t our website www.jaypeebrothers.com, for detailed information of cardiology books, visit our website www.jaypeebrothers.

AN AID TO ELECTROCARDIOGRAM

Harendra Nath Sarker



Contents

PART ONE

Section 1 Elementary Knowledge 1

- □ Definition 1
- □ ECG Paper 1
- □ Leads 1
- □ Einthoven's Triangle 2
  - – *Precordial Leads 2*
- □ Basic ECG Deflexions 3
- □ Direction of Wave 4
- □ Orientation of Leads 4
- □ Mode of Activation of Atria and Ventricles 4
- □ Genesis of Normal ECG Complex 4
  - – *Genesis of the Normal P Wave 4*
  - – *Genesis of QRS Complex 5*
- □ QT Interval 6
- □ Ventricular Activation Time 6
- □ Causes of prolonged QT_C 6
- □ Causes of QT_C Interval Shortening 7
- □ Determination of Frontal Plane Axis 7
  - – *Perpendicular Lead 7*
- □ Causes of Left Axis Deviation 8
- □ Causes of Right Axis Deviation 8

Section 2 ECG Interpretation 9

- □ Determination of Heart Rate 9
  - – *Useful Rules of Thumb 9*
- □ Rhythm 9
- □ Individual Waves 9
  - – *P Wave 9*
  - – *PR Interval 13*
  - – *QRS Complex 14*
  - – *Q Wave 14*
  - – *R Wave 15*
  - – *ST Segment 18*
  - – *T Wave 19*
  - – *Generalized Low Voltage 20*

Section 3 Arrhythmia 21

- □ Definition 21
  - – *Disturbance of Impulse Formation 21*
  - – *Disturbance of Impulse Conduction 29*
  - – *WPW Syndrome (Wolff-Parkinson-White Syndrome) 38*

Section 4 Myocardial Infarction 40

- Summary of ECG Change of Fully Evolved Acute Myocardial Infarction 40
- Sequential Changes of Acute Myocardial Infarction 40
- ECG Changes of Hyperacute Phase of Myocardial Infarction 41
- ECG Changes of Chronic Stabilized Phase of MI 41
- Localization of Myocardial Infarction 41
  - *Left Ventricular Infarction 41*
  - *Right Ventricular Infarction 45*
  - *Subendocardial Infarction 45*
  - *ECG Features of Adverse Prognosis in Acute MI 45*
  - *Chronic Stabilized Phase 46*
  - *Diagnosis of Myocardial Infarction 46*
  - *Ventricular Aneurysm 46*
- Coronary Insufficiency/Myocardial Ischemia 46
  - *Classic Form of Angina (Heberden's Angina) 48*
  - *Prinzmetal's Angina 48*
  - *Criteria of Unstable Angina Pectoris 48*
  - *Prognosis of Coronary Insufficiency 48*
- Exercise Tolerance Test (ETT) 48
  - *Clinical Parameters of Positive ETT 49*
  - *Major Adverse Prognostic Factors 49*
  - *Causes of False-positive ETT 50*
- Causes of Nonprogression of R Wave 50
- Causes of Tall R Wave in Lead V_1 50

PART TWO

Section 5 Systemic ECG 51

- Arrhythmia 51
  - *Sinus Arrhythmia 51*
  - *Sinus Bradycardia 51*
  - *Sinus Tachycardia 53*
  - *Atrial Extrasystole 53*
  - *Atrial Tachycardia (Paroxysmal/Extrasystolic) 55*
  - *Atrial Flutter 56*
  - *Atrial Fibrillation 57*
  - *Supraventricular Tachycardia (SVT) 61*
  - *The Wolff-Parkinson-White Syndrome (WPW Syndrome) 63*
  - *Lown-Ganong-Levine (LGL) Syndrome 65*
  - *Ventricular Extrasystoles/Premature Beats/Ectopic Beats 65*
  - *Ventricular Tachycardia 67*

- *Mechanism 68*
- *Ventricular Fibrillation 70*
- *Pacemakers (Artificial) 70*
- *Sinoatrial Disease (The Sick Sinus Syndrome) 72*
- *Heart Block 73*
- *Sinus Node Arrest 73*
- *Atrioventricular Block (AV Block) 74*
- *Complete Heart Block (Third Degree Heart Block) 76*
- *Bundle Branch Block 78*
- *Fascicular Block 80*
- *Stokes Adams Syndrome 81*

Section 6 Pericarditis 83

□ Acute Pericarditis 83
- *Chronic Pericarditis/Pericardial Effusion 84*
- *Chronic Constrictive Pericarditis 84*
- *Acute Myocarditis 84*

□ Systemic Hypertension 85

□ ECG in Valvular Heart Disease 86
- *Acute Rheumatic Carditis 86*
- *Mitral Stenosis 86*
- *Mitral Incompetence 86*
- *Aortic Stenosis 86*
- *Aortic Incompetence 86*
- *Atrial Septal Defect 86*
- *Ventricular Septal Defect 87*
- *Dextrocardia 87*
- *Mechanical Dextrocardia 87*
- *Hypertrophic Cardiomyopathy 87*
- *Acute Pulmonary Embolism 88*
- *Emphysema (COPD) 89*
- *ECG Evaluation of Pulmonary Function in COPD 90*
- *Digitalis Effect 90*
- *Digitalis Toxicity 90*
- *Hyperkalemia 91*
- *Hypokalemia 92*
- *Hypocalcemia 93*
- *Hypercalcemia 93*
- *Hypothermia 93*
- *Uremia 94*

Index 95

PART ONE — BASIC ECG

Section 1

Elementary knowledge

DEFINITION

ECG is the graphic tracing of electrical activities of the heart (Fig. 1.1).

It is recorded in a calibrated paper called ECG paper by attaching electrodes of the ECG machine which is called leads, in the limbs, left arm, right arm and left leg are called frontal plane leads, and anterior chest wall is called precordial leads.

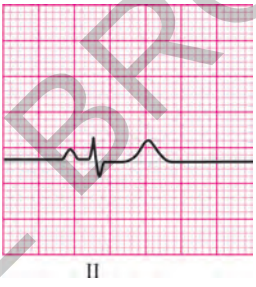


Figure 1.1: Normal ECG and ECG paper

ECG PAPER

Paper is calibrated as follows—

1. 1small sq (horizontally) → 1 mm = 0.04 sec
2. Large sq → 5 mm = 0.2 sec
   Paper speed = 25 mm/sec = 5 large sq /sec
   15 large sq = 3 sec
3. 1mV = 10 mm (10 small sq vertically).

LEADS

All electrodes placed at a distance greater than 15 cm from the heart, may be considered to be equidistant from the heart.

12 conventional leads—

(a) Frontal plane leads—6
   i. Standard leads—I, II, III

JAYPEE BROTHERS

ii. Augmented leads—aVR, aVL and aVF. (Augmented leads increase the voltage by 50%).

(b) Horizontal leads—6
Precordial leads—V_1–V_6.

EINTHOVEN'S TRIANGLE (FIG. 1.2)

The lead axes of the three standard leads form an equilateral triangle with the heart at the center.

S stands for standard bipolar limb leads—

- SI—Left arm positive, right arm negative
- SII—Left leg positive, right arm negative
- SIII—Left leg positive, left arm negative.

V stands for unipolar leads—

(a) Unipolar limb leads—aVR, aVL and aVF.
(b) Unipolar chest leads—V_1–V_6

Augmented unipolar limb leads (aVR, aVL and aVF) are oriented to or face the heart from the roots of the limbs.

- aVR—Oriented to the cavity of the heart, i.e. all deflexions are negative
- aVL—Oriented to anterolateral or superior surface of the left ventricle
- aVF—Oriented to inferior surface of the left ventricle.

Precordial Leads (Fig. 1.3)

- V_1–4th intercostal space right to sternum
- V_2–4th intercostal space left to sternum
- V_3–Midway between V_2 and V_4
- V_4–Left 5th intercostal space at midclavicular line
- V_5–Left 5th intercostal space at anterior axillary line
- V_6–Left 5th intercostal space at midaxillary line.

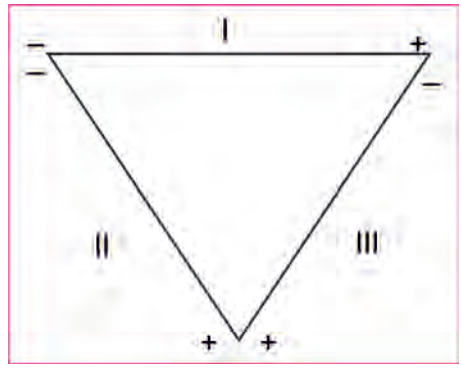


Figure 1.2: Einthoven's triangle

JAYPEE BROTHERS

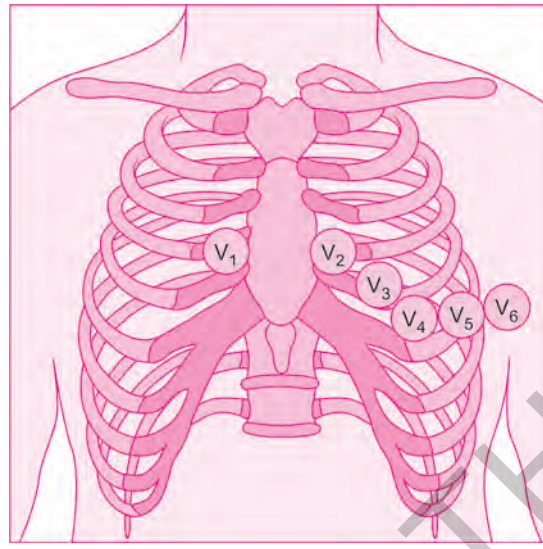


Figure 1.3: Precordial leads

BASIC ECG DEFLEXIONS (FIG. 1.4)

- P wave — Atrial activation or depolarization
- QRS wave — Ventricular activation or depolarization
- T wave — Ventricular recovery or repolarization
- U wave — Uncertain.

ST segment, T wave and U wave—Total duration of ventricular recovery (repolarization).

[ECG waves are denoted by Latin letters. If the amplitude of the wave is over 5 mm the wave is denoted by a capital letter. If the amplitude of the wave is below 5 mm a small letter is used].

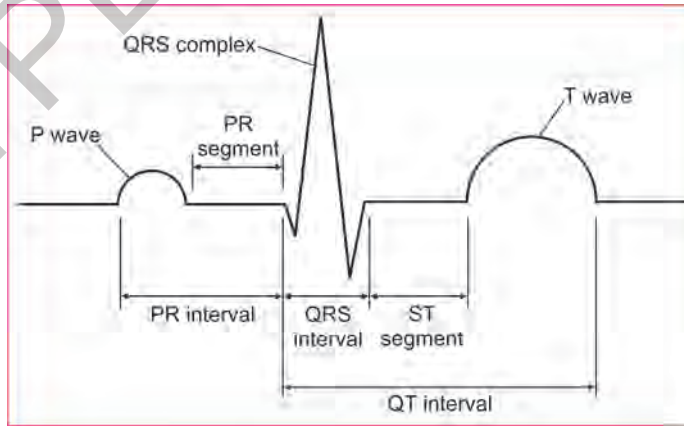


Figure 1.4: Basic ECG deflexions

JAYPEE BROTHERS

DIRECTION OF WAVE

When an electromagnetic force flows towards the positive electrode, it gives a positive wave and when it flows away from the positive electrode, it gives a negative wave.

ORIENTATION OF LEADS

1. Standard leads II, III and lead aVF–Inferior surface
2. Standard leads I and aVL–High left lateral surface
3. aVR and sometimes V_1–Cavity of the heart
4. Precordial leads.
   (a) $V_1 - V_6$—Anterior wall of the heart.
      i) $V_1 - V_4$—Anteroseptal wall of the heart
      ii) $V_5 - V_6$—Anterolateral wall or apical leads.
   (b) $V_1 - V_2$—Right ventricle.
      i) $V_3 - V_4$—Septal
      ii) $V_5 - V_6$—Left ventricle.
   Left oriented leads or lateral leads—Standard lead I, aVL, V_5 and V_6

MODE OF ACTIVATION OF ATRIA AND VENTRICLES

Activation of biatrial chamber occurs longitudinally and contiguity. For this reason, atrial hypertrophy cannot be interpreted electrocardiographically. Such interpretation can be made in terms of atrial enlargement.

Activation of ventricles occurs transversely through the ventricular myocardium and enables to detect ventricular hypertrophy electrocardiographically.

Abnormality should not be read into leads aVR or V_1 unless these leads reflect abnormal positivity.

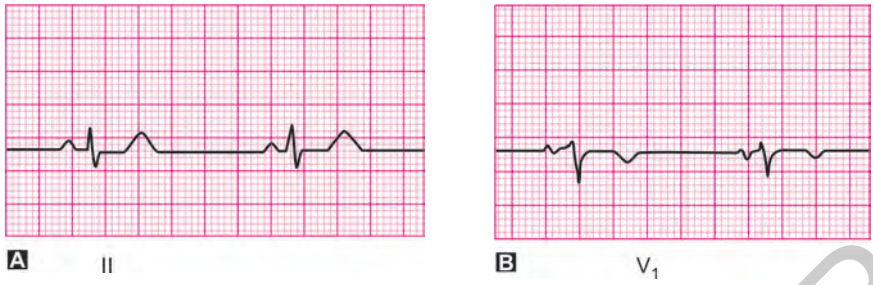
The T wave is normally inscribed in the same direction as the QRS complex.

GENESIS OF NORMAL ECG COMPLEX

Genesis of the Normal P Wave (Figs 1.5 A and B)

The sinoatrial (SA) node is the normal pacemaker of the heart and is situated in the right atrium. The SA node initiates the activation of the atrium first activating the right atrium and then the left atrium. The depolarization of the right atrium produces the ascending limb of the P wave in the frontal plane leads and the initial positive deflection in lead V_1. The left atrial depolarization begins 0.03 sec after right atrial depolarization and constitutes the descending limb of the P wave in the frontal plane leads and terminal negative deflection in V_1.

JAYPEE BROTHERS



Figures 1.5 A and B: Genesis of normal P waves

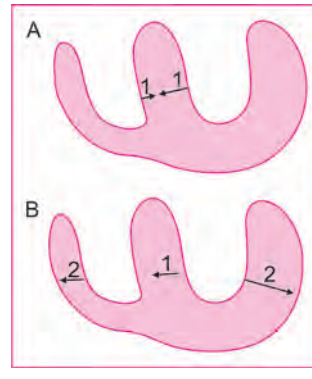
The mean frontal plane direction of atrial activation is inferiorly and to the left. As the mean direction is away from the positive electrode of aVR, P wave is always negative except in dextrocardia.

Genesis of QRS Complex

The QRS complex represents ventricular depolarization. The first negative deflexion after the P wave is termed a Q wave and the upward deflexion after the P wave is called an R wave. The ensuing deflexions are labeled in alphabetical sequence.

The activation of the ventricles begins in the left subendocardial region of the lower third of the interventricular septum, spreading transversely from left to right. It is opposed by a smaller activation force arising in the right subendocardial region of the interventricular septum, spreading transversely from right to left (Fig. 1.6 A). The resultant vector is directed from left to right through the lower third of interventricular septum giving rise to initial r wave in V_1 (rS pattern) and q wave in V_6 (qR pattern) (Fig. 1.7).

Activation of the interventricular septum is followed by activation of the free walls of both ventricles. This occurs transversely from subendocardial to the subepicardial regions of the free walls of both ventricles. The larger right to left force of the free wall of left ventricle counteracts the smaller left to right force of the free right ventricular wall (Fig. 1.6 B).



Figures 1.6 A and B: Genesis of QRS complex

JAYPEE BROTHERS

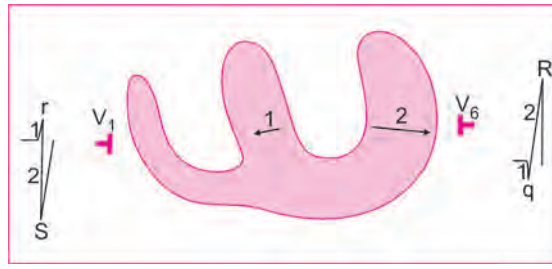


Figure 1.7: Genesis of R wave

The resultant vector is directed from right to left through the free wall of the left ventricle producing S wave in V_1 and large R wave in V_6 (Fig. 1.7).

QT INTERVAL

QT interval—The interval from the beginning of the QRS complex to the end of the T wave. It represents the total duration of ventricular electrical activity, the sum of ventricular depolarization and repolarization.

The QT interval is corrected for which it would theoretically be at a rate of 60 beats/minute. The corrected QT interval is known as QTc interval. The QTc interval is calculated by the formula—

$QTc = QT / \sqrt{(RR)}$

Normal QTc interval = 0.39 ± 0.04 sec.

i. e. 0.35–0.43 sec (9–11 small squares).

QT interval normally does not exceed 0.39 sec (10 small squares) with a normal heart rate of 60–100/min, the QT interval should not exceed half RR interval.

VENTRICULAR ACTIVATION TIME

Interval between beginning of QRS complex and apex of the R wave, normally 0.04 sec.

CAUSES OF PROLONGED QT_C (>11 SMALL SQUARES)

1. Sleep
2. Advanced or complete heart block
3. Acute myocarditis—Acute rheumatic carditis
4. Acute myocardial infarction
5. Drug effect—Quinidine, procainamide, tricyclic and tetracyclic antidepressant
6. Hypocalcemia, hypokalemia, hypomagnesemia
7. Hypothermia
8. Hypertrophic cardiomyopathy
9. The Jervell-Lange-Nielsen syndrome
10. Romano-Ward syndrome.

JAYPEE BROTHERS

CAUSES OF QT_C INTERVAL SHORTENING

1. Digitalis effects
2. Hypercalcemia
3. Hyperthermia
4. Vagal stimulation.

DETERMINATION OF FRONTAL PLANE AXIS

A) 1. Determine the lead in which QRS deflexion is most equiphasic
   2. QRS axis will be on perpendicular lead.

Perpendicular Lead (Figs 1.8 A to C)

(a) SI is perpendicular to aVF

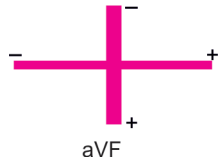


Figure 1.8 A: Perpendicular leads SI and aVF

(b) SII is perpendicular to aVL

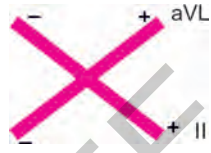


Figure 1.8 B: Perpendicular leads SII and aVL

(c) SIII is perpendicular to aVR



Figure 1.8 C: Perpendicular leads SIII and aVR

B) Axis is on the lead in which there is tallest R and smallest S.

Normal QRS axis in adult	→	0°– 90°
Right axis deviation	→	+ 90° – + 180°
Left axis deviation	→	Slight 0° – – 30°
		Marked—30° – – 90°
QRS axis in infancy (from birth to 3 months)	=	+ 90° – + 140°

JAYPEE BROTHERS

QRS axis in children = + 90° – + 120°
(From 3 months to 12/16 years).

CAUSES OF LEFT AXIS DEVIATION

1. Left anterior hemi block—Commonest cause
2. Inferior wall myocardial infarction
3. Pacing from the apex of left or right ventricle
4. Ventricular tachycardia from a focus in the apex of the left ventricle
5. Emphysema
6. Wolff-Parkinson-White syndrome
7. ASD (Primum type).

** Obesity and pregnancy do not cause left axis deviation.

CAUSES OF RIGHT AXIS DEVIATION

1. Right ventricular dominance
2. Anterolateral myocardial infarction
3. Wolff-Parkinson-White syndrome
4. Left posterior hemi block.
5. ASD (Secundum type).

JAYPEE BROTHERS

Section 2

ECG interpretation

DETERMINATION OF HEART RATE

If rhythm is regular

$$\text{Heart rate} = \frac{1500}{\text{Number of small squares between R–R}}$$

Or

$$\text{Heart rate} = \frac{300}{\text{Number of large squares between R–R}}$$

If rhythm is irregular
Heart rate = Number of R wave in 15 large squares × 20

Useful Rules of Thumb

1. If RR interval is less than 3 large squares, the rate is over 100/min, i.e. sinus tachycardia
2. If RR interval is more than 5 large squares, the rate is less than 60/min, i.e. sinus bradycardia.

RHYTHM

Sinus rhythm—P wave is followed by QRS complex, PP interval is constant and PR interval is fixed.

INDIVIDUAL WAVES

P Wave

Lead—P wave is seen in standard lead II (Fig. 2.1) and V_1 (Fig. 2.2) as the amplitude of P wave is generally the greatest in SII.

- Duration → 0.08 – 0. 10 sec (2 to $2^1/_2$ small squares)
  Maximum 0.11 sec ($2^1/_2$ small squares)

JAYPEE BROTHERS

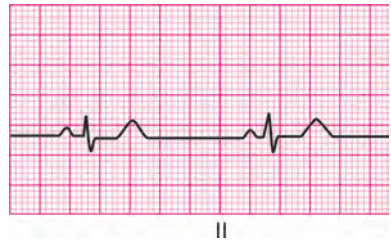


Figure 2.1: Normal P wave in SII

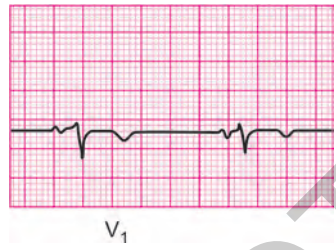


Figure 2.2: Normal P wave in V_1

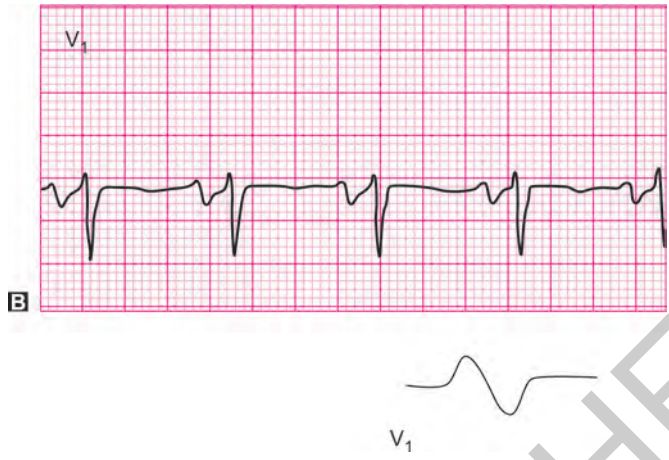
- Amplitude → 2 mm (2 small squares)
  Maximum –2.5 mm ($2\frac{1}{2}$ small squares)
  < 3 mm in bipolar lead
  < 2.5 mm in unipolar lead
- Normal P wave axis → + 45° – + 65°
  Upright, inverted or biphasic in lead III, aVL and V_1.

Left atrial enlargement or P mitrale (Figs 2.3 A and B)

1. In standard lead II or lead I or even lead aVL
   (i) The P wave shows double peaked notched or camel humped P wave
   (ii) An increased duration of the P wave to longer than 0.11 sec (>$2\frac{1}{2}$ Small squares).
   The duration of the notch → The distance between the camel humps is longer than 0.04 sec (>1 small squares).



JAYPEE BROTHERS



Figures 2.3 A and B: Left atrial enlargement or P mitrale

2. In lead V_1 P wave shows a relatively deep delayed and widened terminal negative component.

- Cause of left atrial enlargement → Mitral stenosis.

Right atrial enlargement or P pulmonale (Fig. 2.4)

1. Abnormalities of the P wave.
   (a) A tall peaked P wave in standard lead II
   amplitude > 3 mm (>3 small squares)
   Duration—Within normal limits.
   (b) The initial deflexion of P wave in V_1 lead becomes taller, more pointed with relatively small negative component or no negative component.
   (c) Rapid inscription of the negative component to the P wave in lead V_1.

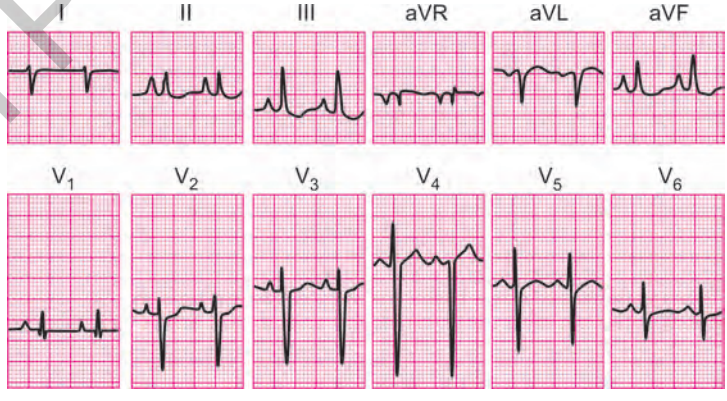


Figure 2.4: Tall peaked P wave. Right atrial enlargement or P pulmonale

JAYPEE BROTHERS

In right atrial enlargement the downslope of P wave <0.03 sec (Fig. 2.5 A).

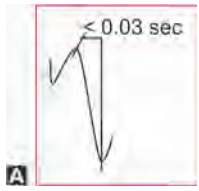


Figure 2.5 A: Downslope of P wave in right atrial enlargement

In left atrial enlargement the downslope of the P wave >0.03 sec (Fig. 2.5 B).

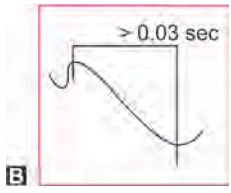


Figure 2.5 B: Downslope of P wave in left atrial enlargement

P pulmonale occurs in acquired heart disease such as cor pulmonale due to COPD. When the tall peaked P wave of right atrial enlargement is associated with right P wave axis deviation in acquired heart disease it is known as P pulmonale.

2. Abnormalities of QRS complex which reflect right atrial enlargement.
   (a) A qR complex in lead V_1.
   (b) Diminution in the size of QRS complex deflexion in lead V_1 with marked increase of QRS amplitude in lead V_2.

Combined right and left atrial enlargement

1. In frontal plane leads and left lateral leads (Precordial) ($V_5 - V_6$)—P wave is wide, notched and of increased amplitude.
2. In lead V_1.
   Initial component of P wave is tall and peaked and terminal component is deep wide and delayed.

Causes of combined left and right atrial enlargement

1. Mitral stenosis associated with marked pulmonary hypertension
2. MS with tricuspid incompetence
3. MS with tricuspid stenosis
4. ASD
5. Lutembacher's syndrome ASD with acquired MS.

P´ WAVE OF RETROGRADE ATRIAL ACTIVATION (AV JUNCTIONAL RHYTHM)

1. Negative p´ wave in standard lead II, III and aVF.
2. Positive p´ wave in aVR.
3. In lead V_1, p wave is a tall, totally positive, narrow and peaked deflexion.

JAYPEE BROTHERS

For detailed information of cardiology books, visit our website www.jaypeebrothers.com, for detailed information of cardiology

AN AID TO ELECTROCARDIOGRAM

Salient Features

- Contains elementary knowledge about electrocardiogram (ECG)
- Includes special knowledge about ECG changes in different diseases
- Contains 123 clear-cut ECG tracings which are special features of this book
- Also contains causes, clinical features and management of the conditions, which have specific ECG changes
- Overall, it is written in simple language.



Harendra Nath Sarker MBBS FCPS (Medicine) MRCP (UK) is an Associate Professor of Medicine at Sher-e-Bangla Medical College, Barisal, Bangladesh. He obtained MBBS in 1984, FCPS in Internal Medicine in 2004 from Bangladesh College of Physicians and Surgeons of Bangladesh and MRCP in 2011 from the Royal College of Physicians, UK. He has been serving as a teacher since 1995 in different medical colleges as Registrar, Assistant Professor and Associate Professor. He is an examiner of undergraduate examination. He is the author of five medical books. He has more than 30 publications in national and international journals.

Shelving Recommendation
CARDIOLOGY

ISBN 978-93-5152-069-6
9 789351 520696

Available at all medical book stores
or buy directly from Jaypee Brothers through online shopping
at www.jaypeebrothers.com

JAYPEE BROTHERS
Medical Publishers (P) Ltd.
www.jaypeebrothers.com