

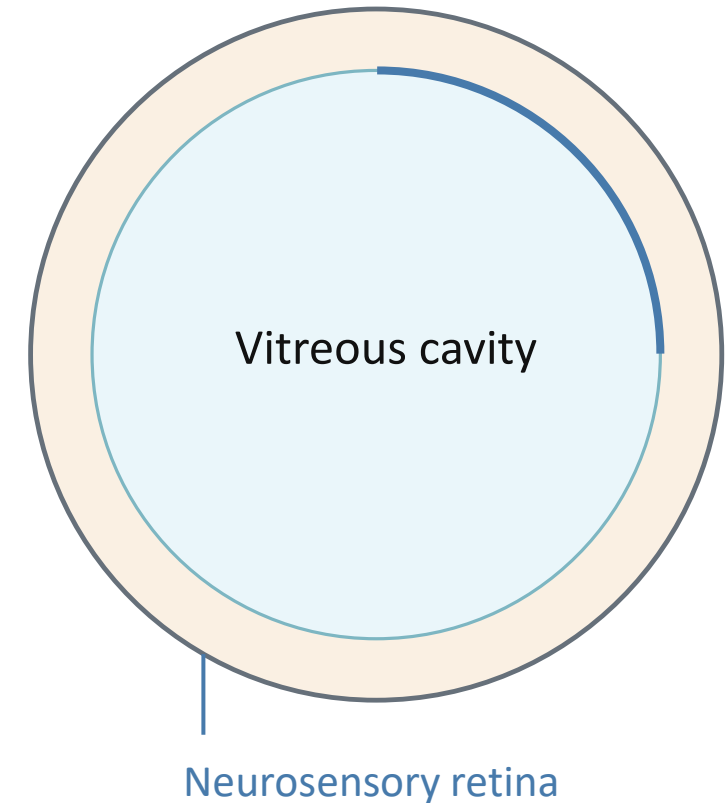
Retina and Vitreous

SCHEMATIC

Morphologic anatomy • vitreoretinal interface

• acute inflammation

- The neurosensory retina is a specialized extension of the central nervous system.
- Retinal layer arrangement governs the appearance of hemorrhages and exudates.
- The vitreoretinal interface becomes clinically important in aging, vascular injury, and infection.



Dr Abu Suraih Sakhri

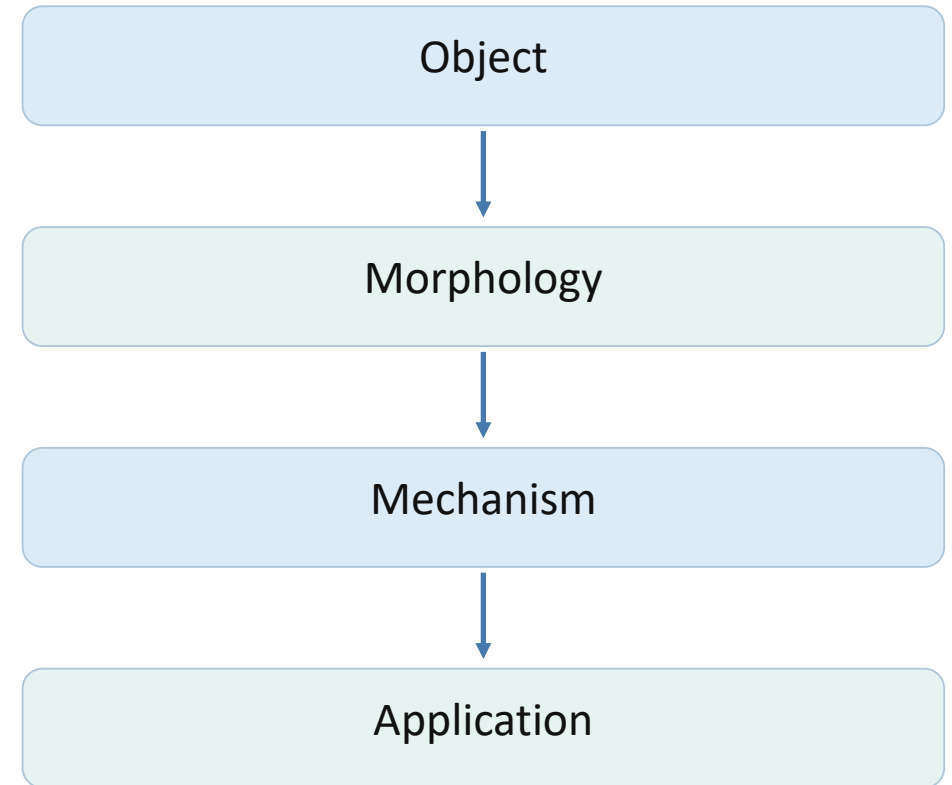
MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning objectives

- Reconstruct the retinal layers and relate their orientation to fundus findings.
- Explain retinal pigment epithelium and vitreous contributions to pathology.
- Distinguish infectious retinitis, endophthalmitis, and panophthalmitis by compartment and route.
- Apply these principles to retinal detachment and neovascular disease.

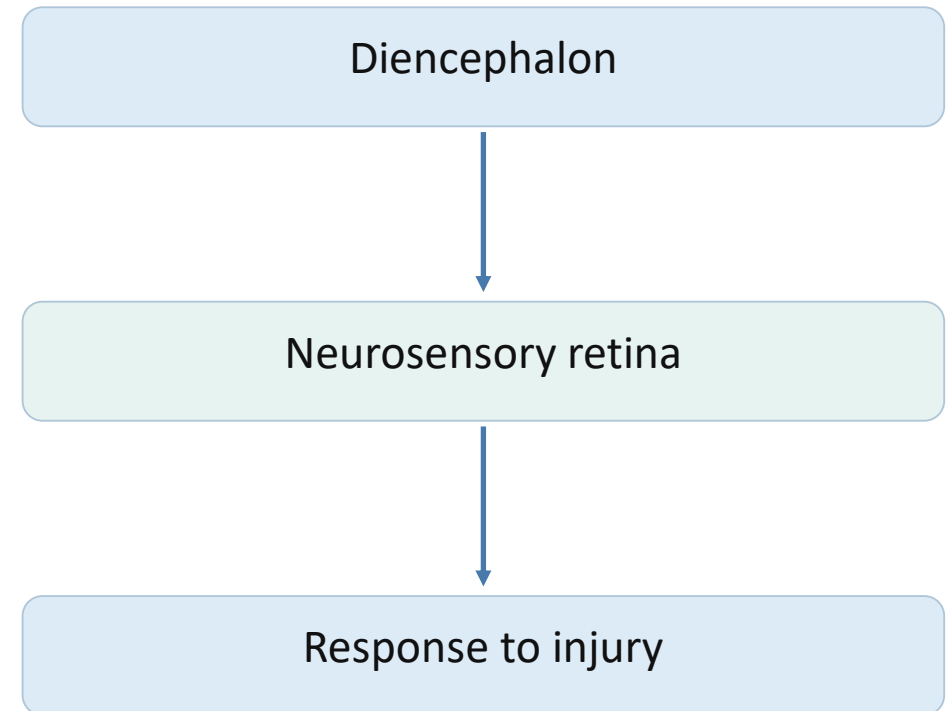
SCHEMATIC



The retina is neural tissue

- Neurosensory retina and optic nerve originate embryologically from the diencephalon.
- The retina therefore shares important biologic responses with the brain.
- Retinal injuries are interpreted through both neurobiology and ocular anatomy.

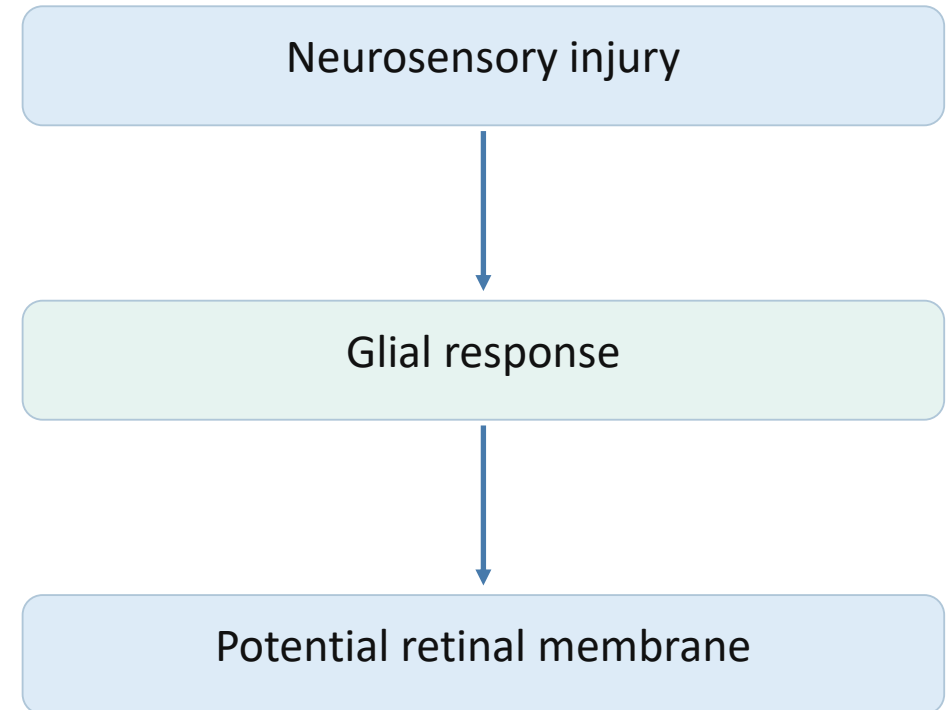
SCHEMATIC



A CNS-type injury response: Gliosis

- Retinal injury elicits gliosis rather than an ordinary fibrous tissue response.
- This response parallels the central nervous system.
- Gliosis becomes relevant to the membranes complicating some retinal detachments.

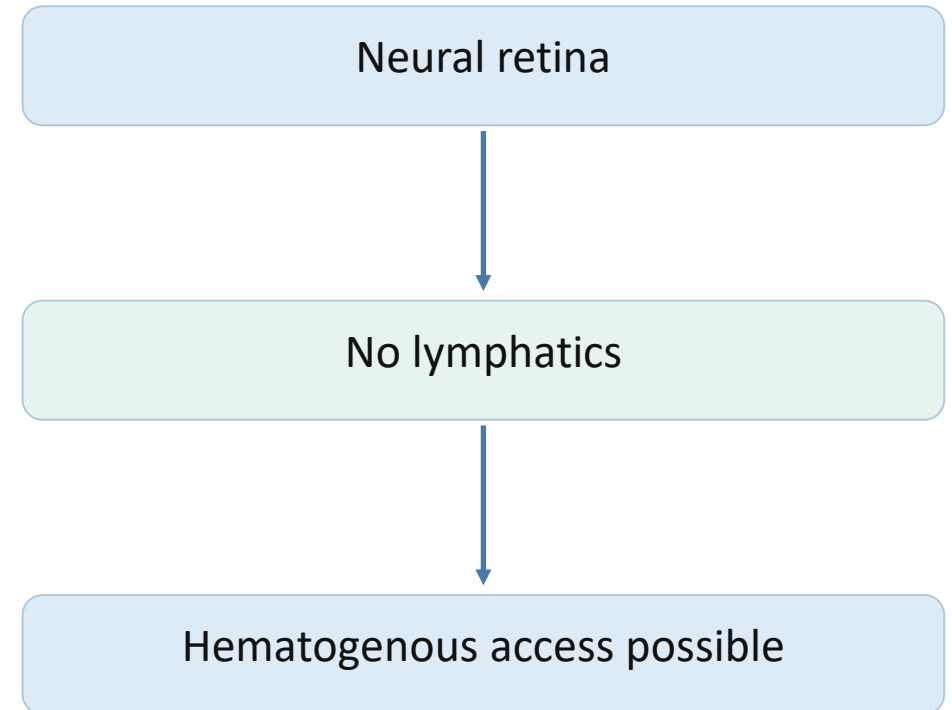
SCHEMATIC



The retina lacks lymphatics

- As in the brain, the neural retina does not have lymphatic vessels.
- This feature distinguishes retinal tissue from many other peripheral organs.
- Blood-borne dissemination nevertheless provides an infectious route to the retina.

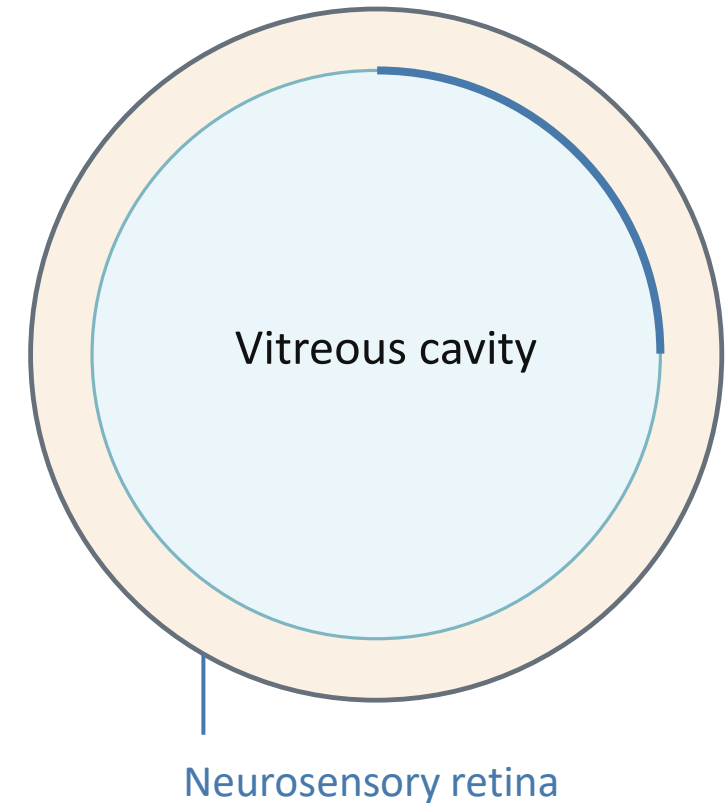
SCHEMATIC



Retina and vitreous occupy adjoining compartments

SCHEMATIC

- The vitreous lies immediately inside the neurosensory retinal surface.
- The retinal pigment epithelium lies external to the neurosensory retina.
- The posterior hyaloid forms a mechanically important interface with retinal inner surface.

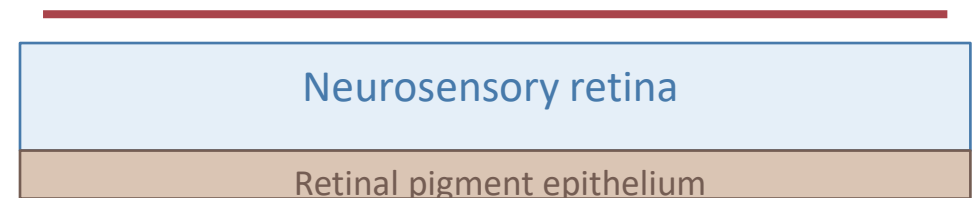


Two retinal components must be distinguished

SCHEMATIC

- Neurosensory retina: Stratified neural tissue participating in vision.
- Retinal pigment epithelium (RPE): Supporting epithelium facing photoreceptor outer segments.
- The potential space between these components is the plane of retinal detachment.

Posterior hyaloid apposed to ILM



Retinal layers: Orientation from vitreous to choroid

- Internal limiting membrane faces the vitreous.
- The nerve fiber and ganglion cell layers are relatively internal.
- Plexiform and nuclear layers occupy the deeper retina.
- Photoreceptors contact the RPE at the outer boundary.

SCHEMATIC

Vitreous / internal limiting membrane
Nerve fiber layer
Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
External limiting membrane
Rods and cones
Retinal pigment epithelium

Internal limiting membrane is a surface landmark

- The internal limiting membrane forms the innermost retinal boundary.
- The posterior hyaloid may remain apposed to or separate from this surface.
- Neovascular tissue crossing this surface is clinically meaningful in proliferative retinopathy.

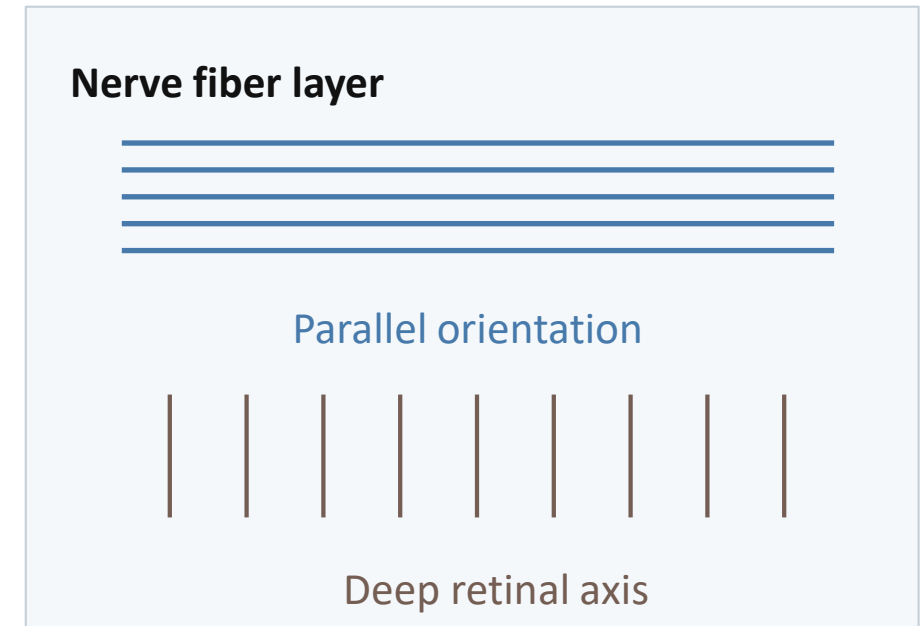
SCHEMATIC

Vitreous / internal limiting membrane
Nerve fiber layer
Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
External limiting membrane
Rods and cones
Retinal pigment epithelium

The nerve fiber layer is oriented tangentially

SCHEMATIC

- Ganglion cell axons run approximately parallel to the retinal inner surface.
- Bleeding confined to this layer follows its elongated anatomical architecture.
- This produces the characteristic superficial flame-shaped pattern.



Ganglion cell and inner plexiform layers

- The ganglion cell layer lies adjacent to the nerve fiber layer.
- The inner plexiform layer sits deeper within the neural retina.
- These landmarks help separate superficial from deep retinal pathology.

SCHEMATIC

Vitreous / internal limiting membrane
Nerve fiber layer
Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
External limiting membrane
Rods and cones
Retinal pigment epithelium

The inner nuclear layer is a depth landmark

- The inner nuclear layer lies between the two plexiform layers.
- Pathologic material deeper than the nerve fiber layer has a different fundusoscopic appearance.
- A three-dimensional lesion may appear rounded in frontal view.

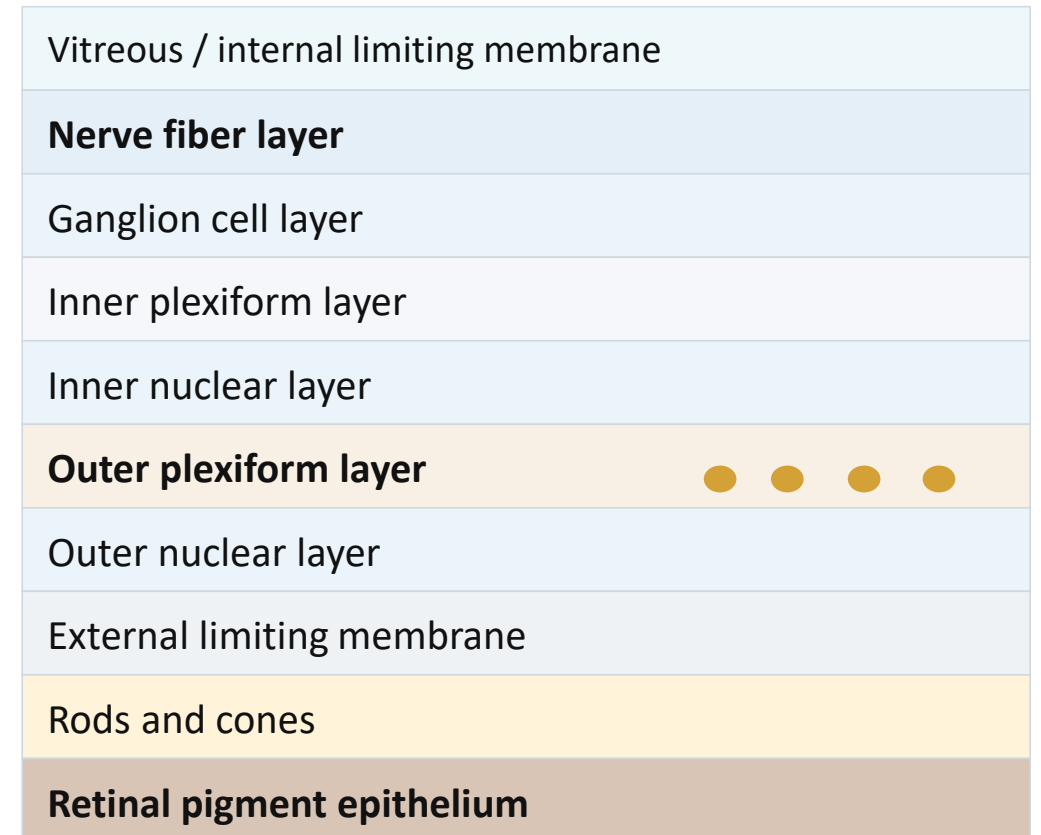
SCHEMATIC

Vitreous / internal limiting membrane
Nerve fiber layer
Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
External limiting membrane
Rods and cones
Retinal pigment epithelium

Outer plexiform layer: Exudate localization

- Fluid leaking from retinal vessels can leave exudative material in the outer plexiform layer.
- This distribution is especially important around the macula.
- Location within the retinal laminae explains a recognizable fundusoscopic pattern.

SCHEMATIC

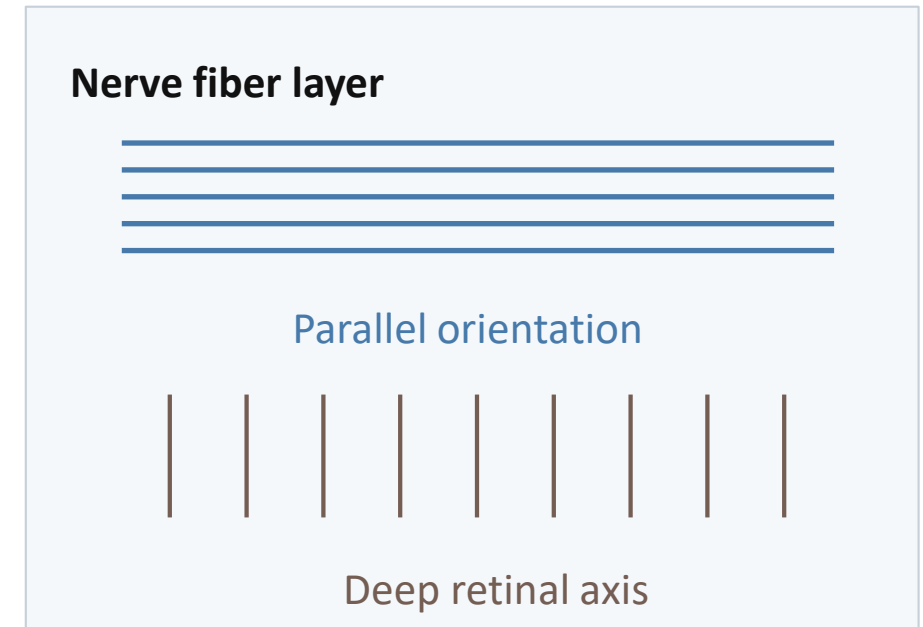


Outer plexiform deposits

Outer nuclear layer and photoreceptor axis

SCHEMATIC

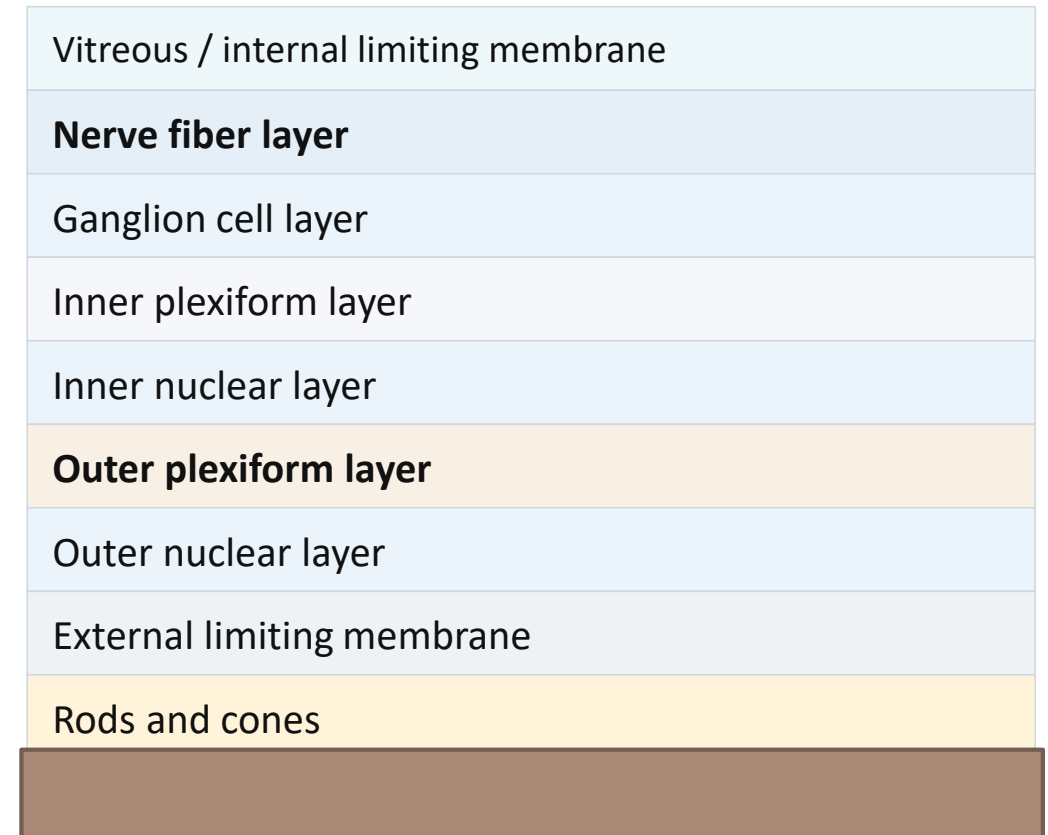
- The outer nuclear layer precedes the photoreceptor segments on the route toward the RPE.
- Deep retinal elements are predominantly perpendicular to the inner limiting membrane.
- This geometric orientation differs from the superficial nerve fiber layer.



Photoreceptor outer segments face the RPE

- Rods and cones sit externally within the neurosensory retina.
- The RPE has a vital role in maintaining photoreceptor outer segments.
- Disease at this interface can compromise retinal function even without a retinal tear.

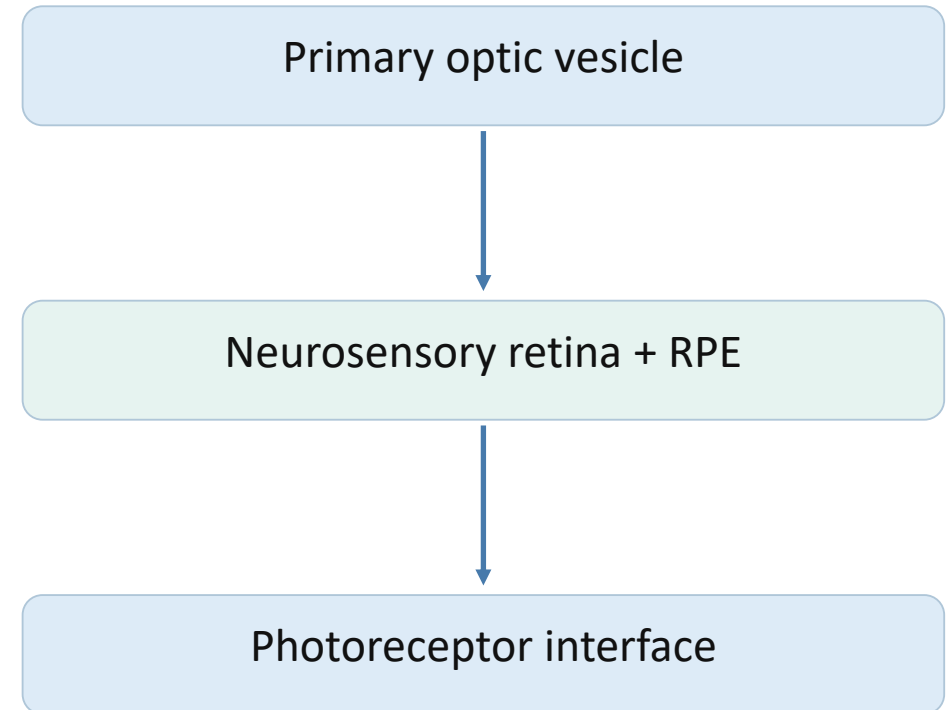
SCHEMATIC



The RPE is derived from the optic vesicle

- The RPE and neurosensory retina share developmental origins in the primary optic vesicle.
- The optic vesicle is an outpouching of the developing brain.
- An embryologic connection explains why both tissues form one functional interface.

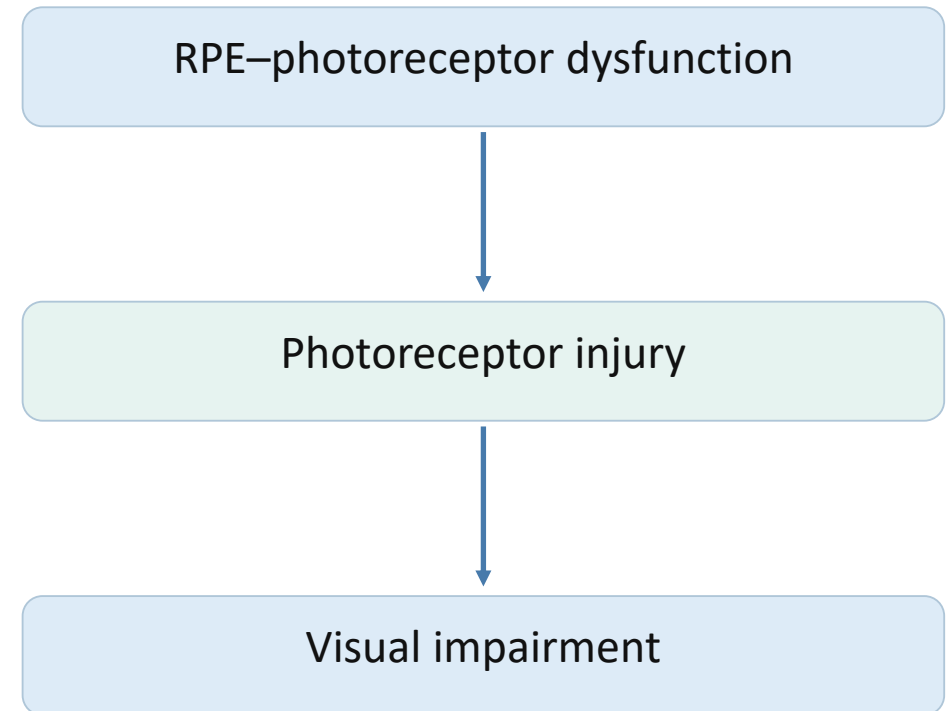
SCHEMATIC



RPE dysfunction links to retinal degeneration

- Damage to the RPE–photoreceptor interface is implicated in hereditary retinal degeneration.
- Retinitis pigmentosa is an example noted by Robbins.
- The distinction is loss of support, not necessarily primary infection or hemorrhage.

SCHEMATIC

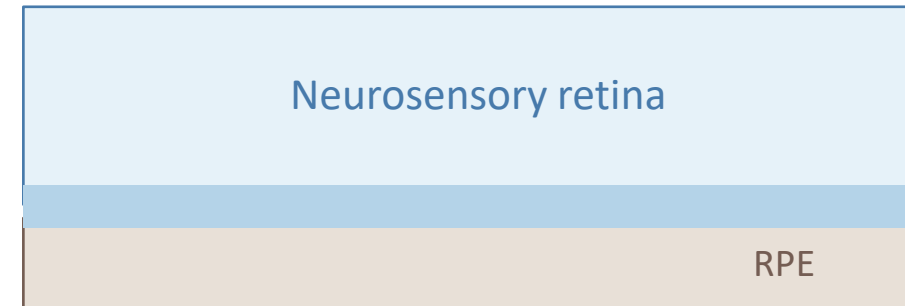


Retinal detachment is a tissue-plane separation

SCHEMATIC

- Detachment separates neurosensory retina from the underlying RPE.
- It is not synonymous with posterior vitreous detachment.
- Fluid or traction can disrupt the normal interface.

Subretinal fluid without retinal break



Layer localization governs what the examiner sees

- The morphology of a hemorrhage depends on the layer in which blood accumulates.
- Superficial and deep hemorrhages can have different shapes in the same eye.
- Vessel leakage also produces exudates at characteristic retinal depths.

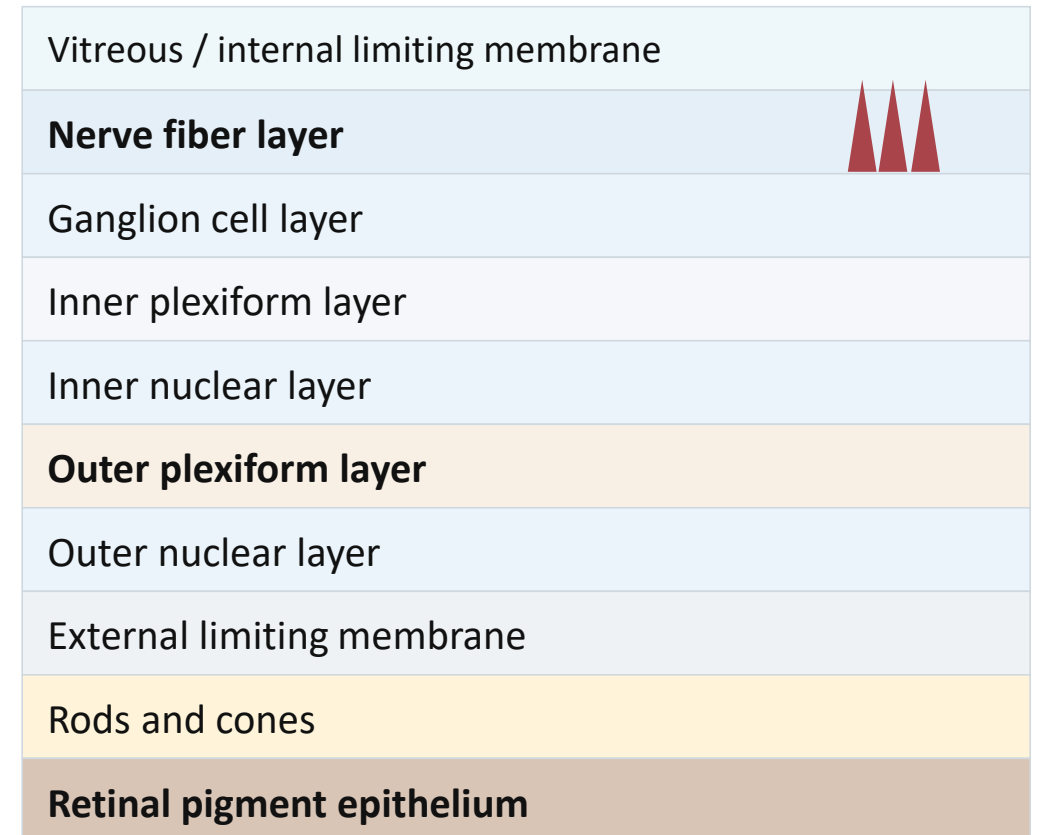
SCHEMATIC

Vitreous / internal limiting membrane
Nerve fiber layer
Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
External limiting membrane
Rods and cones
Retinal pigment epithelium

Flame hemorrhage: The superficial pattern

- Blood within the nerve fiber layer spreads along tangential fibers.
- The resulting ophthalmoscopic appearance is elongated or flame-shaped.
- Shape indicates depth; It does not by itself establish a single etiology.

SCHEMATIC

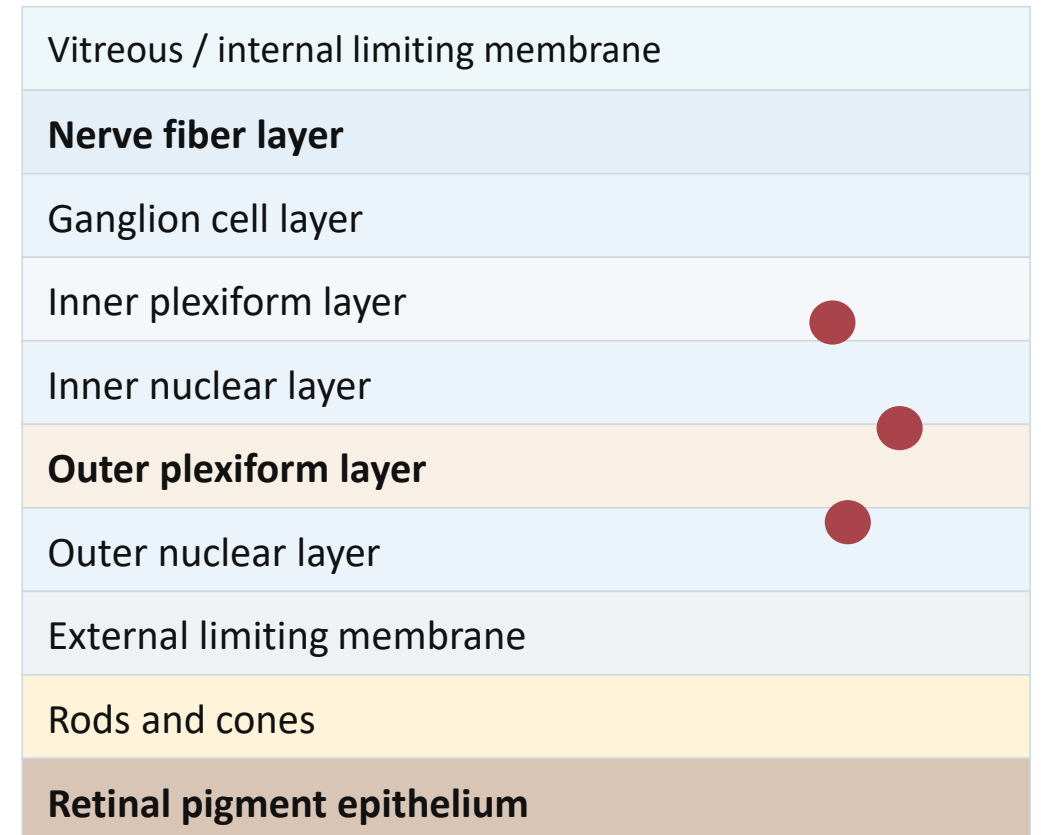


Superficial

Dot hemorrhage: Deep retinal cross-section

- The deeper layers are oriented perpendicular to the retinal surface.
- An intraretinal column of hemorrhage is seen end-on as a dot.
- This accounts for an appearance distinct from nerve fiber layer hemorrhage.

SCHEMATIC



Deeper retina

Flame and dot hemorrhages compared

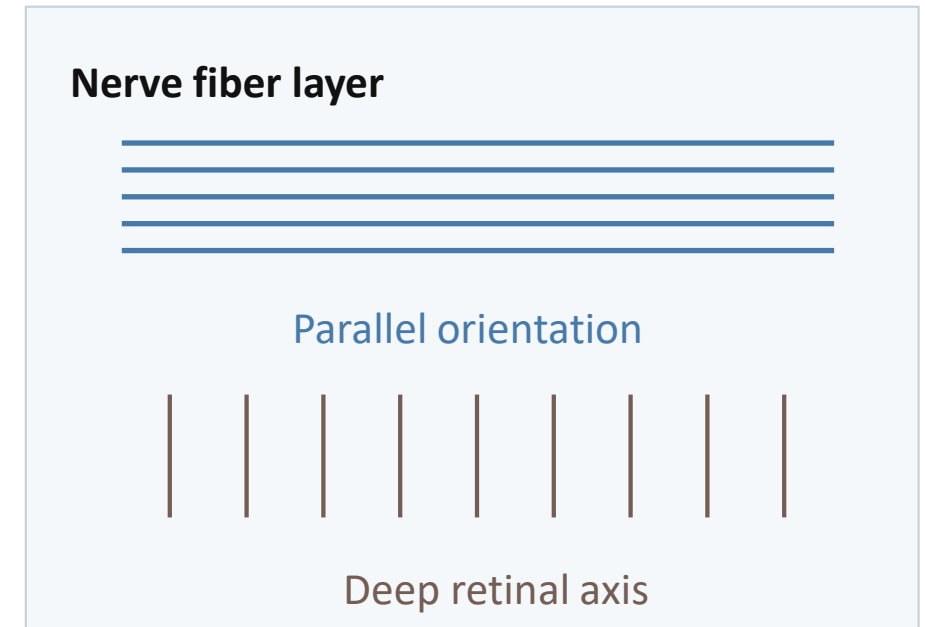
- Flame: Nerve fiber layer, horizontal orientation, elongated appearance.
- Dot: Deeper retinal layers, perpendicular orientation, round appearance.
- The same underlying vascular disorder may produce more than one pattern.

Hemorrhage pattern	Retinal plane	Fundus form
Flame	Nerve fiber layer	Streak or flame
Dot	Deeper retinal layers	Rounded dot

Layer geometry predicts hemorrhage shape

SCHEMATIC

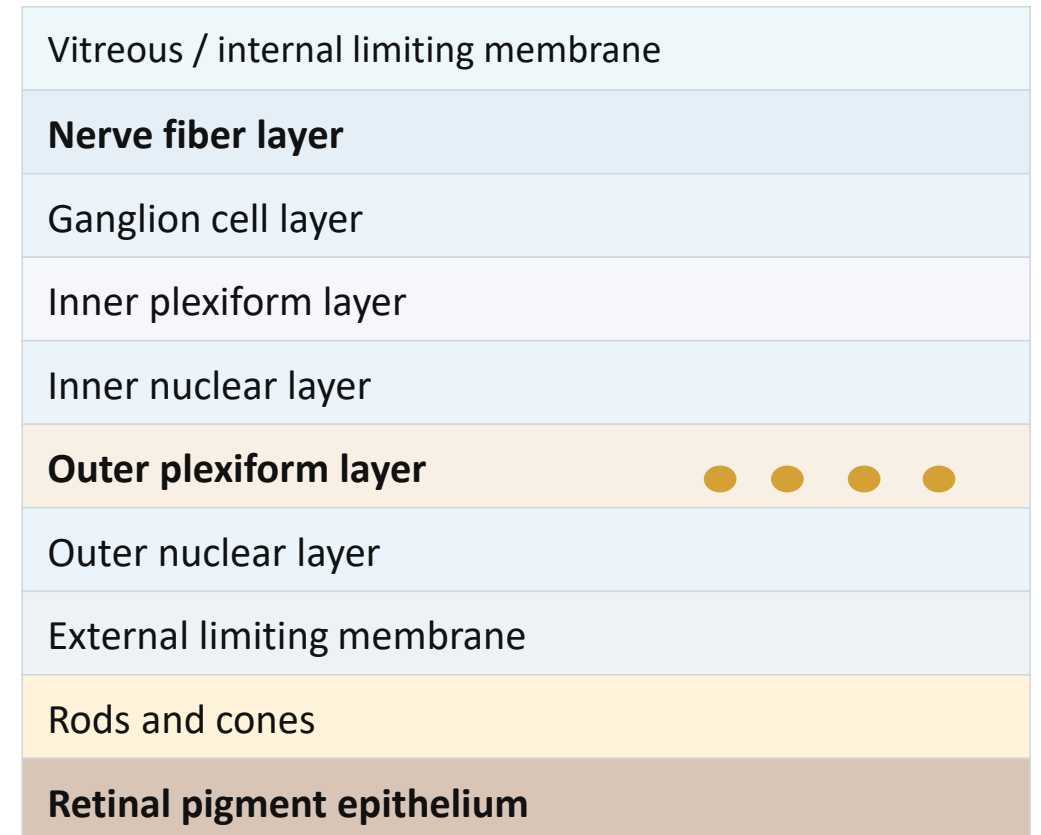
- Blood distribution is constrained by the tissue planes surrounding it.
- Parallel fibers favor longitudinal spread.
- Perpendicular architecture yields rounded cross-sections during ophthalmoscopy.



Exudates reflect vessel permeability

- Leaky retinal vessels release material into retinal tissue.
- Exudates characteristically accumulate in the outer plexiform layer.
- The pathologic finding is therefore both vascular and laminar.

SCHEMATIC

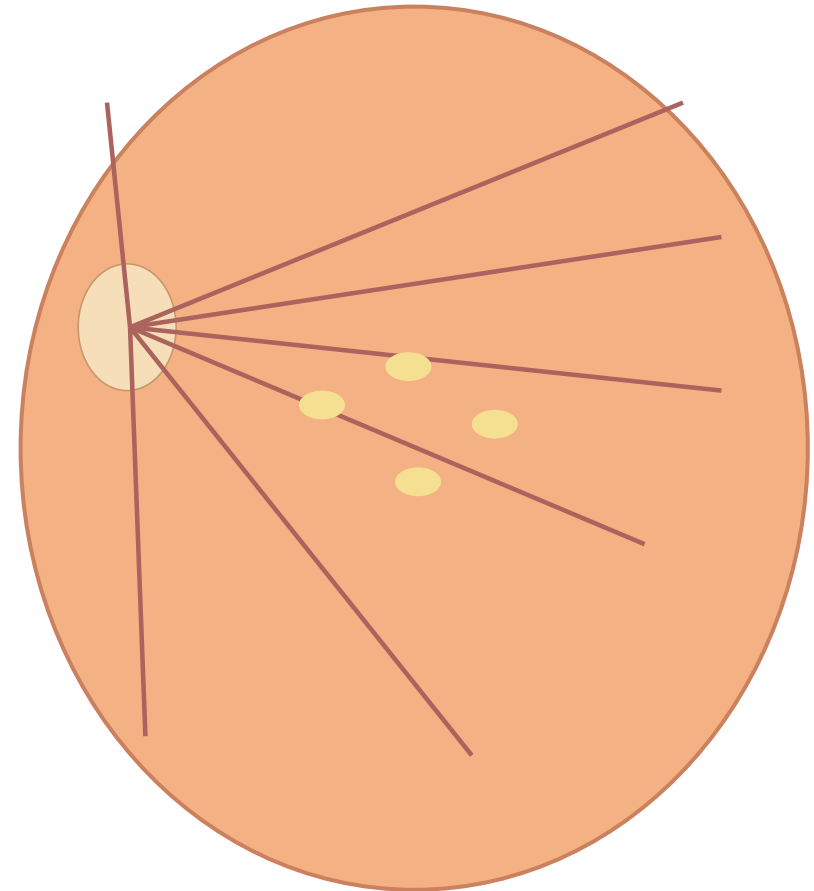


Outer plexiform deposits

Macular location matters for exudates

SCHEMATIC

- Outer plexiform exudation especially within the macula.
- Anatomical localization helps link retinal microangiopathy to visual disturbance.
- Recognize the lesion by its distribution before interpreting its cause.



Stylized fundus — not clinical evidence

Retinal vessel injury: A pattern-based reading

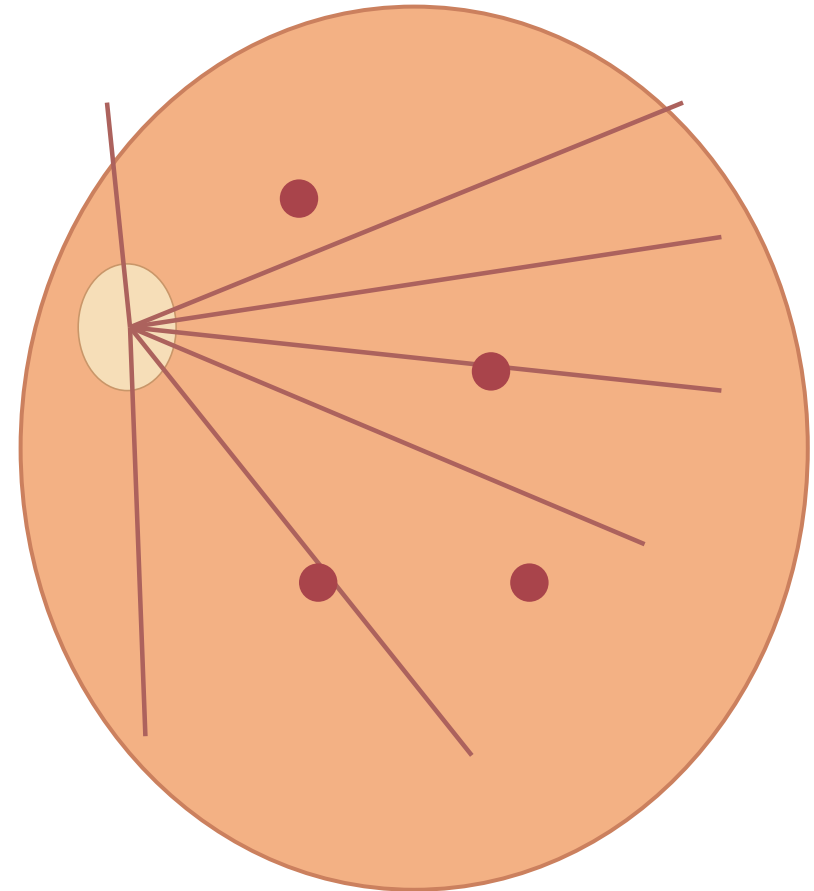
- Hemorrhage indicates extravasated blood in a retinal compartment.
- Exudate indicates leakage of material from vessels.
- Retinal vascular occlusion can produce infarction and secondary structural effects.

Finding	Basic mechanism
Hemorrhage	Blood escapes a vessel
Exudate	Vascular fluid/material leakage
Cotton-wool spot	Nerve fiber layer infarction

Diabetic microangiopathy: An application

SCHEMATIC

- Diabetic retinal vessels develop structural and permeability abnormalities.
- Microaneurysms, hemorrhages, and outer plexiform exudates may coexist.
- This application illustrates why depth, not disease name alone, predicts morphology.



Stylized fundus — not clinical evidence

Cotton-wool spot: A different superficial lesion

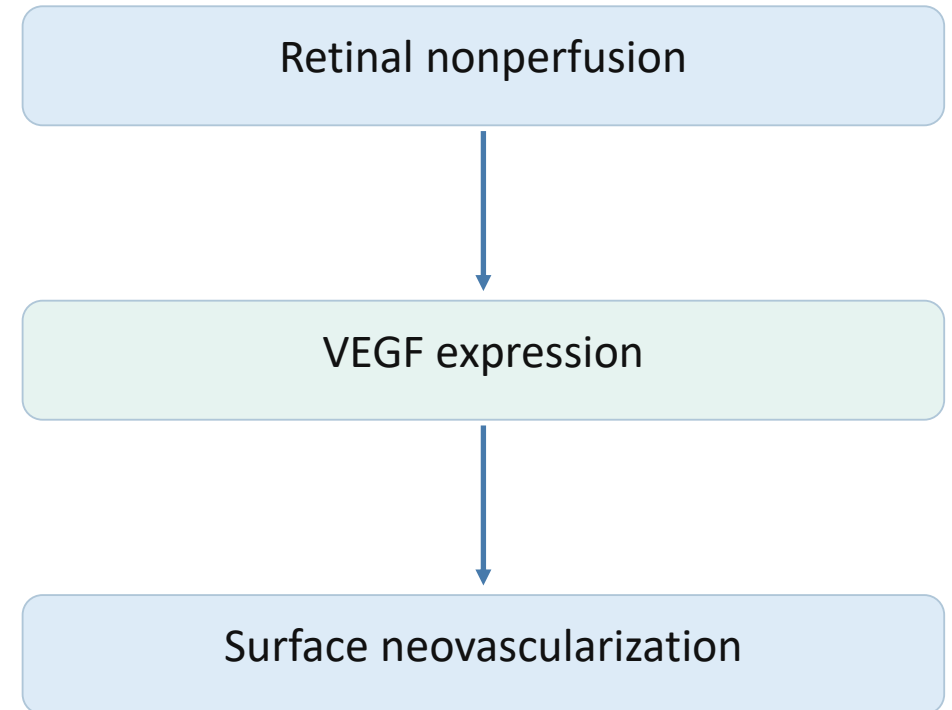
- A cotton-wool spot represents a nerve fiber layer infarct.
- It is not simply a yellow outer plexiform exudate.
- Both lesions may be present in retinal vascular disease, but indicate different processes.

Lesion	Layer	Process
Cotton-wool spot	Nerve fiber layer	Focal infarction
Hard exudate	Outer plexiform layer	Vascular leakage

Diabetic neovascularization reaches the surface

- Ischemic retinal disease can drive VEGF-associated neovascularization.
- Proliferative disease features vessels extending beyond the internal limiting membrane.
- Their relationship to posterior vitreous matters for later hemorrhage and traction.

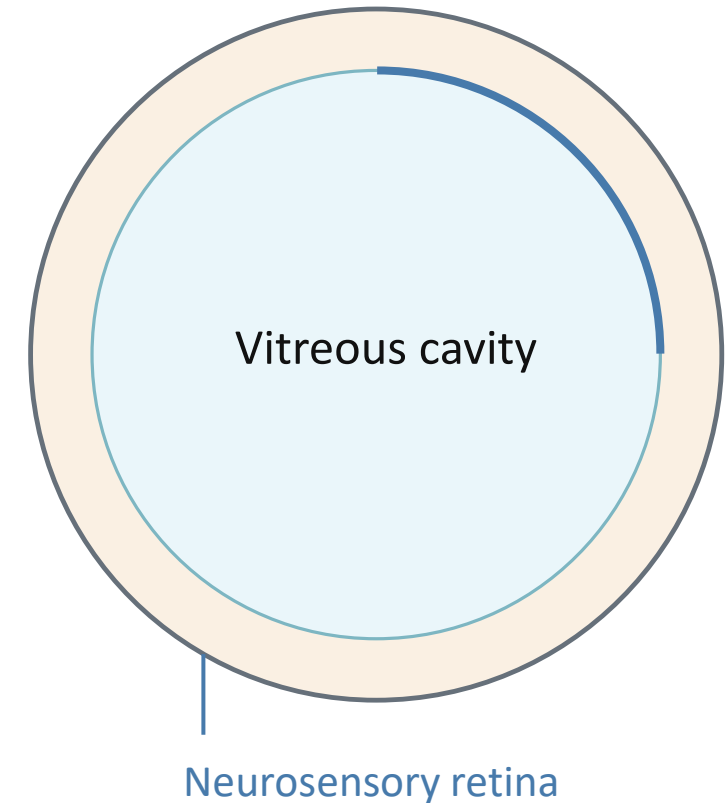
SCHEMATIC



The adult vitreous is avascular

SCHEMATIC

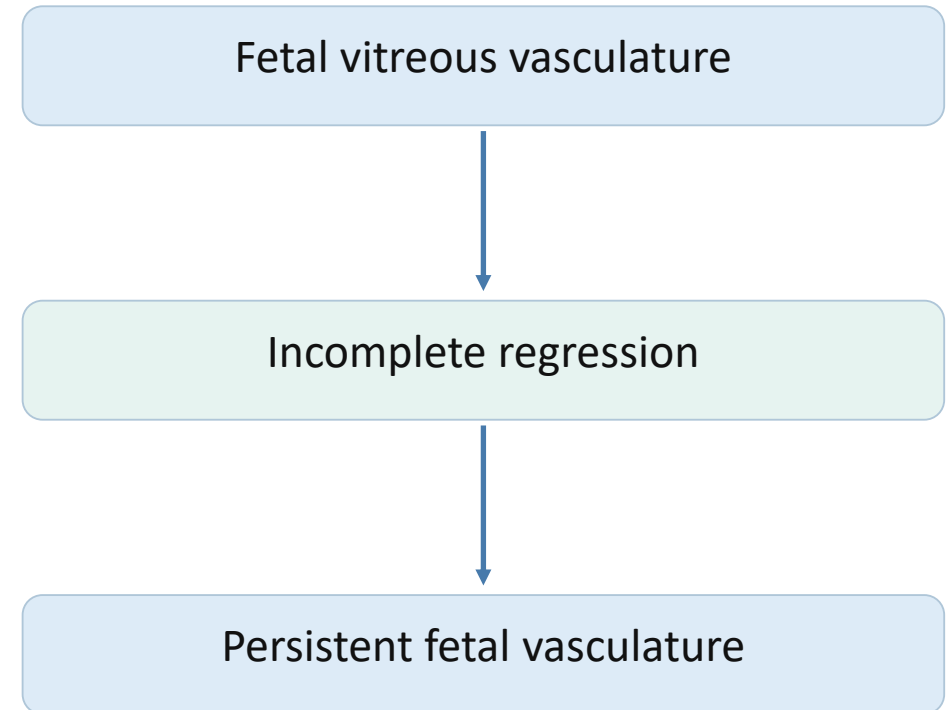
- Normal adult vitreous humor contains no intrinsic blood vessels.
- Vitreous hemorrhage therefore requires blood entering from adjacent tissues.
- Trauma and retinal neovascularization are two source-supported causes.



Developmental fetal vessels normally regress

- Vessels running through fetal vitreous are normally lost during development.
- Incomplete regression can leave persistent fetal vascular tissue.
- The resulting lesion may present as a retrolental mass.

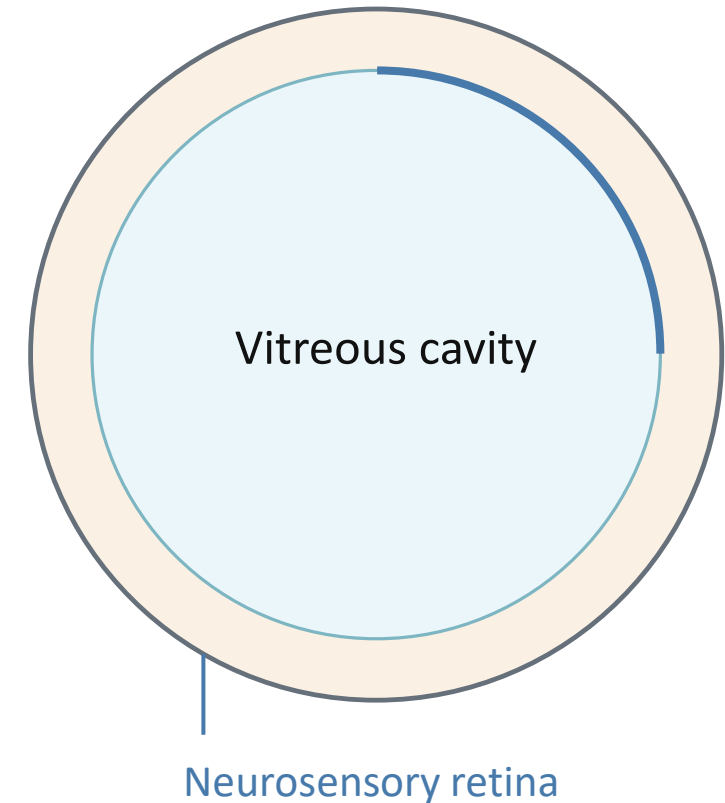
SCHEMATIC



Persistent fetal vasculature: Anatomical consequence

SCHEMATIC

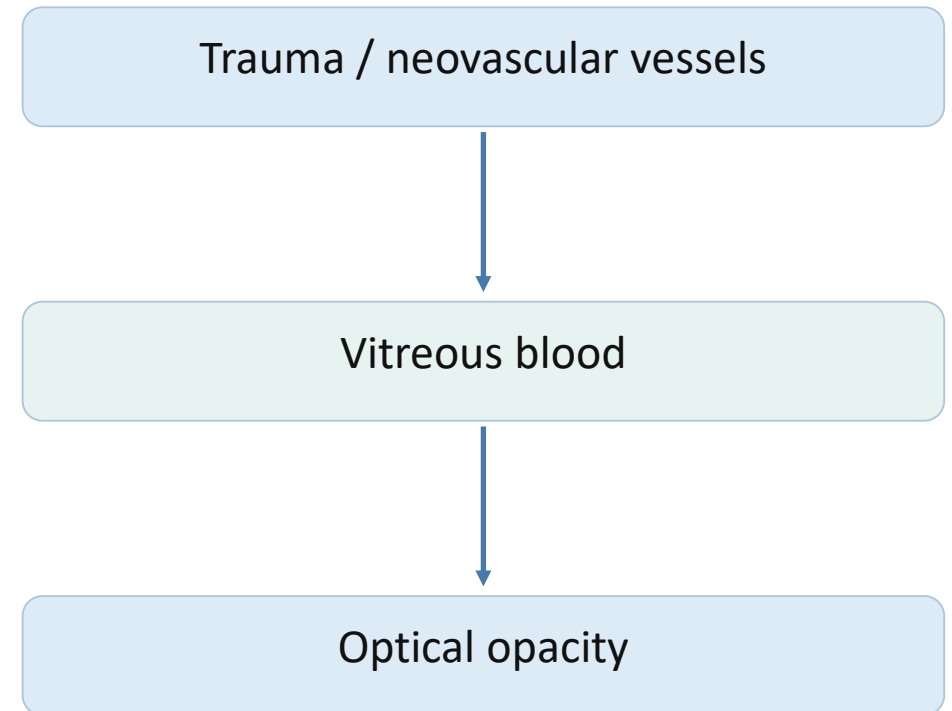
- A retrolental mass can arise when fetal vitreous vasculature persists.
- The lesion originates in abnormal persistence of developmental structures.
- It is distinct from adult vitreous hemorrhage and aging-related degeneration.



Vitreous hemorrhage compromises transparency

- Hemorrhage may opacify a normally transparent vitreous compartment.
- Trauma and pathologic retinal neovascularization are mechanisms.
- The mechanism determines why blood enters an avascular gel.

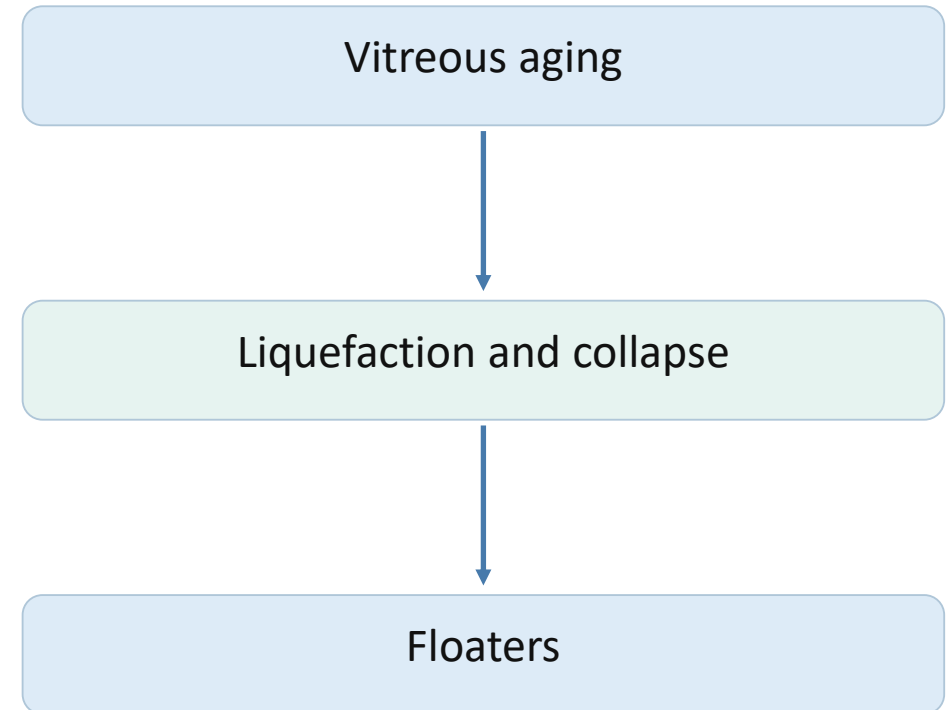
SCHEMATIC



Aging vitreous may liquefy and collapse

- The vitreous gel can liquefy with aging.
- Structural collapse may create the subjective perception of floaters.
- Vitreous alteration precedes or accompanies some posterior interface changes.

SCHEMATIC

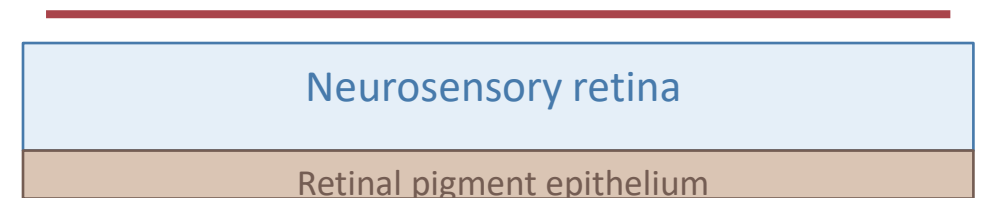


Posterior hyaloid is the vitreoretinal boundary

SCHEMATIC

- The posterior hyaloid is the posterior face of the vitreous.
- It is closely related to the internal limiting membrane of the retina.
- Its adhesion and separation are important in traction-mediated pathology.

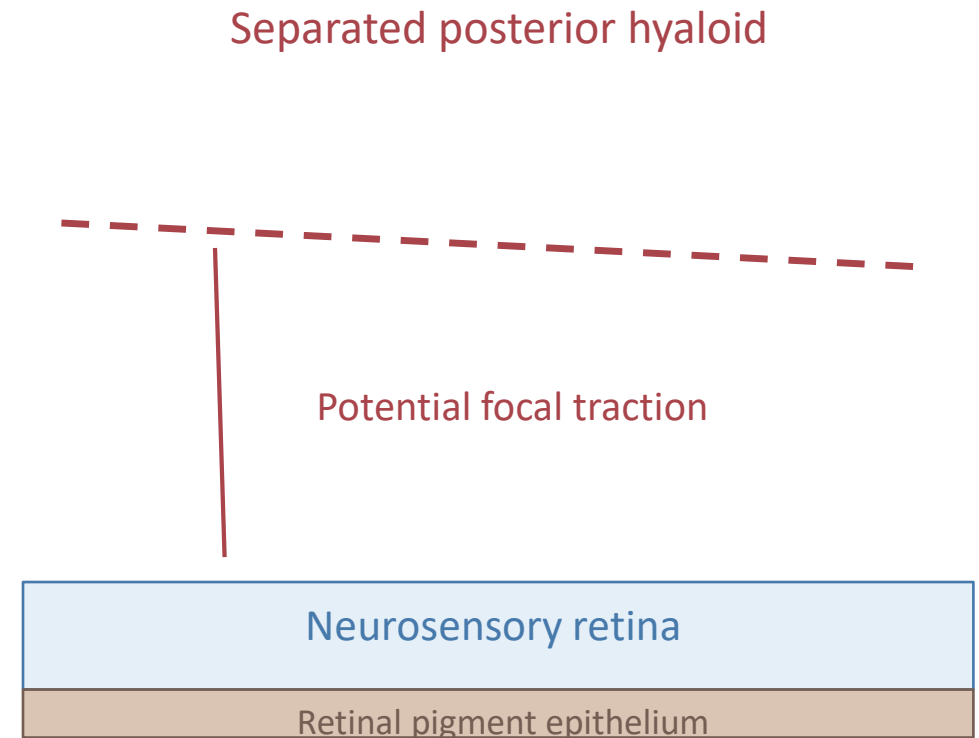
Posterior hyaloid apposed to ILM



Posterior vitreous detachment: The event

SCHEMATIC

- The posterior hyaloid can separate from the neurosensory retina with aging.
- This is a vitreous–retinal surface event.
- It differs anatomically from neurosensory retinal detachment from RPE.



Two different detachments must not be confused

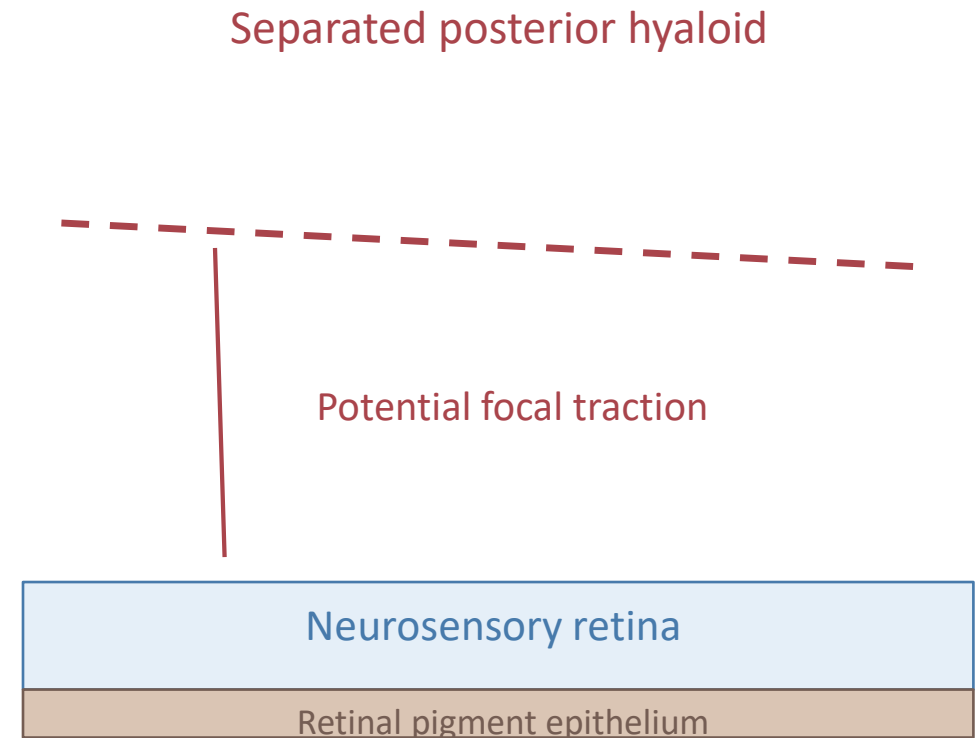
- Posterior vitreous detachment: Posterior hyaloid leaves retinal surface.
- Retinal detachment: Neurosensory retina separates from RPE.
- The former can exert traction contributing to the latter.

Event	Separated structures
Posterior vitreous detachment	Hyaloid from retinal surface
Retinal detachment	Neurosensory retina from RPE

Focal vitreoretinal adhesion can produce traction

SCHEMATIC

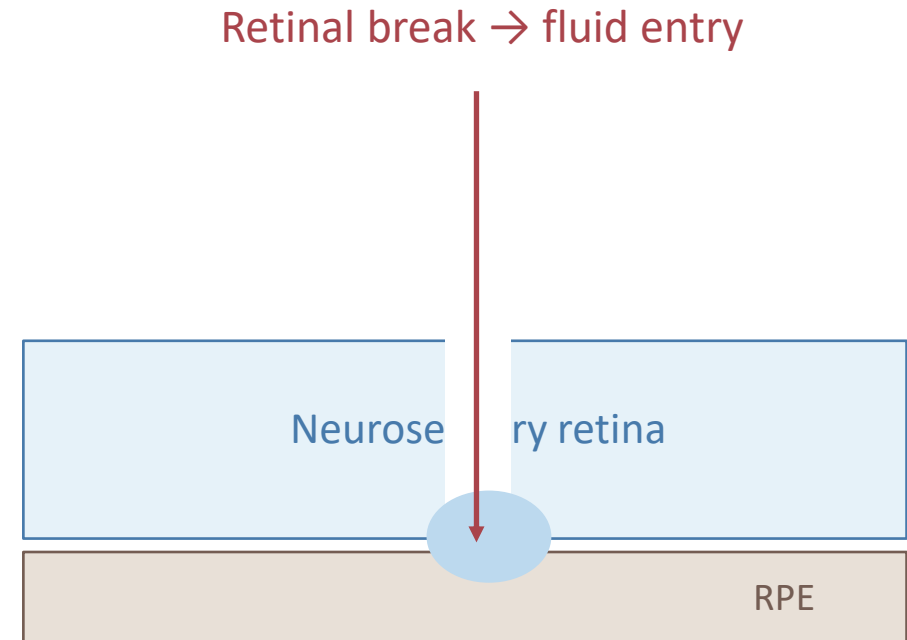
- During hyaloid separation, sites of strong adhesion may remain.
- The separating posterior vitreous can pull on the retina at these sites.
- A sufficiently strong mechanical stress may cause a retinal tear.



Retinal tear provides a route for vitreous fluid

SCHEMATIC

- A full-thickness defect opens access beneath the neurosensory retina.
- Liquefied vitreous can pass through the tear.
- Subretinal fluid separates retina from RPE in rhegmatogenous detachment.

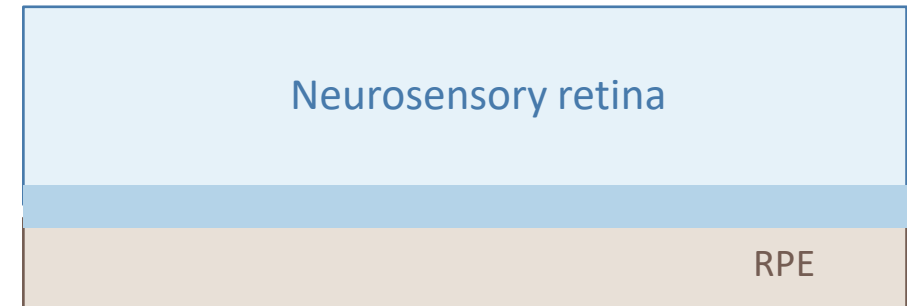


Nonrhegmatogenous detachment has no tear

SCHEMATIC

- Retinal separation also occurs without any full-thickness retinal break.
- Fluid can accumulate beneath retina with vascular exudation or RPE dysfunction.
- Examples include severe hypertension and choroidal tumors.

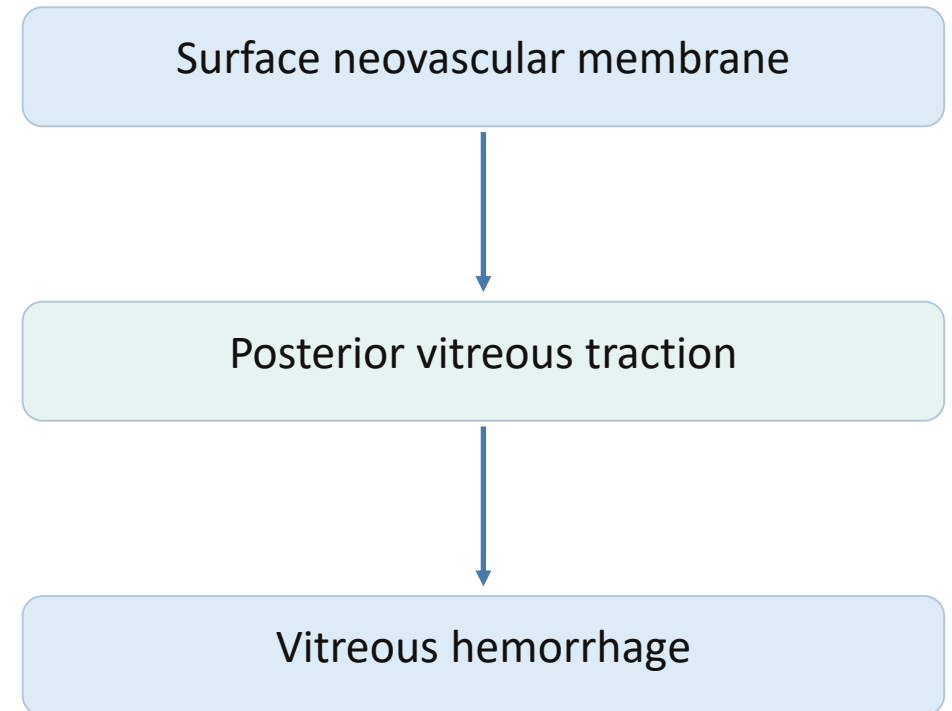
Subretinal fluid without retinal break



Neovascular membranes can couple retina and vitreous

- In proliferative retinopathy, fragile surface vessels may be accompanied by fibroglial tissue.
- The posterior hyaloid can exert force on this neovascular interface.
- Traction can precipitate vitreous hemorrhage.

SCHEMATIC



Vitreoretinal interface: Three separate failures

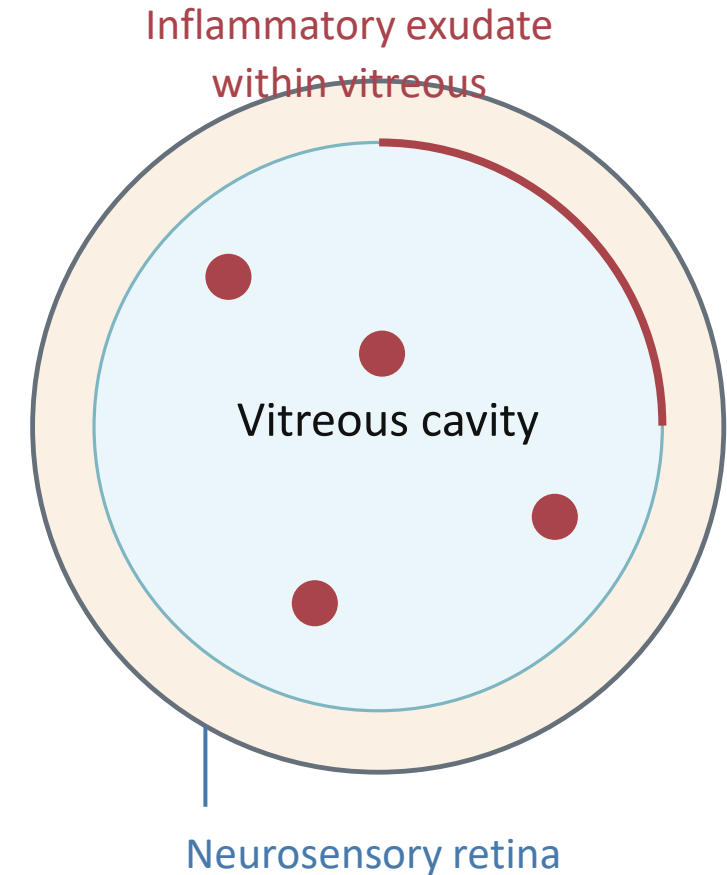
- Mechanical: Focal adhesion transmits traction to retina.
- Vascular: Fragile new vessels bleed into vitreous.
- Barrier: Retinal/RPE fluid imbalance creates subretinal collections.

Failure	Primary result
Traction	Tear / retinal distortion
Fragile new vessels	Vitreous hemorrhage
Subretinal fluid	Nonrhegmatogenous detachment

Infectious retinitis begins within retinal tissue

- A range of pathogens can produce infectious retinitis.
- The retina may be reached by organisms carried in blood.
- Retinitis is not identical to purulent inflammation filling the vitreous.

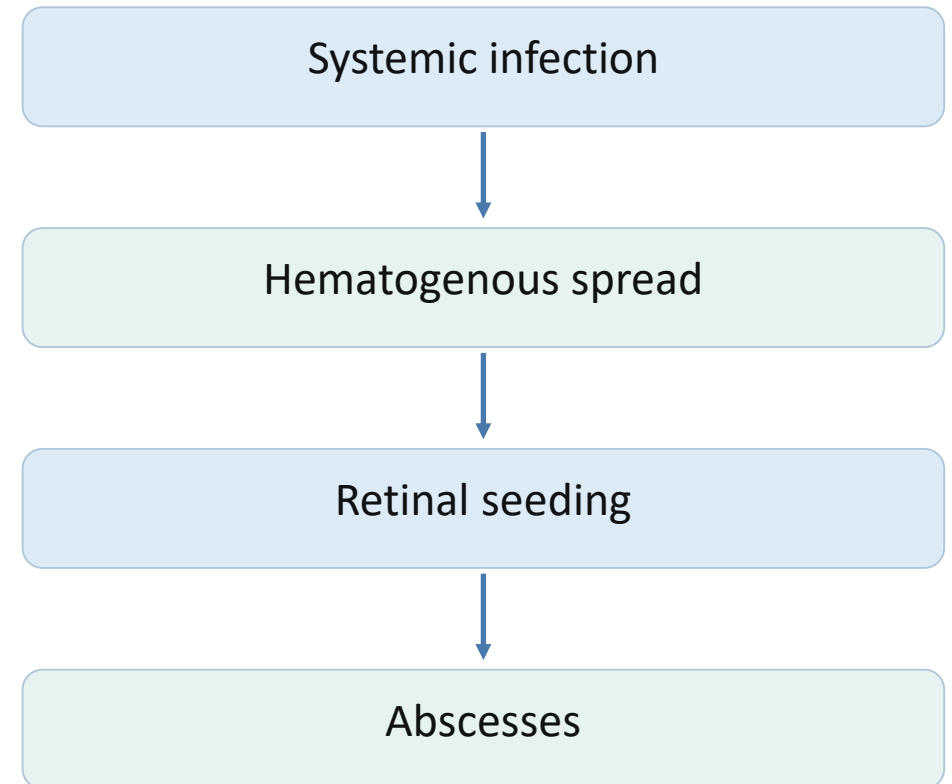
SCHEMATIC



Bloodstream dissemination to the retina

- A hematogenous route allows systemic infection to seed retinal tissue.
- Candida is one example.
- Disseminated disease can yield multiple retinal abscesses.

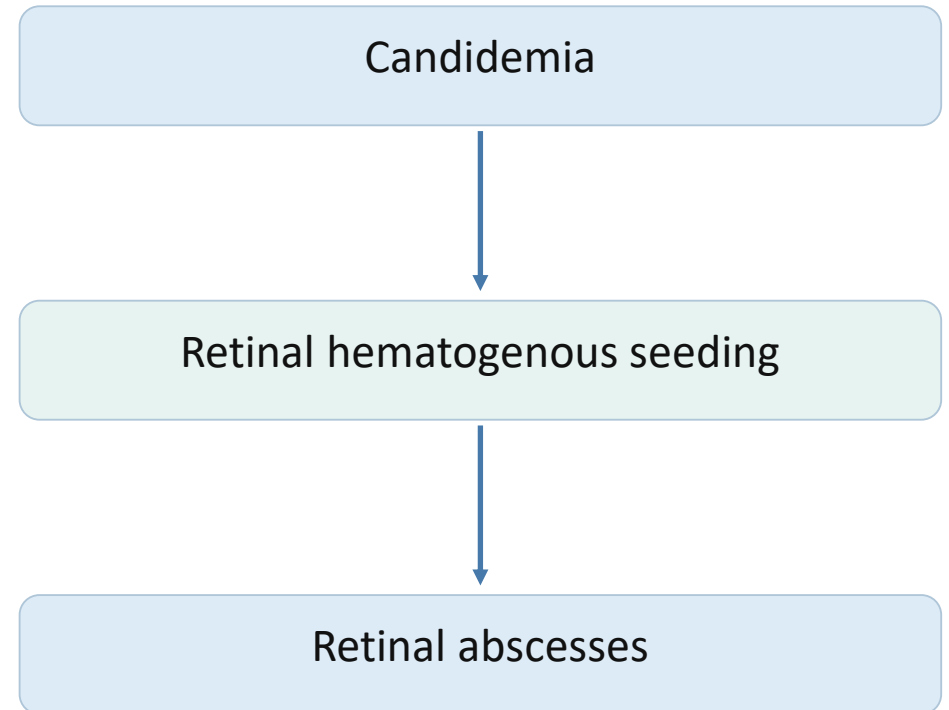
SCHEMATIC



Candida retinitis: Source-supported settings

- Disseminated Candida infection may seed the retina.
- Intravenous drug use and other causes of candidemia.
- Systemic infection supplies the route; Retinal abscesses are a possible tissue consequence.

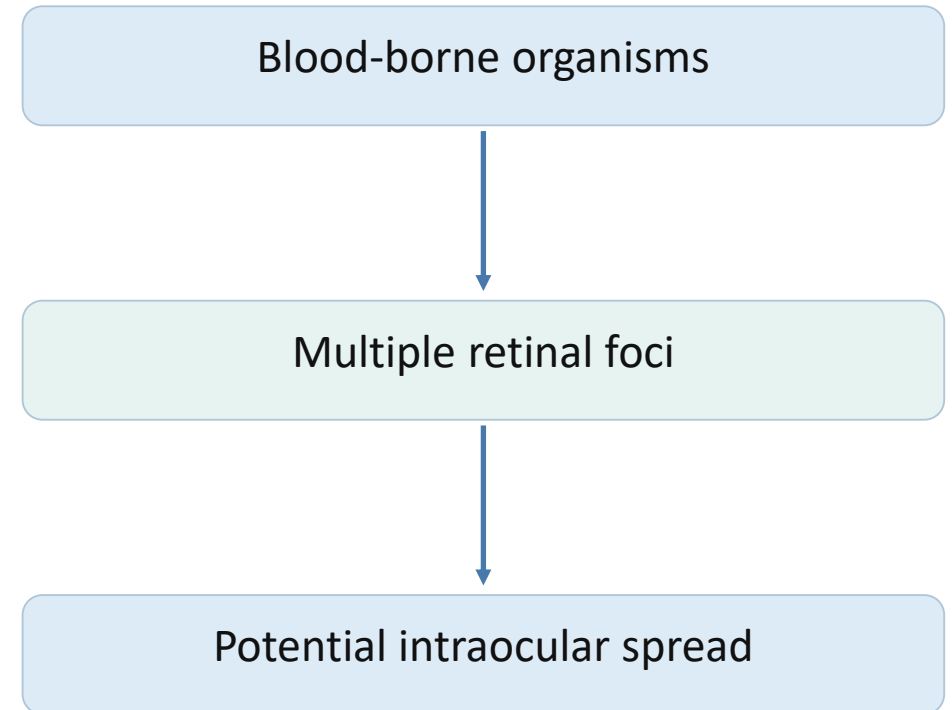
SCHEMATIC



Multiple retinal abscesses imply blood-borne spread

- Hematogenous spread typically produces multiple retinal abscesses.
- Multiplicity is linked here to dissemination rather than a single local wound.
- Determine whether the infection remains retinal or extends into the vitreous.

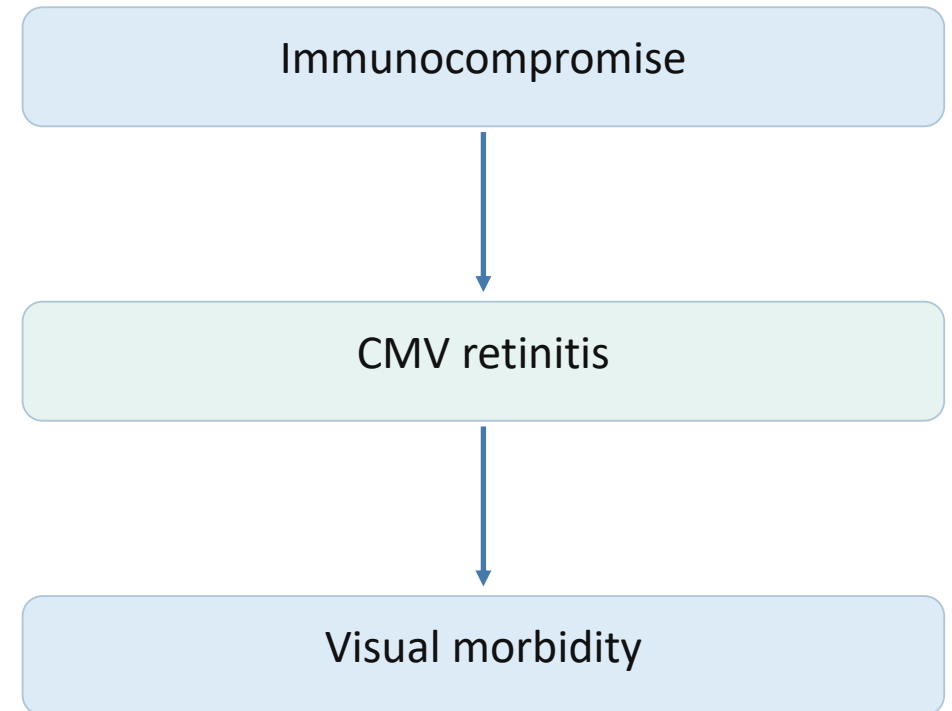
SCHEMATIC



Cytomegalovirus retinitis: Host context

- CMV retinitis is a major source of visual morbidity in immunocompromised individuals.
- The textbook especially emphasizes patients with AIDS.
- Host immune status changes the infectious differential.

SCHEMATIC



The retina and vitreous are different infection sites

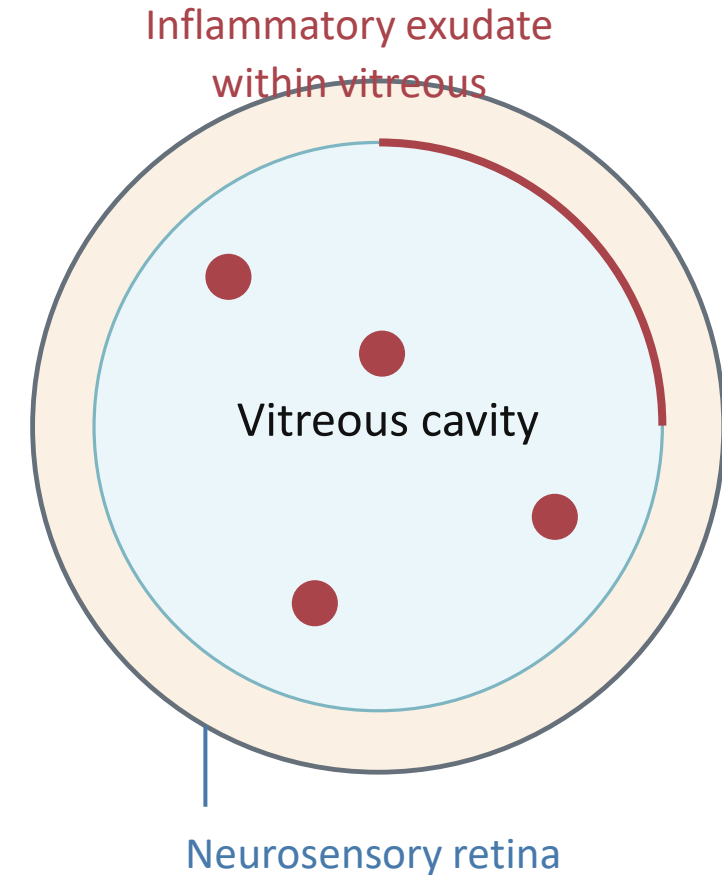
- Retinitis refers to inflammation within retinal tissue.
- Endophthalmitis denotes substantial inflammation involving intraocular compartments, notably the vitreous.
- The tissue compartment predicts the consequence for delicate retinal cells.

Condition	Main compartment
Retinitis	Retinal tissue
Endophthalmitis	Interior of globe / vitreous

Endophthalmitis: An intraocular suppurative process

- Exogenous from endogenous entry routes.
- The retina lines the vitreous cavity and poorly tolerates purulent vitreous inflammation.
- Irreversible retinal injury may develop over only a few hours.

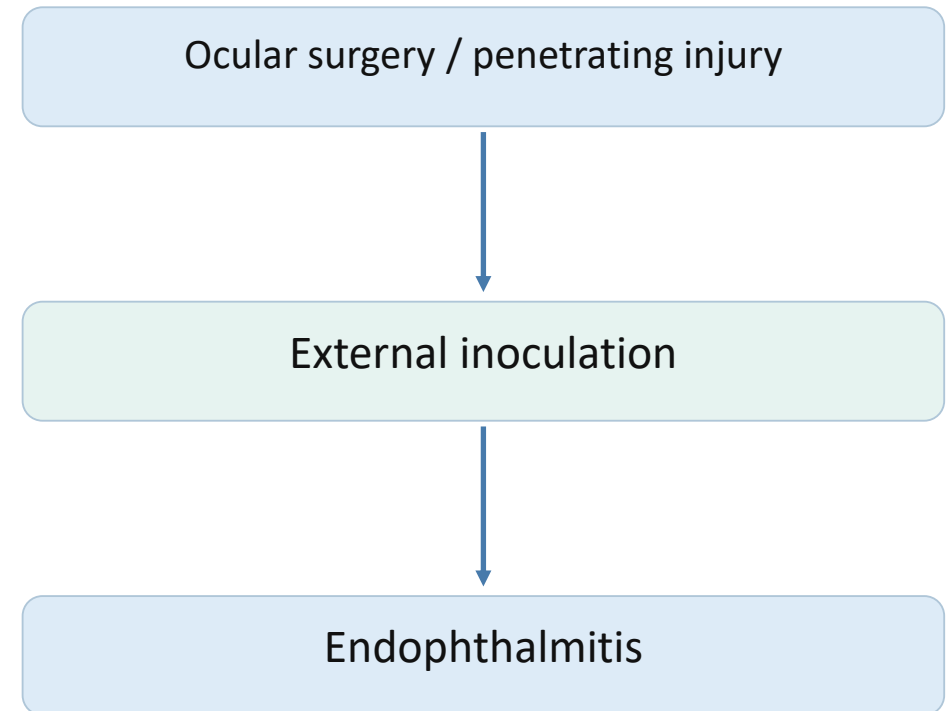
SCHEMATIC



Exogenous endophthalmitis: Direct entry

- Organisms originate in the external environment.
- They access the eye through a wound in the ocular wall.
- Examples include postoperative complications and penetrating ocular injury.

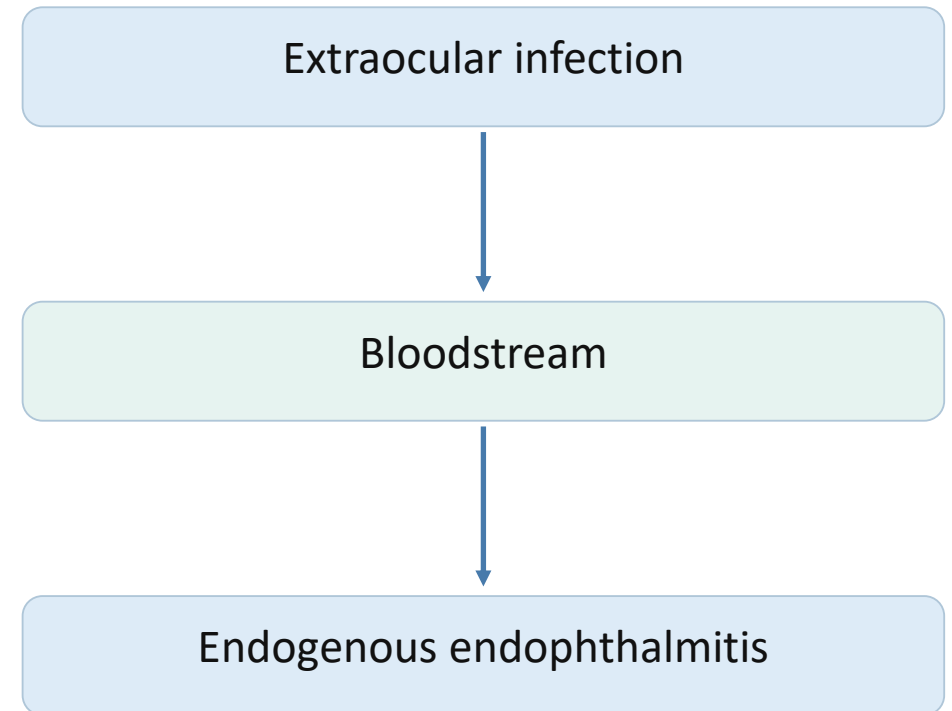
SCHEMATIC



Endogenous endophthalmitis: Hematogenous entry

- Organisms reach the eye through bloodstream dissemination.
- The initial source is outside the eye.
- Robbins gives pulmonary infection as an example of a distant source.

SCHEMATIC



Exogenous versus endogenous routes

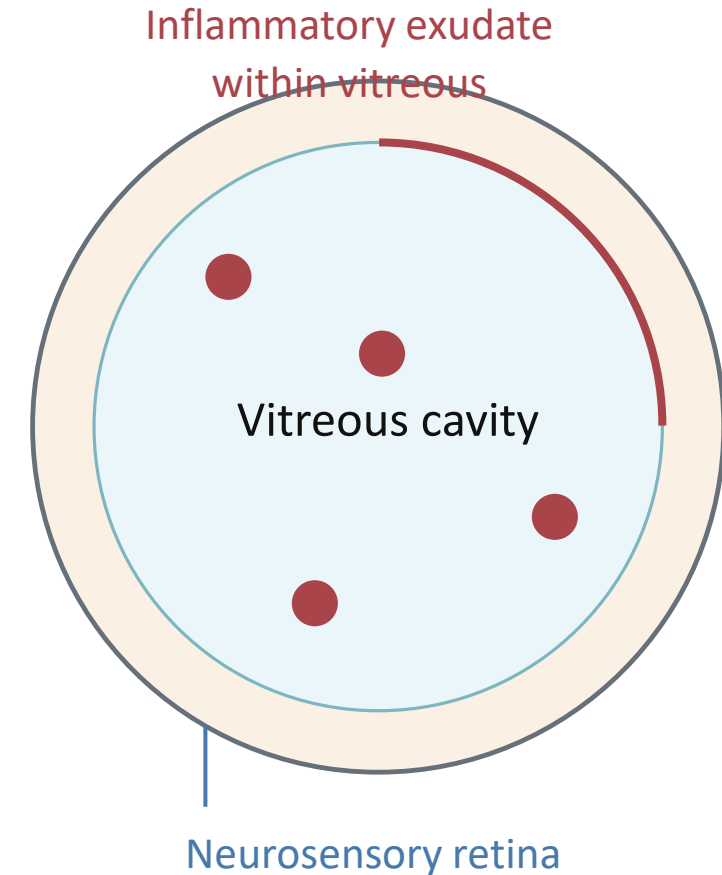
- The designation describes how the organism reaches the eye.
- Exogenous: External access through an ocular wound.
- Endogenous: Bloodstream transport from systemic infection.

Feature	Exogenous	Endogenous
Entry route	Through ocular wound	Through bloodstream
Typical setting	Surgery / penetration	Systemic infection
Primary source	Outside environment	Extraocular infection

Suppurative vitreous inflammation rapidly injures retina

- Retinal tissue borders the vitreous cavity directly.
- Purulent inflammation of the vitreous is especially poorly tolerated.
- Robbins cautions that a few hours can be enough for permanent retinal damage.

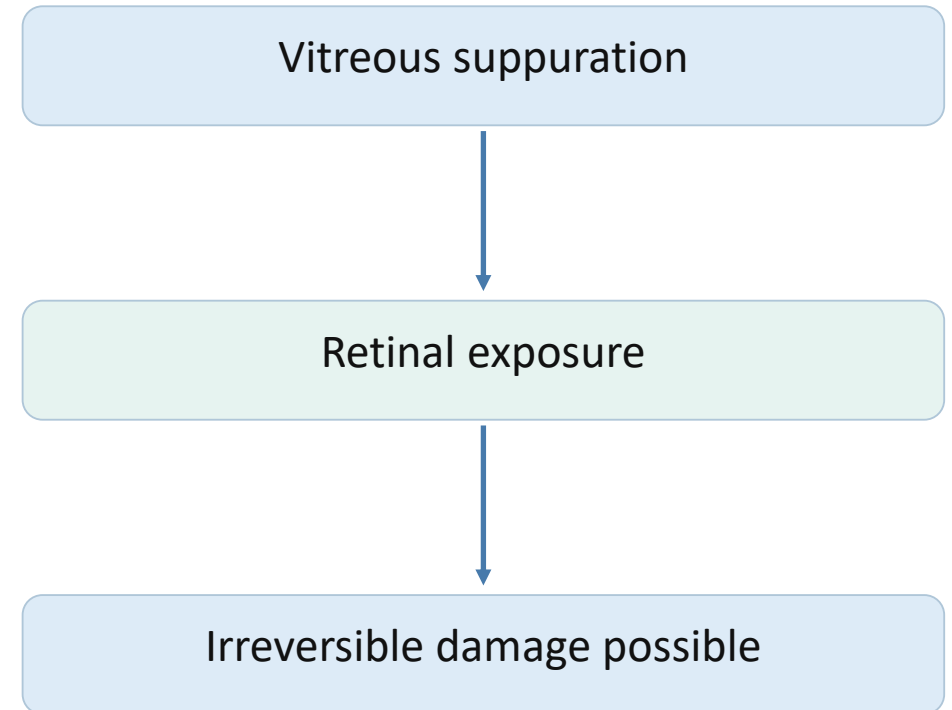
SCHEMATIC



Why endophthalmitis is time-critical

- Anatomy: Retina lines the inflamed vitreous cavity.
- Cellular vulnerability: Neural retina has limited tolerance for destructive inflammation.
- Clinical implication: Suspected endophthalmitis warrants immediate specialist assessment.

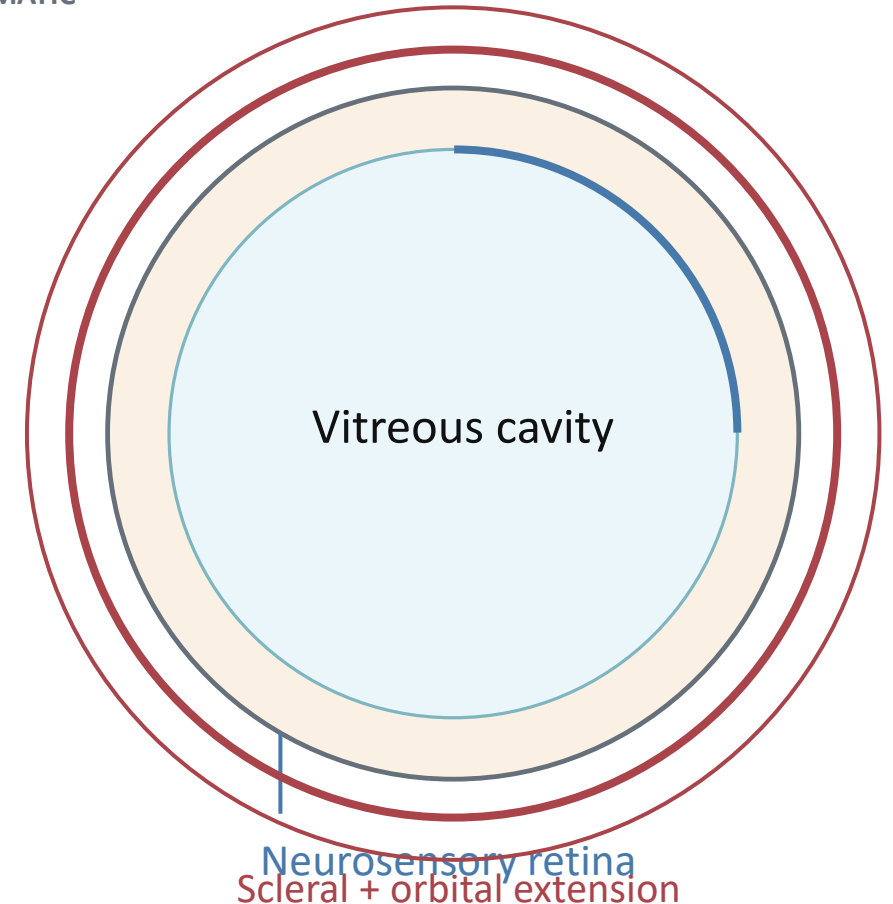
SCHEMATIC



Panophthalmitis extends across ocular coats

SCHEMATIC

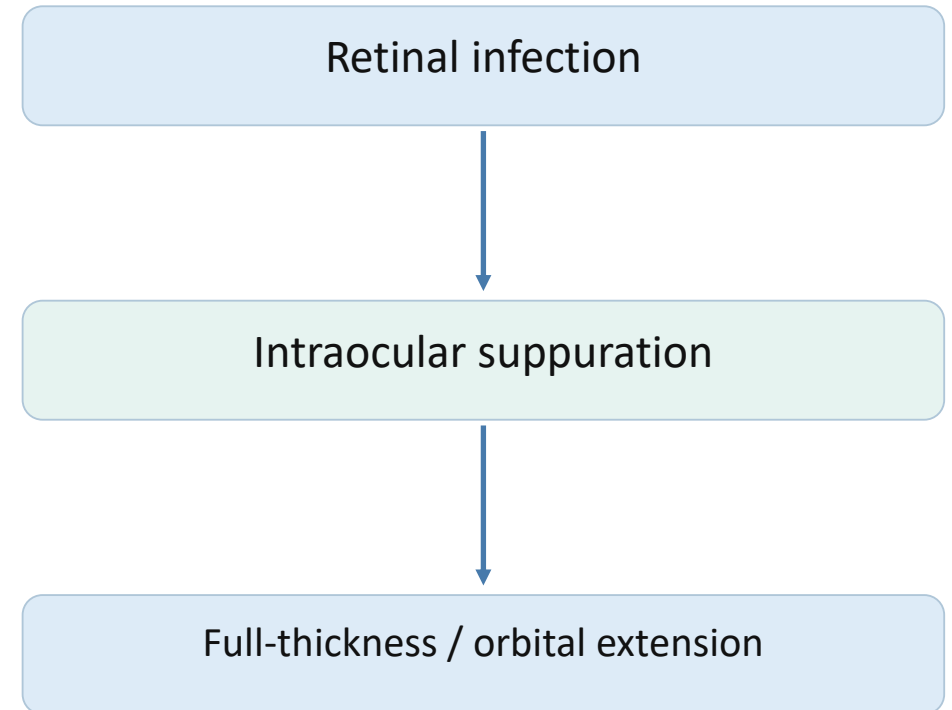
- The process affects retina, choroid, and sclera.
- Inflammation extends beyond the globe into the orbit.
- It therefore denotes a more anatomically extensive process than isolated retinitis.



Intraocular-to-orbital extension: The concept

- Retinitis is anatomically limited to retina.
- Endophthalmitis involves internal ocular compartments.
- Panophthalmitis denotes destructive extension through ocular coats into orbit.

SCHEMATIC



Three inflammatory diagnoses by extent

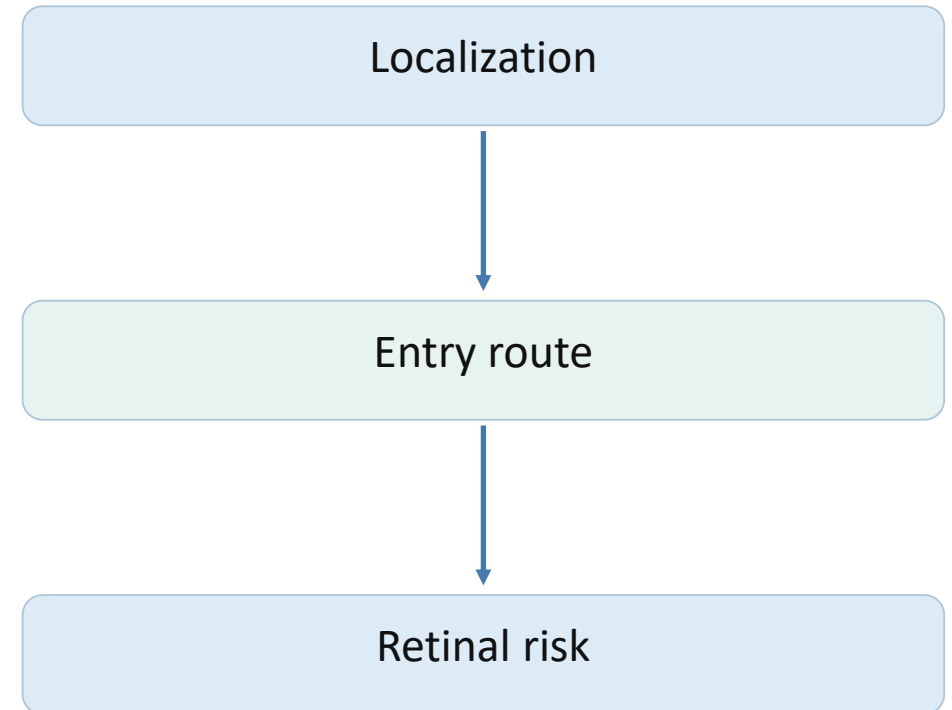
- Retinitis is retinal tissue inflammation.
- Endophthalmitis is infection/inflammation within the globe, often involving vitreous.
- Panophthalmitis spans retina, choroid, sclera, and orbit.

Diagnosis	Anatomical extent
Retinitis	Retina
Endophthalmitis	Interior globe / vitreous
Panophthalmitis	Retina + choroid + sclera + orbit

Compartment anatomy resolves diagnostic ambiguity

- Name the affected tissue first: Retina, vitreous, ocular coats, or orbit.
- Then identify the route: Hematogenous versus direct inoculation.
- Finally assess severity and risk of irreversible retinal injury.

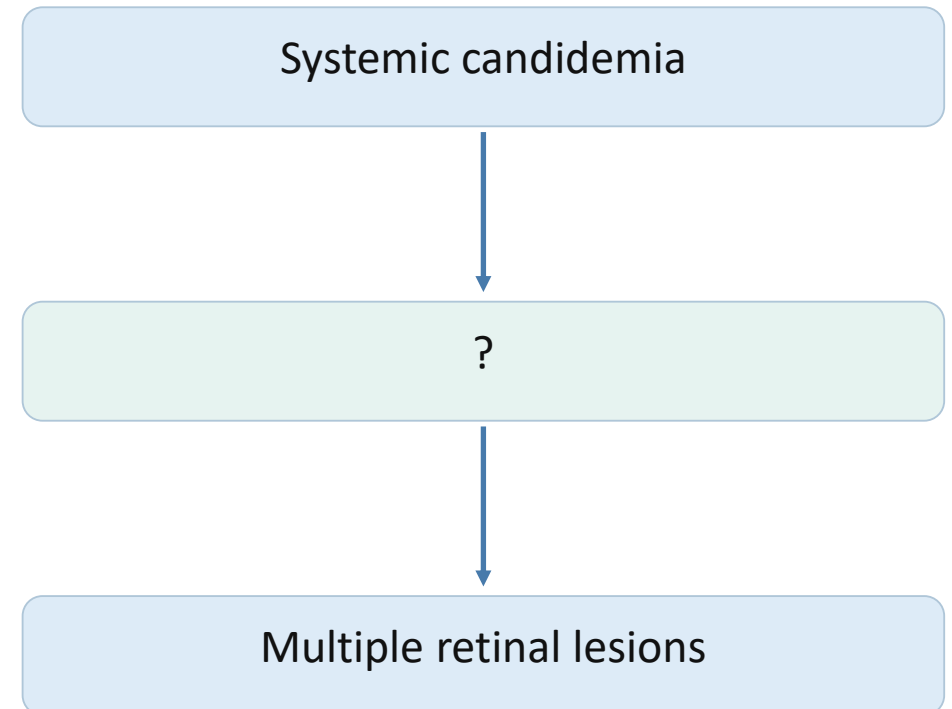
SCHEMATIC



Case: Multifocal retinal inflammation

- A patient has bloodstream Candida infection and multiple separate retinal inflammatory foci.
- The ocular surface has no reported penetrating wound.
- What mechanism most directly explains the retinal distribution?

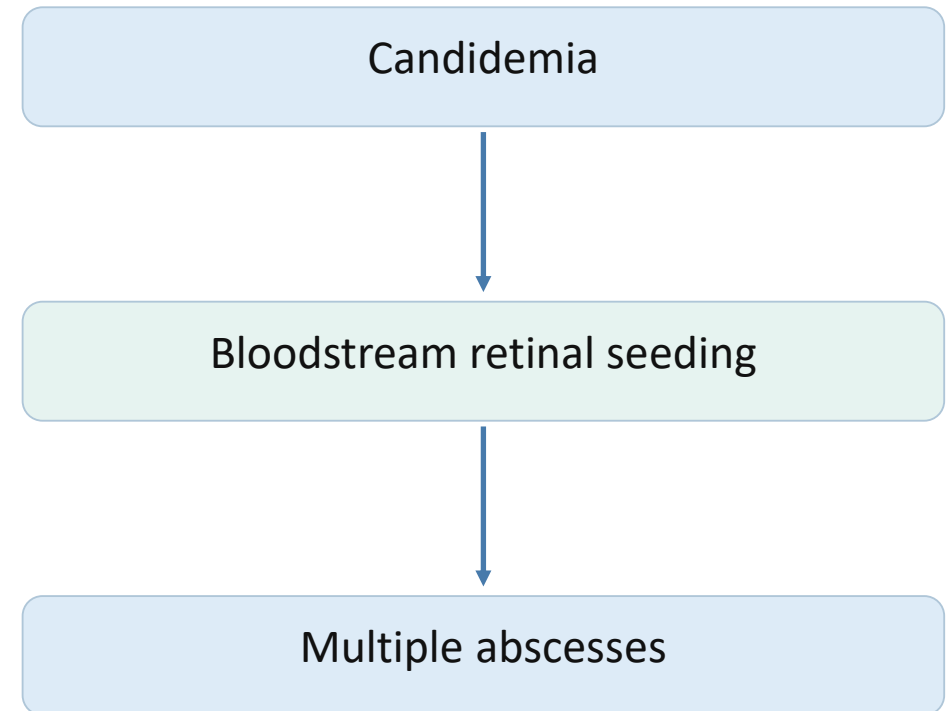
SCHEMATIC



Case analysis: Candida retinitis

- Most consistent mechanism: Hematogenous retinal seeding.
- Dissemination can create multiple retinal abscesses.
- Further clinical evaluation must assess spread beyond retina into vitreous.

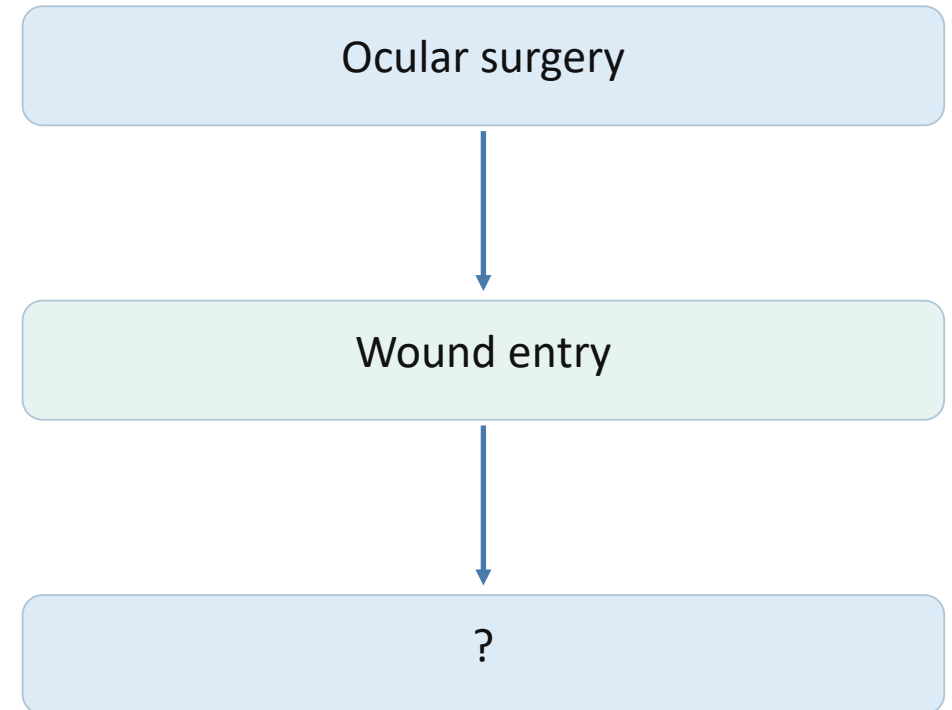
SCHEMATIC



Case: Rapid intraocular inflammation after surgery

- A patient develops a severe intraocular inflammatory syndrome after ocular surgery.
- The suspected organism entered through the surgical wound.
- Which route-based term best categorizes the process?

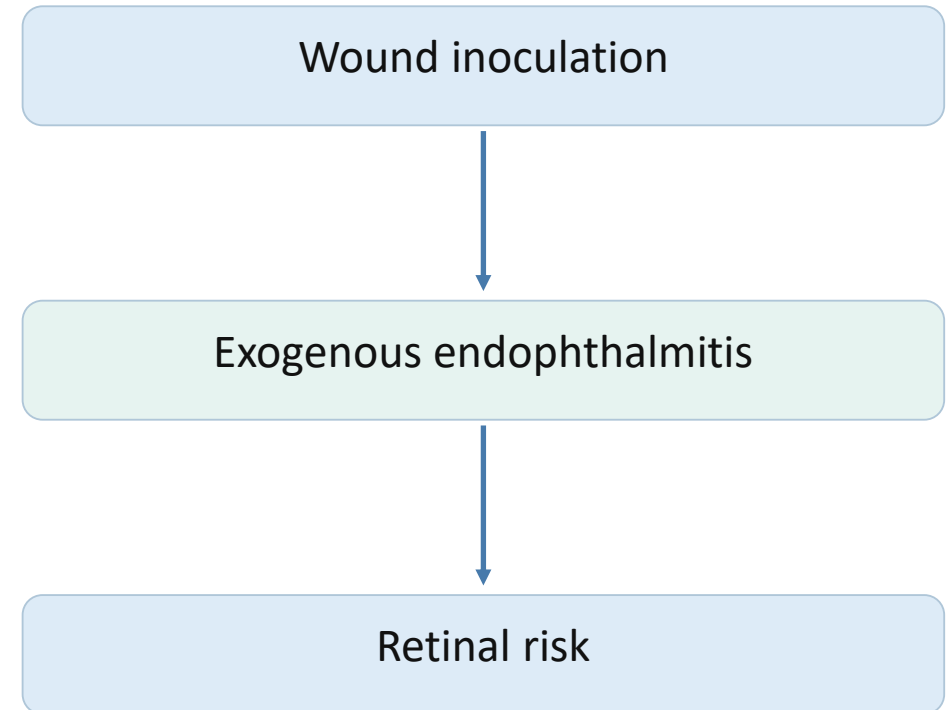
SCHEMATIC



Case analysis: Exogenous endophthalmitis

- The organism arrives from the environment through a surgical opening.
- This is the exogenous route.
- Vitreous suppuration threatens irreversible retinal injury and is time-critical.

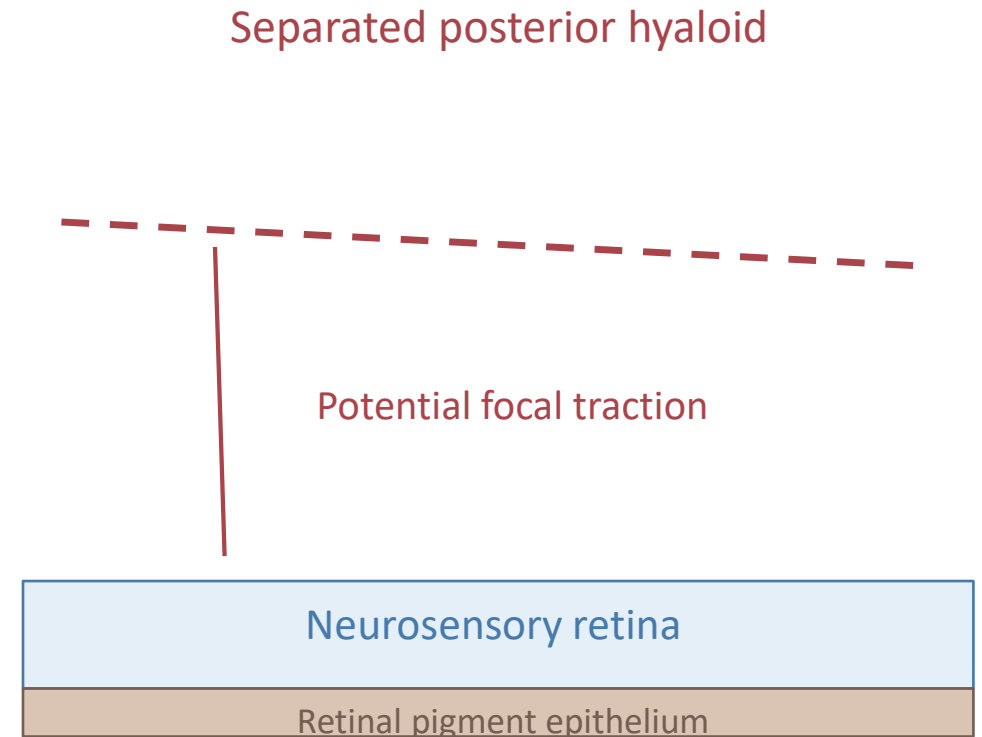
SCHEMATIC



Case: An aging vitreoretinal interface

SCHEMATIC

- An older individual notes new floaters during vitreous structural change.
- Posterior hyaloid separation is detected.
- What nearby anatomical event must remain conceptually distinct?



Case analysis: Vitreous versus retinal detachment

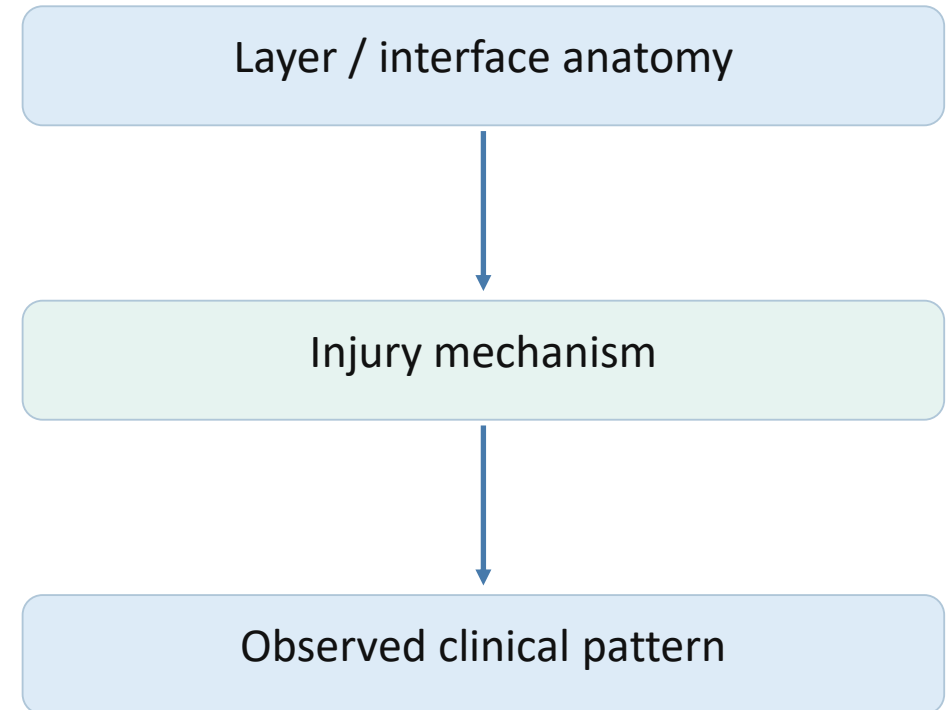
- Posterior vitreous detachment is hyaloid separation from retinal surface.
- Retinal detachment is neurosensory separation from RPE.
- Focal hyaloid traction can contribute to a retinal tear, linking the two events.

Event	Separation plane
PVD	Hyaloid–retinal surface
Retinal detachment	Neurosensory retina–RPE

Integrating the pathology: Structure → pattern

- Retinal layers explain superficial versus deep hemorrhage shape.
- RPE and photoreceptor contact explains vulnerability at the outer interface.
- Posterior hyaloid mechanics explain traction and hemorrhage.
- Vitreous proximity explains urgency of suppurative infection.

SCHEMATIC



Core concepts for clinicopathologic practice

- Identify the retinal layer before interpreting lesion shape.
- Distinguish hyaloid separation from neurosensory retinal detachment.
- Use the route of entry to classify endophthalmitis.
- Recognize that purulent vitreous inflammation can rapidly destroy neural retina.

SCHEMATIC

Vitreous / internal limiting membrane
Nerve fiber layer
Ganglion cell layer
Inner plexiform layer
Inner nuclear layer
Outer plexiform layer
Outer nuclear layer
External limiting membrane
Rods and cones
Retinal pigment epithelium

Selected references

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition. Elsevier; 2026.
- Textbook sections: Retina and Vitreous; Retinal Detachment; Diabetes Mellitus (retinal pathology).