

# \* How to report an ECG:

① patient's name, gender and age

② **Cardiac Rhythm**

**Normal**  
"SA rhythm"

\* Dep. begins in the SA and spreads in it's normal pathway

\* P wave prior to every QRS complex  
\* R-R Interval Regular all the pathway (70,12) sec

**Nodal**  
"AV rhythm"

\* Dep. begins elsewhere and named after the part where it starts.

② **check Heart Rate**

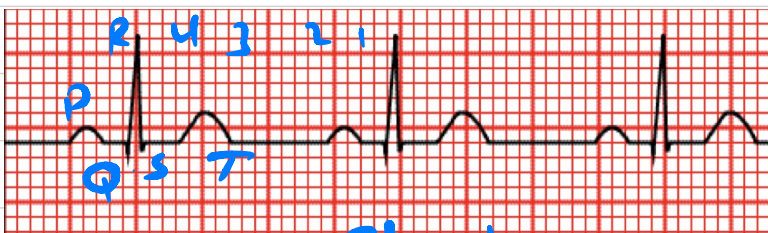
**Normal**  
(60-100) BPM

\* 300 / large squares In R-R Interval

**Ab-normal**

Tachycardia  
> 100 BPM

Bradycardia  
< 60 BPM



Sinus Rhythm

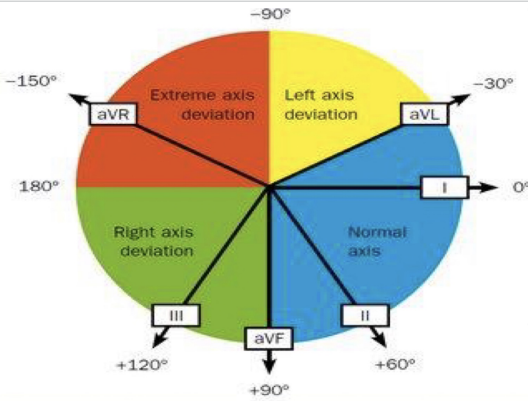
$$HR = \frac{300}{4} = 75 \text{ BPM}$$

$$\underline{\underline{R-R}} \rightarrow 0,04 * 20 = \underline{\underline{0,8}} \text{ normal}$$

④

## Cardiac axis

You can determine cardiac axis in many ways but best one if you have all chest leads by looking at (I and aVF) leads



\* Factors causing Axis deviation:-

### ① Normal QRS duration and Voltage:-

1. Left Angulation of the heart and Left axis:-

- ① Deep Expiration      ③ abdominal fat
- ② Lying down      ④ short and obese

2. Right Angulation of the heart and Right axis:-

- ① Deep Inspiration      ③ Thin and tall
- ② Standing up

### ② Ab-normal QRS duration and Voltage:-

(0,09 - 0,12) s

> 4 mV

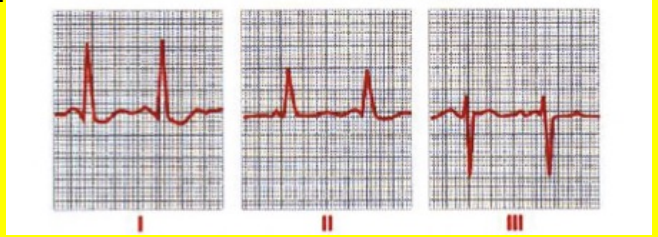
## Ventricular Hypertrophy:-

1. Hypertrophy of left ventricle (left axis shift) caused by hypertension, aortic stenosis or aortic regurgitation
2. Hypertrophy of right ventricle (right axis shift) caused by pulmonary hypertension, pulmonary valve stenosis, interventricular septal defect.

To find the voltage, look at leads I, II and III. Calculate how many small squares are taken up by both the R and S (R+S) for each of them, then add them all together, multiply the resulting number by how many mV a small square is worth.

Example:

- R+S for lead I: 16
- R+S for lead II: 10
- R+S for lead III: 18
- $16+10+18 = 44$
- Assume that each small square is worth 0.1 mV
- $44 \times 0.1\text{mV} = 4.4\text{mV}$



## Ab-Normal QRS duration and Normal voltage :- One Bundle branch block :-

1. Left bundle branch block causes left axis shift
2. Right bundle branch block causes right axis shift

## Description of P waves, QRS, T... :-

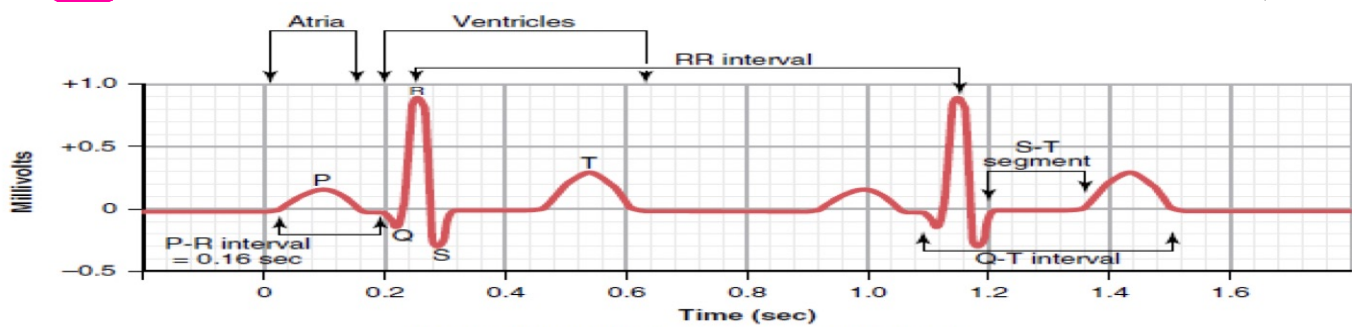


Figure 11-1 Normal electrocardiogram.

## \* Normal Conditions :-

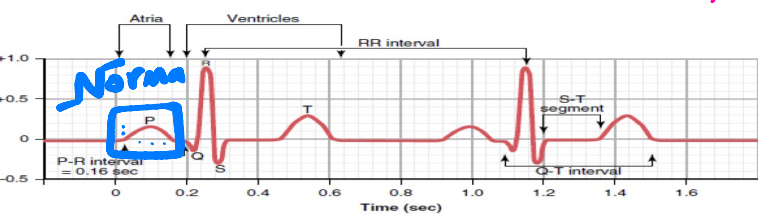
**(A) \* P-wave :- Atrial Depolarization.**

- \* Normally  $\oplus$ ve deflection
- \* maximum high = 2.5 mm

موسمى صغار  
و نوسا  
لغوى

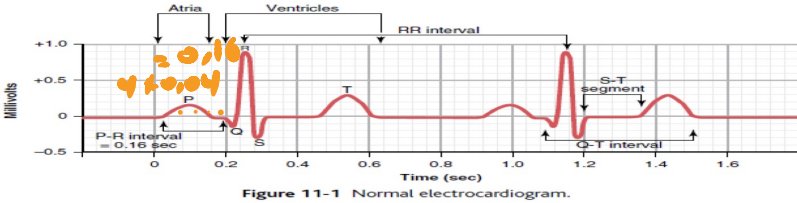
\* Duration  $< 0.12$

أقل من ٣  
مربعات صغار



**B** \* P-R Interval :- from beginning of P wave to beginning of QRS

\* Normally (0,12 - 0,20) من ٣ مريضان له ٥ مريضان صفا ، بالمرضين



$0,04 \times 4 = 0,16 \underline{\underline{N}}$

**C** \* QRS Complex :-  
 Ventricular depolarization  
 \* (0,06 - 0,10) sec  
 \* (0,5 - 2,0) mV ) Normally

\* In limb leads aVR usually  $\ominus$  deflection

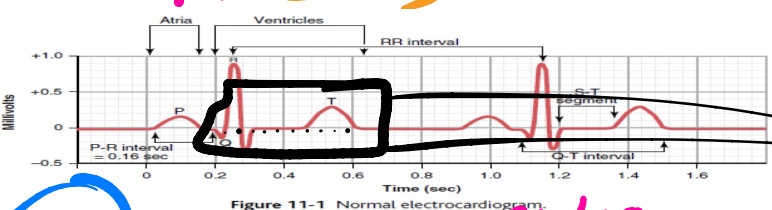
**D** \* T wave :- In normal adults, the T wave is usually upright in all leads, except the aVR and V1 leads

\* Rounded and Asymmetrically with Round peak

\* height of the T wave should not exceed 5 mm in limb leads and 10 mm in chest leads

**E** \* QT Interval :- Begining of QRS to End of T wave.

\* Less than 0,44 sec



أقل من ١١ ربع فيصد

$10 \times 0,04 = 0,4$

**Normal**

**F** \* ST segment :-

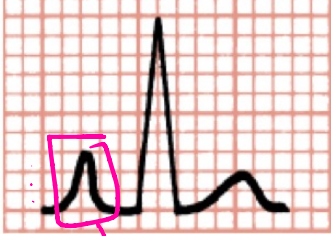
end of the QRS complex to the beginning of the T wave

# \* Ab-Normal Conditions

①

## P-Wave

### P-Pulmonale



\* Right Atrial Enlargement

آکل مرتا مرتین و پهن

\* Peaked, higher and narrower

آکل مرتا ۳ مربعان

### P-Mitrale



\* Left Atrial Enlargement

\* Notched and prolonged

آکل مرتا ۳ مربعان

②

## P-R Interval

Short

Long

Abnormally fast conduction from the atria to the ventricles

First degree heart block

③

## RS Complex

Increased width

low voltage

high voltage

Cardiac hypertrophy or dilatation  
• Bundle branch block

Old myocardial infarctions.  
• Pericardial or pleural effusion

Cardiac hypertrophy

4

# T-wave

## Inversion

- 1. Mild ischemia
- 2. Ventricular hypertrophy
- 3. Bundle Branch Block
- 4. Digoxin Toxicity
- 5. Normal finding in aVR & V1

## Peaked

- 1. Early stages of myocardial infarction
- 2. Hyperkalemia

## Flattened

- 1. Hypokalemia
- 2. Ischemia

5

# QT segment

Prolonged QT interval is seen in Long QT syndrome, hypokalemia, hypercalcemia & hypothyroidism.

6

# ST segment

Depressed or raised in ischemia or myocardial infarction

more than 1 mm of ST segment elevation/ depression in at least two contiguous limb leads (e.g. I and VL; III and VF)

more than 2 mm of ST segment elevation/depression in at least two contiguous chest leads

# # Arrhythmias: Abnormal heart Rhythm

1. Sinus Tachycardia
2. Sinus Bradycardia
3. Sinus arrhythmia
4. Sino-Atrial Block
5. Atrioventricular Block
6. Ventricular fibrillation
7. Re-Entry
8. Atrial fibrillation
9. Atrial flutter

First degree  
Second "  
Third "

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 the impulse through the heart
4. Abnormal pathways of impulse transmission through the heart
5. Spontaneous generation of spurious impulses in almost any part of the heart

## ① Sinus Tachycardia:

- \* Normal ECG components (Normal P, QRS, T)
- \* HR > 100 BPM



$$HR = \frac{300}{T/6} \approx 7100$$

## ② Sinus Bradycardia:

- \* Normal ECG
- \* HR < 60 BPM



$$HR = \frac{300}{8.5} = < 60 \text{ BPM}$$

## ③ Sinus Arrhythmia:



No P wave - BUT Normal ((QRS-T))

## ④ Sino-Atrial Block:



NO P WAVE  
SLOW HR

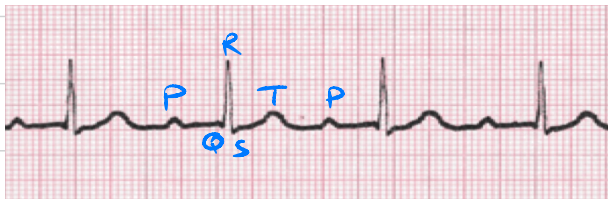
# ⑤ Atrio-ventricular Block:-

- \* Slowed or Blocked Atria → Ventricul
- \* pro-longed P-R Interval
- \* Can cause no P wave conduction.

① First degree:-  $PR > 0,20$

② Each P wave → give QRS Complex

③ Caused by coronary artery disease, acute rheumatic carditis, digoxin toxicity or electrolyte disturbances



PR Interval =  $0,04 \times 7 = 0,28$  prolonged

HR =  $\frac{300}{4,5}$  Normal

④ Second degree:- **Wenckebach**  
**Fixed Ratio**

- \* prolonged P-R (0,25-0,45) sec
- \* action potential is sometimes strong enough to pass through the bundle into the ventricles and sometimes not strong enough to do so

\* Dropped Beats:- P ✓ QRS X

**Wenckebach periodicity**



Tip:-

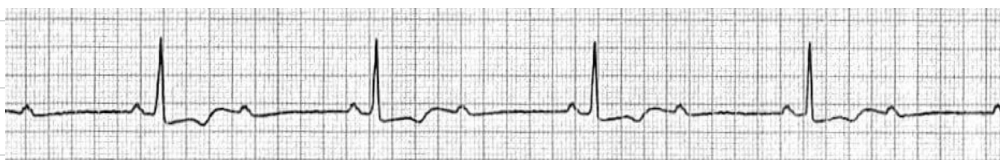
Long Period

... Dropped ... Short Period

Then it Wenckebach

**A** Fixed ratio blocks usually a fixed number of non-conducted P waves for every QRS complex

\* Ab-normality in bundle of His-Purkinj





## ③ Third degree = Complete Block:-

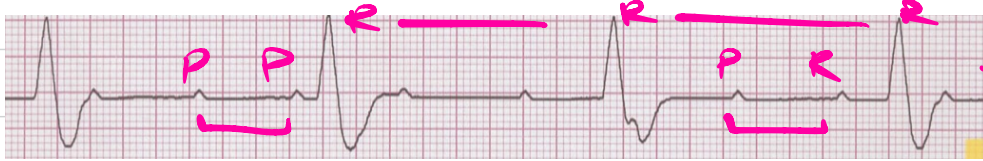
x Total Block

x R-R Intervals are Regular

x P-P " " " "

make it different from first degree-

x Atrial faster than ventricle



⇒ NOTICE THE REGULARITY.

## ⑥ Adams Stroke Syndrome:-

Ventricles stop contracting for 5-30 sec because of overdrive suppression meaning they are used to atrial drive.

⇒

□ Patient faints because of poor cerebral blood flow

## ⑦ Ventricule fibrillation:-

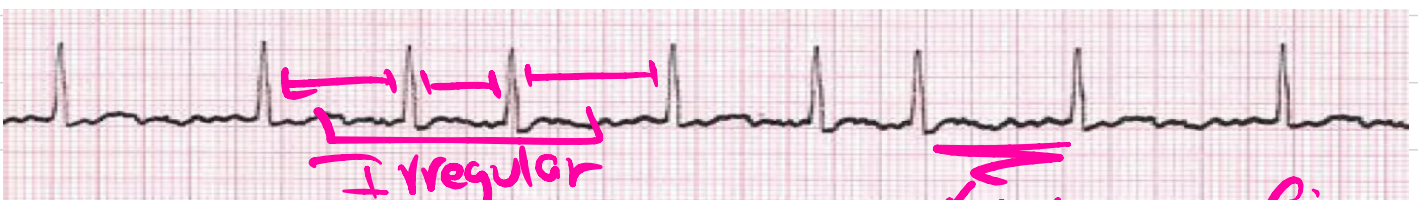
Disorganized rhythm with no identifiable waves.



No discernible rhythm

## ⑧ Atrial fibrillation:-

no P waves are seen or only a fine, high frequency, very low voltage wavy record. The QRS complexes are normal in shape but are irregular



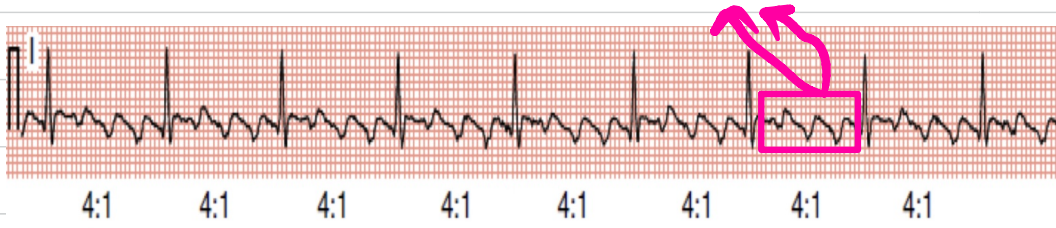
Irregular R-R Intervals

NO or fine P wave

# ⑨ Atrial flutter

Atrial rate is around 300 bpm (200-400)

P waves are strong (saw tooth appearance)

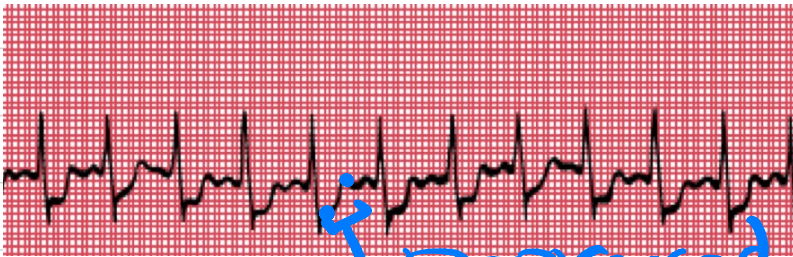


# # Angina Pectoris

noticed while the patient is in pain, once the pain has resolved the ECG returns to normal.

Depression of the ST segment, is usually a sign of ischaemia.

Rem!! ← End of QRS to start of T



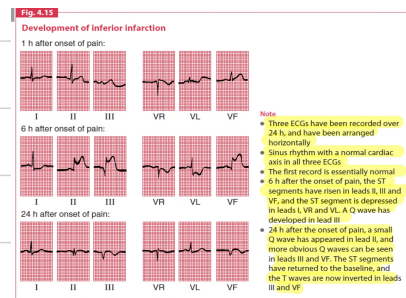
Depressed (Notice here)!!

## # ST segment elevation myocardial infarction (STEMI)

- ① The earliest ECG changes are ST segment elevations
- ② hyperacute T waves

③ Large and transmural MI might produce permanent pathological Q waves within the first day of infarction

④ Hours or days later ST segments return to the baseline and deep T wave inversions appear in the leads that previously showed ST elevations.



## # Non-ST segment elevation myocardial infarction (NSTEMI)

Associated with ST segment depression and wave inversion in the leads corresponding to the site of myocardial damage

	NSTEMI	STEMI
INFARCT LOCATION	Subendocardial	Transmural
LAYERS INVOLVED	Subendocardium (inner 1/3) especially vulnerable to ischaemia	Full thickness of myocardial wall
ECG CHANGES	ST-segment depression, T-wave inversion	ST-segment elevation, pathologic Q waves