

Patent ductus arteriosus

Congenital cardiac shunt • fetal physiology • pulmonary vascular pathology

Anatomy → closure → shunt physiology → clinical consequences

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What this lecture will explain

- Identify the ductus and describe its fetal function
- Explain normal closure and causes of persistence
- Predict shunt direction, pulmonary pathology, and treatment logic

Anatomy



Closure



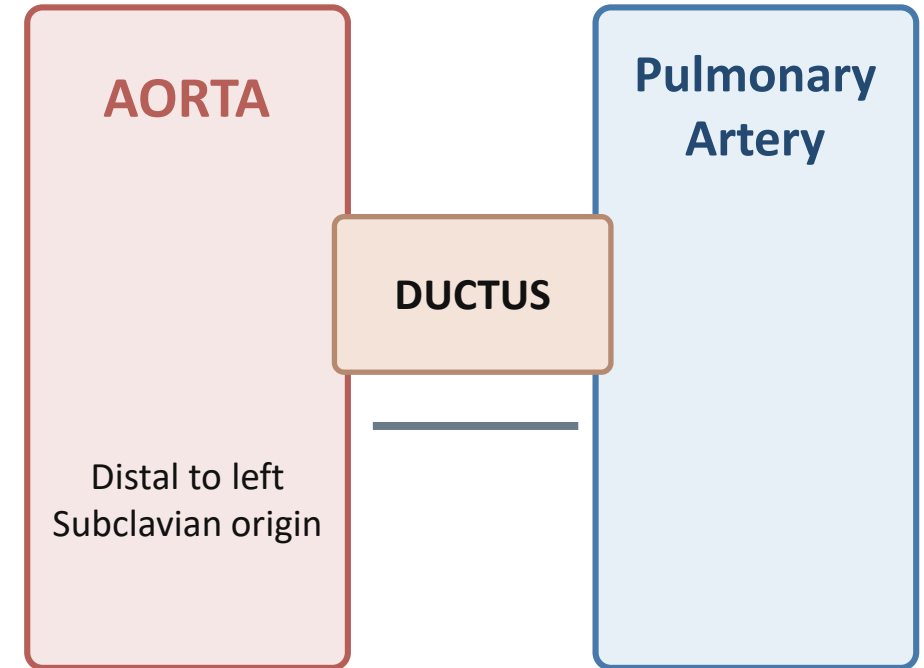
Shunting



Consequences

PDA is a persistent fetal arterial connection

- The ductus connects pulmonary artery to aorta
- Persistence after birth permits abnormal communication
- Unlike ASD and VSD, this is an extracardiac vascular shunt



Persistent arterial connection

PDA belongs to left-to-right shunt lesions

- The common group includes ASD, VSD, and PDA

ASD

VSD

PDA

- They increase flow into pulmonary circulation after birth

- Initial shunt direction does not produce cyanosis

Different locations of abnormal communication

CHD becomes apparent as circulation changes

- Structural anomalies can be tolerated before delivery
- The fetal-to-neonatal transition alters pressure gradients
- Clinical effects may therefore emerge after birth

Fetal circulation



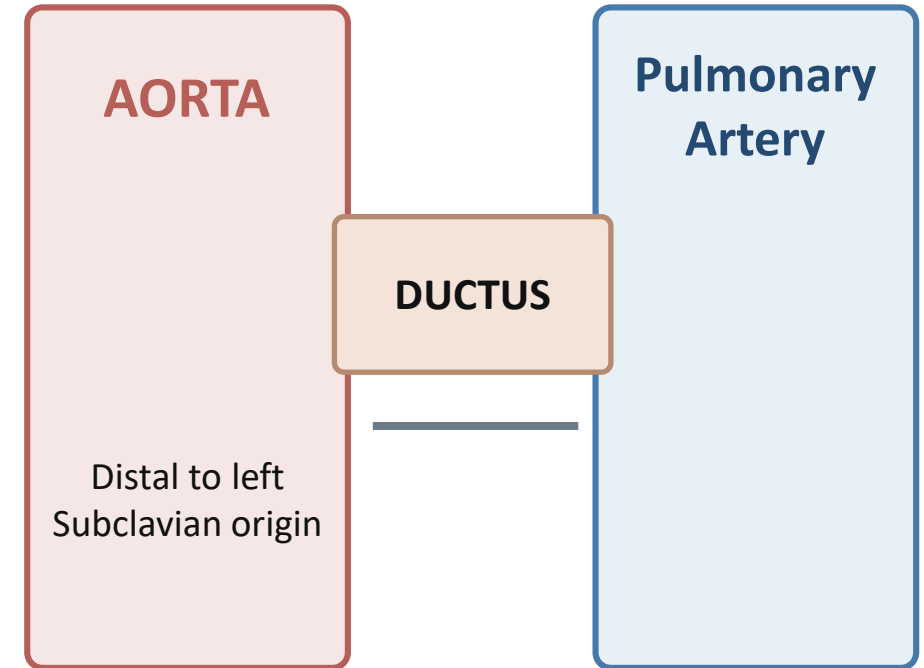
Birth transition



Postnatal hemodynamics

Ductus anatomy: A pulmonary-to-aortic channel

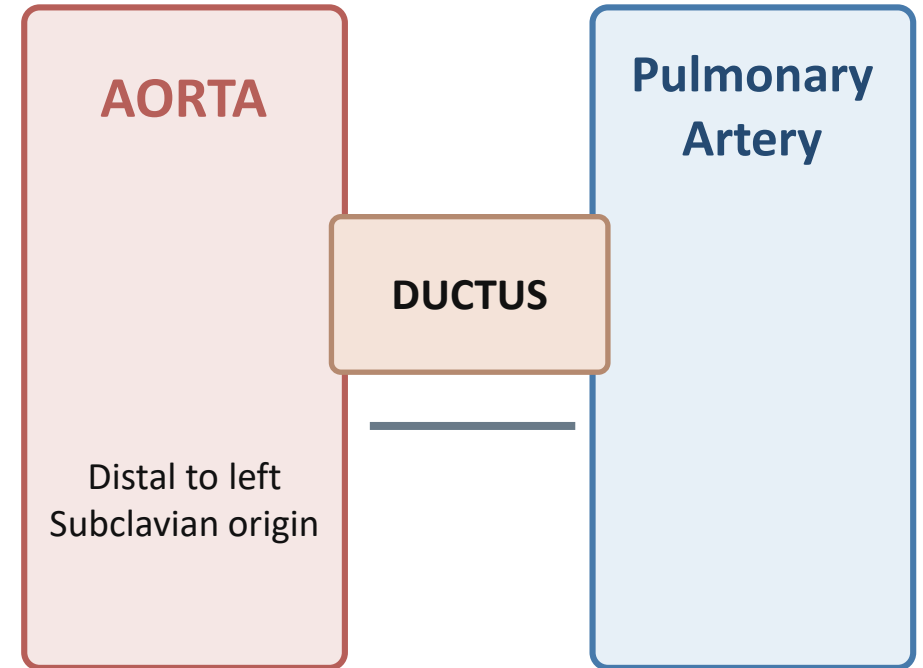
- The ductus originates from the pulmonary artery
- It joins the aorta on the descending side of the arch
- This route bypasses the fetal pulmonary vascular bed



Persistent arterial connection

The aortic insertion lies beyond the left subclavian

- Robbins locates the entry just distal to left subclavian origin
- The ductus therefore connects great vessels, not chambers
- The position matters when explaining flow direction



Persistent arterial connection

A patent ductus is normal before birth

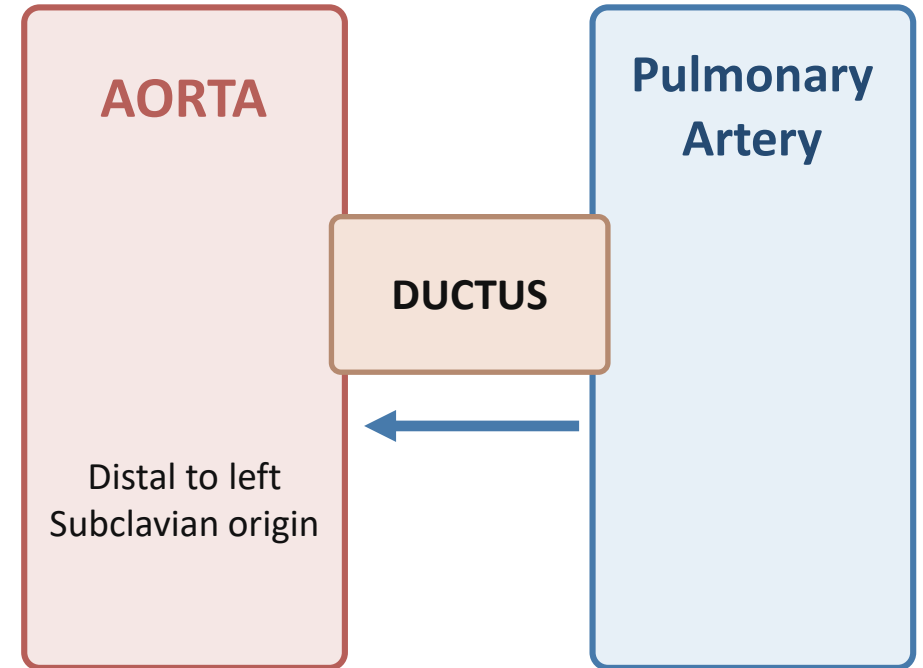
- Fetal lungs are not yet oxygenating the circulating blood
- The ductal pathway conveys blood away from the lungs
- Pulmonary arterial blood enters the aorta via the ductus

Pulmonary artery → ductus → aorta

Fetal physiology

Fetal shunting is pulmonary artery to aorta

- Pulmonary artery supplies the ductal inflow
- The aorta receives the bypassing blood
- The channel reduces dependence on fetal pulmonary flow



Fetal direction: Pulmonary → systemic

Postnatal survival changes ductal requirements

- Oxygenation rises after ventilation begins
- Pulmonary vascular resistance decreases
- Ductal patency is no longer required in normal anatomy

Ventilated lungs



Changed resistance



Ductal constriction

Increasing arterial oxygen favors constriction

- A rise in oxygenation is a closure stimulus
- This response follows the onset of extrauterine respiration
- Hypoxic conditions can delay the constrictive response

Higher arterial O₂



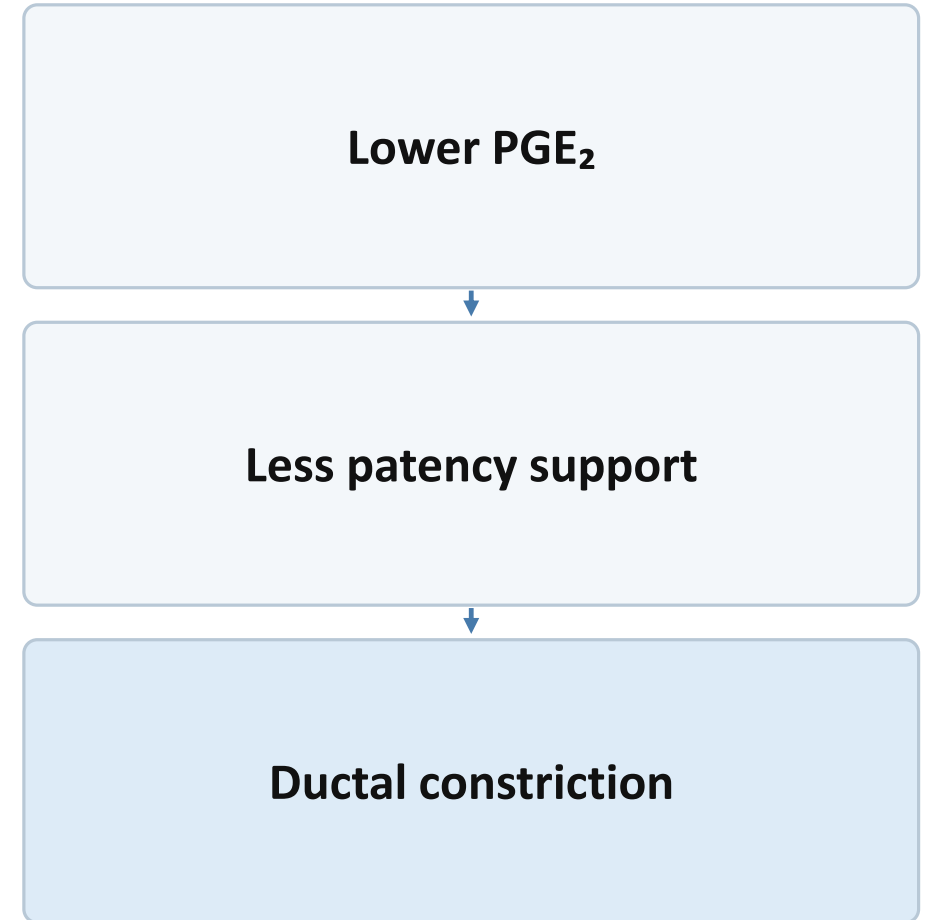
Ductal constriction



Functional closure

Declining prostaglandin E₂ favors closure

- Local prostaglandin E₂ levels fall after birth
- The fall contributes to ductal constriction
- Prostaglandin regulation explains therapeutic opportunities



Falling pulmonary resistance changes flow

- Birth reduces pulmonary vascular resistance
- The postnatal circulation favors aorta-to-pulmonary shunting if ductus remains open
- Direction is therefore not fixed across life

Fetus: Pulmonary → aorta

After birth: Aorta → pulmonary

Normal closure integrates multiple signals

- Higher arterial oxygenation
- Lower local prostaglandin E₂
- Reduced pulmonary vascular resistance accompanies transition

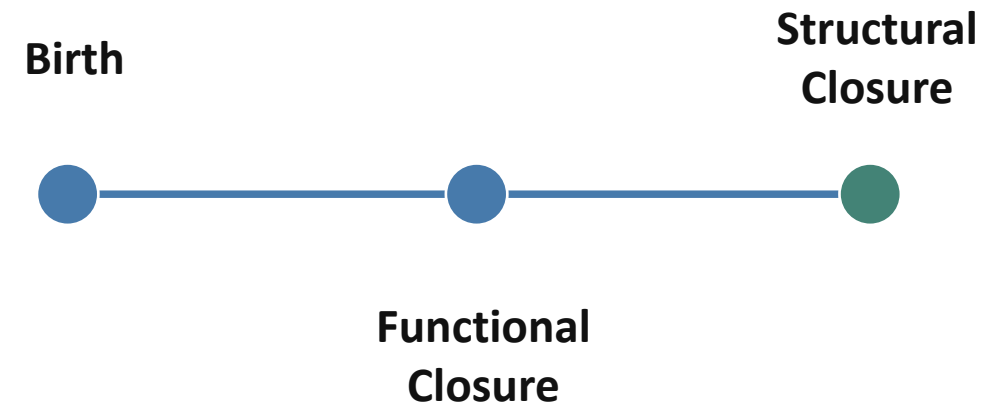
Higher O₂

Lower PGE₂

Lower PVR

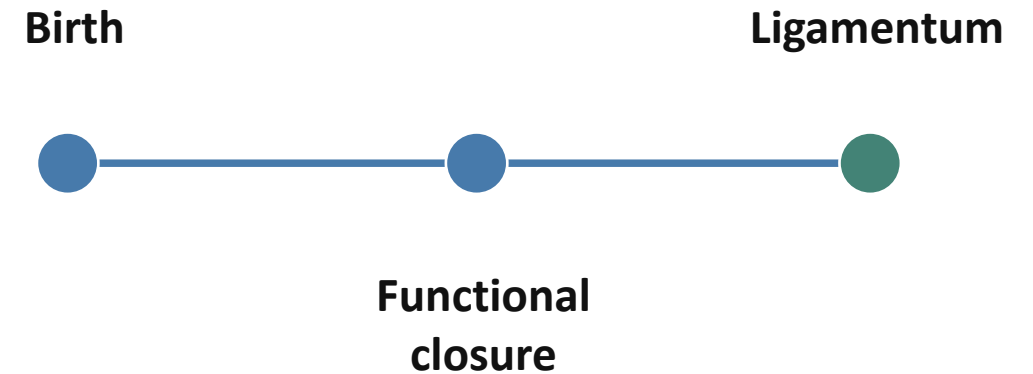
Functional closure usually occurs in 1–2 days

- In healthy term infants the ductus constricts rapidly
- The lumen becomes functionally closed within one or two days
- Functional closure precedes tissue-level obliteration



Anatomic obliteration takes longer

- The ductal structure undergoes permanent obliteration
- Complete closure develops over the first few postnatal months
- The resulting remnant is the ligamentum arteriosum



The ligamentum arteriosum marks closure

- A persistent fetal blood conduit becomes a fibrous remnant
- The remnant no longer carries blood between the vessels
- Structural closure differs from transient smooth-muscle constriction

Patent channel



Obliterated ductus



Ligamentum arteriosum

Functional and structural closure are distinct

- Early constriction stops flow
- Later obliteration removes the arterial channel
- Confusing these time frames obscures the meaning of “persistent”

Phase	Principal change
Functional	Constricted ductus; Blood flow ceases
Structural	Permanent obliteration; Fibrous remnant

PDA means closure has not been completed

- Patency retains a channel between great vessels
- Persistence may be delayed or absent altogether
- The defect must be interpreted in its physiological setting

Persistent lumen



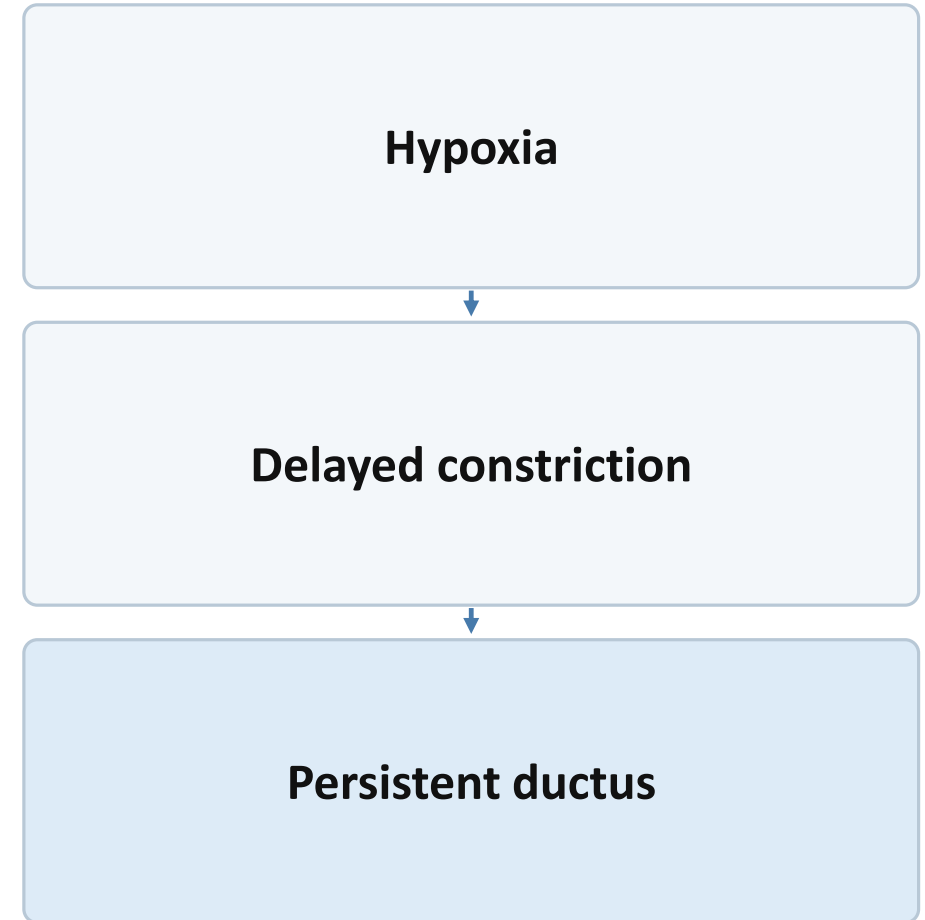
Arterial shunting



Clinical consequences

Hypoxia can prevent timely ductal closure

- Hypoxia with delayed or absent closure
- Respiratory distress is one relevant clinical context
- Other heart disease can also sustain hypoxemia



Other defects can sustain ductal patency

- Coexisting congenital lesions alter pressure and flow
- VSDs that elevate pulmonary pressures
- An apparently simple PDA may coexist with a complex circulation

VSD



Higher pulmonary pressure



Delayed closure

Genetic context: Char syndrome

- The congenital heart disease gene table associates PDA with Char syndrome
- TFAP2B is the listed gene
- The gene product is a transcription factor

Char syndrome

TFAP2B

Transcription factor

Genetic context: CHARGE syndrome

- PDA can occur among multiple cardiac malformations
- CHD7 for CHARGE syndrome
- The table identifies CHD7 as a helicase-binding protein

CHARGE syndrome

CHD7

PDA among cardiac features

Syndromic associations do not explain every PDA

- Many congenital anomalies occur without identified mutations
- One genotype may be associated with several phenotypes
- A syndromic association is not a universal cause of PDA

Genetic susceptibility



Variable expression



Clinical phenotype

PDA contributes about 7% of CHD cases

- The Robbins congenital malformation table lists PDA at 7%
- It is one of the common congenital shunt abnormalities
- This is a proportion among classified CHD cases, not all newborns

PDA: 7% of CHD

Table 12.2

Denominator matters

Most PDAs are isolated lesions

- About 90% as isolated defects
- Isolation does not imply a uniformly benign course
- Large isolated channels can still damage pulmonary vessels

90% isolated

Size matters

Pulmonary vascular risk

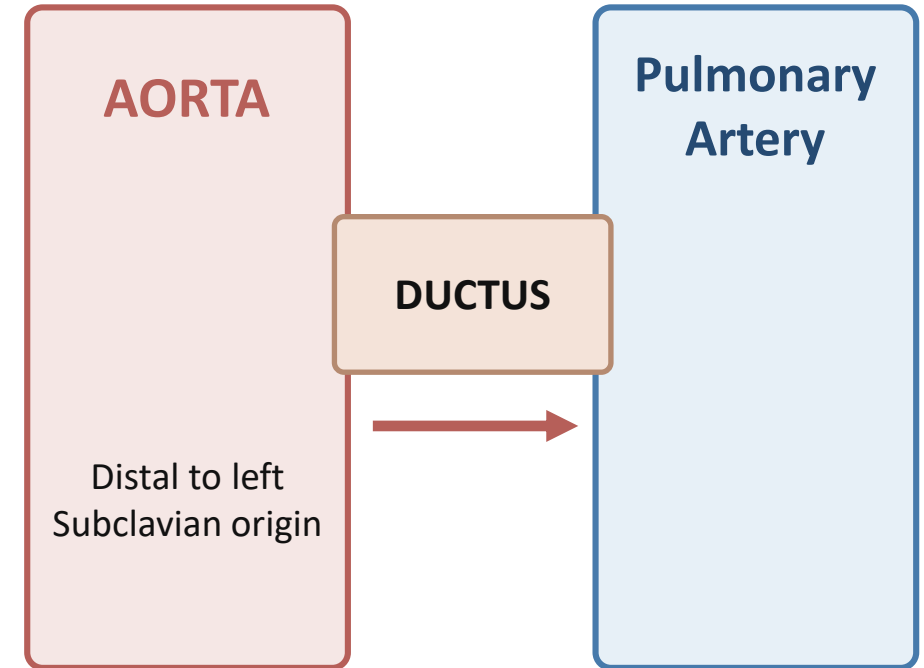
The direction of flow reverses after birth

- Fetal flow: Pulmonary artery to aorta
- Uncomplicated postnatal PDA: Aorta to pulmonary artery
- Late pulmonary vascular disease may reverse it again

Stage	Typical ductal flow
Fetal	Pulmonary artery → aorta
Postnatal PDA	Aorta → pulmonary artery
Advanced disease	Pulmonary artery → aorta

Postnatal PDA follows the pressure gradient

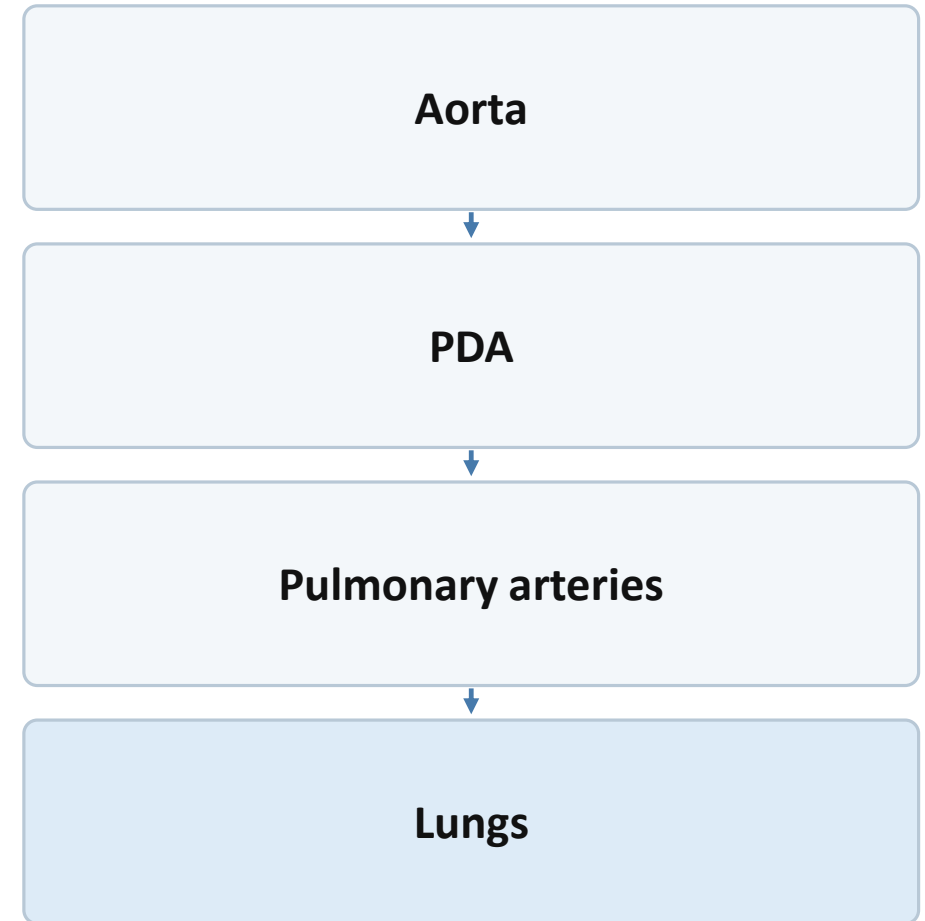
- Blood moves from the systemic to the pulmonary circuit
- This is the initial left-to-right shunt pattern
- Shunt direction reflects relative vascular pressures



After birth: Systemic → pulmonary

PDA connects systemic and pulmonary flow

- Aortic blood re-enters the pulmonary arterial circulation
- Pulmonary flow is increased without an intracardiac defect
- The lungs must accommodate recirculated blood



A small PDA may have little physiological impact

- Ductal diameter influences shunt volume
- A narrow channel may not impair growth or development
- Anatomic patency does not always mean severe disease

Small channel

Limited shunt

Minimal clinical effect

A large PDA may create substantial overload

- A wide duct permits more communication
- Additional pulmonary blood volume and pressure impose stress
- Severity is not determined by the term “PDA” alone

Wide channel

Major shunt

Pulmonary overload

Cardiovascular status modifies PDA severity

- The same opening can behave differently under different hemodynamics
- Pulmonary vascular conditions affect the gradient
- Associated malformations must be interpreted together

Ductal diameter



Pulmonary resistance



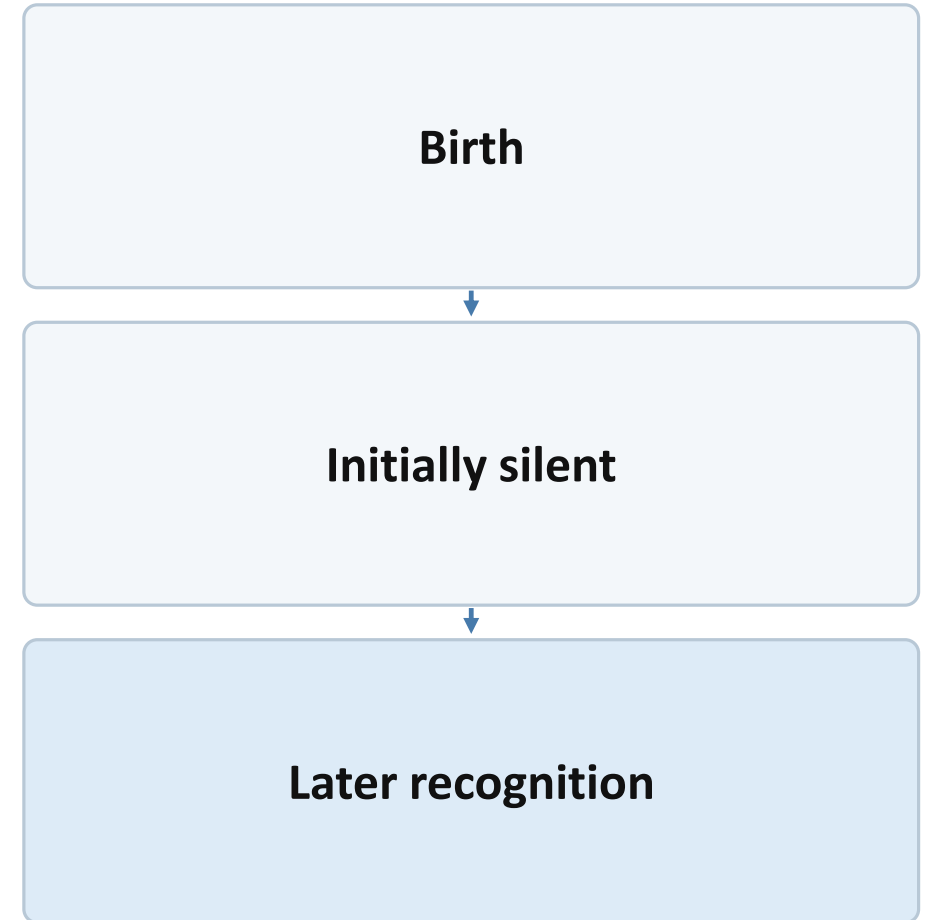
Associated disease



Clinical impact

PDA is commonly asymptomatic at birth

- Robbins explicitly describes initial asymptomatic presentation
- Early tolerance does not exclude a persistent shunt
- Clinical findings can become evident as physiology evolves



A characteristic finding is a continuous murmur

- PDA classically produces a harsh, machinery-like murmur
- The murmur is continuous rather than limited to a single cardiac phase
- Auscultation must be interpreted with the whole hemodynamic picture

Continuous

Harsh

Machinery-like

Continuous flow explains the murmur pattern

- A persistent arterial communication allows ongoing flow
- In a typical postnatal shunt, aortic-to-pulmonary flow is sustained
- The persistent flow supports a continuous auscultatory sound

Patent conduit



Sustained flow



Continuous murmur

Initial left-to-right PDA is not cyanotic

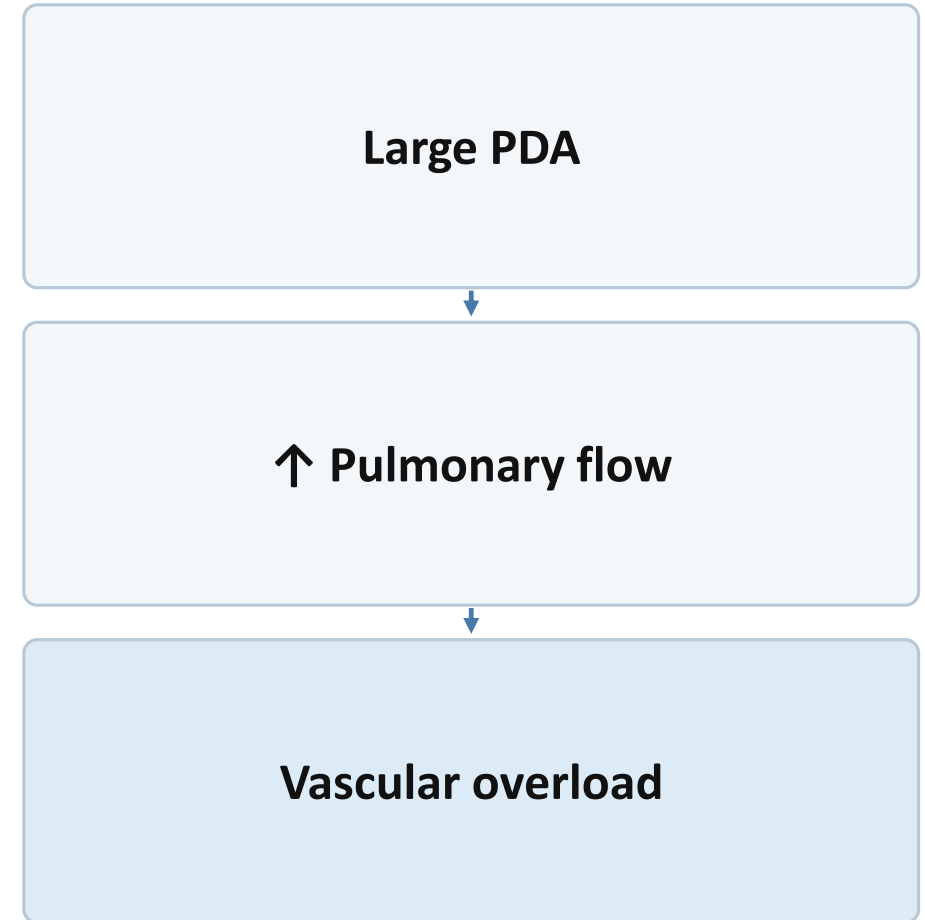
- Oxygenated systemic blood travels toward the lungs
- Systemic arterial blood is not diluted by venous blood initially
- Acyanotic presentation does not exclude a major shunt

Aorta → pulmonary artery

No initial cyanosis

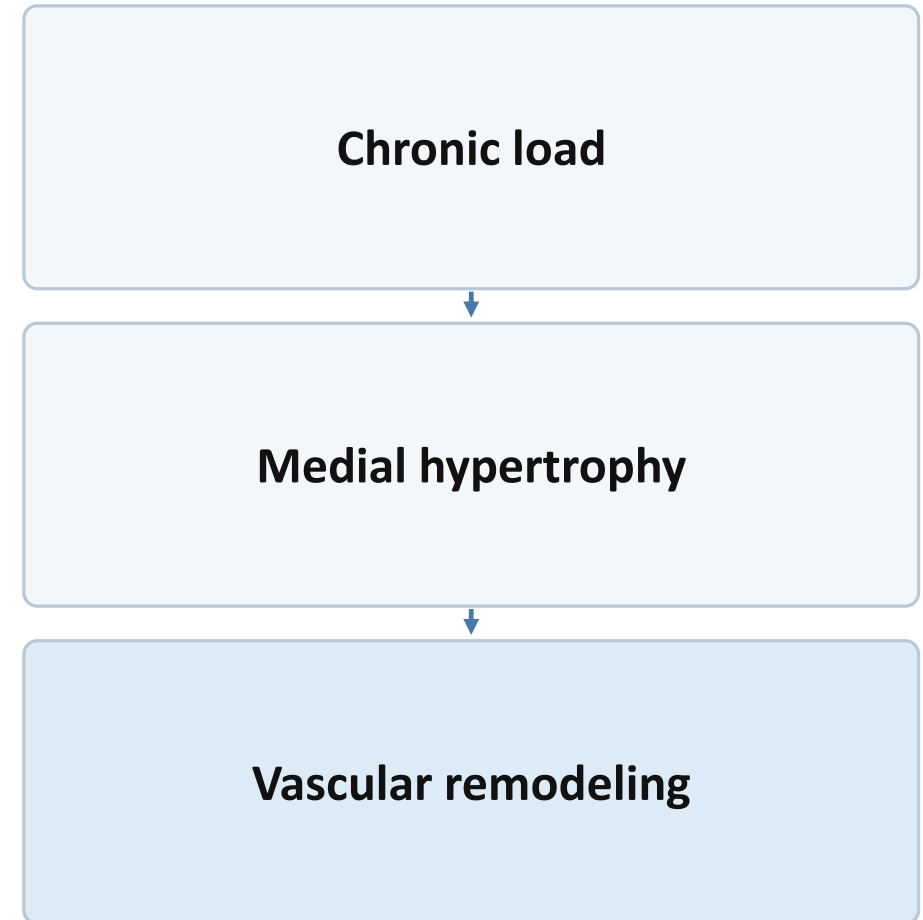
The first major risk is pulmonary overcirculation

- Large left-to-right shunts elevate pulmonary blood flow
- Both volume and pressure loading may increase
- Downstream pulmonary vessels adapt to the increased load



Pulmonary arteries respond to chronic overload

- Pulmonary muscular arteries initially remodel
- Medial hypertrophy is an adaptive change
- The response occurs within a normally low-resistance vascular bed



Pulmonary vasoconstriction is an early response

- Narrowing helps regulate distal capillary pressure
- It simultaneously increases pulmonary vascular resistance
- Persistent stimulus can promote further vascular damage

Elevated flow/pressure



Vasoconstriction



↑ Resistance

Obstructive intimal lesions mark progression

- Prolonged abnormal pressure/flow promotes arterial injury
- Irreversible intimal obstruction can develop
- The vascular pathology may persist even if the initial communication is addressed

Persistent overload



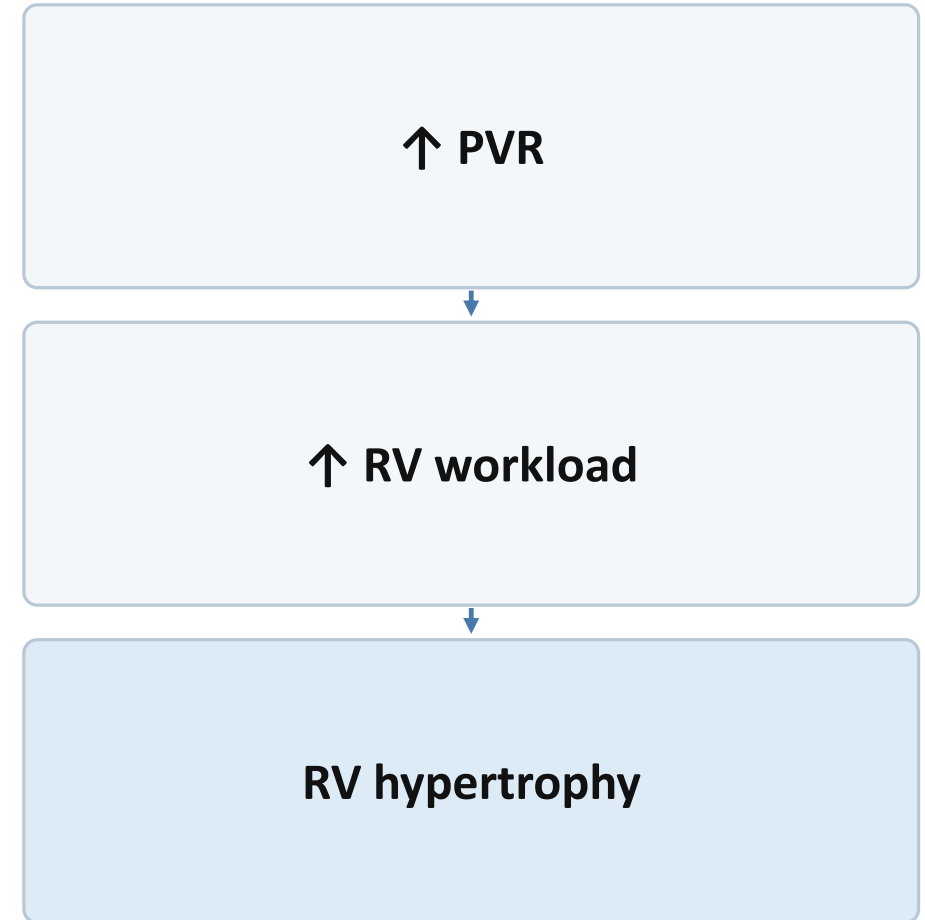
Intimal obstruction



Irreversible disease

The right ventricle responds to rising resistance

- Progressive pulmonary resistance increases right ventricular workload
- The right ventricle undergoes hypertrophy
- This ventricular adaptation reflects pulmonary vascular disease



Pulmonary resistance may approach systemic levels

- The pressure advantage sustaining left-to-right flow diminishes
- Shunt volume and direction can change over time
- Advanced disease may create right-to-left flow

↑ Pulmonary resistance



Gradient narrows



Flow reverses

Eisenmenger physiology is late shunt reversal

- A prior left-to-right shunt becomes right-to-left
- Irreversible obstructive pulmonary vascular disease is the driver
- The resulting systemic desaturation is clinically consequential

Longstanding PDA



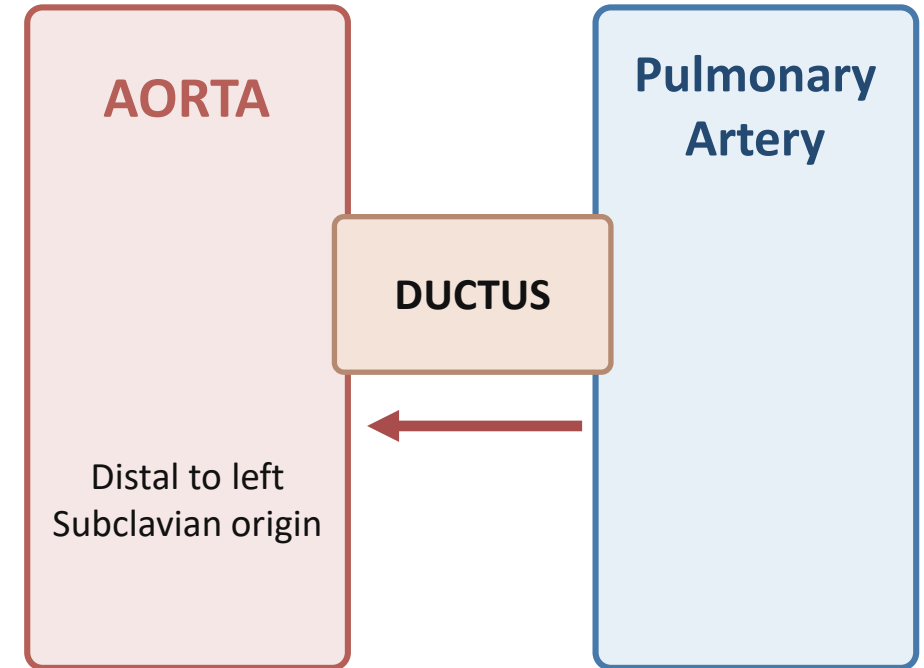
Pulmonary vascular disease



Right-to-left reversal

Right-to-left flow introduces venous blood systemically

- Pulmonary arterial blood can pass into the aorta
- This bypasses oxygenation through the lungs
- Cyanosis becomes possible after reversal



Late reversal: Pulmonary → systemic

Cyanosis is a late warning in uncomplicated PDA

- No cyanosis is expected with the initial left-to-right shunt
- Cyanosis after reversal indicates advanced pulmonary vascular pathology
- The timing and mechanism differ from primary cyanotic CHD

Early PDA	Advanced reversal
Left → right	Right → left
No initial cyanosis	Cyanosis can develop
Pulmonary overload	Obstructive vascular disease

Pulmonary vascular irreversibility changes management

- Longstanding obstruction may make the defect irreparable
- This is why large shunts require early attention
- A simple “close every duct” rule is unsafe in advanced physiology

Reversible overload



Obstructive disease



Irreparable shunt state

Three clinical trajectories should be separated

- Small PDA: Little or no developmental impact
- Large PDA: Pulmonary flow and pressure overload
- Longstanding major shunt: Reversal with cyanotic consequences

Narrow	Large	Advanced
Minimal symptoms	Overcirculation	Shunt reversal
Limited impact	Pulmonary remodeling	Cyanosis

ASD, VSD, and PDA share but differ

- Each can begin with a left-to-right shunt
- ASD typically adds right ventricular/pulmonary volume
- VSD and PDA can elevate both pulmonary flow and pressure

Lesion	Principal communication	Physiology
ASD	Between atria	RV/pulmonary volume
VSD	Between ventricles	Pulmonary flow + pressure
PDA	Between great vessels	Pulmonary flow + pressure

PDA is not the same as a septal defect

- The ductus is a connection between great arteries
- An ASD or VSD is within the cardiac septa
- Physical location changes anatomical interpretation

ASD: Atria

**VSD:
Ventricles**

**PDA: Great
vessels**

**Different locations of abnormal
communication**

Clinical evaluation starts with physiology

- Ask whether the infant is symptomatic or thriving
- Determine whether a characteristic continuous murmur is present
- Assess whether pulmonary overload or late cyanosis is suspected

Presentation



Auscultation



Hemodynamic interpretation

Echocardiography provides structural context

- The parent CHD discussion describes echocardiographic detection of defects
- Imaging can evaluate a suspected congenital communication
- Interpret the visible anatomy together with shunt physiology

Suspected CHD



Echocardiography



Anatomy + physiology

Early closure addresses the cause of overload

- An isolated PDA is generally closed as early as feasible
- Removing the communication stops abnormal recirculation
- The objective is to prevent progressive pulmonary injury

Persistent PDA



Eliminate shunt



Prevent vascular injury

Prostaglandin synthesis inhibition can close PDA

- Prostaglandin synthesis inhibitors as therapy
- The intervention exploits the role of prostaglandins in patency
- Selection depends on individual clinical circumstances

Reduce prostaglandin support



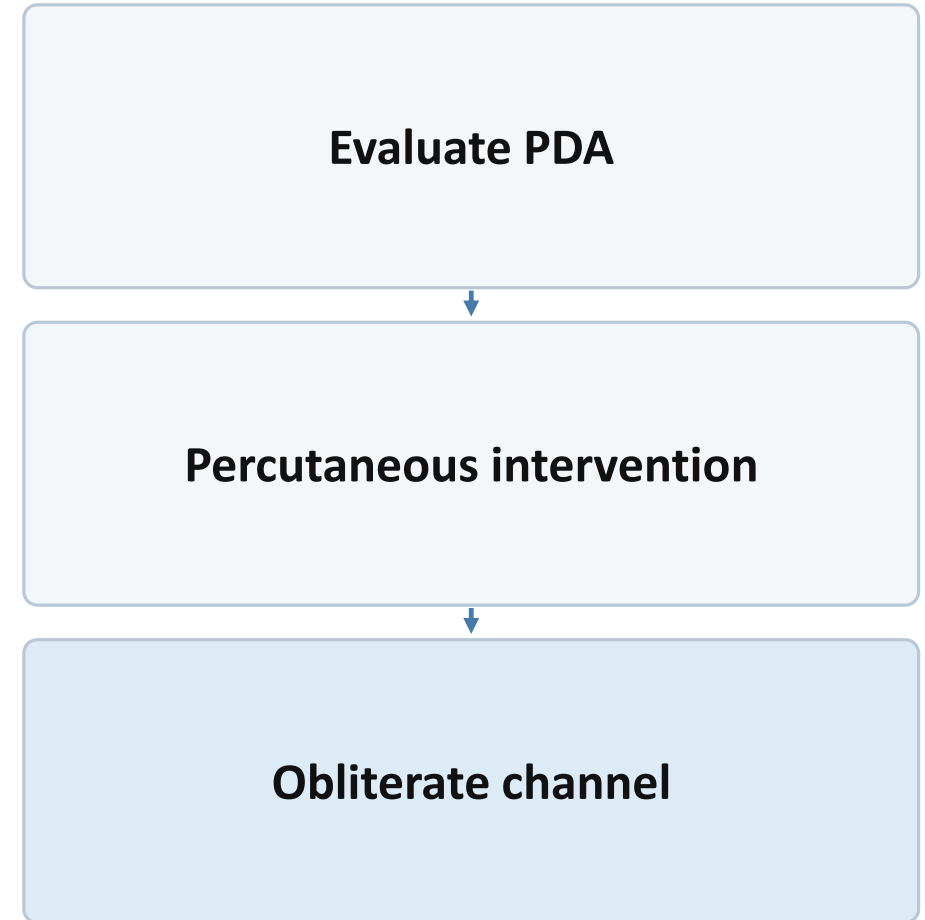
Ductal constriction



Closure

Percutaneous closure offers a mechanical option

- Interventional closure is a recognized strategy
- It interrupts communication between the great vessels
- This option when needed after medical measures



Surgery is another route to closure

- Surgical treatment can interrupt ductal blood flow
- Its use depends on lesion and patient circumstances
- Treatment should match anatomy and clinical risk

Patent communication



Surgical intervention



Stop shunting

Treatment decisions cannot ignore pulmonary disease

- A large patent shunt can progress to irreversible lesions
- Shunt reversal signals that simple closure reasoning has changed
- Intervention is most useful before fixed severe vascular disease

Before vascular injury	After fixed vascular injury
Aimed at prevention	Risk requires reassessment
Early closure feasible	Simple closure not assumed

Some congenital lesions need an open ductus

- A duct may serve as the only route to vital blood flow
- This occurs in lesions obstructing systemic or pulmonary output
- Ductal preservation can be lifesaving

Outflow atresia



Duct-dependent perfusion



Maintain patency

Aortic valve atresia: Ductus supports systemic flow

- Normal ventricular-to-aortic egress is obstructed
- Ductal connection can supply the systemic circulation
- Closing that channel may eliminate essential perfusion

Pulmonary artery → PDA → aorta

Systemic flow depends on duct

Pulmonary valve atresia: Ductus supports lung flow

- Normal right ventricular-to-pulmonary egress is obstructed
- The ductal channel can supply the pulmonary circulation
- Maintaining patency preserves an essential flow route

Aorta → PDA → pulmonary artery

Pulmonary flow depends on duct

Prostaglandin E₁ can maintain ductal patency

- Robbins explicitly describes PGE₁ administration
- The goal in duct-dependent lesions is to keep the channel open
- This is the opposite of treating an isolated large PDA

Duct-dependent defect



Administer PGE₁



Preserve perfusion

The treatment paradox is physiology-dependent

- Isolated PDA with harmful shunting: Aim for closure
- Outflow obstruction with duct-dependent supply: Preserve patency
- The identical vessel may be pathologic or lifesaving

Isolated PDA	Duct-dependent CHD
Abnormal recirculation	Vital bypass pathway
Close when appropriate	Maintain patency with PGE ₁

Case: Asymptomatic infant with small PDA

- A narrow PDA is detected without growth impairment
- Explain why observation of mild impact does not negate anatomy
- Identify which change would raise concern about future risk

CLINICAL REASONING

Small ductal diameter

No growth effect

Evaluate shunt burden

Case: Large PDA with changing physiology

- A persistent large shunt exposes pulmonary arteries to overload
- Name the expected arterial and ventricular adaptations
- Explain why intervention timing matters

CLINICAL REASONING

Pulmonary overcirculation

Arterial remodeling

Prevent irreversible changes

Case: Cyanosis after a longstanding shunt

- A formerly acyanotic patient develops cyanosis
- Infer what happened to pulmonary vascular resistance
- Explain why shunt direction may have reversed

CLINICAL REASONING

↑ Pulmonary resistance

Right-to-left reversal

Eisenmenger physiology

Case: Duct-dependent outflow obstruction

- An infant has pulmonary valve atresia
- The ductus may be essential for pulmonary blood supply
- Determine why routine ductal closure would be dangerous

CLINICAL REASONING

Atretic pulmonary outflow

Duct supplies lungs

Preserve PDA

Integrating PDA pathogenesis

- A fetal conduit persists after the normal closure window
- Left-to-right flow causes pulmonary volume and pressure loading
- Chronic vascular disease can reverse shunting and cause cyanosis

Persistent conduit



Pulmonary overload



Vascular obstruction



Reversal

Four questions determine the clinical meaning

- What is the anatomical connection and direction of flow?
- How large is the duct and what is the pulmonary vascular state?
- Is the communication harmful or supplying essential flow?
- Would closure prevent injury or interrupt perfusion?

Anatomy



Magnitude



Vascular disease



Duct dependence

Primary textbook reference

- Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th edition (2026).
- Congenital heart disease: left-to-right shunts and patent ductus arteriosus.