

Transposition of the Great Arteries

Anatomical discordance • parallel circulation • mixing • systemic ventricular adaptation

Congenital cardiac pathology

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Learning objectives

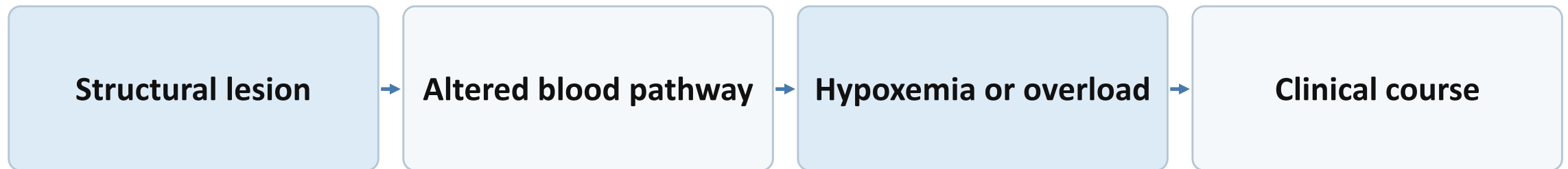
- Trace normal and discordant atrioventricular and ventriculoarterial connections.
- Explain why d-TGA forms parallel circulations and why mixing permits survival.
- Relate shunt closure and systemic ventricular workload to clinical outcomes.
- Differentiate d-TGA from congenitally corrected l-TGA and key congenital comparators.

Congenital heart disease: A structural framework

Congenital heart disease (CHD) changes chamber, valve, or great-vessel connections.

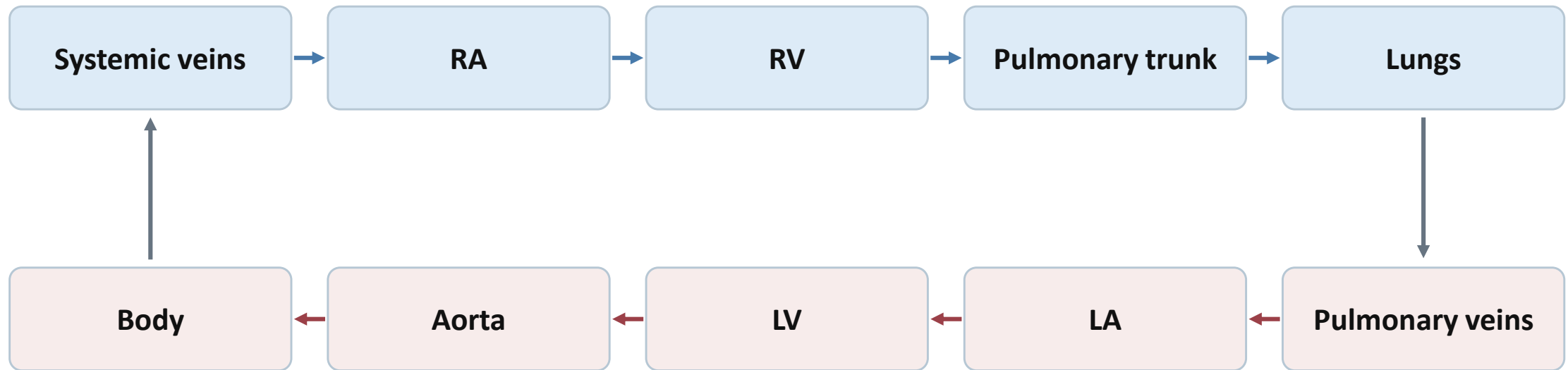
Robbins organizes functional disturbances as shunts, outflow obstruction, or their combination.

TGA is distinguished by the destination of ventricular outflow, not simply a hole between chambers.



Normal circulation depends on two ventricular outflows

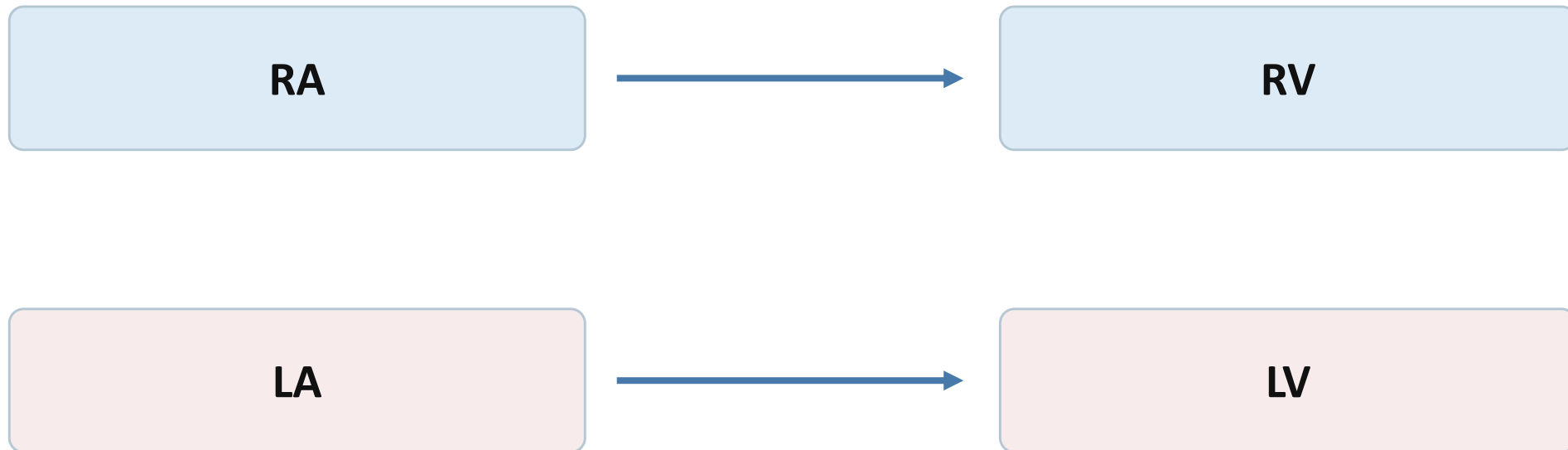
Right atrium → right ventricle → pulmonary artery → lungs.



Single series circuit: Systemic return → lungs → pulmonary venous return → aorta → tissues

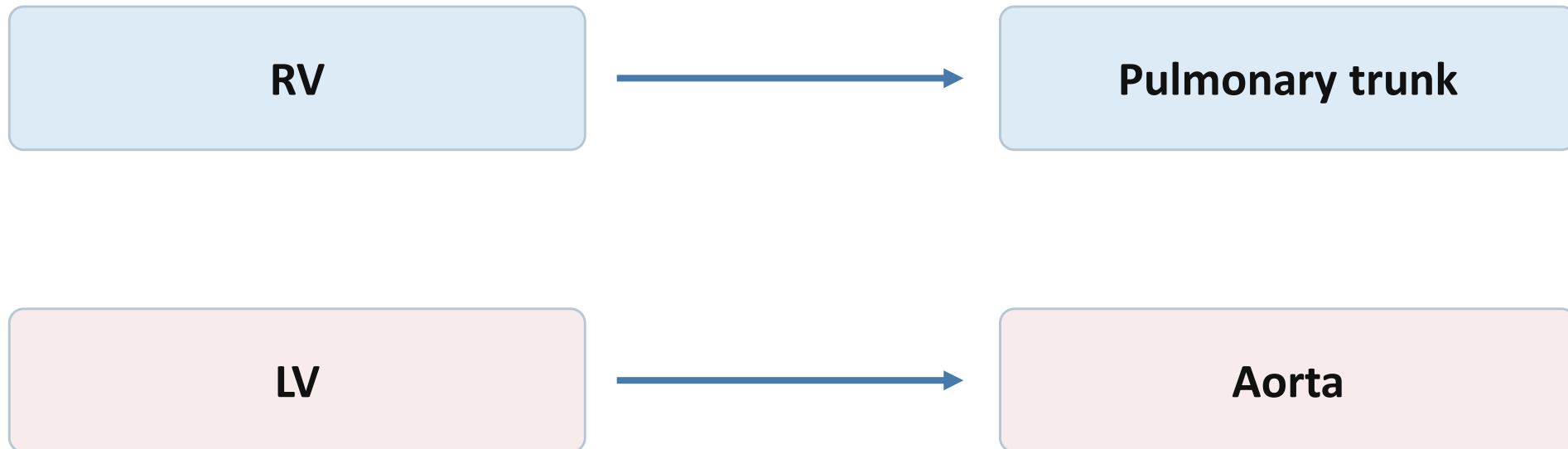
Atrioventricular concordance: Atrium to expected ventricle

Right atrium communicates with the morphologic right ventricle.



Ventriculoarterial concordance: Expected arterial origins

Morphologic right ventricle gives rise to pulmonary trunk.



Discordance describes connections, not chamber position

AV discordance: An atrium connects to a ventricle of unexpected morphology.

Level	Concordant	Discordant
Atrioventricular	RA → RV; LA → LV	RA → morphologic LV; LA → morphologic RV
Ventriculoarterial	RV → PT; LV → aorta	RV → aorta; LV → PT

TGA is a ventriculoarterial connection abnormality

Transposition of the great arteries produces ventriculoarterial discordance.

Variant	AV connection	VA connection
d-TGA	Concordant	Discordant
l-TGA	Discordant	Discordant

How common is TGA in the Robbins CHD table?

Robbins Table 12.2 lists TGA at 388 per million live births.

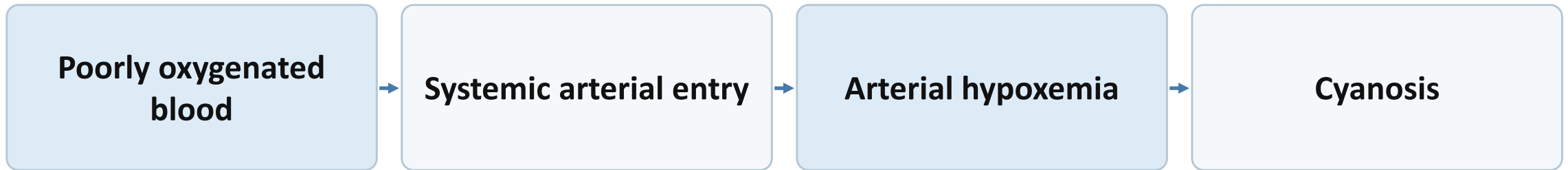
Malformation	Per million live births	Share in source table
Ventricular septal defect	4,482	42%
Tetralogy of Fallot	577	5%
Transposition of great arteries	388	4%

Right-to-left shunt: Why cyanosis develops

Systemic venous blood can bypass pulmonary oxygenation and enter the systemic arterial supply.

Arterial hypoxemia produces cyanosis of skin and mucous membranes.

D-TGA creates a different arrangement—parallel circuits—which likewise threatens systemic oxygen delivery.



A shunt and a transposition are not synonyms

Shunt: An abnormal communication permitting flow between compartments.

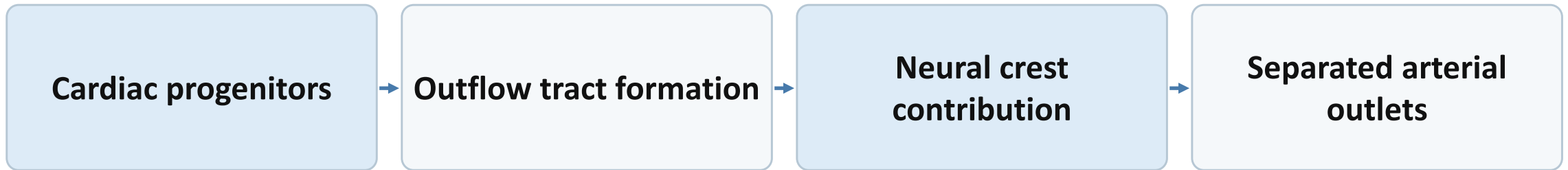
Feature	Shunt	d-TGA
Primary anatomical problem	Communication across compartments	Discordant great-artery origins
Hemodynamic consequence	Flow along pressure gradient	Parallel systemic/pulmonary loops
Role of interconnection	Part of defect	Permits essential mixing

Cardiac outflow development requires coordinated septation

Second-heart-field cells contribute to the cardiac outflow tract and right ventricle.

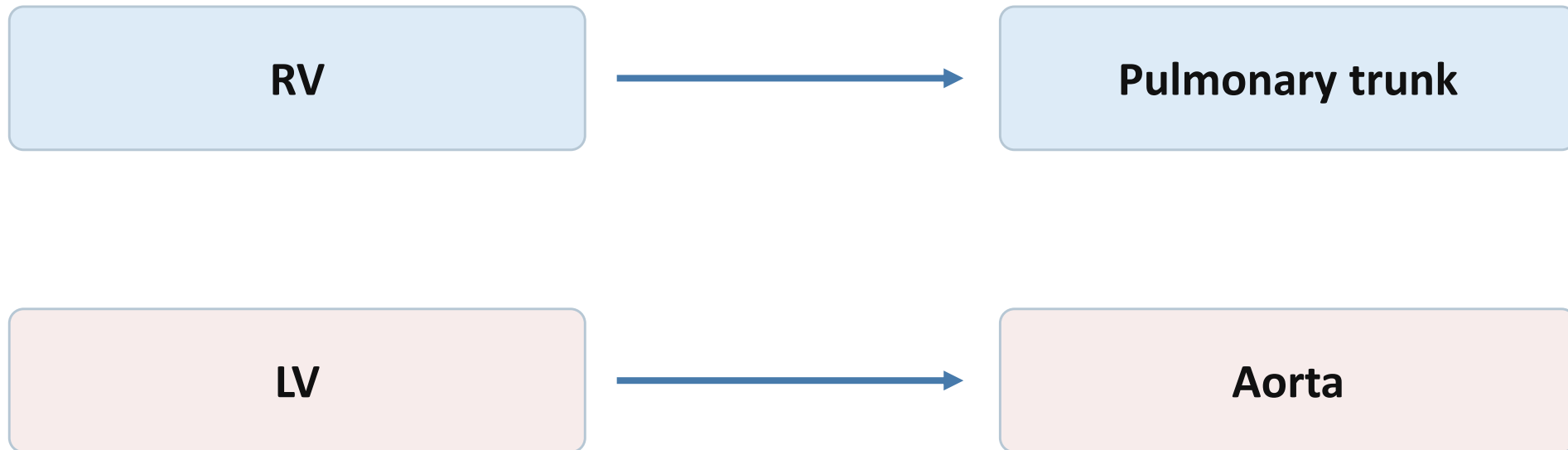
Neural crest–derived cells participate in aortic and pulmonary outflow tract septation.

This developmental context localizes d-TGA to great-vessel partitioning.



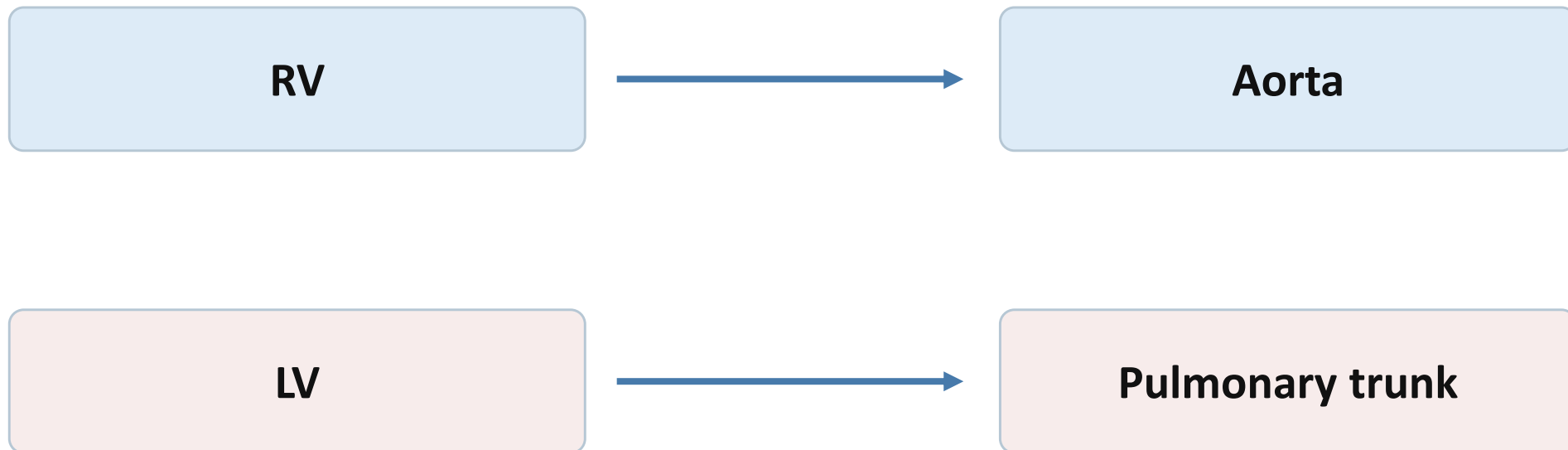
Normal separation depends on spiraling septa

Formation of spiraling truncal and aortopulmonary septa.



D-TGA: Abnormal outflow septation changes arterial alignment

Abnormal development of the spiraling outflow septa is the textbook mechanism.



Embryologic explanation is not a genetic diagnosis

The d-TGA section attributes the defect to abnormal outflow septation.

Supported by the source

- Disordered spiraling outflow septation
- Resultant VA discordance

Not specified for TGA

- A disease-defining mutation
- A TGA-specific inheritance probability

From arterial malposition to circulation in parallel

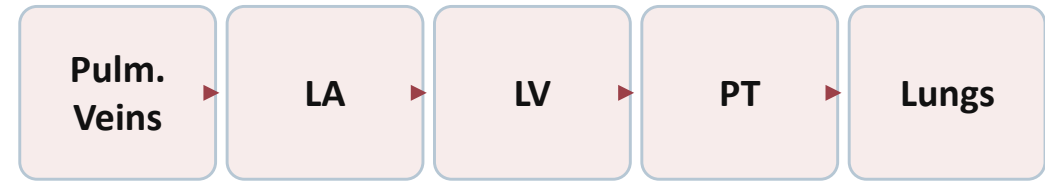
Aorta arising from RV returns systemic venous blood directly to systemic tissues.

Systemic loop — poorly oxygenated return



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Pulmonary loop — oxygenated venous return

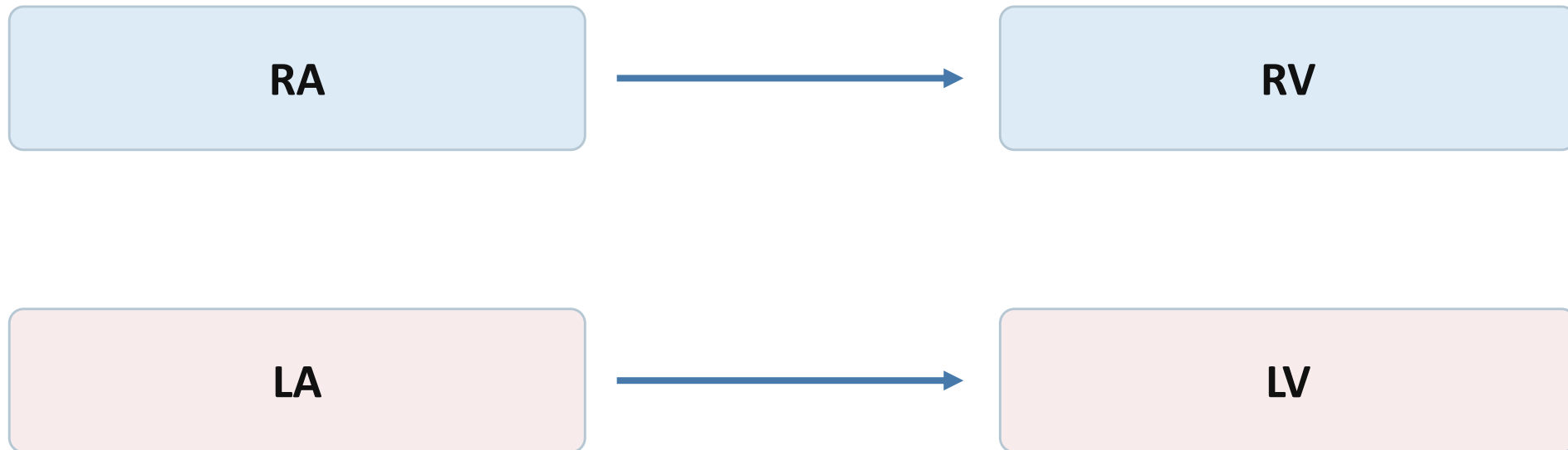


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Parallel-loop schematic • PT = pulmonary trunk; Dashed arrows indicate recirculation

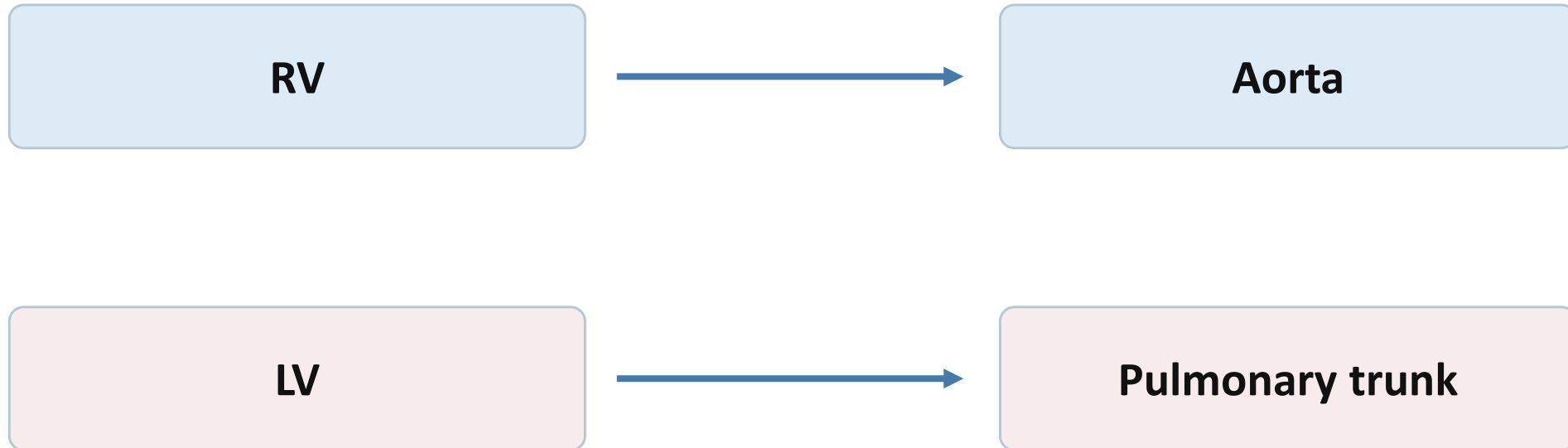
D-TGA preserves both atrium–ventricle connections

Right atrium still enters morphologic right ventricle.



D-TGA reverses the expected great-artery outlets

Aorta originates from the morphologic right ventricle.



Trace the systemic venous pathway in d-TGA

Systemic veins deliver poorly oxygenated blood to RA.

RA → RV remains anatomically concordant.

RV ejects into aorta: Blood can return to body without passage through lungs.



Trace the pulmonary venous pathway in d-TGA

Oxygenated pulmonary venous blood enters LA.

LA → LV remains anatomically concordant.

LV ejects into the pulmonary trunk, sending blood toward lungs again.



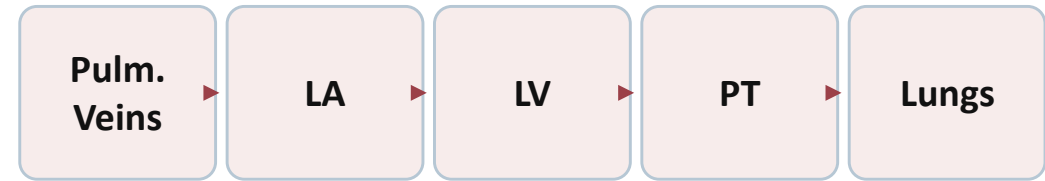
D-TGA produces two circulations in parallel

Systemic circuit loops through RA–RV–aorta–body.

Systemic loop — poorly oxygenated return



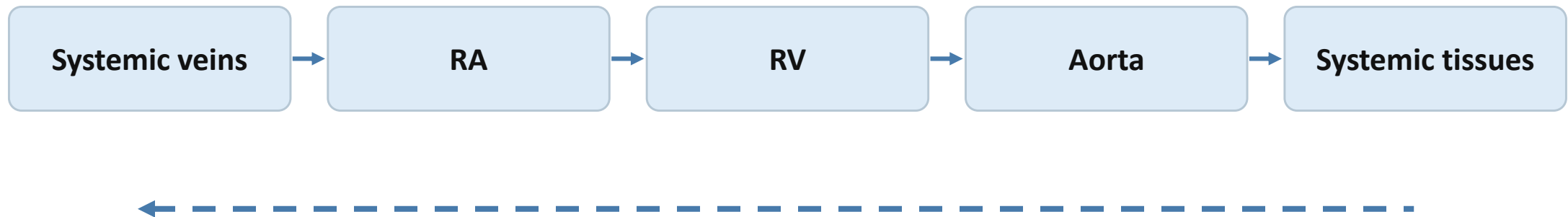
Pulmonary loop — oxygenated venous return



Parallel-loop schematic • PT = pulmonary trunk; Dashed arrows indicate recirculation

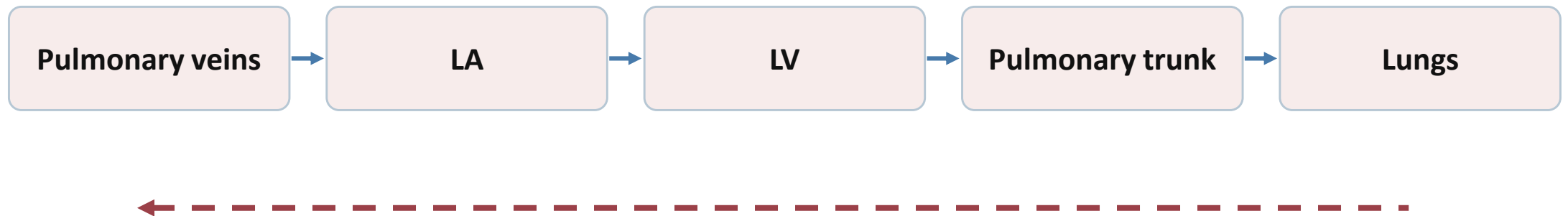
The systemic circuit recirculates venous blood

Oxygen-poor systemic return enters RA and RV.



The pulmonary circuit recirculates oxygenated blood

Pulmonary veins deliver oxygenated blood to LA and LV.



Why the two circuits cannot independently sustain life

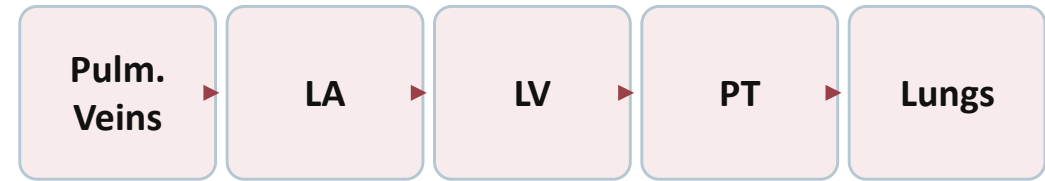
Anatomical continuity from lungs to systemic aorta is absent in uncommunicating d-TGA.

Systemic loop — poorly oxygenated return



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Pulmonary loop — oxygenated venous return



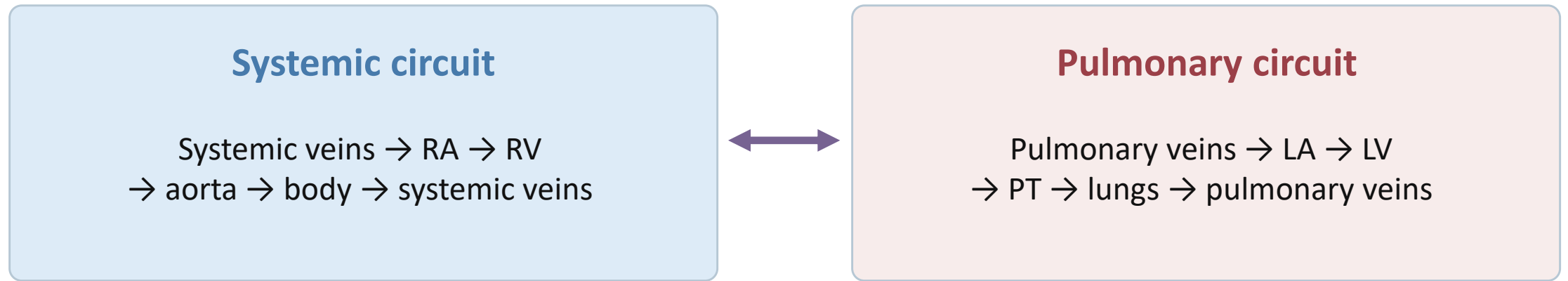
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Parallel-loop schematic • PT = pulmonary trunk; Dashed arrows indicate recirculation

Mixing is the determinant of immediate viability

Robbins explicitly relates survival to the degree of mixing between circuits.

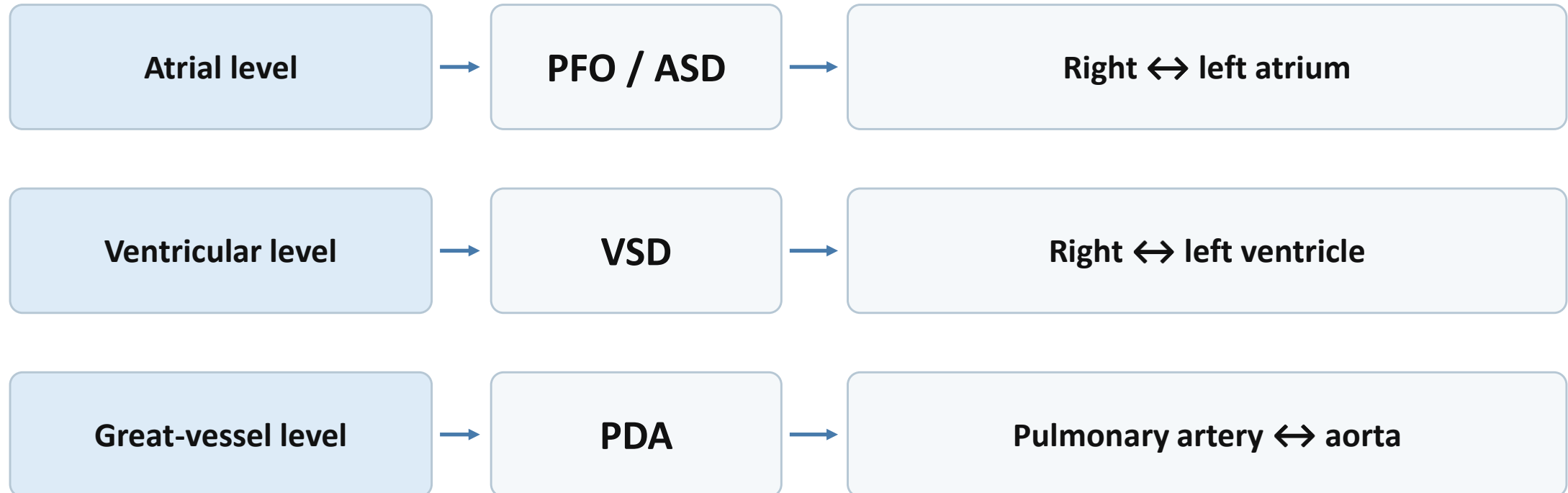
Communication between loops



Schematic mixing between circuits; The label identifies the communicating anatomical level.

Three important sites for communicating flow

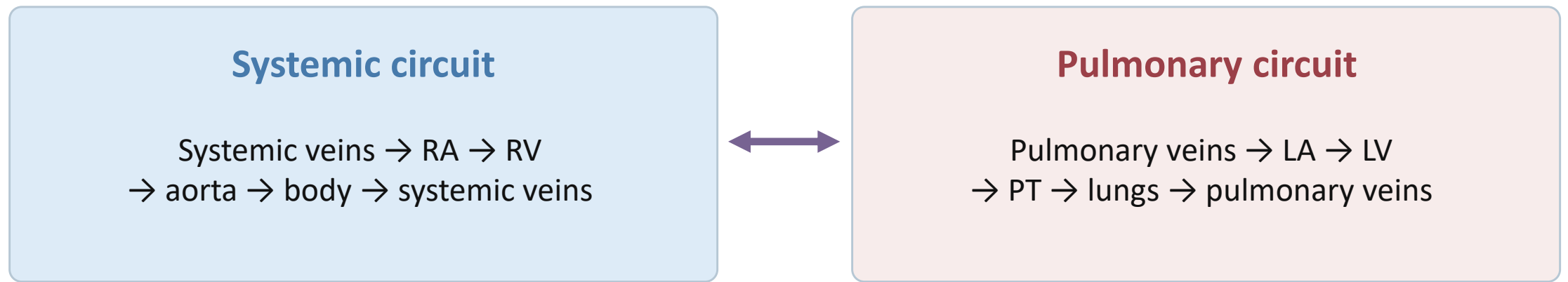
Atrial level: Patent foramen ovale (PFO), or an atrial communication.



VSD permits interventricular mixing

A VSD connects the ventricles otherwise serving separated circuits.

VSD — ventricular level

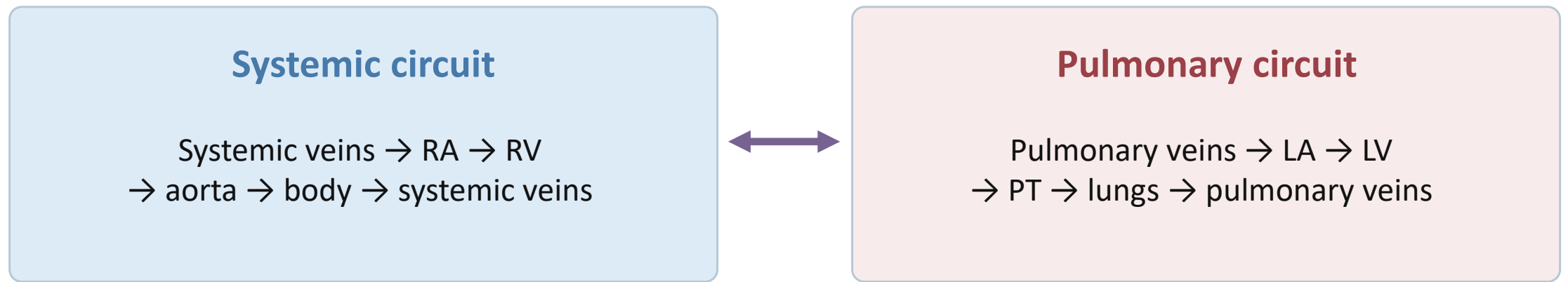


Schematic mixing between circuits; The label identifies the communicating anatomical level.

PFO permits interatrial mixing

The patent foramen ovale is an opening between the atria.

PFO — atrial level

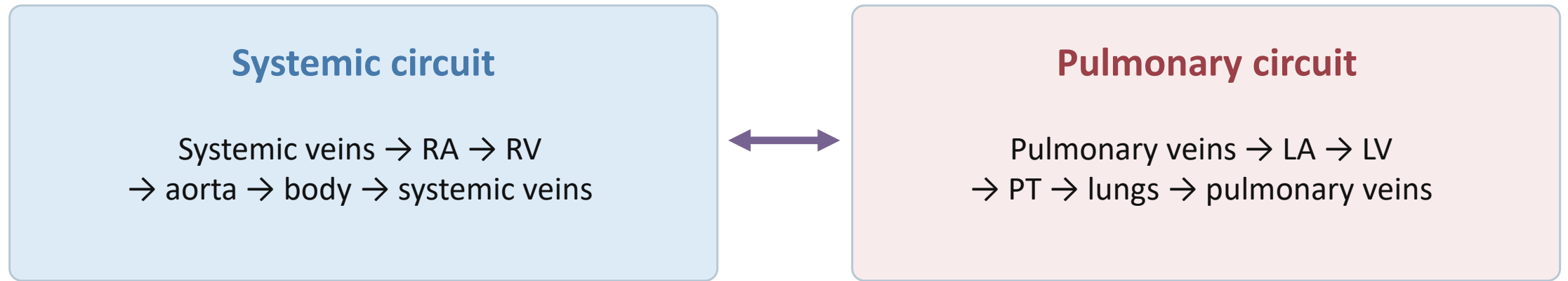


Schematic mixing between circuits; The label identifies the communicating anatomical level.

PDA permits great-vessel mixing

The ductus arteriosus joins pulmonary artery and aorta.

PDA — great-vessel level



Schematic mixing between circuits; The label identifies the communicating anatomical level.

Shunt anatomy predicts stability of blood mixing

Patients with VSD against those dependent on PFO or PDA.

Mixing pathway	Clinical implication in d-TGA
VSD	Often a relatively stable shunt
PFO	Communication vulnerable to functional closure
PDA	Communication vulnerable to physiological closure

Do not mistake mixing proportions for survival probabilities

The source cites VSD in approximately 35% of d-TGA patients.

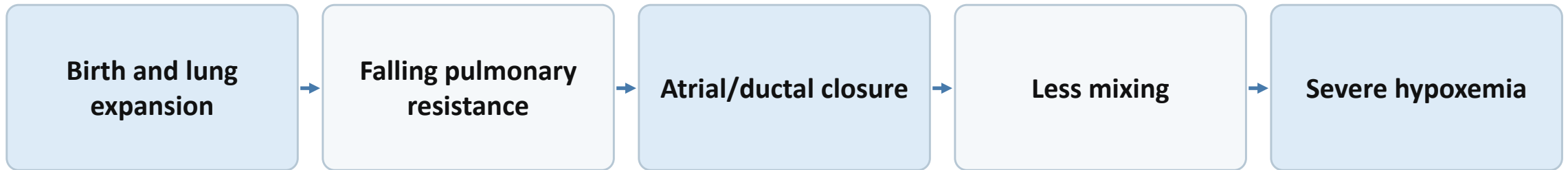
Reported arrangement	Approximate share	Meaning
d-TGA with VSD	35%	Ventricular mixing pathway
PFO/PDA-dependent mixing	65%	Vulnerable postnatal pathways

Postnatal transition can close a vital shunt

In term infants, the ductus normally constricts within 1–2 days of birth.

At birth, lower pulmonary vascular pressure contributes to foramen ovale closure.

These normal events can sharply reduce mixing in d-TGA.

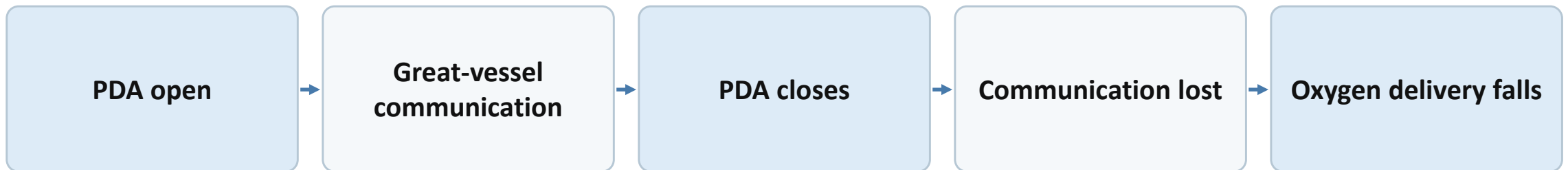


Why closure of the ductus is dangerous in d-TGA

Ductus patency allows connection between aorta and pulmonary trunk.

Functional closure removes that communication.

If other mixing sites are inadequate, systemic oxygenation deteriorates rapidly.

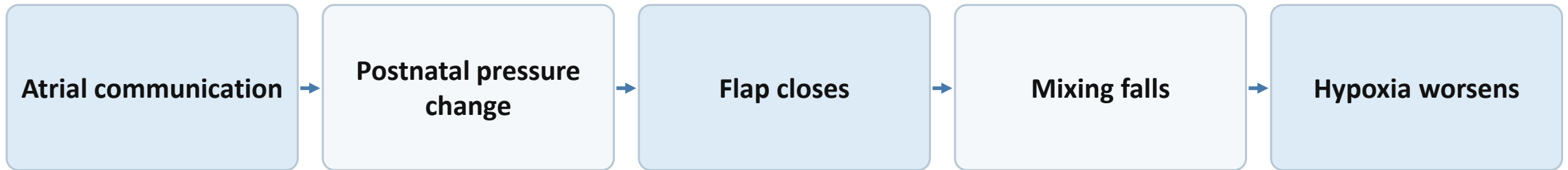


A closing foramen ovale creates a similar risk

The fetal foramen ovale normally permits right-to-left atrial passage.

Postnatal changes in atrial pressures favor flap closure.

A d-TGA infant dependent on this pathway can lose crucial interatrial mixing.



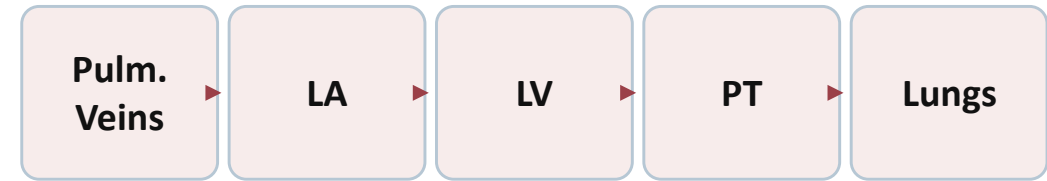
Hypoxemia is a circuit problem, not merely poor lung function

Pulmonary blood can still become oxygenated in the lung circuit.

Systemic loop — poorly oxygenated return



Pulmonary loop — oxygenated venous return



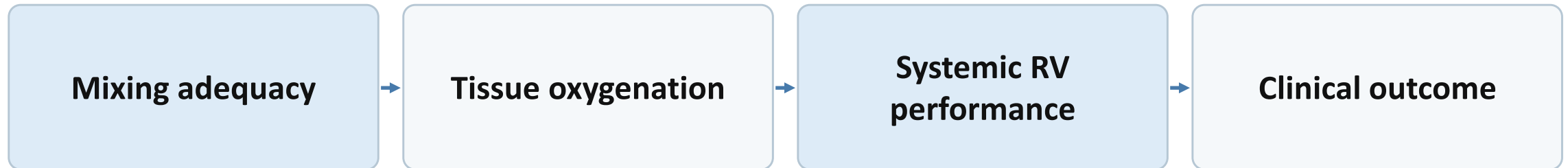
Parallel-loop schematic • PT = pulmonary trunk; Dashed arrows indicate recirculation

Three determinants of outcome in d-TGA

Extent of blood mixing between parallel circulations.

Severity of tissue hypoxia from inadequate systemic oxygen delivery.

Ability of the morphologic right ventricle to sustain systemic circulation.



Early cyanosis reflects inadequate oxygenated systemic flow

TGA is grouped with cyanotic congenital heart lesions.

Mixing failure allows poorly oxygenated blood to supply systemic arteries.

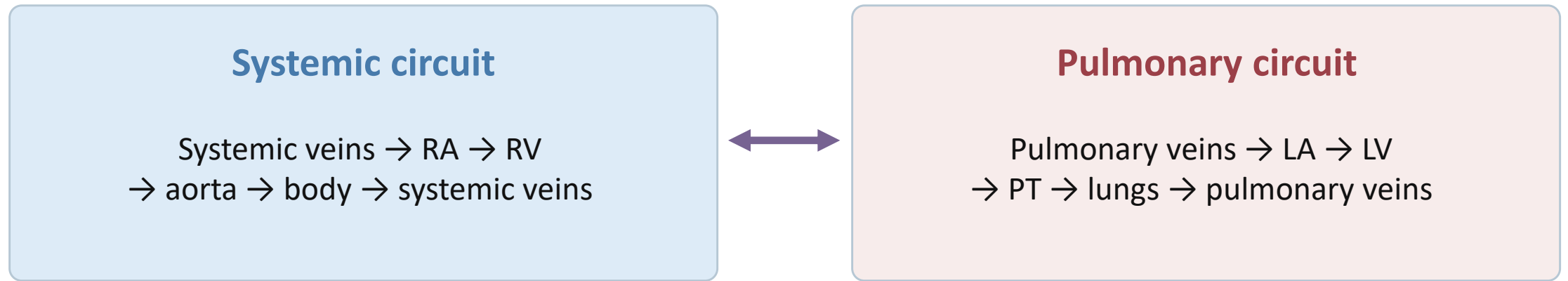
Presentation may become critical as transitional interconnections close.



A VSD can stabilize the newborn circulation

A persistent interventricular opening connects the two pathways.

Persistent VSD: Stable mixing



Schematic mixing between circuits; The label identifies the communicating anatomical level.

PFO/PDA dependence creates a narrow intervention window

The source describes closure of these communications early after birth.

Loss of mixing poses an immediate physiological threat.

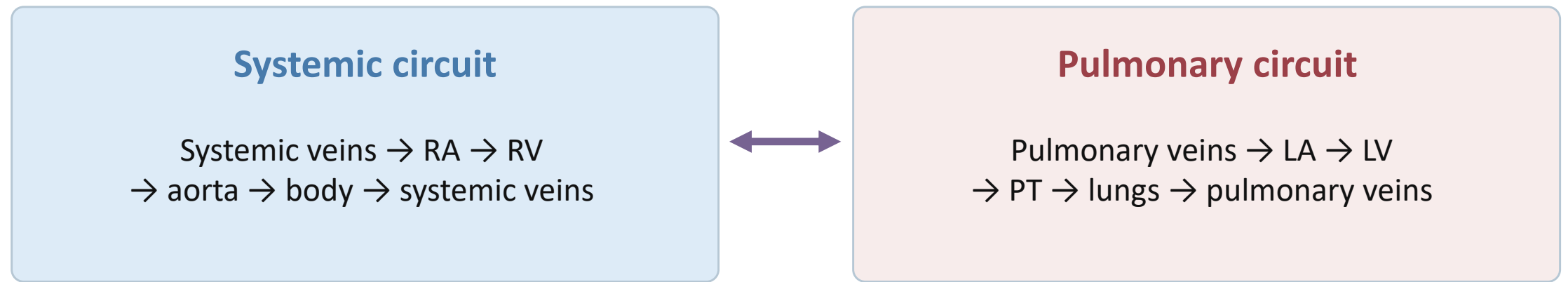
An intervention to establish communication may be required in the first days of life.



Balloon atrial septostomy: Physiological objective

Balloon atrial septostomy as an early intervention.

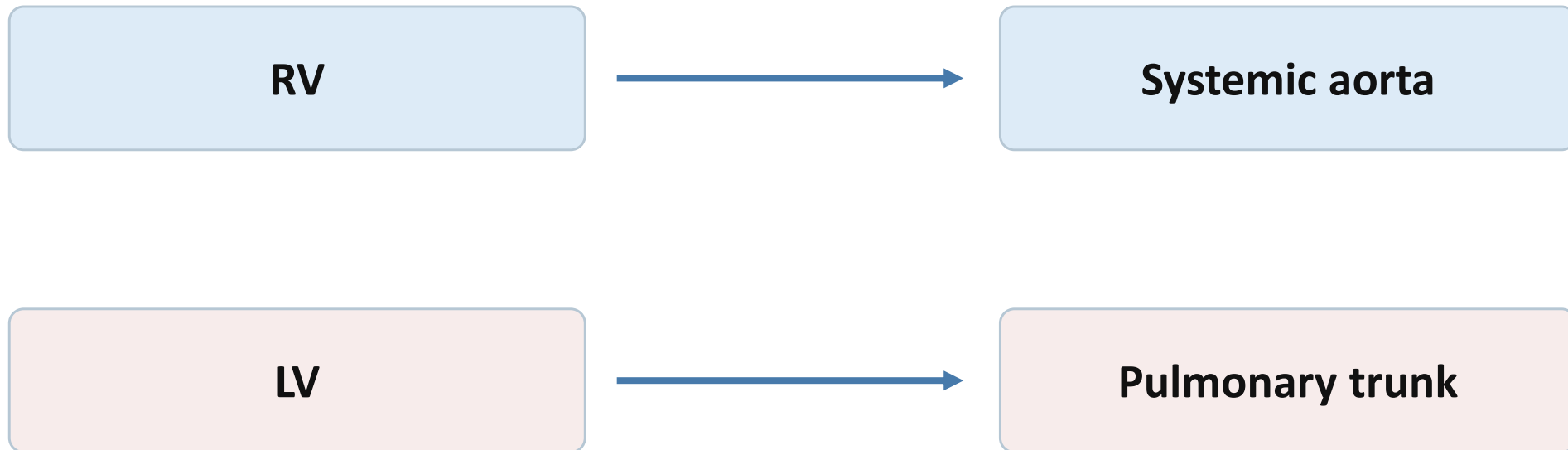
Created atrial communication



Schematic mixing between circuits; The label identifies the communicating anatomical level.

The morphologic right ventricle becomes systemic

In d-TGA, RV pumps directly into the aorta.

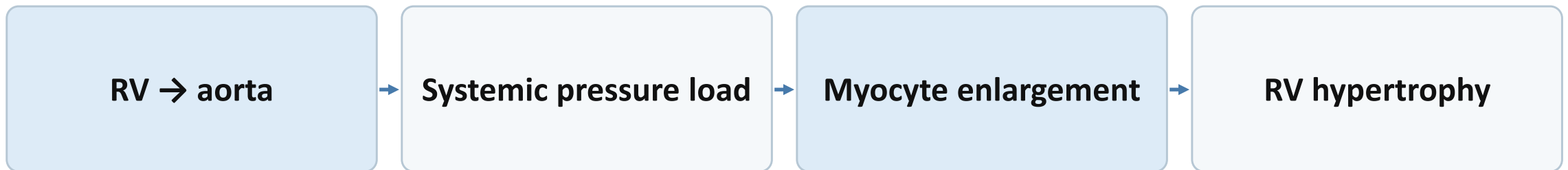


Systemic loading produces right ventricular hypertrophy

The source states that RV hypertrophy becomes prominent over time.

This is a response to maintaining the high-pressure systemic circulation.

The hypertrophy is an acquired hemodynamic consequence, not an additional connection anomaly.

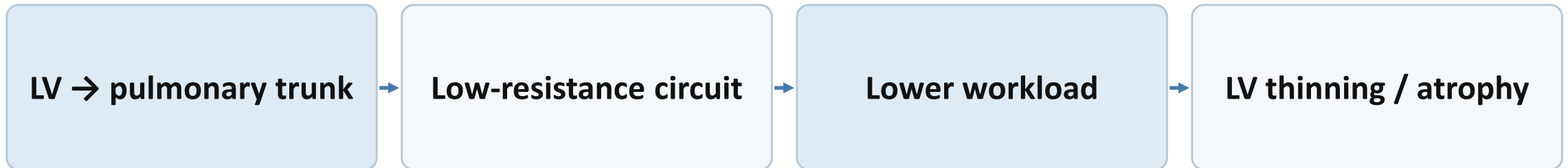


Pulmonary loading produces left ventricular thinning

The morphologic LV supplies the lower-resistance pulmonary circuit.

Over time, a thin-walled, atrophic LV.

The remodeling direction is opposite to that of the systemic RV.



D-TGA ventricular remodeling is asymmetric

Right ventricle: Systemic pressure work → hypertrophy.

Morphologic RV

- Ejects into aorta
- Maintains systemic circulation
- Progressive hypertrophy

Morphologic LV

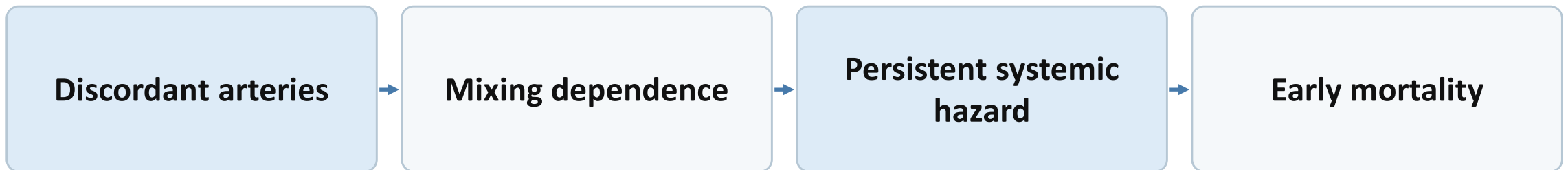
- Ejects into pulmonary trunk
- Supports lower-resistance circuit
- Progressive thinning / atrophy

Without surgical correction, natural history is severe

Most infants with untreated d-TGA die within months.

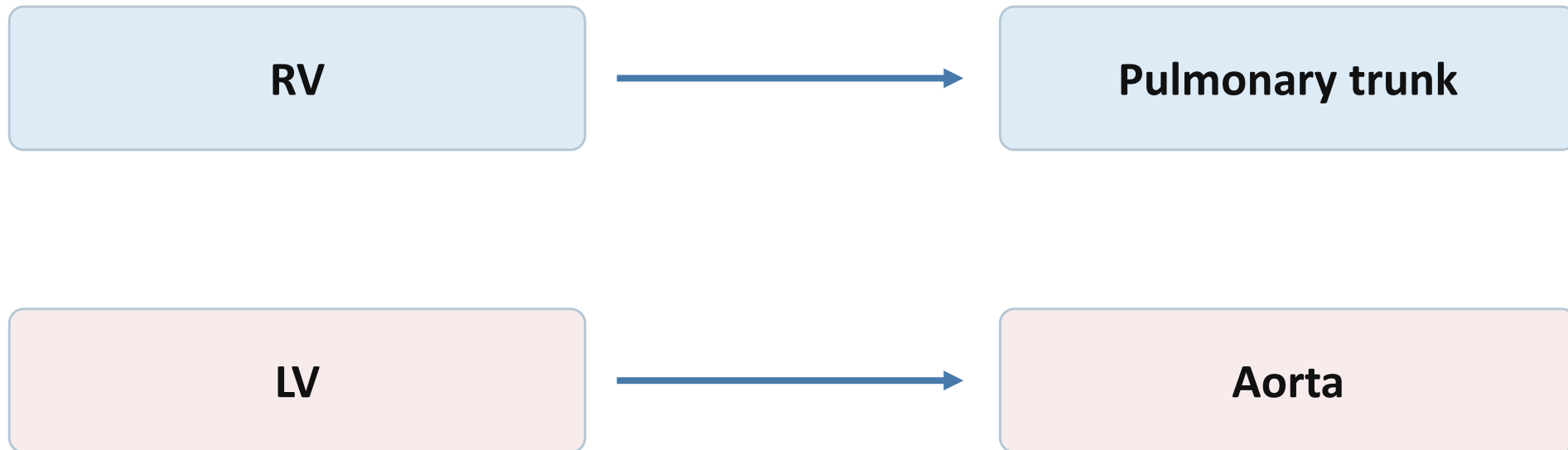
Critical limitations are systemic hypoxia and sustaining systemic output.

A transiently successful shunt is not definitive anatomical correction.



Definitive surgery restores the arterial pathway

Early surgery that repositions the aorta and pulmonary artery.



Interim mixing and definitive correction solve different problems

Balloon atrial septostomy increases communication between parallel streams.

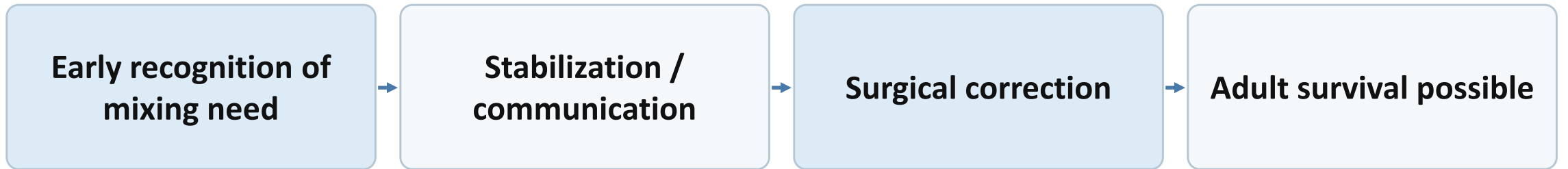
Approach in source	Immediate objective	Changes great-artery origins?
Balloon atrial septostomy	Improve intercirculatory mixing	No
Great-artery repositioning	Correct outlet alignment	Yes

Why survival into adulthood is now possible

Untreated d-TGA has a very poor natural history in the source.

Earlier stabilization and surgical correction alter that course.

Many operated patients now reach adulthood.



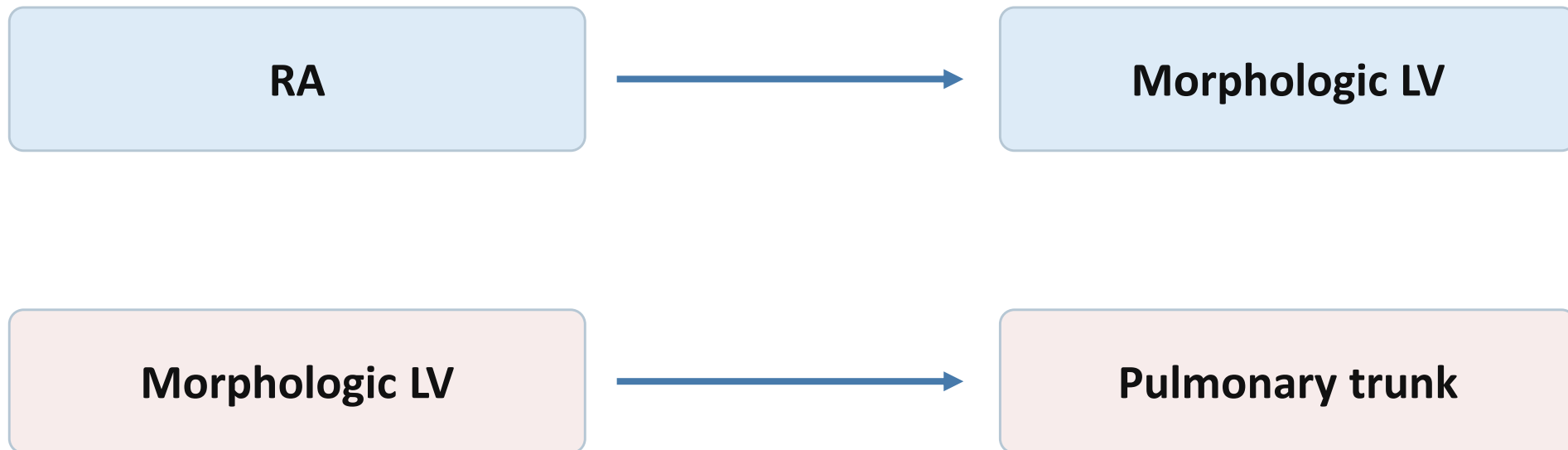
L-TGA adds atrioventricular discordance

Less common l-TGA is also called congenitally corrected transposition.

Connection level	l-TGA arrangement
Right atrial inflow	RA → morphologic LV
Left atrial inflow	LA → morphologic RV
Ventricular arterial outlets	Morphologic LV → PT; morphologic RV → aorta

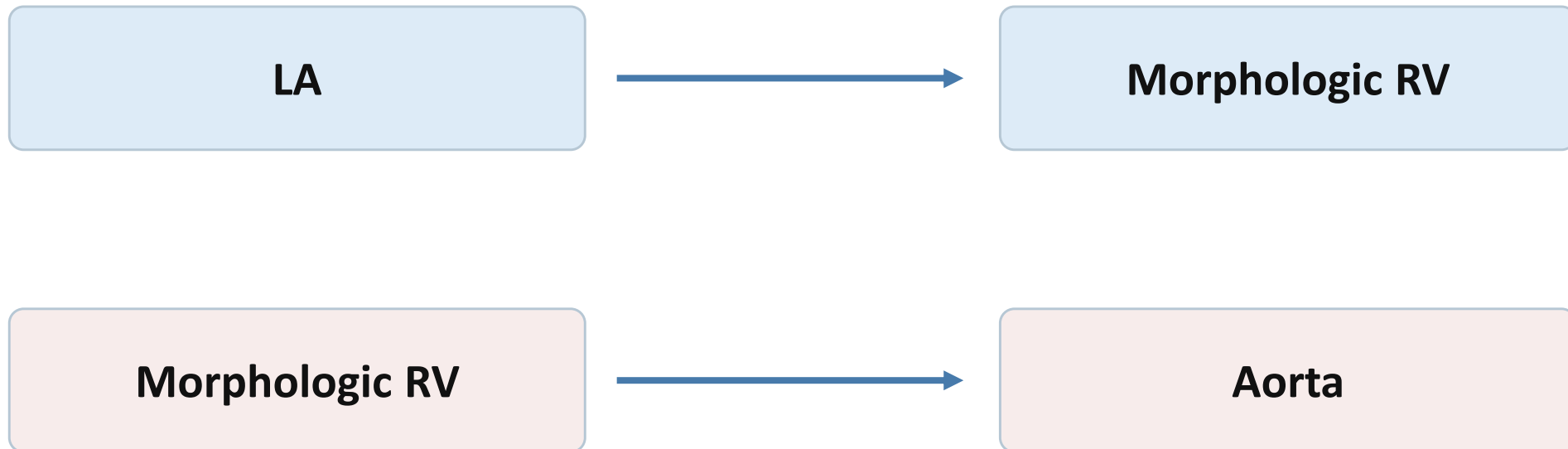
Right atrium drains into the morphologic left ventricle

In l-TGA, the right atrium connects to an LV by internal morphology.



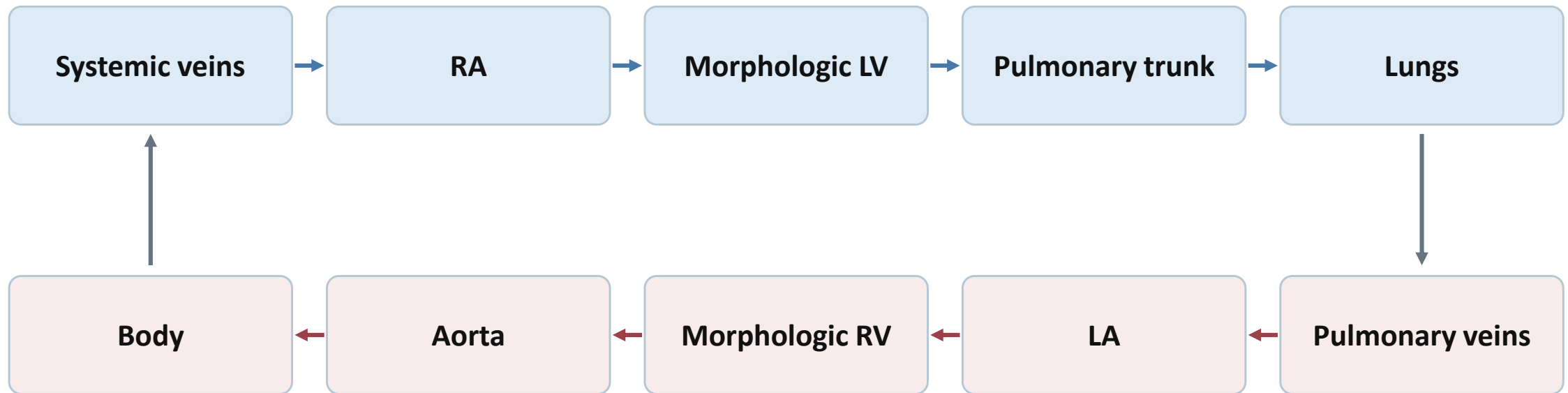
Left atrium drains into the morphologic right ventricle

The left atrium connects to a morphologic right ventricle.



Double discordance produces physiological correction

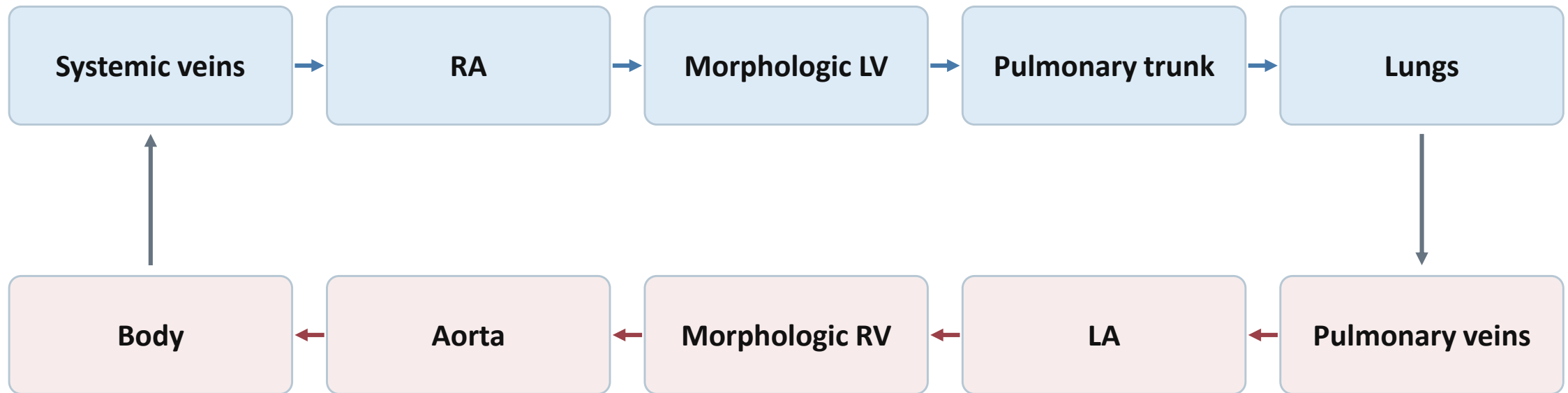
AV discordance sends venous blood into the morphologic LV and oxygenated blood into RV.



Physiological series pathway persists despite two discordant connection levels

Why uncomplicated l-TGA need not cause cyanosis

The right atrial stream reaches lungs through a morphologic LV.



Physiological series pathway persists despite two discordant connection levels

Congenital correction does not mean a normal heart

Physiologically useful blood flow masks two anatomical discordances.

Physiological benefit

- Oxygenated blood reaches aorta
- No intrinsic cyanosis from discordance alone

Anatomical liability

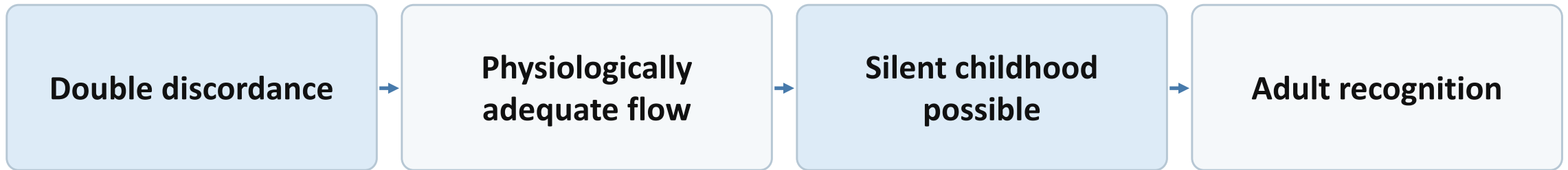
- Systemic morphologic RV
- Potential progressive heart failure

Congenitally corrected TGA may remain asymptomatic

Some people have no early symptoms.

The diagnosis may be made during evaluation for another cardiac concern in adulthood.

Absence of cyanosis must not be taken as proof of normal cardiac connections.



The morphologic RV is exposed to systemic loading

The morphologic RV empties into the aorta in l-TGA.

Longstanding systemic loading produces ventricular hypertrophy.

The same chamber is the long-term vulnerable systemic pump.

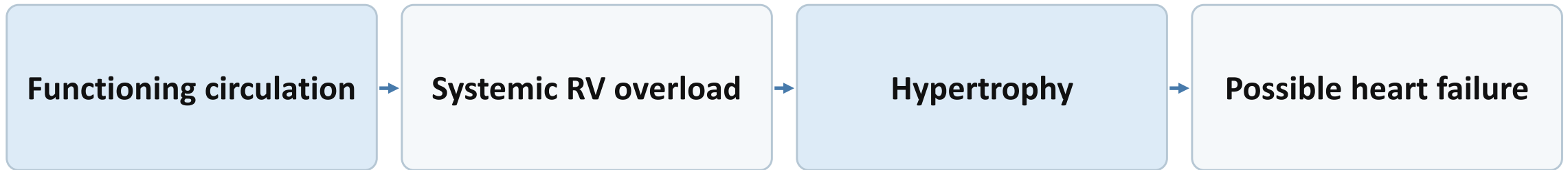


Systemic RV failure is a late clinical consequence

L-TGA can eventually cause heart failure.

The consequence follows long-term reliance on the morphologic RV for systemic output.

Early clinical quiescence and late ventricular dysfunction are compatible.



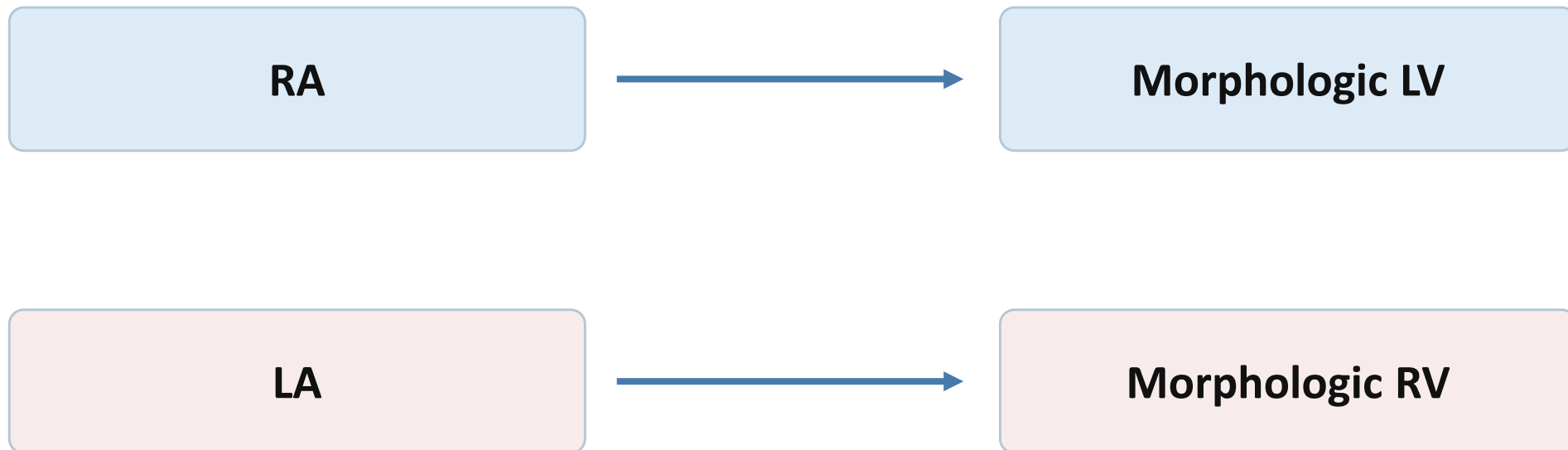
Other congenital defects frequently coexist with l-TGA

The source explicitly lists ventricular septal defect (VSD).

Associated defect	Anatomical level
VSD	Interventricular communication
ASD	Atrial septal communication
PFO	Persistent fetal interatrial flap channel

Morphologic identity—not ventricular side—sets the terminology

In l-TGA, the ventricle connected to RA has left-ventricular morphology.



D-TGA versus l-TGA: Anatomic logic

D-TGA has one discordant connection level: Ventriculoarterial.

Anatomical level	d-TGA	l-TGA
Atrioventricular	Concordant	Discordant
Ventriculoarterial	Discordant	Discordant
Net arrangement	Parallel circuits	Physiological series circulation

D-TGA versus I-TGA: Clinical and ventricular consequences

D-TGA: Urgent mixing dependence and early cyanosis.

Feature	d-TGA	I-TGA
Immediate oxygenation	Mixing needed	Usually maintained
Typical source-described course	Critical infancy untreated	Can be silent until adulthood
Morphologic RV	Systemic pump; hypertrophy	Systemic pump; hypertrophy/failure

TGA and tetralogy of Fallot: Different mechanisms of cyanosis

TOF combines VSD, RV outflow obstruction, overriding aorta, and RV hypertrophy.

Disease	Primary anatomy	Why hypoxemia occurs
Tetralogy of Fallot	VSD plus RV outflow obstruction and overriding aorta	Right-to-left shunt across VSD when obstruction severe
d-TGA	RA→RV→aorta; LA→LV→PT	Inadequate mixing of parallel loops

PDA: A lesion by itself, a lifeline in d-TGA

Isolated PDA ordinarily creates abnormal flow between aorta and pulmonary artery.

Isolated PDA context

- Persistent great-vessel connection
- Often left-to-right flow initially

D-TGA context

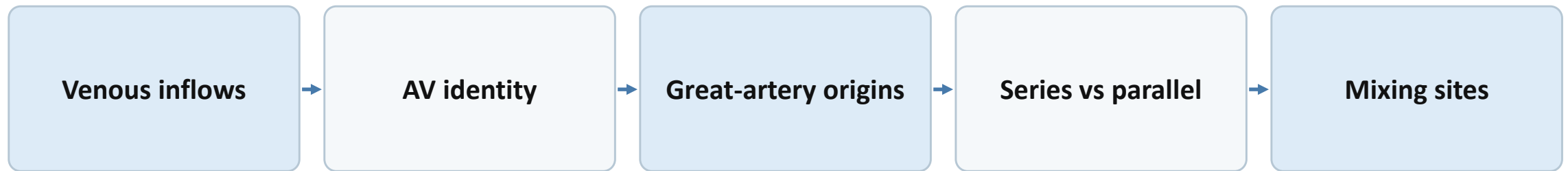
- Bridge between parallel loops
- Closure can sharply impair oxygenation

Read a congenital cardiac diagram in four checks

Identify atrial return: Which atrium receives systemic versus pulmonary veins?

Trace atrium-to-morphologic-ventricle connections.

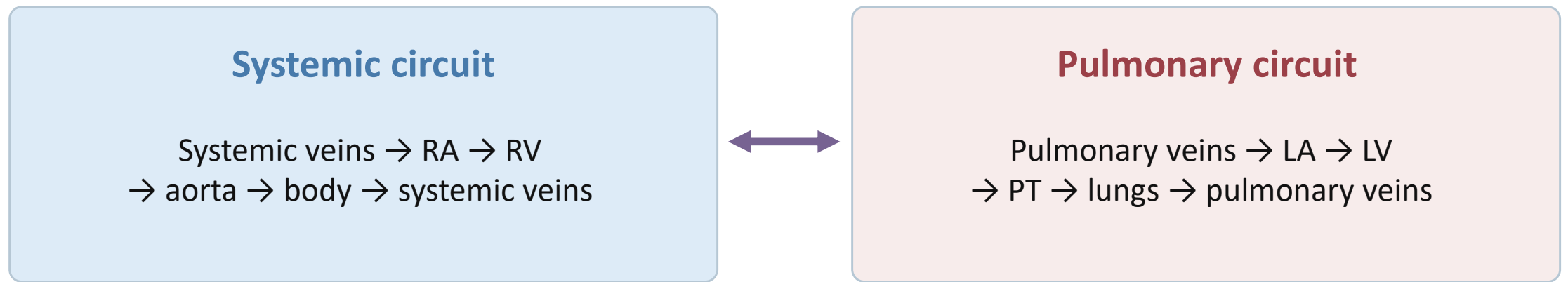
Trace each ventricle to pulmonary trunk or aorta.



Case: Neonatal cyanosis with closing ductus

A newborn has d-TGA without an appreciable VSD; Ductal mixing has been maintaining systemic oxygenation.

PDA closing: Mixing threatened



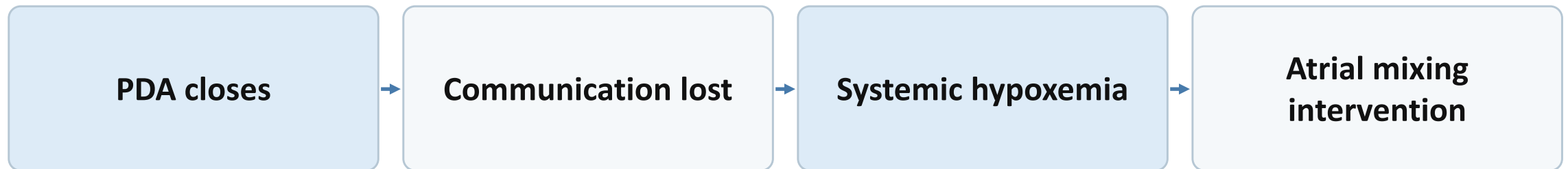
Schematic mixing between circuits; The label identifies the communicating anatomical level.

Case interpretation: Ductal closure eliminates a bridge

D-TGA circuits remain parallel; Lung oxygenation does not by itself supply aorta.

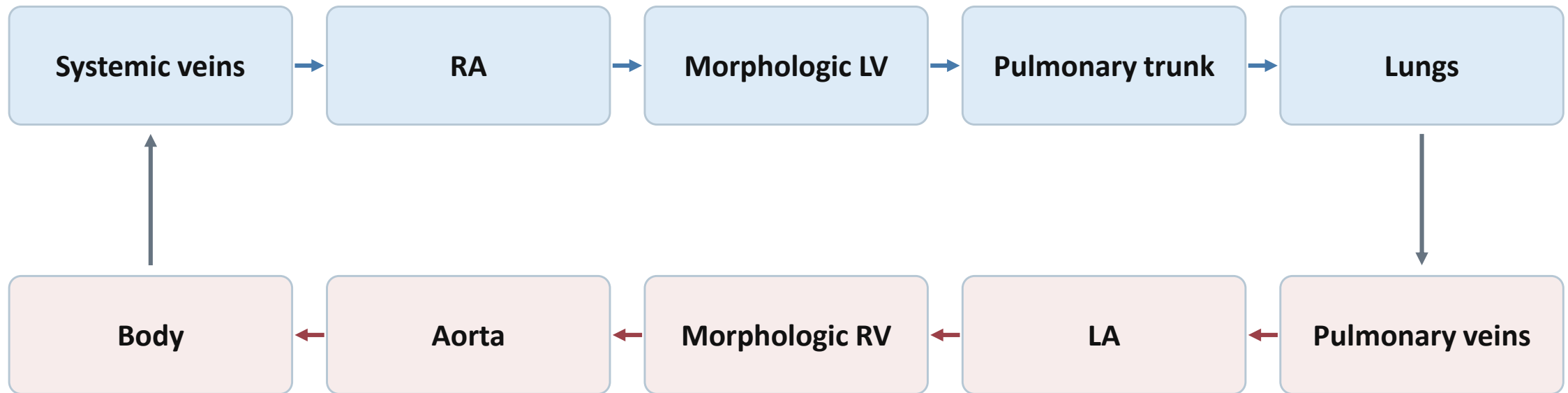
Closure of the PDA reduces cross-circuit communication.

The source supports urgent creation of another mixing route, e.g., atrial septostomy.



Case: Adult without cyanosis but a systemic morphologic RV

An adult evaluated for another cardiac concern has RA → morphologic LV and LA → morphologic RV.



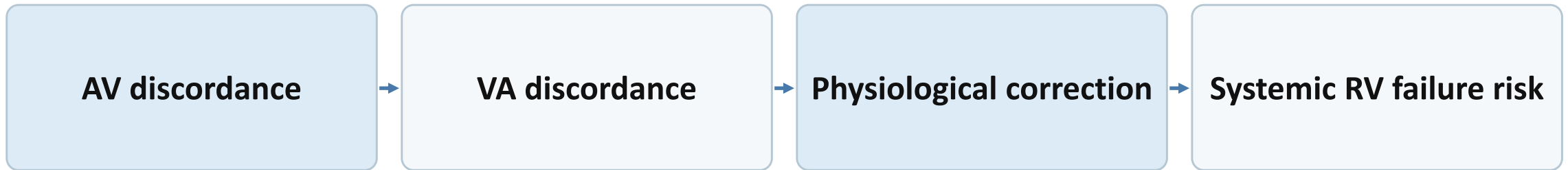
Physiological series pathway persists despite two discordant connection levels

Case interpretation: L-TGA with systemic RV vulnerability

The two discordant connection levels establish congenitally corrected L-TGA.

Effective series flow explains the absence of intrinsic cyanosis.

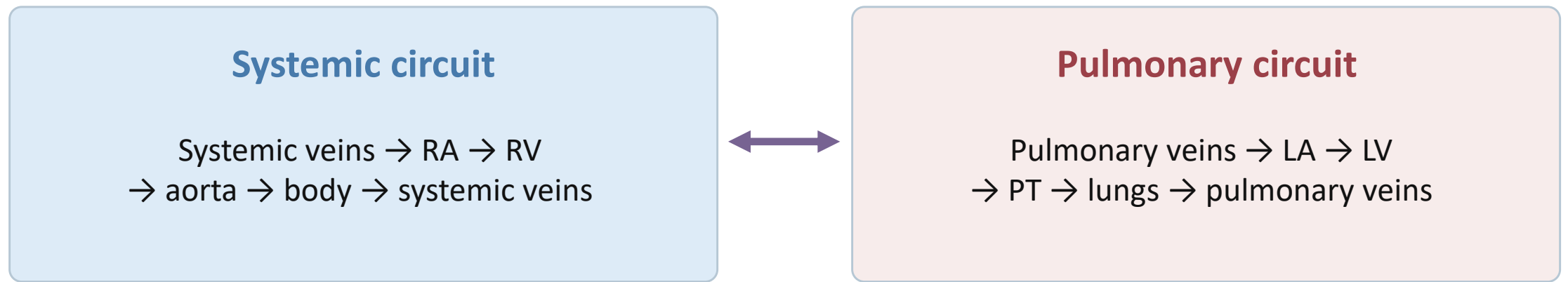
The systemic morphologic RV may hypertrophy and eventually fail.



Case: Communication present despite abnormal outflow

A d-TGA newborn has a VSD providing stable intercirculatory mixing.

VSD permits mixing; Arteries remain discordant



Schematic mixing between circuits; The label identifies the communicating anatomical level.

Case interpretation: Physiological bridge is not repair

A VSD can improve mixing without changing RV–aorta and LV–pulmonary artery origins.

Observed feature	Interpretation
VSD mixing	Acute physiological benefit
Persistent VA discordance	Uncorrected anatomical lesion
Arterial repositioning	Definitive anatomic correction in source

Integrate anatomy, physiology and outcome

D-TGA: VA discordance with AV concordance → parallel loops → mixing-dependent neonatal survival.

Variant	Anatomy	Physiology	Dominant risk
d-TGA	AV concordant; VA discordant	Parallel circuits	Critical hypoxemia without mixing
l-TGA	AV and VA discordant	Physiological series flow	Systemic RV failure over time

Primary reading

Robbins, Cotran & Kumar

Pathologic Basis of Disease • 11th edition (2026)

The Heart — Congenital Heart Disease

Review the sections on right-to-left shunts, transposition, and related congenital lesions.