RABBI ZIDNI ILMA

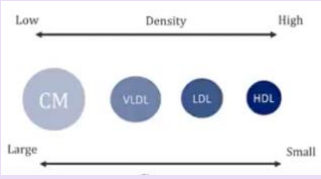
RABBI INNI LIMA ANZALTA ILAYYA MIN KHAYRIN FAQEER

CVS IMP OSPES AND POINTERS

- Apo-B48 is only on chylomicron, required for secretion from enterocyte
- Lipid transport is by two main lipoproteins: VLDL AND HDL

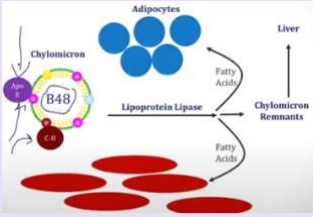
Lipids

- Mostly carbon and hydrogen
- Not soluble in water
- Many types:
  - Fatty acids
  - Triglycerides
  - Cholesterol
  - Phospholipids
  - Steroids
  - Glycolipids



Apolipoproteins

- Proteins that bind lipids
- Found in lipoproteins
- Various functions:
  - Surface receptors
  - Co-factors for enzymes



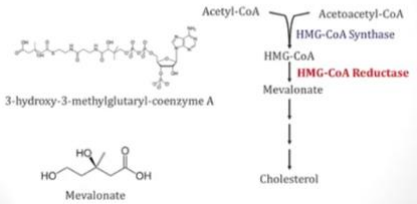
Lipoprotein Lipase

LPL

- **Extracellular** enzyme
- Anchored to **capillary walls**
- Mostly found in adipose tissue, muscle, and heart
  - Not in liver → liver has hepatic lipase
- Converts triglycerides → fatty acids (and glycerol)
- Fatty acids used for storage (adipose) or fuel
- Requires **apo C-II** for activation

Cholesterol Synthesis

- Only the liver can synthesize cholesterol



Absorption of Fatty Acids

- Fatty acids → Triglycerides
- Packaged into **chylomicrons** by intestinal cells
- To lymph → blood stream

Absorption of Cholesterol

- **Cholesteryl esters** formed in enterocytes
- Acyl-CoA cholesterol acyltransferase (**ACAT**)
- Packaged into chylomicrons by intestinal cells
- To lymph → blood stream

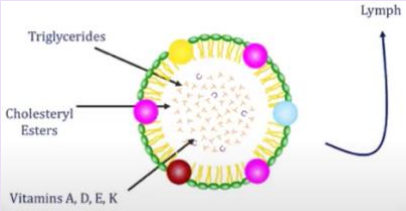
Chylomicron Remnants

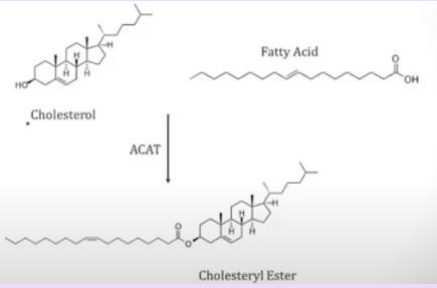
- Apo-E receptors on **liver**
- Take up remnants via receptor-mediated endocytosis
- Usually only present after meals (clear 1-5hrs)
- Milky appearance

Chylomicrons

Summary

- Secreted from enterocytes with Apo48
- Pick up Apo C-II and ApoE from HDL
- Carry triglycerides and cholesteryl esters
- Deliver triglycerides to cells
  - Lipoprotein lipase stimulates (C-II co-factor) breakdown
- Return to liver as chylomicron remnants
  - ApoE receptors on liver





- **C-II**
  - Co-factor for lipoprotein lipase
  - Carried by: Chylomicrons, VLDL/IDL
- **Apo E**
  - Binds to liver receptors
  - Required for uptake of remnants
- Both from **HDL**

Abetalipoproteinemia

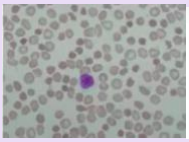
- Autosomal recessive disorder
- Defect in MTP
  - Microsomal triglyceride transfer protein
- MTP forms/secretes lipoproteins with apo-B
  - Chylomicrons from intestine (B48)
  - VLDL from liver (B100)

Clinical Features

- Presents in infancy
  - Steatorrhea
  - Abdominal distension
  - Failure to thrive
- Fat-soluble vitamin deficiencies
  - Especially vitamin E (ataxia, weakness, hemolysis)
  - Vitamin A (poor vision)
- Lipid accumulation in enterocytes on biopsy

Lab Findings

- Low or zero VLDL/IDL/LDL
- Very low triglyceride and total cholesterol levels
- Low vitamin E levels
- Acanthocytosis
  - Abnormal RBC membrane lipids



Foam Cells

- **Macrophages** filled with cholesterol
- Found in atherosclerotic plaques
- Contain LDL receptors and LDL

VLDL

- **Transport** lipoprotein
- Secreted by liver (nascent VLDL)
- Carries triglycerides, cholesterol to tissues

- ApoE2
  - Decreased risk of Alzheimer's
- ApoE4
  - Increased risk of Alzheimer's

HDL

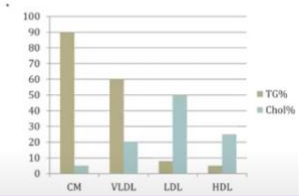
- **Scavenger** lipoprotein
  - Brings cholesterol back to liver
  - "Reverse transport"
- Secreted as small "nascent" HDL particle
- Key apolipoproteins: A-I, C-II, ApoE

- Lecithin-cholesterol acyl transferase (**LCAT**)
  - Esterifies cholesterol in HDL; packs **esters** densely in core
  - Activated by **A-I**
- Cholesteryl ester transfer protein (**CETP**)
  - Exchanges esters (HDL) for triglycerides (VLDL)
- Carries cholesterol back to liver

Hepatic Lipase

- Found in **liver** capillaries
- Similar function to LPL (releases fatty acids)
- Very important for IDL → LDL conversion
- Absence HL → absence IDL/LDL conversion

Lipoprotein Composition



Pancreatitis

- Elevated triglycerides (>1000) → acute pancreatitis
- Exact mechanism unclear
- May involve increased **chylomicrons** in plasma
  - Chylomicrons usually formed after meals and cleared
  - Always present when triglycerides > 1000mg/dL
  - May obstruct capillaries → ischemia
  - Vessel damage can expose triglycerides to pancreatic lipases
  - Triglycerides breakdown → **free fatty acids**
  - Acid → tissue injury → pancreatitis

Lipid Measurements

- Total Cholesterol
- LDL-C
- HDL-C
- TG

Friedewald Formula

$$LDL\text{-}C = \text{Total Chol} - \text{HDL-}C - \frac{TG}{5}$$

Hyperlipidemia

- Elevated total cholesterol, LDL, or triglycerides
- Risk factor for coronary disease and stroke
- Modifiable – often related to lifestyle factors
  - Sedentary lifestyle
  - Saturated and trans-fatty acid foods
  - Lack of fiber

Signs of Hyperlipidemia

- Xanthomas
  - Plaques of lipid-laden histiocytes
  - Appear as skin bumps or on eyelids
- Tendinous Xanthoma
  - Lipid deposits in tendons
  - Common in Achilles
- Corneal arcus
  - Lipid deposit in cornea
  - Seen on fundoscopy

Klaus D. Peter, Gummersbach, Germany

Selected Causes of Hyperlipidemia

Nephrotic syndrome (LDL)
Alcohol use (TG)
Pregnancy (TG)
Beta blockers (TG)
HCTZ (TC, LDL, TG)

Familial Dyslipidemias

- Type I – **Hyperchylomicronemia**
- Severe LPL dysfunction
  - LPL deficient
  - LPL co-factor deficient (apolipoprotein C-II)
- Recurrent pancreatitis
- Enlarged liver, xanthomas
- Treatment: Very low fat diet
  - Reports of normal lifespan
  - No apparent ↑risk atherosclerosis

Familial Dyslipidemias

- Type I – **Hyperchylomicronemia**
- Autosomal recessive
- ↑↑↑TG (>1000; milky plasma appearance)
- ↑↑↑ chylomicrons

- Type II - **Familial Hypercholesterolemia**
  - Autosomal **dominant**
  - Few or zero LDL receptors
  - **Very high LDL** (>300 heterozygote; >700 homozygote)
  - Tendon xanthomas, corneal arcus
  - **Severe atherosclerosis** (can have MI in 20s)

- Type III – **Familial Dysbetalipoproteinemia**
  - Apo-E2 subtype of Apo-E
  - Poorly cleared by liver
  - Accumulation of chylomicron remnants and VLDL
    - (collectively know as β-lipoproteins)
  - Elevated total cholesterol and triglycerides
  - Usually mild (TC>300 mg/dl)
  - Xanthomas
  - **Premature coronary disease**

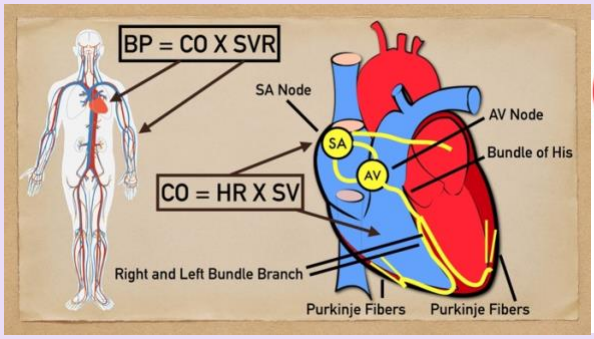
Type IV **Hypertriglyceridemia**

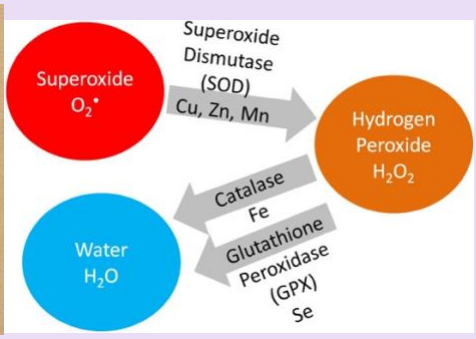
- Autosomal **dominant**
- VLDL overproduction or impaired catabolism
- **↑TG (200-500)**
- ↑VLDL
- Associated with diabetes type II
- Often diagnosed on routine screening bloodwork
- Increased coronary risk/premature coronary disease

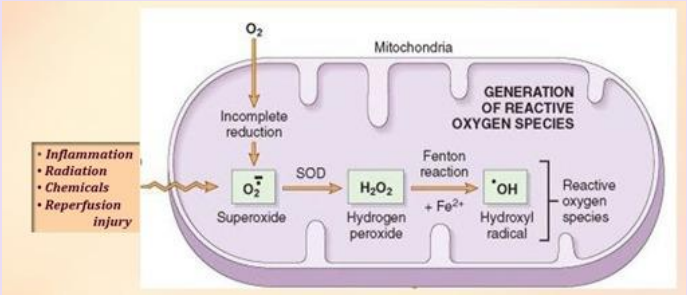
- inferior :RCA
  lateral : LXA
  septal/ant:LAD
- MAP calculated by : (2DP+SP)/3
- Chylomicron and VLDL is mainly TG
- LDL is mainly Cholesterol
- HDL is mainly protein
- Highest TG in chylomicron
- Highest protein in HDL
- Lowest cholesterol in Chylomicron
- In enterocyte, ACAT converts cholesterol to cholesteryl esters
- In HDL, LCAT does this
- 6 groups of lipids :FACTSP (FA,ACYLGYCEROL,CHOLESTROL, TERPENES,SPHINGOLIPIDS,PROSTAGLANDINS)
- lipid lowering drugs are STATINS
- 35% of cholesterol is hdl
- HDL-C (GOOD),LDL-B(BAD)
- In TOF ,vsd appears first
- L-type Ca channels are also called calcium-sodium channels which allow both Ca and Na to flow into the cell causing prolonged depolarization ‘plateau on the graph'

- **Velocity of signal conduction thru atrial and ventricular muscles:** 0.3-0.5 m/sec
- **Velocity of signal conduction thru the conductive system of the heart:** 4m/sec
- **Ventricular refractory period:** 0.25 to 0.30 sec
- **Atrial refractory period:** 0.15 sec **Relative refractory period:** 0.05 sec
- Ca ions coming through L-type Ca channels in T tubules and binding to troponin to cause contraction play an important role in maintaining the strength of cardiac muscle contraction because sarcoplasmic reticulum in cardiac myocytes are less well developed than in skeletal muscle and store less Ca
- Dynamic exercise: DBP does not increase balkay decreases
- Static exercise: DBP increases
- Cardiac Index: is the amount of blood pumped out of ventricle per minute per square meter of body surface area.
- Systolic is largely determined by stroke volume
  Diastolic is largely determined by total peripheral resistance
- Less peripheral resistance(vasodilation) =lower diastolic pressure
- More peripheral resistance(vasoconstriction)= higher diastolic pressure
- More stroke volume more systolic peak
- Height is by stroke volume and depth is by tpr
- Pulse pressure also determined by SV
- Delay: To allow ventricular filling
- 0.03s from SV to AV
- 0.09 with AV
- 0.04 in AV bundle
- 0.09+0.04=0.13 allows ventricular filling
- TERATOLOGY OF FALLOT

Pulmonary stenosis
Overriding of aorta
Hypertrophy of the right ventricle
Interventricular septal defect







MR = holosystolic
MS = opening snap rumbling diastolic 1:43 am

AS = crescendo decrescendo systolic
AR = blowing diasyolic murmur (decrescendo) 1:43 am

If MR occurs with mitral valve prolapse to apna ejection click bhi sunayi degi 1:43 am

S3 sound apna acute heart failure 1:43 am

S4 sound apna ventricular hypertrophy (stiffness) 1:43 am

peRsistant s2 split in rbbb and htn 1:43 am

ParadoxicaL s2 in lbbb and cardiomyopathy 1:43 am

Fixed S2 in asd with systolic ejection click 1:43 am

Inspiration me apna pv late bandh hota h 1:43

Systolic ejection murmur
In AS and HCM 1:43 am

- Rheumatic Myocarditis:
  - Aschoff Bodies: immune-mediated granulomas around perivascular connective tissues (swollen **collagenous fibres**, immune cells (Anitschkow & Aschoff Cells)
  - Anitschkow/Caterpillar Cells: macrophages with prominent caterpillar nucleus chromatin
  - Aschoff Cells: when Anitschkow cells enlarge & become multinucleated
  - Myocardium becomes flabby → poorly contractile → CHF (RVF more severe) (death in acute) (diagnose in echo – larger cardiac shadow)
  - No long-term complications

HEART - PERICARDIUM

PERICARDIUM

- Outer layer - Fibrous
  Blends with adventitia of aorta, pulmonary trunk, superior vena cava (not inferior vena cava), central tendon of diaphragm
- Inner layer - Serous
  - Visceral
  - Parietal
- Blood: pericardiacophrenic & internal thoracic
- Nerve: Phrenic to fibrous and parietal serous layers
  Sympathetic for pain & muscles & vessels of heart
  Nil to visceral layer

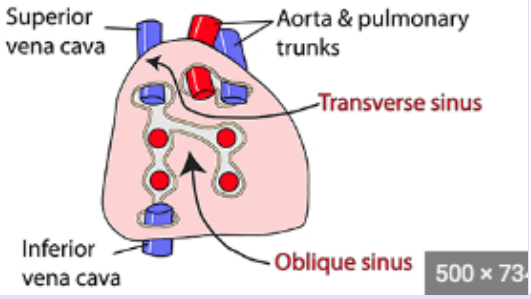
SINUSES OF PERICARDIUM

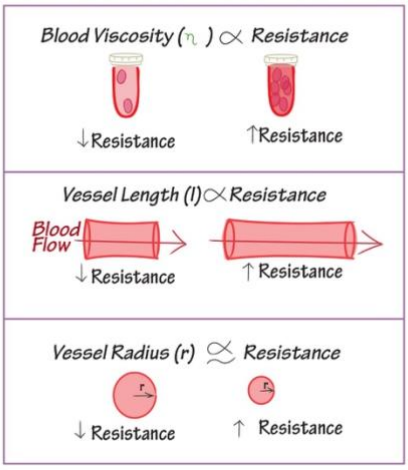
Transverse

Lies between the pulmonary artery and aorta anteriorly and pulmonary veins and superior vena cava posteriorly

Oblique

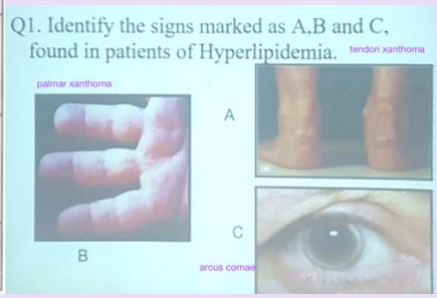
This is a pouch of pericardium between the pulmonary veins at the base of the heart where the visceral pericardium is reflected off the vessels to become the parietal pericardium

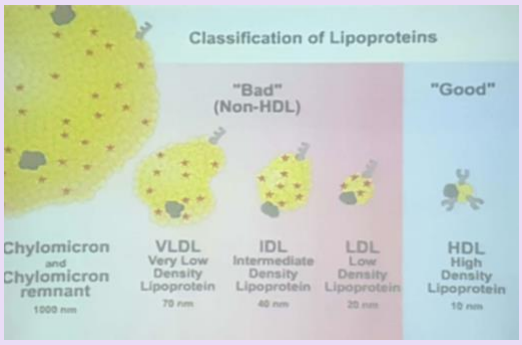




Anterior aortic sonus is for right coronary artery posterior for left coronary

	Medium-sized Artery	Medium-sized Vein
Thickness	Thick wall.	Thin wall.
Lumen	• **Narrow** and Rounded • **Doesn't collapse** after death • **Hasn't blood** after death.	• **Wide.** • **Collapses** after death. • **Has blood** after death.
Valves	Absent.	Present.
T. intima	• **Thick** and **Folded.** • **Rich** in **elastic fibers.** • **Has** clear **IEL.**	• **Thin** and **not folded.** • **Poor** in **elastic fibers.** • **No IEL.**
T. media	• **Thick** • Made of **smooth muscles** and **elastic fibers.** • **EEL may be present.**	• **Thin.** • Made of **smooth muscles, few elastic fibers.** • **No EEL.**
T. adventitia	• **Thin.** • **Rich** in **elastic fibers.**	• **Thick.** • **Few elastic fibers.**





2. Endocardium: It consists of:

- Inner layer of endothelium.
- Middle layer of subendothelial C.T. with smooth muscle cells.
- Deeper layer of C.T., is called subendocardial layer. It is continuous with C.T. of myocardium. Conducting system of the heart is located in this layer.

	1) Continuous (somatic) capillary	2) Fenestrated (visceral) capillary	3) Sinusoidal capillary (Blood sinusoid)
Diameter	**Small & regular**	**Small & regular**	**Large & irregular**
Endothelium	**Continuous** (no pores)	Has pores covered by **diaphragms**	Has pores **without diaphragms**
Tight junctions between endothelial cells	**Present**	**Present**	Has **wide inter-cellular spaces**
Basal lamina	**Continuous**	**Continuous**	**Non- continuous**
Pericytes	**Present**	**Present**	**Absent**
Macrophages	**Absent**	**Absent**	**Present.**
Sites	**All over the body** e.g. C.T., bone, skin, exocrine glands.	- **Intestine**. - **Endocrine gland** - **Renal glomerular capillaries** (No diaphragms).	-**Liver** to carry plasma proteins **Endocrine gland** to carry hormones **Bone marrow** to carry blood cells **Spleen** to carry stored blood cells

	Blood capillaries	Lymphatic capillaries
The beginning	Begin from small arterioles.	Begin with **blind end**
Lumen & permeability	**Smaller** and **less** permeable	**larger** and **more** permeable
Endothelium	- Fenestrated or not. Usually tight junctions between the cells	- Non fenestrated - Wider gaps between the cells.
Basal lamina	Usually **continuous**	**Interrupted**
Pericytes	Usually **present**	**No pericytes** around their walls
	Exchange of materials between blood and tissues.	- Remove **lymph** from interstitial spaces and return it to blood. - Remove large molecules which cannot be carried by capillaries as fat droplets and bacteria.

Pericardium: Double-walled sac contains the heart, has serous and fibrous layers. Serous pericardium is subdivided into two layers, parietal (fused to fibrous pericardium) and visceral (epicardium). In-between parietal and visceral layers there is a pericardial cavity contains a lubricating fluid. Both serous layers lubricate the heart to prevent friction. during its activity.

ii. **Inclusions**: **a. Glycogen granules:** concentrated between myofibrils.

b. Lipofuscin granules: accumulate to be abundant in old age, forming brown atrophy of the heart.

c. Atrial granules: present in muscle cells of heart atria. They contain atrial natriuretic hormones which affect urinary excretion of sodium & water. So, muscle cells of heart's atria also have an **endocrine function**.

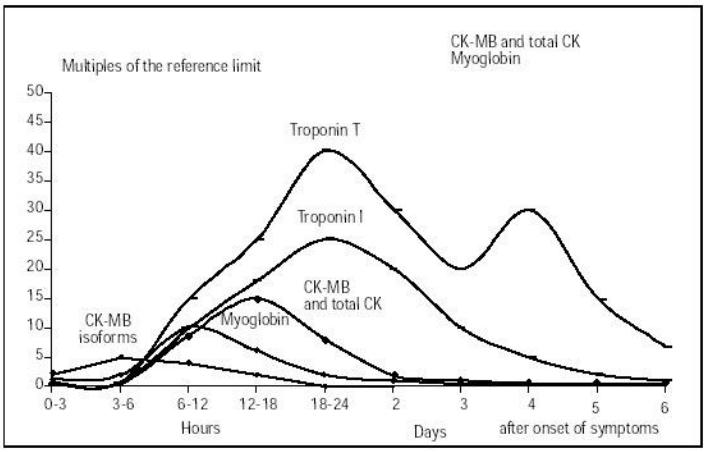
1) **Large elastic** (conducting) arteries: which carry blood from the heart.
2) **Medium-sized muscular** (distributing) arteries: deliver blood to organs.
3) **Small arteries (arterioles)**: regulate the blood flow to capillaries.

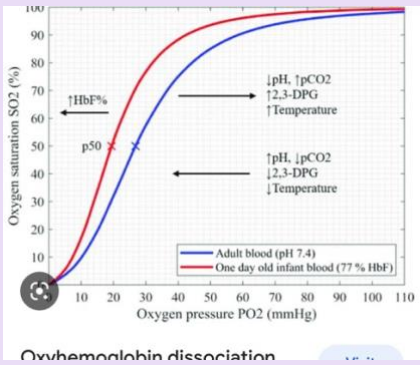
The terminal portions of arterioles that drain into the capillaries are called **metarterioles.** Thickening of their **smooth muscle** forms the **precapillary sphincter** that control blood flow to capillaries.

Increased smooth muscle tone of media in arterioles → hypertension.

Good level: below 200 mg/dl (low risk of heart disease).

Border line: 240mg/dl (if higher at high risk)





Oxyhemoglobin dissociation

Systemic Vascular Resistance (SVR)

$$SVR = \frac{(MAP - CVP)}{CO} * 80$$

- MAP: Mean Arterial Pressure mmHg
- CVP: Central Venous Pressure mmHg
- CO: Cardiac Output l/min
- 80: Conversion – Wood Units (mmHg/l/min) to Metric (dynes/sec/cm^{-5})

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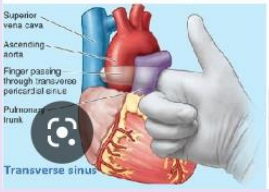
Waveform Component	Phase of Cardiac Cycle	Mechanical Event
a wave	End diastole	Atrial contraction
c wave	Early systole	Tricuspid bulging (IVC)
v wave	Late systole	Systolic filling of the atrium
x descent	Mid systole	Atrial relaxation
y descent	Early diastole	Early ventricular filling

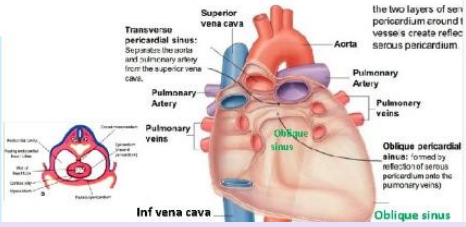
Pericardial sinuses

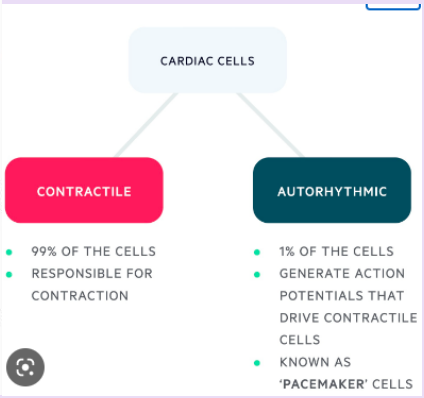
There are two pericardial sinuses: **Transverse sinus** and **oblique Sinus**

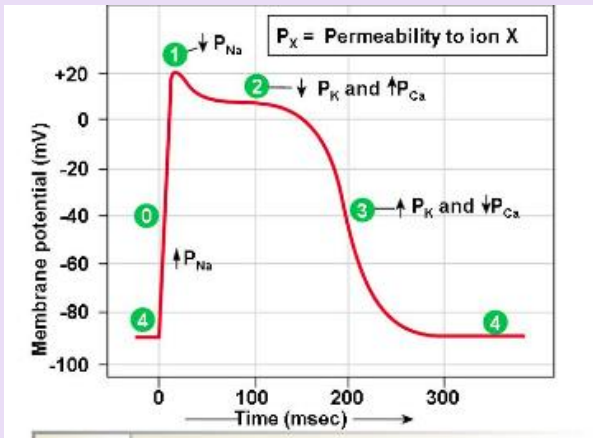
Transverse sinus: located between the **aorta and pulmonary artery anteriorly** and the **superior vena cava posteriorly**

Oblique sinus: is a cul-de-sac sinus, enclosed between the limbs of an inverted U shaped pericardial reflection; it lies posterior to the left atrium. Pericardial reflection is a double layer of visceral mesoderm supporting the heart in the pericardial cavity.

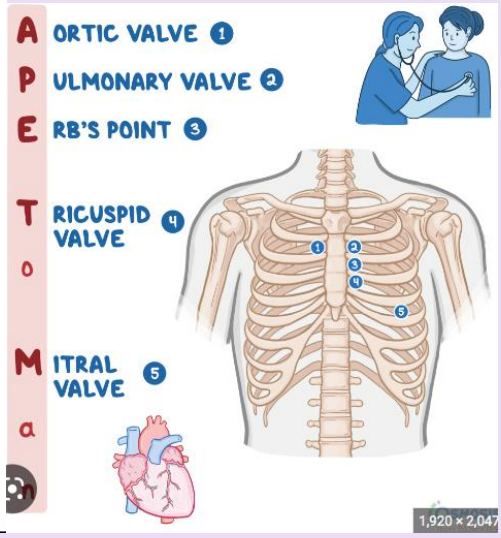








Phase	Membrane channels
0	Na^+ channels open
	Na^+ channels close
	Ca^{2+} channels open; fast K^+ channels close
3	Ca^{2+} channels close; slow K^+ channels open
4	Resting potential



Heart valve projections

Left atrioventricular valve - posteriorly to the left side of the sternum at the level of the left fourth costal cartilage

Right atrioventricular valve - posteriorly to the right side of the sternum at the level of the right fourth costal cartilage

Aortic valve - posteriorly to the left side of the sternum at the level of the third intercostal space

Pulmonary valve - at the junction of sternum and left third costal cartilage

Heart auscultation points

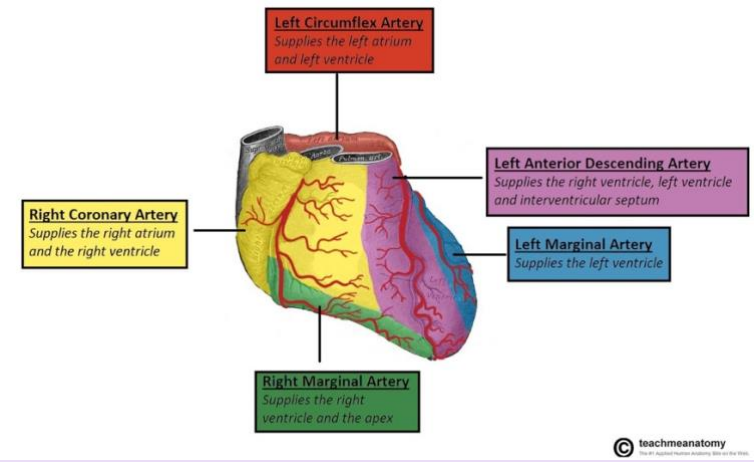
Left atrioventricular valve - the left fifth intercostal space at the midclavicular line

Right atrioventricular valve - left fifth intercostal space, parasternally

Aortic valve - right second intercostal space, parasternally

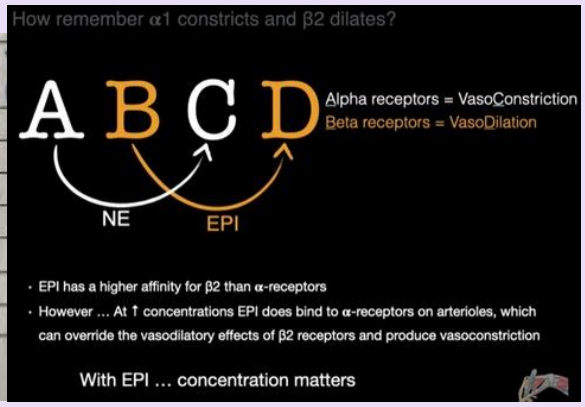
Pulmonary valve - left second intercostal space, parasternally

As chylomicron moves into circulation it takes apo cII and apo e from hdl
Apo cII can act as ligand for lipoprotein lipase found on adipose cardiac and muscle cells so it can be taken up and used or stored
Chylomicron remnant is the one left with bacha kucha thing so hdl says give back my cii and only apo e is left
Apo e is the ligand which can bind to receptor found on liver 2:25 am



Hyperkalemia = cardiac arrest
Hypokalemia = arrhythmia

ECG component	Normal Value (sec)
P wave duration	0.06-0.11
T wave duration	0.16
R-R interval	0.6-1.0
PR interval	0.12-0.20
PR segment	0.08
QRS interval	<0.12
QT interval	0.36-0.44

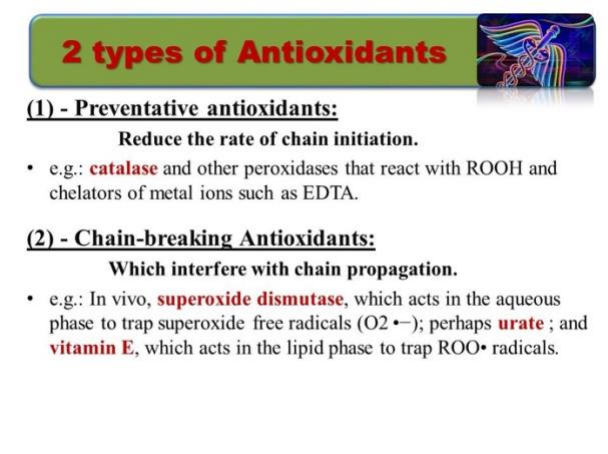


Congenital heart defects

Right to left shunt : cyanotic congenital defect

Left to right shunt: acyanotic congenital defect

Chronotropic - affects heart rate
Ionotropic affects contractility
Dromotropic affects conduction velocitt



2 types of Antioxidants

(1) - Preventative antioxidants:

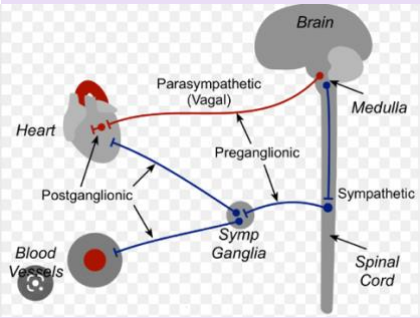
Reduce the rate of chain initiation.

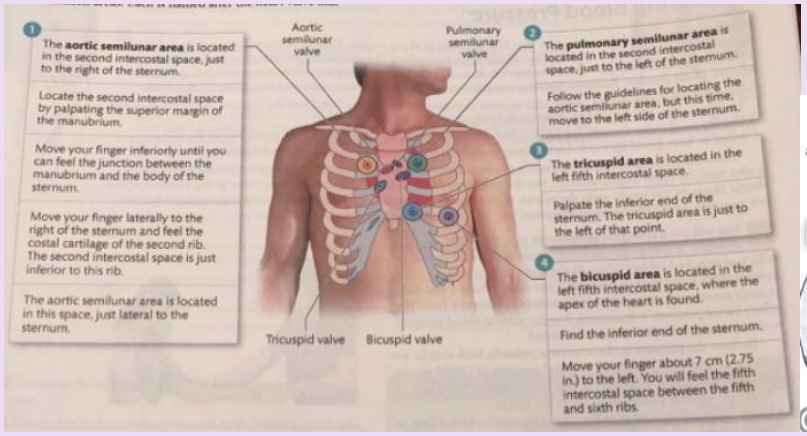
- e.g.: **catalase** and other peroxidases that react with ROOH and chelators of metal ions such as EDTA.

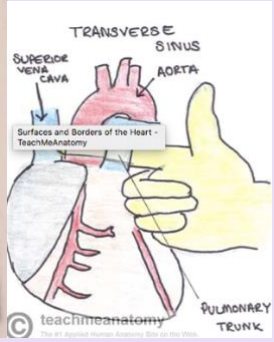
(2) - Chain-breaking Antioxidants:

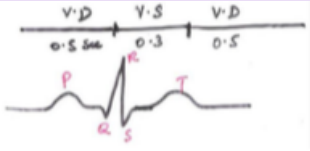
Which interfere with chain propagation.

- e.g.: In vivo, **superoxide dismutase**, which acts in the aqueous phase to trap superoxide free radicals (O2 •−); perhaps **urate** ; and **vitamin E**, which acts in the lipid phase to trap ROO• radicals.









Auditory Changes: Two heart sounds produced

S1: at the onset of ventricular systole.

S2: at the end of ventricular systole. (A_2P_2)

12. Atrioventricular Canal Defect (AVC)

In this heart defect there are three components, the first is a primum atrial septal defect (ASD) which is a hole communicating the two atrial chambers. The second component is an inlet ventricular septal defect (VSD), which is a hole communicating the two ventricular chambers. The third component involves the valves connecting the atrial chambers to the ventricular chambers. Normally there are two atrioventricular valves the mitral and tricuspid valves, in this lesion these two valves are combined together, resulting in a common atrioventricular opening (or canal).

Table 15.1: Derivatives of Aortic arches.

AORTIC ARCHES	DERIVATIVES
First pair	Maxillary arteries
Second pair	Stapedial artery & hyoid artery
Third pair	Common carotid and the proximal part of the internal carotid
Fourth pair	**Left fourth aortic arch** — arch of aorta **Right fourth aortic arch** — proximal part of right subclavian
Fifth pair	—
Sixth pair	**Right sixth aortic arch** — right pulmonary artery **Left sixth aortic arch** — left pulmonary artery & ductus arteriosus

Table 15.2: Important Veins & its Origins.

IMPORTANT VEIN	ORIGIN
Superior vena cava	Right anterior cardinal vein & right common cardinal vein
Inferior vena cava	**Hepatic segment** derived from hepatic vein & sinusoids **Prerenal segment** derived from right subcardinal vein. **Renal segment** derived from sub-supracardinal anastomosis **Post renal segment** derived from right supracardinal vein
Portal vein	Vitelline vein
Hepatic vein	Vitelline vein
Azygos & hemizygos vein	Supracardinal vein

Table 15.3: Fates of Heart Dilatation

EMBRYONIC DILATATION	ADULT STRUCTURE
Truncus arteriosus	Proximal part of aorta. Proximal part of pulmonary artery
Bulbus cordis	Smooth part of right ventricle. Smooth part of left ventricle.
Primitive ventricle	Trabeculated part of right ventricle. Trabeculated part of left ventricle
Primitive atrium	Trabeculated part of right atrium. Trabeculated part of left atrium.
Sinus venosus	Smooth part of right atrium. Coronary sinus.

LIPIDS Levels (mg/dl)

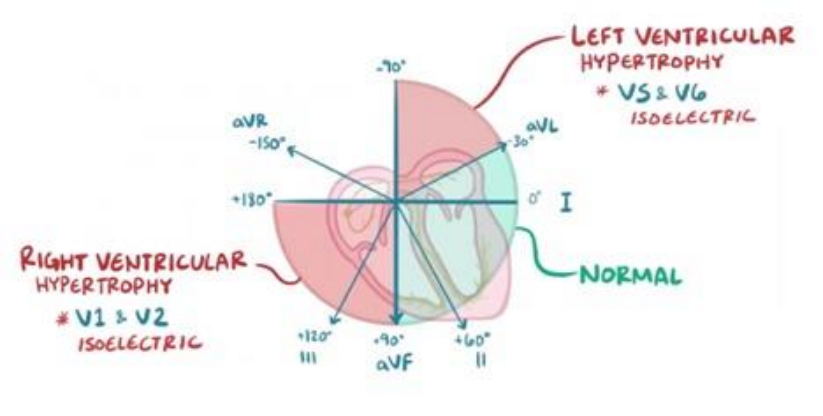
- **LDL**
  - <100 →Optimal
  - 100-129 → Near optimal
  - 130-159 → Borderline
  - 160-189→ High
  - ≥ 190 → Very High
- **Total Cholesterol**
  - < 200 → Desirable
  - 200-239 → Borderline
  - ≥240 → High
  - < 40 → Low
  - ≥ 60 → High
- **Serum Triglycerides**
  - < 150 → normal
  - 150-199 → Borderline
  - 200-499 → High
  - ≥ 500 → Very High

Table 17–1 Blood Flow to Various Organs and Tissues Under Basal Conditions

Organ	Cardiac Output (%)	Flow (ml/min)	ml/min/100 g of Tissue
Brain	14	700	50
Heart	4	200	70
Bronchi	2	100	25
Kidneys	22	1100	360
Liver	27	1350	95
Portal	(21)	(1050)	
Arterial	(6)	(300)	
Muscle (inactive state)	15	750	4
Bone	5	250	3
Skin (cool weather)	6	300	3
Thyroid gland	1	50	160
Adrenal glands	0.5	25	300
Other tissues	3.5	175	1.3
Total	100.0		

Hyperlipidemia: high lipids (all 4)

1. Prim: genetics
   1) Familial Hypercholesterolemia: mutation in LDL receptor --> high cholesterol, TG (xanthomas - stretched skin)
   2) Familial Combined Hyperlipidemia: high VLDL
   3) Type 1 **Hyperlipoproteinemia**: mutation of lipoprotein gene --> high chylomicrons
   4) Type 2a Hyperlipidemia: auto dom tendon xanthoma, xanthelasma, corneal ringing
   5) Type 2b Hyperlipidemia: mixed - high LDL, VLDL --> high cholesterol, TG
   6) Type 3 Hyperlipidemia: palmar xanthomas
   7) Type 4 Hyperlipidemia: eruptive xanthomas
   8) Type 5 Hyperlipidemia:



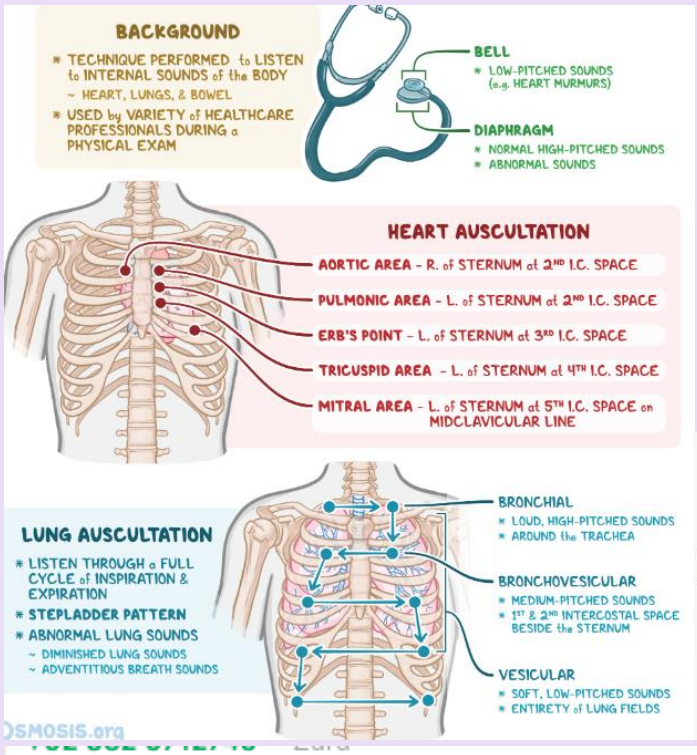
- **Stable angina.** Stable angina is the most common form of angina. It usually happens during activity (exertion) and goes away with rest or angina medication. For example, pain that comes on when you're walking uphill or in the cold weather may be angina.

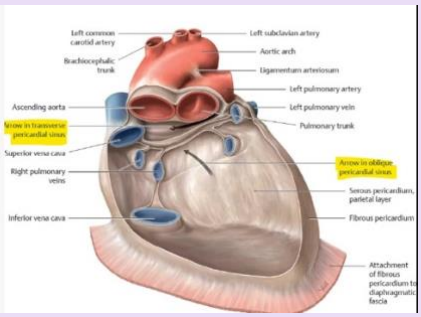
  Stable angina pain is predictable and usually similar to previous episodes of chest pain. The chest pain typically lasts a short time, perhaps five minutes or less.

- **Unstable angina (a medical emergency).** Unstable angina is unpredictable and occurs at rest. Or the angina pain is worsening and occurs with less physical effort. It's typically severe and lasts longer than stable angina, maybe 20 minutes or longer. The pain doesn't go away with rest or the usual angina medications. If the blood flow doesn't improve, the heart is starved of oxygen and a heart attack occurs. Unstable angina is dangerous and requires emergency treatment.

- **Variant angina (Prinzmetal angina).** Variant angina, also called Prinzmetal angina, isn't due to coronary artery disease. It's caused by a spasm in the heart's arteries that temporarily reduces blood flow. Severe chest pain is the main symptom of variant angina. It most often occurs in cycles, typically at rest and overnight. The pain may be relieved by angina medication.

- **Refractory angina.** Angina episodes are frequent despite a combination of medications and lifestyle changes.

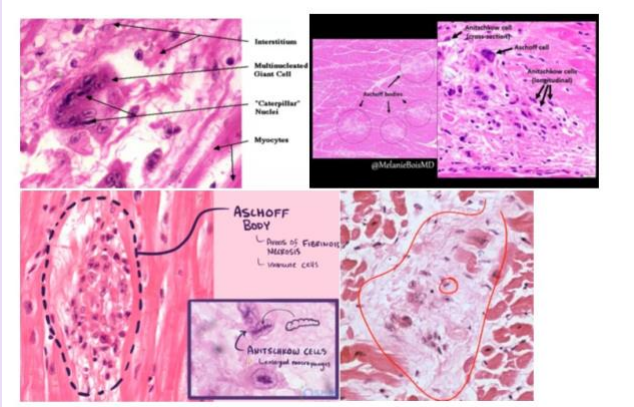




Total peripheral resistance x Cardiac output = Mean arterial pressure aik ye formula he

3:08 pı

Infundinulum is out flow part of right ventricle so should be pulmonary



Can PVCs cause tachycardia?

Abstract. Premature ventricular contraction (PVC), a common arrhythmia affecting 1–2% of the general population, has been considered to have a benign clinical course. However, **people with PVC often develop heart failure and ventricular arrhythmias such as ventricular tachycardia**. 16-Jun-2021

Major apolipoproteins

APOLIPOPROTEIN	FUNCTION	CHYLOMICRON	CHYLOMICRON REMNANT	VLDL	IDL	LDL	HDL
E	Mediates remnant uptake (everything except LDL)	✓	✓	✓	✓		✓
AI	Found only on alpha-lipoproteins (HDL), activates LCAT						✓
CII	Lipoprotein lipase cofactor that catalyzes cleavage	✓		✓	✓		✓
B_{48}	Mediates chylomicron secretion into lymphatics Only on particles originating from the intestines	✓	✓				
B_{100}	Binds LDL receptor Only on particles originating from the liver (I hope I live to Be 100)			✓	✓	✓	

Closure of Foramen Ovale:

(i) Functional- Immediately after birth
(ii) Anatomical- 1 year after birth

4:26 AM

This document contains ink, shapes an...

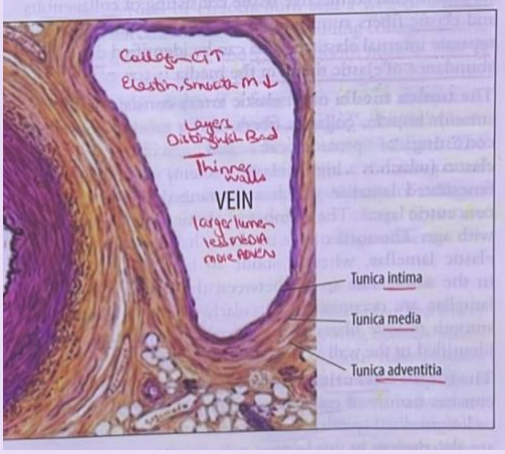
	Voltage gated sodium channels along with some calcium channels that allow slow influx of Calcium
	Phase 1 (+10 to 0): Sodium channels inactivate and potassium channels open up promoting potassium efflux
	L-type calcium channels become a little more active promoting calcium influx
250ms	**Phase 2 (Plateau):** Due to the efflux of K ions and influx of Ca channels net result in change of membrane potential is zero. Ca ions bind to troponin and cause muscle contraction
	Phase 3: L-type Ca channels close, Ca is removed from cells by methods mentioned below. K channels bcm more active pump more K from cell into the EC environment
	Phase 4: Plateau at -90mV until cations flow from nodal cells again to raise potential to -70mV again to repeat cycle again.

Screenshot has been saved to Pictures/Screenshot

cles

MEDIUM-SIZED ARTERIES

These arteries are also known as *muscular arteries* because their thick walls contain a large amount of smooth muscle in the tunica media (Fig. 13.3). The medium-sized arteries perform the function of distribution of blood to various regions and organs of the body by contracting or relaxing the smooth muscle of their wall according to the functional demands of the region concerned. Therefore, these arteries are also referred to as *distributing arteries*. Most of the named arteries of the body belong to this group, e.g., the axillary, brachial, femoral, and popliteal arteries.

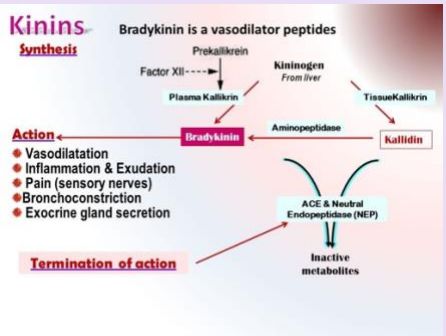


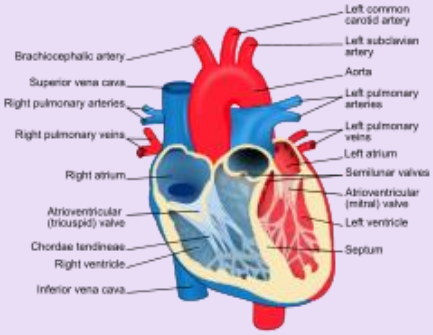
ACAT converts cholesterol to cholesterol ester
It helps in storage of cholesterol when it is excess
LCAT(activated by APO AI) helps transport of Cholesterol from peripheral tissues to hdl

2:09 r

Lipoprotein functions	Lipoproteins are composed of varying proportions of proteins, cholesterol, TGs, and phospholipids. LDL and HDL carry the most cholesterol. Cholesterol is needed to maintain cell membrane integrity and synthesize bile acids, steroids, and vitamin D.
Chylomicron	Delivers dietary TGs to peripheral tissues. Delivers cholesterol to liver in the form of chylomicron remnants, which are mostly depleted of their TGs. Secreted by intestinal epithelial cells.
VLDL	Delivers hepatic TGs to peripheral tissue. Secreted by liver.
IDL	Delivers TGs and cholesterol to liver. Formed from degradation of VLDL.
LDL	Delivers hepatic cholesterol to peripheral tissues. Formed by hepatic lipase modification of IDL in the liver and peripheral tissue. Taken up by target cells via receptor-mediated endocytosis. LDL is Lethal.
HDL	Mediates reverse cholesterol transport from peripheral tissues to liver. Acts as a repository for apoC and apoE (which are needed for chylomicron and VLDL metabolism). Secreted from both liver and intestine. Alcohol ↑ synthesis. HDL is Healthy.

saturated are badd 9:40 am
trans and saturated are bad 9:40 am
poly and mono are good 9:40 am
and one qs said poly is better than mono cuz u get essential fatty acids from it 9:40 am

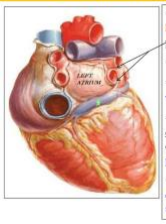




	DESIRABLE
Cholesterol	<200 mg/dl
Triglycerides	<150 mg/dl
HDL cholesterol	60 mg/dl
LDL cholesterol	60-130 mg/dl
Cholesterol/ HDL ratio	4.0

Valve	Number of flaps/cusps	location	prevent backflow of blood
Atrioventricular valves	3 (right), 2 (left)		From the ventricles into the atria
Tricuspid valve	3	between the right atrium and right ventricle.	
Bicuspid or mitral valve	2	between the left atrium and left ventricle.	
Semilunar valves	3 (half-moon shaped) flaps		into the ventricle
Pulmonary semilunar valve	3 (half-moon shaped) flaps	at the opening between the right ventricle and the pulmonary trunk	
Aortic semilunar valve	3 (half-moon shaped) flaps	at the opening between the left ventricle and the aorta	

Base of the Heart (posterior surface)



- It is formed by the 2 atria, mainly left atrium, into which open the 4 pulmonary veins.
- It is directed backwards.
- Lies opposite middle thoracic vertebrae(T5-7)
- Is separated from the vertebral column by descending aorta, esophagus and oblique sinus of pericardium.
- Bounded inferiorly by post part of coronary sulcus , which lodges the coronary sinus

- Rheumatic Endocarditis (Rheumatic **Valvulitis**): (usually mitral → aorta)
  - During fever: tachycardia (due to fever) + inflamed valves → endothelial erosion at line of closure of leaflets → **Rheumatic Vegetations** (fibrin & platelets) – sterile, no embolization like in infective endocarditis (held tightly)
  - After fever: vegetations heal → platelets release PDGF → fibroblasts activated → lay down collagen → vegetations stick so leaflets fuse (**Commissural Fusion/Fish Mouth Appearance**) → adhesions, fibrosis → valve fibrosis & stenosis (LONG TERM COMPLICATION)
  - Diff from Aortic Stenosis: valves have stenosis (not just fibrosis) & commissural fusion
  - Reinfection of Acute RHD → Cumulative damage → Chronic RHD → Valvular Heart Diseases
  - Acute: Regurgitation; Chronic: Stenosis

→ Pancarditis: all 3 layers

LIPIDS Levels (mg/dl)

- **LDL**
  - <100 → Optimal
  - 100-129 → Near optimal
  - 130-159 → Borderline
  - 160-189 → High
  - ≥ 190 → Very High
- **Total Cholesterol**
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  - < 40 → Low
  - ≥ 60 → High
- **Serum Triglycerides**
  - < 150 → normal
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  - 200-499 → High
  - ≥ 500 → Very High

TYPES OF EXERCISE

Depending upon the type of muscular contraction:

1. Dynamic exercise :

Primarily involves the isotonic muscular contraction. It keeps the joints and muscles moving. Examples are swimming, bicycling, walking. Diastolic BP unaltered or decrease

2. Static exercise.

Involves isometric muscular contraction without movement of joints. Example is pushing heavy object. Diastolic BP unaltered or decrease

heart. A negative injury potential found on one of the precordial leads indicates that this electrode is in an area of strong negative potential and that the current of injury originates in the anterior wall of the ventricles. In contrast, a positive T-P segment with respect to the J point indicates the existence of a posterior ventricular wall infarction.

1. Total Cholesterol: <200 mg/dL (240 borderline = **hypercholesterolemia**)
2. LDL: <100-200 mg/dL
3. HDL: >60 mg/dL = heart disease (60-130) (3:1 HDL:LDL)
4. TG: <150 mg/dL (high = fatty liver)
5. BP: 120/80
6. FBS/Glucose: 80-100 (100-120 = prediabetic; >120 mg/dL = diabetic)

Velocity = flow upon area and Area is pir2 so increasing diameter velocity decreases and reynolds number formula is pdv / n it may seem that increasing diamter should increase reynolds number but velocity has greater dominance over diameter and also it isnt "direct " .

11:16 am

by about 0.02 to 0.05 second. A prolonged QRS wave caused by left ventricular hypertrophy is shown in **Figure 12–1**.

- *Blockade of the impulses in the Purkinje system* prolongs the QRS complex because the duration of ventricular depolarization increases in one or both ventricles.

CURRENT OF INJURY (p. 148)

Several abnormalities cause a portion of the heart to remain *depolarized all of the time*, and the current that flows from the depolarized area to the polarized areas of the heart is called the *current of injury*. Some of the abnormalities that can cause a current of injury are as follows:

- Mechanical trauma
- Infectious processes that damage the cardiac muscle membrane
- Coronary ischemia

The Axis of the Current of Injury Can Be Determined With the Electrocardiogram. When a portion of the heart is injured and emits a current of injury, the only time the heart returns to zero potential is at the end of the QRS wave because all of the heart is depolarized at this time (see **Figure 9–1**). The axis of the current of injury is determined in the following way:

1. First, *determine the J point*, which is the point of zero potential at the end of QRS wave.
2. *Determine the level of the T-P segment* with respect to the J point on the three standard leads.
3. *Plot the voltages on the coordinates of the three leads* to determine the axis of the current of injury, and note that the negative end of the vector originates in the *injured* area of the ventricles.

Acute Anterior and Posterior Wall Infarctions Can Be Diagnosed With the Electrocardiogram. The current of injury is also useful for determining whether an infarction is in the anterior or posterior portion of the

Blocks
1) non-progressive
2) progressive
3) irreversible.

Ca^{+} = 1000 - 2000 mg	Cu = 900 µg
Cr = 1800 - 2200 mg	Fe = 8-18 mg
K = 4700 mg	Zn = 8-11 mg
Na = 1500 mg	Se = 55 µg
P = 700 mg	

Vit K = 120 µg — 90 µg
Vit E = 15 mg
Vit D = 15 µg — 20 µg Vit D = 1 µg = 40 IU
Vit A = 900 (RAE) — 700 (RAE) ≈ 900 µg — 700 µg
↳ 1 RAE = 1 µg of retinol
12 µg of β-carotene

Vit B5 = no RDA B3 = Niacin (1.5 g/day) can inhibit lipolysis. ↓ TAG synth
Vit B7 = — TAG (VLDL) cholesterol (LDL) ↓
Vit B2 = — B12 = 2-5 mg stored
Vit B1 = — Pregnant = 0.4 mg/day
Vit B6 = 1.25 mg/day B9 = 0.1 mg/day

Cardiac chamber	Radiological sign of enlargement
Right atrium	Bulging right heart border
Left atrium	•Prominent left atrial appendage on the left heart border
	•Double outline of the right heart border
	•Splayed carina with elevation of the left main bronchus
	•Bulge of the upper posterior border on the lateral view
Right ventricle	•Elevated cardiac apex
	•Bulging of anterior upper part of the heart border on the lateral view
Left ventricle	Bulging lower left cardiac border with depressed cardiac apex

MNEMONIC: PROVe

Components of tetralogy of Fallot

Pulmonary infundibular stenosis
Right ventricle hypertrophy
Overriding aorta
Ventricular septal defect

Layers	Elastic Artery	Muscular Artery	Arteriole
Intima	Endothelium + subendothelial connective tissue (more elastic fibers) + Internal elastic lamina (less prominent)	Well developed Intima with very prominent Internal Elastic Lamina	Subendothelial layer &Internal elastic lamia poorly developed
Media	Mainly Elastic tissue	Mainly Smooth Muscle	1-3 layers of Smooth Muscle cells
Adventitia	Thin with more of elastic fibers Vasa vasorum, Lymphatics + Nerves	Thin layer of fibroelastic tissue, nerves, lymphatics Vasa vasorum not prominent	Loose Connective tissue & few nerve fibers

Familial hyperlipidemia : Summary

Familial Dyslipidemia	Lipoproteins Elevated	Lipids Elevated	Pathophysiology	Main Complication
Type I: Hyperchylomicronemia	Chylomicrons	Cholesterol: + TG: +++	ApoC-2 or LPL deficiency	Pancreatitis, no atherosclerosis
Type IIa: Hypercholesterolemia	LDL	Cholesterol: +++ TG: no change	LDL receptor deficiency	Atherosclerosis
Type IIb: Combined hyperlipidemia	VLDL, IDL	Cholesterol: +++ TG: +++	VLDL overproduction	Atherosclerosis
Type III: Dysbetalipoproteinemia	IDL, chylomicron remnants	Cholesterol: ++ TG: ++	ApoE deficiency	Atherosclerosis
Type IV: Hypertriglyceridemia	VLDL	Cholesterol: + TG: +++	VLDL overproduction	Atherosclerosis
Type V: Mixed hypertriglyceridemia	VLDL, chylomicrons	Cholesterol: + TG: +++	VLDL overproduction	Pancreatitis, no atherosclerosis

The hemodynamic effects of respiration are as follows:
INSPIRATION:
*JVP falls (due to increased venous return to the right heart as a result of fall in the intrathoracic pressure)
*Blood pressure falls (upto 10 mmHg)
*Heart rate increases
*Second heart sound splits (due to the delay in rght ventricular emptying that occurs as a result of increased capacity of the pulmonary vasculature)
EXPIRATION:
*JVP rises
*Blood pressure rises
*Heart rate slows down
*Second heart sound fuses

Explanation generated for Binte Islam at 2022-11-26 14:53:32

The six grades of murmur are as follows:
Grade I: the murmur is only audible on listening carefully for some time.
Grade II: the murmur is faint but immediately audible on placing the stethoscope on the chest.
Grade III: a loud murmur readily audible but with no thrill.
Grade IV: a loud murmur with a thrill.
Grade V: a loud murmur with a thrill. The murmur is so loud that it is audible with only the rim of the stethoscope touching the chest.
Grade VI: a loud murmur with a thrill. The murmur is audible with the stethoscope not touching the chest but lifted just off it.

Note that thrill refers to a palpable murmur, a vibratory sensation that can be felt on touching the precordium.

Case

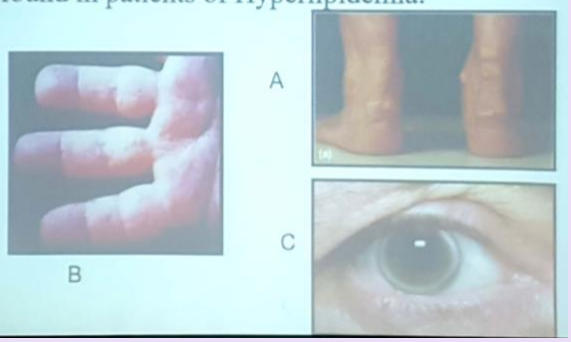
- 40 year old male with hypertension who smokes cigarettes. He has not previously been diagnosed with CHD. His father developed CHD at age 53.
- Fasting labs:
  - glucose 98 mg/dl
  - cholesterol 210 mg/dl
  - triglyceride 150 mg/dl
  - HDL 45 mg/dl

- **Familial Hypercholesterolemia**
  - Codominant genetic disorder, occurs in heterozygous form
  - 1 in 500 individuals
  - Mutation in LDL receptor, resulting in elevated levels of LDL at birth and throughout life
  - High risk for atherosclerosis, tendon xanthomas (75% of patients), tuberous xanthomas and xanthelasmas of eyes.
- **Familial Combined Hyperlipidemia**
  - Autosomal dominant
  - Increased secretions of VLDLs

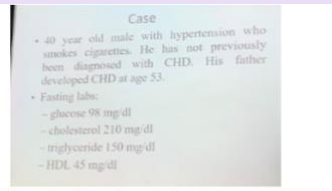
Case

- 49 year old man with no personal or family history of CHD, does not smoke cigarettes and is normotensive. Dietary history revealed high carbohydrate consumption.
- Fasting labs:
  - glucose 135 mg/dl
  - cholesterol 260 mg/dl
  - triglyceride 505 mg/dl
  - HDL 25 mg/dl

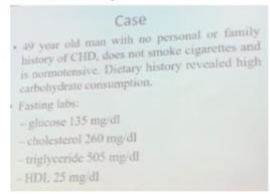
Q1. Identify the signs marked as A,B and C, found in patients of Hyperlipidemia.



- ANSWERS

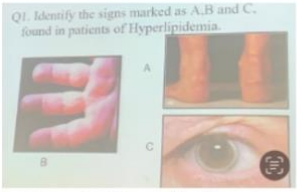


Borderline/at risk



Diabetic

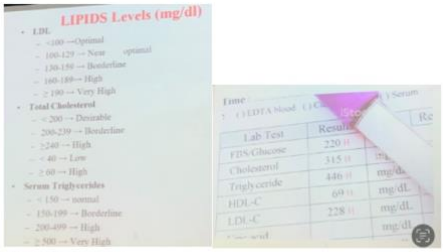
Diabetic



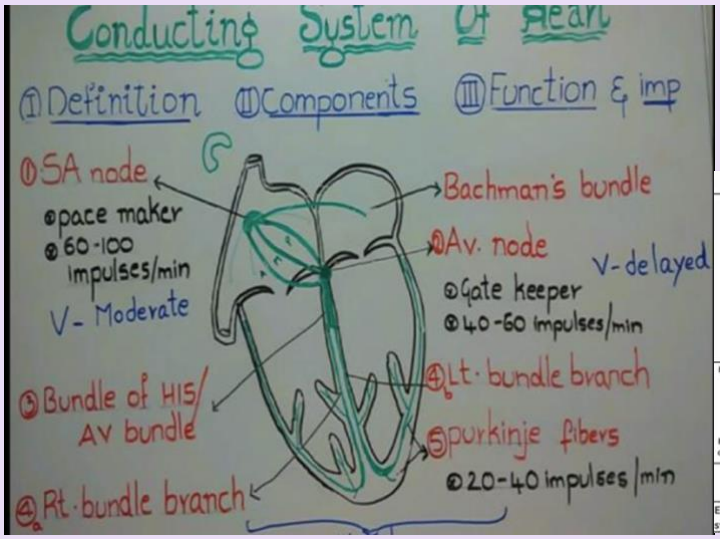
A: Type 2a Hyperlipidemia

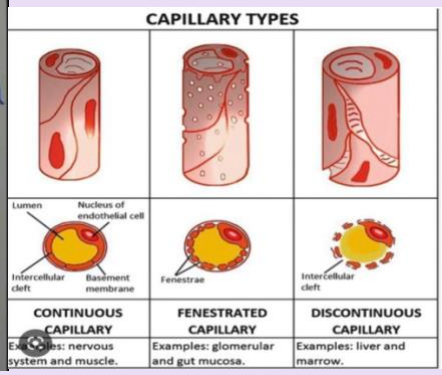
B: Type 3 Hyperlipidemia

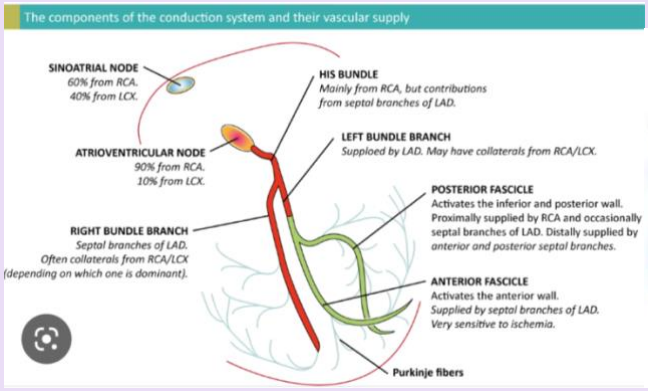
C: Type 2a Hyperlipidemia

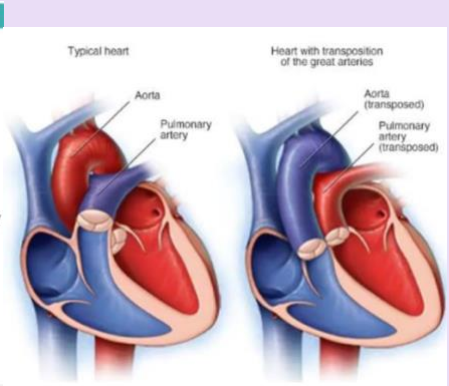


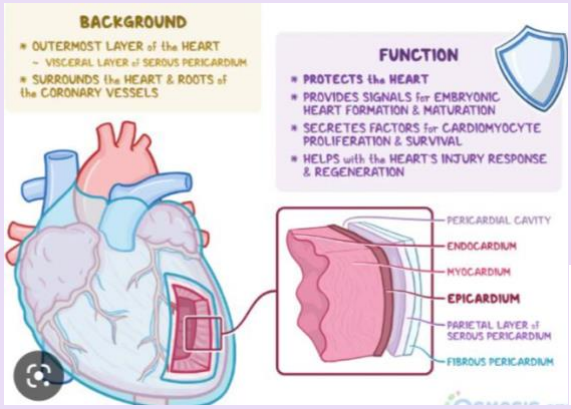
Hyperlipidemia











Congenital heart defects

Right to left shunt : cyanotic congenital defect

Left to right shunt: acyanotic congenital defect

Depolarization of nodal cells and action potential generation:

Membrane potential RMP: -60mV	Membrane channel
-60 to -55 mV	Funny sodium channels (very leaky channels allow sodium to leak continuously into the cell very slowly)
-55mV	T- type Calcium channels(calcium influx)
-40mV (threshold potential)	L-type Calcium channels(Ca influx)
+40mV	L-type Ca channels shut off, K channels open promoting K efflux
+40 to -60mV	Funny sodium channels open up cycle repeats

Pacemaker rates: SA > AV > bundle of His/ Purkinje/ventricles.
Speed of conduction: His-Purkinje > Atria > Ventricles > AV node. He Parks At Ventura AVenue.

Depolarization of contractile cells:

Membrane potential RMP: -90mV	Membrane channels
-90 to -70mv (threshold potential)	By cations flowing in from nodal cells through gap junctions
-70mV	**Phase 0 (-70 to +10) : Voltage gated sodium channels** along with some calcium channels that allow slow influx of Calcium
+10mV	**Phase 1 (+10 to 0):** Sodium channels inactivate and potassium channels open up promoting potassium efflux
0mV	L-type calcium channels become a little more active promoting calcium influx
Plateau at 0mV for 250ms	**Phase 2 (Plateau):** Due to the efflux of K ions and influx of Ca channels net result in change of membrane potential is zero. Ca ions bind to troponin and cause muscle contraction
0mV to -90mV	**Phase 3:** L-type Ca channels close, Ca is removed from cells by methods mentioned below. K channels bcm more active pump more K from cell into the EC environment
-90mV	**Phase 4:** Plateau at -90mV until cations flow from nodal cells again to raise potential to -70mV again to repeat cycle again.

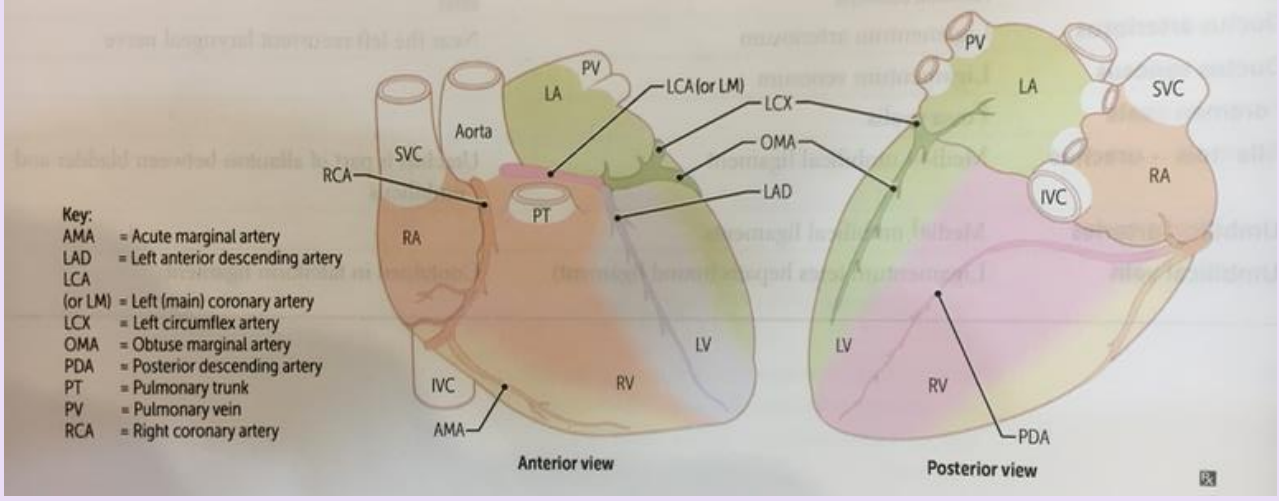
Jugular venous pulse (JVP):
a wave—atrial contraction. Absent in atrial fibrillation.
c wave—RV contraction (closed tricuspid valve bulging into atrium).
x descent—atrial relaxation and downward displacement of closed tricuspid valve during rapid ventricular ejection phase. Reduced or absent in tricuspid regurgitation and right HF because pressure gradients are reduced.
v wave—↑ RA pressure due to ↑ volume against closed tricuspid valve.
y descent—RA emptying into RV. Prominent in constrictive pericarditis, absent in cardiac tamponade.

MNEMONIC: PROVe

Components of tetralogy of Fallot

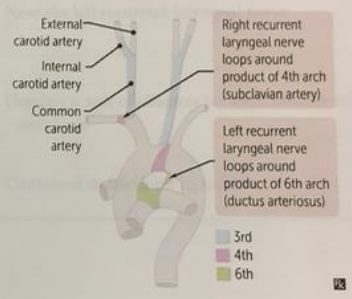
Pulmonary infundibular stenosis
Right ventricle hypertrophy
Overriding aorta
Ventricular septal defect

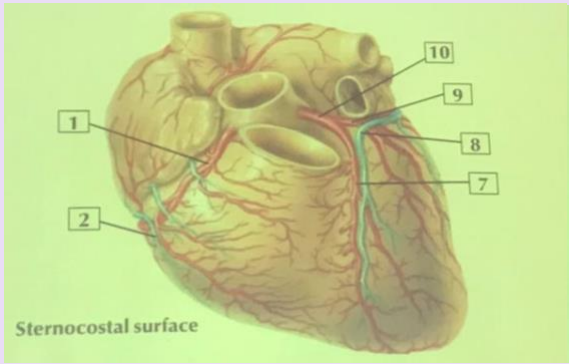
supplies RV. drains into the RA.

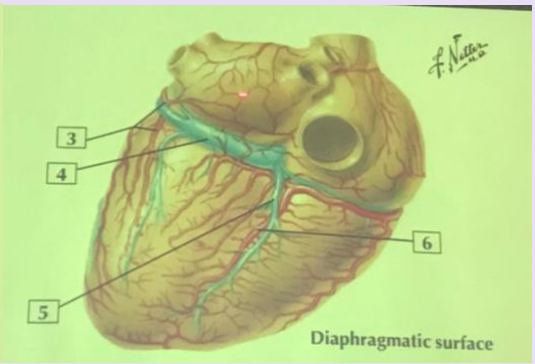


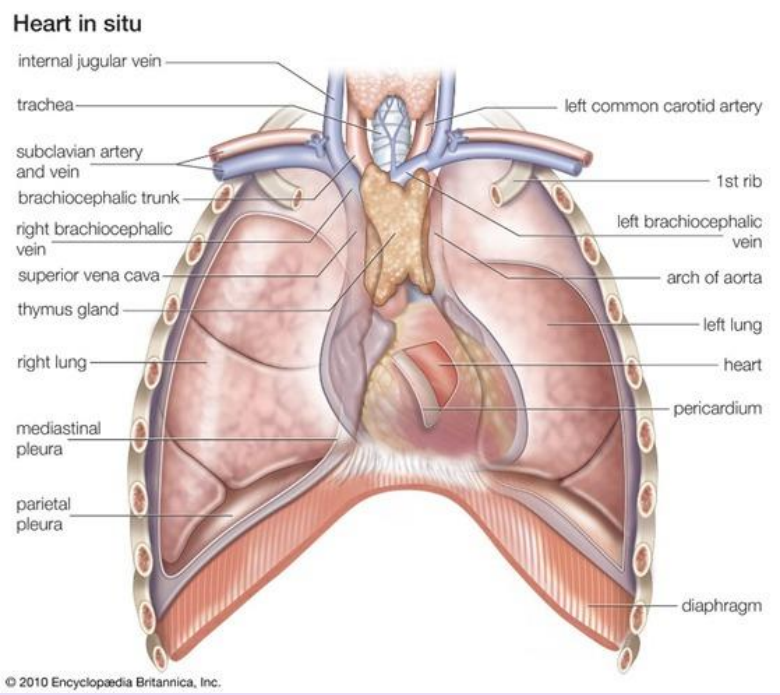
EMBRYONIC STRUCTURE	GIVES RISE TO
Truncus arteriosus	Ascending aorta and pulmonary trunk
Bulbus cordis	Smooth parts (outflow tract) of left and right ventricles
Primitive ventricle	Trabeculated part of left and right ventricles
Primitive atrium	Trabeculated part of left and right atria
Left horn of sinus venosus	Coronary sinus
Right horn of sinus venosus	Smooth part of right atrium (sinus venarum)
Endocardial cushion	Atrial septum, membranous interventricular septum; AV and semilunar valves
Right common cardinal vein and right anterior cardinal vein	Superior vena cava (SVC)
Posterior cardinal, subcardinal, and supracardinal veins	Inferior vena cava (IVC)
Primitive pulmonary vein	Smooth part of left atrium

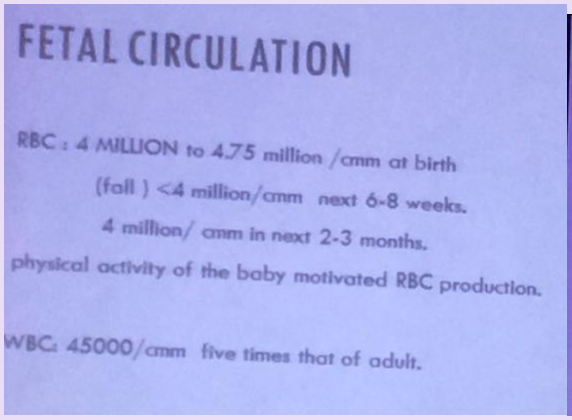
Aortic arch derivatives	Develop into arterial system.
1st	Part of maxillary artery (branch of external carotid). 1st arch is maximal.
2nd	Stapedial artery and hyoid artery. Second = stapedial.
3rd	Common carotid artery and proximal part of internal carotid artery. C is 3rd letter of alphabet.
4th	On left, aortic arch; on right, proximal part of right subclavian artery. 4th arch (4 limbs) = systemic.
6th	Proximal part of pulmonary arteries and (on left only) ductus arteriosus. 6th arch = pulmonary and the pulmonary-to-systemic shunt (ductus arteriosus).

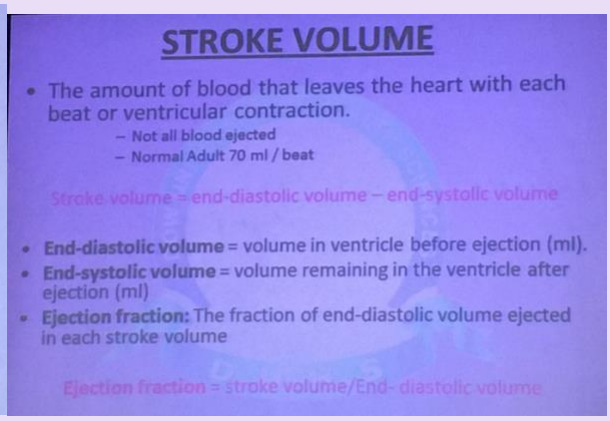


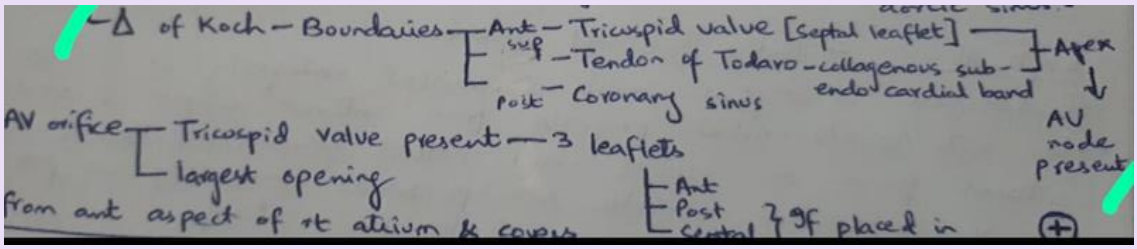


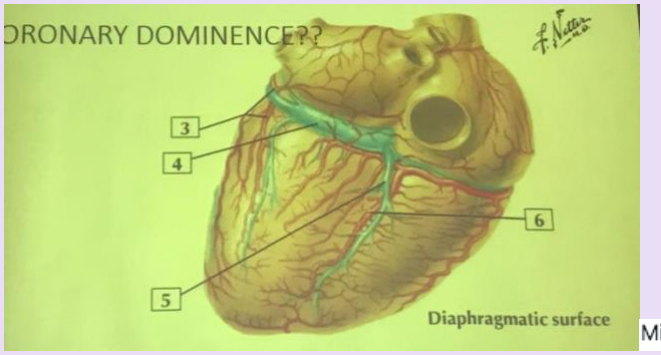












Mitral also known as aped beat

2nd intercostal spaces right = aortic, left = pulmonary
Lower lefy sternal border (4th/5th space) = tricuspid
Midclavicular line 5th space = mitral
6:02 pm

HEART SOUNDS

- The closure of the heart valves is responsible for the characteristic sound produced by the heart, usually referred to as a 'lub-dup' sound.
- The lower-pitched 'lub' sound occurs during the early phase of ventricular contraction. This is produced by closing of the atrioventricular (mitral and tricuspid) valves.
- These valves prevent blood from flowing back into the atria. When the ventricles relax, the blood pressure drops below that in the artery, and the semilunar valves (aortic and pulmonary) close, producing the higher-pitched 'dup' sound.
- Malfunctions of these valves often produce an audible murmur, which can be detected with a stethoscope.

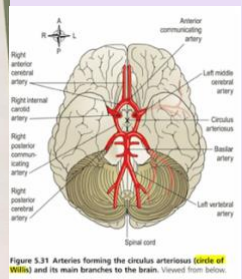


Figure 5.31 Arteries forming the circulus arteriosus (circle of Willis) and its main branches to the brain. Viewed from below.

Circle of wilis is the arterial supply present in the inferior part of brain (as u can see this circle)

Manganese, copper and zinc are required for the activity of superoxide dismutase.

Selenium is required for the activity of glutathione peroxidase.

- Coronary sinus is the **dilatation of great cardiac vein** located in the posterior part of AV groove.
- **Opens** into right atrium b/w inferior vena cava and tricuspid opening guarded by incomplete semicircular **Thebasian valve.**

Tributaries: all have valve **except** oblique vein of left atrium.

FETAL CIRCULATION

Fetal blood volume:

Is 300ml additional 75ml is added if the child is left attached to the umbilical cord post birth.

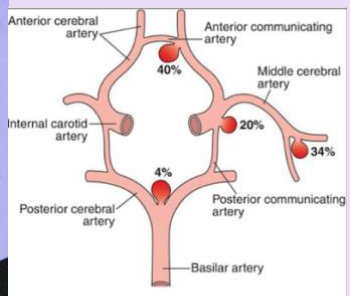
Then plasma moves into tissue spaces and the blood volume comes back to 300ml.

Hematocrit increases.

Cardiac output is about 500ml /min

Blood Pressure is first few days 70-50 mmHg which increase to 90-60 mmHg for months

115-70 mmHg at adolescence.

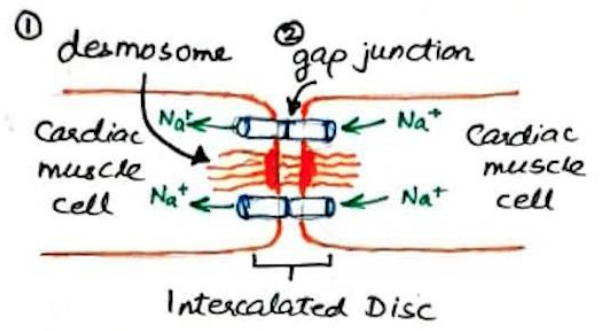


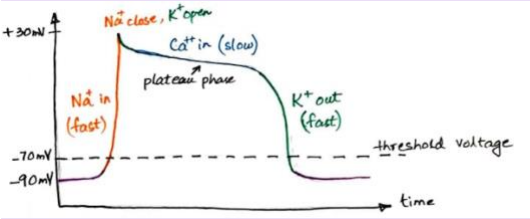
Berry aneurysms (the red berry-like structures)

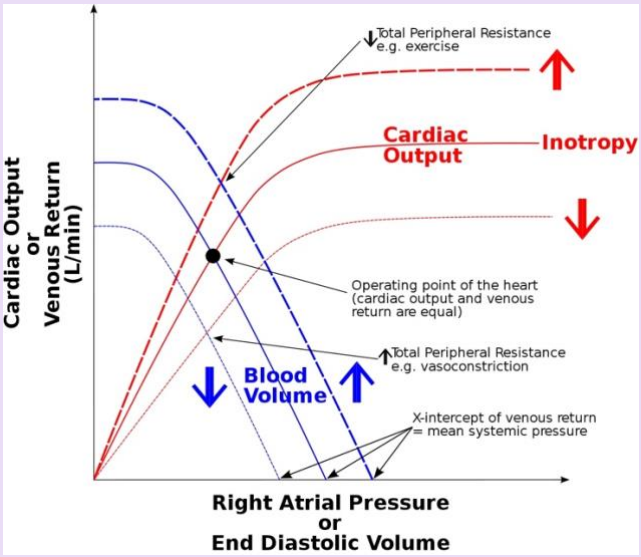
The most important vasodilator substance is probably adenosine but the effects wane off with time

Other vasodilator factors

- (1) potassium ions
- (2) adenosine triphosphate (ATP)
- (3) lactic acid
- (4) carbon dioxide.







FETUS	ADULT
DUCTUS VENOSUS	LIGAMENTUM VENOSUM
FORAMEN OVALE	FOSSA OVALIS
DUCTUS ARTERIOSUS	LIGAMENTUM ARTERIOSUM
UMBILICAL VEIN	LIGAMENTUM TERES
UMBILICAL ARTERY	MEDIAL UMBILICAL LIGAMENT + SUPERIOR VESICAL ARTERY
ALLANTOIS	MEDIAN UMBILICAL LIGAMENT

edv is the volume of blood in isovolumetric contraction that it will eject once the left ventricle's pressure will exceed the aortic pressure

after edv is ejected , the ventricles go into the isovol relaxation phase

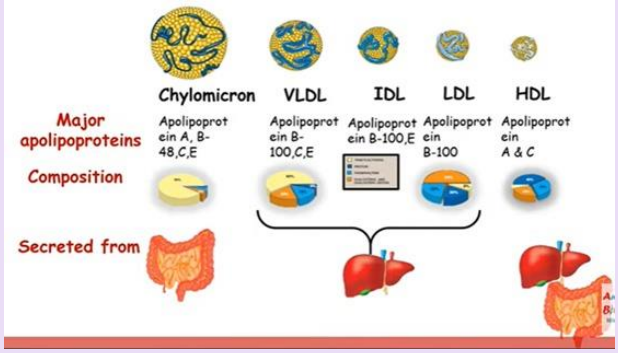
esv is the blood volume (50 ml) that will enter ventricle once mitral valve reopens

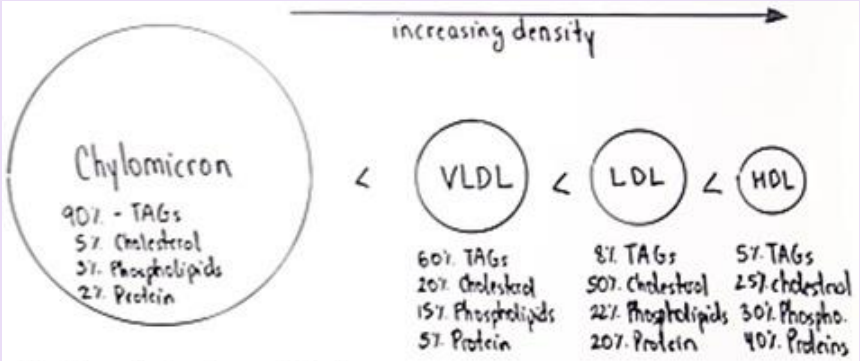
Formulas:

- $BP = CO \times TPR$, $MAP = CO \times TPR$
- $CO = HR \times SV$
- $Flow = CO$
- $Velocity\ of\ Blood\ Flow = Flow/Cross\ sectional\ area$
- $\Delta p = MAP - CVP$, $\Delta p \cong MAP$
- $\Delta p = Flow \times R\ (resistance)$
- $R = 8\eta l/\pi r^4$
- $MAP = \frac{1}{3}SBP + \frac{2}{3}DBP$, $MAP = DBP + \frac{1}{3}PP$
- $PP = SBP - DBP$
- $Re = {}^{dv\rho}/_{\eta}$

Remember:

- CO=HR X SV
- HR is directly proportional to CO
- SV= EDV-ESV
- SV is dependent on Preload, Contractility and Afterload
- SV is directly proportional to CO

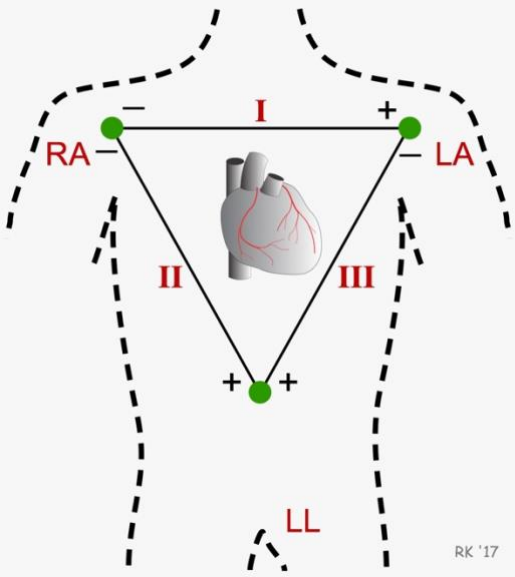




Apolipoproteins (apoproteins)

- Apolipoprotein are proteins that are associated with lipoproteins. They have four major roles:
  (1) guide the formation
  (2) provide structural support
  (3) act as a ligand for receptors
  (4) act as activators or inhibitors of certain enzymes
- In general, apolipoproteins are divided into five major classes (A-E) based on structure and function:

Apo A-I => provides structural support for HDL and activates the LCAT enzyme
Apo A-II => provides support for HDL and activates hepatic lipase
Apo B-48 => provides structural support for chylomicrons
Apo B-100 => provides support for VLDL, LDL, IDL and ligand for LDL receptors
Apo C-II => found in chylomicrons, LDL and HDL and activates LPL
Apo E => found in chylomicron remnants, IDL and HDL; acts as ligand for LDL receptor



12:29 AM 54%

Apex 5th ic space

Mitral valve. Same as above

Pulm valve. Behind 3rd costal cartilage, most audible at 2nd ic space

Bicuspid. Behind 4th costal cartilage audible left 5th ic space MCL

Aortic valve. Oppo to 3rd ic space. Most audible 2nd ic it's closure dub

Tricuspid. Oppo to 4th ic space

Nipple . 4th ic space

Breast 2 to 6th rib

SUpand inf vena cava 3rd costal cartilage and 5th costal cartilage respectively

Sup VC in fetal life upper border of first rib

PATHOLOGY

Holocytolic/ harsh systolic murmur in **VSD**

fixed wide S2 in **ASD** paradoxical emboli

PDA

Associated with congenital rubella

Machine like murmur

TOF

r8 to left shunt, early cyanosis

Boot shaped heart

Transposition Of GV

maternal diabetes

Aortic stenosis

Crescendo decrescendo murmur

Fusion of aortic valve commissures

Consequence of chronic Rheumatoid heart disease

Due to which co exist with mitral stenosis

MHA having schistiocytes

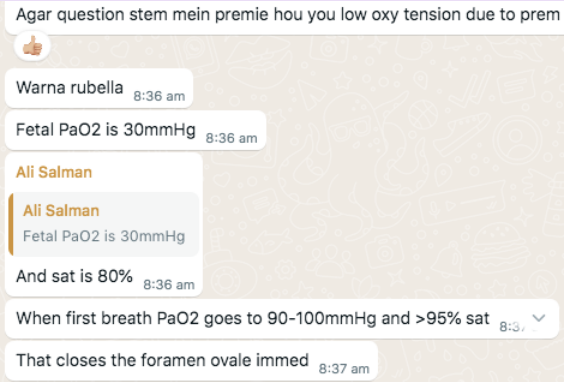
AORTIC REGURGITATION

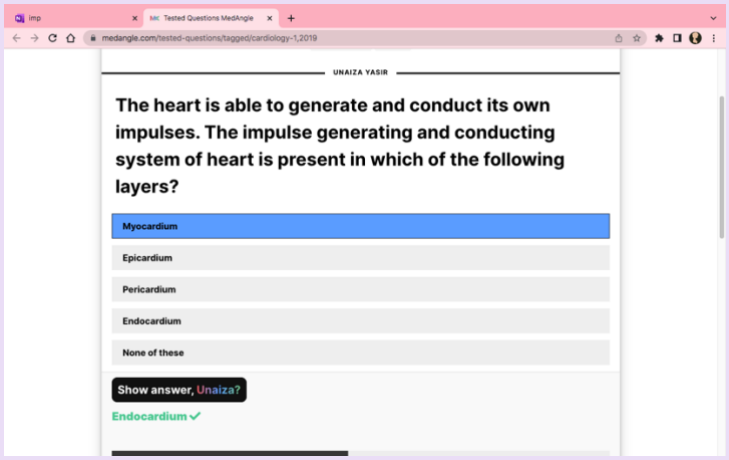
Blowing diastolic murmur

Dys pressure Dec

Sys pressure and SV inc

2. Heart rate: CO is directly proportional to HR, provided the other 3 factors remain constant. Moderate change in HR does not alter the CO. If there is a marked ↑ in HR, diastolic period is shortened & ventricular filling is reduced, decreasing the CO. During marked ↓ in HR, the Diastolic period is prolonged, hence vent. filling is more & CO is more, provided other factors remain constant. But the HR is maintained at normal level constantly by regulatory mechanisms.





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A 46 years man with history of rheumatic fever comes in OPD with chest pain and excessive swelling from 3-4 hours, CPK and LDH levels are normal, troponin I is elevated.

78. The person is suffering from:
A) infarction few hrs back
B) digestive disorder
C) severe stress
D) cardiac arrest.
E) muscular and cellular damage

79. What is your prime suggestion?
A) Immediately check Troponin levels
B) Check Ck and LDH levels after 8-10 hrs.
C) ECG says it all.
D) Check AST and ALT level
E) Ca++needs to be checked

80. What do elevated AST and ALT levels indicate?
A) Liver damage
B) Trauma to heart\
C) Renal failure
D) infection
E) myocardial damage

81. What could be your extended suggestion?

Save a copy to Dropbox

E) myocardial damage

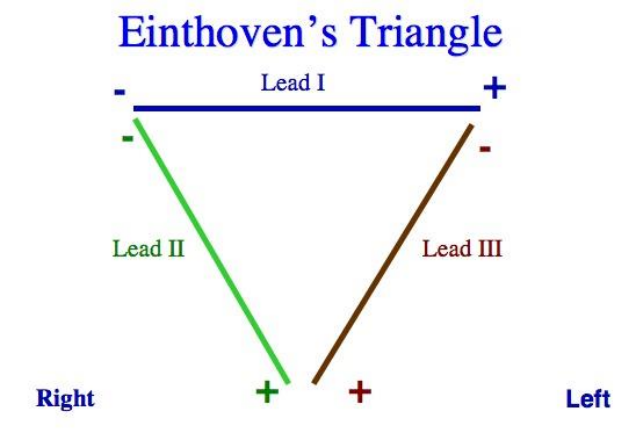
81. What could be your extended suggestion?

A) use a hot water bag to comfort the pain
B) rest
C) eat fibres to improve digestion
D) protein electrophoresis to determine tissue specific CPK or LDH
E) prepare for surgery

82.what suggestion would follow your prime suggestion?
A) get AST and ALT levels checked.
B) apply pressure on pain.
C) intake of water is essential.
D) ECG should be done every 3 hrs
E) get CPK and LDH check again after 8-10 hrs

..mp4 63. Biochemistry....mp4 62. Biochemistry....mp4 61. Biochemistry....mp

to search



1s is rigth arm to left arm 7:4

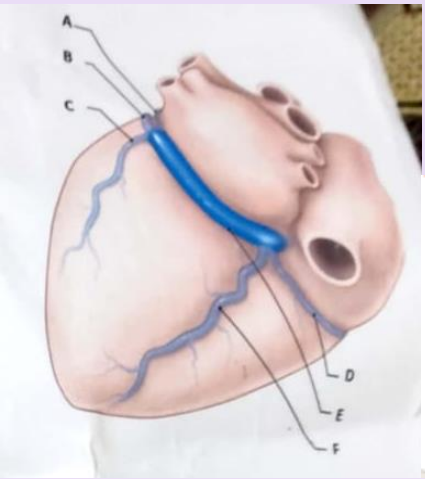
2nd right arm to left leg 7:48

3rdleft arm to left leg 7:48 am

OSPE

STATION 1
1) oxidation def
2) chain breaking antioxidant
3) common oxidants
4) minerals for SOD and Glutathione OP

STATION 3
1) chloride shift in systemic and pulm arteries
2) anion gap and when is it increased
3) hypocholermia ?



A oblique vein of left ventricle
B great cardiac vein
C post vein of left ventricle (koi cnfrm kro)
D small cardiac
E coronary sinus
F middle cardiac/post iv vein 10:08 pm

SOME IMPORTANT TOPICS FOR ASSSESSMENT:

Important O.S.P.Es.:

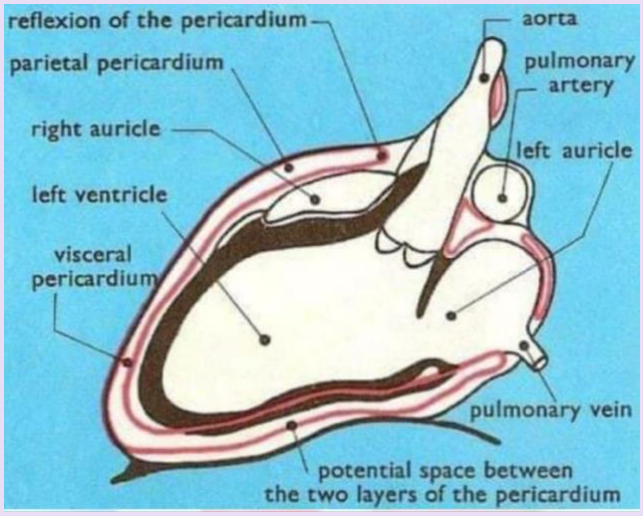
1. Anterior View of Heart (From KLM Anatomy {7th Ed. Pg. 137 Fig. 1.52B})
2. Basal View of Heart (From Netter's Atlas {7th Ed. Plate # 218})
3. Valves of Heart (From Netter's Atlas {7th Ed. Plate # 226})
4. Wigger's Diagram (From Guyton's Physiology {13th Ed. Pg. 114 Fig. 9.7})
5. Normal ECG (From Guyton's Physiology {13th Ed. Pg. 132 Fig. 11.1})
6. Fetal Circulation (From KLM Embryology {8th Ed. Pg. 328 Fig. 13.46} OR see in appendix)
7. Neonatal Circulation (From KLM Embryology {8th Ed. Pg. 329 Fig. 13.47} OR see in appendix)
8. Pulmonary & Systemic Circulation (https://i.pinimg.com/564x/85/59/fa/8559fab677c26e764adef9da479e5500.jpg)
9. Embryonic Heart (From KLM Embryology {8th Ed. Pg. 298 Fig. 13.12D})
10. Pericardium (see in Appendix)
11. Varicose Veins (From KLM Anatomy {7th Ed. Pg. 42 Fig. B1.9})
12. Atherosclerosis (From Robbin's Basis {9th Ed. Pg. 491 Fig. 11.6})
13. Acute Myocardial Infarct (From Robbin's Basis {9th Ed. Pg. 544 Fig. 12.13})

Important C.B.Ls.:

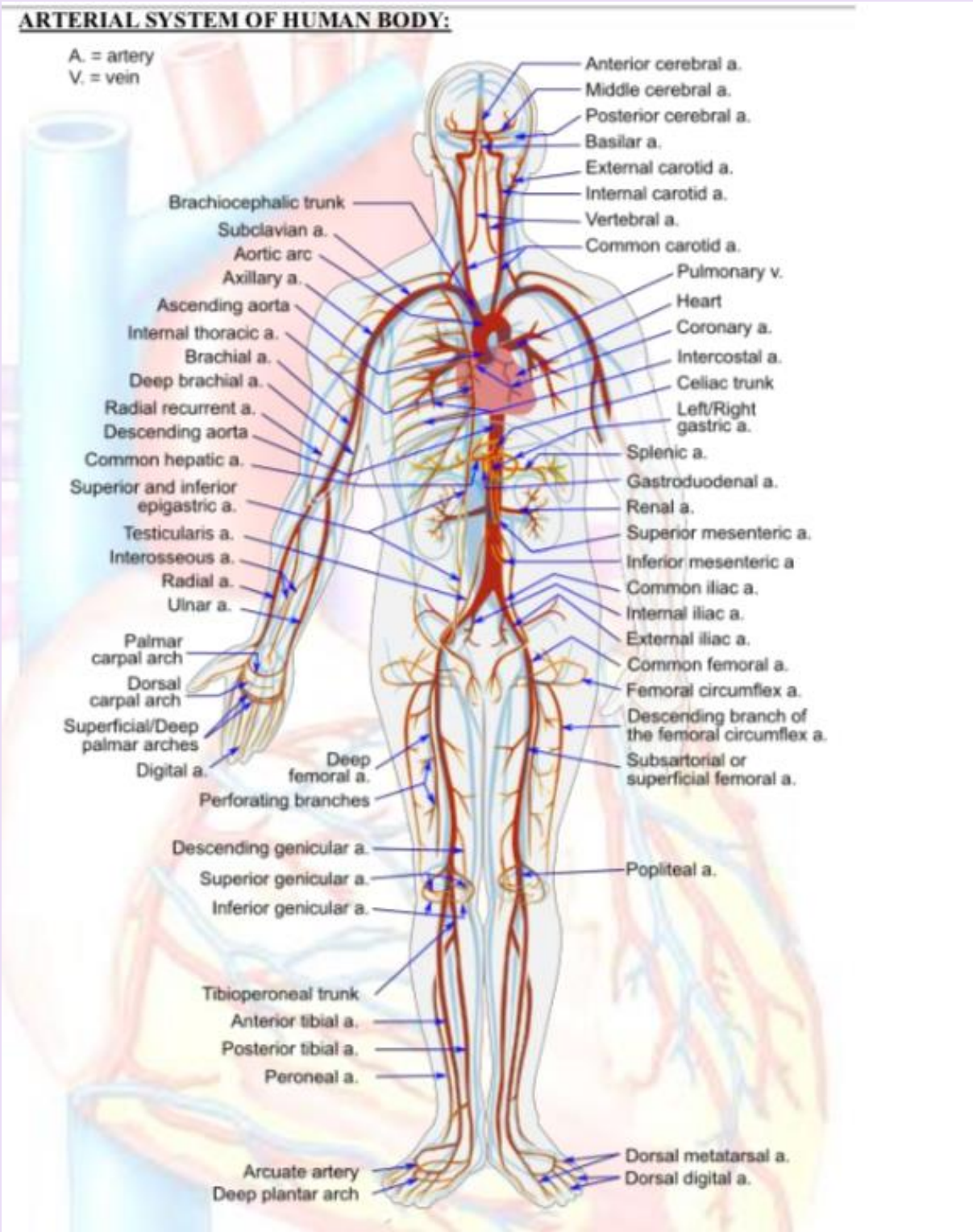
1. Hypertension
2. Myocardial Infarction
3. Rheumatic Heart Disease
4. Tetralogy of Fallot
5. Congenital Heart Disease

IMPORTANT FORMULAE AND VALUES

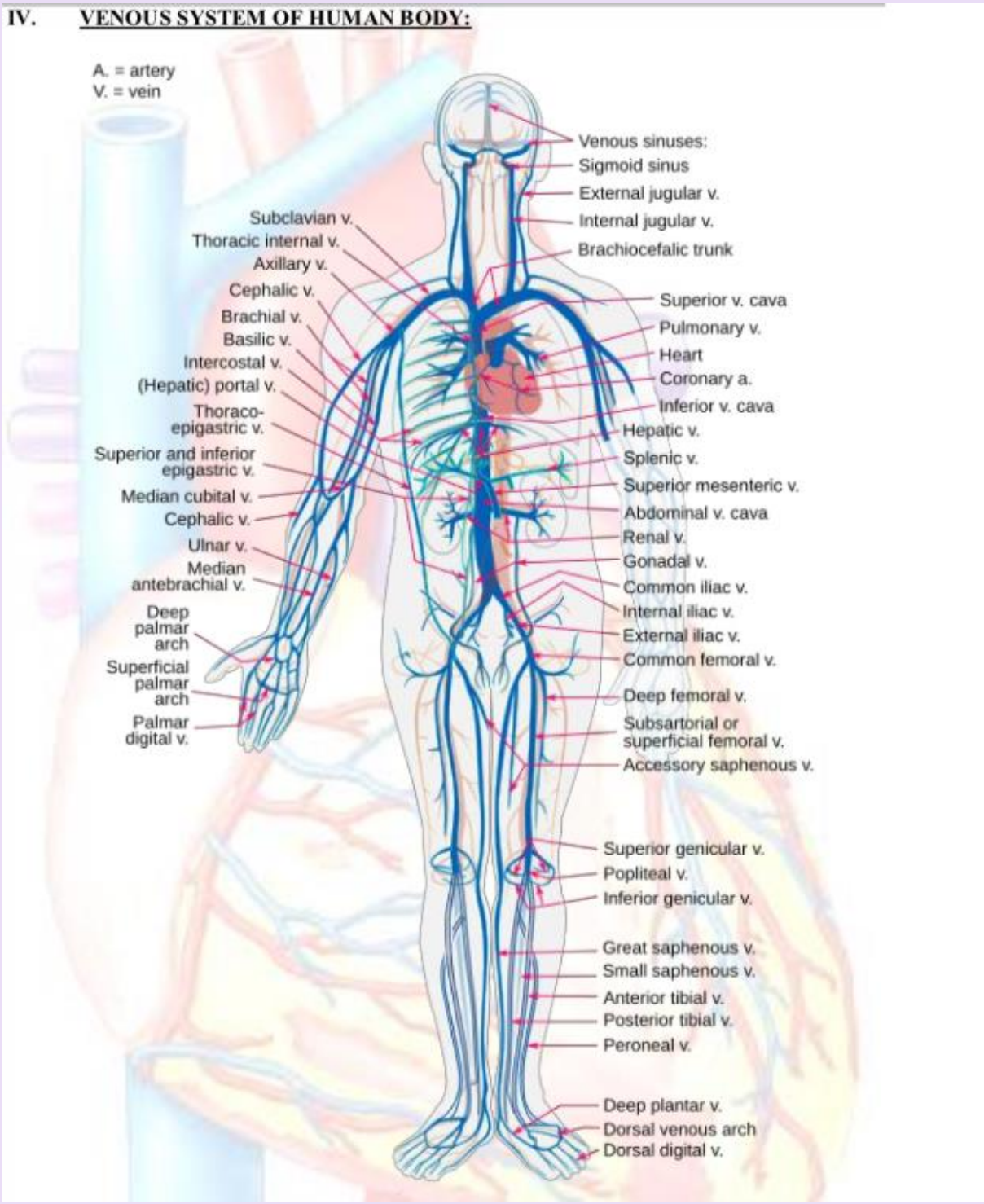
Arterial BP: Diastolic BP + Pulse Pressure/3
SVC Pressure: 0 mm of Hg
Venule Pressure: 10 to 12 mm of Hg
Capillary Pressure: 10 mm of Hg
Semilunar Valve opening Pressure: 80 mm of Hg
Semilunar Valve closing Pressure: 100 mm of Hg
AV Valve opening Pressure: 0 mm of Hg
AV Valve closing Pressure: 4 to 8 mm of Hg
End Diastolic Volume: 120 ml
End Systolic Volume: 50 ml
Stroke Volume: 120 - 50 = 70 ml
Ejection Fraction: 60%
Duration of S1: 0.14 s
Duration of S2: 0.11 s
Reynold's Number: 200 to 400
Duration of Systole: 0.33 s
Duration of Diastole: 0.5 s
Duration of Cardiac Cycle: 0.5 + 0.33 = 0.83s

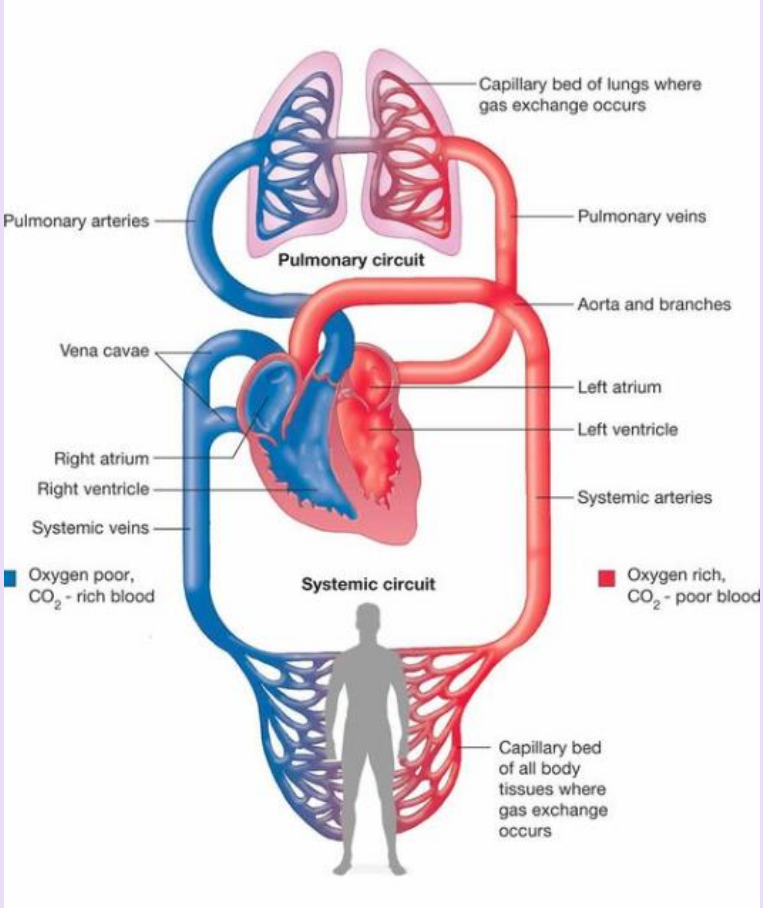


ARTERIAL SYSTEM OF HUMAN BODY:



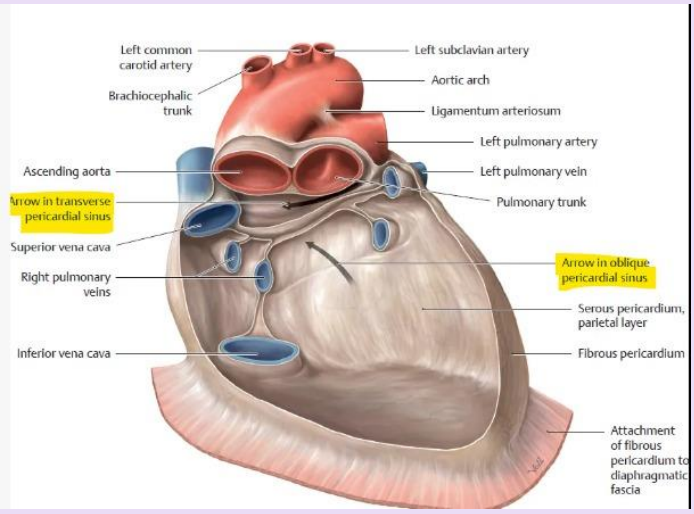
IV. VENOUS SYSTEM OF HUMAN BODY:

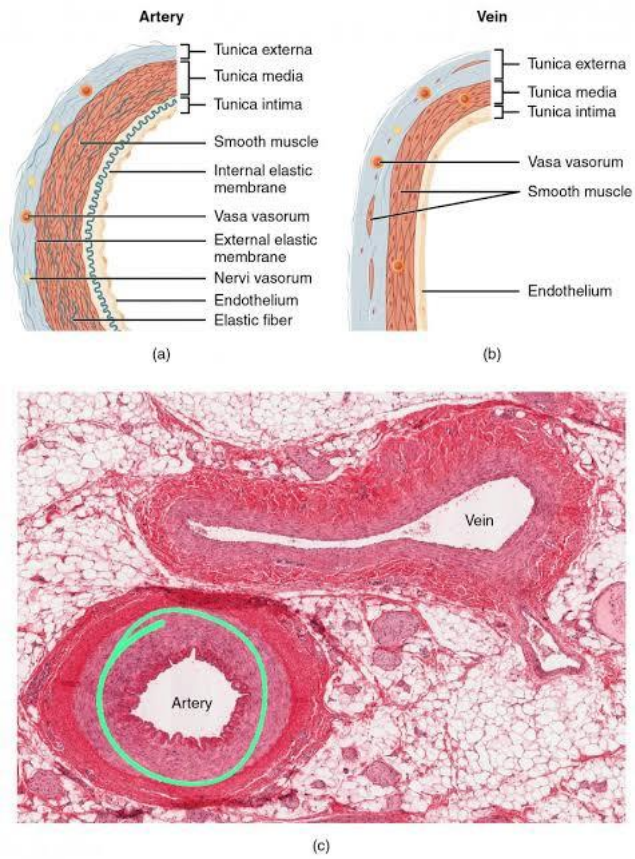


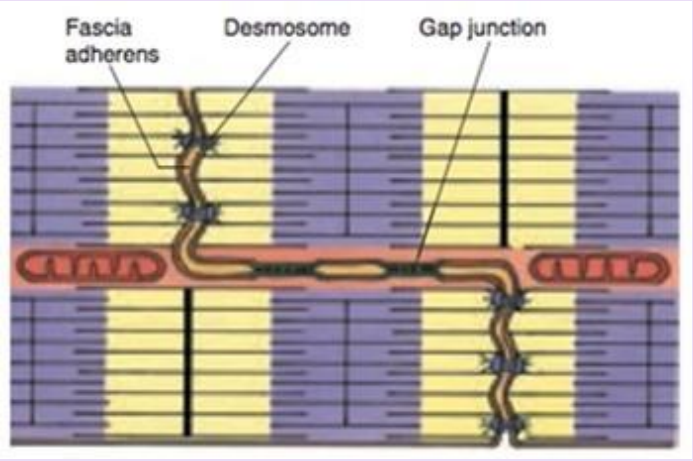


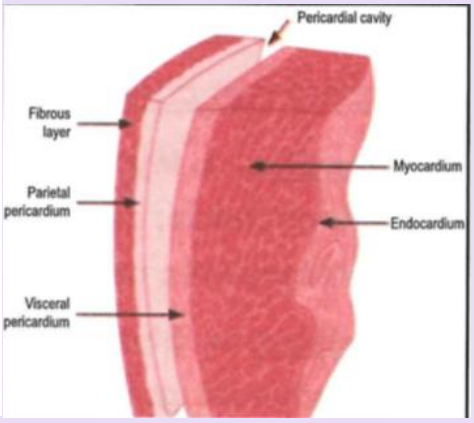
CVS-CASE BASED LEARNING

35 years old male visited a clinic for executive checkup. He admitted having occasional / episodic headache and chest pain on exertion. He has family history of Hypertension and is engaged in a stressful job. Examination revealed obese adult with height of150cm, weighing 100kg with BMI of 33.his BP was 170/110.He started arguing with the doctor that his BP is raised because of stress, as last time his BP was normal about at week ago. He consulted his family physician about three months back when he passed small stone in the urine; his BP was slightly raised as was told by his doctor at that time. His BP was checked on follow up visits and was found to be variable from 140/90 to 160/100. His fasting blood glucose was 120mg/dl, cholesterol 240mg/dl, LDL 160mg/dl, HDL 35, VLDL 45mg/dl, triglycerides 290mg/dl, uric acid 7.4mg/dl, blood urea 40 and creatinine 1.2. Urine detailed report shows trace proteins and few RBCs. His ECG and X-ray chest PA view are shown in pictures.

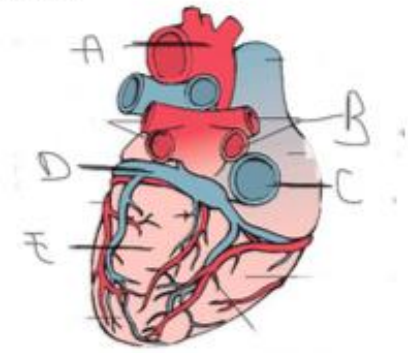

Left common carotid artery
Brachiocephalic trunk
Ascending aorta
Arrow in transverse pericardial sinus
Superior vena cava
Right pulmonary veins
Inferior vena cava
Left subclavian artery
Aortic arch
Ligamentum arteriosum
Left pulmonary artery
Left pulmonary vein
Pulmonary trunk
Arrow in oblique pericardial sinus
Serous pericardium, parietal layer
Fibrous pericardium
Attachment of fibrous pericardium to diaphragmatic fascia


Artery
Vein
Tunica externa
Tunica media
Tunica intima
Smooth muscle
Internal elastic membrane
Vasa vasorum
External elastic membrane
Nervi vasorum
Endothelium
Elastic fiber
(a)
Tunica externa
Tunica media
Tunica intima
Vasa vasorum
Smooth muscle
Endothelium
(b)
Vein
Artery
(c)


Fascia adherens
Desmosome
Gap junction


Pericardial cavity
Fibrous layer
Parietal pericardium
Visceral pericardium
Myocardium
Endocardium

OSPE 2:



96)Which structure Drains the lower part of the body?
a)E
b)B
c)C
d)D
e)A

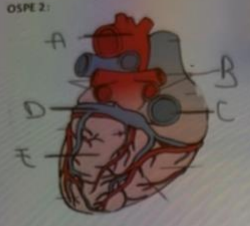
97)The structure labelled D Is called:
a)Coronary sinus
b)Left marginal vein
c)left circumflex vein
d)SVC
e)Middle cardiac vein

98)The thickest layer of myocardium is present in:
a)E
b)B
c)C
d)D
e)A

99)The structure labelled A Provides blood to the rest of the body and is called?
a)Brachiocephalic artery
b)Aorta
c)Subclavian artery
d)Carotid artery
e)Pulmonary artery

100)Structure labelled B brings oxygenated blood back to left atrium:
a)Pulmonary veins
b)Pulmonary artery
c)Aorta
d)Vena cava
e)Cephalic vein

OSPE 2:



96)Which structure Drains the lower part of the body?
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c)C
d)D
e)A

C//

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A//

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B//

99)The structure labelled A Provides blood to the rest of the body and is called?
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B//

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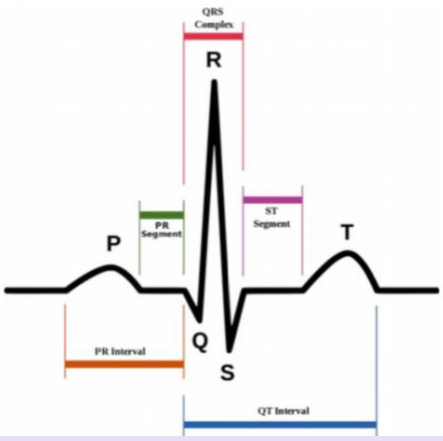
b)Pulmonary artery
c)Aorta
d)Vena cava
e)Cephalic vein

A//

OSPE-3 (93-97)

93. In ECG,the contraction and relaxation of ventricles is represented by
A) QT interval.
B) Q wave
C) P wave.
D) T wave
E) PR interval

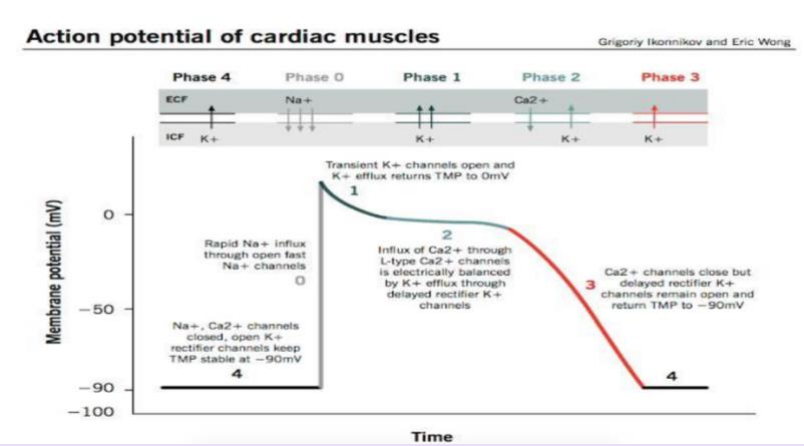


94. The value of PR interval is
A) 0.61 s
B)0.65 s
C) 0.66 s
D) 0.16 s
E) 0.001s

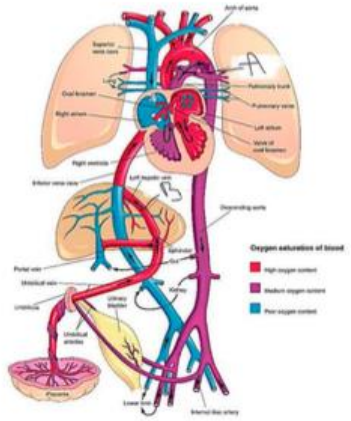
95. QT interval duration in ECG is
A) 0.25s
B) 0.60s
C) 0.61s
D) 0.35s
E) 0.1s

96. P wave is:
A) Depolarization of atria before contraction
B) Re-polarization of atria before contraction
C) Depolarization of ventricles before contraction
D) Re polarization of ventricles before contraction
E) None of the above

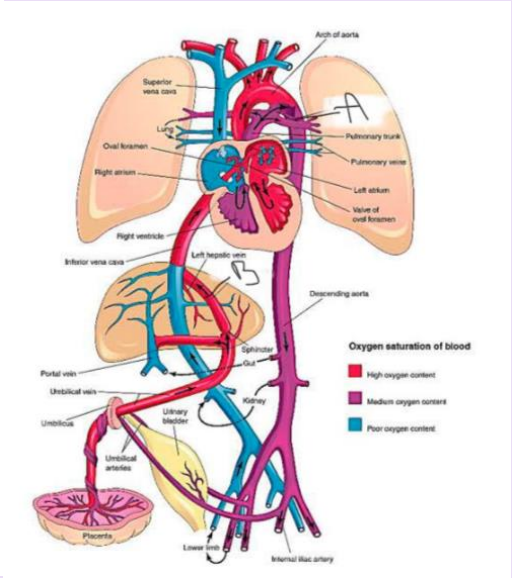
97. T wave represents:
A) Depolarization of atria after contraction
B) Re-polarization of atria after contraction
C) Depolarization of ventricles after contraction
D) Re polarization of ventricles after contraction
E) None of the above

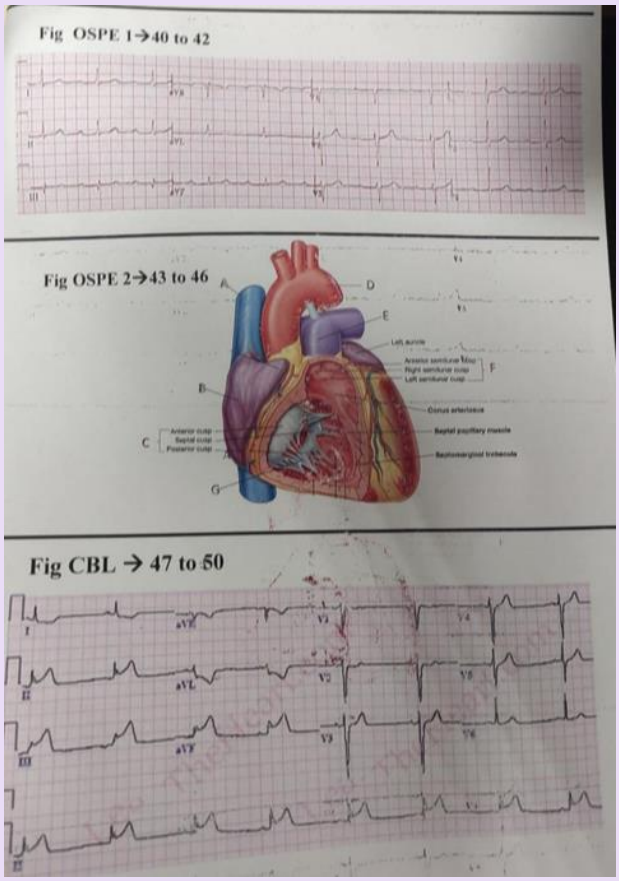


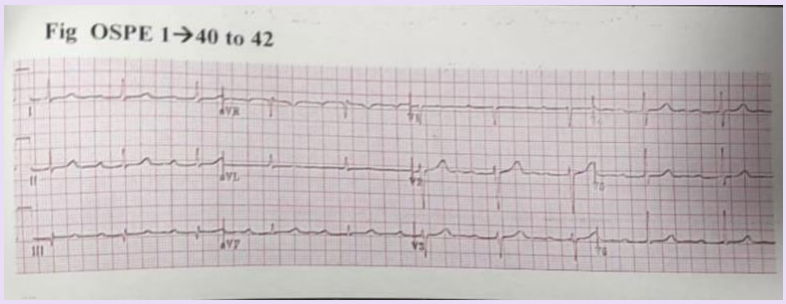
OSPE 1:



82)Choose the correct pairing:
a)Ductus arteriosus -> left subclavian
b)Umblical Arteries -> median umblical ligaments
c)Umblical veins -> ligamentum arteriosus
d)Ductus venosus-> ligamentum venosum







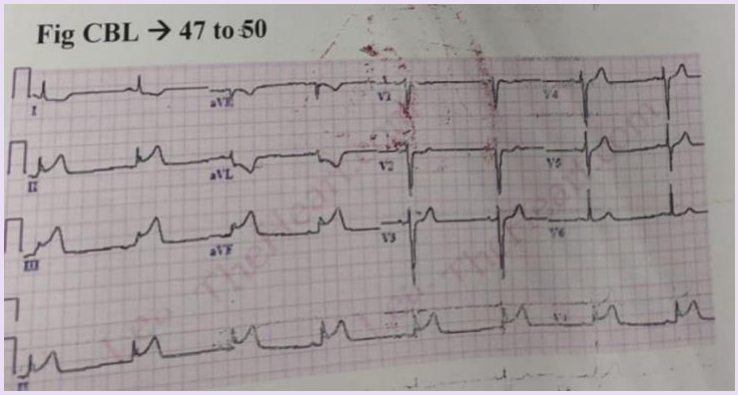


Fig CBL → 47 to 50
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

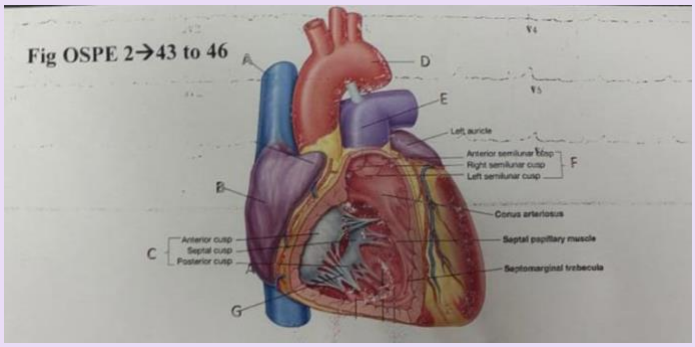
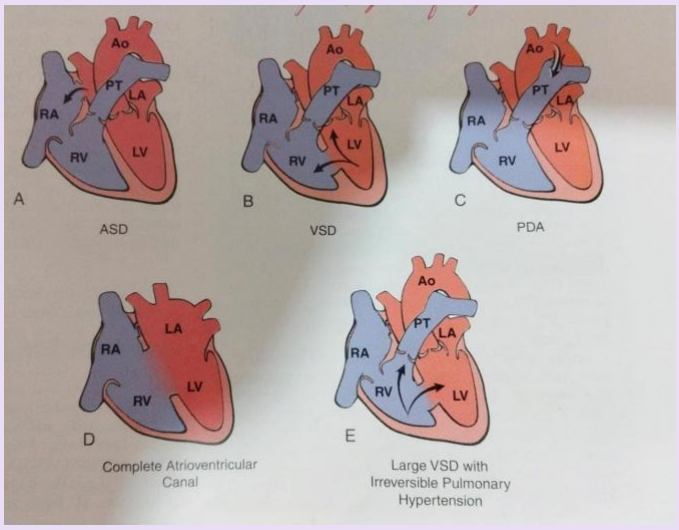
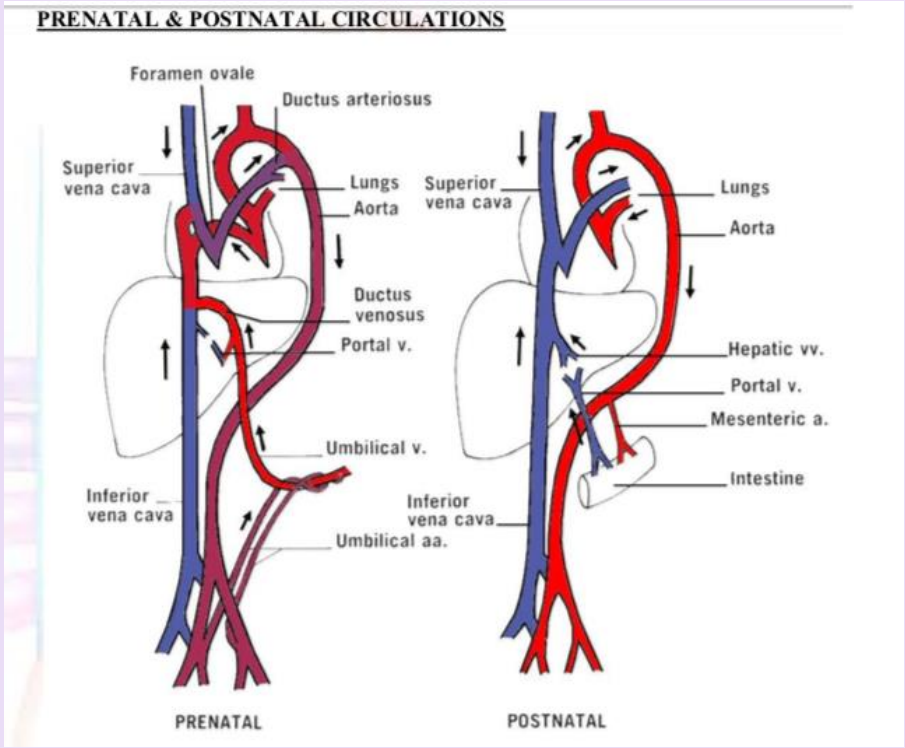
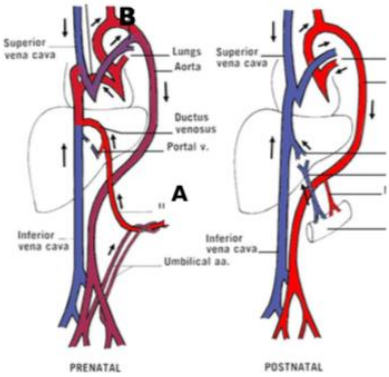


Fig OSPE 2→43 to 46
A
D
E
Left auricle
Anterior semilunar cusp
Right semilunar cusp
Left semilunar cusp
F
B
Conus arteriosus
Anterior cusp
Septal cusp
Posterior cusp
C
Septal papillary muscle
Septomarginal trabecula
G


Ao
PT
LA
RA
RV
LV
A
ASD
B
VSD
C
PDA
D
Complete Atrioventricular Canal
E
Large VSD with Irreversible Pulmonary Hypertension


PRENATAL & POSTNATAL CIRCULATIONS
Foramen ovale
Ductus arteriosus
Superior vena cava
Lungs
Aorta
Ductus venosus
Portal v.
Umbilical v.
Inferior vena cava
Umbilical aa.
Hepatic vv.
Mesenteric a.
Intestine
PRENATAL
POSTNATAL

OSPE-1 (83-87)



83. Which structure is labelled A?
A) Foramen Ovale
B) Ductus venosus
C) Ductus arteriosus
D) Umblical veins
E) Umblical arteries

84. Which structure is labelled B?
A) Foramen Ovale
B) Ductus venosus
C) Ductus arteriosus
D) Umblical veins
E) Umblical arteries

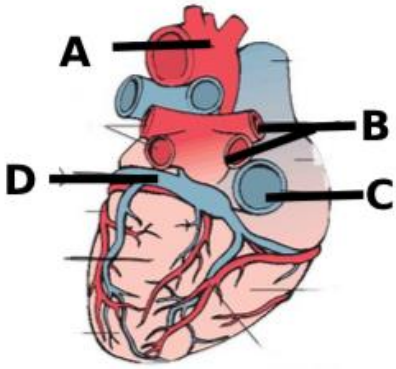
85. Select the incorrect statement:
A) Umblical veins go from navel and enter liver
B) Umblical veins carry oxygenated blood from placenta
C) Pair of Umblical arteries carry dexygenated blood towards placenta
D) Ductus arteriosus connects right ventricle with aorta
E) None of tha above

86. Which of the following is true regarding Foramen ovale?
A) Closes shortly after birth, fuses in 1 year to form fossa ovalis
B) Remains potent after birth
C) Closes shortly after birth, fuses in 2 months to form ligamentum arteriosum
D) Closes late after birth, fuses in 2 years to form ligamentum teres
E) Closes late after birth, in around 10 years

87. Choose the correct pairing:
A) Ductus arteriosus -> left subclavian
B) Umblical Arteries -> median umblical ligaments
C) Umblical veins -> ligamentum arteriosus
D) Ductus venosus-> ligamentum venosum

E) Vitelline veins-> Ligamentum teres

OSPE-2 (88-92)



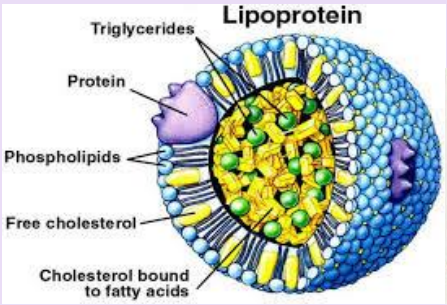
88. The structure labelled D Is called:
A) Coronary sinus
B) Left marginal vein
C) left circumflex vein
D) SVC
E) Middle cardiac vein

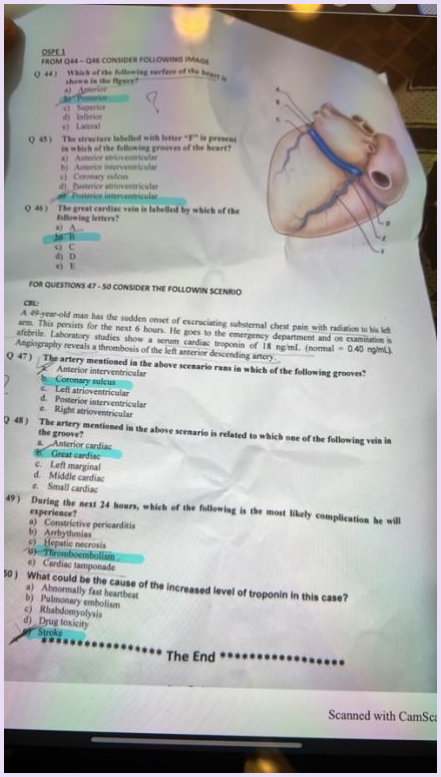
89. The thickest layer of myocardium is present in:
A)E
B) B
C) C
D) D
E) A

90. The structure labelled A Provides blood to the rest of the body and is called?
A) Brachiocephalic artery
B) Aorta
C) Subclavian artery
D) Carotid artery
E) Pulmonary artery

91. Structure labelled B brings oxygenated blood back to left atrium:
A) Pulmonary veins
B) Pulmonary artery
C) Aorta
D) Vena cava
E) Cephalic vein

92. Which structure Drains the lower part of the body?
A) E
B) C
C) D
D) B
E) A





OSPE 1
FROM Q44 – Q46 CONSIDER FOLLOWING IMAGE

Q 44) Which of the following surface of the heart is shown in the figure?
a) Anterior
b) Posterior
c) Superior
d) Inferior
e) Lateral

Q 45) The structure labelled with letter "F" is present in which of the following grooves of the heart?
a) Anterior atrioventricular
b) Anterior interventricular
c) Coronary sulcus
d) Posterior atrioventricular
e) Posterior interventricular

Q 46) The great cardiac vein is labelled by which of the following letters?
a) A
b) B
c) C
d) D
e) E

FOR QUESTIONS 47 - 50 CONSIDER THE FOLLOWIN SCENRIO

CBL:
A 49-year-old man has the sudden onset of excruciating substernal chest pain with radiation to his left arm. This persists for the next 6 hours. He goes to the emergency department and on examination is afebrile. Laboratory studies show a serum cardiac troponin of 18 ng/mL (normal = 0.40 ng/mL). Angiography reveals a thrombosis of the left anterior descending artery.

Q 47) The artery mentioned in the above scenario runs in which of the following grooves?
a. Anterior interventricular
b. Coronary sulcus
c. Left atrioventricular
d. Posterior interventricular
e. Right atrioventricular

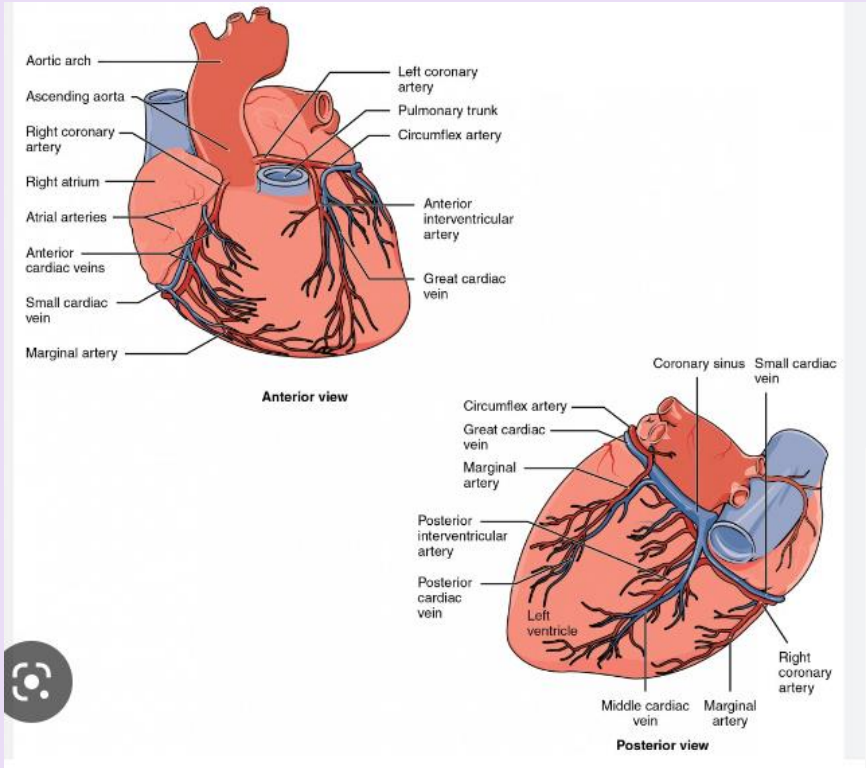
Q 48) The artery mentioned in the above scenario is related to which one of the following vein in the groove?
a. Anterior cardiac
b. Great cardiac
c. Left marginal
d. Middle cardiac
e. Small cardiac

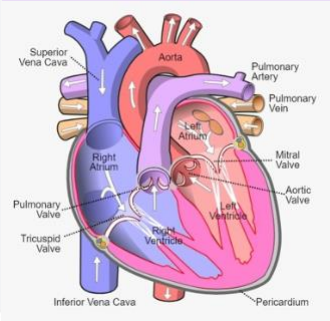
49) During the next 24 hours, which of the following is the most likely complication he will experience?
a) Constrictive pericarditis
b) Arrhythmias
c) Hepatic necrosis
d) Thromboembolism
e) Cardiac tamponade

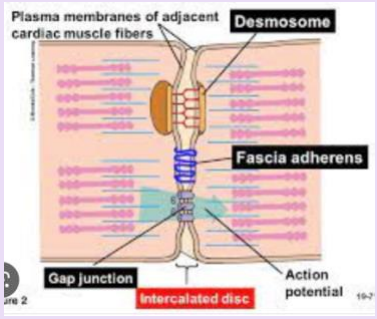
50) What could be the cause of the increased level of troponin in this case?
a) Abnormally fast heartbeat
b) Pulmonary embolism
c) Rhabdomyolysis
d) Drug toxicity
e) Stroke

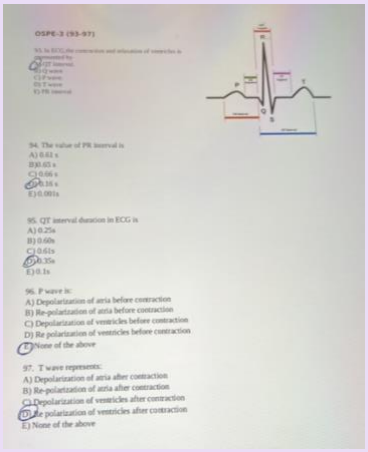
********** The End **********

Scanned with CamSc






Plasma membranes of adjacent cardiac muscle fibers
Desmosome
Fascia adherens
Gap junction
Intercalated disc
Action potential


OSPE-3 (93-97)
94. The value of PR interval is
A) 0.61 s
B) 0.65 s
C) 0.06 s
D) 0.16 s
E) 0.001s
95. QT interval duration in ECG is
A) 0.25s
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96. P wave is:
A) Depolarization of atria before contraction
B) Re-polarization of atria before contraction
C) Depolarization of ventricles before contraction
D) Re polarization of ventricles before contraction
E) None of the above
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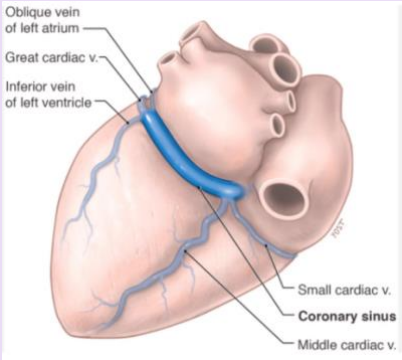
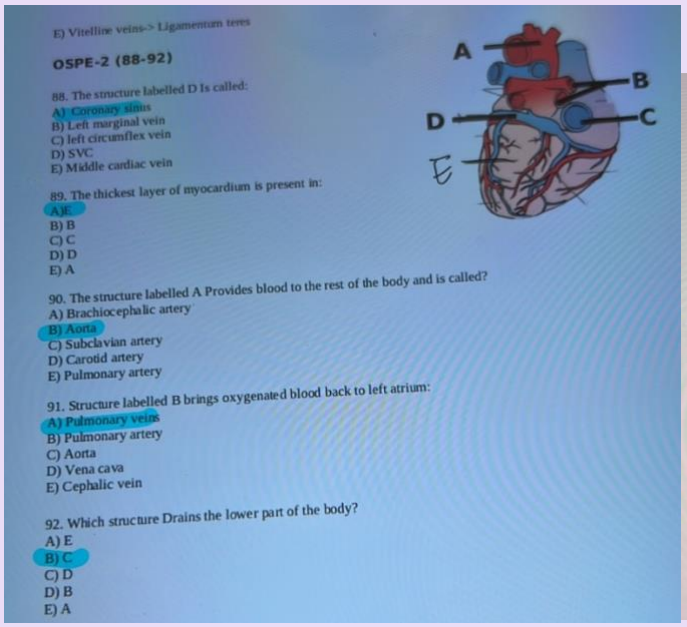
E) None of the above


Oblique vein of left atrium
Great cardiac v.
Inferior vein of left ventricle
Small cardiac v.
Coronary sinus
Middle cardiac v.


E) Vitelline veins-> Ligamentum teres
OSPE-2 (88-92)
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D) SVC
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B) Pulmonary artery
C) Aorta
D) Vena cava
E) Cephalic vein
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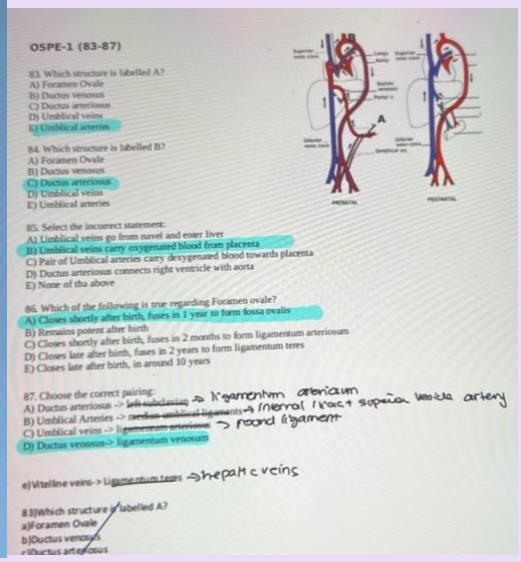
A) E
B) C
C) D
D) B
E) A
A
B
C
D
E


OSPE-1 (83-87)
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B) Ductus venosus
C) Ductus arteriosus
D) Umbilical veins
E) Umbilical arteries
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B) Ductus venosus
C) Ductus arteriosus
D) Umbilical veins
E) Umbilical arteries
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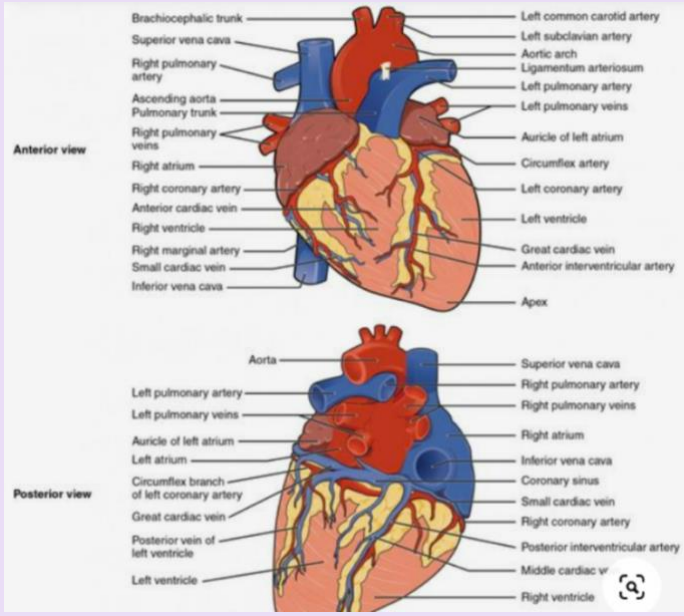
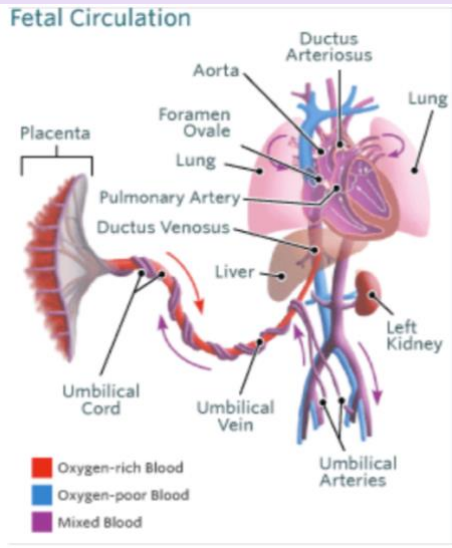
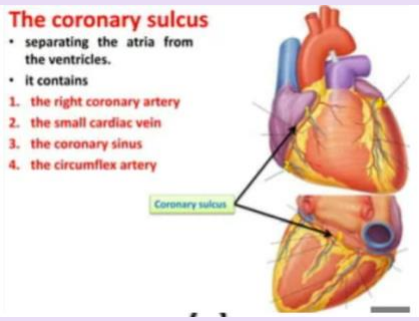
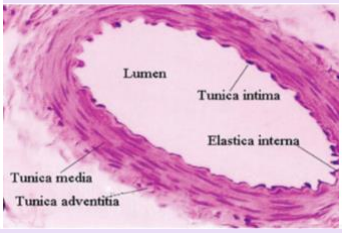
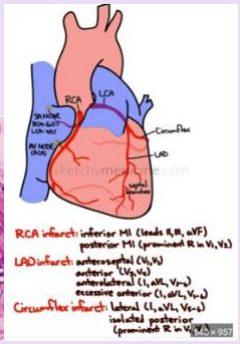
A) Umbilical veins go from navel and enter liver
B) Umbilical veins carry oxygenated blood from placenta
C) Pair of Umbilical arteries carry deoxygenated blood towards placenta
D) Ductus arteriosus connects right ventricle with aorta
E) None of tha above
86. Which of the following is true regarding Foramen ovale?
A) Closes shortly after birth, fuses in 1 year to form fossa ovalis
B) Remains potent after birth
C) Closes shortly after birth, fuses in 2 months to form ligamentum arteriosum
D) Closes late after birth, fuses in 2 years to form ligamentum teres
E) Closes late after birth, in around 10 years
87. Choose the correct pairing:
A) Ductus arteriosus -> left subclavian -> ligamentum arteriosum
B) Umbilical Arteries -> medial umbilical ligaments -> internal iliac + superior vesicle artery
C) Umbilical veins -> ligamentum arteriosum -> round ligament
D) Ductus venosus-> ligamentum venosum
e) Vitelline veins-> Ligamentum teres -> hepatic veins
83)Which structure is labelled A?
a)Foramen Ovale
b)Ductus venosus
c)Ductus arteriosus
A
PRENATAL
POSTNATAL

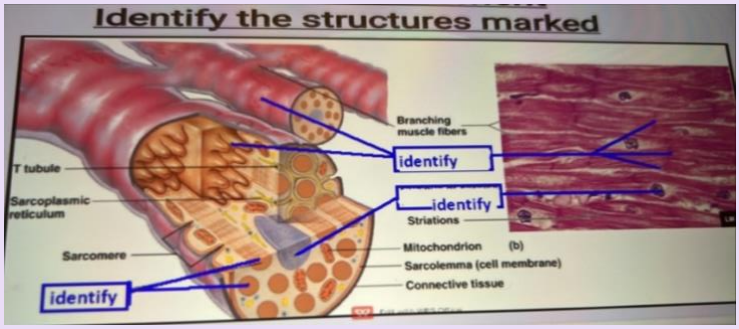

Anterior view
Brachiocephalic trunk
Superior vena cava
Right pulmonary artery
Ascending aorta
Pulmonary trunk
Right pulmonary veins
Right atrium
Right coronary artery
Anterior cardiac vein
Right ventricle
Right marginal artery
Small cardiac vein
Inferior vena cava
Left common carotid artery
Left subclavian artery
Aortic arch
Ligamentum arteriosum
Left pulmonary artery
Left pulmonary veins
Auricle of left atrium
Circumflex artery
Left coronary artery
Left ventricle
Great cardiac vein
Anterior interventricular artery
Apex
Posterior view
Aorta
Left pulmonary artery
Left pulmonary veins
Auricle of left atrium
Left atrium
Circumflex branch of left coronary artery
Great cardiac vein
Posterior vein of left ventricle
Left ventricle
Superior vena cava
Right pulmonary artery
Right pulmonary veins
Right atrium
Inferior vena cava
Coronary sinus
Small cardiac vein
Right coronary artery
Posterior interventricular artery
Middle cardiac v
Right ventricle

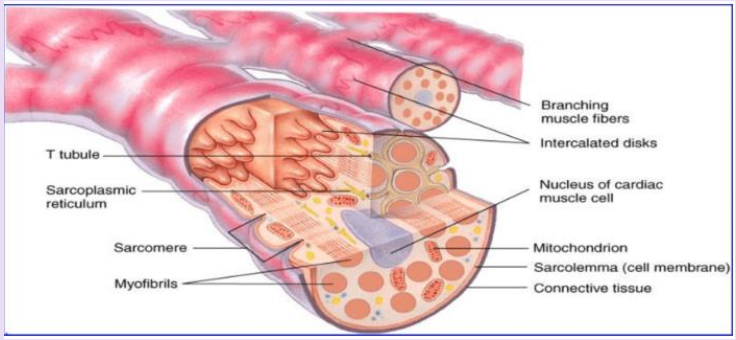

Fetal Circulation
Ductus Arteriosus
Aorta
Lung
Foramen Ovale
Placenta
Lung
Pulmonary Artery
Ductus Venosus
Liver
Left Kidney
Umbilical Cord
Umbilical Vein
Umbilical Arteries
Oxygen-rich Blood
Oxygen-poor Blood
Mixed Blood


The coronary sulcus
separating the atria from the ventricles.
it contains
1. the right coronary artery
2. the small cardiac vein
3. the coronary sinus
4. the circumflex artery
Coronary sulcus


Lumen
Tunica intima
Elastica interna
Tunica media
Tunica adventitia


RCA
LCA
Circumflex
LAD
RCA infarct: inferior MI (leads II, III, aVF)
posterior MI (prominent R in V1, V2)
LAD infarct: anteroseptal (V1, V2)
anterior (V3, V4)
anterolateral (I, aVL, V5-6)
excessive anterior (I, aVL, V5-6)
Circumflex infarct: lateral (I, aVL, V5-6)
isolated posterior
(prominent R in V1)


Identify the structures marked
Branching muscle fibers
identify
T tubule
Sarcoplasmic reticulum
identify
Striations
Sarcomere
Mitochondrion
(b)
Sarcolemma (cell membrane)
Connective tissue
identify


Branching muscle fibers
Intercalated disks
T tubule
Sarcoplasmic reticulum
Nucleus of cardiac muscle cell
Sarcomere
Mitochondrion
Sarcolemma (cell membrane)
Myofibrils
Connective tissue