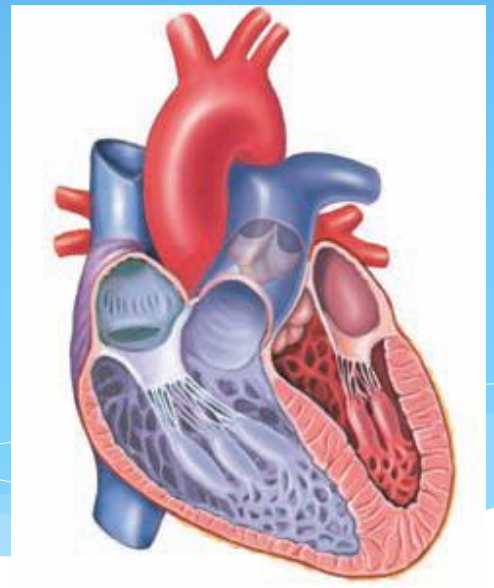
POLTAVA STATE MEDICAL UNIVERSITY

Features of blood circulation of the fetus. Anatomic-physiological features of the heart and blood vessels in children. Semiotics of congenital and acquired diseases of the heart and blood vessels.



Assoc. Professor
Soloviova Halyna

Plan of the lecture

* 1. Fetal and neonatal circulatory systems: shunts and changes at birth.
* 2. Congenital heart diseases.
* 3. Acyanotic defects of the heart.
* 4. Cyanotic defects of the heart.
* 5. Acquired heart disease.
* 6. Tests commonly used to diagnosis congenital heart defects.
* 7. Preventing birth defects.

Fetus

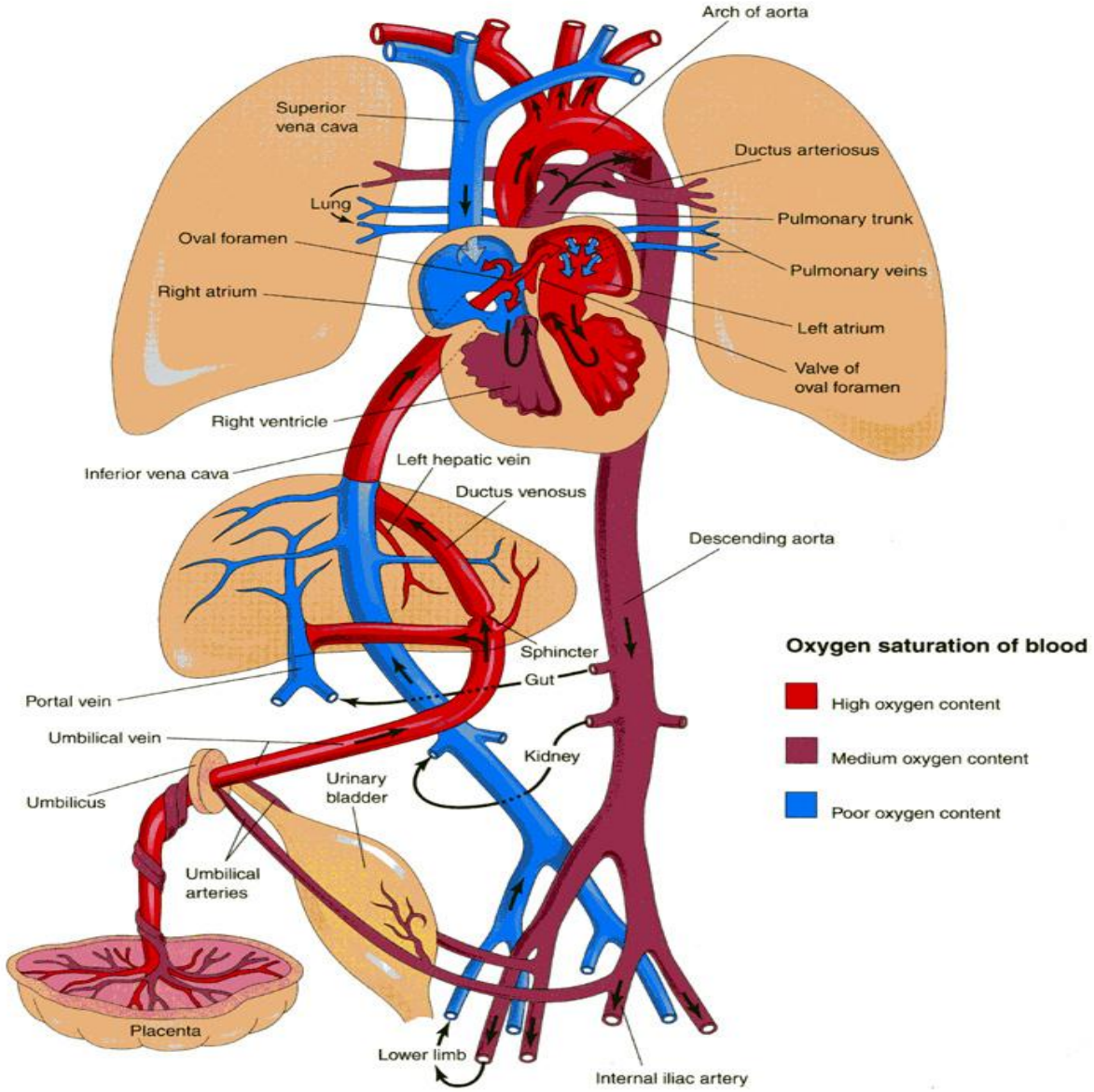


Figure 14 – 46. Fetal circulation. The colors indicate the oxygen saturation of the blood, and the arrows show the course of the blood from the placenta to the heart. The organs are not drawn to scale. Observe that three shunts permit most of the blood to bypass the liver and lungs: (1) ductus venosus; (2) oval foramen; and (3) ductus arteriosus. The poorly oxygenated blood returns to the placenta for oxygen and nutrients through the umbilical arteries.

Neonate

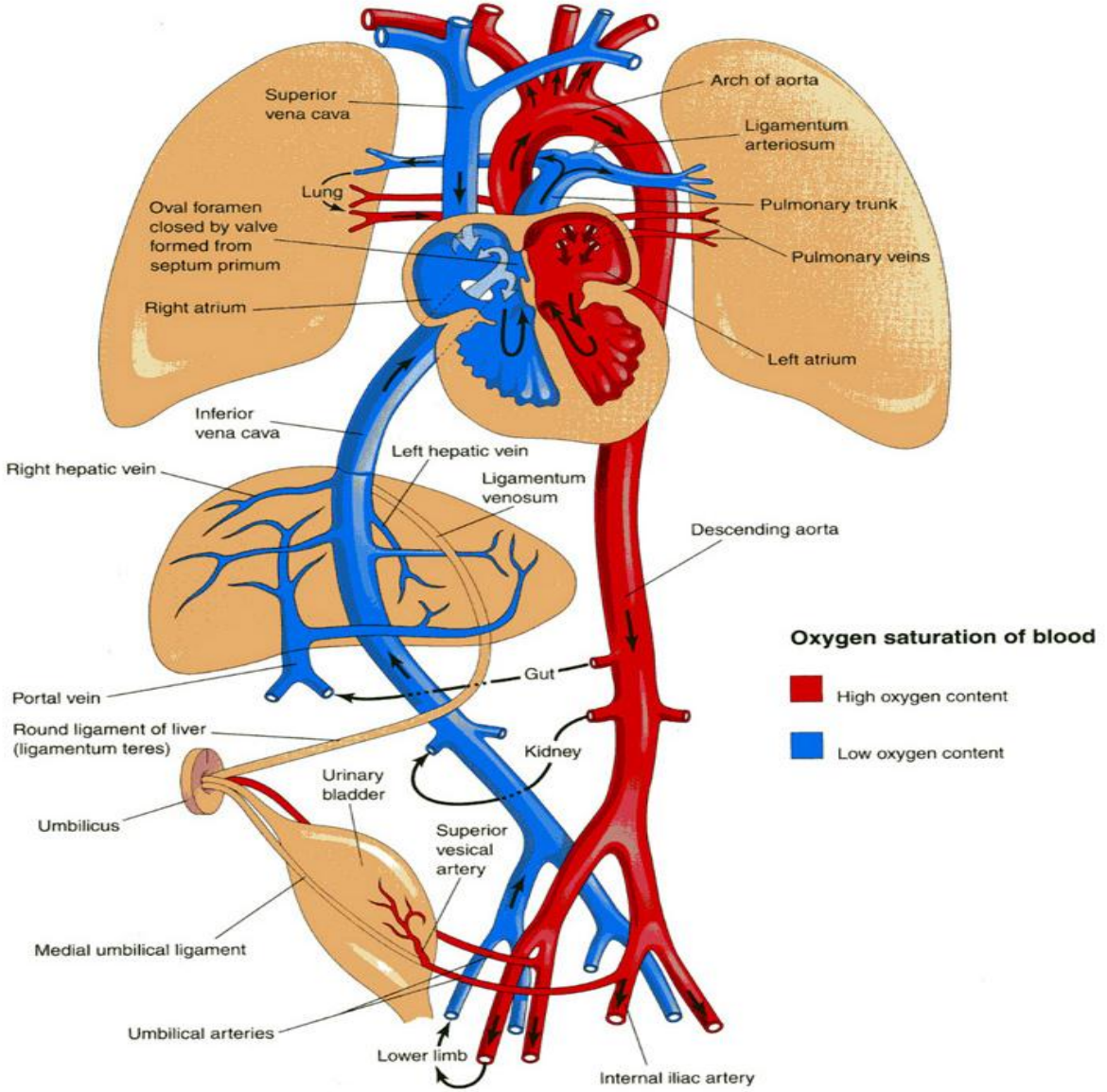


Figure 14 – 47. Neonatal circulation. The adult derivatives of the fetal vessels and structures that become nonfunctional at birth are shown. The arrows indicate the course of the blood in the infant. The organs are not drawn to scale. After birth the three shunts that short-circuited the blood during fetal life cease to function, and the pulmonary and systemic circulations become separated.

Fetal and neonatal circulatory systems: shunts and changes at birth

1. Fetal foramen ovale shunts blood from right to left atrium

   - Adult remnant is fossa ovalis

2. Fetal ductus arteriosus shunts 90% of blood from pulmonary trunk to aorta

   - Adult remnant is ligamentum arteriosum

3. Fetal ductus venosus shunts 50% of blood from umbilical vein to inferior vena cava by passing liver

   - Adult remnant is ligamentum venosum

Differences in circulatory systems

Prenatal:

- Little pulmonary blood flow
- Gas exchange via placenta
- Nutrient delivery to fetus through placenta
- Right to left shunting of blood in heart

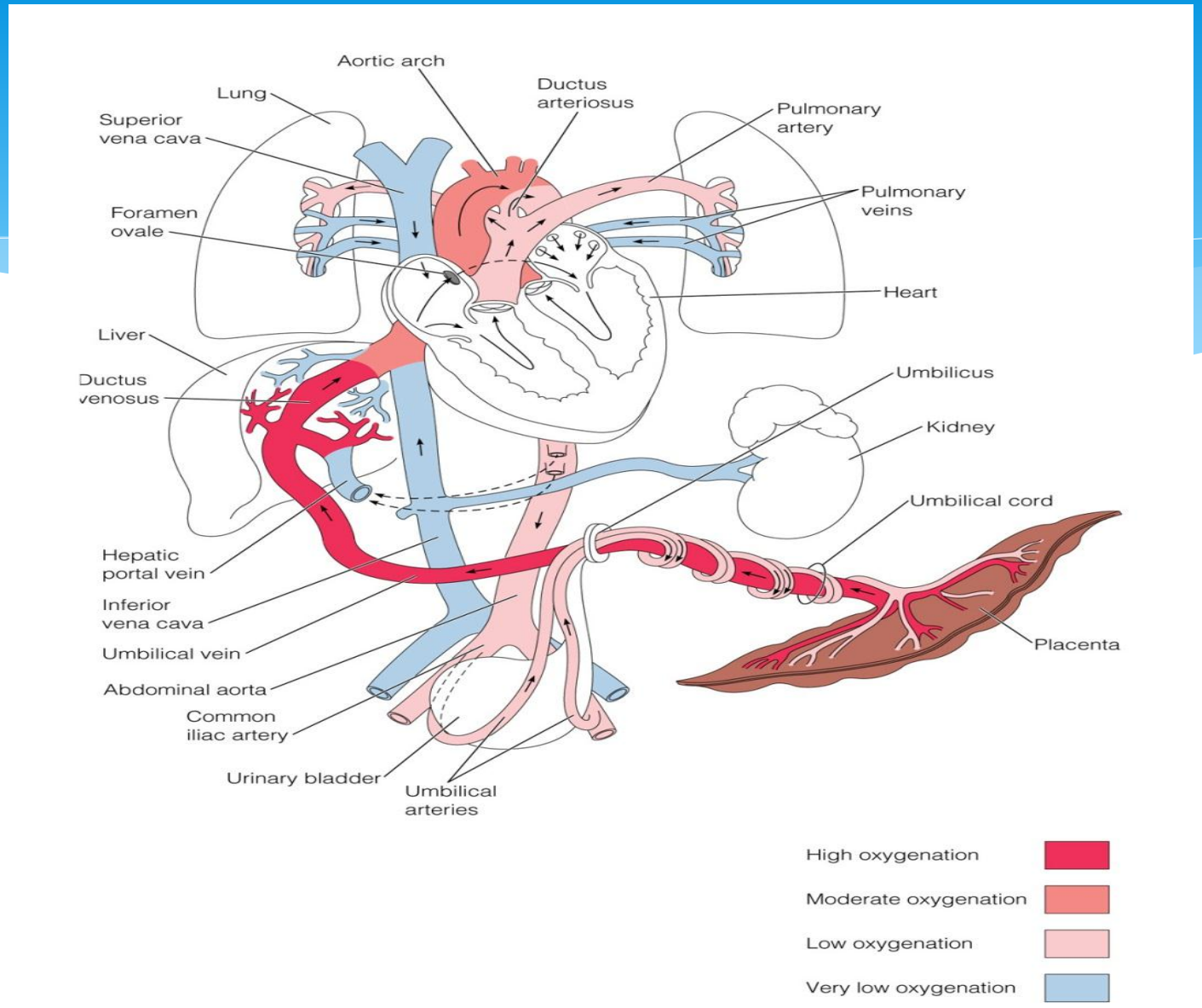
Postnatal:

- Functional pulmonary respiration and gas exchange
- Loss of placental circulation
- Occlusion of right to left shunt in heart and fetal anastomoses

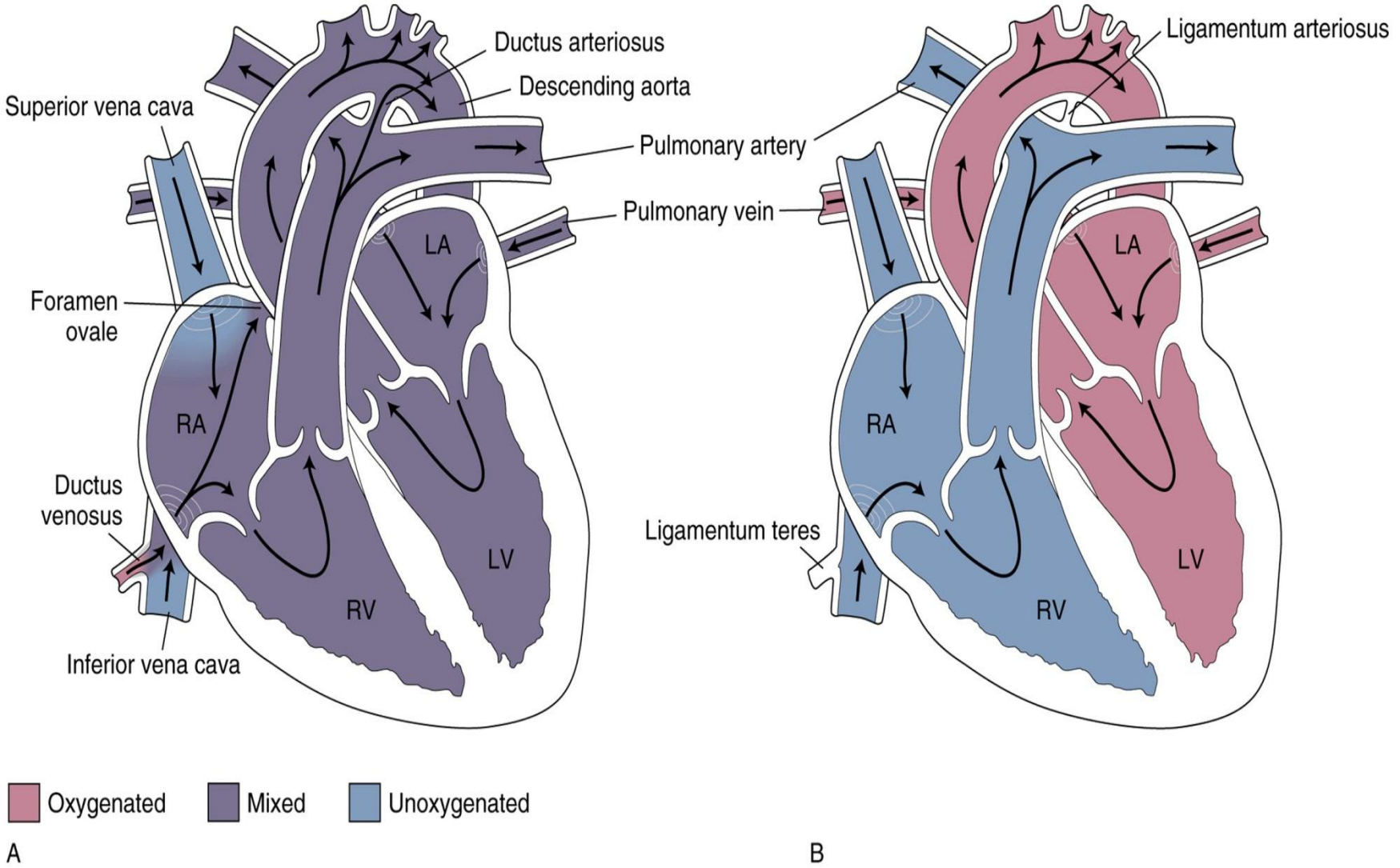
Congenital heart defects

- Most common type of congenital malformations
- Incidence of nearly 1% of live births
- Causes elusive, multifactorial: single gene & chromosome defects, environmental factors, viruses, toxins, alcohol, drugs
- Specific etiology unknown in many cases but most arise during critical period of heart dev. 20-50 days after fertilization
- Well tolerated before birth because of fetal shunts
- Most produce symptoms postnatally

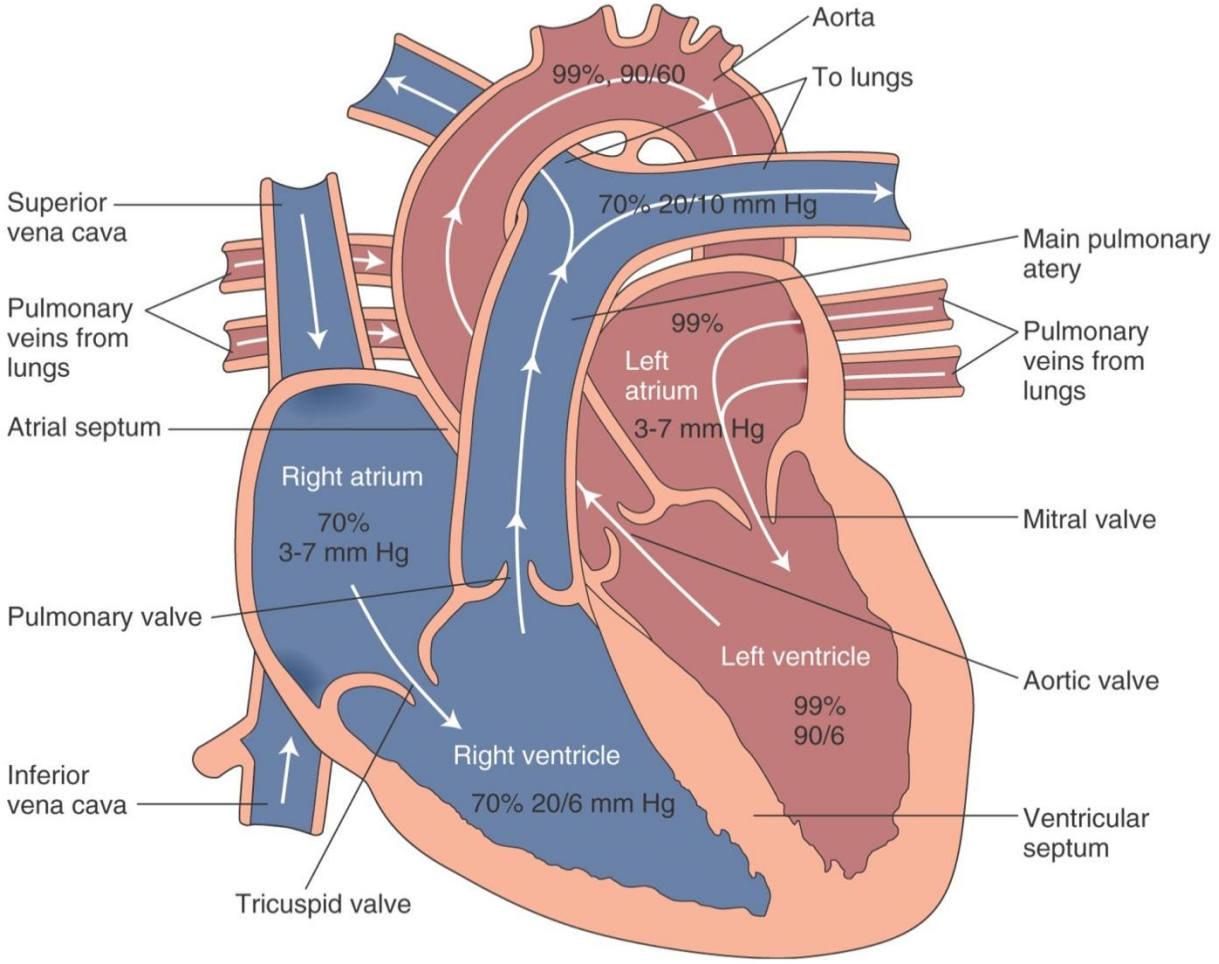
Fetal circulation. Blood leaves the placenta and enters the fetus through the umbilical vein. The ductus venosus, the foramen ovale, and the ductus arteriosus allow the blood to bypass the fetal liver and lungs. After circulating through the fetus, the blood returns to the placenta through the umbilical arteries. .

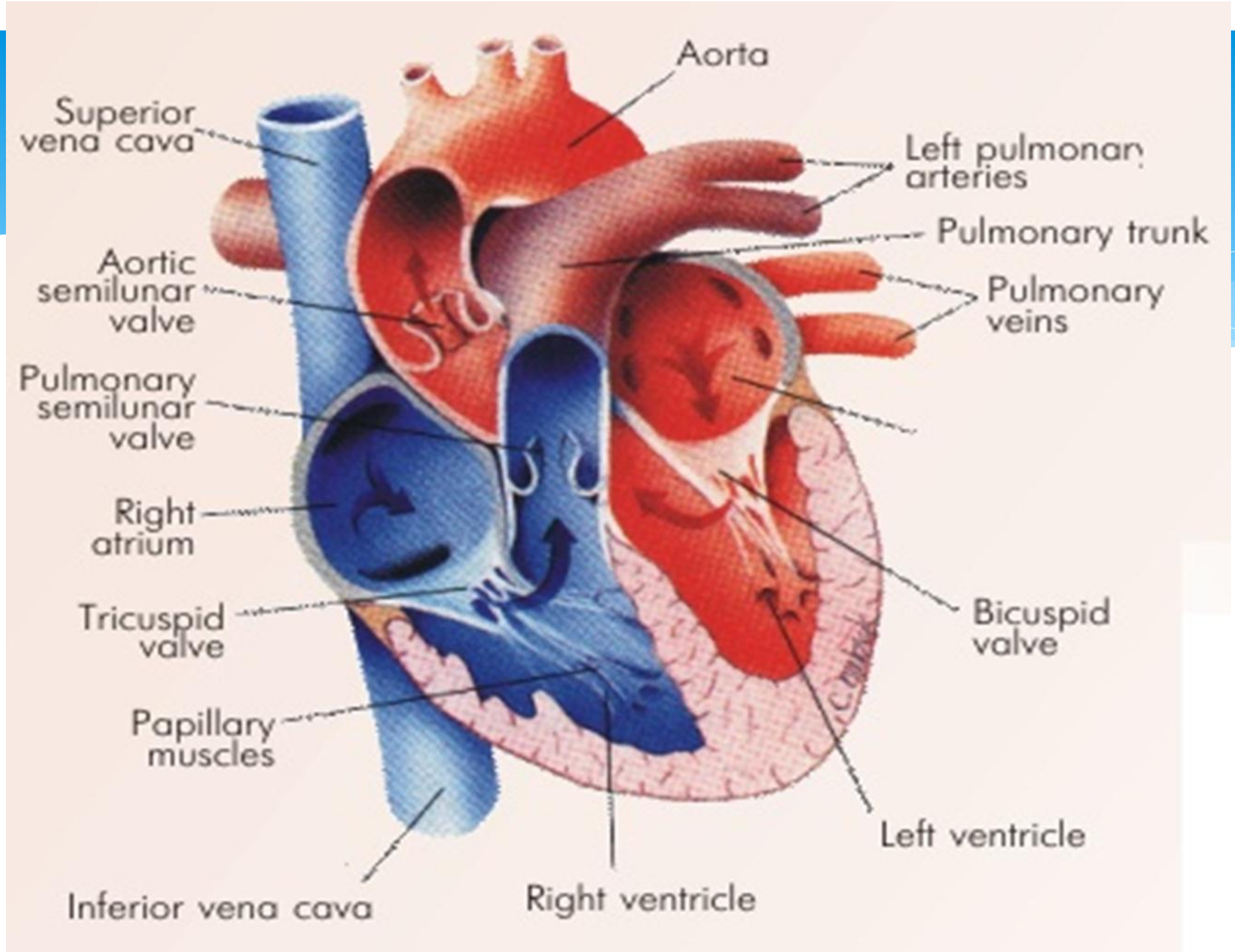


A, Fetal (prenatal) circulation. *B*, Pulmonary (postnatal) circulation. *LA*-left atrium; *LV*-left ventricle; *RA*-right atrium; *RV*-right ventricle.



Normal pressure gradients and oxygen saturation levels in the heart chambers and great vessels. The ventricle on the right side of the heart has a lower pressure during systole than the left ventricle because less pressure is needed to pump blood to the lungs than to the rest of the body.




Aorta
Superior vena cava
Left pulmonary arteries
Pulmonary trunk
Aortic semilunar valve
Pulmonary veins
Pulmonary semilunar valve
Right atrium
Tricuspid valve
Bicuspid valve
Papillary muscles
Left ventricle
Inferior vena cava
Right ventricle

Average pulse rates at rest (beats per minute)

Newborn	140-160
6 months	130-135
1 year	120-125
2 years	110
3 years	105
4 years	100
5 years	98-100
6-7 years	90-85
10 years	78-85
12 years	70-75

The normal rate is not more then 10 % of average

Normal Rate Of Blood Pressure

- Lowest acceptable systolic blood pressure
  - ***Birth – 1 month:*** 60 mmhg
  - ***1 month – 1 year:*** 70 mmhg
  - ***1 year – 10 year:*** 70 + (2 X age in years)
  - ***>10 years :*** 90 mmhg
- Normal systolic
  - 80 + (2 x age in years)
  - or fiftieth percentile
- Diastolic: 1/2-1/3 of systolic

Classification of congenital heart failures in children

Non-cyanotic:

- *ventricular septal defect*
- *atrial septal defect*
- *patent ductus arteriousus*
- *pulmonary stenosis;*
- *coarctation of aorta*
- *atrioventricular canal defect*

Cyanotic:

- *Fallot's tetralogy;*
- *transposition of great arteries*
- *tricuspid atresia*
- *an atrial septal defect*
- *total anomalous pulmonary venous return*
- *hypoplastic left heart*
- *pulmonary valve atresia*
- *a patent ductus arteriosus*
- *Ebstein's anomaly.*

Relative Frequency of Lesions

Ventricular septal defect	25-30
Atrial septal defect (secundum)	6-8
Patent ductus arteriosus	6-8
Coarctation of aorta	5-7
Tetralogy of Fallot	5-7
Pulmonary valve stenosis	5-7
Aortic valve stenosis	4-7
Transposition of great arteries	3-5
Hypoplastic left ventricle	1-3
Hypoplastic right ventricle	1-3
Truncus arteriosus	1-2
Total anomalous pulm venous return	1-2
Tricuspid atresia	1-2
Double-outlet right ventricle	1-2
Others	5-10

Acyanotic defects

- Postnatally these result in left-to-right shunting of blood in the heart
- Since blood has been oxygenated in the lungs cyanosis does not develop
- These shunts in the atria, ventricles or great vessels cause volume or pressure overloads, especially in right side of heart
- Symptom may be excessive fatigue on exertion
- Volume overloads in the case of ASD, VSD and PDA
- Pressure overloads in case of aortic stenosis, pulmonary stenosis and coarctation of aorta.

Ventricular septal defect

- With an incidence of 30%, VSD are the most common life-threatening congenital cardiac anomaly.
- They can occur in either the membranous or muscular portions of the septum; 70% are in the membranous.
- 30-50% of vsd close spontaneously during the first year of life.
- Membranous vsd is caused by incomplete fusion of right truncoconal ridge, left truncoconal ridge and endocardial cushion tissue. This fusion, plus the up growth of the muscular IV septum closes the IV foramen.
- If this does not happen and the defect is large, there can be massive left-to-right shunting of blood through the defect with increased and excessive pulmonary blood flow (with hypertrophy of la and lv and possibly slight rv hypertrophy because of shunting), pulmonary hypertension, dyspnea, deficient systemic blood flow and cardiac failure.

Ventricular septal defect

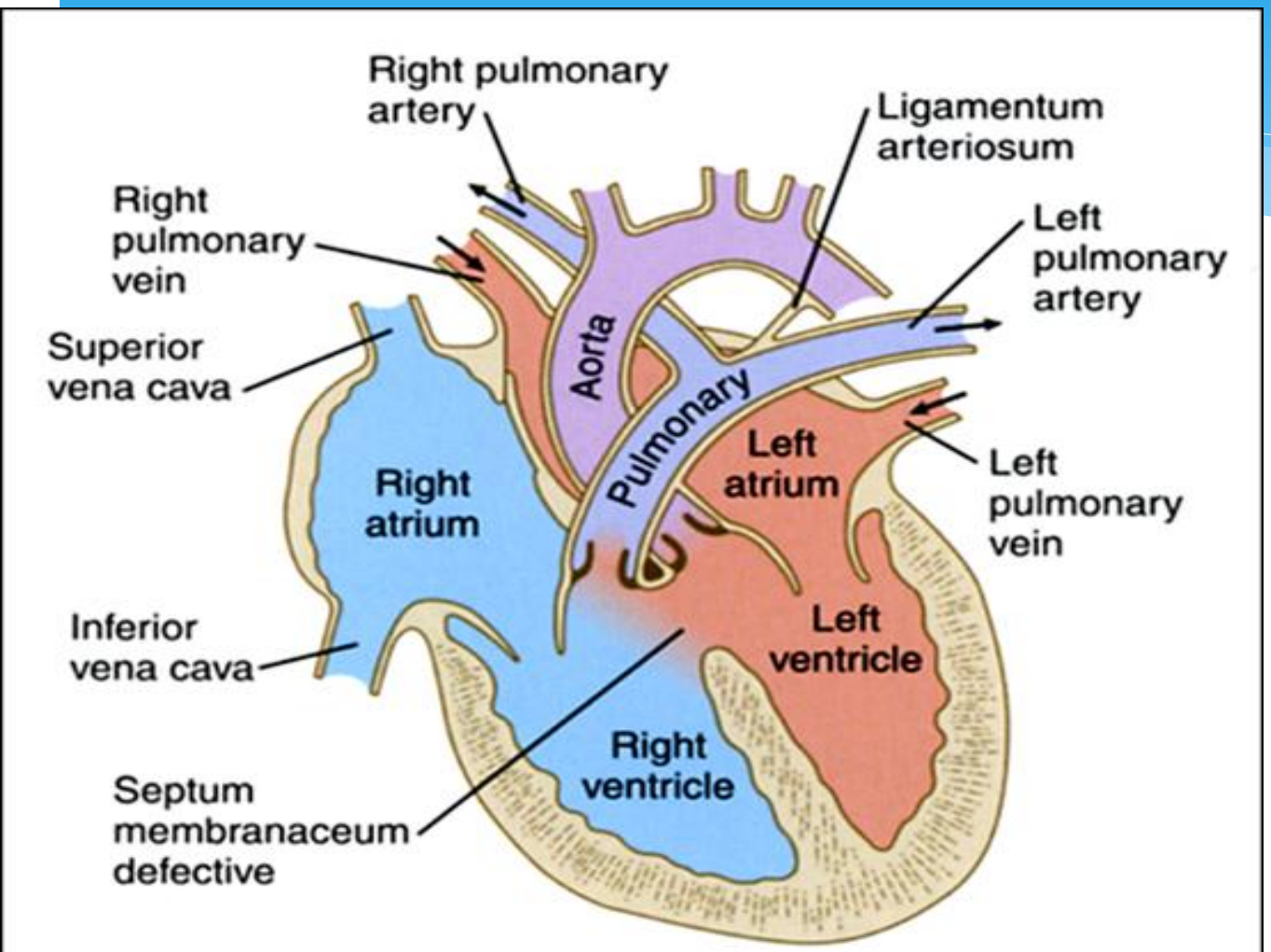
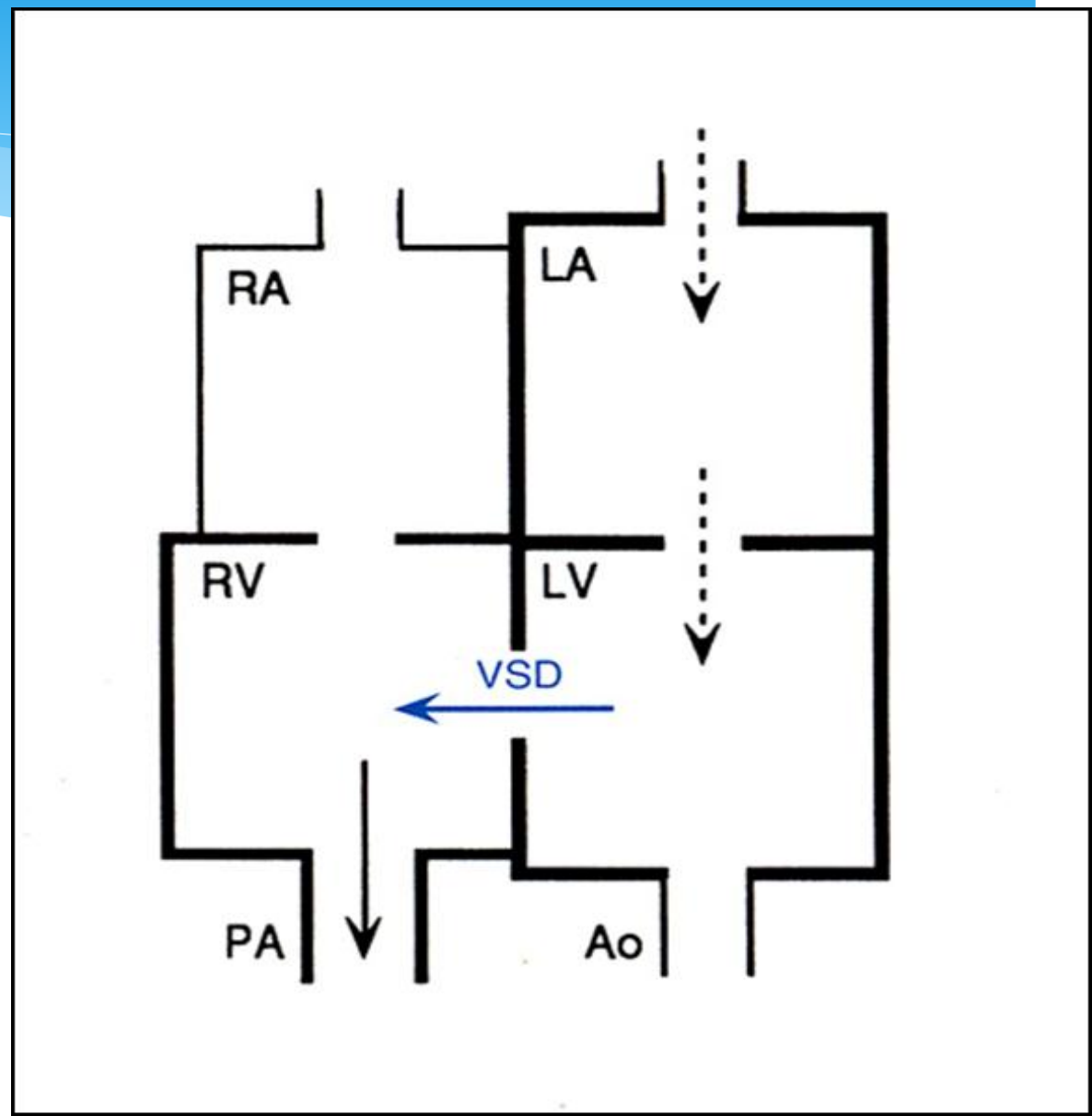


FIGURE 17-31 Interventricular septal defect (membranous portion). Mixing of arterial and venous blood occurs in both outflow tracts but especially in the pulmonary artery.



Atrial Septal Defect

- ✓Defects with increased pulmonary blood flow
- ✓Opening between the atria
- ✓Incidence is 10%
- ✓Heart auscultation: Murmur, second heart sound splitting
- ✓Management - surgery

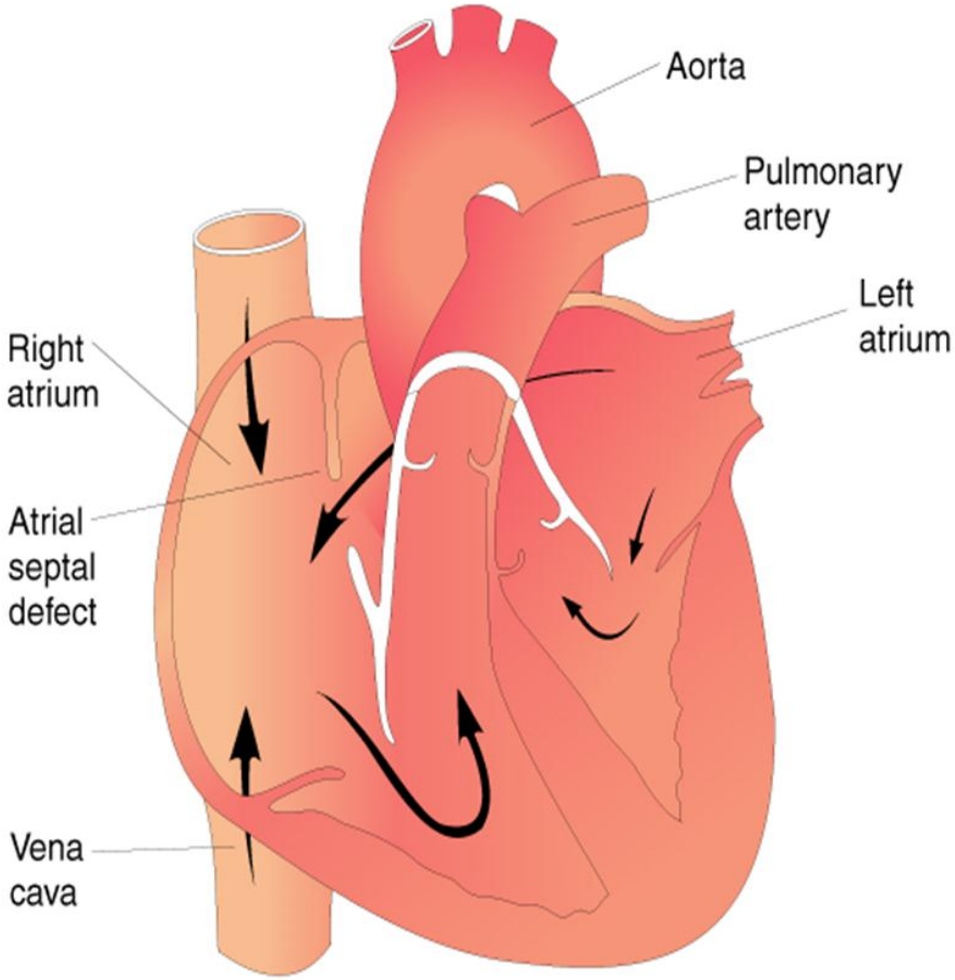


FIGURE 41.9 Atrial septal defect.

Atrial septal defects

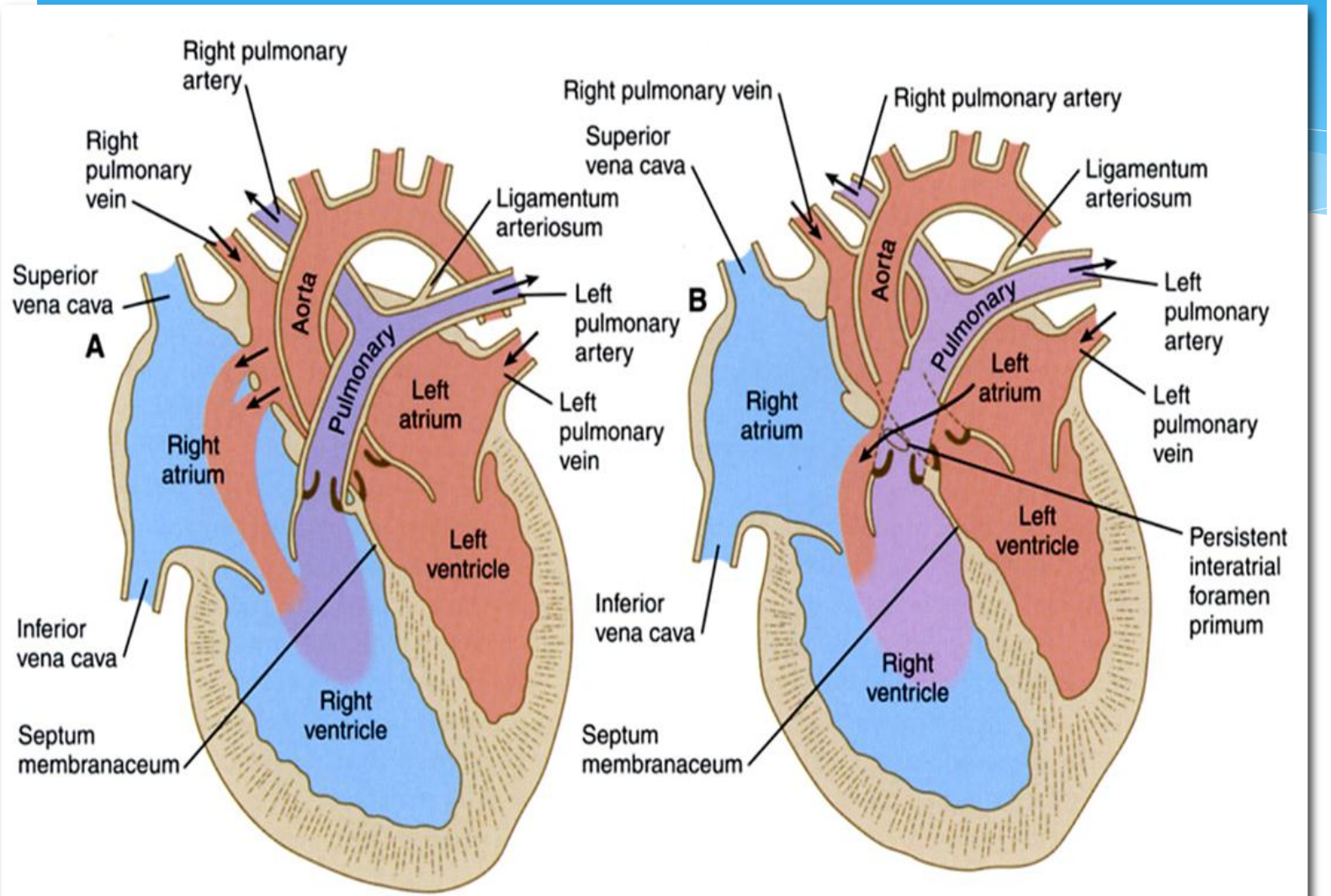
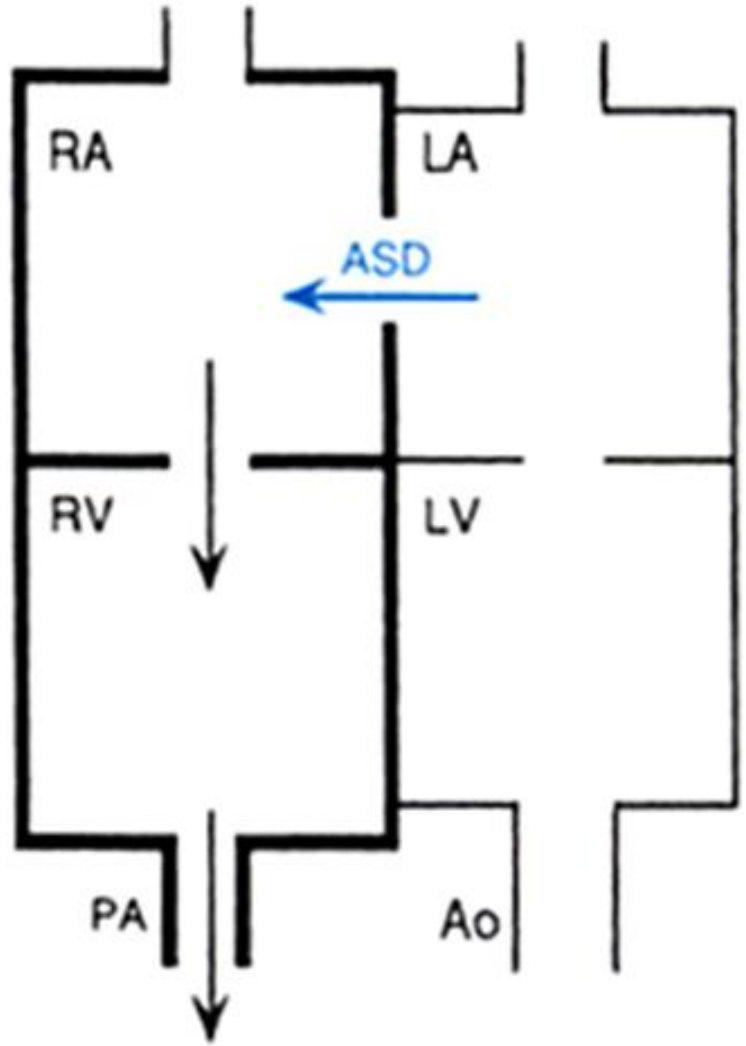


FIGURE 17-27 High **(A)** and low **(B)** atrial septal defects in the heart. Red denotes well-oxygenated arterial blood, blue denotes poorly oxygenated venous blood, and purple denotes a mixture of arterial and venous blood.



Patent foramen ovale (probe patent foramen ovale)

- Present in 20-25% of population
- Not a true atrial septal defect
- A few millimeters in diameter
- Caused by failure of fusion of atrial septa by 6 mo.
- Usually clinically insignificant
- Although one way valve not sealed, it remains closed because left atrial pressure is higher than right
- Significant only if other heart disease develops (with right -to-left shunting, Pulmonary hypertension); implicated in strokes.

Patent ductus arteriosus

- Incidence is 10%.
- The DA begins to construct at birth and is completely closed in a few days and totally fibrosed in a few weeks. During fetal life, high levels of prostaglandin E1 are maintained in response to relatively low levels of O2 in fetal blood. This causes the smooth muscle of the DA to remain relaxed and thus keeps the DA patent. After birth, o2 tension rises because the lungs begin to function and PGE1 levels decline in response to this causing the DA to constrict.
- Prostaglandins are not hormones, but autocrine or paracrine secretions, which are locally acting messenger molecules. They differ from hormones in that they are not produced at a discrete site but in many places throughout the body. Also, their target cells are present in the immediate vicinity of the site of their excretion.

Patent ductus arteriosus

- ✓ Bradykinin (a small peptide) is probably also involved in causing the smooth muscle of the da to contract after birth. It is released by the lungs during their initial inflation. O2 is the most important factor in controlling closure of the DA in full term babies.
- ✓ Pulmonary resistance drops postnatally as the lungs inflate and if the da does not close, there is retrograde (reversed) flow in the da and blood flows from the aorta to the pulmonary arteries instead. This l-to-r shunt causes the pulmonary trunk, LA and LV to become volume overloaded. This can lead to enlargement of the LA and LV and aorta.
- ✓ Clinical features depend on size of patency: dyspnea, fatigue, diminished growth, eventual cardiac failure

Patent ductus arteriosus

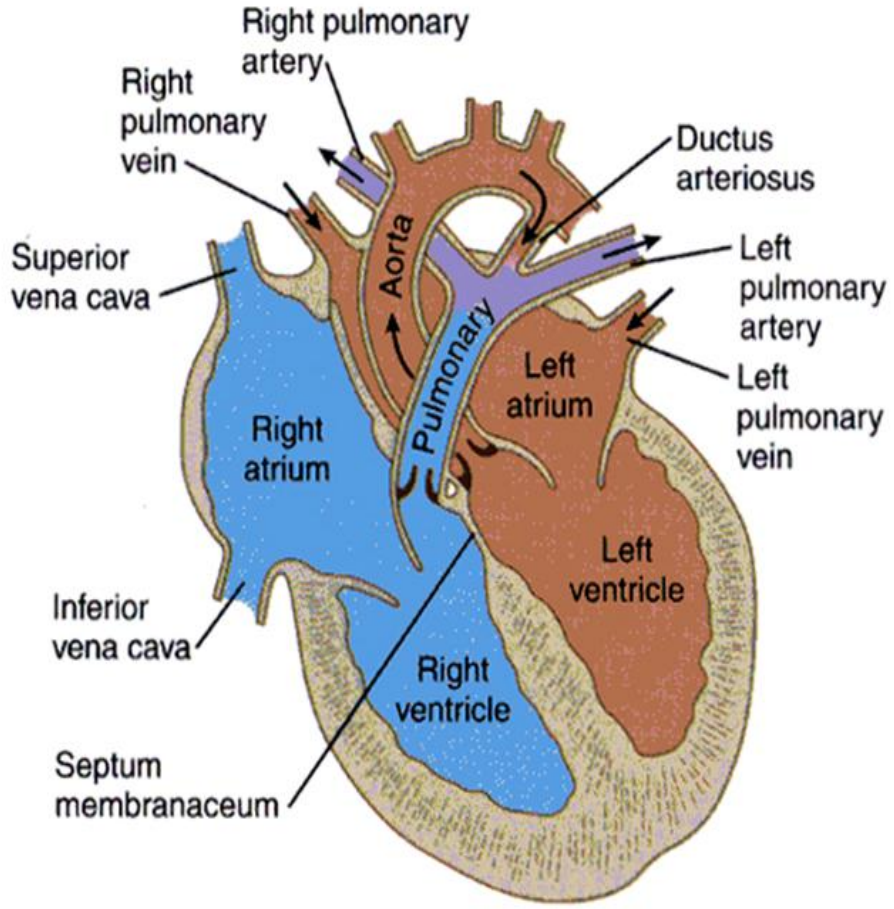
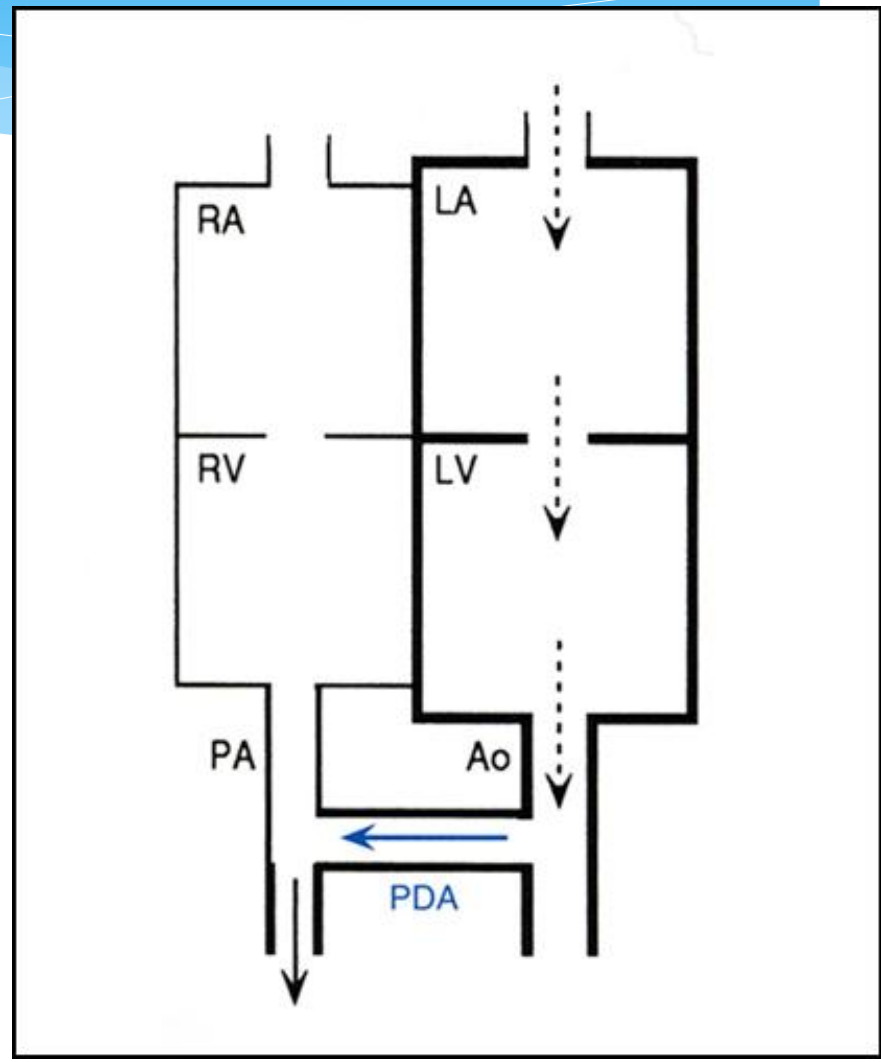
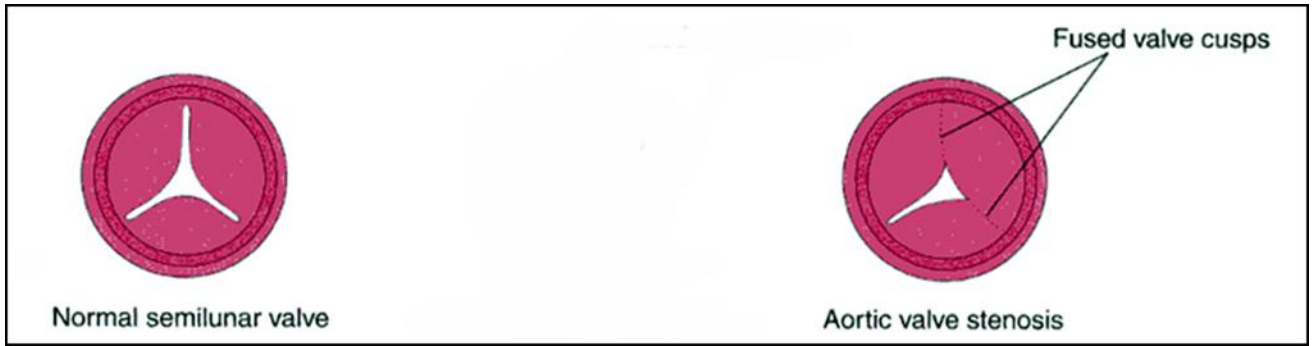


FIGURE 17-41 Patent ductus arteriosus showing the flow of blood from the aorta into the pulmonary circulation. Later in life, pulmonary hypertension may result, causing the reversal of blood flow through the shunt and cyanosis.



Aortic stenosis

- Incidence is 6%
- This defect probably has a neural crest origin since it is caused by asymmetrical septation of the outflow tract by the truncoconal ridges.
- As is usually manifest by abnormal development of the aortic valve (and 20% of the time is accompanied by coarctation of the aorta). The aorta is also narrower than usual.
- Symptoms include insufficiency in cerebral and coronary circulation, dizziness, angina, fatigue, etc.



Aortic stenosis

- The aortic valve is bicuspid instead of tricuspid and this causes the opening to be narrow and eccentric. This usually is not a major problem in infants, but as the cusps fibrose and calcify with age in adults, the stenosis becomes more pronounced.
- Since the valvular orifice is narrowed, lv pressure must increase to pump blood across the valve into the aorta. The LV hypertrophies in response to this increased work and pressure load. The high velocity of the blood impacting the aortic wall may cause dilation of the aorta distal to the valve.

Aortic stenosis

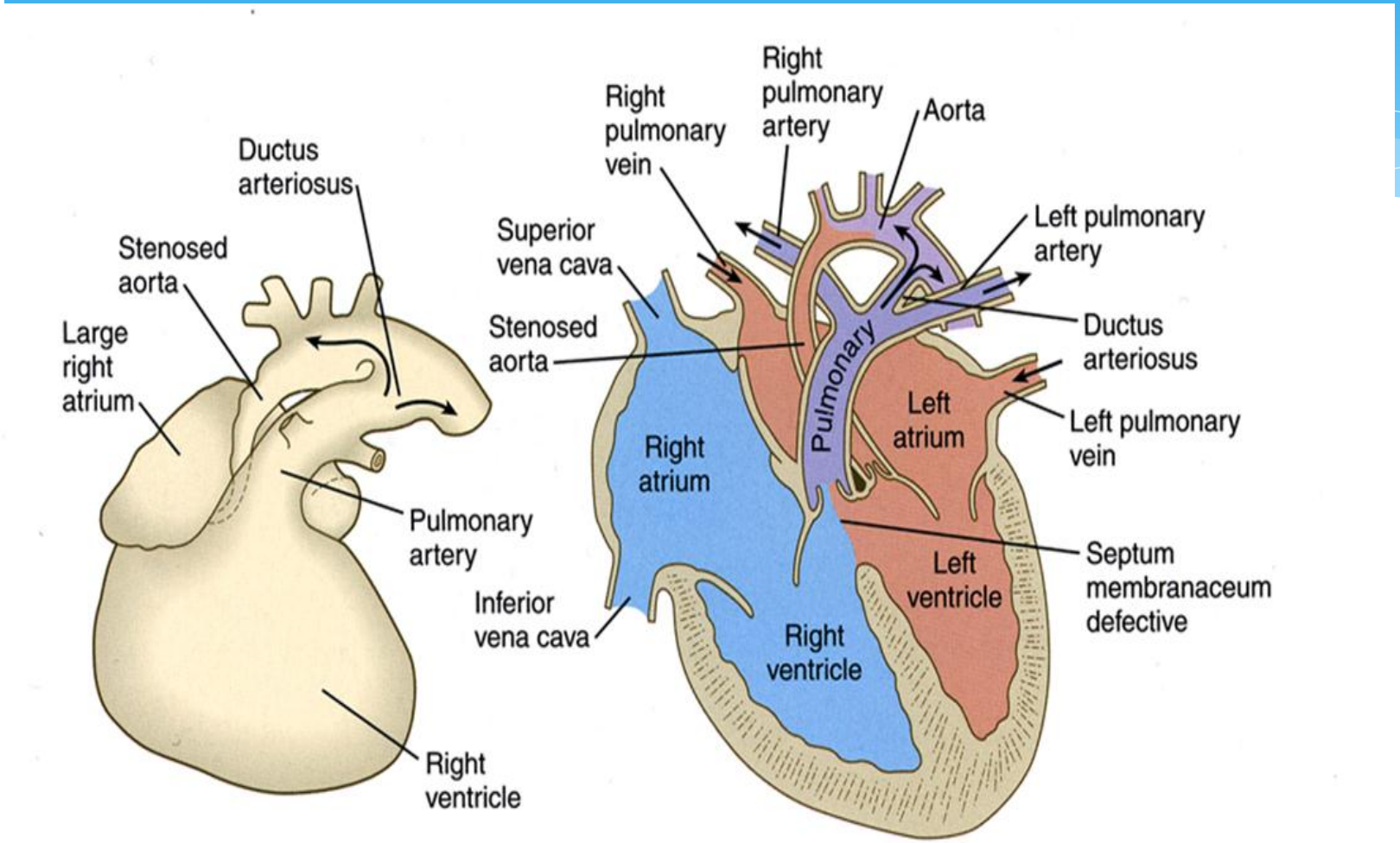
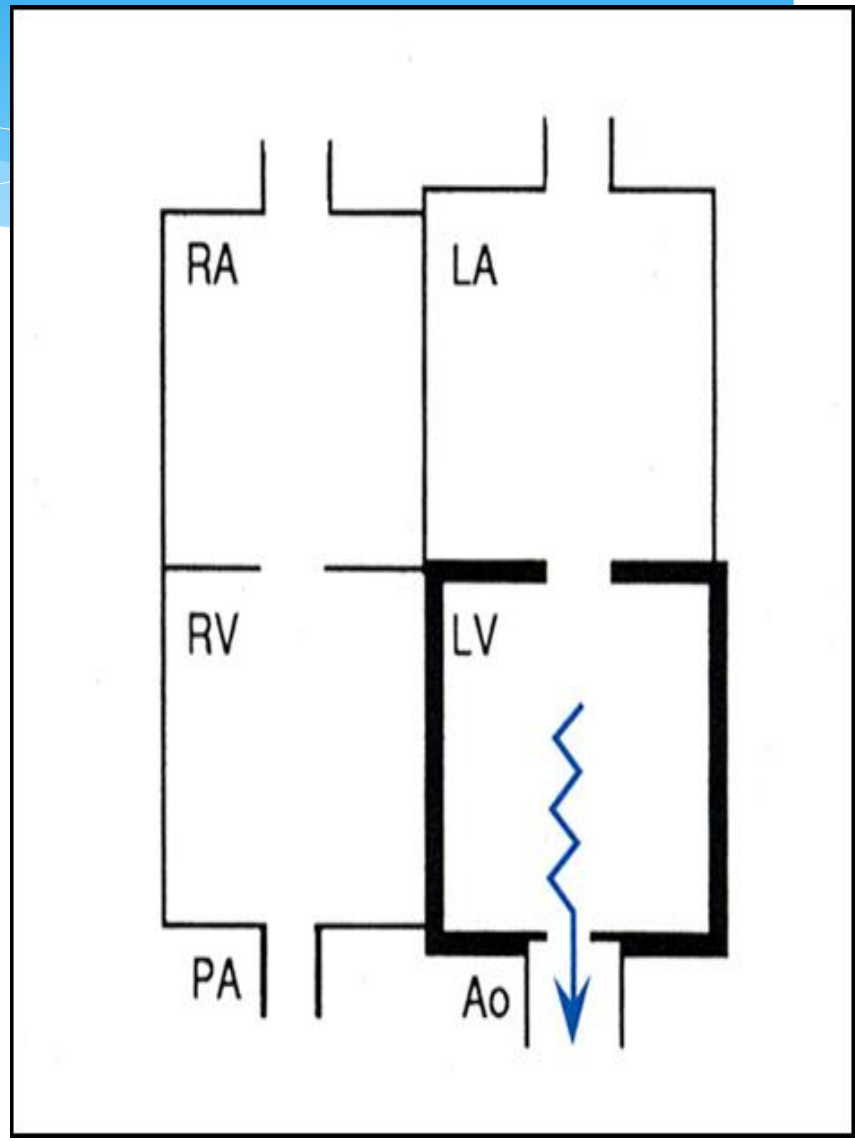


FIGURE 17-35 Aortic stenosis. In severe cases the ductus arteriosus commonly remains patent. *Right*, Mixed arterial and venous blood in the pulmonary artery is shown in purple. Initially, blood from the pulmonary trunk *(purple)* goes through the ductus arteriosus into the aorta, often leading to cyanosis.



Cyanotic defects

* Postnataly these result in right-to-left shunting of blood in the heart.
* Blood has not been oxygenated in the lungs, So cyanosis develops.
* More serious.
* These defects result from faulty rotation &/or partitioning of the truncus arteriosus with respect to the ventricles.
* Also considered neurocristopathies.

Tetralogy of Fallot

Four anomalies:

- **Pulmonary stenosis**
- **VSD**
- **Dextroposition of the aorta**
- **Hypertrophy of right ventricle**

- ✓ Most common cyanotic defect in young children
- ✓ Caused by asymmetrical fusion of truncoconal ridges resulting in misalignment of the ventricular outflow tract and the aortic and pulmonary valves
- ✓ If untreated, only 50% survival rate past age 2.5 years

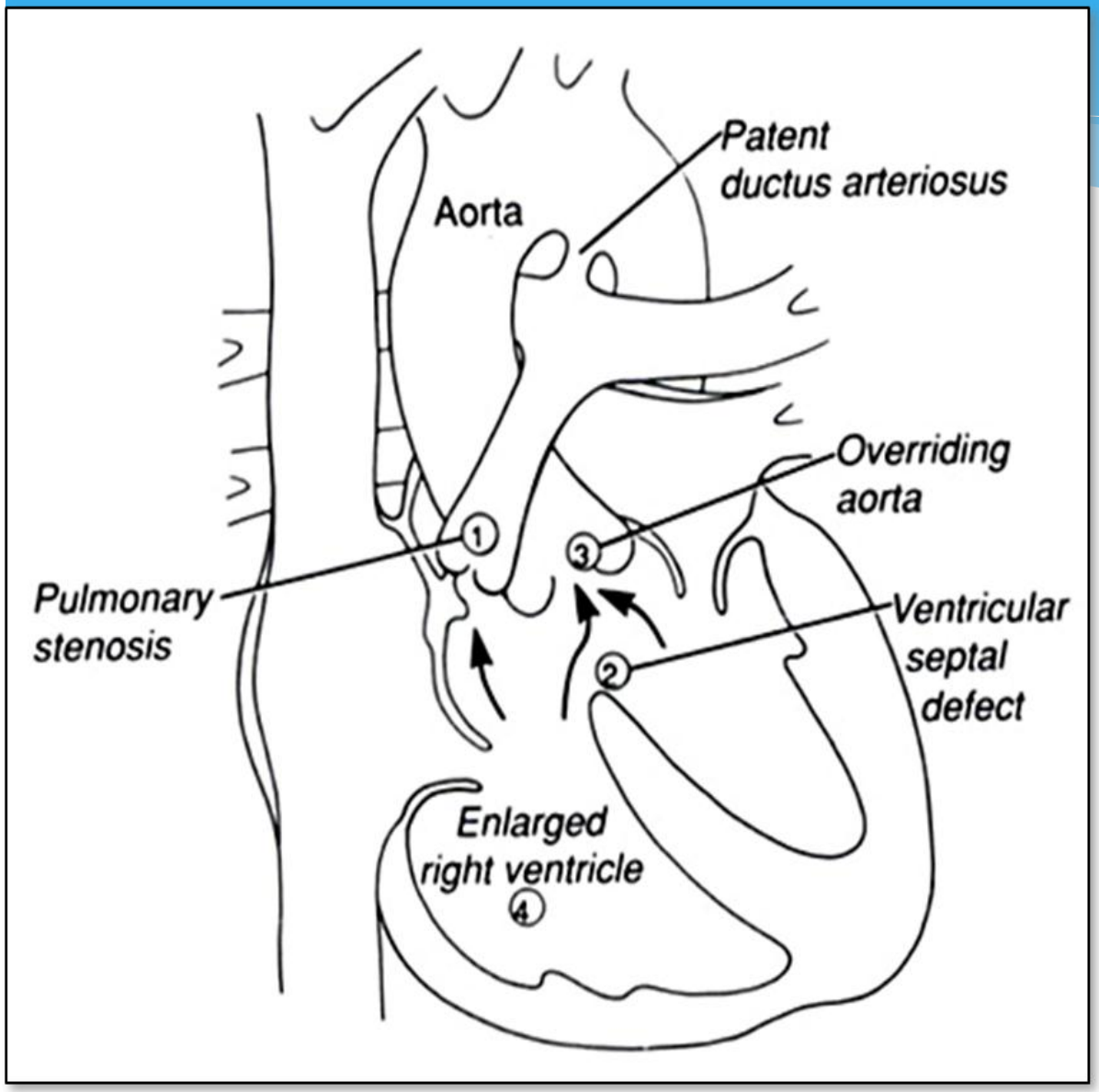
Tetralogy of Fallot

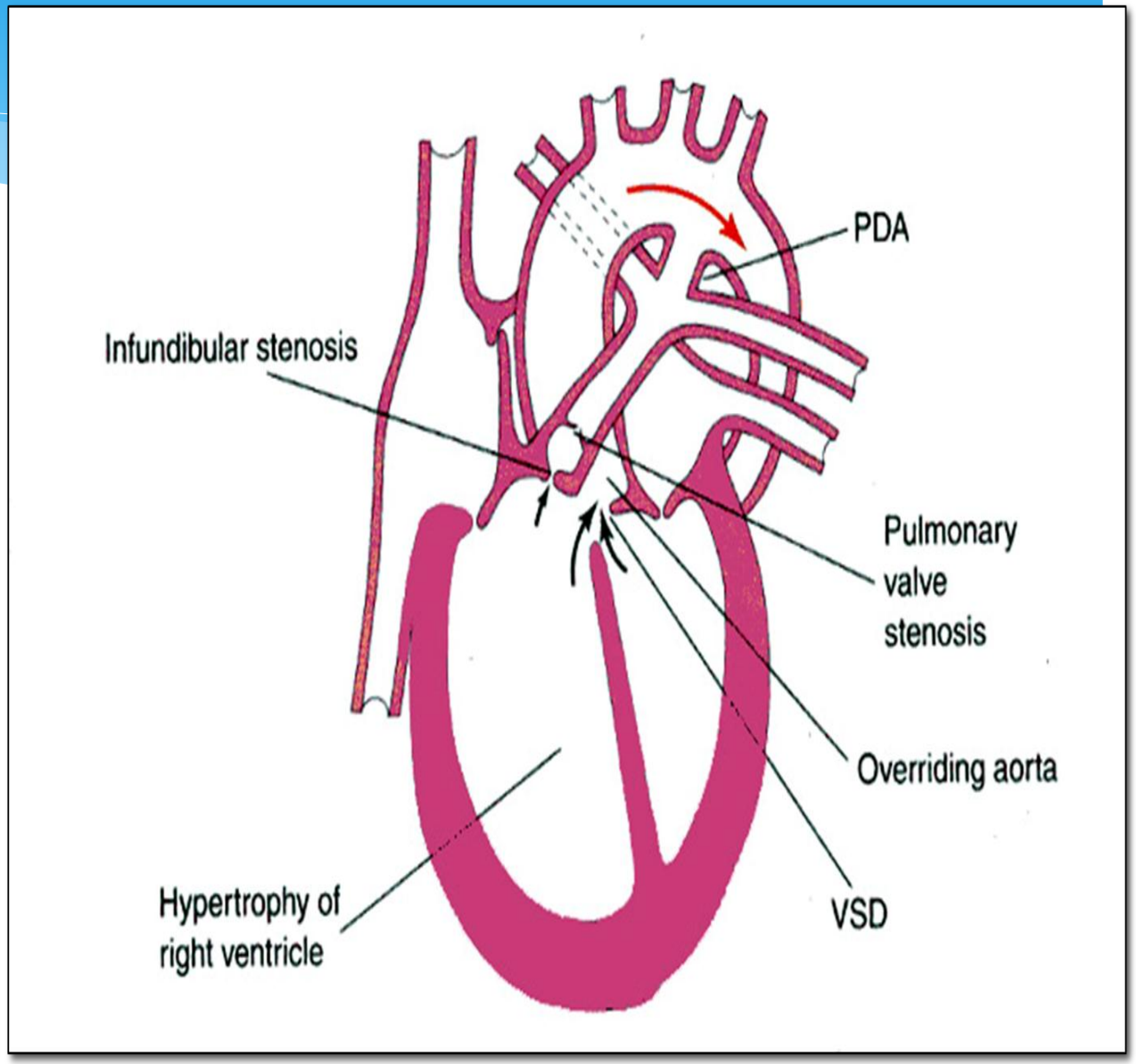
- Clinical picture dependent on degree of right ventricular outflow obstruction.
- The increased resistance by the pulmonary stenosis causes deoxygenated blood blood returning from the systemic veins to be diverted from the rv, through the vsd, to the lv, and into the systemic circulation through the aorta resulting in systemic hypoxemia and cyanosis.
- T of F is a serious heart problem because it obstructs blood from reaching the lungs. Because of the pulmonary stenosis and wider than normal and more right located aortic opening, some poorly oxygenated right ventricular blood leaves via the enlarged aorta. In some babies, the obstruction is severe causing significant cyanosis and very low oxygen levels soon after birth. There is also digital clubbing.

Tetralogy of Fallot

- ✓Although some children are not cyanotic in the first years of life, most babies with T of F become cyanotic at 3-6 months
- ✓Babies with the most severe pulmonary stenosis may become extremely cyanotic within the first few days after birth.
- ✓The degree of patency of the da after birth (the more the better) probably determines the severity of the cyanosis.
- ✓Surgery to correct t of f involves closing vsd & opening narrowed pulmonary trunk.
- ✓T of f is often associated with other cardiac defects.

Tetralogy of Fallot

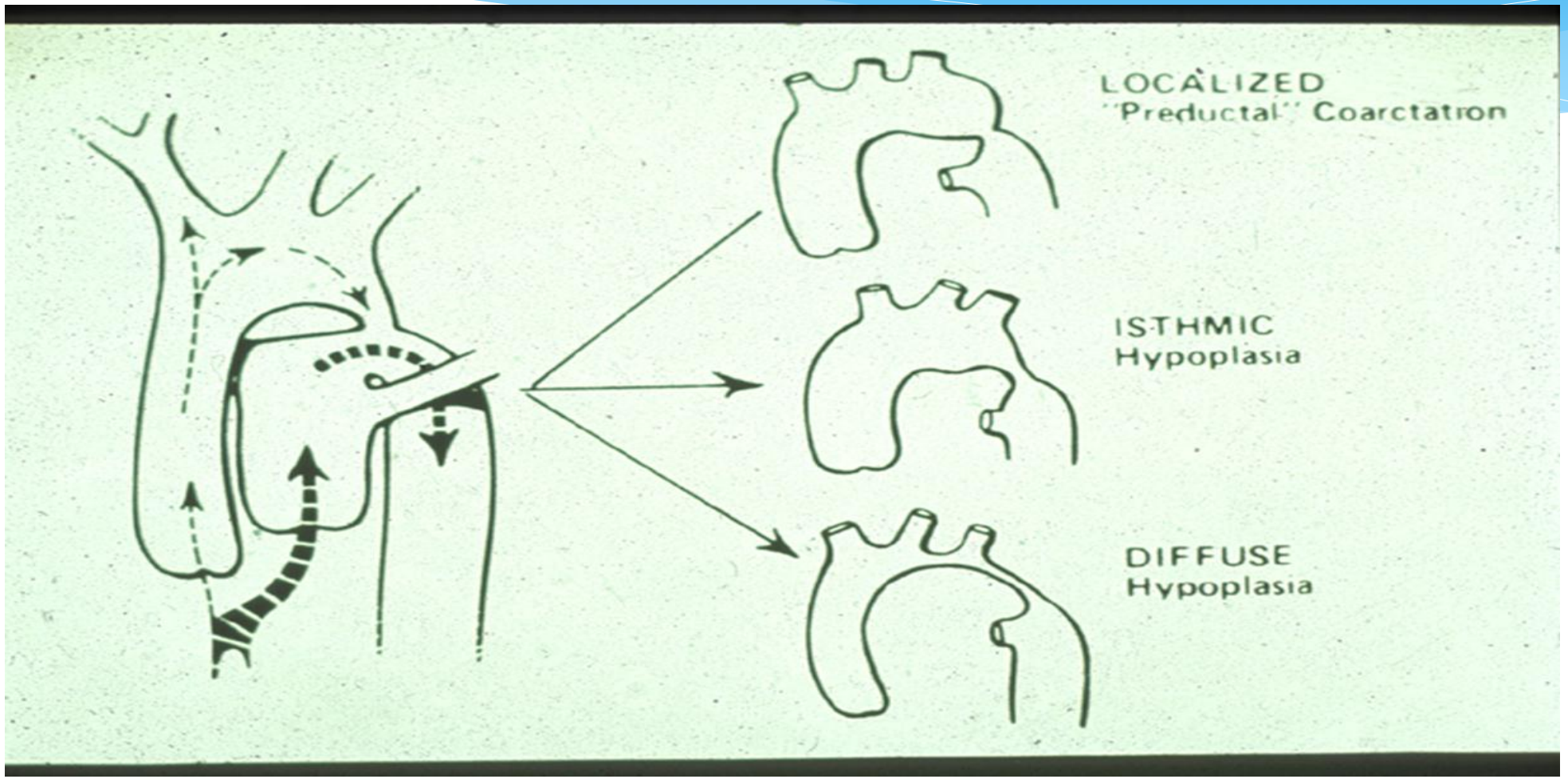




Coarctation of the Aorta

- ✓Aortic constriction
- ✓Pulse – good on the hand, non in the legs.
- ✓Systolical murmur left side parasternal , on the back under the scapulae.
- ✓Abp examined on both hands (differences during coa+ anomalia of subclavicullar veins).
- ✓ECG hypertrophy of left venricle.
- ✓Echocardioscopy

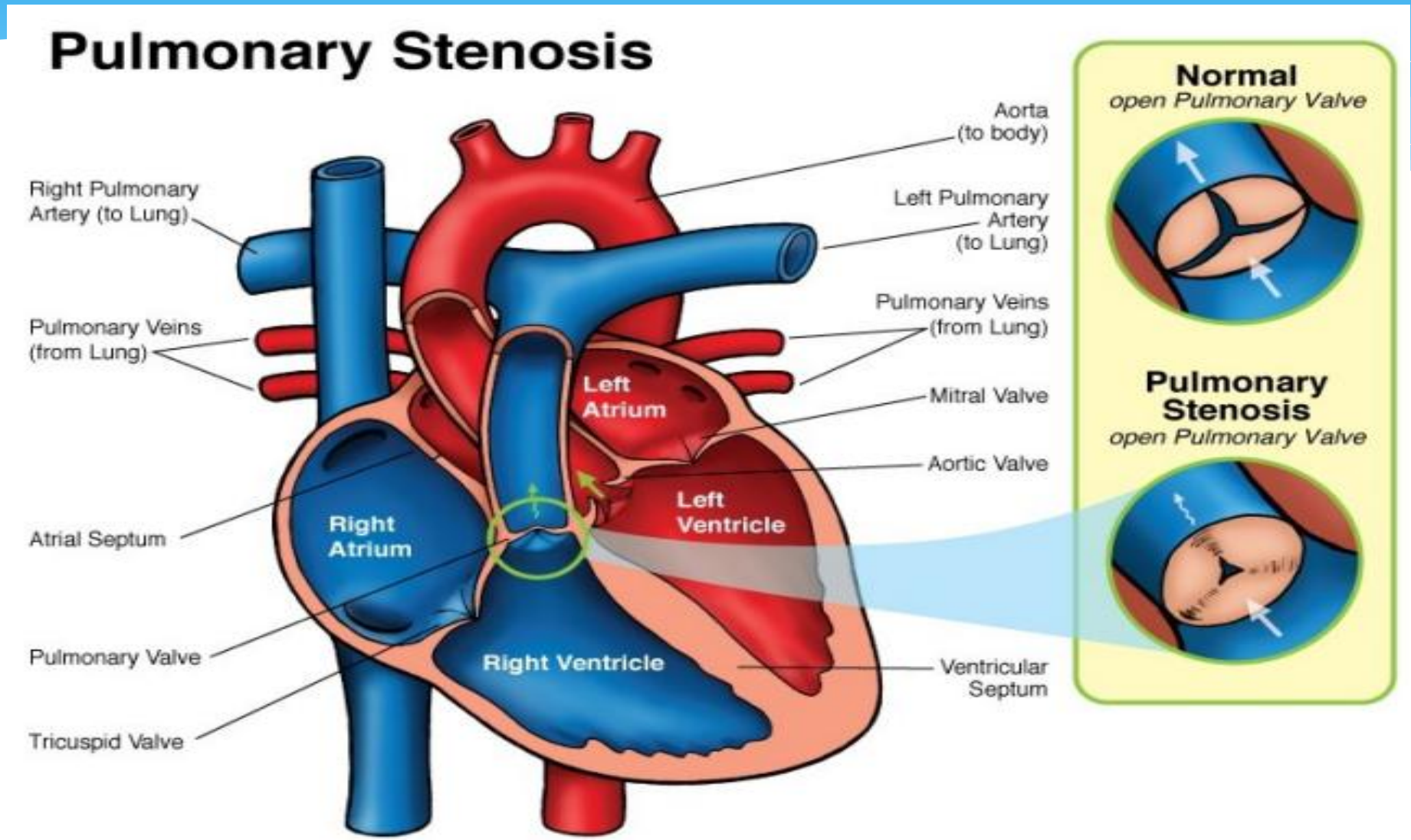
Coarctation of the Aorta



Critical Pulmonary Valve Stenosis

* Clinic of heart insufficiency – from first month of life;
* Clinical features –septic state , hypotension, soft pulse, pale color of the skin; systolic murmur– II intercostal space right side, III intercostal space left side , irradiation on the neck, galop rytm
* Tahypnoe, dyspnea;
* Eho-CG– anatomy of aortic valves.
* Ecg – hypertrophy of right ventricules;
* X-ray – cardiomegaly

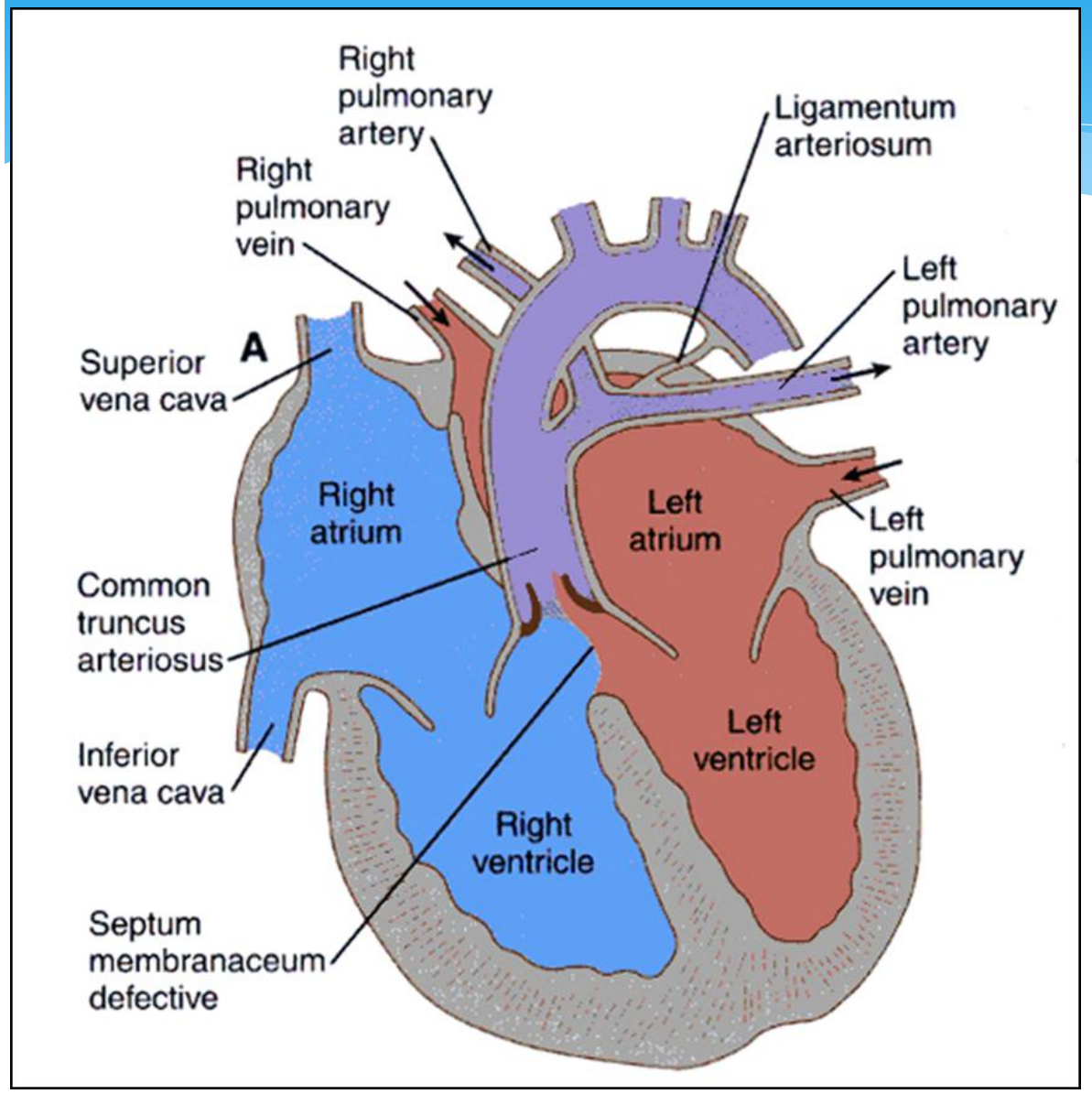
Critical Pulmonary Valve Stenosis

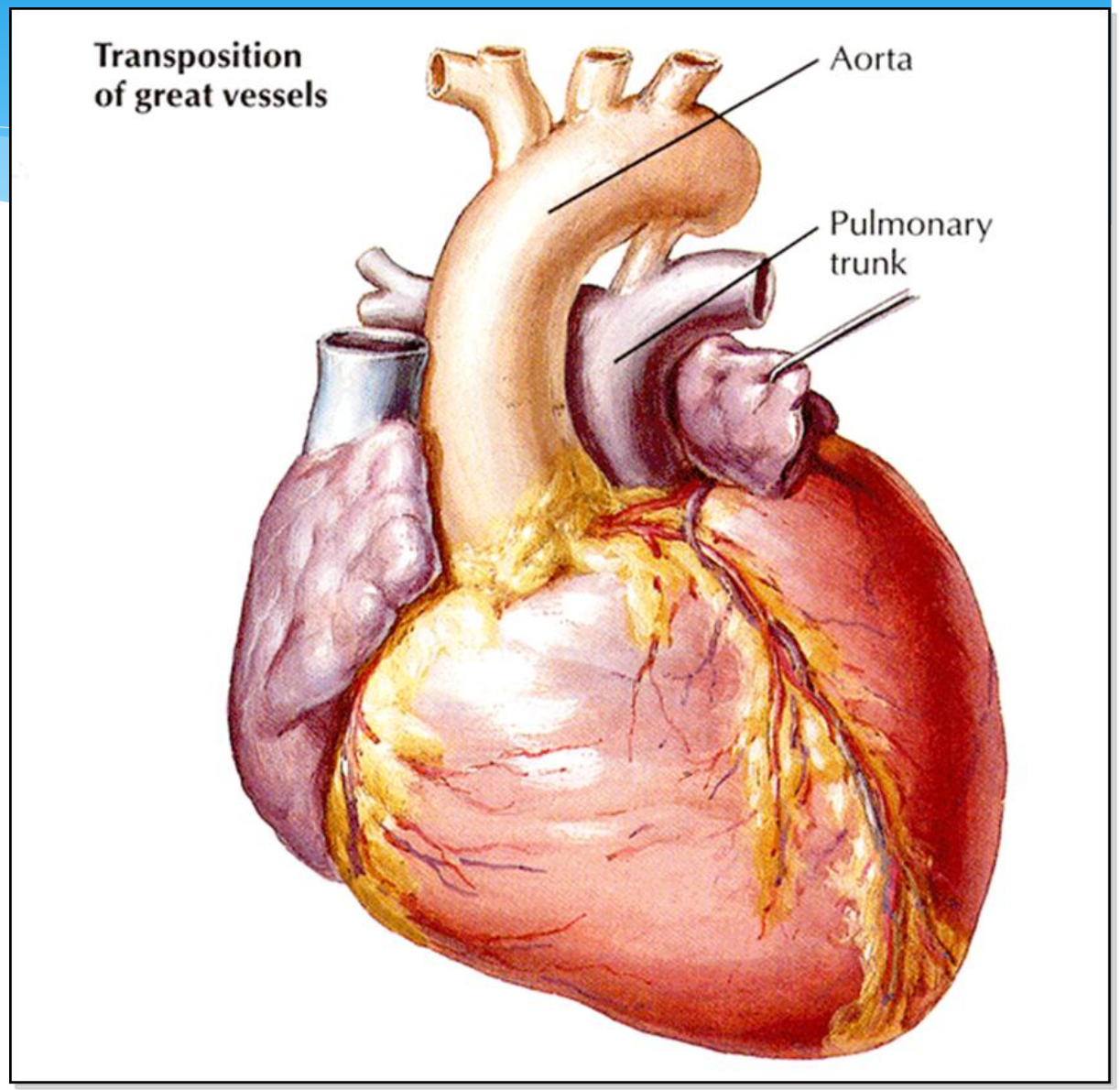


Transposition of the great vessels (arteries)

- Most common cause of cyanosis in newborns
- Truncoconal ridges fail to spiral as they divide the outflow channel
- Two circulations thru heart operate independently
- Compatible with life only if atrial and ventricular septal defects and a patent DA are present
- It is only the truncus arteriosus region of the aorta and pulmonary arteries that is undivided because the portions of the vessels distal to the truncus have different origins --- the aorta is formed from the 4th left aortic arch and the pulmonary arteries from the 6th L and R aortic arches.
- The persistent TA usually overrides the VSD. Since blood in the aorta is a mixture of that from both ventricles, cyanosis is inevitable.
- Usually only one set of semilunar valves.

Transposition of the great vessels (arteries)

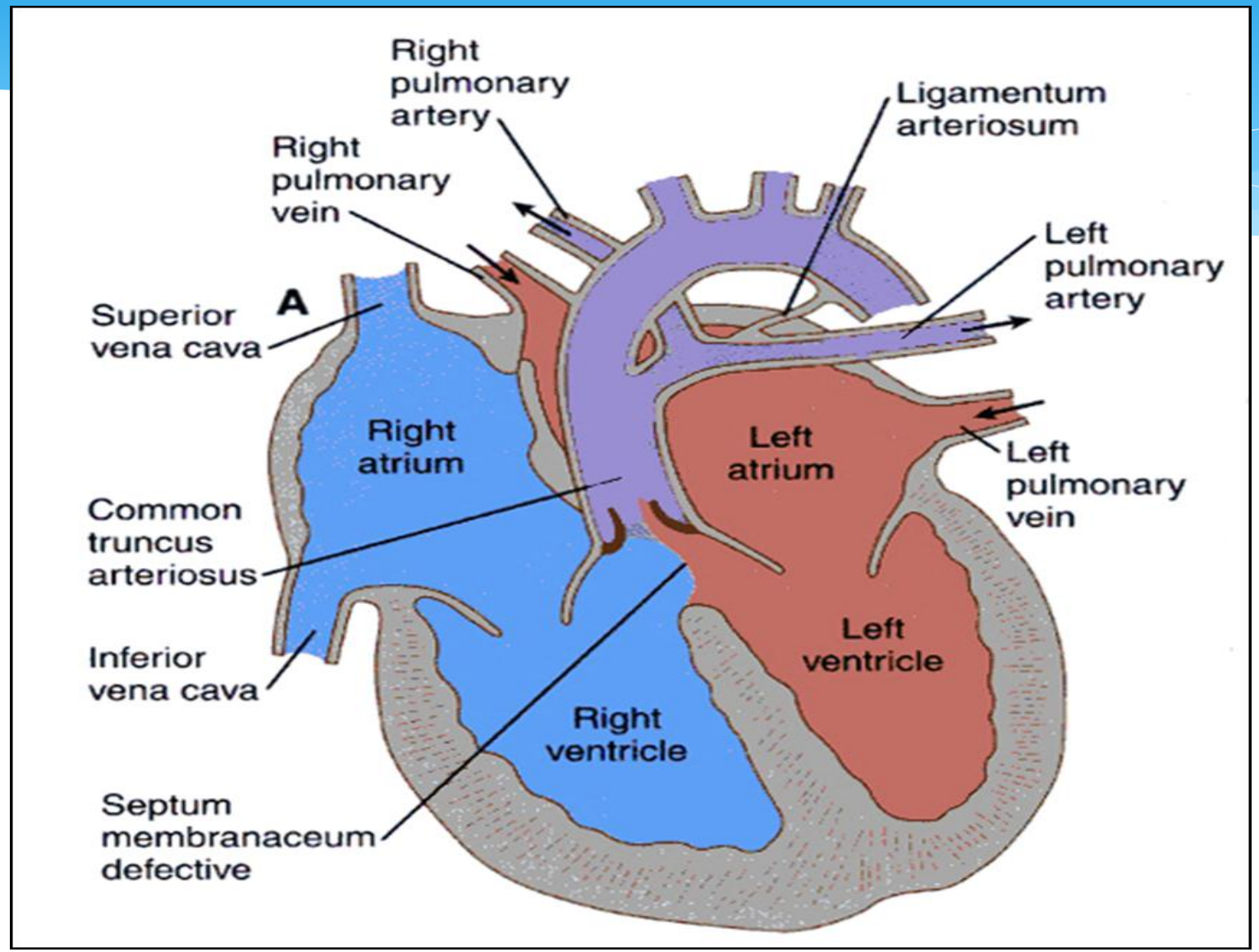




Common arterial trunk

- ✓ Caused by lack of partitioning of the outflow tract by the truncoconal ridges
- ✓ Single outflow vessel leaves ventricles
- ✓ Membranous iv septum frequently lacking because it is also formed in part by t-c ridges
- ✓ W/o treatment, 60-70% mortality by 6 months

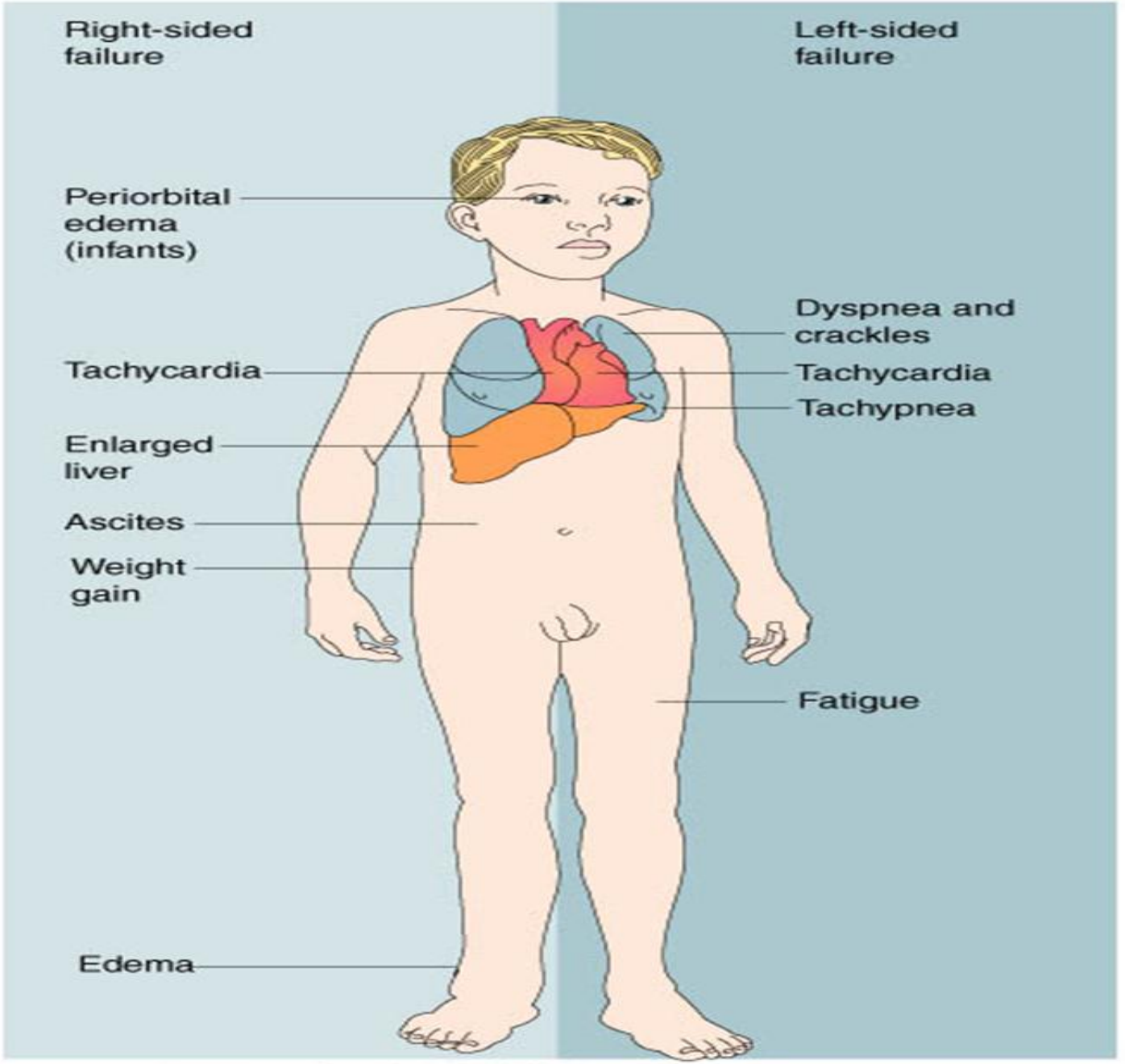
Common arterial duct



ACQUIRED HEART DISEASE

* Congestive Heart Failure
* Tachycardia, tachypnea
* Right sided: increased venous pressure, hepatomegaly
* Left sided: dyspnea, crackles (rales), cyanosis, and, eventually, ride sided failure
* Therapeutic management: Reduce workload of the heart using diuretics, inotropics, and vasodilators

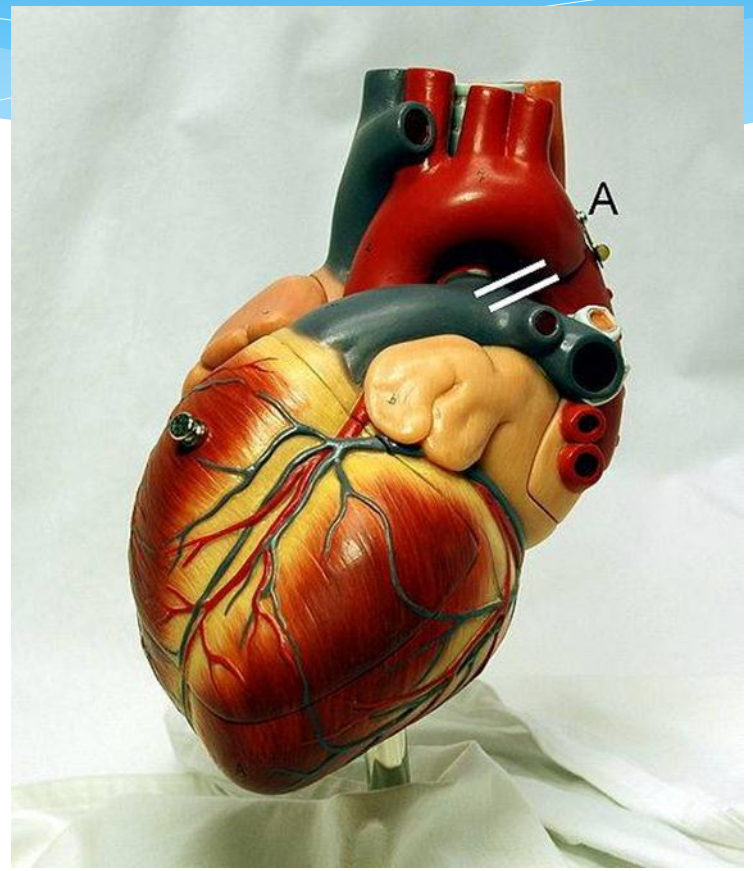
ACQUIRED HEART DISEASE



Assessment 41-2 Assessing the Child With Heart Failure

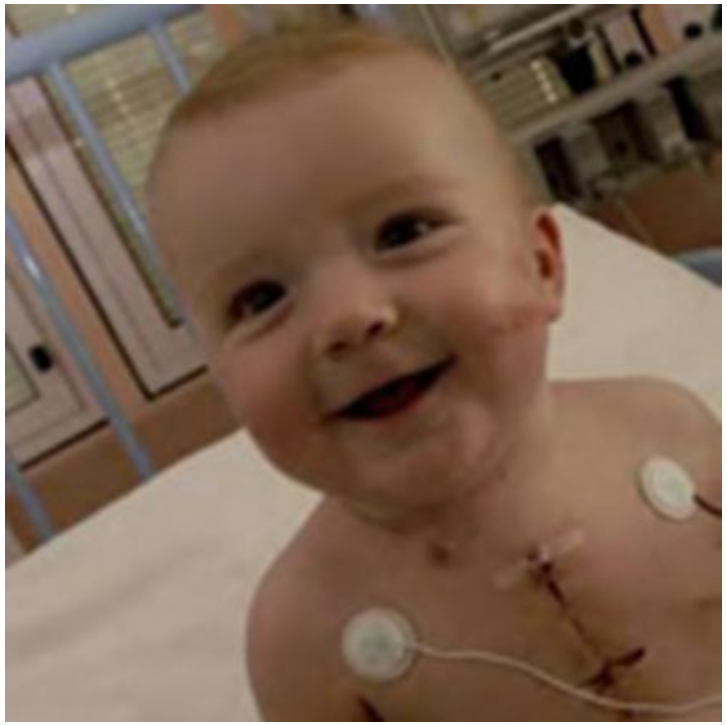
Tests Commonly Used To Diagnosis Congenital Heart Defects

* Blood and urine tests
* ECG (Electrocardiogram)
* Echocardiogram
* Cardiac magnetic resonance imaging (MRI)
* Stress tests
* Chest X Ray
* Pulse Oximetry
* Cardiac Catheterization



Preventing Birth Defects

- Stop smoking
- Avoid drinking alcohol while pregnant
- Take a daily vitamin containing folic acid
- Check with your doctor to make sure any medication (over-the-counter or prescription) is safe to take during pregnancy
- Stop use of any illegal or "street" drugs




Thank you for your attention!

Literature, was used in the lecture

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* Інтернет – ресурси:
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* http://www.who.int/child_adolescent_health/documents/chs_cah_98_1a/en/index.html
* **Key documents WHO**
* A systematic review of the effectiveness of shortening Integrated Management of Childhood Illness guidelines training (2018)
  This is the final report of the Integrated Management of Childhood Illness (IMCI) strategy which has been shown to improve care for ill children in outpatient settings in developing countries. IMCI chart booklet - standard (2018) This revised version of the chart booklet includes new sections on the management of illness in the first week of a child's life. This chart booklet is a job-aid to be used by health workers mostly at first-level health facilities.
* 2011 Manual on paediatric HIV care and treatment for district hospitals 2010 WHO recommendations on the management of diarrhoea and pneumonia in HIV-infected infants and children