CVS

PBL

Modified NO: 4

كتابة: صهيب زعيتر، محمود جرادات

تدقيق: خديجة ناصر

الدكتور: د قيس البلبيسي

Color code
Slides
Doctor
Additional info
Important

"You can't become a good clinician if you don't have a very strong basics"

The doctor's advice.

• Cardiovascular Medicine

• *from Basic to Clinical*

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Cardiovascular Medicine from Basic to Clinical

- Coronary Artery Disease
- Arrhythmias
- Valvular Heart Disease
- Heart Failure

In this lecture we will cover 4 cases.

Let's begin

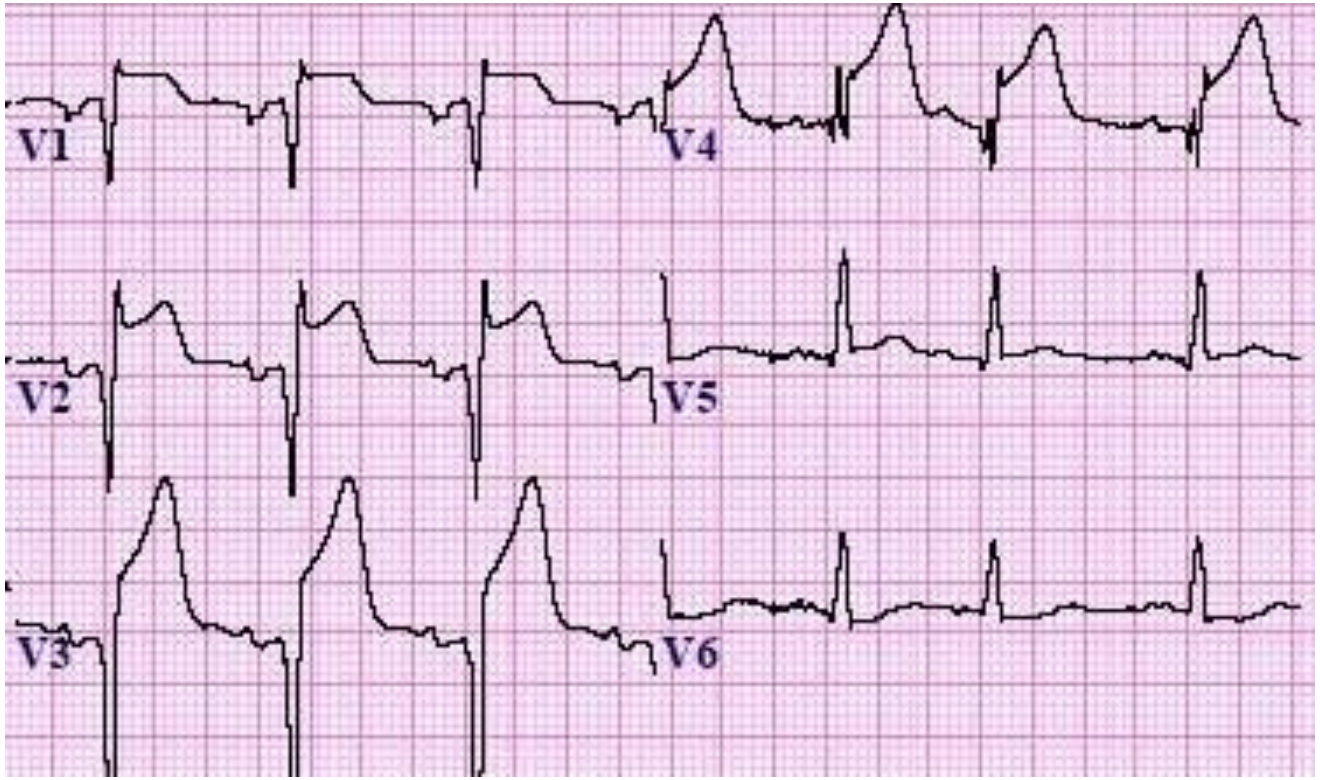
Case 1

Coronary Artery Disease

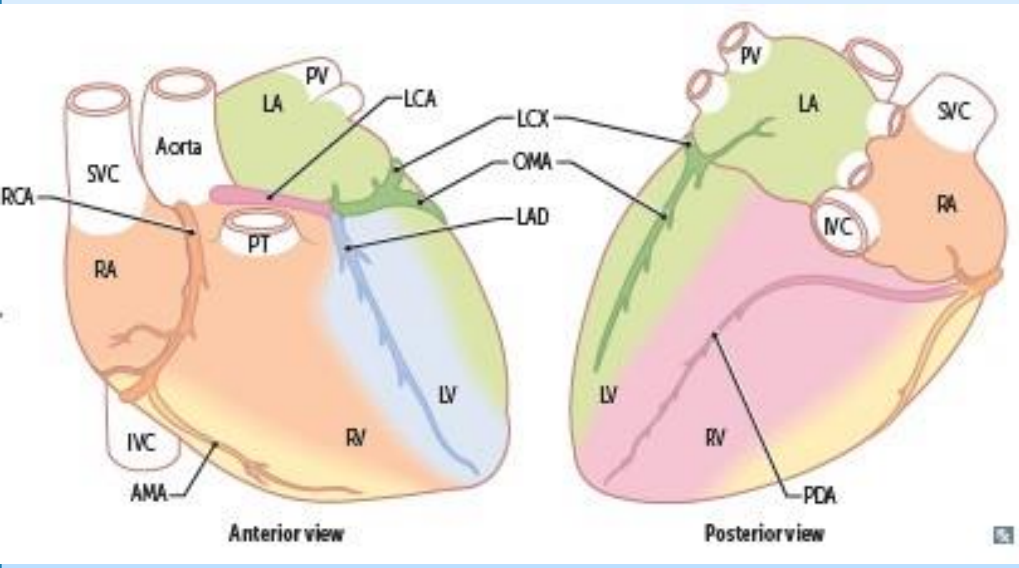
Case 1

The patient is a 65-year-old male, known case of Hypertension, Diabetes, Dyslipidemia, came to ED (emergency department) complaining of **Chest Pain** of 6 hours duration.

This is a very typical day to day case that clinicians get pretty much on daily basis. It is clear that the ECG is abnormal.



Basic – Anatomy & Physiology



You need first to know the anatomy in order to understand the pathophysiology & from where the ECG comes.

Anatomy:

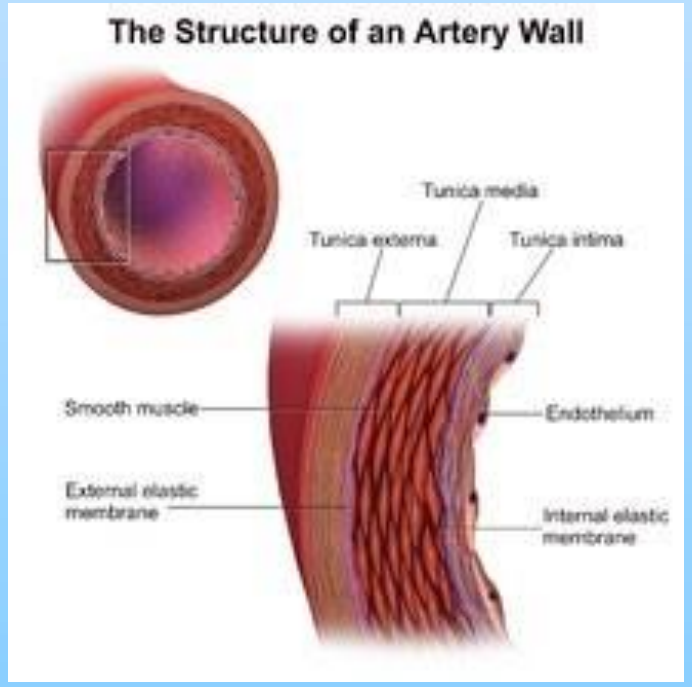
There are certain arteries (left anterior descending artery that supplies the front of the heart, the circumflex that goes to to left and the right coronary artery that goes to the right side of the heart)

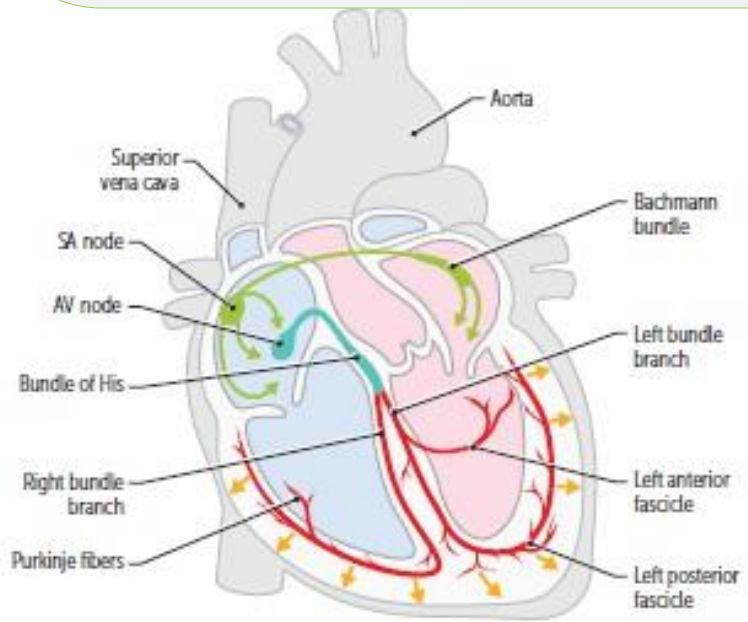
Histology:

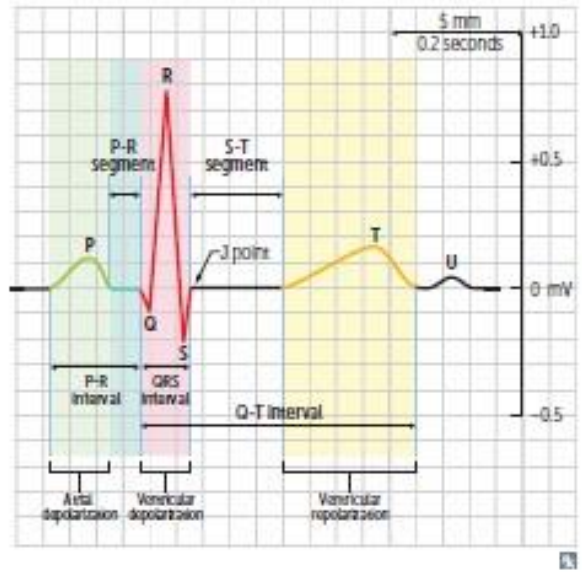
The artery is composed of three layers:

Tunica intima, Tunica media and Tunica adventitia.

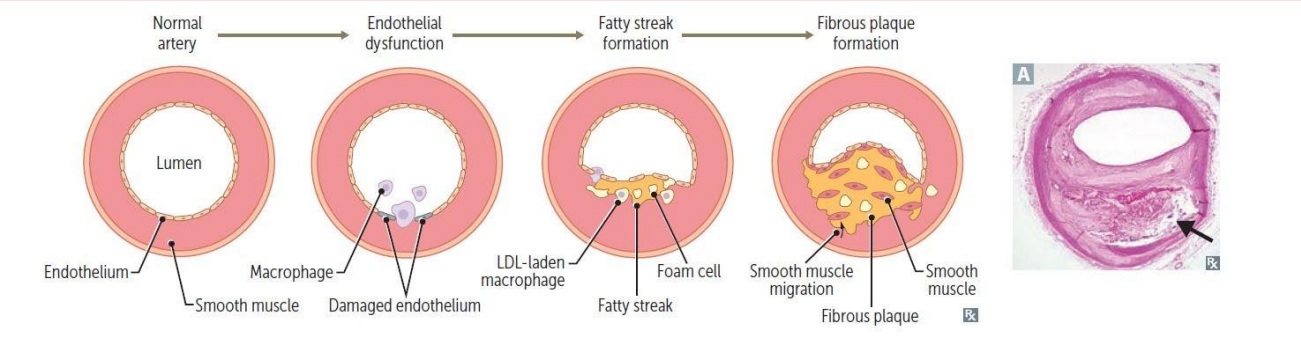
We also should know about atherosclerosis and how it develops.

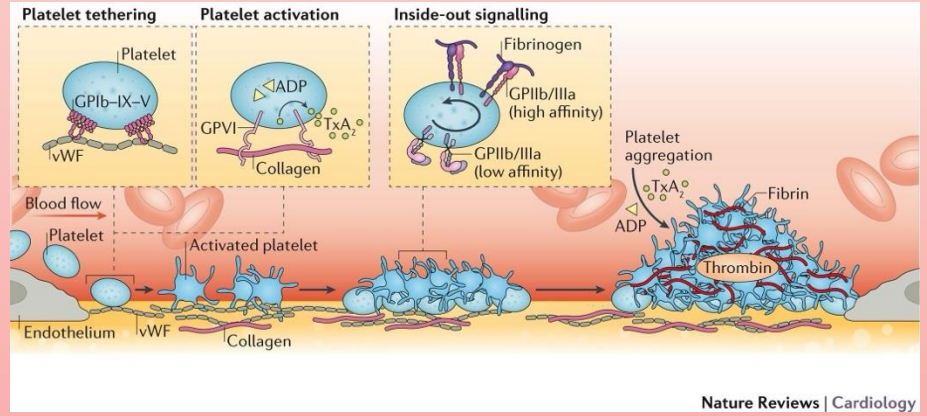


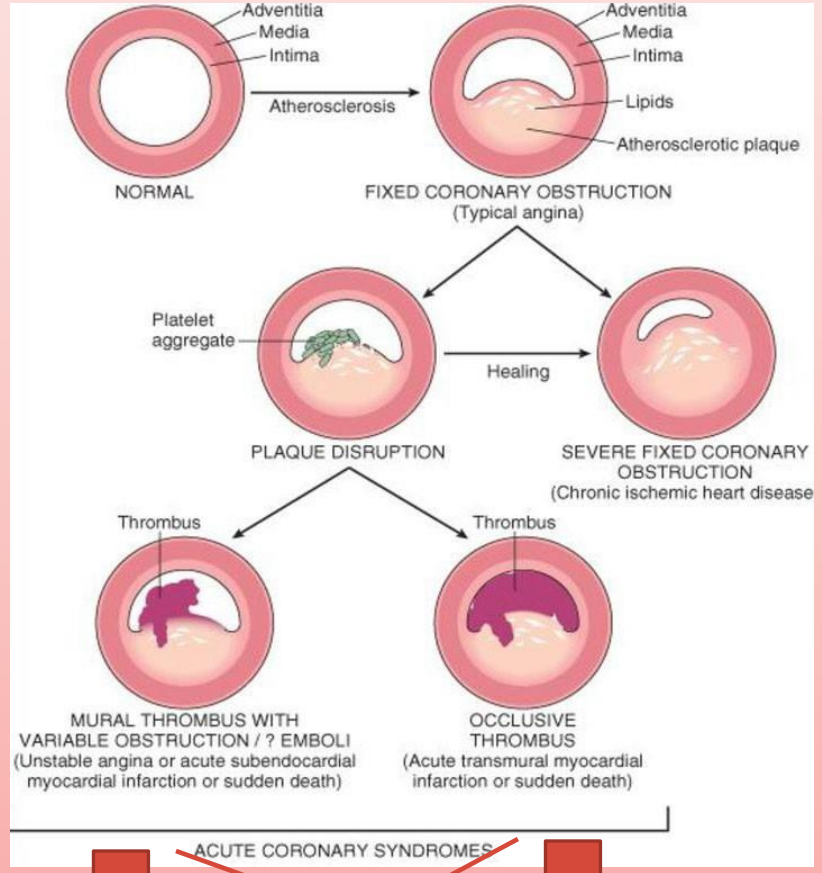




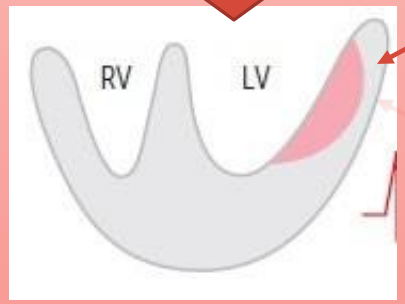
Basic – Pathology

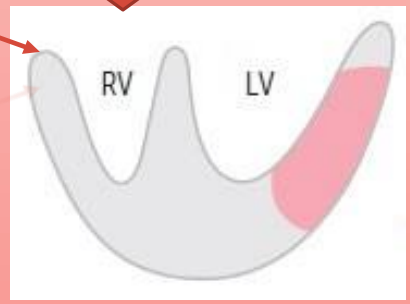






The pathophysiology:
(Explained in the next slide😁)

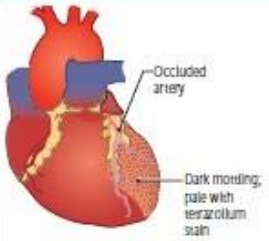
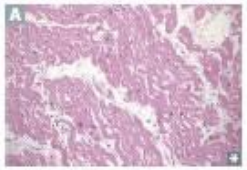
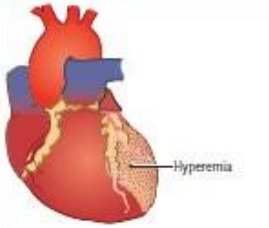
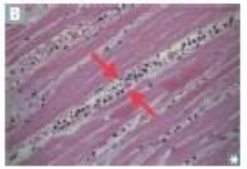
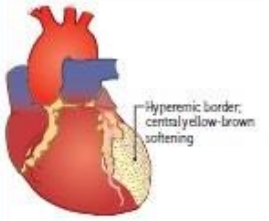
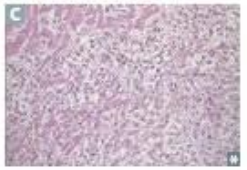
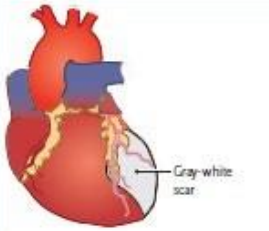
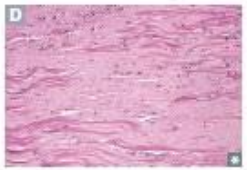


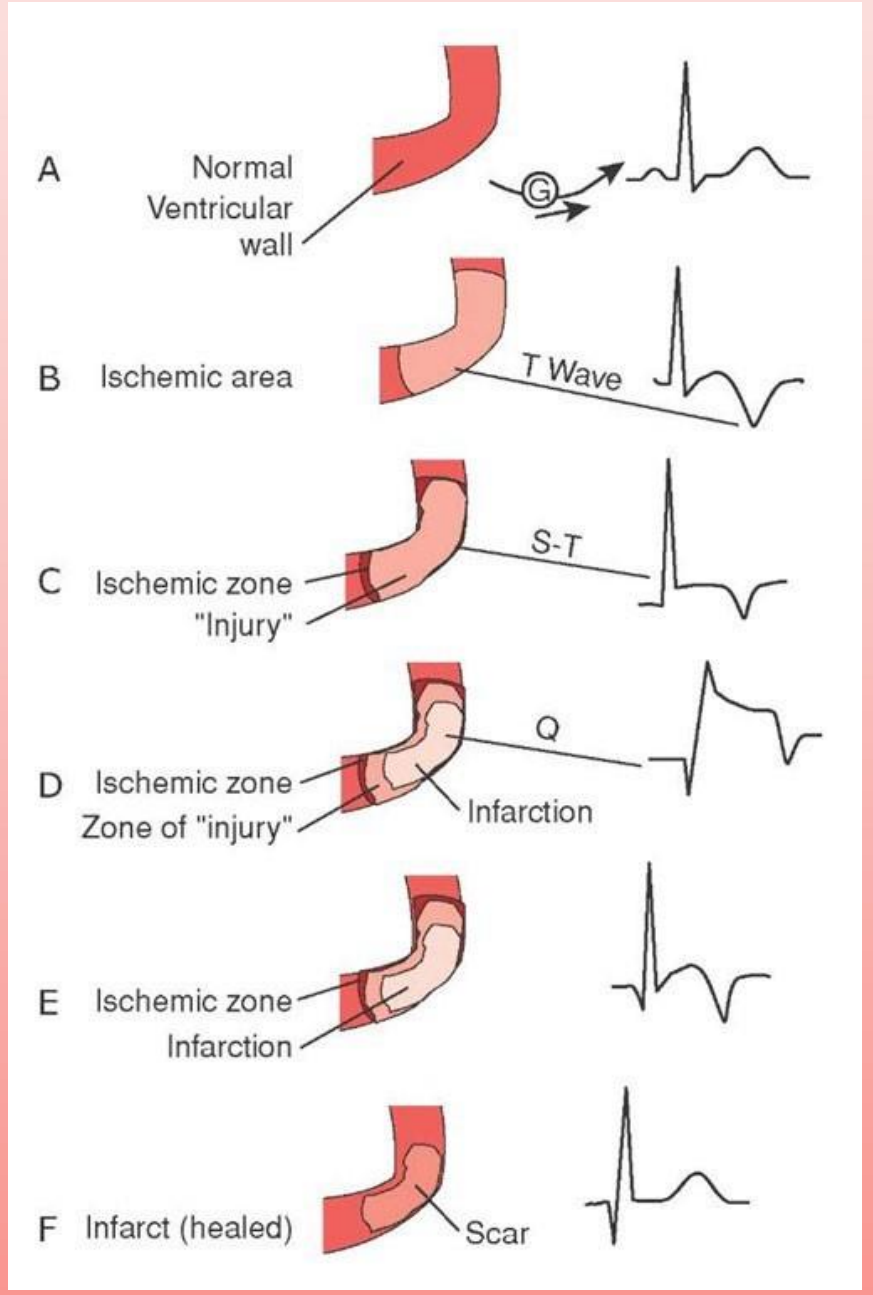


Coronary artery disease (CAD) begins with endothelial injury, followed by cholesterol accumulation, ultimately leading to acute coronary syndrome (ACS).
(Extra: CAD is the underlying chronic disease, while ACS refers to acute events resulting from complications of CAD).
Most heart attacks and acute coronary syndromes (ACS) occur with **non-obstructive lesions** (not severely narrowed) rather than the tight obstructive ones!!
But why?🤔
Because the non-obstructive lesions are simply **more common** and the underlying concept behind ACS is the **instability of the endothelium** (not the obstruction), where cracks expose accumulated materials to the bloodstream, triggering coagulation.
Even if the lesion is occluding only 10–20% of the BV lumen, it can result in a heart attack.

Therefore, the management for ACS depends on treating this pathophysiology and making the atherosclerosis more stable rather than stenting extensively (I am not gonna stent all the vessels!).

To summarise:
The hallmark of ACS is the exposure of cholesterol and inflammatory debris to the blood, leading to coagulation.
One of the cornerstones in treating this condition is anticoagulantion (anticoagulant drugs are commonly used).

TIME	GROSS	LIGHT MICROSCOPE	COMPLICATIONS
0–24 hours		Wavy fibers (0–4 hr), early coagulative necrosis (4–24 hr) A → cell content released into blood; edema, hemorrhage Reperfusion injury → free radicals and ↑ Ca^{2+} influx → hypercontraction of myofibrils (dark eosinophilic stripes) 	Ventricular arrhythmia, HF, cardiogenic shock
1–3 days		Extensive coagulative necrosis Tissue surrounding infarct shows acute inflammation with neutrophils B 	Postinfarction fibrinous pericarditis
3–14 days		Macrophages, then granulation tissue at margins C 	Free wall rupture → tamponade; papillary muscle rupture → mitral regurgitation; interventricular septal rupture due to macrophage-mediated structural degradation → left-to-right shunt LV pseudoaneurysm (risk of rupture)
2 weeks to several months		Contracted scar complete D 	Dressler syndrome, HF, arrhythmias, true ventricular aneurysm (risk of mural thrombus)

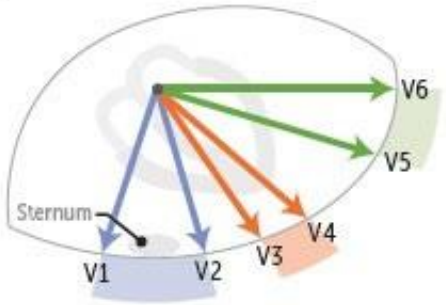


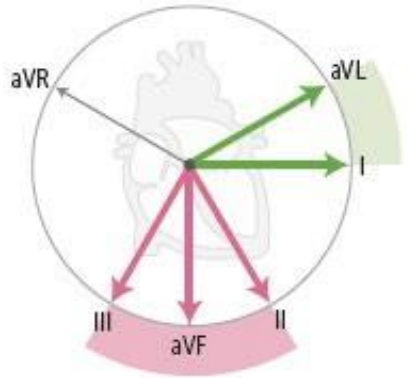
Pathology in the EKG changes:

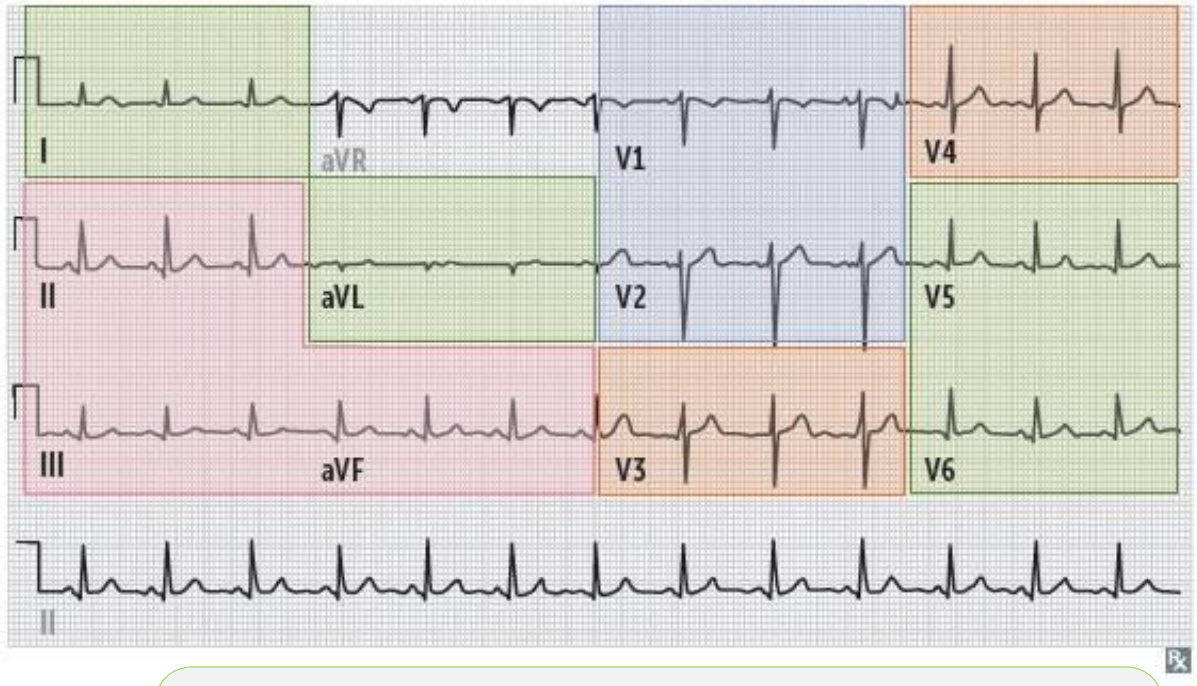
If there is a complete obstruction of a vessel, this will disrupt the repolarization, appearing as STEMI on an ECG.
(STEMI: ST-elevation myocardial infarction)

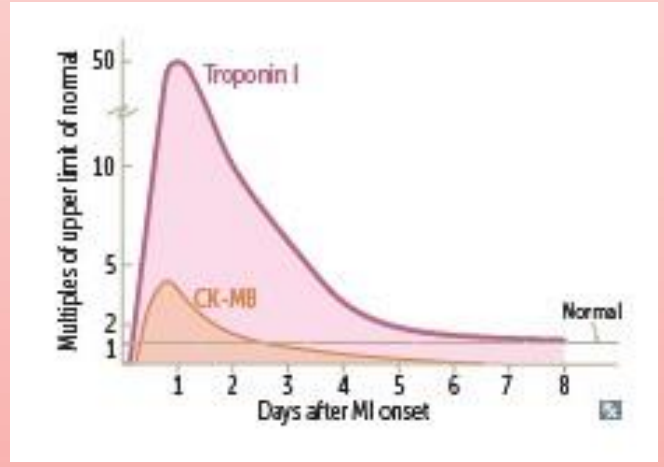
- Therefore ST elevation is an indication that that vessel is completely occluded and if this condition is not treated, the affected area will be lost.
- This makes STEMI an emergency and the area where it is elevated on ECG will tell you which anatomy structure was affected (look at the table in the next slide).

INFARCT LOCATION	LEADS WITH ST-SEGMENT ELEVATIONS OR Q WAVES
Anteroseptal (LAD)	V_1–V_2
Anteroapical (distal LAD)	V_3–V_4
Anterolateral (LAD or LCX)	V_5–V_6
Lateral (LCX)	I, aVL
InFerior (RCA)	II, III, aVF
Posterior (PDA)	V_7–V_9, ST depression in V_1–V_3 with tall R waves

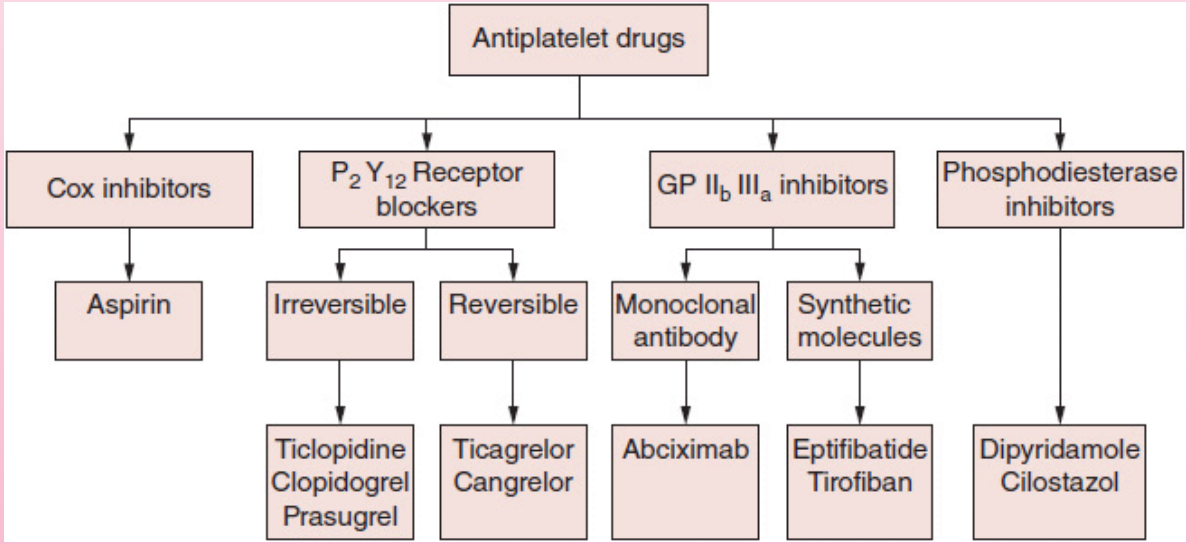


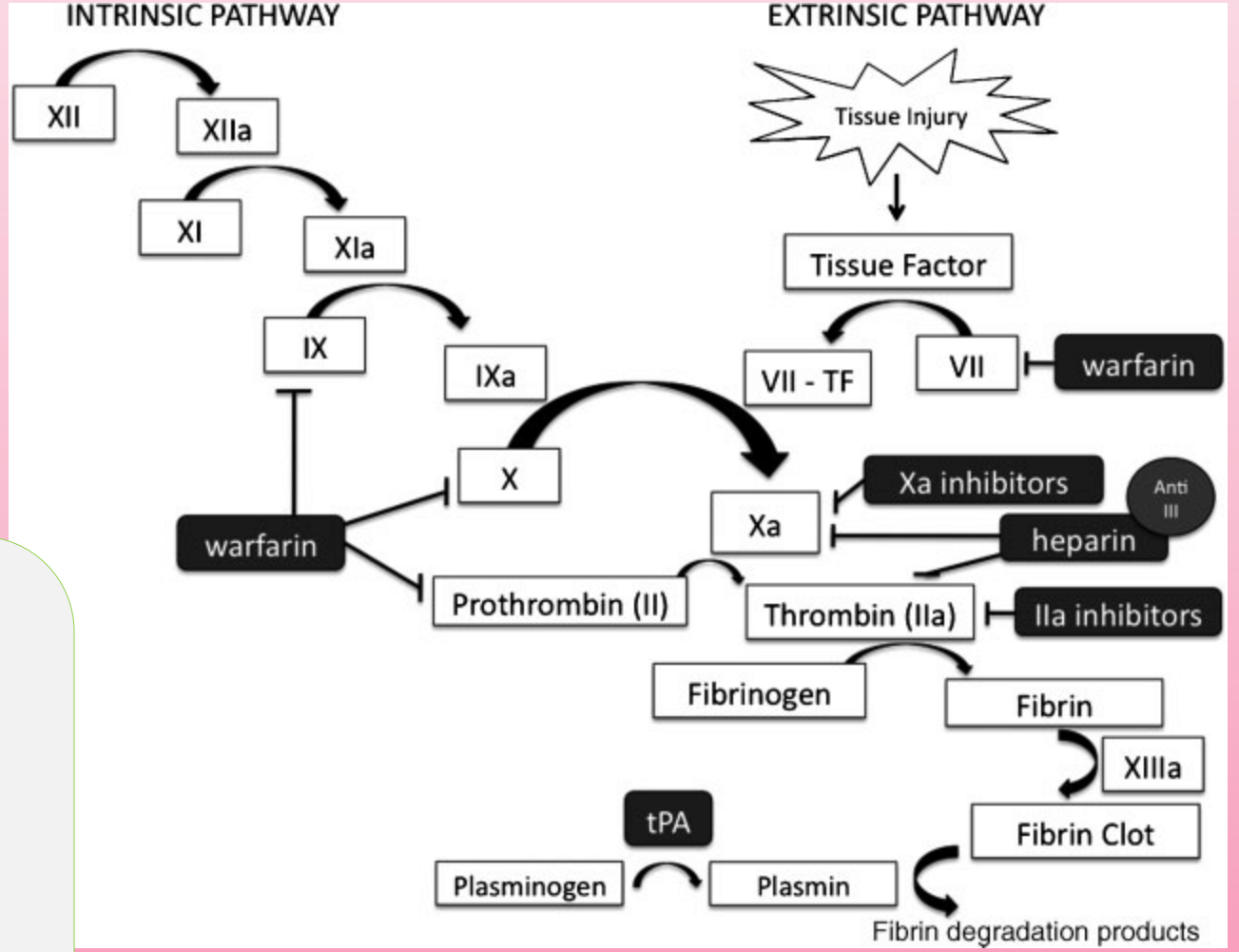






V1 is in front of the heart (anterior) which means LAD is the occluded vessel.
Look at the table above.





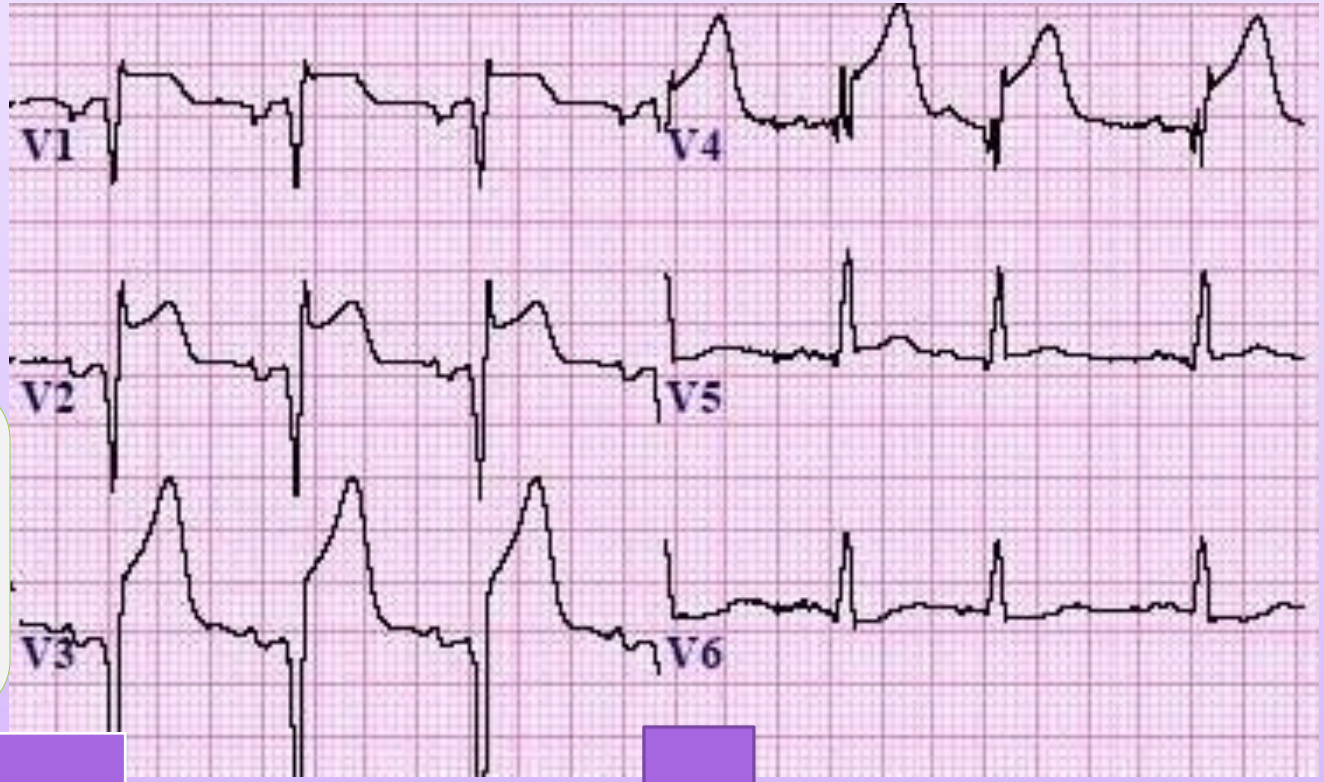
Pharmacology:

The hallmark of ACS is the obstruction caused by coagulation. Preventing further clot formation is a key to treatment.

This can be achieved by administering **anticoagulants** to prevent clot progression or **thrombolytics** to dissolve the clot.

Due to the urgency, the patient cannot wait for oral drug absorption, so these medications are given intravenously.

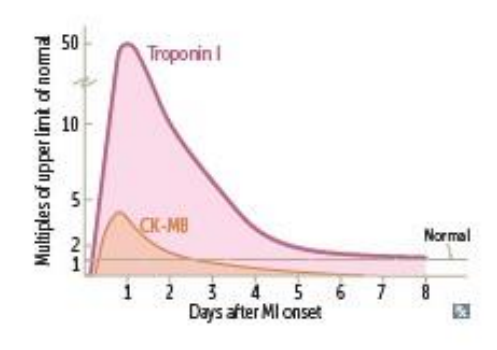
The patient is a 65-year-old male, known case of Hypertension, Diabetes, Dyslipidemia, came to ED complaining of **Chest Pain** of 6 hours duration.



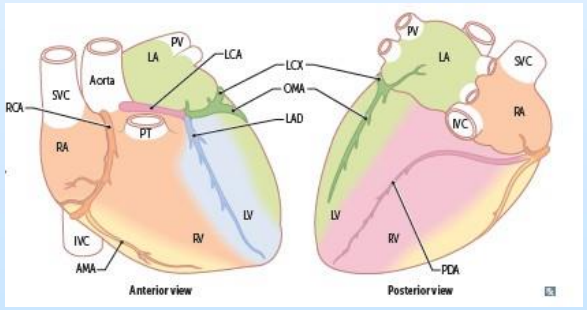
Antiplatelets to prevent formation of the clot
Nitrates to dilate the vessels
Beta blockers to decrease the demand.
(In addition to the previously mentioned anticoagulants and thrombolytics/fibrinolytics)

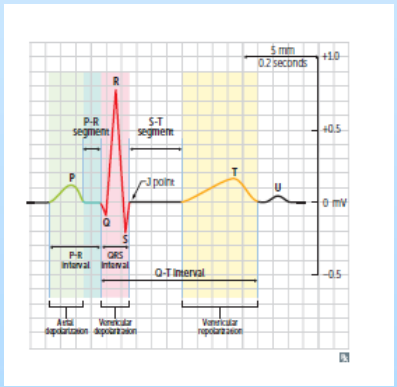
Treatment		
Antiplatelet	Aspirin	P2Y12 Inhibitor
Improve Flow	Nitrates	
Decrease Demand	B-Blockers	
Plaque Stabilization	B-blockers	Statins
Anticoagulant	Heparin	LMWH
Revascularization	Fibrinolytics	PCI

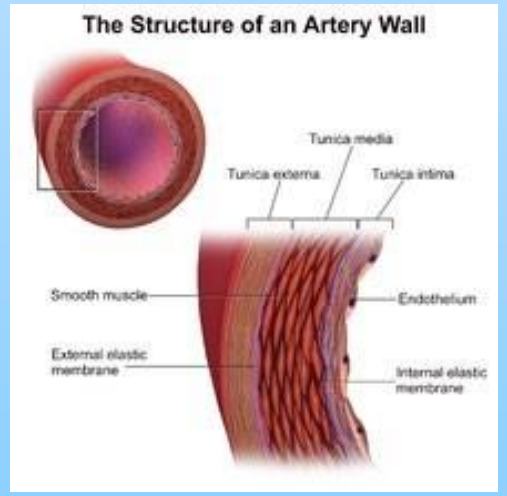
ST Elevation MI



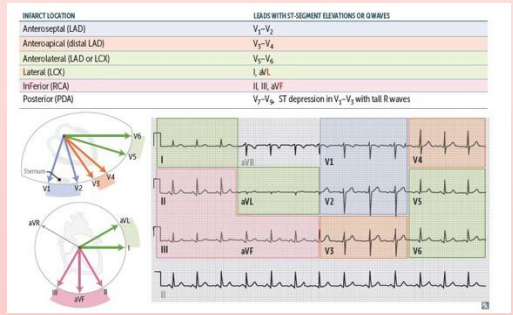
Basic - Physiology

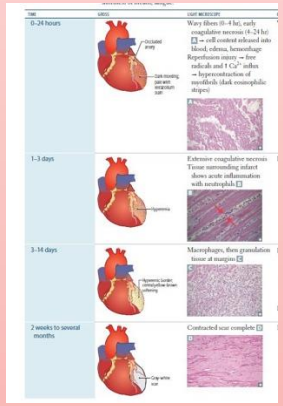


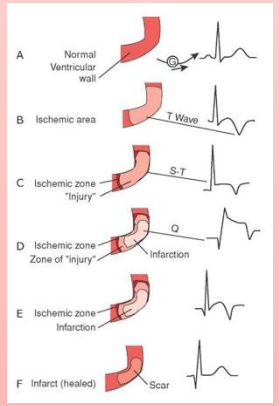


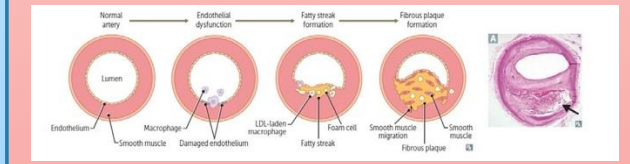


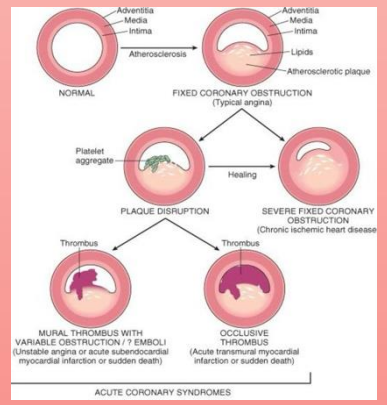
Basic - Pathology



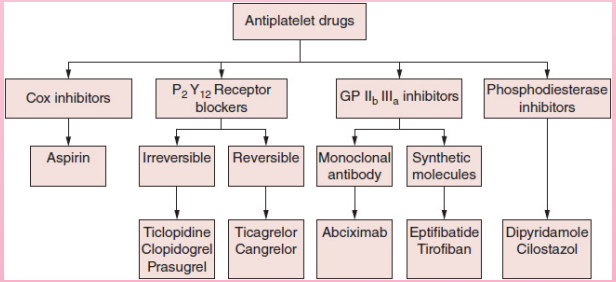


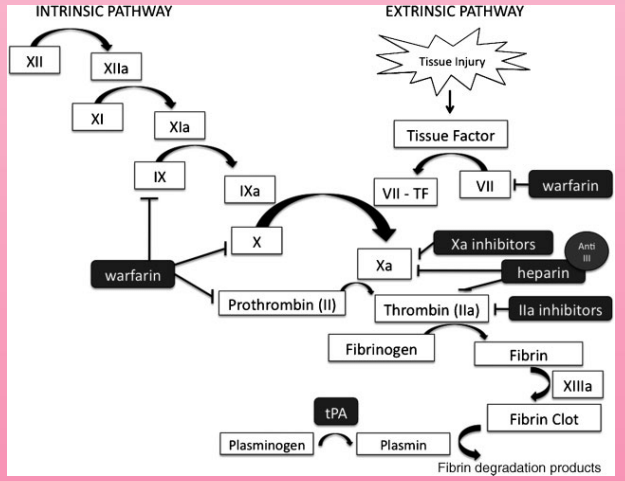






Basic - Pharmacology





Clinical

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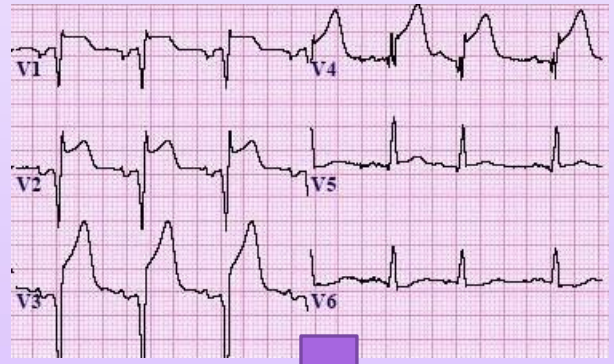
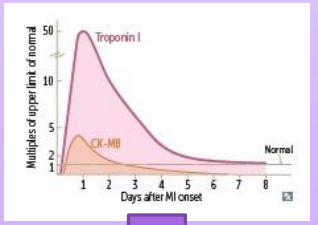


STEMI



Treatment		
Antiplatelet	Aspirin	P2Y12 Inhibitor
Improve Flow	Nitrates	
Decrease Demand	B-Blockers	
Plaque Stabilization	B-blockers	Statins
Anticoagulant	Heparin	LMWH
Revascularization	Fibrinolytics	PCI

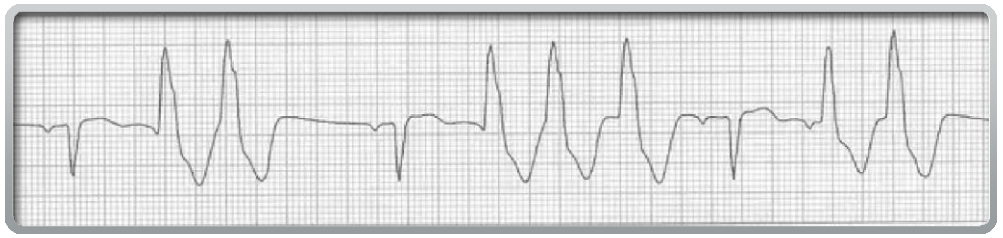
Case 2

Arrythmias ✨⚡

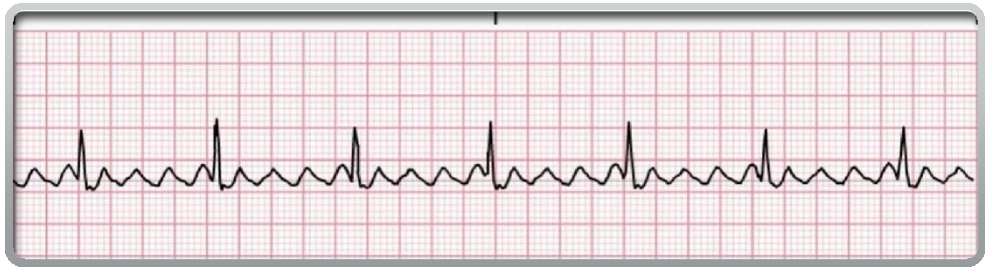
Case 2 Pure clinical

The patient is a 38-year-old female, recent history of Bronchitis treated with Azithromycin, heavy EtOH (alcohol) drinker, came to ED (emergency) complaining of **Palpitations** in last 2 days.

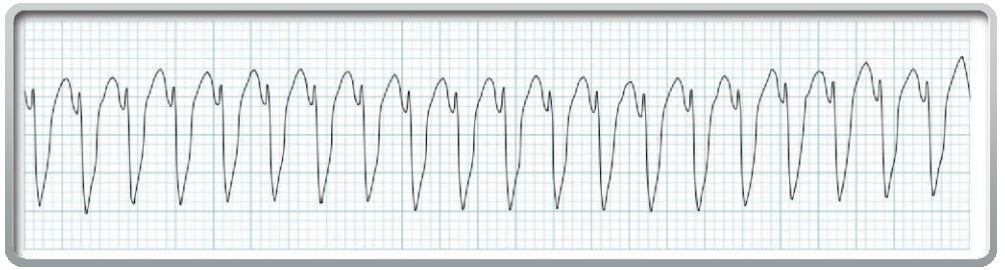
A.



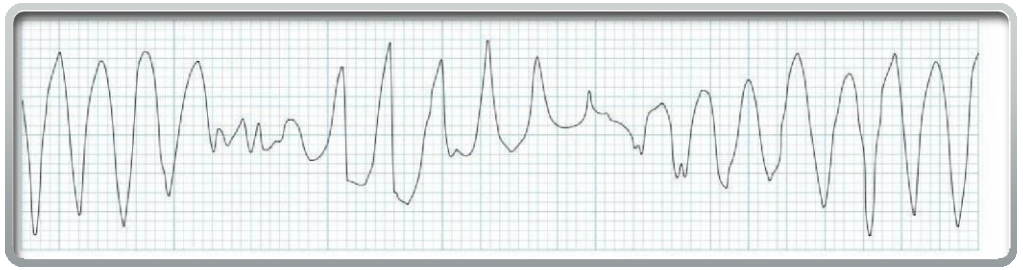
B.



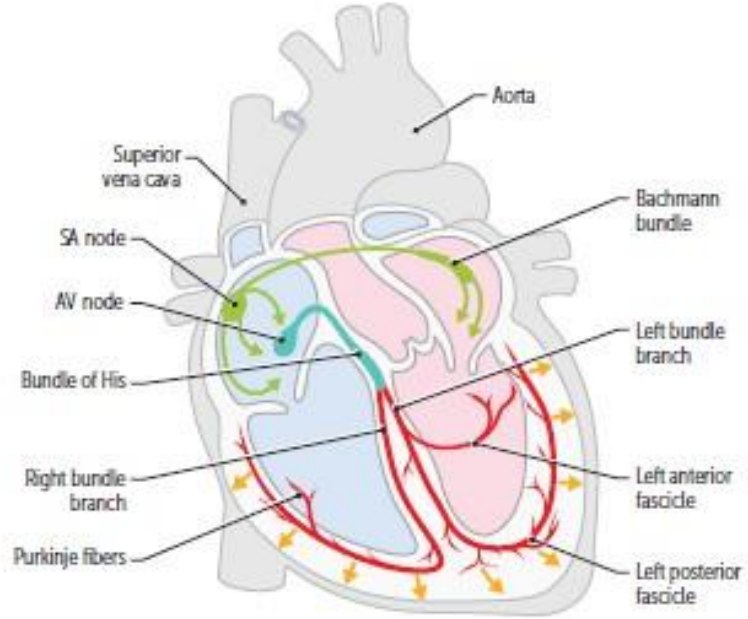
C.

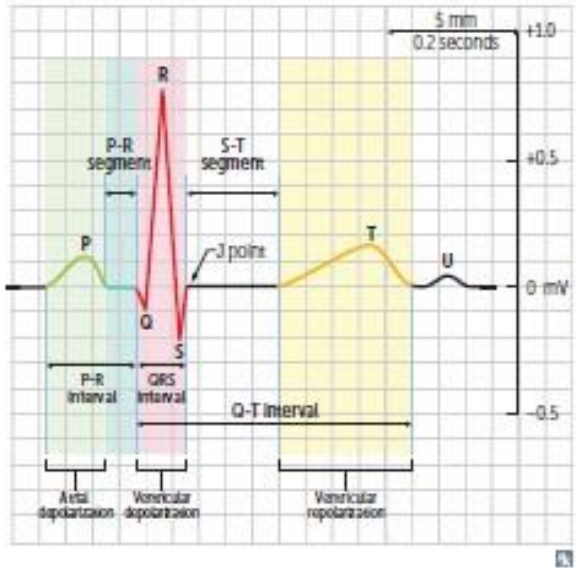


D.

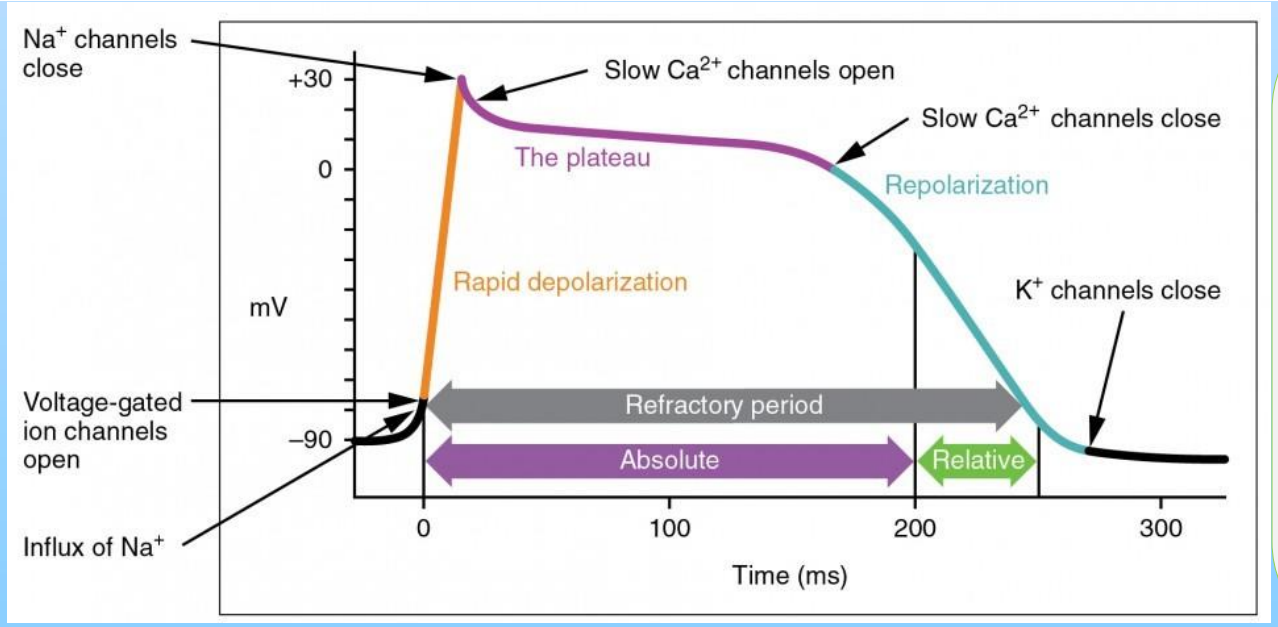


Basic – Anatomy & Physiology





- Phase 0 → Rapid Depolarization
- Phase 1 → Initial Repolarization
- Phase 2 → Plateau Phase
- Phase 3 → Rapid Repolarization
- Phase 4 → Resting Phase



We will discuss action potentials. Since we already know the four phases and the ions responsible for causing them, I won't explain those again.

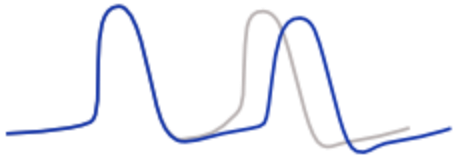
Why is understanding action potentials important in this case?
It is because of the absolute refractory period and the relative refractory period.

Let's move now to Pathophysiology.

Mechanisms of Arrythmias

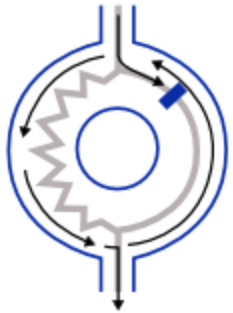
1

Abnormal Automaticity



2

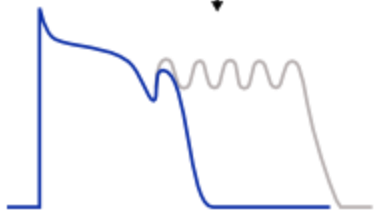
Re-entry



3

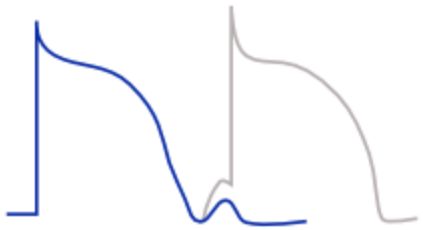
Triggered activity

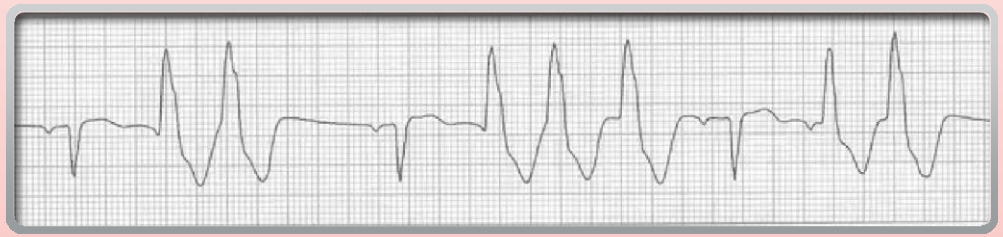
Early Afterdepolarizations

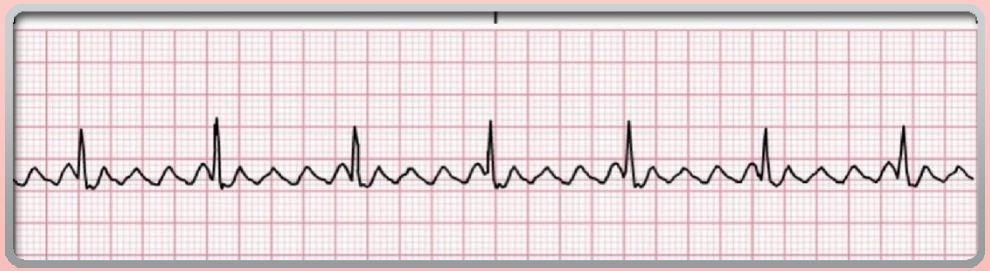


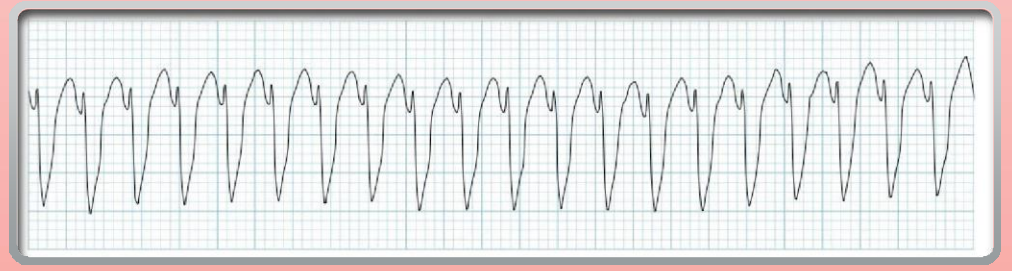
Triggered activity

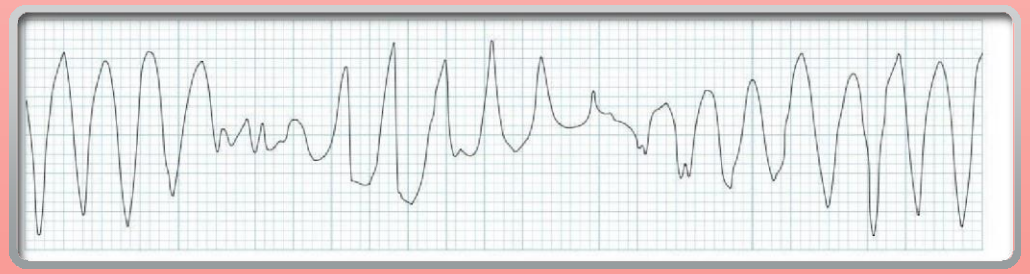
Delayed Afterdepolarizations









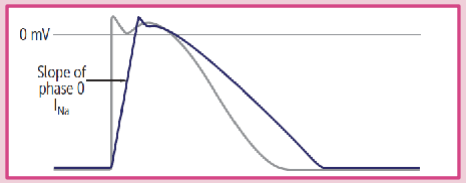
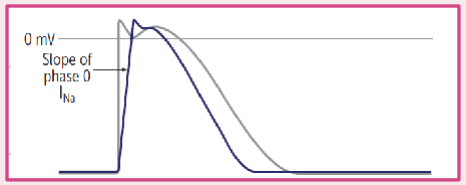
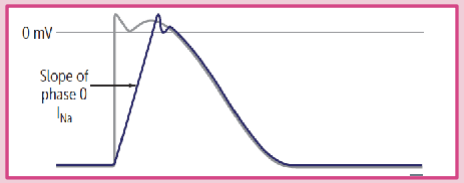
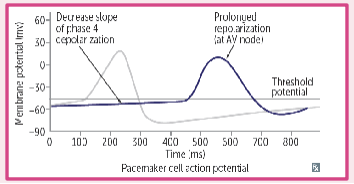
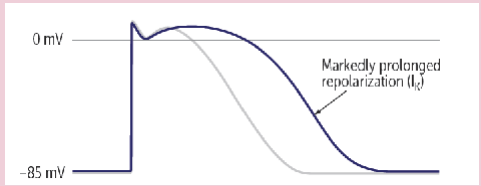
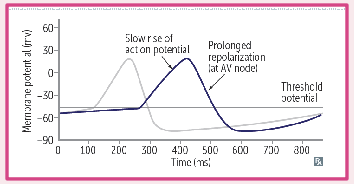


Please refer to the corresponding numbered mechanisms in the previous slide

Mechanisms of Arrhythmia:

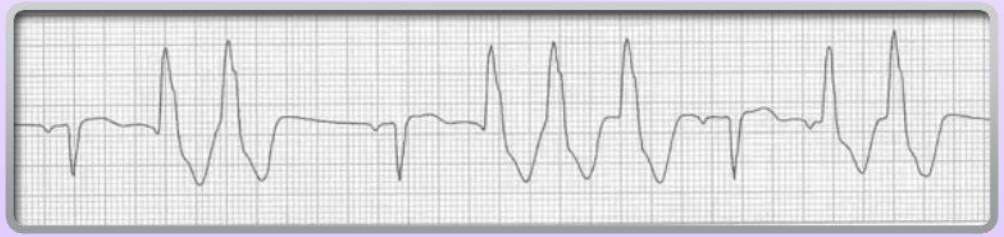
- 1- Increased automaticity --> faster phase 4
- 2- Reentry (the most common form of arrhythmia) and circuits, whether micro or macro.
- 3- Triggered activity occurs during repolarization. If it happens in early phase 3, it is called early afterdepolarization. If it occurs late, it is referred to as late afterdepolarization.
- Each one of these will correlate with a certain type of arrhythmias. So, this very basic is going to move to a very important clinical !!!
- 1- Increased automaticity --> increased premature atrial contractions and premature ventricular contractions (PVCs). (increased automaticity is the most common cause of PVCs).
- 2- Reentry --> ventricular tachycardia (VTs), atrial flutter, and atrial fibrillation.
- 3- Early afterdepolarization is the hallmark of Torsade de Pointes, a type of polymorphic ventricular tachycardia (VT), and is associated with prolonged QT intervals. This is because early afterdepolarization occurs during phase 3 of repolarization, which can increase the QT interval triggering Torsade de Pointes.****

 So, based on this information, I will treat the patient differently, as each class targets a certain phase.

Antiarrhythmic Class	Function	Drugs	Clinical Use	Limitations
Class IA		Quinidine Procainamide Disopyramide	Re-entrant Atrial & Ventricular Arrhythmia	Cinchonism TdP SLE like S/E
Class IB		Lidocaine Phenytoin Mexiletine	Acute VT, esp. Ischemic Digoxin induced Arrhythmias	CNS Toxicity
Class IC		Flecainide Propafenone	SVT	Proarrhythmic
Class II		B-Blockers	Increased Automaticity SVT Rate Control	Bradycardia & Hypotension ED Asthma
Class III		Amiodarone Ibutilide Dofetilide Sotalol	Atrial Fibrillation / Flutter VT	TdP Amiodarone S/E
Class IV		CCB (Verapamil, Diltiazem)	SA / AV Nodal Control	Bradycardia LV Dysfunction

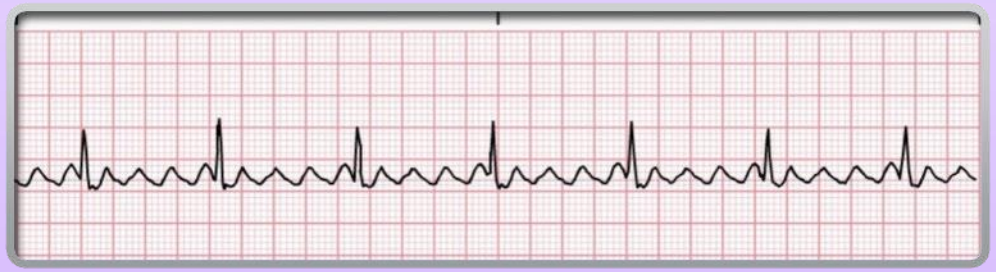
The patient is a 38-year-old female, recent history of Bronchitis treated with Azithromycin, heavy EtOH drinker, came to ED complaining of **Palpitations** in last 2 days.

A.



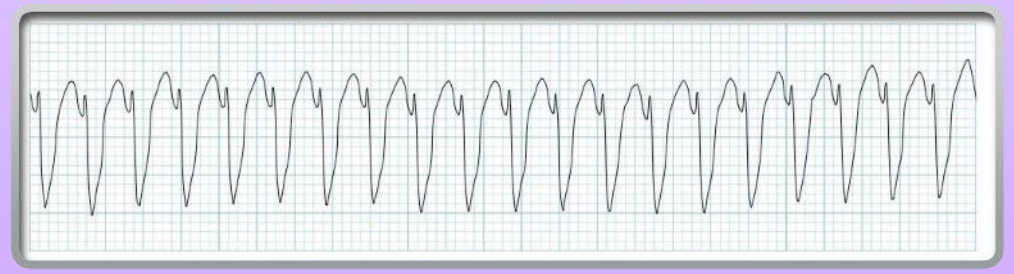
PVC

B.



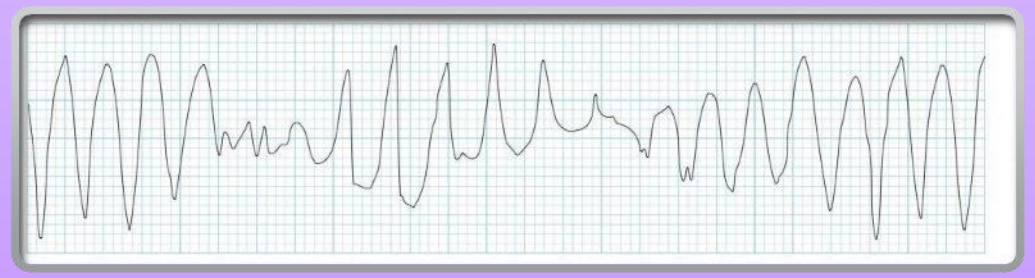
Atrial Flutter

C.



Monomorphic VT

D.



Polymorphic VT (TdP)

Treatment
B-Blockers
Rate Control (B-Blockers, CCB) Rhythm Control (Class III, Class IA/IC)
Amiodarone Class IB
Magnesium

- 1- In cases of increased automaticity, beta-blockers are the best option, as they decrease sympathetic overflow, which increases automaticity.

- 2- In Atrial Flutter and Monomorphic VT, we can:

A. Inhibit the circuit using a drug that affects all phases, such as amiodarone.
B. Alternatively, perform a procedure to ablate the circuit through targeted burning.

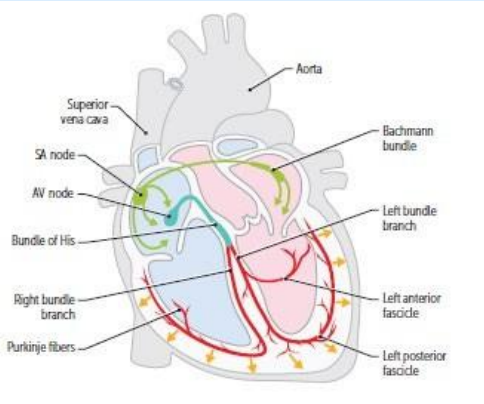
- So, in some cases of atrial flutter and monomorphic VT, treatment with medications may not be necessary. Instead, I can perform an ablation procedure to target and eliminate the circuit.

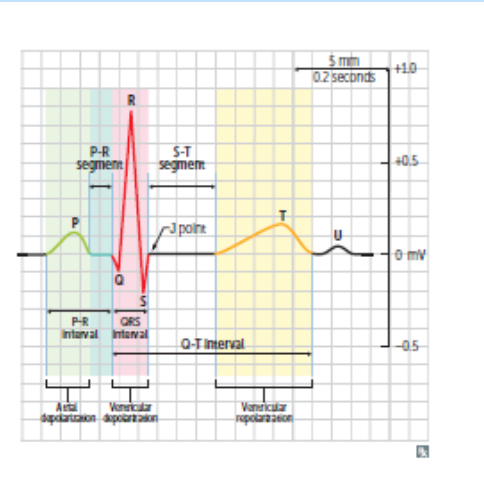
- So, knowing the pathophysiology of arrhythmias will guide you how to treat them.

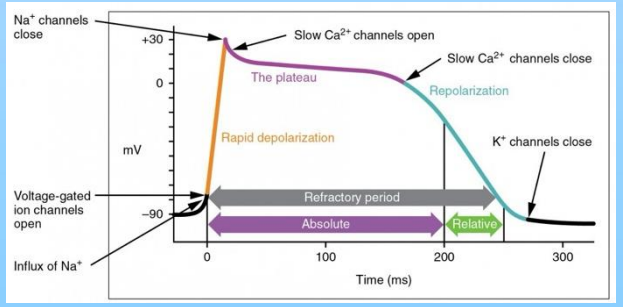
- 3- Since polymorphic VT is caused by early afterdepolarization, it is important to stabilize the myocardium. One of the most effective treatments is administering magnesium and avoiding QT prolongation.

- In our case, the patient is taking azithromycin, which can lead to QT prolongation. Therefore, I should consider withdrawing this drug.

Basic - Physiology

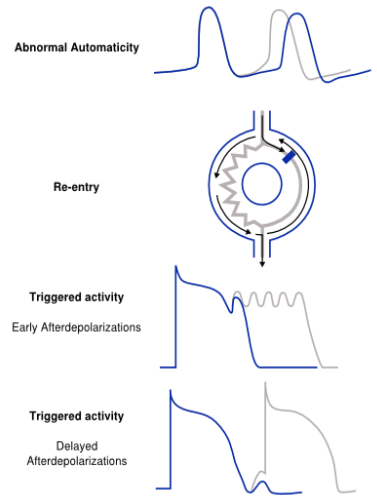






Basic - Pathology

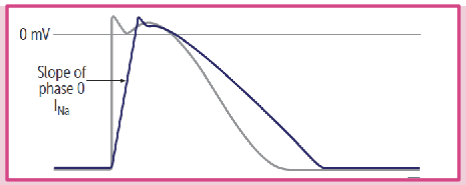
Mechanisms of Arrythmias



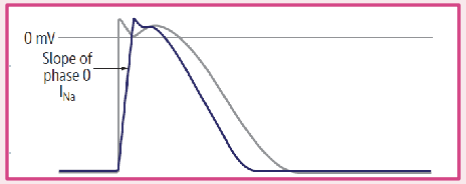
Basic - Pharmacology

Antiarrhythmic Class

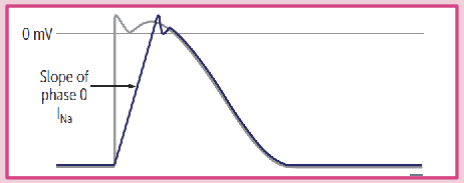
IA



IB



IC



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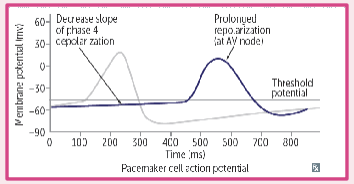
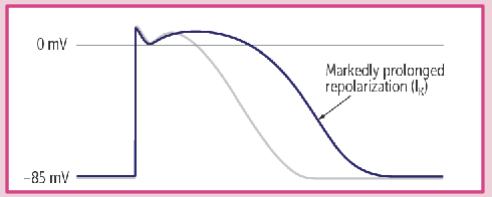
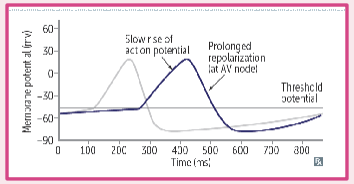


III



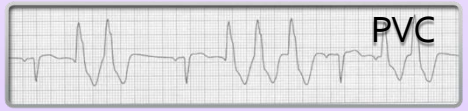
IV



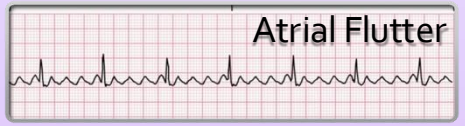
Clinical

The patient is a 38-year-old female, recent history of Bronchitis treated with Azithromycin, heavy EtOH drinker, came to ED complaining of **Palpitations** in last 2 days.

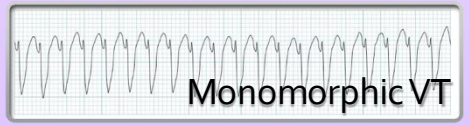
A.



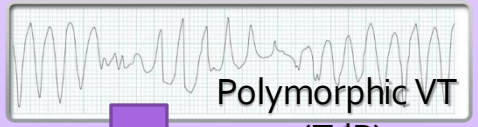
B.



C.



D.



Treatment

B-Blockers
Rate Control (B-Blockers, CCB) Rhythm Control (Class III, Class IA/IC)
Amiodarone Class IB
Magnesium

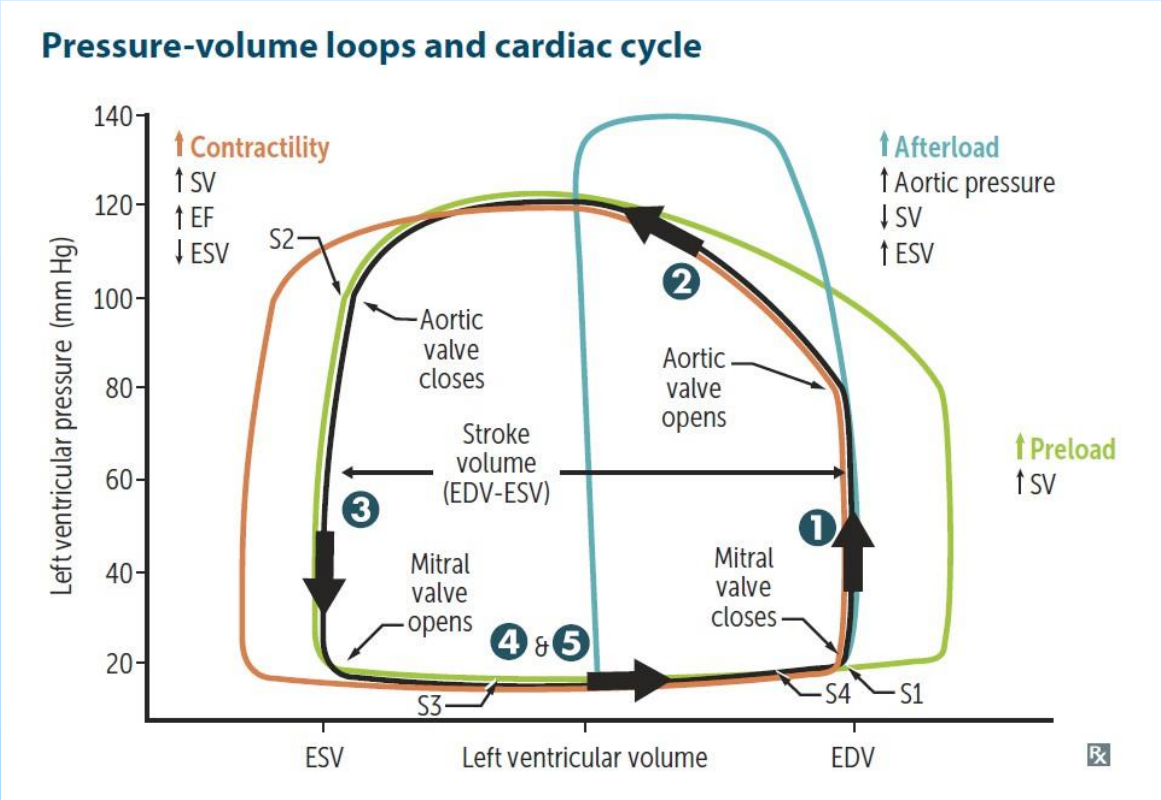
Case 3

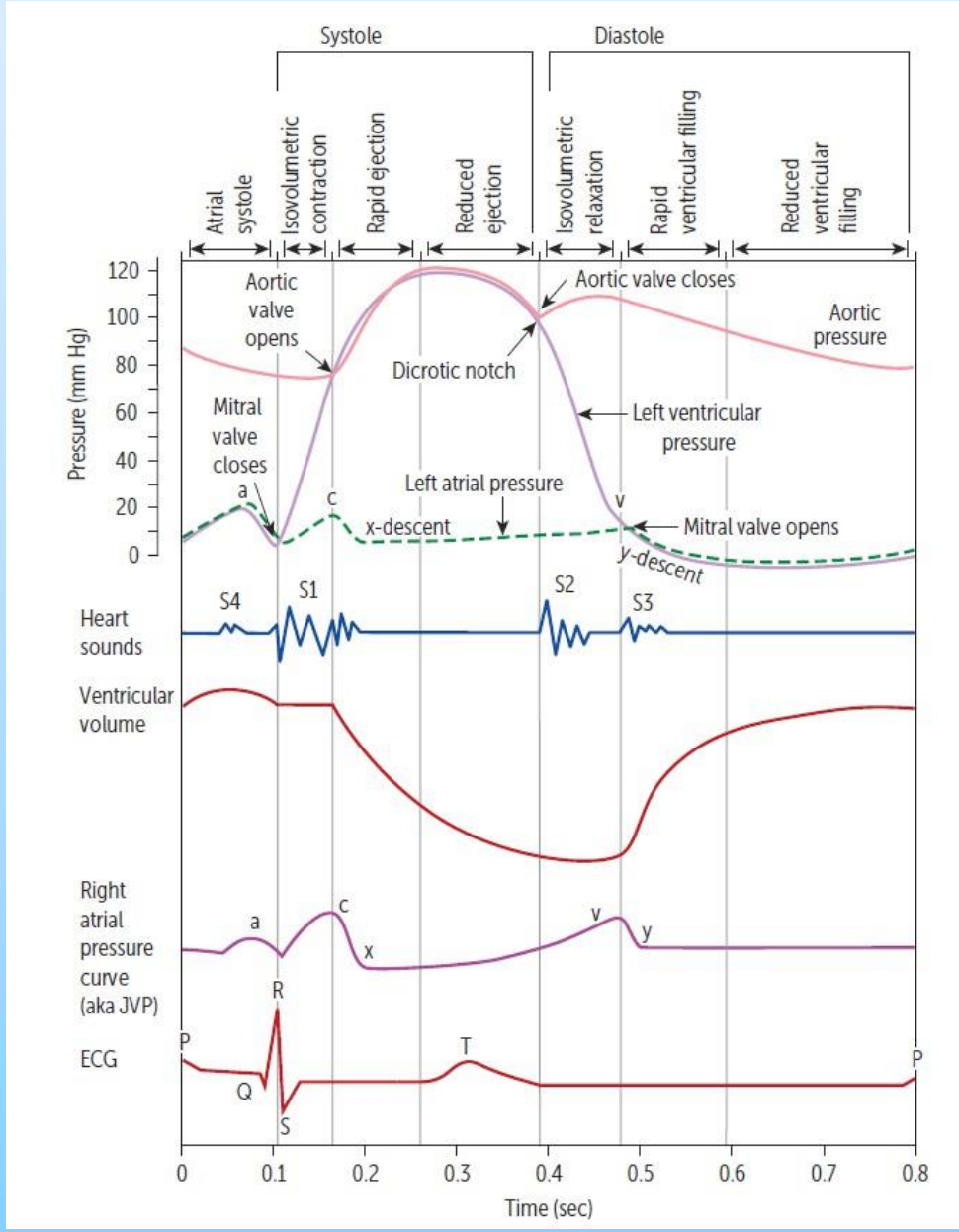
Valvular Heart Disease

Case 3

The patient is a 50-year-old male, came to clinic complaining of **Chest pain on Exertion and Recurrent Syncope** in last 2 months. On Physical Exam, he had an crescendo-decresendo systolic murmur with ejection click.

Basic – Anatomy & Physiology





- First of all, in such cases, I need to understand cardiac hemodynamics and how ESV (end-systolic volume), EDV (end-diastolic volume), and pressure interact with the heart.
- Additionally, I must know when in the cardiac cycle the aortic valve opens and closes, when the mitral valve opens and closes, and when there is flow across them. Depending on this information, I will make my decision.

We will mention some examples why it's important to know anatomy and physiology of the heart:

- When I say "systolic murmur," it means there is abnormal blood flow. A murmur is a sound created by this abnormal flow. For the sound to occur, there must be blood flow, and for blood flow to happen, it has to occur during a specific time in the cardiac cycle when the flow is present.

- For example, in aortic stenosis, there's an abnormal flow of blood across the aortic valve. When does the flow across the aortic valve occur? During systole. On the other hand, mitral regurgitation involves abnormal flow across the mitral valve (when there should be no flow across it). When does this happen? During systole, regurgitation occurs if there is backward flow through the mitral valve. (In these 2 states of abnormal flow, the sound is heard during systole -> systolic murmur).

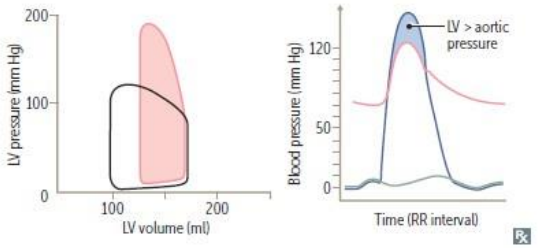
- In contrast, during aortic regurgitation, there should typically be no sound across the aortic valve during diastole. If abnormal sounds are heard during diastole across the aortic valve, it indicates aortic regurgitation, which is an abnormal finding. (So: diastolic murmur)

- Now, the knowledge of the cardiac cycle will help me determine which valve to expect the flow across and where to expect an abnormal sound. Additionally, S3 and S4 are heavily dependent on hemodynamics.

الدكتور بالسلايد السابق كان بعطي امثلة بقصد يورجيك كيف انه البيسك مرتبط بالكلينيكال)وهذا نظامه طول المحاضرة اصلا(، فاذا سألتني هل الامثلة السابقة حفظ؟ يعني كله اخذناه ففش داعي، بس افهم الفكرة العامة، انه في حال اجاك مريض اكون عارف الاساسيات تاعت ال cardiac cycleعشان اعرف اعمل تشخيص واعالج المريض)او احل سؤال الامتحان (😅

Pressure-volume loops and valvular disease

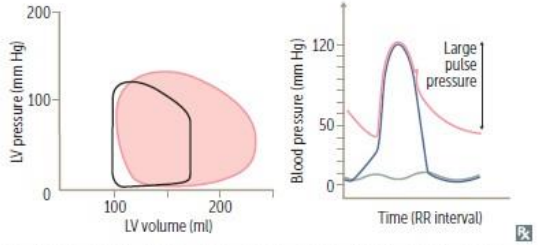
Aortic stenosis



↑ LV pressure
↑ ESV
No change in EDV (if mild)
↓ SV

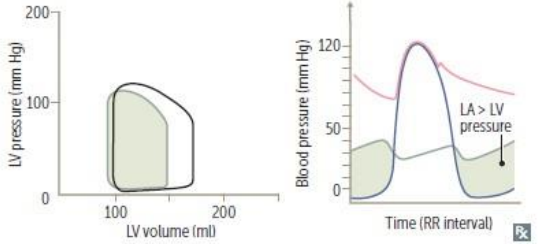
Ventricular hypertrophy → ↓ ventricular compliance → ↑ EDP for given EDV

Aortic regurgitation



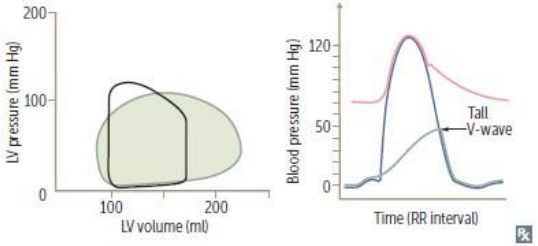
No true isovolumetric phase
↑ EDV
↑ SV
Loss of dichrotic notch

Mitral stenosis



↑ LA pressure
↓ EDV because of impaired ventricular filling
↓ ESV
↓ SV

Mitral regurgitation

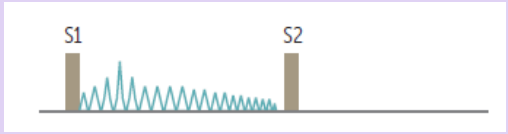
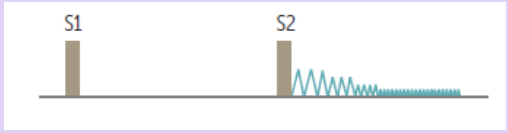
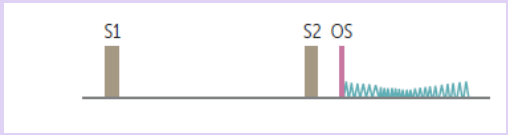
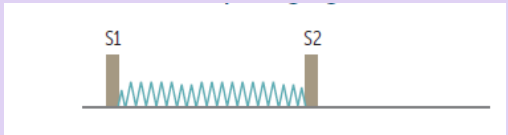


No true isovolumetric phase
↓ ESV due to ↓ resistance and ↑ regurgitation into LA during systole
↑ EDV due to ↑ LA volume/pressure from regurgitation → ↑ ventricular filling
↑ SV (forward flow into systemic circulation plus backflow into LA)

VHD	Key Concepts	Result	Clinical
Aortic Stenosis	↑ LVP	Subendocardial Ischemia	Angina
	↓ SV	↓ Afterload	Syncope
Aortic Regurgitation	Bradycardia	Longer Regurg. Time	↑ HF
Mitral Stenosis	Tachycardia	Shorter Filling time	Pul. Edema
Mitral Regurgitation	↑ Afterload	Increased Regurgitation	↑ HF

Clinical – Diagnosis

The patient is a 50-year-old male, came to clinic complaining of **Chest pain on Exertion and Recurrent Syncope** in last 2 months. On Physical Exam, he had a crescendo-decresendo systolic murmur with ejection click.

Valvular Heart Disease	Symptoms	Physical Examination
Aortic Stenosis	Angina, Dyspnea, Syncope, HF	
Aortic Regurgitation	HF	
Mitral Stenosis	Pulmonary Congestion	
Mitral Regurgitation	HF	

Clinical – Treatment

The patient is a 50-year-old male, came to clinic complaining of **Chest pain on Exertion and Recurrent Syncope** in last 2 months. On Physical Exam, he had an crescendo-decresendo systolic murmur with ejection click.

Valvular Heart Disease	Symptoms	Key Treatment	Treatment
Aortic Stenosis	Angina, Dyspnea, Syncope, HF	↓ LVP Avoid ↓ Afterload	Relief Mech. Obstruction Avoid Vasodilators
Aortic Regurgitation	HF	↓ Afterload Avoid Bradycardia	Diuretics Vasodilators Avoid B-blockers, CCB
Mitral Stenosis	Pulmonary Congestion	Avoid Tachycardia	B-Blockers Diuretics
Mitral Regurgitation	HF	↓ Afterload	Vasodilators Diuretics

The basics have a significant impact on how I will treat the patient. Let's consider 3 examples.

- **First example: Mitral regurgitation.**

When the heart is pumping, blood should flow through the aortic valve, and the mitral valve should close to prevent any backflow.

- **What hemodynamic change makes mitral regurgitation worse?**

- The answer is **increased afterload.** When afterload is increased, the heart has to work harder to pump blood toward the correct direction. And in cases of mitral regurgitation, instead of pumping in the proper direction, the increased afterload causes the blood to flow backward through the mitral valve, worsening the regurgitation.

For instance:

- If the patient is **hypertensive**, the increased afterload will make mitral regurgitation worse.
- If I give the patient a **vasodilator,** it will reduce afterload, allowing the heart to pump more effectively and improving the patient's condition.

Example 2: Mitral Stenosis

- **What is the hemodynamic change that makes mitral stenosis worse?**

The answer is **tachycardia.**

- When the heart rate increases, the time available during diastole for the heart to fill with blood is shortened. Since mitral stenosis involves a narrowed mitral valve, the already compromised filling capacity is further worsened by this shorter diastolic period. As a result:

- **Higher left atrial pressure** develops.

- This leads to **more symptoms and decompensation.**

How can this be improved?

By slowing down the heart rate, the patient's condition can improve. This can be achieved by:

- Administering **beta blockers** to reduce heart rate.
- Preventing **atrial fibrillation,** which can further shorten diastolic filling time and exacerbate mitral stenosis.

Third example: Aortic Regurgitation

- **What is the hemodynamic change that makes aortic regurgitation worse?**

The answer is **bradycardia.**

- When the heart rate is slow, diastole lasts longer. During this extended diastolic period, more blood flows back (regurgitates) into the left ventricle through the incompetent aortic valve, worsening the patient's condition.

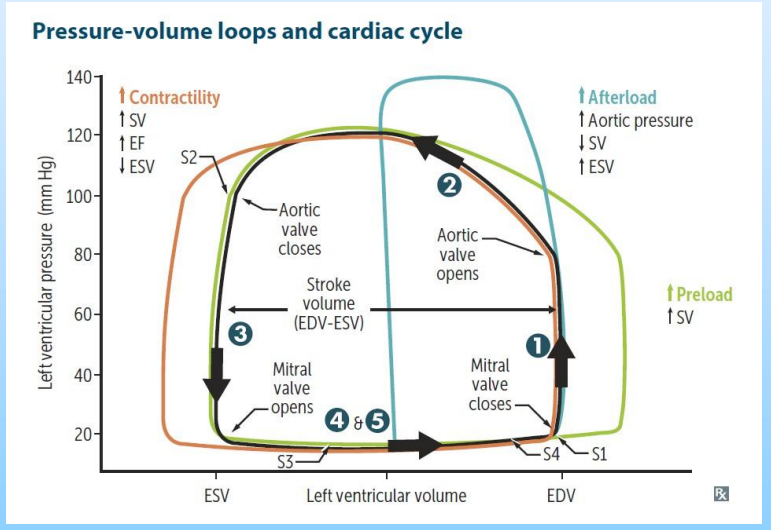
- **How does this affect treatment?**

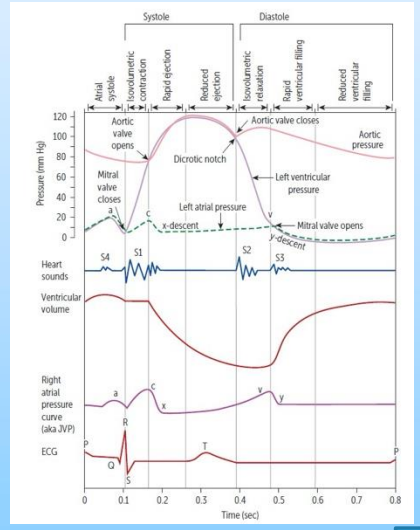
In patients with severe aortic regurgitation:

- **Beta blockers** should be **avoided** because they can slow the heart rate, prolonging diastole and increasing regurgitation.

This demonstrates how understanding the basics can guide you in treating your patient effectively.

Basic - Physiology

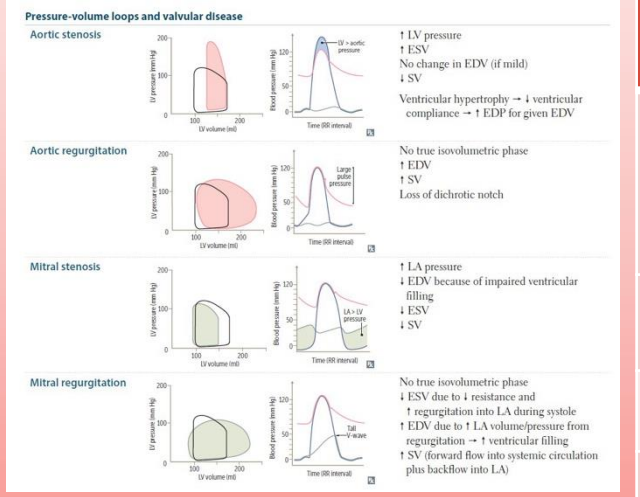




Clinical - Treatment

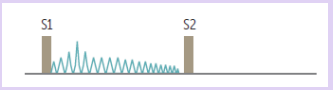
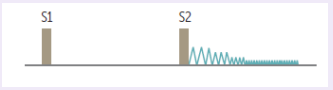
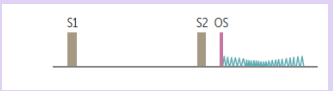
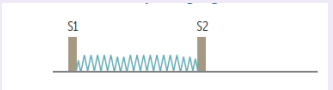
Valvular Heart Disease	Symptoms	Key Treatment	Treatment
Aortic Stenosis	Angina, Dyspnea, Syncope, HF	↓ LVP Avoid ↓ Afterload	Relief Mech. Obstruction Avoid Vasodilators
Aortic Regurgitation	HF	↓ Afterload Avoid Bradycardia	Diuretics Vasodilators Avoid B-blockers, CCB
Mitral Stenosis	Pulmonary Congestion	Avoid Tachycardia	B-Blockers Diuretics
Mitral Regurgitation	HF	↓ Afterload	Vasodilators Diuretics

Basic - Pathology



VHD	Key Concepts	Result	Clinical
Aortic Stenosis	↑ LVP	Subendocardial Ischemia	Angina
	↓ SV	↓ Afterload	Syncope
Aortic Regurgitation	Bradycardia	Longer Regurg. Time	↑ HF
Mitral Stenosis	Tachycardia	Shorter Filling time	Pul. Edema
Mitral Regurgitation	↑ Afterload	Increased Regurgitation	↑ HF

Clinical - Diagnosis

Valvular Heart Disease	Symptoms	Physical Examination
Aortic Stenosis	Angina, Dyspnea, Syncope, HF	
Aortic Regurgitation	HF	
Mitral Stenosis	Pulmonary Congestion	
Mitral Regugitation	HF	

Case 4

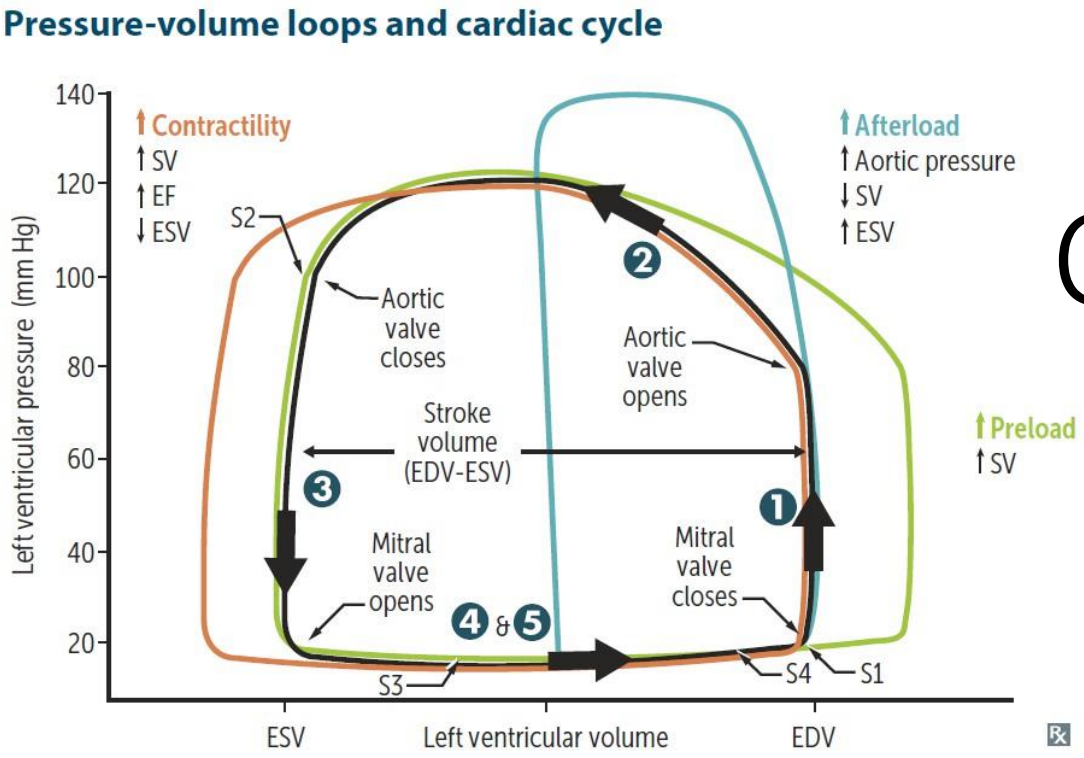
Heart Failure 

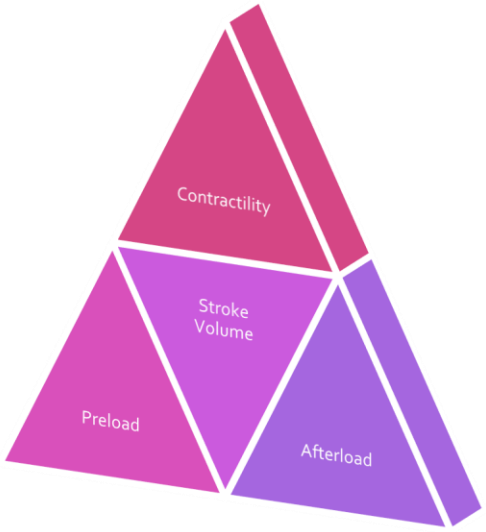
Case 4

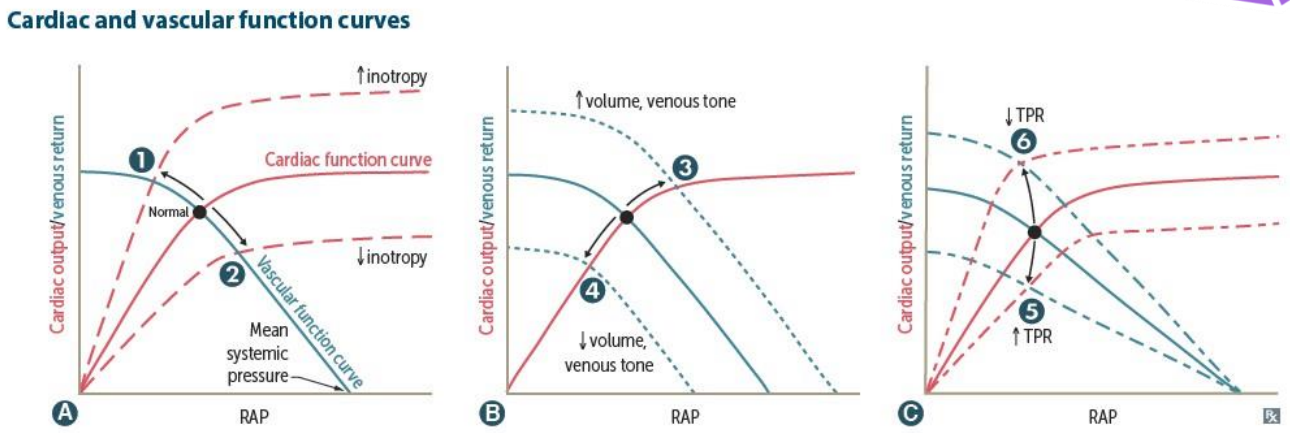
The patient is a 55-year-old female, recent history of COVID Infection 2 weeks ago, came to ED complaining of **Dyspnea and Lower Extremity Swelling** in last week.

CO: Cardiac Output
HR: Heart Rate
SV: Strok Volume

C.O. = HR x SV

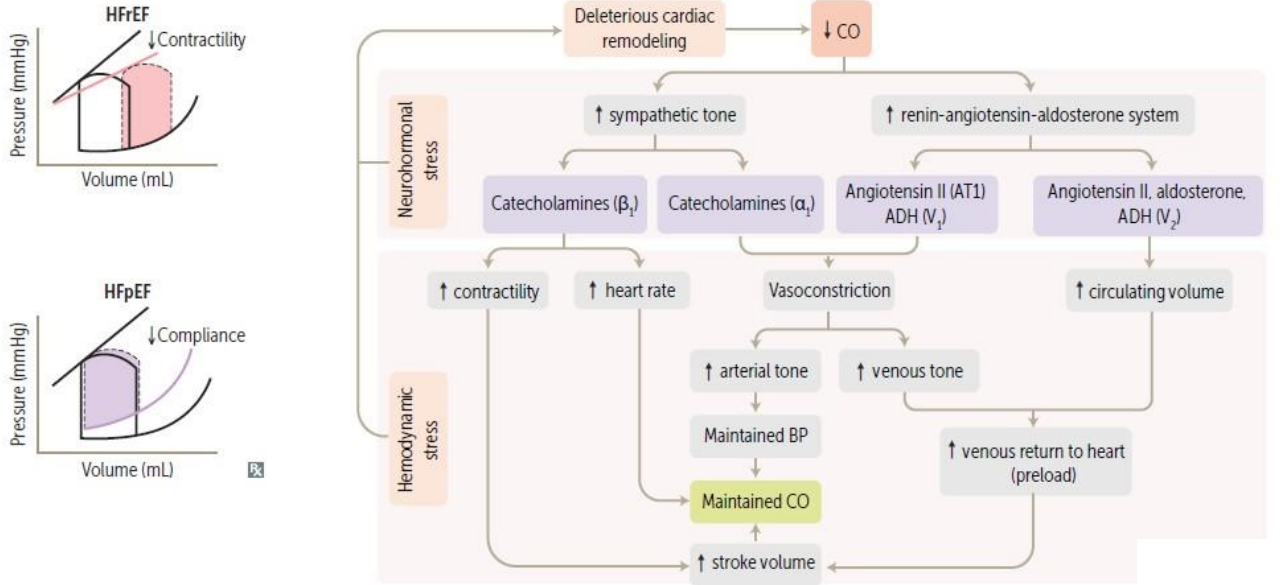






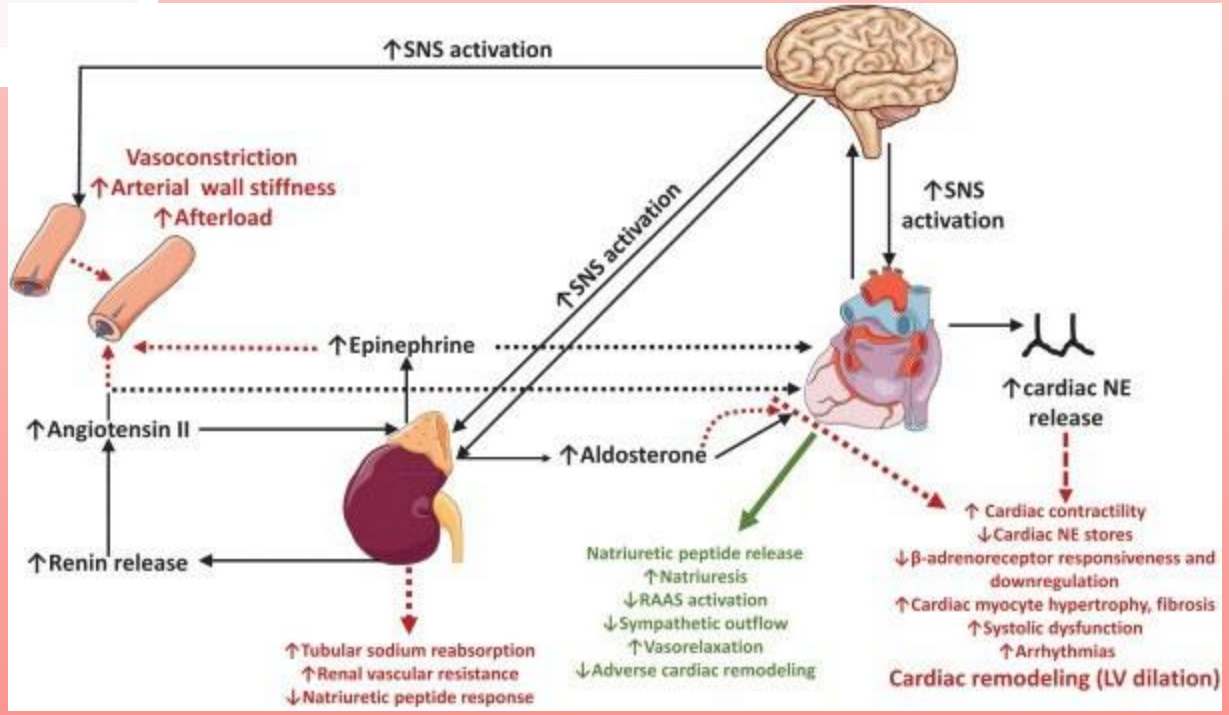
- SV depends on:

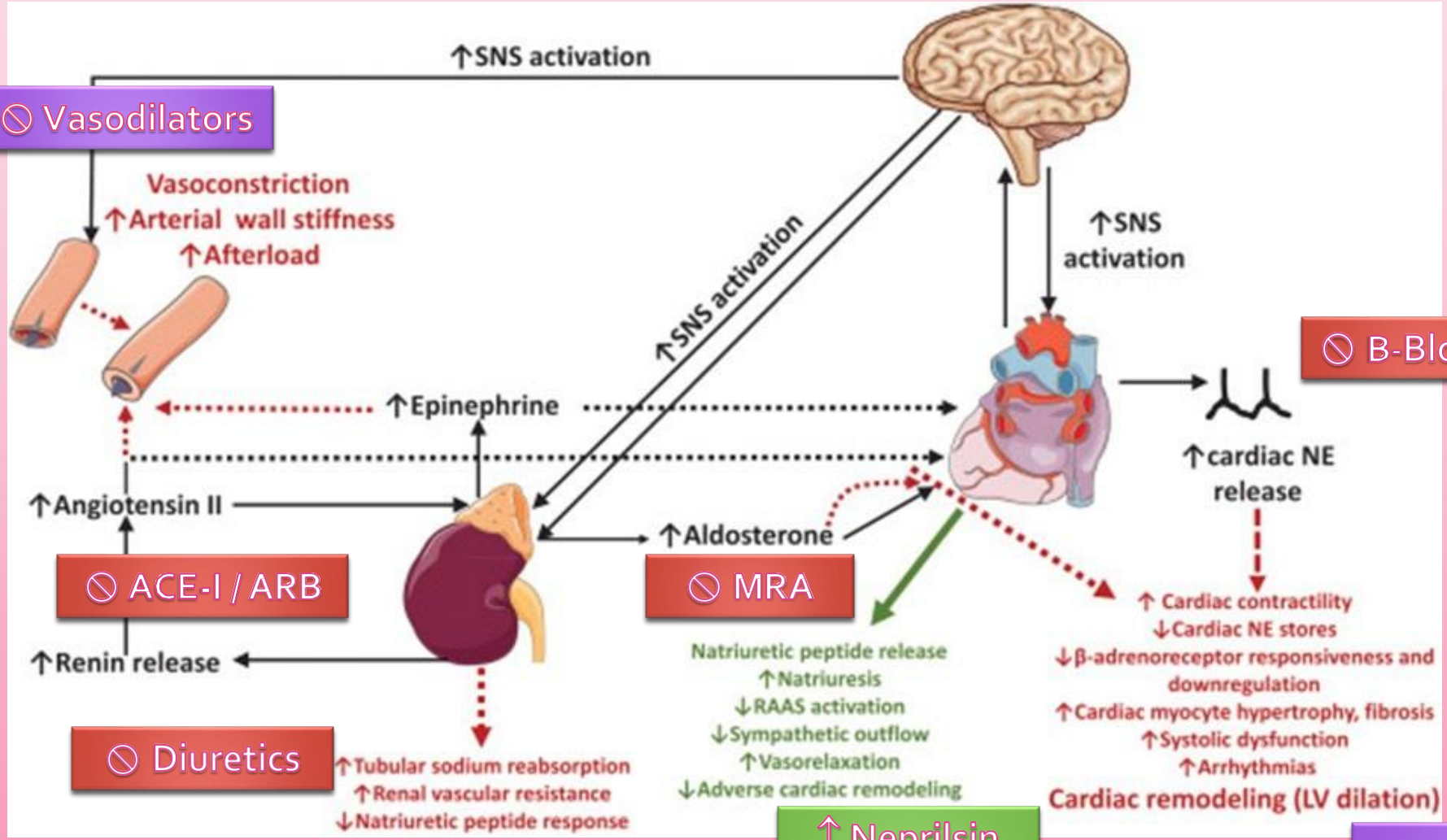
1- contractility
2- afterload
3- preload



Treatment of Heart Failure:
First of all, understanding the pathophysiology in this patient is crucial. Knowing that a viral infection could lead to myocarditis, which can subsequently cause dilated cardiomyopathy, is critical. Dilated cardiomyopathy results in a neurohormonal state that is detrimental, and this is a key point.

- All the compensatory mechanisms we study are beneficial in the short term, but in the long term, they actually become part of the disease itself. Therefore, the goal is to prevent these mechanisms from causing further harm.
- How do I prevent this pharmacologically?




↑SNS activation
⃠ Vasodilators
Vasoconstriction
↑Arterial wall stiffness
↑Afterload
↑SNS activation
↑SNS activation
⃠ B-Blockers
↑Epinephrine
↑cardiac NE release
↑Angiotensin II
↑Aldosterone
⃠ ACE-I / ARB
⃠ MRA
↑Renin release
↑ Cardiac contractility
↓Cardiac NE stores
↓β-adrenoreceptor responsiveness and downregulation
↑Cardiac myocyte hypertrophy, fibrosis
↑Systolic dysfunction
↑Arrhythmias
Cardiac remodeling (LV dilation)
Natriuretic peptide release
↑Natriuresis
↓RAAS activation
↓Sympathetic outflow
↑Vasorelaxation
↓Adverse cardiac remodeling
⃠ Diuretics
↑Tubular sodium reabsorption
↑Renal vascular resistance
↓Natriuretic peptide response
↑ Neprilsin Inhibitor
Inotropes
Cardiac Glycosides

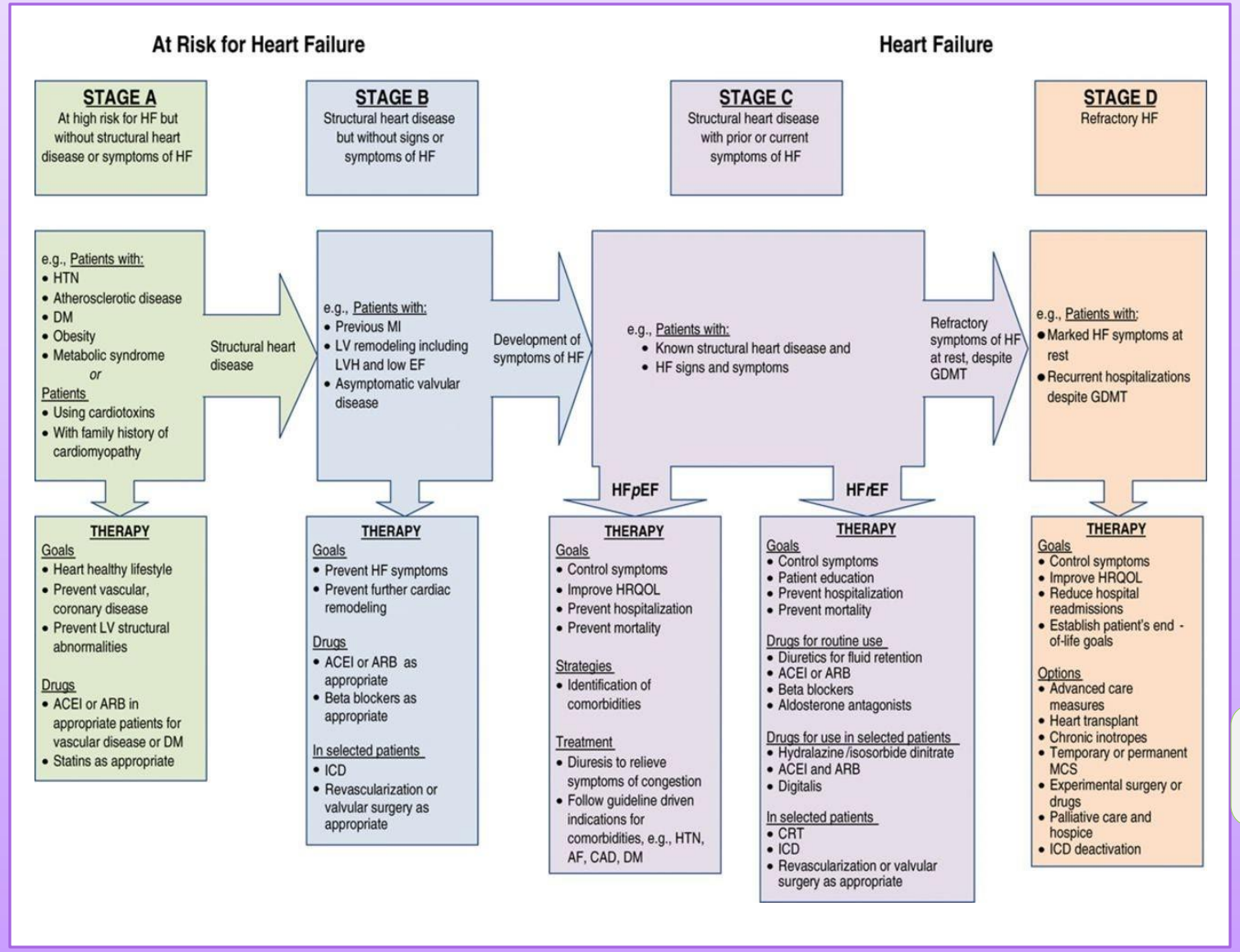
- You have four pillars of treatment for heart failure:

1- Mineralocorticoid receptor antagonists (MRAs)
2- ACE inhibitors (ACEIs) and ARBs
3- Beta blockers
4- Diuretics and SGLT2 inhibitors

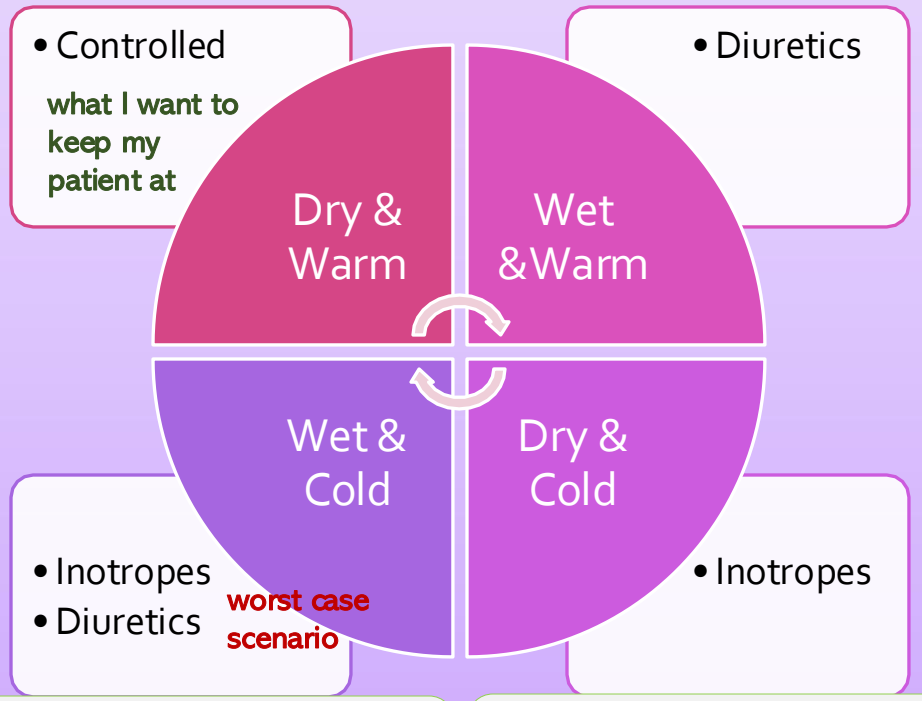
- Here, maintaining balance is crucial. For example:

- If the patient presents with **volume overload**, the focus is on administering **diuretics** to alleviate the excess fluid.
- If the patient presents **dry** (no volume overload) **with low perfusion**, the treatment may involve **vasodilators** or **inotropes** to improve perfusion.
- If the patient presents **wet with no forward output** (low perfusion), the approach combines **inotropes** to enhance contractility and **diuretics** to manage fluid overload.

The patient is a 55-year-old female, recent history of COVID Infection 2 weeks ago, came to ED complaining of **Dyspnea and Lower Extremity Swelling** in last week.



Forrester Classification Of The Heart.

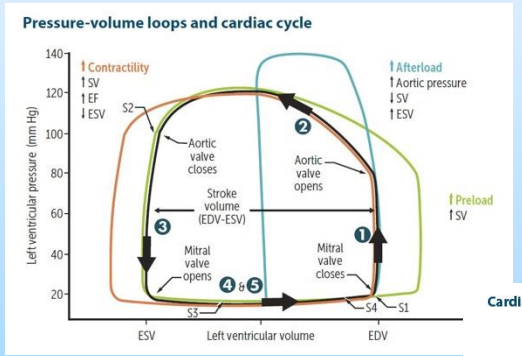


Warm: good perfusion
Cold: low/poor perfusion

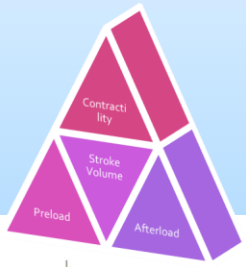
Wet: volume overload
Dry: no volume overload

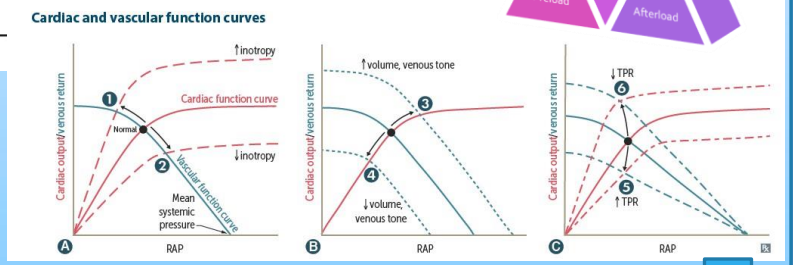
The hemodynamic state is going to dictate how I am going to treat.

Basic - Physiology



C.O. = HR x SV





Clinical – Diagnosis & Treatment

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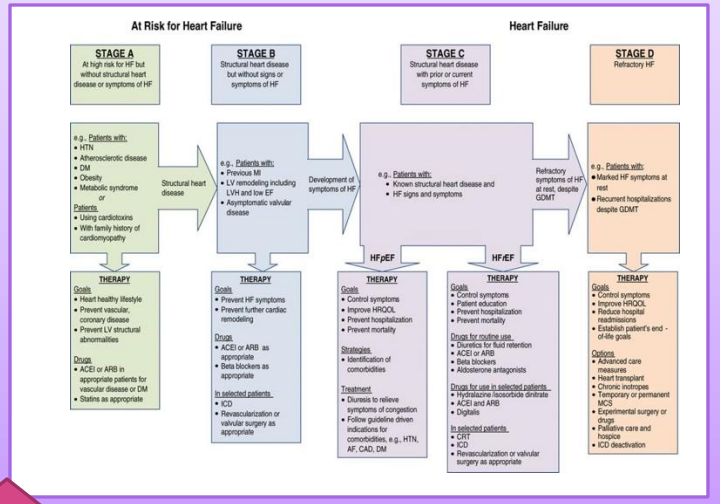
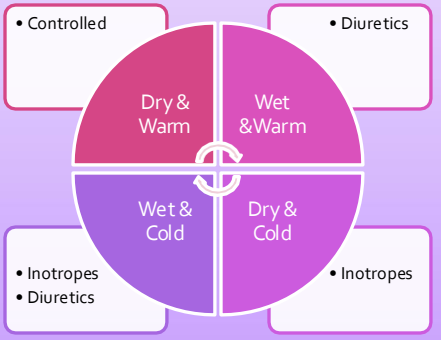
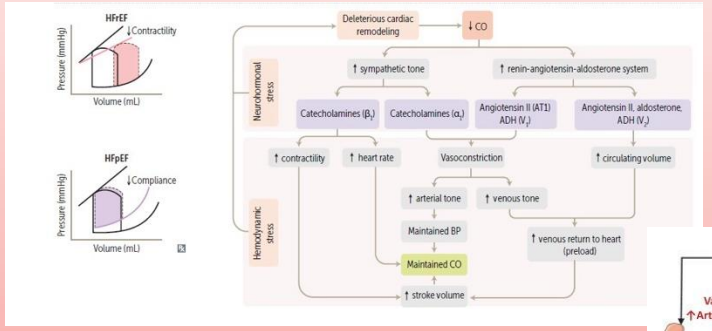
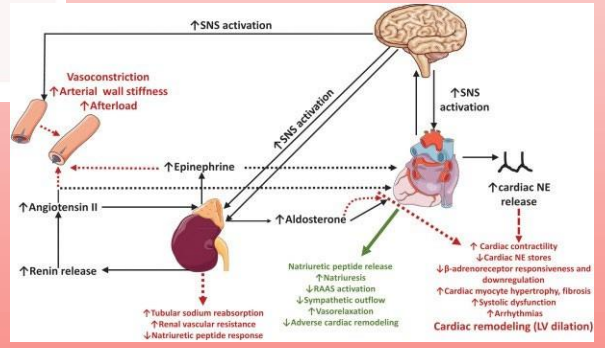


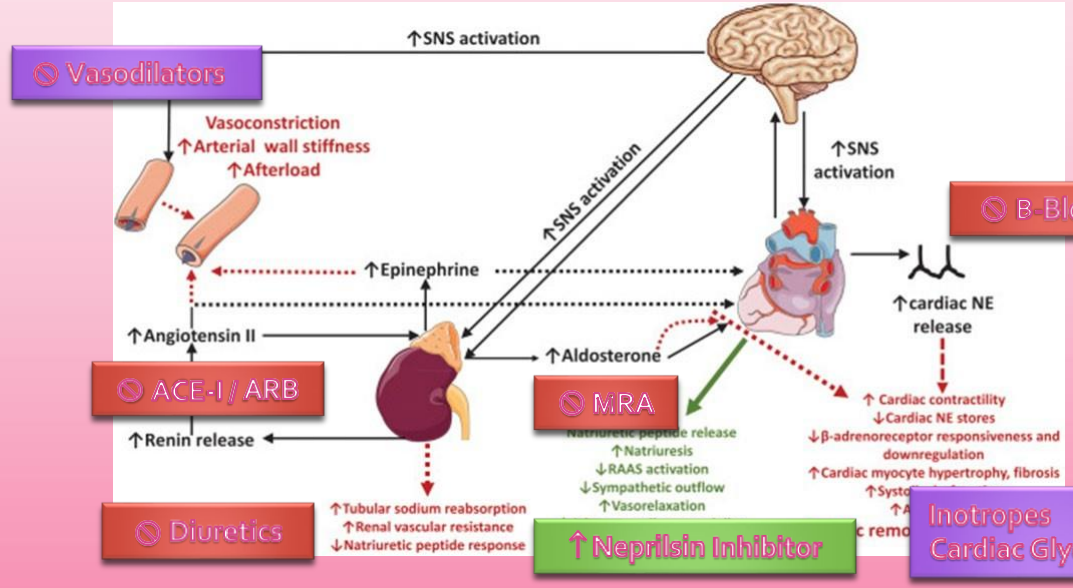


Basic - Pathology

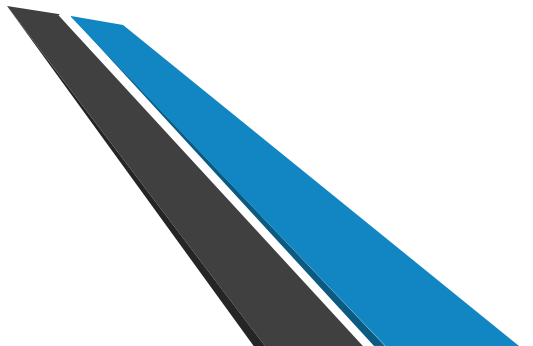




Basic - Pharmacology



Thank You



الآن الآن... سوريا بلا اسد سقط النظام وحرٌ انت يا بلدي

نسأل الله الفرج القريب لاخوتنا في غزة

يا عابدَ الحَرَمينِ لوْ أبصرْتَنا لَعلمْتَ أنّك في العبادةِ تَلْعبُ
مَنْ كان يَخْضِبُ خدَّهُ بدموعِهِ فَنُحُورُنا بِدِمَائِنا تَتَخَضَّبُ

VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2			
V2→V3			

امسح الرمز و شاركنا بأفكارك لتحسين أدائنا !!