"CLINICAL PROFILE OF LEFT BUNDLE BRANCH BLOCK IN TERTIARY CARE HOSPITAL- A CROSS SECTIONAL STUDY"

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LIST OF ABBREVIATIONS USED

AR	-	Aortic Regurgitation
AS	-	Aortic Stenosis
ASMI	-	Antero Septal MI
AV block	-	Atrioventricular Block
AVN	-	Atrio Ventricular Node
BBB	-	Bundle Branch Block
CAD	-	Coronary Artery Disease
CAG	-	Coronary Angiography
CRT	-	Cardiac Resynchronisation Therapy
DBP	-	Diastolic Blood Presssure
DCM	-	Dilated Cardiomyopathy
DDF	-	Diastolic Dysfunction
DVD	-	Double Vessel Disease
ECG	-	Electrocardiogram
ECHO	-	Echocardiography
HBA1C	-	Glycosylated Hemoglobin
HF	-	Heart Failure
LA	-	Left Atrium
LV	-	Left Ventricle
LAD Artery	-	Left Anterior Descending Artery
LAFB	-	Left Anterior Fascicular Block
LBBB	-	Left Bundle Branch Block
LBB	-	Left Bundle Branch

LCX Artery	-	Left Circumflex Artery
LPFB	-	Left Posterior Fascicular Block
LVH	-	Left Ventricular Hypertrophy
LVEDd	-	Left Ventricular End Diastolic diameter
LVESd	-	Left Ventricular End Systolic diameter
MI	-	Myocardial Infarction
MR	-	Mitral Regurgitation
PAH	-	Pulmonary Arterial Hypertension
RA	-	Right Atrium
RV	-	Right Ventricle
RBBB	-	Right Bundle Branch Block
RBB	-	Right Bundle Branch
RCA	-	Right Coronary Artery
RHD	-	Rheumatic Heart Disease
RVH	-	Right Ventricular Hypertrophy
RWMA	-	Regional Wall Motion Abnormality
SAN	-	Sino Atrial Node
SBP	-	Systolic Blood Pressure
STEMI	-	ST Elevation Myocardial Infarction
SVD	-	Single Vessel Disease
TVD	-	Triple Vessel Disease
VT	-	Ventricular Tachycardia
WMA	-	Wall Motion Abnormality

ABSTRACT

Background and Objectives

LBBB is a cardiac conduction defect in which activation of left ventricle is delayed which causes left ventricle to contract later than right ventricle. The objective of the study is to assess the aetiology, clinical features and left ventricular functions on 2D ECHO in patients with LBBB.

Methodology

The present one year cross sectional study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 100 patients diagnosed to have LBBB on ECG, during the period of January 2018 to December 2018. A detailed history, physical examination and routine laboratory investigations were done. ECG and 2DECHO were obtained, QRS duration measured, and was compared to EJECTION FRACTION on 2DECHO.

Results

The mean age of the patients was 63.12 years. Prevalence of LBBB was slightly greater in males. The most common presenting complaint was Breathlessness. Hypertension, diabetes mellitus and smoking were noted to be commonly associated with LBBB. The most common etiology for LBBB was found to be ischemic heart disease. QRS duration, ejection fraction and Left ventricle diameters were found to be statistically significant in association with etiology of LBBB. Left ventricular systolic and diastolic dysfunction was a common finding present in patients with LBBB. Most common valvular lesion associated with LBBB was mitral regurgitation as per our

study.

QRS duration had a negative correlation with ejection fraction which was statistically significant. Among the patients who were subjected to coronary angiography, single vessel disease was found to be the most common lesion. The most common vessel implicated was left anterior descending followed by right coronary artery.

Conclusion and interpretation

LBBB is a cardiac conduction defect which causes both LV systolic and Diastolic dysfunction leading to decreased cardiac performance. Most common presenting symptom is breathlessness and the most common etiology is IHD. QRS duration has a negative correlation with ejection fraction and forms the basis for CRT. Every patient presenting with LBBB should be subjected to extensive cardiac workup to rule out any underlying serious cardiac condition.

Keywords: LBBB; 2D ECHO; Ejection Fraction; Breathlessness; QRS duration; IHD

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INTRODUCTION

LBBB is a cardiac conduction defect in which the normal direction of septal depolarisation is reversed, becomes right to left as the impulse spreads first to the RV via the right bundle branch and then to the LV via the septum [1,2]. In LBBB, conduction abnormality alters the activation sequence of ventricles which results into distortion of QRS complex and ST segment and T wave secondary changes on ECG. Left bundle branch block diagnosis on an electrocardiogram is significantly associated with increased risk of morbidity and mortality [7]. LBBB may be present without any structural heart disease in young population (Isolated LBBB) and is associated with a good prognosis [3-5].However, as the age progresses, LBBB occurs due to underlying heart disease (e.g. hypertension, coronary artery disease, valvular, cardiomyopathy), where it acts as an independent predictor of poorer cardiovascular outcomes [4-7]

The abnormal activation pattern of ventricles in LBBB induces hemodynamic changes that get superimposed on the abnormalities due to underlying cardiac disease. The pattern of ventricular activation is less coordinated and requires much more time because of abnormal sequence of activation of ventricles. The result is asynchronous and prolonged contraction of left ventricles, altered motion of septum and reduced diastolic filling time [9] that results in regional differences in workload; regional changes in flow of blood and metabolism; functional mitral valve dysfunction with mitral regurgitation; negative LV remodeling. As a result, cardiac efficiency is further reduced. Severe left ventricular dysfunction is common, in which there is a delay of more than 60 milliseconds between lateral wall contraction and septal wall contraction.

LBBB prevalence increases from 0.4% at age 50 to 6.5% at age 80[8]. Majority of the patients usually have organic cardiovascular disease such as antecedent hypertension, coronary artery disease or dilated cardiomyopathy at the time of diagnosis of LBBB [2-7]. It can also occur as an isolated abnormality in asymptomatic young patients [3-5]. However, even in isolated LBBB, they will eventually go on to develop one of these cardiovascular abnormalities which translate into a higher mortality. The major causes of death are due to myocardial infarction, heart failure, and arrhythmias including high-grade AV block. In patients with heart failure and LBBB, they carry a poorer prognosis compared to those without LBBB. The prognosis in these patients depends on the duration of QRS complexes. An inverse relationship between duration of QRS complex and ejection fraction of left ventricles has been observed in patients with LBBB [10]. Longer the QRS duration worse the prognosis. The occurrence of LBBB in patients with CAD including acute STEMI correlates with more severe disease, more severe dysfunction of left ventricles and often decreased survival rates [7.12-15]. LBBB associated with left or right axis deviation presents with severe disease symptoms [14, 16, 17]. Left axis deviation in LBBB is associated with severe disease of conduction system involving the main left bundle, and the fascicles. Right axis deviation in LBBB suggests probably dilated cardiomyopathy associated with biventricular enlargement.The Framingham heart study has shown that patients with acquired BBB were more likely to have, or to develop, advanced cardiovascular manifestations-especially the male population with LBBB. Also, sudden death as the first manifestation of heart disease was 10 times higher in male individuals with LBBB than in those without the condition [4,7].Mortality risk in pre-existent LBBB without overt cardiac disease is 1.3, whereas a newly acquired LBBB confers a risk of 10.0[11] .

OBJECTIVES

1. To find out the aetiology, clinical features and left ventricular functions on 2D ECHO in patients with LBBB.

2. To examine the correlation between QRS duration and left ventricular ejection fraction in patients with LBBB.

REVIEW OF LITERATURE

Anatomy of the cardiac conduction system

The human heart has a pacemaker that is capable of mounting an electrical impulse and an electrical circuit that allows the propagation of cardiac impulse in an orderly sequence from atria to ventricles. The pacemaker is the sinus node and the intraventricular conduction system is the electrical circuit.

The sinoatrial node (SAN), the atrioventricular node (AVN), the bundle of HIS, bundle branches right and left, the fascicles and the Purkinje fibers form the conduction system [22-25]. The conduction system is formed by specialized myocytes and the electrical fibers. The SAN (subepicardial) and the AVN (subendocardial) and its atrial components, are in contact with the atrial myocardium [22-23]. While there is no demonstrable morphologically distinct pathway between the SAN and AVN, functional pathways could be responsible for the conduction between the two structures along certain preferential routes due to geometric arrangement of working muscle fibers [23, 24]. The His bundle traverses through the right fibrous trigone (central fibrous body) and before it divides into the right and left ventricular bundle branches, runs at the junction of the membranous and muscular septum [25]. RBB is a cord like structure, 1 mm diameter and it reaches the anterior papillary muscle by traversing along the septal and moderator bands. Whereas the LBBB is a broad sheet of conduction fibers that splits into three indistinct fascicles along the left side of interventricular septum [26].

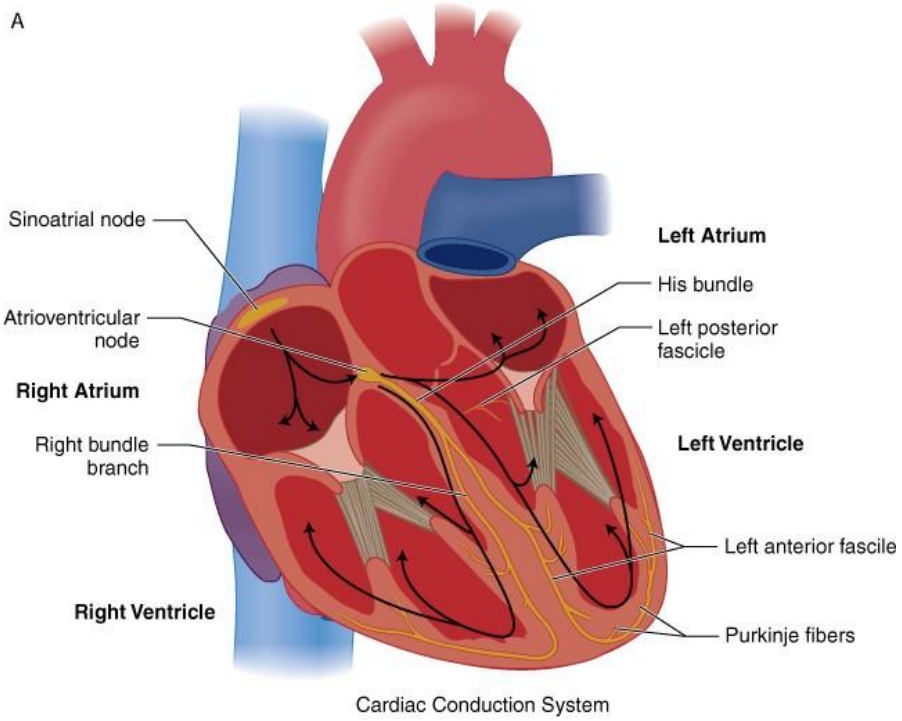


FIGURE 1: CARDIAC CONDUCTION SYSTEM: SOURCE TINTINALI JE, STAPCZYNSKI JS, MA OJ, CLINE DM, CYDULKA RK, MECKLER GD : TINTINALLI'S EMERGENCY MEDICINE : A COMPREHENSIVE STUDY GUIDE, 7TH EDITION

The components of the cardiac conduction system: The various components of the CCS are composed of a distinct set of cardiomyocytes that generate and propagate the electrical impulse required for contraction of the cardiac chambers. The sinoatrial node, which is located at the junction of the superior caval vein and right atrium, generates the impulse that then travels to the atrioventricular node, which delays the signal. The atrioventricular bundle forms the only myocardial connection between atria and ventricles through the non-myocardial atrioventricular junction. Propagation through the left and right bundle branches and the peripheral ventricular conduction system leads to activation of contraction of the ventricles. LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.

Sinus node [27-30]: The sinus node is the pacemaker of the heart. It is located at the junction of the right atrium with superior vena cava at atrial appendage, principally on antero-lateral portion of auriculo-caval junction. The cranial part starts at epicardium and the caudal portion is situated subendocardially. The SAN is made up of specialized cardiac muscle cells, the pacemaker cells that are found in **a**dense matrix of elastic and collagenous fibers which increase which increase with age in quantity [31].Automaticity is the hallmark of these cells and they discharge spontaneously. The various parts of the conduction system as well as components of myocardium are also competent of discharging spontaneously. However, normally the SAN discharges rapidly, with impulse spreading from it to various regions before other parts discharge spontaneously. The SAN therefore is regarded as the heart's pacemaker. It is supplied by the sinus node artery. The right coronary artery gives off SN artery in about **60** percent of subjects and the left circumflex artery in the rest 40% cases [32-33].

Internodal tracts: Three internodal tracts with Purkinje-type fibers connect the SAN to AVN: the anterior, middle and posterior tracts. The significance of these tracts is unknown.

The AV node: The AV node is the only pathway through which the sinus impulse can pass to reach the ventricles. It is smaller than the SAN and is situated at the floor of the right atrium, anterior to the entrance of coronary sinus to the lower right atrium and just above the insertion of the septal leaflet of the tricuspid valve. The AVN is made up of three portions with discrete properties: the upper, lower, and middle regions. The upper *atrionodal (AN)* region connects atrium to the middle portion, *nodal (N)* region. The lower *nodo-His (NH)* region is connected to bundle of His. The AV conduction delay occurs mainly in the middle region. As compared to

upper and lower portion, middle portion has no automatic properties. AV nodal artery which is a branch of the right coronary artery in 90% of patients, supplies the AVN. In the rest 10% population, it arises from the left circumflex coronary artery [34].

The Bundle of His-Purkinje system: The His bundle is a continuation of AVN, It's a short structure that branches into the left and the right bundle branches. The right bundle branch is long and thin cord like structure which is a straight continuation of the bundle of His and continues down towards the right side of the interventricular septum and then towards the apex of right ventricle and finally to the anterior papillary muscle's base. The left bundle branch divides into number of radicles almost immediately after leaving the His bundle. These radicles then continue in two major radiations that form the two dominant divisions or fascicule of the left bundle branch: the *anterosuperior* and *posteroinferior fascicles*. The anterosuperior division ramifies anteriorly and superiorly through the sub endocardium of the left lateral wall. The posteroinferior division ramifies posteriorly and inferiorly through the sub endocardium of the diaphragmatic left ventricular wall. A septal fascicle, in addition to the two, with variable origin and morphology has been described. This fascicle is responsible for the initial left to right activation of septum and the septal vector. The RBB and its fascicles terminate in Purkinje fibers network that spreads just under the endocardial surface of both ventricles. Both right and left coronary artery supply blood to the His bundle, principally by posterior descending coronary artery and branches of septal perforating arteries of the left anterior descending ***co***ronary artery [35, 36].

The Purkinje fibers form the distal part of conduction system [37]. They are very large fibers. They rapidly transmit impulse due to very high permeability of gap junctions between successive cells. The ends of Purkinje fibers penetrate about one third of way into muscle mass and become continuous with cardiac muscle fibers.

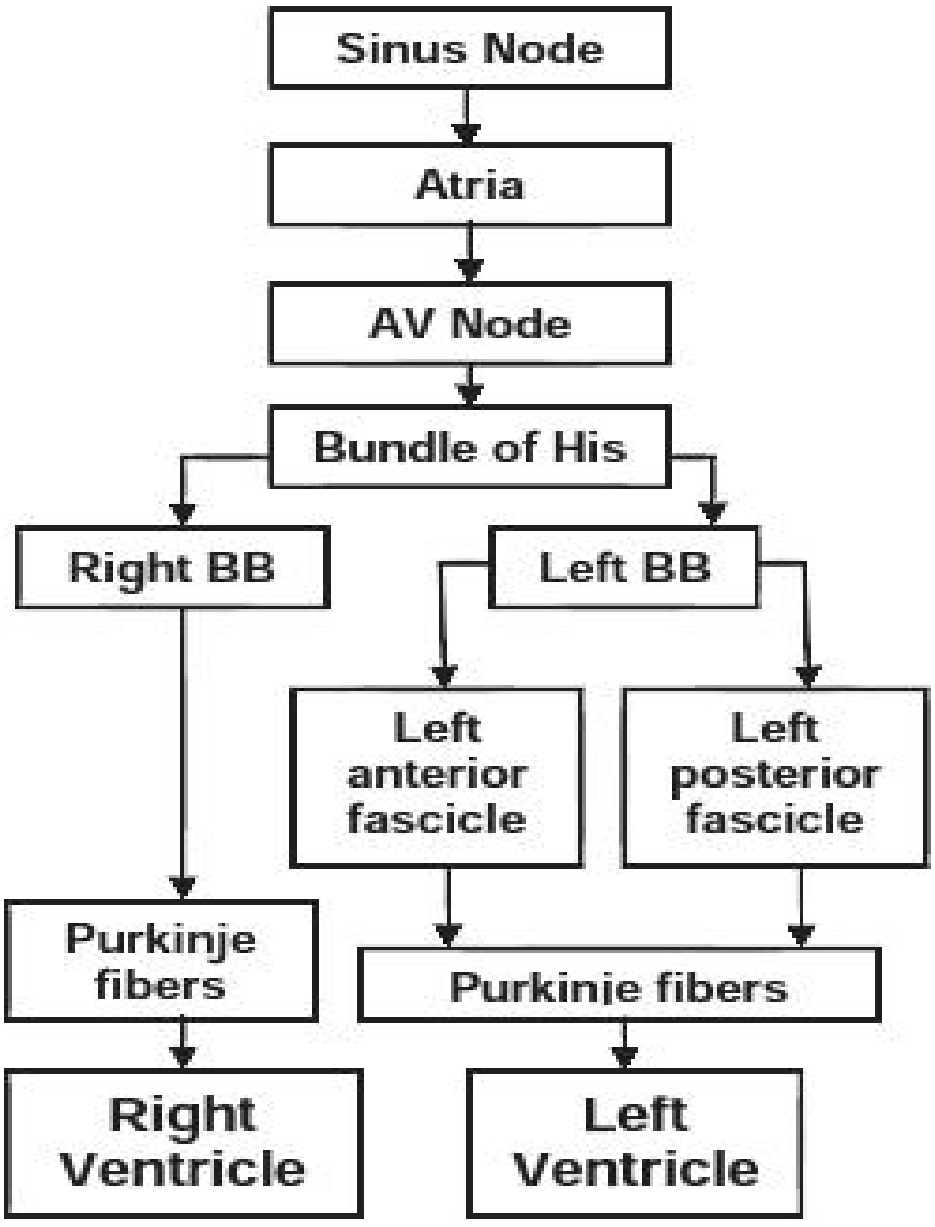


FIGURE 2: FLOWCHART SHOWING SPREAD OF ELECTRICAL ACTIVITY FROM SA NODE TO VENTRICLES.

BLOOD SUPPLY OF CARDIAC CONDUCTION SYSTEM

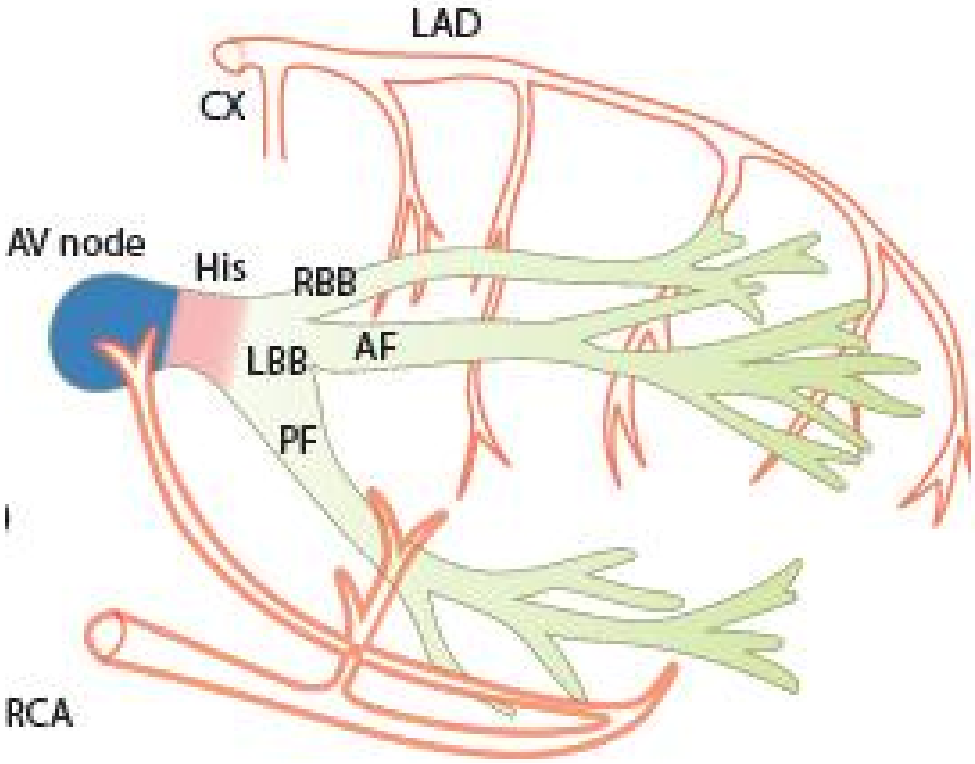


FIGURE 3: THIS FIGURE SHOWS THE BLOOD SUPPLY OF CARDIAC CONDUCTION SYSTEM. SA NODE BY RCA IN 60% CASES, LEFT CIRCUMFLES IN OTHERS.; AV NODE BY RCA IN 90% CASES; HIS BUNDLE BY BOTH RCA AND LAD; RBB BY LAD; LBB BY BOTH LAD AND RCA.

SPREAD OF CARDIAC IMPULSE

Electrical conduction system of the heart was fully accounted for by the discovery of the SA node by Keith and Flack in 1907 [38]. SA node depolarization spreads readily through the atria, through the three inter-nodal pathways, depolarizing it, and then converges on the AV node. Characteristics of the cells, which conduct the cardiac impulse from the SA node to the AV node have not been conclusively defined [39]. Atrial depolarization process is over in about 0.1 seconds. AV nodal delay of about 0.1 occurs because of slow conduction in AVN, before excitation spreads to the ventricles [40]. This delay is shortened by stimulation of the sympathetic nerves and lengthened by stimulation of the vagus and various drugs such as beta blockers, digitalis etc. The impulse then passes through the bundle of His and its branches to reach the purkinje fibers. As the impulse spreads from purkinje fibers directly to the myocardium, it results into synchronous depolarization of the ventricles. It takes 0.08–0.1 seconds for the wave of depolarization to spread from septum to all parts of the ventricles through purkinje fibers. Ventricular activation occurs in three stages- ventricular septum depolarization, depolarization of the free walls of both ventricles, depolarization of the postero-basal wall of the left ventricle and the postero-basal septum.

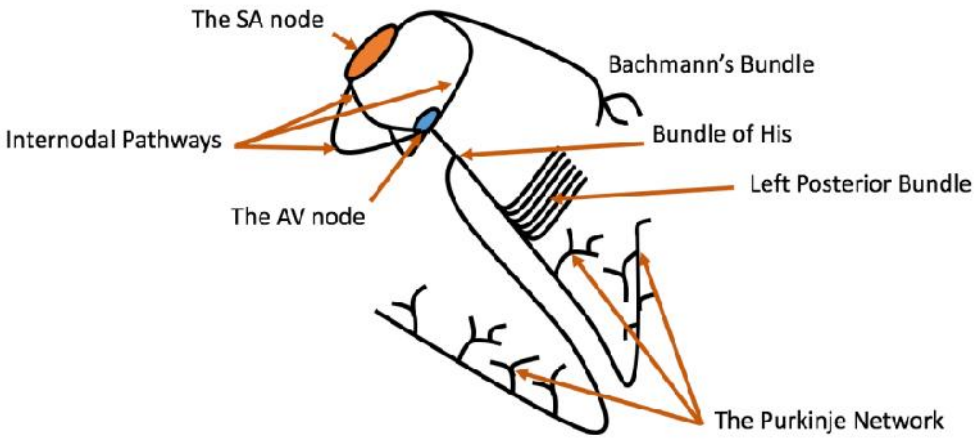


FIGURE 4 : THE CARDIAC CONDUCTION SYSTEM

Tissue	Conduction Rate (m/s)
SA node	0.05
Atrial pathways	1
AV node	0.05
Bundle of His	1
Purkinje system	4
Ventricular muscle	1

CONDUCTION VELOCITIES IN CARDIAC TISSUES.

Intraventricular conduction defects

Conduction disturbances through the intraventricular conduction pathways can occur at all the levels from the His bundle to the myocardium. These conduction disturbances may affect a single fascicle only causing unifascicular block or it may

affect two or more fascicles simultaneously resulting in bifascicular and trifascicular blocks.

A Unifascicular block consists of isolated RBBB, LAFB, and LPFB. Complete LBBB and RBBB with either LAFB or LPFB are bifascicular blocks. RBBB with either LAFB or LPFB and AV block are trifascicular blocks.

Since the activation of intraventricular conduction pathways is not recorded by standard Electrocardiography, any conduction abnormality can be detected only indirectly through its effect on QRS complex.

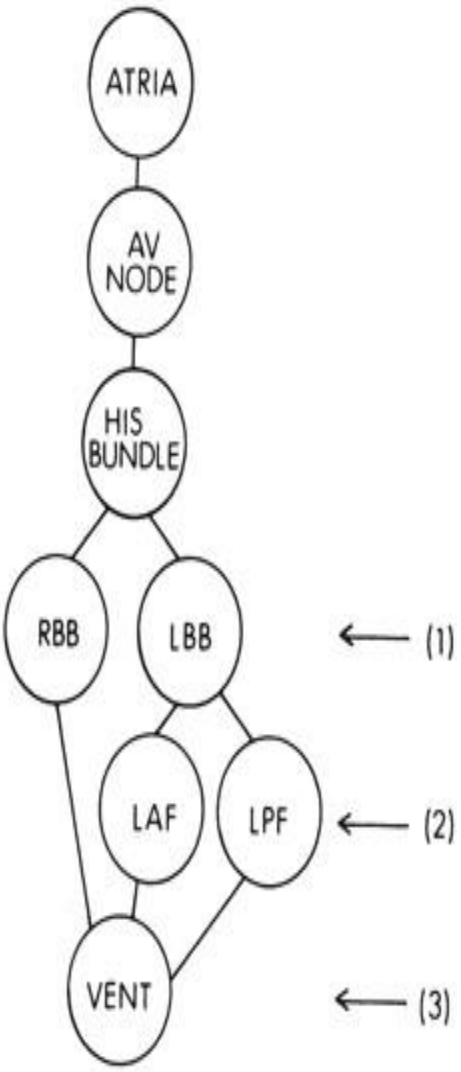


FIGURE 5: POSSIBLE LOCATIONS OF INTRAVENTRICULAR CONDUCTION ABNORMALITIES CAUSING QRS COMPLEX AND T WAVE ALTERATIONS ARE INDICATED BY NUMBERS 1, 2 AND 3. AV, ATRIOVENTRICULAR, LAF, LEFT ANTERIOR FASCICLE; LBBB, LEFT BUNDLE BRANCH BLOCK; LPF, LEFT POSTERIOR FASCICLE; RBB, RIGHT BUNDLE BRANCH BLOCK; VENT, VENTRICLE. (MODIFIED FROM WAGNER GS, WAUGH RA, RAMO BW, CARDIAC ARRHYTHMIAS, NEWYORK; NY: CHURCHILL LIVINGSTONE; 1983:18)

Left Bundle Branch Block

Left bundle branch block was first described around 100 years back on electrocardiogram [41]. Over the years the understanding of the disease has evolved manifold and its management poses a challenge to the clinicians.

LEFT BUNDLE BRANCH BLOCK occurs due to interruption of normal electrical conduction through the LBB of the his-purkinje system, which results in drastic changes in the normal sequence of the left ventricle activation [1, 2]. Here, there is a block in conduction of impulse either within the main trunk of the left bundle branch or both of its fascicles. Activation of left ventricle is through the right bundle branch by muscle to muscle conduction resulting in sequential activation of ventricles rather than concurrent activation.

AETIOLOGY AND EPIDEMIOLOGY

LBBB is rare in individuals below 35 years of age, suggesting it is an acquired condition [42]. Prevalence of LBBB strongly correlates with age with average being 70 years in men and 60 years in women [43]. Prevalence of LBBB increases progressively from less than 0.4% at age of 50 to 6% at the age of 80 years [8]. In asymptomatic adults the prevalence ranges from 0.1 to 0.8% [44, 45].

In several studies, diseases associated with LBBB have been found to be Hypertension, coronary artery disease, valvular heart disease, cardiomyopathy, myocarditis as well as electrographic abnormalities such as left ventricular hypertrophy [43, 46]. In others no risk factors or diseases are associated with LBBB.

Over the last decade, LBBB as a complication of TAVR has become apparent. The incidence varies between 7 to 83% depending on the device used [46, 47]. The mechanism is due to close proximity of atrio-ventricular nodes and the left bundle branch conduction system to the aortic valve and the mechanical interaction between the implanted mechanical valve with this conduction system [46].

Currently available evidence also suggests genetic origin for LBBB. Variations in connexin 40 (expressed in atria, proximal conduction system) and connexin 43 (expressed in Purkinje cells and cardiomyocytes) are associated with cardiomyopathy and can cause LBBB [48].

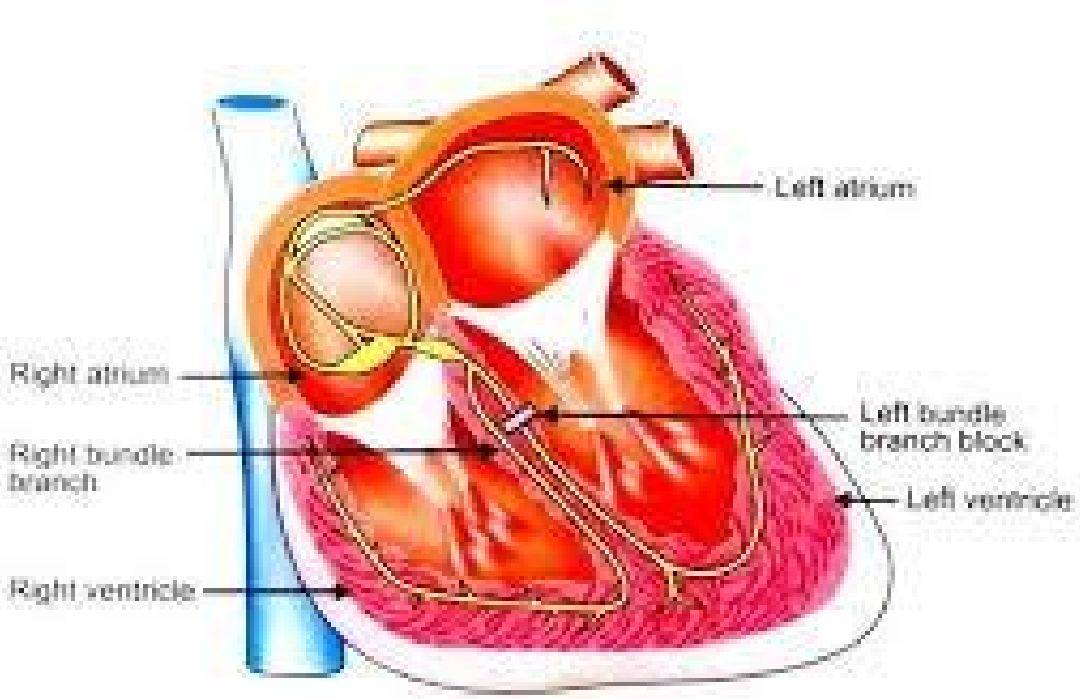


FIGURE 6 : FIGURE SHOWING BLOCK AT LEFT BUNDLE BRANCH TRUNK

Mechanism [49, 50]

Changes in QRS complex: With complete LBBB, depolarization of free wall of the left ventricle and the left side of the interventricular septum is delayed and anomalous in character. Ventricular activation begins in the right side of the interventricular septum and spreads from right to left through the septum. This results in a small right to left vector- vector 1a which is the normal septal vector. However, this is not opposed by a concomitant greater left to right force of the left septal vector. This unopposed vector theoretically manifests as

1. A small initial positive deflection in the leads oriented to the left side of septum.
2. A small negative deflection in leads oriented to the septum's right side. The first component is very small and may not be seen unless sensitive recording apparatus is used.

Following right septal activation, the activation process crosses an intra-septal physiological barrier and activates the interventricular septum's left side. Result is large magnitude vectors that are directed to the left and posteriorly- vector 2. This is reflected by:

1. A tall R wave in left oriented leads
2. A deep S wave in right oriented leads.

Septal activation is followed by delayed and anomalous activation of the free wall of the left ventricle. This results in a large magnitude vector that is directed to the left and posteriorly as well as superiorly- vector 3. This is reflected by:

1. A tall R' deflection in left oriented leads and
2. A deep S wave in right oriented leads.

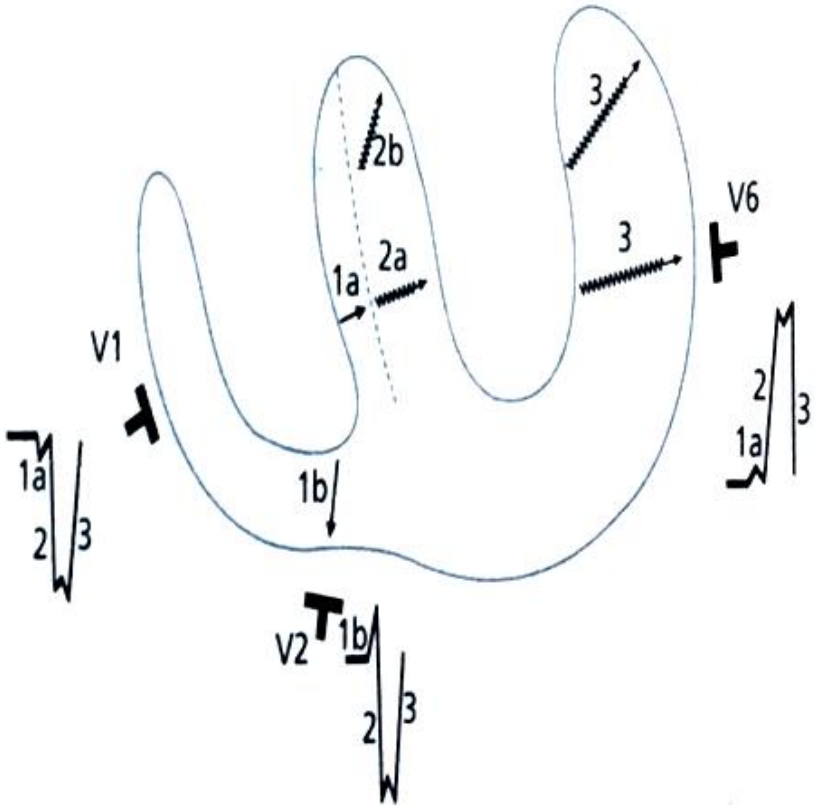


FIGURE 7: FIGURE SHOWING CHANGES IN QRS COMPLEX WITH SEPTAL ACTIVATION

Changes in the ST segment and T waves: In uncomplicated LBBB, the T waves and the ST segment are normally discordant and opposite in direction to the terminal portion of QRS complexes. This reflects the secondary repolarization changes due to abnormal activation of the ventricles. In lead V6, the T waves are inverted with isoelectric or minimally depressed ST segment. Similarly, in lead V1, the T waves are asymmetrical or inverted. The associated ST segment is concave upwards and, at times, minimally depressed. Any deviation from this, i.e. concordant ST segment and T waves, usually represents a primary change and is due to the presence of an intrinsic myocardial disease.

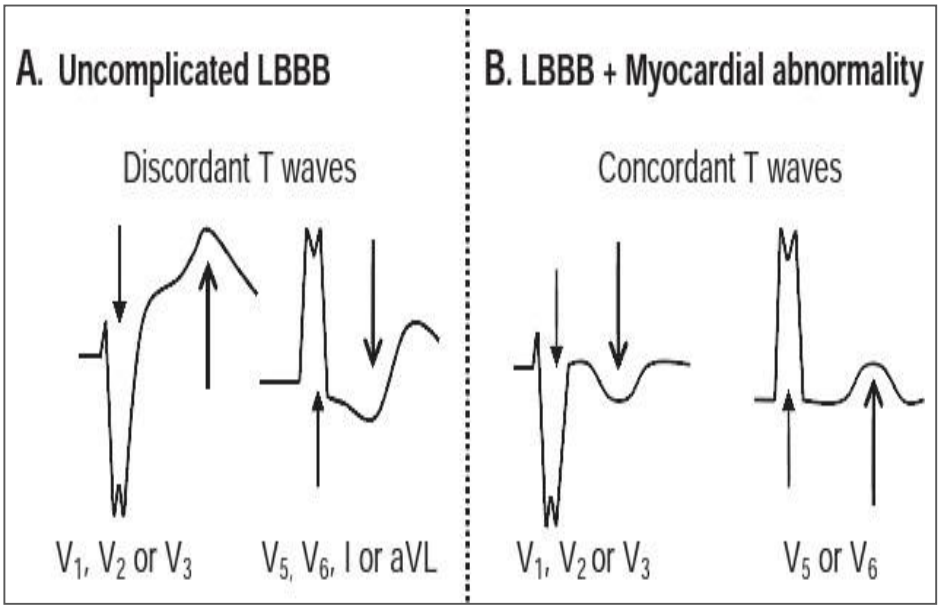


FIGURE 8: FIGURE COMPARING CONCORDANT T WAVES VS DISCORDANT WAVES IN LBBB.

ECG manifestations of Complete LBBB

The ECG changes of LBBB are best seen in the precordial leads V1 and V6. The duration of QRS complexes increase and measures more than 120 milliseconds due to delayed and anomalous activation of left ventricle. It is associated with peculiar morphology of QRS complex in lead V1 such as rS or QS pattern and in leads V6 and I, a monophasic R wave. There might be a delayed intrisicoid deflection of more than 60ms, in lead aVR, QS pattern and discordant ST/T waves [18, 19]. The axis of QRS complex is variable. When LBBB is associated with Right axis deviation, it may be associated with diffuse myocardial disease and biventricular enlargement. When left axis deviation is present, the conduction abnormality is more widespread and often involves the distal fascicles and purkinje system [51].

Leads oriented to the left ventricle reflect the following:

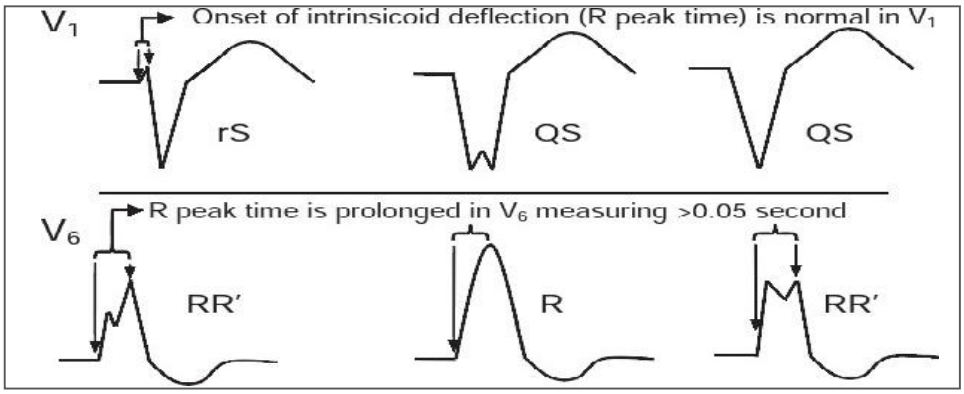


FIGURE 9: QRS COMPLEX IN LEAD V1 AND V6 IN LBBB

1. There is a relatively tall R wave, which is mainly due to depolarization of left side of the septum.

2. There is a terminal R' wave, which is due to late and anomalous depolarization of left free wall.

3. The onset of intrinsicoid deflection in leadV6 is delayed to 50ms or more.

Leads oriented to the right ventricle reflect the following:

1. There is a small initial r wave due to depolarization of the right Para septal region.

2. This is followed by deep, wide and notched S wave, which is mainly due to depolarization of left side of septum and left free wall.

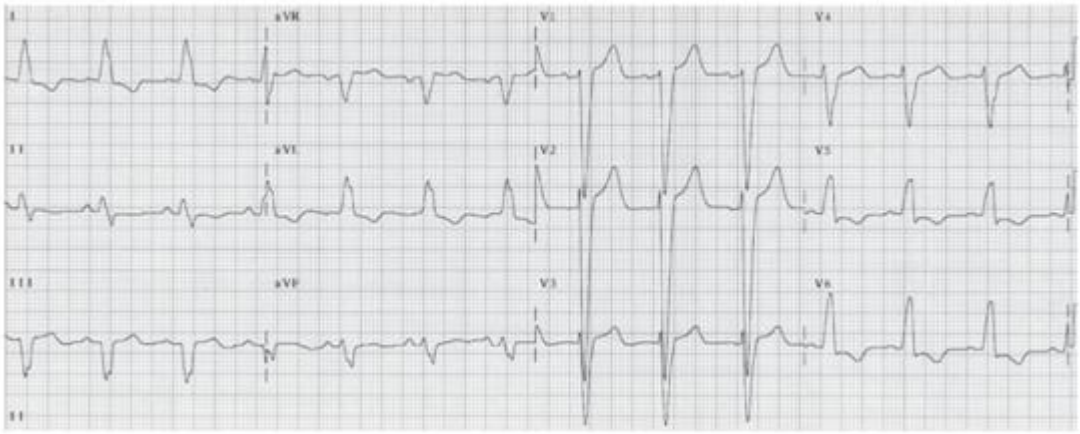


FIGURE 10: 12 LEAD ECG DEPICTING LBBB

Criteria		de Luna	AHA/ACC/HRS	Strauss
QRS duration (m/f, ms)		≥120/≥120	≥120/≥120	≥140/≥130
QRS notching or slurring		-	Broad notched/slurred R wave in leads I, aVL, V5, and V6	Mid-QRS notching/ slurring in ≥ 2 of leads V1, V2, V5, V6, I, and aVL
QS or rS in leads V1 and V2		+	-	+
Delayed intrinsicoid deflection (>60 ms)		Present in leads V6 and I	Present in leads V5 and V6 absent in leads V1, V2, and V3	-
Usually discordant ST and T wave		+	+[a]	-
QS pattern with a positive T wave in aVR		+	-	-
Q waves in leads I, V5, and V6		-	Absent	May be present in patients with concomitant anterior and/or apical infarct
ECG pattern	V1, V2			
	V5, V6			

[+]Criteria included as a criterion.
[-]Not mentioned criteria.
[a]Positive T wave in leads with upright QRS may be normal (positive concordance).

FIGURE 11: DIFFERENT CRITERIA FOR DIAGNOSIS OF LBBB

Incomplete LBBB

Here, conduction through the left bundle branch and its ramifications is still possible but is delayed. The type of electrocardiographic manifestations that occurs with incomplete LBBB depends on the degree of delay within the left bundle branch. A small delay within the left bundle branch causes a delay in the formation and inscription of left sided septal vector. This means the right sided septal vector now has more time to develop and as a result, it equals the magnitude of left sided septal vector. The two septal vectors now cancel or nullify each other, thereby resulting in the disappearance of the resultant normal left to right septal vector. A slight delay in conduction through the left bundle branch will thus cause a disappearance of q waves in leads V5 and V6. There should also be a disappearance of initial r wave in leadv1 [52].

With increasing progression of the incomplete LBBB, a slur appears on the upstroke of QRS complex. This slur is due to the increasing dominance of right septal vector that penetrates the intra-septal barrier to a varying degree. It is allowed to play a more dominant role due to increasing delay within the left bundle branch until the fully developed manifestations of complete LBBB develops.

ECG manifestations of incomplete LBBB:

The following sequence of progressively increasing changes manifests in electrocardiography:

1. There is disappearance of septal q waves in lead V6 resulting in a single tall R wave with disappearance of r wave in lead V1 resulting in QS complex. This is the earliest sign of incomplete LBBB.

2. The disappearance of septal q waves is followed by slurring of the upstroke of the R wave in lead V6.

3. The initial slur becomes increasingly more prominent, the QRS complex becomes progressively wider and the QRS complex eventually develops a notch.

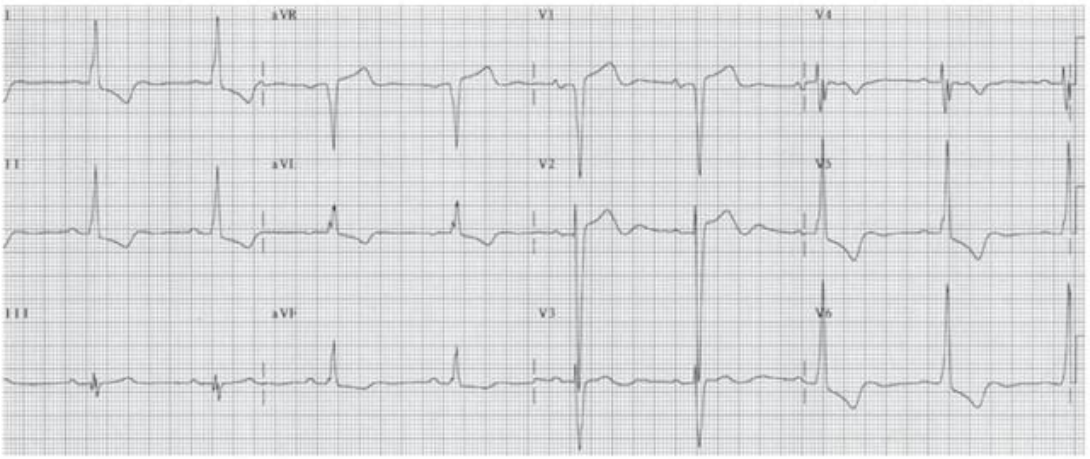


FIGURE 12: 12 LEAD ECG DEPICTING INCOMPLETE LBBB

Intermittent LBBB

LBBB often occurs intermittently and rate related before it becomes fixed. It maybe bradycardia dependent or tachycardia related.

Bradycardia dependent bundle branch block: LBBB occurs only when there is slowing of the heart rate. It is due to phase 4 diastolic depolarization, which is inherently present in cells with properties of automaticity, including cells within the intraventricular conduction system. When there is a long R-R interval as that occurs in bradycardia, cells with automatic properties undergo spontaneous phase 4 diastolic depolarization giving rise to a transmembrane potential turning the inside of the cell

to become less and less negative [53]. Thus, the sinus impulse may not be able to pass through a bundle branch that is partially depolarized.

Tachycardia-related bundle branch block: The refractory period of one bundle branch is usually longer than the refractory period of the other due to its longer action potential duration when compared with the other bundle branch. When the heart rate is normal, both bundle branches are given enough time to repolarize. But when the heart rate is faster, the impulse may arrive well before the branch with a longer refractory period has a chance to recover. This results in bundle branch block that is evident only during tachycardia and resolves when the heart rate slows down to baseline. This type of rate related bundle branch block is called phase 3 aberration.

Clinical significance of LBBB

Etiology: LBBB is an acquired conduction disorder and it is usually a marker of an underlying cardiac abnormality. The majority of patients with LBBB have cardiac disease. LBBB can also occur as an isolated finding in normal, asymptomatic individuals without evidence of cardiac disease but is very rare. If cardiac disease is not apparent, overt cardiac abnormality will subsequently develop. The common causes of LBBB include hypertension, coronary artery disease, cardiomyopathy, valvular heart diseases especially aortic stenosis, acute myocarditis, and degenerative disease of the conduction system. LBBB is the most common conduction abnormality in patients with primary cardiomyopathy.

Hemodynamic abnormalities: The irregular activation pattern of ventricles in LBBB induces further changes in the hemodynamics which get superimposed on the underlying cardiac disease abnormalities. The pattern of ventricular activation with

LBBB is less coordinated and requires much more time due to the sequential activation of ventricles. The result is asynchronous and prolonged left ventricular contraction that results in regional differences in workload; regional changes in blood flow and metabolism; structural remodeling; as well as functional mitral valve dysfunction due to the altered geometry of the apparatus of mitral valve from the changes in contraction patterns causing mitral regurgitation. As a result, cardiac efficiency is further reduced. Severe left ventricular dysfunction is common, with a delay between lateral wall and septal wall contraction of more than 60 milliseconds with QRS durations of 120 to 150 milliseconds.

Auscultatory findings: The presence of LBBB will delay the closure of mitral and aortic valves. Therefore, the first heart sound becomes single and the second heart sound tends to be paradoxically split. With split of the 2^{nd} heart sound being paradoxical, it becomes narrowly split with inspiration and widely split with expiration. A short murmur in systole of MR might be audible during early systole because of asynchrony in contraction of the papillary muscles.

LBBB with LVH: LVH is difficult to diagnose when LBBB is present because of the associated tall voltage and secondary ST-T changes. Nevertheless, approximately 85% of patients with LBBB have left ventricular hypertrophy.

LBBB with Acute MI [53]*:* Acute MI is difficult to diagnose when there is LBBB because ST-T abnormalities and q waves associated with acute MI can be obscured due to the presence of LBBB. Conversely, when LBBB is present, “Q waves or QS complex” may occur in anterior chest leads which may mimic a myocardial infarct. The ST-T changes with LBBB can also be mistaken for current of injury. In the

presence of LBBB, the following findings suggest the possibilities of myocardial infarction:

1. Concordant ST segment elevation of more than 1 mm in a patient with symptoms of myocardial ischemia.

2. Concordant ST segment depression of more than 1 mm in a patient with symptoms of myocardial ischemia.

3. Discordant ST segment elevation of more than 5 mm accompanied by symptoms of ischemia.

4. Cabrera sign, which is notching of the upstroke of the S wave in V3 or V4 and Chapman sign, notching of the upstroke of the R wave in V5 or V6, are highly specific for MI but not very sensitive.

LBBB and ECG stress testing: LBBB may mask the ECG changes of myocardial ischemia and results in a false negative stress test. Conversely, LBBB may also result in a false positive stress test since it can cause secondary ST-T changes in the ECG, which mimics ischemia. Thus, the ACC/AHA guidelines on chronic stable angina do not recommend stress testing using ECG alone as an indicator for ischemia in population with LBBB. Stress testing of patients with LBBB on baseline ECG should always include an imaging modality in addition, preferably a nuclear perfusion scan.

Stress testing with imaging: Nuclear perfusion scan uses perfusion mismatch for detecting myocardial ischemia, whereas Echocardiogram uses wall motion abnormality as the end point for detecting the presence of myocardial ischemia. Nuclear perfusion scan is preferred over echocardiography as the imaging modality

during stress testing, since left ventricular wall motion abnormalities inherently occur when there is LBBB [54]. Exercise can further augment the abnormalities of wall motion in LBBB even if there is no ischemia. Hence Pharmacologic stress testing is preferred over exercise in the presence of LBBB. Dipyridamole or adenosine, but not dobutamine are preferred because both agents do not alter contractility.

Prognosis: LBBB has significant prognostic implications. It is associated with a higher risk of morbidity and mortality from MI, HF, and arrhythmias including high-grade AV block. The risk is increased in all the patients with or without overt heart disease. Majority of the patients usually have antecedent hypertension, coronary artery disease or cardiomegaly at the time of diagnosis of LBBB. Even in isolated LBBB, they will eventually go on to develop one of these cardiovascular abnormalities which translate into a higher mortality. LBBB is also significantly related to an increase in sudden cardiac death.

Among patients with CVD, including acute STEMI, the occurrence of LBBB denotes, widespread disease, and a critical fall in LV ejection fraction and increased mortality rates. In people suffering from heart failure, increased QRS duration has been shown to be associated with poor prognosis.

LBBB with right or left axis deviation is a marker for critical illness. Axial deviation to the left denotes grave conduction system disease involving fascicles and main left bundle. Axis deviation to the right indicates dilated cardiomyopathy with biventricular enlargement.

Treatment: Overall treatment depends on the underlying cardiac disease process. In completely asymptomatic patients without overt cardiac disease, no treatment is

required except periodic screening [7]. LBBB, which is a bifascicular block, can progress to trifascicular block or 3rd degree AV block. In such patients, insertion of a permanent pacemaker is warranted.

Patients with decreased LV ejection fraction and LBBB who continue to have features of heart failure despite receiving optimal medical management may benefit from cardiac resynchronization therapy [46]. Cardiac resynchronization therapy involves insertion of a biventricular pacemaker that can stimulate both the ventricles simultaneously. This significantly decreases the delay in the spread of electrical impulse and therefore improves cardiac output and diminishes mitral regurgitation. Patients who are candidates for CRT should have all of the following features:

1. Wide QRS complexes measuring > 0.12seconds
2. Normal sinus rhythm
3. Systolic dysfunction with ejection fraction <35%
4. NYHA class III / IV HF
5. Features of HF despite optimal medical therapy

The width of the QRS complex is the main indication for biventricular pacing. The patient should be in normal sinus rhythm so that timing of atrial and ventricular contraction can be synchronized.

Role of Echocardiography

Echocardiography is an excellent tool to establish the possible etiology for Left bundle branch block. The findings usually reflect the underlying cardiac disease process.

Isolated LBBB: In the case of isolated LBBB without overt cardiac disease, regional wall motion abnormality may be the only finding. Changes in wall motility due to left bundle branch block exclusively are very obvious in the proximal and mid-anterior septal regions and less obvious in the anterior wall or apex. It usually does not cause a change in LV geometry.

M-mode echo is an excellent way to demonstrate the effects of the LBBB. With the start of ventricular systole, a downward beak is appreciated which is followed by simultaneous movement of the septum anteriorly and thickening of myocardium [55]. Because of conduction delay in LBBB, there is dyssynchrony in commencement of motion in non- involved wall.

Acute Myocardial Infarction: RWMA is the hallmark of an acute MI. The amount of myocardium involved is directly related to degree of wall motion abnormality. It is not necessary that the entire myocardium should be ischemic to result in a regional wall motion abnormality [12]. Ischemia involving 25% of the wall thickness itself can result in dyskinesias of the entire wall.

Ischemia in the LAD territory often results in abnormalities of the wall mobility which can involve the apex and the anterior wall resulting in absence of thickening of myocardium in the systolic phase of ventricular septum. These are often

related with atypical geometry of LV. These can be used to differentiate ischemic from non-ischemic wall motion abnormalities.

ECHO FINDINGS	Ischemic WMA	LBBB
Maximal location	Distal septum, anterior wall and apex	Proximal, mid anterior septum
Thickening	Absent	Partially preserved
Duration	Monophasic	Multiphasic
Abnormal geometry	Common	Uncommon
Temporal dyssynchrony	No	Yes

2D ECHO FINDINGS IN ISCHEMIC WMA VS LBBB

Hypertension: Echocardiography is used to detect the end-organ cardiac damage that occurs with hypertension, including left ventricular hypertrophy, diastolic dysfunction and later on systolic dysfunction. Additionally, left atrial dilation, mitral annulus calcification, and mild degrees of aortic valve insufficiency have a relatively greater prevalence in the hypertensive population. With chronic hypertension, there may be secondary increase in diameter of aorta in the ascending limb and effacement of Sino tubular junction resulting in secondary aortic insufficiency. Atherosclerosis of the large vessels and peripheral vascular disease are also associated with long-standing hypertension.

Diastolic dysfunction is one of the earliest cardiac manifestations of hypertensive heart disease[56]. In early hypertension, diastolic dysfunction is manifested as a reduced E/A ratio of mitral valve inflow due to delayed relaxation of the myocardium as a result of hypertrophy and mild degrees of stiffening. In severe long-standing hypertension, the left ventricle develops systolic dysfunction as well. At this point, more advanced diastolic dysfunction with a normal or high E/A ratio representing pseudo normal filling or restrictive physiology can be detected. However, if left ventricular hypertrophy remains uncomplicated by concurrent systolic dysfunction, no other changes are anticipated. The combination of left ventricular hypertrophy with moderate dilatation and global dysfunction and significant diastolic dysfunction is fairly typical of end stage hypertensive cardiovascular disease.

Dilated cardiomyopathy: 2D-ECHO is a investigation of choice for establishing the diagnosis and severity of DCMP. It establishes the etiology of DCMP in some cases. Dilatation of left ventricles and systolic dysfunction are the important features of DCMP on 2D-ECHO. DDF, secondary PAH, right ventricular dysfunction and secondary regurgitation of mitral and tricuspid valves are some of the secondary features seen in DCMP.

Some secondary findings such as left atrial dilation and right heart involvement are nearly present in all cases and are essential in establishing the diagnosis. Others such as secondary pulmonary hypertension, secondary mitral regurgitation and thrombus formation occur to a variable degree depending on both the severity and duration of cardiomyopathy.

Left atrial dilation denotes more severe and chronic ventricular dysfunction. It is largely due to elevated diastolic pressures in the left ventricle and concurrent mitral regurgitation. It can also be due to a myopathic process in the atrial wall. Left atrial area or volume can be measured from the apical view. It can dilate to substantial dimensions and dimension of more than 6 cm occasionally encountered. All these result in an increased risk of developing atrial fibrillation or flutter. Left atrial spontaneous contrast is also not uncommonly encountered. Occasionally auto contrast may be seen in the left ventricle as well. There is also a strong independent relationship between left atrial area or volume and prognosis in patients with cardiomyopathy. Formation of mural thrombus may occur in patients with dilated cardiomyopathy but is less frequent than in MI. Tricuspid regurgitation is frequently noted in advanced cardiomyopathy because of either concurrent involvement of the right ventricle or secondary pulmonary hypertension.

The etiology of dilated cardiomyopathy cannot be determined quite often by echocardiography alone. However, a clinically relevant distinction can be made between ischemic and non-ischemic etiologies.

Distinguishing features of an ischemic cardiomyopathy include a relatively greater degree of regional variation of systolic function often with areas of frank scar conforming to a well-defined coronary territory or aneurysm formation. Stress echocardiography generally with dobutamine has shown promise for identifying ischemic cardiomyopathy.

METHODOLOGY

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on patients with LBBB during the period of January 2018 to December 2018.

Study design

One year cross sectional study.

Study period

The present study was conducted during January 2018 to December 2018.

METHOD OF COLLECTION OF DATA

SOURCE OF DATA

Patients diagnosed to have LBBB on ECG at KLES Dr. Prabhakar Kore Hospital and MRC Belgaum were selected for the study.

Sample size

A sample size of 100 cases was with LBBB were selected for the study.

Sampling procedure

Sample size is calculated using the formula 4*pq/d where p denotes the prevalence of disease, q=1-p, and d denotes the error range. A sample size of minimum 60 cases with LBBB was calculated using the above formula. A sample size of 100 patients was selected using purposive sampling, with all consecutive patients

with LBBB visiting OPD, GENERAL WARD and ICU of general medicine department.

Selection criteria

Inclusion Criteria

- Patient age more than 18 years.
- Patient diagnosed to have LBBB ON ECG

Exclusion Criteria

- All patients with incomplete LBBB.

Procedure

Patients attending to the out patients department, admitted to the ward and ICU of GENERAL MEDICINE at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum diagnosed to have LBBB ON ECG were evaluated and selected by detailed medical history and physical examination. The study was approved by the Ethical and Research Committee of J. N. Medical College, Belgaum.

After finding the suitability as per inclusion and exclusion criteria they were selected for the study and briefed about the nature of the study, the interventions used and written informed consent was obtained (Annexure–I). Further, descriptive data of the participants like name, age, sex, detailed history, were obtained by interviewing the participants and clinical examination and necessary investigations were recorded on predesigned and pretested pro forma (Annexure-II).

Detailed relevant history regarding duration of symptoms, history of co morbidities and treatment history is taken. Detailed clinical examination, including pulse rate, blood pressure, relevant general physical examination and systemic examination will be done.

STANDARD RESTING SUPINE 12 LEAD ECG IS OBTAINED

LBBB diagnosed on basis of following criteria [18, 19]:

- QRS duration of > 120 ms
- Dominant S wave in V1
- Broad monophasic R wave in lateral leads (I, aVL, V5-V6)
- Absence of Q waves in lateral leads (I, V5-V6; small Q waves are still allowed in aVL)
- Prolonged R wave peak time > 60ms in left precordial leads (V5-6).
- Appropriate discordance: the ST segments and T waves always go in the opposite direction to the main vector of the QRS complex
- Poor R wave progression in the chest leads
- Left axis deviation.

Patients diagnosed with LBBB on ECG will be subjected to 2D ECHO screening.

2-D ECHOCARDIOGRAPHY Studies will be performed in the patient in supine position with echocardiography instrument equipped with 2.5-MHz transducer. Two-dimensional images will be obtained in the standard parasternal and apical views. Chamber dimensions will be noted from recordings in M-mode, taken in parasternal long-axis view and measured according to American Society of Echocardiography [20]. Endocardial echoes of the interventricular septum (IVS) on left side and LV

posterior wall (PW) will be noted in all patients and then LV end diastolic (EDD) and end systolic dimensions (ESD) and IVS thickness will be measured. LV end-diastolic and end-systolic volumes were calculated using modified Simpson's rule technique and the ejection fraction of LV calculated [21]. Following parameters will be noted:

- Asynchronous movement of left ventricle in view of LBBB.
- regional wall motion abnormality
- ejection fraction
- valvular lesions
- left ventricular end diastolic and systolic diameter
- estimated pulmonary artery pressure
- diastolic dysfunction

Other tests

- Complete Blood Count
- Renal function tests
- RBS
- HbA1C if random blood sugar more than 200mg/dl or known diabetic.

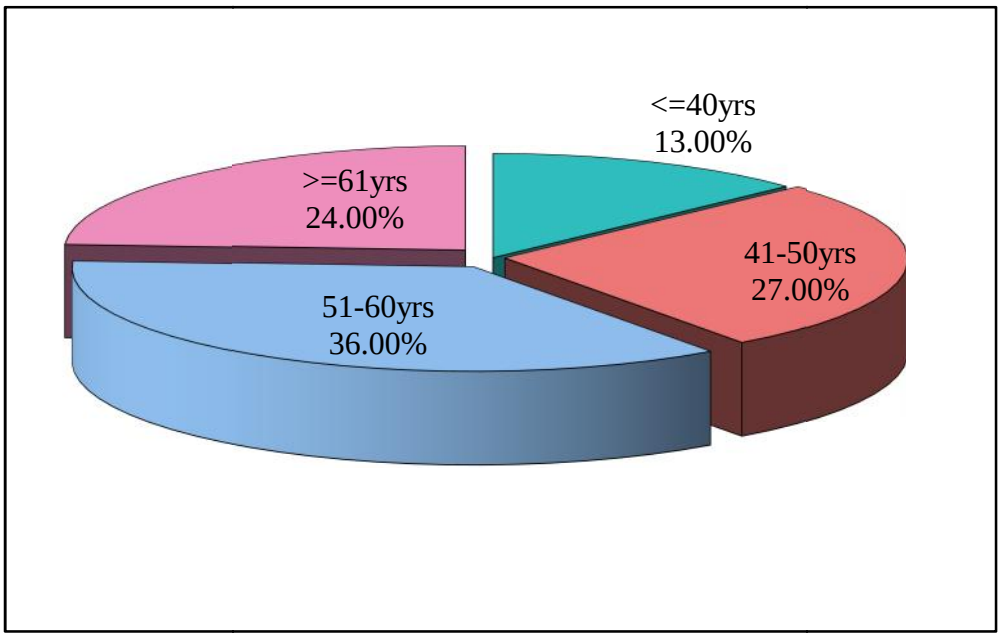
If patient has undergone or undergoes coronary angiography, the report of it will be considered in final analysis.

RESULTS

TABLE 1: AGE WISE DISTRIBUTION

Age groups	No of LBBB patients	%of LBBB patients
<=40yrs	13	13.00
41-50yrs	27	27.00
51-60yrs	36	36.00
>=61yrs	24	24.00
Total	100	100.00
Mean	63.12	
SD	11.80	

GRAPH 1: AGE WISE DISTRIBUTION



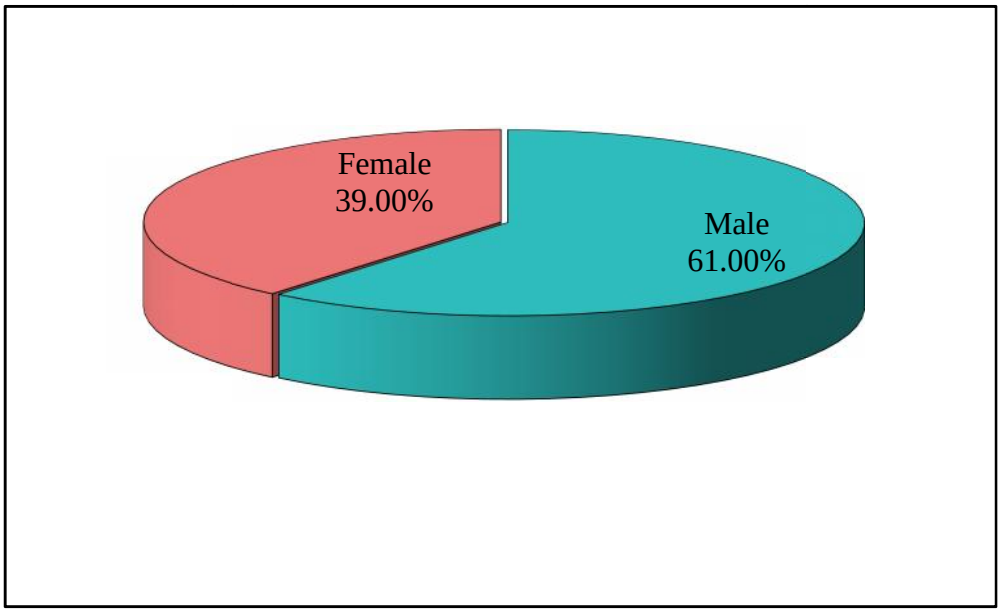
In the present study, among the study population of 100 subjects the age ranges from 18 years to 86 years with the mean age being 63.12 years with a standard deviation of 11.80.

13% of the studied population age was less than 40 years, 27% fell into bracket of age 41 to 50 years, 36% in the age bracket 51 to 60 years and 24% above the age of 60 years.

TABLE 2: GENDER WISE DISTRIBUTION

Gender	No of LBBB patients	%of LBBB patients
Male	61	61.00
Female	39	39.00
Total	100	100.00

GRAPH 2: GENDER WISE DISTRIBUTION

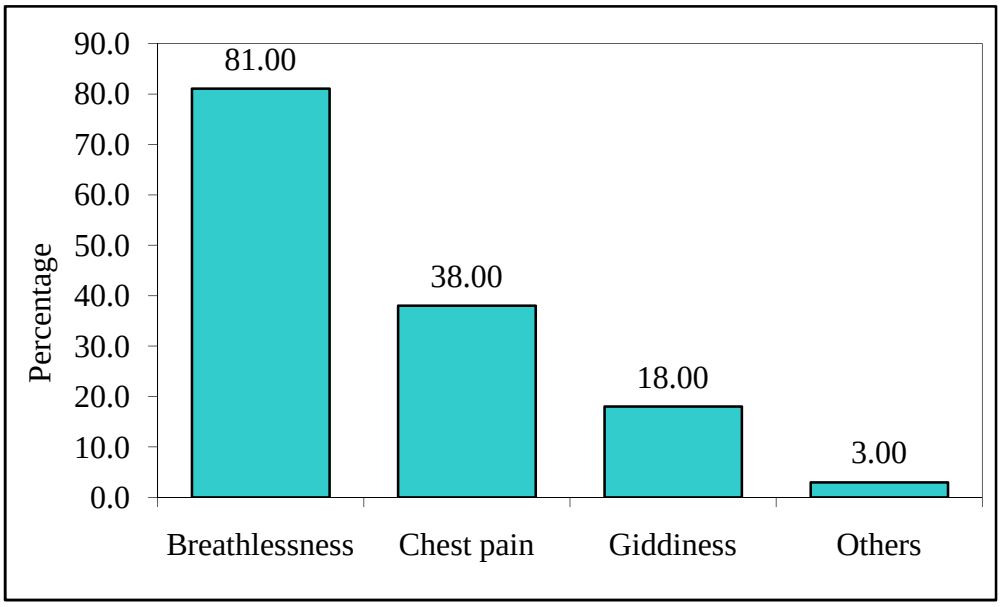


In the present study, among the study population of 100 subjects 61 out of 100(61%) are male and 39 out of 100(39%) are female.

TABLE 3: DISTRIBUTION OF CHIEF COMPLAINTS

Chief complaints	No of LBBB patients	%of LBBB patients
Breathlessness	81	81.00
Chest pain	38	38.00
Giddiness	18	18.00
Others	3	3.00

GRAPH 3: DISTRIBUTION OF CHIEF COMPLAINTS

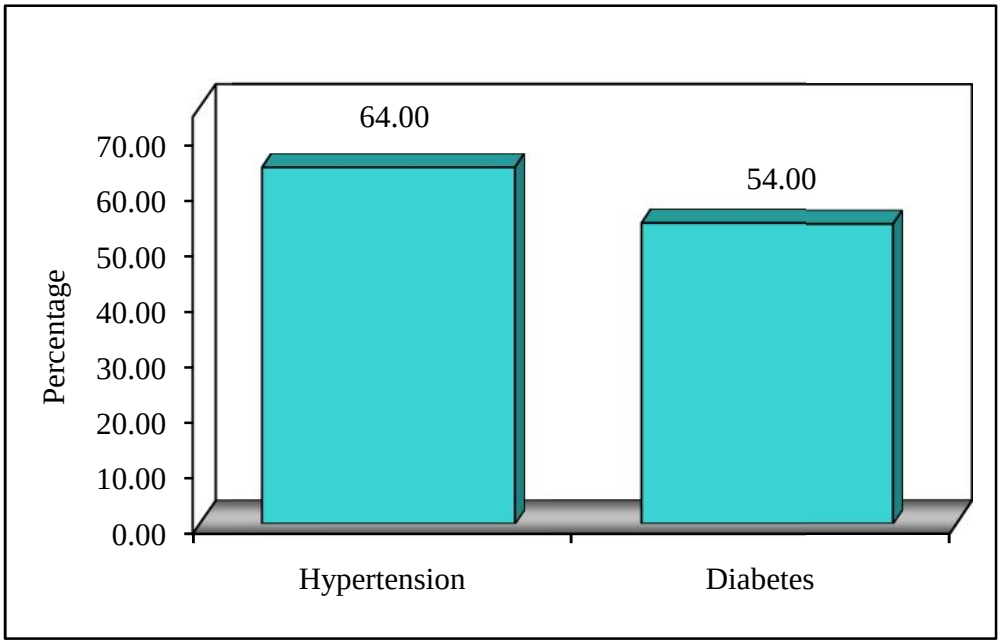


In the study population, among the study population of 100 subjects the main presenting complaint is breathlessness in 81% patients followed by chest pain in 38% subjects and giddiness in 18% subjects. 3 subjects presented with non cardiac complaints and among them 1 was asymptomatic.

TABLE 4: HYPERTENSION AND DIABETES MELLITUS IN LBBB PATIENTS

Co morbidities	No of LBBB patients	%of LBBB patients
Hypertension	64	64.00
Diabetes	54	54.00

GRAPH 4: HYPERTENSION AND DIABETES MELLITUS IN LBBB PATIENTS

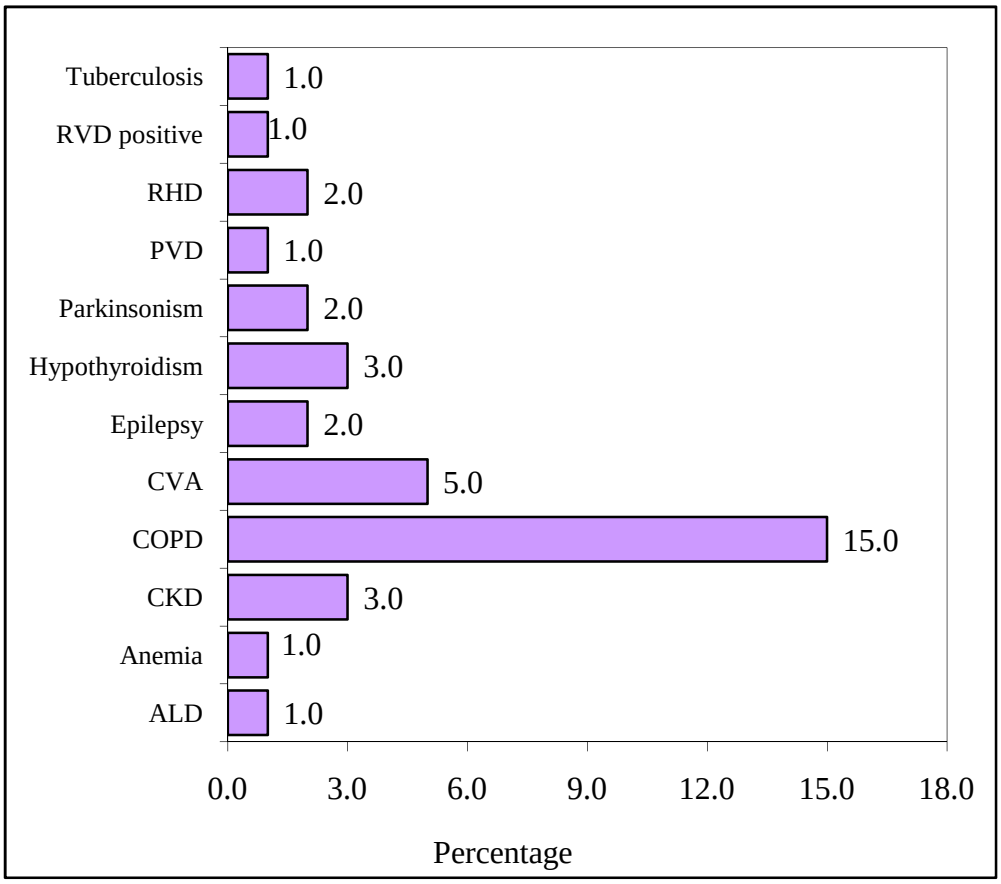


In the study population, among the 100 subjects evaluated, hypertension is the most common co morbidity found in 64 out of 100(64%) subjects followed by diabetes in 54 out of 100(54%) subjects.

TABLE 5: OTHER COMORBIDITIES

Others Co morbidities	No of LBBB patients	%of LBBB patients
ALD	1	1.00
Anemia	1	1.00
CKD	3	3.00
COPD	15	15.00
CVA	5	5.00
Epilepsy	2	2.00
Hypothyroidism	3	3.00
Parkinsonism	2	2.00
PVD	1	1.00
RHD	2	2.00
RVD positive	1	1.00
Tuberculosis	1	1.00

GRAPH 5: OTHER COMORBIDITIES

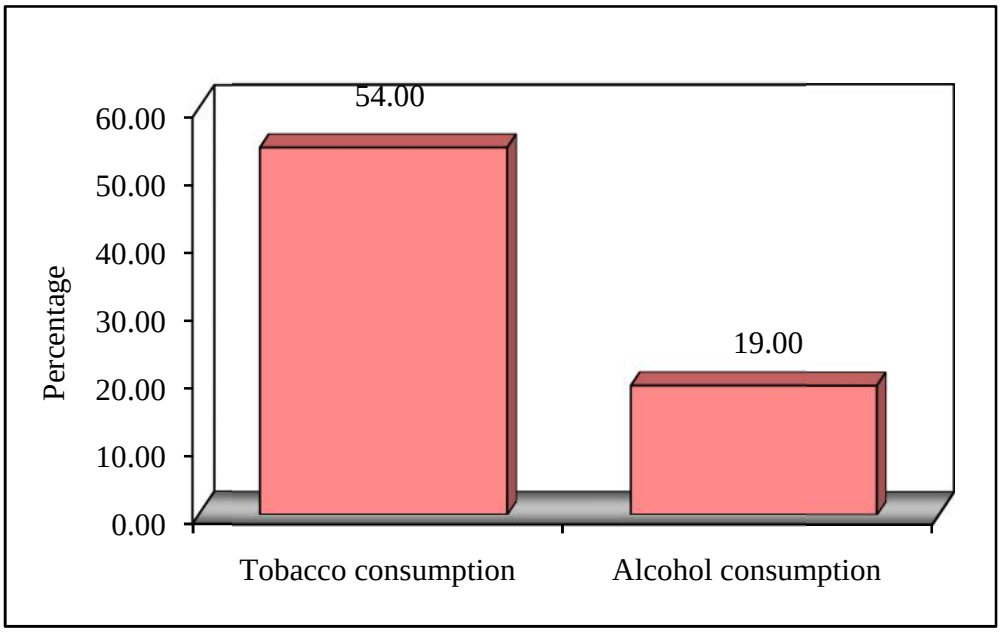


In the study population, among the subjects studied chronic obstructive pulmonary disease as co morbidity is present in 15 subjects (15%), cerebro-vascular accident in 5 subjects (5%) and few have other co morbidities as listed in the Table 5.

TABLE 6: HABITS

Habits	No of LBBB patients	%of LBBB patients
Tobacco consumption	54	54.00
Alcohol consumption	19	19.00

GRAPH 6: HABITS

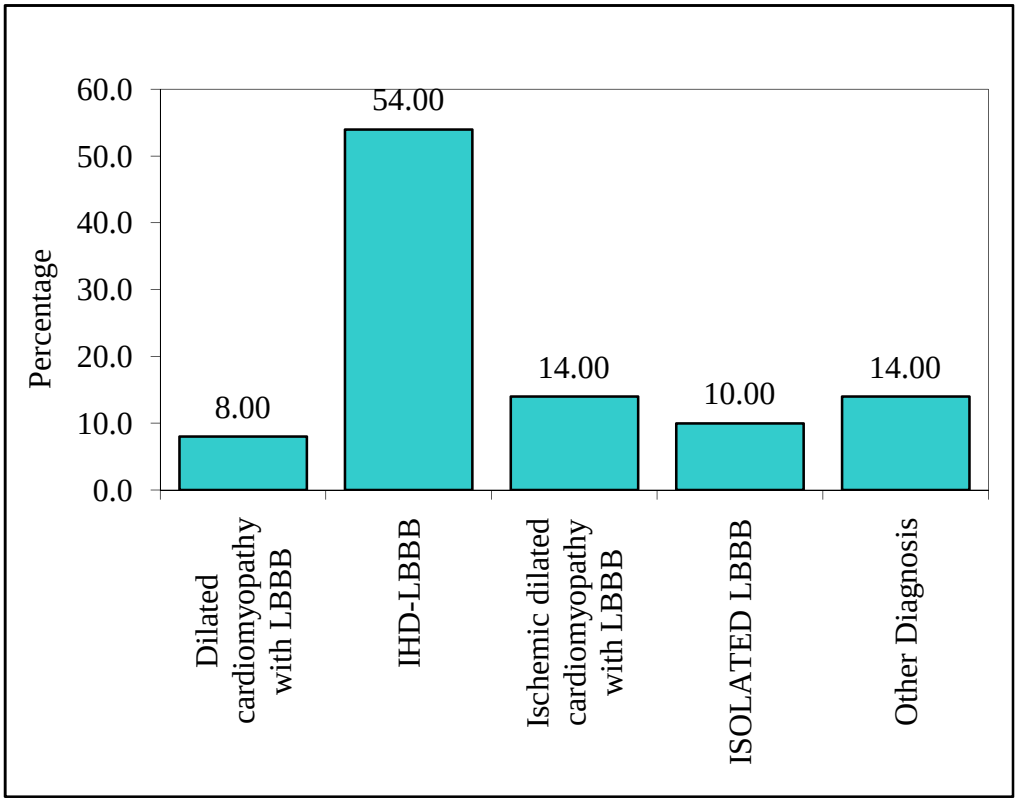


In the present study, among the studied population of 100 subjects 54 out of 100(54%) have tobacco consumption in the form of smoking or gutka chewing as habit and 19 out of 100(19%) have alcohol consumption as habit.

TABLE 7: AETIOLOGY OF LBBB

Diagnosis	No of LBBB patients	%of LBBB patients
Dilated cardiomyopathy with LBBB	8	8.00
IHD- LBBB	54	54.00
Ischemic dilated cardiomyopathy with LBBB	14	14.00
ISOLATED LBBB	10	10.00
Other Diagnosis	14	14.00
Total	100	100.00

GRAPH 7: AETIOLOGY OF LBBB



In the present study among 100 subjects' studied the most common etiology for LBBB is found to be ischemic heart disease in 54 subjects out of 100(54%). This includes patients diagnosed with IHD with new onset LBBB, IHD- unstable angina with LBBB and old cases of IHD with LBBB.

Second most common etiology is ischemic dilated cardiomyopathy in 14 patients out of 100(14%).

Isolated LBBB is found in 10 patients out of 100(10%), followed by dilated cardiomyopathy with LBBB in 8 patients out of 100(8%).

14 patients with LBBB have other diagnosis as listed below in Table 8.

TABLE 8: EXPANDED LIST OF AETIOLOGY OF LBBB

Primary diagnosis	No of LBBB patients	%of LBBB patients
Coarctation of Aorta with aortic arch aneurysm with LBBB	1	1.00
Dilated cardiomyopathy with LBBB	8	8.00
IHD-LBBB	54	54.00
Mitral valve prolapse with LBBB	2	2.00
LBBB, Aortic stenosis	1	1.00
LBBB with 2:1 AV block	1	1.00
Ischemic dilated cardiomyopathy with LBBB	14	14.00
ISOLATED LBBB	10	10.00
LBBB s/p AVR with stuck valve	1	1.00
LBBB with atrial fibrillation status post avr with stuck valve	1	1.00
LBBB with LV dysfunction	2	2.00
IHD- IWMI with LBBB	4	4.00
Secondary PAH- LBBB with AF	1	1.00
Total	100	100.00

TABLE 9: CLINICAL PARAMETERS

Variables	Diagnosis	N	Min	Max	Mean	SD	SE	F-value	P-value
Pulse	Dilated cardiomyopathy with LBBB	8	82.00	130.00	107.00	13.77	4.87	2.4770	0.0490*
	IHD-LBBB	54	60.00	130.00	87.70	16.39	2.23		
	Ischemic dilated cardiomyopathy with LBBB	14	54.00	114.00	88.14	17.05	4.56		
	Isolated LBBB	10	50.00	130.00	81.40	22.90	7.24		
	Other Diagnosis	14	40.00	128.00	89.57	23.74	6.35		
	Total	100	40.00	130.00	88.94	18.72	1.87		
SBP	Dilated cardiomyopathy with LBBB	8	100.00	170.00	122.50	23.75	8.40	4.1730	0.0040*
	IHD-LBBB	54	90.00	220.00	127.89	22.80	3.10		
	Ischemic dilated cardiomyopathy with LBBB	14	80.00	160.00	104.57	20.46	5.47		
	Isolated LBBB	10	110.00	170.00	141.00	20.79	6.57		
	Other Diagnosis	14	90.00	200.00	122.14	28.33	7.57		
	Total	100	80.00	220.00	124.70	24.68	2.47		
DBP	Dilated cardiomyopathy with LBBB	8	60.00	100.00	77.50	14.88	5.26	4.3080	0.0030*
	IHD-LBBB	54	50.00	110.00	77.59	11.48	1.56		
	Ischemic dilated cardiomyopathy with LBBB	14	50.00	100.00	65.00	14.01	3.74		
	Isolated LBBB	10	70.00	100.00	85.00	9.72	3.07		
	Other Diagnosis	14	50.00	110.00	77.14	14.37	3.84		
	Total	100	50.00	110.00	76.50	13.21	1.32		

*$p<0.05$ indicates significant correlation

In the present study, among the study population of 100 subjects, pulse rate ranges from minimum of 40/minute to 130/minute with mean value of 88.94/minute with standard deviation of 18.72.

Systolic blood pressure ranges from 80 mm of HG to 220 mm of HG with mean value of 124.70 mm of HG with standard deviation of 24.68.

Diastolic blood pressure ranges from 50 mm of HG to 110 mm of HG with mean value of 76.50 mm of HG with standard deviation of 13.21.

Ranges of pulse rate, SBP and DBP in individual diagnosis are listed in Table 9.

From the results of the above Table 9, it can be seen that, a significant difference is observed between diagnosis with respect to pulse (f=2.4770, p=0.0490), SBP (f=4.1730, p=0.0040) and DBP (f=4.3080, p=0.0030) at 5% level of significance. It implies that, the patients belonging to different diagnosis have different clinical parameters.

TABLE 10: BIOCHEMICAL PARAMETERS

Variables	Diagnosis	N	Min	Max	Mean	SD	SE	F-value	P-value
HB	DCM with LBBB	8	8.50	13.60	11.73	1.69	0.60	1.1450	0.3400
	IHD-LBBB	54	8.90	15.50	11.88	1.60	0.22		
	Ischemic DCM with LBBB	14	10.00	15.30	12.24	1.52	0.41		
	Isolated LBBB	10	7.40	14.50	11.86	2.27	0.72		
	Other Diagnosis	14	10.10	15.80	12.91	2.04	0.55		
	Total	100	7.40	15.80	12.06	1.74	0.17		
Creatinine	DCM with LBBB	8	0.78	1.72	1.10	0.31	0.11	1.8930	0.1180
	IHD-LBBB	54	0.43	3.34	1.02	0.50	0.07		
	Ischemic DCM with LBBB	14	0.70	3.15	1.46	0.76	0.20		
	Isolated LBBB	10	0.41	2.35	1.13	0.57	0.18		
	Other Diagnosis	14	0.68	2.96	1.19	0.58	0.15		
	Total	100	0.41	3.34	1.12	0.56	0.06		
RBS	DCM with LBBB	8	96.00	298.00	149.50	74.05	26.18	0.4050	0.8050
	IHD-LBBB	54	65.00	601.00	171.06	102.06	13.89		
	Ischemic DCM with LBBB	14	65.00	291.00	157.71	68.29	18.25		
	Isolated LBBB	10	77.00	295.00	173.50	69.58	22.00		
	Other Diagnosis	14	87.00	258.00	141.93	59.95	16.02		
	Total	100	65.00	601.00	163.63	87.27	8.73		
Sodium	DCM with LBBB	8	125.00	146.00	136.13	7.77	2.75	2.3370	0.0610
	IHD-LBBB	54	119.00	144.00	136.09	5.54	0.75		
	Ischemic DCM with LBBB	14	121.00	140.00	131.71	6.71	1.79		
	Isolated LBBB	10	132.00	148.00	138.50	4.90	1.55		
	Other Diagnosis	14	123.00	142.00	135.07	5.18	1.38		
	Total	100	119.00	148.00	135.58	5.96	0.60		

Potassium	DCM with LBBB	8	4.14	5.53	4.51	0.47	0.17	2.2650	0.0680
	IHD-LBBB	54	3.20	5.77	4.31	0.56	0.08		
	Ischemic DCM with LBBB	14	3.78	8.06	4.96	1.20	0.32		
	Isolated LBBB	10	3.71	5.28	4.36	0.54	0.17		
	Other Diagnosis	14	3.49	7.36	4.47	0.94	0.25		
	Total	100	3.20	8.06	4.44	0.75	0.08		
HBA1C	DCM with LBBB	4	5.20	9.10	7.08	1.60	0.80	1.0690	0.3800
	IHD-LBBB	37	5.20	14.00	8.46	2.27	0.37		
	Ischemic DCM with LBBB	10	6.00	16.80	9.34	3.23	1.02		
	Isolated LBBB	6	6.40	9.70	7.47	1.27	0.52		
	Other Diagnosis	6	5.20	10.40	7.78	2.06	0.84		
	Total	63	5.20	16.80	8.35	2.34	0.30		

Among the study population of 100 subjects hemoglobin ranges from 7.40gm/dl to 15.80 gm/dl with a mean value of 12.06 with standard deviation of 1.74

Among the renal function tests, creatinine ranges from 0.41mg/dl to 3.34 mg/dl with a mean value of 1.12mg/dl with standard deviation of 0.56. Sodium ranges from 119meq/l to 148meq/l with a mean value of 135.58meq/l with standard deviation of 5.96. Potassium ranges from 3.20meq/l to 8.06meq/l with a mean value of 4.44 meq/l with standard deviation of 0.75.

The values of random blood sugar ranges from 65mg/dl to 601mg/dl with a mean value of 163.63mg/dl with standard deviation of 87.27

Hba1c was done for 63 subjects who were known cases of diabetes mellitus or random blood sugar was more than 200mg/dl. It ranges from minimum of 5.20 to 16.80 with a mean value of 8.35 with standard deviation being 2.34

Laboratory profile for individual diagnosis for subjects with LBBB is listed in Table 10.

From the results of the above table, it can be seen that, no statistically significant difference is observed between diagnosis with respect to HB, creatinine, RBS, sodium, potassium and HBA1C at 5% level of significance.

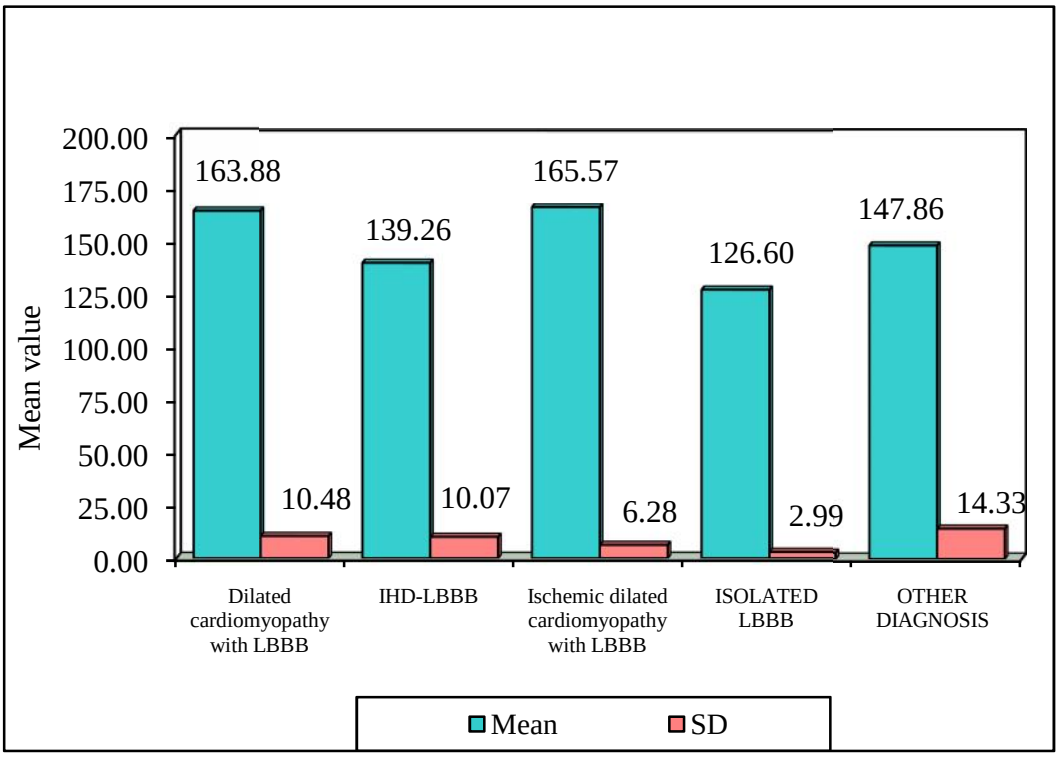
It implies that subjects with LBBB having different diagnosis have similar laboratory profile and there is no statistically significant difference in laboratory parameters such as hemoglobin, creatinine, sodium, potassium, random blood sugar and HBA1C among different diagnosis.

TABLE 11: DURATION OF QRS COMPLEX ON ECG

Diagnosis	N	Min	Max	Mean	SD	SE	F-value	P-value
Dilated cardiomyopathy with LBBB	8	152.00	178.00	163.88	10.48	3.71	35.4700	0.0001*
IHD-LBBB	54	122.00	162.00	139.26	10.07	1.37		
Ischemic dilated cardiomyopathy with LBBB	14	154.00	172.00	165.57	6.28	1.68		
ISOLATED LBBB	10	122.00	132.00	126.60	2.99	0.95		
Other Diagnosis	14	124.00	174.00	147.86	14.33	3.83		
Total	100	122.00	178.00	144.85	15.39	1.54		

*p<0.05 indicates significant correlation

GRAPH 8: DURATION OF QRS COMPLEX ON ECG



In the present study among 100 subjects analyzed with LBBB, QRS duration on ECG ranges from 122 milliseconds to 178 milliseconds with a mean value of 144.85 with standard deviation of 15.39.

Duration of QRS complex and its range in individual diagnosis is listed in Table 11.

It is observed that mean QRS duration in subjects diagnosed with dilated cardiomyopathy and ischemic dilated cardiomyopathy is significantly higher than in subjects diagnosed with IHD- LBBB and is least in subjects diagnosed with isolated LBBB.

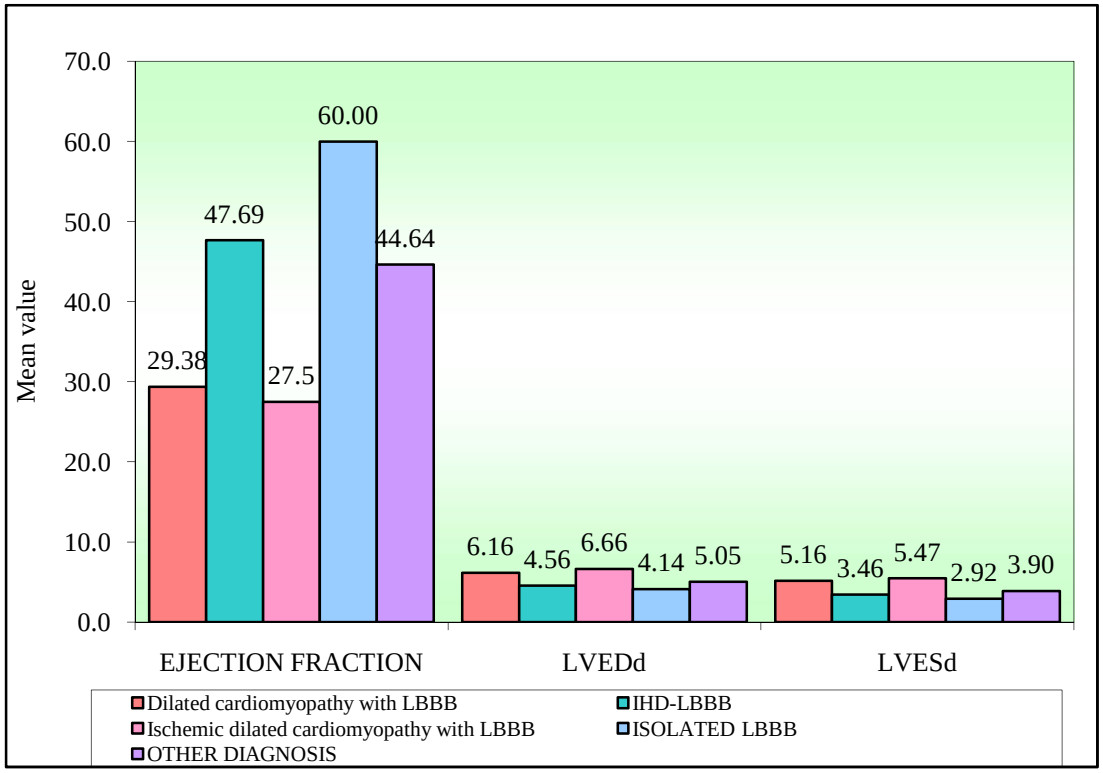
A statistically significant difference is observed between different diagnosis with ECG-QRS scores ($f=35.4700$, $p=0.0001$) at 5% level.

Table 12: EJECTION FRACTION AND DIAMETERS OF LEFT VENTRICLE ON 2D ECHO

Variables	Diagnosis	N	Min	Max	Mean	SD	SE	F-value	P-value
EF	DCM with LBBB	8	20.00	35.00	29.38	4.96	1.75	29.0130	0.0001*
	IHD-LBBB	54	30.00	60.00	47.69	9.70	1.32		
	Ischemic DCM with LBBB	14	25.00	30.00	27.50	2.59	0.69		
	ISOLATED LBBB	10	60.00	60.00	60.00	0.00	0.00		
	Other Diagnosis	14	25.00	60.00	44.64	12.32	3.29		
	Total	100	20.00	60.00	44.20	12.73	1.27		
LVEDd	DCM with LBBB	8	5.00	6.90	6.16	0.65	0.23	45.5440	0.0001*
	IHD- LBBB	54	3.80	5.90	4.56	0.49	0.07		
	Ischemic DCM with LBBB	14	6.10	7.70	6.66	0.46	0.12		
	ISOLATED LBBB	10	4.00	4.50	4.14	0.15	0.05		
	Other Diagnosis	14	3.90	7.60	5.05	1.12	0.30		
	Total	100	3.80	7.70	5.01	1.02	0.10		
LVESd	DCM with LBBB	8	4.00	5.80	5.16	0.71	0.25	41.5160	0.0001*
	IHD- LBBB	54	2.60	4.80	3.46	0.53	0.07		
	Ischemic DCM with LBBB	14	4.90	6.20	5.47	0.42	0.11		
	ISOALTED LBBB	10	2.60	3.20	2.92	0.16	0.05		
	Other Diagnosis	14	2.70	6.70	3.90	1.17	0.31		
	Total	100	2.60	6.70	3.89	1.03	0.10		

*$p<0.05$ indicates significant correlation

GRAPH 9: EJECTION FRACTION AND DIAMETERS OF LEFT VENTRICLE ON 2D ECHO



Among the 100 subjects with LBBB, ejection fraction ranges from minimum of 20% to maximum of 60% with a mean value of 44.20 % with standard of deviation 12.73. Range for ejection fraction and its mean on 2D ECHO in individual diagnosis is listed in Table 12.

LVEDd ranges from 3.80 to 7.70 cm in all subjects with LBBB with a mean value of 5.01 cm with standard deviation of 1.02.

LVESd ranges from 2.60 cm to 6.70 cm with mean value of 3.89 cm with standard deviation of 1.03.

Range and mean value of all three parameters for individual diagnosis is listed in Table 12.

From the results of the above table, it is observed that there is a statistically significant difference between diagnosis with respect to ECHO- EF (f=29.0130, p=0.0001), ECHO- LVEDd (f=45.5440, p=0.0001) and ECHO- LVESd (f=41.5160, p=0.0001) at 5% level of significance.

It implies that the patients belonging to different diagnosis have statistically different ejection fraction and left ventricle diameters.

It is found that patients diagnosed with dilated cardiomyopathy and ischemic dilated cardiomyopathy have significantly lower ejection fraction than patients with IHD-LBBB.

All patients diagnosed as isolated LBBB had 60% as ejection fraction.

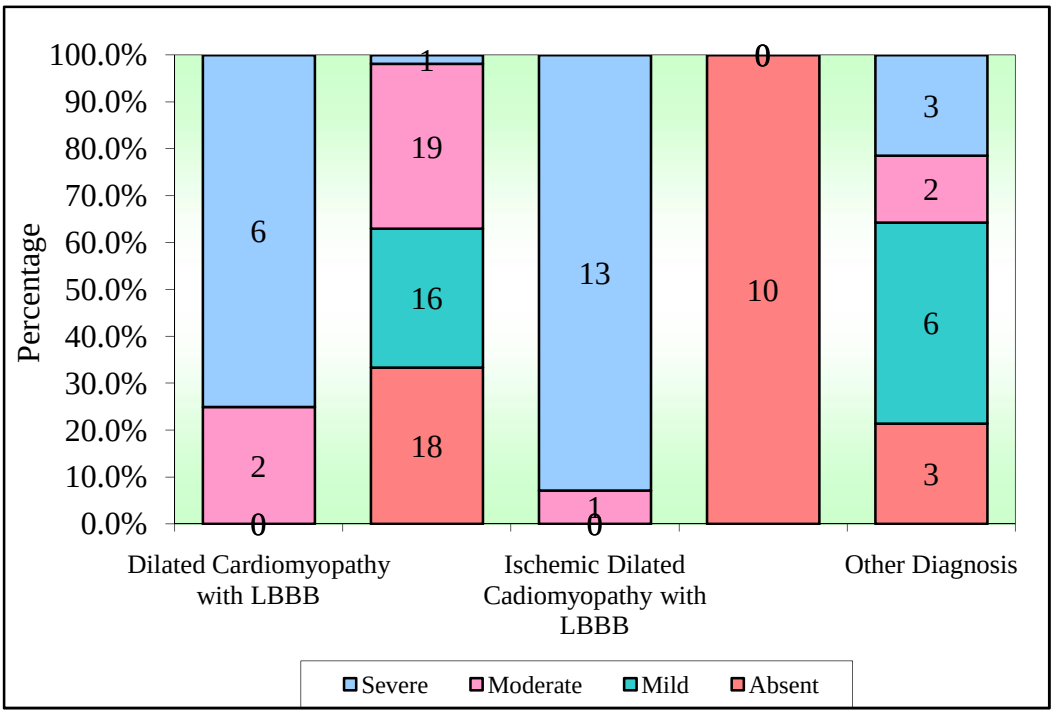
It was also found that the diameters of left ventricle are significantly increased in cases diagnosed with dilated cardiomyopathy and ischemic dilated cardiomyopathy.

TABLE 13: LEFT VENTRICULAR DYSFUNCTION

Diagnosis	Absent	Mild	Moderate	Severe	Total
Dilated cardiomyopathy with LBBB	0	0	2	6	8
IHD- LBBB	18	16	19	1	54
Ischemic dilated cardiomyopathy with LBBB	0	0	1	13	14
ISOLATED LBBB	10	0	0	0	10
Other Diagnosis	3	6	2	3	14
Total	31	22	24	23	100
Chi-square=93.2754, p=0.0001*					

*p<0.05 indicates significant correlation

GRAPH 10: LEFT VENTRICULAR DYSFUNCTION



In the present study, left ventricular dysfunction is present in 69 subjects out of 100 studied (69%) and absent in 31 subjects.

Among the 69 subjects who have LV dysfunction, 22 subjects have mild LV dysfunction, 24 have moderate LV dysfunction, and 23 have severe LV dysfunction.

LV dysfunction in individual diagnosis is as listed in Table 13.

It can be observed that patients with dilated cardiomyopathy and ischemic dilated cardiomyopathy had more severe LV dysfunction.

Patients with isolated LBBB have no LV dysfunction.

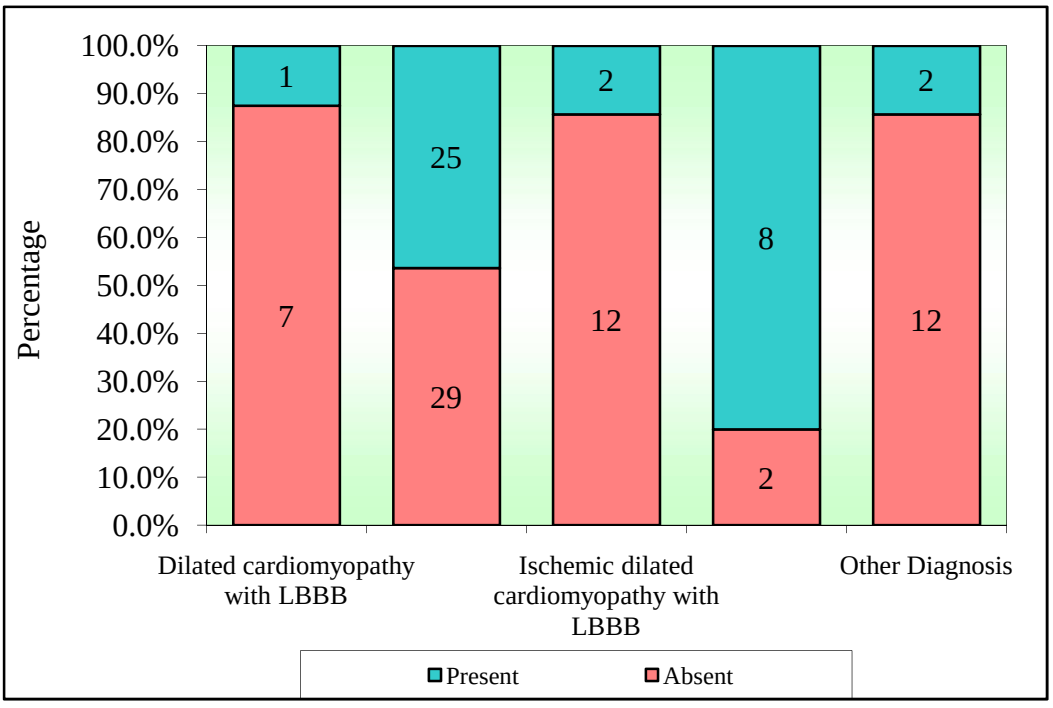
There is a significant association between LV dysfunction and LBBB in our study (chi-square=93.2754, p=0.0001).

TABLE 14: LEFT VENTRICULAR HYPERTROPHY

Diagnosis	Absent	Present	Total
Dilated cardiomyopathy with LBBB	7	1	8
IHD- LBBB	29	25	54
Ischemic dilated cardiomyopathy with LBBB	12	2	14
ISOLATED LBBB	2	8	10
Other Diagnosis	12	2	14
Total	62	38	100
Chi-square=17.9560, p=0.0010*			

*p<0.05 indicates significant correlation

GRAPH 11: LEFT VENTRICULAR HYPERTROPHY



In the present study, among the population studied of 100 subjects, concentric LVH is present in 38 subjects out of 100(38%) and absent in 62 subjects out of 100(62%).

Concentric LVH in individual diagnosis is listed in Table 14.

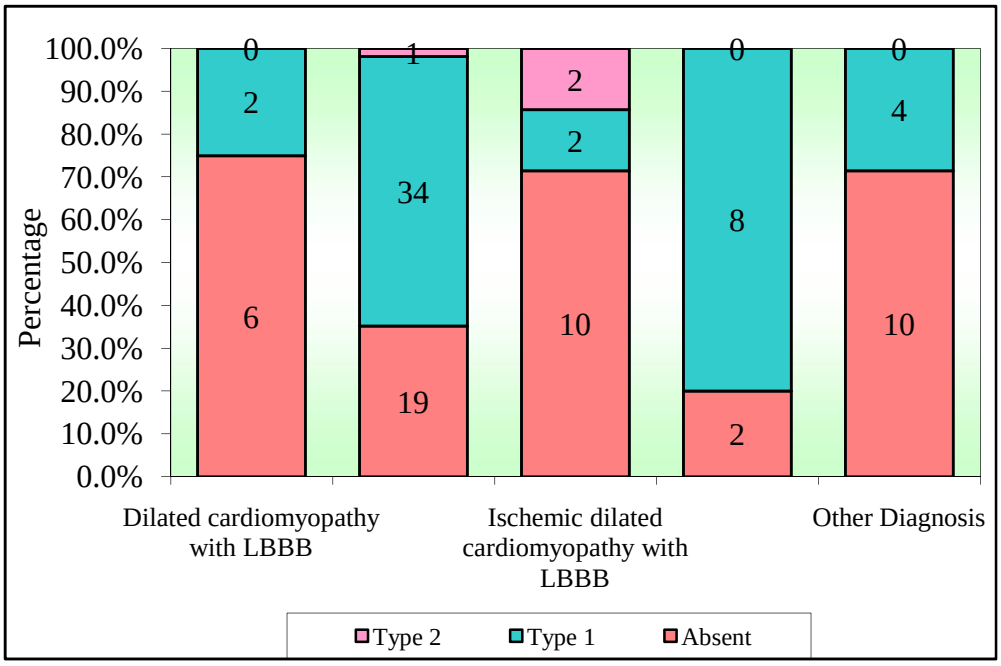
There is a statistically significant association between LVH and LBBB in our study (chi-square=17.9560, p=0.0010).

TABLE 15: DIASTOLIC DYSFUNCTION

Diagnosis	Diastolic dysfunction			
	Absent	Type 1	Type 2	Total
Dilated cardiomyopathy with LBBB	6	2	0	8
IHD-LBBB	19	34	1	54
Ischemic dilated cardiomyopathy with LBBB	10	2	2	14
ISOLATED LBBB	2	8	0	10
Other Diagnosis	10	4	0	14
Total	47	50	3	100
Chi-square=24.6575, p=0.0017*				

*p<0.05

GRAPH 12: DIASTOLIC DYSFUNCTION



In the present study, diastolic dysfunction is present in 53 subjects out 100 and absent in 47 subjects out of 100.

Among 53 subjects in whom diastolic dysfunction is observed, 50 subjects have type 1 diastolic dysfunction and 3 have type 2 diastolic dysfunction.

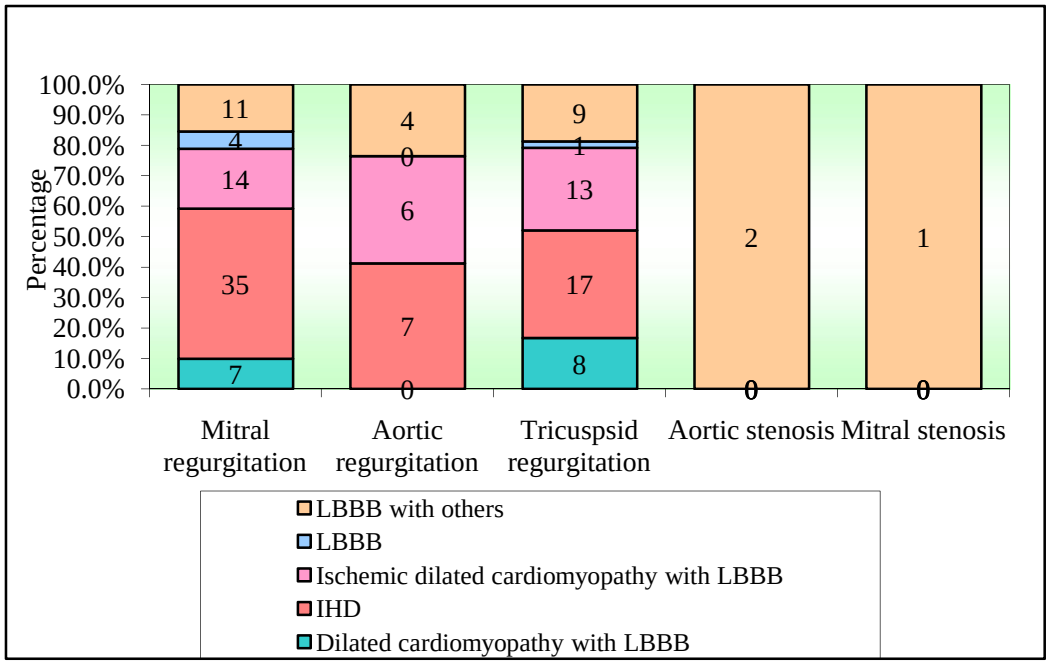
Diastolic dysfunction in individual diagnosis is as listed in table 15

There is a statistically significant association between diastolic dysfunction and diagnosis patients (chi-square=24.6575, p=0.0017).

TABLE 16: VALVULAR LESIONS ASSOCIATED WITH LBBB

Diagnosis	Mitral regurgitation	Aortic regurgitation	Tricuspid regurgitation	Aortic stenosis	Mitral stenosis
Dilated cardiomyopathy with LBBB	7	0	8	0	0
IHD-LBBB	35	7	17	0	0
Ischemic dilated cardiomyopathy with LBBB	14	6	13	0	0
ISOLATED LBBB	4	0	1	0	0
OTHER DIAGNOSIS	11	4	9	2	1
Total	71	17	48	2	1

GRAPH 13: VALVULAR LESIONS ASSOCIATED WITH LBBB



In the present study, mitral regurgitation is present in 71 subjects out of 100; aortic regurgitation is present in 17 subjects and tricuspid regurgitation in 48 subjects.

Aortic stenosis is seen in 2 subjects and mitral stenosis in 1 subject out of 100.

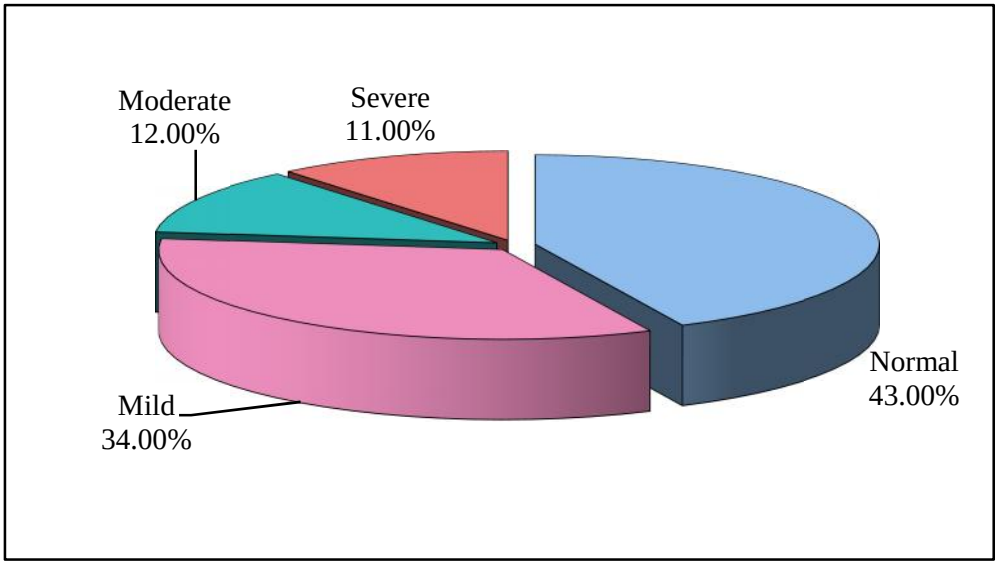
It can be seen in LBBB most common valvular lesion is mitral regurgitation followed by tricuspid regurgitation.

Valvular lesion in individual diagnosis is listed in Table 16.

TABLE 17: PULMONARY ARTERIAL HYPERTENSION

Pulmonary artery hypertension	No of LBBB patients	%of LBBB patients
Normal	43	43.00
Mild	34	34.00
Moderate	12	12.00
Severe	11	11.00
Total	100	100.00

GRAPH 14: PULMONARY ARTERIAL HYPERTENSIO



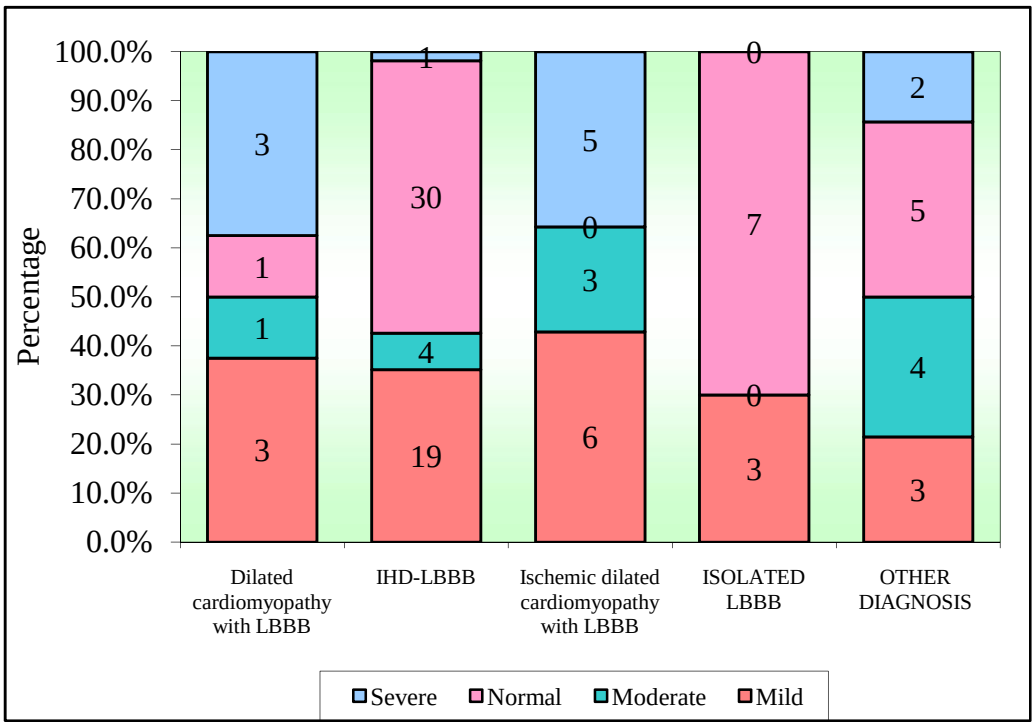
In the present study, among the 100 subjects studied, 57 subjects have pulmonary hypertension out of 100 subjects (57%) and 43 have no pulmonary arterial hypertension (43%).

Out of 57 subjects, 34 have mild PAH, 12 have moderate PAH and 11 have severe PAH.

TABLE 18: PULMONARY ARTERIAL HYPERTENSION IN INDIVIDUAL DIAGNOSIS

Diagnosis	Pulmonary arterial hypertension				
	Mild	Moderate	Normal	Severe	Total
Dilated cardiomyopathy with LBBB	3	1	1	3	8
IHD-LBBB	19	4	30	1	54
Ischemic dilated cardiomyopathy with LBBB	6	3	0	5	14
ISOLATED LBBB	3	0	7	0	10
Other diagnosis	3	4	5	2	14
Total	34	12	43	11	100

GRAPH 15: PULMONARY ARTERIAL HYPERTENSION IN INDIVIDUAL DIAGNOSIS.



Among patients diagnosed with IHD-LBBB, 19 have mild PAH, 4 have moderate PAH and 1 has severe PAH. 30 patients have no PAH on 2D ECHO.

Among patients diagnosed with dilated cardiomyopathy, 3 have mild, 1 has moderate and 3 have severe PAH. 1 patient has no PAH.

Among patients with ischemic dilated cardiomyopathy, 6 have mild, 3 have moderate and 5 have severe PAH.

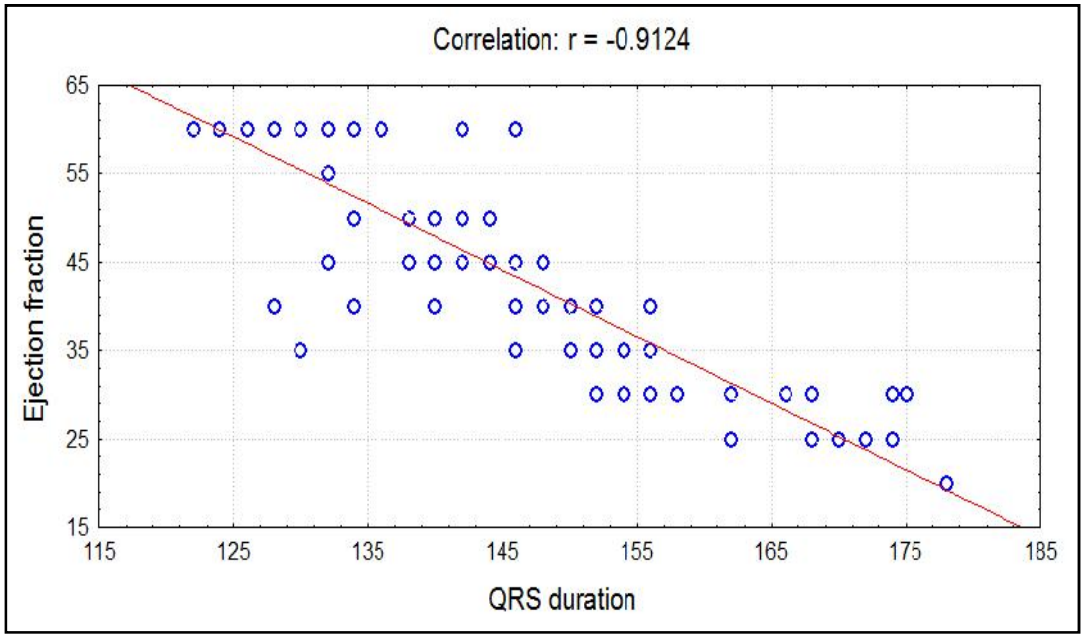
In isolated LBBB patients, 3 have mild PAH, 7 have no PAH.

TABLE 19: CORRELATION BETWEEN QRS DURATION AND EJECTION FRACTION BY KARL PEARSON'S CORRELATION COEFFICIENT

Variables	ejection fraction		
	r-value	t-value	p-value
QRS duration	-0.9124	-22.0663	0.0001*

*p<0.05 indicates negative correlation

GRAPH 16: SCATTER DIAGRAM OF CORRELATION BETWEEN QRS DURATION AND EJECTION FRACTION



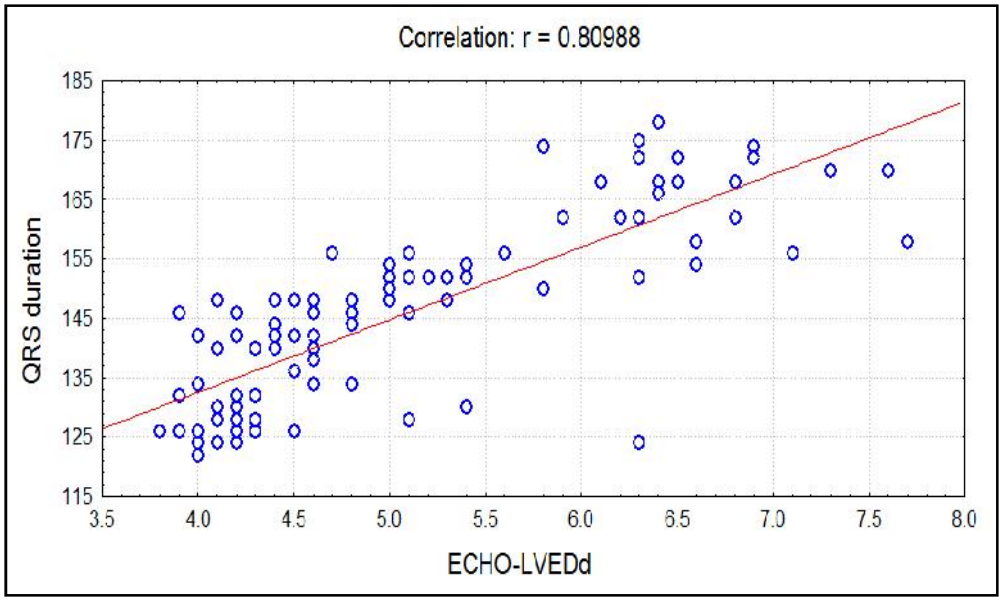
A statistically significant and negative correlation is observed between QRS duration and ejection fraction (r=-0.9124, p=0.0001). It implies that, the QRS duration increases with decrease in ejection fraction or they are dependent on each other.

TABLE 20: CORRELATION BETWEEN QRS DURATION AND LEFT VENTRICLE DIAMETERS BY KARL PEARSON'S CORRELATION COEFFICIENT

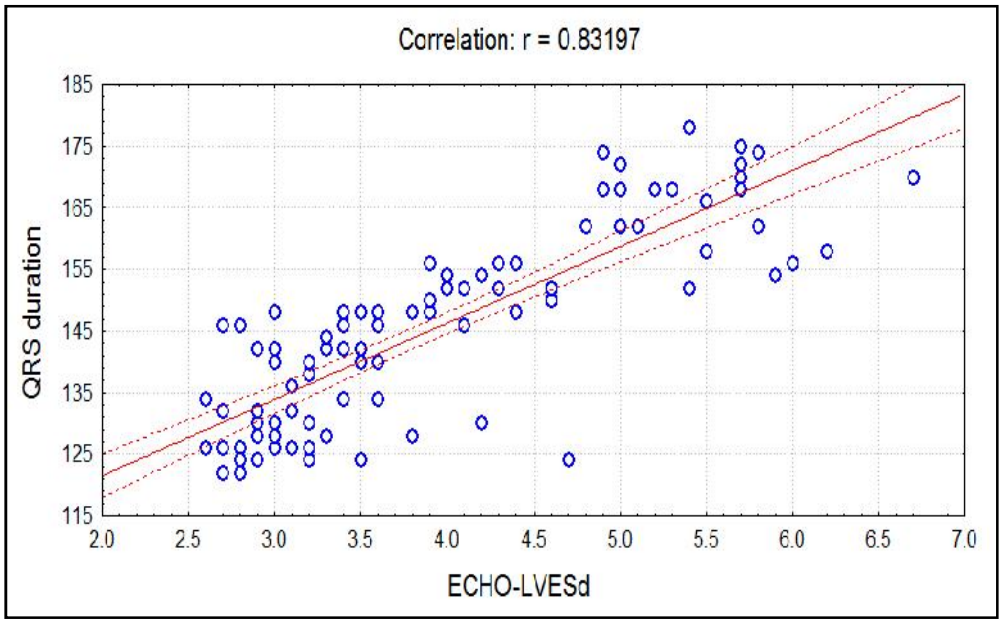
Variables	QRS duration		
	r-value	t-value	p-value
ECHO-LVEDd	0.8099	13.6678	0.0001*
ECHO-LVESd	0.8320	14.8448	0.0001*

*p<0.05 indicates significant correlation

GRAPH 17: SCATTER DIAGRAM OF CORRELATION BETWEEN QRS DURATION AND ECHO-LVEDD



GRAPH 18: SCATTER DIAGRAM OF CORRELATION BETWEEN QRS DURATION AND ECHO-LVESD



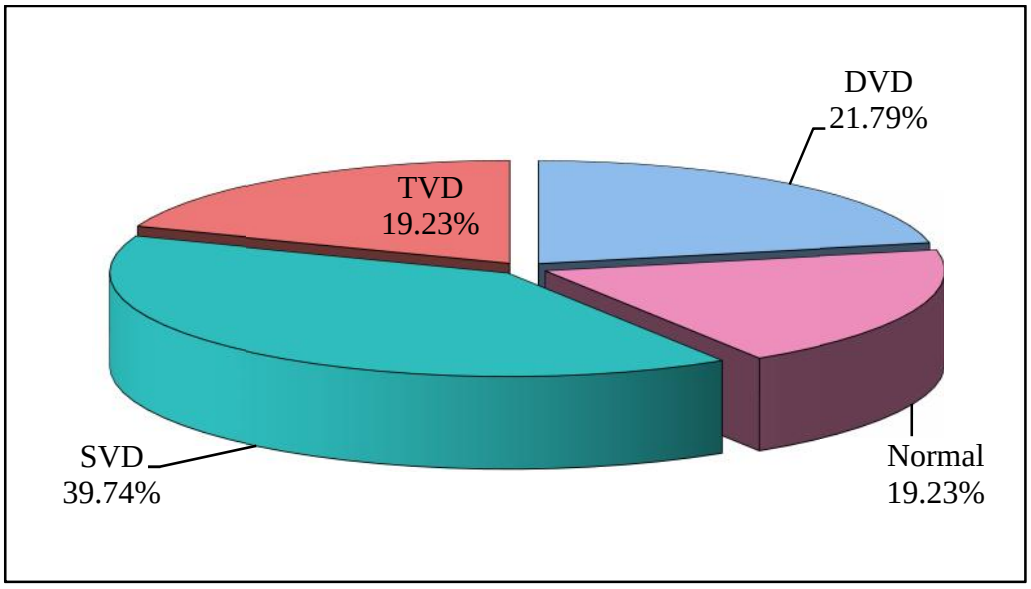
- A statistically significant and positive correlation was observed between QRS duration and LVEDd (r=0.8099, p=0.0001). It other words, the QRS duration increases with increase in LVEDd or they are dependent on each other.

- A statistically significant and positive correlation is observed between QRS duration and LVESd (r=0.8320, p=0.0001). It other words, the QRS duration increases with increase in LVESd or they are dependent on each other.

TABLE 21: CORONARY ANGIOGRAPHY PROFILE OF PATIENTS WITH LBBB

Coronary angiography	No of LBBB patients	%of LBBB patients
NORMAL	15	19.23
SVD	31	39.74
DVD	17	21.79
TVD	15	19.23
Total	78	

GRAPH 19: CORONARY ANGIOGRAPHY PROFILE



In the present study, among the 100 subjects studied 78 underwent coronary angiography and results are as given in Table 21.

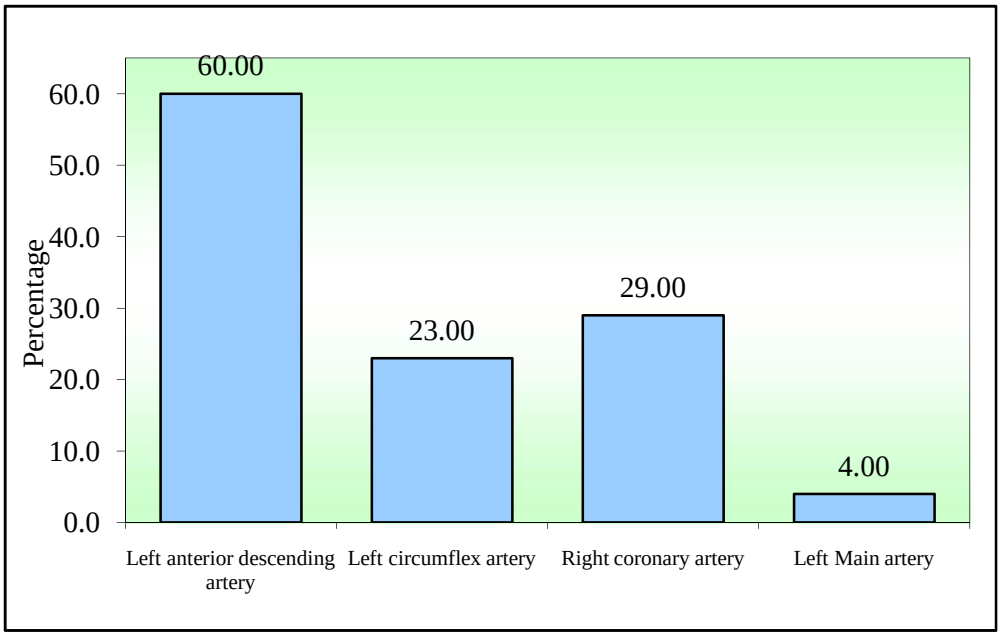
Single vessel disease is the most common coronary angiographic finding, followed by double vessel disease and triple vessel disease.

15 [19.23%] patients out of 78 patients who underwent coronary angiography have normal coronary angiography study.

TABLE 22: CORONARY ARTERY LESIONS

Coronary artery	Yes	No	Total
Left anterior descending artery	60	18	78
Left circumflex artery	23	55	78
Right coronary artery	29	47	78
Left Main	4	74	78

GRAPH 20: CORONARY ARTERY LESIONS

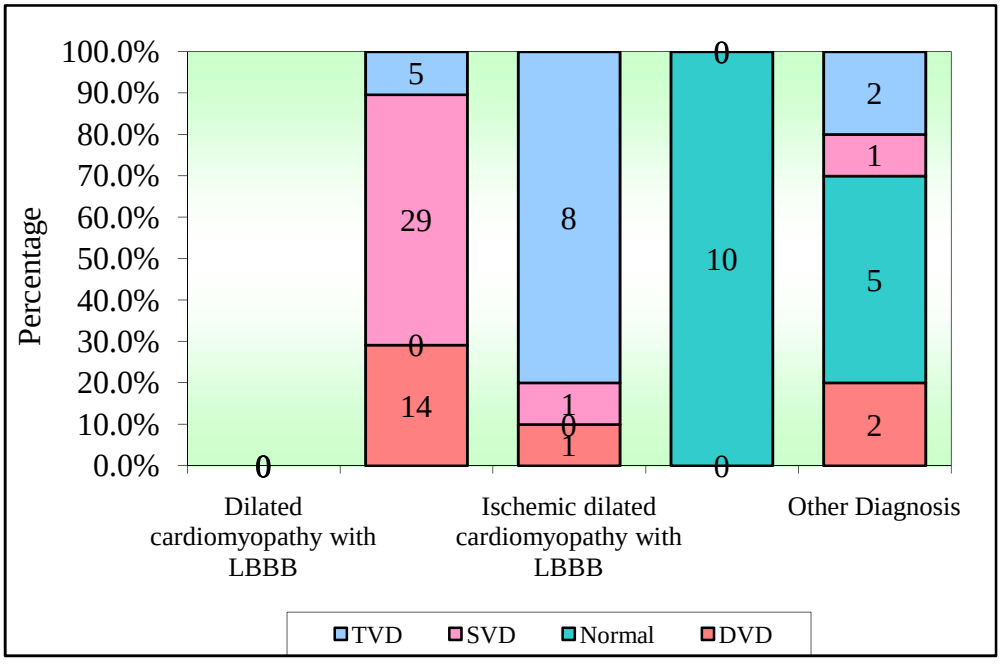


In the study population of 100 subjects' left anterior descending artery is found to be the most commonly involved artery in 60 patients out of 78 who underwent coronary angiography. Right coronary artery is found to be involved in 29 cases of those who underwent coronary angiography and left circumflex is involved in 23 patients. Left main artery is found to be culprit artery in four cases.

TABLE 23: CORONARY ANGIOGRAPHY PROFILE IN INDIVIDUAL DIAGNOSIS

Diagnosis	Coronary angiography				
	DVD	Normal	SVD	TVD	Total
Dilated cardiomyopathy with LBBB	0	0	0	0	0
IHD-LBBB	14	0	29	5	48
Ischemic dilated cardiomyopathy with LBBB	1	0	1	8	10
ISOLATED LBBB	0	10	0	0	10
OTHER DIAGNOSIS	2	5	1	2	10
Total	17	15	31	15	78

GRAPH 21: CORONARY ANGIOGRAPHY PROFILE IN INDIVIDUAL DIAGNOSIS



Among the study population patients diagnosed with IHD-LBBB, 48 out of 54 patients underwent CAG out of which 29 have SVD, 14 have DVD, and 5 have TVD.

Patients diagnosed with ischemic dilated cardiomyopathy, 10 out of 14 have angiography done out of which 8 patients have TVD, 1 has DVD and 1 has SVD.

Isolated LBBB patients all have normal angiography reports.

Patients diagnosed with dilated cardiomyopathy with LBBB, 8 in number didn’t undergo angiography.

Out of 10 patients with other diagnosis, 10 underwent angiography out of which 5 are normal and 5 are abnormal.

DISCUSSION

LBBB is a cardiac conduction defect in which the normal direction of septal depolarisation is reversed, becomes right to left as the impulse spreads first to the right ventricle via the right bundle branch and then to the LV via the septum [1,2]. The present study was undertaken to study the clinical profile and aetiology and left ventricular functions in a patient with LBBB.

In the present study, among the study population of 100 subjects the age ranged from 18 years to 86 years with the mean age being 63.12 ±11.80 years. 13% of the studied population age was less than 40 years, 27% fell into bracket of age 41 to 50 years, 36% in the age bracket 51 to 60 years and 24% above the age of 60 years. Out of 100 subjects, selected in our study 61 out of 100(61%) are male and 39 out of 100(39%) are female. In a study done by Oliver f. Clerc et al in-university hospital Zurich, the mean age of the study population with LBBB was 59.3 years with standard deviation of 11.2 years. In the same study 53% of the subjects with LBBB were male and 47% were female [57]. In a study done by Bino Benjamin et al in Kottayam medical college, India the mean age was 62.7 years and 52.6& patients were male and 47.3% were female [58].

This correlates well with our study population and implies that prevalence of LBBB increases as the age advances and LBBB is slightly more prevalent in males than females. It has been observed that prevalence of LBBB increases from 0.4% at age 50 to 6.5% at age 80[8] due to increased incidence of underlying heart disease such as coronary artery disease, cardiomyopathy etc.

In our study, among the study population of 100 subjects most common presenting complaint was breathlessness in 81% patients followed by chest pain in 38% subjects and giddiness in 18% subjects. 3 subjects presented with non-cardiac complaints and among them 1 was asymptomatic. In a study done by Bharath m.s. et al (2017), breathlessness was present in 35.34% patients, chest pain in 31.89%, giddiness in 10.54% and 20.68% patients were asymptomatic [59]. In the study done by Bino Benjamin et al, breathlessness (35.1%) and chest pain (22.8%) were the most common symptoms.

This correlates with our study where breathlessness as presenting complaint was present in majority of the patients (81%), followed by chest pain in 38% subjects and only 1 patient was asymptomatic. However, the percentage of patients presenting with breathlessness was high in our study.

In our study population among the 100 subjects evaluated, 64 out of 100(64%) subjects are hypertensive and 54 out of 100(54%) subjects are diabetic. Other co morbidities such as COPD was present in 15% subjects and CVA was present in 5% subjects. In the study conducted by Bharath m.s. et al, 50.86% had hypertension and 30.17% had diabetes mellitus as co morbidities. In study conducted by Bino Benjamin et al in 2018, hypertension was present in 54.4% patients and diabetes mellitus was present in 10.75%, COPD in 10.5% patients. Hypertension and diabetes mellitus are known risk factors for left bundle branch block which correlated well with our study.

54 out of 100 subjects (54%) have tobacco consumption in the form of smoking or gutka chewing as habit and 19 out of 100(19%) have alcohol consumption as habit. Smoking is a significant risk factor for ischemic heart disease and majority

of the patients diagnosed with ischemic heart disease with LBBB had smoking as habit.

Among 100 subjects studied, 54 subjects out of 100(54%) were found to have ischemic heart disease with LBBB. This includes patients diagnosed with IHD with new onset LBBB, IHD- unstable angina with LBBB and old cases of IHD with LBBB. Second most common diagnosis was ischemic dilated cardiomyopathy in 14 patients out of 100(14%). Isolated LBBB was found in 10 patients out of 100(10%), followed by dilated cardiomyopathy with LBBB in 8 patients out of 100(8%). From our study it can be seen that the most common etiology for LBBB was ischemic heart disease. Isolated LBBB was present in 10 subjects whose 2d echo and coronary angiographies didn't reveal any major abnormalities. In a study by Schneider et al[7] and Tabuchi et al[60], ischemic heart disease with LBBB was found in 50% cases studied which is similar to our study. Schneider et al observed that LBBB is often a hallmark of advanced ischemic heart disease and associated with increased risk of cardiovascular morbidity and mortality [7].

Pulse rate, systolic blood pressure and diastolic blood pressure were analyzed in all LBBB patients, pulse rate ranged from 40/minute to 130/minute with mean value of88.94/minute±18.72. Systolic blood pressure ranged from 80 mm of hg to 220 mm of hg with mean value of 124.70±24.6. Diastolic blood pressure ranged from 50 mm of hg to 110 mm of hg with mean value of 76.50±13.21. Comparison of vital parameters of pulse rate, systolic and diastolic blood pressure was made between different diagnosis was made and found to be statistically significant a 5% level of significance. Hemoglobin, renal function tests, random blood sugar and hba1c were

analyzed in all LBBB patients and were found to be not statistically different in different diagnosis.

QRS width on ECG was analyzed in all 100 patients with LBBB and in subset of diagnosis with LBBB. QRS duration ranged from 122 milliseconds to 178 milliseconds with a mean value of 144.85±15.39. It is observed that mean QRS duration in subjects diagnosed with dilated cardiomyopathy (163.88±10.48) and ischemic dilated cardiomyopathy (165.57±6.28) was significantly higher than in subjects diagnosed with IHD- LBBB (139.26±10.07) and is least in subjects diagnosed with isolated LBBB (126.60±2.99) which was statistically significant. Johnson, Messer and White [63] noted in a large patient population with LBBB that patients with widest QRS duration had the shortest survival time and they also found correlation between ejection fraction and diameters of left ventricle with QRS duration. In our study among the 100 subjects with LBBB, ejection fraction ranged from minimum of 20% to maximum of 60% with a mean value of 44.20 %±12.73.LVEDd ranged from 3.80 to 7.70 cm in all subjects with LBBB with a mean value of 5.01 cm ±1.02.LVESd ranged from 2.60 cm to 6.70 cm with mean value of 3.89 cm±1.03. It was observed that patients with ischemic dilated cardiomyopathy and dilated cardiomyopathy had lower ejection fraction (27.50±2.59 and 29.38±4.96 respectively) as compared to other diagnosis such as IHD with LBBB and isolated LBBB (47.69±9.70 and 60±0.0 respectively) which was statistically significant.

It was also observed that the left ventricular end diastolic and end systolic diameters were significantly higher in dilated cardiomyopathy (6.16±0.65, 5.16±0.71 respectively) and ischemic dilated cardiomyopathy (6.66±0.46, 5.47±0.42) than in cases with diagnosis as isolated LBBB (4.14±0.15, 2.92±0.16) which was statistically

significant. In study of Recke et al [61], mean LVEF in cases of LBBB with IHD (EF = 54.5+14.1%) and dilated cardiomyopathy (EF =38.6+14.9%) was lower as compared to normal LVEF in patients with no manifest heart disease (LVEF = 68.5+4.6%). In a study done by Chia et al, lower mean LVEF (19.66%) was observed in 6 patients of dilated cardiomyopathy with LBBB [62].

Also, on correlating QRS duration with ejection fraction and diameters of left ventricle in end diastole and end systole, a statistically significant negative correlation was observed between QRS duration and ejection fraction (p- 0.001) and positive correlation between QRS duration and LVEDd and LVESd. This observation was similar to the study conducted by Johnson et al. Patients with LBBB are more predisposed to having LV dysfunction. LBBB patients also have higher mortality when compared with patients with LV systolic dysfunction without LBBB (Fay et al). This can be attributed to higher incidence of coronary artery disease, dilated cardiomyopathy, hypertension and valvular heart disease among patients with LBBB [70]. These findings correlated well with our study. In LBBB, there is alteration of both systolic and diastolic function of left ventricle because of altered septal motion and alteration in diastolic filling time respectively, thereby reducing global cardiac performance. As reported in the study done my Marwan Badri et al [69], this reduced cardiac performance is directly in proportional to the QRS duration.

In our study population, left ventricular dysfunction was present in 69 subjects out of 100 studied (69%) and absent in 31 subjects (31%). Among the 69 subjects who have LV dysfunction, 22 subjects have mild LV dysfunction, 24 have moderate LV dysfunction, and 23 have severe LV dysfunction. In study done by Bino Benjamin, LV dysfunction was observed in 28% of patients studied which was It can

be observed that patients with dilated cardiomyopathy and ischemic dilated cardiomyopathy had more severe LV dysfunction than patients diagnosed as IHD-LBBB. Patients with isolated LBBB have no LV dysfunction. Statistically significant association was observed between LV dysfunction and different diagnosis in patients with LBBB (chi-square=93.2754, p=0.0001). In a study done by Hamby et al it was observed that LBBB associated with symptomatic coronary artery disease is a marker of severe LV dysfunction [64]. Xiao et al reported a similar association of LBBB with deterioration of LV systolic function in patients with cardiomyopathy [65]. Also, this association has been quantified by Zhou et al. Who showed that the LBBB-dependent activation abnormalities had a dominant effect on the deterioration of LV function [66]. Brunekreeft et al. confirmed a significant difference in left ventricular volumes, and LVEF between two groups with and without LBBB [67].

Concentric LVH was present in 38 subjects out of 100(38%) and absent in 62 subjects out of 100(62%). Maximum number of patients who had diastolic dysfunction were diagnosed to have ischemic heart disease with LBBB. 8 patients out 10 patients who were diagnosed to have isolated LBBB had concentric LVH. High prevalence of LVH in IHD and isolated LBBB patients is probably due to higher prevalence of hypertension in such patients.

In the study done by Bharath et al in 2017 among the study population of 116 patients 33.6% patients had left ventricular hypertrophy, which was similar to our study.

Diastolic dysfunction was present in 53 subjects out 100 and absent in 47 subjects out of 100 in our study. Among 53 subjects in whom diastolic dysfunction is observed, 50 subjects have type 1 diastolic dysfunction and 3 have type 2 diastolic

dysfunction. Among 10 patients with isolated LBBB, 8 had type 1 diastolic dysfunction. These finding are similar to the study done by Ozdemir et al in 2004 in turkey [68], in which they found that isolated LBBB patients have increased diastolic filling pressures. This is due to altered electrical activation of left ventricle in patients with LBBB which initiates chain of events leading to delayed contraction of LV, causes global LV abnormalities manifested by abnormal diastolic filling times, LV ejection fraction and IVS motion. This study also showed that occurrence of LBBB in addition to heart failure significantly increased the impairment of diastolic dysfunction. Our study showed that those patients with LBBB not only present with impaired LV systolic function but also impaired diastolic function leading to reduced global cardiac function.

Among the valvular lesions, mitral regurgitation was the most common valvular lesion present in 71 subjects out of 100, followed by tricuspid regurgitation in 48 subjects, aortic regurgitation in 17 subjects. Aortic stenosis was seen in 2 subjects and mitral stenosis in 1 subject out of 100 in our study. Pulmonary hypertension was found to be present in 57 subjects out off 100. Subjects with dilated cardiomyopathy and ischemic dilated cardiomyopathy had more severe forms of PAH, whereas in subjects with isolated LBBB, 3 out 10 had mild PAH.

Left bundle branch has dual blood supply from both the left anterior descending artery and the posterior descending artery through their septal perforating branches. Cad is one of the most common finding in patients with LBBB. In the present study, among the 100 subjects studied 78 underwent coronary angiography. Single vessel disease was the most common coronary angiographic finding present in 31 out of 78 subjects (39.74%, followed by double vessel disease in 17 out of 78

subjects (21.79%) and triple vessel disease in 15 out of 78 subjects (19.23%). 15 patients out of 78 patients (19.23%) who underwent coronary angiography have normal coronary angiography study. This included all the patients diagnosed as isolated LBBB. Left anterior descending artery was found to be the most commonly involved artery in 60 patients out of 78 who underwent coronary angiography. Right coronary artery was found to be involved in 29 cases of those who underwent coronary angiography and left circumflex was involved in 23 patients. Left main artery was involved in four cases. Among the study population patients diagnosed with IHD-LBBB, 48 out of 54 patients underwent CAG out of which 29 had SVD, 14 had DVD, and 5 had TVD. Patients diagnosed with ischemic dilated cardiomyopathy, 10 out of 14 had angiography done out of which 8 patients had TVD, 1 DVD and 1 with SVD. Isolated LBBB patients all had normal angiography reports. Out of 10 patients with other diagnosis, 10 underwent angiography out of which 5 were normal and 5 were abnormal. In the study done by Marwan Badri et al in Lankenau medical research center, USA, among LBBB patients, evidence of coronary artery disease was found in 72% of the patients which is similar to our study [69]. In a study done by Hamby et al, they found SVD in 8 patients out of 51(15.7%), DVD in 15 out of 51 (29.54%) and TVD in 28 out 51 (54.9%) patients with LBBB subjected to CAG. These results were in contrast to our study where single vessel disease was the most common lesion. So, from our study we can observe that hemodynamic evaluation, electrophysiological evaluation and coronary angiographic studies is a must in cases with LBBB.

CONCLUSION

- The risk of LBBB increases as the age increases. in our study the mean age of the patients was 63.12 years.
- Prevalence of LBBB is slightly greater in males.
- The most common presenting complaint was breathlessness followed by chest pain.
- Hypertension, diabetes mellitus and smoking were found to be important risk factors for LBBB.
- The most common etiology for LBBB was found to be ischemic heart disease followed by ischemic dilated cardiomyopathy. isolated LBBB was seen in 10% cases.
- Parameters such as hemoglobin, creatinine, random blood sugar and HBA1C had no significant variation among the various etiologies of LBBB.
- QRS duration, ejection fraction and Left ventricle diameters were found to be statistically significant in association with etiology of LBBB.
- Left ventricle systolic dysfunction was present in 69 patients out of 100. It had statistically significant association with etiology of LBBB.
- Diastolic dysfunction as assessed by echocardiography was present in 53 patients out of 100. It had statistically significant association with etiology of LBBB.

- Most common valvular lesion associated with LBBB was mitral regurgitation as per our study.

- QRS duration had a negative correlation with ejection fraction which was statistically significant.

- Among the patients who were subjected to coronary angiography, single vessel disease was found to be the most common lesion, followed by double vessel disease and triple vessel disease and normal in 19.23%. The most common vessel implicated was left anterior descending followed by right coronary artery. Left main artery lesion was found in 4 cases.

SUMMARY

The present study of 100 patients with Left bundle branch block was conducted in KLES Dr. Prabhakar Kore Hospital and Research Centre from January 1, 2018 to December 31, 2018. The mean age of the patients was 63.12 years. Prevalence of LBBB was slightly greater in males than females (61%vs39%). The most common presenting complaint was breathlessness. Hypertension, diabetes mellitus and smoking were found to be commonly associated with LBBB. The most common etiology for LBBB was found to be ischemic heart disease.

QRS duration, ejection fraction and Left ventricle diameters were found to be statistically significant in association with etiology of LBBB. Left ventricular systolic and diastolic dysfunction was a common finding present in patients with LBBB. Most common valvular lesion associated with LBBB was mitral regurgitation as per our study.

QRS duration had a negative correlation with ejection fraction which was statistically significant. Among the patients who were subjected to coronary angiography, single vessel disease was found to be the most common lesion. The most common vessel implicated was left anterior descending followed by right coronary artery.

We propose that an in-depth study of a larger sample size to compare the above issues with further variables. A prospective follow up study can also be considered to give a broader view into the prognosis of LBBB.

LIMITATIONS OF THE STUDY

1. We found that in our study there were some limitations with the sample size and sample selection which precluded us from getting statistical significance with regard to certain variables.

2. The prognostic and mortality details cannot be determined as it requires long term follow up.

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ANNEXURE I – CONSENT FORM

Title Of Research Study:

CLINICAL PROFILE OF LEFT BUNDLE BRANCH BLOCK IN TERTIARY CARE HOSPITAL- A CROSS SECTIONAL STUDY

Principal Investigator:- **Guide:-**

Introduction and Purpose:-

He/ She is a patient diagnosed with Left Bundle Branch Block on ECG

Procedure:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood and urine samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.

You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study.

If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belagavi as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

1. Dr. Roopa Bellad, Chairman, J.N.M.C Ethical Committee for Human Research 9480275601

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me and has been explained to me in my vernacular language and all my questions have been answered. I will be given a copy of this consent form.

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name :……...

Signature / Left thumb impression:……...

of the participant

Name of the legally authorized :……...

representative / guardian

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

Investigator's name and signature :……...

Date:

Place:

ANNEXURE-II

PROFORMA

CLINICAL PROFILE OF LEFT BUNDLE BRANCH BLOCK IN TERTIARY CARE HOSPITAL- A CROSS SECTIONAL STUDY

Case number:

Name: **Age:** **Sex:**

IP Number

COMPLAINTS AT PRESENTATION:

- **Breathlessness:**
- **Chest pain:**
- **Giddiness:**
- **Others:**

CO MORBIDITIES:

- **Hypertension:**
- **Diabetes mellitus:**
- **Others:**

RISK FACTORS:

- **Tobacco consumption:**
- **Alcohol consumption:**

ADMISSION DIAGNOSIS:

PHYSICAL EXAMINATION:

- **Pulse Rate:**
- **Blood Pressure:**

SYSTEMIC EXAMINATION:

- **CVS:**
- **RS:**
- **PA:**
- **CNS:**

LABORATORY INVESTIGATIONS:

- **Hemoglobin (gm/dl):**
- **Creatinine (mg/dl):**
- **Random Blood Sugar (mg/dl):**
- **Sodium (meq/l):**
- **Potassium (meq/l):**
- **HBA1C (%):**

ECG:

- **QRS Duration:**

ECHO:

- **Asynchronous movement of left ventricle in view of LBBB:**
- **LVEDd (cm):**
- **LVESd (cm):**
- **Ejection Fraction (%):**
- **LV Dysfunction:**
- **Left Ventricular Hypertrophy:**
- **Valvular lesions:**
    - **Mitral valve:**
    - **Tricuspid valve:**
    - **Aortic valve:**
    - **Pulmonary valve:**
- **Diastolic Dysfunction:**
- **Pulmonary Arterial Hypertension:**

CORONARY ANGIOGRAPHY:

- **Report:**
- **Artery:**
    - **Left Anterior Descending artery:**
    - **Left Circumflex artery:**
    - **Right Coronary artery:**
    - **Left Main artery:**

ANNEXURE-III- ETHICAL CLEARANCE LETTER

K.L.E.UNIVERSITY'S
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
(Accredited 'A' Grade by NAAC)

Website: http//www.jnmc.edu
E-Mail : domc@jnmc.edu

Phone: (+ 91-(0)831 Office : 2471350
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 37 **Date: 22/11/2017**

To,

REG NO. BG0117008

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CLINICAL PROFILE OF LEFT BUNDLE BRANCH BLOCK IN TERTIARY CARE HOSPITAL – A CROSS SECTIONAL STUDY",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURES IV - MASTER CHART

S.NO	NAME	IP NO.	AGE	SEX	BREATHLESSNESS	CHEST PAIN	GIDDINESS	OTHERS	CHIEF COMPLAINT	HYPERTENSION	DIABETES	OTHERS	TOBACCO	ALCOHOL	PRIMARY DIAGNOSIS	SECONDARY DIAGNOSIS	PULSE RATE(HR/min)	SBP(mm of HG)	DBP(mm of HG)	HB(gm/dl)	CREATININE(mg/dl)	RBS(mg/dl)	SODIUM(meq/l)	POTASSIUM(meq/l)	HBA1C(%)	ECG-QRS(milliseconds)	ECHO-EF(%)	LV DYSFUNCTION	ECHO-LVEDd(cm)	ECHO-LVESd(cm)	LEFT VENTRICULAR HYPERTROPHY	VALVULAR LESIONS	MITRAL REGURGITATION	AORTIC REGURGITATION	TRICUPSID REGURGITATION	AORTIC STENOSIS	MITRAL STENOSIS	OTHERS	DIASOLIC DYSFUNCTION	PULMONARY ARTERY HYPERTENSION	CORONARY ANGIOGRAPHY	LEFT ANTERIOR DESCENDING ARTERY	LEFT CIRCUMFLEX ARTERY	RIGHT CORONARY ARTERY	OTHERS
1	LAKSHMI PUJARI	926190	52	F	Y	Y	N	N	BREATHLESSNESS, CHEST PAIN	N	Y	N	N	N	IHD -LBBB		116	110	70	14.4	0.63	601	139	3.95	12.2	130	35	MODERATE	5.4	4.2	ABSENT	GRADE 1 MR, GRADE 1 TR	Y	N	Y	N	N		ABSENT	MILD	SVD	Y	N	N	N
2	KUMAR B PAWAR	926169	45	M	Y	Y	N	N	BREATHLESSNESS, CHEST PAIN	N	N	RHD, EPILEPSY	Y	Y	LBBB WITH ATRIAL FIBRILLATION STATUS POST AVR WITH STUCK VALVE		90	100	60	14.5	0.83	109	142	4.2	5.7	124	60	ABSENT	6.3	4.7	PRESENT	STUCK AV VALVE,MILD MS, GRADE 2 MR, GRADE 2 AR, GRADE 1 TR	Y	Y	Y	N	Y	STUCK AV VALVE	ABSENT	MODERATE	ND				
3	LAXMIBAI GADDI	964880	60	F	Y	N	N	B/L LOWER LIMB SWELLING	BREATHLESSNESS, B/L LOWER LIMB SWELLING	Y	Y	COPD	N	N	IHD- LBBB WITH IWMI		80	200	110	12.2	0.9	201	141	4.5	7.4	146	45	MILD	3.9	2.7	ABSENT	GRADE 1 MR, TRIVIAL TR, TRIVIAL AR	Y	N	N	N	N		ABSENT	MILD	TVD	Y	Y	Y	LEFT MAIN
4	CHANDBI AKKHAJI	874809	45	F	Y	N	N	N	BREATHLESSNESS ON EXERTION	Y	N	N	N	N	DILATED CARDIOMYOPATHY WITH LBBB		110	140	90	13.6	1.04	152	144	4.19	5.2	154	35	MODERATE	5	4	ABSENT	GRADE 1 MR, GRADE 2 TR	Y	N	Y	N	N		TYPE 1	NORMAL	ND				
5	RAJ R FERNANDEZ	872249	60	M	N	Y	N	N	CHEST PAIN	Y	N	N	Y	Y	IHD- LBBB		90	110	80	12.5	0.95	134	144	3.9	5.2	148	45	MILD	4.4	3.5	PRESENT	TRIVIAL MR	N	N	N	N	N		TYPE 1	MILD	SVD	Y	N	N	N
6	RAVI KULKARNI	874999	48	M	Y	N	N	N	BREATHLESSNESS	Y	Y	N	Y	N	ISOLATED LBBB		80	110	70	13.5	0.97	207	134	4.91	6.9	130	60	ABSENT	4.1	2.9	PRESENT	ABSENT	N	N	N	N	N		TYPE 1	NORMAL	NORMAL				
7	DROUPADI DHANVANTH	896378	60	F	Y	N	N	B/L LOWER LIMB SWELLING	BREATHLESSNESS ON EXERTION, B/L LOWER LIMB SWELLING WITH FACIAL PUFFINESS	N	N	N	N	N	IHD- LBBB		70	120	70	13.2	1.68	93	142	3.85	ND	128	60	ABSENT	4.3	2.9	ABSENT	GRADE 1 TR	N	N	Y	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
8	KEMPANNA	887182	60	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	N	N	COPD	Y	N	IHD - LBBB WITH IWMI		90	110	80	15.8	1.04	87	134	4.42	ND	138	50	MILD	4.6	3.2	ABSENT	GRADE 1 MR, GRADE 2 TR	Y	N	Y	N	N		ABSENT	NORMAL	DVD	Y	N	Y	N
9	ANNAPOORNA BADIGER	924630	68	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	N	N	N	N	IHD -LBBB		82	150	90	11.4	0.59	95	140	3.45	5.2	132	60	ABSENT	4.3	2.9	PRESENT	TRIVIAL MR,TRIVIAL TR	N	N	N	N	N		TYPE 1	NORMAL	TVD	Y	Y	Y	N
10	YAMANAVVA HOSUR	925088	69	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	N	N	N	Y	N	IHD -LBBB		96	120	70	11.7	0.59	92	132	4.41	ND	148	45	MILD	4.1	3	ABSENT	GRADE 2 MR, TRIVIAL TR AND AR	Y	N	N	N	N		TYPE 1	MILD	SVD	Y	N	N	N
11	NEELAVVA GURAPPOGOUDRA	924262	60	F	Y	N	N	N	BREATHLESSNESS	Y	N	N	N	N	IHD - LBBB		70	110	70	11.5	0.73	95	143	4.05	ND	126	60	ABSENT	3.8	2.8	ABSENT	TRIVIAL MR	N	N	N	N	N		TYPE 1	NORMAL	DVD	Y	N	Y	N
12	REVATI DEVALAPUR	925393	68	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	N	Y	N	N	N	IHD - LBBB		80	140	90	11	0.7	188	136	4.13	9.6	130	60	ABSENT	4.1	3	ABSENT	TRIVIAL MR	N	N	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
13	NINGAVA BAMMANNAVAR	925405	63	F	Y	N	Y	N	BREATHLESSNESS, GIDDINESS	N	Y	N	N	N	IHD -LBBB		100	110	70	12.7	0.69	233	133	4.29	10	146	35	MODERATE	5.1	4.1	ABSENT	GRADE 3 MR, TRIVIAL AR, TRIVIAL TR	Y	N	N	N	N		TYPE 2	MILD	SVD	Y	N	N	N
14	SATAPPA MADER	932774	60	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	N	COPD	Y	Y	IHD -LBBB		60	130	80	15.2	1.46	96	137	4.96	5.8	142	45	MILD	4.4	3.5	PRESENT	GRADE 1 MR, GRADE 2 TR	Y	N	Y	N	N		TYPE 1	MILD	DVD	Y	N	Y	N
15	SANTOSH SEVADATTI	894080	47	M	N	N	Y	N	HGIDDINESS WITH SYNCOPE	N	N	N	Y	N	IHD-LBBB WITH 2:1 AV BLOCK		40	120	70	15.1	1.36	141	137	4.75	5.2	140	50	MILD	4.1	3	ABSENT	TRIVIAL AR	N	N	N	N	N		ABSENT	NORMAL	SVD	N	N	Y	N
16	AKKAVVA NAVI	4481221	55	F	Y	N	N	N	BREATHLESSNESS	Y	Y	N	N	N	IHD- LBBB		72	120	80	10.9	0.54	264	142	4.02	8.9	152	40	MODERATE	5.1	4	PRESENT	GRADE 1 MR, TRIVIAL AR	Y	N	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
17	RAMGOND BIRADERI	894619	53	M	N	Y	Y	N	CHEST PAIN, GIDDINESS	N	N	COPD	Y	N	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		94	90	50	10.4	1.08	194	124	3.93	13.2	158	30	SEVERE	7.7	6.2	ABSENT	SEVERE MR, GRADE 3 TR	Y	N	Y	N	N		TYPE 2	SEVERE	TVD	Y	Y	Y	N
18	DURGAPPA YADAV	933414	70	M	Y	N	N	N	BREATHLESSNESS	Y	Y	PARKINSONISM	Y	Y	IHD- LBBB		110	90	60	13.1	1.45	143	129	5.77	7	148	40	MODERATE	5	3.9	PRESENT	GRADE 1 MR,GRADE 1 AR	Y	Y	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
19	BALWANTRAO DESAI	909268	76	M	N	Y	N	N	CHEST PAIN	Y	N	N	N	N	IHD- LBBB		100	100	60	14.4	1.04	208	137	4.37	5.5	140	40	MODERATE	4.6	3.6	PRESENT	TRIVIAL MR, TRIVIAL TR	N	N	N	N	N		ABSENT	NORMAL	SVD	Y	N	N	N
20	CHANDRU WARAD	893973	40	M	Y	N	N	COUGH WITH EXPECTORATION	BREATHLESSNESS, COUGH WITH EXPECTORATION	Y	N	COPD	Y	Y	DILATED CARDIOMYOPATHY WITH LBBB		106	100	60	10.6	1.72	97	131	4.68	6.9	175	30	SEVERE	6.3	5.7	ABSENT	GRADE 2 MR,GRADE 2 TR	Y	N	Y	N	N		ABSENT	MODERATE	ND				
21	IRAPPA ASUNDI	873007	74	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	Y	COPD	Y	N	IHD- LBBB		82	100	60	11.9	1.19	215	132	4.65	10.7	134	60	ABSENT	4	2.6	PRESENT	GRADE 1 MR	Y	N	N	N	N		TYPE 1	NORMAL	TVD	Y	Y	Y	N
22	RUKHMINI DEVALE	760375	75	F	N	Y	Y	N	CHEST PAIN, GIDDINESS	Y	Y	CVA	N	N	IHD- LBBB		76	220	110	13.3	0.66	110	136	4.8	7.3	136	60	ABSENT	4.5	3.1	PRESENT	GRADE 1 MR, TRIVIAL AR	Y	N	N	N	N		TYPE 1	NORMAL	ND				
23	SHRIKANT PATTN	821875	50	M	N	Y	N	N	CHEST PAIN	Y	Y	N	Y	N	IHD- LBBB		84	150	90	13.5	0.9	345	136	4.31	8.5	146	40	MODERATE	4.8	3.6	PRESENT	GRADE 1 MR, TRIVIAL AR	Y	N	N	N	N		TYPE 1	MILD	SVD	Y	N	N	N
24	SHANKAR PATIL	662615	54	M	N	N	Y	N	GIDDINESS	N	N	N	N	N	ISOLATED LBBB		76	140	80	10.3	0.92	77	132	4	ND	126	60	ABSENT	4.5	2.6	ABSENT	GRADE 1 MR	Y	N	N	N	N		ABSENT	NORMAL	NORMAL				
25	SANJU GADAD	831005	18	M	Y	N	N	EASY FATIGUABILITY	BREATH;ESSNESS	N	N	N	N	N	MITRAL VALVE PROLAPSE WITH LBBB		60	110	80	15.4	1.17	93	136	4.17	ND	136	60	ABSENT	4.5	3.1	ABSENT	GRADE 1 MR, GRADE 2 TR	Y	N	Y	N	N		TYPE 1	MODERATE	NORMAL				
26	BABU MALI	793856	76	M	Y	N	Y	N	BREATHLESSNESS, GIDDINESS	N	N	COPD	Y	Y	DILATED CARDIOMYOPATHY WITH LBBB		82	110	70	12.1	1.18	96	139	4.21	ND	174	25	SEVERE	6.9	5.8	ABSENT	GRADE 3 MR, GRADE 1 TR	Y	N	Y	N	N		ABSENT	MILD	ND				
27	CHANDRASUBHARAYADU	917000	76	M	Y	N	N	WEAKNESS OF LEFT UPPER LIMB, SLURRING OF SPEECH	WEAKNESS OF LEFT UPPER LIMB, SLURRING OF SPEECH, BREATHLESSNESS	N	Y	COPD	Y	Y	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		90	110	70	13	0.72	146	136	3.78	7.8	168	30	SEVERE	6.4	5	ABSENT	GRADE 2 MR, GRADE 2 TR, TRIVIAL AR	Y	N	Y	N	N		TYPE 1	MILD	ND				
28	UMESH	935628	39	M	Y	N	N	ABDOMEN DISTENSION, JAUNDICE	ABDOMEN DISTENSION, JAUNDICE, BREATHLESSNESS ON EXERTION	N	N	ALD	Y	Y	IHD- LBBB		84	100	60	11.2	0.78	96	135	3.44	ND	134	40	MODERATE	4.8	3.4	ABSENT	GRADE 1 MR	Y	N	N	N	N		TYPE 1	NORMAL	ND				
29	MALLAPPA KAKATNUR	850706	61	M	Y	N	N	COUGH	BREATHLESSNESS, COUGH WITH EXPECTORATION	Y	Y	N	Y	N	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		78	80	50	12.4	1.54	109	127	6.28	9.1	168	30	SEVERE	6.1	4.9	PRESENT	GRADE 3 MR, GRADE 2 AR, GRADE 2 TR	Y	Y	Y	N	N		TYPE 2	SEVERE	TVD	Y	Y	Y	N
30	PRABHAKAR KODKANI	850542	65	M	N	Y	N	N	CHEST PAIN	N	N	N	Y	N	IHD- LBBB		110	140	80	9.4	1.11	136	136	4.69	6.5	142	45	MILD	4.2	3	ABSENT	TRIVIAL MR	N	N	N	N	N		ABSENT	MILD	SVD	Y	N	N	N
31	ANNAPPA KHAT	856680	60	M	Y	N	Y	N	BREATHLESSNESS, GIDDINESS	Y	Y	N	Y	Y	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		80	94	60	13.9	0.7	291	132	4.26	16.8	170	25	SEVERE	7.3	5.7	ABSENT	GRADE 3 MR, GRADE 2 TR	Y	N	Y	N	N		ABSENT	MILD	TVD	Y	Y	Y	N
32	DASTAGIR MULLA	859100	71	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	Y	N	Y	N	IHD- LBBB		72	120	80	13.7	1.7	187	144	4	7.4	142	45	MILD	4.6	3.4	PRESENT	GRADE 1 MR, TRIVIAL AR	Y	N	N	N	N		TYPE 1	NORMAL	TVD	Y	Y	Y	N
33	SANGAMMA DHAHED	859723	62	M	Y	N	N	COUGH, FEVER	BREATHLESSNESS, FEVER, COUGH WITH EXPECTORATION	Y	Y	TUBERCULOSIS	Y	N	ISOLATED LBBB		130	170	90	10.6	0.41	265	140	3.83	6.5	124	60	ABSENT	4	2.8	PRESENT	GRADE 1 MR	Y	N	N	N	N		ABSENT	MILD	NORMAL				
34	SUNANDA PAWAR	858841	79	F	Y	N	N	N	BREATHLESSNESS	Y	Y	N	N	N	IHD- LBBB		112	130	90	15.3	1.16	242	131	4.33	9.4	138	45	MILD	4.6	3.2	PRESENT	GRADE 1 MR, GRADE 1 TR	Y	N	Y	N	N		ABSENT	MILD	ND				
35	BALAPPA TALWAR	865890	70	M	Y	N	N	N	BREATHLESSNESS	N	Y	N	Y	N	IHD- LBBB, AORTIC STENOSIS		114	110	70	11.8	1.52	194	129	7.36	9.3	162	25	SEVERE	6.3	5.1	PRESENT	GRADE 3 TR, GRADE 2 MR, SEVERE CALCIFIC AS	Y	N	Y	Y	N		TYPE 1	SEVERE	ND				
36	JINAPPA HARAR	861258	60	M	Y	N	N	DECREASED URINE OUTPUT	BREATHLESSNESS, DECREASED URINE OUTPUT	N	N	RHD	Y	N	LBBB S/P AVR WITH STUCK VALVE		90	90	50	10.1	1	96	137	3.63	ND	170	25	SEVERE	7.6	6.7	ABSENT	SEVERE MR, SEVERE AR, GRADE 1 TR	Y	Y	Y	N	N		TYPE 1	MILD	NORMAL				
37	SIDDHAPPA DHANGAR	925571	68	M	Y	N	N	COUGH	BREATHLESNESS, COUGH WITH EXPECTORATION	N	Y	COPD	Y	Y	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		100	90	50	12.9	3.04	278	122	6.48	8.5	172	25	SEVERE	6.3	5.7	ABSENT	GRADE 2 MR, SEVERE TR	Y	N	Y	N	N		ABSENT	MODERATE	TVD	Y	Y	Y	N
38	MALLAPPA KUDARI	918532	75	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	Y	N	N	N	ISOLATED LBBB		80	130	80	14.4	0.89	178	139	4.3	7.1	128	60	ABSENT	4.1	3	PRESENT	TRIVIAL MR	N	N	N	N	N		TYPE 1	NORMAL	NORMAL				
39	RAMCHANDRA SUTAR	896705	60	M	Y	N	N	N	BREATHLESSNESS	Y	Y	HYPOTHYROIDISM	N	N	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		78	100	70	10	1.15	162	121	5.22	8.7	168	25	SEVERE	6.8	5.7	ABSENT	SEVERE MR, GRADE 3 TR, GRADE 1 AR	Y	Y	Y	N	N		ABSENT	MODERATE	ND				

40	SATYAPPA BASAI	888758	62	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	N	N	N	N	ISOLATED LBBB		70	130	80	14.5	1.12	89	141	4.41	ND	126	60	ABSENT	4	3	PRESENT	ABSENT	N	N	N	N	N		TYPE 1	NORMAL	NORMAL				
41	VEERANNA HOSMANI	858123	73	M	Y	Y	N	N	CHEST PAIN, bREATHLESSNESS	Y	N	N	N	N	IHD-LBBB		74	140	80	13.8	1.13	75	139	3.87	ND	130	60	ABSENT	4.1	3	PRESENT	ABSENT	N	N	N	N	N		TYPE 1	NORMAL	SVD	Y	Y	Y	Y
42	RAMAPPA KUMATANNAVAR	858337	65	M	Y	Y	N	N	BREATHLESSNESS, CHEST PAIN	N	N	COPD	Y	N	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		106	130	80	11.4	0.98	105	140	4.22	ND	168	25	SEVERE	6.1	5.2	ABSENT	GRADE 3 MR, GRADE 2 TR, GRADE 1 AR	Y	Y	Y	N	N		ABSENT	MILD	TVD	Y	Y	Y	N
43	KAMALAVVA GADDI	856038	59	F	Y	Y	N	N	BREATHLESSNESS, CHEST PAIN, COUGH WITH EXPECTORATION	Y	Y	COPD	Y	Y	IHD- LBBB		88	100	70	11.2	0.78	291	138	4.07	14	148	40	MODERATE	4.6	3.4	ABSENT	GRADE 2 MR, GRADE 1 TR	Y	N	Y	N	N		ABSENT	MILD	DVD	Y	Y	N	N
44	RAMAPPA PUJARI	854453	61	M	Y	N	Y	N	BREATHLESSNESS, GIDDINESS	N	N	N	Y	N	LBBB WITH LV DYSFUNCTION		72	110	80	10.5	0.93	94	139	4.2	ND	142	45	MILD	4.6	3.4	ABSENT	TRIVIAL MR, GRADE 1 AR	N	Y	N	N	N		ABSENT	NORMAL	NORMAL				
45	BALVANTARAY DESAI	895421	78	M	N	Y	N	N	CHEST PAIN	Y	N	N	Y	N	IHD-LBBB		86	100	60	11.5	1.1	110	135	4.5	ND	132	45	MILD	4.3	3.1	PRESENT	GRADE 1 MR	Y	N	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
46	TAMMANNNA ADNADDI	895014	62	F	N	Y	N	N	CHEST PAIN	Y	Y	PVD	N	N	IHD- LBBB WITH IWMI		100	130	90	11.8	0.91	241	133	4.78	8.7	156	40	MODERATE	5.1	4.3	PRESENT	GRADE 1 AR, GRADE 1 TR, MODERATE CALCIFIC AS	N	Y	Y	Y	N		ABSENT	MODERATE	TVD	Y	Y	Y	N
47	GUNDAPPA MEDIGOUDRA	895041	78	M	N	N	N	N	ASYMPTOMATIC	Y	N	N	N	N	ISOLATED LBBB		112	150	90	7.4	1.87	137	148	4.47	ND	122	60	ABSENT	4	2.8	PRESENT	GARDE 1 MR	Y	N	N	N	N		TYPE 1	NORMAL	NORMAL				
48	MANIKRAO KULKARNI	894600	71	M	Y	Y	Y	URINARY RETENTION	GIDDINESS, BREATHLESSNESS, URINARY RETENTION	Y	N	COPD,TIA, CA PROSTATE, AF	Y	Y	SECONDARY PAH- LBBB WITH AF		98	100	70	11.9	2.96	172	123	3.49	ND	146	60	ABSENT	3.9	2.8	ABSENT	GRADE 3 MR, GRADE 2 TR	Y	N	Y	N	N		TYPE 1	SEVERE	ND				
49	SUSHILA BING	895531	82	F	Y	N	N	COUGH, FEVER	BREATHLESSNESS, COUGH WITH EXPECTORATION, FEVER	Y	N	CKD	N	N	IHD-LBBB		98	160	90	8.9	1.64	155	123	5.63	5.9	126	60	ABSENT	3.9	2.7	PRESENT	GARDE 1 MR, GRADE 1AR	Y	Y	N	N	N		TYPE 1	NORMAL	ND				
50	SHARDA PATIL	898090	85	F	Y	N	N	WEAKNESS OF RIGHT UPPER AND LOWER LIMB, SLURRING OF SPEECH	BRATHLESSNESS, WEAKNESS OF RIGHT UPPER AND LOWER LIMB, SLURRING OF SPEECH	Y	Y	N	N	N	IHD- LBBB	PNEUMONIA	70	166	100	10.1	0.56	121	142	4.1	6.4	126	60	ABSENT	4.3	3.1	ABSENT	GRADE 1 MR, GRADE 1TR	Y	N	Y	N	N		TYPE 1	MODERATE	TVD	Y	Y	Y	N
51	MANGALA	901471	66	F	N	N	Y	INVOLUNTARY MOVEMENTS OF B/L UPPER LIMB	INVOLUNTARY MOVEMENTS OF B/L UPPER LIMB, GIDDINESS	N	Y	CVA	N	N	IHD-LBBB		90	90	50	9.9	1.88	105	137	5.65	6.6	146	40	MODERATE	4.2	3.4	ABSENT	GRADE 1 MR	Y	N	N	N	N		ABSENT	NORMAL	ND				
52	KASHINATH KAROSHI	907257	27	M	Y	N	N	N	BREATHLESSNESS	N	N	N	N	N	COARCTATION OF AORTA WITH AORTIC ARCH ANEURYSM WITH LBBB		128	110	70	15.3	1.61	106	133	4.12	ND	174	30	SEVERE	5.8	4.9	ABSENT	GRADE 1 MR, GRADE 2 TR	Y	N	Y	N	N		ABSENT	MODERATE	ND				
53	APPANNA HANMARDDI	923461	78	M	Y	Y	N	N	BREATHLESSNESS, CHEST PAIN	Y	Y	COPD	Y	N	IHD-LBBB		122	110	70	11.4	0.77	154	138	5.1	9.1	156	35	MODERATE	4.7	3.9	ABSENT	GARDE 2 MR, GRADE 2 TR	Y	N	Y	N	N		ABSENT	SEVERE	SVD	N	Y	N	N
54	MEERANSAB GOUS	923498	48	M	Y	N	N	DECREASED URINE OUTPUT	BREATHLESSNESS, DECREASED URINE OUTPUT	Y		N	N	N	DILATED CARDIOMYOPATHY WITH LBBB		112	110	70	13.5	0.91	124	125	5.53	ND	178	20	SEVERE	6.4	5.4	ABSENT	TRIVIAL MR, GARDE 2 TR	N	N	Y	N	N		ABSENT	SEVERE	ND				
55	DARSHANA PATHANIA	922468	56	F	Y	N	N	N	BREATHLESSNESS	Y	Y	HYPOTHYROIDISM	N	N	IHD-LBBB		106	140	80	9.9	0.68	196	132	4.52	11.7	128	40	MODERATE	5.1	3.8	ABSENT	GRADE 1 MR, GRADE 1 TR. TRIVIAL AR	Y	N	Y	N	N		ABSENT	MILD	SVD	Y	N	N	N
56	SHOBHA TEGGINAVAR	862755	59	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	Y	N	N	N	IHD-LBBB		100	120	80	12	0.63	137	138	3.99	10.4	162	30	SEVERE	5.9	4.8	PRESENT	GRADE 2 MR, GRADE 1 AR	Y	Y	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
57	MARUTI DESAI	869157	56	M	Y	N	N	FEVER, COUGH	BREATHLESSNESS AT REST, FEVER, COUGH	N	N	N	Y	Y	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB	PNEUMONIA	76	90	60	11.9	1.33	100	138	4.19	ND	154	30	SEVERE	6.6	5.9	ABSENT	SEVERE MR, SEVERE TR	Y	N	Y	N	N		ABSENT	SEVERE	TVD	Y	Y	Y	N
58	NINGAPPA WARIMANI	867979	64	M	Y	N	N	N	BREATHLESSNESS	N	Y	N	N	N	IHD-LBBB		110	130	80	12.9	0.9	330	129	5.17	10	140	45	MILD	4.4	3.2	ABSENT	GRADE 1 MR, TRIVIAL AR	Y	N	N	N	N		ABSENT	NORMAL	SVD	Y	N	N	N
59	SHEKU RATHOD	894486	52	M	N	Y	Y	N	CHEST PAIN, PALPITATIONS, GIDDINESS	N	N	N	Y	Y	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		64	110	70	14.2	1.25	65	138	5.19	ND	156	30	SEVERE	7.1	6	ABSENT	GRADE 3 MR, GRADE 2 TR, GRADE 1 AR	Y	Y	Y	N	N		ABSENT	MILD	SVD	Y	N	N	N
60	SHANTA DESAI	916196	48	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	N	N	N	N	IHD- LBBB		84	130	70	11.9	0.84	109	134	3.93	ND	122	60	ABSENT	4	2.7	PRESENT	GRADE 1 MR, GRADE 1 TR	Y	N	Y	N	N		TYPE 1	MILD	SVD	Y	N	N	N
61	SAVAKKA KADAKAL	897617	64	F	Y	N	N	N	BREATHLESSNESS	N	Y	N	N	N	IHD-LBBB		80	100	70	12.7	0.43	118	138	4.26	10.2	150	35	MODERATE	5.8	4.6	ABSENT	GRADE 3 MR, GRADE 1 TR, GRADE 1 AR	Y	Y	Y	N	N		TYPE 1	MILD	DVD	Y	N	Y	N
62	PARVATEVVA CHIKKANNAVAR	916009	57	F	Y	N	N	N	BREATHLESSNESS	N	N	N	N	N	DILATED CARDIOMYOPATHY WITH LBBB		98	120	80	10.9	0.78	96	142	4.22	ND	162	30	SEVERE	6.8	5.8	ABSENT	GRADE 3 MR, GRADE 2 TR, TRIVIAL AR	Y	N	Y	N	N		ABSENT	MILD	ND				
63	RAGHAVENDRA HAJIRE	935315	38	M	Y	N	N	N	BREATHLESSNESS	N	N	N	Y	N	MITRAL VALVE PROLAPSE WITH LBBB		72	130	80	11.2	1.03	88	139	3.88	ND	132	55	MILD	3.9	2.7	ABSENT	GRADE 2 MR, MVP	Y	N	N	N	N	MVP	ABSENT	NORMAL	NORMAL				
64	IRAVVA KANKER	933311	65	M	N	Y	Y	N	CHEST PAIN, SYNCOPE		N	N	Y	N	IHD-LBBB		70	140	90	9.5	0.8	86	141	4.38	ND	144	50	MILD	4.4	3.3	ABSENT	GRADE 1 MR, GRADE 1 TR	Y	N	Y	N	N		ABSENT	MILD	DVD	Y	N	Y	N
65	YALLAVVA DIVANADAR	932140	70	F	Y	N	N	N	BREATHLESSNESS	N	N	COPD	N	N	LBBB WITH LV DYSFUNCTION		100	130	80	10.9	0.78	107	138	5.1	ND	148	45	MILD	4.5	3.6	ABSENT	GRADE 1 MR, GRADE 1 TR	Y	N	Y	N	N		ABSENT	MILD	NORMAL				
66	CHANDRAKANT HABSE	956848	67	M	Y	N	N	LOWER LIMB SWELLING	BREATHLESSNESS, LOWER LIMB SWEELING	Y	Y	CKD	Y	N	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		104	110	70	11.4	3.15	136	131	4.31	6.9	158	30	MODERATE	6.6	5.5	PRESENT	GRADE 2 MR, GRADE 2 TR, GRADE 1 AR	Y	Y	Y	N	N		TYPE 1	MODERATE	ND				
67	NINGAPPA HEGADIKATTI	953398	68	M	N	Y	N	N	CHEST PAIN	Y	Y	CVA	Y	Y	IHD- LBBB		80	140	80	12.6	1.11	184	141	4.31	7.1	130	60	ABSENT	4.2	3.2	PRESENT	GRADE 1 MR	Y	N	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	LEFT MAIN
68	GURUPAD PATIL	950831	65	M	Y	N	Y	COUGH	BREATHLESSNESS, GIDDINESS, COUGH WITH EXPECTORATION	Y	N	EPILEPSY, PARKINSONISM	Y	N	IHD- LBBB		86	160	90	12.5	0.94	102	142	3.64	ND	152	40	MODERATE	5	4	ABSENT	GRADE 2 MR, GRADE 1 AR, GRADE 1 TR	Y	Y	Y	N	N		ABSENT	MILD	SVD	N	N	Y	N
69	SUSHILA KUDACHE	951803	56	F	Y	N	N	N	BREATHLESSNESS	Y	Y	N	N	N	IHD-LBBB		80	140	80	9.1	0.56	195	136	4.84	7.2	124	60	ABSENT	4.2	2.9	PRESENT	ABSENT	N	N	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
70	SHAMRAO DORGUDE	951743	86	M	Y	N	Y	HEMOPTYSIS	BREATHLESSNESS, GIDDINESS, COUGH WITH EXPECTORATION	Y	N	CKD	Y	N	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		54	160	100	12.5	1.83	111	129	8.06	ND	168	25	SEVERE	6.5	5.3	ABSENT	GRADE 2 MR, GRADE 1 TR, TRIVIAL AR	Y	N	Y	N	N		ABSENT	MILD	ND				
71	SIDDAPPA NAUKUDIKAR	953172	63	M	Y	Y	N	N	BREATHLESSNESS, CHEST PAIN	Y	N	N	Y	N	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		98	100	60	11.3	1.34	132	139	4.42	6	166	30	SEVERE	6.4	5.5	ABSENT	GRADE 2 MR, TRIVIAL TR, TRIVIAL AR	Y	N	N	N	N		ABSENT	SEVERE	DVD	Y	N	Y	N
72	SATTEVVA BADAKAPPAGAL	953032	65	F	Y	N	N	EPISTAXIS	BREATHLESSNESS	Y	N	N	Y	N	IHD- LBBB		98	160	90	12	0.68	90	138	3.91	ND	142	45	MILD	4.4	3.5	PRESENT	GRADE 1 MR, GRADE 1 TR	Y	N	Y	N	N		TYPE 1	MILD	DVD	Y	Y	N	N
73	LAXMIBAI PATIL	945631	64	F	Y	N	N	N	BREATHLESSNESS	Y	Y	N	N	N	ISOLATED LBBB		72	120	80	11.6	0.72	173	142	3.81	8.2	126	60	ABSENT	4.2	3	PRESENT	TRIVIAL MR, TRIVIAL AR	N	N	N	N	N		TYPE 1	NORMAL	NORMAL				
74	LATA KILEKAR	937184	75	F	Y	N	N	N	BREATHLESSNESS	Y	Y	N	N	N	IHD- LBBB		80	130	80	8.9	0.97	99	138	4.06	9.2	124	60	ABSENT	4.1	3.5	ABSENT	ABSENT	N	N	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
75	MALLAPPA SOMANNAVAR	941618	86	M	Y	N	N	COUGH WITH EXPECTORATION	BREATHLESSNESS, COUGH WITH EXPECTORATION	Y	Y	N	Y	N	IHD- LBBB	PNEUMONIA	108	90	50	13.3	1.49	174	140	4.37	6.1	152	35	MODERATE	5.3	4.3	ABSENT	GRADE 1 MR, GRADE 2 TR	Y	N	Y	N	N		ABSENT	MODERATE	ND				
76	ANANDI PATIL	941233	55	F	Y	N	N	COUGH WITH EXPECTORATION	BREATHLESSNESS, COUGH WITH EXPECTORATION	Y	Y	N	N	N	IHD- LBBB		106	140	80	9.8	0.83	136	141	4.15	7.2	152	35	MODERATE	5.2	4.1	ABSENT	SEVERE MR, GRADE 2 TR, TRIVIAL AR	Y	N	Y	N	N		ABSENT	MODERATE	DVD	Y	Y	N	N
77	CHANDRASHEKHAR JAGAJAMPI	940903	76	M	Y	N	Y	LOOSE STOOLS, VOMITTING, DECREASED URINE OUTPUT	BREATHLESSNESS, GIDDINESS, LOOSE STOOLS, VOMITTING, DECREASED URINE OUTPUT	Y	Y	N	N	N	ISOLATED LBBB	ACUTE GE	50	130	80	10.1	2.35	295	138	5.28	6.4	128	60	ABSENT	4.2	3	PRESENT	GRADE 1 MR, GRADE 1 TR	Y	N	Y	N	N		TYPE 1	MILD	NORMAL				
78	RAVINDRA SHETTI	941981	84	M	N	N	Y	N	GIDDINESS	Y	Y	N	Y	N	ISOLATED LBBB		74	160	100	13.1	1.18	179	132	4.92	9.7	132	60	ABSENT	4.2	2.9	PRESENT	TRIVIAL MR, TRIVIAL TR	N	N	N	N	N		TYPE 1	NORMAL	NORMAL				
79	RUDRAPPA TIGADI	941894	69	M	Y	Y	Y	N	BREATHLESSNESS, CHEST PAIN, GIDDINESS	Y	Y	N	Y	N	DILATED CARDIOMYOPATHY WITH LBBB		104	130	90	8.5	0.91	224	134	4.72	9.1	152	30	SEVERE	6.3	5.4	ABSENT	GRADE 2 MR, GRADE 3 TR, TRIVIAL AR	Y	N	Y	N	N		ABSENT	SEVERE	ND				
80	SAVITRI PATIL	943872	70	F	Y	N	N	N	BREATHLESSNESS	Y	N	N	N	N	IHD-LBBB		76	130	80	11.4	1.1	111	134	4.14	ND	128	60	ABSENT	4.3	3.3	PRESENT	GRADE 1 TR	N	N	Y	N	N		TYPE 1	MILD	SVD	Y	N	N	N
81	SURENDRA WALLVEKAR	957607	67	M	Y	N	N	B/L LOWER LIMB SWELLING	BREATHLESSNESS, LOWER LIMB SWELLING	N	Y	N	Y	Y	ISCHEMIC DILATED CARDIOMYOPATHY WITH LBBB		98	110	70	15.3	0.96	142	128	4.62	7.8	172	25	SEVERE	6.5	5	ABSENT	GRADE 3 MR, GRADE 3 TR, GRADE 2 AR	Y	Y	Y	N	N		ABSENT	SEVERE	TVD	Y	Y	Y	N
82	SHAHIDA NAIK	920074	54	F	Y	N	N	N	BREATHLESSNESS	Y	Y	CVA	N	N	IHD- LBBB		86	110	70	12.7	1.91	65	142	4.36	9.7	140	50	MILD	4.3	3.5	PRESENT	GRADE 1 MR, TRIVIAL TR, TRIVIAL AR	Y	N	N	N	N		TYPE 1	MILD	TVD	Y	Y	Y	LEFT MAIN
83	APPASAHEB VAIRAGI	957081	67	M	Y	N	N	FEVER, COUGH	BREATHLESSNESS, COUGH WITH FEVER	Y	Y	COPD	Y	Y	IHD- LBBB		78	120	70	12.2	1.06	235	119	4.87	7.9	142	50	MILD	4.5	3.3	ABSENT	TRIVIAL MR, TRIVIAL TR, TRIVIAL AR	N	N	N	N	N		ABSENT	NORMAL	DVD	Y	N	Y	N
84	AKKAVVA NAVI	933308	55	F	Y	N	N	N	BREATH;ESSNESS	Y	Y	N	N	N	IHD- LBBB		80	130	80	10.9	0.54	264	142	4.02	ND	146	40	MODERATE	4.6	3.6	ABSENT	GRADE 1 MR, TRIVIAL AR	Y	N	N	N	N		ABSENT	NORMAL	SVD	Y	N	N	N

85	MALLAVVA HALYAL	920166	75	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	N	N	N	N	N	IHD-LBBB		120	130	80	12.5	0.8	93	142	3.62	ND	148	40	MODERATE	5.3	4.4	ABSENT	GRADE 2 MR	Y	N	N	N	N		ABSENT	NORMAL	DVD	Y	Y	N	N
86	MAHESH DESAI	954122	56	M	N	N	N	HEADACHE	HEADACHE	Y	N	N	Y	Y	ISOLATED LBBB		70	170	100	13.1	0.82	135	139	3.71	ND	124	60	ABSENT	4.1	3.2	ABSENT	TRIVIAL AR	N	N	N	N	N		TYPE 1	MILD	NORMAL				
87	GULSHANBEE MUJAWAR	744941	68	F	Y	N	N	N	BREATHLESSNESS	Y	N	RVD POSITIVE	Y	N	IHD-LBBB		94	130	80	10.7	0.78	134	131	3.5	ND	126	60	ABSENT	3.8	2.6	PRESENT	TRIVIAL AR	N	N	N	N	N		TYPE 1	NORMAL	DVD	Y	Y	N	N
88	HOUSABAI KADOLKAR	757649	65	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	N	N	N	N	IHD-LBBB	ACUTE KIDNEY INJURY	72	140	80	11	3.34	102	124	3.33	6.5	148	45	MILD	4.8	3.8	PRESENT	TRIVIAL MR	N	N	N	N	N		TYPE 1	NORMAL	DVD	Y	N	Y	N
89	SHRINIVAS DESHPANDE	765433	69	M	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	Y	N	Y	N	IHD-LBBB WITH IWMI		120	160	90	14.3	0.68	258	130	3.94	10.4	156	35	MODERATE	5.6	4.4	ABSENT	GRADE 2 MR, TRIVIAL AR	Y	N	N	N	N		ABSENT	NORMAL	DVD	Y	N	Y	N
90	SUDHABAI	741803	63	F	Y	N	N	N	BREATHLESSNESS	N	N	HYPOTHYROIDIS M	N	N	IHD- LBBB		74	140	90	11.9	0.76	133	130	4.85	ND	124	60	ABSENT	4.1	3.2	ABSENT	GRADE 1 AR	N	Y	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
91	ADAVAYYA VIBHUTI	752323	73	M	Y	N	N	N	BREATHLESSNESS	N	Y	N	Y	Y	IHD-LBBB		80	130	80	15.5	0.77	104	135	4.46	7.2	126	60	ABSENT	4	3.2	ABSENT	GRADE 1 AR	N	Y	N	N	N		TYPE 1	NORMAL	DVD	Y	N	Y	N
92	BALASAHEB KOKITKAR	951539	74	M	Y	N	N	N	BREATHLESSNESS	Y	Y	N	Y	N	ISCHEMIC DILATED CARDIOMYOPATHYWITH LBBB		114	90	50	10.8	1.4	237	139	4.54	8.6	172	25	SEVERE	6.9	5	ABSENT	GRADE 1 MR, GRADE 1 TR, TRIVIAL AR	Y	N	Y	N	N		ABSENT	MILD	TVD	Y	Y	Y	N
93	MEHABOOBI	946479	65	F	Y	N	N	N	BREATHLESSNESS	Y	Y	N	N	N	IHD-LBBB	ACUTE KIDNEY INJURY	130	140	80	11.8	1.76	525	134	4.97	12.9	144	45	MILD	4.8	3.3	ABSENT	GRADE 3 MR, GRADE 3 TR, TRIVIAL AR	Y	N	Y	N	N		TYPE 1	MODERATE	DVD	Y	N	Y	N
94	YASHODA	963582	60	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	N	Y	N	Y	N	IHD-LBBB		82	120	80	12.1	0.8	194	139	3.9	ND	142	45	MILD	4.5	3.4	ABSENT	TRIVIAL MR	N	N	N	N	N		TYPE 1	MILD	SVD	Y	N	N	N
95	GANGUBAI NAYAK	936281	60	F	Y	Y	N	N	BREATHLESSNESS, CHEST PAIN ON EXERTION	Y	Y	N	Y	N	IHD-LBBB		76	130	80	10.8	0.69	229	140	4.29	11.6	152	35	MODERATE	5.4	4.6	ABSENT	GRADE 1 MR, TRIVIAL AR, TRIVIAL TR	Y	N	N	N	N		ABSENT	NORMAL	DVD	Y	Y	N	N
96	JYOTI DUMAL	931989	50	F	Y	N	N	COUGH WITH EXPECTORATION	BREATHLESSNESS ON EXERTION, COUGH WITH EXPECTORATION	N	N	N	N	N	DILATED CARDIOMYOPATHY WITH LBBB	HYPOXIC ISCHEMIC ENCEPHALOPATHY	130	100	60	12.5	1.35	109	146	4.36	ND	162	30	SEVERE	6.2	5	ABSENT	SEVERE MR, GRADE 3 TR	Y	N	Y	N	N		ABSENT	SEVERE	ND				
97	MAHABOOBI DANGE	931674	72	M	Y	N	N	B/L LOWER LIMB SWELLING	BREATHLESSNESS, B/L LOWER LIMB SWELLING	Y	Y	ANEMIA	Y	N	DILATED CARDIOMYOPATHY WITH LBBB		114	170	100	12.1	0.89	298	128	4.14	7.1	154	35	MODERATE	5.4	4.2	PRESENT	SEVERE MR, GARDE 1 TR	Y	N	Y	N	N		TYPE 1	MILD	ND				
98	SHASHIKANTH GUNDABALLE	927188	69	M	N	Y	N	N	CHEST PAIN ON EXERTION	Y	Y	N	Y	N	IHD-LBBB		80	150	70	9.6	0.84	176	137	4.34	7.1	134	50	MILD	4.6	3.6	PRESENT	TRIVIAL MR, TRIVIAL TR	N	N	N	N	N		TYPE 1	NORMAL	SVD	Y	N	N	N
99	SUMITRA SHIVANNAVAR	956401	65	F	Y	Y	N	N	CHEST PAIN, BREATHLESSNESS	Y	Y	N	N	N	IHD- LBBB		64	130	90	12.2	1.15	128	132	4.89	9.9	150	40	MODERATE	5	3.9	ABSENT	GRADE 2 MR, TRIVIAL TR, TRIVIAL AR	Y	N	N	N	N		ABSENT	NORMAL	SVD	Y	N	N	N
100	SRIKANTAYYA	962417	60	M	N	N	Y	ABDOMEN PAIN	GIDDINESS, ABDOMEN PAIN	Y	Y	N	Y	N	IHD-LBBB	PEPTIC ULCER DISEASE	62	120	80	11.8	1.5	174	124	3.2	ND	142	60	ABSENT	4	2.9	PRESENT	TRIVIAL MR, TRIVIAL TR	N	N	N	N	N		TYPE 1	MILD	SVD	Y	N	N	N

ANNEXURE-V

KEY TO MASTER CHART

AF	–	ATRIAL FIBRILLATION
AR	–	AORTIC REGURGITATION
AS	–	AORTIC STENOSIS
AVR	–	AORTIC VALVE REPLACEMENT
DBP	–	DIASTOLIC BLOOD PRESSURE
DVD	–	DOUBLE VESSEL DISEASE
ECHO	–	ECHOCARDIOGRAPHY
EF	–	EJECTION FRACTION
HB	–	HEMOGLOBIN
HBA1C	–	GLYCOSYLATED HEMOGLOBIN
IHD	–	ISCHEMIC HEART DISEASE
IWMI	–	INFERIOR WALL MYOCARDIAL INFARCTION
LBBB	–	LEFT BUNDLE BRANCH BLOCK
LV	–	LEFT VENTRICLE
LVEDd	–	LEFT VENTRICLE END DIASTOLIC DIAMETER
LVESd	–	LEFT VENTRICLE END SYSTOLIC DIAMETER

MR	–	MITRAL REGURGITATION
MS	–	MITRAL STENOSIS
N	–	NO
ND	–	NOT DONE
PAH	–	PULMONARY ARTERY HYPERTENSION
RBS	–	RANDOM BLOOD SUGAR
SBP	–	SYSTOLIC BLOOD PRESSURE
SVD	–	SINGLE VESSEL DISEASE
TR	–	TRICUSPID REGURGITATION
TVD	–	TRIPLE VESSEL DISEASE
Y	–	YES