

Retinal Detachment

Mechanisms, morphology, and clinicopathologic interpretation

Pathology • Retinal anatomy • Mechanism

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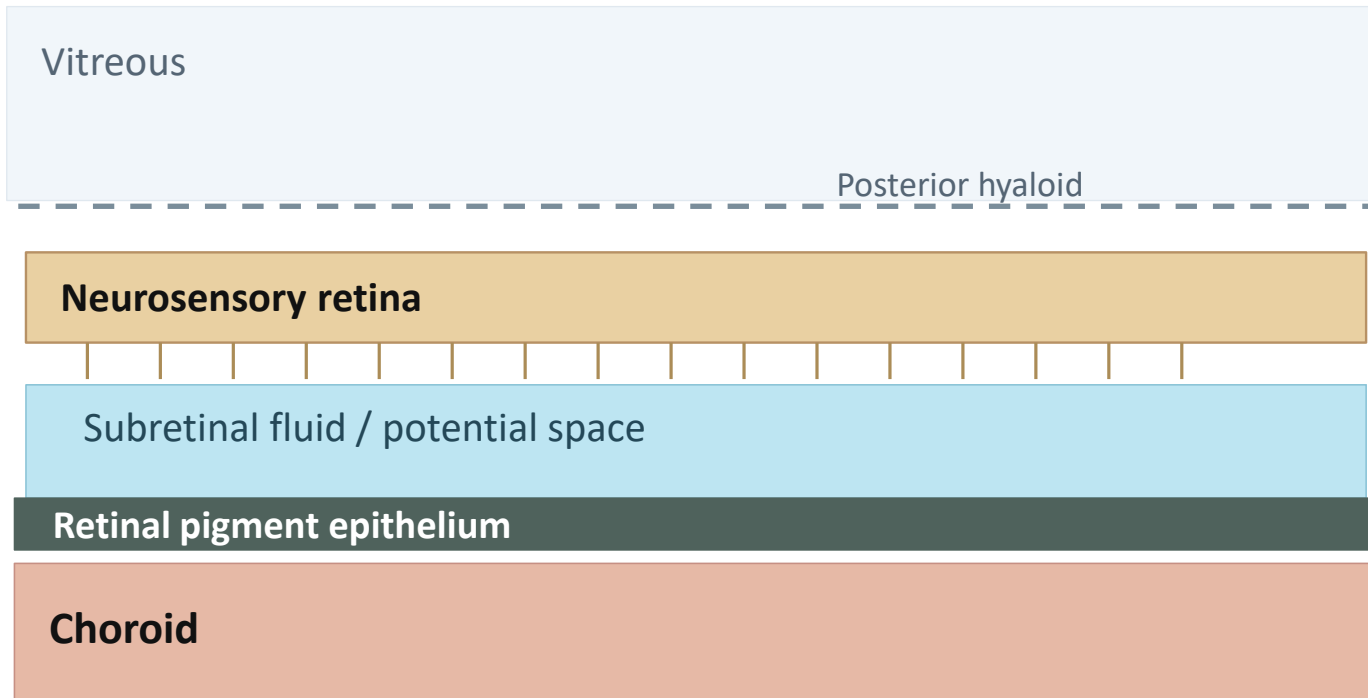
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Learning outcomes

- Define retinal detachment anatomically as separation of neurosensory retina from retinal pigment epithelium.
- Distinguish retinal-break, exudative, and tractional mechanisms using the retinal interface.
- Relate vitreous change and posterior hyaloid traction to full-thickness retinal tears.
- Explain chronic photoreceptor changes, proliferative vitreoretinopathy, and therapeutic principles.
- Apply the mechanism-based differential to retinal vascular disease and clinical presentations.

Retinal detachment: The defining tissue plane

Schematic — tissue planes (not a clinical image)

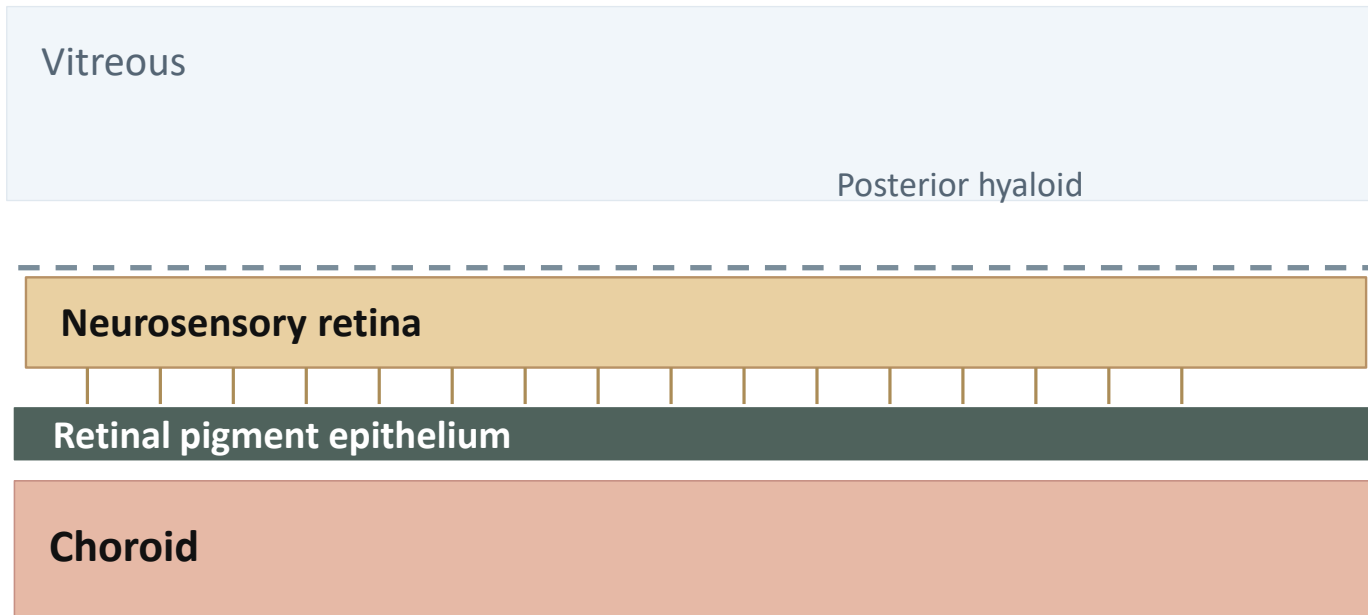


Reading the schematic

- The neurosensory retina separates from the retinal pigment epithelium (RPE).
- The space between the two structures becomes occupied by fluid.
- The identity of the mechanism—not merely separation—determines the classification.

Normal orientation: Vitreous, retina, RPE, choroid

Schematic — tissue planes (not a clinical image)



Reading the schematic

- The internal limiting membrane borders the vitreous cavity.
- The neurosensory retina contains the photoreceptor apparatus.
- The RPE forms the outer interface; The choroid lies beyond it.

Why the retina is vulnerable to separation

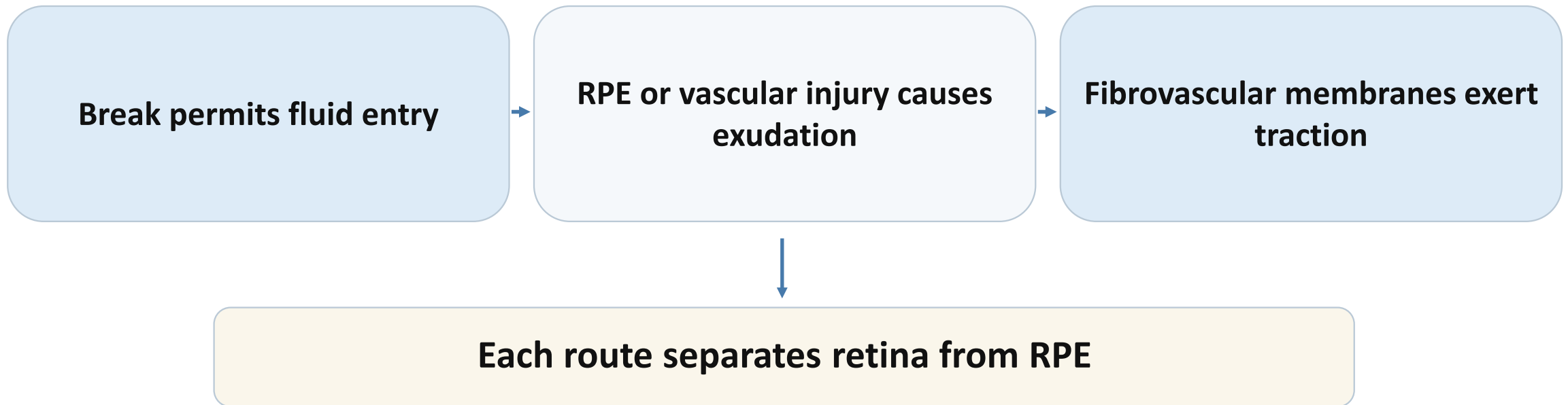
- Neurosensory retina and RPE are closely apposed but distinct embryologic layers.

- The RPE supports maintenance of photoreceptor outer segments.

- Loss of the interface interrupts that normal structural relationship.

- Longer-standing detachment may be accompanied by loss of photoreceptor outer segments.

A mechanism-based framework



- Robbins groups disease by the presence or absence of a retinal break.
- Clinically, traction is also distinguished as an important non-break mechanism.

Two textbook categories: Retinal break present or absent

Rhegmatogenous

- Full-thickness retinal defect
- Liquefied vitreous enters through tear
- Posterior hyaloid traction may initiate break

Nonrhegmatogenous

- No retinal break required
- Exudation or RPE injury permits fluid collection
- Traction may also separate retina without a break

Clinical mechanism categories and textbook terminology

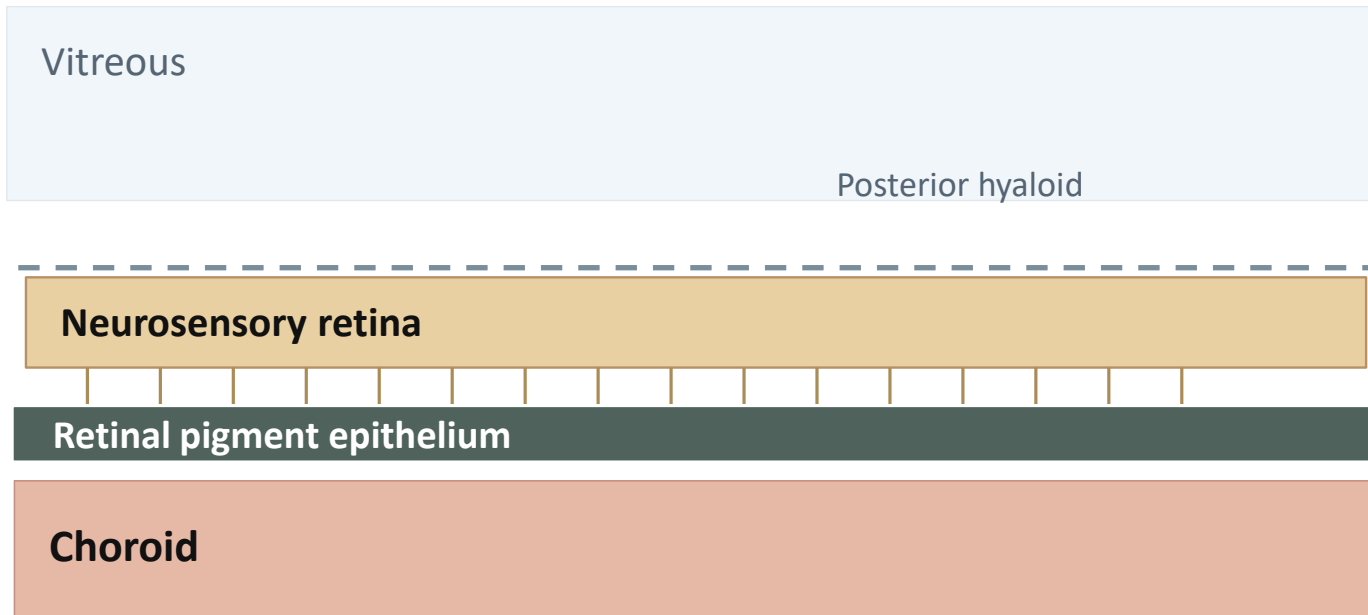
Clinical descriptor	Essential mechanism	Robbins grouping
Rhegmatogenous	Full-thickness break with vitreous access	Rhegmatogenous
Exudative / serous	Fluid accumulates without a break	Nonrhegmatogenous
Tractional	Contracting fibrovascular membrane	Usually nonrhegmatogenous initially
Combined	Traction produces a secondary break	Both mechanisms can coexist

The break is a pathologic discriminator

- A full-thickness defect directly communicates the vitreous cavity with the subretinal space.
- A detached retina with no break requires investigation of other driving mechanisms.
- Strong traction can convert an initially non-break detachment into a combined detachment.

The RPE–photoreceptor interface

Schematic — tissue planes (not a clinical image)



Reading the schematic

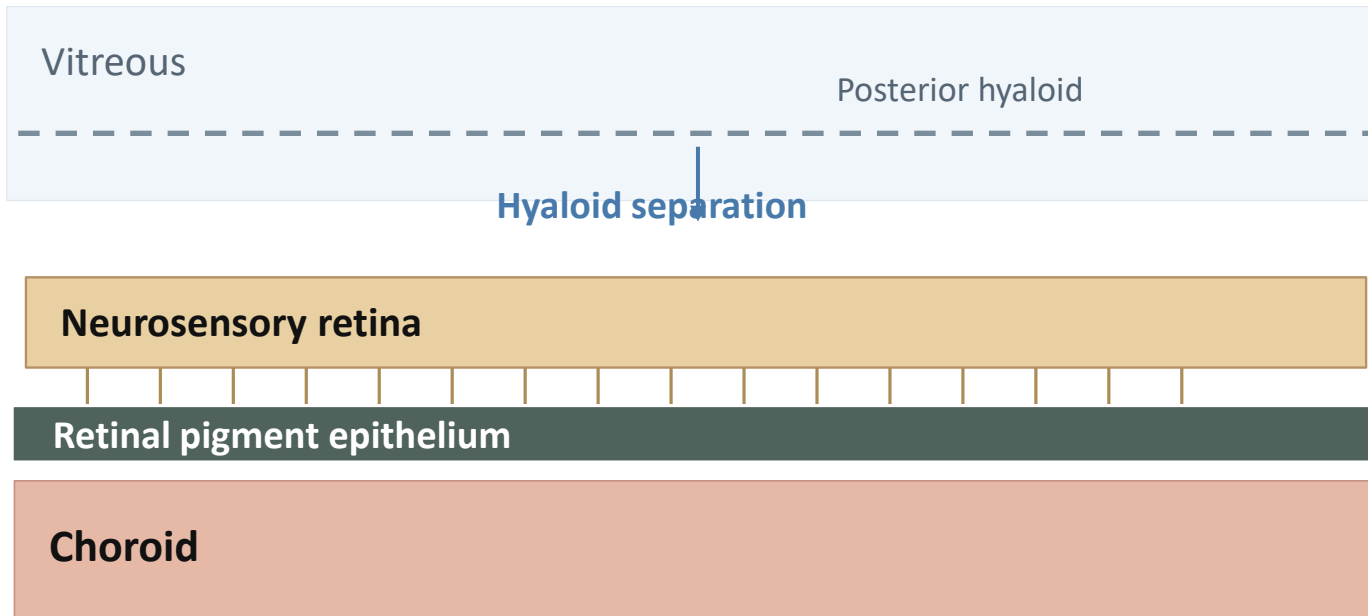
- Photoreceptors lie on the RPE side of the neurosensory retina.
- RPE integrity is essential for maintenance of photoreceptor outer segments.
- Detachment separates photoreceptors from the RPE support surface.

Retinal response to injury

- The neurosensory retina is derived from the diencephalon.
- Like neural tissue, injured retina may respond through gliosis.
- Retinal glial cells contribute to membranes seen in proliferative vitreoretinopathy.
- This reparative response can become mechanically detrimental.

Vitreous aging changes the retinal interface

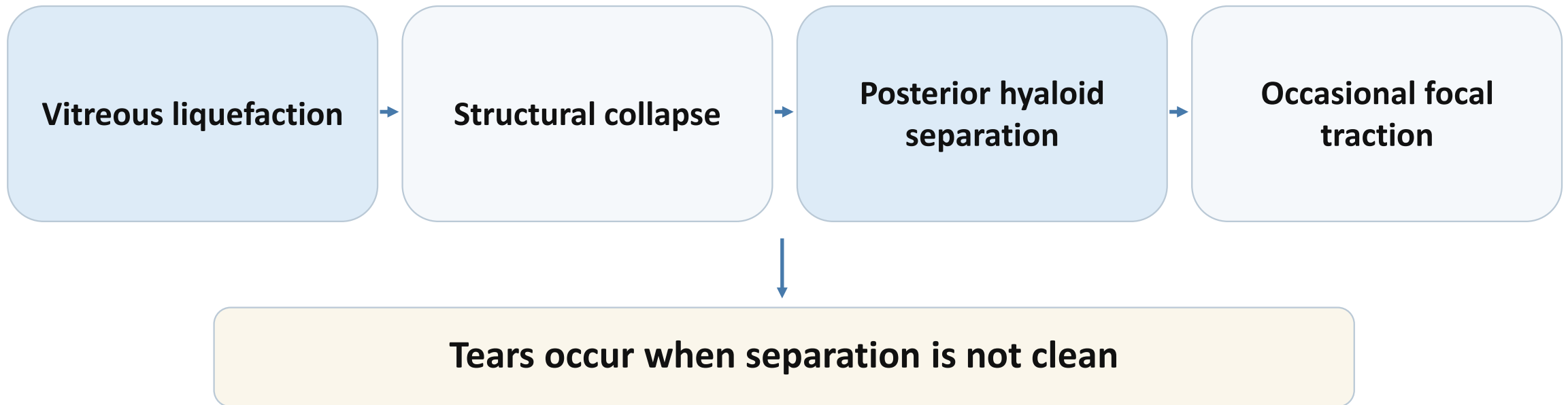
Schematic — tissue planes (not a clinical image)



Reading the schematic

- The adult vitreous humor is avascular.
- Aging vitreous may liquefy and structurally collapse.
- The posterior vitreous face (posterior hyaloid) may separate from retina.

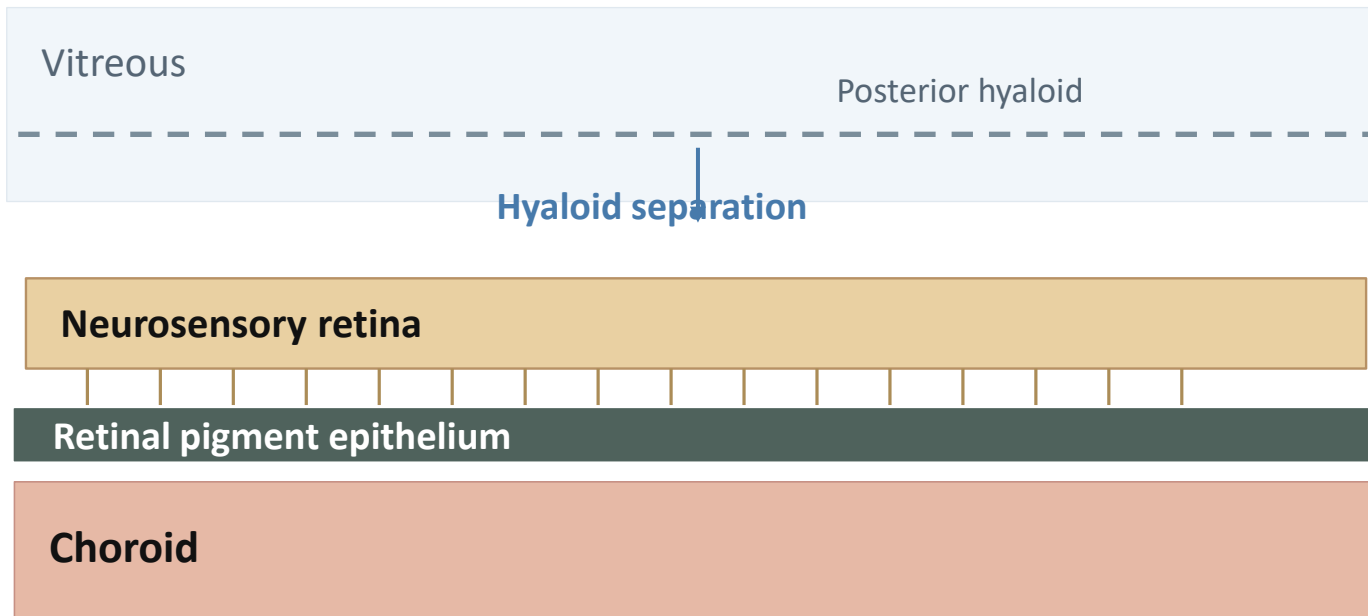
Aging vitreous: The critical sequence



- Posterior vitreous detachment (PVD) is common with aging.
- PVD is not itself equivalent to retinal detachment.

Posterior vitreous detachment is not retinal detachment

Schematic — tissue planes (not a clinical image)



Reading the schematic

- PVD: Posterior hyaloid separates from the internal limiting membrane.
- Retinal detachment: Neurosensory retina separates from the RPE.
- These are anatomically distinct tissue planes.

Clean versus incomplete hyaloid separation

Clean PVD

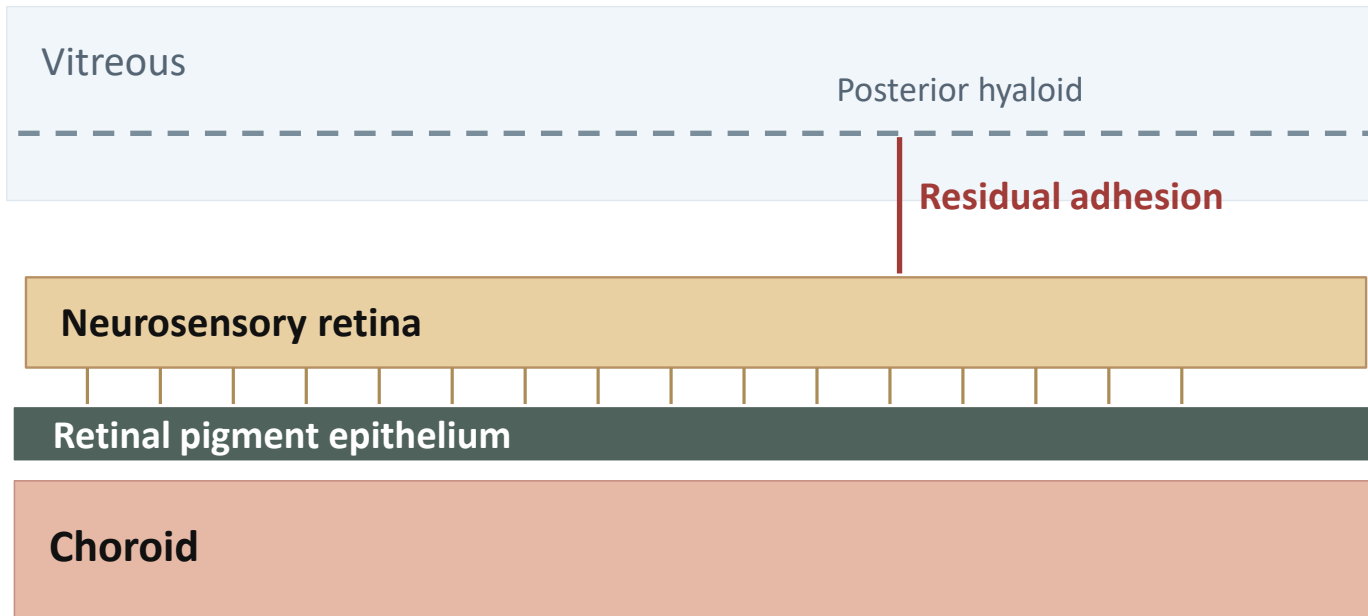
- Hyaloid detaches from retinal surface
- No tear is inherently implied
- Retina remains attached to RPE

Focally adherent PVD

- Strong focal vitreoretinal adhesion persists
- Hyaloid movement pulls retina locally
- Full-thickness retinal tear may follow

Focal adhesion converts movement into traction

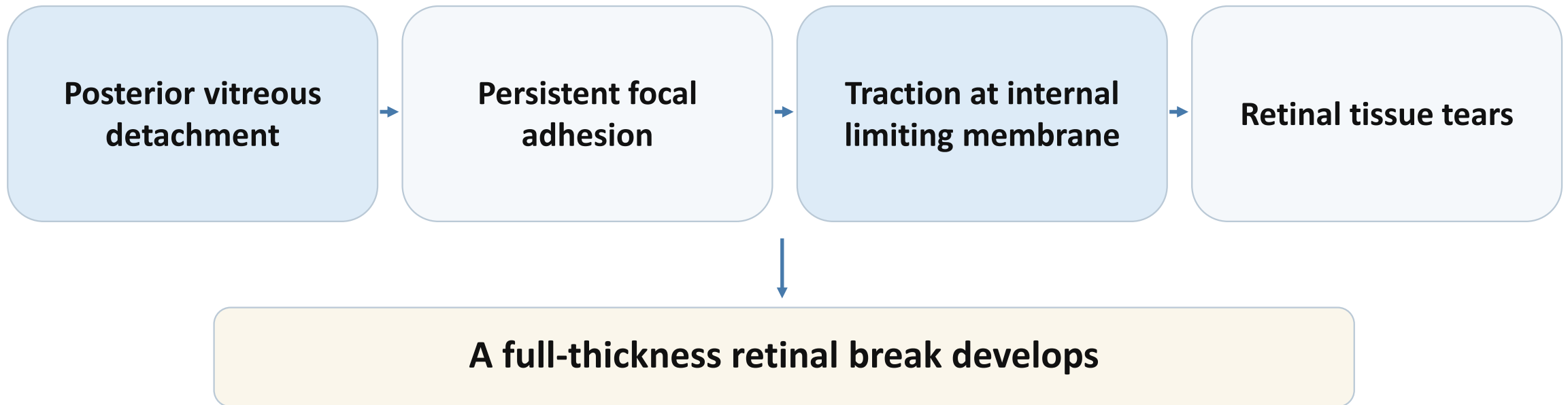
Schematic — tissue planes (not a clinical image)



Reading the schematic

- The posterior hyaloid remains attached at a point of unusually strong adhesion.
- Separation elsewhere transmits force to the remaining attachment.
- Traction is concentrated in the retinal tissue at that point.

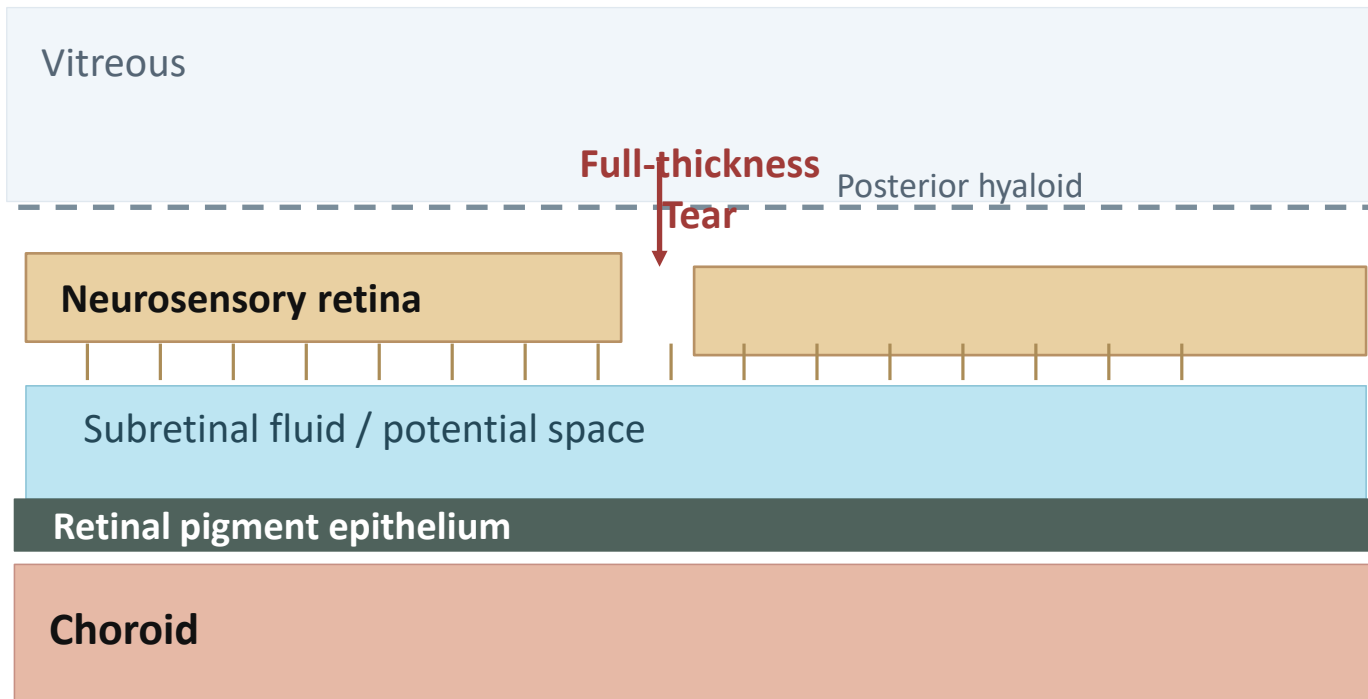
From traction to a full-thickness retinal tear



- The tear creates a pathway for liquefied vitreous.
- Tissue-plane disruption precedes the fluid-mediated detachment.

Rhegmatogenous detachment: Fluid enters via a break

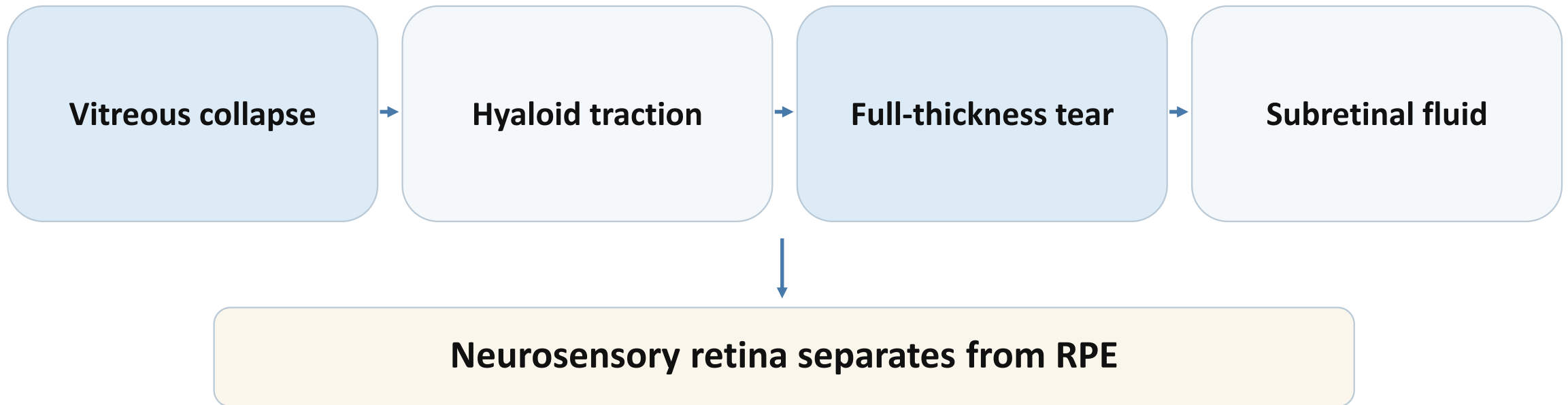
Schematic — tissue planes (not a clinical image)



Reading the schematic

- The tear spans the full retinal thickness.
- Liquefied vitreous traverses the retinal defect.
- Fluid collects between neurosensory retina and RPE.

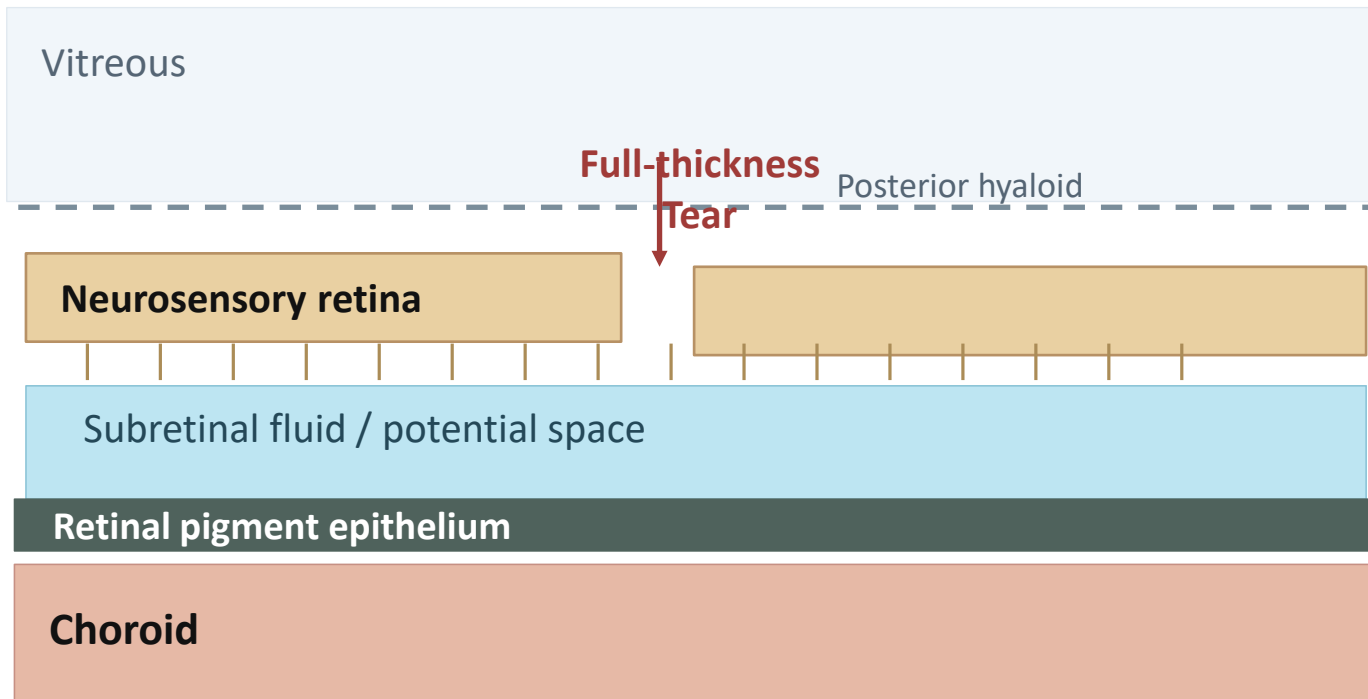
Rhegmatogenous detachment: Four linked events



- A break and fluid movement together explain the detachment.
- Removing traction is a central surgical principle.

Fluid access determines the separation plane

Schematic — tissue planes (not a clinical image)



Reading the schematic

- The retinal defect opens the vitreous compartment to the potential subretinal space.
- The RPE itself need not be the primary site of injury.
- Fluid undermines the detached neurosensory retina.

Retinal tear versus retinal detachment

Retinal tear

- Full-thickness focal defect
- May become an entry point for fluid
- Anatomically a break in neurosensory tissue

Retinal detachment

- Separation of retina from RPE
- Subretinal fluid occupies the intervening space
- May extend away from original break

Break recognition: Clinical significance

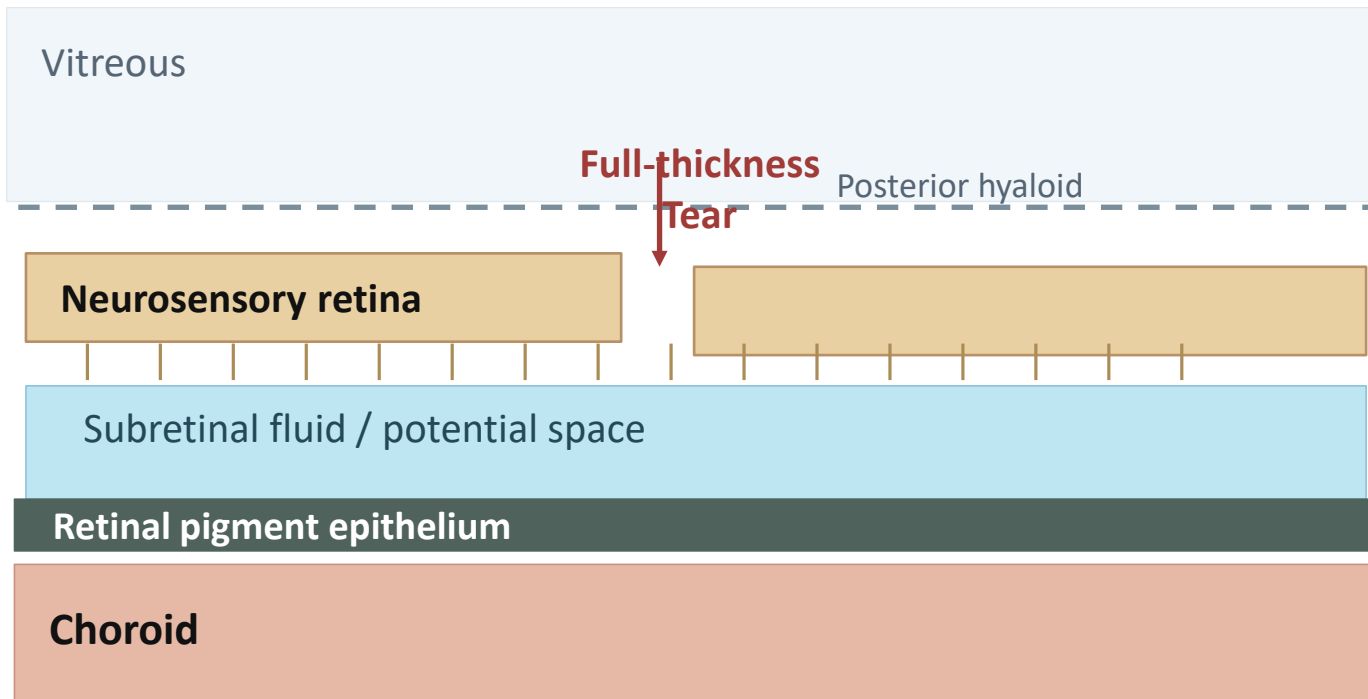
- Finding a full-thickness retinal break favors rhegmatogenous detachment.
- An apparent small break may explain a more extensive separation.
- The clinical goal is to locate retinal breaks and assess the extent of detachment.

Vitreoretinal concepts that must not be conflated

Structure or event	Plane involved	Immediate significance
Posterior hyaloid	Between vitreous and internal limiting membrane	Can exert traction during PVD
Retinal tear	Across neurosensory retinal thickness	Provides access for vitreous fluid
Retinal detachment	Between retina and RPE	Disrupts photoreceptor–RPE interface
Subretinal fluid	Retina–RPE potential space	Maintains physical separation

Acute rhegmatogenous detachment: Outer segments persist

Schematic — tissue planes (not a clinical image)

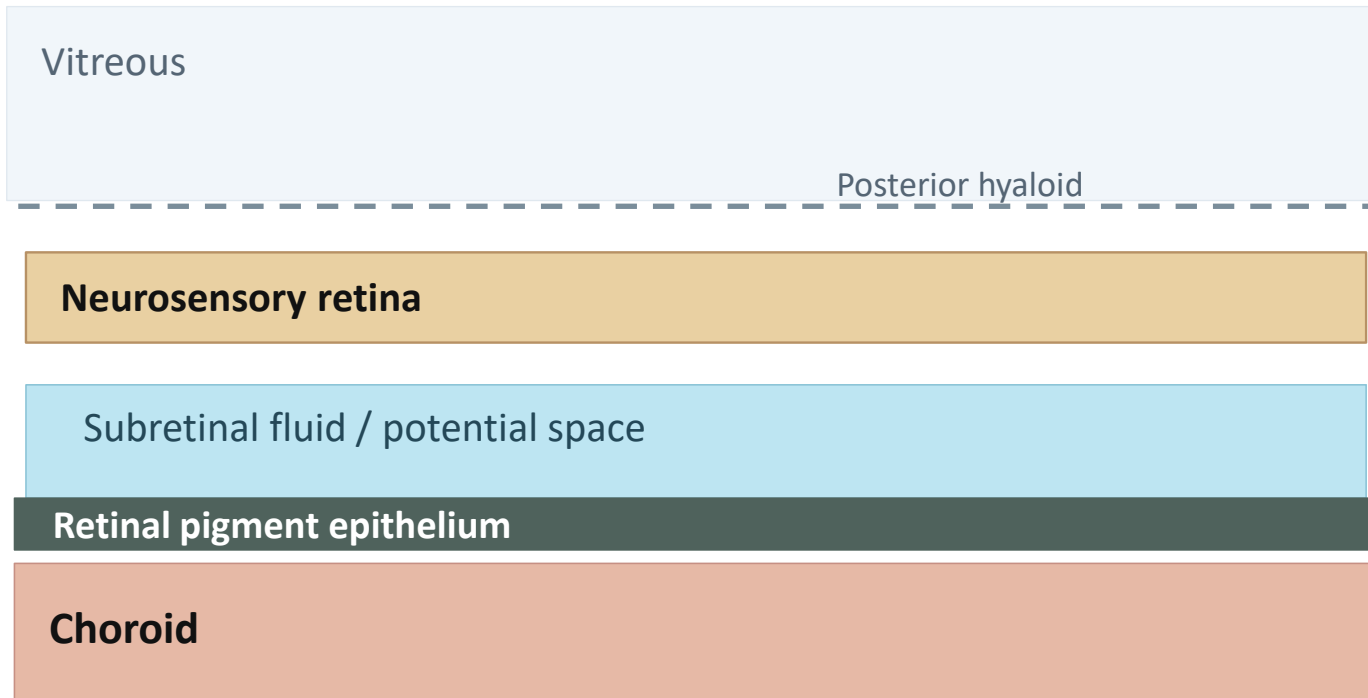


Reading the schematic

- Robbins Fig. 29.17 depicts intact photoreceptor outer segments in an acute example.
- The retina is already separated even while the outer segments remain.
- Preserved outer segments provide a temporal clue—not a different detachment class.

Chronic retinal detachment: Outer segments are lost

Schematic — tissue planes (not a clinical image)



Reading the schematic

- The figure legend describes loss of photoreceptor outer segments in chronic detachment.
- This chronic change can occur in both rhegmatogenous and nonrhegmatogenous disease.
- Do not infer the mechanism from chronicity alone.

Acute versus chronic structural findings

Acute illustrated example

- Neurosensory retina separated
- Photoreceptor outer segments intact
- Textbook illustration uses break-associated example

Chronic illustrated example

- Neurosensory retina separated
- Photoreceptor outer segments missing
- Finding is not specific for detachment mechanism

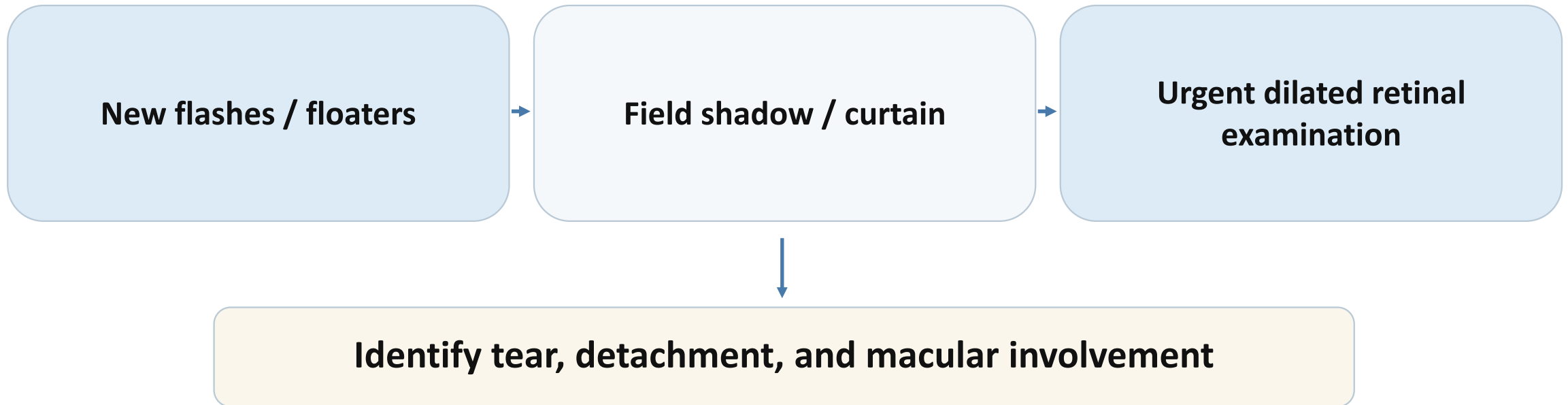
Why duration modifies photoreceptor morphology

- RPE is needed to maintain photoreceptor outer segments.
- Persistent separation interrupts that interface.
- Outer-segment loss is consequently a morphologic sign of chronicity.
- This is a structural principle, not a quantitative prediction of visual recovery.

Clinical warning symptoms

- New flashes of light can accompany vitreoretinal traction.
- A sudden increase in floaters requires prompt assessment.
- A new shadow or curtain in the visual field suggests retinal involvement.
- Absence of symptoms does not exclude a small peripheral detachment.

Symptoms to urgent retinal examination



- Retinal detachment is treated as a medical emergency.
- This is supplementary National Eye Institute clinical guidance.

The dilated examination establishes anatomy

- Inspect the retinal surface and periphery for breaks.
- Determine whether neurosensory retina is elevated from RPE.
- Assess coexisting traction, vascular disease, or choroidal lesions.
- A symptom pattern alone cannot determine the mechanism.

Imaging: What each assessment clarifies

Assessment	Main clinical question
Dilated fundus examination	Is there a retinal tear or detached retina?
Ocular ultrasonography	Where is the retina when the fundus view is inadequate?
Optical coherence tomography	How are retinal layers and the macular interface positioned?

Morphology can suggest the mechanism

Rhegmatogenous pattern

- Retinal break may be visible
- Mobile or corrugated elevation may occur
- Fluid enters from vitreous through break

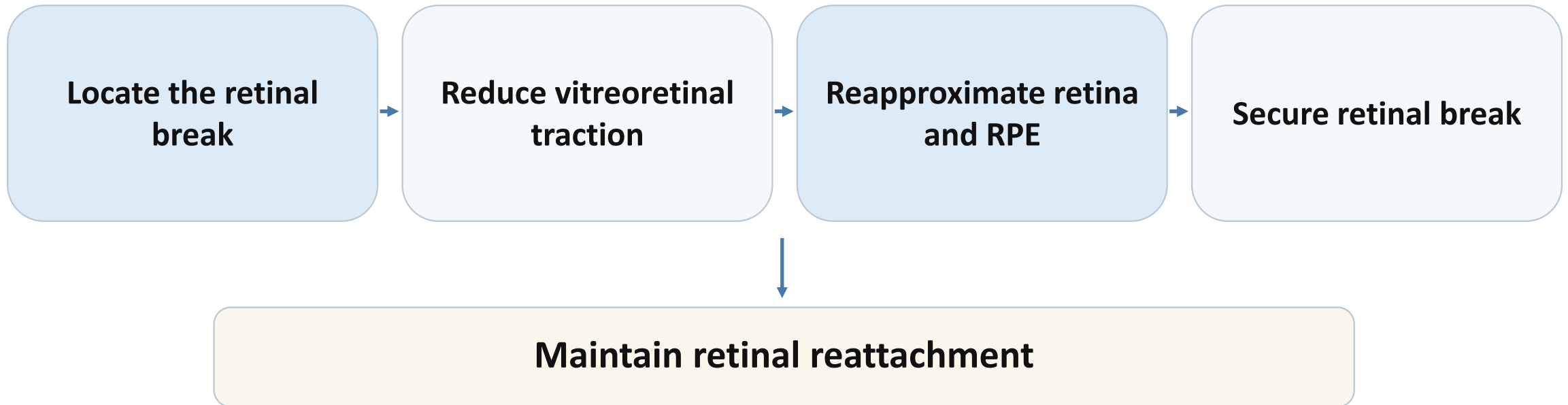
Tractional / exudative patterns

- Tractional: Membrane-related elevation
- Serous: Smooth elevation from subretinal exudate
- A break may be absent or hidden

Do not overread the appearance

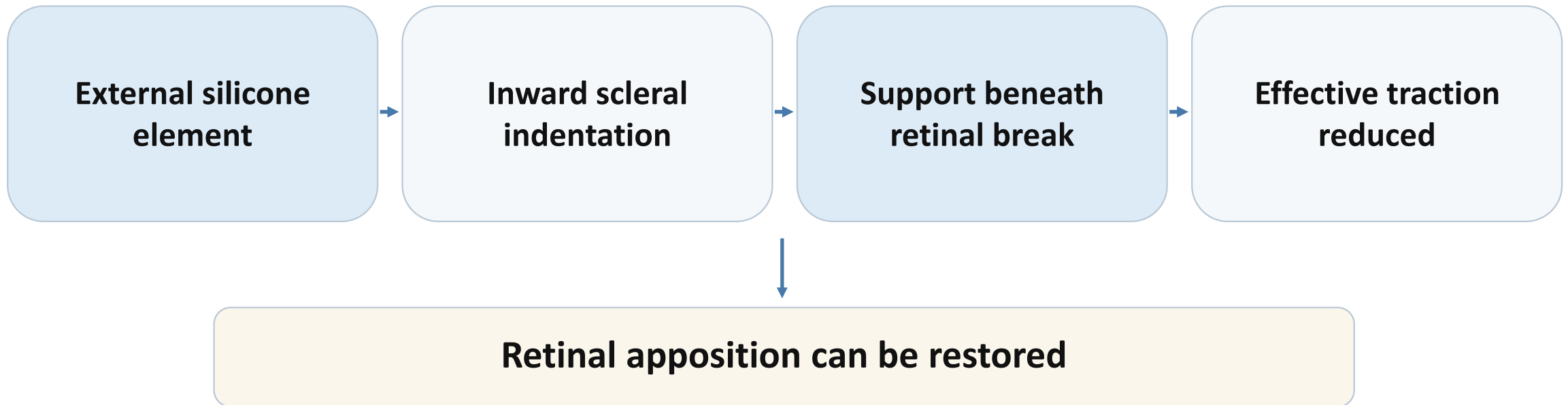
- A retinal break can be difficult to identify when media are opaque.
- An observed elevation is not sufficient to specify its cause.
- Integrate the retinal surface, choroid, vascular findings, and vitreous state.

The therapeutic objective in rhegmatogenous disease



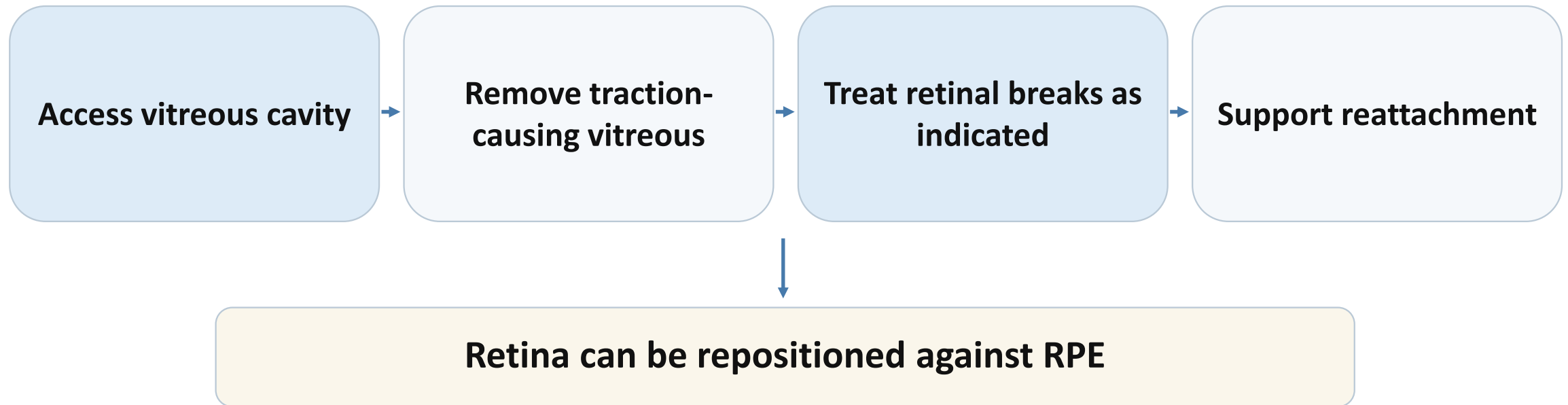
- The primary Robbins mechanism is mechanical relief of vitreous traction.
- Clinical procedures vary with anatomy and detachment extent.

Scleral buckling: Change the geometry of traction



- The buckle acts on the outer globe rather than replacing the vitreous.
- Anatomic indentation is the mechanism emphasized.

Vitrectomy addresses the vitreous component



- Removal of vitreous as a complementary approach.
- Other surgical details are supplementary clinical context.

Retinal repair techniques: Their distinct roles

Technique	Core action	Clinical context
Scleral buckle	External scleral indentation	Relieves traction; Supports a break
Vitrectomy	Removes vitreous and traction	Repairs detachment from within the globe
Laser or cryopexy	Creates adhesion around retinal break	Treats tears; May accompany reattachment
Pneumatic retinopexy	Uses intraocular gas bubble	Selected detachments with appropriate breaks

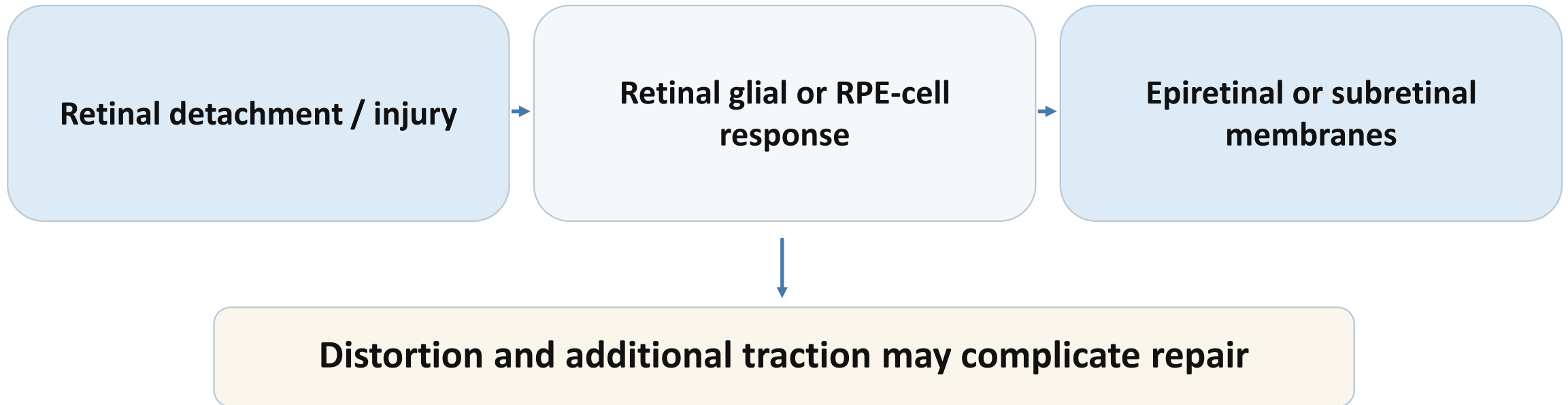
Retinal reattachment is not the only issue

- Traction relief addresses the mechanical initiating factor.
- Retinal break closure limits continued fluid access.
- Proliferative vitreoretinopathy can complicate the repair by creating new membranes.
- The approach is determined by retinal anatomy and associated pathology.

Proliferative vitreoretinopathy: A complicating response

- Rhegmatogenous detachment may be complicated by proliferative vitreoretinopathy (PVR).
- Cell proliferation forms membranes on or below the retinal surface.
- This is an adverse consequence of tissue injury and repair.

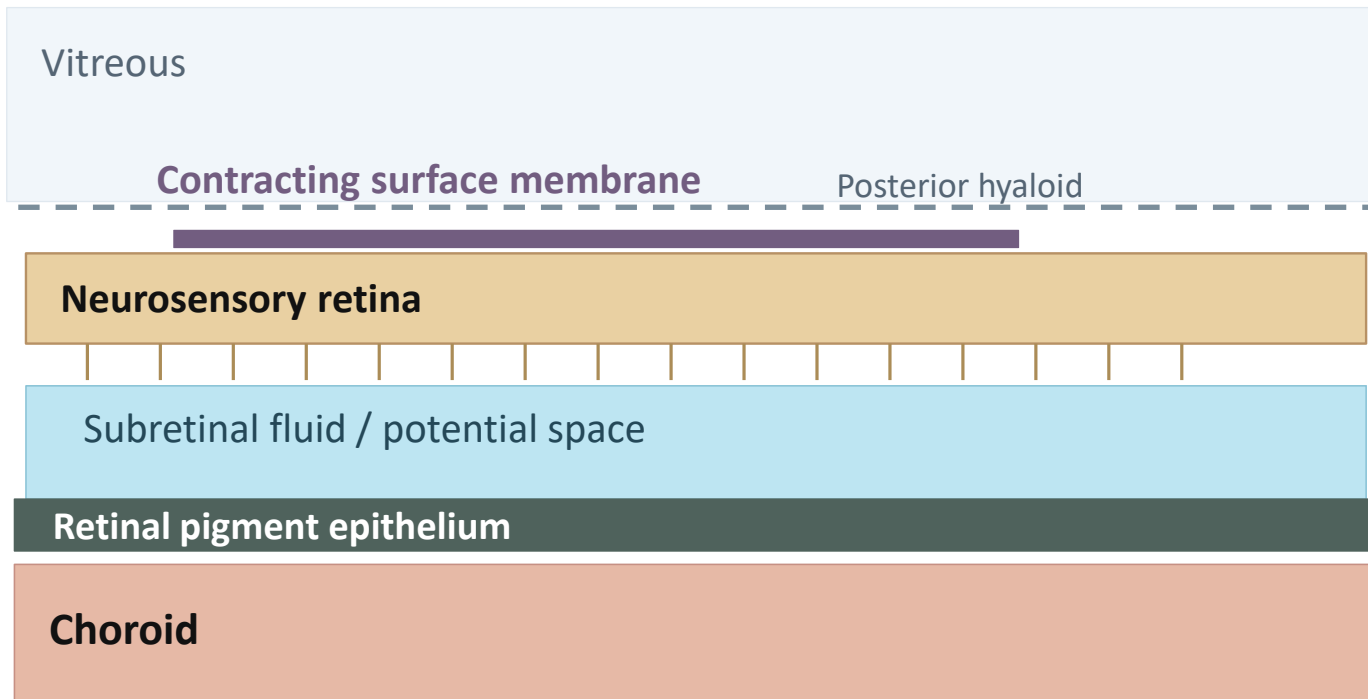
Cells and membranes in PVR



- Glial cells and RPE cells as membrane-forming populations.
- The compartment occupied by membranes is diagnostically informative.

Epiretinal and subretinal membrane locations

Schematic — tissue planes (not a clinical image)



Reading the schematic

- Epiretinal: Membrane is positioned on the vitreous-facing retinal surface.
- Subretinal: Membrane is located between neurosensory retina and RPE.
- The same broad proliferative process can affect either compartment.

Primary rhegmatogenous event versus PVR

Initiating detachment

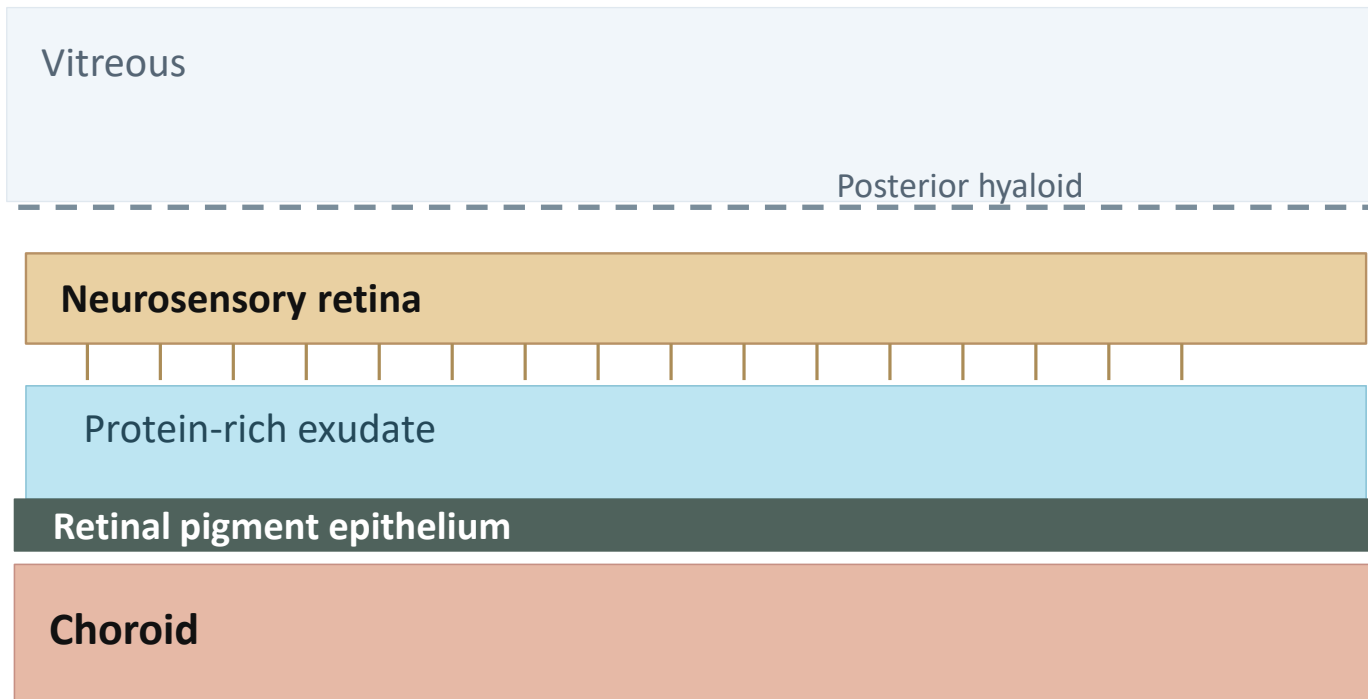
- Hyaloid traction creates a tear
- Liquefied vitreous enters subretinal plane
- Break drives initial fluid access

PVR complication

- Glial or RPE-cell membranes develop
- New tractional or structural obstacles appear
- May complicate surgical reattachment

Nonrhegmatogenous detachment: No tear is necessary

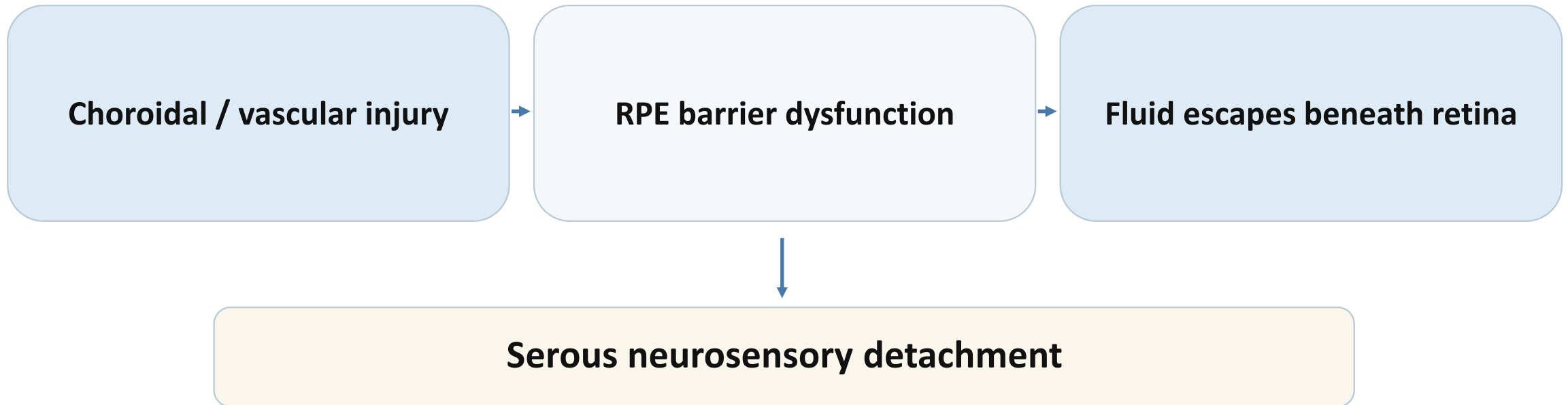
Schematic — tissue planes (not a clinical image)



Reading the schematic

- Fluid accumulates under neurosensory retina without crossing a retinal break.
- RPE injury can permit entry of fluid from choroidal circulation.
- Exudative retinal vascular disorders are another recognized cause.

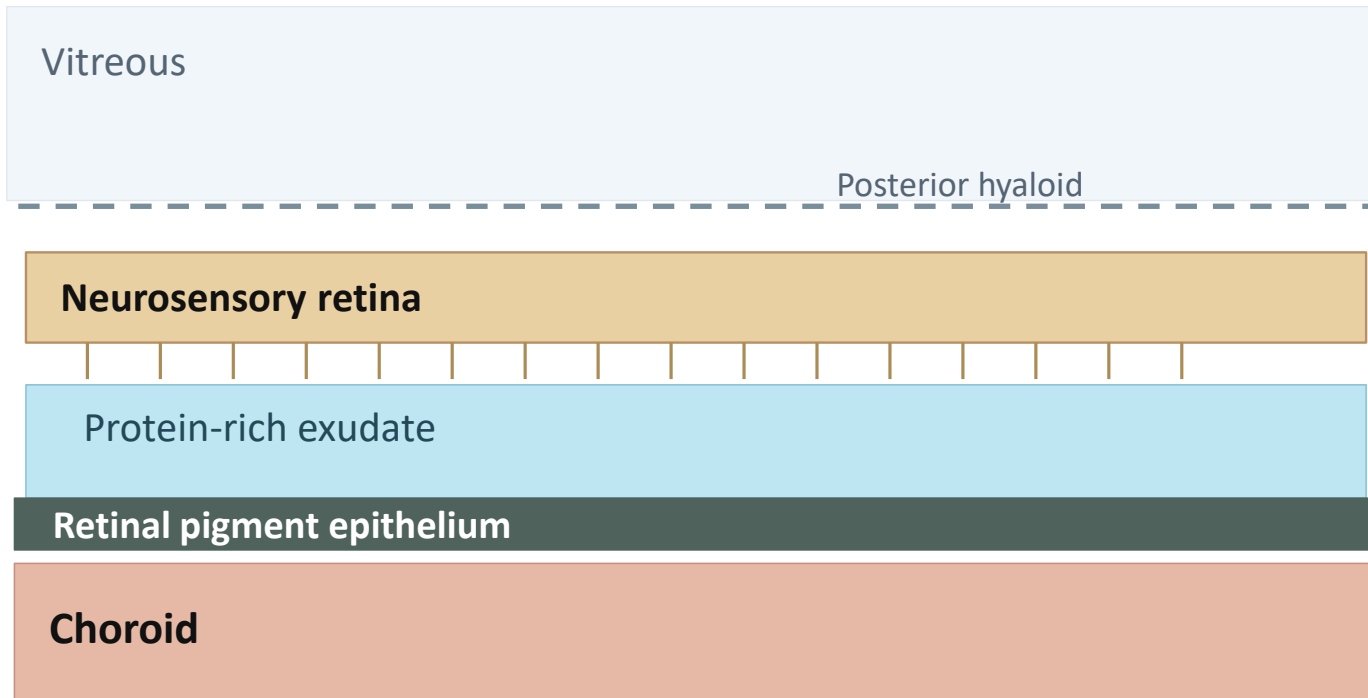
An exudative mechanism begins outside the vitreous



- The source of subretinal fluid differs from rhegmatogenous disease.
- Treating a hypothetical tear would not address the initiating event.

Protein-rich fluid occupies the subretinal space

Schematic — tissue planes (not a clinical image)



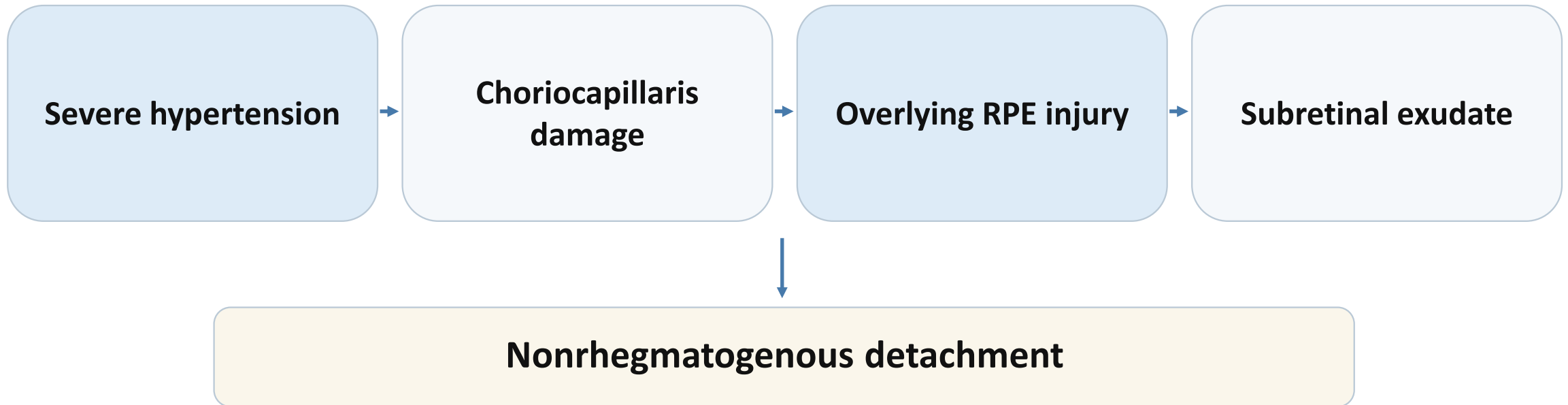
Reading the schematic

- Protein-rich exudate in nonrhegmatogenous detachment.
- The fluid separates retina from RPE while retinal tissue may remain continuous.
- Chronic outer-segment loss can also occur in this category.

Severe hypertension: Choroidal vascular injury

- Severe hypertension can damage retinal and choroidal vessels.
- Choriocapillaris injury may damage overlying RPE.
- Exudate can enter the retina–RPE potential space, causing detachment.

Hypertensive choroidopathy to retinal detachment



- This mechanism is choroid/RPE-driven, not a vitreous tear.
- Choroidal infarcts may be visible clinically as Elschnig spots.

Retinal exudate versus subretinal exudate

Retinal vascular leakage

- Leakage originates from retinal vessels
- Exudate often accumulates in outer plexiform layer
- May create a macular star in severe hypertension

Choroidal / RPE injury

- Leakage originates across injured choroid–RPE interface
- Fluid accumulates between RPE and neurosensory retina
- Can produce exudative detachment

Choroidal tumors: A different source of fluid

- Choroidal tumors are cited among causes of nonrhegmatogenous retinal detachment.
- The primary lesion lies beneath the retina.
- Retinal separation is secondary to disease involving the choroid/RPE interface.
- The detachment must not be mistaken for an isolated primary retinal tear.

Fluid source: Vitreous versus choroid

Rhegmatogenous

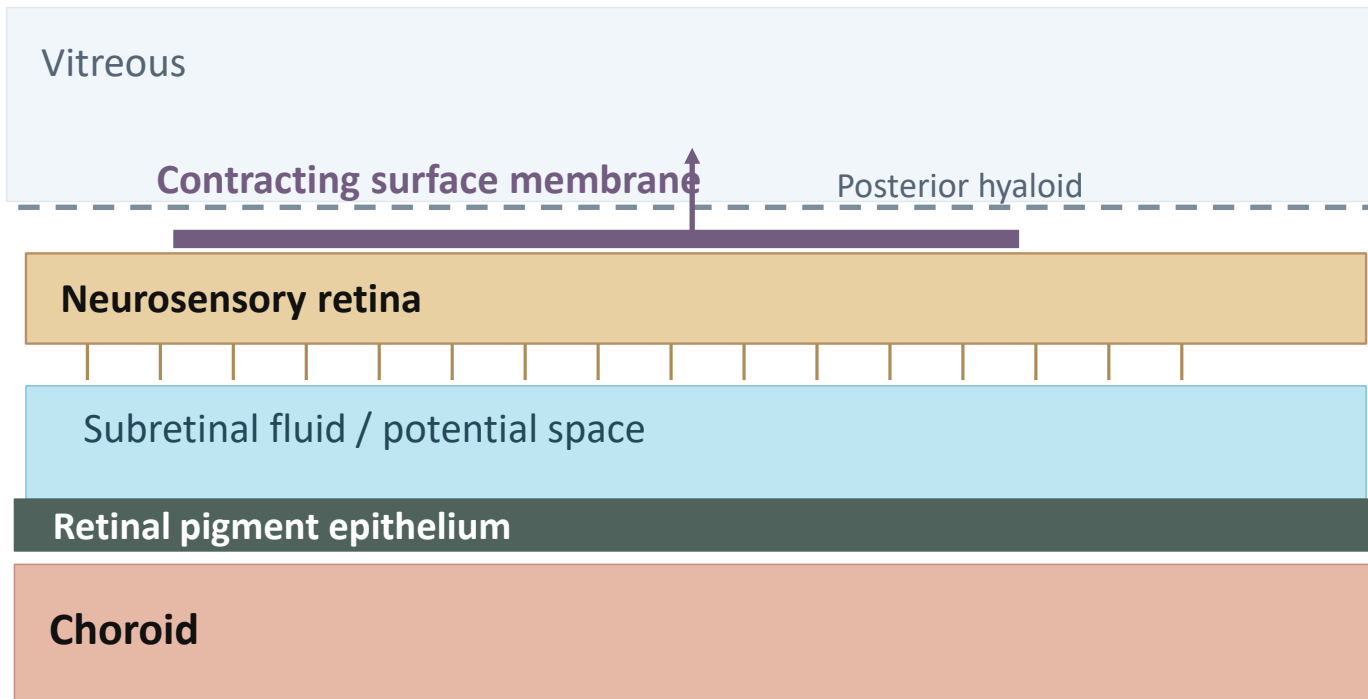
- Requires full-thickness tear
- Fluid arrives from liquefied vitreous
- Posterior hyaloid traction commonly initiates the break

Exudative

- No tear required
- Fluid follows vascular/RPE damage
- Examples: Hypertension, choroidal tumor

Tractional detachment: The retina is physically pulled

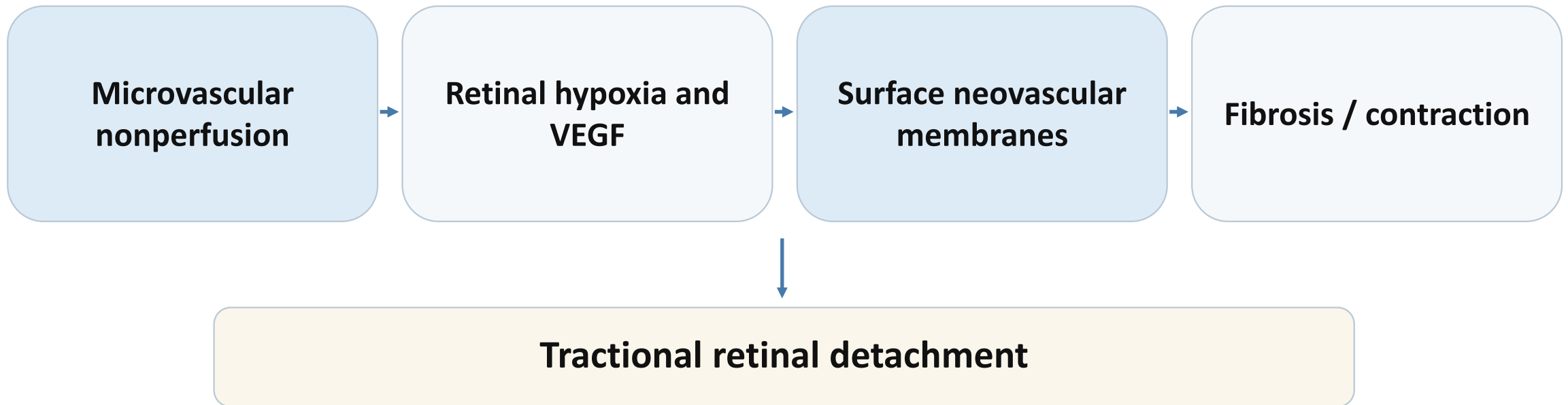
Schematic — tissue planes (not a clinical image)



Reading the schematic

- Fibrovascular membranes can contract against the retinal surface.
- The resulting traction elevates retina away from RPE.
- A tear may be absent initially.

Diabetic microvascular injury leads to traction



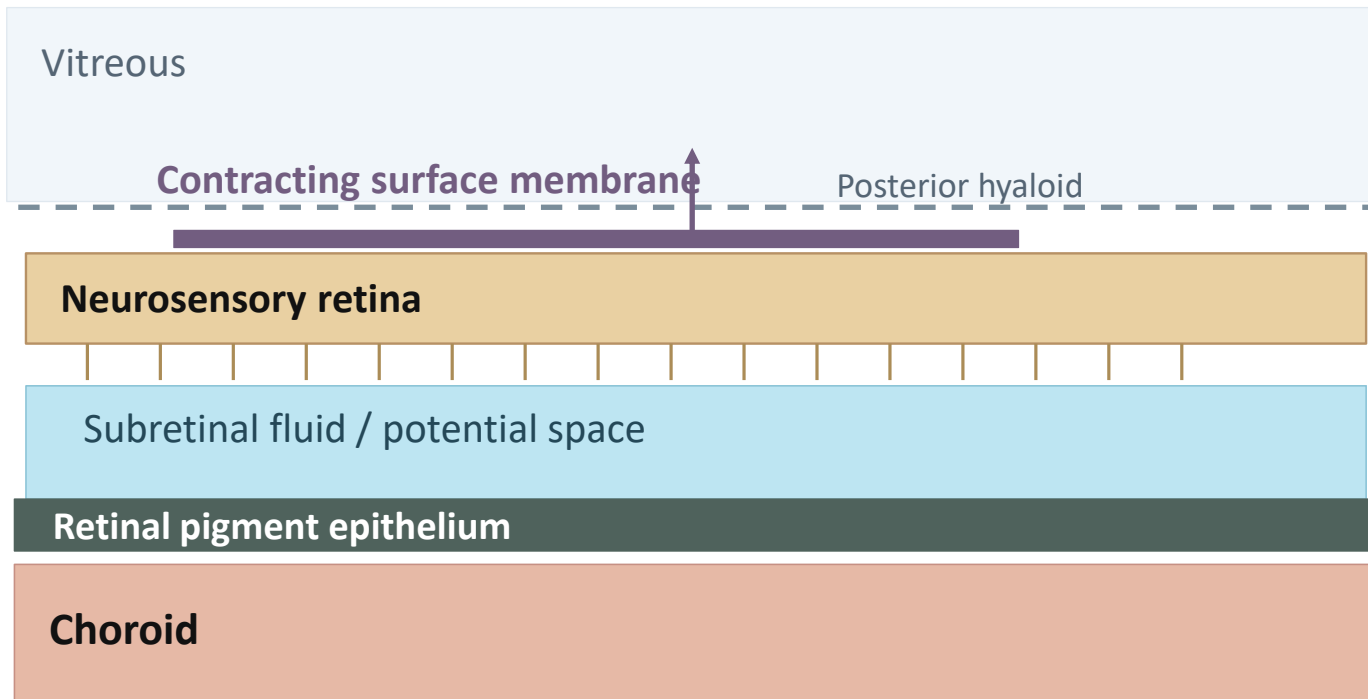
- Robbins specifically describes traction caused by scarring in proliferative diabetic retinopathy.
- This is distinct from primary vitreous entry through a tear.

VEGF connects ischemia and angiogenesis

- Retinal micro-occlusion produces nonperfused tissue.
- Ischemic retina upregulates VEGF.
- New vessels grow in relation to the retinal surface and posterior hyaloid.
- Neovascularization can develop a fibrous or glial scaffold.

The internal limiting membrane is a key boundary

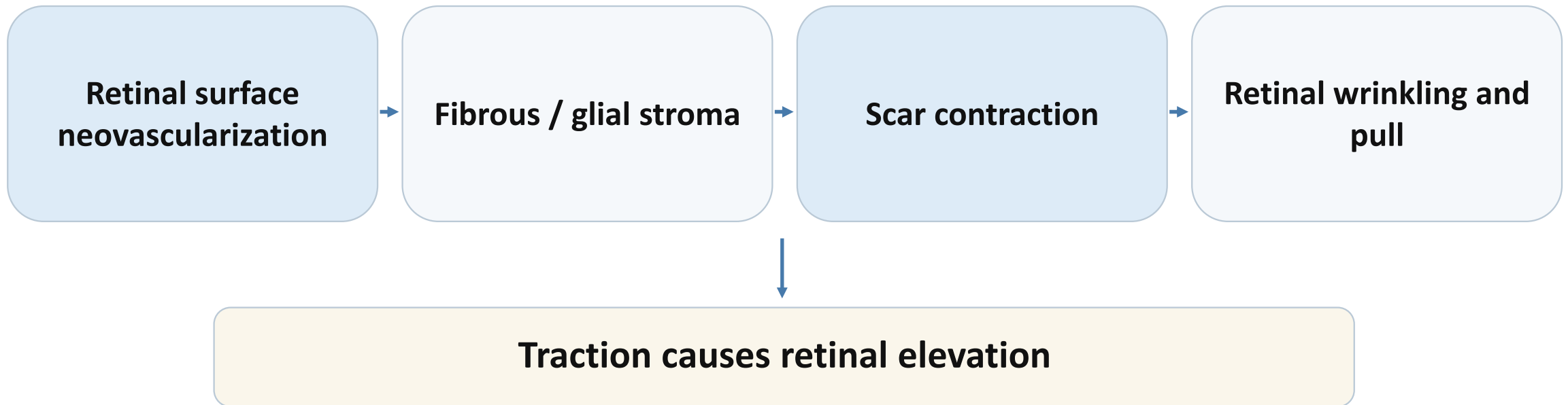
Schematic — tissue planes (not a clinical image)



Reading the schematic

- In proliferative diabetic retinopathy, new vessels extend beyond the retinal internal limiting membrane.
- Neovascular membranes lie on the retinal surface or at the vitreoretinal interface.
- Scarring in this location can act directly on retinal shape.

Membrane contraction links neovascularization to detachment



- Photoreceptor orientation may be disturbed, producing visual distortion.
- Stronger traction can produce frank detachment.

Traction may progress to a combined break-associated detachment

Initial tractional event

- Membrane pulls retina away from RPE
- No retinal break is required initially
- Driving force is mechanical contraction

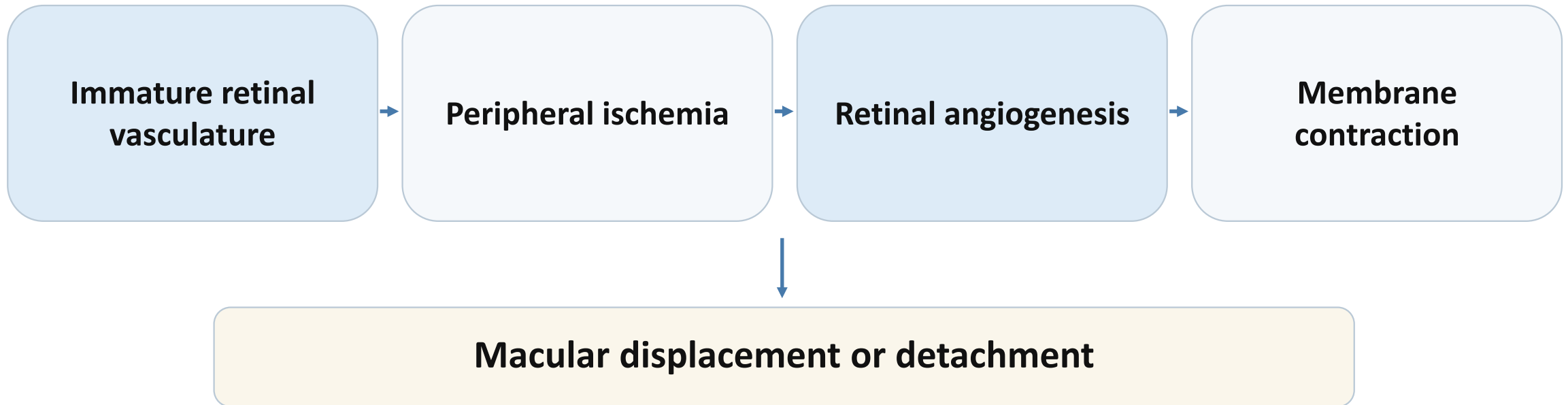
Secondary rhegmatogenous component

- Severe traction tears neurosensory retina
- Vitreous fluid can gain access through new break
- Both tractional and rhegmatogenous mechanisms coexist

Retinopathy of prematurity: Another tractional route

- Immature peripheral retinal vessels may constrict in premature infants exposed to oxygen.
- Peripheral retinal ischemia promotes VEGF and neovascularization.
- Contracting peripheral membranes can drag the retina and macula.
- Severe contraction may produce retinal detachment.

Retinopathy of prematurity: The sequence



- This is a second disease model for traction-driven detachment.
- The initiating problem is abnormal vascular development, not aging vitreous.

Diabetes versus prematurity: Shared mechanics

Proliferative diabetic retinopathy

- Microvascular damage/nonperfusion
- VEGF-driven surface neovascularization
- Membrane scarring exerts traction

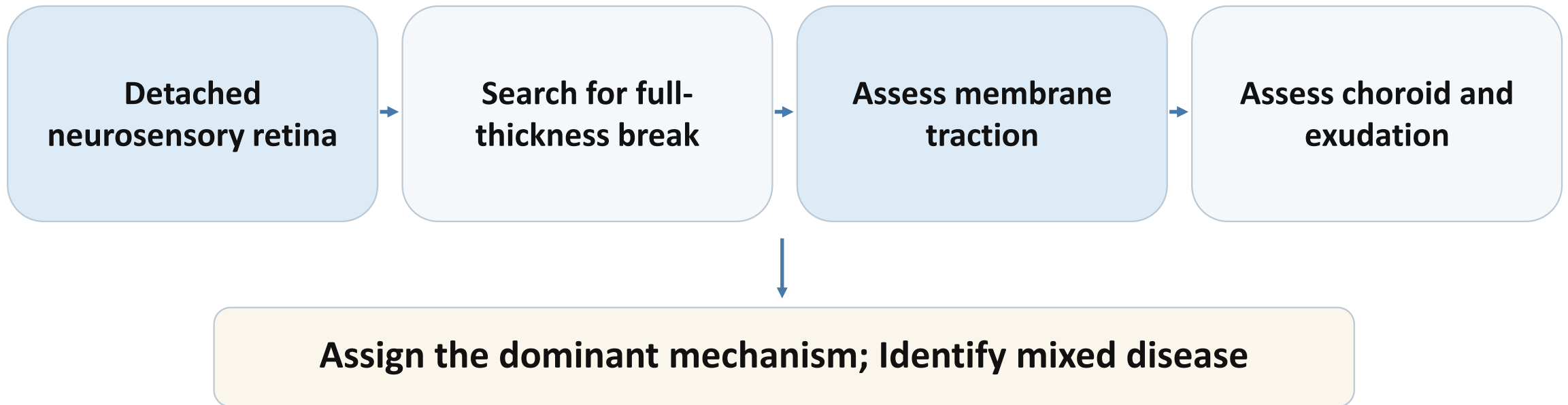
Retinopathy of prematurity

- Immature peripheral retinal circulation
- Ischemia-driven neovascular membrane
- Peripheral contraction can drag retina

Four paths to one anatomical endpoint

Path	Starting lesion	Fluid/force	Break initially?
Rhegmatogenous	Vitreoretinal adhesion + tear	Vitreous fluid	Yes
Exudative	Vascular or RPE damage	Subretinal exudate	No
Tractional	Contracting membrane	Mechanical traction	Usually no
Combined traction + break	Severe membrane traction	Traction plus vitreous fluid	Becomes present

Mechanism-first diagnostic reasoning



- The absence of a visible break alone does not prove exudation.
- Consider coexisting causes and limited visualization.

Morphologic clues mapped to etiology

Finding	Reasoning implication
Full-thickness tear with subretinal fluid	Supports rhegmatogenous route
Protein-rich fluid without tear	Supports exudative nonrhegmatogenous route
Retinal elevation under contracting membrane	Supports tractional mechanism
Missing photoreceptor outer segments	Supports chronicity; Not a unique mechanism
Glial/RPE-cell surface membranes	Suggests proliferative vitreoretinopathy

Case 1 — a focal hyaloid attachment

Aging vitreous has liquefied. Most of the posterior hyaloid has separated, but a small adhesion persists. A full-thickness retinal tear forms at that site.

What sequence explains retinal separation?

Tethered posterior hyaloid → retinal tear → liquefied vitreous enters subretinal space → rhegmatogenous detachment.

Case 2 — subretinal fluid in severe hypertension

Severe hypertension is accompanied by choroidal microvascular injury and focal RPE damage. Subretinal exudate accumulates without an identified break.

Which detachment mechanism fits the findings?

Choriocapillaris/RPE injury allows subretinal fluid → exudative nonrhegmatogenous detachment.

Case 3 — membrane-associated retinal elevation

A retina with proliferative diabetic neovascular membranes becomes wrinkled as the membranes scar and contract. Retinal elevation develops without a full-thickness break.

How should the detachment be classified at this stage?

Tractional, initially nonrhegmatogenous; Later a secondary tear could create a combined detachment.

Case 4 — a chronic detached retina

A detached neurosensory retina has markedly shortened or absent photoreceptor outer segments. No original tear is identified in the limited specimen.

What can and cannot be inferred?

Chronicity is supported; The finding alone cannot distinguish rhegmatogenous from nonrhegmatogenous causes.

Three commonly confused interfaces

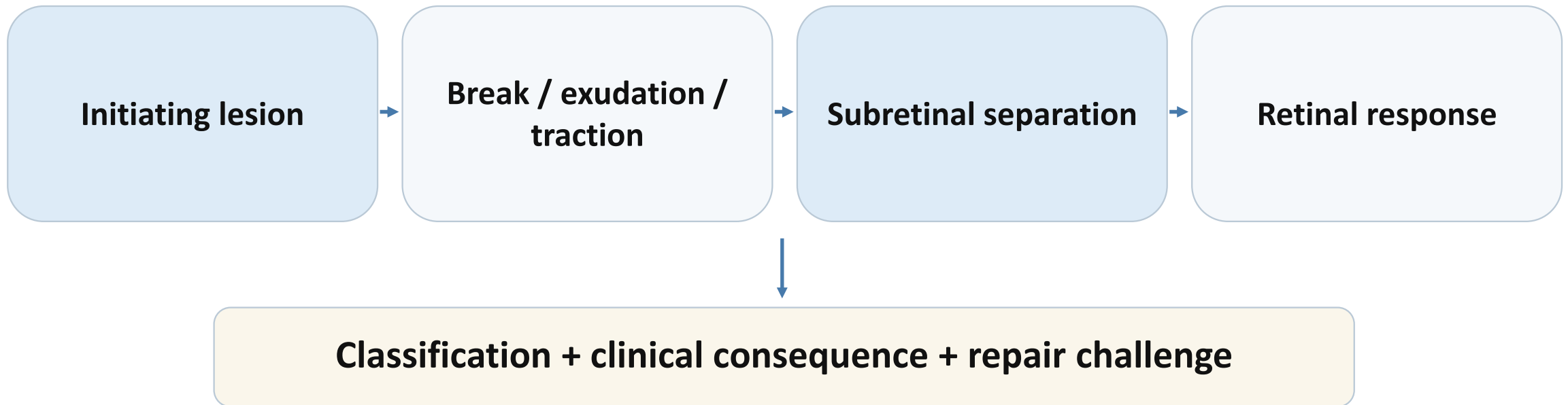
Vitreous–retina

- Posterior hyaloid contacts internal limiting membrane
- PVD occurs here
- Abnormal adhesions create traction

Retina–RPE

- Photoreceptor outer segments face RPE
- Retinal detachment occurs here
- Exudate or vitreous fluid may separate layers

Synthesis: Separate lesion, force, and endpoint



- The same anatomic endpoint has different upstream causes.
- Photoreceptor change and membrane proliferation influence later tissue status.

Core pathology conclusions

- Retinal detachment is separation of neurosensory retina from RPE.
- Rhegmatogenous detachment requires a full-thickness break and fluid entry from vitreous.
- Exudative detachment follows vascular or RPE injury without a retinal break.
- Tractional membranes may detach retina and may subsequently tear it.
- Outer-segment loss indicates chronicity; PVR creates additional membrane pathology.

Selected references

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