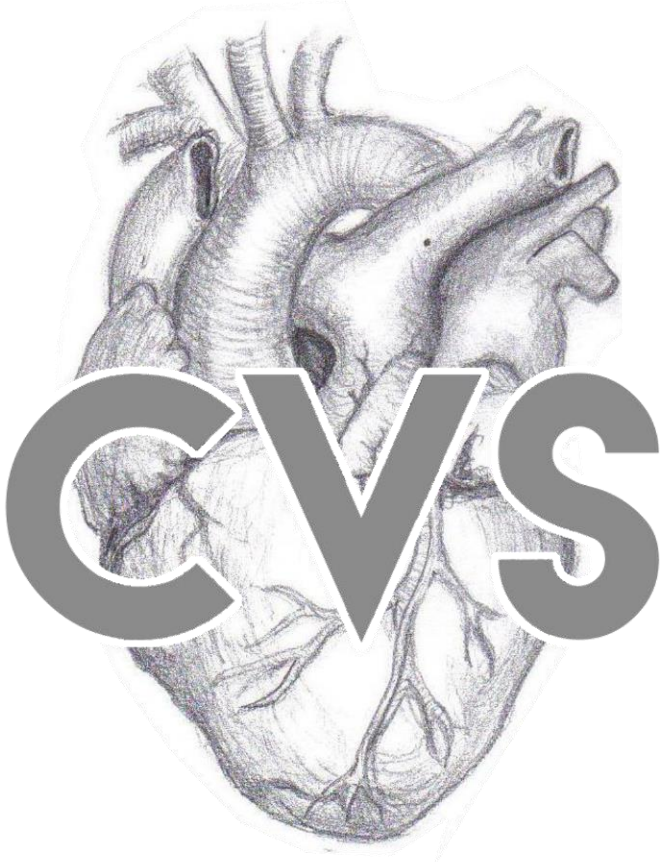

CVS

ANATOMY / HISTOLOGY

☑ Sheet ○ Slide ○ Handout

Number

8

Subject

Fetal circulation &Cardiac Anomalies

Done By

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Corrected by

.....

Doctor

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Date: 00/00/2016

Price:

Fetal circulation

Figure 1

In comparison with an adult the blood circulate in the fetus differently; the major difference is that there is no pulmonary circulation in an embryo.

from that point we can start our great lecture ☺

if there's no pulmonary circulation from where will the fetus get oxygenated blood?

-from the placenta.

why there's no pulmonary circulation?

- the lungs are collapsed (along with the pulmonary vessels)>> collapsed vessels implies very high resistance to the extent that the blood won't flow.

Fetal circulation :

First. The placental blood will pass to the left umbilical vein>> which opens in the Lt branch of portal vein >> that opens in ductus venosus.

Second. This blood will bypass the liver through ductus venosus and reach the IVC

Third. From the IVC the blood will reach the Rt atrium

Fourth. The blood will pass from Rt atrium to the Lt atrium through foramen oval (present at interatrial septum)

Fifth. From the Lt atrium blood will pass to the Lt ventricle then the aorta (through the aortic valve) >> to the upper half of the body (head , neck and brain) .

Sixth. The Rt atrium will also receive deoxygenated blood from SVC from the upper part of the body (less oxygenated than the blood coming from the placenta) this blood will be pumped into the Rt ventricle not the Lt atrium>> from the Rt. Ventricle it will be pumped into the pulmonary trunk >> the blood will not go to the lung –as they are collapsed- instead it will go to descending aorta through ductus arteriosus (which connect the

pulmonary trunk with the end of the aortic arch)>> blood will go to lower part of the body >> then back to the placenta.
by this we can conclude that the Rt. Atrium receive 2 types of blood :

- ✓ Highly oxygenated from IVC, coming from the placenta.
- ✓ Poorly oxygenated (deoxygenated) , coming from upper half of the body.

Mixing of these 2 types of blood occur minimally at some areas mentioned below >> when this mixing occur the highly oxygenated blood will be less oxygenated, as follows:

- ✓ if we assumed that the blood that come from the placenta is 80% oxygenated
- ✓ at the ductus venosus some blood mixing occur (at the level of the liver and its Lt. portal vein).
- ✓ Also mixing occur at the level of IVC>> the oxygenation percentage become 67%
- ✓ Also blood is mixed at the level of the Rt atrium + Lt atrium.
- ✓ Eventually, placental blood will reach the Lt. Atrium with oxygenation % = 62% (less oxygenated than pure placental blood , but still more oxygenated than blood coming to Rt. Atrium from the upper half of the body)

We can notice that there are 3 special shunts in the fetus :

- ✓ Ductus venosus : oxygenated blood of the placenta bypass the Liver only little amount of the blood will pass to the liver.
- ✓ Foramen ovale : oxygenated blood of the placenta bypass the Rt. Ventricle (blood will pass from the RA to the LA as the RA pressure is higher than LA because the LA normally receive its blood from the lungs through pulmonary veins but as the lungs are collapsed in the fetus it receives nth from it)
- ✓ Ductus arteriosus : blood is shunted from the pulmonary trunk to the aorta.

When will these shunts close ?

Both ductus venosus and foramen ovale will be closed when the fetus is born. while ductus arteriosus will close after few hours (or few days) of birth.

How these foramens (shunts) are closed?

To answer such a Q , we must know first what happens after birth (right after the first “waaaah=breathe” of the baby)

1. As the baby takes his first breathe >> his lungs will expand >> the arteries inside the lungs expand>> the blood flow to the lungs from the RV as the pulmonary resistance is massively reduced (no collapsed vessels or lungs) >>the pressure of the pulmonary trunk will fall (as no blood is collected there waiting to be shunted vis ductus arteriosus) also the pressure will fall in the Rt ventricle and Rt atrium (simply because the blood will flow)
2. On the other hand , on the left side , the Lt. atrium and the Lt. ventricle pressure will increase , **why?**
   -look at figure 2
   -the LA and the LV pressures are dependent on the total resistance that the heart pumps against (the after load)
   our body’s different vascular beds (that the heart will pump against) are connected to each others in a parallel way على التوازي and recall from physics that to summate these resistance , we apply this law :

$$\frac{1}{Total\ resistance} = \frac{1}{Renal\ bed\ resistance} + \frac{1}{hepatic\ bed\ resistance} + \frac{1}{placental\ bed\ resistance} ..$$

   also one of these vascular beds is the pulmonary vascular bed that has now lower resistance.
   depending on these findings when we remove the placenta after birth >> the total resistance will increase (as 1/total resistance will decrease depending on the previous law) –Try it ur-self with real numbers. >> when total resistance increases >> the pressure of both LA and LV increases,

   To sum up :
   After birth the systemic resistance will increase because of two changes: the placental resistance will be zero (removed) and the pulmonary resistance will decease massively. When systemic R increase > left heart pressure increases.

by now we can answer our Q :

how foramen ovale is closed ?

After birth the LA pressure increases and becomes more than RA pressure>> the blood flow from Left to right will let the septum premium and secundum fuse together >> foramen ovale is closed.

How ductus Arteriosus closes?

after birth the Aorta pressure become higher than the Pulmonary trunk pressure (because the Aorta is linked to the Lt-side-high-pressure , while the pulmonary trunk is linked to the Rt-side-low-pressure area) then the blood will pass from the aorta to the pulmonary trunk through this duct >> the problem here is that the pulmonary trunk can not tolerate high pressure blood (put on that : that this blood is also deoxygenated) >> to protect the pulmonary trunk >> the ductus Arteriosus will atrophies and become ligamentum arteriosus.

recall from previous lectures , this ligament is between Lt. pulmonary artery and the end of arch of the aorta.

why ductus venosus atrophies ?

obviously >> no placenta >> no blood flow to the Lt. umbilical vein >> no need for “bypass” the liver >> no blood will flow in this duct >>it atrophies >>this ductus will also become ligmentum (ligmentum venosus).

Note the umbilical vein will atrophies , also the umbilical artery (this carry deoxygenated blood) when it atrophies it will become medial umbilical ligament.

Note : two factors contribute in rising the pressure of LA , these are

1. Increased systemic resistance after birth
2. Establishment of pulmonary circulation, the pulmonary veins will pump blood into the LA increasing its pressure (recall in fetal life LA receives blood from RA only via foramen oval)

In patent ductus arteriosus (persistence of the duct after birth- this is one of the Anomalies) what do u expect to happen?

- …
- the blood will pass from the aorta to the pulmonary trunk all the time >> during systole and diastole as the aorta pressure in both systole and diastole is higher

than the pulmonary trunk pressure (aorta 120 /80 , PT 25/8 – systole /diastole) so blood will continue to pass to the lower pressure vessel until the two vessels pressures become equal >> that's why if you enter a catheter in the aorta and the PT to measure their intra-pressure and u find that these two vessels have the same pressure then u must expect >> patent ductus arteriosus (diagnostic).

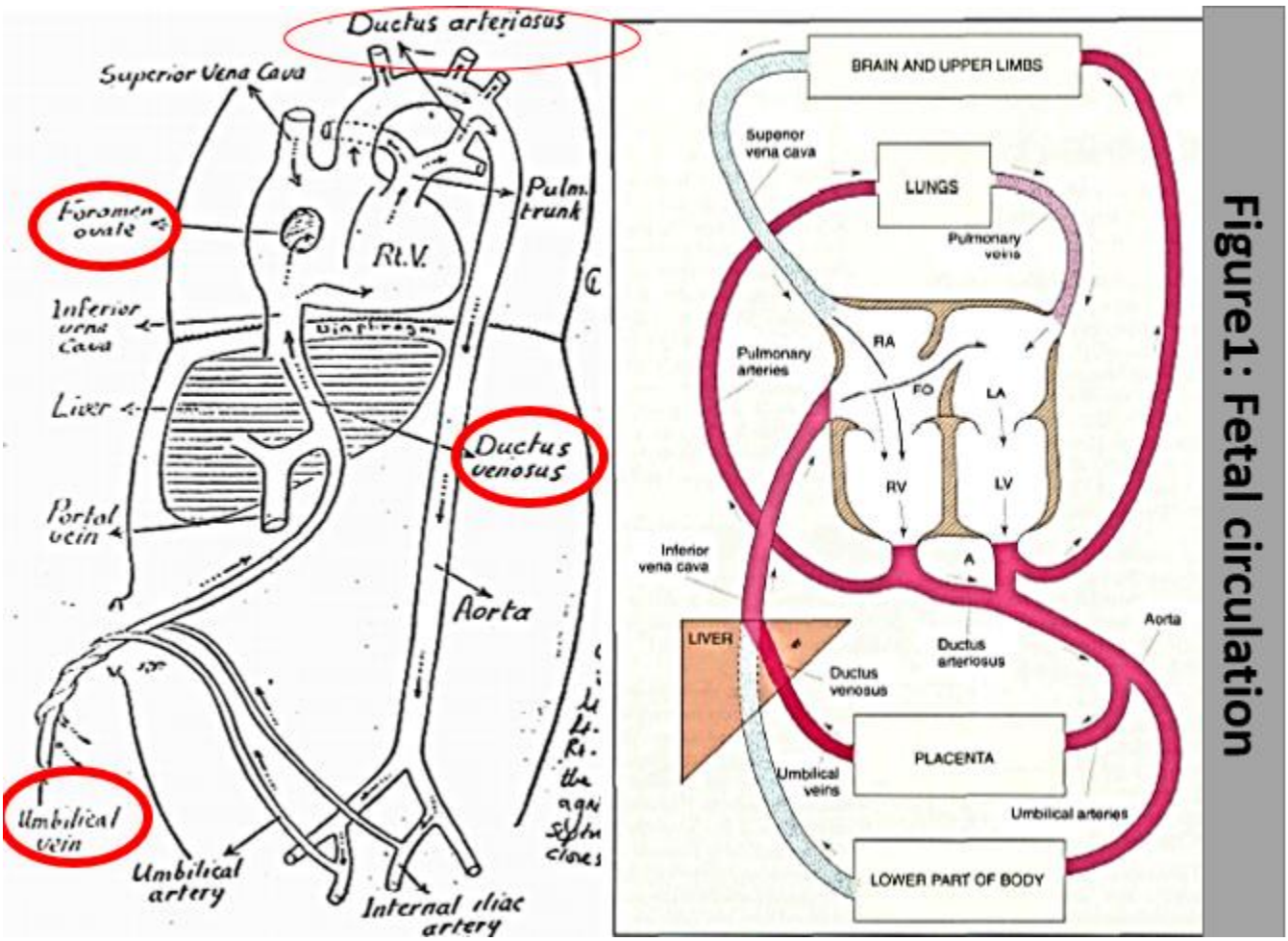
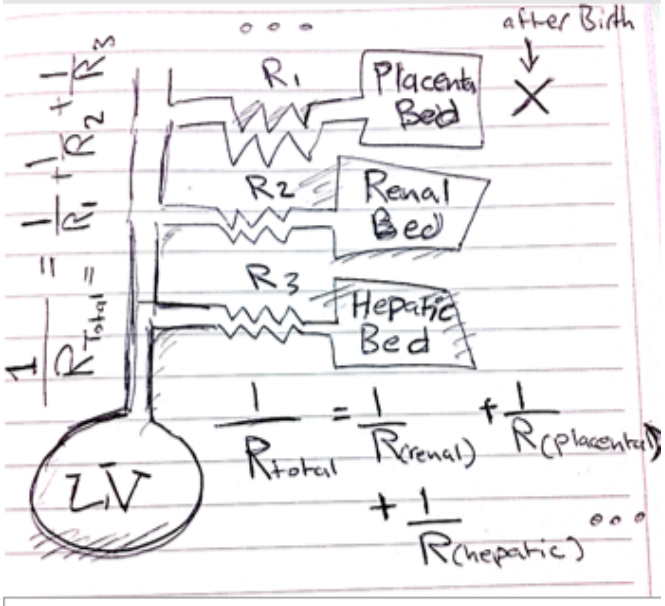


Figure1: Fetal circulation

Figure2 : parallel vascular resistances



The Left heart is affected by the total vascular resistance which is the summation of multiple parallel resistances (the law is shown)
after birth : the placental bed is removed and (Not shown in the figure) the pulmonary resistance get reduced. >> both of these contribute in raising the total R >> raising the

Congenital anomalies of the heart

Remember : any part that is made of many embryo. Origins is more subjected to congenital anomalies. To understand these anomalies u must study the normal embryo very well.

Atrial septal defect (ASD)

figure3

the septal defect can be of many types :

- ✓ Low atrial septal defect = osteum primum defect
  here the septum premium fail to reach the endocardial cushions >> a foramen is produced between septum premium and intermedium.
- ✓ High atrial septal defect = osteum secundum defect
  the septum secundum fail to develop.

- ✓ Patent foramen ovale , most common type .
  here the all septa has developed but the valvular passage remain (no fusion occur)
- ✓ Sometimes the septum intermedium don't develop properly , this is accompanied with no formation of interatrial septum and interventricular septum >> the result is single atria and single ventricle. (this case is present in 20% of mongoloids ie. down syndrome patients)

Ventricular septal defect (VSD)

Recall that the IV septum is made of muscular and membranous parts (the membranous is more commonly defected) when theres VSD the blood will pass from the LV to RV only during systole as>> (LV 120/0 , RV 25/0 >>systole/diastole)

Figure3 : septal defects –read the key

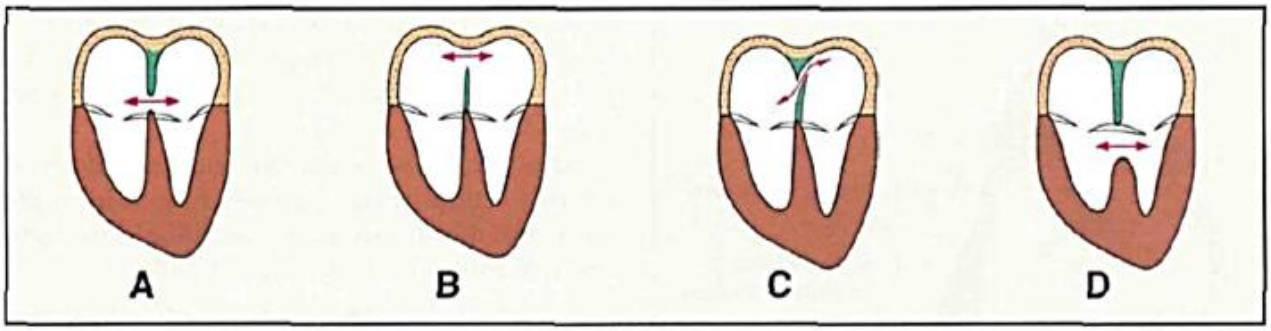


Fig. 15.24 Septal defects: (A) Septum primum defect, (B) Septum secundum defect, (C) Patent foramen ovale, (D) Interventricular septum defect.

note : septum = osteum

Anomalies of Truncus Arteriosus

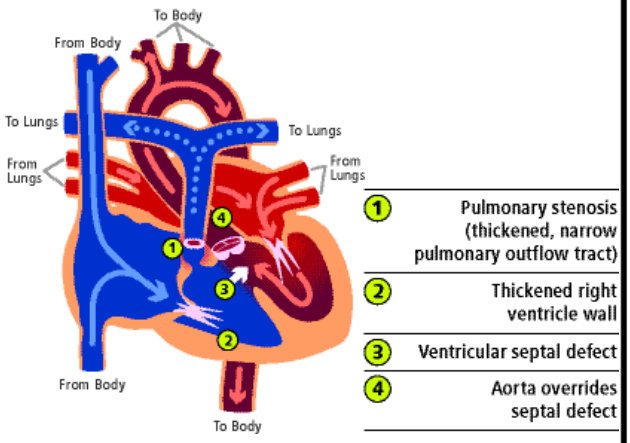
figure4

normally the TA must divide equally into Aorta and pulmonary trunk.
the anomaly here can arise if TA did not divide equally ,
remember that the pulmonary trunk origin is Anterior to the aorta
if the division was unequal , this can happen:

- ✓ The ascending aorta is bigger than the pulmonary trunk , here there are 4 events that will occur ,

  Fallot tetralogy :

  - Pulmonary stenosis: PT is smaller than usual.
  - Cyanosis : Aorta will receive blood from the two ventricles as its lumen is really large >> oxygenated blood of the aorta is mixed with deoxygenated blood of PT >> the body receive some deoxygenated blood >> cyanosis of the tongue , nail bed.. –further explanation later the aorta here is said to be “overridden”
  - Ventricular septal defect.
  - Right ventricle wall hypertrophy >> as it will try to push harder against the narrower lumen of the PT.



- ✓ Persistence truncus arteriosus , look at the figure
  here the TA will not divide proximally then on a higher level it will divide into Aorta and PT , the risk here reside again in mixing oxygenated blood and deoxygenated blood. So here the aorta will receive some deoxygenated blood >> cyanosis and also the PT will receive some oxygenated blood.

Figure4 : TA abnormalities

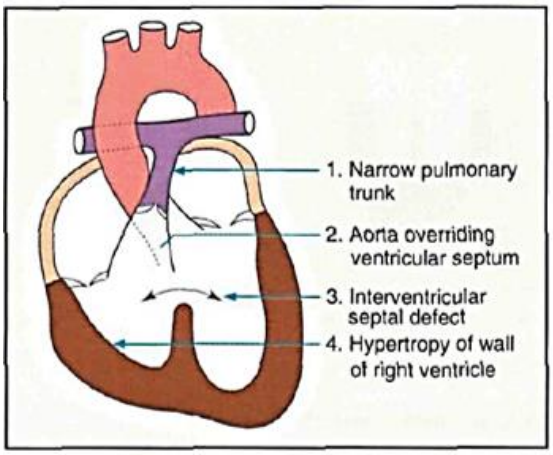
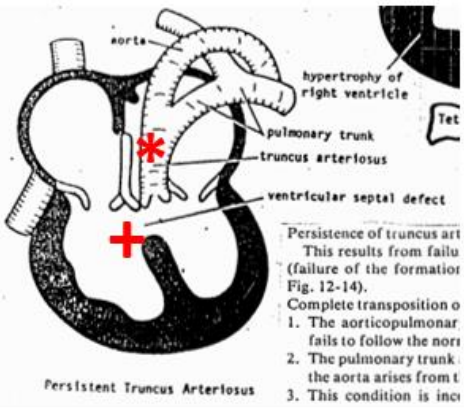


Fig. 15.25 Four features that constitute Fallot's tetrology.



Persistence TA indicated as (*) notice how it is accompanied with VSD (+)

Tricuspid atresia

Figure 5

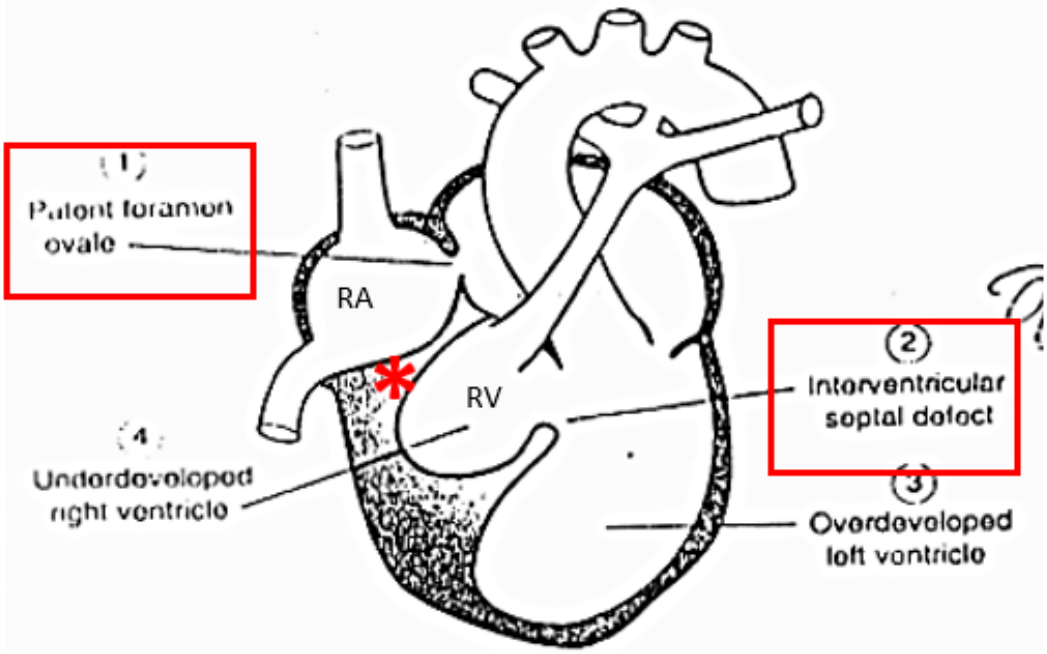
here >>No tricuspid foramen (between RV and RA) this anomaly is accompanied with patent foramen ovale and VSD.

again here we will have sever cyanosis , why?

the blood through foramen ovale will pass from RA to LA then from LA to LV then in the ventricles because of VSD oxygenated blood will be mixed with deoxygenated blood , the aorta receive some deoxygenated blood >> pump it to the body >> cyanosis.

(later in this sheet u will know that cyanosis is the result of increasing reduced Hb in the blood)

Figure5: tricuspid atresia (*)



Transposition of great vessels –discussed in sheet 6- figure 6

Here the normal spiral aorto-pulmonary ridge become straight , this implies that the PT will exit from the LV and the aorta will exit from the RV >> again mixing of blood >> but this time this condition is incompatible with life as the aorta won't just receive SOME deoxygenated blood , it will rather receive JUST deoxygenated blood>> and JUST deoxygenated blood receive the tissue.(the brain cannot tolerate this)
this condition can become compatible with life if there's potent foramen ovale (or any ASD) or VSD as some oxygenated blood can reach the aorta then tissues. (the brain can tolerate mixed blood)

Pulmonary atresia –figure6
rare condition
no foramen at the level of the pulmonary trunk origin from the RV.
this condition is accompanied with sever cyanosis and patent foramen ovale.
why cyanosis the blood is pumped from the RA to the RV >> then in the RV it faces atresia of the pulmonary foramen >> so no exit >> blood get back to RA then through patent foramen ovale blood go to the LA then to LV >> then to the aorta>> by this the aorta has received highly deoxygenated blood >> cyanosis.

Figure6 : transposition of great vessels and pulmonary atresia

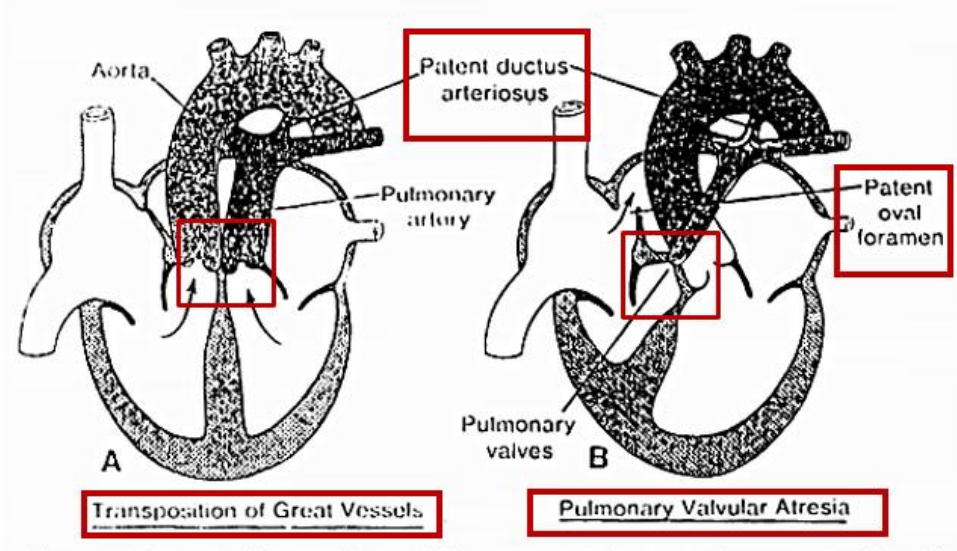


Figure 12-30. A, Transposition of the great vessels. B, Pulmonary atresia with normal aortic root. The only access route to the lungs is by way of the patent ductus arteriosus.

An important note is mentioned at the key of this figure: the only access route to the lungs is by patent ductus arteriosus.(in the two previous conditions)

Coarctation of the aorta التضيق الأورطي

Figure 7
The most important anomaly
the coarctation can happen post or pre ductal (related to ductus arteriosus level) in these situation we are concerned with lower part of the body blood flow as the stenosis threaten it.

Pre-ductal coarctation of the aorta:

- ✓ The stenosis of the aorta happen above the level of ductus arteriosus.
- ✓ Associated with other serious congenital heart defects.
- ✓ Prenatally : there's no real manifestations as the blood will pass normally to the lower part of the body through ductus arteriosus (the blood will be shifted to the pulmonary trunk and from the pulmonary trunk to the descending aorta through ductus arteriosus.
- ✓ Postnatally : ductus arteriosus atrophies and become a ligament. So no escape from fetial manifestations ;the newborn will die.

Post-ductal coarctation of the aorta:

- ✓ Below the level of ductus arteriosus
- ✓ Prenatally : formation of a collateral circulation–look at figure8
  this system is produced above the level of the stenosis and below it in away that able the blood to bypass the coarctated aorta.
  How?
  - Branches above the level of stenosis : one of the aorta branches is the subclavian a>> one of the branches of the subclavian artery is the internal thoracic artery >> this will give arise to **anterior intercostal arteries**.
  - Branches below the level of stenosis : the descending aorta give arise to **posterior intercostal arteries**.
    both ant. And post intercostal arteries anastomose with each others and by this the stenosis is bypassed prenatally.

- ✓ Postnatally:
  - there's no problem now , the blood can easily bypass the stenosis As the collateral circulation is already well established,
  - after birth no significant change occurs in the circulation.
  - This is common finding in Turner's syndrome. (handout only)

As a results of this collateral circulation some related findings can be observed:

1. Pulsation between the two scapulae of the patients (observed at the back of the patient -.-)
2. Notching of lower borders of the ribs as the tortuous arteries are present there. (observed with x-rays of the thorax)
3. The aorta appear doubled on X ray>> the presence of the stenosis make the surrounding aortic wall at each sides appear large >> the tube (aorta) won't appear continuous>> it will appear as two knuckles on the x-rays instead of one continuous tube with one knuckle.
   what enhance this observation is the fact that the blood under the stenosis level flow against its normal route (normally the blood flow from descending aorta to post intercostal arteries to anterior intercostal arteries –from larger arteries to smaller arteries) here the blood flow from ant. Intercostals To post. Intercostals then to the aorta Because of the stenosis.>>as a response to this the aorta distal to the stenosis will enlarge making another knuckle.
4. Different recorded pressure between upper half of the body and the lower half. Blood pressure of the lower limb is lower than the upper limb. As the lower half -because of the stenosis- will still receive lesser amount of blood (despite the anastomosis thing) .
   so when recording high blood pressure in a child , u must also record his lower limb blood pressure to exclude Coarctation of the aorta
   so, Coarctation of the aorta elevate blood pressure in an induvial because of the presence of post-ductal stenosis (this will elevate upper half BP) secondly visceral organs blood flow will also be reduced(as its in the lower part) >> one of these organs are the kidney >> when sense low perfusion it secrets renin >> renin-angiotensin-aldosterone system will work >> elevation of BP.

Figure7 : coarctation of the aorta

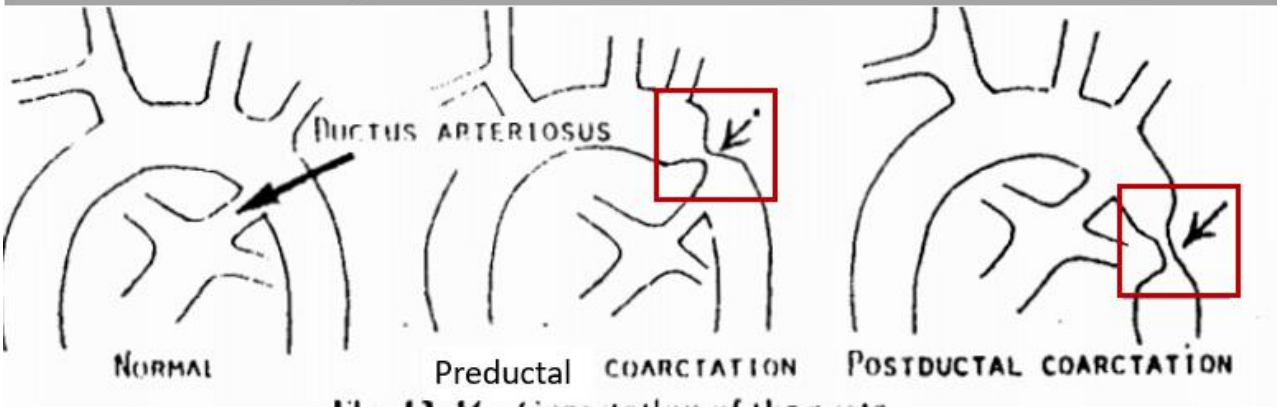
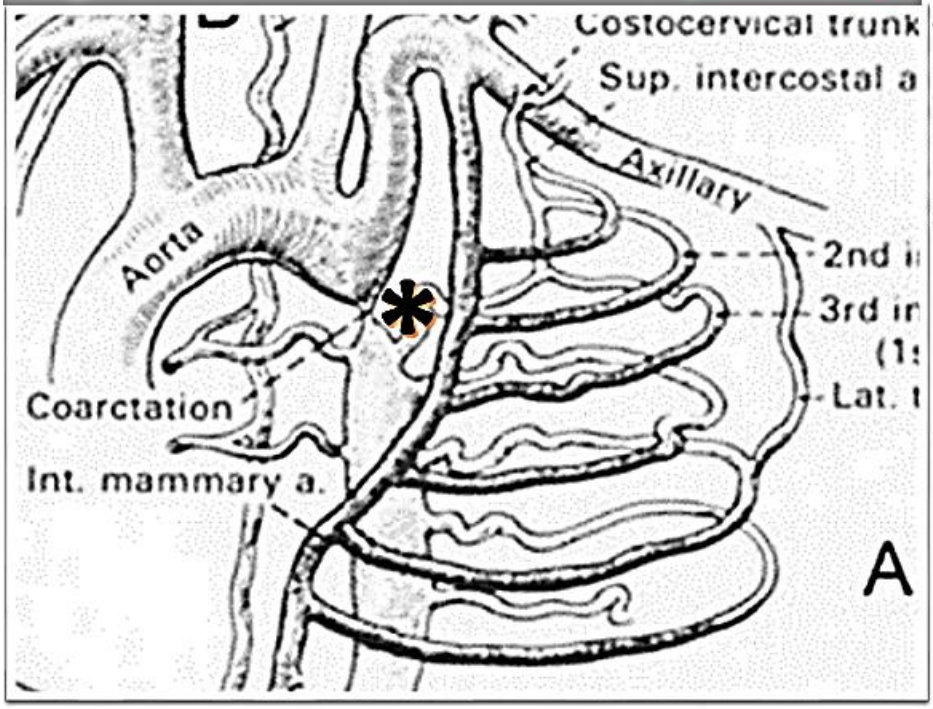
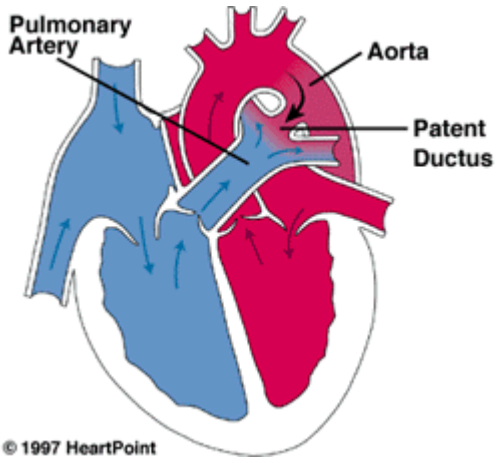


Figure8: collateral anastomosis in postductal



The anterior intercostal arteries (branches of the subclavian arteries) anastomosis with the posterior intercostal arteries (branches of the descending aorta) bypassing the coarctation of the aorta , indicated as (*)

Patent Ductus Arteriosus:



- Usually occurs in babies of pregnant females who had a rubella virus infection "maternal rubella" in the first weeks of pregnancy!
- More common in females
- May be accompanied by other defects
- Diagnosed by **Catheterization**, when pressure in aorta and pulmonary trunk are **equal**!
- Normally in the Aorta: systole 120mm, diastole 80mm
- In the Pulmonary: systole is 25mm diastole 10mm

"the shunt works in systole and diastole" *"because normally in both cases the aortic pressure is higher than the pulmonary pressure"* so pressure is equal to 120-80 in both arteries(Aorta and pulmonary)

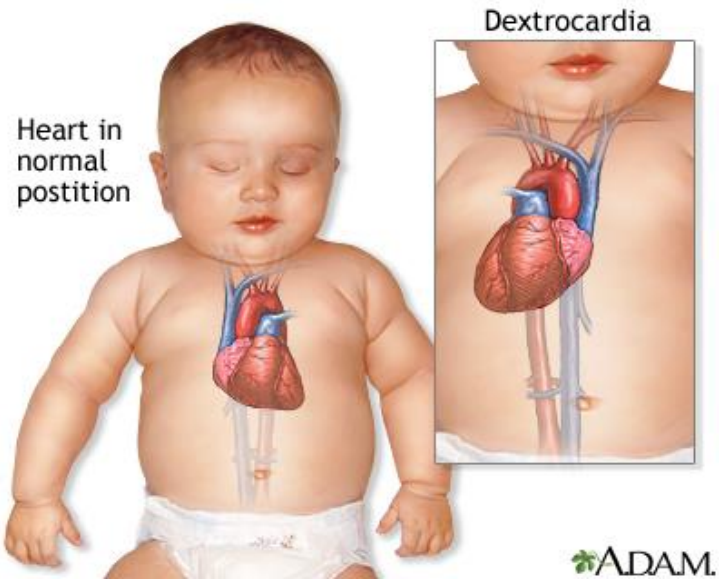
Dextrocardia:

Normally In the embryo the looping of the primitive heart tube to the **Right**,

If the looping is to the **Left** then it is Dextrocardia

Dextrocardia is **Mirror image** of the normal heart, "Apex is downward forward to the right instead of to the left"

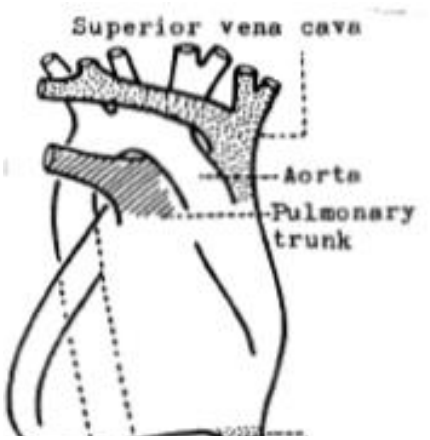
SVC instead of being to the right will be to the **left**

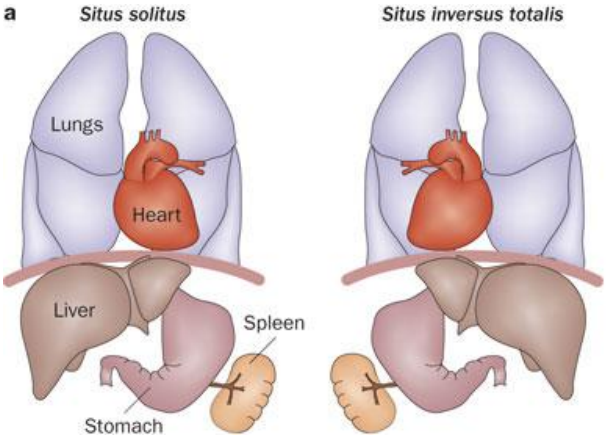


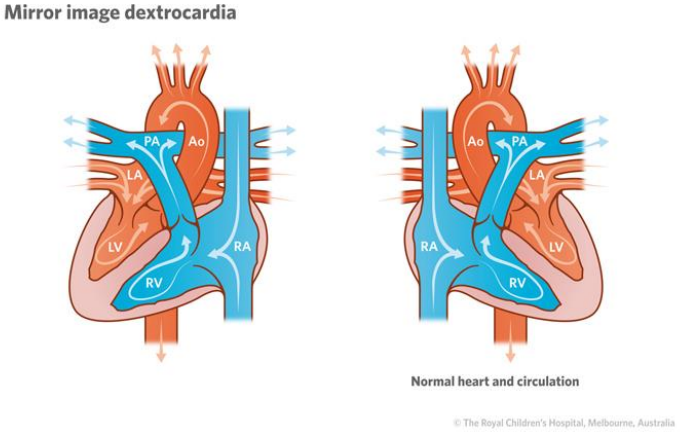
Dextrocardia may be accompanied by **Situs Inversus**
"organs are to their opposite sides!"

E.g. liver is to the left, spleen to the right!

If dextrocardia is accompanied by situs inversus then the heart will be **normal**, "*relatively*"







Cyanosis of Congenital Anomalies:

Definition: Cyanosis is a blue coloration of skin and mucus membrane due to "more than 5g reduced Hb in circulation"

Central vs Peripheral cyanosis:

DIFFERENCES BETWEEN CENTRAL AND PERIPHERAL CYANOSIS

	Central	*Peripheral*
Mechanism	Dimininshed arterial oxygen saturation	Diminished flow of blood to the local part
Sites	On skin and mucous membranes e.g. tongue, lips, cheeks etc.	On skin only
Clubbing and polycythemia	Usally associated	Not associated
Temperature of the limb	Warm	Cold
Local heat	Cyanosis remains	Cyanosis abolished
Breathing pure oxygen	Cyanosis decreased	Cyanosis persists

Central cyanosis:

In Lips, the tongue and also the peripheral parts “fingers and nail beds”

occurs due to **Venoarterial shunt** “mixed blood”:

- Fallot tetralogy “overriding aorta gets blood from right ventricle”
- Tricuspid Atresia “from right atrium to left atrium”
- Persistent truncus arteriosus “left ventricle blood(oxy) mixed with right ventricle blood (deoxy)”

Oxygen tension lower than normal “due to mixing of venal blood with arterial blood”

“If we give a highly oxygenated air to the patient cyanosis will disappear” (hyperbaric chamber)

Peripheral cyanosis:

- only fingers nail beds
- Normal oxygen tension,
- No Mixing of venal blood with arterial blood!!
- Normal looking lips and tongue,
- Occurs due to: slow blood flow, “may be from congestive heart failure, Stenosis of arteries or atherosclerosis”

(Blood won’t reach peripheries at a normal rate ⇨ reduced hemoglobin concentration is higher than normal ⇨ cyanosis)

Classification of Heart Defects Anomalies:

(Either with or without cyanosis)

Without Cyanosis:

1. With right ventricular hypertrophy:
   a) Pulmonary stenosis
   b) ASD

2. With left vent hypertrophy:
   a) Aortic stenosis
   b) Coarctation of aorta
   c) Patent ductus arteriosus “blood from aorta to pulmonary will cause more work on the left ventricle”

3. Biventricular hypertrophy:
   Big ventricular septal defect “VSD”

4. With no ventricular hypertrophy:
   a) Small VSD (Rogeri disease) “small amounts pass so no hypertrophy in either side”
   b) Dextrocardia!

With cyanosis: *cyanotic group*

1. Fallots tetralogy “mixed blood” (the more severe the pulmonary stenosis is the more severe the cyanosis)
2. Tricuspid atresia
3. Persistent truncus arteriosus
   “these three have <u>deep cyanosis</u> , due to mixed blood -> more than 5g of reduced Hb”

☆ Continued: Congenital Anomalies of the Heart:

a. Congenital pulmonary stenosis:

Stenosis "narrowing" in the pulmonary valve or infundibulum,

less blood to the lungs ⇨ lower pressure in pulmonary trunk, increased pressure in right ventricle ⇨ right ventricle hypertrophy ⇨ continued hypertrophy leads to failure and weakness and death of its fibers, why?

Because these fibers are getting bigger without increasing the blood supply to them, so they will be ischemic then infarct. ☠

b. Atrial septal defect (ASD): *"more in females"* *mentioned earlier*

Blood moves from high pressure to low pressure, (From left atrium to right atrium).

"In the fetus the blood moves from right atrium to left atrium normally through foramen ovale!!"

After birth left atrium will have more pressure, so if there was an ASD blood will move to the Right atrium

Less blood will go to the left ventricle ⇨ Lower cardiac output! ⇨ Fatigue …

Note: "ASD or VSD ⇨ less blood to the left ventricle ⇨ less CO"

c. Coarctation of aorta (post ductal) *mentioned earlier*

Remember: pressure in upper limb is higher than in the lower limb,

Angiotensin II also increases blood pressure! "Released due to low blood reaching the kidney"

"We care about post ductal because preductal is considered fatal!"

d. Patent ductus arteriosus: *maternal rubella infection* *mentioned earlier*

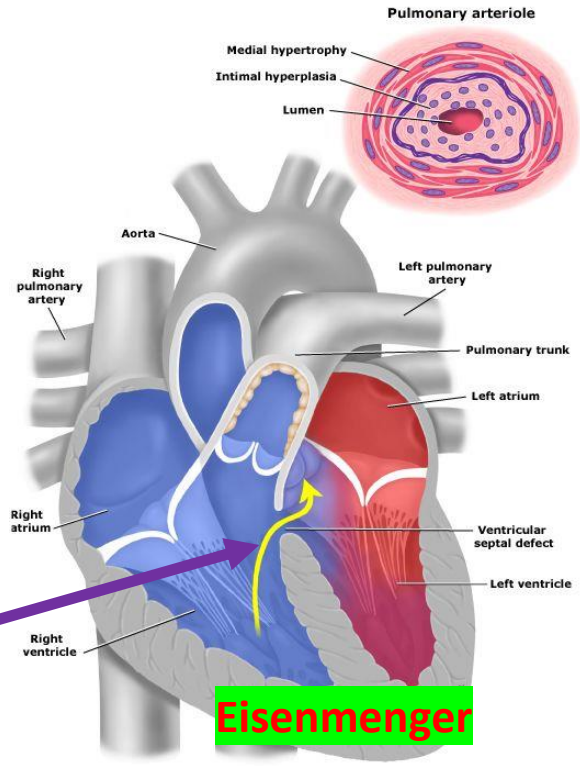
e. Big VSD and Eisenmenger syndrome:

Big ventricular septal defect "shunt from left ventricle to right ventricle **"only in systole"** because in diastole pressure in both ventricles is ZERO (for the millionth time)"

Left will pump blood to aorta and right vent -> hypertrophied, blood in right will be pumped to pulmonary -> more blood will be pumped due to blood coming from left vent -> right hypertrophy!

Resulting in lower CO -> fatigue

After few years shunt will be reversed if not treated, lungs will resist the overflow of blood "يزعلو الرئتين" so they will induce vasoconstriction as a protective mechanism
⇨ vasoconstriction will cause more resistance which will increases the pressure
⇨ pulmonary pressure will be increased
⇨blood will accumulate in the right ventricle and its pressure will be increased until it become more than the pressure of the left ventricle ⇨ then the shunt will be **Reversed**



This state of reversed shunt is called "**Eisenmenger syndrome**"

Eisenmenger is caused by **VSD + Pulmonary hypertension** !!!

Cyanosis occurs with Eisenmenger syndrome, while Big VSD is non cyanotic!!