

Subarachnoid Hemorrhage and Ruptured Saccular Aneurysm

Cerebrovascular pathology · mechanisms, morphology, clinical consequences

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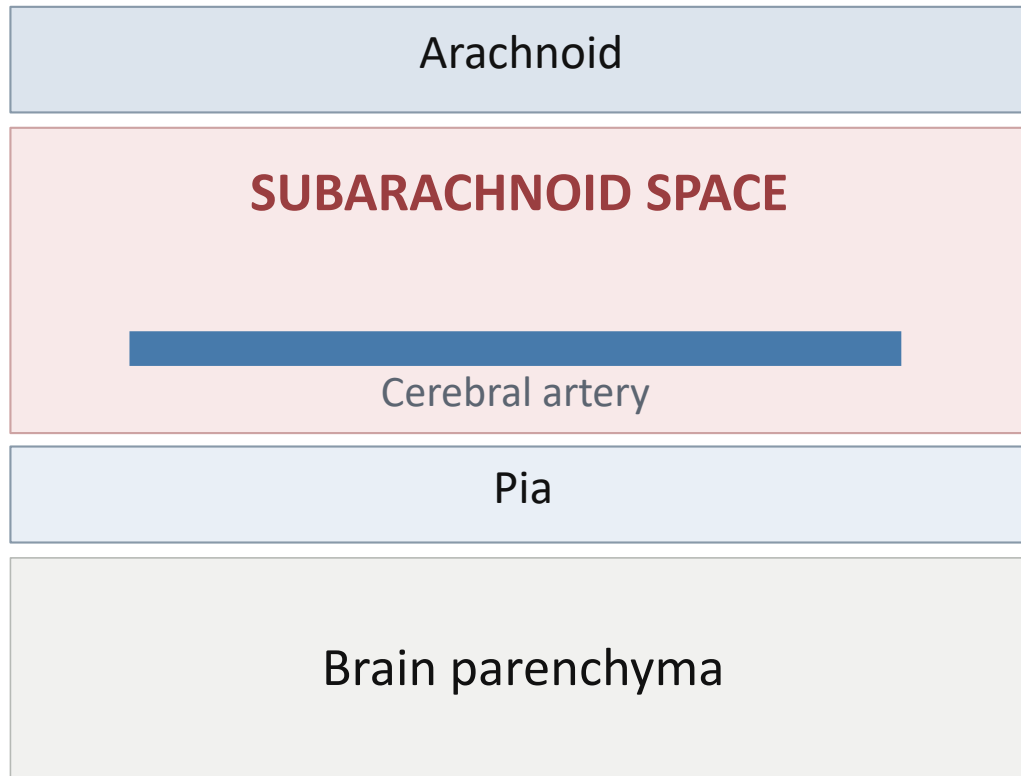
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Learning objectives

- Distinguish subarachnoid hemorrhage from intraparenchymal hemorrhage.
- Explain where saccular aneurysms arise and why their walls are vulnerable.
- Connect aneurysm rupture to immediate symptoms and delayed complications.
- Interpret morphologic features and major clinicopathologic differentials.

Emphasis: Mechanisms, pathologic morphology, and cause–effect reasoning.

Subarachnoid hemorrhage is compartment-defined



- Blood accumulates in the space between the arachnoid and pia mater.
- Cerebral arteries course within the subarachnoid compartment.
- A ruptured artery can therefore expose surrounding vessels and meninges to blood.

The anatomic compartment explains both the initial hemorrhage and secondary effects.

Intracranial hemorrhage: The compartment matters

Subarachnoid space

- Blood outside the brain parenchyma, within leptomeningeal spaces.
- Aneurysm, malformation, or trauma are central etiologic categories.

Intraparenchymal tissue

- Blood dissects or collects within brain substance.
- Hypertension, cerebral amyloid angiopathy, trauma, and tumors are prominent contexts.

Parenchymal hematomas may secondarily extend into the subarachnoid space.

Two broad routes to subarachnoid blood

Traumatic

Vascular injury accompanies head trauma, often with other serious lesions.

Spontaneous

A vascular abnormality—especially a ruptured saccular aneurysm—is the dominant pattern.

Etiology should be inferred from the clinical and morphologic context, not from location alone.

Nontraumatic subarachnoid hemorrhage: Major causes

Saccular aneurysm

Most frequent cause of spontaneous subarachnoid hemorrhage.

Other vascular lesions

Arteriovenous malformations and other vascular lesions may bleed.

Other causes

Hematologic disturbances, tumors, or extension from intracerebral hemorrhage.

“Nontraumatic” does not mean “aneurysmal” in every patient.

Ruptured aneurysm versus extension from brain hemorrhage

Primary arterial rupture

- Saccular aneurysm tears, commonly near the circle of Willis.
- Blood primarily enters the subarachnoid space; It may also enter brain tissue.

Secondary extension

- An intracerebral hemorrhage originates within parenchyma.
- The expanding hemorrhage may reach ventricles or subarachnoid spaces.

Identify the likely primary source of bleeding.

Intracranial aneurysm is not one morphology

Saccular (“berry”)

Focal, sac-like arterial outpouching; The common intracranial type.

Fusiform

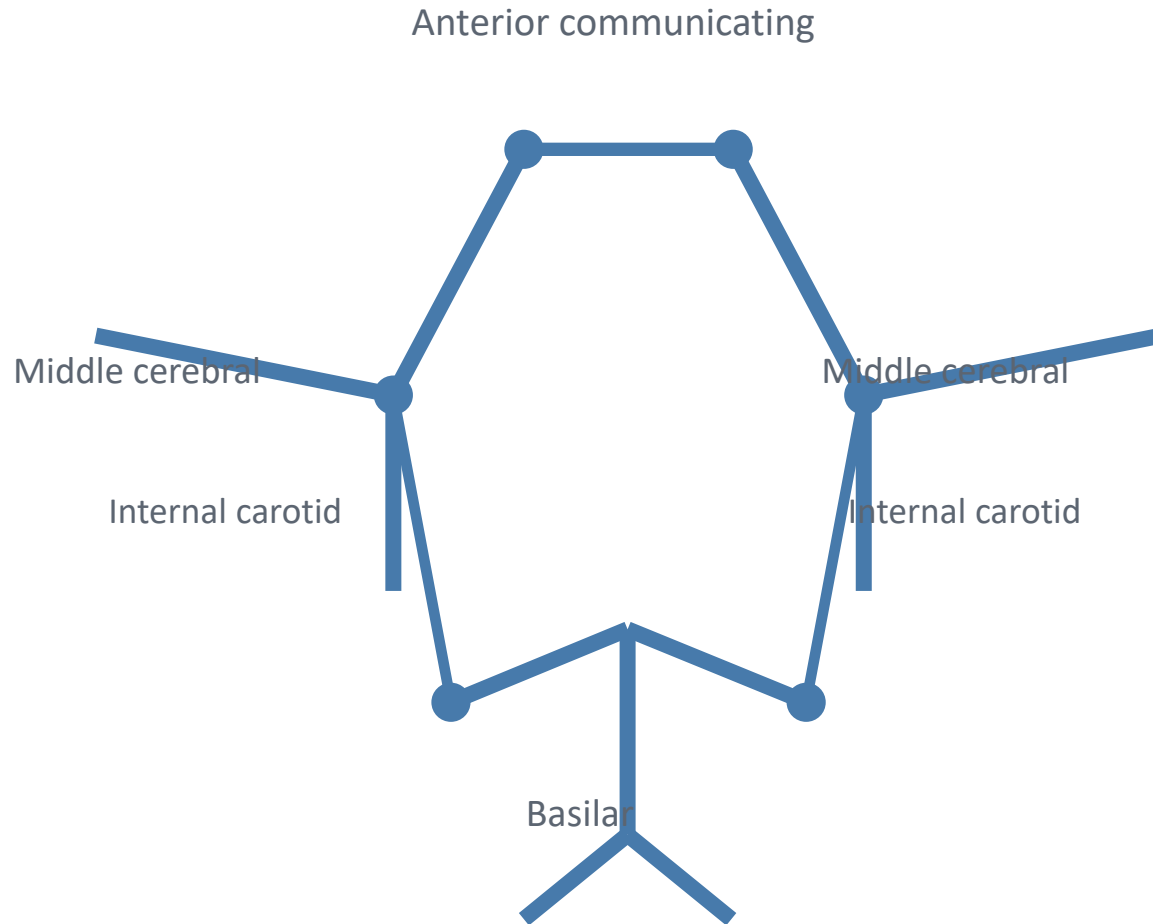
Circumferential enlargement;
Atherosclerotic examples often affect the basilar artery.

Other patterns

Mycotic, traumatic, and dissecting aneurysms have distinct pathologic origins.

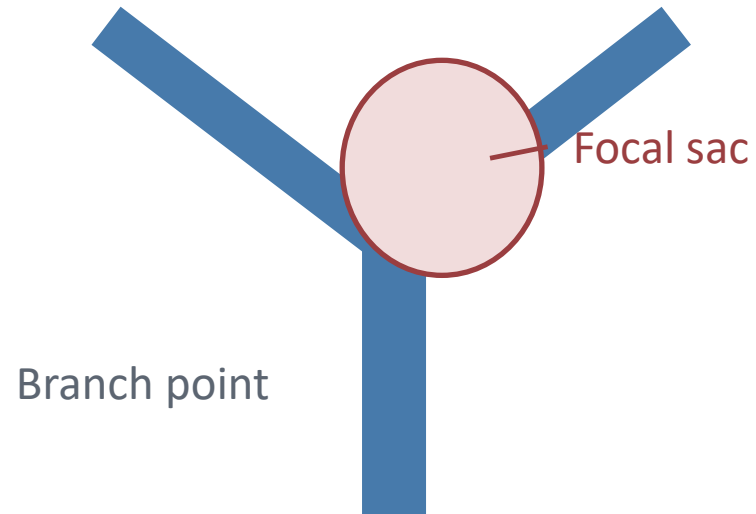
Use vascular geometry and underlying pathology—not the word “aneurysm” alone.

The circle of Willis: Key arterial connections



- Internal carotid arteries contribute to the anterior circulation.
- Anterior communicating artery links the anterior cerebral arteries.
- Basilar and posterior cerebral arteries connect to the posterior circulation.

Why arterial branching points are important



- Saccular aneurysms usually appear at branching sites or immediately beyond them.
- These are regions exposed to complex hemodynamic forces.
- A localized vessel-wall deficit creates a weak point under pulsatile pressure.

Vascular topology and wall structure must be considered together.

Distribution: Anterior circulation predominates

- Approximately 90% of saccular aneurysms near major branch points in the anterior circulation.
- The circle of Willis and vessels just beyond it are important locations.
- A distribution pattern helps frame gross examination and vascular mapping.

Anatomic predominance is not a substitute for locating an individual lesion.

Multiple saccular aneurysms are possible

- According to the source, approximately 20%–30% of affected individuals have multiple aneurysms.
- Finding one sac does not establish that it was the source of hemorrhage.
- Look for evidence of rupture and the relationship to adjacent blood distribution.

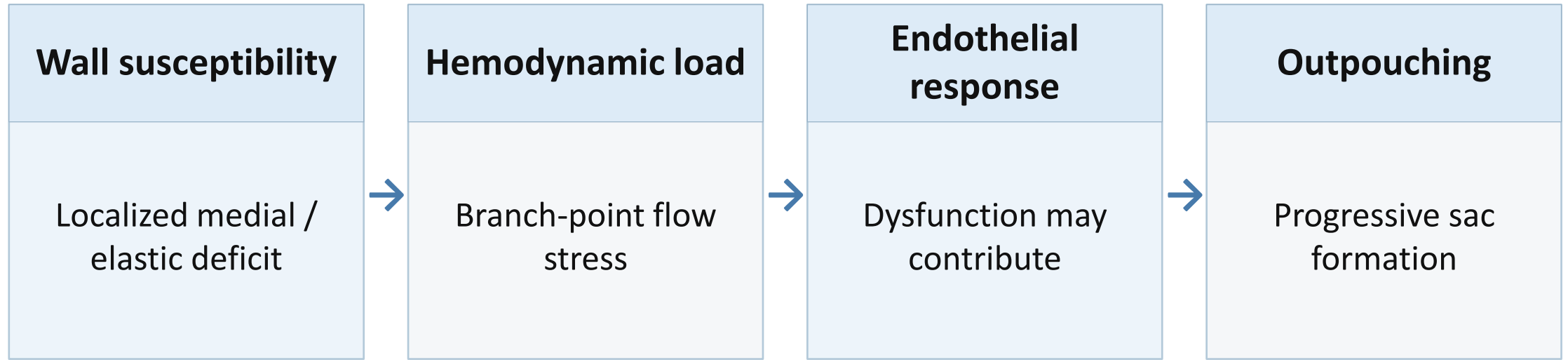
The causal lesion must be distinguished from a coincidental unruptured aneurysm.

Incidental aneurysm and ruptured aneurysm differ

- Community-based radiologic studies in the text identify saccular aneurysms in about 2% of the population.
- The presence of an aneurysm does not itself demonstrate current bleeding.
- Clinical presentation and pathologic evidence establish whether rupture occurred.

Prevalence describes presence; Rupture concerns a subsequent event.

Mechanistic overview of aneurysm development



The source presents a multifactorial model rather than a single proven cause.

The initiating cause remains incompletely defined

- The etiology of most saccular aneurysms remains uncertain.
- Abnormal smooth muscle and elastic architecture imply a structural predisposition.
- Hemodynamic and endothelial influences are proposed contributors, not a complete causal explanation.

Separate established morphologic findings from proposed mechanisms.

“Congenital aneurysm” is an imprecise term

Underlying predisposition

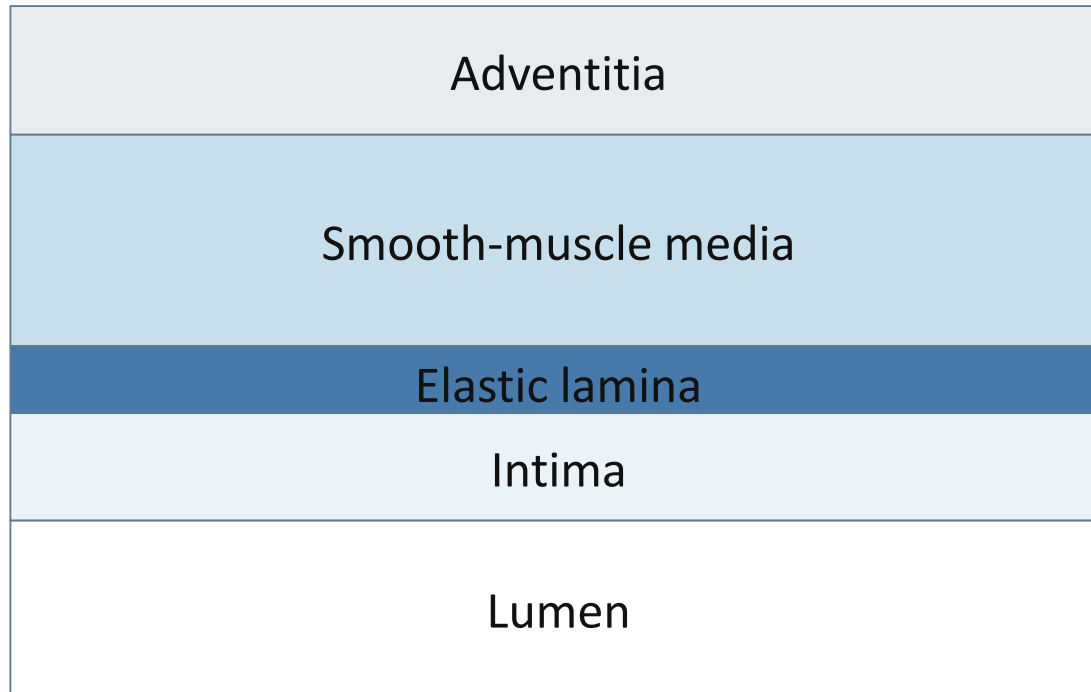
- The abnormal vessel-wall architecture suggests a developmental susceptibility.
- Inherited connective-tissue conditions support a role for structural vulnerability.

Actual aneurysm formation

- Sacs are not present at birth.
- The outpouching develops over time under the influence of local vessel-wall forces.

A congenital predisposition does not imply a congenital visible aneurysm.

Normal arterial wall: Layers that resist stress



- Intima lines the arterial lumen.
- Internal elastic lamina and muscular media support mechanical integrity.
- Adventitia provides an external connective-tissue investment.

Schematic cross-sectional layering; Not a histologic micrograph.

Saccular aneurysm wall: Key structural losses

Adventitia
Hyalinized intima
No elastic lamina / media
Lumen

- Smooth muscle and the internal elastic lamina are absent within the aneurysm sac.
- The wall largely comprises thickened, hyalinized intima with adventitial covering.
- The absence of normal media makes the sac structurally distinct from its parent artery.

Structural deficiency supplies a pathologic explanation for rupture.

Parent vessel versus aneurysm sac

Parent artery near the neck

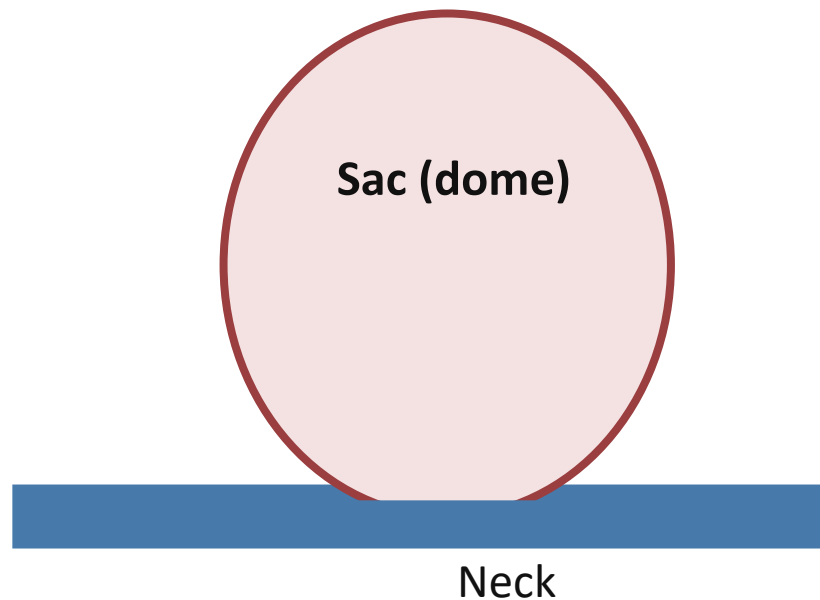
- May show intimal thickening.
- The adjacent media can be attenuated.
- Wall architecture transitions toward the aneurysm neck.

Aneurysm sac

- Internal elastic lamina absent.
- Smooth muscle absent.
- Fibrous hyalinized intima and adventitial covering persist.

Abrupt change in load-bearing layers helps define the weak segment.

The aneurysm neck and dome are not equivalent



- The neck joins the parent arterial lumen; It can be narrow or wide.
- The thin-walled dome expands away from the artery.
- Rupture commonly occurs near the sac apex.

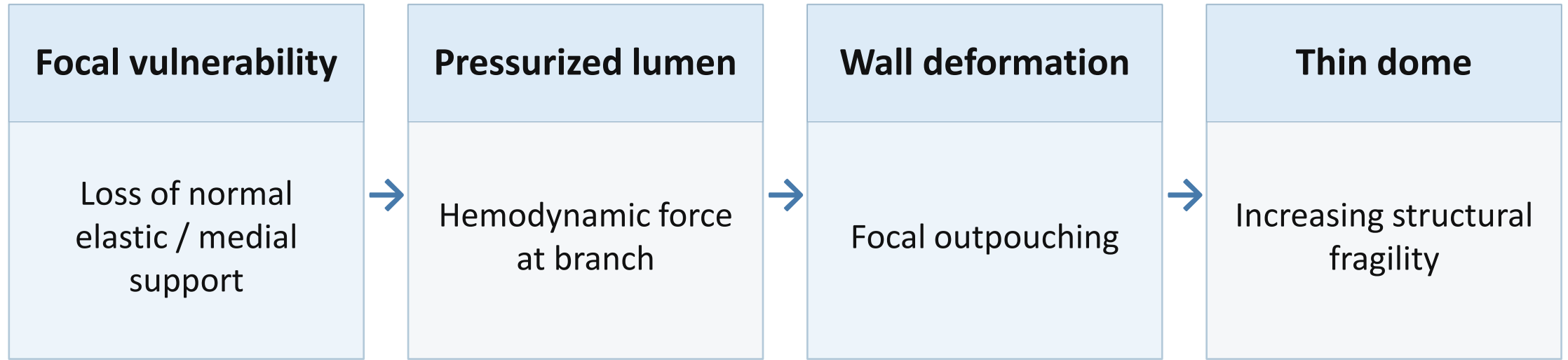
Localizing the rupture point requires examining the sac, not merely identifying it.

Endothelial dysfunction: A proposed link

- Complex flow at a branching artery produces localized hemodynamic stress.
- The textbook proposes endothelial dysfunction as an important contributor.
- A weakened elastic–muscular wall may subsequently remodel into a sac.

Do not infer that endothelial dysfunction alone is sufficient for aneurysm formation.

Pathology connects wall defect to outpouching



This diagram depicts the explanatory sequence, not a proven universal chronology.

Most aneurysms are sporadic

- Most cases occur without a recognized hereditary syndrome.
- However, first-degree relatives of affected patients have an increased frequency of aneurysms.
- Familial enrichment indicates genetic susceptibility in at least a subset.

“Sporadic” and “genetically influenced” are not mutually exclusive.

Inherited conditions associated with aneurysms

Vascular connective tissue

Vascular Ehlers–Danlos syndrome (type IV) and Marfan syndrome.

Other genetic disorders

Autosomal dominant polycystic kidney disease and neurofibromatosis type 1.

Associated arterial conditions

Coarctation of the aorta and extracranial fibromuscular dysplasia.

Associations are susceptibility clues; No single condition accounts for most cases.

Smoking and hypertension are important risk factors

- Smoking and hypertension are listed as predisposing factors.
- Both can coexist with structural or familial vulnerability.
- Risk-factor recognition is relevant even when no Mendelian disorder is present.

Aneurysm formation is better framed as a multi-hit vessel-wall process.

Susceptibility operates at different levels

Inherited structure

Predisposition affecting
vessel wall integrity.

Local anatomy

Arterial branch points
concentrate complex flow.

Acquired exposures

Smoking and hypertension
add risk during life.

None of these alone provides a definitive explanation for every aneurysm.

Gross appearance of an unruptured sac

- Usually a bright red, shiny, thin-walled outpouching.
- The wall may appear translucent on gross examination.
- The lesion lies at an arterial branch point along or just beyond the circle of Willis.

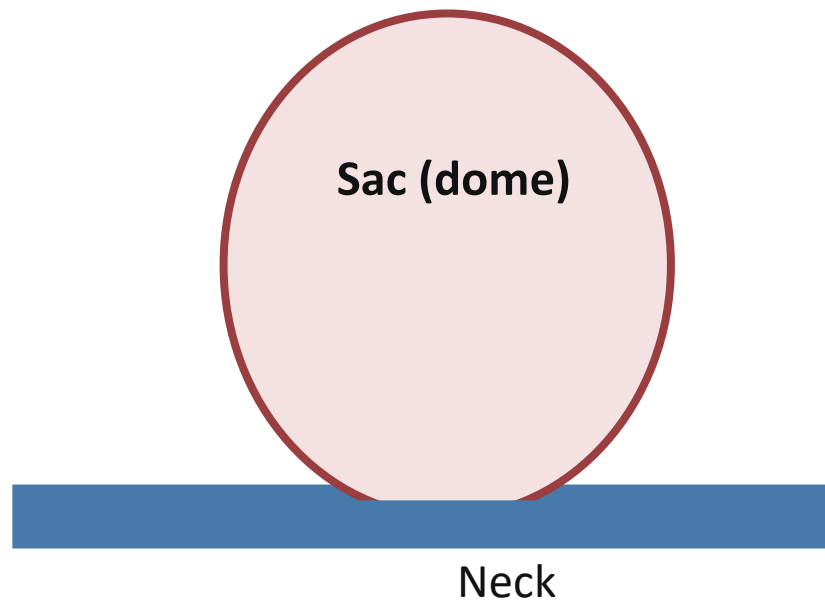
Distinguish a true focal arterial sac from enlarged segments of adjacent vessels.

Gross size is variable

- Observed diameters range from a few millimeters to several centimeters.
- Examples reaching 2–3 cm.
- Larger size is associated with greater risk of rupture, but a small sac is not necessarily harmless.

Sac size contributes to risk but does not replace assessment of wall morphology.

Gross anatomy: Lumen, neck, and sac



- The sac communicates with the parent artery through its neck.
- Wide and narrow neck configurations occur.
- The outer contour does not necessarily reveal mural atherosclerosis or thrombus.

The diagram is schematic; Gross examination remains essential.

Secondary changes in the aneurysm wall or lumen

Atheroma

Atherosclerotic plaques may alter the vessel wall.

Calcification

Mineralization may be encountered in the sac wall.

Thrombus

Intraluminal thrombotic material may partly occupy the sac.

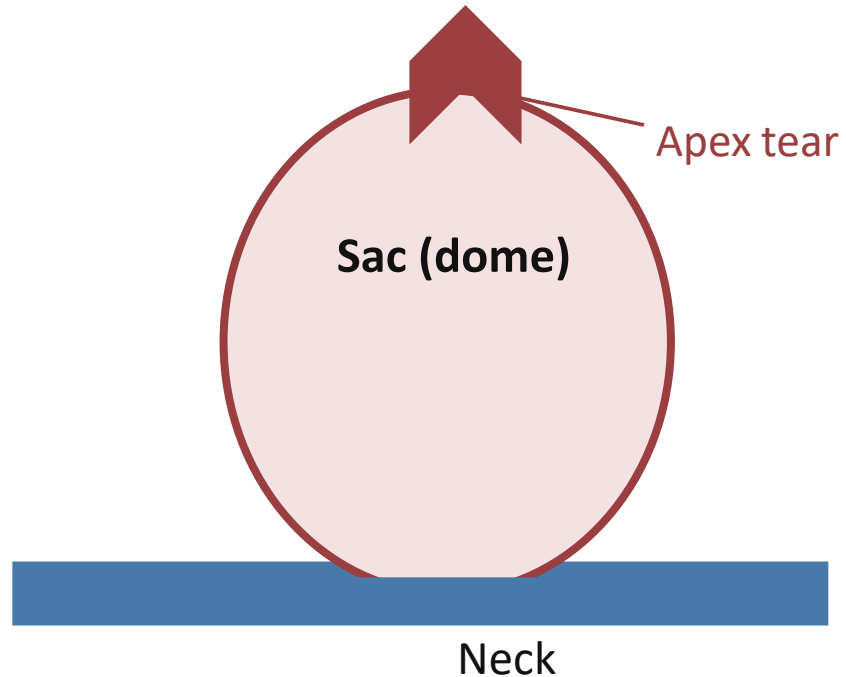
Secondary findings modify morphology but do not define the basic saccular pattern.

Old bleeding can leave a macroscopic clue

- Orange-brown discoloration of adjacent meninges or brain suggests preceding hemorrhage.
- This pigmentary change may coexist with an otherwise unruptured-appearing sac.
- Historical bleeding should not be confused with evidence of a new rupture.

Pigment supports prior hemorrhage; It does not by itself date the current event.

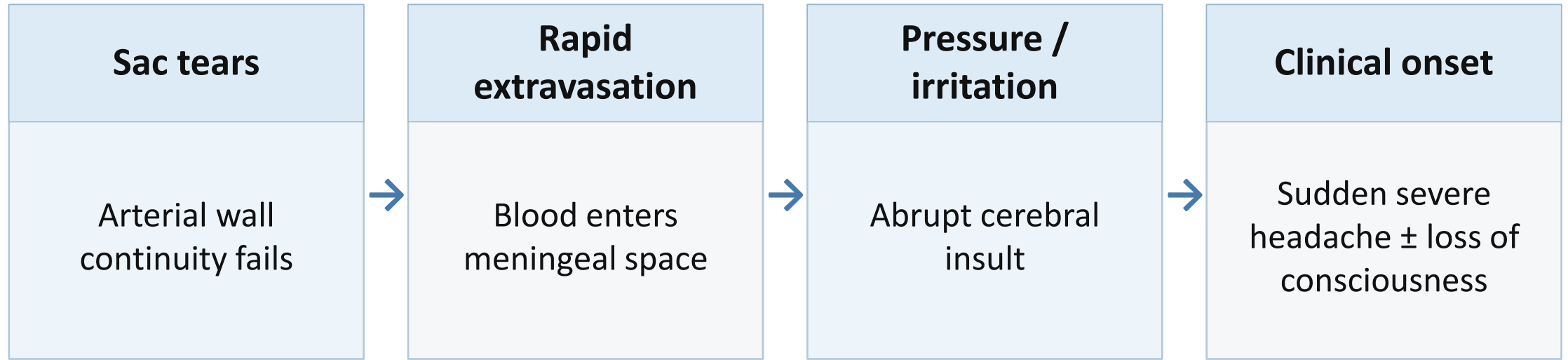
Rupture favors the aneurysm apex



- A focal defect at the dome permits escape of arterial-pressure blood.
- The immediate destination is usually the subarachnoid compartment.
- Blood may also dissect into nearby brain tissue.

Apex localization links structural morphology to distribution of hemorrhage.

Rupture: From arterial lumen to clinical injury



The rapid onset follows blood entering the subarachnoid space under arterial pressure.

Pure subarachnoid versus mixed hemorrhage

Subarachnoid distribution

- Blood tracks through leptomeningeal spaces and basal cisterns.
- Major surrounding arteries may be bathed in blood.

Combined parenchymal involvement

- Rupture may also extend into brain substance.
- A mixed distribution does not negate an aneurysmal origin.

Search for the initial vascular event when compartments overlap.

Age and sex patterns at rupture

- Rupture is reported most frequently in the fifth decade.
- Women are described as slightly more frequently affected than men.
- Demographic patterns are tendencies, not diagnostic criteria.

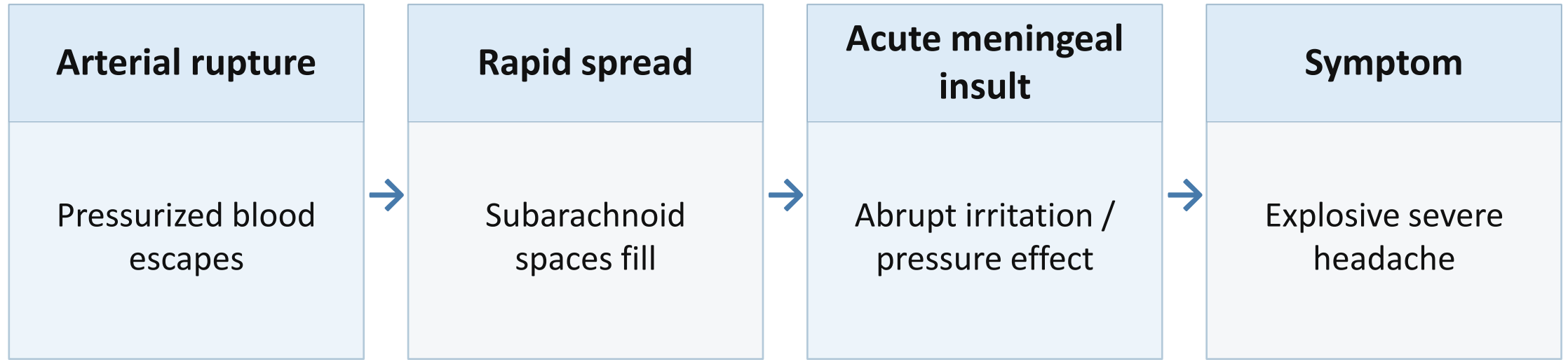
Clinical suspicion must not be restricted to one age or sex group.

Exertion may precede rupture

- An aneurysm can rupture without a recognizable trigger.
- The source notes an association with acute pressure-related exertion in a subset.
- Straining at stool or sexual orgasm are provided as examples.

A precipitating activity is neither necessary nor sufficient to prove rupture.

Why headache begins suddenly



The tempo is characteristic: Onset is abrupt rather than gradual.

The cardinal symptom: Thunderclap headache

- Abrupt, excruciating headache is a typical presentation.
- Patients may describe it as the worst headache experienced.
- Rapid deterioration or loss of consciousness may accompany onset.

The clinical syndrome is an emergency pattern; Morphology supplies its causal explanation.

Loss of consciousness can evolve rapidly

- Very early impairment of consciousness reflects the severity of the abrupt intracranial event.
- The source describes patients who lose consciousness quickly after rupture.
- Patients surviving the initial event may regain consciousness within minutes.

Early clinical improvement does not exclude a catastrophic vascular lesion.

The first hemorrhage may be fatal

- Substantial immediate mortality after first rupture.
- A large hemorrhage can produce abrupt neurologic collapse.
- Early survivors remain vulnerable to further hemorrhage and ischemic complications.

Initial survival does not mean the vascular lesion is no longer dangerous.

Rebleeding worsens prognosis

- Recurrent hemorrhage is common among survivors.
- The timing of a subsequent bleed is unpredictable.
- Each new hemorrhagic episode is associated with a worse prognosis.

Recognize ongoing vulnerability after the index event.

Aneurysm size influences rupture risk

- The source identifies aneurysm size as a predictor of bleeding risk.
- Larger aneurysms have greater propensity to rupture.
- Rupture remains possible across the size spectrum.

Size modifies risk but does not determine the individual course.

Immediate injury and delayed injury are distinct

At rupture

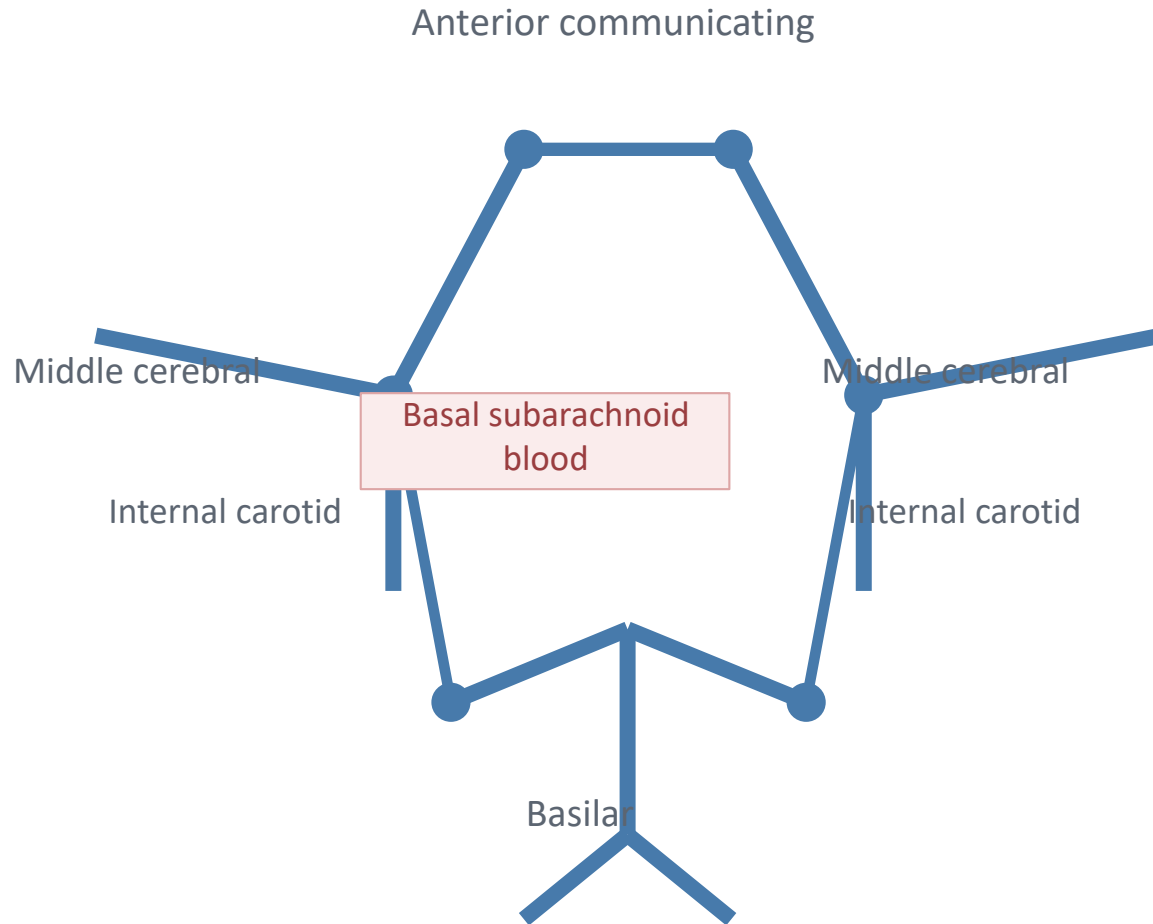
- Arterial blood suddenly enters subarachnoid space.
- Severe headache and abrupt loss of consciousness may occur.
- Repeat bleeding remains possible.

After rupture

- Vasospasm may create additional ischemic injury.
- Meningeal fibrosis may compromise CSF flow or resorption.
- Manifestations can arise after initial recovery.

Clinical risk evolves over time; The acute event is not the entire disease.

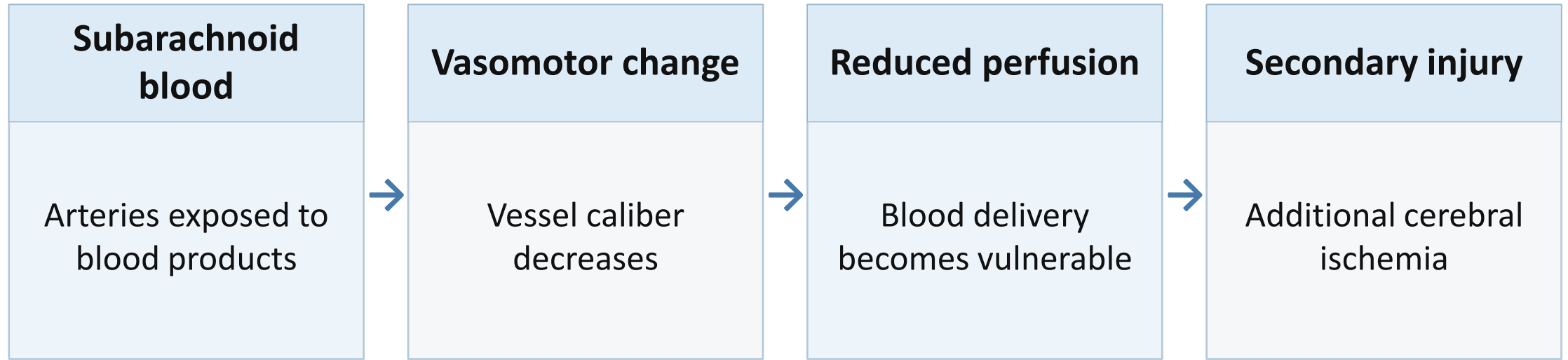
Basal subarachnoid blood threatens major arteries



- The circle of Willis lies in basal subarachnoid spaces.
- Extravasated blood may surround large conducting vessels.
- Vasospasm in these vessels can compromise major vascular territories.

Basal hemorrhage creates a particularly important setting for vasospasm.

Posthemorrhagic vasospasm: Clinicopathologic sequence



Additional ischemic damage can appear after the initial hemorrhage.

Vasospasm develops over the first days

- Vasospasm-related ischemic risk emerges during the first days after hemorrhage.
- It can occur after subarachnoid hemorrhage of different causes.
- The concern is greatest when blood bathes major vessels of the circle of Willis.

Timing and vessel exposure connect hemorrhage distribution to risk.

Mediators of vasospasm: Proposed pathways

- Endothelins have been proposed as contributors.
- Nitric oxide signaling is implicated in hypotheses of vasomotor dysregulation.
- Arachidonic acid metabolites may also participate.

These are proposed mediators in the source, not a single definitive mechanism.

Hemorrhage and ischemia can coexist in one illness

Primary mechanism: Hemorrhage

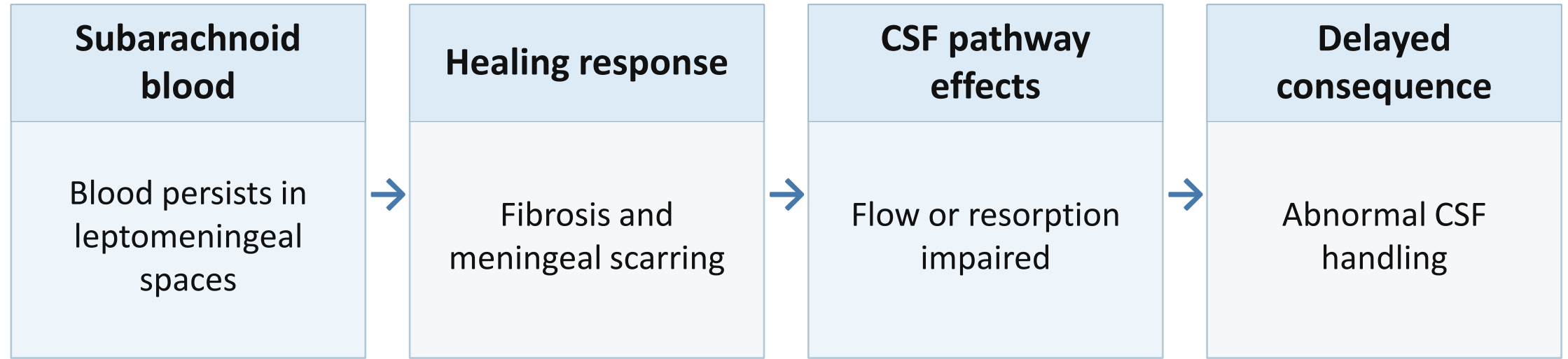
- A ruptured arterial wall is the initiating event.
- Blood enters the subarachnoid space.

Secondary mechanism: Ischemia

- Arteries exposed to blood may constrict.
- Reduced perfusion can produce additional cerebral injury.

The delayed infarct risk does not transform the original event into an ischemic stroke.

Late meningeal response after hemorrhage



Fibrotic repair can turn an acute hemorrhage into a chronic CSF problem.

Meningeal fibrosis follows the healing phase

- The leptomeninges respond to extravasated blood during resolution.
- Fibrosis and scarring can alter spaces through which CSF normally circulates.
- The damage is architectural rather than a renewed arterial rupture.

Late scarring is a distinct mechanism from acute hemorrhage.

CSF flow and resorption are vulnerable

Ventricular CSF



Subarachnoid spaces



Resorption pathways

Scarring affects flow and/or resorption

- Subarachnoid spaces participate in CSF circulation and resorption.
- Adhesions can obstruct flow through these pathways.
- Meningeal scarring can also interfere with normal CSF resorption.

Posthemorrhagic disturbance of CSF handling is structurally plausible.

Obstructed CSF flow versus reduced resorption

Flow obstruction

- Fibrotic adhesions may narrow available CSF pathways.
- The problem concerns movement between communicating spaces.

Impaired resorption

- Scarring can interrupt the usual CSF absorption route.
- The problem concerns fluid clearance rather than only transit.

The source describes both processes without quantifying their relative frequency.

Saccular aneurysm versus arteriovenous malformation

Saccular aneurysm

- Localized arterial sac at a branch point.
- Sac lacks normal media and internal elastic lamina.
- Apex rupture can cause subarachnoid hemorrhage.

Arteriovenous malformation

- Tangled enlarged channels with high-flow artery-to-vein shunting.
- Abnormal vascular walls are separated by gliotic tissue.
- May cause intraparenchymal or subarachnoid hemorrhage.

These abnormalities both bleed but have very different architectures.

Aneurysmal SAH versus hypertensive brain hemorrhage

Aneurysmal SAH

- Commonly begins with a ruptured branch-point sac.
- Arterial blood enters subarachnoid spaces.
- Acute vasospasm and later meningeal scarring are important.

Hypertensive intraparenchymal bleed

- Typically arises from fragile small vessels in deep brain regions.
- A parenchymal clot compresses adjacent brain and may enter ventricles.
- Secondary tissue ischemia can follow compression.

The dominant pathologic compartments and vessel lesions differ.

Aneurysmal SAH versus lobar amyloid-related bleed

Aneurysmal SAH

- Saccular arterial-wall defect at a major branch.
- Subarachnoid blood may be basal and widespread.

CAA-associated lobar hemorrhage

- Amyloid deposition weakens cortical and leptomeningeal vessels.
- Bleeding originates in lobar parenchyma and may reach the subarachnoid space.

Extension into the subarachnoid space does not establish a saccular aneurysm.

Morphologic checklist for a suspected saccular aneurysm

Location	Sac and neck	Wall and rupture
At an arterial branch of the circle of Willis or nearby major vessel.	Focal outpouching with a narrow or broad communication to parent artery.	Hyalinized fibrous wall; Smooth muscle / elastic lamina absent in the sac; Inspect apex.

A comprehensive interpretation integrates location, shape, wall, and bleeding.

Application: Abrupt collapse with severe headache

A middle-aged adult develops instantaneous, excruciating headache and quickly loses consciousness. At examination, blood is concentrated within basal subarachnoid spaces.

Question

What vascular lesion should be high on the differential?

Ruptured saccular aneurysm near the circle of Willis.

Arterial-pressure bleeding from a thin-walled branch-point outpouching explains the presentation.

Application: Deterioration after initial survival

A patient survives an acute subarachnoid hemorrhage and briefly improves, but develops new neurologic deficits during the following days.

Question

Which secondary mechanism is highlighted by Robbins?

Vasospasm with additional ischemic injury.

Arteries surrounded by extravasated blood may constrict, especially in basal hemorrhage.

Application: Delayed CSF disturbance

After a subarachnoid hemorrhage, a survivor develops a later disturbance in CSF circulation without a documented new bleed.

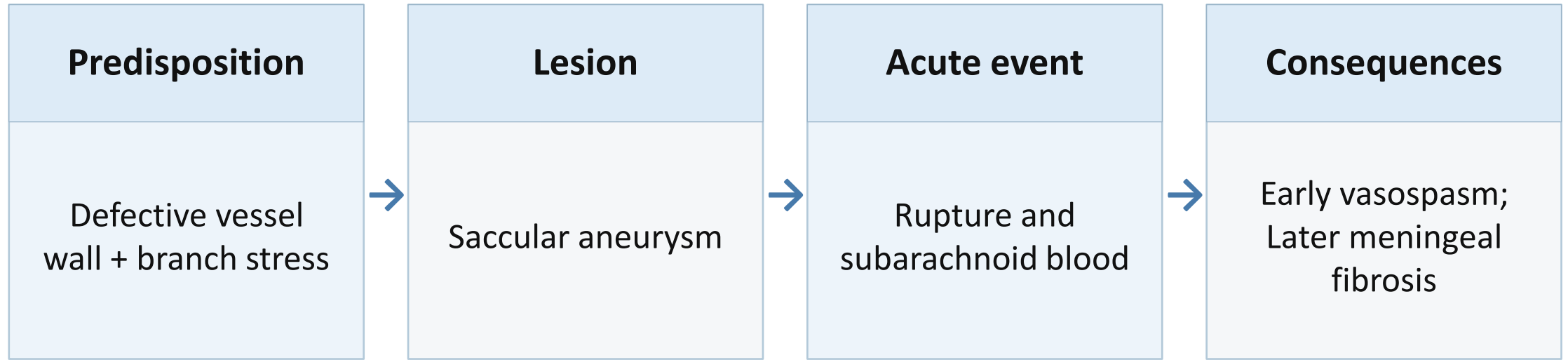
Question

What healing-phase lesion explains the complication?

Fibrosis and scarring of the leptomeninges.

Adhesions may obstruct CSF movement or interfere with its normal resorption.

Integrated sequence: Anatomy to late effects



Two downstream mechanisms can continue to injure the CNS after rupture.

Three diagnostic pathology distinctions to retain

Site

Subarachnoid versus
intraparenchymal bleeding.

Source

Saccular aneurysm versus
AVM or secondary
extension.

Time

Immediate hemorrhagic
effects versus early
vasospasm and late
fibrosis.

These axes support a concise cause-based clinicopathologic explanation.

Core concepts

- Saccular aneurysm is the leading cause of spontaneous subarachnoid hemorrhage in the source.
- Branch-point geometry, absent media / elastic lamina, and a thin fibrous sac explain its vulnerability.
- Rupture produces abrupt severe headache, possible collapse, and risk of rebleeding.
- Vasospasm threatens early ischemia; Meningeal scarring later threatens CSF circulation.

Interpret anatomy, pathology, and clinical time course together.

Reference and source interpretation

Primary source

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th edition (2026). Chapter 28: The Central Nervous System, pp. 1141–1143.

Figure convention

- Diagrams simplify anatomical and morphologic relationships and are labeled schematics—not diagnostic photomicrographs.
- An aneurysm is best interpreted by its arterial location, gross configuration, wall structure, and relationship to the site of hemorrhage.