

Aneurysms and Dissection

Vessel-wall pathology • Pathogenesis • Morphology • Clinical correlations

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Learning objectives

- Distinguish true aneurysm, pseudoaneurysm, and dissection by the location of blood relative to the wall.
- Explain extracellular matrix, smooth muscle, and vasa vasorum contributions to wall weakness.
- Compare abdominal and thoracic aortic aneurysms and recognize dissection-related complications.
- Link gross and microscopic morphology to clinical manifestations.

The spectrum of major aortic lesions

Wall dilation

True aneurysm

Wall breach

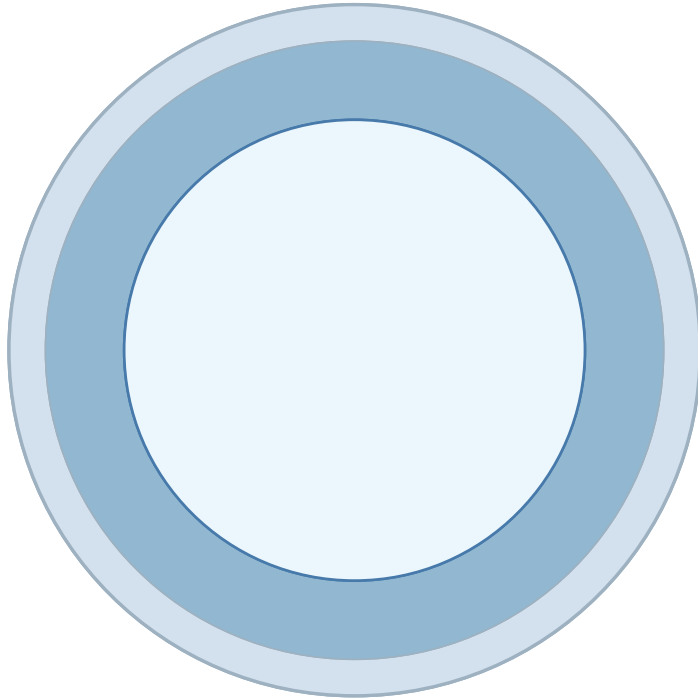
Pseudoaneurysm

Intramural channel

Dissection

These are distinct structural phenomena rather than interchangeable terms.

Normal aortic wall: Structure and resilience



Intima

Inner lining and subendothelial matrix

Media

Smooth muscle and elastic lamellar units

Adventitia

Outer connective tissue and vasa vasorum

Three layers have different mechanical roles

- Intima provides the endothelial interface and can develop atherosclerotic thickening.
- Elastic lamellar units and smooth muscle cells within the media accommodate pulsatile flow.
- Adventitia supports the vessel and contains vessels supplying outer wall regions.
- Wall integrity requires ongoing synthesis, remodeling, and repair of extracellular matrix.

Extracellular matrix homeostasis

ECM synthesis

Smooth muscle production

ECM breakdown

Proteolytic activity

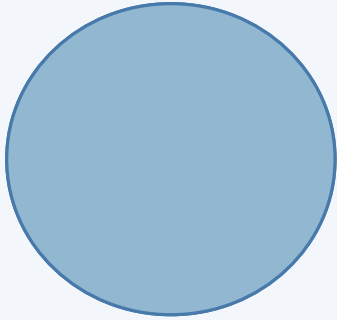
Balanced remodeling

Preserved wall strength

Aneurysm: The defining lesion

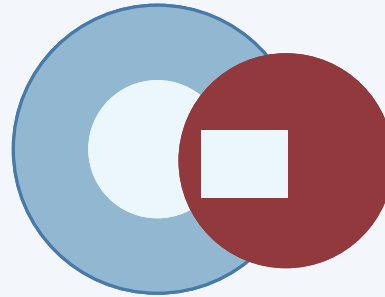
- A localized abnormal dilation of a blood vessel or cardiac chamber.
- May be congenital or acquired.
- True aneurysm contains the native wall, although it may be markedly attenuated.
- Potential consequences include rupture, mural thrombosis, and embolization.

Three structural categories are not synonyms



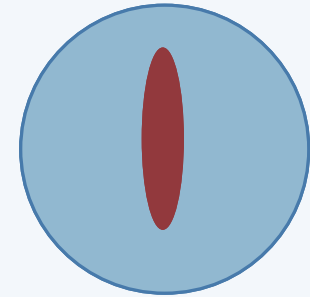
True aneurysm

Dilation with attenuated but
continuous native wall



Pseudoaneurysm

Wall defect; Blood contained by
extravascular tissues



Dissection

Blood splits laminar planes
within arterial wall

True aneurysm: Wall continuity persists

- Dilation incorporates all layers of an intact but attenuated arterial wall.
- Examples include atherosclerotic and congenital arterial aneurysms.
- Postinfarction ventricular aneurysms also meet the definition of a true aneurysm.
- An intact wall does not imply a low rupture risk.

Pseudoaneurysm: Blood outside the wall

- A focal arterial or cardiac wall defect creates communication with an extravascular hematoma.
- Containment depends on adjacent tissue rather than all native wall layers.
- Examples include a contained ventricular rupture after infarction or a vascular graft anastomotic leak.
- The cavity can be pulsatile because it communicates with the lumen.

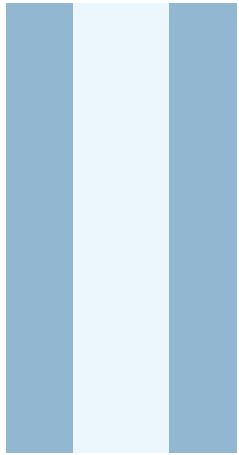
Dissection: Blood tracks through the wall

- Blood enters the wall and separates lamellar planes, typically in the media.
- The resulting intramural channel may extend longitudinally in either direction.
- Dissection may occur with or without conspicuous preexisting dilation.
- Rupture outward or obstruction of branch vessels produces severe complications.

Compare aneurysm, pseudoaneurysm, dissection

True aneurysm	Pseudoaneurysm	Dissection
• Dilation is the primary change • Native wall continuous but attenuated • May develop mural thrombus	• Full-thickness wall breach • Hematoma bounded by outside tissues • Communication with the lumen	• Blood within wall lamellar planes • True and false lumens may form • Possible branch-vessel compromise

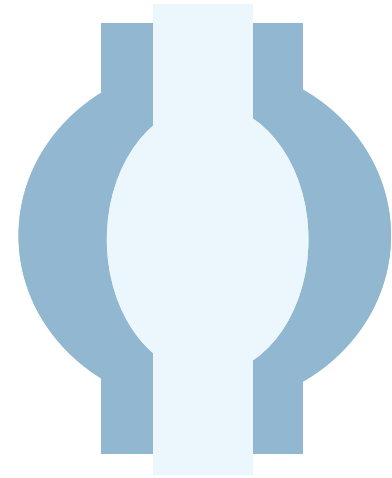
Saccular and fusiform geometry



Healthy vessel



Saccular aneurysm



Fusiform aneurysm

Saccular aneurysm: Focal wall outpouching

- Essentially spherical outpouching involving only a portion of circumference.
- Can occur in intracranial arteries and the aorta.
- May contain thrombus, particularly in large aortic lesions.
- Shape alone does not specify the underlying cause.

Fusiform aneurysm: Circumferential dilation

- Diffuse dilation of a longer vascular segment and much of its circumference.
- May involve aortic arch, abdominal aorta, and iliac arterial territories.
- Can coexist with extensive atherosclerotic disease.
- The term describes morphology, not a distinct etiologic diagnosis.

Shape does not establish pathogenesis

What shape tells you

- Distribution of outward dilation
- Length and circumference involved
- Potential anatomic extent

What shape does not tell you

- Specific genetic or acquired cause
- Whether the lesion is infected
- Its clinical course without other context

Major complications of aneurysms

Wall weakening

Progressive dilation

Luminal changes

Thrombus / emboli

Structural failure

Rupture / hemorrhage

Local effects

Compression /
obstruction

Rupture: Conversion to hemorrhage

- Failure of the thinned aortic wall permits blood to escape into surrounding spaces.
- Intraperitoneal, retroperitoneal, thoracic, or pericardial bleeding may occur depending on site.
- Rapid blood loss makes rupture a major cause of sudden death.
- Anatomical site determines the immediate clinical syndrome.

Mural thrombosis and embolization

Aneurysmal dilation

Altered local flow



Mural thrombus

Surface thrombosis



Distal emboli

Downstream ischemia

Compression and branch-vessel effects

- Aneurysm mass effect may impinge on neighboring structures.
- Extension across branch ostia or superimposed thrombus may compromise perfusion.
- Renal, mesenteric, iliac, and other branches may be affected.
- Clinical findings depend on the vascular territory involved.

Wall weakness is a final common pathway

Matrix degradation

Loss of elastic support

Abnormal signaling

Disordered remodeling

Smooth-muscle loss

Inadequate repair

Separate etiologies converge on reduction of mechanical integrity.

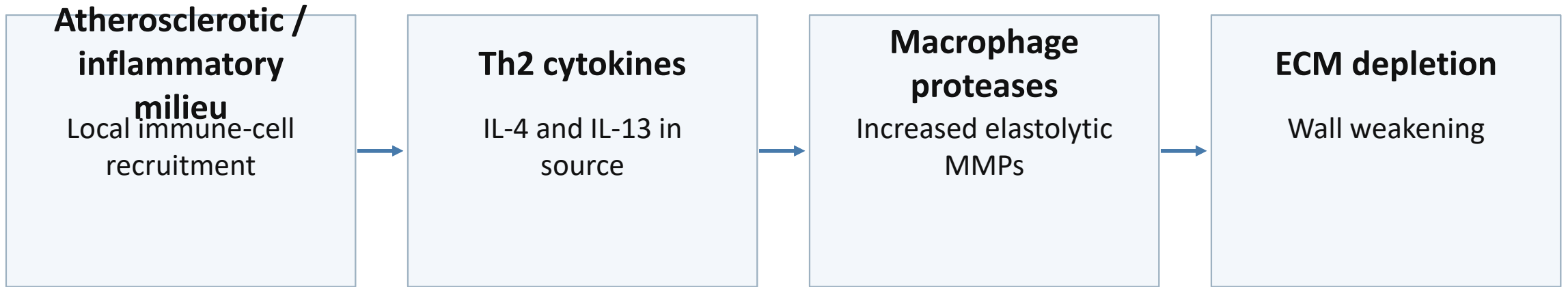
Matrix metalloproteinases in aneurysm

- Inflammation in atherosclerosis and aortitis can increase matrix metalloproteinase (MMP) activity.
- Macrophage-derived MMPs degrade elastin, collagen, and other matrix components.
- Loss of elastic fibers impairs recoil during diastole.
- The net effect is weakened media and progressive wall dilation.

Tissue inhibitors regulate matrix breakdown

- Tissue inhibitors of metalloproteinases (TIMPs) normally restrain MMP activity.
- Reduced inhibition favors unbalanced extracellular matrix proteolysis.
- The balance between proteases and inhibitors matters more than either alone.
- ECM loss can coexist with unsuccessful compensatory matrix synthesis.

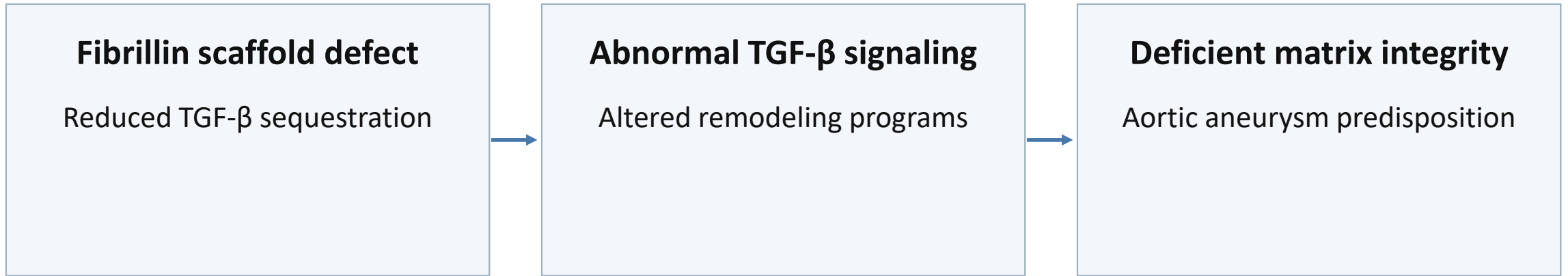
Inflammatory cytokines and the AAA wall



Type III collagen and vascular Ehlers–Danlos

- Vascular Ehlers–Danlos syndrome features defective type III collagen synthesis.
- Poor connective tissue quality reduces intrinsic arterial wall strength.
- Structural fragility differs mechanistically from acquired matrix degradation.
- A genetic cause should be considered in a syndromic or unusually young patient.

Fibrillin and dysregulated TGF- β signaling



Simplified source-derived pathway, not a complete receptor signaling network.

Marfan syndrome: Matrix and signaling

- Fibrillin normally participates in extracellular sequestration of transforming growth factor β (TGF- β).
- Fibrillin defects alter the local bioavailability of TGF- β .
- Disordered wall remodeling particularly affects the ascending aorta.
- Clinical skeletal features may provide a clue to inherited aortopathy.

Loeys–Dietz syndrome: Pathway dysfunction

- Variants involving TGF- β receptors and pathway components can affect aortic wall integrity.
- The Robbins section names TGF- β receptors, SMAD3, and TGF- β 3 as relevant components.
- Aneurysms may rupture at comparatively small diameters.
- Morphology alone cannot confirm the underlying molecular diagnosis.

Inherited aortopathy: Mechanistic contrast

Vascular Ehlers–Danlos

- Type III collagen defect
- Intrinsic matrix fragility
- Weak arterial wall

Marfan syndrome

- Fibrillin scaffold defect
- Altered TGF- β sequestration
- Ascending aortic involvement

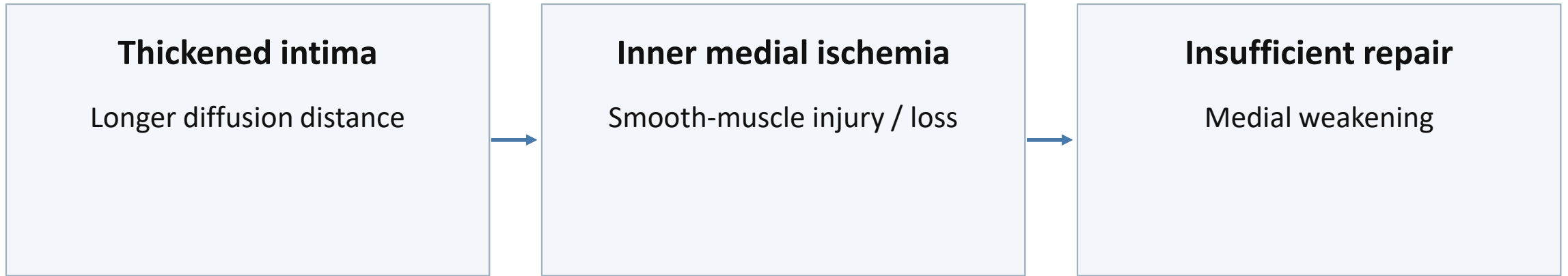
Loeys–Dietz syndrome

- TGF- β signaling pathway variants
- Disordered wall remodeling
- Rupture possible at small sizes

Smooth-muscle cells maintain aortic media

- Medial smooth-muscle cells synthesize and remodel elastic and collagenous matrix.
- Smooth-muscle loss diminishes capacity for structural repair.
- Ischemia and inflammatory injury can accelerate cell attrition.
- Medial degeneration combines cellular loss with matrix abnormalities.

Atherosclerotic thickening compromises nutrition



Hypertension and the vasa vasorum

Systemic hypertension

Arteriolar narrowing



Vasa vasorum compromise

Outer media ischemia



Smooth-muscle loss

Reduced matrix integrity

Infectious aneurysm: Three entry routes

Septic embolus

- May follow infective endocarditis
- Organisms lodge in vessel wall

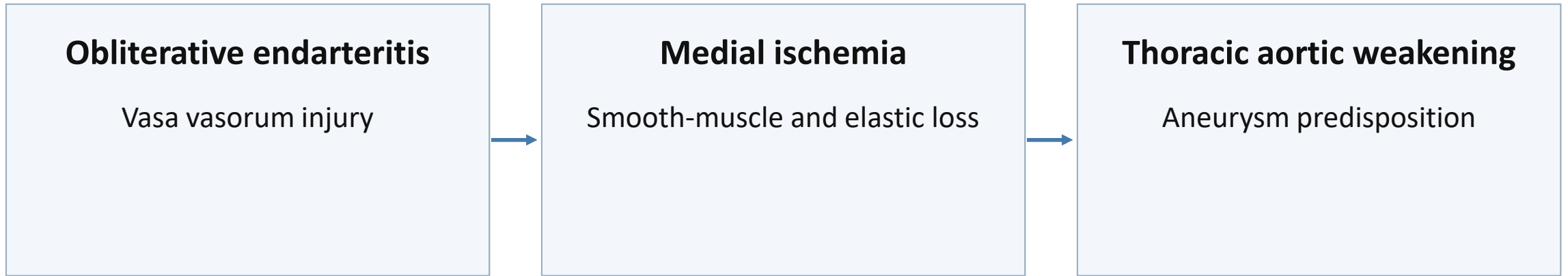
Local extension

- Adjacent suppurative focus
- Inflammation spreads to artery

Hematogenous infection

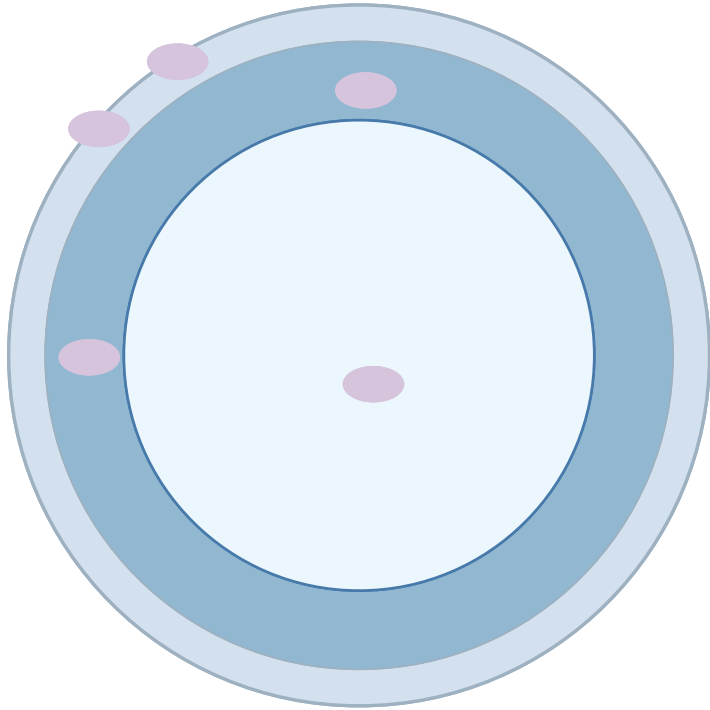
- Circulating organisms invade wall
- Suppuration damages media

Syphilitic aortitis: Vascular nutrition failure



Rare in contemporary practice, included for textbook pathology.

Medial degeneration: The histologic pattern



Elastic-fiber fragmentation

Reduced recoil / lamellar integrity

Mucoïd ECM accumulation

Expanded proteoglycan-rich zones

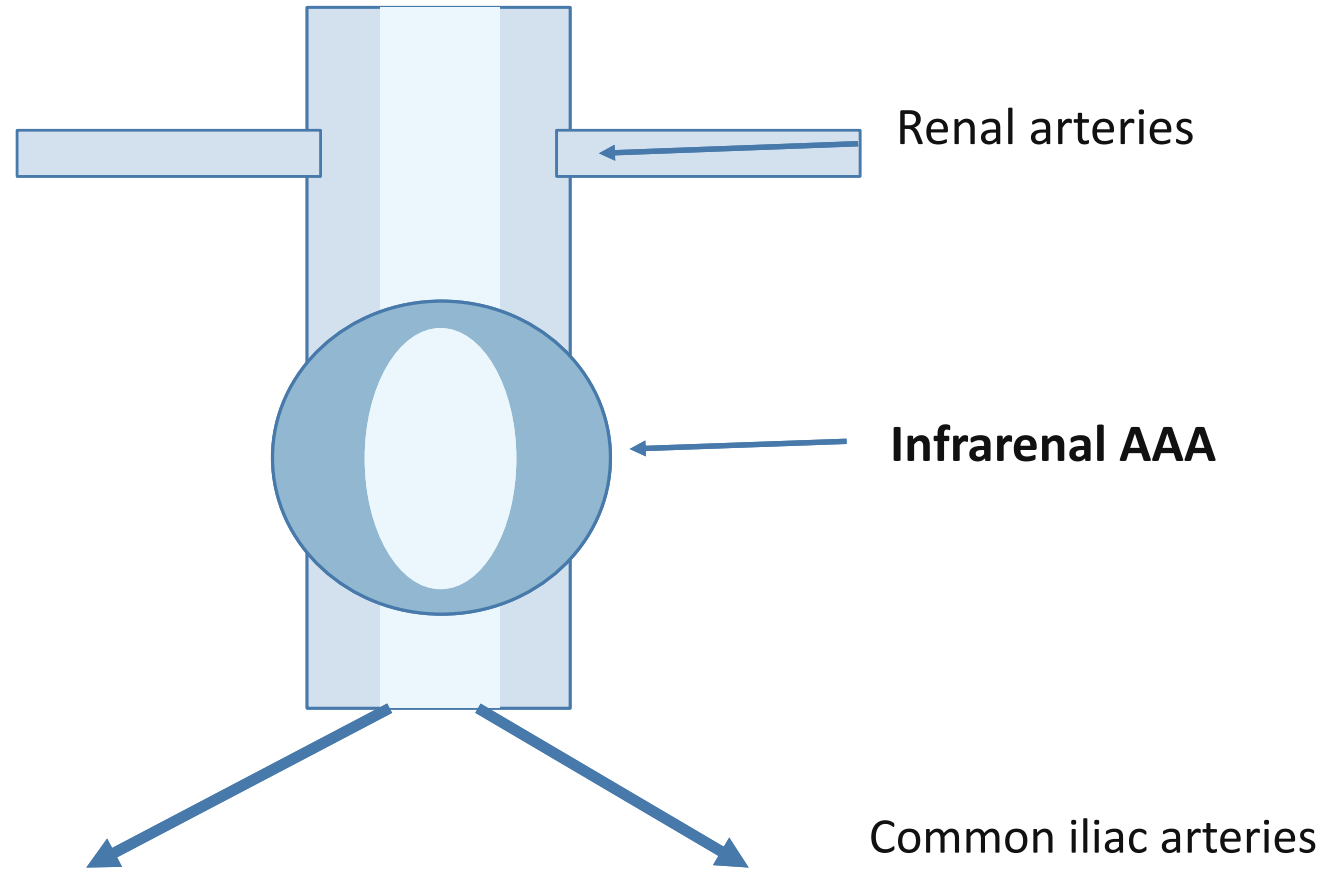
Smooth-muscle attrition

Reduced matrix maintenance and repair

Medial degeneration is not etiologically specific

- Fragmentation and loss of elastic fibers disturb lamellar architecture.
- Muroid extracellular matrix accumulation separates normal structures.
- Smooth-muscle-cell attrition contributes to reduced repair capacity.
- Similar alterations occur across inherited and acquired aortic disease.
- Cause must be inferred from clinical and other pathologic context, not from degeneration alone.

Abdominal aortic aneurysm: Characteristic site

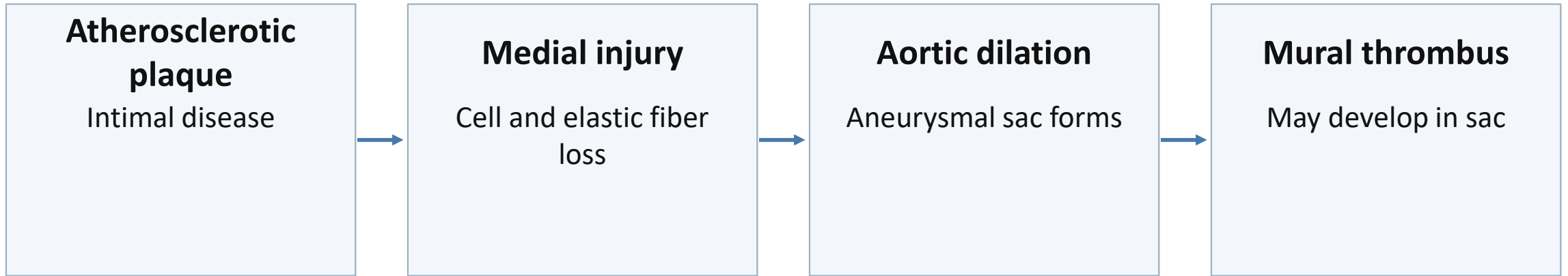


Schematic aortic segment; Branches and proportions simplified

AAA: Core epidemiologic associations

- Typically occurs in the abdominal aorta, often with associated iliac aneurysms.
- More frequent in men and in smokers; Uncommon before age 50.
- Atherosclerosis is a major contributor, but does not fully explain susceptibility.
- Inflammation, altered matrix turnover, and smooth-muscle damage also contribute.

AAA: Severe atherosclerosis and medial destruction



AAA gross morphology

- Most often located below the renal arteries and above the aortic bifurcation.
- May have saccular or fusiform configuration.
- Commonly shows a thinned, distorted wall with advanced atherosclerosis.
- Laminated but poorly organized mural thrombus is frequently present.

Mural thrombus in an AAA

- An aneurysmal lumen often contains a bland, poorly organized thrombus.
- The thrombus can become layered with ongoing deposition.
- Fragments may embolize to downstream arterial beds.
- Thrombus or sac expansion may impair a branch-vessel ostium.

Abdominal aneurysm variants

Conventional AAA

- Atherosclerosis prominent
- Medial attenuation
- Mural thrombus common

Inflammatory AAA

- Prominent inflammation
- Dense periaortic fibrosis
- May involve IgG4-related disease

Mycotic AAA

- Infected vessel wall
- Suppurative medial destruction
- Risk of rapid dilation / rupture

Inflammatory AAA: Fibrosis and inflammation

- Accounts for 5%–10% of abdominal aortic aneurysms.
- Dense lymphoplasmacytic infiltrates and periaortic scarring may extend into retroperitoneum.
- Back pain and elevated inflammatory markers may be presenting clues.
- Most cases lack overt inflammation in other arterial territories.

IgG4-related disease and inflammatory AAA

- A subset of inflammatory aneurysms belongs to IgG4-related disease.
- Storiform fibrosis and IgG4-positive plasma cells can occur in involved tissues.
- Retroperitoneal fibrosis may obstruct ureters and produce hydronephrosis.
- The systemic clinical context is important; Not every inflammatory AAA is IgG4-related.

Mycotic AAA: Infected wall is fragile

- Seeding by circulating microorganisms can establish infection in the aortic wall.
- Suppuration amplifies tissue destruction and weakens the media.
- Rapid aneurysm expansion and rupture can follow.
- “Mycotic” is a historical term and does not imply a fungal pathogen.

AAA: Routes to clinical injury

Aneurysm growth

Often silent

Rupture

Hemorrhage

Thrombus / plaque

Distal embolism

Mass / branch effects

Ischemia or
compression

AAA rupture risk rises with size

AAA diameter interval	Rupture risk stated in Robbins
≤4 cm	Reported as negligible
4–5 cm	Approximately 1% per year
5–6 cm	Approximately 11% per year
>6 cm	Approximately 25% per year

Textbook estimates for pathology teaching; Not a contemporary patient-specific risk calculator.

AAA: Incidental detection versus emergency

Often asymptomatic

- Incidentally found on examination or imaging
- May present as pulsatile abdominal mass
- Can coexist with extensive systemic atherosclerosis

Symptomatic / complicated

- Back or abdominal pain
- Sudden hemorrhage with rupture
- Limb or visceral ischemia from emboli or ostial compromise

AAA treatment concepts in the source

- Open graft repair and femoral-access endovascular stent grafting.
- Rupture carries far greater mortality than repair before rupture.
- Aneurysm size and change over time are major considerations in risk assessment.
- Treatment thresholds and surveillance schedules require current, patient-specific clinical guidance.

AAA indicates systemic vascular disease

- Aortic atherosclerosis often coexists with atherosclerosis in other territories.
- Patients with AAA may also have ischemic heart or cerebrovascular disease.
- Consequences are not confined to the aneurysmal aortic segment.
- The pathological diagnosis informs broader vascular risk assessment.

Thoracic aortic aneurysm: Etiologic spectrum

Acquired associations

- Hypertension
- Aortitis / inflammatory conditions
- Historically, tertiary syphilis

Genetic and structural associations

- Marfan syndrome
- Loeys–Dietz syndrome
- Bicuspid aortic valve with aneurysm

Hypertension and ascending aortic injury

- Hypertension is strongly associated with ascending aortic aneurysms.
- Vasa vasorum arteriolar narrowing can compromise outer medial perfusion.
- Medial smooth-muscle loss and maladaptive matrix turnover weaken the wall.
- The disease process differs in emphasis from atherosclerosis-associated infrarenal AAA.

TAA: Inherited and inflammatory causes

- Abnormal ECM scaffolding or TGF- β signaling predisposes to ascending aortic dilation.
- Aortitis may directly damage media and adventitia.
- Bicuspid aortic valve can coexist with thoracic aortopathy.
- An inherited syndrome may be suspected from age, family history, and extra-aortic features.

TAA: Symptoms follow anatomical compression

- May remain clinically silent before dissection or rupture.
- Chest pain can result from enlargement or erosion into neighboring structures.
- Compression of coronary arteries may cause myocardial ischemia.
- Esophageal, recurrent laryngeal nerve, or bronchial compression may cause dysphagia, hoarseness, or respiratory symptoms.

Abdominal versus thoracic aneurysm

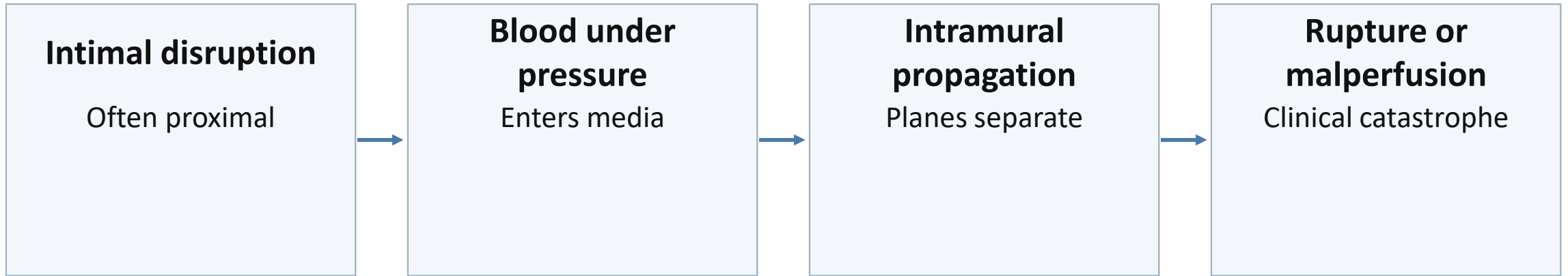
Abdominal aorta

- Atherosclerosis prominent
- Often infrarenal
- Mural thrombus common
- Rupture, embolization, branch obstruction

Thoracic / ascending aorta

- Hypertension a major association
- Genetic aortopathy important
- Vasa vasorum injury may contribute
- Compression, regurgitation, dissection or rupture

Aortic dissection: Wall separation by blood



Aortic dissection: Two major patient groups

Middle-aged / older adults

- Commonly men aged 40–60 in textbook series
- Marked association with hypertension
- May lack syndromic phenotype

Younger patients

- Connective tissue syndromes
- Marfan and related aortopathies
- Often additional clues to wall fragility

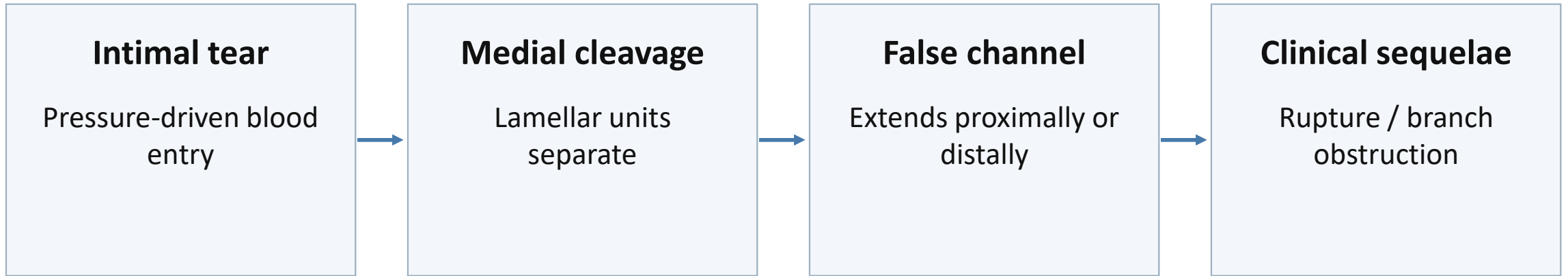
Other dissection contexts

- Iatrogenic injuries may occur during catheterization or cardiovascular procedures.
- Pregnancy-related dissection is rare and may relate to peripartum vascular and hemodynamic changes.
- Blunt chest trauma can produce intimal tearing near the ligamentum arteriosum.
- The initial trigger is unknown in many spontaneous dissections.

Dissection pathogenesis: Vulnerable media

- Hypertension is the major risk factor in the source.
- Inherited ECM or TGF- β signaling defects may predispose to injury.
- Medial degeneration is common but not required for dissection.
- Histologic degeneration alone is not a reliable predictor of an acute tear.

Intimal tear and medial propagation



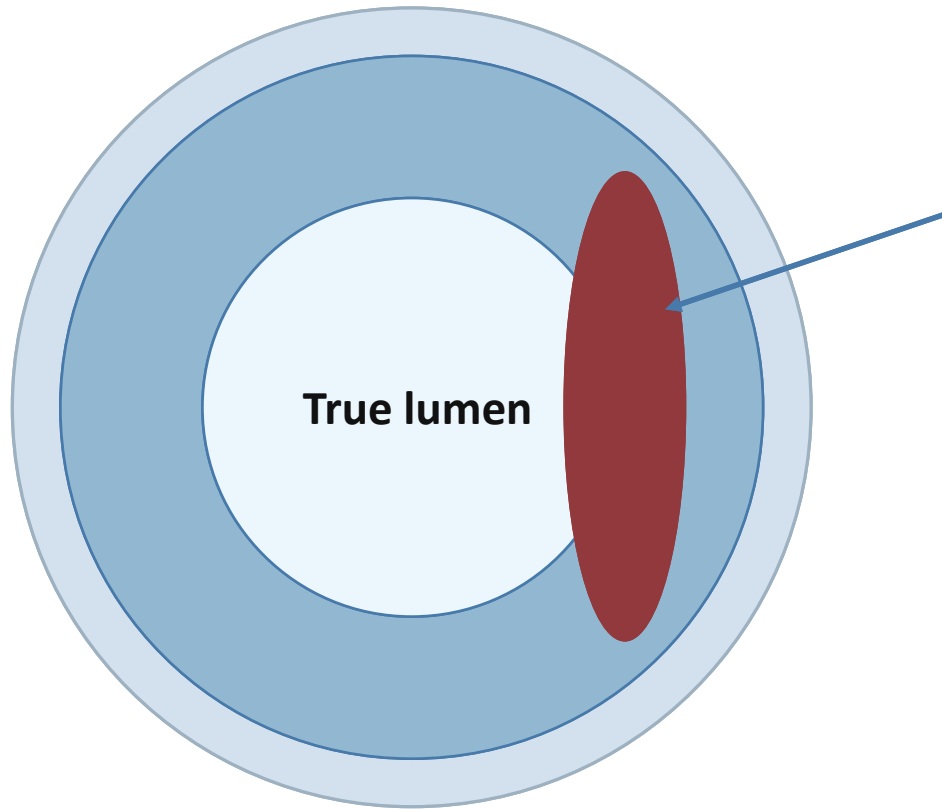
Intramural hematoma without an intimal tear

- Disruption of penetrating vessels from vasa vasorum can produce intramural hemorrhage.
- No initiating intimal communication need be visible.
- Blood still accumulates within the wall and compromises its structural integrity.
- This mechanism is related to, but should not be confused with, a classic entry tear.

Morphologic hallmarks of dissection

- Most spontaneous intimal tears begin in the ascending aorta.
- The tear may be transverse, sharp, and irregular.
- Blood tracks along media or a medial–adventitial plane.
- Propagation may extend toward the heart or into distal aorta and branches.

Dissection morphology: Characteristic planes



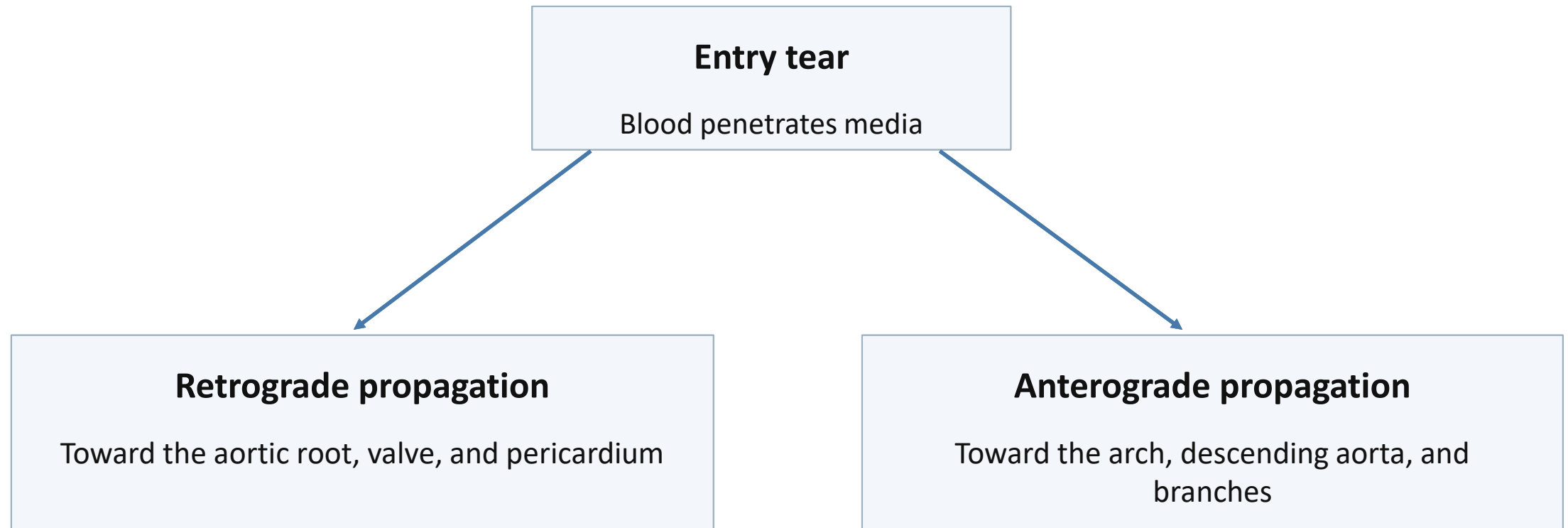
Intramural false channel

Blood separates medial lamellar planes

Outer wall may eventually rupture

Schematic cross-section; No stain or specimen represented

Dissection can propagate in either direction



Both directions may occur in the same dissection.

Re-entry may create a double-barreled aorta

- A distal second intimal tear can reconnect the false channel to the true lumen.
- The re-entry pathway may avert some extraaortic hemorrhage.
- Persistent false channels may endothelialize with chronicity.
- A double-lumen vessel is a morphological consequence, not evidence of a benign process.

Stanford and DeBakey classification

Type A (proximal)

- Ascending aorta involved
- May extend into descending aorta
- Includes DeBakey I and II
- Higher risk of proximal complications

Type B (distal)

- Ascending aorta spared
- Usually starts beyond left subclavian artery
- Corresponds to DeBakey III
- Often initially managed without operation

Classification is based on the anatomical extent of the dissection.

Type A dissection: Proximal involvement

- Any dissection involving the ascending aorta is Stanford type A.
- DeBakey I extends from the ascending aorta distally; DeBakey II is confined proximally.
- Root and pericardial complications make these especially dangerous.
- Prompt recognition and urgent operative assessment.

Type B dissection: Ascending aorta spared

- Usually begins after the origin of the left subclavian artery.
- Corresponds to DeBakey type III classification.
- Still may cause rupture or malperfusion of visceral or peripheral branches.
- The source describes many uncomplicated cases as initially treated with antihypertensive therapy.

Aortic rupture: Dangerous spaces

Proximal / thoracic

- Hemopericardium and tamponade
- Hemothorax
- Rapid exsanguination

Distal extension

- Hemoperitoneum or retroperitoneal bleeding
- Distal arterial compromise
- Visceral and limb ischemia

Aortic root and valve complications

Proximal / retrograde extension

Aortic root and adjacent pericardial region

Aortic valve annulus affected

Acute aortic regurgitation / insufficiency

Rupture into pericardium

Hemopericardium with cardiac tamponade

Branch involvement produces malperfusion

Head / heart

- Carotid or coronary compromise
- Neurologic deficits or myocardial ischemia

Abdomen / limbs

- Renal or mesenteric ischemia
- Iliac obstruction and limb ischemia

Spinal circulation

- Potential spinal cord ischemic injury
- Serious neurologic deficits

Clinical presentation of aortic dissection

- Classic pattern is sudden onset of severe chest pain radiating to the back.
- Pain may migrate as the dissection propagates longitudinally.
- The presentation can resemble acute myocardial infarction.
- Hemodynamic instability or organ ischemia indicates major complications.

Dissection: Pathology informs urgency

- Hemodynamic pressure drives progression of the dissecting hematoma.
- Reduction of arterial pressure and shear stress is a key immediate management principle.
- Type A disease generally requires urgent surgical evaluation.
- Type B management depends on whether rupture, malperfusion, or other complications are present.

Clinical case: Infrarenal aneurysm

Clinical vignette

An older smoker has an incidentally identified infrarenal aortic dilation with layered mural thrombus.

Discussion question

Which structural changes favor aneurysm formation, and what complications should the pathologist anticipate?

Answer and explanation are in the speaker notes.

Clinical case: Sudden severe chest pain

Clinical vignette

A patient with long-standing hypertension develops abrupt chest pain radiating between the scapulae.

Discussion question

Which aortic lesion must be considered, and why does ascending involvement alter prognosis?

Answer and explanation are in the speaker notes.

Integrated comparison of aortic wall lesions

Aneurysm	Pseudoaneurysm	Dissection
• Wall dilation • Wall weakening dominates • Rupture / thromboembolism	• Contained wall rupture • Extravascular hematoma • Rupture / compression	• Blood within the wall • Lamellar separation • Rupture / malperfusion

Four mechanisms converge on aortic weakness

ECM proteolysis

MMP–TIMP imbalance

Poor matrix quality

Collagen / fibrillin
defects

Altered signaling

TGF- β pathway

SMC loss

Medial ischemia

Mechanisms can overlap in one patient.

Clinicopathologic synthesis

- True aneurysm, pseudoaneurysm, and dissection are structurally distinct.
- Extracellular matrix and smooth-muscle integrity determine resistance to pulsatile stress.
- Atherosclerosis predominates as a cause of infrarenal AAA; Hypertension is prominent in ascending aortic disease.
- Medial degeneration is frequent but nonspecific.
- Rupture and branch-vessel ischemia account for many acute presentations.

Primary reference and source boundaries

Robbins, Cotran & Kumar: Pathologic Basis of Disease

- 11th edition (2026), Chapter 11: Blood Vessels.
- Aneurysms and Dissection (p. 458 onward).
- Adjacent parent-context sections: Abdominal Aortic Aneurysm, Thoracic Aortic Aneurysm, and Aortic Dissection (pp. 460–463).
- Source basis: supplied extracted chapter Markdown and topic unit files; original page PDFs not provided.