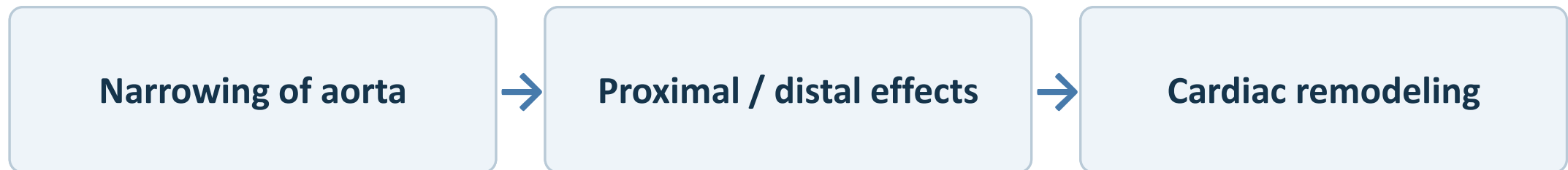


Coarctation of the Aorta

Congenital aortic obstruction: anatomy, morphology and hemodynamics



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Learning objectives

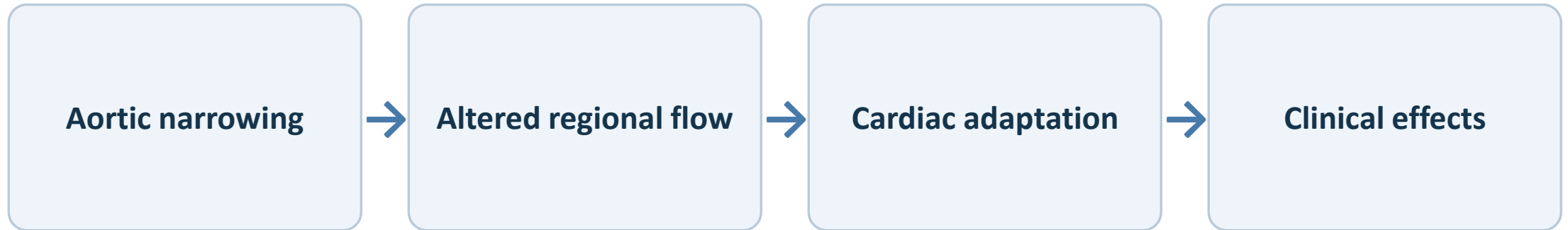
Localize the narrowing relative to the left subclavian artery and ductus arteriosus.

Distinguish the classic preductal and postductal patterns.

Predict cardiac remodeling, regional perfusion and presenting signs.

Explain collateral vessels, rib notching and the rationale for repair.

Coarctation is an obstructive congenital defect



Coarctation means a localized or tubular constriction of the aorta.

The primary lesion impedes flow through a great vessel rather than constituting a primary septal shunt.

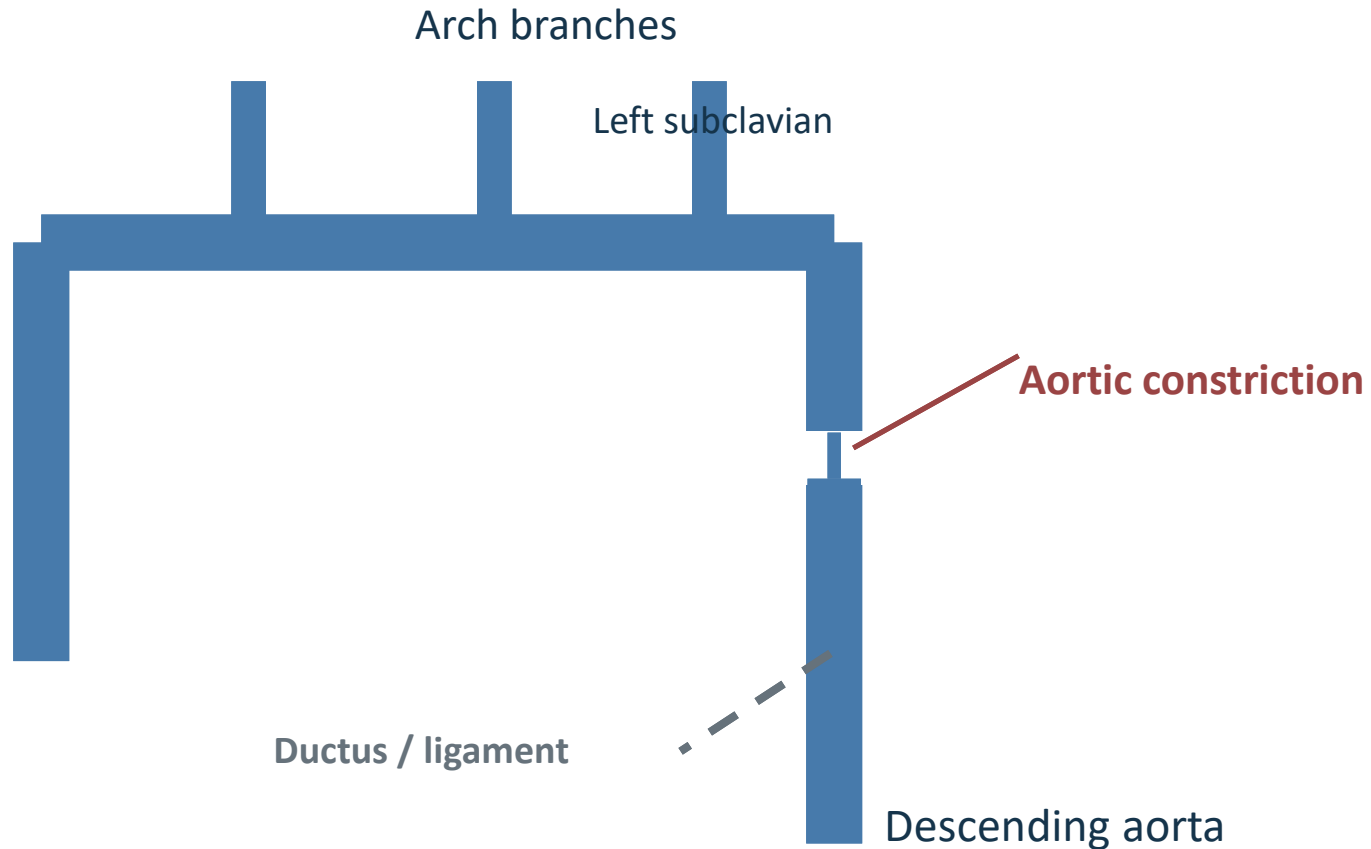
Clinical effects depend strongly on severity and ductal patency.

Where the lesion belongs in congenital heart disease

Category	Representative lesion
Great-vessel obstruction	Aortic coarctation
Valvular obstruction	Aortic or pulmonary stenosis
Associated shunt	PDA may coexist with coarctation

Obstructive lesions may involve a great vessel, cardiac valve or chamber.

The arch–ductus relationship determines the form

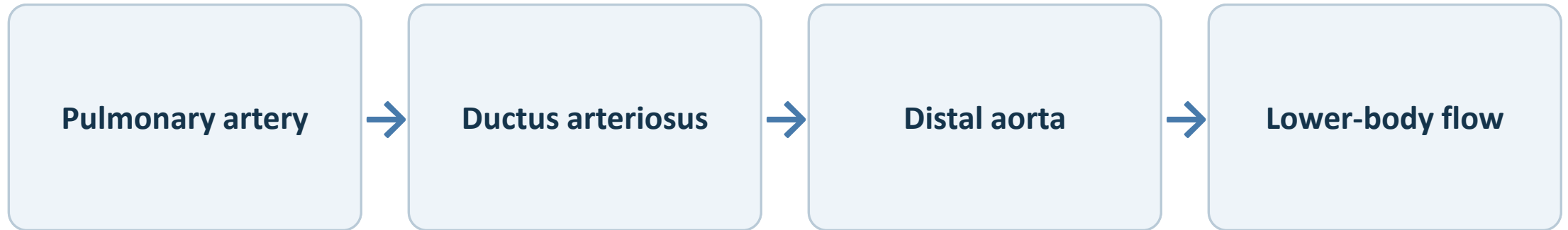


The ductus joins the aorta distal to the left subclavian artery.

The left subclavian origin anchors the proximal landmark.

Narrowing may lie proximal to a patent ductus or adjacent to its closed remnant.

Fetal ductal flow bypasses the lungs

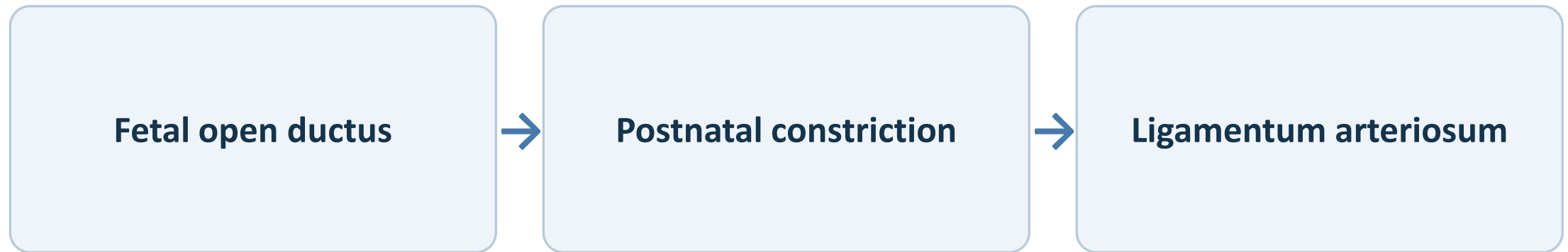


In fetal life, the ductus connects the pulmonary artery to the descending aorta.

Flow through this connection bypasses the unventilated fetal lungs.

The relationship becomes crucial if the arch is obstructed.

Normal postnatal ductal closure changes the circuit



In a healthy term infant, functional ductal closure usually occurs within 1–2 days.

The closed ductus later becomes the ligamentum arteriosum.

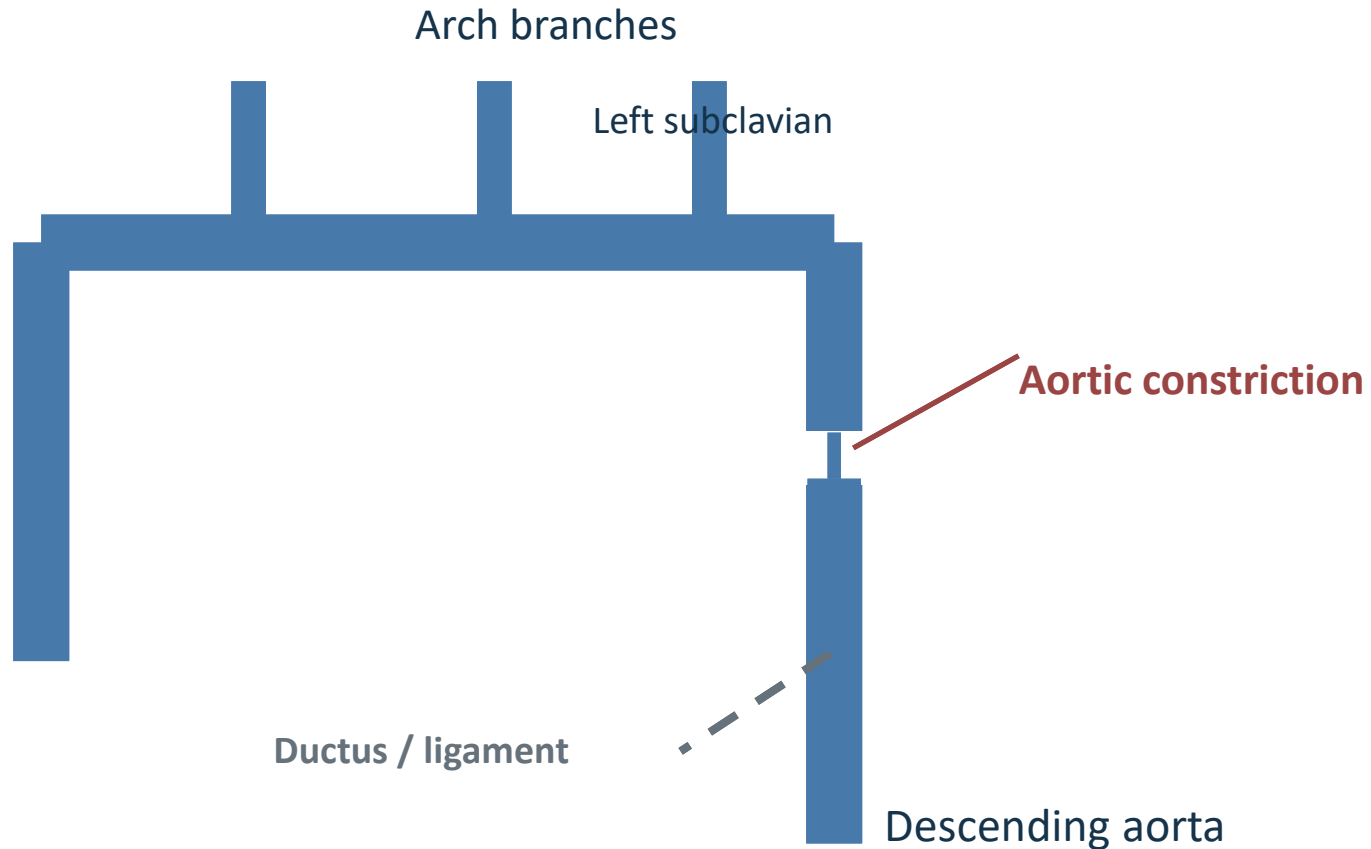
An infant with severe proximal obstruction may depend on the open ductus for distal flow.

Two classic patterns are described

Feature	Preductal pattern	Postductal pattern
Narrowing	Tubular hypoplasia	Discrete ridge
Ductus	Often patent	Typically closed
Classical onset	Early life	Later recognition

Infantile or preductal: Tubular hypoplasia proximal to a PDA.

Preductal and postductal are anatomic terms



“Preductal” identifies a lesion proximal to the ductal junction.

“Postductal” describes the ridge-like lesion at the ligamentum arteriosum.

Age at diagnosis alone cannot substitute for description of the anatomy.

Distribution: Male predominance

Coarctation as twice as frequent in males as in females.

This is a descriptive epidemiologic association, not an anatomic diagnostic feature.

Examination must still be driven by perfusion and pulse findings.

Turner syndrome is a notable association



Coarctation is often encountered in females with Turner syndrome.

The syndromic association links aortic abnormalities to congenital cardiac evaluation.

It does not imply that coarctation is confined to patients with a recognizable syndrome.

Bicuspid aortic valve commonly coexists

Finding	Site	Importance
Coarctation	Aortic segment	Fixed vessel obstruction
Bicuspid valve	Aortic valve	Associated valve anatomy

A bicuspid aortic valve in approximately 50% of coarctation cases.

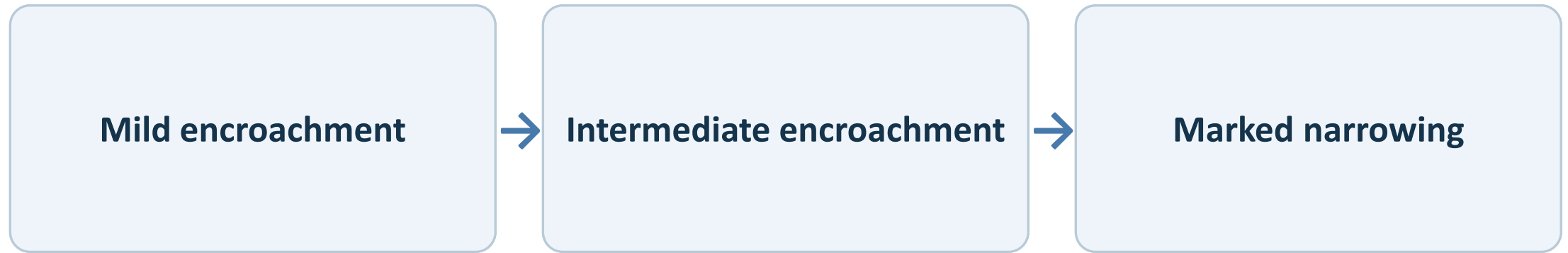
Other structural associations may coexist

Less frequent associations include congenital aortic stenosis, atrial septal defect and ventricular septal defect.

Mitral regurgitation and berry aneurysms of the circle of Willis are also listed.

Associated abnormalities may change the overall clinical picture.

The residual aortic lumen can vary greatly

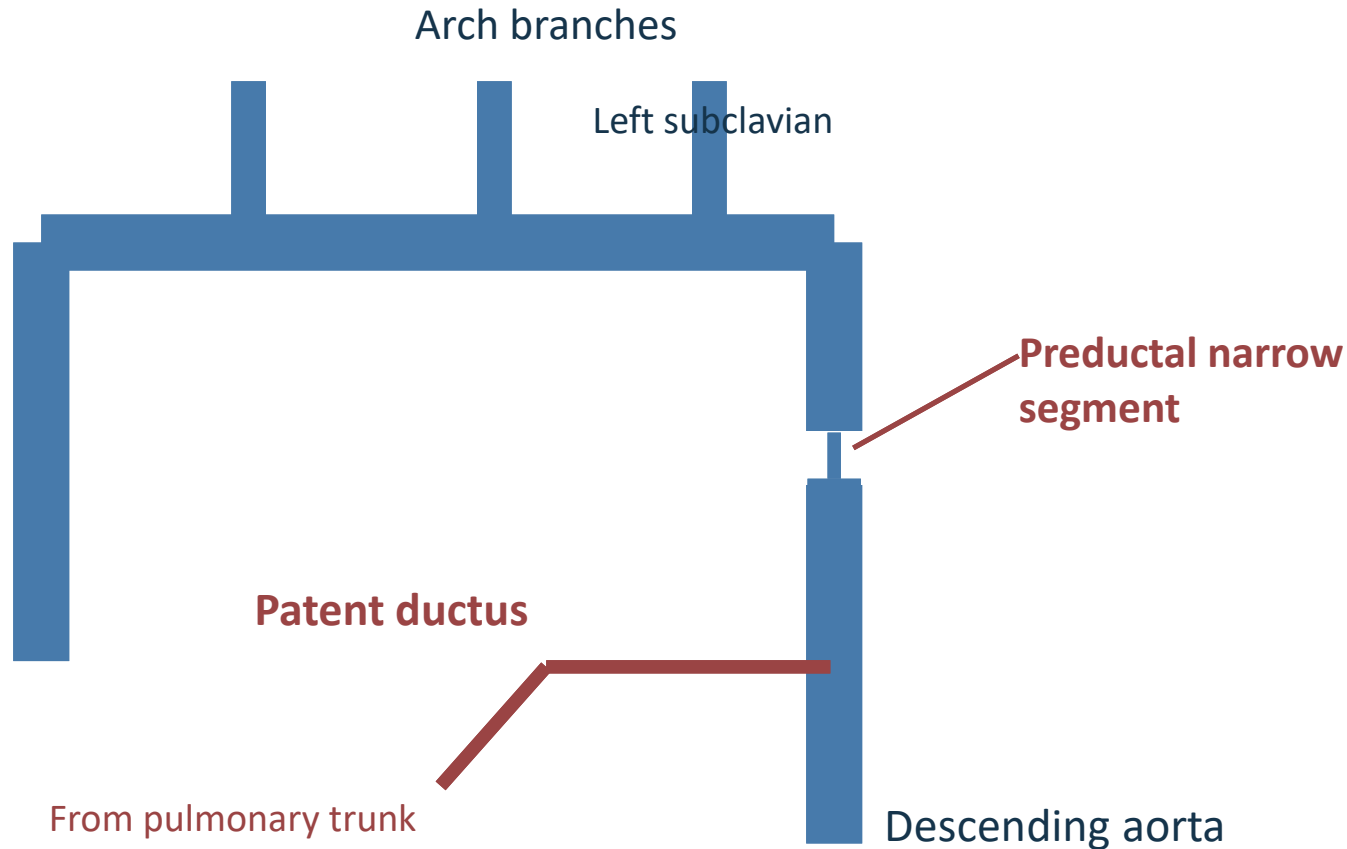


One coarctation may leave only a small residual channel.

Another produces minimal luminal compromise.

The anatomic label alone therefore does not quantify the functional obstruction.

Preductal morphology: A hypoplastic arch segment

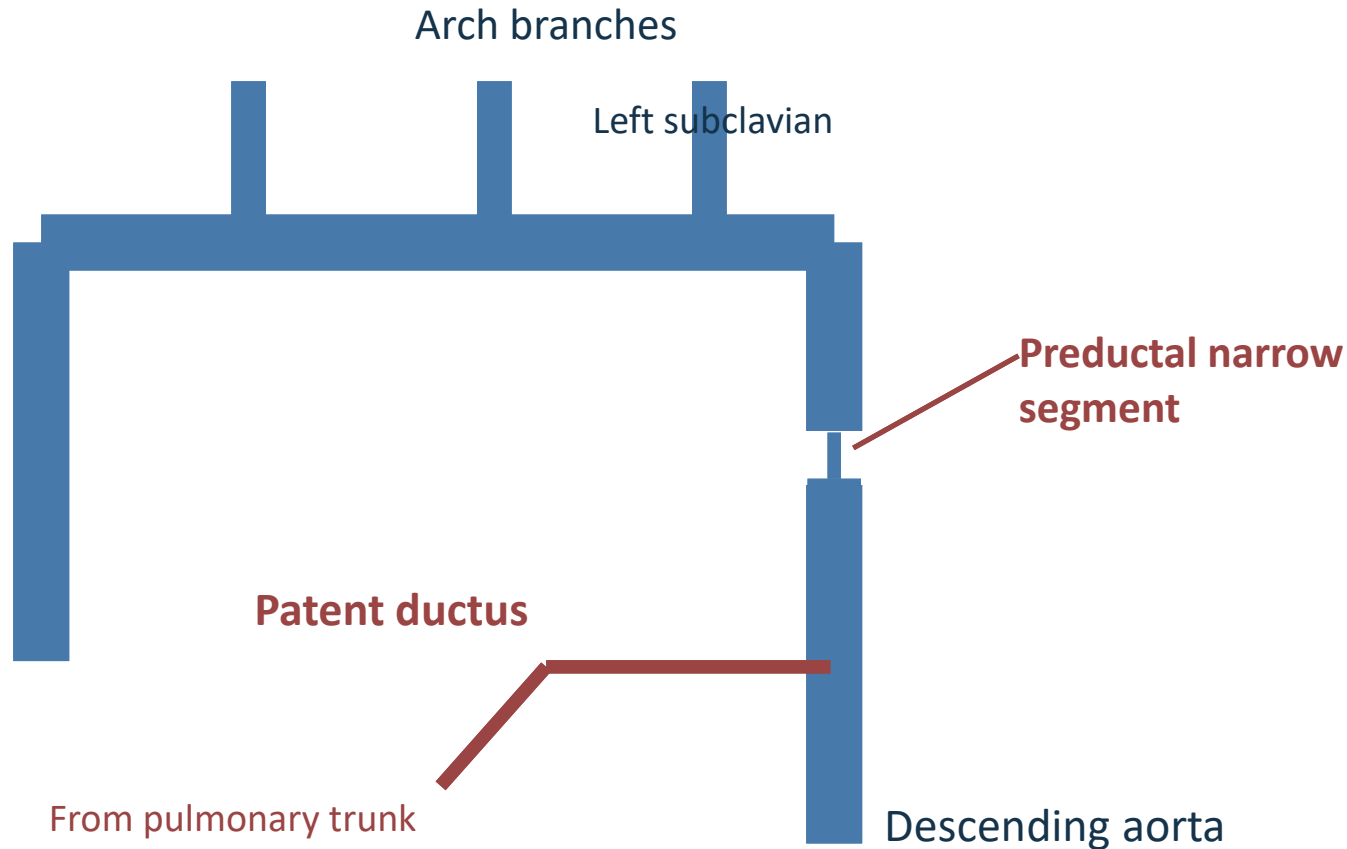


The infantile pattern features tubular hypoplasia of the arch proximal to a PDA.

This differs from a short, ridge-like adult-pattern narrowing.

Obstruction extends along a segment rather than merely across a focal ridge.

Preductal narrowing has a defined location

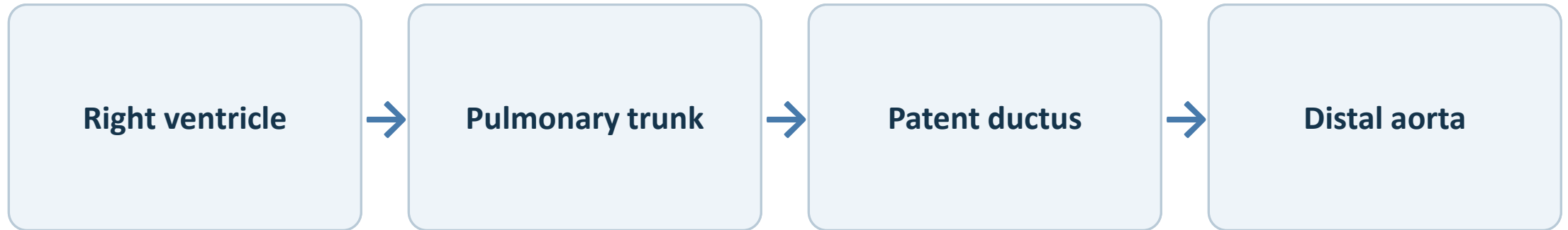


The constricted segment lies between the left subclavian artery and ductus arteriosus.

The narrowing is circumferential.

The distal aorta may receive much of its perfusion from the ductus.

A patent ductus can sustain distal aortic supply

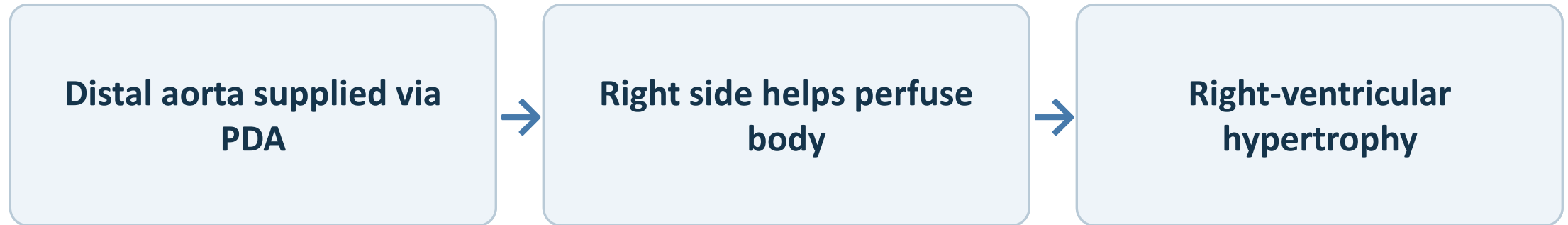


With severe preductal coarctation, the ductus supplies blood to the distal aorta.

The main supplying route is then from the pulmonary arterial circuit.

Distal perfusion is consequently linked to right-sided cardiac output.

Why the right ventricle becomes hypertrophied



With ductal-dependent distal flow, the right heart contributes to systemic perfusion.

This adds a substantial functional burden to the right ventricle.

Right-ventricular hypertrophy is the characteristic adaptation described in this pattern.

Why the pulmonary trunk can dilate

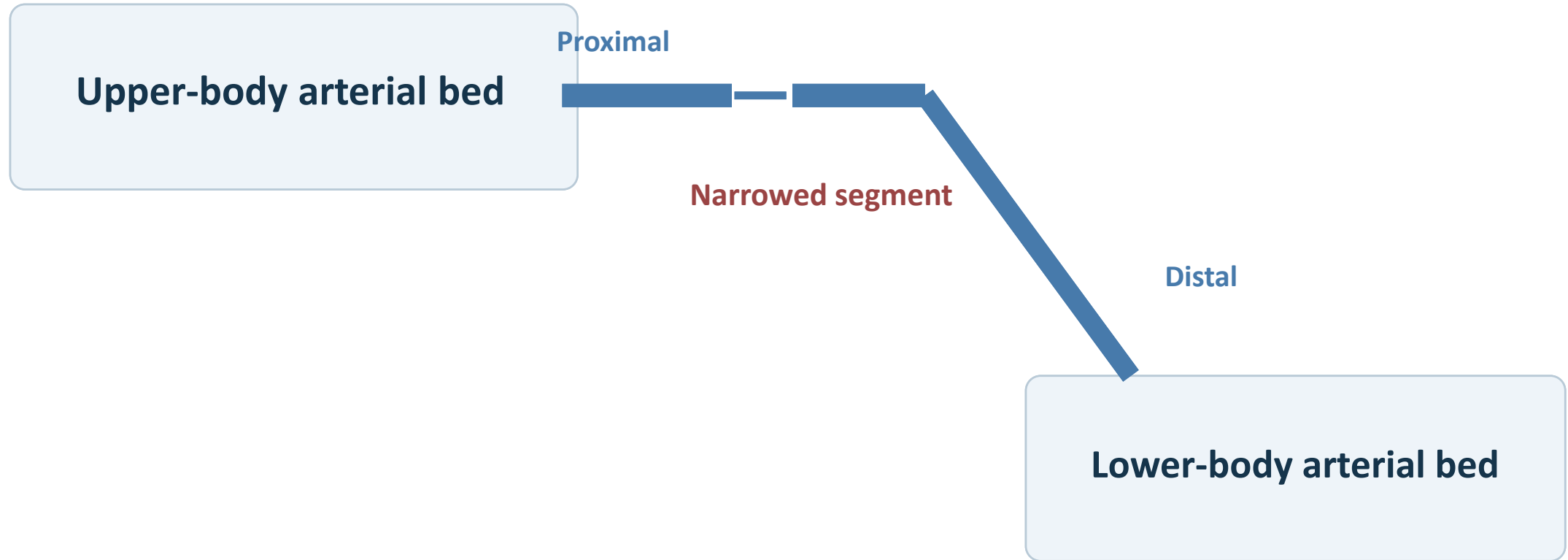


The pulmonary trunk accommodates increased flow in the ductal-dependent configuration.

Increased throughput is associated with dilation of the pulmonary trunk.

The enlargement reflects hemodynamics, not a separate pulmonary valve stenosis.

Preductal perfusion creates an oxygenation divide



Interpret the territory contrast through the position of the stenosis.

Simplified schematic

Differential cyanosis: The key mechanism

Territory	Predominant supply	Clinical correlate
Upper body	Aortic arch branches	Relatively better oxygenation
Lower body	Ductus to distal aorta	Differential cyanosis

The upper body receives blood from vessels arising proximal to the obstruction.

The infantile pattern often declares itself early

Coarctation with a PDA commonly produces manifestations in early life.

CASE INTERPRETATION

Link the observations to the site and direction of blood flow.
pathway.

**Neonate with lower-body
cyanosis**

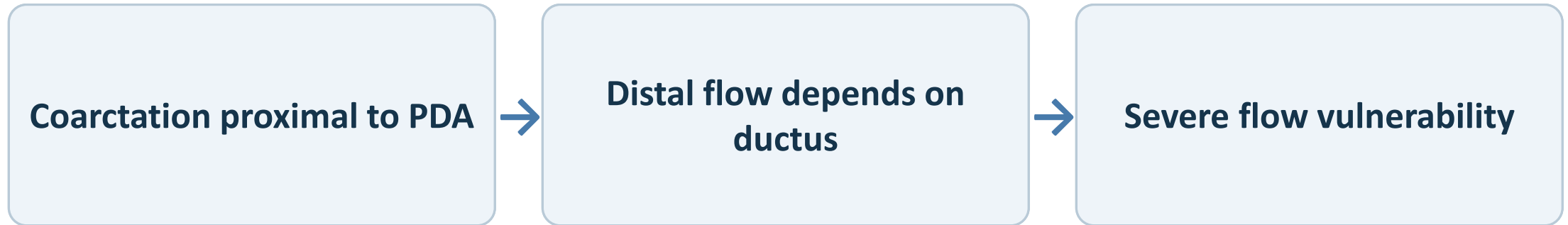


Ductus remains patent



**Which circulation is
supplying the legs?**

Ductal dependence creates neonatal vulnerability

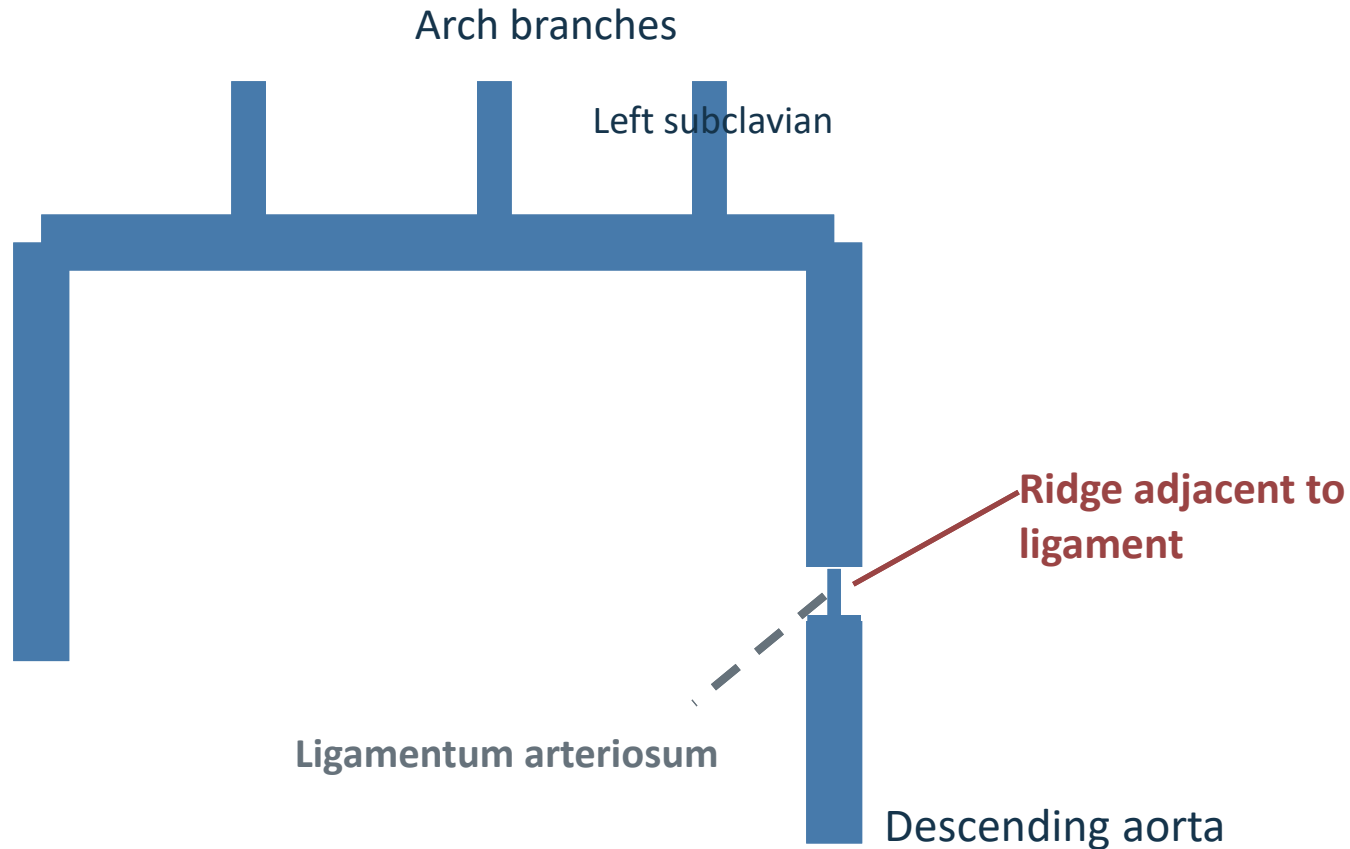


If distal systemic flow relies on a patent ductus, losing ductal flow jeopardizes systemic perfusion.

The source describes high neonatal mortality in severe cases without intervention.

The need for timely treatment follows from the altered circuit, not merely from the measured diameter.

Postductal morphology: A discrete ridge

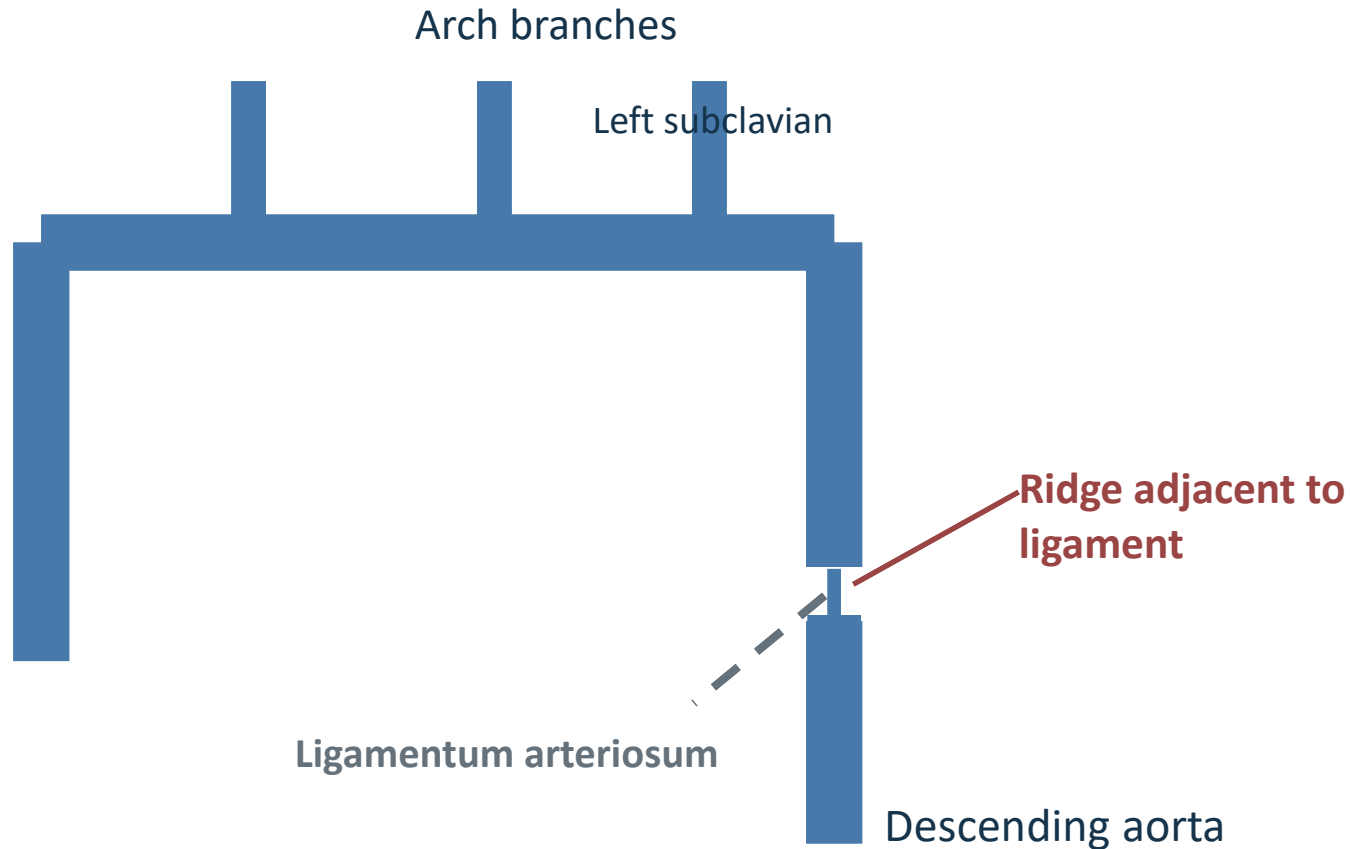


The more common adult pattern has a sharply constricted aortic segment.

A ridge-like infolding sits next to the nonpatent ligamentum arteriosum.

The lesion is focal rather than uniformly tubular.

The ductus has become a ligament in this pattern



Postductal coarctation is classically beside the ligamentum arteriosum.

The ligament is the fibrous remnant of the closed ductus.

The descending aorta must receive its flow across the narrowed segment.

The ridge derives from the aortic media

Layer or tissue	Morphologic role
Smooth muscle	Component of constricting ridge
Elastic fibers	Component of constricting ridge
Aortic media	Source of ridge constituents

The constricting tissue contains smooth muscle and elastic fibers.

A tight focal ridge restricts distal flow

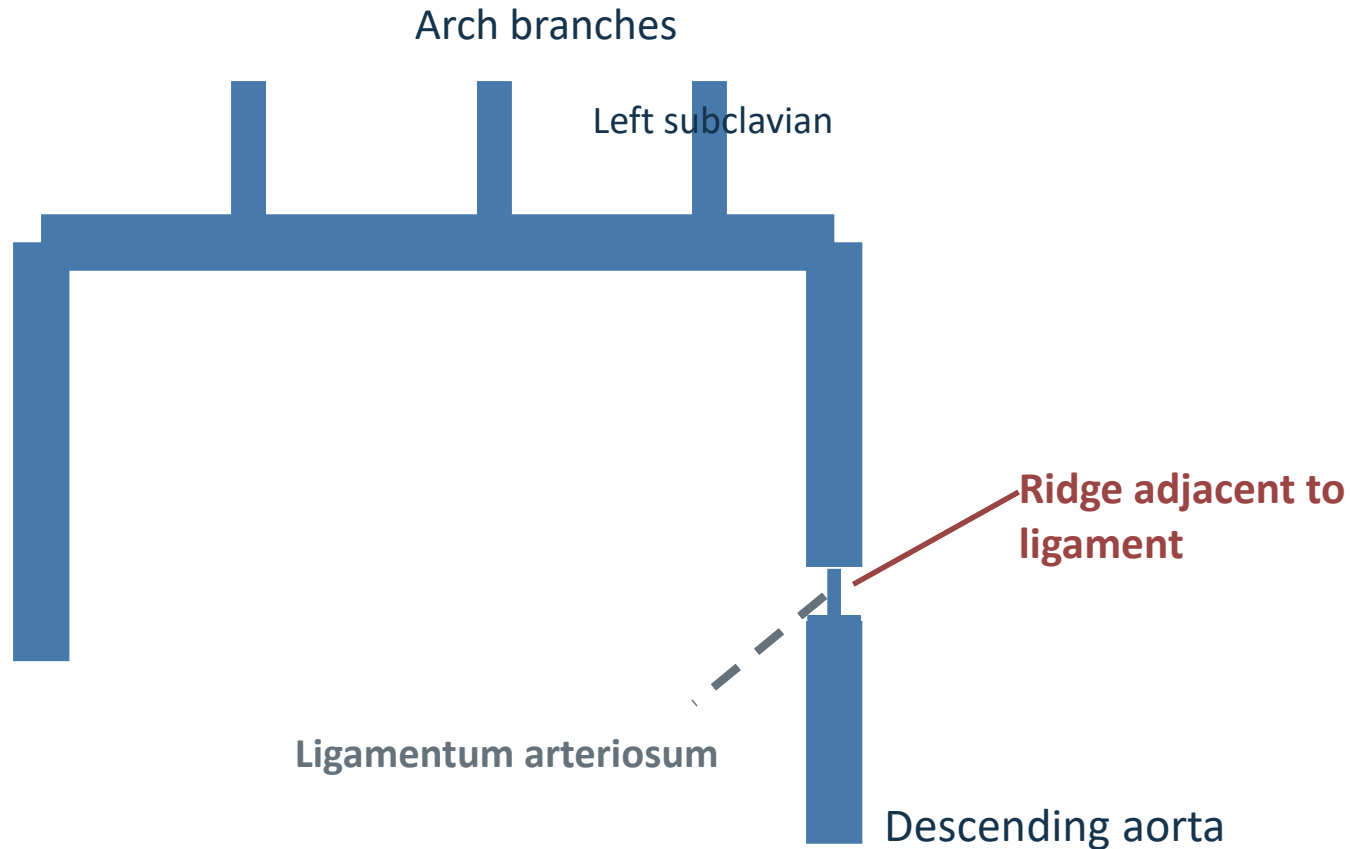


The degree of narrowing varies among postductal lesions.

A tight constriction creates resistance between arch and descending aorta.

Proximal and distal vascular beds can therefore have markedly different pressure and perfusion.

Proximal dilatation is a characteristic response

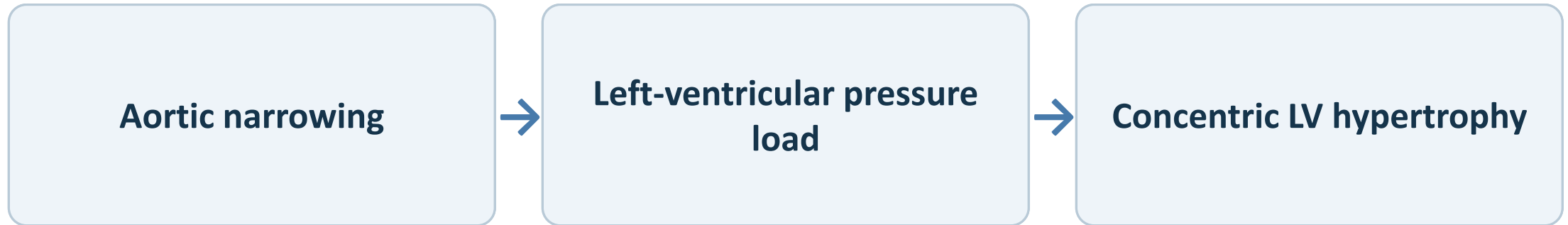


The aortic arch and major branches proximal to a postductal lesion may be dilated.

Their enlargement is part of the morphology.

The location of proximal vessels helps orient gross anatomic interpretation.

The left ventricle faces a pressure load

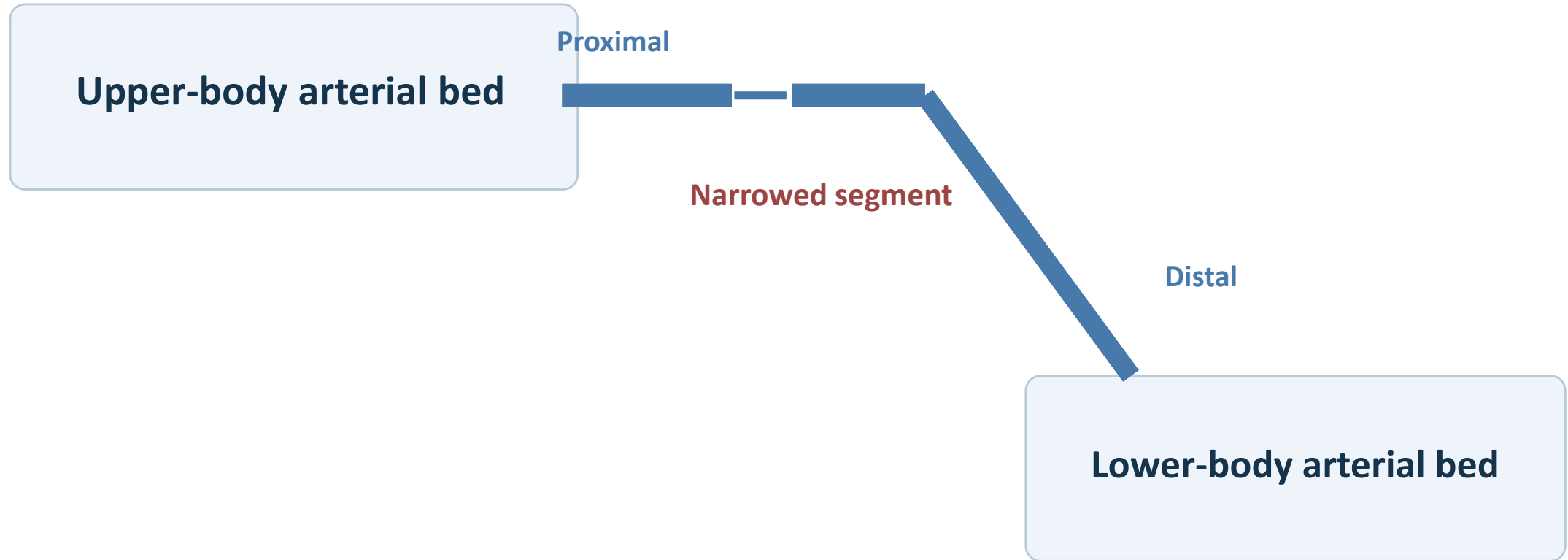


The left ventricle ejects against the narrowed aortic outflow pathway.

The chronic increased workload produces left-ventricular hypertrophy.

A concentric pattern with long-standing pressure overload.

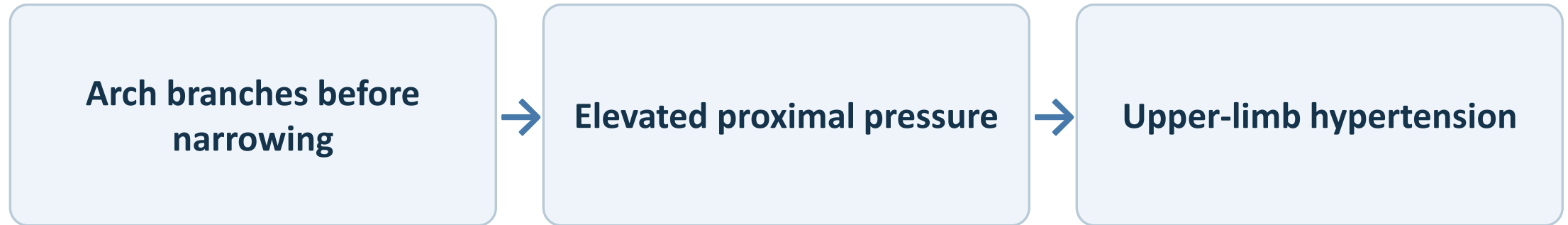
The central hemodynamic split is proximal versus distal



Interpret the territory contrast through the position of the stenosis.

Simplified schematic

Upper-extremity hypertension is an important clue



The arm circulation arises from proximal arch vessels in the classic pattern.

Upper-extremity hypertension is consequently characteristic.

The finding is particularly informative when contrasted with weak lower-limb pulses.

Lower-extremity hypotension follows distal obstruction



The legs are supplied via the aorta beyond the narrowed segment.

Reduced distal pressure is a hallmark of the typical postductal configuration.

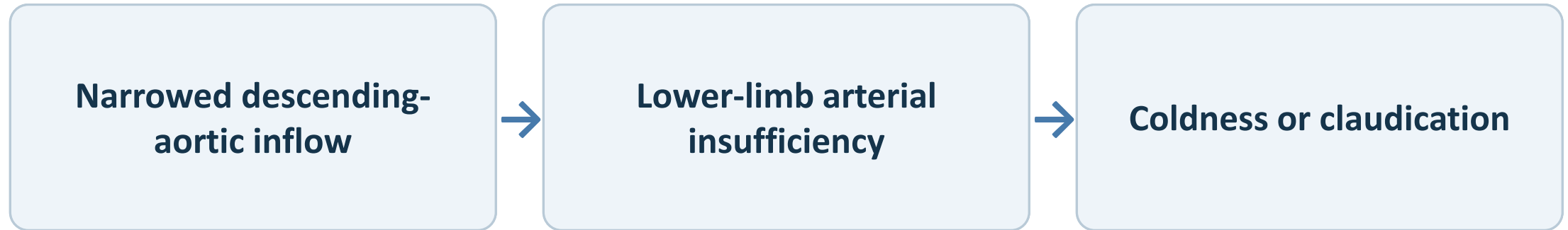
Severity and collateral flow can modify the clinical expression.

Weak leg pulses reflect delayed distal perfusion

Upper limbs	Lower limbs
Higher pressure	Lower pressure
Stronger pulses	Weaker pulses
Proximal territory	Distal territory

Pulse strength in the legs is often diminished compared with the arms.

Leg ischemic symptoms have a vascular basis



Cold lower extremities reflect reduced arterial delivery to distal tissues.

Claudication reflects exercise-related arterial insufficiency.

The same aortic lesion explains both systemic hypertension above and underperfusion below.

Collateral flow may bypass a chronic obstruction

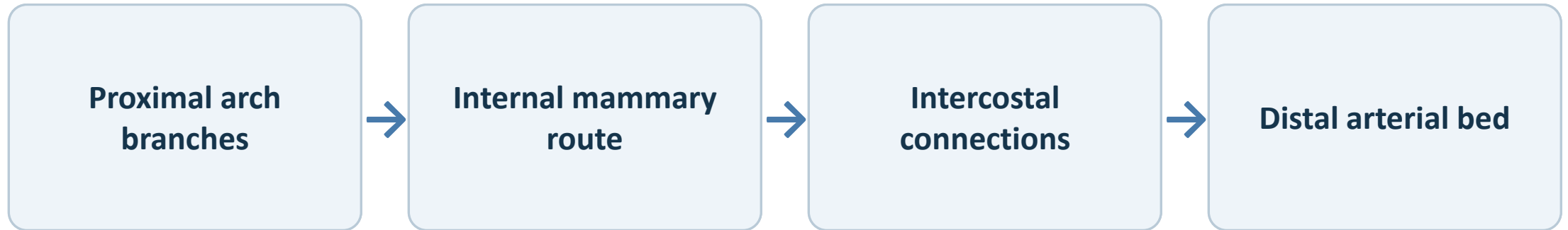


Over time, alternative arterial pathways link pre- and post-coarctation vessels.

Internal mammary and intercostal arteries enlarge as collateral conduits.

These networks can preserve distal flow despite the persistent anatomic narrowing.

Internal mammary arteries participate in collaterals



The source identifies enlarged internal mammary arteries as part of the collateral circulation.

These channels help connect the proximal circulation to intercostal pathways.

Their enlargement is a compensatory response to chronic outflow obstruction.

Intercostal arteries form visible collateral routes



Intercostal arteries are specifically identified as enlarged collateral vessels.

Their course along the rib surfaces explains associated bony changes.

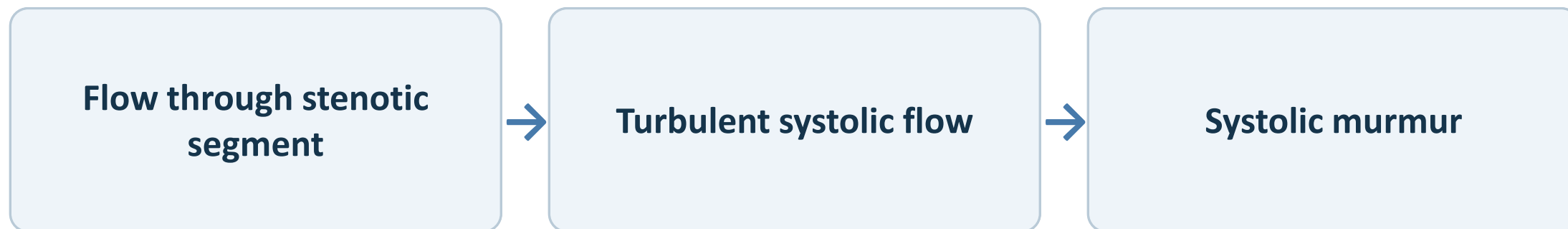
This is an adaptation to chronic stenosis rather than the primary lesion itself.

Inferior rib notching is the radiographic correlate

Observation	Pathologic explanation
Enlarged intercostal arteries	Collateral channels
Rib undersurface erosion	Chronic vascular remodeling
Rib notching on radiograph	Bony sign of collateral pathway

Enlarged intercostal arteries may erode the undersurfaces of ribs.

Murmurs arise in significant coarctation



Hemodynamically significant lesions can produce systolic murmurs.

The murmur persists through systole description.

The auscultatory finding should be interpreted alongside limb pressures and pulses.

A palpable thrill may accompany the murmur

Some significant coarctations cause a vibratory thrill.

A thrill indicates a palpable mechanical correlate of disturbed flow.

Its absence does not exclude a clinically important narrowing.

Cardiac remodeling differs by ductal physiology

Preductal / PDA	Postductal / no PDA
Right side supplies distal aorta	LV ejects across narrowing
RV hypertrophy	Concentric LV hypertrophy
Pulmonary trunk may dilate	Proximal arch may dilate

In the severe preductal duct-dependent configuration, the right ventricle may hypertrophy.

Severity and ductal status jointly determine presentation

Determinant	Clinical implication
Lumen compromise	Magnitude of flow obstruction
Ductal patency	Alternative route to distal aorta
Duration of lesion	Time for hypertrophy and collaterals

Tight narrowing with a patent ductus can generate early systemic dependence and differential cyanosis.

A child may be asymptomatic despite coarctation

Postductal disease may be clinically silent in childhood.

Recognition can be delayed into adult life.

A normal symptom history cannot replace upper-versus-lower extremity vascular assessment.

Two contrasting clinical courses

Infantile presentation	Later presentation
Often patent ductus	Usually no PDA
Lower-body cyanosis	Arm–leg pressure discrepancy
Right-ventricular burden	Left-ventricular burden

With ductal-dependent severe narrowing: Early cyanosis and critical perfusion vulnerability.

Bedside assessment follows vascular territories

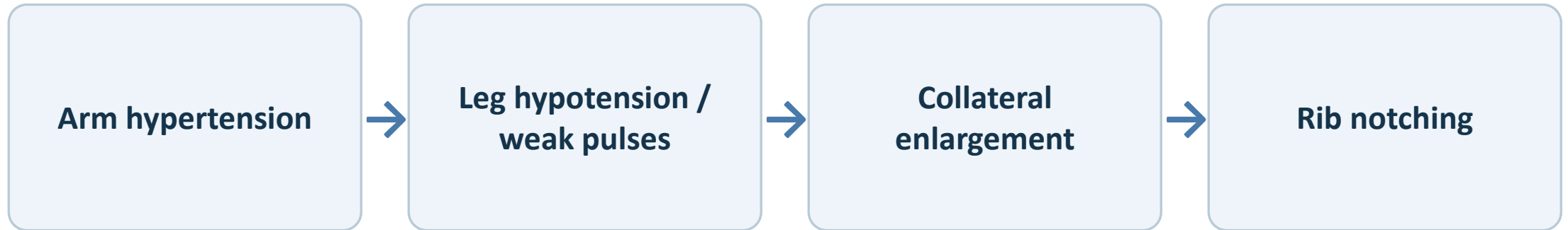
Compare blood pressure in upper and lower extremities.

Palpate and compare arm and leg pulses.

Assess for systolic murmur, lower-limb coldness or claudication.

Consider associated structural lesions when coarctation is identified.

Clinical evidence forms a coherent chain



Upper-limb hypertension localizes pressure proximal to the obstruction.

Weak leg pulses and leg hypotension localize the perfusion deficit distally.

Collateral-related rib notching demonstrates long-standing compensatory supply.

Coarctation with PDA is not isolated PDA

Isolated PDA	Coarctation with PDA
Shunt lesion	Obstruction plus ductal connection
May be asymptomatic at birth	May cause immediate neonatal effects
Effect depends on shunt flow	Ductus may supply distal systemic flow

A PDA alone is a communication between pulmonary artery and aorta.

The associated defects can modify findings

Associated anomaly	Additional anatomic feature
Bicuspid aortic valve	Abnormal valve cusp number
Aortic stenosis	Valvular outflow narrowing
ASD / VSD	Interatrial / interventricular communication

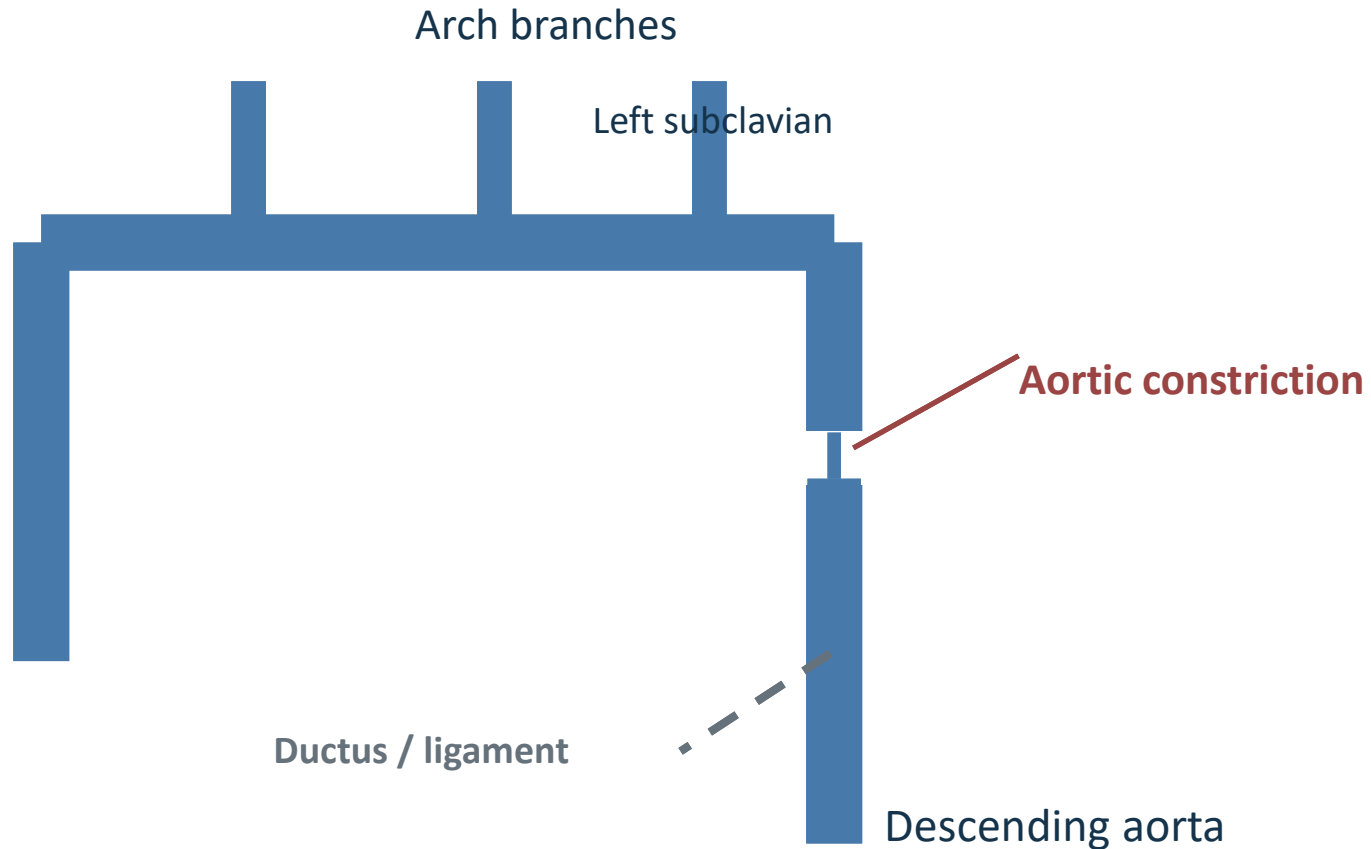
A bicuspid aortic valve is the common associated lesion named by Robbins.

Other obstruction sites must be distinguished

Entity	Primary obstruction
Aortic coarctation	Aortic arch / isthmus
Aortic stenosis	Aortic valve
Pulmonary stenosis	Pulmonary valve

Aortic stenosis obstructs flow at the valve rather than at the descending arch.

Gross morphology: What to identify



Identify the arch branches and descending aorta to orient the lesion.

Determine whether there is a long hypoplastic segment or a focal ridge.

Locate the constriction relative to the ductal insertion or ligament.

Inspect for proximal dilatation and other associated structural findings.

Simplified schematic

Morphology predicts which ventricle remodels

Structural configuration	Hemodynamic adaptation
Preductal + PDA	RV hypertrophy; Pulmonary-trunk dilation
Postductal + closed ductus	LV hypertrophy; Proximal aortic dilation

Preductal obstruction with PDA recruits the right ventricle for distal systemic flow.

Case 1 · Early neonatal cyanosis

A neonate has cyanosis that is more marked in the lower extremities.

CASE INTERPRETATION

Link the observations to the site and direction of blood flow.

Patent ductus arteriosus

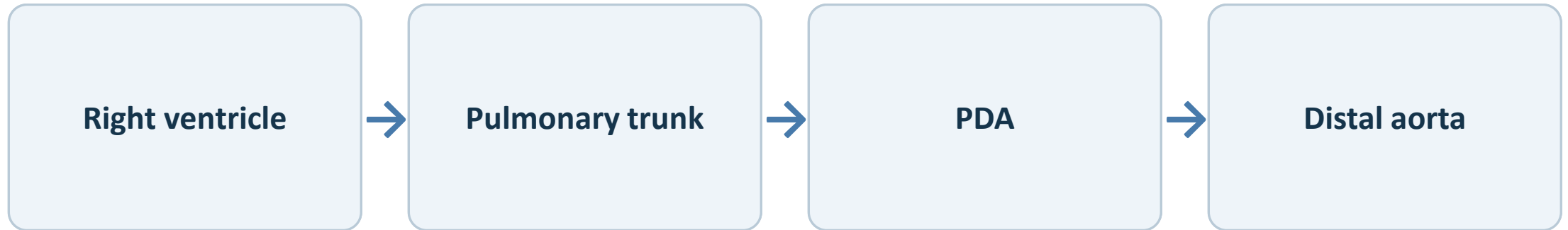


Preductal narrowing



Lower-body cyanosis

Case 1 · Reconstruct the blood-flow route



Pulmonary-trunk blood traverses the PDA to reach the distal aorta.

Its lower oxygen saturation explains the regional cyanosis.

Right-ventricular hypertrophy and pulmonary-trunk dilation are expected adaptations.

Case 2 · Hypertension with diminished leg pulses

An older patient has upper-extremity hypertension but weaker lower-limb pulses.

CASE INTERPRETATION

Link the observations to the site and direction of blood flow.

Proximal hypertension

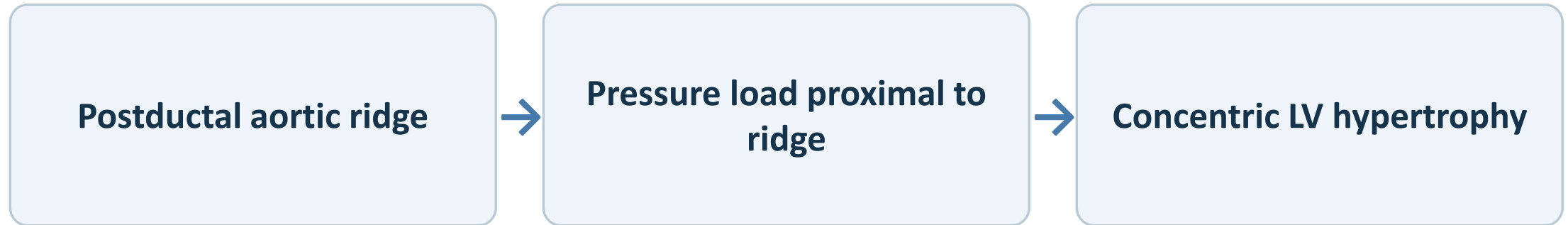


Distal pulse diminution



Focal ridge near ligament

Case 2 · Link the signs to the lesion



The left ventricle pumps against a narrowed systemic outflow tract.

The resulting chronic pressure load produces concentric left-ventricular hypertrophy.

A proximal–distal pressure disparity explains arm–leg findings.

Case 3 · Rib notching in a minimally symptomatic adult

An adult with few symptoms has upper-body hypertension and inferior rib notching.

CASE INTERPRETATION

Link the observations to the site and direction of blood flow.

Upper-arm hypertension

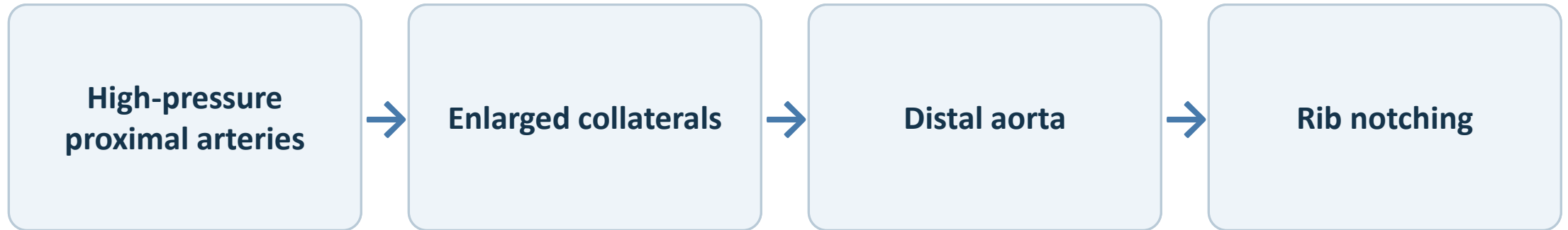


Rib undersurface notching



Few lower-limb complaints

Case 3 · Collateral circulation explains the image



Enlarged intercostal and internal mammary pathways bypass the aortic narrowing.

Intercostal enlargement can erode rib undersurfaces.

Collateral perfusion can reduce distal symptoms without removing the constriction.

Case 4 · Evaluate an associated cardiac lesion

A patient has coarctation and an additional abnormality of the aortic valve.

CASE INTERPRETATION

Link the observations to the site and direction of blood flow.

Aortic arch constriction



Abnormal aortic valve



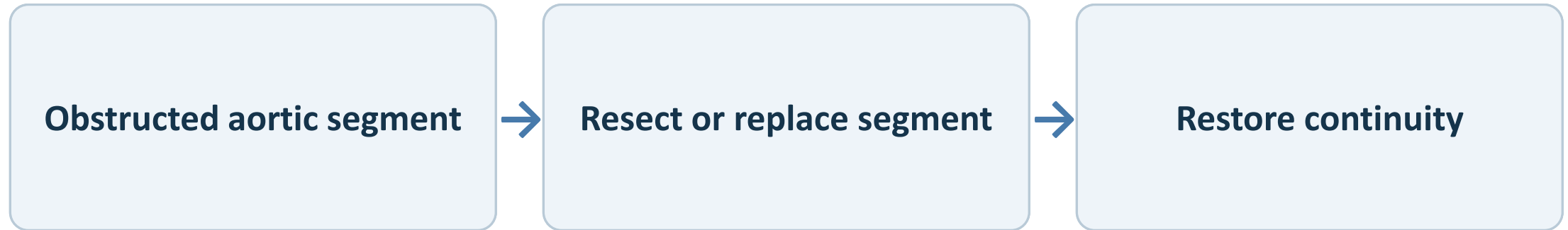
Two structural levels

Case 4 · Separate valve and vessel disease

Lesion	Principal location
Coarctation	Aortic vessel segment
Bicuspid aortic valve	Aortic valve cusps

Coarctation and bicuspid valve involve different anatomic sites.

Repair aims to remove the flow obstruction



For uncomplicated coarctation, resection with end-to-end anastomosis.

Replacement of an affected aortic segment by prosthetic graft is another described surgical approach.

The therapeutic target is the narrowed aortic segment.

Anatomic relief addresses the two pressure territories



Removing the fixed bottleneck restores a more direct pathway to the distal aorta.

Intervention aims to improve distal flow and reduce proximal outflow resistance.

These are physiologic interpretations of the surgical approaches described.

Outcomes must be qualified by complexity

Excellent results with surgical repair of uncomplicated coarctation.

This statement should not be extrapolated to all neonates or those with complex associated malformations.

The coexisting cardiac anatomy remains essential to prognosis.

Five findings that connect anatomy to presentation

Preductal PDA: Distal supply from pulmonary arterial blood.

Postductal ridge: Proximal hypertension and distal hypotension.

Right- versus left-ventricular hypertrophy follows the route of systemic support.

Internal mammary/intercostal collateral vessels explain rib notching.

Bicuspid aortic valve is an important coexisting defect.

Rapid mechanism check

1. Why can cyanosis predominate in the lower half of the body in preductal disease?

2. Why does a postductal ridge produce left-ventricular hypertrophy?

3. What causes rib notching when longstanding collateral vessels enlarge?

Primary source and scope

Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026).

Congenital heart disease: obstructive lesions and coarctation of the aorta.

Related reading: patent ductus arteriosus and the overview of congenital defects.