



Shortcut to
ECG

FIRST EDITION



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*Shortcut to
Electrocardiography*



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Shortcut to Electrocardiography

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As the name suggests this book provides you only a shortcut to interpret an ECG. This book in any way does not replace the need of standard textbooks.

After going through this book, you may access some sample 12-lead ECGs at www.themedicalpost.com/ecg

This book is dedicated to our parents.

Shortcut to Electrocardiography

Preface

In the preface of our first book we would like to share that why we had chosen this topic “Electrocardiogram”.

As doctors we all know the ultimate need for understanding an ECG. Its importance is known to everyone as it is the simplest source of diagnosing a large number of diseases and condition not only related to the heart but other systems too.

As titled the book provides with a shortcut that you can rely on for understanding and describing an electrocardiogram.

This book cannot change the need of standard textbooks but it will provide you with all the specific points to learn and remember, helping you to interpret the 12 lead ECG within seconds.

One important feature of this book is the Treatment box. With every disease or a condition, specific treatment regimens and protocols are described in brief. In this way this book will not only help you to diagnose conditions easily but will also help you to remember important drugs and doses which may even be life saving in many cases.

I hope our first project will make a good impression on everyone and will benefit medical students specially the interns to the maximum.

We would like to thank **Manbir Singh** for guiding us and to make this project a success.

We would also like to thank **Ram Kaji Baniya** and **Shankar Baral** for their help.

GURKEERAT SINGH
JASKEERAT SINGH
RAVI KUMAR SHAH

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Chapter 1. Important basics you need to know.

Let us first let be familiar with common terminologies. Electrocardiography is the interpretation of the electrical activity of the heart over a period of time, as detected by electrodes attached to the outer surface of the skin and recorded by a device external to the body. The recording produced by this procedure is termed as Electrocardiogram.

There are 3 types of cells in the heart.

1. *Pacemaker cells* – These cells are responsible for the cardiac rhythm. They can further be grouped in atrial, junctional and the cells in the ventricles. Remember those pacemaker cells that are dominant takes over the rhythm. Normally the dominant cells are located high up in the right atrium. These groups of cells are called as the Sino Atrial (SA) node. In normal conditions it fires at a rate of 60-100 times a minute.
2. *Electrical conducting cells* – They are like wires of an electrical circuit. They carry current rapidly and efficiently to distant regions of the heart.
3. *Myocardial Cells* – These are responsible for heavy labour of contracting and relaxing of the heart. Composed of abundant contractile proteins – actin and myosin. When the wave of depolarization reaches the myocardial cells Calcium is released from the cells causing it to contract. This is the famous excitation contraction coupling.

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The conduction pathway in brief –

SA NODE → ATRIAL CONDUCTION SYSTEM → AV
NODE → BUNDLE OF HIS → DIVISION INTO –

- Right bundle branch
- Left bundle branch → Septal fascicle, Left anterior fascicle and Left posterior fascicle.



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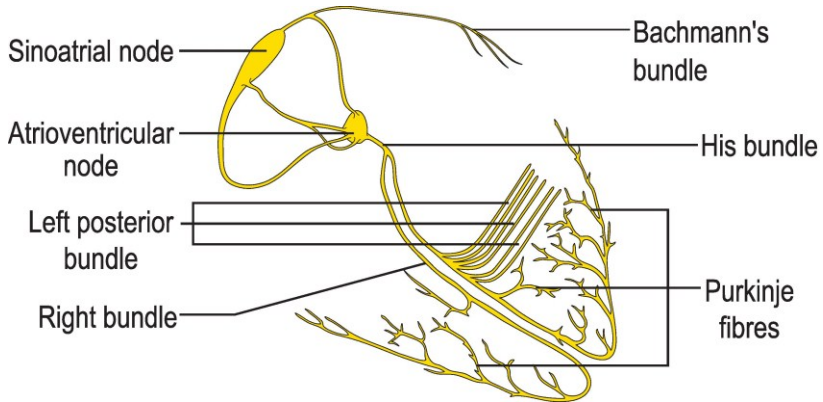


Fig 1.1 – Electrical conduction of the heart. [Source – Wikipedia. Author – Madhero88].

Note that all the lines/waves we see on the ECG are produced as a result of the function of the myocardial cells. Rests of the cells do not generate sufficient voltage to be recorded by the surface electrodes.

History

Willem Einthoven used his invention “String galvanometer” and assigned the letter P, Q, R, S and T to the various deflections and described the electrocardiographic features of a number of cardiovascular disorders. In 1924, he was awarded the Nobel prize in medicine for his discovery.

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ECG Paper

Let us understand the ECG paper now –

- Light lines describe small squares each of 1 x 1 mm size.
- Dark lines describe large squares each of 5 x 5 mm size.
- X axis denotes time – 1 small square = 0.04 seconds.
- Y axis denotes the amplitude of the wave produced – 1 small square = 0.1 mV.

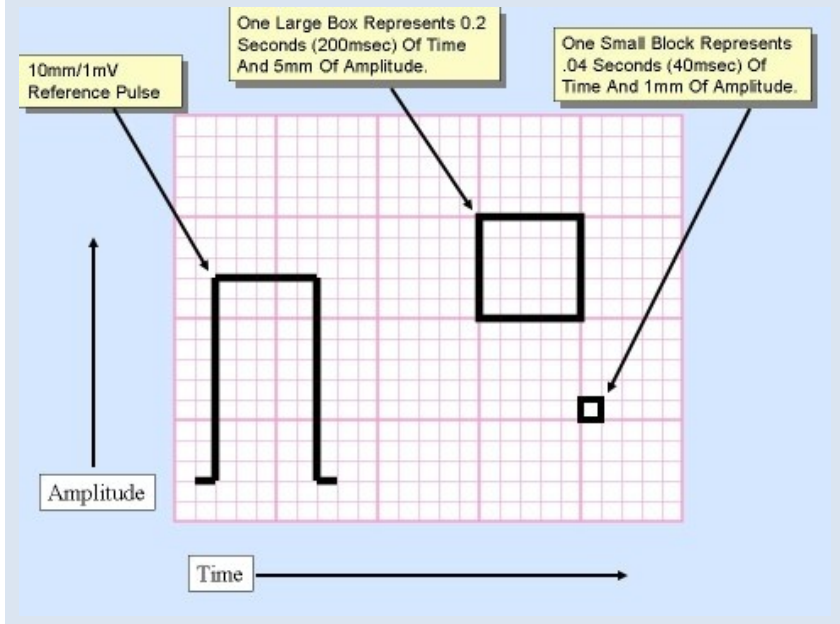
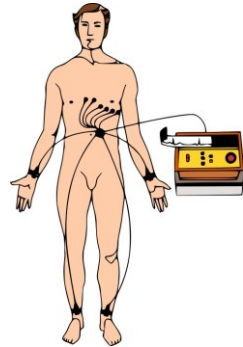


Fig 1.2 – Dimensions of the ECG paper.

Chapter 2. ECG leads.

There are two types of leads – Limb leads and precordial (chest) leads.

The reason we use these two leads is that the heart is a 3 dimensional structure, thus a single lead will not be able to describe its electrical activity properly. Hence the standard ECG consists of 12 leads. Each leads views the heart at a unique angle enhancing its sensitivity to a particular region of the heart.



Limb Leads

These leads view the heart in the vertical plane i.e. records the electrical forces (depolarization and repolarization) moving up, down, left and right.

To produce the 6 limb leads with 4 actual leads. Each of the electrodes is variably designed as positive and negative. This is done automatically by the machine.

Various limb leads again further grouped into Standard limb leads and Augmented limb leads.

The standard limb leads are,

Lead 1 – (0 degree): Left arm is made +ve and Right arm -ve.

Lead 2 – (60 degrees): Legs +ve and Right arm -ve.

Lead 3 – (120 degrees): Legs +ve and Left arm -ve.

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The augmented limb leads are,

AVR – (-50 degrees): Right arm is +ve and other limbs are –ve.

AVL – (-30 degrees): Left arm +ve and other limbs are –ve.

AVF – (90 degrees): Legs are +ve and other limbs are –ve.

Note these augmented are so named as they amplify the tracings to get an adequate recording.

Precordial leads

The precordial leads view the electrical forces moving anteriorly and posteriorly.

These are,

V1: Placed in the 4th intercostal space right to the sternum.

V2: Placed in the 4th intercostal space left to the sternum.

V3: Placed between leads V2 and V4.

V4: Placed in the 5th intercostal space in the mid clavicular line.

V5: Placed between the leads V4 and V6.

V6: Placed in the 5th intercostal space in the mid axillary line.

Leads specifying different region of the heart

Inferior leads: Leads II, III and AVF. They view the inferior surface of the heart more effectively.

Left lateral leads: Leads I, AVL, V5 and V6. They best view the left lateral wall of the wall.

Anterior leads: V1, V2, V3 and V4.

Note: The right ventricle lies anteriorly and medially and the left ventricle lies posteriorly and laterally. Thus;

V1 and V2 – lies directly over the right ventricle.

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V3 and V4 – lies over the interventricular septum.

V5 and V6 – lies over the left ventricle.

Remembering the region specific leads is of great importance in Ischemic heart diseases.

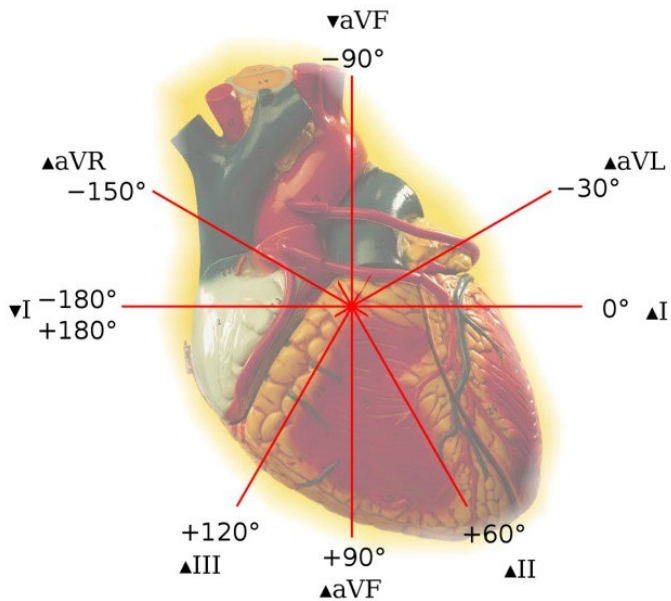


Fig 2.1 – Figure showing angle of orientation of all the limb and chest leads.

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Chapter 3. Naming the lines.

Before we can start naming the lines it is important to understand them first.

A segment is a line connecting 2 waves.

An interval is composed of at least one wave plus a connecting straight line.

We'll be discussing these waves/segments as the cardiac cycle continues.

P wave

As the SA node fires (an event which is not seen on the ECG) the wave of depolarization spreads outward into the atrial myocardium which results in depolarization of the atrial myocardial cells producing atrial contraction. This forms the p wave on the ECG.

Hence p wave is the recording of the spread of wave of depolarization. Now as the SA node is located in the right atrium thus the right atrium will be depolarized first and so the first half of the p wave is formed by the right atrial depolarization and the remaining half by the left atrium.

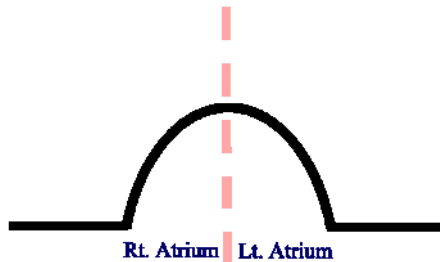


Fig 3.1 – Figure showing the two components of the p wave.

Now the wave of depolarization is prevented from communicating the ventricles by the valves. Thus the electrical conduction funnels its way along the interventricular septum.

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Here a structure known as the AV node slows down the conduction to a crawl but this pause only lasts for a few fractions of seconds.

Why is this pause necessary? It's really important as it allows the atria to contract completely and allows them to empty their whole content before the ventricles start to contract.

The wave of depolarization now enters the ventricular conduction systems. The wave first enters the bundle of His which then divides into right and left supplying the respective ventricles. The left bundle of His further divides into 3 fascicles – Left anterior f., Left posterior f. and the septal fascicle.

The wave then passes to the ventricles causing depolarization. This is marked by the QRS complex on the ECG.

Note that the amplitude of the QRS complex is much more than the P wave as because of its obvious size.

Components of the QRS complex are,

Q wave: First downward/negative deflection.

R wave: First upward/positive deflection.

S wave: Second downward/negative deflection.

R'/R prime wave: It is the second upward/positive deflection.

After the ventricular depolarization now the myocardial cells go to a refractory period which is then followed by repolarization. This wave of repolarization is marked on the ECG paper as the T wave.

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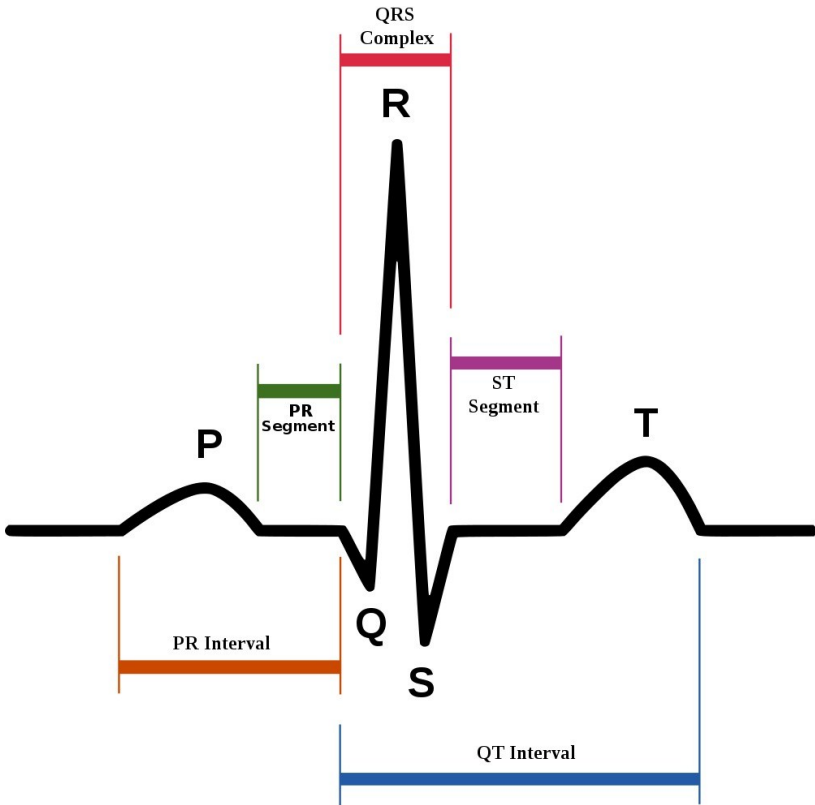


Fig 3.2- Figure showing segments and intervals.

To summarize

PR interval: From the p wave to the QRS interval, it means the time starting from the atrial depolarization to the start of ventricular depolarization.

ST segment: Line connecting the end of QRS complex to the beginning of the T wave, it means the time starting from the end of ventricular depolarization to the start of ventricular repolarization.

QT interval: Comprises of QRS complex + ST segment and the

T wave, it means the time starting from the start of ventricular depolarization till the end of ventricular repolarization.

By seeing an ECG, till a long time you must be thinking that why there are positive deflections in some leads but negative in the other leads?

The reason to this is that a wave of depolarization moving towards an electrode produces a positive deflection of the ECG paper and similarly the wave of depolarization which is moving away from the positive deflection produces a negative deflection on the ECG paper.

It is also important to understand one more concept, If the electrode is placed in the middle of a cell, initially as the wave front approaches the electrode, the ECG records a positive deflection but when the wave reaches the electrode the positive and negative charges are balanced and the ECG recording returns to the baseline, further as it recedes a negative deflection is inscribed.

Note that a perpendicularly placed lead will produce a biphasic wave on the ECG.

Hence this concept can easily be applied to the entire heart; the electrodes placed on the surface of the body will record waves of depolarization and repolarization as they sweep through the heart.

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Chapter 4. Relationship of different waves, segments and intervals in different leads.

P wave

As we know the right atrium depolarizes first, then the left atrium depolarizes. Thus the average current flow for the atrium points from right to left and slightly inferiorly.

Thus the left lateral leads – V5, V6, I, AVL and the inferior leads- II, III and AVF will show a positive deflection.

Lead III and V1 is actually lying perpendicular the atrium current. Thus often records a biphasic p wave.

Lead AVR in most of us sees the current as moving away from it, so we expect these leads to show a negative deflection.

The p wave is normally less than 2.5mm in amplitude.

PR interval

This interval includes the delay in conduction that occurs at the AV node.

Normally it ranges from 0.12 to 0.2 seconds i.e. 3-5 mm on the ECG paper.

QRS complex

As the left ventricle is much larger and thicker therefore the current flow swings leftwards.

Thus large positive deflections are seen in many of the left lateral and the inferior leads.

Lead AVR lying right wards records a deep negative deflection.

Leads V1 and V2 records S waves negative as the current moves away from them.

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Leads V5 and V6 records tall R (positive) waves.

Leads V3 and V4 represents a transition zone and one of them records a biphasic wave i.e. R wave and a S wave of equal or near equal amplitude.

T wave

The T wave is highly susceptible to all kinds of influences both cardiac and non cardiac. Hence it is variable in nature. It is usually positive in leads with tall R waves.

QT interval

The duration of the QT interval is proportionate to the heart rate. The faster the heart beats the faster it must repolarize to prepare the heart for the next contraction and vice versa.

In general, the QT interval comprises about 40% of the cardiac cycle as measured from once R wave to the next.

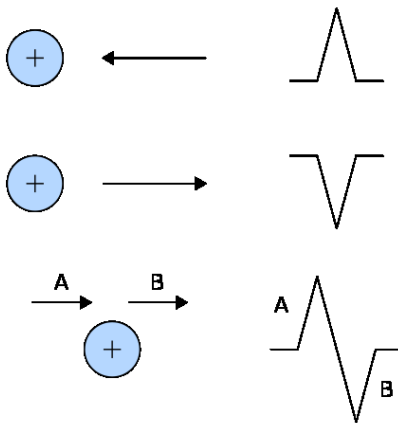


Fig 4.1 – Figure showing the relationship between positive electrodes, depolarization wave fronts (or mean electrical vectors), and complexes displayed on the ECG.

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Chapter 5. Axis.

Axis determination is a very important clue which helps us to get an idea that where the problem lies approximately. But do note that this funda cannot be applied in all the cases for eg in Hemi Blocks.

Axis is the direction of the mean vector or the mean electrical axis.

Normally the axis lies between -30° to 100° .

For quickly determining the QRS axis we look at the leads I and AVF or I and III.

In left axis deviation: deflections moves away from each other (remember in LAD the deflections have left each other).

In right axis deviation: deflections moves towards each other (remember in RAD the deflections are kissing each other and are doing the right thing).

Axis	Lead I	Lead AVF/III	Angle $^\circ$
Normal axis	+ve	+ve	-30 to +100
Left axis deviation	+ve	-ve	-30 to -90
Right axis deviation	-ve	+ve	+100 to +180
Extreme right axis	-ve	-ve	+180 to -90

For defining the axis very precisely –

Step 1: Find out the biphasic wave in any of the leads.

Step 2: The lead which shows the biphasic wave must be lying perpendicular to the actual axis.

Step 3: Check whether it's left or right axis.

For calculating the actual axis the lead-axis-degree diagram should be remembered thoroughly.

Conditions associated with axis deviations –

LAD – Seen in normal variants, left ventricular hypertrophy, anterior fascicular block, inferior wall infarction.

RAD – Normal variants especially in young and children, right ventricular hypertrophy, lateral wall infarction, dextrocardia, left pneumothorax.

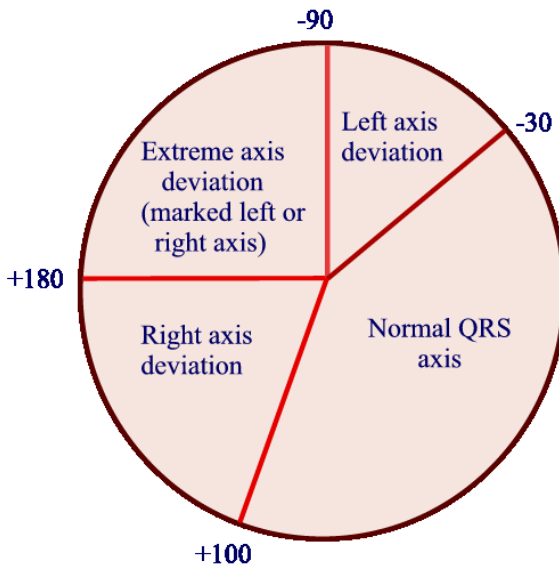


Fig 5.1- Figure showing axis deviations according to the angle of orientation.

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Chapter 6. Hypertrophy and Enlargement.

Let us first understand these terminologies.

Hypertrophy refers to the increase muscle mass. Caused by the pressure overload in which the heart is forced to pump blood against an increased resistance. E.g., in long term hypertension and aortic stenosis.

Enlargement refers to dilatation of a particular chamber. Caused by volume overload. The chamber dilates to accommodate an increased amount of blood.

These two conditions commonly co exists.

Also why is that axis deviation and hypertrophy related to each other?

In long standing cases of sustained hypertension the left ventricle is forced to do more work and thus it hypertrophies. Thus its electrical dominance over the right ventricle therefore becomes more profound. Hence in the axis is further drawn leftward and results in left axis deviation.

Due to enlargement or hypertrophy 3 things can happen –

- The can increase in duration.
- The wave can increase in amplitude.
- There can be axis deviation.

Important points to remember –

- The amplitude of p wave should not exceed 2.5 mm.
- For right atrial enlargement – P waves with an amplitude exceeding 2.5 mm in the inferior leads. No change in the duration of the p wave.

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Possible right axis deviation. Also called as p pulmonale.
Common cause – severe lung disease.

- For left atrial enlargement – The amplitude in the terminal portion of the p wave may be increased and must descent at least 1 mm below the iso-electric line (especially in the lead V1). Duration of the p wave may be increased. No significant axis deviation. Also called as p mitrale. Causes – any mitral valve disease.

Ventricular hypertrophy

Normally the R wave's amplitude enlarges as we proceed from the lead V1 and V5. But in case of right ventricular hypertrophy the R wave amplitude increases i.e. large in lead V1 which lies over the hypertrophied right ventricle and small R wave in lead V6 which overlies the normal left ventricle. Similarly the S wave in the lead V1 is small whereas S wave in V6 is large.

<i>Causes</i>	
Right ventricular hypertrophy	Left ventricular hypertrophy
Pulmonary disease and congenital heart disease.	Systemic hypertension and valvular heart disease.

To summarize the criteria –

- RVH: Right axis deviation and R wave is larger than S wave in V1 where as S wave is larger than R wave in V6.
- LVH: R waves in V5 or V6 plus S waves in V1 or V2 should exceed 35 mm.
R wave in AVL exceed 13 mm.

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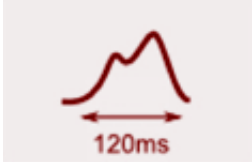
Interesting fact: ECG changes of left ventricular hypertrophy are present only in 50 % of the cases whose ECHO demonstrates a thickened left ventricle. Thus the sensitivity is low. But if the ECG pattern is present then around 90 % of the patient will show hypertrophy in ECHO.

Associated secondary repolarization abnormalities of ventricular hypertrophy–

- Down sloping ST segment depression.
- T wave inversion.

Note that these changes are more evident in those leads with tall R waves and presence of these abnormalities indicates long term disorders.

Repolarization abnormalities usually accompany severe hypertrophy.

	<p>Fig 6.1- Figure showing p wave > 2.5mm; P pulmonale.</p>
	<p>Fig 6.2- Figure showing p wave duration i.e. it's too wide; P mitrale. Note- the characteristic notch is seen.</p>

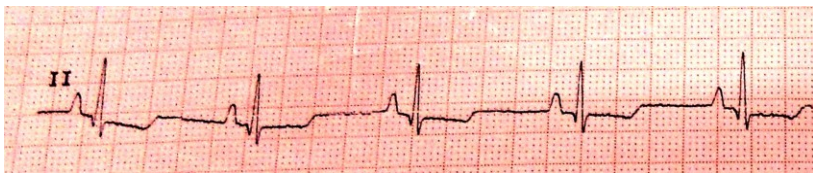


Fig 6.3 – ECG showing right atrial enlargement in a patient of chronic pulmonary hypertension.

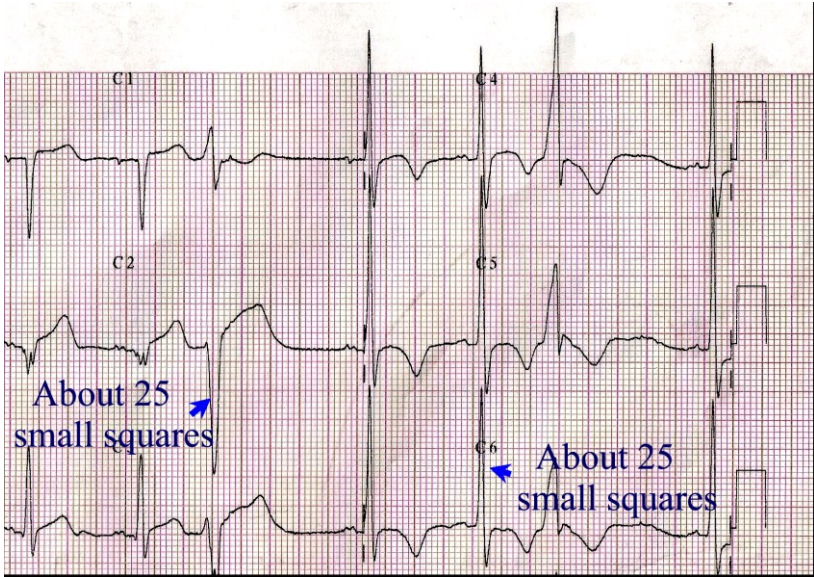


Fig 6.2 – ECG showing extreme left ventricular hypertrophy. Note the marked S waves in C2 and R waves in C6. Adding C2 and C6 = 50 small boxes approximately; which fulfills the criteria for left ventricular hypertrophy.

Arrhythmias are one of the most important subjects to be studied, as prompt measures when taken can be life saving in most of the individuals.

How can we suspect an arrhythmia clinically?

The patient may present in the ER or in the OPD with symptoms of palpitations, symptoms due to decreased cardiac output like light headedness and syncope, chest pain or sometimes it may cause sudden death.

To understand an arrhythmia we should first know how to calculate the heart rate.

Calculating Heart Rate with an ECG-

For a quick estimate calculate the number of large squares between two R waves and divide 300 by it.

For more accuracy, Heart rate = 1500 divided by the number of small squares between the two R waves.

However this method is not accurate when the rhythm is irregular or irregularly irregular. In such conditions count the number of R waves in 6 seconds (i.e. 3 large boxes) and multiplied it by 10.

Escape beats

If sinus arrest in an individual is followed by a normal beat then the later beat is the escape beat. Junctional non sinus pacemakers are the most common for providing one or continual series of escape beats.

Characteristics of these escape beats:

They escape the atrial depolarization hence no p waves are seen.

Retrograde p waves may be seen which are due to the depolarization moving backward from the AV node.

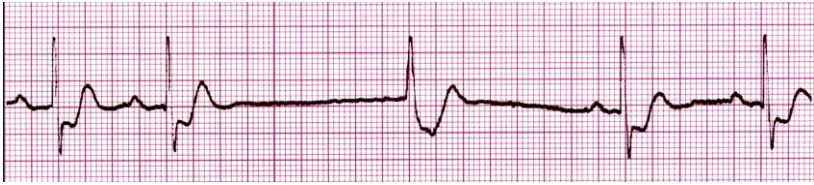


Fig 7.1- ECG showing escape beats.

Treatment Box

Treatment depends upon the cause –

- No pharmacological therapy is required for asymptomatic individuals.
- Individuals with high degrees of heart blocks, symptomatic sick sinus syndrome; pacemakers may be required.
- If due to digitalis toxicity; atropine and digoxin immune Fab may be required.

Arrhythmias can be classified as:

1. Arrhythmias of sinus origin.
2. Ectopic rhythms.
3. Conduction blocks.
4. Pre excitation syndromes.

Arrhythmias of sinus origin

Sinus tachycardia is said to occur if the rhythm is regular and the heart rate is more than 100 beats per minute. Common causes are Pain, anxiety, fever, hyperthyroidism, congestive cardiac failure, severe lung disease, anemia, pheochromocytoma, etc.

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Fig 7.2 – ECG showing sinus tachycardia.

Treatment Box

Physiological sinus tachycardia requires no treatment.

For inappropriate sinus tachycardia:

Beta-blockers such as propranolol or metoprolol are effective in slowing down the rhythm.

Sinus node ablation.

Sinus bradycardia is said to occur if the rhythm is regular and the heart rate is less than 60 beats per minute. Common causes are healthy athletes, sleep, myocardial infarction and any condition which enhances the vagal tone there by decreasing the heart rate. Other important causes one should rule out are sick-sinus syndrome, hypothermia, hypothyroidism, cholestatic jaundice, raised intracranial pressure and drugs like beta blockers, digoxin and verapamil.



Fig 7.3 – ECG showing sinus bradycardia.

Treatment Box

- Intravenous access, supplemental oxygen and cardiac monitoring are must.
- Intravenous atropine can be given in symptomatic patients. Dose – 0.6 -1.2 mg iv stat and may be repeated again.
- Sinus bradycardia which is due to drugs like digoxin, calcium

channel blockers, beta blockers, etc; simple discontinuation of the drug with observation is enough.

- Implantation of pacemaker may be required in refractory cases.

Ectopics

These are abnormal rhythms that arise from elsewhere than the sinus node. These are different from escape beats as these are sustained rhythms not just one or few beats.

Pathology behind these ectopics is either due to enhanced automaticity or/and re entry phenomenon.

Important points for identifying ectopics:

1. Check for normal p waves- absence of the p waves indicates that the ectopics are arising below the atria i.e. AV node/ ventricles.
2. Check the QRS complexes – A narrow QRS complex indicates that the origin of the rhythm must be at or above the AV node. A wide QRS complex implies that depolarization is initiated within the ventricular myocardium not through the normal conduction system. Therefore it spreads much more slowly.
3. Relationship of the p waves and QRS complexes. When both these are correlated in the usual one to one fashion i.e. one p wave followed by one QRS complex it indicates the rhythm is almost atrial in origin. But when there is no correlation between them then it indicates there is AV dissociation.
4. Is the rhythm regular or irregular.

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Atrial pre mature beats

Supra ventricular ectopics are those which arise from the atria or the AV node.

Atrial premature beats are a common phenomenon and doesn't indicate any cardiac problem or a disease.

APB can be distinguished from a normal sinus beat by the contour of the p wave (it is quite different from the other p waves) and the timing of the beat (it comes too early).

In junctional premature beats no visible p waves or retrograde p waves are seen.

Please note a compensatory pause may be seen as the premature impulse requires some extra time to travel through the atrial muscle before it discharges from the SA node.



Fig 7.4 – ECG showing Atrial premature beats.

Treatment Box

- Often no treatment is required, reassure the patient.
- In symptomatic patient, beta blockers like atenolol, metoprolol are useful.
- In very severe or frequent PACs, ablation can be done.

Paroxysmal supraventricular tachycardia

It is quite common, sudden in onset, usually preceded by a supra ventricular tachycardia and is known to terminate abruptly.

Various causes are,

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Seen in normal hearts too, sudden excitement, Reentrant circuit looping within the AV node (evident by the retrograde p waves).

Identifying features are,

- No/retrograde p waves.
- Tachycardia.
- Narrow QRS complexes.
- Continuous waves.

Role of carotid message is a very commonly used non pharmacological method for relieving PSVT.

Mechanism:

Baroreceptors are present at the bifurcation of the common carotid artery. Normally these baroreceptors senses the change in blood pressure and stimulates the vagus nerve when the blood pressure rises resulting in its decrease. While applying carotid message we try to stimulate the baroreceptors which eventually leads to stimulation of the vagus nerve thereby causing parasympathetic action which causes the following –

- Interrupts the re-entrant circuit thereby terminating the arrhythmia.
- Or at least slows the arrhythmia so that the presence or absence of the p waves can be recognized and the type of arrhythmia can be diagnosed.

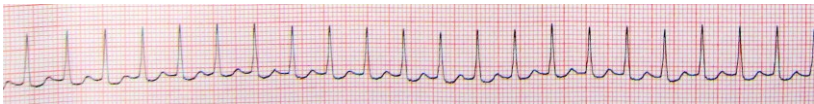


Fig 7.5 – ECG showing PSVT. [Source – Wikipedia, Author – James Heilman, MD].

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Treatment Box

- Try vagal maneuvers like carotid massage and valsalva maneuvers.
- Drug therapy:
 - Adenosine – First choice of drug in PSVT. Dose: 3mg iv stat. Second dose of 6mg and a third dose of 12mg can be given at 2 minutes interval till the arrhythmia is terminated. Note that half-life of adenosine is only 8-10 seconds; So it acts very fast.
 - Verapamil is the second drug of choice. Dose: 2.5-10mg is given over 5-10 minutes. Followed by the oral dose of 40-120mg/day.
 - Other drugs like beta blockers can also be used.
- Other modalities –
 - Overdrive pacing.
 - Radiofrequency ablation.

Atrial flutter

It is not commonly seen. Often associated with a cardiac pathology.

ECG shows classical saw tooth pattern, p waves appears at a rate of 250 – 350 beats per minute.

As the AV node cannot handle atrial impulses at this rate. Thus atrial flutter is commonly associated with a conduction block i.e. after 2 p waves 1 QRS complex is seen.

In a case of atrial flutter, carotid massage increased the degree of block i.e. from 2:1 to 4 or 5:1 and the arrhythmia will not terminate as seen in PSVT. This is because the arrhythmia is arising from above the AV node.

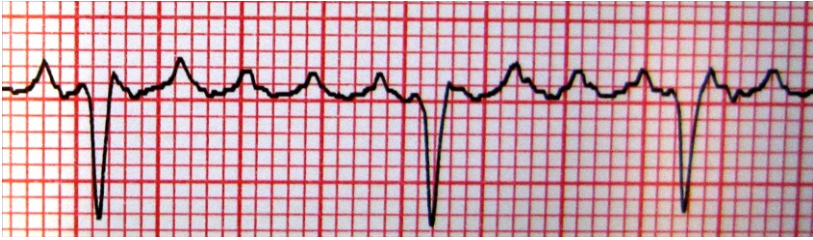


Fig 7.6 – ECG showing atrial flutter. [Source – Wikipedia, Author – James Heilman, MD].

Treatment Box

- Drug therapy-
Digoxin, beta blockers, verapamil are used to control the ventricular rate.
Amiodaronem propatenon or flecamide may be effective.
- In resistant cases DC cardioversion may be tried.
- Catheter ablation is advised for reccurent cases.

Atrial fibrillation

The atrial activity is completely chaotic. The atrium beats more than 500 times a minute. The AV node allows only occasional impulses to pass through at variable intervals generating an irregularly irregular rhythm.

Important identifying features are –

- No p waves.
- Irregular appearance of QRS complexes.
- Undulating base line.

Causes of atrial fibrillation are –

IHD (acute and chronic), RHD (mitral valve disease), Heart failure, Hypertension, Thyrotoxicosis, Cardiomyopathies, Congenital(ASD), Pericardial diseases, Pulmonary embolism, Pneumonia, COPD,

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MVP, After alcohol binge, Lone AF.

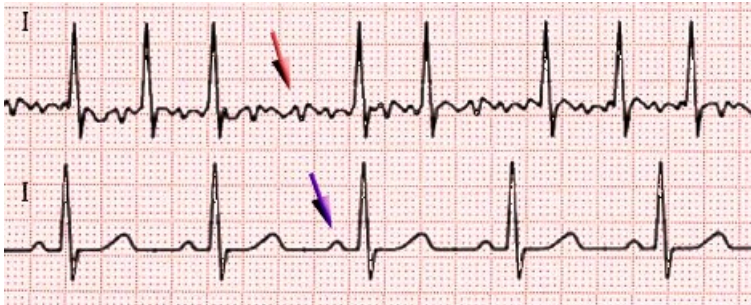


Fig 7.7 – ECG showing comparison between atrial fibrillation and normal sinus rhythm. [Source – Wikipedia, Author – Heuser].

Treatment Box

- Rhythm control / With hemodynamic compromise –
 - a. Electric cardioversion – Synchronised DC shock is preferred when the AF has been present less than 48 hours and there is clot in the left atrial appendage. Most of the patient revert back to normal sinus rhythm with this method.
 - b. Pharmacological cardioversion –

Drug of choice is intravenous Ibutilide. Dose: 1 mg over 10 minutes. Repeat dose may be required.

Amidarone, Dose: 150mg in 100ml of NS in 10 minutes. Followed by an infusion of 1 mg/min for the next 6 hours.
- Rate control / Without hemodynamic compromise –

Diltiazem, Dose: loading dose- 0.25mg/kg over 2 minutes → 0.35 mg/kg after minutes → Infusion of 5-15 mg/hour for 24 hours if needed. Respond to bolus dose of diltiazem is evident within 5 minutes in more than 85 % of the cases.

Amiodarone, Metoprolol can also be given.

Digoxin- It is not useful in cases of acute AF as its onset of action takes several hours. However it is indicated in chronic AF and chronic heart failure due to left ventricular systolic dysfunction. Dose: Tab Digoxin 0.5 mg every 8 hours x 3 doses followed by 0.25 mg/day.

- Anti-thrombotic strategies – Indicated in all patients with AF more than 48 hours, patients with rheumatic heart diseases, prosthetic heart valve, etc.

Heparin 5000 units iv as bolus and infuse 1000-2000 units / hour. Continue Heparin for 4-5 days.

Start loading of Warfarin 10 mg on the same day of starting heparin and continue with a maintenance dose of 2-5 mg / day. Note our aim is to achieve an INR of 2-3. With the use of antithrombotics; the risk of thromboembolism is known to decrease to a large extent.

Premature ventricular ectopics

These are very common.

For identifying PVCs-

- Wide QRS complexes.
- No or retrograde p waves
- A prolonged compensatory pause before the next beat is usually present.

Bigeminy means that the PVC is present in a ratio of 1:1. Similarly, *Trigeminy* means that the PVC is present in the ratio of 2:1 and so on; 2 VPCs when seen in a row then is called as *couplet*. Similarly 3 VPCs in a row is called triplet.

When should we worried for these PVCs –

- When it occurs in a case of a MI.

Shortcut to Electrocardiography

- In cases of frequent PVCs.
- When 3 or more PVCs occur consecutively in a row.
- When PVC falls on a T wave of a previous beat; also known as R on T phenomenon.
- Multiform PVCs.

When associated with these above conditions, premature ventricular contractions can trigger ventricular tachycardia and fibrillations.

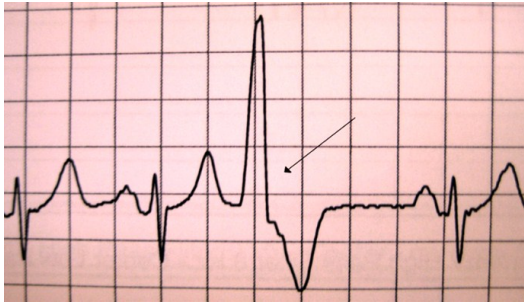


Fig 7.8 – ECG showing a ventricular premature contraction. [Source – Wikipedia, Author – James Heilman, MD].

Treatment Box

- In normal healthy individual no treatment is required. Reassurance must be done.
- Eliminate triggers like caffeine and tobacco.
- Drug therapy – Beta blockers, calcium channel blockers, anti-arrhythmic drugs like amiodarone can be prescribed in symptomatic individuals.

Ventricular tachycardia

A run of 3 or more consecutive PVCs is called as ventricular tachycardia. It is an emergency which commonly leads to cardiac arrest.

The rate is usually 120 to 200 beats per minute.

Ventricular tachycardia may be difficult to distinguish from supra ventricular tachycardia with bundle branch block or pre excitation syndrome (WPW syndrome).

Features in favor of ventricular tachycardia -

- History of MI.
- AV dissociation (pathognomonic).
- Capture or fusion beats (pathognomonic).
- Extreme left axis deviation.
- Very broad QRS complexes more than 140 msec.
- No response to carotid sinus massage or intravenous adenosine.

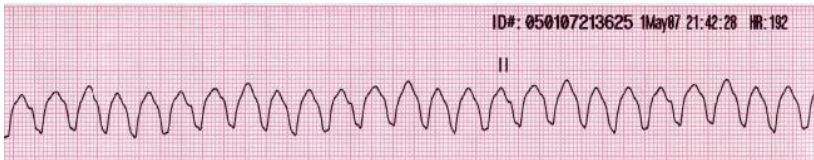


Fig 7.9 – ECG showing ventricular tachycardia.

Treatment Box

- Note that VT is a “medical emergency”.
- Modes of treatment are –
 - a. Electrical cardioversion. – DC cardioversion is the treatment of choice if the blood pressure is less than 90 mm hg.
 - b. Medical cardioversion. – Amiodarone iv bolus followed by infusion. Beta blockers may also be effective.
 - c. Catheter radiofrequency ablation.

Shortcut to Electrocardiography

Torrside pointes –

It is a French word which means “twisting of the points”. It is a rare type of ventricular tachycardia which is encountered in patients having prolonged QT intervals.

Causes are hypocalcemia, hypomagnesaemia, hypokalemia, drugs like Drsopyramide sotalol, amiodarone, amitryptilline, chlorpromazine, erythromycine.

Characteristic feature: The QRS complexes spiral around the baseline changing their axis and amplitude.



Fig 7.10 – ECG showing torsade pointes.

Ventricular fibrillation –

It usually follows ventricular tachycardia and it is a pre terminal event.



Fig 7.11 – ECG showing ventricular fibrillation.

Atrio-Ventricular Blocks

It refers to conduction block between the sinus node and purkinje fibres.

These can be easily diagnosed by examining the relationship of the p waves to the QRS complexes.

1st degree:

Shortcut to Electrocardiography

It is not really a block but a delay in conduction of the electrical impulse.

Mechanism – The impulse of depolarization spreads normally from the SA node but upon reaching the AV node; the conduction/passage is prolonged.

ECG feature – Prolonged PR interval i.e. longer than 0.2 seconds.

Remember all the impulses pass through the AV node in 1st degree AV block; they are just delayed.

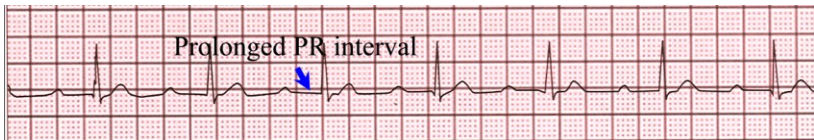


Fig 7.12 – ECG showing 1st degree heart block.

Treatment Box

- Correct all the underlying conditions like maintaining electrolyte balance, withdrawing any offending medication.
- Usually does not progress into higher forms of heart block.
- In general, no treatment is required for asymptomatic patients.

2nd degree:

Not all the impulses pass through the AV node into the ventricles.

It is of 2 types.

Mobitz type 1 – The block is variable. Each atrial impulse suffers from a longer and longer delay in the AV node until one impulse fails to conduct or make through it.

Shortcut to Electrocardiography

It is due to block high up in the AV node and it rarely progresses into 3rd degree heart block.

ECG – Shows a progressive lengthening of the PR interval then which is not followed by a QRS complex at all.

We usually describe this type of block in ratios. For e.g., 4:3 or 5:3. Lesser the ratio more severe it is.

It is also called as Wenckebach phenomenon.

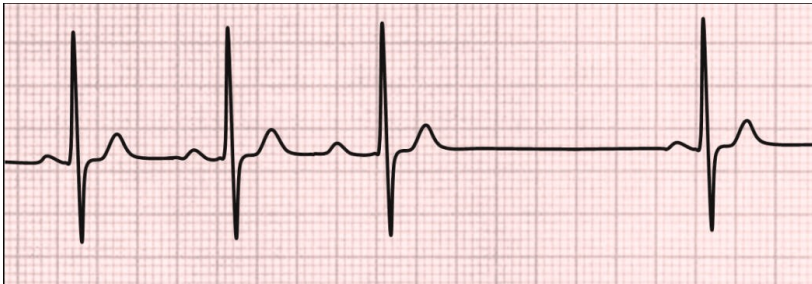


Fig 7.13 – ECG showing Wenckebach phenomenon.

Treatment Box

- No treatment is required for asymptomatic individuals.
- If symptomatic, give intravenous atropine to improve the conduction through the AV node and thereby increasing the heart rate.

Mobitz type 2 - This type is very similar to that of type 1 but it differs in such a way that there is no progressive lengthening seen. The block is constant. For e.g., either 3:2, 2:1 etc.

It commonly progresses into 3rd degree heart block.

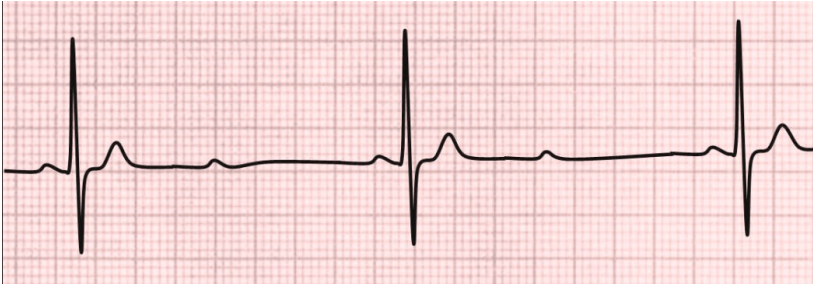


Fig 7.14 – ECG showing Mobitz type 2 heart block.

In 2:1 AV blocks, alternate p waves are conducted so it is impossible to distinguish between Mobitz type 1 and type 2 blocks.

Treatment Box

- Admit all patients in cardiac care units.
- Temporary pacemakers are indicated in such patients.

3rd degree heart block:

Often called as complete heart block. It implies that no impulses are being conducted through the AV node. It is characterized by total AV dissociation.

ECG – Regular p waves beating at a rate of 60-100 beats per minute.

Regular QRS complexes at a rate of 30-40 beats per minute.

No relationship between p waves and QRS complexes.

Complete heart blocks can be congenital in origin or may be acquired like in idiopathic fibrosis, myocardial infarction, trauma, trauma, drugs (digoxin, beta blockers).

Shortcut to Electrocardiography

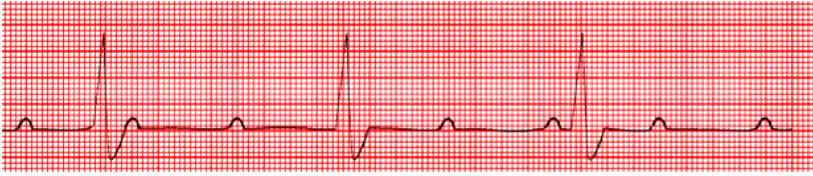


Fig 7.15 – ECG showing 3rd degree heart block.

Treatment Box

- If associated with inferior wall infarction, the patient can be observed as it may revert back to normal sinus rhythm. If symptomatic or persistent, pacemaker should be inserted immediately.
- If associated with anterior wall infarction, pacemaker should be installed immediately.

Chapter 8. Ischemic heart diseases (MI).

What is the simple basic pathology behind any infarction?

It all starts with atherosclerosis → Gradual narrowing of the coronary arteries → Complete occlusion of a particular coronary artery (usually occurs due to superimposed thrombosis and or coronary artery spasm). → Leads to myocardial infarction.

Diagnosis of MI involves several of signs, symptoms and lab parameters.

Symptoms – Retrosternal/central chest pain radiating to the jaw and or left arm. Nausea, excessive sweating and shortness of breath.

Note that silent myocardial infarction is seen in elderly and diabetics.

Lab parameters – Troponin I and CKMB are the markers which are commonly assessed in every patient giving a history suggesting of MI.

Troponin I rises earlier than CKMB. CKMB levels rises 6 hours after an infarction and they return to normal levels within 48 hours.

ECG changes seen in myocardial infarction

In an acute stage; the ECG changes occurs through the following stages –

1. Changes in the T waves.
2. ST segment.
3. Q waves.

With the onset of infarction T waves becomes tall and narrow. This is known as peaking; which is referred to as hyper acute T waves.

Shortcut to Electrocardiography

Few hours later these T waves invert. Changes of the t waves reflect ischemia.

If the blood flow is restored, t wave changes revert to normal.

ST segment elevation signifies myocardial injury i.e. infarction and not just ischemia. ST segment elevation more than 1 mm above the iso-electric lines in 2 consecutive leads is diagnostic of myocardial infarction.

Q waves are pathognomic of myocardial infarction. It must be greater than 0.04 seconds in duration and the depth should be at least $1/3^{\text{rd}}$ the height of R waves in the same QRS complex.

Serial ECG Changes in MI

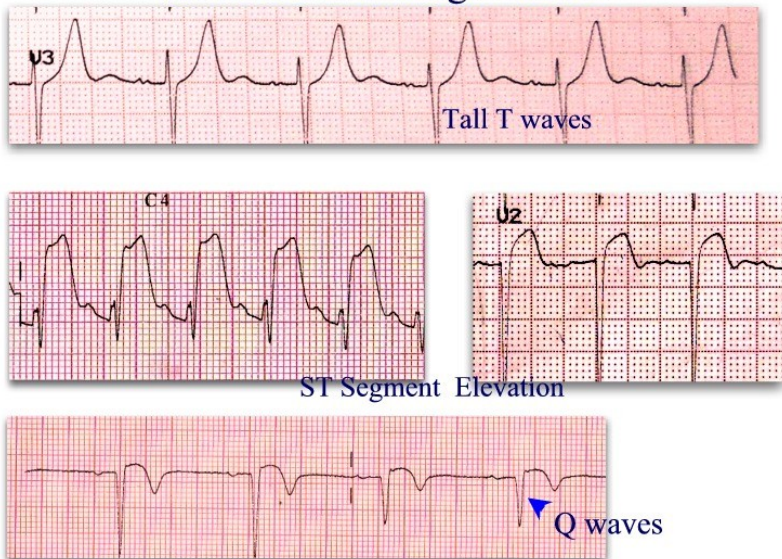


Fig 8.3 – Figure showing serial ECG changes as evolution of myocardial infarction occurs.

Treatment Box

Management of acute myocardial infarction –

- General –
 - a. Secure and IV line.
 - b. Start oxygen at 4-6 liters per minute and achieve an oxygen saturation more than 95%.
 - c. Admit the patient in cardiac intensive unit.
- Antiplatelet therapy –
 - a. Aspirin, dose: 300 mg per oral (to be chewed and swallowed) → 75 mg PO/day.
 - b. Clopidogrel, dose: 300 mg per oral (to be chewed and swallowed) → 75 mg PO/day.
- Pain –
 - a. Isosorbtrate-dinitrate to be given sub linguallly 5 mg stat.
 - b. Nitroglycerine sub linguallly 0.5 mg x 3 doses at 5 minutes intervals.
 - c. Morphine 2.5 mg iv every 5-10 minutes till the pain the relieved or a total of 15 mg.
 - d. If the pain still persists; start Nitroglycerine iv drip if the SBP>100 mm hg.
- Reperfusion therapy –
 - a. Thrombolysis –

Dissolves the clot and restores the circulation. But best benefit if done within 4-6 hours.

Drugs used: Recombinant tissue plasminogen activators like alteplase, reteplase or tenecteplase. Steptokinase is also widely used (commonly available).

Alteplase: 15 mg iv bolus followed by 50mg over

Shortcut to Electrocardiography

30 minutes and 35 mg over the next 60 minutes.

Streptokinase: 1.5 million units iv in 1000 ml of normal saline should be infused over 1 hour.

b. Percutaneous intervention –

Consists of coronary angiography to find the clot followed by balloon dilatation with or without stent insertion.

• ACE inhibitors –

Started if there is no hypotension. Captopril 6.25 mg PO TDS can be started or Enalapril 5-10 mg PO per day is started.

Long term therapy prevents ventricular remodeling and prevents heart failure.

• General measures –

- a. Reassurance is very important.
- b. Strict bed rest for at least 48 hours.
- c. Relieve anxiety by giving anti-anxiety drugs.
- d. Keep the patient on liquid diet for 12 hours followed by soft and normal diet.
- e. Care of the bowel can be done by giving mild laxatives.
- f. Gradual mobilization of the patient plays an important role in the treatment of MI.

Chapter 9. Pre excitation syndromes.

Excitation syndromes are opposite of conduction blocks. Here the electrical current flows more quickly than the usual.

There are two main types of pre excitation syndrome –

Wolf Parkinson White syndrome and Lown Ganong Levine syndrome.

Mechanism behind these syndromes –

These syndromes are characterized by an accessory conduction pathway which helps the impulse to make a shortcut to arrive to the ventricles ahead of time and depolarize them prematurely.

WPW syndrome:

The bypass pathway from the atria to ventricles is called as the “bundle of kent”. It can be on the left or on the right side.

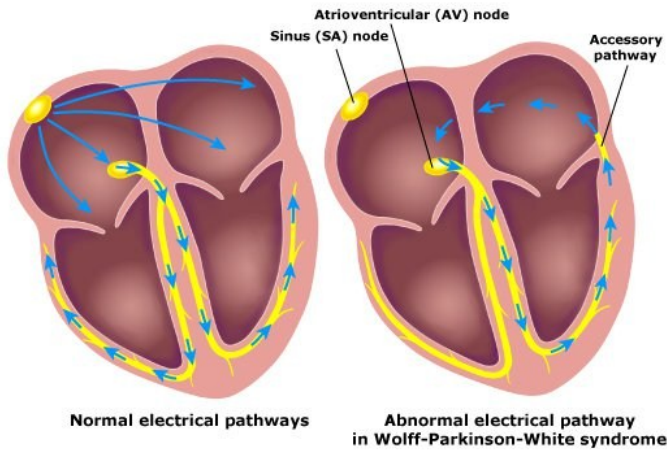


Fig 9.1 – Figure showing the bundle of kent (accessory pathway). [Source – wikipedia, Author – Tom Lück].

Characteristic features are –

- Short PR interval.
- Widened QRS complex as a fusion beat is formed.

Shortcut to Electrocardiography

- Presence of delta waves (a small region of myocardium that is depolarized early gives the QRS complex a characteristic slurred initial upstroke).

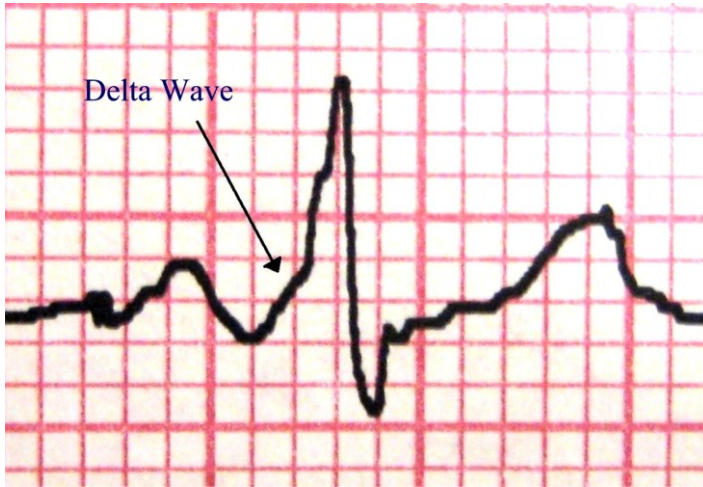


Fig 9.2 – ECG showing WPW syndrome. .[Source – Wikipedia, Author – James Heilman, MD].

Lown Ganong Levine syndrome:

The bypass/accessory pathway is called as the “James fibres”.

Here an intra nodal pathway is created. It bypasses the delay within the AV node. Thus no delta waves are formed and normal QRS complex is present but a short PR interval is there.

These phenomenon are important to be diagnosed as these can cause arrhythmias like PSVT and atrial fibrillation.

Chapter 10. Bundle branch blocks.

These are diagnosed by looking at the width and configuration of the QRS complexes. As we know these right and left bundles supplies the depolarization wave to the respective ventricles, thus it is easier to different these blocks according to the overlying leads.

Right bundle branch block –

The conduction to the right bundle branch is impaired/obstructed → Thus right ventricular depolarization is delayed → Hence wide QRS complexes are formed on the ECG.

ECG – a unique diagnostic shape is seen in those leads overlying the right ventricle i.e. V1 and V2. The typical complex formed is RSR' / M shaped / Rabbit ears.

Formulae- V1 (MORROW) V6.

Causes – may be present as a normal variant, in right ventricular hypertrophy, pulmonary embolism, congenital (e.g. ASD), coronary artery diseases.

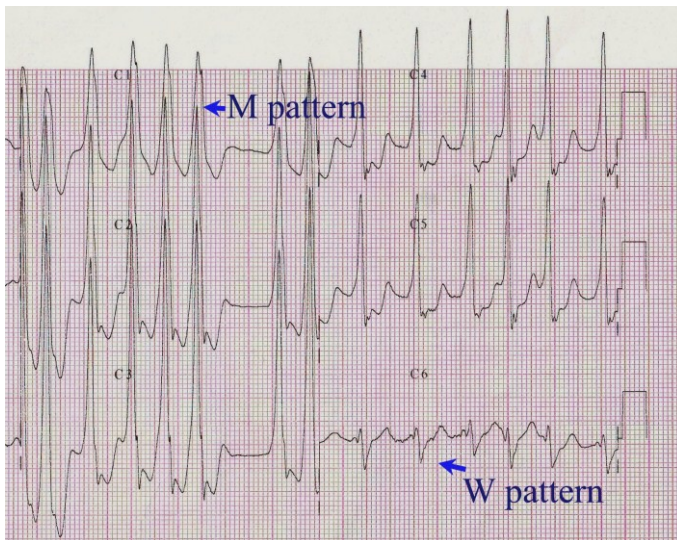


Fig 10.1 – ECG showing right bundle branch block.

Shortcut to Electrocardiography

Left bundle branch block –

Here the conduction through the left bundle branch is impaired or obstructed.

ECG changes are seen in the QRS complexes in the leads overlying the left ventricle (V5, V6, I and AVL).

As these leads V5 and V6 normally have tall R waves, thus the change i.e. seen is marked prolongation in the rise of the tall R waves and they are either broad or notched.

Formulae – V1 WILIAM V6

Causes – coronary artery disease, hypertension, aortic valve disease, cardiomyopathy.

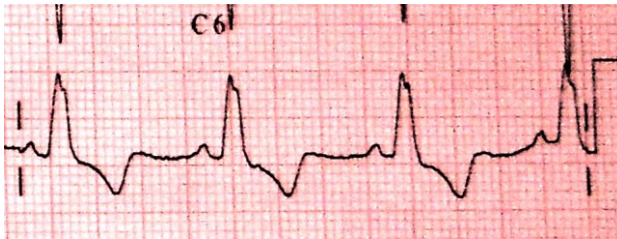


Fig 10.2 – ECG showing left bundle branch block. Note the RSR' (M) pattern formed in lead C6.

Note other associated features seen on ECG with bundle blocks are – ST segment depression and T wave.

Note that changes are seen in right precordial leads in RBBB and in the left lateral leads in LBBB.

Chapter 11. ECG changes seen in electrolyte imbalances.

Hyperkalemia

Presence of ECG changes is considered to be a good measure/marker of potassium toxicity than the serum potassium levels.

ECG changes seen with increasing potassium levels –

- Tall peaked T waves across the entire 12 lead ECG.
- PR interval is prolonged and gradually it flattens or disappears.
- With extremely high potassium levels the QRS complexes widens and merges with the T waves; forming the sine wave pattern.
- Ventricular fibrillation may soon be formed.

Hypokalemia

ECG changes seen with decreasing potassium levels –

- ST segment depression.
- Flattening of the T wave.
- Appearance of U wave.

Hypo/Hyper calcemia

Changes in the serum calcium levels primarily affect the QT interval. Hypocalcemia is associated with prolonged QT interval; and hypercalcemia is associated with short QT interval.

Shortcut to Electrocardiography

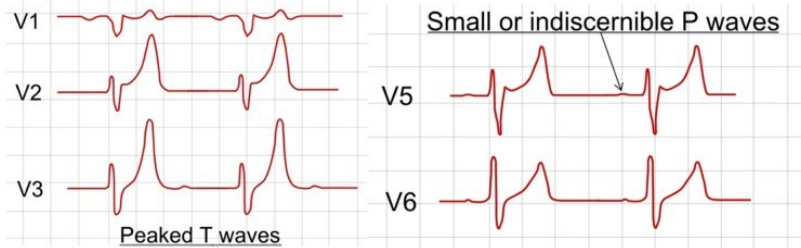


Fig 11.1 – ECG showing features of hyperkalemia.

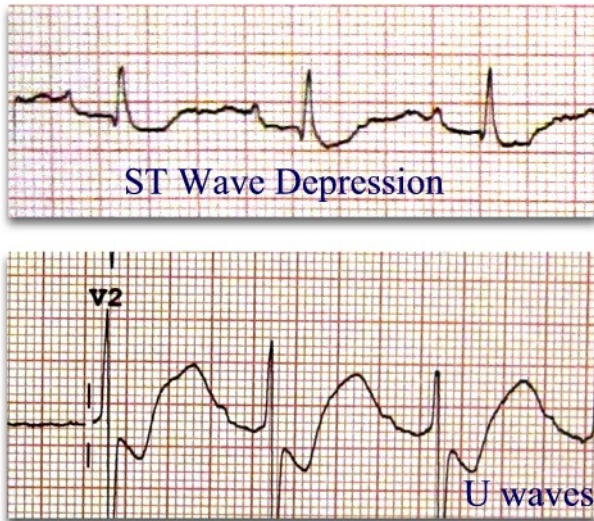


Fig 11.2 – ECG showing features of hypokalemia. [Source- Wikipedia, Author - James Heilman, MD].

Chapter 12. ECG changes seen in other systems.

Chronic obstructive pulmonary disorder

ECG features seen are –

- Low voltage ECG – specially seen in longstanding emphysema because of the dampening effects of the expanded residual air trapped in the lungs.
- Right axis deviation – the expanded big lungs forces the heart into a vertical or even rightward oriented position, also the pressure overload hypertrophy may be responsible for it.
- Right atrial enlargement (p pulmonale) – characteristic of patient in cor pulmonale.

Acute pulmonary embolism

ECG features seen –

- Right ventricular hypertrophy with strain.
- Right bundle branch block.
- S1Q3 pattern – i.e. presence of a large S wave in lead I and a deep Q wave in lead III.
- May be associated with arrhythmias like sinus tachycardia and atrial fibrillation which are more common.

Central nervous disorders like hemorrhage/cerebral infarctions

Diffuse T wave inversion and prominent U waves with sinus bradycardia are commonly seen in such patients. It is probably due to the involvement of the autonomic system.

Shortcut to Electrocardiography

Summary : Stepwise approach for reading an 12-lead ECG.

<i>Step 1</i>	Make sure that the standardization mark on ECG paper is correct. Make sure that the paper speed is correct.
<i>Step 2</i>	Calculate the heart rate.
<i>Step 3</i>	Measure intervals – PR interval, QT interval, width of the QRS complex.
<i>Step 4</i>	Find out the axis.
<i>Step 5</i>	Access the rhythm – normal p waves present or not? QRS complex wide or narrow? Relationship of p waves and QRS complex, rhythm regular or irregular?
<i>Step 6</i>	Find out if there is any AV block.
<i>Step 7</i>	Find out if there is any bundle branch block.
<i>Step 8</i>	Look for any features suggestive of enlargement or hypertrophy.
<i>Step 9</i>	Look for changes of coronary artery disease – look Q waves, ST segment and T waves changes.
<i>Step 10</i>	In case of confusion, never hesitate for assistance from a more experienced physician.