# Rapid Review of Clinical ECG

**Saleh Mohammed AL-awdhaly**

**M.B.Ch.B, F.I.C.M.S, C.A.M.B**

**A.Prof. faculty of medicine**

**Thamar University**

**First Edition**

**2008**

# Author

**Saleh Mohammed Al-awdhaly**

**Assist, Prof. Consultant physician, department of medicine, faculty of medicine**

**Thamar University**

# Dedication

**To my colleagues**

**To my students.**

**To those who helped me and for the hours they spent at the ECG drafts and computer that made this work possible,**

**"Thank you" Never seems enough**

## Preface

**The aim of this book (rapid review of clinical electrocardiography)is to provide medical students and general Practitioners some of the basic practical skills in electrocardiography. We advise students to be involved directly with the patients because practical skills in the ECG can not be learned from only the book.**

**We insist once more that our medical students and practitioners should be systemically interpret ECG paper and also with close relation to the patient's history and examination and other laboratory investigations.**

**Author**

## Reviewers

**Dr.Fathi Maklady (FRCP) (UK) (Suez Canal University) Egypt**

**Dr.Mohamed Reda Bassiouny (MD, MHPE) (Mansoura University) Egypt**

**Dr. Yahya Alezzy (MD) (Sana'a University)**

**Dr.Dhaifallah Geed (PhD)**

**Dr. Amin Abdelrab (MD)**

**Dr. Motea Alaolaqi. (MD)**

**Dr. Mohammed Aldholae (MD)**

**Dr. Ahmed Ali Kaied (PhD)**

**Dr. Ahmed. S. Almansoob (MD) (Aden University)**

**Dr. Yassen. A. Yassen (MD) (Aden University)**

**Dr. Ahmed. A. ALshahari (MD)**

**Dr. Mohammed Moustafa**

**Dr. Anwar M. Othman (M.B.Ch.B)**

**Dr. Abdullah Almortatha (M.B.Ch.B)**

**Dr.Abduh Alsherabi (M.B.Ch.B)**

**Dr. Salah Alshoki (M.B.Ch.B)**

I Reviewed this book thoroughly. it is a very good effort. well structured. very basic and take the subject in a sequential way. Easy to understand and Comprehend. takked all aspects of E.C.G. basic and clinical. I recommend this book to medical students, High nursing schools. and post graduate students of Internal medicine. I Congratulate Dr. Saleh. for this basic standard Book.

F. MAKLady
21/11/2007

**Dr.Fathi Maklady (MBBCH, DM, FRCP) (UK)**

**Professor of cardiology**

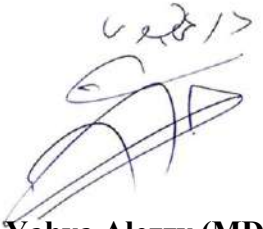
**(Suez Canal University) Egypt**

An excellent work essential for postgraduate teaching. Paediatric ECG differs from adult in some aspects that need to be highlighted in a separate chapter.
The book covers basic concepts and practical aspects.

M R Bassiouny
Prof of Pediatrics, Mansoura University

**Dr.Mohamed Reda Bassiouny (MD, MHPE)**
**Professor of pediatrics/ Neonatology**
**(Mansoura University) Egypt**

I reviewed this book and I found it's anew add to scintific product.
And Would help students Under and postgraduate.



**Ass. Prof. Yahya Alezzy (MD)**
**(Sana'a University)**

**Editors**

***Dr. Saleh Mohammed Al-awdhaly. (F.I.C.M.S, C.A.M.B)***

***Dr. Waleed Ismail M. Althahbi. (M.B.Ch.B)***

***Dr. Ibraheem Algubani (M.B.Ch.B)***

***Senior. Haytham  Ahmed Almalahy.***

***Printed by***

***Senior. Hamed Mohammed Al-selah.***

**Acknowledgments**

**I am grateful for prof. Ahmed M. AL-Hadrani F.R.C.S."Galaxo"**

**And prof. Amin A. AL-Hemyeri Ph.D . for encouraging me to complete the task of writing this book.**

**Many colleagues encouraged and provided me with constructive comments. It would be difficult to try to name them all.**

**Finally, I am grateful to my students who taught me how to teach, and to my family, on whose time this book was written.**

## Abbreviation

**The following abbreviation are some times will be used throughout the text without further explanation:**

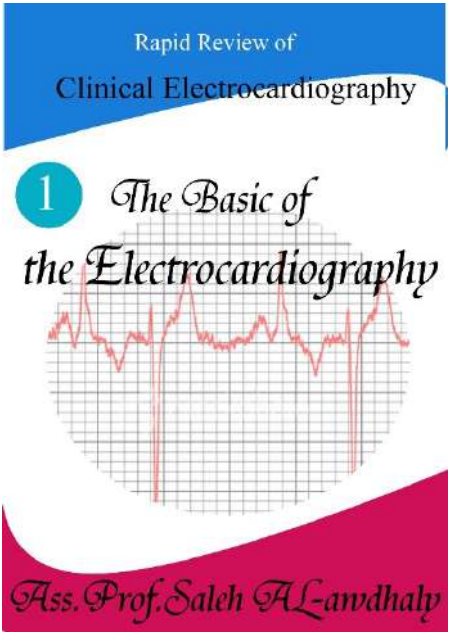
**AF = Atrial Fibrillation.**
**APB = Atrial Premature Beat.**
**A-V = Atrioventricular.**
**BBB = Bundle Branch Block.**
**ECG = Electrocardiography.**
**LAA = Left Atrial Abnormality.**
**LAD = Left Axis Deviation.**
**LAFB = Left Anterior Fascicular Block.**
**LBBB = Left Bundle Branch Block.**
**LPFB = Left Posterior Fascicular Block.**
**LV = Left Ventricle.**
**LVH = Left Ventricular Hypertrophy.**
**MAT = Multifocal Atrial Tachycardia.**
**MI = Myocardial Infarction.**
**MM = Millimeter.**
**MV = Millivolt.**
**RAA = Right Atrial Abnormality.**
**RAD = Right Axis Deviation.**
**RBBB = Right Bundle Branch Block.**
**RV = Right Ventricle.**
**RVH = Right Ventricular Hypertrophy.**
**S = Second.**
**S-A = Sino-Atrial.**
**VF = Ventricular Fibrillation.**
**VPB = Ventricular Premature Beat.**
**VT = Ventricular Tachycardia.**
**WPW = Wolf-Parkinson-White.**
**CAD = Coronary Artery disease.**
**IHD = Ischemic Heart disease.**
**LGL = Lown-Ganong-Levine syndrome.**
**AVNRT = Atrioventricular Nodal re-entery Tachycardia.**
**AVRT = Atrioventricular re-entry Tachycardia**

## Contents

**Chapter 1**

**Basic of ECG**

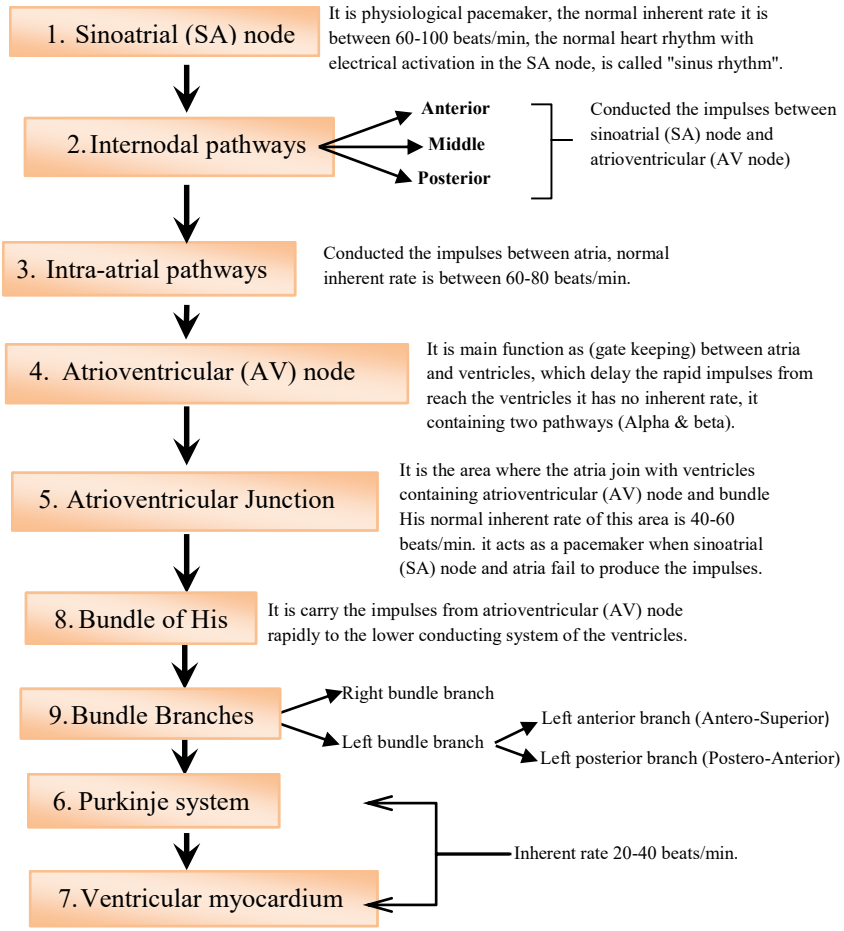
`



# The Basic of Electrocardiography

ECG definition: - is a graphic reading of electrical potentials generating by the heart.

**Electrical conductions system of the heart.** **(Fig.1-1)**



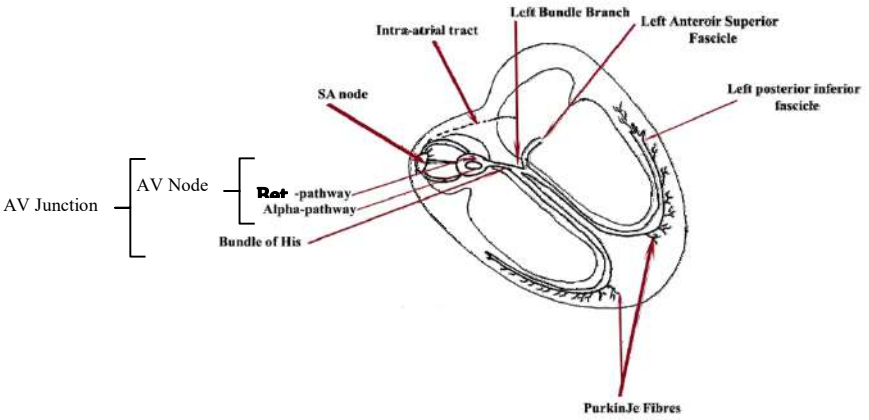



Fig (1-1) the pathway of the conduction

**The backup pacemaker sites and their inherent rates summarized in the following.**

1. SA node: 60-100 beats/min.
2. Atrial sites: 60-80 beats/min.
3. AV junction: 40-60 beats/min.
4. Ventricular sites (Purkinje system): 20-40 beats/min.

## Normal Sequence of Cardiac Depolarization and Repolarization and Consequent QRS complexes

The entire cycle that produces the contraction of myocardium tissue can be divided into three phases of electrical activity as the following:-

1. During resting state (polarized state), the cardiac muscle has negative charge inside and positive charge outside. (Fig.1-2A)

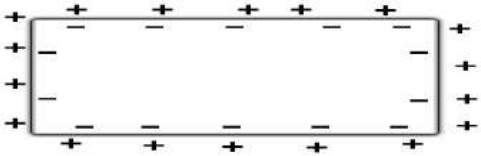
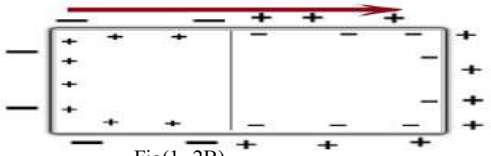



Fig (1-2A)

2. When the cardiac muscle is stimulated by electrical current, its charge becomes reveres, i.e. positive inside and negative outside, this change is called (depolarization). (Fig.1-2B)




Fig(1- 2B)

3. When wave of excitation is over again, there is reversal to original state there is negative inside and positive outside the cardiac muscle. This process of reversal to resting state is called (repolarization). (Fig.1-2C)

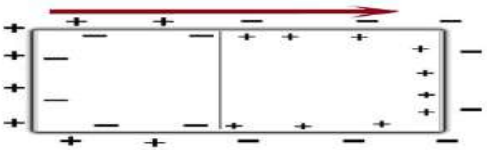
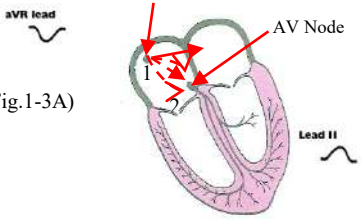



Fig (1-2c)

**Spread of the impulses from the atria to the ventricles** (step 1, 2).

Step 1, Impulses are initiated in the SA node and activate the atrium almost simultaneously by electrical potential, which represents by electrical vector that directed downward to the left produces positive upward deflection (P wave) in the leads lie with the same direction of the current flow, e.g. lead II and negative deflection (inverted P wave) in the leads lie in opposite direction of current flow, e.g. aVR. (Fig.1-3A)

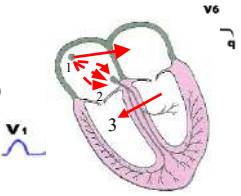



(Fig.1-3A)

Step 2, Next step the impulses pass slowly

through the AV node, introduce a delay

so that the ventricular depolarization is delayed. (Fig1-3B)




(Fig.1-3B)

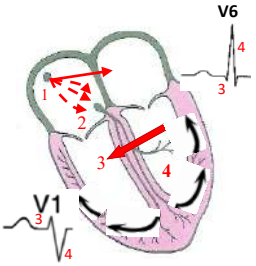
PR interval

The period from the beginning of the P wave to the end of this delay is known as PR interval.

**Ventricular depolarization (step 3 and 4)**

step 3, After brief delay of the impulses at AV node it spreads through common bundle of His and right and left bundle branches then enter interventricular septum, causing myocardial depolarization, activation of the septum occurs from the left to the right, this results in small negative (downward) deflection in lead V6 (septal Q wave) and positive (upward) deflection in lead V1 (septal R wave). (Fig.1-3C & Fig.5-1A)

(Fig.1-3C)




Step 4, The ventricular depolarization starts form endocardial surface and spreads outward from inside (from endocardial to the epicardial surface) as the left ventricle has large muscle mass than the right (and has a larger potential electrical force) and both ventricles activated simultaneously, the larger left ventricle force counteract the smaller right ventricle.

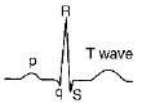
The result is a single force from right to the left, causes large positive (upward) deflection (R wave) in the left ventricle leads which lies in the same direction of current flow e.g. V5- V6. And negative deflection (downward) S wave in right ventricle leads e.g. V1, V2.(Fig.1-3C & Fig.5-1B)

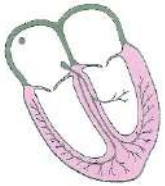
The relative increase in the size of the R wave toward the left precordial leads is called normal R wave progression

The lead aVR lies opposite the direction of the current flow, then the QRS complex is completely negative in this lead.

**Ventricular repolarization (step 5)** -

Step 5, when the heart is fully depolarized, there is no electrical activity for brief period (ST segment) the repolarization starts in the epicardium toward endocardium, producing electrical vector (axis) directed downward and to the left, causing upward (positive) deflection (T wave). A period of electrical activity follows, with tracing at baseline until next impulse originate again at SA node. See (Fig 1-3D )



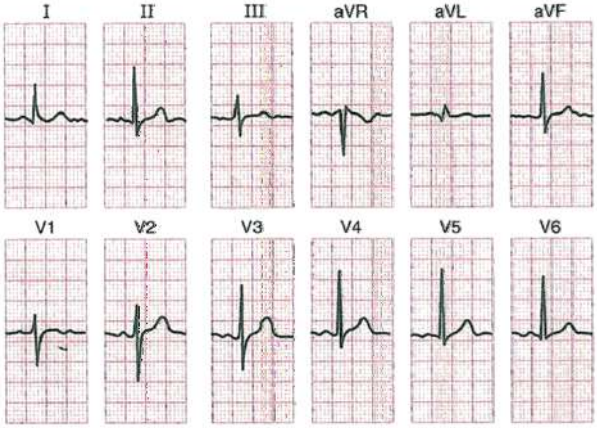



(Fig 1-3D )

Electrical current generated during repolarization of the ventricles are reflected in the ST system and the T wave

## The end result of the above mentioned steps (step 6)

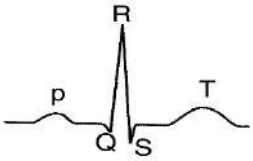
Step 6, The normal 12 lead electrocardiography is the end result of atrial depolarization and ventricular depolarization repolarization sequence.(see Fig.




(Fig. 1-3E) Normal 12-lead electrocardiogram. Note the progression in the upright deflection from r over the right ventricle (V1) to an 'R' over the left ventricle (V6).

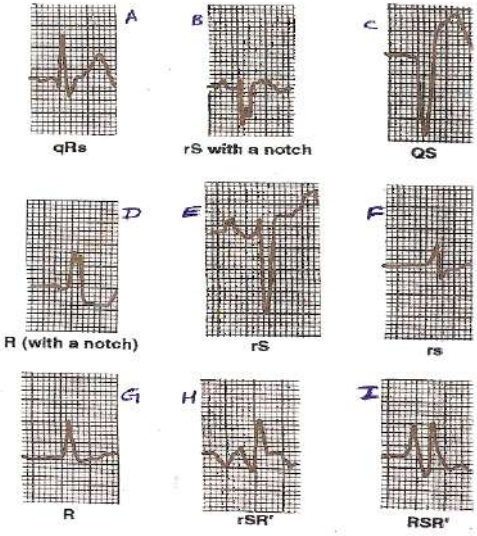
## The shape of the ECG

- The muscle mass of the atria is small compared with that of the ventricles, and the electrical changes accompanying the contraction of the atria is therefore small wave on ECG called "P". but the ventricles depolarization produces a large deflection of the ECG called "QRS" complex. The T wave of the ECG is associated with the ventricular repolarization. The basic shape of the normal ECG is shown in (figure.1-4) and the different parts of the QRS complex are shown in (fig.1-5).




(Fig 1-4) basic shape

In the (Fig.1-5 ) the waves are termed small q, r, s or capital (larger) letter QRS according to their amplitude (height) occasionally the QRS complex contains more than one deflection of the same wave, in such case the extra wave are called R (R˜ prime wave) as shown in (Fig.1-5 H & I)




(Fig 1-5 ) different parts of the QRS complex

**ECG paper**

1. ***Horizontal measurement:*** the Horizontal measurements represents the time.(Fig.1-6A)

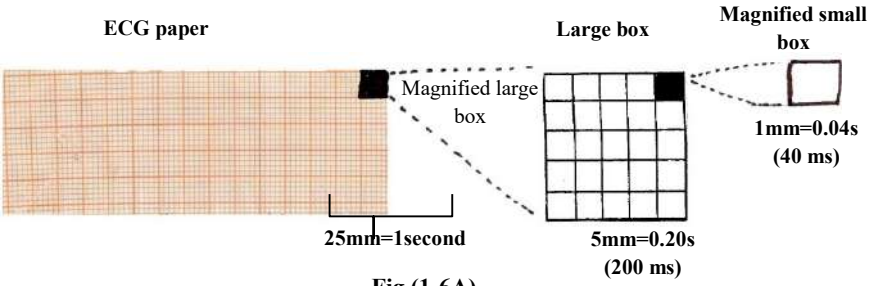


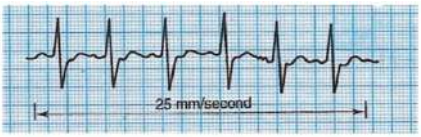

**Fig (1-6A)**

A- ECG paper composed of number of small boxes, each small box equal to 1mm wide=0.04 second= 40 milliseconds =1/25 second.

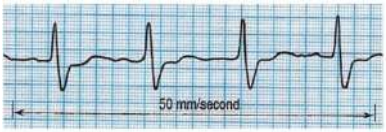
B- Each 5 small boxes (5mm) wide represents 0.20 second (i.e. 5× 0.04=0.20 seconds) = one large box in horizontal measurements.

C- Each five large boxes (25mm) wide represents 1 second (which means 300 big boxes/minute, 5× 60=300 or 1500 small boxes/minute, 25× 60=1500 small box/minute).

D- The ECG paper routinely moves through the machine at constant speed of 25mm/second. The paper speed can be increase to 50mm/second or slower to 12.5mm/second with some types of monitoring machine. Any change in the speed of the paper should be noted because it will change the basic pattern of the ECG.(see Figs.1-6B & C for normal and abnormal speed respectively)



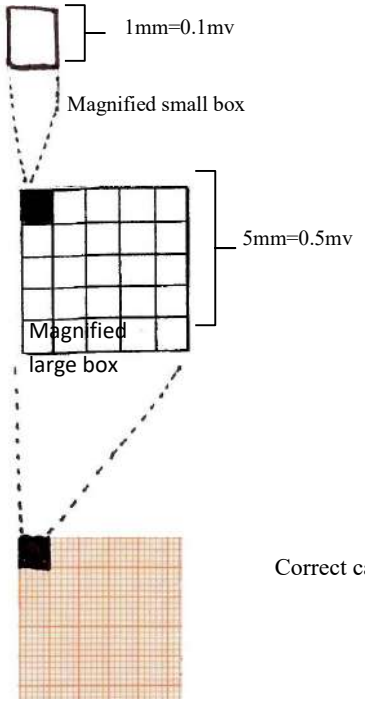

(Fig 1-6B) normal paper speed i.e. 25mm/sec




(Fig 1-6C) incorrect paper speed
Waves are abnormally wide at higher paper speeds

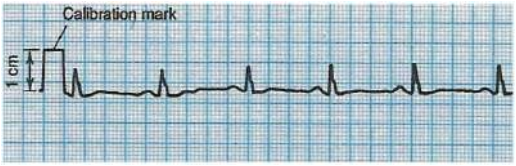
## 2. *Vertical measurements* (Figs. 1-7 A&B)

The vertical axis measurement on ECG paper represents voltage.



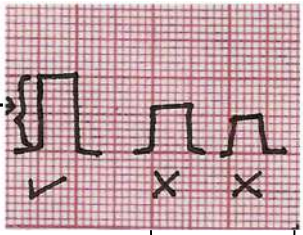

ECG paper (Fig 1-7A)

A- Vertically, the small box represents the amount of electrical potential of 0.1mv=1mm.
B- Each large box 5mm represents 0.5mv.
C- Machine is standardized in such manner that an impulse of 1 millivolt will cause deflection of 10 small box=10mm=two large boxes = 1cm.




Correct calibration
Note 1cm calibration mark
1mV=1 cm

Correct calibration



**(Fig 1-7B)**

Incorrect calibration

**How to Calculate Heart Rate**

***Any of several methods can be used to calculate the heart rate.***

1. **When the heart rhythm is regular (sinus rhythm)**

   A- Count the number of large boxes between two consecutive R waves and divided into 300. As already has been discussed ECG paper moves through the machine at a constant speed of 25mm/second, i.e. 25 small boxes (5 large boxes/second) minute is 60 seconds= 60 × 5= 300 large boxes/minute. (Fig.1-8A)

   Thus, we know for example, that if there is a QRS complexes every 2nd large box, the heart rate would be 300/2=150 beats/min.

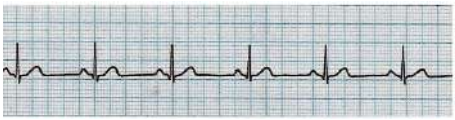
   - ***If there are two big squares, the heart rate will be 150/min.***
   - ***If there are three big squares, the heart rate will be 100/min.***

   B- Count the number of small boxes between two consecutive R waves and divided into 1500.

   As already discussed above the rate of ECG paper is 25mm/sec, i.e. 25 small boxes (5 large squares/second).

   In one minute 60 × 25=1500mm/1minute (i.e. 1500 small boxes/min)

   For example if there is 25 small boxes between two R peaks the heart rate will be 1500/25=60 beats/min. (Fig.1-8A)



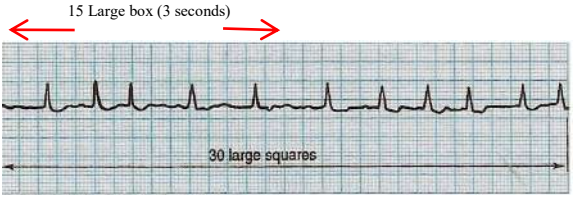
(Fig 1-8A) calculating heart rate when the rhythm is regular.

2. **When the heart rhythm is irregular or the QRS complex will be at variable distances. So, we calculate the heart rate as follows:**

A- Count the QRS complexes falling in 30 large squares and multiply it with (10). This will give the heart rate/min.

Or

B- Count the QRS complexes falling in 15 large squares and multiply it with (20) this will give the heart rate/min.(Fig.1-8B)




(Fig 1-8B) calculating heart rate when the rhythm is irregular
Key point -30 large squares contain 11QRS complexes.
So the heart rate 11×10= 110 beats /minute

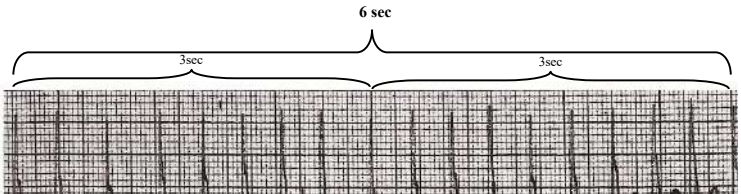
C- The ECG is usually and conventionally recorded at a paper speed of 25mm per second (i.e. 25 small box=5 large boxes) per second, and fifteen large box represent 3 seconds and thirteen large box represent 6 seconds. (see Fig.1-8B)

Most recording paper has every fifteenth large box a 3 second interval marked with vertical line on the upper border (see fig.1-8C) so we calculate the heart rate as follow:

1. Count the number of QRS per 3 seconds interval, then simply multiply the result by 20 to get the number of beats per minute.

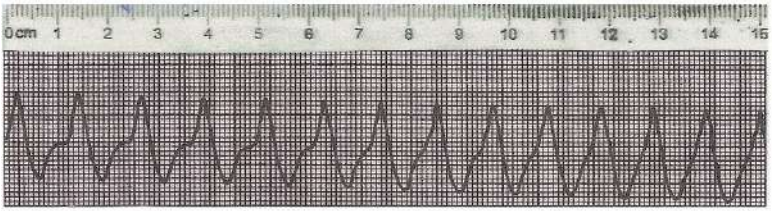
   Or

2. Count the number of QRS per 6 seconds interval (i.e. in two-3 seconds interval) then simply multiply the result by 10 to get the number of beats per minute.



(Fig 1-8C) When the rhythm is irregular, heart rate can be determined by counting the R waves in a 6-second strip and multiplying by 10. (paper speed is 25 mm/sec)
In this example 6 second contain 18 QRS complexes so the Heart rate is 18×10= 180 bpm & 3 sec contain 9 QRS complexes so the heart rate is 9×20 = 180 bpm.

D- The speed of ECG paper is 25mm/second =2.5cm/sec, the 6 seconds =2.5× 6=150mm=15cm, so count the number of QRS complexes per 15 cm interval, then multiply the result by 10 to get the number of beats per minute. (see Fig.1-8D)

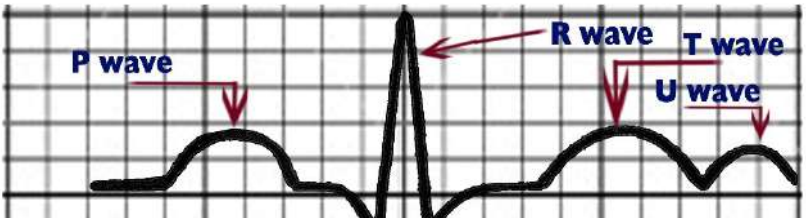



(Fig 1-8D) Heart rate can be determined by counting the R waves in 15 cm strip and multiplying by 10 in this example 15 cm contain 14 QRS complexes so the heart rate is 14×10 = 140 bpm

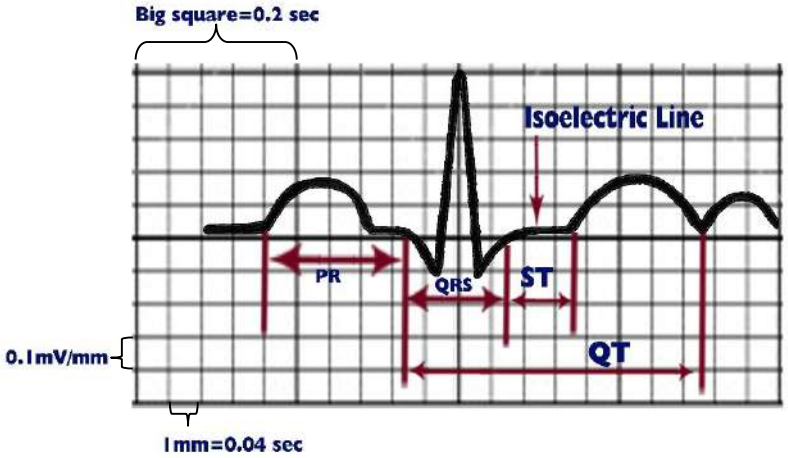
***Practical points***

1. If the RR interval is less than three large squares, the rate generally is over 100 per minute.
2. If the RR interval is more than five large squares, the rate generally is less than 60 per minute.

## Electrocardiography, Waves, Interval and Segments.

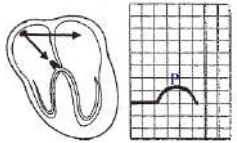
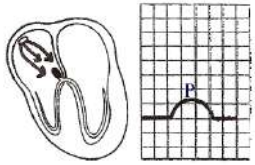


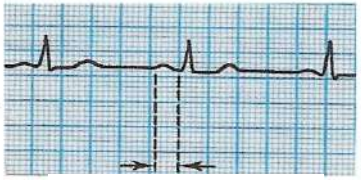
(Fig 1-9A) waves of the ECG




(Fig 1-9B) ECG Intervals and Segments

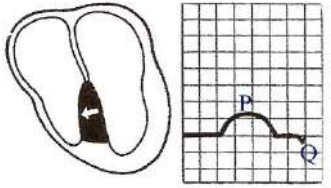
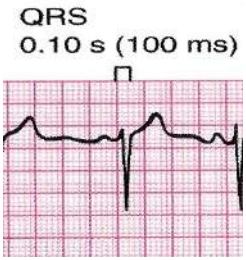
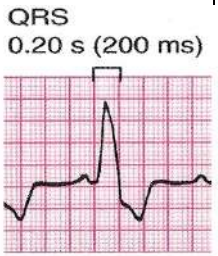
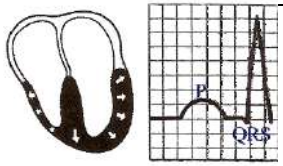
# Electrocardiography, Waves, Interval and Segments. (See Figs.1-9A&B)

| | Duration | Amplitude | Events in the heart | |
|---|---|---|---|---|
| P wave | Two and one-half small boxes. 0.10 second (2.5mm)<br><br>0.10 second = 100 millisecond | Two and one-half small boxes (2.5mm) | Atrial depolarization |  <br>P wave represents atrial depolarization |
| PR interval | From the beginning of P wave to beginning of QRS complex<br><br>The normal PR interval is range from 0.12 to 0.20 second (three to five small boxes) | PR segment usually isoelectric | Represents the time interval taken by an impulses to pass from the SA node to ventricular muscle. |  <br>during the PR segment also His-purkinje system is being activated, this is another silent event, not seen on the ECG |

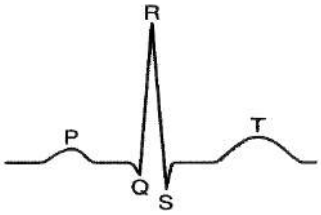
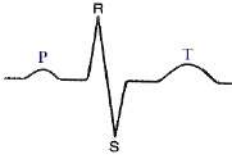
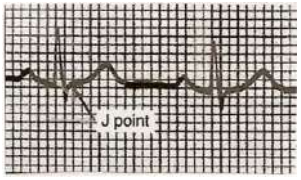



the PR interval

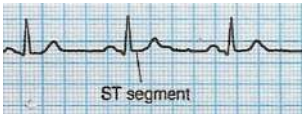
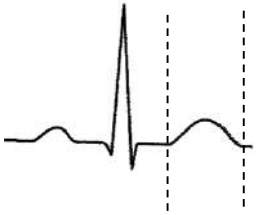
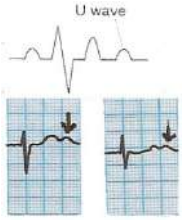
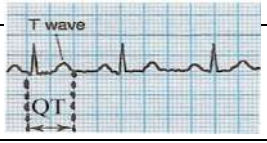
Continue ⟶

| | | | | |
|---|---|---|---|---|
| **Q wave** | **Less than one small box (<0.04 second) = 0.04 second = 40 MS** | **Less than 25% of R wave height in the same lead** | **First negative deflection represents of the septal activation (when recorded from the left precordial lead)** |  **septal activation from the left to the right produces a small, narrow, negative deflection (Q wave) in V5-V6 (septal Q wave)** |
| **QRS** | **Beginning of QRS complex to J point where QRS joint ST segment. Less than two and one-half small boxes (<0.10 sec)**  **normal QRS**  **Wide QRS** | | **Ventricular depolarization** |  **depolarization of the ventricles, mainly left ventricle produces a tall positive deflection in the left ventricular leads** |

Continue ⟶

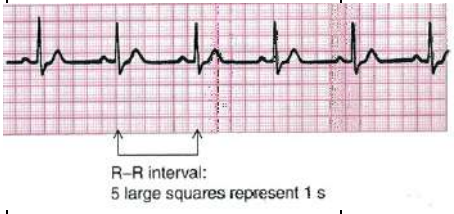
| | Amplitude | Events in the Heart | |
|---|---|---|---|
| **R wave** | **A-Upper limit for R wave amplitude: In leads V4, V5, V6: 25mm or may be 30 mm in young adult**<br><br>**B-Lower limit for R wave amplitude in lead I, II, III: 6mm (total value of negative and positive components) below 5mm in the three standard leads (i.e. below of 15mm in all of these standard limb leads) consider as low voltage QRS complex** | **First positive deflection of QRS**<br><br>**Its represents depolarization of the ventricles, the height is larger depending on the much thicker muscle of the left ventricle (R wave)** |  |
| **S wave** | **A-Upper limit for S wave amplitude in V1-V2: 26mm**<br><br>**B-Lower limit for S wave amplitude in V1: 3mm** | **The second negative deflection follow R wave.** |  |
| **J point** | **The J point defines the end of the QRS complex and begging of the ST segment.**<br><br>**A J wave may seen in the following conditions.**<br><br>**1.Hypothermia**<br><br>**2.Hypercalcaemia**<br><br>**3.Early repolarization** | |  <br>**the J point** |

Continue ⟶

| | Duration | Amplitude | Events in the heart | |
|---|---|---|---|---|
| **ST segment** | **The interval between the end of the QRS complex and the beginning of the T wave** | **Isoelectric (i.e. neither positive not negative)**<br><br>**Normally slightly elevated or depressed but not more than 1mm** | **It is may represents the pause in ventricular activity before repolarization** |  <br>**The ST segment** |
| **T wave** | | **1. In the standard limb leads less than 6mm (less than 6 small boxes).**<br><br>**2. In the chest leads it is also less than 6mm in women but may be taller in men including in V2-V3, but usually not more than 12mm (12 small boxes).** | **Represents the recovery (repolarization) of the myocardium.** |  |
| **U wave** | **A wave on the ECG that follows the T wave** | **In clinical practice prominent U wave is sign of:**<br><br>**Hypokalaemia, Drug toxicity (quinidine), Cerebrovascular accident, Hypercalcaemia, Hyperthyroidism.** | **Represents the last phase of the ventricular repolarization** |  |
| **QT interval** | **For the guidance of the beginning ECG reader, one might generalize that with** | | **Represents the total ventricular depolarization and repolarization** |  |



Continue ⟶

| | | | | |
|---|---|---|---|---|
| | **normal heart rate, 60-100/minute, QT interval is 0.30-0.43sec.**<br><br>**QT interval extends from the beginning of the QRS complex to the end of T wave and not the U wave (if present).** | | | **the T wave an QT interval**<br><br>**The faster the heart rate, the shorter the QT interval, it is essential to correct the QT interval to decide whether its normal or not, use the following formula:-**<br><br>$QTc = \frac{QT}{\sqrt{RR}}$<br>**This Bazett's formula** |
| **RR interval** | **Is the time between two consecutive R waves and be either measured directly from ECG or calculated by dividing 60 by the patient heart rate example at the heart rate 80 beats.min the RR interval is 60/80 =0.75 seconds** | | **RR interval is determined the ventricular rate on the ECG**<br><br>  <br> | |

# ECG Leads

**A standard ECG is composed of six limb and six chest leads.**

## Limb leads

These are recorded by placing electrodes on the right, left arms and left leg respectively.

A- **Bipolar (standard) leads (I, II, III):** called bipolar because it records the electrical potential difference, between voltages from the heart detected at two extremities (a positive and a negative). (See Fig.1-10A)

**Lead I**: the positive electrode is placed at the left arm the negative electrode at the right arm. I = (LA-RA) i.e. records the difference in the voltage between left arm and right arm.

**Lead II**: this is recorded by placing –ve electrode on right arm, while +ve electrode is on left Leg. (II= LL- RA) records voltage difference between left leg (LL) and right arm (RA).

**Lead III**: this is recorded by placing +ve electrode on the left leg, while the -ve electrode is on the left arm, records voltage difference between left leg (LL) and left arm (LL- LA).

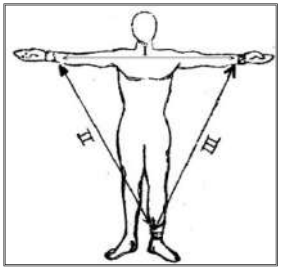
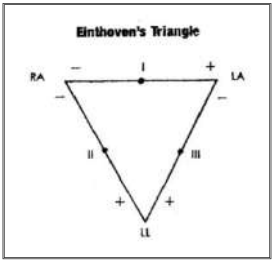



Fig.1-10A

The bipolar standard limb

leads I, II, III, represented

by Einthoven triangle (Fig.1-10B),




(Fig.1-10B)

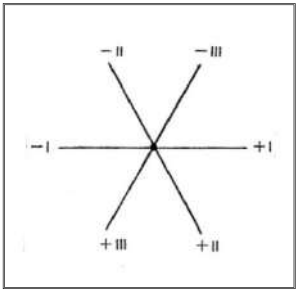
Einthoven triangle has been redrawn that lead I, II, and III intersect at a common central point, the result is the triaxial diagram in the (fig.1-10C), the bipolar leads are related by the following equation:

II=III+I

Lead I = (LA - RA)

Lead III = (LL - LA)

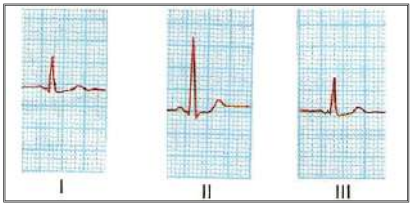
LL- RA = II




(Fig.1-10C ) B- the triangle is converted to a triaxial diagram.

(I.e. II= I+III)

Example

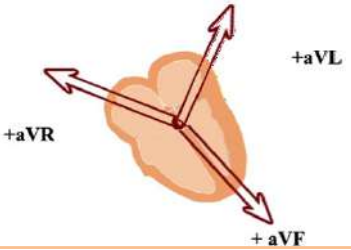



ECG example, with normal Heart axis.
Lead II = I+III

B- **Unipolar Leads**, Augmented* extremity leads aVR, aVL, and aVF.

**Unipolar**:- because the centre of the heart (Fig.1-11A) is used as a reference point and the electrode (positive +) is placed on the limbs and used as other point. i.e. records the electrical voltage at one location related to zero potential rather than relative to the voltages at another extremity as in the case of bipolar leads.

(Fig.1-11A)




*__Augmented__:- In order to monitor a lead in this manner amplify (augmented) the voltage in the ECG machine is needed, to get tracing of the same magnitude as leads I, II, III this increase the size of potential by 50% without any change in configuration from the Non augmented record

- aVR, aVL , aVF, the abbreviations as following :-

- a = augmented.

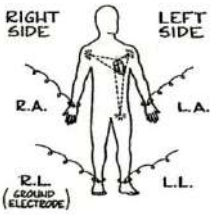
- V = voltage.

- R =
- L =
- F =

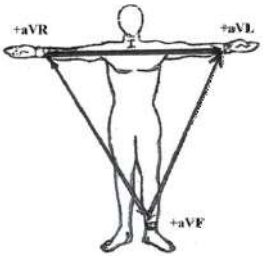
Refers to the limbs — Right arm, Left arm, Left Foot

In the augmented limb leads, one limb electrode is used for the positive electrode and other two are joined to form a ground reference.(Fig.1-11B)

(Fig.1-11B) Limb electrode sites

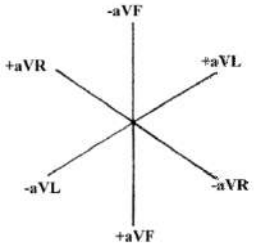



The Unipolar leads are represented by triangle. (Fig.1-11C)



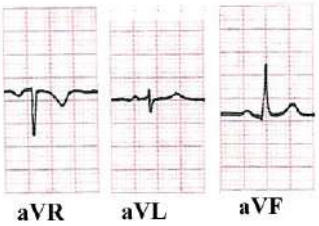

(Fig.1-11C)

The triangle is converted to a triaxial diagram. (Fig




(Fig.1-11D)

The Unipolar (augmented) limb leads also related by the simple equation (aVR + aVL + aVF = Zero) For example




**aVR = -10mm**

**aVL = +3mm – 3mm**

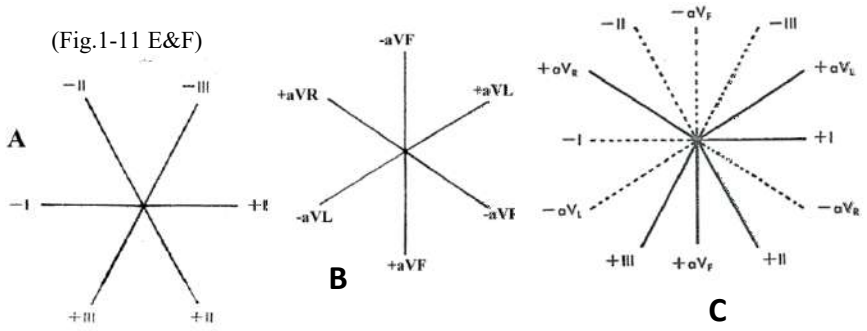
**aVF = + 10mm**

**aVR +aVF + aVL = - 10 + (+3-3) + (+10) = 0**

## C- Derivation of hexaxial lead diagram

The three unipolar and three bipolar Triaxial leads can be show on the same diagram, producing (Hexaxial lead diagram).

(Fig.1-11 E&F)

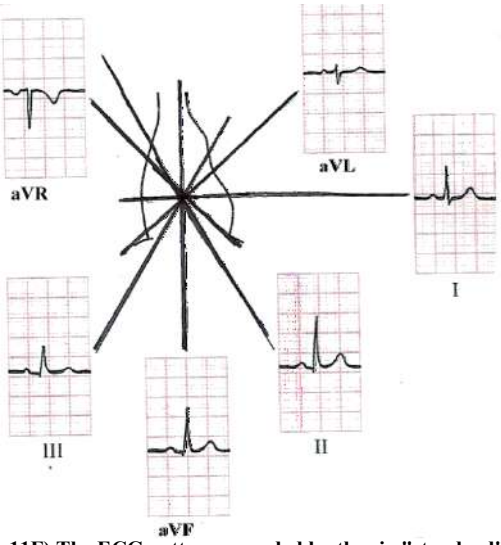



**(Fig.1-11E)**

A- Triaxial diagram of the bipolar leads (I, II, III).

B- Triaxial diagram of the Unipolar leads (aVR, aVL, aVF).

C- The two triaxial diagrams can be combined into hexaxial diagram.




**(Fig.1-11F) The ECG pattern recorded by the six "standard" leads**

**aVL is perpendicular to the lead II.**

**aVR is perpendicular to the lead III.**

**aVF is perpendicular to the lead I.**

## Chest Leads

**Precordial (chest) leads (V1 to V6): obtained by direct recording over the chest itself.** (Fig.1-12A)

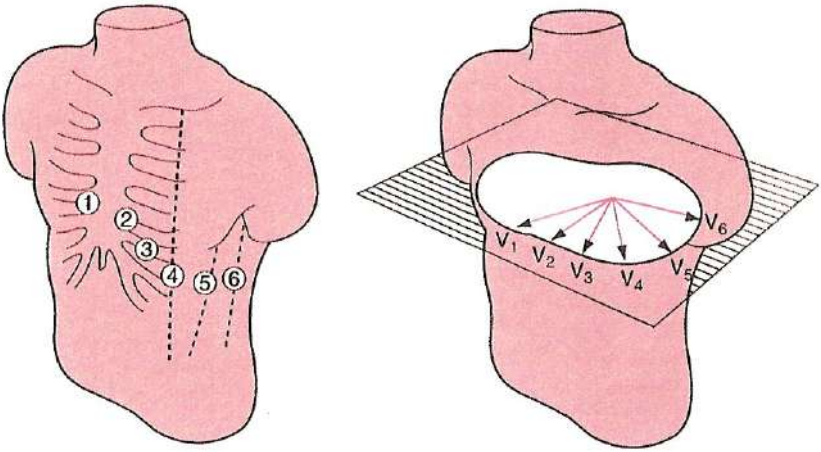



*Fig. 1.*12AThe sites of electrode placement on the precordium.

V1: Fourth intercostal space at right sternal border.

V2: Fourth intercostal space at left sternal border.

V3: Midway between V2 and V4.

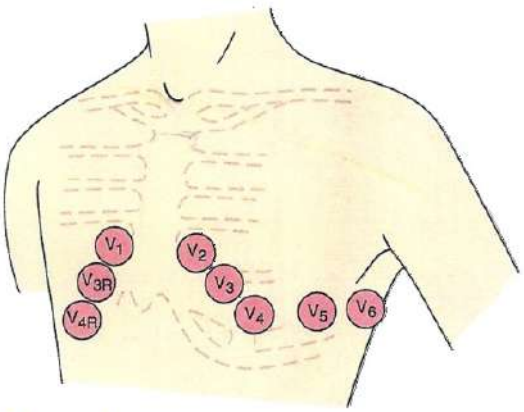
V4: Midclavicular line at fifth intercostal space.

V5: The left anterior axillary line at fifth intercostal space.

V6: The left midaxillary line at the same level with V4 and V5.

Other lead may occasionally be added V7: at posterior left axillary line.

Right ventricular leads V2R, V3R, V4R on the right chest comparable to V2, V3, V4 respectively. (Fig.1-12B)



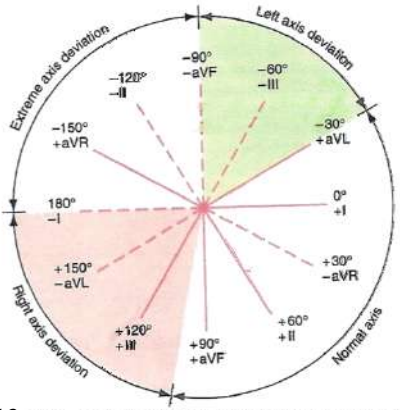

**(Fig .1-12B ) The horizontal plane (chest or precordial) leads are obtained with electrodes in the locations shown.**

## Determining electrical axis

## Cardiac Axis

Axis is an indicator of general direction of the electrical current through the heart.

In the ECGs of most normal people the normal axis of QRS lies between (-30° and 100°).(Fig.1-13)




Upper half of the circle Negative

Lower half of the circle Positive

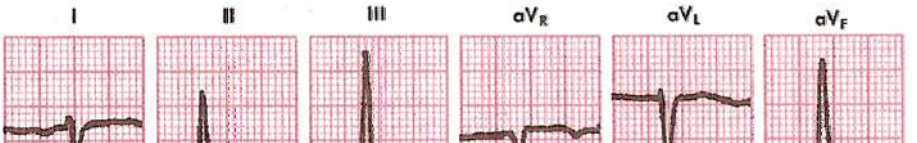
**(Fig.1-13 ) frontal plane (extremity or limb) leads are represented on a hexaxial diagram. Each ECG lead has a specific spatial orientation and polarity. The positive pole of each lead axis (solid line) and negative pole (hatched line) are designated by their angular position relative to the positive pole of lead I (0 ). The mean electrical axis of the QRS complex is measured with respect to this display.**

**Right axis deviation (RAD)** is exist if the QRS axis is found to be +100° or more positive.(See Figs.1-13,1-14, 5-6B & 5-7)

**Causes of RAD**

Occasionally occurs in normal individual, but sometimes indicates one of the following:-

1- Right ventricular hypertrophy.
2- WPW syndrome (type A).
3- Anterolateral MI.
4- Dextrocardia.
5- Left posterior hemiblock (Rare).



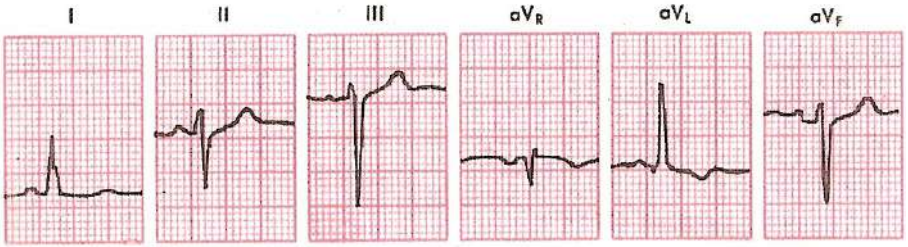
**Left axis deviation (LAD)** exist if the QRS axis is found to be -30° or more negative. (see Figs.1-13, 1-15, 5-4A&B, 5-6A)

## Causes of LAD

Left axis deviation (LAD) more often indicates one of the following:-

1. Left anterior hemiblock (LAH*).
2. Wolff-Parkinson-white (WPW) syndrome type (B).
3. Inferior myocardial infarction.
4. Ventricular tachycardia from left ventricular focus.

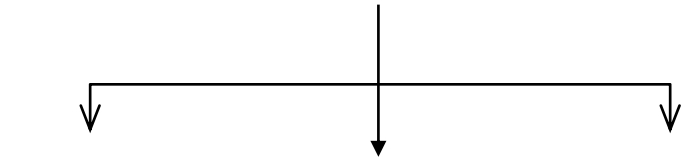
*LAH may caused by hypertension, aortic valve disease, ischemic heart disease, and other disease predominantly affecting the left ventricle as well as intraventricular conduction. See also chapter 5



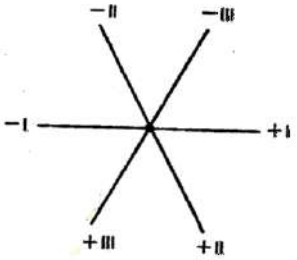

(Fig.1-15) **Notice the rS complex in lead II, from a patient with left axis deviation.**

***Practical Guide***

# Determining of the Electrical

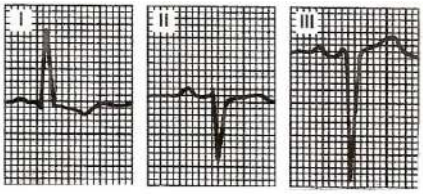



1. **Triaxial method of axis determination involves the following:-**
   - **A-** Draw the triaxial diagram of the Bipolar standard limb leads (I, II, III). (Fig.1-16)
   - **B-** Determining the sum of small boxes below the baseline (i.e. negative deflection of QRS) and small boxes above the base line (positive deflection of the QRS)in lead I or II and lead III.
   - **C-** Input the QRS sum of lead I or II and lead III on the same leads in (see the example 1-1).
   - **D-** Draw perpendicular line to lead I or II and lead III.
   - **E-** Draw a line from central point through intersection of perpendicular.
   - **F-** Intersection of the line with the circle prove approximate angle of electrical axis in regarding case.



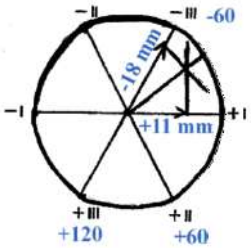

(Fig.1-16) triaxial diagram
of standard limb leads (I, II, III).

**(Example 1-1)**: Based on the triaxial limb leads (I, II, III) shown what is the approximate mean QRS axis?



***The answer***

1. Draw triaxial diagram which represent lead I, II, III (Fig.1-17).
2. To work out exact angle of the axis (use sine, cosine, or tangent).
3. In this case we use lead I and lead III to calculate the mean QRS axis as the following:-
   - A- In lead I we should calculate the number of small boxes below and above the baseline (i.e. negative and positive side respectively) -1mm + 12 = +11mm.
   - B- In lead III same as lead I. +1-19= -18.
   - C- Input the sums of the QRS for lead I and lead III on diagram (Fig.1-17).
   - D- Draw the perpendicular to lead I and lead III axis.
   - E- Draw a line from central point through intersection of the perpendicular.
   - F- Intersection of the line with the circle provide approximate angle of electrical axis (about - 45° in tl




**(Fig.1-17) triaxial diagram of standard limb leads (I, II, III).**

**2. Hexaxial method for estimating the electrical axis**

- In this method, use the standard limb leads (I, II, III, aVR, aVL, aVF).
- Using this method provides a quick approximation of the axis, because in biphasic* lead the QRS complex with Zero net amplitude then the axis is rapidly determined from the lead which perpendicular on it.
- Hexaxial method involves the following steps:-

A- Examine the all six extremity leads (I, II, III, aVR, aVL, aVF).(see the example 1-2)

B- Select the lead with biphasic QRS complex (i.e. when the positive and negative deflections are equal) or the lead with smallest QRS complex.

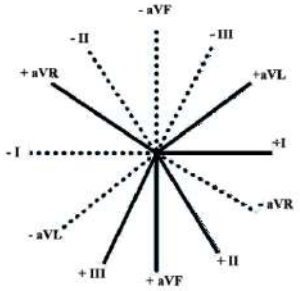
C- Select the lead whose perpendicular to the previously selected lead. For example:- Lead I is perpendicular on lead aVF and vice versa.

Lead II is perpendicular on lead aVR and vice versa.

Lead III is perpendicular on lead aVL and vice versa. (See Fig.1-18).

* Biphasic lead: is the lead with equal positive and negative deflection which means the QRS complex of this lead is zero net amplitude

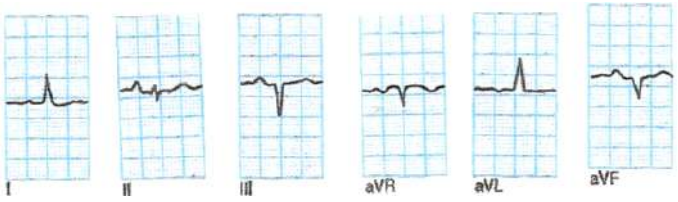
D- The angle of selected perpendicular lead is the angle of the electrical axis
(plus minus 20 degree).




**(Fig.1-18) Hexaxial diagram of standard limb leads (I, II, III, aVR, aVL, aVF)**

**(Example 1-2)**

Base on the hexaxial limb leads (I, II, III, aVR, aVL, aVF) shown what is the approximate mean QRS axis?




1. After careful inspection of six leads, we found the lead II with biphasic QRS complex (i.e. the number of small boxes above baseline is equal to the number of small boxes below baseline).
2. The lead aVL is the lead that is perpendicular to the lead II.(see fig.1-19)

3. The lead aVL = (-30°), thus the approximate angle of electrical axis in this example is (-30°) ± 20°.

**Note**: If the ECG of limb leads consist of two leads one with small QRS complex and the other with biphasic QRS complex, select one of them either the biphasic or the smallest QRS complex.

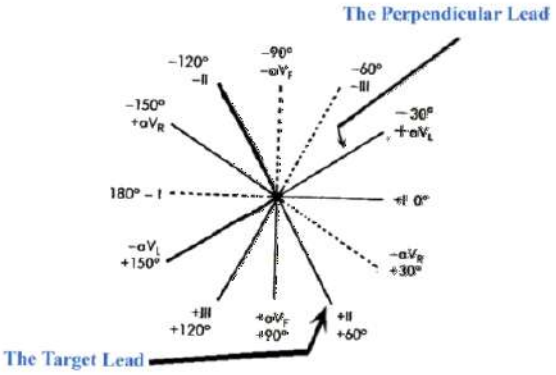
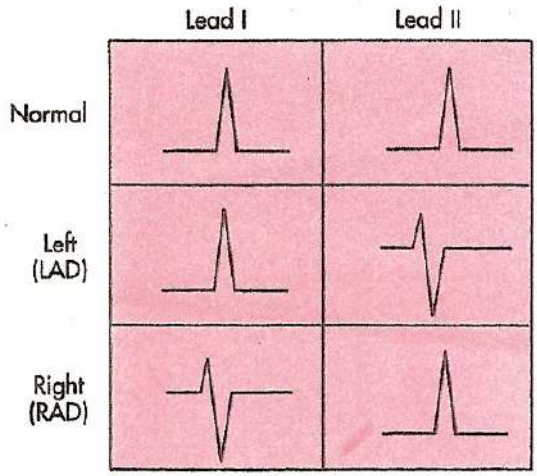



**Fig.1-19**

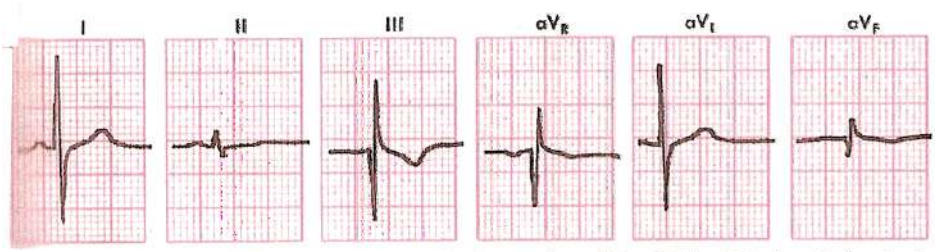
3. **The practical method for estimating electrical axis** (Fig.1-20)
   - This method allows the experienced cardiologist to quickly estimate the electrical axis by inspecting various leads of the ECG in particular order.
   - Example lead I and lead II if both of these leads positive that mean the QRS axis is normal.
   - Lead I and lead aVF if both of them are positive the axis is normal.
   - As general practical rule if lead I QRS is positive the right axis deviation is excluded.
   - If the lead I is positive and lead aVF is negative may indicated left axis deviation. (See Fig.5-6A)
   - If the lead I is negative and the lead aVF is positive may indicated right axis deviation.(See Fig.5-6B)




**(Fig.1-20)** Simple method for telling whether the QRS axis is normal using leads I and II. LAD, left axis deviation; RAD, right axis deviation.

**Indeterminate axis**

On rare occasions, all six limb leads show biphasic (QR or RS) complexes, which makes it impossible to calculate the mean QRS axis. In such cases the term indeterminate axis is used (Fig. 1-21). An indeterminate axis may occur as a normal variant or it may be seen in a variety of pathological setting.




**(Fig. 1-21)** Indeterminate axis. Notice the biphasic complexes (RS or QR) in all six frontal plane leads.

# Chapter 2

## Routine ECG

# Routine ECG approach

***The following points are important to consider when interpret an ECG:***

A- Never give undue weight to single ECG investigation particularly if the result does not fit with clinical finding.

B- ECG abnormalities may be seen in normal healthy person, in the absence of organic heart disease, these ECG finding include the following:-

1. Early repolarization. (see Fig.7-12)
2. High left ventricular voltage. (see Fig.3-3B)
3. Juvenile T wave.
4. Athletic T wave. — *See further description in the glossary end of this book*
5. Insignificant Q waves in lead aVL, I,V5, V6.(see chapter 6)
6. Right axis deviation.(see chapter 1)
7. Low voltage QRS complex in obese individual. (see chapter 7)
8. Short PR interval. (see chapter.4)
9. First degree and wenkbach AV block.

C- ECG may be normal or Uninterpreted in the presence of organic heart disease, include the following conditions:-

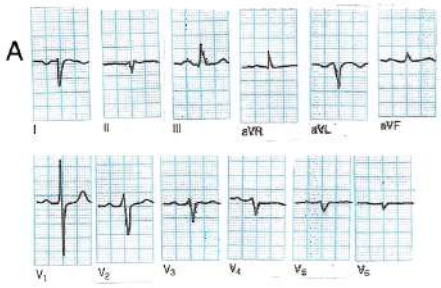
1. Acute MI may not show diagnostic ST-T changes especially in the early presentation or the diagnosis of MI may be masked by LBBB, WPW syndrome with preexcitation, electronic ventricular pacemaker.
2. Patients with sever coronary disease, may not show diagnostic ECG changes during stress testing.
3. Acute pulmonary embolism the ECG may be normal or with non specific changes.
4. In some cases with significant LVH and/or RVH the ECG may looks normal.
5. The ECG may be normal between the attacks of the intermittent arrhythmias like like:
   A- Paroxysmal atrial fibrillation.
   B- Paroxysmal supraventricular tachycardia.
   C- Ventricular tachycardia and bradycardia.

D- ECG abnormalities may be seen secondary to the extracardiac disease, in otherwise healthy heart, that provide important clues in the evaluation of such medical conditions which described in detail on chapter of miscellaneous ECG changes as:-

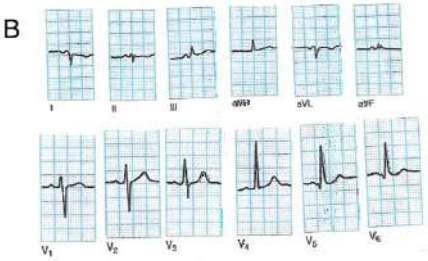
1. Cerbrovascular accident (especially intracranial hemorrhage).
2. Drug toxicity.
3. Electrolytes disorders.
4. Endocrine disorders.
5. Hypothermia.

E- Always bear the following artifact in mind when interpreting an ECG:-

1. *Reversed limb leads*; for example, reversal of the left and right arm electrodes can cause an apparent rightward QRS axis shift as a general rule, when lead I shows a negative P wave and a negative QRS, reversal of the left and right arm electrodes should be suspected this is called technical dextrocardia, to differentiate it from true dextrocardia, chest leads should be studied. In reversal limb leads (technical dextrocardia) chest leads will be normal, while in true dextrocadia the chest lead ECG does not show the normal progressive increase in R wave height across them, the right sided chest leads will show the pattern normally seen on the left (i.e. prominent R wave in V1 and V2) (Fig.2-1A&B).

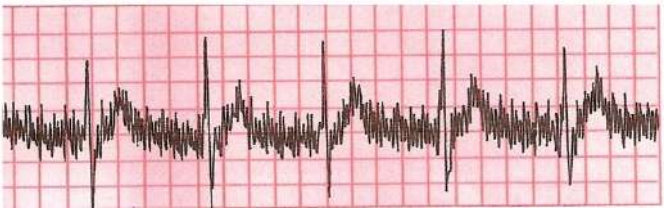



**(Fig.2-1 ) A- Dextrocardia the ECG shows a negative P, QRS in Lead I and decrease in R wave height across chest Leads**




**B- limb lead reversal (technical dextrocardia) the ECG shows a negative P wave and negative QRS complex in lead I but with normal R wave progression in chest leads.**

2. Many ECGs are mistakenly thought to show "high" or "low" voltage when the voltage is actually normal but the standardization marker is set at half standardization or two times normal sensitivity, the normal standardization is 1mv makes the recording needle more 10 mm = 2 large boxes, every ECG must include a calibration mark so that the gain setting can be checked, sometimes it is necessary to alter the calibration (Standardization), particularly when the QRS complexes appear too big or too small, it is good practice to record this clearly by writing a note on the ECG. (See chapter 1) (Fig.1-7B).
3. *Incorrect paper speed*
   the standard ECG recording speed is 25 mm/s, so that 1 small (1mm) box equal to 0.04s. if the paper is run at double the speed (50 mm/s) the waves will double in width. Always label every ECG you record with the paper speed used and if you use a non-standard setting, it is good practice to document this clearly at the top of the ECG. (See chapter 1) (Fig.1-6C).
4. *External electrical interference*
   A 50-Hz electrical interference from domestic appliances has been reported as significant cause of ECG artifact, and this can make the ECG difficult or even impossible to interpret correctly. This stray electric current can be detected by cardiac monitor and appears as wide, dark baseline on the ECG, this type of artifact is produced most often by electrical equipment that contains electric motors, such as intravenous pumps, ventilators, beds, mobile, and so on. You can usually eliminate this interference by switching the ECG plug to a different outlet or turning off other electrical appliance in the room. (Fig.2-2)



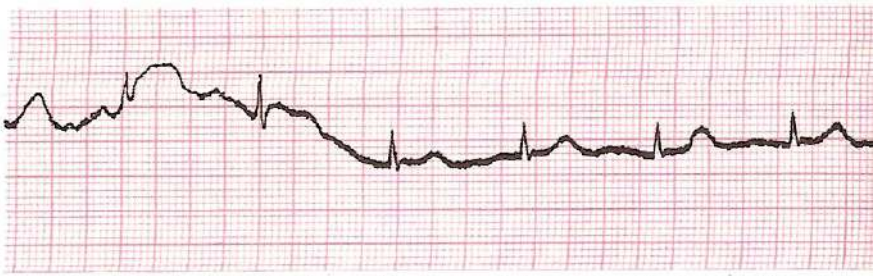
**(Fig. 2-2)** Common ECG artifact produced by 60-Hz electrical interference.

5. *Patient movement*

   Skeletal muscle activity is also picked up on the ECG, and it is important for patient to lie still and relaxed, while their ECGs are recorded unfortunately, this is not always possible, particularly if the patient is:

   - Uncooperative or agitated.
   - In respiratory distress.
   - Suffering from a movement disorder.

   However, signal-averaged recording can also be misleading by introducing artifactual changes of their own, and such recording should always interpreted with discretion. (Fig.2-3)



**(Fig.2-3)** Wandering baseline resulting from patient movement or loose electrode contact.

6. *Poor electrode contact*, loose electrodes, broken cables, or broken wire. This pattern (Fig.2-4) has a wandering baseline that is often interspersed with wide, unusual wavelike fluctuations, this type of artifact is simple to eliminate by either replacing the electrodes, the broken wires, or both.

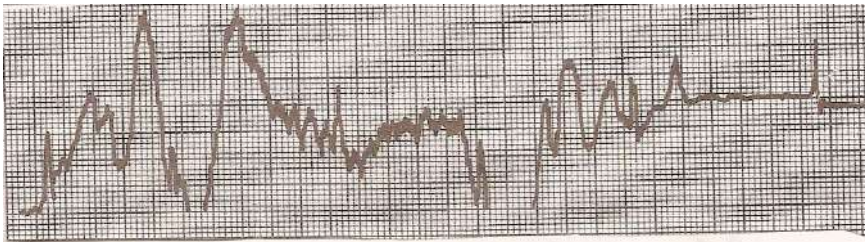


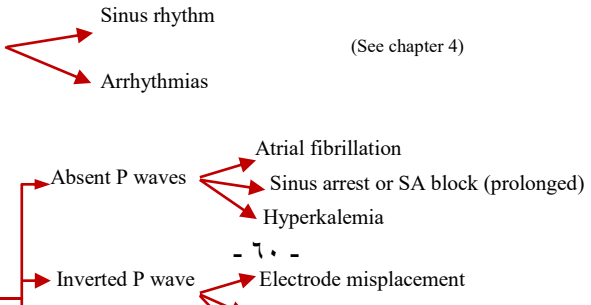
Fig.2-4

# How to Interpret an ECG

ECG interpretation is largely a matter of experience and pattern interpretation. However, while building experience, it is useful to develop a method of systematic "ECG" analysis. This is most easily performed by asking oneself a number of questions in logical sequence about P, QRS and T

A simple system is presented in the following sequence:

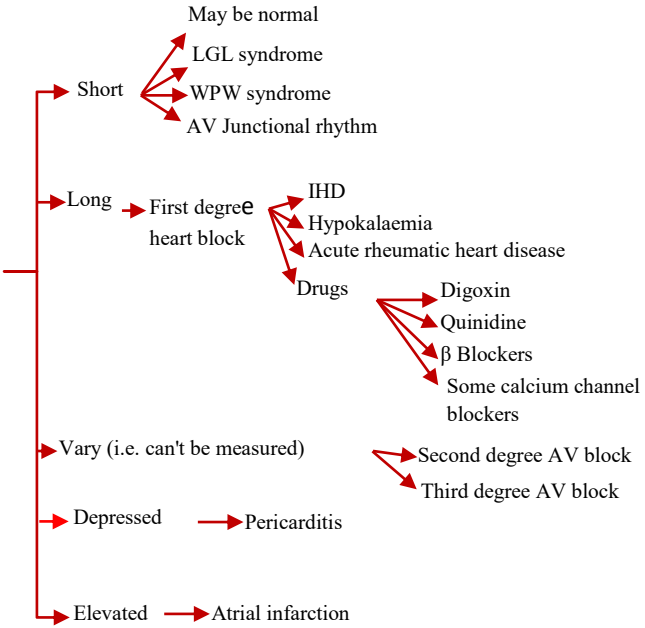
1. ***Standardization***:
   An ECG recording is standardized so that 1mv gives a deflection of 10 mm on the paper; the height of a deflection therefore indicates its voltage. (see Chapter1)
2. ***Paper speed:***
   Normally the ECG are recorded at paper speed 25 mm/second i.e. 5 large squares/second, then / minute ECG tracing covers 300 large squares. (see Chapter1)
3. ***Rate:***
   Calculation of the heart rate is simple and can be done is several ways. (see Chapter1)
   As a general rule, a regular heart rhythm with a rate between 60 and 100 beats/ min is normal.
   If the heart rate below 60 beats/min is bradycardia.
   If the heart rate above 100 beats/min, the patient is tachycardia.



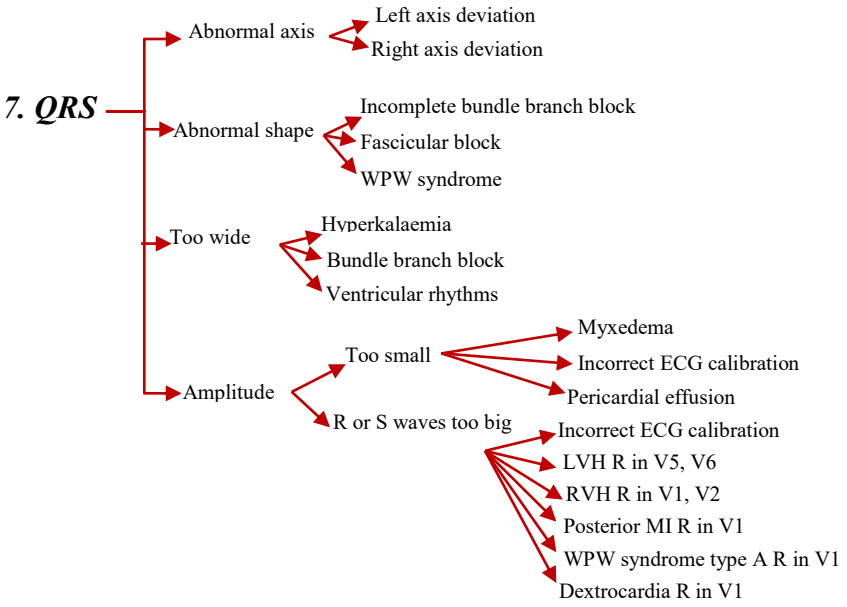
4. ***Rhythm***

5. ***P wave***

### 6. *PR interval*




### 7. *QRS*



## 8. Q wave

- Q waves are usually absent from most of the leads of a normal ECG.
- Physiological small Q waves are normal in leads that look at the heart from the left : aVL, I, V5, V6 (septal q waves) results from septal depolarization from left to right.
- Pathological (i.e. > 25 % of R height or more than one small box wide are significant) may be due to: - 1. MI. 2. LVH. 3. BBB. 4. Pulmonary embolism

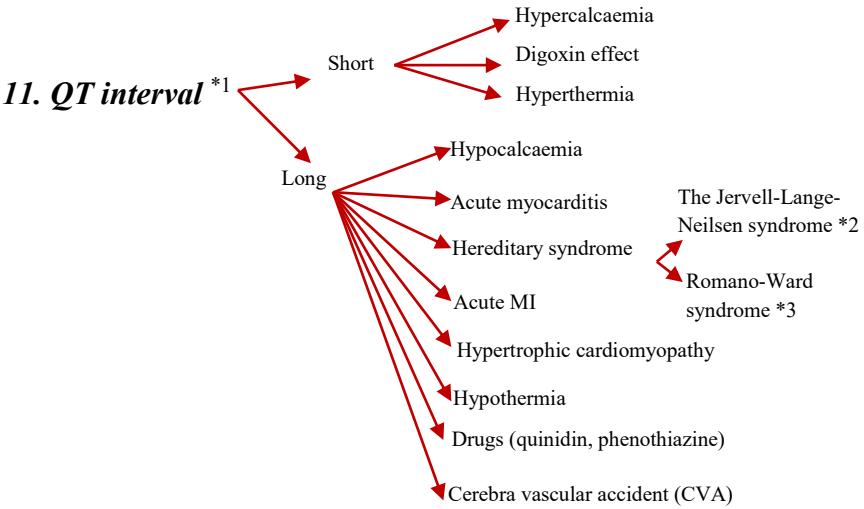
## 9. ST segment

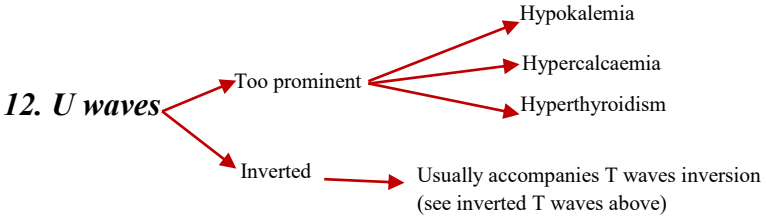
- Depressions
  - Myocardial ischemia
  - Acute posterior MI
  - Drugs (digoxin, Quinidine)
  - Ventricular hypertrophy with "strain"
  - Reciprocal changes of MI
  - Hypokalemia
- Elevation
  - Acute MI
  - Pericarditis
  - LV aneurysm
  - Early repolarization
  - Prinzmetal angina
  - Metastasis of myocardium

## 10. T waves

- Too tall
  - Hyperkalemia
  - Acute MI
- Too small
  - Hypokalemia
  - Hypothyroidism
- Inverted T waves
  - Normal (aVR, V1)
  - Normal variants (V2, V3) Juvenile pattern
  - Myocardial ischemia and infarction
  - Ventricular hypertrophy with "strain"
  - Digoxin toxicity
  - Paroxysmal tachycardia
  - Hyperventilation, cholecystitis
  - (MVP) mitral valve prolaps
  - (SAH) subarachnoid hemorrhage
  - Pulmonary embolism

N.B In the last four conditions the ECG is not diagnostic




## *1 QT interval

**A-** the rate corrected QTc is obtained by dividing the actual QT by the square root of the RR interval (Bazett formula) both measured in seconds QTc = (see chapter 1).

**B-** the Bazett formula recently modified by Hodges and Coworkers, and Macfarlane and Veitch Lawire as follow.

QTc = QT + 1.75 (ventricular rate - 60) .

**C-** The normal range QTc interval at a heart rate 60 beats/min is 0.35-0.43s.

**D-** Rule of thumb

$$\frac{QT}{RR}$$

Memory aid: students looking for a mnemonic to help recall key features of ECG can try using (IR-WAX) as the following.

- I- is for intervals (RR, PR, QRS, QT)
- R- is for rhythm. (Sinus or others)
- W- is for five waves (P, QRS, T & V)
- AX- is for electrical axis.

*2 ***the Jervell-Lange-Nielsen syndrome***

In 1957, Jervell and Lange-Nielsen, described an unusual syndrome of congenital deafness, a family history of syncopal attacks and sudden death due to paroxysmal ventricular tachyarrhythmias in individuals whose only manifest cardiac abnormality was a prolonged QT interval, the syndrome is inherited as an autosomal recessive trait.

*3 ***Romano-Ward syndrome***

**Pediatric ECG**

rd syndrome is a related syndrome which manifests without the deafness and which is apparently inherited as an autosomal dominant trait.

***The topic of pediatric ECG falls outside the scope of this book, but a few critical points of difference between pediatric and adult ECG are mentioned briefly:-***

1. Before the pediatric ECG can be interpreted, the age of the child must be known, as must information about the indication for the testing, clinical diagnosis, medications, and electrolytes.

2. Interpretation of the neonatal ECG is the most challenging because of the rapid hemodynamic changes taking place causing the ECG to change rapidly during the first few weeks of life.

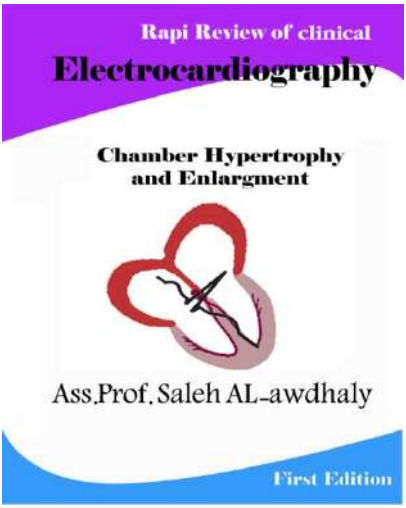
3. At 3 years of age the child's ECG begins to resemble the adult's, for example normal heart rate in newborn varies from 110to200 beats/min in the first week of the life the average rate is less than 140 beats/min in the first year; it is less than 120 beats/min. sinus bradycardia is rare in normal, healthy children. It may be seen in hypothyroidism, hypothermia, hypopituitrism, obstructive Jaundice, typhoid fever.

   Reflex sinus bradycardia occurs because of increase intracranial pressure.

   Right axis deviation gone in 2-3 years, although there remains significant, persistent difference, as big R wave in V1 may persist to age 5 years, anterior T wave inversions out to V4 may last into early teens.

# Chapter 3

# Chamber Hypertrophy and Enlargment



## Chamber hypertrophy and enlargement

The ECG diagnosis of right or left ventricular hypertrophy has a low sensitivity 50% but high specificity > 90%. Thus approximately half of the individuals with ventricular hypertrophy cannot be recognized by the ECG.

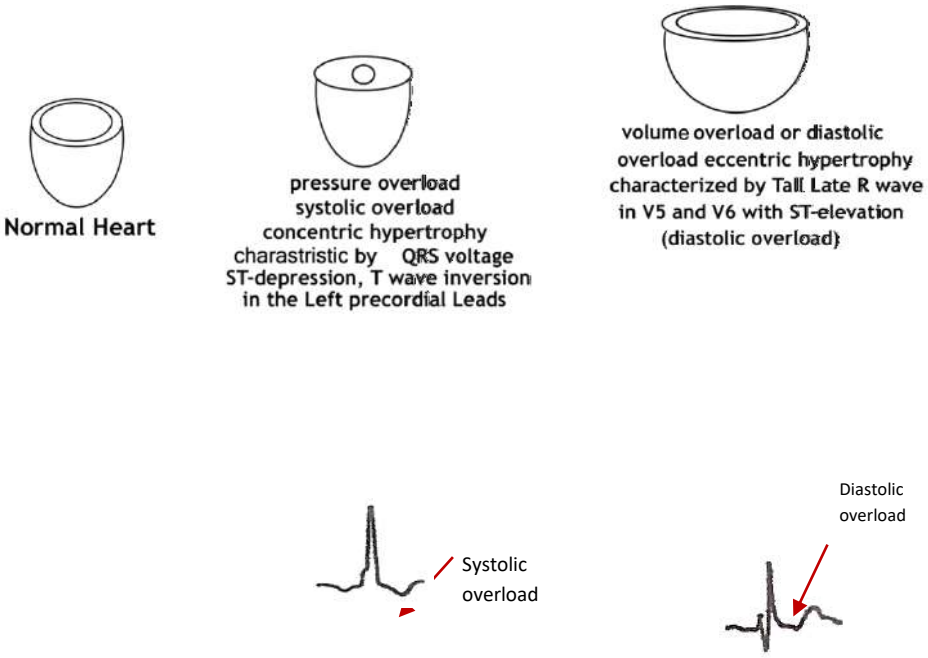



Fig. 3-1

## Left ventricular hypertrophy and enlargement.(Fig.3-1)

## A-Left ventricular hypertrophy (Fig 3-2E)

- *Pathophysiology of the LVH*
  - Increase muscle thickness of the left ventricle cause shift of QRS axis toward the left precordial leads (more muscle, more voltage) results by a deep S wave on the right-oriented leads and tall R waves in the left oriented leads. (voltage criteria). (Fig.3-2A)

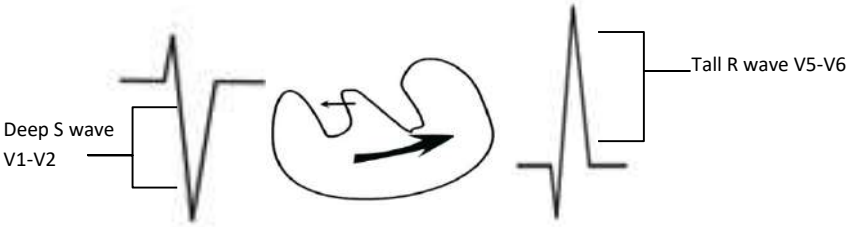



Fig.3-2A

  - The hypertrophied left ventricle is under strain probably the blood supply of the subendocardium is compromised causing ST-T changes opposite main QRS complex that look ischemic (referred to as strain pattern) in the left oriented leads V5 and V6.(Fig.3-2B)

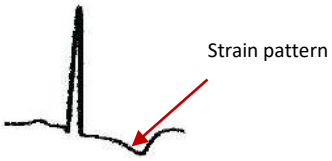
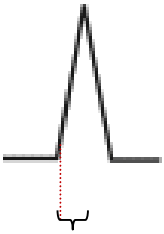



Fig 3-2B

  - The extra thickness of the left ventricle prolongs QRS and ventricular activation time also is prolonged. (Delay the intrinsicoid deflection).
    (Fig.3-2C)



>0.05 sec (normal is =0.04 sec)

- The delay in depolarization of the left ventricle may sometime cause slight widening of QRS complex i.e. ≥ 0.09 sec
- High left ventricular pressure is reflected to the left atrium causing atrial (P wave) abnormalities on the ECG.(Fig.3-2D)

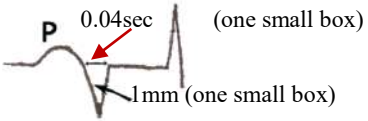



Fig 3-2D

- Long standing hypertrophy cause fibrosis of the left anterior branch of the left bundle branch (anterior hemiblock) results in left axis deviation (LAD). (see chapter 1)
- In advanced cases LVH cause pressure on the septum results in partial LBBB, in some person complete LBBB appear which completely mask the ECG finding of the LVH.(See chapter 5)

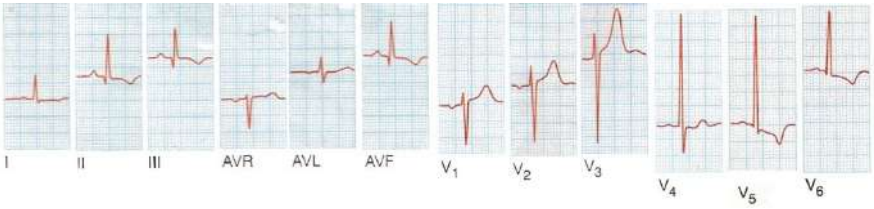



Fig (3-2E) **Left ventricular hypertrophy: QRS**

**complexes in limb leads have increased amplitude with a very large R wave in V5 and S wave in V2**. There is ST depression and T wave inversion in lead II, III, AVF, V5 and V6 (left ventricular strain pattern)

B-Left ventricular dilatation *(eccentric hypertrophy) (volume overload) (diastolic overload)*. (Fig 3-3)

Left ventricle dilatation, the useful ECG sign is voltage "Discordance" increase diameter but not thickness of LV cause lateral displacement of LV results in low R wave amplitude on limb leads as well as anterior chest leads with prominent R wave in V6 more than V5 with upward ST-T changes. (Fig 3-3)

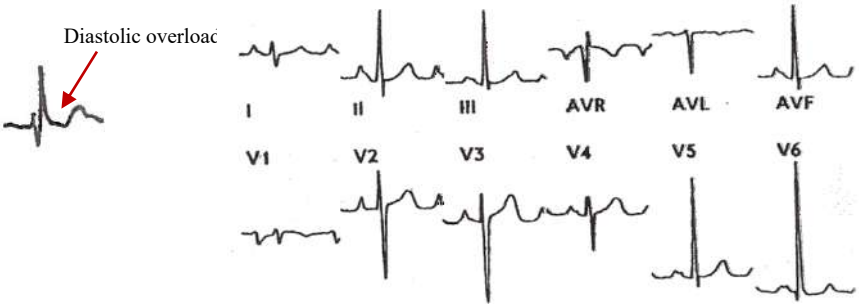



Fig (3-3A) left ventricular dilatation due to diastolic overload, also called LVH due to diastolic overload.

Key points:-

- Tall R wave in V6 than V5.
- Prominent q wave in V6.
- Mild elevation of ST-T wave in lateral precordial leads

- **A number of ECG criteria have been developed for diagnosis of LVH as following:-**

1. The Romhitt and Estes point score system.

A- Increase QRS amplitude voltage criteria (3 points)
- R or S wave in any limb leads=20mm.
- S wave in V1 or V2 =30mm.
- R wave in V5 or V6 =30mm.

B- ST-T abnormalities (3 points) (without digitalis) but if digitalis 1 point.
C- A P wave of left atrial enlargement (3 points).
D- Left axis deviation (2 points).
E- Increase ventricular activation time* (1 point).
F- QRS duration ≥ 0.09 sec (1 point)

- *LVH= 5 or more points.*
- *Probable LVH= 4 points*

**see glossary for details*

2. Other ECG voltage criteria for the diagnosis of LVH as a follow:-

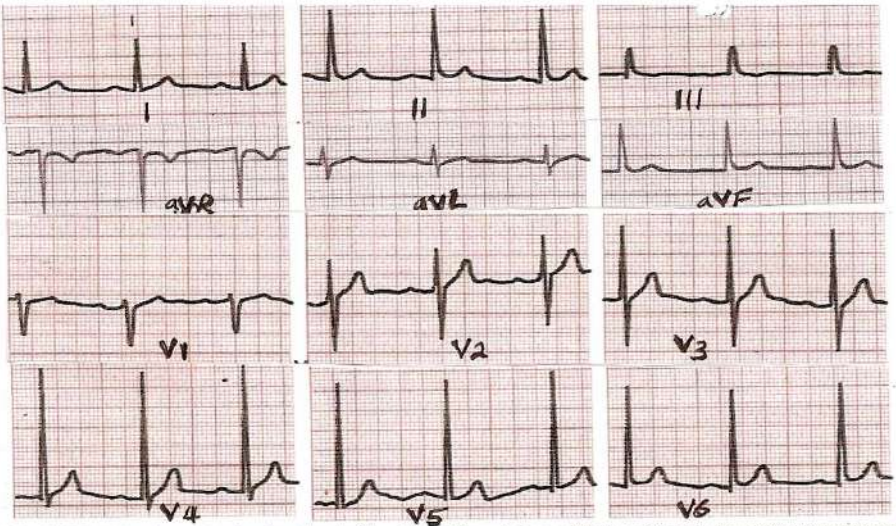
A- Cornell voltage criteria:-
S in V3 + R in aVL > 24mm (men).
S in V3 + R in aVL > 20 mm (women).
B- Limb leads voltage criteria for LVH
R in aVL ≥ 11 mm or if left axis deviation : R in aVL ≥ 13mm plus S in lead III ≥ 15mm.
C- Chest leads voltages criteria (Sokolow-Lyon criteria for LVH) S wave in V1 plus R wave in V5 or V6 ≥ 35mm

The voltage criteria has limitation in clinical practice because

1. High voltage QRS complex may be normal variants in young people. (Fig.3-3B)
2. Normal ECG does not exclude LVH.
3. In the presence of other diseases like pericardial effusion, emphysema, myocardial infarction may change the voltage criteria of LVH.

***Comment***

- In patient with marked LVH and septal hypertrophy R wave in V1,V2, V3 may be absent simulating anterior wall myocardial infarction. See anterior myocardial infarction.
- Also Q or QS in II, III, aVL or I and V6 may be present simulating inferior or lateral wall MI. (see also Fig. of pseudo infarction in the glossery)




(Fig.3-3B) Tall voltages in the chest leads ($S_{V_1} + R_{V_5} = 36$ mm) from a 20-year-old man represent a common normal ECG variant, particularly in athletic or thin young adults. The ST-T complexes are normal, without evidence of repolarization (ST-T) abnormalities or left atrial abnormality.

## Right Ventricular Hypertrophy (RVH) (Fig.3-

The electrical forces of the hypertrophied RV (Fig.3-4A)

is directed to the right because an increase in RV voltage over the right chest leads, and an associated shift QRS axis toward the right. There is often strain pattern in leads reflecting high RV voltage.

(Fig 3-4A)

ECG changes of right ventricular hypertrophy

- Increase positive forces in V1-V2 (i.e. ↑ R wave amplitude).
- ST-T wave changes in V1-V2 opposite main QRS direction. (i.e. strain pattern) it's may be due to subendocardial ischemia.
- Increase negative forces in left precordial lead V5-V6, i.e. reversal of the normal pattern.
- Often, right axis deviation.

**ECG diagnosis of Right Ventricular Hypertrophy**

Suggestive Criteria

- R/S (ratio) in V1 ≥ 1, or
- R in V1 ≥7mm, or
- R in V1 + S in V5 or V6 ≥ 10.5mm

Supportive findings

- Right axis deviation ≥ 110°.
- Right atrial abnormality (RAA)
- ST depression + T wave inversion in V1 or V2 (RV strain).
- Normal QRS duration if no right bundle branch block.

***As a general rule:*** *the tall R wave in V1 being the most sensitive for diagnosis of RVH because of its proximity to the right ventricle.*

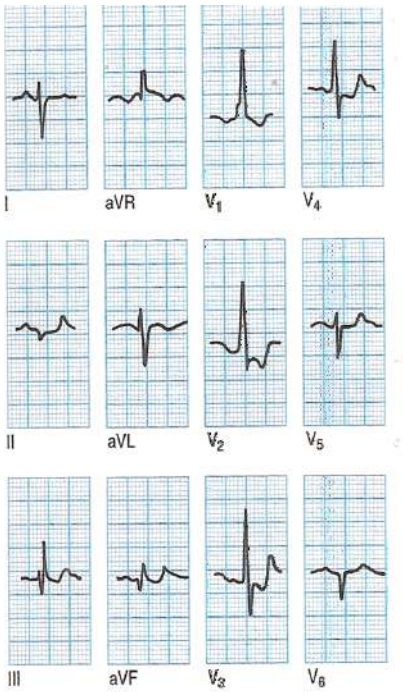



Fig (3-4B) Right ventricular hypertrophy with "Strain"

Key points

- Dominant R waves in lead V1, V2.
- Right axis deviation.
- ST segment depression/ T wave inversion in leads V1, V3

***Differential diagnosis of prominent R wave in V1 has been discussed in (chapter 6).***

**Practical Guide**

1. An R wave exceeding the S wave in the lead V1 is suggestive but not diagnostic of RVH.
2. The appearance of the following ECG pattern is very strong evidence of RVH which are:-
   - Tall right precordial R waves.
   - RAD.
   - ST depression and T wave inversion in V1-V6.
3. Advanced state of mitral stenosis produces LAA and right ventricle hypertrophy.
4. RVH and RBBB in V1 can produced by the most common type of ASD (i.e. ostium secundum ASD )

## Biventricular Hypertrophy (Fig 3-5)

Enlargement of both ventricles is suggested if any of the following combinations of ECG changes are present:-

- Voltage criteria for LVH in the precordial leads combined with right axis deviation in the limb leads.
- Criteria for LVH in the left precordial leads combined with prominent R waves in the right precordial leads.
- A low amplitude S wave in the lead V1 combined with a very deep S wave in lead V2.
- Left atrial enlargement as the sole criterion for LVH combined with any criterion suggestive of RVH.
- The Katz-Wachtel phenomenon consisting of biphasic complexes in two or more limb leads and in the mid precordial leads. This is seen in many congenital lesions, but is perhaps most common in VSD.
- In rare occasions the ECG may look normal because of partial cancellation of electrical forces with clinical evidence of biventricular hypertrophy.

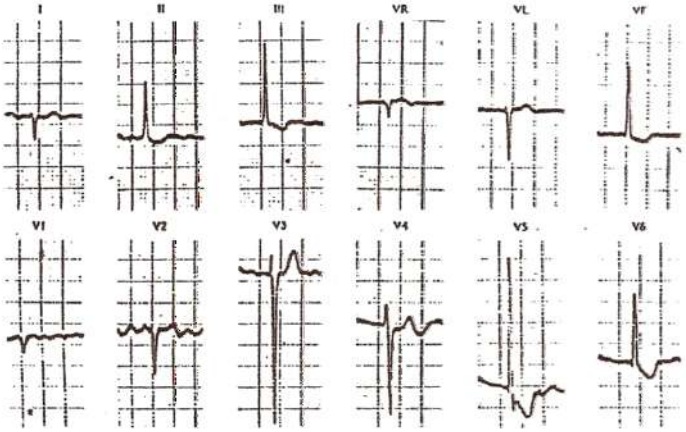
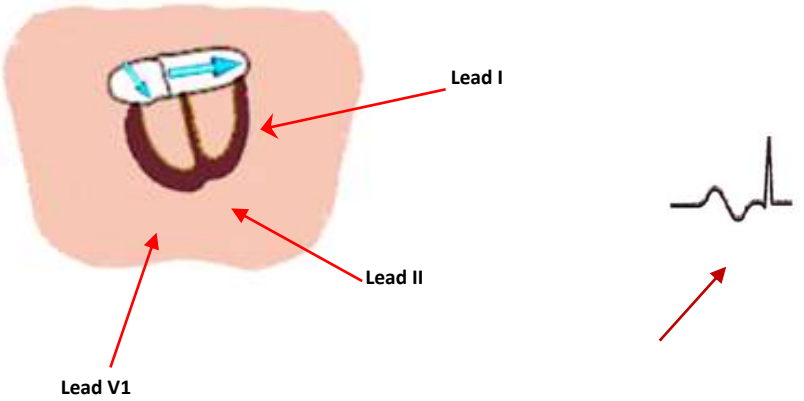



Fig (3-5) ECG: Biventricular hypertrophy

## Atrial Enlargement (Fig. 3-




*Normal Biphasic P wave in V1*

Fig. 3-6

**Mechanism**

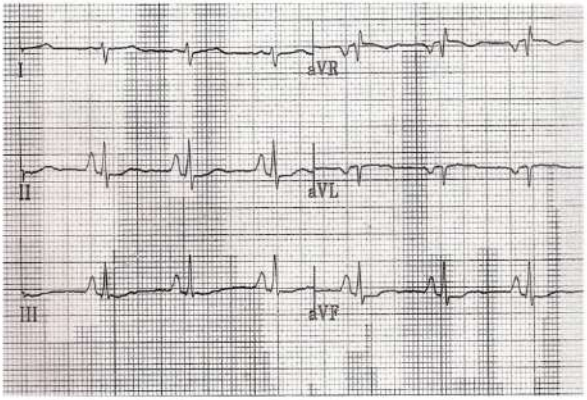
- The P wave represents the contraction of both atria, the right atrium depolarizes first represents by the initial peak of P waves followed by left atrial depolarization.
- In lead V1 normally the first peak of P wave is the depolarization of the right atrium, because the electrical force is toward that lead, and the second deflection less than (1mm) is the depolarization of the left atrium is negative because its opposite to the direction of the electrical activity of the left atrium. (Fig.3-6)

***What happen if one atria is abnormal?***

1. **Right atrial enlargement (Fig.3-7A&B)**

Fig. 3-7A

- Arrows indicating major atrial electrical vectors, associated with hypertrophy of the right atrium,
  with a shift in the direction of the P wave axis downward toward the feet. That is in the direction of the positive poles of lead II,III, aVF, the P wave is characteristically tall mid peaked in these leads of more than 2.5mm (2.5 small box) with normal duration.




**(Fig.3-7B)Right atrial abnormality (RAA). Tall, peaked P wave in the inferior leads (at least 2.5mm in one of the leads)**

## 2. Left atrial enlargement (Fig.3-8A,B &C)

- P wave is wide ≥ 0.12 (more than or equal 3 small boxes) in all leads.
- Exaggeration of the normal pattern lead to increase negative deflection of P wave in V1 ≥ small box ≥ 0.01mv, also delay depolarization of LA cause P wave widening ≥ one small box in V1.

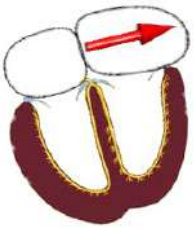


Fig. 3-8A

- In other leads including lead I there is bifid P wave (P mitral) the initial peak is the activity of the right atrium the second wide peak is the activity of the left atrium.(Fig. 3-8B&C)


Fig. 3-8B

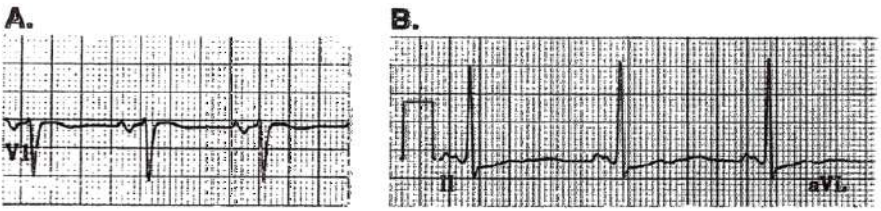



**Fig (3-8C) Left atrial abnormality (LAA). Two ECG findings may be used to make the diagnosis. A: Biphasic P wave in lead $V_1$; the negative deflection should be 1 mm deep and wide. B: Broad, notched P wave in one of the limb leads, most commonly II, III, or aVF, as the P wave vector is aimed at the inferior leads.**

**Comments**:-

- left atrial enlargement may be the only sign of LVH in the presence of LBBB.
- Left atrial enlargement may be the earliest and only ECG sign of the systemic

## 3.Bi atrial hypertrophy

- The ECG changes are Just exaggeration of the normal pattern of both atria. The result is peak, wide P wave e.g. lead I.(Fig.3-9A)

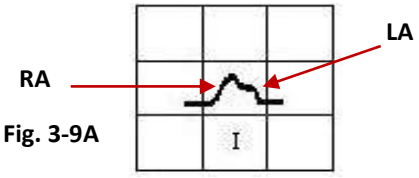



Fig. 3-9A

- peak positive component and deep negative component of P wave in V1 due to RA, LA abnormalities respectively.(Fig.3-9B)

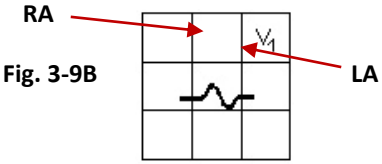
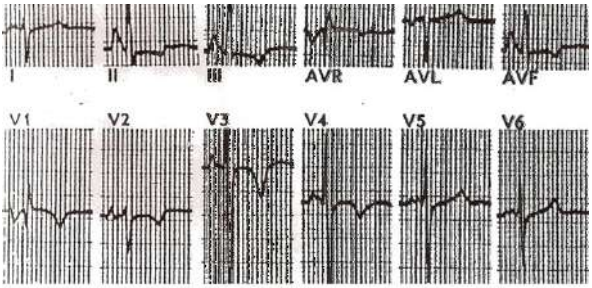



Fig. 3-9B

- combined right and left atrial enlargement occurs with the following conditions (Fig.3-9C):-

1. Mitral stenosis associated with marked pulmonary hypertension.
2. Mitral stenosis associated with tricuspid incompetence.
3. Mitral stenosis associated with tricuspid stenosis.
4. Atrial septal defect.
5. Lutembacher's syndrome: ASD associated with acquired mitral stenosis.

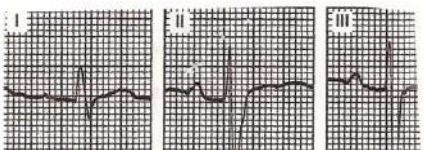



**(Fig.3-9C) Biatrial Hypertrophy**

# Typical ECG Changes in Selected Congenital Heart Diseases

There are certain rare congenital heart diseases that are associated with typical combinations of abnormalities on the ECG.

1. *Dextrocardia with situs inversus*: P waves, QRS complex and T wave are all inverted in lead I.(See Chapter 7)
2. *Anomalous left coronary artery originating from the pulmonary artery*; Q waves, ST elevation and T inversion in lead I, aVL, V4, V5 and V6.
3. *Ostium primum atrial septal defect* (common AV canal) there is marked left axis deviation of the QRS complex in frontal plane with typical appearance of left anterior fascicular block. There is also right bundle branch block and other changes described above for right ventricular dilatation. (see Fig.3-10)
4. *VSD and PDA*: there is combined right and left ventricular hypertrophy this is characterized by high voltage biphasic QRS complexes in the midprecordial leads, as discussed above under biventricular hypertrophy.
   Frequently, there are also prominent Q wave in the left precordial leads or inferiorly oriented limb leads.
5. *Ebstein's abnormality*:- there is a combination of extremely tall P waves indicative of right atrial enlargement without evidence of right ventricular involvement, prolonged QRS duration with the initial slowing of ventricular preexcitation, in many instances the QRS is positive in V1, presenting an appearance of an atypical RBBB, there also generally low QRS voltage.

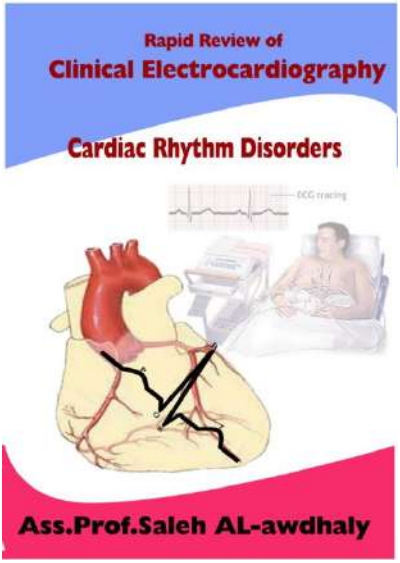


# Chapter 4

# Cardiac

# Rhythm

# Disorder



## Normal sinus rhythm (Fig 4-1)

The characteristic features of sinus rhythm are:-

1. P waves are upright in lead II and inverted in lead aVR.
2. Every P wave of similar shape and axis.
3. PR interval is fixed (between 3-5 small boxes).
4. QRS is normally preceded by P wave.
5. RR interval, very slight-irregularity i.e. apparently regular.
6. Heart rate between 60-100 beats/min.

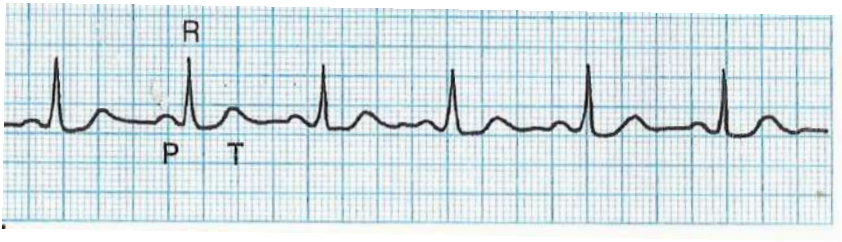


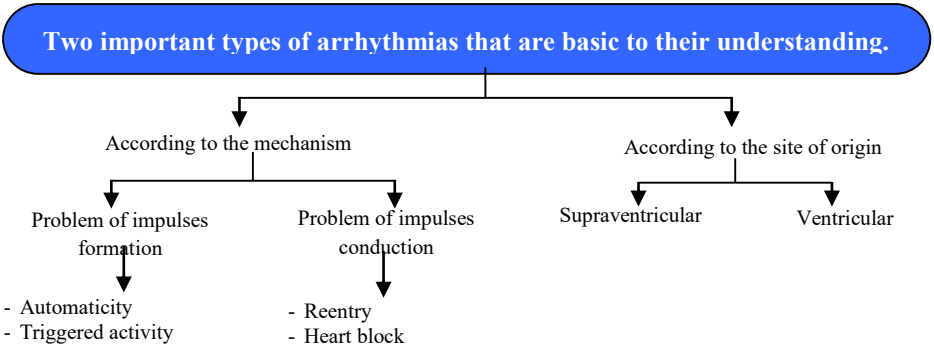

Fig (4-1)sinus rhythm

**Key point:**

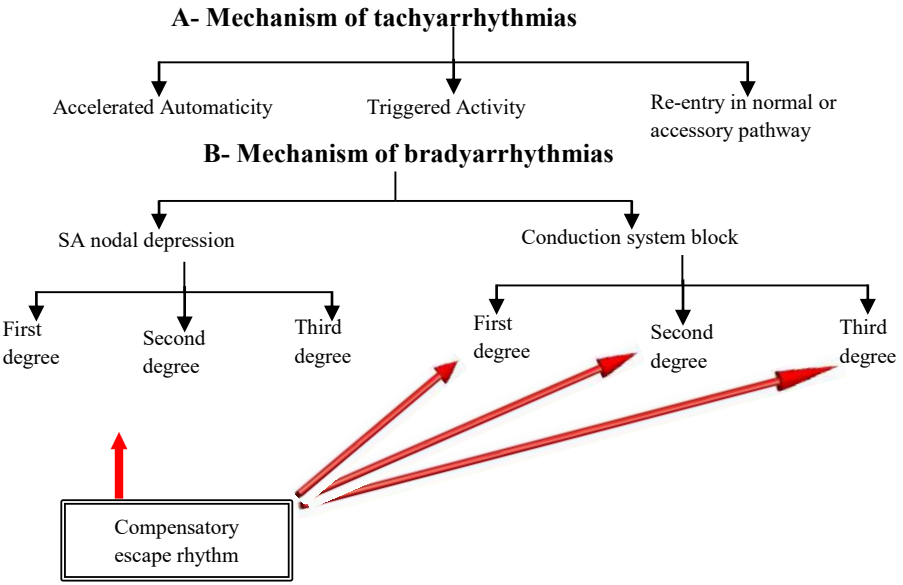
- P wave are upright (lead II)
- QRS complex after every P wave.

# CARDIAC ARRHYTHMIAS

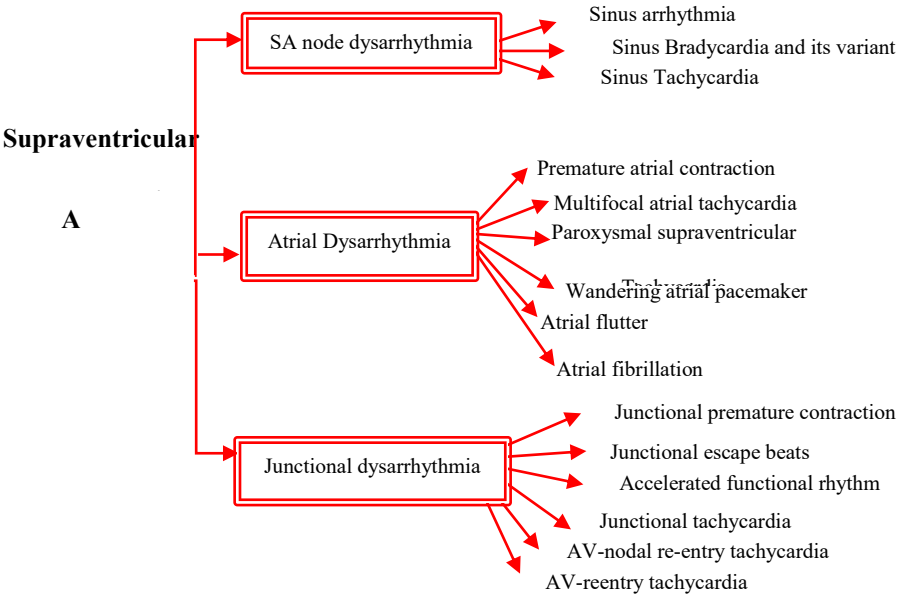
Arrhythmias are implying an abnormality of impulses formation or conduction, most of the arrhythmias produce abnormalities in the heart rate or rhythm or both.

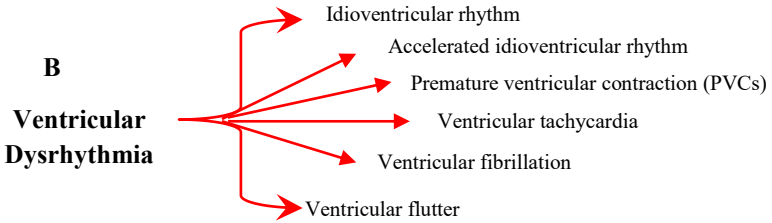


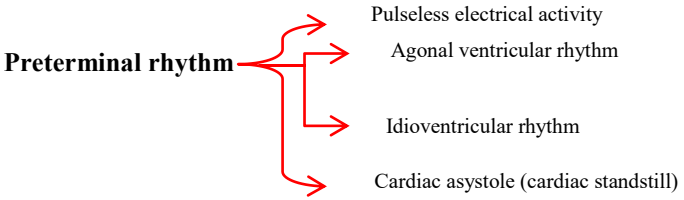

## I- According to the mechanism



## II- According to the site of the




## SA nodal arrhythmias

## 1. Sinus arrhythmia (Fig 4-2)

- Normal sinus P waves and PR intervals.
- Irregular PP or RR intervals, the variation between the shortest and longest interval is >0.16s (i.e. four small boxes).
- Every P wave followed by QRS complex. i.e. 1:1 AV conduction.
- It is normal finding in children and young adults tend to disappear with advanced age.
- During the long intervals of sinus dysrhythmia, junctional escape beats may occur.
- In the phasic variety, the SA rate accelerates during inspiration and decelerates during expiration.

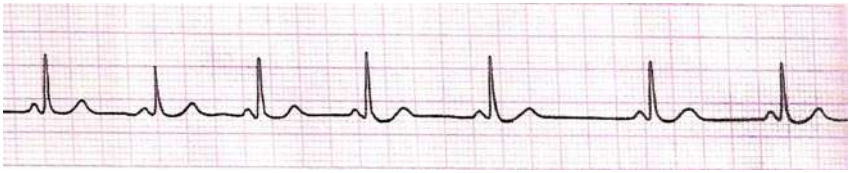


Fig (4-2) sinus arrhythmia

## 2. Sinus Bradycardia :-(Fig 4-3)

Is a sinus rhythm with heart rate less than 60 beats/min.

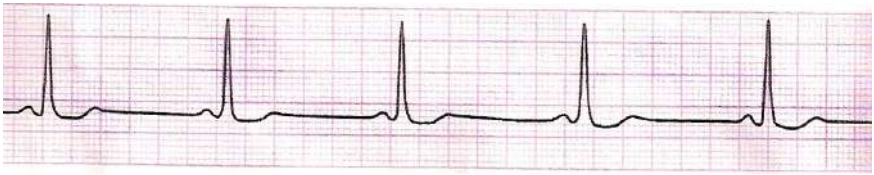


Fig (4-3) sinus Bradycardia

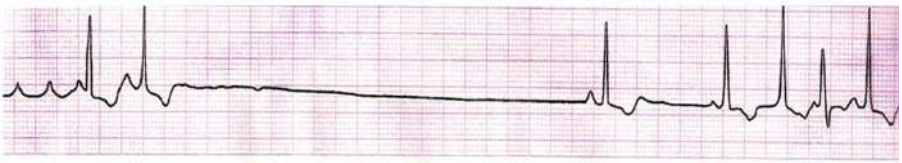
The following syndromes are among the causes of the Bradycardia:

Sick sinus syndrome(SSS):- (Fig 4-4A)

Due to degenerative changes and or ischemia of SA node, commonly in elderly the condition is characterized by a variety of arrhythmia as in the following:-

1. **Bradyarrhythmia**
   . Sinus Bradycardia.
   . SA block*.
   . AV block

2. **Tachyarrhythmia.**
   - PSVT-paroxysmal supraventricular tachycardia.
   - Paroxysmal atrial fibrillation.



(Fig 4-4A)SSS

***Sino-atrial block may be:**

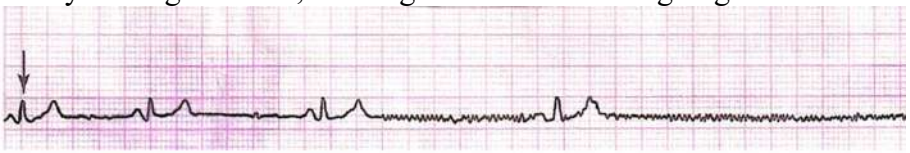
First degree SA block denote a prolonged conduction time from SA node to surrounding atrial tissue. It can not recognized on slandered ECG.

Second degree SA block denote the intermittent failure of conduction of sinus impulses to surrounding atrial tissue. Its manifested as intermittent absence of P waves (Fig.4-4B)

(Fig.4-4B) Second-degree sinoatrial exit block. Surface ECG denoting abrupt absence of P wave during sinus rhythm.

A third degree SA block or complete SA block is characteristic by lack of atrial activity on ECG its can not be distinguished from sinus arrest (Fig.4-4A)

A. Carotid Sinus Syndrome (Fig 4-5) usually occurs in elderly that may result in sinus bradycardia, is due to stimulation of the carotid sinus by turning the neck, wearing stiff collars or coughing.

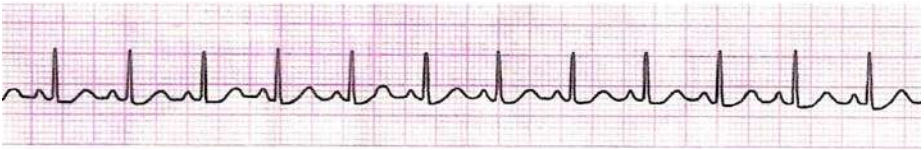


(Fig 4-5) carotid sinus syndrome

B. Neurocardiogenic (vasovagal) syndrome:- Bradycardia and/or vasodilatation, usually in young, adult, but may presenting for a first time in elderly.

## 3. Sinus tachycardia (Fig 4-6)

Is a sinus rhythm with heart rate above 100 beats/min.



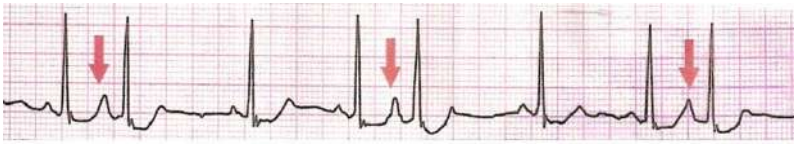
(Fig 4-6) sinus tachycardia

Note : Tachycardia in a sleeping patient may be caused by : - fever, bleeding and thyrotoxicosis.

# Atrial arrhythmias

## 1.Premature atrial contraction (Fig 4-7)

- P waves, ectopic P waves have different shape and axis, and appear sooner (prematurely) than next expected beat.
- PP intervals usually are irregular because of ectopic beats.
- PR intervals usually normal, but may be prolonged i.e. P wave unconducted (followed by pause) .
- QRS narrow: unless there is abnormal conduction.
- Usually followed by incomplete compensatory pause .



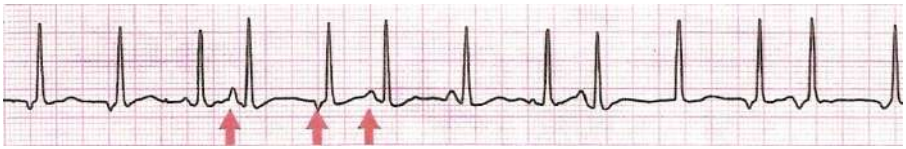
(Fig 4-7) premature atrial contraction

## 2. Multifocal Atrial Tachycardia (MAT) (Fig 4-8 A&B)

- Usually associated with severe pulmonary disease.
- Impulses originated irregularly and rapidly at different points in atria.

(Fig.4-8A)

- P waves shape, PR intervals, PP, and thus RR intervals all may vary.
- Atrial rhythm 150(100-180 beats/min).



**(Fig 4-8B) multifocal atrial tachycardia**

## 3. Paroxysmal Supraventricular Tachycardia (PSVT)

- It differs from sinus tachycardia in that the impulses are generated by ectopic foci or re-entry than sinus node.

**PSVT**

Ectopic SVT fig (4-9)

- Ectopic SVT usually originated in the atria.
- The regular P wave can be mistaken for atrial flutter or sinus rhythm.

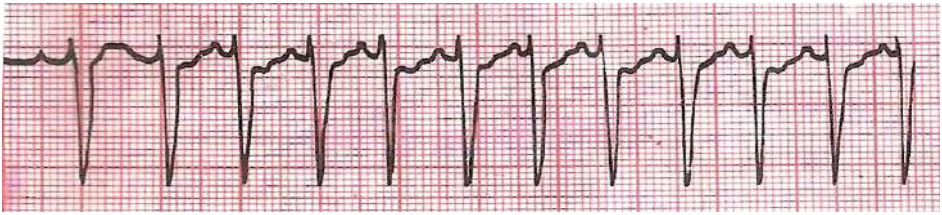
Reentrant SVT

- Within the AV node fig (4-17) (AVNRT)[1].
- With bypass tracts. Fig (4-20B) (AVRT) [2].

1. AVNRT= Atrioventricular nodal re-entry tachycardia.

2. AVRT=Atrioventricular Re-entry tachycardia

- It has sudden onset, sudden offset and short duration.
- P wave is not visible, abnormal shaped usually before QRS complexes but may be absent (fused with previous T wave).
- Heart rate greater than 100 beats/min usually between 150-250 beat/min.

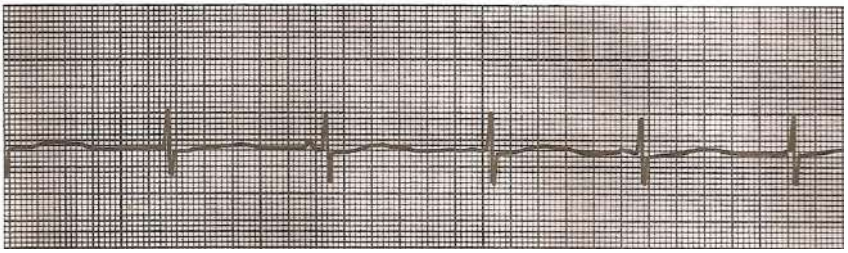


(Fig.4-9)

## 4. Wandering Atrial Pacemaker (WAP) (Fig 4-10)

- The impulses originate from different points in atria, called migratory impulse, at one time from the SA node and other time from ectopic site in the atria or from AV junction.
- The contour or shape of P waves varies from beat to beat in single lead, often associated with variation of PR, PP, and the RR interval. It seems likely that the site of atrial depolarization is varying (i.e. wandering atrial pacemaker).

▲ Example of wandering pacemaker.



(Fig 4-10)

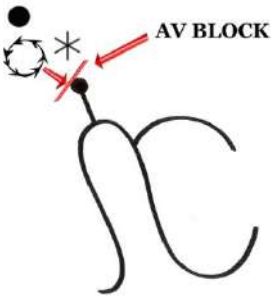
## 5.Atrial Flutter

*Is the second most common tachyarrhythmia.*

- It is usually paroxysmal, lasting from seconds to hours and occasionally even days.
- Pathophysiology.
    - It is the result of re-entry in the right atrium, rarely of left atrial re-entry.
- Atrial flutter can be classified into.

A. **Typical atrial flutter**:- (Fig 4-11A)

The re-entrant impulses travel in counter clockwise regular rotation (Down the right atrial free wall and up the intraatrial septum).

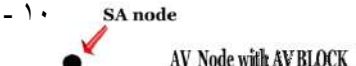



(Fig.4-11A)

B. **Atypical atrial flutter**:- (Fig 4-11B)

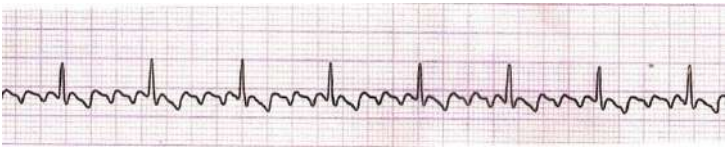
The re-entrant impulses travel in clockwise regular fashion.





*The AV node acts as gatekeeper to prevent those rapid impulses from reaching the ventricles by block one out of two atrial impulses (2:1), two of three (3:1), three of four (4:1).*

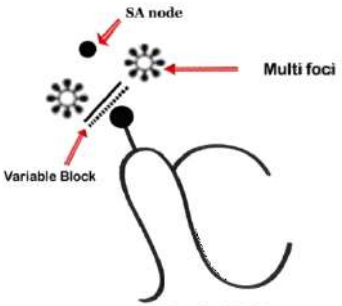
- **The ECG characteristic**:- (Fig 4-11C)
    - Typical flutter shows the classic negatively directed saw-tooth in the inferior lead, II, III, aVF, no isoelectric baseline is present.
    - Atypical flutter shows positive atrial depolarization in the inferior leads.
    - QRS complexes have normal duration, although aberrant conduction could be occurring.
    - The ventricular response can be irregularly irregular due to varying degree of block (2:1, 4:1, etc), but is more typically regular.
    - If the F wave are not clearly visible it is worth accentuating them by slowing AV conduction by carotid sinus massage or by administration AV nodal blocking drugs such as adenosine or verapamil.



(Fig.4-11C)

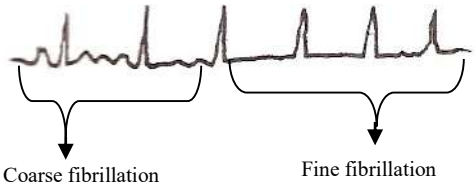
## 6. Atrial Fibrillation

- The most common sustained arrhythmia.
- It occurs in :-
  A. Paroxysmal pattern (an episode lasting minutes to hours).
  B. Persistent pattern (an episode lasting two days-weeks can be terminated by electrical or chemical cardio version).
  C. Permanent pattern (an episode lasting months to years, no expectation of restoring sinus rhythm).
- Pathophysiology (Fig 4-12A)
  Two theories existed.
  - Enhanced automaticity involving one or more foci firing rapidly.
  - Multiple re-entrant wavelet hypotheses.



(Fig.4-12A)

- The ECG characteristic (Fig.4-12B):-
  A- P waves are absent.
  B- ECG baseline coarsely or finely irregular



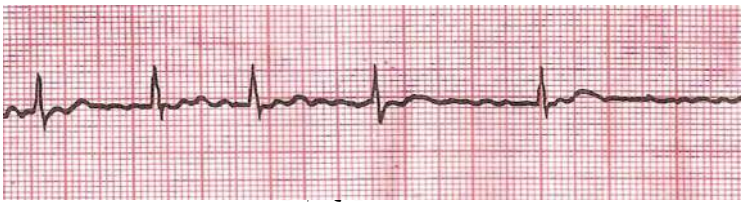

(Fig 4-12 B)

C- Ventricular rhythm is quite irregularly, irregular, slow (Fig 4-12C) or rapid (Fig 4-12D), because the ventricles are stimulated in random fashion due to variable block of rapid atrial impulses at AV node (in case of atrial flutter the block are usually constant)

***Practical points***

In case of atrial fibrillation if the ventricular response became regular it may suggested the following possibilities:-

- Conversion to sinus rhythm.
- Conversion to atrial flutter.
- Complete AV block (the response is slow and regular).
- Digitalis toxicity (4-12E).



Fig(4-12C) Atrial fibrillation with irregular slow ventricular response

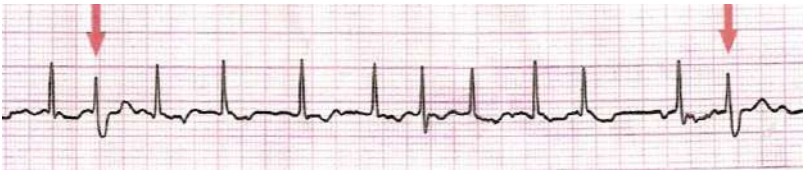
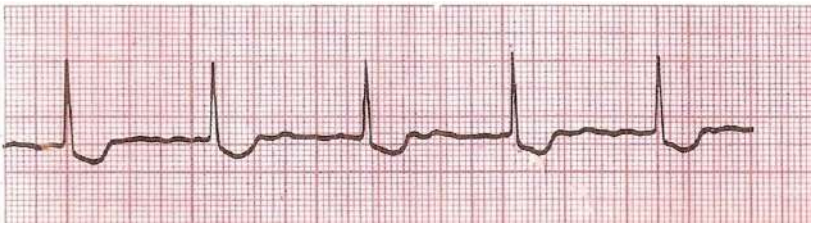


Fig (4-12D) Atrial fibrillation with irregular rapid ventricular response



(Fig 4-12E) Atrial fibrillation and digitalis effect

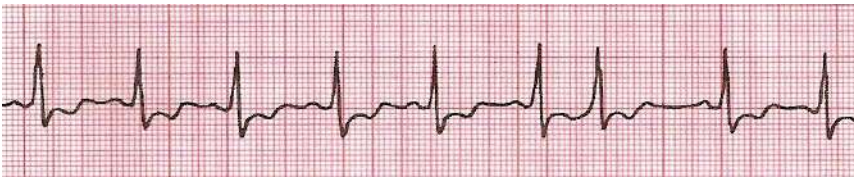
***Practical point***

Unexplained AF (or sinus tachycardia at rest) should prompt a search for hyperthyroidism.

## Junctional Dysarrhythmia

### 1. Junctional premature contraction (fig.4-13)

- P wave inverted may before or after QRS.
- Narrow QRS complex unless there is conduction abnormality.
- May be isolated, multiple (as in bigeminy, trigeminy, unifocal, multifocal).



(Fig 4-13)

## 2.Junctional escape beats (40-60 beats/min) (fig.4-14)

- Usually occur late, not premature, when there is SA node arrest.
- P wave, often inverted, may be buried in QRS or follow QRS complexes.
- QRS narrow unless there is conduction disturbance.
- Rate slow.

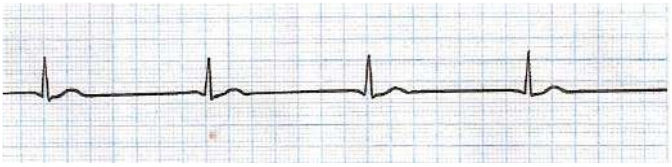


Fig (4-14)

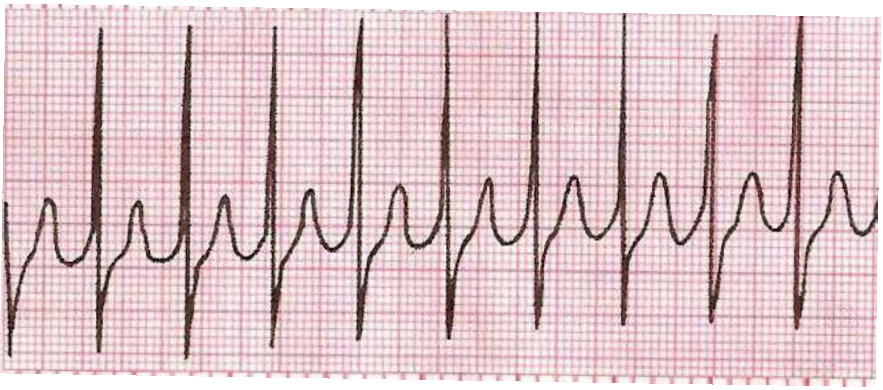
## 3. Accelerated junction rhythm

- Rate (60-100 beats/min).

- P wave, inverted, buried in or follow QRS complexes.
- QRS complexes narrow, unless associated with conductions abnormality.

### 4. Junctional tachycardia (fig.4-15)

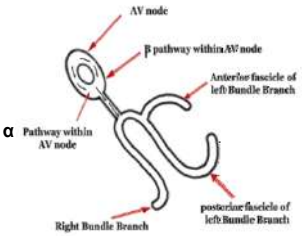
- Rate (>100 beat/min).
- P wave usually not visible.
- QRS complexes narrow, unless there is conduction problem.



(Fig.4-15)

### 5.AV- node re-entry tachycardia mechanism (AVNRT)

A- Under normal circumstances the P impulses should proceed from AV node downward, passing antegrade from up-down in both alpha and β pathway in relatively uniform fashion.

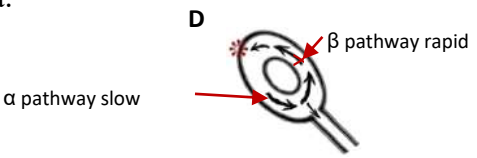


A

B- The impulses are blocked from proceeding down through β pathway due to ischemia.


C- The impulses that pass through α pathway is then able to return back through β pathway and able to re-excite of that area.


D- The cycle is repeated over and over again rapidly produces so called re-entry tachycardia.




***Note:-***

## *ECG of AV nodal re-entry tachycardia (AVNRT ) (Fig.4-16 &4-17)*

- Atrial rate 160 to 220 beat/min.
- P wave regular and often inverted.
- QRS usually regular sometimes irregular.

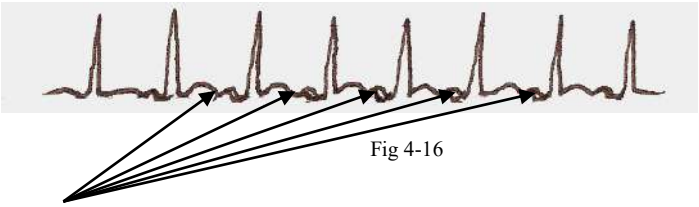


Fig 4-16

- P wave usually precede the QRS complex because the impulses depolarize atria before the ventricles.

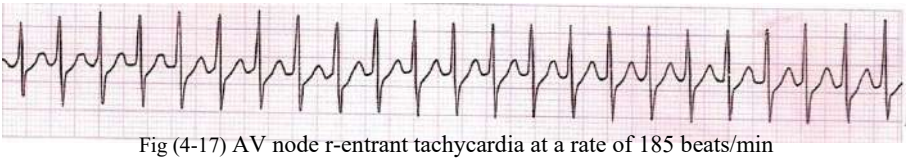


Fig (4-17) AV node r-entrant tachycardia at a rate of 185 beats/min

**Note** In 10% of the person the block occur in α pathway (slow pathway) then the impulse depolarize the ventricle before the atrium, resulting in a retrograde P wave that occurs after the T wave (long PR tachycardia) (Fig 4-18&4-19)

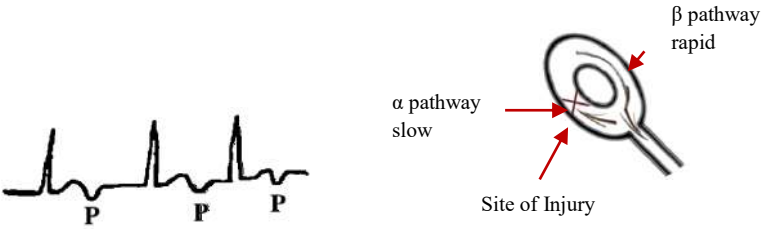



Fig (4-18)

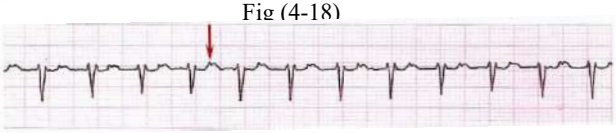


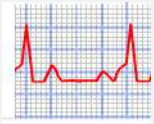
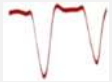
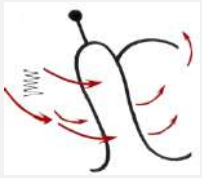
Fig (4-19)

AV node re-entrant tachycardia at a rate of 146 beats/min the retrograde P waves are clearly seen altering the normal T wave contour (arrow)

## ATRIOVENTRICULAR-RECIPROCATING TACHYCARDIA (AVRT) (PREEXCITION SYNDROME)

- Normally the AV node is the only way to conduct the impulses from atria to the ventricles.
- Accessory pathway (bundle of Kent) are abnormal inheritance tract conduct the impulses very rapidly from the atria to the ventricles, it's bypass AV node, resulting in early activation of the ventricles (preexcitation).
- WPW-syndrome is common in men.
- WPW-syndrome and LGL syndrome account the most common causes of preexcitation syndrome.

**Wolf-Parkinson-white syndrome (WPW-syndrome classically classified into three types):-**

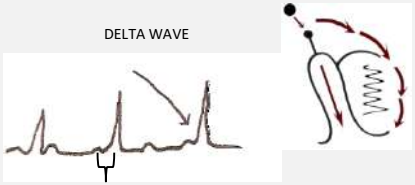
| ***Type (A) WPW syndrome*** *(See Fig. Below)* | ***Type (B) WPW syndrome*** *(See Fig. Below)* | ***Type (C) WPW syndrome*** |
|---|---|---|
| *1. It is the most common type.*<br>*2. Impulses travel through the left bundle of kent.*<br>*3. The left ventricle depolarize first then the septum lastly the right ventricle.*<br>*4. Produce upright R wave in right precordial leads V1-V2*<br>   | *1. Impulses are travel through the right bundle of kent.*<br>*2. The right ventricle depolarize firstly then the septum lastly the left ventricle.*<br>*3. Produce Negative QRS in the right precordial lead V1-V2*<br>   <br>*V1 V2* | *1.Has been suggested that the accessory pathway may found in the area of interventricular septum.*<br>*2.It's difficult to identify on ECG and its morphology often identical to LGL syndrome* |

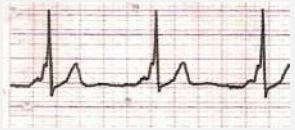
# ECG PATTERNS IN WPW-SYNDROME

***Sinus rhythm***

1. *The ventricles are partially depolarized by both AV node and accessory pathway.*
2. *ECG (Fig.4-20A)*

- *Short PR interval.*
- *Delta wave.*
- *Wide QRS complex*



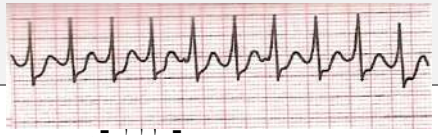



(Fig 4-20A)

***Re-entrant orthodromic tachycardia.***

1. *It is the most common.*
2. *The ventricles activate by the normal AV node the impulses conducted antegrade through AV node and retrograde through the accessory pathway. (Fig.4-20B)*

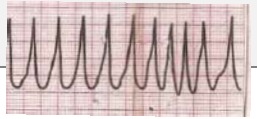

*ECG narrow complex tachycardia with loss of Delta wave*



(Fig 4-20B)

***Re-entrant antidromic tachycardia with atrial fibrillation.***

1. *It is rare.*
2. *The ventricles activated by the accessory pathway the impulses bypass the AV node rapidly produces preexcitation of the ventricles.(Fig.4-20C)*

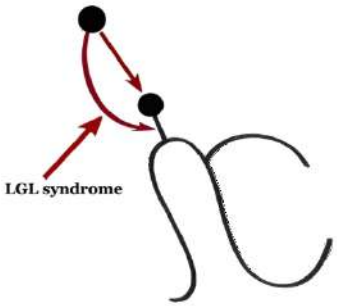
| | | |
|---|---|---|
| | | (Fig 4-20C) |

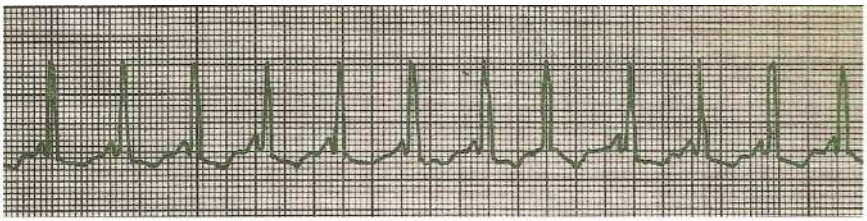
## II. Lown-Ganong-Levine syndrome (LGL) (Fig.4-21)

In the LGL syndrome the accessory pathway is the extension of the normal intranodal conduction pathway between the atria and bundle of His then the conduction to the ventricles from bundle of His is normal.

ECG see fig below

- Normal QRS complexes.
- No delta wave.
- Show PR interval.



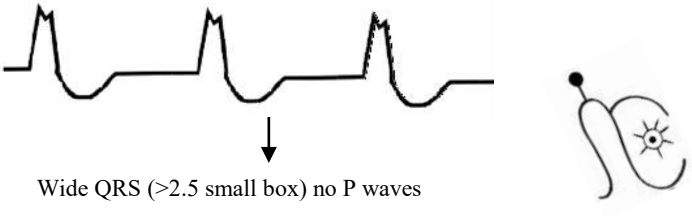
(Fig 4-21)

**Note:-** A relatively short PR interval may be seen as a normal variant, without a bypass tract, because of accelerated AV conduction, therefore you should not "overread" an ECG on which the only note worthy finding is short PR interval.

# VENTRICULAR DYSARRHYTHMIAS

## 1. Idioventricular Rhythm (Fig 4-22)

- The term idio indicates the ability for self initiating and self containing an impulse.
- Ventricular myocardium assume the pacemaker role, when the primary pacemaker fails or the conduction between atria and ventricles is blocked the resultant ventricular escape rhythm usually at rate 30-40 beats/min.
- ECG Characteristics(Fig.4-22)
  - P wave absent, or not related to the QRS complexes.
  - wide QRS complexes, typically T wave will be opposite the direction to that of QRS.



Wide QRS (>2.5 small box) no P waves

Fig 4-22

## 2. Accelerated Idioventricular Rhythm (AIVR)

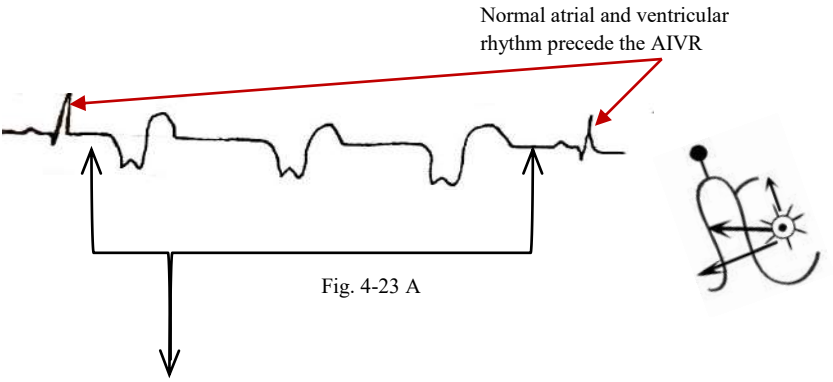



Fig. 4-23 A

Short bursts (usually <20 seconds) of AIVR often a few days after successful reperfusion therapy of myocardial infarction, it is usually Asymptomatic with no progression to ventricular tachycardia or ventricular fibrillation.

*ECG criteria.*

- Wide and regular QRS complexes (>0.10 second).
- Absence of P wave.
- A rate usually between 40-100 beats/min

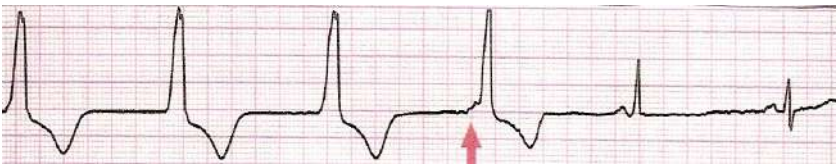


Fig (4-23B)

Look the AIVR usually not preceding by a P wave but the fourth complex is preceded by a P wave and the subsequent QRS is slightly narrower than those preceding it, the following QRS are sinus bradycardia with normal AV conduction.

## 3.Premature Ventricular Contractions (PVCs) (Fig. 4-24)

- PVCs are one of the most common of all Dysarrhythmia, and the most common of all ventricular dysrhythmia, also is the most common dysrhythmia after the MI.
- PVCs may occur normally or with organic heart disease
- ECG characteristic of PVCs

1. Occur early before sinus beat is expected.(Fig.4-24A)

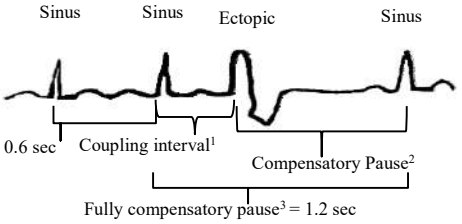



**Fig. 4-24A**

1. Coupling interval is applied to the interval between PVCs and the preceding normal beat.
2. Compensatory pause:- is the pause between PVCs and the next normal beat.
3. Fully compensatory pause:- is applied to the interval between the two sinus beat that surround the PVC which equal twice the normal RR interval (2×RR).

2. QRS complexes usually >0.12 second and the T wave point in opposite QRS direction.(Fig.4-24B)

**Fig. 4-24B**

3. PVCs may appear with varying following frequency and shape:-

a- Couplet:- two consecutive premature beats.(Fig.4-24C)

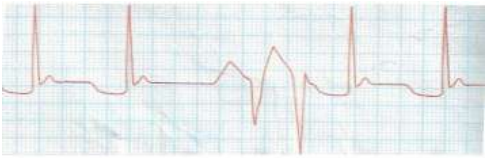


Fig (4-24C) couplet

b- Bigeminy: every sinus beat is followed by premature beat (Fig.4-24D)

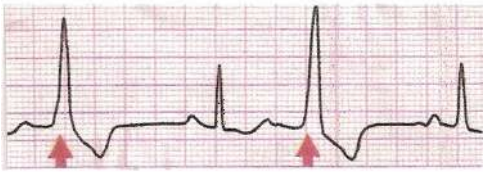


Fig (4-24D) Bigeminy uniform PVCs

c- Trigeminy: every second sinus beat is followed by premature

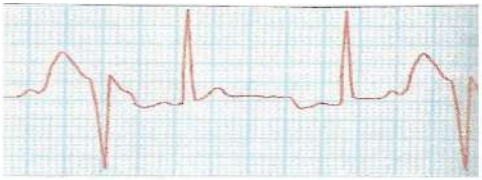


Fig (4-24E) Trigeminy VPCs

d- Quadrigeminy: every three sinus beat is followed by premature beat.

(Fig.4-24F)

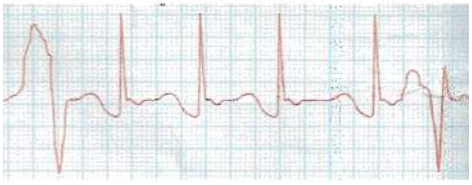


Fig (4-24F) quadrigeminy

e- Three or more consecutive premature beats constitutes ventricular tachycardia.(Fig.4-24G)

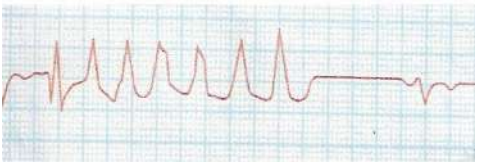
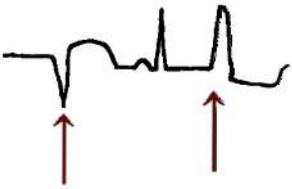


Fig (4-24G) non sustained VT

f- A uniform VPCs: with same shape in single lead. (Fig.4-24G)

g- A multiform VPCs: with a different shape in same lead. (Fig.4-24H)



(Fig.4-24H) Bigemany Multiform PVCS

h- Interpolated: premature beat occur between two normal beats, which may not followed by a pause as usual PVCs.(Fig.4-24I)

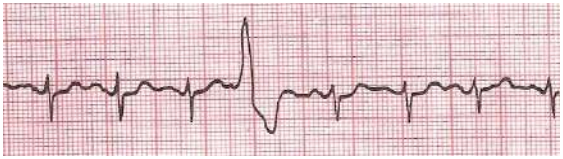


Fig (4-24I)

i- Multifocal: premature beats originating from two or more different ventricular locations.

## Clinical significance of the VPCs

1. In healthy subject:-
   - Frequently found in normal people, increase with age.
   - More prominent at rest and tend to disappear with exercise. In contrast to AF which precipitated by exercise
   - The outlook is excellent and treatment is unnecessary, although low dose beta blocker is sometimes used to suppress anxiety and palpitation.
   - Even in healthy subject the VPCs may be a manifestation of subclinical coronary artery disease in this regard no evidence that antiarrhythmic therapy is beneficial in such patient, but the discovery of frequent VPCs might reasonably prompt some general cardiac investigation.
2. With heart disease
   - Frequent VPCs during acute MI are of no prognostic significant, and require no treatment, but in patient who have survived the acute phase of MI the following criteria may indicated poor prognosis:-

     - More than 6-8/min
     - Multifocal. (Fig.4-24H)
     - Three or more successive beats(non sustained ventricular tachycardia). (Fig.4-24G)
     - R on T phenomenon (R wave of the ectopic beat on the T wave of preceding sinus beat) (Fig.4-25)

- Unfortunately antiarrhythmic therapy does not improve, and may even worsen the prognosis in that patient.

- In patient with heart failure are associated with poor prognosis, but again the outlook is not change if they are suppressed by antiarrhythmic drugs, effective treatment of heart failure may suppress the ectopic beats.
- VPCs are also feature of digoxin toxicity.
- Sometimes occurs in mitral valve prolaps.

Anyway the treatment of PVCs is directed toward underlying cause

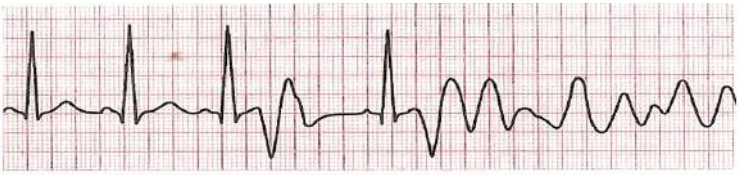


Fig.4-25 Ventricular ectopic beats interrupting the T wave of preceding beats (R-on-T phenomenon). The second such beat initiates ventricular fibrillation.

## Parasystole (Fig. 4-26)

- parasystole is a special type of rhythm produced by cardiac cells, that functioning as a protective natural pacemaker without the capability to sense the surrounding cardiac activity it has the following criteria:-
  1. Variable intervals between premature ventricular contraction and preceding sinus beat (coupling interval) in contrast to the PVCs which has fixed coupling interval. (Figs.4-24A& 4-26)
  2. Constant interectopic intervals.
  3. An occasionally fusion beat.
  4. Unchanged QRS configuration of the parasystolic ventricular premature complex.

**Clinical significant**

- **Parasystole is uncommon arrhythmia.**
- **Generally it is benign.**

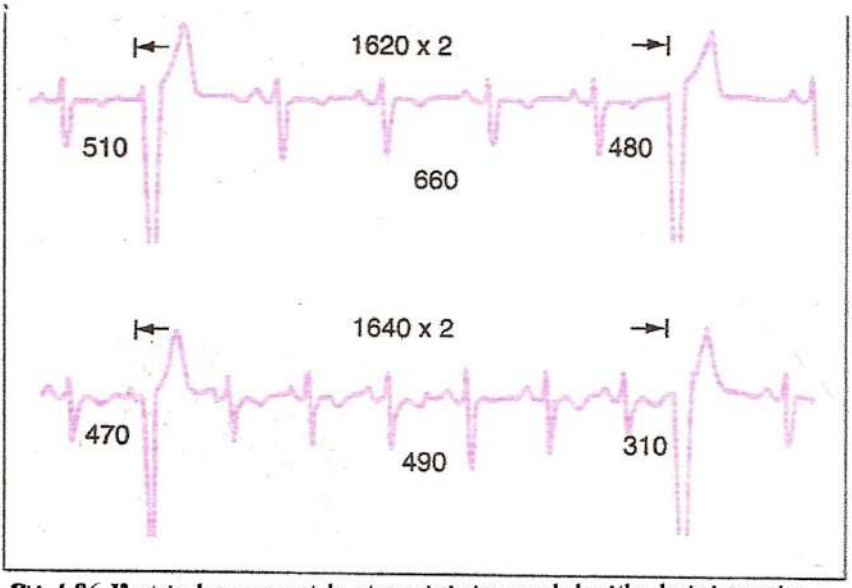


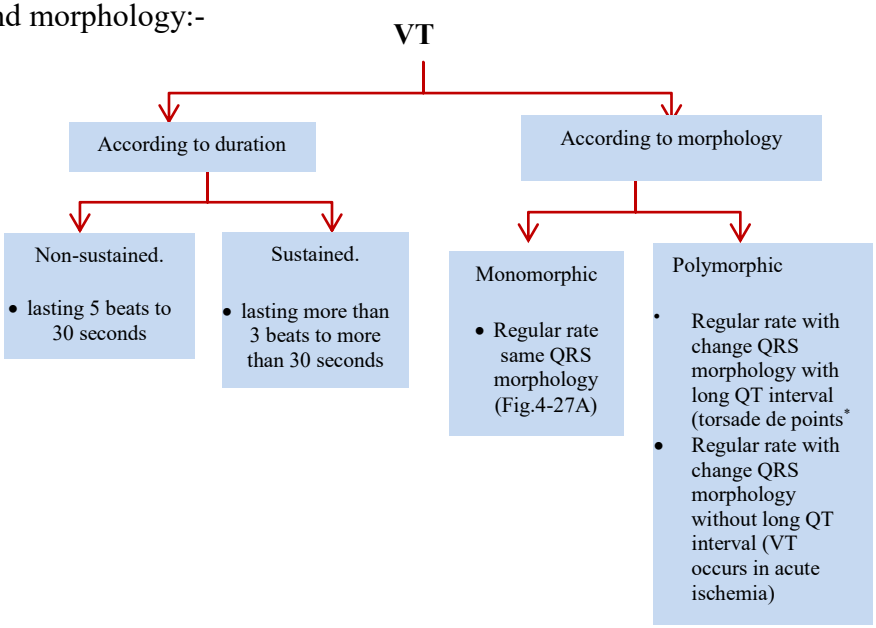

**Fig. 4-26. Ventricular parasystole. at varying sinus cycle lengths during exercise, interectopic intervals remain constant at 1620-1640ms. however; the coupling intervals between sinus and ectopic complexes vary between 510 & 310 ms.**

## 4.Ventricular Tachycardia

ECG characteristic of ventricular tachycardia:-

1. Wide QRS complexes (>0.14sec).
2. Rate greater than 100 beats/min, usually between 150-200 beats/min.
3. Rhythm usually regular, although there may be some beat-to-beat variations.
4. QRS axis usually constant.

The ventricular tachycardia generally classified according to the duration, and morphology:-

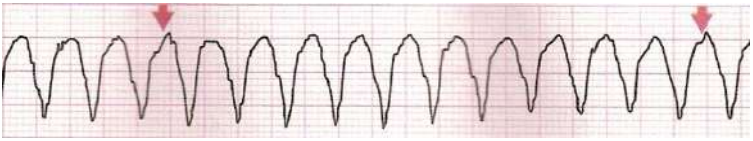



***=Torsades depoints (twisting points)**

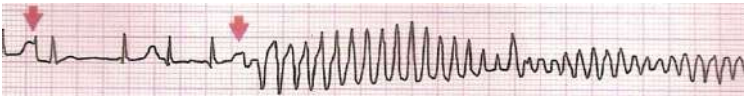
This form of the polymorphic VT, with long QT interval, is characteristic by rapid irregular complexes that oscillate from upright to an inverted position, and seen to twist around the baseline as the mean QRS axis changes.(Fig.4-27B)

***Ventricular tachycardia needs to be distinguished from other cause of wide complexes tachycardia as supraventricular tachycardia with aberrant conduction*:-**

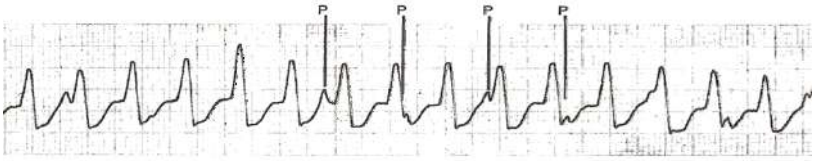
1. In cases of doubt ventricular tachycardia (VT) should be diagnosed because it is safer to treat VT which is by far the commonest cause of broad QRS complexes, including in old age or known case of cardiac disease patients. A good general practical role is that broad complex tachycardia is always assumed to be VT unless proven other wise.
2. Ventricular tachycardia is more likely when there are:-
   A. A very broad QRS (>0.14s).
   B. Atrioventricular dissociation (pathognomonic) (Fig.4-27C).
   C. Concordant same polarity QRS in all chest lead (V1-V6) (Fig.4-27A & D).
   D. Capture/ fusion beats (pathognomonic) (Fig.4-27D & E).
   E. No response to carotid sinus massage or IV adenosine.



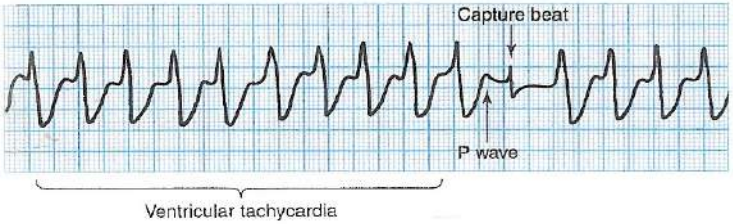
(Fig.4-27A) monomorphic ventricular tachycardia, P waves are seen to be occasionally alter QRS morphology (AV dissociation) (Arrows)



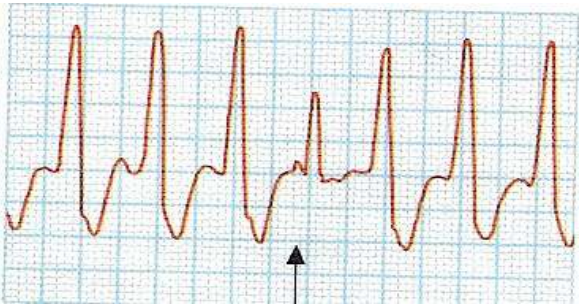
(Fig.4-27B) polymorphic ventricular tachycardia,(Torsades de point) And several QRS complexes occur at the peak of preceding T waves (Arrows) R-on-T that initiate VT.




(Fig. 4-27C) AV dissociation. The regular, independent sinus P waves during the tachycardia can be seen. The P waves are easily found when a distortion is seen in the QRS, ST segment, or T wave of one cycle that is not seen in another.




(Fig. 4-27D) capture beat occur when atrial impulse stimulate (capture) the ventricles causing normal QRS complex preceded by P wave.

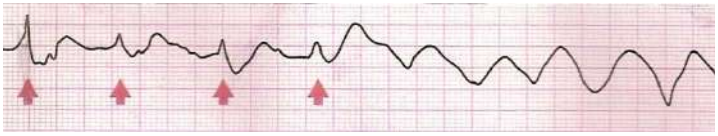


(Fig. 4-27E). fusion beat' the normal sinus conducted impulses fuses with an impulse from the tachycardia.

i.e. the ventricles are activated by 2 impulses from the atrial and from the Ectopic forces at the same time. The 2 impulses fuse together produce a beat with deformity appearance that looks neither like the normal beat nor the Ectopic ventricular beat called (fusion beat)

## 5.Ventricular fibrillation (VF) (Fig. 4-28)

- No clear P wave or QRS complexes, rate usually in determined the usual ECG manifestation is A fine to coarse zigzag pattern.



(Fig.4-28) VF an agonal rhythm is initially present (arrows) but deteriorate into VF

## 6.Ventricular flutter (Fig. 4-29)

Very rapid ventricular tachycardia that give modified pattern on ECG (Regular zigzag) without clearly formed QRS without definite demarcation between QRS and T waves.

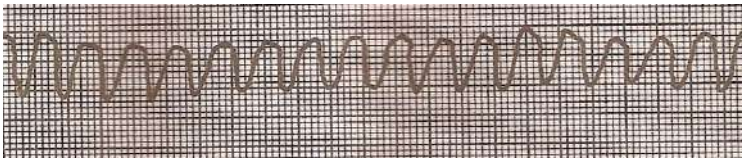


Fig (4-29)

## Differential Diagnosis between Ectopic and Escape Rhythm

| | Ectopic beat<br><br>Fig (4-30 A) | Escape Beat<br><br>Fig (4-30B) |
|---|---|---|
| mechanism | Premature contraction are probably manifestation of un usual irritability of Automatic cells. | Occurs as a safety mechanism when the sinus or other dominant rhythm temporarily pause. |
| Onset | Usually occur early before the second sinus beat is expected. | Occur late after a pause |
| Site of origin | -Atria<br><br>-Junctional<br><br>-Ventricular | -Atria<br><br>-Junctional<br><br>-Ventricular |

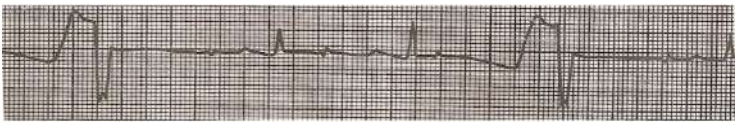


Fig (4-30A) Ventricular Ectopic Beat

Fig (4-30B) Ventricular Escape Beat

## Preterminal rhythm

1. *Agonal rhythm*: -Is the occurrence of very broad and irregular ventricular complexes at a slow rate, usually without associated ventricular contractions.(Fig.4-31A)

   Agonal rhythm

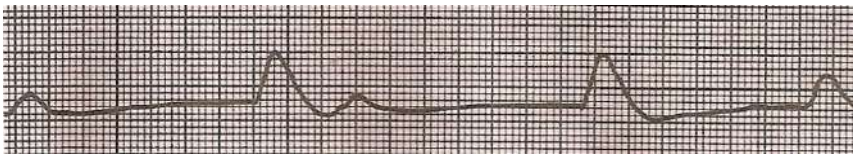


Fig (4-31A)

2. *A systole*: asystole implies there is no spontaneous cardiac activity, and thus no QRS complexes shown on the ECG.(Fig.4-31B)

   A systole

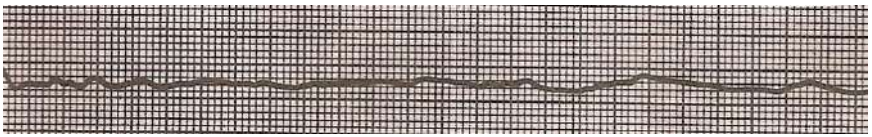


Fig (4-31 B) No spontaneous electrical activity and thus no QRS complexes.

3. *Pulseless electrical activity (PEA)* PEA is some times called electromechanical dissociation and occurs when the heart continues to work electrically (the ECG continues to show QRS complexes with any of cardiac rhythm even sinus rhythm) but fails to provide a circulation.
4. *Idioventricular rhythm (IVR).*

## Atrioventricular Conduction Disturbance

- The atrioventricular conduction is assessed by examining the relationship between the P waves and QRS complexes.

The following questions are whether P waves are:

1. Always related to the QRS complexes.
2. Sometimes related to the QRS complexes.
3. Never related to the QRS complexes.

## 1.P wave always related to the QRS

P waves always preceding QRS complexes but PR interval prolonged >0.20sec (>5 small boxes) (Fig.4-32A)

Fixed but prolonged PR interval

↓

First degree heart block

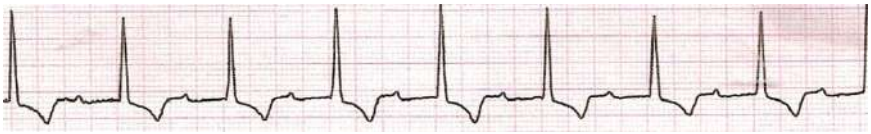
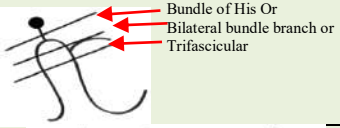
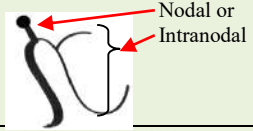
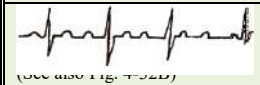
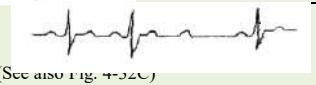


Fig (4-32A)

## 2. P wave sometimes related to the QRS complexes

| | | | |
|---|---|---|---|
| Type of the block | Second degree heart block Mobitz type 1 (Wenckebach) | Second degree heart block Mobitz type 2 | Second degree heart block 1:1 or 2:1 advanced block this block is neither Mobitz I nor II |
| Pattern of block | Progressive lengthening of PR interval with intermittent dropped beat | Sudden dropped QRS without prior prolonged | Occurs when every second or third P waves conduct to the ventricle |
| location | AV node  |   |   |
| ECG |  (See also Fig. 4-32D) |  (See also Fig. 4-32C) | Depends on setting in which occurs see (See also Fig. 4-32D) |
| Occurrence with acute MI | Usually inferior MI | Usually anterior MI | Depends on setting in which occurs |
| Risk progression to complete heart block | Low | High | |
| Indication or permanent passing | Not usually | usually | |

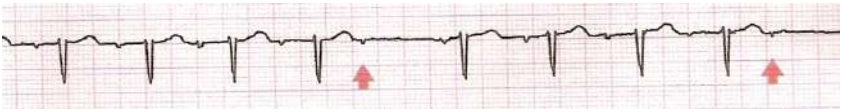
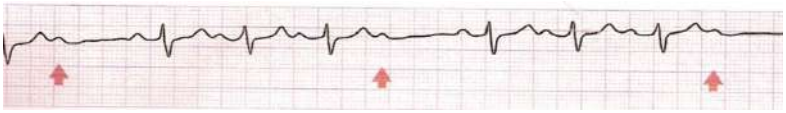
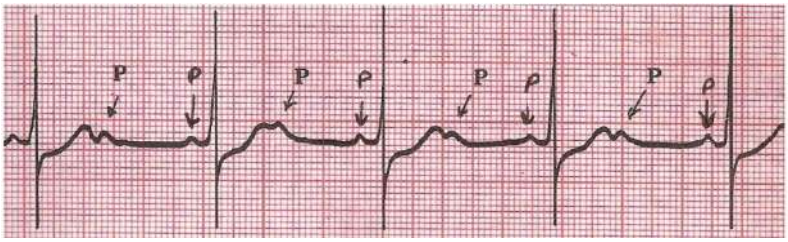


Fig (4-32B) second degree heart block mobtiz type I



Fig(4-32C) second degree heart block




**Second degree block (2:1 type)**

Fig (4-32D) second degree heart block with fixed 2:1 block; some P waves or alternative P waves are not conducted. It's difficult to differentiate this type of second degree AV block is neither Mobitz I nor II.

## 3. P wave never related to QRS

QRS rate is slower than P rate (i.e. ventricular rate slower than atrial rate)

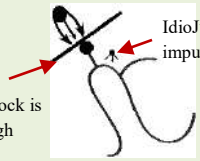
**Third degree(complete) AV block**

QRS rate faster than P rate (i.e. ventricular rate faster than atrial rate)

**AV dissociation**

### AV nodal block

SA node




1). Impulses originated in SA node and below the site of block (idiojunctional)

2). Atria and ventricles depolarize independently

3). The QRS:-

A. less frequent.

B. Regular.

C. at rate 40-60/min.

D. normal shape

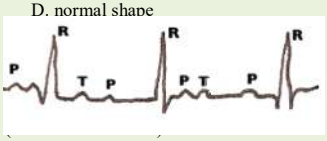
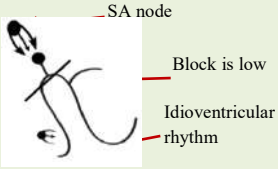



Fig (4-33A)

### Infranodal block




1). Impulses originated at both SA node (P wave) and also below site of block (idioventricular).

2). Atria and ventricles depolarize independently

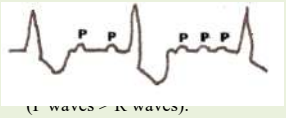
3). The QRS:-

A. less frequent.

B. Regular.

C. at rate 20-40/min

D. Wide abnormal shape




(P waves > R waves).

Fig(4-33B)

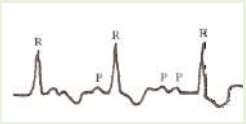
4). Wide complexes complete block is quite dangerous

### AV Dissociation

1). Slower supraventricular rhythm.

2). Rapid ventricular rhythm, which cannot conduct retrograde to atria or shut off sinus.

3). P waves less frequent than QRS complexes (opposite complete heart block) and totally unrelated to them




(R wave >P wave).

Fig.(4-33C)

4). AV dissociation implies more than complete heart block (i.e. heart block may

4). Acute onset, narrow complexes block may be transient and may response to the atropine.

5). Chronic narrow complexes is may be congenital require pacing if symptomatic

Dizziness and blackout (stoke-Adams attacks) often occur.

5). In young usually caused by IHD.

6). In elderly usually caused by fibrosis of distal conduction system (lev's disease)

be considered as a part from AV dissociation).

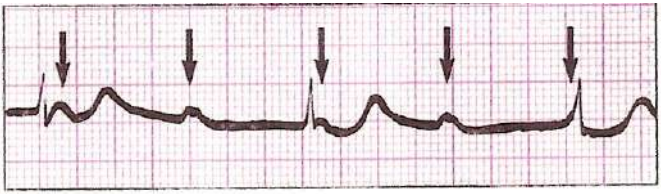
5). In AV dissociation some P wave may be conducted through the AV node, whereas with complete heart block, no P waves reaches the ventricles. The following are among the cause of AV Dissociation.

1- may be physiological

fig(4-33C) .

2- complete heart block .

3- Atrial flutter, Atrial fibrillation.



(Fig.4-33A) Bradycardia with complete heart block narrow complexes idiojunctional escape rhythm, this patient has a more rapid rate (40-50 beats/min) than patient in fig (B) the level of block is probably the AV node.

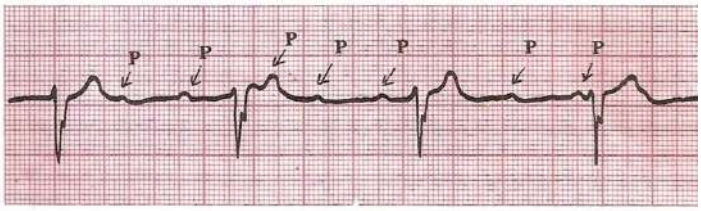
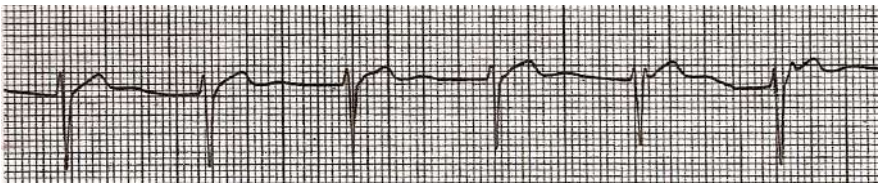



Fig (4-33B) Bradycardia with complete heart block wide complexes idioventricular rhythm this patient has slow rate 38 bpm comparing with the patient above indicating infra-nodal block



(Fig.4-33C) This type of (AV) dissociation is characterized by transient desynchronization of the SA and AV node; the P waves appear to slide in and out of the QRS. This type of AV dissociation must be distinguished from AV dissociation caused by complete AV block.

## Practical diagnosting approach for common tachy-brady-arhythmias

1. Tachyarrhythmia

A- If the heart rate above 100 bpm and regular the following differential diagnosis should be considered:-

- Sinus tachycardia

-Ventricular tachycardia.

- Atrial flutter Fixed 2-1 block.

B- If the heart rate above 100 bpm and irregular the following differential diagnosis should be considered.

- Ventricular ectopic beat (may occur as irregularly irregular if multiple or regular irregular when arise after many normal QRS).

- Atrial fibrillation (its total chaotic abnormal rhythm).

- Atrial flutter with varying block.

- Sinus arrhythmia (see Fig.4-2) occurs as regular irregular.

C- If the heart rate above 100 bpm with normal QRS duration (*narrow complex tachycardia*) the following differential diagnosis should be considered:-

- Sinus tachycardia.

- Paroxysmal supraventricular tachycardia.

- Atrial flutter.

- Atrial fibrillation.

- A-V re-entery tachycardia.

D- If the heart rate above 100 bpm with QRS duration more than three small squares (wide complex tachycardia) the following differential diagnosis should be considered:-

- Ventricular tachycardia.

- Supraventricular tachycardia with aberrant conduction.

2. Brady arrhythmia

A- If the heart rate below 60 bpm with normal QRS duration the following differential diagnosis should be considered:-

- Sinus bradycardia.

- Sinoatrial block.

- high nodal complete AV block (see Fig.4-33A) .

- First and second degree heart block.

B- If the heart rate below 60 bpm with QRS duration more than three small squares, the following differential diagnosis should be considered:-

- Infra-nodal complete heart block (see Fig.4-33B) and AV dissociation.

- Escape rhythm (mainly ventricular).

- Drug induced conduction defect.

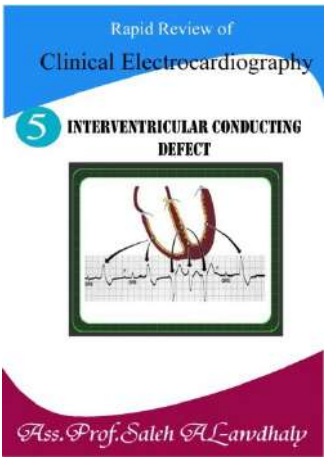
***As a general rule in wide complex tachycardia the IV administration of verapamil should not be done until the cause is clarified.***

# Chapter 5

# Intraventricular

# Conduction

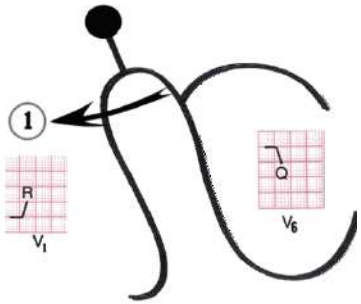
# Defect



# *Intraventricular Conduction Defect*

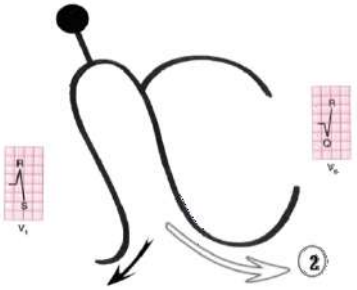
## The sequence of normal ventricular

- *The normal septal activation from the left to right, produces R wave in V1 and small septal q wave in V6 (Fig. 5-1A) .*




*(Fig 5-1A) Arrow 1 represent the normal septal depolarization*

- *The left and right ventricles depolarized simultaneously, the left ventricle is much thicker than the right, generate more voltage in the left precordial leads produces deep S wave in (V1) and tall R wave in (V6) (Fig. 5-1B).*

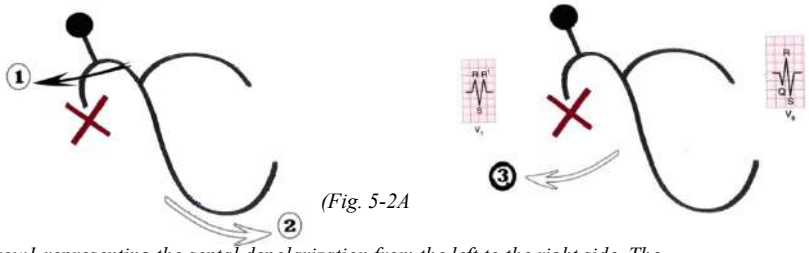



*(Fig 5-1B) ventricular depolarization; the large and small arrow represent the left and right ventricular electrical force respectively the left ventricle is responsible for most of the heart forces (arrow 2)*

*The result of normal depolarization of ventricle is normal QRS in morphology and duration, preceding by normal P waves and PR interval , when this QRS is differ in morphology and/or wide than normal , with normal a trial P wave and PR duration is the basic diagnostic criteria for bundle branch block .*

## *A- Right bundle branch block (RBBB)*

*Remember in RBBB the Activation of septum and LV is the same as normal (Fig. 5-2A). only the terminal period of depolarization is effected, because the right bundle branch is blocked. current must move from the left ventricle to the right and this occur late produces new R wave in right precordial leads mainly (VI) and deep s wave in left precordial leads V5, V6.*




*(Fig. 5-2A*

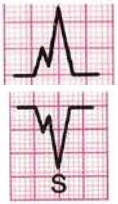
*An arrow1 representing the septal depolarization from the left to the right side. The second phase involves depolarization of the left ventricle (arrow 2).*
*The (late) depolarization forces travel toward the right ventricle (arrow 3) this causes the last part of the wide QRS complex to be positive (R wave) in lead V1 and negative (S wave) in leads V5, V6.*

## ECG criteria for complete RBBB (Fig. 5-2B)

1- *QRS duration ≥ 0.12 sec.*

2- *Broad notched Ŕ wave in VI*

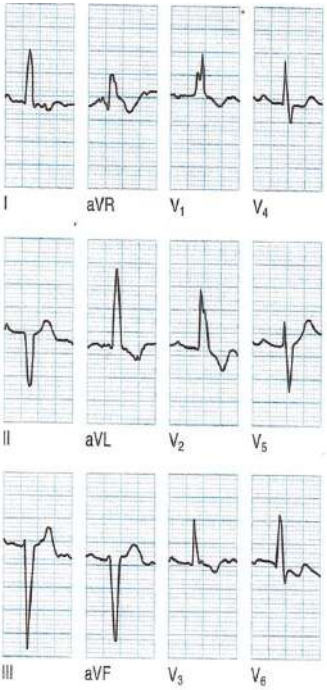
3- *Deep S wave in V5-V6*




4- *ST-T wave inversion in (VI-V2) but ıf ınvertea ın the left precordial leads V5-V6 may indicate ischemia.*

5- *Normal septal q wave in left precordial leads*

RBBB recognition RSR pattern in V1 and QRS ≥

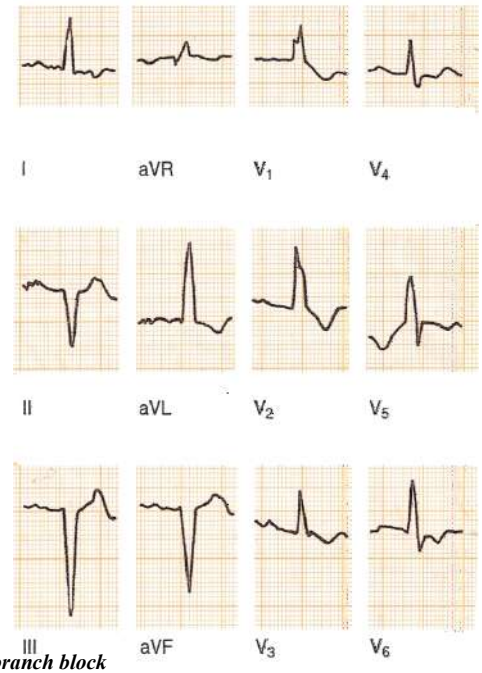



***(Fig. 5-2B) right bundle branch block (RBBB)***
***Key point :-***

- ***R in V1***
- ***S in V6***

**EEG criteria for incomplete RBBB is the same pattern as complete RBBB but with normal QRS complex duration.** **(Fig.5- 2C)**

RSR pattern in V1 and QRS duration <




*(Fig. 5-2C) incomplete right bundle branch block*

***Key point :-***

- ***Morphology of the RBBB.***
- ***Normal QRS duration < 0.12 sec.***

**Practical point**

M-shaped QRS complex over right precordial leads may help you to remember the QRS complex of RBBB

## *B- Left bundle branch block (LBBB)*

*The sequence of ventricle activation is almost opposite of that occur in RBBB.*

- *The septum is activated from the right to left produce small q in V1 and R in V6 ( there is no septal q wave V5-V6 ) if it present may indicate ischemia (Fig. 5-3A).*


*Fig. 5-3A*

- *Activation of thin – walled Right Ventricle from left to right produces little current .(Fig.5-3B)*


*Fig. 5-3B*

- *The LV is depolarized late by current working with terminal QRS forces are oriented toward left produces : broad positive complex- often notched in left side leads*

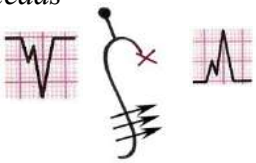


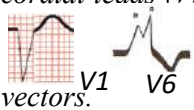
*Fig. 5-3C*

**Practical point**
LBBB Recognition: M-shaped QRS complex over leads Oriented to the left ventricle (V5-V6-1-avL)

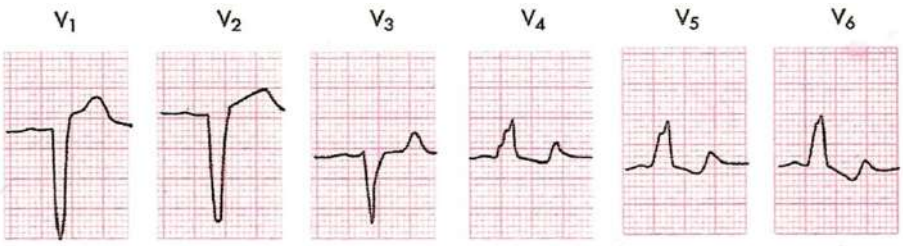
**Note**: step 1 and step 2 may not appear in surface ECG and by step 3 that represent the left ventricle give wide deep S wave in lead 1 and wide R wave in lead V6. (Fig.5-3C)

**ECG criteria of LBBB (Fig. 5-3D)**

- *QRS duration more than (> 0.12 sec)*
- *Broad slurred or notched R wave in Lateral precordial leads (V5-V6-1-avl)*

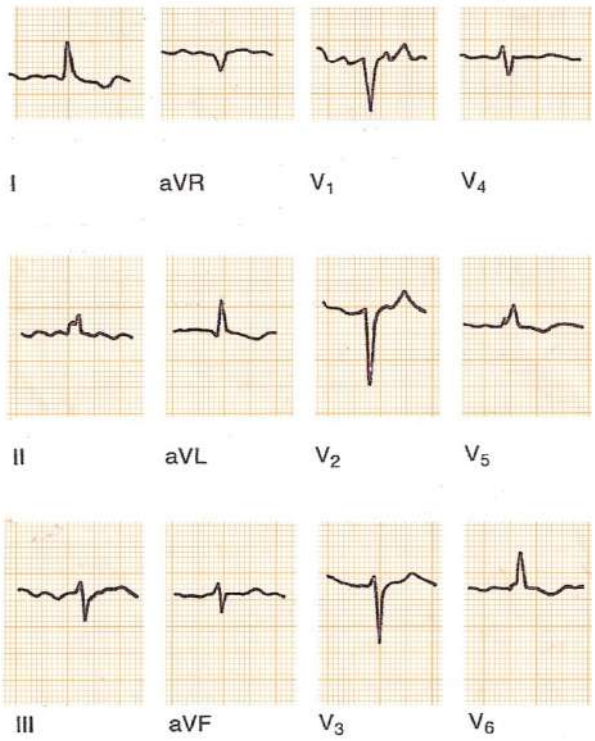



- *ST-T wave vectors opposite to the terminal QRS vectors.*
  - *i.e. ST-depression and T inversion in left precordial leads.*
  - *But in right precordial leads ST-T changes opposite the complex, i.e. upward.*
- *QS or rS pattern in the anterior precordial leads (V1-V2 )*




*(Fig. 5-3D) Left bundle branch block*
*Essential Features:-*
*Broad QRS complexes.*
*QS in V1, V2, V3*
*Notched R wave in V5, V6*

## Incomplete LBBB the same pattern of complete LBBB with normal QRS duration ( Fig.5-3E)

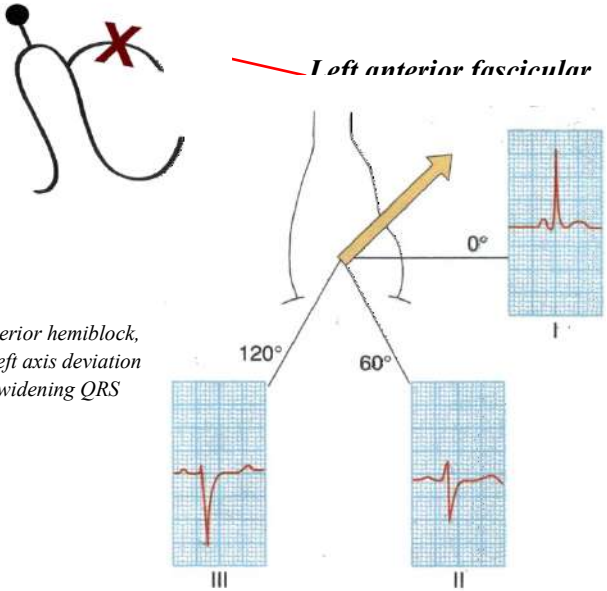



*(Fig. 5-3E) Incomplete left bundle branch block.*
*Essential points:-*

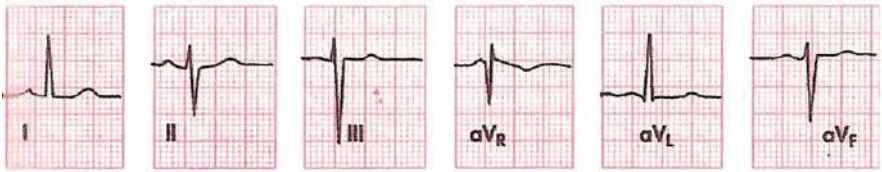
- *LBBB morphology.*
- *Normal QRS duration.*

# Fascicular hemiblock

## *A-anterior ( anterio-superior ) hemi block (Fig.5-4A&B)*




*(Fig. 5-4A) Left anterior hemiblock, notice the marked left axis deviation without significant widening QRS complex.*



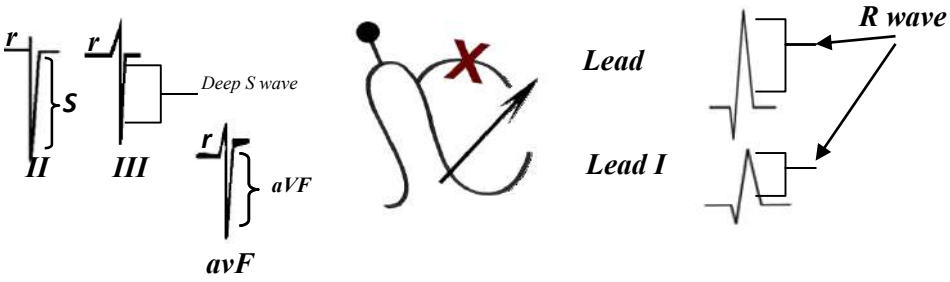

*(Fig. 5-4B) Left axis deviation as a result of left anterior hemiblock without significant widening of the QRS duration.*

**Anatomical considerations**

- ***The anterio-superior or left anterior division of LBB is more vulnerable to interruption than posterior (posterio-inferior division ) due to the following reasons:***
    - *The anterio-superior division is long and thin, whereas the posterior-inferior is relatively short and thick.*
    - *the posterior ( posterio-inferior division ) has double blood supply , in contrast to anterior (anterio-superior ) which has a single blood supply by sepal branch of anterior descending artery , which also supply the right bundle branch this is one of the reasons for frequents manifestation of RBBB with left anterior hemi block .*
    - *The anterior (anterio-superior) division is closer to the aortic valve, and is therefore more likely to be involved in disease affecting the aortic valve.*

## Mechanism of left anterior hemi

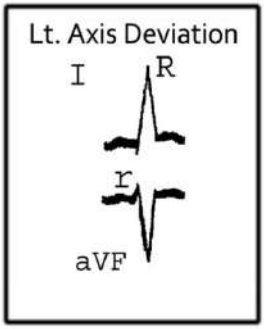
*Electrical vector directed for left i.e. marked left axis deviation (Fig. 5-5).*



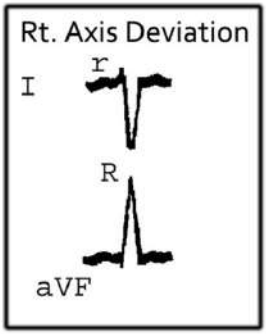

**ECG criteria of left anterior hemi block.**

1- *Marked left axis deviation LAD (more negative - 45 degree) with normal QRS morphology and duration.*

2- *the marked LAD can easily recognized because deep S wave in aVF , and tall R wave in lead I.* *(Figs.5-4B& 5-6A)*

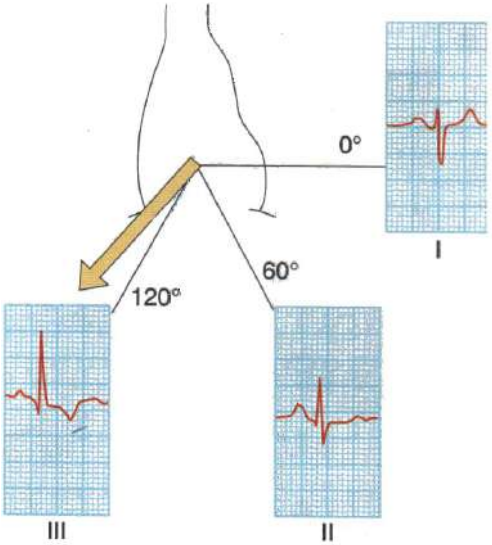



(Fig. 5-6A)




(Fig. 5-6B)

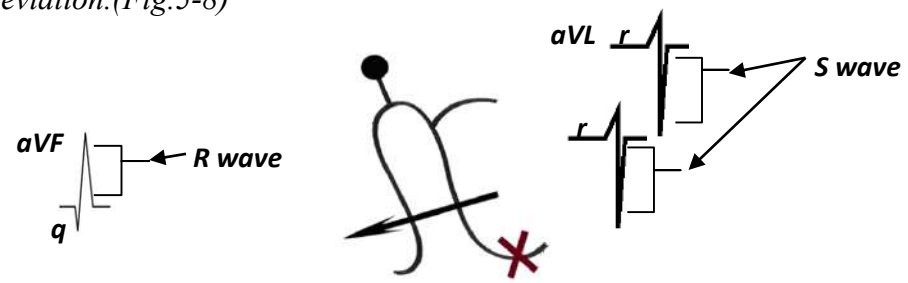
### B- *Left posterior (posterior inferior) fascicular block.* *(Fig.5-7)*




*(Fig. 5-7) posterior hemiblock, marked right axis deviation, normal QRS duration.*

## *Mechanism of left posterior (posterior inferior) block*

*Electrical vector directed to the right, with marked right axis deviation.(Fig.5-8)*




*(Fig. 5-8)*

**ECG criteria (opposite left anterior hemi block)**

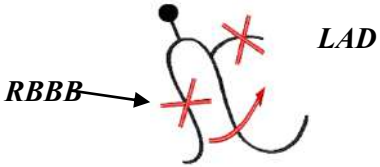
*1- Marked right axis deviation (RAD) of 120◦ or more with normal*

## C- Bifascicular block. *(Fig. 5-9)*

***Is the right bundle branch block with one of the following:-***

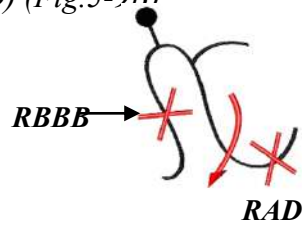
*A- Left anterior hemi block (LAH) produces a RBBB pattern with marked LAD*

*(Figs. 5-9A&C).*

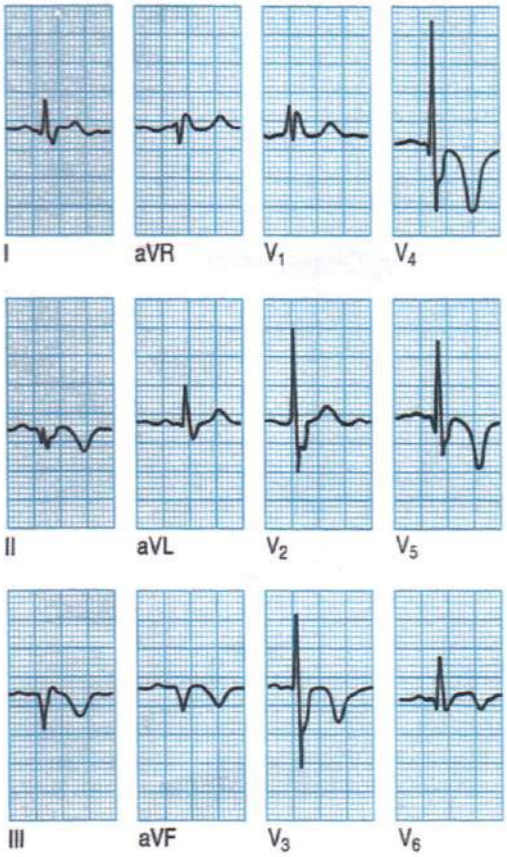



*(Fig.5-9A)*

*B- left posterior hemi block (LPH) produces a RBBB pattern with marked right axis deviation .(RAD) (Fig.5-9B)*




*(Fig.5-9B)*



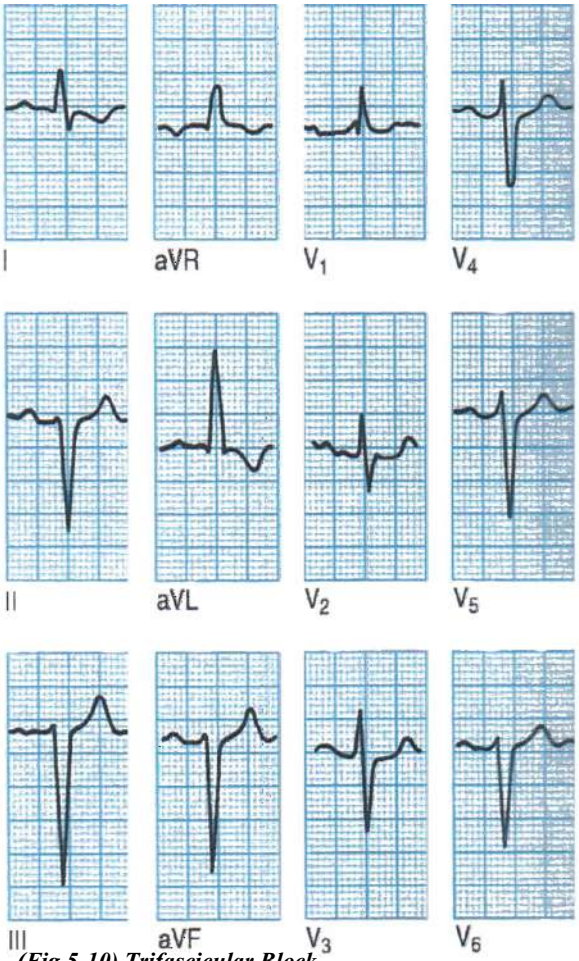

*(Fig. 5-9C) Bifascicular Block*

*Essential feature:-*

- *Left axis deviation (axis is 60◦)*
- *Right Bundle Branch Block*

## D- *Tri fascicular block.* *(Fig.5-10)*

*Is A bifascicular block with prolonged PR interval.*




***(Fig.5-10) Trifascicular Block***

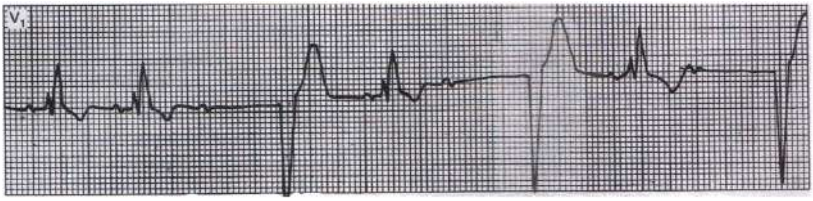
***Essential features:-***

- ***Left axis deviation (axis is -80◦)***
- ***Right Bundle Branch Block***
- ***First degree AV Block (Pr interval is 0.24s)***

## *Alternating and intermittent bundle branch block*

1- *Conduction abnormality involving both the right and left bundle branches.*

2- *Indicated by presence of some alternating beats , with RBBB and other with LBBB or by AV block located distal to the common bundle (Fig.5-11)*

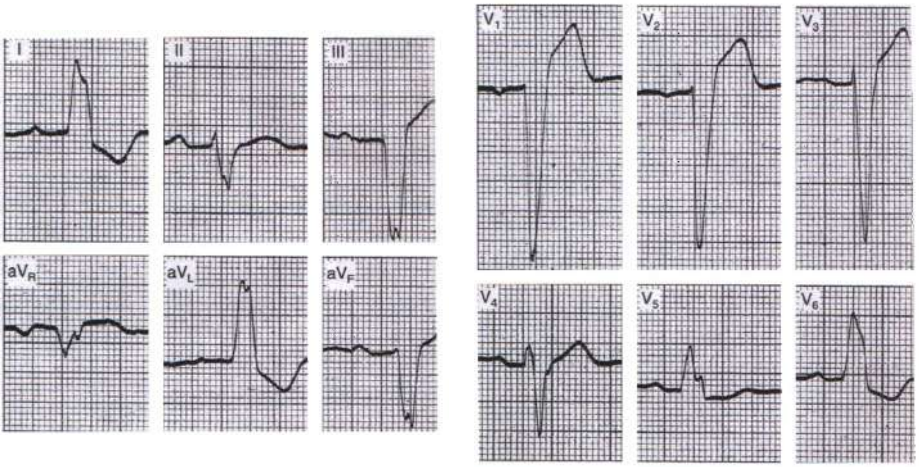



***(Fig.5-11)*** **Alternating LBBB and RBBB. In the first two beats there is RBBB. The left bundle is able to conduct as long as the rate is slow enough. When the rate speeds up, the left bundle blocks, unmasking the slow conduction through the right bundle branch (note the long PR interval with the LBBB beats). The next P wave (hidden) blocks in both bundles, and the sequence begins again: (1) a short PR interval and conduction over the left bundle (RBBB), (2) block in the left bundle and slow conduction over the right bundle (LBBB with a long PR interval), and (3) block in both bundles (hidden P wave).**

## *Practical Comments to Remember*

- *Left anterior or posterior hemi block, the diagnosis is made from the QRS axis, without changes in QRS morphology or duration.*
- *LBBB and RBBB the diagnosis is made from the QRS pattern (morphology and duration) without change of the mean QRS axis.*
- *The diagnosis of ventricular hypertrophy in the presence of bundle branch blocks is difficult in clinical practice. A few general guidelines are helpful*
    - *When RVH occurs with RBBB, RAD is often present; a tall peaked P wave with RBBB should also suggest underlying RVH.*
    - *The finding of LBBB or RBBB and evidence of left a trial abnormality virtually ensures the diagnosis of LVH.*
- *The ECG diagnosis of MI in the presence of bundle branch block is discussed in (chapter 6) ischemic heart disease.*
- *In case of LBBB, the left precordial leads – leads V5 and V6 , as well as standard lead cannot reflect an internal septal q wave or a terminal S wave.*
- *Complete LBBB, indicates organic heart disease. It is commonly associated with ischemia and hypertensive heart disease.*
- *The commonest cause of left axis deviation (LAD) is left anterior hemi block due to fibrosis that may results of (IHD, chronic HF, chronic hypertension and chronic cardiomyopathy).*
- *When RBBB or LBBB associated with axis deviation mainly left axis. ( Fig.5-12) is worse prognosis than when LBBB with normal directed QRS complex*
- *The combination of RBBB and LAD is associated with ostium primum ASD. In the rare Brugada* syndrome, RBBB with ST segment elevation in the right precordial ECG leads, lead to high incidence of sudden death secondary to ventricular tachyarrhythmias.*

**see chapter 6 (Fig.6-36)*



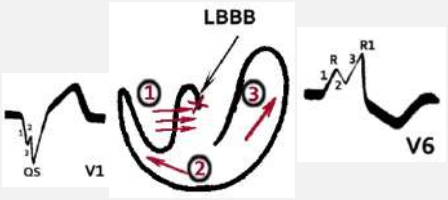
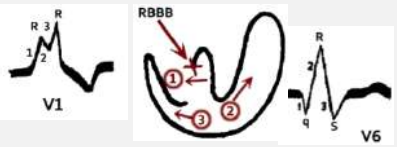

*(Fig.5-12)* **LBBB and left axis deviation.**

*The student and General practitioner must understand that Wide QRS complex is not restricted to BBB but has many differential diagnosis*

***Differential diagnosis of ECG patterns with a wide QRS complex includes four major classes of abnormities:-***

1. Right or Left Bundle Branch Block.
2. Toxic conduction delay caused by hyperkalemia or certain drugs.
3. Ventricular premature beats or pacemaker beats.
4. WPW syndrome.

## *Summary of pathophysiology and ECG changes of Bundle Branch Block*

| *Complete Left Bundle Branch Block (LBBB)* | *Complete Right Bundle Block (RBBB)* |
|---|---|
|  |  |
| ***1.*** *the septal activation is occurs from right to left (a reversal of the normal pattern) produces Q wave in V1 and R wave in V6 (arrow 1 above).*<br><br>***2.*** *Right ventricle depolarize normally produces r wave in V1 and S wave in V6 (arrow 2 above).*<br><br>***3.*** *Delay depolarization of the left ventricle due to LBBB, produces S wave in V1 and R wave in V6, and so the QRS complex is wider than (3 small boxes) with abnormal shape (arrow 3 above)* | *1. Septum depolarization occurs from left to right (same of the normal pattern) (arrow 1 above) produces R wave in V1, small septal q wave in V6.*<br><br>*2. Left ventricle depolarize normally produces S wave in V1, R wave in V6 (arrow 2 above).*<br><br>*3. Right ventricle depolarize late (by the left ventricle) produces R wave in V1, S wave in V6 (arrow 3 above). So the QRS is wider than 3 small boxes with abnormal morphology.* |

# Chapter 6

# Myocardial Ischemia and Infarction

## Ischemic Heart Disease (IHD)

- IHD remains the leading cause of morbidity and mortality in the world.
- Coronary artery disease (CAD) represent stable angina (angina pectoris), coronary artery syndrome, Heart failure (HF), silent ischemia, sudden death.
- Acute coronary syndrome represents a spectrum of clinical presentation of acute myocardial ischemia referred to as unstable angina (UA), Non ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI).
- From the clinical point of view, the division of acute MI into ST-segment elevation and Non ST-segment elevation types is useful since the efficacy of acute reperfusion therapy is limited to the former group.
- The ECG has a central role for diagnosis of IHD in patient present with chest pain, but before your comment on ECG changes there are many things to remember.
    - The ECG has important limitation in both sensitivity and specificity in the diagnosis of IHD.
    - A single normal ECG does not exclude ischemia or acute MI.
    - A prolonged chest pain without diagnostic changes in serial ECG should be always prompts a careful search for other non-coronary causes of chest pain.

- A chest pain with ECG changes not always caused by ischemic heart disease but there are many factors that mimic ECG changes of MI, that discussed in the appropriate section but the following are the most important factors:-

| Diagnosis | ECG findings mimicking MI | Diagnostic evaluation |
|---|---|---|
| Pericarditis | ST elevation | Echocardiography |
| Myocarditis | ST elevation, Q wave | echocardiography |
| Acute aortic dissection | ST elevation or depression, non specific ST-and-T-wave changes | from esophageal echocardiography, CT of the chest, aortography or MRI |
| Pneumothorax | New poor R-wave progression in leads V1-V6, acute QRS axis shift | Chest radiography |
| Pulmonary embolism | Inferior ST elevation, ST shift in lead V1-V3 | Ventilation perfusion scan |
| Acute cholecystitis | Inferior ST-elevation | Gall bladder ultrasound or radioisotope scan |

- The diagnostic changes of acute or evolving ischemia are often masked by the presence of the following conditions:-

1. Left bundle branch block LBBB.
2. Electronic ventricle pacemaker pattern.
3. WPW Preexcitation syndrome

- Generally the ECG changes in IHD described are those of

  A. Ischemia.  B. Injury.  C. Necrosis.

- Zone of Ischemia which cause ST segment depression with or without T wave inversion
- Zone of injury cause ST segment elevation with or without loss of R wave progression.
- Zone of Necrosis (infarction) cause a deep Q wave as a result of absence of depolarization

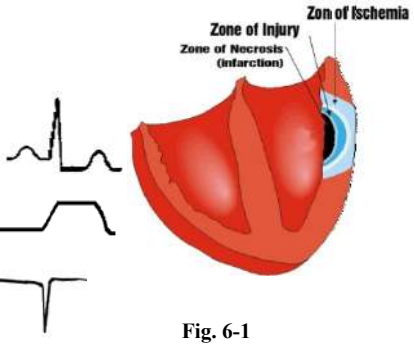
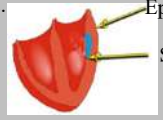
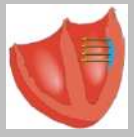
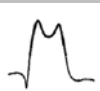
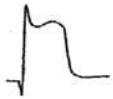



**Fig. 6-1**

# Ischemic Heart Disease, ECG Changes

| Diagnosis | ECG changes | Time of ECG changes | Pathophysiology |
|---|---|---|---|
| **Angina pectoris** | ST depression[1*] is the most common changes.<br><br>T wave inversion.<br><br>T wave pseudo-normalization[3*] | Coincidental with a chest pain or during exercise test[2*]<br><br>(see figs. 6-2&6-3) | - Transient, reversible subendocardial ischemia due to fixed stenotic artery, demand supply mismatch of O2.<br>    <br>Epicardium<br>Subendocardium<br>**ST depression**<br>- With acute subendocardial ischemia the electrical forces are deviated toward the inner layer of the heart causing ST depression or T inversion in leads which face the ischemic area see fig above. |
| **Coronary artery spasm (Prinzmetal angina)** | Reversible ST elevation | Coincidental with chest pain<br><br>(Fig.6-4) | - Spasm may occur in a normal artery or at the site of plaque; usually total occlusion, short duration (minutes); transmural ischemia temporary.<br><br>- With acute transmural ischemia electrical forces (arrows) are deviated towards the outer layer of<br><br>the heart causing ST elevation in the overlying lea<br>   <br>ST elevation |

Continue ⟶

| | | | |
|---|---|---|---|
| **Unstable angina[4*] and**<br><br>**Non ST segment elevation MI** | ST depression<br><br>T wave inversion | During chest pain may be permanent ECG changes<br><br>(Fig.6-5) | - Stenosed artery, but with some blood flow subendocardial ischemia, then necrosis.<br>- Same mechanism of angina pectoris but with progressive changes.<br>   |
| **ST segment elevation MI** | • **Tall upright T wave.**<br>• **Elevated segment.**  | Minutes afterwards<br><br>(Figs.6-10&6-11) | -Total occlusion artery, full thickness ischemia injury, necrosis.<br><br>-When transmural ischemia is persist causing more injury to cardiac muscle, the electrical forces are deviated towards the outer layer causing ST elevation in the overlying leads and reciprocal changes in opposite leads. |
| | • **Progressive loss of R wave.**<br>• **Terminal T inversion.**<br>• **Developing Q wave.**  | Hours afterwards | -With progressive ischemia, injury and necrosis, the electrical forces conduction decreased, resulting in decrease of R wave progression in overlying leads with small Q wave.  ST elevation |
| | • **ST segment return to normal.**<br>• **Deep Q wave.**  | Days afterwards | -Lastly when myocardial necrosis is established at the end of evolving phase, loss of large forces resulting in the mean forces now being oriented in an opposite direction, the electrode overlying the area of necrosis records a negative deflection(Q wave) |
| | • **T wave inversion.**<br>• **Persist deep Q wave.**<br>• **T wave may become less marked or may be**  | Weeks afterwards |   Q wave |

**permanent or return to normal.**

***Practical point:-***

**How to identify ST segment elevation/depression.**

ST segment of a given complex is compared with followed TP segment and preceeded PR segment of the same complex.

1*. The form of ST segment depression

A- Horizontal ST segment depression usually indicates subendocardial ischemia. (Fig.6-2B)

B- Upward-sloping ST depression:-

In this case the J point, the junction between QRS complex and the beginning of ST segment is depressed bellow the baseline, but the ST segment moves rapidly upward and its non-specific finding may occur in normal people.

C- Downward sloping ST segment depression usually indicates ischemia. (Fig.6-3)

2* - Exercise ECG test (Fig.6-3)

1. Is useful in establishing the diagnosis of CAD, because in many patient normal rest ECG does not exclude coronary artery disease (CAD).
2. Stress ECG is usually performed while the patient walks on treadmill or pedicle a bicycle.
3. The test is stopped when the patient develops anginal pain, fatigue or diagnostic ST-T changes or when heart rate reaches 85% to 90% of maximum predicted rate, predetermined according to the patient age, this approach is known as submaximal testing.
4. Normal exercise test does not exclude coronary artery disease.
5. ST depression of at least 1mm or more lasting at least 0.08s is positive (abnormal test), ST depression less than 1mm or upward slopping are considered as negative (normal) test response.

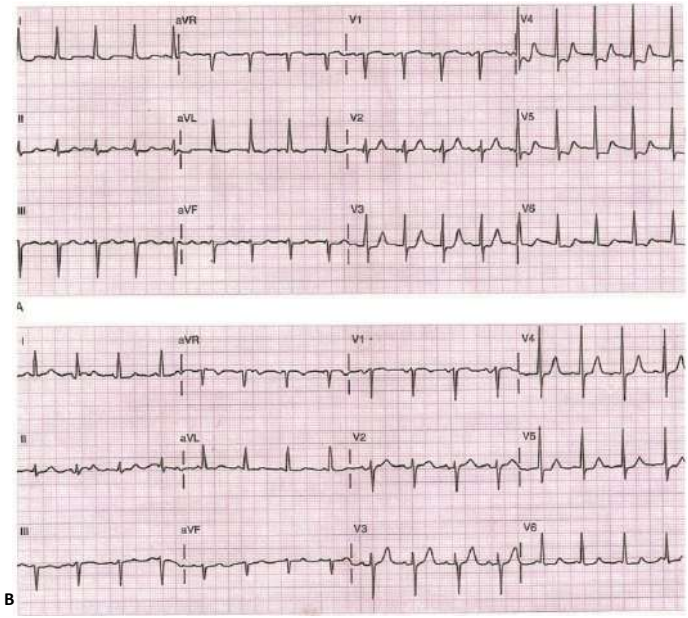



Fig. 6-2 . Electrocardiogram obtained during angina (A) and after administration of sublingual nitroglycerin and subsequent resolution of angina (B). During angina in the patient there is transient ST segment depression and T wave abnormalities.

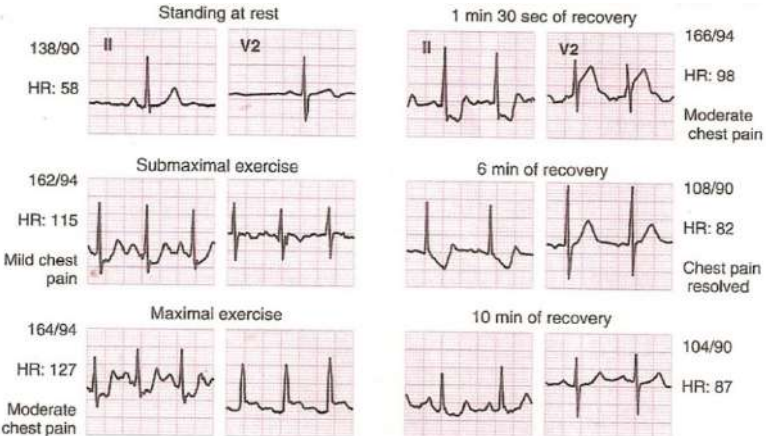



Fig. 6-3 .Treadmil exercise test demonstrating a markedly ischemic ECG response. The resting ECG was normal. The test was stopped on reproduction of angina at a relatively low workload, accompanied by ST segment depression in lead II and ST segment elevation in lead V2. These changes worsened early in recovery and resolved after administration of sublingual nitroglycerin. Only lead II and V2 are shown; however, ischemic changes were seen in 10 of 12recorded leads. Severe atherosclerotic disease of all coronary arteries was documented at subsequent cardiac catheterization.

[3*] pseudonormalization:- in patient with evolving MI T waves are inverted, during ischemic episode T waves become normally upright in these leads called pseudonormalization.

[4*]-Non-ST segment elevation myocardial infarction and unstable angina have The same ECG manifestation, the distinction between these two conditions is ultimately made on the basis of presence or absence of cardiac enzymes

***The diagnosis of myocardial infarction is established if at least two of the following three criteria are consistent with myocardial infarction :-***

1. Patient history.
2. ECG changes.
3. Changes in the cardiac markers*.

*Enzymatic changes are essential to differentiate the MI from other Causes of chest pain with ECG changes e.g. unstable angina even it may increase the cardiac enzymes but not to the same extent of MI.

# ECG localization of ischemia and myocardial infarction

***Practical point:-***

The most important thing in ischemia and MI is that ECG changes are limited to the area which affected by specific coronary artery with reciprocal changes* not as other myocardial disease as myocarditis for example it has diffuse non specific ECG changes or even pericarditis etc….

*Reciprocal changes: - means opposite changes, either as ischemic changes secondary to myocardial infarction in opposite lead (see localization of MI below) or as primary diagnostic of MI as posterior MI.
Reciprocal changes appear in the opposite leads as.

- Increase R wave amplitude.
- Depressed ST segment.
- Upright high T wave.

# Localization of MI

| Infarction location | Leads depicting primary changes | Reciprocal changes | Likely vessel involved |
|---|---|---|---|
| Inferior MI | II, III, aVF (Fig.6-15) | I, aVL,V1,V2 | RCA |
| Septal MI | V1,V2 | II,III, aVF | LAD |
| Anterior MI | V3, V4 | II, III, aVF | LAD |
| Anteroseptal MI | V1-V4 (Fig.6-10) | II, III, aVF | LAD |
| Extensive anterior MI | LI, aVL, V1-V6 (Fig.6-11) | II, III, aVF | LAD |
| Lateral MI | I, aVL, V5-V6 (Fig.6-17) | II, III, aVF | CIRC |
| High lateral MI | I, aVL | II, III, aVF | CIRC |
| Posterior* MI | Esophageal leads | V1, V2 | RCA, CIRC |
| Right ventricular** infarction | V1, V2R, V3R, V4R, ST-elevation (For ECG changes see RVI) | | RCA |

- CIRC= circumflex artery
- LAD= left anterior descending artery
- RCA= right coronary artery

*usually associated with inferior or lateral MI
**usually associated with inferior MI.

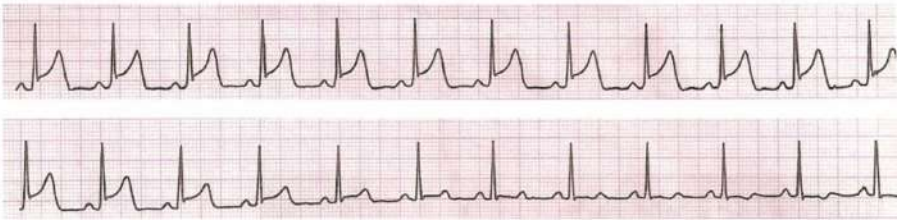


**Fig. 6-4**. Continuous ECG recording depression in a patient with prinzmetal's (variant) angina Spontaneous onset of chest discomfort began during the top strip, accompanied by transient ST segment elevation. By the bottom strip (several minutes later), both discomfort and ST elevation have resolved.

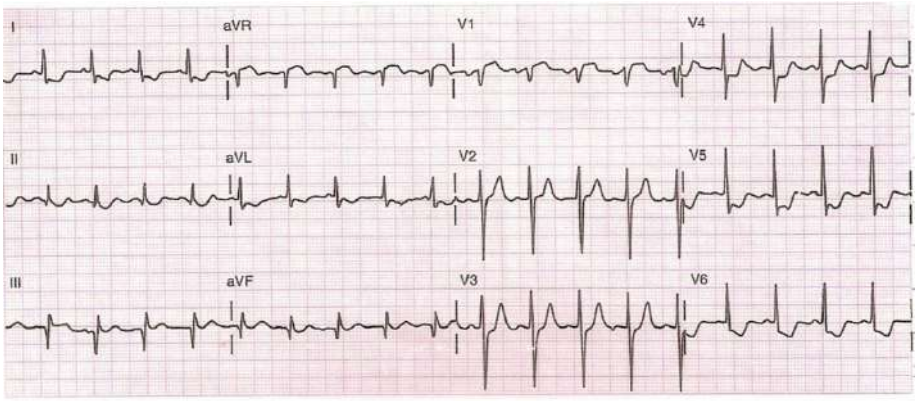
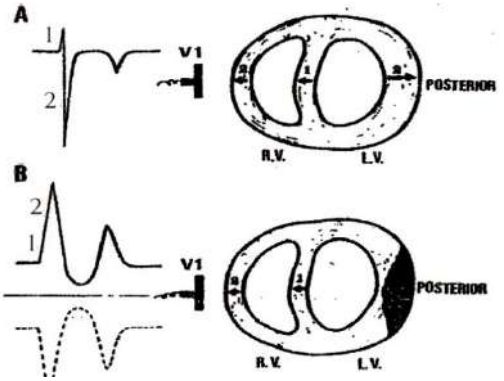



**Fig. 6-5**. Marked ST segment depression in a patient with prolonged chest pain resulting from an acute non-ST elevation myocardial infarction One to 3 mm of ST segment depression is seen in leads V4 to V6 and in leads I and aVL. The patient was known to have had a prior Q wave inferior myocardial infarction.

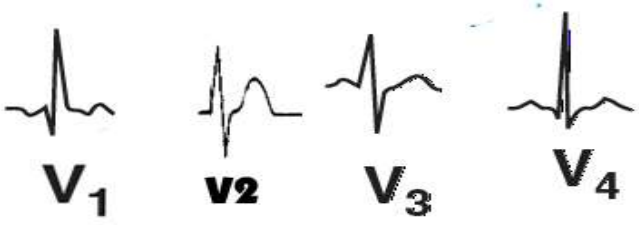
## Posterior myocardial infarction

Acute posterior MI has the following ECG changes. (Figs.6-6 , 6-7 & 6-8)

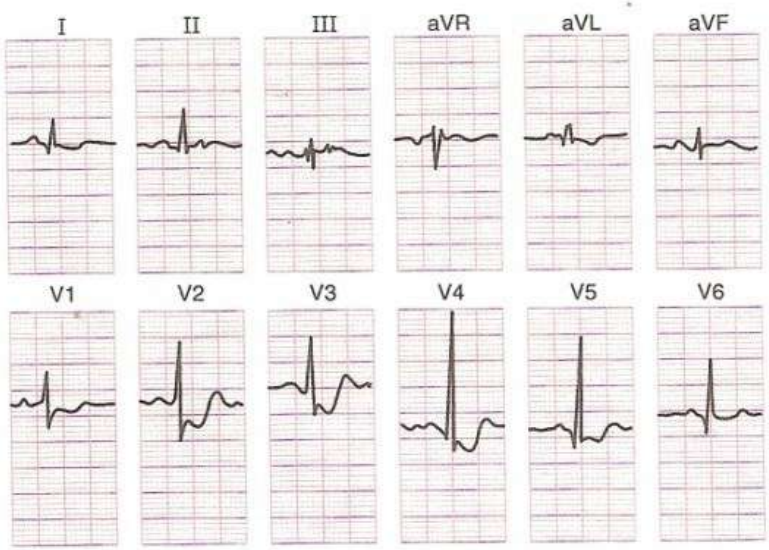
1. Dominant R wave in V1-V3.
2. ST segment depression in V1-V3.
3. Upright, tall T waves in the V1-V3.
4. In the most cases of posterior MI, the infarction extend either to the lateral wall producing characteristic changes in the lead V6 or to the inferior wall producing characteristic changes in the leads II, III, aVF.
5. Because of overlap between inferior and posterior MI the more general term inferio-posterior MI can be used when the ECG shows changes consistent with either inferior or posterior infarction.(Fig.6-9)



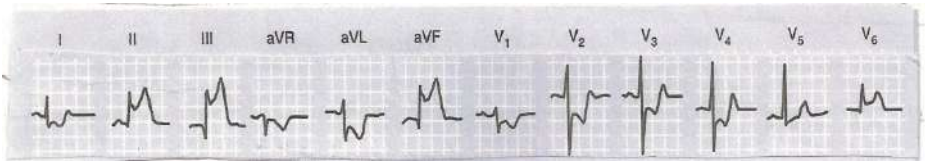

(Fig.6-6) Diagram illustrating (A) normal ventricular depolarization.
(B) Ventricular depolarization during true posterior wall infarction in ECG mainly V1 is present as a mirror image of the QRS complex.




(Fig.6-7)

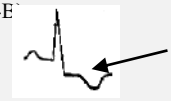
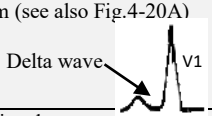
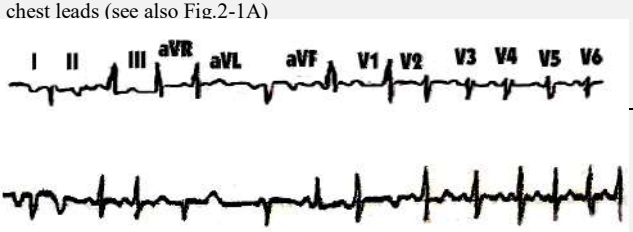



**(Fig.6-8) posterior myocardial infarction. Tall R waves are present in leads V1 and V2 accompanied by deep ST depression. These are reciprocal changes arising from a posterior**




**(Fig.6-9) posterinferior myocardial infarctions, ECG recorded early Q waves, marked ST elevation, hyperacute T waves in leads II, III, aVF, in addition a large R wave, ST segment depression and elevated T waves in V1-V2.**

# Differential diagnosis of tall R wave in V1

| N | Causes | Confirmatory clues |
|---|---|---|
| 1 | Normal variants | No other abnormalities |
| 2 | Posterior MI | ST depression, tall T wave (V1-V2) (See also Fig.6-8)  |
| 3 | Right ventricular hypertrophy | Secondary ST-T changes strain pattern, right axis deviation, right atrial enlargement (See also Fig.3-4E   |
| 4 | Ventricular septal hypertrophy | Associated Q wave, sign of left ventricular hypertrophy (See pseudo infarction in the glossary) |
| 5 | Right bundle branch block | Wide QRS complex if complete, broad S in V6, R peaked late in V1 (See also Fig.5-2B)  |
| 6 | WPW-syndrome type A | Short PR interval – delta waves Diagram (see also Fig.4-20A)   |
| 7 | Dextrocardia* | Inverted P wave in lead I, with progressive decrease of R wave in the chest leads (see also Fig.2-1A)   |
| 8 | Incorrect position of the leads** | |
| | | |

** The same ECG manifestation will occur with Dextrocardia and incorrectly placed (reversed) limp leads. In the former the precordial leads will reflect a reversed QRS pattern, the QRS complex begin tallest in the lead V1 and diminished progressively in lead V6. While in the latter the precordial pattern will be normal.

*&** are discussed in detail in section of miscellaneous cardiac change.

## Anteroseptal myocardial infarction. (Fig.6-10)

Probably the most common mistake made in every day ECG interpretation is in diagnosing Anteroseptal infarction because the tracing lacks R waves in lead V1 and V2. Although this is always a possibility, QS pattern in those two leads is more likely Owing to LVH with or without incomplete LBBB, than to an anterior infarction other rare cause is cardiac amvloidosis.

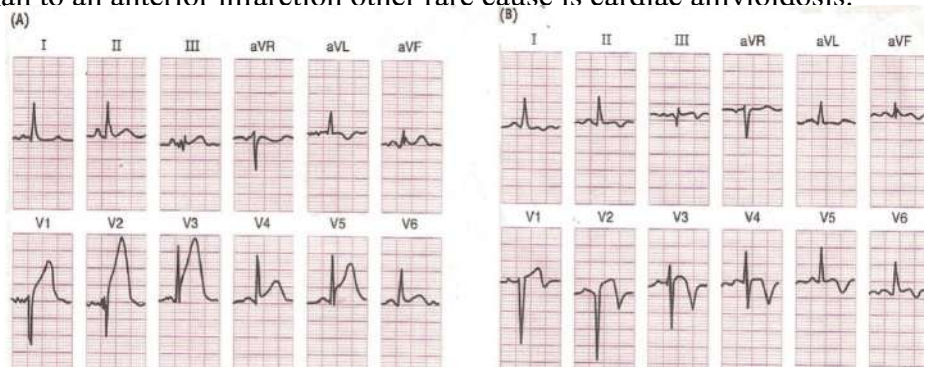



Fig.6-10. Anteroseptal myocardial infarction, (A) 12-lead ECG recorded within one hour of onset of symptom. Hyperacute changes are evident with marked ST elevation across the anterior chest lead. Tall peaked T waves are present in lead V2 and V3. (B) 12h later the acute ST changes have largely resolved, and have been succeeded by T wave inversion. A deep Q wave is present in lead V2.

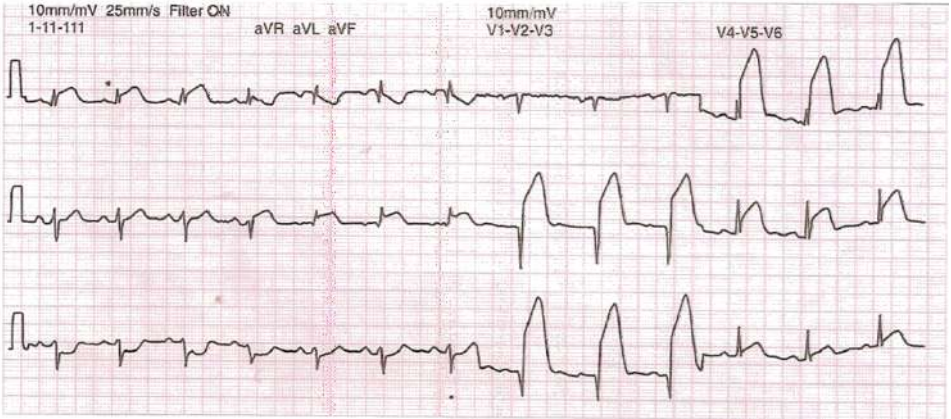
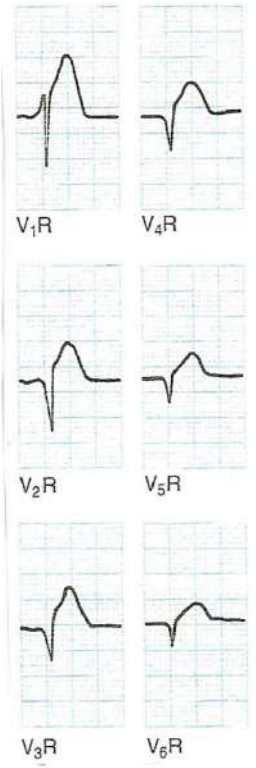
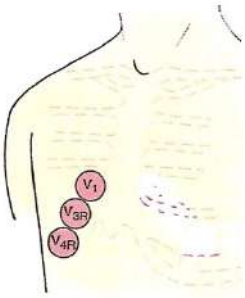



Fig.6-11. Acute anterolatral myocardial infarction. There is ST segment elevation (current of injury) across the precordial leads (V2 to V6) and in leads I and aVL reciprocal ST segment depression is seen in the inferior leads (leads II, III, and aVF). Deep Q waves have developed in leads V2 and V3.

# Right ventricle infarction (RVI)

A- To make the diagnosis of RVI, you must perform another ECG using right sided ECG leads, look for ST segment elevation in lead aV4R or any one of extra right precordial leads V4R to V6R.(Figs.6-12, 6-13)

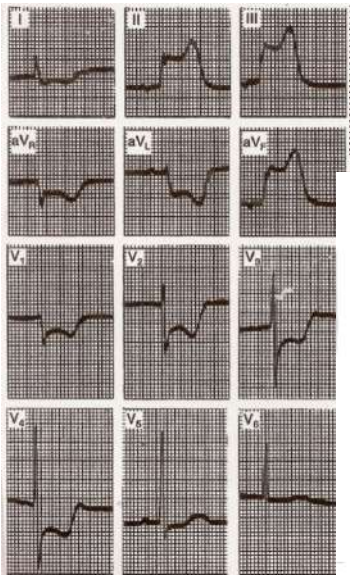


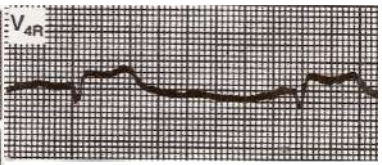

**(Fig 6-12) Positioning of right sided chest leads**

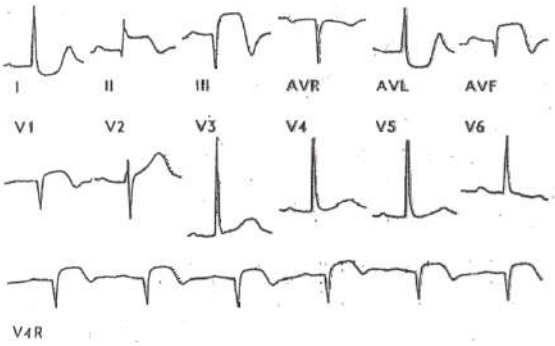
**(Fig 6-13) Right ventricular involvement**
**Key points:**
- **Only right sided chest leads are shown.**
- **ST segment elevation in all leads (including V4R)**

B- Right ventricular infarction suspected if in the clinical setting of acute inferior wall MI, there is ST segment elevation of 1mm or more in lead V1 (normally in case of inferior MI there is reciprocal ST segment depression in right precordial leads i.e. V1-V2 leads). (Figs.6-14 & 6-15)




(Fig.6-15 ) inferior myocardial infarction, ST elevation in leads II, III, aVF with reciprocal ST depression in leads V1, V2.




(Fig. 6-14) inferior MI, Q waves, ST elevation and T wave inversion in lead II, III, aVF and Q wave ST elevation in V1.

**In the above both patient in (fig.6-14)and (fig.6-15)the right ventricular lead V4 R was positive for right ventricular infarction.**

C- Occasionally RVI may be associated with ST elevation in leads V1 as well as other of the conventional precordial leads (V2 to V5) it may thus mimic anterior MI, however the magnitude of ST elevation in leads V1 to V5 with right ventricular infarction decreases from right to left chest leads i.e. ST elevation being maximum in the V1 of course concomitant evidence of inferior MI is further corroborative evidence that precordial ST elevation in leads V1 to V5 are due to right ventricular, rather left ventricular infarction.

D- In some situations there is ST elevation in V1, with ST depression in V2 a discordant relationship it also suggests the presence of right ventricular infarction.

***Practical guide lines***

***Practical point***

- ✓ An elevation of ST segment in aVR offers the highest specificity and efficacy in diagnosis of RVI.
- ✓ A diagnosis of RVI should only be considered if there is concomitant ECG evidence of inferior or inferoposterior MI

## Anterior MI infarction

In the clinical practice the anterior MI are subdivided into a number of subsets depending on the leads showing disturbance changes.

1. Anterior MI (V3-V4).
2. Anteroseptal MI (V1-V2).
3. Anterolateral or anteroapical MI (V1-V6).
4. High lateral (LI- aVL).

ECG changes of anterior MI. (Fig.6-11)

- QS complex in the anterior lead.
- Absence of the R waves in leads V1-V2.
- ST elevation in V1-V2.

## Atrial infarction

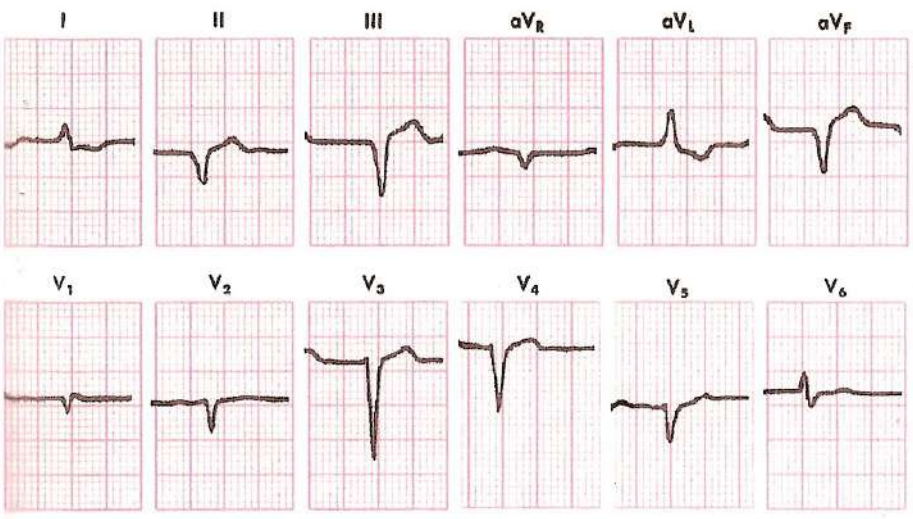
ECG recognition :-
One of the following is present:-

A- PR-segment elevation is greater than 0.5mm in leads V5 with PR segment depression in leads V1 and V2.
B- Pr-segment elevation is greater than 0.5mm in the lead I with PR-segment depression in leads II and III.
C- PR-segment depression is greater than 1.5mm in precordial leads and greater than 1.2mm in lead I, II, III, combined with atrial arrhythmia.
D- P waves may also be abnormal in shape (W-shaped), M shaped, notched, or irregular in configuration.

# The age of infarction

1. *Recent (acute) infarction* is diagnosed on ECG by
   A. ST segment elevation with or without pathological Q wave and T wave inversion.
   B. ST depression with or without pathological Q wave.
   C. Hyperacute T wave.
   D. Poor progression of R wave.
   E. Some times may appear as LBBB with masking infarction sign.

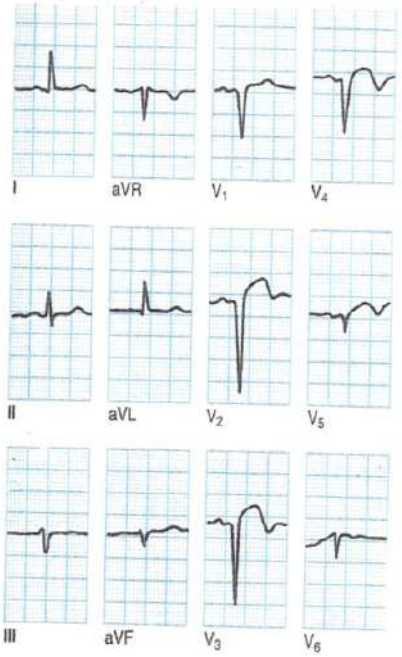
2. *Old infarction* is diagnosed by the presence of pathological Q wave without ST elevation of recent MI.
3. *Multiple infarction* acute or recent infarctions may develop in patient with previous myocardial infarction. (See the ECG below)



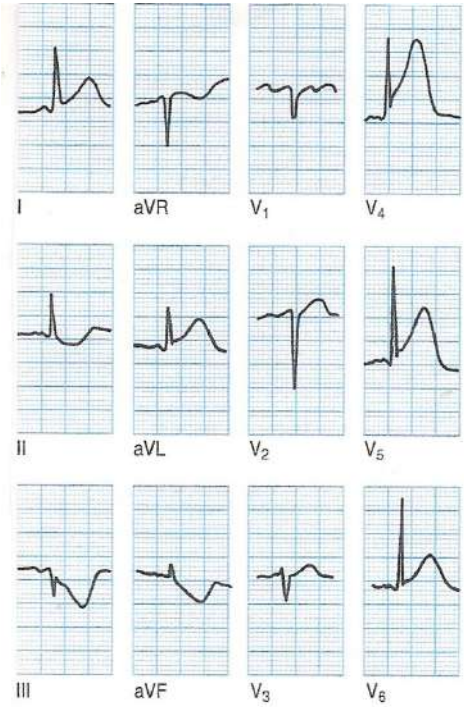

ECG of multiple myocardial infarctions. This ECG shows evidence of previous anterior and inferior walls infarction. Notice the slow R wave progression and QS complexes in the chest leads V1-V6 as well as the QS waves in leads II, III, aVF.

## *Ventricular aneurysm* (Fig.6-16)

in case of presence of pathological Q wave and persistent elevation of ST segment more than many weeks it's may indicated ventricular aneurysm in the corresponding leads of MI in this condition you need to look at previous ECGs.




**(Fig.6-16) ventricular aneurysm 6 months following an anterior MI persistent ST elevation in V1- V5**




**(Fig.6-17) Lateral myocardial infarction.**

**ST elevation in lead I, aVL and leads V5, V6 hyperacute T waves in leads V4, V5.**

# Diagnosis of Myocardial Infarction in the presence of Bundle Branch Block

## 1. Right bundle branch block RBBB

### A. RBBB without MI

Initially remember that RBBB effects primarily terminal phase of ventricular depolarization only as the following:-

*Step one* the septum is depolarized from the left produce R in V1 and Q in V6.(Fig.6-18)

Fig. 6-18


*Step two* the LV (left ventricle) is depolarized from the right produce S in V1 and R in V6. (Fig.6-19)

Fig. 6-19


*Step three* the right ventricle is depolarized slowly (because the right bundle branch is blocked) from left side produce wide R wave in V1 and S in V6. (Fig.6-20)


Fig. 6-20

## B. RBBB with MI

MI in case of RBBB affects both early and late depolarization with the following ECG changes:-

*Step one* septal activation does not take place because of necrotic and injured tissue.

*Step two* the left ventricle is depolarized from the right produce Q wave in V1 and R wave in V6. (Fig.6-21)

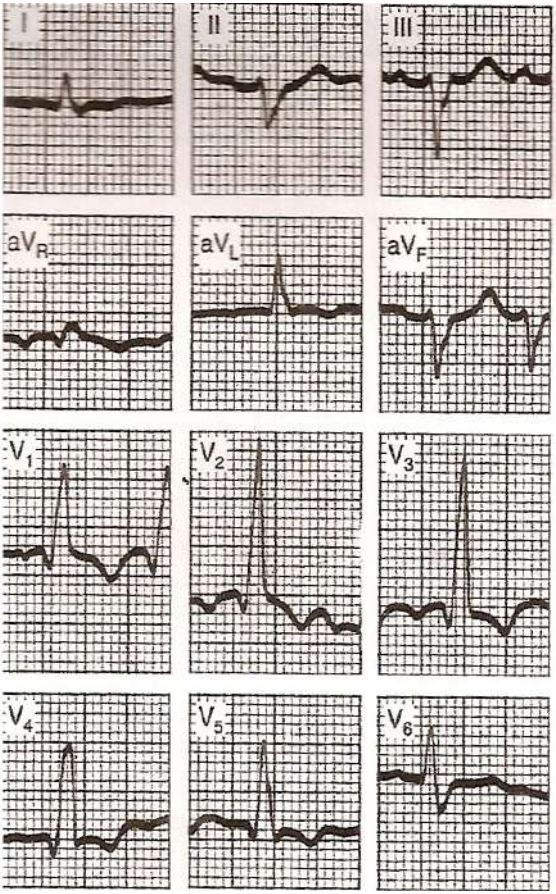

Fig. 6-21

*Step three* the right ventricle is depolarized from left produce R in V1 and S in V6. (Fig.6-22)


Fig. 6-22

### *Practical point*

1. Generally, the diagnosis of MI and RBBB is possible.
2. Abnormal Q wave in right precordial leads may indicate infarction.
3. ST elevation with Q wave in the chest leads with anterior MI and in lead II, III, aVF with an inferior MI. (Fig.6-23)
4. ST depression with tall T wave in V1-V2 with a posterior MI.




**(Fig.6-23) RBBB and left anterior hemiblock in a patient with acute Anteroseptal myocardial infarction.** **(look the Q waves in the Anteroseptal leads)**

## 2. Left Bundle Branch Block (LBBB)

### A- LBBB without MI

1. The septum depolarized from the right to the left produce Q in V1 and R in v6 (i.e. loss of normal R in V1 and septal Q in V6). (Fig.6-24)


**Fig. 6-24**

2. The total time for left ventricular depolarization is prolonged with LBBB the QRS is abnormally wide in V1 wide negative QS complex, and wide notched R wave without an initial Q wave in V6. (Fig.6-25)


**Fig. 6-25**

N.B. in some cases of LBBB the depolarization of right ventricle may appear as R wave in V1 (with wide S wave)but without opposite septal Q wave. (Fig.6-26)


**Fig. 6-26**

## B- LBBB with MI Example (anteroseptal MI)

1. The septal activation does not take place because of necrotic and injured tissue, initially the right ventricle depolarized produce tall, thin R in V1 and Q wave in V6 (similar to the normal activation). (Figs.6-27A&B)


**Fig. 6-27A**

2. The left ventricle (LV) is activated next producing a deep wide S in V1 and wide R in V6.

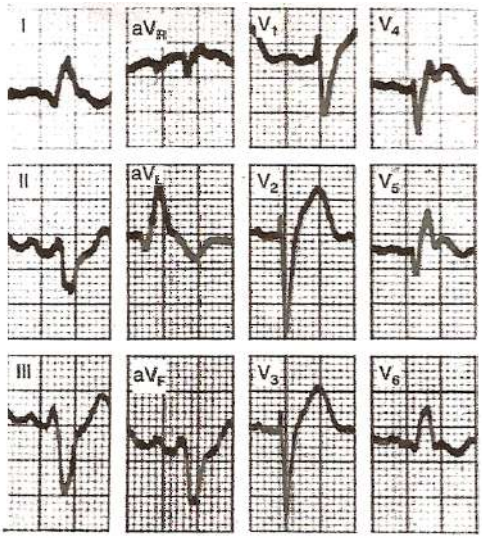



**Fig. 6-27B** LBBB and anteroseptal MI. The Q wave in lead $V_5$ and $V_6$ and the R wave in lead $V_1$ are evident. (From Lyon LJ: *Basic electrocardiography handbook*, New York, 1977, Van Nostrand Reinhold.)

***Practical point***

As general rule, if a patient with ischemic chest pain including those patient with cardiovascular risk factors (as hypertension etc) and ECG finding of LBBB is myocardial ischemia until prove otherwise and the cardiac enzymes should obtained to confirm the diagnosis, because the diagnosis of MI in the presence of LBBB generally is masked or may the LBBB suggests false diagnosis of MI, then the clinical judgment is important.

*Three strategies that may help in the correct ECG interpretation of LBBB with MI are as follows.*

1. *Serial ECGs demonstrating ischemic changes.*
2. *Comparison to previous ECG.*
3. *Knowledge of the expected ST segment and T wave morphologies in uncomplicated LBBB and thus the ability to recognize ischemia.*

The following ECG finding in LBBB were thought to correlate with myocardial ischemia. (Fig.6-27B, 6-28)

1. Presence of Q waves > one small box in the left chest leads with LBBB is suggestive of underling MI (V5-V6), I, aVL.
2. Presence of T wave inversion in the right chest leads (V1-V2).
3. ST segment elevation in the left chest leads (V5-V6), I, aVL.
4. ST segment depression in the right chest leads (V1-V2).
5. Rapid serial ST segment and T wave changes.



(Fig.6-28) LBBB with ischemia

Note. Loss of R wave progression in precordial leads or ST elevation in those leads not enough for the diagnosis of the MI with LBBB

## Differential Diagnosis of ST Segment Depressions

1. myocardial ischemia or infarction
   - A- Acute subendocardial infarction or non Q wave myocardial infarction.
   - B- Reciprocal changes with acute transmural infarction (ischemia).
2. Abnormal non coronary pattern.
   - A- Left or right ventricular hypertrophy (strain) pattern.
   - B- Secondary ST-T changes.
     - Left bundle branch block (LBBB).
     - Right bundle branch block (RBBB).
     - WPW precipitation pattern.
   - C- Drugs (e.g. digitalis).
   - D- Metabolic conditions (e.g. hypokalaemia)
   - E- Miscellaneous condition (e.g. cardiomyopathy)
3. physiological and normal variant*.

*with physiological and normal variants the ST-segment depression are usually less than 1mm and are seen especially with exertion or hyperventilation

**Differential Diagnosis of QS complexes in the Anteroseptal leads.**

1. Anteroseptal MI, usually accompany ischemic changes in other leads.
2. Chronic left ventricular hypertrophy.
3. Left bundle branch block.
4. System hypothermia.
5. Brugada syndrome.

## Differential Diagnosis of ST segment Elevation

1. Ischemia / myocardial infarction
   non infarction transmural ischemia (Prinzmetal's angina pattern)
   acute myocardial infarction
   postmyocardial infarction (ventricular aneurysm pattern)
2. Acute pericarditis.
3. Normal variant (early repolarization pattern).
4. Left ventricular hypertrophy/left bundle branch block.
5. Other (rare)
   - Brugada* syndrome (right bundle branch block-like pattern with ST elevation in right precordial leads).
   - Hyperkalaemia. (usually limited to V1 & V2)
   - Hypothermia (J wave / Osborn wave).
   - Myocardial injury.
   - Myocarditis
   - Tumor invading left ventricle.
   - Trauma to ventricles. (e.g. DC shock, needles)

*Brugada syndrome:-
In 1992 Brugada and Brugada (Brugada Brothers) described eight patients without structural heart disease, including three children, who had a history of aborted sudden cardiac death resulting from ventricular fibrillation (VF), right precordial leads (RBBB), and unique type of ST elevation in the right precordial leads (V1 to V3). Recognition of this ECG pattern is important because it permits early identification and treatment of those at risk of sudden death. (FIG.6-30)

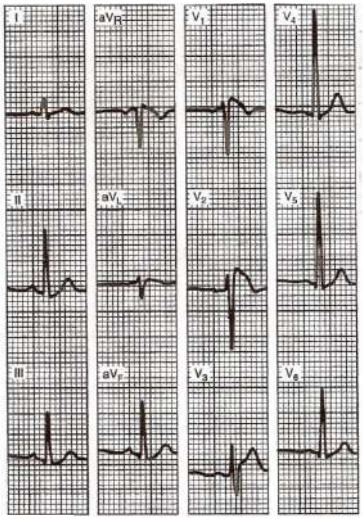



Fig. 6-30

Brugada syndrome in a 39-year-old patient. Note the typical coved ST segment elevation in $V_1$ and $V_2$. This patient previously experienced two unexplained episodes of syncope. (From Hermida JS, Lemoine JL, Aoun FB et al: Prevalence of the Brugada syndrome in an apparently healthy population, *Am J Cardiol* 86:91-94, 2000.)

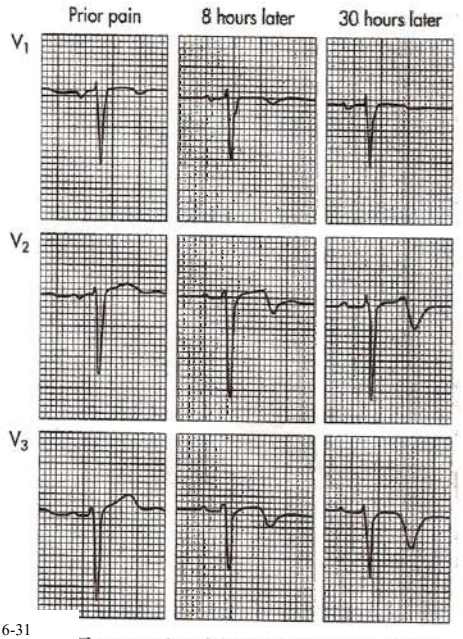



Fig. 6-31

The progression of Wellens syndrome from admission in the emergency department to 30 hours later, before angiography. Note the dramatic changes in the T waves of $V_2$ and $V_3$ compared to the insignificant changes in $V_1$.

# Differential Diagnosis of T wave changes

## *T wave inversion*

1. Primary T wave inversion.
   - A- Normal variants (e.g. Juvenile T wave pattern).
   - B- Myocardial ischemia or infarction.
   - C- Wellens syndrome.*
   - D- Left and right ventricular overload (Strain Pattern).
   - E- Cerebrovascular accident (CVA).
   - F- Post pacemaker T wave pattern.
   - G- Post tachycardia T wave pattern.
   - H- Miscellaneous conditions.
     - Cardiomyopathy.
     - Intermittent left bundle branch block.
     - Myocardial tumor.
2. Secondary T wave inversions.
   - A- Left bundle branch block.
   - B- Right bundle branch block.
   - C- WPW-preexcitation pattern.
   - D- Ventricular paced beats.
3. Idiopathic global T wave inversion syndrome.

*Wellens syndrome

Consists of specific ST-T wave changes in V2-V3 during the pain free period in patients with unstable angina, indicating critical stenosis high in the left anterior descending (LAD) coronary artery.

ECG pattern of Wellens syndrome. (Fig.6-31)

- Progressive deep symmetrical T inversion.
- No loss of R wave progression.

Clinical significance of recognition of Wellens syndrome and confirmation by subsequent cardiac catheterization identity the need for bypass grafting or PTCA.

## *Tall Positive T waves*

1. Ischemic causes
   A- Hyperacute phase of myocardial infarction.
   B- Acute transient transmural ischemia (Prinzmetal's angina).
   C- Chronic (evolving) phase of myocardial infarction (tall positive T waves reciprocal to primary T wave inversion).
   D- True posterior myocardial infarction.
2. Non ischemic causes
   A- Normal variants (early repolarization patterns).
   B- Hyperkalaemia.
   C- Cerebrovascular accident (most commonly T inversion).
   D- Left ventricular hypertrophy (LVH)
      - Left precordial leads usually in association with diastolic overload conditions (e.g. aortic or mitral regurgitations).
      - Right precordial leads usually in conjunction with left precordial ST depressions.
   E- Left bundle branch right precordial leads.
   F- Acute pericarditis (occasionally).

## *Non-specific T wave variants.*

Non specific T wave inversion may occur in the following conditions.

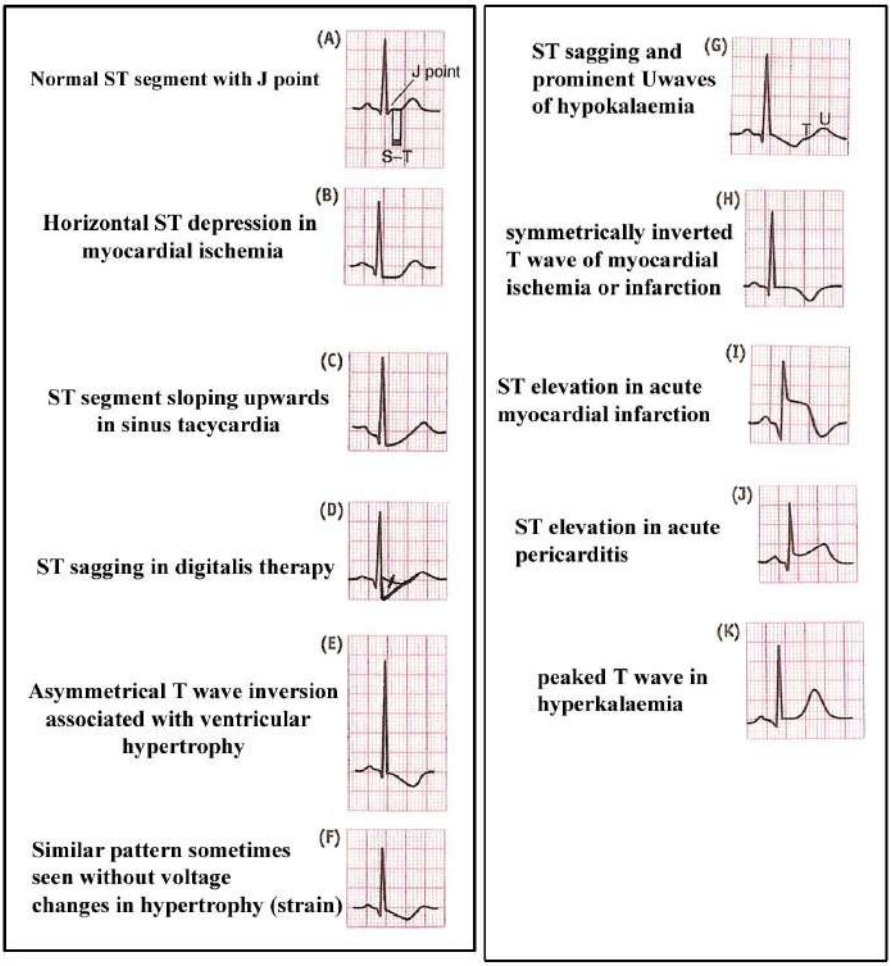
1. As a response to anxiety or fear.
2. As an orthostatic response.
3. As an postprandial response.
4. As a result of hyperventilation.
5. For no apparent reason.

This manifestations may frequently be normalized by recording the ECG after administration the potassium salt.

in some normal individuals, particularly young black, slight ST elevation

## *Differential Diagnosis of Q waves*

1. Physiological or positional factors.
   A- Normal variant "septal Q wave".
   B- Normal variant Q waves in V1 to V2, (see Fig.6-32A) and in leads



Note: The ischemic ECG changes may occur with normal angiography of the coronary arteries in :-

A- Hypertrophic obstructive cardiomyopathy.
B- Aortic stenosis.
C- Hypertensive heart disease.
D- Sever anemia.
E- Thyrotoxicosis

*Dextrocardia with situs inversus: P waves, QRS complexes and T waves are all inverted in lead I. see differential diagnosis of tall R waves

[a] small or absent R wave in the right to mid-precordial leads.
[b] progressive decrease in R wave amplitude from V1 to mid or lateral precordial leads.

***Comments***

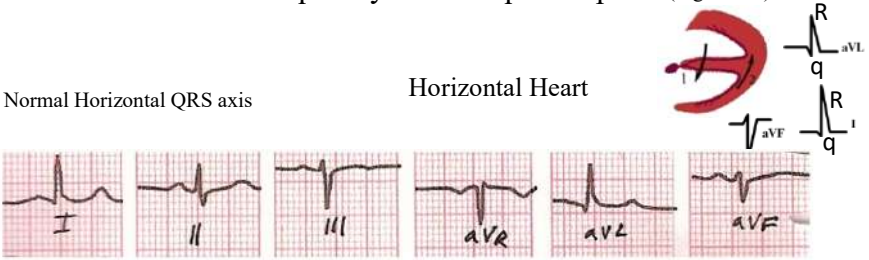
- Normal or physiological Q wave reflect normal septal activation from the right to the left results in septal R wave V2 and septal Q waves in V5-V6 (septal Q wave <0.04sec in width and <25% of R wave in the same lead in depth)


- The Q wave in myocardial infarction result from necrosis or dead tissue of the heart muscle that cannot transmitted the electrical activity (i.e. electrically inactive) in this regard the pathological Q wave (>0.04 width, >25% of R wave depth).


Necrotic muscle.

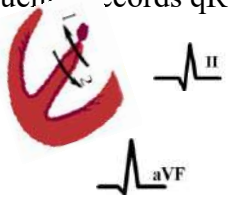
(looking through the window to the opposite wall of the heart)

- Other Q wave may be caused by the changes in anatomical position of the heart; the heart was theoretically thought to rotate around two axis, anteroposterior axis and oblique or longitudinal axis.
  1. Anteroposterior axis:- Rotation around this axis was thought to result in a horizontal or a vertical position
     *First* the horizontal position was thought to occur when the main body of the left ventricle was oriented upward and to the left, i.e. toward the standard lead I and aVL the high lateral leads, these leads would consequently records qR complex. (Fig.6-32A)




(Fig.6-32A) with a horizontal QRS position (axis), leads I and aVL, show qR complexes, lead II shows an RS complex, and leads III and aVF, show rS complexes.

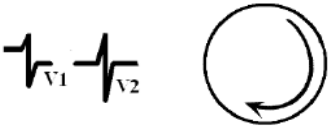
*Second* the vertical position was thought to occur when the anatomical position of the heart was vertical so that the main body of left ventricle wall was now dominantly oriented to standard lead II and aVF, which would consequent[illegible]ecords qR complex. (Fig.6-32B)



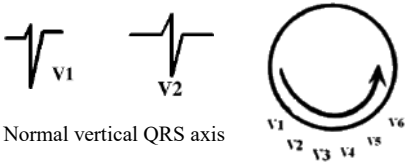

Vertical heart

2. Rotation of the heart around the oblique or longitudinal axis the terms clockwise and counter-clockwise rotation is still used, such changes are for example brought by increased electrical forces which are generated from the right or left ventricles, when these structures are hypertrophied respectively.

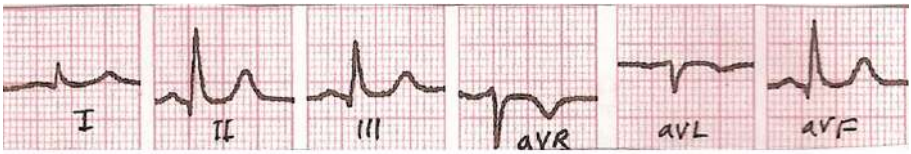
A- Clockwise rotation may result in formation of rS pattern in all or most of the precordial chest leads (e.g. RVH) (Fig.3-4B)




B- Counter-clockwise rotation may result in formation of Rs pattern manifest in leads V1 or V2 (V3) V4 to V6. (e.g. LVH) (Fig.3-2A)



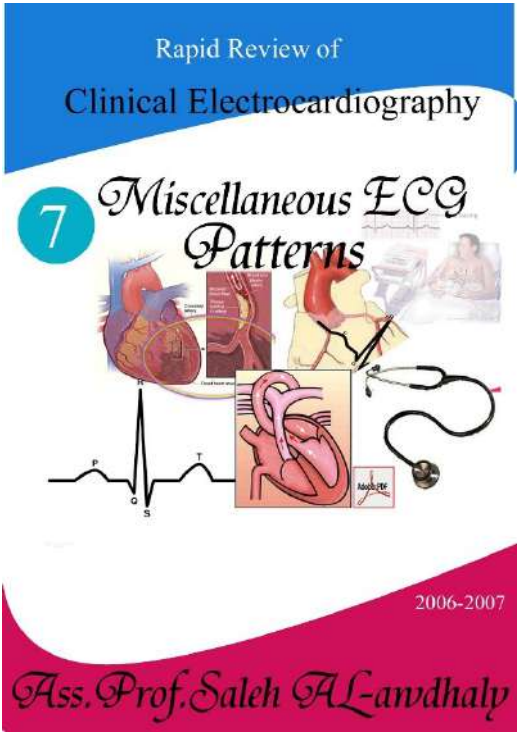

Normal vertical QRS axis



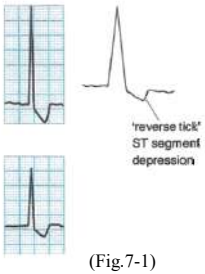
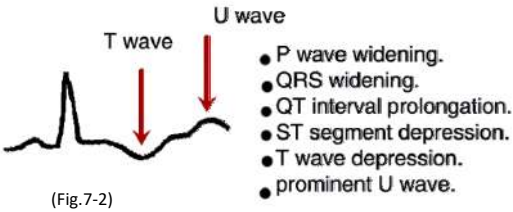

(Fig.6-32B) with a vertical QRS position (axis), leads II, III, and aVF show qR complexes, but lead aVL (and sometimes lead I) shows an RS complex. This is the reverse of the pattern that occurs with a normal horizontal axis.

**Chapter 7**

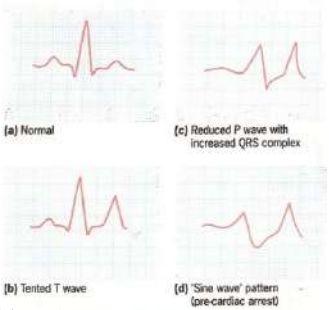
**Miscellaneous**



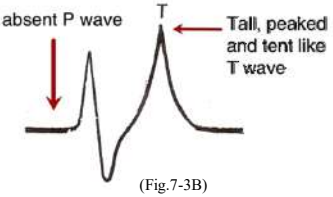
# Miscellaneous ECG changes

| | |
|---|---|
| ***I-Drugs***<br><br>1.Digoxin<br><br>A-Therapeutic effect.<br><br>(Therapeutic effect does not imply digitalis toxicity) (Fig.7-1)<br><br>B-Toxic effect | • ST segment depression (reversed tick).<br>• Reduction in the T wave size.<br>• Shortening of the QT interval.<br><br>  <br>(Fig.7-1)<br><br>• T wave inversion (change in the T wave during digitalis administration are often the earliest signs of digitalis toxicity).<br>• Any type of nearly every known arrhythmia. Especially ventricular ectopy.<br>• Digitalis does not effect the QRS complexes, because digitalis does not cause bundle branch block, as well as mobitiz type II second degree Heart block.<br>• Appearance of ectopic rhythm in the course of digitalis administration is nearly always signs of toxicity until prove other wise. |
| 2.Quinidine and related drugs like: (Fig.7-2)<br><br>procainamide<br><br>Disopyramide<br><br>Phenothiazine<br><br>Tricyclic<br><br>Antidepressant<br><br>Amiodarone | • Prolonged QT interval, mainly due to prolongation T wave duration.<br>• QRS prolongation.<br>• Incr<br><br>  <br>(Fig.7-2)<br><br>Amiodarone is the drug that typically prolonged the QT interval even at therapeutic doses. |
| II- ***Electrolyte***<br><br>A- Hyperkalemia<br><br>(Fig.7-3) | • The earliest ECG evidence usually appear in the T waves. The variety of the changes include:<br>1. Mild to moderate hyperkalemia (5-7 Meq/L)<br>▪ Tall symmetrical peaked "tents" T waves with narrow base (narrow base T wave may help to differentiate the peaking T wave of the hyperkalemia from other causes like acute ischemia)<br>2. More sever hyperkalemia (8-11 Meq/L)<br>▪ Widening of the QRS complexes.<br>▪ PR segment prolonged (i.e. delay of the AV conduction). |

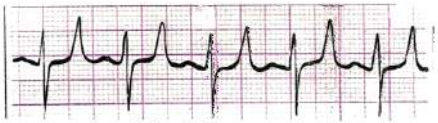
- In severe cases the ECG resembles, a sine wave.
- P wave disappearance (a trial arrest)



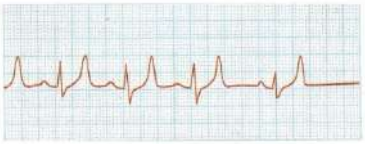

(Fig.7-3A) **Progressive ECG changes with increasing hyperkalaemia.**




(Fig.7-3B)



(Fig.7-3D) **Severe hyperkalaemia** is a common complication of acute renal failure and can be diagnosed on ECG, which shows peaked and symmetrical T waves. Hyperkalaemia is an indication for urgent dialysis; other indications include pulmonary oedema, severe acidosis and pericarditis.



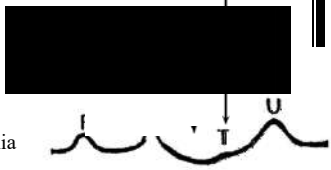
(Fig.7-3C) **Changes in the ECG associated with hyperkalaemia.** The T waves are tall, peaked and tent-like, and the PR interval and QRS complexes are lengthened. In severe cases, the P wave disappears and the QRS complex widens further, with loss of the T wave.

B- Hypokalaemia

(Fig.7-4A,B&C)

Mild (3-3.5) to moderate (2.5-3Meq/L) hypokalaemia

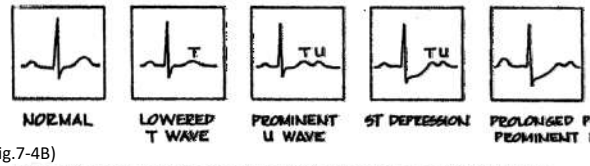
1. Progressive ST Segment depression.
2. Progressive decrease in T wave ampl
3. Increase U wave amplitude




(Fig.7-4A) Hypokalaemia

Sever hypokalaemia (<2.5Meq/L).

1. Fusion of T and U waves.
2. Increase QRS duration and amplitude.
3. Increase P wave duration and amplitude.
4. QT interval usually slightly prolonged.
5. Premature beats and sustained tachyarrhythmia.




(Fig.7-4B)

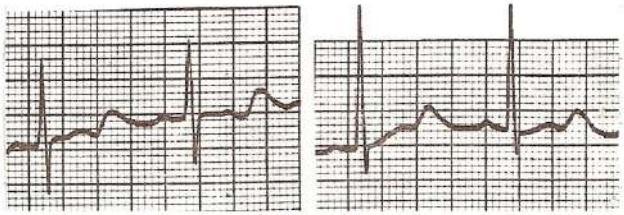
ECG patterns of hypokalemia sequentially as potassium level decreases.

C- Hypercalcaemia

(Fig.7-5A)

D- Hypocalcaemia

(Fig.7-5B& 7-6)

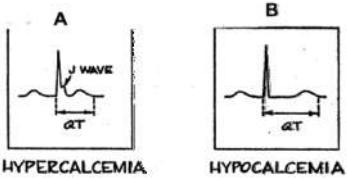


(Fig.7-4C) ECG changes of hypokalaemia

- Marked shortening of the QT interval due to shortening of the ST segment.

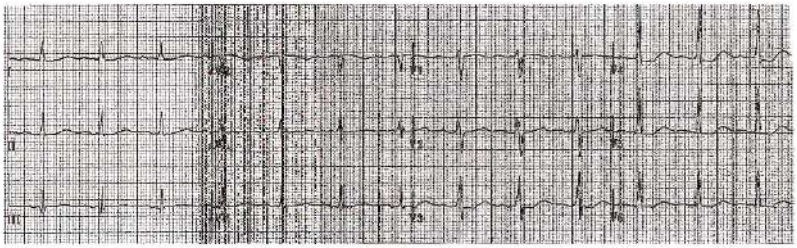
- Hypocalcaemia is the only condition which can prolong the ST segment without effecting the T wave.

Note the duration of the QT interval is inversely proportional to the serum calc




**(Fig.7-5)**

A, ECG changes in *hypercalcemia*, shortening of the *QT* interval primarily by shortening of the ST segment. A *J wave* may also be seen. B, *hypocalcemia*, lengthening of the *QT* interval primarily by lengthening of the ST segment.



**(Fig.7-6)the QT interval is clearly longer than half of the RR interval. this ECG is for a patient on antiarrhythmic agent quinidine, which may be used to treat paroxysmal AF.**

## Renal Failure

The triad of LVH (caused by hypertension), peaked T waves (caused by hyperkalemia) and prolonged QT interval (caused by hypocalcemia) should suggest chronic renal failure. (Fig.7-3D)

## Acute pericarditis

- May cause ST segment elevation and chest pain, raising the possibility of acute myocardial infarction. (Fig.7-7A)
- Acute pericarditis almost invariably associated with sinus tachycardia this is potential differentiating feature from acute myocardial infarction which may be associated with either sinus tachycardia or sinus bradycardia.
- Other feature that may help the differentiation of ST elevation of pericarditis from that caused by ischemia are as the following:

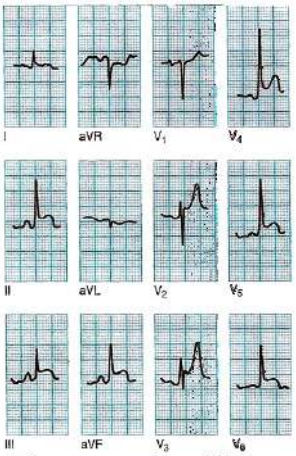
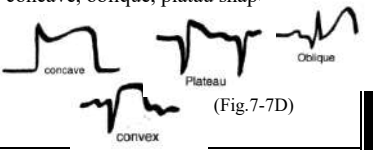
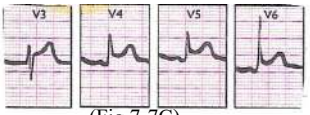
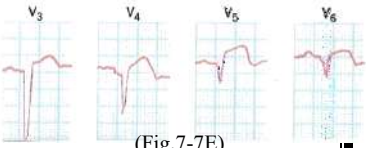



**Fig. 7-7A) pericarditis**
**key point:**
**widespread saddle-shape**
**ST segment elevation**

## ST segment elevation:- pericarditis versus ischemia

| | | Pericarditis<br><br>(Figs.7-7A,B&C) | Myocardial ischemia<br><br>(Figs.7-7D&E) |
|---|---|---|---|
| 1 | Leads distribution | In most of the ECG leads (diffuse distribution) does not localize in to right or left coronary artery distribution. | Reflect the area which effected by concerned coronary arteries. |
| 2 | Reciprocal ST depression | Absent | Usually present |
| 3 | ST segment shape | Upwardly concave<br>  <br><br>(Fig.7-7B) | Usually upwardly convex, but may be concave, oblique, platau shap<br>  <br><br>(Fig.7-7D) |
| 4 | Q wave | Absent | Present in Q wave MI |
| 5 | PR depression<br>  | Present, although it is specific but less sensitive, rarely seen | Absent |
| 6 | Timing of T inversion | T waves inversion, occur only after ST segment become, isoelectric. | T wave invert while ST segment still elevated |
| | Example<br><br>ECG strip |  <br><br>(Fig.7-7C) |  <br><br>(Fig.7-7E) |

## Pericardial effusion

Atrial of ECG changes that is virtually diagnostic of pericadial effusion are:

A- Low voltage QRS complexes[*1].

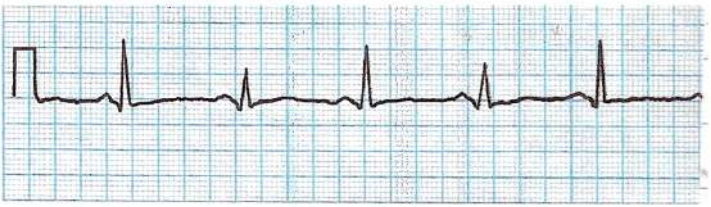
B- Low to inverted T waves in most leads.

C- Total electrical alternans[*2].

[*1]low QRS complexs is defined:- as a total amplitude of QRS complexes, in every one of the six extermaity leads is 5mm (0.5mv) or less; or over all voltage of QRS in I+II+III<15mm. which this is the case the amplitude in each of precordial chest leads(V1-V6) is usually less than 10mm, but this is not necessary for the diagnosis.(Fig.7-9)

[*2]Electrical alternans

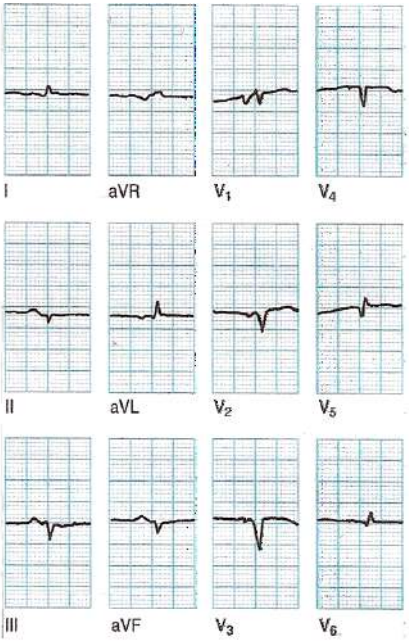
Is Beat-to-beat shift in the QRS axis associated with mechanical swinging of the heart to-and-fro in a large accumulation of fluid. Is virtually pathognomenic of cardiac tamponade, although not every patient with tamponade manifests this pattern.(Fig.7-8)



**(Fig.7-8) QRS electrical alternans. Note the alteration in QRS height in every other beat in this patient with large pericardial effusion**

## Differential diagnosis of low voltage QRS complexes

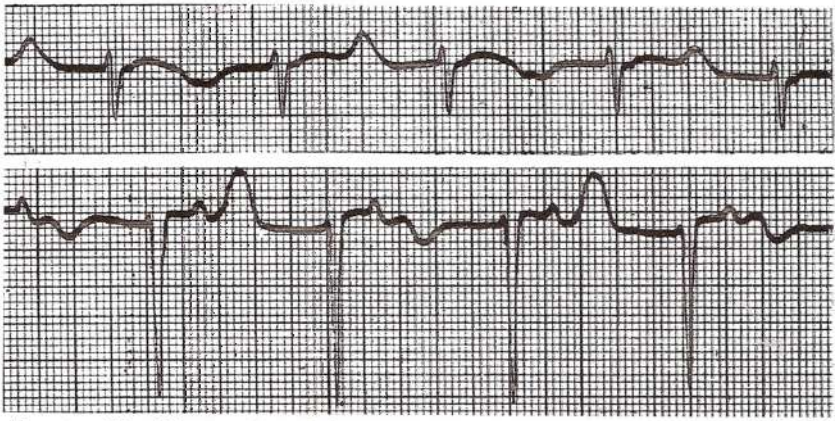
1. Artificats unrecognized standarization of ECG at one half usual gain (i.e. 1mv=5mm).
2. Normal variants (may also seen in pregnancy and obesity).
3. Pericardial tamponads (usually with sinus tachycardia).
4. Pleural effusion.
5. Chronic obstructive pulmonary disease (COPD).
6. Extensive myocardial infarction.
7. Myxoedema (usually with sinus bradycardia-"low and slow").
8. Cardiac inflitration (especially cardiac amyloid).
9. Cardiomyopathy (dilation, usually with diffuse fibrosis).
10. Left pneumothorax (mid left chest leads).




**(Fig.7-9) low voltage ECG**

**Repolarization alternans.**(Fig.7-10)

These alteration accompying only repolarization stage i.e. the alternation are limited to the T and/or U wave without QRS alternation. It is unexplained phenomenon that has been described in among other condition like left ventricular failure

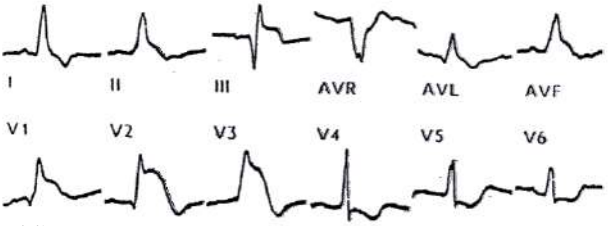


(Fig.7-10)- Alternation of T and U waves without QRS alternans. From a 64-year-old black man with hypertension, hyperkalemia, hypocalcemia, respiratory arrest, ventricular fibrillation, and seizures.

**Acute myocarditis**.(Fig.7-11)

The acute myocarditis may manifest as:

- Non specific T waves changes: increase amplitude of upright or inverted T wave.
- Depression or elevation of the ST segments.
- A prolonged QTc interval.
- Irregularities in the inscription of the QRS deflexion, resulting in notching or slurring, and, at times, and atypical intraventricular con[illegible]




**(Fig.7-11)** electrocardiogram was recorded from 12-year-old girl with acute diphtheritic myocarditis. it shown the following features:

A- Most leads reflect an increased ventricular activation time.
B- Slurred and irregular QRS deflections.
C- Elevation ST segment in leads V1, V2, V3, aVL, aVF.
D- Low to inverted T waves in all leads Except aVR.

## Early repolarization (high take-off ST segment).(Fig.7-12)

1. Characteristically the ST elevation as the following:-

   A- May rise 2 to 3 mm above the baseline.

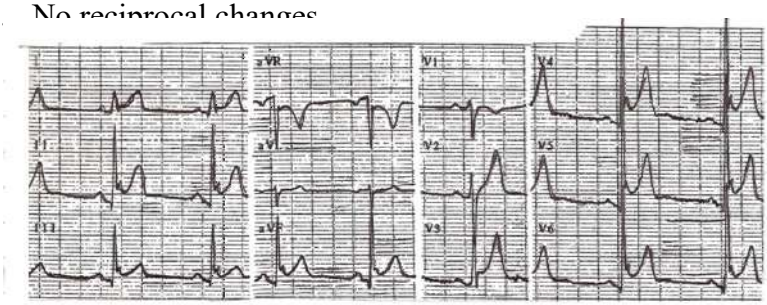
   B- Elevation are stable not undergo the evolutionary sequence of the pericarditis or MI.

   C- Always follows S wave.

2. Tall R waves in the left precordial leads.

3. Relatively tall and frequently symmetrical T wave, rarely T waves inversion present.

4 No reciprocal changes




**(Fig .7-12) "Early repolarization " Normal Variant.**
**early repolarization has to be differentiated from acute pericarditis.**
**Note**
**(1) a little barb at the J point, that with ST-T, produces a"Fishhook" in several leads.**
**(2) in V6 the degree of ST elevation is usually less than 25 percent the height of the T wave.**

***In diagnosing early repolarization, the clinical history is very important. Comparison with old ECGs and obtaining serial tracings may be necessary to show no change or evolution of the ST elevation.***

***Practical point***

## ECG changes in Cerbrovascular accidents (CVA)(Fig.7-13)

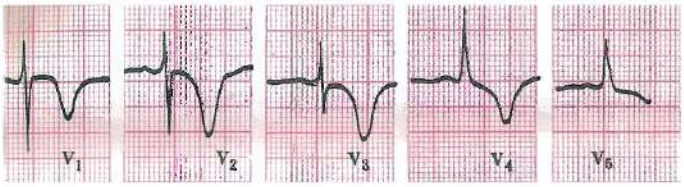
Usually, ECG changes occur in association with strokes either intracerebral hemorrhage or subarachnoid hemorrhages and others.

These ECG changes occur mainly during ventricular repolarization and are believed to be mediated through the autonomic nervous system and altered sympathetic tone.

The ECG changes of CVA include the following:

1. Abnormal and widened T waves that may be deeply inverted or tall and peaked.
2. Prominent U waves.
3. Prolonged Q-T interval.

These changes are termed "CVA pattern" and usually resolve with time.



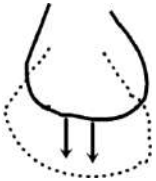

(Fig. 7-13) ECG "CVA pattern" observe the wide deeply inverted T waves. and prolonged QT interval in this patient with an intracerebral haemorrhage.

## Chronic obstructive pulmonary disease (COPD)(Fig.7-14)

COPD influences the electrical events of the heart in the following basic respects:-

1. The volaminous lungs impair electrical transmission, resulting in diminished QRS voltage.

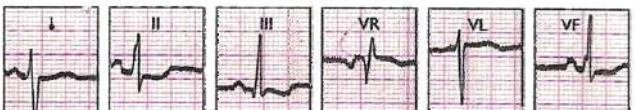
2. Due to lowering of the diaphragm, resulting with low anatomic position of the heart. This anatomic changes of the heart causes decrease progression of the R wave amplitudes in the precordial leads, with low voltage QRS complexes, especially in the left precordial leads. (Fig.7-14A)



(Fig.7-14A)

3. Pulmonary hypertension, will result in right ventricular hypertrophy and dilatation as well as right atrial enlargement (See fig.7-14B), which result with the following ECG changes. (See also Fig. 3-7B)

   - Increase P waves amplitude in lead II, III, aVF (P-pulmonale).
   - Right axis deviation.
   - Occasionally complete or incomplete right bundle branch block.



4. Posterior displacement of the apex. The terminal forces of ventricular depolarization, which are oriented superiorly and to the right, giving rise to SI, SII, SIII syndrome*.

5. Occasionally, there may be left axis deviation, the frontal planes QRS axis being directed upward and to the left, this occur in about 10% of cases. The mechanism still speculative.

***Summary ECG manifestation of COPD***

1. Right axis deviation is characteristic finding.
2. Absent of R wave in precordial leads simulate anterior MI.
3. Prominent R waves in right precordial leads (V1-V2) also ST segment depression may occur in these leads, due to right ventricular hypertrophy as a consequence of pulmonary hypertension.
4. Prominent P wave in leads II, III, aVF (P-pulmonale) due to right atrial abnormality.
5. Occasionally SI, SII, SIII syndrome may be present*.
6. Rarely in 10% of the patient left axis deviation may occur

*The SI SII SIII syndrome differential diagnosis.

The SI SII SIII syndrome may occur under the following circumstances:-

# Thyroid abnormalities.

**A- Hypothyroidism.**

a- Low voltage ECG.

b- Sinus bradycardia

c- Inverted T waves without ST segment deviation in many or all leads (slow and low ECG) is hypothyroidism until prove otherwise.

**B- Thyrotoxicosis.**

a- Unexplained AF(or sinus tachycardia at rest).

b- High voltage ECG, which may simulate LVH (left ventricular hypertrophy).

c- Decrease QT interval.

d- A prominent U waves in association with tachycardia should prompt you to think of hyperthyroidism.

## Obesity

A- Displacement of the heart by elevated diaphragm to the left but within normal range QRS axis.

B- Increasing the distance between the heart and the recording electrodes although the true low voltage QRS amplitude is rarely appears.

## Pregnancy

The pregnancy even at maximal distention at full term does not associate with left QRS axis deviation or with any significant changes of QRS axis.

**Note**
In full term pregnancy and obesity the QRS axis deviated leftward but within normal range, that is not further to the left, the further leftward >-30° in obese or pregnant women probably represents a pathological abnormality.

## Pulmonary embolism (Acute cor-pulmonale)(Fig.7-15)

May produce any of the following ECG patterns:

1. Sinus tachycardia, which consider the most common ECG finding.
2. Right ventricular strain, appearance of ST-T changes in right precordial leads (V1-V2).
3. SI QIII TIII (S in lead I, Q in lead III, T inversion in lead III, "see Fig.7-15" these changes are more specific but less sensitive) which may simulate that produced by acute inferior myocardial infarction is probably due to acute right ventricular dilatation.
4. Right axis shift, due to dilatation and strain of the right ventricle.
5. ST depression resulting from subendocardial ischemia.
6. Acute right bundle branch block (RSR pattern in V1) either complete or incomplete. This results from strain and dilatation of the right ventricle.

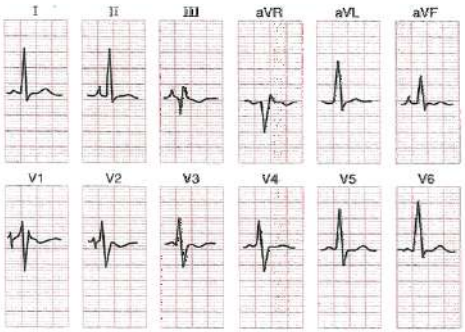



**Fig.7-15** ECG appearances in pulmonary embolism. Note tall, peaked P waves, partial right bundle branch block (rSr in V1), S in lead I, Q and negative T in III, and inverted T waves in V1 to V3.

## Mitral Valve prolaps

The ECG manifestations

1. Inverted T wave in standard leads II and III and lead aVF with a wide frontal plane QRS-T angle.
2. T wave inversion in the precordial leads, these T wave changes may normalize after effort, they may normalize spontaneously and unrelated to effort, emotion or changes in heart rate.
3. The isolated T wave negativity syndrome, which may only occurs in the midprecordial leads-leads V3 and V4- the right and left precordial leads reflecting upright T waves termed this the "isolated T wave negativity syndrome".
4. ST segment abnormalities:

   The billowing mitral leaflet syndrome may manifest with abnormalities of ST segment which may be indistinguishable from those associated with myocardial ischemia, these manifestations are usually seen in inferior leads, and the left precordial leads.
5. Prolongation of QT interval has also been reported.
6. Ectopic ventricular rhythms.
7. Sinoatrial block, LBBB, RBBB and left anterior hemiblock have also been observed in association with mitral valve prolaps.

## Comments

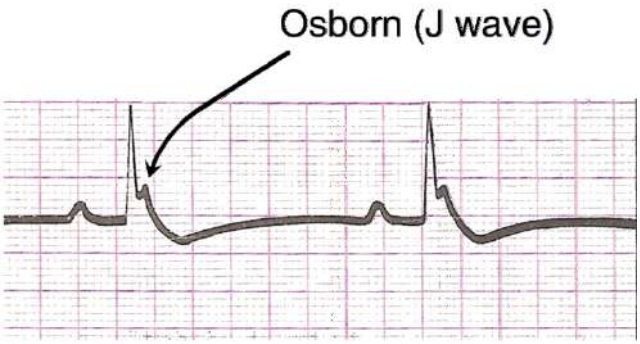
- The ECG manifestation of mitral valve prolaps, its non specific, may vary

## Hypothermia.(Fig.7-16)

1. The most characteristic ECG sign is the J or "Junctional wave" "Osborn wave".
2. Prolongation of QRS complexes.
3. Depression of the ST segment.
4. T wave depression.
5. Prolongation of QT interval.
6. Sinus bradycardia.
7. First and second degree heart block.
8. Ectopic rhythm.

**Note**

**The ECG** manifestation of hypothermia is completely reversible with a return to normal body temperature**.**




**(Fig.7-16)** the ECG changes in hypothermia. the Osborn wave (J wave) is frequently seen.

## **Amyloidosis**.(Fig.7-17)

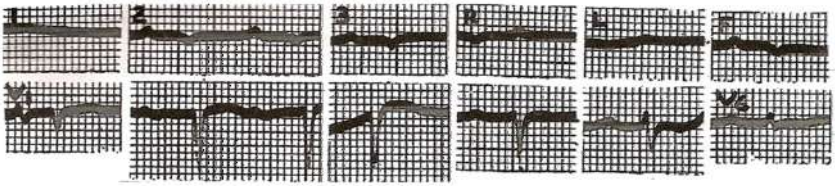
Amyloidosis may be suspected when the following combination ECG changes appear.

A- Low voltage of all waveforms in the limb leads.

B- Marked left axis deviation.

C- QS or minimal R waves in leads V1-V3 or in lead V4.

D- Prolonged AV conduction time.



**(Fig.7-17)** ECG in patient with Heart failure due to Amyloidosis.

# Chapter 8

# Artificial Cardiac

# Pacemaker

# Artificial Cardiac Pacemakers: A Rapid Review

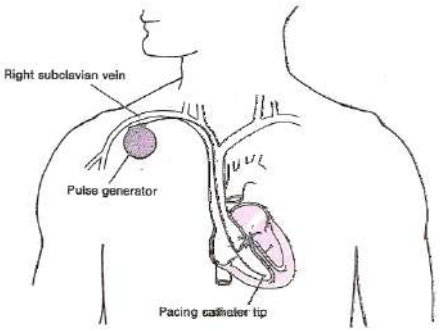
The topic of artificial pacemaker falls outside the scope of this book, but a few important aspects of artificial cardiac pacemakers are mentioned briefly.

## Key Definitions

### 1. Artificial Cardiac Pacemaker

An artificial pacemaker is an electronic device that delivers a depolarizing impulse to the myocardium when the heart's own internal pacemaker mechanism fail.

A pacemaking system consist of a pulse generator which contains the pacemaker electronics and power source, and pacemaker catheter or lead electrode (or electrodes in multichamber pacemakers) (Fig.8-1)




(Fig.8-1) implanted pacemaker generator (battery) with a wire electrode inserted through the right subclavian vein into the right ventricle.

2. **Pacing:-**

It is setting of the pace, or regulation of the rate of the heart.

3. **Capture:-**

Capture is the response by the heart to an pacemaker impulse that results in atrial and ventricular depolarization; every pacing spike should be followed by heart depolarization wave.

4. **Sensing:-**

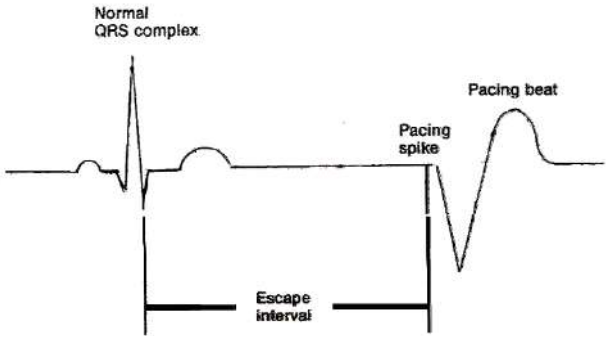
Sensing is the ability of the pacemaker to "see" electrically what the heart is doing by other mean is the ability to detect the intrinsic activity of the heart, and operate only when there is no ventricular complex or when the patient spontaneous beat below the preset rate.

5. **Escape Interval:-**

In "demand" pacemakers the escape interval is the time measured from a sensed normal intrinsic QRS complex to the next paced beat, they emit pulse only when the spontaneous heart falls below the escape rate of the pacemaker (e.g. 70 beats/min), they do not emit pulses when the patient spontaneous heart rate is faster than the escape rate of the pacemaker.

The pacemaker only provide impulses, where is no intrinsic QRS activity through escape interval. (Fig.8-2)

(Fig .8-2) An example of escape interval




The escape interval in single-chamber pacemakers is measured from the intrinsic QRS complex to the pacing spike. This interval is the longest time the pacemaker will allow the heart to go without contracting. Measured in milliseconds, the average escape interval for adults ranges between 1091 ms (55 beats/min) to 857 ms (70 beats/min).

## 6. Pacing interval:-

Is the time interval between the two consecutive pacing beats (Fig.8-3)

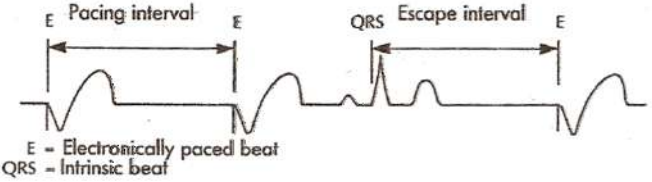



**Fig.8-3** A ventricular demand (QRS-inhibited) pacemaker emits an electronic pulse only when the intrinsic heart rate falls below the escape rate of the pacemaker.

***The pacing interval is further divided into***

A- Refractory period in which a ventricular demand pacemaker does not sense any electrical activity However if spontaneous ventricular beats or ventricular premature complex occurs during the refractory period, they are not inhibited but appear in ECG.

B- The alert period is follow the refractory period, during which the pacemaker can sense an R wave. If no intrinsic normal QRS

complex is sensed by the end of the alert period, the pacemaker emits another pulse (Fig.8-4).

## 7. Demand pacemakers:-

A demand pacemaker is a type of artificial cardiac pacing it allow the heart to beat normally, for example, if the pulse generator was set to deliver 60 impulses/min, it would not discharge pacing impulses until the patient own rate fall below 60 impulses/min.

## 8. Fixed rate pacemaker:-

A fixed rate pacemakers is a type of artificial cardiac pacing in which the pacemaker deliver pacing impulses at a preset rate not matter with the heart is doing in its own. for example a fixed rate pacemaker set to deliver 60 impulses/min would discharge impulses even if the patient's own intrinsic rate was above 60 beats/min.

This type of activity leads to what called competition between the pacemaker and the intrinsic normal rhythm of the heart.

The major potential problem associated with fixed rate pacing is the occurrence of pacing impulses during the relative refractory period of the normal cardiac cycle (a type R on T effect) that could put the patient into ventricular tachycardia or ventricular fibrillation fixed rate pacemaker are seldom used.

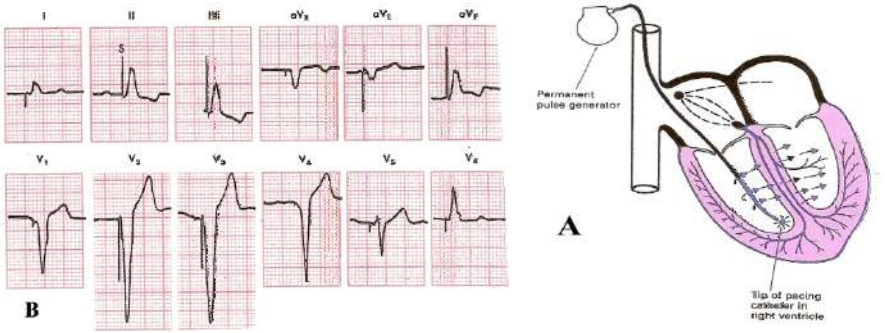
## 9. ECG pattern of the artificial pacemaker:-

The most common type of pacemaker uses a transvenous pacing catheter with a bipolar tip, which is ideally lodged in or near the apex of the right ventricle

Where an impulse is delivered from the pulse generator through the pacing catheter to the right ventricle depolarization of the myocardium occurs. (Fig.8-5A)

Because the depolarization originates from a ventricular focus, the QRS complex produced does not appear normal, the current travels in the heart from right ventricle across the interventricular septum to the left ventricle, because the left ventricular stimulation is delayed, the QRS complex will appear wide and the ECG show a left bundle branch (LBBB) pattern (Fig.8-5B)

the ECG may show right bundle branch block pattern when the epicardial pacing catheter is inserted through the pericardium after thoracic wall incision and pace the left ventricle directly then the right ventricle depolarized slowly from the left.

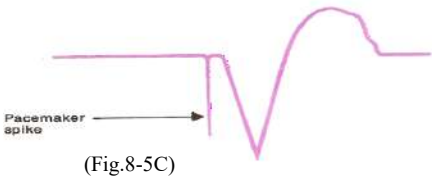



(Fig.8-5 A&B )

A- The current flow in the heart when the depolarization is initiated in the right ventricle.

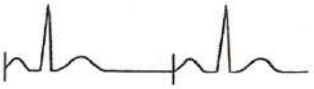
B- A pacemaker inserted in the right ventricle generally produces a pattern resembling that of left bundle branch block, with wide QS in lead V1 and a wide R wave in lead V6. This pattern caused by delayed depolarization of the left ventricle. Notice the pacemaker spike in each lead preceding the QRS complex. In some leads (e.g. II) the spike (S) is positive; in others (V1 to V6) is negative.

- When the pacemaker "Fires" the electrical activity produced by pulse generator is recorded on the ECG as a pacing "spike". (Fig.8-5C)




(Fig.8-5C)

- When the atria are being paced, the pacing spike will be followed by a P wave, this may conducted normal pathway and followed by a normal QRS complex. (Fig.8-5D)



(Fig.8-5D)

- In ventricular pacing, a pacing will be followed by broad QRS complexes. (Fig.8-5E)

(Fig.8-5E)

- In dual chamber pacemaker (DDD) when the electrode placed in both the right atrium and the right ventricle, the pacemaker spike seen before the P waves and before the QRS complexes. (Fig.8-5F)

(Fig.8-5F)

- Pacemaker sensing with normal intrinsic cardiac activity, the pacemaker will not discharge pacing impulses, until these rate fell below preset rate. (Fig.8-5G)

(Fig.8-5G)

**10. Generally the pacemakers are classified into**

A- Temporary pacemakers.

B- Permanent pacemakers.

***Practical guide lines***

*A percussion pacing is performed by delivering gentle blows to the precordium (alongside the lower left sternal edge) to stimulate intrinsic cardiac rhythm the technique can be remarkably effective and can buy enough time to arrange further treatment appropriate.*

**11. Summary of the indications for pacemakers**

To summarize pacemakers generally are indicated for:-

A- Symptomatic bradycardia commonly, these are

1. AV blocks (second-degree mobitiz II, high grade, or third-degree).

2. Sinus node dysfunction.

3. Carotid sinus hypersensitivity.

4. Drug overdose (e.g. digoxin, β blocker, verapamil)

B- In asymptomatic patients pacing commonly should be considered in the following condition:-

1. Complete heart block (particularly with escape < 40 beats/min or pause > 3 seconds).

2. Mobitiz II block (especially associated with bifascicular or trifascicular block).

3. Postoperative AV block.

4. Preoperative: if the surgery is required in general anesthesia patient with type 2 or complete heart block, temporary pacing may be needed.

***Practical guide lines***

- Surgeons and anesthetics must always be made aware if a patient undergoing surgery has a permanent pacemaker, to avoid interference with, or damage to, the pacemaker from dithering, to minimize the dangers, place the active diathermy electrode at least 15 cm from the box as possible.

## 12. Pacemaker codes

Three-position pacemaker code is often used to described pacemaker's functions and mode of the response.

- The 1st letter indicates the chamber paced (A = atria, V = ventricles, D = Dual chamber).
- The 2nd letter identifies the chamber sensed (A = atrium, V = Ventricles, D = Dual, O = none).
- The 3rd letter indicates the pacemaker response to sensing (T = Triggered, I = Inhibited, D = Dual both chambers, O = none).

  The basic three letters has been expanded to five letters.

- The 4th letter defined programmable capabilities (R = Rate-responsive).
- The 5th letter indicates antitachycardia function (P = Means that in tachycardia the pacemaker will pace the patient, S= means In tachycardia the pacemaker chocks the patient, D = Dual ability to pace and shock, O = None)

***In practice***, relatively few modes are actually used examples are as follows.

- VVI (ventricular paced, ventricular sensed, inhibited in response to a ventricular events).
- VVIR (same as VVI but also has rate responsiveness*).
- DDD (Dual chambers, atria/ and ventricular pacing and sensing with triggered and inhibited response to a sensed atrial or ventricular events).
- DDDR (same as DDD but also has rate responsiveness).

*Rate responsiveness (the "R") is used to increase heart rate of the pacemaker in response to physical demand, so that the rate at which pacing occur is appropriate to metabolic demands (i.e. sensor that responds to body motion, respiratory rate, blood temperature etc….).

## 13. Pacemaker malfunction (See also the glossary)

1. Failure to sense.
2. Failure to pace.
3. Oversensing.
4. Pacemaker-mediated tachycardia.
5. Pacemaker-induced tachycardia.
6. Pacemaker syndrome.

## 14. Complications of pacemaker therapy

1. Infection.
2. Erosion.
3. Pocket hematoma.
4. Lead displacement.
5. Occasionally a pacemaker also paced the muscles of the chest wall or the diaphragm, resulting in patient discomfort.

## 15. Electromagnetic interference

The following factors may interfere with pacemaker function include:

- High tension cables.

- Arc-Welding equipment and some medical equipment.
- High energy radars.
- Digital cellular telephone may cause similar problem but only when the telephone is held in very close proximity to the pulse generator.

**N.B.**

For more details about artificial pacemakers, Text Book of cardiology should be consulted.

# Glossary

***Aberrant ventricular conduction*** : the temporarily abnormal intraventricular conduction of supraventricular impulse , usually associated with a change in cycle length .Aberrancy differs from "normal" conduction in two ways : (a)the impulse does not travel normally through the ventricular Purkinje system to reach the ventricular myocardium and (b)it is not the usual conduction pattern for that individual . (see preexcitation syndrome)

***Accelerated automaticity*** : increase in the rate of impulse

formation within the pacemaker cells .

***Accelerated rhythm*****:** the rate is increased above its normal limit.

***Afterpotential*** : also termed after depolarization .A small electrical potential that occurs following the completion of repolarization which may produce or "trigger" a second automatic activation of the cell termed triggered activation .

***Anterior infarction*** : infarction in the distribution of the left anterior descending coronary artery , involving primarily the middle and apical sectors of the anterior-septal quadrant of the left ventricle . (see ch.5)

***Antidromic tachycardia*** : an Reentrant Junctional Tachycardia of the AV bypass variety produced by macroreentry in which the impulse recycles sequentially through an accessory AV bypass pathway ,a ventricle , the AV node ,and an atrium . (Fig.4-20C)

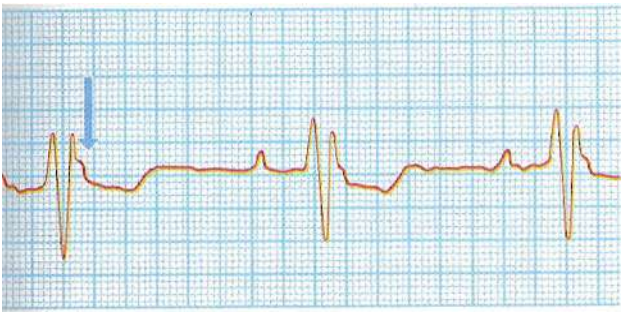
***Apical infarction*** : infarction in the distribution of any of the major coronary arteries , involving primarily the apical sectors of the posterior-lateral and inferior quadrants of the left ventricle

***Arrhythmia*** : any cardiac rhythm other than sinus rhythm.

***Arrhythmogenic right ventricular cardiomyopathy*****:** is

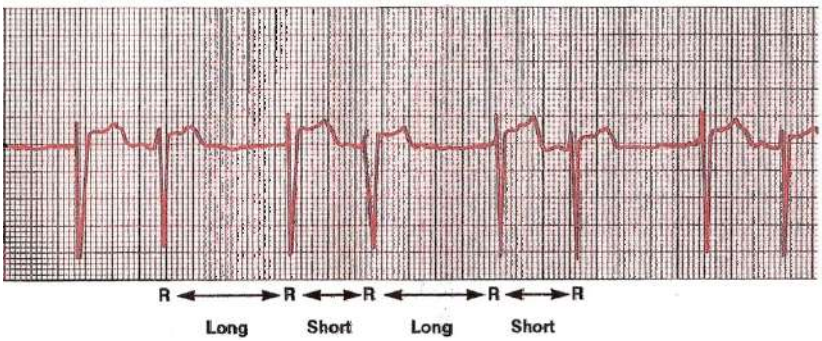
characterized by progressive fibrofatty replacement of the right ventricular myocardium. This leads to ventricular arrhythmia and risk of sudden death in its

early stages and right ventricular or biventricular failure in its later stages. It is familial in at least 50% of cases, most commonly with an autosomal dominant pattern of inheritance.



ECG from an adult with ARVC demonstrate RBBB and precordial T wave inversion with epsilon wave (arrow)

***Ashman's syndrome*** : Ashman's pattern of long RR intervals followed by short R-R interval, may occur in the atrial fibrillation and also in other types of dysarrhythmia, it caused by a conduction phenomena in the ventricles.




***A systole*** : a rhythm synonymous with sinus pause in which there is a period of slowing of the heart rate with neither atrial nor ventricular activity present on the ECG .(see Fig.4-31B)

***Athletes T wave*:** in the athletes heart the T waves may be peaked, tall, biphasic, isoelectric, or even frankly inverted. T wave inversion in the precordial or limb leads have been seen in up to 30% of endurance athletes, these T waves changes may normalize with exercise or with isoproterenol infusion.

***Atrial fibrillation*** : the tachyarrhythmias at the rapid end of the flutter/fibrillation spectrum produced by macroreentry within multiple circuits in the atria and characterized by irregular multiform f waves .

***Atrial flutter*** : A rapid atrial rhythm sustained by a macro-reentrant circuit Atrial tachycardia : A single focus firing rapidly.

***Atrial flutter/fibrillation*** : the tachyarrhythmia in the middle of the flutter/fibrillation spectrum having some aspects of flutter and some of fibrillation .

***Atrial flutter/fibrillation spectrum***: a range of tachyarrhythmias caused by macroreentry in the atria which extends from flutter with an atrial rate of 200 beats/min through flutter-fibrillation and coarse fibrillation to fine fibrillation with no atrial activity detectable on the body surface .

***Atrial rhythm*** : a rhythm with rate less than 100 beats/min with abnormally directed p waves (indicating origination from a site in the atria other than the sinus node) preceding each QRS complex .

***Atrioventricular(AV) block*** : a conduction abnormality between the atria and the ventricles . Both the severity and the location should be considered.

***Atrial premature beat(APB) :*** a P wave produced by an impulse that originates in the atria and appears prior to the expected time of the next P wave generated from the sinus node .

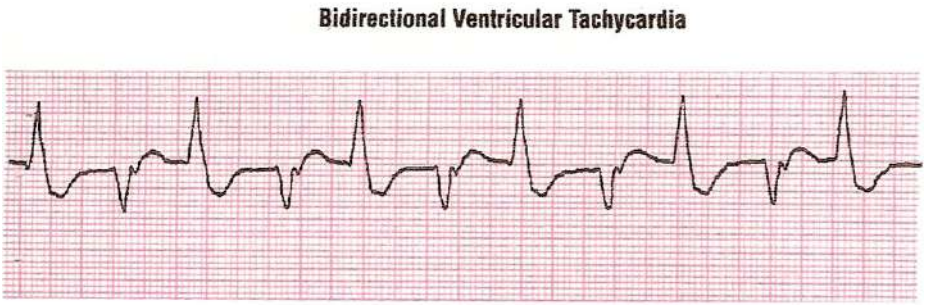
***Automaticity*** : the ability of specialized cardiac cells to achieve spontaneous depolarization and function as "pacemakers" to form new impulses .

***AV dissociation*** :a condition of independent beating of the atria and ventricles caused either by block of the atrial impulse in the AV junction or by interference with conduction of the atrial impulse by ventricular impulse.(see AV conduction disturbance)

***AV junction*** : the cardiac structures that electrically connect the atria and ventricles normally including the AV node and common (His) bundle and abnormally an accessory AV conduction (Kent) bundle .

***AV nodal tachycardia***: an Reentrant Junctional Tachycardia (RJT) produced by microreentry within the AV node.

***Bidirectional tachycardia*** : VT with a regular rate and two QRS morphologies with opposite polarities . (See Fig. below)




***Bigeminy*** :a rhythm pattern in which every sinus beat is followed by a premature beat .

***Block*** : either a delay or total failure of the impulse conduction through a part of the heart .

***Bradyarrhythmia*** : any rhythm with ventricular rate <60 beats/min .

***Bundle of Kent*** : a congenital abnormality in which a bundle of myocardial fibers connects the atria and ventricles .

***Calcium antagonist*** : a drug that diminishes calcium entry into cells and slows conduction through the AV node .

***Capture beat*** : atrial impulses that activates the ventricles .The term is used when this interrupts an independent ventricular rhythm .

***Cardioversion*** : application of electric shock in order to restore a normal heartbeat.

***Carotid sinus stimulation*** : contact with area in the neck overlying the bifurcation of the carotid artery which contains receptors capable of enhancing parasympathetic nervous activity .

***Chaotic atrial tachycardia*** **:** another term used for multifocal atrial tachycardia .

***Coarse fibrillation*** : prominent f waves in some of the ECG leads .

***Collateral blood supply*** : the perfusion of an area of myocardium via arteries that have developed to compensate for an obstruction of one of the principal coronary arteries .

***Compensatory pause*** : the long cycle length (pause) following a premature beat (PB) completely "compensates for "the short cycle length preceding the PB . This is identified when the interval between the beginning of the P waves of the sinus beats preceding and following a PB is equal to two PP intervals of sinus beats not associated with PBs .

***Concealed AV bypass pathway*** : a Kent bundle that is only capable of AV conduction and, therefore, is incapable of producing ventricular pre-excitation.

***Concealed conduction*** : conduction of an impulse that is recognizable only by its effect on the subsequent beat or cycle.

***Concordant precordial negative*** : an abnormally wide QRS complexes are predominately negative in all six of the precordial leads.

***Concordant precordial positive*** : abnormally wide QRS complexes are predominately positive in all six of the precordial leads.

***Couplet*** : two consecutive premature beats.

***Coupling intervals*** : the time durations between the ectopic sinus beats and preceding beats.

***Defibrillation*** : termination of either atrial or ventricular fibrillation using an extrinsic electrical current.

***Degree*** : a measure of the severity of AV block .

***Delta wave*** : a slowing of the initial aspect of the QRS complex caused by premature excitation (Preexcitation) of the ventricles via a bundle of Kent.

***Demand mode*** : an artificial pacemaking system with capability to sense , and to be inhibited by , intrinsic cardiac activity.

***Digitalis*** : a drug which occurs naturally in the foxglove plant , that is used both to increase the contraction of the cardiac muscle and decrease conduction through the AV node.

***Digitals toxicity*** **:** an arrhythmia produced by the drug digitalis.

***Dysarrhythmia*** : synonym (by usage) for arrhythmia.

***Echo beat*** : an atrial premature beat(APB) produced by reentry within the AV node.

***Entrainment*** : The ability to capture the reentry circuit with atrial pacing.

***Epicardial injury*** : the deviation of the ST segments that occurs with transmural ischemia or with pericardial irritation.

***Escape beat*** : a beat from a part of the pacemaking and conduction system which ends in a cycle that is longer than the basic cycle.

***Escape rhythm*** : rhythms that originate from sites in the pacemaking and conduction system other than the sinus node following a pause created by the failure of either the normal sinus impulse formation or atrioventricular impulse conduction.

***Excitable gap*** : A cyclic area of non-refractory tissue within the pathway of a reentry circuit.

***F waves*** : the irregular multiform atrial activity characteristic of fibrillation.

***F waves*** : the regular uniform sawtooth-like atrial activity characteristic of flutter.

***Fast- slow AV nodal tachycardia*** :An RJT of the AV nodal variety produced by microreentry in which the impulse travels down the fast pathway and up the slow pathway .

***Fine fibrillation*****:** either minute f waves or no atrial activity at all in any of the ECG leads.

***First degree AV block*** :atrial impulses conducted to the ventricles with PR intervals >0.20 sec .

***Fixed-rate artificial pacemaker*****:** a device capable only of generating cardiac impulses without sensing the patient's own intrinsic rhythm.

***Flutter*** :A continuously waving pattern on the ECG without isoelectric baseline in at least one lead ,irrespective of the rate .

***Footprints of the wenckebach*** : the patterns of clusters of beats in small groups with gradually decreasing intervals between beats preceding a pause which is equal to less than two times the length of the shortest interval .

***Heart block*** : another term used for AV block .

***His bundle electrograms*** : intracardiac recordings obtained via a catheter positioned across the tricuspid valve adjacent to the common or His bundle . They are used clinically to determine the location of the AV block when this is not apparent from the surface ECG recordings .

***Holter monitoring*** : continuous ECG recording of one or more leads , either for the detection of abnormalities of morphology suggestive of ischemia or abnormalities of rhythm .

***Hyperacute T waves*** : the tall peaked T waves that occurs during early phase of transmural ischemia .

***Hypertrophic cardiomyopathy*** : a condition in which the cardiac performance is decreased because the thickened myocardium has decreased contraction capability .

***Hysteresis*** : delay built into a demand pacemaker to provide a longer period before initiating an impulse following a period of inhibition by intrinsic rhythm than that between consecutive generated impulses .

***Implantable Cardiovertor Defibrillator ICD:*** internal cardiac placement of a device capable of delivering electrical shocks to the heart to interrupt a life-threatening run of ventricular tachycardia or ventricular fibrillation and thereby prevent syncope or sudden death. This therapy is used as secondary prevention in patients who have already been resuscitated from an episode of cardiac arrest due to VT or VF. And it is also recommended in primary prevention or as prophylactic therapy (see text for details)

***Infarct expansion*** : partial disruption of the myocardial wall in the area of a recent infarction which results in thinning of wall and dilation of the chamber .

***Inferior infarction*** : infarction in the distribution of the posterior descending coronary artery , involving primarily the basal and middle sectors of the inferior quadrant of the left ventricle , but often extending into the posterior aspect of the right ventricle.

***Infranodal block*** : AV block that occurs distal or below the AV node and ,therefore , within either the common bundle or in both the right and left bundle branches .

***Interectopic intervals*****:** the times between consecutive premature beats.

***Interpolated*** : occurring between normal beats .

***Ischemic heart disease*****:** cardiac abnormality caused by decreased blood flow to the myocardium usually because of atherosclerosis with or without superimposed thrombosis in the coronary arteries .

***Isorhythmia*****:** the atria and ventricles are beating independently(dissociation), but at very similar rates.

***Isorhythmic dissociation*** : AV dissociation with atria and ventricles beating at the same or almost the same rate .

***Junctional premature beat(JPB):*** a P wave and QRS complex produced by an impulse that originates in the AV node, His bundle , or kent bundle and appears prior to the expected time of the next P wave and QRS complex generated from the sinus node.

***Junctional rhythm*** :a rhythm with a rate less than 100 beats/min with an inverted P wave direction visible in the frontal plane leads and normal appearing QRS complexes . The P waves may precede or follow the QRS complexes or may be obscured because they occur during the QRS complexes.

***Juvenile T wave*****:** T wave inversion may occur as normal variant in the right side to mid chest leads of the children (The Juvenile T wave pattern) and may persist to adulthood.

***Lateral infarction*** : infarction in the distribution of the a"diagonal" or "marginal" coronary artery , involving primarily the basal and middle sectors of the anterior-superior quadrant of the left ventricle .

***Left coronary dominance*** : unusual coronary artery anatomy in which the posterior descending artery is a branch of the left circumflex coronary .

***LENĒGRE'S (LEV'S)disease*** : both Lenegre and Lev described variation of fibrosis of ventricular Purkinje in the absence of other significant cardiac disease .

***LGL syndrome*** : the clinical combination of a short PR interval , normal QRS appearance , and supraventricular tachyarrhythmias .

***Lidocaine*****:** a compound with local anesthetic properties that is used in treatment of reentrant tachyarrhytthmias .

***Lone fibrillation*** **:** atrial fibrillation occurring in an individual with no evidence of cardiac disease .

***Low voltage*** : total amplitude of the QRS complex less than 0.50 mV in any limb lead and less than 1.0 mV in any precordial lead .

***Macroreentry*** : recycling of an impulse around a circuit that is large enough for its own activation to be represented on the surface ECG .

***Mahaim tract*** : a rarely occurring congenital anomaly in which Purkinje fibers from the common (HIS) bundle lead into the septal myocardium .

***Microreentry*** : recycling of an impulse around a circuit that is too small for its own activation to be represented on the surface ECG.

***Mitral valve disease*** **:**either abnormally tight(stenotic) or loose(insufficient) valve between the left atrium and left ventricle.

***Mobitz type 1 (type 1)*** : a pattern of AV block in which there are varying PR intervals . It is typical of block in the AV node that is capable of wide variations in conduction time . Wenckebach sequences are the classic form of type 1 block .

***Mobitz type 11 (type11)*** : a pattern of AV block in which there are constant PR intervals despite varying RP intervals . It is typical of block in the ventricular Purkinje system which is incapable of significant variations in conduction time .

***Monomorphic VT*** :VT with a regular rate and consistent QRS morphology .

***Multifocal atrial tachycardia (MAT):*** a rapid rhythm produced by pacemakers located at multiple sites within the atria.

***Multifocal VPBs*** : premature beats originating from two or more different ventricular locations .

***Multiform VPBs*** : premature ventricular beats with two or more different morphologies in a single ECG lead .

***Myocardial rupture*** : complete disruption of the myocardial wall in the area of a recent infarction which results in leakage of blood out of the chamber .

***Necrosis*** : death of a living tissue; termed an infarction when it is caused by insufficient supply of oxygen via the circulation .

***Neurocardiogenic syncope*** : occurs when an individual experiences vasovagal reactions because of some factor which causes increased parasympathetic or decreased sympathetic nervous system activity . It may be detected using head-up tilt test.

***Nonsustained VT*** : VT less than 30 sec in duration .

***Orthodromic tachycardia*** : an RJT of the AV bypass variety produced by macroreentry in the impulse recycles sequentially through the AV node ,a ventricle , an accessory AV bypass pathway ,and an atrium . (Fig.4-20B)

***Osborn waves*** : abnormal ECG waveforms caused by hypothermia . (Fig.7-16)

***Overdrive suppression*** : a decrease in the rate of impulse formation resulting from premature activation of the pacemaking cells .

***Oversensing :*** abnormal function of an artificial pacemaker in which electrical signals other than those representing activation of the myocardium are sensed and inhibit impulse generation .

***Pacemaker cells*** : specialized cardiac cells that are capable of automaticity .

***Pacemaker –induced tachyarrhythmias*** : a rapid rate produced by the artificial pacemaker . A paced impulse occurring during the time when the myocardium is

vulnerable to induction of a reentrant circuit because it is just emerging from total refractoriness .

***Pacemaker –mediated tachyarrhythmias*** : a rapid rate occurring with a dual chambered pacing system in which some of the beats are intrinsic and some are from the artificial device .

***Pacemaker spikes*** : high frequency signals appearing on an ECG which represent impulses generated by an artificial pacemaker .

***Pacemaker***: a cell in the heart or an artificial device that is capable of the formation or generation of an electrical impulse.

***Pacing electrodes*** :in contrast to the electrodes used to record the ECG , pacing electrodes are designed to transmit an electrical impulse to the myocardium . In pacing systems with sensing capability , however , they also transmit the intrinsic impulses to the artificial device.

***Palpitation*** : a sensation felt in the chest as a result of ventricular contraction .

***Parasystole***: an arrhythmia produced by a cardiac cell that is functioning as a pacemaker without the capability to sense the surrounding cardiac activity. (Fig.4-26)

***Paroxysmal atrial tachycardia(PAT) with block***: a tachyarrhythmia ,commonly caused by digitalis toxicity, in which a rapid atrial rhythm Is accompanied by failure of some of the impulses to be conducted through the AV node of the ventricles.

***Paroxysmal***: the sudden occurrence of an arrhythmia.

***Pericardial effusion*** : an increase in the amount of fluid in the pericardial sac .

***Pericardial sac*** : the fluid-filled space between the two layers of the pericardium .

***Pericardial tamponade*** : filling the pericardial sac with fluid which restricts the relaxation of the cardiac chambers .

***Pericarditis*** : acute or chronic inflammation of the pericardium .

***Polymorphic VT*** : VT with a regular rate but with frequent changes in QRS morphology.

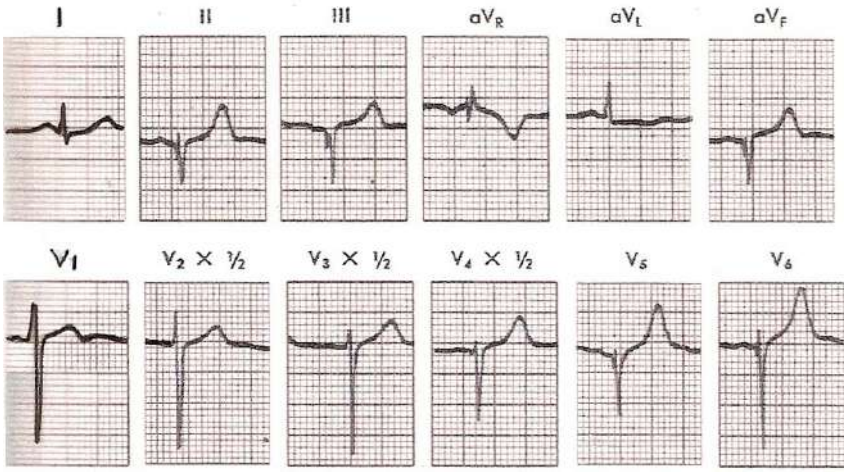
***Posterior infarction*** : infarction in the distribution of the left circumflex coronary artery , involving primarily the basal and middle sectors of the posterior-lateral quadrant of the left ventricle.

***premature beat*** : a beat that occurs prior to the time when the next normal beat would be expected to appear.

***Pro-arrhythmic effects*** : cardiac arrhythmias produced by therapeutic agents that are typically used to prevent or terminate arrhythmias .

***Procainamide*:** a compound related to the local anesthetic procaine that is used in the treatment of reentrant tachyarrhythmias.

***Pseudo infarction:*** is prominent of Q waves in the absence of MI called pseudo infarction. "see the differential diagnosis of Q wave"




Hypertrophic obstruction cardiomyopathy (HOCM). Notice the prominent pseudoinfarction Q waves, which are the result of septal hypertrophy. Leads V2 to V4 are recorded at 1/2 the usual voltage calibration.

***Pseudo normalization :*** in patient with evolving MI infarction. T waves are inverted, during ischemic episodes temporarily upright T waves in these leads is called T wave pseudo normalization.

***Pseudo aneurysm :*** is a rupture of the myocardium with full thickness penetration through the wall with the rupture being contained by pericardial.

***Pulse generator*** : the device that produces electrical impulses as the key component of an artificial pacing system .

***Quinidine*** : a drug , which occurs naturally in the bark of the cinchona tree , that prolongs myocardial recovery time and protects against some tachyarrhythmias . However, quinidine and other related drugs may also produce tachyarrhythmias by over prolongation of the recovery time .

***Reciprocal*** : deviation of the ST segments in the opposite direction from the maximal deviation .

***Reentrant junctional tachyarrhythmias*** : any of the

tachyarrhythmias (RJTs) produced by continual recycling of an impulse through structures which are present either normally or abnormally between the atria and the ventricles .

***Reentry circuit*** : a circular course traveled by a cardiac impulse , created by reentry, and having the potential for initiating premature beats and tachyarrhythmias .

***Relative refractory*** : cells that have only partially recovered from their previous activation and are , therefore , capable of slow conduction of another impulse .

***Retrograde atrial activation*** : spread of the impulse from the AV junction through the atrial myocardium toward the SA node .

***Right coronary dominance*** : the usual coronary artery anatomy in which the posterior descending artery is a branch of the right coronary .

***Right VPBs*** : premature beats originating from right ventricle , always with a V1 negative morphology .

***R-on-T VPB*** : a VPB that occurs so premature that it occurs during the T wave of the previous beat.

***RP interval*** : the time between the beginning of the previously conducted QRS complex and the beginning of the next conducted P wave .

***RP/PR reciprocity*** : the inverse relationship between the interval since the previously conducted beat (RP interval) and the time required for AV conduction (PR interval). This occurs in type 1 AV block .

***Second degree AV block*** : some atrial impulses are conducted to the ventricles while some fail to be conducted .

***Sick sinus node*****:** a term that is loosely used clinically to describe any abnormal low sinus rate. These bradyarrhythmias are more likely caused by increased parasympathetic nervous activity than by disease in the sinus node.

***Sick sinus syndrome*** : inadequate function of cardiac cells with pacemakung capability , resulting in continuous or intermittent slowing of the heart at rest and an inability to appropriately increase the rate with exercise .

***Silent ischemia*** : the appearance of ECG changes that indicate ischemia or infarction without ischemic pain.

***Slow-fast AV nodal tachycardia*** : an RJT of the AV nodal variety produced by micro-reentry in which the impulse travels down the slow pathway and up the fast pathway.

***Spontaneous depolarization*** : the ability of specialized cardiac cell to activate by altering the permeability of its membrane sufficient to attain threshold potential without any external stimulation .

***Stokes-Adams attacks*** : syncopal episodes caused by periods of cardiac arrest .

***Subaortic stenosis*** : narrowing of the outflow passage from the left ventricle proximal to the aortic valve sufficient to obstruct the flow of blood .

***Subendocardial injury*** : a term used for the deviation of the ST segments that occurs with subendocardial ischemia .

***Subendocardial ischemia(SEI)*** : deviation of the ST segments away from the ventricle (always the left ventricle ) in which only the inner part of the myocardium is ischemic .

***Supraventricular*** : any cardiac area above the branching of the common bundle and , therefore , capable of initiating a beat that could be conducted normally through the ventricles .

***Supraventricular premature beat (SVPB)*** : either an APB or a JPB .

***Sustained VT*** :VT at least 30 sec in duration or requiring an intervention to terminate .

***Syndrome X:*** is a term used to describe a commonly found constellation of metabolic derangements that include insulin resistant (with or with out DM), hypertension, dilepidemia, obesity, and endothelial dysfunction and is associated with accelerated cardiovascular disease.

***Tachyarrhythmia*** : an abnormal cardiac rhythm with a ventricular rate >100 beats/min .

***Tachycardia-bradycardia syndrome*** :both rapid and slow rhythms are present The rapid rhythms tend to appear when the rate slows abnormally while the slow rhythms are prominent immediately following the sudden cessation of a rapid rhythm .

***Third degree AV block*** : non of the atrial impulses is conducted to the ventricles . It is often referred to as complete AV block .

***Torsades de pointes*** : a variety of ventricular tachycardia resulting from prolongation of the ventricular recovery time . The term is French for "turning of the point "

***Total electrical alternans*** : alternation in the amplitudes of all of the ECG waveforms in the presence of regular cardiac cycle lengths .

***TP segment*** : time from the end of the T wave to the onset of the P wave .

***Transient Left Ventricular apical Ballooning without coronary Artery Stenosis:*** Recently a new syndrome has been described by Tsuchihashie et al that mimics acute MI. it is characterized by transient left ventricular apical ballooning without coronary stenosis. These patients present with chest discomfort and ECG changes mimicking MI, and have elevated CK. Some patients present with pulmonary odema, cardiogenic shock, or ventricular fibrillation there is no obstructive coronary artery disease and most patients have complete recovery quite rapidly.

***Trigeminy*** : a rhythm pattern in which every second sinus beat is followed by a premature beat .

***Triggered activation*** : spontaneous impulse formation produced by early after depolarizations.

***V1 negative*** : an abnormally wide QRS complex that is predominantly negative in lead V1 ; sometimes called "LBBB-like."

***V1 positive*** : an abnormally wide QRS complex that is predominantly positive in lead V1 ; sometimes called "RBBB-like."

***Vagal maneuver*****:** an intervention that increases parasympathetic activity in relation to the amount of sympathetic activity.

***Vasovagal reaction (reflex)*** : sudden slowing of the heart rate by either decreased impulse formation (sinus pause )or decreased impulse conduction (AV block) resulting from increased parasympathetic or decreased sympathetic nervous system activity . The slowing of the cardiac rhythm is accompanied by peripheral vascular dilation .

***Vasovagal syncope*** : loss of consciousness caused by a vasovagal reaction . Consciousness is almost always regained when the individual falls into a recumbent position because this results in increased venous return to the heart .

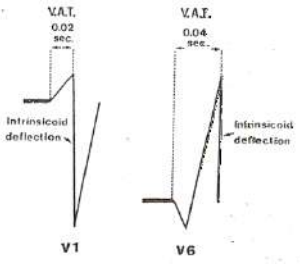
***Ventricular*** : any cardiac area beyond the branching of common bundle which is , therefore , is incapable of initiating a beat that could be conducted normally through the ventricles .

***Ventricular Activation Time*** : it is time required for ventricular depolarization .This is from the onset of QRS complex to the peak of R wave. Normally it is 0.03 to 0.05 second for left ventricle and 0.02 sec for right ventricle, and the downward deflection that follows is the intrinsicoid deflection. (see fig. below)

***Ventricular aneurysm*** : the extrem /hich the wall becomes so thin that it bulges outward (dysk

***Ventricular fibrillation*** : rapid and

ventricular activity with no discern waves .



***Ventricular flutter*** : rapid organized ventricular activity with no discernible QRS complexes or T waves.

***Ventricular flutter/fibrillation***: the spectrum of ventricular tachyarrhythmias with no discernible QRS complexes or T waves ,ranging from gross undulations to no discernible electrical activity .

***Ventricular preexcitation*** : premature activation of the ventricular myocardium via an abnormal AV pathway called a bundle of kent.

***Ventricular premature beat (VPB)*** : a QRS complexes produced by an impulse originating from the ventricles and appearing prior to the expected time of the next QRS complex generated from the sinus node or other basic underlying rhythm .

***Ventricular rhythm*** : a rhythm with a rate less than 100beats/min with abnormally wide QRS complexes . There may be either retrograde association or AV dissociation .

***Ventricular strain*** : deviation of the ST segments and T waves away from the ventricles in which there is either a severe systolic overload or marked hypertrophy .

***Vulnerable period*** : the time in the cardiac cycle prior to complete repolarization when reentrant tachyarrhythmia may be induced by the introduction of a premature impulse .

***Wenckebach sequence*** : the classic form of type $2^{nd}$ degree AV block that would be expected to occur in the absence of autonomic influences on either the SA or AV nodes .

***Wide-QRS tachycardia*** : rhythm with a rate over 100 beats/min with QRS complexes at least 0.12 sec in duration .

***WPW syndrome*** : the clinical combination of a short PR interval , an increased QRS duration caused by an initial slow deflection (delta wave) , and supraventricular tachyarrhythmias .

## References


1. **Abdulla AM. et al. Value of ventricular Complex (VPC).**
2. **Alwan SM. Clinical medicine 1st Edit: Quraish printing press. Aden, 2004.**
3. **Bennett DH. Cardiac Arrhythmia, 5th Edit. Oxford: Butterworth-Heinemann; 1997.**
4. **Boon, NA, et al. Disease of the Cardiovascular System. Davidson principles and practice of medicine 18th Edit. Edinburgh : Churchill Livingstone.**
5. **Catalano, Joseph T. Guide to ECG analysis, 2nd Edit: By Lippincot Williams & Wilkins; 2002.**
6. **Convor MB. Understanding electrocardiography, 8th Edit, St louis, Mosby, 1996.**
7. **Falk RH, Atrial fibrillation. New England Journal of medicine 344:1067-1078(2001).**
8. **Ganz LI, Freidman PL: Supraventricular tachycardia. New England Journal of Medicine 162:332 (1995).**
9. **Goldberger AL. Clinical electrocardiography: A simplified Approach, 6th Edit, St. Louis, Mosby, 1999.**
10. **Goldberger AL. Electrocardiography: A simplified Approach, 2006.**
11. **Hampton, JR. The ECG in practice, 3rd Edit. Edinburg : Churchill Livingstone (1997).**
12. **Hampton, JR. The ECG made easy. 6th Edit: Churchill Livingstone.**
13. **Goldberger, A.L. Electrocardiography. In : Braunwald, E, disease of the cardiovascular system, Harrison's principles of internal medicine, 16th Edit. (2005): Mc Graw Hill.**

14. **Houghton AR and Gray D. making sense of the ECG. 2nd Edit. Arnold, member of the Holder headline Group. London, 2003.**

15. **Khalid SA. Manual of ECG. Ain shamus University.**

16. **Kumar P, Clark M, cardiovascular disease electrocardiography in clinical medicine, 5th Edit. UK : WB Saunders; 2002; 719-721.**

17. **Mangrum JM, Dimarco JP. The evaluation and management of bradycardia. New England Journal of medicine. 342:703-709(2000).**

18. **Marriot HJ. Pearls and pitfalls in electrocardiography, 2nd Edit, Williams & wilkins; 1998.**

19. **Mirvis DM, Goldberger AL : Electrocardiography, in Zipes D, Lippy, Bonow RO, Braunwald E : Braunwald's Heart disease : atext of cardiovascular Medicine, 7th Edit, Philadelphia, WB Saunaders, 2004.**

20. **Shamroth, L. An Introduction to Electrocardiography, 7th Edit, Oxford: Black Well Scientific Publication, 1990.**

21. **Scheidt. S,Netter FH, "Basic electrocardiography" in interactive ECG. DXR Development Group. Inc. 1996.**

22. **Wagner GS. Marriot's, Practical Electrocardiography, 9th Edit. Williams & wilkins; 1995.**

**Index**

Defibrillation

Depolarization,

Electrical axis deviation,

first-degree Heart Block,

Inferior myocardial infarction

MAT (multifocal atrial tachycardia),

Mobitz

Multiform premature ventricular

Narrow-complex tachycardia,

P wave,

PAC(s) (premature atrial contraction

Pacemaker rhythm,

pacemaker,

Paroxysmal Supraventricular Tachycardia

PEA (pulseless electrical activity),

Pediatric ECG,

PJC(s) (premature junctional contraction[s]),

Posterior myocardial infarction,

PR interval,

Purkinje system,

Q wave

QRS complex

QRS interval

QT interval

Quadrigeminy,

R wave,

Repolarization

Right bundle branch block,

second-degree Heart Block,

Sinoatrial block,

Sinoatrial node

Sinus arrest (sinus pause),

Sinus arrhythmia,

Sinus bradycardia,

ST segment depression,

ST segment elevation,

T wave

third-degree Heart Block,

U wave,

Ventricular arrhythmias,

Ventricular bigeminy

Wenckebach,

Wide-complex tachycardia,

Wolff-Parkinson-White (WPW) syndrome,