dystrophy; d) Enlarged blind spot in eccentric nasal retinal dysfunction






Indication

- Diagnosis of generalized retinal degenerations
- In cases of suspected reduced visual acuity and presence of nystagmus at birth
- Measuring retinal function in cases of opaque media
- If functional visual loss is suspected

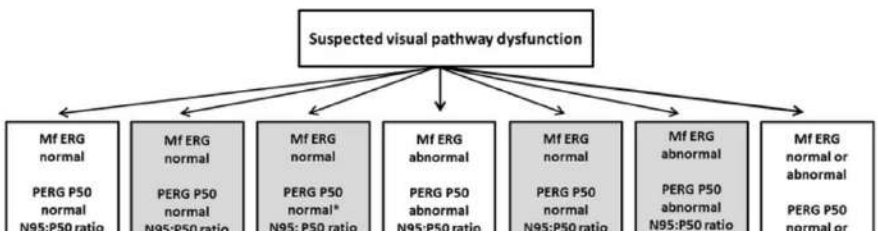
Examples

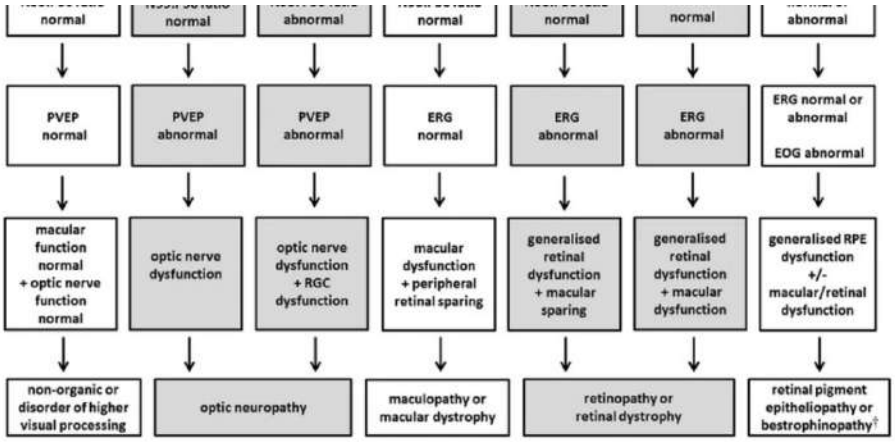
- Central Retinal Artery Occlusion (CRAO): Normal a-wave (as the photoreceptor layer is supplied by the choroid), missing b-wave
- Ischemic Central Retinal Vein Occlusion (CRVO): Reduced amplitude of b-wave, extended implicit time
- Retinitis Pigmentosa: Reduced amplitude (usually of the b-wave) and extended implicit time; in advanced stages -> no rod and cone response to bright light stimuli
- Multiple Evanescent White Dot Syndrome (MEWDS): Reduced a-wave
- Glaucoma, Congenital Rubella, Optic Atrophy/Neuropathy: Normal ERG (as ganglion cells are affected)

Electrooculogram (EOG)

- Measures corneoretinal potential
- Assesses the function of the RPE and the interaction of photoreceptors with RPE
- Arden Ratio: Maximum potential height in light divided by minimum potential height in darkness
  - Normal: > 1.85/ > 185%
  - Pathological: < 1.65/ < 165%
- The ERG is pathological in all cases where the EOG is abnormal **except**:
  - Normal ERG, pathological EOG:
    - Best's disease as a classic example
    - Pattern dystrophies, Chloroquine retinopathy
  - Pathological ERG, normal EOG:
    - X-linked retinoschisis, CSNB (congenital stationary night blindness)

Test Strategy Algorithm[1]






Sources


- EyeWiki Electroretinogram
- EyeWiki Electrooculogram
- The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease; Kalla Gervasio MD, Travis Peck MD et al; Lippincott Williams&Wilkins; 8th Edition (2021)
- Kanski's Clinical Ophthalmology: A Systematic Approach; John E Salmon MD; Elsevier; 9th Edition (2019)
- †Robson, A.G., Nilsson, J., Li, S. *et al.* ISCEV guide to visual electrodiagnostic procedures. *Doc Ophthalmol* **136,** 1–26 (2018). https://doi.org/10.1007/s10633-017-9621-y
- International Society for Clinical Electrophysiology of Vision – iscev.org
- American Academy of Ophthalmology; 2017-2018 Basic and Clinical Science Course (BCSC), Section 12 Retina and Vitreous

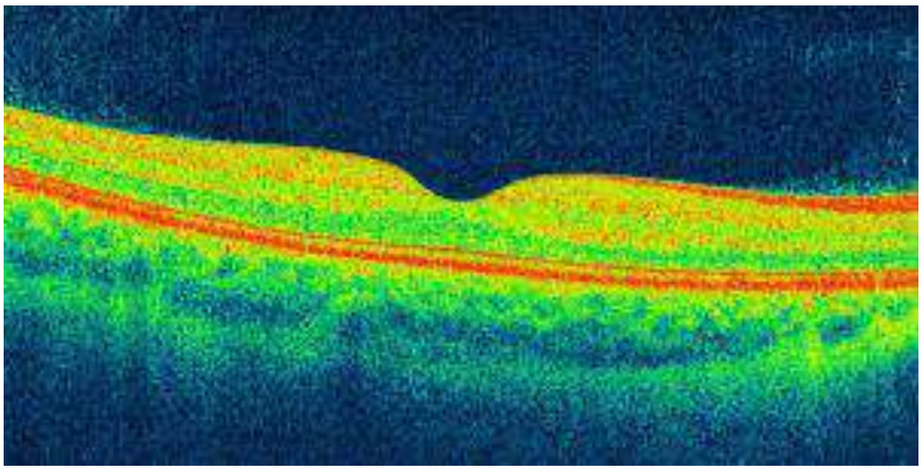
**How to read OCTs: 8 fundamental diseases**

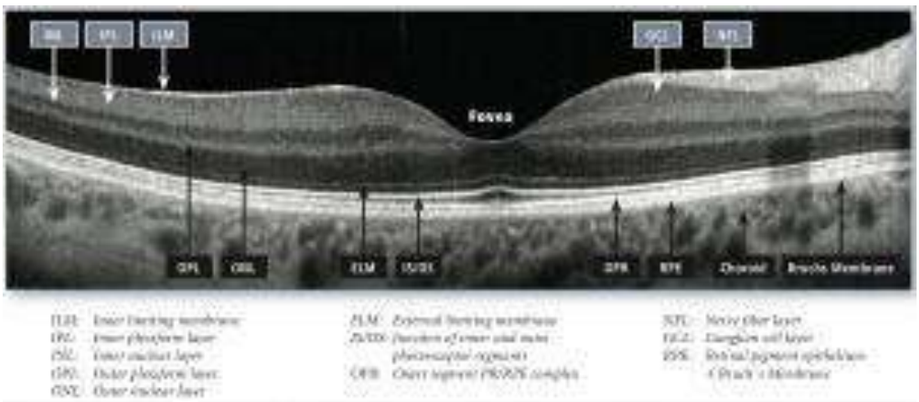
Listed here are 80-90% of the OCTs that you are going to be seeing. Most OCT is used for imaging the retina, so that's what we'll focus on. It's revolutionized the field of retina, helped us revise the pathophysiology of multiple diseases based on OCT evidence, and is a standard for the treatment of multiple macular diseases.

**About OCT**

Optical coherence tomography is a non-contact, high-resolution, in vivo imaging modality. It produces cross-sectional tomographic images just like ultrasound. Decreased OCT image quality can be attributable to cataracts which block light, patient motion artifact, or any other media opacity.

**View of the normal retina with OCT**



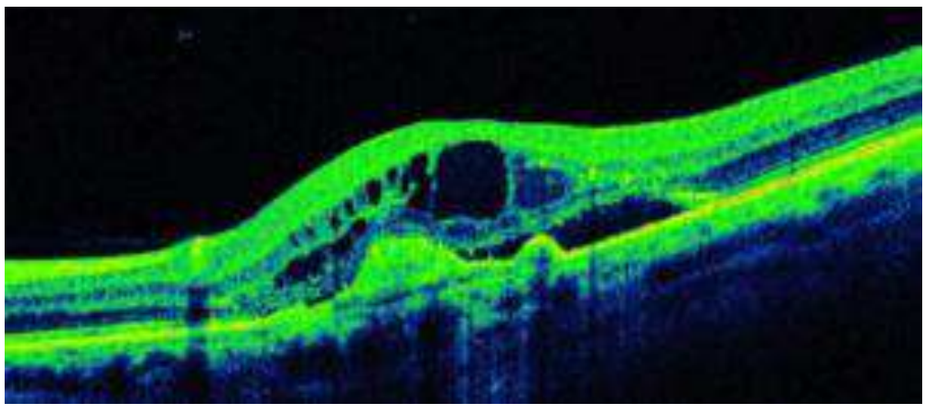



The macular OCT can be used to evaluate the premacular vitreous, macula, and choroid. We'll look at the OCT through a number of common diseases.

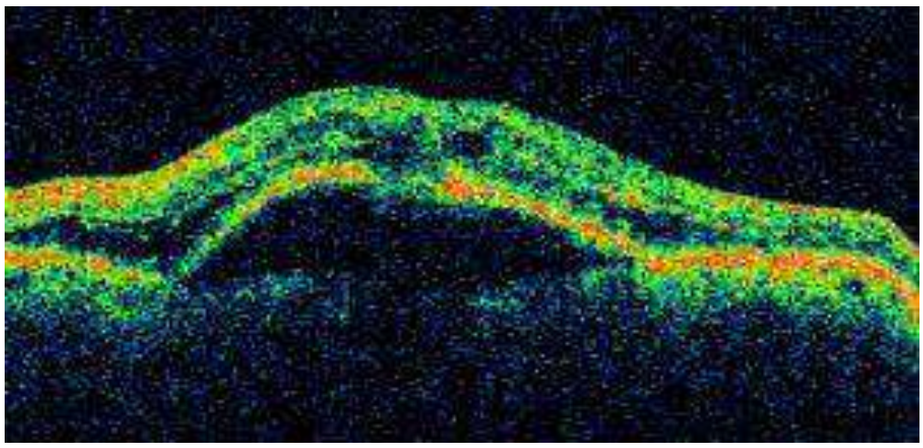
Below, we've highlighted a few diseases with their common OCT findings:

**View of the retinal pathologies with OCT**

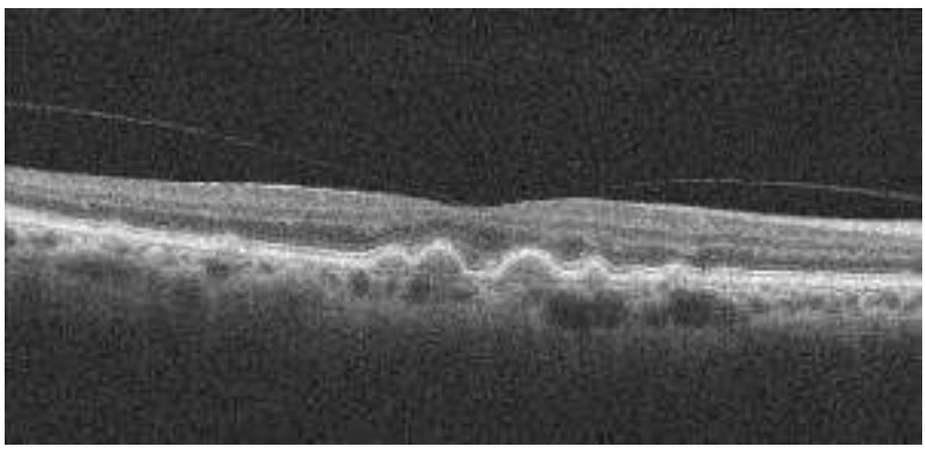
**Age-related macular degeneration (AMD)**



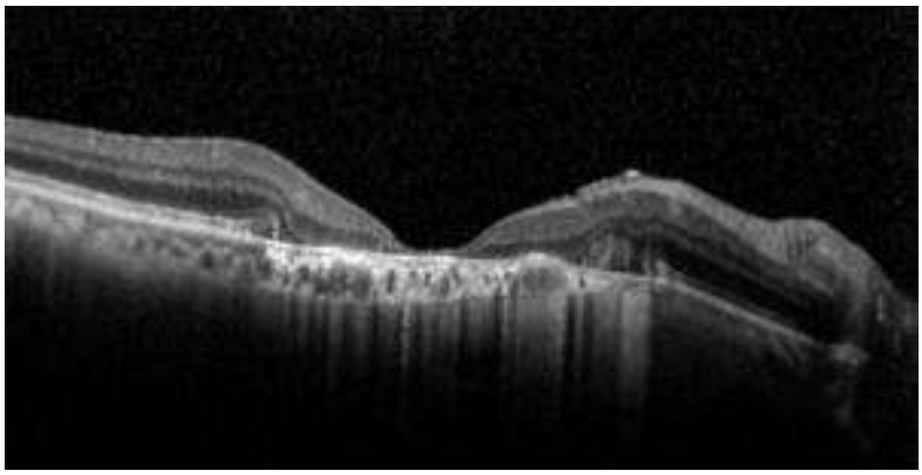
Wet AMD leads to intraretinal fluid (IRF) and subretinal fluid (SRF) accumulation. The choroidal neovascular membrane (CNV) can be visualized.



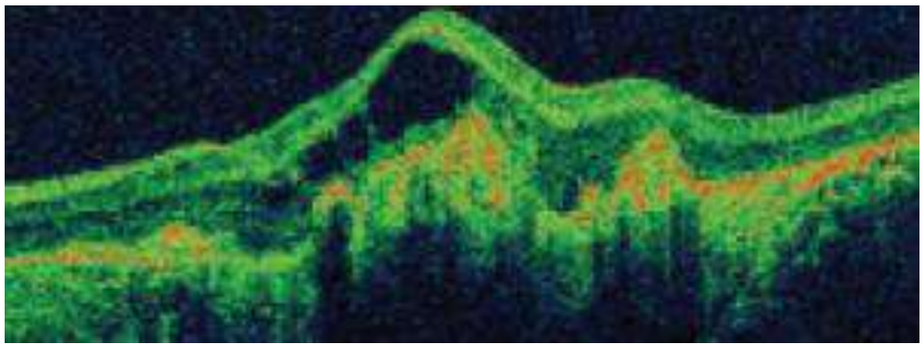
This is an OCT of a pigment epithelial detachment (PED) in wet AMD with some adjacent subretinal fluid (SRF) and an overlying area of focal intraretinal fluid (IRF).



Drusen: Lumps of deposits under the RPE.

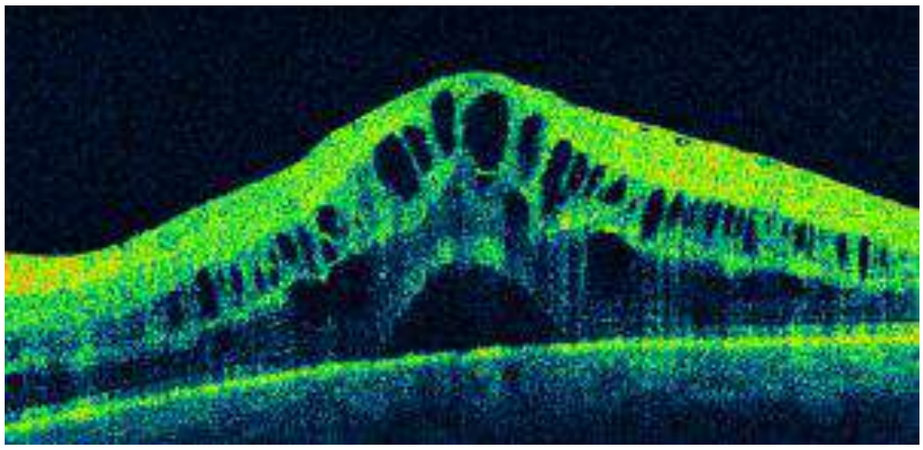


Geographic atrophy: Atrophy of the outer retinal layers with OCT signal penetrating deeper into the choroid.



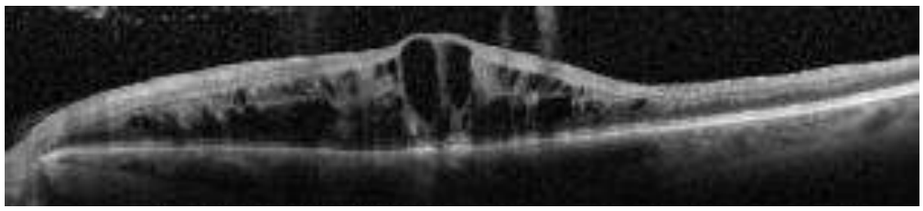
Another example of wet AMD: Eyes show fibrovascular pigment epithelial detachments (PEDs), neovascular membranes and subretinal fibrosis along with fluid.

**Diabetic macular edema (DME)**

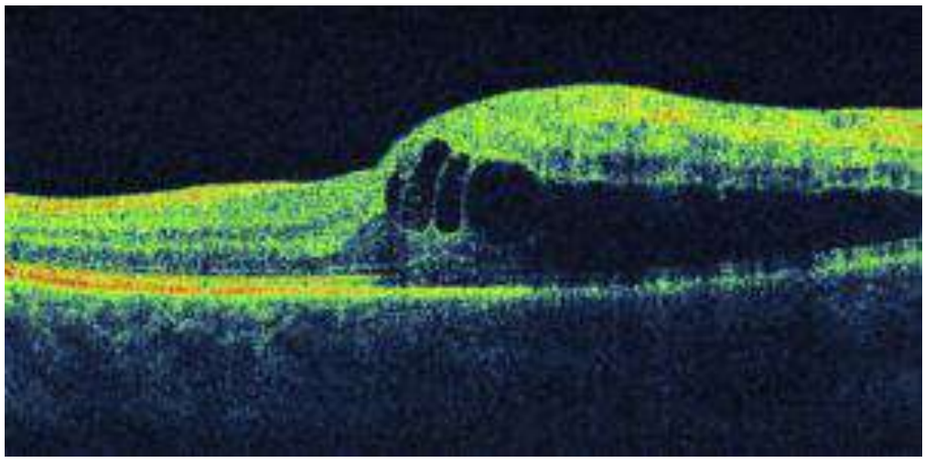


This is  classic DME with cystoid intraretinal fluid pockets in the outer plexiform layer (OPL). Subretinal fluid (SRF), which is present in severe DME, is also seen here.

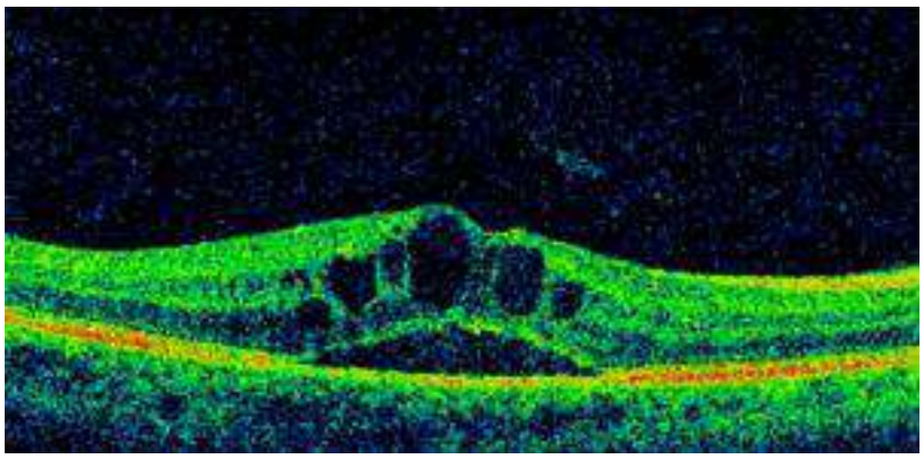
**Retinal vein occlusions (RVOs)**



This central retinal vein occlusion (CRVO) causes severe cystoid macular edema (CME). There isn't a clear differentiation between CME from CRVOs and CME from diabetic retinopathy on OCT, but the fundus appearance is obvious.

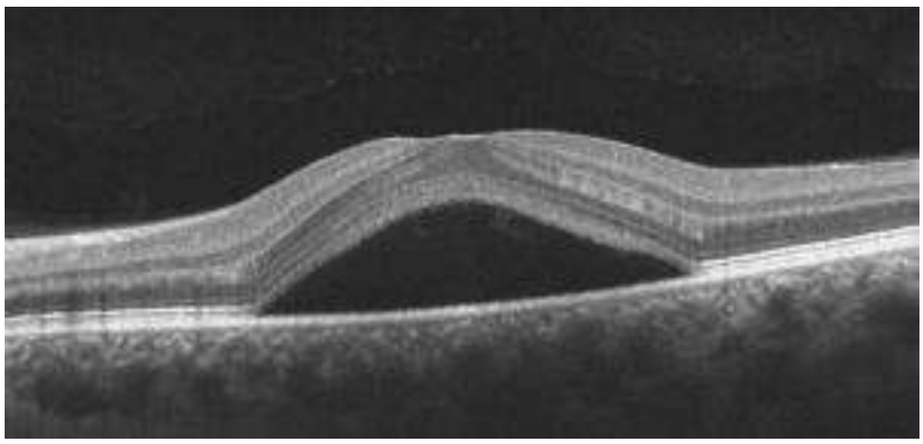


In comparison to CRVO, this branch retinal vein occlusion (BRVO) shows retinal edema on the temporal side of the macula, which is a more common finding in BRVO. You know it's the temporal side because the nasal side of the OCT has a thicker retinal nerve fiber layer (RNFL).

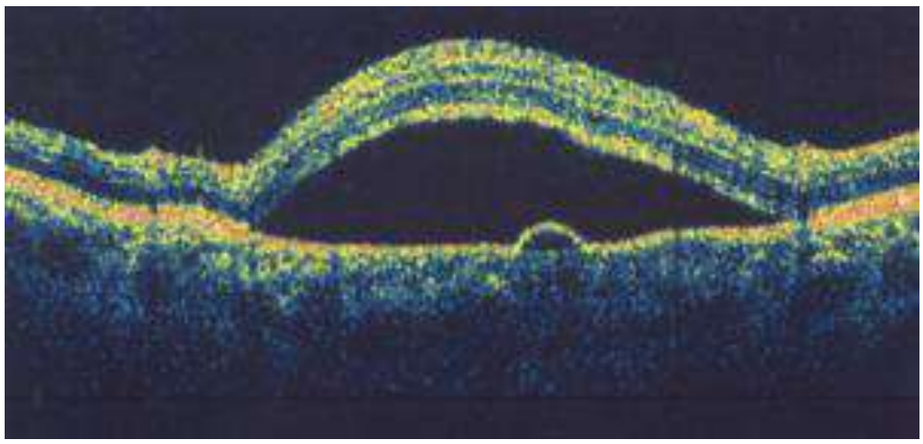


Here is a BRVO with central macular edema. Chronic RVOs lead to inner retinal atrophy, which is also characteristic of the disease.

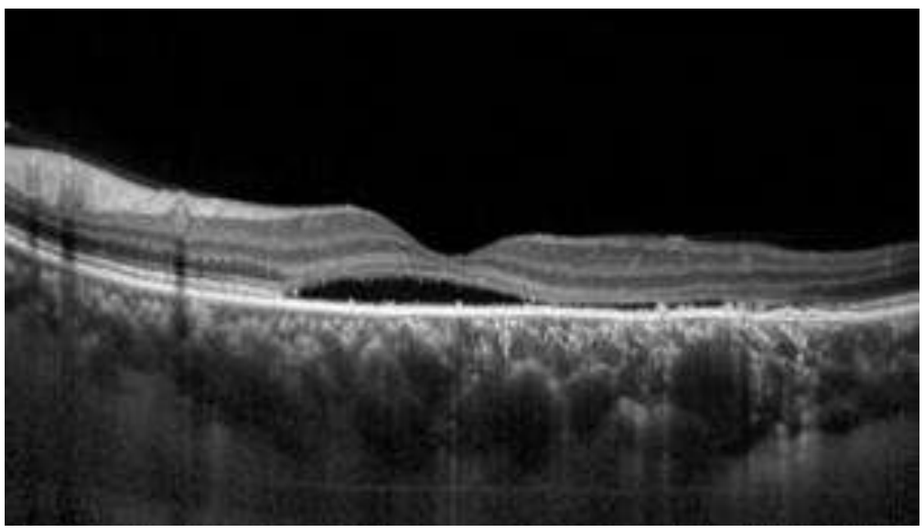
**Central serous chorioretinopathy (CSR)**



CSR has a central SRF collection, no intraretinal fluid (IRF), and a thickened choroid.

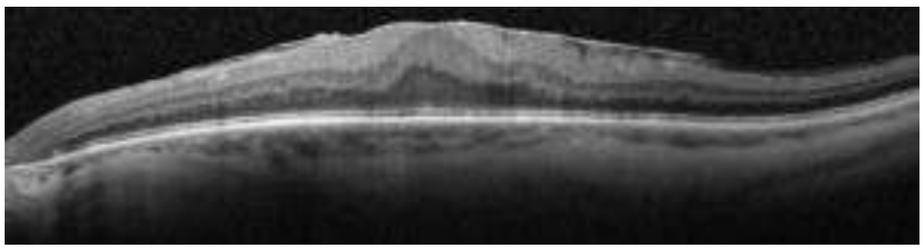


With CSR, there can often be a component of pigment epithelial detachment (PED) inside the area of serous detachment. These PEDs can be quite large.

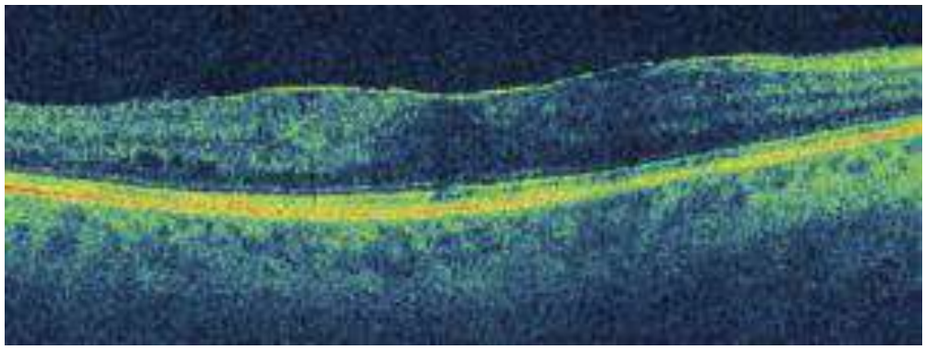


This example of CSR displays a very thick choroid.

**Epiretinal membrane (ERM)**

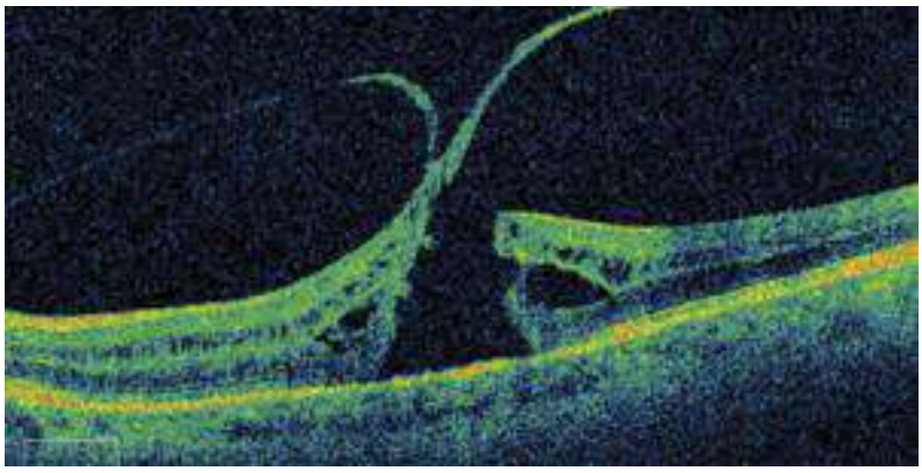


A dense epiretinal membrane (ERM) can be seen here leading to inner retinal wrinkling and distortion of the foveal contour. A severe ERM can also be associated with cystoid macular edema.

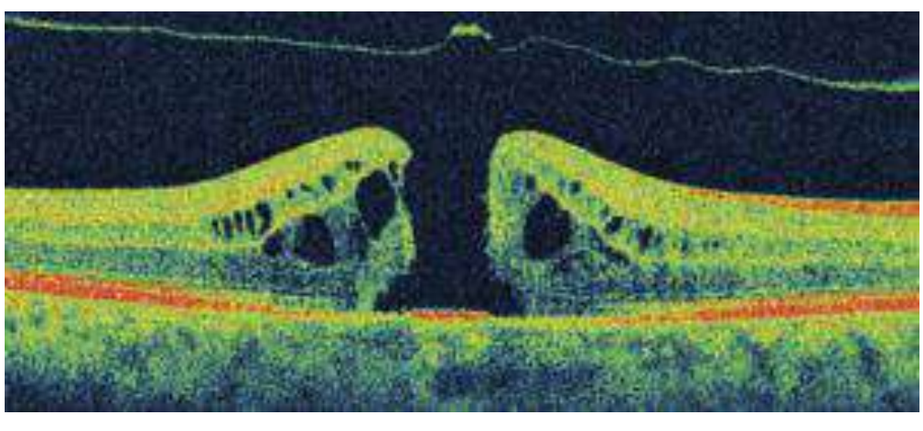


A mild-moderate ERM.

**Macular Hole (MH)**

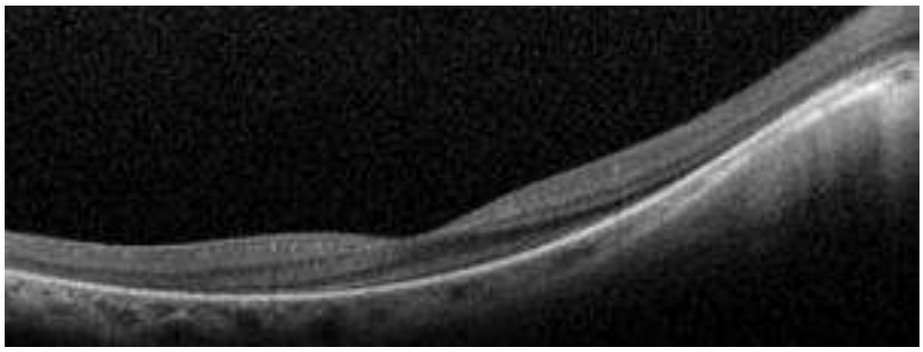


Full-thickness macular holes (FTMH) are very easy to diagnose with OCT. They are always a foveal, full-thickness defect that can have associated cystoid macular edema. Here, there is traction from the posterior hyaloid membrane that opens the hole in a “can opener” effect.

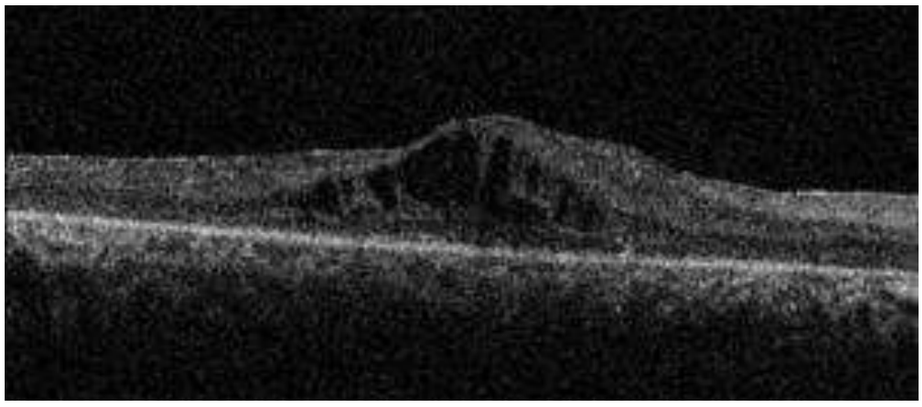


Here, the posterior hyaloid has separated, leaving a central operculum and a full thickness defect.

**Retinitis pigmentosa (RP)**

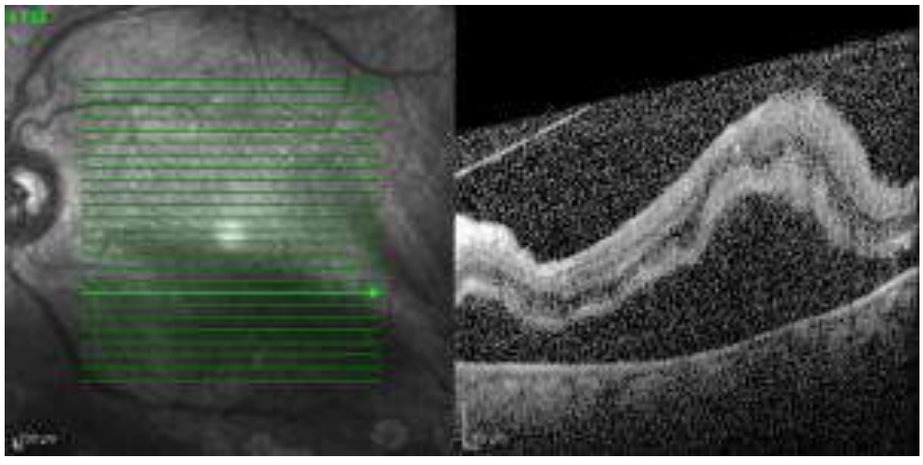


RP is a rod-cone dystrophy. The photoreceptor layer is completely lost except for a central island. Along with it comes thinning of the outer nuclear layer (ONL), which is where the cell bodies of the photoreceptor cells reside.

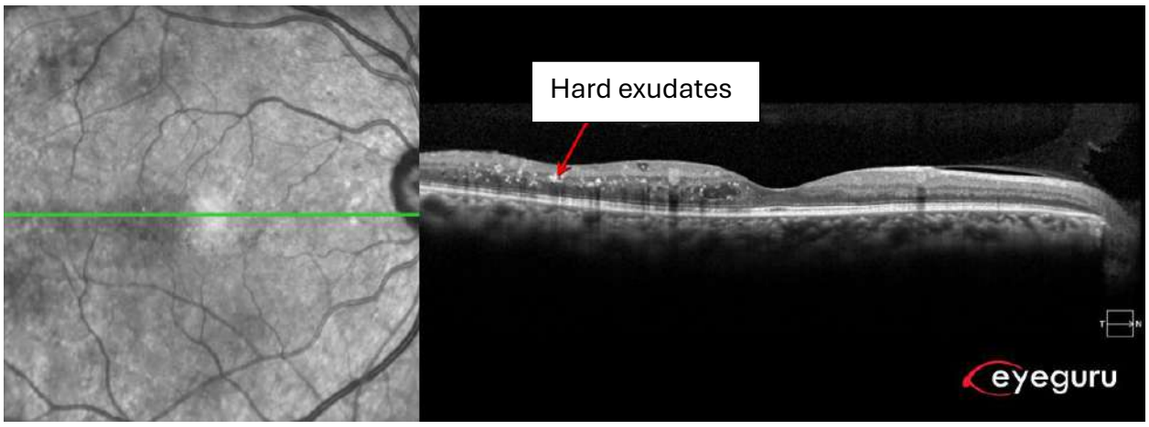


Vision loss from RP can also come from CME (cystoid macular edema) which is something to keep in mind for your RP patients. You can treat this with topical dorzolamide.

**Retinal detachment**

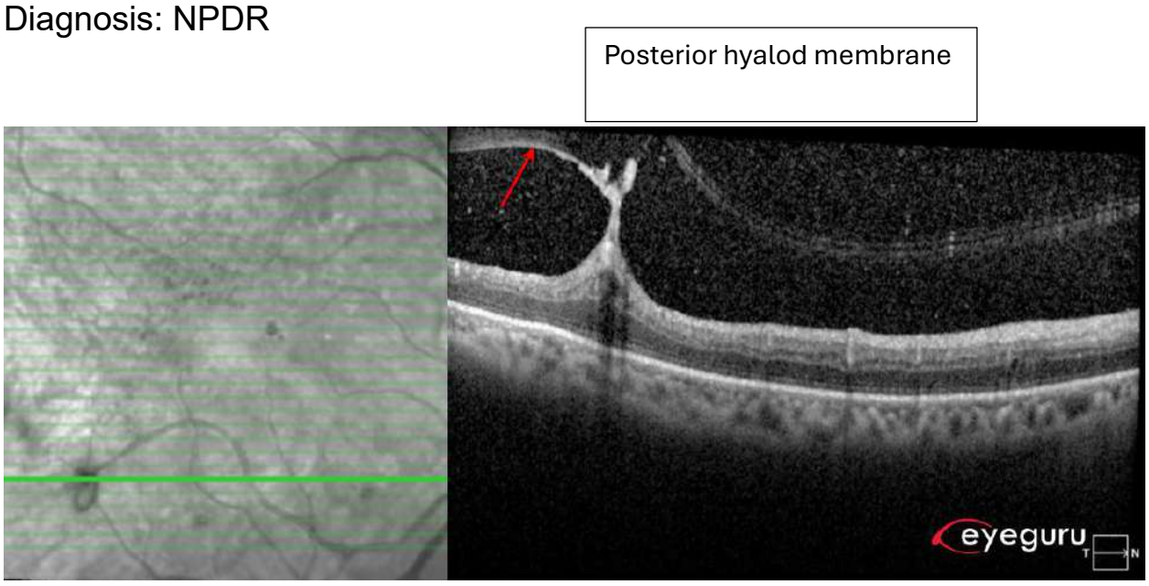


A retinal detachment is usually diagnosed clinically and with exam, but shallow macular detachments are sometimes hard to appreciate early on. If any doubt, a retinal OCT can demonstrate a detachment easily.




Hard exudates (HEs) are deposits of lipid and protein that result from vascular leakage. These are classically seen in diabetic retinopathy and wet age-related macular degeneration. On fundoscopy, they will appear as small, bright yellow bodies. Most of the time, HEs are not visually significant. However, a large HE located within the fovea may cause decreased visual acuity.

Diagnosis: NPDR




Seen here is the posterior hyaloid membrane, which is essentially an interface that becomes visible when the posterior vitreous detaches from the retina. It is important to note that this is technically NOT a complete PVD (posterior vitreous detachment). Differentiating between incomplete and complete PVDs is important for retina surgeries and is out of the scope of this beginner module.

The three strongest primary attachments of the vitreous to the retina are at the:

1. (1) vitreous base
2. (2) margins of the optic disc
3. (3) macula

With age, the Jello-like vitreous begins to contract and liquify. As this occurs, the vitreous can separate from the retina - this is known as a posterior vitreous detachment (PVD). A PVD can most easily be visualized on exam as vitreous debris floating anterior to the optic nerve.

The patient can see flashes and floaters during this progressive process of remodeling, traction and release. In some cases, the separation of vitreous from retina can result in a retinal tear or hole. This allows fluid to flow underneath the retina and increases the risk of a retinal detachment.

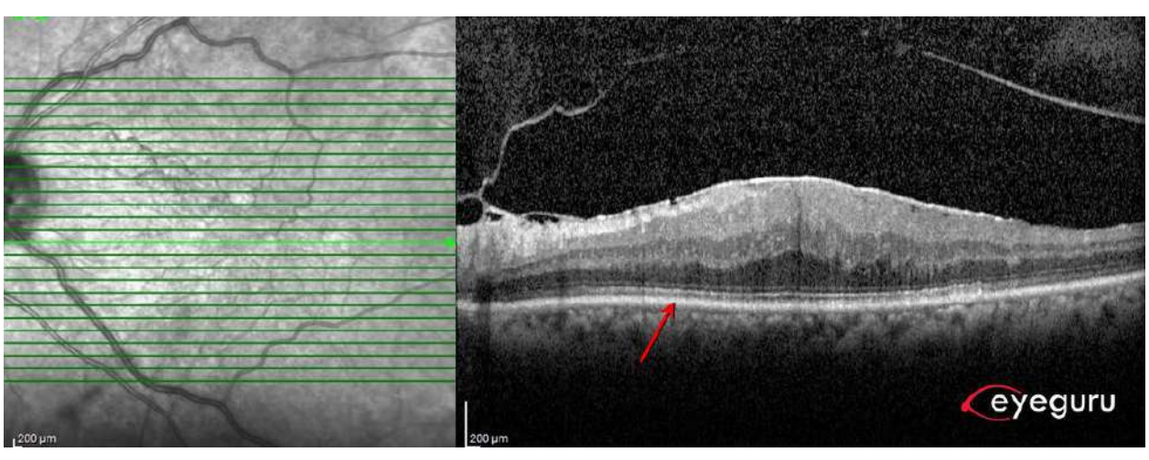
All patients with new symptoms of flashes or floaters should have a complete retinal exam to search for retinal tears, holes, or detachments. If a patient has a PVD in one eye, he or she should be counseled on the warning signs of retinal detachments and the increased risk of a PVD in the other eye.

Diagnosis: Proliferative diabetic retinopathy

**RPE (normal retinal pigment epithelium):** This is an important outer retinal layer to identify, as it is used as a landmark to differentiate between subretinal fluid (SRF) and pigment epithelial detachments (PEDs). The RPE is a layer of pigmented cells that helps to transfer nutrients and waste between retinal photoreceptors in the ellipsoid zone (EZ) and the underlying choroidal circulation.

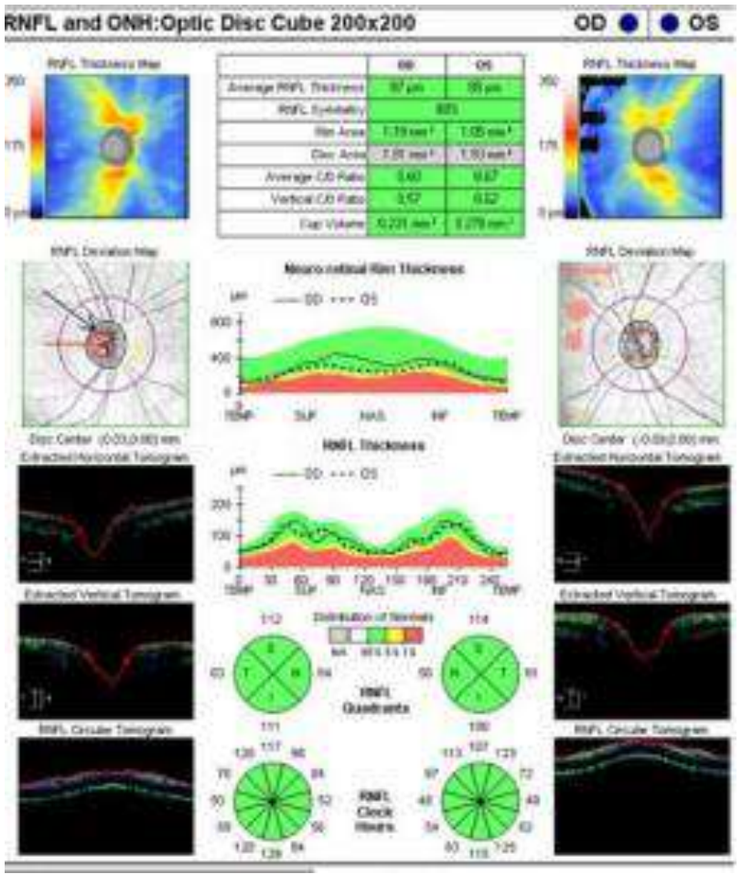
If a retinal detachment results from a tear or a hole (rhegmatogenous retinal detachment), pigment is released from the RPE and enters the vitreous. The pigment can be visualized as small brown flecks floating in the vitreous - this is known as a positive Shafer's sign. If you ever are trying to rule out a retinal detachment, make sure to look for and document a negative Shafer's sign! One study showed that Shafer's sign has a sensitivity of up to ~96% for retinal tears

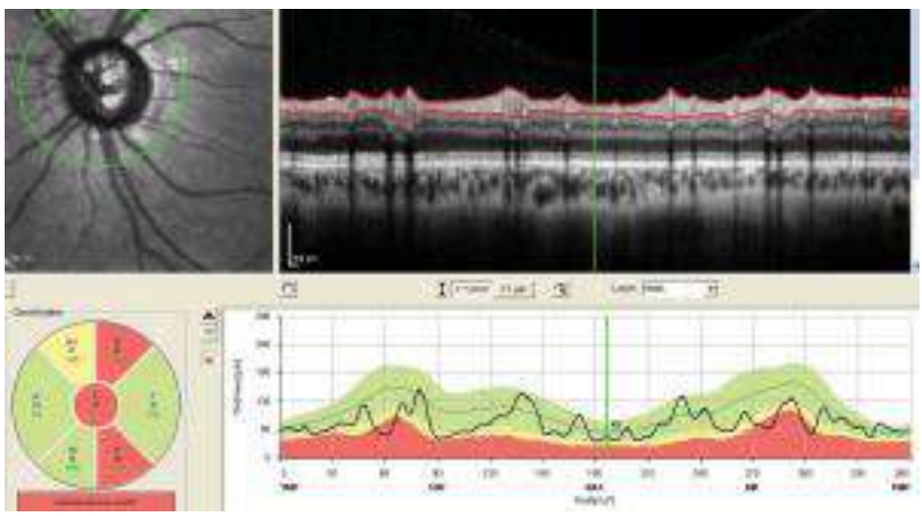
Diagnosis: Significant epiretinal membrane causing loss of foveal contour



## Optic nerve OCT

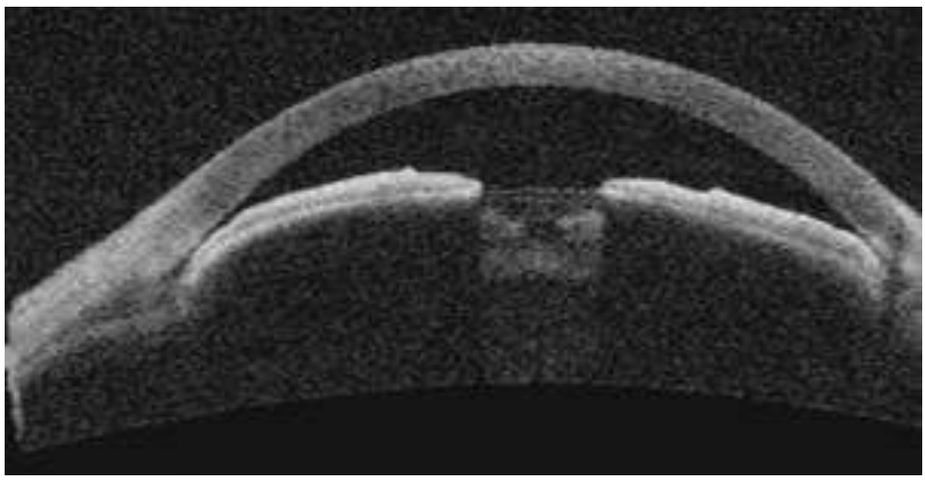
Optic nerve and nerve fiber layer OCT helps in the management of glaucoma. The OCT machines provide automated, serial analysis of the nerve fiber layer thickness, cup-to-disc ratio, and other measurements. They can compare the patient's optic nerve and nerve fiber measurements against age-matched normal patients to show areas of loss. These analyses have become an important adjunct to visual field testing in the treatment of glaucoma. It can also be used to track optic nerve edema.



## Anterior segment OCT (AS-OCT)

Anterior segment OCT is most commonly used to evaluate the iridocorneal angle, such as for patients with narrow angles. It can also be used for corneal biometry to measure the thickness and steepness of the cornea.



AS-OCT of an eye with narrow angles.

**Conclusion**

1. OCT is a non-contact, cross-sectional imaging modality providing high-resolution images of the macula.
2. Summary of the diseases in this article:
    1. Wet age-related macular degeneration (AMD)
        1. Intraretinal fluid (IRF) and subretinal fluid (SRF) accumulation
        2. Pigment epithelial detachments (PEDs)
    2. Diabetic macular edema (DME)
        1. Cystoid macular edema (CME), intraretinal fluid pockets in the outer plexiform layer
        2. SRF (subretinal fluid) if severe
    3. Central retinal vein occlusions (CRVO)
        1. Severe CME
    4. Branched retinal vein occlusions (BRVO)
        1. Retinal edema on temporal side of macula
        2. Chronic RVOs lead to inner retinal atrophy, which is characteristic of the disease
    5. Central serous chorioretinopathy (CSR)
        1. Central SRF (subretinal fluid) collection, no IRF (intraretinal fluid), and a thickened choroid
        2. Can have PED (pigment epithelial detachment) inside the area of SRF (subretinal fluid) accumulation
    6. Epiretinal membrane (ERM)
        1. Inner wrinkling and distortion of foveal contour
        2. Cystoid macular edema if severe

7. Macular hole
    1. Foveal, full-thickness defect
    2. Can have associated
8. Epiretinal membrane (ERM)
    1. Inner retinal wrinkling and distortion of foveal contour
    2. Cystoid macular edema if severe
9. Macular hole
    1. Foveal, full-thickness defect
    2. Can have associated CME (cystoid macular edema)
10. Retinitis Pigmentosa
    1. Loss of photoreceptor layer, with sparing of a central island
    2. Thinning of outer nuclear layer (ONL)
    3. CME can be present (cystoid macular edema)
11. Retinal detachment
    1. Usually diagnosed clinically and with exam, but OCT can be used to check shallow macular detachments

**Interpret Visual Fields: 5 Most Common Patterns**

**What is automated perimetry?**

Here, we'll only talk about the Humphrey visual field perimeter, which is used for 99% of visual field tests. It's an **automated, static** perimeter (unlike Goldmann kinetic perimetry which requires a human operator, and uses a moving target). The Humphrey uses fixed points of light which are shown at different intensity levels. The software automatically varies the intensity of the points of lights at each location to determine the **threshold** – the intensity of light where the patient can see it 50% of the time.

**Which subtest should I order?**

**HVF 24-2**

This is ordered for 90% of glaucoma patients. This is your baseline exam that all glaucoma suspects and glaucoma patients need at routine intervals.

**HVF 10-2**

This is ordered for the 10% of glaucoma patients who are so advanced that the HVF 24-2 is mostly black, with only a central island of remaining vision. Macular diseases including plaquenil toxicity exams also need 10-2.

**HVF 30-2**

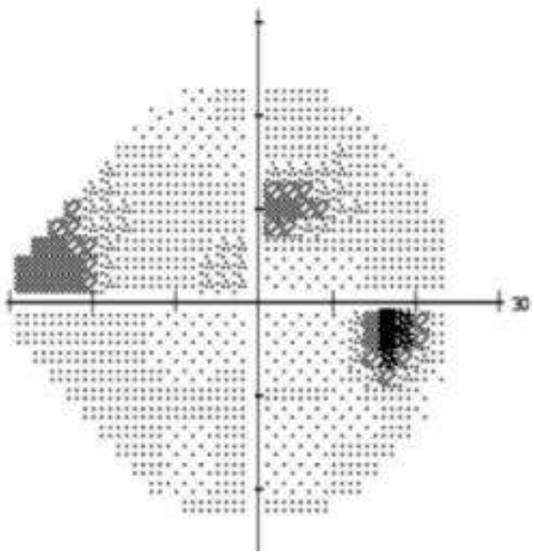
Generally, we order this for neuro patients. It has a wider angle and can capture peripheral field defects.

**Reliability indices**

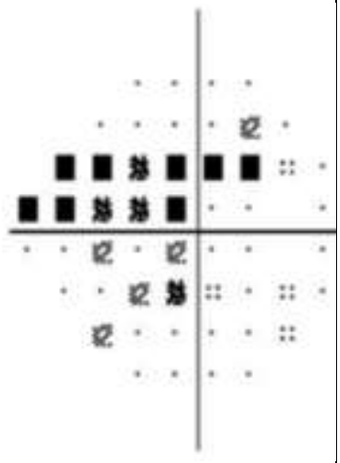
- Name, demographics, etc: Make sure you are looking at the right patient!
- Fixation loss: The HVF will routinely flash dots in the patient's physiological blind spot to check if the patient has his / her gaze fixated on the center. If the patient can actually see the spot, then it's recorded as a fixation loss. Reliable tests have below 20% fixation loss (although many people have their own opinions about these upper limits).
- False positive: The user pressed the button when there was no stimulus. They were "trigger happy". Reliable tests have below 33% false positives.
- False negative: The user did not see a stimulus which was brighter than one they saw earlier in the same test. Reliable tests have below 33% false negatives.
- Stimulus characteristics: 99% of visual fields (VFs) will use the size 3 white stimulus. Other sizes and colors are used for patients with late disease or retinal disease.

**Which picture do I look at?**

Yes, there are a lot of graphs. The two most important to look at are the Grayscale Map and the Pattern Deviation. The rest of this article will explain how to interpret these.



Grayscale Map



Pattern Deviation

**How do I tell if things are changing over time?**

This is the million dollar question. This is what every patient will want to know and how you will decide whether to step up drop therapy, add laser, or take the patient to the operating room.

As a very, very general guideline, you can look at the density / size of the field defect, the pattern standard deviation, and the mean deviation (MD) to see if it is worsening. However, your decision should also take into account the normal variability between each visual field, the optic nerve head appearance, pressures, patient compliance, OCT, visual symptoms, etc.

This is a very complex topic and somewhat beyond the discussion of this post, so talk to your seniors and your attendings if you aren't sure!

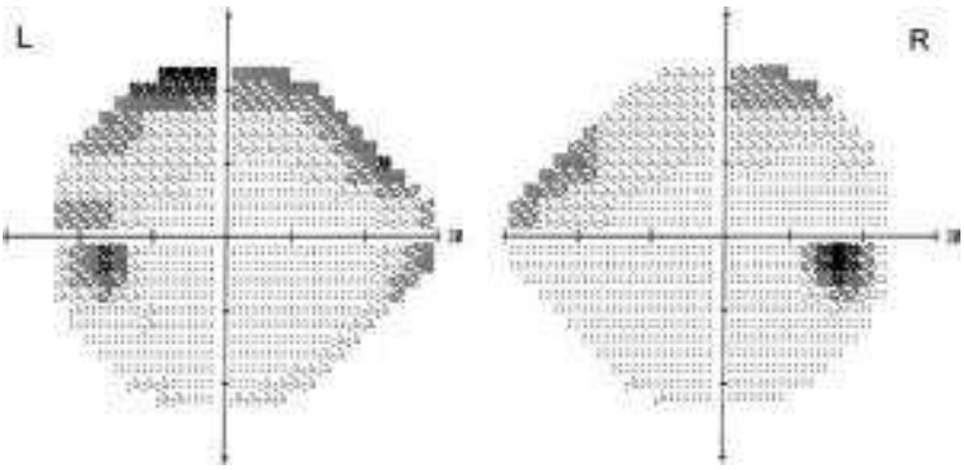
**Top 5 most common visual field patterns**

**1) Nonspecific / low Reliability / inattention / patient hungry**

For every interpretable, reliable visual field you get, you will also get another in which the patient thinks he should be scanning the dome for lights the whole time, is poorly positioned, is exhausted from waiting in your clinic for hours, or is too elderly and arthritic to push the button in time.

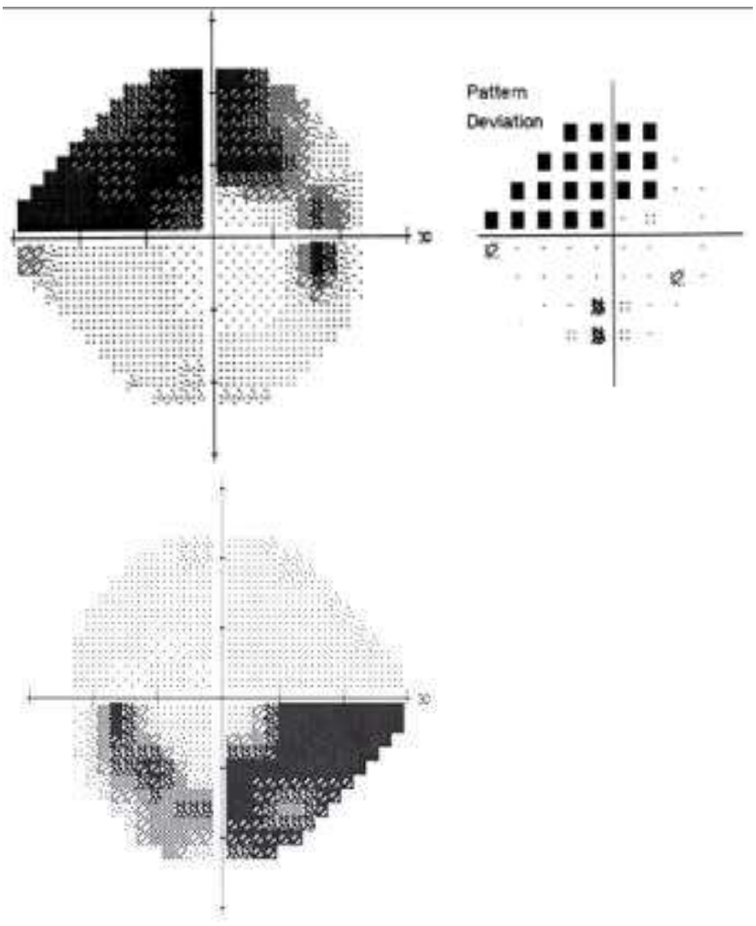
These types of inattention errors will usually register as high fixation losses, false positives, or false negatives. Or, the visual field could just be patchy all over.

If these errors are not too bad, the general gist of the field can be deduced, especially if compared to prior fields. Most often, as long as everything else is stable (IOP, ONH appearance), we just reorder these fields in a few month's time. If you simply cannot get a visual field due to patient cooperation or attention, you can order an optic nerve OCT to follow the optic nerve head objectively (though thinning does not always necessarily correlate to field loss).



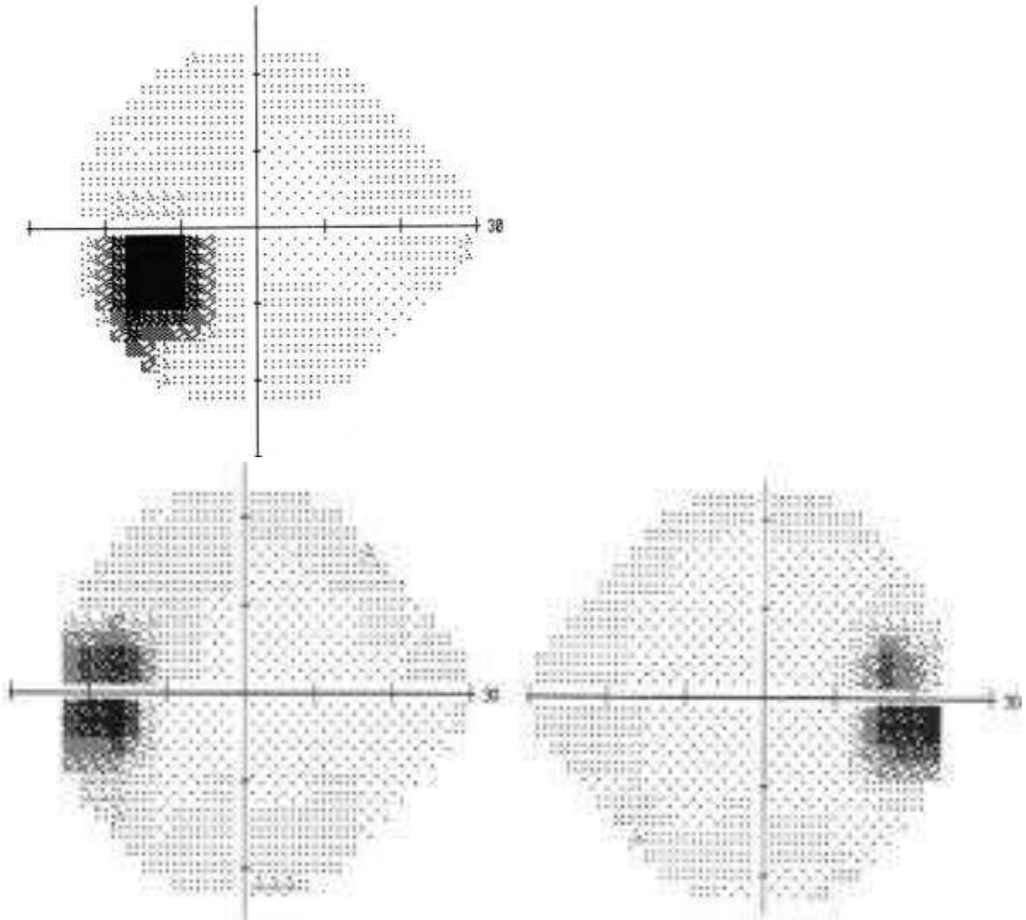

**2) Superior / inferior arcuate defect**

The most common early to mid stage glaucomatous field. The reason these look like arcs and come off the blind spot is that they represent the loss of bundles of nerves as they come out of the optic nerve head. The horizontal border is the horizontal raphe, which is an imaginary line dividing the upper and lower hemispheres of the retina. These are probably 25%-35% of the fields we see.



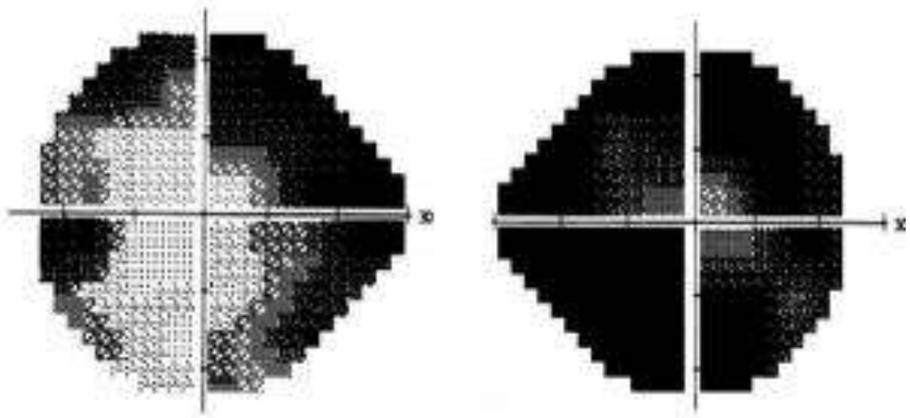

**3) Blind spot enlargement**

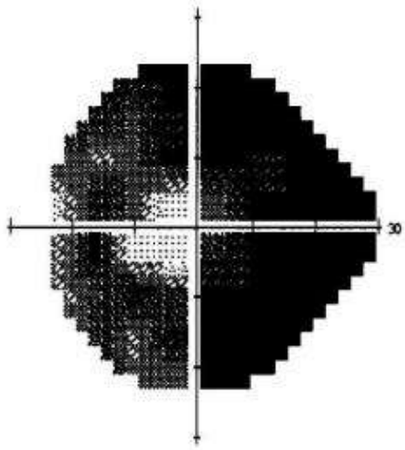
This can be seen in glaucoma, but also can occur with papilledema and optic nerve head swelling. This would likely be seen in patients with idiopathic intracranial hypertension (aka pseudotumor cerebri).



**4) Severe constriction with a central island**

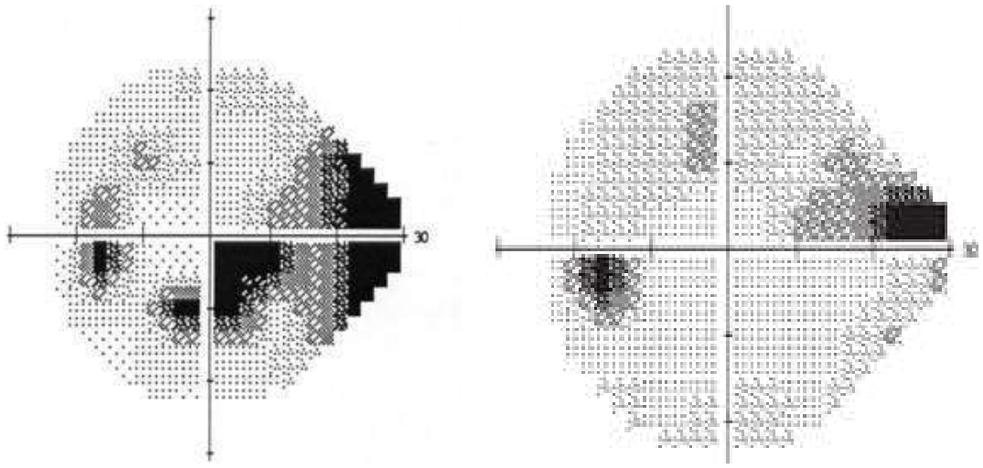
Unfortunately, this is end stage glaucoma. At this point, many patients still have great central vision of 20/20 to 20/50, but peripheral vision is nearly gone. Here, we switch patients over to an HVF 10-2 to better follow their progression.





**5) Nasal steppe**

This is another common glaucomatous field. About 10% of fields show this.



**Conclusion**

Use this order to interpret your Humphrey visual field every time:

1. Confirm it's the right patient with name and date of birth
2. Confirm it's the right/left eye
3. Look at the reliability indices
4. Look at the pattern

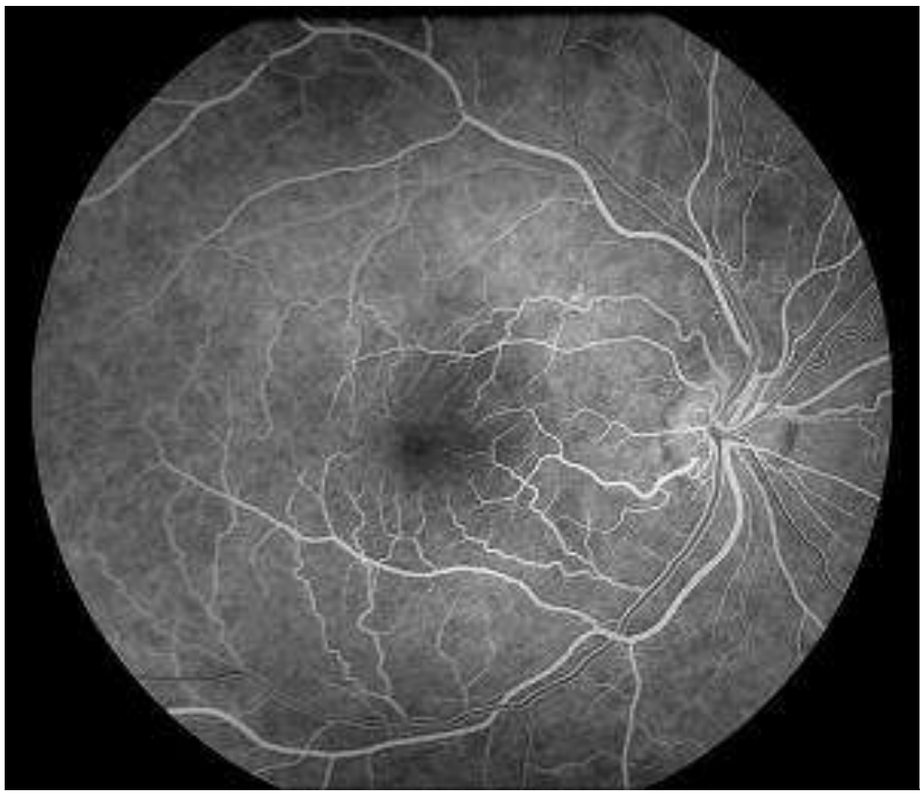
5. Look at the GHT, mean deviation, VFI, and pattern standard deviation
6. Compare to the previous visual fields

**Interpret Fluorescein Angiography: 6 Types Of Defects**

**How does fluorescein angiography (FA) work?**

Fluorescein is an organic dye. When blue light is shined on fluorescein, it fluoresces yellow-green. We do this commonly when looking at the cornea after instilling fluorescein. The same dye can be injected intravenously. A blue light camera can take pictures of the retinal circulation, and the emitted fluorescence is then passed through a yellow-green filter and sent to the camera for the final image.

In a normal eye, fluorescein can not permeate through the endothelial cells of the retinal blood vessels nor can it pass through tight junctions in the retinal pigment epithelium (RPE). The yellow-green wavelength is also heavily absorbed by the RPE so the choroidal fluorescence is blocked. This makes FA good for evaluating the retinal vasculature, not the choroidal vasculature.


A normal FA

In contrast with FA, indocyanine green (ICG) dye is great for evaluating the choroidal circulation. Almost all of the ICG molecules are protein bound, so they do not readily produce retinal leakage or staining. ICG fluoresces in the infrared wavelength and readily passes through the RPE (retinal pigment epithelium).

**Phases of the angiogram**

1. *9-15 seconds* = Choroidal phase (AKA pre-arterial phase): The choroidal hyperfluorescence is present. A cilioretinal artery if there is one will fill in this phase. Delayed choroidal filling time happens in ocular ischemic syndrome (OIS).
2. *1-3 seconds later* = Arterial phase: Arteries are bright, but the veins remain dark.

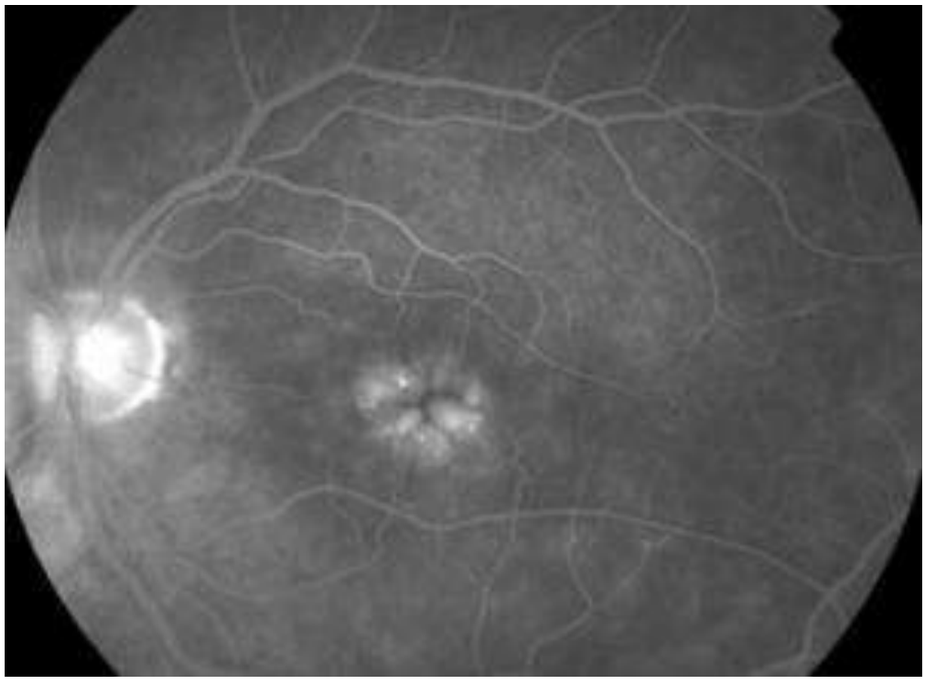
3. Arteriovenous phase: Laminar flow in the veins – the walls of the veins are bright while the center of the vein is still dark.
4. *By 30 seconds* = Venous phase: Complete filling of the veins.
5. *30 seconds – 10 minutes* = Late phase: Dye has recirculated. Things that are going to leak or pool will have done so already.

**Types of hyperfluorescence**

There are 4 types of hyperfluorescence (brightness) in FA:

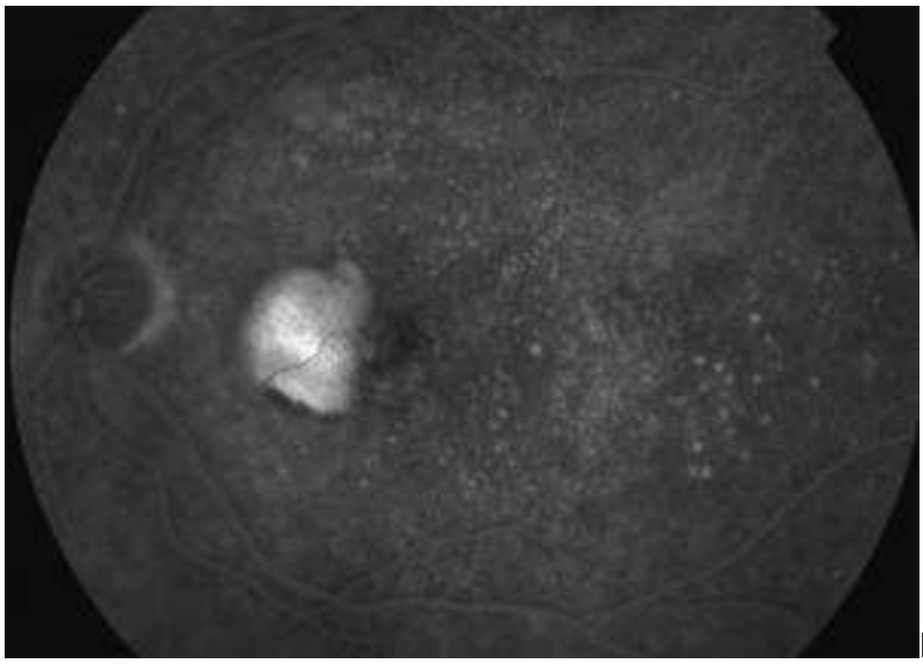
1. Leakage: Hyperfluorescence **progressively enlarges** with fuzzy borders. The dye permeates out of leaky, incompetent blood vessels in the setting of neovascularization, retinal vasculitis, vascular malformations, tumors, or disc edema (dye leaks from prepapillary capillaries).
2. Pooling: Hyperfluorescence progressively enlarges to fill the fluid cavity and then **becomes fixed in size**. Usually the dye fills a cavity like the subretinal space or sub-RPE space (in a PED).
3. Staining: Late hyperfluorescence due to accumulation of fluorescein dye. The hyperfluorescence gradually gets brighter, but the **size stays the same**. Usually a mild amount of fluorescence is seen, but it is never very bright. The optic disc always stains. Additionally, drusen and fibrosis will stain.
4. Window defect: Defect in the RPE allows transillumination of the choroidal hyperfluorescence. Remains **static in size and brightness** and becomes fluorescent with the choroidal phase before the arteries even fill in the early frames.

**Leakage**

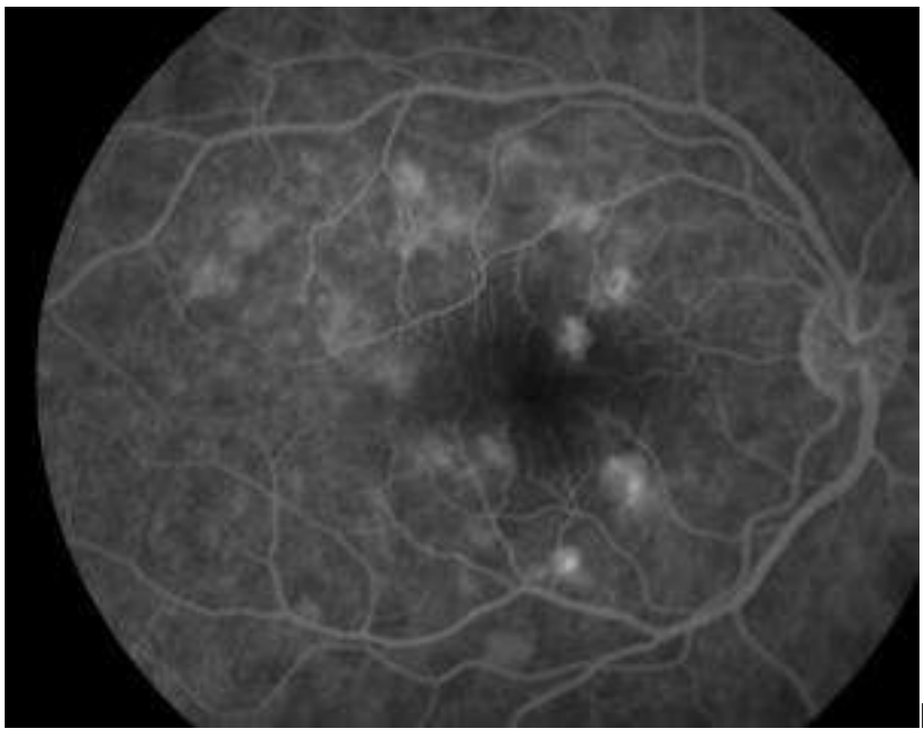


Petaloid leakage from cystoid macular edema

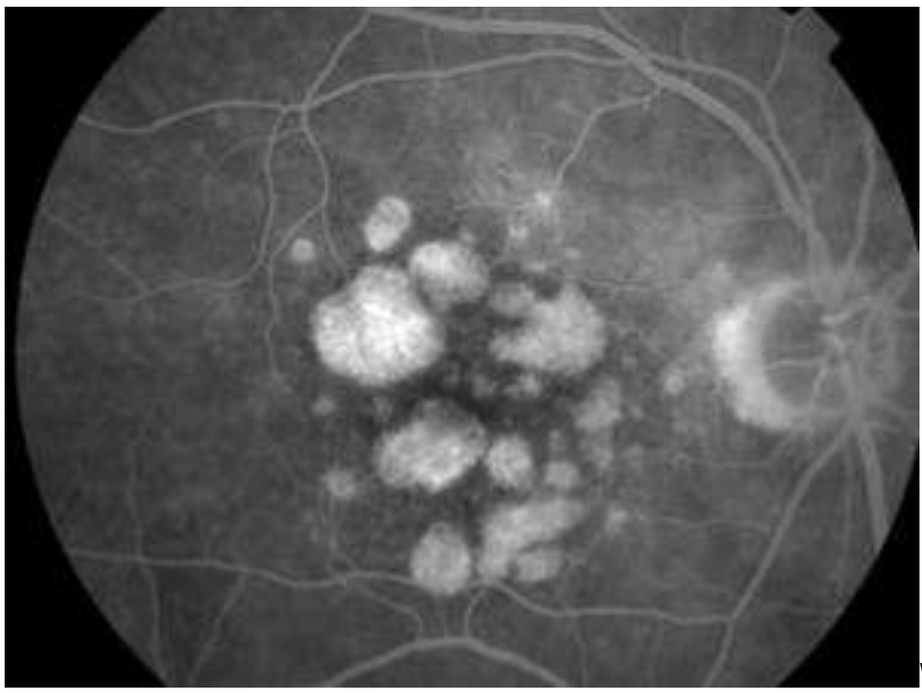
**Pooling**


Pooling from a serous pigment epithelial detachment

**Staining**

 Late staining of acute posterior multifocal placoid pigment epitheliopathy (APMPPE) lesions.

**Window defect**

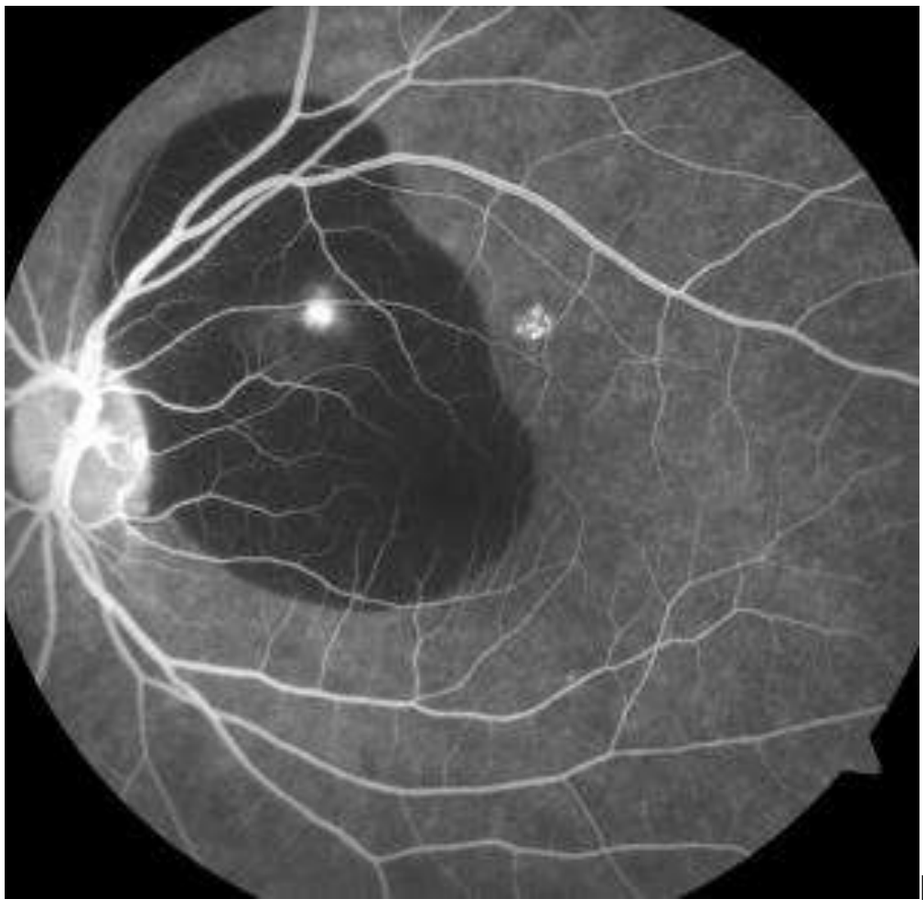
 Window defect from geographic atrophy in AMD

**Types of hypofluorescence**

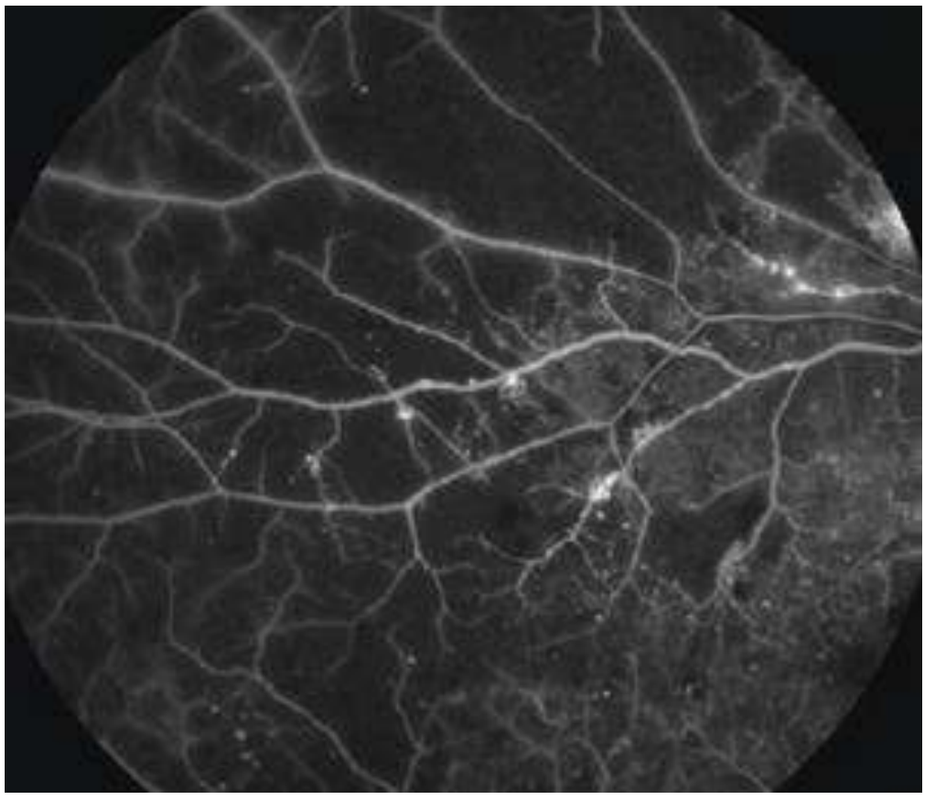
There are 2 major types of hypofluorescence:

1. Blocking: Blood or other opacities block the fluorescence. Blockage of the retinal fluorescence can happen due to preretinal or vitreous hemorrhage. Blockage of the choroidal fluorescence can happen due to nevi or melanomas, Stargardt's disease (lipofuscin blocking choroidal flush leading to a "dark choroid"), or subretinal blood.
2. Filling defect – lack of retinal perfusion due to capillary dropout, retinal artery occlusion and other causes.

**Blocking**


Blocking of choroidal hyperfluorescence from subretinal blood. You know it’s subretinal because the retinal vessels are overlying the dark area.

**Filling defect**


Nonperfusion from diabetic capillary dropout.

## Conclusion

1. Fluorescein angiography (FA) is a great way to evaluate retinal circulation.
2. Nowadays, OCT has greatly reduced the number of FAs performed, though FA still remains a very important modality for assessing many circulatory dysfunctions of the retina.
3. FAs can be evaluated based on distinct phases of dye circulation.
4. Various pathology can cause structures to be hyperflourescent or hypoflourescent. It is important to know generally what pathology correlates with what FA appearance.

## Interpret Ophthalmic Ultrasound: 5 Most Common Scans

### Uses for ophthalmic ultrasound

There are a few different types of ultrasound that can be performed on the eye. The “A scan” is performed by placing an ultrasound transducer directly on the cornea or using an immersion shell after some numbing drops are applied. It performs a single line scan which can be used to measure axial length or numerically compare the reflectivity of different structures. The “B scan” is usually performed by placing the transducer on the patient’s closed eyelid. It creates the typical 2D image that you associate with ultrasound. Ultrasound biomicroscopy (UBM) is performed with a much higher frequency than the A and B scans and is primarily used to evaluate the anterior chamber angle and ciliary body.

Here is a summary of what the different ultrasound scans are used for:

1. B scan: Used for evaluating the retina, retinal detachments, vitreous choroidal masses, tumors, and nevi.
2. A scan: Used for ultrasound biometry (e.g. calculating axial length).
3. Ultrasound biomicroscopy (UBM): Used for evaluating the iridocorneal angle and ciliary body.

### Basics of B scan

We’re going to focus on B scan in this article. Here are the key concepts you need to know to understand B scan ultrasound for the eye.

### Scan orientations

First, for the purposes of ophthalmic ultrasound, the posterior of the eye is centered on the optic nerve, not the fovea.

There are 3 scan orientations.

1. Axial: The probe is placed in the visual axis. A horizontal axial view would show the optic nerve and macula.
2. Longitudinal: The probe is placed on the eye with the plane of the ultrasound beam facing towards the pupil. The ultrasound images will show the retinal periphery to the posterior pole.
3. Transverse: The probe is placed on the eye, but oriented so the plane of the ultrasound beam is parallel to the limbus. If the entire retina were a clock, the ultrasound image from a transverse scan would show several clock hours of the retinal periphery in one frame.

Of the 3 orientations, transverse is used the most because it allows you to pan the probe and integrate that quadrant of the retinal periphery into a 3D mental image. This is something that you will understand and get much better at with a little practice in clinic.

### Dynamic scans

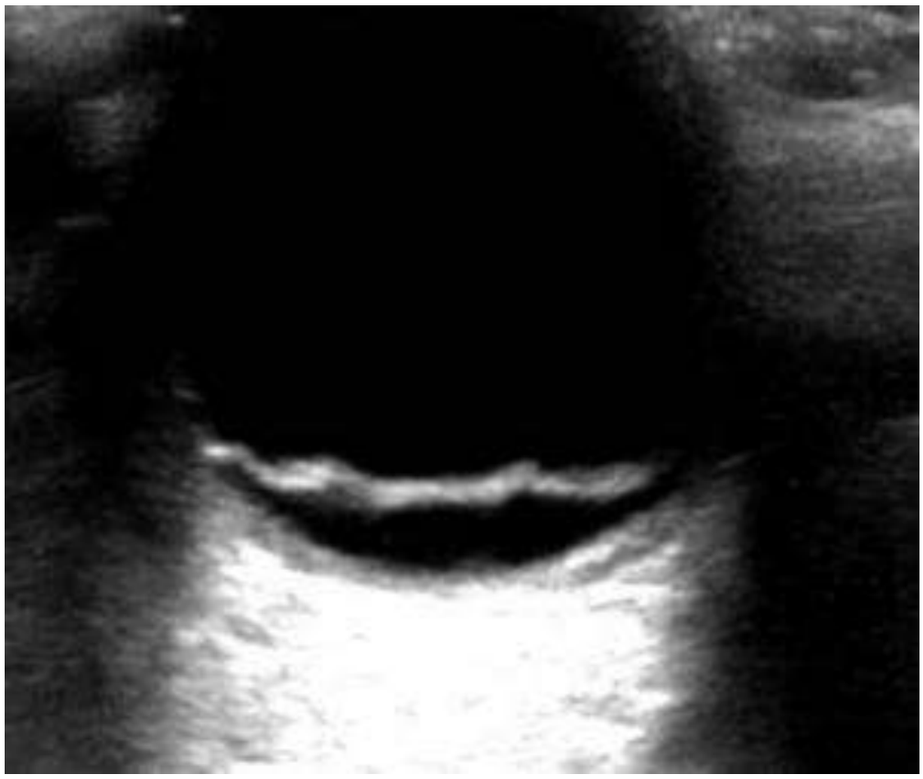
Performing a dynamic scan just means having the patient move their eye back and forth to evaluate how the structures are moving. This is most

important in differentiating vitreous detachments from more emergent retinal detachments. The posterior hyaloid membrane (which separates the vitreous from the retina) moves rapidly and tumbles loosely in the eye, like a “washing machine”. The retina undulates more slowly and is more echogenic than the hyaloid.

**Example ultrasounds**

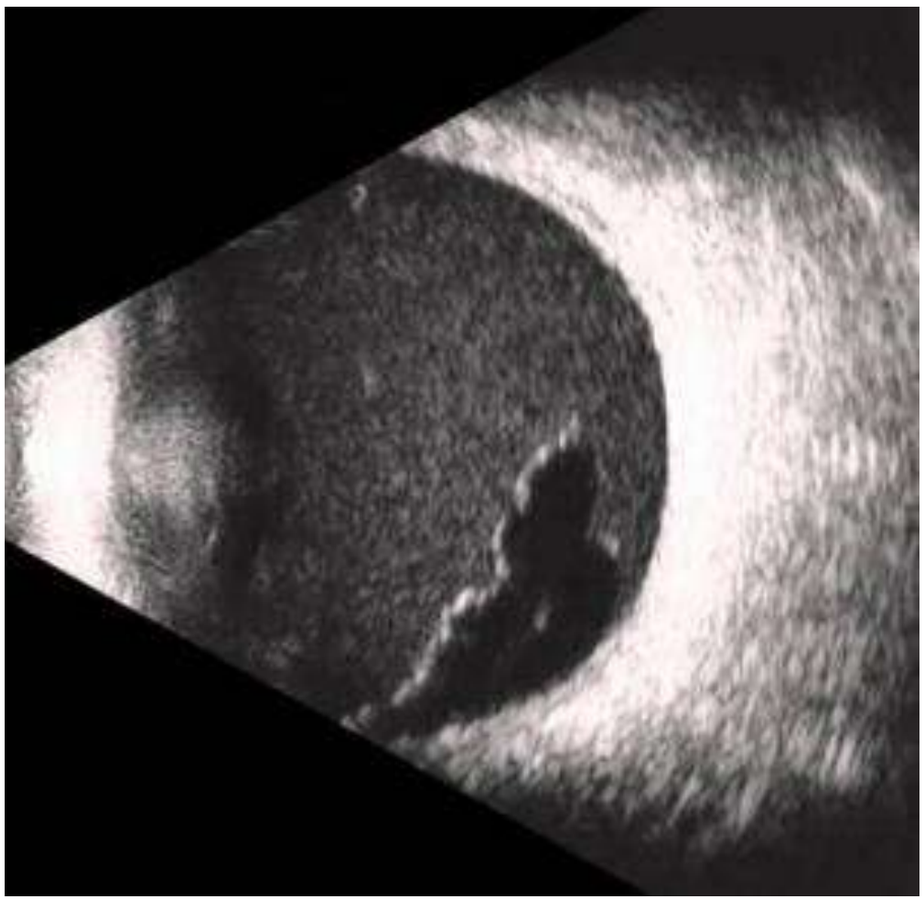
**Retinal detachment**

Retinal detachments have a characteristic undulating movement of the retina with dynamic scans. The retina moves at a slower speed than the posterior hyaloid and its reflectivity is higher. You can use ultrasound using the axial, transverse and longitudinal views to delineate where the retina has detached.



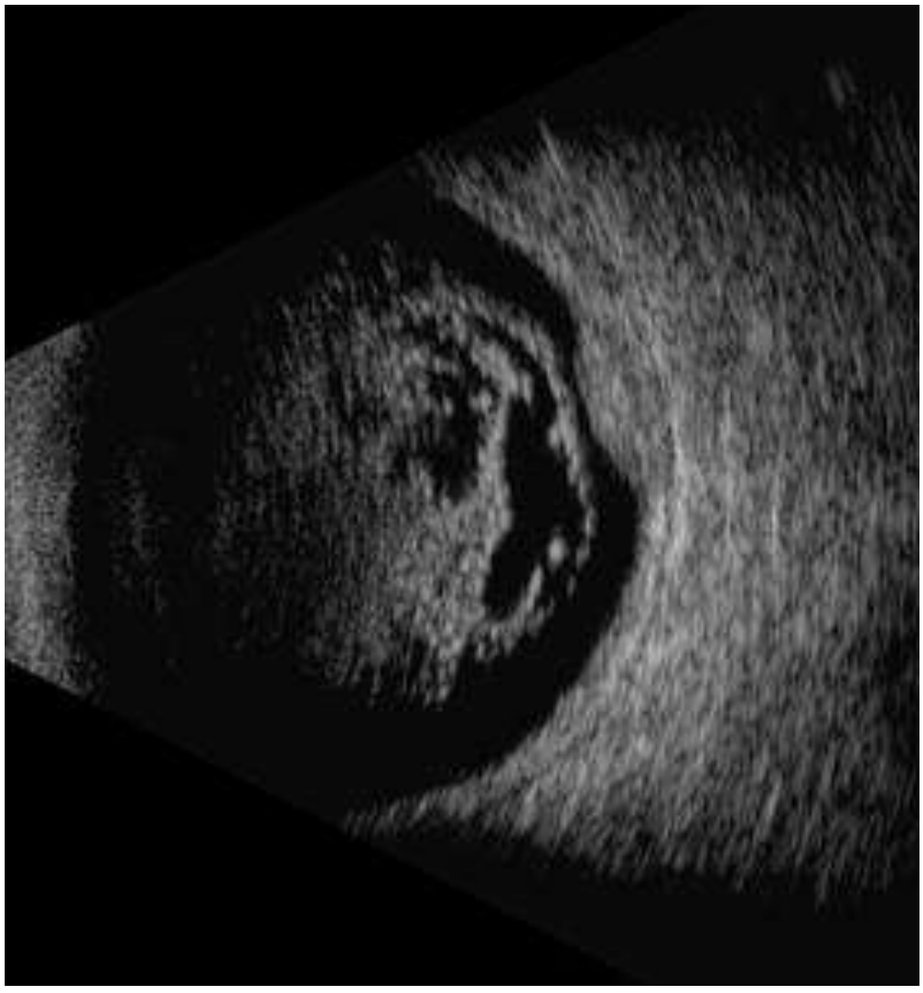
**Vitreous hemorrhage**

Vitreous hemorrhages are seen commonly in the setting of acute vision loss in one eye. They can happen in diabetic retinopathy and almost every other retinal neovascular disease.



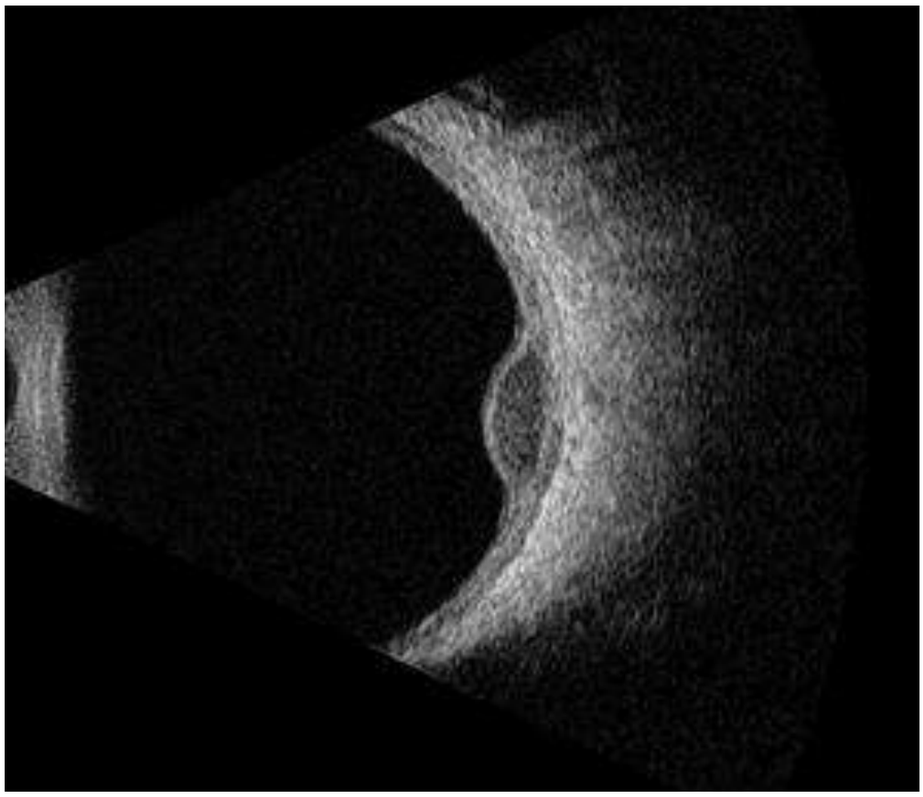
**Choroidal nevus**

Choroidal nevi can be differentiated from choroidal melanoma in that they have a uniform, high internal reflectivity.



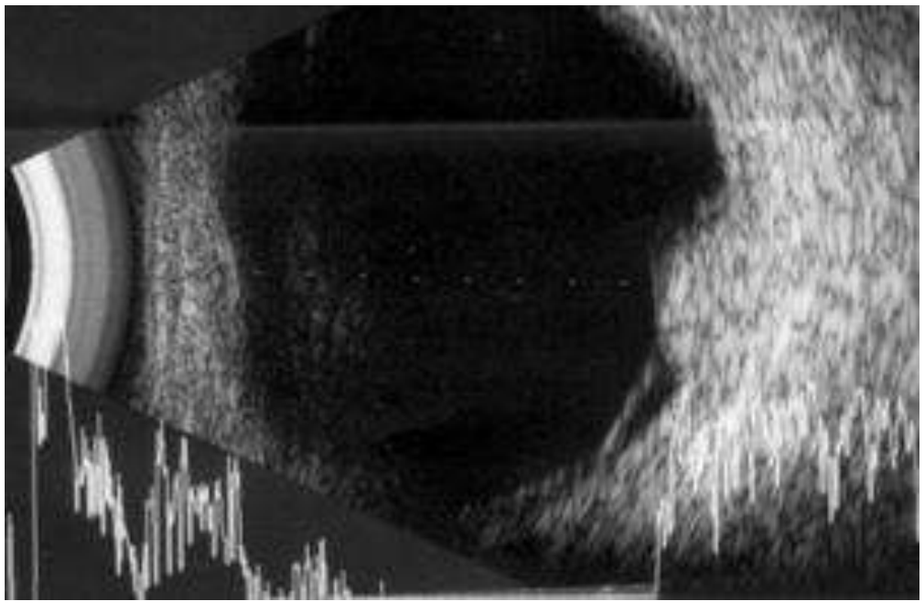
**Choroidal melanoma**

Choroidal melanomas have a low to medium internal reflectivity due to the fact that they are more vascular.



**Choroidal hemangioma**

Choroidal hemangiomas have a uniform, high-internal reflectivity.



## Interpret Corneal Topography: 5 Clinical Uses

***Note:*** Technically, topography and tomography are different imaging modalities (explained below). However, both are colloquially referred to as topography. Except for our section differentiating between them, we will also refer to both as topography.

In this article, we will review what corneal topography and tomography are, why they are useful, and how to interpret a normal Pentacam scan. We will also review 5 clinical uses for topography that will prepare you well for cornea clinic.

### Topography vs. Tomography

This is the technical distinction between topography and tomography:

1) Corneal to**p**ography is a non-invasive imaging technique for mapping the surface curvature and shape of the **anterior** corneal surface.

- How it's done:
    - Placido disc (topography): Evaluates the cornea based on the reflection of concentric rings (mires).
        - Widely spaced rings = flatter
        - Closely spaced rings = steeper
- **Devices: Orbscan, Atlas, NIDEK OPD**

2) Corneal to**m**ography computes a 3-D image of the cornea and assesses the entire cornea, **anterior** and **posterior** surfaces. Nowadays, tomography is most commonly used.

- How it's done:
    - Scheimpflug imaging (tomography): Evaluates the cornea using a camera that captures cross-sections of the cornea as it rotates
- **Devices: Pentacam, Galilei, Sirius**

### Utility

- Management of astigmatism in cataract surgery and after corneal transplant

- Screening candidates for refractive surgery by identifying irregular astigmatism and helping estimate postoperative ectasia risk
- Detection of ectatic disorders such as keratoconus, pellucid marginal degeneration and post-LASIK ectasia
- Determining visual significance of corneal and conjunctival lesions, such as pterygia and Salzmann's nodular degeneration
- Guiding suture removal and placement of corneal relaxing incisions

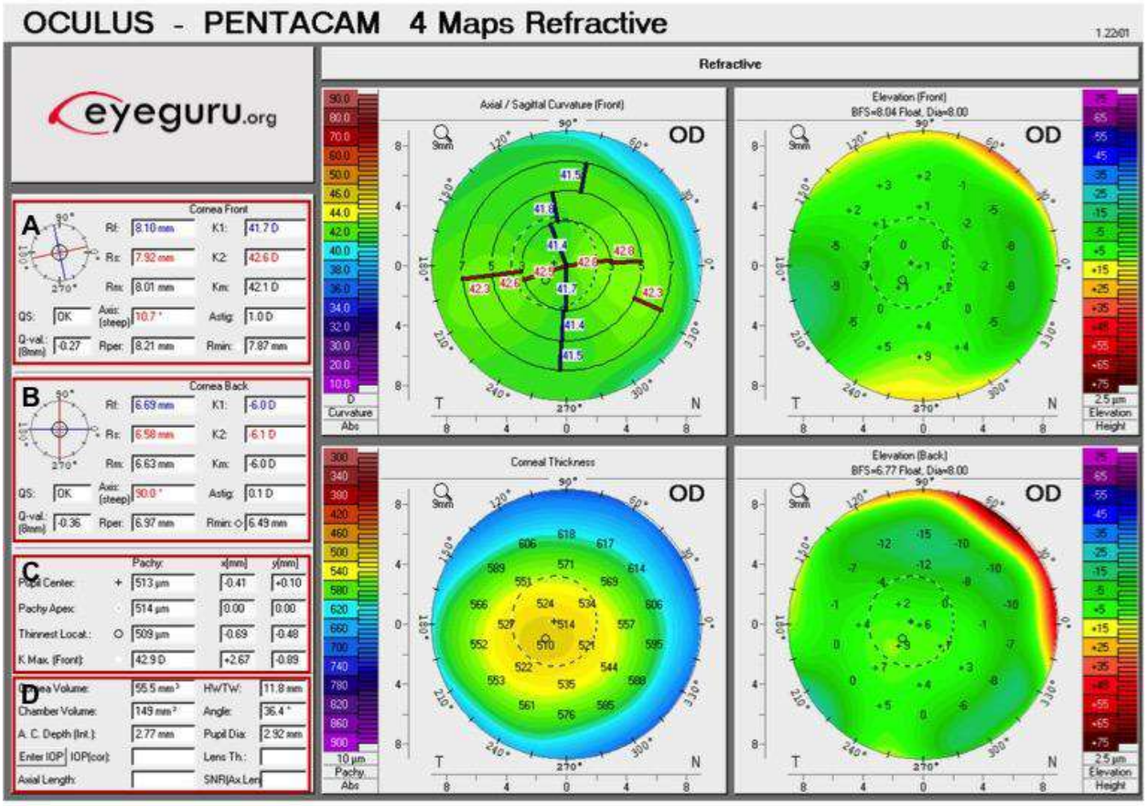
**Basic Principles**

Colored Maps: You will see a rainbow of colors on every topographic map. These range from warm colors (red, orange, yellow), to neutrals (green) to cool colors (blue, purple). On our representative Pentacam images below, you will see four different types of maps.

- 1) Axial map (top left)
    - Useful for assessing irregularity of astigmatism and planning suture removal after PK
    - **Warm** colors = steep (think "*steep*ing *warm* tea")
    - **Cool** colors = flat
- 2) Corneal thickness, aka pachymetry map (bottom left)
    - Displays distribution of corneal thicknesses across the entire measured area.
    - **Warm** colors = thin (think "in the *heat* wear *thinner* layers")
    - **Cool** colors = thick (think "in the *cold* wear *thicker* layers")
- 3) Anterior elevation map (top right)
    - Useful for assessing regularity of astigmatism, location of astigmatism and surgical planning for AK, toric planning
    - Warmer colors indicate where the cornea is elevated above the best fit sphere; cooler colors indicate where the cornea is depressed below the best fit sphere
- 4) Posterior elevation map (bottom right)
    - Useful for identifying forme fruste keratoconus
    - Warmer colors indicate where the cornea is elevated above the best fit sphere; cooler colors indicate where the cornea is depressed below the best fit sphere

**Normal Cornea**

**Expected topography:** Progressive flattening from center to the periphery by 2-4D, with the nasal area flattening more than the temporal area.




## Interpreting Pentacam Values[1]

### A) Anterior corneal values

- $K_1$, $K_2$, $K_m$: The two major meridians (**$K_1$, $K_2$**), determined using the 3mm ring, are 90 degrees from each other. Red corresponds with the steep meridian whereas blue corresponds with the flat meridian. **$K_m$** is the average of **$K_1$** and **$K_2$**
- $R_f$, $R_s$, $R_m$: Radii corresponding with **$K_1$, $K_2$, and $K_m$**, respectively
- QS: Quality score (I.e. “OK,” “Data gaps,” “Fix,” “Model”) may alert the technician to retake the exam due to suspect quality
- Q-val: Describes the corneal shape factor, or eccentricity of the cornea. The ideal value is -0.26.
    - More negative values may suggest keratoconus or hyperopic correction whereas positive values may suggest myopic correction.
- Axis: The meridian that requires no cylinder power to correct astigmatism
- Astig: The central corneal astigmatism
- $R_{per}$: Average radius of curvature between the 6mm and 9mm zone center
- $R_{min}$: Smallest radius of curvature in entire field measurement
    - $R_{min}$ may be elevated in keratoconus

### B) Posterior corneal values

The same variables described for the front of the cornea are used to characterize the back of the cornea.

**C) Pupil center:** Calculated by finding the center point based on edge detection on the iris then the distance is calculated in mm

- Pachy apex: Corneal thickness at the apex
- Thinnest Location: Thinnest point over anterior corneal surface
- K Max (Front): Steepest point over anterior corneal surface

**D) Values used in IOL calculations** (out of scope of this article)

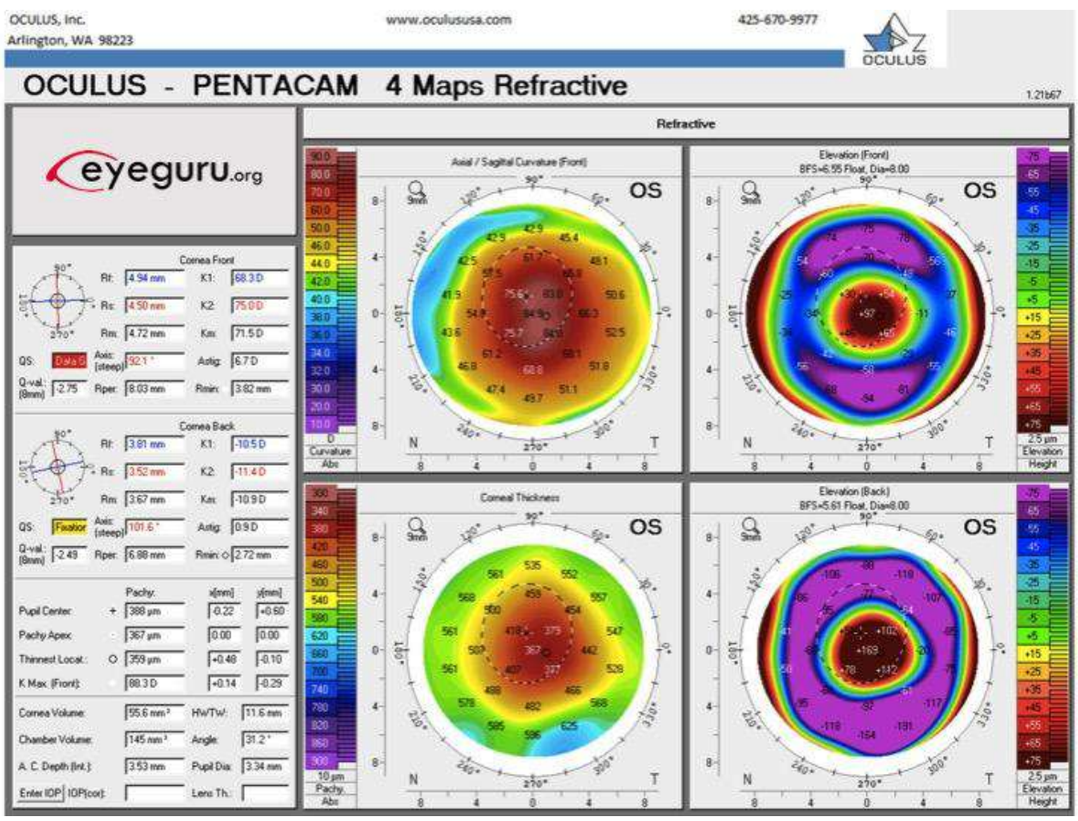
**5 Clinical Uses**

**1) Keratoconus**

Topographic diagnosis of keratoconus is suggested by:

1. High central corneal power
2. Large difference between the power of the corneal apex and periphery
3. Differences in steepness between the two corneas of a given patient.

**Expected topography:** inferior steepening on anterior axial map and corresponding thinning on pachymetry map. There are many systems to grade keratoconus. Here are some examples of various systems, but these are not necessarily the only criteria by which to rule in or rule out keratoconus.

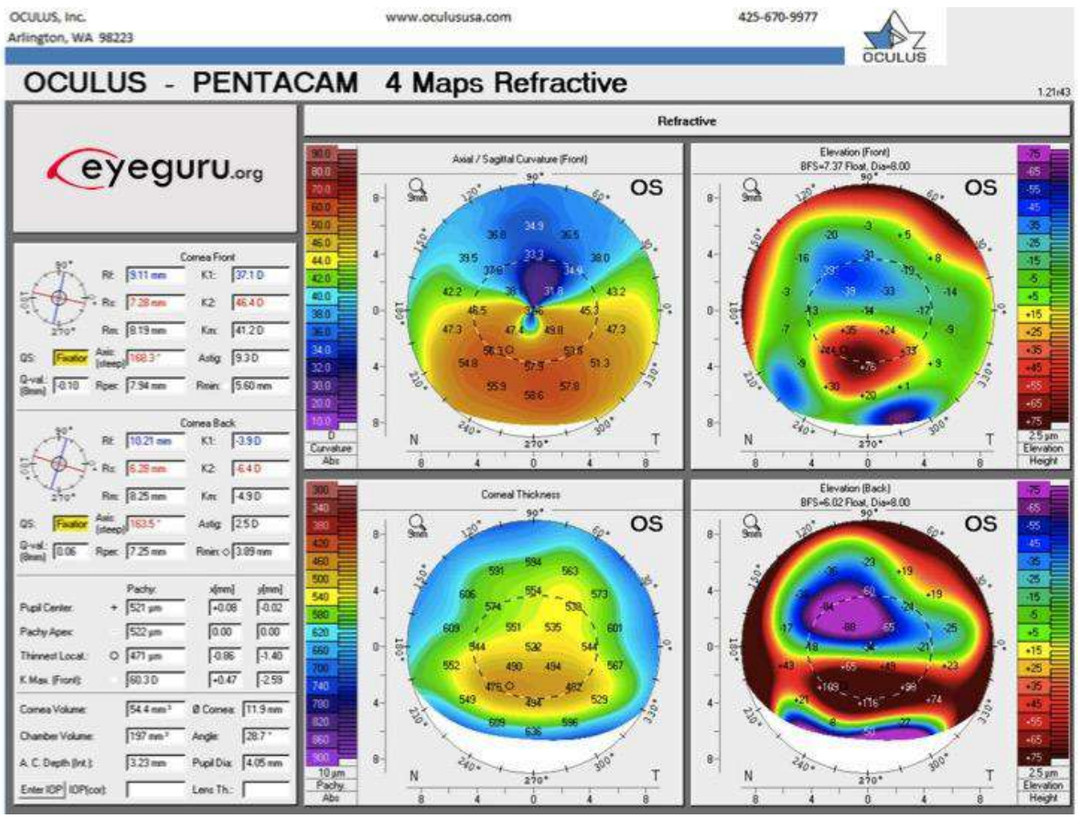



| | Cutoff points for Keratoconus |
|---|---|
| Central $K^2$ | Normal <47.2D<br>Forme Fruste Keratoconus 47.2-48.7D<br>Keratoconus >48.7D |

| | |
|---|---|
| Inferior-superior asymmetry index | >1.2D |
| Astigmatism | >2.5D |
| Orbscan II topography posterior elevation[3] | ≥35µm subclinical keratoconus<br>≥51µm keratoconus |
| Orbscan II topography anterior elevation[3] | ≥16µm subclinical keratoconus<br>≥19µm keratoconus |
| Pentacam Scheimpflug corneal tomography posterior elevation[4] | Normal ≤+17µm<br>Suspicious +18µm to +20µm<br>Risky >+20µm |
| Pentacam Scheimpflug corneal tomography anterior elevation[3] | Normal ≤+12µm<br>Suspicious +13µm to +15µm<br>Risky >+15µm |

**2) Pellucid marginal corneal degeneration**

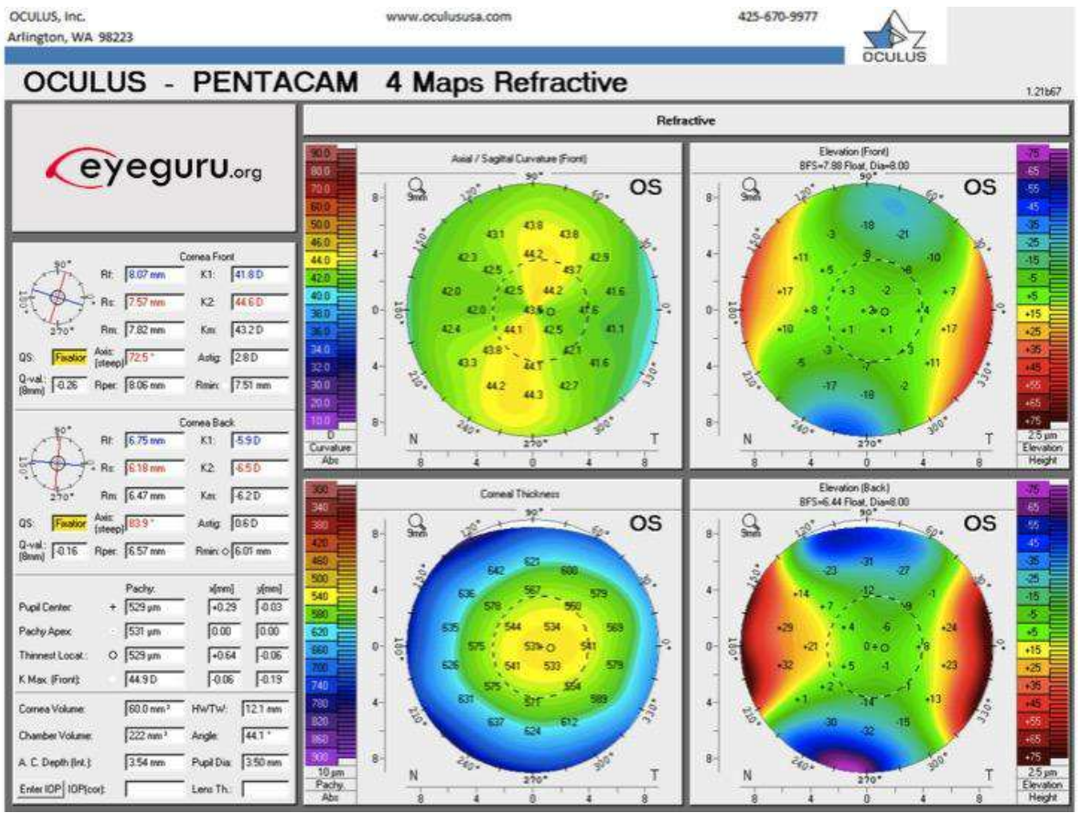
**Expected topography: against-the-rule** “**crab claw**” or “butterfly” pattern on axial map




**3) Astigmatism**

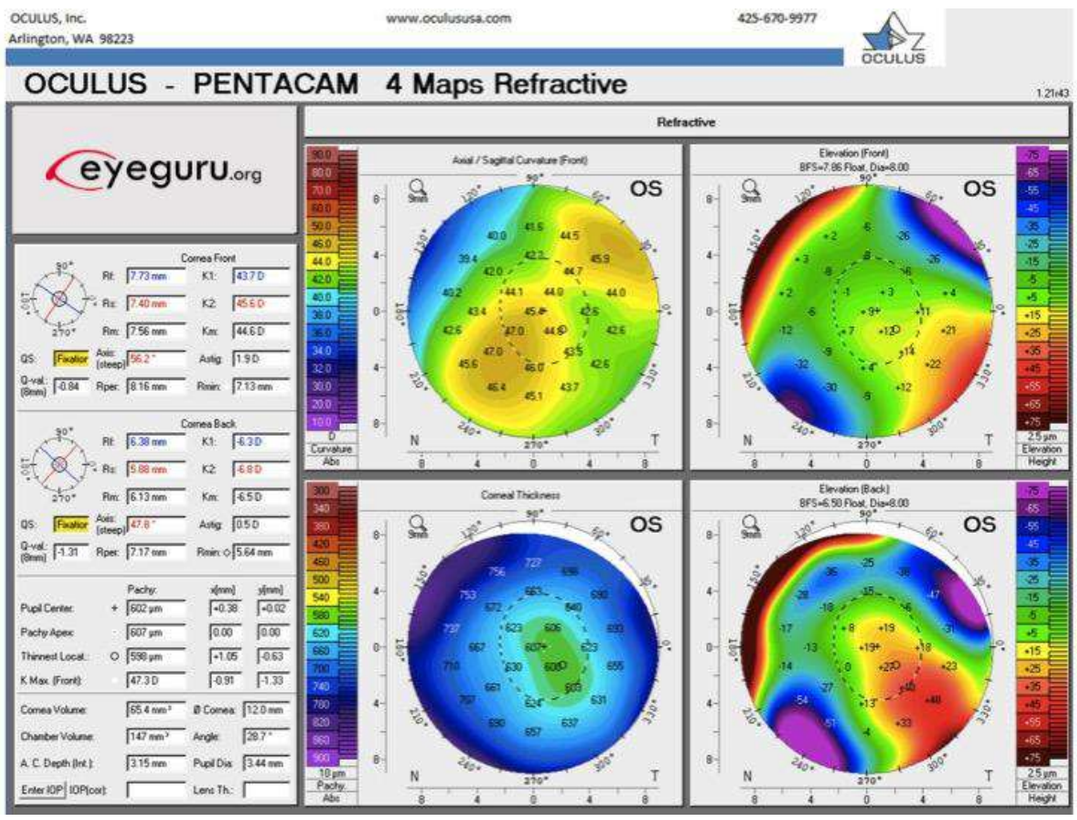
***Regular astigmatism*:** uniform steepening along a single corneal meridian that can be fully corrected with a cylindrical lens (BCVA of 20/20 or better)

**Expected topography: symmetric** “**bow-tie**” along a single meridian

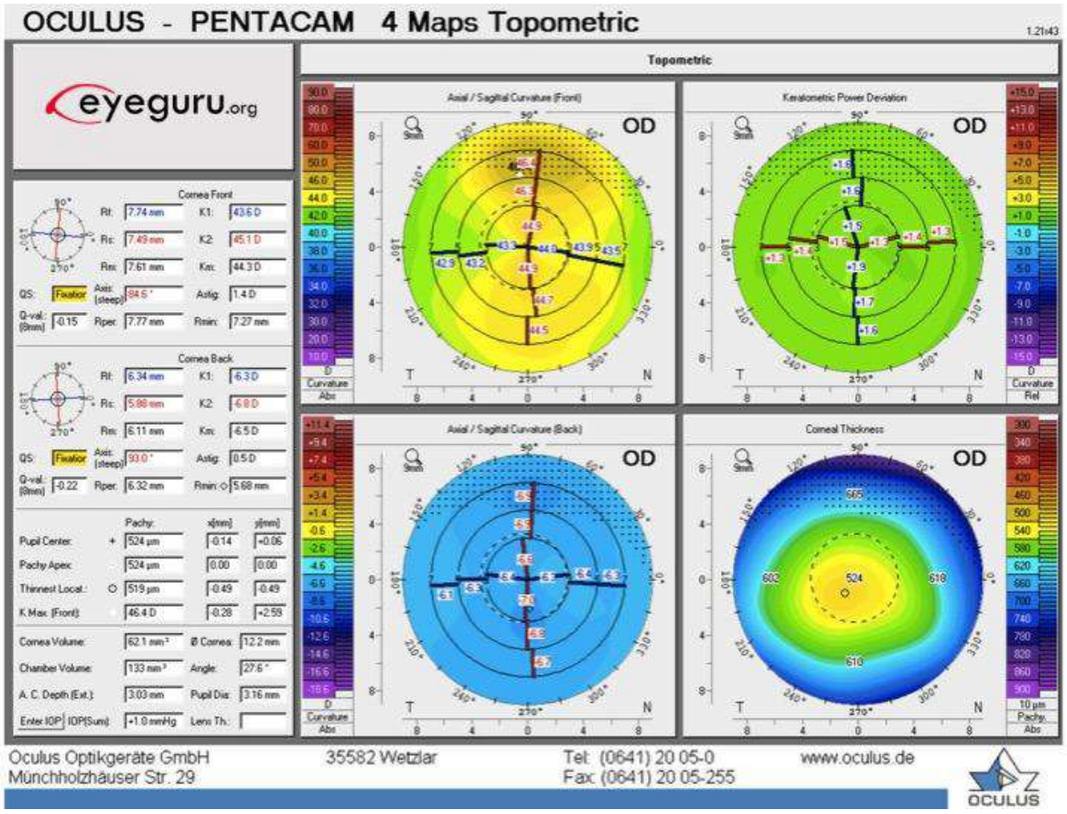



***Irregular astigmatism***: nonuniform steepening that cannot be corrected by cylindrical lens (BCVA of 20/50 or worse due to irregular astigmatism).

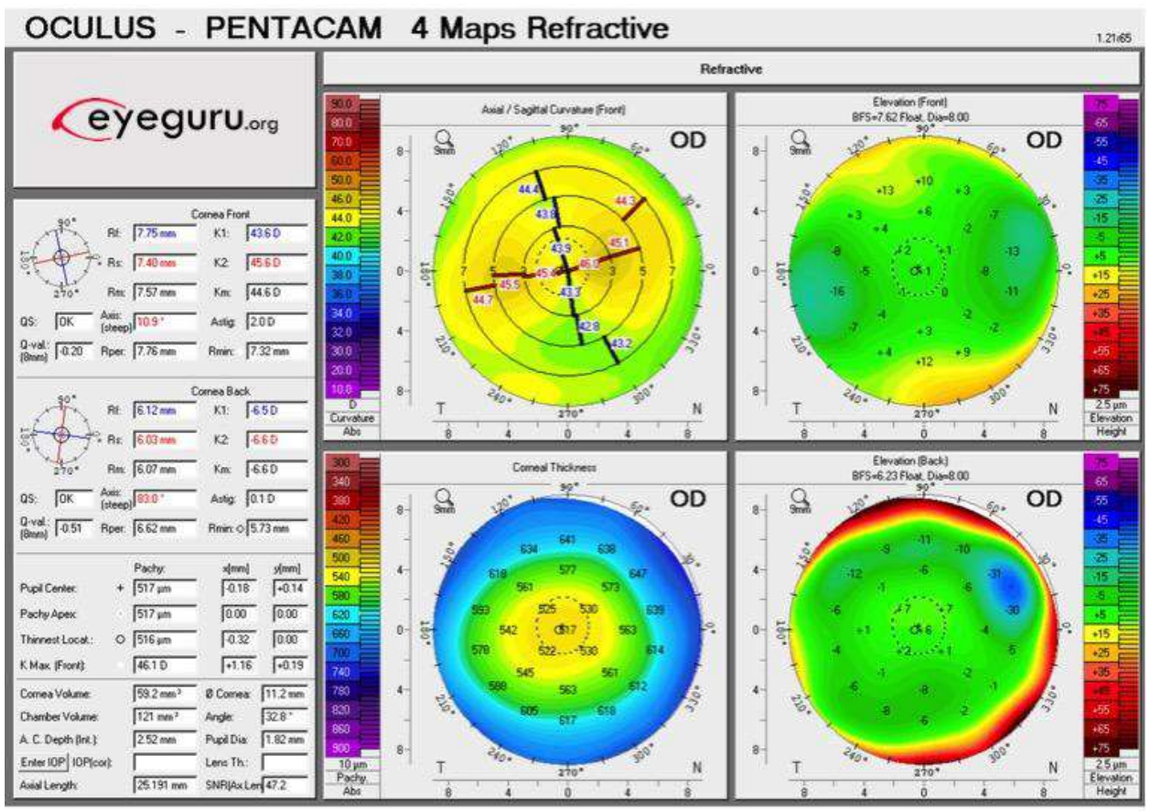
**Expected topography: steep** and **flat axes less or more** than **90 degrees** apart




***With-the rule*** *astigmatism*: Steeper in the **vertical** meridian




***Against-the-rule*** *astigmatism*: Steeper in the **horizontal** meridian




**4) Refractive error**

**Myopia** is associated with **steeper** central corneal curvature

**Hyperopia** is associated with **flatter** central corneal curvature

However, these are not hard and fast rules, as axial length plays a big role in the overall myopia/hyperopia of the eye.

**5) Refractive surgery**

Preoperative assessment should be performed to rule out ectactic (e.g. irregular) patterns, which occur in disorders like keratoconus and pellucid

marginal degeneration. If an ectactic disorder is suspected, LASIK is not recommended.

Refractive surgery itself can induce corneal ectasia. In preoperative planning, percent tissue altered (PTA) is used to estimate the risk of inducing a cornea ectasia. Generally, a PTA < 40% is accepted as a lower risk in a normal eye.[5]

**PTA = (FT + AD)/CCT**

PTA: percent tissue altered; FT: flap thickness; AD: ablation depth; CCT: preoperative central corneal thickness

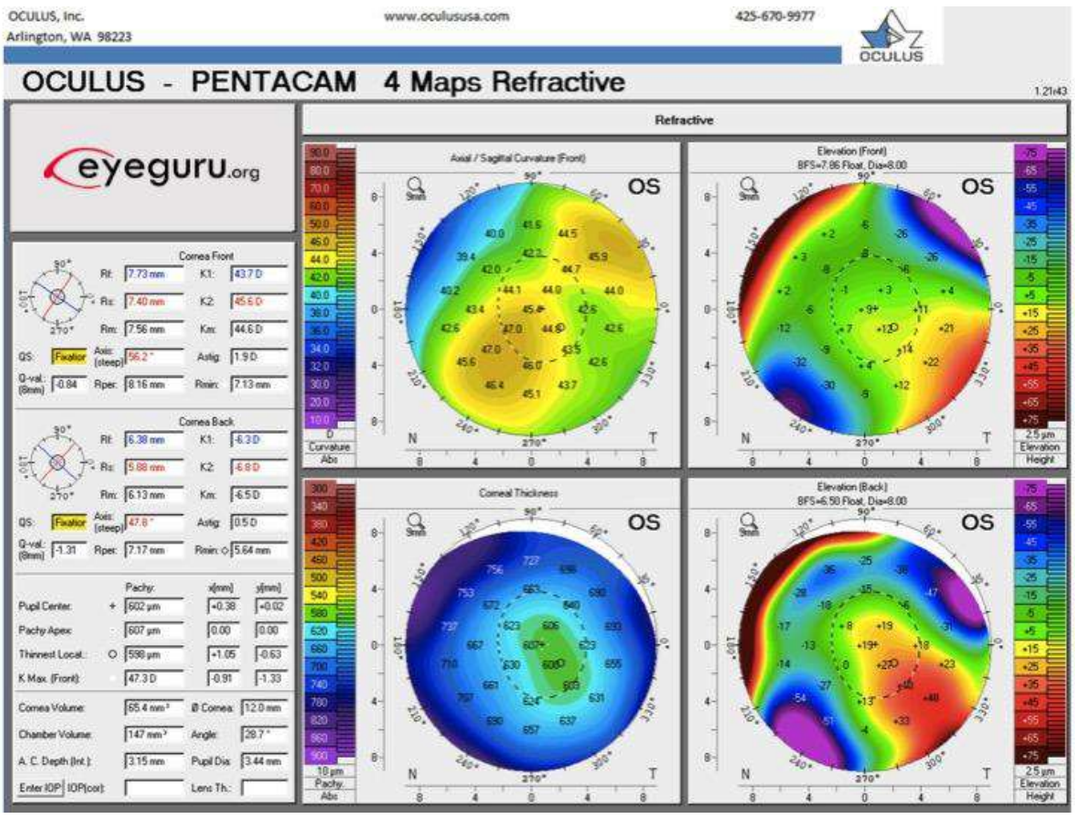
Postoperative assessment is performed to evaluate any dioptric changes at the corneal level and to rule out decentered or incomplete ablation, ectasia or other changes.

**Quick Tips for Clinic**

- Choose which post-keratoplasty suture to remove based on the steep axis of astigmatism on topography. Removing sutures along the steep axis leads to corneal flattening.
- Candidates for Toric IOL will have regular symmetrical astigmatism, appearing as a “bowtie” or “figure-eight” pattern on topography (can be with or against-the-rule)
- Keratoconus or pellucid eyes will have an irregular astigmatism, often with inferior steepening and corneal thinning,

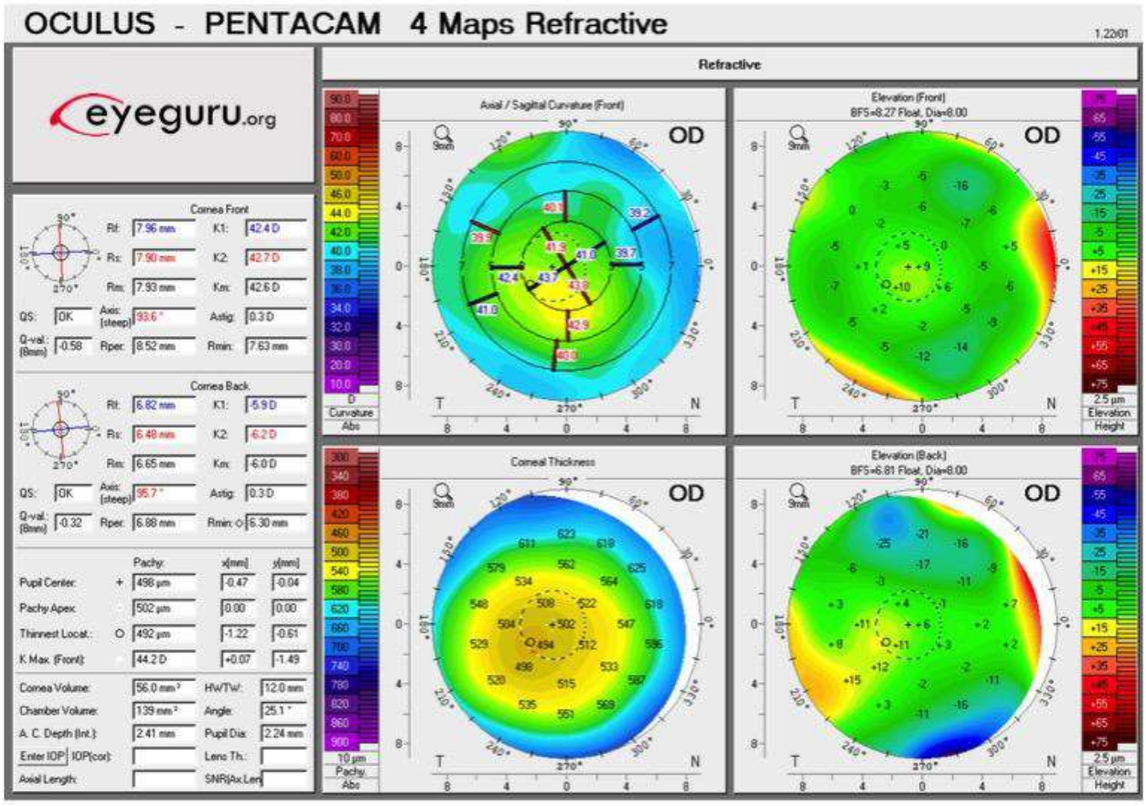
**Review Questions**

***1) Based on this patient’s topography, are they a candidate for Toric IOL?***



***Answer***: **No**, the topography shows irregular astigmatism. Only patients with **regular astigmatism** are **good candidates** for **Toric IOLs**.

***2) Based on this patient's topography post-LASIK, what refractive error did they have prior to LASIK?***




***Answer:*** This post-LASIK topography demonstrates central corneal steepening ("steeping warm tea" reminds us that warmer colors correspond with steeper corneal curvature). Therefore, the patient was being corrected for **hyperopia.**