PERIPHERAL RETINA AND VITREOUS

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ORIENTATION

In order to examine and document findings of the peripheral retina, a method of orientation and representation of the fundus as a whole is necessary. The fundus is traditionally depicted on a circular flat surface that is divided into 3 sections that are based on several anatomical structures used to demarcate certain regions:

1 **Posterior Pole**: area delimited by the ONH and the major superior and inferior vascular arcades (~6mm)
2 **Mid - Periphery**: area between superior and inferior vascular arcades and the equator
3 **Periphery**: area from the equator to and slightly beyond the ora serrata (~ 5mm)

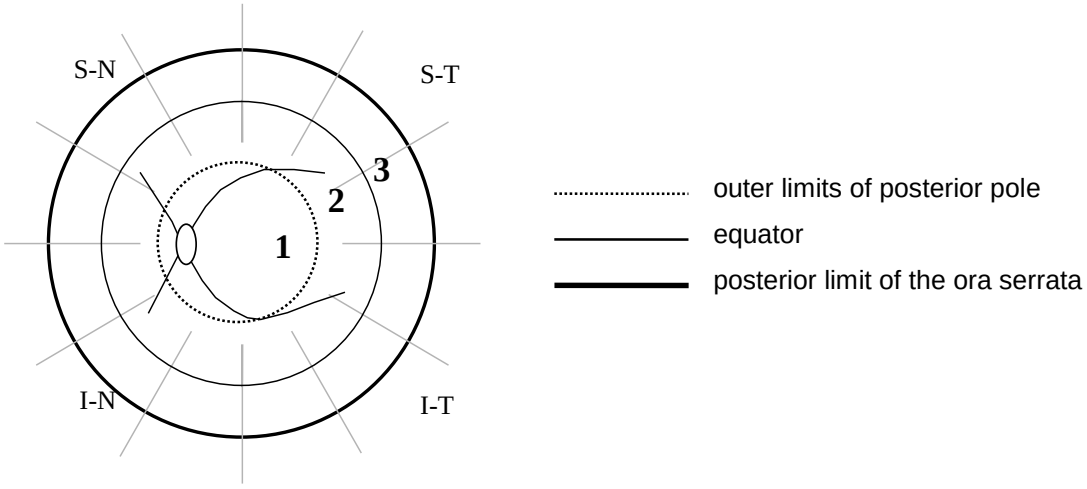

Figure 1: *A method of orientation and representation of the fundus*

By illustrating the **spherical eye** on a **flat surface**, certain distortions are created. The disc and macula are relatively minified while the equator, peripheral retina and ora are magnified. Therefore, on the diagram, the ora appears larger than the equator but is not so in reality. The equator is the largest circumference of the eye.

For location purposes, the eye is divided further into **4 quadrants** centered on the fovea. One can also refer to an **imaginary clock face** centered on the fovea with the 12 o'clock position superiorly. The terms **inner and outer** are used to denote location with respect to the center of the globe. Anything closer to the center of the globe is "inner" compared to anything further from it. Finally, the **Disc Diameter** unit (1DD ~ 1.5mm) is used to indicate the size and location of lesions with respect to the optic disc.

ANATOMICAL LANDMARKS

VORTEX VEIN AMPULLAE

The Vortex Vein Ampullae are collecting channels for the multiple, thin, curved venous tributaries that cover most of the fundus. They are usually four (1 per quadrant) but can be fewer or as many as 10-15. They are located around the equator and mark the **equatorial** region but they are not always visible. They are red-orange, **octopus-shaped** and are commonly found with significant **surrounding pigmentation** (RPE hyperplasia). The vortex ampullae drain into vortex veins going through the sclera posteriorly.

LONG POSTERIOR CILIARY ARTERIES AND NERVES (LPCA & LPCN)

The LPCA and LPCN appear as two straight lines, typically white to yellowish in color, with pigmented borders. They run in the suprachoroidal space stretching from the **ora to the equator** at the 3 and 9 o'clock positions and **divide the retina** into superior and inferior halves. The artery usually runs below the nerve temporally and above the nerve nasally. The nerves are usually more visible than the arteries.

SHORT POSTERIOR CILIARY ARTERIES AND NERVES (SPCA & SPCN)

The SPCA and SPCN are short, straight, white to yellowish lines with pigmented borders found in the peripheral retina. They are 10 to 20 in number but usually only 4 to 8 are visible. They extend from the mid-periphery to the periphery usually congregating near the vertical meridian but they can be scattered anywhere. They are not as consistently visible as the LPCA and LPCN.

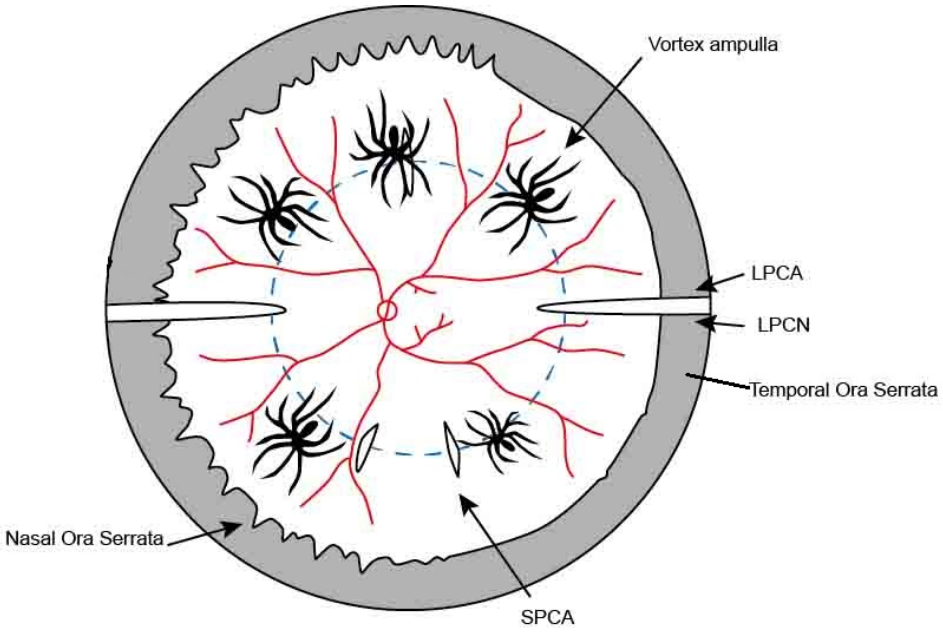

Figure 2: *Short posterior ciliary arteries and nerves*

PERIPHERAL VESSELS

The peripheral arteries and veins run parallel to the ora serrata and come to about 1.5 mm from the ora serrata. As a result, a 1.5 mm capillary-free band exists posterior to the edge of the ora serrata.

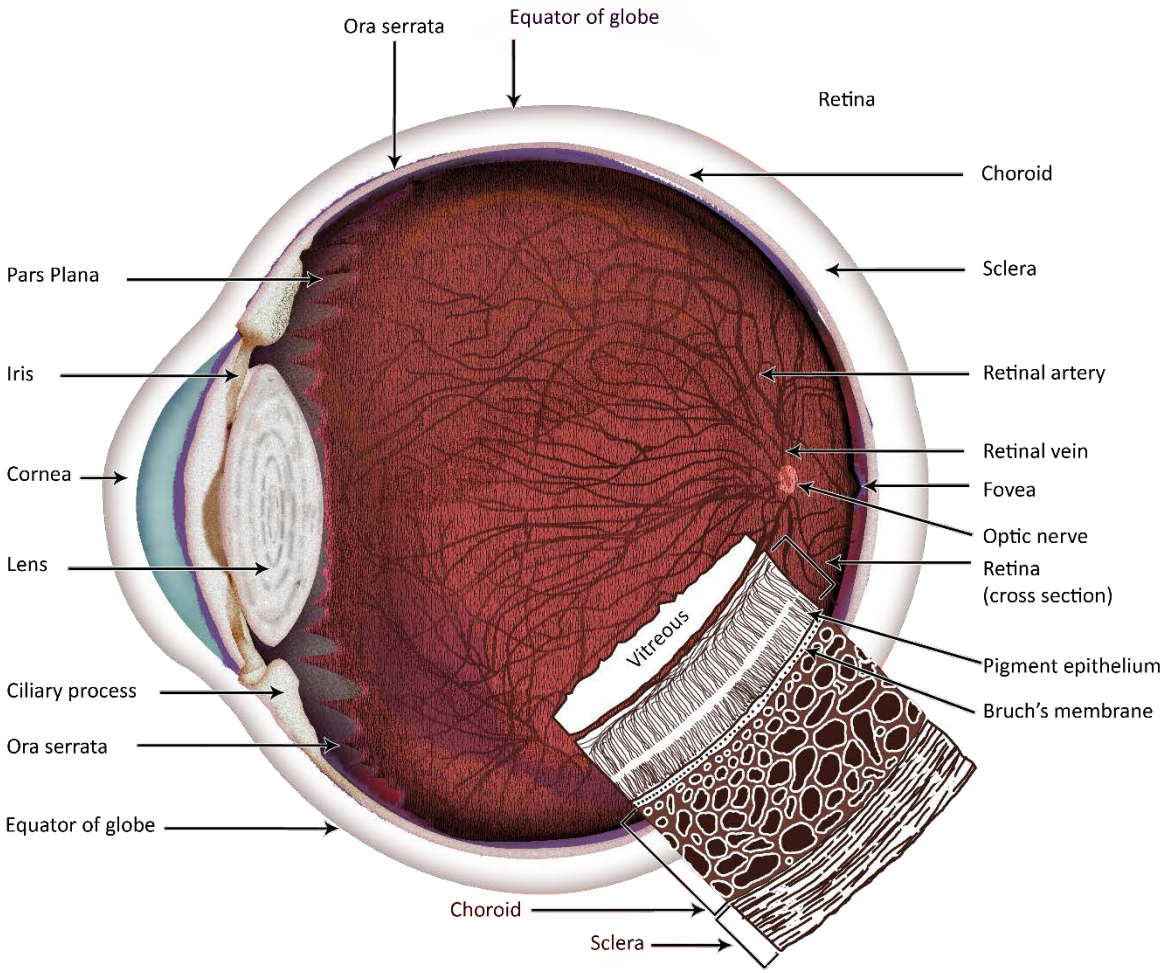

Figure 3: *Peripheral vessels*

ORA SERRATA

The ora serrata represents the anterior limit of the neural "seeing" retina. It is a 360° junctional band that is more narrow temporally (~1 mm) than nasally (~2 mm). It is **scalloped** (nasally > temporally) with 20 to 30 **dentate processes** per eye. The brown rounded areas that extend posteriorly from the ora are called **Oral Bays** while the whitish anterior retinal extensions into the bays are called **Oral Teeth.**

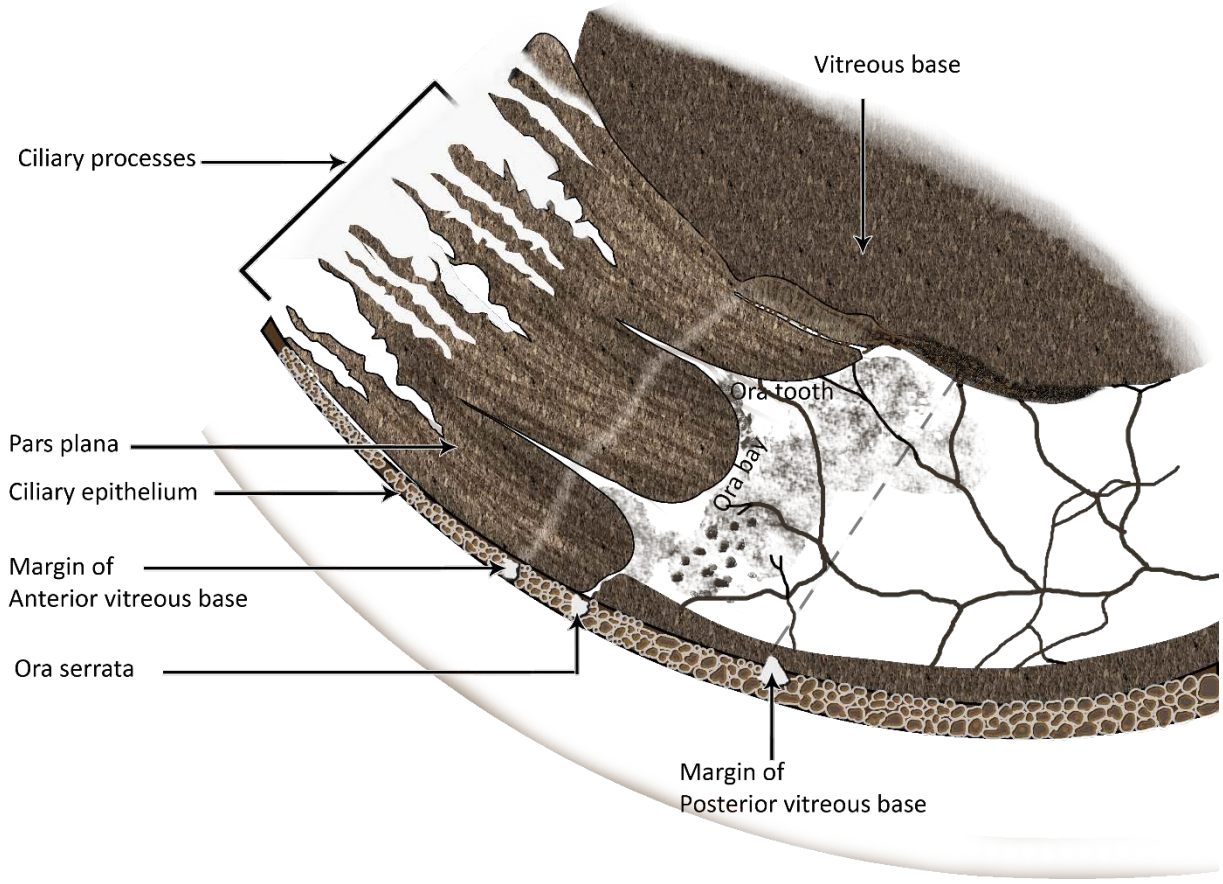

Figure 4: *Anatomy of the ora serrata*

PARS PLANA

The Pars Plana is a 4-5 mm region that extends from the ora serrata to the ciliary processes (60-70 / eye). It is composed of an inner non-pigmented epithelium and an outer pigmented epithelium. The Pars Plana is chocolate color. The ciliary processes are also brown in reality but appear **cream color** during ophthalmoscopy because of the tangential illumination used to see them.

The vitreous is not truly a major anatomic landmark since it is usually invisible but is included here because of the prominent role it plays in the appearance and development of peripheral findings. One must understand the close vitreoretinal relationships that exists throughout the fundus which may be altered during the development of peripheral lesions or which may themselves cause the formation of peripheral retinal anomalies.

The vitreous is a type of gel, semi-solid to liquid in consistency, made up of 99% water, 1% hyaluronic acid, to allow maximum light transmission. The hyaluronic acid provides elasticity and a viscous quality. The vitreous also contains type II collagen which supports its shape. It occupies 67-75% of the ocular volume. It is clear and usually retains its clarity through most of a person's life. Vitreous strands and floaters, which are debris of vitreous material, are relatively common and may be seen with the ophthalmoscope.

Asteroid hyalosis occurs in the vitreous of 0.05 to 0.5% of the population. It is made up of white or yellow-white calcium-laden lipids that usually occur unilaterally. The opacities can be annoying to the patient, although vision is usually not severely affected. Synchysis scintillans has a similar appearance, though the crystals are made of cholesterol rather than calcium. It usually occurs bilaterally in young patients though is found more rarely than asteroid hyalosis, sometimes occurring secondary to ocular disease or trauma.

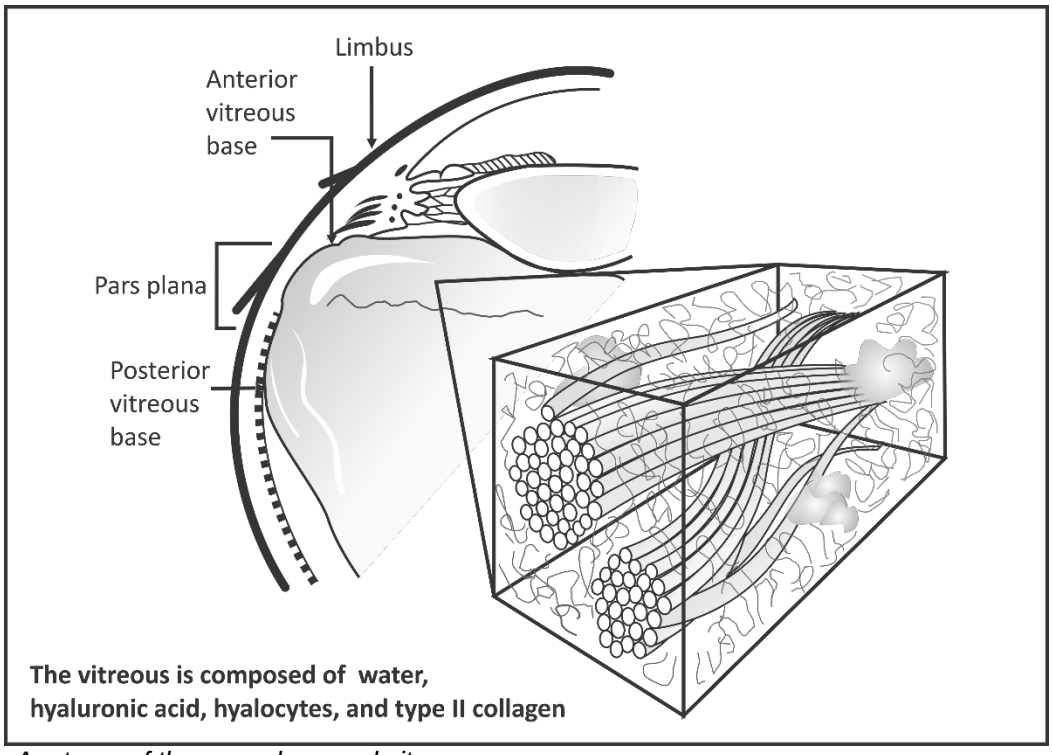

Figure 5: *Anatomy of the pars plana and vitreous*

The vitreous is attached at several locations in the eye. It is attached **strongly** at the **posterior lens surface** in young persons, a fact that renders the extraction of cataracts difficult in the young. It is attached at the **vitreous base** with a **strong 2-4 mm** connection that straddles the ora serrata and holds the vitreous cortex, sensory retina and pars plana together. It is attached at the **ONH margin** with a **strong ring-form** attachment that is observable as the "Weiss ring" following a PVD. Finally it is **weakly** attached at the **macula and peripheral retinal vessels**.

VITREOUS BASE (VB)

The vitreous base, while usually not visible, is also considered a landmark. The VB is a 2-4 mm band that straddles the ora and represents the limit between anterior and posterior vitreous cortex. The anterior limit is sometimes visible as a whitish linear haze on and parallel to the pars plana. The posterior limit is usually invisible unless it protrudes or is altered by severe vitreous traction in which case it is called a **prominent VB**. A prominent VB looks like a thin elevated white line parallel to the ora. The VB is the most adherent vitreal attachment. Its limits can sometimes be denoted by the increased pigmentation that results from RPE hyperplasia at that location.

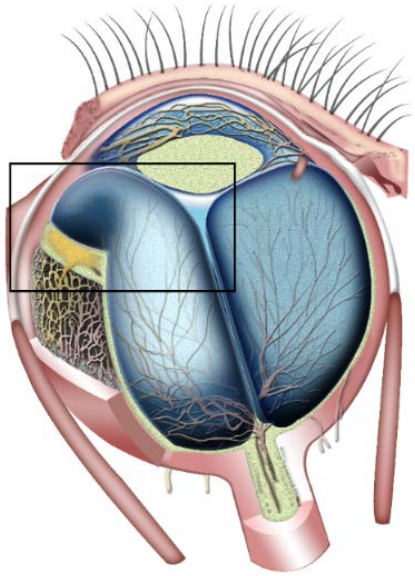

Figure 6: *Location of the vitreous base*

DIMENSIONS OF EYE

Length (antero-posterior)	~ 22mm
Ora to ora	~ 45mm
Limbus to posterior ora limit	~ 7 mm
Ora to equator	~ 7 mm
Corneal diameter	~ 12mm
Circumference	~ 71mm

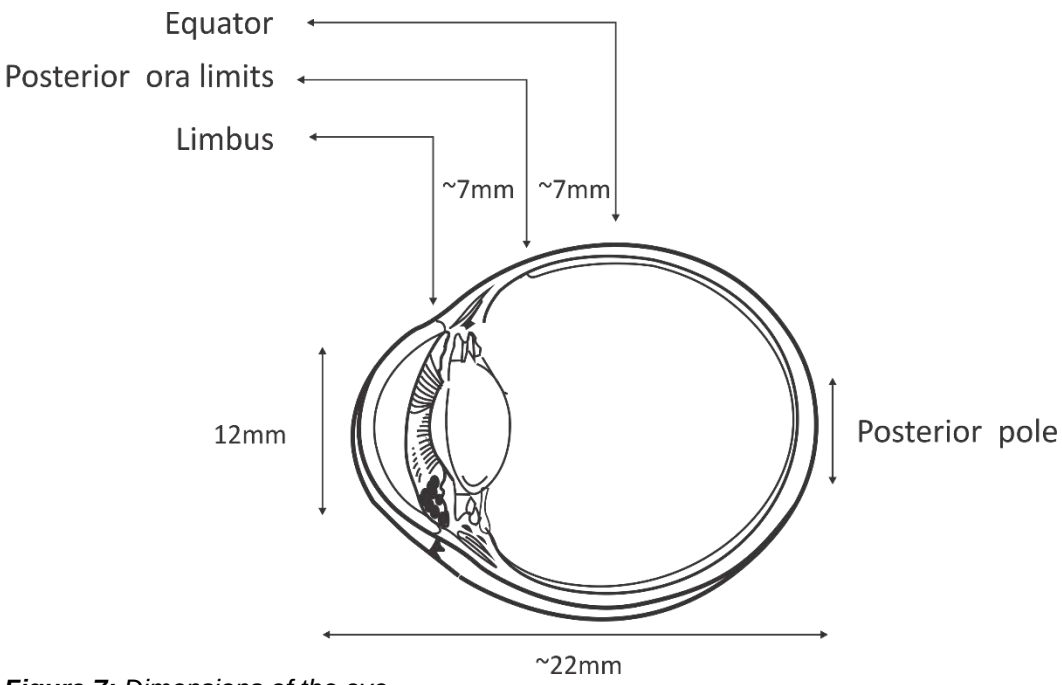

Figure 7: *Dimensions of the eye*

DEVELOPMENTAL ANOMALIES

ENCLOSED ORAL BAY

S: Asymptomatic

O: Brownish segment from the ora serrata that is completely or partially surrounded by dentate processes or sensory retina.

Prevalence	6%
Bilaterality	8%
Preferred location	S-T
Risk of retinal break	15-20%
Risk of RD	rare
Associations	(-)

A: DDx: Atrophic retinal hole; R/O break or RD

P: No treatment; Educate patient of Si/Sx of RD; Monitor annually

MERIDIONAL FOLDS

S: Asymptomatic

O: Meridional folds are **radial elevated** folds (1/2 - 4 DD in length) of degenerated superfluous retina and proliferated glial cells that develop as a result of vitreous traction. **Perpendicular** to the ora, they are located in the oral area usually within a tooth but occasionally at the end of a bay. Gray-white in color, the folds often contain cystoid degeneration. Small retinal breaks may be found at the posterior end or along the lateral margins. Since these folds are altered retinal tissue composed of a proliferation of glial cells, the possibility of RD is present. They may increase with age.

Prevalence	25%
Bilaterality	50%
Preferred location	SN
Risk of retinal break	present
Risk of RD	present
Associations	(-)

A: R/O breaks or RD

P: No treatment
Educate patient of Si/Sx of RB and RD
Monitor annually

ORAL PEARL

S: Asymptomatic

O: Single, smooth, glistening white spheres near the tip of a dentate process that represent the histopathologic equivalent of posterior pole drusen. They may be pigmented.

Prevalence	20%
Bilaterality	+++ (⊂ symmetry)
Preferred location	S-T
Risk of retinal break	(-)
Risk of RD	(-)
Associations	(-)

A: Oral Pearl

P: No treatment, benign

PARS PLANA CYST

S: Asymptomatic

O: Pars plana cysts are clear, bullous, blister-like elevations (<3DD) that represent a separation between the non-pigmented and pigmented ciliary epithelium. They represent somewhat the histopathologic equivalent of a sensory retinal detachment. Most are acquired and are either idiopathic or possibly associated to ocular diseases such as posterior uveitis or RD.

Prevalence	3-18% (↑ ⊂ age)
Bilaterality	33%
Preferred location	temporal
Risk of retinal break	(-)
Risk of RD	(-)
Associations	Ocular diseases?

A: Pars Plana Cyst

P: No treatment, benign

CONGENITAL HYPERTROPHY OF THE RPE (CHRPE) (A.K.A. HALO NEVUS)

S: Asymptomatic

O: CHRPE are brown to black flat lesions that are one to several DD in size, usually round and most often found in the peripheral retina. They have well defined margins sometimes with a distinctive depigmented halo that is **pathognomonic** of the lesion. Chorioretinal atrophy, in the form of punched-out holes through which the choroid is visible, frequently develops in the lesion producing **lacunae** and gives the area a **"Swiss cheese"** appearance.

CHRPE is the result of a congenital **increase in size** of the RPE cells. Because the larger cells contain more melanin granules, the involved area is darker. The halo is produced by adjacent pigment epithelial cells which are almost devoid of melanin granules. These lesions are benign and stable, except for slight enlargement over time and the development of lacunae.

Grouped pigmentation such **as "Bear Tracks"** is a variation of CHRPE which presents like a series of animal-like footprints on the retina. The "footprints" are grouped in clusters and extend usually in a **wedge shape** from the posterior pole into the periphery.

An association between CHRPE and Familial Adenomatous Polyposis (FAP) is reported. FAP is an AD hereditary disorder where polyps develop in the large intestine and tend to become malignant (50% of

cases). Although the relation is uncertain, patients with four or more lesions in both eyes and a familial Hx of FAP should have periodic colorectal evaluations to R/O malignancy.

Prevalence	??
Bilaterality	15%
Preferred location	none
Risk of retinal break	(-)
Risk of RD	(-)
Associations	FAP; relative scotoma

A: DDX: choroidal nevi, melanoma, RPE hyperplasia, chorioretinal scars; Assess possibility of FAP association

P: No treatment, benign; R/O FAP if necessary

PERIPHERAL DEGENERATIONS

CYSTOID DEGENERATION

S: Asymptomatic

O: Cystoid degeneration is area of thickened retina that usually extends about 1/2 DD from the ora. Hazy gray in color with enclosed hazy red dots, it grows slowly in a posterior direction. Cystoid degeneration involves **vacuolar** changes (small bubbles) in the **outer plexiform layer** of the retina. These may coalesce and form larger cystic spaces and evolve into retinal schisis with inner layer holes. The cystoid can progress posteriorly and extend to the equator.

The inner surface of cystoid can “burst” to produce a “pseudo-hole” or inner layer hole. These require no treatment since they do not allow liquid to sip under the retina and hence do not produce RD. More rarely, both inner and outer layer holes occur and treatment may be warranted.

Prevalence	**100% by age 8!**
Bilaterality	100%
Preferred location	T > N; S > I
Risk of retinal break	rare hole (inner > outer)
Risk of RD	(-)
Associations	retinoschisis

A: R/O retinoschisis and rare inner and outer holes

P: No treatment; Monitor annually

PERIPHERAL RETICULAR PIGMENTARY DEGENERATION

(a.k.a. Peripheral Senile Degeneration and Peripheral Tapetochoroidal Degeneration)

S: Asymptomatic

O: Peripheral reticular pigmentary degeneration presents as areas of hypo- and hyper-pigmentation in a circumferential band. The affected area takes on a granular appearance with the pigment laid down in a **typical** sprinkled pattern or in a criss-crossing **reticular or bone-spicule** pattern.

Peripheral senile pigmentary degeneration results from the breakdown of RPE cells which probably results from the loss of perfusion of the peripheral choriocapillaris. The degenerating RPE releases melanin granules which are dispersed into the overlying sensory retina or carried by macrophages toward retinal venules causing **pigment cuffing.**

Prevalence	20% > 40 y/o (↑ ⊂ age)
Bilaterality	100%
Preferred location	Nasal
Risk of retinal break	(-)
Risk of RD	(-)
Associations	age

A: DDx: Retinitis Pigmentosa

P: No treatment, benign

WHITE WITHOUT PRESSURE (WSP) / WHITE WITH PRESSURE (WCP)

S: Asymptomatic

O: WsP is a fairly common retinal finding that causes the retinal surface to have a thin translucent gray-whitish appearance. The gray often fades as it approaches the ora anteriorly. WsP is bounded posteriorly by a discrete border that often appears like a narrow zone of **optically darker retina**. Retinal vessels stand out against the whitened retina but choroidal details are not visible.

Anywhere in the periphery between the ora and equator, it can appear in patches or as a 360° band. Islands of normal retina can be found within large areas of WsP. WsP appears to be **migratory** in nature and its appearance and location can change between examinations. Scleral depression enhances the whitish appearance of the entity and the posterior margins.

The exact cause of WsP is not known but it appears to be related to peripheral vitreous traction and an **unusual vitreoretinal relationship** that may cause disorganization of the ILM, NFL, retinal neural elements or even RPE. Alternatively, it may represent intraretinal oedema that results from the prolonged vitreoretinal insult.

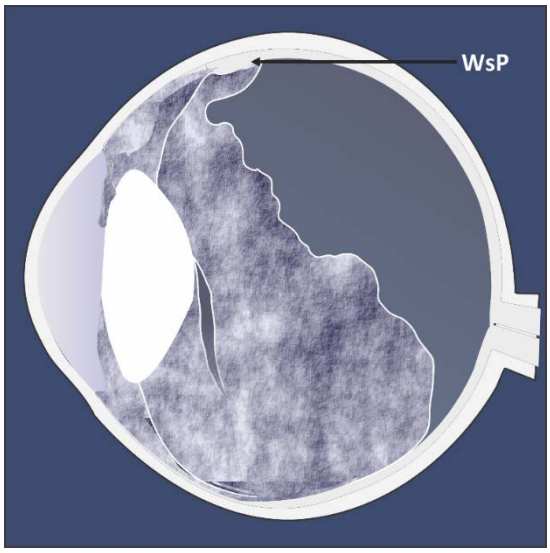

Figure 8a: *White without pressure*

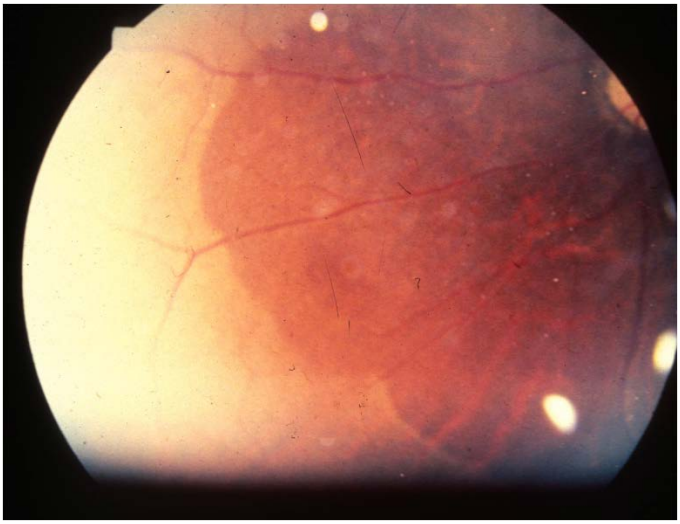

Figure 8b: *White without pressure*

WcP is a similar optical phenomenon to WsP with the same possible causes and characteristics except that the retina appears whitened like WsP upon scleral indentation otherwise it appears normal without pressure. WcP is fairly common (32% of eyes) and it is totally benign.

Prevalence	30% (↑⊂ age); 10 x ↑ in Blacks: ↑ in myopes
Bilaterality	+++
Preferred location	ST
Risk of retinal break	15-20%
Risk of RD	rare
Associations	lattice, staphyloma, vitreous degeneration, PVD, RS

A: DDx: RD, RS *with scleral indentation!*

P: No treatment; monitor annually for rare associated breaks
Monitor bi-annually if WsP is with irregular scalloped borders, elevated tractional membranes, progressive vitreous degeneration or near a lattice degeneration.

PERIPHERAL DRUSEN

S: Asymptomatic

O: Peripheral drusen are discrete, small, round, evenly spaced, white-yellow crystalline spots that can appear in variable pattern and in any quadrant of the peripheral retina. Peripheral drusen are less common than posterior pole drusen and may be found as an isolated finding. Peripheral drusen represent hyaline thickening of Bruch's membrane but unlike posterior pole drusen, they are rarely associated with complications (e.g. SRNV, haemorrhages or leakage). Vision is unaffected and all visual functions and electrophysiology tests remain intact. It is uncertain if peripheral drusen is an age-related anomaly or an AD hereditary disease.
When drusen present around the equator, the condition is called "equatorial drusen".

Prevalence	???
Bilaterality	100%
Preferred location	variable
Risk of retinal break	(-)
Risk of RD	(-)
Associations	rare SRNV

A: Recognize, R/O other “yellow-spot” conditions of the fundus

P: No treatment.

CHORIORETINAL ATROPHY

(a.k.a.: Pavingstone or Cobblestone Degeneration)

S: Asymptomatic

O: Discrete, depressed, rounded depigmented foci of **retinal and choroidal thinning** between the ora and the equator. The **“punched-out”** lesions can be 0.1 mm to several DD in size. They appear yellow-white due to the sclera being partially visible and have pigmented edges. Adjacent lesions can coalesce and form larger lesions with scalloped margins. When the lesions are found in a chain-like fashion in the peripheral retina, it is known as *pavingstone or cobblestone* degeneration.

Chorioretinal atrophy is thought to be due to vascular occlusion of compartments of the choriocapillaris which results in the degeneration of the overlying five layers of the sensory retina. The inner retinal layers remain intact since they are not supplied by choroidal blood flow. Due to the loss of both the choriocapillaris and the pigmented epithelium, the remaining semi-transparent inner layers of the retina allow a "window-like" view of the underlying choroidal vessels and the sclera. Because the inner retinal layers are intact, there is no risk of RD since holes do not form.

Prevalence	27% (↑⊂ age)
Bilaterality	33%
Preferred location	**80% I-T or 5-7 o’clock**
Risk of retinal break	(-)
Risk of RD	(-)
Associations	(-)

A: DDx: retinal holes, lattice, chorioretinal scars

P: No treatment, benign

VITREORETINAL TUFTS

(a.k.a. granular tufts of pigment clumps)

S: Usually asymptomatic; Photopsia may be reported with eye movement

O: A retinal tuft is produced by a small amount of grayish-white irregular pieces of proliferated glial tissue in the vitreoretinal interface. The lesions are found between the ora and equator and usually appear elevated. VR tufts can be non-cystic, cystic, or zonular traction tufts.

The **non-cystic type** is irregularly shaped with pointed projections on its surface. It is small with a base of < 0.1mm and usually occurs in clusters which break off spontaneously later in life to appear as small fragments in the vitreous.

The **cystic type** is composed of tiny cystic spaces that result from retinal degeneration. It appears as a sharply circumscribed, opaque gray-white irregular clump with a “honeycomb” appearance due to the microcystic spaces within it. It is firmly attached to the vitreous and it may break off spontaneously or with a PVD and float in the vitreous. It is commonly associated with retinal breaks, either flap tears or operculated holes and **accounts for 7% of primary RD**.

The **zonular traction type** appears like anterior projections of retina attached to one or more thickened **zonular fibers**. It is distinguished from other tufts by the larger size, closeness to the ora, a triangular base

that thins to a pointy apex and its anterior angulation. Zonular tufts are associated with a high incidence of retinal breaks but a low incidence of RD since they are located in the vitreous base.

VR tufts are areas of **stronger adhesion** between the vitreous and retina and consequently of higher traction. They are usually associated to **RPE hyperplasia** that is probably secondary to the retinal insult that they cause. Because of the increased traction, these areas are closely associated with retinal tear and retinal detachment formation especially after a recent PVD. A VR tuft can frequently be found on the flap or the operculum of a retinal tear.

Scleral depression can enhance the vitreous condensation over these lesions.

	Non-Cystic	Cystic	Zonular
Prevalence	72 %	5-7 %	15%
Bilaterality	50 %	6 %	15 %
Preferred location	VB	(-)	Nasal; Ora
Risk of retinal break	present	present	present
Risk of RD	present	1%	present
Associations	(-)	(-)	(-)

A: Assess retinal traction using scleral depression; R/O associated retinal breaks.

P: No treatment
Educate on Si/Sx of RD
Monitor annually
Monitor more frequently if symptoms or high amount of traction are present
Monitor more closely following a PVD

ACQUIRED RETINOSCHISIS (RS)

(a.k.a. Senile or Adult Retinoschisis)

S: Asymptomatic until advanced
Possible photopsia and VF defects
Decrease in VA if macula is involved (rare)

O: Acquired RS is caused by retinal degeneration which results in the separation of the sensory retina at the **outer plexiform layer and inner nuclear layers**. It may represent a progression of cystoid degeneration where the cystic spaces expand into schisis cavities. In fact, cystoid is <u>always</u> present between the schisis and ora and it is difficult to say where cystoid ends and schisis begins. The resultant cavity can take two forms:

The typical or flat RS - this represents an advanced form of cystoid degeneration. The cavity remains small, minimally elevated, acquires no holes and remains confined to the areas anterior to the equator.

The bullous or reticular form - the cavity fills up with a viscous fluid and as the cavity enlarges, the lesion balloons forward and elevates like a dome into the vitreous. The RS appears thin, taut, smooth and does not undulate on eye movements.

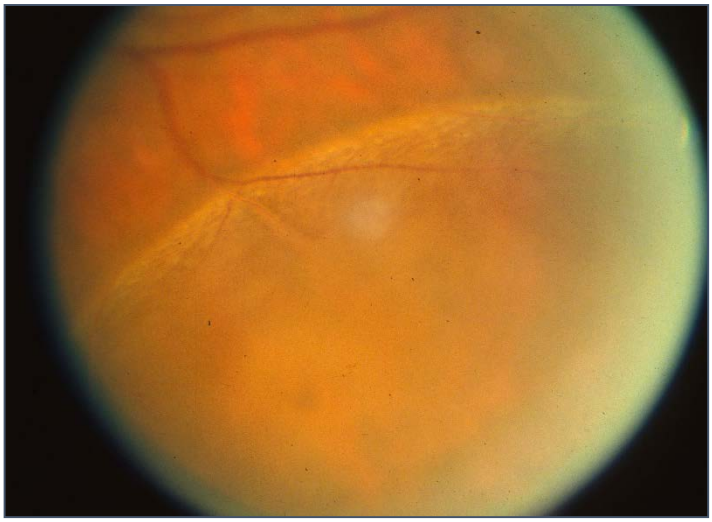

Figure 9: *Retinoschisis*

The layers of the schisis take on a **honeycomb** appearance. The inner layer can be transparent or translucent and underlying choroidal details are dimmed. Sometimes it is possible to see the **shadow** from a retinal vessel in the inner layer on the underlying RPE and choroid. The retinal vessels can separate with the inner or the outer layer or they can be shared between both layers. Sheathed, sclerotic or occluded vessels may appear on the surface of the RS. Vitreal condensations, or remnant of glial tissues from the inner retinal layers, may be seen on the RS inner layer as white or yellowish **snowflakes.**

Multiple small holes may develop in RS especially in the reticular type. Inner layer holes are small and of little consequence since the fluid from the vitreous cavity will just enter the cystic space. Outer layer breaks are larger and may have rolled up edges. They may have a small surrounding detachment caused by the viscous fluid from the cavity, but alone, with limited amount of fluid available, they tend to remain stationary and not cause complications. **Coexisting inner and outer layer breaks** in a RS can allow fluid from the vitreous to pass to the subretinal space and produce an RD. A PVD must also be present to provide the fluid necessary to produce the RD.

The presence of a white or **pigmented demarcation** line is often visible at the posterior margin of the schisis. When pigmented, it usually indicates a stationary (**>3 months**) detachment of the ocular schisis layer.

Most RS become stationary spontaneously or progress very slowly posteriorly in a circular fashion. Progression may be related to vitreous traction and it may be halted with PVD and the relief of traction. Less than 10% show progression, expansion, or development of retinal break or detachment outside the RS. 70%, however, reach post-equatorial borders.

Prevalence	4% ; (↑ ⊂ age)
Bilaterality	38-80%; ⊂ symmetry
Preferred location	**70% I-T**
Risk of retinal break	28% of RS 23% in outer layer 3% in inner layer 2-10% in both layers
Risk of RD	6% of RS
Associations	**Absolute scotoma** Vitreous degeneration (60%)

A: Scleral depression to enhance view of breaks
DDx: RD - based on several findings as shown below:

	Retinoschisis	**Retinal Detachment**
Motion	No	Yes
Scotoma	Absolute	Relative
Surface	Smooth	Corrugated
Choroidal detail visibility	Faint	No
Pigmented vitreous floaters	No	Yes
Sheated vessels	Yes	No
Snowflakes/cystoid	Yes	No
Bilaterality	70%	No
Symptoms	No	(+/-)
Demarcation	No (+/-)	Yes

P: If no breaks are present or
If only inner layer breaks anterior to equator exist,

No treatment; Monitor bi-annually with drawing or pictures
Educate on Si/Sx of RD.

If outer layer holes only exist,

Consider a retinal consult or manage as atrophic retinal holes (see later)

If RS is progressing beyond equator or
If both inner and outer layer breaks exist or
If traction or an RD are present,

Obtain a retinal consult.

LATTICE DEGENERATION

(a.k.a. Palisade, Equatorial & Snail Track degeneration)

S: Usually asymptomatic; flashes or floaters may be reported with eye movement

O: Lattice degeneration is a sharply demarcated, oval or elliptical peripheral lesion. Lattice can be singular or multiple in number, usually 1-4 DD in length and 1.5-2 DD in width with the long axis **parallel to the ora serrata**. Posterior lesions tend be wider and are usually radially or obliquely oriented. Lattice is frequently

associated with WsP along its borders and 80% show **RPE hyperplasia** giving it a mottled look. Most lesions have small white-yellow flakes on the surface, giving lattice a granular appearance. The base is "jagged" and of uneven thickness, giving the lesion a "moth-eaten" appearance. The name "lattice" refers to the appearance produced by attenuated or sheathed vessels within it.

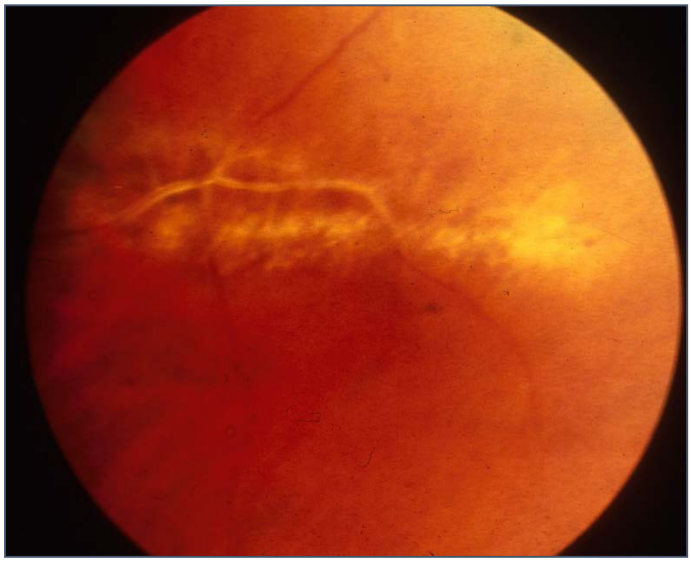

Figure 10: *Lattice degeneration*

Lattice usually appears in the **2nd decade, developing** in 3 slowly progressive stages:
1. Lattice begins with a grayish granular appearance of retinal thinning often around a vessel.
2. The vessel then sheaths (hyalinizes) but remains patent giving it a sclerotic appearance and associated RPE hyperplasia forms.
3. Finally larger vessels are affected, the lesion enlarges and the pigmentation increases. Holes can develop at this stage.

Lattice is caused by vitreoretinal degenerative process characterized by **retinal thinning** and abnormal pockets of liquefied vitreous **(lacunae)** in the adjacent vitreous. Progressive retinal thinning and VR changes produce characteristic atrophic holes within lattice in 18-42% of lesions. These are small and round, darker than the surrounding whitened lattice and can be partial or full thickness. The holes within lattice have no traction. Without disturbance of the vitreous, lattice with holes typically remains quiet for years with a low 3-14% rate of RD.

The VR changes can also result in operculated, linear and flap tears, which can lead to RD. The VR changes produce vitreal condensations with very **strong adhesions** at the edges of the lesions. A PVD, ocular surgery or other insult to the vitreous can produce traction at the sites of strong VR adhesions and result in retinal breaks. Lattice with traction tears at the margins have a 35-38% risk of RD development and require prophylactic treatment.

Lattice degeneration is significant in that it is the most commonly associated retinal finding in rhegmatogenous detachments requiring surgery: **20-35% of eyes with RD have lattice.**

Snail track degeneration appears to be a variant of lattice degeneration, with all the inherent risks of lattice. Snail tracking has no pigmentary changes and appears like **the tracks of a snail as shiny silver-white broad streaks,** in the peripheral retina. Snail track lattice may be an early stage of typical lattice, but most likely it is a distinct variant of typical lattice. Scleral depression makes these lesions appear bright white.

The development of lattice is thought to be triggered by:
- relative retinal ischemia
- basic VR degenerative changes
- genetic factors.

Prevalence	6-10% (↑ ⊂ myopia)
Bilaterality	50% (⊂ symmetry)
Preferred location	I (5-7o'clock) ; S (11-1 o'clock)
Risk of retinal break	16-25% (1-2% tears; rest are holes)
Risk of RD	0.1-0.5%.
Associations	WsP, WcP, Chorioretinal atrophy

A: R/O breaks and significant traction using scleral indentation
Manage lattice patients weighing **RD Risk Factors** (see below)

P: If no symptoms:

Monitor annually
Educate on Si/Sx of RD

If symptoms of flashes or floaters or
If lattice has atrophic holes, is asymptomatic and has no RD risk factors

Monitor bi-annually
Educate on Si/Sx of RD

If RD risk factors are present

Consider a retinal consult or F/U at a closer interval

If marginal breaks are present (28-35% chance of RD)

Obtain a retinal consult

RETINAL BREAKS (RB)

GENERALITIES

RB are defined as a loss of retinal tissue in the form of **atrophic holes or tears**. Retinal holes can be partial (a.k.a. excavations, erosions or pits) or full thickness. Retinal tears can be of the **operculated, horseshoe or "dialysis**" type.

RB occur in 4-18% of the population. About 75-80 % of these are atrophic retinal holes, 10-15% are operculated breaks, and 10% are flap tears.

RB occur mostly in the periphery probably because of the thinner retina, relative vascular insufficiency and strong VR adhesions existing there.

RB are **by far mostly asymptomatic.** When symptoms are present, they are in the form of transient and episodic photopsia and sudden floaters. Symptomatic RB have 30-40% chance of becoming an RD.

RB are found in 30% of eyes with non-traumatic RD, 70% of these are above 40 years of age.

ATROPHIC RETINAL HOLES

S: Usually asymptomatic; F&F may be reported with eye movement

O: Atrophic retinal holes are small (< 2DD) dark red areas of retinal thinning and atrophy in the peripheral retina. Holes may be **partial** (a.k.a. retinal excavations, erosions or pits) **or full thickness**. Full thickness holes allow the choroid to show through vividly giving it the typical dark red appearance. With indentation,

atrophic holes have a “volcano-like” or “fish-mouth” appearance. Holes are usually between the ora and equator.

Figure 11: *Atrophic retinal holes*

Atrophic retinal holes are possibly caused by localized vascular insufficiency in the retina and choriocapillaris which causes localized degenerative retinal changes. Because of the lack of traction and PVD, atrophic holes usually seal down and remain stationary with the borders pigmented and apposed to the RPE. **Pigmentation indicates that the hole has been present for at least 3 months.**

Atrophic holes can progress into a RD. This occurs when existing vitreous syneresis makes fluid available to filter under the edges of the hole and push the sensory retina forward off the RPE. Degenerative change in the RPE must also exist and affect the close apposition between the RPE and the photoreceptors to allow the edges of the hole to lift off.

Prevalence	2-5%
Bilaterality	20%
Preferred location	Temporal
Risk of RD	7%
Associations	Vasculopathies

A: Use scleral indentation to assess the size of retinal fluid cuff.
Assess associated RD risk factors to opt for prophylactic treatment or monitoring.

P: If asymptomatic and fluid cuff is small **(<1DD)**

Monitor annually (or bi-annually if no RPE hyperplasia is formed yet)
Educate on Si/Sx of RD.

If a “gray zone” fluid cuff **(1-2 DD)** is present

Consider **RD Risk Factors**
Monitor at closer intervals or consider a retinal consult

If fluid cuff (>**2DD)** is present

Obtain a retinal consult

OPERCULATED RETINAL TEARS

S: Usually asymptomatic; flashes or floaters may be reported when produced

O: Operculated retinal tears result from vitreous traction on a small area of retina which pulls a plug of sensory retina (**operculum**) off into the vitreous cavity. A VR tuft, which may have been the source of traction, is

often seen on the operculum which usually floats in front of the hole. Operculated tears commonly look like atrophic holes. They are round, small and red because of the visible underlying choroid and are surrounded by atrophic gray retinal tissue. Over time, the detached operculum shrinks due to the lack of nutritional blood supply and distinguishing operculated and atrophic holes becomes difficult.

An operculated tear often occurs following a PVD but it may be found in younger patients without a PVD. When an operculated tear is secondary to PVD, it is common to have a surrounding localized RD due to the large amount of fluid, available from the vitreous, seeping under the retina. Compared to flap tears, operculated tears do not frequently cause clinically significant RDs because vitreous traction is released when the operculum is formed.

Prevalence	< 1%
Bilaterality	(-)
Preferred location	Temporal
% of Breaks	10-15%
Risk of RD	low
Associations	VR changes & traction

A: Recognize operculum; assess surrounding cuff and traction; assess age of lesion; R/O RD
Use scleral indentation:

P: Manage like atrophic retinal holes

HORSESHOE RETINAL TEARS

(a.k.a. Linear or Flap Tears)

S: Most are asymptomatic
Photopsia
Sudden appearance of floaters

O: Retinal tears usually result from traction on abnormally strong vitreoretinal adhesions commonly at the posterior borders of lattice, vitreous base, retinal tufts or chorioretinal scars. The tear usually occurs **during the time of PVD**. As the PVD progresses, the torn flap assumes a horseshoe shape with the apex pointing towards the posterior pole. The anterior margins of the tear remain attached to the retina. The flap appears whitish because of oedema and lack of nutrient supply, and it shrinks over time. Vitreous heme may appear at the time of the tear due to tearing of blood vessels.

Retinal tears are usually larger than atrophic or operculated holes. They are the leading cause of rhegmatogenous RD and have a 30-35% chance of progressing into a RD. **The most important factor in RD formation is the persistent vitreous traction on the flap.**

Prevalence	0.4-2%; 10-15% of PVD
Bilaterality	
Preferred location	Superior
% of Breaks	10%
Risk of RD	30-35%
Associations	VR changes and traction

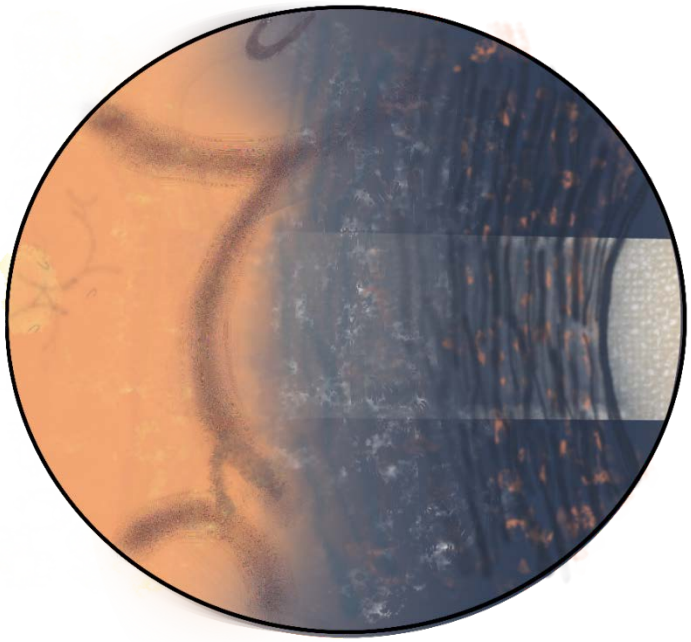

Figure 13: *Horseshoe retinal tear*

A: Use scleral depression to localize the break.

P: Obtain a retinal consult even though many are not treated!

RETINAL DIALYSIS

S: Asymptomatic (60%)

O: A retinal dialysis occurs when a tear is produced at the ora serrata pulling the retina away from the ora. Usually less than 90°, the tear takes on a crescent shape and stops at the VB.

The exact cause of retinal dialysis is questionable; it may be spontaneous or post-traumatic. The spontaneous form seems to happen more commonly bilaterally in males in the infero-temporal retina. Traumatic dialysis is usually supero-nasal and unilateral, and occurs in patients prone to trauma who present with other signs of trauma.

Retinal dialysis may not appear immediately after a trauma. **4-5 months** after it occurs, retinal dialysis **slowly, but almost definitely, progresses into RD.**

A: R/O RD;
R/O late dialysis formation by assessing post-trauma patients 60-90 days after the event

P: Obtain a retinal consult

RHEGMATOGENOUS RETINAL DETACHMENT (RRD)

S: Light flashes
Sudden onset of floaters
Shower of dust or cobwebs
Curtain (obscuration) or shadow dropping over field of vision
Decreased vision

O: A retinal detachment is produced when the sensory retina separates from the RPE. The detached sensory retina appears translucent and whitish, floats up into the vitreous cavity, develops small and large folds and **undulates** upon eye movements.

A shallow detachment does not undulate as much as a bullous RD. Retinal vessels are tortuous and appear darker on the whitened detached retina, and choroidal details are obscured. Retinal vessels may cast a shadow on the underlying choroid. RD may produce **Schaeffer's Sign or "Tobacco Dust"** when free RPE or blood cells float in the vitreous. **The prevalence of RD is 0.01% (1/10000) with a peak between 50-60 years old.**

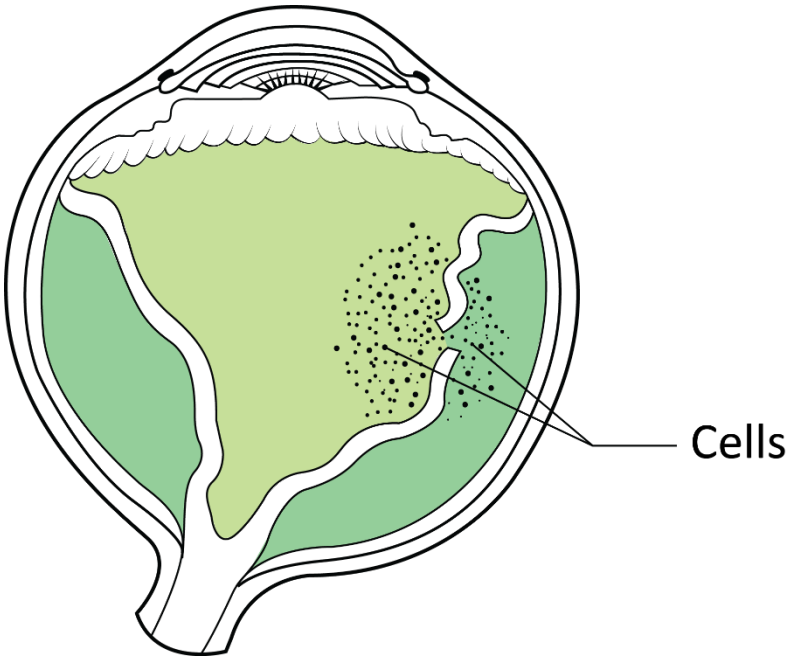

Figure 14: *Cells in the vitreous*

A **RRD** is defined as a RD that results from a **full thickness retinal break**. These occur in association with vitreoretinal changes that probably result from underlying relative vascular insufficiencies. Studies have confirmed that **vasculopaths** show a higher rate of RRD than age-matched normals.

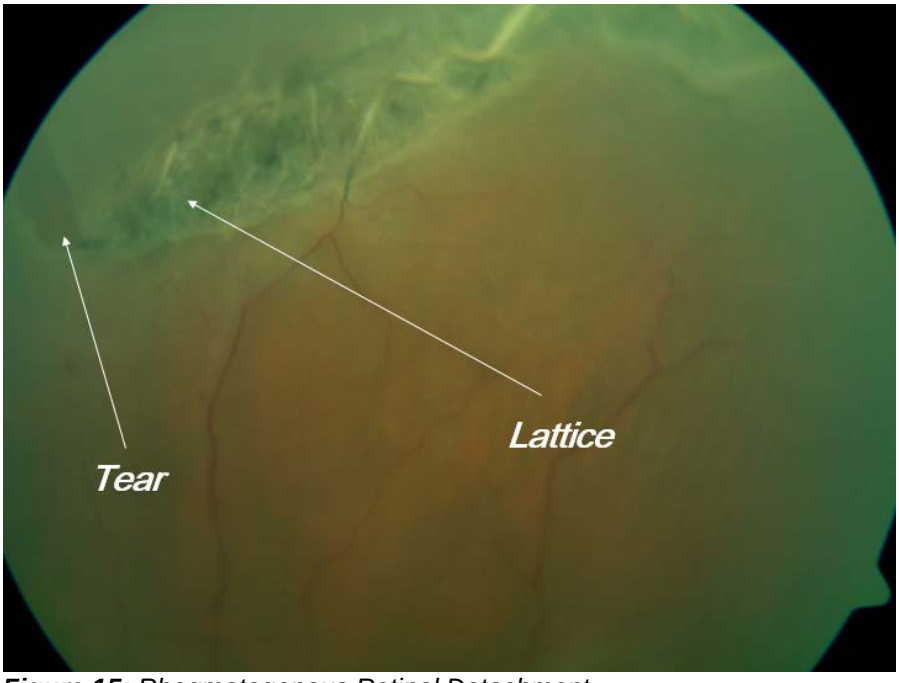

Figure 15: *Rhegmatogenous Retinal Detachment*

Several factors favor separation of the sensory retina from the RPE in a RRD:

- the bond between sensory retina and RPE is weak
- fluid from the vitreous can percolate through the RB and force the layers to dissect
- existence of VR degeneration with accompanying loss of “shock absorbing” quality
- tractional forces pulling on the sensory retina or on flap/edges of breaks
- "whip-like" action on the detached retina upon eye movements

A: RRD vs. RS vs. Tractional RD vs. Exudative RD

P: Refer immediately to a retinal specialist for prompt treatment..

RISK FACTORS

Several factors exist that raise the risk for RD development. These must be assessed and weighted for each patient in the management of retinal breaks and conditions that may progress to a RD.

POSTERIOR VITREOUS DETACHMENT & VITREORETINAL DEGENERATION

S: Flashes (25-50%), Floaters, Metamorphopsia, Blurred vision

O: A PVD results from the progressive syneresis (liquefaction) and eventual separation of the posterior cortical vitreous from the retinal surface.

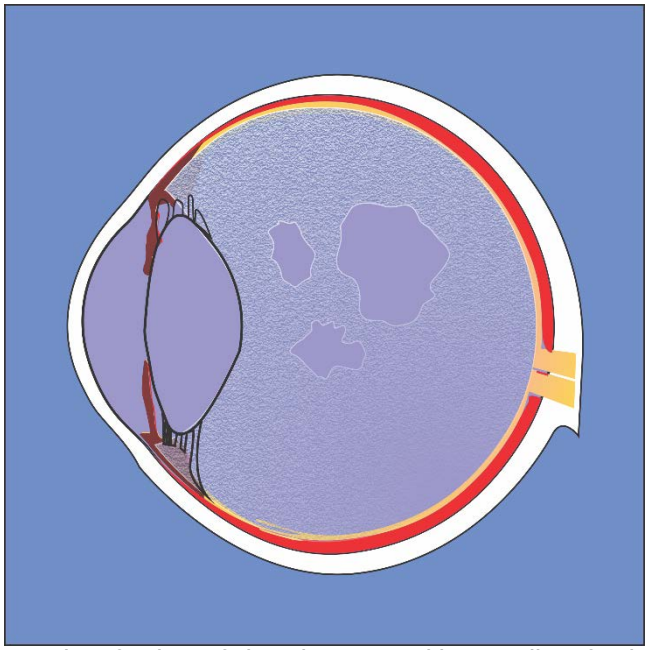

Figure 16: *Liquefaction of the vitreous. With age, liquefaction due to reduction in hyaluronic acid causes loss of support.*

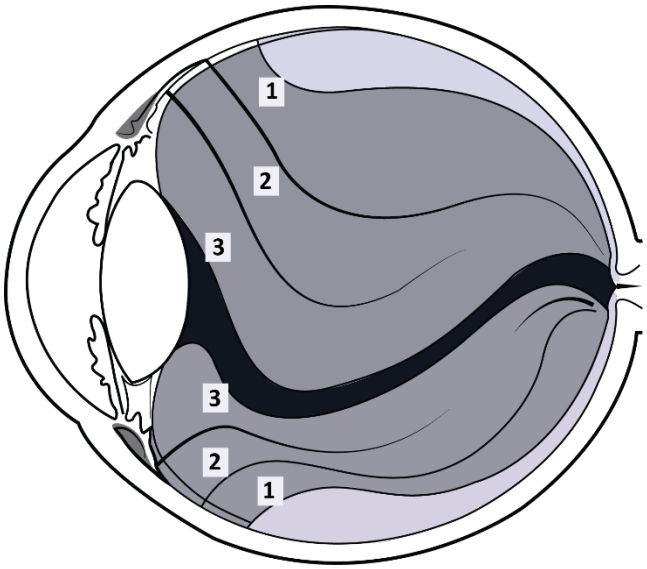

Figure 17: *Syneresis of the vitreous. Vitreous shrinkage, contraction and collapse can cause traction.*

PVD proceeds anteriorly from posterior attachments and stops at the firm posterior margins of the VB. The PVD is complete only when the vitreous detaches from its ONH attachment. Objectively, vitreous opacities are visible, but can only truly confirm a full PVD when a Weiss ring is observed. The "ring", often partial, corresponds to the circular attachment of the vitreous at the ONH.

The occurrence of PVD is of significant clinical importance since it is associated with vitreous traction and the formation of a large reservoir of fluid in the vitreous cavity, both of which are closely related with RD formation. The majority of retinal tears in people over 50 are directly related to vitreous traction. In fact, approximately 10% of PVD develop a RB. The observation of pigment in the anterior vitreous (called **Schaeffer's sign** or **Tobacco dust**) is pathognomonic of retinal breaks and should be looked for carefully during a PVD assessment.

PVDs cause photopsia due to the traction on the underlying retina. Floaters may result from the formation of condensed collapsed vitreous or the separation of the glial ring from the optic nerve. Floaters can sometimes be the result of traction on the superficial retinal vessel which can produce small vitreous bleeds. Macular holes, macular oedema, and ONH oedema may also be complications of PVD.

At 50 years old, approximately 50% of people develop a PVD. The ratio increases by about 10% for each per decade of life. PVD occurs earlier in myopes, aphakes and in patients with a history of trauma and inflammation.

A: Recognize through Si/Sx; Rule-out RB

P: Educate and monitor closely (majority of cases)

If Sx are present,

monitor every 2-4 weeks;
double the time interval as Sx decrease

If hemes are noticed,

monitor every 1-2 weeks;
double the time interval as hemes resolve

If a RB is present,

Obtain a retinal consult

MYOPIA (> 3.00D)

The precise role of myopia in RD is unclear but 1-3% of high myopes (esp. axial) develop RD. Myopia does not seem to cause specific detachment producing lesions but it is frequently associated with conditions that may predispose patients to RDs (lattice, WsP, holes, etc.). Myopia may facilitate the development of RD through the existence of a weaker photoreceptors-RPE bond. A non-complicated hole in the oral area of a myopic eye, is **35 X more likely** to produce a RD than in a non-myopic eye.

PHOTOPSIA

The presence of photopsia (flashes) signals the presence of traction on the retina. The development of RD is favored with the existence of pulling and tugging activity on the retina. Symptomatic breaks have a 30-40% chance of RD.

LOCATION, SIZE AND DEPTH OF A RB

A RB that is located superiorly is generally riskier due to gravitational forces that may force downward any liquid within the break. Logically, larger and deeper (full thickness) breaks are at a higher risk of progressing into RD.

FELLOW EYE WITH A RD

In phakic individuals, the fellow eye to an eye with a RD has an 11-31% risk of also developing a RD. In aphakic eyes, that risk is 21-36%. About 50% of fellow eyes with breaks will produce an RD, therefore all breaks in fellow eyes should be treated prophylactically.

FAMILY HX OF A RD

Although it is a weak association, a family history of RD may increase the risk of developing a RD.

APHAKIA / PSEUDOPHAKIA

Aphakia accounts for 23-40% of RD. Aphakia has a risk of RD development of 2-3% compared to 0.01% (↑ 200-300X) in the phakic population. 1.7-3% of aphakic eyes develop a RD, 50% within the 1st year post operatively. Pseudophakia also raises slightly the risk of RD, but not as significantly as aphakia does.

RACE

Interestingly, RD occurs 10 X more in White than in Black populations. This may be due to the increased rate of myopia, incidence of lattice changes and decreased pigmentation in the RPE that occurs in the White population.

OCULAR SURGERY / TRAUMA

Intraocular surgery may increase the likelihood of RD by causing a direct insult to the retina, by producing breaks, by promoting vitreal traction through the formation of vitreal membranes or by producing vitreous volume loss or shift. Pre-operative patients require a fundus investigation to R/O RB which may become a problem post-operatively. Post-op patients require regular check-ups to R/O any new breaks, assess any changes to pre-existing breaks and to R/O RD.

MIOTIC AGENTS

The use of miotic agents may increase the likelihood of RD formation. However, this is uncertain, given that in many studies showing this, no baseline fundus exam was done to rule out prior retinal breaks.

PRERETINAL MEMBRANES

The cortical vitreous may be adherent to the preretinal formation and cause massive traction at the level of the membrane.

VITREOUS HAEMORRHAGE

Following a vitreous haemorrhage, the clinician may observe vitreous liquefaction and increased VR traction through the formation of a preretinal membrane and increased retraction of an existing membrane.

CHORIORETINAL INFLAMMATION

Chorioretinitis and peripheral uveitis have effects on the vitreous that are somewhat similar to those of a vitreous haemorrhage by causing syneresis and partial liquefaction. They may additionally precipitate an exudative non-rhegmatogenous detachment.

PATIENT ACTIVITIES

The patient's activity should always be considered in the assessment of RB and RD. "Rough" activities that involve intense physical activities or contact may increase the pulling and tugging on areas of retinal traction. The development of a RB and a RD may be more likely to occur in such situations. Interestingly, 75% of traumatic RDs occur in males!

AGE

70% of eyes with non-traumatic RD occur in persons over the age of 40 with a peak between 50-60 years. Age is therefore an important determinant of the management of RB, which may require closer attention and more timely intervention in older individuals.