A Dissertation on

Analysis of 200 cases of conduction disturbances in acute ST elevation Inferior wall myocardial infarction related to causal comparison

Dissertation Submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

With partial fulfillment of the regulations

for the award of the degree of

M.D. GENERAL MEDICINE

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(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

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Course : M. D. GENERAL MEDICINE.

Period of Study : ONE YEAR.

College : COIMBATORE MEDICAL COLLEGE

Dissertation Topic : ANALYSIS OF 200 CASES OF CONDUCTION DISTURBANCES IN ACUTE ST ELEVATION INFERIOR WALL MYOCARDIAL INFARCTION RELATED TO CAUSAL COMPARISON.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not~~ accepted and you are permitted / ~~Not~~ permitted to proceed with the above Study.

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CERTIFICATE

Certified that this is the bonafide dissertation done by ***Dr.A. RADHA KRISHNAN*** *and submitted in partial fulfillment of the requirements for the Degree of* ***M.D.,General Medicine****, Branch I of* ***The TamilnaduDr. M.G.R. Medical University, Chennai.***

Date: **Guide,Professor &Chief**
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Date: **Dean**
Coimbatore Medical College
Coimbatore

DECLARATION

I solemnly declare that the dissertation titled **"Analysis of 200 cases of conduction disturbances in acute ST elevation Inferior wall myocardial infarction related to causal comparison"** was done by me from JULY 2016 to JUNE 2017 under the guidance and supervision of Professor **Dr.K.SWAMINATHAN. M.D.,**

This dissertation is submitted to **The TamilnaduDr.M.G.R.Medical University** towards the partial fulfilment of the requirement for the award of MD Degree in General Medicine(Branch I).

Place: Coimbatore **Dr.A.RADHA KRISHNAN**

Date:

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Lastly, I am ever grateful to the **ALMIGHTY GOD** for always showering His blessings on me and my family.

DATE: **Dr.A.RADHA KRISHNAN**

CERTIFICATE – II

This is to certify that this dissertation work titled **"Analysis of 200 cases of conduction disturbances in acute ST elevation inferior wall myocardial infarction related to causal comparison"** of the candidate Dr.A.RADHA KRISHNAN with registration Number 201511310 for the award of M.D in the branch of General Medicine I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 1% (one percentage) of plagiarism in the dissertation.

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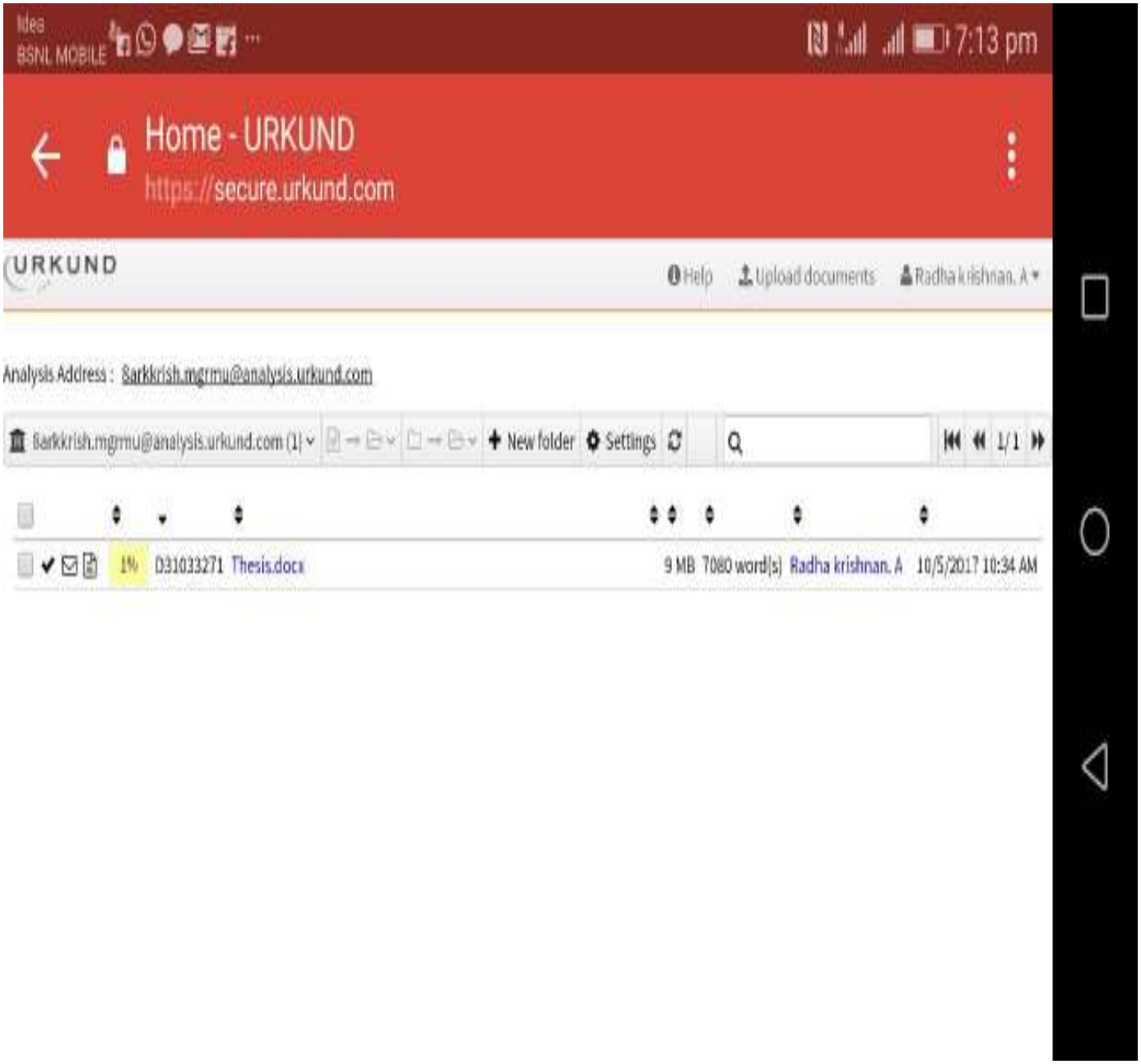
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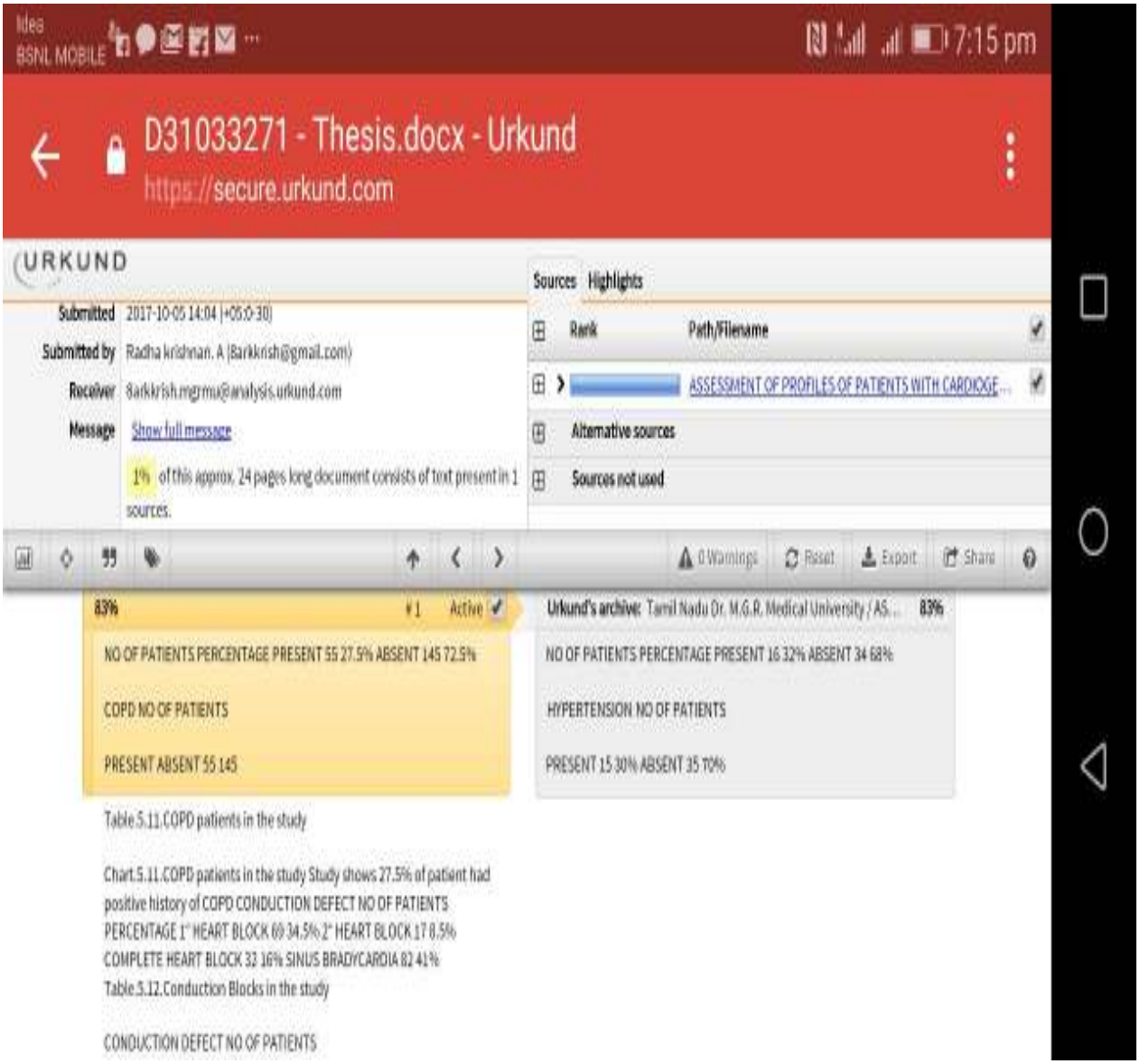
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PRESENT 15 30% ABSENT 35 70%
Table 5.11.COPD patients in the study
Chart 5.11.COPD patients in the study Study shows 27.5% of patient had positive history of COPD CONDUCTION DEFECT NO OF PATIENTS PERCENTAGE 1° HEART BLOCK 69 34.5% 2° HEART BLOCK 17 8.5% COMPLETE HEART BLOCK 33 16% SINUS BRADYCARDIA 82 41%
Table 5.12.Conduction Blocks in the study
CONDUCTION DEFECT NO OF PATIENTS

LIST OF ABBREVIATIONS USED CONTENTS

AWMI	-	Anterior Wall Myocardial Infarction
AV BLOCK	-	Atrio Ventricular Block
CAD	-	Coronary Artery Disease
CHB	-	Complete Heart Block
ECG	-	Electro Cardio Graph
HDL	-	High Density Lipoprotein
IWMI	-	Inferior Wall Myocardial Infarction
LDL	-	Low Density Lipoprotein
LWMI	-	Lateral Wall Myocardial Infarction
PWMI	-	Posterior Wall Myocardial Infarction
RVMI	-	Right Ventricular Myocardial Infarction
TC	-	Total Cholesterol

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INTRODUCTION

Coronary artery disease remains a major disease problem in world even though a development of diagnosis management and prevention of risk factors. Prevalence of coronary artery disease increased two-fold in last 20 years in both rural and urban population. Acute myocardial infarction is a major cause of death in developed and develop countries including india. Coronary artery disease is responsible for 1 in 5 deaths in united states .

In India CAD is the leading cause of death 32% of all deaths. Commonly anterior wall myocardial infarction , inferior wall myocardial infarction, lateral wall myocardial infarction, posterior wall myocardial infarction occur.

Inferior wall myocardial infarction account for 40-50% of all myocardial infarction .conduction disturbances in acute myocardial infarction occur anywhere in the heart which is divided into four major categories according to location of block sino atrial block ,intra atrial block, atrio ventricular block, interventricular block. sino atrial, atrio ventricular blocks are more common in inferior wall myocardial infarction. This is because occlusion of dominant artery more than 70% cases.

AV nodal block can also be due to increased vagal tone due to stimulation of afferent nerves adjacent to AV node by ischeamic cells. It may also be due to release of chemical mediators like potassium and adenosine from ischeamic cells.

William Einthoven invented first practical electrocardiogram.(fig.1.1)

Harold Ensign Bennet Pardee helped to define in an organized manner. The electrocardiographic patterns associated with coronary insufficiency and myocardial infarction.(fig.1.1)

Paul Dudley White founder of preventive cardiology.(fig.1.1)

Inge Edler father of Echocardiography.(fig.1.2)

Charles Theodore Dotter described angioplasty.(fig.1.4)

Andreas Ronald Gruntz develop balloon angioplasty.(fig.1.3)

Bernard Lawn developer of DC defibrillator and the cardioverter. (fig.1.4)

FIG 1.1:The pioneers of Electrocardiogram

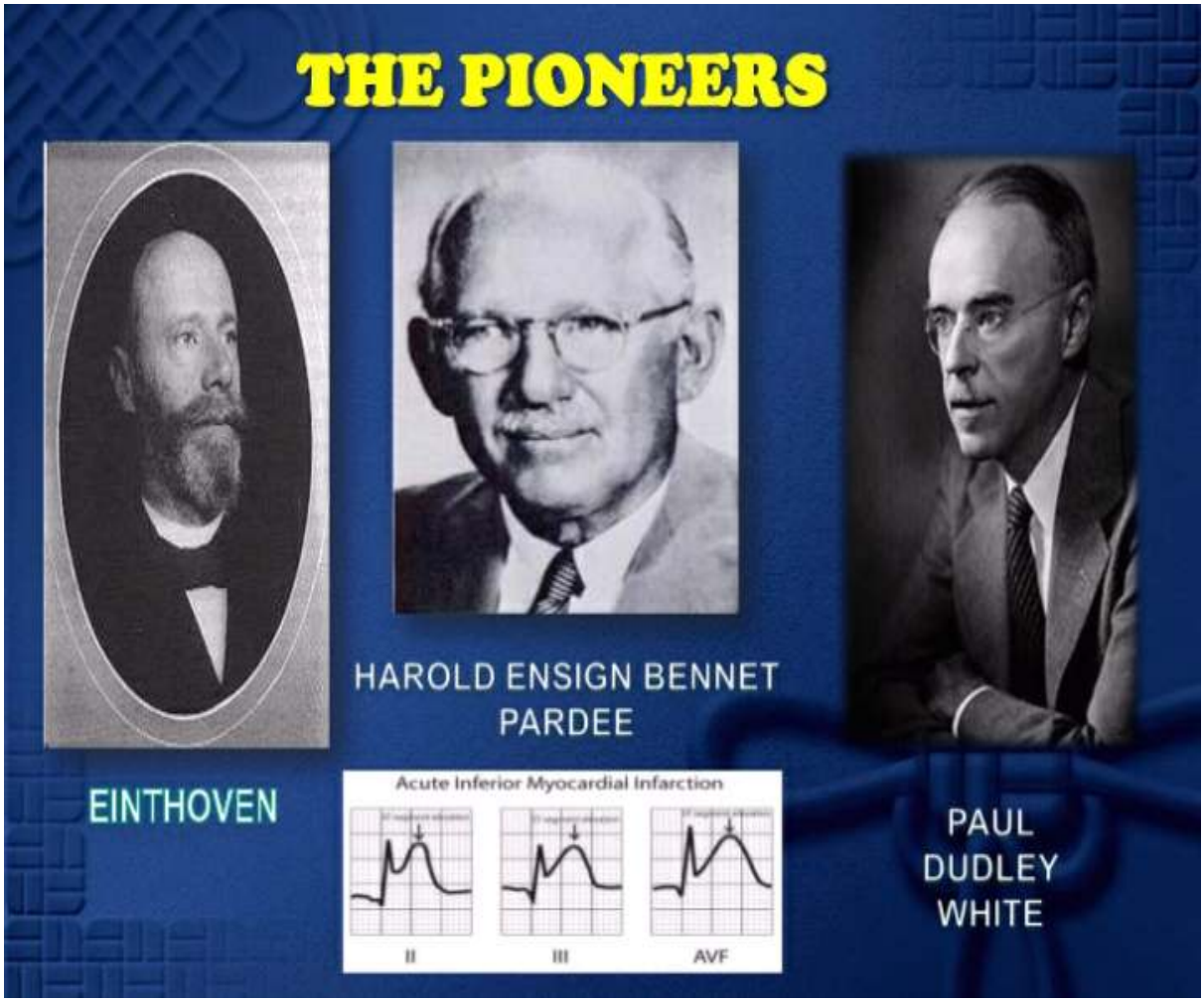

FIG.1.2:Inventors of ECHO

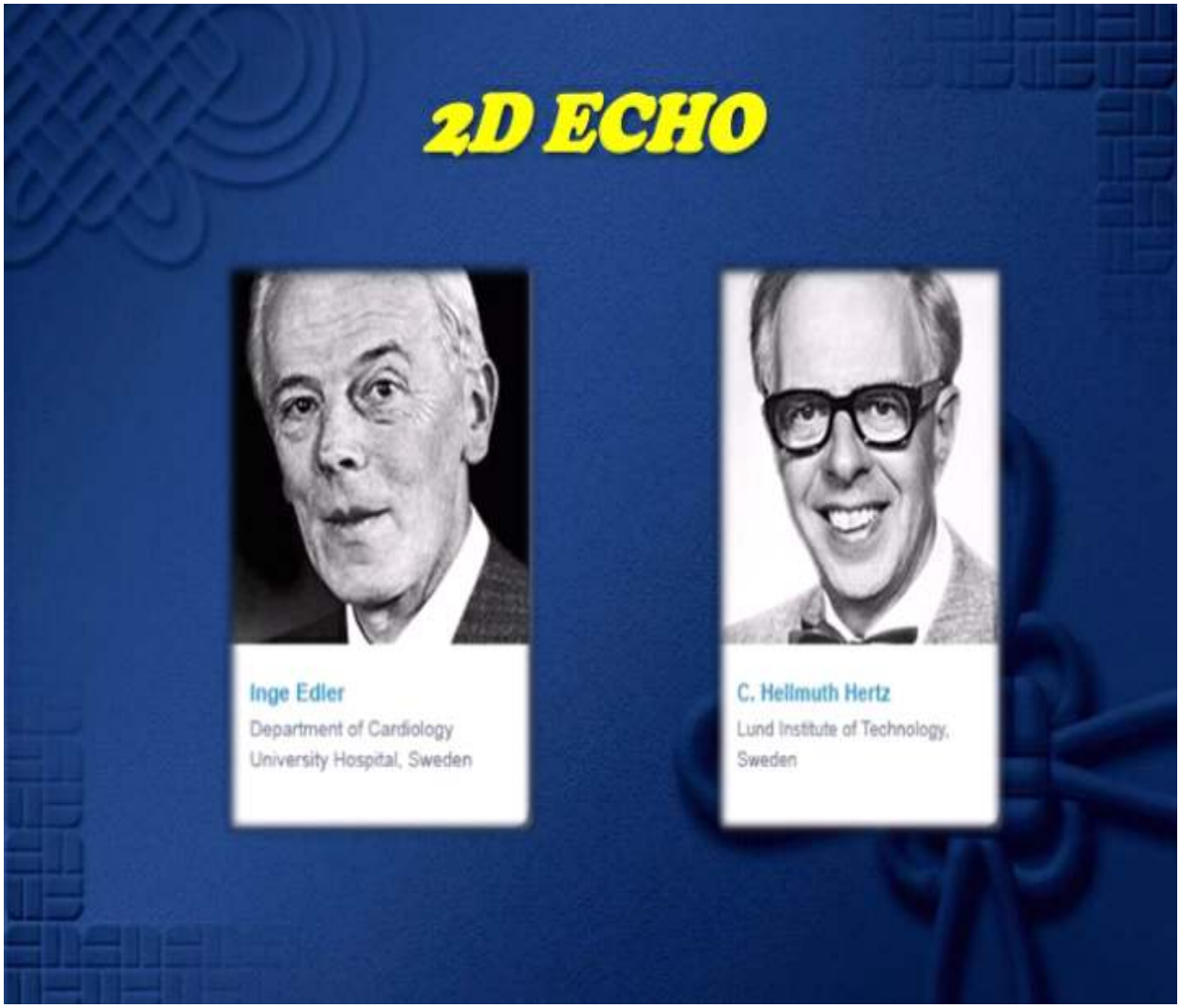

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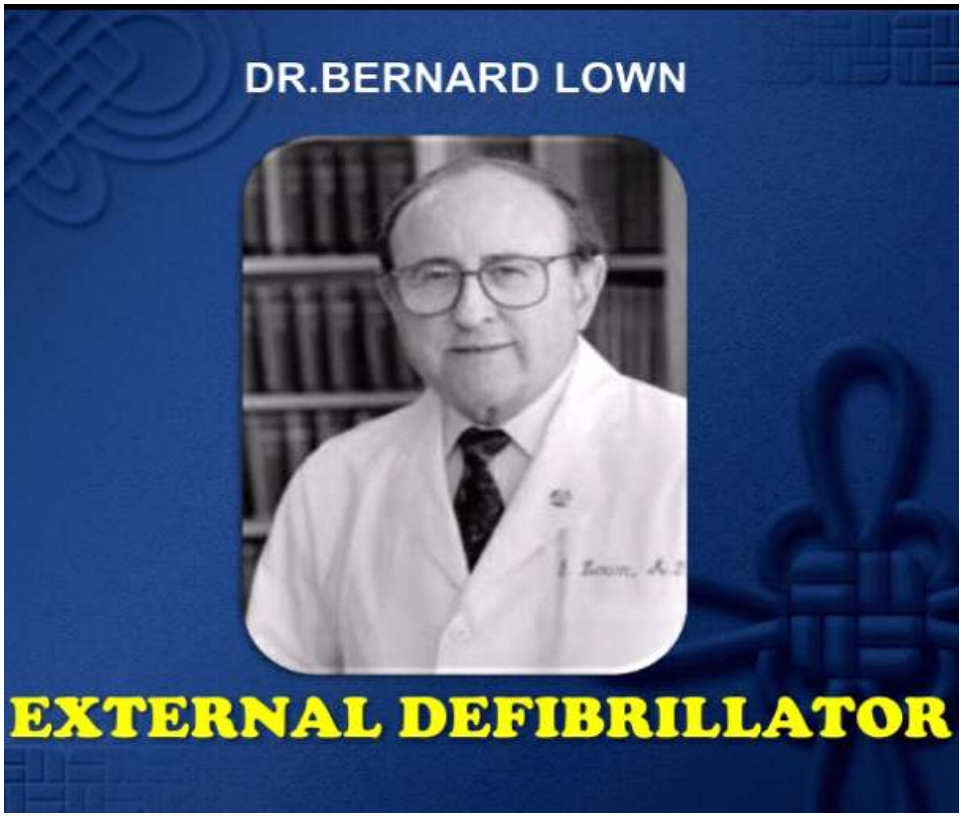
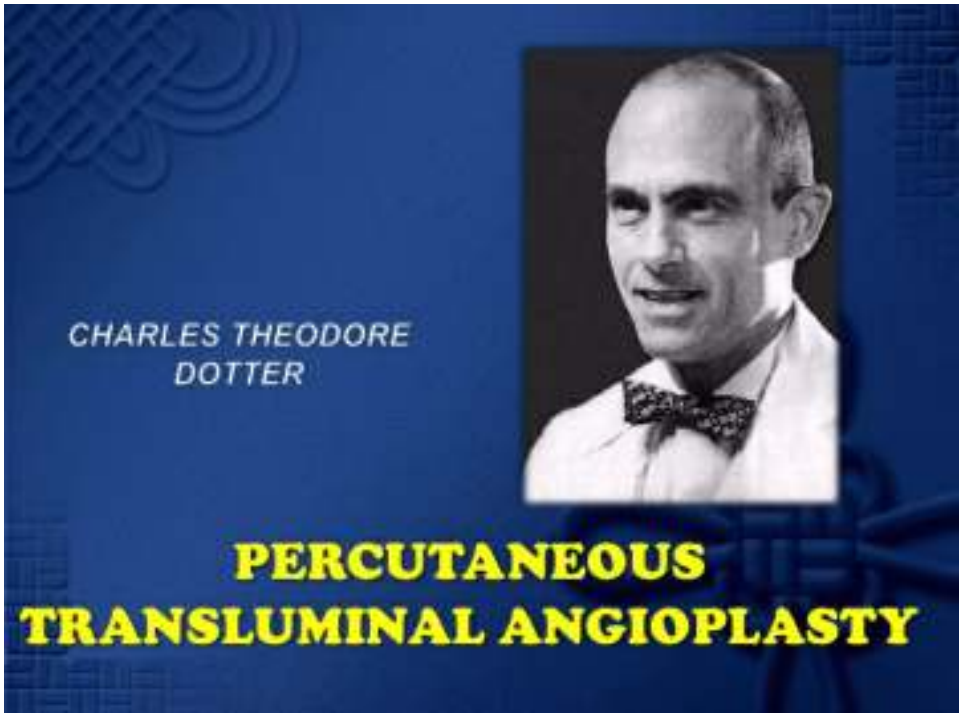
FIG.1.3:Inventor of Baloon Angioplasty

FIG.1.4:Inventors of percutaneous transluminal Angioplasty and External Defibrillator





AIMS AND OBJECTIVES OF THE STUDY

The aim to analyze 200 cases of conduction disturbances in patients with acute ST elevation inferior wall myocardial infarction related to following causal comparisions.

Age, sex

Smoking

Alcoholism

hypertension

diabetes mellitus

total cholesterol

HDL

LDL

COPD

To find out any association of each comparisions group with ST elevation inferior wall myocardial infarction particularly on the risk of conduction disturbances.

MATERIALS AND METHODS

In this, observational cross sectional study. Total of 200 cases of acute inferior wall myocardial infarction with conduction disturbances admitted in intensive care unit of Coimbatore medical college hospital during one year period from JULY 2016 to 30 JUNE 2017 were enrolled.

The subjects fulfilling the following criteria were included in this study. Age above 18 years. Patient fulfilling ECG criteria for acute inferior wall ST elevation myocardial infarction.ST elevation > 1 mm in 2,3,avf leads.

Patients below 18 years of age, anterior wall myocardial infarction, old inferior wall myocardial infarction and patients on drugs like calcium channel blockers, beta blockers, digoxin and other anti arrhythmic drugs were excluded.

Subjects fulfilling the inclusion criteria were analyzed. A Proforma was used to record the variables including patients name, age address. A detailed history was recorded to obtain typical symptoms and risk factors .History of hypertension, diabetes, smoking, alcoholism, dyslipidemia and COPD were noted.

A detailed clinical examination was carried out. Investigations like complete hemogram, random blood sugar, blood urea serum creatinine, electrolytes, lipid profile and electrocardiogram were taken.ECG fulfilling the criteria of inferior wall myocardial infarction with conduction disturbances were taken.

Hypertension is defined as a previous record of at least two blood pressure recording > 140/90 mmHg or the requirement of regular intake of antihypertensive drugs.

Diabetes is defined as random blood sugar >200 mg/dl with symptoms of diabetes or the requirement of regular hypoglycemic drugs.

Dyslipidemia is defined as total cholesterol level >200mg/dl LDL cholesterol level >130 mg/dl, HDL cholesterol level < 50mg/dl.

Patients were identified as non smoker if they never smoked or they gave up smoking 15 years back, as an ex-smoker if they stopped smoking >3 months and as current smoker if they still smoked within last 3 months.

Patients were identified as non alcoholic if they never drinked or they gave up drinking 15 years back, ex-alcoholic if they stopped drinking >3 months and as a current alcoholic if they still drinked with in last 3 months COPD patients were identified by only previous evidence of COPD history or patient on bronchodilators.

Patients with acute ST elevation inferior wall myocardial infarction with conduction disturbances evidenced by electrocardiogram were taken. These patients are analyzed with their risk factors

REVIEW OF LITERATURE

Acute myocardial infarction is the one of the most common diagnosis in hospitalized patients .commonly anterior wall ,inferior wall, lateral wall ,posterior wall myocardial infarction occur .Mortality and morbidity is more common in anterior wall myocardial infarction than inferior wall myocardial infarction.

CORONARY CIRCULATION

Coronary circulation is the circulation of blood in the blood vessels of the heart muscle (myocardium).The vessels that deliver oxygen rich blood to the myocardium are the coronary arteries. The vessels that remove the deoxygenated blood from the heart muscle are known as cardiac veins .The arteries when healthy capable of auto regulation to maintain coronary blood flow to the needs of cardiac muscles. Relative narrow of vessels commonly affected by atherosclerosis causing angina or heart attack . Coronary arteries are end arteries that represent only source of blood supply to myocardium. Coronary arteries supply blood to myocardium mother components of heart. Two coronary arteries originate from the left side of heart at the beginning of aorta, just after

the aorta exits the left ventricle. There are three aortic sinuses (dilations)in the walls of aorta just superior to aortic semilunar valve. Two of these ,left posterior aortic sinus and anterior aortic sinus give rise to left and right coronary arteries respectively.

Coronary vessel branches that remain on the surface of the artery and follow the sulci of the heart are called epicardial coronary arteries.

Left coronary supplies blood to left side of heart, left atrium, left ventricle and interventricular septum. Circumflex artery arises from left coronary artery and follows the coronary sulcus to the left. Left anterior descending artery is the second major branch arising from left coronary artery. It follows anterior interventricular sulcus around pulmonary trunk. Right coronary artery follows along coronary sulcus and distributes blood to the right atrium, portion of both ventricles, and the heart conduction system.(fig.3.1)

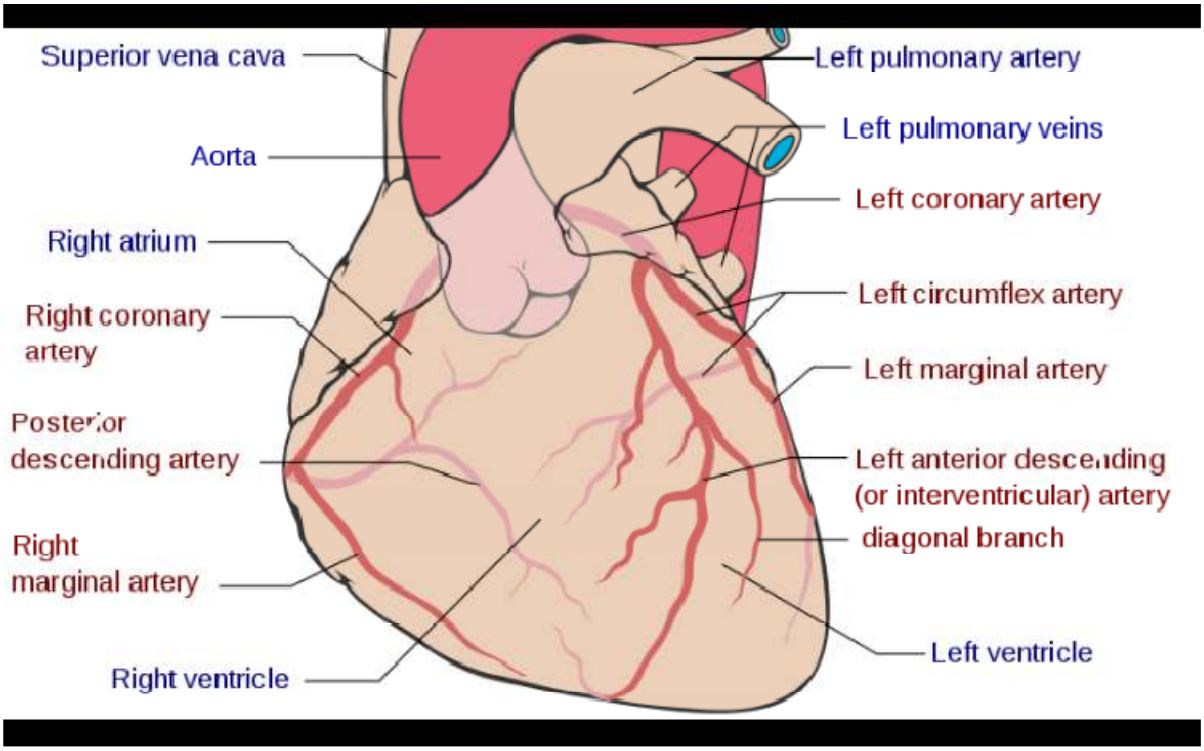


FIG.4.1:Coronary Circulation

AETIOPATHOGENESIS OF MYOCARDIAL INFARCTION

1.ATHEROSCLEROSIS:

Atherosclerosis is characterized by atheroma or fibro fatty plaque formation. consists of raised focal plaque with in intima, having core of lipid and a covering of fibrous cap. Atherosclerosis is the single most etiological factor for coronary artery disease. The cause and pathogenesis of atherosclerosis explains varies hypothesis are

A) REACTION TO INJURY HYPOTHESIS:

This is widely accepted theory by ROSS and Glomset in 1976 and modified 1986. The lesions of atherosclerosis is initiated as a response to injury to arterial endothelium. Injury leads to attachment of monocytes, platelets and proliferation of smooth muscle cells in the intima and deposition of intracellular and extracellular lipids.(fig.4.3).

B) LIPID INSUDATION OR INFILTRATION HYPOTHESIS:

This modified inhibition hypothesis by Virchow in 1856 .Cellular proliferation in intima is a form of low grade inflammation which leads to increased infiltration of plasma protein and lipids from blood.

C) ENCRUSTATION OR THROMBOGENIC HYPOTHESIS:

Described by Rokitansky that explains small thrombi composed of platelet ,fibrin and leucocytes collected over foci of endothelial injury organized and their gradual growth result in plague formation.

HISTOPATHOLOGY OF ATHEROSCLEROTIC LESION

Stary 1 lesion:

The endothelium express surface adhesion molecules like E selectin and P selectin which attracts more polymorphonuclear cells and monocytes in subendothelial space.

Stary 2 lesion:

Macrophages takes large amount of LDL

Stary 3 lesion:

Process continues macrophages become foam cells

Stary 4 lesion:

Lipid exudes into the extracellular space begins to form lipid core.(fig.4.2)

Stary 5 lesion:

Fibroblasts and smooth muscle cells moves forming fibroatheroma with inner lipid cores and outer fibrous cap.

Stary 6 lesion:

Fibrous cap ruptures forming thrombosis which leads to acute coronary syndrome.

Stary 7 lesion:

Lesions stabilize become fibrocalcification

Stary 8 lesion:

Finally fibrotic with extensive collagen content

2)NON-ATHEROSCLEROSIS:

1) Injury or spasm of coronary artery

2) Dissection of coronary artery or aorta

3) Embolic phenomena from prosthetic valves ,infective endocarditis tumors calcium ,paradoxical embolus,etc

4) Coronary arteritis due to Takayasu disease, Polyarteritis Nodosa, SLE, Syphilis,Kawasaki disease

5) Congenital anomalies like single coronary artery, atresia of coronary ostium, myocardial bridges, coronary AV fistula

6) Metabolic disorders like mucopolysaccharidoses, Homocystenemia fabry's disease and Amyloidosis.

7) Substance abuse like cocaine, amphetamine

8) Myocardial oxygen demand and supply disproportion due to aortic stenosis, systemic hypertension, carbon monoxide poisoining, thyrotoxicosis. 9) Intimal proliferation due to cardiac

transplantation irradiation, fibromuscular dyaplasia.

10) Misceelaneous cause like hypercoagulable state,diabetes mellitus,HOCM.

FIG.4.2:Lipid core formation in Atherosclerosis

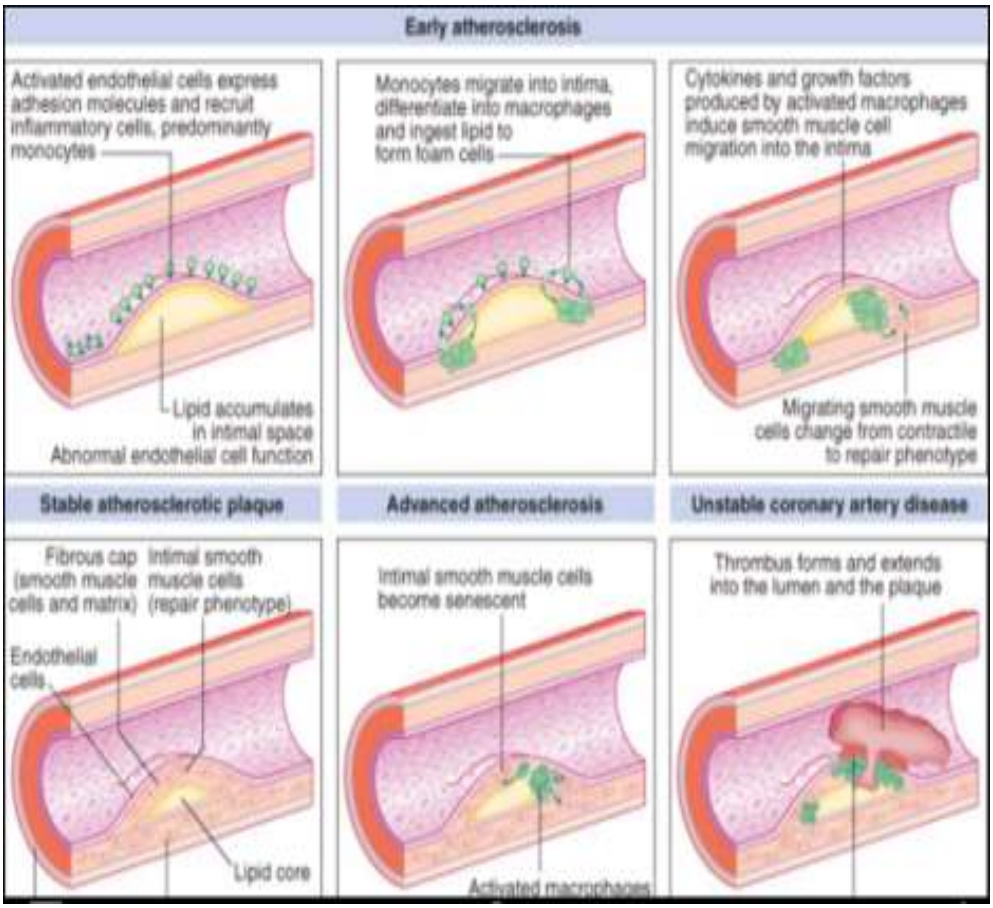
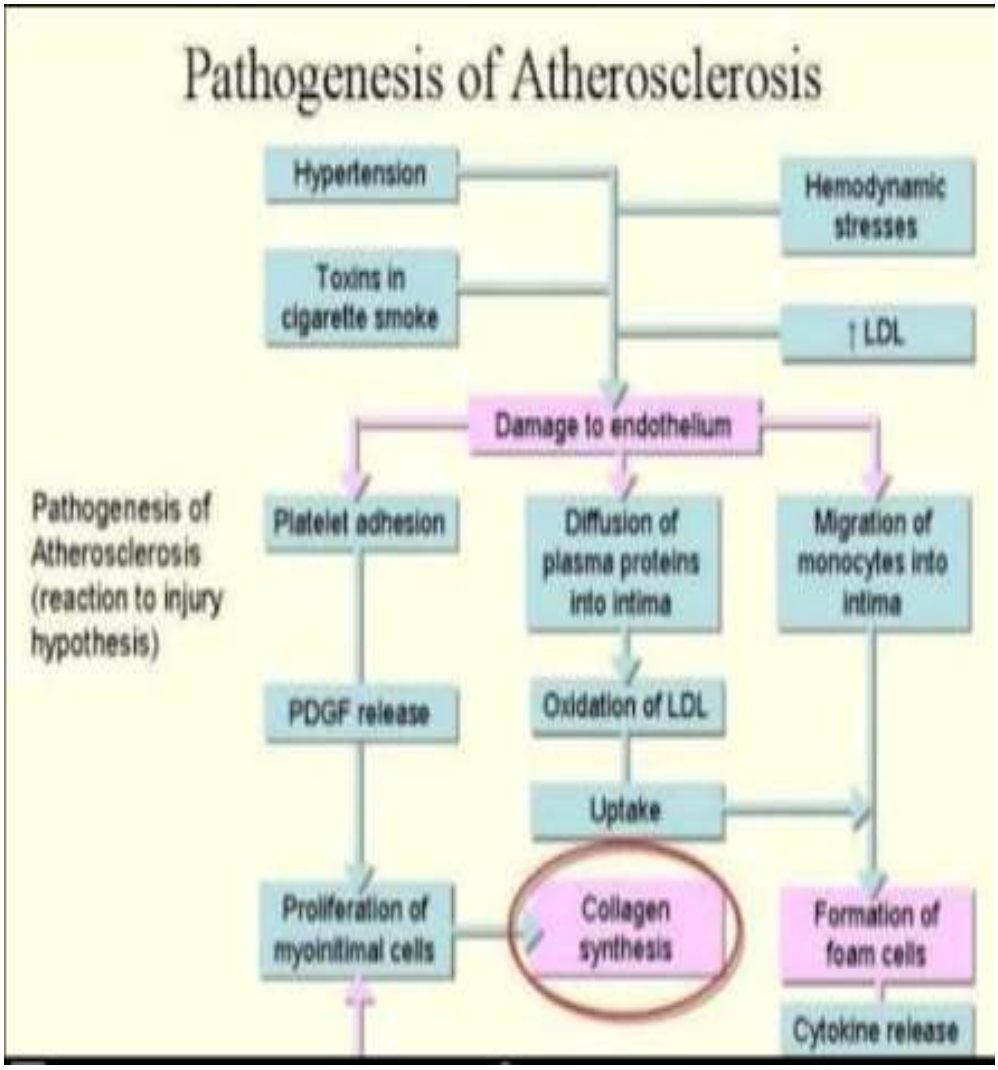


FIG.4.3:Pathogenesis of Atherosclerosis



RISK FACTORS FOR MYOCARDIAL INFARCTION:

NON MODIFIABLE :

Age

Male sex

family history

MODIFIABLE :

Smoking

Hypertension

Lipid disorders

Diabetes mellitus

Obesity

Sedentary life style

Haemostatic variables

Mental stress personality

Oral contraceptive pills

Hyperhomocysteinemia, Inflammation.

AGE:

Age is the strongest risk factor for the development of coronary artery disease. Elderly persons have higher morbidity and mortality rate. Atherosclerosis progress as age advances. Atherosclerosis rarely present In childhood. Except in familial hyperlipidemia.

SEX:

Mens are more affected than women because of protective effect of the female sex hormones. But after menopause incidence of atheroma rises. Fall in HDL levels in postmenopausal women which plays in development of clinical manifestation.

FAMILY HISTORY:

Coronary artery disease may run in families. Family history of premature atherosclerosis may increase the risk of premature death.

SMOKING:

Smoking increases the risk of coronary artery disease because carbon monoxide content of smoke decreases oxygen carrying capacity of hemoglobin. Nicotine and other substances in smoke affects vascular smooth muscles and platelets may initiate thrombotic events when circulation is compromised by atherosclerosis.

Smoking may increase the risk of arrhythmias. Smoking decreases the HDL Cholesterol level. It increase the risk of atherosclerosis causing coronary artery disease. Continuation of smoking increases the risk of restenosis after percutaneous transluminal angioplasty and coronary artery byepass grafting .

HYPERTENSION:

The incidence of coronary artery disease increases when blood pressure rises. The risk is related to both systolic and diastolic blood pressure. In Framingham study the incidence of coronary artery disease increased 5 times in middle aged person when blood pressure exceeding 160/95mmHg that in normotensive. There is 37%increase in risk of coronary artery disease for each 10 mmHg increase in diastolic blood pressure.

DIABETES MELLITUS:

It is important risk factor for hyperlipidemia by which increases the risk for atherosclerosis .The term Diabetic Dyslipidemia which means abnormal lipid profile with insulin resistance (small dense LDL, low HDL, elevated triglycerides) increases the cardiovascular risk.

DYSLIPIDEMIA:

High level of total cholesterol or low density lipoprotein(LDL) and low level of high density lipoprotein (HDL) increases the risk of atherosclerosis by which increases the risk of coronary artery disease. Low density lipoprotein is highly atherogenic HDL helps to remove cholesterol from arterial wall by which it has protective effect.LDL/HDL ratio calculation is a way to assess atherosclerosis. Ratio more than 4.5 is more atherogenic. The LDL cholesterol is phenotypically is subdivided into pattern A,B and C of which phenotype B is mostly small dense LDL particles which is linked to an increased risk of coronary artery disease independent of total LDL cholesterol levels.

OBESITY:

It is a significant risk factor for atherosclerosis. It may be associated with hypertension ,diabetes mellitus and physical inactivity. Increasing in body weight increases the other risk factor for atherosclerosis like increase in blood pressure, cholesterol, triglycerides, blood glucose levels and decrease in HDL cholesterol level.

SEDENTARY LIFE STYLE:

Atleast 30 min of intense physical activity increases HDL level, lowers blood pressure, reduces blood clotting and development of collateral vessel formation.

HAEMOSTATIC FACTORS:

Fibrinogen and factor 7 are important factors which increases the risk of coronary artery disease. Arterial thrombus stability mainly depends upon the balance between fibrinolytic factors like plasmin and inhibitors like plasminogen activator inhibitor. Apolipoprotein has structural homology with plasminogen. so which inhibits cell surface mediated endogenous fibrinolysis by reducing the formation of plasminogen activator.

MENTAL STRESS AND PERSONALITY:

Stress increases the catecholamine levels and increases the blood pressure by which increases the risk of coronary artery disease. Type A individuals who is always aggressive, impatient and competitive prone for coronary artery disease.

ORAL CONTRACEPTIVE PILLS:

It increases inactivity of factor 7 and 10 by which increases the clotting mechanism. It increases platelet adhesiveness by increases platelet sensitivity to ADP

HYPERHOMOCYSTEINEMIA:

It is the main cause for young myocardial infarction. Mutation in enzymes involved in homocysteine metabolism causing homocysteine accumulation which correlate with thrombosis and coronary risk.

INFLAMMATION:

Inflammatory cells induce plaque thrombogenicity by inducing tissue factor which is procoagulant that activates clotting factors .Inflammatory cells in the plaque producing matrix degrading metalloproteinases which induce plaque destabilization and smooth muscle apoptosis.

MYOCARDIAL INFARCTION:

Myocardial infarction resulting from abrupt reduction in coronary blood flow to a segment of myocardium .

Anterior wall myocardial infarction (AWMI):(fig.4.4).

Defined by ST elevation >2mm in V1-V6 leads

Inferior wall myocardial infarction(IWMI):(fig.4.5).

Defined by ST elevation >1 mm in 2,3 avf leads.

Lateral wall myocardial infarction(LWMI):(fig.4.6).

Defined by ST elevation >2mm in 1,avl,v5,v6.

Posterior wall myocardial infarction(PWMI):(fig.4.7).

Defined by ST depression in leads v1-v3.

Right ventricular infarction(RVMI):(fig.4.8)

It is diagnosed with ST elevation in V4R. Inferior wall myocardial infarction is mainly associated with posterior wall myocardial infarction and right ventricular infarction.

Fig.4.4.Anterior wall myocardial infarction

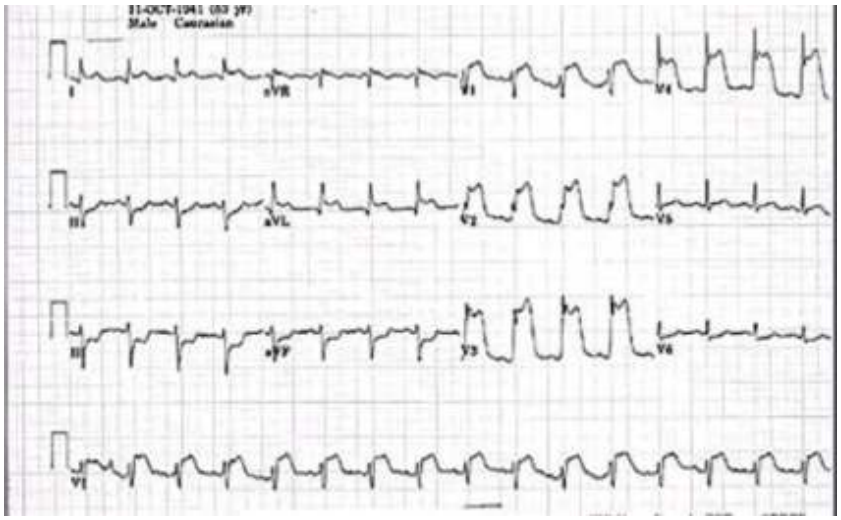


Fig.4.5.Inferior wall myocardial infarction

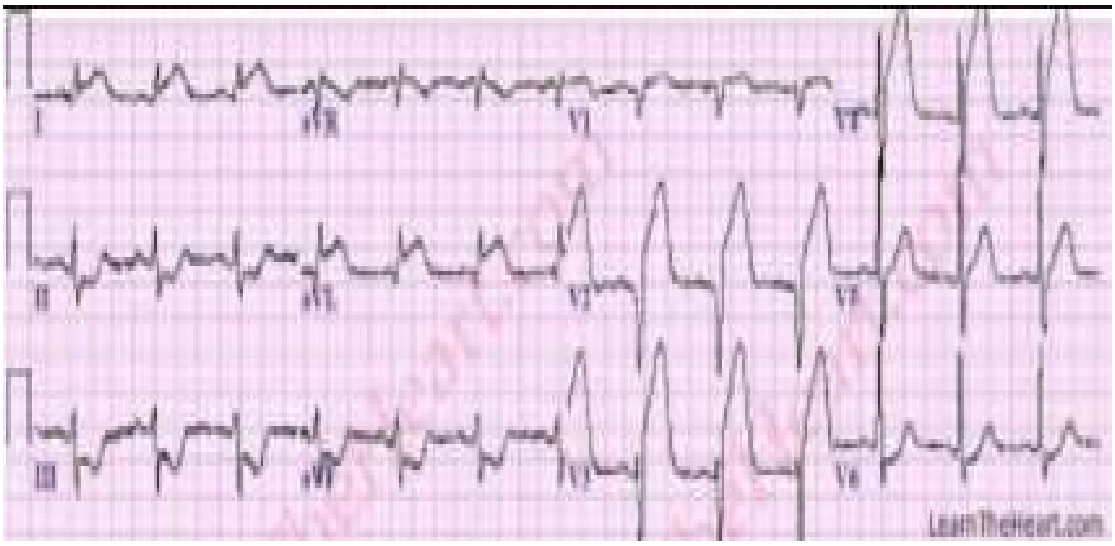


Fig.4.6.Lateral wall myocardial infarction

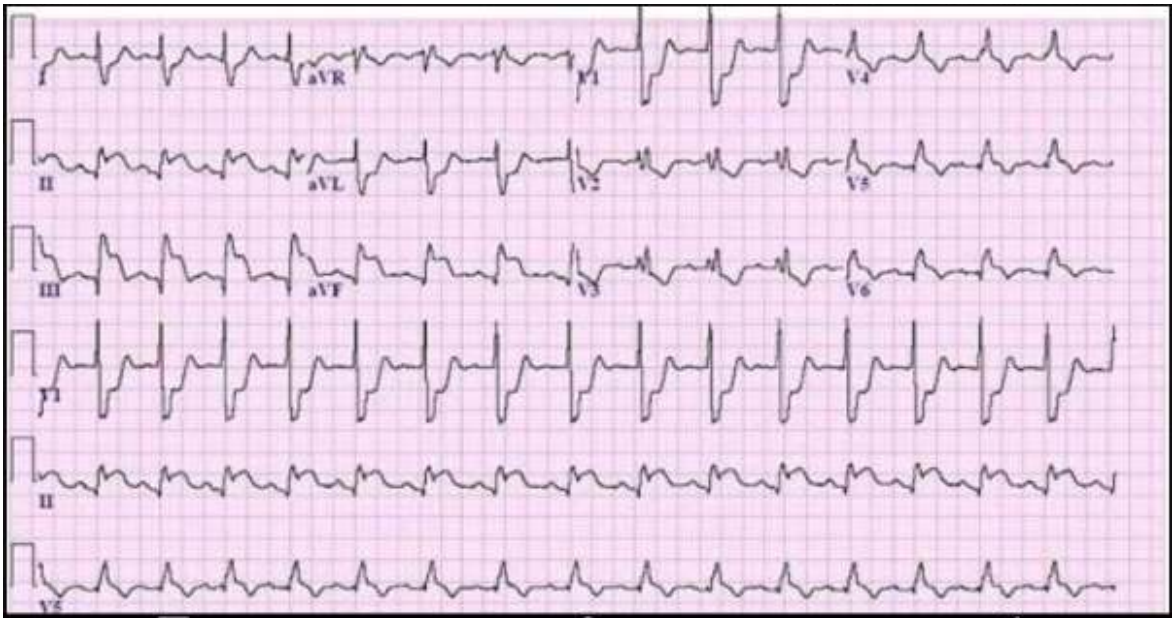


Fig.4.7.Posterior wall myocardial infarction

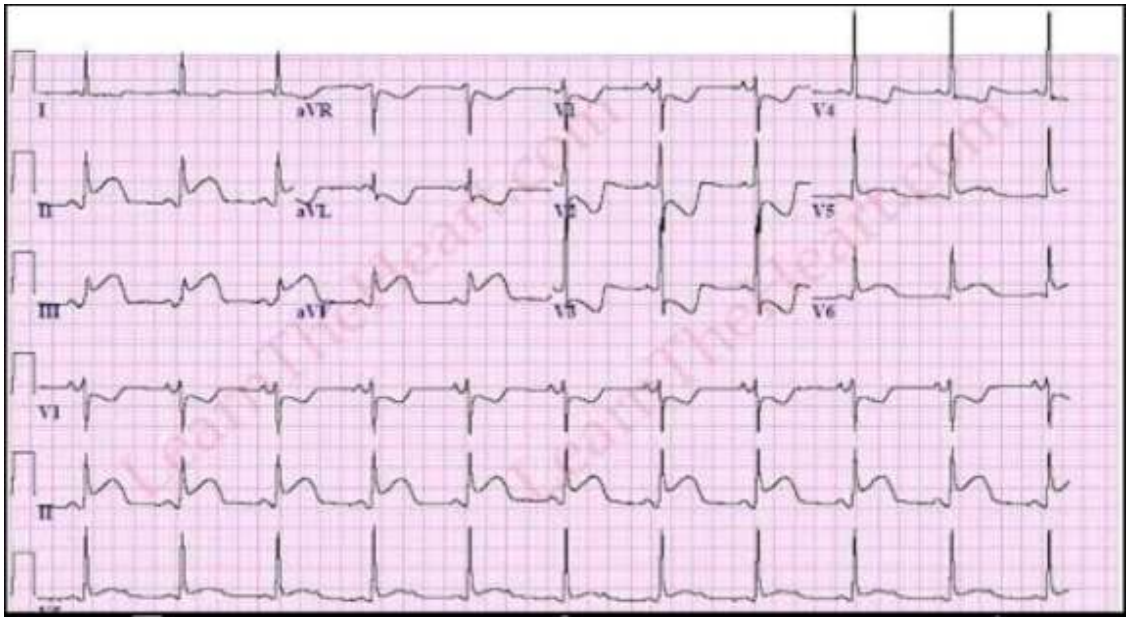
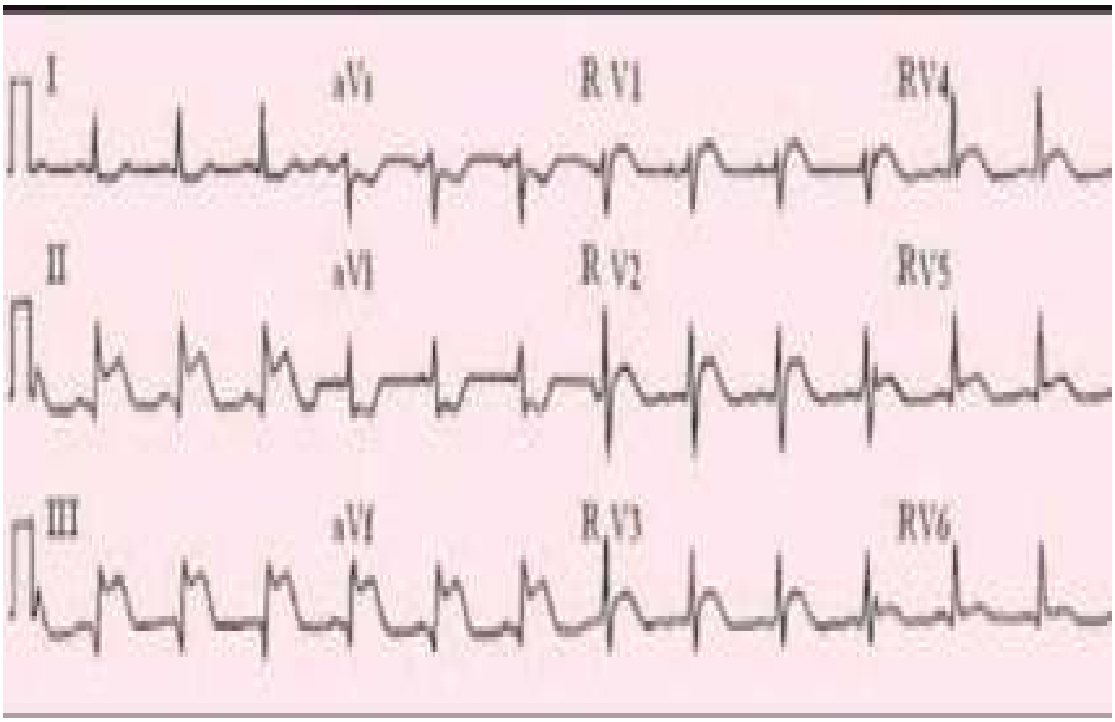


Fig.4.8. Right ventricular infarction



INFERIOR WALL MYOCARDIAL INFARCTION

Defined by ST elevation > 1 mm in 2,3,avf leads

CLINICAL FEATURES:

HISTORY:

Typical retrosternal chest pain which is mainly associated with sweating, palpitation. Pain may radiate to left arm forearm, neck or back .which may be associated with breathlessness. some patients may experience atypical symptoms like acute confusion, giddiness, syncope, stroke, gastro intestinal symptoms like nausea, vomiting, heartburn and abdominal pain. Some patients particularly with the history of diabetes mellitus didn't have any classical symptoms. Giddiness and syncope due profound hypotension as in case of inferior wall myocardial infarction. vomiting due to vagal stimulation as in case of inferior wall myocardial infarction.

PHYSICAL:

Pulse rate; Bradycardia is more common in inferior wall myocardial infarction, tachycardia is common other type of myocardial infarction High or low blood pressure. Hypotension reflects hemodynamic compromise which reflects poor outcome.

Tachypnoea

Diaphoresis

Syncope

Pedal edema.

Heart failure:

Heart failure symptoms may indicate cardiogenic shock .Presence of S3 gallop indicates reduced left ventricular function. Presence of S4 gallop indicates failure. Appearance of heart murmurs indicates poor prognosis.

Respiratory system:

Rales and rhonchi

INVESTIGATIONS:

Complete blood count

Random blood sugar

Blood urea

Serum creatinine electrolytes

Lipid profiles

Total cholesterol

LDL

HDL

ELECTROCARDIOGRAM:

1)T Waves:

Peaked upright T waves may be the first ECG manifestation of myocardial infarction

2) ST Changes:

- Convex ST segment elevation > 1mm in 2 consecutive lead with peaked or inverted T waves is indicative of myocardial injury.
- Inferior wall myocardial infarction is recognized By ST segment elevation >1mm in leads 2,3,avf.

- Posterior wall myocardial infarction is recognized by ST depression in leads V1-V3.
- Right ventricular infarction is recognized by ST elevation in V4R.

3) Q Waves:

Development of new pathological Q waves

4) New bundle branch block

SERUM MARKERS:

a) Creatine kinase with MB isozymes:

Level increases with in 3-12 hours of chest pain, peak at 24 hours and return to baseline after48-72 hours. It has >95% sensitivity and specificity for myocardial injury.

b)Troponins(I or T):

Important biomarker for the diagnosis of ST segment elevation myocardial infarction. level increases 3-12 hours of chest pain, peak at 24-48 hours, return to baseline after 5-14 days.

c) Lactate dehydrogenase and isozymes

CHEST X-RAY:

It shows complications of coronary artery disease like cardiac enlargement ,signs of cardiac failure, pericardial effusion and ventricular aneurysm. These signs are important in assessing degree of cardiac involvement.

SPECIAL TESTS:

a) Specific lipid studies small dense LDL-C level , Apoprotein profile

b) Miscellaneous tests:

Homocysteine level, Inflammatory markers(CRP)

IMAGING STUDIES:

Echocardiography

a) Transthoracic echocardiography - Assess left ventricular function, wall motion abnormalities and mechanical complication

b) Transesophageal echocardiography- Assessing possible aortic dissection in the setting of acute myocardial infarction

C) Stress echocardiography**:** used to evaluate hemodynamically significant stenosis who are thought to have coronary artery disease.

d)Treadmill echocardiography stress testing provide equivalent predictive values

Nuclear imaging studies(myocardial perfusion imaging):

Useful in assessing significant coronary artery stenoses.

Electron beam CT scanning:

It is a noninvasive method to identify calcium content of coronary artery. **Magnetic resonance angiography**

PROCEDURES:

Coronary angiography:

Coronary arterial luminography for significant flow limiting stenoses that must be revascularized through percutaneous or surgical intervention to improve prognosis.

Intravascular ultrasound:

Demontrates the luminal dimensions and tissue composition of vascular

COMPLICATIONS OF INFERIOR WALL MYOCARDIAL INFARCTION:

ARRHYTHMIAS:
1**)BRADYARRHYTHMIAS AND CONDUCTION DISTURBANCES:**

Sinus node dysfunction;

May present as sinus bradycardia, sinoatrial block or sinus arrest. It usually due to vagal stimulation as in case of inferior wall myocardial infarction because of sinus node ischaemia. Normal conduction system is explained in (fig.3.9). symptoms of hypotension shock may occur sometimes asystole and cardiac arrest may occur. Treatment consists of IV atropine should be given. If it is ineffective temporary pacemaking is necessary.

Av Nodal Block:

First degree heart block, second degree heart block, complete heart block. It is usually occurs in inferior wall myocardial infarction. These blocks are usually transient and respond to IV atropine.

If giddiness, hypotension or other evidence of hemodynamic compromise occurs or if the ventricular rate is less than 50/mt temporary pacing may be required. Permanent pacemaker required in comple heart block associated with bradycardia ,second degree AV block with symptomatic bradycardia or asymptomatic complete heart block with ventricular rate 40/mt.

Distal conduction defects:

Consists of right bundle and the anterior and posterior fascicles of the left bundle. Distal conduction system supplied by left coronary artery. this type of conduction defect is more common in anterior wall myocardial infarction.

Clinical symptoms consists of syncopal attacks ,hypotension may lead to cardiac arrest. Temporary pacemaker may necessary. patients with bifascicular block who develop trifascicular block need permanent pacemaker.

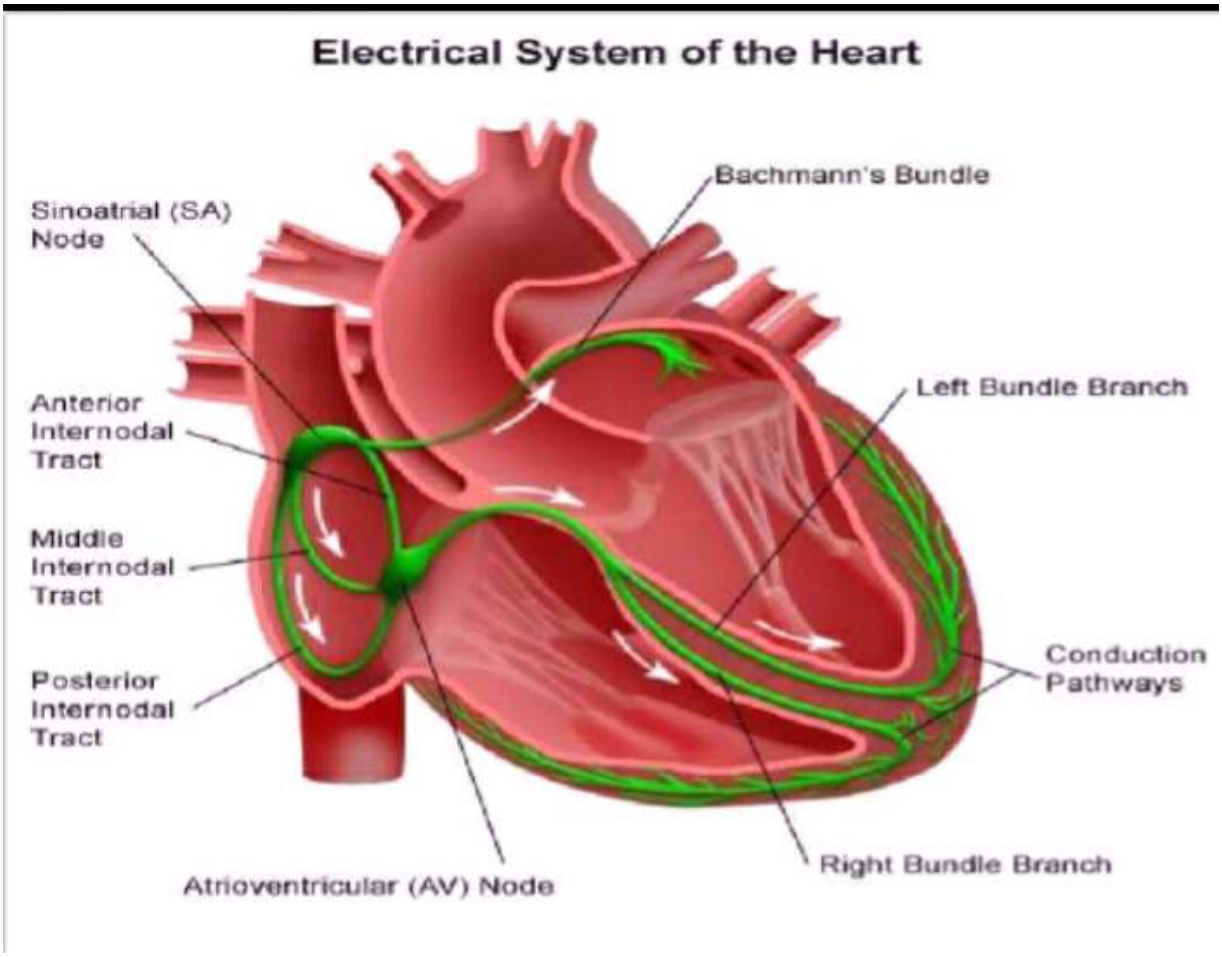


Fig.4.9.Normal conduction system

Fig.4.10.Sinus Bradycardia

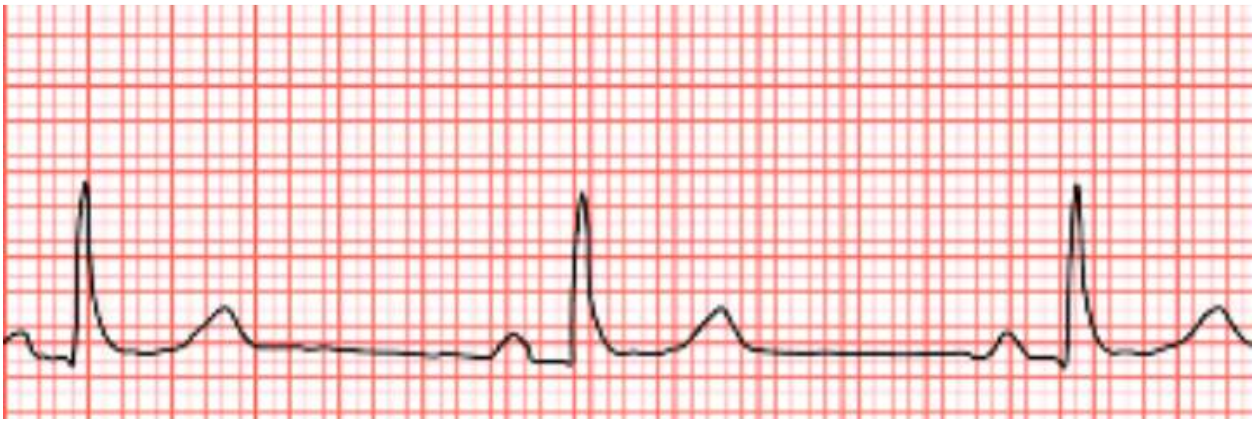


Fig.4.11.First Degree Heart Block

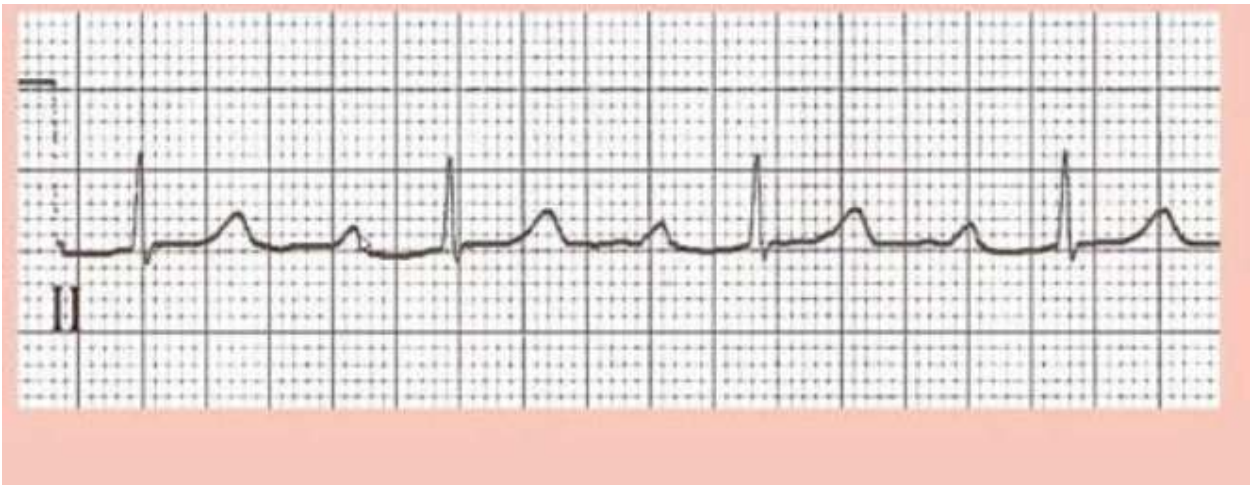
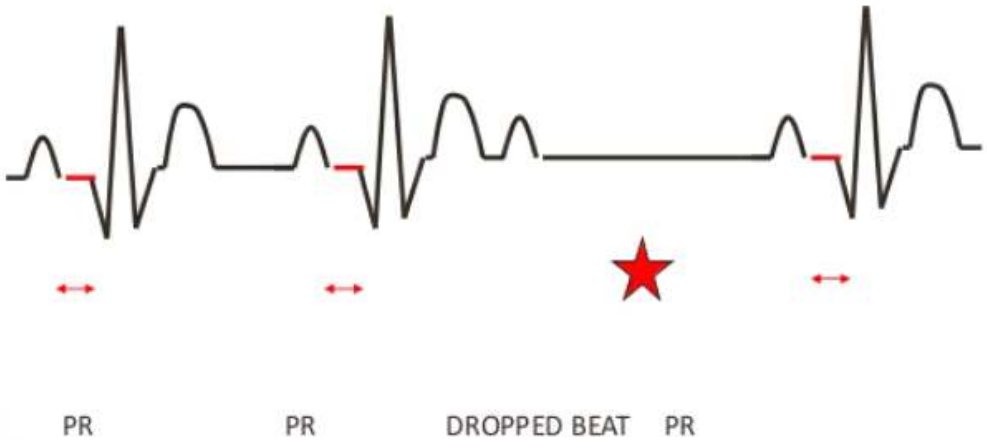


Fig.4.12.Second Degree Heart Block



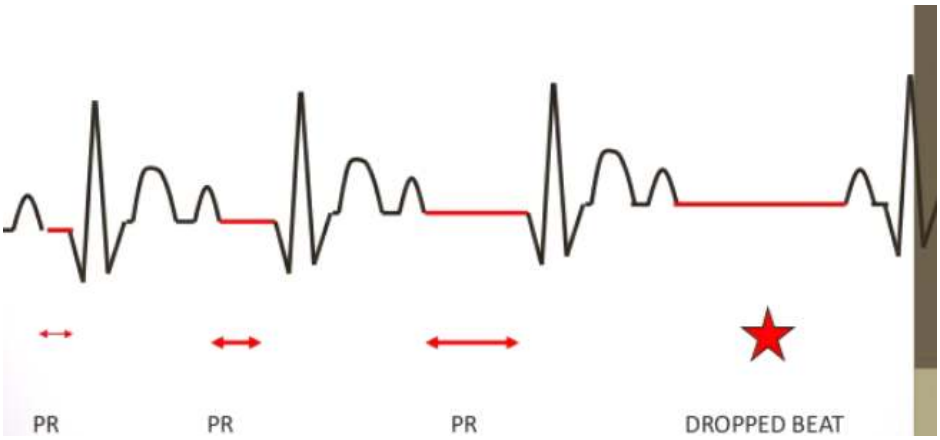
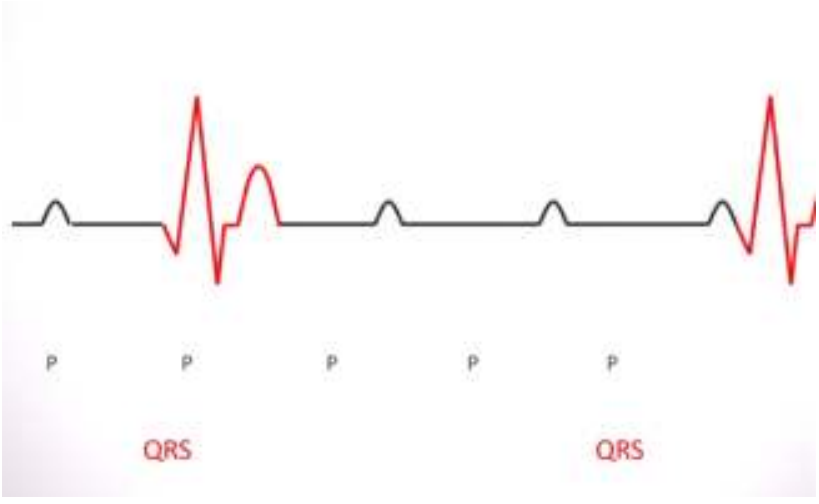


Fig 4.13 COMPLETE HEART BLOCK



TACHYARRHYTHMIAS:

Premature ventricular complex:

Commenest arrhythmia. This is very important because this may be fore runner of ventricular tachycardia, ventricular fibrillation. This can be treated with IV lignocaine 100 mg bolus.

Ventricular Tachycardia:

This is life threatening complication after myocardial infarction. This can be treated with lignocaine, amiodarone or mexilitene. If medical management fails DC shock of 150-200 joules is effective. Sometimes pacemaker may be effective.

Ventricular Fibrillation:

Mostly patient is pulseless . Defibrillation should be done by DC shock of 200-400 joules. If patient had repeated episode means intravenous amiodarone, mexilitene or lignocaine.

Supraventricular Arrhythmias:

Sometimes atrial fibrillation, atrial flutter may occur. This can be treated with verapamil or digoxin.

POST INFARCTION ISCHEAMIA:

It may occur upto 50% of patients. due to residual stenosis in infarct related vessel despite successful thrombolysis

ACUTE CIRCULATORY FAILURE:

This results in cardiogenic shock which carries bad prognosis. If inferior wall myocardial infarction is occur there may associated right ventricular myocardial infarction, posterior wall myocardial infarction. Patient went for severe hypotension and cardiogenic shock.

PERICARDITIS:

It is common in second and third day of myocardial infarction. That is recognized by worsening of chest pain on inspiration. Dressler's syndrome which occur 2 weeks and 3 months after myocardial infarction. It is due to autoimmune effect usually accompanied by pleural and pericardial effusions. Usually treated by steroids.

RIGHT VENTRICULAR MYOCARDIAL INFARCTION AND POSTERIOR WALL MYOCARDIAL INFARCTION:

It is the important complications of inferior wall myocardial infarction. If coronary artery occlusion involves right coronary artery means there may be associated right ventricular and posterior wall myocardial infarction may occur.

OTHER COMPLICATIONS:

Mitral Regurgitation

Mainly due to rupture of papillary muscle .if it is trivial no clinical significance. But it is severe mitral regurgitation means produce life threatening complications like left ventricular failure and cardiogenic shock.

Ventricular Septal Defect:

Due to rupture of infracted interventricular system. It produce pansystolic murmur in left sterna border. It may need immediate surgical intervention. Left ventricular aneurysm and thrombo embolism are other complications.

TREATMENT:

a) Oxygen may be given to improve oxygen saturation of myocardium.

b) For chest pain inj. morphine sulphate is the drug of choice. Then nitrates and beta blockers also be used.

c) Anti platelets like aspirin should be given.

d) Subcutaneous heparin 5000u 6^{th} hourly should be given.

e) Streptokinase

It should be given for acute myocardial infarction if window period less than 12 hours.

f) Early administration of beta blockers and angiotensin converting enzyme inhibitors in acute myocardial infarction reduce the morbidity and mortality

g) Anti arrhythmic drugs may be used in a case of ventricular tachyarrhythmias. Patients with supra ventricular may be treated with beta blockers and digoxin.

h) Reperfusion therapy:

Medically treated with inj.streptokinase. surgically by percutaneous coronary angioplasty.in elderly patients medical management is preffered.

i) Diuretics may be used if patient in failure.

j) Pacemackers:

Indications are acquired atrio ventricular block like complete heart block, bradycardia with symptoms presumed to be due to AV block, second degree AV block with symptomatic bradycardia and marked first degree AV block (>0.30 sec)in patients with left ventricular dysfunction and symptoms of congestive cardiac failure and after acute myocardial infarction.

LONG TERM TREATMENT:

Modifiable risk factors like smoking, alcohol, hypertension and diabetes should be controlled. long term drug therapy like aspirin, clopidogrel, nitrates, diuretics, beta blockers and angiotensin converting enzyme inhibitors should be use.

OBSERVATIONS AND RESULTS

The study populations consists of 200 patients who had fulfilled inclusion and exclusion criteria.

Age and Sex of study population was depicted below. Most of the cases were > 50 years with male predominant

Table.5.1.Sex Distribution in the Study

SEX	NO OF PATIENTS	PERCENTAGE
MALE	152	76%
FEMALE	48	24%

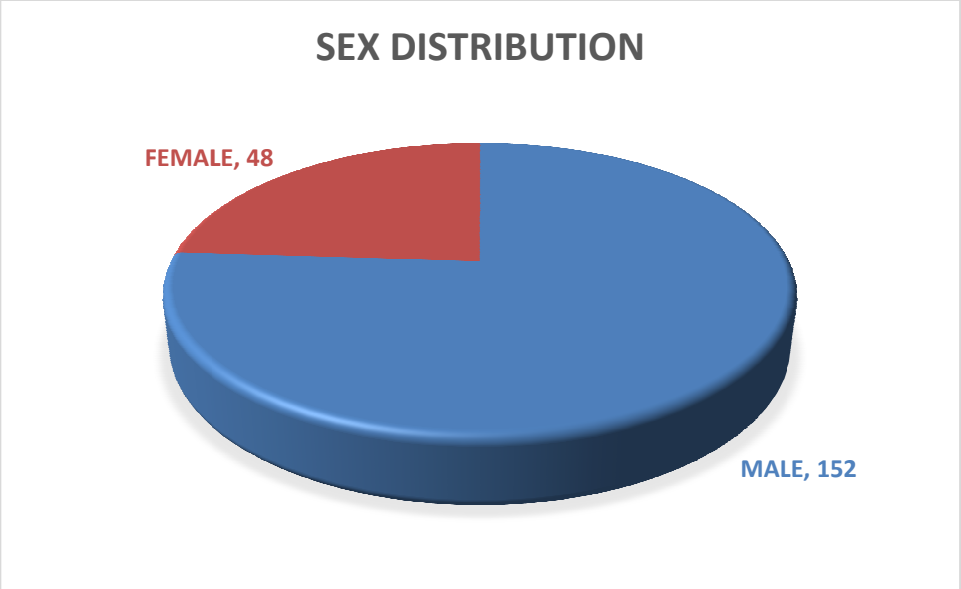


Chart.5.1.Sex distribution in the study

Table.5.2.Age Distribution in the Study

AGE (IN YEARS)	NO OF PATIENTS	PERCENTAGE
< 30	9	4.5%
31-40	16	8%
41-50	20	10%
51-60	69	34.5%
61-70	74	37%
> 70	12	6%

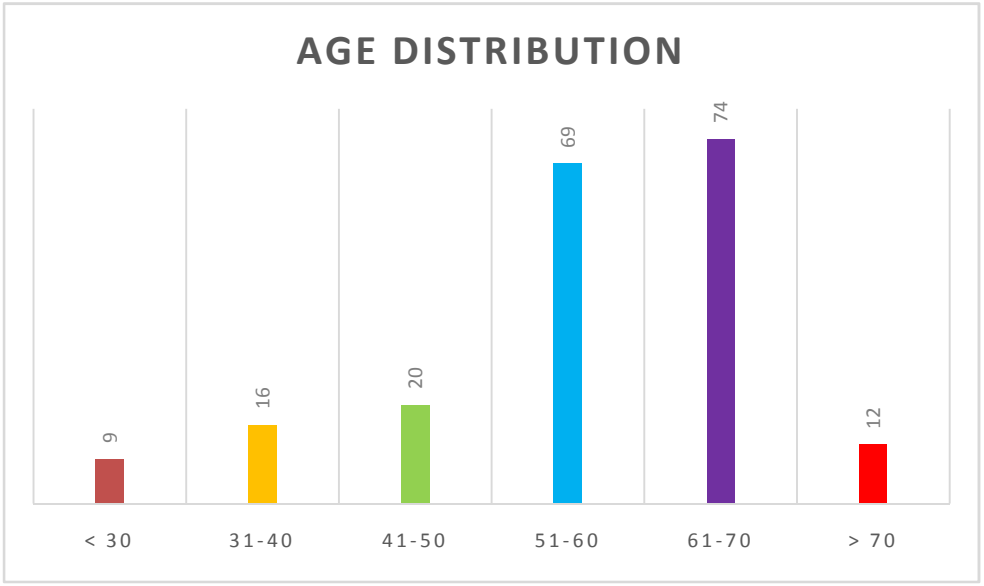


Chart.5.2.Age Distribution in the study

Table.5.3.Smoking percentage in the study

SMOKING	NO OF PATIENTS	PERCENTAGE
PRESENT	124	62%
ABSENT	76	38%

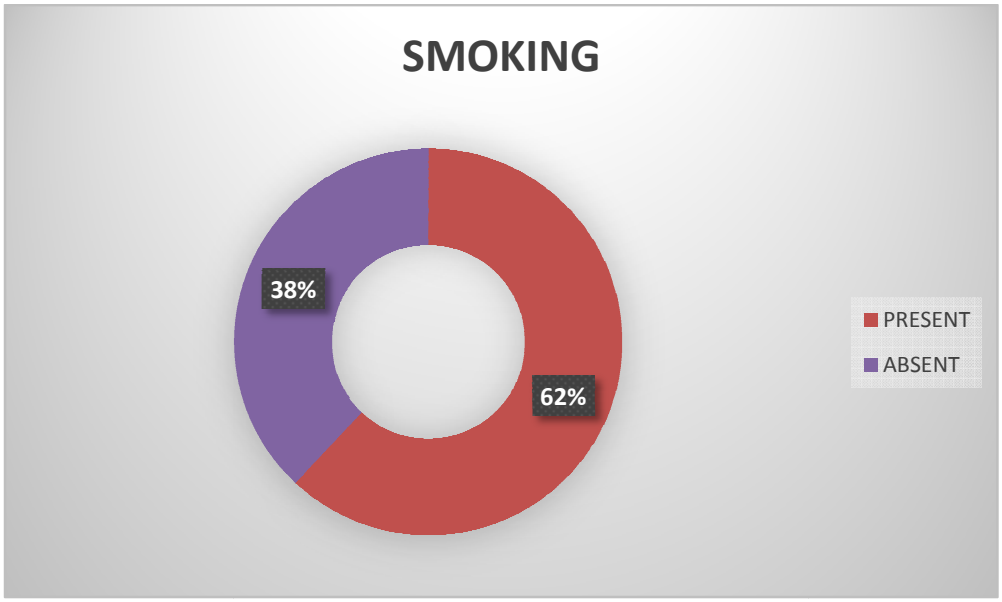


Chart.5.3. Smoking percentage in the study

Study shows 62%of the patient had smoking history.

Table.5.4.Alcohol percentage in the study

ALCOHOL INTAKE	NO OF PATIENTS	PERCENTAGE
PRESENT	111	55.5%
ABSENT	89	44.5%

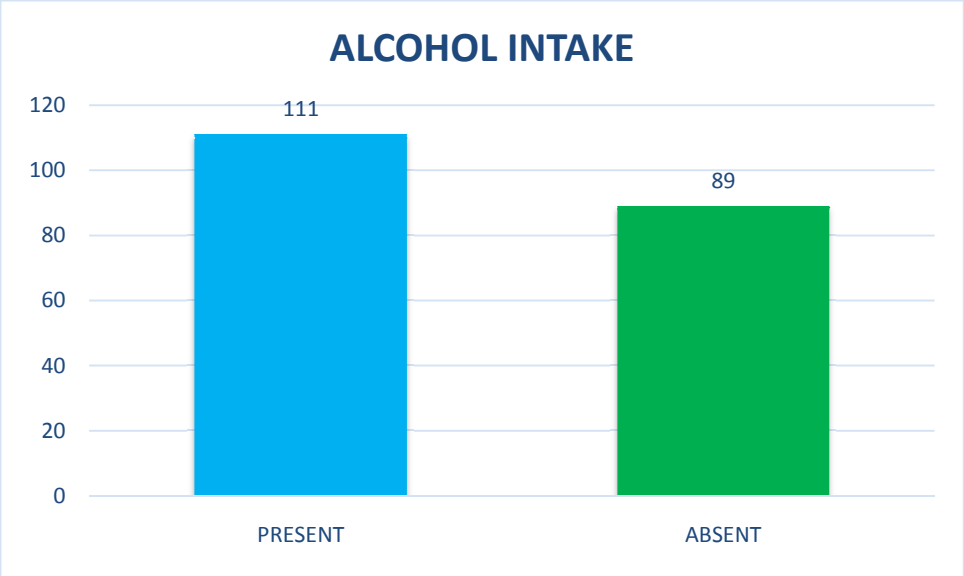


Chart.5.4.Alcohol percentage in the study

Study shows 55.5% had positive history of alcoholism

Table.5.5.Hypertension percentage in the study

HYPERTENSION	NO OF PATIENTS	PERCENTAGE
PRESENT	135	67.5%
ABSENT	65	32.5%

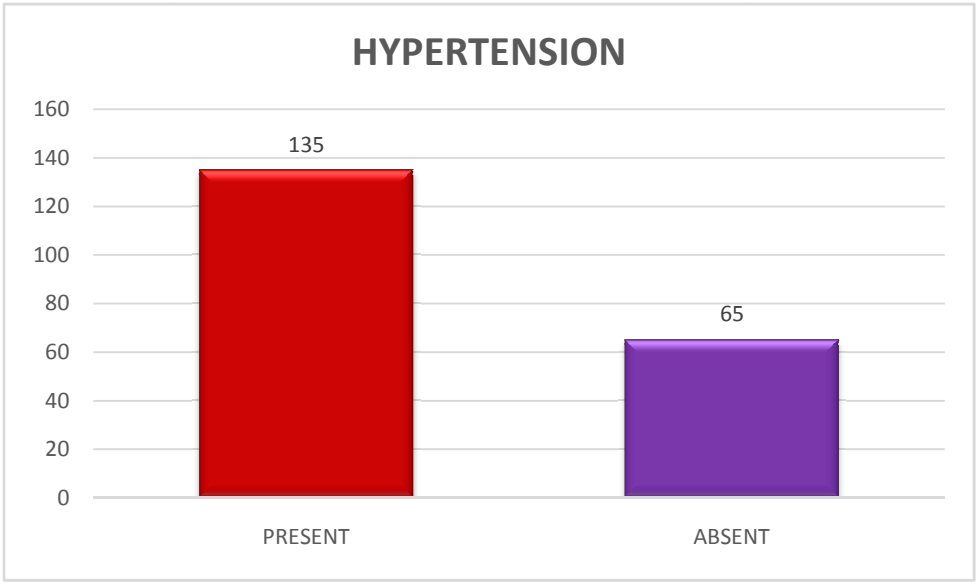


Chart.5.5.Hypertensive patients in the study

Study shows 67.5% patient had positive history of hypertension

Table.5.6.Diabetic percentage in the study

DIABETES MELLITUS	NO OF PATIENTS	PERCENTAGE
PRESENT	127	63.5%
ABSENT	73	36.5%

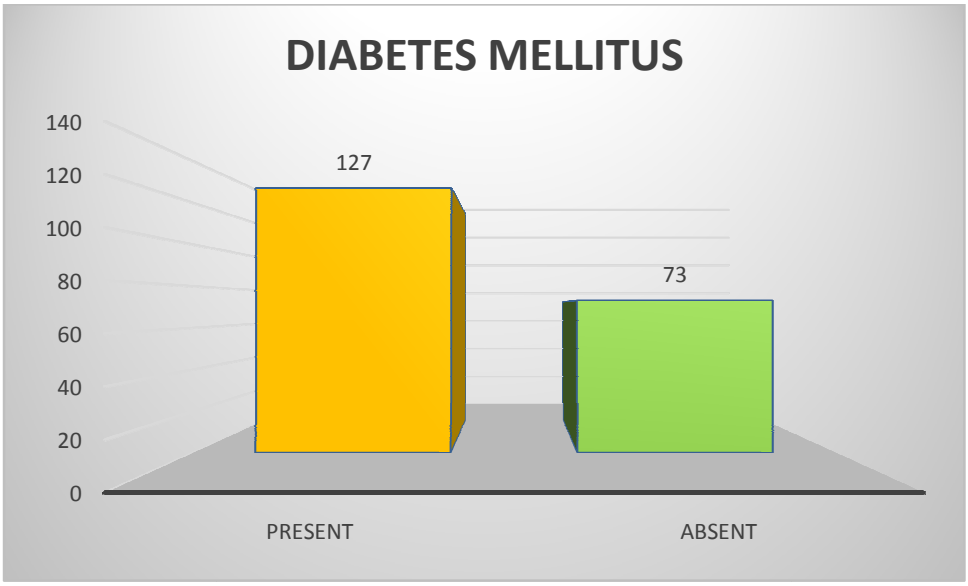


Chart.5.6.Diabetic patients in the study

Study shows 63.5% patient had positive history of Diabetes mellitus

Table.5.7.Cholesterol level in the study

TOTAL CHOLESTEROL	NO OF PATIENTS	PERCENTAGE
HIGH	104	52%
NORMAL	96	48%

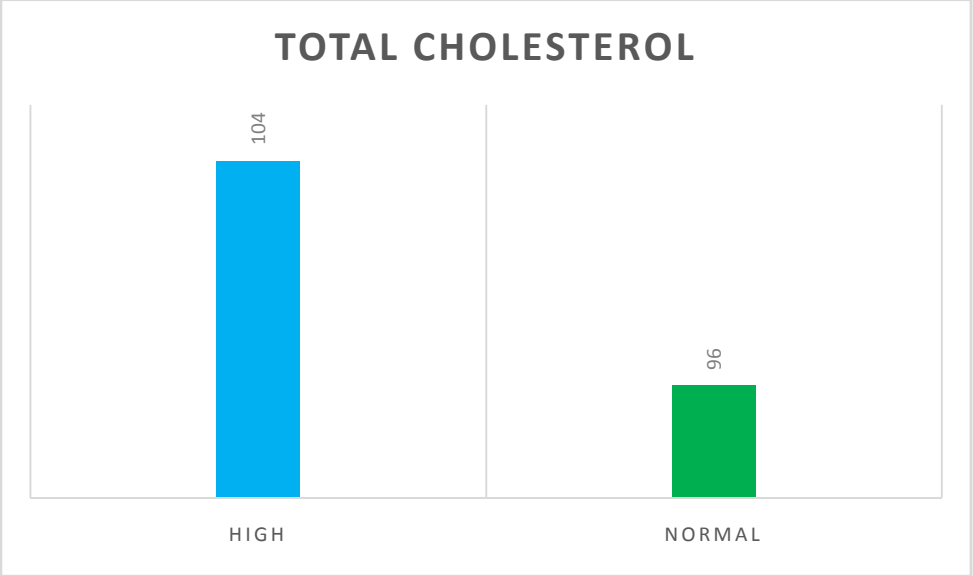


Chart.5.7.Cholesterol level in the study

Study shows 52% patient had high total cholesterol level

Table.5.8.HDL level in the study

HDL	NO OF PATIENTS	PERCENTAGE
LOW	105	52.5%
NORMAL	95	47.5%

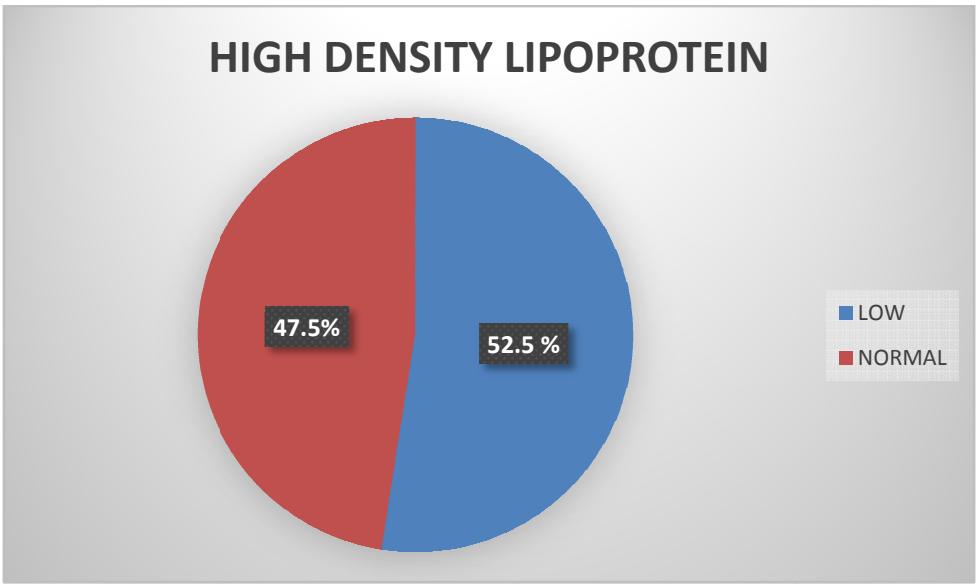


Chart.5.8. HDL level in the study

Study shows 52.5% patient had low HDL level

Table.5.9.LDL level in the study

LDL	NO OF PATIENTS	PERCENTAGE
HIGH	104	52%
NORMAL	96	48%

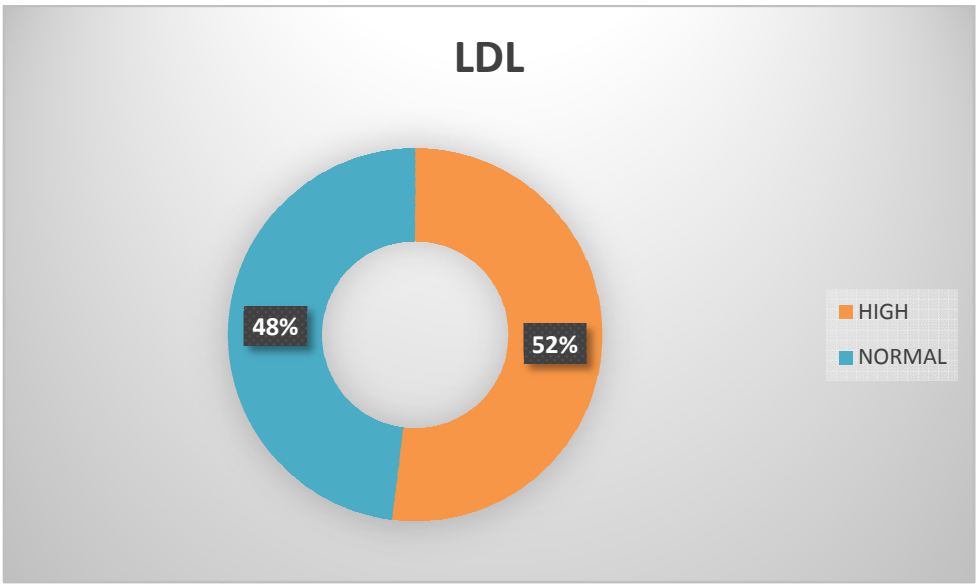


Chart.5.9.LDL level in the study

Study shows 52% of patient had high LDL level

Table.5.10.COPD patients in the study

COPD	NO OF PATIENTS	PERCENTAGE
PRESENT	55	27.5%
ABSENT	145	72.5%

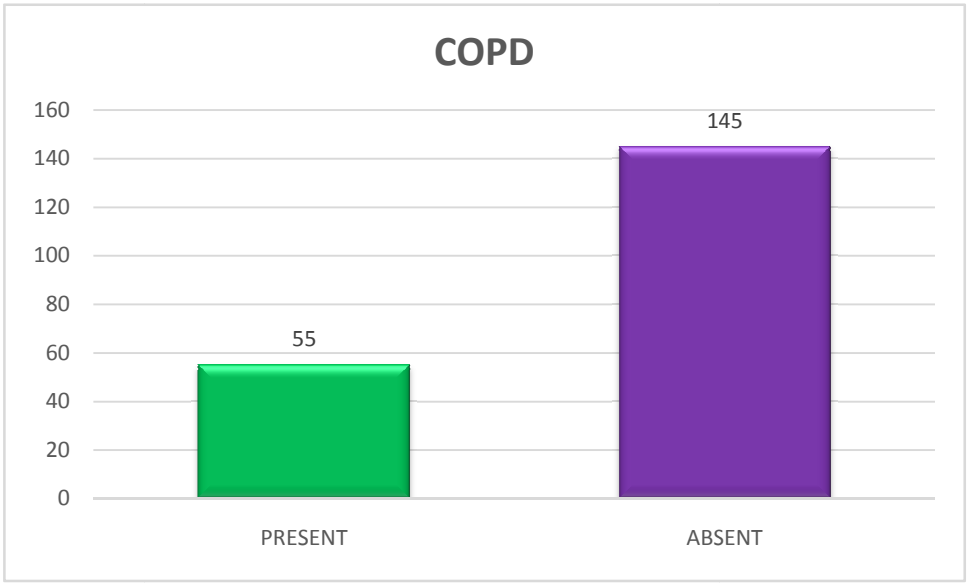


Chart.5.10.COPD patients in the study

Study shows 27.5% of patient had positive history of COPD

Table.5.11.Conduction Blocks in the study

CONDUCTION DEFECT	NO OF PATIENTS	PERCENTAGE
1° HEART BLOCK	69	34.5%
2° HEART BLOCK	17	8.5%
COMPLETE HEART BLOCK	32	16%
SINUS BRADYCARDIA	82	41%

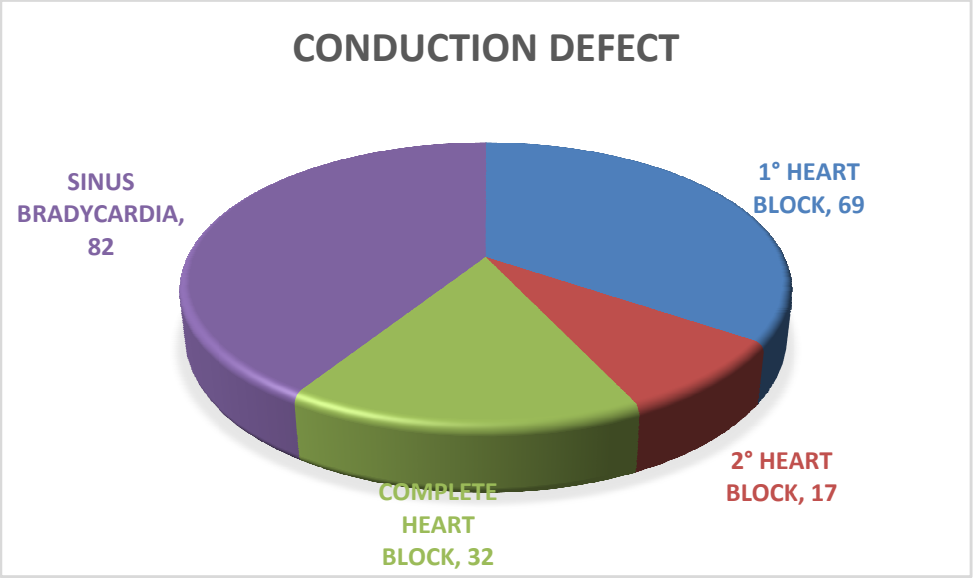


Chart.5.11.Conduction Blocks in the study

Study shows sinus bradycardia is the most common conduction defects followed by first degree heart block .

Table.5.12.Prognosis of the patients in the study

PROGNOSIS	NO OF PATIENTS	PERCENTAGE
DEATH	23	11.5%
ALIVE	177	88.5%

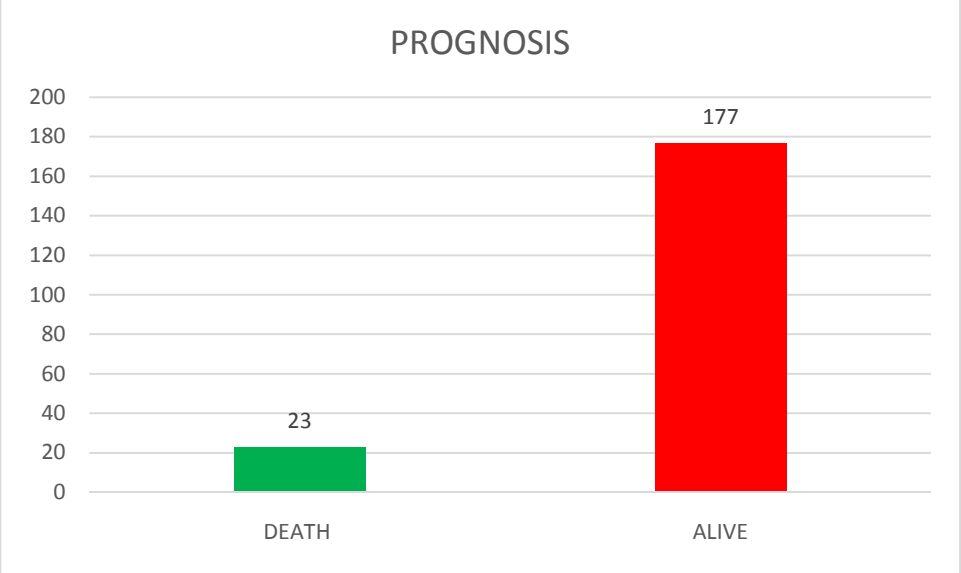


Chart.5.12.Prognosis of the patients in the study

Study shows 11.5% of patient were died

Table.5.13.Correlation between conduction defects and sex

	SEX	
CONDUCTION DEFECT	MALE	FEMALE
1° HEART BLOCK	55	14
2° HEART BLOCK	15	2
COMPLETE HEART BLOCK	26	6
SINUS BRADYCARDIA	56	26
KRUSKAL WALLIS TEST		
P VALUE - 0.167		
NON SIGNIFICANT		

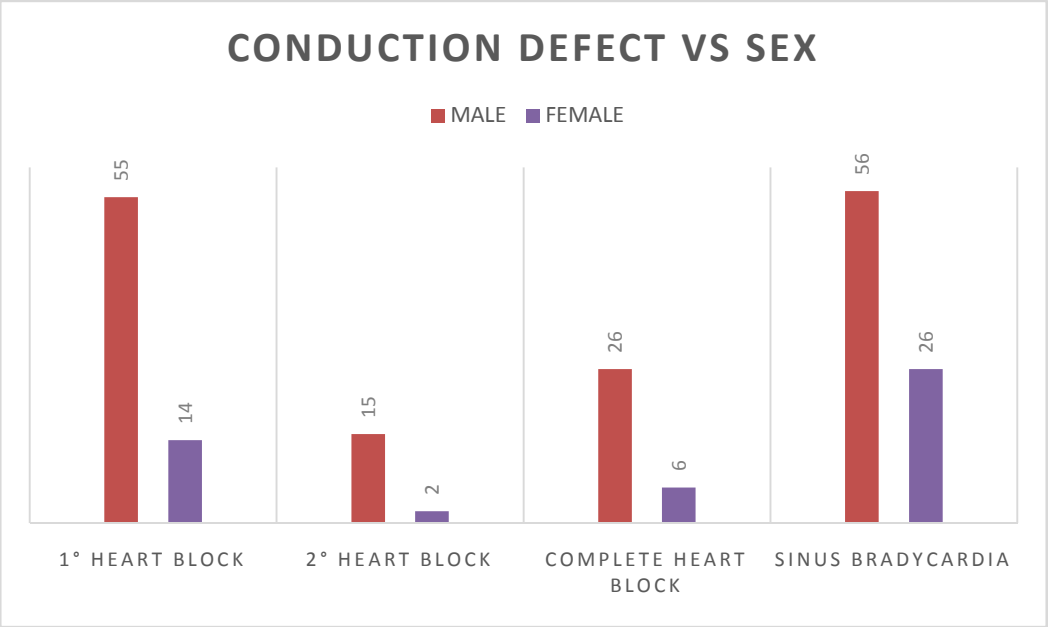


Chart.5.13.Correlation between conduction defects and sex

Study shows no significant correlation between conduction disturbances and sex.

Table 5.14.Correlation between conduction defects and age

AGE (IN YEARS)	1° HEART BLOCK	2° HEART BLOCK	CHB	SB
< 30	2	1	1	5
31-40	3	1	2	10
41-50	7	4	2	7
51-60	29	4	10	20
61-70	23	7	14	30
> 70	5	0	3	4
P VALUE - 0.626				
NON SIGNIFICANT				
KRUSKAL WALLIS TEST				

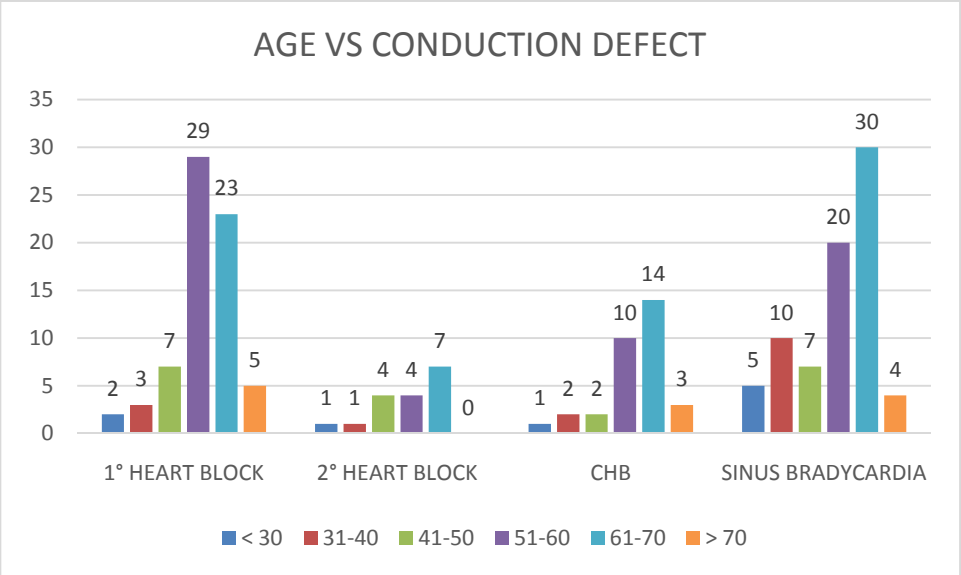


Chart.5.14.Correlation between conduction defects and age

Study shows conduction defects are more common in above 50 years of age

Table.5.15.Correlation between conduction defects and smoking

	SMOKING	
CONDUCTION DEFECT	**PRESENT**	**ABSENT**
1° HEART BLOCK	42	27
2° HEART BLOCK	12	5
COMPLETE HEART BLOCK	23	9
SINUS BRADYCARDIA	47	35
KRUSKAL WALLIS TEST		
P VALUE - 0.448		
NON SIGNIFICANT		

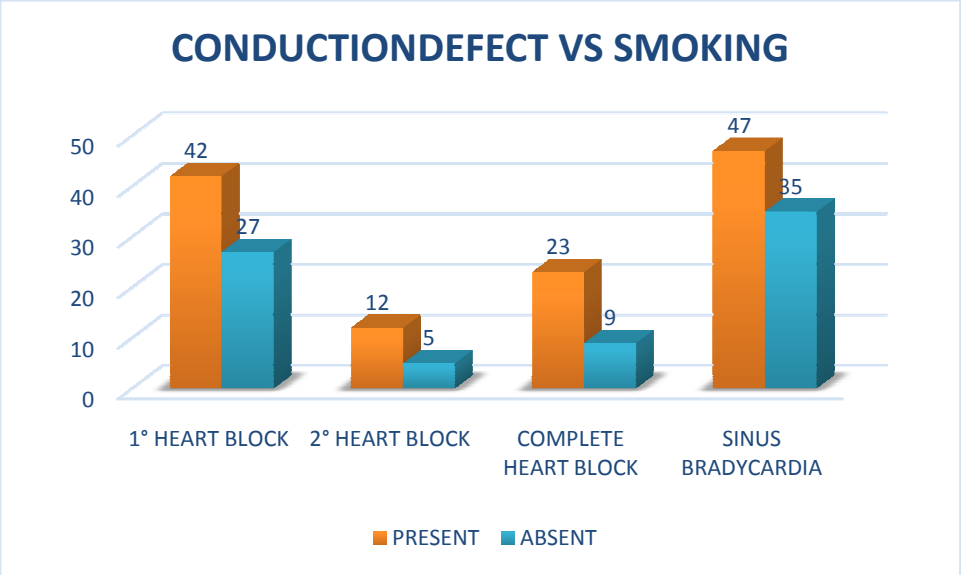


Chart.5.15.Correlation between conduction defects and smoking

Study shows there is no significant correlation between conduction defects and smoking.

Table.5.16.Correlation between conduction defects and Alcohol

	ALCOHOL INTAKE	
CONDUCTION DEFECT	**PRESENT**	**ABSENT**
1° HEART BLOCK	38	31
2° HEART BLOCK	9	8
COMPLETE HEART BLOCK	19	13
SINUS BRADYCARDIA	45	37
KRUSKAL WALLIS TEST		
P VALUE - 0.167		
NON SIGNIFICANT		

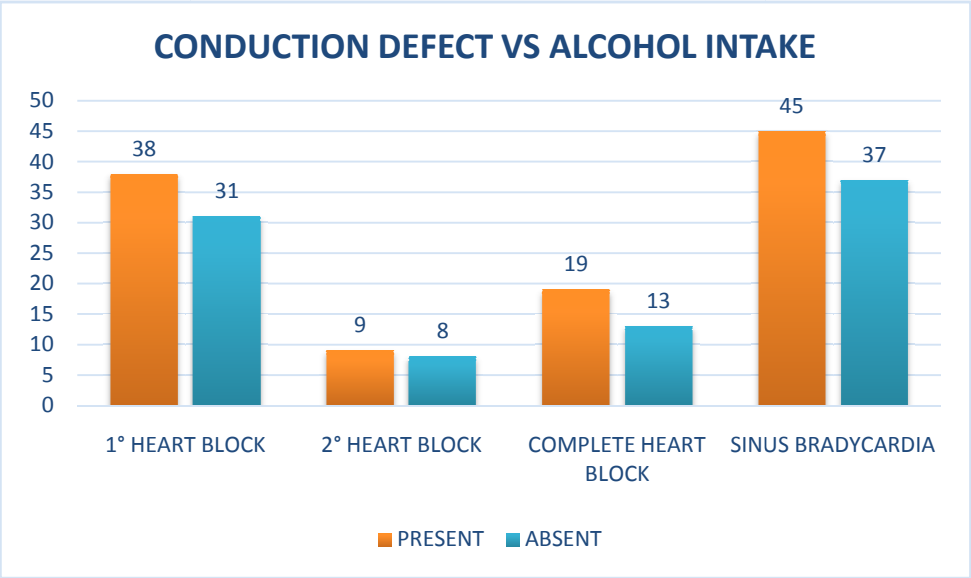


Chart.5.16.Correlation between conduction defects and Alcohol

Study shows no significant correlation between conduction defects and alcoholism.

Table.5.17.Correlation between conduction defects and Hypertension

	HYPERTENSION	
CONDUCTION DEFECT	**PRESENT**	**ABSENT**
1° HEART BLOCK	51	18
2° HEART BLOCK	12	5
COMPLETE HEART BLOCK	22	10
SINUS BRADYCARDIA	50	32
KRUSKAL WALLIS TEST		
P VALUE - 0.039		
SIGNIFICANT		

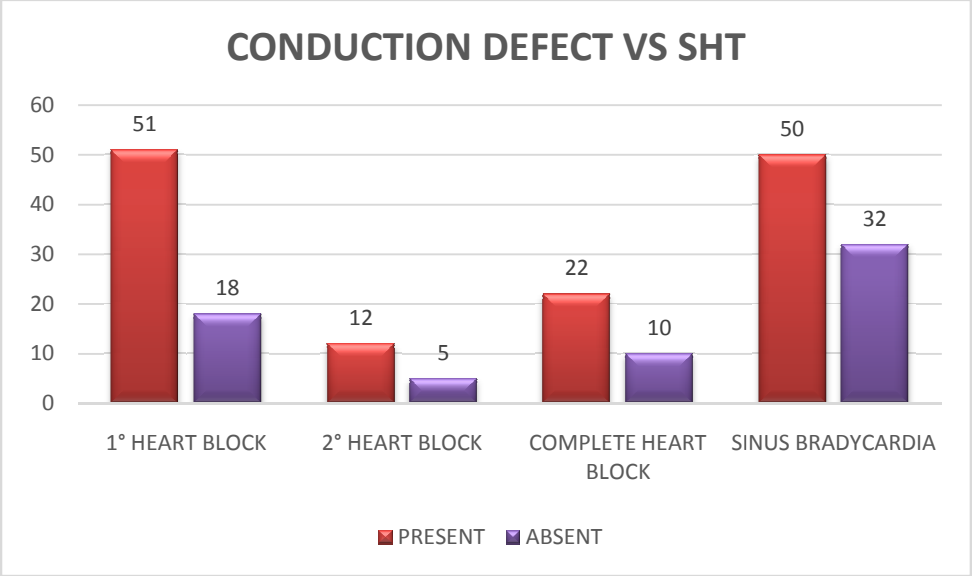


Chart.5.17.Correlation between conduction defects and Hypertension

Study shows significant correlation between conduction defects and Hypertension

Table.5.18.Correlation between conduction defects and Diabetes mellitus

	DIABETES MELLITUS	
CONDUCTION DEFECT	**PRESENT**	**ABSENT**
1° HEART BLOCK	43	26
2° HEART BLOCK	12	5
COMPLETE HEART BLOCK	16	16
SINUS BRADYCARDIA	56	26
KRUSKAL WALLIS TEST		
P VALUE - 0.029		
SIGNIFICANT		

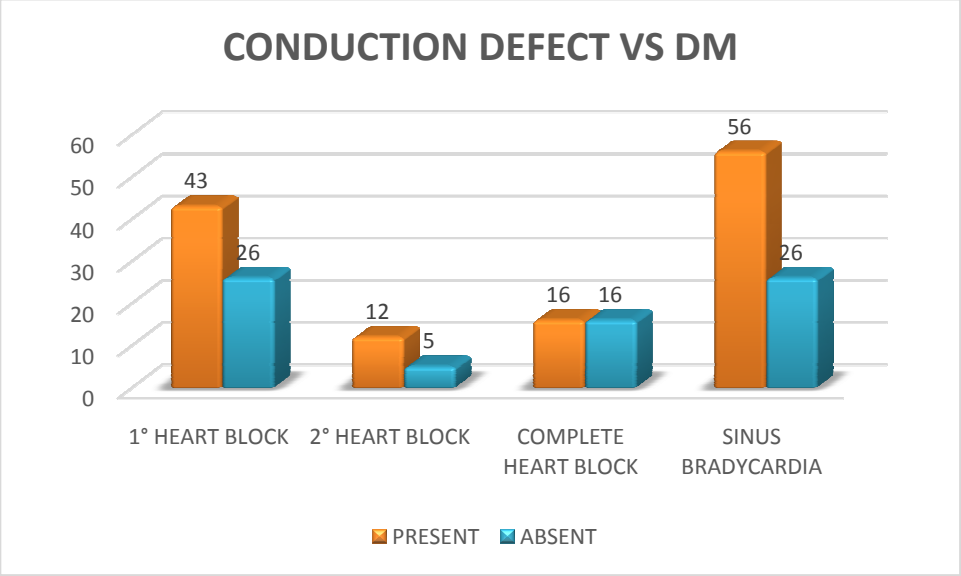


Chart.5.18.Correlation between conduction defects and Diabetes mellitus

Study shows significant correlation between conduction defects and Diabetes mellitus

Table.5.19.Correlation between conduction defects and cholesterol

	TOTAL CHOLESTEROL	
CONDUCTION DEFECT	**HIGH**	**NORMAL**
1° HEART BLOCK	33	36
2° HEART BLOCK	7	10
COMPLETE HEART BLOCK	21	11
SINUS BRADYCARDIA	43	39
KRUSKAL WALLIS TEST		
P VALUE - 0.301		
NON SIGNIFICANT		

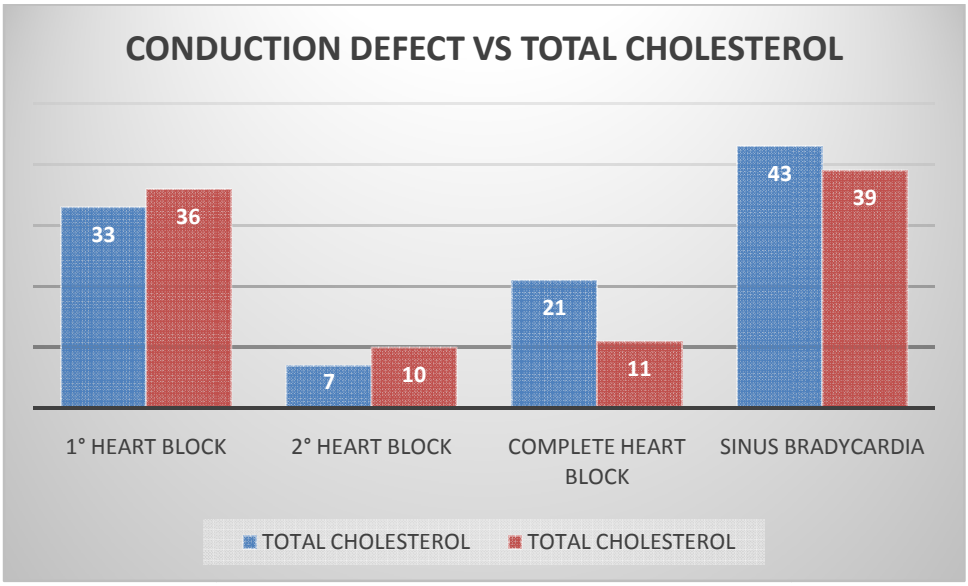


Chart.5.19.Correlation between conduction defects and cholesterol

Study shows no significant correlation between conduction defects and cholesterol level

Table.5.20.Correlation between conduction defects and HDL level

	HIGH DENSITY LIPOPROTEIN	
CONDUCTION DEFECT	**LOW**	**NOMAL**
1° HEART BLOCK	34	35
2° HEART BLOCK	7	10
COMPLETE HEART BLOCK	21	11
SINUS BRADYCARDIA	43	39
KRUSKAL WALLIS TEST		
P VALUE - 0.338		
NON SIGNIFICANT		

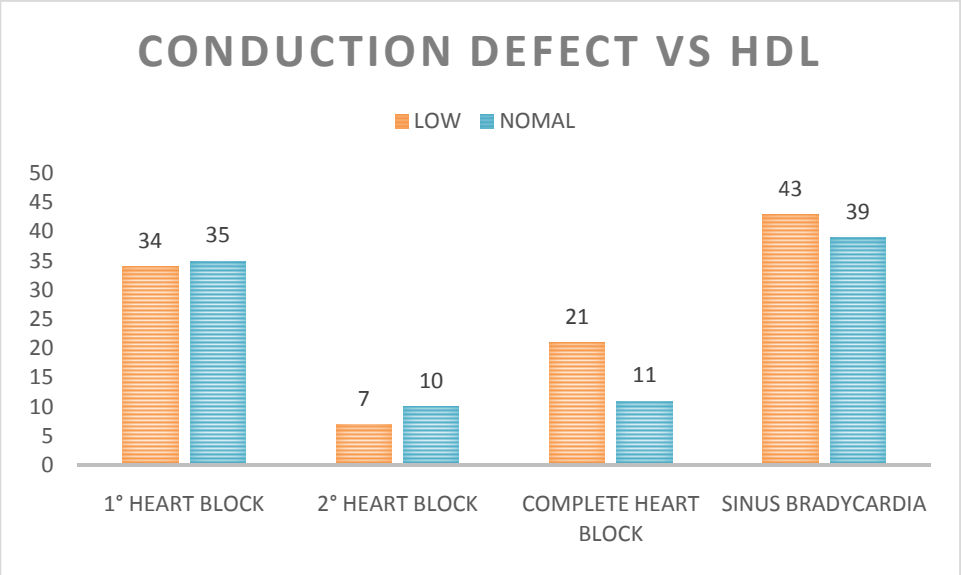


Chart.5.20.Correlation between conduction defects and HDL level

Study shows no significant between conduction defects and HDL level

Table.5.21.Correlation between conduction defects and LDL level

	LDL	
CONDUCTION DEFECT	**HIGH**	**NORMAL**
1° HEART BLOCK	34	35
2° HEART BLOCK	7	10
COMPLETE HEART BLOCK	21	11
SINUS BRADYCARDIA	42	40
KRUSKAL WALLIS TEST		
P VALUE - 0.489		
NON SIGNIFICANT		

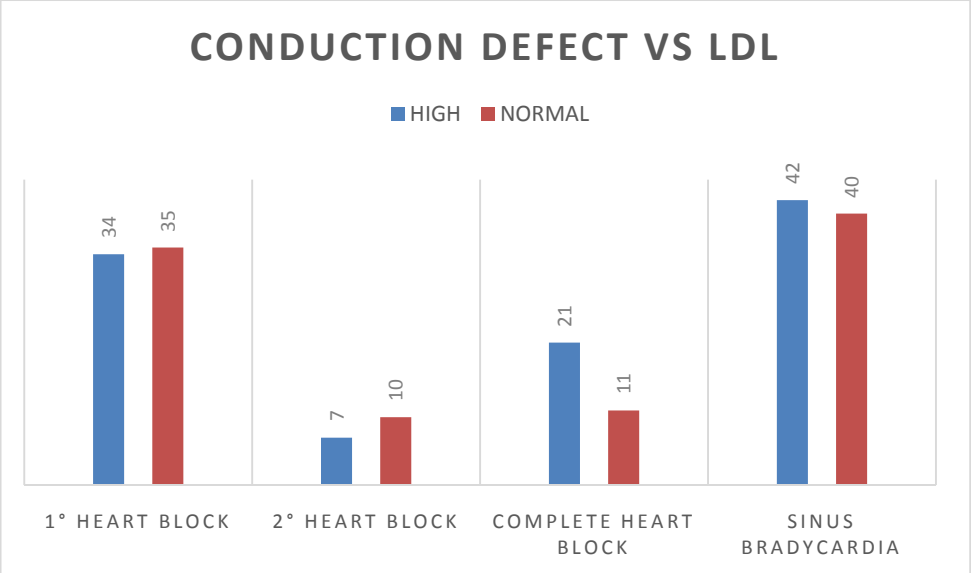


Chart.5.21.Correlation between conduction defects and LDL level

Study shows no significant correlation between conduction defects and LDL level

Table.5.22.Correlation between Conduction defects and COPD Patients

	COPD	
CONDUCTION DEFECT	**PRESENT**	**ABSENT**
1° HEART BLOCK	18	51
2° HEART BLOCK	6	11
COMPLETE HEART BLOCK	10	22
SINUS BRADYCARDIA	21	61
KRUSKAL WALLIS TEST		
P VALUE - 0.811		
NON SIGNIFICANT		

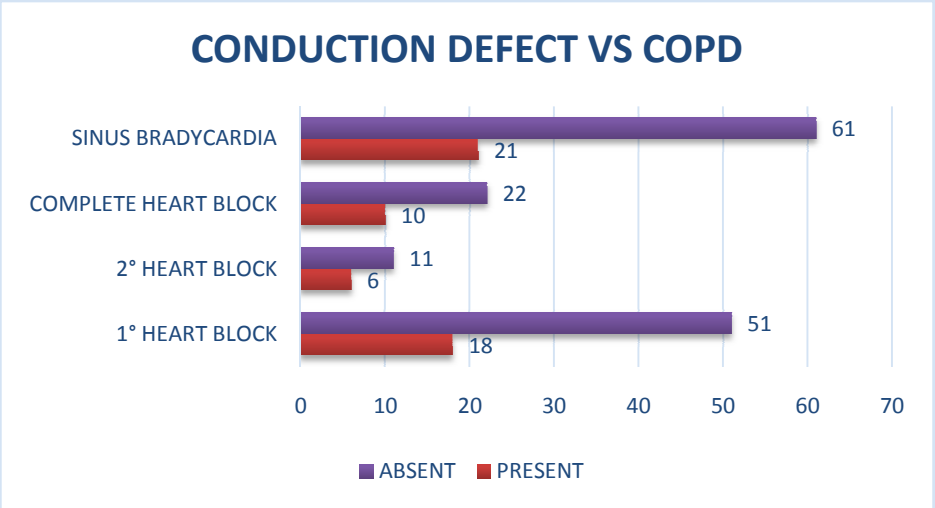


Chart.5.22.Correlation between Conduction defects and COPD Patients

Study shows no significant correlation between conduction disturbances and COPD

Table.5.23.Correlation between conduction defects and prognosis of the patient

	PROGNOSIS	
CONDUCTION DEFECT	**DEATH**	**ALIVE**
1° HEART BLOCK	6	63
2° HEART BLOCK	3	14
COMPLETE HEART BLOCK	6	26
SINUS BRADYCARDIA	8	74
KRUSKAL WALLIS TEST		
P VALUE - 0.382		
NON SIGNIFICANT		

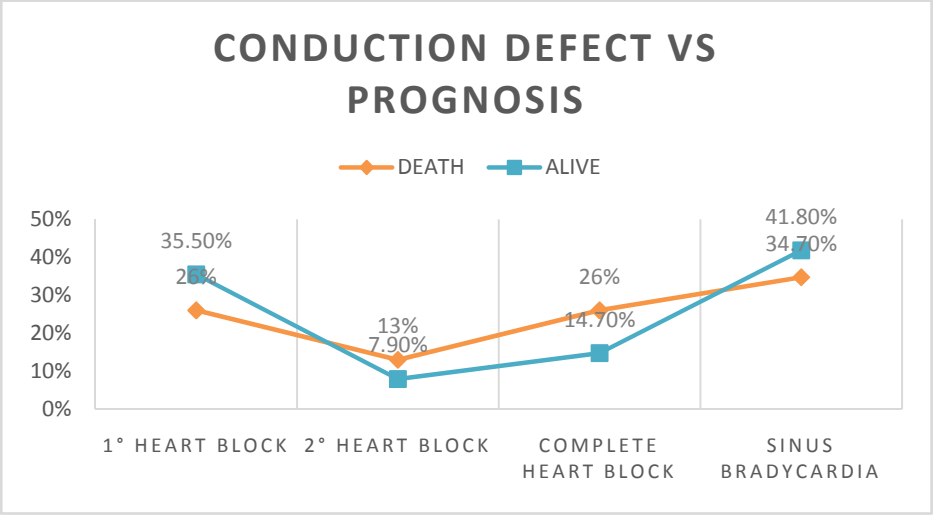


Chart.5.23.Correlation between conduction defects and prognosis of the patient

Study shows no significant correlation between conduction defects and prognosis of patient

Table.5.24.Correlation between mean cholesterol and prognosis of the patient

	CHOLESTEROL	
PROGNOSIS	**MEAN**	**SD**
DEATH	226.83	43.85
ALIVE	182.21	36.62
P VALUE - 0.001		
SIGNIFICANT		
UNPAIRED T TEST		

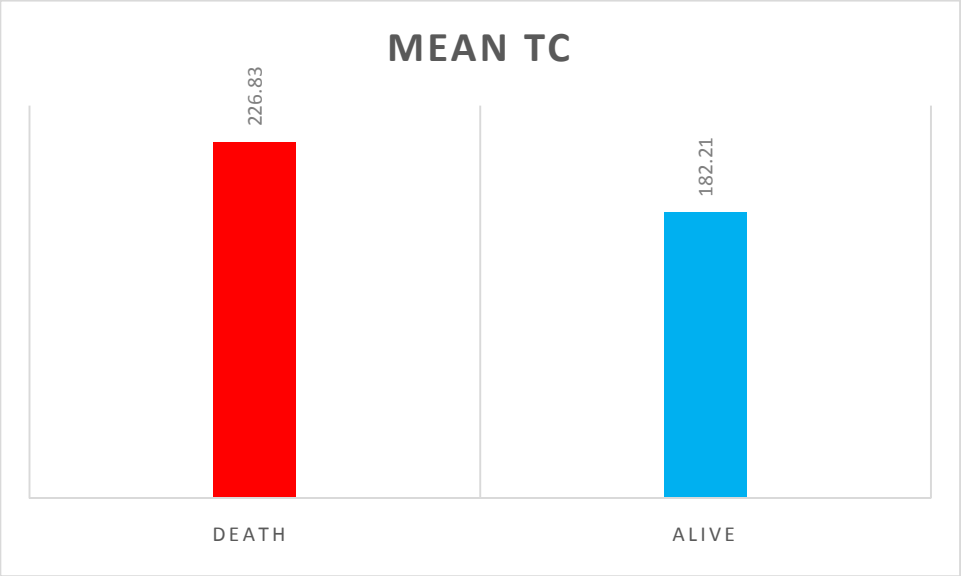


Chart.5.24.Correlation between mean cholesterol and prognosis of the patient

Study shows significant correlation between mean cholesterol and prognosis of patient.

Table.5.25.Correlation between mean HDL and prognosis of the patient

	HDL	
PROGNOSIS	**MEAN**	**SD**
DEATH	40.83	8.09
ALIVE	49.75	7.96
P VALUE - 0.001		
SIGNIFICANT		
UNPAIRED T TEST		

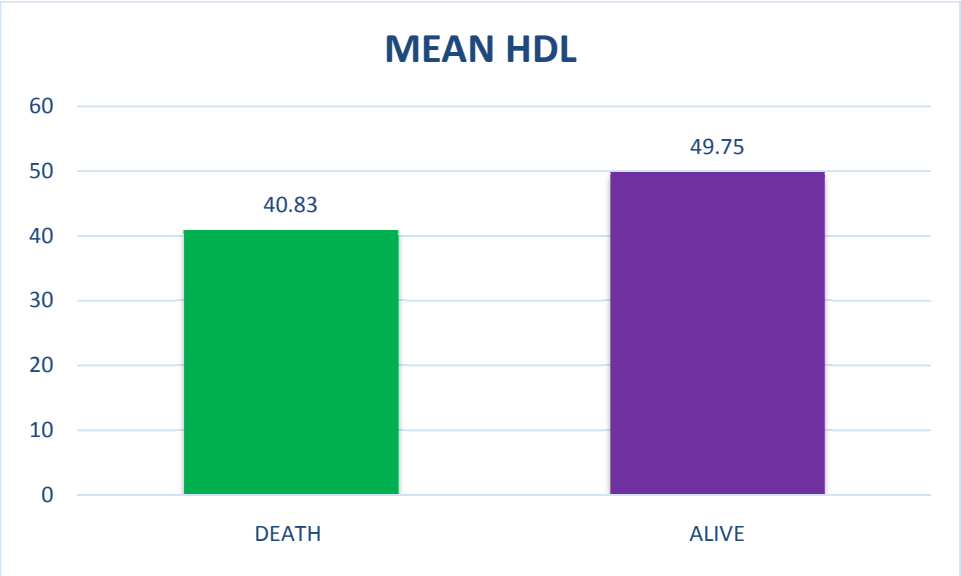


Chart.5.25.Correlation between mean HDL and prognosis of the patient

Study shows significant correlation between mean HDL and prognosis of patient.

Table.5.26.Correlation between mean LDL and prognosis of patient

	LDL	
PROGNOSIS	**MEAN**	**SD**
DEATH	153.39	27.02
ALIVE	123.24	27.44
P VALUE		
SIGNIFICANT		
UNPAIRED T TEST		

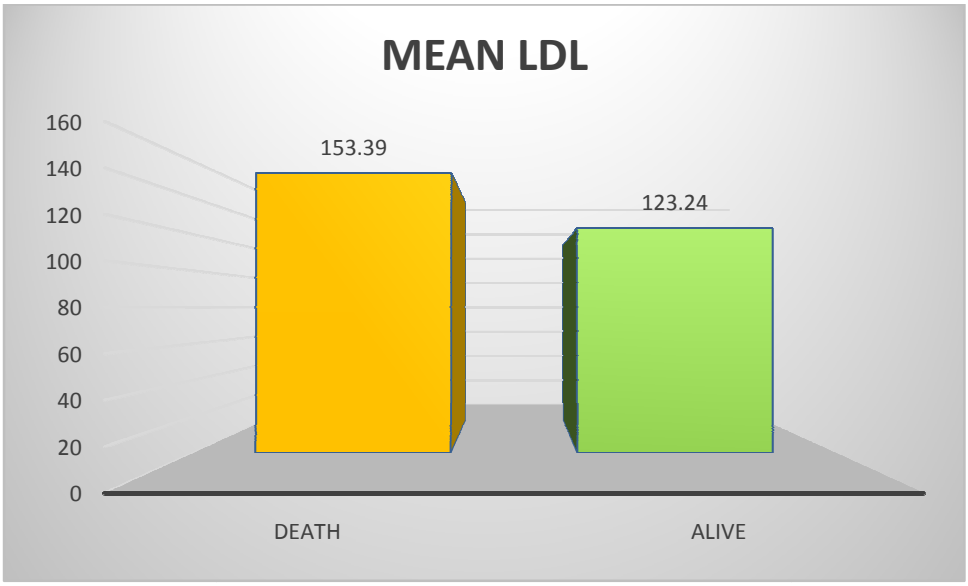


Chart.5.26.Correlation between mean LDL and prognosis of patient

Study shows significant correlation between mean LDL level and prognosis of patient.

Table.5.27. Correlation between mean cholesterol and conduction defects

	TOTAL CHOLESTEROL	
CONDUCTION DEFECT	**MEAN**	**SD**
1° HEART BLOCK	123.74	25.58
2° HEART BLOCK	120.11	30.11
COMPLETE HEART BLOCK	135.91	30.11
SINUS BRADYCARDIA	124.7	24.6
P VALUE - 0.122		
SIGNIFICANT		
ANOVA		

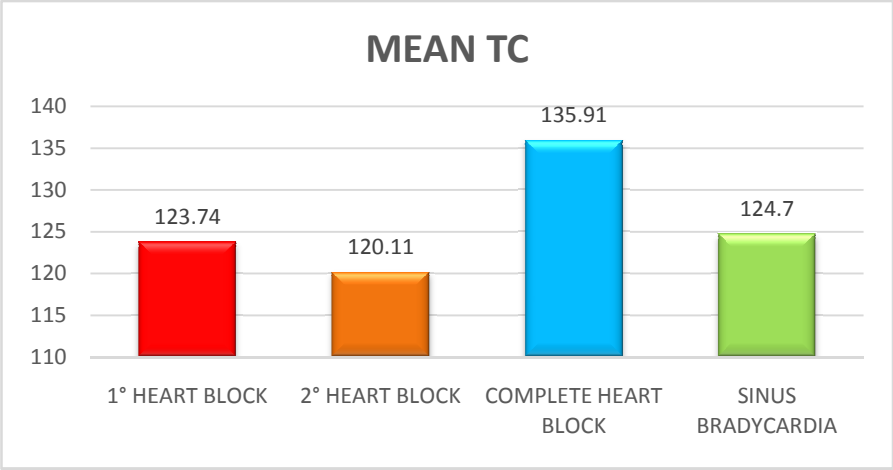


Chart.5.27.Correlation between mean cholesterol and conduction defect

Study shows significant correlation between mean cholesterol and conduction disturbances.

Table.5.28.Correlation between mean HDL level and conduction defects

	HDL	
CONDUCTION DEFECT	**MEAN**	**SD**
1° HEART BLOCK	48.9	8.65
2° HEART BLOCK	49.12	7.69
COMPLETE HEART BLOCK	46.88	9.44
SINUS BRADYCARDIA	49.27	8.08
P VALUE - 0.604		
NON SIGNIFICANT		
ANOVA		

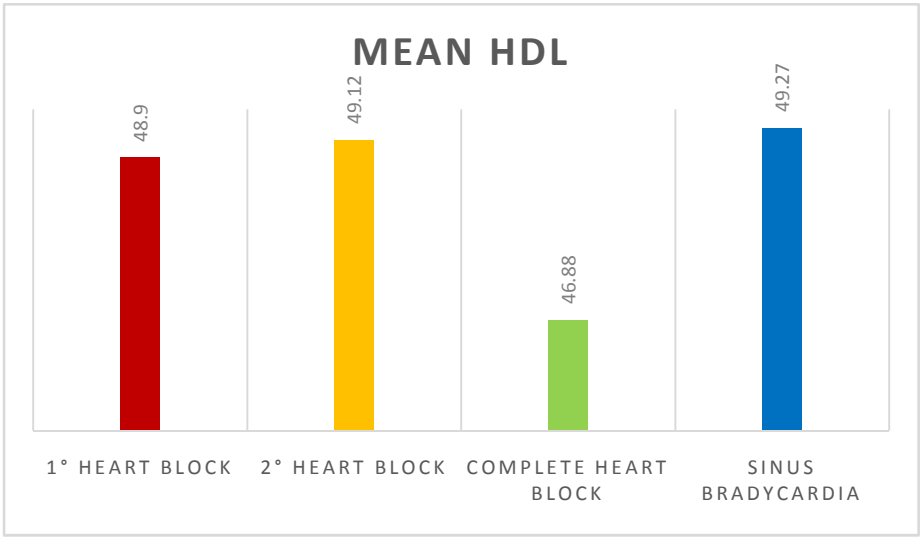


Chart.5.28.Correlation between mean HDL level and conduction defects

Study shows no significant correlation between mean HDL and conduction defects.

Table.5.29.Correlation between mean LDL level and conduction disturbances

	LDL	
CONDUCTION DEFECT	**MEAN**	**SD**
1° HEART BLOCK	183	35.54
2° HEART BLOCK	181	54.86
COMPLETE HEART BLOCK	194	41.78
SINUS BRADYCARDIA	189	39.55
P VALUE -0.481		
NON SIGNIFICANT		
ANOVA		

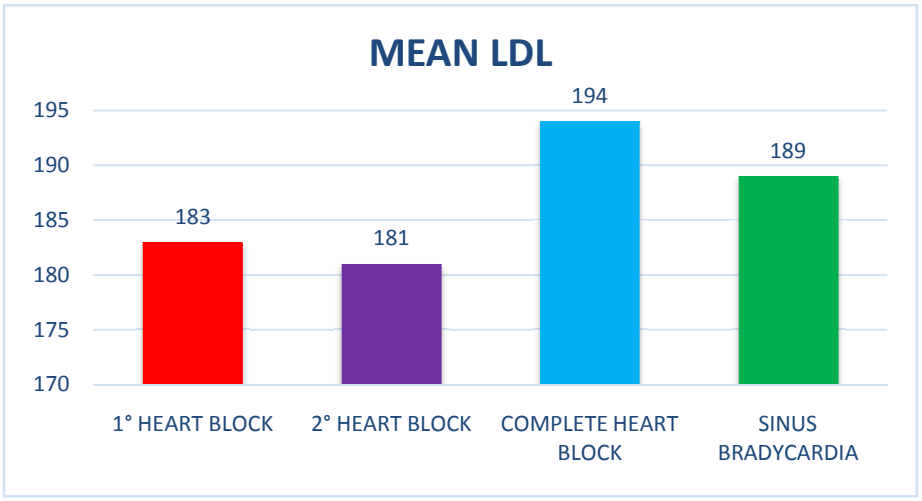


Chart.5.29.Correlation between mean LDL level and conduction disturbances

Study shows no significant correlation between mean LDL and conduction defects

DISCUSSION

AGE:

The present study shows that increasing age the occurrence of conduction disturbances is more. More percentage of conduction disturbance occur between age group of 61-70 years of age. Of the conduction disturbance sinus bradycardia is more common. Because when age increases there is fibrosis of conduction system. This may be because when age increases atherosclerosis is likely to increase. In addition cumulative effect of smoking and the development of hypertension. According to the study conducted by Nimetz et al the mean age was 63 years.

SEX:

In present study there is male preponderance ratio is 4:1.This is because occurrence other risk factor like smoking and alcoholism is more common in males. Study conducted by Godmen et al there was 75% males and 25% females. In this study 76% males and 24% females.

SMOKING:

In this study smoking constitutes 62% of patients. Study shows there is increase incidence of conduction disturbances among smokers .This can be explained by that smoking increase the severity of coronary atherosclerosis and so there is diffuse coronary artery damage among smokers .but statistically is not significant.($p>0.001$).

ALCOHOLISM:

In this study alcoholism constitutes 55.5% of patients. Study shows there is increase occurrence of conduction disturbances among alcoholic groups. But stastically is not significant.($p>0.001$).

HYPERTENSION:

Present study shows 67.5% of patients had hypertension. Study shows increase occurrence of conduction disturbances among hypertension. This can be explained by hypertensive patients had more chance of atherosclerosis. By this more diffuse coronary artery damage. Statistically significant.($p<0.001$).

DIABETESMELLITUS:

In this study 63.5% of patients had diabetes mellitus .study shows increase incidence of conduction disturbances among diabetic patients because diabetic patients had more chance of atherosclerosis by this more diffuse coronary artery damage. statistically significant.($p<001$)

DYSLIPIDEMIA:

Patients who had normal or high total cholesterol level the occurrence of conduction disturbances is not statistically significant. Patients who had normal

or low HDL cholesterol the occurrence of conduction disturbances is not statistically significant. Patients who had normal or high LDL cholesterol level the occurrence of conduction disturbances is not statistically significant when mean value of total cholesterol, HDL and LDL is taken there is direct influence on the prognosis of the conduction disturbances which is stastically significant. But the mean value of dyslipidemia is not influence conduction disturbances which is not statistically significant.

COPD:

In present study there is no correlation between COPD and conduction disturbances. Among the conduction disturbances sinus bradycardia accounts for 41%.first degree heart block accounts for 34.5%.Complete heart block accounts for 16%.Second degree heart block accounts for 8.5%.in the present study sinus bradycardia is the more common conduction disturbances followed by first degree heart block followed by complete heart block and second degree heart block.

The increased incidence of Sinus bradycardia and AV nodal block is more common in inferior wall myocardial infarction are caused by occlusion of dominant artery in more than 70% of cases. AV nodal block is more common due to increased vagal tone due to stimulation of afferent nerves adjacent to the AV node by ischeamia.AV nodal block

may also be due to release of chemical mediators such as potassium and adenosine from ischeamic cells.

Some of the patients with inferior wall myocardial infarction also associated with posterior wall myocardial infarction and right ventricular infarction.In that patient AV nodal involvement is due to coronary artery occlusion proximal to AV nodal artery.

HOSPITAL MORTALITY:

In the present study ,there was 23 (11.5%) patients were died during hospital stay who had conduction defects in acute inferior wall myocardial infarction.0f the 23 patients 8 patients (35%) were died of sinus bradycardia.6 patients (26%) were died of complete heart block.6 patients (26%) were died of first degree heart block.3 patients (13%) were died of second degree heart block more percentage of death occur in a case of sinus bradycardia followed by complete heart block

COMPARISION WITH OTHER STUDIES (Table.6.1)

STUDIES	1st DEGREE(%)	2ND DEGREE (%)	CHB(%)
Michael C Hindan	39	6	19
Scanlan et al	-	-	13.7
Peter Ciemmensen	-	-	13
Tans et al	-	-	-
Boris Strasberg et al	-	-	-
Present Study	34.5	8.5	16

CONCLUSION

From the study I conclude that,

Sinus bradycardia is the commonest conduction disturbance in acute ST elevation.

Myocardial infarction followed by first degree heart block then complete heart block and second degree heart block.

With increasing age the occurrence of conduction disturbances increases with male preponderance.

Hypertension and diabetes mellitus are important and statistically significant risk factor for conduction disturbances.

Smoking and alcoholism is somewhat influence the conduction disturbances but is not statistically significant.

Among hospital mortality sinus bradycardia group had high mortality because of need for immediate pacemaker.

There is no significant correlation between conduction disturbances and other risk factors like dyslipidemia and COPD.

But there is significant correlation between mean value of dyslipidemia and prognosis of conduction disturbances.

SUMMARY

From the study and literature it is well known that the incidence of conduction disturbances in acute inferior wall myocardial increases when age increases with male preponderance. Sinus bradycardia is the common conduction disturbances. Hypertension and diabetes mellitus are important risk factor for increase incidence of conduction disturbances. Mean value of Dyslipidemia is correlate with prognosis of conduction disturbances.

KEY WORDS:

Coronary artery disease

Atherosclerosis

Acute inferior wall myocardial infarction

Conduction disturbances

12 lead electrocardiogram

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ANNEXURE - I

PROFORMA

AGE

SEX

ADDRESS

CHIEF COMPLAINTS

PERSONALHISTORY: SMOKING,ALCOHOLISM

FAMILY HISTORY:

PAST HISTORY: HYPERTENSION,DIABETES

MELLITUS,CORONARY ARTERY DISEASE AND COPD.

TREATMENT HISTORY:

GENERAL EXAMINATION

SYSTEM EXAMINATION

INVESTIGATIONS :

COMPLETE BLOOD COUNT

RANDOM BLOOD SUGAR

RENAL FUNCTION TEST

ELECTROCARDIOGRAM

FASTING LIPID PROFILE

CHEST X-RAY, ECHO CARDIOGRAM

DIAGNOSIS

TREATMENT

ANNEXURE -2

KEY TO MASTER CHART

HT-HYPERTENSION

DM-DIABETES MELLITUS

TC-TOTAL CHOLESTEROL

HDL-HIGH DENSITY LIPO PROTEIN

LDL-LOW DENSITY LIPO PROTEIN

COPD-CHRONIC OBSTRUCTIVE PULMONORY DISEASE

CHB-COMPLETE HEART BLOCK

SB - SINUS BRADYCARDIA

CONDUCTION CODE-1-FIRST DEGREE HEART BLOCK

2-SECOND DEGREE HEART BLOCK

3-COMPLETE HEART BLOCK

4- SINUS BRADYCARDIA

ANNEXURE -3

CONSENT FORM

Yourself Mr./Mrs./Ms…….. are being asked to be a participant in the research study titled "Analysis of 200 cases of conduction disturbances in acute ST elevation inferior wall myocardial infarction related to causal comparison" in CMC Hospital, Coimbatore, conducted by DR. A. RADHA KRISHNAN , Post Graduate Student, Department of General Medicine, Coimbatore Medical College. You are eligible after looking into the inclusion criteria. You can ask any question you may have before agreeing to participate.

Research Being Done

Analysis of 200 cases of conduction disturbances in acute ST elevation inferior wall myocardial infarction related to causal comparison

Purpose of Research

The aim to analyze 200 cases of conduction disturbances in patients with acute ST elevation inferior wall myocardial infarction related to following causal comparisons like Age, sex, Smoking, Alcoholism, hypertension, diabetes mellitus, total cholesterol, HDL, LDL & COPD .

To find out any association of each comparisions group with ST elevation inferior wall myocardial infarction particularly on the risk of conduction disturbances.

Decline from Participation

You have the option to decline from participation in the study existing protocol for your condition.

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me, and I may ask questions at any time.

---	---
Signature /Left thumb impression (volunteer)	Date
---	---
Signature of witness	Date

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி :

கோவை அரசு மருத்துவக் கல்லூரி மருத்துவமனையில் மருத்துவர் ஆ. ராதாகிருஷ்ணன் தலைமையில் நடைபெறும் இந்த ஆய்வில் முழு சம்மதத்துடன் கலந்து கொள்ள சம்மதிக்கிறேன். இந்த ஆய்வில் என்னை பற்றி விவரங்களை பாதுகாப்புடன் இந்த ஆய்வில் வெளியிட ஆட்சேபணை இல்லை என்று தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் ஆய்வில் இருந்து எந்த நேரத்திலும் விலக்கிக் கொள்ளும் உரிமை உண்டு என்று அறிவேன்.

இடம் :

தேதி :

Sl.o.	NAME	SEX	AGE		SMOKING	ALCOHOL	HTN	DM	TC	TC mg	HDL	HDL mg	LDL	LDL mg	COPD	CONDUCTION DITURBANCES	COND CODE	DIED
1	MARIAMMAL	F	45	3	NO	NO	NO	yes	HIGH	205	LOW	45	HIGH	132	no	2 degree	2	NO
2	KUMAR	M	28	1	yes	yes	yes	NO	N	130	N	51	N	121	no	sinus bradycardia	4	NO
3	MARIAPPAN	M	65	5	yes	yes	yes	yes	HIGH	206	LOW	45	HIGH	133	y	1 degree	1	NO
4	SANTHANAM	M	65	5	yes	yes	NO	NO	N	131	N	52	N	79	no	CHB	3	NO
5	BUVANESWARI	F	69	5	NO	NO	yes	YES	HIGH	321	LOW	35	HIGH	171	no	sinus bradycardia	4	yes
6	SURESH	M	51	4	yes	yes	yes	yes	N	140	N	53	N	75	no	1degree	1	NO
7	SRINIVASAN	M	43	3	yes	yes	yes	NO	HIGH	212	LOW	46	HIGH	134	y	2 degree	2	NO
8	BALAN	M	26	1	yes	yes	NO	yes	N	141	N	54	N	76	no	sinus bradycardia	4	NO
9	LAKSHMI	F	64	5	NO	NO	yes	YES	HIGH	301	LOW	37	HIGH	165	no	sinus bradycardia	4	yes
10	NANDHAKUMAR	M	52	4	yes	yes	NO	NO	N	150	N	51	N	81	y	1degree	1	NO
11	VARUN	M	43	3	yes	yes	yes	yes	N	151	N	52	N	84	no	2 degree	2	NO
12	GNANASEKAR	M	66	5	yes	yes	NO	yes	HIGH	252	LOW	46	HIGH	135	y	sinus bradycardia	4	NO
13	MUNIAMMAL	F	78	6	NO	NO	yes	YES	N	185	N	51	N	121	no	1degree	1	yes
14	KALIAPPAN	M	53	4	yes	yes	yes	NO	HIGH	211	LOW	47	HIGH	136	y	2 degree	2	NO
15	KUMARESAN	M	42	3	yes	yes	yes	yes	N	132	N	53	N	86	no	sinus bradycardia	4	NO
16	GURU	M	27	1	yes	yes	NO	NO	HIGH	275	LOW	35	HIGH	165	no	CHB	3	yes
17	MAREESWARI	F	63	5	NO	NO	yes	YES	N	190	N	52	N	95	no	sinus bradycardia	4	yes
18	SAKTHIRAJ	M	54	4	yes	yes	yes	yes	HIGH	214	LOW	45	HIGH	137	no	2 degree	2	NO
19	MANI	M	35	2	yes	yes	yes	NO	N	142	N	54	N	94	y	1degree	1	NO
20	THAYAMMAL	F	67	5	NO	NO	NO	yes	HIGH	205	LOW	48	HIGH	139	no	sinus bradycardia	4	NO
21	VIMALRAJ	M	55	4	yes	yes	yes	yes	N	133	N	55	N	96	no	1degree	1	NO
22	KALIAMMMAL	F	62	5	NO	NO	yes	yes	HIGH	222	LOW	45	HIGH	141	no	sinus bradycardia	4	NO
23	JAVEED	M	68	5	yes	NO	yes	yes	N	143	N	53	N	92	y	2 degree	2	NO
24	DURAISAMY	M	56	4	yes	yes	NO	NO	HIGH	215	LOW	45	HIGH	142	no	1degree	1	NO
25	RAJA	M	36	2	yes	yes	yes	yes	N	162	N	52	N	84	y	CHB	3	NO
26	KALEESWARI	F	61	5	NO	NO	yes	yes	HIGH	203	LOW	46	HIGH	143	no	sinus bradycardia	4	NO
27	FAROOQABDULLA	M	57	4	yes	yes	NO	NO	N	154	N	51	N	86	no	sinus bradycardia	4	NO
28	HILANRAJ	M	69	5	yes	NO	NO	YES	N	180	N	52	N	121	y	2 degree	2	yes
29	SARAVANAN	M	60	4	yes	yes	yes	NO	HIGH	256	LOW	35	HIGH	145	no	1degree	1	yes
30	BANUMATHI	F	47	3	NO	NO	NO	yes	HIGH	212	LOW	47	HIGH	132	no	sinus bradycardia	4	NO
31	YASIR	M	25	1	yes	NO	NO	NO	N	123	N	54	N	82	no	sinus bradycardia	4	NO
32	ESAKKIAPPAN	M	58	4	yes	yes	NO	NO	HIGH	221	LOW	48	HIGH	145	no	1degree	1	NO

33	MADHIVANAN	M	64	5	yes	yes	yes	yes	HIGH	205	LOW	49	HIGH	153	y	CHB	3	NO
34	LUBAINA	F	48	3	NO	NO	yes	yes	N	102	N	56	N	87	no	2 degree	2	NO
35	CHANDRAN	M	59	4	yes	yes	NO	NO	N	148	N	52	N	83	no	1degree	1	NO
36	SANMUGAM	M	87	6	yes	NO	yes	yes	HIGH	231	LOW	43	HIGH	154	y	sinus bradycardia	4	NO
37	RAJKUMAR	M	62	5	yes	yes	yes	yes	N	152	N	51	N	92	no	1degree	1	NO
38	FATHIMA	F	51	4	NO	NO	NO	yes	HIGH	222	LOW	44	HIGH	136	no	sinus bradycardia	4	NO
39	ABDULJALEEL	M	52	4	yes	yes	yes	yes	N	123	N	50	N	102	y	2 degree	2	NO
40	MEERAMYDEEN	M	49	3	yes	NO	yes	NO	HIGH	205	LOW	45	HIGH	133	no	CHB	3	NO
41	VINOTH	M	25	1	yes	yes	NO	yes	N	142	N	53	N	105	no	sinus bradycardia	4	NO
42	SUNDARI	F	67	5	NO	NO	yes	yes	HIGH	206	LOW	37	HIGH	134	no	1degree	1	NO
43	ADHIKESAVAN	M	53	4	yes	yes	yes	NO	HIGH	207	LOW	48	HIGH	147	y	1degree	1	NO
44	RAMASAMY	M	30	1	yes	yes	NO	yes	N	154	N	52	N	108	no	2 degree	2	NO
45	SUDALI	F	66	5	NO	NO	yes	yes	HIGH	208	LOW	45	HIGH	152	no	sinus bradycardia	4	NO
46	PUVIARASAN	M	54	4	yes	yes	yes	NO	HIGH	265	LOW	34	HIGH	152	y	CHB	3	yes
47	YUVARAJ	M	31	2	yes	yes	NO	yes	N	123	N	54	N	110	no	sinus bradycardia	4	NO
48	KARUPPASAMY	M	62	5	yes	yes	yes	yes	HIGH	214	LOW	41	HIGH	134	y	1degree	1	NO
49	SUMATHI	F	55	4	NO	NO	yes	NO	N	124	N	52	N	114	no	sinus bradycardia	4	NO
50	BALAJI	M	61	5	yes	yes	NO	yes	HIGH	215	LOW	39	HIGH	187	no	2 degree	2	NO
51	BABU	M	29	1	yes	yes	yes	yes	N	152	N	51	N	122	no	1degree	1	NO
52	RAMESH	M	56	4	yes	yes	yes	yes	HIGH	216	LOW	45	HIGH	175	y	CHB	3	NO
53	GNANASELVI	F	66	5	NO	NO	yes	NO	N	189	N	52	N	125	no	sinus bradycardia	4	yes
54	RAJESH	M	32	2	yes	yes	NO	yes	HIGH	222	LOW	46	HIGH	165	no	sinus bradycardia	4	NO
55	JASVINRAJ	M	65	5	yes	yes	NO	yes	HIGH	205	LOW	42	HIGH	132	no	1degree	1	NO
56	PALANIAPPAN	M	56	4	yes	yes	NO	NO	N	132	N	53	N	108	no	1degree	1	NO
57	PARAMESWARI	F	68	5	NO	NO	yes	yes	HIGH	222	LOW	43	HIGH	152	no	sinus bradycardia	4	NO
58	TAMILSELVAN	M	57	4	NO	NO	yes	yes	N	145	N	51	N	86	no	sinus bradycardia	4	NO
59	KRISHNAN	M	33	2	yes	yes	NO	NO	HIGH	206	LOW	39	HIGH	147	no	1degree	1	NO
60	VASANTH	M	67	5	yes	yes	NO	YES	HIGH	207	LOW	31	HIGH	165	y	sinus bradycardia	4	yes
61	KUMARESAN	M	58	4	yes	yes	yes	NO	N	125	N	65	N	88	no	2 degree	2	NO
62	SELVI	F	66	5	NO	NO	yes	YES	HIGH	214	LOW	35	HIGH	171	no	1degree	1	yes
63	SALEEM	M	65	5	NO	NO	NO	yes	N	121	N	66	N	75	no	CHB	3	NO
64	MAHADEVAN	M	64	5	yes	yes	yes	NO	HIGH	216	LOW	38	HIGH	156	no	1degree	1	NO
65	SELVANAYAGI	F	34	2	NO	NO	yes	yes	N	132	N	62	N	110	no	sinus bradycardia	4	NO

66	PANDARAM	M	59	4	yes	yes	yes	yes	HIGH	205	LOW	37	HIGH	132	no	1degree	1	NO
67	JEYARAJ	M	63	5	yes	yes	NO	NO	N	142	N	64	N	114	y	sinus bradycardia	4	NO
68	AVUDAIAMMAL	F	62	5	NO	NO	yes	yes	N	125	N	52	N	120	no	CHB	3	NO
69	ASHOKKUMAR	M	35	2	NO	NO	yes	yes	HIGH	205	LOW	35	HIGH	134	no	2 degree	2	NO
70	LAKSHMANAN	M	77	6	yes	yes	yes	NO	N	148	N	58	N	115	y	sinus bradycardia	4	NO
71	SANTHI	F	50	3	NO	NO	NO	yes	HIGH	207	LOW	36	HIGH	173	no	1degree	1	NO
72	KALIGOUNDER	M	85	6	yes	yes	yes	yes	HIGH	208	LOW	41	HIGH	137	no	sinus bradycardia	4	NO
73	ESWARAN	M	61	5	NO	NO	yes	NO	N	214	N	56	N	121	no	2 degree	2	yes
74	ARUMAINAYAGAM	M	36	2	yes	yes	yes	yes	N	166	N	59	N	116	no	sinus bradycardia	4	NO
75	AYYAMMAL	F	65	5	NO	NO	NO	yes	N	196	N	57	N	120	no	sinus bradycardia	4	NO
76	SEKAR	M	60	4	yes	yes	yes	NO	HIGH	222	LOW	40	HIGH	135	y	1degree	1	NO
77	ESAKKIAMMAL	F	51	4	NO	NO	yes	NO	N	125	N	65	N	125	no	CHB	3	NO
78	VIMALKUMAR	M	52	4	yes	yes	yes	yes	HIGH	205	LOW	42	HIGH	133	no	1degree	1	NO
79	ESWARAPANDI	M	37	2	yes	yes	NO	NO	HIGH	231	N	54	N	124	no	sinus bradycardia	4	NO
80	IYYAPPAN	M	69	5	yes	yes	yes	yes	N	153	N	55	N	109	no	2 degree	2	NO
81	BUVANA	F	68	5	NO	NO	yes	NO	HIGH	205	N	52	N	103	no	sinus bradycardia	4	NO
82	WASIM	M	53	4	NO	NO	NO	yes	HIGH	206	LOW	43	HIGH	131	no	sinus bradycardia	4	NO
83	LALITH	M	67	5	yes	yes	yes	yes	N	142	LOW	44	HIGH	132	y	1degree	1	NO
84	ABDULRAHMAN	M	38	2	yes	yes	NO	NO	HIGH	208	LOW	45	HIGH	134	no	sinus bradycardia	4	NO
85	NAMBIAMMAL	F	61	5	NO	NO	yes	yes	HIGH	214	LOW	41	HIGH	136	no	1degree	1	NO
86	CHARAN	M	54	4	yes	yes	yes	yes	N	141	LOW	42	HIGH	138	no	sinus bradycardia	4	NO
87	BALAKUMAR	M	62	5	NO	NO	yes	NO	N	159	N	57	N	89	no	1degree	1	NO
88	SYED AKBAR	M	39	2	yes	yes	yes	NO	N	147	N	58	N	95	no	CHB	3	NO
89	NALINI	F	65	5	NO	NO	NO	yes	HIGH	205	LOW	43	N	106	no	sinus bradycardia	4	NO
90	RAMACHANDRAN	M	55	4	yes	yes	NO	yes	N	123	N	59	N	122	y	sinus bradycardia	4	NO
91	GANAPATHY	M	40	2	NO	NO	yes	NO	HIGH	206	LOW	44	HIGH	141	no	1degree	1	NO
92	RAVEENDRAN	M	64	5	yes	yes	yes	yes	HIGH	216	LOW	45	HIGH	143	y	sinus bradycardia	4	NO
93	SUBHA	F	56	4	NO	NO	yes	yes	N	156	N	61	N	118	no	sinus bradycardia	4	NO
94	SARANAKUMAR	M	64	5	yes	yes	yes	NO	HIGH	205	LOW	35	HIGH	184	y	CHB	3	yes
95	PALANI	M	41	3	yes	yes	yes	yes	N	158	N	62	N	106	no	sinus bradycardia	4	NO
96	DIRAVIUM	M	60	4	NO	NO	yes	yes	N	152	N	56	N	89	no	sinus bradycardia	4	NO
97	KALIDASAN	M	57	4	yes	yes	NO	yes	HIGH	222	LOW	49	HIGH	145	y	1degree	1	NO
98	DURAISELVI	F	32	2	NO	NO	yes	NO	N	145	N	58	N	95	no	sinus bradycardia	4	NO

99	NAVEEN	M	69	5	yes	yes	yes	yes	N	132	N	59	N	113	no	sinus bradycardia	4	NO
100	MADASAMY	M	58	4	yes	yes	yes	yes	HIGH	205	LOW	48	HIGH	143	no	1degree	1	NO
101	IYYAVU	M	68	5	yes	yes	yes	YES	HIGH	206	LOW	36	HIGH	156	no	1degree	1	yes
102	MUTHURANI	F	43	3	NO	NO	yes	NO	N	182	N	58	N	115	no	sinus bradycardia	4	NO
103	KARUPPAN	M	67	5	NO	NO	yes	NO	HIGH	214	LOW	47	HIGH	143	no	1degree	1	NO
104	KUPPUSAMY	M	61	5	yes	yes	yes	yes	HIGH	231	LOW	45	HIGH	139	y	sinus bradycardia	4	NO
105	VELLAIYAN	M	59	4	yes	yes	NO	yes	HIGH	222	LOW	46	HIGH	137	no	sinus bradycardia	4	NO
106	MERLIN	F	44	3	NO	NO	yes	yes	N	175	N	57	N	106	no	1degree	1	NO
107	IYYASAMY	M	76	6	NO	NO	yes	YES	N	206	LOW	37	HIGH	135	no	sinus bradycardia	4	yes
108	VELAN	M	58	4	yes	yes	yes	NO	HIGH	207	LOW	43	HIGH	135	no	sinus bradycardia	4	NO
109	HELENA	F	50	3	NO	NO	yes	yes	HIGH	208	LOW	44	HIGH	138	no	CHB	3	NO
110	ALEXANDER	M	45	3	NO	NO	yes	yes	N	195	N	56	N	108	no	sinus bradycardia	4	NO
111	DURAIAPPAN	M	81	6	yes	yes	NO	yes	HIGH	215	LOW	45	HIGH	141	y	CHB	3	NO
112	JANNADH	F	51	4	NO	NO	yes	yes	N	124	N	54	N	121	no	1degree	1	NO
113	BOOPATHI	M	57	4	yes	yes	yes	NO	HIGH	222	LOW	46	HIGH	134	no	sinus bradycardia	4	NO
114	NIRMALRAJ	M	46	3	yes	yes	yes	yes	N	153	N	59	N	102	y	sinus bradycardia	4	NO
115	BEGAM	F	60	4	NO	NO	yes	yes	HIGH	222	LOW	47	HIGH	135	no	1degree	1	NO
116	BOOPALAN	M	52	4	yes	yes	NO	yes	N	164	N	58	N	108	no	sinus bradycardia	4	NO
117	VAGEESWARAN	M	65	5	NO	NO	yes	NO	N	142	N	52	N	75	no	1degree	1	NO
118	PANDI	M	56	4	yes	yes	yes	yes	N	153	N	51	N	72	no	sinus bradycardia	4	NO
119	VIGNESWARAN	M	47	3	yes	yes	yes	yes	N	123	N	58	N	79	y	1degree	1	NO
120	RANI	F	53	4	NO	NO	yes	NO	N	132	N	61	N	84	y	sinus bradycardia	4	NO
121	IYYANRAJ	M	67	5	yes	yes	yes	yes	HIGH	231	LOW	42	HIGH	136	no	CHB	3	NO
122	LOORDUMUHAMED	M	64	5	yes	yes	NO	yes	HIGH	222	LOW	43	HIGH	134	y	sinus bradycardia	4	NO
123	PAPPATHI	F	55	4	NO	NO	yes	yes	N	111	N	65	N	82	y	1degree	1	NO
124	CHINNASAMY	M	48	3	NO	NO	yes	NO	HIGH	206	LOW	39	HIGH	141	no	sinus bradycardia	4	NO
125	MARAPPAN	M	54	4	yes	yes	yes	NO	HIGH	207	LOW	48	HIGH	156	no	sinus bradycardia	4	NO
126	MARIASELVAM	M	62	5	yes	yes	NO	NO	N	124	N	69	N	89	y	1degree	1	NO
127	CHITRA	F	54	4	NO	NO	yes	yes	HIGH	214	LOW	47	HIGH	184	no	sinus bradycardia	4	NO
128	SUMAN	M	27	1	yes	yes	yes	yes	N	152	N	64	N	95	no	sinus bradycardia	4	NO
129	DHEENADAYALAN	M	49	3	NO	NO	yes	yes	HIGH	216	LOW	48	HIGH	176	no	1degree	1	NO
130	JALEEL	M	55	4	yes	yes	NO	yes	HIGH	222	LOW	45	HIGH	145	y	sinus bradycardia	4	NO
131	VANI	F	53	4	NO	NO	yes	NO	N	155	N	62	N	93	no	sinus bradycardia	4	NO

132	TIRUMALAISAMY	M	66	5	yes	yes	yes	YES	HIGH	222	LOW	34	HIGH	178	no	1degree	1	yes
133	KESAVAN	M	52	4	yes	yes	yes	NO	HIGH	231	LOW	46	HIGH	165	y	CHB	3	NO
134	ESWARI	F	40	2	NO	NO	NO	yes	HIGH	222	LOW	49	HIGH	142	no	sinus bradycardia	4	NO
135	PANDIDURAI	M	56	4	NO	NO	yes	yes	N	165	N	63	N	102	no	1degree	1	NO
136	IYYAMPERUMAL	M	51	4	yes	yes	yes	NO	N	152	N	65	N	108	y	CHB	3	NO
137	SANKARAMMAL	F	67	5	NO	NO	yes	yes	HIGH	207	LOW	44	HIGH	134	no	sinus bradycardia	4	NO
138	SUBRAMANI	M	66	5	yes	NO	NO	yes	HIGH	208	LOW	41	HIGH	133	y	CHB	3	NO
139	SUBBAIAH	M	50	3	yes	yes	yes	NO	N	167	N	56	N	112	y	1degree	1	NO
140	PITCHIMANI	M	31	2	NO	NO	yes	NO	HIGH	215	LOW	42	HIGH	132	no	sinus bradycardia	4	NO
141	LALITHA	F	57	4	NO	NO	yes	NO	HIGH	216	LOW	40	HIGH	141	no	CHB	3	NO
142	PITCHAIAH	M	65	5	yes	yes	NO	YES	N	222	N	52	N	125	no	1degree	1	yes
143	VELMURUGAN	M	32	2	yes	yes	yes	NO	HIGH	205	LOW	39	HIGH	136	no	sinus bradycardia	4	NO
144	MAMTHA	F	59	4	NO	NO	yes	yes	HIGH	222	LOW	38	HIGH	138	no	sinus bradycardia	4	NO
145	MARIRAJ	M	58	4	NO	NO	yes	yes	N	189	N	58	N	125	no	1degree	1	NO
146	SAKTHISELVAM	M	64	5	yes	yes	NO	yes	HIGH	214	LOW	37	HIGH	142	no	sinus bradycardia	4	NO
147	MUNIANDI	M	58	4	yes	yes	yes	NO	HIGH	216	LOW	35	HIGH	141	y	sinus bradycardia	4	NO
148	MUMTAJ	F	59	4	NO	NO	yes	yes	N	195	N	54	N	114	no	1degree	1	NO
149	SADAIANDI	M	75	6	NO	NO	NO	yes	HIGH	231	LOW	32	HIGH	140	no	CHB	3	NO
150	KANDHASAMY	M	50	3	yes	yes	yes	yes	N	175	N	57	N	109	y	1degree	1	NO
151	THIRAVIUM	M	57	4	yes	yes	yes	NO	HIGH	207	LOW	41	HIGH	171	no	CHB	3	yes
152	RAJARAJESWARI	F	72	6	NO	NO	NO	yes	N	185	N	65	N	98	no	1degree	1	NO
153	SAMBATH	M	61	5	yes	yes	yes	yes	HIGH	215	LOW	48	HIGH	139	y	sinus bradycardia	4	NO
154	MURUGAIAH	M	56	4	NO	NO	yes	yes	N	125	N	62	N	84	no	1degree	1	NO
155	MUNIAPPAN	M	88	6	yes	yes	NO	NO	HIGH	205	LOW	35	HIGH	138	y	1degree	1	NO
156	LILLY	F	68	5	NO	NO	yes	NO	HIGH	222	LOW	49	HIGH	135	no	CHB	3	NO
157	RAJAPPAN	M	55	4	yes	yes	yes	NO	N	195	N	58	N	79	y	sinus bradycardia	4	NO
158	PERUMAL	M	71	6	yes	NO	yes	NO	HIGH	214	LOW	36	HIGH	136	no	1degree	1	NO
159	VAITHEESWARAN	M	67	5	yes	yes	NO	YES	HIGH	216	LOW	42	HIGH	175	y	sinus bradycardia	4	yes
160	PITCHAMMAL	F	54	4	NO	NO	yes	yes	N	132	N	54	N	105	no	1degree	1	NO
161	GNANASELVAM	M	66	5	NO	NO	NO	yes	N	145	N	57	N	105	no	CHB	3	NO
162	SHANMUGAVEL	M	53	4	yes	yes	NO	NO	N	165	N	61	N	112	y	sinus bradycardia	4	NO
163	KUPPAN	M	67	5	yes	yes	NO	NO	HIGH	207	LOW	48	HIGH	140	y	1degree	1	NO
164	MANIMEKALAI	F	66	5	NO	NO	yes	yes	HIGH	208	LOW	47	HIGH	145	no	CHB	3	NO

165	MANIRAJ	M	52	4	NO	NO	yes	yes	HIGH	215	LOW	36	HIGH	143	no	sinus bradycardia	4	NO
166	SANGILI	M	28	1	yes	NO	NO	NO	N	175	N	60	N	108	no	1degree	1	NO
167	VIJAYAKUMAR	M	65	5	yes	yes	yes	yes	N	185	N	52	n	125	no	CHB	3	NO
168	MANIMALA	F	51	4	NO	NO	yes	yes	N	185	N	52	n	114	no	1degree	1	NO
169	KULANDAISAMY	M	62	5	NO	NO	NO	NO	HIGH	333	LOW	36	HIGH	181	no	2 degree	2	yes
170	RAMAIAH	M	60	4	yes	yes	yes	yes	N	165	N	51	n	124	no	sinus bradycardia	4	NO
171	MUTHAIAH	M	50	3	yes	yes	yes	yes	HIGH	214	LOW	35	HIGH	142	y	1degree	1	NO
172	DEVI	F	69	5	NO	NO	NO	yes	N	154	N	53	n	111	no	sinus bradycardia	4	NO
173	ESWARAN	M	59	4	NO	NO	yes	NO	HIGH	222	LOW	47	HIGH	154	no	1degree	1	NO
174	PAVANRAJ	M	67	5	yes	yes	yes	yes	HIGH	231	LOW	42	HIGH	146	no	CHB	3	NO
175	BUVANESWARAN	M	66	5	yes	yes	NO	yes	N	198	N	54	n	102	y	sinus bradycardia	4	NO
176	CHANDRAKALA	F	58	4	NO	NO	yes	yes	HIGH	207	LOW	45	HIGH	142	no	1degree	1	NO
177	HARIHARAN	M	65	5	yes	yes	yes	NO	HIGH	208	LOW	47	HIGH	145	y	sinus bradycardia	4	NO
178	KANNAPPAN	M	73	6	NO	NO	NO	yes	N	157	N	61	n	120	no	1degree	1	NO
179	ISMAIL	M	57	4	yes	yes	NO	yes	N	147	N	65	n	89	no	sinus bradycardia	4	NO
180	MUHAMMED	M	61	5	yes	yes	NO	yes	HIGH	205	LOW	41	HIGH	151	no	sinus bradycardia	4	NO
181	THIAGARAJAN	M	56	4	yes	NO	yes	NO	HIGH	214	LOW	43	HIGH	149	y	1degree	1	NO
182	SAMINATHAN	M	74	6	yes	yes	yes	NO	HIGH	216	LOW	44	HIGH	145	no	CHB	3	NO
183	CHAKRATIS	M	55	4	NO	NO	yes	NO	N	186	N	62	n	95	no	1degree	1	NO
184	CHARLES	M	62	5	yes	yes	NO	yes	N	166	N	60	n	100	y	CHB	3	NO
185	ROBERT	M	63	5	yes	yes	yes	yes	HIGH	222	LOW	42	HIGH	141	no	1degree	1	NO
186	JOSEPH	M	64	5	yes	NO	yes	yes	N	135	N	51	N	99	y	2 degree	2	NO
187	MURUGAN	M	54	4	yes	yes	NO	yes	HIGH	208	LOW	35	HIGH	176	no	CHB	3	yes
188	VELAUTHAM	M	62	5	NO	NO	yes	NO	N	145	N	52	N	105	no	1degree	1	NO
189	MANIKANDAN	M	64	5	yes	yes	NO	yes	HIGH	216	LOW	41	HIGH	139	y	sinus bradycardia	4	NO
190	SANJAI	M	53	4	yes	yes	yes	NO	N	125	N	54	N	116	no	CHB	3	NO
191	AVUDAIAPPAN	M	66	5	yes	NO	yes	yes	N	156	N	53	N	123	no	1degree	1	NO
192	SANKAR	M	68	5	yes	yes	NO	NO	N	180	N	51	N	129	y	sinus bradycardia	4	yes
193	ESAKKIPANDI	M	60	4	NO	NO	yes	yes	HIGH	214	LOW	39	HIGH	151	no	1degree	1	NO
194	GANESH	M	69	5	yes	yes	NO	yes	N	157	N	56	N	124	no	1degree	1	NO
195	MUPPIDATHI	M	52	4	yes	NO	yes	NO	HIGH	222	LOW	41	HIGH	142	no	CHB	3	NO
196	MUTHUKUMAR	M	65	5	yes	yes	yes	NO	N	185	N	55	N	105	y	1degree	1	NO
197	MARAPPAN	M	63	5	NO	NO	yes	NO	HIGH	222	LOW	42	HIGH	135	no	sinus bradycardia	4	NO

198	PARAMESWARAN	M	61	5	yes	yes	yes	yes	N	122	N	51	N	108	no	1degree	1	NO
199	RAMAN	M	51	4	yes	yes	NO	yes	N	124	N	52	N	112	no	1degree	1	NO
200	MUHAMED IRFAN	M	65	5	yes	NO	NO	NO	HIGH	215	LOW	35	HIGH	201	no	CHB	3	yes