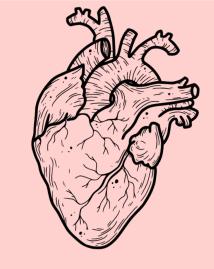






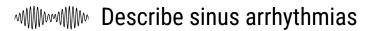
# **ARRYTHMIAS**



Editing File



# **Objectives**



Describe the main pathophysiological causes of cardiac arrhythmias

Explain the mechanism of cardiac block

Enumerate the common arrhythmias and describe the basic ECG changes



## Read This Before you Continue

- 1. We recommend you study the Physiology ECG lecture before you study this lecture.
- 2. In terms of the exam, we don't know how the questions will come. It may come as a an ECG strip picture and you will be asked to identify the rhythm.
- 3. ECG is a very complex topic with many intricacies above a 1st year's level. However there are important basic topics that you should be able to identify, like Sinus rhythm, Atrial Fibrillation/Flutter and AV Blocks.
- 4. Everything in slides is included. However there are things we see as more **important** from our point of view and reviewing the objectives. so we wrote it in **red**. But **nothing is from the doctor**.
- 5. The Lecture is simple if you understand it. We highly recommend you check the summary and the stepwise approach alongside the video of our explanation it in the next 3 slides. It covers all content covered in this lecture in an organized manner (if you understand it, it will be ان شاء الله more than enough for your level.)





00:00 - 51:01

### Our Approach and Summary

Don't be scared, the next 3 pages contain the basic approach to ECG for a medical student level + a summary of all ECGs mentioned in the lecture with clear pictures + how to identify them





## Stepwise Approach to Reading ECG



Extra slide

Intro + Approach 00:00 - 11:25

#### Step 1: are there p waves?

sinus p waves = originate in SA node = upright in lead II, III, AVF (inferior)

#### Step 2: Regular/Irregular R-R Interval?

- P waves present + Regular → Most likely Sinus Rhythm
- No p waves + Irregularly Irregular → Atrial Fibrillation  $\circ$

#### Step 4: Check the Intervals?

- PR (normal <200ms = 5 small boxes = 1 big box)
  - Prolonged in AV block
    - Constant PR + No drop beat  $\rightarrow$  1st degree AV Block
    - Progressive PR Prolongation + Drop Beat → Mobitz I (2nd degree AV Block)
    - Constant PR + Drop Beat → Mobitz II (2nd degree AV Block)
    - Complete AV Dissociation (no relationship between P waves and QRS)  $\rightarrow$  3rd degree AV Block (complete)
- QT (normal <1/2 R-R interval)

#### Step 3: Wide or Narrow?

- Narrow QRS (<120ms; 3 small boxes)
  - His-Purkinje system works and No bundle branch blocks present
  - The only way you get a narrow QRS is if conduction goes through the AV node → bundle branches →Purkinje fibers (normally).
- Wide ORS
  - Most likely → bundle branch block
  - Ventricular rhythm → (i.e. Ventricular Tachycardia, Ventricular Fibrillation )

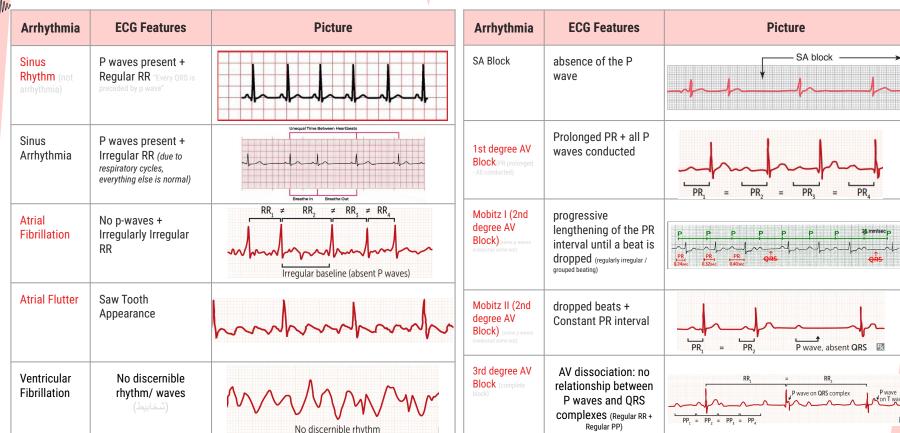
#### **Step 5: ST segments?**

- T wave abnormalities
  - Inverted → ischemia
  - Peaked  $\rightarrow$  Early ischemia, hyperkalemia ( $\uparrow K$ )
  - Flat/U waves  $\rightarrow$  Hypokalemia ( $\downarrow$ K)
- ST Depression → Subendocardial (partial) Ischemia or infarction (Angina or NSTEMI)
- ST Elevation → Transmural (complete) ischemia or infarction (STEMI or Vasospastic Angina)



## Summary (1/2)





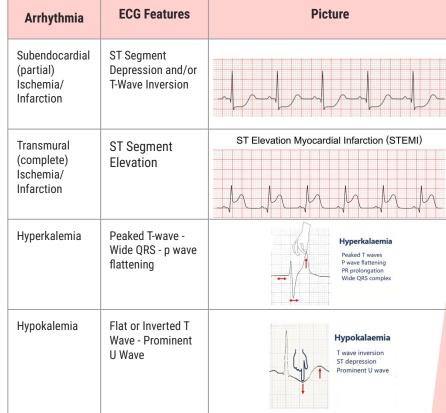


## **Summary (2/2)**

Video: Summary 2/2 *26:55 - 41:20* 



Arrhythmia	ECG Features	Picture
PACs (Premature Atrial Contractions	Sinus rhythm then → Early P wave + Narrow QRS complex	25 mm/s
PVCs (Premature Ventricular Contractions)	Sinus Rhythm then → Early Wide QRS + no p wave	
SVT (Supraventricula r Tachycardia)	No p wave (hidden) + Regular RR + Narrow QRS Tachycardia	MAHAHAHAHAHA
VT (Ventricular Tachycardia)	Regular RR + Wide QRS Tachycardia	\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\

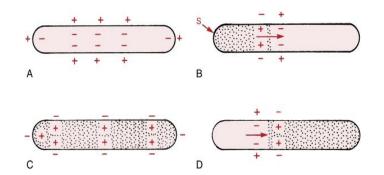


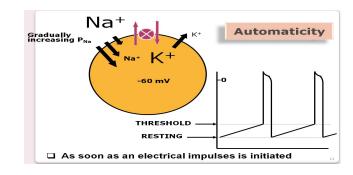
# **Quick Review**

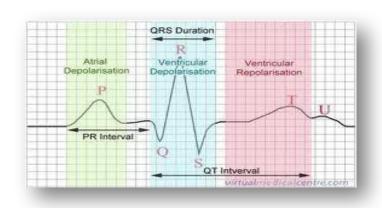
The pictures in this section are in slides but not in lecture objectives so just skim them as a review

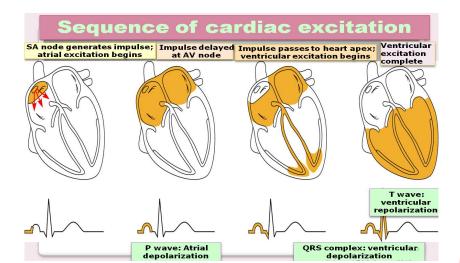


## **Depolarization and Repolarization**



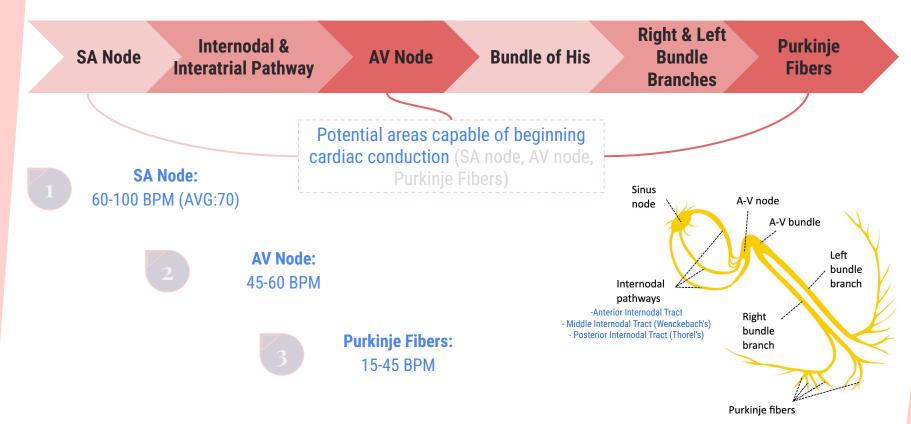


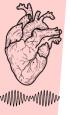






#### **Electrical conduction and normal conduction system**





#### **Sequence of Cardiac Excitation**

not in slides, we added hetter quality ones

1 mm = 0.04 seconds**Limb leads Chest leads** RA (white) LA (black) RL (green) LL (red)

Am 5.0 = mm 8.0 mm 8.0

1 Small Box on Y-axis = 0.1mv = 1mm 1 Big Box on Y-axis = 5 small boxes = 0.5mv = 5mr

V<sub>1</sub> 4th intercostal space, right sternal border

 $V_2$  4th intercostal space, left sternal border

 $V_3$  Midway between  $V_2$  and  $V_4$ 

 $V_4$  5th intercostal space, left midclavicular line

5 mm = 0.20 seconds

 $V_{\mathfrak{s}}$  Left anterior axillary line, same level as  $V_{\mathfrak{s}}$ 

V<sub>6</sub> Left midaxillary line, same level as V<sub>4</sub> and V<sub>5</sub>

RL (green): right leg, slightly proximal to ankle
RA (white): right arm, slightly proximal to wrist
LL (red): left leg, slightly proximal to ankle
LA (black): left arm, slightly proximal to wrist

These were not mentioned here but know them for physiology lecture

# 3

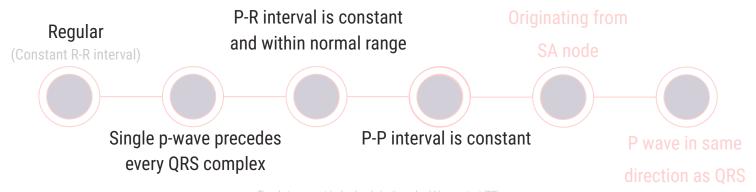
## **Sinus Rhythm**

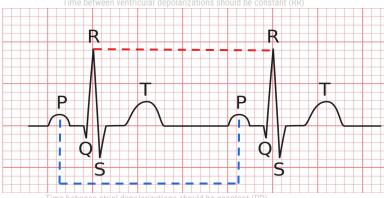
This section is just to understand what normal rhythm look like.

"If you know what's normal, then you know what's abnormal" - someone smart



# **Normal Sinus Rhythm**







## **Interpretation & Rate**

γν	Approach to ECG	Method	
	Rate (60-100 -> Normal)	a) Regular R-R Interval - 300/#big boxes between R-R interval b) Irregular R-R Interval - Count all R waves then multiply by 6 (each strip is 10 sec)	
	Rhythm (including intervals and blocks)	Anything other than previous slide (Sinus Rhythm) is considered abnormal rhythm	
	Axis	Not mentioned in slides (but for your own information watch this <u>video</u> )	
	Hypertrophy	-	
	Ischemia	ST segment changes	

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



## **Abnormal Sinus Rhythm**

You can try it yourself (take a deep inspiration and you will feel your heart beating faster, expire and you will see it going slower)

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Was		Tachycardia an increase in the heart rate	Bradycardia	Sinus Arrhythmia
	Rate	Heart Rate > 100 Beats Per Minute (Normal = 60-100)	Heart Rate < 60 Beats Per Minute (Normal = 60-100)	A variation of the heart rate during respiration( <i>R-R Intervals are irregular</i> ), which is normal and common in young adults.
	Causes	<ul><li>Sympathetic stimulation</li><li>Increased Body temperature</li><li>Drugs: digitalis</li><li>Inspiration</li></ul>	- Parasympathetic stimulation - Expiration	- Result from spillover of signals from the medullary respiratory center into the adjacent vasomotor center during inspiration and expiratory cycles of respiration.  - The spillover signals cause alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart
	Picture	hhhhhhhluhhhhl	A	Hail: Guyton and Hail Textbook of Medical Physiology, 12th Edition

# 4

## **Overview of Arrhythmias**

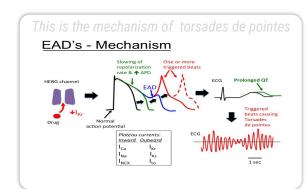
Here we cover basic mechanisms of arrhythmias. In addition to how they are classified

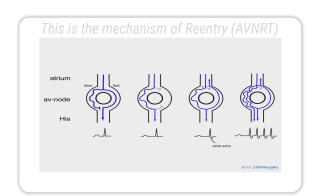


### Causes/Mechanisms of Cardiac Arrhythmias



- 1. Abnormal rhythmicity of the pacemaker
- 2. Shift of the pacemaker from the sinus node to another place in the heart
- 3. Blocks at different points in the spread of impulse through the heart
- 4. Trigger
- 5. Reentry
- 6. Abnormal pathways of impulse transmission through the heart
- 7. Spontaneous generation of spurious impulses in almost any part of the heart







#### **Classification / Causes of Cardiac Arrhythmias**



- Rate Above or Below Normal (Tachyarrhythmias vs. Bradyarrhythmias)
- Regular or irregular rhythm
- Narrow or Broad Rhythm QRS Complex

(Narrow QRS = Fast ventricular depolarization = conduction went through Bundle branches→ purkinje → myocytes) | (Wide QRS = Slow Ventricular Depolarization)

- Relation to P Waves (absent p waves?)
- Supraventricular vs. Ventricular (Supraventricular = originates above AV node) | (Ventricular = originates below AV node)



## **COMMON ARRHYTHMIAS**

This table is only female slides and in our opinion it's not important to memorize all. Because some are not even mentioned in next slides.



Origin	Bradyarrhythmia	Tachyarrhythmia
SA node	- Sinus Bradycardia - Sick Sinus Syndrome	- Sinus Tachycardia
Atria		<ul> <li>Atrial Premature Beats - Atrial Flutter</li> <li>Atrial Fibrillation</li> <li>Paroxysmal SVT</li> <li>Multifocal Atrial Tachycardia</li> </ul>
AV node	- Conduction Blocks (1st,2nd, 3rd) - Junctional escape rhythm	-
Ventricles	- Ventricular Escape Rhythm	<ul><li>Ventricular Premature Beats</li><li>Ventricular Tachycardia</li><li>Torsades de pointes</li><li>Ventricular Fibrillation</li></ul>



## Differential Diagnosis of Tachycardia

This table is only female slides and in our opinion it's not important to memorize all.Because some are not even mentioned in next slides.



Tachycardia	Narrow Complex	Wide Complex
Regular	<ul><li>Sinus Tachycardia</li><li>Supraventricular Tachycardia</li><li>Atrial Flutter</li></ul>	<ul> <li>Sinus Tachycardia with aberrancy</li> <li>Supraventricular Tachycardia with aberrancy</li> <li>Ventricular Tachycardia</li> </ul>
Irregular	<ul> <li>Atrial Fibrillation</li> <li>Atrial Flutter with variable conduction</li> <li>Multifocal Atrial Tachycardia</li> </ul>	<ul> <li>Atrial Fibrillation with aberrancy</li> <li>Atrial Fibrillation with WPW</li> <li>Ventricular Tachycardia</li> </ul>



# 5

#### **Conduction Blocks**

 $SA \ node \rightarrow (block) \rightarrow Atrial \ Muscle = SA \ Block \ (no \ p \ wave)$ 

 $SA \ node \rightarrow Atrial \ Muscle \rightarrow AV \ node + His \ Purkinje \ system \rightarrow (block) \rightarrow Ventricle = Atrioventricular (AV) \ block (PR \ interval \ abnormal)$ 





#### **Abnormal Cardiac Rhythms that Result from Impulse Conduction Block**

	Sinoatrial (SA) Block	A-V (heart) Block
What is Blocked?	- The impulse from the S-A node is blocked before it enters the atrial muscle	When impulse from the AV node is blocked
Causes	- Ischemia of the node - Compression of the A-V node by Scar Formation - Inflammation of the A-V node (Aging) - Strong vagal stimulation	
Pictures	SA block  W  Nell Capton and Nell Tentrock of Medical Physiology, 12th Edition Copyright © 20.51 by Standards, an impart of Standard, an inspect of Standards.	-
Characteristics Cessation of P Wave		1st, 2nd, 3rd degree block



# 1st, 2nd & 3rd Degree A–V Block

			Y			
W			ECG Pattern			
A	/ Block	Description	P-R Interval Duration (Prolonged when > 0.2s or 1 big box)	Constant or Progressive P-R Interval?	Is a Drop Beat Present? (P wave not followed by QRS)	Picture
First Degree		1st Degree = Prolonged PR	Prolonged	Constant	Without Drop Beat	Ph Ph Ph Ph
Second	Mobitz (I) (Physiolog ical)	- 2nd degree = prolonged PR + Drop Beat - Drop Beat mechanism:	Prolonged	Progressive	With Drop Beat	P P P P P P
Degree	Mobitz (II) (Pathologi cal)	Only few impulses pass to the ventricles→ Atria beat faster than ventricles → "dropped beat" of the ventricles	Prolonged	Constant	With Drop Beat	-A A A A A A A A A A
Thir	d Degree	- The ventricle escape from the influence of S-A node.		Complete dissociation of P wave and QRS waves (Atrial Rate is 100 beats/min)   (Ventricular Rate is 40		- (P-P interval is constant)   (R-R interval is constant)   (no relationship between P waves and QRS complexes)
Tilliu Degree		- Pathological Condition Related : Stokes-Adams Syndrome : AV Block comes and goes.	beats/min)		intricular Nate 15 40	P P P P P P P P P

# 6

## **Premature Contractions (PACs/PVCs)**



## **Premature Contractions (Ectopic beat)**

What's it?

**Results from?** 

Originate in?

Causes?



Contractions that occur early before the next normal sinus impulse (before P wave send the impulse out), thus we'll see QRS complex occurs prior to the next expected heat



(Extrasystoles) or ectopic beat result from ectopic foci that generate abnormal cardiac impulses (Pulse deficit). Ectopic foci can cause premature contractions that originate in: The atria (but not in SA node), A-V junction

Ischemia,
Irritation of
cardiac muscle
by calcified foci
Drugs like





## **Premature Contractions (Ectopic beat)**

	Premature Contractions	PACs (Premature Atrial Contractions)	PVCs (Premature Ventricular Contractions) Causes: Drugs, Caffeine, Smoking, Lack of Sleep, Emotional Irritations.
	QRS Complex	Normal Narrow QRS because impulse went through AV node → Bundle of HIS →Purkinje system	<ul> <li>Prolonged QRS Complex:</li> <li>because the impulses are carried out with myocardial fibers with slower conduction rate than Purkinje fibers.</li> <li>Increase QRS Complexes Voltage:</li> <li>because QRS wave from one ventricle can not neutralize the one from the other ventricle</li> </ul>
	Features	<ul> <li>Short P-R Interval: Depending on how far the ectopic foci from the AV node.</li> <li>Pulse Deficit: If there is no time for the ventricles to fill with blood.</li> <li>Compensatory Pause: The time between the premature contraction and the succeeding (next) beat is increased.</li> </ul>	T Wave has an electrical potential of opposite polarity of that of the QRS: because of the slow conduction in the myocardial fibers, the fibers that depolarizes first will Repolarize first.
ı	Picture	Premature beat	"*\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\

# 7

## Fibrillation / Flutter

Atrial Fibrillation is the most common arrhythmia



## **Atrial Fibrillation**

Cause / mechanism	Same mechanism as Ventricular Flbrillation - it can occur only in atria without affecting the ventricles - The Atria do not pump if they are fibrillating - The efficiency of ventricles pumping/filling is decreased 20-30%, thus the cardiac output will decrease 20-30%
Features	No P wave, or high frequency of low voltage P wave Irregularly irregular (means its irregularity doesn't have a pattern) Narrow QRS complex
Causes	It occurs more frequently in patients with enlarged heart
Treatment	DC shock





#### **Atrial Flutter**



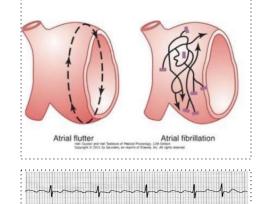
A single large wave travels around and around in the atria

(notice in the picture that atrial fibrillation has multiple impulses generated from different locations, while the atrial

flutter has only one that is large) (Reentry Circuit) (It's more organized than Atrial Fibrillation)

Atrial Rate: The Atria contracts at high rate (250 bpm/min)

 Amount of Blood: Because one area of the atria is contracted and another one is relaxed, the amount of blood pumped by the atria is slight
 Rhythm: The refractory period of the AV node causes 2-3 beats of atria for one single ventricular beat; 2:1 or 2:3 rhythm.





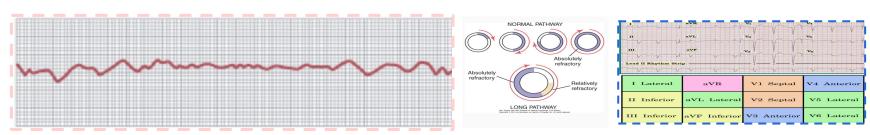




### **Ventricular Fibrillation**

#### The most serious of all arrhythmias

Cause	impulses stimulate one part of the ventricles, then another, then itself, many parts contract at the same time while other parts relax (circus movement)	
Features	Tachycardia, Irregular rhythm, Broad QRS complex, No P wave, low amplitudes, Chaotic and rapid rate	
Causes	Sudden Electrical Shock, Ischemia	
Treatment	DC shock	

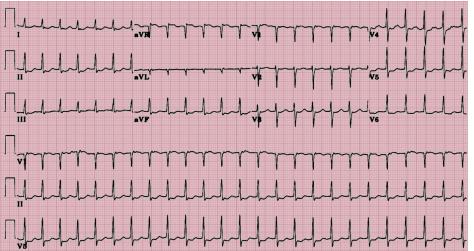


No p waves and the whole baseline is fibrillatory. Yes slides says its wide QRS, because it's a ventricular rhythm. But actually all the waves in VFib are indiscernible.

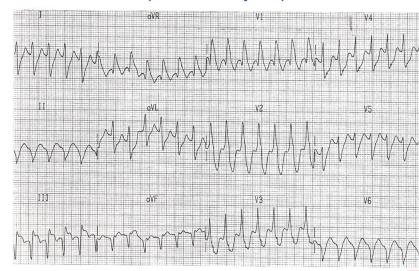


# SVT and VT

#### AVRT-Narrow Complex (Supraventricular Tachycardia - SVT)



## Ventricular Tachycardia (Wide Complex)



# 8

## Ischemia/Potassium & The ECG

This section in slides, but M&F doctors hinted it may not come. Still its mentioned in other lectures (Physiology) and it's not very hard to grasp the basics



## **Ischemia And The ECG**

Ischemia = Decreased blood flow to the heart.

It has two types:

1- Reversible: <b>Angina Pectoris</b>	2- Irreversible: Myocardial Infarction  • Complete loss of blood supply to the myocardium resulting in necrosis or death of tissue  • Ischemia -> Injury -> Infarction
Inverted T Wave - ST Segment depression	ST Segment Elevation - Deep Q Wave STEMI : ST-Elevation Myocardial

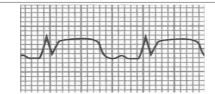
Infarction

One of the common uses of the ECG is in acute assessment of chest pain.



I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral







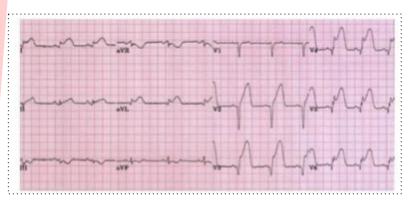
### **Ischemia And The ECG Cont..**



7:05 - 6:18

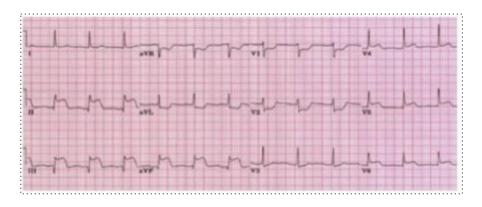


#### Antero-Lateral MI



- Occlusion of the Left Anterior Descending Artery (LAD)
- ST elevation in leads V2-V6, I, aVL
- Reciprocal ST depression in leads III, aVF

#### Infero-Posterior MI



- Occlusion of the Right Coronary Artery (RCA)
- ST elevation in leads II, III, aVF
- Reciprocal ST depression in aVL





### Potassium and the ECG

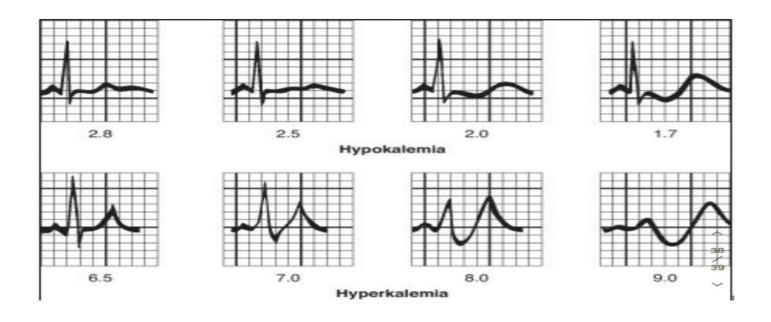


Hypokalemia:

- Flat T Wave

#### Hyperkalemia:

- Tall Peaked T Wave





# 8

## Questions

In a 12-Lead ECG the best lead to assess most things (rhythm, p wave, PR interval) is Lead II. so look at it first

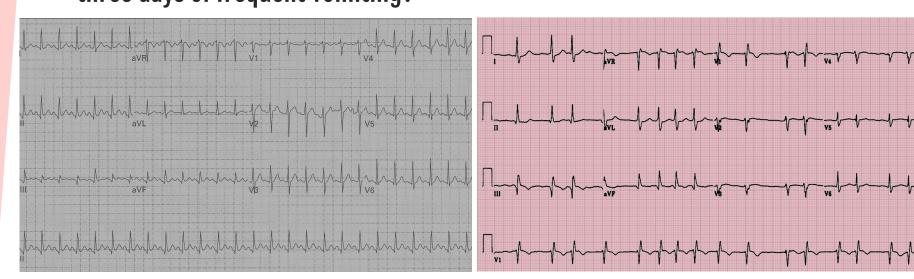


### MCQ



# 1) 24 year-old pregnant woman with three days of frequent vomiting?

#### 2) What do you see in this ECG?



A) SVT
B) VT
C) Sinus Tachycardia
D) Atrial Flutter

A) Atrial FlutterB) Atrial FibrillationC) SA BlockD) Sinus Tachycardia

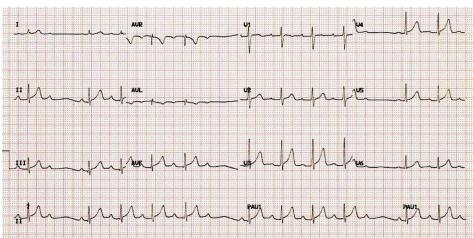




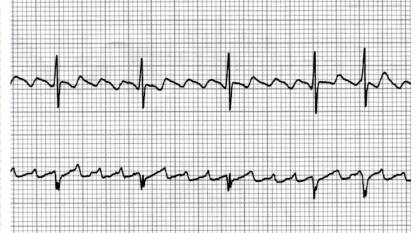
## MCQ



#### What do you see in this ECG



#### What do you see in this ECG?



- A) 1st degree AV Block
- B) 2nd degree AV Block
- C) 3rd degree AV Block
- D) Sinus Bradycardia

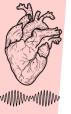
A) Atrial Flutter

B) Atrial Fibrillation

- C) PAC
- D) SVT







### SAQ

#### **Questions:**

Q1: Why does sinus arrhythmia occur?

**Q2: How is Atrial/Ventricular Fibrillation treated?** 

Q3: Premature Contractions (Ectopic beat) originate in?

Q4: give features of ventricular fibrillation?

#### **Answers:**

Due to the changes in autonomic tone during respiratory cycles

DC shock

Ectopic foci can cause premature contractions that originate in: The atria (but not in SA node), A-V junction, The ventricles.

Tachycardia, Irregular rhythm, Broad QRS complex, No P wave



#### **Meet Our Team**



#### • Team Leaders:

خالد ابراهيم العوهلي ميس احمد الطخيس

#### • Team Members:

عبدالعزيز بندر العنزي شادن راضي الشمري