

Retinal Artery and Vein Occlusions

Mechanisms • morphology • clinicopathologic interpretation

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Learning outcomes: Explain the vascular lesion

Distinguish arterial infarction from venous occlusive injury.

Relate arterial territory to the distribution of retinal opacity.

Explain how emboli and local atherosclerotic thrombosis arise.

Learning outcomes: Interpret the visible fundus

Derive the cherry-red spot from retinal and foveal anatomy.

Explain why storage diseases can yield the same ophthalmoscopic sign.

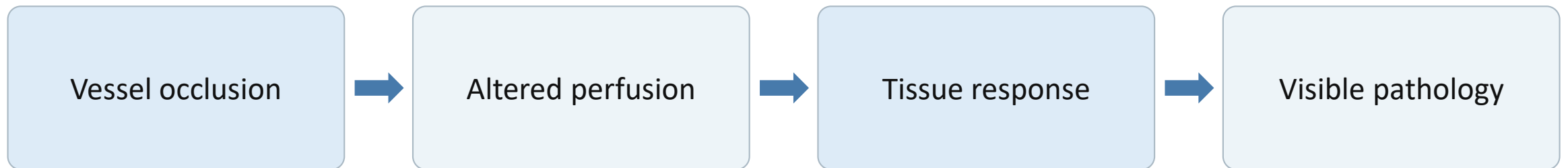
Contrast ischemic and nonischemic retinal vein occlusion.

Occlusion produces different forms of injury

Arterial obstruction deprives retinal tissue of its blood supply.

Venous obstruction is associated with hemorrhage, exudation, and/or ischemia.

Ischemia may induce a secondary angiogenic response.

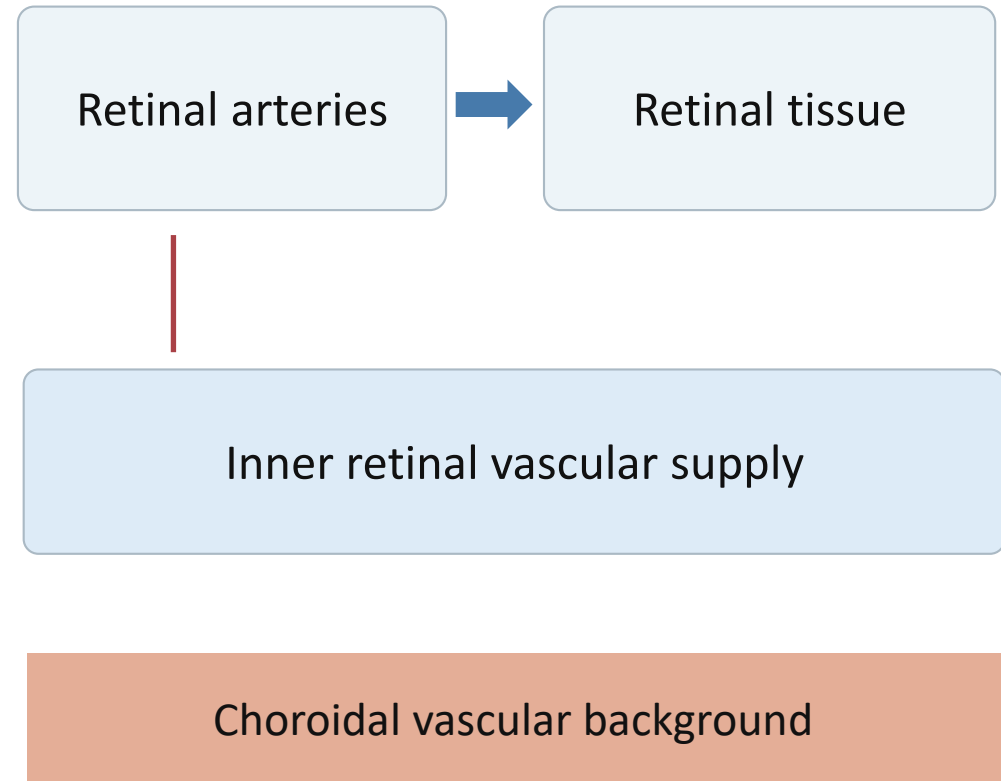


Two vascular backgrounds must be distinguished

Retinal tissue has a vascular supply vulnerable to arterial occlusion.

The choroid is richly vascular and normally supplies an orange-red fundus background.

Retinal opacity changes how much choroidal color is visible.



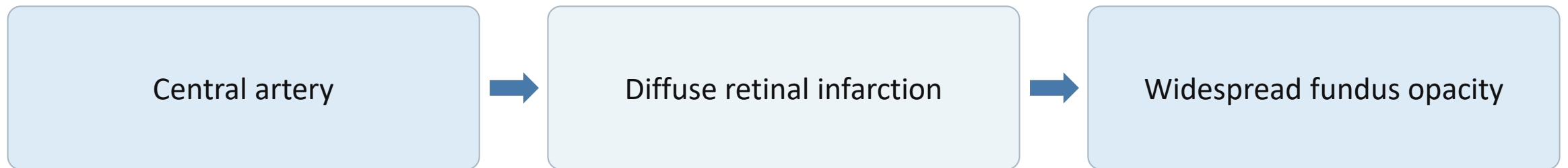
Conceptual orientation • two vascular compartments

Central and branch vessels define lesion extent

Central-vessel obstruction threatens a broad retinal territory.

A branch-vessel lesion produces a more limited segmental distribution.

The topography of injury follows the supply of the occluded vessel.

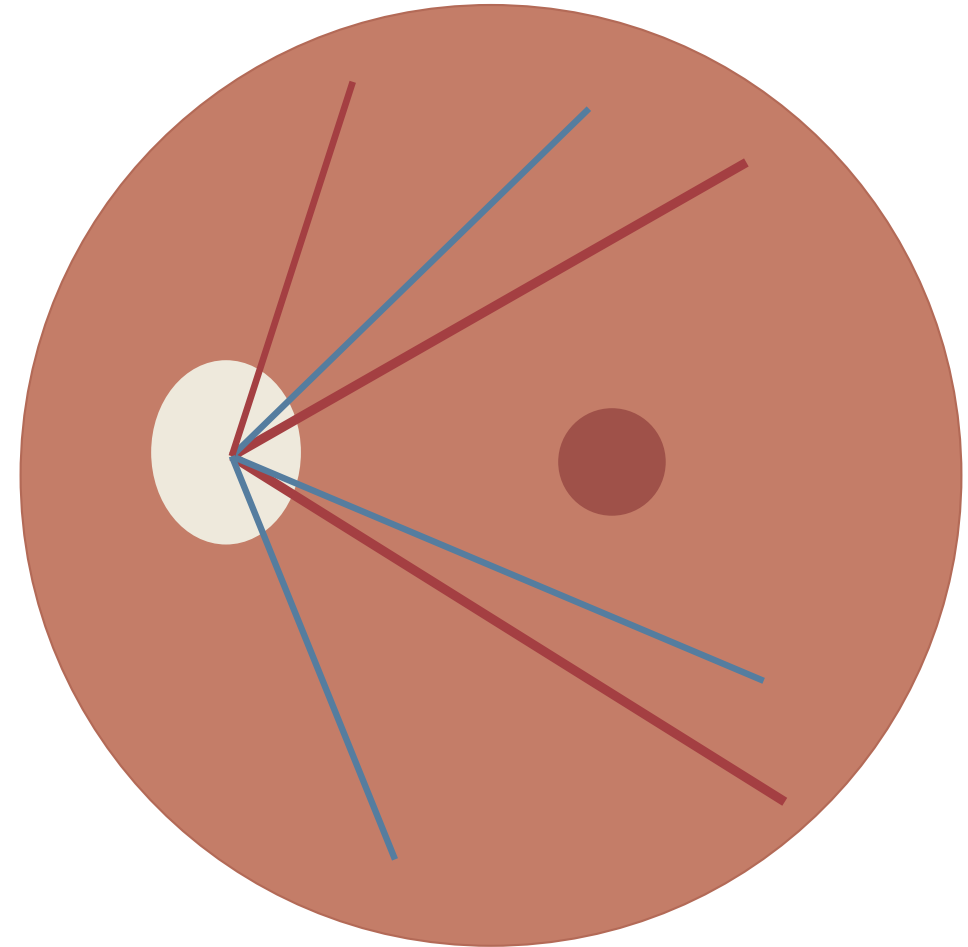


Anatomy explains the color of the normal fundus

The retina is relatively transparent under normal viewing conditions.

The choroidal circulation contributes the usual red-orange background.

Opaque retinal tissue can mask this underlying vascular color.



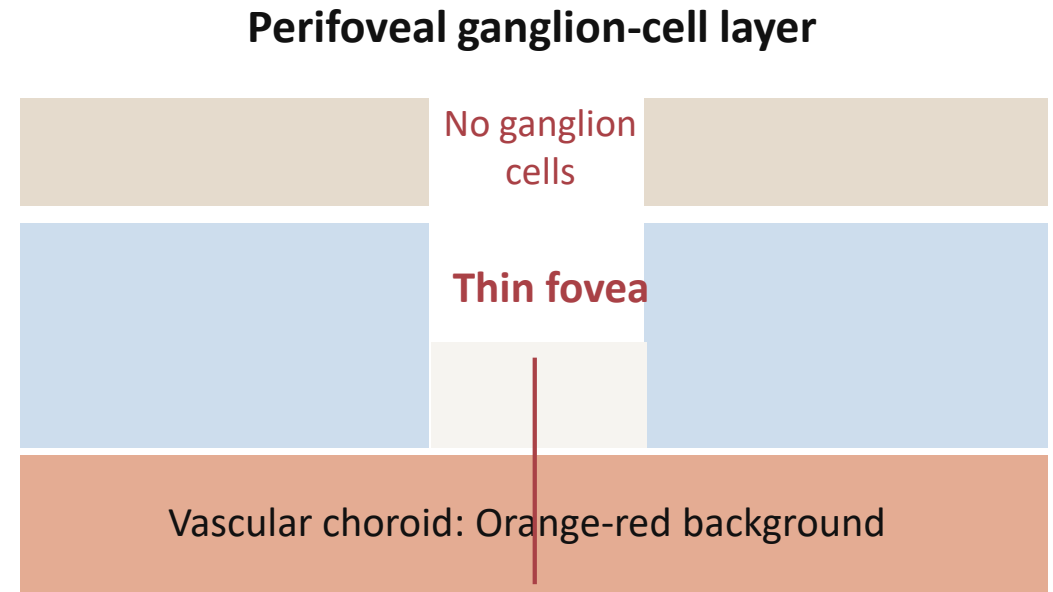
Simplified fundus schematic

The fovea is anatomically distinct

Fovea and foveola are particularly thin.

The foveal center lacks ganglion cells.

This thin central area remains comparatively transparent when surrounding retina becomes opaque.



Foveal thinness permits a choroidal color window

Cross-sectional concept • schematic; Not histology

Arterial versus venous obstruction: A framework

Feature	Retinal artery	Retinal vein
Primary event	Loss of arterial inflow	Venous occlusive injury
Dominant textbook lesion	Retinal infarction	Ischemic or nonischemic consequences
Visible tissue changes	Acute whitening/opacity	Hemorrhage, exudate, edema; Neovascularization if ischemic
Major complication	Severe visual loss/blindness	Neovascular glaucoma in ischemic disease

Thromboembolism is the leading arterial mechanism

Thromboembolism as the most common cause of retinal artery occlusion.

The thrombus may form locally in a diseased central artery.

Alternatively, an embolus may originate upstream and lodge in the retinal circulation.

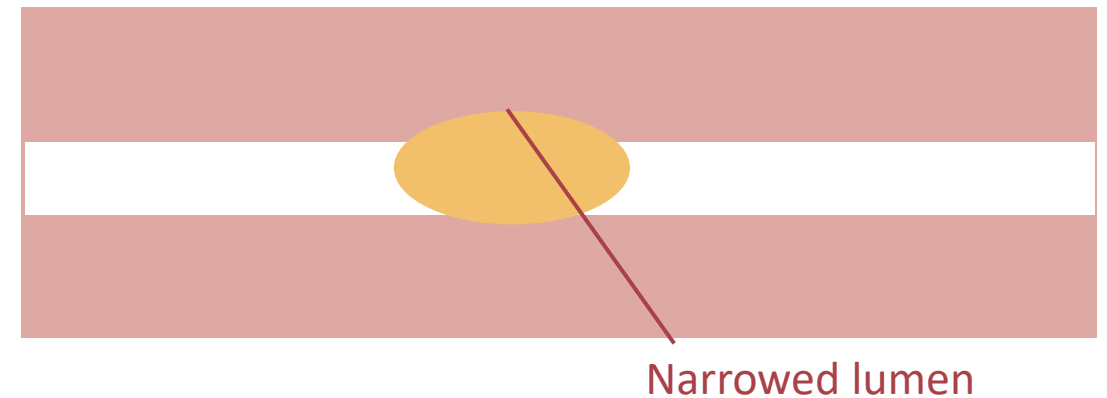
Atherosclerosis predisposes to local thrombosis

Atherosclerotic plaque narrows the central retinal arterial lumen.

Luminal narrowing creates conditions that favor thrombosis.

The resulting obstruction interrupts distal retinal perfusion.

Plaque-related luminal narrowing



Atherosclerotic
narrowing



Local thrombosis risk

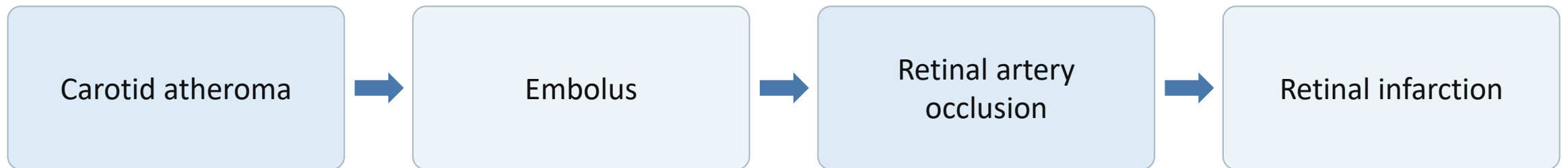
Simplified artery longitudinal section

The carotid artery can supply an embolic source

An ulcerated carotid atheromatous plaque can release embolic material.

Embolization to the central retinal artery interrupts flow downstream.

The clinically observed retinal lesion is therefore potentially a consequence of proximal vascular disease.

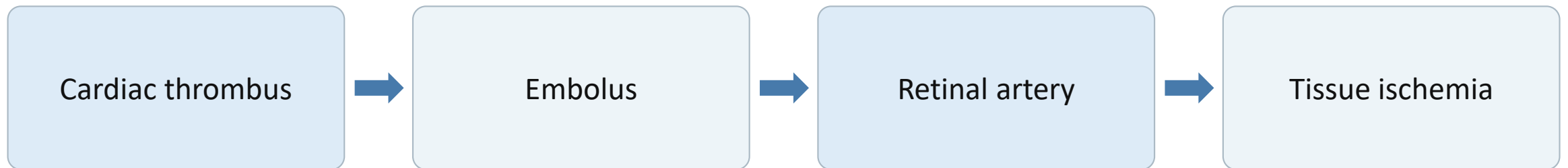


Cardiac thrombi can also embolize

A thrombus within the heart is another source of retinal arterial emboli.

A cardiac-source embolus and a carotid-source embolus may converge on the same retinal end-organ lesion.

The retinal appearance reflects the vascular insult; It does not itself identify the embolic source.



Local thrombosis and embolization are distinct

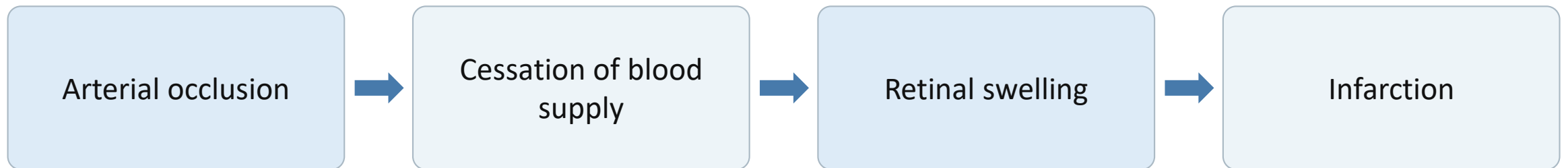
Question	Local arterial thrombosis	Retinal arterial embolus
Origin	At a diseased arterial segment	Proximal heart or carotid artery
Predisposing process	Atherosclerotic luminal narrowing	Thrombus or ulcerated atheromatous plaque
Shared endpoint	Interruption of retinal arterial flow	Interruption of retinal arterial flow
Interpretive limit	Fundus infarction alone does not prove which mechanism occurred	Fundus infarction alone does not prove which source embolized

Retinal arterial obstruction initiates infarction

Cessation of arterial supply causes abrupt deprivation of perfusion.

The affected retina undergoes acute injury and swelling.

Sufficiently severe arterial occlusion produces an infarct.

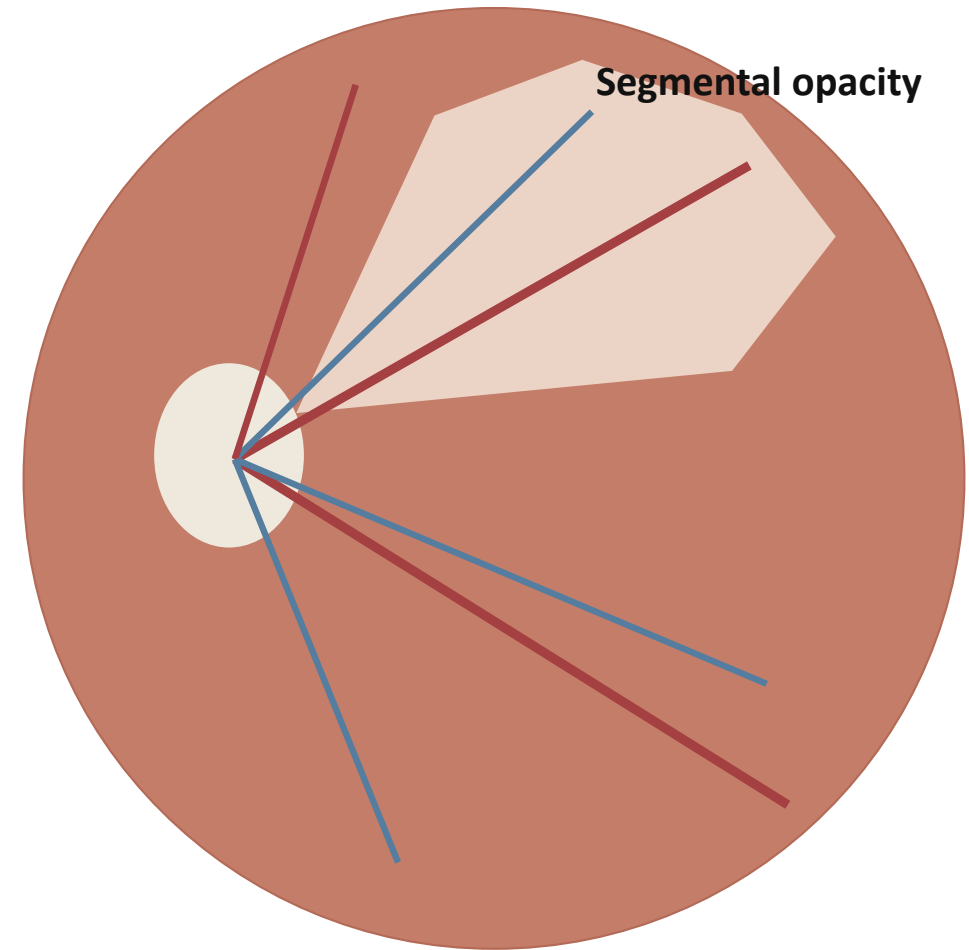


A branch-artery lesion has a segmental footprint

Total occlusion of one retinal arterial branch produces segmental infarction.

Opacity is confined to the affected vascular territory.

Fundus distribution therefore helps localize the arterial obstruction.



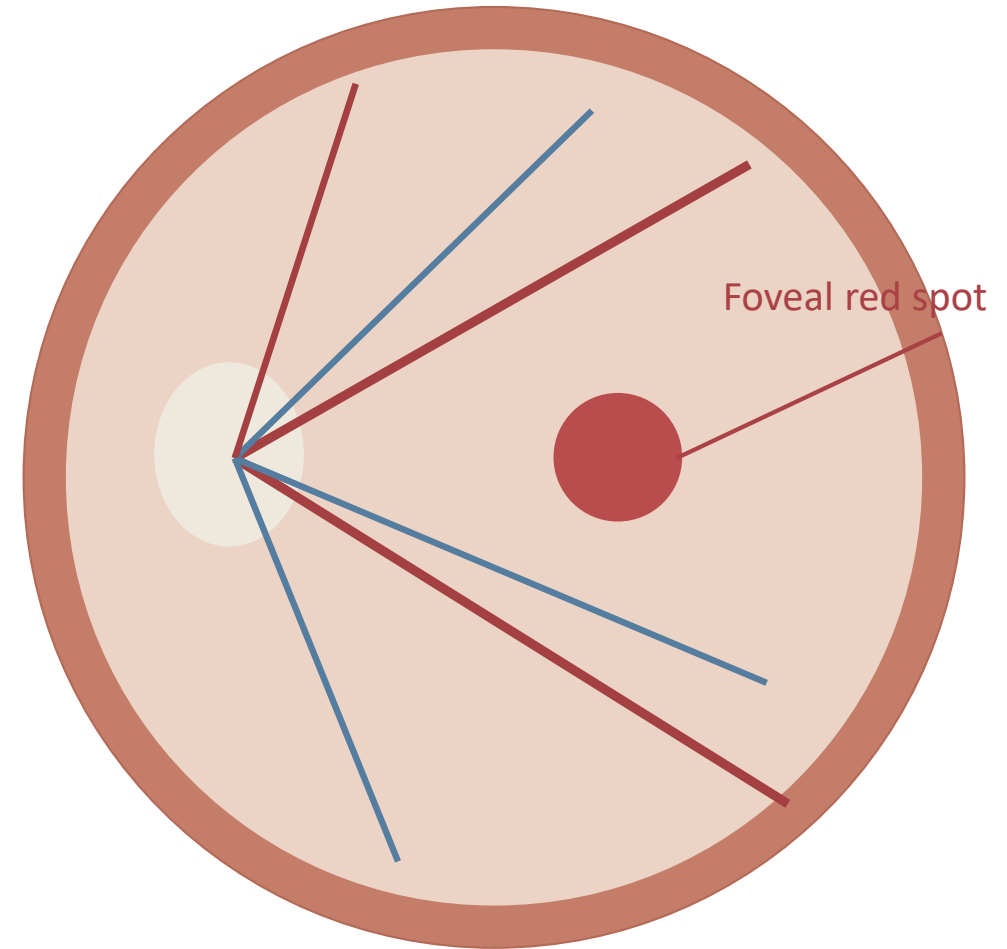
Simplified fundus schematic

Central artery occlusion has a diffuse footprint

Total central retinal artery occlusion can produce diffuse retinal infarction.

Much of the retina becomes optically opaque.

The thin foveal region remains a conspicuous exception.



Simplified fundus schematic

Vascular geography determines infarct geography

Characteristic	Branch artery occlusion	Central artery occlusion
Occluded level	Arterial branch	Central retinal artery
Infarct pattern	Segmental	Diffuse
Fundus opacity	Regional area of whitening	Widespread retinal whitening
Optical foveal contrast	Depends on distribution	Classically explains cherry-red appearance

Acute retinal injury causes swelling

The acutely swollen retina with the brain, from which it is derived.

Swelling is an immediate structural consequence of sudden ischemia.

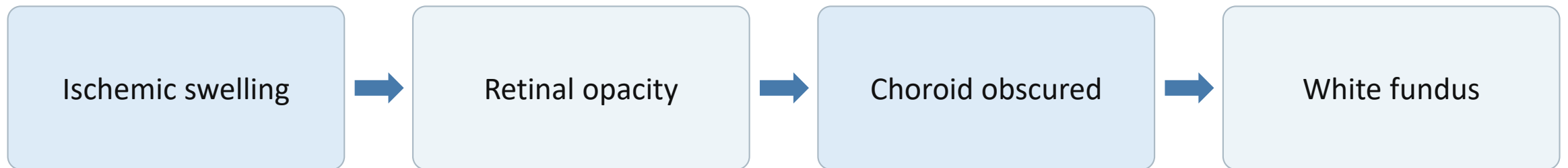
The altered tissue becomes optically opaque rather than transparent.

Why retinal infarction looks white

The normal red-orange choroid lies beneath the retina.

The infarcted, swollen retina blocks that choroidal background.

The affected fundus appears white rather than the usual orange-red.

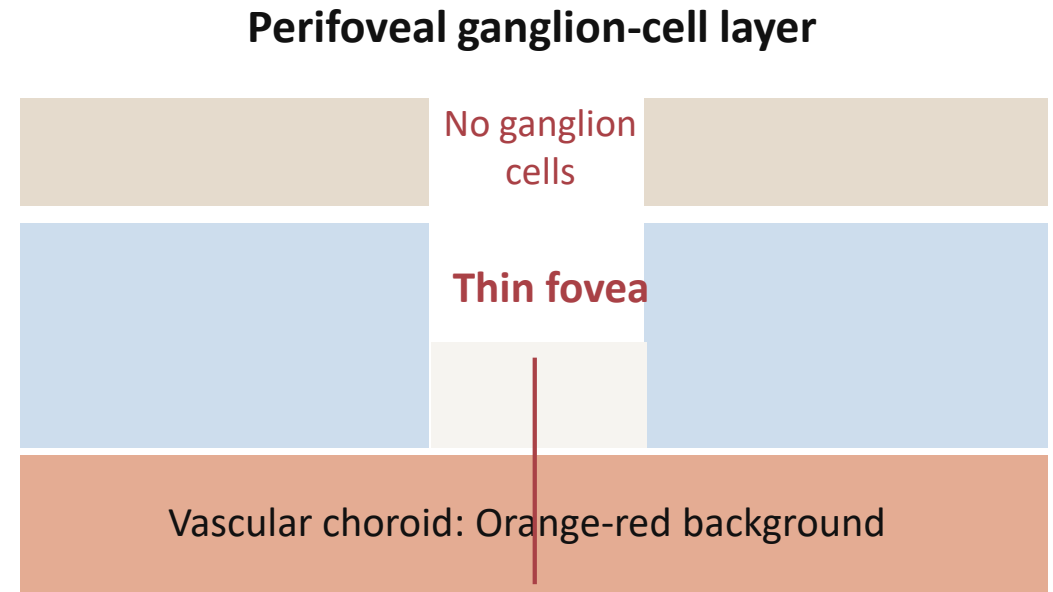


The apparent whiteness is an optical effect

The color change does not require the retina itself to contain white pigment.

It results from opaque swollen retinal tissue over a red vascular bed.

Interpret tissue transparency and background color together.



Foveal thinness permits a choroidal color window

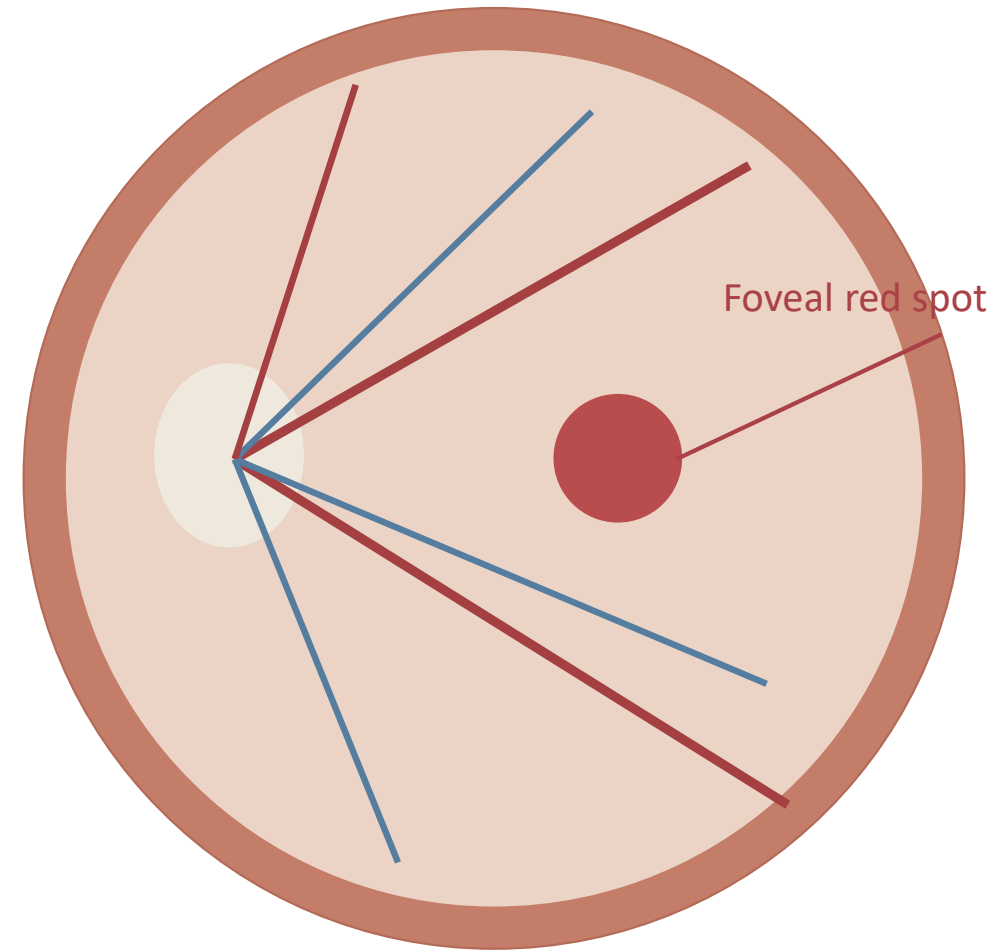
Cross-sectional concept • schematic; Not histology

Diffuse whitening leaves a foveal color window

In central artery occlusion, surrounding retinal tissue becomes opaque.

Fovea and foveola are much thinner than neighboring retina.

The red-orange choroid remains visible centrally.



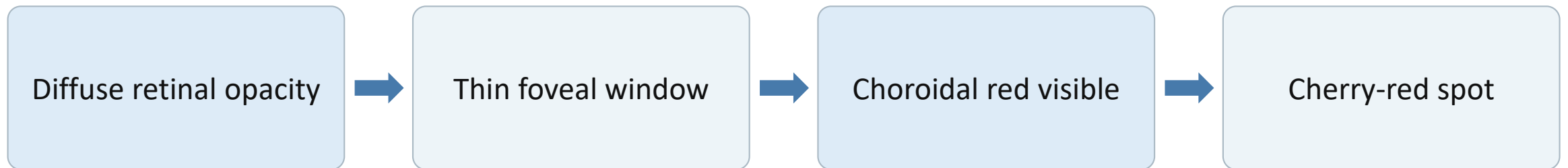
Simplified fundus schematic

A cherry-red spot arises from contrast

The central red area is highlighted by pale, infarcted surrounding retina.

It represents choroidal color seen through thin foveal tissue.

The surrounding white retina creates the diagnostic visual contrast.

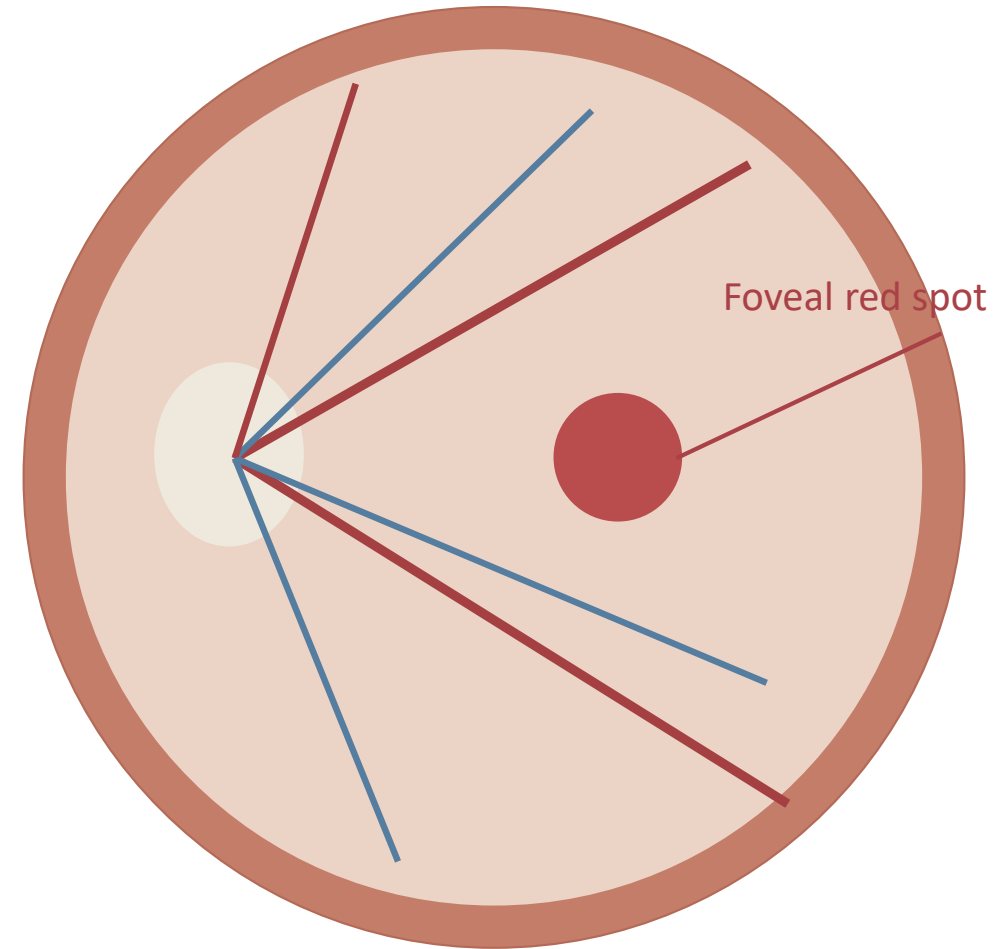


Cherry-red does not mean central hemorrhage

The textbook mechanism is optical transmission of the choroidal background.

The spot is not defined by extravasated blood in the fovea.

The distinction matters because the same appearance occurs through different mechanisms.



Simplified fundus schematic

The fundus links anatomy to pathology

Structure	Normal optical role	During central retinal artery occlusion
Choroid	Orange-red vascular background	Background persists
Nonfoveal retina	Relatively transparent	Swollen, white, opaque
Fovea / foveola	Thin window over choroid	Remains comparatively transparent
Composite appearance	Orange-red fundus	Pale retina with central red spot

Arterial infarction may cause blindness

The central retinal artery supplies a large vulnerable retinal territory.

Total occlusion can lead to diffuse infarction.

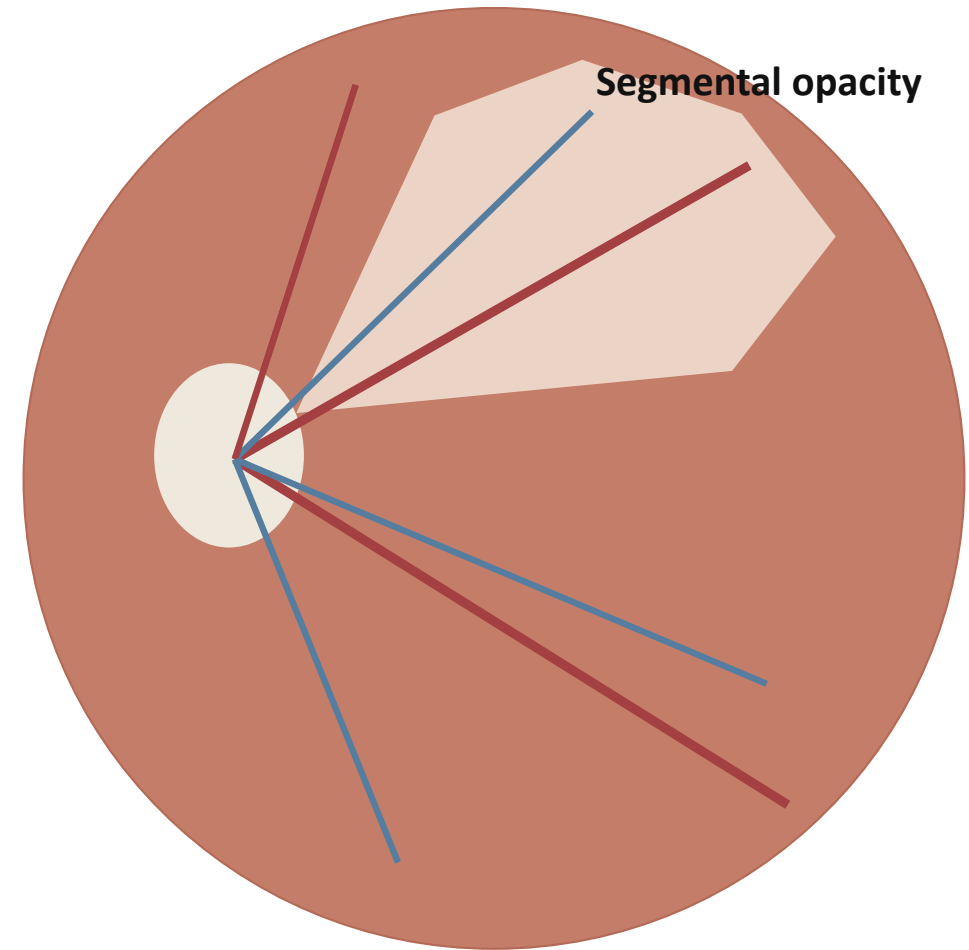
Robbins explicitly identifies blindness as a possible consequence of arterial occlusion.

Reason from distribution before naming a lesion

Diffuse whitening suggests a central supply interruption.

Sectoral whitening favors interruption of a branch territory.

Topographic reasoning is more informative than color alone.



Simplified fundus schematic

Case 1: Distinguish level of occlusion

A fundus schematic shows a white sector rather than diffuse retinal opacity.

Which arterial territory is more likely to be occluded?

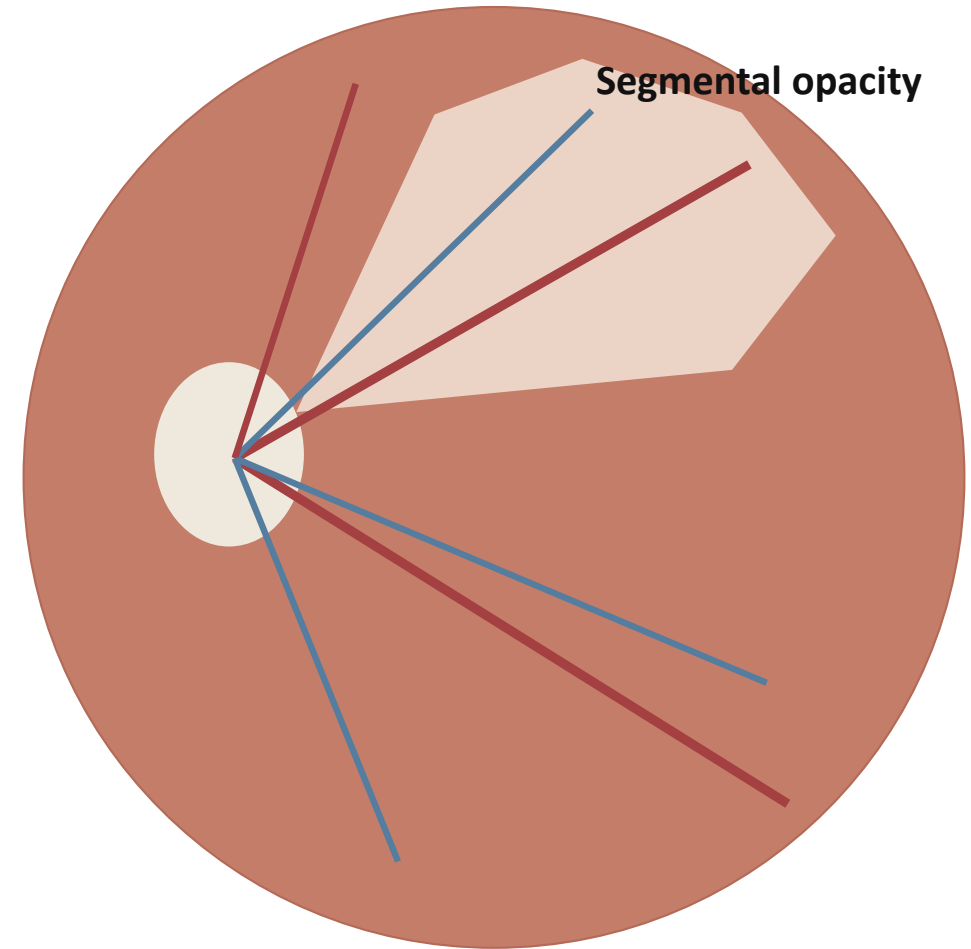
What tissue event explains the white sector?

Case 1: Tissue and vessel correlate

A branch-vessel obstruction best explains a segmental infarct.

Acute ischemic swelling makes that segment optically opaque.

The opacity obscures the orange-red choroid only in the involved area.



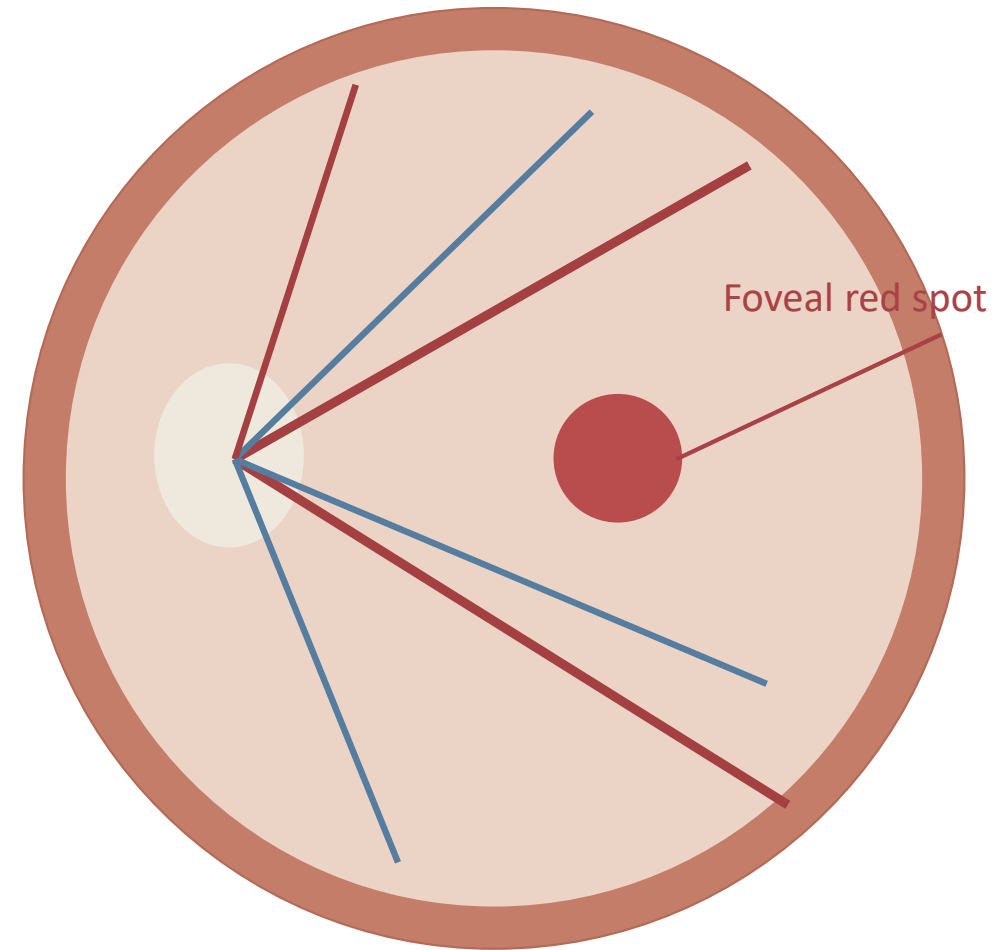
Simplified fundus schematic

The cherry-red spot is not disease-specific

The sign in central retinal artery occlusion.

It may also occur in certain rare lysosomal storage diseases.

An identical visible sign can therefore arise from different retinal pathology.



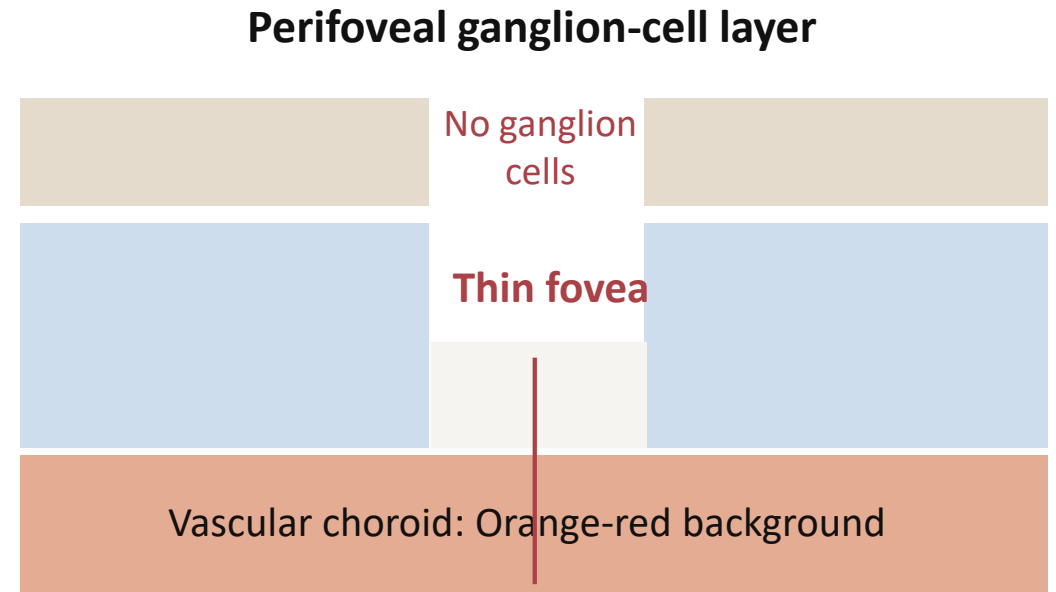
Simplified fundus schematic

Tay–Sachs and Niemann–Pick: Examples

Both are named in the Robbins passage as conditions with cherry-red spots.

Storage material accumulates in retinal ganglion cells.

Ganglion-cell-rich retinal tissue surrounding the fovea becomes relatively opaque.



Foveal thinness permits a choroidal color window

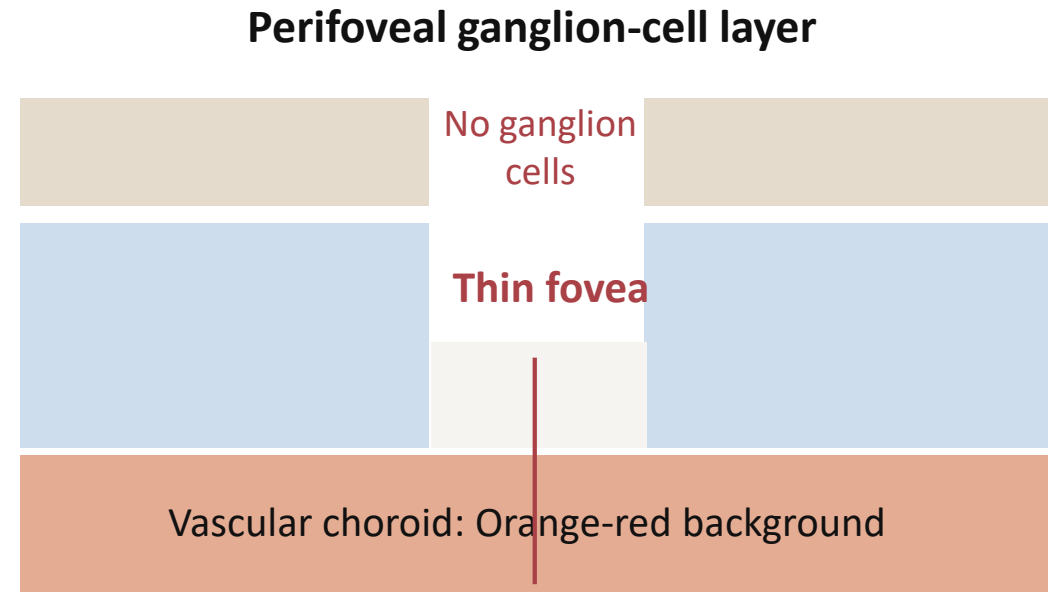
Cross-sectional concept • schematic; Not histology

Storage material changes perifoveal transparency

The ganglion cell layer is comparatively thick around the fovea.

Intracellular storage material reduces transparency of those adjacent cells.

The foveal center lacks ganglion cells and is relatively spared by this mechanism.



Foveal thinness permits a choroidal color window

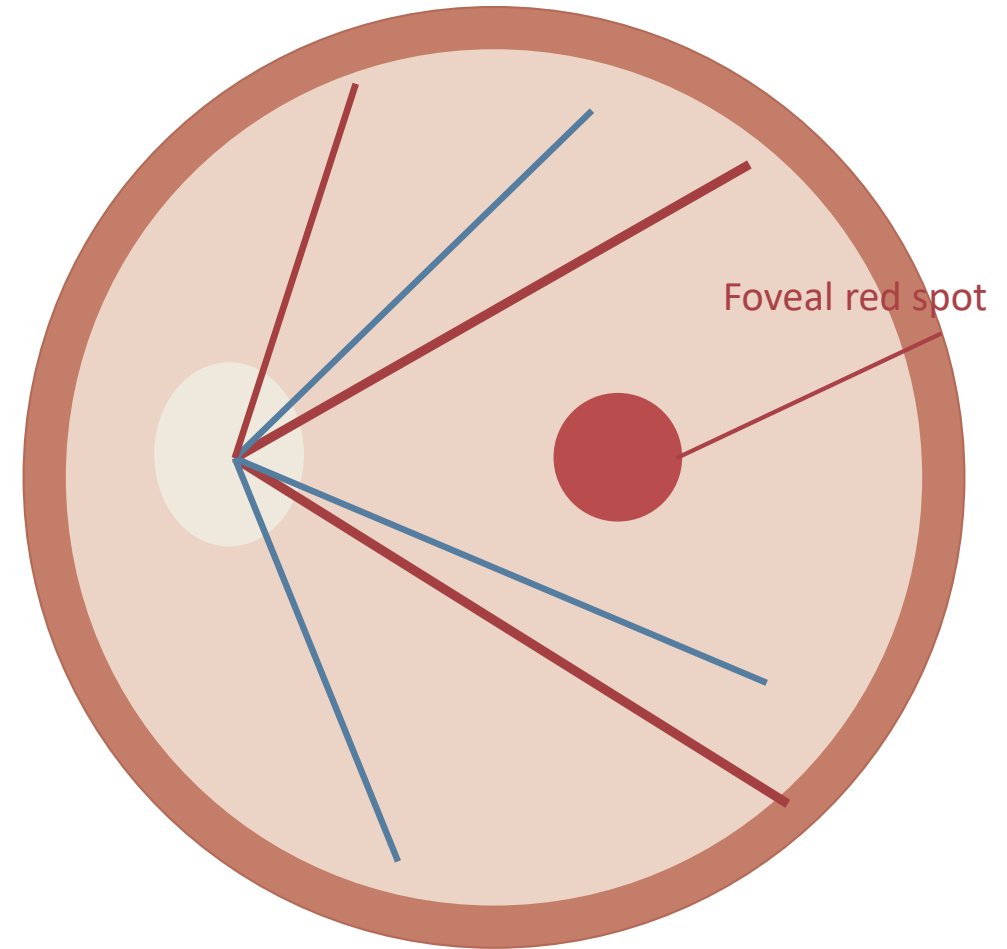
Cross-sectional concept • schematic; Not histology

The foveal center remains visibly red in storage

No retinal ganglion cells occupy the center of the fovea.

The ganglion-cell storage process therefore spares this central optical window.

Choroidal orange-red remains visible through the central macula.



Simplified fundus schematic

Same sign, different mechanisms

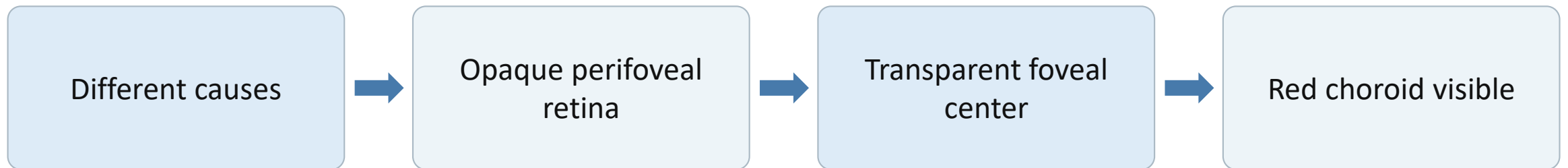
Element	Retinal artery occlusion	Lysosomal storage disease
Why surrounding retina is opaque	Acute ischemic swelling	Stored material in retinal ganglion cells
Why fovea remains red	Foveal thinness	Absent ganglion cells in the center
Source of red color	Underlying choroid	Underlying choroid
Interpretation	Acute vascular mechanism	Storage-related cellular mechanism

A shared appearance is not shared pathogenesis

Both diseases increase contrast between the fovea and adjacent retina.

Arterial infarction changes tissue hydration and optical opacity.

Storage diseases change ganglion-cell content and transparency.



Case 2: Avoid overcalling arterial occlusion

A fundus has a central red area surrounded by pale retina.

Is the red spot alone sufficient to establish thromboembolism?

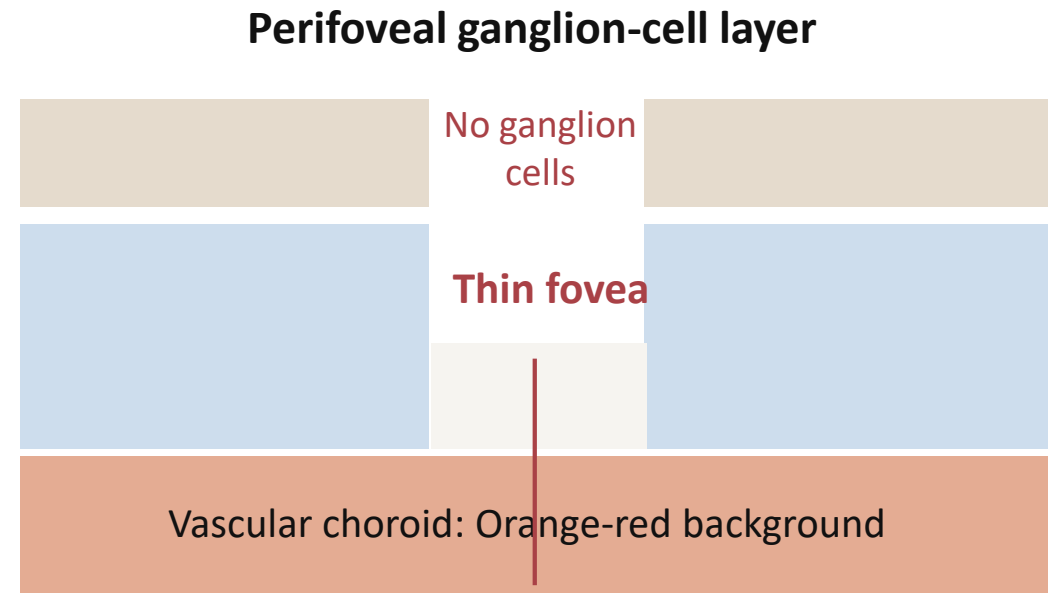
Which alternative pathogenic category is?

Case 2: The differential is mechanistic

No: The cherry-red sign is not unique to central retinal artery occlusion.

Ganglion-cell storage disorders are one mechanistically distinct explanation.

Use accompanying evidence rather than assigning etiology from this sign alone.



Foveal thinness permits a choroidal color window

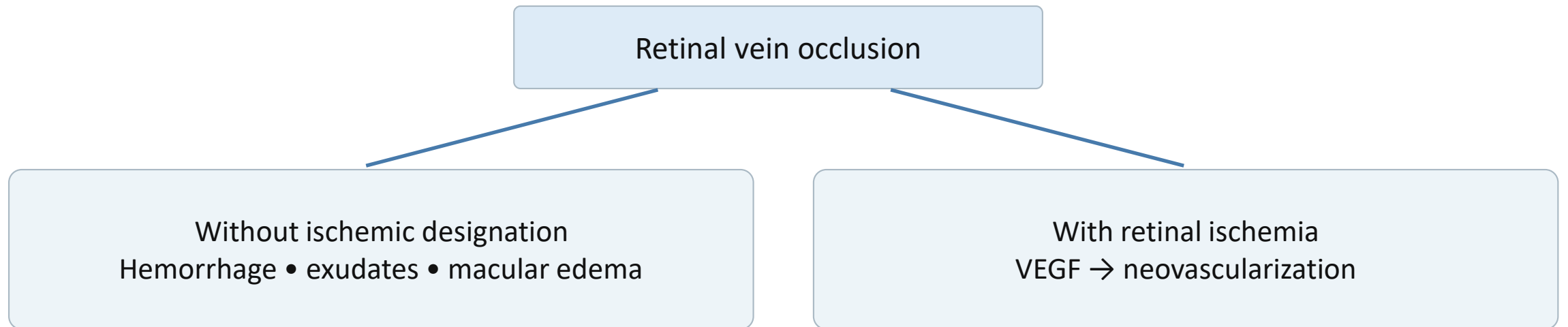
Cross-sectional concept • schematic; Not histology

Retinal vein occlusion has two broad patterns

The focused textbook passage divides retinal vein occlusion by the presence or absence of ischemia.

Ischemic disease may induce retinal angiogenic activity.

Nonischemic disease may be complicated by hemorrhage, exudates, and macular edema.

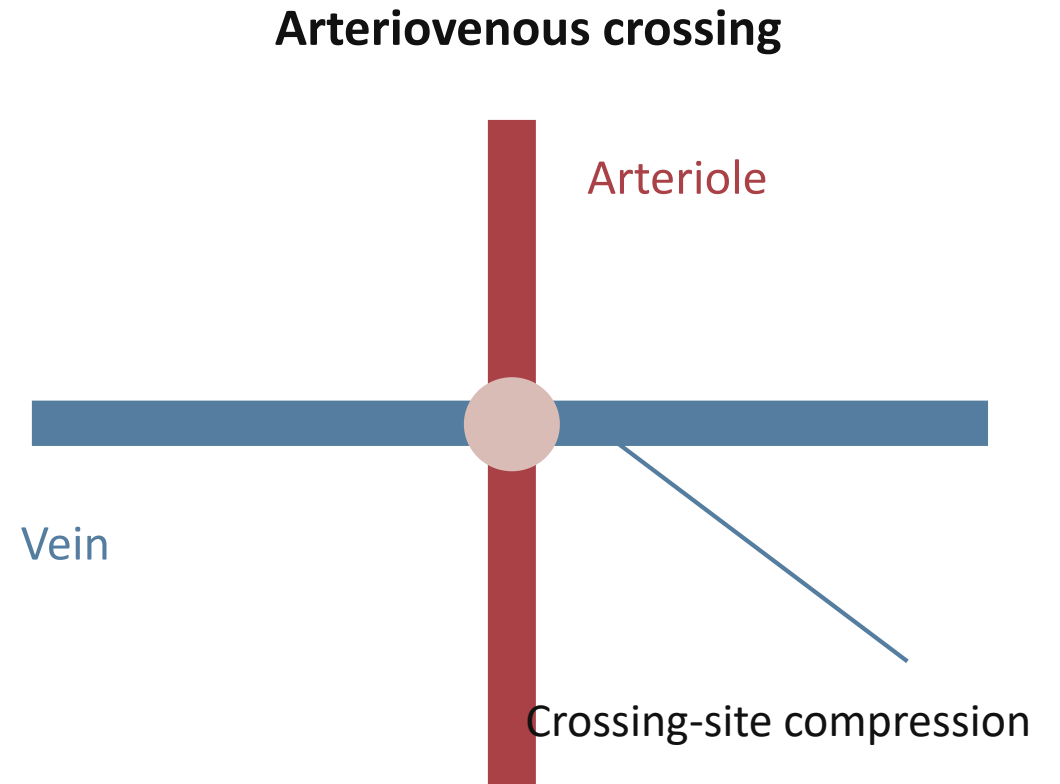


Arteriovenous crossings matter to branch veins

Retinal arterioles and veins share a common adventitial sheath at their crossings.

Pronounced arteriolosclerosis can compress an adjacent vein.

Distal venous stasis may precipitate branch retinal vein occlusion.



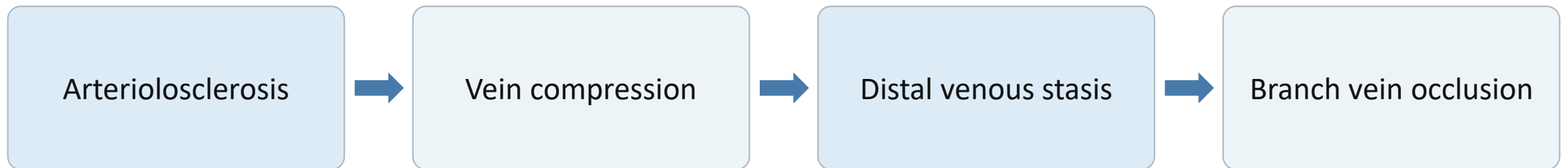
Conceptual schematic • not a vessel micrograph

Retinal hypertension creates a venous mechanism

A thickened retinal arteriolar wall can produce crossing-site compression.

This mechanism differs from an arterial embolus lodging downstream.

Retinal vascular diseases may interact anatomically at shared vessel crossings.

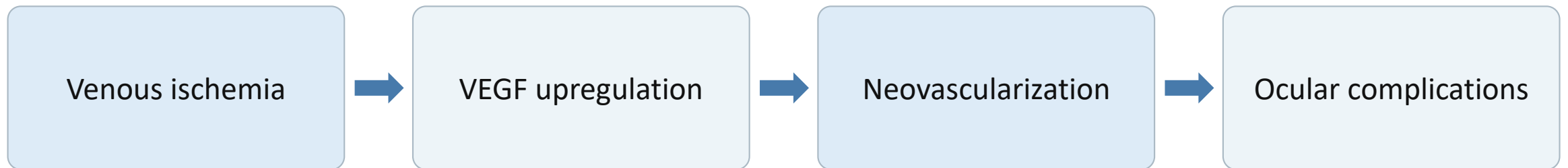


Ischemic vein occlusion is an angiogenic trigger

Retinal ischemia causes upregulation of VEGF and other proangiogenic factors.

The angiogenic response promotes formation of new vessels.

Their location determines complications involving the retina, optic disc, and iris.



VEGF links retinal hypoxia to vessel growth

Vascular endothelial growth factor is a molecular mediator of retinal angiogenesis.

Hypoxic retina is the stimulus described.

A molecular response can create clinically important secondary pathology.

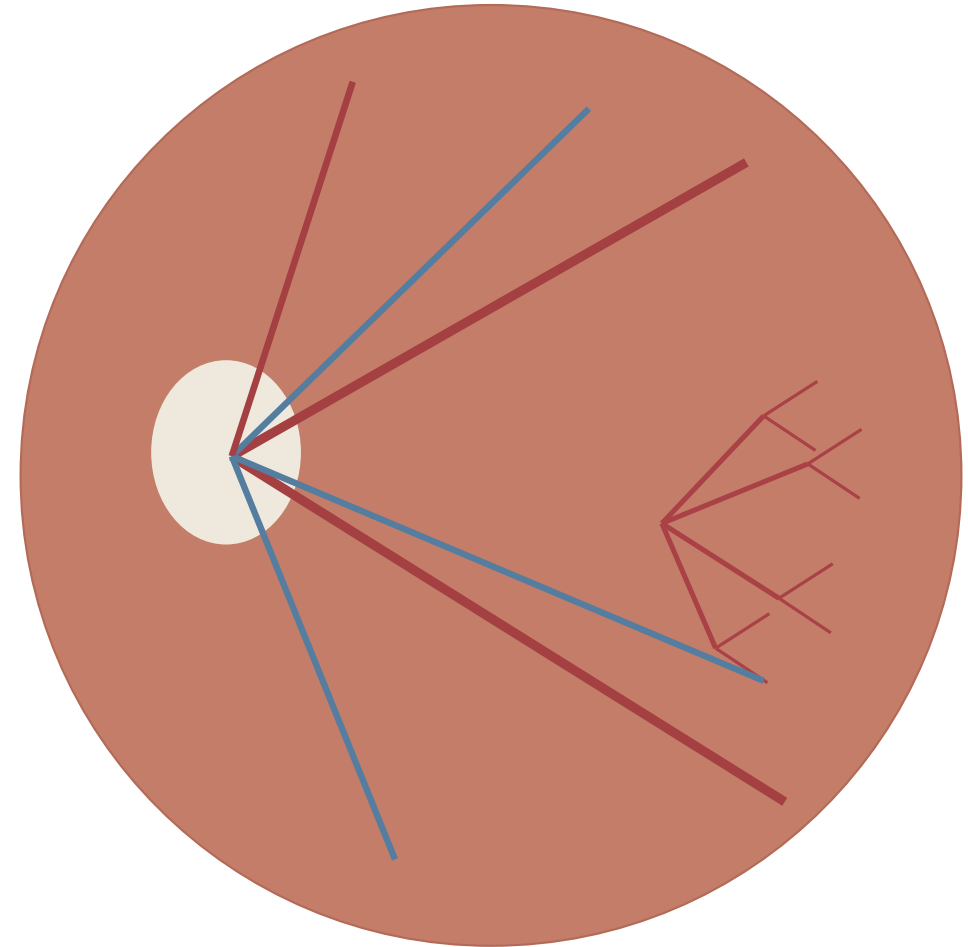


The retinal surface can develop new vessels

Ischemic retinal vein occlusion can cause neovascularization on the retina.

Surface neovascularization is distinct from a normal retinal vascular tree.

The pathologic vessels reflect a response to nonperfused retina.



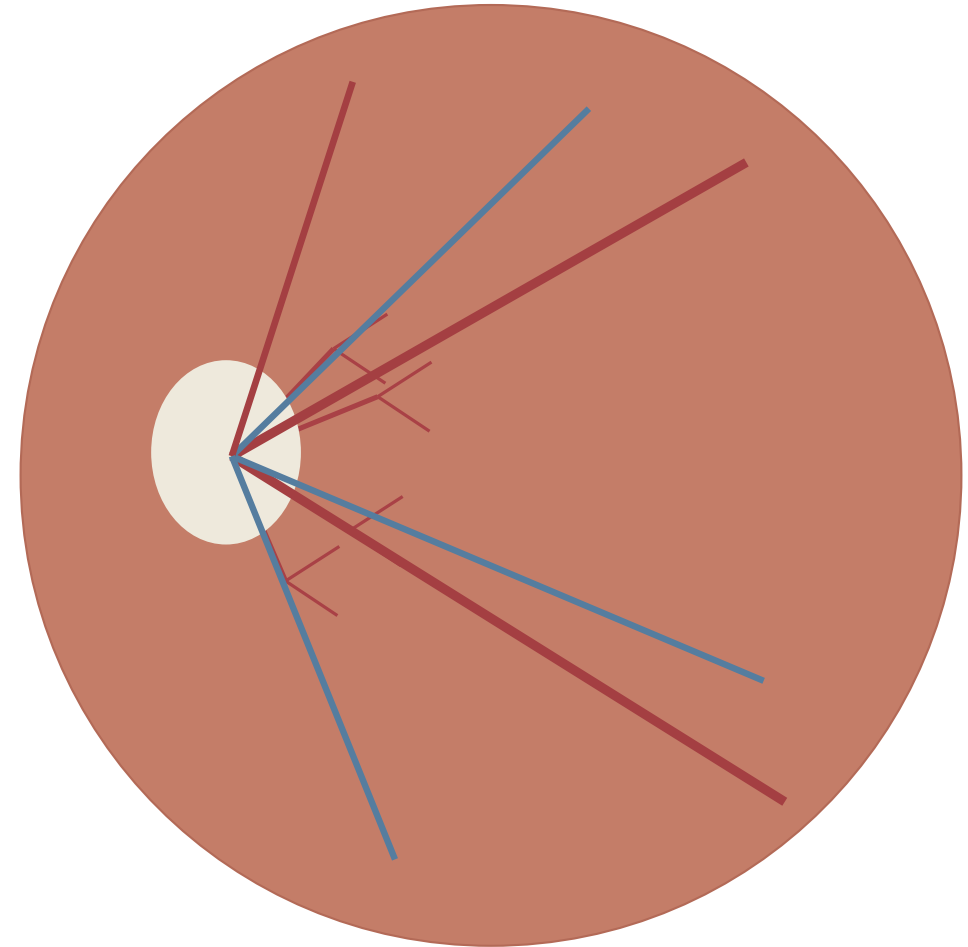
Surface neovessels — schematic

The optic nerve head is another site

Ischemia-driven neovascularization may develop on the optic nerve head.

Robbins also identifies retinal surface neovascularization.

The distribution of new vessels matters when interpreting ocular complications.



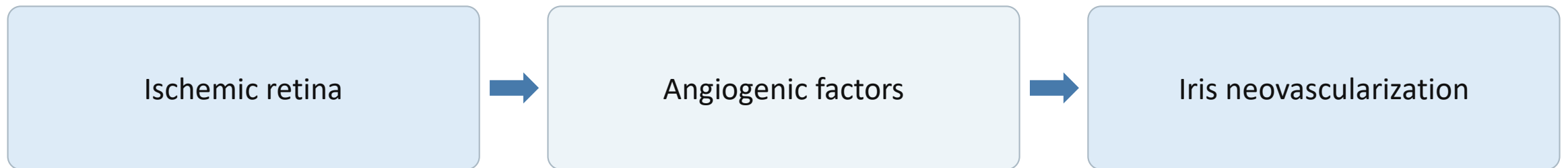
Surface neovessels — schematic

Retinal ischemia can affect the anterior segment

Ischemic retinal vein occlusion may stimulate new vessels on the iris.

This is an anterior-segment consequence of posterior retinal ischemia.

The pathologic linkage is mediated by angiogenic factors.



Iris neovascular membranes can contract

VEGF-associated iris neovascularization may produce a fibrovascular membrane.

Contraction can bring the iris toward the trabecular meshwork.

Peripheral anterior synechiae obstruct aqueous outflow.

Anterior-chamber outflow angle

Aqueous humor pathway

Trabecular meshwork

Iris surface membrane

Membrane contracts

Outflow obstructed

Mechanism schematic • not scaled anatomy

Neovascular glaucoma is an outflow disorder

The trabecular meshwork is a principal route of aqueous humor drainage.

An iris fibrovascular membrane can close off this route.

Obstructed drainage raises intraocular pressure: Secondary angle-closure (neovascular) glaucoma.

Anterior-chamber outflow angle

Aqueous humor pathway

Trabecular meshwork

Iris surface membrane

Membrane contracts

Outflow obstructed

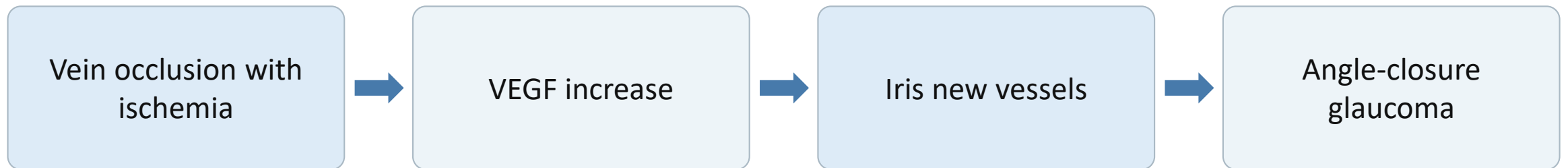
Mechanism schematic • not scaled anatomy

The pathway from vein occlusion to glaucoma

Glaucoma is not simply caused by hemorrhage within the retina.

The source links ischemia to VEGF, then iris neovascularization and angle closure.

Every step explains a distinct tissue or functional change.



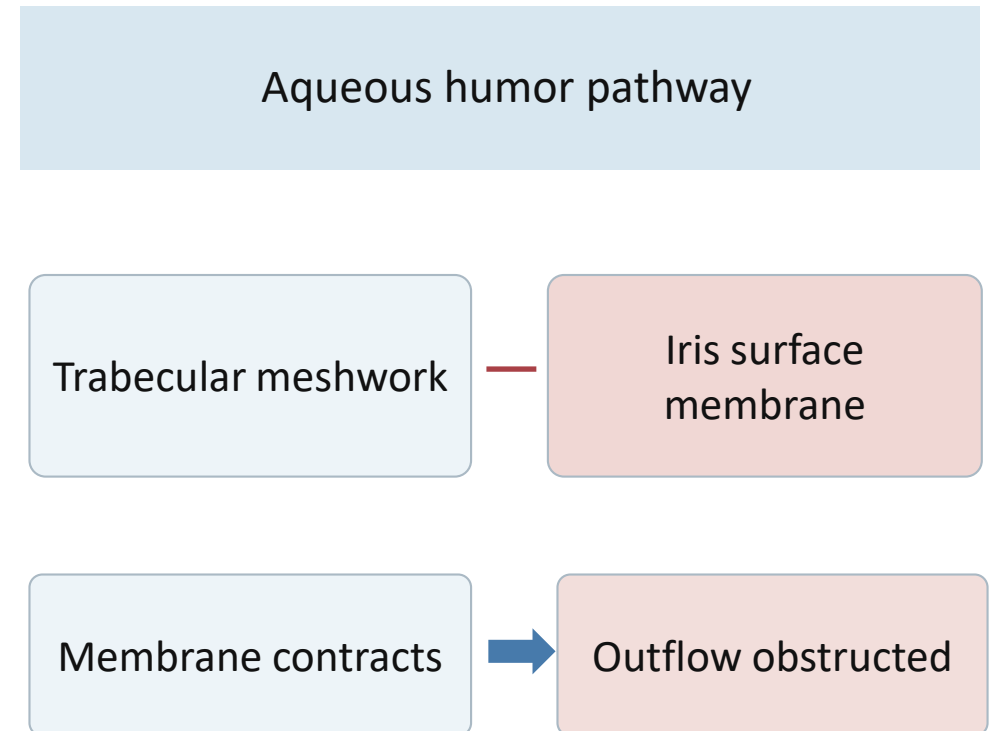
Posterior ischemia has distant consequences

The primary nonperfusion occurs in the retina.

The angiogenic response may extend beyond the site of nonperfusion.

Iris pathology can become the immediate cause of impaired aqueous drainage.

Anterior-chamber outflow angle



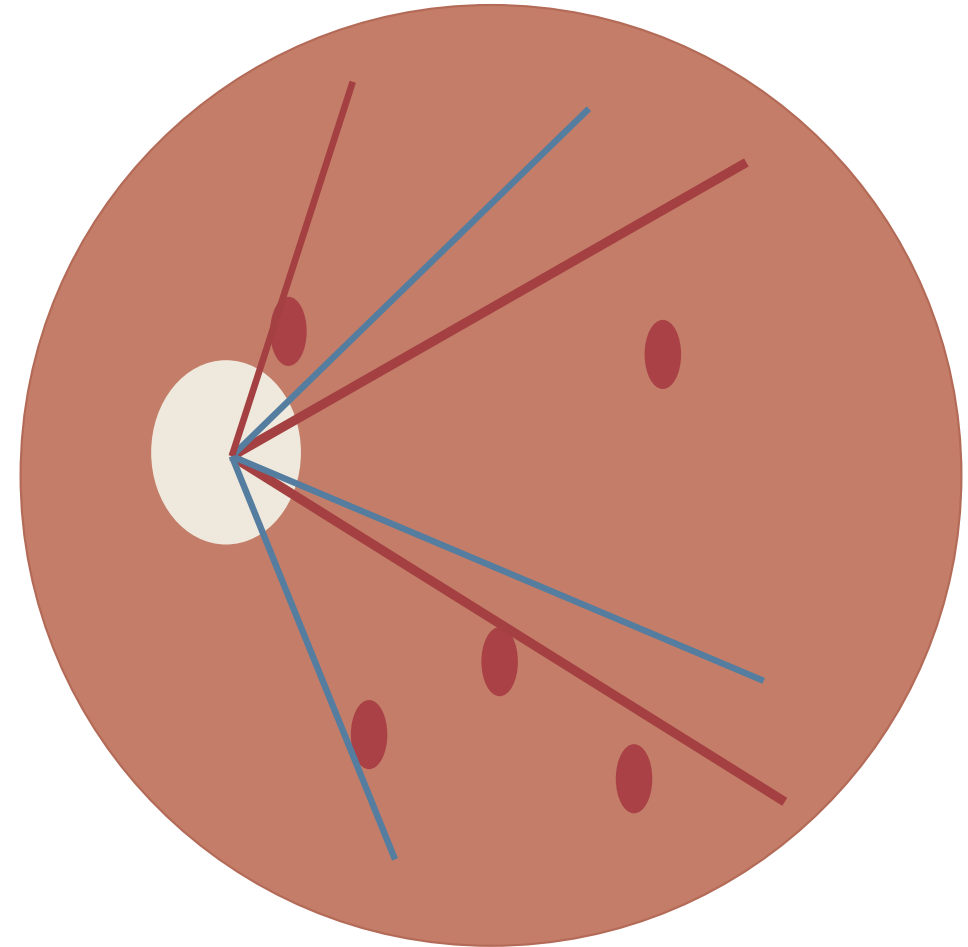
Mechanism schematic • not scaled anatomy

Nonischemic vein occlusion can cause hemorrhage

Retinal hemorrhages as a complication of nonischemic vein occlusion.

Hemorrhage reflects vascular injury rather than an arterial white infarct.

Hemorrhage alone should not be equated with the ischemic neovascular pathway.



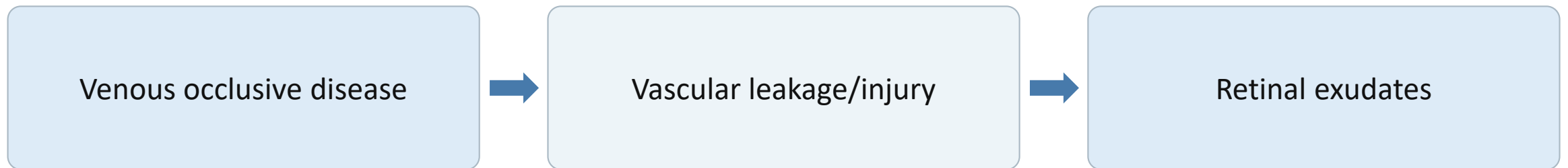
Hemorrhages — schematic

Exudates reflect a second venous consequence

Exudates may complicate nonischemic retinal vein occlusion.

Material accumulating outside vessels changes the retinal appearance.

This differs mechanistically from the acute whitening of arterial infarction.

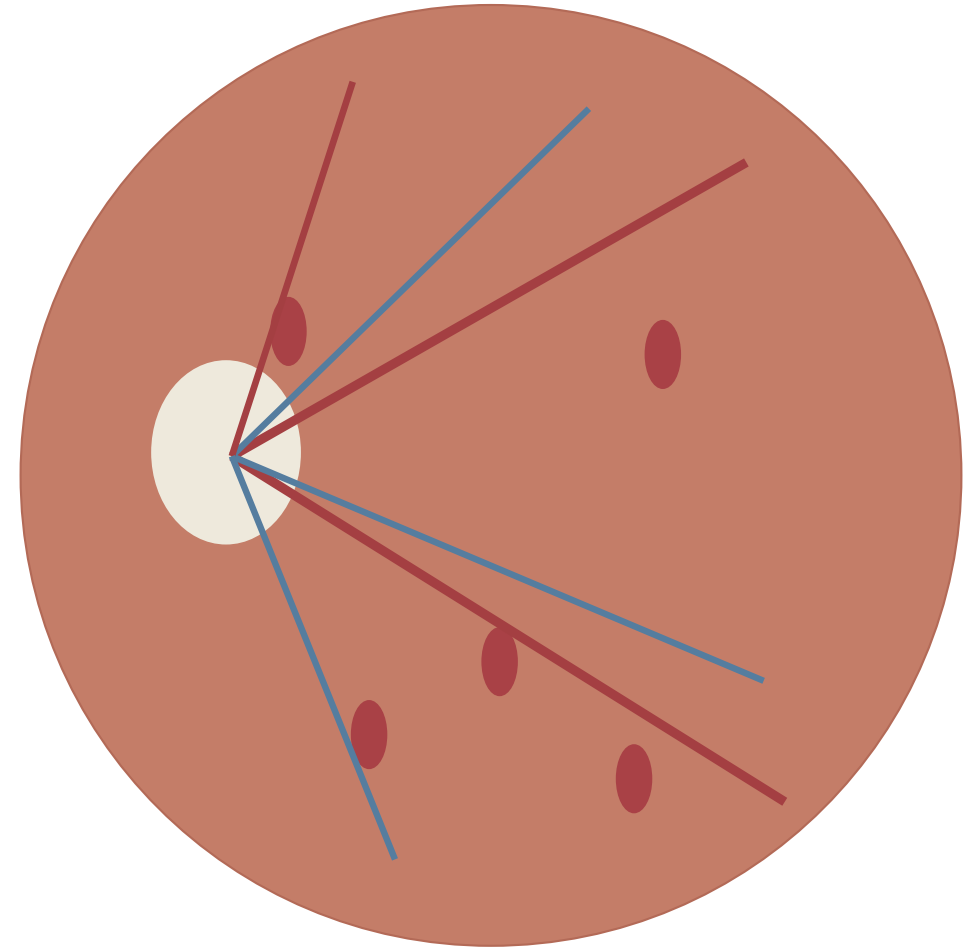


Macular edema threatens a critical retinal region

Macular edema is named as a complication of nonischemic vein occlusion.

The macula is central to the appearance being discussed in retinal vascular disease.

Edema is distinct from the diffuse infarct pattern of a central artery occlusion.



Hemorrhages — schematic

Venous occlusion need not be ischemic

Aspect	Nonischemic RVO	Ischemic RVO
Perfusion descriptor	Without significant ischemic designation	With retinal ischemia
Textbook complications	Hemorrhage, exudates, macular edema	VEGF upregulation
Neovascular sites	Not specified in the focused passage	Retina, optic nerve head, iris
Additional complication	Macular edema	Neovascular angle-closure glaucoma

Interpret retinal hemorrhage separately from ischemia

A venous occlusion may produce hemorrhage or edema without the neovascular sequence.

Ischemic venous disease has an additional angiogenic pathway.

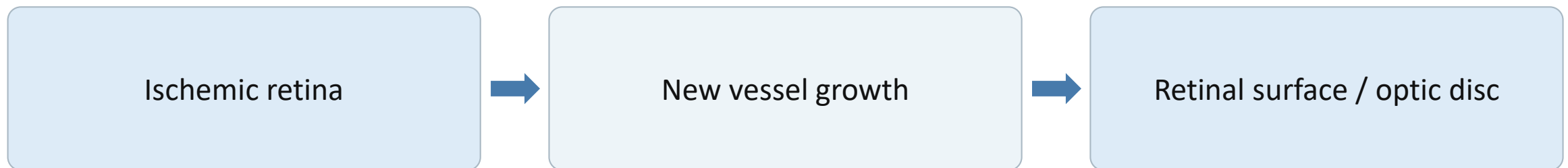
Do not infer all complications of every case from a single visible abnormality.

Retinal neovascularization has a defined location

Adjacent Robbins material distinguishes intraretinal changes from new surface vessels.

Surface new vessels breach the internal limiting membrane.

The source names new vessels on the optic disc or elsewhere on the retina.

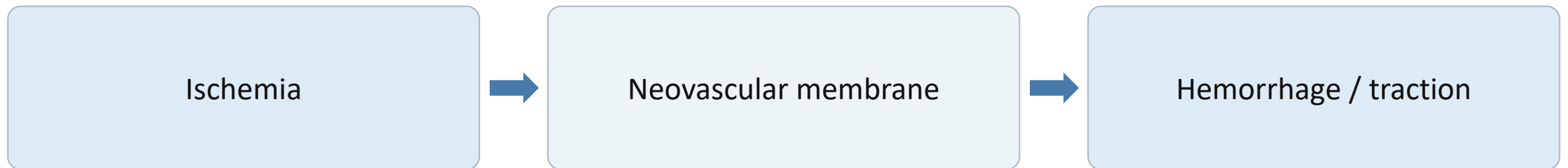


VEGF-mediated vessels may cause further injury

In adjacent retinal-vascular disease sections, fragile neovascular membranes are associated with hemorrhage and traction.

These are mechanisms shared across ischemic retinal vasculopathies.

They provide context, not a claim that every vein occlusion has these sequelae.



Retinal artery versus vein: Morphology

Morphologic clue	Arterial occlusion	Venous occlusion
Primary tissue event	Infarction	With or without ischemia
Retinal color change	Whitening/opacity	Hemorrhages and exudates may be present
Macular change	Foveal red contrast in central occlusion	Macular edema in nonischemic disease
Angiogenesis	Not the characteristic mechanism in the focused section	VEGF-driven in ischemic disease

Retinal artery versus vein: Mechanism

Step	Arterial mechanism	Venous mechanism
Initiator	Thromboembolism; Possibly atherosclerotic thrombosis	Venous occlusion; Branch stasis may relate to AV compression
Direct consequence	Abrupt loss of arterial supply	Ischemic or nonischemic consequences
Structural change	Acute swelling and opacity	Hemorrhage/exudate/edema or neovascularization
Important outcome	Retinal infarction and blindness	Neovascular glaucoma if iris angiogenesis develops

The same fundus feature can mislead

A cherry-red spot alone is not specific for thromboembolic occlusion.

Retinal whitening depends on optical masking of choroidal color.

Hemorrhage and exudate are not interchangeable with infarct opacity.

Case 3: Ischemic retinal vein occlusion

After retinal venous occlusion, new vessels appear on the iris.

Which retinal signal links ischemia to the iris?

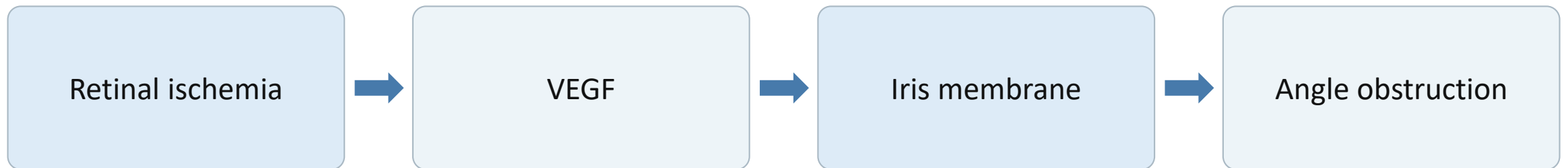
How can these new vessels eventually elevate intraocular pressure?

Case 3: Derive the complete causal sequence

Ischemic retina upregulates VEGF and other proangiogenic mediators.

Iris fibrovascular membrane formation and contraction obstruct the drainage angle.

Reduced trabecular outflow produces secondary neovascular glaucoma.



Case 4: Nonischemic venous consequences

A venous occlusive process is associated with macular edema, exudates, and hemorrhage.

Which of the two textbook venous patterns emphasizes these lesions?

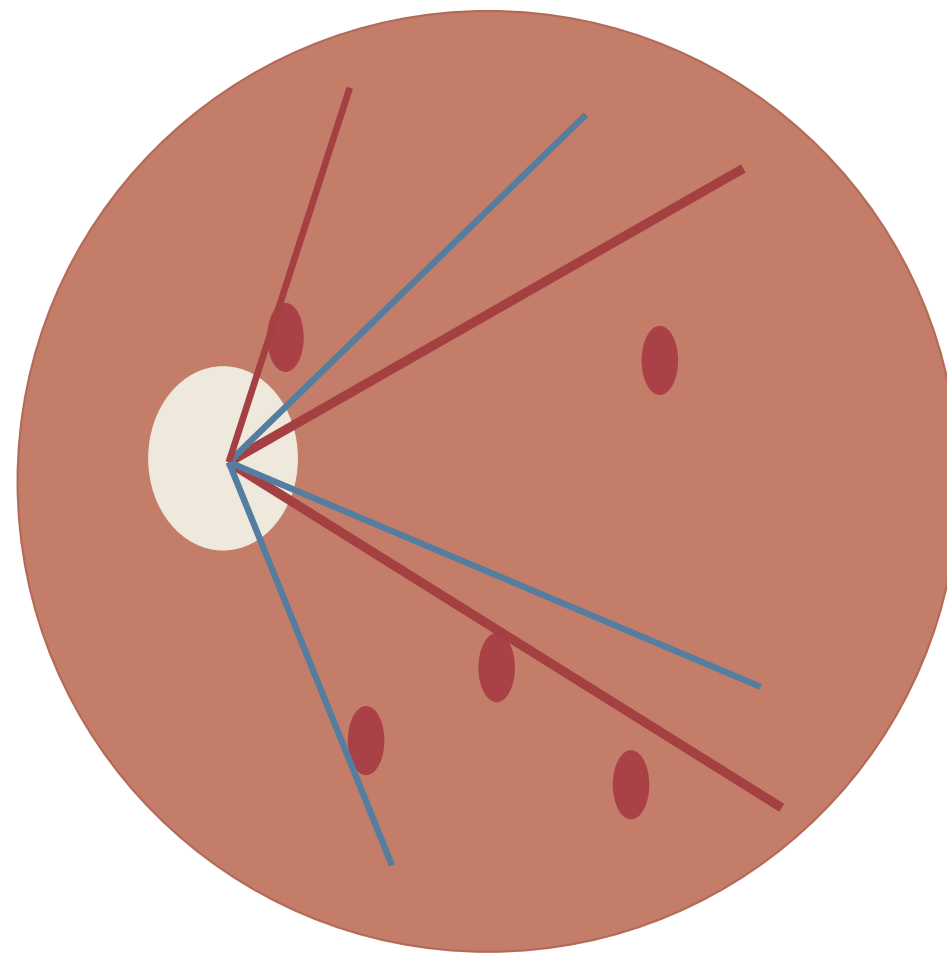
Does their presence alone prove iris neovascularization?

Case 4: Avoid an unwarranted inference

The nonischemic pattern is explicitly associated with these retinal changes.

The ischemic form has the prominent VEGF-driven neovascular sequence.

A specific ischemic classification cannot be assigned from these findings alone.



Hemorrhages — schematic

A mechanism-based approach to fundus signs

Visible clue	Immediate interpretation	Potential lesion to consider
White retinal territory	Swollen opaque retina masks choroid	Retinal artery infarction
Red fovea on pale retina	Relatively transparent foveal window	Central artery occlusion or storage condition
Hemorrhage, exudate, edema	Venous-associated retinal injury	Nonischemic vein occlusion
Retinal / disc / iris new vessels	Response to retinal ischemia	Ischemic vein occlusion

Final integration: Four causal pathways

Atherosclerotic narrowing or emboli → arterial interruption → infarction.

Infarct swelling → retinal opacity; Spared thin fovea → red spot.

Venous ischemia → VEGF → retinal/iris neovascularization.

Iris membrane contraction → angle obstruction → neovascular glaucoma.

Five high-yield distinctions

Branch-artery infarct is segmental; Central-artery infarct may be diffuse.

The red spot is transmitted choroidal color, not diagnostic of a unique etiology.

Storage disease and arterial occlusion cause different forms of perifoveal opacity.

Nonischemic RVO: Hemorrhage, exudates, macular edema.

Ischemic RVO: VEGF-driven angiogenesis and possible neovascular glaucoma.

References and source boundaries

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. (2026), Chapter 29, “The Eye,” pp. 1201–1205.

Primary topic: “Retinal Artery and Vein Occlusions,” p. 1204.

Context: “Retinal Vascular Disease,” “Diabetes Mellitus,” and “Glaucoma” sections in the same chapter.

Fundus, retinal-layer, vessel, and outflow visuals here are original editable schematics, not clinical photographs.