“STUDY OF CONDUCTION BLOCKS IN ACUTE MYOCARDIAL INFARCTION”

By

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Dissertation submitted to BLDE University, Vijayapur

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

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Dr. NEELU MAHENDRA SUNKAVALLI

LIST OF ABBREVIATIONS USED

ACS	-	Acute Coronary Syndrome
MI	-	Myocardial Infarction
AV	-	AtrioVentricular
CAD	-	Coronary Artery Disease
Lp (a)	-	Lipoprotein (a)
SA	-	Sinoatrial
RCA	-	Right Coronary Artery
LCA	-	Left Coronary Artery
LAD	-	Left Anterior Descending
RBB	-	Right Bundle Branch
LBB	-	Left Bundle Branch
LDL	-	Low Density Lipoprotein
HDL	-	High Density Lipoprotein
BP	-	Blood Pressure
CRP	-	C Reactive Protein
IL	-	Interleukin
BMI	-	Body Mass Index
TC	-	Total Cholesterol
CK	-	Creatinine Kinase
LDH	-	Lactate dehydrogenase
CTn	-	Cardiac specific troponin
ECG	-	Electrocardiogram
VT	-	Ventricular Tachycardia
EF	-	Ejection Fraction

AMI	-	Acute Myocardial Infarction
IV	-	Intravenous
CHF	-	Congestive Heart Failure
HR	-	Heart Rate
LV	-	Left ventricle
RV	-	Right Ventricle
BBB	-	Bundle branch block
LBBB	-	Left Bundle Branch Block
RBBB	-	Right Bundle Branch Block
LAHB	-	Left anterior hemiblock
LPHB	-	Left posterior hemiblock
LAFB	-	Left Anterior Fascicle Block
LPFB	-	Left Posterior Fascicle Block
ICU	-	Intensive Care Unit
CB	-	Conduction Blocks
LV	-	Left Ventricle
RV	-	Right Ventricle

ABSTRACT

Introduction:

Conduction blocks occur in nearly 15.8% of the patients suffering from acute myocardial infarction (AMI). They are one of the important predictors of poor outcome in acute MI cases. They are associated with higher rates of morbidity (cardiovascular events like hypotension, left ventricular failure, cardiogenic shock, complete AV block and cardiac arrest) and in-hospital mortality. This prospective clinical study was undertaken to assess the association of conduction blocks in acute MI patients with site, timing, complications and disease outcome.

Methods:

One hundred and fifty consecutive patients (118 men, 38 women) who were admitted with the diagnosis of AMI in Shri B.M. Patil Medical College & Hospital were assessed. The initial ECG, recorded immediately after the patient's admission to the emergency department, was considered as baseline. Any heart blocks occurring over the following days were noted by comparing the relevant ECGs with this baseline ECG. Cases were selected taking into consideration inclusion and exclusion criteria.

Results:

The mean age of patients with conduction blocks was 56.97 years and 59.22 years in patients without conduction blocks. The conduction blocks were not found to be associated with the patients' age and sex, whereas cigarette smoking, hypertension, and diabetes mellitus were found to be important risk factors for development of conduction blocks. Conduction blocks were significantly more common among patients with inferior wall MI than anterior wall MI, but bundle branch blocks (BBB)

were more common in anterior wall MI. Mortality is higher in patients with conduction blocks (28.9%) as compared to patients without blocks (14.3%)

Conclusions:

Conduction blocks are associated with higher mortality and morbidity in the form of various cardiovascular events during hospital stay. It has important prognostic significance. So early detection and prompt management of these patients is required to reduce morbidity and mortality.

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INTRODUCTION

Coronary artery disease is the commonest form of heart disease and the leading cause of morbidity and mortality throughout the world[1]. Its prevalence among Indians has doubled during the past two decades. Myocardial infarction is one of the most common diagnoses in hospitalized patients. Immediate and late mortality following acute myocardial infarction is dependent upon the size of the infarction, beside other factors.[2]

Asian Indians have much higher incidence of coronary artery disease compared to other ethnic groups. In India, prevalence of coronary artery disease from 1960s to 1990 has increased two fold (2% to 4%) in rural areas and three fold (3.45% to 9.45%) in urban areas. Prevalence in south India is even higher, 13% in urban areas and 7% in rural areas. In Indians coronary artery disease(CAD) appears a decade earlier with peak in the age group of 51 to 60 yrs[3]

The presence of conduction defects complicating acute myocardial infarction (MI) is relatively frequent and is associated with increased short and long term mortality rates.[4,5]Thrombolytic therapy, although has been established to reduce the mortality in acute myocardial infarction, however its role in reducing the incidence of conduction defects is less clearly defined. Mortality and morbidity associated with conduction defects also remain unchanged.[6]

Conduction blocks are important predictors of poor outcome in patient with acute myocardial infarction and are associated with higher in hospital mortality and morbidity. So, early detection and prompt management of such conduction blocks is necessary to reduce mortality.[7]

Over all prevalence of heart blocks is 15.8%. The development of heart blocks are more common among those patients treated with thrombolytic therapy. Among that Incidence rates of atrio-ventricular blocks is 35%, 1st degree AV blocks 18%, 2nd degree AV block 1-2%, 3rd degree AV block 3%, and incidence of BBB is 65%, LAHB is 30%, LPHB is 6-7%, LBBB is 19% and RBBB is 9.5%.[8]

Further study in this field will contribute to the better understanding of clinical disturbance in acute myocardial infarction (AMI). The purpose of this study is to document the frequency of various conduction defects in cases of acute myocardial infarction who will be observed in coronary care unit, and to know about their relationship with complications and in hospital clinical outcome.

The present study will enlighten the co-relation of conduction defects with the site of infarction.

AIMS AND OBJECTIVES OF STUDY

1. To determine the type of conduction defects in acute myocardial infarction.
2. To co-relate the site of infarction with the type of conduction defect.

REVIEW OF LITERATURE

HISTORICAL ASPECTS[9]

Seneca (4 BC – 65 AD) recorded the earliest description of anginal syndrome.

Andreas Vesalius (1514-1564) described the general structure of heart and coronary vessels.

Raymond de vieusseus (1641-1716) described the course of coronary arteries.

William Heberden (1710-1801) published classical description of angina pectoris, which he called “pectoris dolor”.

Edward Jenner (1786) recognized the symptoms of Angina pectoris in his friend John hunter and predicted that he had Coronary artery disease (CAD), which was proved, later by postmortem study when Hunter died in 1793.

Thomas LaudesBrunton (1844-1916) recommended amyl nitrate for treatment of angina pectoris.

A.F. Stanley Kent (1863-1958) published cardiac conduction studies.

Karl Weigert (1845-1904) described myocardial infarction.

Ernst Von Leyden (1832-1910) contributed to the relationship between diseased coronary arteries and changes in the myocardium.

Zeigler (1849-1905) described pathology of myocardial infarction.

Jones Herrick (1912) concluded by autopsy studies that acute myocardial infarction results from coronary artery disease.

INCIDENCE OF CONDUCTION BLOCKS

Go As et al evaluated the incidence of bundle branch block in 297,832 patients admitted to a hospital with an acute MI between 1994 and 1997: 6.7 percent of patients had LBBB and 6.2 percent had RBBB on the initial ECG[10]

A similar study by Stenestrand et.al, showed that rate of LBBB (9 percent) was noted in a prospective analysis of over 88,000 patients in Sweden[11]

Meine et al- studied the incidence high degree AV block in the fibrinolytic era comes from a review of almost 76,000 patients with ST-elevation MI (STEMI) enrolled in four large randomized trials[12]The overall incidence was 6.9 percent: 9.8 percent with inferior MI and 3.2 percent with anterior MI.

Gang UJ et al studies concerning AV block in acute MI since widespread use of PCI, the incidence of AV block appears to have declined. Among 2073 STEMI patients treated with primary PCI in the Danish National Patient Register, only 67 (3.2 percent) had documentation of second or third degree AV block at presentation or during hospitalization [13]

Auffret et al examined 6662 STEMI patients enrolled in a prospective registry in Brittany, France between 2006 and 2013[14.]About 74 percent of patients underwent primary PCI and 90% had PCI at some point in the index hospitalization. A total of 234 patient (3.5 percent) had Mobitz II or third degree AV block; 149 (2.2 percent) on admission and 85 (1.3 percent) later in the hospitalization. AV block was more common among those with right coronary occlusion (5.9 percent) than those with other infarct-related arteries (1.5 percent). Rates of AV block developing during hospitalization were lower for patients undergoing primary PCI (1.2 percent) or thrombolysis (0.5 percent) than those with no reperfusion treatment (2.6 percent).

Kim HL et al studied that high degree AV block occurred in 3.2 percent of patients, 5.9 percent of patients with right coronary artery occlusion, and in 1.5 percent of patients with other infarct-related arteries[13-15]

Singh S M et al studied 59,229 patients with acute coronary syndromes at 126 hospitals in 14 countries [16]Mobitz II or third degree AV block occurred in 2.9 % of patients . AV block occurred in 54 % at presentation and in 46 % later in the hospitalization. The right coronary artery was the culprit vessel in 64.7 percent of patients with AV block and 31.3% of patients without AV block ($p<0.001$). 35% of patients with AV block had temporary pacemaker inserted and 5.9% received a permanent pacemaker. Temporal analysis showed a declining incidence of AV block between 1999 and 2007 (-0.2 percent per year, $p<0.001$).

GUSTO TRIAL showed incidence of complete heart block (CHB) in acute MI is about 4 to 5 percent [17-21]and the combined end point of CHB or second degree AV block in 7 to 10 percent [12,20]

PROGNOSIS

Harikrishnan P et al Among patients with an anterior MI, the 3.2 percent with AV block had a significant and substantial increase in mortality at 30 days (41 versus 8 percent in those without AV block; odds ratio 3.0, 95% CI 2.2-4.1). The increase in mortality did not increase further at one year. In the United States National Inpatient Sample registry from the PCI era, CHB in the setting of anterior STEMI increased in-hospital mortality fourfold, versus only two fold in inferior STEMI patients[22]

Wong C K et al, discussed , BBB was present of 5.1 percent of initial (ECGs), but new BBB developed in only 0.9 percent of patients on a second ECG taken 60

minutes later [23.] Reports from case series and registries generally define the presence of BBB on the basis of the initial ECG. This finding reflects the prevalence of BBB among patients presenting with acute MI, not the incidence of new BBB due to the infarction.

Aplin M et al showedHigh degree AV block — High (second or third) degree atrioventricular (AV) block is associated with an increase in mortality in patients with an inferior or anterior (MI),The increase in mortality risk is largely seen within the first 30 days; among 30-day survivors, subsequent mortality does not appear to be increased[24,25]

Hindman et al studied the progression to second or third degree AV block is associated with twice the in-hospital mortality compared with those who do not progress [26]

Goldberg et al studied high grade AV block in patients with an inferior MI is associated with increased in-hospital mortality. An increase in mortality with high degree AV block of about 15% at 30 days is seen in patients with an inferior MI treated with a thrombolytic agent [27-29,30]and High degree AV block in patients with an anterior wall MIis associated with a greater increase in in-hospital and 30-day mortality than seen with an inferior wall infarction, probably due to more extensive myocardial involvement and a higher incidence of hemodynamic complications.

Dubois C et al showed 30-day mortality was significantly increased only in patients with RBBB at baseline and an anterior MI (odds ratio 2.48) and in those with new LBBB (odds ratio 2.97) or new RBBB with an anterior MI (odds ratio 3.84). The increase in risk with RBBB and an anterior MI has been noted in other studies [31]

Sgarbossa EB et al of thrombolytic therapy, studied patients with a BBB were more likely to experience cardiogenic shock (19 versus 11 percent), AV block or asystole (30 versus 19 percent), and to require a pacemaker (18 versus 11 percent). Mortality was higher when the BBB was persistent (20 percent versus 12 and 8 percent in the 24 percent of patients who had partial or complete resolution of the BBB, respectively).[32]

Guerrero M et al studied patients undergoing primary PCI, the presence of BBB on the baseline ECG is associated with increased mortality. This was illustrated in a review of 3053 patients in the PAMI trials; LBBB was present in 1.6 percent and RBBB in 3.1 percent. [33] In-hospital mortality was 14.6, 7.4, and 2.8 percent in patients with LBBB, RBBB, and no BBB, respectively. On multivariate analysis, LBBB was an independent predictor of in-hospital mortality (odds ratio 5.5).[34]

Melgareso-Moreno A et al confirmed the association of new BBB with increased mortality in acute MI. Among 5570 patients with acute MI between 1998 and 2008 enrolled in a Spanish registry, BBB was present in 17.3 percent (RBBB 10.6 percent, LBBB 6.7 percent) [35.]

Rathore SS et al studied patients with BBB who did not have a contraindication to thrombolytics received this therapy (17 and 32 percent for LBBB and RBBB, respectively, compared with 67 percent of those without a BBB) and fewer with LBBB or RBBB received a beta blocker within the first 24 hours (24 and 32 versus 40 percent). Patients with permanent pacemakers are also less likely to receive reperfusion therapy [36.]

Vivas et al, in primary PCI era suggest that both RBBB and LBBB remain significantly associated with increased short- and long-term mortality[37,38]

Newly KH et al[39] studied 721 patients of AMI enrolled in GUSTO-1 and TAMI-9 trials, 23.6% had BBB, with transient block in 18.4% and permanent in 5.3% RBBB was found in 13%, LBBB in 7% and alternating BBB in 3.5% patients. Patients with BBB had lower EF, more diseased vessels, higher mortality rate (8.7% VS 3.5%, P<0.007), those with persistent block had even higher mortality rate (19.4% VS 5.6%, P<0.001).

Moreno AM et al[40] performed a multicentre prospective study of 1238 patients with AMI for 1 year. 135 patients (10.9%) were found to have RBBB (new cases-51, old cases – 46, indeterminate time of orgin-38 cases). Patients withRBBB were frequently associated with heart failure (46% VS 24%,p<0.001), AV block (11% VS 3.6%,P<0.001) and higher 1 year mortality (40.7% VS 17.6, P<0.001). Patients with new RBBB, permanent RBBB had even higher mortality.

Mujamdar AA et al [41] study showed higher incidence of conduction disturbances in inferior wall MI than anterior wall MI (56.8% and 31.8% respectively). Conduction disturbances were mostly (92%) atrio-ventricular in inferior myocardial infarction and mostly (72%) intraventricular in anterior myocardial infarction. Patients with conduction disturbances developed more complications (84%) than those without conduction disturbances (54%). Mortality rate was higher in patients with conduction defects (25%) than in patients without conduction defects (3.6%) with a overall rate of 13%. Mortality

rate was higher in anterior myocardial infarction (50%) than in inferior myocardial infarction (25%) when complicated by complete atrioventricular block.

Bates ER et al[42] studied 932 patients of Q wave MI, of them 178 had RBBB. Patients with RBBB showed an increased incidence of LV failure (72% Vs 52%, $p<0.001$) increased in hospital mortality rate (17% Vs 7%, $p<0.001$). Thus RBBB after MI is an independent marking of poor prognosis

Beher s et al[43] studied 2273 patients of inferior wall MI enrolled in SPRINT trail. 251 patients (11%) had complete heart block. These patients exhibited more serious arrhythmic and mechanical complications during hospitalization. The in hospital mortality is also higher in patients with complete heart block (37% Vs 11%, $p<0.001$) than in those without AV block.

Anatomy of the conduction system[44 -47]

The specialized conduction system consists of

1. The Sino — atrial (S.A.Node)
2. Preferential intra — atrial &internodal pathways.
3. The atrio — ventricular (A.V Node)
4. The bundle of His (The common A.V. Bundle)
5. The right Bundle branch
6. The left bundle branch
7. The left bundle branch which divides into two major
    a) The anterio- superior division.
    b) The postero- inferior division.
8. The Purkinje Fibers.

1. SINO-ATRIAL NODE (S.A.NODE):

It is also called as primary pacemaker of the heart. The Sino-atrial node is a crescent structure, having a "head", "body" and "tail", located in the right atrium 1mm below the epicardium of the sulcus terminals with a well defined summit which is always present on the lateral aspect of the junction between the superior venacava and right atrium. There is no overlying muscle. It tapers both medially and laterally and runs backwards to the left and onwards curling around the superior venacava. The Sino-atrial artery, which also supplies a large adjacent area of myocardium supplies the SA node. The Sino-atrial node consists of various types of cells including Purkinje cells, smaller Sino-atrial nodal cells and stellate cells. The purkinje cells are smaller nodal- fibers are organized as preferential pathways –which leave the Sino-atrial node

to enter the atria and then on to atrio-ventricular node. The stellate shaped cells have pacemaker properties.

2. THE PREFERENTIAL INTER — NODAL PATHWAY:

Activation of the atria was for a long time considered to be a sequential and circumferential process proceeding outwards from the S.A. node "like the ripples caused by a pebble thrown into a pond". Current physiological evidence indicates that this is not so, the activation process, infarct, occurs through preferential or Semi-Specialized pathways.

There are three such 'Preferential' pathways between Sino-atrial and A.V.Node.

a) The anterior internodal tract: This has two braches — Bachman's bundle and a descending branch.
b) The middle internodal tract (Wenckebach's Bundle)
c) The posterior internodal tract (Thorel's Pathways)

a. The anterior internodal tract:

The anterior internodal tract leaves the anterior margin of the S.A.Node and passes anterior to the superior venacava where it divides into two branches.

i. The inter-atrial branch (Bachmann's bundle) — this branch Or bundle courses within the anterior inter-atrial myocardial band from the right atrium to the left atrium.
ii. The descending branch — this branch descends in the inter-atrial septum to enter the crest of the A.V.Node.

b. The middle internodal (pathway) tract : (Wenckebach's Bundle)

The middle internodal tract leaves the posterior margin of the Sino-atrial node and passes posteriorly to the superior venacava where it descends in the inter-atrial septum

anterior to the fossa ovalis and merges with the fibers of the anterior internodal tract to enter the superior crest of the A.V.Node.

c. The posterior internodal tract : (Thorel's Pathway)

The posterior inter-nodal tract leaves the posterior margin of the Sino-atrial node and runs within the crista terminals. It curves through the valve of the inferior Venacava the Bustachian Ridge and then above the opening of the coronary sinus to enter the posterior margin of the A.V. node. (This tract is longer than the other two tract).

3. THE 'BY PASS' TRACT :

The by-pass tract is a short tract, which is mainly a continuation of the posterior inter-nodal tract, but also receives fibers from the anterior and middle inter-nodal tracts. This tract is so named because it by-passes the main body of the A.V. node to enter it distally. It may also enter the bundle of His directly. Fibers from all three inter-nodal tracts bend or intermingle just proximal to the AV node where all three tracts divide into two divisions, one entering the crest of the AV node, and the other contribution to the by-pass tract.

4. ATRIO — VENTRICULAR NODE (A V NODE):

AV node is a flattened oblong structure concave on one side and convex on the other. The AV node is situated on the right side of the central fibrous body that anchors the mitral value annulus. The concave surface lies directly on the right atrial side of the central fibrous body. The posterior margin of this AV node lies close to the ostium of the coronary sinus. The anterior or distal end blends with the bundle of his. The AV node is supplied by the AV nodal artery. The AV node is connected with the atrial myocardial through the anterior middle and posterior internodal tracts. It is

continued distally as the bundle of His. In addition specialized fibers known as para specific fibers, may leave the AV node and end with intraventricular septum.

5. THE BUNDLE OF HIS — THE COMMON A.V.NODE:

The distal end of the AV node continues as the bundle of His. The external morphology does not reveal a sharp demarcation between the two, the AV node blending imperceptibly with the bundle of His. The internal structure of the AV node, however, differs markedly from that of the bundle of His. The main body of the AV node has a Labyrinthine structure of interweaving strands of cells. The bundle of His is characterized by fibers, which are organized in parallel strands. The bundle of His penetrates the central fibrous body and proceeds anteriorly descending towards the intraventricular septum where it divides into right and left bundle branches. The AV node and the bundle of His are sometimes collectively referred to as 'AV Junction'.

6. THE RIGHT BUNDLE BRANCH:

The right bundle branch is a relatively well defined and easily dissectable structure situated rather more deeply beneath the epicardium than the left main bundle branch. The right bundle branch is 1-2 mm in width and 45-50 mm in length. It passes down the right side of the intraventricular septum and along the free edge of the moderator band to the base of the anterior papillary muscle. It gives off relatively few branches until it reaches the anterior papillary muscle where it begins to ramify, breaking up into a network of small branches.

7. THE LEFT BUNDLE BRANCH:

The left bundle branch is 4-8 mm in length and 8-10 mm in width. The left bundle branch passes down the left side of the intra-ventricular septum and emerges below the posterior cusp of the aortic value. In contrast to the structure of the right bundle

branch, the left bundle branch breaks up almost immediately into a number of small branches or rootlets which proceed onwards in two major sweeps or radiation's.

a) **The anterior - superior division.**

This is the more important division which supplies the antero-superior aspect of the left ventricle — the greater part of the left ventricle. Delay or block within this division will result in a left anterior hemiblock.

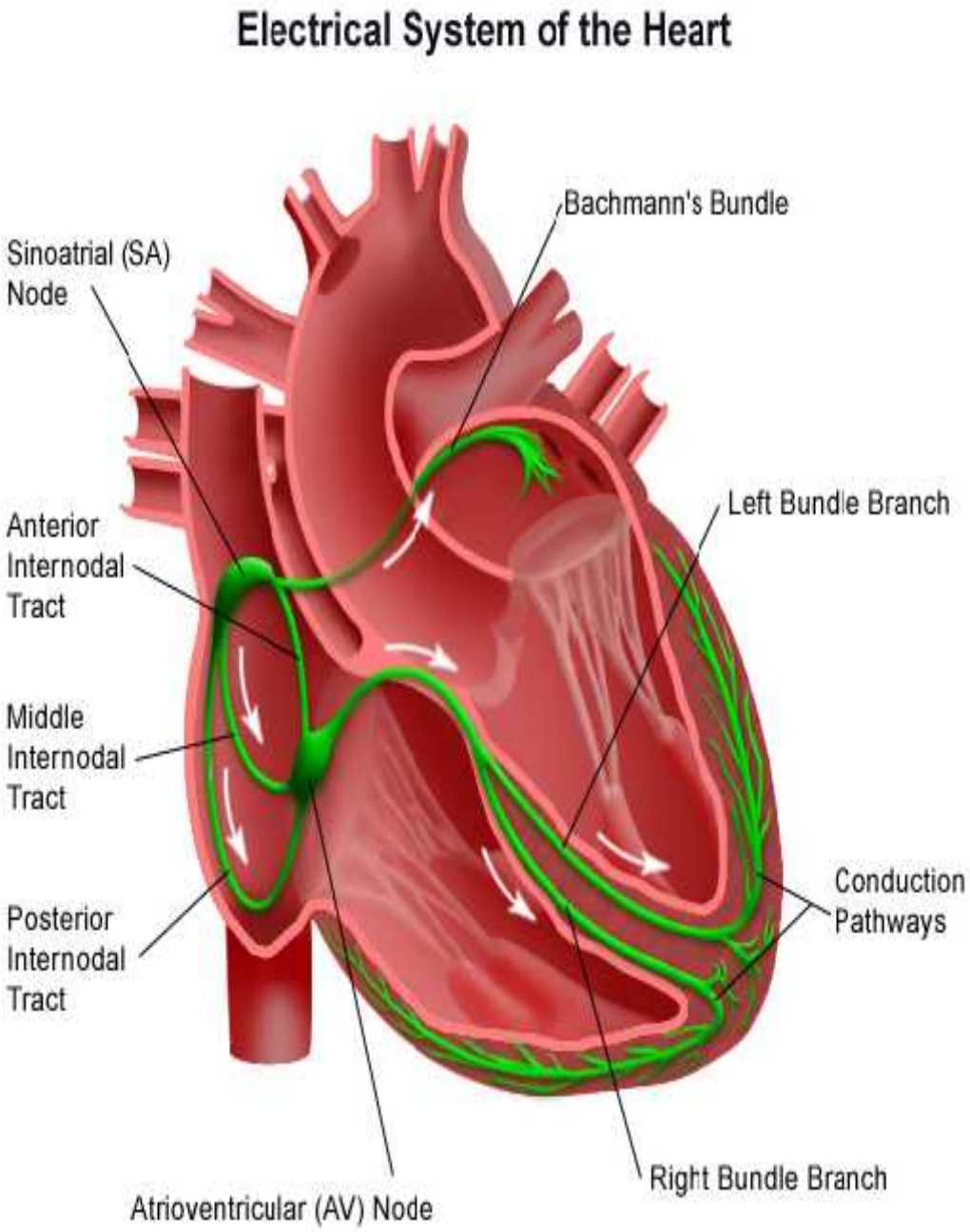
b) The postero-inferior division.

A division which supplies the postero-inferior aspect of the left ventricle. Delay or block within this division will result in a posterior hemiblock.

8. PURKINJE FIBERS

The Purkinje fibers are terminal part of the conducting system of the heart, which are complex fibers and continuous structures from the Right Bundle Branch and Left Bundle Branch. The Purkinje fibers are distributed diffusely in every portion of the ventricles expect for the central upper portion of the ventricle septum.

Fig 1.


Electrical System of the Heart
Bachmann's Bundle
Sinoatrial (SA) Node
Left Bundle Branch
Anterior Internodal Tract
Middle Internodal Tract
Conduction Pathways
Posterior Internodal Tract
Right Bundle Branch
Atrioventricular (AV) Node

Review the vascular supply of the different components of the conduction system:

Sinoatrial node –Supplied by the right coronary artery (RCA) in 60 percent of patients, by the left circumflex artery (LCX) in 40 percent.

Atrioventricular (AV) node – Supplied by the RCA in 90 percent (AV nodal branch); by the LCX in 10 percent of patients.

His bundle – Supplied by the RCA (AV nodal branch) with a minor contribution from the septal perforators of the left anterior descending artery (LAD).

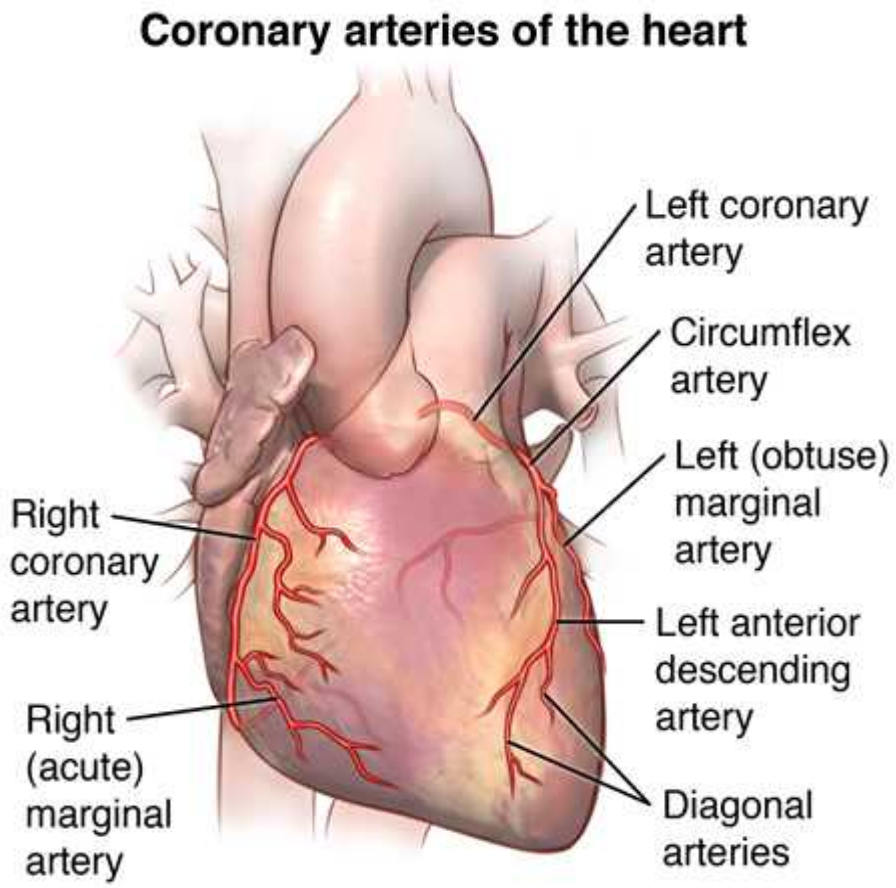
Main or proximal left bundle branch – The LAD provides most of the blood supply for the left bundle branch, particularly for the initial portion. There may be some collateral flow from the RCA and LCX systems.

Left posterior fascicle – The proximal portion of the left posterior fascicle is supplied by the AV nodal artery and, at times, by septal branches from the LAD. The distal portion has a dual blood supply from both anterior and posterior septal perforating arteries.

Left anterior fascicle – The left anterior and mid-septal fascicles are supplied by septal perforators of the LAD and, in about one-half of subjects, by the AV nodal artery.

Right bundle branch – The right bundle branch receives most of its blood supply from septal perforators from the LAD coronary artery, particularly in its initial course. It also receives some collateral supply from either the RCA or LCX coronary systems, depending upon the dominance of the coronary system.

Fig 2



The Autonomic supply of the Heart :

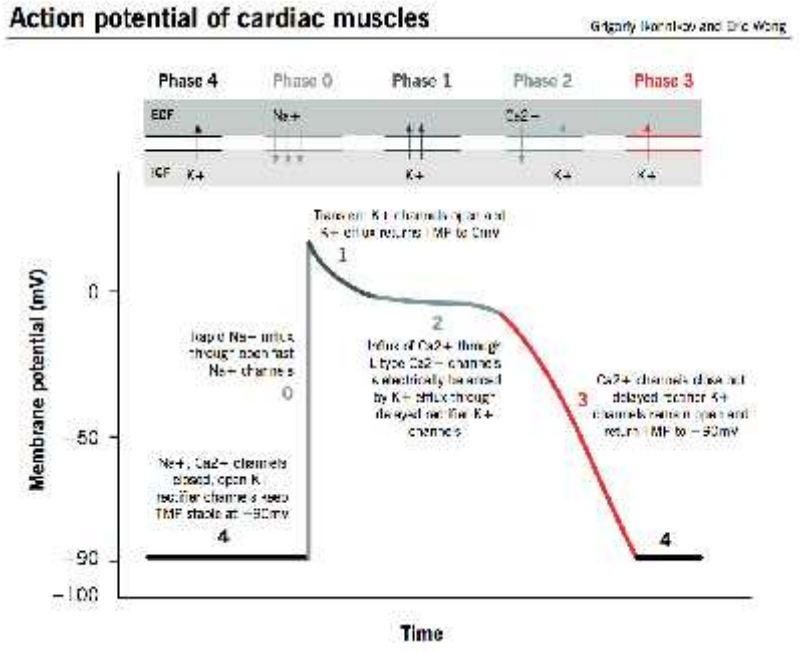
The heart is supplied by both parasympathetic and sympathetic fibers. Parasympathetic innervation consists of branches from the right vagi. The right vagus supplies the Sino-atrial node. The left vagus supplies the atrio-ventricular node. The parasympathetic system does not innervate the ventricles. The sympathetic system innervates both the atria and ventricles.

ELECTROPHYSIOLOGY OF THE HEART[44,48,49]

The knowledge of electrophysiology of cardiac excitation has been greatly extended by direct intra cellular recording. A microelectrode inside a single myocardial fiber paired with an extracellular electrode enables to measure the electrical potential inside and outside the muscle fiber and changes that occur during the activity to be recorded (Hoftman and Cranefield, 1960). The difference in potential is termed transmembrane potential and the changes during the activity are termed action current.

During the resting phase when the fiber is said to be polarized the inside is electrically negative compared with its outside. The resting transmembrane potential is surprisingly large measuring 90mv. During the first phase of action current the transmembrane potential falls rapidly and transiently overshoots (Phase 2), so that the inside of the fiber becomes approximately 30mv positive to the outside. This transient overshoot is followed by a plateau (Phase 3) and then the more gradual downstroke of repolarization (Phase 4) follows.

Fig 3



The ionic concentration of the intracellular fluid of cardiac muscle fibers like that of all living cells is markedly different from that of extracellular fluid. All ions by definition carry electric charges and the resting transmembrane potential largely results from the high concentration of positively charged sodium ions in the extracellular fluid. Since the cell membrane is ordinarily permeable to all ions, it is necessary to explain how the ionic composition of the intracellular fluid is maintained. The generally accepted view is that the cells contain metabolic sodium pumps, which continuously eject sodium ions from the cell. Since this is achieved in phases of both electrical and chemical gradients, active metabolic work is involved. The electrical changes represented by the action current are brought about by rapid refluxes of ions across the cell membrane. It is believed that both depolarization and repolarization are passive events resulting from abrupt changes in the permeability of cell membranes to sodium ions. Sodium ions then rapidly enter the cell as a result of electrochemical gradient and the upstroke of the action current is recorded.

Repolarization is initially achieved by an increase in potassium permeability so that a rapid exit of positively charged potassium ions from the cell occurs and the transmembrane potential is restored. This egress of potassium ions is facilitated by a large chemical gradient which disappearance following depolarization. At the end of action current the inside fibers will have gained sodium ions and lost potassium ions.

Following activity the original differential ionic concentrations are restored by the metabolic pump.

The action current of atrial and ventricular muscle fibers is that the transmembrane potential remains constant during diastole. On the other hand, the action current of pacemaker cells is characterized by slow spontaneous depolarization during diastole. For reasons not clear, once the transmembrane potential has fallen to

threshold value, which is approximately 40 mv, rapid depolarization follows and this automatically fires off a propagated response which will be conducted over the heart. (The AV node is activated early during the P wave at surface electrocardiogram).

The main delay in AV transmission occurs at the atrial margin of the AV node where the conduction velocity has a very low value of 50 mms/sec within the node. Conduction velocity progressively increases to reach 1000 to 1500mms/sec, in the bundle of His. In the Purkinje fibers, velocity reaches 4000mms/sec. During the retrograde conduction from ventricles to atria, the same conduction velocity occurs except at the atrial margin of the node where retrograde conduction is even slower than antegrade.

The development of intra-cardiac electrocardiographic recording and stimulation techniques during the past five years has brought about a renaissance in cardiac electrophysiology. It records electrical activity that is inconspicuous or un-recordable in the surface electrocardiograms. Local electrocardiograms may be recorded in the infarct human heart from the atrial tissue adjacent to the SA node, low intra-atrial septum, coronary sinus, bundle of His, right and left bundle branch.

Amplification of atrial activity that is in apparent in the surface ECG has been accomplished by intra-esophageal lead and intra-atrial electrograms. Recording of the AV conduction system was first discovered by Scherlang and his colleagues in 1969.

DISTURBANCE OF IMPULSE CONDUCTION[50-54]

The abnormalities in conduction known as "heart block" may be classified as: -

1. Sino-atrial block (SA block).
2. Atrio-ventricular block (AV block).
3. Bundle branch block including intra-ventricular block.

I. SINO-ATRIAL BLOCK (SA BLOCK):

In SA block, the sinus impulse is blocked within the SA junction i,e., the junction betweenthe SA node and the surrounding atrial myocardium.

As a result, neither atrial nor ventricular activation takes place, and no P wave or QRS complex is recorded in this form of exit block. SA block usually occurs irregularly and unpredictably as isolated instances.

There are three types of SA Block :

1) First degree Sino-atrial exit block.
2) Second degree Sino-atrial exit block.
3) Third degree Sino-atrial exit block.

1) First degree Sino-atrial block.

In this there is prolonged conduction time from the sinus node to the surrounding atrial tissue. It cannot be recognized on a standard ECG, but requires invasive procedures.

2) Second degree Sino-atrial block.

In this there is intermittent failure of conduction of sinus impulses to the surrounding atrial tissue. It is manifested as the intermittent absence of P wave.

3) Third degree or complete sino-atrial block.

This is characterized by a lack of atrial activity or by the presence of an ectopic subsidiary atrial pacemaker.

II.ATRIO-VENTRICULAR BLOCK (AV BLOCK)

Atrioventricular (AV) block is defined as a delay or interruption in the transmission of an impulse from the atria to the ventricles due to an anatomic or functional impairment in the conduction system. The conduction disturbance can be transient or permanent, with conduction that is delayed, intermittent, or absent. Commonly used terminology includes:

i. First degree AV block – Delayed conduction from the atrium to the ventricle (defined as a prolonged PR interval of >200 milliseconds) without interruption in atrial to ventricular conduction.
ii. Second degree AV block – Intermittent atrial conduction to the ventricle, often in a regular pattern (eg, 2:1, 3:2), or higher degrees of block, which are further classified into
    - Mobitz type I (Wenckebach)
    - Mobitz type II second degree AV block.
iii. Third degree (complete AV) block – No atrial impulses conduct to the ventricle.
iv. High-grade AV block – Two or more consecutive blocked P waves.

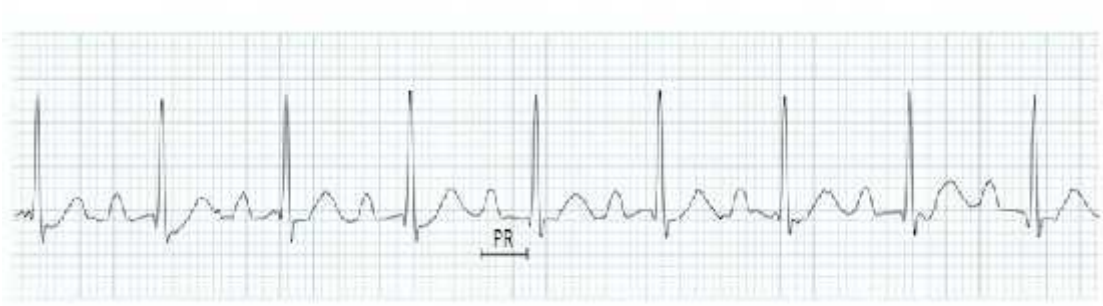
i. First degree AV block:

It is a delay in conduction through the AV conducting system. It is reflected by a prolonged PR interval beyond the normal limit of 0.20 sec.

ECG abnormalities

- õ PR interval greater than 0.20 sec
- õ Both P and QRS are morphologically normal.

Electrocardiogram (ECG) showing type I atrioventricular (AV) block and (Figure 4)



ii. Second degree AV block:

It is an intermittent failure or interruption of AV conduction. The sinus impulses after leaving SA node and activating atria to produce P wave are blocked in the AV conducting system. Thus there are regularly occurring P waves some of which are not followed by QRS complexes. The sinus impulses may be blocked at regular or irregular intervals and blocked impulse may be preceded by a constant or progressively increasing AV conduction times (PR Interval).

Types of second degree AV block

1)Mobitz type 1 AV block

2)Mobitz type 2 AV block

1) Mobitz type 1 AV block (Wenckebach phenomenon) .

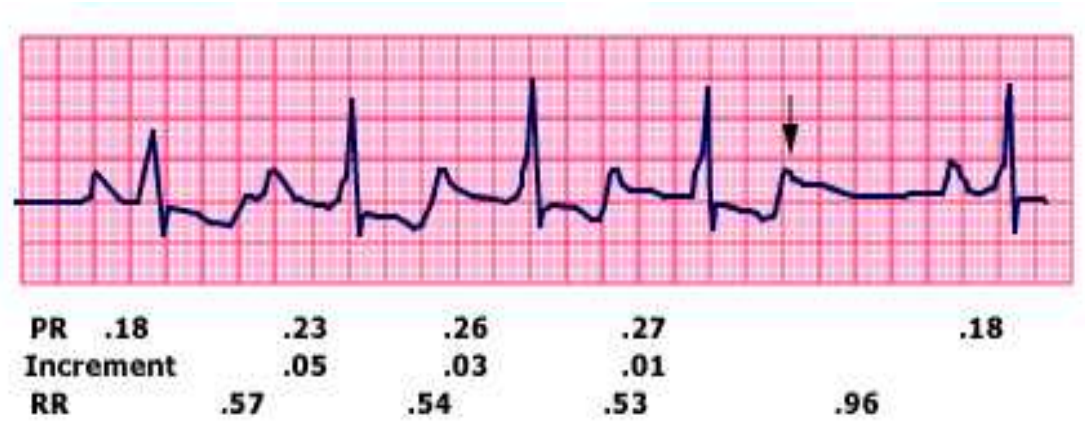
In this type transmission through the conducting system becomes increasingly difficult until it fails completely and a beat is dropped. The pause

occasioned by the dropped beat allows the conducting system to recover and the sequence is repeated. The defect is usually in the AV node.

ECG abnormalities

- Sequential/gradual prolongation of PR interval terminated by a non conducted P wave.
- Prolongation of RR interval occurs in progressively shorter increments.
- Duration of pause following the non conducted P wave is less than the
- Sum of any two consecutively conducted beats.
- Decreased PR interval following the pause when compared to prepause PR interval.
- Grouped beating, a pattern of repeated groups of QRS complexes.

Electrocardiogram (ECG) showing Mobitz type I (Wenckebach) Atrioventricular (AV) block (Figure 5)



Single lead electrocardiogram (ECG) showing Mobitz type I (Wenckebach) second degree AV block with 5:4 conduction. The characteristics of this arrhythmia include: a progressively increasing PR interval until a P wave is not conducted (arrow); a progressive decrease in the increment in the PR interval; a progressive decrease in the RR interval; and the RR interval that includes the dropped beat (0.96 sec) is less than twice the RR interval between conducted beats

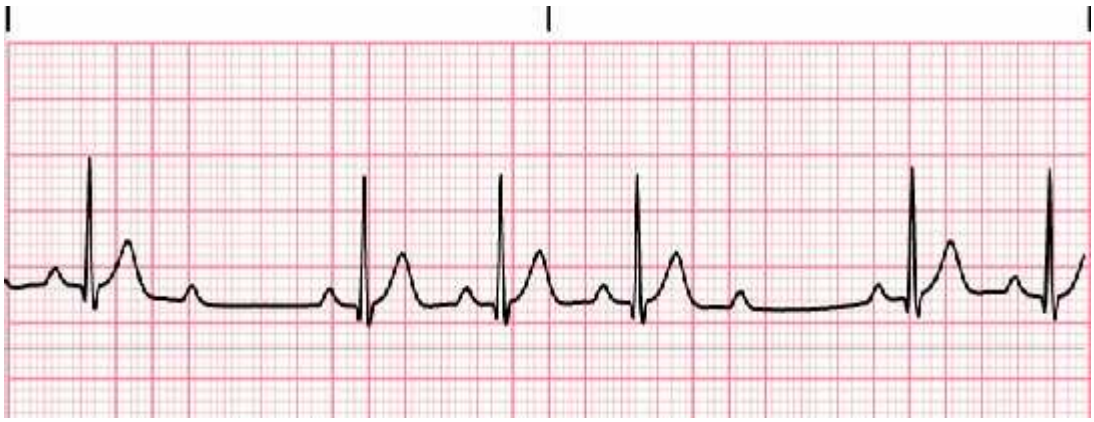
2) Mobitz type II AV block

In this type transmission through AV conduction system fails abruptly. The defect is usually situated in the bundleofHis

ECG abnormalities:

- PR interval of all the conducted supraventricular impulses is constant with a sudden non conducted P wave.
- Each QRS may have multiple P waves
- Associated QRS complex in frequently abnormal.

Mobitz Type II blocks, the impulse is blocked in the bundle of His. Every few beats there will be a missing beat but the PR Interval will not lengthen. (Figure 6)



iii. Third degree AV block (Complete AV block)

It is characterized by a complete or permanent interruption of AV conduction that is all supra-ventricular impulses are blocked within the conducting system. The atria are thus activated by one pacemaker (usually sinus node) and ventricles by another (idionodal / idioventricular) pacemaker.

The two rhythms are independent and asynchronous so that there is complete dissiciation of atrial and ventricular electrical activity.

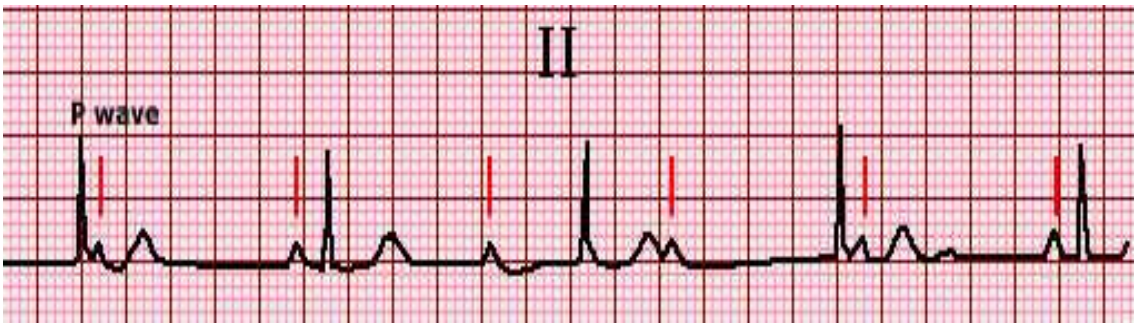
ECG Abnormalities

- **AV dissociation**: P waves bear no relationship with QRS complexes.
- Slow ventricular rate 30-35bpm with idioventricular and 35-40bpm with idionodal pacemaker. With pacemaker in lower AV node (Below the block) or bundle of His, QRS complexes are normal or near normal in shape as ventricular activation occurs via normal pathway. With pacemaker in ventricular musculature activation of ventricles is bizarre and QRS is abnormal (broad, notched, slurred and bizarre).

  3rd degree Complete heart block. The atrial rate is faster than ventricular rate, and no association exists between the atrial and ventricular activity.

Third degree (complete) atrioventricular block with narrow QRS escape rhythm

(Figure 7)



The P waves are completely dissociated from the QRS complexes. The QRS complexes are narrow, indicating a junctional escape rhythm. The atrial and ventricular rates are stable; the former is faster than the latter.

III. INTRAVENTRICULAR BLOCKS

Normal ventricular depolarization occurs after an impulse traverses the atrioventricular (AV) node and the bundle of his. This bundle of specialized conducting tissue splits into two main branches, the right and left bundles, that rapidly transmit depolarization impulses to the right and left ventricular

myocardium, respectively, via the purkinje fibers. The main left bundle bifurcates into two primary subdivisions: a left anterior fascicle and a left posterior fascicle. Any delay in this conduction pathway leads to intraventricular blocks, classified as

1)Right Bundle Branch Block

a) Complete Right bundle branch block

b) Incomplete right bundle branch block

2)Left Bundle Branch block

a) Complete left bundle branch block

b) Incomplete bundle branch block

3)Fascicular blocks

a) Left anterior fascicular block

b) Left posterior fascicular block

c) Bifascicular block

d) Trifascicular block

Right bundle branch block:

It results from conduction delay in any part of the right sided conduction system.

Sequence of ventricular activation in RBBB[55]

Activation starts in left lower third of inter-ventricular septum progressing from left to right (r wave in V1 and q wave in V5, V6). This is followed by activation of paraseptal area of LV and then LV free wall form endocardial to epicardial surface transversely (S wave in V1 and R wave in V5, V6). Now activation front jumps, inter-ventricular septal barrier, and activates RV paraseptal area and free wall slowly with variable participation of specialized

conduction system by conduction from muscle cell to muscle cell in longitudinal direction (Slurred, bizarre R` wave in V1 and S wave in V5, V6, prolongation of QRS duration) LV recovers earlier then RV so recovery vectors are directed towards left and away from right (negative ST-T waves in V1).

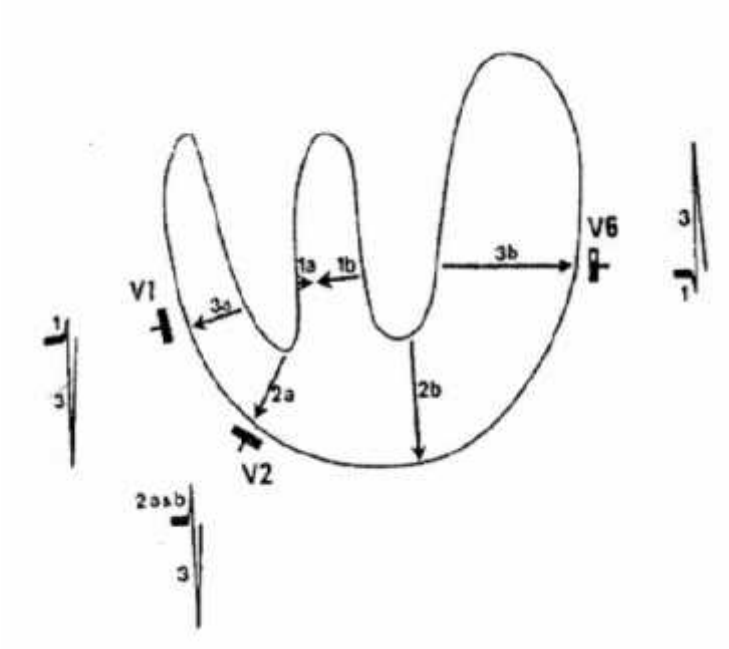


Fig 8: Normal intra ventricular conduction

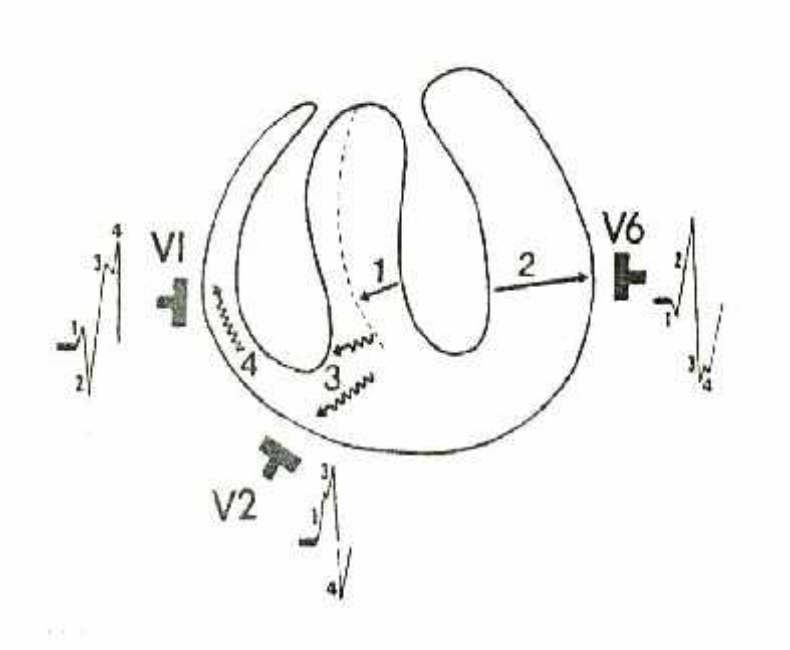
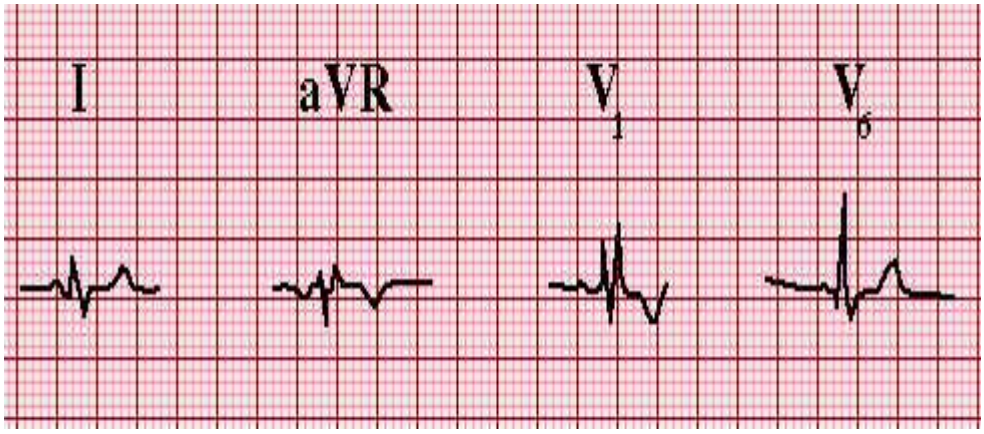


Fig 9: Mechanism of RBBB

COMMON DIAGNOSTIC CRITERIA FOR COMPLETE RBBB[55-57]

- QRS duration $\geq$ 0.12 sec.
- Broad, notched R waves (rsr`, rsR`, rSR` patterns) in right precordial leads
- (V1, V2).
- Wide and deep S wave in left precordial leads (V5, V6) and lead I.
- R peak time >0.05 sec in VI.

Complete right bundle branch block (Figure 10)



The initial myocardial activation is normal; thus, there is a normal septal q wave in leads 1 and V6, followed by a tall R wave. Similarly, there is a normal initial septal R wave followed by a deep S wave in leads aVR and V1. However, the subsequent abnormal right ventricular activation occurs from left to right and goes through the ventricular myocardium instead of the His-Purkinje system; thus, there will be a tall and broad secondary R wave (R') in leads aVR and V1 (a RSR' complex), and a deep and broad S wave in leads I and V6. The width of the QRS complex is >0.12 sec.

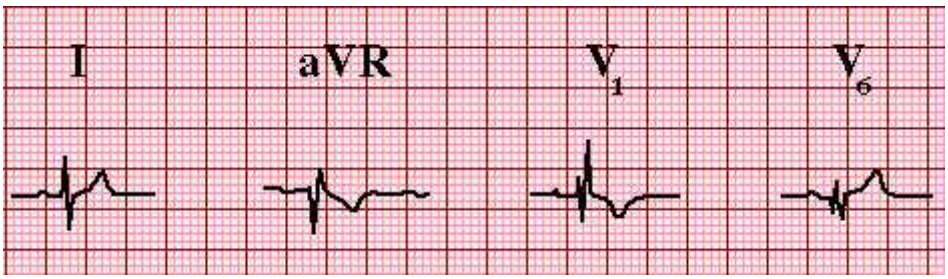
Incomplete RBBB[56]

Here transmission through right bundle is not totally interrupted but delayed

Common diagnostic criteria for incomplete RBBB

- QRS duration 0.1 – 0.12 sec
- rSr`, rsR`, rSR` or M shaped (R>R) pattern in VI
- Deep wide S wave in leads I, V5, V6.

Incomplete right bundle branch block (Figure 11)



Delayed conduction through the right bundle branch does not affect the initial portion of the QRS complex (septal activation), but alters the terminal portion. There is an rsR' pattern in leads aVR and V1, and a qRs morphology in leads I and V6. The width of the QRS complex is only slightly prolonged, between 0.10 and 0.12 seconds(figure 11)

Right bundle branch block with acute myocardial infarction [50]

The effect of right bundle branch block (RBBB) must be considered in both Q wave (ST elevation) and non-Q wave (non-ST elevation) infarctions.

Q wave MI — Right bundle branch block does not usually interfere with the diagnosis of a Q wave myocardial infarction (MI). MI most often involves the left ventricle and therefore affects the initial phase of ventricular depolarization, sometimes producing abnormal Q waves. In contrast, right bundle branch block (RBBB) primarily affects the terminal phase of ventricular depolarization, producing a wide R' wave in the right chest leads and a wide S wave in the left chest leads.

These changes are due to delayed depolarization of the right ventricle, while depolarization of the left ventricle is not affected.

The net effect is that the ECG patterns are combined when complete RBBB and a Q wave infarct occur together, and the criteria for the diagnosis of a Q wave MI are the same as in patients with normal conduction:

- Due to the bundle branch block, the QRS complex will be abnormally wide (0.12 sec or more), lead V1 will show a terminal positive deflection, and lead V6 will show a terminal negative deflection (wide S wave).
- If the infarction is anterior, there will be a loss of R wave progression with abnormal Q waves in the anterior leads and characteristic ST-T changes; if the infarction is inferior, Q waves will appear in leads II, III, and aVF.

Non-Q wave MI — There may be some diagnostic difficulties in interpreting the ECG in patients with RBBB who have a non-Q wave MI. RBBB is typically associated with secondary ST-T changes due to abnormal right ventricular repolarization. Thus, leads with an R' wave (leads V1, V2, and sometimes V3) will show T wave inversions. In contrast, ST depressions or T wave inversions in leads with a terminal S wave (leads V5 and V6) cannot be attributed to the RBBB alone. Such ST-T changes may be due to ischemia, or to other factors such as drug effects (digoxin) or electrolyte abnormalities, such as hypokalemia. Note that the Brugada pattern may simulate RBBB with acute or evolving ST elevation MI.

Left bundle branch block (LBBB)

It results from delay in any part of the left sided conduction system.

Sequence of ventricular activation in LBBB[55]

Initial right septal activation occurs from right to left followed by activation of RV free wall (QS complex or small r wave in VI).

Then activation front jumps interseptal physiological barrier to activate left side of septum in a delayed and anomalous manner and still later activates superior portions of interventricular septum (R wave in lateral leads). Septal activation is followed by delayed and anomalous activation of LV free wall by spread of activation wave from muscle cell to muscle cell (R` in lateral leads, S wave in V1 and prolonged QRS duration). RV recovers earlier then LV so recovery forces are directed right (negative ST-T waves in lateral leads).

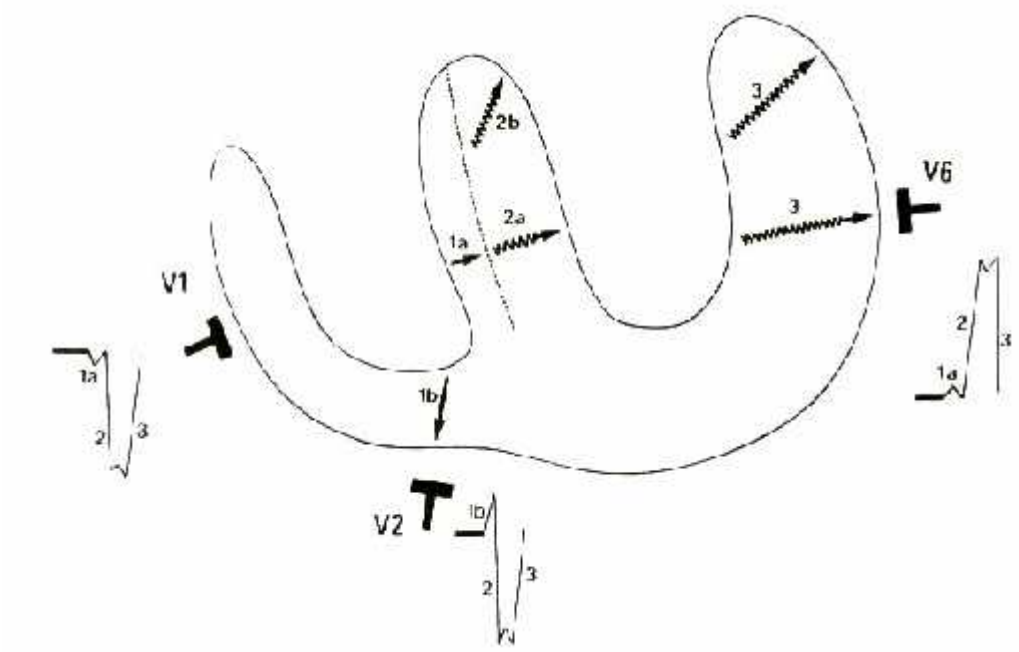
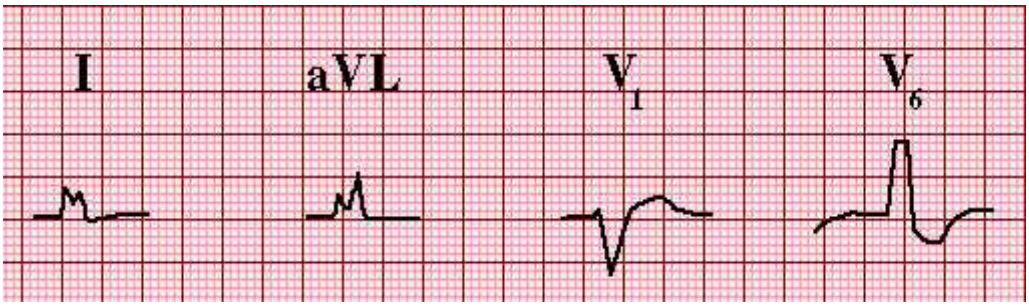


Fig 12: Mechanism of LBBB

Common diagnostic criteria for LBBB[56]

- QRS duration ≥ 0.12 sec
- Broad, notched slurred R waves in lateral precordial leads (V5, V6) and leads
- 1 and avL.
- Small or absent initial r waves followed by deep broad S waves in right
- precordial leads (V1, V2).
- Absent septal q waves in left sided leads
- Prolonged intrinsicoid deflection (>0.060 sec) in V5 and V6.
- Transition zone displaced to left.

Left Bundle Branch Block (Figure 13)



Delayed and abnormal activation of the left ventricular myocardium, and a diffuse slowing of conduction throughout the left ventricle leads to the following changes on the ECG: there is a tall monophasic and broadened R wave in leads I, aVL, and V6 instead of a septal Q wave; there is a QS complex which is abnormal and widened in V1 instead of a small initial R wave due to septal activation; the QRS interval is prolonged >0.12 seconds; myocardial repolarization changes including T wave inversion and ST segment depression are evident.

Incomplete LBBB [56]

It results from lesser degrees of conduction delays in LBB system, much of LV activation occurs through the normal conduction system.

Common diagnostic criteria.

- Loss of septal q waves in 1, V5, V6
- Slurring and notching of upstroke of R wave in left precordial leads.
- QRS duration 0.1-0.12 sec.
- R peak time $\geq$ 0.06 sec in left precordial leads.

Left bundle branch block with acute myocardial infarction[50]

Left bundle branch block (LBBB) is present in approximately 7 percent of acute infarctions[58]. The diagnosis of myocardial (MI) in the presence of LBBB is considerably more complicated and confusing than that of right bundle branch block (RBBB). The reason is that LBBB alters both the early and the mid to late phases of ventricular depolarization, and also produces secondary ST-T changes **.**

Acute MI — The sequence of repolarization is altered in LBBB, with the ST segment and T wave vectors being directed opposite to the QRS complex. These changes may mask the ST segment depression and T wave inversion induced by ischemia. On the other hand, the diagnosis of an acute MI or ischemia can occasionally be made in a patient with underlying LBBB if certain ST-T changes are seen, particularly if the ST-T vectors are in the same direction as the QRS complex as in the Sgarbossa criteria described below.

Sgarbossa criteria — A large, historic trial of thrombolytic therapy for acute MI (GUSTO-1) provided an opportunity to revisit the issue of the electrocardiographic diagnosis of evolving acute MI in the presence of LBBB[59] Among 26,003 North American patients who had a myocardial infarction confirmed by enzyme studies, 131 (0.5 percent) had LBBB. A scoring system, often called the

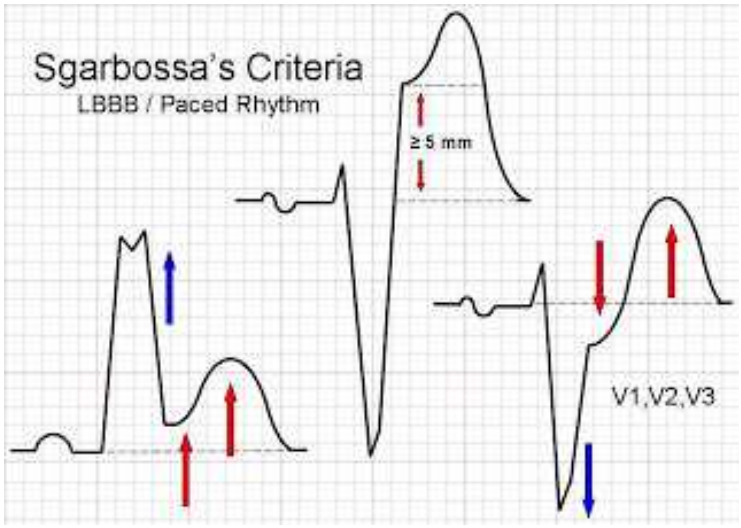
Sgarbossa criteria, was developed from the coefficients assigned by a logistic model for each independent criterion, on a scale of 0 to 5.

The three ECG criteria with an independent value in the diagnosis of acute infarction and the score for each were:

- ST segment elevation of 1 mm or more that is in the same direction (concordant) as the QRS complex in any lead – score 5.
- ST segment depression of 1 mm or more in any lead from V1 to V3 – score 3.
- ST segment elevation of 5 mm or more that is discordant with the QRS complex (ie, associated with a QS or rS complex) – score 2.

A Sgarbossa score of ≥3 was highly specific (ie, few false positives) but much less sensitive (36 percent) in the validation sample in the original report[59]. The sensitivity may increase if serial or previous ECGs are available[60.]

(Figure 14)



Cabrera's sign refers to prominent (0.05 sec) notching in the ascending limb of the S wave in leads V3 and V4; a similar finding is prominent notching of the ascending limb of the R wave in lead V5 or V6 (Chapman's sign)[61]. These signs have a specificity that approaches 90 percent. However, there may be a high degree of inter observer variability in accurate identification and their sensitivity is quite low.

Fascicular blocks

It is delay or block in conduction within one of the two major divisions of the left bundle branch. They are:

Left anterior fascicular block

Left posterior fascicular block

Bifascicular block

Trifascicuclar block

Left anterior fascicle block

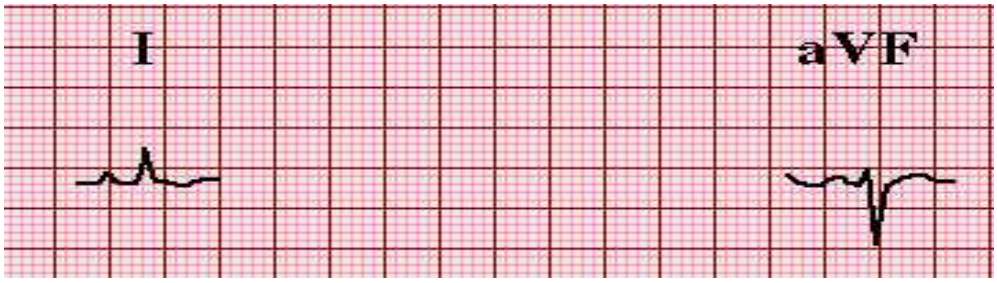
Sequence of LV activation in LAFB [55]

Initial activation occurs in inferior septal mass, inferior region of LV free wall (resulting in initial forces directed inferiorly and to right). Later delayed activation of anterolateral region of free LV wall occurs (resulting in dominant and terminal QRS forces directed superiorly and to left).

Common diagnostic criteria for LAFB[56]

- Frontal plane mean QRS axis of – 45 to 90 degrees
- QRS duration <0.120 sec
- rS pattern in leads II, III, avF
- qR pattern in lead 1, avL
- R peak time in 1, avL$\geq$ 0.045 sec.

Left anterior fascicular block



An upright QRS complex in lead I and negative QRS complex in aVF is characteristic of a left anterior hemiblock. The QRS duration is normal (figure 15)

Left posterior fascicle block

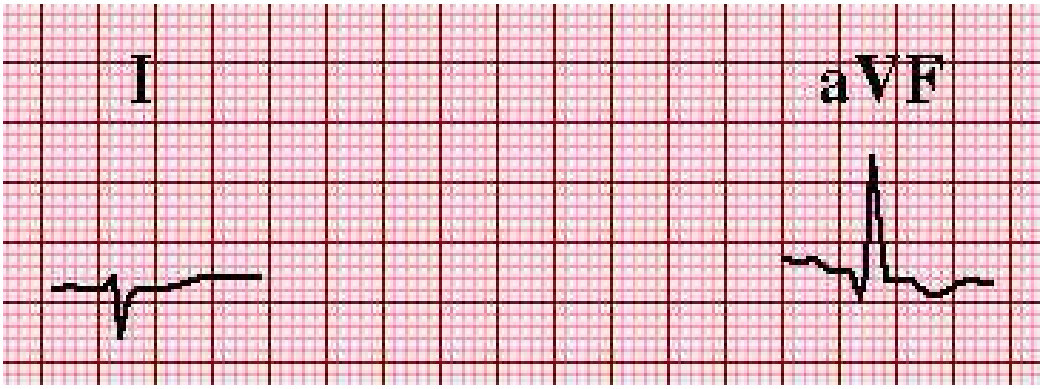
Sequence of LV activation in LPFB[55]

Activation starts in anterosuperior LV free wall followed by activation of inferioposterior aspect of LV.

Common diagnostic criteria for LPFB[56]

- Frontal plane mean QRS axis (>120)
- rS pattern in leads 1, avL
- qR pattern in leads II, III, avF (1 wave≤ 0.04 sec)
- QRS duration <0.12 sec
- Exclusion of other factors causing Right axis deviation

Left posterior fascicular block(Figure 16)



A negative QRS complex in lead I and positive QRS complex in aVF is characteristic of a left posterior hemiblock. The QRS duration is normal

Bifascicular blocks[56]

The combination of right bundle branch block with either left anterior or posterior divisional block or the combination of left anterior and let posterior divisional blocks(i.e. left bundle branch block) is known as bidivisional or bifascicular block.

RBBB with LAFB [55,56]

Sequence of ventricular activation:

Initial activation begins in posterior paraseptal area of ventricle (forces directed inferiorly, anteriorly and to right). The impulse then spreads through inferior wall and apex (forces directed inferiorly leftward). Subsequently activation occurs in anterolateral and posterobasal regions (with forces directed posteriorly, leftwards, superiorly). Later activation occurs in right septum and right ventricular free wall (with forces directed anteriorly and to right).

Common diagnostic criteria

- QRS duration > 0.12 sec
- QRS axis of –45 to -90
- qR pattern in lead avL
- Right precordial leads show rsr`, rsR`, rSR` or M shaped pattern with R`>R
- Wide S wave in leads I, V5, V6 and S wave duration > R wave or >0.40 sec

RBBB with LPFB [55,56]

Sequence of ventricular activation

Initial activation occurs in anterior paraseptal area of LV, then anterior and anterolateral wall (with vectors directed anteriorly, superiorly and left ward). Activation then proceeds in retrograde way to inferior and posterobasal areas of

LV (with forces directed inferiorly and to right). Later delayed activation of right septum and RV free wall occurs (with forces directed anteriorly and to right).

Common diagnostic criteria

- QRS $\geq$ 0.12sec
- Frontal mean QRS axis +90° to + 180°
- rS pattern in leads 1 and avL
- qR pattern in leads II, III and avF (Q wave duration$\leq$ 0.04 sec)
- rsr`, rsR`, rSR` or M shaped right precordial pattern with R`>R
- Wide S wave (duration > 0.04 sec) in leads I, avL, V5 and V6
- R peak time >0.05 s in V1 with normal in V5, V6.

Trifascicular blocks[50]

It is simultaneous block (complete or incomplete) in any three of the five ventricular conducting fascicles – Bundle of His, Right bundle branch, Left bundle branch, Left anterior fascicle and Left posterior fascicle.

The term trifascicular block is used specifically when block simultaneously involves three peripheral fascicles – right bundle branch, left anterior fascicle and left posterior fascicle. It usually results in bifascicular block (RBBB with LAFB or LPFB) with first or second degree AV block.

CONDUCTION DISTURBANCES BASED ON INFARCT LOCATION

Inferior MI — Conduction disturbance in inferior MI can occur acutely or after hours or days. Sinus bradycardia, Mobitz type I (Wenckebach), and complete heart block (CHB) are commonly seen, since the sinoatrial (SA) node, AV node, and His bundle are primarily supplied by the right coronary artery (RCA) [62]

- Sinus bradycardia is the most common arrhythmia associated with inferior MI. It is present in up to 40 percent of patients in the first two hours, decreasing to 20 percent by the end of the first day. It is usually attributable to increased vagal tone in the first 24 hours after infarction. Transient sinus node dysfunction occurring later may be due to sinus node or atrial ischemia.
- First degree AV block can arise in the AV node, the bundle of His, or the bundle branches. It is common after occlusion of the coronary artery (right or circumflex) which gives rise to the AV nodal artery. RCA occlusion can lead to first degree AV block via ischemia of the AV node, by enhanced acetylcholine release from the inferoposterior myocardium, or perhaps by making the AV node hypersensitive to the action of acetylcholine. First degree AV block due to occlusion of the RCA with involvement of the AV node is usually transient, generally resolving in five to seven days and requiring no therapy.

Occlusion of the left circumflex artery, which often manifests as an inferior MI on the electrocardiogram, may affect the AV node directly in the 10 percent of individuals in whom it supplies the AV node.

- Inferior MI is typically associated with the more benign second degree AV block of the Wenckebach type (Mobitz type 1). Mobitz type II is uncommon

in this setting, generally occurring with anterior MI .Mobitz type I block is usually transient, resolving in most cases within five days.[63]

Complete AV block (CHB) with inferior MI generally results from an intranodal lesion. It is associated with a narrow QRS complex, and develops in a progressive fashion from first to second to third degree block. It often results in an asymptomatic bradycardia (40 to 60beats/min) and is usually transient, resolving within five to seven days. [64]

Anterior MI — Conduction disturbances occurring with anteroseptal MI are less frequent but more serious, and the degree of arrhythmic complications is usually directly related to the extent of infarction.

- First degree AV block – Prolongation of the PR interval due to slowed AV nodal conduction does not occur in anterior MI, since the AV node is usually supplied by the right or circumflex coronary artery, as opposed to the left anterior descending coronary artery, which is the cause of anterior infarctions in most cases.
- High (second or third) degree AV block – Second degree AV block with anterior MI is usually at the level of the AV node or below and is almost exclusively a Mobitz type II block. The clinical course may be unpredictable, with CHB developing with little warning.

Complete heart block with anterior MI generally occurs abruptly in the first 24 hours. It can develop without warning or may be preceded by the development of right bundle branch block with either a left anterior fascicular block or left posterior fascicular block (bifascicular or trifascicular block)[65]. The escape rhythm is wide and unstable and the event is associated with a high mortality from both arrhythmias and

pump failure. Heart block in this setting is thought to result from extensive necrosis that involves the bundle branches traveling within the septum [65.]

Less commonly, anterior MI produces first degree AV block below the level of the AV node, a situation that should be suspected if first degree AV block occurs in the presence of a widened QRS complex

MANAGEMENT OF AV BLOCK

Atrioventricular (AV) block may be asymptomatic or symptomatic. An important consideration in the setting of an acute myocardial infarction (MI) is that a bradycardia, even if asymptomatic or transient, can cause decreased coronary blood flow and reduced myocardial perfusion. The most commonly used therapies for symptomatic bradycardia in the setting of acute MI are atropine or temporary transcutaneous or right ventricular pacing.

Atropine — Bradyarrhythmias that occur early in the setting of an inferior MI (within the first 24 hours) may respond to atropine, while those occurring later are often atropine resistant [62,66]

Treatment is not indicated in asymptomatic patients, but is useful if symptoms are present such as dizziness, syncope, or confusion from reduced cardiac output. Atropine is administered in 0.5 or 1 mg increments to a total of 3 mg. Caution should be used with administering atropine in the setting of active ischemia or in the setting of bundle branch block (BBB) or Mobitz Type 2 AV block because precipitation of ventricular fibrillation has been described [67]

Refractory hypotension after correction of the bradycardia with atropine usually indicates volume depletion or concurrent right ventricular infarction. Volume infusion is the treatment of choice in this setting.

Temporary transvenous pacing — Reperfusion strategies have significantly reduced the need for temporary and permanent cardiac pacing since there is often less myocardial damage and a lower chance that bradycardia and conduction abnormalities will occur. Temporary transcutaneous or transvenous pacing is necessary for many patients with highly symptomatic bradyarrhythmias and maybe considered for those at high risk of developing CHB as a consequence of acute MI.[68]

Temporary transvenous pacing can be considered in the following circumstances:

- Complete (third-degree) AV block.
- Alternating right and left bundle branch block (LBBB), or right bundle branch block (RBBB) with alternating left anterior fascicular block (LAFB) or left posterior fascicular block (LPFB).
- New or age-indeterminate bifascicular block (RBBB with LAFB or LPFB or LBBB) with PR prolongation.
- Asystole.
- Symptomatic bradycardia of any etiology, including sinus bradycardia and Mobitz type I second degree AV block, if hypotension is present and the bradyarrhythmia is not responsive to atropine.
- Mobitz type II second-degree AV block.
- Bradycardia-induced tachyarrhythmias, such as torsades de pointes.

Third Universal definition of myocardial infarction.[69]

Criteria for acute myocardial infarction

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

I. Detection of a rise and/or fall of cardiac biomarker values[preferably cardiac troponin t [cTn] with at least one value above the 99th percentile upper reference limit[URL] and with at least one of the following:
   - Symptoms of ischemia
   - New or presumed new significant ST-T wave changes or new left bundle branch block(LBBB)
   - Development of pathological Q waves in ECG.
   - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
   - Identification of an intracoronary thrombus by angiography or autopsy.

II. Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased

III. PCI related MI is arbitrarily defined by elevation of cTn values (>5 * 99th percentile URL) in patients with normal baseline values (<= 99th percentile URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either
   - Symptoms suggestive of myocardial ischemia

     or

- New ischemic ECG changes

  or

- Angiographic findings consistent with a procedural complications

  or

- Imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required

IV. Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and fall of cardiac biomarkers values with at least one value above the 99^{th} percentile of URL

V. Coronary artery bypass grafting related MI is arbitrarily defined by elevation of cardiac biomarker values ($>10*99^{th}$ percentile URL) in patients with normal baseline cTn values ($\leq 99^{th}$ percentile URL). In addition, either

- New pathological Q wave or new LBBB,

  or

- Angiographic documented new graft or new native coronary artery occlusion,

  or

- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Criteria for prior myocardial infarction

Any one of the following meets the diagnosis of prior myocardial infarction

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

WHO definition and criteria for Acute Myocardial Infarction (AMI) [70]

According to the WHO criteria as revised in 2000, a cardiac troponin rise accompanied by either typical symptoms, pathological Q waves, ST elevation or depression or coronary intervention are diagnostic of MI. A patient is diagnosed with myocardial infarction if two (probable) or three (definite) of the following criteria are satisfied:

1. Clinical history of ischemic type chest pain lasting for more than 20 minutes
2. Changes in serial ECG tracings
3. Rise and fall of serum cardiac biomarkers such as creatinine kinase-MB fraction and troponin

Table 1: RISK FACTORS FOR CORONARY ARTERY DISEASE [69]

Modifiable	Non modifiable
Hypertension	Male age > 25 years
Smoking / tobacco abuse	Female age > 45 years
Diabetes mellitus / insulin resistance syndrome	Family history of premature CAD (at age <55 years)
Obesity / BMI > 22	
Homocysteine > 10 micro mol/L	
High PAI – 1	
Lipid profile	Total Cholesterol >150 mg%
	Triglycerides > 150 mg%
	LDL : > 100 mg%
	Apo (B) : > 100 mg%
	O9 HDL Males < 40 mg%
	Females < 50 mg%
	TC/HDL > 4.5
	LDL/HDL > 3.5

Clinical Features of Acute Myocardial Infarction [72-74]

Acute myocardial infarction presents itself as a sudden catastrophic incident and its definite clinical picture may be established without warning.

1. The clinical picture can be classified as following

1. Cases presenting with chest pain.
2. Cases presenting with shock.
3. Cases presenting with pulmonary edema of other evidence of L.V. failure.
4. Cases presenting with the gradual development of CCF.
5. Cases presenting other complication.

Chest pain

In 80-85% of cases, this is a presenting complaint. It is a deep visceral pain, involving the central portion of the chest and epigastrium, described as tightness, heaviness or constriction in the chest. In 25% of cases it radiates to the arms and less commonly it may radiate to epigastrium, back, jaw and neck. The pain is often accompanied by fatigue, sweating, nausea, giddiness and anxiety. It rarely occurs during exertion or emotional outbursts, not relieved with rest & makes the patient to move about in an attempt to find a comfortable position.

Breathlessness

Secondmost important symptom, breathlessness may be sudden in onset or if may be exertional. It is common in those who had "painless myocardialinfarction" particularly diabetics and aged individuals and those having complication like cardiogenic shock and pulmonary edema.

Other presenting symptoms.

Sudden loss of consciousness, a confusion state, a sense of profound weakness or unexplained fall in blood pressure with giddiness, syncope and or convulsions may be a presenting complaint. Some patients present with gradual onset of breathlessness. Paroxysmal nocturnal dyspnea and pain in abdomen with oliguria and swelling of lower limbs, a picture that of congestive cardiac failure. In rare cases, the infarct may go unrecognized until endocardial thrombosis resulting from it leads to systemic embolism.

Physical sign:

Most commonly, patient will be anxious, restless, sweating and attempting to relieve the pain by moving about in bed stretching, belching or even including vomiting. Patient may come with the hand on his precordium indicating the site of

maximum intensity of pain (Levine sign). Pallor is common, often associated with perspiration and coolness of extremities. Cyanosis may be there, when the patient is having severe pulmonary edema or cardiogenic shock.

Pulse

It may show bradycardia, normal sinus rhythm, tachycardia with or without irregularities, depending upon the presence or absence of arrhythmias and the type of arrhythmia.

Blood pressure

Usually shows an initial rise because of pain, anxiety or unfamiliarity of the environment, which will become normal within 3 or 4 days. Fall in the blood pressure may be due to cardiogenic shock, arrhythmia or due to `BezaldJarisch reflex, which is due to increased vagal tone that occurs in inferior wall infarction.

J.V.P. (Jugular Venous Pressure)

May be elevated when the patient is first seen, which may be due to cardiac failures or to anxiety. Collapse of neck veins occurs when that patient is in shock. Cannon a-waves can be made out in complete heart block, in which they are irregular and in nodal tachycardia, in which they are regular.

Precordium

Is usually quiet and the apical impulse may be difficult to palpate. In about one fourth of the patients with anterior wall infarction, an abnormal systolic pulsation develops in the periapical area within the first few days of illness, which may resolve later, which represents a transient, palpable systolic bulging of the infracted ventricle. Other physical signs of ventricular dysfunction that may be present are muffled heart sounds, atrial (S_4) and ventricular (S_3) gallop sounds and paradoxical splitting of the 2^{nd} sound. A transient apical systolic murmur due to

mitral regurgitation secondary to papillary muscle dysfunction during acute infarction may occur. A pericardial friction rub is audible, in most cases of transmural infarction. Temperature elevations in the range of 37 to 38°C are common during the first 3 to 4 days due to myocardial necrosis.

Respiratory system

Tachypnea is common and crepitations are heard at the base or all over the lung fields depending upon the amount of pulmonary congestion.

Gastro-intestinal system

Indigestion with epigastric pain and enlarged tender liver will be present, when a patient is in congestive cardiac failure.

Central nervous system

Anxiety, restlessness, stupor, coma, focal neurological delicate may occur, when the patients is having fall in blood pressure and / or thromboembolic phenomenon.

Renal system

Oliguria may be present, if the patient is having fall in blood pressure.

Table 2: Complications of Acute Myocardial Infarction[73]

Complication Type	**Manifestations**
Ischemic	Angina, re-infarction, infarct extension
Mechanical	Heart failure, cardiogenic shock, mitral valve dysfunction, aneurysms, cardiac rupture
Arrhythmic	Atrial or ventricular arrhythmias, sinus or conduction blocks.
Embolic	Central nervous system or peripheral embolization
Inflammatory	Pericarditis

Investigations for Diagnosis of Acute Myocardial Infarction

Laboratory diagnosis

The laboratory tests of value in confirming the diagnosis of myocardial infarction are grouped into three categories

1. Non-Specific indices of tissue necrosis and inflammation.
2. The electrocardiogram.
3. Serum enzyme studies

1. Non-specific reaction to myocardial injury.

- Polymorphonuclearleucocytosis of 12,000 — 15,000/mm, which appears within a few hours after myocardial infarction and persists for 3-7 days is common. Higher white cell counts are associated with larger infarcts.
- Erythrocyte sedimentation rate (ESR) rise slowly, peaks during the first week, and remains elevated for several weeks. The height of elevations does not correlate with the severity of the infarct.
- C-Reactive protein(CRP) is seen in transmural infarction
- Blood coagulability: These are three phases of coagulability of blood immediately following the infarction.
  - A very constant period of hypocoagulability occupying the first 24-48 hours.
  - Period of spontaneous secondary hypocoagulability from 2-8 days.
  - Period of hypercoagulability, variable in degree from 15 days and lasting for several weeks or months. These figures are= important for the control of anticoagulant therapy.
- Hyperglycemia and impaired glucose tolerance. — occurs due to relative insulin resistance and decrease in the insulin secretion from the pancreas.

These are due to the fall of blood supply to the pancreas, increased catecholamine release, and stress.

Increased catecholamine release is proportionate to severity of myocardial infarction and the serum levels are in proportion to SGOT levels.

2. The Electrocardiogram [55,56]

The importance of E.C.G. is :

i. To diagnosis myocardial infarction.

ii. To diagnosis type of conduction defects.

Presence of abnormal Q waves, loss of R waves and typical ST elevation > 2mm and coving are suggestive of myocardial infarction. The site of infarction and their relation to various leads is show in table below (Table 3).

Table 3 Leads reflecting site of infarction

Sl no	Site of infarction	Leads reflecting infarct
1	Anterior	V_2-V_6
2	Anterior septal	V_1-V_4
3	Inferior	AVF LEAD L_2-L_3
4	Anterolateral	L_1.AVL. V_4-V_6
5	Apical	V_5-V_6
6	Right ventricular	RV_2, V_3,V_4,V_5

3. Cardiac Markers [75-77]

These are released by necrosis of myocyte into blood stream, which are normally concentrated within cardiac cells.

i. Creatinine phosphokinase:

It starts rising within 4-8 hrs peaks at 24 hrs and returns to normal by 48 hrs. The isoenzyme CKMB is more specific for myocardium. Changes suggestive of AMI are:

- Serial increase then decrease of plasma CKMB with a change >25% between any two values.
- CKMB > 10-13 u/liter or >5% of total CK activity
- Increase in CKMB activity >50% between any two samples separated by at least 4 hours.
- If only a single sample available CKMB elevation >2 fold

ii. Lactate dehydrogenase(LDH):

It starts rising after 24-48 hrs peaks at 3-6 days and returns to normal by 8-14 days. Isoenzyme LDH-1 is specific.

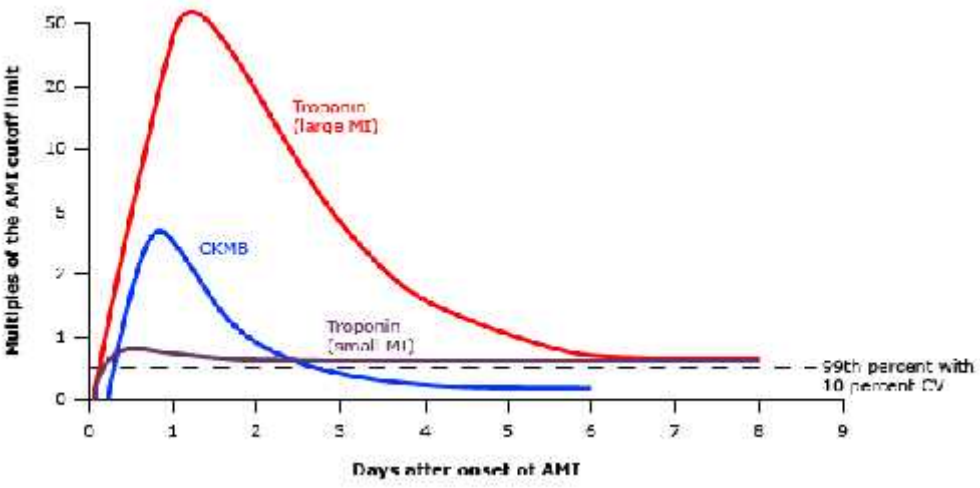
iii. Myoglobin:

It is the earliest to be released into serum (1-4 hrs). It lacks cardiac specificity and is rapidly excreted in urine and blood levels returns to normal within 24 hrs of onset of infarction.

iv. Cardiac specific troponin: cTnT and cTnI:

Levels of cTnI may remain elevated for 7 to 10 days and cTnT may remain elevated for up to 10 to 14 days after acute MI. Thus these are of value when patient presents late.

Figure 17



CARDIAC IMAGING [72,73]

Chest X-ray may be normal or may show signs of left ventricular failure or cardiomegaly. Two dimensional echocardiography is useful in detecting wall motion abnormalities, accessing left ventricular function diagnosis of right ventricular infarction, ventricular aneurysm or thrombus. Doppler echocardiography helps in detecting mitral regurgitation and ventricular septal defect. Radionuclide studies like Technetium 99m and Thallium 201 help in diagnosis and localizing infarction.

MATERIALS AND METHODS

Source of data:

Cases of acute myocardial infarction admitted in BLDE hospital and research center, Vijayapura were taken in the study.

Method of collection:

All the patients of acute myocardial infarction admitted in BLDE hospital during the study period January 2015 to June 2016 were evaluated by detailed history, clinical examination and the required investigations. The patients will be observed for conduction defects for 5 days from the day of their admission or until they stay in the hospital whichever is the earlier. Patients were selected taking in to consideration inclusion and exclusion criteria.

Inclusion criteria:

- Patients of acute myocardial infarction
- Diagnosis of acute myocardial infarction by WHO[70] criteria
- Diagnosis of conduction blocks based on ECG

Exclusion criteria

- Patients with known cases of conductions defects
- Patients on drugs which may cause conductions defects.
- Patients with cardiomyopathy
- Patients with congenital or rheumatic heart disease

Sample size:

At 95% confidence level and expected prevalence of coronary vascular disease as 6.4% , the minimum sample size is 145 at the rate of ± 4% margin of error.

Formulae used= $Z\alpha^2 * p (1-p) / d^2$

where n = sample size(145≈150)

Z = 1.96 at α= 5%

P = prevalence (6.4%) [78]

d = margin of error.

Study design:

A Prospective clinical study consisting of 150 patientsof acute myocardial infarction were undertaken to investigate the relationship of Conduction blocks with site, timing, complications and outcome.

Statistical tests:

The data collected was analyzed using proper tests.

- Student t test.
- Chi-square test for association

STATISTICAL ANALYSIS:

The data was compiled in Microsoft (MS) Excel work sheet and analyzed using SPSS (Statistical Package for Social Sciences) software version 16.0.

Results were subjected to following statistical analysis:

All characteristics were summarized descriptively. For continuous variables, the summary statistics of N, mean, standard deviation about the arithmetic mean (SD) were used. For categorical data, the number and percentage were used in the data summarized. Chi square test (χ^2) was employed to determine the significance of associations between variables for categorical data. Student t test was employed to determine the significance of associations between variables of continuous data. The associations with p value < 0.05 were considered to be statistically significant.

RESULTS

The present study includes a total of 150 patients of acute myocardial infarction who were admitted in Shri. B.M.Patil Medical College, Hospital and Research centre, Vijayapur during January 2015 to June 2016. Out of these 150 cases of acute myocardial infarction, 38 patients developed various types of conduction blocks.

Table-4 incidence of Conduction blocks in type of MI

MI site	Total	Conduction block present in	%
Anterior wall	78	12	31.65%
inferior wall	65	26	68.45%
Total	150	38	p value = 0.001

In the present study the total incidence of various types of conduction blocks were 25.3% (38 patients). Among 78 patients with anterior and anterior septal myocardial infarction, 12 (31.6%) patients had conduction blocks. Among 65 patients with inferior wall myocardial infarction, 26 (68.4%) patients had conduction blocks. So it was seen that the incidence of conduction blocks was higher in inferior wall myocardial infarction (68.4%) than in anterior wall myocardial infarction (31.6%).

Table-5 Sex incidence of conduction blocks

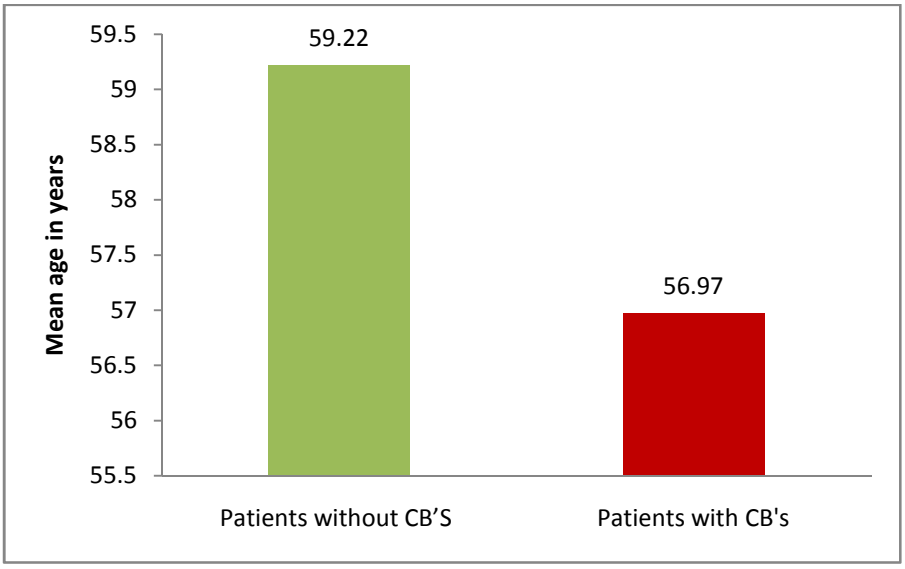
Myocardial infarction with conduction defects	Male	Female	Total
Anterior wall	10(26.3%)	2(5.2%)	12(31.5%)
inferior wall	18(47.3%)	8(21%)	26(68.4%)
Total	28	10	38

In the present study, among 38 patients of myocardial infarction with conduction blocks, 28 were male and 10 were female in the ratio of 2.8:1.

Table-6 Mean age of patients

	MEAN AGE IN YEARS
WITH OUT CB'S	59.22
WITH CB'S	56.97

Graph 1:Mean age of patients

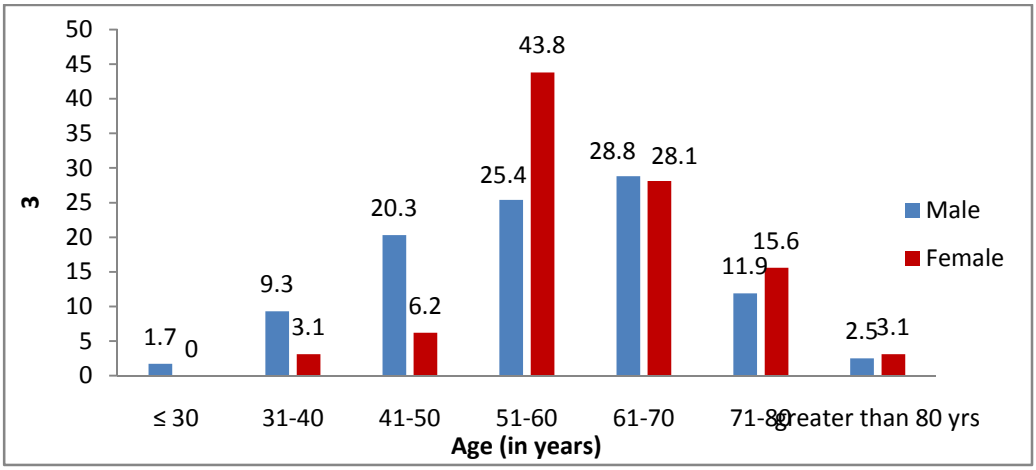


The above graph shows the mean age of patients. The mean age of patients with conduction blocks was 56.97years whereas in patients without blocks was 59.22 years.

Table-7 Distribution of study population according to age and sex

	Male		Female		Total	
Age in years	frequency	Percent	frequency	percent	frequency	percent
≤ 30	2	1.7	0	0	2	1.3
31-40	11	9.3	1	3.1	12	8
41-50	24	20.3	2	6.2	26	17.3
51-60	30	25.4	14	43.8	44	29.3
61-70	34	28.8	9	28.1	43	28.7
71-80	14	11.9	5	15.6	19	12.7
greater than 80 yrs	3	2.5	1	3.1	4	2.7
Total	118	100	32	100	150	100
Mean ± SD	57.58± 12.53		62.59±9.89		58.65±1.22	

Graph 2: Distribution of study population according to age and sex



Age ranged between 30-86 years,

The above graph shows the distribution of study population according to age and sex. In the present study 118 (78.66%) patients were male and 32(21.33%) were female patients. Of which the age of youngest patient was 30 years and the age of the oldest is 84 years. The present study shows male preponderance.

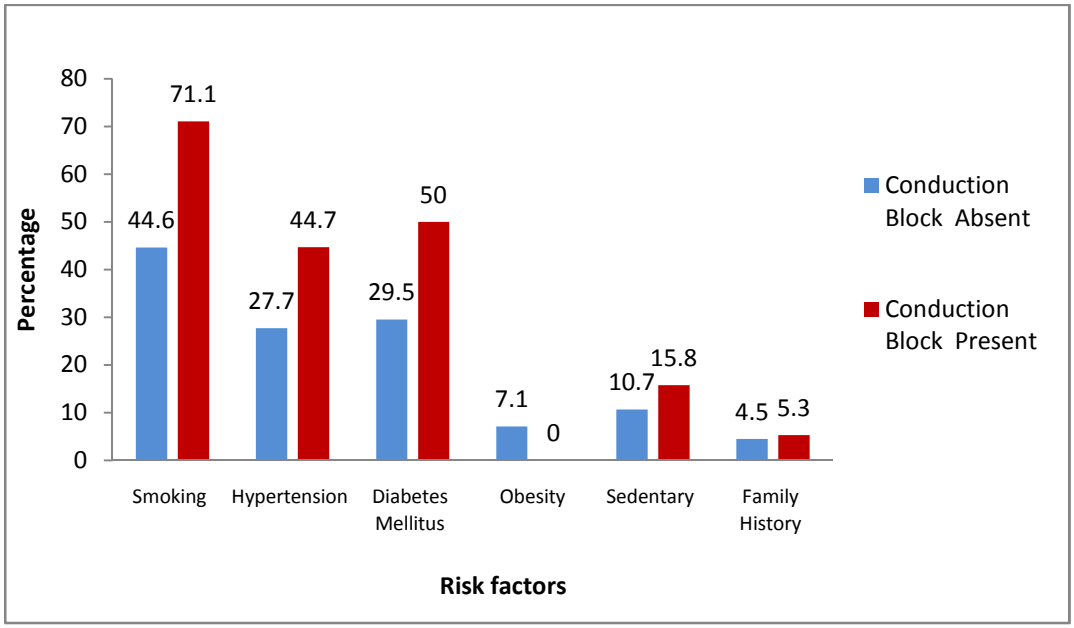
Majority of the male patients belonged to age group of 61-70 years (28.8%) and majority of female patients belonged to age group of 51 to 60 years (43.8%).

Table-8 Risk factors

Risk factors (Present)	Conduction Block		Total (n=150)	p value	Odd's ratio (OR)
	Absent (n=112)	Present (n=38)			
Smoking	50 (44.6%)	27 (71.1%)	77	0.005	3.04
Hypertension	31 (27.7%)	17 (44.7%)	48	0.05	2.12
Diabetes Mellitus	33 (29.5%)	19 (50.0%)	40	0.02	2.39
Obesity*	8 (7.1%)	-	8	-	-
Sedentary	12 (10.7)	6 (15.8%)	18	0.405	1.56
Family History	5 (4.5%)	2(5.3%)	7	0.840	1.19

***BMI > 30kg/m^2**

Graph 3: Risk factors



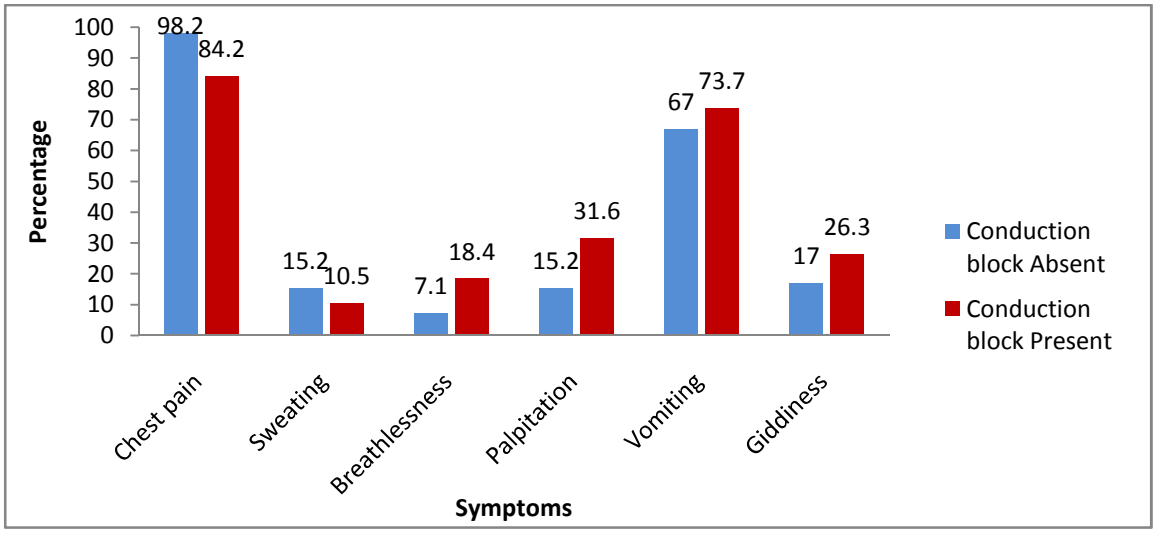
The above graph shows the distribution of risk factors among the patients who developed acute myocardial infarction. Present study noted Smoking history (OR = 3.04) was the most common risk factor. Diabetes and Hypertension were the other common risk factors noted (OR = 2.39 and 2.12 respectively).

Among the patients with acute myocardial infarction Smoking history was present in 50 (44.6%) patients without blocks and 27 (71.1%) patients with blocks which was significant($p<0.05$) . Similarly, Diabetes mellitus was present in 33 (29.5%) of patients without conduction blocks and 19 (50.0%) of patients with conduction blocks and hypertension was present among 31 (27.7%) of patients without conduction blocks and 17 (44.7%) of patients with conduction blocks were also found to be significant in the study.

Table-9 Distribution of patients according to Symptoms

Symptoms (Present)	Conduction Block		Total (n=150)	P value	OR
	Absent (n=112)	Present (n=38)			
Chest pain	110 (98.2%)	32 (84.2%)	142	0.001*	0.097
Sweating	17 (15.2%)	4 (10.5%)	21	0.475	0.657
Breathlessness	8 (7.1%)	7 (18.4%)	15	0.045*	2.935
Palpitation	17 (15.2%)	12 (31.6%)	29	0.027*	2.58
Vomiting	75 (67.0%)	28 (73.7%)	103	0.440	1.381
Giddiness	19 (17.0%)	10 (26.3%)	29	0.207	1.748

Graph 4: Distribution of patients according to Symptoms

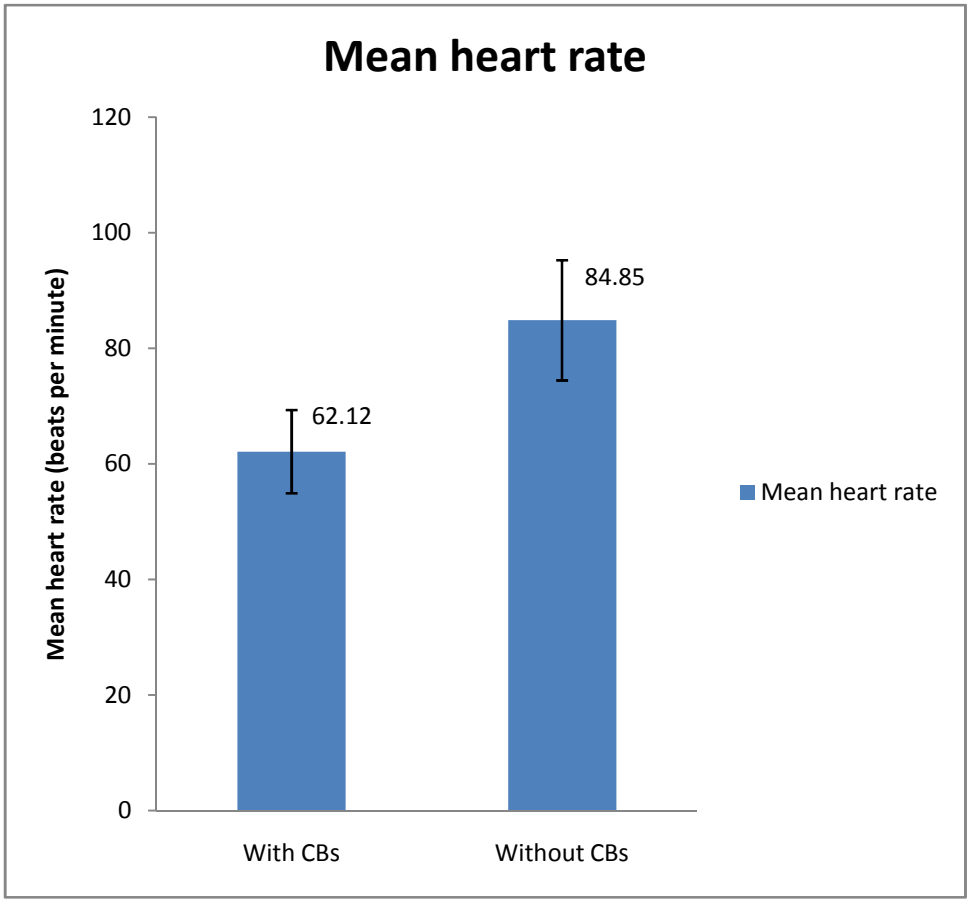


The above graph shows the distribution of patients according to the symptoms. In the present study Chest pain was the most common symptom in both the groups. It was present in 110 (98.2%) patients without blocks and 32 (84.2%) patients with blocks. Vomiting and giddiness are the other two common symptoms seen in the patients of both the groups.

Table 10: Comparison of Mean heart rate

	Mean heart rate	Standard deviation	
With CBs	62.12	7.2	$p < 0.05$
Without CBs	84.85	10.4	

Graph 5: Comparison of Mean heart rate

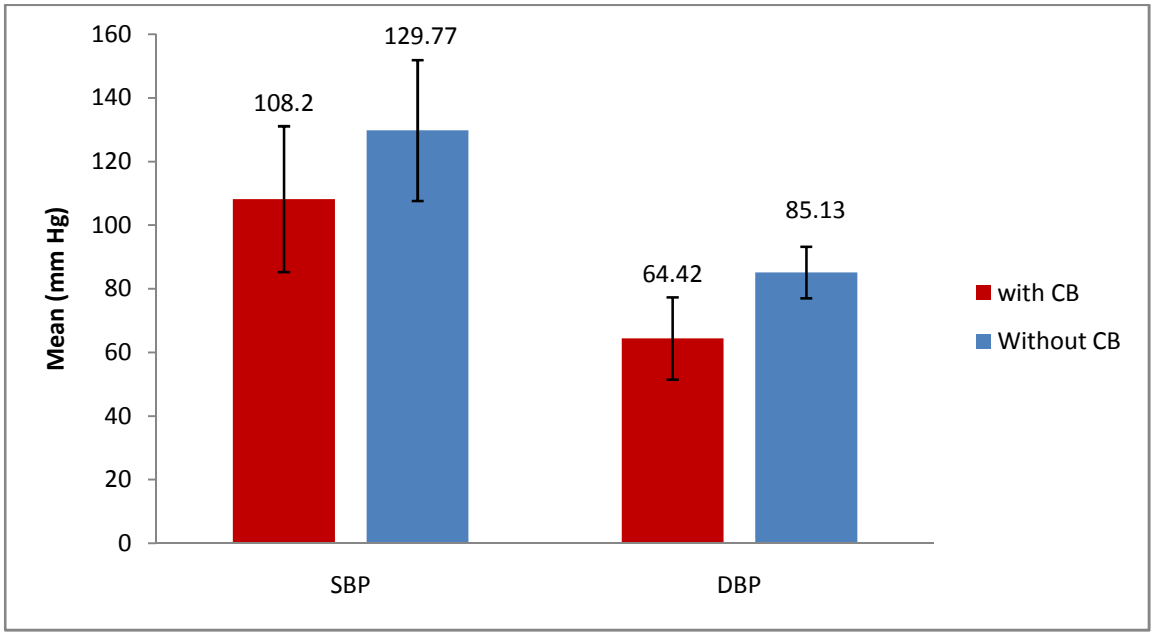


The mean heart rate was 62.12 ± 7.2 in patients with conduction blocks where as it was 84.85 ± 10.4 in patients without blocks. Mean heart rate was significantly different among patients with conduction blocks when compared to patients without conduction blocks.

Table-11 Comparison of mean systolic and diastolic blood pressures among patients with and without blocks

	WITH CB	WITH OUT CB	p value
SYS BP (mm Hg)	108.20 ± 22.92	129.77 ± 22.12	0.044
DIA BP (mm Hg)	64.42 ± 12.95	85.13 ± 8.11	0.001

Graph 6: Comparison of mean systolic and diastolic blood pressures among patients with and without blocks



The above graph shows the comparison of mean systolic and diastolic blood pressures in patients with and without conduction blocks. In the present study the mean systolic and diastolic blood pressures in patients with blocks (108.20 ± 22.92 and 64.42 ± 12.95 respectively) were significantly lesser than the mean systolic and diastolic blood pressures of patients without blocks (129.77 ± 22.12 and 85.13 ± 8.11 respectively).

Table-12 Association of Site with Conduction Block

CONDUCTION BLOCKS								
	Atriventricular blocks				Intraventricular blocks			
MI site	Type 1 block	Mobitz type 1	Mobitz type 2	CHB	RBBB	LBBB	Fascicular Blocks	Total
Anterior wall	1 (11.1%)	-	-	1 (6.2%)	6 (66.7%)	4 (100.0%)	-	12
inferior wall	8 (88.9%)	-	-	15 (93.8%)	3 (33.3%)	0 (0)	-	26
Total	9 (100.0%)	-	-	16 (100%)	9 (100.0%)	4 (100.0%)	-	38

MI site	Conduction block present (Total = 38)	Conduction block absent (Total = 112)
Anterior wall	12 (31.6%)	66 (58.9%)
Inferior wall	26 (68.4%)	39 (34.8%)
		p value = 0.001

Atrioventricular blocks:

In the present study 25 patients had various types of atrioventricular blocks (i.e.16.66%).

Table-13 Atrioventricular blocks:

		Conduction Block		
Risk factors (Present)	Type 1 degree block	Second degree block		Third degree block
		Mobitz type 1 (n=112)	Mobitz type 2 (n=38)	
Anterior wall	1(0.6%)	-	-	1(0.66%)
Inferior wall	8(5.33%)	-	-	15 (10%)
Total	9 (6%)	-	-	16(10.67%)

The present study shows that AV blocks are common in inferior wall myocardial infarction.

First degree AV block

The above table shows that out of 25 patients with various types of AV blocks, 9(6%) patients had first degree AV block and the incidence was higher in inferior wall myocardial infarction than the anterior wall myocardial infarction.

Second degree AV block

In the present study there were no patients of myocardial infarction with second degree AV block.

Third degree AV block (CHB)

In the present study 16(10.6%) patients had third degree AV block and the incidence was more common in inferior wall myocardial infarction than the anterior wall infarction.

Intraventricular conduction defects:

In the present study 13(8.6%) patients had various type of intraventricualr blocks.

Table-14 Intraventricular conduction defects:

MI	RBBB	LBBB	Fascicular blocks
Anterior wall	6 (66.7%)	4 (100.0%)	-
inferior wall	3 (33.3%)	0	-
Total	9	4	-

The present study shows intraventricular conduction defects were more common in anterior wall myocardial infarction than inferior wall myocardial infarction.

In the present study 9(6%) patients had right bundle branch block among whom 6 patients had anterior wall myocardial infarction and 3 patients had inferior wall infarction. Thus right bundle branch block was more common in anterior wall myocardial infarction than inferior wall myocardial infarction.

In the present study 4(2.66%) patients had left bundle branch block, and all the 4 had anterior wall myocardial infarction. Thus left bundle branch block is more common in anterior wall myocardial infarction than inferior wall myocardial infarction.

In the present study there were no patients with myocardial infarction who developed fascicular blocks.

Complications:

Table-15 Association type of conduction block with complications

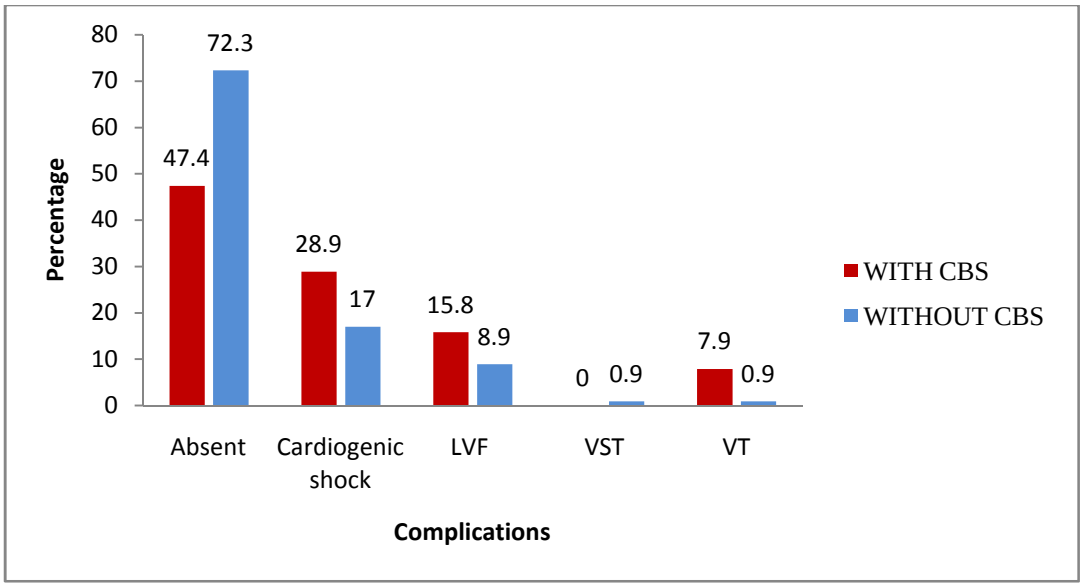
	conduction blocks types				
Complications	type 1 block	RBBB	LBBB	CHB	Total
CS	2 (22.2%)	5 (55.6%)	0 (0)	4 (25.0%)	30 (20.0%)
LVF	0 (0)	2 (22.2%)	2 (50.0%)	2 (12.5%)	16 (10.7%)
SVT	0 (0)	0 (0)	0 (0)	0 (0)	1 (0.7%)
VT	0 (0)	0 (0)	0 (0)	3 (18.8%)	4 (2.7%)
Absent	7(77.8%)	2(22.2%)	2(50.0%)	7(43.8%)	99(66.0%)
Total	9 (100.0)	9 (100.0)	4 (100.0)	16 (100.0)	150 (100.0)

The above table shows the association of type of conduction blocks and the complication they developed. Out of 9 patients with type 1 AV block 2 patient developed cardiogenic shock and 7 patients improved without any complications. Among 9 patients who developed right bundle branch block 5 patients developed cardiogenic shock, 2 patients developed left ventricular failure and 2 patients improved without any complications. Among 4 patients of left bundle branch block 2 patients developed left ventricular failure and 2 patients improved without any complications. Among 16 patients with complete heart block 4 patients developed cardiogenic shock, 3 patients developed ventricular tachycardia, 2 patients developed left ventricular failure and 7 were improved without any complications.

Table-16 Association of conduction block with complications

Type of Complications	WITH CBS (Total = 38)
VT	3(7.9%)
Cardiogenic shock	11 (28.9%)
Left Ventricualr Failure	6 (15.8%)
SVT	0 (0)
absent	18(47.4)

Graph 7: Association of conduction block with complications



The above graph showed the association of type of conduction block with complications they developed during the hospital stay. In the present study the occurrence of cardiogenic shock was more common in patients with conduction blocks than in the patients without conduction blocks.

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Table-17 Death in patients associated with type of conduction block

		Conduction block				
	type 1 block (n =9)	RBBB (n = 9)	LBBB (n = 4)	CHB (n = 16)	Absent (n=112)	Total =150
Death	2 (22.2%)	5 (55.6%)	1 (25.0%)	3 (18.8%)	16 (14.3%)	27

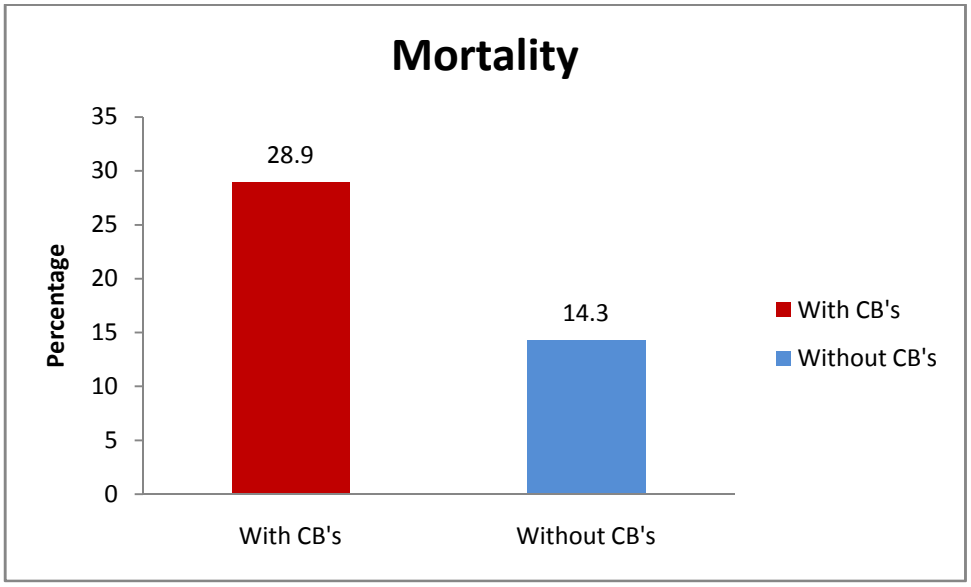
The above table shows death among the patients who developed conduction blocks. 2 out of 9 patients who developed type 1 AV block expired. (These two patients had inferior wall MI). 5 (4 with anterior wall MI and 1 with inferior wall MI) out of 9 patients with right bundle branch block expired and one (with anterior wall MI) out of 4 patients with left bundle branch block expired. 3 (2 had inferior wall MI and one had anterior wall MI) out of 16 with complete heart block expired.

Mortality was high among the patients who developed right bundle branch block (55.6%)

Table-18 Death in patients with conduction block

	WITH CBS (Total = 38)	WITHOUT CBS (Total = 112)
MORTALITY	11 (28.9%)	16 (14.3%)

Graph 8: Death in patients with conduction block



The above graph show death among the patients who developed conduction blocks. In the present study there was 22.2 % among type 1, 55.6 % among RBBB, 25 % among LBBB and 18.8 % among CHB. The mortality was higher in patients with blocks (28.9%) as compared to patients without blocks (14.3%)

DISCUSSION

The present study consisted of 150 patients of acute myocardial infarction admitted in the ICCU during January 2015 to June 2016. All patients fulfilled the clinical and investigational criteria of acute myocardial infarction.

Incidence

In the present study, out of 150 patients of acute myocardial infarction, 38 developed various types of conduction defects, which make an incidence of 25.33%. Wooks et al (1990)[79]reported 27.53% incidence of conduction blocks in acute myocardial infarction. Archbold RA, Sayer JW et al (1998)[21] reported that the conduction blocks complicated acute myocardial infarction in 16%.

Site of infarction

In the present study, out of 78 patients with anterior wall & anterior septal myocardial infarction, 12 (19%) patients had conduction blocks, and out of 65 patients with inferior wall myocardial infarction, 26 (31.6%) patients had conduction defects. Thus conduction blocks were more frequently in inferior wall myocardial infarction.

Braunwald (2014)[73] , Wooks et al (1990)[79] have reported similarly

Sex incidence

In the present study group 78.66% were males and 21.33% were females, showing male preponderance and ratio of 2.8:1

Our results were similar to the study done by Joseph tharakan, K.S.Varadhcharay et al and Newby KH et al. Joseph tharakan, K.S.Varadhcharay et al (1986)[80] reported 3:1 ratio of male to female in patients of acute myocardial infarction with conduction block. Newby KH et al [39], in their study reported that the patients who developed conduction blocks were 78.66 % male and 28.90% female.

AGE

In the present study the mean age of patients with conduction blocks was 59.22 and that of patients without conduction blocks was 56.297. On comparison with other studies done by Abidov et al[30] and Newby KH et al [39] where the mean age was 61.7 and 63.6 respectively.

Table-19 Comparison of mean age of patients in present study and other studies

Studies	Mean age in years	
	With CBs	Without CBs
Present study	56.97	59.22
Abidov et al [30]	61.7	58.3
Newby KH et al [39]	63.6	58.1

Patients in our study are younger than those in other studies. It may be because CAD appears earlier in Indians as compared to developed world.

Risk Factors

In the present study the most common risk factor noted was smoking in both the groups, followed by hypertension and diabetes mellitus. On comparison with studies done by Abidov et al and Newby KH et al, Our values of smoking was higher than those of study by Abidov et al and lesser than those of Newby et al. Our values of hypertension and diabetes mellitus are similar to those of Newby et al.

Table 20 Comparison of risk factors in patients of present study and other studies:

Studies		Smoking (%)	HTN (%)	DM (%)
Present study	With CBs	71.1	44.7	50
	Without CBs	44.6	27.7	29.5
Abidov et al [30]	With CBs	43	37	24
	Without CBs	47	33	20
Newby KH et al [39]	With CBs	71.9	46	19.9
	Without CBs	74.4	41	15.8

Conduction defects

In the present study, 38(25.33%) patients developed various type of conduction defects among whom 25(16.6%) patients developed atrioventricular blocks and 13 (8.6%) patients developed intraventricular blocks.

Atrioventricular blocks

In the present study, atrioventricular blocks developed in 25 (16.6%) patients.

Meine TJ et al[12] reported 6.9% incidence of atrioventricular blocks and Wooks et al (1990)[79] reported 11.3% incidence of atrioventricular block in acute myocardial infarction.

In the present study, atrioventricular blocks were more commonly seen in inferior wall than anterior wall myocardial infarction.

Meine TJ et al[12] reported overall incidence of atrioventricular blocks in higher in inferior wall myocardial infarction (9.8%) than in anterior wall myocardial infarction (3.2%). Auffret V, Loirat A, Leurent G et al[14] showed, atrioventricular block were higher in inferior wall myocardial infarction (5.9%) than anterior wall myocardial infarction (1.5%)

First degree AV block

In the present study, 1.3% of patients with acute myocardial infarction had first degree atrioventricular block.

Thomas David, Jaison T.M, Mathew M et al (1988)[81] reported 6.18% incidence of first degree AV block in acute myocardial infarction. Chin Hock Lim, Charles C.S.Toh et al (1971)[81] reported 1.5 % of incidence of first degree atrioventricular blocks with acute myocardial infarction.

Second degree AV block

In the present study, there were no cases of myocardial infarction with Type 2 AV block (Mobitz type 1 AV block and Mobitz type 2 AV block.)

Complete heart block

The present study shows 10.66% incidence of complete AV block in patients with acute myocardial infarction.

Berger PB, Ruocco NA, Jr. Ryan T.J et al (1992)[28] reported 12% incidence of complete heart block with acute myocardial infarction. Goldberg RJ, Zevallos J et al (1992)[27] reported 5 to 8% incidence of complete heart block with acute myocardial infarction. Archbold RA, Sayer JW, Ray S, Wilkinson P et al (1998)[21] reported 5.3% incidence of complete AV block with acute myocardial infarction.

The present study shows third degree AV block to be more common in inferior wall myocardial infarction than anterior wall myocardial infarction.

Hindman MC, Wagner GS et al (1978)[26] observed that complete heart block is twice as common with inferior wall myocardial infarction than with anterior wall myocardial infarction.

Intraventricular conduction defects

In the present study, 8.66% patients developed various type of intraventricular blocks.

Hindman MC, Wagner et al (1978)[26] observed that intraventricular conduction delays including bundle branch block and fascicular block, occurred in about 10 — 20% of patients with myocardial infarction. Godman MJ, Lasscrs BW et al (1970)[83] reported 8 to 15% incidence of intraventricular block with myocardial infarction.

In the present study intraventricular conduction blocks to be more common in anterior and anterior septal myocardial infarction than the inferior wall myocardial infarction. KC Garg, NS Negi, PK Pathak et al(1988)[84] reported intraventricular conduction defects to be predominant in anterior wall myocardial infarction.

Right bundle branch block

In the present study, 6% incidence of right bundle branch block with acute myocardial infarction was observed.

Archbold RA, Sayer JW, Ray S et al (1998)[21] reported 2.4% incidence of right bundle branch block with myocardial infarction. In the present study right bundle branch block was more common in anterior and anterior septal myocardial infarction. Hindman MC, Wagner et al (1978)[26] observed right bundle branch block to be more frequently associated with anterior wall myocardial infarction.

Left bundle branch block

In the present study, 2.66% incidence of left bundle branch block with acute myocardial infarction was observed.

Godman et al (1970)[83] reported 2% incidence of left bundle branch block in acute myocardial infarction. Wooks et al (1990)[79] reported 3% incidence of left bundle branch block in acute myocardial infarction.

In the present study left bundle branch block was more common in anterior and anteroseptal myocardial infarction. Paolo Rizzon, Di Biasc, Baissusi et al(1974)[85] reported left bundle branch block to be more common in anterior wall myocardial infarction than inferior wall myocardial infarction.

Fascicular blocks

In the present study there were no cases of myocardial infarction with fascicular blocks

Mortality

In our study, patients with right bundle branch block (RBBB) mortality rate was 55.6% as compared with total mortality rate of 14.3% in patients without blocks. On comparing with studies done by Go As et al[10] and Moreno AM et al[40], where the mortality was 23% and 25.9% respectively, present study showed higher mortality with RBBB .

Table-21 Comparison of mortality in patients with RBBB in present study and others studies

Studies	Mortality rate (%)	
	With RBBB	Without Block
Present study	55.6	14.3
GO AS et al[10]	23	13.1
Moreno AM et al [40]	25.9	9.9

Table 22 Comparison of mortality in patients with III block in present study and others studies

Studies	Mortality rate (%)	
	With III Block	Without Block
Present study	18.8	14.3
Bates ER et al [42]	20	4
Goldberg et al[27]	41.9	14
Beher S et al[43]	37	11

In our study the mortality rate in patients in patients with complete AV block was 18.8%, which is slightly higher than that of patients without blocks that is 14.3%. On comparison with studies done by Bates ER et al, Goldberg et al, and Beher S et al, where the mortality rate was 20%, 41.9% and 37% respectively.

All other studies had much higher values as compared to our study. As the patients with III degree block were referred to other hospitals for need of permanent pace maker our values might have been low.

Comparison of mortality in patients with blocks and without blocks

In present study the mortality rate of patients with conduction blocks was 28.9% compared to 14.3% in patients without conduction blocks.

In the study done by Mujamdar A.A et al [41] showed mortality rate of 25% in patients with conduction blocks and 3.6% in patients without conduction blocks. Similar results were seen in study done by Sgarbossa EB, Pinski SL, Topol EJ et al [32] showed in hospital mortality is high in patients with conduction blocks (18%) versus patient without conduction blocks (11%)

Cardiogenic shock

In the present study cardiogenic shock was seen in 33.3% patients with complete heart block which was higher when compared to 14.3% in patients without block. The study done by Beher S et al showed, that frequency of cardiogenic shock was high (23%) in patients with complete heart block. Similar results were reported in the study done by Goldberg RJ et al[27] where 23.4% of patients with complete heart block developed cardiogenic shock.

Table-23 Comparison of frequency of cardiogenic shock in patients with III block in present study and other studies

Studies	With III Block (%)	Without Block (%)
Present study	25	17
Beher S et al [43]	23	4
Goldberger RJ et al [27]	23.4	6.5

SUMMARY

1. In this study 150 patients of Acute Myocardial Infarction were included, 118 0f them were males and 32 were females.
2. Among the 150 patients 38 had conduction blocks.
3. Majority of the male patients belonged to age group of 61-70 years (28.8%) and majority of female patients belonged to age group of 51 to 60 years (43.8%)
4. The mean age of patients with conduction blocks is 54.95 whereas in patients without blocks is 55.75.
5. Smoking, diabetes and hypertension were the common risk factors noted.
6. Chest pain was the most common symptom in both the groups. Vomiting and sweating were the other two common symptoms seen in the patients of both the groups
7. The mean heart rate was 62 in patients with conduction blocks where as it is 84 in patients without blocks.
8. The mean systolic and diastolic blood pressure in patients with blocks (108.26 and 64.42 respectively) was lesser than that of patients without blocks (129.79 and 85.13 respectively).
9. The conduction blocks were significantly more common among patients with inferior wall MI than the anterior wall MI (P=0.001). Bundle branch blocks were more common in anterior wall MI than inferior wall MI whereas atrioventricular blocks were common in inferior wall MI.
10. Bradycardia, Hypotension and raised JVP were more common in patients with conduction blocks as compared to patients without conduction blocks.

11. The most common complication was cardiogenic shock followed by left ventricular failure in both the groups. Other complications seen were SVT and VT. These complications were seen more commonly in patients with blocks.
12. Mortality was higher in patients with blocks (28.9%) as compared to patients without blocks (14.3%).

CONCLUSION

Bradyarrhythmias and conduction disturbances are well-recognized complications of acute myocardial infarction (MI). They are caused by either autonomic imbalance or ischemia/necrosis of the conduction system.

Atrioventricular (AV) block with inferior MI generally results from ischemic and autonomic effects on the AV node. It is associated with a narrow QRS complex and develops in a progressive fashion from first to second to third degree block. It often results in an asymptomatic bradycardia (40 to 60 beats/min) and is usually transient, resolving within five to seven days.

AV block with anterior MI generally occurs abruptly in the first 24 hours. It can develop without warning or may be preceded by the development of right bundle branch block (RBBB) with either a left anterior or posterior fascicular block pattern (bifascicular or trifascicular block). The escape rhythm is wide and unstable and the event is associated with a high mortality from both arrhythmias and pump failure. In-hospital and 30-day mortality are higher than seen with an inferior wall infarction.

Bradyarrhythmias that occur early in the setting of an inferior MI (within the first 24 hours) may respond to atropine, while those occurring later are often atropine resistant. Treatment is not indicated in asymptomatic patients, but is useful if symptoms are present, such as dizziness, syncope, or confusion from reduced cardiac output.

We consider temporary transvenous pacing in the following circumstances

- Complete (third-degree) AV block.
- Alternating right and left bundle branch block (LBBB).
- RBBB with alternating left anterior or posterior fascicular block.

- New or age indeterminate bifascicular block (RBBB with left anterior or posterior fascicular block or LBBB) with PR prolongation.
- Asystole.
- Symptomatic bradycardia of any etiology, including sinus bradycardia and Mobitz type I second degree AV block, if adverse hemodynamic effects are present and the bradyarrhythmia is not responsive to atropine.
- Mobitz type II second-degree AV block.
- Bradycardia-induced tachyarrhythmias, such as torsades de pointes.

To conclude Conduction blocks are associated with higher in hospital mortality and morbidity in the form of other cardiovascular events during hospital stay.

- Conduction blocks are important predictors of poor outcome in patients with Acute Myocardial Infarction.

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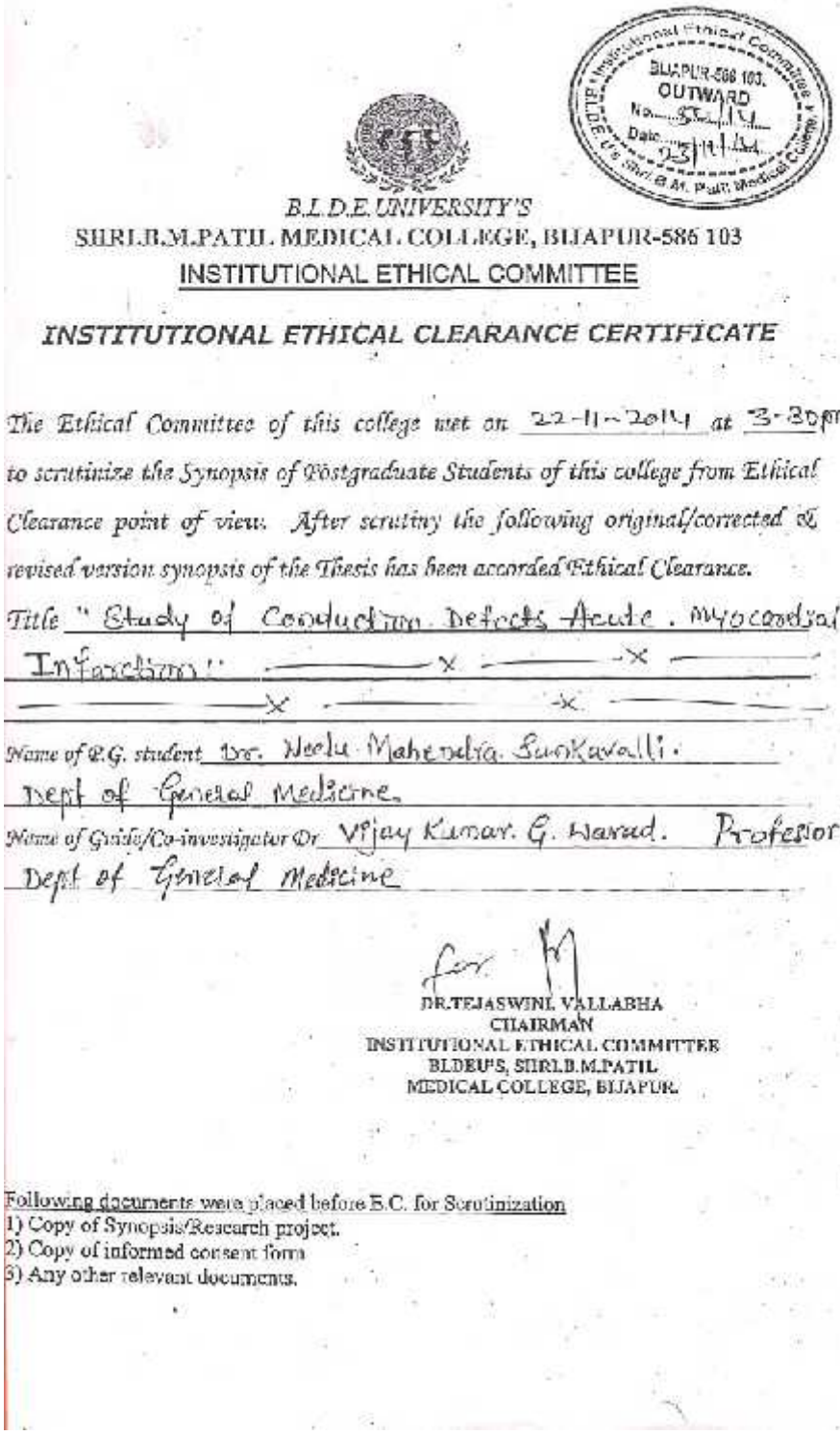
ANNEXURE-I

ETHICAL CLEARANCE CERTIFICATE



B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL. MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 22-11-2014 at 3-30pm to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.

Title "Study of Conduction Defects Acute. Myocardial Infarction"

Name of P.G. student Dr. Neelu Mahendra Bookawalli.
Dept of General Medicine.

Name of Guide/Co-investigator Dr Vijay Kumar. G. Warad. Professor.
Dept of General Medicine

for

DR.TEJASWINI VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevent documents.

ANNEXURE-II

INFORMED CONSENT FOR PARTICIPATION IN

DISSERTATION/ RESEARCH

I the undersigned----------S/O.D/O.W/O----------aged ------years ordinarily resident of----------do here by state/declare that **Dr Neelu Mahendra Sunkavalli** of **Shri B.M.Patil Medical College and Hospital** has examined me thoroughly on ----------at----------(place) and has explained to me in my own language ----------that I am suffering from ----------disease (condition) and this disease/condition mimic following diseases ----------Further **Dr Neelu Mahendra Sunkavalli** informed me that she is conducting dissertation/research titled **"STUDY OF CONDUCTION DEFECTS IN ACUTE MYOCARDIAL INFARCTION"** Under the guidance of **Dr Vijaykumar.G.Warad** requesting my participation in the study. Further Doctor has informed me that my participation in this study help in evaluation of results of the study which is useful reference for treatment of other similar cases in near future, and also I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made/ photographs/video graphs taken upon me by the investigator will be kept secret and not accessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary based on information given to me, I can ask any clarification during the course of

treatment/study related to Diagnosis, Procedure of treatment, result of treatment or prognosis. At the same time I have been informed that I can withdraw from my participation in this study at any time if I want or investigator can terminate me from the study at any time from the study but not the procedure of treatment & follow up unless I request to discharge.

After understanding the nature of dissertation or research, Diagnosis made, mode of treatment I the under signed Shri/Smt----------under my full conscious state of mind I agree to participate in the said research/Dissertation .

Signature of patient:

Signature of Doctor:

Witness 1

Witness 2

ANNEXURE-III

BLDE'S SHRI B.M.PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR

CONDUCTION DEFECTS IN ACUTE MYOCARDIAL INFARCTION SCHEME OF CASE TAKING

Name: CASE NO:

Age: IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Residence:

Presenting complaints with duration:

History of presenting complaints:

Past History:

Personal History:

Diet Appetite Sleep Bladder and bowel habits:

Smoking/Tobacco chewing/Snuff Inhalation

Duration

Number of cigarettes/beedis pack year smoked

Amount of tobacco chewed/snuff inhaled

Alcohol

Duration

Quantity/Frequency

Type

Sexual history

History of multiple sexual partners.

Family History:

Treatment History:

GENERAL PHYSICAL EXAMINATION:-

Pallor: Present/absent

Icterus: present/absent

Clubbing: present/absent

Generalized lymphadenopathy: present/absent

Built:

Nourishment:

Vitals

PR:

BP:

RR:

Temp:

Hair;

Eyes:

Nose;

Ears:

Oral cavity;

Neck:

Upper limbs:

Chest:

Abdomen:

Genetilia;

Lower limbs:

Skin:

SYSTEMIC EXAMINATION.

Cardiovascular system

Respiratory System

Per abdomen

.

Central Nervous System

INVESTIGATIONS

- **HAEMATOLOGY –**
    - HAEMOGLOBIN

- TOTAL WBC COUNT
- PLATELET COUNT
- ESR

- **URINE EXAMINATION-**
  - URINE ALBUMIN
  - URINE SUGAR
  - URINE MICROSCOPY
- **BIOCHEMISTRY–**
  - CREATINE KINASE
  - RANDOM BLOOD SUGAR
  - TROPONIN-T
  - LIPID PROFILE
  - SERUM ELECTROLYTES

- **ELECTRO-CARDIO-GRAPHY**

- **2D-ECHOCARDIOGRPHY**

  LVEF

- **CHEST X RAY**

FINAL DIAGNOSIS:

- Type of conduction block
- Site of infarction

KEY TO MASTER CHART

VT	-	Ventricular tachycardia
CS	–	Cardiogenic shock
L	-	Living
D	–	Death
IW	-	Inferior wall
AW	-	Anterior wall
GL	-	Global
PSM	–	Pan systolic murmur
S3	-	Third heart sound
S4	-	Fourth Heart Sound
SBP	-	Systolic blood pressure
DBP	-	Diastolic blood pressure
Y	-	Yes
N	-	No
P	-	Present
A	-	Absent
C	–	crepitations
N	-	Normal
CHB	-	Complete heart block
I	-	Type 1 AV block
RBBB	-	Right bundle branch block
LBBB	-	Left bundle branch block
NA	-	Not available

Sl. No	Name of patient	IP Number	Age in years	sex	jvp	Heart sounds	Additional sounds	Murmurs	Tachycardia	Bradycardia	SBP	DBP	hypotension	Hypertension	Pulse	Bradycardia	Chest pain	sweating	breathlessness	palpitation	Vomiting	giddiness	Hypertension	Rub	Resp system	CK	RBS	Total cholesterol	LDL	HDL	TG	Thrombolysis	Smoking	HTN	DM	Obesity	Sedentary life	Family h/o	Coonduction block Present/Absent	SITE	Type	Timing	Com	Outcome	Causes of death
1	chanasab mulk	28502	54	Male	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	38	183	173	43	29	80	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	RBBB	NA	A	L	0
2	ashik laxman	28359	70	Male	Normal	N	A	PSM	Absent	Absent	110	**84**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Present	Present	Present	**0**	N	C	109	151	135	40	60	83	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	CS	D	PF
3	putalabai	26051	55	Female	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Present	Absent	**1**	N	N	51	112	NA	NA	NA	NA	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	D	VT
4	kallapan	28564	72	Male	Raised	N	S3	A	Absent	Present	142	**82**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Present	Present	**1**	N	N	68	112	126	55	89	53	Y	Absent	Present	Present	Absent	Present	Absent	Present	IW	CHB	T	CS	L	0
5	vitabhai	28547	50	Female	Normal	N	A	A	Absent	Absent	130	**90**	**Absent**	**Present**	78	Absent	Present	Absent	Present	Absent	Present	Absent	**1**	N	N	40	-	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	IW	RBBB	PT	CS	D	PF
6	panehappa biradar	28476	86	Male	Raised	N	A	A	Present	Absent	120	**88**	**Absent**	**Absent**	110	Absent	Present	Absent	Absent	Present	Present	Absent	**0**	N	N	33	-	210	46	91	95	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
7	sukudev ram	27539	58	Male	Normal	N	A	A	Absent	Absent	122	**84**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	N	52	89	132	28	215	61	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	PF
8	Tibunnisa	26850	60	Female	Raised	N	A	A	Absent	Absent	124	**82**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	C	54	91	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	VF
9	siddanagouda	8761	60	Female	Raised	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Absent	Present	**1**	N	N	18	229	157	26	74	116	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	GL	A	NA	A	L	0
10	manohar laxman	26929	64	Male	Normal	N	A	A	Absent	Absent	120	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	10	203	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	I	T	A	L	0
11	layappa	9361	68	Male	Normal	N	A	A	Absent	Absent	134	**82**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	50	68	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
12	mehabub	9078	65	Male	Normal	N	A	A	Absent	Absent	110	**84**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Absent	Absent	**0**	N	N	52	118	151	21	650	101	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
13	somalingappa	27532	38	Male	Raised	N	S3	A	Present	Absent	60	**50**	**Present**	**Absent**	126	Absent	Present	Absent	Absent	Absent	Absent	Present	**0**	N	C	58	156	148	35	140	85	Y	Absent	Absent	Present	Absent	Absent	Absent	Present	IW	I	T	CS	L	0
14	nagappa	15210	71	Male	Normal	N	S4	A	Absent	Absent	162	**92**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Absent	Present	**1**	N	N	80	246	166	26	173	105	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
15	vijay kumar	14404	60	Male	Normal	N	A	A	Absent	Absent	110	**86**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	44	128	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	A	L	0
16	lakkamma	11028	60	Female	Normal	N	A	A	Absent	Absent	134	**84**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	96	132	215	39	100	156	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
17	gouramma	11551	68	Female	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	76	Absent	Present	Absent	Absent	Absent	Absent	Absent	**1**	N	N	100	207	208	31	198	111	Y	Present	Present	Absent	Present	Present	Absent	Absent	AW	A	NA	A	L	0
18	siddabai	40187	65	Female	Normal	N	A	A	Present	Absent	142	**94**	**Absent**	**Present**	108	Absent	Present	Absent	Absent	Absent	Present	Absent	**1**	N	C	120	130	290	42	311	185	Y	Present	Present	Absent	Present	Absent	Absent	Absent	AW	A	NA	LVF	L	0
19	ashok basavaraj	39437	63	Male	Normal	N	A	A	Absent	Present	90	**82**	**Absent**	**Absent**	52	Present	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	49	154	226	45	125	156	Y	Present	Absent	Present	Present	Absent	Absent	Absent	IW	A	NA	A	L	0
20	apanna	820	52	Male	Normal	N	A	A	Absent	Absent	126	**84**	**Absent**	**Absent**	62	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	27	188	-	-	-	-	Y	Absent	Absent	Present	Present	Absent	Absent	Absent	AW	A	NA	A	L	0
21	ningamma	2219	85	Female	Normal	N	A	A	Absent	Absent	128	**86**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	120	127	202	34	169	134	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
22	chambasappa	18225	58	Male	Raised	N	A	A	Absent	Absent	146	**84**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Absent	Absent	**1**	N	C	72	360	206	45	272	106	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
23	siddanagouda	8761	65	Male	Raised	N	S3	PSM	Present	Absent	130	**84**	**Absent**	**Absent**	102	Absent	Present	Absent	Present	Absent	Present	Present	**0**	N	C	87	267	187	40	287	98	Y	Present	Absent	Present	Absent	Present	Present	Absent	AW	A	NA	CS	L	0
24	amasidda ramanna	21939	60	Male	Normal	N	A	A	Absent	Present	100	**86**	**Absent**	**Absent**	58	Present	Present	Present	Absent	Absent	Present	Present	**0**	N	N	180	90	165	25	110	118	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
25	sidramappa	8279	66	Male	Normal	N	A	A	Absent	Absent	190	**120**	**Absent**	**Present**	84	Absent	Present	Absent	Absent	Absent	Absent	Present	**1**	N	R	109	345	245	29	343	147	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
26	mehabub	9078	65	Male	Normal	N	A	A	Absent	Absent	110	**82**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	N	55	109	181	42	135	112	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
27	sharanappa	26111	65	Male	Raised	N	S3	A	Absent	Absent	88	**58**	**Present**	**Absent**	92	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	C	54	110	-	-	-	-	N	Absent	Absent	Present	Absent	Absent	Absent	Absent	IW	A	NA	SVT	L	0
28	padmavathi	11914	50	Female	Normal	N	A	A	Absent	Present	90	**60**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Present	Absent	**1**	N	N	100	130	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Present	IW	CHB	T	A	L	0
29	nanasaheb	11968	67	Male	Normal	N	A	A	Absent	Absent	110	**80**	**Absent**	**Absent**	76	Absent	Present	Absent	Present	Absent	Present	Absent	**0**	N	N	>200	120	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	I	T	A	L	0
30	laxmibai	24389	68	Female	Raised	N	A	A	Absent	Present	144	**88**	**Absent**	**Present**	52	Present	Present	Absent	Absent	Present	Present	Absent	**1**	N	N	54	132	-	-	-	-	Y	Absent	Present	Absent	Absent	Absent	Present	Present	IW	CHB	T	A	L	0
31	hanamanth	9888	35	Male	Normal	N	S3	A	Absent	Absent	142	**86**	**Absent**	**Present**	60	Absent	Present	Absent	Absent	Present	Absent	Present	**1**	N	C	200	204	149	40	245	60	Y	Present	Present	Present	Absent	Absent	Absent	Present	IW	CHB	T	VT	L	0
32	shivappa	9887	42	Male	Normal	N	A	A	Absent	Absent	126	**84**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Present	Present	**0**	N	N	106	126	-	-	-	-	Y	Absent	Absent	Absent	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
33	ranganath	24379	38	Male	Raised	N	A	A	Present	Absent	156	**84**	**Absent**	**Present**	130	Absent	Present	Absent	Absent	Present	Absent	Absent	**1**	N	C	130	230	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	PF
34	abdulhamid	9780	48	Male	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Present	Present	**0**	N	N	122	123	-	-	-	-	Y	Present	Absent	Absent	Present	Absent	Absent	Present	AW	LBBB	PT	A	L	0
35	siddanna	23700	66	Male	Normal	N	A	A	Absent	Absent	124	**82**	**Absent**	**Absent**	72	Absent	Present	Absent	Absent	Present	Present	Absent	**0**	N	N	200	130	295	45	268	200	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
36	channappa	24257	45	Male	Normal	N	A	A	Absent	Absent	110	**60**	**Absent**	**Present**	82	Absent	Absent	Absent	Absent	Present	Present	Absent	**1**	N	N	43	350	140	36	68	70	Y	Present	Present	Absent	Present	Absent	Absent	Present	IW	I	NA	A	D	VF
37	nazeer	9773	60	Male	Normal	N	A	A	Absent	Absent	130	**88**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	N	44	131	172	31	107	119	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
38	malkappa	15559	48	Male	Normal	N	A	A	Absent	Absent	120	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	**0**	N	N	48	210	105	32	250	83	Y	Present	Absent	Present	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
39	sayyad	11718	55	Male	Normal	N	A	A	Absent	Absent	100	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	**0**	N	N	379	176	160	27	140	105	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
40	bavasingh	24397	70	Male	Raised	N	A	A	Absent	Present	120	**80**	**Absent**	**Absent**	54	Present	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	N	53	90	181	23	124	133	Y	Present	Absent	Absent	Absent	Present	Absent	Present	IW	CHB	T	LVF	L	0
41	nagu	23755	40	Male	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Absent	Absent	**1**	N	N	60	187	-	-	-	-	Y	Present	Present	Present	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
42	bandappa basappa	8512	68	Female	Normal	N	A	A	Absent	Absent	122	**82**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Present	**0**	N	N	62	132	224	45	221	134	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
43	layappa kesappa	9361	30	Male	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	89	Absent	Present	Present	Absent	Absent	Absent	Absent	**1**	N	N	89	160	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
44	devanagouda	16070	68	Male	Normal	N	A	A	Absent	Absent	110	**88**	**Absent**	**Absent**	84	Absent	Present	Absent	Present	Absent	Present	Absent	**0**	N	N	84	110	-	-	-	-	Y	Absent	Absent	Present	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
45	basappa mallappa	16221	48	Male	Normal	N	A	A	Absent	Absent	110	**84**	**Absent**	**Absent**	82	Absent	Present	Absent	Present	Absent	Absent	Absent	**0**	N	N	36	120	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
46	gurulingappa	17645	72	Male	Normal	N	A	A	Absent	Absent	130	**86**	**Absent**	**Absent**	72	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	76	123	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
47	channabasappa	39217	60	Male	Raised	N	A	A	Absent	Absent	128	**82**	**Absent**	**Absent**	68	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	C	50	132	-	-	-	-	N	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	vt
48	shivappa konappa	26424	71	Male	Raised	N	A	A	Absent	Absent	110	**82**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	C	93	83	198	41	280	101	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
49	gangubai narayan rao	26675	55	Female	Raised	N	A	A	Absent	Present	130	**80**	**Absent**	**Absent**	52	Present	Present	Present	Present	Present	Present	Absent	**0**	N	N	47	106	231	38	229	147	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	CHB	PT	A	L	0
50	bhagavant vittal	26775	80	Male	Raised	N	A	A	Absent	Absent	86	**58**	**Present**	**Absent**	85	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	C	104	123	-	-	-	-	N	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
51	balawwa ningappa	26903	68	Female	Normal	N	A	A	Absent	Absent	110	**80**	**Absent**	**Absent**	74	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	60	109	215	42	70	159	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
52	hanamantraya	26581	53	Male	Raised	N	S3	A	Absent	Present	100	**88**	**Absent**	**Absent**	52	Present	Absent	Absent	Absent	Absent	Present	Absent	**0**	N	C	86	91	180	38	374	67	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	IW	A	NA	CS	L	0
53	dhamiji	27347	35	Male	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	68	Absent	Present	Present	Absent	Absent	Present	Present	**0**	N	N	85	132	-	-	-	-	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
54	mahadevi	27444	34	Female	Normal	N	A	A	Absent	Absent	120	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	N	96	110	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
55	malakubai vittal	27131	62	Male	Normal	N	A	A	Present	Absent	146	**88**	**Absent**	**Present**	102	Absent	Absent	Absent	Absent	Present	Present	Absent	**1**	N	N	87	385	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	CS	D	PF
56	rafiq khajasab	27336	48	Male	Normal	N	A	A	Absent	Absent	110	**60**	**Absent**	**Absent**	98	Absent	Present	Present	Absent	Absent	Present	Absent	**0**	N	N	130	54	233	37	184	159	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	CHB	T	A	L	0
57	shivappa	27783	72	Male	Raised	N	A	A	Absent	Present	90	**60**	**Absent**	**Absent**	46	Present	Absent	Absent	Absent	Absent	Absent	Absent	**0**	N	N	408	110	-	-	-	-	Y	Absent	Absent	Present	Absent	Absent	Absent	Present	IW	CHB	PT	CS	D	PF
58	mahadevappa	27625	63	Male	Normal	N	S3	A	Absent	Absent	110	**86**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	120	112	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
59	shivashankreppa	27581	58	Male	Normal	N	A	A	Absent	Absent	120	**80**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Absent	Present	Absent	**0**	N	N	98	121	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
60	siddu kashiraya	28147	41	Male	Normal	N	A	A	Absent	Absent	120	**80**	**Absent**	**Absent**	87	Absent	Present	Absent	Present	Absent	Present	Absent	**0**	N	N	64	86	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Present	IW	I	T	A	L	0
61	mahantappa	28213	45	Male	Normal	N	A	A	Absent	Absent	158	**84**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Present	Absent	**1**	N	N	74	121	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0

64	bashsab	6426	56	Male	Raised	N	S3	A	Absent	Absent	146	**86**	**Absent**	**Present**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	1	N	C	124	99	226	42	358	112	Y	Absent	Present	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	CS	D	PF
65	lakkavva	3436	75	Female	Normal	N	A	A	Absent	Absent	100	**82**	**Absent**	**Absent**	72	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	40	110	195	40	60	143	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
66	shantabai	41410	55	Female	Normal	N	A	A	Absent	Absent	142	**82**	**Absent**	**Present**	96	Absent	Present	Absent	Absent	Absent	Absent	Absent	1	N	C	50	101	129	30	111	76	Y	Absent	Present	Absent	Absent	Absent	Absent	Present	AW	LBBB	PT	LVF	D	VT
67	babusab	20152	75	Male	Normal	N	S3	A	Present	Absent	158	**84**	**Absent**	**Present**	102	Absent	Present	Absent	Absent	Absent	Present	Absent	1	N	C	56	132	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
68	bhimarayya	31298	65	Male	Normal	N	A	A	Absent	Absent	126	**82**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	40	92	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
69	shivarudrappa	31087	70	Male	Raised	N	A	A	Absent	Present	156	**92**	**Absent**	**Present**	58	Absent	Present	Absent	Absent	Absent	Absent	Absent	1	N	N	86	92	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
70	ramu	31309	61	Male	Raised	N	S3	A	Present	Absent	162	**88**	**Absent**	**Present**	120	Absent	Present	Present	Absent	Absent	Absent	Present	1	N	C	110	96	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
71	rajujawar	30964	61	Male	Normal	N	A	A	Absent	Absent	110	**70**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	86	120	-	-	-	-	Y	Present	Absent	Absent	Present	Absent	Absent	Present	AW	CHB	NA	VT	D	0
72	medinabai	31135	75	Female	Raised	N	S3	A	Absent	Absent	130	**80**	**Absent**	**Absent**	98	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	34	132	132	44	167	88	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
73	shivappa	30007	56	Male	Normal	N	A	A	Absent	Absent	164	**86**	**Absent**	**Present**	72	Absent	Present	Present	Absent	Absent	Absent	Absent	1	N	N	86	180	124	36	265	126	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
74	gugas	30865	55	Male	Normal	N	A	A	Present	Absent	120	**80**	**Absent**	**Absent**	110	Absent	Present	Present	Absent	Present	Present	Absent	0	N	C	64	120	234	40	180	102	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
75	amaranagouda	21249	72	Male	Normal	N	A		Absent	Absent	130	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	0	N	N	46	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
76	mamtaz	30981	60	Female	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	38	183	173	43	29	80	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
77	ishwarappa	29860	50	Male	Normal	N	A	PSM	Absent	Absent	110	**86**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Present	Present	Present	0	N	C	109	151	135	40	60	83	Y	Absent	Absent	Absent	Absent	Absent	Present	Absent	GL	A	NA	CS	D	PF
78	gurusidappa	31559	60	Male	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Present	Absent	1	N	N	51	112	NA	NA	NA	NA	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
79	irappa	26716	53	Male	Raised	N	S3	A	Absent	Present	142	**84**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Present	Present	1	N	N	68	112	126	55	89	53	Y	Absent	Present	Absent	Absent	Absent	Absent	Present	IW	CHB	T	CS	L	0
80	shivalingappa	27771	30	Male	Normal	N	A	A	Absent	Absent	130	**90**	**Absent**	**Present**	78	Absent	Present	Absent	Present	Absent	Present	Absent	1	N	N	40	-	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Present	IW	RBBB	PT	A	L	0
81	basappa	29513	60	Male	Raised	N	A	A	Present	Absent	132	**84**	**Absent**	**Absent**	110	Absent	Present	Absent	Absent	Present	Present	Absent	0	N	N	33	-	210	46	91	95	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
82	yallawwa	28652	58	Female	Normal	N	A	A	Absent	Absent	134	**84**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Absent	Absent	0	N	N	52	89	132	28	215	61	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	VT	L	0
83	shivasharaneppa	35663	80	Male	Raised	N	A	A	Absent	Absent	136	**84**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Absent	Absent	0	N	C	54	91	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Present	Absent	IW	A	NA	CS	D	VF
84	basavraj	21085	35	Male	Raised	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Absent	Present	1	N	N	18	229	157	26	74	116	Y	Absent	Present	Absent	Absent	Absent	Present	Absent	GL	A	NA	A	L	0
85	ashok gidaveer	41189	70	Male	Normal	N	A	A	Absent	Absent	120	**80**	**Absent**	**Present**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	1	N	N	10	203	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Present	IW	I	T	A	L	0
86	kambale	2075	68	Male	Normal	N	A	A	Absent	Absent	134	**80**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	50	68	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
87	krishnappa	861	33	Male	Normal	N	A	A	Absent	Absent	110	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Absent	Absent	0	N	N	52	118	151	21	650	101	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
88	lata joshi	38603	65	Female	Raised	N	S3	A	Present	Absent	50	**30**	**Present**	**Absent**	126	Absent	Present	Absent	Absent	Absent	Absent	Present	0	N	C	58	156	148	35	140	85	Y	Present	Absent	Present	Absent	Absent	Absent	Present	IW	I	T	CS	L	0
89	somashekar	41794	75	Male	Normal	N	S4	A	Absent	Absent	156	**88**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Present	Absent	1	N	N	80	246	166	26	173	105	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
90	madarsab	20152	75	Male	Normal	N	A	A	Absent	Absent	110	**80**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	44	128	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	A	L	0
91	shrimanth	38770	72	Male	Normal	N	A	A	Absent	Absent	134	**80**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	96	132	215	39	100	156	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
92	parusuram	2075	48	Male	Normal	N	S3	A	Present	Absent	160	**100**	**Absent**	**Present**	108	Absent	Present	Absent	Absent	Absent	Absent	Absent	1	N	C	100	207	208	31	198	111	Y	Present	Present	Present	Present	Present	Absent	Absent	AW	A	NA	LVF	L	0
93	pushpa	20571	59	Female	Normal	N	A	A	Absent	Absent	154	**84**	**Absent**	**Present**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	1	N	N	120	130	290	42	311	185	Y	Present	Present	Absent	Present	Absent	Absent	Absent	AW	A	NA	A	L	0
94	lalmohamad	20356	60	Male	Normal	N	A	A	Absent	Present	90	**80**	**Absent**	**Absent**	52	Present	Present	Absent	Absent	Absent	Present	Absent	0	N	N	49	154	226	45	125	156	Y	Present	Absent	Absent	Present	Absent	Absent	Absent	IW	A	NA	A	L	0
95	saimuddinasaheb	19571	62	Male	Normal	N	A	A	Absent	Absent	132	**82**	**Absent**	**Absent**	62	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	27	188	-	-	-	-	Y	Absent	Absent	Absent	Present	Absent	Absent	Absent	AW	A	NA	A	L	0
96	amiramza	3582	36	Male	Normal	N	A	A	Absent	Absent	134	**86**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	120	127	202	34	169	134	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
97	kamala	2191	53	Female	Raised	N	A	A	Absent	Absent	160	**88**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Absent	Absent	1	N	C	72	360	206	45	272	106	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
98	husensab	2351	50	Male	Raised	N	S3	PSM	Present	Absent	130	**82**	**Absent**	**Absent**	102	Absent	Present	Absent	Present	Absent	Present	Present	0	N	C	87	267	187	40	287	98	Y	Present	Absent	Present	Absent	Present	Present	Absent	AW	A	NA	CS	L	0
99	murali	2953	58	Male	Normal	N	A	A	Absent	Present	100	**86**	**Absent**	**Absent**	58	Present	Present	Present	Absent	Absent	Present	Present	0	N	N	180	90	165	25	110	118	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
100	ravi	3374	62	Male	Normal	N	A	A	Absent	Absent	160	**90**	**Absent**	**Present**	84	Absent	Present	Absent	Absent	Absent	Absent	Present	1	N	R	109	345	245	29	343	147	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
101	sadappa satalagoan	1295	60	Male	Raised	N	A	A	Absent	Present	142	**82**	**Absent**	**Present**	52	Present	Present	Absent	Absent	Present	Present	Absent	1	N	N	54	132	-	-	-	-	Y	Absent	Present	Absent	Present	Absent	Present	Present	IW	CHB	T	A	L	0
102	razak	2095	45	Male	Normal	N	S3	A	Absent	Absent	142	**86**	**Absent**	**Present**	60	Absent	Present	Absent	Absent	Present	Absent	Present	1	N	C	200	204	149	40	245	60	Y	Present	Present	Present	Absent	Absent	Absent	Present	IW	CHB	T	VT	L	0
103	dongaresab	3137	64	Male	Normal	N	A	A	Absent	Absent	132	**82**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Present	Present	0	N	N	106	126	-	-	-	-	Y	Absent	Absent	Absent	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
104	tuppad	5635	50	Male	Raised	N	A	A	Present	Absent	166	**88**	**Absent**	**Present**	130	Absent	Present	Absent	Absent	Present	Absent	Absent	1	N	C	130	230	-	-	-	-	N	Present	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	PF
105	mallapa	5736	56	Male	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Present	Present	0	N	N	122	123	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Present	AW	LBBB	PT	A	L	0
106	anil	7151	45	Male	Normal	N	A	A	Absent	Absent	134	**84**	**Absent**	**Absent**	72	Absent	Present	Absent	Absent	Present	Present	Absent	0	N	N	200	130	295	45	268	200	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
107	laxmikanth	8085	42	Male	Normal	N	A	A	Absent	Absent	110	**60**	**Absent**	**Absent**	82	Absent	Absent	Absent	Absent	Present	Present	Absent	0	N	N	43	350	140	36	68	70	Y	Present	Absent	Absent	Absent	Present	Absent	Present	IW	I	NA	A	D	VF
108	madukar	10168	65	Male	Normal	N	A	A	Absent	Absent	130	**84**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Absent	Absent	0	N	N	44	131	172	31	107	119	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
109	ningayya irayya	8232	42	Male	Normal	N	A	A	Absent	Absent	120	**80**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	0	N	N	48	210	105	32	250	83	Y	Absent	Absent	Present	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
110	dundappa huchappa	22642	63	Male	Normal	N	A	A	Absent	Absent	110	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	0	N	N	379	176	160	27	140	105	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
111	laxmibai	7398	75	Female	Raised	N	A	A	Absent	Present	120	**80**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Absent	Absent	1	N	N	53	90	181	23	124	133	Y	Present	Present	Present	Absent	Present	Absent	Present	IW	CHB	T	LVF	L	0
112	mallikarjun	268	47	Male	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Absent	Absent	1	N	N	60	187	-	-	-	-	Y	Present	Present	Absent	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
113	babu masali	490	60	Female	Normal	N	A	A	Absent	Absent	132	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Present	0	N	N	62	132	224	45	221	134	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
114	shantabai	17294	74	Female	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	89	Absent	Present	Present	Absent	Absent	Absent	Absent	1	N	N	89	160	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
115	sahebanna	49229	47	Male	Normal	N	A	A	Absent	Absent	112	**80**	**Absent**	**Absent**	84	Absent	Present	Absent	Present	Absent	Present	Absent	0	N	N	84	110	-	-	-	-	Y	Absent	Absent	Present	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
116	vasant	22805	57	Male	Normal	N	A	A	Absent	Absent	110	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Present	Absent	Absent	Absent	0	N	N	36	120	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
117	vachu	821	55	Male	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	72	Absent	Present	Absent	Present	Absent	Present	Absent	0	N	N	76	123	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
118	guramma sajjan	4242	65	Male	Raised	N	A	A	Absent	Absent	124	**86**	**Absent**	**Absent**	68	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	C	50	132	-	-	-	-	N	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	vt
119	nijalingappa	21914	63	Male	Raised	N	A	A	Absent	Absent	110	**80**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	C	93	83	198	41	280	101	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
120	tayawwa hadappa	2398	60	Female	Raised	N	A	A	Absent	Present	126	**80**	**Absent**	**Absent**	52	Present	Present	Present	Present	Present	Present	Absent	0	N	N	47	106	231	38	229	147	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	CHB	PT	A	L	0
121	dhanasigh	10930	39	Male	Raised	N	A	A	Absent	Absent	84	**56**	**Present**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	C	104	123	-	-	-	-	N	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
122	shankar	15076	60	Male	Normal	N	A	A	Absent	Absent	110	**80**	**Absent**	**Absent**	74	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	60	109	215	42	70	159	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
123	basavaraj ningappa	15485	65	Male	Raised	N	S3	A	Absent	Present	100	**84**	**Absent**	**Absent**	52	Present	Absent	Absent	Absent	Absent	Present	Absent	0	N	C	86	91	180	38	374	67	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	L	0
124	sangappa	30686	39	Male	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	68	Absent	Present	Present	Absent	Absent	Present	Present	0	N	N	85	132	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
125	ganapati	17050	68	Male	Normal	N	A	A	Absent	Absent	120	**86**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	0	N	N	96	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
126	jagadish	24226	67	Male	Normal	N	A	A	Present	Absent	144	**84**	**Absent**	**Present**	102	Absent	Absent	Absent	Absent	Present	Present	Absent	1	N	N	87	385	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	CS	D	PF
127	gangadhar	30865	45	Male	Normal	N	A	A	Absent	Absent	110	**60**	**Absent**	**Absent**	98	Absent	Present	Present	Absent	Absent	Present	Absent	0	N	N	130	54	233	37	184	159	Y	Present	Absent	Present	Absent	Absent	Absent	Present	IW	CHB	T	A	L	0
128	ningappa	25890	83	Male	Raised	N	A	A	Absent	Present	90	**60**	**Absent**	**Absent**	46	Present	Absent	Absent	Absent	Absent	Absent	Absent	0	N	N	408	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Present	Absent	Present	IW	CHB	PT	CS	D	PF
129	prabhu	26720	60	Male	Normal	N	S3	A	Absent	Absent	110	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	120	112	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
130	sanjay	27425	53	Male	Normal	N	A	A	Absent	Absent	120	**80**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Absent	Present	Absent	0	N	N	98	121	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
131	nagamma	24821	65	Female	Normal	N	A	A	Absent	Absent	100	**80**	**Absent**	**Absent**	87	Absent	Present	Absent	Present	Absent	Present	Absent	0	N	N	64	86	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Present	IW	I	T	A	L	0
132	shivappa	27783	63	Male	Normal	N	A	A	Absent	Absent	158	**86**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Present	Absent	1	N	N	74	121	-	-	-	-	Y	Present	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
133	madiwalawwa	2869	75	Female	Normal	N	S3	A	Absent	Absent	120	**70**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Present	Present	0	N	C	64	106	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	LVF	L	0
134	mallanna	3253	45	Male	Normal	N	A	A	Absent	Absent	110	**84**	**Absent**	**Absent**	78	Absent	Present	Absent	Absent	Absent	Present	Absent	0	N	N	78	112	186	38	80	132	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0

137	madiwalappa	5730	49	Male	Normal	N	A	A	Absent	Absent	146	**84**	**Absent**	**Present**	96	Absent	Present	Absent	Absent	Absent	Absent	Absent	**1**	N	C	50	101	129	30	111	76	Y	Present	Present	Present	Absent	Absent	Absent	Present	AW	LBBB	PT	LVF	L	0
138	mallappa	5736	58	Male	Normal	N	S3	A	Present	Absent	154	**92**	**Absent**	**Present**	102	Absent	Present	Absent	Absent	Absent	Present	Absent	**1**	N	C	56	132	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
139	baganna	721	57	Male	Normal	N	A	A	Absent	Absent	134	**84**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	40	92	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
140	babu masali	27550	84	Male	Raised	N	A	A	Absent	Present	158	**90**	**Absent**	**Present**	58	Absent	Present	Absent	Absent	Absent	Absent	Absent	**1**	N	N	86	92	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
141	mahadev	25535	66	Male	Raised	N	S3	A	Present	Absent	160	**96**	**Absent**	**Present**	120	Absent	Present	Present	Absent	Absent	Absent	Present	**1**	N	C	110	96	-	-	-	-	N	Absent	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
142	sonabai	17617	55	Female	Normal	N	A	A	Absent	Absent	110	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	86	120	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
143	prakash	24018	42	Male	Raised	N	S3	A	Absent	Absent	130	**80**	**Absent**	**Absent**	98	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	34	132	132	44	167	88	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
144	ratnabai	33532	56	Female	Normal	N	A	A	Absent	Absent	168	**90**	**Absent**	**Present**	72	Absent	Present	Present	Absent	Absent	Absent	Absent	**1**	N	N	86	180	124	36	265	126	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
145	krishnappa	37550	38	Male	Normal	N	A	A	Present	Absent	120	**80**	**Absent**	**Absent**	110	Absent	Present	Present	Absent	Present	Present	Absent	**0**	N	C	64	120	234	40	180	102	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
146	yamanappa	3036	75	Male	Normal	N	A		Absent	Absent	136	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	N	46	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
147	naik	3374	56	Male	Normal	N	A	A	Absent	Absent	130	**80**	**Absent**	**Absent**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	**0**	N	N	38	183	173	43	29	80	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
148	mahadevi	895	70	Female	Normal	N	A	PSM	Absent	Absent	110	**82**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Present	Present	Present	**0**	N	C	109	151	135	40	60	83	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	CS	D	PF
149	kamalapur	12216	67	Female	Normal	N	A	A	Absent	Absent	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Present	Absent	**1**	N	N	51	112	NA	NA	NA	NA	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
150	madukar	10168	75	Male	Normal	N	A		Absent	Absent	134	**86**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**0**	N	N	46	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0

Sl. No	Name of patient	IP Number	Age in years	sex	jvp	Heart sounds	Additional sounds	Murmurs	Tachycardia	Bradycardia	SBP	DBP	hypotension	Hypertension	Pulse	Bradycardia	Chest pain	sweating	breathlessness	palpitation	Vomiting	giddiness	Hypertension	Rub	Resp system	CK	RBS	Total cholesterol	LDL	HDL	TG	Thrombolysis	Smoking	HTN	DM	Obesity	Sedentary life	Family h/o	Coonduction block Present/Absent	SITE	Type	Timing	Com	Outcome	Causes of death
1	chanasab mulk	28502	54	Male	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	38	183	173	43	29	80	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	RBBB	NA	A	L	0
2	ashik laxman	28359	70	Male	N	N	A	PSM	A	A	110	**84**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Present	Present	Present	**A**	N	C	109	151	135	40	60	83	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	CS	D	PF
3	putalabai	26051	55	Female	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	51	112	NA	NA	NA	NA	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	D	VT
4	kallapan	28564	72	Male	Raised	N	S3	A	A	P	142	**82**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Present	Present	**P**	N	N	68	112	126	55	89	53	Y	Absent	Present	Present	Absent	Present	Absent	Present	IW	CHB	T	CS	L	0
5	vitabhai	28547	50	Female	N	N	A	A	A	A	130	**90**	**Absent**	**Present**	78	Absent	Present	Absent	Present	Absent	Present	Absent	**P**	N	N	40	-	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Present	IW	RBBB	PT	CS	D	PF
6	panehappa biradar	28476	86	Male	Raised	N	A	A	P	A	120	**88**	**Absent**	**Absent**	110	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	33	-	210	46	91	95	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
7	sukudev ram	27539	58	Male	N	N	A	A	A	A	122	**84**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	52	89	132	28	215	61	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	PF
8	Tibunnisa	26850	60	Female	Raised	N	A	A	A	A	124	**82**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	C	54	91	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	VF
9	siddanagouda	8761	60	Female	Raised	N	A	A	A	A	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Absent	Present	**P**	N	N	18	229	157	26	74	116	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	GL	A	NA	A	L	0
10	manohar laxman	26929	64	Male	N	N	A	A	A	A	120	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	10	203	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	I	T	A	L	0
11	layappa	9361	68	Male	N	N	A	A	A	A	134	**82**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	50	68	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
12	mehabub	9078	65	Male	N	N	A	A	A	A	110	**84**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Absent	Absent	**A**	N	N	52	118	151	21	650	101	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
13	somalingappa	27532	38	Male	Raised	N	S3	A	P	A	60	**50**	**Present**	**Absent**	126	Absent	Present	Absent	Absent	Absent	Absent	Present	**A**	N	C	58	156	148	35	140	85	Y	Absent	Absent	Present	Absent	Absent	Absent	Present	IW	I	T	CS	L	0
14	nagappa	15210	71	Male	N	N	S4	A	A	A	162	**92**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	80	246	166	26	173	105	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
15	vijay kumar	14404	60	Male	N	N	A	A	A	A	110	**86**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	44	128	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	A	L	0
16	lakkamma	11028	60	Female	N	N	A	A	A	A	134	**84**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	96	132	215	39	100	156	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
17	gouramma	11551	68	Female	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	76	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	N	100	207	208	31	198	111	Y	Present	Present	Absent	Present	Present	Absent	Absent	AW	A	NA	A	L	0
18	siddabai	40187	65	Female	N	N	A	A	P	A	142	**94**	**Absent**	**Present**	108	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	C	120	130	290	42	311	185	Y	Present	Present	Absent	Present	Absent	Absent	Absent	AW	A	NA	LVF	L	0
19	ashok basavaraj	39437	63	Male	N	N	A	A	A	P	90	**82**	**Absent**	**Absent**	52	Present	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	49	154	226	45	125	156	Y	Present	Absent	Present	Present	Absent	Absent	Absent	IW	A	NA	A	L	0
20	appanna	820	52	Male	N	N	A	A	A	A	126	**84**	**Absent**	**Absent**	62	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	27	188	-	-	-	-	Y	Absent	Absent	Present	Present	Absent	Absent	Absent	AW	A	NA	A	L	0
21	ningamma	2219	85	Female	N	N	A	A	A	A	128	**86**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	120	127	202	34	169	134	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
22	chambasappa	18225	58	Male	Raised	N	A	A	A	A	146	**84**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	C	72	360	206	45	272	106	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
23	siddanagouda	8761	65	Male	Raised	N	S3	PSM	P	A	130	**84**	**Absent**	**Absent**	102	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	C	87	267	187	40	287	98	Y	Present	Absent	Present	Absent	Present	Present	Absent	AW	A	NA	CS	L	0
24	amasidda ramanna	21939	60	Male	N	N	A	A	A	P	100	**86**	**Absent**	**Absent**	58	Present	Present	Present	Absent	Absent	Present	Present	**A**	N	N	180	90	165	25	110	118	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
25	sidramappa	8279	65	Male	N	N	A	A	A	A	190	**120**	**Absent**	**Present**	84	Absent	Present	Absent	Absent	Absent	Absent	Present	**P**	N	R	109	345	245	29	343	147	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
26	mehabub	9078	65	Male	N	N	A	A	A	A	110	**82**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	55	109	181	42	135	112	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
27	sharanappa	26111	65	Male	Raised	N	S3	A	A	A	88	**58**	**Present**	**Absent**	92	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	C	54	110	-	-	-	-	N	Absent	Absent	Present	Absent	Absent	Absent	Absent	IW	A	NA	SVT	L	0
28	padmavathi	11914	50	Female	N	N	A	A	A	P	90	**60**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	100	130	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Present	IW	CHB	T	A	L	0
29	nanasaheb	11968	67	Male	N	N	A	A	A	A	110	**80**	**Absent**	**Absent**	76	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	N	>200	120	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	AW	I	T	A	L	0
30	laxmibai	24389	68	Female	Raised	N	A	A	A	P	144	**88**	**Absent**	**Present**	52	Present	Present	Absent	Absent	Present	Present	Absent	**P**	N	N	54	132	-	-	-	-	Y	Absent	Present	Absent	Absent	Absent	Present	Present	IW	CHB	T	A	L	0
31	hanamanth	9888	35	Male	N	N	S3	A	A	A	142	**86**	**Absent**	**Present**	60	Absent	Present	Absent	Absent	Present	Absent	Present	**P**	N	C	200	204	149	40	245	60	Y	Present	Present	Present	Absent	Absent	Absent	Present	IW	CHB	T	VT	L	0
32	shivappa	9887	42	Male	N	N	A	A	A	A	126	**84**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Present	Present	**A**	N	N	106	126	-	-	-	-	Y	Absent	Absent	Absent	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
33	ranganath	24379	38	Male	Raised	N	A	A	P	A	156	**84**	**Absent**	**Present**	130	Absent	Present	Absent	Absent	Present	Absent	Absent	**P**	N	C	130	230	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	PF
34	abdulhamid	9780	48	Male	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Present	Present	**A**	N	N	122	123	-	-	-	-	Y	Present	Absent	Absent	Present	Absent	Absent	Present	AW	LBBB	PT	A	L	0
35	siddanna	23700	66	Male	N	N	A	A	A	A	124	**82**	**Absent**	**Absent**	72	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	200	130	295	45	268	200	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
36	channappa	24257	45	Male	N	N	A	A	A	A	110	**60**	**Absent**	**Present**	82	Absent	Absent	Absent	Absent	Present	Present	Absent	**P**	N	N	43	350	140	36	68	70	Y	Present	Present	Absent	Present	Absent	Absent	Present	IW	I	NA	A	D	VF
37	nazeer	9773	60	Male	N	N	A	A	A	A	130	**88**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	44	131	172	31	107	119	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
38	malkappa	15559	48	Male	N	N	A	A	A	A	120	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	48	210	105	32	250	83	Y	Present	Absent	Present	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
39	sayyad	11718	55	Male	N	N	A	A	A	A	100	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	379	176	160	27	140	105	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
40	bavasingh	24397	70	Male	Raised	N	A	A	A	P	120	**80**	**Absent**	**Absent**	54	Present	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	53	90	181	23	124	133	Y	Present	Absent	Absent	Absent	Present	Absent	Present	IW	CHB	T	LVF	L	0
41	nagu	23755	40	Male	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	N	60	187	-	-	-	-	Y	Present	Present	Present	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
42	bandappa basappa	8512	68	Female	N	N	A	A	A	A	122	**82**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	62	132	224	45	221	134	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
43	layappa kesappa	9361	30	Male	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	89	Absent	Present	Present	Absent	Absent	Absent	Absent	**P**	N	N	89	160	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
44	devanagouda	16070	68	Male	N	N	A	A	A	A	110	**88**	**Absent**	**Absent**	84	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	N	84	110	-	-	-	-	Y	Absent	Absent	Present	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
45	basappa mallappa	16221	48	Male	N	N	A	A	A	A	110	**84**	**Absent**	**Absent**	82	Absent	Present	Absent	Present	Absent	Absent	Absent	**A**	N	N	36	120	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
46	gurulingappa	17645	72	Male	N	N	A	A	A	A	130	**86**	**Absent**	**Absent**	72	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	N	76	123	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
47	channabasappa	39217	60	Male	Raised	N	A	A	A	A	128	**82**	**Absent**	**Absent**	68	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	C	50	132	-	-	-	-	N	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	vt
48	shivappa konappa	26424	71	Male	Raised	N	A	A	A	A	110	**82**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	C	93	83	198	41	280	101	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
49	gangubai narayan rao	26675	55	Female	Raised	N	A	A	A	P	130	**80**	**Absent**	**Absent**	52	Present	Present	Present	Present	Present	Present	Absent	**A**	N	N	47	106	231	38	229	147	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	CHB	PT	A	L	0
50	bhagavant vittal	26775	80	Male	Raised	N	A	A	A	A	86	**58**	**Present**	**Absent**	85	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	C	104	123	-	-	-	-	N	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
51	balawwa ningappa	26903	68	Female	N	N	A	A	A	A	110	**80**	**Absent**	**Absent**	74	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	60	109	215	42	70	159	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
52	hanamantraya	26581	53	Male	Raised	N	S3	A	A	P	100	**88**	**Absent**	**Absent**	52	Present	Absent	Absent	Absent	Absent	Present	Absent	**A**	N	C	86	91	180	38	374	67	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	IW	A	NA	CS	L	0
53	dhamiji	27347	35	Male	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	68	Absent	Present	Present	Absent	Absent	Present	Present	**A**	N	N	85	132	-	-	-	-	Y	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
54	mahadevi	27444	34	Female	N	N	A	A	A	A	120	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	96	110	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
55	malakubai vittal	27131	62	Male	N	N	A	A	P	A	146	**88**	**Absent**	**Present**	102	Absent	Absent	Absent	Absent	Present	Present	Absent	**P**	N	N	87	385	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	CS	D	PF
56	rafiq khajasab	27336	48	Male	N	N	A	A	A	A	110	**60**	**Absent**	**Absent**	98	Absent	Present	Present	Absent	Absent	Present	Absent	**A**	N	N	130	54	233	37	184	159	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	CHB	T	A	L	0
57	shivappa	27783	72	Male	Raised	N	A	A	A	P	90	**60**	**Absent**	**Absent**	46	Present	Absent	Absent	Absent	Absent	Absent	Absent	**A**	N	N	408	110	-	-	-	-	Y	Absent	Absent	Present	Absent	Absent	Absent	Present	IW	CHB	PT	CS	D	PF
58	mahadevappa	27625	63	Male	N	N	S3	A	A	A	110	**86**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	120	112	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
59	shivashankreppa	27581	58	Male	N	N	A	A	A	A	120	**80**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Absent	Present	Absent	**A**	N	N	98	121	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
60	siddu kashiraya	28147	41	Male	N	N	A	A	A	A	120	**80**	**Absent**	**Absent**	87	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	N	64	86	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Present	IW	I	T	A	L	0
61	mahantappa	28213	45	Male	N	N	A	A	A	A	158	**84**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	74	121	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0

64	bashsab	6426	56	Male	Raised	N	S3	A	A	A	146	**86**	**Absent**	**Present**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	C	124	99	226	42	358	112	Y	Absent	Present	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	CS	D	PF
65	lakkavva	3436	75	Female	N	N	A	A	A	A	100	**82**	**Absent**	**Absent**	72	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	40	110	195	40	60	143	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
66	shantabai	41410	55	Female	N	N	A	A	A	A	142	**82**	**Absent**	**Present**	96	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	C	50	101	129	30	111	76	Y	Absent	Present	Absent	Absent	Absent	Absent	Present	AW	LBBB	PT	LVF	D	VT
67	babusab	20152	75	Male	N	N	S3	A	P	A	158	**84**	**Absent**	**Present**	102	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	C	56	132	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
68	bhimarayya	31298	65	Male	N	N	A	A	A	A	126	**82**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	40	92	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
69	shivarudrappa	31087	70	Male	Raised	N	A	A	A	P	156	**92**	**Absent**	**Present**	58	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	N	86	92	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
70	ramu	31309	61	Male	Raised	N	S3	A	P	A	162	**88**	**Absent**	**Present**	120	Absent	Present	Present	Absent	Absent	Absent	Present	**P**	N	C	110	96	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
71	rajujawar	30964	61	Male	N	N	A	A	A	A	110	**70**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	86	120	-	-	-	-	Y	Present	Absent	Absent	Present	Absent	Absent	Present	AW	CHB	NA	VT	D	0
72	medinabai	31135	75	Female	Raised	N	S3	A	A	A	130	**80**	**Absent**	**Absent**	98	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	34	132	132	44	167	88	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
73	shivappa	30007	56	Male	N	N	A	A	A	A	164	**86**	**Absent**	**Present**	72	Absent	Present	Present	Absent	Absent	Absent	Absent	**P**	N	N	86	180	124	36	265	126	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
74	gugas	30865	55	Male	N	N	A	A	P	A	120	**80**	**Absent**	**Absent**	110	Absent	Present	Present	Absent	Present	Present	Absent	**A**	N	C	64	120	234	40	180	102	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
75	amaranagouda	21249	72	Male	N	N	A		A	A	130	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	46	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
76	mamtaz	30981	60	Female	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	38	183	173	43	29	80	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
77	ishwarappa	29860	50	Male	N	N	A	PSM	A	A	110	**86**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Present	Present	Present	**A**	N	C	109	151	135	40	60	83	Y	Absent	Absent	Absent	Absent	Absent	Present	Absent	GL	A	NA	CS	D	PF
78	gurusidappa	31559	60	Male	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	51	112	NA	NA	NA	NA	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
79	irappa	26716	53	Male	Raised	N	S3	A	A	P	142	**84**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Present	Present	**P**	N	N	68	112	126	55	89	53	Y	Absent	Present	Absent	Absent	Absent	Absent	Present	IW	CHB	T	CS	L	0
80	shivalingappa	27771	30	Male	N	N	A	A	A	A	130	**90**	**Absent**	**Present**	78	Absent	Present	Absent	Present	Absent	Present	Absent	**P**	N	N	40	-	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Present	IW	RBBB	PT	A	L	0
81	basappa	29513	60	Male	Raised	N	A	A	P	A	132	**84**	**Absent**	**Absent**	110	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	33	-	210	46	91	95	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
82	yallawwa	28652	58	Female	N	N	A	A	A	A	134	**84**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	52	89	132	28	215	61	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	VT	L	0
83	shivasharaneppa	35663	80	Male	Raised	N	A	A	A	A	136	**84**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	C	54	91	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Present	Absent	IW	A	NA	CS	D	VF
84	basavraj	21085	35	Male	Raised	N	A	A	A	A	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Absent	Present	**P**	N	N	18	229	157	26	74	116	Y	Absent	Present	Absent	Absent	Absent	Present	Absent	GL	A	NA	A	L	0
85	ashok gidaveer	41189	70	Male	N	N	A	A	A	A	120	**80**	**Absent**	**Present**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	10	203	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Present	IW	I	T	A	L	0
86	kambale	2075	68	Male	N	N	A	A	A	A	134	**80**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	50	68	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
87	krishnappa	861	33	Male	N	N	A	A	A	A	110	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Absent	Absent	**A**	N	N	52	118	151	21	650	101	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
88	lata joshi	38603	65	Female	Raised	N	S3	A	P	A	50	**30**	**Present**	**Absent**	126	Absent	Present	Absent	Absent	Absent	Absent	Present	**A**	N	C	58	156	148	35	140	85	Y	Present	Absent	Present	Absent	Absent	Absent	Present	IW	I	T	CS	L	0
89	somashekar	41794	75	Male	N	N	S4	A	A	A	156	**88**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	80	246	166	26	173	105	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
90	madarsab	20152	75	Male	N	N	A	A	A	A	110	**80**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	44	128	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	A	L	0
91	shrimanth	38770	72	Male	N	N	A	A	A	A	134	**80**	**Absent**	**Absent**	96	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	96	132	215	39	100	156	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
92	parusuram	2075	48	Male	N	N	S3	A	P	A	160	**100**	**Absent**	**Present**	108	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	C	100	207	208	31	198	111	Y	Present	Present	Present	Present	Present	Absent	Absent	AW	A	NA	LVF	L	0
93	pushpa	20571	59	Female	N	N	A	A	A	A	154	**84**	**Absent**	**Present**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	120	130	290	42	311	185	Y	Present	Present	Absent	Present	Absent	Absent	Absent	AW	A	NA	A	L	0
94	lalmohamad	20356	60	Male	N	N	A	A	A	P	90	**80**	**Absent**	**Absent**	52	Present	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	49	154	226	45	125	156	Y	Present	Absent	Absent	Present	Absent	Absent	Absent	IW	A	NA	A	L	0
95	saimuddinasaheb	19571	62	Male	N	N	A	A	A	A	132	**82**	**Absent**	**Absent**	62	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	27	188	-	-	-	-	Y	Absent	Absent	Absent	Present	Absent	Absent	Absent	AW	A	NA	A	L	0
96	amiramza	3582	36	Male	N	N	A	A	A	A	134	**86**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	120	127	202	34	169	134	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
97	kamala	2191	53	Female	Raised	N	A	A	A	A	160	**88**	**Absent**	**Present**	100	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	C	72	360	206	45	272	106	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
98	husensab	2351	50	Male	Raised	N	S3	PSM	P	A	130	**82**	**Absent**	**Absent**	102	Absent	Present	Absent	Present	Absent	Present	Present	**A**	N	C	87	267	187	40	287	98	Y	Present	Absent	Present	Absent	Present	Present	Absent	AW	A	NA	CS	L	0
99	murali	2953	58	Male	N	N	A	A	A	P	100	**86**	**Absent**	**Absent**	58	Present	Present	Present	Absent	Absent	Present	Present	**A**	N	N	180	90	165	25	110	118	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
100	ravi	3374	62	Male	N	N	A	A	A	A	160	**90**	**Absent**	**Present**	84	Absent	Present	Absent	Absent	Absent	Absent	Present	**A**	N	R	109	345	245	29	343	147	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
101	sadappa satalagoan	1295	60	Male	Raised	N	A	A	A	P	142	**82**	**Absent**	**Present**	52	Present	Present	Absent	Absent	Present	Present	Absent	**P**	N	N	54	132	-	-	-	-	Y	Absent	Present	Absent	Present	Absent	Present	Present	IW	CHB	T	A	L	0
102	razak	2095	45	Male	N	N	S3	A	A	A	142	**86**	**Absent**	**Present**	60	Absent	Present	Absent	Absent	Present	Absent	Present	**P**	N	C	200	204	149	40	245	60	Y	Present	Present	Present	Absent	Absent	Absent	Present	IW	CHB	T	VT	L	0
103	dongaresab	3137	64	Male	N	N	A	A	A	A	132	**82**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Present	Present	**A**	N	N	106	126	-	-	-	-	Y	Absent	Absent	Absent	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
104	tuppad	5635	50	Male	Raised	N	A	A	P	A	166	**88**	**Absent**	**Present**	130	Absent	Present	Absent	Absent	Present	Absent	Absent	**P**	N	C	130	230	-	-	-	-	N	Present	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	D	PF
105	mallapa	5736	56	Male	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Present	Present	Present	**A**	N	N	122	123	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Present	AW	LBBB	PT	A	L	0
106	anil	7151	45	Male	N	N	A	A	A	A	134	**84**	**Absent**	**Absent**	72	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	200	130	295	45	268	200	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
107	laxmikanth	8085	42	Male	N	N	A	A	A	A	110	**60**	**Absent**	**Absent**	82	Absent	Absent	Absent	Absent	Present	Present	Absent	**A**	N	N	43	350	140	36	68	70	Y	Present	Absent	Absent	Absent	Present	Absent	Present	IW	I	NA	A	D	VF
108	madukar	10168	65	Male	N	N	A	A	A	A	130	**84**	**Absent**	**Absent**	88	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	44	131	172	31	107	119	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
109	ningayya irayya	8232	42	Male	N	N	A	A	A	A	120	**80**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	48	210	105	32	250	83	Y	Absent	Absent	Present	Absent	Present	Absent	Absent	IW	A	NA	A	L	0
110	dundappa huchappa	22642	63	Male	N	N	A	A	A	A	110	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Present	Present	Absent	**A**	N	N	379	176	160	27	140	105	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
111	laxmibai	7398	75	Female	Raised	N	A	A	A	P	120	**80**	**Absent**	**Present**	54	Present	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	N	53	90	181	23	124	133	Y	Present	Present	Present	Absent	Present	Absent	Present	IW	CHB	T	LVF	L	0
112	mallikarjun	268	47	Male	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	N	60	187	-	-	-	-	Y	Present	Present	Absent	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
113	babu masali	490	60	Female	N	N	A	A	A	A	132	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Present	**A**	N	N	62	132	224	45	221	134	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
114	shantabai	17294	74	Female	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	89	Absent	Present	Present	Absent	Absent	Absent	Absent	**P**	N	N	89	160	-	-	-	-	Y	Absent	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
115	sahebanna	49229	47	Male	N	N	A	A	A	A	112	**80**	**Absent**	**Absent**	84	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	N	84	110	-	-	-	-	Y	Absent	Absent	Present	Absent	Present	Absent	Absent	AW	A	NA	A	L	0
116	vasant	22805	57	Male	N	N	A	A	A	A	110	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Present	Absent	Absent	Absent	**A**	N	N	36	120	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
117	vachu	821	55	Male	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	72	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	N	76	123	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
118	guramma sajjan	4242	65	Male	Raised	N	A	A	A	A	124	**86**	**Absent**	**Absent**	68	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	C	50	132	-	-	-	-	N	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	vt
119	nijalingappa	21914	63	Male	Raised	N	A	A	A	A	110	**80**	**Absent**	**Absent**	84	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	C	93	83	198	41	280	101	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	LVF	L	0
120	tayawwa hadappa	2398	60	Female	Raised	N	A	A	A	P	126	**80**	**Absent**	**Absent**	52	Present	Present	Present	Present	Present	Present	Absent	**A**	N	N	47	106	231	38	229	147	Y	Present	Absent	Absent	Absent	Absent	Absent	Present	IW	CHB	PT	A	L	0
121	dhanasigh	10930	39	Male	Raised	N	A	A	A	A	84	**56**	**Present**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	C	104	123	-	-	-	-	N	Absent	Absent	Present	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
122	shankar	15076	60	Male	N	N	A	A	A	A	110	**80**	**Absent**	**Absent**	74	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	60	109	215	42	70	159	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
123	basavaraj ningappa	15485	65	Male	Raised	N	S3	A	A	P	100	**84**	**Absent**	**Absent**	52	Present	Absent	Absent	Absent	Absent	Present	Absent	**A**	N	C	86	91	180	38	374	67	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	CS	L	0
124	sangappa	30686	39	Male	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	68	Absent	Present	Present	Absent	Absent	Present	Present	**A**	N	N	85	132	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
125	ganapati	17050	68	Male	N	N	A	A	A	A	120	**86**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	96	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
126	jagadish	24226	67	Male	N	N	A	A	P	A	144	**84**	**Absent**	**Present**	102	Absent	Absent	Absent	Absent	Present	Present	Absent	**P**	N	N	87	385	-	-	-	-	N	Present	Present	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	CS	D	PF
127	gangadhar	30865	45	Male	N	N	A	A	A	A	110	**60**	**Absent**	**Absent**	98	Absent	Present	Present	Absent	Absent	Present	Absent	**A**	N	N	130	54	233	37	184	159	Y	Present	Absent	Present	Absent	Absent	Absent	Present	IW	CHB	T	A	L	0
128	ningappa	25890	83	Male	Raised	N	A	A	A	P	90	**60**	**Absent**	**Absent**	46	Present	Absent	Absent	Absent	Absent	Absent	Absent	**A**	N	N	408	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Present	Absent	Present	IW	CHB	PT	CS	D	PF
129	prabhu	26720	60	Male	N	N	S3	A	A	A	110	**80**	**Absent**	**Absent**	82	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	120	112	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
130	sanjay	27425	53	Male	N	N	A	A	A	A	120	**80**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Absent	Present	Absent	**A**	N	N	98	121	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
131	nagamma	24821	65	Female	N	N	A	A	A	A	100	**80**	**Absent**	**Absent**	87	Absent	Present	Absent	Present	Absent	Present	Absent	**A**	N	N	64	86	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Present	IW	I	T	A	L	0
132	shivappa	27783	63	Male	N	N	A	A	A	A	158	**86**	**Absent**	**Present**	72	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	74	121	-	-	-	-	Y	Present	Present	Present	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
133	madiwalawwa	2869	75	Female	N	N	S3	A	A	A	120	**70**	**Absent**	**Absent**	100	Absent	Present	Absent	Absent	Absent	Present	Present	**A**	N	C	64	106	-	-	-	-	Y	Present	Absent	Present	Absent	Absent	Absent	Present	AW	RBBB	PT	LVF	L	0
134	mallanna	3253	45	Male	N	N	A	A	A	A	110	**84**	**Absent**	**Absent**	78	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	78	112	186	38	80	132	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0

137	madiwalappa	5730	49	Male	N	N	A	A	A	A	146	**84**	**Absent**	**Present**	96	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	C	50	101	129	30	111	76	Y	Present	Present	Present	Absent	Absent	Absent	Present	AW	LBBB	PT	LVF	L	0
138	mallappa	5736	58	Male	N	N	S3	A	P	A	154	**92**	**Absent**	**Present**	102	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	C	56	132	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
139	baganna	721	57	Male	N	N	A	A	A	A	134	**84**	**Absent**	**Absent**	76	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	40	92	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
140	babu masali	27550	84	Male	Raised	N	A	A	A	P	158	**90**	**Absent**	**Present**	58	Absent	Present	Absent	Absent	Absent	Absent	Absent	**P**	N	N	86	92	-	-	-	-	Y	Present	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
141	mahadev	25535	66	Male	Raised	N	S3	A	P	A	160	**96**	**Absent**	**Present**	120	Absent	Present	Present	Absent	Absent	Absent	Present	**P**	N	C	110	96	-	-	-	-	N	Absent	Present	Absent	Absent	Absent	Absent	Absent	AW	A	NA	CS	D	PF
142	sonabai	17617	55	Female	N	N	A	A	A	A	110	**84**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	86	120	-	-	-	-	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
143	prakash	24018	42	Male	Raised	N	S3	A	A	A	130	**80**	**Absent**	**Absent**	98	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	34	132	132	44	167	88	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
144	ratnabai	33532	56	Female	N	N	A	A	A	A	168	**90**	**Absent**	**Present**	72	Absent	Present	Present	Absent	Absent	Absent	Absent	**P**	N	N	86	180	124	36	265	126	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
145	krishnappa	37550	38	Male	N	N	A	A	P	A	120	**80**	**Absent**	**Absent**	110	Absent	Present	Present	Absent	Present	Present	Absent	**A**	N	C	64	120	234	40	180	102	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	LVF	L	0
146	yamanappa	3036	75	Male	N	N	A		A	A	136	**82**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	46	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0
147	naik	3374	56	Male	N	N	A	A	A	A	130	**80**	**Absent**	**Absent**	90	Absent	Present	Absent	Absent	Absent	Present	Absent	**A**	N	N	38	183	173	43	29	80	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
148	mahadevi	895	70	Female	N	N	A	PSM	A	A	110	**82**	**Absent**	**Absent**	86	Absent	Present	Present	Absent	Present	Present	Present	**A**	N	C	109	151	135	40	60	83	Y	Absent	Absent	Absent	Absent	Absent	Absent	Absent	GL	A	NA	CS	D	PF
149	kamalapur	12216	67	Female	N	N	A	A	A	A	160	**100**	**Absent**	**Present**	88	Absent	Present	Absent	Absent	Absent	Present	Absent	**P**	N	N	51	112	NA	NA	NA	NA	Y	Absent	Present	Absent	Absent	Absent	Absent	Absent	IW	A	NA	A	L	0
150	madukar	10168	75	Male	N	N	A		A	A	134	**86**	**Absent**	**Absent**	86	Absent	Present	Absent	Absent	Absent	Absent	Absent	**A**	N	N	46	110	-	-	-	-	Y	Present	Absent	Absent	Absent	Absent	Absent	Absent	AW	A	NA	A	L	0