

# Vascular Anomalies

---

Structural anomalies • hemodynamic consequences • clinicopathologic recognition

Cerebral aneurysms | Arteriovenous fistulas | Fibromuscular dysplasia | Anomalous coronary origin

**Dr Abu Suraih Sakhri**

MBBS, MD (Pathology)

[www.abusuraihsakhri.com](http://www.abusuraihsakhri.com)

# Learning outcomes

- Recognize how vascular wall architecture relates to blood flow and structural failure.
- Explain the morphogenesis and major consequences of each of the four anomalies.
- Differentiate aneurysm, direct arteriovenous shunt, stenosing dysplasia, and anomalous vessel course.
- Apply the mechanisms to focused clinical and anatomic cases.

**Recognize wall and flow**



**Explain four anomaly mechanisms**



**Predict anatomic complications**



**Apply clinicopathologic clues**

# An anomaly can be clinically silent yet consequential

- Many variations of the normal vascular supply produce no symptoms.
- Unrecognized anatomy can complicate surgery or an endovascular procedure.
- Clinical risk depends on vessel wall integrity, flow routing, and adjacent structures.

**Saccular aneurysm**

**AV fistula**

**Outpouching**

**Direct shunt**

**Fibromuscular dysplasia**

**Coronary anomaly**

**Stenosis / wall thinning**

**Abnormal origin / course**

# The four lesions disrupt different features

- Berry aneurysm: Focal cerebral arterial wall outpouching.
- Arteriovenous fistula: Direct artery–vein communication bypassing capillaries.
- Fibromuscular dysplasia: Abnormal arterial wall thickening and stenosis, sometimes with aneurysm formation.
- Anomalous coronary origin: A vessel originates or travels along an abnormal route.

**Saccular aneurysm**

**AV fistula**

**Outpouching**

**Direct shunt**

**Fibromuscular dysplasia**

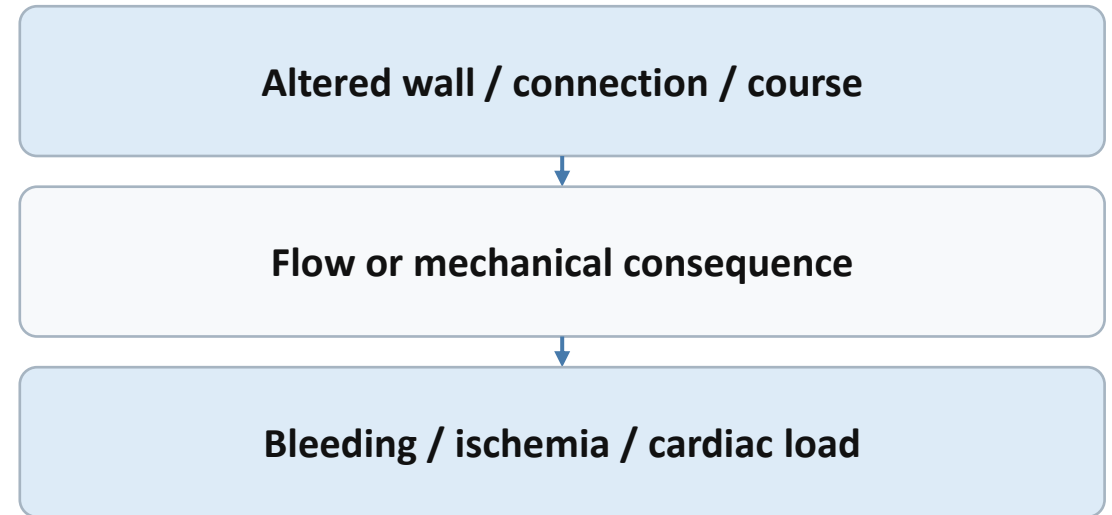
**Coronary anomaly**

**Stenosis / wall thinning**

**Abnormal origin / course**

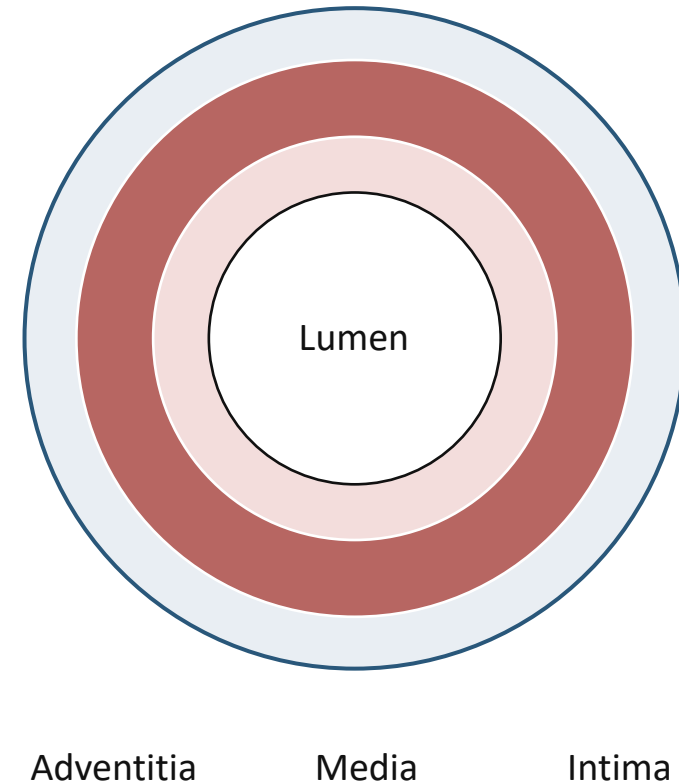
# Why the same variant may cause different outcomes

- Site determines whether hemorrhage threatens brain, kidney or myocardium.
- Structural weakness favors dilatation or rupture; Luminal loss favors ischemia.
- Large shunts impose volume load; Vulnerable coronary courses may limit perfusion during exertion.



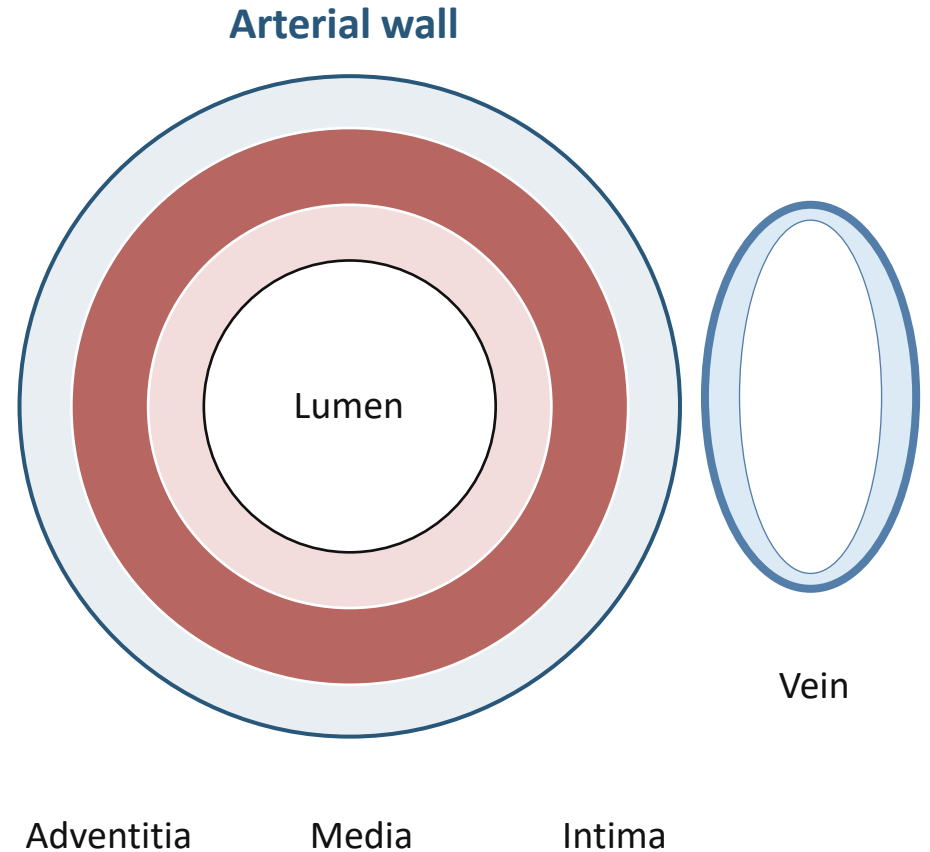
# Large-vessel walls have three basic layers

- Intima: Endothelial lining that interfaces directly with flowing blood.
- Media: Smooth muscle and elastic components adapted to local forces.
- Adventitia: Supportive extracellular matrix and connective tissue.
- Capillaries lack a conventional muscular media.



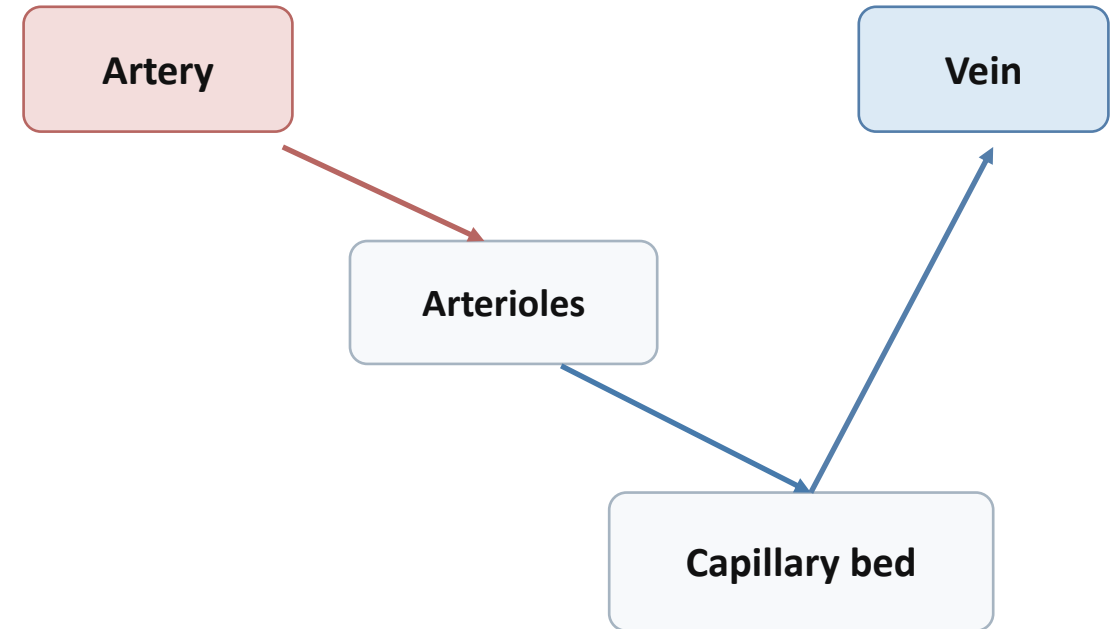
# Arteries and veins face different demands

- Arteries sustain higher pressures and have relatively developed media.
- Veins have more capacious lumens and thinner, less organized walls.
- Capillaries trade structural complexity for efficient exchange.



# A normal microvascular circuit prevents direct shunting

- Arterial inflow supplies arterioles and a capillary exchange bed.
- Flow then reaches postcapillary venules and larger veins.
- A direct artery-to-vein connection bypasses the exchange bed and alters hemodynamics.



# The site of a lesion matters as much as its size

- A cerebral arterial branch point may form a weak-wall outpouching.
- A renal artery lesion can reduce perfusion even without rupture.
- An interarterial coronary course can become hazardous during exercise.

**Saccular aneurysm**

**AV fistula**

**Outpouching**

**Direct shunt**

**Fibromuscular dysplasia**

**Coronary anomaly**

**Stenosis / wall thinning**

**Abnormal origin / course**

# Wall structure explains two divergent complications

- Weakness or attenuated media predisposes to aneurysmal dilatation.
- Focal intimal or medial hyperplasia plus fibrosis may narrow the lumen.
- The same disorder can have thickened segments adjacent to thinned, vulnerable ones.

**Wall attenuation → aneurysm**



**Fibrosis / hyperplasia → stenosis**



**Fistulous connection → shunting**

# A functional framework for classifying anomalies

- Assess whether the abnormality is an outpouching, a shunt, a narrowed segment, or a displaced course.
- Identify the affected vascular territory and the direction of flow.
- Predict bleeding, ischemia, hypertension, or cardiac loading from anatomy rather than the label alone.

**Saccular aneurysm**

**AV fistula**

**Outpouching**

**Direct shunt**

**Fibromuscular dysplasia**

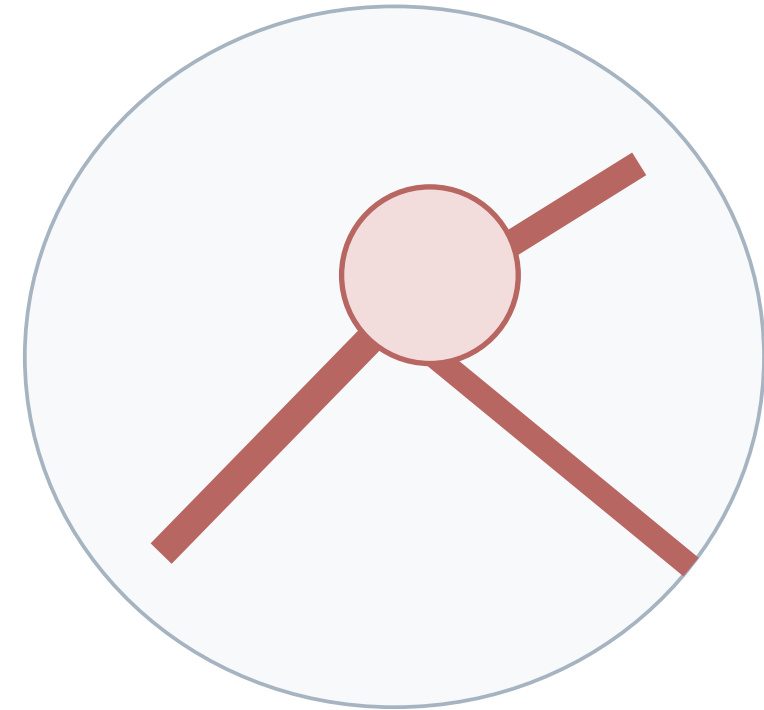
**Coronary anomaly**

**Stenosis / wall thinning**

**Abnormal origin / course**

## Saccular (“berry”) aneurysm: Core definition

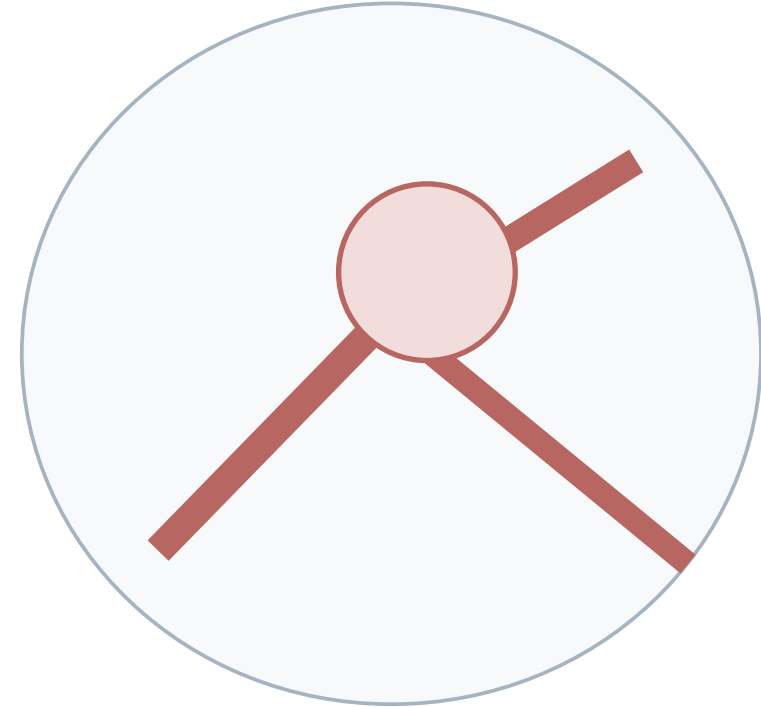
- A thin-walled saccular outpouching develops near cerebral arterial bifurcations.
- Saccular aneurysms are the most common type of intracranial aneurysm.
- Their principal catastrophic consequence is rupture with hemorrhage.



Saccular outpouching

# Why cerebral branch points are vulnerable

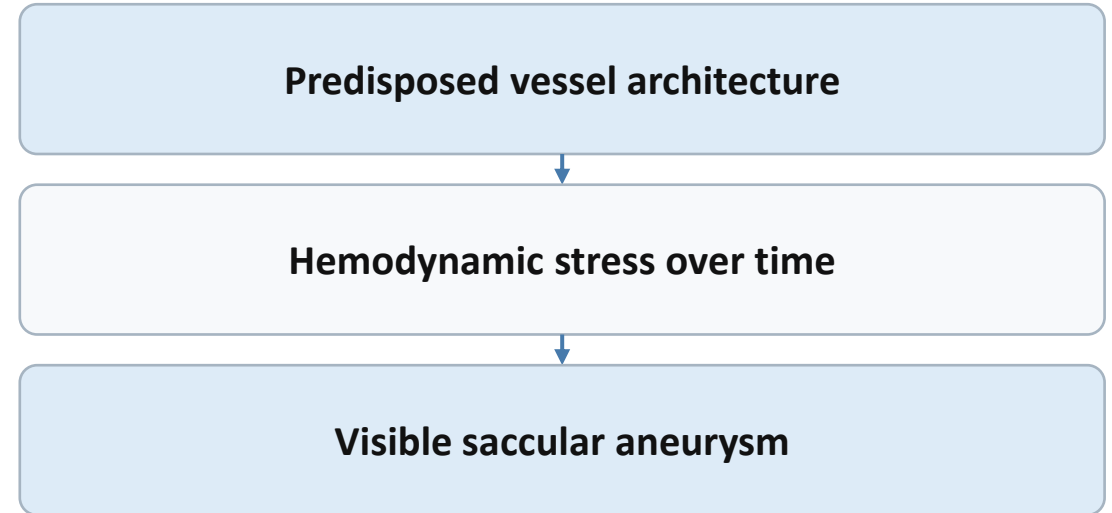
- Bifurcations concentrate complex hemodynamic stresses on the vessel wall.
- The affected wall lacks normal smooth muscle and internal elastic lamina in the sac region.
- Underlying media defects and acquired flow stress contribute to aneurysm development.



Saccular outpouching

## **“Congenital” does not mean present at birth**

- An underlying structural predisposition rather than a visible sac at birth.
- Aneurysms develop over time at susceptible arterial sites.
- Genetic and hemodynamic factors help explain why only some sites evolve into lesions.



# The critical wall defect is loss of reinforcement

- Normal cerebral artery depends on an intact media and internal elastic lamina.
- The sac lacks these structural components and is sustained by attenuated, fibrotic tissue.
- Blood pressure acts on a wall with diminished mechanical support.

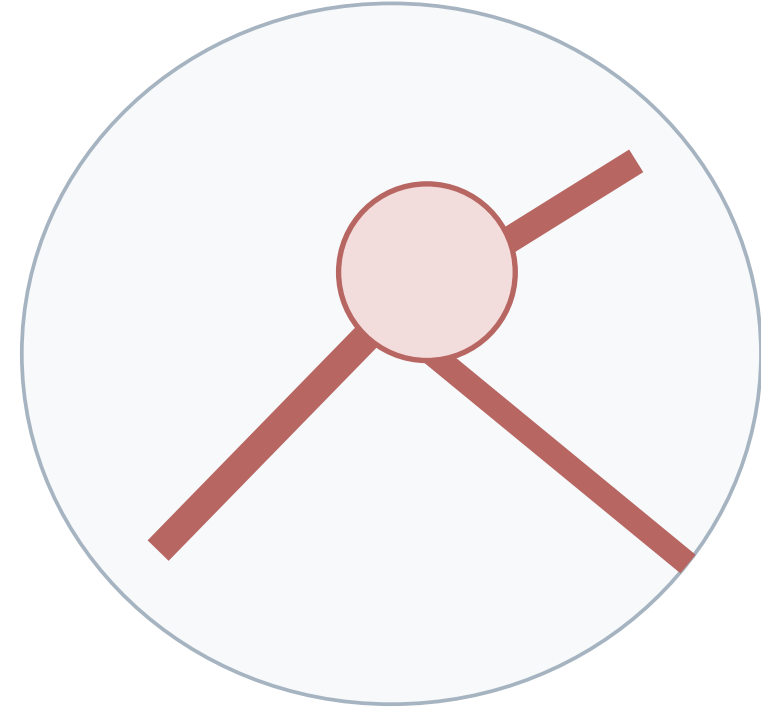


Parent artery: Reinforced wall

Sac: Attenuated fibrous wall

# Saccular aneurysms favor the circle of Willis

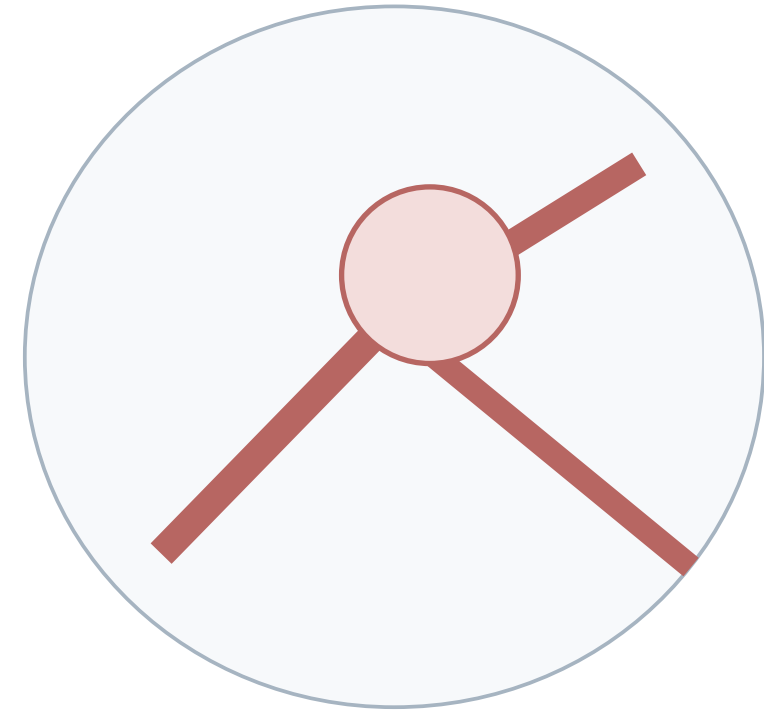
- Approximately 90% of saccular aneurysms occur around branch points in the anterior circulation.
- They lie along the circle of Willis or major proximal cerebral arteries.
- Anatomic localization helps explain the pattern of basal subarachnoid hemorrhage.



Saccular outpouching

## Saccular aneurysms can be multiple

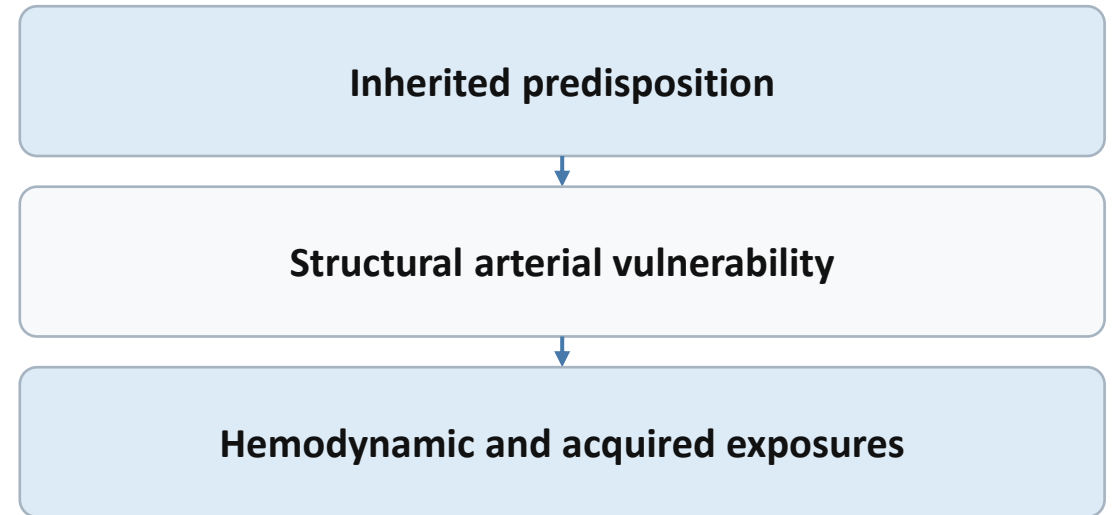
- Multiple aneurysms in roughly 20–30% of cases.
- Finding one aneurysm does not establish that no others exist.
- Multiplicity matters when interpreting neurovascular anatomy and hemorrhage sources.



Saccular outpouching

# Susceptibility is usually multifactorial

- Most saccular aneurysms occur sporadically.
- Risk is increased among first-degree relatives of affected individuals.
- Certain heritable connective-tissue and vascular disorders are associated.

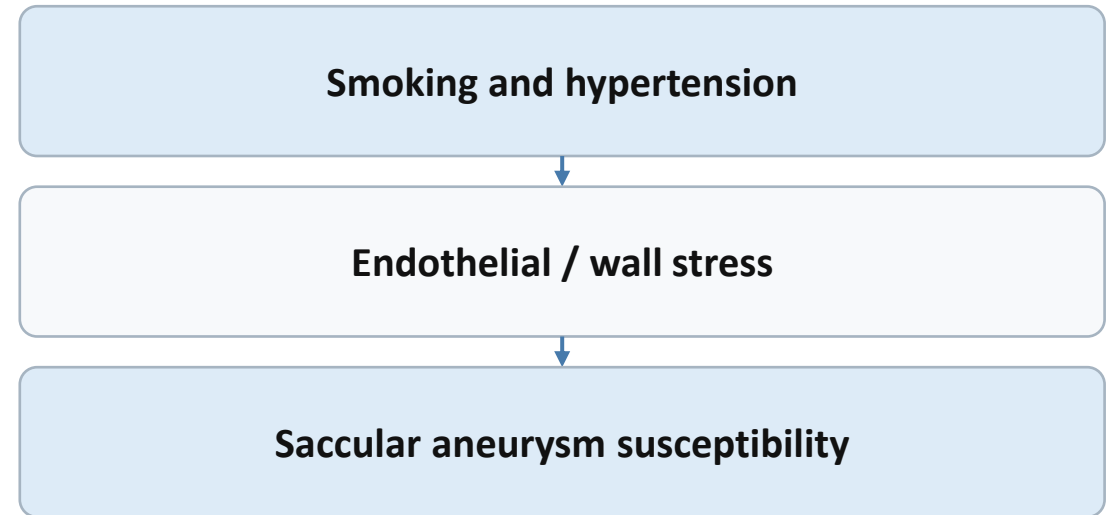


## Examples of associated disorders

- Autosomal dominant polycystic kidney disease is a recognized association.
- Other reported associations include vascular Ehlers–Danlos syndrome, neurofibromatosis type 1, Marfan syndrome and aortic coarctation.
- Fibromuscular dysplasia of extracranial arteries also occurs in association.

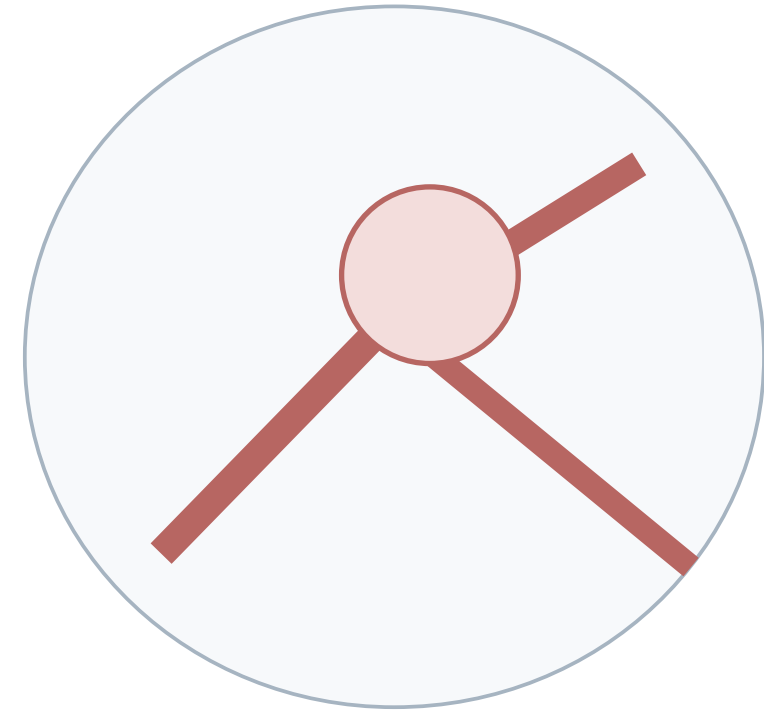
## Smoking and hypertension add acquired stress

- Both smoking and hypertension are described as predisposing factors.
- Hemodynamic stress may induce endothelial dysfunction at branch points.
- An underlying vulnerable wall can enlarge progressively rather than remaining static.



# Unruptured aneurysm: The anatomic lesion

- The lesion is an outpouching rather than a diffuse longitudinal narrowing.
- The sac has a thin fibrotic wall and communicates with the parent artery.
- An intact aneurysm may be detected before any hemorrhage occurs.



Saccular outpouching

# Histologic logic of a saccular aneurysm

- At the sac, normal arterial wall layering is no longer preserved.
- Loss of smooth muscle and elastic support contrasts with the adjacent parent artery.
- A hyalinized fibrous aneurysm wall can be recognized histologically.

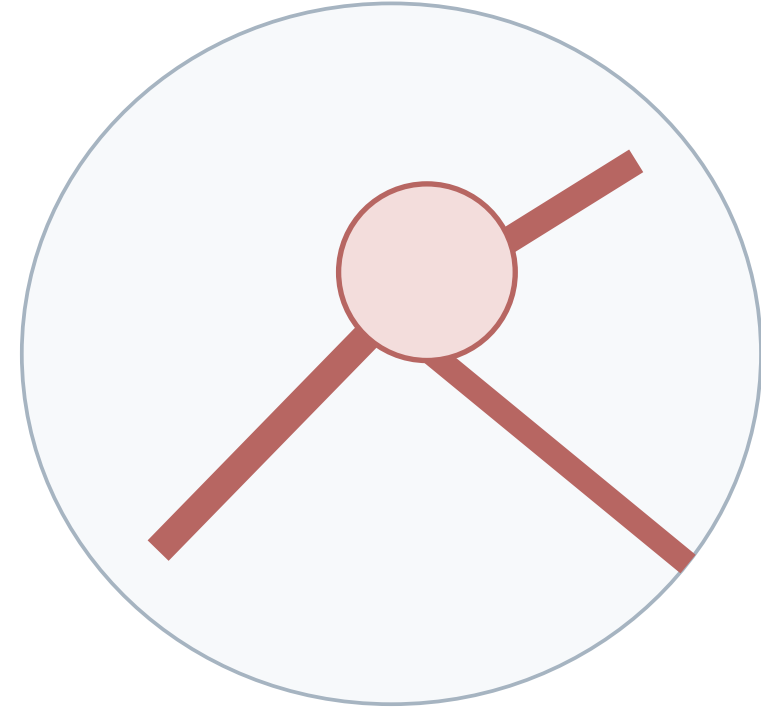


Parent artery: Reinforced wall

Sac: Attenuated fibrous wall

# Rupture leads most characteristically to SAH

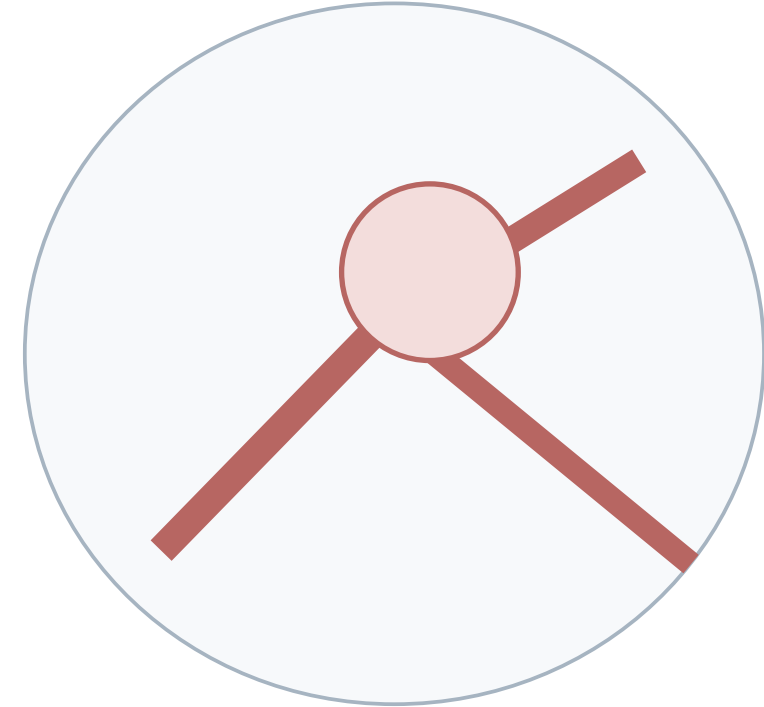
- Rupture of a cerebral saccular aneurysm is the most common cause of spontaneous subarachnoid hemorrhage.
- Blood escapes into the subarachnoid compartment surrounding the brain.
- The event can be immediately life-threatening.



Saccular outpouching

# Subarachnoid and parenchymal bleeding differ

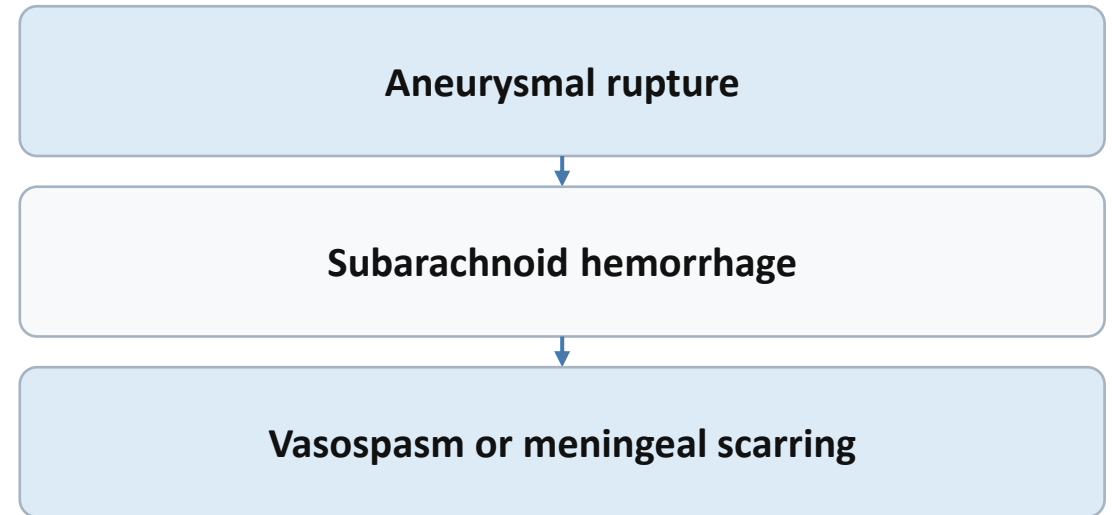
- Subarachnoid hemorrhage disperses blood into CSF-containing spaces.
- Intraparenchymal hemorrhage directly damages adjacent brain tissue.
- An intracranial hemorrhage pattern must be interpreted in its anatomic context.



Saccular outpouching

## Secondary complications extend beyond the rupture

- Vasospasm after subarachnoid hemorrhage can compromise cerebral perfusion.
- Healing can involve meningeal fibrosis and impaired CSF resorption.
- These secondary responses help explain morbidity beyond the initial bleed.



## **Berry aneurysm: Focused application**

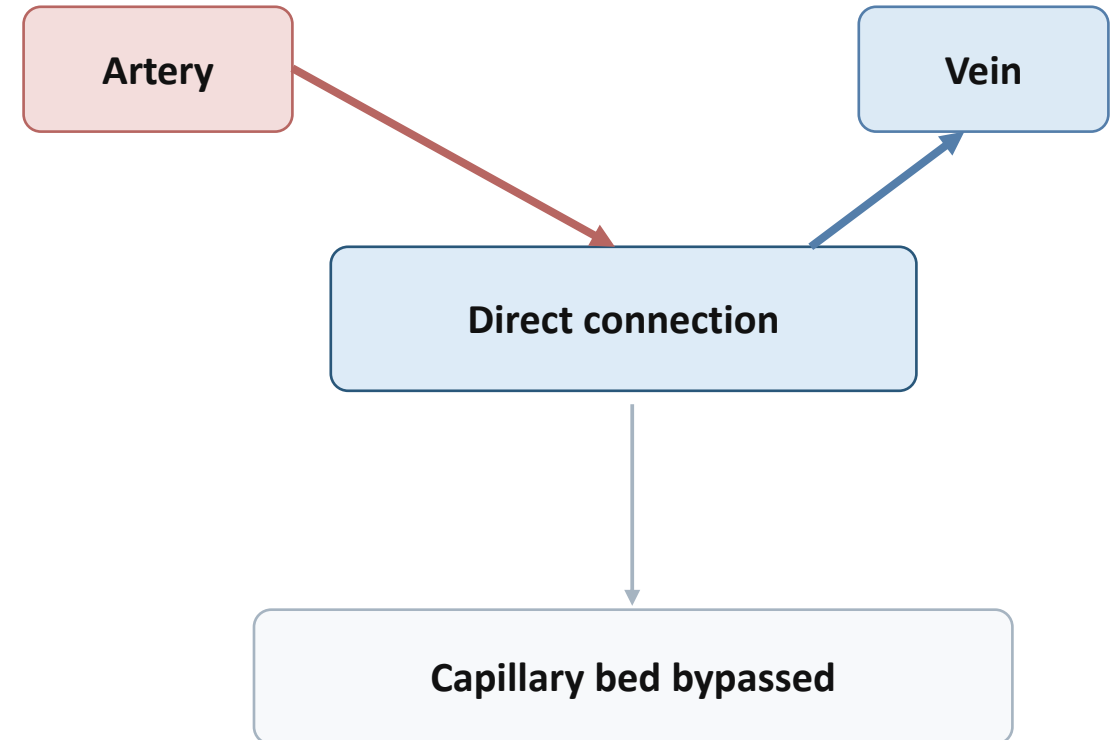
- Illustrative case: Sudden severe headache with CT evidence of basal subarachnoid blood.
- Which vascular defect is a leading spontaneous cause?
- Predict the location and the principal wall abnormality before seeing angiography.

## Case interpretation: Aneurysmal SAH

- A ruptured saccular aneurysm is the leading consideration within the supplied Robbins differential.
- Look near cerebral arterial branch points, particularly in the anterior circulation.
- The characteristic defect involves loss of normal medial smooth muscle and elastic lamina.

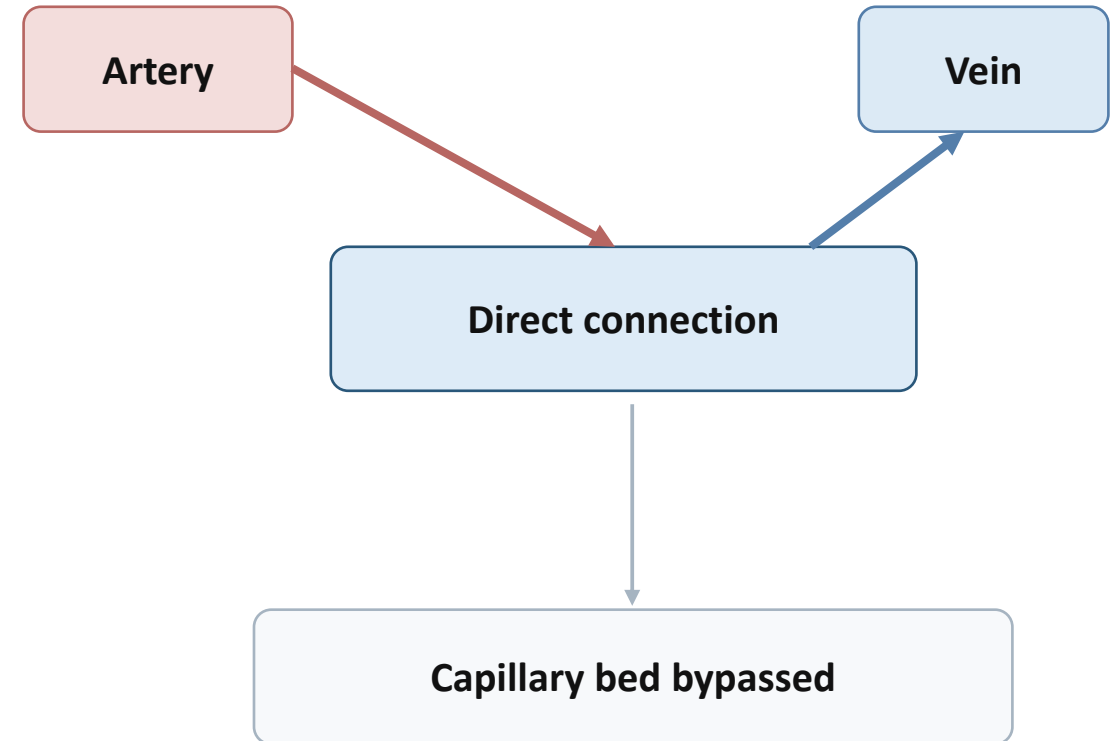
# Arteriovenous fistula: Defining topology

- An arteriovenous fistula directly connects an artery to a vein.
- The communication bypasses intervening capillary exchange channels.
- A fistula is defined by flow routing—not by a tumor-like mass.



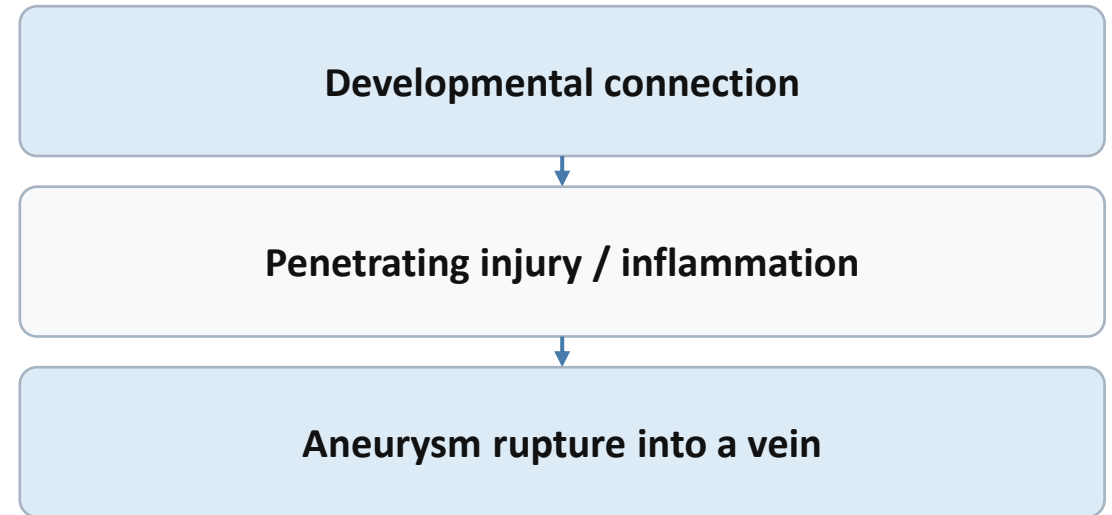
# A shunt changes the normal circulatory pathway

- A normal circuit passes through arterioles, capillaries and venules.
- A fistula diverts some arterial blood directly to the venous system.
- The magnitude of the shunt helps determine systemic consequences.



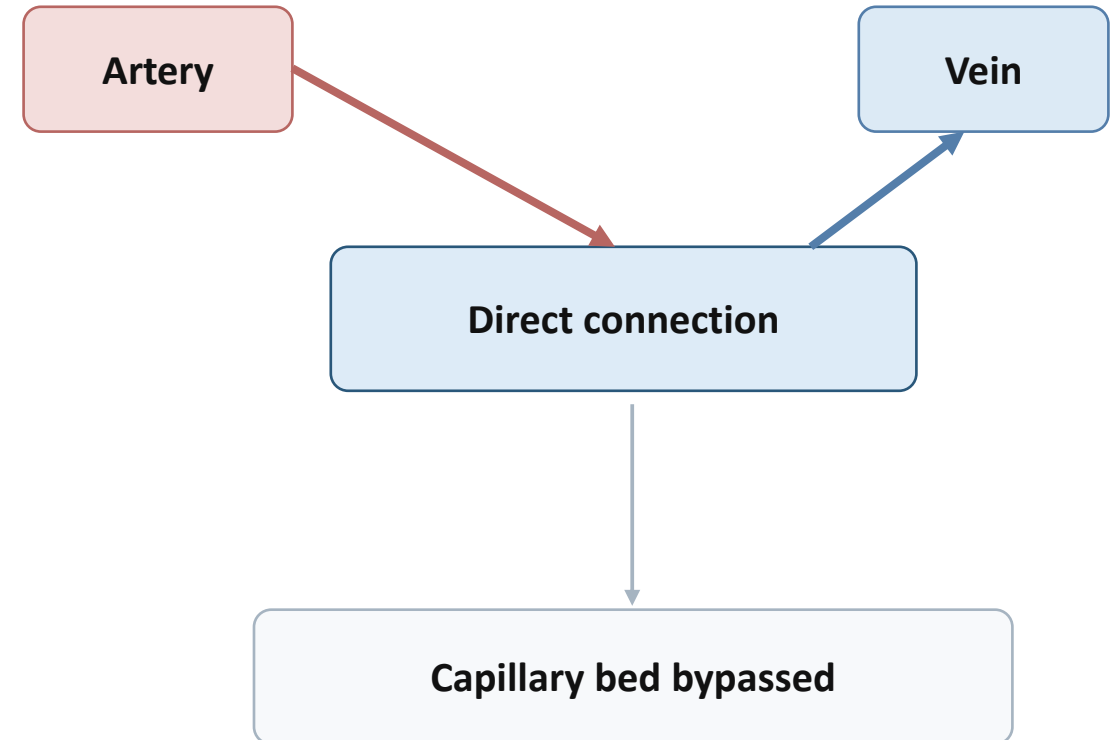
# Fistulas may be congenital or acquired

- Some are developmental defects.
- Others arise when an arterial aneurysm ruptures into a nearby vein.
- Penetrating injury or destructive inflammation can also connect adjacent vessels.



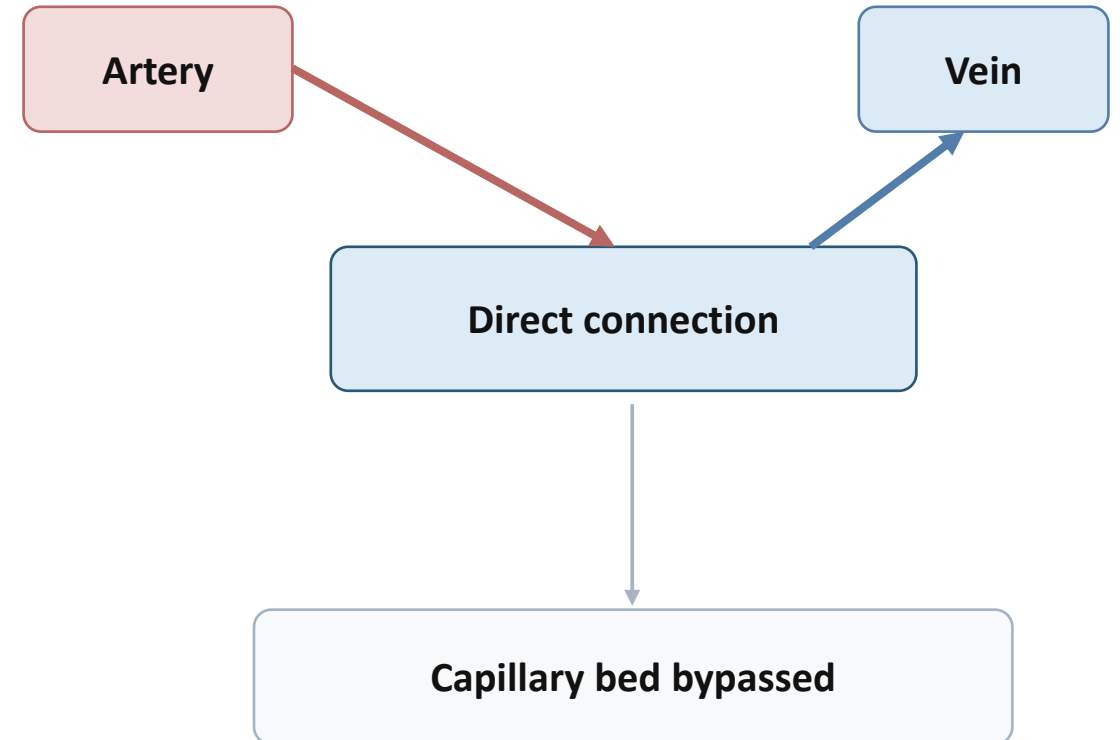
# Penetrating trauma can create a fistula

- A wound crossing an artery and neighboring vein may establish a persistent channel.
- Arterial pressure then drives blood into the lower-pressure venous circulation.
- The mechanism is an acquired connection, not an intrinsic vascular neoplasm.



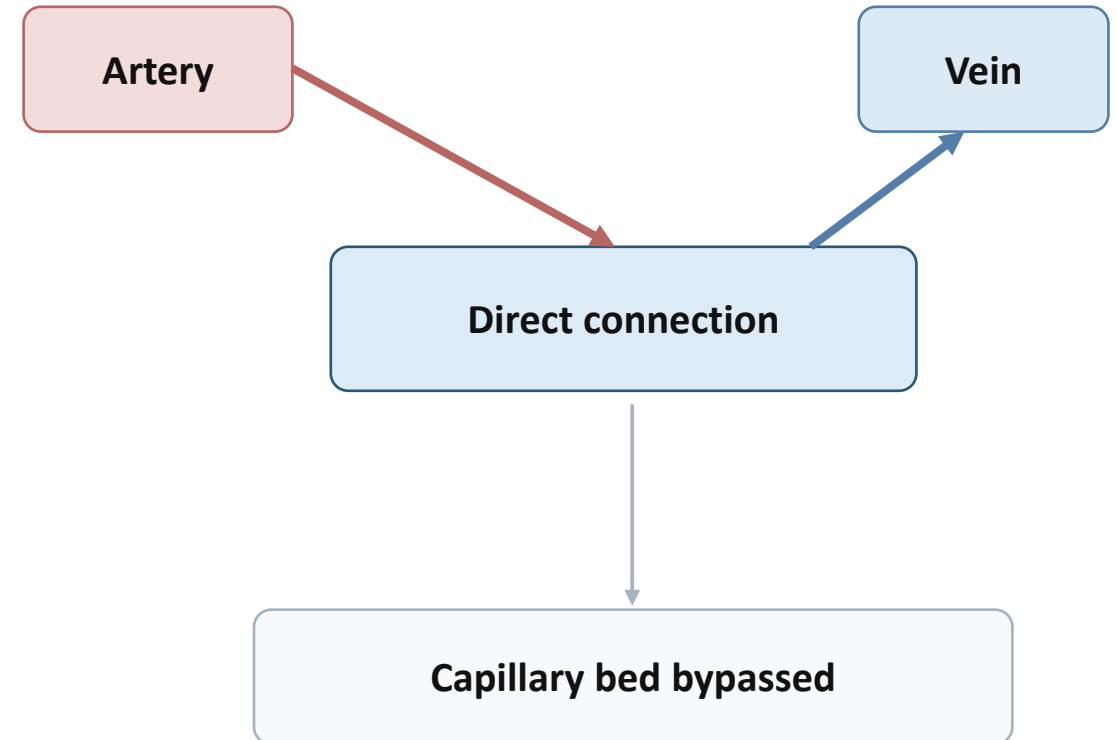
# An aneurysm can rupture into an adjacent vein

- A weakening arterial wall may communicate directly with a nearby venous lumen.
- Such a connection combines a wall defect with pathologic shunting.
- The consequence differs from an aneurysm rupturing freely into tissue.



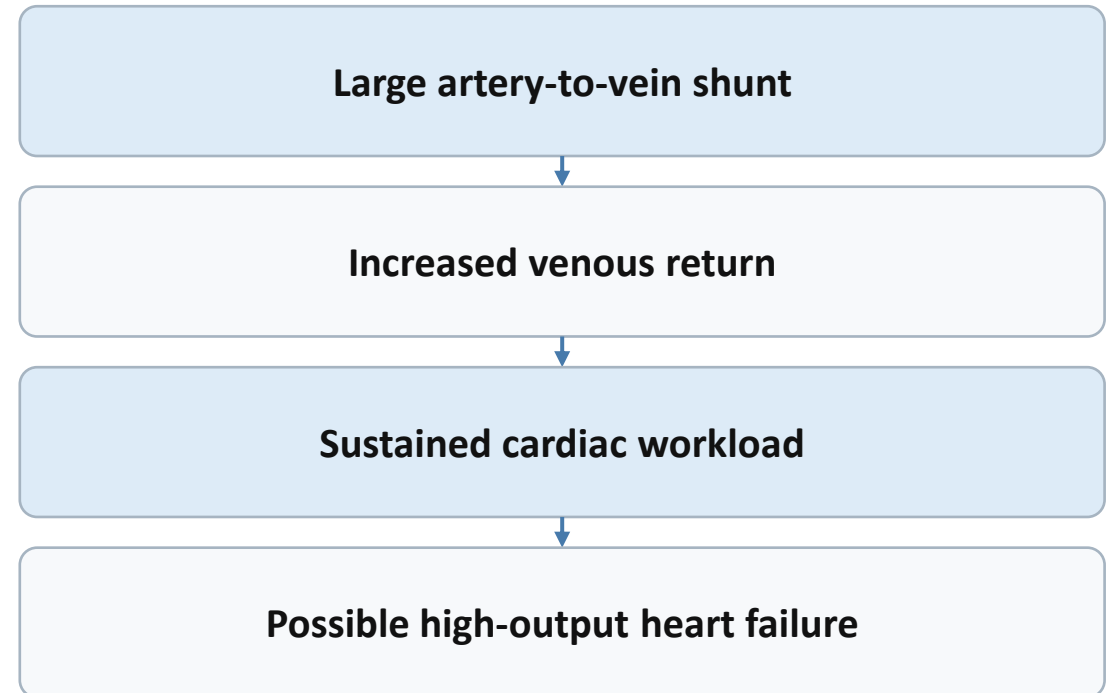
# Inflammation or necrosis can erode adjacent walls

- Destruction of closely apposed arterial and venous tissue may breach both lumens.
- The resulting channel creates a direct path for flow.
- The etiologic route differs from a congenital anatomic connection.



# Large fistulas can overload the heart

- Substantial arterial-to-venous shunting increases venous return.
- Sustained high-flow shunts increase cardiac workload.
- A sufficiently large shunt can produce high-output cardiac failure.



# Rupture is another fistula complication

- Fistulas, like aneurysms, can rupture.
- The hemorrhagic risk is distinct from the hemodynamic burden of a persistent shunt.
- Complications depend on location, vessel integrity and shunt magnitude.

**Saccular aneurysm**

**AV fistula**

**Outpouching**

**Direct shunt**

**Fibromuscular dysplasia**

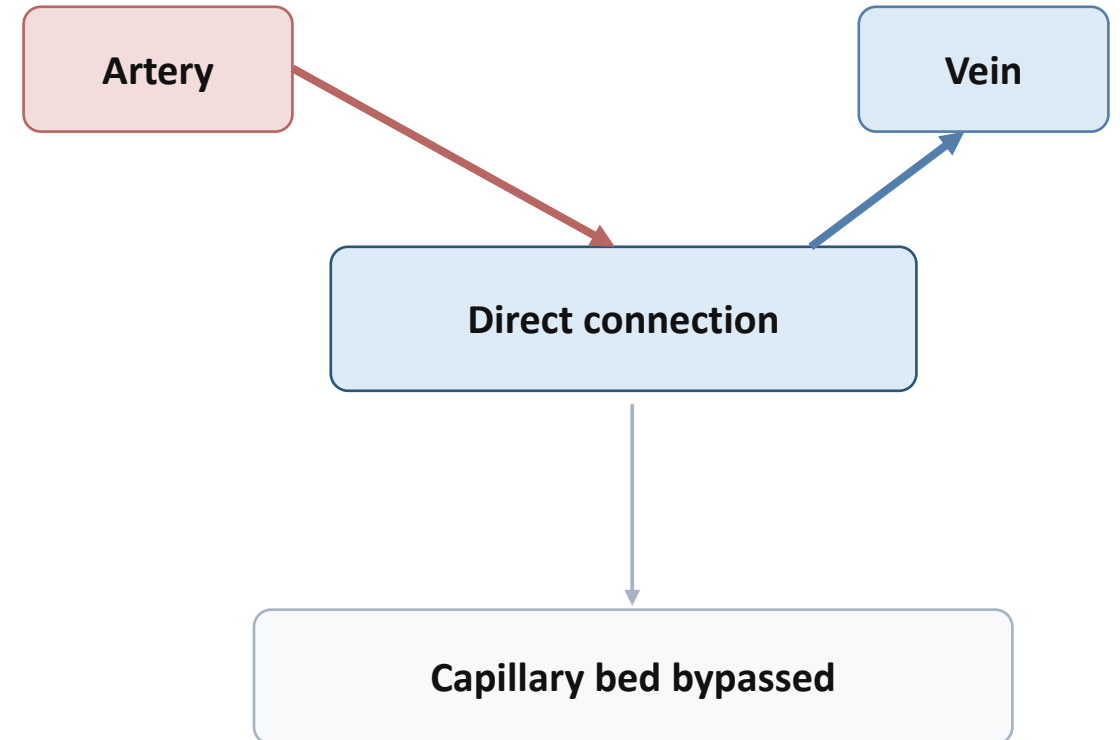
**Coronary anomaly**

**Stenosis / wall thinning**

**Abnormal origin / course**

# Not every fistula is pathologic

- Surgeons deliberately create arteriovenous fistulas for hemodialysis access.
- The therapeutic fistula uses a high-flow artery-to-vein circuit to provide repeated vascular access.
- An identical structural idea can therefore be purposeful or harmful depending on context.



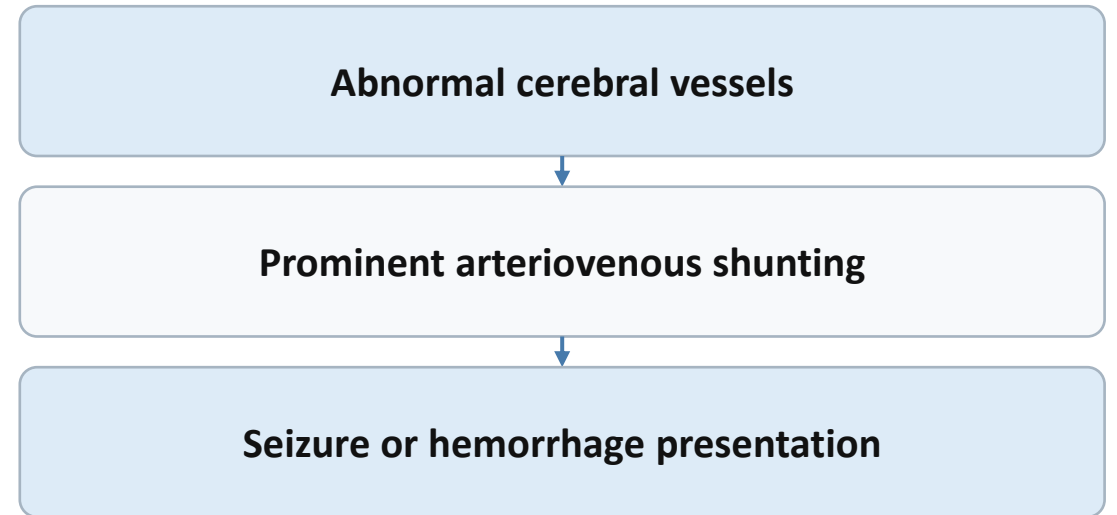
# Arteriovenous fistula versus AV malformation

- A simple fistula denotes a direct connection between artery and vein.
- Cerebral arteriovenous malformations contain abnormal networks of interconnected vessels.
- Both can shunt blood; However, their architecture and clinical evaluation differ.

| Pattern          | Architecture               |
|------------------|----------------------------|
| AV fistula       | Direct artery–vein channel |
| Cerebral AVM     | Tangled, high-flow network |
| Cavernous lesion | Low-flow dilated channels  |

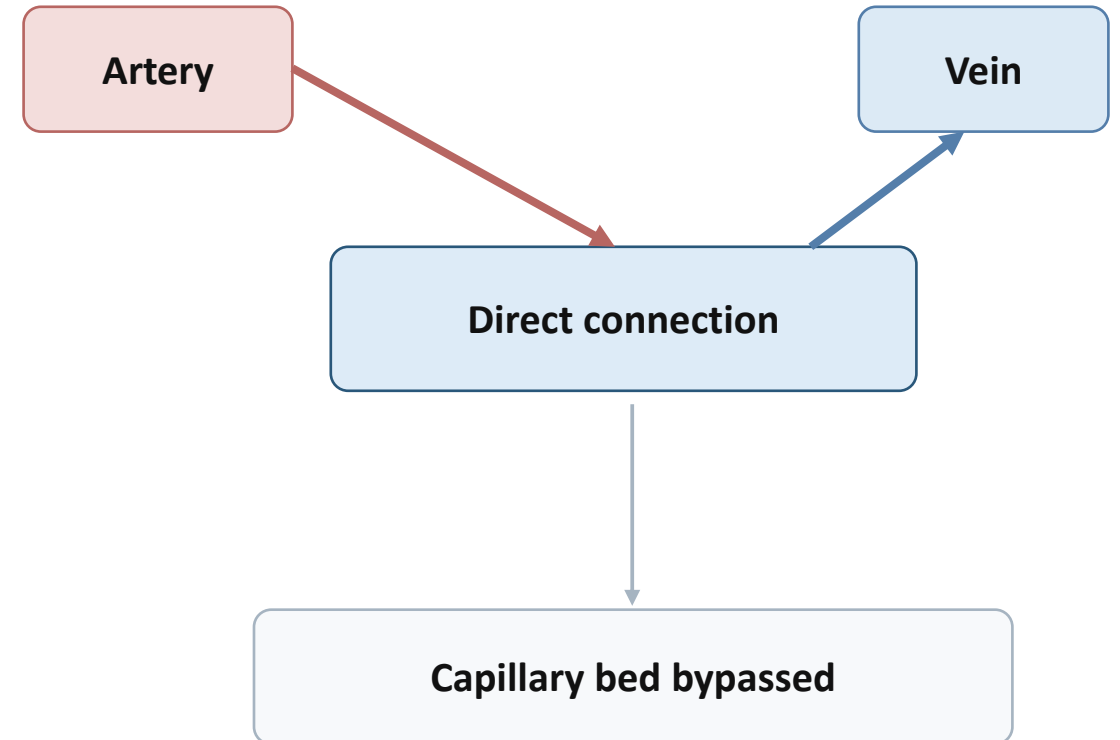
## Cerebral AVM: Relevant contrast

- Cerebral AVMs as tangled, high-flow vascular channels with intervening gliotic tissue.
- They may cause seizures or intracranial hemorrhage.
- This contrasts with a single direct fistulous communication and with low-flow cavernous malformations.



# Large neonatal cerebral shunts: An extreme example

- Major arteriovenous malformations in newborns can produce congestive cardiac failure.
- A vein of Galen malformation may connect malformed arteries directly to veins.
- The example illustrates the impact of high-flow shunting on the circulation.



## Arteriovenous shunt: Focused application

- Illustrative case: An acquired direct artery–vein connection after penetrating injury.
- Blood bypasses a capillary bed; A large shunt progressively increases cardiac output.
- What mechanism connects the local lesion to systemic cardiac dysfunction?

## **Case interpretation: High-output state**

- Persistent shunting diverts arterial blood into veins, increasing venous return.
- The heart compensates by increasing output and may ultimately fail under sustained loading.
- Shunt magnitude is the critical physiologic variable.

# Fibromuscular dysplasia: Core lesion

- Fibromuscular dysplasia (FMD) produces focal, irregular thickening of muscular arteries.
- The lesion involves hyperplasia and fibrosis, frequently in the media or intima.
- Its major consequences are arterial stenosis and local wall weakness.



Alternating stenosis and dilatation

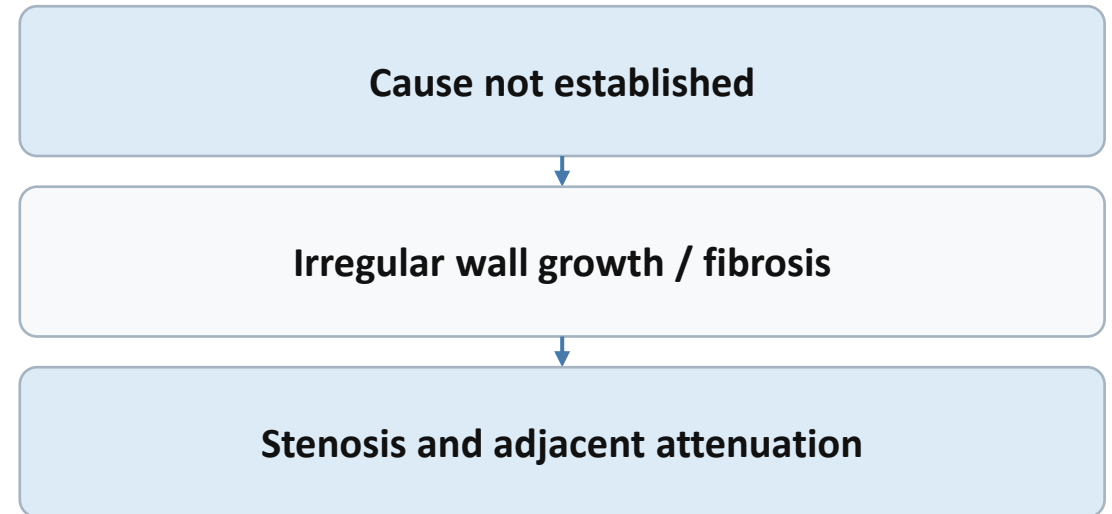
## FMD affects several muscular arterial beds

- Renal, carotid, splanchnic and vertebral arteries.
- The same wall disorder therefore has potential renal, cervical and visceral consequences.
- The affected vessel determines whether ischemia, hypertension or an aneurysm is clinically prominent.

| Vessel              | Potential effect              |
|---------------------|-------------------------------|
| Renal               | Renovascular hypertension     |
| Carotid / vertebral | Cervical arterial involvement |
| Splanchnic          | Visceral arterial involvement |

# The cause of fibromuscular dysplasia is unknown

- Robbins does not establish a single causal genetic or environmental mechanism.
- The morphologic substrate is uneven growth and fibrosis of the vascular wall.
- A named arterial dysplasia should not be presumed inflammatory or atherosclerotic.



## Thickened arterial segments narrow the lumen

- Focal intimal and medial hyperplasia increases wall thickness.
- Fibrosis further reduces effective luminal caliber.
- The resulting stenosis can reduce downstream organ perfusion.



Alternating stenosis and dilatation

## Thin segments can alternate with stenotic areas

- Some arterial segments have markedly attenuated media.
- These portions alternate with thickened, stenosing regions.
- The resulting morphology explains the characteristic segmental angiographic pattern.



Alternating stenosis and dilatation

## “String of beads” is a structural descriptor

- Angiography can show successive narrowings and relative expansions.
- The beaded contour follows alternating focal stenosis and more dilated or attenuated segments.
- It is not synonymous with inflammation or lipid-rich atherosclerotic plaque.



Alternating stenosis and dilatation

## Wall thinning adds risk beyond stenosis

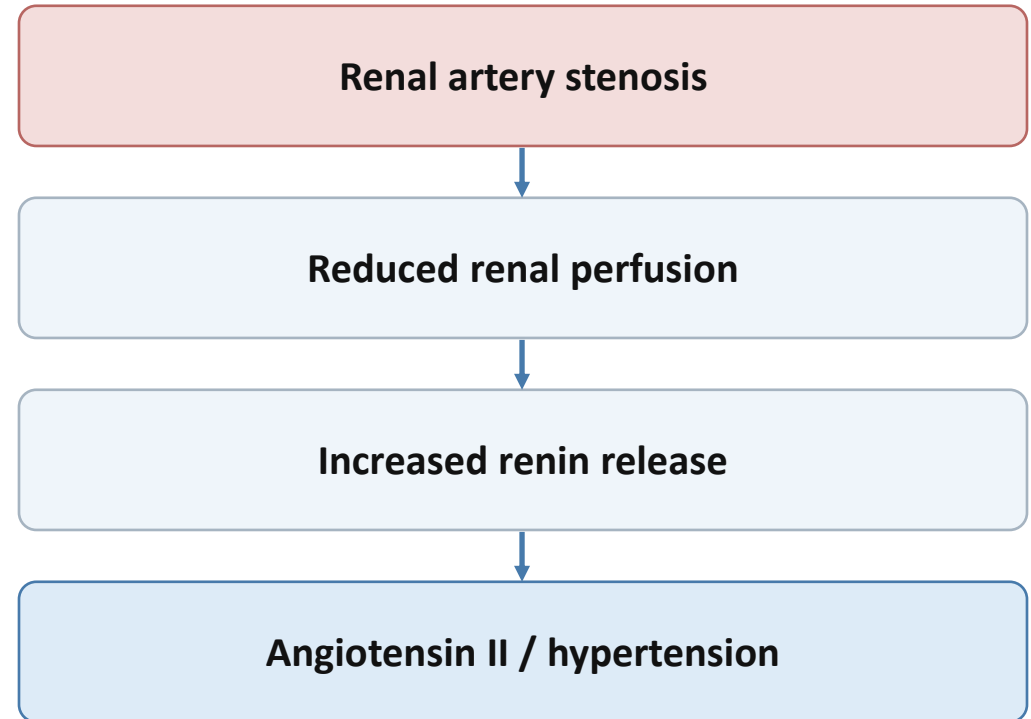
- Attenuated medial areas are less mechanically supported.
- Aneurysms may arise in these weakened segments.
- An aneurysm can rupture; FMD is therefore not solely a narrowing disorder.



Alternating stenosis and dilatation

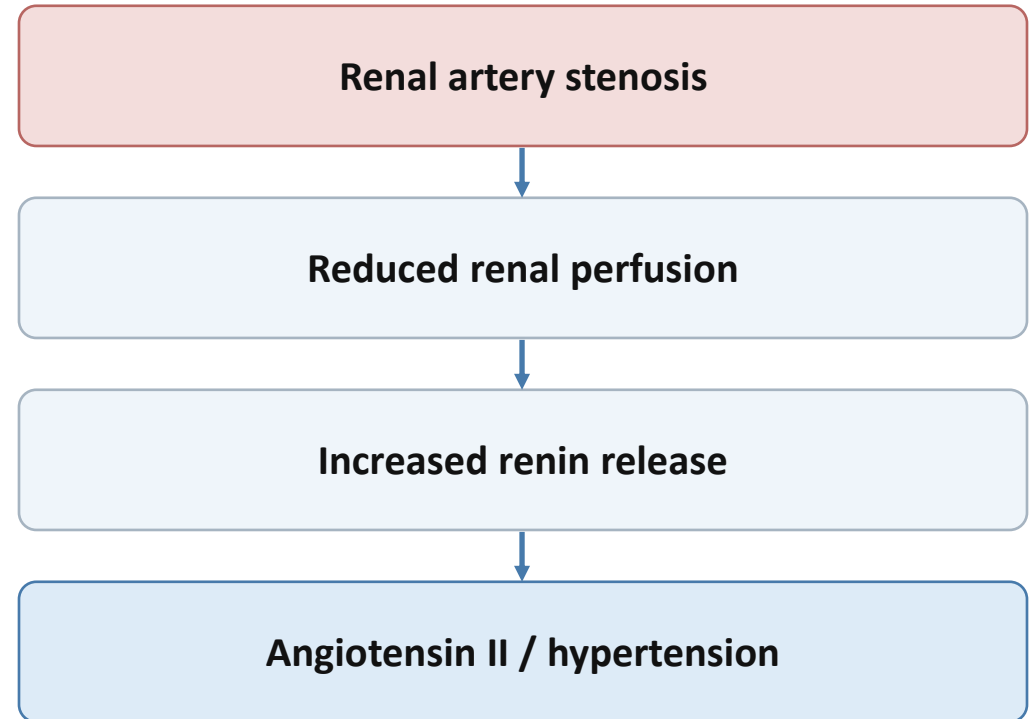
# Renal FMD can initiate renovascular hypertension

- Narrowing of a renal artery reduces the pressure and flow reaching the kidney.
- The ischemic kidney increases renin production.
- Renin–angiotensin signaling promotes systemic hypertension.



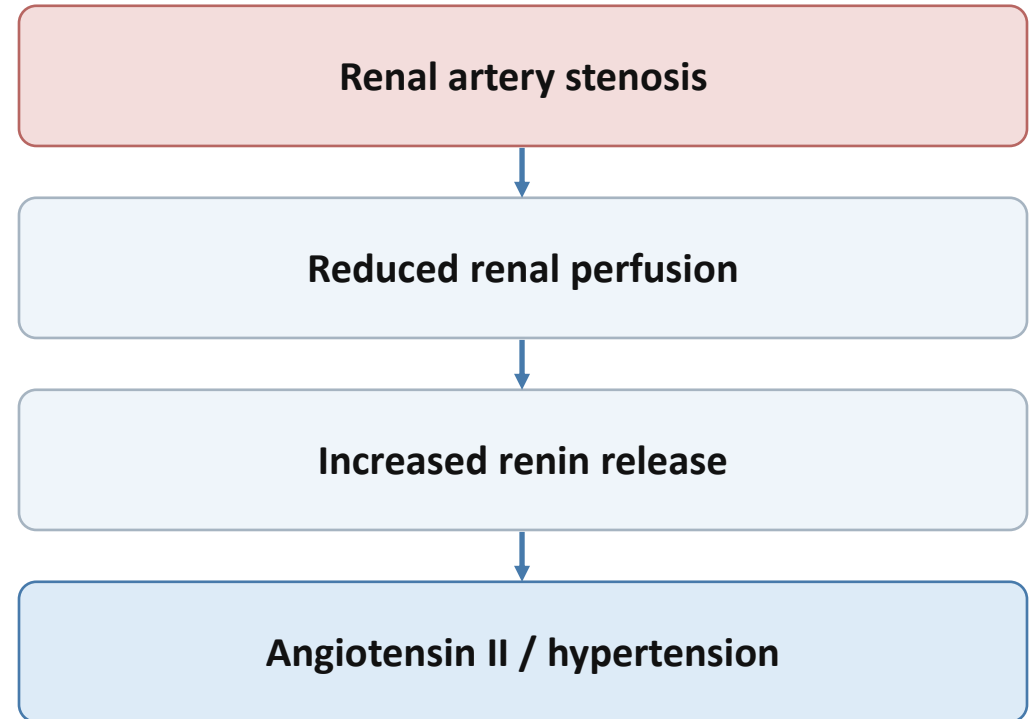
# Renin links renal ischemia to vascular tone

- Juxtaglomerular renin release activates the renin–angiotensin system.
- Angiotensin II contributes to vasoconstriction and elevated systemic blood pressure.
- Sodium retention can contribute to sustained renovascular hypertension.



# Chronic renal artery stenosis changes the kidney

- The ischemic kidney may shrink and develop diffuse atrophy.
- Histology can include crowded glomeruli, tubular atrophy and interstitial fibrosis.
- The contralateral kidney experiences the effects of increased systemic pressure.



# Renal FMD differs from atherosclerotic stenosis

- Atherosclerosis commonly narrows the renal arterial origin.
- FMD is a heterogeneous dysplastic arterial thickening, often in younger patients.
- FMD is more common in females than males.

| Feature       | Atherosclerosis   FMD            |
|---------------|----------------------------------|
| Mechanism     | Intimal plaque   dysplasia       |
| Renal pattern | Ostial plaque   segmental lesion |
| Context       | Often older   often younger      |

# Fibromuscular disease has heterogeneous wall patterns

- Renal FMD may involve intima, media or adventitia.
- Do not infer a single histologic subtype from the diagnosis alone.
- A medial-type lesion can show marked fibrous thickening and luminal narrowing.



Alternating stenosis and dilatation

## Interpreting a renal artery “beading” vignette

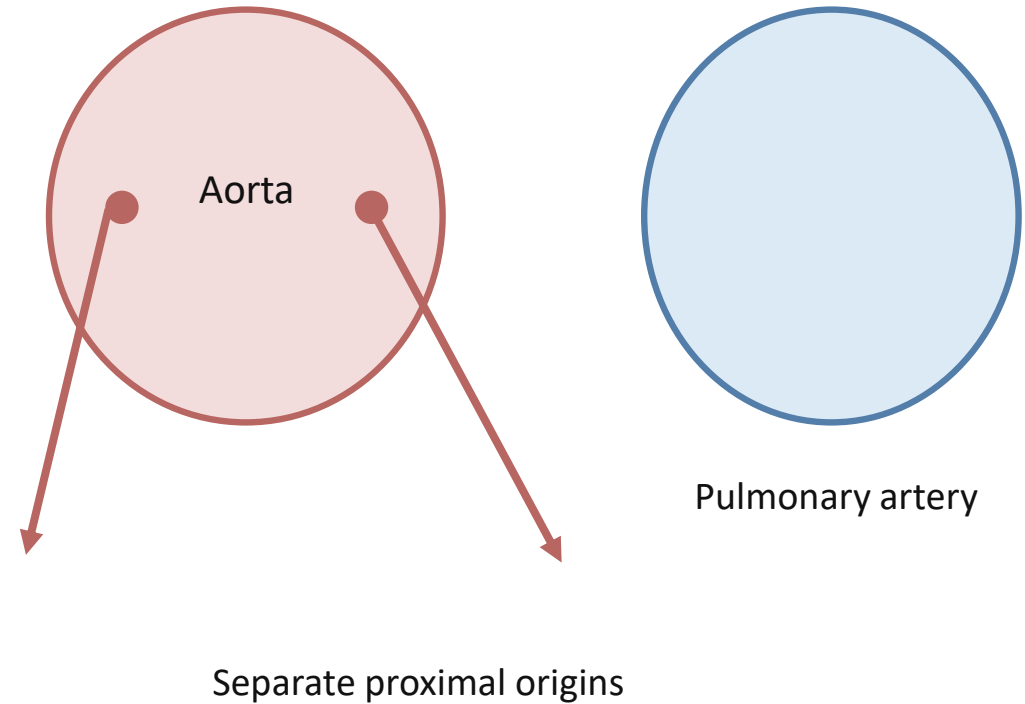
- Illustrative case: A young adult with hypertension and multiple segmental renal arterial narrowings separated by expansions.
- Identify the abnormal arterial wall process.
- Explain the hypertension and a second structural complication beyond stenosis.

## **Case interpretation: FMD of a renal artery**

- An irregular beaded muscular artery supports fibromuscular dysplasia.
- Reduced renal perfusion stimulates renin release and the angiotensin system.
- Medial attenuation can predispose to aneurysm formation and rupture.

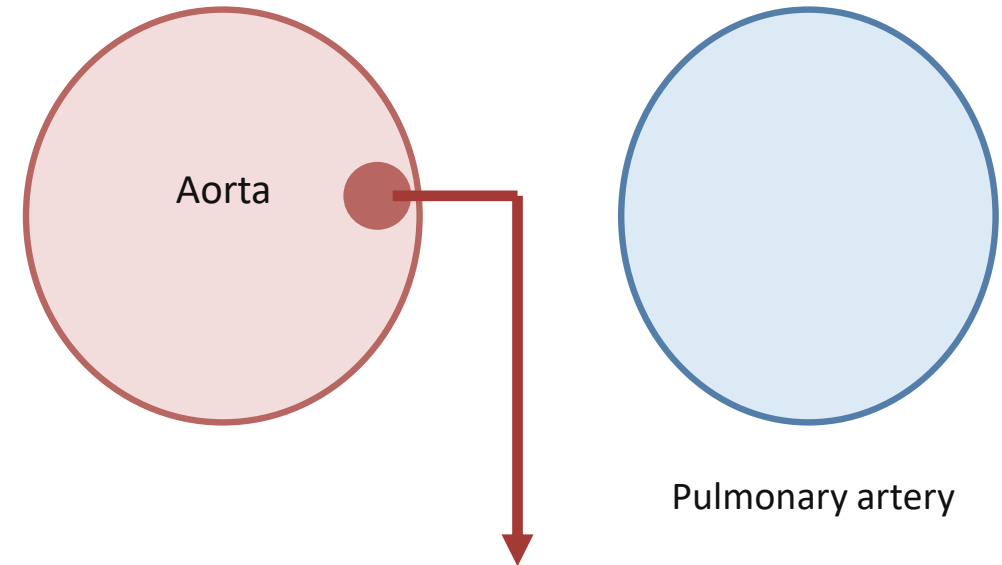
# Normal coronary origin is paired and separated

- Ordinarily, the left and right coronary arteries arise from their respective aortic sinuses.
- Their proximal courses distribute oxygenated blood to the myocardium.
- Origin and trajectory are both essential parts of coronary anatomy.



# Anomalous coronary origin changes the ostium

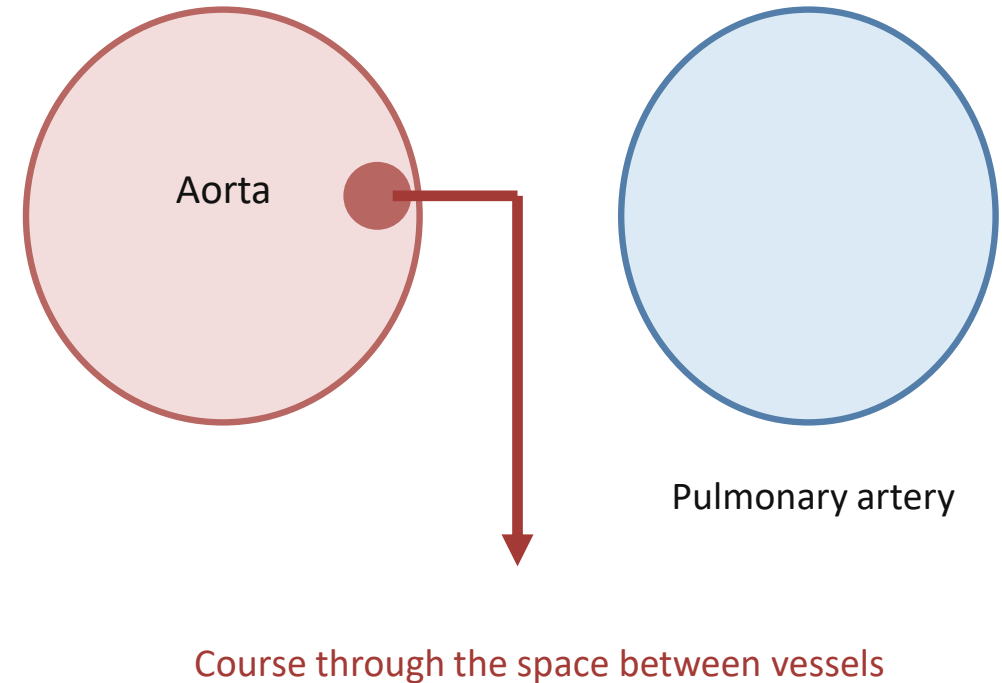
- Both coronary arteries originating above the same aortic valve cusp.
- This is a developmental variant of coronary anatomy.
- An abnormal origin alone does not determine clinical danger; Course is pivotal.



Course through the space between vessels

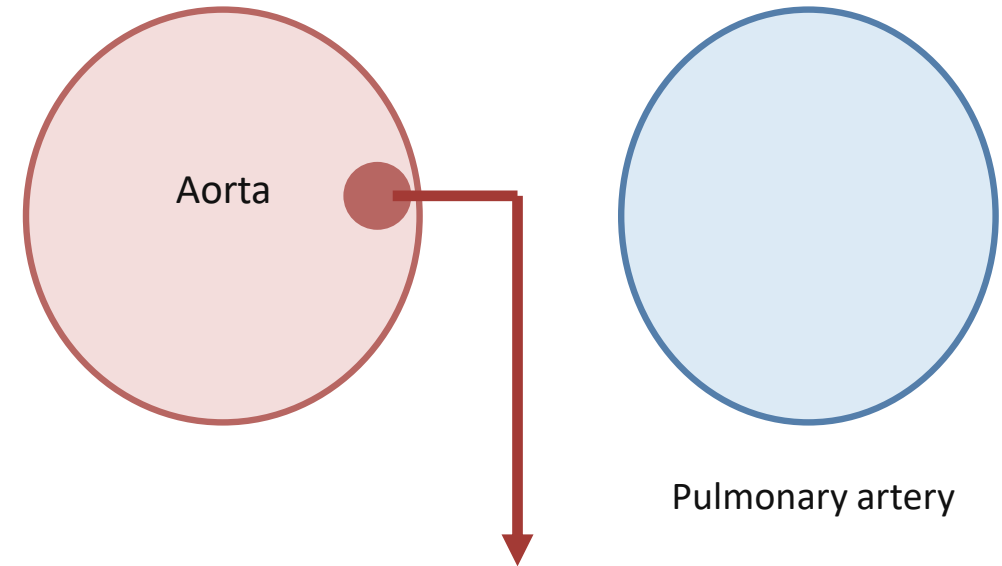
# An interarterial course defines an important hazard

- An anomalous artery may pass between the aorta and pulmonary artery.
- That position exposes the vessel to potential external compression.
- The anatomic relationship becomes especially important during exertion.



# Exercise may compromise an interarterial vessel

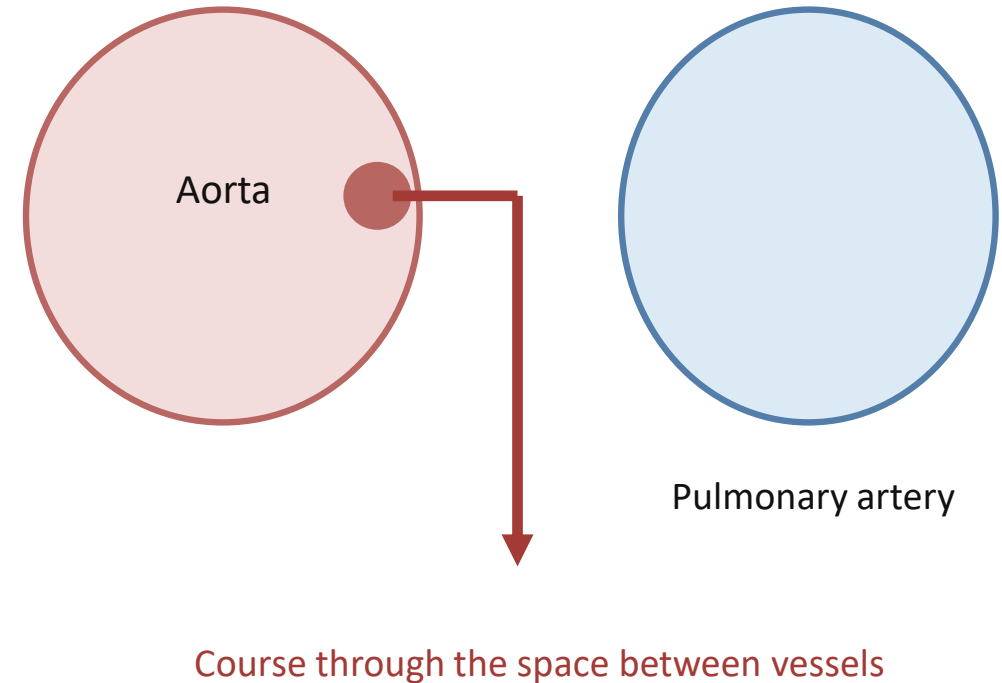
- Expansion and dynamic forces during exertion may compress a vulnerable coronary segment.
- Restricted coronary flow can cause myocardial ischemia.
- In the high-risk configuration, sudden death can occur.



Course through the space between vessels

# Most anomalous origins are not equally dangerous

- Anomalous coronary origin is often benign.
- The concerning scenario is an anomalous vessel passing between the great arteries.
- Risk assessment requires understanding the actual proximal vessel course.



# Compression differs mechanistically from fixed plaque

- Anomalous interarterial coronary artery: Course-dependent mechanical compromise.
- Atherosclerotic coronary disease: Luminal narrowing from an intimal plaque.
- Both can restrict myocardial blood supply, but the initiating pathology differs.

| Mechanism        | What limits flow?             |
|------------------|-------------------------------|
| Coronary anomaly | Course-related compression    |
| Atherosclerosis  | Plaque-related fixed stenosis |

## **Coronary origin: Focused application**

- Illustrative case: Collapse during exertion in a young person; An anomalous coronary artery arises above the same cusp as the other.
- Its proximal segment runs between the ascending aorta and pulmonary trunk.
- Identify the dangerous relationship and explain the potential loss of coronary flow.

## **Case interpretation: Course-dependent ischemia**

- The interarterial coronary segment may be compressed by adjacent great vessels during exertion.
- Impaired myocardial perfusion can provoke life-threatening ischemia.
- The decisive feature is the artery's trajectory, not merely an atypical ostium.

## Four anomalies: Morphology predicts consequence

- Saccular aneurysm: Weakened bulging arterial wall → hemorrhage risk.
- Arteriovenous fistula: Direct arterial–venous communication → high-flow shunt.
- FMD: Alternating thickening and attenuation → stenosis ± aneurysm.
- Anomalous coronary course: Great-vessel proximity → possible dynamic flow restriction.

**Saccular aneurysm**

**AV fistula**

**Outpouching**

**Direct shunt**

**Fibromuscular dysplasia**

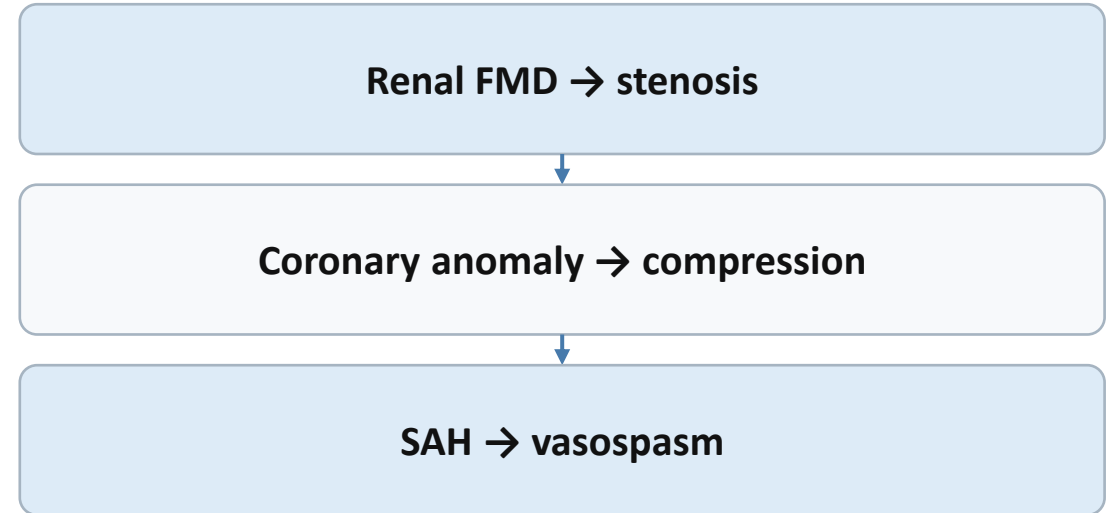
**Coronary anomaly**

**Stenosis / wall thinning**

**Abnormal origin / course**

# Three distinct causes of reduced tissue perfusion

- Arterial narrowing in FMD reduces renal inflow and may stimulate hypertension.
- An interarterial coronary may compromise myocardial inflow mechanically during exertion.
- Post-aneurysmal subarachnoid vasospasm can secondarily reduce cerebral perfusion.



# Hemorrhage risk: Three different anatomical sources

- Berry aneurysm can rupture into subarachnoid spaces.
- Arteriovenous fistulas can rupture from the abnormal connection.
- FMD-associated aneurysms develop in thinned arterial segments and may rupture.

**Saccular cerebral aneurysm**



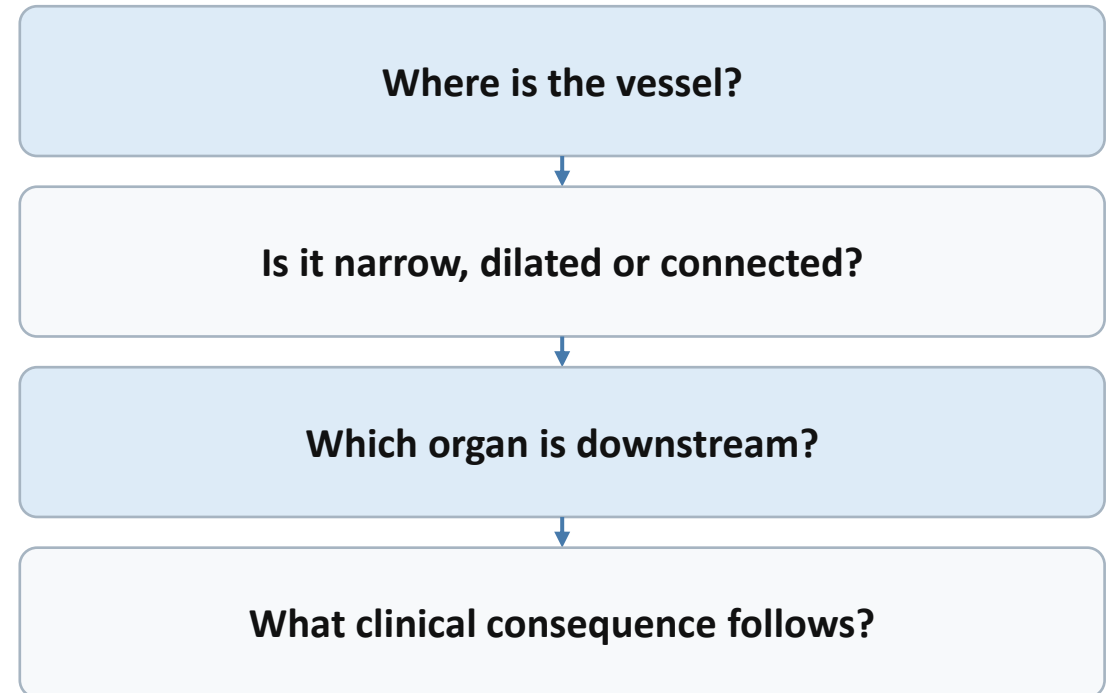
**Arteriovenous fistula rupture**



**FMD-associated aneurysm rupture**

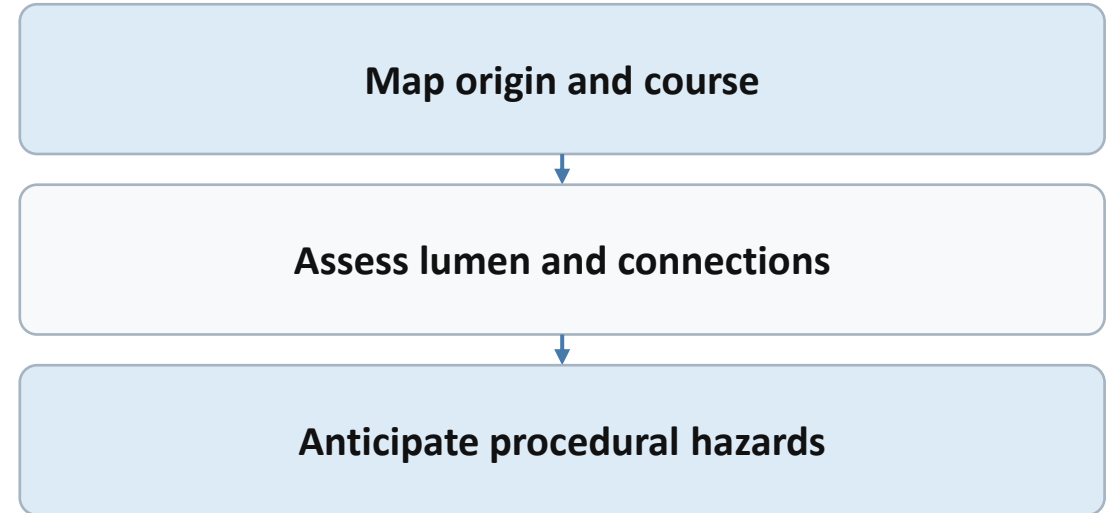
# Recognize the anomaly from the dominant clue

- Branch-point cerebral outpouching points to a saccular aneurysm.
- An artery directly entering a vein points to a fistula.
- Segmental stenoses with beading suggest FMD; An interarterial coronary requires origin-and-course analysis.



# Anomaly recognition in procedures

- Identify unexpected branches and variant ostia before placing devices or operating.
- A catheter or stent cannot be planned solely from an assumed normal vascular map.
- Where a lesion is incidental, the relevant risk still depends on course, wall integrity and flow pattern.



## **Integrated teaching case: Four observations**

- A: Basal subarachnoid blood following sudden headache.
- B: High cardiac output with a direct artery–vein communication.
- C: Renin-associated hypertension with a beaded renal artery.
- D: Exercise-associated ischemia and an interarterial coronary segment.

## **Integrated case: Assign the underlying lesion**

- A: Ruptured saccular aneurysm at a cerebral branch point.
- B: Large arteriovenous fistula causing high-flow shunting.
- C: Renal fibromuscular dysplasia causing stenosis and ischemia.
- D: Anomalous coronary origin with a high-risk interarterial course.

## Essential distinctions for clinical pathology

- Structural dilatation, direct shunting, stenosing dysplasia and anomalous course are not interchangeable diagnoses.
- Morphology should be tied to the mechanism of hemorrhage, ischemia, hypertension or cardiac loading.
- Cross-sectional vascular anatomy guides interpretation even when an anomaly is asymptomatic.

## References and source boundaries

- Primary: Robbins, Cotran & Kumar, Pathologic Basis of Disease, 11th ed. (2026), Chapter 11, pp. 441–444.
- Additional passages from the same text: Chapter 28, pp. 1141–1143, cerebral aneurysms/vascular malformations.
- Additional passages: Chapter 20, pp. 852–853, renovascular hypertension/FMD; Chapter 10, pp. 432–433, pediatric malformations.
- Visuals are original editable schematics; no reproduced pathology images or proprietary textbook figures.