# CLINICAL STUDY OF CONDUCTION BLOCK IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION

**A Dissertation Submitted to**

**THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY**

**CHENNAI**

In Partial Fulfillment of the Regulations

For the Award of the Degree of

**M.D. (GENERAL MEDICINE) - BRANCH – I**


**GOVERNMENT KILPAUK MEDICAL COLLEGE**

**CHENNAI**

**APRIL – 2017**

# BONAFIDE CERTIFICATE

This is to certify that "**CLINICAL STUDY OF CONDUCTION BLOCK IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION"** is a bonafide work done by **Dr. SANGEETHA. K**, Post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in partial fulfillment of rules and regulations of the Tamil Nadu Dr. M.G.R Medical University, for the award of M.D. Degree Branch I (General Medicine) during the academic period from June 2014 to June 2017.

**Prof. Dr. S. Ushalakshmi M.D., FMMC.,**
Guide for the study,
Professor and Head of the Department,
Department of Medicine,
Govt. Kilpauk Medical College,
Chennai.

**Prof. Dr. R. Narayana Babu M.D., D.C.H.,**
The DEAN
Govt. Kilpauk Medical College
Chennai - 600 010

# DECLARATION

I solemnly declare that this dissertation "**CLINICAL STUDY OF CONDUCTION BLOCK IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Prof. Dr. S. Ushalakshmi M.D., FMMC,** Professor and Head of the Department, Department of Internal 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 fulfillment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

Place: Chennai-10 **Dr. SANGEETHA. K**

Date:

# ACKNOWLEDGEMENT

At the outset, I would like to thank my beloved Dean, Kilpauk Medical College, **Prof. Dr. R. Narayana Babu M.D., D.C.H.,** for his kind permission to conduct the study in Kilpauk Medical College.

I express my indebtedness to **Prof. Dr. S. Ushalakshmi M.D., FMMC.,** my thesis guide and Professor & HOD of Medicine for her continuous motivation, affectionate guidance, valuable suggestions, sympathetic, helping nature and encouragement enabled me to complete the dissertation.

I am extremely thankful to my unit Assistant Professors, **Dr. M. Bathragiri M.D., Dr. A. Marimuthu M.D., & Dr. T. Mohanasundaram M.D.,** for their valuable suggestions and guidance.

I would always remember with extreme sense of thankfulness for the valuable time, co-operation , criticism and support provided by my fellow post graduates , juniors , C.R.R.I's and friends.

I also extend my thanks to all the laboratory technicians for their valuable support throughout my dissertation work.

I would like to take this opportunity to show gratitude to my friends & family for their never ending support in completing this thesis.

I also extend my thanks to **Mr. Albert** for providing statistical assistance.

Finally, I wholeheartedly thank all my patients for their active co-operation in this study, without whom this would not have become a reality.

# TABLE OF CONTENTS

# INTRODUCTION

# INTRODUCTION

Conduction delay and block is the most commonly witnessed complications of acute infarction of myocardium.

It may be a delay in AV nodal conduction (or) block which may be of II,III degree AV blck depending on the site of abnormality in the conducting system.The abnormality in conduction is due to autonomic dysfunction,decreased blood flow,to the conducting tissue,or an infarction.

As various factors contribute to the conduction block in acute infarction it may be due to transient or fatal.

It is important to anticipate particular tye of conduction abnormality in specific type of lesion in the coronary arteries, as fatal abnormalities can be managed promptly.

Incidence and clinical implications cannot be determined accurately in myocardial infarction as,

i)most studies are retrospective

ii)Low incidence in studies done before reperfusion era.

iii)To identify Old and New conduction abnormalities is not possible.

iv)Transition from one type of the block to another And various factors other than the infarction of myocardium, leading to conduction block.

But still pattern of blocks in different types of myocardial infarctin(based on site of lesion) can guide in anticipating the risk and assessing the Prognosis there by planning treatment accordingly.

Knowledge in anatomy of conducting system and its blood supply is important in understanding the significance Of association between type of infarction,site and the Degree of conduction disturbance.

There are Various studies on Incidence of conduction block amongst acute MI In Pre PCI era.In PCI Era only few studies are available Which showed that the incidence of conduction abnormality is Low compared to Pre PCI era.

According to the Retrospective study among ST elevation MI patiens in Brittany in Year 2006 to 2013 "AV block is more common in The right Coronary artery occlusion when compared to Left coronary Artery occlusion"(1)".Itis often Transient due to increased parasympathetic tone.Sinus BradyCardia is the most Common arrhythmia.

" In RCA occlusion Incidence Of AV block is5.9% with 1.5% in Other arterial infarctions".

2.6% incidence of AVblock in patients not reperfused with1.2%in patiets who underwent PCI and 0.5% among patients who are not thrombolysed.(1)

In TRACE study incidence of III Degree AV block in acute infarction of myocardium is 4-5%.(2-6),

II and III Degree AV block incidence is 7-10%.(1).and is high in initial 48 hours. "Transition to II or III degree block leads to two fold increased risk of mortality than with out transition"(7).

With respect to bundle Branch Block ,RBBB is more than LBBB According to NREM-2 Study.

In HERO-2,the new BBB is not common in acute MI,but carries poor prognosis.Mortality in anterior wall MI with AV Conduction abnormality is more than its association with Inferior MI.

NEED FOR THE STUDY:

It is mandotary to know the pattern of conduction Abnormality and its relation to the Site of infarction,because of prognostic implications and prompt intervention through various modes: (i) Phrmacological

(ii)Pacing Either temporary or permanent.

## AIMS & OBJECTIVES

1. To study the incidence of conduction block in acute myocardial infarction.

2.To study various patterns of conduction blocks occurring in acute myocardial infarction.

3.To study the prognostic implications of conduction blocks occurring in acute myocardial infarction.

# REVIEW OF LITERATURE

## REVIEW OF LITERATURE

ANATOMY OF CONDUCTING SYSTEM OF HEART:-

Conducting system of heart is made of specialized myocardial cells and conducting fibres,capable of initiating And conducting Electrical impulses. “The functioning of conducting system should be regular and rhythmic for effective Synchronisation of cardiac Events,so that heart can effectively receive and pump out blood”(8,9).

Conducting System is comprised of:-

Sinoatrial node

Intra atrial and Internodal Pathways

(i)Anterior(,Bachman ),Middle ,Posterior

AV Node

Bundle of His

Bundle Branches

Purkinjie fibres

SINO ATRIAL NODE:

It consist of spindle shaped cells.It is 20 mm long 2-3 mm breadth,located just 1 mm beneath the epicardial surface At the junction of superior vena cava with the Right atrium.

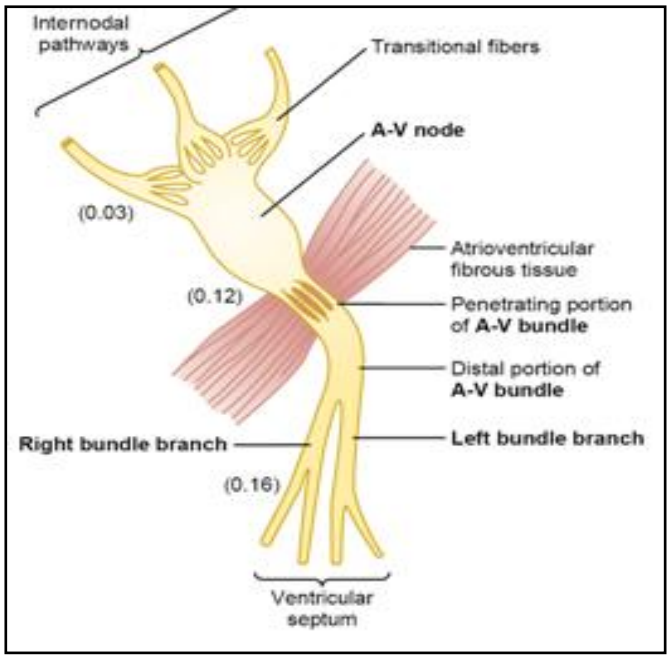
BLOOD SUPPLY: RCA in 55-60%

LCA in 40-45%

INNERVATION:- Densely innervated 3 times more than Atrial tissue by sympathetic and parasympathetic Nerves through B1 and B2 ,muscuranic receptors.(10)

INTERNODAL PATHWAYS:-

Anterior intermodal path is the one which start in anterior margin of sinoatrial node to end in superior margin of left Atrium.Middle starts from both superior and posteriormargin via intermodal septum to end in superior margin of AV Node.Posterior tract extends from posterior margin to travel posteriorly through to interatrial septum and joins AVnode.




They continue as transitional fibres to end inAV node

ATRIOVENTRICULAR NODE:-

LOCATION: RA endocardium at the apex of Kochs triangle formed

ABOVE:Tendon of todaro

BELOW:Tricuspid valve septal leaflet

BASE:Opening of coronary sinus

BLOOD SUPPLY: RCA=85-90%

LCx=10-15%

SIGNIFICANCE:The distal part of AV Node is capable of automaticity. "AV node allows for travel of electrical impulse from the atria to the

ventricle,giving time for emptying atrial blood into ventricle and coordinating contraction."(11,12)

HIS BUNDLE:

LOCATION:Begins inAV Nodethen penetrates central fibrous Body to end in mmembranous septum.

BLOOD SUPPLY:Both Anterio and Posterior descending Coronary artery.

SIGNIFICANCE " His bundle:Ischemia is rare unless it is widespread due to dual supply."(13)

BUNDLE BRANCH:

LOCATON:Begins in upper margin of muscular Portion of the interventriclar septum dividing into i)Left Bundle Branch

ii)Right Bundle Branch

Left pass through interventricular septum to divide into anterior and posterior fascicle.

Right bundle pass intramyocardially to supply Right Ventricle.

BLOOD SUPPLY: RBB=LAD

LBB=LAD

LAF=LAD

LPF=Proximally:AV Nodal artery

Distally:Dual supply from both Anterior and Posterior septal perforating arteries

According to a study conducted on 30 hearts to define principal blood suppyof SA and AV node and the result of the study also proved the same.(ref)

PURKINJIE FIBRES:

Forms network of fibres in endocardial surface of the ventricles exciting both the ventricles at the same time.

It is less dense at Base and Papillary muscle tip.Resistant to decreased blood supply when compared toother fibres.

| Coronary Artery | Cardiac Muscle | Conduction System |
|---|---|---|
| RCA | RV-lateral/posterior wall<br>LV-inferior wall | SA node (45%)*<br>AV node (90%)*<br>Bundle of His<br>Right Bundle |
| LAD | RV-anterior wall<br>LV-septum/apex/<br>anterior wall | Left Bundle |
| LCx | LV-lateral/posterior wall | Left Bundle<br>SA node (55%)*<br>AV node (10%)* |

INNERVATION OF CONDUCTING SYTEM OF HEART:

Autonomic supply o heart is comprised of Vagus (parasympathetic) and sympathetic supply through the stellate ganglion of Cervical sympathetic chain.

Adrenergic receptors: B1;B2 receptors

Cholinergic receptors:M I (muscuranic)

ON SA Node, the parasympathetic fibres ,"vagal to SA node also sends fibres to SVC ,Aortic root pad then only to ICV andRight pulmonary pad"(14).More amount of Ach is present in SA node than rest of right atrium and left atrium.

Ach concentration:SA NODE > R A>L A>VENTRICLES.

POSITIVE CHRONOTROPIC EFFECT(ref)

"Adrenaline +BI receptor

⇩

activates Adenyl Cyclase

⇩

↑ Intracellular Camp

⇩

activates protein kinase A

⇩

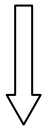
Phosphorylation of ion handling Proteins"(15)

⇩

↑ Discharge of impulses.

NEGATIVE CHRONOTROPIC EFFECT:

Acetyl choline + M 2 receptor



Activate G(inhibitory) receptor

↓ Intracellular Camp

It also modulates G protein receptor to open potassium channelthere by allowing its outward flow leading to decreased heart rate.

"The AV node is not uniformly innervated with inferior portion of AV node is densely innervated than compact node"(16).

Autonomic nervous system exhibit sidedness to some extent ,inspite of overlap.Right side is concerned more with SA node and it alsodecreases the doration of refractory time of the anterior aspect of ventricle.Left side isconcerned more with AV node and it decreases the refractory period of posterior part of ventricle Stimulation of nerves does not cause any change in the conduction of Bundle of His.

On stimulation of vagal nerve the response is fast and short,whereas stimulation ofsympathetic causes delayed and prolonged response.

Tonic stimulation of both the vagal nerve in the background continuous stimulation of stellate ganglion leads to

decreased impulse conduction inSA node but in AV node , it is summation of effect of both vagal and sympathetic nerves.

Phasic vagal stimulation is causechange in the heart rate during respiration .it leads to change insinus rate with out causing AV node delay. “Neuropeptide Y is involved in the long term regulation of autonomic responses”(17).

CARDIAC ACTION POTENTIAL:-

Transmembrane potential(TMP) of cardiac cells is -90 mv due to outflux of potassiumions across sarcolemma of cardiomyocytes.There are two types of ion channels in cardiac cells

i)Voltage sensitive channel: Operate in specific range of TMP.

ii)Time sensitive channel:opens for a fraction of time as TMP changes.

PHASES OF CARDIAC ACTION POTENTIAL:

RESTING PHASE: RMP(-90mv)

This phase is maintained by inward rectifier K+ channel.

Sodium and calcium channel remains in closed state. . “Cardiac glycosides inhibits Na+K+ATP ase pump,thereby rises sodium

concentration inside cell and also decreases the movement of calcium through Na+/Ca2+ pump leading to increased contrantion of myocardium”(18)

PHASE O DEPOLARISATION: TMP >-90mv

At -70mv fast Na+ channel open leading to rapid depolarization of of membrane potential to little more than -Omv leading to overshoot.At this instant fast Na+ channel closes,followed by opening of long acting Ca2+ channel. “Thus Inward NA and Calcium current are very important during upstroke of action potential and also “funny”current,also known as pacemaker current”(19)

PHASE I EARLYREPOLARISATION:

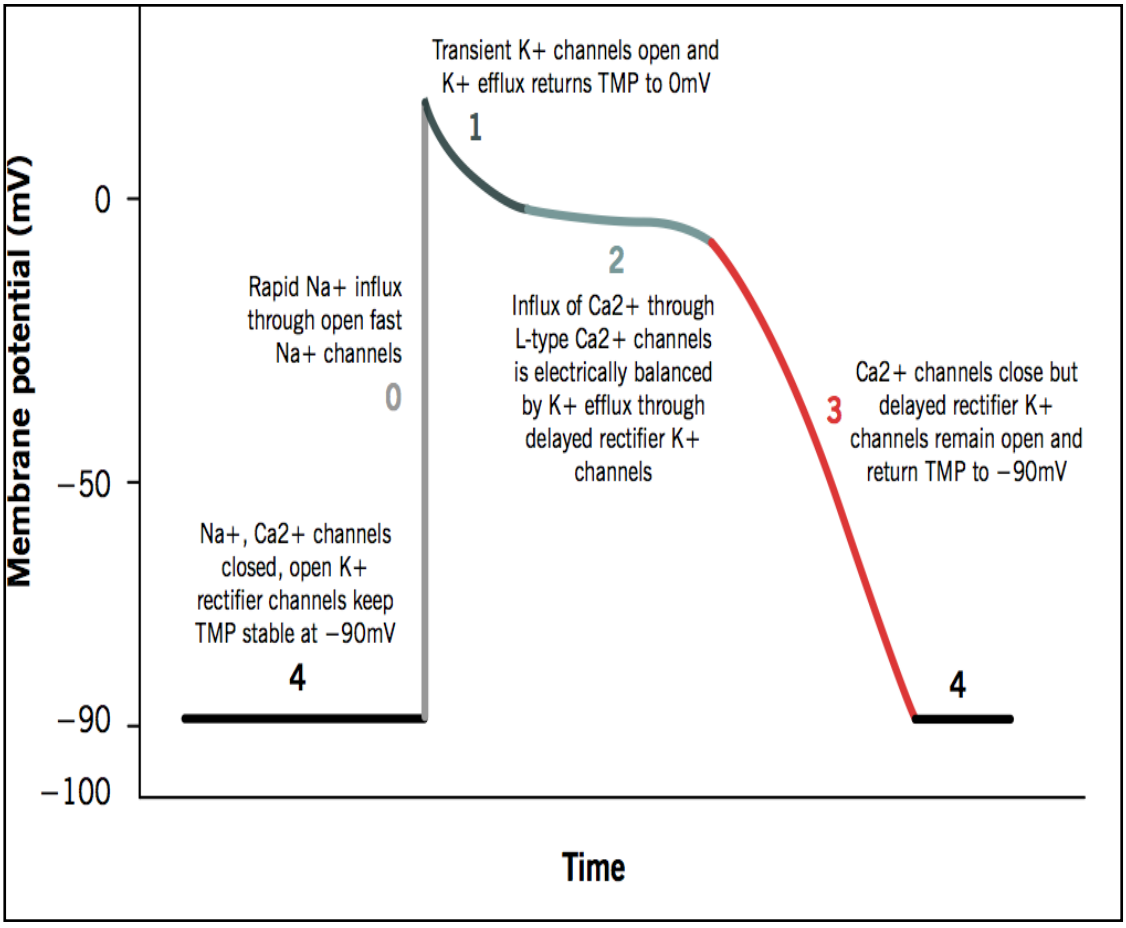
Opening of K+ channel leading to decrease inTMP to “O” mv.

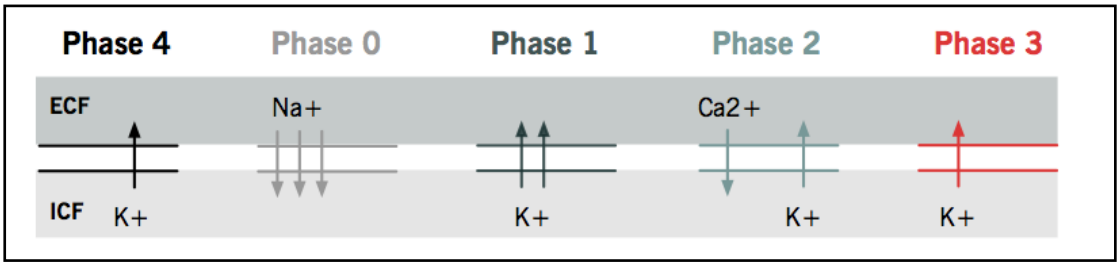
PHASE II: PLATEAU PHASE

Influx of Ca2+ through L Type channel and outflux of K+ balance each other leading to TMP just below “O”mv. “Steady concentration of Inward NA+,long acting Ca current also contributes to plateau phase”(20).

PHASE III: REPOLARISATION

K+ continues to flow out with L- type Ca channel closed leading to return of membrane potential to resting state.

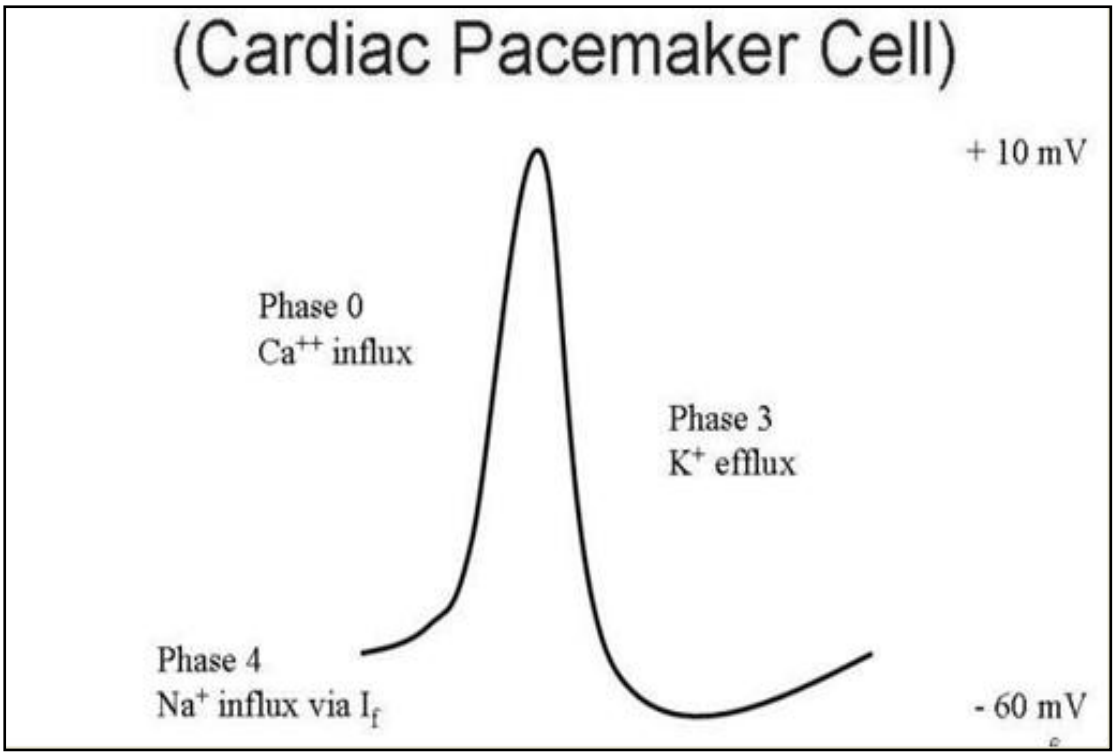


CARDIAC PACEMAKER POTENTIAL:

Pacemaker cell have inherent capacity to initiate action potential without any external stimuli.TMP of pacemaker cell is "-60mv",due to less number of K+ channels compared to other cells of heart.As TMP cannot fall to -90mv at which fast Na+ channels become active,rapid depoolarisation phase is absent.

" Due to autonomic activity the site or locus of pacemaker can shift from SA node to outside the node leading to either faster or slower discharge rate"(21).




CARDIAC CONDUCTION BLOCK:

Conduction abnormality is due to abberency in conducting system either structural,function resulting in delay or block of conduction.

MAJOR TYPES OF CONDUCTION BLOCK IN ACUTE MYOCARDIAL INFARCTION: i) Right bundle branch block

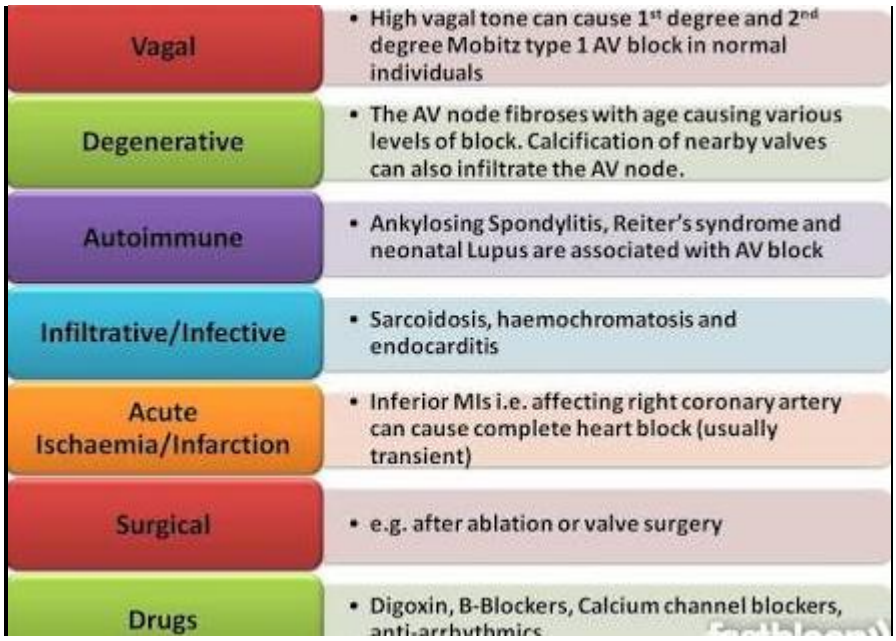
ii)Left bundle branch block

iii) I degreeblock

iv) II degree block

v) III degree or Complete heart block.

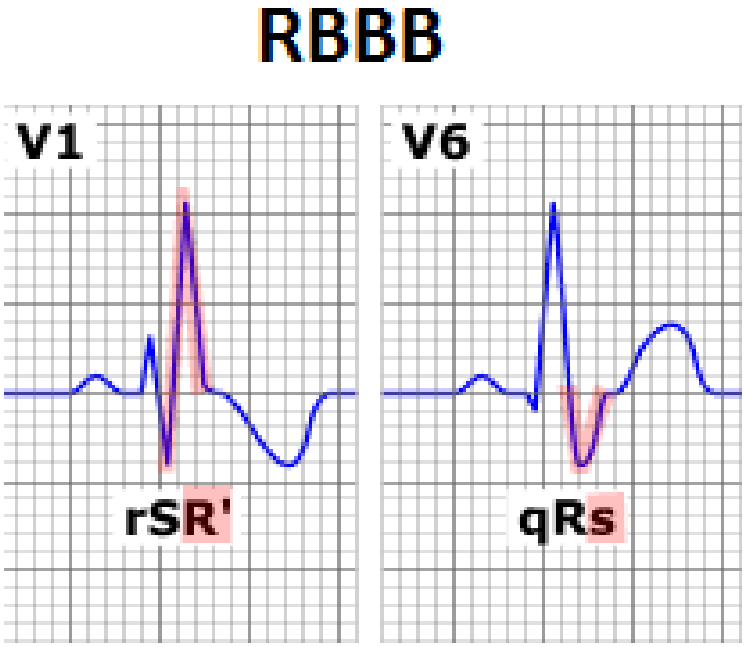
CAUSES OF CONDUCTION BLOCK:




BUNDLE BRANCH BLOCK:block in Conduction of electric impulse through either of branch in ventricle results in delay of contraction of respective ventricle.

RBBB:

RBBB is alteration in the sequence of electrical activity of His purkinjie system leading to changes in QRS,R and S waves in the form of wide QRS complex.QRS morphology can vary with axis of heart and previous MI.




Complete BBB : QRS complex is wide > 120ms.

Incomplete BBB:QRS<120ms.

"Among 3 levels of block in Right bundle,Proximal block is common than Distal and Terminal"(22,23).Terminal block is due to interruption of terminal branches during surgery.

ETIOLOGY:i) Cor pulmonale due to gradual elevation of RV pressure.

ii)Pulmonary embolism due to acute stretch of ventricle.

iii)Myocardial infarction(RBBB does not

iv)Iatrogenic(during catheterization of Right heart)

v)Functional(Rate related Block)

vi)Hyperkalemia.(ref).

Causes of RBBB which are not that common are "Hypertension,Lev's disease which is the cause of idiopathic progressive conduction disease"(25,26).

CRITERIA(AHA criteria)(24) QRS duration ≥120 sec.

Rsr', rsR' in V1(or) V2 with r',R' wider than R.

S wave of greater duration >40ms in Lead 1,V6.

R peak time > 50ms in V1 and normal in V5, V6.

Differential diagnosis: Incomplete RBBB.

Ventricular pacing.

Ventricular tachycardia

Pseudo RBBB (Brugada syndrome).

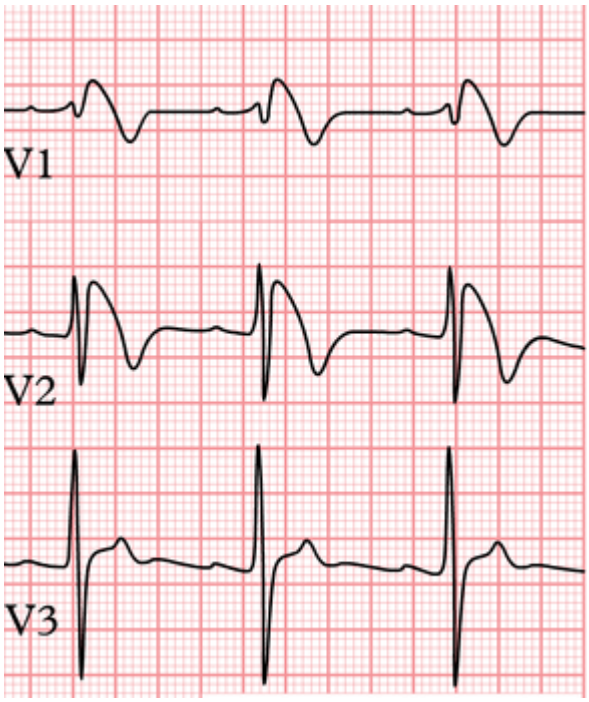
Incomplete RBBB:

In Incomplete RBBB the duration of QRS complex is less than 0.10 sec.

Ventricular pacing: " In biventricular pacing , the presence of pacemaker spike differentiate from RBBB".

Ventricular tachycardia: In Ventricular tachycardia and Idioventricular rhythm the wide QRS complex is associated with A-V Dissociation.

Brugada syndrome: “ Pseudo RBBB pattern with ST segment elevation in lead V1 to V3.”(anterior leads)without the presence of other heart disease”(27).Downsloping of ST segment ,T wave inversion,absent wide S wav in lateral leads differentiate between Pseudo RBBB and RBBB. “In this syndrome ,the transient ST changes can be brought out bydrugs belonging to sodium channel blockers”(28,29).




“Presence of RBBB in acute infarction (CVD) is associated with significant mortality”(30).,even after thrombolysis.

LEFT BUNDLE BRANCH BLOCK:

Interruption of conduction in left bundle in His purkinjie system leading to alteration in sequential activation of vntricles.

“Presence of LBBB is associated with underlying cardiovascular disease”(31) than RBBB and implies poor prognosis in case of coexistence with acute MI and decrease in ejection fraction in case of heart failure.Block can be present proximally or distally.

Etiology:

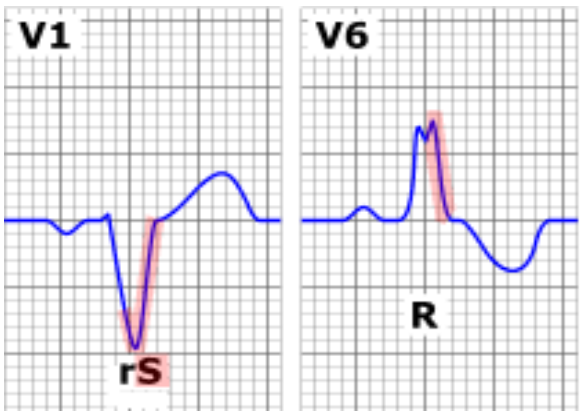
(i) Anterior MI

(ii) Myocardial fibrosis (Sytemic hypertension ,CAD ,DCM).

(iii) Functional (rate related).

(iv) Ventricular tachycardia

(v) “Hyperkalemia ,is a rare cause of LBBB”(34,35 ).



CRITERIA: (AHA(24)

" Durastion of QRS complex ≥ 120ms.

Broad R wave in lead 1,avL,V5,V6.

Absent q waves in lead I, V1,V6.

R peak time > 60ms inV5, V6.

ST-T disconcordance

Negative QRS –T concordance may be associated with underlying ischemia.

"LBBB in TYPE II DM and CHD is independent predictor of mortality"(32).

Differential diagnosis: Incomplete LBBB

Ventricular hypertrophy.

Myocardial ischemia

Ventricular tachycardia.

Ventricular pacing.

Ventricular pre excitation (WPW syndrome).

Incomplete LBBB: Duration of QRS complex 0.10-0.12 sec.

Pseudo Delta wave in V6 and lead I.

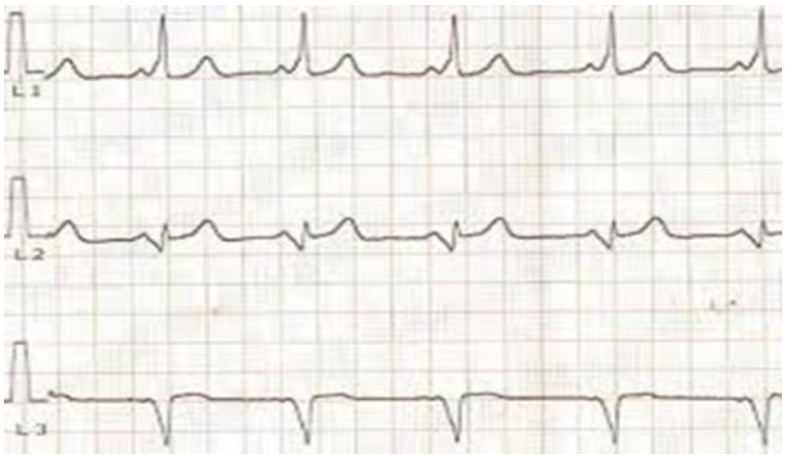
"Incomplete LBBB may also occur as median fasicular block"(33).

Ventricular tachycardia:

Wide QRS associated with A-V dissocation.

Ventricular pacing: pacemaker spikes along with LBBB pattern.

Ventricular pre excitation:




Resembles LBBB pattern but with shortened PR interval.

Unlike RBBB,there is a need for pacemaker therapy in many circumstances.

LBBB+Mod to severe LV dysfunction.

LBBB+ Symptomatic AV block.

LBBB+II,III degree AV block.

LBBB+Acute MI (Temporary pace maker).

LBBB+Right heart catheterization.

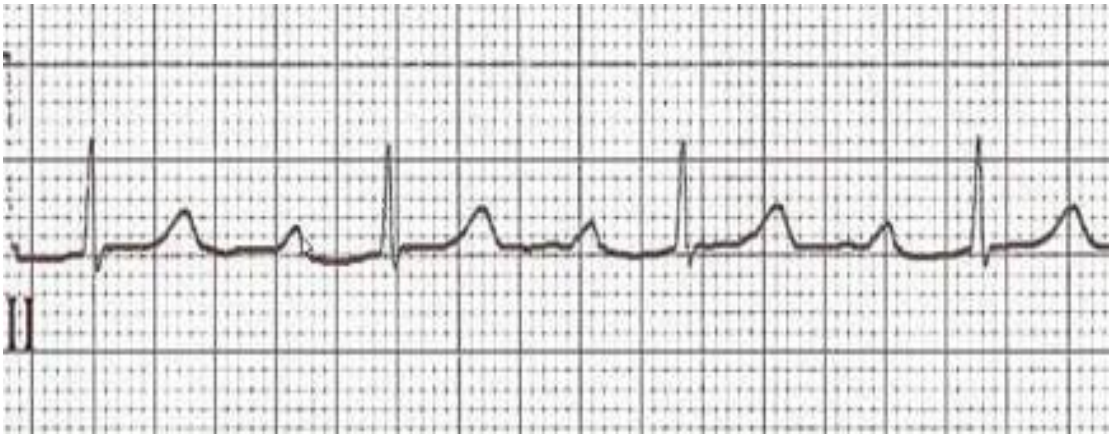
Asymptomatic LBBB with no CHD does not require treatment.

HEART BLOCK:

" Delay (or) interruption in conduction of electrical impulse from atrium down to ventricle leading to dyssynchronisation of both atria and ventricular depolarization".

FIRST DEGREE HEART BLOCK:

Delay in the form of prolonged duration of PR with out block in conduction.




ETIOLOGY:

- Increase in vagal tone either physiologic (or) pathologic'

i)physiologic as in athlete

ii)pathologic as in autonomic dysfunction or drugs like Digoxin which is vagotonic.

- Myocardial infarction:
- (i) In IWMI AV node delay is because of blood supply(i.e)both are supplied by RCA.

  (ii) In AWMI it is associated with BBB resulting in wide "QRS" complex when compared to IWMI which results in narrow QRS COMPLEX.

  Diagram
- Structural defect in AV node.

- Drugs like non dihydropyridine ca2+ blockers (interferes with depolarizing current),Digoxin, 'Na2+' channel blockers causing block in Bundle of his.
- DCM
- Lyme disease
- Lev disease.

It is usually asymptomatic ,diagnosed using (i)Routine ecg

(ii)EPS

Criteria:- (i)PR duration >200ms in ECG

(ii) prolonged AH duration>300ms.

(iii) Infranodal:HV interval>100ms.

History of cardiac disease ,drug intake ,lifestyle(athlete) should be considered.Atropine causes rapid conduction in AV node and also SA node.It thereby reverts AV node delay but worsens the block in infra nodal delay due to increased duration of refractory period ,because of increased rate of SA nodal discharge.

Usually benign,not associated with increased mortality in Acute MI.In some conditions there is need for pacemaker as folloes

(i)Pacemaker syndrome it occurs due to decreased time available for atrium to fill as it immediately follows ventricular contraction, leading to "awareness of one's own heart beat".

(ii) In neuromuscular disease, as it may progress to high degree.

(iii) Wide QRS complex(HV>100ms) in infranodal delay.

(iv) I degree block with AV dissociation.

II DEGREE AV BLOCK:-

This is due to intermittent block of electric impulse through AV node , which may be of (i) regular pattern

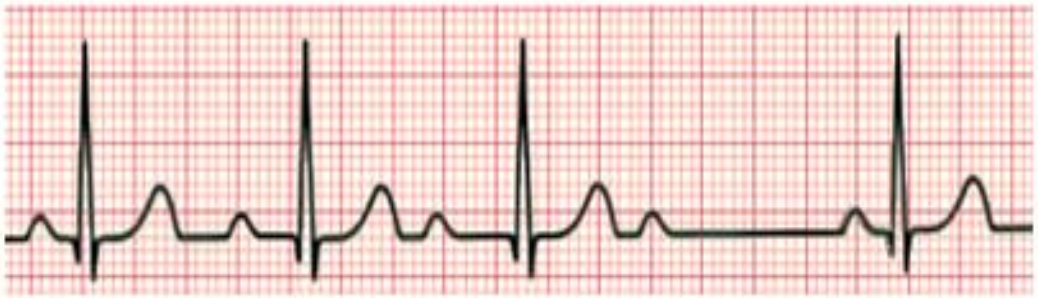
(ii) Mobitz type 1 and II

(iii) high grade.

Regular pattern :the block,variable ,may be of 2:1,3:1 due to one non conducted P at a time .

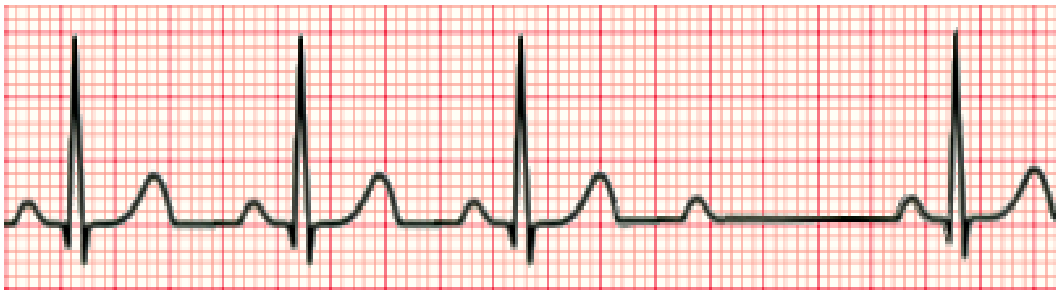
In mobitz type 1,PR duration is prolonged in progressive way ultimately resulting in a drop beat then conducting normally followed by similar cycle.

Mobitz I or Wenckebach

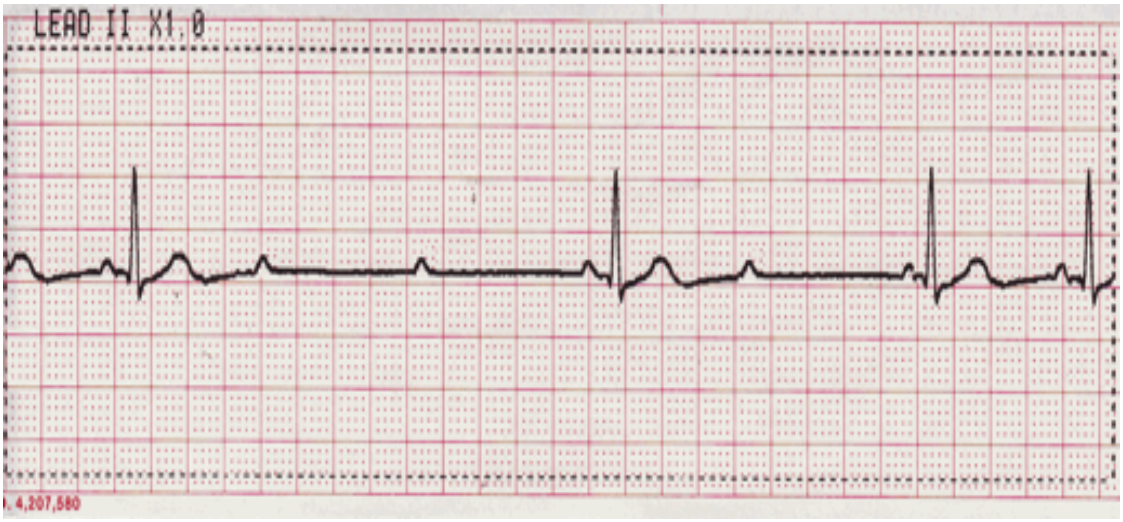


In mobitz type II PR interval is not changed , before and after a non conducted “P” WAVE.

Mobitz II



High grade: in which more than one P wave are not conducted




CAUSE OF II DEGREE BLOCK:

(i)Physiologic

(ii) Increased vagal tone

(iii) Myocardial infarction

(iv) Myocarditis

(v) Endocarditis

(vi) Hyperkalemia

Type 1 is usually benign,may cause irregular pulse,syncope and in acute MI it may lead to decreased cardiac output.it responds to Atropine and is worsened by carotid sinus massage.

Diagnosed by EPS ,which is indicated in syncope,the findings are progressive A-H and constant H-V ,finally no H-V electrogram,

THERAPY:

(i) Atropine(in hemodynamically unstable)

(ii) Temporary pacemaker (in Acute MI)

(iii) Permanent pacemaker(in hemodynamically stable).

MOBITZ TYPE 2 BLOCK:

Unlike Type 1 Mobitz block,it is mostly associated with heart disease. It usually presents with varying symptoms and are rarely stable, due to decreased cardiac output and irregular pulse.the symptoms are severe in patients with bradycardia and heart failure.”the blockis below AV Node and “ in twenty percent it is at bundle of HIS”(ref).In type 2 mobitz block increased heart rate ,as in case of exercise and atropine can get worse if block is below his bundle .carotid sinus massage may improve it.

It is diagnosed by EPS study,here “ His deflection follows non conducted A wave,but in case of more proximal block It is not so” , leading to misdiagnosis as AV Node block.(ref)

THERAPY(i) Atropine and temporary pacing :

–in case of hemodynamically unstable ,usually in reversible cases like MI,decreased Thyroid,lyme disease and drugs.

(ii) Permanent pacing :in case of irreversible causes (ref).

III DEGREE AV BLOCK:

It is a type of conduction block in which the electrical impulse from the atria does not reach the ventricle .

CAUSE: The etiology of III degree block is same as that of other A V blocks.

| Nonpharmacologic Causes of Atrioventricular Dissociation | |
|---|---|
| Degenerative diseases | Lenègre disease<br>Nail-patella syndrome<br>Mitochondrial myopathy |
| Infectious causes | Lyme borreliosis<br>*Trypanosoma cruzi*<br>Rheumatic fever<br>Chagas disease<br>*Aspergillus* myocarditis<br>Varicella zoster virus |
| Rheumatic diseases | Ankylosing spondylitis<br>Reiter syndrome<br>Rheumatoid arthritis<br>Scleroderma |
| Infiltrative processes | Amyloidosis<br>Sarcoidosis |
| Metabolic disorders | Hypoxia<br>Hyperkalemia<br>Hypothyroidism |
| Neuromuscular disorders | Becker muscular dystrophy<br>Myotonic muscular dystrophy |

'Sarcoidosis should be considered in case of idiopathic block in elderly as it is present in 25-35% of patients'.

Almost all patiens present with symptoms of varying severity, especially when the ventricular rate is decreased.

The complete heart block is diagnosed by

(i)ECG:P and QRS waves are not related to one another ,with atrial rate >ventricular rate.

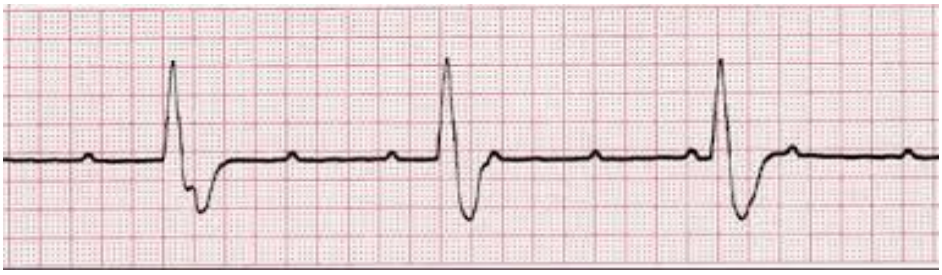
The origin of escape rhythm [ arising from other than SA node] is determined by QRS complex

AV node or junction :narrow QRS complex

Below AV node , Bundle of His : narrow QRS complex

Below bundle of His : Wide QRS complex

DIAGNOSIS:



(I)ECG

(II)EPS : to detect site of block

THERAPY

In unstable patients atrophine and temporary pacemaker are used, especially in reversible causes like myocardial infarction, Lymes disease etc.

Along with that vasopressors like Dopamine,Dobutamine [ In heart failure] is used

After stabilization permanent pacemakers are used, especially if the cause is irreversible.

MYOCARDIAL INFARCTION

Injury to the myocardium due to inadequate blood supply

ACUTE CORONARY SYNDROME

Suspicious of ischemia

- STEMI
- NSTEMI
- UA

| | Stable Angina | Unstable Angina | STEMI | NSTEMI |
|---|---|---|---|---|
| Character of pain | Exertional pain | Rest pain | Rest pain | Rest pain |
| Relievers | Responds to GTN | No GTN effect | No GTN effect | No GTN effect |
| Enzymes | Normal | Normal | Elevated | Elevated |
| ECG | Often normal | Often ST depression | ST segment elevation | No ST segment elevation |

DIAGNOSIS

(I) ECG

(II) CARDIAC BIOMARKERS

(III) IMAGING MODALITY

(IV) CORONARY ANGIOGRAPHY

CRITERIA FOR ACUTE MI :

Rise or fall in cardiac biomarkers especially Troponin with ≥ one value higher than 99 percentile of the upper limit plus one of the following criteria

(i)Symptoms of ischemia

(ii) Formation of pathological Q wave

(iii) New LBBB or ST-T change

(iv) Angiographic detection of intracoronary thrombus

(v) Imaging evidence of regional wall motion abnormality,loss of viability of myocardium.

- For type I MI the above criteria is used [ conditions resulting in thrombus formation in coronary artery]
- For type III MI cardiac biomarkers cannot be obtained due to sudden of cardiac death
- For type IVA above criteria with biomarkers > 5X 99 percentile[in patients with baseline value in normal limits] or more than 20% if the values are higher but stable or lower than normal limits.
- For type IV B above criteria with identification of thrombus in the stent either during angiography or autopsy
- For type V additionally the cardiac enzymes should be more than 10X 99 percentile of upper limit

TYPES OF MI

I.Spontaneous

II.Secondary to ischemic imbalance

III Sudden cardiac death

IVA PCI related

IVB Stent thrombus

V CABG related

ST Elevation MI:

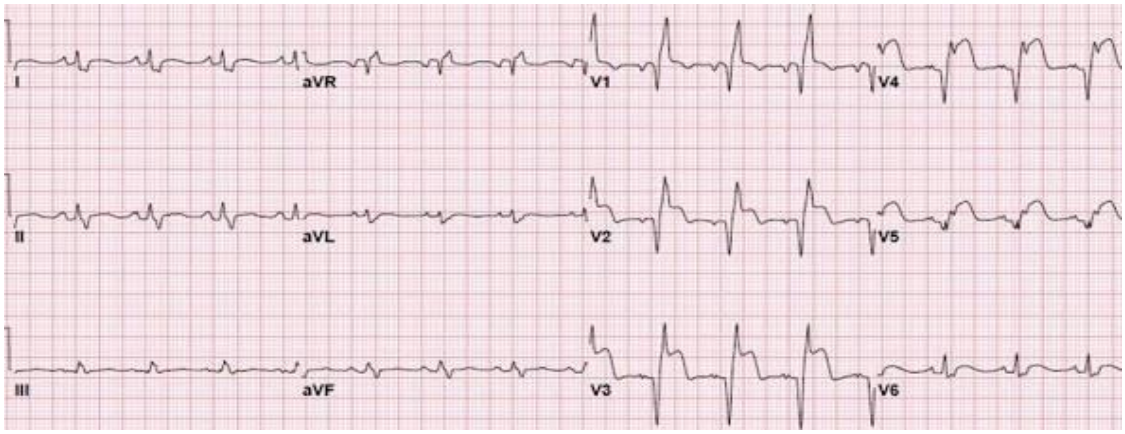
ECG CRITERIA:according to AHA criteria ",New ST segment elevation of>0.1mv in two contiguous leads other than lead V2,V3 for which

(I) Elevation of ≥0.2 in male ≥ 40 years of age

(II) Elevation of≥0.25 mv in male less than 40 years of age

(III) Female with elevation of ST segment ≥0.15m,v.

Initially it starts as tall " T" wave then in hours to two weeks there occurs Q waves,T inversion ,reduced R wave amplitude.




In patients with noQ waves in ST elevation ECG the prognosis is found to be good.

New onset LBBB and posterior wall MI with ST segment elevation of 0.5mv and above is also considered.

| | |
|---|---|
| **Cardiac** | |
| | Pericarditis<br>Ventricular Aneurysm<br>Brugada Syndrome<br>Left Bundle Branch Block |
| **Intracranial** | Stroke<br>Raised Intracranial Pressure<br>Intracranial Haemorrhage |
| **Abdominal** | Peritonitis |
| **Drugs** | Digoxin<br>Isoprenaline<br>Quinidine<br>Procainamide |
| **Metabolic** | Hypothermia<br>Hyperventilation<br>Hyperkalaemia |
| **Other** | Spinal Cord injury<br>Pulmonary Embolism |

NSTEMI:

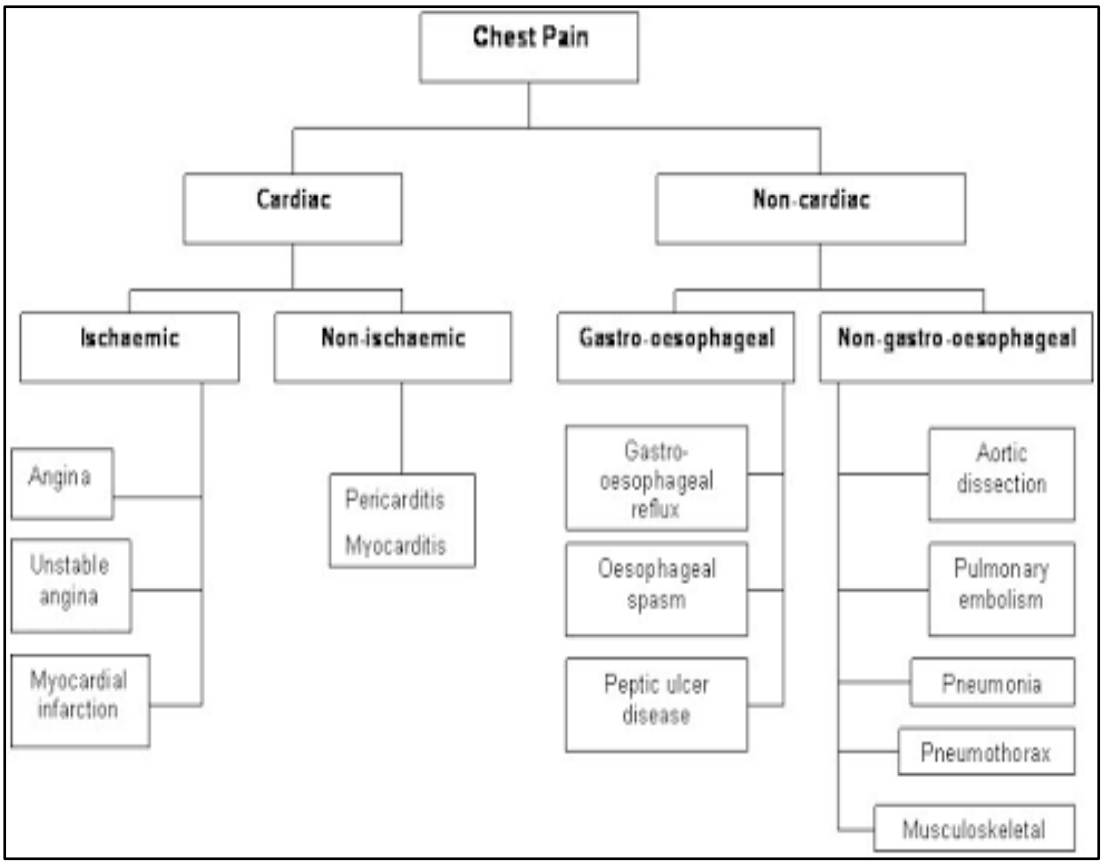
Here there is no ST segment elevation instead there is ST-T change with elevated cardiac enzymes.,and those changes are almost persistent.

UNSTABLE ANGINA:

Here there is ST-T change other than elevation with no increase in cardiac enzymes as it represents ischemia rather than infarction.,and ST-T changes are temporary.

ANGINA PECTORIS represents classical angina pain and also increase in duration,intensity,even less work precipitating pain in chronic stable angina represents active damage of myocardium.

There are other conditions which cause chest pain which are considered in differential diagnosis of chest pain. ,like in CVS ,Respiratory and GI conditions.




CARDIAC BIOMARKERS:

Troponin T is both sensitive and specific than other markers ,especially CK MB.The level of elevation of Troponin are significant in a specific way for different MI as mentioned above in the criterias above.It is mostly elevated in 3 hours of MI , so levels are checked after 6 hours of initial reading and after 12 hours it can be checked again to rule out.However if it is persistently elevated more than 2 weeks, then chronic event should be suspected .if the levei increases >20% of previous value in apatient with MI then “REINFARCTION” should be considered.

The “CARDIAC INJURY” is the term used for conditions other than MI leading to elevated cardiac biomarkers as follows.

(i)Heart failure

(ii)myocarditis,pericarditis

(iii)chronic kidney disease

(iv)Sepsis

(iv) Subendocardial infarction

(v) Atrial fibrillation.

Mortality rate is increased in patients with elevated TEROPONIN when compared to those with negative troponin,CK MB elevation.

SITE OF INFARCT AND ECG CHANGE WITH BLOOD SUPPLY

| lead | border | Arterial supply |
|---|---|---|
| V3 & V4 | anterior Right Ventricle | RCA |
| V1 & V2 | Septum | LAD |
| a VL, V5 & V6 | Lateral Left Ventricle | LCX |
| II+III+AVF | inferior borderof right ventricle | RCA |

## MATERIAL AND METHOD:-

**Study design** : Prospective Observational Study

**Study period** : 6 months

**Study Area** : GovtKilpauk Medical College,Chennai.

**Study population:** Myocardial Infarction patients admitted in Intensive Coronary Care unit, GovtKilpauk Medical College Hospital,Chennai.

**Sample Size** : **200**

## CASE DEFINITION:

Conduction block is defined as delay or interruption in conduction of the atrial impulse through the specialized conducting system of heart.

It includes AV conduction blocks,Bundle blocks and Fasicular blocks.

ST elevation MI is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent ECG ST elevation .

Acute ST Elevation myocardial infarction as per W.H.O. criteria[8]as follows;A new ST elevation at the J point in atleast two contiguous leads

of ≥ 2mm in men or ≥1.5 mm in women in leads V2-V3 and/or of ≥ 1mm in other contiguous chest leads or the limb leads.

**INCLUSION CRITERIA:**

Patients having Acute ST elevation myocardial infarction.

**EXCLUSION CRITERIA:**

1. patients with cardiomyopathy.

2. patients with congenital or rheumatic heart disease.

3. patients with history of intake of drugs causing conduction

blocks like clonidine, methyl dopa, verapamil, digoxin etc.

**METHODOLOGY**

The study is a clinical, prospective and observational study conducted at GovtKilpauk Medical College Hospital,Chennai.All patients included in the study will be explained about the procedure in detail and issued patient information sheet.Informed/written consent will be taken in each case.patient will be monitored for serial changes in ECG for 24

hours by trained medical personnel.Twice daily ECG printout along with any change in type of conduction block and other events will also be recorded through out the inhospital stay period.All the investigations and interventions will be done under the direct supervision and guidance of our guide.

After obtaining a detailed history, a complete general physical and systemic examination, the patients will be subjected to following investigations.

Complete blood count

Urine analysis

Renal function test

Liver function test

Lipid profile

Chest Xray

ECG

Echocardiogram

The complete data will be recorded in a specially designed Case Recording Form. The data collected will be transferred into a Master Chart which is subjected to statistical analysis. Patients are selected with the following Inclusion and Exclusion Criteria.

**DATA ANALYSIS:**

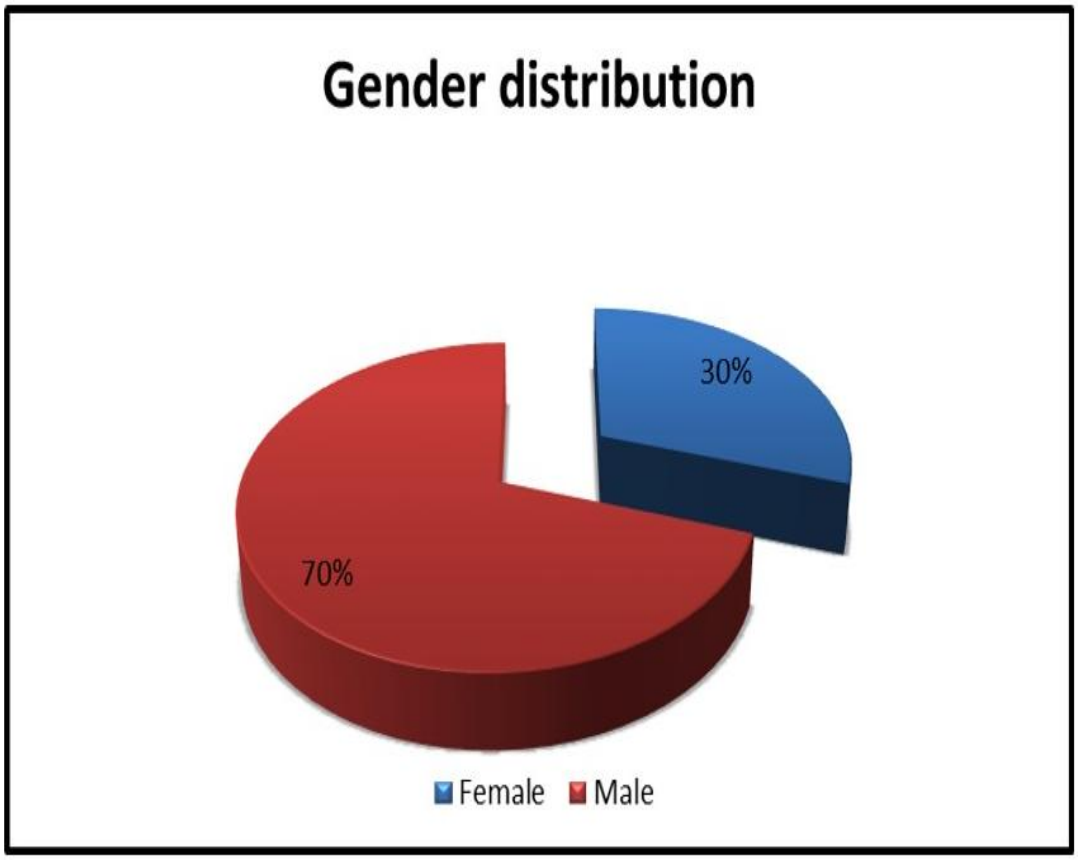
Incidence of conduction block will be provided as %.Different patterns of conduction block will be provided as % with confidence interval of 95.The prognostic implications in terms of mortality will be provided as %

# OBSERVATION

# & RESULTS

## OBSERVATION AND RESULTS

In this study of 200 patients, presenting with acute STEMI ,number of female patients were found to be 60 and number of male was 140.therefore incidence of MI is more when compared to female in male.



**SEX**

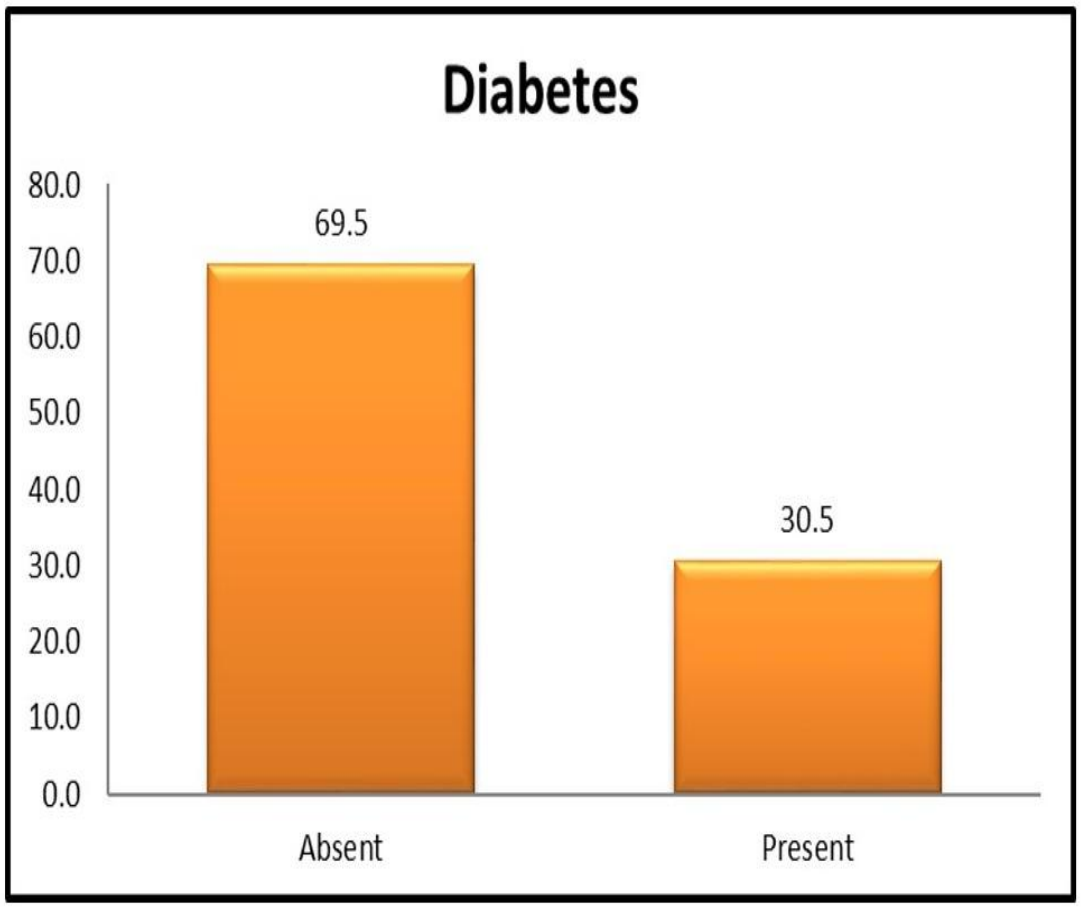
| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Female | 60 | 30.0 | 30.0 | 30.0 |
| | Male | 140 | 70.0 | 70.0 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

DIABETES AND MYOCARDIAL INFARCTION :

The distribution of diabetes among MI patient in this study is ,among 200 patients, 61 patients were found to be having Diabetes and 139 were not having Diabetes.The percentage of people having Diabetes with MI was 30.5% and those presented with MI not having Diabetes is 69.5%.

**DIABETES**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Absent | 139 | 69.5 | 69.5 | 69.5 |
| | Present | 61 | 30.5 | 30.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

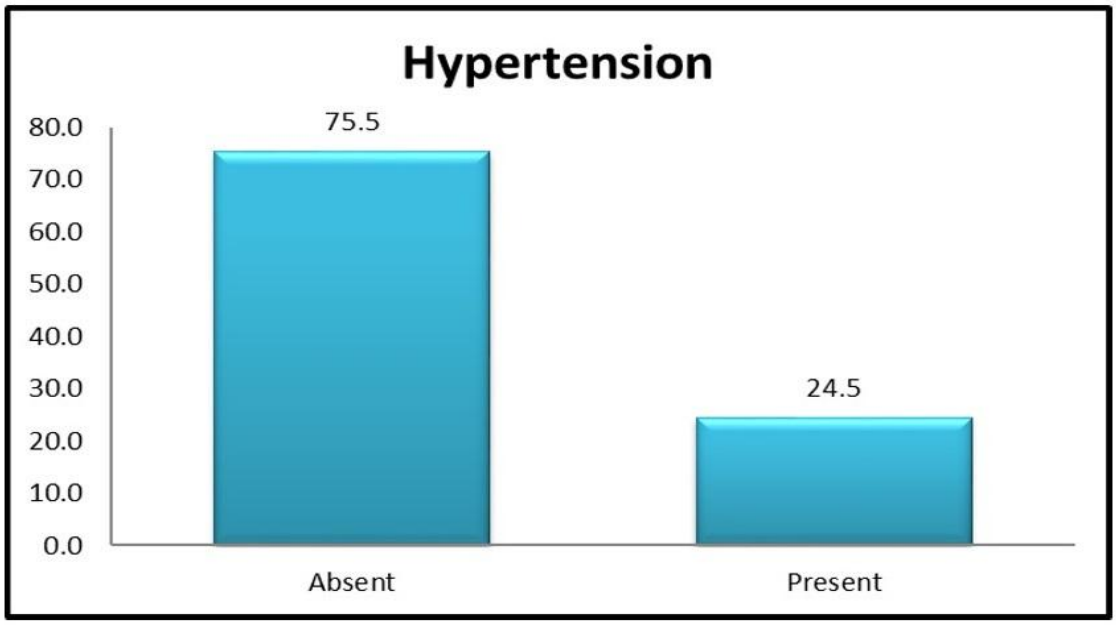



HYPERTENSION AND MYOCARDIAL INFARCTION:

Amongst 200 patients of acute STEMI 49 were hypertensives and 151 were not having hypertension.The distribution of hypertensionin patients with MI inthis study is 24.5%.

**HYPERTENSION**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Absent | 151 | 75.5 | 75.5 | 75.5 |
| | Present | 49 | 24.5 | 24.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

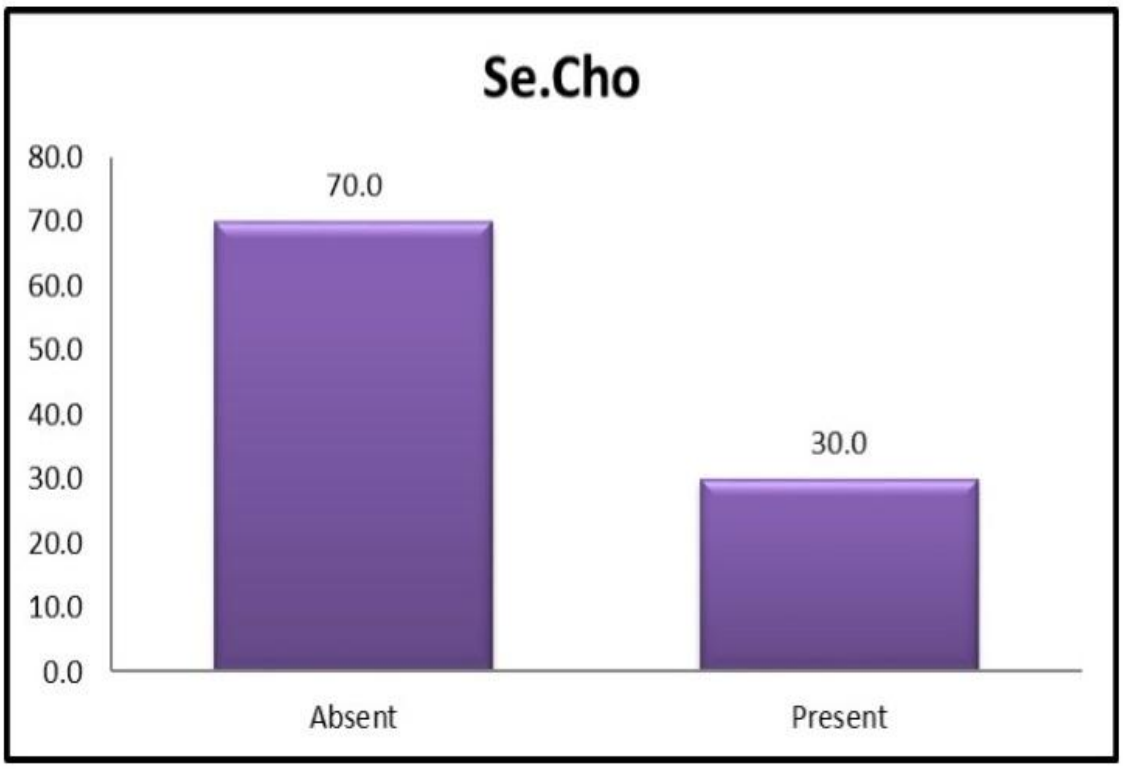


SERUM CHOLESTEROL:

The patients with MI having elevated serum cholesterol is 60.those not having elevated serum cholesterol is 140.the percentage of MI patients (200) ,having raised serum cholesterol is 30% and those with out elevated serum cholesterol is 70%.

**SE.CHO**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Absent | 140 | 70.0 | 70.0 | 70.0 |
| | Present | 60 | 30.0 | 30.0 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

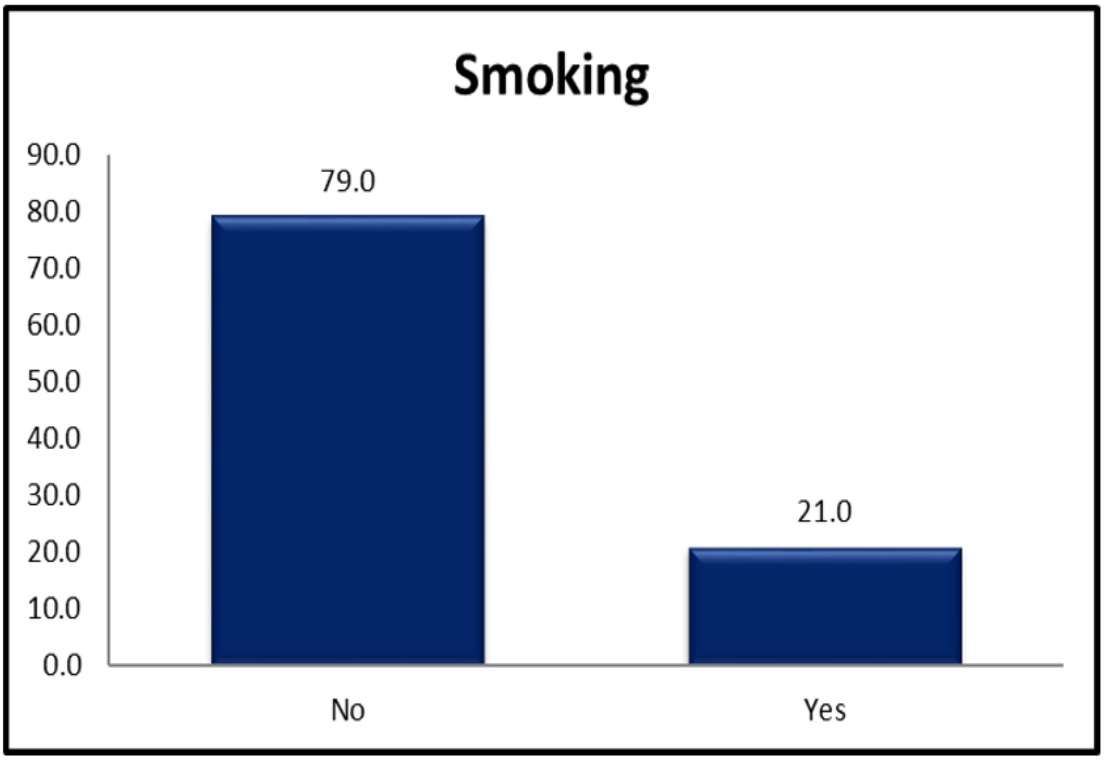



SMOKING:

42 patients with Acute MI were having smoking history,and 158 patients were non smokers with total number of 200.the percentage of MI patients who are smokers was21% .the percentage of non smokers among 200 MI patients was 79%.

**SMOKING**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | No | 158 | 79.0 | 79.0 | 79.0 |
| | Yes | 42 | 21.0 | 21.0 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

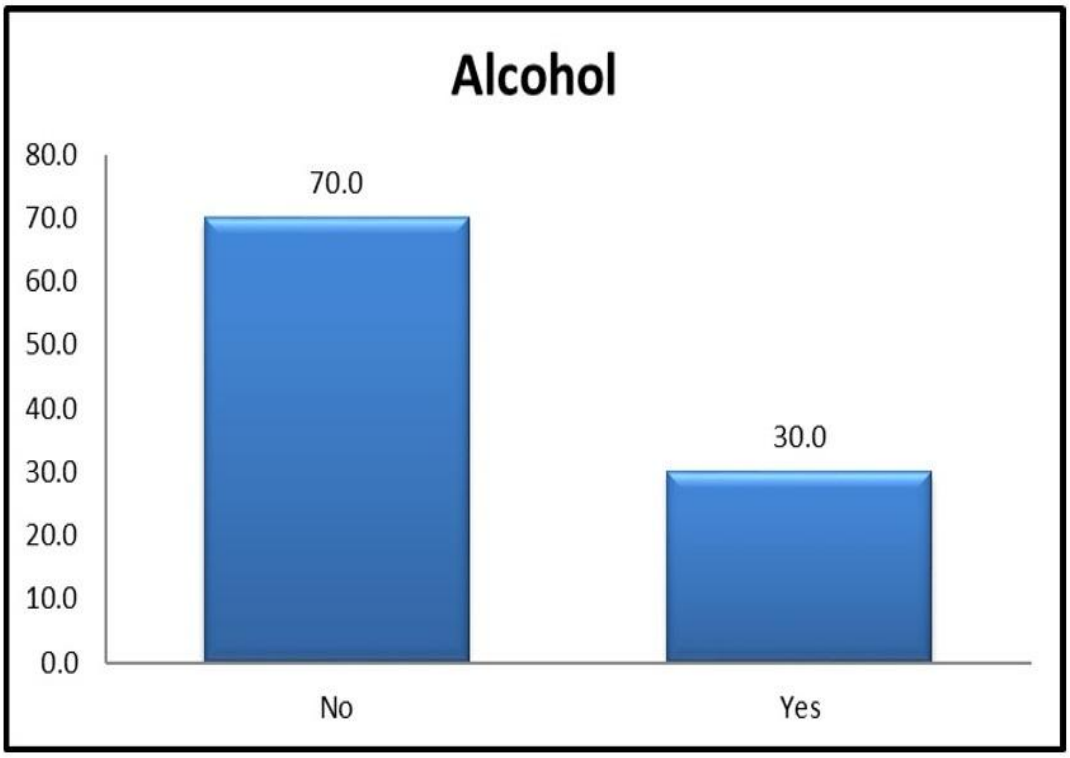


ALCOHOL:

Among 200 MI patients 60 are Alcoholic and 140 are non alcoholic.the percentage of Alcoholics in Acute MI patients was 30% and non alcoholics in 200 MI patients was70

**ALCOHOL**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | No | 140 | 70.0 | 70.0 | 70.0 |
| | Yes | 60 | 30.0 | 30.0 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

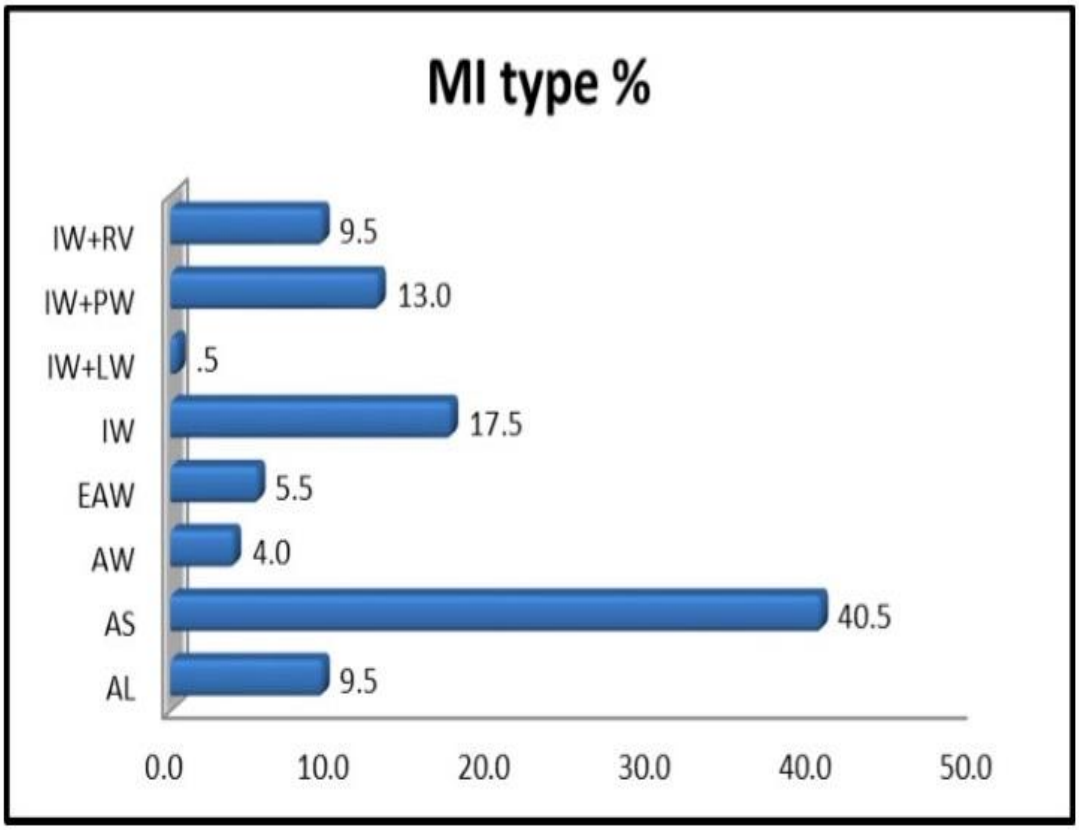



MYOCARDIAL INFARCTION TYPE:

Among 200 patients presented with MI ,19 were having ALMI,81 had ASMIwhich is majority type ,8 patients had AWMI,and 11 patients were having EAWMI.

With IWMI 35 had only IW infarction ,26 had both IW+PWMI .19 patients han IW+RVMI.where as 1 patient presented with IW+LWMI.

Among inferior wall MI involvement of only iw is found in majority of patients




**AI**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Inferior | 81 | 40.5 | 40.5 | 40.5 |
| | Anterior | 119 | 59.5 | 59.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

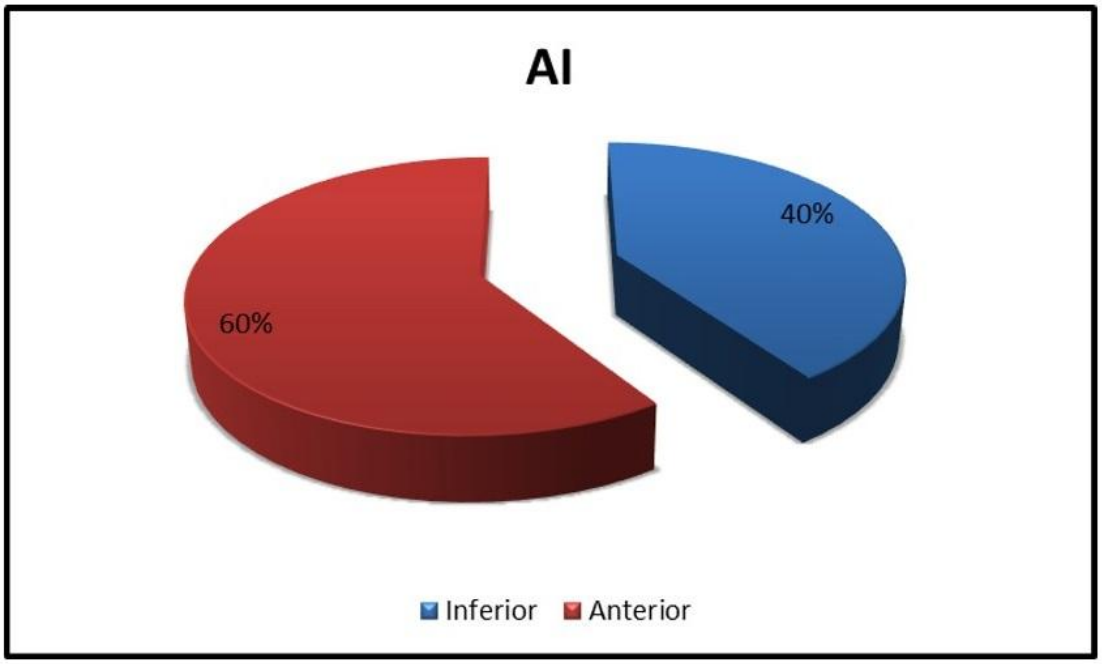
**MI TYPE**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | AL | 19 | 9.5 | 9.5 | 9.5 |
| | AS | 81 | 40.5 | 40.5 | 50.0 |
| | AW | 8 | 4.0 | 4.0 | 54.0 |
| | EAW | 11 | 5.5 | 5.5 | 59.5 |
| | IW | 35 | 17.5 | 17.5 | 77.0 |
| | IW+LW | 1 | .5 | .5 | 77.5 |
| | IW+PW | 26 | 13.0 | 13.0 | 90.5 |
| | IW+RV | 19 | 9.5 | 9.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

ANTERIOR AND INFERIOR INFARCTION:

Among 200 people presented with Acute MI,the frequency of occurrence of Anterior wall associated infarctions

are more than Inferior infarctions.119 out of 200 MI patients had Anterior infarction while 81 had infarction associated with Inferior wall.The percentage of Inferior wall associated MI is 40.5%.The percentage of anterior wall associated MI is 59.5%.The patients who presented with both Anterior and Inferior wall MI is included in both Anterior wall MI and Inferior wall MI .



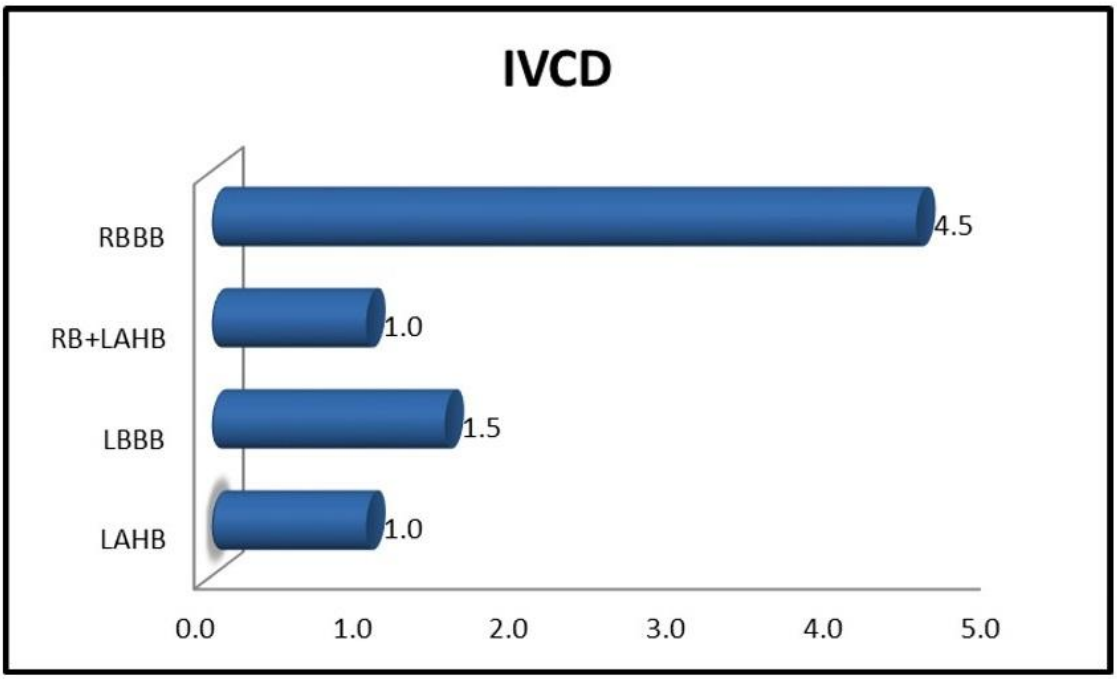
**IVCD**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | LAHB | 2 | 1.0 | 1.0 | 1.0 |
| | LBBB | 3 | 1.5 | 1.5 | 2.5 |
| | N | 184 | 92.0 | 92.0 | 94.5 |
| | RB+LAHB | 2 | 1.0 | 1.0 | 95.5 |
| | RBBB | 9 | 4.5 | 4.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

INTRAVENTRICULAR CONDUCTION DELAY IN MI

Amongst 200 MI patients,16 patients presented with IVCD.In these 19 patients with IVCD ,RBBB was present among 9 patients.3 had LBBB.2 patients had both RBBB and LAHB .Only LAHB was present in 2 patients.The remaining 184 patients did not develop BBB.

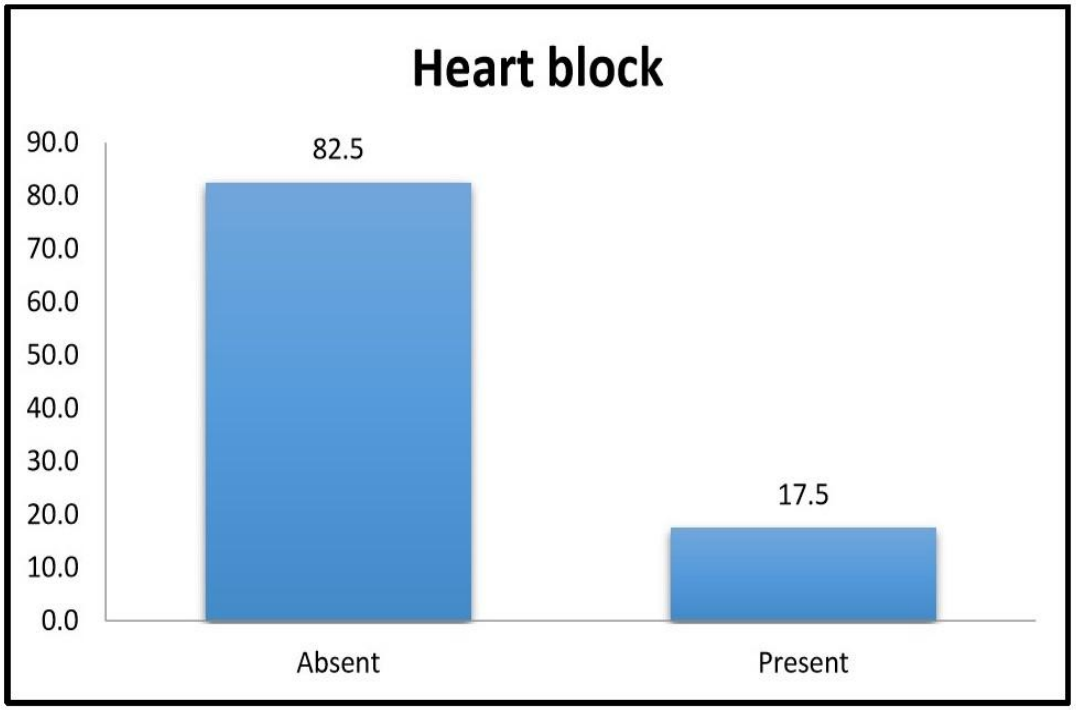
In this study RBBB is more common than LBBB.


IVCD
RBBB
RB+LAHB
LBBB
LAHB
4.5
1.0
1.5
1.0
0.0
1.0
2.0
3.0
4.0
5.0

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Absent | 165 | 82.5 | 82.5 | 82.5 |
| | Present | 35 | 17.5 | 17.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

ATRIOVENTRICULAR BLOCK:

In 200 patients with Acute MI, 178 had no AV block.Remaining 22 patients had AV block.The occurrence of type of AV block as follows.,12 patients had type 1 AV block.Type 1,3 transition was present in 5 patients.2 patients had Type 2 AV block .2,3 transition was present in 1 patient.Type 3 block was present among 2 patients.Transition to Type 3 block has occered in 6 patients.

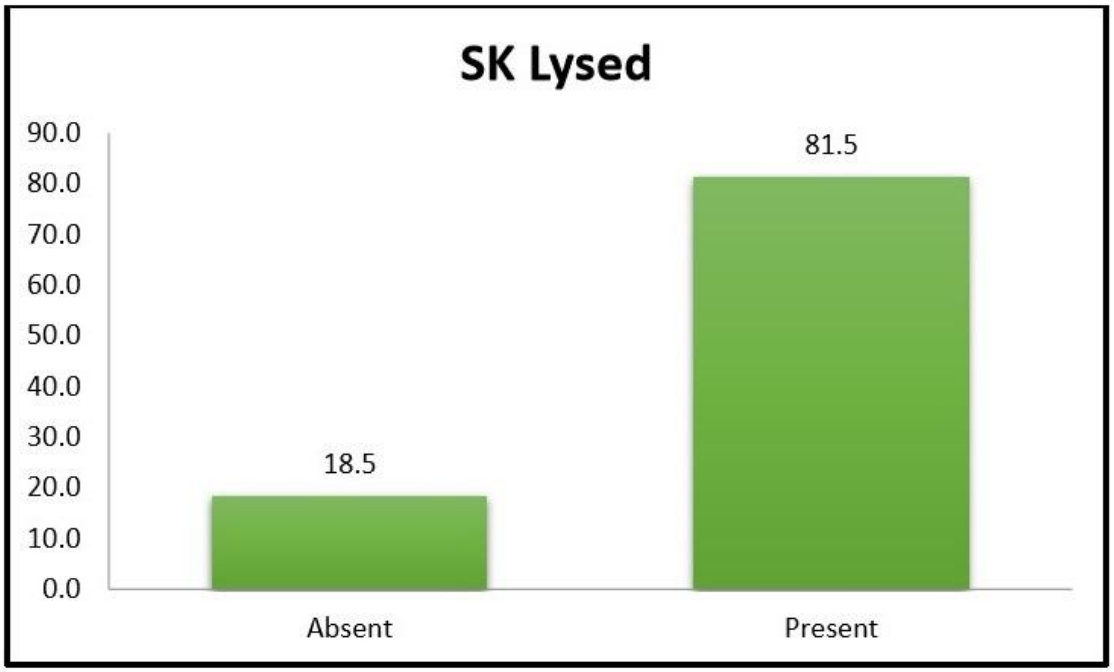



STREPTOKINASE LYSED MI:

**SK LYSED**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Absent | 37 | 18.5 | 18.5 | 18.5 |
| | Present | 163 | 81.5 | 81.5 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

Among 200 patients admirtted with Myocardial infarction, 163 patients were treated with streptokinase and37 out of 200 patients were not thrombolysed with streptokinase.

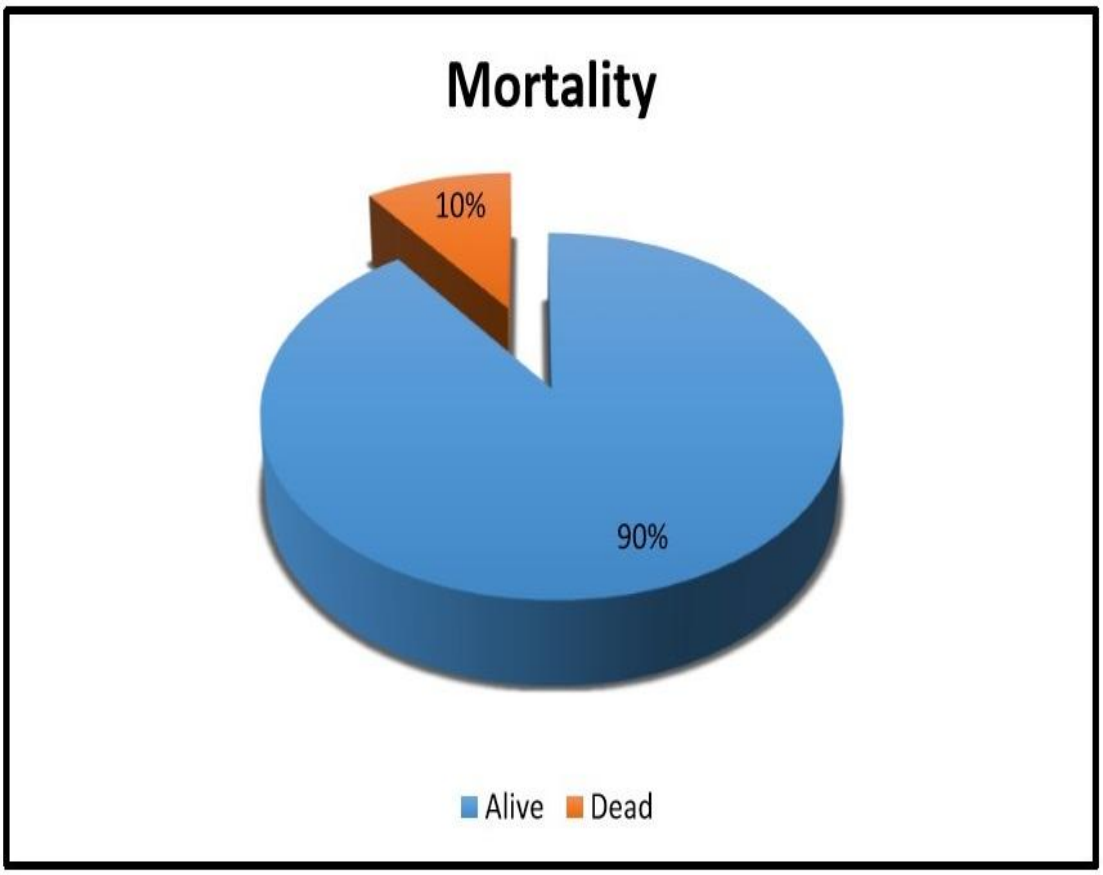

SK Lysed
90.0
80.0
70.0
60.0
50.0
40.0
30.0
20.0
10.0
0.0
18.5
81.5
Absent
Present

**MORTALITY**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Alive | 180 | 90.0 | 90.0 | 90.0 |
| | Dead | 20 | 10.0 | 10.0 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |

Among 200 patients admitted with Myocardial Infarction,180 people were found to be alive where as 20 out of 200 expired.

The percentage of alive patients is 90% ,the percentage of patients expired were 90%.

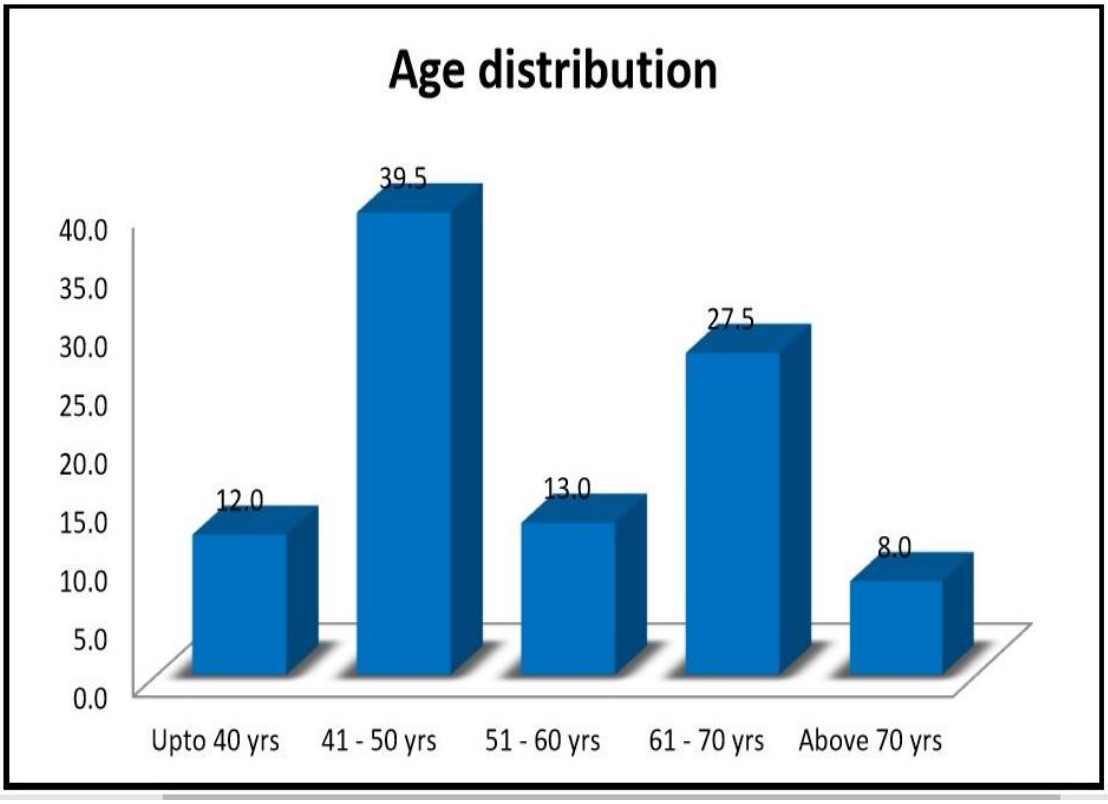



AGE DISTRIBUTION AND MYOCARDIAL INFARCTION:

Out of 200 MI patients 24 were less than 40 years.79 patients were between 49 to 50 years of age.26 patients were between 51 to 60 years of age.among patients between 61 to 70 years of age 55 were present.above 70 years 16 patients were there'.

**AGERANGE**

| | | Frequency | Percent | Valid Percent | Cumulative Percent |
|---|---|---|---|---|---|
| Valid | Upto 40 yrs | 24 | 12.0 | 12.0 | 12.0 |
| | 41 - 50 yrs | 79 | 39.5 | 39.5 | 51.5 |
| | 51 - 60 yrs | 26 | 13.0 | 13.0 | 64.5 |
| | 61 - 70 yrs | 55 | 27.5 | 27.5 | 92.0 |
| | Above 70 yrs | 16 | 8.0 | 8.0 | 100.0 |
| | Total | 200 | 100.0 | 100.0 | |



Maximum percentage of people with MI were between 41 to 51 years of age’

The minimum age of patient presented with MI was 29.

The maximum age was 78.

**Descriptive Statistics**

| | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| AGE | 200 | 29 | 78 | 53.59 | 11.320 |
| Valid N (listwise) | 200 | | | | |

**Descriptive Statistics[a]**

| | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| AGE | 140 | 29 | 78 | 51.62 | 11.873 |
| Valid N (listwise) | 140 | | | | |

a. SEX = M

The minimum age of male presented with MI was 28.

The maximum age of male pfesented with MI was 78.

The minimum age of female presented withMI was46.

The maximum age of female presented with MI was78.

**Descriptive Statistics[a]**

| | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| AGE | 60 | 46 | 78 | 58.17 | 8.341 |
| Valid N (listwise) | 60 | | | | |

a. SEX = F

Amongst both male and female patient male presented at earlier age than female'.

SEX AND HEART BLOCK:

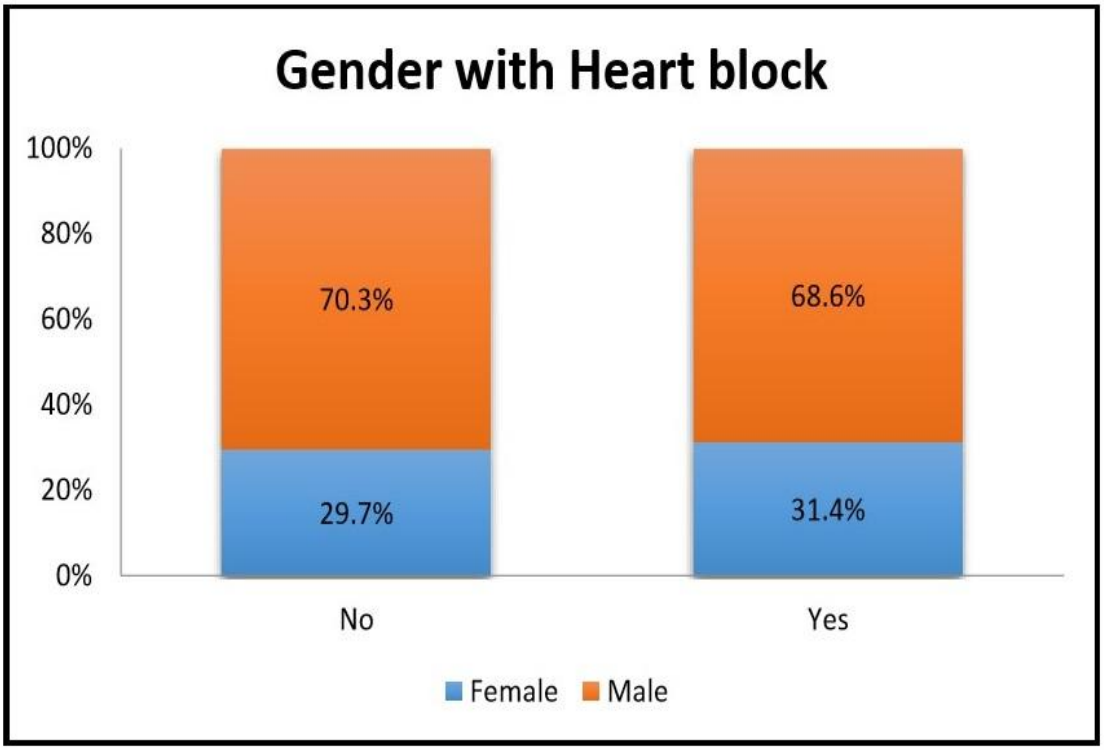
Among 49 female patients,11 presented with heart block and myocardial infarction.,

Among  116 male patient with MI 24 presented with heaqrt block.

| SEX * HEART BLOCK | | | | | |
|---|---|---|---|---|---|
| | | | | | |
| **Crosstab** | | | | | |
| | | | HEART BLOCK | | Total |
| | | | No | Yes | |
| SEX | F | Count | 49 | 11 | 60 |
| | | % within HEART BLOCK | 29.7% | 31.4% | 30.0% |
| | M | Count | 116 | 24 | 140 |
| | | % within HEART BLOCK | 70.3% | 68.6% | 70.0% |
| Total | | Count | 165 | 35 | 200 |
| | | % within HEART BLOCK | 100.0% | 100.0% | 100.0% |

Male patients comprised of68.6% where as female comprises of 31,4 %.as the incidence of MI in this study is more in male the heart block is also more iin male.

| | | No | Yes |
|---|---|---|---|
| | Female | 29.7% | 31.4% |
| | Male | 70.3% | 68.6% |



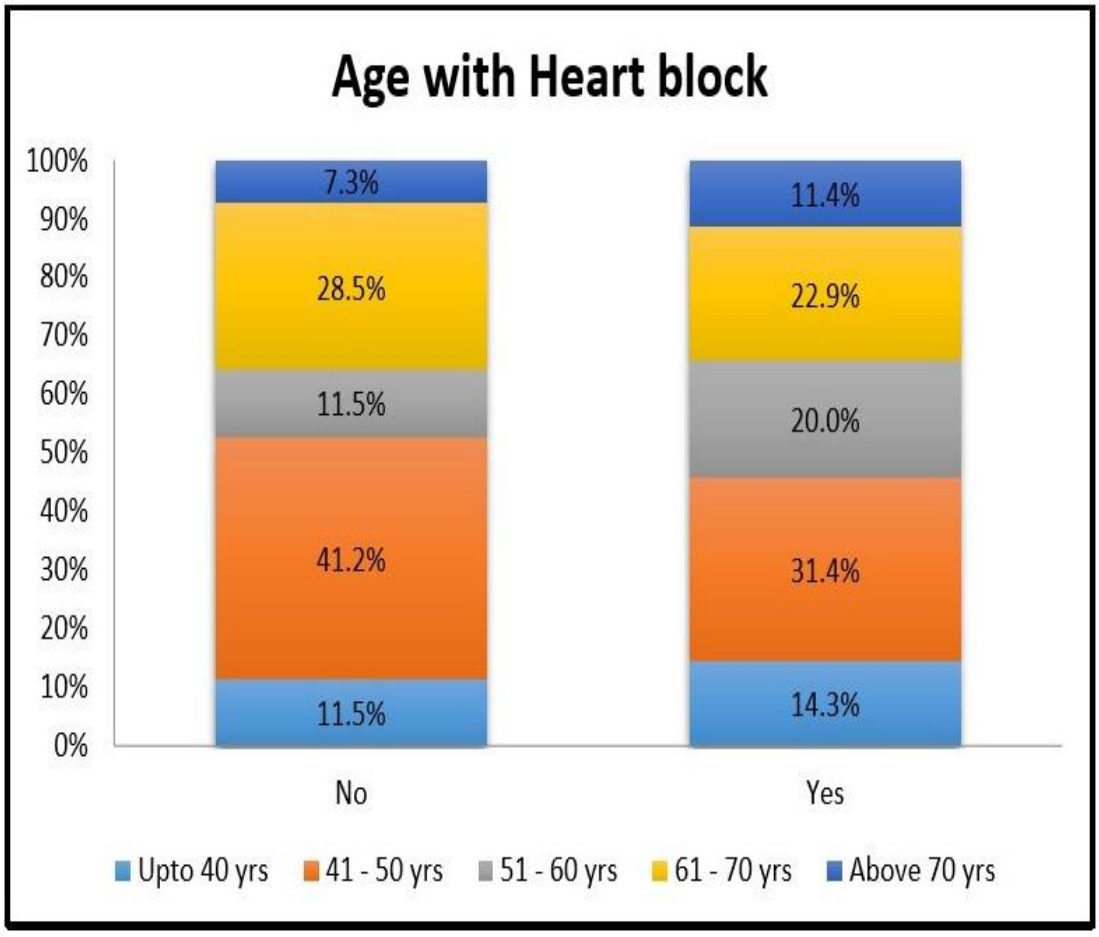
AGE AND HEART BLOCK:

## AGERANGE * HEART BLOCK

**Crosstab**

| | | | HEART BLOCK | | |
|---|---|---|---|---|---|
| | | | No | Yes | Total |
| AGERANGE | Upto 40 yrs | Count | 19 | 5 | 24 |
| | | % within HEART BLOCK | 11.5% | 14.3% | 12.0% |
| | 41 - 50 yrs | Count | 68 | 11 | 79 |
| | | % within HEART BLOCK | 41.2% | 31.4% | 39.5% |
| | 51 - 60 yrs | Count | 19 | 7 | 26 |
| | | % within HEART BLOCK | 11.5% | 20.0% | 13.0% |
| | 61 - 70 yrs | Count | 47 | 8 | 55 |
| | | % within HEART BLOCK | 28.5% | 22.9% | 27.5% |
| | Above 70 yrs | Count | 12 | 4 | 16 |
| | | % within HEART BLOCK | 7.3% | 11.4% | 8.0% |
| Total | | Count | 165 | 35 | 200 |
| | | % within HEART BLOCK | 100.0% | 100.0% | 100.0% |

myocardial infarction which is more common in Male as per this study that too in the age group of41 to 50 years ,the presence of heart block is more in age group of41 to 50 years then only61 to 70 years.



DIABETES AND HEART BLOCK:

Among patients with DM comprising of 61 ,heart block is present in 46 people.

Among non diabetics comprising of 139 ,heart block is present only in 20 people

## DIABETES * HEART BLOCK

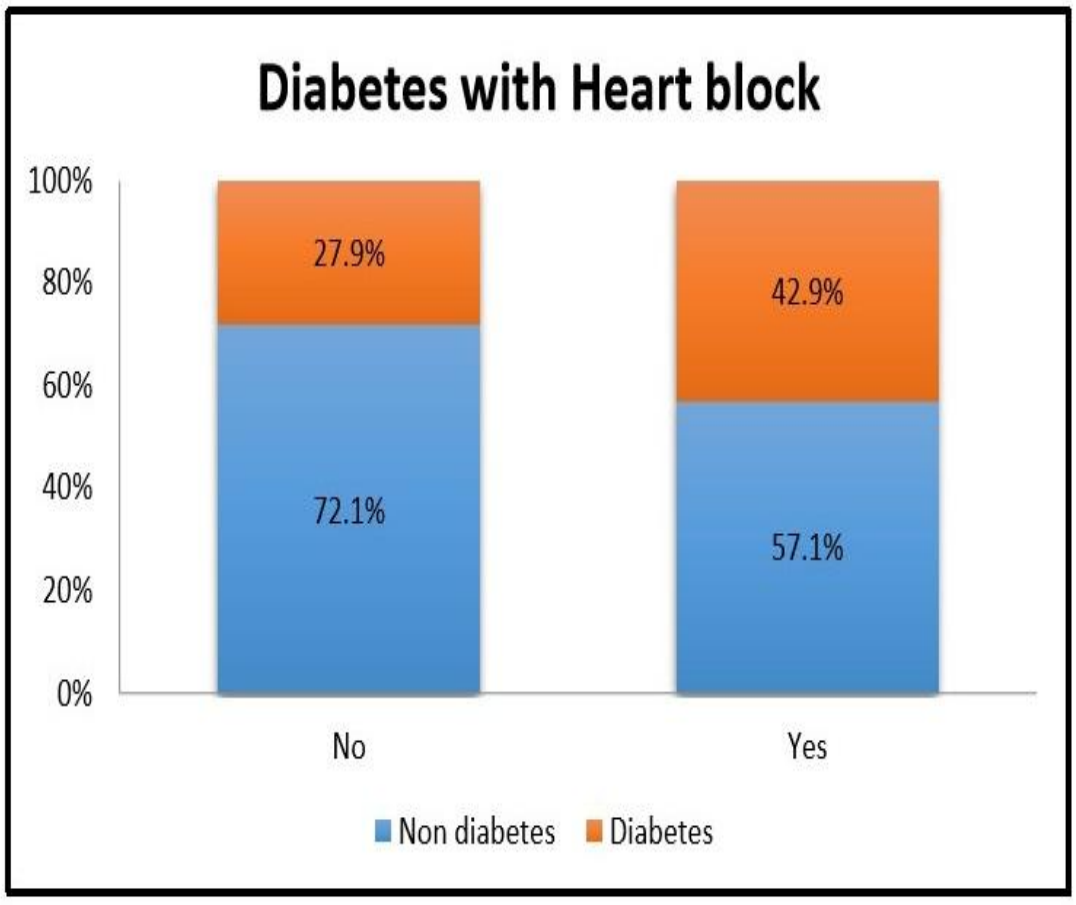
**Crosstab**

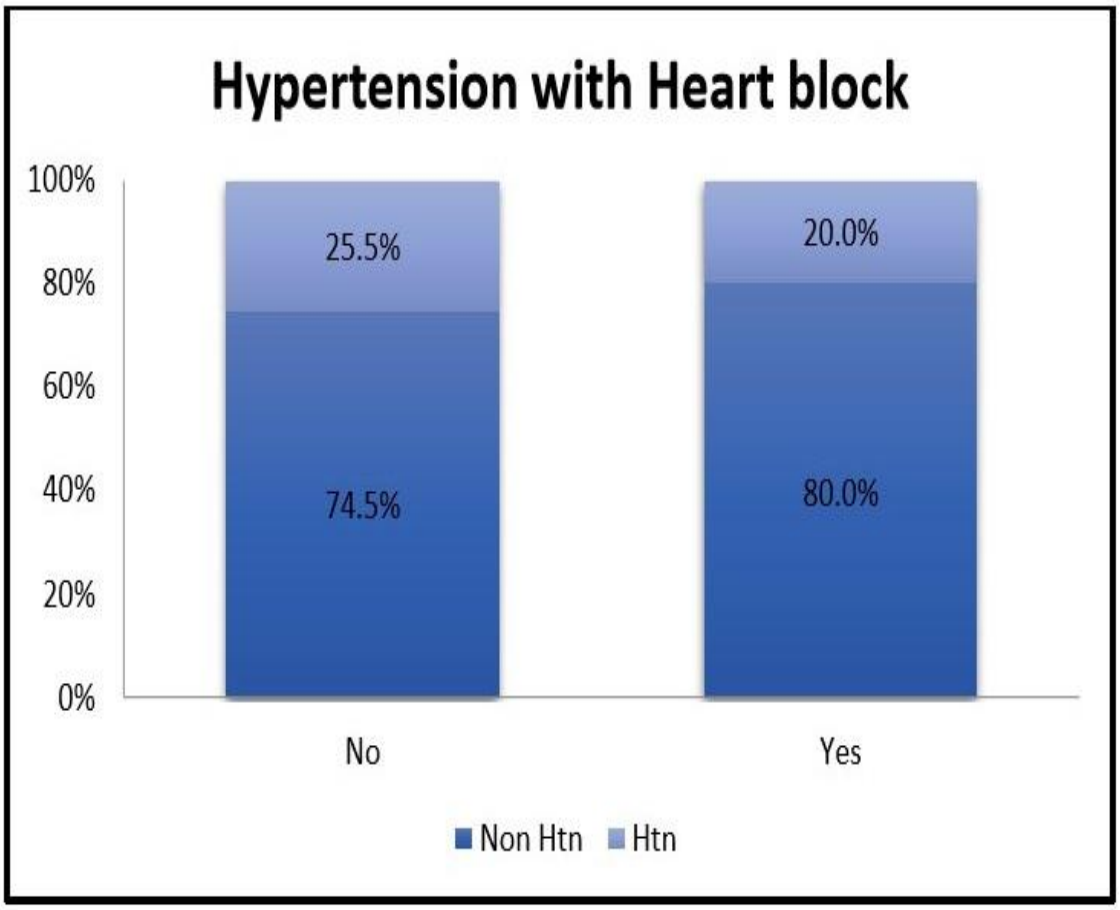
| | | | HEART BLOCK | | |
|---|---|---|---|---|---|
| | | | No | Yes | Total |
| DIABETES | N | Count | 119 | 20 | 139 |
| | | % within HEART BLOCK | 72.1% | 57.1% | 69.5% |
| | Y | Count | 46 | 15 | 61 |
| | | % within HEART BLOCK | 27.9% | 42.9% | 30.5% |
| Total | | Count | 165 | 35 | 200 |
| | | % within HEART BLOCK | 100.0% | 100.0% | 100.0% |

Among non diabetics 57.1% had heart block.

Among diabetics 42.9 had heart block.

This is of greater significance that heart block is more prevalent in diabetic patients.






HYPERTENSION AND HEART BLOCK:

Among 200 patients ,49 patients were found to be hypertensive.in tat 49, only 7 patient had heart block where as remaining 42 patients had no heart block.

The percentage of patients with both heart block and hypertension is 20%.the percentage of patients having heart block and not hypertensive is80%.

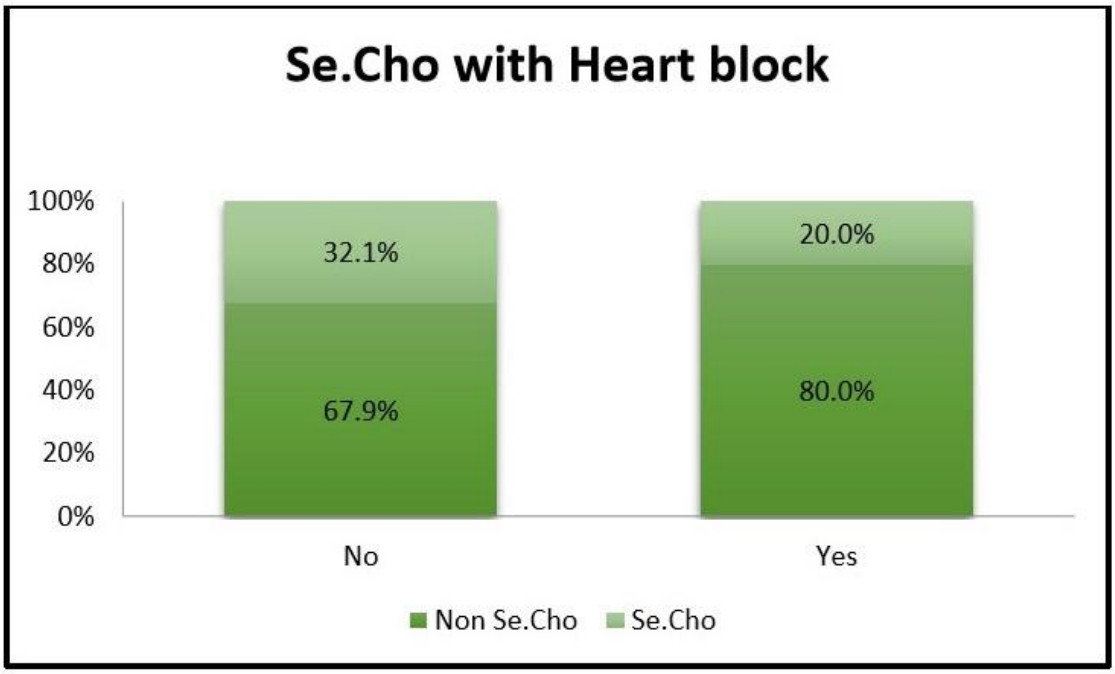
so,there is no significant association between heart block and hypertension.

| | | | | | | |
|---|---|---|---|---|---|---|
| | **HYPERTENSION * HEART BLOCK** | | | | | |
| | | | | | | |
| | **Crosstab** | | | | | |
| | | | | HEART BLOCK | | Total |
| | | | | No | Yes | |
| | HYPERTENSION | N | Count | 123 | 28 | 151 |
| | | | % within HEART BLOCK | 74.5% | 80.0% | 75.5% |
| | | Y | Count | 42 | 7 | 49 |
| | | | % within HEART BLOCK | 25.5% | 20.0% | 24.5% |
| | Total | | Count | 165 | 35 | 200 |
| | | | % within HEART BLOCK | 100.0% | 100.0% | 100.0% |

SERUM CHOLESTEROL AND HEART BLOCK:

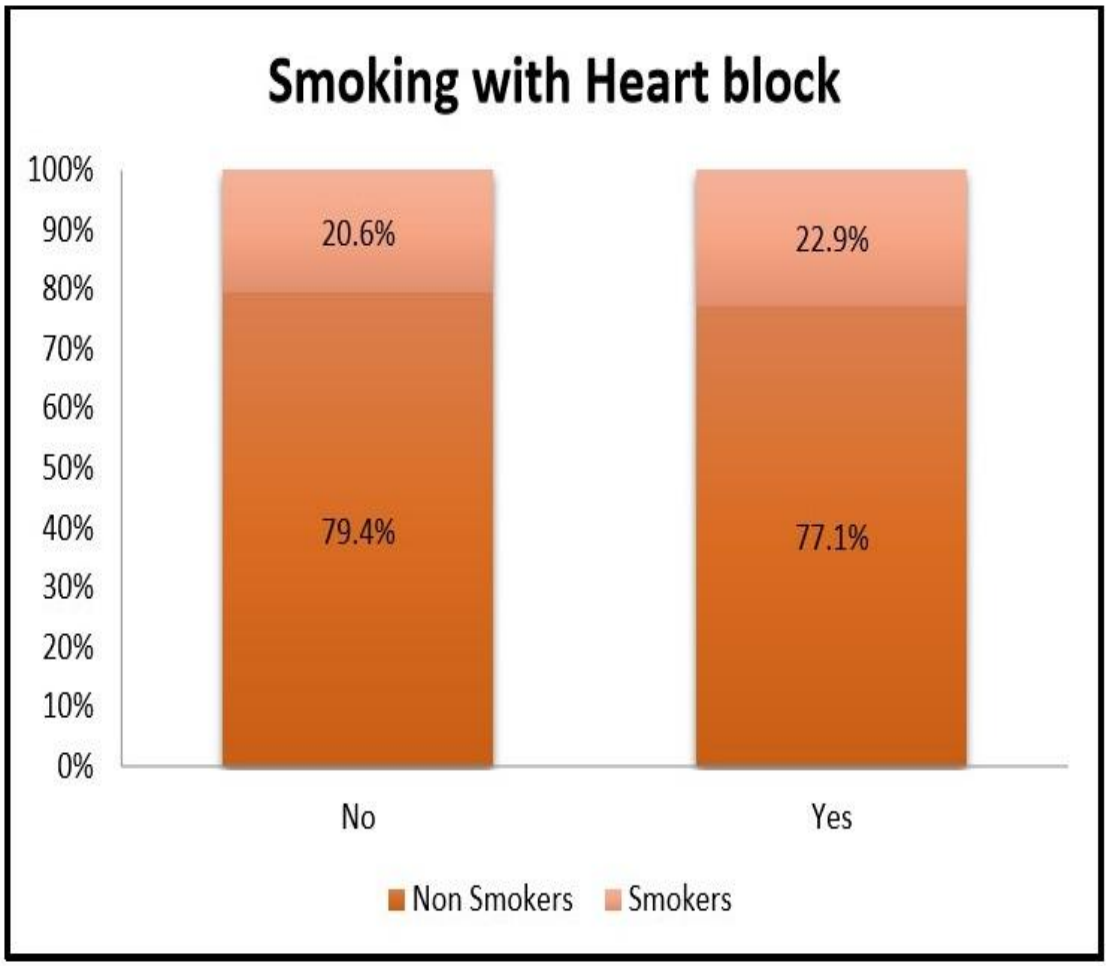
Among 200 patients of acute MI,60 had elevated serum cholesterol.in those 60 hyperlipidemic patients,only 7 presented with heart block and hypertension.53 patients who had hypertension did not have heartblock.the oercentage ofpatients with hypertension and heart block is 20%.this is not significant.

| SE.CHO * HEART BLOCK | | | | | |
|---|---|---|---|---|---|
| | | | | | |
| **Crosstab** | | | | | |
| | | | HEART BLOCK | | Total |
| | | | No | Yes | |
| SE.CHO | N | Count | 112 | 28 | 140 |
| | | % within HEART BLOCK | 67.9% | 80.0% | 70.0% |
| | Y | Count | 53 | 7 | 60 |
| | | % within HEART BLOCK | 32.1% | 20.0% | 30.0% |
| Total | | Count | 165 | 35 | 200 |
| | | % within HEART BLOCK | 100.0% | 100.0% | 100.0% |




SMOKING AND HEARTBLOCK:

Among 200 patients with MI ,42 people were smokers. In those 42 patients 8 had heart block .remaining 34 did not have heart block. The percentage of patients with both heart block and smoking history was 22%.ther was no significant association between smoking and heart block.




Among patients with heart block22.9% were smokers.in patients with out heart block 20.6% were smokers.

Thus smoking factor is more or less equally distributed in patients with and with out heart block.

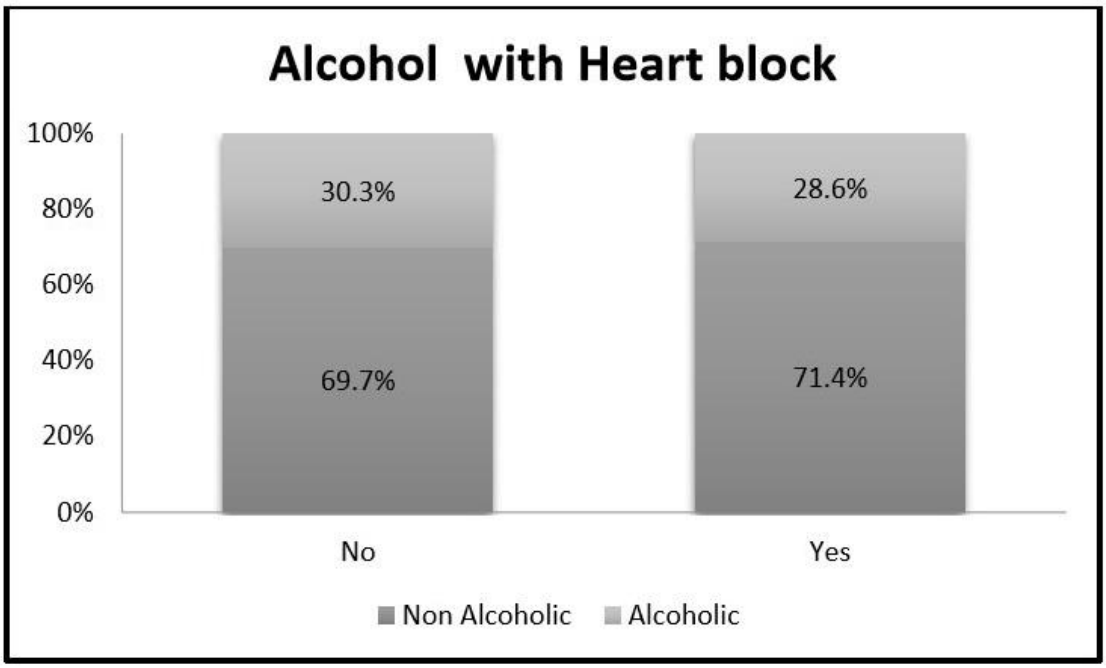
| SMOKING * HEART BLOCK | | | | | |
|---|---|---|---|---|---|
| | | | | | |
| **Crosstab** | | | | | |
| | | | HEART BLOCK | | Total |
| | | | No | Yes | |
| SMOKING | N | Count | 131 | 27 | 158 |
| | | % within HEART BLOCK | 79.4% | 77.1% | 79.0% |
| | Y | Count | 34 | 8 | 42 |
| | | % within HEART BLOCK | 20.6% | 22.9% | 21.0% |
| Total | | Count | 165 | 35 | 200 |
| | | % within HEART BLOCK | 100.0% | 100.0% | 100.0% |

ALCOHOL AND HEART BLOCK:

There were totally 60 patients with history of alcohol consumption.among those 60,

| | | | | | | |
|---|---|---|---|---|---|---|
| | **ALCOHOL * HEART BLOCK** | | | | | |
| | | | | | | |
| | **Crosstab** | | | | | |
| | | | | HEART BLOCK | | Total |
| | | | | No | Yes | |
| | ALCOHOL | N | Count | 115 | 25 | 140 |
| | | | % within HEART BLOCK | 69.7% | 71.4% | 70.0% |
| | | Y | Count | 50 | 10 | 60 |
| | | | % within HEART BLOCK | 30.3% | 28.6% | 30.0% |

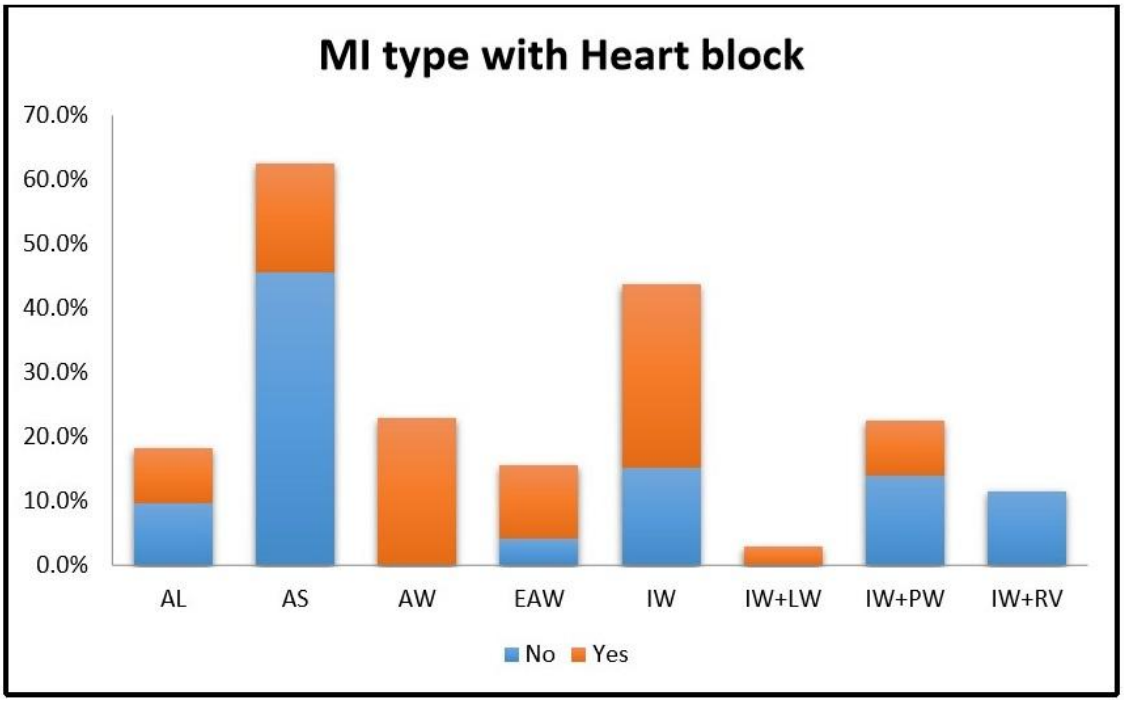
%.

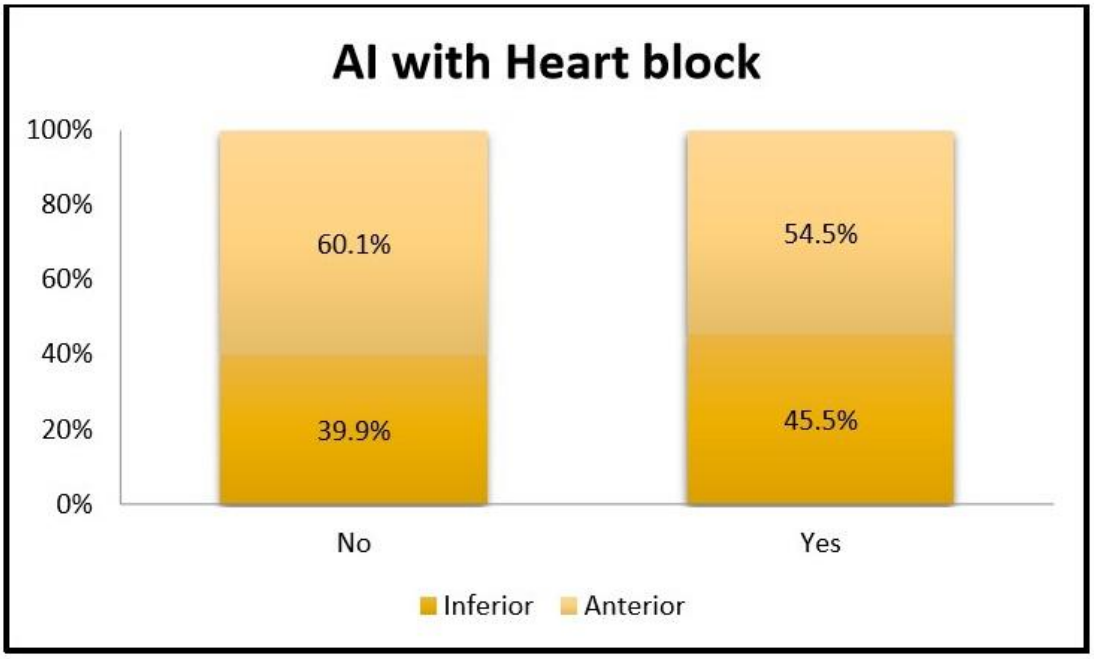



HEART BLOCK AND MI:

The total heart block is 35. Among that 35,22 were AV block and 13 were IVCD. The distribution of heart block among various Type of MI as represented in the bar diagram.

The presence of heart block inanterior wall MI is more than in Inferior wall MI.


MI type with Heart block
70.0%
60.0%
50.0%
40.0%
30.0%
20.0%
10.0%
0.0%
AL
AS
AW
EAW
IW
IW+LW
IW+PW
IW+RV
No
Yes


AI with Heart block
100%
80%
60%
40%
20%
0%
60.1%
39.9%
54.5%
45.5%
No
Yes
Inferior
Anterior

In the study, 6 RBBB was in Inferior wall MI and 3 LBBB in Inferior wall MI

.

LBBB is 3 in Anterior wall MI.

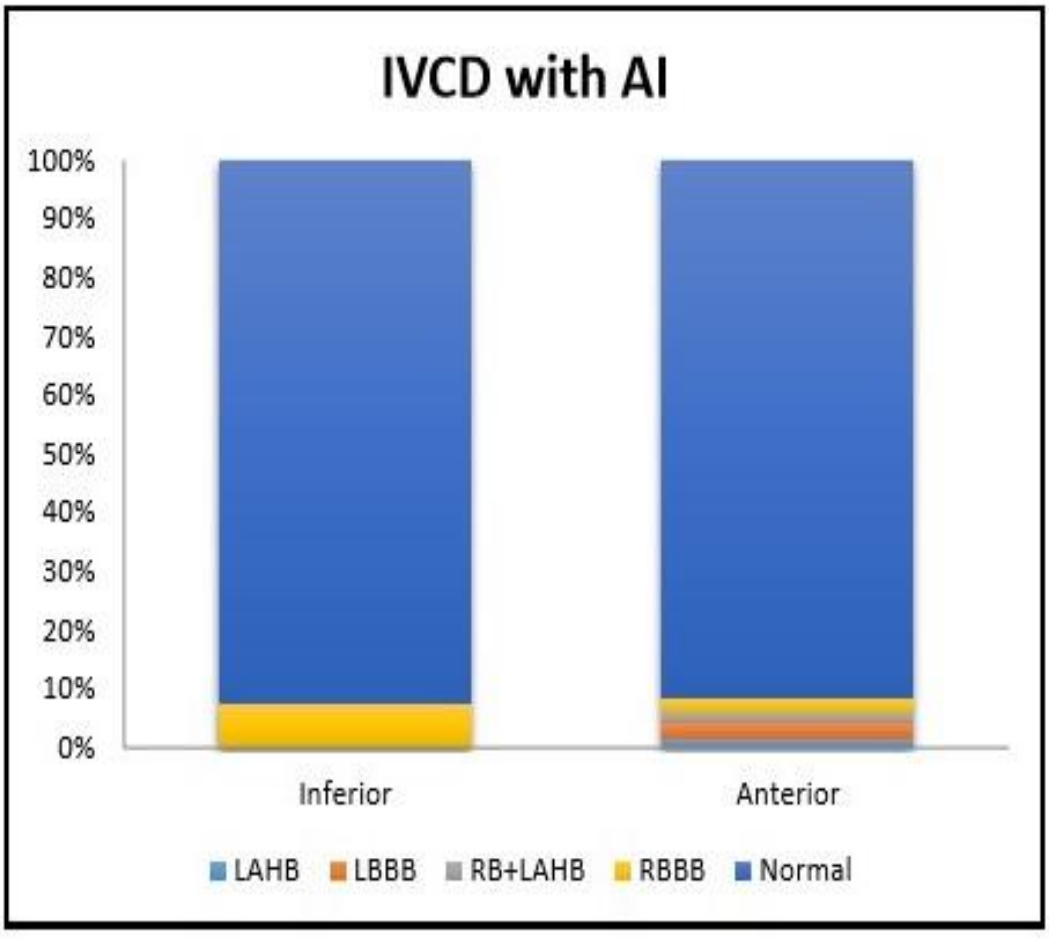
2 RBBB with LAHB is present in antererior wall MI with Inferior wall MI.

But for analysis purpose it is included in Anterior wall MI.and 2 LAHB in anterior wall MI.

So, the RBBB is more in Inferior wall MI than in Anterior wall MI.LBBB is more or less equally distributed.

| | | | | | | |
|---|---|---|---|---|---|---|
| | **IVCD * AI** | | | | | |
| | | | | | | |
| | **Crosstab** | | | | | |
| | | | | AI | | Total |
| | | | | Inferior | Anterior | |
| | IVCD | LAHB | Count | 0 | 2 | 2 |
| | | | % within AI | 0.0% | 1.7% | 1.0% |
| | | LBBB | Count | 0 | 3 | 3 |
| | | | % within AI | 0.0% | 2.5% | 1.5% |
| | | N | Count | 75 | 109 | 184 |
| | | | % within AI | 92.6% | 91.6% | 92.0% |
| | | RB+LAHB | Count | 0 | 2 | 2 |
| | | | % within AI | 0.0% | 1.7% | 1.0% |
| | | RBBB | Count | 6 | 3 | 9 |
| | | | % within AI | 7.4% | 2.5% | 4.5% |
| | Total | | Count | 81 | 119 | 200 |
| | | | % within AI | 100.0% | 100.0% | 100.0% |

The Intraventricular conduction delay is more or less equally present in Anterior and Inferior wall MI.



ATRIOVENTRICULAR BLOCK:

The AV block of I degree ,8 in number is present in Inferior wall MI.

The AV block of II and III degree is present 2 in number,each In ANTERIOR wall MI.

The III degree block ,2 in Inferior and 3 In Anterior wall Myocardial Infarction..

Transition from 1 degree to III degree and II dgree to III degree block is more with Anterior wall MI.

There is no 2 degree AV block in Inferior wall MI.

There were 2 second degree AV block in Anterior wall MI and 2 ,III degree block with out transition from I degree av block.

## AV BLOCK * AI

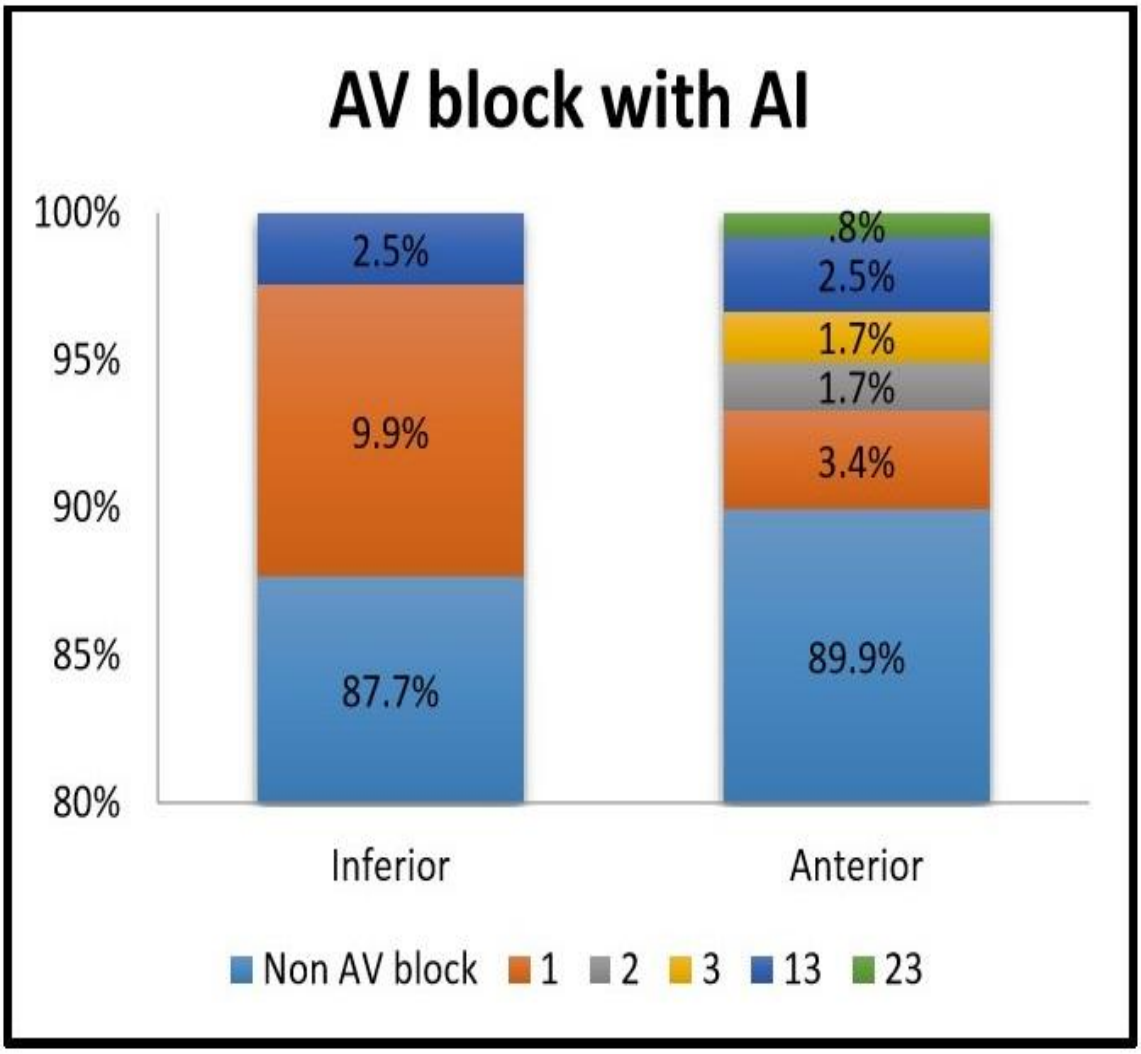
**Crosstab**

| | | | AI | | |
|---|---|---|---|---|---|
| | | | Inferior | Anterior | Total |
| AV BLOCK | No | Count | 71 | 107 | 178 |
| | | % within AI | 87.7% | 89.9% | 89.0% |
| | 1 | Count | 8 | 4 | 12 |
| | | % within AI | 9.9% | 3.4% | 6.0% |
| | 2 | Count | 0 | 2 | 2 |
| | | % within AI | 0.0% | 1.7% | 1.0% |
| | 3 | Count | 0 | 2 | 2 |
| | | % within AI | 0.0% | 1.7% | 1.0% |
| | 13 | Count | 2 | 3 | 5 |
| | | % within AI | 2.5% | 2.5% | 2.5% |
| | 23 | Count | 0 | 1 | 1 |
| | | % within AI | 0.0% | .8% | .5% |
| Total | | Count | 81 | 119 | 200 |
| | | % within AI | 100.0% | 100.0% | 100.0% |

The I degree AV block is more in Inferior wall MI than in Anterior wall MI.

The II degree and III degree AV block is present inAnterior infarction alone.

The III degree AV block with out transition from I degree is in Anterior wall MI.



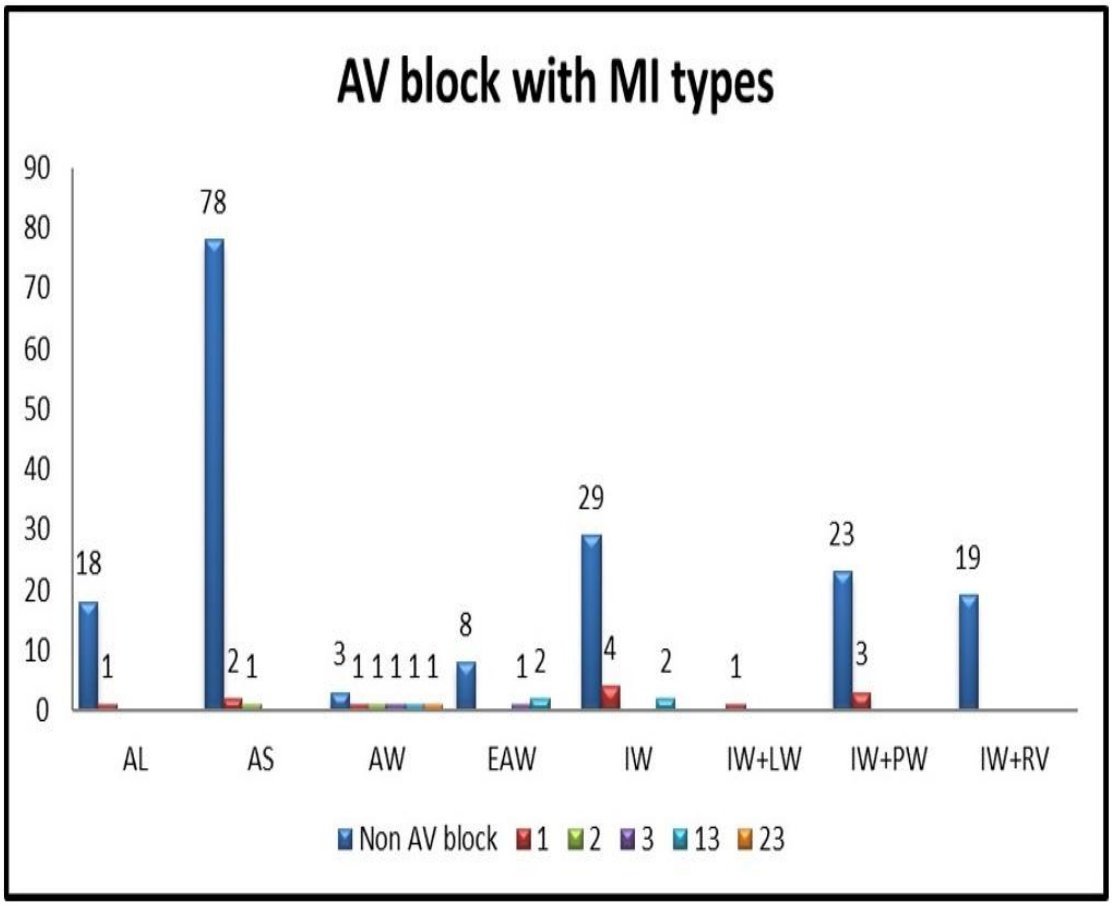

According to this table the distribution of different blocks in various subtype of Anterior and inferior wall MI were presented.

The I degree block is more ininferior wall MI.

Various grades of block is present in Anterior wall MI.

The extensive ante=erior wall MI is associated with III degree AV block.

| MI TYPE * AV BLOCK Crosstabulation | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Count | | | | | | | | |
| | | AV BLOCK | | | | | | Total |
| | | Non AV block | 1 | 2 | 3 | 13 | 23 | |
| MI TYPE | AL | 18 | 1 | | | | | 19 |
| | AS | 78 | 2 | 1 | | | | 81 |
| | AW | 3 | 1 | 1 | 1 | 1 | 1 | 8 |
| | EAW | 8 | | | 1 | 2 | | 11 |
| | IW | 29 | 4 | | | 2 | | 35 |
| | IW+LW | | 1 | | | | | 1 |
| | IW+PW | 23 | 3 | | | | | 26 |
| | IW+RV | 19 | | | | | | 19 |
| Total | | 178 | 12 | 2 | 2 | 5 | 1 | 200 |




TYPE OF BLOCK AND MORTALITY

| Type of block | MORTALITY |
|---|---|
| I BLOCK | 4 |
| II | 1 |
| III | 4 |
| LBBB | 3+2(RB+LAHB) |
| RBBB | 2 |

| TYPE OF MI | MORTALITY |
|---|---|
| AWMI | 14 |
| AW+IW | 0 |
| IWMI | 6 |

In this study death percentage ofdeath in III degree block and transition from I toIII degree block is more.Similarly Death inRBBB +LAHB is more.

Mortality is as such more in anterior wall MI with block.

# DISCUSSION

## DISCUSSION:

The main objective is to study " incidence of heart block,which is17.5% which falls between the literature incidence of12 to25%"(40).

In our study among risk factors " DIABETES is significantly associated with AV block"(32) . Among patients with DM comprising of 61 ,heart block is present in 46 people.

Among non diabetics comprising of 139 ,heart block is present only in 20 people

As per previous studies on risk factors complications and mortality is increased in Myocardial infarction with block.But in our study other risk factors like HT ,smoking,alcohol are not significantly associated.

As per literature no consistent association exist between age and gender inacute MI with new onset block.

Association with myocardial infarction which is more common in Male as per this study that too in the age group of41 to 50 years ,the presence of heart block is more in age group of41 to 50 years then only61 to 70 years,but it is not significant.

Association of various pattern of blocks in MI is as follows

"Sinus Bradycardia is commonly present in IWMI

RBBB and LBBB are of more or less equal incidence in Acute MI(6.7%LBBB VS6.2%RBBB)"(43), but on the whole RBBB more common in IWMI.

" 1 degree block is common in occlusion of artery predominantly supplying AV node that too when proximally occluded"(41).

"Narrow high grade block is 90% present in IWMI and is less mortality than with AWMI"(42).

"Increased mortality in high grade and RBBB with LAHB in AWMI"(44,45).

The presence of heart block inanterior wall MI is more than in Inferior wall MI.

In the study, 6 RBBB was in Inferior wall MI and 3 LBBB in Inferior wall MI

.LBBB is 3 in Anterior wall MI.

2 RBBB with LAHB is present in antererior wall MI with Inferior wall MI.

. So, the RBBB is more in Inferior wall MI than in Anterior wall MI.LBBB is more or less equally distributed.

In this study death percentage ofdeath in III degree block and transition from I toIII degree block is more.Similarly Death inRBBB +LAHB is more.

Mortality is as such more in anterior wall MI with block.

According to literature "mortality is more inRBBB+LAHB due to decreased ejection fraction."(43)

"Low mortality in high grade block with Inferior MI due to Transient nature when compared with Anterior wall MI."(44)

"Mortality more in RBBB+LAHB and new LBBB IN MI".(45)

# CONCLUSION

- The Incidence of conduction block(AV block and Intraventricular conduction delay) is 17.5%.
- There is significant association of Heart Block with diabetes.
- The percentage of Heart block is more in Age between 51-60 years.
- RBBB and I degree Heart block is common in Inferior wall MI.
- RBBB with LAHB implies worse prognosis
- Increased death in Acute MI with high grade blocks.
- Transition from I degree to III degree is Transient in IWMI .

# BIBLIOGRAPHY

# BIBLIOGRAPHY

1. Auffret V, Loirat A, Leurent G, et al. High-degree atrioventricular block complicating ST segment elevation myocardial infarction in the contemporary era. Heart 2016; 102:40.

2. Harpaz D, Behar S, Gottlieb S, et al. Complete atrioventricular block complicating acute myocardial infarction in the thrombolytic era. SPRINT Study Group and the Israeli Thrombolytic Survey Group. Secondary Prevention Reinfarction Israeli Nifedipine Trial. J Am Coll Cardiol 1999; 34:1721.

3. GISSI-2: a factorial randomised trial of alteplase versus streptokinase and heparin versus no heparin among 12,490 patients with acute myocardial infarction. GruppoItaliano per lo Studio dellaSopravvivenzanell'InfartoMiocardico. Lancet 1990; 336:65.

4. Grines CL, Browne KF, Marco J, et al. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction. The Primary Angioplasty in Myocardial Infarction Study Group. N Engl J Med 1993; 328:673.

5. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. The GUSTO investigators. N Engl J Med 1993; 329:673.

6. Archbold RA, Sayer JW, Ray S, et al. Frequency and prognostic implications of conduction defects in acute myocardial infarction since the introduction of thrombolytic therapy. Eur Heart J 1998; 19:893.

7. Hindman MC, Wagner GS, JaRo M, et al. The clinical significance of bundle branch block complicating acute myocardial infarction. 1. Clinical characteristics, hospital mortality, and one-year follow-up. Circulation 1978; 58:679.

8. Agur AMR, Dalley AF, Moore KL. The thorax. *Clinically Oriented Anatomy*. 5th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 2005.

9. Kiani J, Agarwal SK, Kamireddy S, Adelstein E, Saba S. Relationship of electro-mechanical remodeling to survival rates after cardiac resynchronization therapy. *Tex Heart Inst J*. 2013.40(3):268-73.

10.Schwartz PJ, Zipes DP. Autonomic modulation of cardiac arrhythmias. Zipes DP, Jalife J, eds. *Cardiac Electrophysiology: From Cell to Bedside*. 3rd ed. Philadelphia, Pa: WB Saunders; 1999. 300-14.

11.Libby P, Bonow RO, Mann DL, Zipes DP, eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*. 8th ed. Philadelphia, Pa: Saunders Elsevier; 2008. Vol 1

12.Ellenbogen KA, Wood MA, eds. *Cardiac Pacing and ICDs*. 5th ed. Hoboken, NJ: Blackwell Publishing; 2008.

13.Wu J, Zipes DP. Mechanisms underlying atrioventricular nodal conduction and the reentrant circuit of atrioventricular nodal re-entrant tachycardia using optical mapping. *J CardiovascElectrophysiol*. 2002 Aug. 13(8):831-4.

14.. Schwartz PJ, Zipes DP: Autonomic modulation of cardiac arrhythmias. In Zipes DP, Jalife J (eds): Cardiac Electrophysiology: From Cell to Bedside. 3rd ed. Philadelphia, WB Saunders, 1999, pp 300-314

15.Barbuti A, Terragni B, Brioschi C, et al: Localization of f-channels to caveolae mediates specific β2-adrenergic receptor modulation of rate in sinoatrial myocytes. J Mol Cell Cardiol 42:71, 2007.

16.Hucker WJ, Nikolski VP, Efimov IR: Autonomic control and innervation of the atrioventricular junctional pacemaker. Heart Rhythm 4:1326, 2007

17.Jacques D, Abdel-Samad D: Neuropeptide Y (NPY) and NPY receptors in the cardiovascular system: Implication in the regulation of intracellular calcium. Can J PhysiolPharmacol 85:43, 2007.

18.Reuter H, Henderson SA, Han T, et al: The Na+-Ca2+ exchanger is essential for the action of cardiac glycosides. Circ Res 90:305, 2002.

19.Baruscotti M, Bucchi A, DiFrancesco D: Physiology and pharmacology of the cardiac pacemaker ("funny") current. PharmacolTher 107:59, 2005

20.Song Y, Shryock JC, Belardinelli L: An increase of late sodium current induces delayed afterdepolarizations and sustained triggered activity in atrial myocytes. Am J Physiol Heart CircPhysiol 294:H2031, 2008.

21.. Lakatta EG, Maltsev VA, Vinogradova TM: A coupled SYSTEM of intracellular Ca2+ clocks and surface membrane voltage clocks controls the timekeeping mechanism of the heart's pacemaker. Circ Res 106:659, 2010.

22.Horowitz LN, Alexander JA, Edmunds LH Jr. Postoperative right bundle branch block: identification of three levels of block. Circulation 1980; 62:319.

23.Josephson, ME. Clinical Cardiac Electrophysiology: Techniques and Interpretations, 2nd, Lea &Febiger, Philadelphia 1993.

24.Surawicz B, Childers R, Deal BJ, et al. AHA/ACCF/HRS recommendations for the standardization and interpretation of the

electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am CollCardiol 2009; 53:976.

25.LENEGRE J. ETIOLOGY AND PATHOLOGY OF BILATERAL BUNDLE BRANCH BLOCK IN RELATION TO COMPLETE HEART BLOCK. ProgCardiovasc Dis 1964; 6:409.

26.LEV M. ANATOMIC BASIS FOR ATRIOVENTRICULAR BLOCK. Am J Med 1964; 37:742.

27.Mizusawa Y, Wilde AAM: Brugada syndrome. Circ Arrhythm Electrophysiol 5:606, 2012.

28.Krishnan SC, Josephson ME. ST segment elevation induced by class IC antiarrhythmic agents: underlying electrophysiologic mechanisms and insights into drug-induced proarrhythmia. J CardiovascElectrophysiol 1998; 9:1167.

29.Brugada R, Brugada J, Antzelevitch C, et al. Sodium channel blockers identify risk for sudden death in patients with ST-segment elevation and right bundle branch block but structurally normal hearts. Circulation 2000; 101:510.

30.Zhang ZM, Rautaharju PM, Soliman EZ, et al. Mortality risk associated with bundle branch blocks and related repolarization abnormalities (from the Women's Health Initiative [WHI]). Am J Cardiol 2012; 110:1489.

31.Fahy GJ, Pinski SL, Miller DP, et al. Natural history of isolated bundle branch block. Am J Cardiol 1996; 77:1185.

32.Ozeke O, Aras D, Deveci B, et al. Comparison of presence and extent of coronary narrowing in patients with left bundle branch block without diabetes mellitus to patients with and without left bundle branch block but with diabetes mellitus. Am J Cardiol 2006; 97:857.

33.Dabrowska B, Ruka M, Walczak E. The electrocardiographic diagnosis of left septal fascicular block. Eur J Cardiol 1978; 6:347.

34.Ohmae M, Rabkin SW. Hyperkalemia-induced bundle branch block and complete heart block. ClinCardiol 1981; 4:43.

35.Bashour T, Hsu I, Gorfinkel HJ, et al. Atrioventricular and intraventricular conduction in hyperkalemia. Am J Cardiol 1975; 35:199.

36.Siegman-Igra Y, Yahini JH, Goldbourt U, Neufeld HN. Intraventricular conduction disturbances: a review of prevalence,

etiology, and progression for ten years within a stable population of Israeli adult males. Am Heart J 1978; 96:669.

37.OSTRANDER LD Jr, BRANDT RL, KJELSBERG MO, EPSTEIN FH. ELECTROCARDIOGRAPHIC FINDINGS AMONG THE ADULT POPULATION OF A TOTAL NATURAL COMMUNITY, TECUMSEH, MICHIGAN. Circulation 1965; 31:888.

38.Auricchio A, Fantoni C, Regoli F, et al. Characterization of left ventricular activation in patients with heart failure and left bundle-branch block. Circulation 2004; 109:1133.

39.Das MK, Cheriparambil K, Bedi A, et al. Prolonged QRS duration (QRS >/=170 ms) and left axis deviation in the presence of left bundle branch block: A marker of poor left ventricular systolic function? Am Heart J 2001; 142:756.

40. James TN: Arrhythmias and conduction disturbances in acute myocardial infarction. AM Heart J 1962;64:416-426

41. Braat SH, de Zwaan C, Brugada P, et al: Right ventricular involvement with acute inferior wall myocardial infarction identifies high risk of developing atrioventricular nodal conduction disturbances. Am Heart J 1984;107:1183-1187.

42.Hindman MC, Wagner GS, JaRo M,et al: The clinical significance of bundle branch block complicating acute myocardial

infarction, 1: clinical characteristics, hospital mortality, and one-year follow up. Circulation 1978;58:679-688.

43.Meine TJ, Al-Khatib SM, Alexander JH, et al: Incidence, predictors, and out-comes of high-degree atrioventricular block complicationg acute myocardial infarction treated with thrombolytic therapy. Am Heart J 2005;149:670-674.

44. Go AS, Barron HV, Rundle AC, et al: Bundle-branch block and in-hospital mortality in acute myocardial infarction. National Registry of Myocardial Infarction 2 Investigators. Ann Intern Med 1998;129:690-697.

45.Piotrowicz K, Zareba W, McNitt S, et al: Repolarisation duration in patients with conduction distrubances ater mmyocardial inarction. Am J Cardiol 2007;99:163-168.

# ANNEXURES

**PROFORMA:**

Name: Age: Sex:

Educational status: Occupation:

Address: Ht in cms:

Wt in kgs: BMI [kg/m2].

Clinical presentation on admission:

Time of onset of symptoms:

Chest pain Typical/Atypical

Syncope.

Palpitation.

Shortness of breath.

Cerebral symptoms.

**PAST HISTORY:**

Diabetes:

Hypertension:

Myocardial infarction:

Angina pectoris:

Cerebrovascular diseases:

**PERSONAL HISTORY:**

Diet: vegetarian/non vegetarian:

Smoking:

Alcohol:

Tobacco chewing:

Sedentary habits:

**Menstrual and Obstetric History**:

**FAMILY HISTORY:**

Hypertension:

Diabetes:

Ischemic heart disease:

Hypercholesteremia:

**GENERAL EXAMINATION**:

Anemia: Jaundice: Cyanosis:

Clubbing: Edema:

**EXAMINATION OF CARDIOVASCULAR SYSTEM:**

Pulse Rate

Rhythm.

Character.

Volume.

Peripheral pulses.

BP:

JVP:

APICAL IMPULSE:

AUSCULTATION:

Heart sounds:

Murmur:

**EXAMINATION OF RESPIRATORY SYSTEM:**

Air entry:

Adventitious sounds:

**EXAMINATION OF ABDOMEN:**

Free fluid

Organomegaly

**EXAMINATION OF CENTRAL NERVOUS SYSTEM:**

Level of consciousness:

Any focal neurological deficit:

**INVESTIGATIONS**

1. Urine R/E. Albumin Sugar Deposits

2.BLOOD TC DC ESR HB Sugar Urea

3. ECG ANALYSIS.

1. Site of infarction.
2. Type of conduction disturbance present.
3. Time of conduction disturbance with relation to onsetof symptoms.
4. Duration of conduction disturbance.

5.Sequential Changes in type of block.

6. Specific treatment given.

| S:NO | SEX | AGE | DIABETES | HYPERTENSION | SE.CHO | SMOKING | ALCOHOL | MI TYPE | IVCD | AV BLOCK | SK LYSED | MORTALITY |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | M | 51 | y | N | N | N | N | EAW | N | 3 | Y | Y |
| 2 | M | 76 | Y | N | N | Y | N | IW+PW | N | 1 | y | N |
| 3 | M | 71 | N | N | N | N | N | AL | RBBB | N | y | N |
| 4 | F | 47 | Y | N | N | N | N | AS | N | 1 | y | N |
| 5 | F | 68 | N | Y | Y | N | N | AL | N | N | Y | N |
| 6 | F | 68 | Y | Y | Y | N | N | AS | N | N | Y | N |
| 7 | M | 61 | N | N | N | N | Y | AS | N | N | Y | N |
| 8 | F | 63 | N | N | N | N | N | IW | N | N | Y | N |
| 9 | M | 53 | Y | N | N | N | Y | IW | N | 1 | Y | N |
| 10 | M | 39 | N | N | Y | N | N | AS | N | N | Y | N |
| 11 | M | 56 | Y | Y | N | Y | N | IW | N | N | N | N |
| 12 | M | 46 | N | N | Y | N | Y | AS | N | N | Y | N |
| 13 | M | 68 | Y | Y | N | Y | Y | AS | N | N | Y | N |
| 14 | F | 49 | N | N | N | N | N | IW+PW | N | 3 | Y | N |
| 15 | M | 46 | N | N | N | N | N | IW | N | N | Y | N |
| 16 | M | 49 | N | Y | N | N | N | AL | N | N | Y | N |
| 17 | F | 78 | N | N | N | N | N | IW+PW | N | N | N | N |
| 18 | M | 58 | Y | N | Y | Y | N | IW+RV | N | N | Y | Y |
| 19 | M | 44 | N | Y | N | N | N | AS | N | N | N | N |
| 20 | F | 49 | N | N | N | N | N | IW+RV | N | N | Y | N |
| 21 | F | 49 | N | N | N | N | N | AS |  | 1 | Y | N |
| 22 | M | 48 | N | N | N | N | N | IW+RV | N | N | Y | N |
| 23 | M | 39 | Y | Y | N | Y | Y | IW | N | N | Y | N |
| 24 | M | 41 | N | N | Y | N | Y | IW+RV | N | N | Y | N |
| 25 | M | 72 | N | Y | N | Y | N | IW+RV | N | N | N | N |
| 26 | F | 68 | N | Y | N | N | N | IW | N | N | Y | N |
| 27 | F | 59 | N | N | N | N | N | AS | N | N | Y | N |
| 28 | M | 44 | N | N | N | Y | Y | IW | N | 1,3 | Y | N |
| 29 | M | 43 | N | N | Y | N | N | AS | N | N | N | N |
| 30 | M | 33 | N | Y | N | N | N | AL | N | N | Y | N |
| 31 | M | 62 | N | Y | N | Y | Y | AW | RBBB | N | Y | N |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 32 | M | 36 | N | N | Y | Y | Y | AS | LAHB | N | Y | N |
| 33 | M | 72 | N | Y | Y | N | N | EAW | N | 1,3 | Y | Y |
| 34 | M | 71 | N | N | N | N | N | IW | N | N | N | N |
| 35 | F | 54 | N | N | N | N | N | AS | N | N | N | Y |
| 36 | F | 46 | N | N | N | N | N | AS | N | N | Y | N |
| 37 | F | 70 | N | N | N | N | N | IW+PW | N | N | N | N |
| 38 | M | 48 | Y | Y | Y | Y | Y | AL | N | N | Y | N |
| 39 | M | 49 | N | N | Y | N | Y | AS | N | N | Y | N |
| 40 | M | 48 | N | N | N | N | N | AS | N | N | N | N |
| 42 | F | 54 | N | N | N | N | N | IW | N | 3 | Y | N |
| 43 | M | 49 | N | N | N | Y | N | IW | N | N | Y | N |
| 44 | F | 62 | Y | N | N | N | N | EAW | N | N | Y | N |
| 45 | F | 69 | Y | N | Y | Y | Y | IW+PW | N | N | N | N |
| 46 | M | 49 | N | N | Y | N | Y | IW+PW | N | N | Y | N |
| 47 | M | 37 | N | N | N | Y | N | IW+RV | N | N | Y | N |
| 48 | F | 63 | Y | Y | N | N | Y | AS | RBBB | N | N | Y |
| 49 | M | 72 | Y | Y | N | N | Y | IW+PW | N | N | Y | N |
| 50 | F | 55 | N | N | N | N | N | IW | N | N | Y | N |
| 51 | M | 38 | Y | N | N | N | N | AS | N | N | Y | N |
| 52 | M | 70 | N | N | N | N | N | AS | N | N | N | N |
| 53 | M | 63 | N | Y | N | N | N | AS | N | N | Y | N |
| 54 | M | 73 | N | N | Y | Y | N | EAW | N | N | Y | Y |
| 55 | F | 67 | N | N | N | N | N | IW | RBBB | N | N | N |
| 56 | M | 47 | Y | N | Y | N | Y | AL | N | N | Y | N |
| 57 | M | 47 | N | N | N | N | N | EAW | N | N | Y | N |
| 58 | F | 61 | Y | N | N | N | N | AL | N | N | Y | N |
| 59 | M | 37 | N | N | N | Y | N | AS | N | N | Y | N |
| 60 | F | 47 | Y | N | N | N | Y | AS | N | N | Y | N |
| 61 | F | 51 | N | N | N | N | Y | AS | N | N | N | N |
| 62 | M | 44 | Y | N | N | N | N | AS | N | N | Y | N |
| 63 | M | 46 | Y | N | N | N | Y | EAW | RBBB+LAHB | N | Y | y |
| 64 | M | 44 | Y | N | N | Y | N | AS | N | N | Y | N |

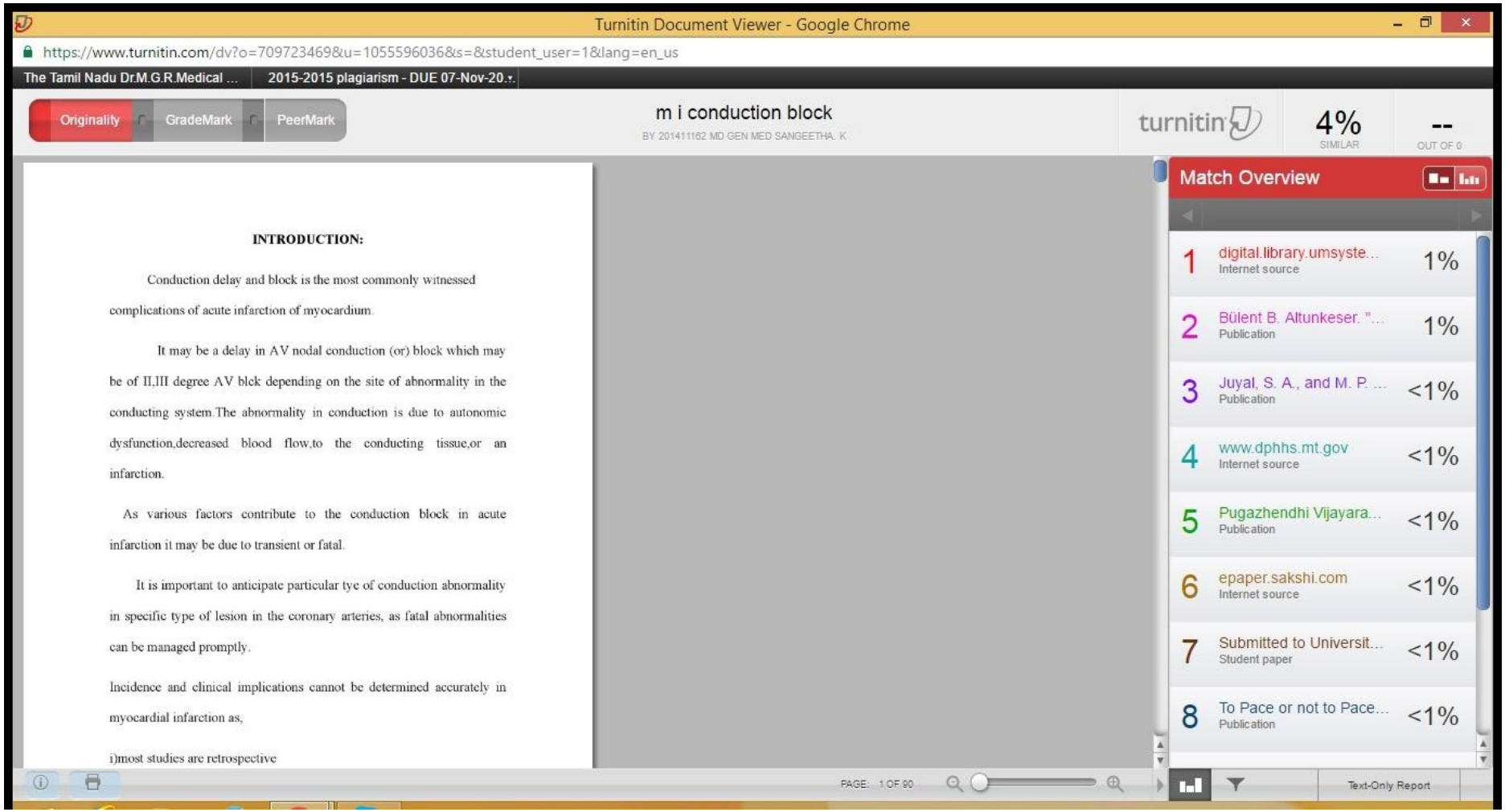
| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 65 | F | 49 | N | Y | Y | N | N | EAW | N | N | Y | N |
| 66 | F | 67 | N | Y | N | N | N | AS | N | N | N | N |
| 67 | M | 38 | N | N | N | N | N | AS | N | N | Y | N |
| 68 | M | 49 | N | N | N | N | N | AS | N | N | Y | N |
| 69 | M | 71 | N | N | Y | N | N | IW+PW | N | N | Y | N |
| 70 | M | 47 | | Y | N | N | Y | IW+RV | N | N | Y | N |
| 71 | M | 61 | Y | N | N | Y | N | IW | N | N | Y | N |
| 72 | F | 51 | N | Y | N | N | N | IW+PW | N | 1 | Y | Y |
| 73 | M | 47 | N | Y | N | N | N | IW | N | N | Y | N |
| 74 | M | 42 | N | N | N | N | Y | AS | N | N | Y | N |
| 75 | M | 44 | Y | N | N | N | N | AS | N | N | Y | N |
| 76 | M | 49 | Y | N | Y | N | Y | IW+RV | N | N | Y | N |
| 77 | M | 42 | Y | N | N | N | N | AS | N | N | Y | N |
| 78 | M | 33 | Y | N | N | N | N | EAW | RBBB+LAHB | N | Y | Y |
| 79 | M | 72 | N | N | Y | Y | N | IW+PW | N | N | Y | N |
| 80 | M | 47 | N | Y | N | N | Y | IW+RV | N | N | Y | N |
| 81 | M | 46 | N | Y | N | N | Y | AS | N | N | Y | N |
| 82 | F | 64 | N | N | N | N | N | AS | N | N | Y | N |
| 83 | M | 43 | N | N | Y | N | N | AS | N | N | Y | N |
| 84 | M | 68 | N | N | Y | N | Y | IW+PW | N | N | Y | N |
| 85 | M | 62 | N | N | N | N | N | IW | N | N | Y | N |
| 86 | M | 44 | Y | N | N | Y | Y | AL | N | N | Y | N |
| 87 | M | 68 | N | N | N | N | N | AL | N | N | N | Y |
| 88 | M | 67 | Y | N | N | N | N | IW | N | 2 | N | Y |
| 89 | M | 39 | N | N | Y | N | N | IW+RV | N | 1 | Y | Y |
| 90 | F | 48 | N | Y | N | N | N | IW+PW | N | N | Y | N |
| 91 | F | 67 | N | N | Y | N | N | IW+PW | N | N | Y | N |
| 92 | M | 54 | Y | N | N | Y | N | AL | N | N | Y | N |
| 93 | M | 45 | Y | N | Y | N | N | IW | N | N | Y | N |
| 94 | M | 39 | N | N | N | N | Y | IW+PW | N | N | Y | N |
| 95 | F | 48 | N | N | N | N | N | IW | N | N | Y | N |
| 96 | F | 69 | Y | N | N | N | N | AL | N | N | Y | N |

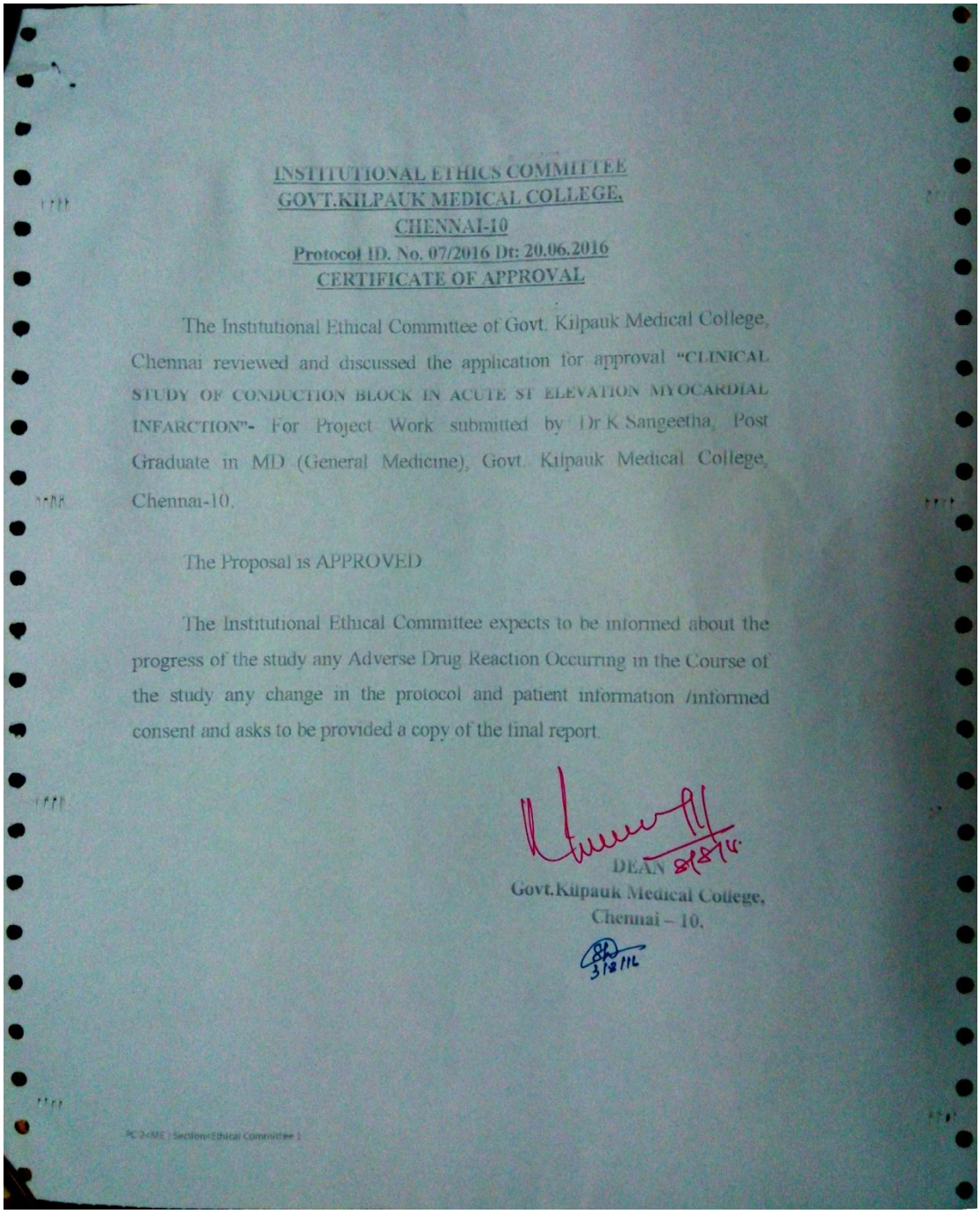
| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 97 | M | 47 | N | N | Y | N | Y | AW | N | 3 | Y | Y |
| 98 | M | 61 | Y | N | N | Y | N | IW+PW | N | N | N | N |
| 99 | M | 44 | N | N | Y | N | Y | IW+RV | N | N | Y | N |
| 100 | M | 49 | N | N | N | N | N | IW | N | N | Y | N |
| 101 | M | 62 | N | N | Y | N | N | AL | N | N | Y | N |
| 102 | M | 64 | N | N | Y | N | N | EAW | N | N | Y | N |
| 103 | M | 49 | N | N | N | Y | Y | IW | N | N | N | N |
| 104 | F | 48 | N | N | N | N | N | AW | LAHB | N | Y | Y |
| 105 | M | 43 | N | N | Y | Y | N | AS | N | N | Y | N |
| 106 | M | 49 | N | N | N | N | N | AS | N | N | N | N |
| 107 | M | 39 | N | S | Y | N | Y | AS | N | N | Y | N |
| 108 | M | 41 | Y | N | N | N | N | IW | N | 1,3 | Y | N |
| 109 | F | 52 | N | Y | N | N | N | IW+RV | N | N | N | N |
| 110 | F | 58 | Y | N | N | N | N | AL | N | N | Y | N |
| 111 | M | 47 | N | Y | N | Y | N | AS | N | N | Y | N |
| 112 | M | 55 | Y | N | Y | Y | Y | AS | N | N | Y | N |
| 113 | M | 47 | | Y | N | N | N | AS | N | N | Y | N |
| 114 | F | 61 | Y | N | Y | N | Y | IW | N | N | N | N |
| 115 | M | 61 | N | N | N | Y | Y | AL | LBBB | N | Y | Y |
| 116 | F | 62 | N | N | Y | N | N | AS | N | N | N | N |
| 117 | F | 49 | N | Y | N | N | N | IW+RV | N | N | Y | N |
| 118 | M | 46 | Y | N | N | N | N | AS | N | N | N | N |
| 119 | M | 38 | N | N | Y | N | Y | AS | N | N | Y | N |
| 120 | M | 46 | N | Y | N | N | N | AS | N | N | Y | N |
| 121 | F | 62 | Y | N | N | N | Y | AS | N | N | Y | N |
| 122 | F | 56 | Y | N | N | N | N | IW | N | N | Y | N |
| 123 | F | 48 | N | Y | N | N | N | IW+PW | N | N | Y | N |
| 124 | M | 50 | N | N | Y | Y | N | AS | N | N | Y | N |
| 125 | M | 38 | N | Y | N | N | N | AS | N | N | N | N |
| 126 | F | 49 | N | N | Y | N | N | AS | N | N | Y | N |
| 127 | M | 68 | N | N | Y | Y | Y | AS | N | N | Y | N |
| 128 | F | 66 | N | Y | N | N | N | AS | N | N | Y | N |
| 129 | M | 39 | N | N | Y | N | N | AS | N | N | Y | N |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 130 | F | 73 | Y | N | Y | N | N | AL | N | 1 | Y | Y |
| 131 | M | 78 | Y | N | N | N | N | IW+PW | N | N | N | N |
| 132 | M | 63 | N | N | N | Y | N | IW+RV | N | N | Y | N |
| 133 | M | 39 | N | N | N | N | Y | AS | N | N | Y | N |
| 134 | M | 42 | N | N | N | N | Y | AS | N | N | Y | N |
| 135 | M | 74 | Y | N | N | N | N | IW+PW | N | N | Y | N |
| 136 | M | 64 | N | N | N | Y | N | IW+RV | N | N | N | N |
| 137 | M | 69 | N | N | N | N | N | IW+PW | N | N | Y | Y |
| 138 | M | 36 | Y | N | Y | N | Y | IW+RV | N | N | Y | N |
| 139 | F | 58 | N | N | N | N | N | AS | 3 | N | Y | N |
| 140 | f | 60 | Y | Y | N | N | N | AS | N | N | Y | N |
| 141 | M | 62 | N | N | N | N | N | AS | N | N | Y | N |
| 142 | F | 56 | Y | Y | Y | N | N | AS | N | N | Y | N |
| 143 | M | 64 | N | N | N | Y | Y | IW | RBBB | N | Y | Y |
| 144 | F | 68 | N | Y | N | N | N | AS | N | N | Y | N |
| 145 | F | 56 | N | N | N | N | N | IW | N | N | N | N |
| 146 | M | 48 | N | N | Y | N | Y | EAW | N | N | Y | N |
| 147 | M | 48 | N | N | Y | N | Y | IW | N | N | Y | N |
| 148 | M | 32 | N | N | N | Y | N | AL | N | N | Y | N |
| 149 | F | 70 | N | N | N | N | N | IW+PW | N | N | Y | N |
| 150 | F | 69 | N | N | N | N | N | AS | N | N | N | N |
| 151 | F | 62 | N | N | N | N | N | IW+PW | N | N | Y | N |
| 152 | F | 52 | N | Y | N | N | N | IW | N | N | Y | N |
| 153 | F | 49 | Y | N | Y | N | N | AS | N | N | Y | N |
| 154 | M | 29 | N | N | Y | N | Y | AL | N | N | Y | N |
| 155 | M | 72 | N | N | Y | Y | Y | IW | N | N | Y | N |
| 156 | M | 68 | N | N | Y | N | Y | AL | N | N | N | N |
| 157 | M | 34 | N | N | N | N | N | IW | N | N | Y | N |
| 158 | M | 47 | Y | N | Y | N | N | AS | N | N | Y | N |
| 159 | M | 63 | N | Y | N | N | N | AS | N | N | Y | N |
| 160 | M | 45 | Y | N | Y | N | Y | AS | N | N | N | N |
| 161 | M | 49 | N | Y | N | Y | N | AS | N | N | Y | N |
| 162 | F | 65 | N | N | N | N | N | IW+PW | N | N | N | N |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 163 | M | 45 | Y | N | N | Y | Y | IW | N | N | N | N |
| 164 | M | 61 | Y | N | N | N | Y | EAW | N | 3,1 | Y | N |
| 165 | M | 36 | N | N | N | N | N | AS +IW | LAHB+RB | N | Y | N |
| 166 | M | 44 | Y | Y | N | N | N | IW | N | N | Y | N |
| 167 | M | 48 | Y | N | N | N | N | IW+PW | N | N | Y | N |
| 168 | M | 42 | Y | N | N | N | N | IW+RV | N | N | Y | N |
| 169 | M | 48 | N | N | Y | N | N | AS | N | N | Y | N |
| 170 | F | 51 | N | N | N | N | N | IW+RV | N | N | Y | N |
| 171 | M | 61 | N | N | Y | N | Y | AS | N | N | Y | N |
| 172 | M | 37 | N | N | N | Y | N | AS | N | N | Y | N |
| 173 | F | 48 | N | Y | N | N | N | AS | N | N | N | N |
| 174 | M | 49 | N | N | Y | N | Y | AS | N | N | Y | N |
| 175 | M | 63 | N | N | Y | N | Y | AS | N | N | Y | N |
| 176 | M | 68 | N | N | N | N | N | IW+LW | N | 1 | Y | N |
| 177 | M | 43 | Y | N | N | N | N | AL | LBBB | N | Y | N |
| 178 | M | 73 | N | N | N | Y | N | IW+PW | RBBB | 1 | Y | N |
| 179 | M | 44 | Y | Y | N | N | N | IW+RV | N | N | Y | N |
| 180 | M | 44 | N | N | N | N | N | AS | N | N | Y | N |
| 181 | F | 63 | N | Y | N | N | N | AS | N | N | Y | N |
| 182 | F | 59 | Y | N | Y | N | N | AW | N | 1,3 | Y | Y |
| 183 | M | 64 | N | N | N | N | N | AS | N | N | Y | N |
| 184 | M | 58 | N | Y | N | N | N | AS | N | N | N | N |
| 185 | M | 68 | N | Y | Y | N | Y | AS | N | N | Y | N |
| 186 | M | 45 | N | N | Y | N | Y | AS | N | N | Y | N |
| 187 | M | 34 | Y | N | N | N | Y | IW | N | 1 | Y | N |
| 188 | F | 60 | N | N | N | N | Y | AS | N | N | Y | N |
| 189 | M | 72 | N | N | Y | N | Y | IW | N | N | Y | N |
| 190 | M | 63 | Y | N | N | N | Y | IW+PW | N | N | Y | N |
| 191 | F | 53 | N | N | N | N | N | IW | RBBB | N | Y | N |
| 192 | M | 45 | N | N | N | Y | N | IW+RV | N | N | Y | N |
| 193 | M | 38 | N | N | N | Y | Y | IW+PW | N | N | Y | N |
| 194 | M | 61 | N | N | Y | N | Y | AS | N | N | N | N |

| | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 195 | M | 64 | N | N | N | Y | N | AS | N | N | Y | N |
| 196 | M | 49 | N | Y | N | Y | N | IW+PW | N | N | Y | N |
| 197 | M | 46 | N | N | N | N | N | IW | RBBB | N | Y | N |
| 198 | M | 48 | Y | Y | N | Y | N | AW | RBBB | N | Y | Y |
| 199 | M | 52 | N | Y | N | N | N | AW | N | 1 | Y | N |
| 200 | M | 48 | Y | Y | Y | N | N | AS | LBBB | 1 | Y | Y |


Turnitin Document Viewer - Google Chrome
https://www.turnitin.com/dv?o=709723469&u=1055596036&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical ...
2015-2015 plagiarism - DUE 07-Nov-20...
Originality
GradeMark
PeerMark
m i conduction block
BY 201411162 MD GEN MED SANGEETHA. K
turnitin
4%
SIMILAR
--
OUT OF 0
INTRODUCTION:
Conduction delay and block is the most commonly witnessed complications of acute infarction of myocardium.
It may be a delay in AV nodal conduction (or) block which may be of II,III degree AV blck depending on the site of abnormality in the conducting system.The abnormality in conduction is due to autonomic dysfunction,decreased blood flow,to the conducting tissue,or an infarction.
As various factors contribute to the conduction block in acute infarction it may be due to transient or fatal.
It is important to anticipate particular tye of conduction abnormality in specific type of lesion in the coronary arteries, as fatal abnormalities can be managed promptly.
Incidence and clinical implications cannot be determined accurately in myocardial infarction as,
i)most studies are retrospective
PAGE: 1 OF 90
Match Overview
1 digital.library.umsyste... Internet source 1%
2 Bülent B. Altunkeser. "... Publication 1%
3 Juyal, S. A., and M. P. ... Publication <1%
4 www.dphhs.mt.gov Internet source <1%
5 Pugazhendhi Vijayara... Publication <1%
6 epaper.sakshi.com Internet source <1%
7 Submitted to Universit... Student paper <1%
8 To Pace or not to Pace... Publication <1%
Text-Only Report



INSTITUTIONAL ETHICS COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No. 07/2016 Dt: 20.06.2016
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval **"CLINICAL STUDY OF CONDUCTION BLOCK IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION"**- For Project Work submitted by Dr.K.Sangeetha, Post Graduate in MD (General Medicine), Govt. Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

DEAN
Govt.Kilpauk Medical College,
Chennai – 10.

PC-2-ME | Section-Ethical Committee 1

# ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு:

"கடுமையான எஸ்-டி ஏற்ற மாரடைப்பில் உள்ள கடத்தல் தடுப்பு பற்றிய மருத்துவ ஆய்வு"

பெயர்: தேதி:

வயது: உள்நோயாளி எண்:

பால்: ஆராய்ச்சி சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

கடுமையான எஸ்-டி ஏற்ற மாரடைப்பில் ஏற்படும் கடத்தல் தடுப்பின் பாதிப்புகள் மற்றும் ,அதனைக் கண்டறிய மேற்கொள்ளப்படும் பரிசோதனைகளைப் பற்றியும் ஆராய்ச்சியாளர் கூற முழுவதும் விளங்கப்பெற்றேன்.

மேற்கொண்ட பரிசோதனையின் போது ஏற்படக்கூடிய பின்விளைவுகளையும் முழுவதும் உணர்ந்து இந்த பரிசோதனைக்கு மனமார சம்மதிக்கிறேன்.

கையொப்பம்

## ஆராய்ச்சி தகவல் தாள்

சென்னை கீழ்பாக்கம் அரசு மருத்துவக் கல்லூரி மருத்துவமனையில் ஆராய்ச்சி ஒன்று நடைபெற்றுவருகிறது. "கடுமையான எஸ்-டி ஏற்ற மாரடைப்பில் உள்ள கடத்தல் தடுப்பு பற்றிய மருத்துவ ஆய்வு" என்பதே இதன் தலைப்பாகும்.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

தேதி: