Review Article

Basics of Electrocardiogram (ECG) and Its Application in Diagnosis of Heart Ailments: An Educational Series

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ABSTRACT

Electrocardiogram (ECG) is a record of electrical activity of the heart. PQRST waves represent the electrical activities of atria and ventricles. A complete three-dimensional electrical activity is possible to be recorded using a 12-lead ECG. The normal and different routinely-met clinical ECG are elaborated and discussed. This routine, normal and abnormal ECG, like arrhythmias and heart block records as well as their clinical notes shall be educational information for the medical students.

Keywords: ECG, lead, conduction system, arrhythmia, heart block

INTRODUCTION

Electrocardiogram is the graphical record of different electrical activities of the heart. Electrocardiograph is the instrument or machine that is used to record the electrical activity generated by different parts of the heart such as conducting system, atria and ventricles. The basic functionality of ECG is diagnostic but not therapeutic in nature. ECG helps to specifically point out the abnormalities and an aid in the diagnosis which further enable the therapeutic approach.

FUNCTIONAL ANATOMY OF THE HEART

Human heart is made up of four chambers; two atria and two ventricles. It is connected to both oxygenated and deoxygenated system of blood flow wherein these two separate blood circulations never mix but rather exchange in such a way that oxygenation and removal of waste products continue throughout the human life. Greater circulation starts at left ventricle and via the aorta and subsequent successive generations of arteries, arterioles and capillaries blood are propelled forward while delivering oxygen from the capillaries to the tissues. Tissues exchange the waste and carbon dioxide in return and divert it via the lesser or pulmonary circulation to the alveoli in lungs where waste and carbon dioxide are removed while oxygen is loaded to make it oxygenated blood. Heart, greater and lesser, circulation helps in the oxygenation and de-oxygenation and this mechanical event is governed by a well-synchronized electrical impulse that is regularly generated at the SA node and transmitted along the conducting tissues of the heart.

CONDUCTION SYSTEM OF THE HEART AND SPREAD OF CARDIAC IMPULSE

SA node generates the impulse at regular intervals which is traversed down along the AV node, bundle of His, right and left bundle branches and Purkinje system. Two important unique aspects of this conducting system is that, AV nodal delay and functional continuity of atria and ventricles through AV node. This makes atria and ventricle to function as separate units and giving enough time for the systole, diastole in return, filing and emptying of atria and ventricles into their respective vessels in a synchronized fashion. All these functions are possible because of the electrical impulses that are traversing rhythmically and systematically generated at SA node of a healthy heart.

As the impulse traverses along both the atria, almost 100 million atrial cells contract simultaneously in a short duration of one-third of a second as result of instantaneous depolarization of atrial myocardium.

Simultaneously, the same impulse traverses the AV node and because of AV nodal delay takes a brief time to proceed further to reach the bundle of His and further along the conducting pathway and subsequently to both the ventricles. AV nodal delay is a nature's gift as it allows the atria to completely pump the blood into the ventricles at the same time maintaining the synchrony and pace of atrial emptying and ventricular filling. As the impulse traverses subsequently into the ventricles around 400 million cells of the ventricles depolarize simultaneously that result in ventricular contraction. Atria and ventricles discharge separately in unison because of the intercalated discs among the myocardial cells so that atria and ventricles act as primer and power pumps to direct the blood forward along the closed cardiovascular circuitry.

Each SA nodal impulse results in single heart beat or a single cardiac cycle. This involves, atrial depolarization, ventricular depolarization and their repolarization electrically and atrial systole, ventricular systole and their diastoles mechanically.

HEARTBEAT

A single pacemaker potential of SA node lead to atrial depolarization, ventricular depolarization and atrial and ventricular repolarization. This potential as it is transmitted along the atria, P wave is developed since atria is a syncytium followed by ventricular QRS and then ventricular repolarization – T wave. This is enabling the atria and ventricle to undergo atrial systole, diastole and ventricular systole and diastole with an average 0.8 sec interval in a normal heart that covers a single heartbeat. Voluntary consent from the patients was obtained to publish the recordings and data of ECG.

ELECTROCARDIOGRAPH

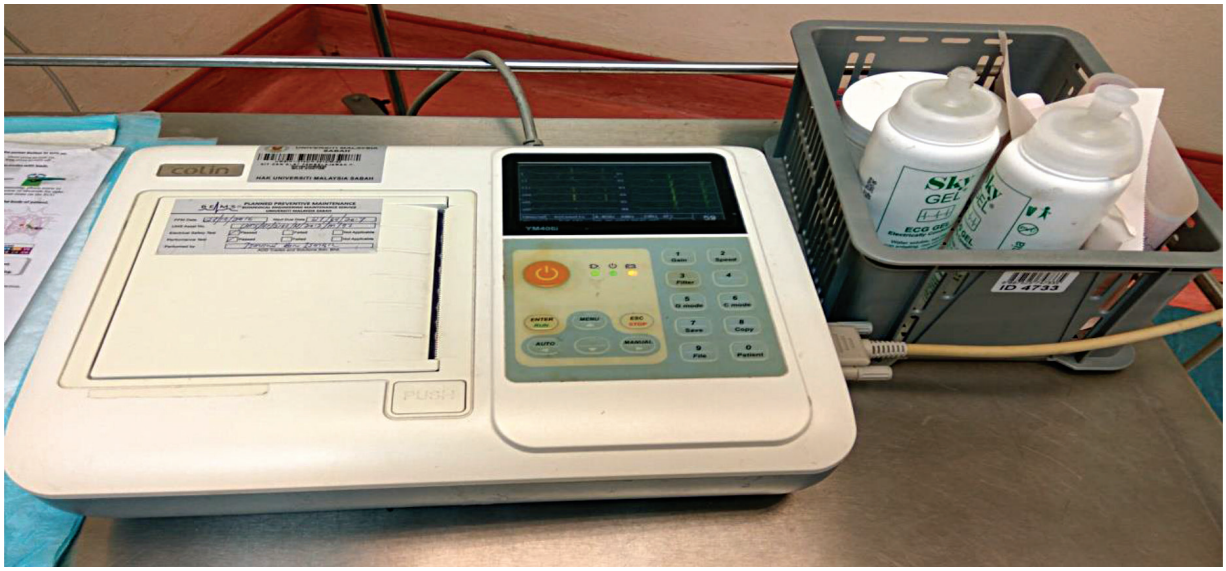

Figure 1 Electrocardiograph
[CoLin 6 channel ECG Ref: YM406i, SN MD 094120800233-2012-08 (Korea)]

Electrocardiograph or ECG machine is able to pick the electrical deflections that are produced during atrial and ventricular depolarization and repolarization from the specific points of the skin in a non-invasive manner and is able to record simultaneously the deflection on a grid of ECG paper.[1] Pacemaker potential helps in initiating the discharge of SA node. It is a rhythmic impulse that is able to produce a depolarization wave followed by repolarization and is able to traverse along the conducting system of the heart and simultaneously able

to spread along the atrial muscle fibres. This results in complete depolarization of both the atria resulting in a deflection which is picked up by the ECG electrodes resulting in 'P' wave. P wave is a depolarization wave of atria, in other words, it is the resultant summated depolarization of every atrial fibres. This is followed by atrial repolarization. In the ECG record due to the ventricular depolarization already making its way, atrial repolarization will be masked or submerged in the ventricular depolarization wave-QRS complex. Atria and ventricles are connected functionally only through AV node as there is a fibrous tissue or ring that separates the atria and ventricle electrically and AV node is the only link that connects the two functionally. It is a nature's gift that aids the mechanical component of atria and ventricle and aids in the continuous forward flow of blood along the closed cardiovascular circuitry. This is supported by AV nodal delay as the impulse is delayed for a few milliseconds duration because of the resistant nature of AV node, which gives enough time for the ventricle to fill, atrial systolic phase. The impulse from AV node traverses along the bundle of His, right and left bundle branches and then on into the Purkinje fibres which travels into the interiors of ventricular musculature and as a result the whole ventricular musculature depolarizes simultaneously and a 'QRS' complex which is a positive deflection in the electrocardiogram is recorded. Followed by this, ventricles repolarize fully with a resultant T wave in the ECG.

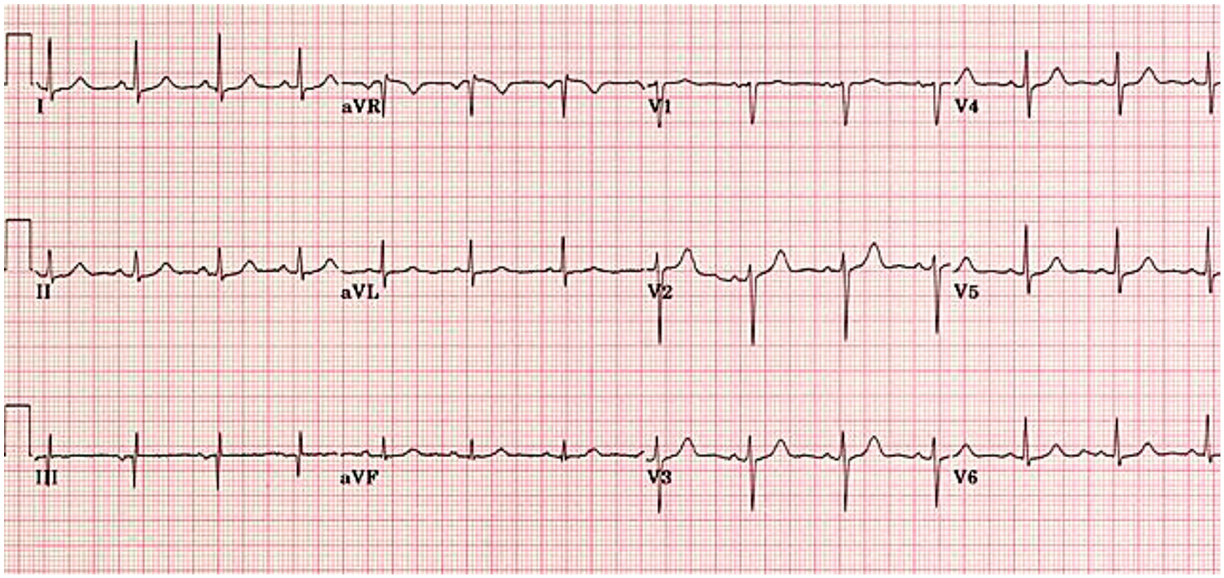

Figure 2 12-lead ECG record, normal sinus rhythm of heart rate 100/min

ECG Leads

A lead is an electrode with positive and negative terminals connected to the specific areas on the body surface. Leads pick up the electromotive force that is generated by the heart at different specific locations of the body. Generally, these terminals of leads are fixed on the skin surface as per the international standard locations. There are practically 12 leads that need to be used to record the electrical activity to know the complete three dimensional view of the heart. Accordingly, leads are classified as:

Standard limb leads: Lead I, Lead II and Lead III

Augumented standard limb leads: aVR, aVL and aVF

Precordial or chest leads: V_1, V_2, V_3, V_4, V_5 and V_6

12 different points on the body surface which are woven together to interpret a cohesive electrical activity and in return the functionality of the heart.

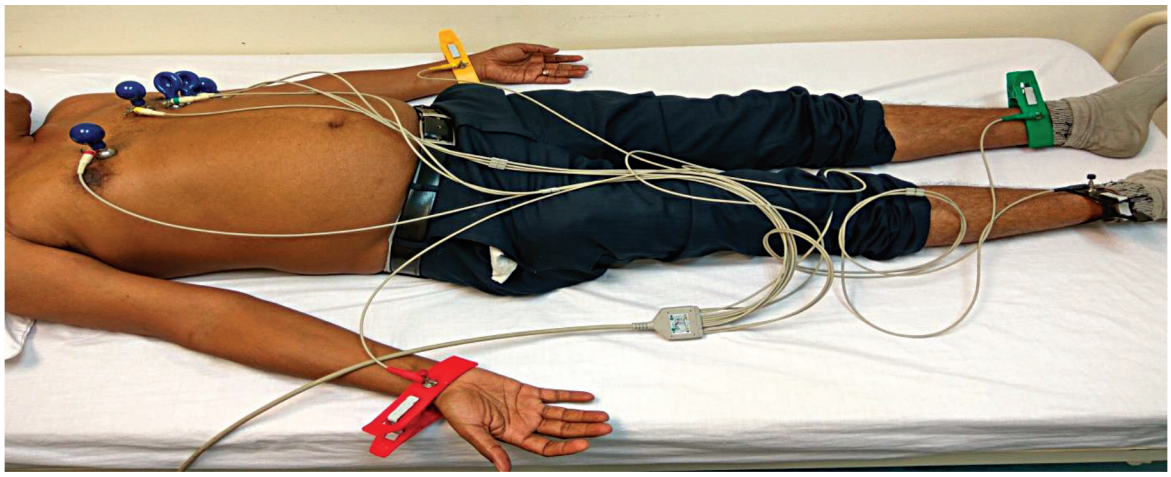

Figure 3 12-lead electrode placement

Limb electrode placements:

RA (Right Arm): Anywhere between the right shoulder and right elbow
RL (Right Leg): Anywhere below the right torso and above the right ankle
LA (Left Arm): Anywhere between the left shoulder and the left elbow
LL (Left Leg): Anywhere below the left torso and above the left ankle

Vertical plane (Frontal Leads):

By using 4 limb electrodes, 6 frontal leads that provide information about the heart's vertical plane

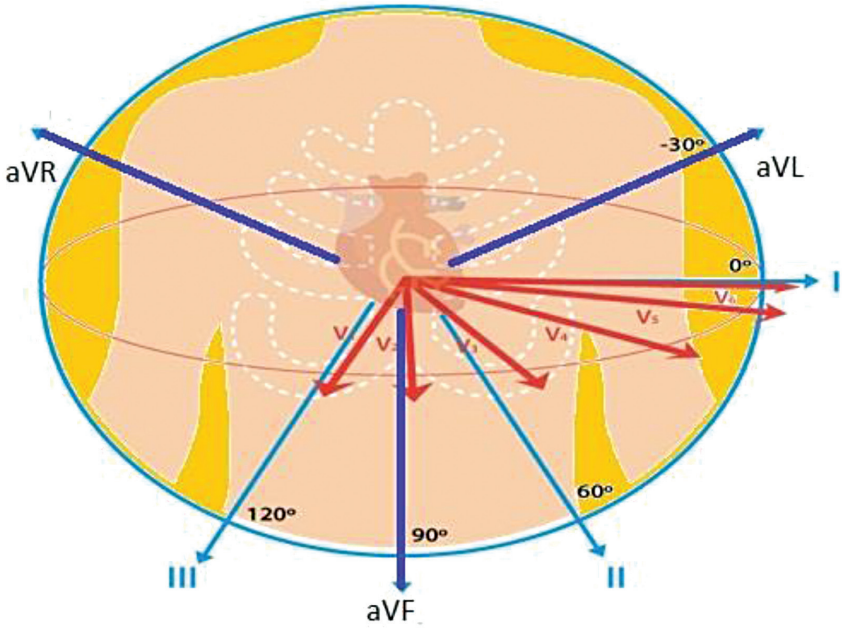

Figure 4 Electrical cardiac axis; mean electrical axis −30° to 90°

Lead I
Lead II
Lead III
Augmented Vector Right (aVR)
Augmented Vector Left (aVL)
Augmented Vector Foot (aVF)

Leads I, II, and III use negative and positive electrodes (as they are bipolar). The augmented leads; aVR, aVL, and aVF use only a positive electrode (as they unipolar).

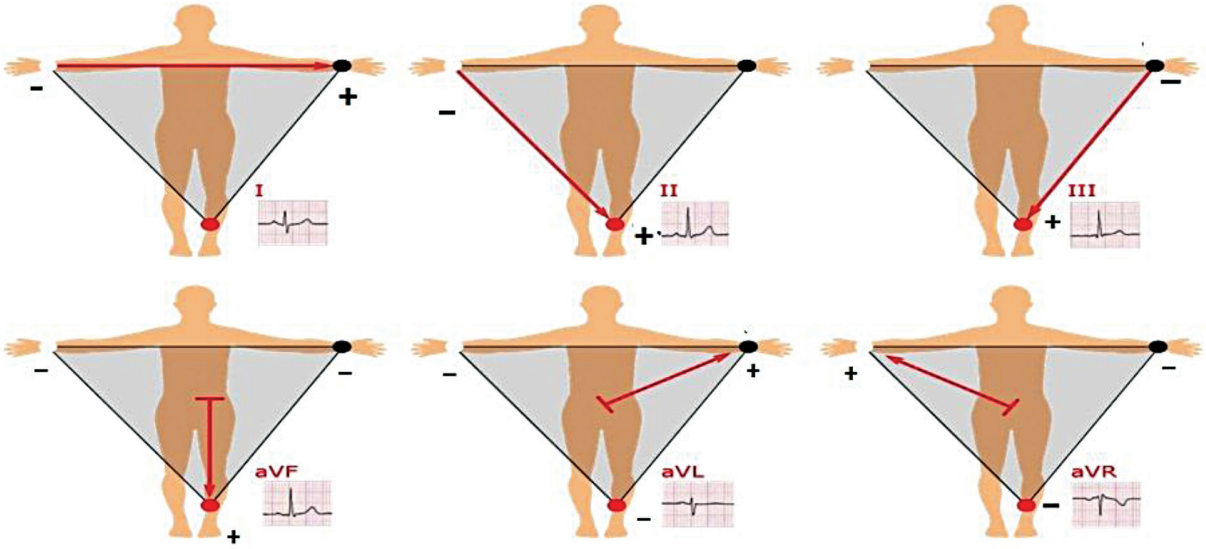

Figure 5 Einthoven Triangle with different lead axes

The Einthoven's triangle explains the basis of 6 frontal leads when there are only 4 limb electrodes.

The principle behind Einthoven's triangle demonstrates the role of individual electrodes RA, LA and LL where they record the electrical activity of the heart in relation to themselves through the aVR, aVL and aVF leads. They also correspond with each other to form lead I (RA to LA), II (RA to LL) and III (LL to LA).

When each lead corresponds with each other, they form an equilateral triangle called Einthoven's triangle. All standard limb leads form an equilateral triangle which is named after Willem Einthoven who pioneered the first practical ECG.

Right foot (RL) is a neutral point and known as point zero where the electrical current is measured. Generally RL does not come up in ECG readings, and is a grounding lead that helps to minimize ECG artefact.

Horizontal Plane (Transverse Leads)

By using 6 precordial or chest electrodes, transverse leads (6) are formed; provide information of heart's horizontal plane: V_1, V_2, V_3, V_4, V_5 and V_6.

Transverse leads are unipolar, they are the active electrodes, needs only a positive electrode. The negative pole of all 6 leads is found at the centre of the heart. This point is called central Wilsons terminal which is maintained at zero. This is calculated with the ECG.

Simonson (1953) has confirmed a decrease in the amplitude of R wave, deviation of axis to the right and ST segment depression in a normal exercising heart.[2] Though it is normal phenomenon in long term exercising heart, decreased R wave amplitude indicates left ventricular dysfunction, coronary vessel obstruction or both.[3, 4] Variability in the R wave amplitude as a result of exercise can be correlated which needs further ECG analysis to differentiate it from a diagnostic ECG of a patient. Postulations are that, correction of ECG measurements from the same ECG record with regard to the heart rates shall improve the diagnostic features of ECG and vector analysis is useful in assessing the haemodynamic functional as of the heart. Based on controversies and gaps, it needs to be further evaluated and a classification of the routine heart ailments with the ECG may help the under graduate medical students to have a thorough knowledge on different ECGs of different pathological conditions.

Determination of Heart Rate (HR) from ECG

On ECG paper horizontal or X-axis represents the time interval and 250 mm is equivalent to 1 second which is 5 large squares. To estimate

the HR, the number of squares in between two successive QRS complex is counted. If the number of squares between two successive QRS is 2, HR is 150/min and if 5, HR is 60 beats per minute.

1. P wave:

It is upright in leads I, aVF and $V_3 – V_6$. Normal duration is less than or equal to 0.11 seconds.

Its polarity is positive in leads I, II, aVF and $V_4 – V_6$; biphasic in lead V_1; negative in aVR. Shape is generally smooth, not notched or peaked.

2. PR interval:

It is between 0.12 and 0.20 seconds.

3. QRS complex:

QRS complex duration is less than or equal to 0.12 seconds, amplitude greater than 0.5 mV in at least one standard lead, and greater than 1.0 mV in at least one precordial lead. Upper limit of normal amplitude is 2.5 – 3.0 mV. Small septal Q waves in lead I, aVL, V_5 and V_6 duration is less than or equal to 0.04 seconds; amplitude is less than 1/3 of the amplitude of the R wave in the same lead. It is represented by a positive deflection with a large, upright R in leads I, II, $V_4 – V_6$ and a negative deflection with a large, deep S in aVR, V_1 and V_2. In general, proceeding from V_1 to V_6, the R waves get taller while the S waves get smaller. At V_3 or V_4, these waves are usually equal. This is called the transitional zone.

4. ST segment:

It is iso-electric, slanting upwards to the T wave in the normal ECG. It may be slightly elevated up to 2.0 mm in some precordial leads. ST segment never depressed greater than 0.5 mm in any lead.

5. T wave:

T wave deflection should be in the same direction as the QRS complex in at least 5 of the 6 limb leads. Normally rounded and asymmetrical, with a more gradual ascent than descent, should be upright in leads $V_2 – V_6$, inverted in aVR. Amplitude of at least 0.2 mV in leads V_3 and V_4 and at least 0.1 mV in leads V_5 and V_6. Isolated T wave inversion in an asymptomatic adult is generally a normal variant.

6. QT interval:

Durations normally less than or equal to 0.40 seconds for males and 0.44 seconds for females.

CLINICAL DIAGNOSIS OF ECG

All the clinical ECGs obtained were original records of the patients who had given their voluntary consent to publish for educational purpose. ECG is an important tool used to diagnose cardiac abnormalities. It is the basis of cardiac conditions. ECG is useful and plays a major significant role in diagnosis.

Mean electrical axis is the average direction of electro-motive force through the ventricle It is also called as cardiac vector. The mean electrical axis in the frontal plane extends between +30° and –110° and in the transverse plane it is between +30° to –30°. When this electrical axis deviates more towards right, it indicates right ventricular hypertrophy generally found in congenital heart diseases, severe pulmonary hypertension, cor-pulmonale, COPD and emboli in the lungs. If left ventricular hypertrophy, the axis is deviated to the left generally found in hypertension, IHD, conduction defects and aortic stenosis.

Arrhythmias

In a normal beating heart, the pacemaker is the Sino-atrial (SA) node, which regularly fires electrical impulse in a rhythmic fashion which

is conducted along the conducting pathways. This yield one cardiac cycle which equals to one beat of the heart with systole and diastole (mechanical event). In arrhythmia the normal conduction pathway is disturbed because of conduction blocks or additional ectopic foci. In an ECG paper, the regularity of electrical event is measured by finding the distance between the successive QRS complexes in a horizontal manner. This aids in measuring the atrial and ventricular rate and rhythm separately. This helps also in estimating the characteristics of ECG waves.

Atrial Fibrillation

Atrial fibrillations can occur because of the diseased sinus node or chaotic atrial contraction or sino-atrial conduction disturbances. The rate in atrial fibrillation is around 300 to 600, and also may not be able to detect the P waves separately in an ECG paper. Atrial fibrillation may or may not be associated with fast ventricular rate depending on the A-V conduction pathway. Atrial fibrillation apart from thromboembloism also can lead to myocardial infarct and cardiac failure.[4]

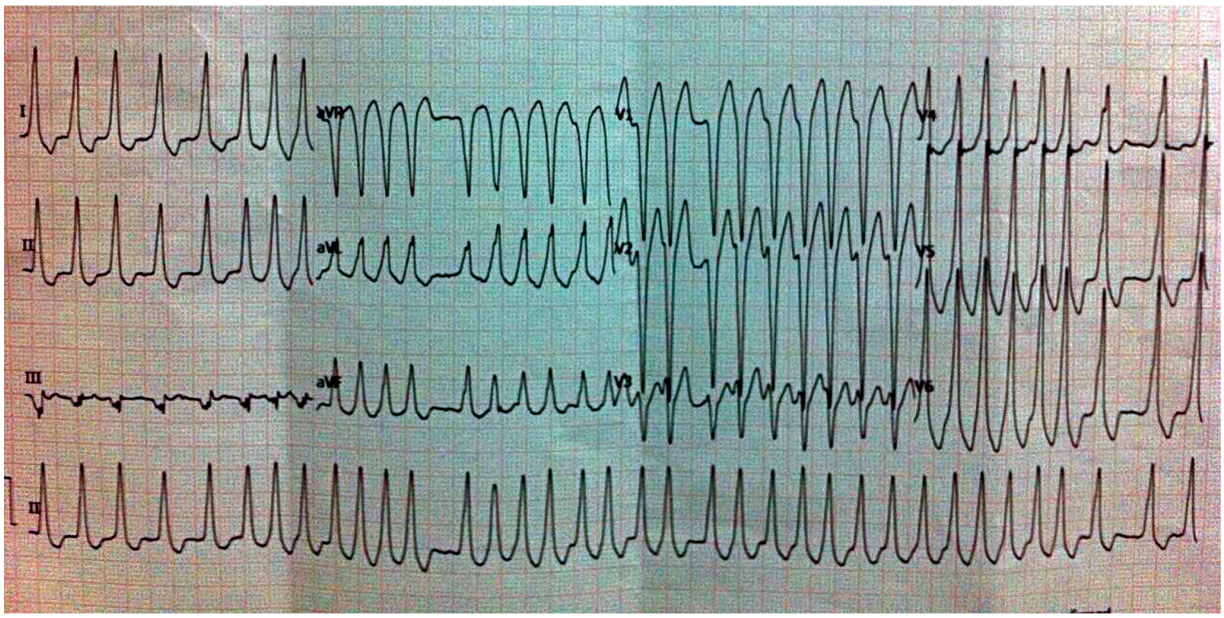

Figure 6 Atrial Fibrillation with fast ventricular response with aberrant conduction: Changing R-R interval, inconspicuous P wave (P wave invisible) and broad QRS complex. This type is commonly seen in rheumatic heart disease, ischaemic heart disease and underlying WPW syndrome, COPD and thyrotoxicosis.

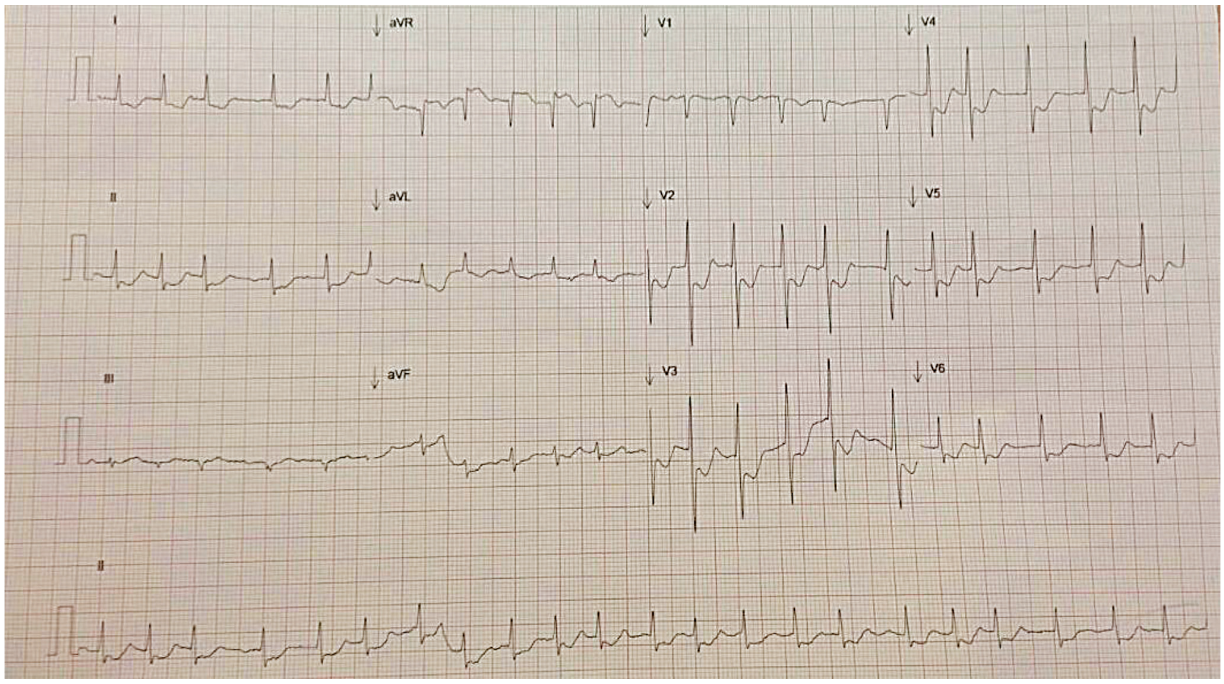

Figure 7 Atrial fibrillation with fast ventricular response

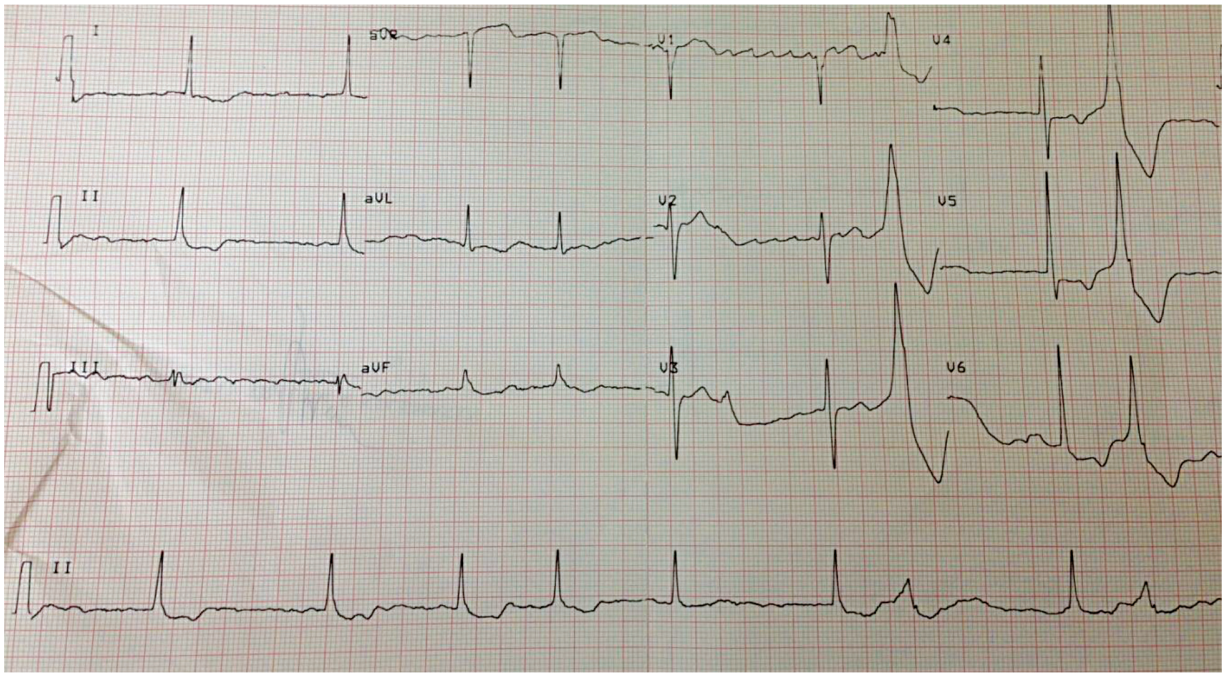

Figure 8 Atrial flutter and fibrillation: Re-entrant circuit within the atria causes atrial contraction at the rate of 250 – 350 causing saw-tooth appearance of the P waves and having variable blocks to the ventricular conduction.

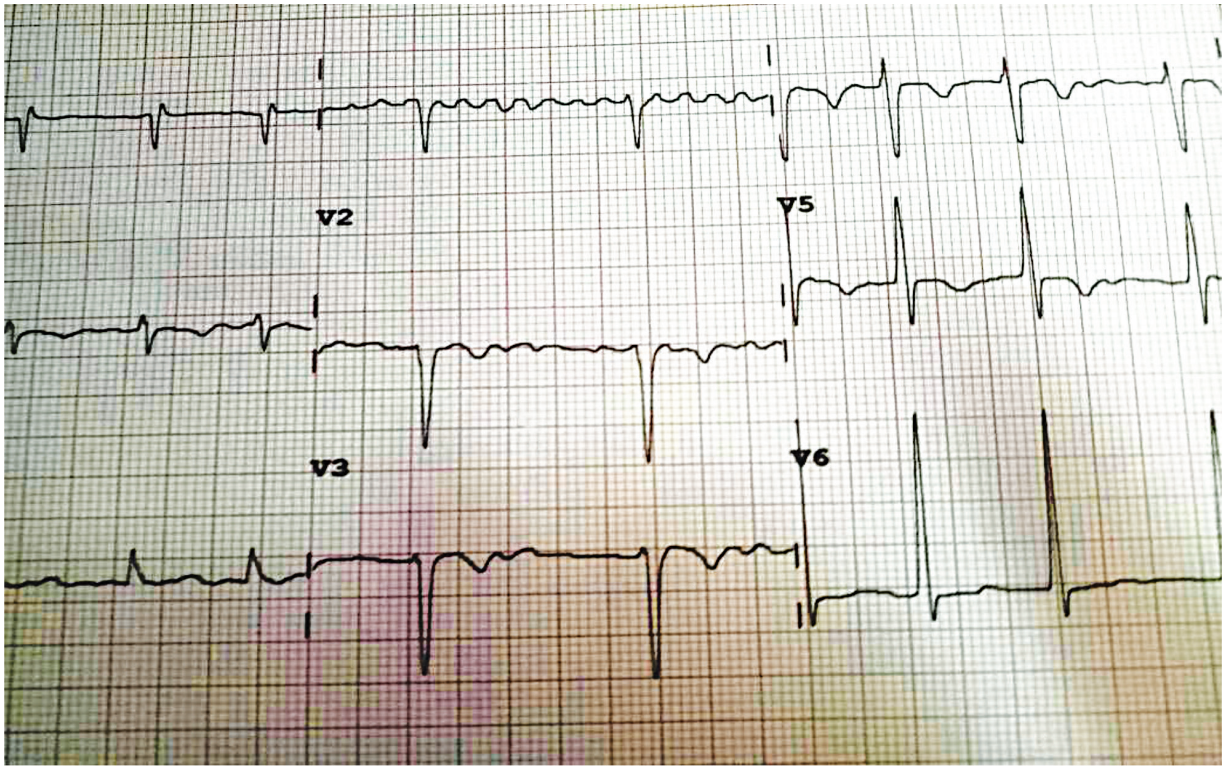

Figure 9 Atrial flutter in V_1 in the form of saw-tooth with atrial rate of more than 300, also seen associated atrial fibrillation with changing R-R interval with occasional ventricular premature beat. It can be seen in rheumatic heart disease, ischaemic heart disease, drug induced and COPD.

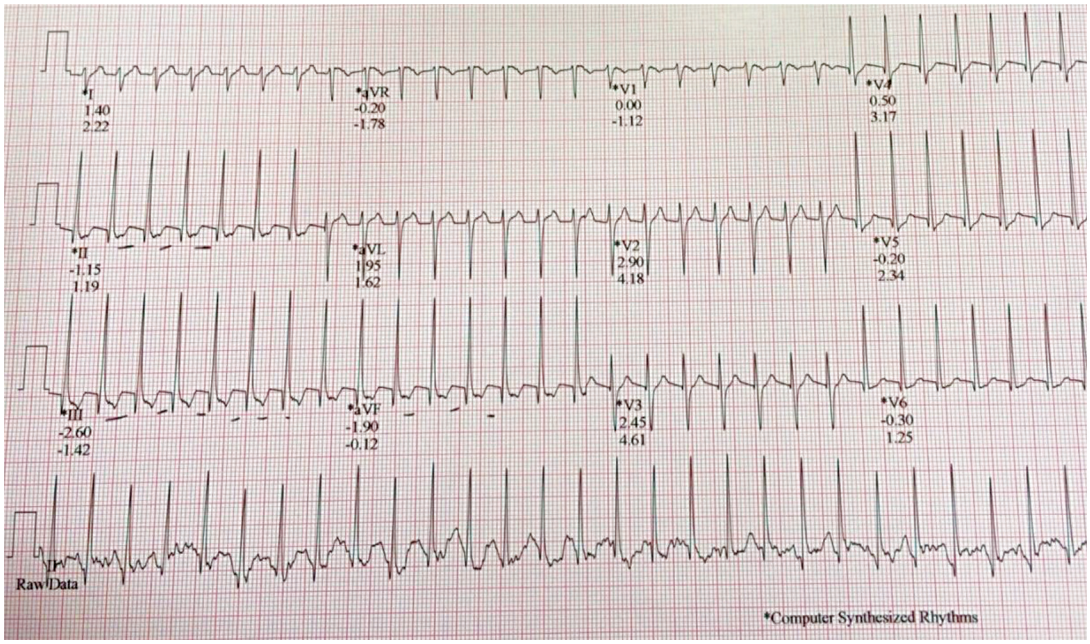

Figure 10 Supra-ventricular tachycardia (SVT)

It is also called paroxysmal supraventricular tachycardia. They are originated above the ventricles. In congenital tricuspid valve dysplasia SVT is common.[6] Atrial and ventricular rate is more than 150/min, narrow complex QRS. It is commonly seen in rheumatic heart disease, IHD, congenital heart disease.

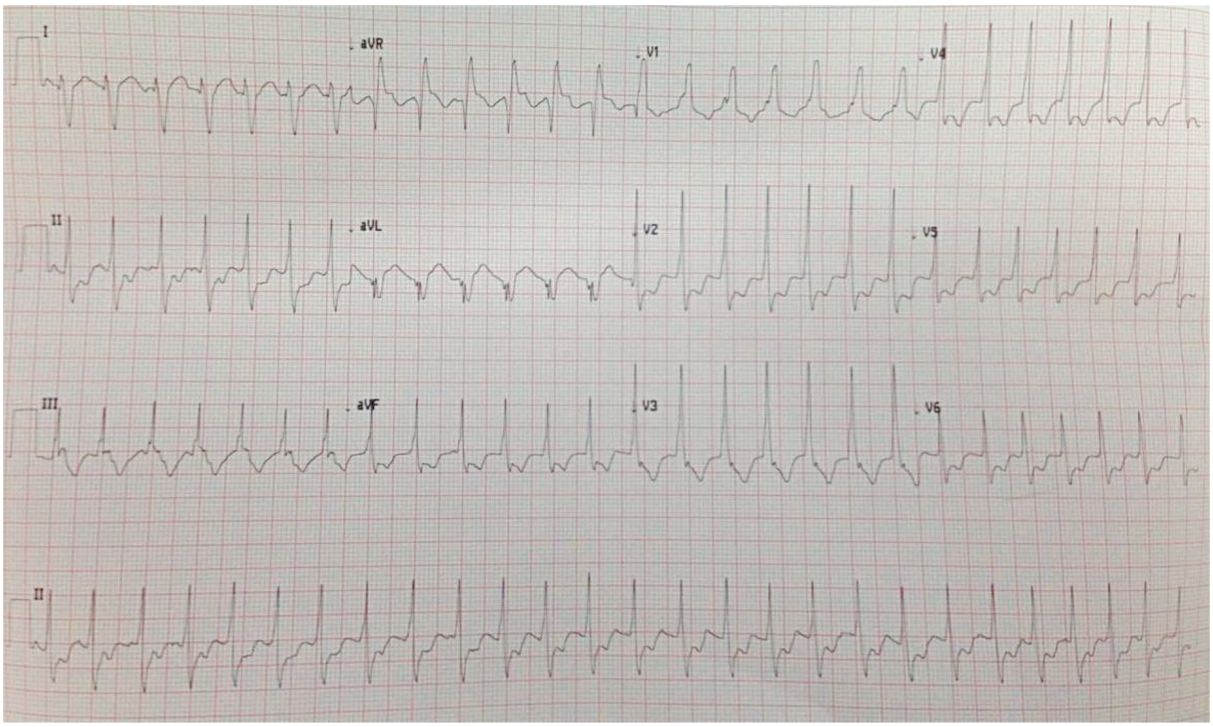

Figure 11 Supraventricular tachycardia (SVT) with aberrant conduction

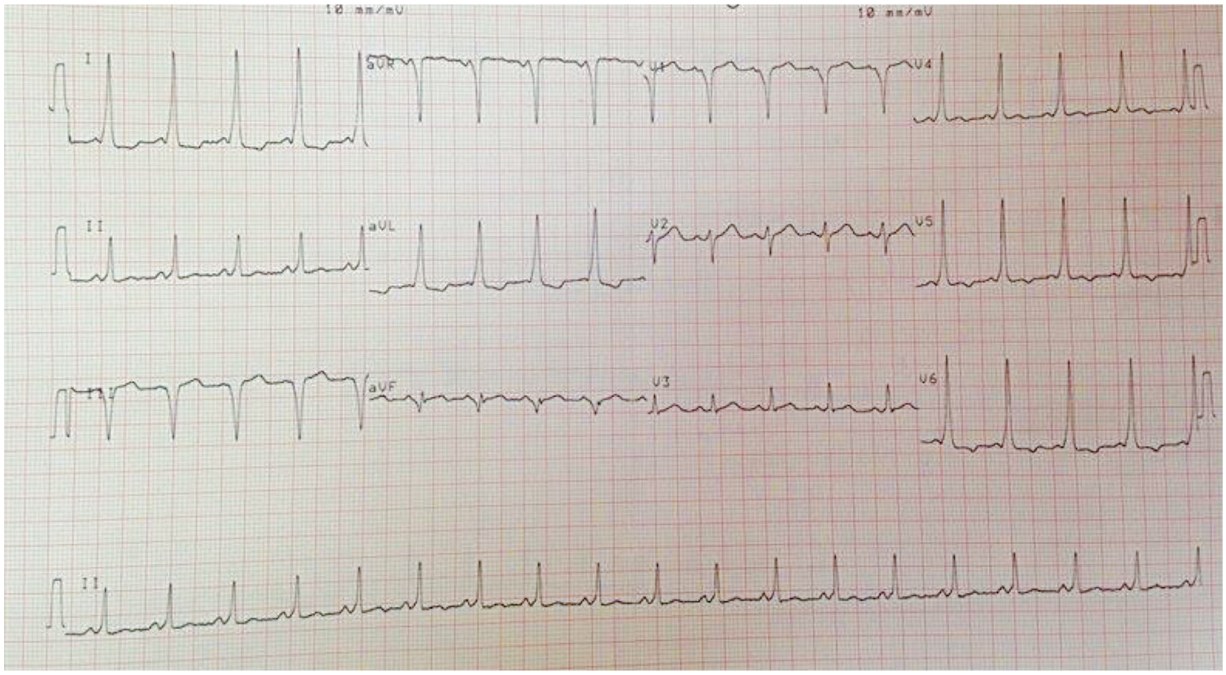

Figure 12 Wolf Parkinson White (WPW) syndrome – Characterized by short P-R interval and slurring of the ascending limb of the R wave, also known as delta wave suggestive of aberrant conduction pathway from atria to ventricle.

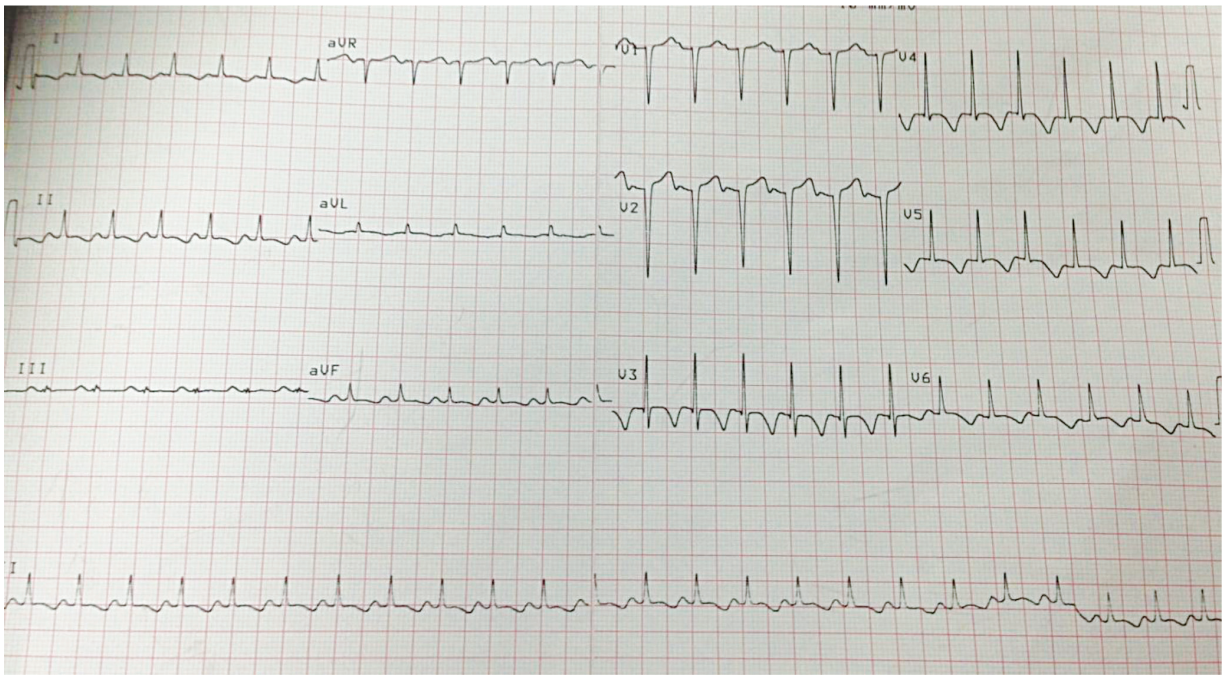

Figure 13 Atrial tachycardia

Ventricular Arrhythmias

They arise from ectopic beats in the ventricles coming successively more than 3 beats considered as runs of ventricular tachycardia. They may come in duplex or triplex and also may be multifocal.

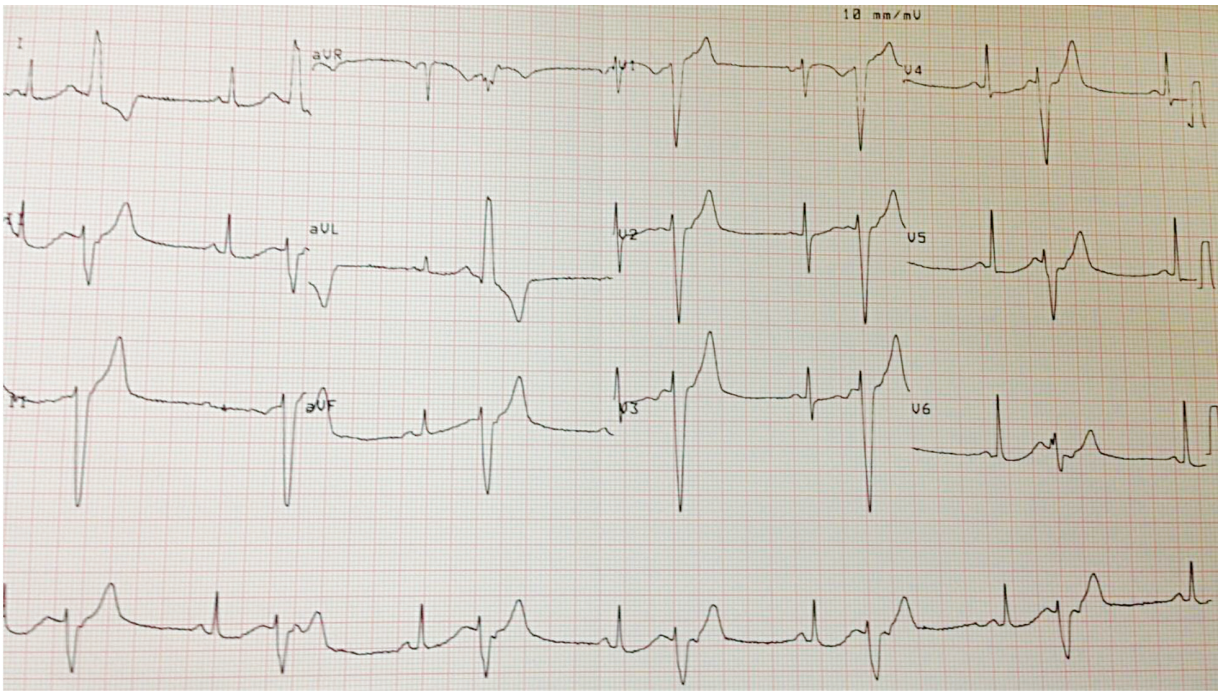

Figure 14 Ventricular bigeminy – ventricular premature beat which is broad and bizarre coming prematurely followed by a pause, featured by a T wave which always opposite to the main QRS complex. This complex may or may not be preceded by P wave. When such beats are coming, alternating with normal sinus rhythm, it is called ventricular bigeminy.

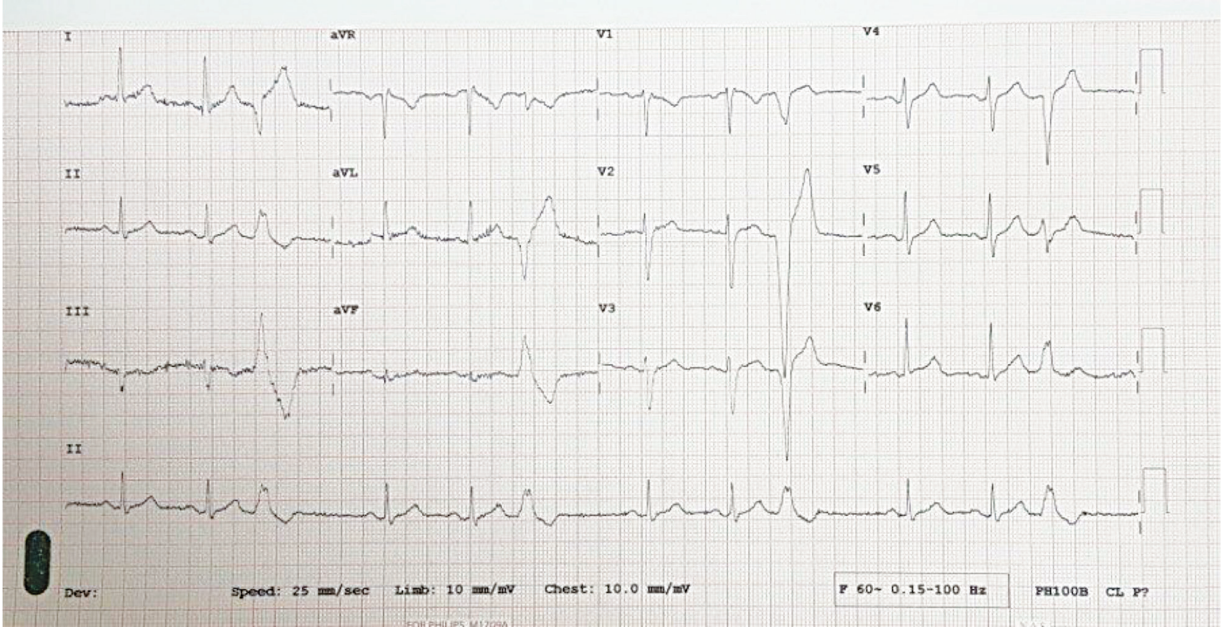

Figure 15 Ventricular trigeminy – It is characterized by ventricular premature complex (VPC) following two successive sinus rhythm. Since the configuration of all VP beats, is same in a given lead, it is called multiple unifocal VP beats when the configuration of the VP beats are changing, it is called multifocal VP beats.

Ventricular bigeminy and trigeminy are commonly seen in IHD, hypertensive heart diseases, congenital heart diseases, drug induced, electrolyte imbalances and excessive smoking, tea or coffee consumption.

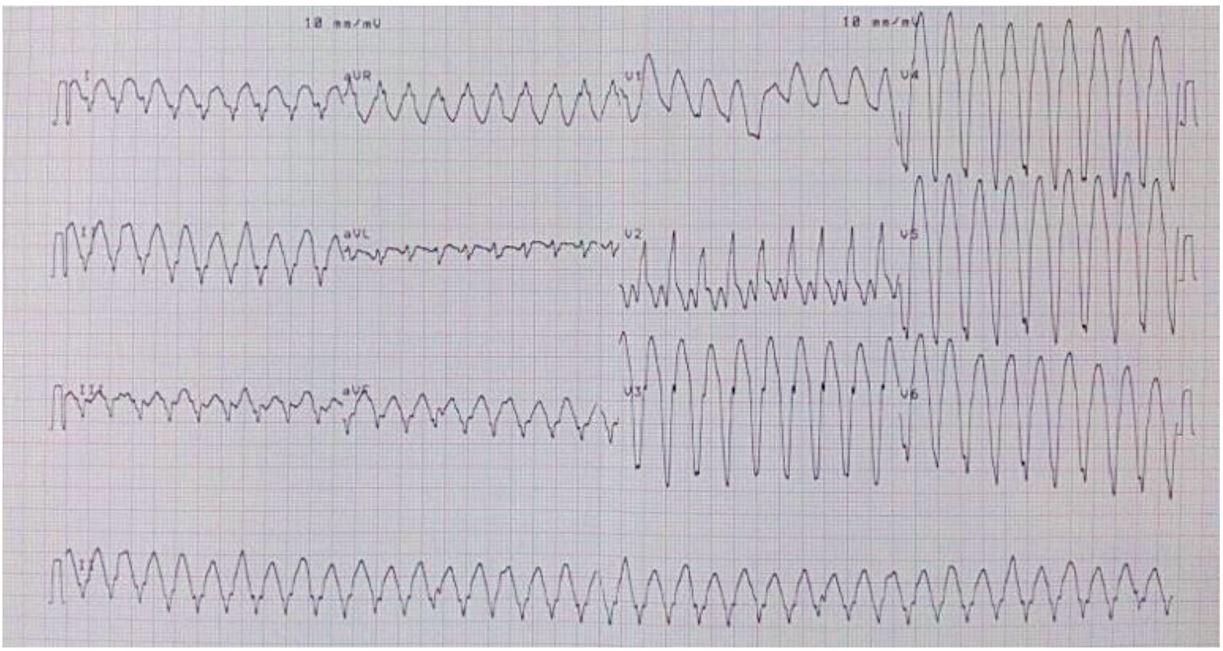

Figure 16 Ventricular tachycardia

Clinically, a serious condition causing faster haemodynamic deterioration with ECG findings of ventricular rate ranging 150 – 200 with broad bizarre QRS complex with changing R-R interval and changing QRS width with polymorphic nature. It may be precipitated by a ventricular premature beat. It should be differentiated from atrial fibrillation with conduction defect.

Brady-arrhythmias

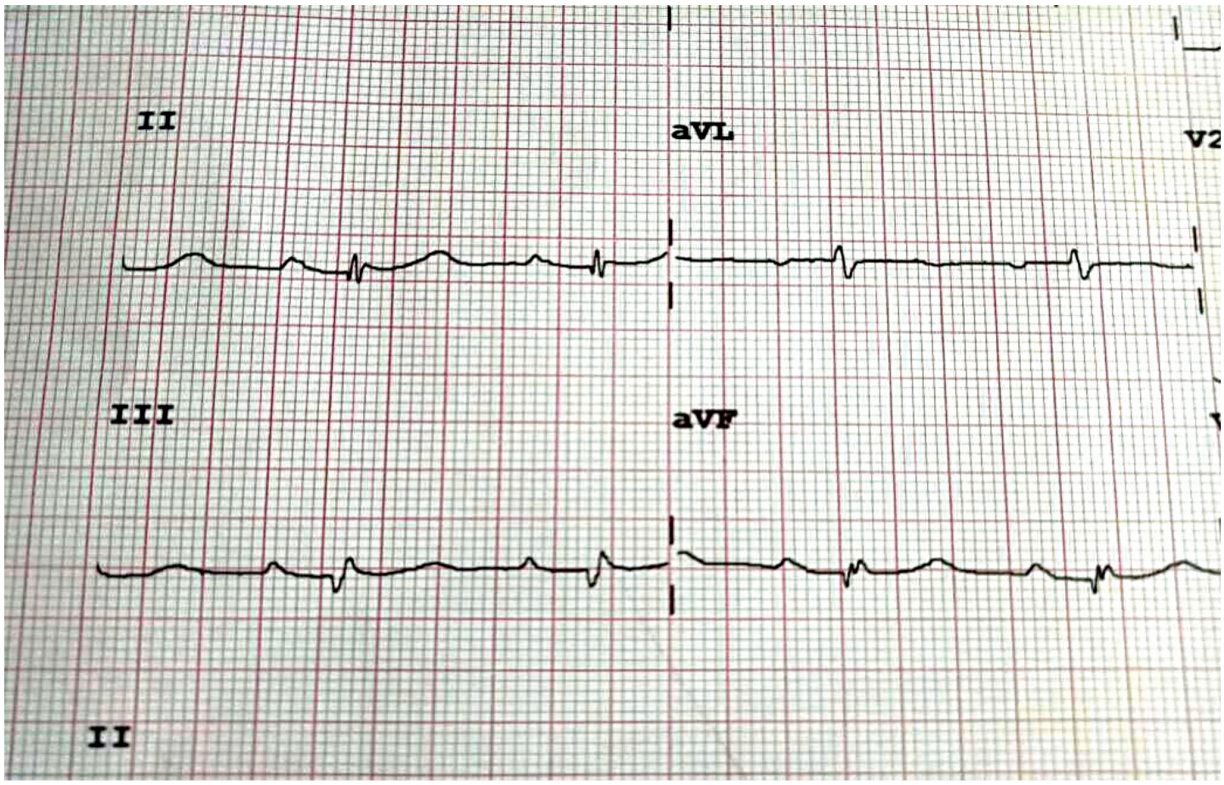

Figure 17 1st degree heart block characterized by prolonged P-R interval more than 0.2 seconds. It is commonly seen in drug induced and degenerative diseases of the conducting system.

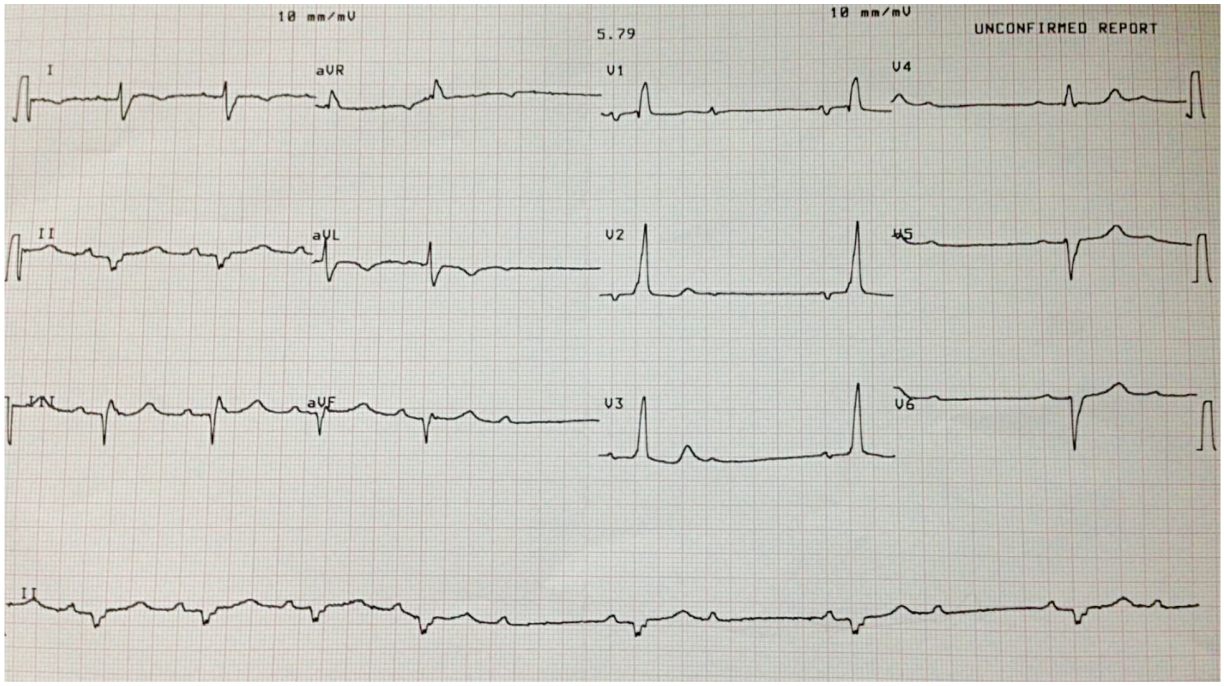

Figure 18 2nd degree heart block – type II, commonly seen with fixed blocks, either every second or third P wave is blocked.

In type I Wenckebach phenomenon, the P-R interval is gradually prolonged in successive beats till one P wave is blocked and the same cycle repeats. Normally seen in rheumatic, ischaemic and hypertensive heart diseases and cardio-myopathies and drug induced.

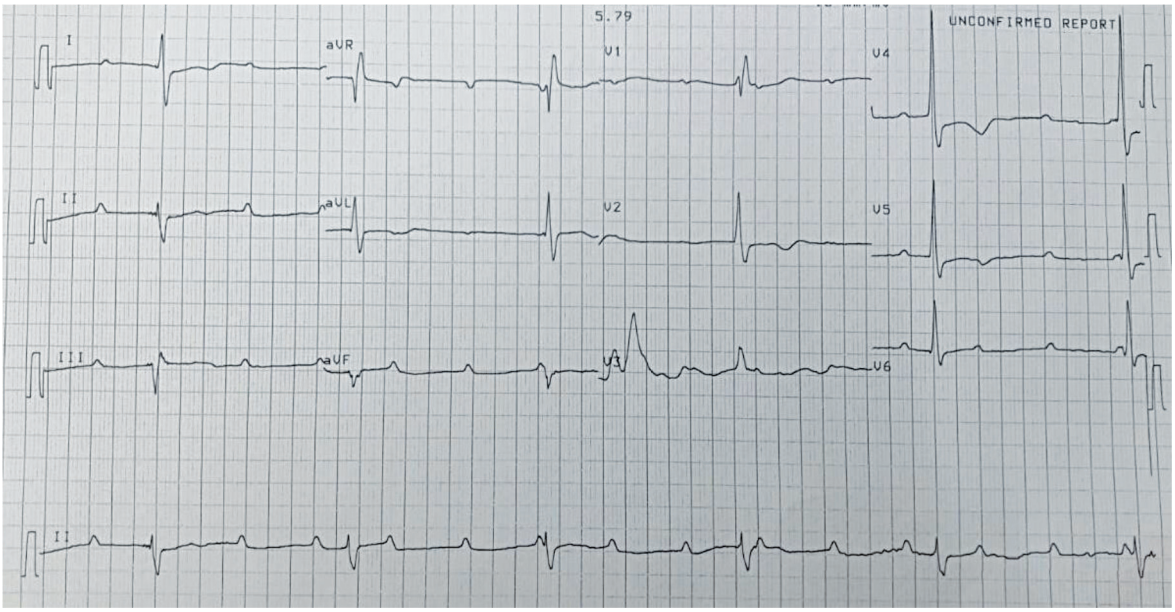

Figure 19 Complete heart block. Also called 3rd degree heart block

P wave is marching through the QRS, no relationship between atrial and ventricular contractions (independently). Commonly seen in IHD, cardio-myopathies, drug induced and electrolyte imbalance.

Pacemaker

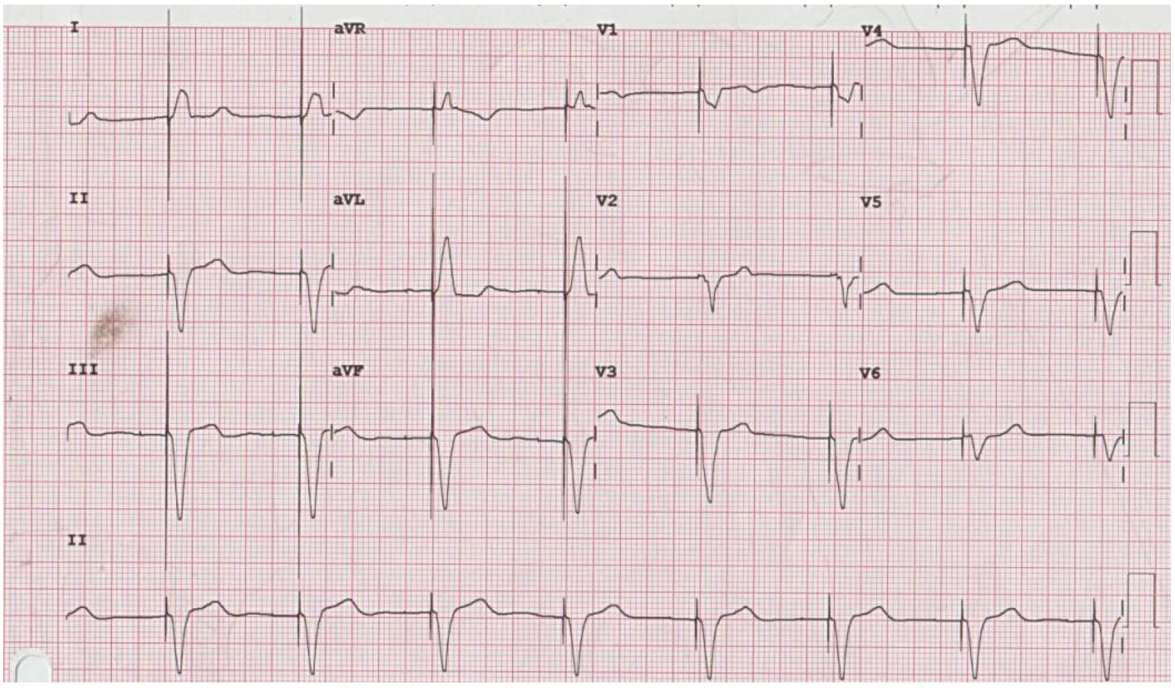

Figure 20 Ventricular pacemaker; indication complete heart block and tri-fascicular block and congenital complete heart block.

Bundle Branch Block

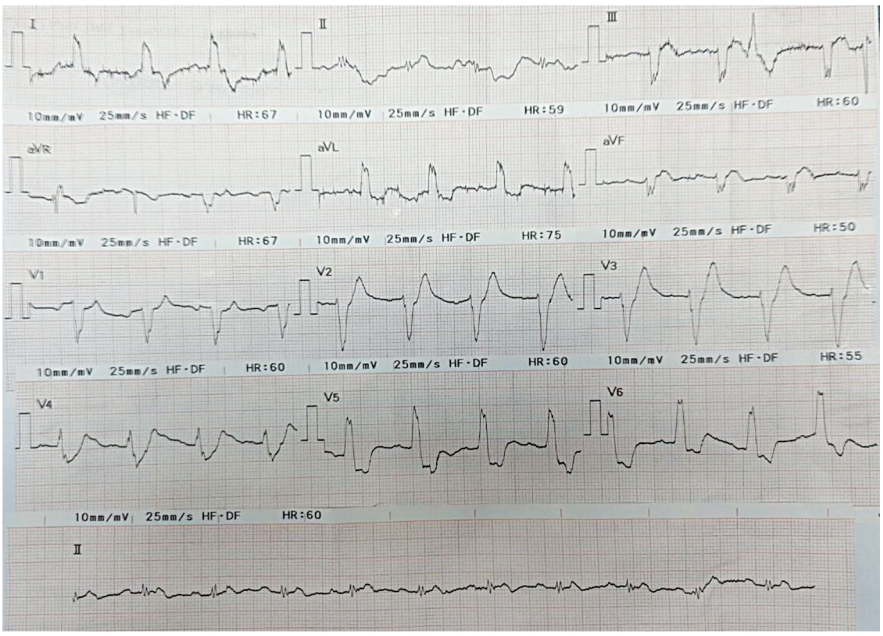

Figure 21 Left bundle branch block: Broad QRS complex with tall R waves in V_5 and V_6 and slurred S wave in V_1 and V_2.

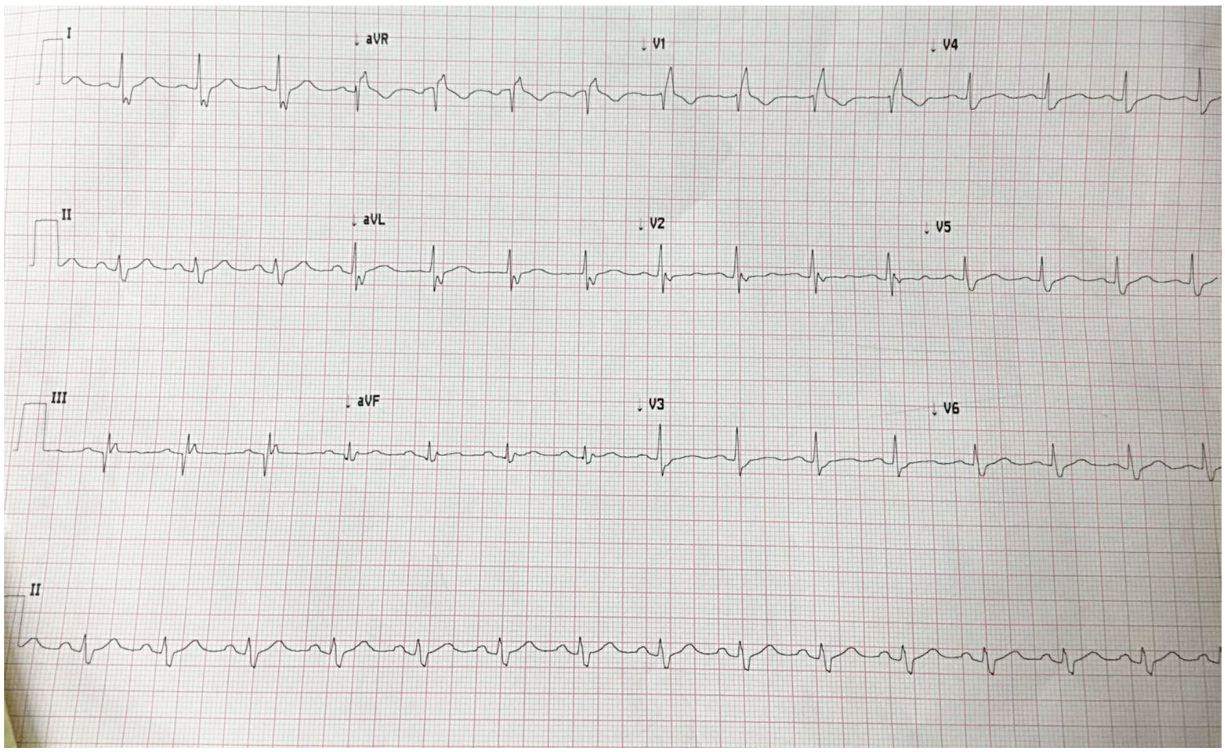

Figure 22 Right bundle branch block: broad QRS, tall R wave in V_1 and slurred S wave in V_5 and V_6 and L_1. It is seen in acute myocardial infarction

ST Segment Abnormalities

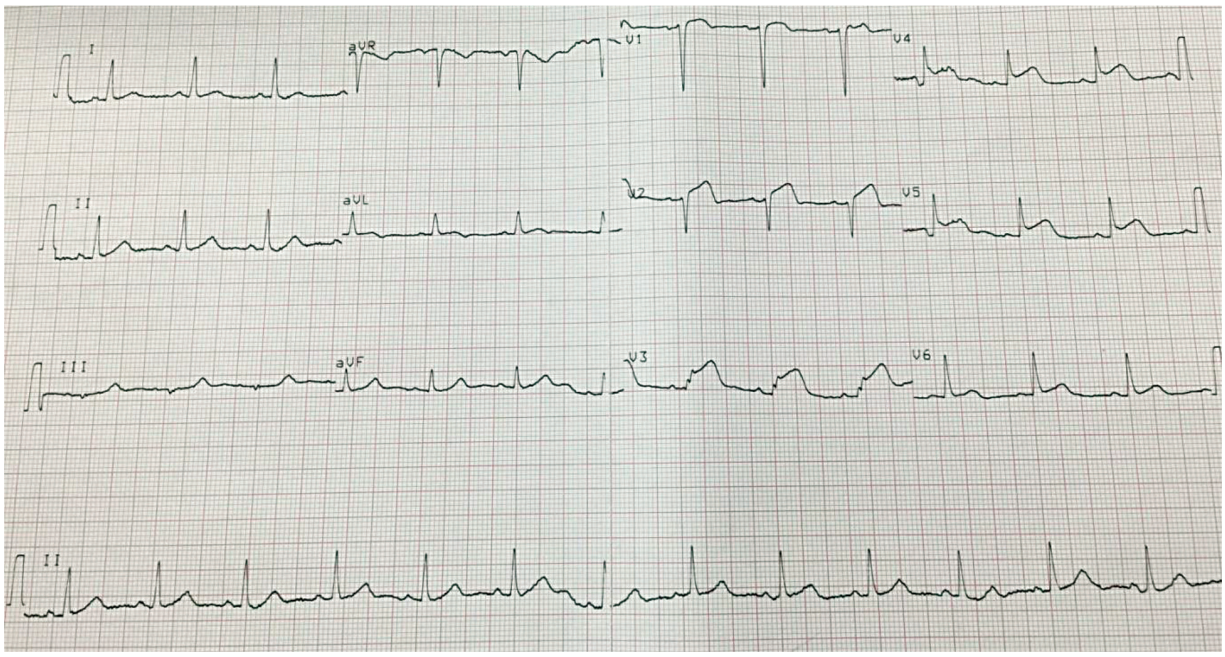

Figure 23 Acute anterior wall infarction characterized by marked ST elevation in V_1, V_2, V_3, V_4 and V_5.

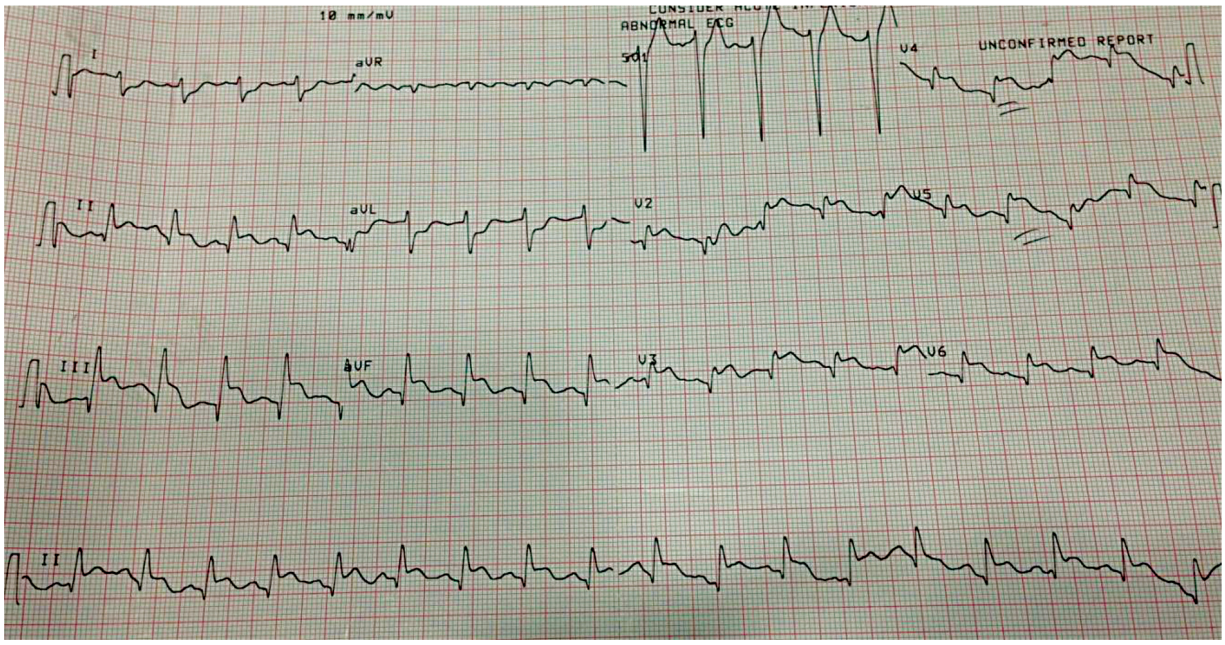

Figure 24 Acute inferior wall infarction with ST elevation in Lead II, Lead III, aVF with reciprocal ST depression in Lead 1 and aVL. Global infarct in view of ST elevation in V_1, V_2, V_3, V_4, V_5 and V_6.

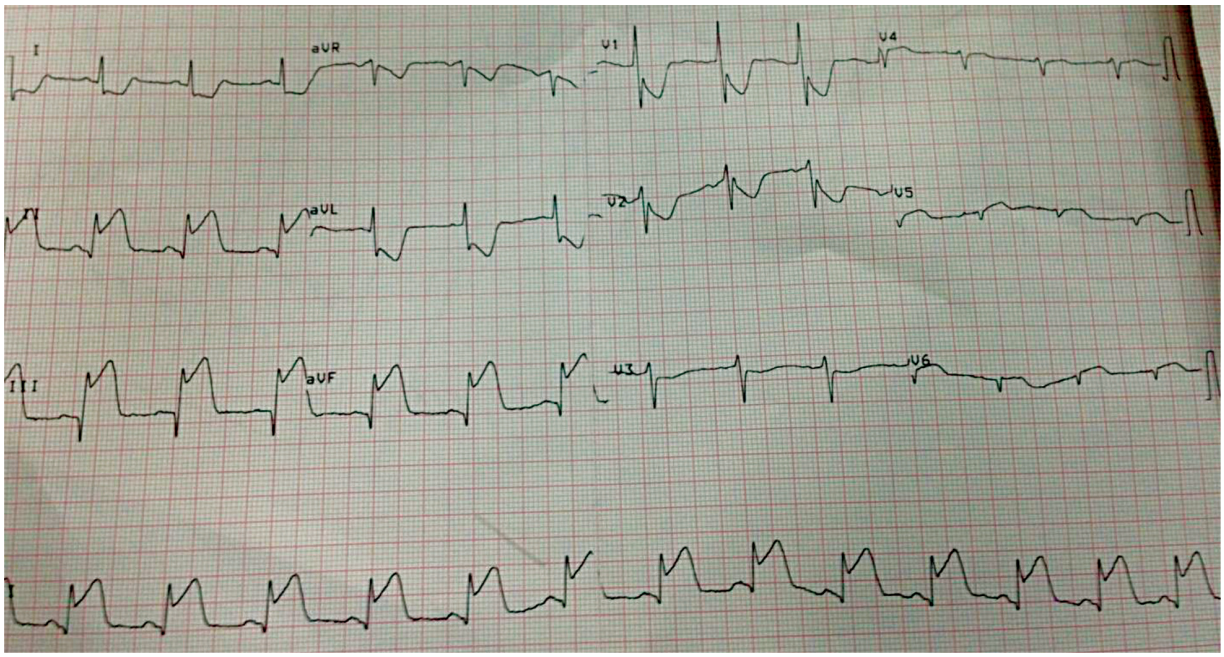

Figure 25 Acute inferio-lateral wall infarction with ST elevation in Lead II, Lead III, aVF and V_5, V_6 reciprocal ST depression in V_1, V_2, Lead I and aVL.

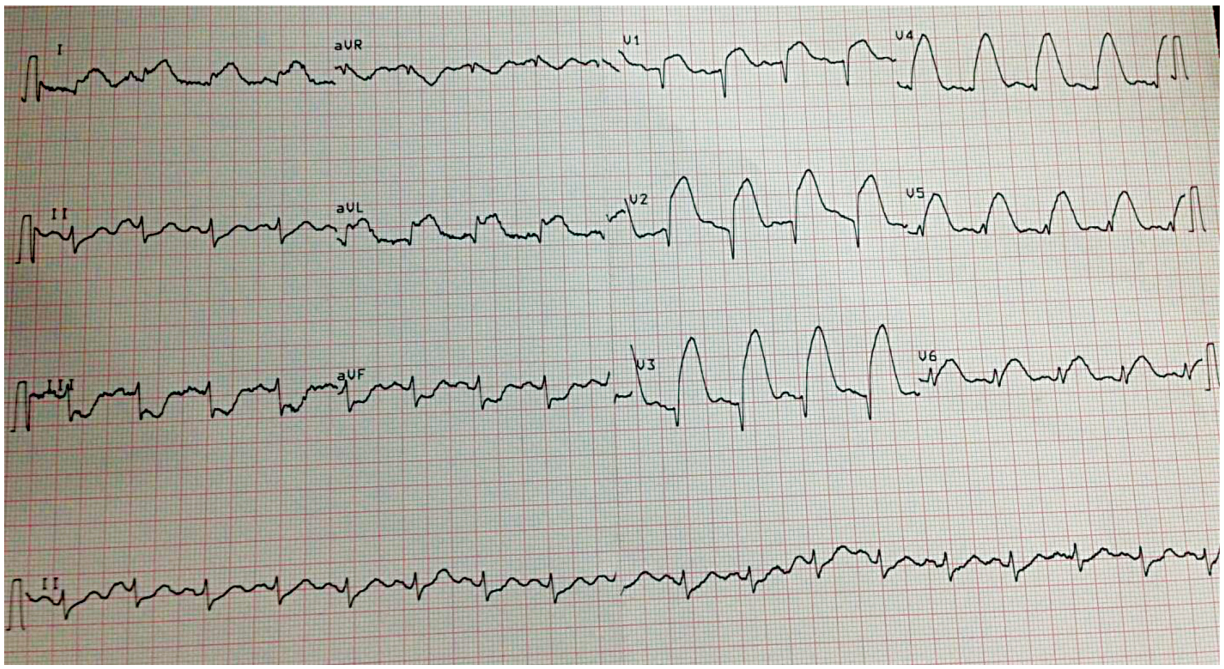

Figure 26 Extensive anterior wall infarction in the form of ST elevation from V_1 to V_6, Lead I and aVL with reciprocal ST depression in inferior wall Lead II, Lead III and aVF.

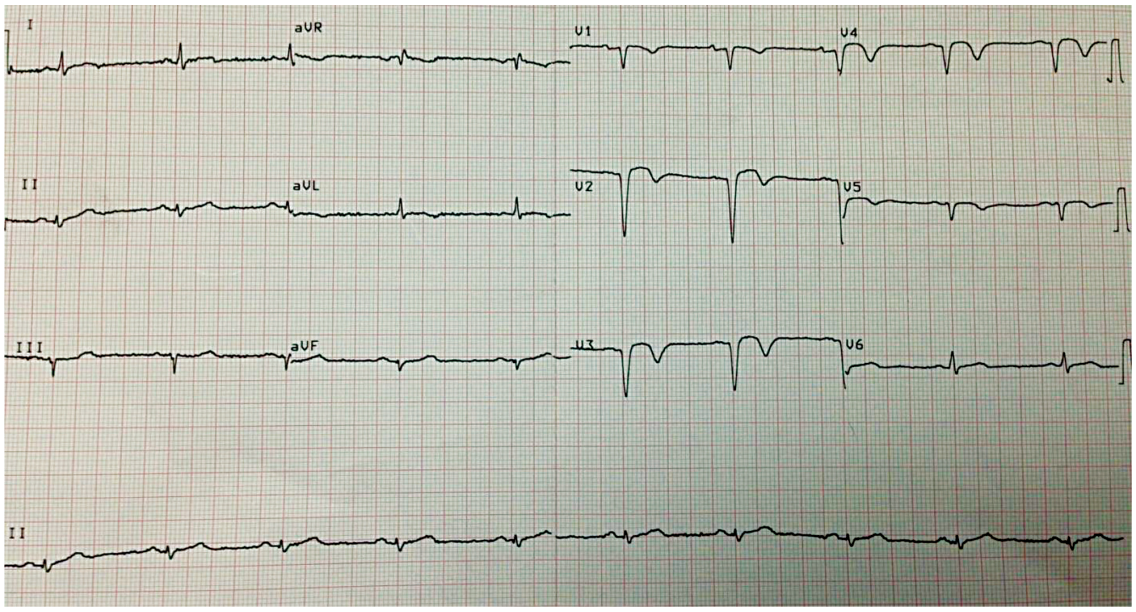

Figure 27 Healed antero-septal infarct in the form of deep Q waves in V_1, V_2 and V_3 with ST segment almost normalizing with T inversion in the same area.

The Cardiac Axis

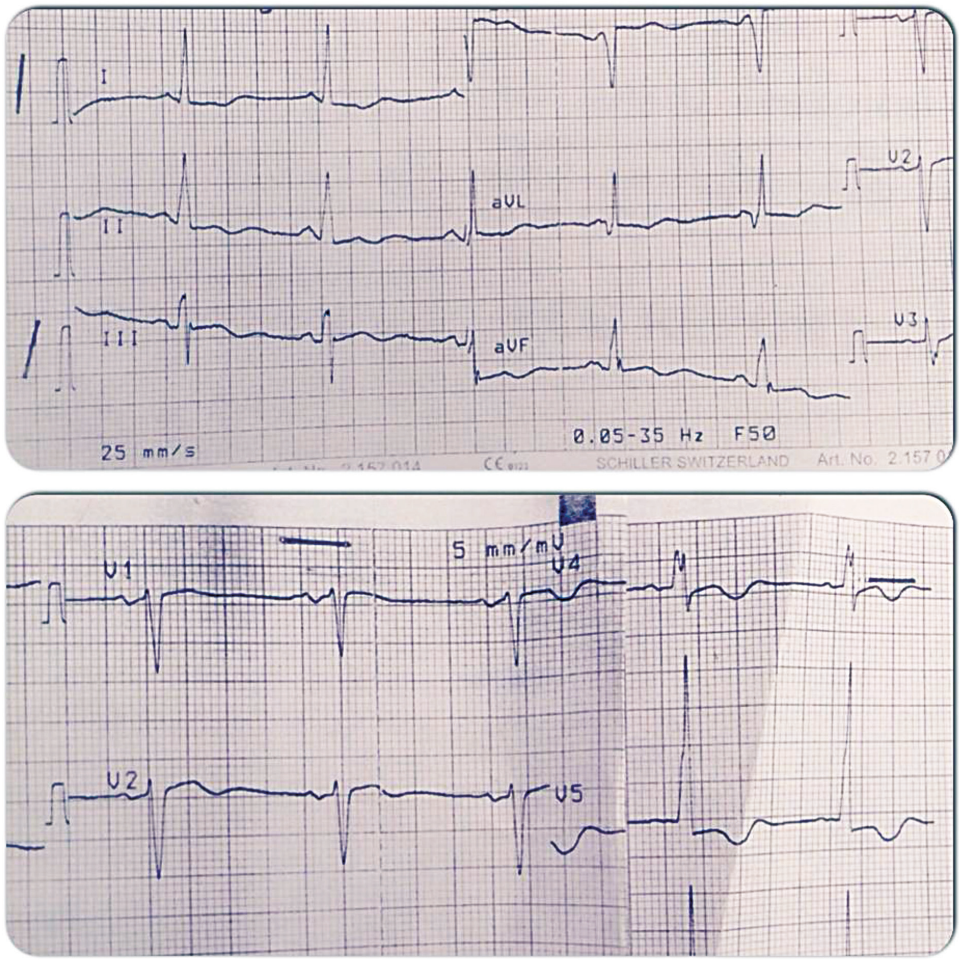

Figure 28 Normal axis – Both Lead 1 and aVF are positive complexes.

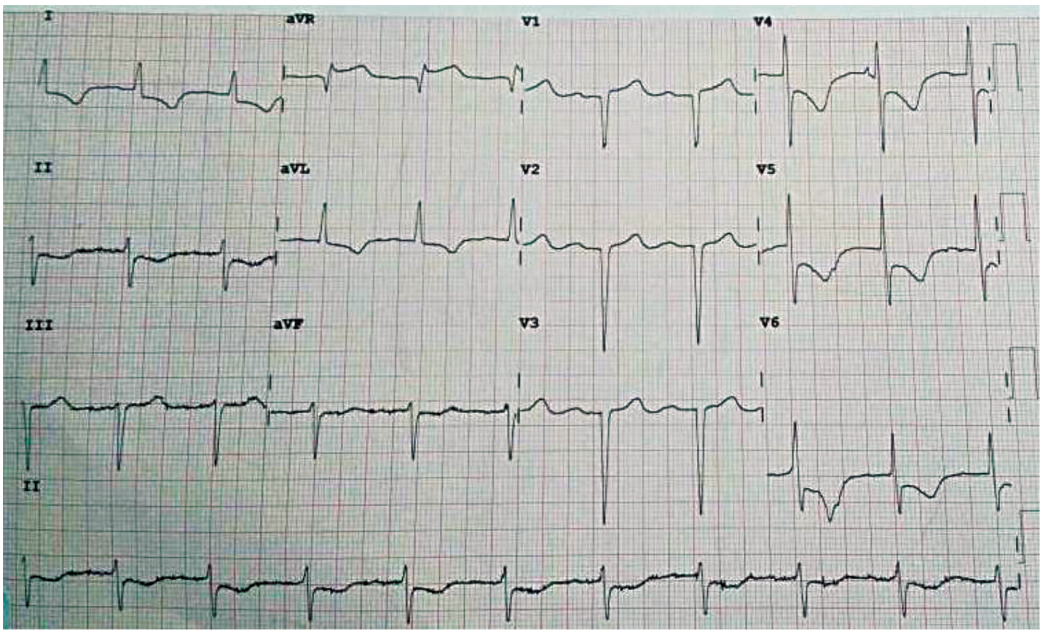

Figure 29 Left axis deviation: aVF negative, Lead 1 positive, can be seen in left anterior hemi-block and hypertensive heart diseases.

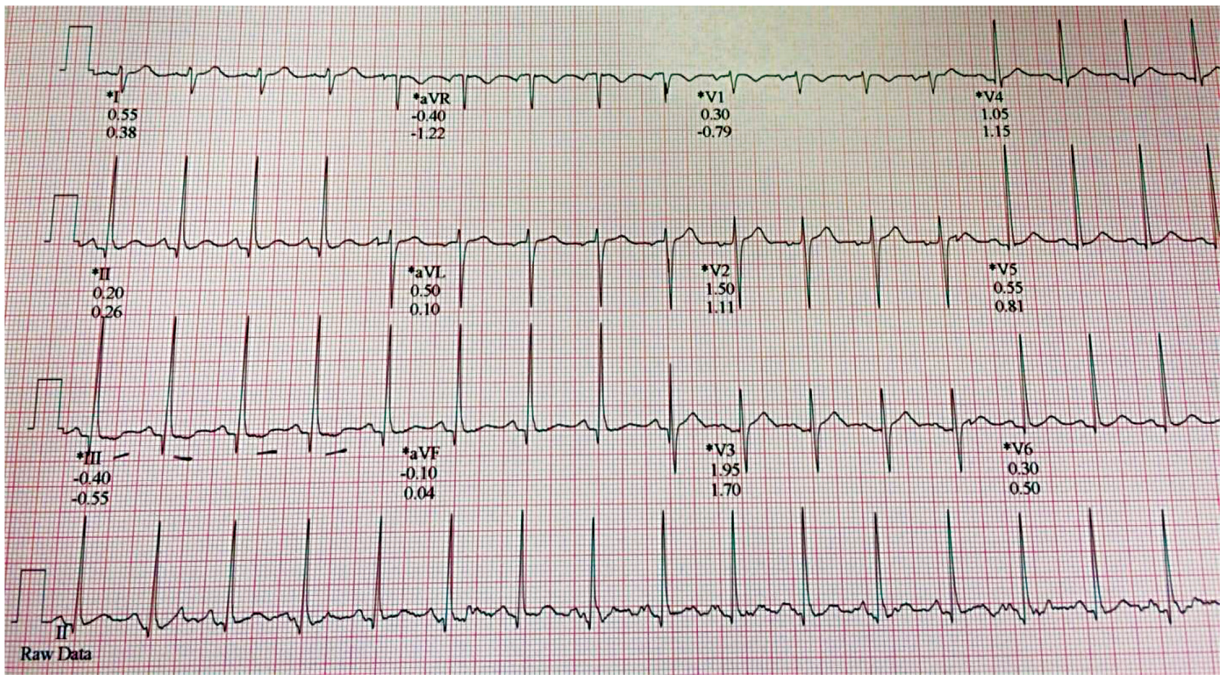

Figure 30 Right axis deviation: Lead 1 negative, aVF positive – right axis deviation is commonly seen in right ventricular hypertrophy, dextrocardia, technical dextrocardia, left posterior hemi-block.

CONCLUSION

An over-all discussion of normal ECG, 12 leads and 12-lead ECG records were discussed. Along with the normal routinely-met abnormal clinical ECG such as atrial fibrillation, ventricular tachycardia, conduction block, arrhythmias and axis deviation cases were presented. This article shall be an educational series that will be useful to every undergraduate medical student for their learning and reflection.

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