DISSERTATION ON

"A STUDY ON THE CORRELATION OF QRS AMPLITUDE IN ECG IN ST ELEVATED MYOCARDIAL INFARCTION TO ITS OUTCOME AND EXTENT OF VESSEL INVOLVEMENT IN CORONARY ANGIOGRAM"

Dissertation submitted to

THE TAMIL NADU DR MGR MEDICAL UNIVERSITY

In partial fulfilment of the requirement

For the award of the degree

M.D. GENERAL MEDICINE - BRANCH I

Submitted by

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Dr. R. ARAVIND

GOVERNMENT KILPAUK MEDICAL COLLEGE,

INSTITUTIONAL ETHICS COMMITTEE

Reg.No. ECR/1385/Inst/TN/2020

CERTIFICATE OF APPROVAL

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PRINCIPAL INVESTIGATOR	: DR. ARAVIND R
DESIGNATION	: IIND YEAR POST GRADUATE
DEPARTMENT:	: DEPT OF GENERAL MEDICINE GOVT. ROYAPETTAH HOSPITAL, GOVT. KILPAUK MEDICAL COLLEGE, CHENNAI-10.

The request for an approval from the Institutional Ethics Committee (IEC) was considered on the IEC meeting held on **29/04/2022** at Government Kilpauk Medical College, Chennai-10.

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DECLARATION

I solemnly declare that this dissertation "**A STUDY ON THE CORRELATION OF QRS AMPLITUDE IN ECG IN ST ELEVATED MYOCARDIAL INFARCTION TO ITS OUTCOME AND EXTENT OF VESSEL INVOLVEMENT IN CORONARY ANGIOGRAM**" was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Prof. Dr. P.PARANTHAMANM.D,** Professor and HOD of General Medicine, Department of General Medicine, Government Kilpauk Medical College and Hospital, Chennai. This dissertation is submitted to **The Tamil Nadu Dr. M.G.R.Medical University, Chennai** in partial fulfilment of the Universityregulations for the award of the degree of **M.D. Branch I (General Medicine)**.

DR. R. ARAVIND

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PLAGIARISIM CERTIFICATE

This is to certify that this dissertation work titled "**A STUDY ON CORRELATION OF QRS AMPLITUDE IN ECG IN ST ELEVATED MYOCARDIAL INFARCTION TO ITS OUTCOME AND EXTENT OF VESSEL INVOLVEMENT IN CORONARY ANGIOGRAM**" of the candidate **Dr.R.ARAVIND** with registration number – **200120102003** for the award of M.D in the branch of **GENERAL MEDICINE**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **12** percentage of plagiarism in the dissertation.

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ABSTRACT

OBJECTIVE

Primary objective :

To assess the value of QRS amplitude in STEMI patients as a prognostic indicator with the extent of Vessel involvement in coronary angiogram.

Secondary objectives:

To assess outcome in terms of ECHO findings with respect to Ejection fraction.

To assess Age and Sex distribution

To assess correlation with respect to Smoking, Alcoholism, Comorbidites - Diabetes and Hypertension.

METHOD

Study design: Comparative study

Period of Study: May 2022 to November 2022

Duration of Study: 6 months

Place of Study: Government Royapettah Hospital, Chennai-600014 and Government Kilpauk Medical College, Chennai - 600010

RESULT

Our study reports significant differences in the QRS amplitude with a correlation between Ejection Fraction (EF%) and vessel obstruction. The comparison yielded with Pearson correlation stated a sig. (2-tailed) value of 0.031 for EF% and a significant difference with vessel obstruction % with angiography (p-value <0.0001)

CONCLUSION

ECG is one of the predictive tools which can be used to detect the outcome of the disease, Patients with a low QRS amplitude score are more prone to complications and longer hospital stays. Therefore, it is essential to correlate the reciprocal changes in the ECG and angiography for a better clinical assessment and to provide an adequate therapeutic outcome. A lower QRS amplitude score has also affected the ejection fraction (EF%) and can be correlated with the % vessel obstruction. Hence, ECG can be used as an early diagnostic tool for predicting the severity of STEMI and non-STEMI disease in patients.

A STUDY ON THE CORRELATION OF QRS AMPLITUDE IN ECG IN ST ELEVATED MYOCARDIAL INFARCTION TO ITS OUTCOME AND EXTENT OF VESSEL INVOLVEMENT IN CORONARY ANGIOGRAM

TABLE OF CONTENTS

ABBREVIATION

MI	-	Myocardial infarction
ACS	-	acute coronary syndrome
ECG	-	electrocardiogram
STE	-	ST-elevation
STEMI	-	ST-elevation myocardial infarction
NSTEMI	-	non-ST-elevation myocardial infarction
LV	-	left ventricular
ESVI	-	end-systolic volume index
IS	-	infarct size
EF	-	Ejection Fraction
AHA	-	American heart association
VF	-	ventricular fibrillation
BF	-	Bifascicular block
SA	-	sinoatrial

AV	-	atrioventricular
ECHO	-	Echocardiogram
WD	-	Wave Doppler
DI	-	Doppler Imaging

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INTRODUCTION

Myocardial infarction is one of the most frequently encountered reasons for hospital admission and is commonly seen in all populations worldwide. Theoretically, infarction of the myocardial muscle can be detected based on certain pathological features like coagulation necrosis, causing the loss of muscle cells (myocytes). When such a pathological change occurs, it triggers inflammation, leading to fibrosis and healing with a scar. The 12-lead electrocardiogram is one of the essential diagnostic factors that can be used to detect acute coronary syndrome (ACS). [1]

The clinical approach to a patient suspected of acute coronary syndrome consists of detailed history taking, electrocardiogram (ECG) changes, cardiac biochemistry evidence and imaging. The reliability of each of these modalities as a diagnostic tool depends on multiple factors, the most significant being the window period between the infarction and the time the patient seeks medical attention. [1]

The current approach to patients presenting with typical or atypical features of myocardial ischemia or infarction starts with making the provisional diagnosis of the acute coronary syndrome. Then, depending on the changes seen on the twelve lead ECG, the acute coronary syndrome is classified into ST-elevation

myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI). [1]

ST-elevation myocardial infarction (STEMI) is a clinical syndrome characterised by the symptoms of ischemia in correlation with the persistent electrographic ST-elevation (STE) and subsequent release of biomarkers of myocardial necrosis. STE elevations have been significant for detecting acute coronary artery occlusion with collateral circulation, which can be used to assess myocardial injury and infarction that requires immediate reperfusion therapy. [2]

Improving the PCI techniques and adjunctive pharmacological therapy can reduce mortality rates compared to thrombolytic therapy. [3] Despite major leaps in the diagnostic tools and treatment of myocardial infarction, ST-segment elevation MI persists as a leading cause of ill health in the developed and developing world. The prevalence of coronary disease and infarction is on the rise in developing countries, with further worsening of adverse cardiovascular events due to disadvantages like inadequate primary prevention policies and limited availability of medical assistance.

The meteoric advances in acute coronary care and resuscitation since the twentieth century have led to a considerable decline in mortality and morbidity

rates from STEMI. In the early twentieth century, therapy was centred around passive observation and monitoring rather than active intervention. However, significant advances have opened the gates to the current reperfusion therapy, which along with intensive hemodynamic monitoring, has improved the standards of acute coronary care and emergency management. As a result, the approach to ST elevation MI is increasingly leaning towards practice guidelines and evidence-based medicine.

Extensive research has been done in the field of cardiovascular medicine focused on ST-elevation myocardial infarction and the complications surrounding it. One particular area of interest in recent studies appears to be the study of the extent of infarction over imaging modalities and its correlation with various events in the course of infarction. Other parameters gaining importance are non-traditional ECG changes like reciprocal changes, QRS sloping, etc. Myocardial infarction mortality and probability of death can be substantially determined by the cumulative left ventricular (LV) structural and functional derangement. The end-systolic volume index (ESVI) and infarct size (IS) are alternatives used to assess the Ejection Fraction (EF). [1] [3] [5]

The simplest and oldest diagnostic tool for myocardial infarction is the 12-lead electrocardiograph. In daily practice, even though the ECG remains an important test in the diagnosis and detection of the progression of the disease,

there remains ample potential for its role as a prognostic marker. The fundamental advantage that can be gained by realising this potential is the time it saves for immediate intervention. In addition, it can serve as an instant and cost-effective method for risk assessment instead of waiting for biochemical and angiography results. This study explores the possibility of ECG being a prognostic marker in terms of left ventricular outcome and extent of vessel involvement.

LITERATURE REVIEW

History – The word electrocardiography is derived from the Greek terms electro, cardio, and graph, which respectively mean "electrical activity," "heart," and "to write". [6] Willem Einthoven recorded the ECG with a string galvanometer in 1901, the first therapeutically useful innovation. [7] He also designated the letters P, Q, R, S, and T to the deflections detected in ECGs. [8]

Later, Lewis continued Einthoven's work by writing books and papers about the electrophysiology of the heart after completing many experiments to examine dysrhythmias. [9] In his writings, he addressed the advantages of ECG in the healthcare profession. After conducting some experiments, American doctors Williams and James concluded that the ECG offered "a new point of view of the normal and pathological functioning of the heart." [9]

The electrocardiograph, which for a long time was known as the string galvanometer, underwent a series of modifications over time to become a helpful portable device anywhere. [10] The internal cardiac defibrillator and the Holter monitoring system can use the same fundamental concept because of current technology. [9]

ST ELEVATION MYOCARDIAL INFARCTION (STEMI)

Definition - According to the American heart association, evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia is when the term acute myocardial infarction (MI) should be used. [11] The diagnosis of MI is made if any one of the following criteria is fulfilled under these conditions: (Table 1)

TABLE 1 – DEFINITION OF MYOCARDIAL INFARCTION. [11]

Definition of myocardial infarction

Criteria for acute myocardial infarction

The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischaemia. Under these conditions any one of the following criteria meets the diagnosis for MI:

- Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:
  - Symptoms of ischaemia.
  - New or presumed new significant ST-segment–T wave (ST–T) changes or new left bundle branch block (LBBB).
  - Development of pathological Q waves in the ECG.
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
  - Identification of an intracoronary thrombus by angiography or autopsy.
- Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.
- Percutaneous coronary intervention (PCI) related MI is arbitrarily defined by elevation of cTn values (>5 x 99th percentile URL) in patients with normal baseline values (≤99th percentile URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischaemia or (ii) new ischaemic ECG changes or (iii) angiographic findings consistent with a procedural complication or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.
- Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarker values with at least one value above the 99th percentile URL.
- Coronary artery bypass grafting (CABG) related MI is arbitrarily defined by elevation of cardiac biomarker values (>10 x 99th percentile URL) in patients with normal baseline cTn values (≤99th percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Criteria for prior myocardial infarction

Any one of the following criteria meets the diagnosis for prior MI:

- Pathological Q waves with or without symptoms in the absence of non-ischaemic causes.
- Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischaemic cause.
- Pathological findings of a prior MI.

Clinical Features - The development of myocardial infarction is divided into three stages. The first stage is an acute stage that can be seen anywhere from a few hours to seven days. The second phase is the healing stage, lasting between seven and twenty-eight days. This is followed by the healed phase, which lasts more than twenty-nine days. [11] The continuous phase is seen in which the healed phase begins and lasts more than 29 days. [11]

Events like stress (physical or mental), sickness, or surgery appear to be catalysts for ST-elevation myocardial infarction in up to 50% of instances. The complaint that is most commonly seen is pain. Patients report the distinctive pain of MI as being heavy, crushing, and squeezing. [11]

Usually, the pain originates underneath the xiphoid process and the epigastrium and spreads to various body parts, including the occipital region and the level of the umbilicus. Other symptoms include vomiting, sweating, weakness, worry, and concern about dying. Those with diabetes and the elderly are more likely to experience silent infarction. [11]

Patients may seem nervous when being examined. You could notice pallor, diaphoresis, and cold hands and feet. Blood pressure and heart rate may be normal during the first hour after infarction. Still, roughly 50% of patients with inferior infarcts show parasympathetic hyperactivity, while 25% of patients with anterior infarcts may exhibit sympathetic hyperactivity. [11]

The apical impulse could be hard to feel while palpating. In the first few days of sickness, aberrant systolic pulsations may develop in people with anterior infarcts in the peri-apical region. Third and fourth heart sounds, paradoxical splitting of S2, and decreased intensity of S1 are all potential indicators of ventricular failure. Pericardial rub and the mid or late-systolic murmur are two possible adventitious noises. Reducing the carotid pulse, which represents the stroke volume, is possible. [11]

Complications

Ventricular Dysfunction - Modification of the left ventricle after STEMI signals the onset of heart failure, which may manifest months or years after the infarct. After a STEMI, the left ventricle enlarges due to a combination of acute modifications that prolong the infarcted region and subsequent changes that stretch the non-infarcted area (figure 1). [12]

As shown by increased dilatation in cases of MI affecting the anterior wall and apex with a worse prognosis, factors including the location and size of the infarct impact the chamber's total dilatation. The most crucial factor in predicting death in STEMI is LV failure. Diastolic or systolic dysfunction may exist. Pulmonary venous hypertension and congestion are two symptoms of diastolic dysfunction. [12]

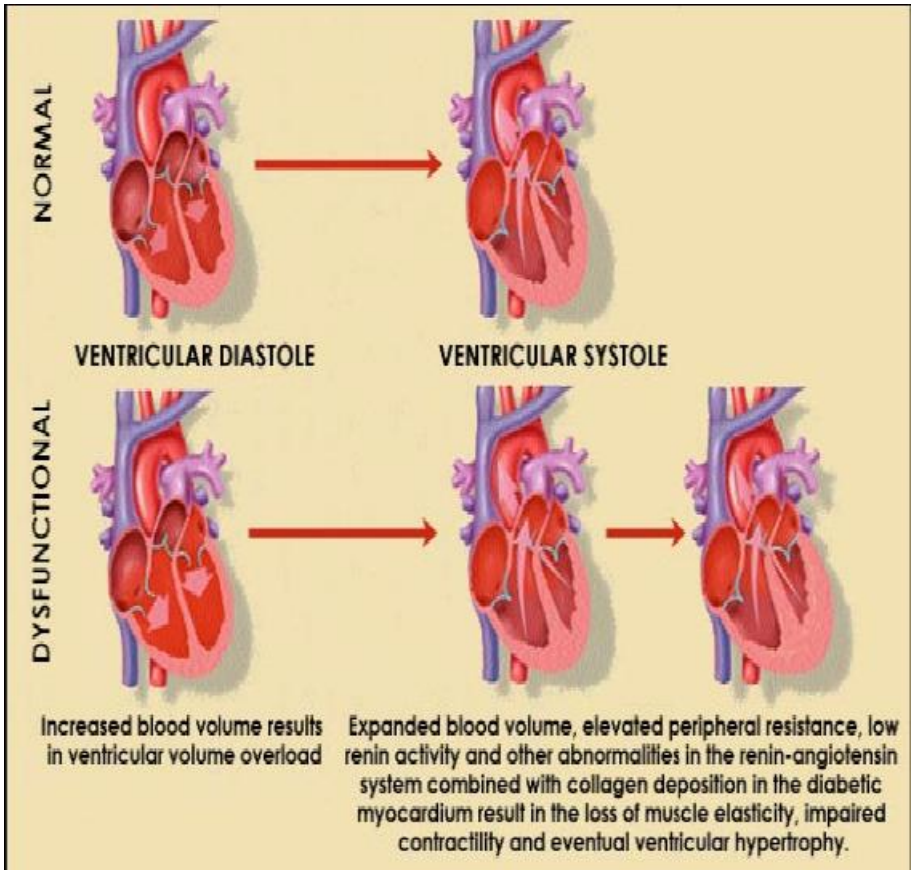


FIGURE 1 – COMPARISON OF NORMAL AND DYSFUNCTIONAL HEART.

Systolic heart failure – ventricular function alteration

Many patients with heart disease might have signs and symptoms that hint at the ventricle's impaired systolic performance. The primary cause of this cardiovascular depression is a dysfunctional or insufficient heart pump mechanism, which prevents the heart from producing its maximum energy output and maintaining the mean arterial pressure. Systolic function declines

when heart failure worsens, first during physical effort and then later while at rest, limiting daily activities and negatively impacting the quality of life. [13] [14]

Systolic heart failure has a molecular and cellular foundation that includes other processes in addition to abnormalities in proteins involved in myocyte contraction or biochemical irregularities in calcium homeostasis. They consist of altered ion channel function, metabolic failure or irregularities at the mitochondrial level, structural alterations such as myocytolysis and cell loss, electrical remodelling, inflammatory changes such as fibrosis, or changes in signal transduction. [13] [14]

Understanding the pathophysiology is crucial because it affects the development of treatments that target these processes to prevent and treat left ventricular remodelling while also improving the systolic function of the heart. [15] [16]

The length and force of a myocyte with the membrane removed may be studied at the molecular level utilising tissue specimens to reveal the presence of abnormal cardiac cell activity. But due to several complicating elements, such as the heart's current stress, dysfunction brought on by heart failure, the complex structure of the ventricular chamber, remodelling, etc., this is difficult in an intact heart in a live individual. Ejection fraction measurement is the most often used indicator of heart failure. [17] This is impacted by all of the confounding factors mentioned above, making it a less-than-ideal instrument for determining the heart's fluctuating systolic capacity. [18]

The symptomatology does not necessarily correspond to the anatomical remodelling of the heart. [17] For instance, a patient presenting with class III dyspnea may have dilated cardiomyopathy and a poor ejection fraction, as can a patient with NYHA class I symptoms accompanied by MI. In both instances, increased end-diastolic volume due to the chamber's dilatation is the primary cause of the decreased EF. But a distinct factor contributes to this mechanism. [18]

In an infarction, the major disease is remodelling caused by a region of ischemia with possible normal reserve capacity, characterising the rest of the myocardium. With cardiomyopathy, the disease is extensive, and the reserve capacity is decreased. Regular workup and ejection fraction assessment may yield identical results in both situations. Invasive pressure-volume loops can be used to measure the severity of contractile dysfunction. [18]

The left ventricular function seems to be the primary indicator of STEMI prognosis. [19] (figure 2). There are two forms of left ventricular dysfunction: systolic and diastolic. Patients suffering from a myocardial infarction may experience either systolic or diastolic dysfunction. [19]

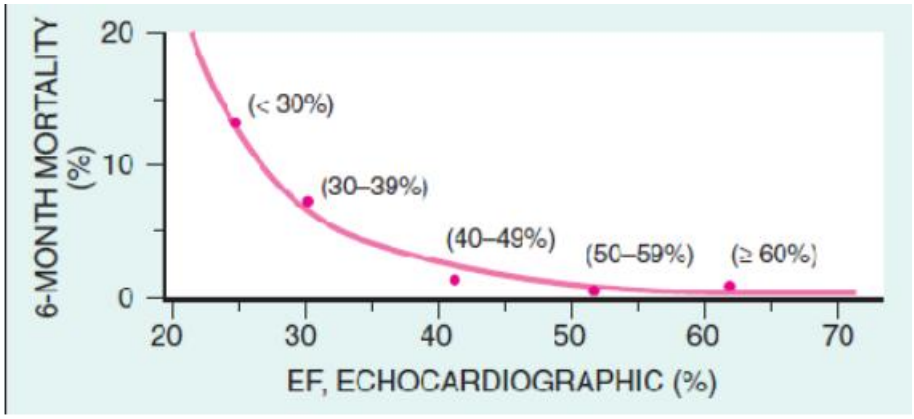


Figure 2 – Effect of LV function on survival following MI

Hemodynamic instability

Pump failure is the primary cause of death, and how severe it depends on how widespread the infarction is. Four subgroups of acute MI patients can be distinguished based on clinical manifestations, cardiac output, and wedge pressure. [19] Table 2.

Table 2 – Comparison between clinical examination and invasive monitoring.

*Based on Clinical Examination**		*Based on Invasive Monitoring*	
CLASS	**DEFINITION**	**SUBSET**	**DEFINITION**
I	Rales and S_3 absent	I	Normal hemodynamics; PCWP < 18 mm Hg, CI > 2.2
II	Crackles, S_3 gallop, elevated jugular venous pressure	II	Pulmonary congestion; PCWP > 18 mm Hg, CI > 2.2
III	I rank pulmonary edema	III	Peripheral hypoperfusion; PCWP < 18 mm Hg, CI < 2.2
IV	Shock	IV	Pulmonary congestion and peripheral hypoperfusion; PCWP > 18 mm Hg, CI < 2.2

CI = cardiac index; PCWP = pulmonary capillary wedge pressure.
*Modified from Killip T, Kimball J: Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. Am J Cardiol 20:457, 1967.
†Modified from Forrester J, Diamond G, Chatterjee K, et al: Medical therapy of acute myocardial infarction by the application of hemodynamic subsets. N Engl J Med 295:1356, 1976.

Hypovolemia

When a STEMI occurs, hypotension can develop for several reasons. Hypovolemia can result from excessive perspiration, vomiting, diuretics, or a reduction in fluid intake. Relative hypovolemia may exist without these conditions and with a normal essential vascular volume because of diminished ventricular compliance. [19]

Cardiogenic shock

Recent years have seen a decline in the prevalence of cardiogenic shock due to better medical care. 90% of patients experience this problem while being hospitalised. The most severe type of left ventricular injury is cardiogenic shock. The myocardium is typically injured, and the other body parts may have mechanical flaws, such as ruptured ventricular septal or papillary muscles or right ventricular infarction**.** [19]

Arrhythmias

Arrhythmias in MI patients are brought on by electrolyte imbalances, ANS imbalances, and delayed conduction in ischemic myocardium zones. Most deaths from arrhythmia happen within the first few hours after infarction. [19]

Ventricular Premature Beats

In STEMI, sporadic and infrequent premature ventricular depolarisations (figure 3) do not require treatment. Prophylactic anti-arrhythmic therapy is unnecessary

unless there are ventricular tachyarrhythmias. Hypokalaemia and hypomagnesemia are precipitating factors that should be avoided and addressed if they are present. [19]

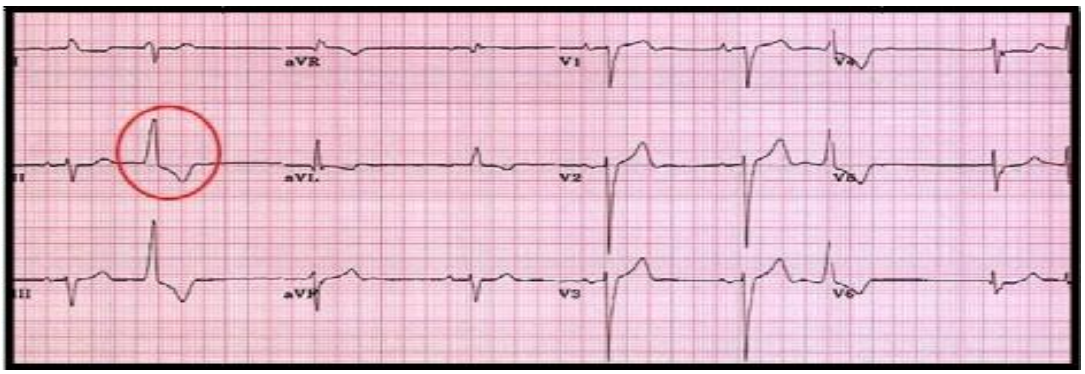


FIGURE 3 – ECG SHOWING VENTRICULAR PREMATURE COMPLEXES

Ventricular Tachycardia and Ventricular Fibrillation

The danger of death does not seem to be present in paroxysmal and clinically minor bouts of ventricular tachycardia. However, the prognosis is likely to worsen if individuals with trans-mural infarction and left ventricular dysfunction experience prolonged ventricular tachycardia later in their disease (Figures 4 and 5). [19]

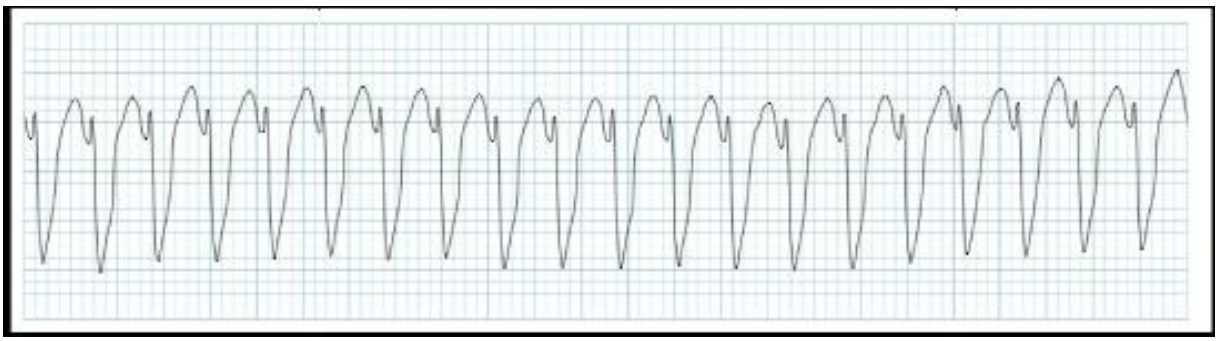


FIGURE 4 – ECG SHOWING MONOMORPHIC VENTRICULAR TACHYCARDIA.

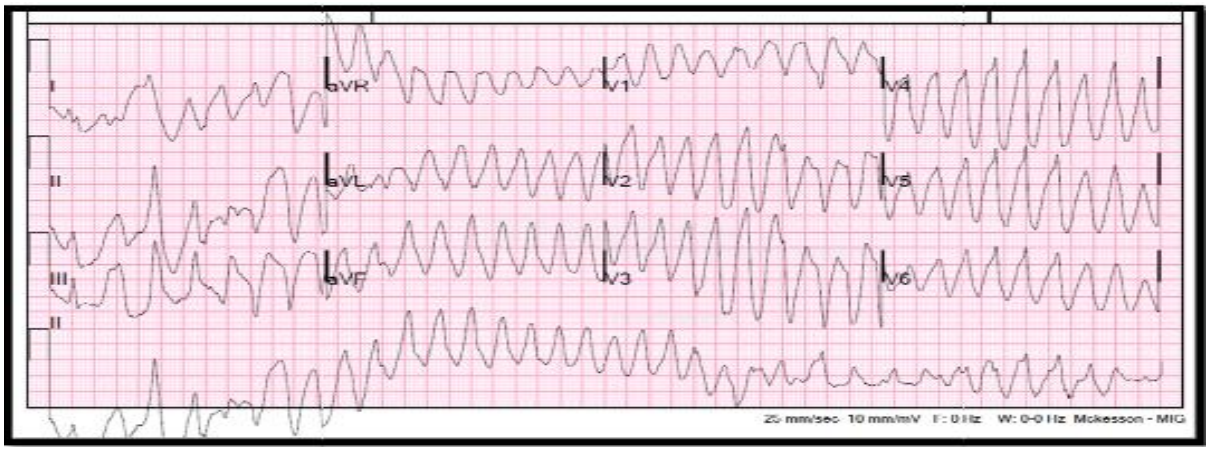


FIGURE 5– ECG SHOWING POLYMORPHIC VENTRICULAR TACHYCARDIA

Patients admitted with STEMI, ventricular fibrillation (VF) (figure 6), may seem to comprise three clinical conditions; primary, secondary, and late ventricular fibrillation. Any sudden arrhythmia without warnings is characterised as a ventricular failure, known as primary VF. The secondary ventricular failure occurs as a terminal event secondary to an already worsened left ventricle. If the duration extends for more than 48 hours or is seen after STEMI, it is known as a late type of VF, which is more prevalent in patients with ventricular dysfunction and large infarct. [19]

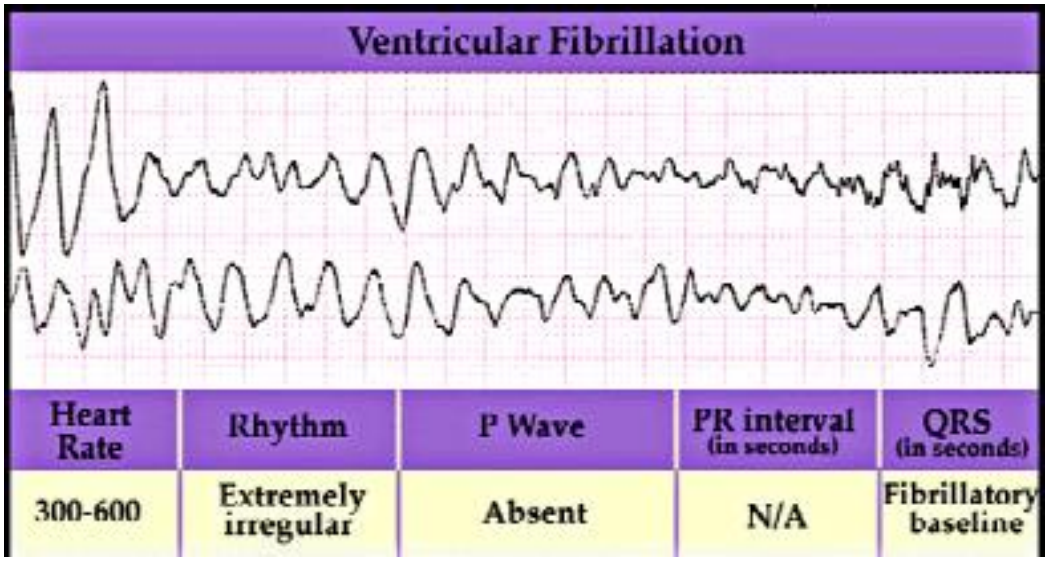


Figure 6 – ECG PORTRAYING VENTRICULAR FIBRILLATION

Sinus bradycardia

Sinus bradycardia (figure 7) is seen during the initial phases of STEMI, presented with inferior and posterior infarction. The treatment is warranted if the heart rate falls below 40 bpm or if the patient is hemodynamically unstable. [19]

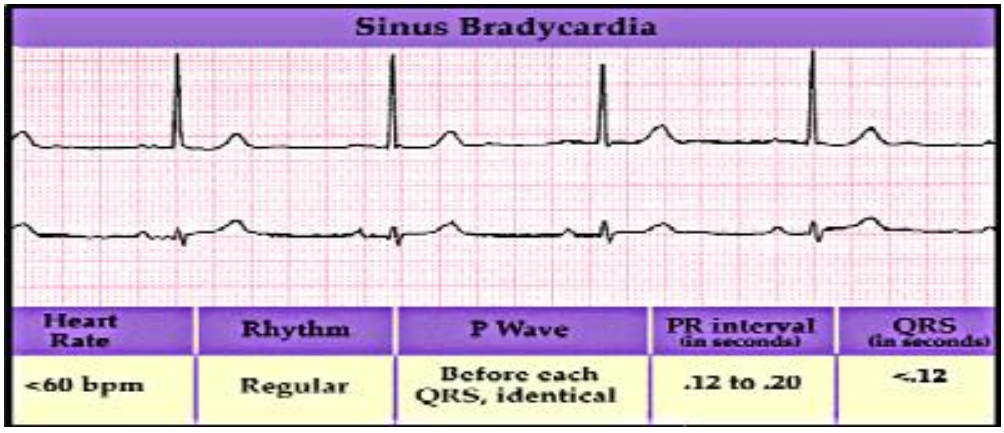


FIGURE 7 – ECG SHOWING SINUS BRADYCARDIA

Intraventricular blocks

Intraventricular blocks are seen in almost 2% to 5% of the patients. Isolated fascicular blocks affecting the posterior division pose a higher risk of death as the major cause is a large infarct. In addition, Right Bundle Branch Block in anterior STEMI presenting with congestive heart failure can be associated with an elevated mortality rate. New onset of the lesion can result in ventricular fibrillation in hospitalised patients. [19]

Bifascicular block can progress to the complete atrioventricular block, whereas extensive myocardial infarction can lead to a conduction defect in similar patients. [19]

Sinus Tachycardia

The clinical presentation associated with myocardial infarction, such as pain anxiety, pericarditis, left ventricular failure, hypovolemia, and sympathetic over-activity due to atropine, can lead to sinus tachycardia. The dysfunction can be seen commonly with anterior wall infarcts and left ventricular dysfunction. [19]

Beta-Blockers are indicated for arrhythmia, such as tachycardia due to hyperdynamic circulation. This is also applicable to patients newly diagnosed with STEMI. Hypotension and hemodynamic instability can be a contraindication for the use of Beta-Blocker. [19]

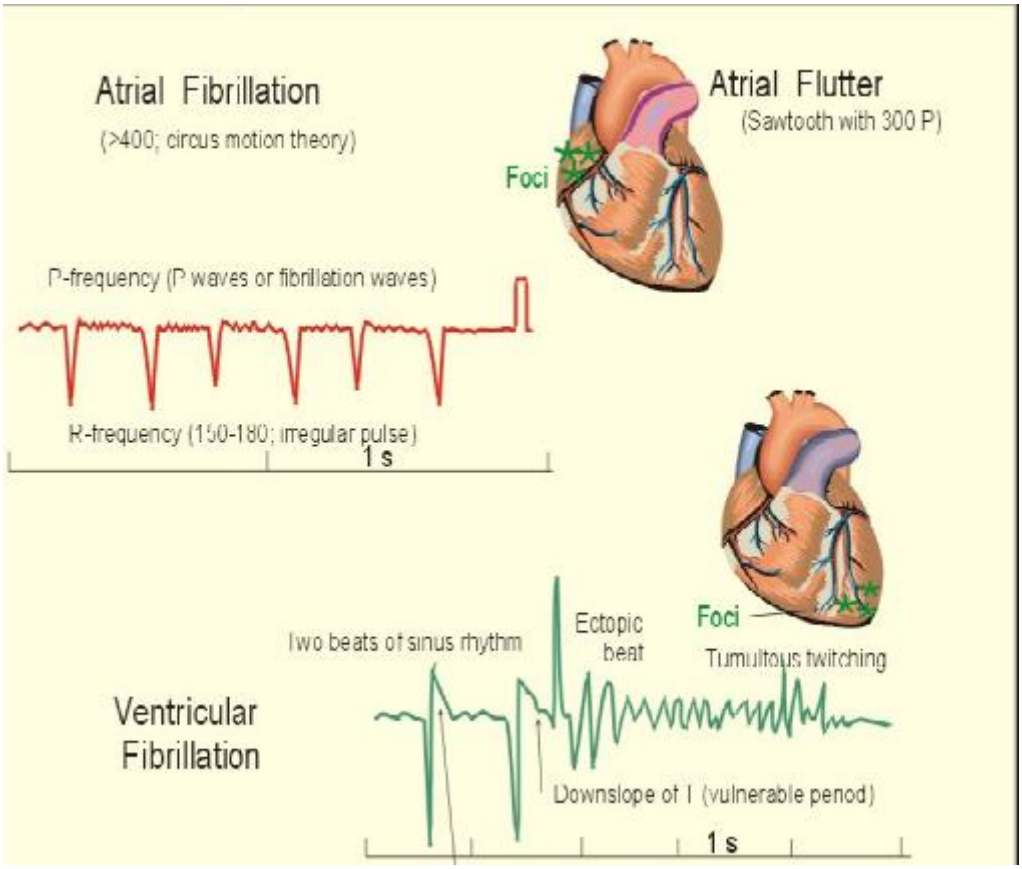


FIGURE 8 – ATRIAL FIBRILLATION AND ATRIAL FLUTTER

Atrial fibrillation or atrial flutter can be precipitated due to pericarditis, right ventricular infarction, and atrial ischemic injury. In STEMI, atrial fibrillation is generally associated with stroke and increased mortality primarily due to anterior wall myocardial infarction, which is majorly seen due to poor prognosis. [19]

Complications associated with STEMI

Recurrent chest discomfort is one of the classical symptoms seen in 25% of the patients admitted with STEMI due to a high prevalence of post-thrombolysis cases. [19]

Dressler syndrome can be seen during the first to eight weeks post-infarction, also known as post-myocardial infarction syndrome. The clinical features include malaise, fever, pericardial pain, elevation in white blood cells, high erythrocyte sedimentation rate, and pericardial effusion. High doses of aspirin (650mg) can be given with a frequency of four hours. [19]

Pericarditis is the complication seen in patients with epicardial involvement in STEMI manifested by friction rubs or pain. It is essential to characterise pericardial pain as ischemic pain to avoid the irrelevant use of anticoagulants which can lead to further complications. [19]

Thromboembolism can cause significant complications in 10% of patients with STEMI. Precipitant factors include extensive anterior infarction, left ventricular thrombus (in echo), and congestive heart failure. Hemiparesis and hypertension complications can be seen when the cerebral or renal vasculature is affected. [19]

ELECTROCARDIOGRAM

The heart's principle is to provide oxygen-rich blood into the systemic circulation. By rhythmically contracting, the heart pumps blood. The contraction and relaxation of the heart are enabled by the electrical impulses generated by the cardiac muscle. Specific cells and conduction are generated based on the type of tissues in the heart. [20]

The heart's contraction is due to the generation of electrical impulses through the cardiac muscle; the impulses are generally initiated by pacemaker cells and propagated further from conduction tissues and cardiac muscle. [22]

Cardiac electrical impulses start in the sinoatrial (SA) node (pacemaker), automatically self-generated and propagated to the right and left atrium. The electrical signals pass through improvised conduction tissue in the atrioventricular (AV) junction. [21]

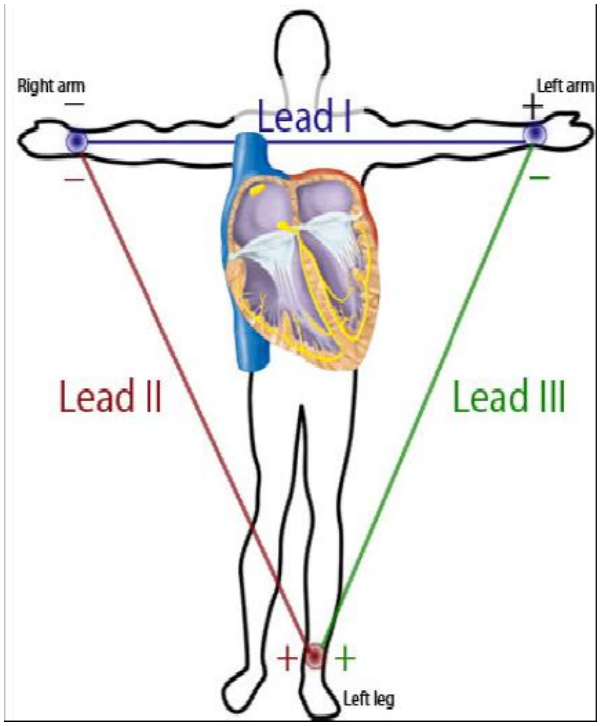
Between the atria and the ventricles, the AV junction acts as a relay junction with the proximal portion termed AV node, and the distal portion termed His bundle. The electrical impulse reaches the left and right ventricular myocardium from the bundle of His through left and right bundle branches and, finally, Purkinje fibres. Electromechanical coupling is the electrically stimulated commencement of cardiac contraction aided by releasing calcium ions by the cardiac muscle cells of the atrium and ventricles. The electrical activity progressing through the heart (within seconds) recorded on a specialised graph is called the electrocardiogram (ECG), recorded with the aid of an electrocardiograph machine. The ECG records currents produced by only the working heart muscle. The smaller currents produced by AV and SA nodes are invisible. [21]

The ECG is recorded by the placement of electrodes in the chest, arms, and legs of a person. The standard ECG consists of twelve leads showing voltage

variations among electrodes on the body's surface. The twelve leads consist of 6 precordial (chest) (figure 10) and six extremities (limb) leads. The purpose of the limb and the extremity leads is to record voltages transmitted onto the body's frontal and horizontal planes, respectively. [21]

The extremity leads are recorded by placing electrodes on the wrists and ankles, where the right leg electrode acts as an electrical ground. The heart voltages are relayed through the trunk to the limbs. From the extremity leads, three are bipolar leads (I, II, III) (figure 9) and unipolar augmented leads (aVR, aVL & aVF). The difference in voltages between the left arm and right arm is recorded by lead I. [22]

The difference in voltages between the left leg and right arm is recorded by lead II. The difference in voltages between the left leg and left arm is recorded by lead III. Augmented leads record voltages at one point concerning another electrode nearer to zero potential.



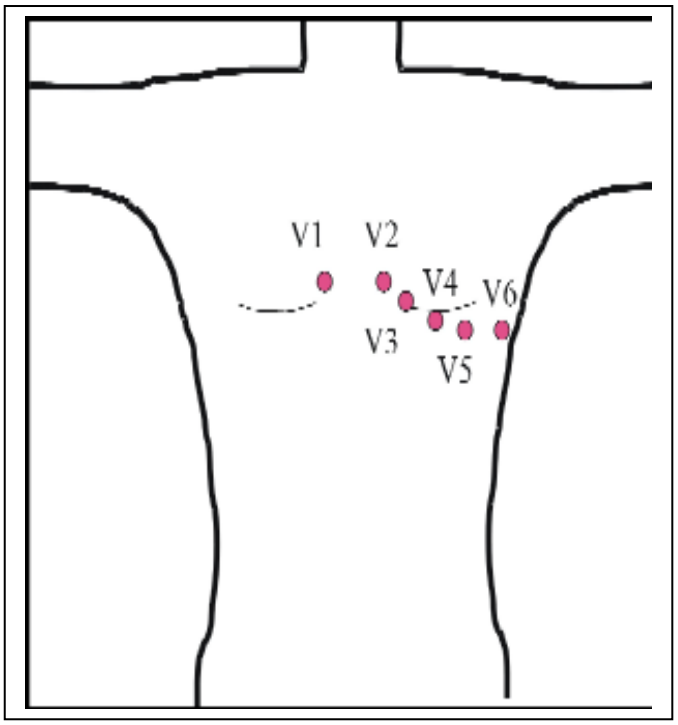


FIGURE 9 – EINTHOVEN'S TRIANGLE **FIGURE 10 – PRECORDIAL LEAD**

The stimulus is spread through the cardiac muscle resulting in cardiac muscle cell stimulation called depolarization. The activated cardiac muscle reverts to its resting state called repolarisation. [21]

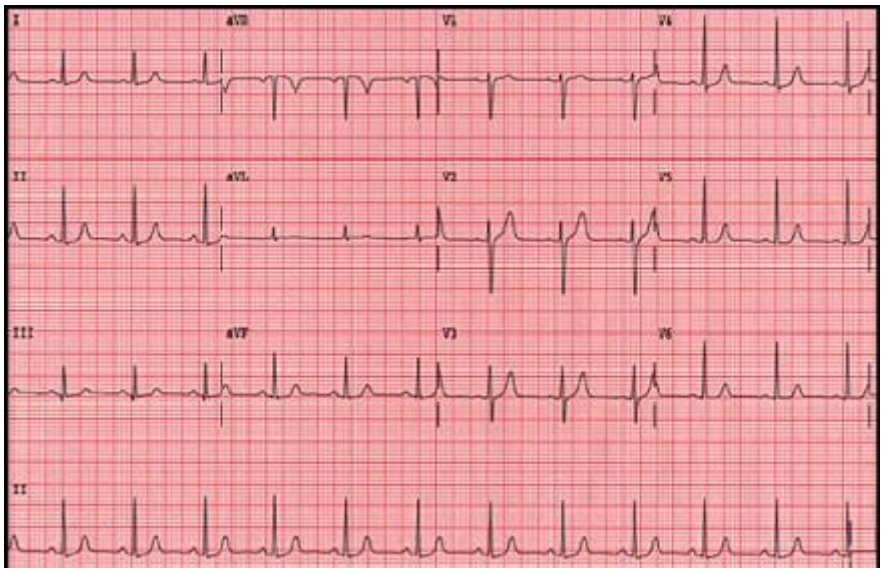


FIGURE 11 – STANDARD ECG GRAPH PAPER SHOWCASING THE LEAD

The deflections are recorded on the special paper, which consists of grids called ECG graph paper (figure 12). A small box has an area of 1 mm speed is 25 mm/second. Hence each small unit constitutes 0.04 seconds horizontally. In the vertical direction, the standard ECG machine is calibrated to showcase a deflection of 10 mm amplitude for every one mV of electrical impulse transmitted. [22]

The standard ECG consists of P wave, QRS complex, ST segment, T wave, and U wave depolarization. The QRS complex is representative of ventricular depolarization. The ST segment, T wave & U wave represent the ventricular repolarisation, with the U wave being the terminal phase. The time required for the propagation of electrical impulses via the AV junction is represented in the ECG as the PR interval, which is the distance between the start of the P wave and the start of the QRS complex. The normal PR interval in adults is generally 0.1-0.2 seconds (figure 13). [21]

QRS Complex

The QRS complex denotes the dispersion of electrical impulses in the ventricles. The first negative deflection in the QRS complex is called the Q wave, and the positive deflection is known as the R wave. The subsequent

negative deflection is the S wave. Sometimes, the QRS complex can be seen as complete positivity or negative, called an R wave and a QS wave. [21]

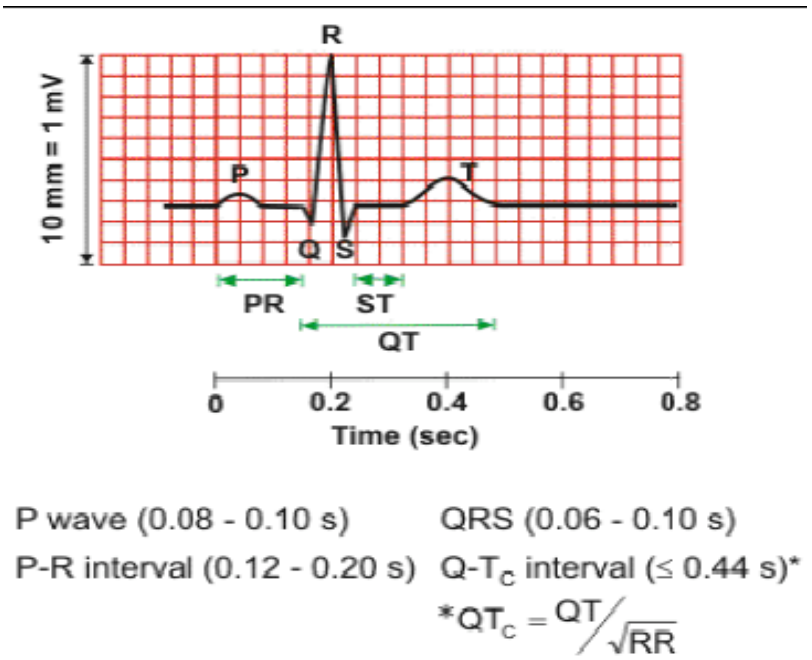


FIGURE 12 – QRS COMPLEX ON AN ECG [21]

The time interval taken for the electrical impulse to pass through the ventricles is known by measuring the QRS width. The normal time interval is 0.1 seconds.

The characteristics of the QRS complex vary in different leads according to how they are placed about the heart, which in common language, is the way the respective leads "look" at heart. [21] The QRS complex normally is positive (which means above the baseline) in apico-lateral leads because they are oriented in such a way that they look at the heart from the left side. The same applies to the leads that look at the heart from below, as from under the heart (II, III & aVF). But if the QRS complex is negative in the leads V1, V2, and

aVR, it records the heart's electrical activity from the right side. The QRS complex will be partly above and partly below the baseline (known as biphasic) in the anterior leads like V3 & V4. The amplitude of the QRS complex is usually not more than 25 mm. [22]

QRS AMPLITUDE

Prominent variation can occur in the amplitude and morphology of the QRS complex due to certain influencing factors like age, gender, and ethnicity. [23] [24] [25,26] The amplitude is inversely proportional to age, but it is said that the variation is less significant after forty years of age. Females tend to have a lower amplitude than males, and Caucasians have lower amplitudes than those of African American ethnicity. [27]

ECG CHANGES IN ST-ELEVATION MYOCARDIAL INFARCTION

Ischemia or necrosis with a complete left ventricular wall thickness is called transmural MI. Depolarisation and repolarisation are generally seen in such conditions, and sequential changes can be reflected in the ECG.

In the acute phase of STEMI, due to alteration in the flow of electrical conduction, we can see an elevation of the ST segment and occasional positive T waves in several leads, which can be similar to the ST elevation. In addition, depression of the ST segment can be seen in the reciprocal leads. The changes are seen as early, within minutes of blood flow occlusion. In the evolving phase

(after a few hours to days), the ST elevation begins to recede to baseline. Simultaneously, the hyperacute J waves become inverted, and a deep T wave inversion is seen. [28]

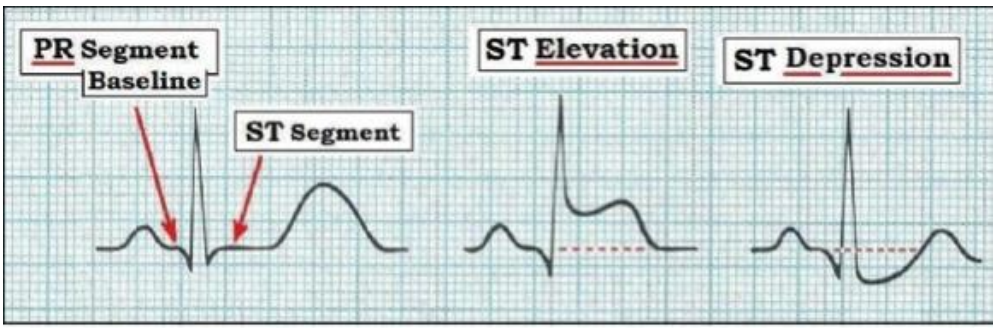


FIGURE 13 – ECG SHOWING ST CHANGES IN MI

The area infarcted is known by its representative lead in the ECG. In the case of anterior wall infarction, the ST-T changes can be evident on the interior leads, lead I, aVL, and V1 to V6 whereas, if the inferior wall is involved, the sequential ECG changes of STEMI will become evident in leads II, III, and aVF. The absolute aetiology and physiology of ST elevation are not established completely. Physiologically, the normal ST segment is isoelectric. The alteration in the ST segment can be due to abnormal injury due to infarction or ischemia affecting the outermost layer of cardiac tissue called the epicardium, which can result in STEMI. [28]

Effect of Myocardial injury on QRS Complex

The presence of myocardial injury is seen in the QRS complex as the loss of positive amplitude in the ECG lead representation of the affected area, causing the appearance of QR, QS complex, or loss of R voltage. (Figure 15). [28]

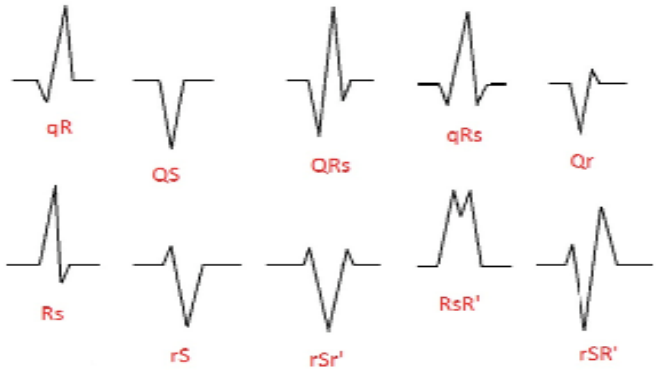


FIGURE 14 – DEFLECTIONS AND COMPLEXES OF THE QRS

Table 3 shows the overview of infarcts and the leads involved in detecting MI.

TABLE 3 – OVERVIEW OF INFARCTS

Location of Infarct	Arterial Supply	Indicative Changes	Reciprocal Changes
Anterior	LAD	**V1-V4**	II, III, aVF
Inferior	RCA	**II, III, aVF**	I, aVL
Lateral	Circumflex	I, aVL, V5, V6	V1
Posterior	Posterior Descending (RCA)	None	V1, V2
Septal	Septal Perforating (LAD) Posterior Descending (RCA	Loss of R wave in V1, V2, or V3	None

ECG as a prognostic marker in myocardial infarction

Much research has been conducted to evaluate the ECG as one of the prognostic markers in myocardial infarction. The GUSTO-1 database (Table 4) was conducted among 1021 patients with multivariate analysis for thirty days' infarction and mortality with a correlation to ECG's predictive behaviour in MI. [29]

TABLE 4 – ECG VARIABLES PREDICTIVE OF 30 DAYS MORTALITY – GUSTO-1 DATABASE [29]

ECG variable	*Characteristic predictive of thirty day mortality*	*Odds ratio*
Heart rate (beats per minute)	More than 84	2.47
Sum total of ST elevation and depression	More than or equal to 19 mm	1.53
QRS complex width in case of anterior wall infarcts	Duration more than or equal to 100 milli seconds	1.55
In case of inferior infarct	ECG showing signs of previous infarct in acute inferior infarction	2.47

The study also evaluated two additional variables observed to be predictive of poor prognosis, including the occurrence of Q waves of new-onset and the absence of resolving pattern in the ST-T wave changes visible after the infarction. [29]

Further trials were conducted to obtain substantial evidence. "The GISSI-1" study revealed that ST segment deviation total strongly predicts patient mortality. The observations were seen one-month post-infarction and proved consistent after six months of primary events. [30] In addition, it was also noted

that patients with greater infarction had the therapeutic advantage of revascularisation after thrombolysis. [31]

The area of infarction was also compared in a few studies showing the comparison of anterior and inferior wall myocardial infarction, in which the anterior wall STEMI appeared to have a poorer prognosis than the inferior wall infarction. [32] [33] [34]

Few explanations in the literature have indicated that the anterior infarct was more likely to have a larger affected area of ischemic damage. In addition, another study stated that the difference in outcome was due to the higher incidence of complications arising in anterior infarcts, such as poor left ventricular function, arrhythmias, or heart failure. [35]

Echocardiogram (ECHO)

Echo is based on the principles of Ultrasound. The most comprehensive and widely used heart imaging tool is Echocardiography. Cardiac valvular function, the morphology of cardiac chambers (size & shape), real-time assessment of hemodynamics inside the heart, and diastolic & systolic functions can be evaluated during Echocardiography. Echocardiography has the advantage of being low in cost and non-invasive, resulting in minimal patient inconvenience. [19]

Image generation

The basic principle of ultrasounds is used for Echocardiography. The transducer consists of piezoelectric crystals which generate sound waves of high-frequency type (1 to 10 Mhz), which interact with tissues and return to the transducer after passing through many structures inside the body. The ultrasound machines compute the depth of the reflecting structure based on the time the waves travel to and fro. [19]

M – MODE (motion mode) – wherein the singular scan line (from singular ultrasound beam) recorded on a moving paper/screen plots time and depth on the horizontal and vertical axis, respectively. Though becoming obsolete, this mode is thought to be best suited for linear measurement assessment wherein the temporal resolution is high. [19]

TWO DIMENSIONAL MODE – wherein the phased array transducers are electronically directed and work based on the pulse-echo principle (Figure 16). The ultrasound pulses are repeatedly produced, and echoes are recorded sequentially to produce moving images. The elevated tissue depth and restricted ultrasound speed regulate the pulse repetition frequency (PRF). [19]

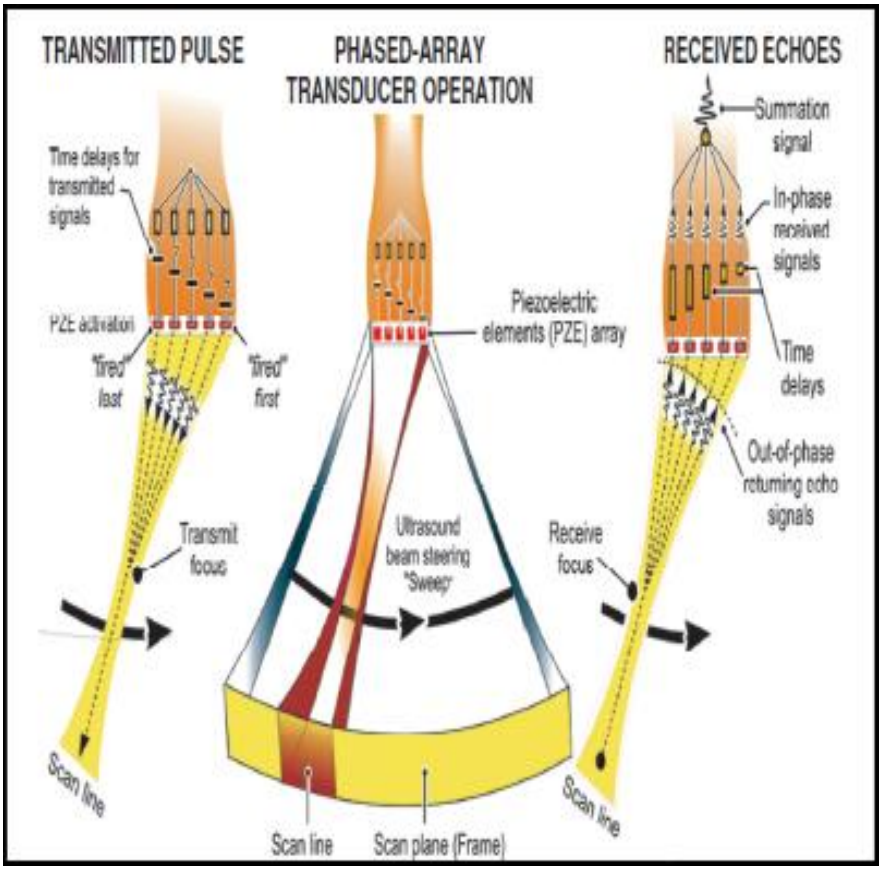


FIGURE – 15 – TWO DIMENSIONAL ECHO PHYSIOLOGY

Doppler Imaging

Cardiac chamber movement quantification and evaluation of the flow of blood (velocity) through various chambers and major vessels can be done based on the Doppler principle. *Doppler principle:* The frequency of waveform emitted from a moving object will be discerned as greater or less than the actual frequency based on the object's movement to or away from the observer. [19]

Continuous and Pulse Wave Doppler

These are two main varieties of Doppler Imaging. Ultrasound waves are sent out continuously and received by separate piezoelectric elements in Continuous Wave Doppler (CW Doppler) (Figure 17). In CW, Doppler depth analysis is not feasible, but there is no restriction on the measured velocities. Ultrasound waves are sent at discrete intervals, which are reflected off moving objects and recorded by the transducer in Pulse Wave Doppler (PW Doppler).

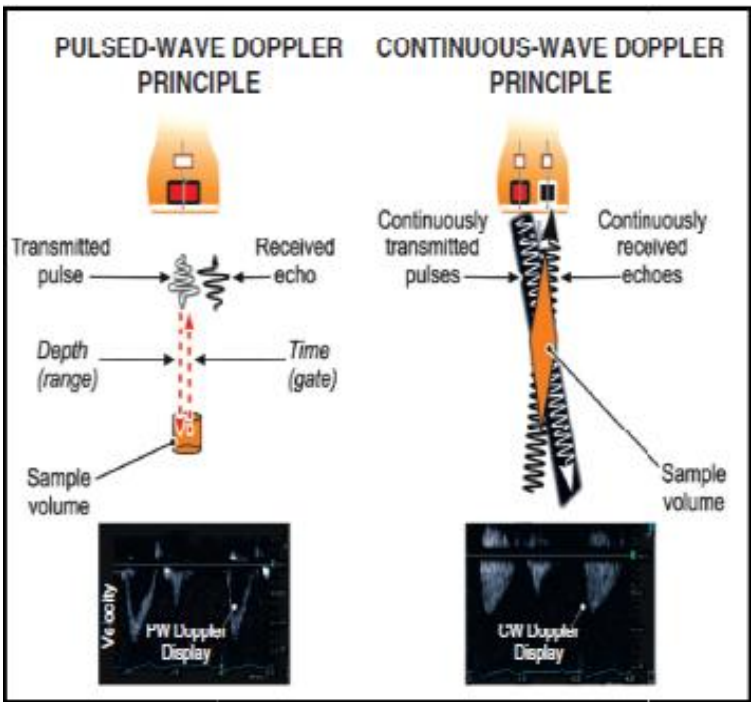


FIGURE 16 – PULSE WAVE & CONTINUOUS WAVE DOPPLER

Standard Adult Transthoracic Echo Examination

Doppler imaging, two dimensional and M-mode combination constitute standard adult TTE. The protocol recommended for a complete examination includes best image acquisition wherein three main components describe echo views. [19]

1. The standard transducer window
2. The orthogonal echo imaging planes
3. The interested anatomical region

Figure 18 describes the different echocardiographic image planes

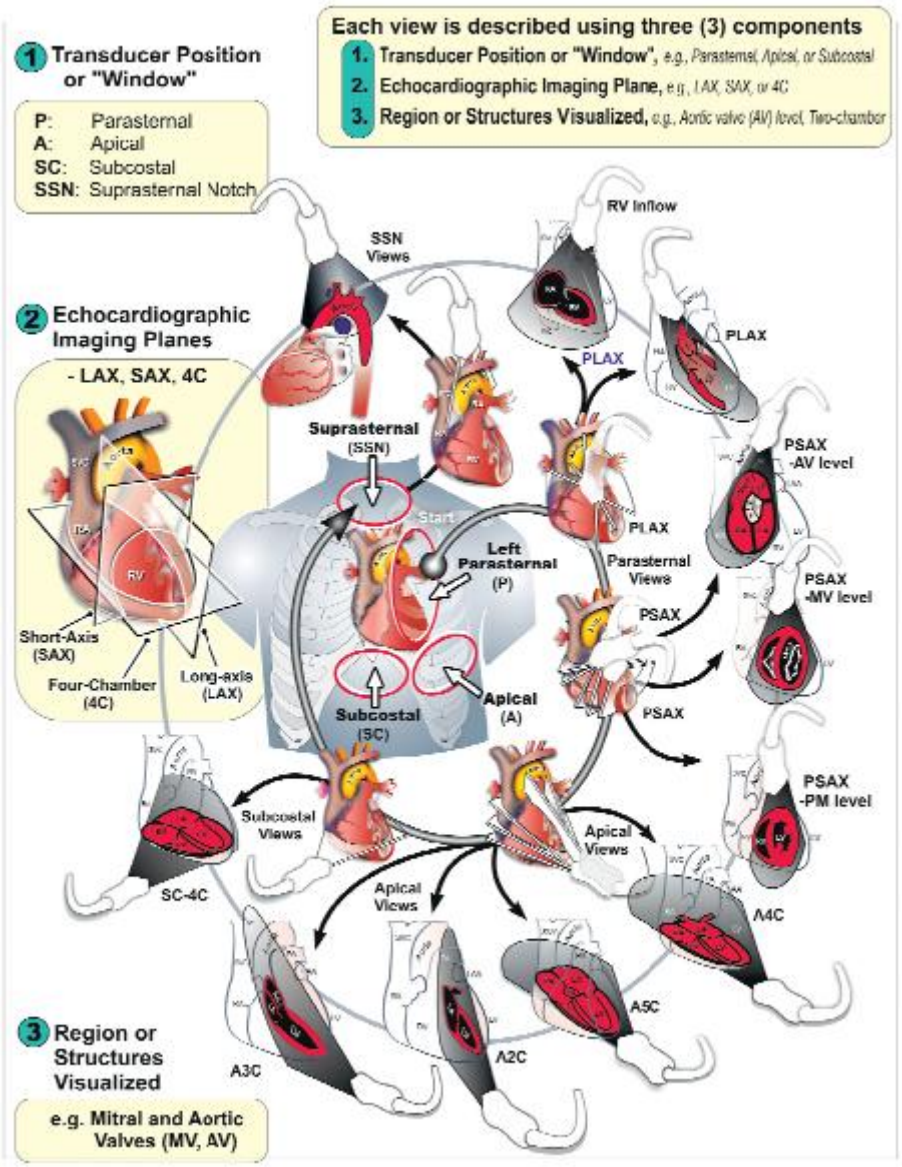


FIGURE 17 – ECHOCARDIOGRAPHIC PLANES

Left ventricular assessment

Structure: Size and Mass

The demonstrated method is the single/biplane Simpson method, favoured due to the accuracy and independent assessment of rigid geometric assumptions. [19]

The method involves; four or two chambers view after recognising the endocardial border with the assistance of a computer, and the ventricular diameter is measured from which the cross-sectional area is calculated. [19]

The limitation of this method is the difficulty in identifying the endocardial border when the image quality is poor. Errors in volumetric estimation due to alterations in transducer angle can lead to fore-shortening of the ventricle. Chamber size and wall thickness are used in several formulae to assess the left ventricular mass though their correctness can be diminished when the geometry of the ventricle is altered. [19]

Left Ventricular Systolic Function

The ejection fraction of the LV can be devised by subtracting the end-systolic volume from the end-diastolic volume and then dividing it by the end-diastolic volume. It is one of the most widely used methods for the detection and risk assessment of many cardiovascular pathologies. Though LV ejection fraction calculation by the volumetric method is ideal, due to multiple variables visual method is more accurate in seasoned echo cardiographer. Myocardial strain

imaging is a new way of estimating the heart's function and can also be performed using Doppler imaging. [19]

Regional Function of the Left Ventricle

When a particular coronary blood vessel is affected in acute myocardial infarction, regional wall motion abnormalities according to its area of distribution, scoring systems have been developed to calculate regional wall motion abnormality, of which the most extensively practised method was devised by the American Society of Echocardiography which is based on the seventeen-segment model. [19]

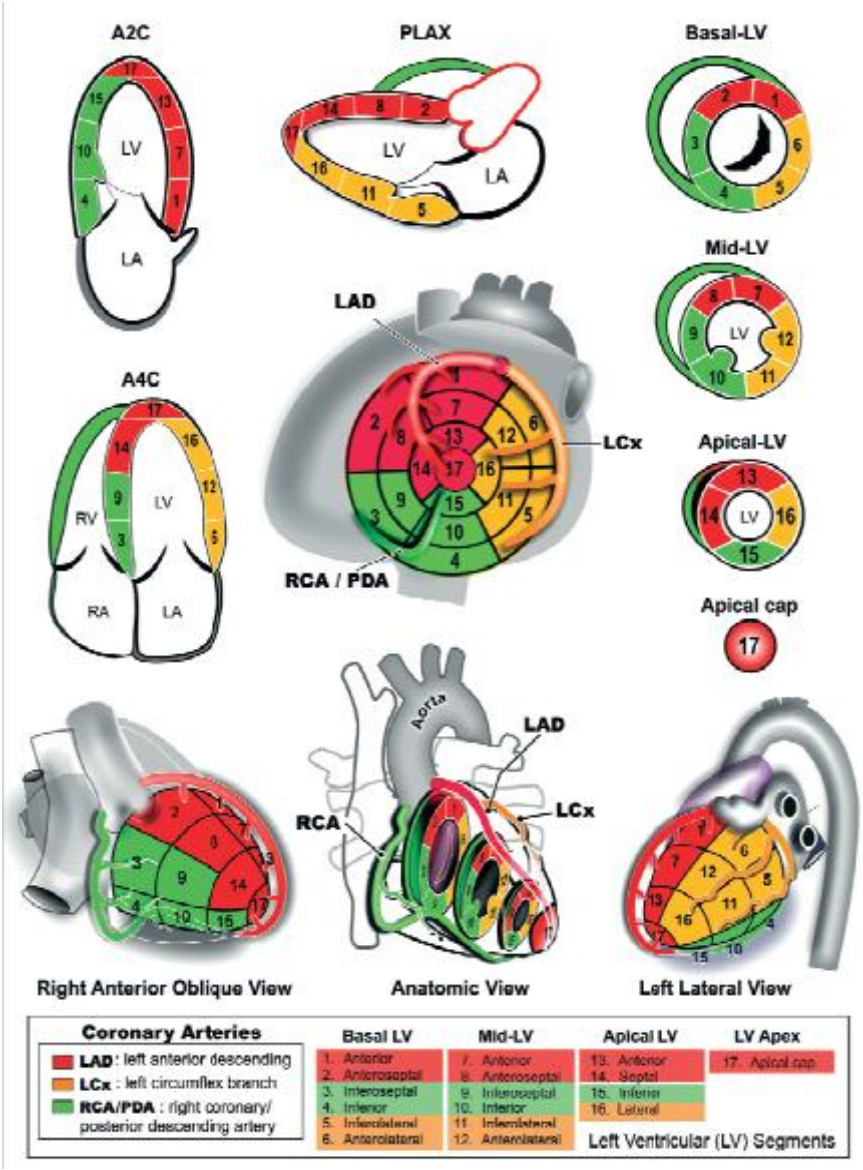


FIGURE 18 – SEVENTEEN SEGMENTS OF THE LEFT VENTRICLE

The scoring method is represented in Figure 20, which includes the following;

1. Dyskinetic – 4 points
2. Akinetic – 3 points
3. Hypokinetic – 2 points
4. Normal – 1 point

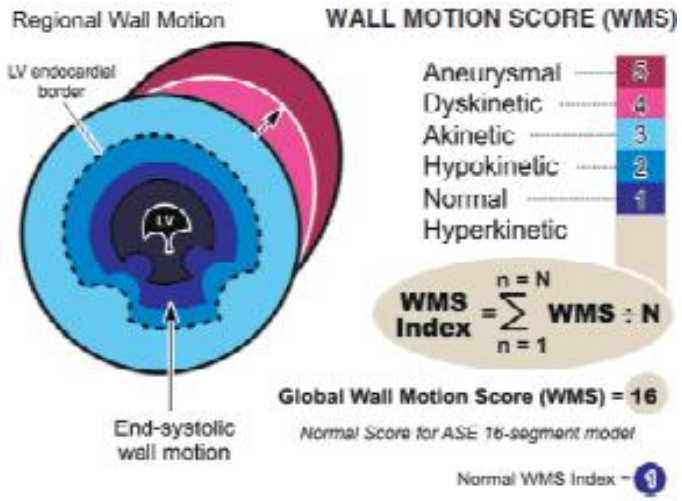


FIGURE 19 – WALL MOTION SCORE INDEX

$$\text{Wall motion score index (WMSI)} = \frac{\text{Sum of these grades}}{\text{Number of segments visualized}}$$

WMSI = 1.0 (normokinetic ventricle)

WMSI > or equal to 1.7 (physical examination findings of heart failure

Marta Lopez Castillo et al	Madrid, Spain, 2014	A prospective observational study of 144 ICU patients over five hospitals	Multivariate logistic regression analysis showed that QRS width (odds ratios [OR] 1.05, *p* = .001) on admission ECG and the sum of Q-wave depth (OR 1.06, *p* = .002) on discharge ECG were independent predictors of LVSD development.

			Moreover, QRS width on admission ECG was related to an increased risk of HF or death (OR 1.03, p = .026).
Kenneth Wood et al	Minnesota. USA, 2015	Retrospective study, 162 ICU patients	The sum of the maximum deflection of the QRS amplitude across all leads (ΣQRS) was smaller in patients with ACO than those without ACO (101.5 mm vs 132.5 mm; $p < 0.0001$), and a cutoff of ΣQRS < 90 mm was 92% specific.
Nigel S Tan et al	Toronto, Canada, 2015	Retrospective study, 12409 patients with STEMI or NSTE-ACS from GRACE	Low voltage was independently associated with higher in-hospital mortality (adjusted OR 1.77, 95% CI 1.13–2.78, P = 0.013) and mortality/re-infarction (adjusted OR 1.42, 95% CI 1.05–1.93, P = 0.023), but not 6-month mortality (adjusted

			OR 1.25, 95% CI 0.85–1.84, P = 0.27).
Akihito Kobayashi et al	NewYork, USA, 2017	Retrospective study, 190 ICU patients	Patients with low voltage had a higher rate of multi-vessel disease (MVD) (76% vs 52%, p = 0.01). In addition, patients with low voltage were more likely to undergo coronary artery bypass grafting (CABG) during admission (11% vs 2%, p = 0.028).

JUSTIFICATION:

It can help rapidly assess the severity, disease progression, duration of stay, and outcome at the initial presentation stage. In addition, it is highly cost-effective since it requires only a basic investigation- ECG.

AIM AND OBJECTIVES

Aim

To observe the association of ECG changes with left Ventricular function and clinical outcome in patients with first-time ST-elevation myocardial infarction and the association between QRS amplitude and angiographic findings.

Objectives

Primary objective:

To assess the value of QRS amplitude in STEMI patients as a prognostic indicator with the extent of Vessel involvement in the coronary angiogram.

Secondary objectives:

To assess outcome in terms of ECHO findings for Ejection fraction.

To assess Age and Sex distribution

To assess the correlation concerning Smoking, Alcoholism, Comorbidities - Diabetes and Hypertension, NYHA grading, and Killip's classification.

METHODOLOGY

A comparative study will be conducted in the Government Royapettah Hospital, Chennai-600014, and the Government Kilapauk Medical College, Chennai-600010. The comparative study will be conducted for seven months in both hospitals.

Study design: Comparative study

Period of Study: May 2022 to November 2022

Duration of Study: 6 months

Place of Study: Government Royapettah Hospital, Chennai-600014, and Government Kilpauk Medical College, Chennai – 600010.

Study Population

Inclusion criteria:

- Aged above 18 years with first-time myocardial infarction with ST elevation in chest leads.
- Presented within 6 hours after initial chest pain.
- Lysed with Inj Streptokinase immediately after admission.

Exclusion criteria:

- STEMI with ST elevation in limb leads
- Bundle branch blocks
- Ventricular paced rhythms
- Pre-existing cardiac illness
- Hypothyroidism
- Obesity
- COPD / Emphysema / Pneumothorax

Sample size:

The sample size is calculated using the multi-vessel disease as a primary outcome measure. Using data from the reference studies attached below, the Proportion in group I is 76%, and the Proportion in group 2 is 52% Risk difference is estimated as 24%. We estimated 84 subjects with a power of 80% and 5% alpha error.

Two Proportion - Hypothesis Testing - Large Proportion - Equal Allocation	
Proportion in group I	0.76
Proportion in group II	0.52
Estimated risk difference	0.24
Power (1- beta) %	80
Alpha error (%)	5
1 or 2 sided	2
The required sample size for each arm	42
total	84

Formula

$H_o : P_1 = P_2;$ $\quad H_a : P_1 \neq P_2$

$$n = \frac{\left\{ Z_{1-\frac{\alpha}{2}} \sqrt{2\,\overline{P}\,(1-\overline{P})} + Z_{1-\beta} \sqrt{P_1\,(1-P_1) + P_2\,(1-P_2)} \right\}^2}{(P_1 - P_2)^2}$$

Where,

$$\overline{P} = \frac{P_1 + P_2}{2}$$

P_1 : Proportion in the first group

P_2 : Proportion in the second group

α : Significance level

$1-\beta$: Power

Sampling Method: *Convenience Random Sampling*

Hazards of study: *Nil*

Conflict of Interests: *Nil*

METHODS

This study will be conducted in Government Royapettah Hospital as outlined. Patients hospitalised at GRH, Chennai, with ST-elevation myocardial elevation on electrocardiogram between May 2022 to November 2022, meeting the specified inclusion criteria, were included in the study. Informed consent was obtained, and history will be taken from the patients.

The patient's age, gender, and risk factors for myocardial infarction must be documented at admission. The first electrocardiogram (ECG) taken will be studied for QRS morphology. The R wave amplitude in mm (from the upper limit of the baseline to the top of the positive deflection) will be taken in all the leads of the ECG, and the sum was noted as the QRS amplitude score.

The type of management done for the patient, whether medical or interventional, will be documented. Details of echocardiogram done 48-72 hours after infarction – left ventricular function and regional wall motion abnormality will be noted. The left ventricular function will be assessed based on ejection fraction using eyeballing method.

Patients will be graded as having mild Left ventricular dysfunction (LVD) if the ejection fraction is 45- 50 %, moderate LVD if the ejection fraction is 35 – 45%, and severe LVD if the ejection fraction is less than 35%. Regional wall

motion abnormality will be assessed using the American Society of Echocardiography (ASE) method by grading the wall motion of all seventeen left ventricular segments. This is represented as a Wall motion score. For each of the individual 17 segments, a score of 1 is taken as normal regional wall motion, a score of 2 is given for hypokinesia, a score of 3 for akinesia, a score of 4 for dyskinesia, and 5 for an aneurysm. So if a patient's echo showed normal (> 45%) left ventricular function, the total Wall motion score is 17 for all segments.

The patient's clinical outcome will be assessed in terms of the New York Heart Association (NYHA) classification based on symptoms at the first follow-up after discharge from the hospital (commonly three to four weeks after infarction).

NYHA Grading	Functional Capacity
Class I	No limitations. Ordinary physical activity does not cause undue fatigue, dyspnoea or palpitations (asymptomatic LV dysfunction)
Class II	Slight limitation of physical activity. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina pectoris (mild CHF).
Class III	Marked limitation of physical activity. Less than ordinary physical activity leads to symptoms (moderate CHF).
Class IV	Unable to carry on any physical activity without discomfort. Symptoms of CHF present at rest (severe CHF).

All patients will undergo a coronary angiography after lysis with streptokinase and five days of heparinisation. Obstructive coronary artery disease (CAD) is

defined as stenosis ≧ 50% in the left main coronary artery and 70% in other epicardial coronary arteries. Multi-vessel disease (MVD) is present if there is obstructive CAD in more than two major epicardial coronary arteries. If obstructive CAD exists in all three major epicardial coronary arteries, the three-vessel disease is present. Coronary blood flow is graded according to the TIMI criteria.

OPERATIONAL DEFINITIONS:

STEMI: ST-segment elevation≧0.2mV in lead V2 and V3, and ST-segment elevation≧0.1mV in other leads (except for lead aVR)

QRS amplitude score: The R wave amplitude in mm (from the upper limit of the baseline to the top of the positive deflection) will be taken in all the leads of the ECG, and the sum was noted

Wall Motion Score: For each of the individual 17 segments, a score of 1 is taken as normal regional wall motion, a score of 2 is given for hypokinesia, a score of 3 for akinesia, a score of 4 as dyskinesia and 5 for the aneurysm.

Coronary Angiogram: Obstructive coronary artery disease (CAD) is defined as stenosis≧50% in the left main coronary artery and 70% in other epicardial coronary arteries. Multi-vessel disease (MVD) is present if there is obstructive CAD in more than two major epicardial coronary arteries. If obstructive CAD

exists in all three major epicardial coronary arteries, the three-vessel disease is present.

ANALYSIS

The collected data was entered for analysis in Microsoft Excel. This data will be exported to Statistical Package for Social Sciences software (SPSS) version 22.0. Mean, standard deviations, and range will be employed to describe continuous variables, while frequency distributions will be obtained for dichotomous variables.

RESULTS

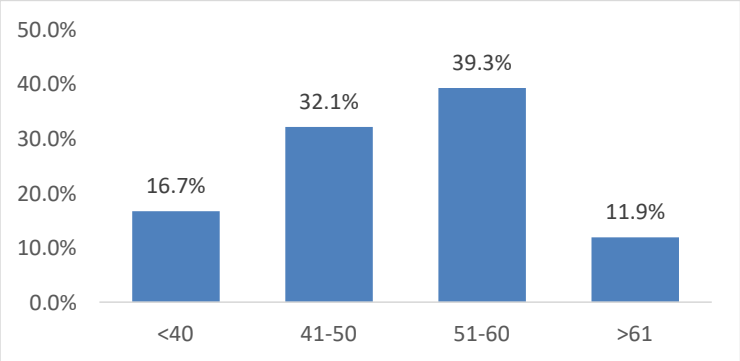
1. Demographic distribution of patients

The study observed that the age group 51-60 was most affected (39.3%), followed by the age group 41-50 (27%). A less prevalence was seen in the age group >61 (11.9%).

Table 1- Age distribution

Age group	Frequency	Percent
<40	14	16.7%
41-50	27	32.1%
51-60	33	39.3%
>61	10	11.9%
Total	84	100.0%

Figure 1 - % age distribution

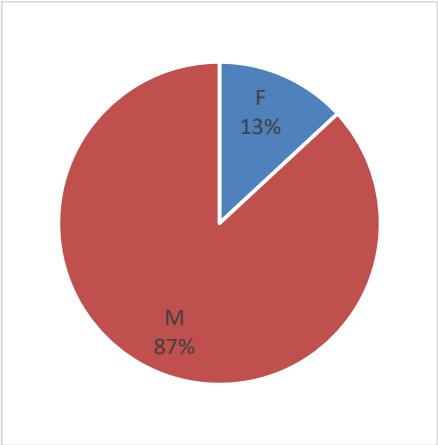


A male predominance was seen in the current study, which reports 73 males affected (86.9%) out of 84 patients enrolled in the study.

Table 2 – Gender distribution among patients

Sex	Frequency	Percent
F	11	13.1%
M	73	86.9%
Total	84	100.0%

Figure 2 - % of gender distribution



2. **Comorbid conditions**

Diabetes Mellitus was prevalent among the patients (51.2%), whereas 41 patients were not diagnosed with diabetes. In addition, hypertension was reported in 35 patients accommodating 41.7% of total patients.

Table 3 – Prevalence of diabetes

DIABETES	Frequency	Percent
No	41	48.8%
Yes	43	51.2%
Total	84	100.0%

Figure 3 - % Prevalence of diabetes

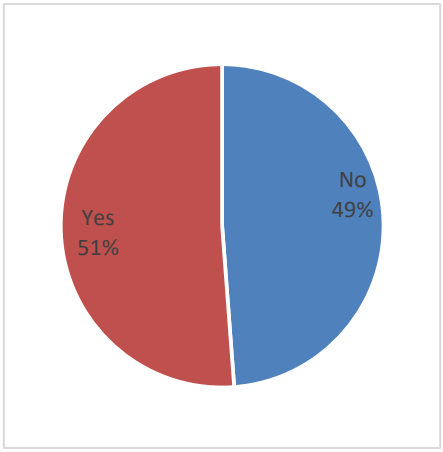
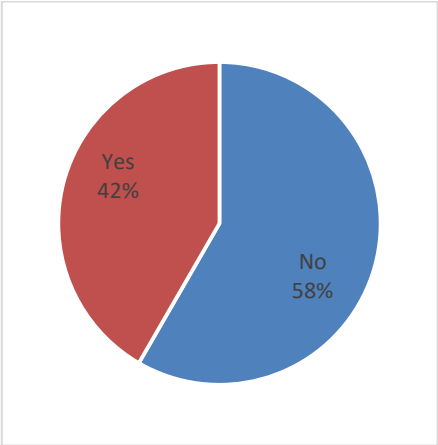


Table 4 – Prevalence of hypertension

HYPERTENSION	Frequency	Percent
No	49	58.3%

Yes	35	41.7%
Total	84	100.0%

Figure 4 - % prevalence of hypertension

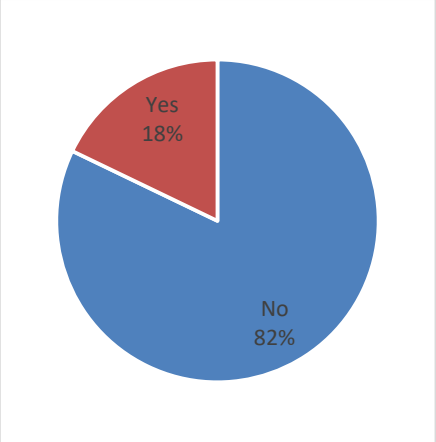


Dyslipidemia was less prevalent in the current study, accounting for 15 (17.9%) out of 84 patients.

Table 5 – Prevalence of dyslipidemia

DYSLIPIDEMIA	Frequency	Percent
No	69	82.1%
Yes	15	17.9%
Total	84	100.0%

Figure 5 - % Prevalence of dyslipidemia



3. **Social history of patients**

Social history and smoking habits were reported in 11 patients (13.1%), and 73 patients (86.9%) did not report a social habit of smoking. In addition, alcohol consumption was observed in 3 patients (3.6%) out of 84 patients.

Table 6 – Smoking history of patients

SMOKER	Frequency	Percent
No	73	86.9%
Yes	11	13.1%
Total	84	100.0%

Figure 6 - % incidence of smoking

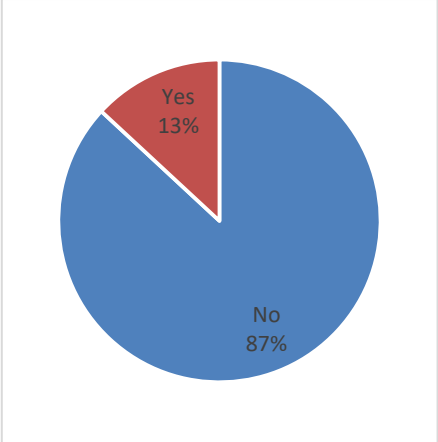
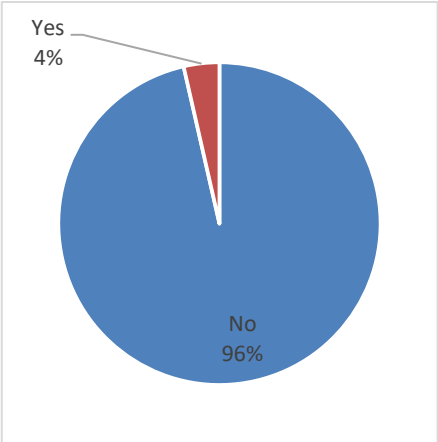


Table 7 – Alcohol consumption in patients

ALCOHOLIC	Frequency	Percent
No	81	96.4%
Yes	3	3.6%
Total	84	100.0%

Figure 7 - % prevalence of alcohol consumption



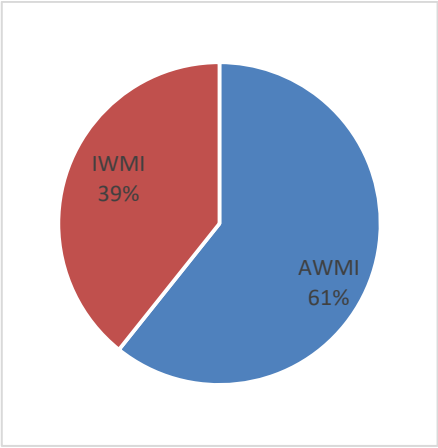
4. Clinical diagnosis of patients

Out of 84 patients, 51 were diagnosed with AWMI, accounting for 60.7% prevalence, and IWMI was reported in 33 patients (39.3%).

Table 8 – Type of Myocardial infarction

Type of MI	Frequency	Percent
AWMI	51	60.7%
IWMI	33	39.3%
Total	84	100.0%

Figure 8 – Type of MI

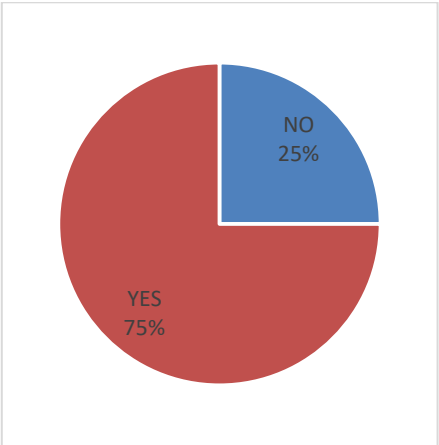


Lysed was reported in 63 patients (75%), whereas 21 patients were not presented with lysed.

Table 9 – Lysed in patients

Lysed	Frequency	Percent
NO	21	25.0%
YES	63	75.0%
Total	84	100.0%

Figure 9 - % distribution of Lysed



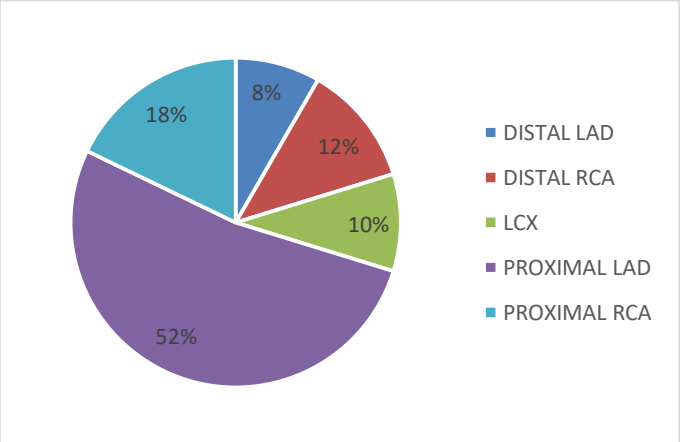
5. ECG findings

ECG localisation findings revealed that most of the patients were presented with MI affecting the proximal LAD (52.4%), followed by proximal RCA (17.9%), distal RCA (11.9%), LCX (9.5%), and distal LAD (8.3%).

Table 10 – ECG localisation

ECG localisation	Frequency	Percent
DISTAL LAD	7	8.3%
DISTAL RCA	10	11.9%
LCX	8	9.5%
PROXIMAL LAD	44	52.4%
PROXIMAL RCA	15	17.9%
Total	84	100.0%

Figure 10 - % distribution of ECG localisation

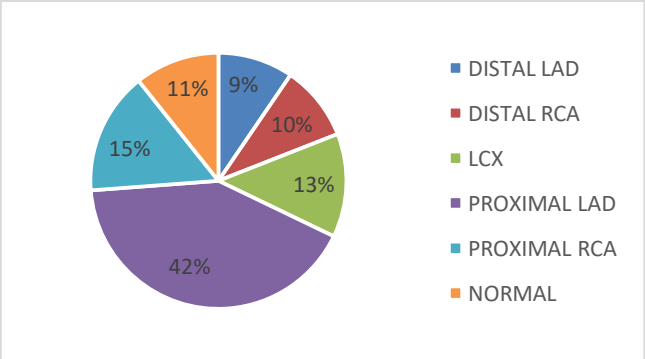


Angiographic localisation revealed that the majority of the patients were affected with MI in the proximal LAD (41.7%), followed by proximal RCA (15.5%), LCX (13.1%), and 10.7% of the patients were reported with normal functioning. In addition, distal LAD and distal RCA were reported as 9.5%, respectively.

Table – 11 – Angiographic localisation

Angiographic localisation	Frequency	Percent
DISTAL LAD	8	9.5%
DISTAL RCA	8	9.5%
LCX	11	13.1%
PROXIMAL LAD	35	41.7%
PROXIMAL RCA	13	15.5%
NORMAL	9	10.7%
Total	84	100.0%

Figure 11 – Angiographic localisation % distribution



6. Correlation of ECG localisation and Angiographic localisation

The study reports a significant difference, with a p-value <0.0001 in the correlation between ECG and angiographic localisation. Furthermore, both methods were able to diagnose proximal LAD with a higher prevalence rate and proximal RCA among the patients.

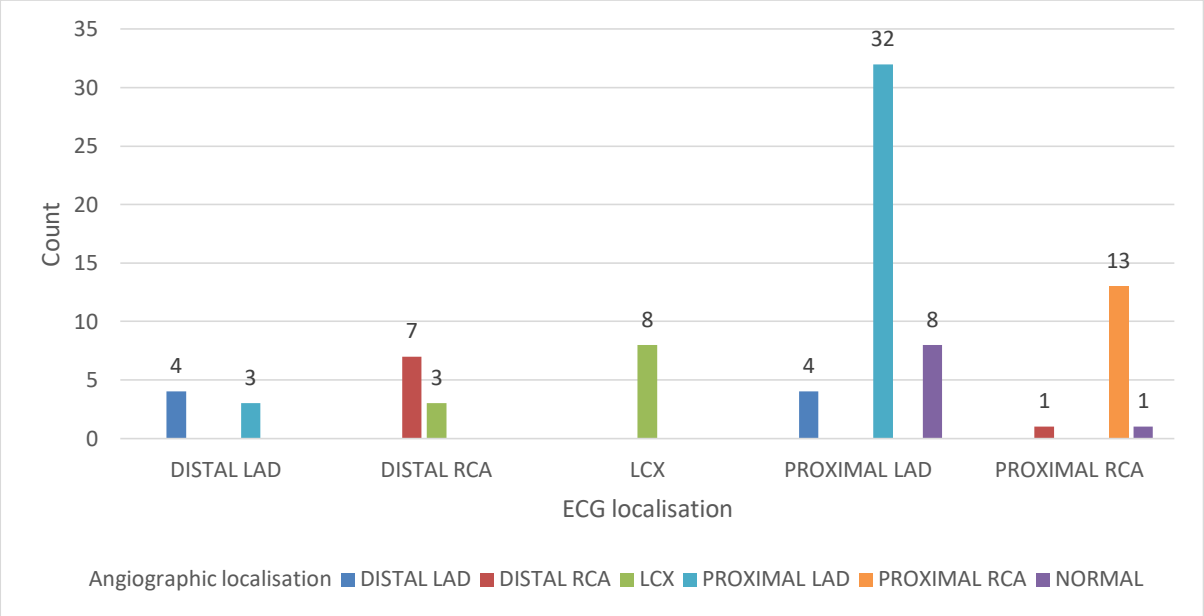
Table 12 – Correlation of ECG localisation and Angiogrpahic localistion

			Angiographic localisation						Total	P value
			DISTAL LAD	DISTAL RCA	LCX	PROXIMAL LAD	PROXIMAL RCA	NORMAL		
ECG localisation	DISTAL LAD	Count	4	0	0	3	0	0	7	<0.0001
		% within ECG	57.1%	0.0%	0.0%	42.9%	0.0%	0.0%	100.0%	

		localisation								
	DISTAL RCA	Count	0	7	3	0	0	0	10	
		% within ECG localisation	0.0%	70.0%	30.0%	0.0%	0.0%	0.0%	100.0%	
	LCX	Count	0	0	8	0	0	0	8	
		% within ECG localisation	0.0%	0.0%	100.0%	0.0%	0.0%	0.0%	100.0%	
	PROXIMAL LAD	Count	4	0	0	32	0	8	44	
		% within ECG	9.1%	0.0%	0.0%	72.7%	0.0%	18.2%	100.0%	

		localisation								
	PROXIMAL RCA	Count	0	1	0	0	13	1	15	
		% within ECG localisation	0.0%	6.7%	0.0%	0.0%	86.7%	6.7%	100.0%	
Total		Count	8	8	11	35	13	9	84	
		% within ECG localisation	9.5%	9.5%	13.1%	41.7%	15.5%	10.7%	100.0%	

Figure 12 – Comparison of ECG and angiographic localisation



7. ST-segment correlation with clinical findings

a. RCA angiographic correlation ST-segment

We observed ST-elevation in the RCA angiographic finding with a significant difference of p-value <0.0001. A total of 21 patients presented ST-elevation in L3 > L2. In addition, ST-depression aVL> L1 was also observed in 21 patients with a significant difference (p-value <0.0001) in RCA angiographic findings.

Table 13 – ST-elevation in RCA angiographic finding

			ST elevation in L3 > L2		Total	P value
			NO	YES		
RCA Angiographic	NO	Count	59	4	63	<0.0001
		% within RCA Angiographic	93.7%	6.3%	100.0%	
	YES	Count	2	19	21	
		% within RCA Angiographic	9.5%	90.5%	100.0%	
Total		Count	61	23	84	
		% within RCA Angiographic	72.6%	27.4%	100.0%	

Figure 13 – RCA angiographic correlation with ST-elevation

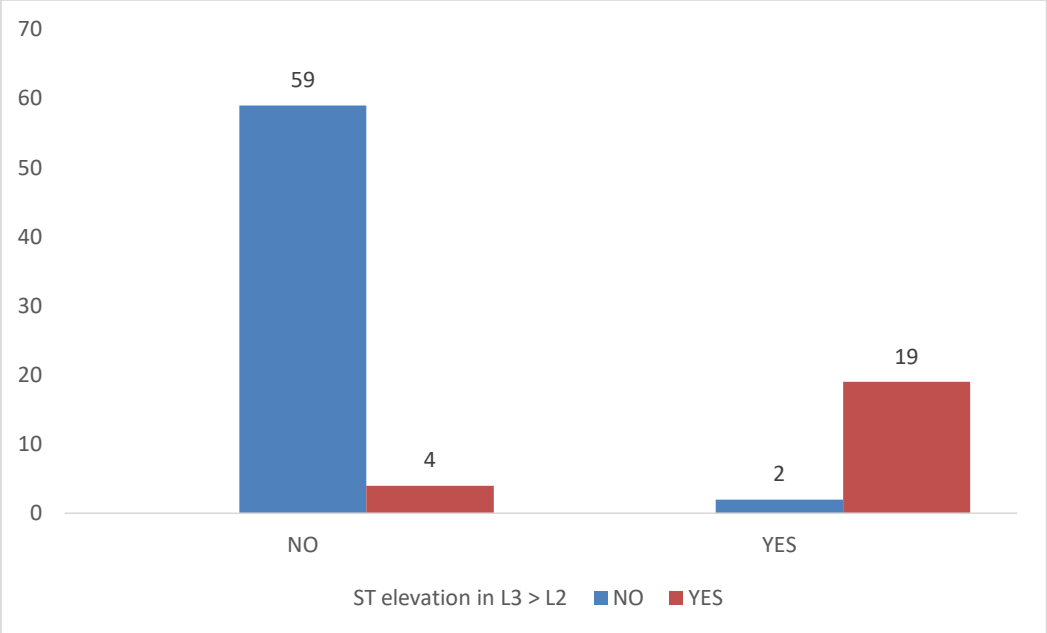
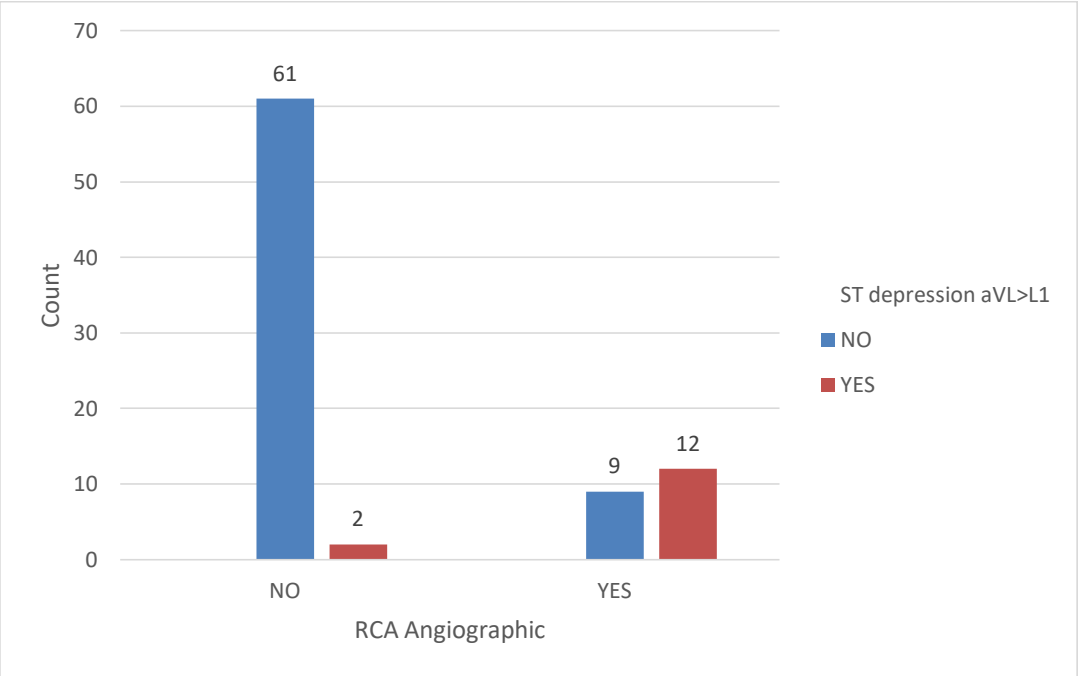


Table 14 – RCA angiographic and ST-depression

			ST depression aVL>L1		Total	P value
			NO	YES		
RCA Angiographic	NO	Count	61	2	63	<0.0001
		% within RCA Angiographic	96.8%	3.2%	100.0%	
	YES	Count	9	12	21	
		% within RCA Angiographic	42.9%	57.1%	100.0%	
Total		Count	70	14	84	

	% within RCA Angiographic	83.3%	16.7%	100.0%	

Figure 14 – RCA correlation with ST-segment depression



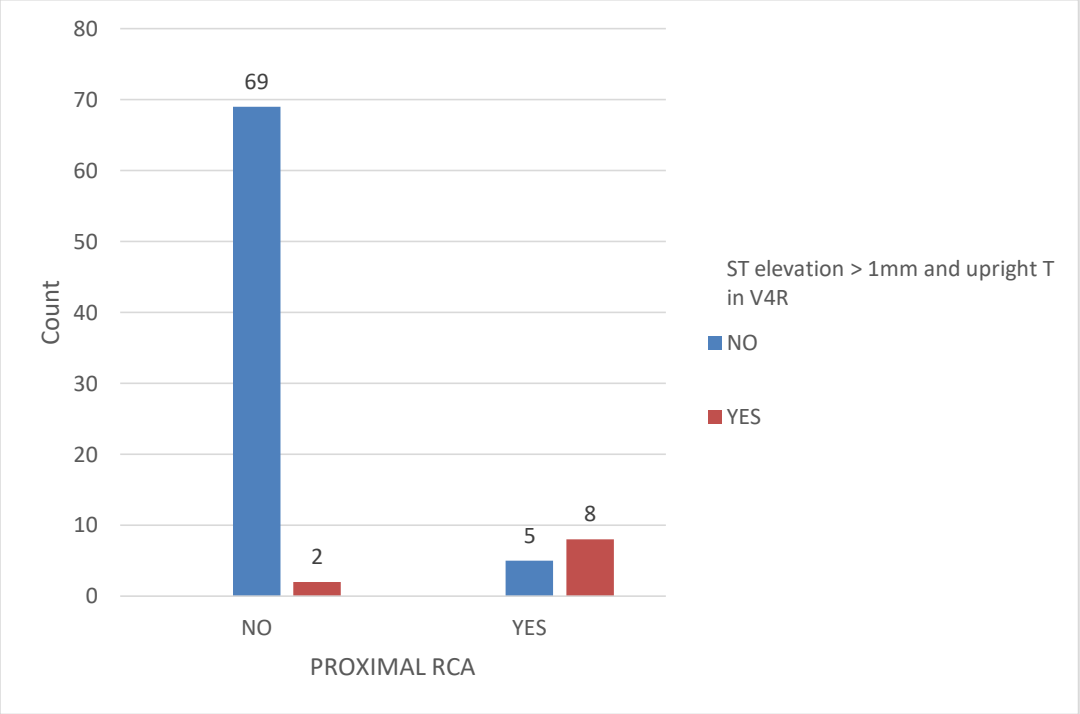
b. Proximal RCA correlation with ST segment

The study reports a significant difference (p-value < 0.0001) in patients with ST-elevation > 1 min and upright T in V4R in patients presented with proximal RCA.

Table 15 – Proximal RCA correlation with ST elevation

			ST elevation > 1mm and upright T in V4R		Total	P value
			NO	YES		
PROXIMAL RCA	NO	Count	69	2	71	<0.0001
		% within PROXIMAL RCA	97.2%	2.8%	100.0%	
	YES	Count	5	8	13	
		% within PROXIMAL RCA	38.5%	61.5%	100.0%	
Total		Count	74	10	84	
		% within PROXIMAL RCA	88.1%	11.9%	100.0%	

Figure 15 – ST segment correlation with proximal RCA patients

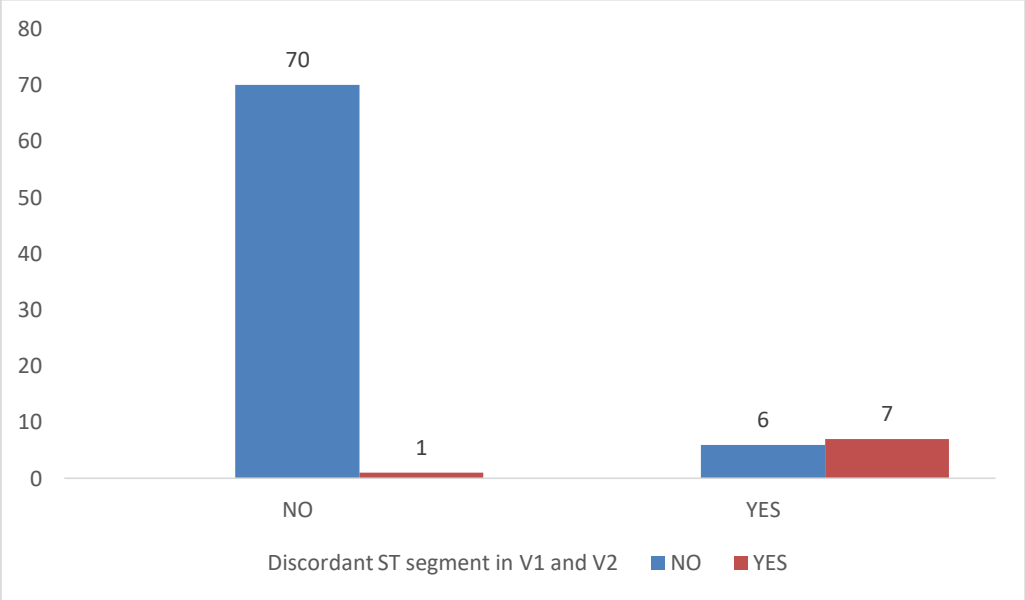


The also reports a significant difference in discordant ST-segment in V1 and V2 with p-value <0.0001. In addition, the study found that seven patients presented with proximal RCA.

Table 16 – Proximal RCA with discordant ST segment

			Discordant ST segment in V1 and V2		Total	P value
			NO	YES		
PROXIMAL RCA	NO	Count	70	1	71	<0.0001
		% within PROXIMAL RCA	98.6%	1.4%	100.0%	
	YES	Count	6	7	13	
		% within PROXIMAL RCA	46.2%	53.8%	100.0%	
Total		Count	76	8	84	
		% within PROXIMAL RCA	90.5%	9.5%	100.0%	

Figure 16 – Proximal RCA correlation with ST-segment discordant



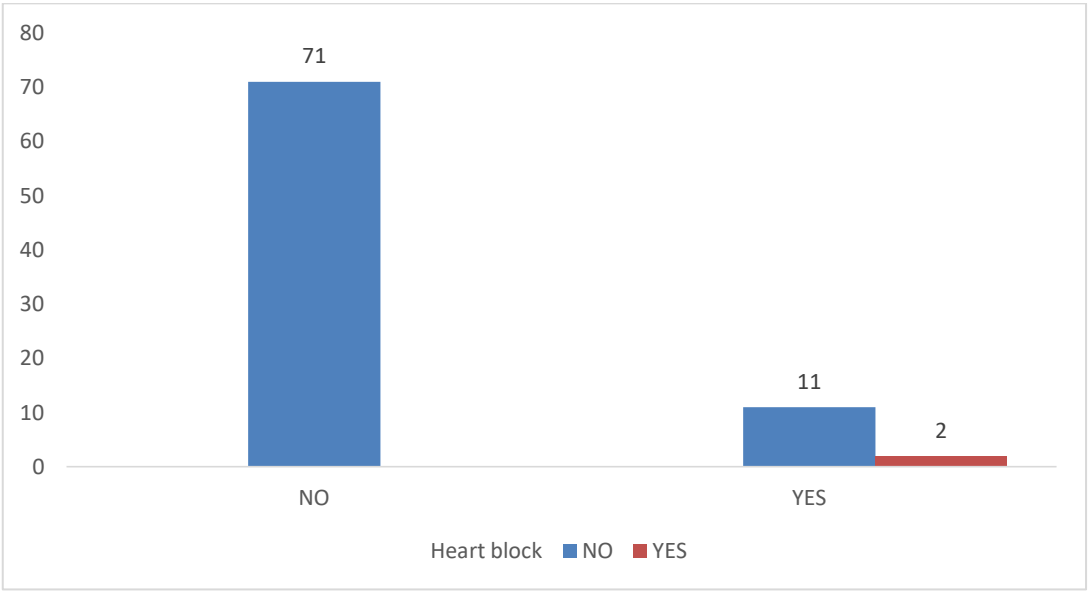
Proximal RCA patients were also seen with heart block, including a total of 13 patients out of 84 patients. The study did not find a significant correlation between the proximal RCA and heart block among patients (p-value = 0.001).

Table 17 – Proximal RCA correlation with heart block

			Heart block		Total	P value
			NO	YES		
PROXIMAL RCA	NO	Count	71	0	71	0.001
		% within PROXIMAL RCA	100.0%	0.0%	100.0%	

	YES	Count	11	2	13
		% within PROXIMAL RCA	84.6%	15.4%	100.0%
Total		Count	82	2	84
		% within PROXIMAL RCA	97.6%	2.4%	100.0%

Figure 17 – Heart block in proximal RCA patients



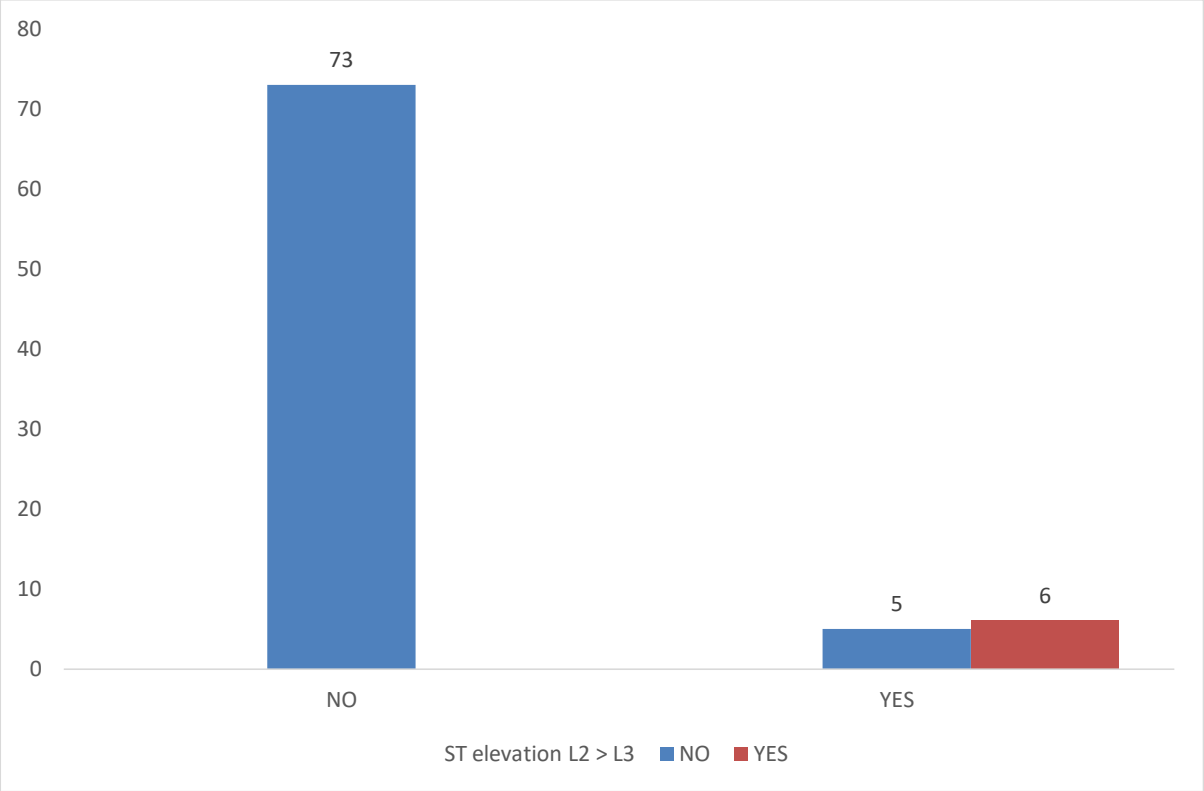
c. LCX correlation with ST segment

The study reports an ST-segment elevation L2> L3 prevalence with a total presentation of 11 patients. Our study reported a significant difference in the LCX and ST-elevation (p-value <0.0001).

Table 18 – LCX correlation with ST segment

			ST elevation L2 > L3		Total	P value
			NO	YES		
LCX	NO	Count	73	0	73	<0.0001
		% within LCX	100.0%	0.0%	100.0%	
	YES	Count	5	6	11	
		% within LCX	45.5%	54.5%	100.0%	
Total		Count	78	6	84	
		% within LCX	92.9%	7.1%	100.0%	

Figure 18 – LCX correlation with ST-segment elevation



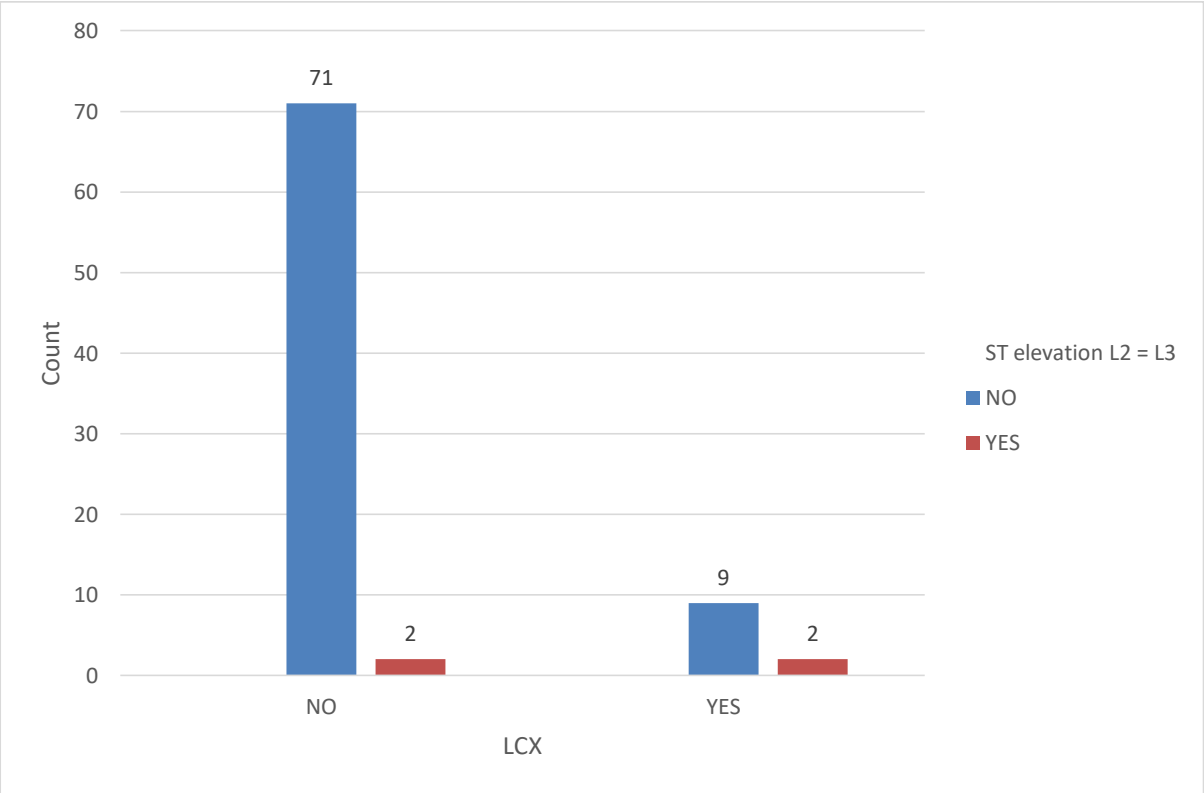
The study did not find a significant difference in ST-elevation L2 = L3 with LCX patients (p-value 0.025).

Table 19 – LCX patients with ST-elevation

			ST elevation L2 = L3		Total	P value
			NO	YES		
LCX	NO	Count	71	2	73	0.025
		% within LCX	97.3%	2.7%	100.0%	
	YES	Count	9	2	11	

		% within LCX	81.8%	18.2%	100.0%	
Total		Count	80	4	84	
		% within LCX	95.2%	4.8%	100.0%	

Figure 19 – LCX patients with ST elevations



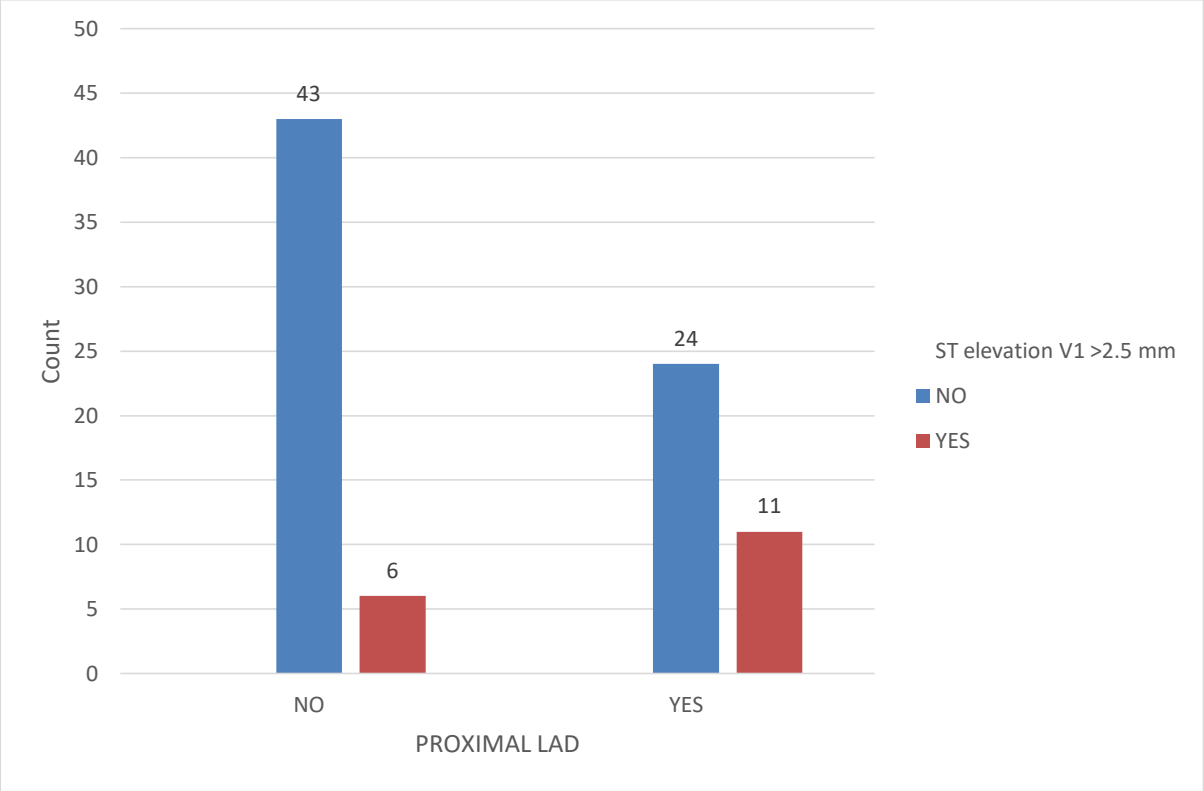
d. Proximal LAD and ST segment

The study did not find any significant correlation with ST-elevation V1> 2.5mm with proximal LAD patients (p-value = 0.031).

Table 20 – Proximal LAD and ST elevation

			ST elevation V1 >2.5 mm		Total	P value
			NO	YES		
PROXIMAL LAD	NO	Count	43	6	49	0.031
		% within PROXIMAL LAD	87.8%	12.2%	100.0%	
	YES	Count	24	11	35	
		% within PROXIMAL LAD	68.6%	31.4%	100.0%	
Total		Count	67	17	84	
		% within PROXIMAL LAD	79.8%	20.2%	100.0%	

Figure 20 – Prevalence of ST elevation in proximal LAD patients



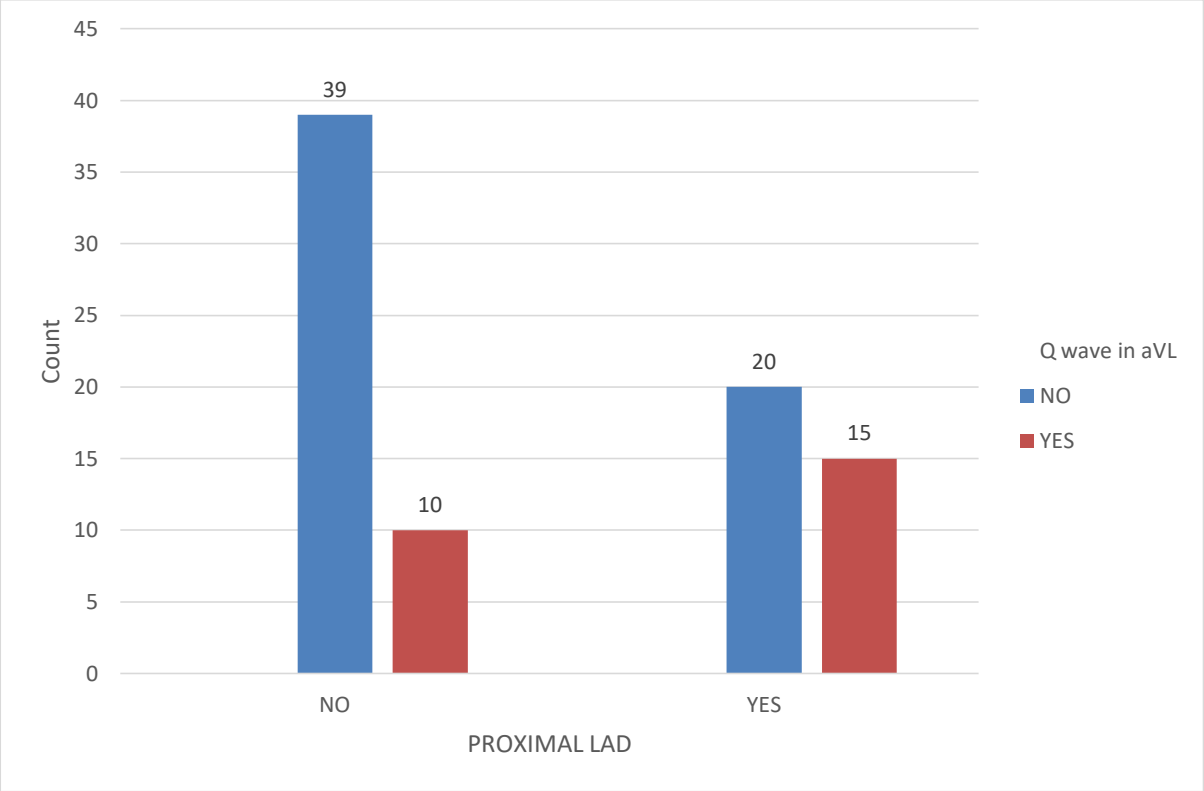
ST elevation was noted in 11 patients with proximal LAD. However, six patients reported ST elevation without the diagnosis of proximal LAD.

ST depression and proximal LAD did show any significant difference when evaluated.

Table 21 – ST depression correlation with proximal LAD

			ST depression L2, L3, aVF		Total	P value
			NO	YES		
PROXIMAL LAD	NO	Count	42	7	49	0.015
		% within PROXIMAL LAD	85.7%	14.3%	100.0%	
	YES	Count	22	13	35	
		% within PROXIMAL LAD	62.9%	37.1%	100.0%	
Total		Count	64	20	84	
		% within PROXIMAL LAD	76.2%	23.8%	100.0%	

Figure 21 – Proximal LAD and ST depression Q wave in aVL

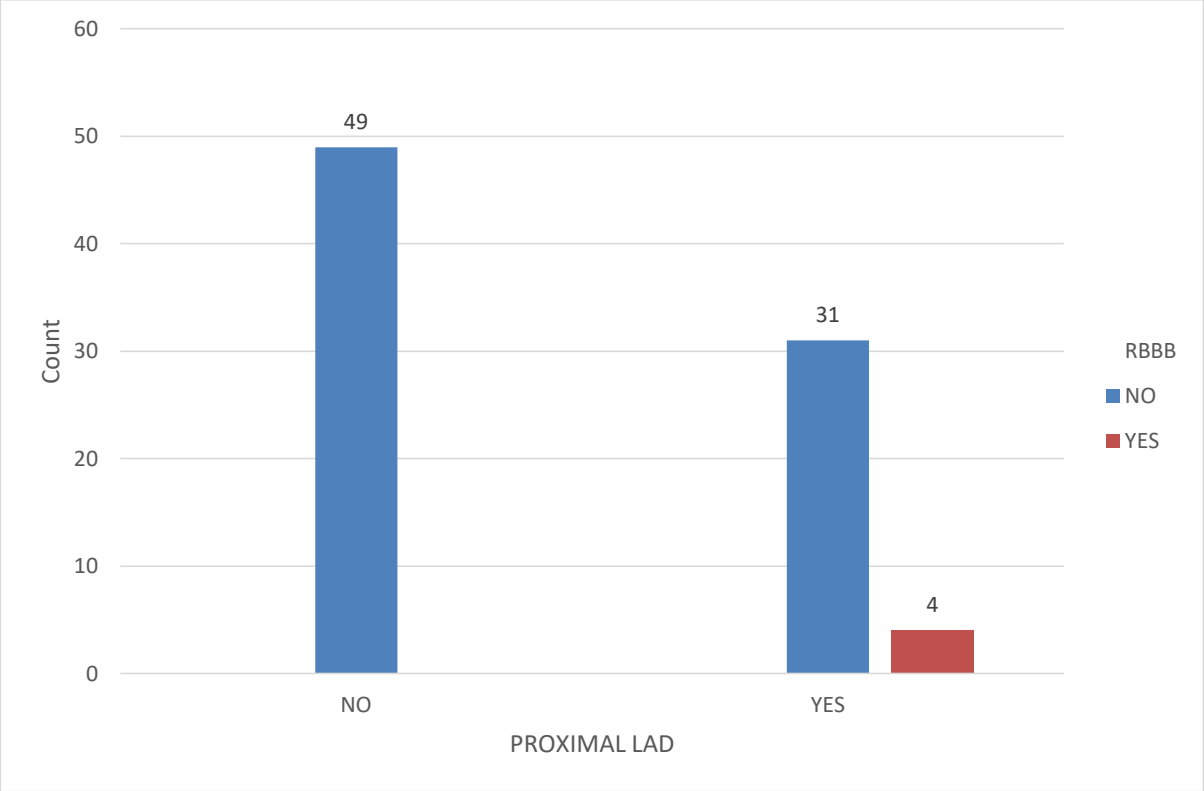


The prevalence of RBBB was seen in 4 patients with proximal LAD involvement. However, the study did not find any significant difference between proximal LAD and RBBB (p-value 0.015).

Table 22 – Proximal LAD correlation with RBBB

			RBBB		Total	P value
			NO	YES		
PROXIMAL LAD	NO	Count	49	0	49	0.015
		% within PROXIMAL LAD	100.0%	0.0%	100.0%	
	YES	Count	31	4	35	
		% within PROXIMAL LAD	88.6%	11.4%	100.0%	
Total		Count	80	4	84	
		% within PROXIMAL LAD	95.2%	4.8%	100.0%	

Figure 22 – Proximal LAD presented with RBBB



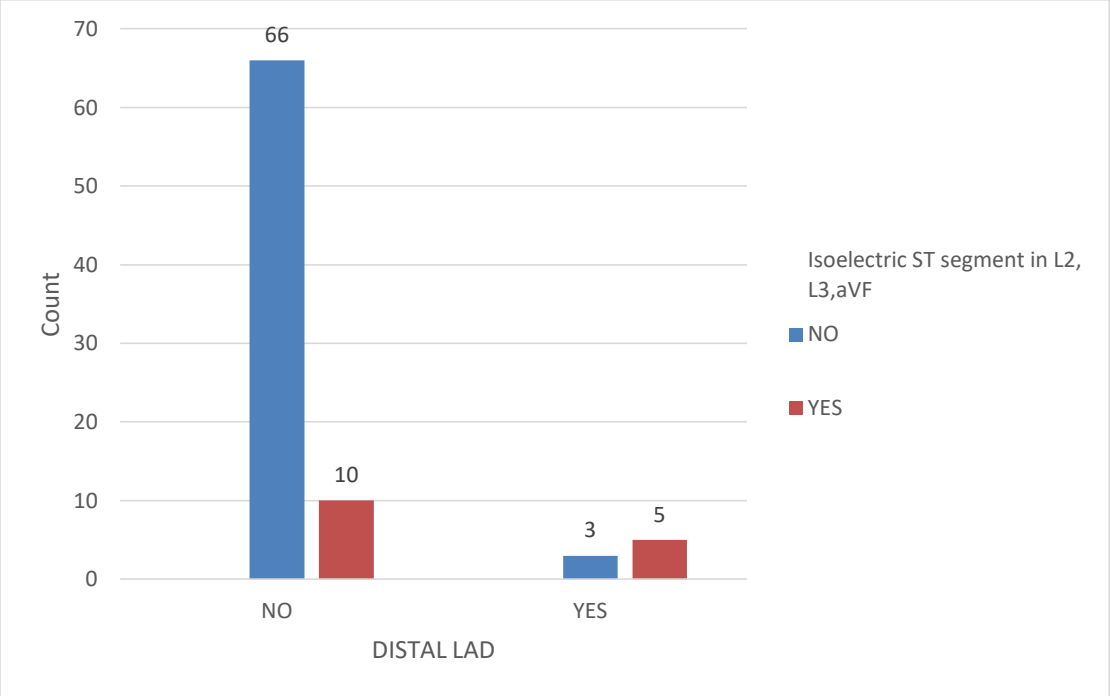
e. Distal LAD correlation with ST segment

Patients with distal LAD were diagnosed with ST-segment elevation accounting for five patients. In addition, the study did not find a significant difference (p-value = 0.001).

Table 23 – Distal LAD and ST segment

			Isoelectric ST segment in L2, L3,aVF		Total	P value
			NO	YES		
DISTAL LAD	NO	Count	66	10	76	0.001
		% within DISTAL LAD	86.8%	13.2%	100.0%	
	YES	Count	3	5	8	
		% within DISTAL LAD	37.5%	62.5%	100.0%	
Total		Count	69	15	84	
		% within DISTAL LAD	82.1%	17.9%	100.0%	

Figure 23 – Distal LAD and isoelectric ST segment in L2, L3, and aVF



8. ECG and angiographic localisation correlation with QRS amplitude

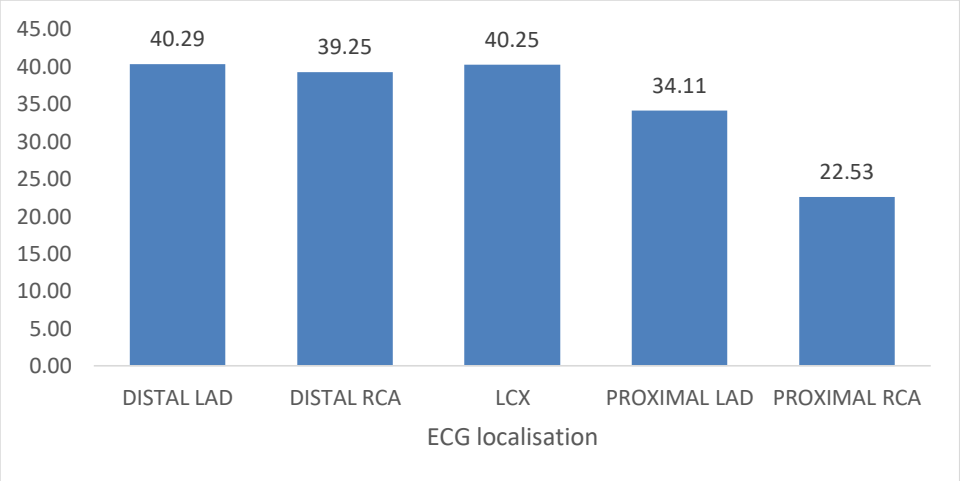
In the ECG localisation method, the study did not find any significant correlation with QRS amplitude (p-value 0.018).

Table 24 – ECG localisation and QRS amplitude

	QRS AMPLITUDE		P value
	Mean	Standard Deviation	

ECG localisation	DISTAL LAD	40.29	20.62	0.018
	DISTAL RCA	39.25	13.25	
	LCX	40.25	13.18	
	PROXIMAL LAD	34.11	14.73	
	PROXIMAL RCA	22.53	14.63	

Figure 24 – ECG localisation

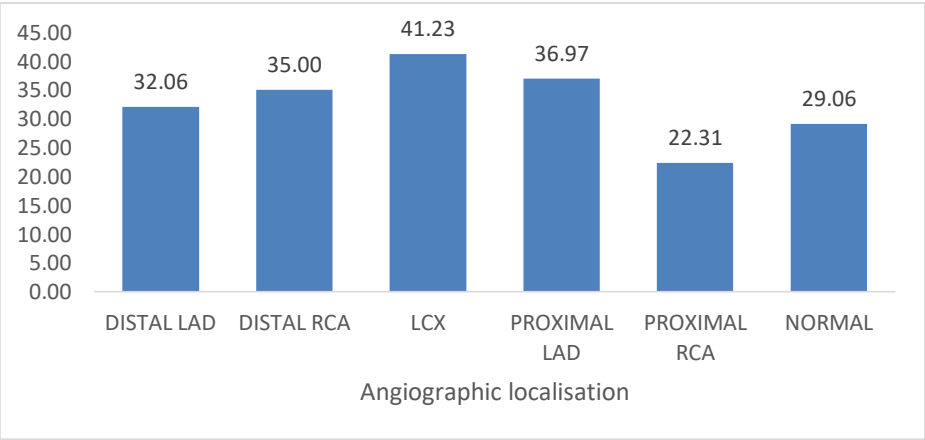


Angiographic localisation and comparison with QRS amplitude did not reveal a significant difference. However, the prevalence of LCX with higher with QRS amplitude (mean 41.23 SD 13.03), followed by proximal LAD (mean 36.97 SD – 16.64).

Table 25 – Angiographic localisation with QRS amplitude

		QRS AMPLITUDE		P value
		Mean	Standard Deviation	
Angiographic localisation	DISTAL LAD	32.06	16.11	0.032
	DISTAL RCA	35.00	13.78	
	LCX	41.23	13.03	
	PROXIMAL LAD	36.97	16.64	
	PROXIMAL RCA	22.31	15.65	
	NORMAL	29.06	6.94	

Figure 25 – Prevalence of QRS amplitude with angiographic findings



9. QRS amplitude

Using the Pearson correlation, the study reports a significant difference between the QRS amplitude and targeted angiographic findings, which can be beneficial in diagnosing vessel obstruction. By using the sig. (2-tailed) method, a p-value <0.0001 was reported in the current study.

Table 26 – QRS amplitude and Pearson correlation

		EF%	Vessel obstruction % (Angiography)
QRS AMPLITUDE	Pearson Correlation	0.235	-0.776
	Sig. (2-tailed)	0.031	<0.0001

Figure 26 – QRS amplitude and EF%

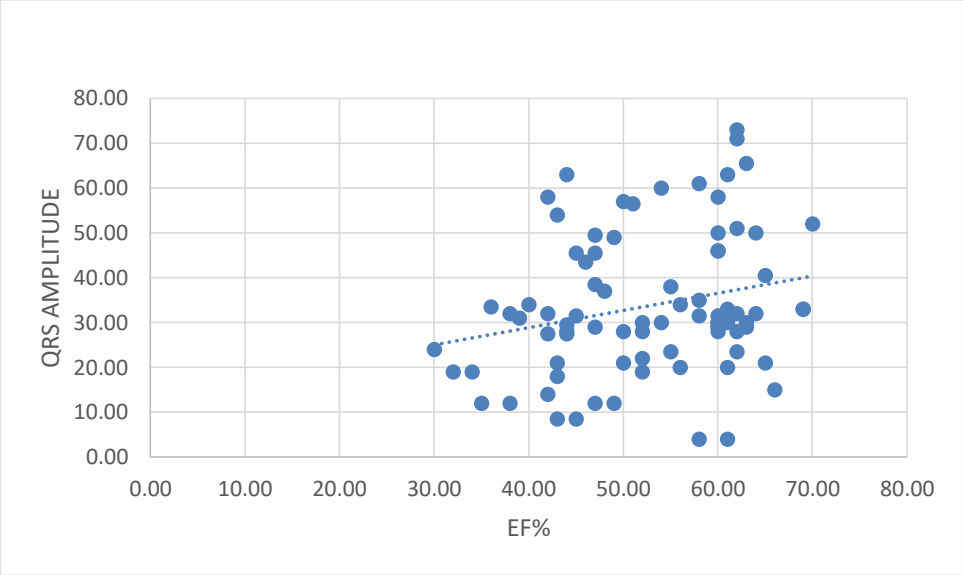
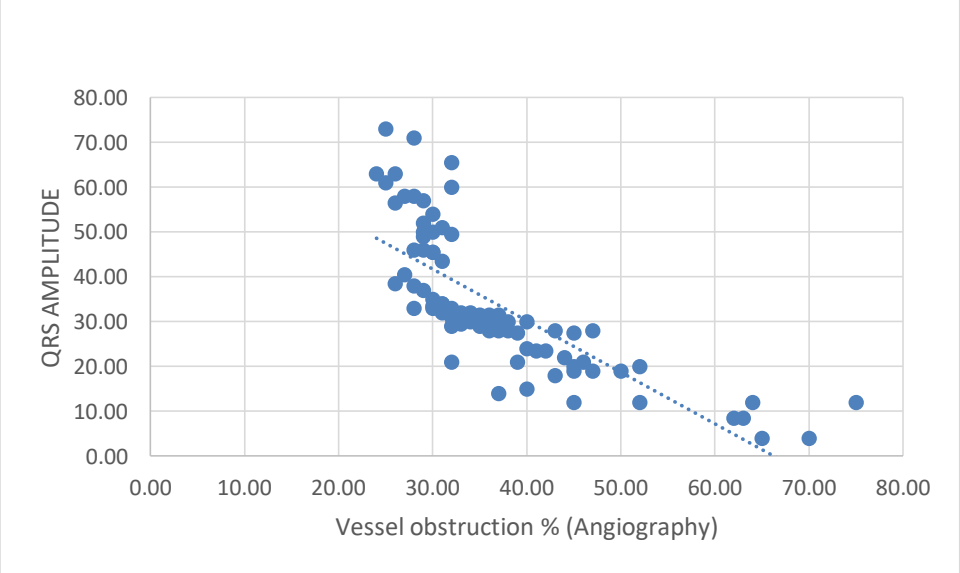


Figure 27 – QRS amplitude and Vessel obstruction %



DISCUSSION

The ST-segment elevation is one of the signs of transmural ischemia, and early revascularisation therapy can be used for positive outcomes. Several clinical studies in patients with ACS have established the relation between ST-segment elevation on the ECG and coronary artery occlusion. [36] LAD coronary obstruction is most often correlated with ST-segment elevation in the precordial leads, including V1-V4 (anterior and anteroseptal patterns). [37] In rare conditions, ST-segment elevation in the lead V1-V4 can be related to RCA occlusion, including right ventricular infarction. The isolated ST elevation in leads V4-V6 without the prevalence of V1-V3 can be a correlation to an occlusion of the LCX or the distal diagonal branch rather than the major LAD artery. [36] The major vessels in inferior MI cases can be RCA or the LCX artery. [37]

The current study reports reciprocal changes in the ECG constituting most of the patients with AWMI (60.7%) and IWMI (39.3%). The most prevalent age group was between the age range of 51-60, accounting for 39.3% of patients, followed by the age group of 41-50 (32.1%). A male predominance was reported in the current study, with 73 males (86.9%) out of 84 individuals. The current findings were similar to the study by Sanaani et al., who identified 131 patients with AWMI and 1-vessel CAD with a high male predominance (74%) and a mean age of 64 years ± 13 years. [38]

Our study reported comorbidities with diabetes affecting 51.2% of the total patients, followed by hypertension (41.7%) and dyslipidemia in 17.9% of the individuals. The history of smoking and alcohol was less prevalent in the current study, with 13.1% and 3.6% of the patients, respectively. In contrast to the current study finding, Sanaani et al. reported a high prevalence of hypertension (64.9%) and a moderate prevalence of diabetes (25.9%) out of 131 patients. However, a social history of smoking was much higher in the study, with 57.3% of individuals. [38]

ECG localisation has revealed that most patients presented with proximal LAD (52.4%), followed by proximal RCA (17.9%). Whereas, the angiographic localisation revealed a similar prevalence of proximal LAD (41.7%), followed by proximal RCA (15.5%), and LCX (13.1%)

The correlation of ECG and angiographic localisation revealed a significant difference (p-value <0.001), which signifies the accuracy of the diagnostic method for AWMI and IWMI.

The RCA angiographic revealed a significant difference when compared with ST-elevation L3> L2 with a p-value <0.0001, which diagnoses the prevalence of ST elevation in 19 patients. In addition, the ST depression aVL > L1 was found in 21 patients with a significant difference (p-value <0.0001). In addition, proximal RCA was also found to be associated with ST-elevation > 1mm and upright T in V4R in a total of 13 patients, indicating STEMI (p-value <0.0001).

The proximal RCA also revealed a discordant ST-segment in V1 and V2, affecting 13 individuals with ST-segment elevation and heart block. The left circumflex artery was seen to be associated with ST-elevation L2> L3 affecting a total of 11 individuals (p-value < 0.0001). However, Proximal Lad did not reveal significant differences in ST elevation, ST depression, and RBBB. The distal LAD was more prevalent in 8 individuals with isoelectric ST-segment in L2, L3, and aVE.

The current study did not find a significant correlation between QRS amplitude with ECG localisation. However, ECG localisation found a high prevalence of QRS amplitude with Distal LAD (mean – 40.29), followed by LCX (mean – 40.25), and Distal RCA (mean – 39.25). Similarly, no significant difference was reported during the comparison with the angiographic localisation. In contrast, the QRS amplitude with higher in the LCX (mean – 41.23), Proximal LAD (mean – 36.97), and Distal RCA (mean – 35.00).

Our study reports significant differences in the QRS amplitude with a correlation between Ejection Fraction (EF%) and vessel obstruction. The comparison yielded with Pearson correlation stated a sig. (2-tailed) value of 0.031 for EF% and a significant difference with vessel obstruction % with angiography (p-value <0.0001). The correlation signifies that a high QRS amplitude score can be associated with heart functioning and the degree of myocardial infarction.

The overall study findings revealed the reciprocal changes and clinical outcome, which implies the presence of ST-elevation in myocardial infarction with the reciprocal changes seen in ECG.

Similar findings were reported by Katragadda et al., who found that ECG changes can be used as a predictive tool for identifying STEMI. In addition, the reciprocal changes seen in the ECG can signify a poor outcome on follow-up. [39] In addition, Sannani et al. also reported a prevalence of ECG ischemic abnormalities with LAD disease in 97% of the patients with NSTEMI. However, the prevalence of ECG ischemic abnormalities was more prevalent with LAD than with the RCA or LCX disease, similar to our study findings.

The current study findings also correlated with Zoghi et al., who reported that patients with AWMI and reciprocal ST depression had significant differences in ejection fraction, as our study reported a significant difference between the ejection fraction and QRS amplitude. [40] In addition, Atroush et al. reported that patients with AWMI with pericardial ST segment depression had seen complications during their hospital course on a higher level when compared with the patients with no reciprocal changes. [41]

CONCLUSION

The current study reports a higher prevalence of AWMI (60.7%) and IWMI (39.3%), with a male predominance of 86.9%. In addition, the most affected age group was found to b 51-60 years of age, along with comorbidities such as diabetes (51.2%), hypertension (41.7%), and dyslipidemia (17.9%). The risk factor involved in MI is the social history of smoking and alcohol consumption. However, the study found a low prevalence of smoking (13.1%) and alcohol consumption (3.6%).

The study findings revealed that patients with STEMI with reciprocal ECG changes are more prone to develop LAD, LCX, or proximal RCA disease. However, the involvement of specific diseases is significant in some cases. ECG is one of the predictive tools which can be used to detect the outcome of the disease, Patients with a low QRS amplitude score are more prone to complications and longer hospital stays. Therefore, it is essential to correlate the reciprocal changes in the ECG and angiography for a better clinical assessment and to provide an adequate therapeutic outcome. A lower QRS amplitude score has also affected the ejection fraction (EF%) and can be correlated with the % vessel obstruction. Hence, ECG can be used as an early diagnostic tool for predicting the severity of STEMI and non-STEMI disease in patients.

REFERENCES

1. Tan NS, Goodman SG, Yan RT, Tan MK, Fox KAA, Gore JM, et al. Prognostic significance of low QRS voltage on the admission electrocardiogram in acute coronary syndromes. Int J Cardiol 2015;190:34–39.
2. Hwang C, Levis JT. ECG diagnosis: ST-elevation myocardial infarction. Perm J. 2014;18(2):e133. doi:10.7812/TPP/13-127
3. Kiatchoosakun S, Wongwipaporn C, Pussadhamma B. Prognostic factors of in-hospital mortality in all comers with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Heart Asia 2016;8:13–17.
4. Sorajja P, Gersh BJ, Cox DA, McLaughlin MG, Zimetbaum P, Costantini C, et al. Impact of multi-vessel disease on reperfusion success and clinical outcomes in patients undergoing primary percutaneous coronary intervention for acute myocardial infarction. Eur Heart J 2007;28:1709–1716.
5. Burns RJ, Gibbons RJ, Yi Q, Roberts RS, Miller TD, Schaer GL, et al. The relationships of left ventricular ejection fraction, end-systolic volume index and infarct size to six-month mortality after hospital discharge following myocardial infarction treated by thrombolysis. J Am Coll Cardiol 2002;39:30–36.

6. Cooper JK. Electrocardiography 100 years ago. Origins, pioneers, and contributors. N Engl J Med 1986; 315:461–4.
7. Electrocardiograph TF. Einthoven's String Galvanometer 2008; 35:174–8.
8. Hurst JW. The naming of the Waves in the ECG, With a Brief Account of Their Genesis. Circulation 1998; 98:1937–42.
9. Sattar Y, Chhabra L. Electrocardiogram. [Updated 2022 Jun 13]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK549803/
10. Barold SS. Willem Einthoven and the Birth of Clinical Electrocardiography a Hundred Years Ago 2003:99–104.
11. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, et al. Third universal definition of myocardial infarction. Circulation 2012;126:2020–35.
12. Mudd JO, Kass DA. Tackling heart failure in the twenty-first century. Nature 2008; 451:919–28
13. Heineke J, Molkentin JD. Regulation of cardiac hypertrophy by intracellular signalling pathways. Nat Rev Mol Cell Biol 2006; 7:589–600
14. Ii GWD, Force T. Review series Protein kinase cascades in the regulation of cardiac hypertrophy 2005;115
15. Sano M, Minamino T, Toko H, Miyauchi H, Orimo M, Qin Y, et al. p53-induced inhibition of Hif-1 causes cardiac dysfunction during pressure overload. Nature 2007;446:444–8.

16. Shiojima I, Sato K, Izumiya Y, Schiekofer S, Ito M, Liao R, et al. Disruption of coordinated cardiac hypertrophy and angiogenesis contributes to the transition to heart failure 2005;115
17. Douglas L. Mann. Heart Failure: A Companion to Braunwald's Heart Disease. Saunders; 2011.
18. Kass DA et al. comparative influence of load versus inotropic states on indexes of ventricular contractility: an experimental and theoretical analysis based on pressure-volume relationships. Circulation 1987; 76:1422–36.
19. Antman EM, Morrow DA, Mann DL, Oxide N, Messenger U, Signaling V, et al. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. Ninth Edit. Elsevier; 2012.
20. Franz MR, Bargheer K, Rafflebeul W et al. Monophasic action potential mapping in human subjects with normal electrocardiograms: direct evidence for the genesis of the T wave. Circulation 1987;75:381.
21. Standard L, Leads L. CHAPTER 3 ECG Leads n.d.:16–25.
22. Ary L.Goldberger. Clinical electrocardiography: A simplified approach. 8th ed. Elsevier; 2013.
23. Green LS, Lux RL, Haws CW et al. Effect of age, sex, and body habitus on QRS and ST-T potential maps of 1100 normal subjects. Circulation 1985;71:244.
24. Lamb LE. Electrocardiography and Vectorcardiography. Philadelphia: Saunders; 1965.

25. PiPberger HV, Goldman MJ LD et al. Correlations of the orthogonal electrocardiogram and vectorcardiogram with constitutional variables in 518 normal men. Circulation 1967;35:536.
26. Franz MR, Bargheer K, Rafflebeul W et al. Monophasic action potential mapping in human subjects with normal electrocardiograms: direct evidence for the genesis of the T wave. Circulation 1987;75:381.
27. Lepeschkin E. duration of electrocardiographic deflections and intervals: man. Respir Circ 1971:277.
28. Leo Schamroth. An introduction to electrocardiography. 7th ed. Blackwell Science; 2007.
29. Lee, Kerry L., Lynn H. Woodlief, Eric J. Topol, W. Douglas Weaver, Amadeo Betriu J, Col, Maarten Simoons, Phil Aylward, Frans Van de Werf and RMC. "Predictors of 30-Day Mortality in the Era of Reperfusion for Acute Myocardial Infarction Results From an International Trial of 41 021 Patients." Circulation 1995;91:1659–68.
30. Mauri, Francesco, Maria Grazia Franzosi, Aldo Pietro Maggioni, Eugenio Santoro and L, Santoro. "Clinical value of 12-lead electrocardiography to predict the long-term prognosis of GISSI-1 patients." J Am Coll Cardiol 2002;39:1594–600.
31. Hathaway WR, Peterson ED, Wagner GS, Granger CB, Zabel KM, Pieper KS, et al. Prognostic Significance of the Initial Electrocardiogram in Patients With Acute Myocardial Infarction 1998;279:387–91.

32. Califf RM, Pieper KS, Lee KL, Van de Werf F, Simes RJ, Armstrong PW, et al. Prediction of 1-Year Survival After Thrombolysis for Acute Myocardial Infarction in the Global Utilisation of Streptokinase and TPA for Occluded Coronary Arteries Trial. Circulation 2000;101:2231–8.
33. Stone PH et al. prognostic significance of location and type of myocardial infarction: independent adverse outcome associated with the anterior location. Am J Cardiol 1988;11:453.
34. Haim M et al. comparison of short-term and long-term prognosis in patients with anterior wall versus the inferior or lateral wall, SPRINT study group. Am J Cardiol 1997;79.
35. Al FC et. very early assessment of risk for in-hospital death among 11,483 patients with myocardial infarction. Am J Cardiol 1999;138.
36. Birnbaum Y, Drew BJ. The electrocardiogram in ST elevation acute myocardial infarction: correlation with coronary anatomy and prognosis. *Postgrad Med J* 2003;79:490-504.
37. Zimetbaum PJ, Josephson ME. Use of the electrocardiogram in acute myocardial infarction. *N Engl J Med* 2003;348:933-40.
38. Sanaani A, Yandrapalli S, Jolly G, Paudel R, Cooper HA, Aronow WS. Correlation between electrocardiographic changes and coronary findings in patients with acute myocardial infarction and single-vessel disease. Ann Transl Med. 2017 Sep;5(17):347.

39. Katragadda S, Alagesan M, Rathakrishnan S, Kaliyaperumal D, Mambatta AK. Correlation of Reciprocal Changes and QRS Amplitude in ECG to Left Ventricular Dysfunction, Wall Motion Score and Clinical Outcome in First Time ST Elevation Myocardial Infarction. J Clin Diagn Res. 2017 Jul;11(7):OC04-OC08.
40. Zoghi M, Gugun C, Yavuzgi O, Turko Iu I, Kultusay H, Akilli A, et al. The angiographic correlation between ST segment depression in non-infarcted leads and the extent of coronary artery disease in patients with acute inferior myocardial infarction: A clue for multi-vessel disease. Can J Cardiol. 2003;19:67–71.
41. Atroush HE, Effat H, Shehata M, Emara H. Reciprocal ST segment changes in acute inferior myocardial infarction: clinical, hemodynamic and angiographic implications. The Egyptian Heart Journal. 2012;64:97–103.

																	RCA exclusion		LCX exclusion		RCA exclusion			Proximal LAD					Distal LAD		
S.no.	Age	Age group	Sex	DIABETES	HYPERTENSION	DYSLIPIDEMIA	SMOKER	ALCOHOLIC	Type of MI	Lysed	ECG localisation	Angiographic localisation	Angio RCA	PROXIMAL RCA	LCX	PROXIMAL LAD	DISTAL LAD	ST elevation in L3 > L2	ST depression aVL>L1	ST elevation L2 > L3	ST elevation L2 = L3	ST elevation > 1mm and upright T in V4R	Discordant ST segment in V1 and V2	Heart block	ST elevation V1 >2.5 mm	ST elevation in aVR	ST depress- ion L2, L3, aVF	Q wave in aVL	RBBB	Isoelectric ST segment in L2, L3,aVF	QRS AMPLITUDE	EF%
1	56	3	M	No	No	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	22	52
2	48	2	M	Yes	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	YES	NO	NO	19	52
3	52	3	M	Yes	Yes	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	30	54
4	68	4	M	Yes	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	YES	NO	NO	38	55
5	53	3	M	No	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	34	56
6	42	2	M	Yes	No	No	No	No	IWMI	NO	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	12	38
7	45	2	M	Yes	Yes	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	33.5	36
8	52	3	F	No	No	Yes	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	52	70
9	52	3	M	Yes	Yes	Yes	No	No	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	YES	NO	NO	19	34
10	56	3	F	Yes	Yes	No	No	No	IWMI	NO	DISTAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	20	56
11	54	3	M	Yes	Yes	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	12	49
12	51	3	M	No	No	No	No	Yes	AWMI	NO	DISTAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	63	44
13	46	2	M	No	No	No	Yes	Yes	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	31.5	58
14	52	3	M	Yes	No	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	50	60
15	50	2	M	Yes	No	No	No	No	AWMI	YES	PROXIMAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	29.5	44
16	40	1	M	Yes	No	No	No	No	IWMI	NO	DISTAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	46	60
17	40	1	M	No	No	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	31.5	60
18	54	3	M	No	No	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	4	61
19	35	1	M	No	No	No	No	No	AWMI	YES	DISTAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	31	61
20	39	1	M	No	No	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	63	61
21	65	4	M	No	No	Yes	No	No	IWMI	YES	DISTAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	57	50
22	48	2	M	Yes	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	58	42
23	50	2	M	No	No	No	Yes	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	YES	NO	NO	NO	60	54
24	67	4	M	No	No	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	33	69
25	52	3	F	Yes	No	No	No	No	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	YES	NO	NO	NO	37	48
26	51	3	M	No	No	No	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	31.5	45
27	50	2	M	Yes	No	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	56.5	51
28	53	3	M	Yes	Yes	Yes	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	32	42
29	47	2	M	Yes	Yes	No	No	No	IWMI	YES	DISTAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	35	58
30	46	2	M	No	No	No	Yes	No	AWMI	YES	DISTAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	71	62
31	55	3	M	No	No	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	28	44
32	38	1	M	No	No	No	No	No	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	29	47
33	56	3	M	Yes	Yes	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	NO	YES	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	28	62
34	56	3	M	No	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	65.5	63
35	44	2	M	Yes	No	No	No	No	IWMI	YES	DISTAL RCA	LCX	NO	NO	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	45.5	45
36	48	2	M	Yes	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	49	49
37	61	4	M	Yes	Yes	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	YES	14	42
38	36	1	M	No	No	Yes	No	No	IWMI	YES	PROXIMAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	19	32
39	62	4	M	No	Yes	Yes	No	No	AWMI	YES	PROXIMAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	YES	21	43
40	52	3	M	No	Yes	No	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	23.5	55

41	58	3	M	No	No	No	Yes	No	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	29	60
42	53	3	M	Yes	Yes	Yes	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	YES	54	43
43	40	1	F	No	No	No	No	No	IWMI	NO	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	30	60
44	48	2	F	Yes	Yes	No	No	No	IWMI	YES	DISTAL RCA	LCX	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	58	60
45	38	1	M	Yes	Yes	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	YES	33	61
46	30	1	M	No	Yes	No	No	No	IWMI	YES	PROXIMAL RCA	NORMAL	NO	NO	NO	NO	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	29	60
47	52	3	M	No	Yes	No	Yes	Yes	AWMI	YES	DISTAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	24	30
48	43	2	M	No	No	No	Yes	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	YES	73	62
49	54	3	M	Yes	No	No	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	49.5	47
50	57	3	M	No	No	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	YES	21	50
51	67	4	M	No	No	No	No	No	AWMI	YES	PROXIMAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	YES	NO	NO	20	61
52	55	3	M	No	No	Yes	No	No	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	YES	NO	NO	18	43
53	53	3	F	No	No	No	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	43.5	46
54	49	2	M	Yes	No	No	Yes	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	27.5	44
55	62	4	M	Yes	No	Yes	No	No	AWMI	YES	DISTAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	32	62
56	39	1	M	No	No	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	12	35
57	65	4	F	Yes	Yes	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	12	47
58	38	1	M	No	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	51	62
59	55	3	F	Yes	No	No	No	No	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	32	38
60	49	2	M	Yes	No	No	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	29	63
61	50	2	M	No	No	No	No	No	IWMI	NO	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	YES	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	50	64
62	47	2	M	Yes	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	21	65
63	37	1	M	No	No	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	YES	NO	NO	40.5	65
64	48	2	M	Yes	No	No	Yes	No	IWMI	NO	DISTAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	45.5	47
65	50	2	M	No	No	Yes	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	30	63
66	54	3	M	No	No	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	NO	YES	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	8.5	43
67	53	3	M	No	No	No	Yes	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	YES	30	52
68	49	2	M	Yes	Yes	No	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	61	58
69	66	4	M	Yes	Yes	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	30	61
70	46	2	M	No	No	No	Yes	Yes	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	38.5	47
71	59	3	M	Yes	Yes	No	No	No	IWMI	YES	DISTAL RCA	LCX	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	28	52
72	56	3	M	Yes	Yes	Yes	No	No	AWMI	YES	PROXIMAL LAD	NORMAL	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	YES	NO	NO	NO	30	60
73	49	2	M	Yes	Yes	Yes	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	31	39
74	40	1	F	Yes	No	Yes	No	No	IWMI	NO	DISTAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	23.5	62
75	48	2	M	No	No	Yes	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	YES	28	50
76	47	2	M	Yes	No	No	No	No	AWMI	NO	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	27.5	42
77	46	2	M	Yes	No	No	No	No	IWMI	YES	LCX	LCX	NO	NO	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	32	64
78	36	1	M	No	No	No	No	No	AWMI	YES	DISTAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	46	60
79	53	3	F	Yes	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	4	58
80	54	3	M	Yes	Yes	No	No	No	IWMI	YES	PROXIMAL RCA	PROXIMAL RCA	YES	YES	NO	NO	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	8.5	45
81	48	2	M	Yes	Yes	No	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	33	69
82	64	4	F	Yes	No	No	No	No	IWMI	YES	DISTAL RCA	DISTAL RCA	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	34	40
83	55	3	M	No	Yes	Yes	No	No	AWMI	YES	PROXIMAL LAD	PROXIMAL LAD	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO	28	60
84	54	3	M	No	Yes	No	Yes	No	AWMI	NO	DISTAL LAD	DISTAL LAD	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	15	66