CLINICAL

بسم الله الرحمن الرحيم

FINAL | Lecture 2

Clinical Pediatric Cardiology

وَلَقَدْ خَلَقْنَا الْإِنسَانَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِ نَفْسُهُ ۖ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ الْوَرِيدِ

اللهم إنّا نعوذ بك من شرور أنفسنا ومن سيئات أعمالنا

Written by: Sadeel Al-hawawsheh

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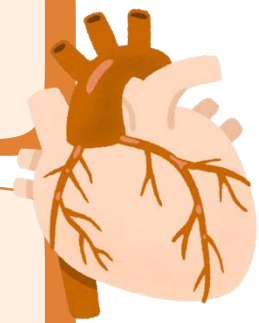

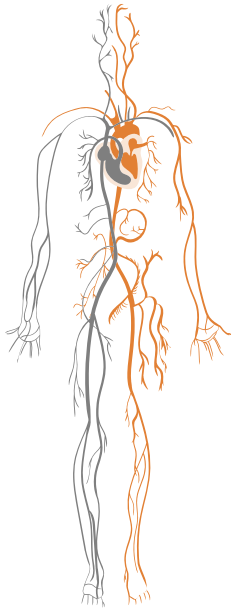

وَلِلَّهِ الْأَسْمَاءُ الْحُسْنَى فَادْعُوهُ بِهَا

المعنى: الذي له العلوّ المطلق من جميع الوجوه، علوّ الذات، وعلوّ القدر والصفات، وعلوّ القهر والغلبة، وهو المتعالي على خلقه، وكل شيء هو تحت قهره وسلطانه.

الورود: ورد اسم (العلي) ٨ مرات، أما اسم (الأعلى) فورد مرتين، واسم (المتعال) مرة واحدة.

الشاهد: ﴿وَهُوَ ٱلْعَلِيُّ ٱلْعَظِيمُ﴾ [البقرة:٢٥٥]، ﴿سَبِّحِ ٱسْمَ رَبِّكَ ٱلْأَعْلَى﴾ [الأعلى:١]، ﴿عَٰلِمُ ٱلْغَيْبِ وَٱلشَّهَٰدَةِ ٱلْكَبِيرُ ٱلْمُتَعَالِ﴾ [الرعد:٩].

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اضغط هنا لشرحٍ أكثر تفصيلًا

Clinical Pediatric Cardiology
CVS- 3rd year

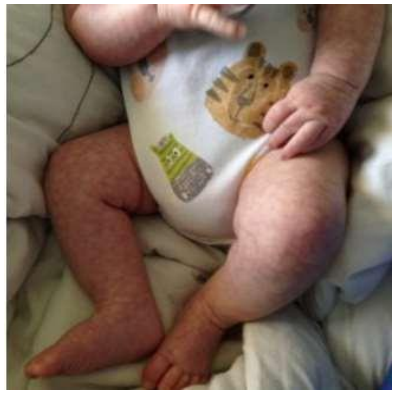
Prof. Iyad AL-Ammouri
Pediatric Cardiology

Introduction

- Pediatric cardiac disease are largely congenital defects. *Congenital* means that the disease develops **before birth**, so the baby is **born** with it, even though signs and symptoms may **not** appear **immediately**; they may show up right after delivery or months to years later.
- Some acquired heart disease may happen in Pediatrics but they are far less common.
- Will present 4 common cases of children with heart disease
- In each case we will focus on:
  - Embryologic consideration leading to the anatomic defect
  - Physiology and hemodynamics leading to the clinical picture

Case 1

- Two month old infant who has:
  - Rapid breathing (tachypnea)
  - Difficulty of feeding
  - Not gaining weight appropriately



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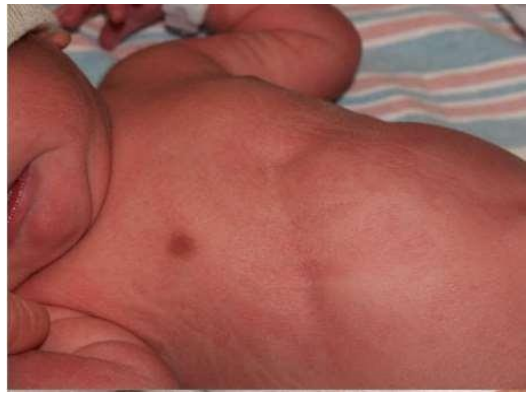

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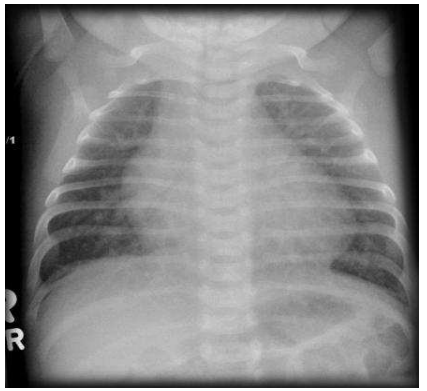

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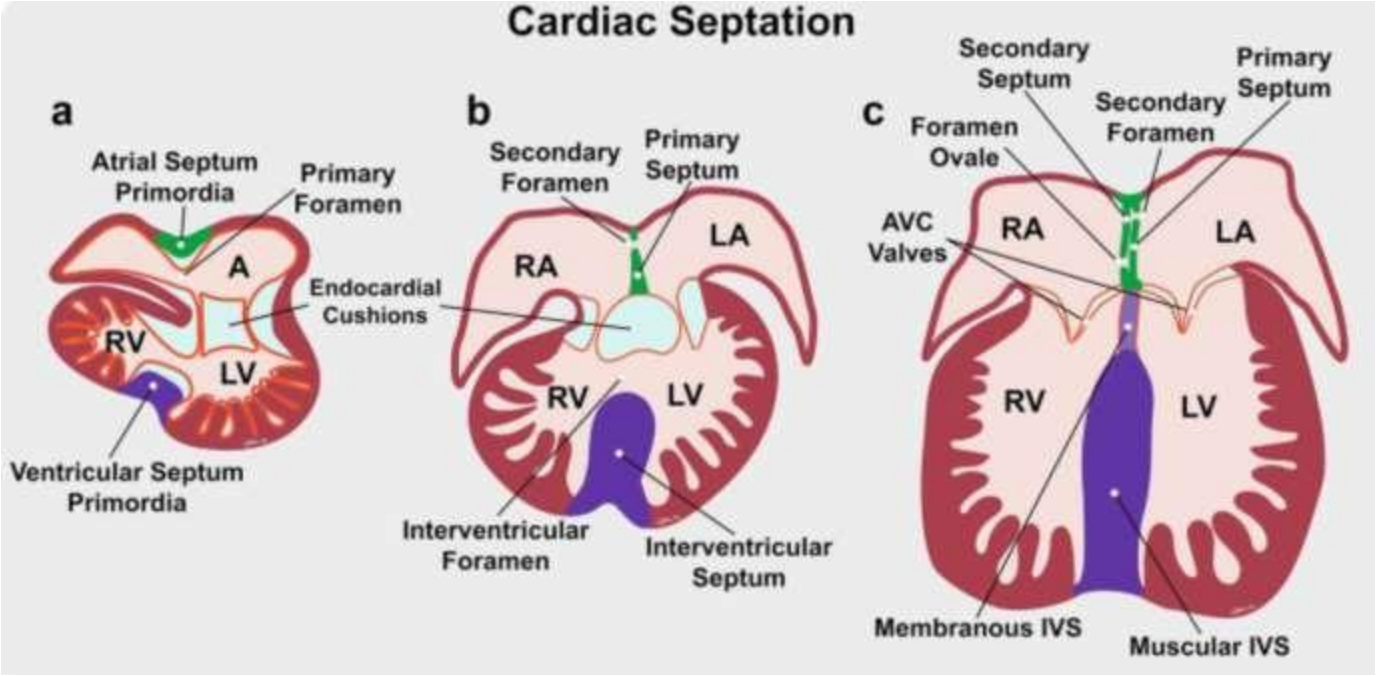
- Examination:
  - Signs of respiratory distress (tachypnea, use of accessory muscles, and visible chest retractions)
  - Rapid heart rate (tachycardia)
  - Weak pulses, skin hypo-perfusion reflected by prolonged capillary refill, cold extremities, pallor, and mottled (net of capillaries) (see 1) skin.
  - Systolic heart murmur which indicates turbulent blood flow. Normally, heart sounds should be smooth without murmurs, but structural abnormalities in the heart can create abnormal flow patterns that produce a murmur.

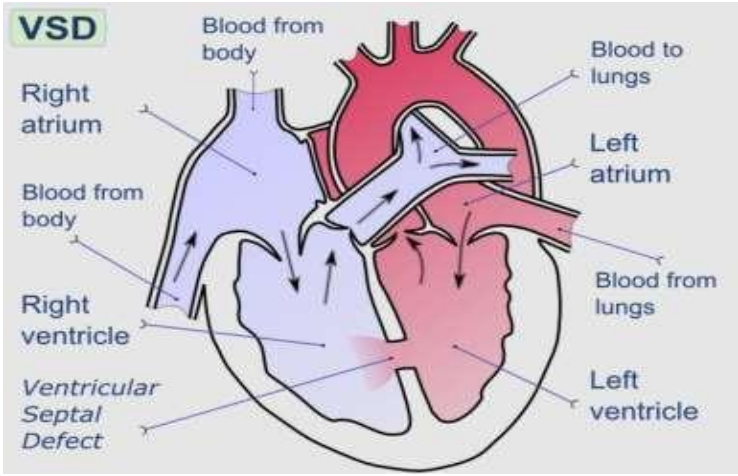
- Chest X ray showed (see 3):
  - Enlarged heart (cardiomegaly)
  - Congested lungs , meaning they contain excess fluid, making them heavier than normal.

Diagnosis: Ventricular septal defect

- Embryologic defect: failure of ventricular septation
  - Septation starts after cardiac looping
  - Interventricular foramen
  - Ventricular septum is composed of muscular part, membranous part, and endocardial cushings

- Anatomical result:
  - Defect between the ventricles
  - Types (muscular, membranous, inlet/canal types..etc)





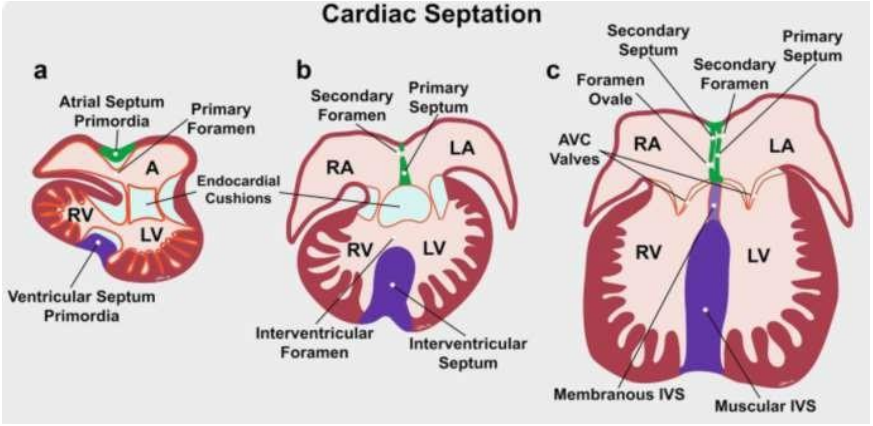
سبحان الله العظيم

Diagnosis: Ventricular septal defect

A ventricular septal defect (VSD) is an abnormal opening between the right and left ventricles. The fetal heart begins as a simple tube containing a **single atrium**, **a single ventricle**, **the sinus venosus**, and the **Bulbus cordis (and the truncus arteriosus above)**. As development progresses, this tube undergoes looping and starts to septate, gradually dividing into separate **right- and left-sided chambers.**

As the ventricles loop, they split into distinct right and left portions. The separation between them occurs through a coordinated process involving three major components:

1. **The muscular septum**, which grows upward from the apex of the developing ventricle
2. **The membranous septum**, a thinner structure located near the outflow tracts
3. **The endocardial cushiongs**, which also contribute to the formation of the mitral and tricuspid valves



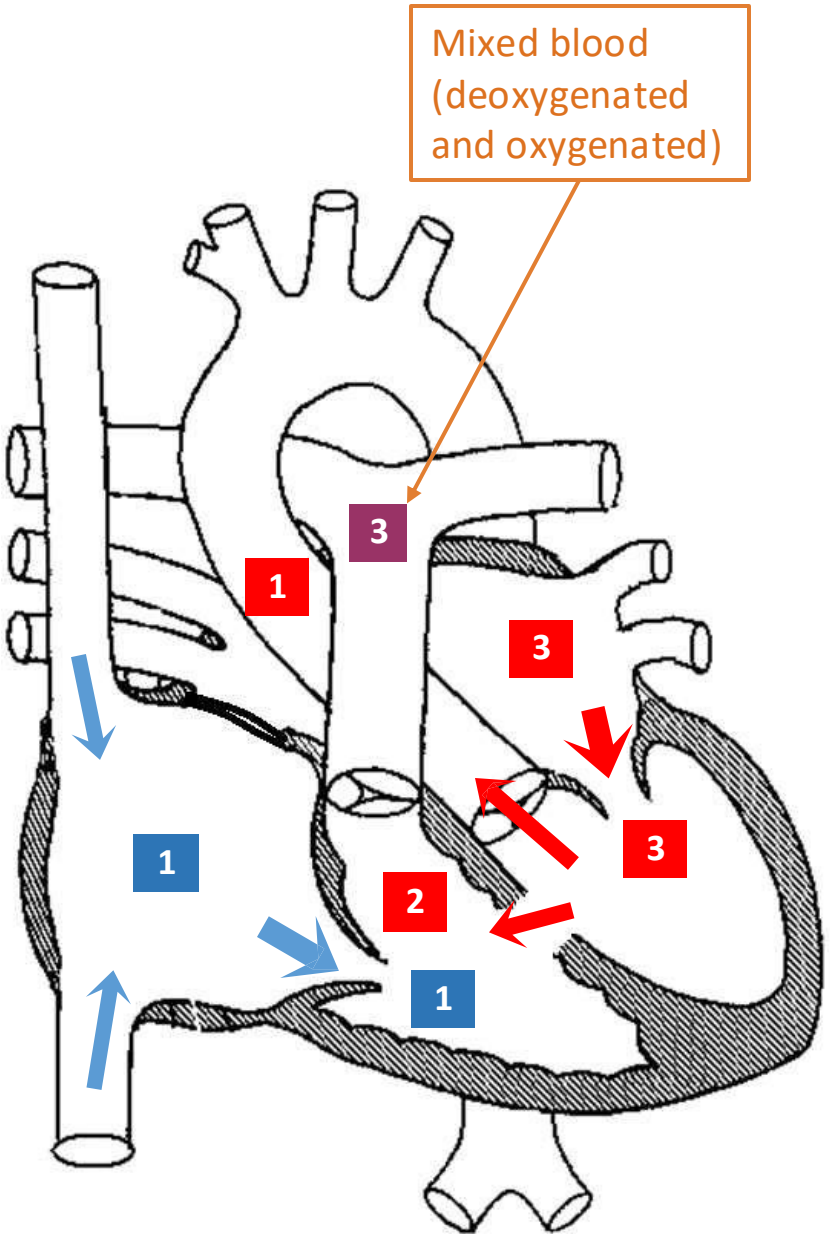
Diagnosis: Ventricular septal defect

During this process, a temporary opening—called the interventricular foramen—exists between the developing ventricles.

As the muscular septum continues to grow and the membranous septum completes the upper portion, the foramen closes, forming the complete interventricular septum. A VSD results when any part of this septation process fails, leaving an opening between the two ventricles.

Depending on which component is affected, the defect may be muscular, membranous, or inlet/canal type. Despite these anatomical differences, the overall clinical features are generally similar.

Physiology of VSD ⟶ clinical picture



In a normal heart with an intact septum, the right and left ventricles pump **equal** amounts of blood. The **right** ventricle delivers **deoxygenated** blood to the **lungs** (pulmonary circulation), and the **left** ventricle delivers **oxygenated** blood to the **systemic circulation.**

When a **VSD** is present, blood may move—or **shunt**—from one side to the other (depending on the pressure & resistance). Blood always flows from **high pressure to low pressure** (in other words: from higher resistance to lower resistance). Because the **left** ventricle has much **higher** pressure than the right (due to higher **systemic** resistance and the resistance in the lungs is much lower due to high total-surface-area), blood usually flows from the left ventricle to the right ventricle **(a left-to-right shunt).**

So during **systole**, instead of all blood leaving the left ventricle into the aorta, part of it crosses the defect into the **right** ventricle, then enters the **pulmonary** artery. This results in excess blood flow to the lungs.
For example:
- The **systemic** output may remain **1 unit** (enough to survive).
- But the **pulmonary** circulation may receive **3 units**—the **normal 1** from the body plus **2** additional units **shunted** from the left ventricle.

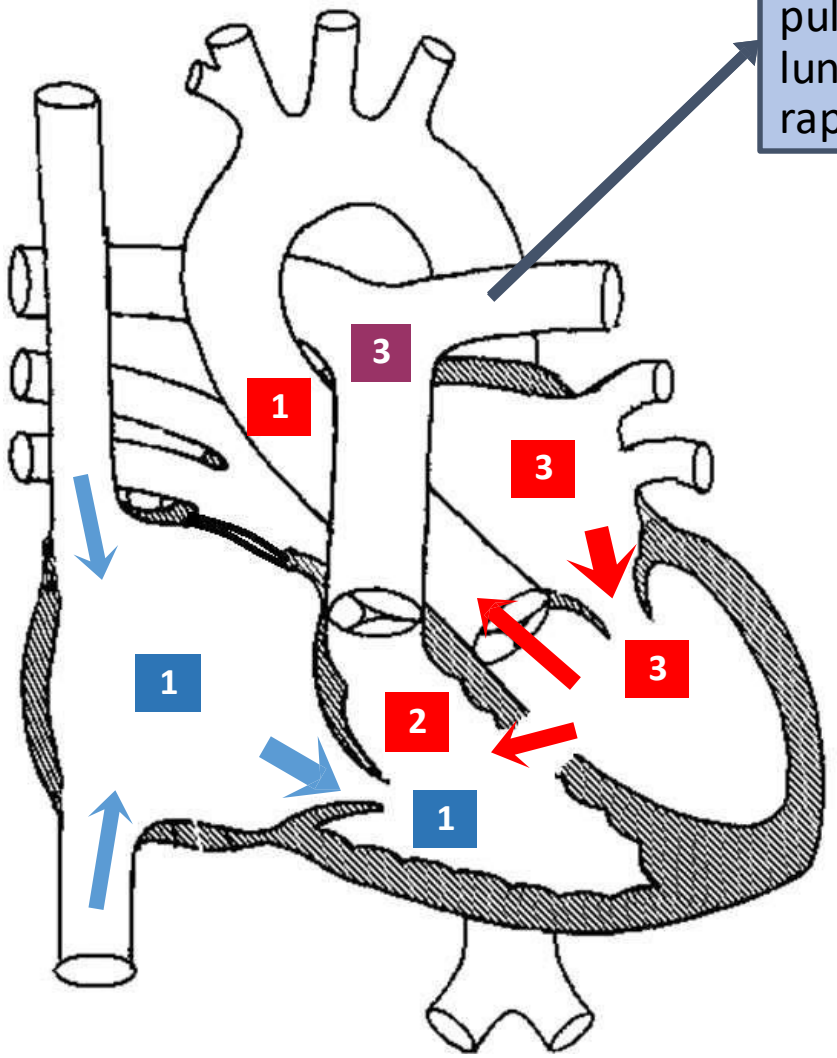
Much of this excess flow is already **oxygenated (the ones shunted)**, so it provides no benefit to the lungs but **increases their workload.**

Diagnosis: Ventricular septal defect

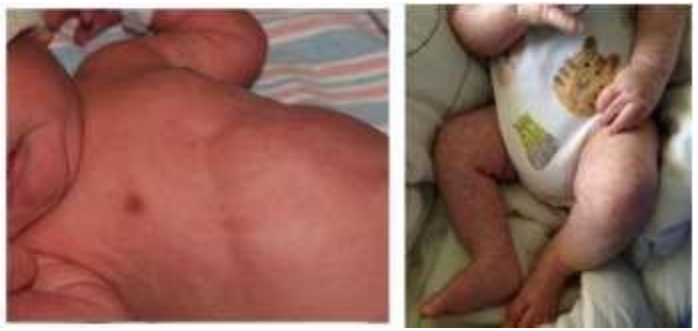
Why Symptoms Appear Around 4–6 Weeks of Age?

In fetal life, a VSD typically causes no symptoms because the fetal circulation largely bypasses the lungs. **Pulmonary vascular resistance (PVR) is high at birth**, then gradually drops to adult levels by **4–6** weeks. When PVR **decreases**, more blood is able to flow through the **lungs**, the **left-to-right shunt** increases dramatically and the lungs become **overloaded** with blood. This is why infants with significant **VSDs become symptomatic around 1–2 months of age**.

Physiology of VSD ⟶ clinical picture



Increased pulmonary blood flow and pulmonary pressure➔ congested lungs ➔ breathing difficulty and rapid breathing



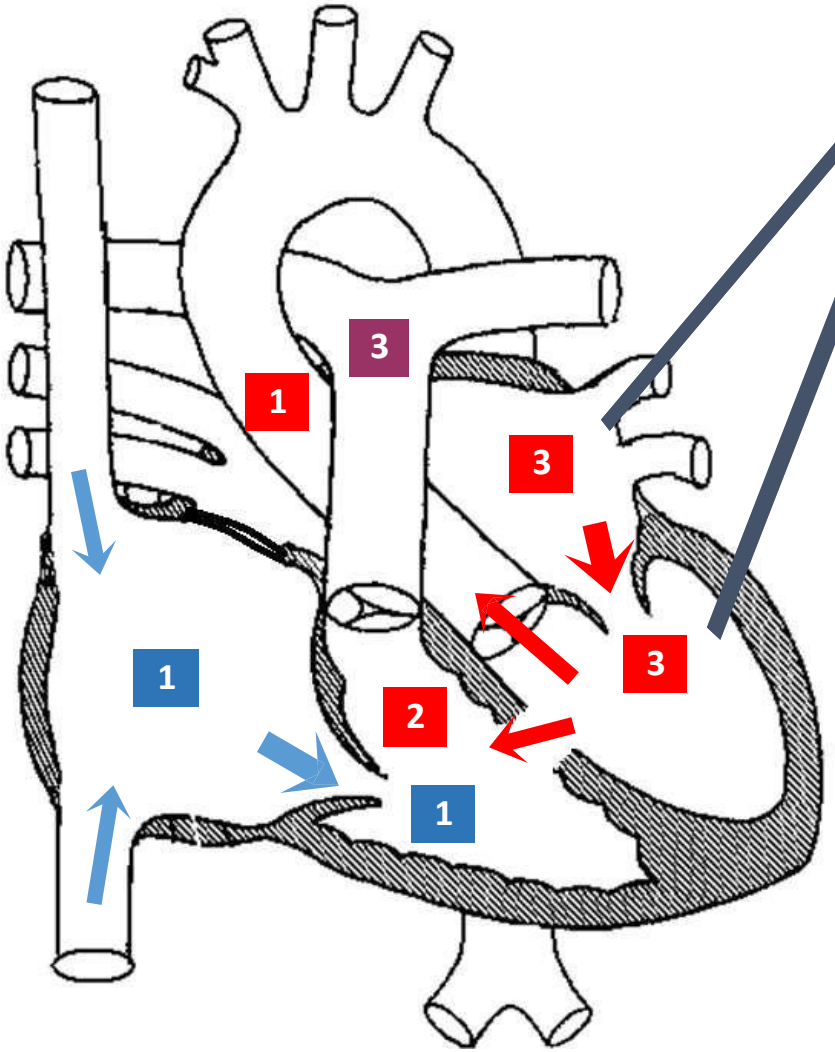
The **excessive** blood volume entering the pulmonary circulation leads to Pulmonary **congestion** , Pulmonary **edema** and Pulmonary **hypertension**

Because the lungs fill with fluid and become heavy —like a wet sponge instead of a dry one— the baby must work harder to expand them. This results in:

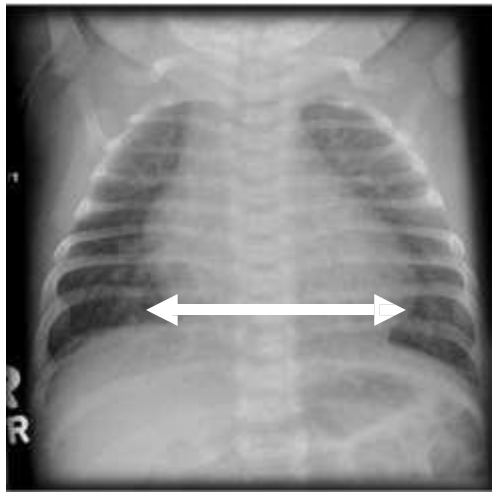
- **Dyspnea (difficulty breathing)**
- **Tachypnea (rapid breathing)**
- **Use of accessory muscles**
- **Chest retractions**

Since **tidal volume** becomes smaller due to stiff, congested lungs, the baby **compensates** by breathing faster to maintain **adequate minute ventilation.**

Physiology of VSD ⟶ clinical picture



Increased left atrial and left ventricular filling ➔ dilatation (cardiomeglly)

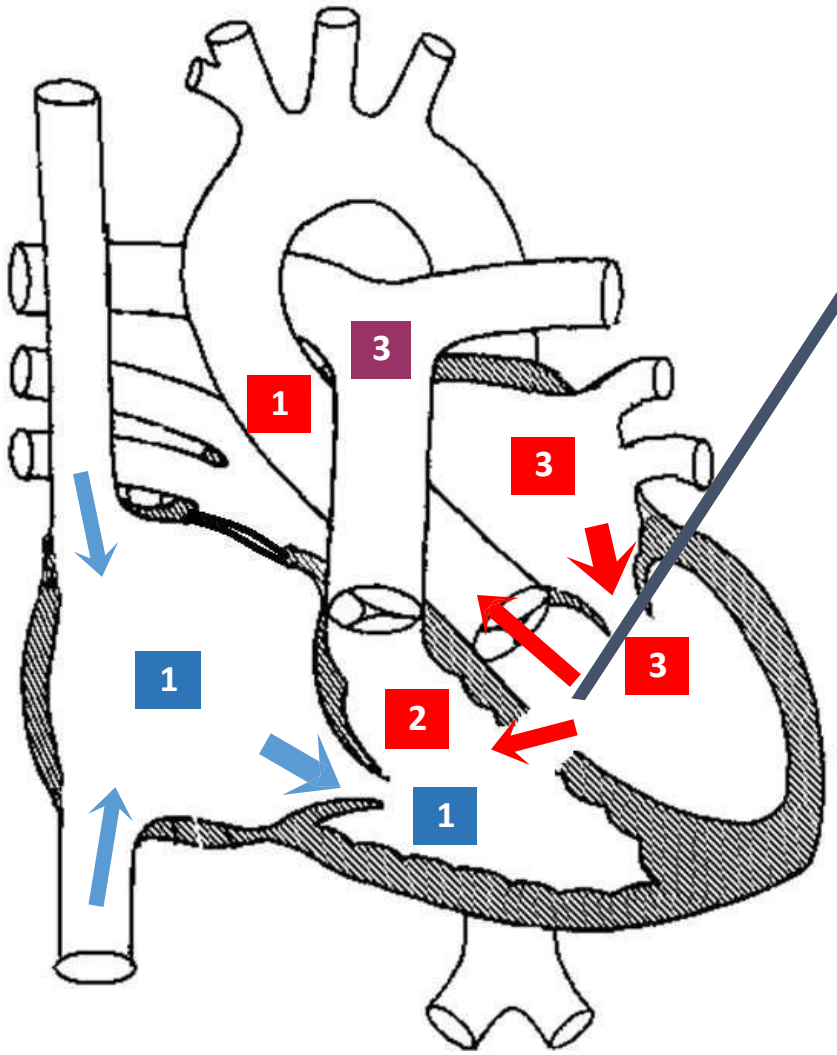


Because of **the left-to-right shunt in a VSD**, the amount of blood returning to the left atrium and left ventricle becomes **approximately three times the normal amount.** This causes volume overload of the left-sided chambers. As a result, the left atrium and left ventricle **dilate**, leading to an enlarged heart. This explains the cardiomegaly seen on physical examination and on chest X-ray.

Plethoric, whitish lung = more perfused

استغفر الله

Physiology of VSD ⟶ clinical picture



Flow between high pressure and low pressure chamber ➔ systolic murmur (turbulence)

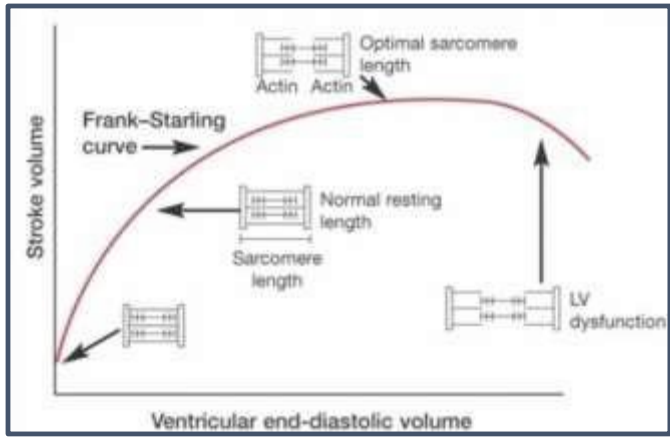
There is continuous blood flow across the VSD from the higher-pressure left ventricle to the lower-pressure right ventricle. The blood moves **rapidly** across the defect (due to different pressure and velocity), **creating turbulent flow**, which produces a **murmur**.

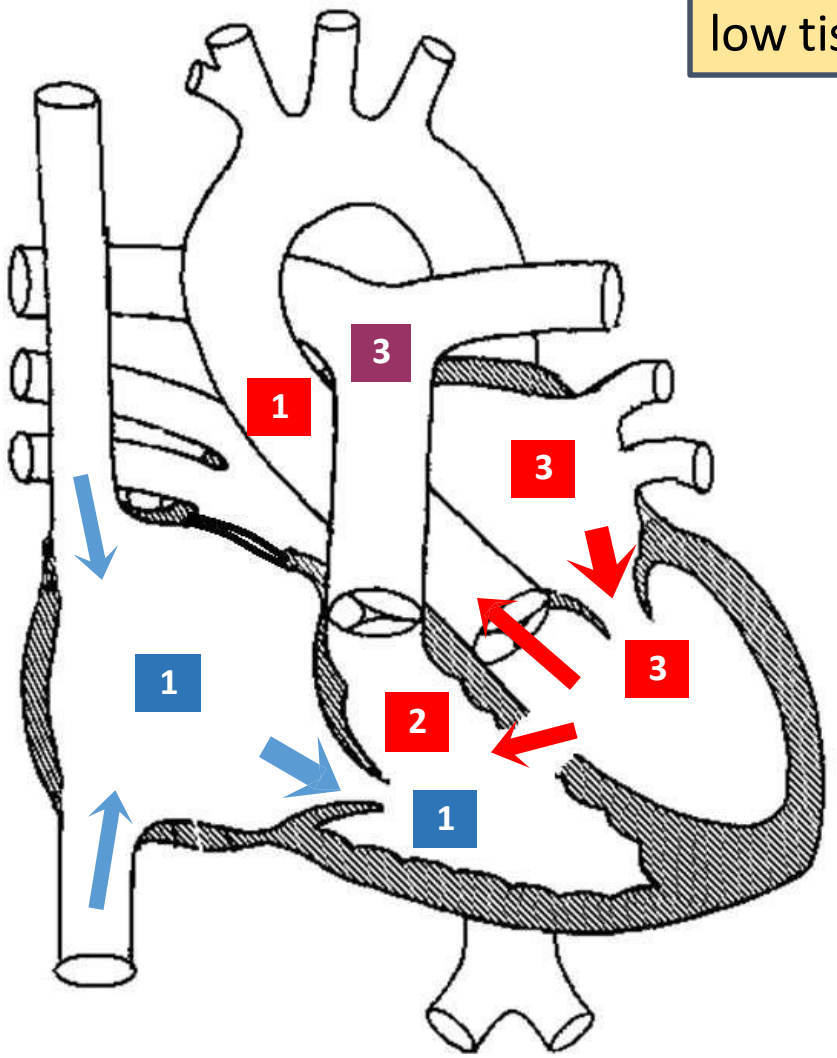
- **Small defects** create **higher-velocity** jets, so the turbulence is greater and the murmur is **louder**. A loud murmur usually suggests a smaller VSD, which is often **favorable** because small defects tend to close over time.

- **Large defects** may produce little or no murmur, because the **pressures** in the two ventricles become nearly **equal**, **reducing** the **speed** of the flow.

Physiology of VSD ⟶ clinical picture

Excessive dilatation eventually leads to systolic dysfunction ➔ low tissue perfusion



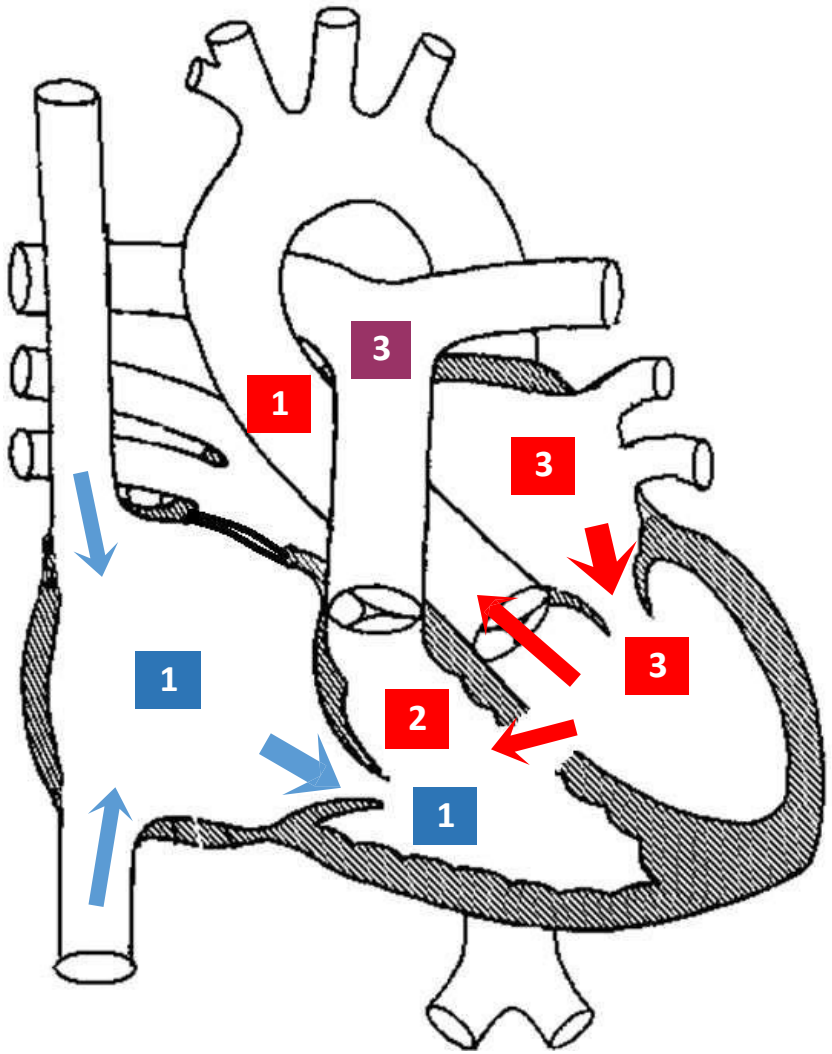


The **chronic** volume overload causes progressive **dilation** of the left ventricle. Initially, according to the **Frank–Starling law**, stretching of the ventricular muscle fibers **increases** contractility and improves output.

However, if the muscle is stretched **beyond its optimal length**, the **actin and myosin filaments no longer overlap effectively**. This leads to a **decline** in contractility. Over time, the child may develop **left ventricular dysfunction and symptoms of decompensated heart failure.**

Although heart failure patients may remain compensated for a period -able to maintain adequate cardiac output through mechanisms like tachycardia, increased sympathetic tone, fluid retention, and tachypnea- these mechanisms eventually fail in children how suffer from VSD. When they are no longer sufficient to meet metabolic demands, the child becomes **decompensated**, showing **symptoms** of **heart failure even at rest**.

Physiology of VSD ⟶ clinical picture



Right Side changes

During **systole**, when the shunt occurs, the **pulmonary valve is already open.** This means that the right ventricle does **not** experience a major volume overload. Instead, it experiences a **pressure load**, because the left ventricular systolic pressure is transmitted across the defect, so it hypertrophies.

As a result:

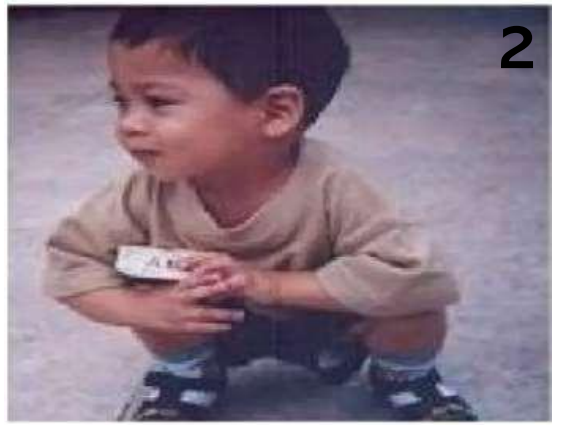
- **The left ventricle dilates** (due to volume overload during diastole)
- **The right ventricle hypertrophies** (due to pressure overload during systole), often becoming nearly as thick as the left ventricular wall.

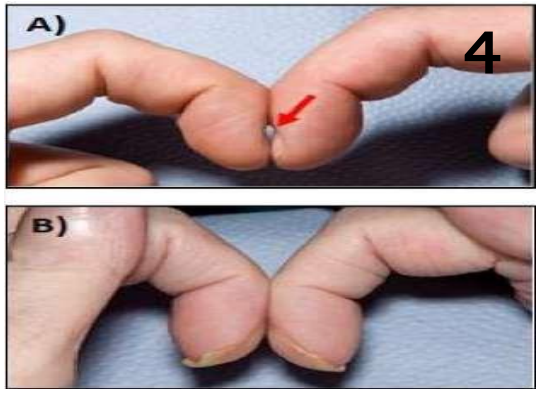
Case 2

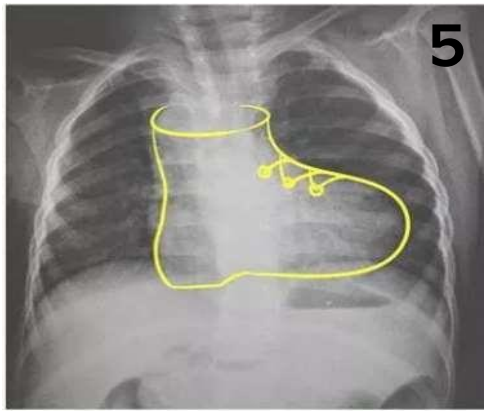
- Two year old child who has:
  - Cyanosis that increases when he plays and cries
  - Occasional squatting - specific body position (see 2)
  - Generally growing well with good weight
- Examination:
  - Oxygen saturation of 80% (Normal is >95%)
  - Plethoric (reddish or bluish) face (opposite of pale)
  - Good pulse and perfusion
  - Systolic heart murmur
  - Finger clubbing which is loss of the normal angle between the nail and the nail bed, so that when the nails of corresponding fingers are placed together, the normal diamond-shaped space is lost (see 3&4)

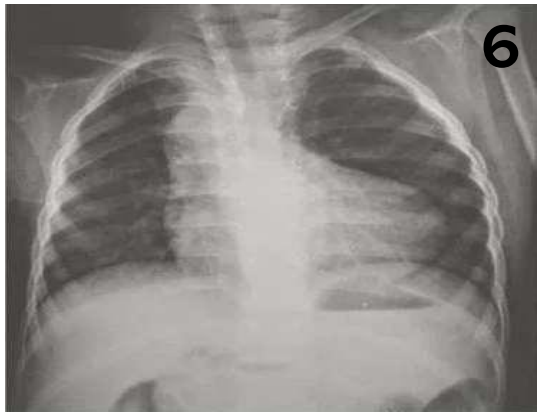
- Chest X ray showed (see5&6):
  - Heart not enlarged, but boot shaped, with dark lungs - the apex of the heart is uplifted, and the pulmonary artery appears small, with a concave shape.

- Hemoglobin level was 17 gm/dL (polycythemia) – Normal is 12-13 gm/dl





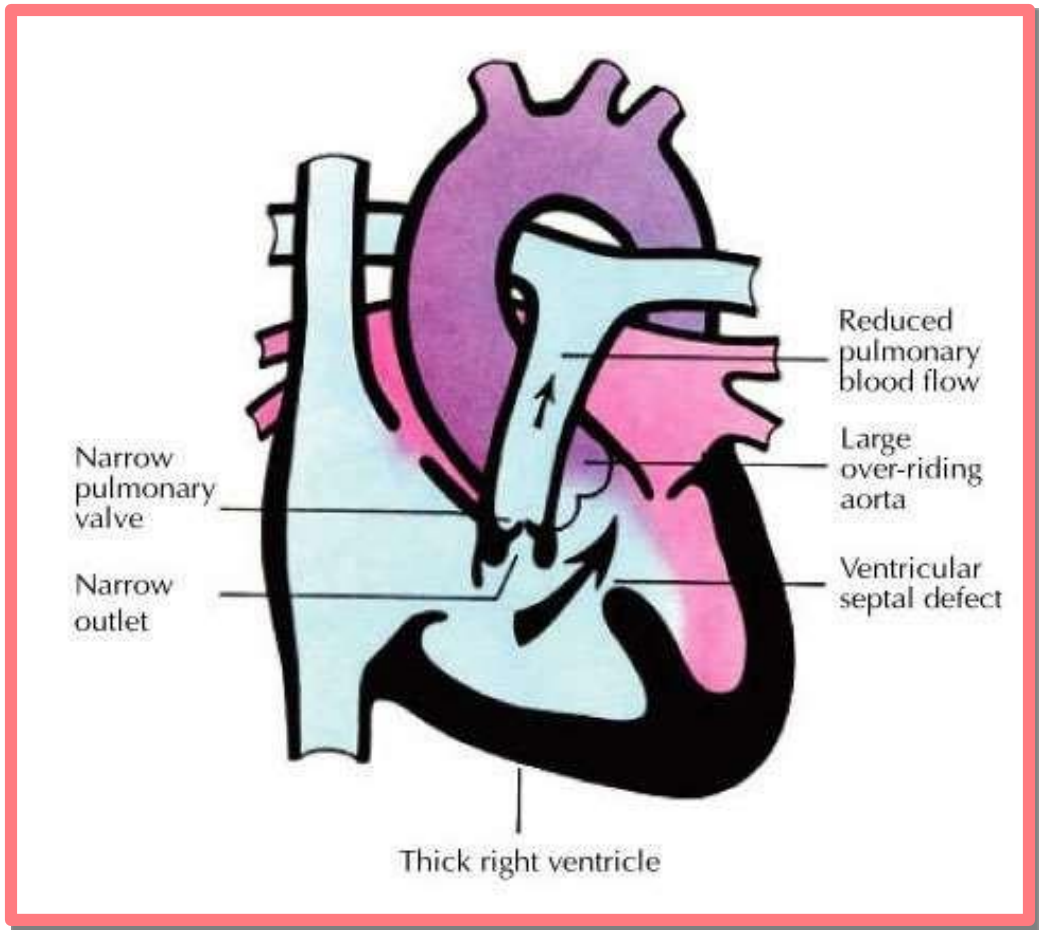




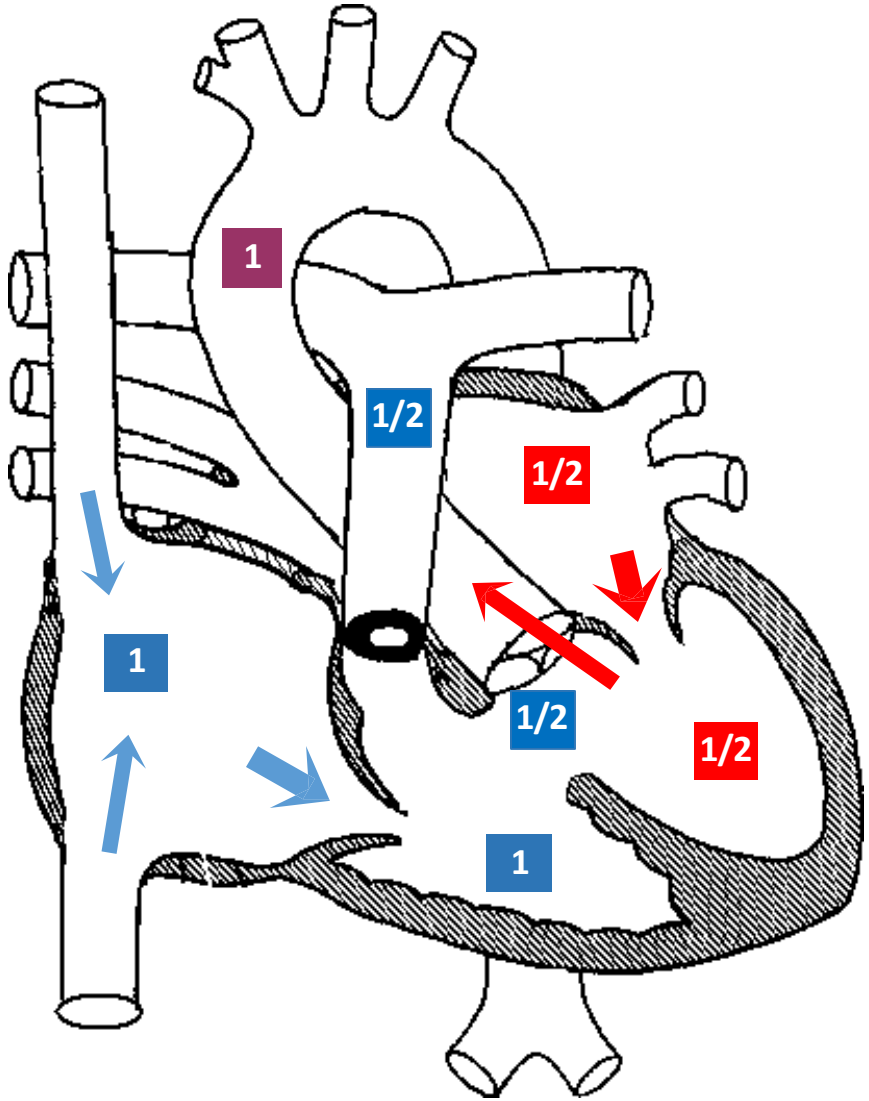
سبحان الله وبحمده عدد خلقه ورضا نفسه وزنة عرشه ومداد كلماته

Diagnosis: Tetralogy of Fallot

- Embryologic defect: anterior and rightward deviation of the "conal" septum. Normally, the conal septum should grow downward and align perfectly with the rest of the interventricular septum. However, in this condition, the conal septum deviates to the right instead of forming along the normal axis. Because it fails to align with the developing muscular septum, several structural abnormalities occur.
- Anatomical result:
  - Failure of the muscular ventricular septum to fuse with conal septum (VSD)
  - Sub-pulmonary and pulmonary stenosis (crowding beneath the pulmonary valve due to rightward deviation)
  - Aorta overrides the septum (the aorta is shifted rightward and appears to arise from both ventricles)
  - Secondary right ventricular hypertrophy (due to pressure overload on the right and equalized ventricular pressures)



Physiology of TOF ⟶ clinical picture



Although this patient also has a VSD, the clinical picture is very different from the first child because there is significant **pulmonary valve stenosis**. This stenosis is the key determinant of symptoms.

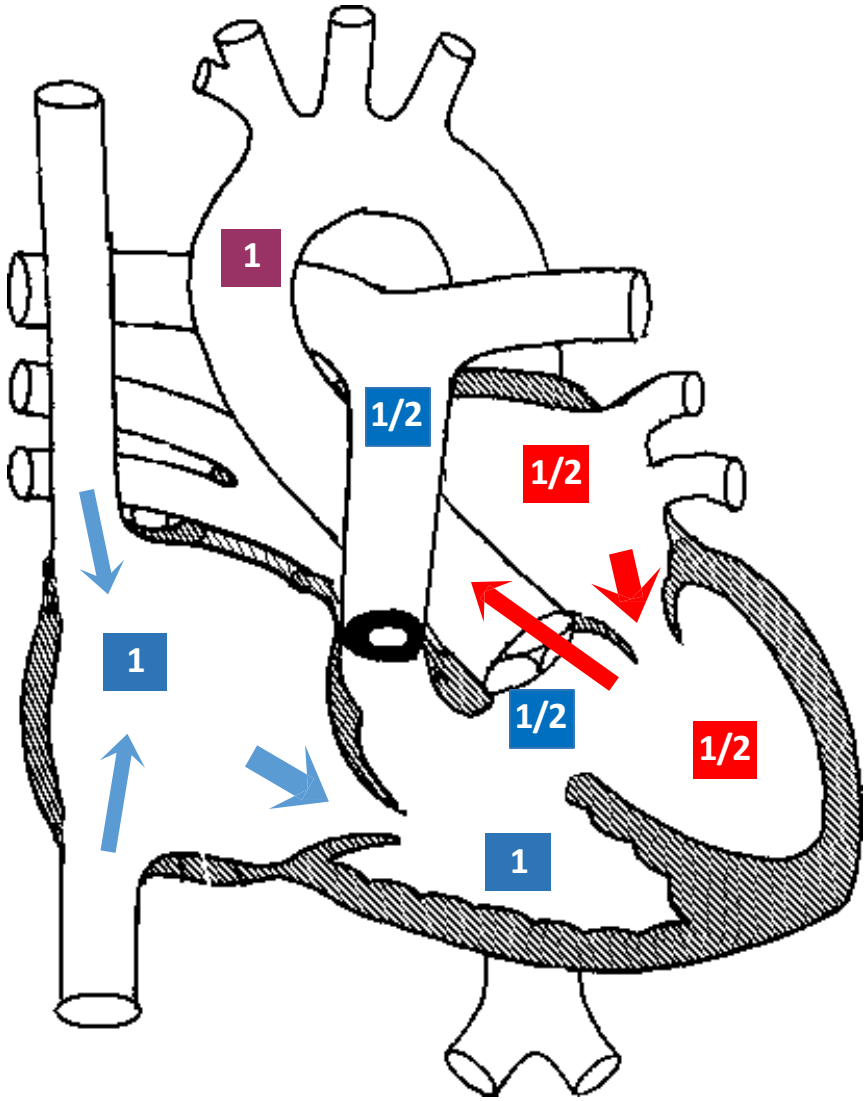
Assume the child has a cardiac output of 1 unit, which meets his basic metabolic needs. However, his oxygen saturation is **about 80%,** meaning his blood contains a mixture of **oxygenated and deoxygenated hemoglobin.**

Blood returns from the body to the right atrium fully **deoxygenated**, then moves to the right ventricle. With a **large** VSD, pressures between the two ventricles **equalize**, so shunting direction depends primarily on **relative resistance.** Because the right ventricular outflow tract is **stenotic**, **resistance** to blood flow into the lungs is much higher than the resistance into the aorta.

As a result:

- Only half of the cardiac output (from right ventricle) can pass through the **narrowed pulmonary** outflow tract to the **lungs**.
- The other half crosses the VSD into the **left ventricle and enters the aorta without** being oxygenated.

Physiology of TOF ⟶ clinical picture



The blood that does reach the lungs becomes oxygenated, returns to the left atrium, and mixes with the deoxygenated blood entering the left ventricle through the VSD. This mixture explains the **reduced oxygen saturation (80%).**

- If more blood is **deoxygenated**, saturation may be **70%.**
- If more reaches the **lungs,** saturation may be **90%.**
- The **degree of pulmonary stenosis** determines how much blood can be oxygenated.

This is why patients with Tetralogy of Fallot **do not have fixed oxygen saturation**; instead, it fluctuates depending on physiological conditions.

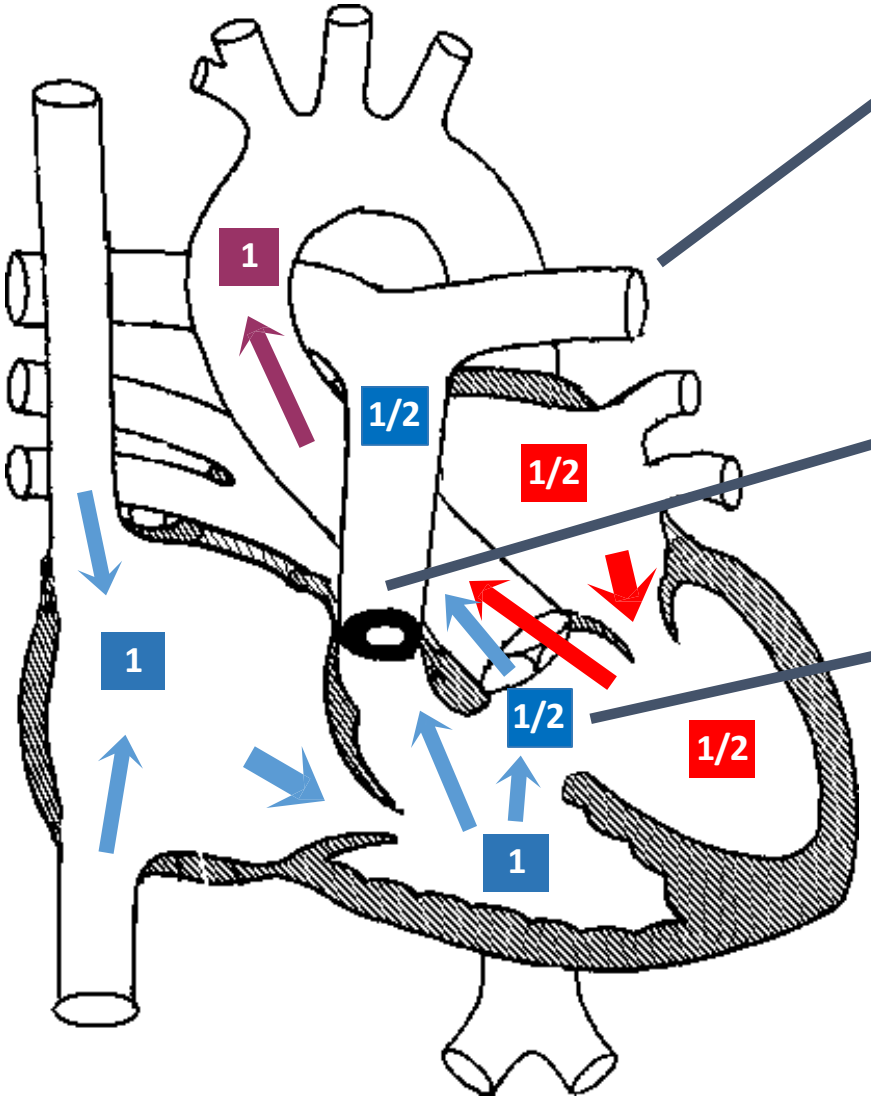
Why Oxygen Saturation Changes

Several factors influence the proportion of blood going to the lungs versus the systemic circulation:

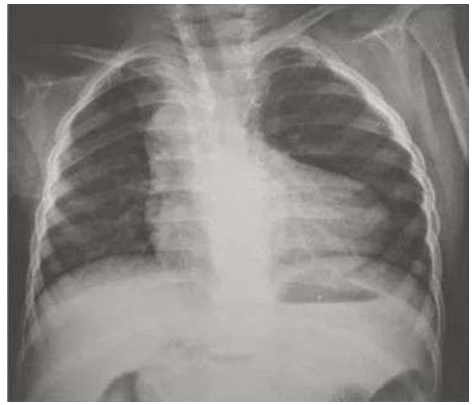
- Exercise or playing → systemic vasodilation → less resistance in systemic circulation → more right-to-left shunting → lower oxygen saturation
- Fever or illness → higher metabolic demand and lower systemic resistance → increased shunting → lower saturation
- Sleep → lowest metabolic demand → more blood can reach the lungs → highest saturation values

Physiology of TOF ⟶ clinical picture

The amount of pulmonary blood flow is lower than normal, so the lungs appears **black or dark** on the X-ray (less perfused)
Small pulmonary arteries because it doesn't receive adequate amounts of blood, so it doesn't grow adequately



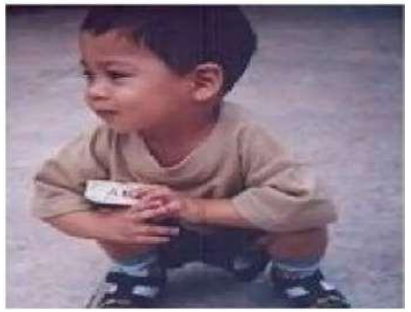
Decreased pulmonary blood flow ➔ No respiratory distress or rapid breathing, and darker lungs on the X ray, small pulmonary artery



Obstruction leads to turbulence of blood flow ➔ systolic murmur

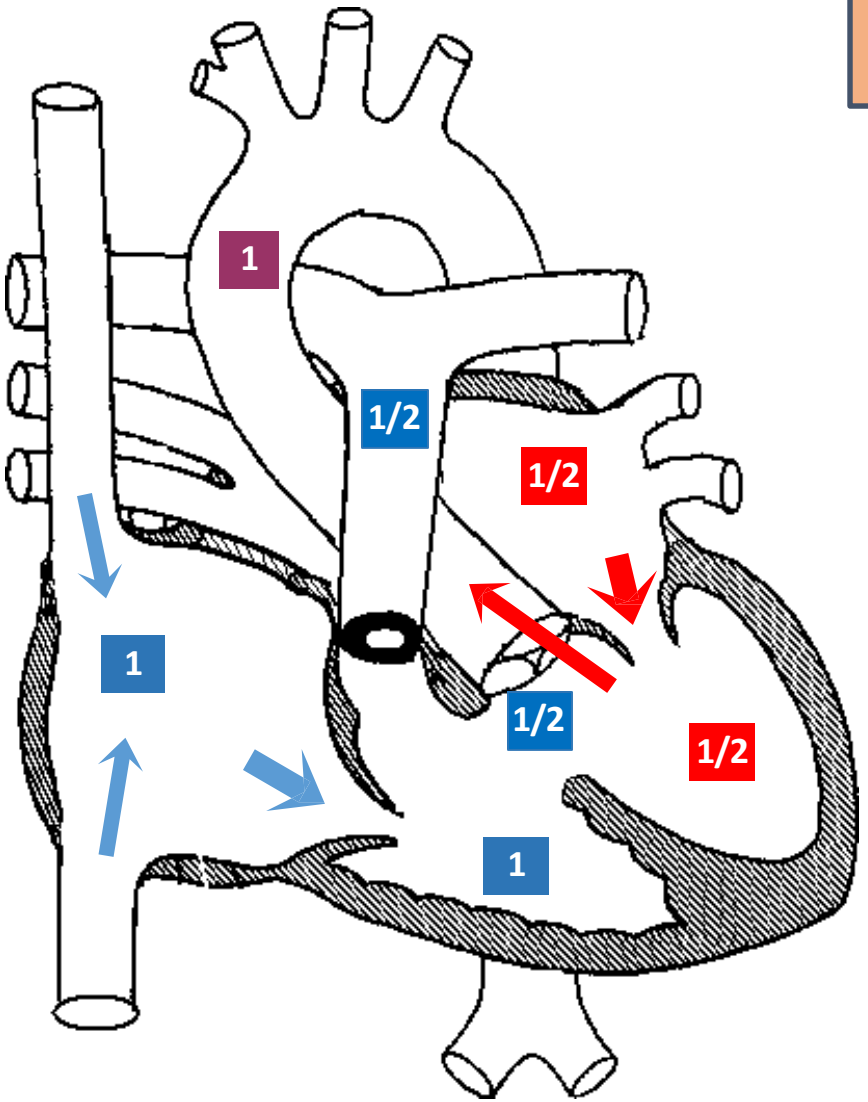
Shunting of deoxygenated blood to the aorta ➔ cyanosis ➔ polycythemia, clubbing

Squatting helps the child to decrease the right to left shunting ➔ alleviates the cyanotic episodes



Physiology of TOF ⟶ clinical picture

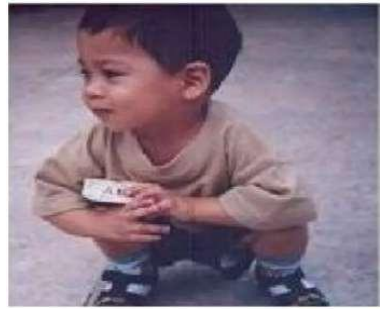
Shunting of deoxygenated blood to the aorta ➔ cyanosis ➔ polycythemia, clubbing



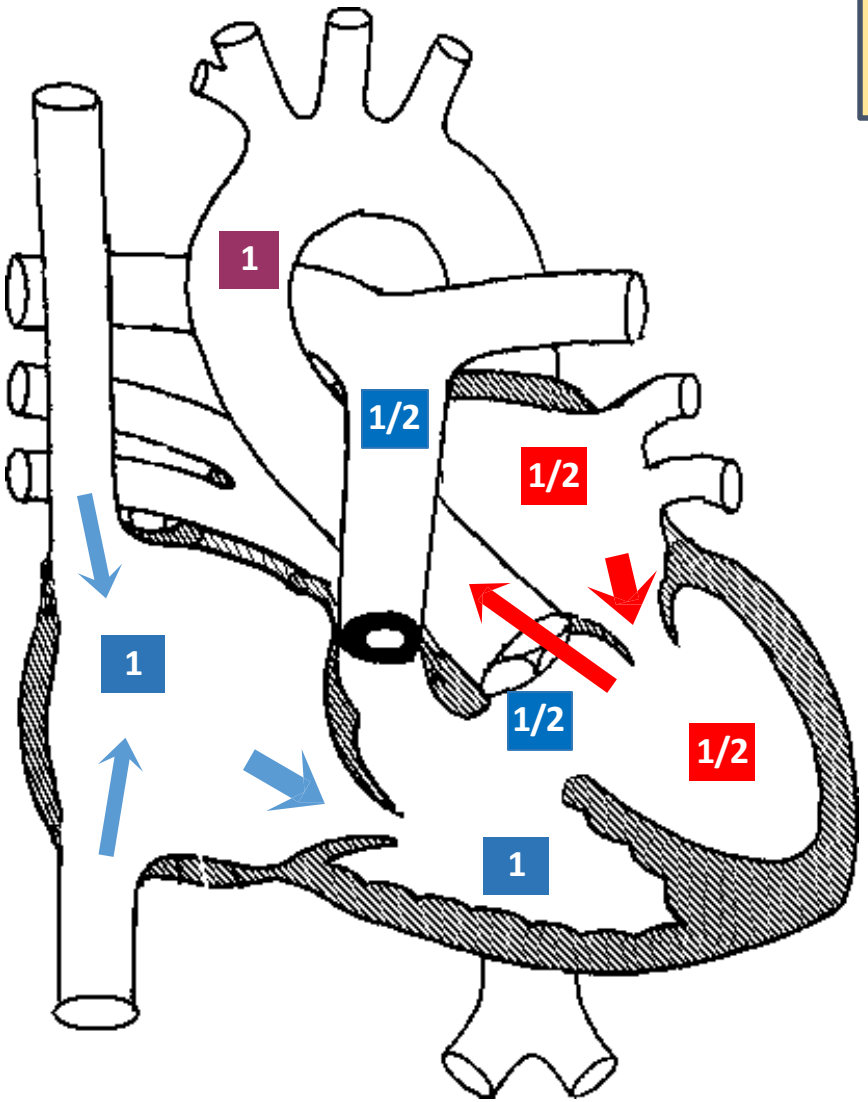
As a compensation of hypoxia, the bone marrow becomes more active and produces more RBCs. (Polycythemia) increases the **oxygen-carrying capacity**. For example, if a patient has a hemoglobin level of 17 g/dL with an oxygen saturation of 80%, he effectively has about 14 g of oxygenated hemoglobin and around 3 g that is not oxygenated - similar to a child who has 14 g/dL hemoglobin at 100% saturation. In contrast, having a hemoglobin concentration of around 10 g/dL is quite inadequate. Therefore, polycythemia is initially beneficial as a compensatory mechanism. However, once hemoglobin levels reach 20–21 g/dL, blood viscosity becomes excessively high, leading to increased resistance and reduced perfusion to vital organs.

(Finger Clubbing) Chronic cyanosis stimulates proliferation of tissues in an attempt to **increase hemoglobin production**. In infants, bone marrow is present throughout almost all bones, including **those beneath the nail bed**. This increased activity leads to nail-bed proliferation and eventually **clubbing**. Similar proliferative changes may occur in the **liver** and the **reticulo-endothelial** system due to increased hematopoietic activity. **Clubbing** can occur with any condition that leads to **chronic cyanosis**, such as **chronic lung diseases**.

Physiology of TOF ⟶ clinical picture



Squatting helps the child to decrease the right to left shunting ➔ alleviates the cyanotic episodes

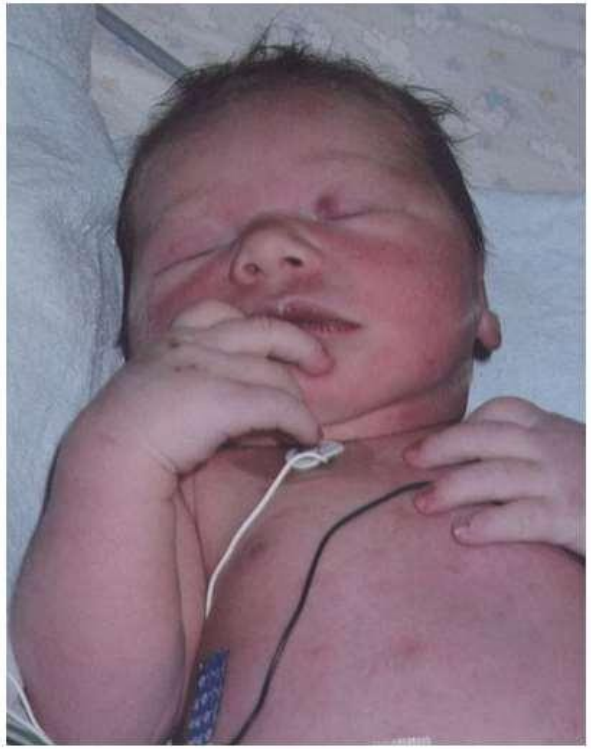


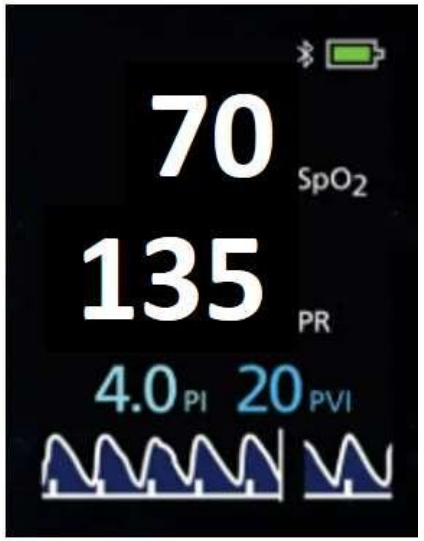
(Squatting) When the child is active or his oxygen saturation drops, he instinctively squats. Activity causes systemic vasodilation, which promotes more right-to-left shunting and further reduces oxygen saturation, causing symptoms. Squatting **compresses** the **femoral** arteries and **increases systemic vascular resistance**, **counteracting vasodilation**. This forces more blood toward the pulmonary circulation. In addition, squatting compresses the **abdomen and lower-extremity veins**, increasing **venous return to the heart**. This helps expand the right ventricle slightly and reduces the dynamic obstruction caused not only by the valve but also by the subvalvular muscle. It is also a kind of resting

In very young infants—such as a 2-month-old—with severe pulmonary stenosis, **squatting is not possible.** These infants may develop severe cyanosis, known as **hypercyanotic spells**, which are dangerous and can be fatal. Although most infants have only mild pulmonary stenosis that worsens with growth, some are born with critically severe obstruction. These infants appear cyanotic from birth and cannot compensate, making early surgical intervention necessary.

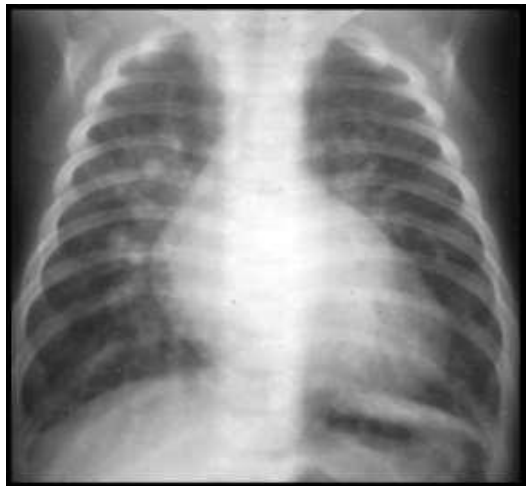
Case 3

- One day old newborn who has:
  - Severe cyanosis (looks blue)
  - No respiratory distress
- Examination:
  - Oxygen saturation of 70%
  - Good pulse and perfusion
  - No murmur. If he had a very loud murmur, we would suspect Tetralogy of Fallot with severe pulmonary stenosis — but in his case, he does not.
- Chest X ray showed:
  - Egg shaped heart, narrow mediastinum in contrast to the normal mediastinum where the pulmonary artery and the aorta next to each others.
- Cyanosis is getting worse with time



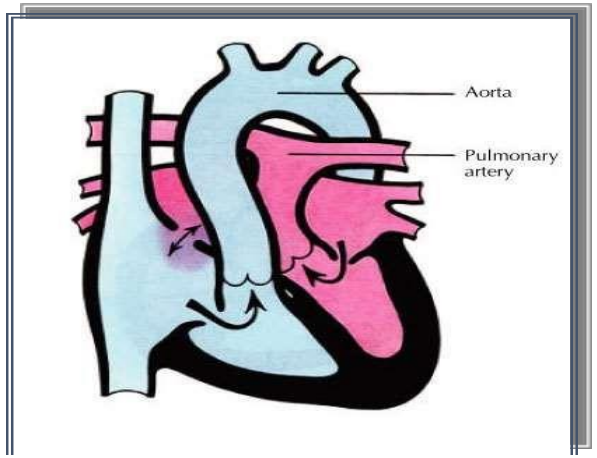


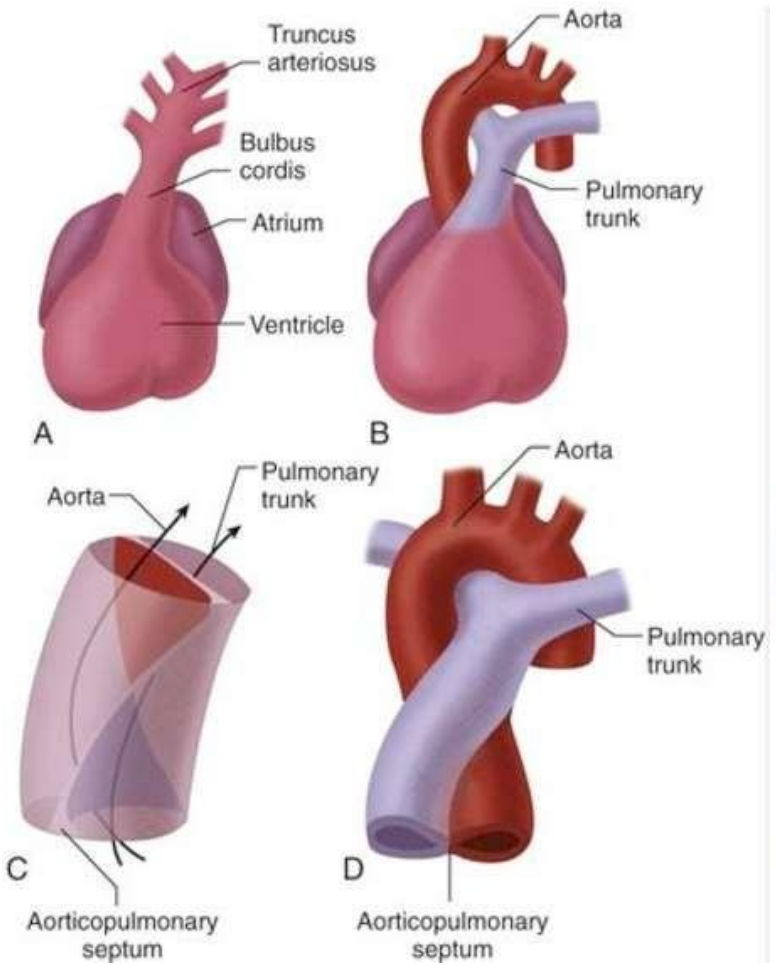
Normal heart rate for a neonate



Diagnosis: Transposition of the Great Arteries

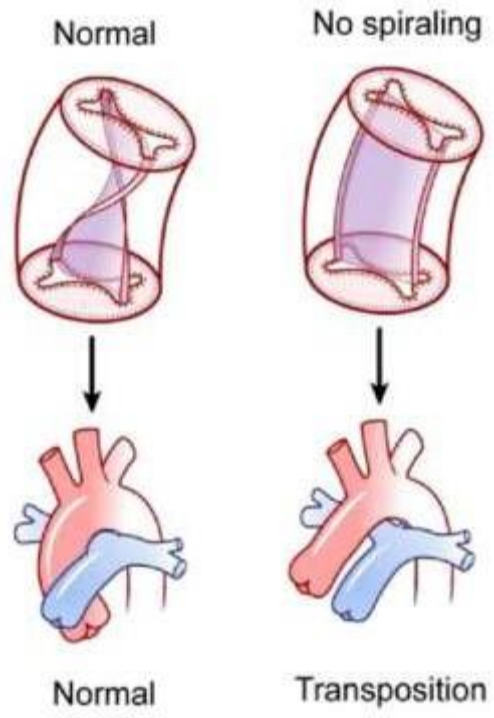
- Embryologic defect: Abnormal septation of the truncus arteriosus (a single vessel that will eventually divide to form the two major arteries: the pulmonary artery and the aorta)
- (failure of spiral septation) resulting in transposition of the great arteries
- **Anatomical result:**
  - **Aorta arises from the right ventricle**
  - **Pulmonary artery arises from the left ventricle**



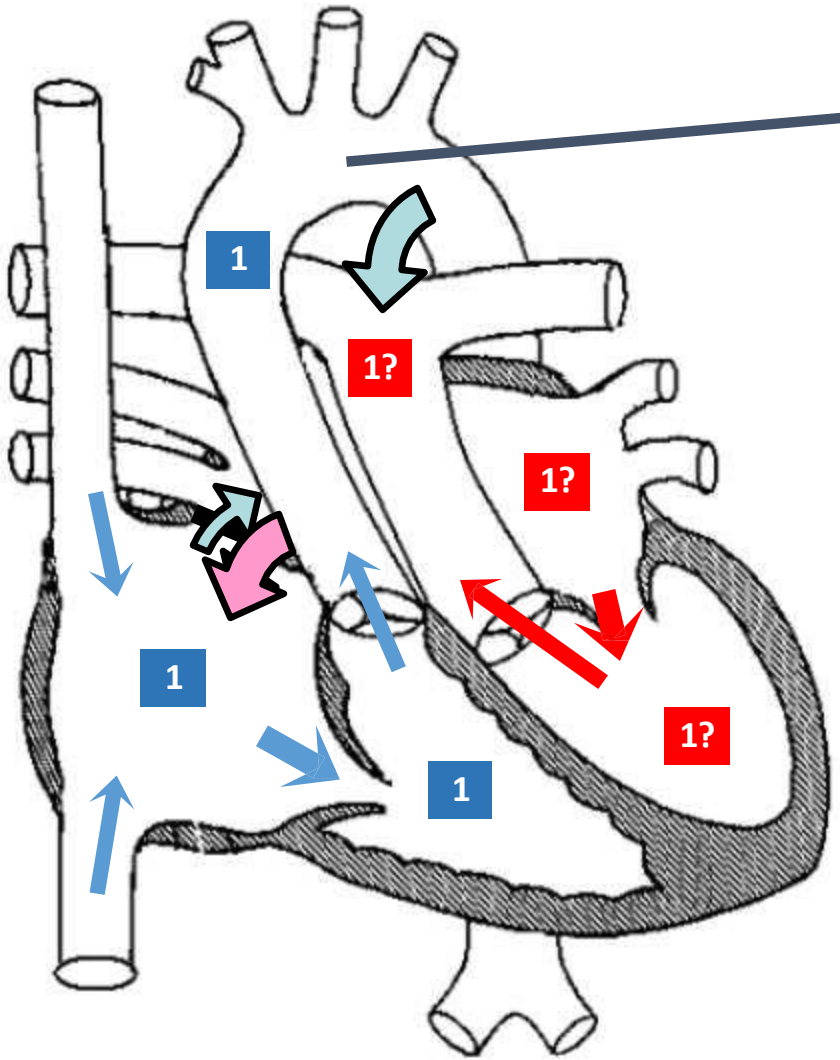


Normal spiraling of AP septum

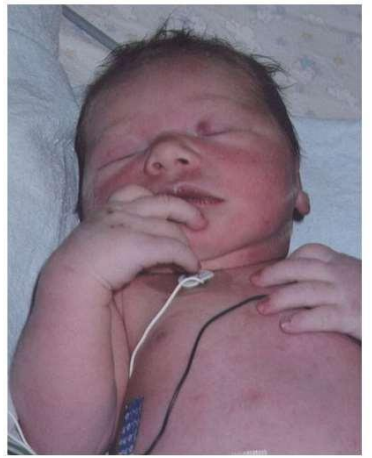
Normal vs abnormal spiraling

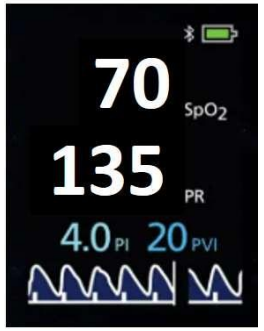


Physiology of TGA ⟶ clinical picture



The two circulations are parallel, systemic and pulmonary blood re-circulate, resulting is severe cyanosis





For survival, atrial level mixing is mandatory (bidirectional flow)

Allows for some oxygenated blood to body, and deoxygenated blood to lung

Emergency balloon septostomy can be life-saving

Additional shunting via patent ductus arteriosus can also help the baby survive until surgical repair is done

Prostaglandin infusion can be life-saving

Physiology of TGA ⟶ clinical picture

In this abnormal arrangement:

Systemic (Aortic) Circuit

Deoxygenated blood:

Right atrium → right ventricle → aorta → systemic circulation → returns even more deoxygenated → right atrium again

Each cycle makes the blood progressively more deoxygenated, so the baby can only survive minutes because the oxygen content rapidly drops - from 80% → falling to 0% as hemoglobin becomes fully deoxygenated.

Pulmonary Circuit

Oxygenated blood:

Left atrium → left ventricle → pulmonary artery → lungs → left atrium again

This creates an isolated oxygenated circuit that keeps recirculating in the lungs with no effect on the body.

Physiology of TGA ⟶ clinical picture

For survival, oxygenated blood must reach the aorta, and deoxygenated blood must reach the lungs. This requires shunts that allow mixing. The most important are:

1. **Atrial-Level Shunting (PFO or ASD)**

The foramen ovale (PFO) -normally- is essential for fetal survival because:

- The right ventricle does not send blood to the lungs
- The left ventricle does not receive pulmonary blood

Therefore, fetal circulation depends on the PFO to allow blood to pass between the atria. If it remains open after birth, it allows bidirectional mixing, helping the baby survive.

2. Ductus Arteriosus (PDA)

The PDA connects:

Aorta → pulmonary artery (Because aortic pressure is higher, even though it arises from the right ventricle; because pressure depends on systemic resistance, not on the ventricle itself.)

Physiology of TGA ⟶ clinical picture

Through the PDA:
Deoxygenated aortic blood → pulmonary artery → lungs → left atrium (This increases pulmonary flow and improves oxygenation).
The ASD/PFO then becomes:
Left atrium (higher pressure) → right atrium
Providing oxygenated blood → systemic circulation.

Atrial mixing volumes are small (low-pressure-difference btw. them) so PDA provides a larger, more unidirectional flow:
Aorta → pulmonary (deoxygenated to lungs) → oxygenated → left atrium → increase pressure and help more unidirectional flow → ASD/PFO → right atrium & ventricle → systemic (oxygenated to body)

Which is better?
PFO is required (must have)
PDA alone is not enough
ASD alone might be enough but having **PDA** would be better.

Physiology of TGA ⟶ clinical picture

What Happens After Birth

After birth, two things naturally close the PDA:

- Cutting the umbilical cord → ↓ prostaglandins
- First breaths → ↑ oxygen saturation

This causes PDA constriction → ligaments arteriosum within few days.

If PDA closes and the ASD/PFO is small:

→ Mixing becomes severely limited

→ Saturation drops dangerously

→ Baby becomes critically ill

Emergency management:

Give IV prostaglandin to make the PDA relaxed and open:

Aorta → pulmonary artery → lungs → left atrium → ASD/PFO → right atrium → systemic circulation

This restores oxygen delivery to the body.

Physiology of TGA ⟶ clinical picture

Why PFO cannot be relied on

The PFO is just a flap, and once left atrial pressure rises after birth:
Left atrium pressure rises → closes the flap
So mixing becomes restricted within days, making the baby very sick.

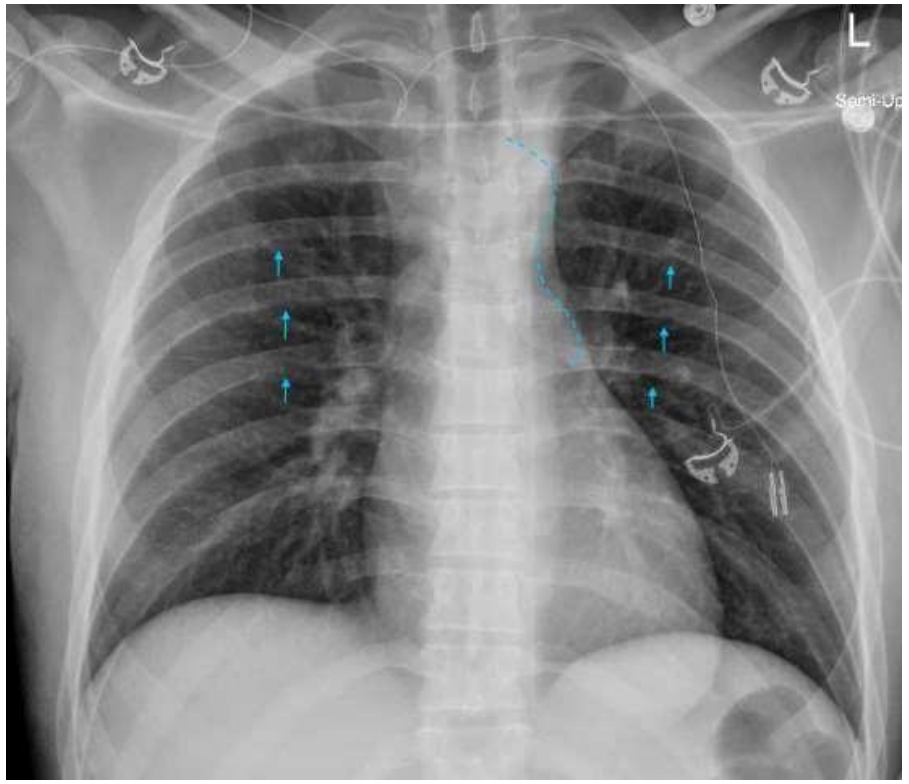
Emergency Septostomy (Balloon Atrial Septostomy)

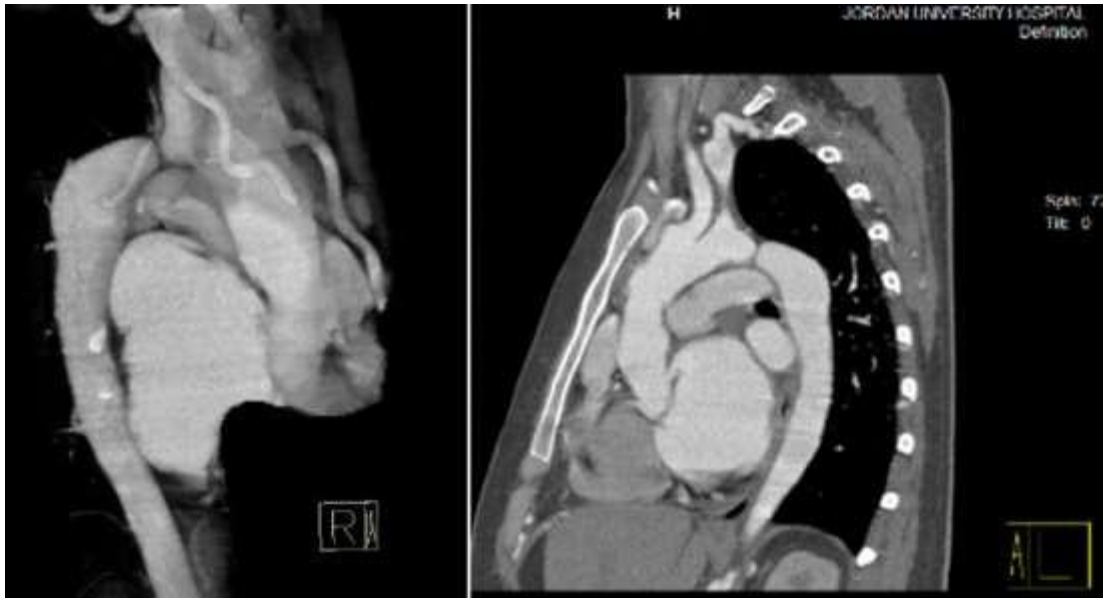
To reopen the atrial septum:
Femoral vein → right atrium → PFO/ASD → left atrium
Inflate balloon → pull back forcefully → tear septum → create a large opening

This procedure buys days to weeks of stability until corrective surgery is performed.

Case 4

- 12 year old child complains of headache, and occasional chest pain with exercise.
- He reports cramps of his leg when he walks for long distances (claudication).
- Examined by school nurse, and was found to have blood pressure of 160/100
- Other findings on examination:
  - Good radial pulse, but poor femoral pulse
  - BP in the leg measured 110/70
  - Cardiac palpation showed strong apical impulse (apical heave)
  - ECG showed left ventricular hypertrophy
- Chest X ray showed:
  - Notched ribs

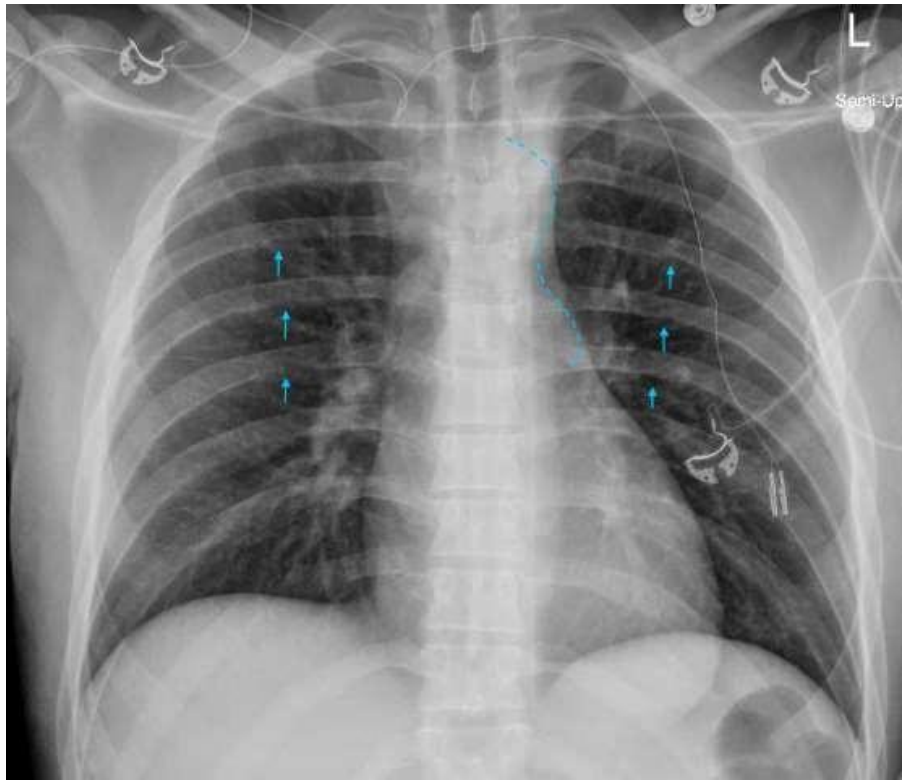


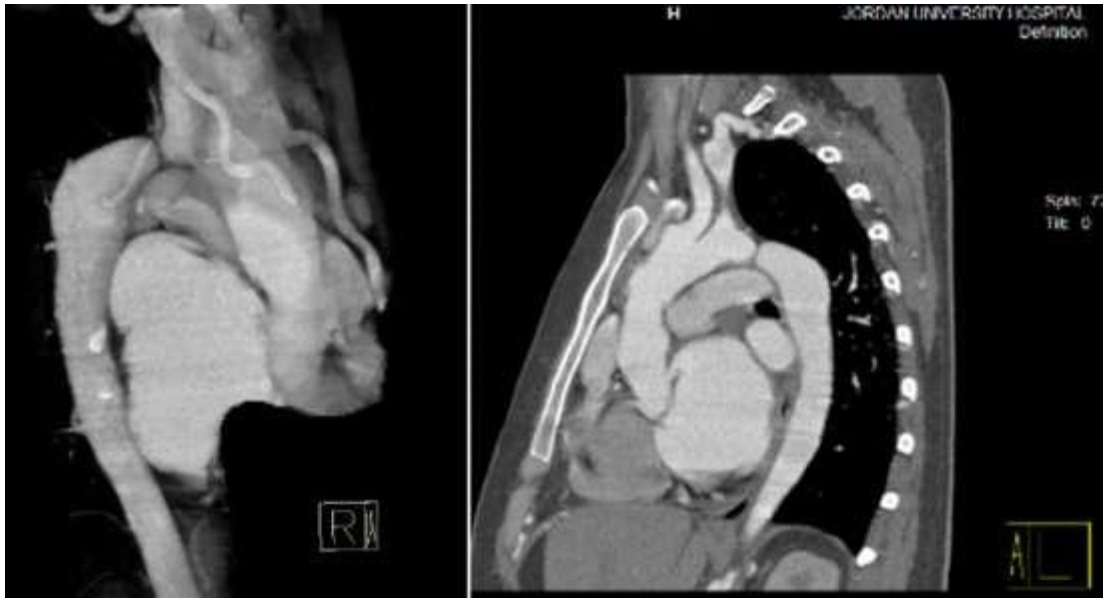


The heart size is **not** significantly enlarged, and the lungs appear **normal**—neither plethoric nor hypoperfused.

However, there is **notching of several ribs**, with irregularities along the inferior borders of some of them.

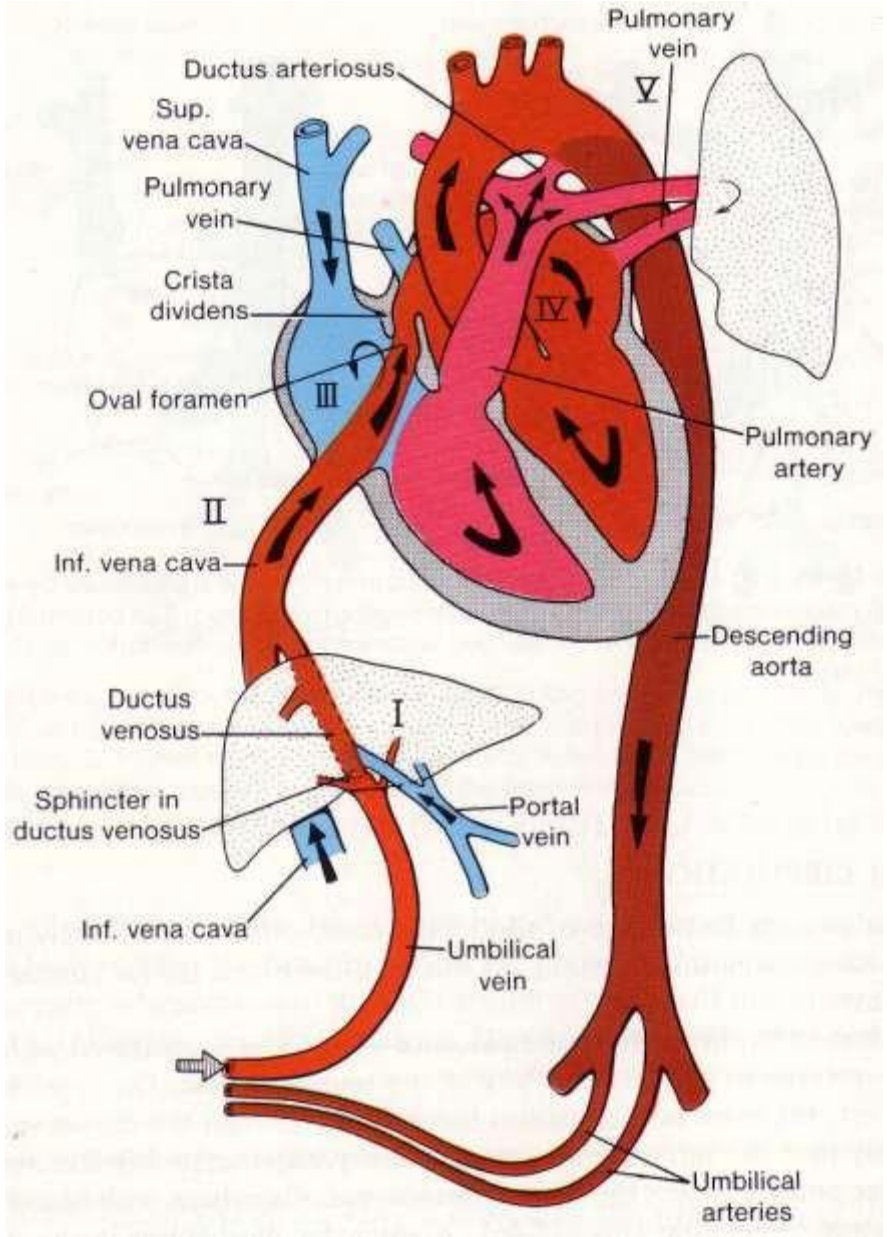
A CT scan demonstrates a significant obstruction in the distal aortic arch, which is consistent with a diagnosis of **coarctation of the aorta.**

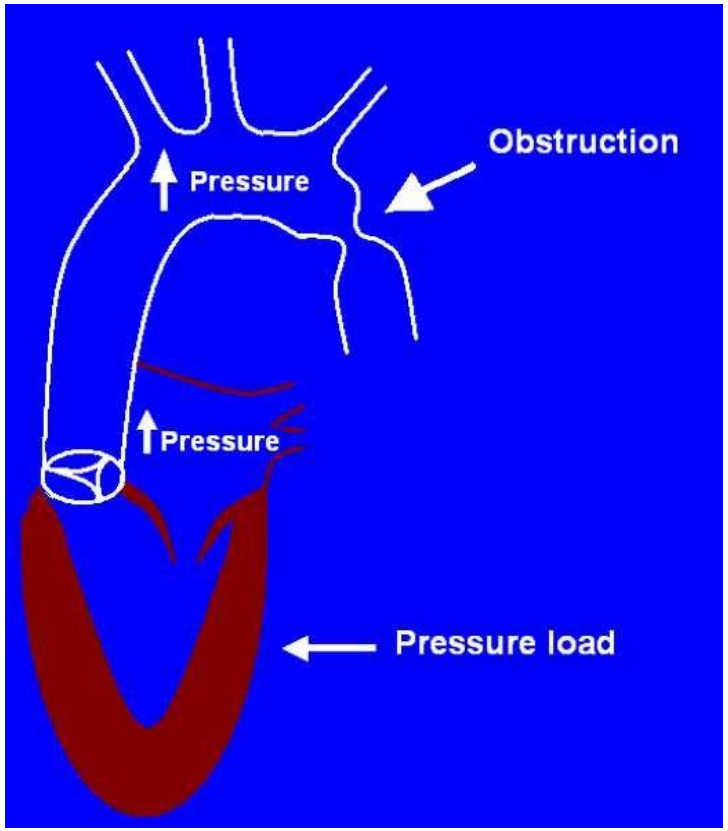




Diagnosis: Coarctation of Aorta

- Embryologic defect: decreased flow across the distal aortic arch during fetal life (most accepted theory)
- Anatomical result:
  - Narrowing of the distal arch (Isthmus), usually distal to the left subclavian artery





Diagnosis: Coarctation of Aorta

We have an obstruction of the aorta just distal to the left subclavian artery

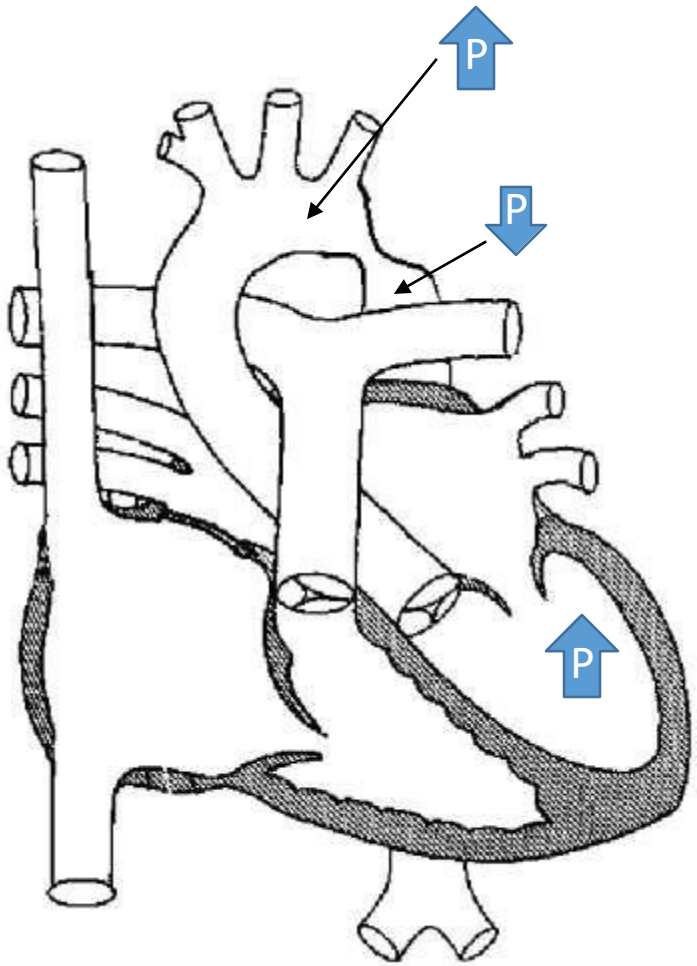
Fetal Circulation

• In fetal life, all cardiac structures are present: left atrium, left ventricle, right atrium, right ventricle, pulmonary arteries, and the aorta.
• The heart develops within the first two months of gestation and subsequently grows in size as pregnancy progresses.
• Fetal structures that guide blood flow include:
  - Ductus arteriosus (DA): blood flows from the right ventricle → pulmonary artery → descending aorta.
  - Foramen ovale (FO): blood flows from the right atrium → left atrium → left ventricle to fill the systemic circulation.
• This setup constitutes normal fetal circulation.

Pathophysiology of Coarctation

• In some fetuses, for various reasons, there is reduced blood flow from the left ventricle into the aortic arch.
• Normally, the left ventricle might supply ~50% of the cardiac output to the aorta; in these cases, it may only supply ~30%, with the majority (70%) supplied by the right ventricle via the ductus arteriosus.
• Because the brain requires a substantial portion of blood during development, there is insufficient flow across the aortic arch to the descending aorta.
• Low flow across the arch limits its growth, particularly in the segment distal to the left subclavian artery.
• This results in narrowing of the aortic arch, which is the anatomical basis of coarctation of the aorta.

Physiology of TGA ⟶ clinical picture

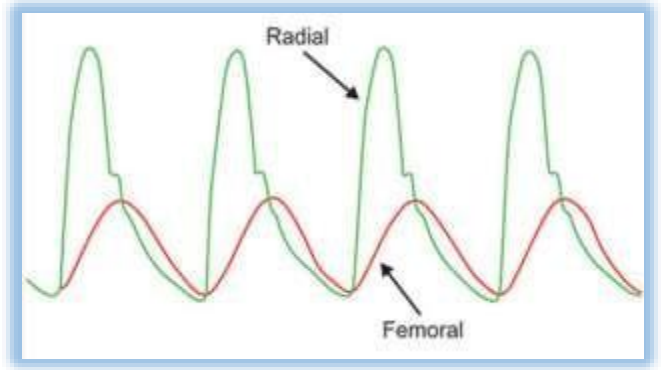


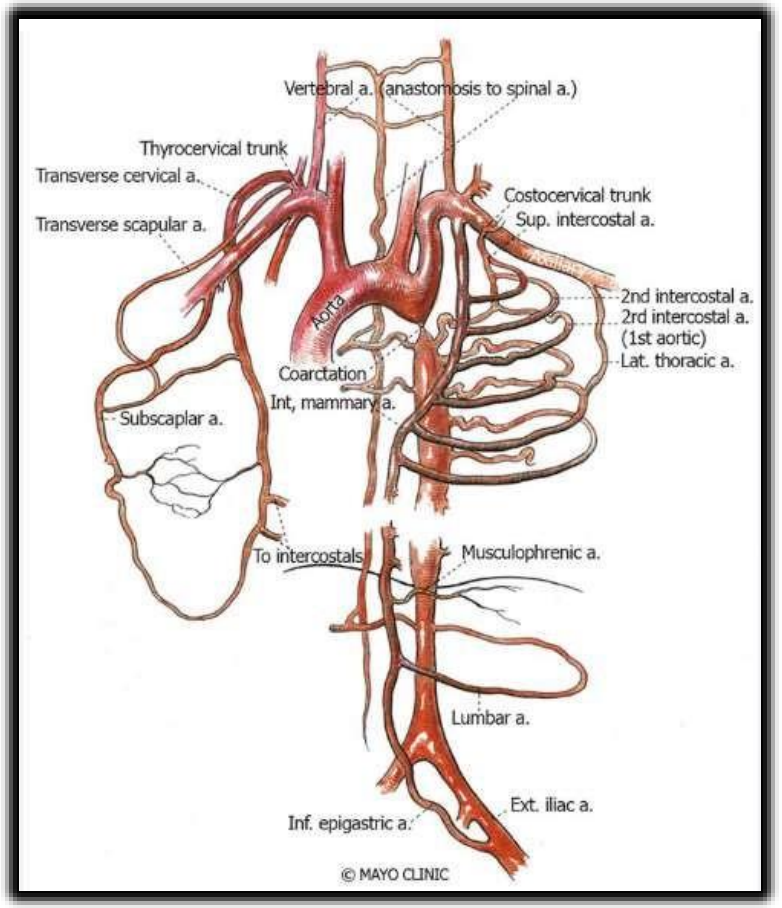
Hypertension in upper limbs, with normal or low pressure in lower limb

Left ventricle becomes hypertrophic to be able to handle the high pressure➔ increased myocardial oxygen demand ➔ susceptible for exertional ischemia and exertional angina

Collateral circulation develops slowly, involving the intercostal arteries ➔ ribs are notched

Lower limb blood supply may get compromised with exercise leading to muscle pain and cramping (claudications)





Physiology of TGA ⟶ clinical picture

Consequences of Coarctation of the Aorta

- **Blood Pressure Changes**

- Proximal to the narrowing: Pressure is elevated, resulting in hypertension in the upper limbs.
- Distal to the narrowing: Pressure is normal or low, so the lower limbs may be hypotensive.

- **Left Ventricular Hypertrophy (LVH)**

The left ventricle faces high afterload due to the proximal hypertension → it "trains" to pump against higher pressure. This causes muscle thickening and strengthening, which is why the apical impulse is very strong. Over time, excessive LVH can lead to diastolic dysfunction:

- Elevated left atrial pressure
- Pulmonary congestion
- Symptoms on exertion such as dyspnea
- Exertional chest pain: Thickened myocardium has increased oxygen demand, which may exceed coronary blood flow despite normal coronary arteries. This explains why some young patients with LVH develop anginal pain during exercise (the cause is LVH but NOT coronary artery disease).

حسبي الله لا إله إلّا هو عليه توكّلت وهو ربّ العرش العظيم

Physiology of TGA ⟶ clinical picture

- **Rib Notching and Collateral Circulation**

In coarctation, blood flow to the lower body is reduced, but organs like the kidneys and abdominal structures still require adequate perfusion. The body compensates via collateral circulation:

- Intercostal arteries (from descending aorta)
- Internal mammary arteries (branches from subclavian arteries)

These vessels connect to supply the lower body.

High flow through intercostal arteries → vessels become engorged and enlarged, causing rib notching on imaging.

- **Claudication**

Although collateral vessels supply the lower limbs at rest, they are insufficient during exercise, leading to fatigue or pain in the legs (claudication).

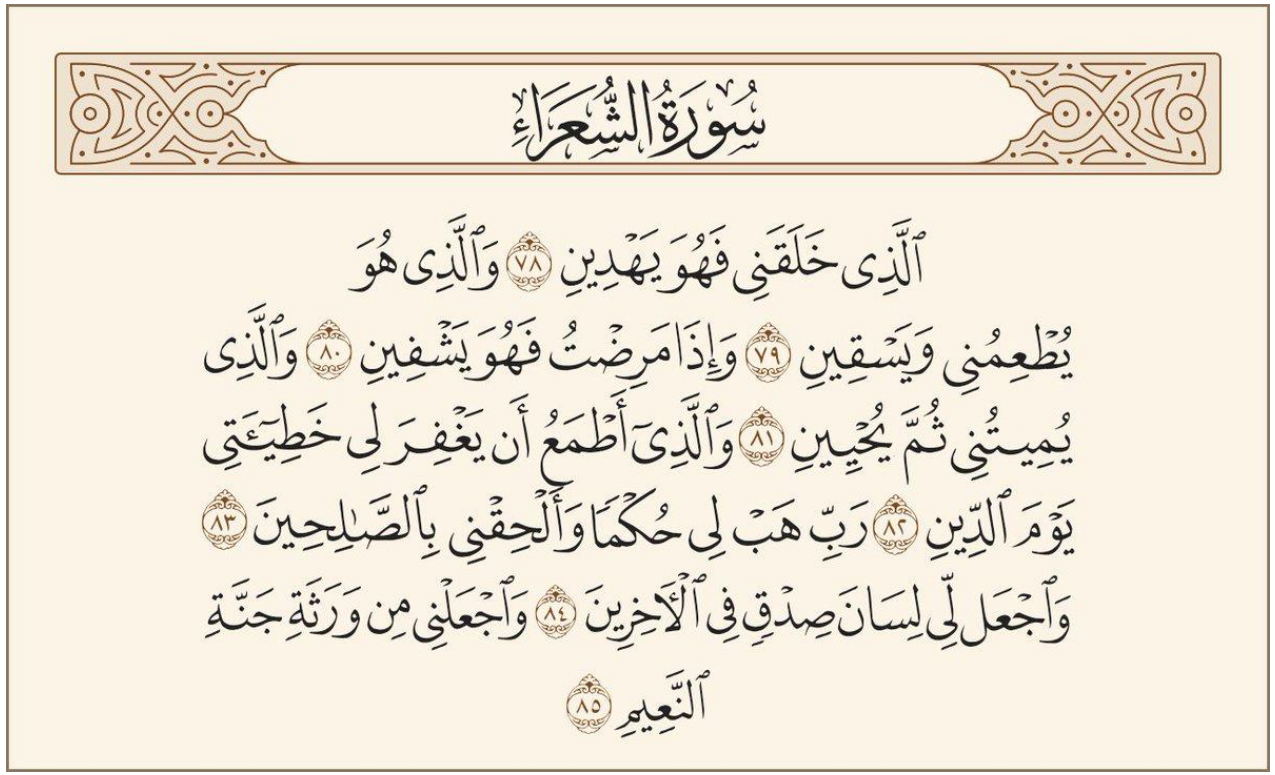
END

This file was prepared quickly, I hope it was clear and easy to follow

CLINICAL QUIZ LECTURE 2

رسالة من الفريق العلمي

لا إله إلا الله وحده لا شريك له ، له الملك و له الحمد و هو على كل شيء قدير


سُورَةُ الشُّعَرَاءِ

ٱلَّذِى خَلَقَنِى فَهُوَ يَهۡدِينِ ﴿٧٨﴾ وَٱلَّذِى هُوَ
يُطۡعِمُنِى وَيَسۡقِينِ ﴿٧٩﴾ وَإِذَا مَرِضۡتُ فَهُوَ يَشۡفِينِ ﴿٨٠﴾ وَٱلَّذِى
يُمِيتُنِى ثُمَّ يُحۡيِينِ ﴿٨١﴾ وَٱلَّذِىٓ أَطۡمَعُ أَن يَغۡفِرَ لِى خَطِيٓـَٔتِى
يَوۡمَ ٱلدِّينِ ﴿٨٢﴾ رَبِّ هَبۡ لِى حُكۡمًا وَأَلۡحِقۡنِى بِٱلصَّٰلِحِينَ ﴿٨٣﴾
وَٱجۡعَل لِّى لِسَانَ صِدۡقٍ فِى ٱلۡأٓخِرِينَ ﴿٨٤﴾ وَٱجۡعَلۡنِى مِن وَرَثَةِ جَنَّةِ
ٱلنَّعِيمِ ﴿٨٥﴾

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Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			