A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENTS

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CHENNAI – 600 003

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENTS**" submitted by **Dr. R.NAGENDRAN** appearing for M.D. Branch I - General Medicine Degree examination in MAY-2018 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfilment of regulations of the TamilNadu Dr. M.G.R. Medical University, Chennai. I forward this to the TamilNadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENTS"** is done by me at Madras Medical College & Rajiv Gandhi Govt. General Hospital, Chennai during 2017 under the guidance and supervision of **Prof.Dr.G.SUNDARAMURTHY., M.D.** The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

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ABBREVIATIONS

AR	-	Aortic Regurgitation
AS	-	Aortic Stenosis
ASMI	-	Antero Septal MI
AV block	-	Atrioventricular Block
AV node	-	Atrio Ventricular Node
CAD	-	Coronary Artery Disease
CRT	-	Cardiac Resynchronisation Therapy
DCM	-	Dilated Cardiomyopathy
ECG	-	Electrocardiogram
ECHO	-	Echocardiography
LAD Artery	-	Left Anterior Descending Artery
LAF	-	Left Anterior Fascicular Block
LBBB	-	Left Bundle Branch Block
LPFB	-	Left Posterior Fascicular Block
LVH	-	Left Ventricular Hypertrophy
MI	-	Myocardial Infarction

MR	-	Mitral Regurgitation
RBBB	-	Right Bundle Branch Block
RHD	-	Rheumatic Heart Disease
RVH	-	Right Ventricular Hypertrophy
SA node	-	Sino Atrial Node
STEMI	-	ST Elevation Myocardial Infarction
VT	-	Ventricular Tachycardia
WMA	-	Wall Motion Abnormality

CONTENTS

INTRODUCTION

INTRODUCTION

Left bundle branch block is an electrocardiographic diagnosis that is significantly associated with a higher than normal risk of morbidity and mortality. 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 dilated non ischemic cardiomyopathy at the time of diagnosis of LBBB. It can also occur as an isolated abnormality in asymptomatic patients. 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.

The abnormal ventricular activation pattern of LBBB itself induces further hemodynamic changes that are superimposed on the abnormalities caused by the underlying cardiac disease process. The pattern of ventricular activation 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 remodelling; as well as functional mitral valve dysfunction with mitral regurgitation. As a result, cardiac efficiency is further reduced. Severe left ventricular dysfunction is common, with a delay of more than 60 milliseconds between septal and lateral wall contraction with QRS durations of 120 to 150 milliseconds.

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. Longer the QRS duration, worser the prognosis.

Among patients with coronary artery disease, including acute STEMI, the presence of LBBB correlates with more extensive disease, more severe left ventricular dysfunction and often reduced survival rates.

LBBB with associated left or right axis deviation presents with more severe disease manifestations. Left axis deviation is associated with more severe conduction system disease involving the fascicles and the main left bundle. Right axis deviation suggests dilated cardiomyopathy with biventricular enlargement.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

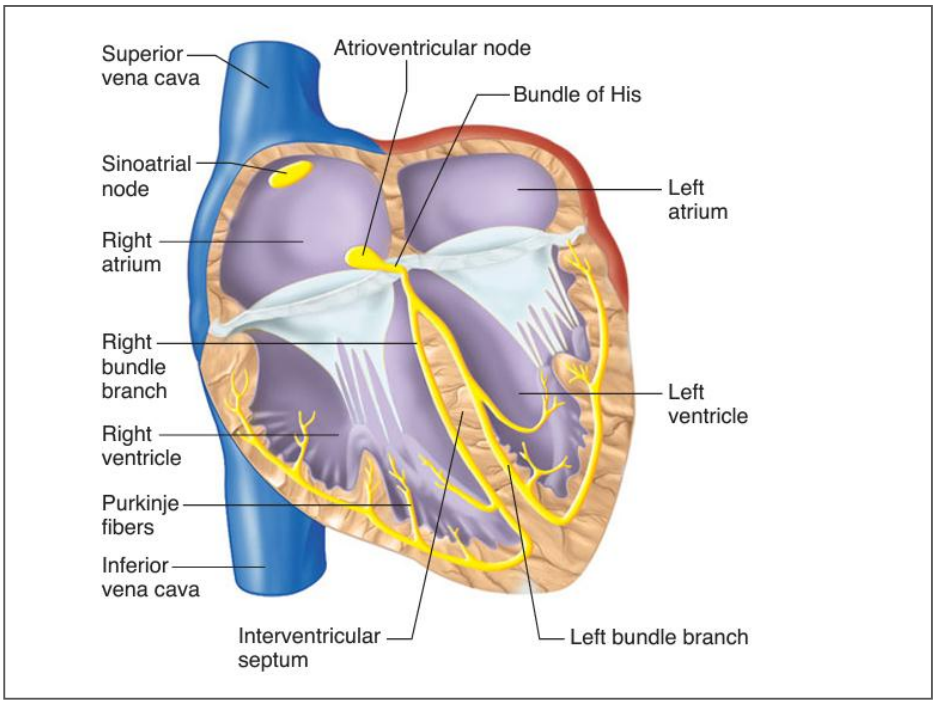
To study the clinical and echocardiographic profile in left bundle branch block patients in a tertiary care institute

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Anatomy of the cardiac conduction system

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



Sinus node: The sinus node is the pacemaker of the heart and the cardiac impulse originates here. It is located high within the right atrium in the superior and lateral border. The most cranial part starts from the

epicardium at the junction of superior vena cava with right atrium. Its most caudal portion is located subendocardially. The sinus node contains cardiac pacemaker cells that are distributed widely throughout its entire length. These cells have properties of automaticity and are capable of spontaneous discharges. The various parts of the conduction system as well as parts of the myocardium are also capable of discharging spontaneously. However, the SA node normally discharges rapidly, with depolarization spreading from it to the other regions before they discharge spontaneously. The sinus node therefore is regarded as the pacemaker of the heart. The sinus node is supplied by the right coronary artery 60% to 65% of the time through its sinus node branch. In the rest, the vascular supply originates from left circumflex coronary artery.

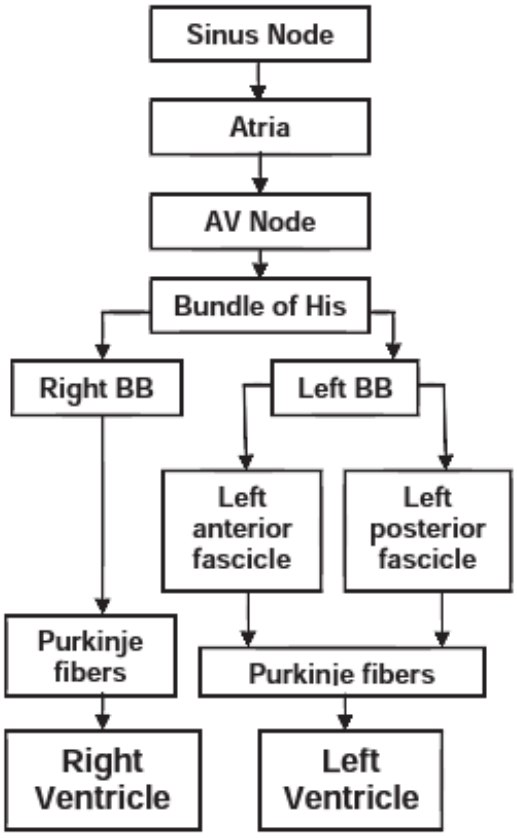
Internodal tracts: Three internodal tracts with Purkinje-type fibers connect the SA node to the AV node: the anterior, posterior, and middle internodal tracts. The significance of these tracts is uncertain because the sinus impulse passes through the atrial musculature to reach the AV node.

The AV node: The AV node is the only pathway through which the sinus impulse can pass to reach the ventricles The AV node is smaller than the sinus node and is located at the floor of the right atrium, anterior to the entrance of the coronary sinus to the lower right atrium and just above the

insertion of the septal leaflet of the tricuspid valve. The AV node consists of three portions with distinct properties: the upper, middle, and lower regions. The upper *atrionodal (AN)* region connects the atria to the middle portion, *nodal (N)* region. The lower *nodo-His (NH)* region connects with the bundle of His. The AV conduction delay occurs mainly in the middle region. It also has no automatic properties in contrast to the upper and lower portion, which contain cells with properties of automaticity. The AV node is supplied by AV nodal artery, a branch of the right coronary artery in 90% of patients. In the remaining 10%, the AV node artery arises from the left circumflex coronary artery.

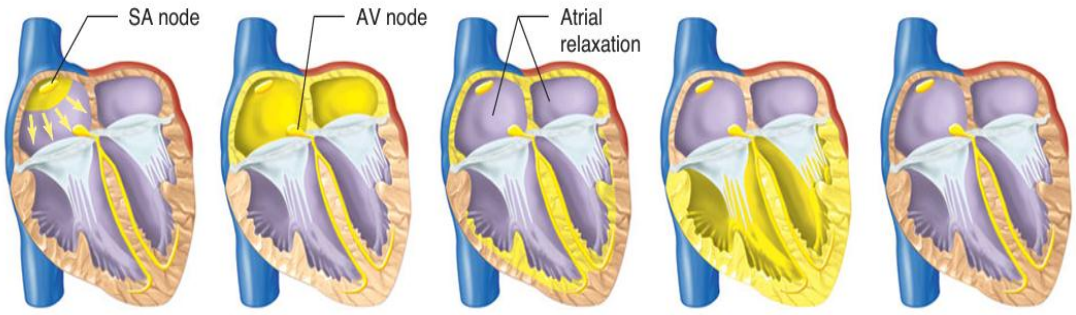
The Bundle of His-Purkinje system: The AV node continues into the His bundle, a short structure that immediately divides into the right and the left bundle branches. The right bundle branch, a direct continuation of the bundle of His is long and thin and continues down the right side of the interventricular septum toward the apex of right ventricle and base of the anterior papillary muscle. 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*. These fascicles are interconnected with each other. The anterosuperior division ramifies anteriorly and superiorly

through the subendocardium of the left lateral wall. The posteroinferior division ramifies posteriorly and inferiorly through the subendocardium 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 right bundle branch and the fascicles end in a network of Purkinje fibers that spreads just under the endocardial surface of both ventricles. The blood supply of the His bundle comes from both anterior and posterior descending coronary arteries through their septal branches.



SPREAD OF CARDIAC EXCITATION

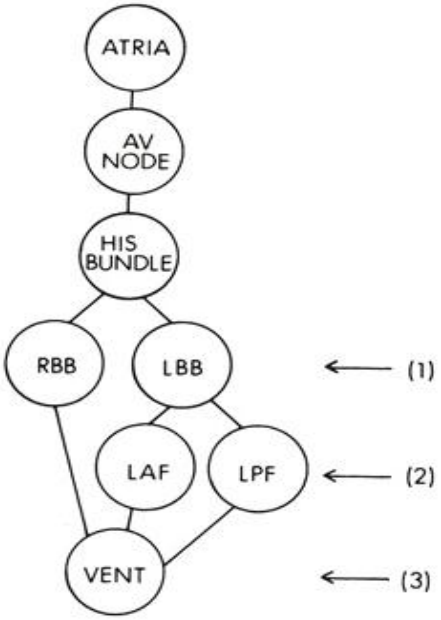
Depolarization initiated in the SA node spreads radially through the atria depolarising it, then converges on the AV node. Atrial depolarization is complete in about 0.1 s. AV nodal delay of about 0.1 s occurs before excitation spreads to the ventricles. This delay is shortened by stimulation of the sympathetic nerves and lengthened by stimulation of the vagi. The impulse then passes through the bundle of His pass and its branches to reach the purkinje fibers. The purkinje fibers conduct the impulse directly to the myocardium causing synchronous depolarisation of both the ventricles. From the top of the septum, the wave of depolarization spreads in the Purkinje fibers to all parts of the ventricles in 0.08–0.1 s. Ventricular activation then occurs in three stages- depolarization of the ventricular septum, depolarization of the free walls of both ventricles, and depolarization of the posterobasal wall of the left ventricle and the posterobasal septum.



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

Intraventricular conduction defects

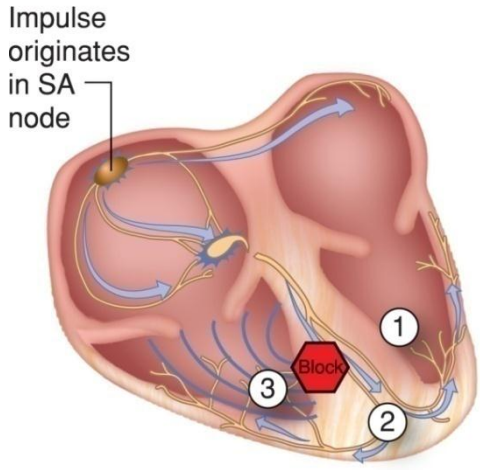
Any conduction disturbances through the intraventricular conduction pathways can occur at all the levels from the His bundle to the myocardium. These conduction disturbances may affect a single fascicle only causing unifascicular block or it may affect two or more fascicles simultaneously resulting in bifascicular and trifascicular blocks. Isolated RBBB, LAFB and LPFB are considered unifascicular blocks. Complete LBBB and RBBB with either LAFB or LPFB are bifascicular blocks. RBBB with both LAFB and LPFB and alternating bundle branch 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.

Right Bundle Branch Block

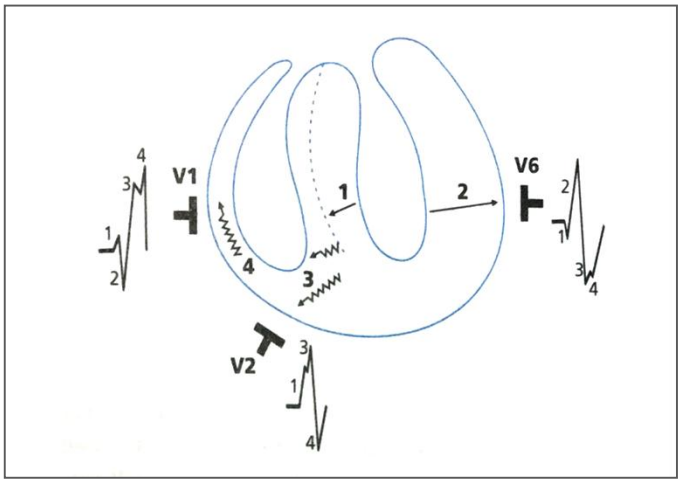
Here, there is a block in conduction of impulse within the right bundle branch causing delayed right ventricular depolarisation. Activation of right ventricle is through the left bundle branch by muscle to muscle conduction. This results in sequential activation of ventricles rather than concurrent activation.



Mechanism

Changes in QRS complex: With complete RBBB, ventricular activation begins in the left lower third of interventricular septum and spreads through the septum transversely from left to right- vector 1. This left to right vector is not however opposed by the normal right to left septal vector which originates from the right bundle branch in the right side of the interventricular septum. And since the vector 1 from left to right septum is no longer opposed, it is therefore increased in magnitude

but minimally[1]. This results in slightly more prominent initial r wave in lead V2. Normal paraseptal activation, which occurs next, spreads transversely from endocardial to epicardial surface- vector 2b. This is followed by endocardial to epicardial activation of the left ventricular free wall- vector 2 which is directed to the left[2].



Activation of right side of the interventricular septum and the right ventricle and its free wall is effected by the activation front that arises in the left side of the interventricular septum. This crosses a physiological intraseptal barrier within the septum and is then conducted through ordinary myocardial tissue, instead of the specialised conducting tissue, within the right ventricular wall. The Purkinje system is responsible for the rapid transmission of the impulse from a central distributing point which is normally in the subendocardium and the synchronous activation of the ventricles[3]. Activation entering the system from another direction i.e. within the myocardium is also transmitted but in a relatively slow,

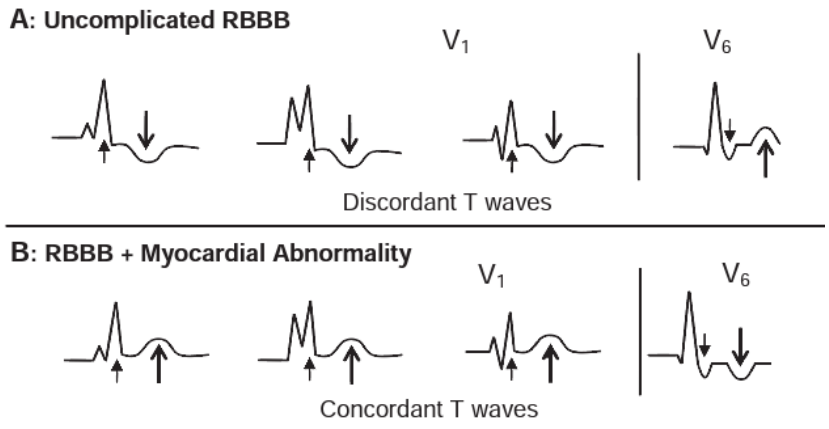
anomalous, ineffective and bizarre manner. This results in slow abnormal depolarisation of the right side of the interventricular septum, right paraseptal region and the right ventricle. This depolarisation is reflected electrocardiographically by large, slowly inscribed vectors- vector 3 and 4 which are directed anteriorly and to the right.

The right paraseptal vector 3 occurs simultaneously with vector 2- the vector of left free wall, but is opposite in direction to vector 2. This results in a marked diminution in the amplitude of the S wave in lead V1 which in advanced degrees of RBBB, may disappear completely. This also results in attenuation of the R wave in left oriented leads such as lead V5, V6. Unlike the effect on lead V1, however, the R wave in lead V1is only attenuated but not abolished completely. This is because lead V6, which is oriented to the lower free wall of the left ventricle, is influenced to a degree by the left paraseptal vector- vector 2b also. This also contributes to the positivity of the R wave in lead V6[4].

Activation of the free wall of the right ventricle is the last to occur. This results in a large unopposed vector- vector 4, which is directed anteriorly and to the right. This is reflected by a wide R' reflection in lead V1 along with a prominent slurred and delayed S wave in lead v6. The abnormal, slow, anomalous form of activation is responsible for the

marked notching and slurring of the terminal bizarre and wide deflections.

Changes in the ST segment and T waves: In uncomplicated RBBB, 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 repolarisation changes due to abnormal activation of the ventricles. In lead V1, the T waves are inverted with isoelectric or minimally depressed ST segment[5]. Similarly, in lead V6, the T waves are upright. 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.

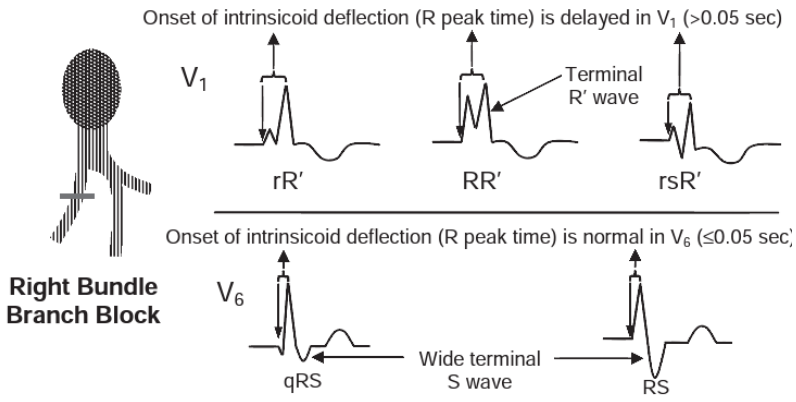


ECG manifestations of Complete RBBB

The ECG changes of RBBB are best seen in the precordial leads V1 and V6. The duration of QRS complexes is prolonged to 120 msec or more due to delayed and anomalous activation of left ventricle. The axis of QRS complexes usually remains normal and unchanged unless if there is a fascicular block or ventricular hypertrophy.

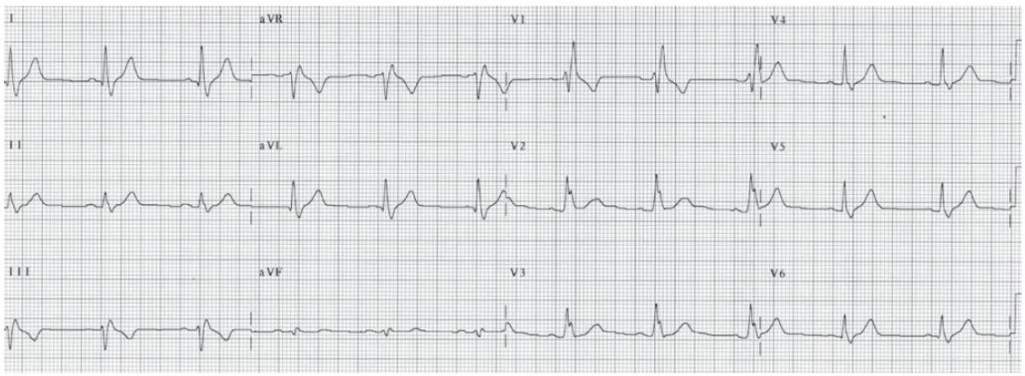
Leads oriented to the right ventricle reflect the following:

1. There is a small initial r wave due to depolarisation of the left side of the septum.
2. This is followed by an S or s wave, which is mainly due to depolarisation of left free wall.
3. There is a terminal bizarre and slurred R wave- R’ due to late and anomalous right septal and right free wall depolarisation.
4. The onset of intrinsicoid deflection in V1 may be delayed up to 50 msec or more.



Leads oriented to the left ventricle reflect the following:

1. There is a small initial q wave due to depolarisation of the left side of the septum.
2. There is a relatively tall R wave, which is mainly due to depolarisation of left free wall.

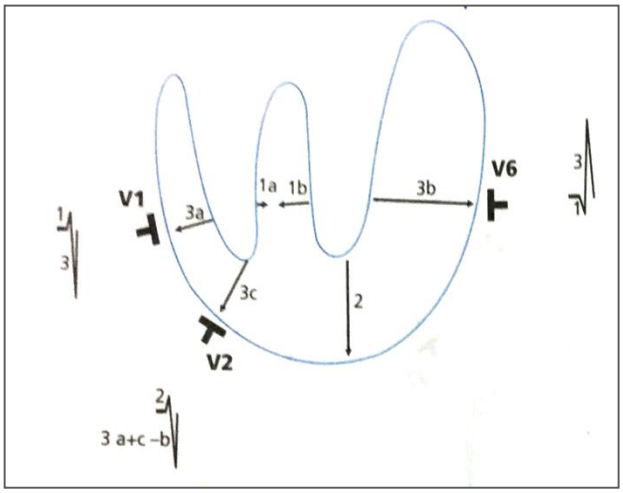


3. There is a terminal bizarre and slurred S wave, which is due to late and anomalous right septal and right free wall depolarisation.

Incomplete RBBB

Here, conduction through the right bundle branch is still possible but is delayed. A small delay within the right bundle branch causes delayed inscription of the right paraseptal vector- vector 3c. This vector now occurs synchronously with the free wall forces- vectors 3a and 3b[6]. Since vector 3c is directed slightly opposite to vector 3b, the magnitude of resultant vector 3 that is responsible for the S wave will be diminished

in lead V2. A slight delay in conduction will thus cause a diminution of S wave of lead V2. There should be a diminution of R wave in lead V6 also but not consistently. A possible reason is that lead V6 tends to be more obliquely oriented towards the left paraseptal vector as well as the left free wall vector- vector 2 and 3b. Therefore the influence of the left paraseptal vector will also contribute to the amplitude of R wave in this lead.



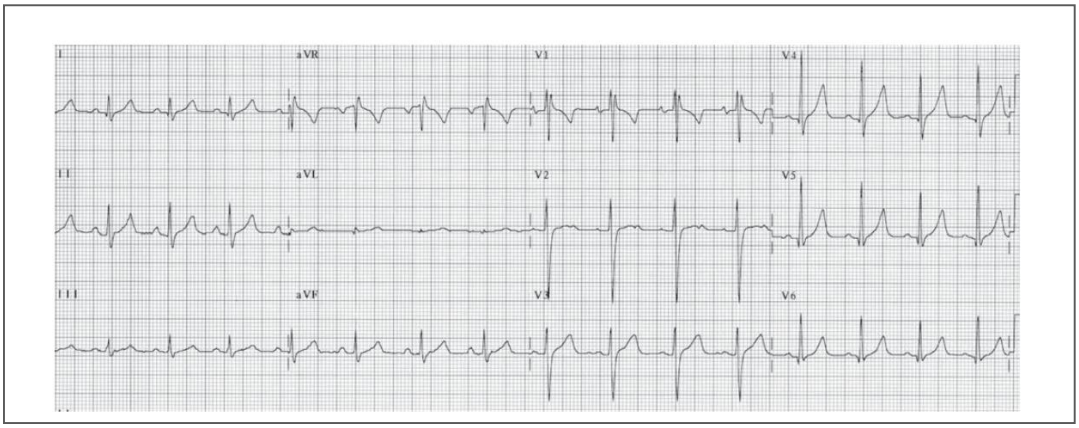
As the delay increases within the right bundle branch, activation of the free wall of the right ventricle will also be increasingly delayed. When the delay is of such a degree, that activation of the right ventricular free wall occurs after the activation of the left ventricular free wall, an r' deflection appears in the right oriented leads[7]. This is because right ventricular free wall activation is then no longer completely opposed by the left ventricular free wall activation.

The r’ deflection progressively gets taller as the delay increases since a larger part of right ventricular free wall activation will occur after left ventricular free wall activation. However the R’ deflection of maximal delay is not widened beyond 40 msecs. When the R’ deflection is more than 40msecs, the block is considered to be complete and conduction can no longer occur through the right bundle branch but through ordinary myocardium.

ECG manifestation of incomplete RBBB

The following sequence of progressively increasing changes manifests in electrocardiography:

1. There is diminution of S wave in lead V2. This is the earliest sign of incomplete RBBB and maybe the only sign.
2. This is followed by slurring of the upstroke of the S wave in lead V2. There is further diminution of S wave.
3. A small r’ deflection then appears in lead V2. This results in a rsr’ deflection. This is usually associated with further attenuation of S wave.
4. The amplitude of R’ deflection in lead V2 then increases and the S wave is further diminished. The configuration is that of an rsR’ pattern.



Anatomical length of Right bundle branch

The length of right bundle branch also plays a significant role in the genesis of RBBB. The normal right bundle branch measures 40 mm in length. The normal conduction velocity is 2 m/s. Thus, for each cm increase in the length of the right bundle branch, the conduction time increases by 5msec. Therefore to begin with, if uncomplicated intraventricular conduction results in QRS duration of 0.09 secs, a 4 cm increase in the length increases the duration to 0.11 s.[7] If there is a conduction delay in addition, the increase would be obviously greater. Therefore the diagnosis of incomplete RBBB depends mainly on morphology alone and not the duration.

Intermittent RBBB

RBBB may initially occur intermittently before it becomes fixed. When RBBB is intermittent, it is usually rate related. The block occurs

only when the heart rate increases above a certain threshold. When the heart rate slows down to baseline, normal conduction is restored. The presence of rate-related RBBB is often suspected when the wide QRS complexes normalize after the long compensatory pause of a premature ectopic impulse.[8]

Common errors in diagnosing RBBB

Ectopic ventricular impulses originating from the ventricles have wide QRS complexes. These impulses are wide since they do not follow the normal conduction system and spread through the ventricles by muscle cell to muscle conduction. RBBB may be frequently mistaken for these Ectopic ventricular impulses. In true RBBB, the impulse should originate from sinus node or it is supra ventricular[9]. Whereas in VT, the impulse originates from the left ventricle and spread to the right ventricle, causing the QRS complexes to have a RBBB pattern. This can be used to differentiate between RBBB and VT.

Clinical significance of RBBB

RBBB is a common finding in the general population without any evidence of structural heart disease. The common causes of RBBB include acute MI, cardiomyopathy, valvular heart disease especially

aortic stenosis, congenital heart disease such as tetralogy of Fallot and Ebstein's anomaly, sclerodegenerative changes of the conduction system and infiltrative diseases such as sarcoidosis and amyloidosis, Chagas disease. Since the right bundle branch is partially subendocardial in location, it is vulnerable to sudden and severe increases in right ventricular pressure. RBBB therefore can occur in the setting of acute pulmonary embolism or pulmonary hypertension and cor pulmonale. The Right bundle branch is also more prone for local injury and can occur as a complication of cardiac surgery or interventional procedures such as cardiac catheterization, percutaneous coronary intervention.

Auscultatory findings: The presence of RBBB will delay the closure of the pulmonary valve resulting in wide splitting of the second heart sound. It may be associated with delayed closure of the tricuspid valve resulting in wide splitting of the first heart sound.

RBBB with RVH: The diagnosis of right ventricular hypertrophy is more difficult with RBBB because of the prominent positive waves in lead V1. RVH is suggested, by the presence of an R wave >1.5 mV in lead V1 and a rightward shift of the mean QRS axis.[10]

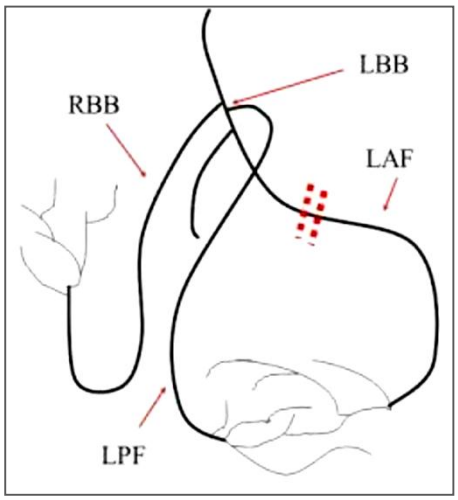
RBBB with acute MI: The presence of RBBB generally does not conceal the ECG changes associated with Q wave MI. Therefore changes

in the Q waves or QRS complexes can still be used for diagnosis. When STEMI is complicated by RBBB, the ST segments become concordant in relation to the terminal portion of the QRS complex. Similar concordant changes may be seen in leads II, III, and aVF in the presence of inferior MI.

Prognosis: In patients with no manifest cardiac disease, RBBB do not increase the risk of cardiac morbidity or mortality. Ventricular dyssynchrony, though present, is lesser than LBBB. However, when cardiac disease is present, the coexistence of RBBB suggests advanced cardiac disease and reduced long-term survival. RBBB and LAFB is a common combination because the right bundle branch and the left anterior fascicle are adjacent to each other, straddling both sides of the ventricular septum. Left anterior descending coronary artery supplies both these fascicles. Therefore a concurrent RBBB and LAFB can occur as a common complication of acute anteroseptal MI. When RBBB with LAFB is due to acute ASMI, the myocardial damage is usually extensive, resulting in a higher incidence of AV block, pump failure, and ventricular arrhythmias with a mortality rate more than 20%[11].

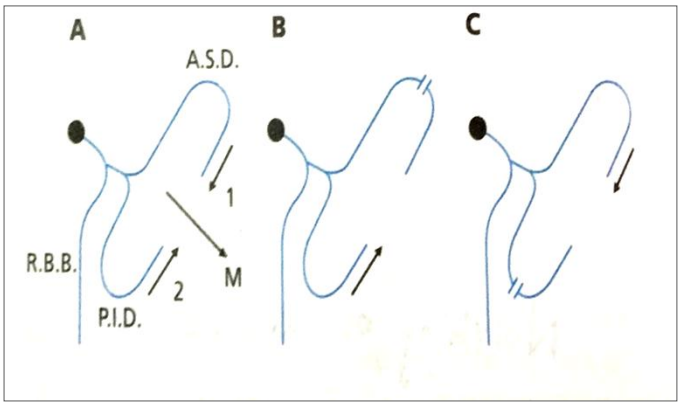
Left Anterior fascicular block

In the presence of LAFB, conduction is delayed or interrupted in the anterosuperior division of left bundle branch. When such interruption or delay occurs, activation proceeds entirely through the posteroinferior division of the Left bundle branch.



Mechanism

Changes in the QRS complex- The left anterior fascicle of the left bundle branch normally activate the anterior and superior portions of the left ventricle. With a block in the left anterior fascicles, the area supplied by it is the last to be activated. This causes the mean manifest frontal plane QRS axis to shift superiorly and to the left. The mean QRS axis is located within a range extending from -30^0 to -80^0 counter clockwise[29]. QRS axes which range from 0^0 to 30^0 reflect minor degrees of LAFB or incomplete LAFB.



A - Normal conduction

B - Left Anterior Fascicular Block

C - Left Posterior Fascicular block

In LAFB, the left ventricle is initially activated by the left posterior fascicle of the left bundle branch. Thus, the initial 20 msec QRS vector is directed inferiorly and to the right to the region of $+90^0$ to $+120^0$ on the frontal plane hexaxial system. This is reflected by 1.Prominent initial q waves in the standard lead I and lead aVL i.e. initial q waves in these leads become accentuated. 2. Prominent initial r waves in the standard leads II, III and lead aVF.

The area supplied by the anterior fascicle, that is the high anterolateral region, is the activated at the end. This results in a delay in the inscription of the intrinsicoid deflection in leads oriented to the high lateral aspect of the left ventricle, particularly lead aVL. The ventricular activation time may be delayed up to 45 ms or longer. This delay may also be reflected in lead aVR due to incorporation of a terminal r wave and, may at times be seen only in lead aVR. There may be slurring,

notching or irregularities of R wave in lead aVR and lead I as well as S waves in leads V5 and V6. There may be slurred terminal r or r' deflection in lead aVR[30].

The leftwards directed QRS vectors are frequently increased in magnitude. This is due to lack of opposing forces from the blocked anterosuperior division. This result in tall R waves in leads I and aVL and a deep S wave in lead III and lead II[31]. The QRS complex is not widened because of the presence of two overlapping sets of Purkinje fibres from the left anterior and posterior fascicles. Hence the left ventricle is activated by the intact left posterior fascicle and both the ventricles remain synchronously activated. There may be a slight increase in QRS duration, but, this is usually < 110 msec.

With the advent of LAFB, the QRS forces become more aligned with the frontal plane. The QRS deflections therefore become larger in the frontal plane leads than in the horizontal plane leads.

Changes in the ST segment and T waves- LAFB is associated with secondary changes of repolarisation that are secondary to abnormal intraventricular conduction and have no primary significance. These, in particular, affect the T wave. The T wave vector tends to deviate in the opposite direction to main QRS vector. As a result, inverted T waves are

seen in leads with dominant positive QRS complex- leads I and aVL. Leads with dominant negative QRS complex tend to have upright T waves.

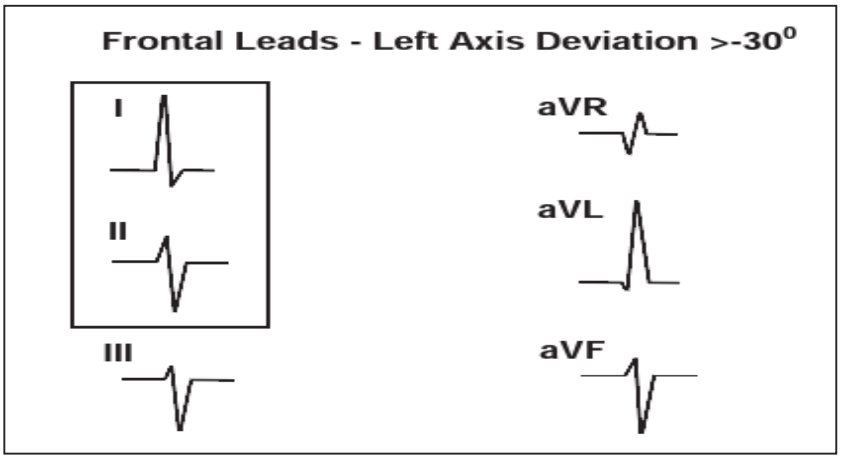
ECG manifestations of LAFB

LAFB results in the following basic disorders of intraventricular conduction

1. Left Axis Deviation of mean manifest frontal plane QRS axis.
2. Delay in the activation of the left ventricular free wall, particularly the high left anterolateral region, resulting in an increased ventricular activation time.
3. An increase in magnitude and change in direction of the initial 20 ms vector, which is directed inferiorly and commonly somewhat to right.
4. Secondary T wave repolarisation changes.

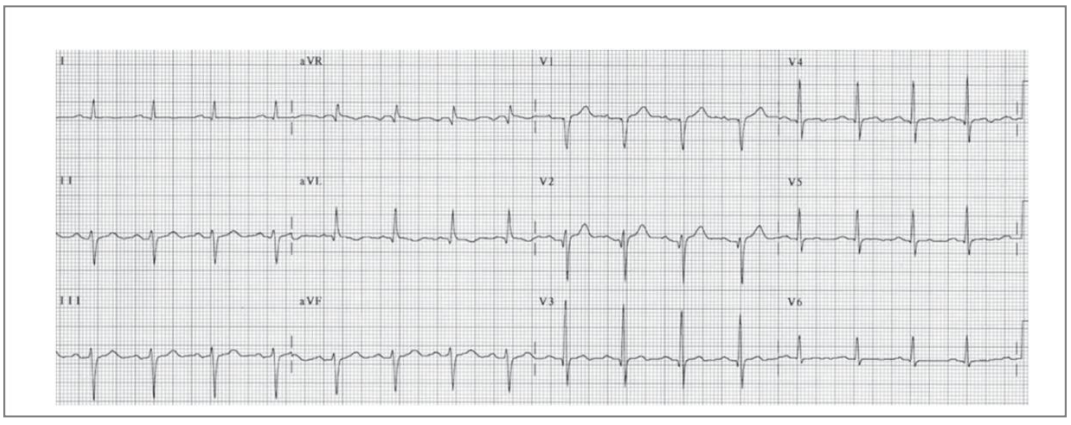
Effects on Standard lead II- Lead II reflects an rS complex with a relatively prominent initial r wave. This is an expression of prominent initial vector that is directed to right and inferiorly. Lead II does not show a terminal R wave. This is an important differentiating feature from LAD of Inferior wall MI, where lead II presents a qR complex[32].

Effects on Standard lead I and aVL- Leads I and aVL both reflect a initial q wave followed by a tall R wave. The small initial q wave may sometimes disappear in lead I. Lead aVL has a relatively prominent R wave that reflects the increased ventricular activation time.



Effects on Lead aVR- Lead aVR may reflect a slurred terminal r wave. This reflects the delayed activation of anterosuperior regions.

Effects on precordial leads V5and V6- 1.There is a tendency for the small initial septal q waves in V5 and V6 to disappear. This reflects the change in pattern of left ventricular activation from the normal initial left- right septal activation to



initial posterior- inferior ventricular wall activation. 2. Attenuation of the R waves in left oriented precordial leads, which reflects the superiorly directed dominant QRS vector. 3. Prominent S waves develop in the left precordial leads that also reflect the superiorly directed dominant QRS vector[33].

Clinical significance of LAFB

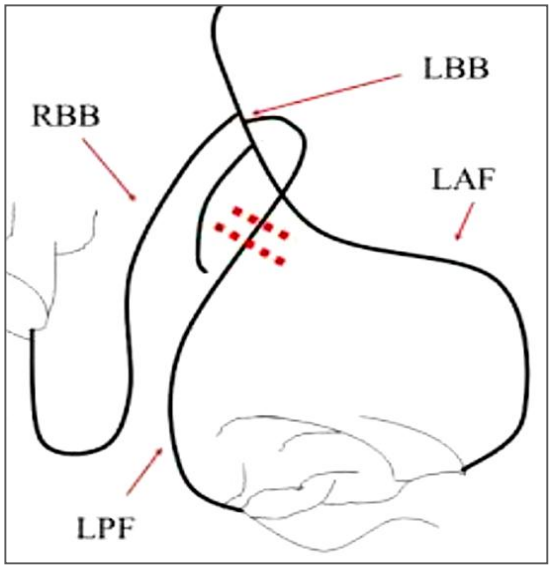
LAFB is the most common intraventricular conduction abnormality because of its structure and location. The left anterior fascicle is relatively long and thin, courses subendocardially in the direction of the outflow tract of left ventricle closer to the aortic valve, and therefore subjected to higher intraventricular pressure than the rest of the conduction system. LAFB is more commonly the result of hypertension, cardiomyopathy, ischemic heart disease, acute myocarditis, aortic valve disease and fibrosis of the conduction system[34].

Prognosis: Isolated LAFB in the absence of overt cardiac disease, as such, does not constitute a risk factor. Rather, it points to the possible presence, or the potential development of cardiac disease. When LAFB occurs with RBBB, it usually connotes an adverse prognosis and may precede complete AV block. When it occurs together with LBBB, all

aspects related to morbidity and mortality is far worse than LBBB with normal QRS axis.

Left Posterior Fascicular block

When conduction is interrupted in the posteroinferior division of left bundle branch, a rare occurrence, it results in Left Posterior Fascicular block. Activation then proceeds entirely through the anterosuperior division of the left bundle branch.



Mechanism

Changes in the QRS complex- The left posterior fascicle of the left bundle branch normally activate the posterior and inferior portions of the left ventricle. With a block in the left posterior fascicles, the area supplied by it is the last to be activated. This causes the mean manifest frontal plane QRS axis to shift downwards and to the right[35]. The mean QRS axis is

located within a range extending from $+90^0$ to $+120^0$ clockwise. In LPFB, the left ventricle is initially activated by the left anterior fascicle of the left bundle branch. Thus, the initial 20 msec QRS vector is directed superiorly and to the left to the region of -50^0. This result in 1.Prominent initial q waves in the standard lead II, III and lead aVF 2.Prominent initial r waves in the standard leads I. The area supplied by the posterior fascicle is activated at the end. This results in 1. Prominent S waves in standard lead I and aVL 2. Tall R waves in standard II, III, aVF.

The rightwards directed QRS vectors are frequently increased in magnitude. This is due to lack of opposing forces from the blocked posteroinferior division. The QRS complex is not widened because of the presence of two overlapping sets of Purkinje fibres from the left anterior and posterior fascicles[36]. Hence the left ventricle is activated by the intact left anterior fascicle and both the ventricles remain synchronously activated.

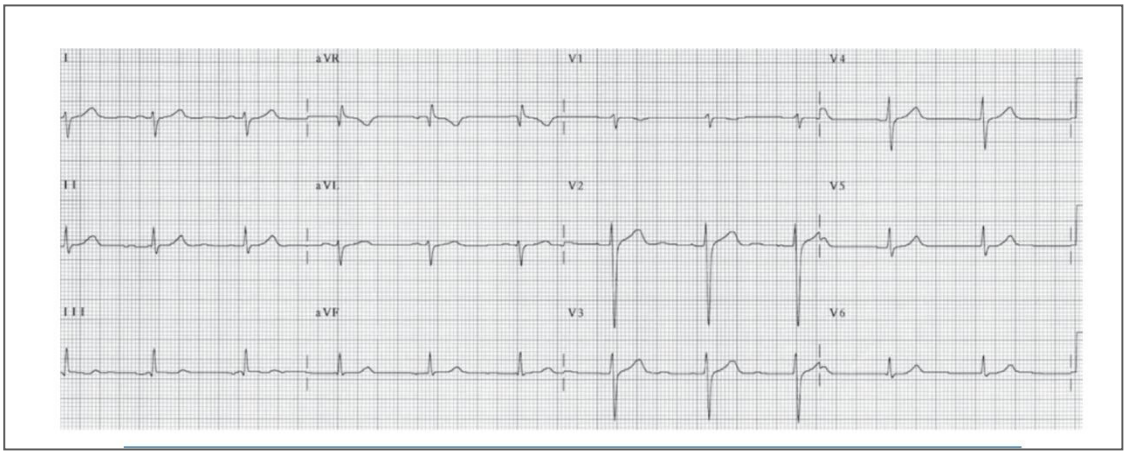
Changes in the ST segment and T waves- As with other intraventricular conduction disturbances, LPFB is associated with secondary changes of repolarisation that are secondary to abnormal intraventricular conduction and have no primary significance. These, in particular, affect the T wave. The T wave vector deviates in the opposite direction to main QRS vector. As a result, inverted T waves are seen in leads with dominant positive

QRS complex (leads II, III and aVF). Leads with dominant negative QRS complex (lead I) tend to have upright T waves.

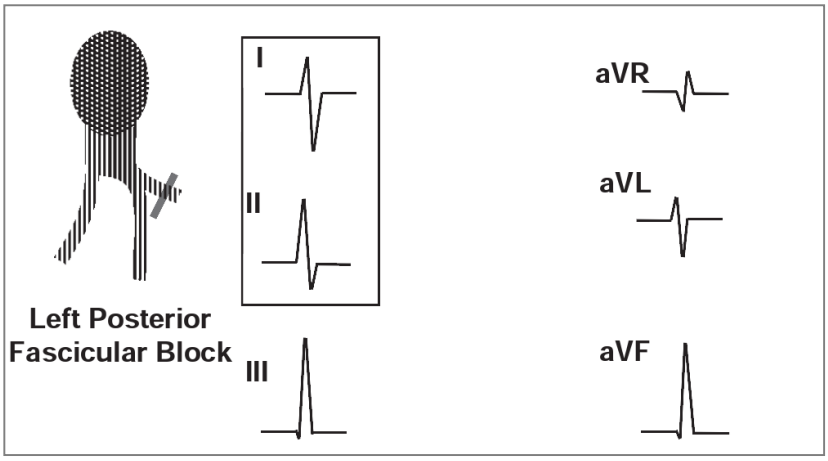
ECG manifestations of LPFB

LPFB results in the following basic disorders of intraventricular conduction

1. Right Axis Deviation of mean manifest frontal plane QRS axis.
2. An increase in magnitude and change in direction of the initial 20 ms vector, which is directed superiorly and commonly to left.
3. Secondary T wave repolarisation changes.



Effects on Standard lead I and aVL- Leads I and aVL both reflect an rS complex with a relatively prominent initial r wave. This is an expression of prominent initial vector that is directed to superiorly and to left. These leads do



not show a terminal R wave. This is an important differentiating feature from LAD of Inferior wall MI, where lead I present a qR complex.

Effects on Standard lead II, III and aVF- Inferior leads reflect a initial q wave followed by a tall R wave.

Clinical Significance

LPFB is the least common among all intraventricular conduction abnormalities because of its structure, location, and blood supply. The left posterior fascicle is short, broad and thick and courses along the inflow tract of the left ventricle before terminating in a network of Purkinje fibres[37]. It has a dual blood supply, originating from the septal perforating branches of the LAD anteriorly and from the septal perforating branches of posterior descending artery posteriorly. It is therefore much protected and subjected to less intraventricular pressure compared with the left anterior fascicle.

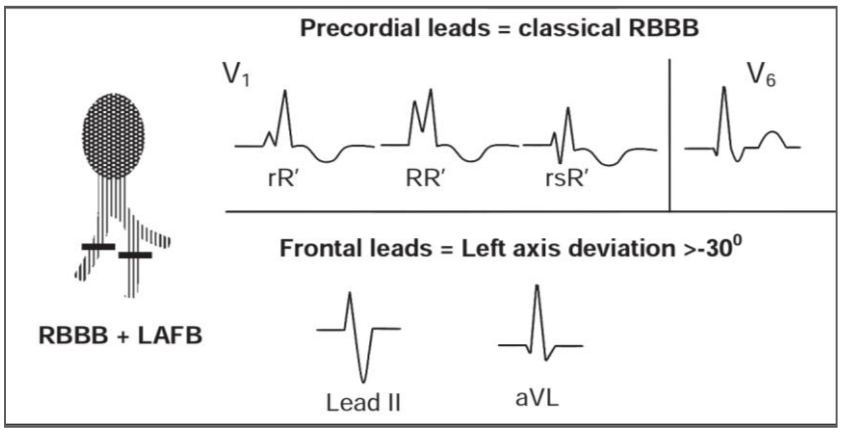
Since LPFB is relatively uncommon, it is a diagnosis of exclusion. Before the diagnosis of LPFB is considered, other common causes of right axis deviation should first be excluded[38]. The occurrence of isolated LPFB is very rare. LPFB is most commonly a result of coronary artery disease, hypertension, cardiomyopathy, acute myocarditis, Valvular heart disease especially aortic stenosis, and degenerative diseases of the conduction system.

Prognosis- Because the left posterior fascicle is the least vulnerable defect, LPFB seldom occurs independently and is frequently seen in combination with RBBB or with LAFB. LPFB, therefore, indicates a more advanced and more significant form of conduction abnormality than LAFB[39]. LPFB in combination with LAFB can result in left bundle branch block.

RBBB with Left Anterior Fascicular block

RBBB can occur in combination with a fascicular block. The sinus impulse is simultaneously interrupted at the right bundle branch as well as left anterior fascicular branch. Activation of the ventricles can occur only through the remaining intact left posterior fascicle. RBBB in combination with LAFB is an example of a bifascicular block.

ECG findings: The typical features of RBBB are seen in the chest leads and that of LAFB in the frontal plane leads.



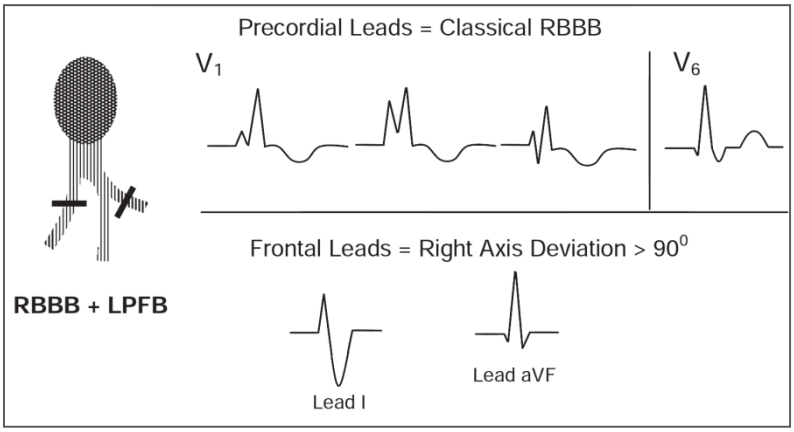
- Precordial leads: The QRS complexes are wide measuring 120 msecs or more. The characteristic rR' or rsR' pattern is present in lead V1, and wide S waves are present in lead V6.
- Limb leads: In the frontal plane, the axis of the QRS complex is -30^0 with rS in lead II and tall R wave in lead aVL.

RBBB with Left Posterior Fascicular block

In the presence of RBBB with LPFB, the sinus impulse is simultaneously interrupted at the right bundle branch as well as left posterior fascicular branch. Activation of the ventricles can occur only through the remaining intact left anterior fascicle.

ECG findings: The typical features of RBBB are seen in the chest leads and that of LPFB in the frontal plane leads.

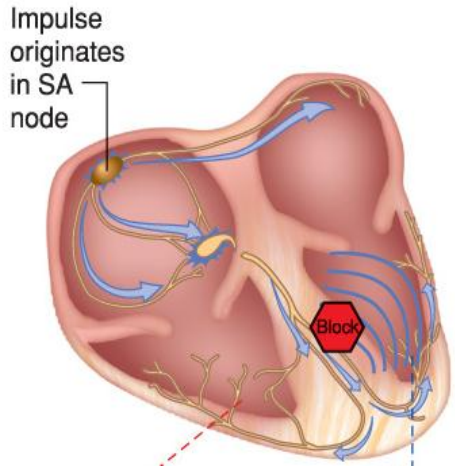
- Precordial leads: The QRS complexes are wide measuring 120 msecs or more. The characteristic rR' or rsR' pattern is present in lead V1 and wide S waves are present in lead V6.



- Limb leads: In the frontal plane, the axis of the QRS complex is $+90^0$ or more with rS in lead I and tall R wave in lead aVF.

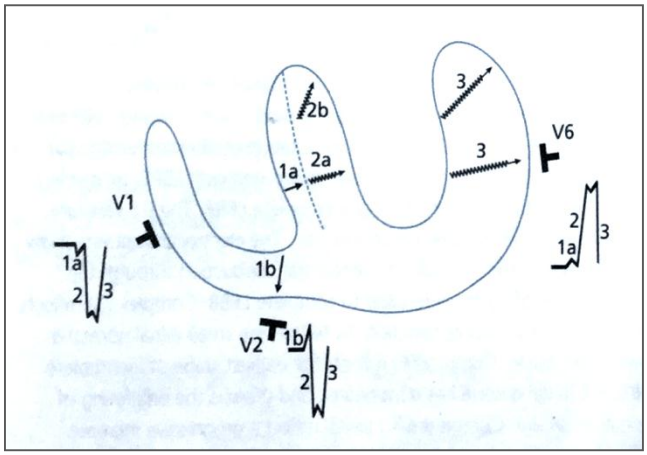
Left Bundle Branch Block

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.



Mechanism

Changes in QRS complex: With complete LBBB, activation of the left side of the interventricular septum and the free wall of the left ventricle 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 and 2. A small negative deflection in leads oriented to the right side of the septum[12]. The first component is very small and may not be seen unless sensitive recording apparatus is used.

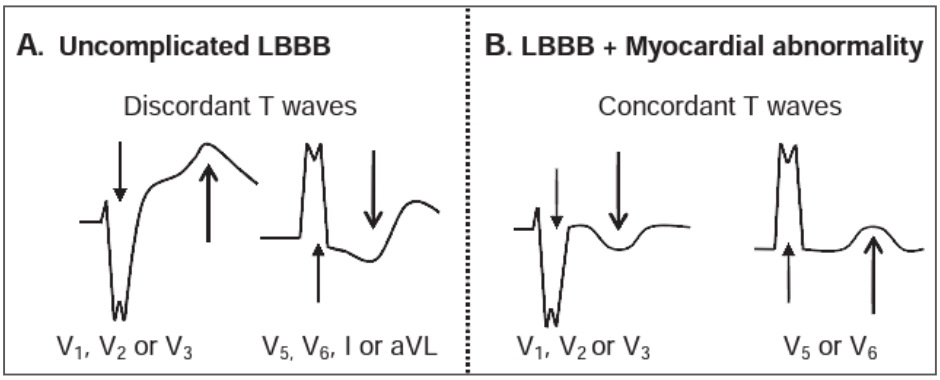


Following right septal activation, the activation process crosses an intraseptal physiological barrier and activates the left side of the interventricular septum. This results in vectors of large magnitude that are directed to the left and posteriorly- vector 2. This is reflected by 1.A tall R wave in 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.

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 repolarisation 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.

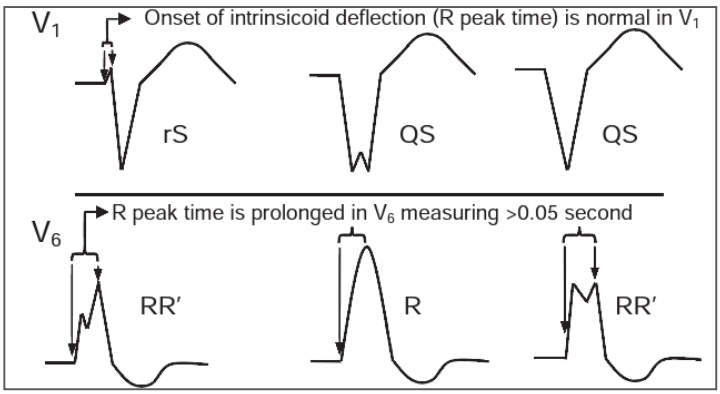


ECG manifestations of Complete LBBB

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 msec due to delayed and anomalous activation of left ventricle. 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[13].

Leads oriented to the left ventricle reflect the following:

1. There is a small initial r wave due to depolarisation of the right side of the septum. The septal q waves are absent.

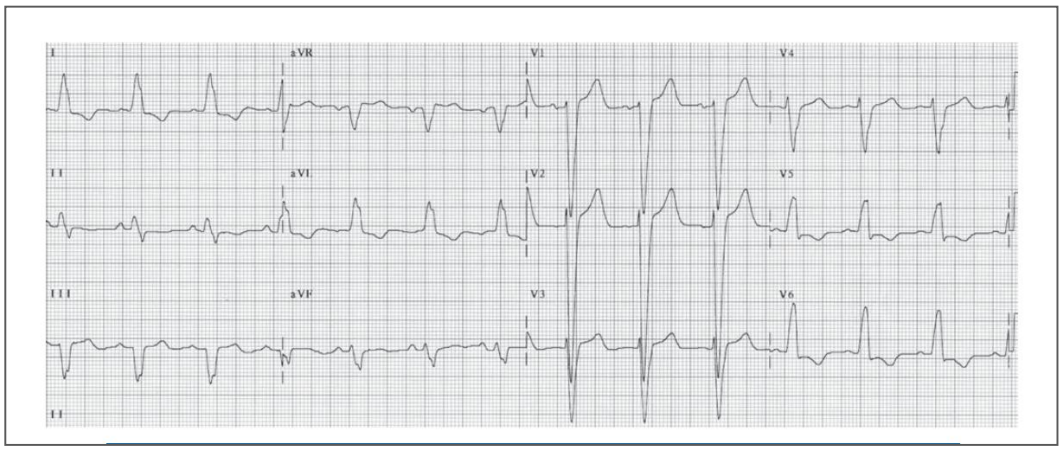


2. There is a relatively tall R wave, which is mainly due to depolarisation of left side of the septum.
3. There is a terminal R' wave, which is due to late and anomalous depolarisation of left free wall.
4. The onset of intrinsicoid deflection in leadV6 is delayed to 50ms or more.

Leads oriented to the right ventricle reflect the following:

1. There is a small initial r wave due to depolarisation of the right paraseptal region.

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



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 lead v1[14].

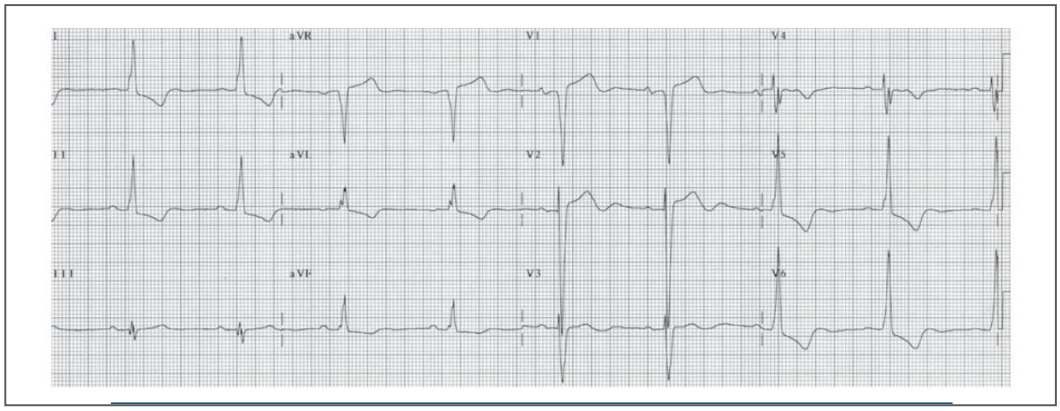
With increasing progression of the incomplete LBBB, a slur appears on the upstroke of QRS complex. This slur is due to the increasing dominance of right septal vector that penetrates the intraseptal barrier to a varying degree. It is allowed to play a more dominant role due to increasing delay within 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.



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[15]. 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

Aetiology: 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 abnormal ventricular activation pattern of LBBB itself induces further hemodynamic changes that are superimposed on the abnormalities caused by the underlying cardiac disease process. 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 remodelling; as well as functional mitral valve dysfunction with mitral regurgitation due to the altered geometry of the mitral valve apparatus from the changes in contraction patterns. As a result, cardiac efficiency is further reduced. Severe left ventricular dysfunction is common, with a delay of more than 60 milliseconds between septal and lateral wall contraction 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 paradoxical splitting of the second heart sound, the second heart sound becomes narrowly split with inspiration and widely split with expiration. A short systolic murmur of mitral regurgitation may be audible during early systole because of asynchrony in contraction of the papillary muscles.

LBBB with Left ventricular hypertrophy: Left ventricular hypertrophy 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: 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 complexes can occur in the anterior precordial leads and this 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 a marker for myocardial ischemia in patients 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[16]. Exercise can further augment the wall motion abnormalities of LBBB even in the absence of 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. LBBB is associated with a higher than normal risk of morbidity and mortality from myocardial infarction, heart failure, and arrhythmias including high-grade AV block[17]. 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[18]. LBBB is also significantly related to an increase in sudden cardiac death.

Among patients with coronary artery disease, including acute STEMI, the presence of LBBB correlates with more extensive disease, more severe left ventricular dysfunction and often reduced survival rates. In patients with heart failure, increased QRS duration has been shown to be associated with poor prognosis[19].

Patients with associated left or right axis deviation have more severe disease manifestations[20]. Left axis deviation is associated with more severe conduction system disease involving the fascicles and the main left bundle. Right axis deviation suggests 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[21]. LBBB, which is a bifascicular block, may progress to trifascicular block or complete AV block. In such patients, insertion of a permanent pacemaker is warranted.

Patients with left ventricular systolic dysfunction and LBBB who continue to have symptoms of heart failure despite receiving optimal medical management may benefit from cardiac resynchronization therapy[22]. 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 > 0.12 seconds
2. Normal sinus rhythm
3. Systolic dysfunction with ejection fraction < 35%
4. New York Heart Association functional class III or IV heart failure
5. Symptoms of heart failure in spite of 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 serves as a definitive tool to establish the possible etiology of 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. Wall

motion abnormalities due to left bundle branch block exclusively are most prominent in the proximal and mid-anterior septal regions and less obvious in the anterior wall or apex. There is often multiphasic motion of the septum. They typically do not result in alteration of left ventricular geometry or any regional dilation.

M-mode echocardiography is the most definitive method for demonstrating the mechanical effects of the left bundle branch block. A classic early downward beak is noted with the onset of ventricular systole followed by concurrent anterior motion of the septum and myocardial thickening[23]. There is often marked dyssynchrony between the onsets of motion in the noninvolved walls since the wall motion abnormality in left bundle branch block is due to conducting delay.

Acute Myocardial Infarction: A regional wall motion abnormality is the echocardiographic hallmark of an acute ischemic syndrome. In the presence of chest pain with ECG changes, detection of a regional wall motion abnormality in ECHO is direct evidence of myocardial ischemia. And the extent of wall motion abnormality is directly related to the volume of myocardium in jeopardy. It is not necessary that the entire myocardium should be ischemic to result in a regional wall motion

abnormality[24]. Ischemia involving 25% of the wall thickness itself can result in dyskinesias of the entire wall.

Ischemia in the left anterior descending territory results in wall motion abnormalities that often extend to the anterior wall and apex and loss of systolic thickening of the myocardium in the ventricular septum. These are often associated with abnormal geometry of the left ventricular cavity. 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

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 dilation of the ascending aorta and effacement of the sinotubular junction resulting in secondary aortic insufficiency[26]. 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[27]. 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 pseudonormal filling or restrictive physiology can be detected. However, if left ventricular hypertrophy remains uncomplicated by concurrent systolic dysfunction, no other changes are anticipated[28]. 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: Echocardiography serves as a definitive tool for establishing the diagnosis and severity of cardiomyopathy as well as to define the etiology to some extent. The primary diagnostic features of dilated cardiomyopathy are left ventricular dilatation and systolic dysfunction. Secondary features are also fairly common and contribute to symptoms and prognosis substantially. These include diastolic dysfunction with chronic elevation of left atrial pressure, secondary pulmonary hypertension with concurrent right ventricular dysfunction and secondary mitral and tricuspid regurgitation.

Left ventricular dilation is ubiquitous and a requisite component for establishing the diagnosis of dilated cardiomyopathy. The degree of dilatation can be mild or substantial with left ventricular internal dimension measuring 9 cm or more occasionally encountered. The distribution of systolic dysfunction within the left ventricular walls is dependent on the aetiology of cardiomyopathy whether ischemic or non ischemic. With an ischemic aetiology, there is usually greater regional variation in systolic dysfunction than if the process is nonischemic. Regional variation in systolic dysfunction occurs in non ischemic

cardiomyopathy also, typically with the proximal inferoposterior and posterior lateral walls having relatively preserved function. As a result of dilation and systolic dysfunction, the left ventricle becomes more spherical that contributes further to the systolic dysfunction as the spherical geometry interferes with the efficiency of contraction. Normally, the long axis dimension of the left ventricle exceeds the minor axis dimension by a ratio of 1.6:1 or greater. With progressive dilation, the minor axis increases disproportionately and a ratio of less than 1.5:1 implies pathological remodelling.

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 increasing spherical geometry results in lateral and apical displacement of the papillary muscles. This reduces the length of the mitral valvular apparatus and results in functional mitral regurgitation as the leaflets coapt only at their tips. In some patients with profound ventricular remodelling and concurrent diastolic dysfunction, diastolic mitral regurgitation may develop as a result of a marked increase in pressure and reversal of the left atrial to left ventricular pressure gradient.

The aetiology 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.

MATERIALS AND METHODS

MATERIALS AND METHODS

Study centre : Institute of Internal Medicine, Madras Medical College & Rajiv Gandhi Government General Hospital.

Study Design : Single centre observational prospective study

Venue : Rajiv Gandhi Government General Hospital, Chennai

Collaborating Departments:

Institute of Cardiology, MMC& RGGGH, Ch-3.

Duration: Study was conducted from March 2017 - August 2017

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. The required sample is around 50 using the above formula. About hundred patients who attended our outpatient or emergency department with ECG findings of Left bundle branch block were selected so that the sample error in the study could be minimised. A complete history was taken either from the patient or his/ her attendee including past history of Diabetes, hypertension, coronary artery disease, seizures, Jaundice, CVA,

COPD, CKD, H/o any prior surgery, malignancy and retroviral status. His/her personal habits were enquired.

A complete physical examination was done with monitoring of vitals including temperature, pulse rate, respiratory rate and blood pressure. Detailed cardiovascular examination done. Electrocardiography findings were confirmed. The patients were then subjected to detailed Echocardiographic examination. Treatment was started based on clinical and Echocardiographic presentation and patients were advised regular followup.

Inclusion Criteria:

Patients with ECG changes of complete LBBB

Exclusion Criteria:

Patients with ECG showing Incomplete LBBB

Statistical Analysis Plan :

Data analysed using statistical package - SPSS Software

Consent:

All participants / attenders gave written informed consent.

Ethical Committee Approval:

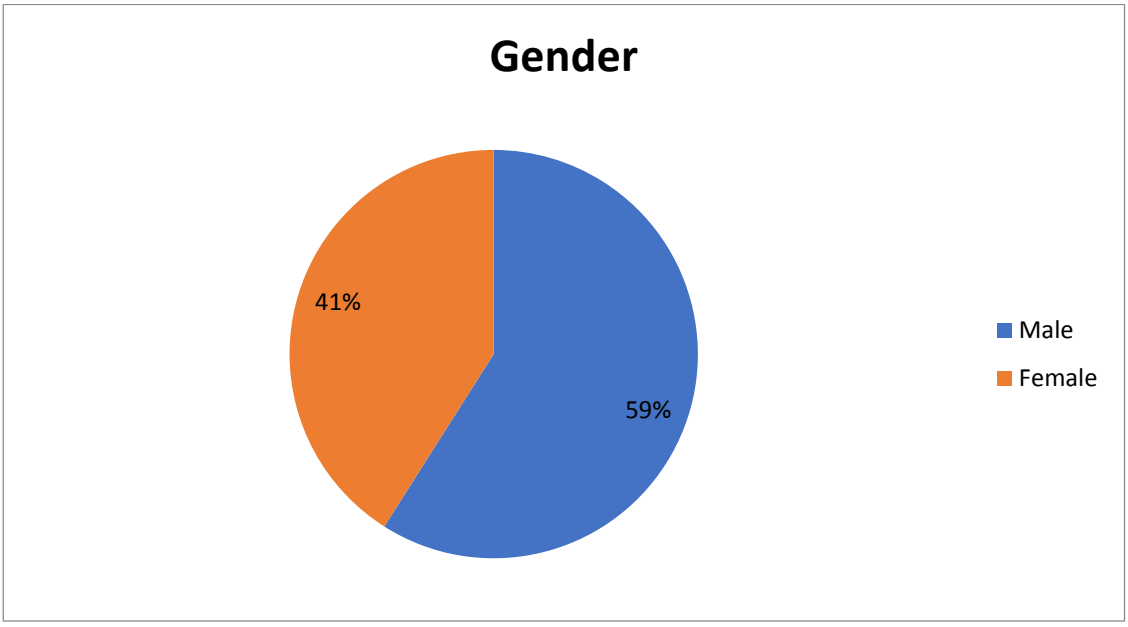
Institutional Ethics Committee of Madras Medical College approved the study.

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

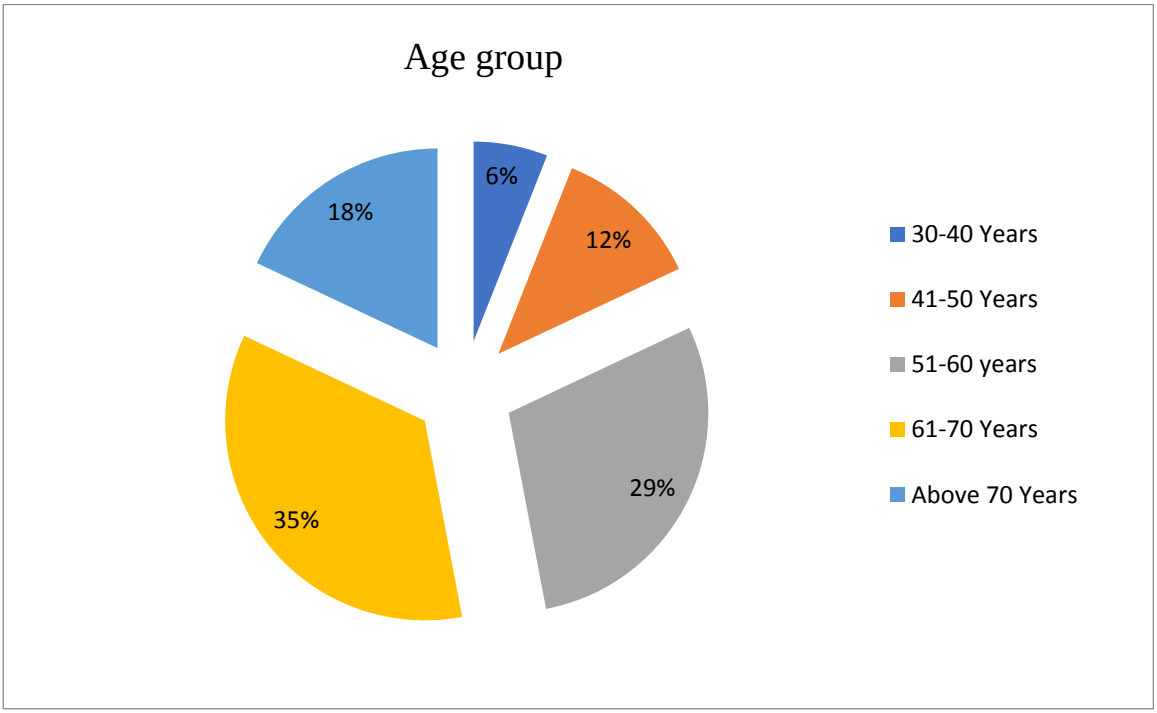
GENDER

GENDER	Frequency	Percent
Male	59	59.0
Female	41	41.0
Total	100	100.0



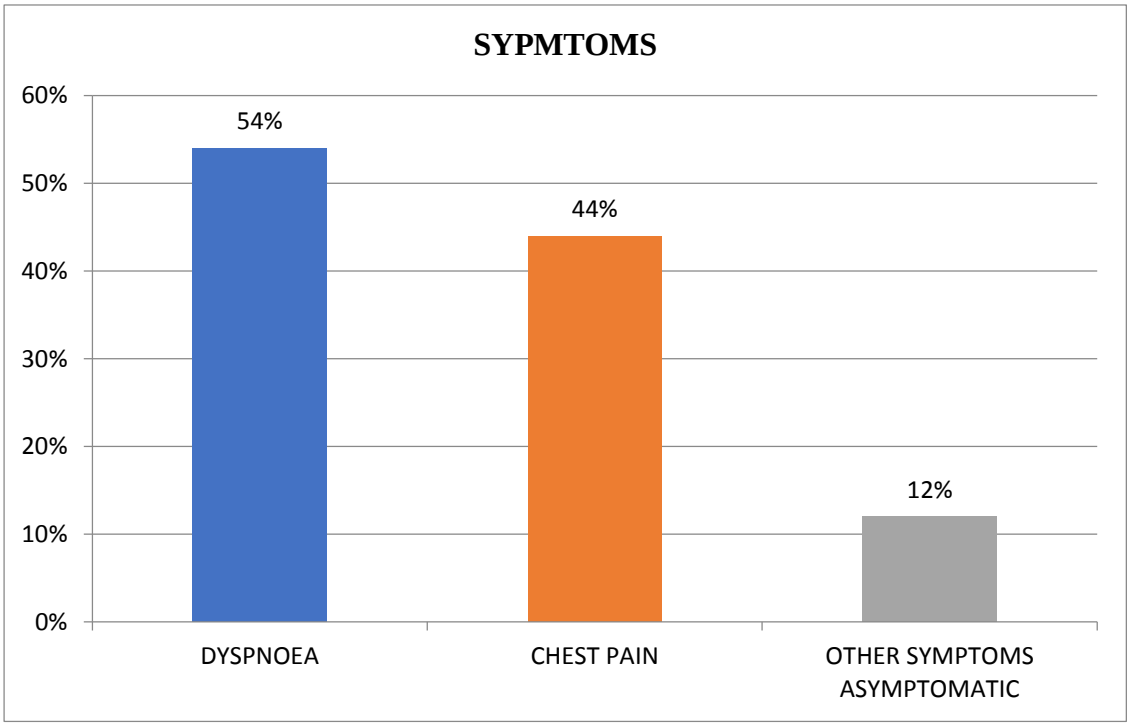
AGE GROUP

Age Group	Frequency	Percent
30-40 Years	6	6.0
41-50 Years	12	12.0
51-60 years	29	29.0
61-70 Years	35	35.0
Above 70 Years	18	18.0
Total	100	100.0



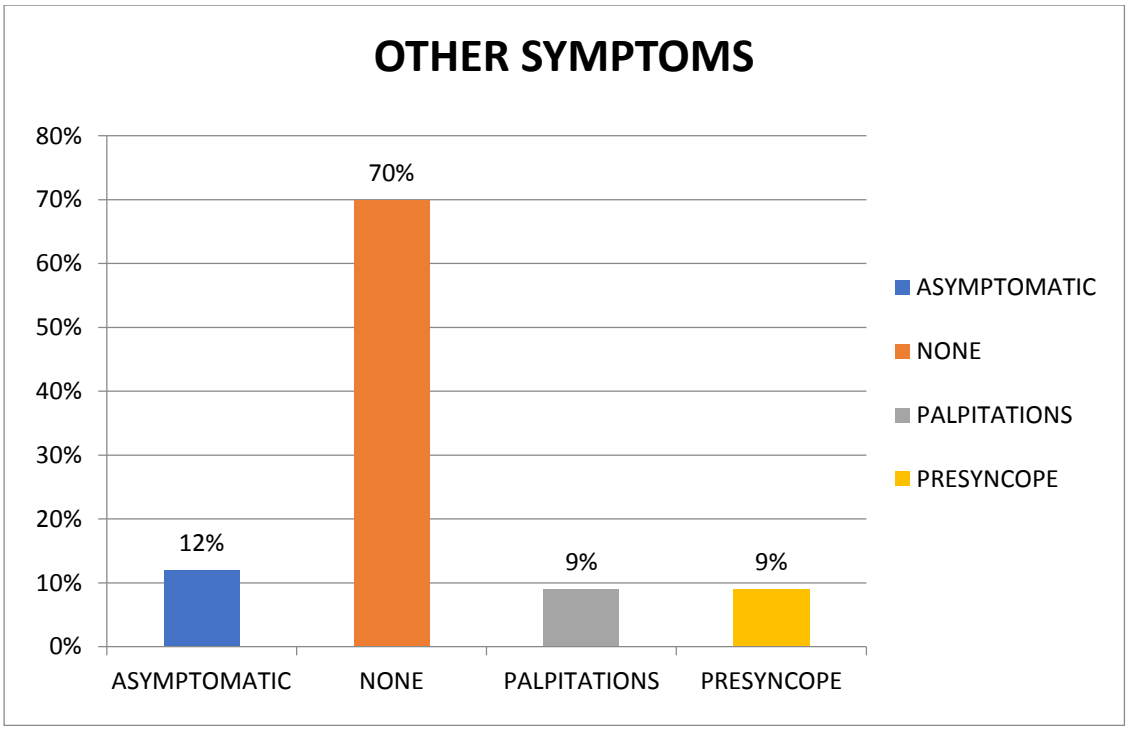
SYMPTOMS

SYMPTOMS		Count	Table N %
DYSPNOEA	Yes	54	54
CHEST PAIN	Yes	44	44
OTHER SYMPTOMS	ASYMPTOMATIC	12	12
	NONE	70	70
	PALPITATIONS	9	9
	PRESYNCOPE	9	9



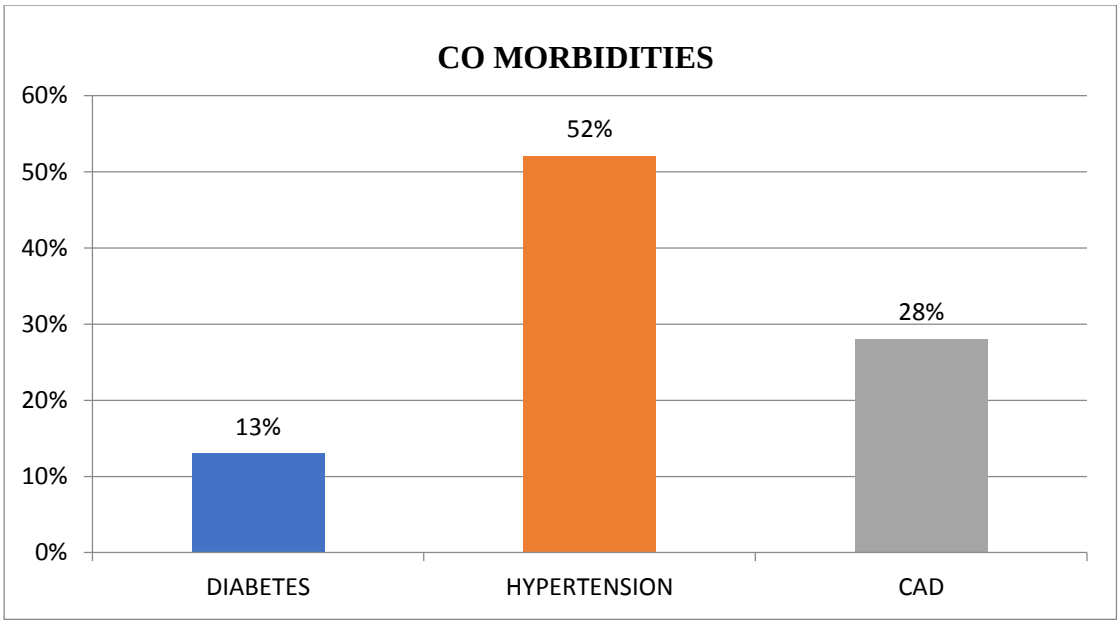
OTHER SYMPTOMS

OTHER SYMPTOMS	Frequency	Percent
ASYMPTOMATC	12	12.0
PALPITATIONS	9	9.0
PRESYNCOPE	9	9.0



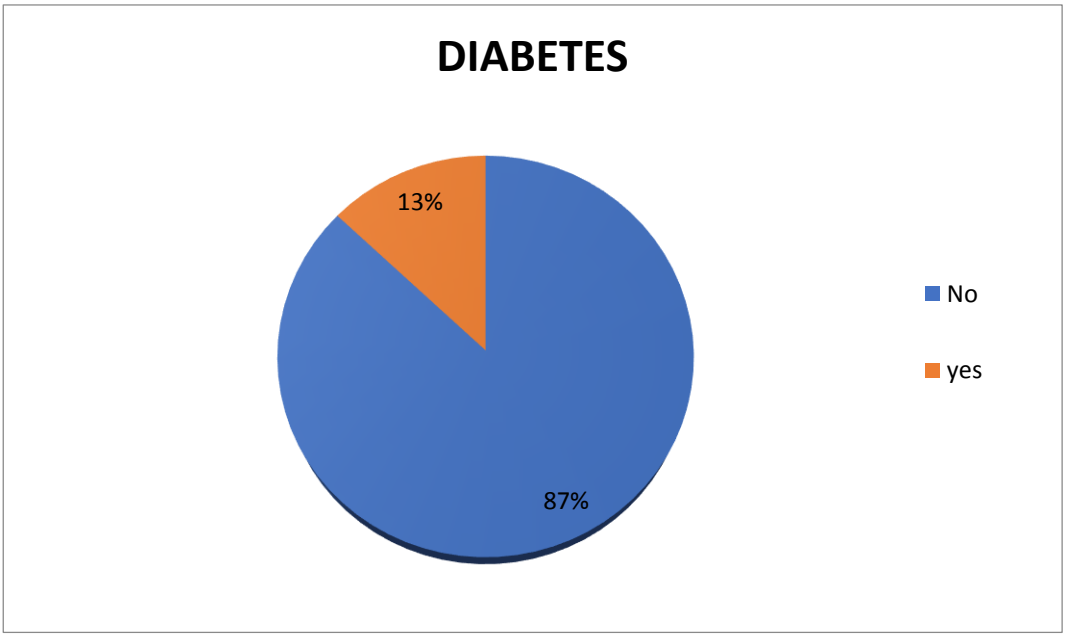
CO MORBIDITIES

	Count	Table N %
DIABETES	13	13
HYPERTENSION	52	52
CAD	28	28



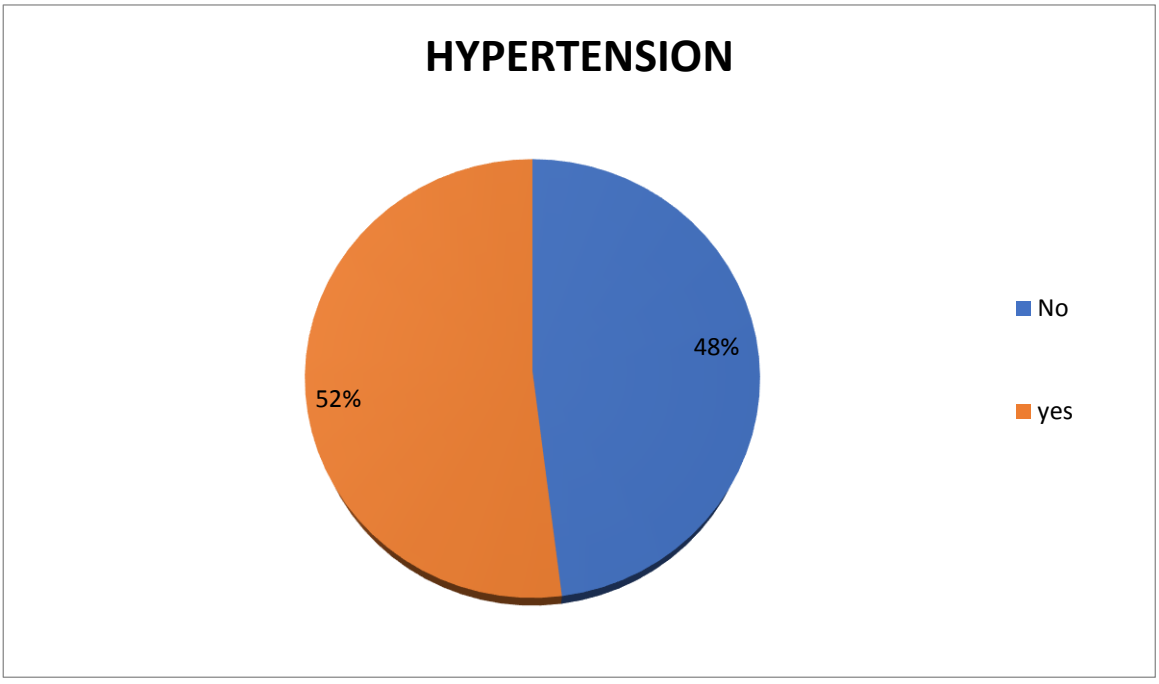
DIABETES MELLITUS

Diabetes	Frequency	Percent
No	87	87.0
Yes	13	13.0
Total	100	100.0



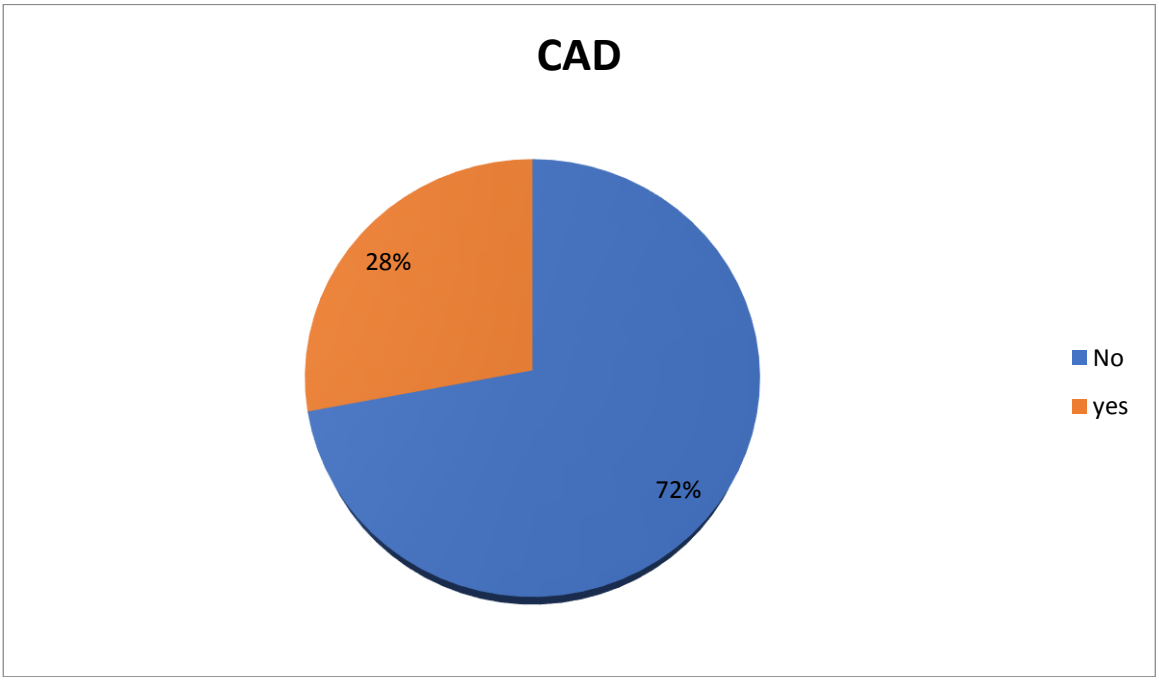
HYPERTENSION

Hypertension		Frequency	Percent
	No	48	48.0
	Yes	52	52.0
	Total	100	100.0



CORONARY ARTERY DISEASE

CAD		Frequency	Percent
	No	72	72.0
	Yes	28	28.0
	Total	100	100.0



SMOKER

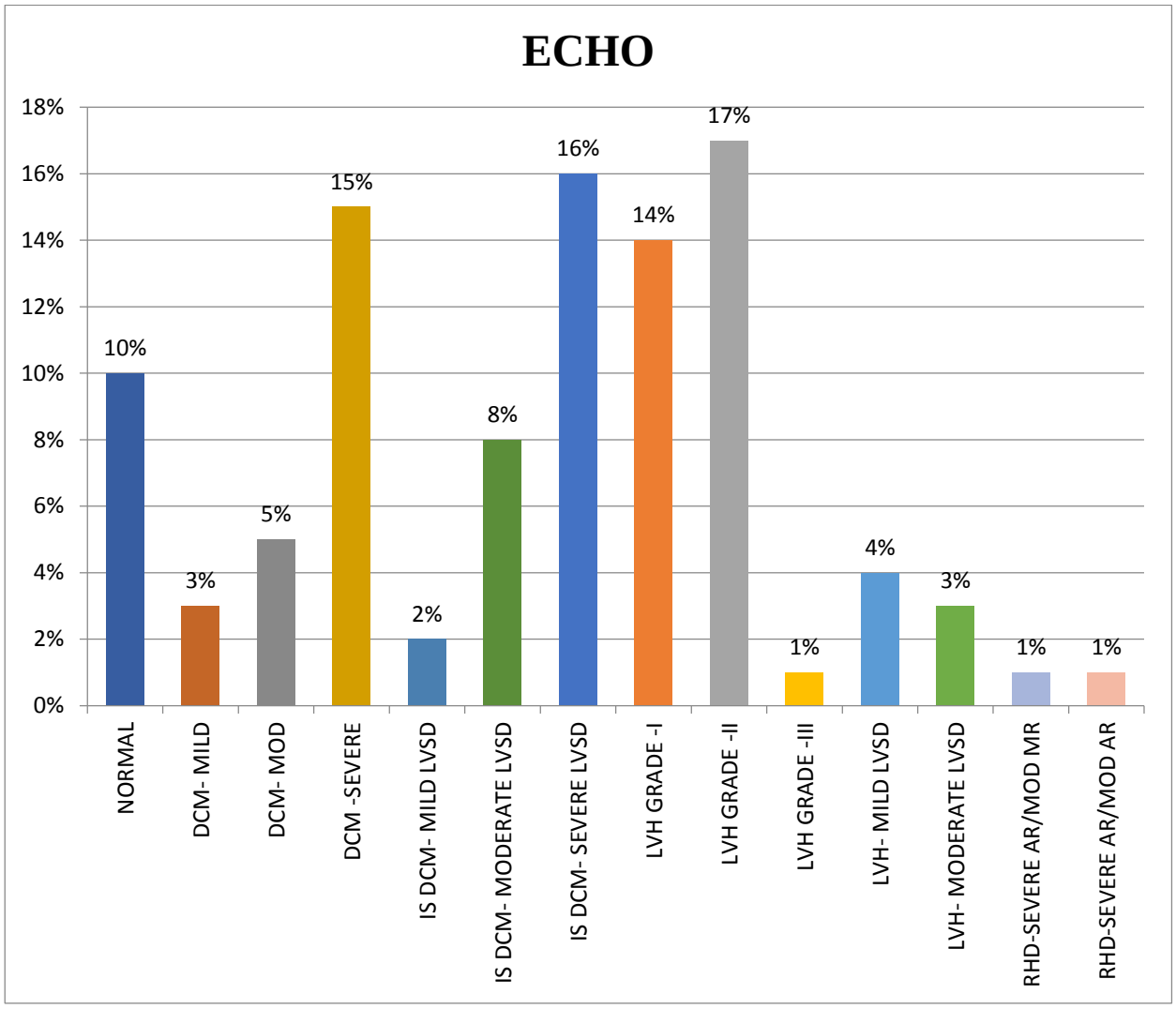
Smoker		Frequency	Percent
	No	65	65.0
	Yes	35	35.0
	Total	100	100.0

ALCOHOLIC

Alcoholic		Frequency	Percent
	No	71	71.0
	Yes	29	29.0
	Total	100	100.0

ECHOCARDIOGRAPHY

ECHOCARDIOGRAPHY		Frequency	Percent
	NORMAL	10	10.0
	DCM- MILD LVSD	3	3.0
	DCM- MOD LVSD	5	5.0
	DCM –SEVERE LVSD	15	15.0
	ISCHEMIC DCM- MILD LVSD	2	2.0
	ISCHEMIC DCM- MOD LVSD	8	8.0
	ISCHEMIC DCM- SEV LVSD	16	16.0
	LVH GRADE –I DD	14	14.0
	LVH GRADE –II DD	17	17.0
	LVH GRADE –III DD	1	1.0
	LVH- MILD LVSD	4	4.0
	LVH- MODERATE LVSD	3	3.0
	RHD-SEVERE AR/MOD MR	1	1.0
	RHD-SEVERE AS/MOD AR	1	1.0
	Total	100	100.0


ECHO
18%
16%
14%
12%
10%
8%
6%
4%
2%
0%
10%
3%
5%
15%
2%
8%
16%
14%
17%
1%
4%
3%
1%
1%
NORMAL
DCM- MILD
DCM- MOD
DCM -SEVERE
IS DCM- MILD LVSD
IS DCM- MODERATE LVSD
IS DCM- SEVERE LVSD
LVH GRADE -I
LVH GRADE -II
LVH GRADE -III
LVH- MILD LVSD
LVH- MODERATE LVSD
RHD-SEVERE AR/MOD MR
RHD-SEVERE AR/MOD AR

DILATED CARDIOMYOPATHY AND CLINICAL PROFILE

			Echo			Total
			DCM-MILD	DCM-MOD	DCM - SEVERE	
Age group	51-60 years	Count	1	2	1	4
		% within Echo	33.3%	40.0%	6.7%	17.4%
	61-70 Years	Count	2	1	8	11
		% within Echo	66.7%	20.0%	53.3%	47.8%
	Above 70 Years	Count	0	2	6	8
		% within Echo	0.0%	40.0%	40.0%	34.8%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=5.227 P= 0.265

			Echo			Total
			DCM-MILD	DCM- MOD	DCM -SEVERE	
GENDER	Male	Count	0	2	9	11
		% within Echo	0.0%	40.0%	60.0%	47.8%
	Female	Count	3	3	6	12
		% within Echo	100.0%	60.0%	40.0%	52.2%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=3.764 P= 0.152

			Echo			Total
			DCM-MILD	DCM-MOD	DCM - SEVERE	
DYSPNOEA	No	Count	3	2	0	5
		% within Echo	100.0%	40.0%	0.0%	21.7%
	Yes	Count	0	3	15	18
		% within Echo	0.0%	60.0%	100.0%	78.3%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=15.947** P<0.001

			Echo			Total
			DCM- MILD	DCM- MOD	DCM - SEVERE	
CHEST PAIN	No	Count	1	2	8	11
		% within Echo	33.3%	40.0%	53.3%	47.8%
	Yes	Count	2	3	7	12
		% within Echo	66.7%	60.0%	46.7%	52.2%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=0.558 P= 0.757

			Echo			Total
			DCM- MILD	DCM- MOD	DCM -SEVERE	
DIABETES	No	Count	2	3	14	19
		% within Echo	66.7%	60.0%	93.3%	82.6%
	Yes	Count	1	2	1	4
		% within Echo	33.3%	40.0%	6.7%	17.4%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=3.511 P= 0.173

			Echo			Total
			DCM- MILD	DCM- MOD	DCM -SEVERE	
HYPERTENSION	No	Count	1	4	1	6
		% within Echo	33.3%	80.0%	6.7%	26.1%
	Yes	Count	2	1	14	17
		% within Echo	66.7%	20.0%	93.3%	73.9%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=10.553** P= 0.005\

			Echo			Total
			DCM- MILD	DCM- MOD	DCM -SEVERE	
CAD	No	Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=14.822** P<0.001

			Echo			Total
			DCM- MILD	DCM- MOD	DCM -SEVERE	
SMOKER	No	Count	3	3	10	16
		% within Echo	100.0%	60.0%	66.7%	69.6%
	Yes	Count	0	2	5	7
		% within Echo	0.0%	40.0%	33.3%	30.4%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=1.588 P= 0452

			Echo			Total
			DCM- MILD	DCM- MOD	DCM -SEVERE	
ALCOHOLIC	No	Count	3	4	13	20
		% within Echo	100.0%	80.0%	86.7%	87.0%
	Yes	Count	0	1	2	3
		% within Echo	0.0%	20.0%	13.3%	13.0%
Total		Count	3	5	15	23
		% within Echo	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=0.664 P= 0.717

LEFT VENTRICULAR HYPERTROPHY AND CLINICAL PROFILE

			Echo					Total
			LVH GRADE –I DD	LVH GRADE – II DD	LVH GRADE – III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
Age Group	30-40 Years	Count	2	0	0	0	0	2
		% within Echo	14.3%	0.0%	0.0%	0.0%	0.0%	5.1%
	41-50 Years	Count	2	1	0	0	1	4
		% within Echo	14.3%	5.9%	0.0%	0.0%	33.3%	10.3%
	51-60 years	Count	3	3	0	4	1	11
		% within Echo	21.4%	17.6%	0.0%	100.0%	33.3%	28.2%
	61-70 Years	Count	7	7	1	0	1	16
		% within Echo	50.0%	41.2%	100.0%	0.0%	33.3%	41.0%
	Above 70 Years	Count	0	6	0	0	0	6
		% within Echo	0.0%	35.3%	0.0%	0.0%	0.0%	15.4%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=26.818* P=0.044

GENDER			Echo					Total
			LVH GRADE - I DD	LVH GRADE –II DD	LVH GRADE -III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	Male	Count	9	12	0	3	1	25
		% within Echo	64.3%	70.6%	0.0%	75.0%	33.3%	64.1%
	Female	Count	5	5	1	1	2	14
		% within Echo	35.7%	29.4%	100.0%	25.0%	66.7%	35.9%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=3.537 P=0.472

DYSPNOEA			Echo					Total
			LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	No	Count	13	10	0	2	2	27
		% within Echo	92.9%	58.8%	0.0%	50.0%	66.7%	69.2%
	Yes	Count	1	7	1	2	1	12
		% within Echo	7.1%	41.2%	100.0%	50.0%	33.3%	30.8%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square= 7.487 P=0.112

CHEST PAIN			Echo					Total
			LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	No	Count	4	8	1	3	1	17
		% within Echo	28.6%	47.1%	100.0%	75.0%	33.3%	43.6%
	Yes	Count	10	9	0	1	2	22
		% within Echo	71.4%	52.9%	0.0%	25.0%	66.7%	56.4%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=4.395 P=0.355

OTHER SYMPTOMS		Echo					Total
		LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
ASYMPTOMATIC	Count	2	0	0	0	0	2
	% within Echo	14.3%	0.0%	0.0%	0.0%	0.0%	5.1%
NONE	Count	9	14	1	3	3	30
	% within Echo	64.3%	82.4%	100.0%	75.0%	100.0%	76.9%
PALPITATIONS	Count	2	2	0	0	0	4
	% within Echo	14.3%	11.8%	0.0%	0.0%	0.0%	10.3%
PRESYNCOPE	Count	1	1	0	1	0	3
	% within Echo	7.1%	5.9%	0.0%	25.0%	0.0%	7.7%
Total	Count	14	17	1	4	3	39
	% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=7.229 P=0.842

DIABETES			Echo					Total
			LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	No	Count	10	16	0	4	3	33
		% within Echo	71.4%	94.1%	0.0%	100.0%	100.0%	84.6%
	Yes	Count	4	1	1	0	0	6
		% within Echo	28.6%	5.9%	100.0%	0.0%	0.0%	15.4%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=9.822* P=0.044

HYPERTENSION			Echo					Total
			LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	No	Count	14	6	0	2	0	22
		% within Echo	100.0%	35.3%	0.0%	50.0%	0.0%	56.4%
	Yes	Count	0	11	1	2	3	17
		% within Echo	0.0%	64.7%	100.0%	50.0%	100.0%	43.6%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=19.144** P=0.001

CAD			Echo					Total
			LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	No	Count	14	17	1	2	1	35
		% within Echo	100.0%	100.0%	100.0%	50.0%	33.3%	89.7%
	Yes	Count	0	0	0	2	2	4
		% within Echo	0.0%	0.0%	0.0%	50.0%	66.7%	10.3%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=20.893** P=0.001

SMOKER			Echo					Total
			LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	No	Count	8	9	1	1	2	21
		% within Echo	57.1%	52.9%	100.0%	25.0%	66.7%	53.8%
	Yes	Count	6	8	0	3	1	18
		% within Echo	42.9%	47.1%	0.0%	75.0%	33.3%	46.2%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=2.462 P=0.652

ALCOHOLIC			Echo					Total
			LVH GRADE –I DD	LVH GRADE –II DD	LVH GRADE –III DD	LVH-MILD LVSD	LVH-MODERATE LVSD	
	No	Count	9	11	1	2	2	25
		% within Echo	64.3%	64.7%	100.0%	50.0%	66.7%	64.1%
	Yes	Count	5	6	0	2	1	14
		% within Echo	35.7%	35.3%	0.0%	50.0%	33.3%	35.9%
Total		Count	14	17	1	4	3	39
		% within Echo	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

Pearson Chi-Square=0.917 P=0.922

DISCUSSION

DISCUSSION

The observational study done at MMC and RGGGH, Chennai during March 2017 to August 2017 in the Institute of Internal medicine examined the clinical and Echocardiographic profile in Left bundle branch block patients. A total of 100 patients were enrolled in the study. Cases were selected from Medicine Outpatients.

Out of 100 patients selected in our study, 59 patients were male and 41 were female. 29% were in 51 to 60 years age group, 35 % in 61 to 70 years age group and 18 % in 71 to 80 years age group. The median age group was 62.03 years. According to Framingham study conducted in 1979 (5,209 subjects, 55 with LBBB), the mean age of onset was around 62 years.

The most common presenting symptom was dyspnoea in 54 % and chest pain in 44%. 12 % of the patients were asymptomatic. 13% were known diabetic, 52 % hypertensive and 28 % had coronary artery disease. The Framingham Study also showed a clear association between LBBB and major cardiovascular diseases such as hypertension, cardiac enlargement and coronary heart disease.

The most common finding in echocardiography was Left ventricular hypertrophy in 39 patients; 32 had diastolic dysfunction and 7

had systolic dysfunction. 26 of these patients had ischemic DCM with systolic dysfunction. 23 had non ischemic DCM. 2 patients had Rheumatic heart disease. 10 had normal Echocardiogram. How many of these develop cardiovascular disease on follow up remains to be seen. Left ventricular systolic dysfunction was identified in 56 patients; severe LVSD in 31 patients; moderate LVSD in 16 patients; mild LVSD in 9 patients.

Correlation study was done between clinical profile and Echocardiographic findings. Significant association was found between dyspnoea and DCM showing breathlessness as the chief presenting complaint in the setting of DCM. Association was also found between hypertension & coronary artery disease with DCM which shows these factors as main aetiological factors.

There is a significant association between Left ventricular hypertrophy and age which reflects increasing age as a risk factor for LVH. There is also a strong association between Diabetes, Hypertension and Coronary artery disease with LVH. This suggests a possible role of the above factors in the aetiology of LVH.

CONCLUSION

CONCLUSION

1. The prevalence of Left bundle branch block increases with increasing age.
2. Majority of patients had antecedent cardiovascular disease at the time of diagnosis.
3. Most common causes include hypertension and dilated cardiomyopathy.
4. Even isolated LBBB ultimately lands in major cardiovascular disease.
5. Most common presenting symptom is dyspnoea followed by chest pain.
6. The most common finding in Echocardiogram is Left Ventricular Hypertrophy followed by Dilated Cardiomyopathy.

LIMITATIONS OF STUDY

LIMITATIONS OF 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 like Acute Myocardial infarction.

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

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BIBLIOGRAPHY

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ANNEXURES

A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENT

Name :

Age/Sex :

OP/IP No :

Occupation :

Address :

Contact No. :

SYMPTOMS

- ◊ Dyspnea
- ◊ Chest pain
- ◊ Palpitations
- ◊ Syncope
- ◊ Cough
- ◊ Leg swelling
- ◊ Asymptomatic

Patient Characteristics

- ◊ Smoker
- ◊ Alcoholic
- ◊ Diabetic
- ◊ Hypertensive
- ◊ Coronary Artery Disease
- ◊ Bronchial Asthma/COPD
- ◊ Congenital heart disease
- ◊ Others

EXAMINATION

- ◊ Blood Pressure
- ◊ Pulse Rate
- ◊ JVP
- ◊ Heart sounds
- ◊ Murmurs

ECG

ECHO

ETHICAL COMMITTEE APPROVAL

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.R.Nagendran
II Year Post Graduate in MD General Medicine
Institute of Internal Medicine
Madras Medical College
Chennai 600 003

Dear Dr.R.Nagendran,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENTS" - NO.19022017 (II)**

The following members of Ethics Committee were present in the meeting hold on **21.02.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD., Prof.of Pharmacology.,MMC,Ch-3	: Member
5.Prof.K.Ramadevi,MD.,Director,Inst.of Bio-Che,MMC,Ch-3	: Member
6.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
7.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
8.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PLAGIARISM SCREENSHOT

URKUND

Urkund Analysis Result

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Instances where selected sources appear:

1

PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled "**A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENTS**" of the candidate **Dr.R.NAGENDRAN** with registration Number **201511019** for the award of **M.D** in the branch of **GENERAL MEDICINE.** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 1 percentage of plagiarism in the dissertation.

Guide & Supervisor sign with Seal.

INFORMATION SHEET

We are conducting a study on **"A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENTS"** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us. The purpose of this study is to identify the etiology and left ventricular function in patients with LBBB.

We are selecting certain cases and if you are found eligible, we may perform extra tests and special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator Signature of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail	:	**A STUDY OF CLINICAL AND ECHOCARDIOGRAPHIC PROFILE IN LEFT BUNDLE BRANCH BLOCK PATIENTS**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.	☐
I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.	☐
I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted	☐

in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests.

Signature/thumb impression

Patient's Name and Address

Signature of Investigator

Dr.R.Nagendran

MASTER CHART

MASTER CHART

Sl.NO	AGE (Yrs)	GENDER	DYSPNOEA	CHESTPAIN	OTHER SYMPTOMS	DIABETES	SHTN	CAD	SMOKER	ALCOHOLIC	ECHOCARDIOGRAPHY
1	54	F	N	N	PRESYNCOPE	N	Y	N	N	N	DCM- MILD LVSD
2	63	F	N	Y	NONE	N	Y	N	N	N	DCM- MILD LVSD
3	64	F	N	Y	NONE	Y	N	N	N	N	DCM- MILD LVSD
4	58	F	Y	N	PALPITATIONS	N	Y	N	N	N	DCM- MOD LVSD
5	56	F	N	Y	PALPITATIONS	Y	N	N	N	N	DCM- MOD LVSD
6	79	M	Y	Y	PALPITATIONS	N	N	N	Y	N	DCM- MODERATE LVSD
7	75	F	N	Y	NONE	Y	N	N	N	N	DCM- MODERATE LVSD
8	67	M	Y	N	NONE	N	N	N	Y	Y	DCM- MODERATE LVSD
9	61	F	Y	Y	PRESYNCOPE	N	Y	N	N	N	DCM- SEVERE LVSD
10	63	M	Y	N	NONE	N	Y	N	Y	Y	DCM- SEVERE LVSD
11	66	F	Y	N	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
12	77	F	Y	N	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
13	76	M	Y	N	NONE	N	N	N	Y	N	DCM- SEVERE LVSD
14	67	M	Y	N	NONE	Y	Y	N	N	Y	DCM- SEVERE LVSD
15	78	M	Y	N	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
16	66	F	Y	Y	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
17	67	F	Y	Y	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
18	60	M	Y	N	NONE	N	Y	N	Y	N	DCM- SEVERE LVSD
19	76	F	Y	Y	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
20	65	M	Y	Y	NONE	N	Y	N	Y	N	DCM- SEVERE LVSD
21	74	M	Y	Y	NONE	N	Y	N	Y	N	DCM- SEVERE LVSD
22	68	M	Y	N	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
23	77	M	Y	Y	NONE	N	Y	N	N	N	DCM- SEVERE LVSD
24	47	M	N	N	PRESYNCOPE	N	Y	N	N	Y	IS DCM- MILD LVSD
25	59	M	N	Y	NONE	N	Y	N	Y	Y	IS DCM- MILD LVSD
26	68	M	Y	Y	NONE	N	Y	Y	Y	Y	IS DCM- MODERATE LVSD
27	57	F	Y	N	NONE	N	Y	Y	N	N	IS DCM- MODERATE LVSD
28	78	F	Y	Y	NONE	N	Y	Y	N	N	IS DCM- MODERATE LVSD

29	65	F	N	Y	PALPITATIONS	N	N	Y	N	N	IS DCM- MODERATE LVSD
30	56	M	Y	Y	PRESYNCOPE	Y	Y	Y	N	N	IS DCM- MODERATE LVSD
31	71	M	Y	N	NONE	N	Y	Y	Y	N	IS DCM- MODERATE LVSD
32	57	M	N	Y	NONE	N	N	Y	Y	N	IS DCM- MODERATE LVSD
33	63	M	Y	Y	PALPITATIONS	N	Y	Y	Y	Y	IS DCM- MODERATE LVSD
34	51	M	N	Y	NONE	Y	N	N	Y	N	LVH- GRADE I DD
35	55	M	N	N	PRESYNCOPE	N	Y	N	Y	Y	LVH- MILD LVSD
36	66	F	N	Y	NONE	N	N	N	N	N	LVH- GRADE II DD
37	71	M	N	Y	NONE	N	N	N	N	N	LVH- GRADE II DD
38	72	F	Y	N	NONE	N	Y	N	N	N	LVH- GRADE II DD
39	58	M	N	Y	NONE	Y	N	N	N	Y	LVH- GRADE II DD
40	43	M	N	N	ASYMPTOMATIC	N	N	N	Y	Y	NORMAL
41	34	F	N	N	ASYMPTOMATIC	N	N	N	N	N	NORMAL
42	64	M	N	Y	NONE	N	N	N	Y	Y	LVH- GRADE I DD
43	68	M	N	Y	NONE	N	N	N	Y	Y	LVH- GRADE I DD
44	55	F	N	Y	NONE	N	N	Y	N	N	LVH- MILD LVSD
45	48	F	Y	N	NONE	N	Y	Y	N	N	LVH- MODERATE LVSD
46	63	F	N	Y	NONE	N	Y	Y	N	N	LVH- MODERATE LVSD
47	53	M	Y	N	NONE	N	N	Y	Y	N	IS DCM- SEVERE LVSD
48	77	M	N	Y	NONE	N	Y	N	Y	N	LVH- GRADE II DD
49	36	F	N	N	ASYMPTOMATIC	N	N	N	N	N	NORMAL
50	69	M	N	Y	NONE	N	Y	N	Y	Y	LVH- GRADE II DD
51	52	F	Y	Y	NONE	N	Y	Y	N	N	IS DCM- SEVERE LVSD
52	56	M	Y	N	NONE	N	Y	N	Y	Y	LVH- MILD LVSD
53	49	M	N	N	PRESYNCOPE	Y	N	N	N	N	LVH- GRADE I DD
54	62	F	Y	N	PRESYNCOPE	Y	Y	Y	N	N	IS DCM- SEVERE LVSD
55	73	M	Y	N	NONE	N	Y	N	N	N	LVH- GRADE II DD
56	59	M	N	Y	NONE	N	Y	N	Y	Y	LVH- MODERATE LVSD
57	61	M	N	Y	NONE	N	N	N	Y	Y	LVH- GRADE I DD
58	69	F	Y	N	NONE	N	Y	Y	N	N	IS DCM- SEVERE LVSD
59	55	F	Y	N	NONE	N	Y	N	N	N	LVH- GRADE II DD
60	42	M	N	N	ASYMPTOMATIC	N	N	N	Y	Y	LVH- GRADE I DD
61	54	M	Y	N	NONE	N	Y	Y	N	N	IS DCM- SEVERE LVSD
62	54	M	Y	N	NONE	N	N	Y	Y	Y	IS DCM- SEVERE LVSD
63	64	F	Y	N	NONE	N	Y	N	N	N	LVH- GRADE II DD
64	37	M	N	N	ASYMPTOMATIC	N	N	N	N	N	LVH- GRADE I DD

65	71	F	Y	N	NONE	N	N	N	N	N	LVH- GRADE II DD
66	52	M	N	Y	PALPITATIONS	N	Y	N	N	Y	LVH- GRADE II DD
67	60	M	Y	N	NONE	N	N	Y	N	Y	IS DCM- SEVERE LVSD
68	45	M	N	N	ASYMPTOMATIC	N	N	N	N	N	NORMAL
69	72	M	Y	N	NONE	N	Y	N	Y	Y	LVH- GRADE II DD
70	31	F	N	N	ASYMPTOMATIC	N	N	N	N	N	NORMAL
71	50	M	Y	Y	NONE	N	N	N	N	Y	RHD-SEVERE AS/MOD AR
72	62	M	N	Y	NONE	Y	N	N	N	Y	LVH- GRADE I DD
73	62	F	N	Y	PALPITATIONS	N	N	N	N	N	LVH- GRADE I DD
74	51	F	N	N	ASYMPTOMATIC	N	N	N	N	N	NORMAL
75	65	M	Y	N	NONE	N	N	Y	N	Y	IS DCM- SEVERE LVSD
76	39	M	N	N	ASYMPTOMATIC	Y	N	N	N	N	NORMAL
77	68	F	Y	N	NONE	Y	Y	N	N	N	LVH- GRADE III DD
78	55	M	N	Y	NONE	Y	N	N	Y	N	LVH- GRADE I DD
79	39	F	N	Y	NONE	N	N	N	N	N	LVH- GRADE I DD
80	76	F	Y	N	NONE	N	Y	Y	N	N	IS DCM- SEVERE LVSD
81	66	M	N	Y	PALPITATIONS	N	N	N	Y	Y	LVH- GRADE II DD
82	48	M	N	N	PRESYNCOPE	N	Y	N	Y	N	LVH- GRADE II DD
83	53	F	Y	Y	NONE	N	Y	Y	N	N	IS DCM- SEVERE LVSD
84	58	F	Y	N	NONE	N	N	Y	N	N	IS DCM- SEVERE LVSD
85	51	M	N	N	ASYMPTOMATIC	N	N	N	N	N	NORMAL
86	69	M	Y	N	NONE	N	Y	N	Y	N	LVH- GRADE II DD
87	73	M	Y	N	NONE	N	Y	Y	N	N	IS DCM- SEVERE LVSD
88	45	F	Y	N	NONE	N	Y	N	N	N	RHD-SEVERE AR/MOD MR
89	62	M	Y	N	NONE	N	Y	Y	Y	Y	IS DCM- SEVERE LVSD
90	53	M	Y	N	NONE	N	N	Y	Y	N	LVH- MILD LVSD
91	67	F	N	Y	NONE	N	N	N	N	N	LVH- GRADE I DD
92	42	M	N	N	ASYMPTOMATIC	N	N	N	N	Y	NORMAL
93	70	M	N	Y	NONE	N	N	N	Y	Y	LVH- GRADE II DD
94	59	F	N	Y	PALPITATIONS	N	N	N	N	N	LVH- GRADE I DD
95	65	F	Y	N	NONE	N	N	N	N	N	LVH- GRADE I DD
96	64	M	Y	N	NONE	N	N	Y	Y	N	IS DCM- SEVERE LVSD
97	49	F	Y	N	PRESYNCOPE	N	N	Y	N	N	IS DCM- SEVERE LVSD
98	44	M	N	N	ASYMPTOMATIC	N	Y	N	N	N	NORMAL
99	56	M	Y	N	NONE	N	N	Y	N	Y	IS DCM- SEVERE LVSD
100	61	M	N	Y	NONE	N	Y	N	Y	N	LVH- GRADE II DD