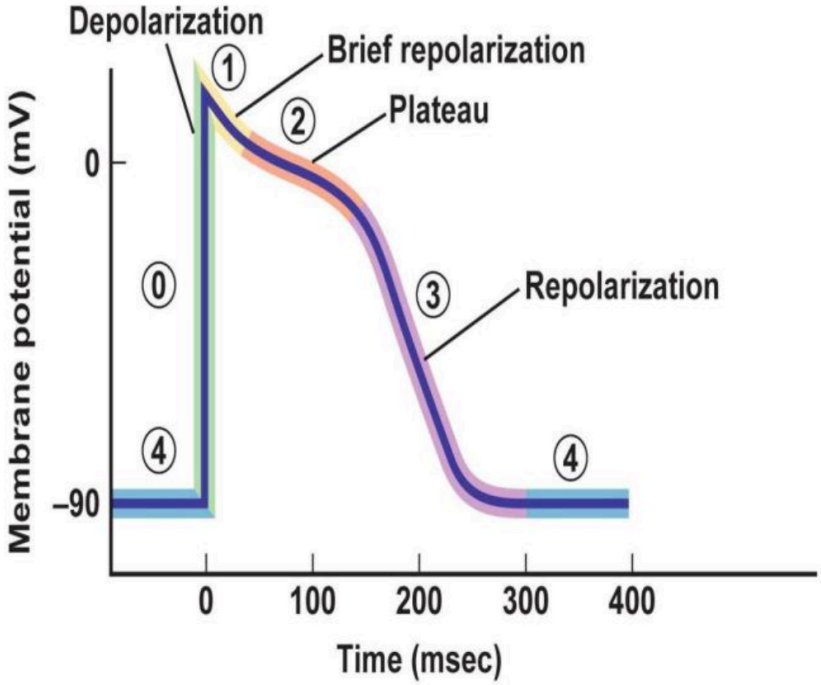
Action potential in Cardiac muscles:

- Resting membrane potential = -90 mV
- Duration of the cardiac action potential = 0.4 s

Phase	Ionic changes	Value
• **Phase 0 :** Depolarization "Rapid depolarization"	• Fast **Na+** channel open , **Na+** flow into the cell.	+20 mV
• **Phase 1 :** Initial "partial" repolarization	• **K+** flow out the cell	5-10 mv
• **Phase 2 :** Action potential Plateau	• **Slow** calcium channels open • Ca++ moving in slowly. • membrane permeability to **K+** will decrease	0 mv
• **Phase 3 :** Rapid repolarization	• **Ca+** channels close • **K+** outflow . • End of plateau .	-
• **Phase 4 :** Resting membrane potential	back to normal level	–90 mV

They might choose a point and ask you what it represents:



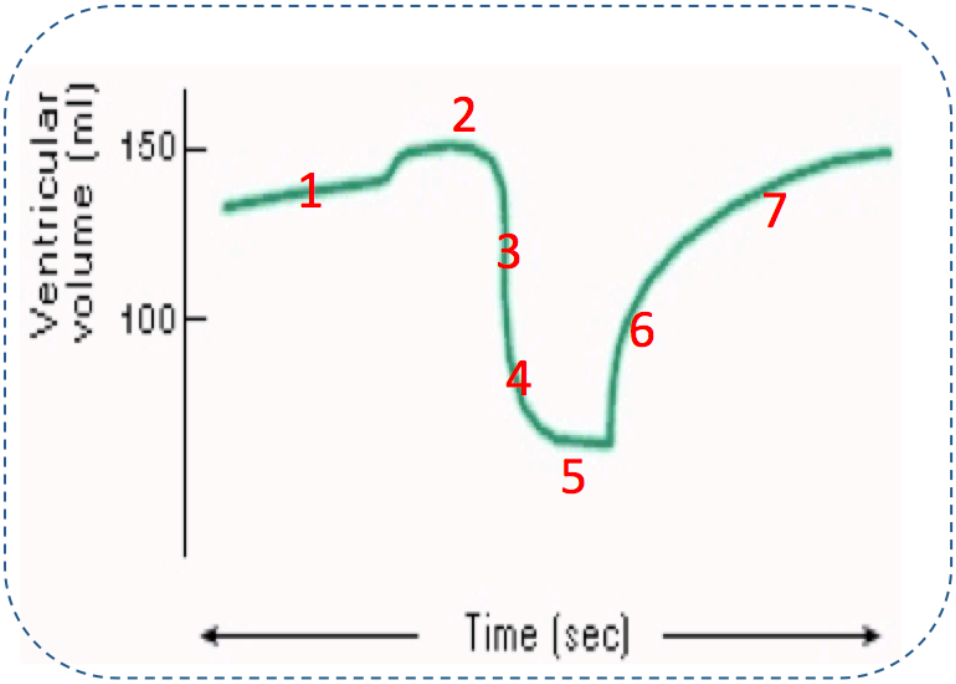
Differentiate between the normal pacemaker, ectopic pacemaker and blockage pacemaker:

	Blockage pacemaker	Ectopic pacemaker	SA Node
Definition	Resulted from blockage of transmission of the cardiac impulse from the sinus node to the other parts of the heart.	A pacemaker elsewhere other than the sinus node.	Located in the superior lateral wall of the right atrium near the opening of the superior vena cava.
Cause	-	Developing of a rhythmical discharge rate that is more rapid than that of the sinus node at any part or the heart.	○ Highest Frequency ○ Is capable of originating **Action Potential.** ○ Its rate of rhythmic discharge is greater than any other part in the heart
Examples	A-V blockage	• A-V node • Purkinje fibers	-
Consequences	Cardiac impulses fail to pass from atria into the ventricles. ↓ Atria beats with its normal rate of rhythm of the (S-A) node. ↓ New pacemaker developed in purkinje fibers with a new rate.	Abnormal heart beats	-

Phase	Definition	Valves	Time	Pressure changes	Sounds	Volume changes
Atrial systole	Atrial systole (atrial contraction) is at the end of ventricular diastole . Atrial systole is **Preceded** by **atrial depolarization**.	AV-vs open (semilunar- vs closed.)	**0.1** sec	↑ Atrial pressure.	4th Heart sound In elderly & pathological conditions	Tops off last 27-30% of ventricular filling. Blood arriving the heart can't enter atria, it flows back up jugular vein.
Isovolumetric Contraction	○ It is the first phase (beginning) of ventricular systole. ○ Period **between** closure of AV- vs & opening of Semilunar- vs. ○ It is **Preceded** by ventricular **depolarization**. Ventricle in this phase is a **closed chamber**	• Starts with closure of **AV- vs**. – Semilunar are already closed • Aortic valve **opens** at the **end** of this phase, when left ventricle **exceeds 80 mmHg**	**0.04** sec	↑ Ventricular pressure	1st Heart sound heard (LUB)	- volume of blood in ventricle = **EDV** - Ventricle contracts with **no changes** in volume.
Rapid Ejection	This is the second phase of the systole phases "rapid ejection phase", the blood will flow from aorta to the rest of the body which will cause reduction of the ventricle pressure Almost **75%** of ventricular blood is ejected, i.e. 75% of **stroke volume**	Semilunar- vs open at beginning of this phase ,when LV pressure exceeds **80 mmHg**.	-	Ventricular pressure reaches **120 mmHg**. which equal to aortic pressure.	-	-
Reduced Ejection	It is the **End** of **systole**. Almost **25%** of ventricular blood is ejected, i.e. 25% of **Stroke volume**	Aortic- v **closes** at the end of this phase when left ventricle pressure reaches **110 mmHg**.	-	-	-	-
Isovolumetric Relaxation	Period **between** closure of semilunar- vs & opening of AV- vs. Happens at the **Beginning** of diastole. It is **Preceded** by ventricular **repolarization**.	AV- vs **open** at the end of this phase; semilunar –vs **close**	**0.04** sec	-	2nd Heart sound heard.	• LV is **a closed chamber,** i.e. **no changes in volume.** • Volume of blood in ventricle = **ESV**
Rapid Filling	From the atrium **60-70%** of blood passes **passively** move to the ventricles along pressure gradient.	AV- vs open. - Semilunar valves are closed	-	Atrial pressure > ventricular pressure	3rd Heart sound heard. (children)	-
Reduced Filling (Diastasis)	Remaining atrial blood flows slowly into ventricles	AV- vs still open - Semilunar valves are closed	-	-	-	-

Volume changes:

Ventricular volume	Phases
↑	1. Atrial systole.
Constant	2.Isometric contraction phase.
↓ Rapidly	3. Rapid ejection phase.
↓ Slowly	4. Reduced ejection phase.
Constant	?.Protodiastole.
Constant	5. Isometric relaxation phase.
↑ rapidly	6. Rapid filling phase.
↑ slowly	7. Reduced filling phase.

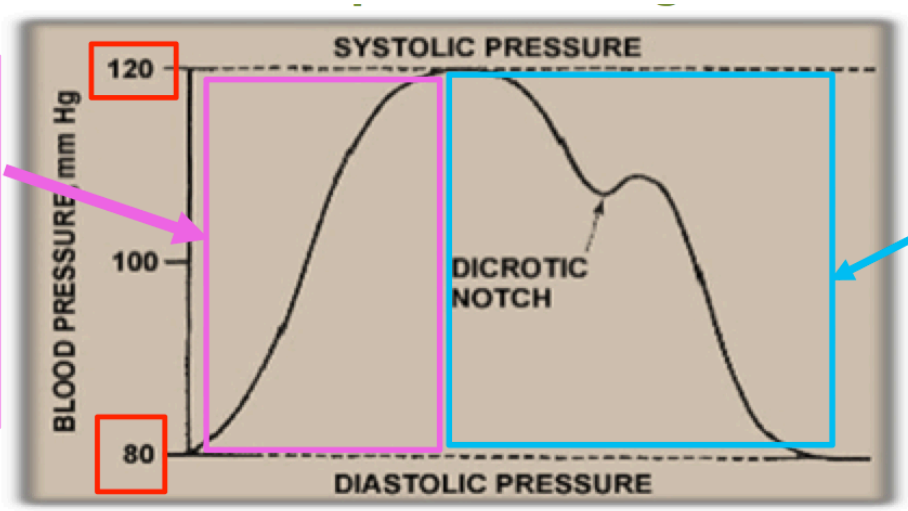


End-diastole volume (EDV)	• Volume of blood in ventricles at the end of diastole. • ≈ **110-130**ml
Stroke volume (SV)	• Amount of blood ejected from ventricles during systole. • ≈ **70**ml/beat
End-systolic volume (ESV)	• Amount of blood left in ventricles at the end of systole. • ≈ **40-60**ml • SV = EDV - ESV
Ejection fraction (EF)	• Fraction of end-diastolic volume that is ejected. • ≈ **60-65**% • $EF = \frac{SV\ (EDV-ESV\ )}{EDV} \times 100$

Pressure changes:

Aortic pressure changes:

Ascending or anacrotic limb:
- With rapid ejection phase.
- Aortic press ↑ up to 120 mmHg.



Descending or catacrotic limb:
- Passes in 4 stages

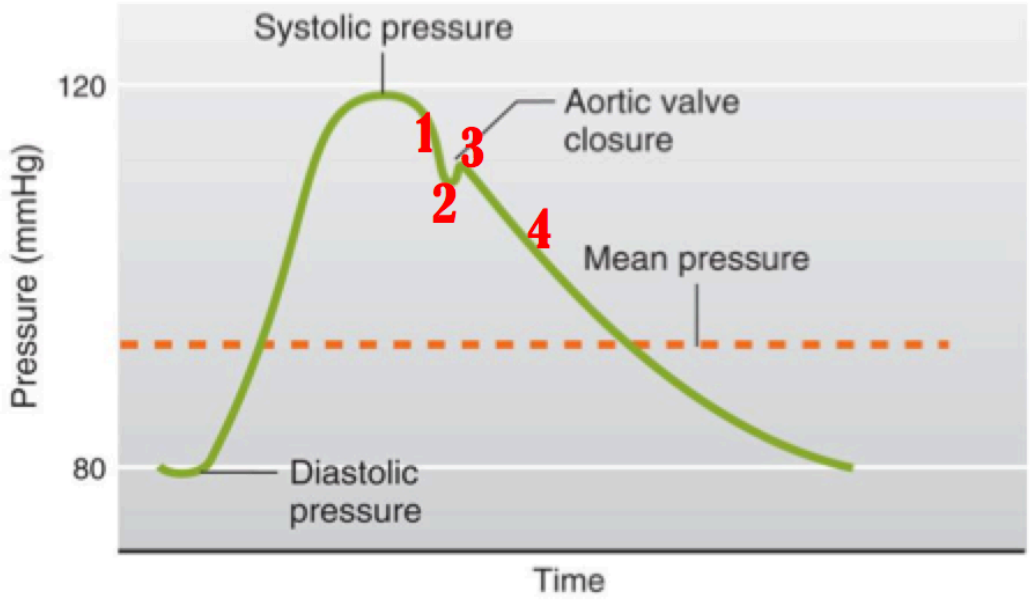
Descending/Catacrotid Limb:

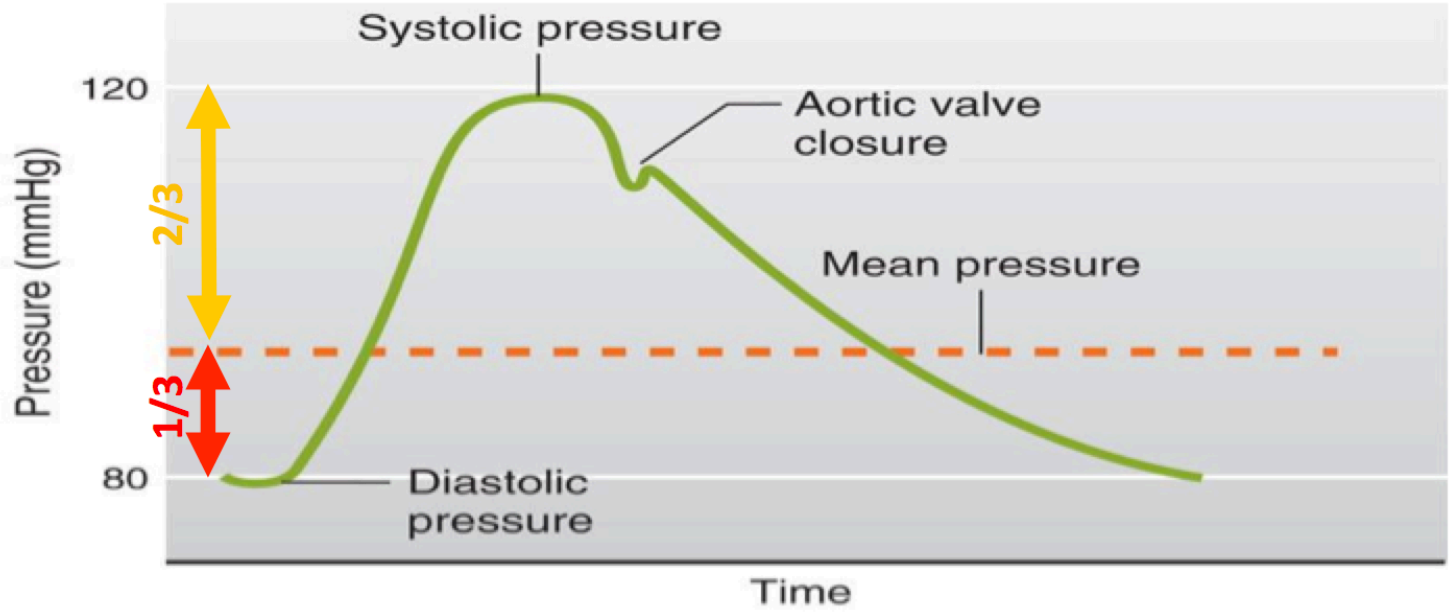
1 ↓ In Aortic pressure:
In reduced ejection phase. When amount of blood that enters aorta is less than the blood that leaves it

2 Incisura (dicrotic notch):
"Due to closure of aortic valve"
Causes sudden drop in aortic pressure.
Happens at the end of ventricular systole.

3 Dicrotic wave:
"Due to elastic recoil of aorta"
Causes slight increase in aortic pressure.

4 Slow decrease of aortic pressure:
Up to 80 mmHg.
"Due to continued flow of blood from aorta to systemic circulation.





Mean pressure:

Diastolic pressure + 1/3 (systolic pressure – diastolic pressure)

Pulse pressure:

Systolic pressure – diastolic pressure

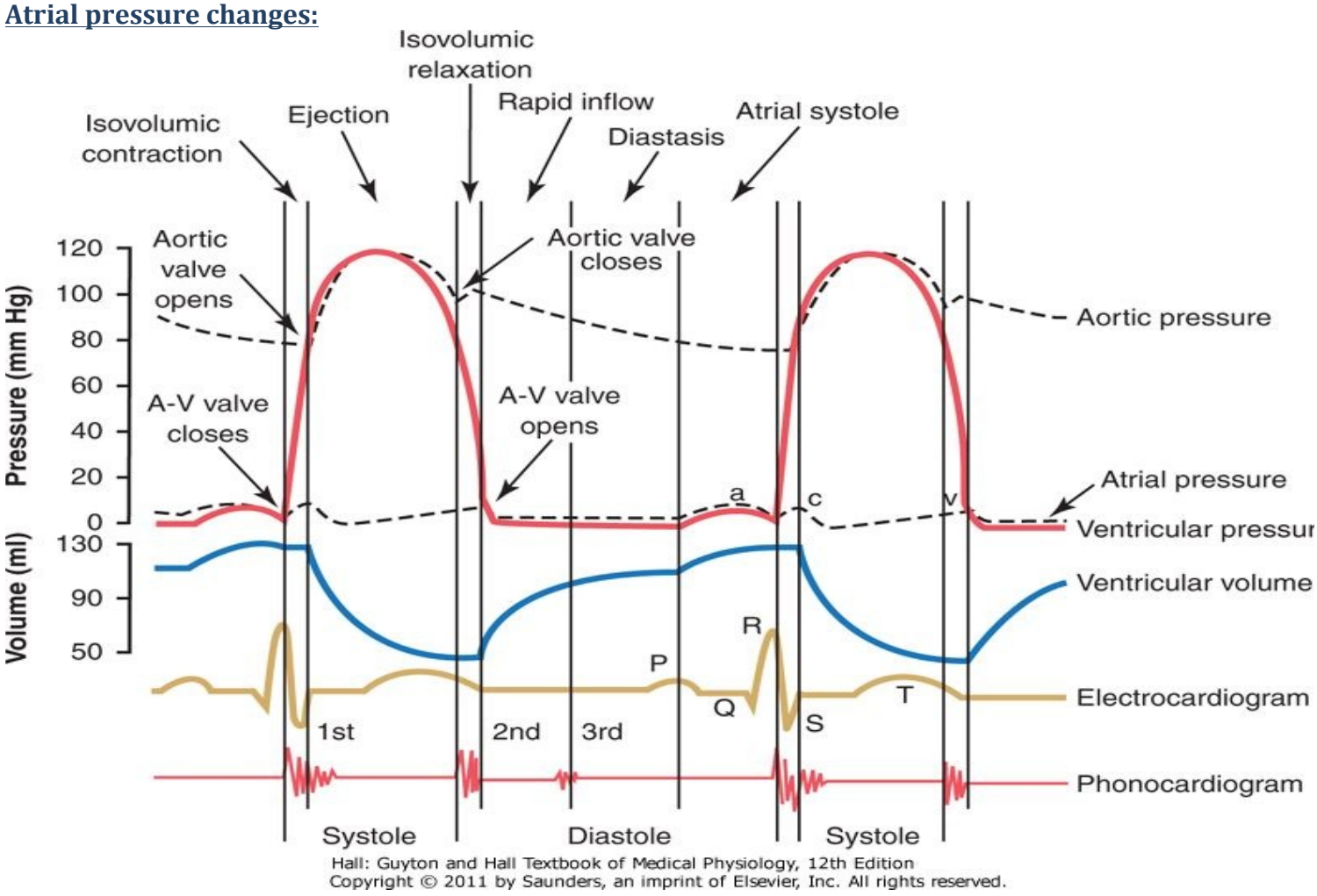
Aortic pressure: is 120/80

Differences between arteries and aorta:

Arterial pressure changes	Pulmonary artery pressure changes

- **110-130/70-85**
  - Similar to aortic pressure waves, but **sharper.**
  - (Its waves are Sharper than aortic pressure waves.)
  - Reflects a systolic peak pressure of **110-130 mmHg** & a diastolic pressure **70-85 mmHg**.
	- **25-30/4-12**
	- Similar to aortic pressure changes, but **with difference in magnitude.**
	- (Differs from aortic pressure changes by magnitude)

Atrial pressure changes:



Causes of Atrial pressure waves:

- 3 upward deflections. (a, c & v)
- 2 downward deflections. (x & y)

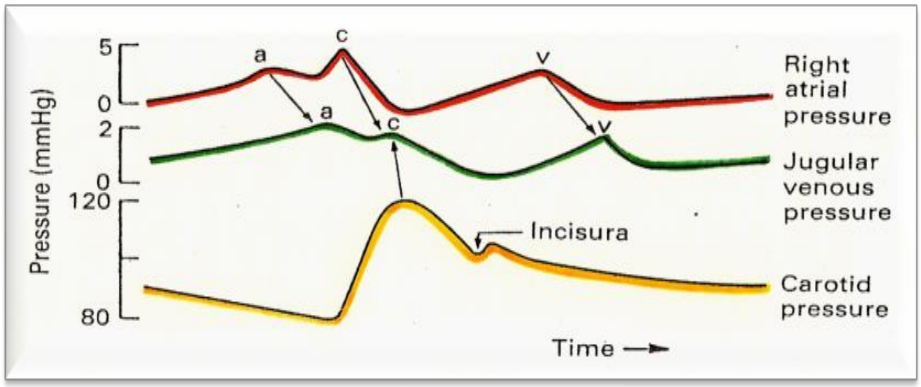
a wave:
(atrial systole)
-Increase in atrial pressure during atrial systole.

c wave:
(ventricular systole)
+ve → bulging of AV valves into atria during "isovolumetric contraction phase"
-ve → pulling of the atrial muscle & AV cusps down during "rapid ejection phase".

v wave:
+ve: increase in atrial pressure due to the increase in venous return during "atrial diastole".
-ve: decrease in atrial pressure during "rapid filling phase"

x decent:
Downward displacement of AV valves during "reduced ejection phase"

y decent:
Decrease in atrial pressure during "reduced filling phase"



- +ve = **increase** in pressure
- -ve = **decrease** in pressure
- The 3 waves a,c&v are equal to **ONE cardiac cycle = 0.8 sec.**

Jugular venous pressure (JVP):

Note that it has similar recordings of transmitted delayed **atrial** waves:
3 upward waves: **a, c, & v**
2 downward waves: **x & y**

Normal heart sounds

S1	S2	S3	S4
Due to closure of the AV- vs.	Due to closure of semilunar-vs.	S3 is usually not audible (very low pitch.)	S4 is usually not audible (very low pitch.)
Recorded at the beginning of the 'isovolumetric contraction phase.'	Recorded at the beginning of the 'isovolumetric relaxation phase.'	Recorded during the ' rapid filing phase ' due to rush of blood into the ventricle.	Recorded during 'atrial systole.'
It marks beginning of ventricular systole.	Marks the beginning of ventricular diastole.	It can be heard in children.	It can heard in elderly & pathological conditions
Long in duration 0.15 sec	Short in duration 0.11-0.125 sec	0.05 sec.	0.04 sec.
Of low pitch (LUB), Loud.	Of high pitch (DUB) Soft & Sharp.	-	-
25-35 Hz.	50 Hz.	-	-
Best heard at Mitral & Tricuspid areas.	Best heard at Aortic & Pulmonary areas.	Best heard at Mitral area.	Best heard at Mitral area.
-	S2 splits physiologically into 2 sounds during inspiration due to delay closure of pulmonary valve = physiological splitting	-	-

ECG:

The axis of ECG graph:

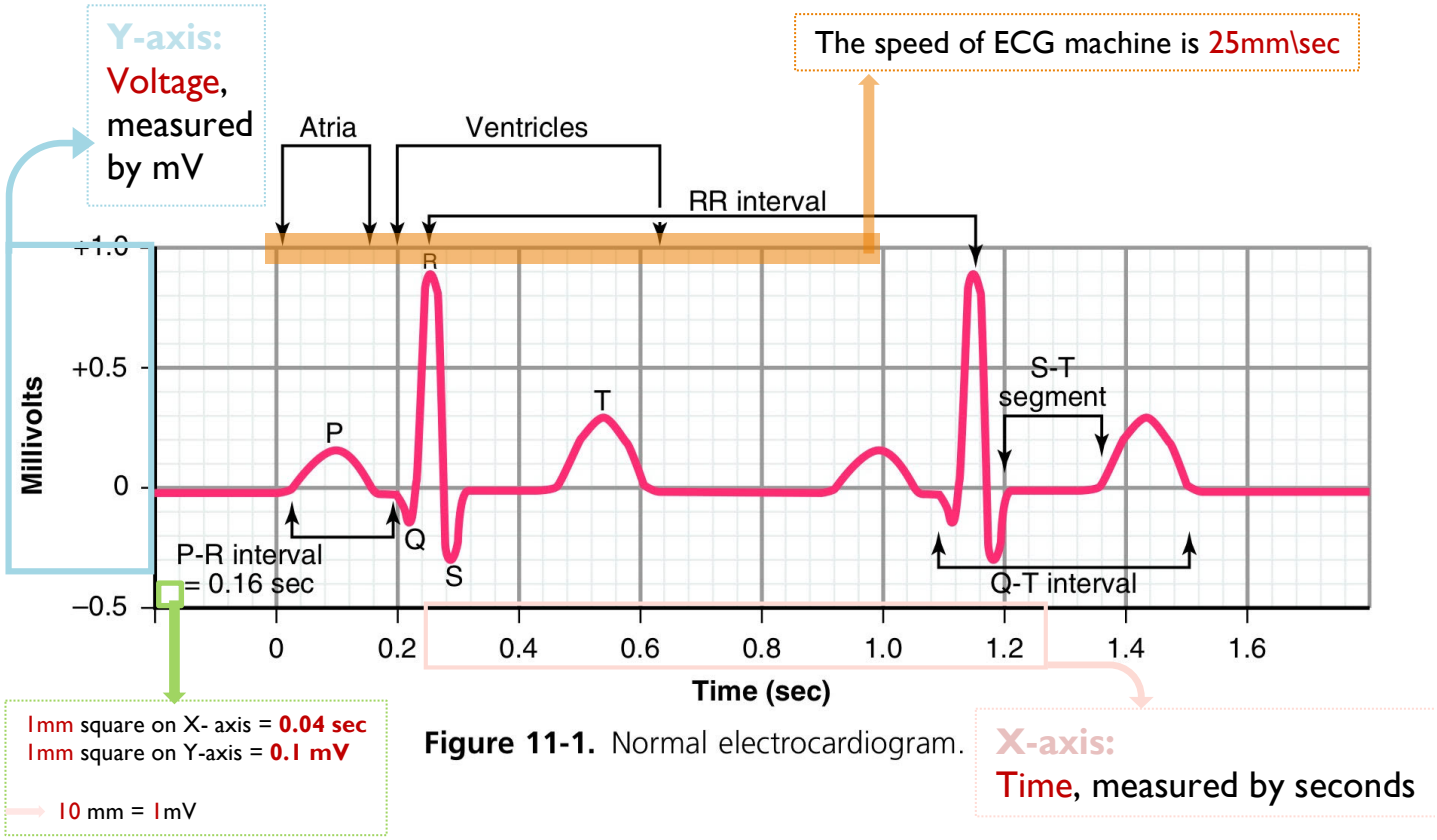
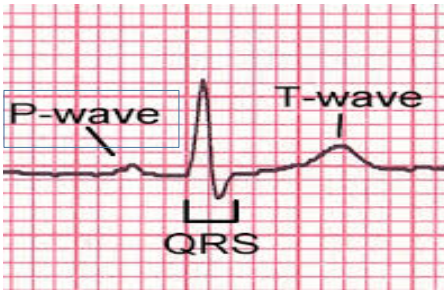
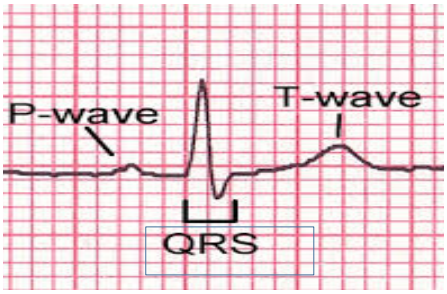
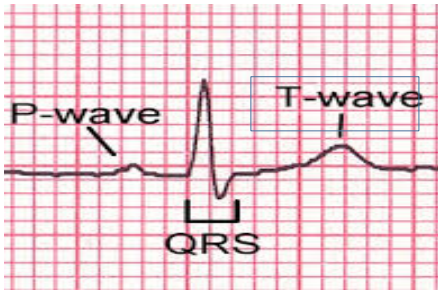


Figure 11-1. Normal electrocardiogram.

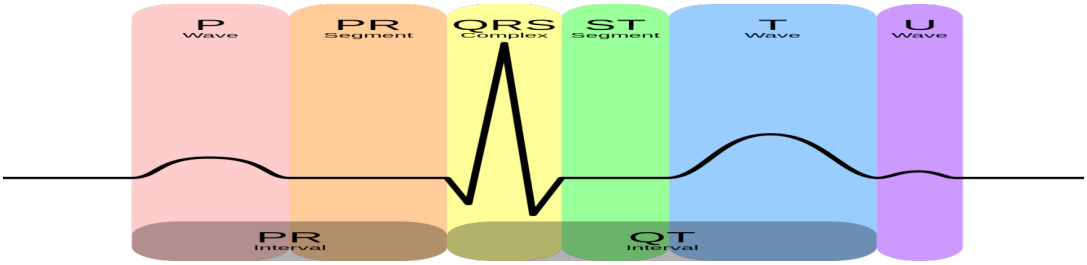
One heartbeat is normally recorded as:

2 segments	**2 interval between waves**	**3 waves**	
PR segment (start from the end of P to the beginning of QRS)	PR interval (stare from the beginning of P to the beginning of Q)	P-wave	
ST segment (start from the end of QRS to the beginning of T)	QT interval (start from the beginning of Q to the end of T)	QRS-complex	
		T-wave	
		2 (-) waves (Q,S)	3 (+) waves (P,R,T)

Name of the waves	T-wave	QRS-complex	P-wave
Diagram showing the waves			
Due to	Ventricular repolarization	Ventricular depolarization	Atrial depolarization
Recorded before	The onset of ventricular Diastole (isometric relaxation phase)	The onset of ventricular systole (isometric contraction phase)	The onset of the Atrial systole

Ventricular depolarization and repolarization (Q-T interval)	Ventricular repolarization (T wave)	Ventricular repolarization (S-T segment)	Ventricular depolarization (QRS)	P-R interval	Atrial depolarization (p-wave)
Including the **QRS** complex , **ST** segment and **T** wave	The repolarization of both ventricles is represented by **T** wave. The **ST** segment and the **T** wave are sensitive indicators of the oxygen demand - oxygen supply status of the ventricular myocardium	Earlier phase repolarization of both ventricles extends from the end of the **QRS** to the beginning of the **T** wave. The point at which the **ST** segment joins the QRS is known as the **J** (junction)- point	Ventricular depolarization is indicated by the **QRS** complex. The R wave is the initial positive deflection; the negative deflection before the **R** wave is the **Q** ; the negative deflection after the **R** wave is the **S** wave	Electrical transmission from the atria to the ventricles. Including the **P** wave and **PR** segment.	The depolarization of both atria is represented by the **P** wave, the **P** wave is the first ECG deflection

QRS	ST segment	QT interval	PR segment	PR interval
<0.10 sec	0.3 - 0.32 sec	0.35 - 0.45 sec	0.06 - 0.11 sec	0.12 - 0.20 sec



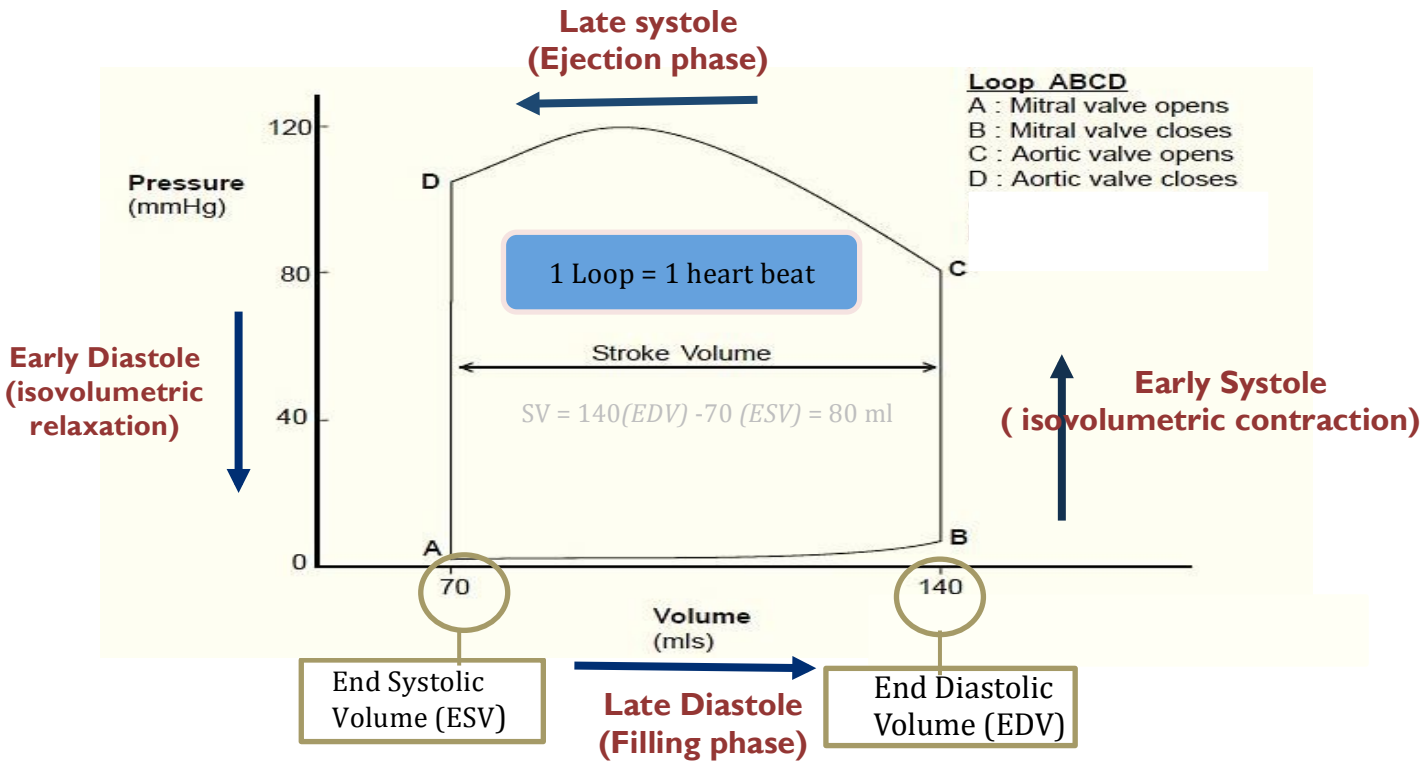
Left Ventricular Pressure - Volume Loop:

Both ventricular systole and diastole can be divided into:

Early phase	Diastole	Systole
	Early diastole : Isovolumetric Relaxation	Early systole Isovolumetric Contraction
Late phase	Late diastole : Isotonic Relaxation *(Filling Phase)*	Late systole : Isotonic Contraction *(Ejection Phase)*

The following Volume-Pressure loop shows plots of the left ventricle pressure values *(Y-axis)* and volume values *(X-axis)* through one complete cardiac cycle:

- **Systole** is divided into early and late systoles. (Begins at B & end at D)
- **Diastole** is divided into early and late diastoles. (Begins at D & end at B)



1- At point A the mitral valve opens then it closes at point B between them the diastolic filling occurs (A & B).
2- At point C the aortic valve opens then closes at D point between them ejection phase occurs (C & D).

Stroke Volume: It is the volume of blood pumped from one ventricle of the heart with each beat.
Stroke Volume = End diastolic - End Systolic (SV = EDV – ESV)

ECG:

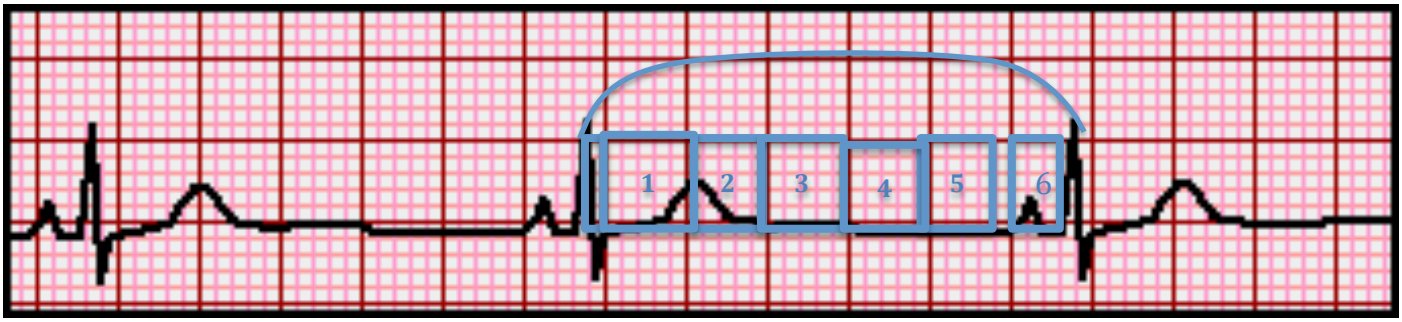
Einthoven's Law:

- It states that if the electrical potential of any two of the three bipolar limb leads are known, the third one can be determined mathematically by summing the first two.

  (Note the +ve and -ve signs).
- The sum of voltages in **Lead I** and **Lead III** is equal to the voltage in **Lead II**.

Arrhythmias:

Rate= divide **300** by the number of boxes between each QRS e.g.:



Rate= 300/6= 50 bpm

Number of big boxes	Heart Rate
1	300
2	150
3	100
4	75
5	60
6	50

>100 bpm is tachycardia

<60 bpm is bradycardia

60-100 bpm is normal

Heart rate variable depends on:

- Age
- Activity
- Time of the day

Common arrhythmias:

Rate / Location	Bradyarrhythmia	Tachyarrhythmia
SA node	Sinus bradycardia Sick sinus syndrome	Sinus tachycardia
Atria	---	Atrial premature beats - Atrial flutter - Atrial fibrillation Paroxysmal SVT (supraventricular tachycardia) - Multifocal atrial tachycardia
Av node	Conduction blocks Junctional escape rhythm	---
ventricles	Ventricular escape rhythm	Ventricular premature beats - Ventricular tachycardia Torsade's de pointes - Ventricular fibrillation

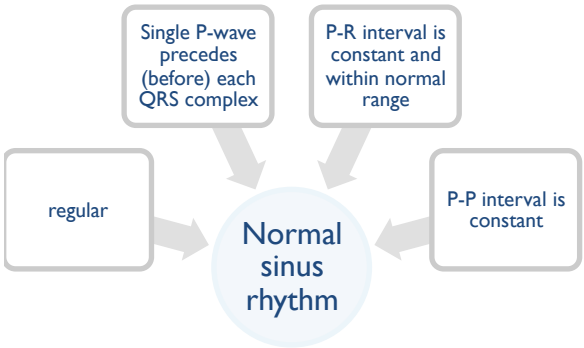
Differential diagnosis of tachycardia:

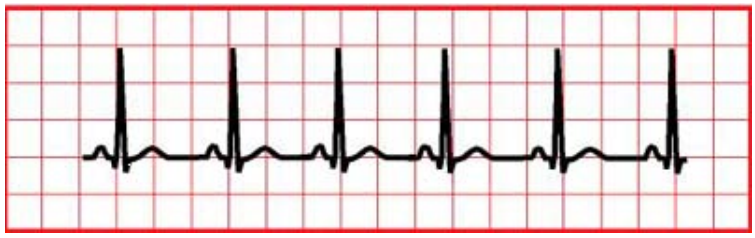
Tachycardia	Narrow Complex	Wide Complex
Regular	**ST** (sinus tachycardia) **SVT** (supraventricular tachycardia) **Atrial flutter**	**ST** w/ **aberrancy** **SVT** w/ **aberrancy** **VT** (ventricular tachycardia)
Irregular	**A-fib** (atrial fibrillation) **A-flutter** w/ variable conduction **MAT** (multifocal atrial tachycardia)	**A-fib** w/ **aberrancy** **A-fib** w/ **WPW** **(Wolff-Parkinson-White Syndrome)** **VT**

Causes of Cardiac Arrhythmias:

- Spontaneous generation of impulses at any part of the heart
- Abnormal rhythmicity of the pacemaker *(means SA node stops working)*
- Shift of the pacemaker from the sinus node to another place in the heart
- Blocks at different points during the spread of the impulse through the heart
- Abnormal pathways of impulse transmission through the heart
- Rate above or below normal
- Regular or irregular rhythm
- Narrow or broad QRS complex
- Relation to P waves

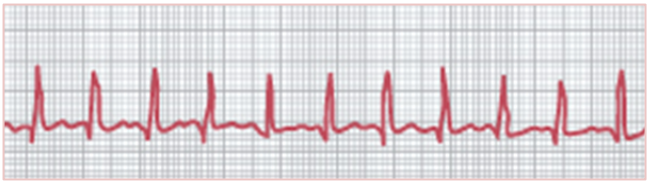
Normal sinus rhythm:





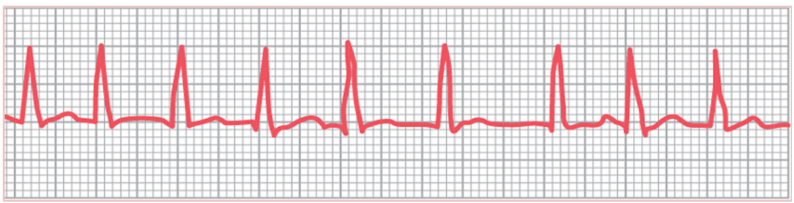
Abnormal sinus rhythm:

Tachycardia	Bradycardia
An increase in the heart rate **Heart rate:** **More than100 beats per minute** **Causes:** • Increased body temperature • Sympathetic stimulation • Drugs such as: digitalis • Inspiration	Means a decrease in heart rate **Heart rate:** Below than **60** beats per minute **Causes:** • Parasympathetic (vagal) stimulation • Expiration



Abnormal Cardiac Rhythms that Result from Impulse Conduction Block:

1- Sinoatrial Block:



- Blockade of the S-A node impulse before entering atrial muscle
- Cessation of P wave *(No SA node activity)*

Causes:

- Ischemia of the A-V node
- Compression of the A-V node by scar formation
- Inflammation of the A-V node
- Strong vagal stimulation

2- A-V Block

When impulse from the S-A node is blocked

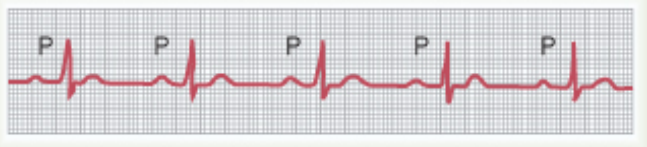
Causes:

- Ischemia of the A-V node
- Compression of the A-V node by scar formation
- Inflammation of the A-V node
- Strong vagal stimulation

Types of the A-V Block

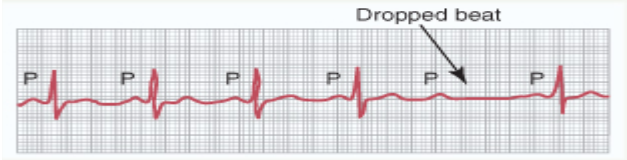
First degree block:

- Prolong P-R interval (0.2 seconds).
- "NO" beats Drop



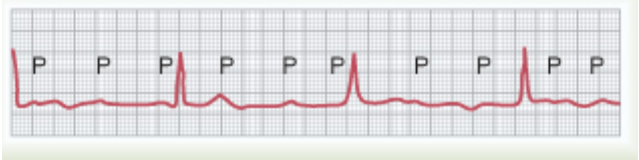
Second degree block:

- P-R interval > **0.25** second.
- Only few impulses pass to the ventricles.
- → atria beat faster than ventricles.
- →"dropped beats" of the ventricles.



Third degree block (complete):

- Complete dissociation of P wave and QRS waves.
- Ventricle escape from the influence of S-A node.
- Atrial rate is **100** beats/min.
- Ventricular rate is **40** beats/min.
- *Stokes-Adams Syndrome*: AV block comes and goes.

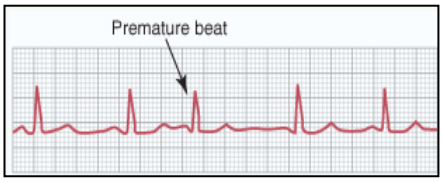
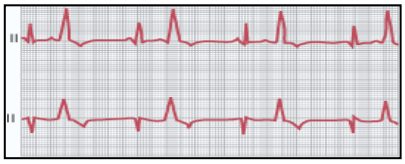


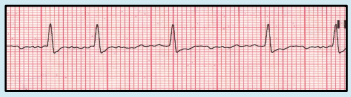
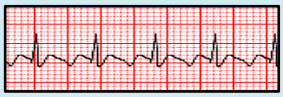
Premature contractions:

Premature contractions, *extrasystoles, or* ectopic beat: result's from *ectopic foci* that generate abnormal cardiac impulses (pulse deficit).
Ectopic foci are abnormal pacemaker sites within the heart (outside of the SA node)
The premature contractions can originate in **atria**, **A-V** junctions or **ventricles**.

Caused By:

- Drugs like caffeine
- Myocardium irritations
- Ischemia

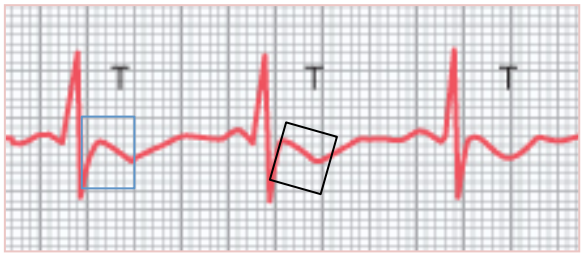
Atrial	**Ventricular (PVC)**
Signs: 1- Short P-R interval (Depends on how far the ectopic foci from the AV node). 2- Pulse deficit If there is no enough time for ventricles to fill with blood. 3- The time between the premature contraction and the next beat is increased (compensatory pause). 	**Signs:** 1- Prolonged QRS: because the impulses are carried out with myocardial fibers with slower conduction rate than purkinje fibers. 2- Increased QRS voltage; QRS from a ventricle cannot neutralize the one from other ventricle. 3- After PVCs, T wave has an electrical potential of opposite polarity of that of QRS because of the slow conduction in myocardial fibers; the fibers that depolarize first will repolarize first. **Causes:** Drugs, Caffeine, Smoking. Lack of sleep, Emotional irritations. 

	Ventricular fibrillation (V-fib)	Atrial fibrillation (A-fib)	Atrial flutter
What is it?	The most serious form of arrhythmias. It is the uncoordinated contraction of the cardiac muscle of the ventricles in the heart. As if the ventricles are quivering not pumping.	Same mechanism as ventricular fibrillation, but as the name suggests it affects ATRIA. 	- A single large wave travels around and around in the **atria**, which results in abnormal heart rhythm. - Contraction of atria occurs at **250** Beats/minute. (high rate) - The refractory period of the AV node causes 2-3 beats of atria for one single ventricular beat **2:1** or **2:3** rhythm. 
Causes	❖ Sudden electric shock or Ischemia. ❖ Impulses stimulate one part of the ventricles, then another, then itself. Many part contracts at the same time while other parts relax (Circus movement).	Frequent in patients with enlarged hearts	
Manifestations	• Tachycardia • Irregular rhythm • Broad QRS complex • No P wave	The efficiency of ventricular filling is decreased 20 to 30%. As seen in the ECG above you either see no P wave or high frequency of low voltage P wave.	
Treatment	Direct current shock (DC).		

Ischemia and ECG:

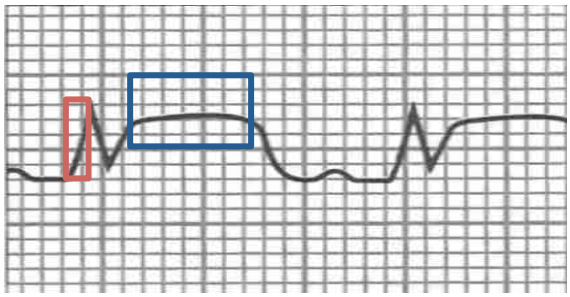
One of the common uses of the ECG is in acute assessment of chest pain
Cause: restriction of blood flow to the myocardium, either:

- Reversible: angina pectoris:
  - Inverted T wave as shown in blue box
  - ST segment depression as shown in black box



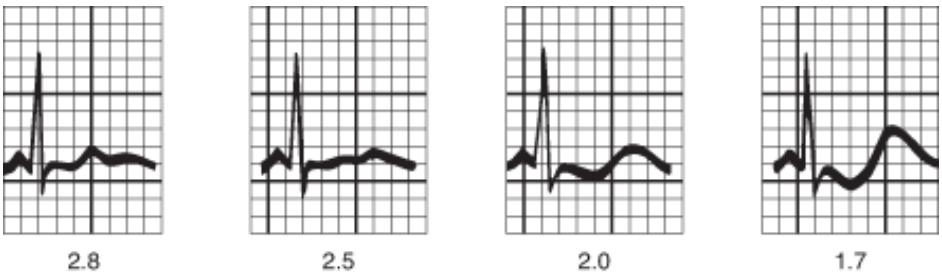
- Irreversible: myocardial infarction

  Cause:
  Complete loss of blood supply to the myocardium resulting in necrosis or death of tissue
  - ST segment elevation as shown in blue box
  - Deep Q wave as shown in red box



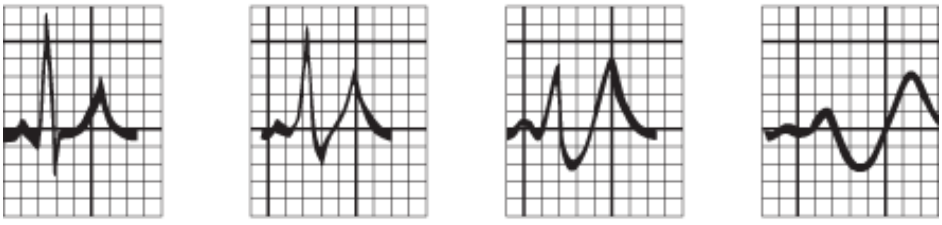
  Ischemia → injury → infarction

K+ and the ECG:



Hypokalemia

Hypokalemia: Flat T wave



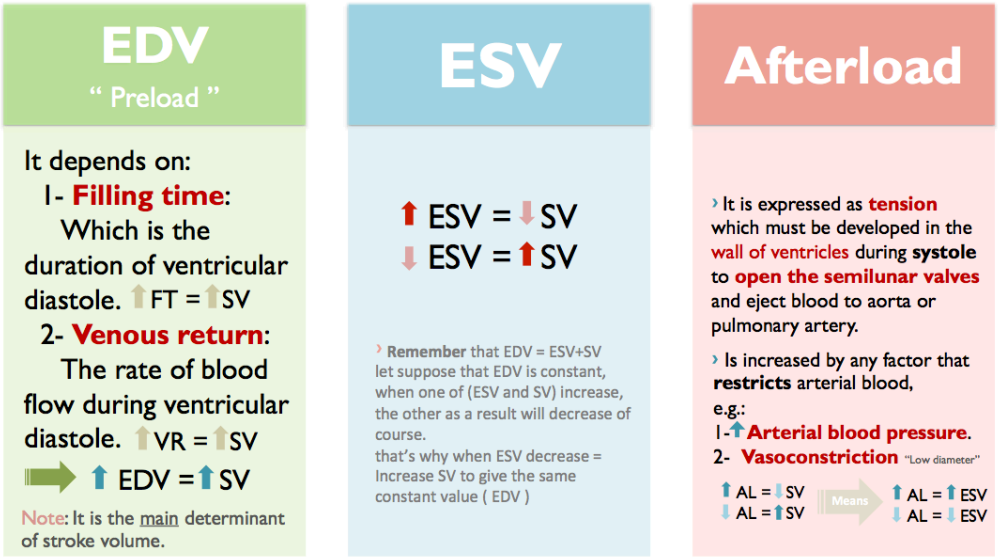
Hyperkalemia

Hyperkalemia: Tall peaked T wave

Stroke Volume:

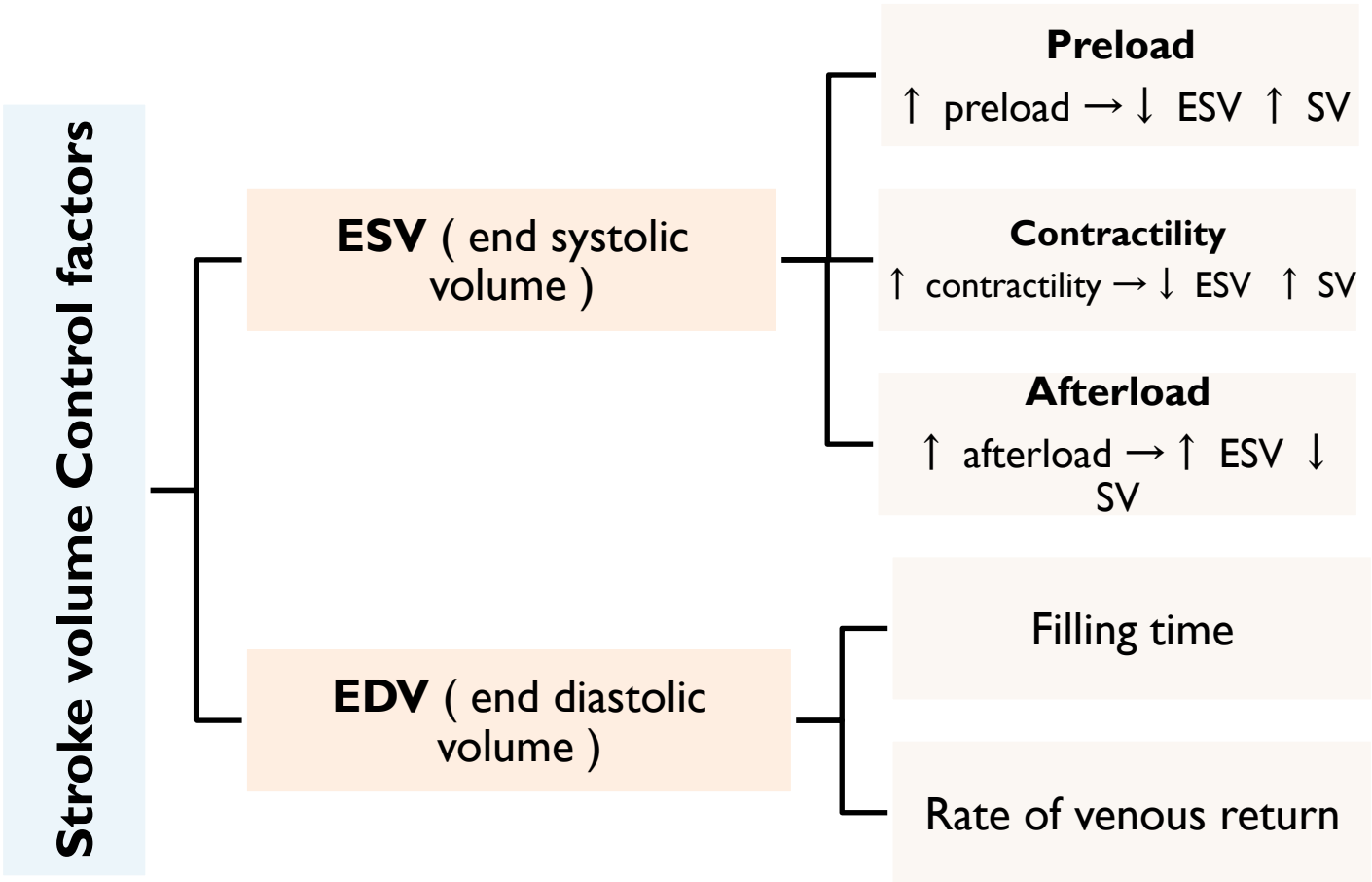
EDV = ESV + SV

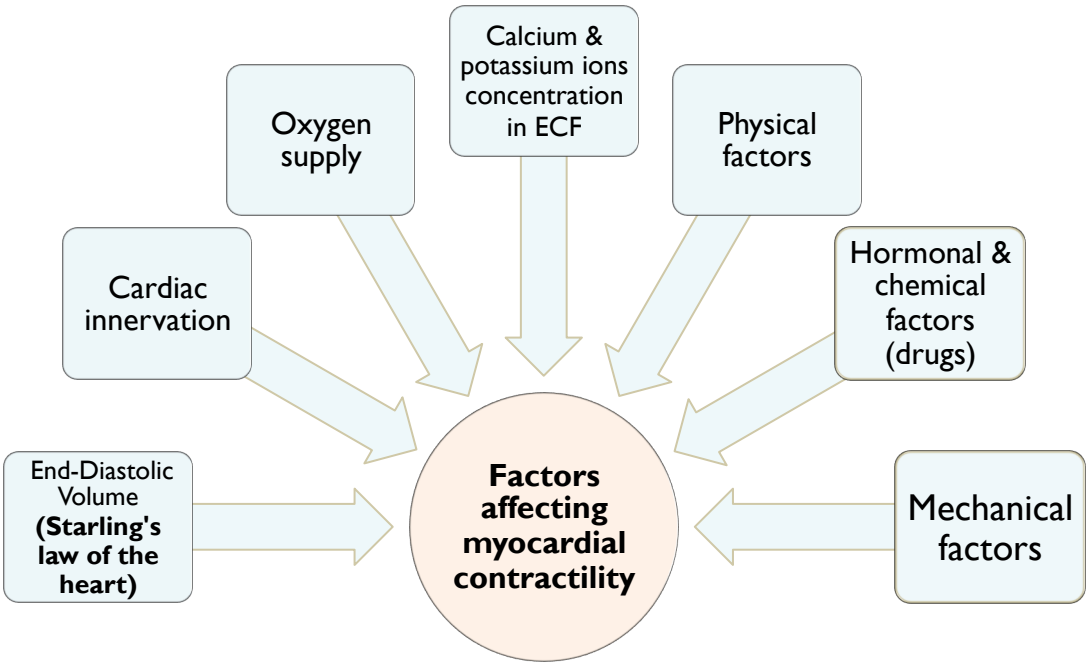
- **EDV (End diastolic volume):**
  The volume of blood present in each ventricle at the end of ventricular diastole. It also called Preload .
  Value: 120-130 mL
- **ESV (End systolic volume):**
  The volume of blood present in each ventricle at the end of ventricular systole "After pumping the blood" Value: 50-60 mL
- **SV (Stroke volume):**
  The volume of blood ejected by each ventricle during each ventricular systole. Value: 70-80 mL\beat



The Frank–Starling Principle (Starling's law of the heart):

- It is based on the length-tension relationship within the ventricle.
- If EDV (preload) ↑ it follows that the ventricular fiber length is also ↑ resulting in an increased 'tension' of the muscle.
- Cardiac output is directly related to venous return.
- The most important determining factor is preload.
- The contraction has a direct proportion to SV, and therefore SV in response to changes in venous return is called the Frank-Starling mechanism (Starling's Law of the heart).





1- Starling's law of the heart:

- Within limits, the power of contraction is *directly proportional* to the initial length of The muscle fiber.
- Overstretching the fiber as in *heart failure* its power of contractility *decreases*.

2- Cardiac innervation:

- Sympathetic NS:

  ↑ Force of contraction.
- Parasympathetic NS (vagus)

  ↓ Atrial force of contraction with no significant effect on *Ventricular* contraction.

3- Oxygen supply:

Hypoxia → ↓ contractility.

4- Calcium and potassium ions concentration

- ↑ Ca^{2+} → ↑ contractility.
- ↑ K^{+} → ↓ contractility.

5- Physical factors:

- Warming → ↑ contractility.
- Cooling → ↓ contractility.

6- Hormonal & chemical factors (drugs):

Positive inotropics:	Negative inotropics:
• Adrenaline	• Acetylcholine
• Noradrenaline	• Acidosis
• Alkalosis	• Ether
• Digitalis	• Chloroform
• Ca^{2+}	• some bacterial toxins (diphtheria toxins).
• caffeine	• K^{+}

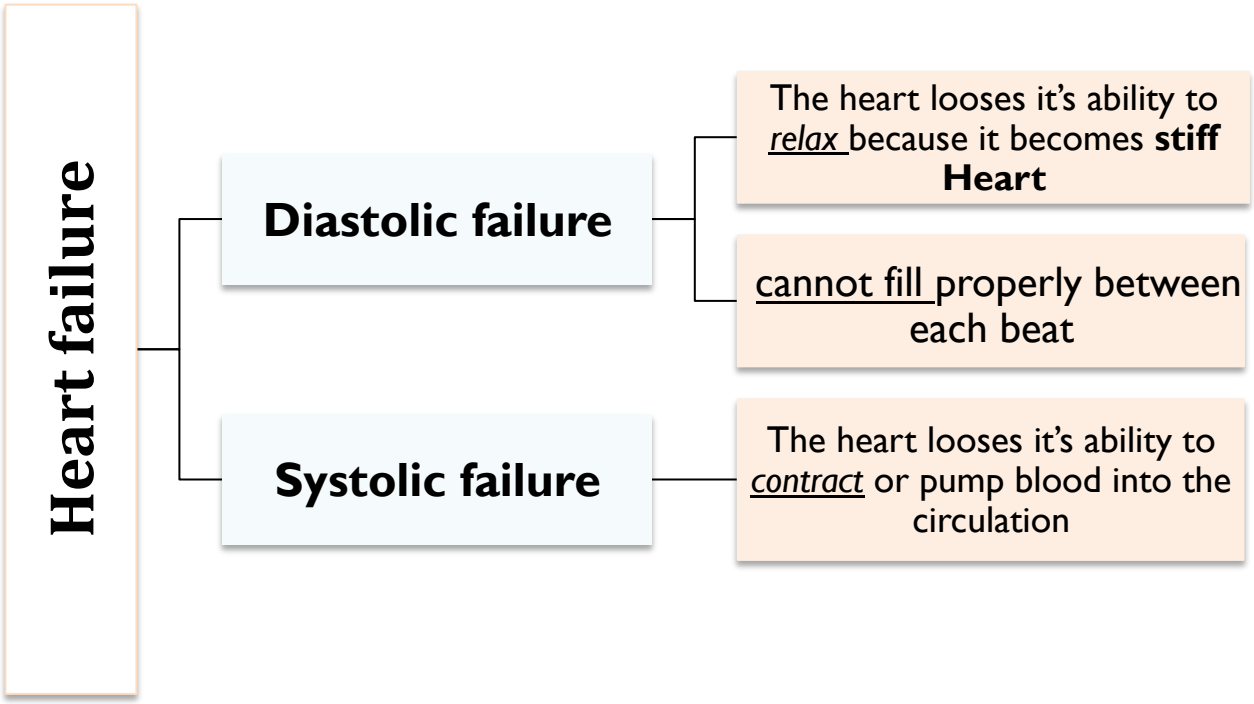
7- Mechanical factors:

- Cardiac muscle obeys "all or none law":
  Minimal or threshold stimuli lead to *maximal* cardiac contraction, because cardiac muscle behaves as a syncytium.
- Cardiac muscle cannot be stimulated while it is contracted, because its excitability during contraction is *zero* due to *long* absolute refractory period, so it *cannot be tetanized.*
- Cardiac muscle can perform both *isometric & isotonic* types of contractions.

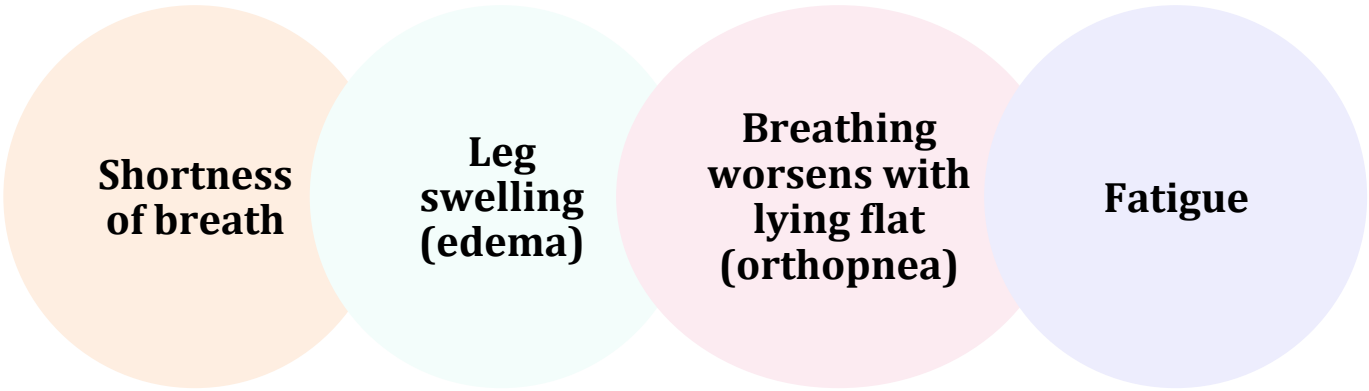
Heart Failure:

It is the pathophysiological process in which the heart as a pump is *unable* to meet the metabolic requirements of the tissue for oxygen and substrates *despite* the venous return to heart is either *normal* or *increased.*

- Occurs when either side of the heart cannot keep up with the flow of blood.
- Can involve the *left* or *right* side of the heart or both BUT Usually the left side is affected first.



Symptoms of congestive heart failure:



Heart failure signs and symptoms:

	SYMPTOM	WHY?
Persistent Cough or Wheezing	Coughing that produces white or pink blood-tinged sputum	Fluid "backs up" in the lungs
Edema	- Swelling in feet, ankles, legs or abdomen - Weight gain	Decreased blood flow out of the weak heart Blood returning to the heart from the veins "backs up" causing fluid to build up in tissues
Tiredness, fatigue	- Constant tired feeling - Difficulty with everyday activities	Heart can't pump enough blood to meet needs of bodies tissues Body diverts blood away from less vital organs (muscles in limbs) and sends it to the heart and brain
Lack of appetite/ Nausea	Feeling of being full or sick to your stomach	The digestive system receives less blood causing problems with digestion

Left heart failure:

- **Systolic** and **diastolic** heart failure are treated with different types of medications. **Both** types can cause:
  - The 'backing-up' of blood back into the lungs. "Pulmonary Edema".
  - As fluid builds up in tissues throughout the body.

Pathogenesis

Increase Aortic pressure causes backward pressure travels to Left ventricle → Then Pulmonary Veins → finally goes to the lung → Sort of accumulation of the blood in the lung that can not go back to the heart, , Causing Pulmonary Edema present as area of consolidation on the X-ray imaging.

Signs and Symptoms:

- Dyspnea
- Orthopnea and paroxysmal nocturnal dyspnea
- Cheyne Stokes breathing (abnormal pattern of breathing which is deeper and faster than normal).
- Fatigue, Anxiety
- Rales (abnormal sound heard from unhealthy lung by stethoscope)
- Pallor, cyanosis
- Increased heart rate and blood pressure.

Right Heart Failure:

- Usually occurs as a result of left heart failure.
- Occasionally isolated right heart failure can occur due to lung disease or blood clots to the lung (pulmonary embolism).

Pathogenesis:

Increase Right Ventricle pressure causes backward pressure on Right Atria → Increasing right atrial pressure → Stagnation of the blood On the:

1- Superior Vena Cava → increased Jugular venous pressure
2- Inferior Vena Cava → Ascites (An accumulation of fluid in the peritoneal cavity –Abdomen-).

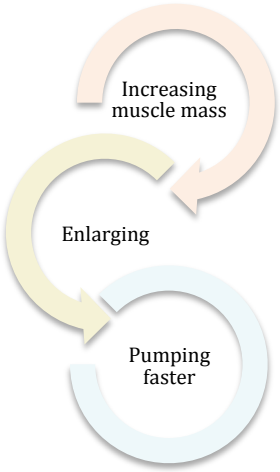
Signs and Symptoms:

- Fatigue
- Weakness
- Lethargy
- Weight gain, including abdominal girth
- Anorexia
- Elevated neck veins
- Hepatomegaly
- Pitting edema (present in the extremity)

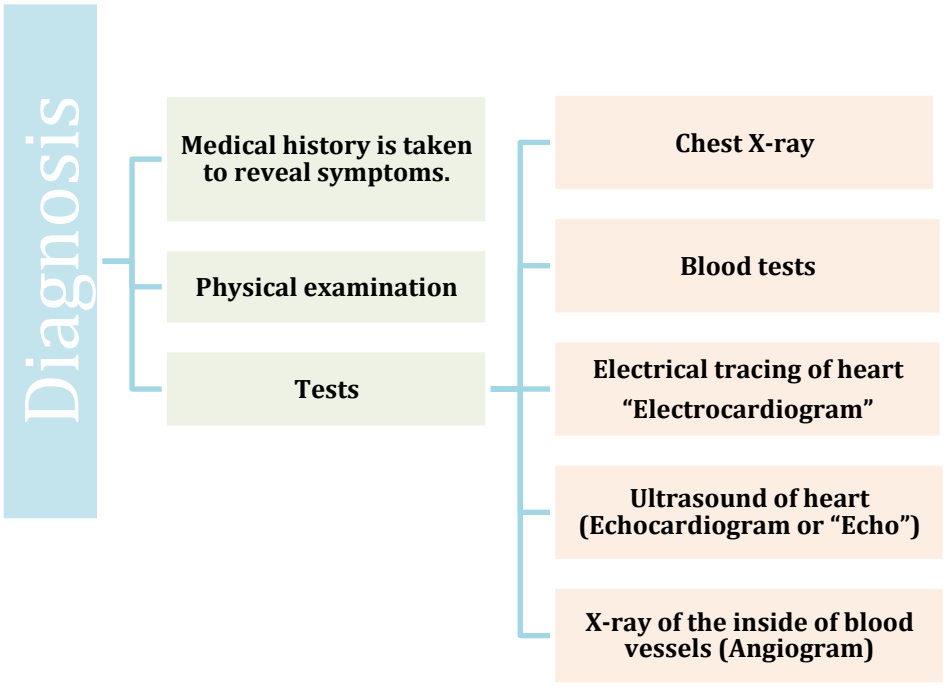
Causes of heart failure:

Acute non cardiac condition	Increased cardiac workload:	Impaired cardiac function:
• Volume overload • Fever • Hyperthyroidism • Infection	• Hypertension • Valvular disorders • Anemias • Congenital heart defects	• Coronary heart disease • Cardiomyopathies (muscle disease) • Rheumatic fever • Endocarditis

Compensating mechanism:



How is it diagnosed?



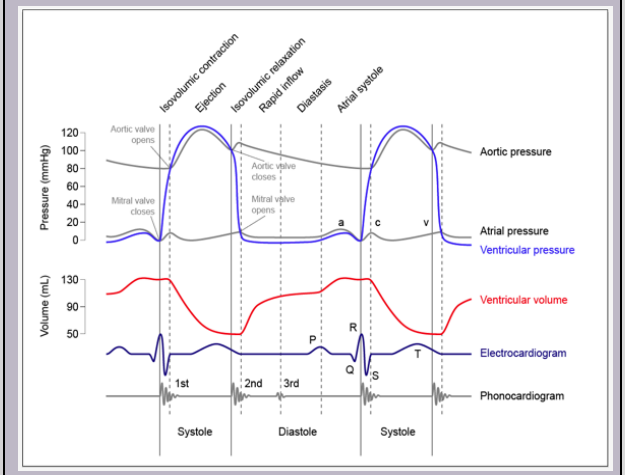
Indicator for diagnosing heart failure:

Ejection fraction (EF):
The percentage of blood that is pumped out of your heart during each beat.

How do we calculate EF?
It is the fraction of end-diastolic volume ejected during a heartbeat.

Ejection Fraction = Stroke volume / End diastolic volume

$$EF = \frac{SV}{EDV} = \frac{70\ ml}{130\ ml} = 0.54\ (no\ unit)$$

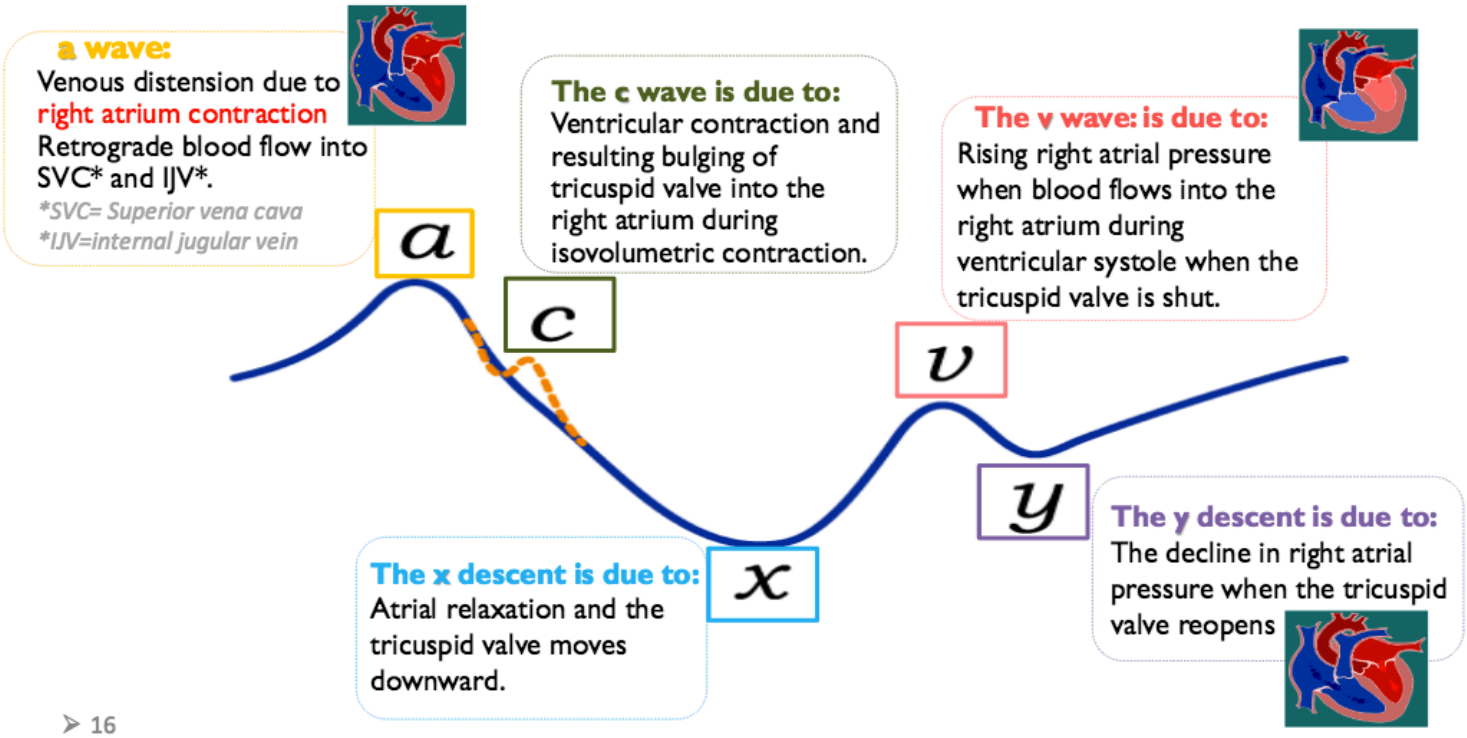
Heart sounds:	Normal				Abnormal "Murmurs"				
Divided into	S1	S2	S3	S4	Systolic PASS*		Diastolic PAID*	Continuous	
					Pulmonary + Aortic	Mitral +tricuspid	Pulmonary + Aortic	Mitral + tricuspid	-
Caused by:	Closure of AV valves	Closure of Semilunar valve	Rush of blood	Contraction of atrial muscle	Stenosis	insufficiency	insufficiency	stenosis	ventricular septal defect Patent Ductus Arteriosus
Characters	LUB 50-60Htz	DUB Physiological splitting 80-90Htz	Heard in children 20-30Htz	Heard in elderly < 20htz	Mid ejection between S1-S2	Pan, begins with S1 until S2	Early	Mid-late	Begins in systole, continue into diastole.
Best heard	AV VALVES area	SEMILUNAR VALVE area	Mitral area	Mitral area	**an easy mnemonic to remember abnormal heart sounds: PASS: Pulmonary Aortic Stenosis, Systolic. PAID: Pulmonary Aortic insufficiency, Diastolic And the opposite in the mitral and tricuspid valves.				
Relation to cardiac cycle									

Venous return:

What is it	is the quantity of blood flowing from large veins into the right atrium each minute
Factors controlling venous return	Pressure drop during inspiration → ↑venous return -Forceful expiration (Valsalva maneuver) → ↓venous return. ↑Blood volume → ↑venous return. ↑Pressure gradient → ↑venous return

jugular venous pulse

- The normal JVP reflects phasic pressure changes in the right atrium and consists of three positive waves and two negative descents.



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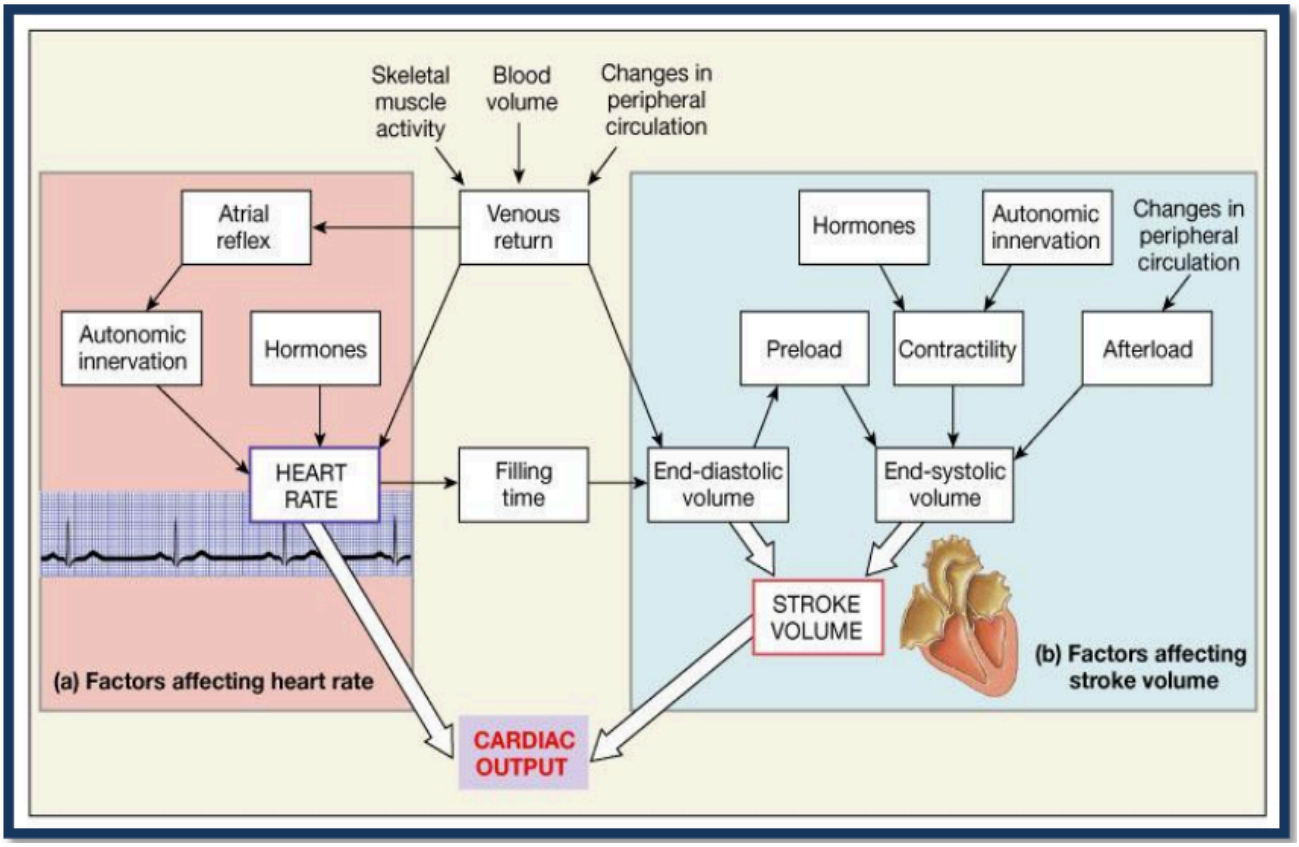
Causes of raised JVP			
1- increased right ventricular filling pressure. e.g.: in heart failure, fluid overload.	**2-Obstruction of blood flow from right atrium to right ventricle.** e.g.: tricuspid stenosis.	**3-superior vena caval obstruction.** e.g.: retrosternal thyroid goitre.	**4- positive intrathoracic pressure.** e.g.: pleural effusion, pneumothorax.

What is Cardiac output	Cardiac output is the volume of blood ejected from the right or left ventricle per minute. (C.O. = Heart Rate x Stroke Volume)
Values	Normal H.R.= 72 beats\min S.V.= 70ml\min C.O.=5l\min



PHYSIOLOGY TEAM 435

Measurement Of C.O

The Direct Fick's Method:

It states that, the amount or volume of any substance taken up by an organ or by the whole body is equal to:

(The arterial level of the substance — the venous level) X blood flow.

$$Blood\ flow = \frac{Amount}{(Arterial\ level - Venous\ level)}$$

Thus :

$$\left[\{Arterial\ level\ of\ the\ substance - The\ venous\ level\} \times \{\frac{Amount\ of\ substance}{Artirial\ level\ Venous\ level}\}\right]$$

- **Guyton corner :**

In animal experiments, one can cannulate the aorta, pulmonary artery, or great veins entering the heart and measure the cardiac output using a flowmeter. An electromagnetic or ultrasonic flowmeter can also be placed on the aorta or pulmonary artery to measure cardiac output.
In humans, except in rare instances, cardiac output is measured by indirect methods that do not require surgery. Two of the methods that have been used for experimental studies are the *oxygen Fick method* and the *indicator dilution method.*

Arterial blood pressure:

Definition	is the force exerted by blood against a vessel wall.
In normal	Arterial blood pressure is not constant, it raises during ventricular systole & falls during ventricular diastole. 120/80.
Greatest drop in pressure	Arterioles
Systolic BP	The peak (highest) blood pressure, it is measured during ventricular systole, it is 120 mmHg in a young person at rest.
Diastolic BP	The minimum blood pressure, it is measured at the end of ventricular Diastole, it is 80 mmHg in a young person at rest.
Pulse pressure	It is the difference between systolic BP and diastolic BP. Pulse pressure = systolic BP - diastolic BP
Mean BP	Mean BP = diastolic pressure + 1/3 (systolic pressure – diastolic pressure), depends on 2 factors: { Mean BP= C.O. x total peripheral resistance }
Hemodynamics	It is the branch of physiology concerned with The physical principles governing : Pressure, Flow, Resistance, Volume, and Compliance
Resistance Vessels	Arterioles & small arteries
Compliance	Venous system has a large compliance & acts as a blood reservoir (high volume & low pressure)

Types of blood flow	1- Laminar (Stream-lined) flow	2-Turbulent flow
-	Smooth flow at a steady rate. The central portion of blood stays in the center of the vessel → Less friction.	High flow rate Blood flow in all directions (Mixing), leading to increase in resistance (by narrowing in vessel wall) → slow the flow rate. Fluid passes a constriction, sharp turn, rough surface.

Types of blood flow	1- Laminar (Stream-lined) flow	2-Turbulent flow
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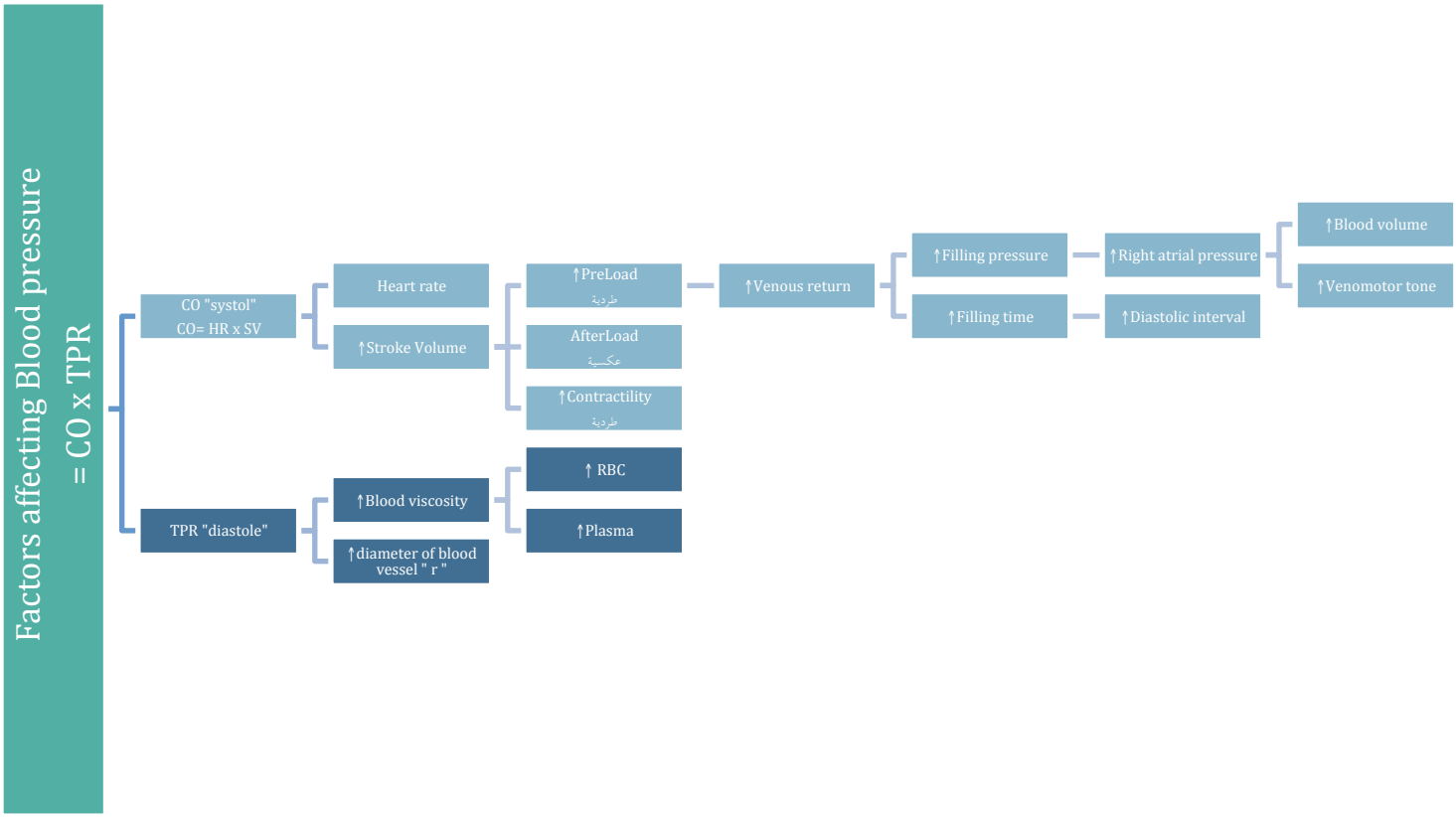
Elastic rebound	During systole:	During diastole :
maintains blood flow in arteries when ventricle diastole.	arterial BP and the volume of blood will ↑ so the artery walls expand to handled the extra amount of blood pumped by the ventricles.	the arterial BP falls and the Volume of blood will ↓ so the arteries wall will recoil to their original dimensions

Blood flow = CO	Viscosity	Resistance	Compliance	TPR	
Amount of blood moving through a vessel in a given time period. $Q = \frac{\Delta P}{R}$	$V = \frac{Q}{A}$ When Cross sectional area increase: ⇓ Diameter of vessel ⇓ viscosity of blood flow	tendency of vascular system to oppose flow. Poiseuille's Law = $R = \frac{8\eta L}{\pi r^4}$ $Q = \frac{\Delta P \pi r^4}{8\eta L}$ Resistance ⇑ Radius ⇓ Radius⇑ blood flow ⇑	the volume of blood that the vessel can hold at a given pressure. $C = \frac{V}{P}$	Systemic Circulation: $TPR = \frac{AP - RAP}{Flow}$ AP=Aortic pressure RAP= right atrial pressure	Pulmonary Circulation: $Pul.R = \frac{Pul.Art.P - LAP}{flow}$ Pul.art.p= pulmonary artery pressure LAP= left atrial pressure



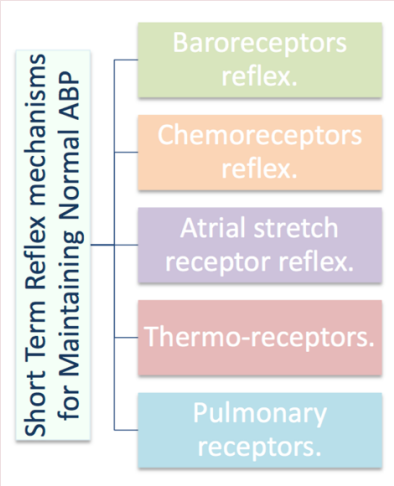
Circulatory shock types								
Divided into	Low output shock ⇓CO					High to normal output shock ⇓PR		
Ty	Cardiogenic	Hypovolemic	Obstructive			Distributive Neural	Distributive Vasogenic	
							Anaphylactic	septic	Psychogenic
Due to	-Failure of myocardial pump -loss of > 40% LV function	(loss of 15-25% \ 1-2L). ⇓ venous return	Reduced by vascular obstruction			- Generalized peripheral vasodilation - Loss or drop in vasomotor (vascular) tone / spinal cord injury	Histamine triggers peripheral vasodilation and increase capillary permeability	Bacterial endotoxin	Decrease HR & vessels dilate
Causes:	-Myocardial infarction -Cardiomyopathy -Cardiac tamponade - Acute valvular dysfunction -Sepsis	-loss of blood -loss of fluid -loss of plasma	Obstruction of venous return: Vena Cava syndrome (usually neoplasms)	Compression of the heart: hemorrhagic pericarditis → cardiac tamponade.	Obstruction of the outflow of the heart: Aortic dissection, Massive pulmonary embolism., pneumothorax	- Acute spinal cord injury - Deep general anesthesia depresses the vasomotor center - Spinal anesthesia blocks the sympathetic nervous system - Brain damage	-allergic reaction - IgE- mediated hypersensitivity	peripheral vasodilatation and endothelial injury	stress, pain, or fright
symptoms	Congestion of lungs & viscera (Chest X-ray): -Interstitial pulmonary edema. -Alveolar edema. -Cardiomegaly	-hypotension -Tachycardia -Tachypnea -Cold, pale skin. -Oliguria -restlessness				- Behaves like hypovolemic shock - Blood volume remains normal - CO is severely reduced as blood is pooled in peripheral veins	-	flushed & warm due to Hyperdynamic state.	- Simple fainting (syncope) - Brain becomes hypoperfused - Loss of consciousness
others	High mortality rate	-				-	-	Hyperdynamic state	-

Pathophysiology of shock:

↓ capillary perfusion -> Inadequate tissue oxygen -> anaerobic metabolism -> Metabolic acidosis -> Release of free radicals & oxidative stress -> Tissue damage -> Apoptosis.

Stages of shock:

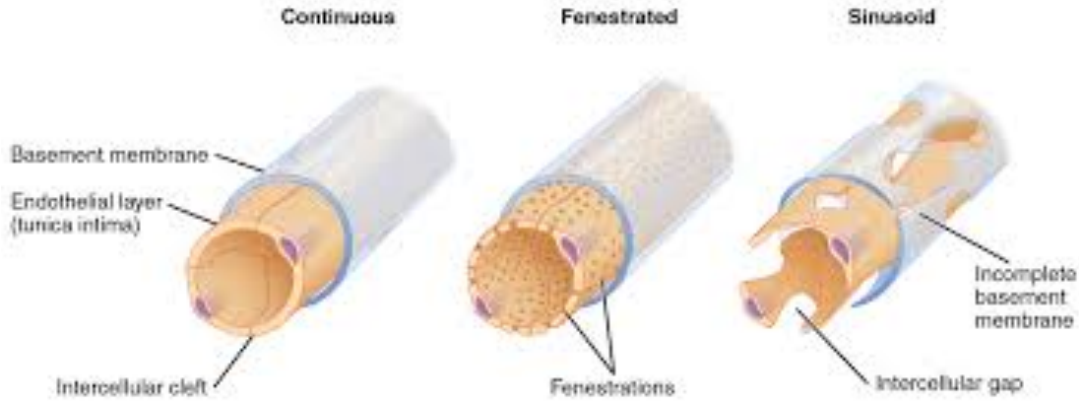
Reversible	Progressive	Irreversible
- Changes can be reversed by compensatory mechanism (neurohormonal activation) or by treatment - Defense mechanisms are successful in maintaining perfusion - Non-progressive 1- ↓ *BP*-> ↑ *sympathetic* 2- ↑ *sympathetic*: skin vasoconstriction, vasoconstriction to the kidney, Release of epinephrine and nor-epinephrine, increase heart rate and force of contraction 3- Hypoxia Vasoconstriction to the kidney: ⇓ urine output ⇑ renin secretion ⇑Angiotensin II vasoconstrictor ⇑ aldosterone ⇑ Na reabsorption and H2O retention	- Defense mechanisms begin to fall - Multi-organ failure - Loss of more than 30% of blood volume - The deterioration of the CVS 1- Leads to myocardial ischemia, and a decrease in cardiac output → More decrease in Blood pressure below 60 2- Decrease in blood volume → decrease in blood velocity → increase Viscosity. Platelets aggregate → Clot formation → Obstruction 3- caused by lysosome rupture and decrease activity of mitochondria → decrease active transport and general metabolism 4- Leads to acidosis	- Complete failure of compensatory mechanisms - Heart deteriorates until it can no longer pump blood - can lead to death Possible Mechanism in development irreversible Shock: 1- Shock Stimulus 2- Lysosome Activation , Release Proteases 3- Splitting of Plasma Proteins 4- Vasoactive Peptides, Amines, etc. 5- Hypotension, Fluid Loss 6-

Regulation of blood pressure		
Rapidly acting control mechanism short	Long term regulation	Intermediate Mechanisms Regulating Arterial Blood Pressure
acts within. seconds / minutes. Concerned by regulating Cardiac output & Peripheral resistance. Reflex mechanisms that act through autonomic nervous system: Centers in medulla oblongata: • Vasomotor Center (VMC) ... Sympathetic nervous system. • Cardiac Inhibitory Center (CIC) .. Parasympathetic nervous system	• Hormonally mediated. • Takes few hours to begin showing significant response. [slow response (Long-term). • Concerned in regulating blood volume.	Intermediate Mechanisms Regulating Arterial Blood Pressure: 1. Renin-angiotensin vasoconstrictor mechanism. 2. Stress-relaxation of the vasculature. 3. Fluid Shift mechanism. During this time, the nervous mechanisms usually become less and less effective. The mechanisms are activated within 30 minute to several hours.
	1- Renin-Angiotensin-Aldosterone System. 2- Vasopressin [Anti-diuretic hormone (ADH)] Mechanism 3- Atrial Natriuretic Peptide Mechanism (Low-pressure volume receptors.) 4- EPO (erythropoietin.)	1. The Renin-Angiotensin System 2. Fluid Shift Mechanism 3. stress relaxation mechanism

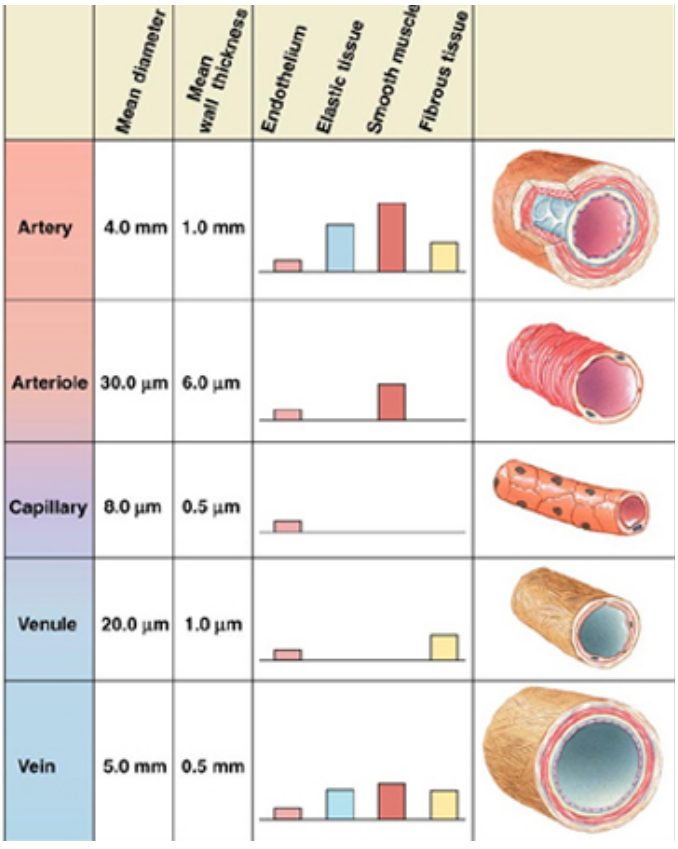
HIGH BP	LOW BP
1- ↓ Antidiuretic hormone (ADH) secretion. 2- ↓ Angiotensin II hormone secretion 3- ↓ Aldosterone hormone secretion 4- ↓ Erythropoietin hormone secretion. 5- ↑ Atrial natriuretic peptides (ANP) hormone secretion.	1- ↑ Antidiuretic hormone (ADH) secretion. 2- ↑ Aldosterone hormone secretion 3- ↑ Angiotensin II hormone secretion . 4- ↑ Erythropoietin hormone secretion → ↑ RBCS,take few days. 5- ↓ Natriuretic peptides (ANP) hormone secretion

Capillary circulation:

Functions	Serve the requirements of the tissues
Circulation divided into	aorta “elastic recoil”, arteries “muscular+ low resistance vessels , arterioles “resistance vessels” , capillaries “exchange blood vessels” and venules &veins “capacitance vessels”.
Characteristic of capillaries	Most permeable- the larges cross Sectional area.
Types of capillaries	classified by diameter \ permeability : 1- Continuous : Do not have fenestrae. 2- Fenestrated: Have pores. 3- Sinusoidal: Large diameter with large fenestrae. “the most permeable”
The important processes that move Materials across capillary walls :	1-Diffusion 2- Filtration 3-Reabsorption



BLOOD VESSEL COMPARISON:



DISTRIBUTION OF BLOOD WITHIN THECIRCULATORY SYSTEM AT REST:

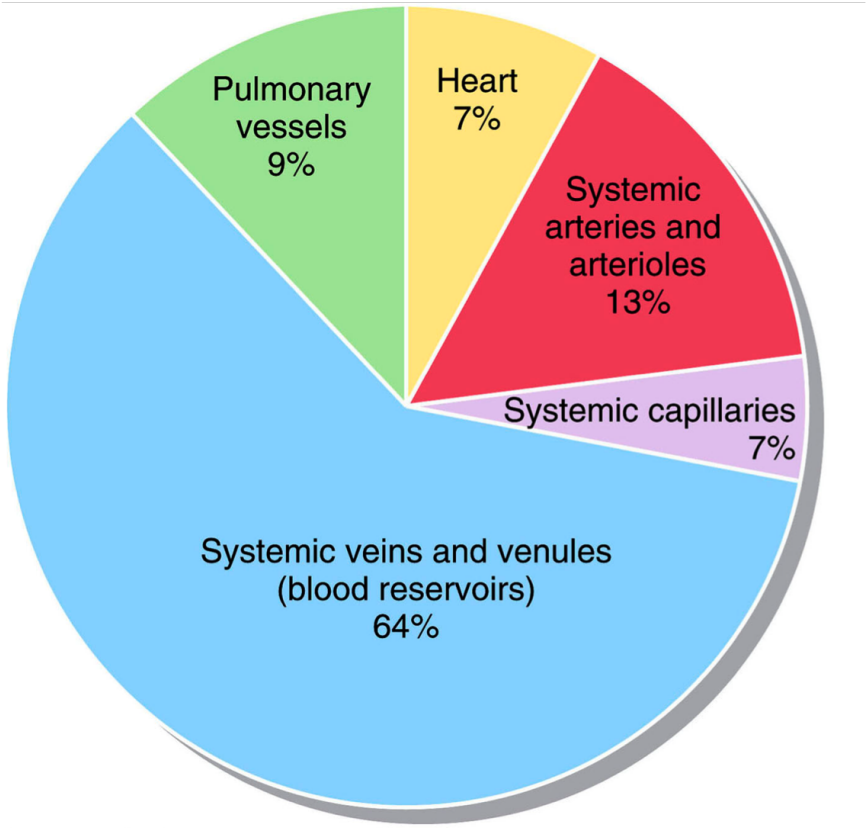
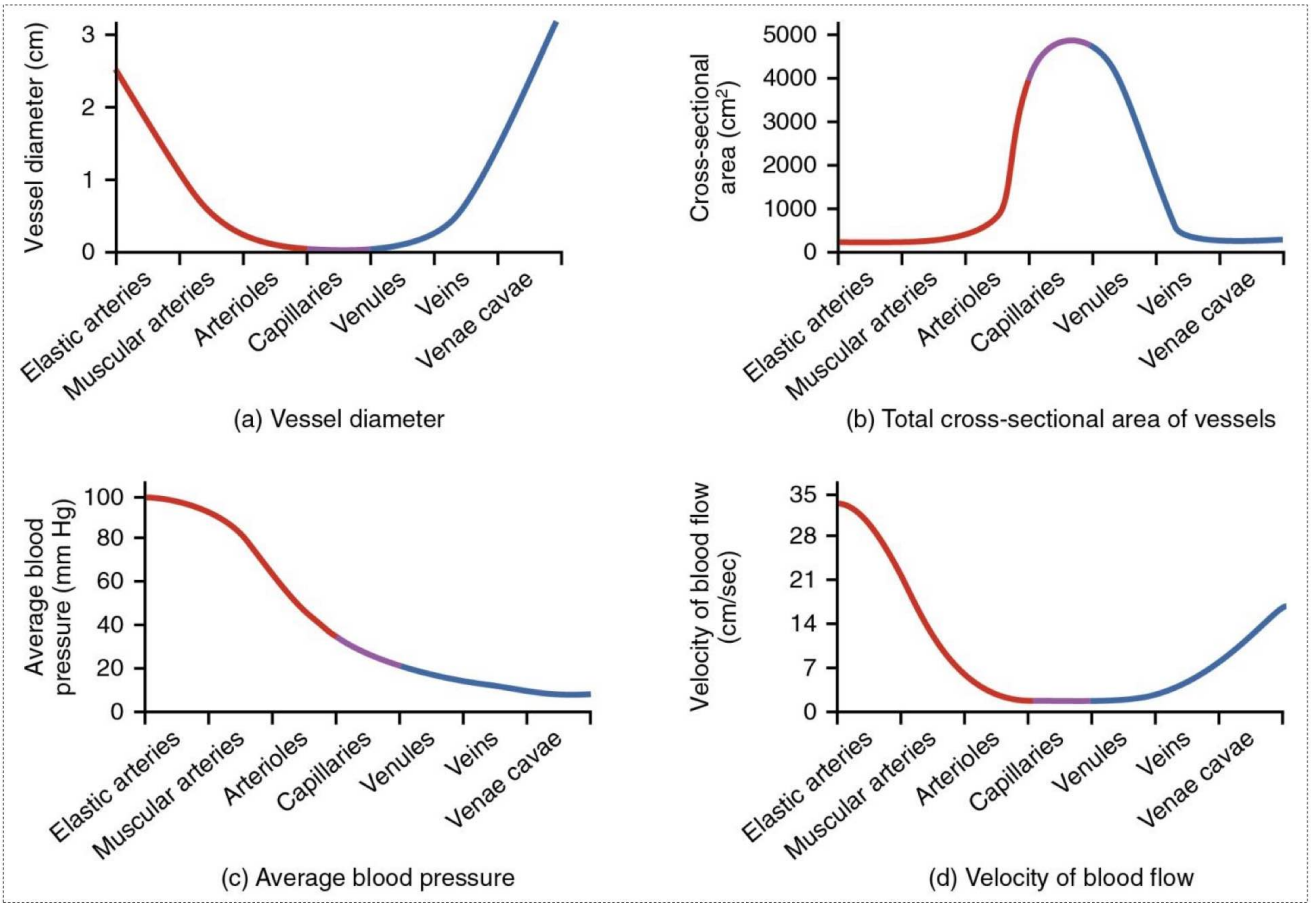


Figure 21.06 Tortora - PAP 12/e

Relationships among Vessel Diameter, Cross Sectional Area, Blood Pressure, and Blood Velocity:



Starling Forces

There are Four Forces that determine Fluid Movement Across the Capillary Membranes:

1. **Pc** (Capillary Pressure) → Tends to move fluid out of the capillary.
2. **Pi** (Interstitial Fluid Pressure) → Tends to move fluid into the capillary.
3. **Πc** (Plasma Colloid Osmotic Pressure) → Tends to cause Osmosis of fluid into capillary.
4. **Πi** (Interstitial fluid colloid osmotic pressure) → Tends to cause osmosis of fluid out of the capillary.

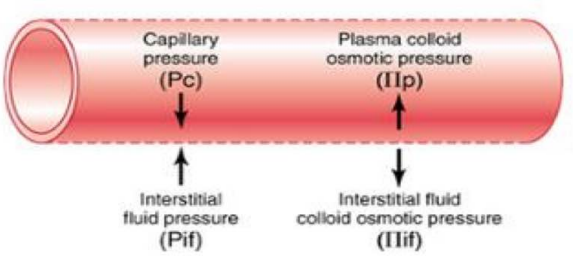
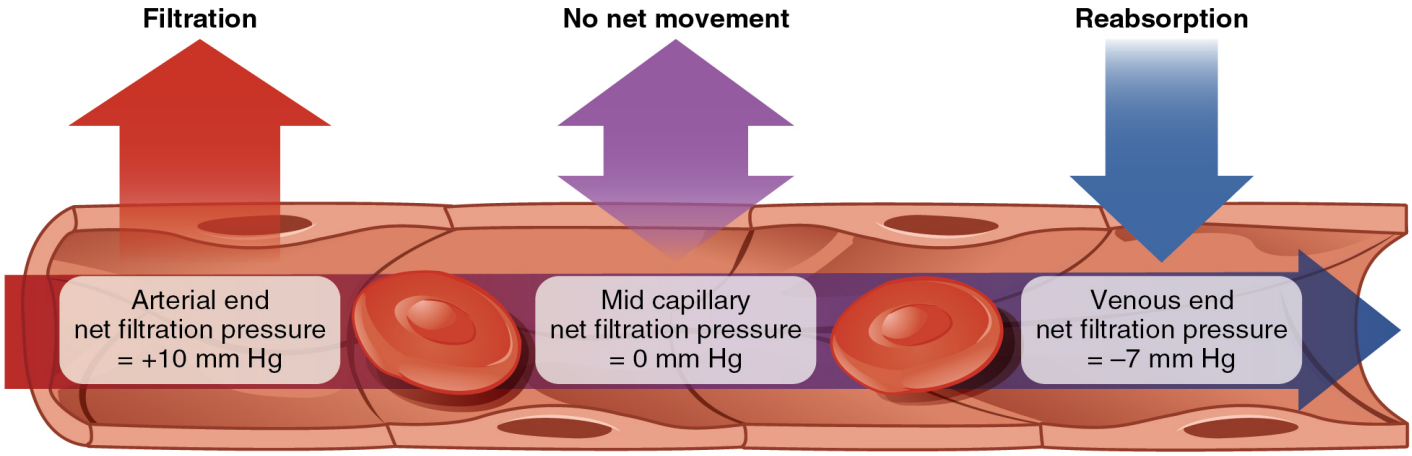


Figure 16-5 shows the four primary forces that determine whether fluid will move out of the blood into the interstitial fluid or in the opposite direction. These forces, called "Starling forces" in honor of the physiologist who first demonstrated their importance

- **Net Filtration Pressure** = P_{net} = [(Pc-Pi) – (Πc- Πi)]
- **Fluid movement** = Kf [(Pc – Pi) – (Πc - Πi)]

- **(Pc-Pi)** : Net hydrostatic pressure
- **(Πc- Πi):** Net osmotic pressure
- **Kf :** filtration coefficient



Fluid exits capillary since capillary hydrostatic pressure (35 mm Hg) is greater than blood colloidal osmotic pressure (25 mm Hg)

No net movement of fluid since capillary hydrostatic pressure (25 mm Hg) = blood colloidal osmotic pressure (25 mm Hg)

Fluid re-enters capillary since capillary hydrostatic pressure (18 mm Hg) is less than blood colloidal osmotic pressure (25 mm Hg)

At the arterial end of the capillary, capillary hydrostatic pressure (CHP) is greater than blood colloid osmotic pressure (BCOP), so fluid moves out of the capillary **(filtration).** "occurs More than reabsorption"

Near the venule, CHP is lower than BCOP,so fluid moves into the capillary **(Reabsorption).**

What is edema?	Is an excessive amount of fluid in the interstitial Spaces .
Signs	Pitting edema of the feet , Elephantiasis (Lymphaticfilariasis)
Causes	1-Increased hydrostatic blood pressure. 2-Decreased blood osmotic pressure. 3- Increased interstitial hydrostatic pressure (lymphatic capillary blockage). 4-Leaking capillary wall.

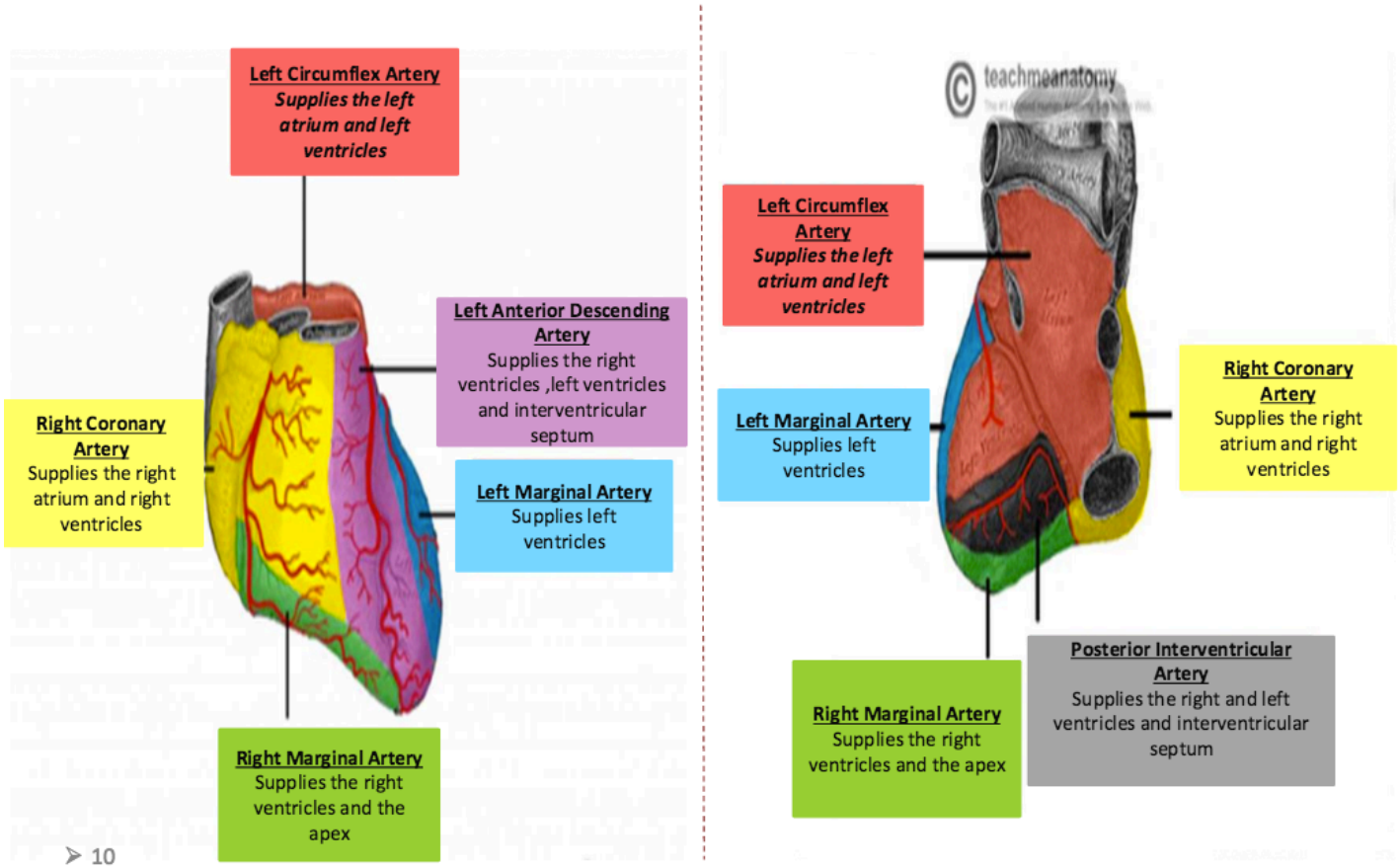
Location of lymphatic vessels:
Lymphatic vessels present between capillaries.
Function (Aim):
1- Lymphatic system is responsible for bringing the interstitial fluid to vascular compartment. 2- Drain excess interstitial (tissue) fluid back to the blood in order to maintain original blood volume. 3- Transports absorbed fat from small intestine to the blood. 4- Helps provide immunological defenses against pathogens.
Value:
Normal 24 hours lymph flow is → 2- 4 L
Mechanism:
Lymphatic capillaries lie in interstitial fluid close to vascular capillaries ,these capillaries merge into large lymphatic vessels & eventually into largest vessel, thoracic duct *which empties into large veins .*

coronary circulation:

	Left coronary artery (LCA)	**Right coronary artery (RCA)**
Size	larger	smaller
origin*	**Left posterior aortic sinus**	**Anterior aortic sinus**
Termination	By anastomosing with the right coronary artery	By anastomosing with the left coronary artery
Branches	• Left Anterior Descending (LAD) (= Anterior interventricular) • Marginal artery • Circumflex artery (CX)	• Posterior Descending Branch (= posterior interventricular) • Marginal artery

PHYSIOLOGY TEAM 435

Areas of Distribution of Coronary Arteries



Coronary dominance depends on which artery (or arteries) gives rise to the posterior descending artery (PDA) :
Right dominance(80-85% of cases) or left dominance or co-dominance.

Coronary Blood Flow (CBF) at rest	225-250 mL/min (5% of cardiac output.) Heart extracts 60-70%of O2
Phasic changes in CBF during systole & diastole	During Systole: Coronary arteries are compressed,so Blood flow to the left ventricle is reduced. During Diastole: Blood flows to the subendocardial portion of the left ventricle occurs only during diastole, and is not there during systole.
Factors Affecting Coronary Blood Flow	* Chemical factor: Lack of oxygen. Increased local concentration of Co2. Increased local concentration of H+ ion. Increased local concentration of k + ion. Increased local concentration of Lactate ,Prostaglandin ,Adenosine, Adenine nucleotides. * Neural factors: Sympathetic stimulation, Parasympathetic stimulation.

Short summary of rules:

- $EF = \frac{SV\ (EDV-ESV\ )}{EDV} \times 100$
- **SV = EDV – ESV**
- **EDV = ESV + SV**
- **Rate=** divide **300** by the number of boxes between each QRS
- $EF = \frac{SV}{EDV}$
- $Q = \frac{\Delta P}{R}$
- $V = \frac{Q}{A}$
- $R = \frac{8\eta L}{\pi r^4}$
- $Q = \frac{\Delta P \pi r^4}{8\eta L}$
- $C = \frac{V}{P}$
- $TPR = \frac{AP-RAP}{Flow}$
- $Pul.R = \frac{Pul.Art.P-LAP}{flow}$

- **Net Filtration Pressure =** P_{net} = [(Pc-Pi) – (Πc- Πi)]
- **Fluid movement =** Kf [(Pc – Pi) – (Πc - Πi)]

A huge thanks to our physiology team!
Done by: Nouf Altwaijri, Muneera AlOmari & Razan Alsabti
Good Luck.