"STUDY OF PROGNOSIS OF AV CONDUCTION BLOCKS VERSUS INTRAVENTRICULAR CONDUCTION BLOCKS IN ACUTE MYOCARDIAL INFARCTION

By

Dr. N SRICHANDHAN REDDY, MBBS

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IN

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Under the guidance of

Dr. B.N. RAGHAVENDRA PRASAD. MD

DEPARTMENT OF GENERAL MEDICINE
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

APRIL 2016

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Date :

Place: Kolar

Dr. B.N. RAGHAVENDRA PRASAD, MD
Professor
Department of GENERAL MEDICINE
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

ENDORSEMENT BY THE HOD,
PRINCIPAL / HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"STUDY OF PROGNOSIS OF AV CONDUCTION BLOCKS VERSUS INTRAVENTRICULAR CONDUCTION BLOCKS IN ACUTE MYOCARDIAL INFARCTION"** is a bonafide research work done by **Dr N SRI CHANDHAN REDDY** under the guidance of **Dr. B.N. RAGHAVENDRA PRASAD, MD, PROFESSOR**, Department of General Medicine.

Dr. PRABHAKAR . K,MD
Professor & HOD
Department of General Medicine
Sri Devaraj Urs Medical College,
Tamaka, Kolar

Date:
Place: Kolar

Dr.M.L.HARENDRA KUMAR
Principal,
Sri Devaraj Urs Medical College,
Tamaka, Kolar

Date:
Place: Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH TAMAKA, KOLAR, KARNATAKA

ETHICS COMMITTEE CERTIFICATE

This is to certify that the Ethics committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved

Dr. N SRI CHANDHAN REDDY

Post-Graduate student in the subject of GENERAL MEDICINE

at Sri Devaraj Urs Medical College, Kolar

To take up the Dissertation work entitled

"STUDY OF PROGNOSIS OF AV CONDUCTION BLOCKS VERSUS INTRAVENTRICULAR CONDUCTION BLOCKS IN ACUTE MYOCARDIAL INFARCTION"

To be submitted to the

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH TAMAKA, KOLAR, KARNATAKA,

Member Secretary
Sri Devaraj Urs Medical College,
Kolar–563101

Date :

Place : Kolar

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Dr. N SRICHANDHAN REDDY

Date :

Place : Kolar

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Dr. N SRICHANDHAN REDDY

ABSTRACT

INTRODUCTION: Myocardial infarction is a Global epidemic, and is intimidatinglarge as the new epidemic afflicting population worldwide. According to the National Commission on Macro-economics and Health, there were around 62 million patients with Coronary Artery Disease (CAD) by 2015 in India, and of these, 23 million were younger than 40 years of age[1.]The present study will enlighten the correlation of Atrioventricular conduction blocks (AV BLOCKS) versus intraventricular conduction blocks (IV BLOCKS) in acute myocardial infarction after thrombolytic era.

AIMS AND OBJECTIVES: To study the prognosis of atrioventricular conductionblocks versus intraventricular conduction defects in patients with acute myocardial infarction

MATERIALS AND METHODS: It is a prospective and comparative cohort study.72Patients admitted in RLJH diagnosed as acute myocardial infarction who are with AV conduction blocks (atrioventricular blocks) and myocardial infarction with intraventricular conduction blocks are included in the study. That is 36 patients with acute myocardial infarction with atrioventricular conduction blocks compared with 36 patients of myocardial infarction (MI) with intraventricular conduction blocks (IV BLOCKS). 7 days follow up is done to assess the prognosis of AV blocks versus intraventricular conduction blocks in acute myocardial infarction.

RESULTS: Both atrioventricular (75%) and intraventricular (80%) blocks are more inmales but no significant difference between AV and IV blocks. Chest pain (86%) is the

common presentation for conduction disturbances in acute MI. Breathlessness is more specific for intraventricular blocks in acute MI. Anterior wall (52.8%) is involved more in intraventricular conduction blocks compared to AV Blocks (27.8%). Inferior wall (55.6%) is involved in AV blocks more than anterior Wall (41.7%). In Killips staging most of AV blocks presented in stage 3 compared to IV block which have less risk (stage1) and better prognosis. TPI insertion is needed in 25% of patients of AV block and 5.6% of patients of IV block. Based on mortality AV blocks have more mortality of 33.3% compared to Intraventricular blocks (8.3%) in acute MI.

CONCLUSION: Taking all variables together into consideration according to thispresent study atrioventricular blocks are associated with poor prognosis compared to intraventricular block in patients with acute myocardial infarction. Patients with conduction defects are at high risk of developing complications and increased mortality. They need close monitoring and optimum clinical care to reduce mortality and morbidity. Temporary external pacing and transvenous pacing definitely reduce the mortality in conduction blocks due to AMI.

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LIST OF ABBREVATIONS

Apo A	-APOPROTEIN-A
Apo B	-APOPROTEIN-B
AMI	-ACUTE MYOCARDIAL INFARCTION
AVB	-ATRIOVENTRICULAR BLOCK
AV BLOCKS	-ATRIOVENTICULAR BLOCKS
AVN	-ATRIOVENTRICULAR NODE
CAD	-CORONARY ARTERY DISEASE
CHD	-CONGENITAL HEART DISEASE
DDD	-DUAL PACING, DUAL SENSING, DUAL RESPONSE
ECG	-ELECTROCARDIOGRAPHY
ESC	-EUROPEAN SOCIETY COMMITTEE
HDL	-HIGH DENSITY LIPOPROTEIN
IV BLOCKS	-INTRAVENTRICULAR BLOCKS
IVB	-INTRAVENTRICULAR BLOCK
LA	-LEFT ATRIUM
LAD	-LEFT ANTERIOR DESCENDING ARTERY
LBBB	-LEFT BUNDLE BRANCH BLOCK
LCX	-LEFT CIRCUMFLEX ARTERY
LDL	- LOW DENSITY LIPOPROTEIN
LV	-LEFT VENTRICLE

MI	-MYOCARDIAL INFARCTION
RA	-RIGHTATRIUM
RBBB	-RIGHT BUNDLE BRANCH BLOCK
RCA	-RIGHT CORONARY ARTERY
RV	-RIGHT VENTRICLE
SAN	-SINO ATRIAL NODE
SND	-SINUS NODE DYSFUNCTION
TC	-TOTAL CHOLESTEROL
TPI	-TEMPORARY PACE MAKER INSITU

INTRODUCTION

INTRODUCTION

Myocardial infarction (MI) is a Global epidemic and is intimidating large as the new epidemic afflicting population worldwide. Myocardial infarction is one of the most common diagnoses in hospitalized patients. According to the National Commission on Macro-economics and Health, there were around 62 million patients with Coronary Artery Disease (CAD) by 2015 in India, and of these, 23 million were younger than 40 years of age .[1]

Myocardial ischemia is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium. It typically occurs when there is an imbalance between myocardial oxygen supply and demand. The most leading cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery (or arteries) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium.

As the incidence of myocardial infarction is increased in modern thrombolytic era. Conduction defects are one of the most common complications which occur following AMI, which results in increased mortality in these patients.[2] Conduction defects occur during AMI, have varied presentation. Atrioventricular (AV) blocks associated with inferior wall infarction and bundle branch blocks are more commonly associated with anterior wall MI.

CAD is the commonest form of heart disease and the leading cause of morbidity and mortality throughout the world. Its prevalence among Indians has doubled during the past two decades. Immediate and late mortality following acute myocardial infarction is dependent upon the size of the infarction, beside other factors.

The mortality rate associated with uncomplicated infarctions is less than 20 % but the mortality rate when some form of bundle branch block is present may be as high as 60%. The disorder of conduction disturbance is a source of great clinical interest due to the discovery of new drugs, advance techniques of pacing, and improved success with intervention.

Brady arrhythmias and conduction blocks are a common clinical finding and may be a physiologic reaction (for example in healthy, athletic persons) as well as a pathologic condition. Arbitrarily, Brady arrhythmias are defined as a heart rate below 60 beats per minute (bpm). These can be further categorized on the basis of the level of disturbances in the hierarchy of the normal cardiac conduction system.[3]

The two major categories are sinus node dysfunction (SND) and atrioventricular (AVB) conduction disturbances or blocks. The present study will enlighten prognosis of AVB versus IVB in AMI after thrombolytic era.

OBJECTIVES

OBJECTIVES

1. To study the prognosis of AV conduction blocks versus Intraventricular conduction defects in patients with acute myocardial Infarction.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The term "myocardial infarction" focuses on the myocardium and the changes that occur in it due to the sudden deprivation of circulating blood. The main change is necrosis of myocardial tissue. The word "infarction" comes from the Latin "infarcire" meaning "to plug up or cram." It refers to the clogging of the artery.

Globally CAD accounts for 12 million deaths annually. Incidence of coronary artery disease (CAD) is doubled in India in the last 25 years. In rural India CAD prevalence increased from 2% to 4%. In urban India it is increased by three fold from 3.45% to 9.45%.

Myocardial infarction (AMI) can be recognized by clinical features, including electrocardiographic (ECG) findings, elevated values of biochemical markers (biomarkers) of myocardial necrosis, and by imaging, or may be defined by pathology. It is a major cause of death and disability worldwide.

The serendipitous discovery of William Tillet in 1933 followed by many years of work with student Sol sherry laid foundation for use of streptokinase in thrombolysis, which changed the outcome of myocardial infarction tremendously.

Figure 1: Sir William Tillet.

BASICS OF CORONARY CIRCULATION

The left main and right coronary arteries arise from left and right coronary sinuses of aortic root, distal o the aortic valve. Within 2.5cms from its origin left main coronary divides into left anterior descending (LAD) artery which descends in the anterior interventricular groove and left circumflex (LCX) artery, which runs in the atrioventricular groove. LAD gives branches to supply the anterior part of the septum (septal perforators) and the anterior, lateral and apical walls of the left ventricle (diagonals). LCX gives obtuse marginal branches that supply lateral, posterior and inferior segments of the left ventricle (LV).

Right coronary artery (RCA) runs in the right atrioventricular groove, giving acute marginal branches, that supply Right atrium (RA), Right ventricle (RV) and inferior posterior aspects of the LV. The posterior descending artery runs in the interventricular groove and supply the inferior part of the interventricular septum. It arises from RCA in right dominant system and LCX in left dominant system.

Right coronary artery (RCA) supply SAN in about 60% of individuals, remaining 40% of patients SAN is supplied by LCX. Atrioventricular node (AVN) is supplied by small AV nodal artery which arises from dominant coronary artery (RCA in 85% of patients). Proximal occlusion of RCA, therefore results in sinus bradycardia and cause AV nodal block. Abrupt occlusion in the RCA, due to coronary thrombosis result in infarction of inferior part of LV and often RV.

Abrupt occlusion of the LAD leads to anterior or anteroseptal MI.

LCX causes infarction in the anterolateral territory of LV. Acute occlusion of left main coronary artery is usually fatal.

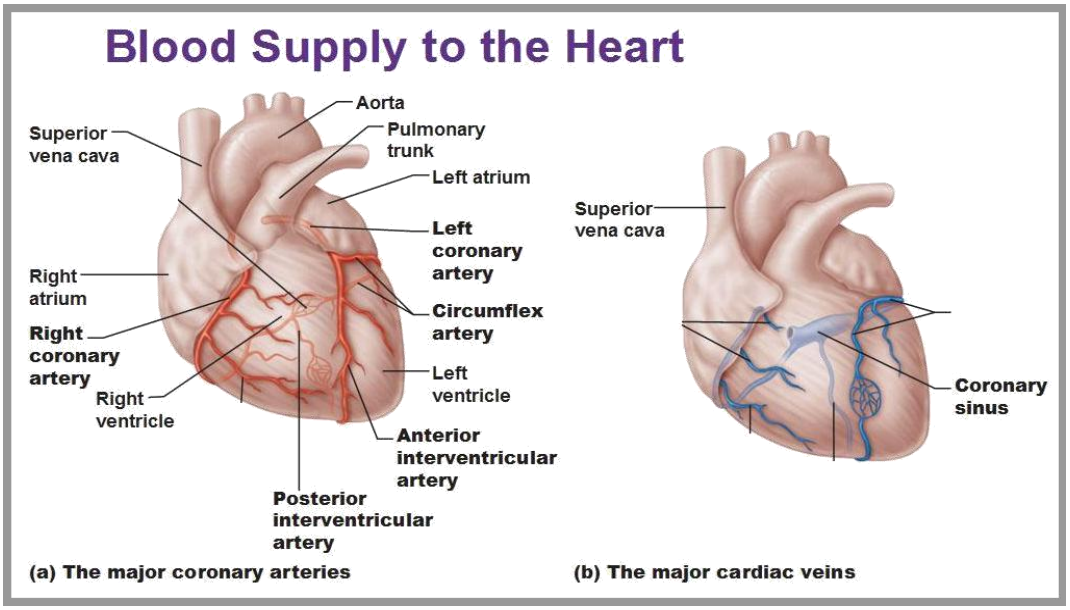


Figure 2: Blood supply of heart.

In anterior wall infarction, the heart block is usually related to ischemic malfunction of all three fascicles of the conduction system, and this commonly results only from extensive myocardial necrosis.[4]

RISK FACTORS FOR CLINICAL ATHEROSCLEROSIS:

Risk factors are classified into non modifiable and modifiable.

Non Modifiable Risk Factors:-

- Age
- Male gender
- Family history of congenital heart disease (CHD)
- Presence of CHD
- Menopause

Modifiable Risk Factors:-

- Dyslipidemia
- Hypertension
- Diabetes
- Abdominal obesity
- Smoking
- Diet

<u>Risk Factors for Asian Indians:-</u>

Non Modifiable:

- Male age >35 years
- Female age >45 years
- Family history of premature CAD (age <55 years)

Modifiable - Non Lipid:

- Hypertension
- Cigarette smoking/ tobacco abuse
- Diabetes mellitus / Insulin resistance syndrome.
- Apple obesity or body mass index >23
- Homocysteine >10 m mol/L

Modifiable - Lipid:

- Total cholesterol >150 mg / dl
- Triglycerides> 150mg/dl
- LDL cholesterol >100 mg/dl

- Apo A lipoproteins <100 mg / dl
- HDL < 40 mg/dl males, <50 mg/dl females

Modifiable: Lipoprotein Ratios

- TC/Hal > 4.5
- LDLc / HDL c > 3.5
- Apo A/ Apo B <1.2

Among all these smoking and Apo B/ApoA-1 are associated with increased incidence of MI in young patients.

Spectrum of Myocardial Ischemia:-

Ischemic Heart Disease can present as effort angina, unstable angina, non Q wave MI, Q wave MI, heart failure and sudden cardiac death.

Clinical features:-

Poorly localized retrosternal discomfort with radiation to neck, shoulders, arms, jaw, and epigastrium or back usually. Angina can be triggered by emotional activity, emotional stress, and exposure to cold, consuming meal or smoking. Pain is not specifically localized, chest discomfort may be squeezing type, burning, choking, heaviness, hot or cold sensation, dyspnea altered sensorium, and syncope these symptoms are called ―Angina equivalents‖.

Pain lasts for 2 minutes to 8 minutes. Ischemia seldom lasts more than 30 minutes, without causing AMI.

Pain relieved by rest and sublingual nitrates in 2-5 minutes. It is less likely to be angina , if it is not localized and less than 30 seconds or more than 30 minutes without AMI (except unstable angina).

Location of pain and its relation to exertion are two important factors in the history to determine pain. If both features are present chances of AMI is 90%. If one of the features is present chances of AMI is 50 %. If both are absent chances of acute mi is very less.

Decubitus angina is due to shift of blood volume into the lungs, nocturnal angina is associated with night mares. Prinzmetal angina is intense coronary vasospasm. It is atypical pattern of pain at rest, precipitated by cold, emotional stress and smoking.

Conduction System of Heart

Normal anatomy and physiology of the conduction system

The physiologic conduction system consists of the sinus node, the AV node, and the bundle of His including the right and left bundle branch as well as the Purkinje system. The conduction system can be considered as a hierarchy of pacemakers with the sinus node being the primary pacemaker of the heart.

The sinus node was first identified as the region responsible for the primary activation of the heart by Keith and Flack in 1907. It is a crescent-shaped structure which lies epicardial in the sulcus terminalis between the superior vena cava and the right atrium. Although the sinus node is often depicted as a small, localized area in medical textbooks, this is not consistent with electrophysiological findings.[5, 6]

According to experimental animal models (especially in rabbits), the sinus node is more likely to be a diffuse and extensive area between the superior and inferior vena cava.[5,7] It consists of spontaneously depolarizing pacemaker cells with a unique pattern of in channels necessary for the generation and the propagation of action potentials. The sinus node is supplied with blood via the sinus node artery

which originates from the right (about 60%) or the left (40%) circumflex coronary artery and approaches the sinus node from a clockwise or counterclockwise direction around the superior vena cava. [8]

It has long been believed that impulses from the sinus node are conducted to the AV node via 3 intra-arterial pathways (the anterior, middle and posterior internodal tract), but more recent studies suggest that atrial fiber orientation may account for preferred ways of conduction. [9]

Apart from patients with accessory pathways the AV node is the sole connection between the atria and the ventricles. Impulses from the atria to the ventricle are modulated by the AV node. One of the main functions of the AV node is to delay and to limit the number of atrial impulses reaching the ventricle. Furthermore, the inferior nodal extensions of the AV node can act as a subsidiary pacemaker in cases of AV block.[10]

The AV node is part of the AV junction which can be divided into three different regions based on the marked heterogeneity in action potential waveform: the transitional zone, the compact portion or the AV node itself and the penetrating part of the AV bundle (His bundle).[10] The compact portion of the AV node is located beneath the right atrial endocardium, anterior to the coronary sinus ostium and above the insertion of the septal leaflet of the tricuspid valve.[10] When entering the central fibrous tissue the AV node becomes the penetrating portion of the His bundle. Impulses are then conducted from the His bundle to the right and left bundle. The proximal part of the AV node is supplied by the AV nodal artery, whereas the distal part has a dual blood supply which makes it less vulnerable to ischemia. The AV nodal artery arises in 80% to 90% of humans from the right coronary artery and in 10% to 20% from the circumflex artery. Therefore, conduction abnormalities of the

AV node during acute myocardial infarction are usually caused by an inferior myocardial infarction.

The cardiac conduction system is innervated by a rich supply of both, the sympathetic and parasympathetic nervous system. Stimulation of the sympathetic nervous system increases automaticity, enhances conduction, and shortens refractory periods. The parasympathetic influence has the opposite effect. The conduction in the His bundle, though, is neither influenced by sympathetic nor by vagal stimulation.[11]

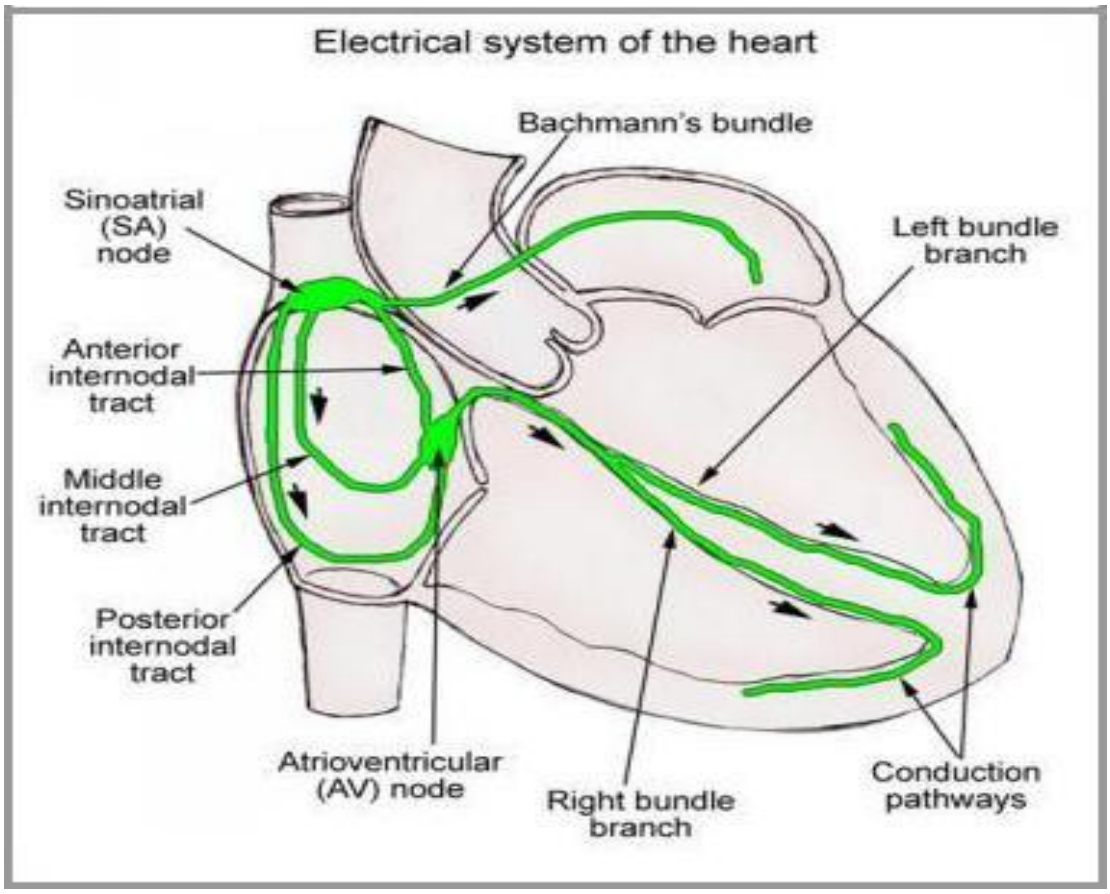


Figure 3: Normal Conduction System of Heart

Sinus Node Dysfunction

SND (also called sick sinus syndrome in symptomatic patients) comprises a variety of disturbances affecting sinus node impulse generation and transmission within the atria and may lead to bradarrhythmias but also tachyarrythmias. It is sort of a spectrum of disorders. Possible electrocardiographic manifestations are:

1. Sinus pauses or arrest.
2. Sinoatrial exit block
3. Chronotropic incompetence
4. Atrial tachycardia (including atrial fibrillation or atrial flutter), and
5. Bradycardia-tachycardia syndrome.

Today, SND is still one of the major causes of pacemaker implantations other than AV block. It accounts for approximately 50% of pacemaker implantations in the United States, between 30-50% in Europe, and approximately 40% in Spain in 2009 and 2010.[11, 12] In general, SND is a chronic progressive disorder and primarily occurs in the elderly with the incidence doubling between the fifth and sixth decades of life and the peak incidence in the seventh and eighth decades of life. [13, 14] although exact numbers on the incidence of SND are unavailable. SND is estimated to occur in 150 to 200 patients per million people.[15]

Sinus pause/ Sinus arrest:

Failure of atrial activation due to inability of sinus node to made impulses [16]. Distinctive features of sinus pause is absence of ―p‖ waves for more than 1.5 seconds. Sinus arrest or pauses imply failure of an expected atrial activation. [17] This may be due to a problem of impulse generation in the sinus node or a failure of impulse conduction to the atrium. Though there are currently no cut-off values, pauses of 3s or

more are uncommon and warrant implantation of a pacemaker in symptomatic patients. Pauses of 3s or more, however, do not seem to be predictive of heightened mortality according to a newer study.[17]More severe form of sinus pause lasting more than 3 seconds.

Sino Atrial block:

Sino atrial exit block occurs when there is failure in conduction of sinus impulse into atria. Absence of normal ―p‖ waves and duration of pause recognizable as an exact multiple of preceding PP interval, unlike sinus pause/ arrest.

Pathophysiology of Sinus Node Dysfunction:

SND can result from various conditions, which cause depression of the automaticity in and electrical conduction from the sinus node, perinodal and atrial tissue. These conditions may be intrinsic (diseases that directly alter the sinus node or sinoatrial structure) or extrinsic (most often cardiovascular drugs or systemic illnesses such as sleep apnea). The most common cause of SND is idiopathic degenerative fibrosis of nodal tissue which is associated with aging.[18, 19] Fibrosis is thought to lead to a loss of pacemaker cells and a shift from central to inferior pacemaker cells within the sinus node.[20] Spontaneous diastolic depolarization is slower in those cells, which results in bradycardia.

Diagnosis of Sinus Node Dysfunction

Due to the predominantly intermittent and often unpredictable nature of SND this can be very difficult. Apart from a thorough medical history, a 12-lead surface ECG, Holter ECG recording (long-term ECG), and exercise testing are usually

adequate. Whenever surface ECG and repetitive Holter recordings are incapable of documenting the cause of a patient_s symptoms, an external event recorder or an implantable loop recorder should be considered. In patients with symptoms occurring more than once a month an external event recorder which can be kept for a maximum of 30 days is often sufficient. An implantable loop recorder may be used in patients with infrequent and transient symptoms in whom none of the aforementioned electrocardiographic recordings could achieve diagnostic information.

Treatment of sinus node dysfunction:

Treatment should be restricted to those patients in whom a strong symptom-rhythm correlation has been documented.[21] Patients with asymptomatic SND do not require specific treatment. The first step is to rule out or treat reversible extrinsic causes of SND and to exclude physiologic sinus bradycardia. Pharmacologic therapy is not effective in SND. If there are no reversible conditions causing SND, cardiac pacing should be implemented to relieve symptoms. The mode of pacing has been a subject of numerous studies (Pacemaker Selection in the Elderly trial Canadian Trial of Physiological Pacing[22]Mode Selection Trial in Sinus-Node Dysfunction[23] Danish trial[24]).

Taking into account that atrial tachyarrhythmia‘s, particularly atrial fibrillation, are common in patients with SND and thromboembolism is the most important cause of mortality in SND, oral anticoagulation should be considered in each patient with SND and a history of intermittent tachycardia‘s. Oral anticoagulation should be implemented according to the latest ESC guidelines for the management of atrial fibrillation[25].

Prognosis of SND:

The natural course of SND can be highly variable and is often unpredictable. However, patients with a history of syncope due to SND are likely to have recurrent syncope. Development of concomitant complete AV block is considered to be low with a median annual incidence of 0.6% (total prevalence of 2.1%) and so does not dominate the clinical course of SND. The incidence of sudden death seems to be low, too, and pacemaker therapy does not seem to improve overall survival, but improves morbidity. Progression and prognosis of SND depend on several factors: age, coexistent cardiovascular diseases, concomitant AV conduction block, and atrial fibrillation resulting in a higher risk of thromboembolic complications.

Disorders of AV Conduction

AV conduction block is a disorder in which atrial impulses are conducted with a delay or are not at all conducted to the ventricles at a time when the AV conduction pathway is not physiologically refractory. Historically, it was the first indication for cardiac pacing and still remains the major reason (approximately 50%) for pacemaker implantation. The incidence of AV conduction disturbances increases with age and is estimated to be up to 30% in selected groups.[26]

Based on ECG criteria, AV block is traditionally classified as first, second, or third-degree (complete) AV block.

First degree AV block:

By convention, first degree AV block is defined as an abnormal prolongation of the PR interval (>0.2s). Every P wave is followed by a QRS complex, but with a

constantly prolonged PR interval. Prolongation of the PR interval can derive from delayed conduction within the atrium, AV node (AH interval) or His-Purkinje system (HV interval) but most commonly is due to delayed conduction within the AV node. Patients with first-degree AV block are usually asymptomatic. However, if a marked prolongation of the PR interval (>0.3s) occurs (Figure 1) patients may suffer from a pacemaker-like syndrome owing to AV dyssynchrony. Many of these patients are particularly symptomatic during exercise because the PR interval does not shorten appropriately as the R-R interval decreases.

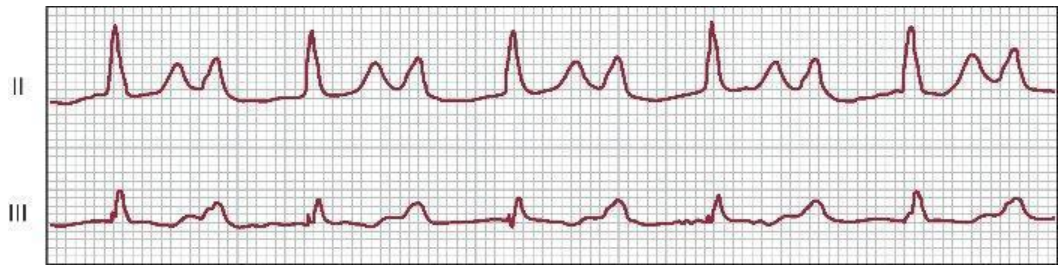


Figure 4: An example of a patient with asymptomatic first-degree atrioventricular block with marked prolongation of the PR interval (PR 0.4 s).

Second-Degree AV Block

The term second-degree AV block is applied when intermittent failure of AV conduction occurs. Second-degree AV block can be divided into 2 types based on ECG patterns: type I (Mobitz I or Wenckebach) and type II (Mobitz II). This classification should not be used to describe the anatomical site of the block because the terms type I and type II only refer to a certain ECG conduction pattern. To avoid mistakes and pitfalls often associated with the diagnosis of second-degree AV block, it is important to adhere to a correct definition[27].

The classic Mobitz type I second-degree AV block is characterized by a progressive PR interval prolongation prior to the non-conducted P wave (Wenckebach behavior).

The first conducted P wave after the non-conducted P wave has the shortest PR interval of such a cycle and so the pause between the QRS complexes encompassing the non-conducted P wave will be less than twice the P-P interval.[28] With stable sinus rhythm, the block cycle normally has a fixed P: R ratio (in classic type I ratios of 3:2, 4:3 or 5:4). However, many type I second-degree AV block sequences are atypical and do not show the classical progressive prolongation of the PR interval.[29] (Figure 2)

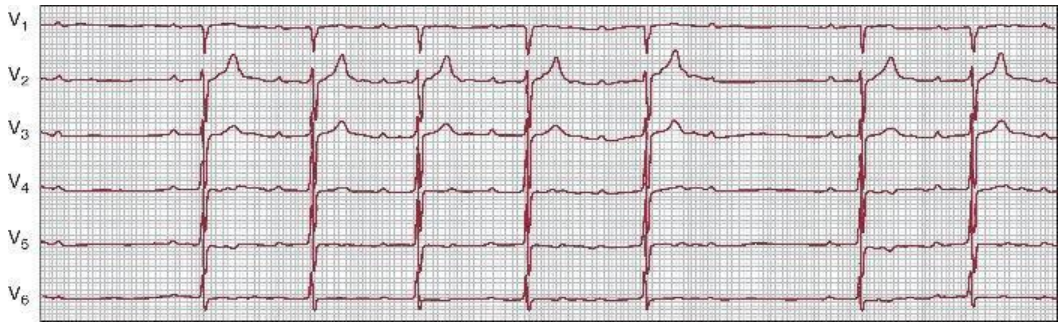


Figure 5: Atypical second-degree Mobitz type I (Wenckebach) atrioventricular block with a 6:5 ratio.

According to the statements of the World Health Organization and the American College of Cardiology a more appropriate definition of type I second-degree AV block is occurrence of a single non conducted P wave associated with inconstant PR intervals before and after the blocked impulse as long as there are at least 2 consecutive conducted P waves (i.e., 3:2 AV block) to determine the behavior of the PR intervals.[39]

Type II second-degree AV block (Figure 4) is defined as the occurrence of a single non-conducted P wave associated with constant PR intervals before and after a single blocked impulse (PP and RR intervals are constant) [30,31]. The pause encompassing the blocked P wave equals 2 P-P cycles. Type II second-degree AV block typically occurs in conjunction with intraventricular block.

2:1 AV Block:

With only one PR interval before the blocked P wave a 2:1 AV block (Figure3), also called ―ADVANCED AV BLOCK‖, cannot be classified as type I or II second degree AV block based on a single (short) recording of the surface ECG. The anatomic site of the block can be in the AV node or in the His-Purkinje system and both type I or II second-degree AV block can progress or regress to a 2:1 block. The presence of intraventricular block indicates a block distal to the AV node, whereas a block with a small QRS complex is usually within the AV node. Considering that second-degree AV block type II is a class I indication for permanent pacing it is of huge therapeutic importance to make the exact diagnosis. Recording a long surface ECG strip, carotid sinus pressure test as well as giving atropine or exercise can reveal the correct type of second-degree AV block. If Wenckebach cycles are observed during long-term ECG recording (or sometimes during longer recordings of the standard ECG) of a patient with 2:1 AV block, this serves as an indication that in this case, 2:1 AV block most probably is the extreme form of a Wenckebach cycle.

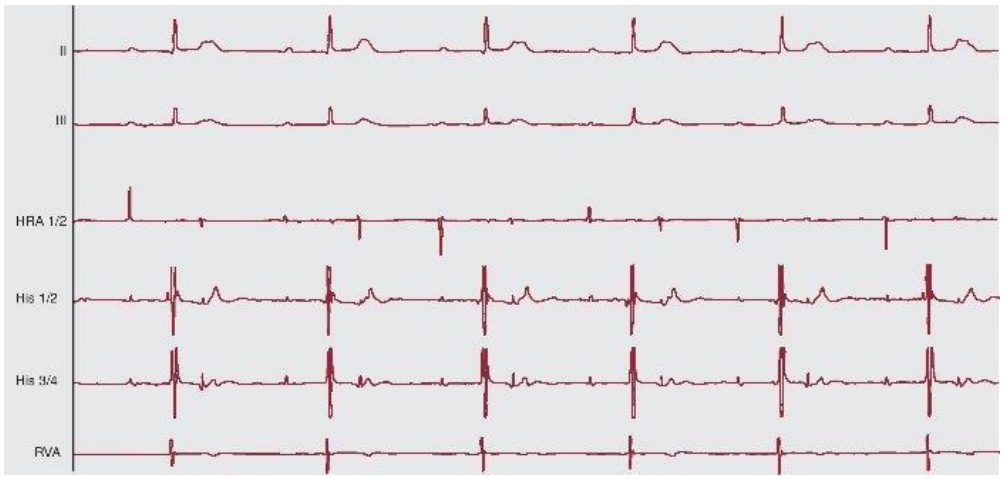


Figure 6: A 15-year-old patient with second-degree atrioventricular.

Third-degree AV block:

Third-degree or complete AV block is characterized by the failure of each P wave or each atrial impulse to conduct to the ventricle resulting in complete AV dissociation with atrial rates higher than the ventricular ones (Figure 4).

It can be congenital or acquired and can be localized to the AV node, the His Bundle, or the ramifications of the right and left bundles. The ventricular escape rhythm reveals the anatomic site of the block: complete AV block with an escape rhythm of 40 to 60 bpm and a narrow QRS complex on surface ECG is usually within the AV junction, which is often seen in congenital AV block (Figure 2). A wide QRS complex and/or a rate of 20 to 40 bpm imply a block in the His-Purkinje system, which is most often the case in acquired AV blocks.

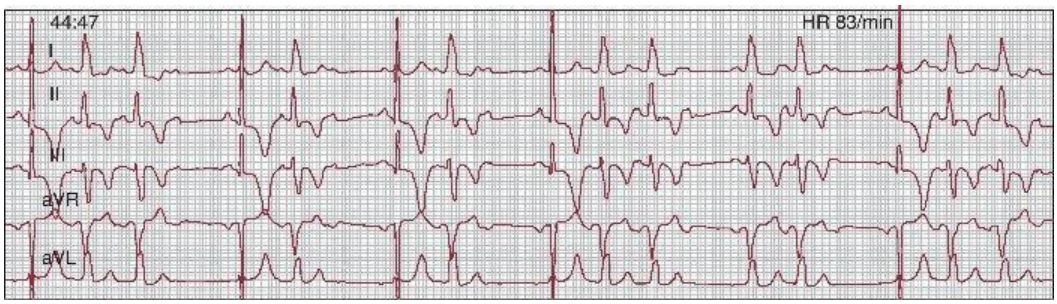


Figure 7: An example of third-degree atrioventricular block with complete atrioventricular dissociation and an atrioventricular junctional escape rhythm with narrow QRS complexes.

Etiology and Pathophysiology of AV Block

Acquired AV block can be caused by a number of extrinsic and intrinsic conditions which were already discussed with SND. Idiopathic progressive degeneration of the cardiac conduction system, referred to as Lenegre or Lev disease, accounts for approximately one half of cases of AV block. In addition to the causes listed under SND progressive AV conduction disturbances may be seen in

neuromuscular disorders (muscular dystrophy, Kearns-Sayre syndrome), systemic diseases (e.g., cardiac sarcoidosis, amyloidosis), neoplastic disorders (i.e., primary cardiac lymphoma [32], and/ or post radiation therapy), or after catheter ablation of septal accessory pathways or slow or fast AV pathway for AV nodal reentrant tachycardia. In younger individuals, Lyme disease should always be considered as a possible reversible cause of AV block. Congenital complete AV block may occur as isolated disease which is frequently due to intrauterine exposure to maternal antibodies (Rho, La) or may be associated with any congenital heart disease.

Pathologically, there are 4 types of congenital AV block: lack of connection between the atria and the peripheral conduction system, interruption of the AV bundle, bundle branch disease, and abnormal formation or interruption of the AV bundle. Complete AV block is a relatively frequent manifestation of the rare entity of congenitally corrected transposition of the great arteries.

Diagnosis of AV block:

Patients presenting with advanced AV block generally complain of dizziness, vertigo and/or syncope, but may also suffer from any of the above mentioned symptoms of brad arrhythmias. Diagnosis of AV block can be achieved in most of these cases noninvasively. The surface ECG (if the recording is sufficiently long) usually provides the information to characterize the type and localize the level of the block. In patients with intermittent AV block, Holter ECG and exercise testing are important to establish a correlation between symptoms and rhythm. With rare exceptions such as persistent 2:1 AV block or failure to establish a symptom-rhythm correlation, invasive electrophysiological study does not make a significant contribution to the management of patients with complete AV block.

Treatment of AV Blocks

As with SND, treatment of AV block should start with looking for potentially reversible causes as for example Lyme disease or myocardial ischemia. Drugs resulting in a conduction delay within in AV node (e.g., digitalis, calcium channel blockers) should be discontinued, if possible. In the acute setting, symptomatic AV block can be treated with intravenous vagolytic agents as atropine and/or catecholamine (orciprenalin). If these drugs are not effective, a temporary pacemaker is indicated. In the emergency treatment of severe symptomatic brad arrhythmias (no escape rhythm) transcutaneous stimulation may be applied. Transient and permanent cardiac pacing is the definite therapy of choice in most cases of symptomatic complete AV block. The indication depends on the type and location of the AV block, present symptoms, the prognosis, and concomitant diseases.

Clinical indication for pacing:

1. Chronic symptomatic third or second-degree (Mobitz I or II) Atrioventricular block
2. Neuromuscular diseases (e.g., myotonic muscular dystrophy, Kearns-Sayre syndrome, etc.) with third- or second-degree atrioventricular block
3. Third- or second-degree (Mobitz I or II) atrioventricular block
4. After catheter ablation of the atrioventricular junction
5. After valve surgery when the block is not expected to resolve
6. Asymptomatic third- or second-degree (Mobitz I or II) atrioventricular block
7. Symptomatic prolonged first-degree atrioventricular block
8. Neuromuscular diseases (e.g., myotonic muscular dystrophy, Kearns-Sayre syndrome, etc.) with first-degree atrioventricular block

9. Asymptomatic first-degree atrioventricular block
10. Asymptomatic second-degree Mobitz I with supra-Hisian conduction block
11. Atrioventricular block expected to resolve

Patients with first-degree AV block usually do not need cardiac pacing. If the PR interval, though, fails to adapt to heart rate during exercise and is long enough (most often > 0.3 s) to cause symptoms due to loss of AV synchrony, implantation of a DDD pacemaker should be considered (class II a).Asymptomatic type I second-degree AV block (Wenckebach) is almost always considered a benign condition with excellent prognosis in young persons or well-trained athletes at rest. However, some controversy exists about the prognosis and the need for permanent pacing of chronic type I second-degree AV block in elderly patients (>45 years). Thus, older patients with asymptomatic type I second-degree AV block should at least be monitored closely. In patients with congenital complete AV block, the decision to implant a pacemaker is usually based on several factors including its natural history, the patient's age (significance of bradycardia is age-dependent) and symptoms, and the concomitant structural/congenital heart disease. The indications for permanent cardiac pacing in congenital complete AV block are still evolving. However, there is a consensus among pediatricians that the presence of an underlying severe heart disease, symptoms, and a heart rate below 50 to 55 bpm are an indication to implement cardiac pacing. Nowadays, we also know that even asymptomatic patients with isolated congenital heart disease have an unpredictable risk of syncope, so that pacing should be strongly considered in each patient with congenital complete AV block.

Prognosis of AV block:

The prognosis of patients with AV conduction disturbances depends on the site of the block, but also particularly on the concomitant or underlying heart disease. The natural history of the different types of AV block dates back to the era before pacemaker therapy was available as there is no alternative therapy for patients with symptomatic AV block.

First-degree AV block carries an excellent prognosis because the risk of progression to third-degree AV block is extremely low. Controversy exists about the prognosis of chronic, type I second-degree AV block as mentioned above. In healthy young patients with normal QRS width, it is considered to be a benign condition. In older patients (>45 years) and in patients with associated bundle branch block suggesting an infranodal location prognosis seems to be worse compared with age- and sex-matched individuals unless a pacemaker is implanted. The natural course of type II second-degree AV block is characterized by a high rate of progression to complete AV block. Patients have a significantly lower 5-year survival rate than patients who had a pacemaker implanted for second-degree AV block.

Prognosis of patients with congenital AV block is largely dependent on the presence of congenital heart disease and time of diagnosis. The prognosis of isolated congenital complete heart block is a more favorable one compared to those with concomitant structural heart disease. However, the stability of escape rhythms and the incidence of syncope are unpredictable. Cardiac pacing should be strongly considered even in asymptomatic patients with isolated congenital AV block. The occurrence of complex ventricular arrhythmias may also argue for pacemaker implantation in asymptomatic individuals.

Intraventricular conduction abnormalities

Intraventricular conduction abnormalities including right bundle branch, left bundle branch, fascicular block, or a combination of these are commonly seen on routine ECG of elderly patients but may also been seen in younger patients either as an isolated finding or in association with dilative cardiomyopathy. The incidence was estimated to be 11% in men and 5% in women over 60 years according to an analysis of the Framingham study and is increasing with age.

Pathophysiology of IV Block:

Intraventricular conduction abnormalities and bundle branch blocks can be due to ischemia, i.e., in myocardial infarction, after cardiothoracic surgery or can be mechanically induced after (mostly) aortic valve replacement surgery and after transcatheter aortic valve implantation. It can also be the consequence of surgery in congenital heart disease. Left bundle branch block (LBBB) that is defined by a prolongation of QRS above 0.11s in combination with a delay of the intrinsic deflection in leads V_5 and V_6>60 ms (and no septal Q waves in leads I, V_5 and V_6) often occurs in association with dilative cardiomyopathy. However, the majority of chronic bundle branch block is idiopathic and seems to be associated with fibrosis of the conduction system, though only a few studies have investigated the underlying pathophysiology.

Prognosis of IVB:

Bundle branch block (especially LBBB) and bifascicular block are generally associated with a higher mortality compared to sex and age matched control persons, but some conditions such as isolated right bundle branch block are considered to be

benign. The higher mortality is rather explained by the associated heart disease, especially coronary artery disease, than by the conduction abnormalities. However, LBBB itself may be a cause or an aggravating factor in left ventricular systolic failure due to the reduced pumping performance which results from asynchronous electrical activation of the ventricles in LBBB. In some cases a LBBB may be the first sign of a developing latent dilated cardiomyopathy. The annual incidence of progression to advanced or complete AV block and so the risk of death from Brady arrhythmias is low. Syncope and death seem to result more often from tachyarrhythmias and/or myocardial infarction than from conduction abnormality itself.

Diagnosis of IVB:

The ECG and the Holter ECG (in intermittent conduction delay) provide the information to identify the type of conduction delay. In patients with intraventricular conduction delays and a history of syncope invasive electrophysiological study may be helpful. If the HV interval is more than 100ms, implantation of a pacemaker should be discussed. According to the 2007 ESC guidelines an electrophysiological study is also pathologic, if a high-degree His-Purkinje block is unmasked by intravenous administration of ajmaline.

Furthermore, every patient with bundle branch block should be evaluated for an underlying structural heart disease due to the high incidence of coronary artery and / or hypertensive heart disease. In general, the incidence is higher with left bundle branch than with right bundle branch.

Therapy of IVB:

Because of the low incidence of complete AV block, asymptomatic patients with isolated right or left or bifascicular block with or without first-degree AV block (often wrongly referred to as ―trifascicular‖ block) do not require permanent cardiac pacing. According to the ESC guidelines, a cardiac pacemaker should be implanted in patients with true trifascicular block (i.e., alternating bundle branch block), chronic bifascicular block, and second-degree. Mobitz II AV block, or intermittent complete AV block.

Clinical indication for pacing:

1. Intermittent third-degree atrioventricular block
2. Second-degree Mobitz II atrioventricular block
3. Alternating bundle branch
4. Findings on electrophysiological study of markedly prolonged HV interval (≥100 ms) or pacing-induced infra-His block in patients with symptoms
5. Syncope not demonstrated to be due to atrioventricular block when other likely causes have been excluded, specifically ventricular tachycardia
6. Neuromuscular diseases (e.g., myotonic muscular dystrophy, Kearns-Sayre syndrome, etc.) with any degree of fascicular block
7. Incidental findings on electrophysiological study of markedly prolonged HV interval (≥100 ms) or pacing-induced infra-His block in patients without symptoms
8. Bundle branch block without atrioventricular block or symptoms
9. Bundle branch block with first-degree atrioventricular block without symptoms

Apart from brady arrhythmias patients with LBBB and dilative cardiomyopathy should be evaluated for cardiac resynchronization therapy.

Brady arrhythmias associated with AMI:

Brady arrhythmias arising in the setting of acute myocardial infarction are common and result from abnormalities in impulse formation or impulse conduction.[32] Sinus bradycardia is one of the most common rhythm disorders related to myocardial infarction, especially in right coronary involvement (about 30%-40%).[33,34] The major conduction abnormalities associated with myocardial infarction are AV and intraventricular conduction disorders. Despite new techniques such as thrombolysis and percutaneous coronary intervention the incidence of intraventricular conduction disturbances has not changed significantly; the absolute incidence of AV block, however, has decreased but remains still high. [34,] AV block occurs in 6% to 7% of cases of acute myocardial infarction and is 2 to 3 times as commonly associated with inferior than anterior infarction.[34,] Intraventricular conduction delays occur in a transient form in up to 18% of patients and in approximately 5% in a persistent form.

Pathophysiology of Brady arrhythmias Associated With AMI:

The pathophysiologic mechanisms underlying most bradyarrhythmias in myocardial infarction are: reversible ischemia, irreversible necrosis of the conduction system, or other conditions like altered autonomic function, such as increased parasympathetic tone, electrolyte disturbances, systemic hypoxia, or local increases in adenosine.¯According to histologic studies, obvious structural damage to the conduction system (necrosis) seems to be rare and is usually due to an extensive anterior myocardial infarction with necrosis of the septum.

Treatment of Bradyarrhythmias Associated With AMI:

Acute management of symptomatic high-grade AV block includes intravenous drugs such as atropine or temporary cardiac pacing. Implantation of a permanent cardiac pacemaker is rarely necessary in acute myocardial infarction, especially in inferior myocardial infarction because truly persistent AV block is uncommon. Recommendations for permanent cardiac pacing according to the ESC are

1. Persistent third-degree heart block preceded or not by intraventricular conduction disturbances.
2. Persistent Mobitz type II second-degree heart block associated with bundle branch block, with or without PR prolongation.
3. Transient Mobitz type II second- or third-degree heart block associated with new onset bundle branch block.

. The huge problem with the recommendations for cardiac pacing in acute myocardial infarction is the definition of ―persistent‖. According to the ESC guidelines conduction disturbances are persistent if they do not resolve after more than 14 days. However, this has been and still is a subject of discussion.

Prognosis of bradyarrhythmias associated with AMI:

Despite the use of thrombolytic therapy and of percutaneous coronary intervention, AV block, and intraventricular conduction disturbances complicating acute myocardial infarction are still associated with a high risk of short-term mortality.[33, 34, 35]

Literature from the pre thrombolytic era have shown that intraventricular and atrioventricular conduction defects were associated with a greater in hospital morbidity and mortality in patients with acute myocardial infarction.

The impact of thrombolytic therapy on acute myocardial infarction mortality has been widely confirmed by scientific evidence.. A few literature reports have suggested that reperfusion of an artery related to the infarction can lower the incidence of conduction disturbances, because thrombolysis decreases the size of the infarction. Others have suggested that the appearance of complete atrioventricular block may be signaling successful reperfusion.

However, the prognosis associated with intraventricular and atrioventricular conduction disturbances has been viewed with reserve during this era of thrombolytic therapy.

By 2020 it is estimated that ACS will become a major cause of death in all the regions of the world. Many of these deaths are attributed to the development of arrhythmias during periods of myocardial infarction.[36]

Conduction defect is an independent prognostic factor and is an indicator of mortality in AMI[37.] Early recognition and prompt treatment will definitely reduce the mortality in AMI due to conduction blocks, [38] as Conduction defects are common even in this thrombolytic era.[39] Sometimes thrombolysis may not be required in AV block it may be due to vagal mediated benign AV Block, it gets relieved on itself it is a benign condition.[40] Hence our study is to assess prognosis of AV block versus intraventricular blocks which are most common blocks in acute myocardial infarction and have better prognosis when diagnosed early.

MATERIALS AND METHODS

MATERIALS AND METHODS

Source of data:

- It is a prospective and comparative cohort study.
- Seventy two patients admitted in RLJH diagnosed as acute myocardial infarction who are with AV conduction blocks and myocardial infarction with intraventricular conduction blocks are included in the study. That is 36 patients with acute myocardial infarction with atrioventricular conduction blocks compared with 36 patients of myocardial infarction with intraventricular conduction blocks.
- Seven days follow up is done to assess the prognosis of AV blocks versus intraventricular conduction blocks in acute myocardial infarction.
- This is done by assessing following variables like area of infarction in myocardium, Killips staging, hypotension, mortality and ejection fraction of left ventricle in the two groups of patients.

Inclusion Criteria:-

1. Patients satisfying W.H.O criteria of myocardial infarction , that is 2 of the 3 of following [41]
   a) Symptoms of ischemia.
   b) Evolutionary ECG changes are seen
   c) A rise of cardiac markers

The diagnosis of arrhythmia was carried out as per AHA guidelines and treated accordingly

2. Patients above the age of 18 years are included in the study
3. Patients having myocardial infarction with atrioventricular blocks (AV blocks) and intraventricular conduction blocks (IV blocks).

Exclusion criteria:-

1. Patients with preexisting conduction blocks.
2. Patients with preexisting heart disease (congenital heart disease, cardiomyopathy, rheumatic heart diseases).
3. Patients taking drugs that cause conduction blocks like clonidine, methyldopa, verapamil and digoxin.

Method of collection of data:

After taking history of patients and doing clinical examination investigations including ECG, serum cardiac markers and ECHO are done, acute myocardial infarction diagnosed based on above mentioned criteria. Follow up for a period of 7 days after acute myocardial infarction by series of ECG'S and the complications are observed. All data for 7 days prognosis is analyzed and assessed. The assessment is based on following variables they are area of infarction, mortality, hypotension, Killips staging and ejection fraction. Few pictures of the machines used for investigations are as follows.

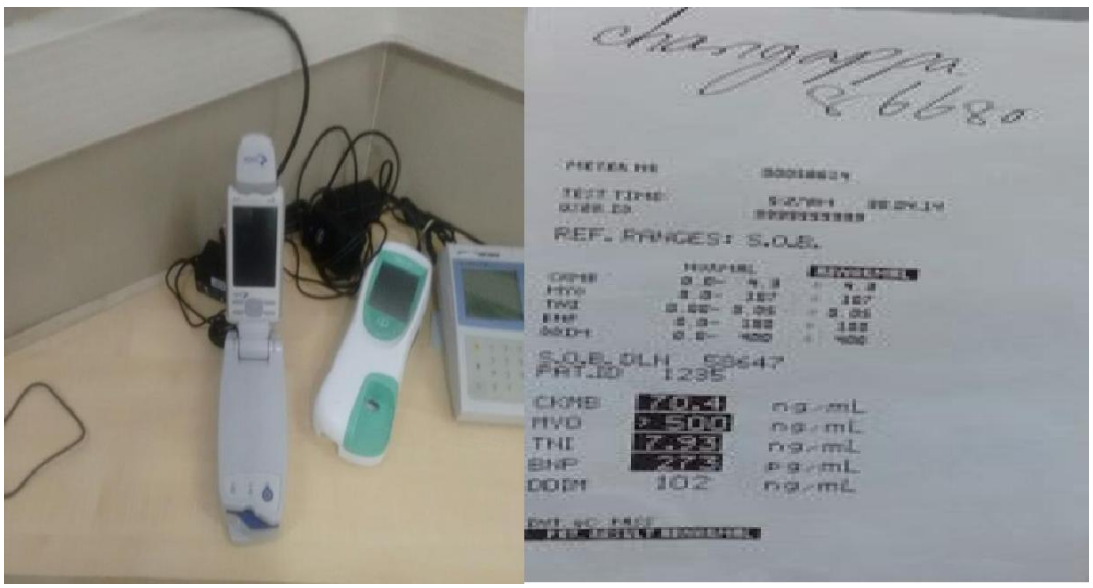


FIGURE 8: Troponin Analyzer and Report

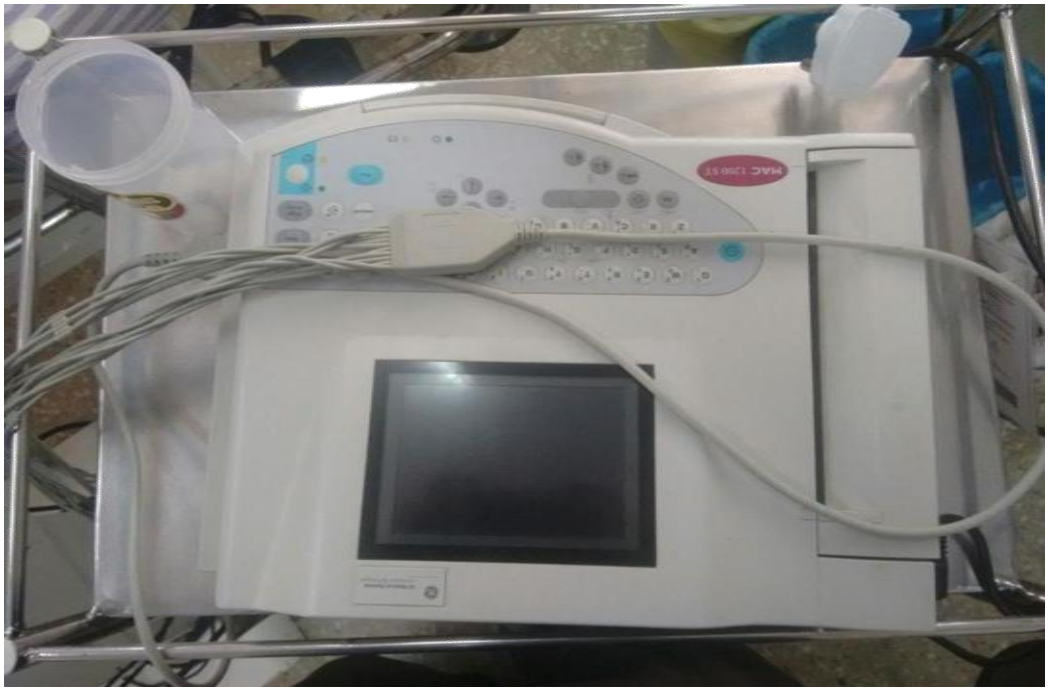


FIGURE 9: ECG machine

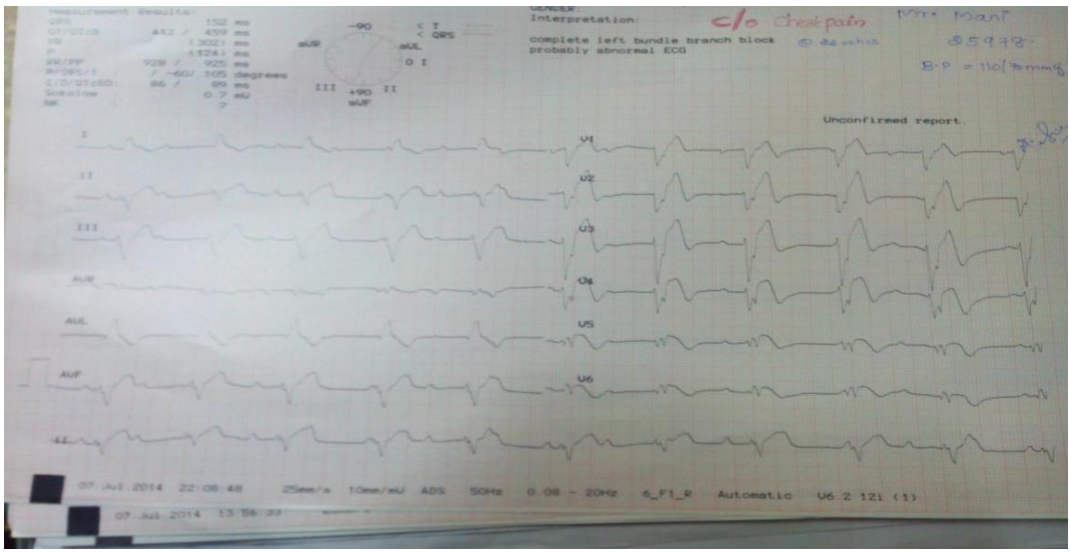


FIGURE 10: ECG SHOWING LBBB

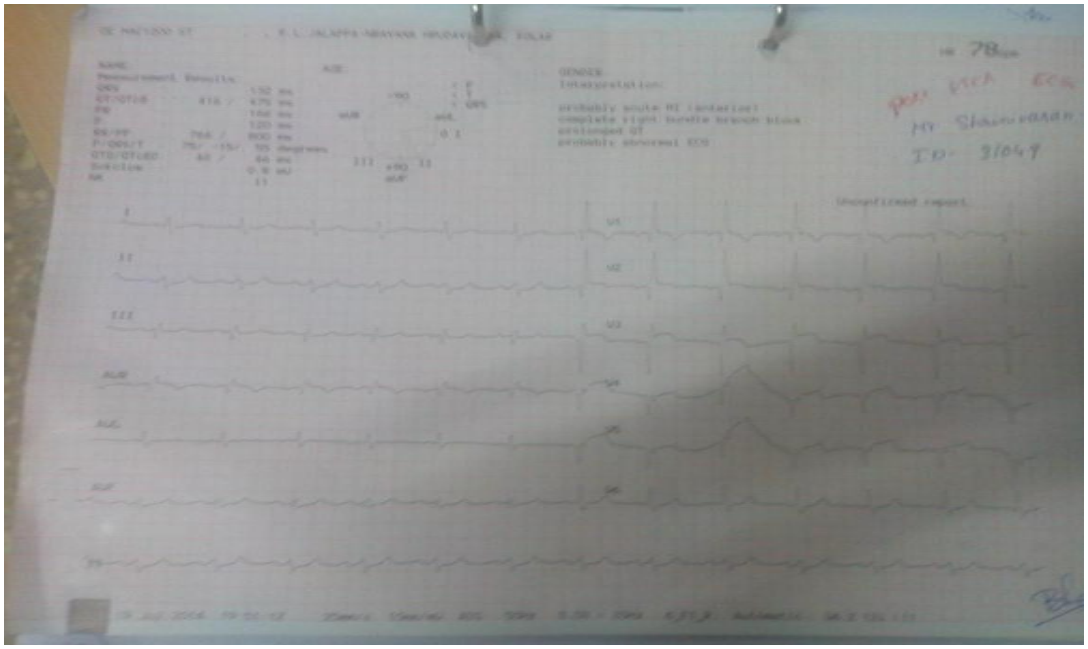


FIGURE 11: ECG SHOWING RBBB

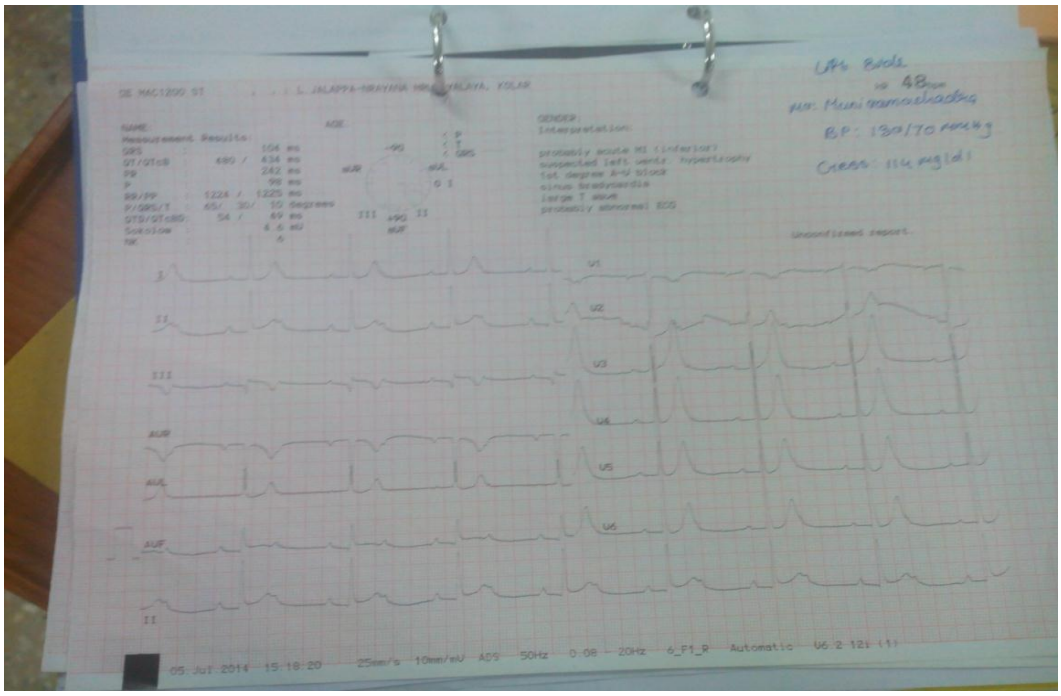


FIGURE 12: ECG SHOWING FIRST DEGREE AV BLOCK

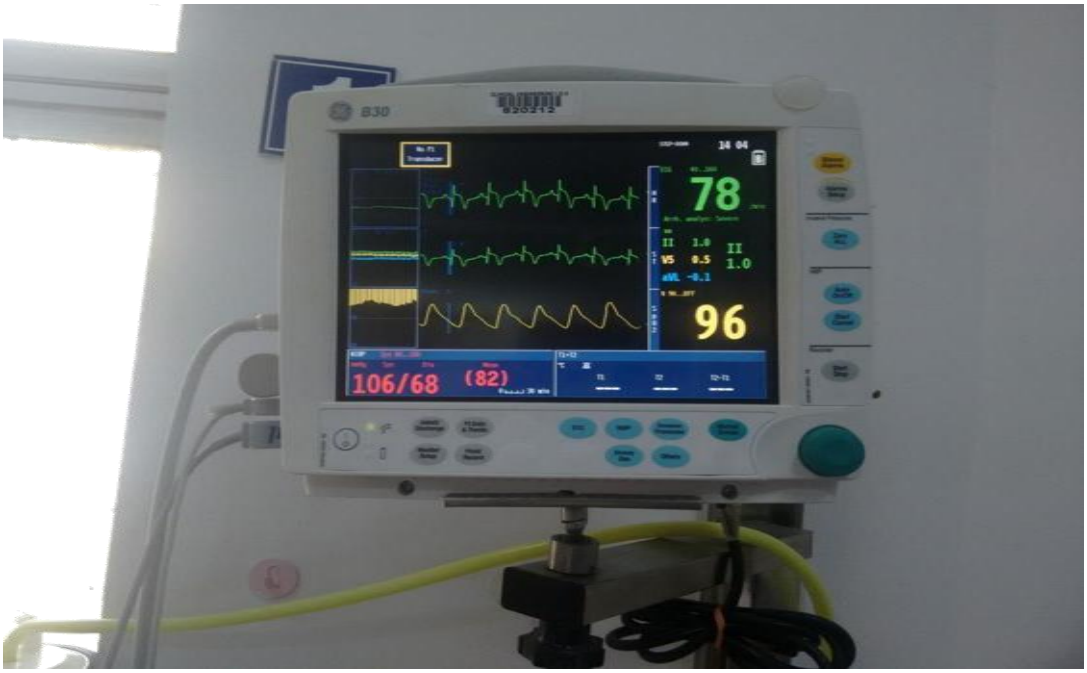


FIGURE 13: Cardiac Monitor

FIGURE 14: Taking ECG for A Patient in Cardiac Care Unit

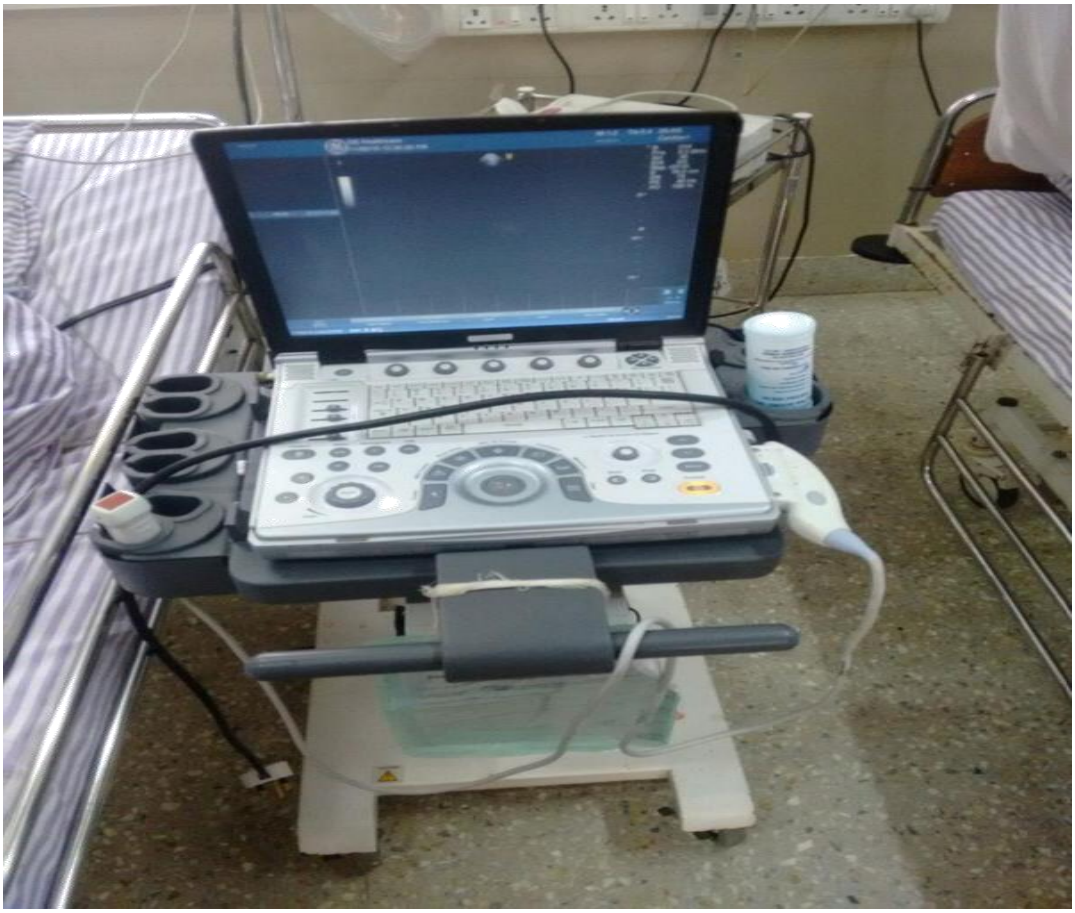


FIGURE 15: 2D ECHO machine

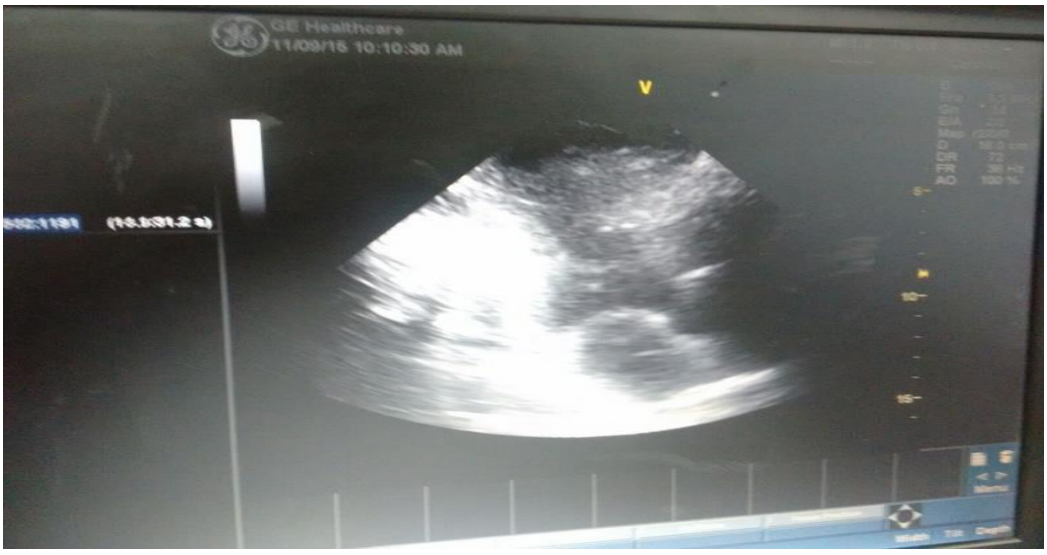


FIGURE 16:2D ECHO four chambered view with inferior wall hypokinetic.

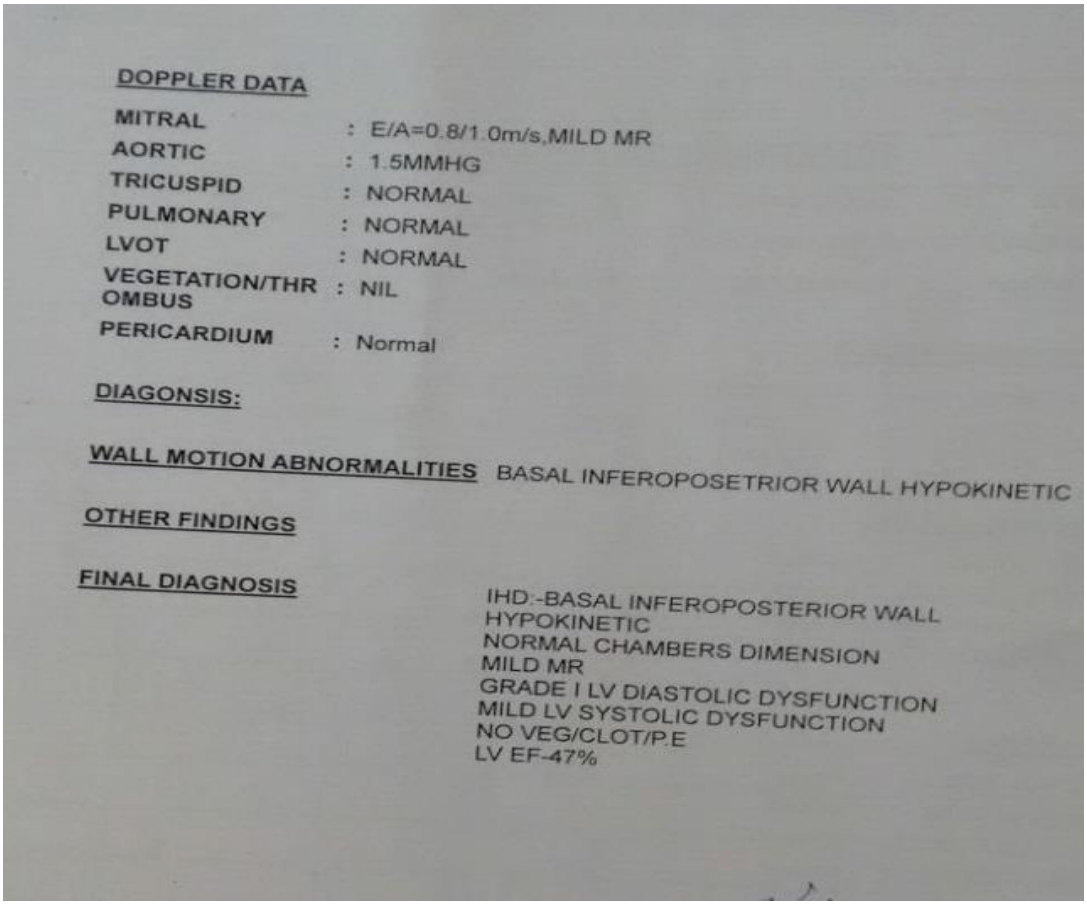


FIGURE 17: 2D ECHO report of patient

Sample size:

As per the previous studies of conduction blocks taking prevalence of conduction blocks of 15.8% of conduction blocks in acute myocardial infarction. Using formula

$$n= 4pq/d^2$$

Sample size is around 52. At 95% confidence limit. Considering 90% confidence limit sample size of 36 patients is taken in each group and compared.

Study design:

It is a prospective cohort study.

Statistical Methods:

- Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software.
- Categorical data was represented in the form of Frequencies and proportions.
- Chi-square was used as test of significance.
- Continuous data was represented as mean and SD.
- Independent t test was used as test of significance to identify the mean difference between two groups.
- Mann Whitney U test was used for quantitative variables not following normal distribution.
- P value <0.05 was considered as statistically significant.

RESULTS

RESULTS

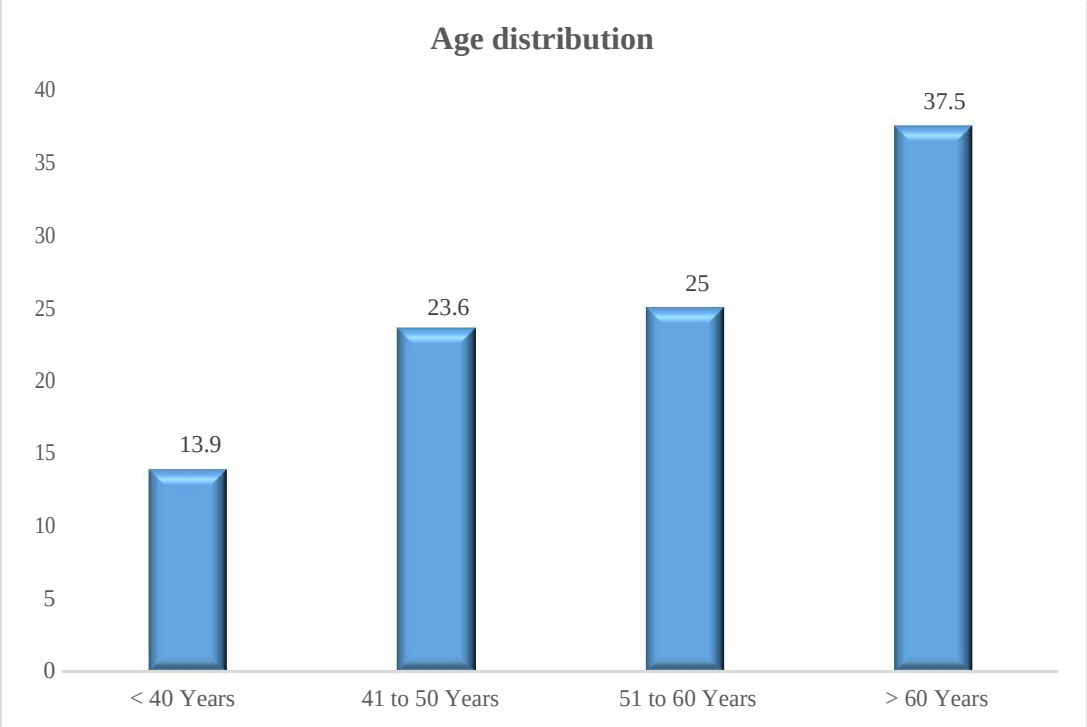
A sample of 72 patients are taken under the study out of them 36 are AMI with AV blocks and 36 are AMI with IVB both are analyzed and compared in variables like age, gender, blood pressure, area of infarction, ejection fraction and mortality.

Table 1: Age distribution of subjects

		Frequency	Percent
Age	< 40 Years	10	13.9
	41 to 50 Years	17	23.6
	51 to 60 Years	18	25.0
	> 60 Years	27	37.5
	Total	72	100.0

Age		
Mean		57.00
Median		57.50
Std. Deviation		13.121
Range		55
Minimum		30
Maximum		85
Percentiles	25	46.50
	50	57.50
	75	65.00

Mean age of subjects in the study was 57 ± 13.12 years. Majority of subjects were in the age group > 60 years (37.5%).

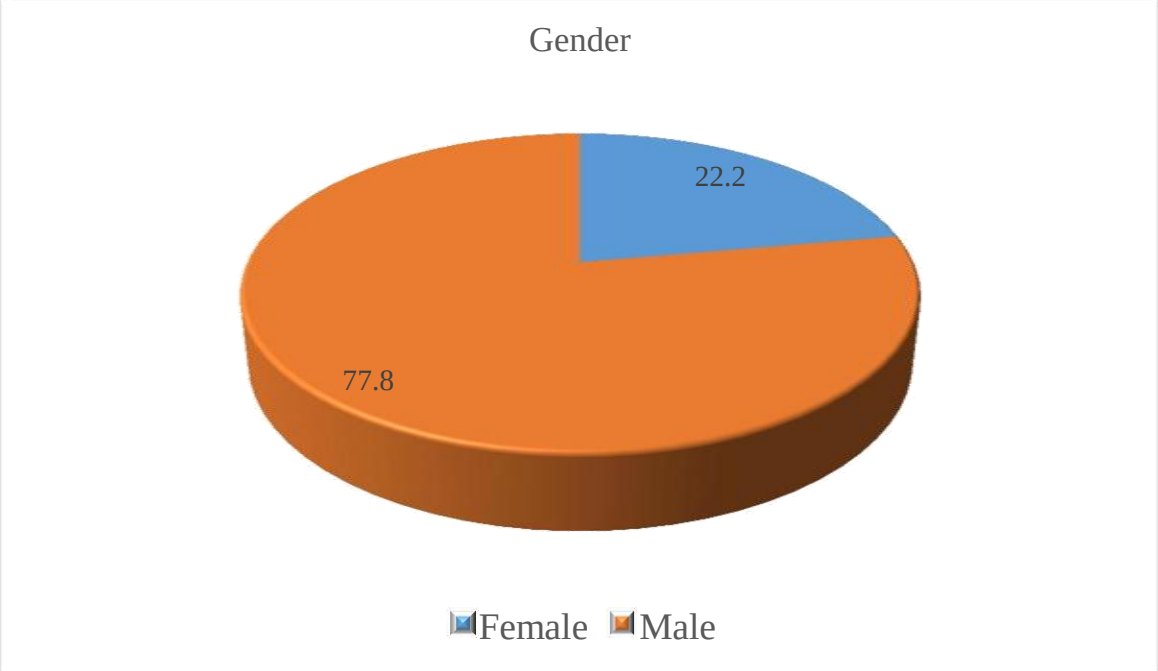


Graph 1: Bar diagram showing Age distribution of subjects

Table 2: Gender distribution of subjects

		Frequency	Percent
Gender	Female	16	22.2
	Male	56	77.8
	Total	72	100.0

Majority of subjects were Males (77.8%) and 22.2% were females.

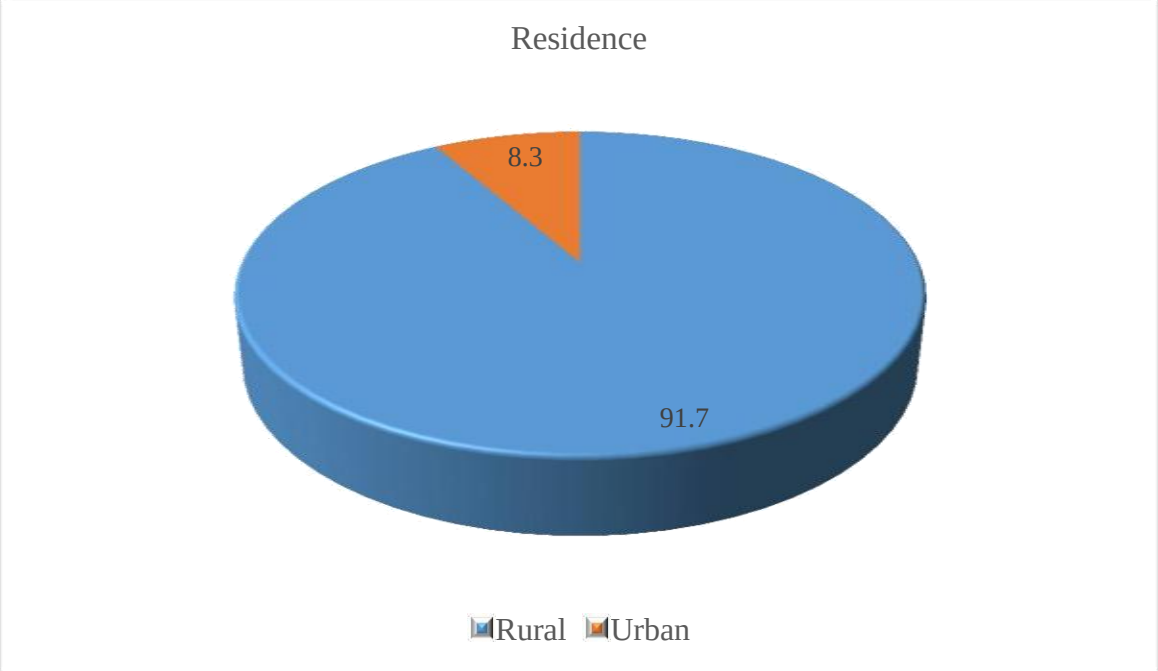


Graph 2: Pie diagram showing Gender distribution of subjects

Table 3: Distribution of subjects according to Residence

		Frequency	Percent
Residence	Rural	66	91.7
	Urban	6	8.3
	Total	72	100.0

In the study majority of subjects were from rural residence (91.9%) and 8.3% were from urban area.

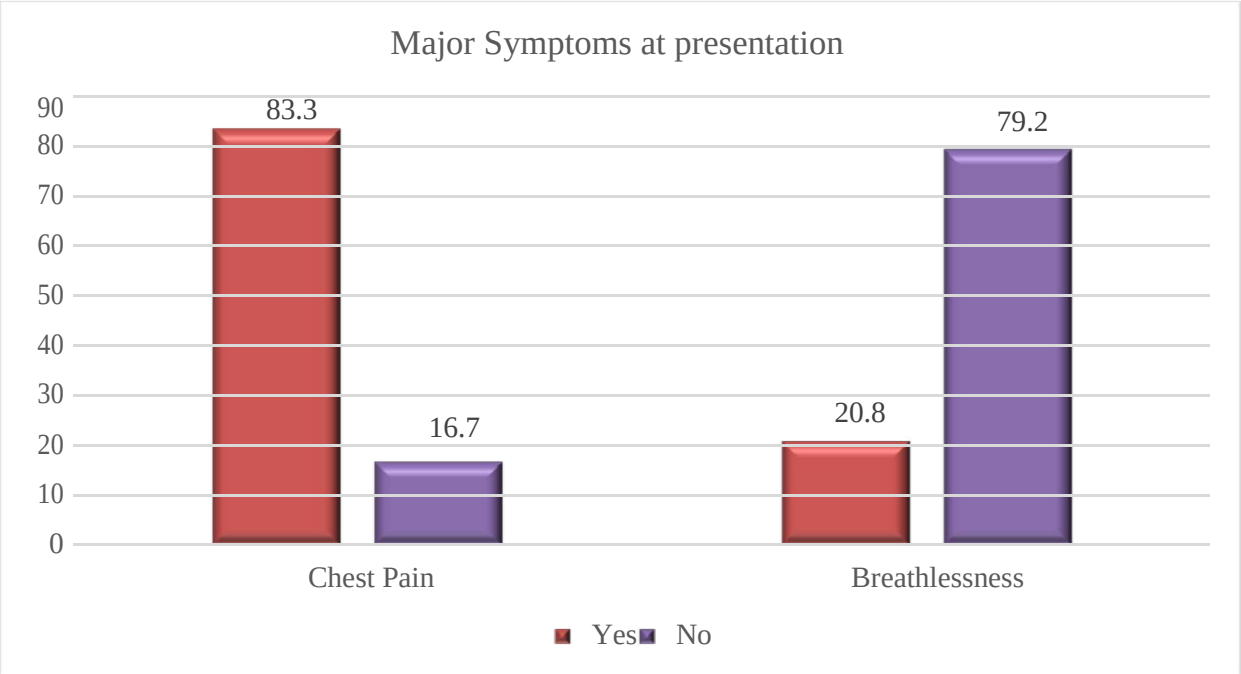


Graph 3: Pie diagram showing distribution of subjects according to Residence

Type 4: Distribution of subjects according to Major Symptoms at presentation

	Yes		No	
	Frequency	Percent	Frequency	Percent
Chest Pain	60	83.3	12	16.7
Breathlessness	15	20.8	57	79.2

83.3% of subjects presented with chest pain and 20.8% presented with breathlessness.

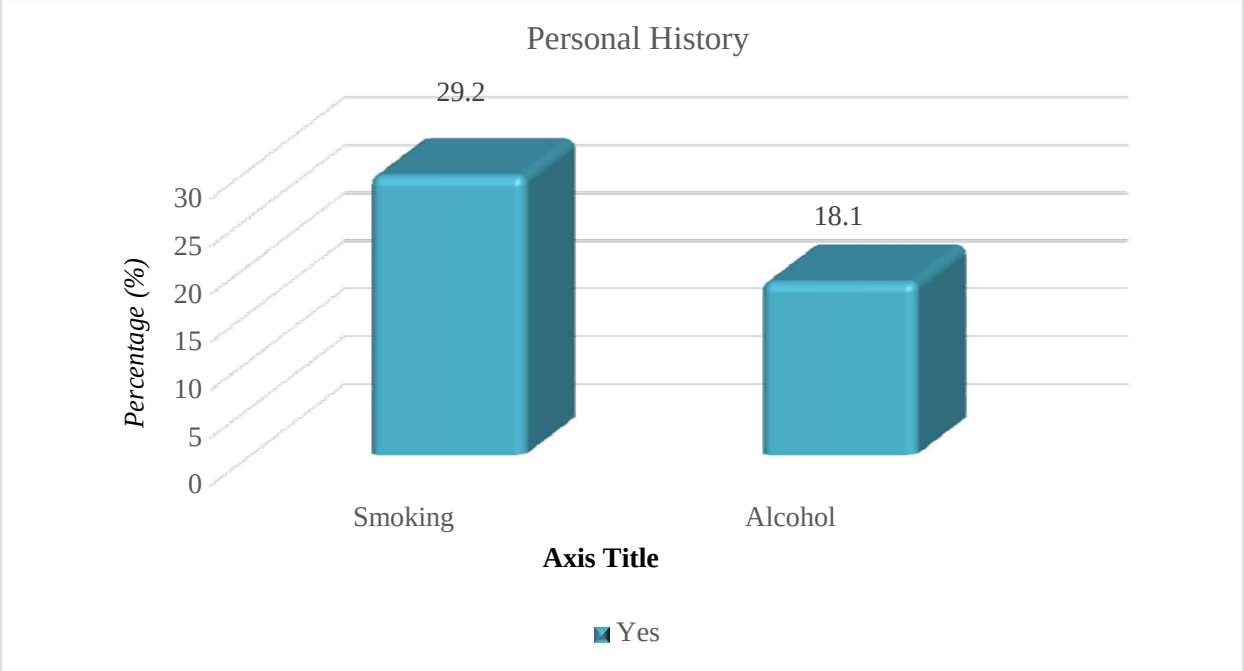


Graph 4: Bar diagram showing Symptoms at Presentation

Table 5: Distribution of subjects according to Personal History

	Yes		No	
	Frequency	Percent	Frequency	Percent
Smoking	21	29.2	51	70.8
Alcohol	13	18.1	59	81.9

29.2% of subjects were smokers and 18.1% were alcoholics.

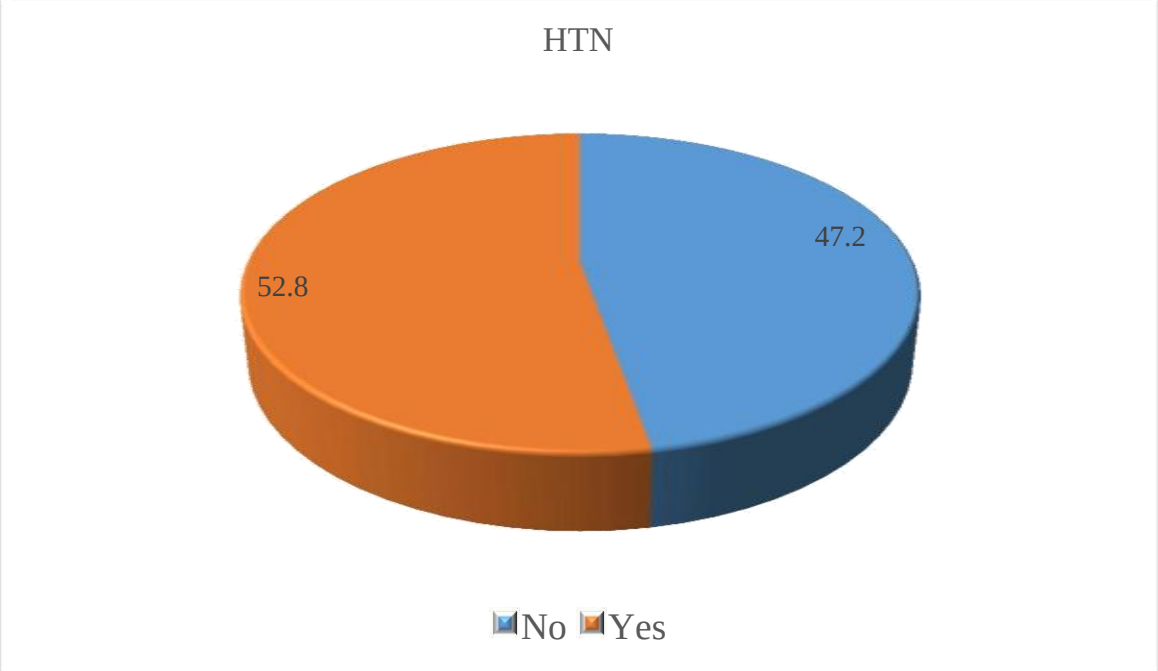


Graph 5: Bar diagram showing Distribution of subjects according to Personal History

Table 6: Distribution of subjects according to Hypertension

		Frequency	Percent
HTN	No	34	47.2
	Yes	38	52.8
	Total	72	100.0

52.8% of subjects had history of HTN in the study.

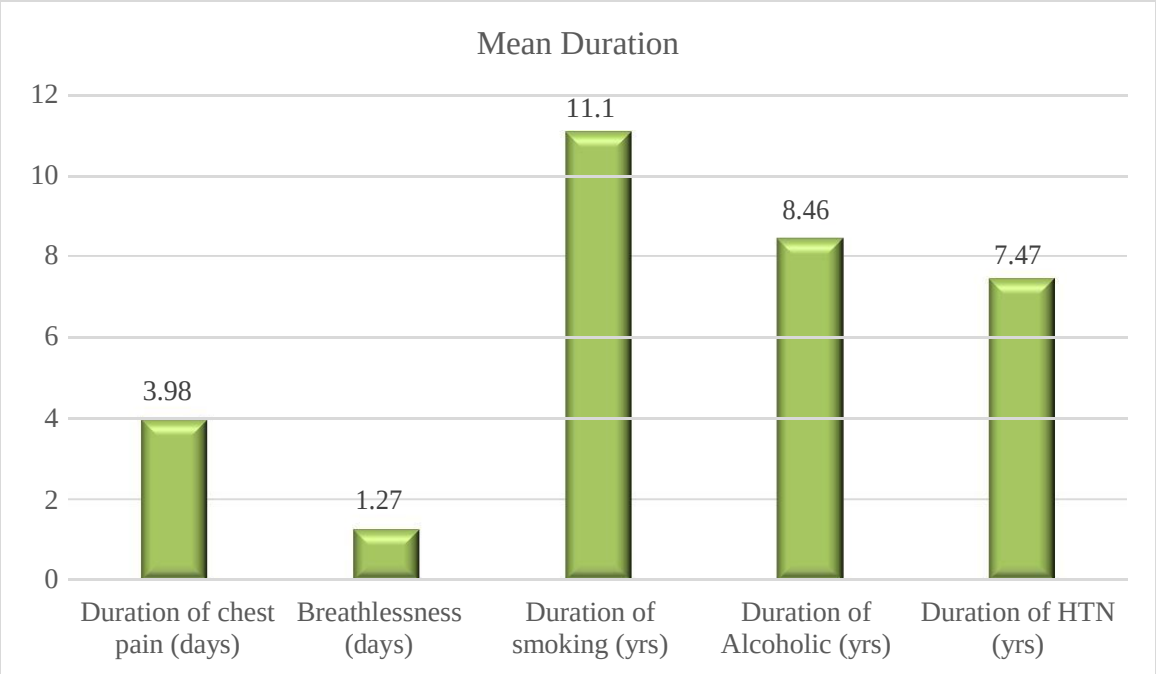


Graph 6: Pie diagram showing distribution of subjects according to HTN

Table 7: Duration of Symptoms and Smoking, Alcohol intake and HTN in subjects

	Duration of chest pain (days)	Breathlessness (days)	Duration of smoking (yrs)	Duration of Alcoholic (yrs)	Duration of HTN (yrs)
N	60	15	21	13	38
Mean	3.98	1.27	11.10	8.46	7.47
SD	13.650	.594	5.718	3.072	4.310
Minimum	1	1	5	4	1
Median	1.00	1.00	10.00	10.00	6.50
Maximum	90	3	25	15	20
Range	89	2	20	11	19

Descriptive statistics of durations are given in above table.

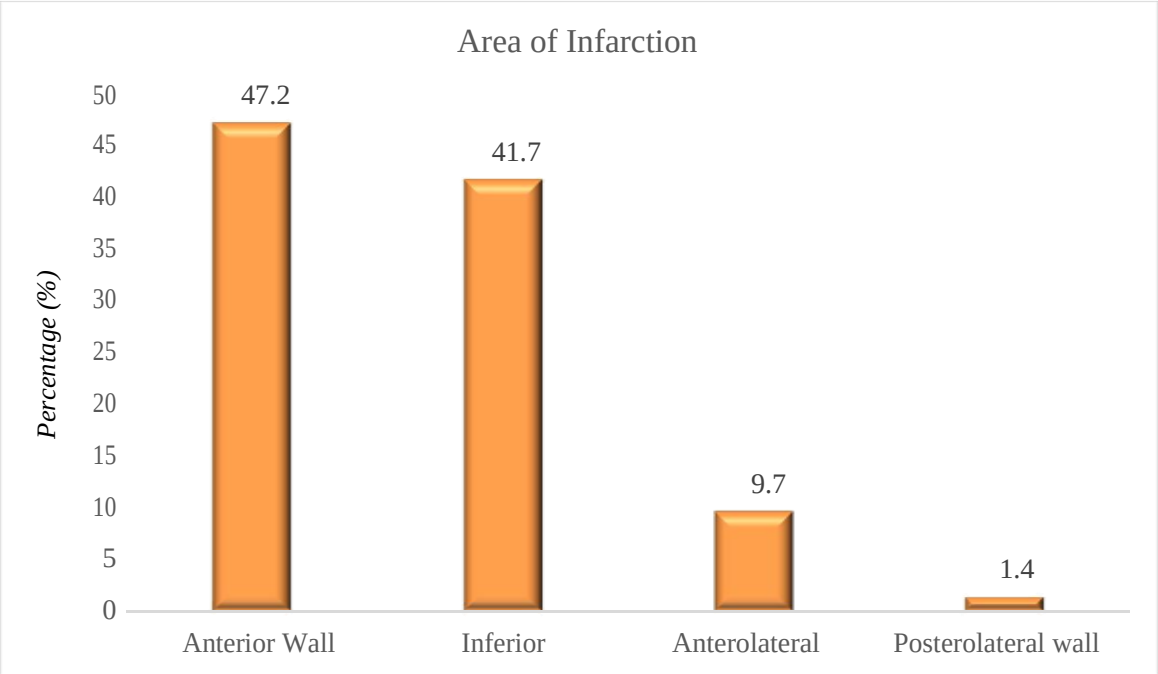


Graph 7: Bar diagram showing Mean duration

Table 8: Distribution of subjects according to Area of Infarction

		Frequency	**Percent**
Area of Infarction	Anterior Wall	34	47.2
	Inferior	30	41.7
	Anterolateral	7	9.7
	Posterolateral wall	1	1.4
	Total	72	100.0

Majority of subjects had anterior MI (47.2%), followed by inferior MI (41.7%), anterolateral MI (9.7%) and posterolateral MI (1.4%).

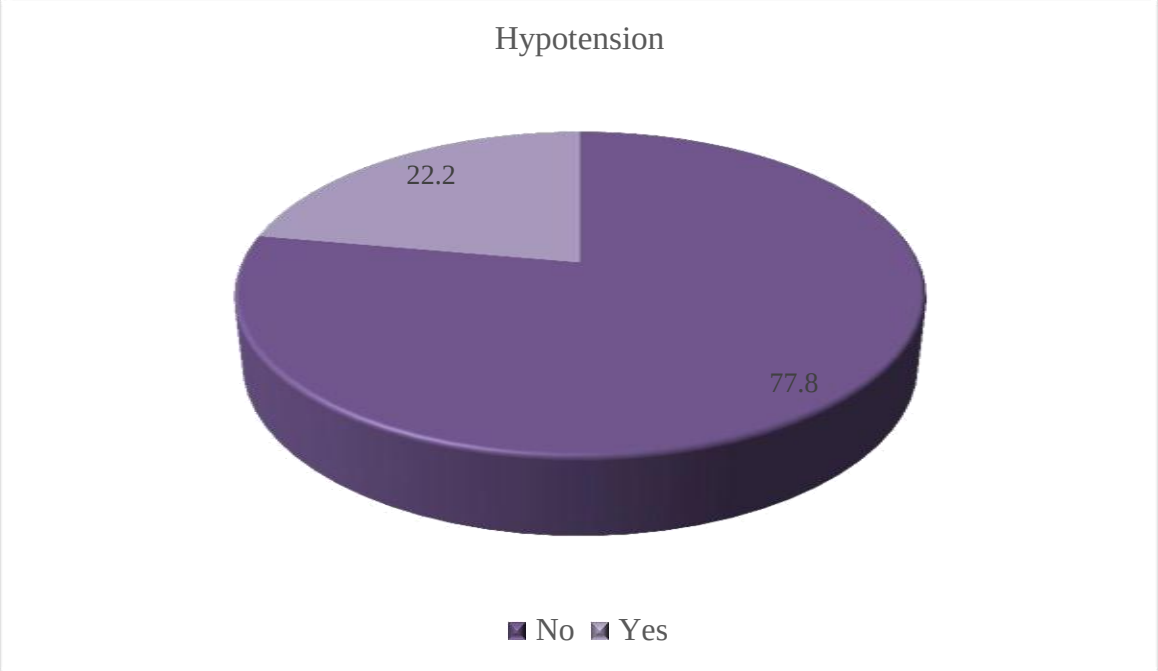


Graph 8: Bar diagram showing Area of Infarction

Table 9: Distribution of subjects according to Hypotension

		Frequency	Percent
Hypotension	No	56	77.8
	Yes	16	22.2
	Total	72	100.0

In the study 22.2% of subjects had hypotension on admission.



Graph 9: Pie diagram showing distribution of subjects according to Hypotension

Table 10: Descriptive statistics of Pulse rate, SBP and DBP in subjects

	PR in bpm	SBP	DBP
N	70	67	62
Mean	75.91	114.09	75.03
Std. Deviation	23.498	27.960	14.539
Minimum	30	60	40
Median	74.00	110.00	70.00
Maximum	150	210	110
Range	120	150	70

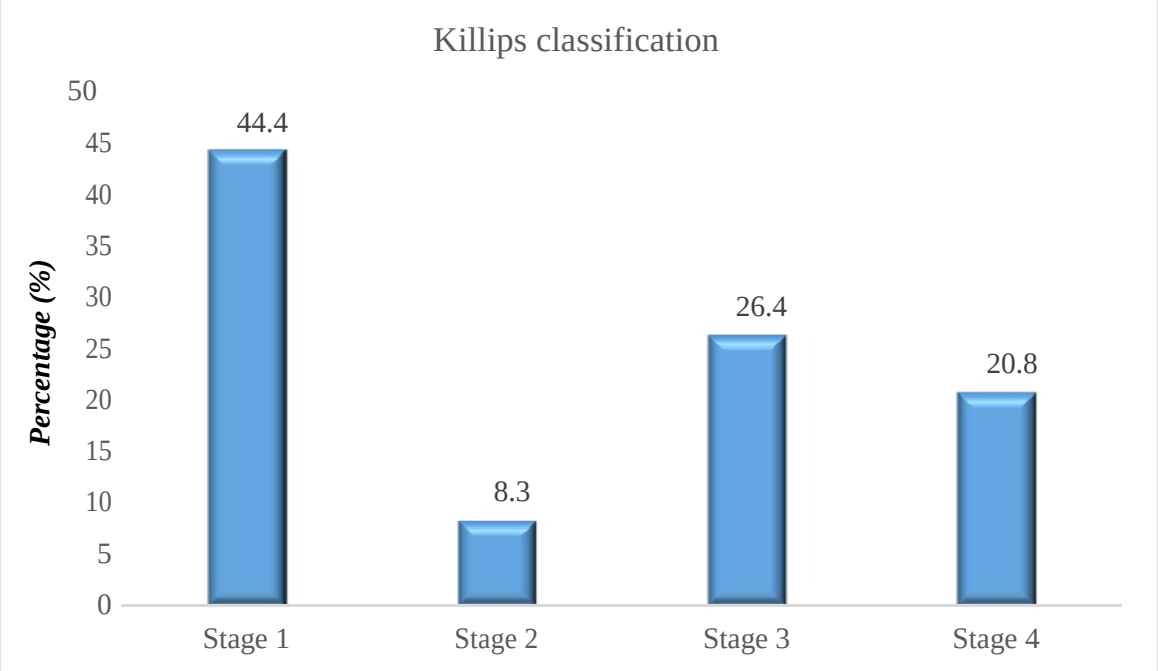
Descriptive statistics of Vital signs are given in above table.

Graph 10: Bar diagram showing Mean SBP and DBP in subjects

Table 11: Killips classification in Subjects

		Number of patients	Percent
Killips classification	Stage 1	32	44.4
	Stage 2	6	8.3
	Stage 3	19	26.4
	Stage 4	15	20.8
	Total	72	100.0

According to Killips classification 44.4% were stage 1, 8.3% were stage 2, 26.4% were stage 3 and 20.8% were Stage 4.

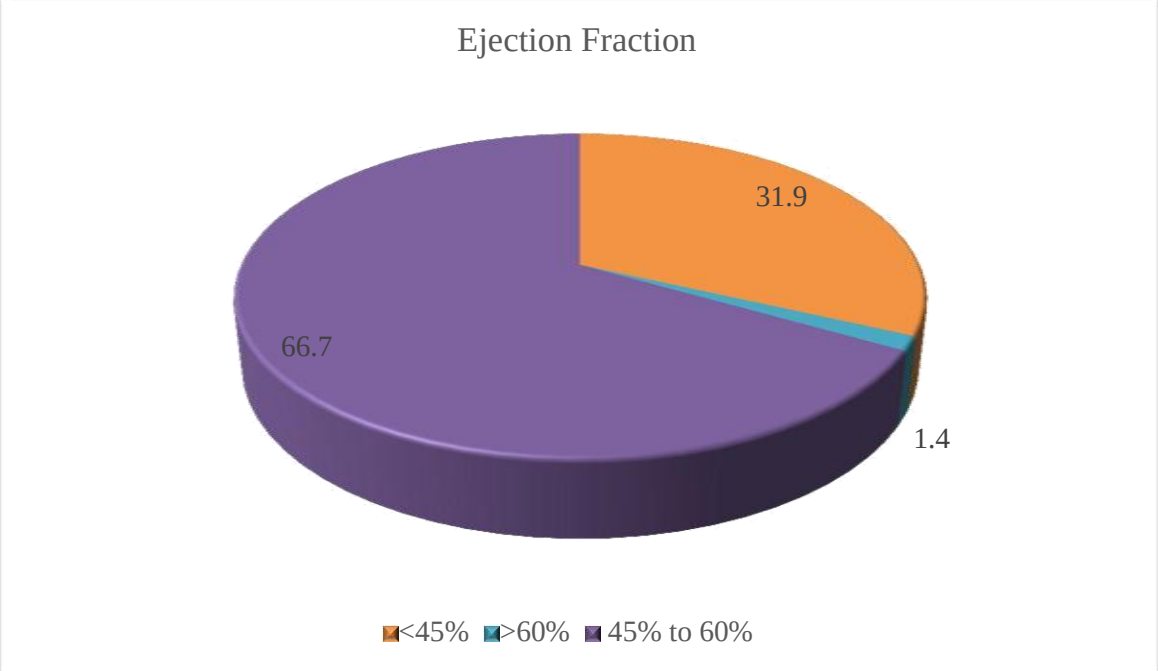


Graph 11: Bar diagram showing Killips classification

Table 12: Based On Ejection Fraction

		Frequency	Percent
Ejection Fraction	<45%	23	31.9
	>60%	1	1.4
	45% to 60%	48	66.7
	Total	72	100.0

On ECHO 31.9% had Ejection fraction < 45%, 66.7% had 45 to 60% EF and 1.4% had EF > 60%.

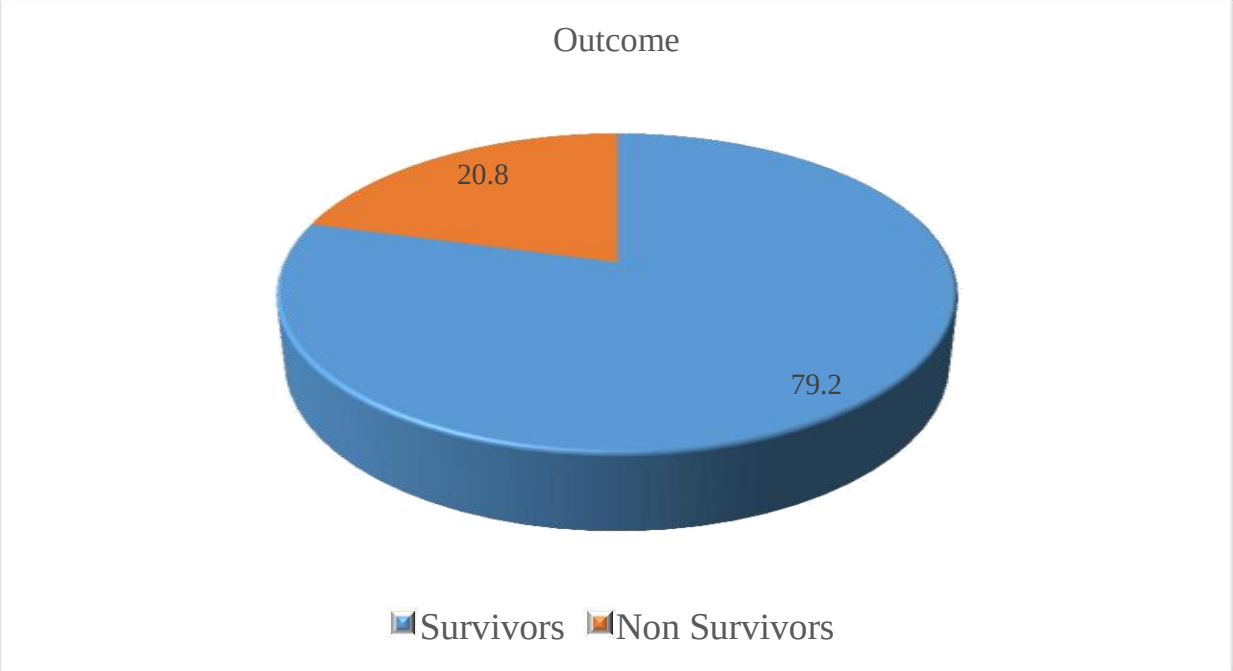


Graph 12: Pie diagram showing Ejection Fraction in subjects

Table 13: Outcome in the study

		Frequency	Percent
Outcome	Survivors	57	79.2
	Non Survivors	15	20.8
	Total	72	100.0

In the study 79.2% had good outcome and 20.8% had mortality during the course of treatment.



Graph 13: Pie diagram showing Outcome in the study

Table 14: TPI insertion in subjects

		Frequency	Percent
TPI insertion	No	61	84.7
	Yes	11	15.3
	Total	72	100.0

In 15.3% of subjects TPI was inserted.

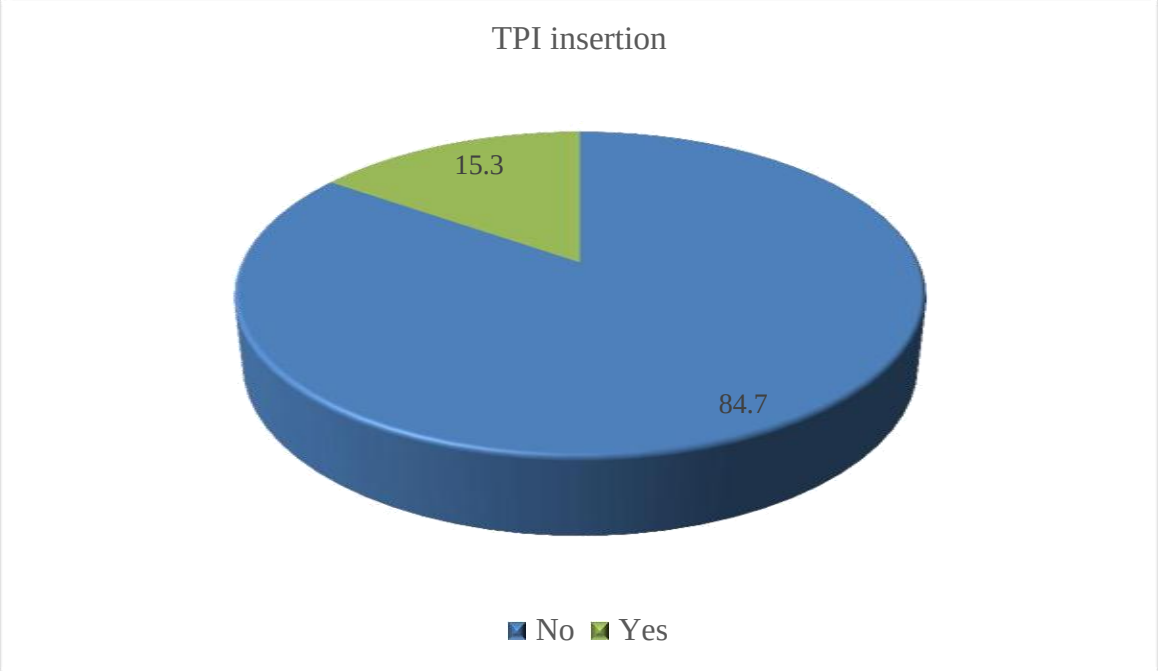
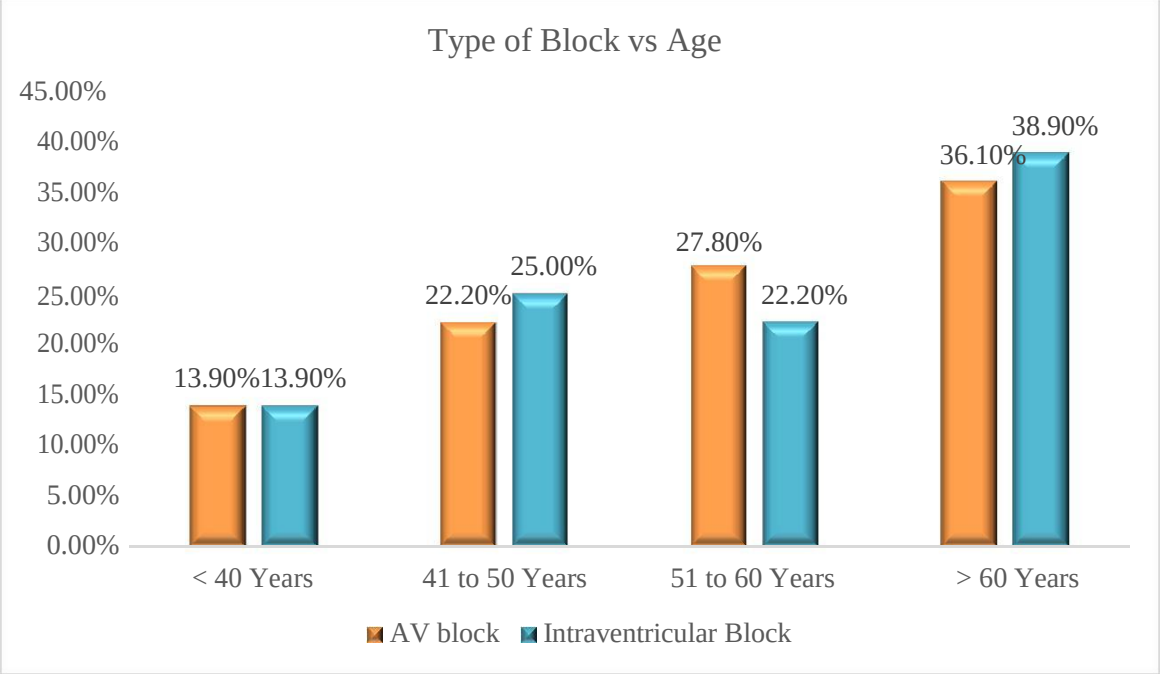


Figure 14: Pie diagram showing TPI insertion in subjects

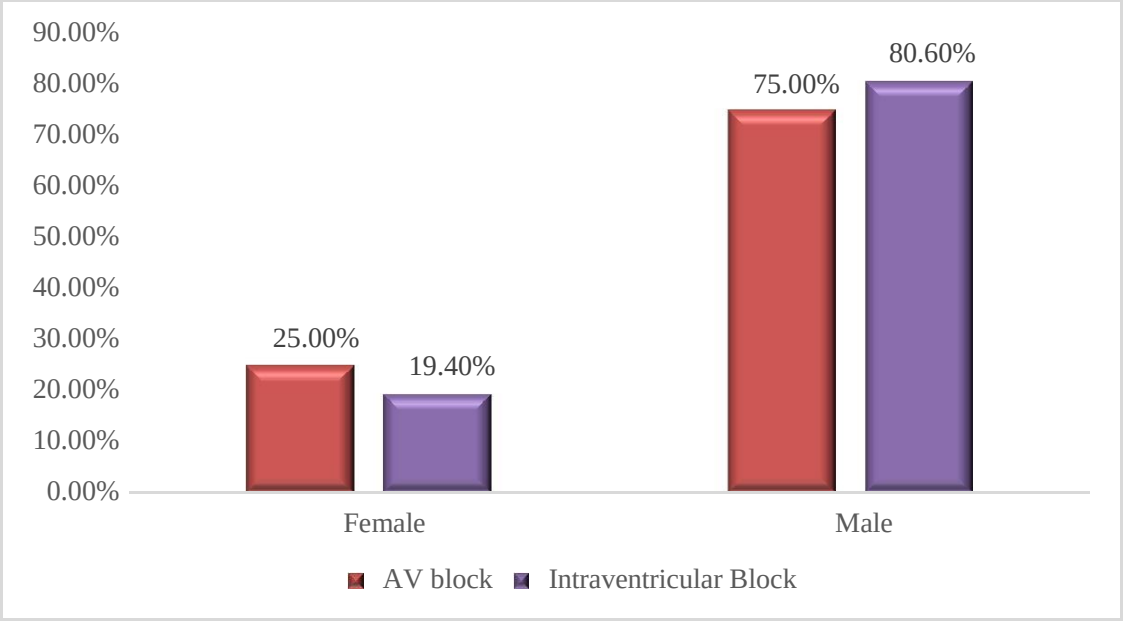
Table 15: Association between Type of Block and General Profile of subjects

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Age	< 40 Years	5	13.9%	5	13.9%	0.957
	41 to 50 Years	8	22.2%	9	25.0%	
	51 to 60 Years	10	27.8%	8	22.2%	
	> 60 Years	13	36.1%	14	38.9%	
Gender	Female	9	25.0%	7	19.4%	0.571
	Male	27	75.0%	29	80.6%	
Residence	Rural	34	94.4%	32	88.9%	0.394
	Urban	2	5.6%	4	11.1%	

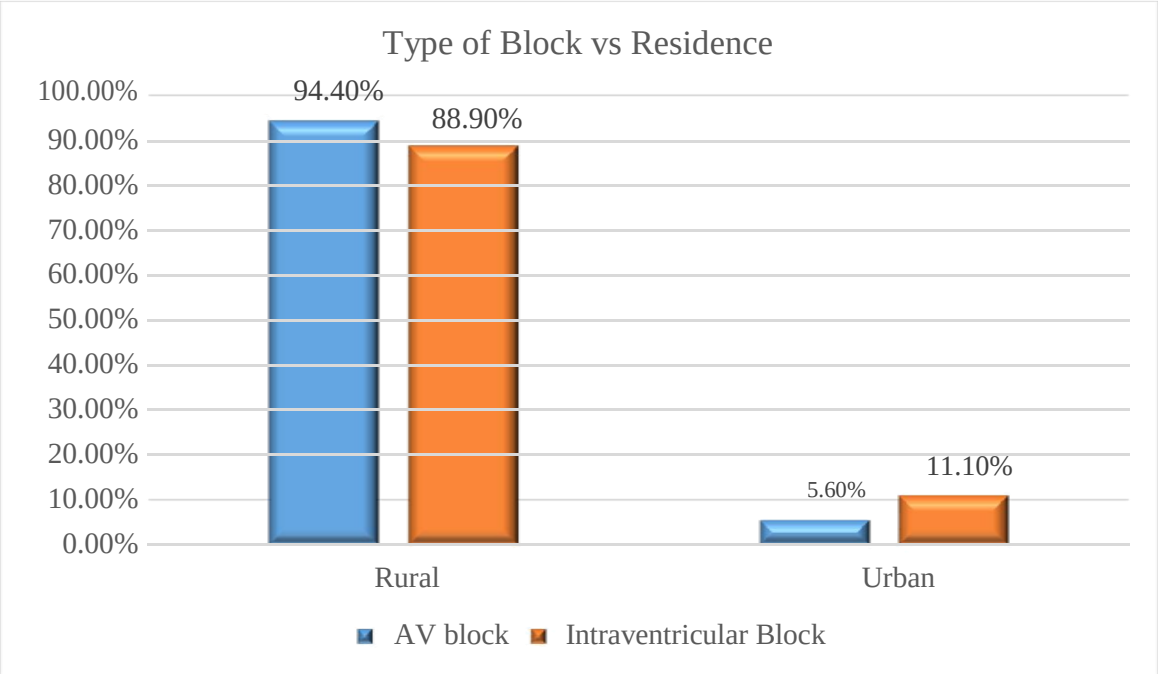
There was no significant association between Type of block and Age, Gender and Residence. Hence confounding was removed by matching the subjects in both groups.



Graph 15: Bar diagram showing association between age and Type of block



Graph 16: Bar diagram showing association between Type of Block and gender

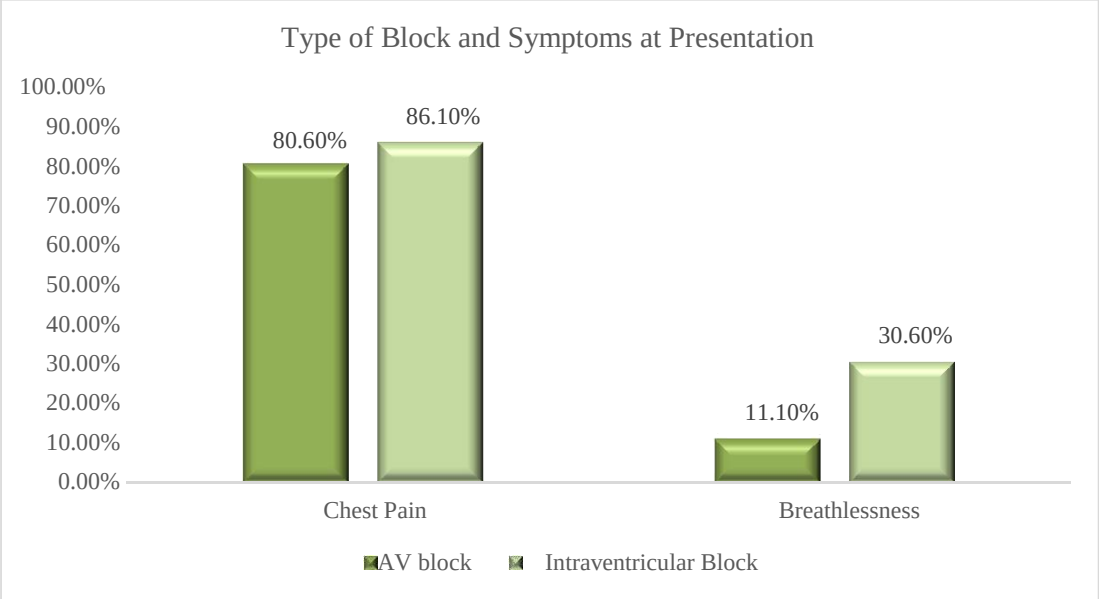


Graph 17: Bar diagram showing association between Type of Block and Residence

Table 16: Association between Type of Block and Symptoms at Presentation

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Chest Pain	No	7	19.4%	5	13.9%	0.5271
	Yes	29	80.6%	31	86.1%	
Breathlessness	No	32	88.9%	25	69.4%	0.042*
	Yes	4	11.1%	11	30.6%	

Majority of subjects in both block presented with Chest pain. There was no significant difference. Were as 30.6% of IV block presented with breathlessness and only 11.1% in AVB presented with breathlessness. This was statistically significant.



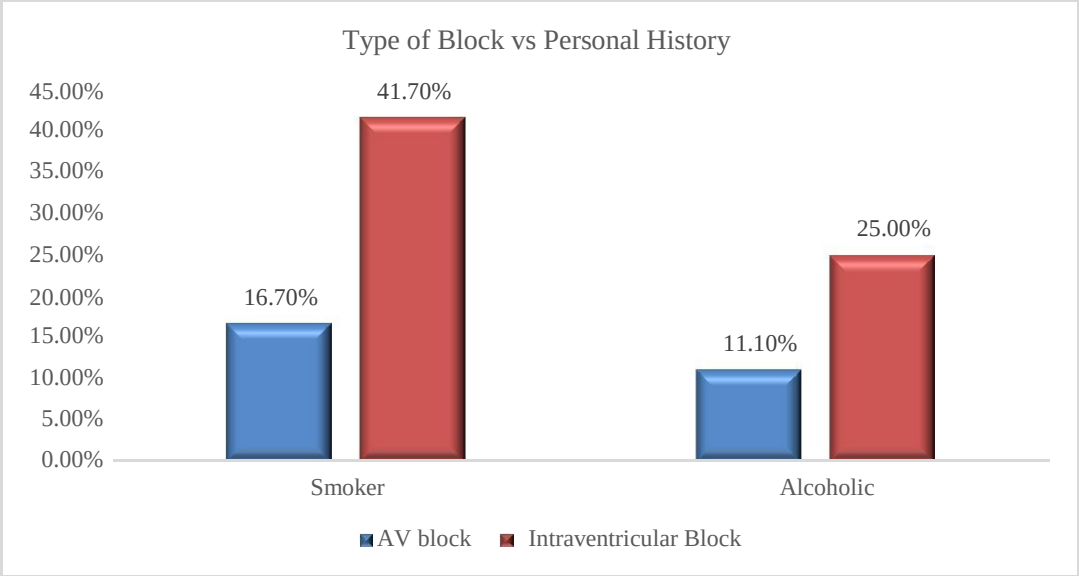
Graph 18: Bar diagram showing Association between Type of Block and Symptoms at Presentation

Table 17: Association between Type of Block and Personal History of subjects

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Smoker	No	30	83.3%	21	58.3%	0.020*
	Yes	6	16.7%	15	41.7%	
Alcoholic	No	32	88.9%	27	75.0%	0.126
	Yes	4	11.1%	9	25.0%	

41.7% of IV block subjects were smokers & 16.7% of AV block subjects were smokers. This observation was statistically significant.

There was no significant association between type of block and alcohol intake.

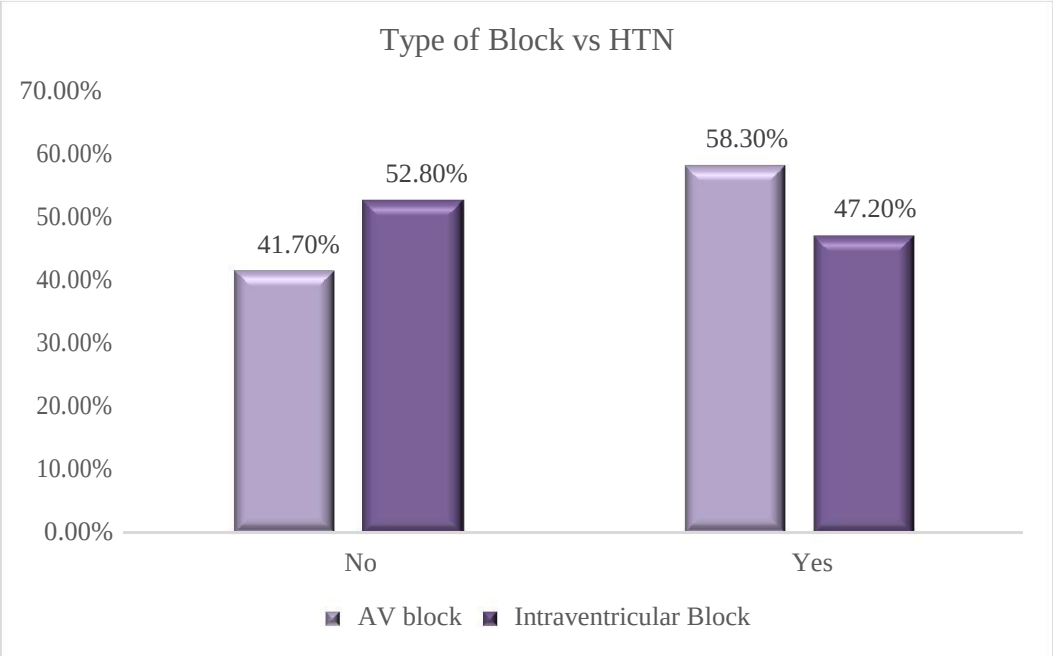


Graph 19: Bar diagram showing association between Type of Block and Personal History

Table 18: Association between Type of Block and HTN in subjects

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
HTN	No	15	41.7%	19	52.8%	0.345
	Yes	21	58.3%	17	47.2%	

There was no significant association between type of block and HTN history.

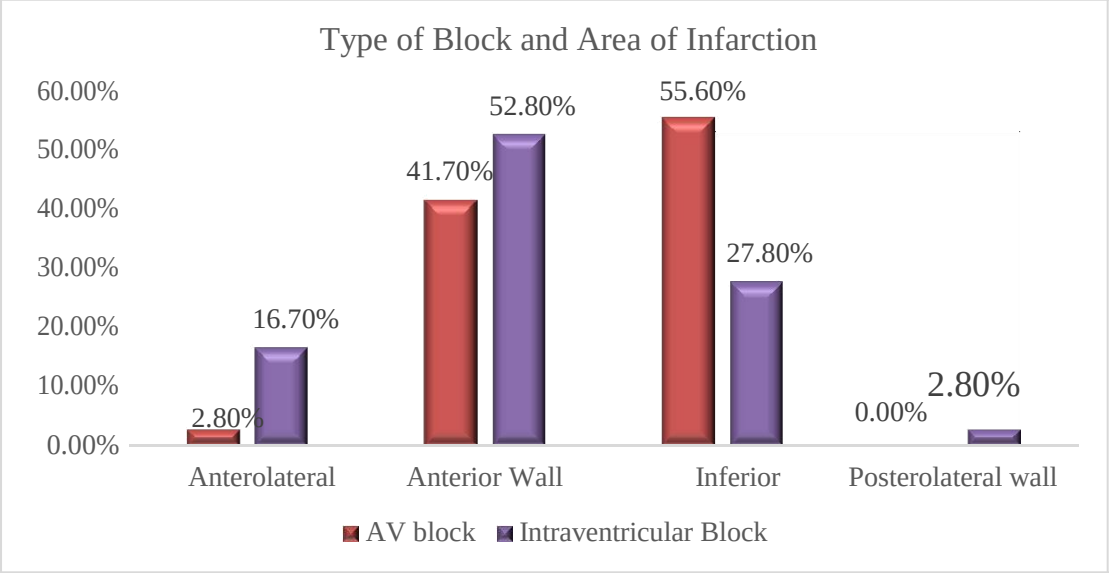


Graph 20: Bar diagram showing Association between Type of Block and HTN

Table 19: Association between Type of Block and Area of Infarction

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Area of Infarction	Anterolateral	1	2.8%	6	16.7%	0.039*
	Anterior Wall	15	41.7%	19	52.8%	
	Inferior	20	55.6%	10	27.8%	
	Posterolateral wall	0	0.0%	1	2.8%	

There was significant association between type of block and Area of Infarction. I.e. Majority of AVB subjects had Inferior wall MI (55.6%) and Majority of IVB subjects had anterior wall (52.8%) MI.

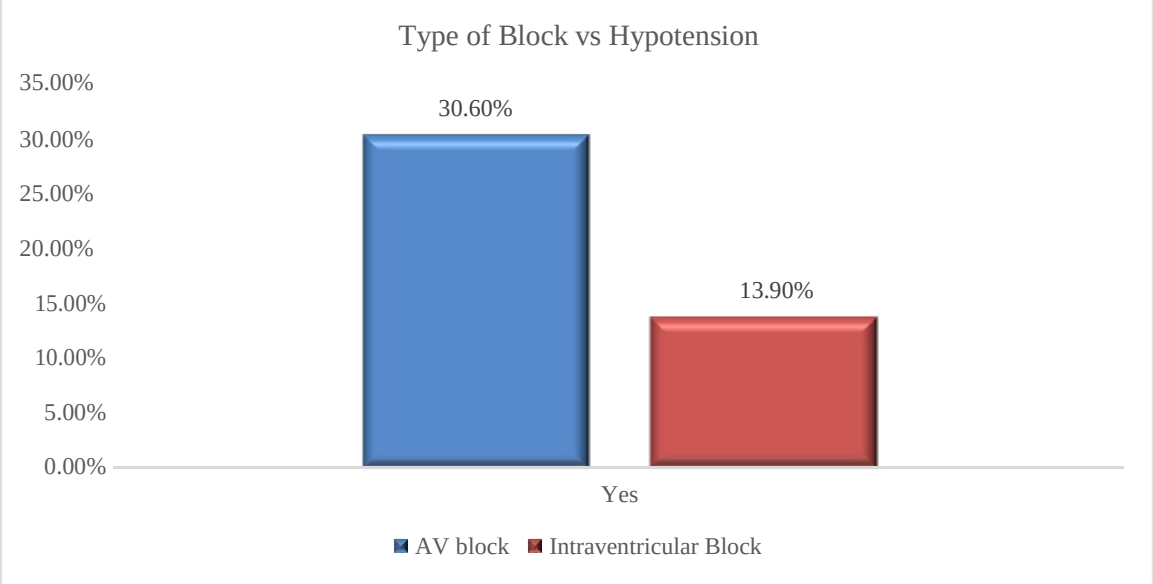


Graph 21: Bar diagram showing Association between Type of Block and Area of Infarction

Table 20: Association between Type of Block and Hypotension in subjects

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Hypotension	No	25	69.4%	31	86.1%	0.089
	Yes	11	30.6%	5	13.9%	

There was no significant difference between type of block and hypotension.

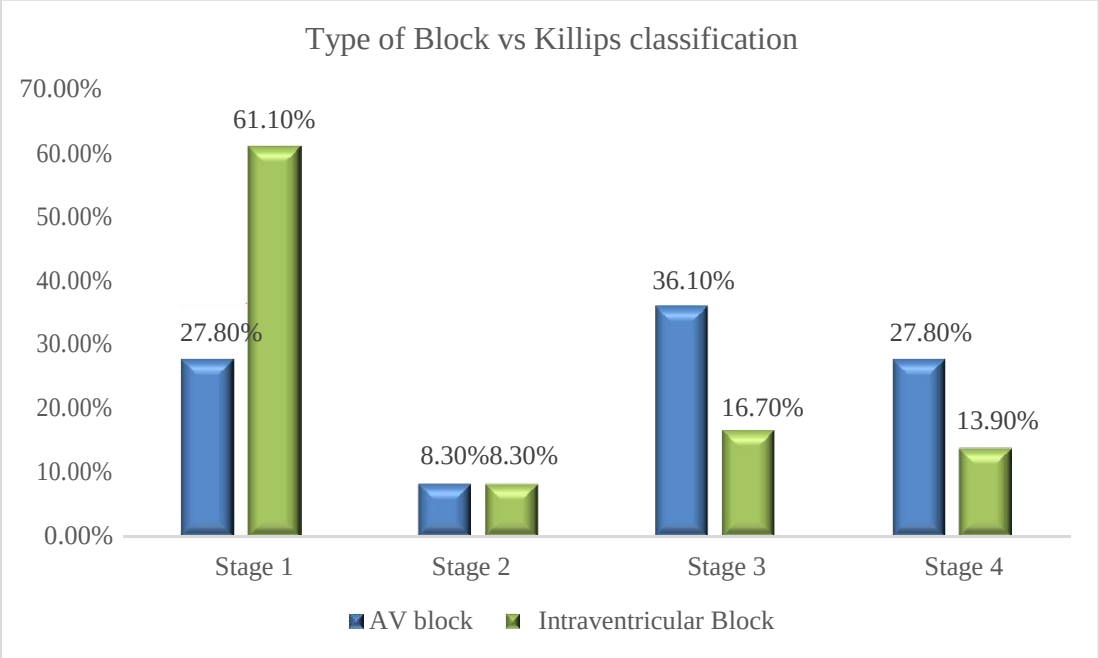


Graph 22: Bar diagram showing Association between Type of Block and Hypotension

Table 21: Association between Type of Block and Killips classification

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Killips classification	Stage 1	10	27.8%	22	61.1%	0.033
	Stage 2	3	8.3%	3	8.3%	
	Stage 3	13	36.1%	6	16.7%	
	Stage 4	10	27.8%	5	13.9%	

In the study majority of subjects in AV block had stage 3 Killips classification (36.1%) and in IV block majority of them had Stage 1 Killips classification (61.1%). This observation was statistically significant.

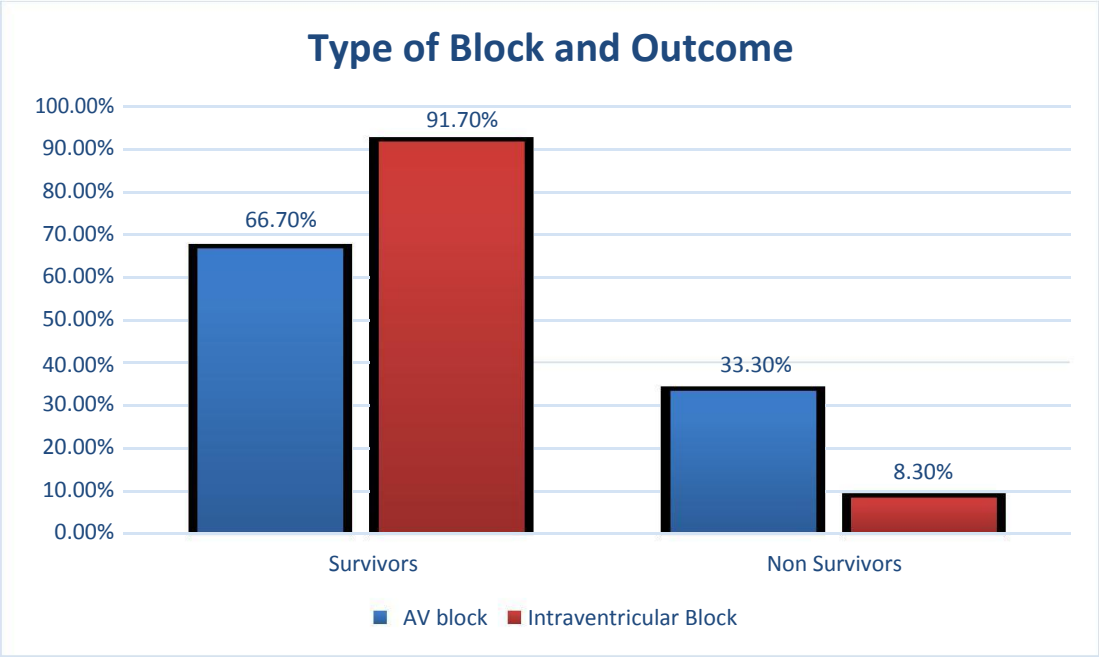


Graph 23: Bar diagram showing Association between Type of Block and Killips classification

Table 22: Association between Type of Block and Outcome

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Outcome	Survivors	24	66.7%	33	91.7%	0.009
	Non-Survivors	12	33.3%	3	8.3%	

In the study 33.3% of subjects in AV block had mortality and in IV block 8.3% had mortality. This observation was statistically significant.

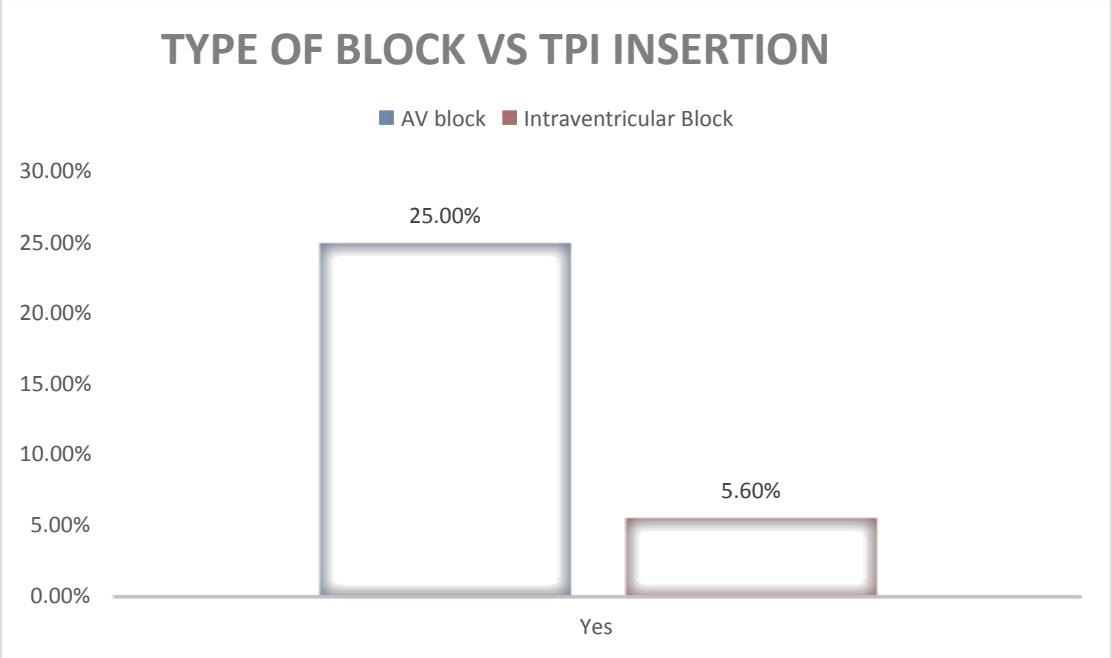


Graph 24: Bar diagram showing Association between Type of Block and Outcome

Table 23: Association between Type of Block and TPI insertion

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
TPI insertion	No	27	75.0%	34	94.4%	0.022
	Yes	9	25.0%	2	5.6%	

In the study 25% of subjects in AV block were inserted with TPI and in IV block 5.6% had TPI insertion. This observation was statistically significant.

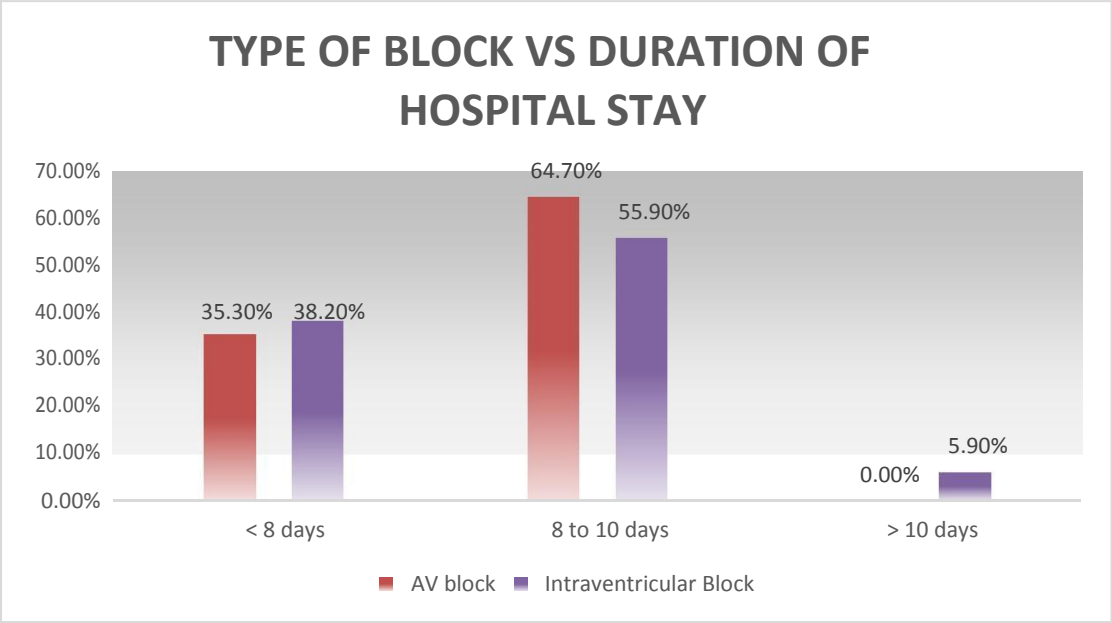


Graph 25: Bar diagram showing Association between Type of Block and TPI insertion

Table 24: Association between Type of Block and Duration of hospital stay

		Type of Block				P value
		AV block		Intraventricular Block		
		Count	%	Count	%	
Duration of hospital stay	< 8 days	12	35.3%	13	38.2%	0.323
	8 to 10 days	22	64.7%	19	55.9%	
	> 10 days	0	0.0%	2	5.9%	

There was no significant association between Duration of hospital stay and Type of block.

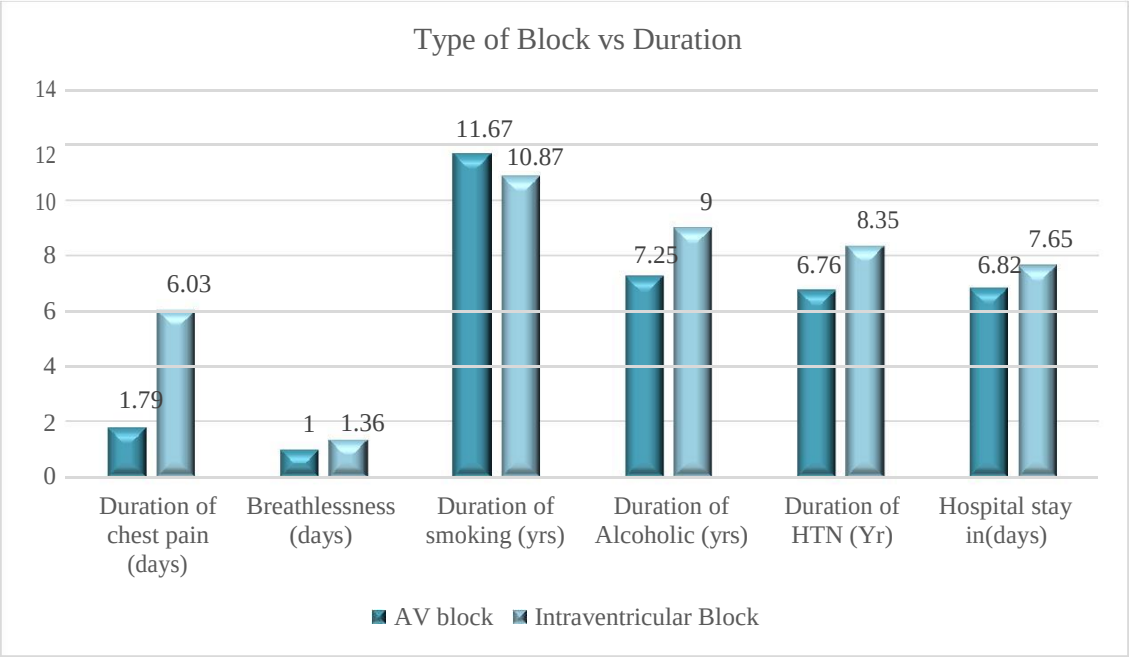


Graph 26: Bar diagram showing Association between Type of Block and Duration of hospital stay

Table 25: Association between Type of Block and Duration

	Type of Block				P value
	AV block		Intraventricular Block		
	Mean	SD	Mean	SD	
Duration of chest pain (days)	1.79	1.74	6.03	18.83	0.232
Breathlessness (days)	1.00	0.00	1.36	0.67	0.311
Duration of smoking (yrs)	11.67	7.53	10.87	5.13	0.780
Duration of Alcoholic (yrs)	7.25	5.19	9.00	1.73	0.366
Duration of HTN (Yr)	6.76	3.00	8.35	5.50	0.264
Hospital stay in(days)	6.82	2.71	7.65	2.60	0.206

There was no significant difference in duration of symptoms, smoking, alcohol intake, HTN and Hospital stay between two groups.



Graph 27: Bar diagram showing Mean duration in Type of Block

DISCUSSION

DISCUSSION

Conduction Blocks in myocardial infarction is dreaded complication. It is associated with fatal outcome, many of the deaths are due to the development of arrhythmias during MI. Myocardial ischemia is characterized by ionic and biochemical alterations, creating an unstable electrical substrate capable of initiating and sustaining arrhythmias and infarction creates areas of electrical inactivity and blocks conduction, which also promotes arrhythmogenesis [42].

It has been found that many serious arrhythmias develop before hospitalization, even before the patient is brought to hospital. At least 75% of patients with AMI have arrhythmia in the infarct period, and also that majority of deaths occur secondary to development of arrhythmias [42]. The etiology of AVB in the setting of STEMI is thought to be multifactorial and dependent on the location of the culprit lesion [43-46].

The AV nodal artery normally arises from the right coronary artery [47] and the ischemic insult caused by STEMI is thought to be sufficient to cause a transient dysfunction of the conduction fibers. The conduction tissue of the AV node is usually resistant to permanent damage from ischemia due to the high intracellular contents of glycogen, the rich complex arterial blood supply, and the capability of nutrient and oxygen absorption by diffusion from surrounding venous sinusoids.

In addition, AVB is thought to be provoked by enhanced parasympathetic tone or local release of potassium or adenosine.[48]These mechanistic considerations contribute to our understanding of the transiency of the majority of AVB events, as demonstrated in our study. In the thrombolytic era, it was shown that thrombolytic therapy may paradoxically precipitate the development of AVB[49]. It was suggested

that the reperfusion of the obstructed coronary artery induces a surge of afferent vagal activity that in turn induced a transient AVB.

Similarly, almost a quarter of the patients in our cohort developed AVB following PCI, thus it appears that reperfusion by coronary stenting may occasionally have a similar effect. In the present study there was a lower incidence of high degree AVB compared with reports from the thrombolytic era. Despite the low incidence and the fact that all cases in our cohort resolved prior to discharge, high degree AVB was associated with a significantly increased risk of both short and long term mortality, independently of other clinically important confounders. High degree AVB has consistently been found to mark an adverse short-term mortality, whereas the long-term impact remained questionable [49,50,51] . Gang et al. [52] investigated the incidence and outcomes associated with high degree AVB in a large cohort of STEMI patients treated with PPCI [52]. In that cohort the incidence of AVB was 3%, similar to our results; however, 20% of AVB were related to left anterior descending artery lesions, while no such lesions were found in our cohort. In addition, there were missing data regarding LVEF and peak CK in the patient groups. Moreover, while in the report by Gang et al. short- and long-term mortality up to 5 years of follow-up among patients with high degree AVB.[52]

In the present study we assess the prognosis of atrioventricular blocks versus intraventricular blocks in acute MI. This is based on 6 variables and comparing them for a duration of 7 days. Conduction blocks chiefly seen in age group > 60 years (37.1%). Conduction disturbances in MI our study show male (77%) predominance. It might be due smoking history more in males or male sex is a risk factor for myocardial infarction. Majority of the patients included in the study are from a rural area (91.1%). among all the patients included in the study majority of them presented

with chief complaint of chest pain (83.3%), followed by breathlessness (20.8%). Few cases presented with loose stools and back pain. Smoking history is present in 29.2% of patients. Anterior wall is most commonly involved in myocardial infarction with conduction disturbances.

Hypotension is present in 16 patients (22.2%), whereas the rest are normotensive. Majority of IVB presented in Killips stage 1 (44.4%) followed by Killips stage 3 (26.4%) to the hospital. Ejection fraction in majority of patients (66.7%) included in the study is between 45-60%.About 20% of patients died in spite of the above treatment. Pacemaker was placed in 15% of patients.

ASSESS AVB VERSUS IVB IN DIFFERENT AGE GROUPS:-

AV blocks are more (36%) in age group more than 60 years compared to other age groups. In intraventricular blocks are more (38%) in age group more than 60 years compared to other age groups. But as the ―p‖ value is insignificant we cannot establish a correlation between age group and different types of block.

Ahmadalli Shirafkhan, Mita Mehrad Study of conduction disturbances in acute myocardial infarction: clinical study and brief review of literature. The incidence of AVB and IVB in elderly (>60 years) is 33% and 64% respectively.

TABLE 26: Comparing Age Groups in the Present Study with Ahmadalli Shirafkhan,Mita Mehrad Study

	Ahmadalli Shirafkhan, Mita Mehrad Study	**Present Study**
AV BLOCK(>60 years)	33%	**36%**
INTRAVENTRICULAR BLOCK(>60 years)	64%	**38%**

COMPARE AVB VERSUS IVB IN DIFFERENT GENDER:-

About 27 (75%) members of the 36 AV blocks are present in males. About 29 (80.1%) members of the 36 Intraventricular blocks seen in males. But as the ―p‖ value is insignificant we cannot establish a significant correlation between gender and the type of block. . In a previous study atrioventricular blocks and bundle branch blocks in acute myocardial infarction males have an incidence of 62.5%, and 65.5% respectively. Females have incidence of 65.71% and 34.29% respectively.

TABLE 27: Comparing IVB gender differences in the Present Study with Macarie C,Năstase-Melicovici D study.

	Macarie C, Năstase-Melicovici D study.	**Present study**
TYPE OF BLOCK	IV BLOCK	**IV BLOCK**
Male	62.57%	**80.1%%**
Female	37.43%	**19.9%**

TABLE 28: Comparing AVB gender differences in the Present Study with MacarieC, Năstase-Melicovici D study

	Macarie C, Năstase-Melicovici D study.	PRESENT STUDY
TYPE OF BLOCK	AV BLOCK	AV BLOCK
Male	65.71%	75%
Female	34.29%	25%

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK IN DIFFERENT RESIDENCES:-

About 95% of the AV blocks seen in rural population. About 88.7% of INTRAVENTRICULAR (IV) blocks seen in rural population. But as the ―P‖ value is insignificant we cannot establish the correlation between residence and the type of block.

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK IN DIFFERENT PRESENTATIONS:-

Majority of them presented with chest pain (83%) as the main symptom, second most common complaint is breathlessness. As 30.6% of IV block presented with breathlessness and only 11.1% in AV block presented with breathlessness. This observation was statistically significant.

Breathlessness is specific symptom for those who have MI with intraventricular block than AV block.

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK IN PERSONAL HABITS:

Two things considered are the smoking and alcohol intake among those smoking is more commonly associated with IV blocks. 41.7% of IV block subjects were smokers & 16.7% of AV block subjects were smokers. This observation was statistically significant. There was no significant association between type of block and alcohol intake. Alcohol intake and condition blocks have no significant relation. In another study 30% increase in risk of IV blocks noticed in them patients. In other study done by Macarie C, Năstase-Melicovici D study atrioventricular blocks and bundle branch

blocks in acute myocardial infarction.[53] Smoking patients 24% risk of IV blocks compared to AV blocks that is 20%.

TABLE 29: Comparing AVB and IVB personal habits in the Present Study withMacarie C, Năstase-Melicovici D study

	Macarie C, Năstase-Melicovici D study	**PRESENT STUDY**
	Smoking	**Smoking**
AV BLOCK	20%	**16.7%**
INTRAVENTRICULAR BLOCK	24.4%	**41.7%**

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK IN NORMOTENSIVE AND HYPERTENSIVE PATIENTS:

Percentage of people with AV BLOCK (58%) versus INTRAVENTRICULAR BLOCK (47%).But the ―P‖ value is insignificant. Hence there was no significant association between type of block and HTN history. In other study done by Macarie C, Năstase-Melicovici D study atrioventricular blocks and bundle branch blocks in acute myocardial infarction. Atrioventricular blocks and bundle branch blocks in acute myocardial Infarction [53]. Hypertension patients AV blocks incidence is 56.6% and for IV blocks is 60.6%.

TABLE 30: Comparing AVB and IVB in relation to hypertension in the PresentStudy with Macarie C, Năstase-Melicovici D study

	Macarie C, Năstase-Melicovici D study	**Present study**
AV BLOCK	56.2%	**58%**
IV BLOCK	60.6%	**47%**

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK IN VIEW OF AREA OF INFARCTION:

There was significant association between type of block and Area of Infarction. I.e. Majority of AV block subjects had Inferior wall MI (55.6%) and anterior wall (41.7%) and Majority of IV block subjects had anterior wall (52.8%) MI

TABLE 31: Comparing AVB in relation to area of infarction in the Present Studywith Macarie C, Năstase-Melicovici D study

AV BLOCK	**Macarie C, Năstase-Melicovici D,study**	**PRESENT STUDY**
INFERIOR WALL	80%	**55.6%**
ANTERIOR WALL	14%	**41.7%**

TABLE 32: Comparing IVB in relation to area of infarction in the Present Study withMacarie C, Năstase-Melicovici D study

IV BLOCK	**Macarie C, Năstase-Melicovici D,study**	**PRESENT STUDY**
ANTERIOR WALL	42%	**52%**
INFERIOR WALL	49%	**27%**

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK IN HYPOTENSIVE AND NORMOTENSIVE PATIENTS:

The Percentage of people with hypotension with conduction block in the present study .There was no significant difference between type of block and hypotension in this study. But in the study done by Elena B. Sgarbossa, MD, Sergio,

Pinski,: Acute myocardial infarction and complete bundle branch block a hospital admission: Clinical Characteristics and Outcome in the Thrombolytic Era‖[55] the incidence of hypotension in AV blocks is 38.8% and intraventricular block is 92%.

TABLE 33: Comparing AVB and IVB in relation to hypotension in the Present Studywith Elena B. Sgarbossa, study.

	HYPOTENSION IN Elena B. Sgarbossa, study	**HYPOTENSIONIN PRESENT STUDY**
AV BLOCK	38.8%	**30.6%**
INTRAVENTRICULAR BLOCK	92.%	**13.9%**

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK BASED ON KILLIP CLASSIFICATION:

In the study majority of subjects in AV block presented in stage 3 Killips (36.1%) and in IV block majority of them presented in Stage 1 Killips classification (61.1%). This observation was statistically significant. Hence AV block is associated with poor prognosis, as most of them presented with Killips stage 3. Arrhythmias during the 1st week of acute Myocardial infarction: an observational Cross-sectional study. ELENA B. SGARBOSSA, MD, SERGIO L. PINSKI, acute myocardial infarction and complete bundle branch block a hospital .Admission: Clinical Characteristics and Outcome in the Thrombolytic Era‖[54] Study most of AV blocks and intraventricular blocks in stage 4 are 38.8% and 92% respectively IV block has poor prognosis.

TABLE 34: Comparing AVB and IVB in relation to Killips staging in the PresentStudy with Elena B. Sgarbossa, study.

	ELENA B. SGARBOSSA, MD study	**PRESENT STUDY**
	KILLIP STAGING	**KILLIP STAGING**
AV BLOCK	Stage4 (38.8%)	**STAGE4 (27.8%)**
INTRAVENTICULAR BLOCK	Stage 4 (92%)	**STAGE4 (13.9%)**

Hence a significant risk is associated with AV block in acute MI than intraventricular block in this present study.

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK BASED ON EJECTON FRACTION:

AV blocks WITH EF< 45% is 27% and Intraventricular blocks with EF < 45% is 36%. But there is no significant relation. So the ejection fraction in patients with intraventricular conduction defects is associated with EF < 45%. A previous study was done by AHMADALLI SHIRAFKAN, MITRAMEHAD STUDY shows no significant relation between type on conduction blocks and ejection fraction this support our study.

TABLE 35: Comparing AVB and IVB in relation to ejection fraction in the PresentStudy.

	Present study
AV BLOCK (EF<45%)	**27%**
INTRAVENTRICULAR BLOCK (<45%)	**36%**

COMPARE AV BLOCK VERSUS INTRAVENTRICLAR BLOCK IN VIEW OF MORTALITY:-

In the study 33.3% of subjects in AV block had mortality and in IV block 8.3% had mortality. This observation was statistically significant. Hence AV blocks are associated high mortality compared to intraventricular blocks in view of mortality. In a previous study by M. SCHEINMAN, M.D., AND B. BRENMAN, B.A. Clinical and Anatomic Implications of Intraventricular Conduction Block in Acute Myocardial Infarction study mortality of AV block and IV block are 38% and IV block is 18%. This supports our study further. [58]

TABLE 36: Comparing AVB and IVB in relation to mortality in the Present Studywith by m. Scheinman, M.D., and b. Brenman, B.study.

	By M. SCHEINMAN, M.D., AND B. BRENMAN, B.A. Study	**Present study**
AV BLOCK MORTALITY	38%	**33%**
Intraventricular blocks	18.%	**8.3%**

Hence based on the variables chosen Killips staging AV block presented in stage3. And mortality is also significant in the AV block compared to Intraventricular block. Involving anterior wall is more risk of mortality than other walls of the heart, intraventricular blocks commonly involves anterior wall, AV bocks most commonly involves inferior wall followed by anterior wall.

Hence taking all things together AV blocks are associated with greater risk or poor prognosis compared to intraventricular blocks.

SUMMARY

SUMMARY

Seventy two patients of acute myocardial infarction with conduction blocks were included in the study. Mean age of subjects in the study was 57 ± 13.12 years. Majority of subjects were in the age group > 60 years (37.5%). Male to female ratio is 3:1 approximately (77.8% are Males and 22.2% are females). Most common presenting complaint is chest pain (83.2%) and 2.7% of the patients presented with sudden collapse. In the study majority of subjects were from rural residence 91.9% and 8.3% were from urban area. Smoking (29.2%) and alcohol intake (18.1%) are the common risk factors for conduction blocks in acute MI. Hypertension was found to be a significant risk factor in 52% of patients. Anterior was ischemia (47.2%) was found to be most common area involved in acute myocardial infarction with conduction block followed by inferior wall ischemia (41.7%). Cardiogenic shock was seen in 22% of patients.

In this study we assess the prognosis of patients with AMI with AVB and AMI with IVB by following variables with 7 days follow up.

1. Area of infarction (anterior wall or inferior wall),
2. Killips staging at the time of presentation,
3. Hypotension
4. Low Ejection fraction of left ventricle,
5. TPI requirement
6. Mortality.

In this study group with patients of AMI with AV blocks, 55.6% had inferior wall ischemia, 63% of the patients presented with Killips stage 3 and above, 30.6% patients developed cardiogenic shock at the time of presentation or during the

course of hospital stay, low ejection fraction was found in 27% of patients, 25% of patients needed pacing and mortality was noted to be high in this group (33.3%).

In the other group with patients of AMI with IVB, 52% had anterior wall ischemia, 30.6% of the patients presented with Killips stage 3 and above, 13.9% patients developed cardiogenic shock at the time of presentation or during the course of hospital stay, low ejection fraction was found in 36% of patients, 5.6% of patients needed pacing and mortality was 8.3%.

Taking all variables together into consideration according to this present study atrioventricular blocks are associated with poor prognosis (63% Killips stage 3 and above , 30.6% cardiogenic shock, 25% TPI requirement, 33.3% mortality) compared to intraventricular block in patients with acute myocardial infarction.

CONCLUSION

CONCLUSION

Taking all variables together into consideration according to this present study atrioventricular blocks are associated with poor prognosis compared to intraventricular block in patients with acute myocardial infarction. Conduction defects are common even in this thrombolytic era. Patients with conduction defects are at high risk of developing complications and increased mortality. They need close monitoring and optimum clinical care to reduce mortality and morbidity. Temporary external pacing and transvenous pacing definitely reduce the mortality in conduction blocks due to AMI.

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ANNEXURES

ANNEXURES

PROFORMA

1. OP/IP No.: 2. Date: 3. Serial No.:

4. Name: 5. Age: 6. Gender:

7. Occupation:

8. Date of Admission: 9. Date of Discharge:

10. Socioeconomic status:

11. Address with Phone no.:

12. Chief Complaints:

13. Past history:

14. Family history:

15. Personal History:

16. General Physical Examination: (At admission)

PR: BP: Temp:

Respiratory Rate: Spo2:

Pallor: Icterus: Cyanosis: Clubbing: Lymphdenopathy: Oedema:

17. Systemic examination:

CVS:

RS:

CNS:

PA:

18. Diagnosis:

19. Duration of hospital stay:

20. Course in the hospital:

21. INVESTIGATIONS:

I. Hemoglobin:

II. Random Blood sugar: III.

ECG for 7 days follow up IV.

Troponin T:

V. CK-MB:

VI. Other relevant investigations:

INFORMED CONSENT

Name of the investigator: **Dr. N SRI CHANDHAN REDDY**

Organization: R L JALAPPA Hospital and Research Centre attached to Sri Devaraj

Urs Medical College

Name of the participant: OP/IP no:

Study prognosis of atrioventricular block versus intraventricular conduction blocks in acute myocardial infarction

I/we the patient‘s attenders have been invited to take part in this research study. The information in this document is meant to help me to decide whether or not to take part. I have clarified my doubts regarding this study with the principal investigator.

I/we the patient‘s attenders request and authorize Dr. N Sri Chandhan Reddy to perform clinical examination on me and the designated tests for my blood sample. My signature below constitutes my acknowledgment that the benefits, risks and limitations of this testing have been explained to my satisfaction by a qualified health professional.

Participation is totally voluntary and there would be no payment for sample collection. All test results are treated with medical confidentiality and will not be disclosed to any outsider except if it is required by the law.

I/we the patient‘s attenders give my consent to allow my sample to be used for medical research, test validation or education as long as my privacy is maintained.
I/we the patient‘s attenders understand that I remain free to withdraw from this study at any time and this will not change my future care.
I/we the patients attenders have read this document and I understand the information

Subject name and signature/ Thumb impression DATE:

Parents / Guardians name / Thumb impression DATE:

Signature of the person taking consent DATE: