A Dissertation on

INFLUENCE OF LEFT VENTRICULAR SYSTOLIC DYSFUCTION ON THE SURFACE ELECTROCARDIOGRAM – A CASE CONTROL STUDY

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CERTIFICATE

This is to certify that **Dr. S.J. AJEET ARULKUMAR,** Post - Graduate Student (July. 2003 to September 2006) in the Department of Medicine, Kilpauk Medical College, Chennai- 600 010, has done this dissertation on **"INFLUENCE OF LEFT VENTRICULAR SYSTOLIC DYSFUCTION ON THE SURFACE ELECTROCARDIOGRAM – A CASE CONTROL STUDY"** under my guidance and supervision in fulfillment of the regulations laid down by the Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in September 2006.

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CONTENTS

Introduction

Heart failure is a significant public health concern worldwide. Heart failure affects an estimated 1 percent of adults 50 to 60 years of age and 10 percent of adults in their 80s. This clinical syndrome is the most frequent cause of hospitalization in patients older than 65 years. Half of all patients with a diagnosis of heart failure will die within 4 years, and in patients with severe heart failure, more than half will die in one year. [1, 2]

The prevalence of heart failure is higher in our country than in developed countries. This is because coronary artery disease in Indians occurs at a younger age, is more severe and extensive and follows a malignant course. [3.] By the year 2020, the burden of cardiovascular disease in India will surpass that in other regions of the world[4]. The higher prevalence is directly related to the higher incidence and prevalence of hypertension and diabetes. This problem is particularly exacerbated by a lack of access to health care and to substandard preventive health care in India.

While modern methods of diagnosing heart failure such as the echocardiogram, perfusion scan, Radionuclide angiography, Left ventricular angiography, Magnetic resonance imaging (MRI) and ultra fast or cine computed tomography etc are available in big cities, the

majority of our population must be content with the treatment and investigations available in the primary health center. Often the only investigations available here to evaluate a patient with suspected cardiac disorder are an electrocardiogram and a chest x ray. Therefore the treating physician has to make a decision regarding the presence of left ventricular dysfunction based on his clinical assessment and with the help of electrocardiogram and Chest X-ray. Diagnosis of heart failure is definitely possible with the help of a thorough clinical examination and simple tools such as electrocardiogram and chest X-Ray.

Numerous studies have shown that left ventricular systolic dysfunction is very unlikely in a patient with a normal electrocardiogram. This means that left ventricular systolic dysfunction screening could be concentrated towards patients with abnormal electrocardiograms. Hence, especially in a developing country like ours it would be a cost effective approach to use the electrocardiogram as the initial investigation. In a study done by AP Davie et al if screening is restricted to those with major ECG abnormalities, the incidence of left ventricular systolic dysfunction increased from 18% to 37%.[5]. If the tracing is normal other diagnoses should be considered. Only if these have been excluded should an echocardiogram be performed.

Patients with congestive heart failure have a poor prognosis and many studies have shown mortalities of around 15% in the first year

and 30% in the second year. Even among patients with ischemic heart disease, the single most important prognostic factor is the degree of LV dysfunction. Although significant progress has been made in the treatment of heart failure, patients continue to have a poor quality of life and an unacceptably high mortality.

The question often arises in our mind – if most patients with significant LV dysfunction have electrocardiographic abnormalities then, are there any specific electrocardiographic abnormalities specific for diagnosing systolic heart failure?

Aims of the Study

1. To study specific ECG changes in patients with and left ventricular systolic dysfunction.
2. To determine if there is an association between decreasing Ejection fraction and the electrocardiogram.
3. To quantify the association between certain electrocardiographic criteria and left ventricular systolic dysfunction.

Review of the literature.

Heart failure is primarily a disease of the elderly.[6] It is responsible for 5 to 10 percent of all hospital admissions. Approximately 6% to 10% of people older than 65 years have heart failure.[7] Around 80% of patients hospitalized with heart failure are more than 65 years old.[8] Heart failure causes or contributes to approximately 250,000 deaths every year. Although significant progress has been made in the treatment of heart failure, patients continue to have a poor quality of life and an unacceptably high mortality.

Definition

By definition Heart failure is the pathophysiologic state in which the heart, via an abnormality of cardiac function (detectable or not), fails to pump blood at a rate commensurate with the requirements of the metabolizing tissues and/or pumps only from an abnormally elevated diastolic filling pressure.[9]

Pathophysiology

Heart failure may be caused by myocardial failure but may also occur in the presence of near-normal cardiac function under conditions of high demand. Heart failure always causes circulatory failure, but the converse is not necessarily the case because various noncardiac conditions (e.g., hypovolemic shock, septic shock) can

produce circulatory failure in the presence of normal, modestly impaired, or even supranormal cardiac function.

Inadequate adaptation of the cardiac myocytes to increased wall stress in order to maintain adequate cardiac output following myocardial injury (whether of acute onset or over several months to years, whether a primary disturbance in myocardial contractility or an excessive hemodynamic burden placed on the ventricle, or both), is the inciting event in CHF.

Most important among these adaptations are the (1) Frank-Starling mechanism, in which an increased preload helps to sustain cardiac performance; (2) myocardial hypertrophy with or without cardiac chamber dilatation, in which the mass of contractile tissue is augmented; and (3) activation of neurohumoral systems, especially the release of norepinephrine (NE) by adrenergic cardiac nerves, which augments myocardial contractility and the activation of the renin-angiotensin-aldosterone system (RAAS) and other neurohumoral adjustments that act to maintain arterial pressure and perfusion of vital organs.

The primary myocardial response to chronic increased wall stress includes myocyte hypertrophy and remodeling, usually of the eccentric type. The reduction of cardiac output following myocardial injury sets into motion a cascade of hemodynamic and neurohormonal derangements that provoke activation of neuroendocrine systems, most

notably the above-mentioned adrenergic systems and renin-angiotensin-aldosterone system. The release of epinephrine (E) and NE, along with the vasoactive substances endothelin-1 (ET-1) and vasopressin (V), causes vasoconstriction, which increases afterload, and, via an increase in cyclic adenosine monophosphate (cAMP), causes an increase in cytosolic calcium entry. The increased calcium entry into the myocytes augments myocardial contractility and impairs myocardial relaxation (lusitropy).

The calcium overload may also induce arrhythmias and lead to sudden death. The increase in afterload and myocardial contractility (known as inotropy) and the impairment in myocardial lusitropy lead to an increase in myocardial energy expenditure and a further decrease in cardiac output. The increase in myocardial energy expenditure leads to myocardial cell death, resulting in heart failure and further reduction in cardiac output, thus starting an accelerating cycle of further increased neurohumoral stimulation and further adverse hemodynamic and myocardial responses as described above.

The activation of the renin-angiotensin-aldosterone system leads to salt and water retention, resulting in increased preload and further increases in myocardial energy expenditure. Increases in renin, mediated by decreased stretch of the glomerular afferent arteriole, reduced delivery of chloride to the macula densa, and increased beta1-adrenergic activity as a response to decreased cardiac output, results in an increase in

angiotensin II levels and, in turn, aldosterone levels. This results in stimulation of release of aldosterone. Angiotensin II, along with ET-1, is crucial in maintaining effective intravascular homeostasis mediated by vasoconstriction and aldosterone-induced salt and water retention.

As heart failure advances and/or becomes progressively decompensated, there is a relative decline in the counter regulatory effects of endogenous vasodilators, including nitric oxide (NO), prostaglandins (PGs), bradykinin (BK), atrial natriuretic peptide (ANP), and B-type natriuretic peptide (BNP). This occurs simultaneously with the increase in vasoconstrictor substances from the renin-angiotensin-aldosterone system and adrenergic systems. This fosters further increases in vasoconstriction and thus preload and afterload, leading to cellular proliferation, adverse myocardial remodeling, and antinatriuresis with total body fluid excess and worsening CHF symptoms.

Natriuretic peptides

ANP and BNP are endogenously generated peptides activated in response to atrial and ventricular volume/pressure expansion. ANP and BNP are released from the atria and ventricles, respectively, and both promote vasodilation and natriuresis. Their hemodynamic effects are mediated by decreases in ventricular filling pressures, owing

to reductions in cardiac preload and afterload. BNP, in particular, produces selective afferent arteriolar vasodilation and inhibits sodium reabsorption in the proximal convoluted tubule. BNP inhibits renin and aldosterone release and, possibly, adrenergic activation as well.

Both ANP and BNP are elevated in chronic heart failure. BNP, in particular, has potentially important diagnostic, therapeutic, and prognostic implications. A large study has confirmed that BNP could help differentiate cardiac from respiratory acute breathlessness in the emergency room setting.[10] In particular; the negative predictive accuracy was 97%. The positive predictive value was also high at 70%.[11,12]

Systolic and diastolic heart failure

There are two types of heart failure – systolic and diastolic. In systolic heart failure, there is reduced cardiac contractility (decreased left ventricular systolic function with an ejection fraction less than 50%), whereas in diastolic heart failure there is impaired cardiac relaxation and abnormal ventricular filling (symptoms and signs of heart failure in the presence of normal ejection fraction). Both systolic and diastolic heart failure result in a decrease in stroke volume. This leads to activation of peripheral and central baroreflexes and chemoreflexes that are capable of eliciting marked increases in sympathetic nerve traffic.

In individuals with systolic dysfunction, the neurohormonal responses to decreased stroke volume result in temporary improvement in

systolic blood pressure and tissue perfusion. However, in all circumstances, the existing data support the notion that these neurohormonal responses accelerate the downward spiral of myocardial dysfunction in the long term.

In diastolic heart failure, the same pathophysiologic processes to decreased cardiac output that occur in systolic heart failure also occur, but they do so in response to a different set of hemodynamic and circulatory environmental factors that depress cardiac output.

Etiology of heart failure

There are several causes for heart failure. However in about two thirds the cause of heart failure is coronary artery disease.[13] The etiology differs in doth systolic and diastolic heart failure but a significant overlap occurs. The principal causes are mentioned below.

Dominant systolic heart failure

1. Ischemic myocardial disease, coronary artery disease
2. Alcoholic cardiomyopathy
3. Diabetic cardiomyopathy
4. Cocaine cardiomyopathy
5. Drug-induced cardiomyopathy (e.g., doxorubicin)
6. Idiopathic cardiomyopathy
7. Peripartum cardiomyopathy
8. Myocarditis

9. Preterminal valvular heart disease
10. Congenital heart disease with severe pulmonary hypertension

Dominant diastolic heart failure

1. Hypertension
2. Severe aortic stenosis
3. Hypertrophic cardiomyopathy
4. Restrictive cardiomyopathy
5. Ischemic myocardial disease, coronary artery disease
6. Acute heart failure
7. Acute mitral or aortic regurgitation
8. Rupture of valve leaflets or supporting structures
9. Infective endocarditis with acute valve incompetence
10. Myocardial infarction
11. High-output heart failure
12. Anemia
13. Systemic arteriovenous fistulas
14. Hyperthyroidism
15. Beriberi heart disease
16. Paget disease of bone

Precipitating factors

In a study conducted by Chin MH et al out of 435 patients admitted with heart failure, precipitating factors could be identified in 66%.[14] A well compensated patient with heart failure may become decompensated due to numerous precipitating factors and not necessarily due to worsening heart failure. The most common of these are inappropriate reduction in the intensity of treatment, be it dietary sodium and fluid restriction or pharmacological therapy. Other important precipitating factors include arrhythmias, systemic infection, physical, emotional and environmental stress, pulmonary disease and development of an unrelated illness.

Stages

The evolution of heart failure from an asymptomatic to a symptomatic stage has recently been classified.[15]

ACC/AHA Classification of Chronic Heart Failure

Stage Description

A. High risk for developing heart failure. (Hypertension, diabetes mellitus, CAD, family history of cardiomyopathy)

B. Asymptomatic heart failure. (Previous MI, LV dysfunction, valvular heart disease)

C. Symptomatic heart failure. (Structural heart disease, dyspnea and fatigue, impaired exercise tolerance)

D. Refractory end-stage heart failure. (Marked symptoms at rest despite maximal medical therapy)

The clinical syndrome of heart failure manifests when cellular respiration becomes impaired. The Framingham, Duke and Boston criteria were established before noninvasive techniques for assessing systolic and diastolic dysfunction became widely available. The three sets of criteria were designed to assist in the diagnosis of heart failure. The Boston criteria[16] have been shown to have the highest combined sensitivity (50 percent) and specificity (78 percent). All of these criteria are most helpful in diagnosing advanced or severe heart failure, a condition that occurs in 20 to 40 percent of patients with a decreased ejection fraction.

Boston Criteria for Diagnosing Heart Failure

Category I: history	Points
Rest dyspnea	4
Orthopnea	4
Paroxysmal nocturnal dyspnea	3
Dyspnea while walking on level area	2
Dyspnea while climbing	1
Category II: physical examination	
Heart rate abnormality (1 point if 91 to 110 beats per minute; 2 points if more than 110 beats per minute)	1 or 2
Jugular venous elevation (2 points if greater than 6 cm H2O; 3 points if greater than 6 cm H2O plus	2 or 3

hepatomegaly or edema)	
Lung crackles (1 point if basilar; 2 points if more than basilar)	1 or 2
Wheezing	3
Third heart sound	3
Category III: chest radiography	
Alveolar pulmonary edema	4
Interstitial pulmonary edema	3
Bilateral pleural effusion	3
Cardiothoracic ratio greater than 0.50	3
Upper zone flow redistribution	2

No more than 4 points are allowed from each of three categories; hence the composite score (the sum of the subtotal from each category) has a possible maximum of 12 points. The diagnosis of heart failure is classified as "definite" at a score of 8 to 12 points, "possible" at a score of 5 to 7 points, and "unlikely" at a score of 4 points or less.

Early diagnosis of heart failure is essential for successfully addressing underlying diseases or causes and, in some patients, preventing further myocardial dysfunction and clinical deterioration. However, initial diagnosis may be difficult because the presentations of heart failure can change from no symptoms to pulmonary edema with cardiogenic shock. It is estimated that heart failure is correctly diagnosed initially in only 50 percent of affected patients.[17]

The first step in diagnosing heart failure is to obtain a complete clinical history. The patient should be questioned about dyspnea, cough, nocturia, generalized fatigue and other signs and symptoms of heart failure. Peripheral edema, raised jugular venous pressure and hepatomegaly are the characteristic feature of congestion of systemic veins.[18,19]

Dyspnea, a cardinal symptom of a failing heart, often progresses from dyspnea on exertion to orthopnea, paroxysmal nocturnal dyspnea and dyspnea on rest. Cough, usually nocturnal and nonproductive, may accompany dyspnea and often occurs in similar settings (i.e., on exertion or when the patient is supine).

The NYHA functional classification scheme is used to assess the severity of heart failure and correlates fairly well with prognosis.[20] New York Heart Association Heart Failure Symptom Classification System

I No symptom limitation with ordinary physical activity

II Ordinary physical activity somewhat limited by dyspnea (i.e., long distance walking, climbing 2 flights of stairs)

III Exercise limited by dyspnea at mild work loads (ie, short distance walking, climbing one flight of stairs)

IV Dyspnea at rest or with very little exertion

Nocturia, also a frequent sign of heart failure, occurs secondary to increased renal perfusion when the patient is supine.[203] Generalized fatigue (caused by the low perfusion state) and peripheral edema with inability to wear usual footwear are frequent complaints.

With severe, longstanding heart failure, cardiac cachexia (emaciation resulting from heart disease) may develop secondary to protein-losing enteropathy and increased levels of certain cytokines, such as tumor necrosis factor. Cardiac cachexia may mimic the cachexia seen in patients with disseminated malignant disease.

Confusion and altered mental status may occur because of decreased cerebral perfusion or cardiac cirrhosis. In heart failure, cirrhosis develops secondary to chronic passive congestion of the liver.

The patient should be asked about previous chest pain or myocardial infarction because coronary artery disease is responsible for up to 75 percent of cases of heart failure with decreased left ventricular function.[5] A history of myocardial infarction has a better combination of sensitivity, specificity and positive and negative predictive value for heart failure compared with other symptoms or aspects of the medical history.[5]

Once heart failure is suspected, the functional class of the patient should be determined. The New York Heart Association (NYHA)

functional classification of congestive heart failure is commonly used in clinical practice.

Physical Examination

A complete physical examination is the second component in the diagnosis of heart failure. The patient's general appearance should be assessed for evidence of resting dyspnea, cyanosis and cachexia.

Blood Pressure and Heart Rate

The patient's blood pressure and heart rate should be recorded. High, normal or low blood pressure may be present. The prognosis is worse for patients who present with a systolic blood pressure of less than 90 to 100 mm Hg when not receiving medication (angiotensin-converting enzyme [ACE] inhibitors, beta blockers or diuretics). Tachycardia may be a sign of heart failure, especially in the decompensated state. The heart rate increases as one of the compensatory ways of maintaining adequate cardiac output. A decrease in the resting heart rate with medical therapy can be used as a surrogate marker for treatment efficacy. A weak, thready pulse and pulsus alternans are associated with decreased left ventricular function. The patient should also be monitored for evidence of periodic breathing (Cheyne-Stokes respiration).

Jugular Venous Distention

Jugular venous distention is assessed while the patient is supine with the upper body at a 45-degree angle from the horizontal plane. The top of the waveform of the internal jugular venous pulsation determines the height of the venous distention. A height of more than 4 to 5 cm from the sternal angle is considered significant.

Elevated jugular venous pressure is a specific (90 percent) but not sensitive (30 percent) sign of elevated left ventricular filling. The reproducibility of the jugular venous distention assessment is low.[23]

Point of Maximal Impulse

The point of maximal impulse of the left ventricle is usually located in the midclavicular line at the fifth intercostal space. Cardiomegaly usually displaces the cardiac impulse laterally and downward.

Third and Fourth Heart Sounds

A double apical impulse can represent an auscultated third heart sound (S3). Just as with the displaced point of maximal impulse, a third heart sound is not sensitive (24 percent) for heart failure, but it is highly specific (99 percent).[5]

Patients with heart failure and left ventricular hypertrophy can also have a fourth heart sound (S4). The physician should be alert for

murmurs, which can provide information about the cause of heart disease and also aid in the selection of therapy.

Pulmonary Examination

Physical examination of the lungs may reveal crepitations and pleural effusions. Despite the presence of pulmonary congestion, crepitations can be absent because of increased lymphatic drainage and compensatory changes in the perivascular structures that have occurred over time. Wheezing may be the sole manifestation of pulmonary congestion. Frequently, asthma is erroneously diagnosed in patients who actually have heart failure.

Liver Size and Hepatojugular Reflux

The key component of the abdominal examination is the evaluation of liver size. Hepatomegaly may occur because of right-sided heart failure and venous congestion.

The hepatojugular reflux can be a useful test in patients with right-sided heart failure. This test should be performed while the patient is lying down with the upper body at a 45-degree angle from the horizontal plane. The patient keeps the mouth open and breathes normally to prevent Valsalva’s maneuver, which can give a false-positive test. Moderate pressure is then applied over the middle of the abdomen for 30 to 60 seconds. Hepatojugular reflux occurs if the height of the neck veins

increases by at least 3 cm and the increase is maintained throughout the compression.[23]

Lower Extremity Edema

Lower extremity edema, a common sign of heart failure, is usually detected when the extracellular volume exceeds 5 L. The edema may be accompanied by stasis dermatitis, an often chronic, usually eczematous condition characterized by edema, hyperpigmentation and, commonly, ulceration.

Valsalva's Maneuver

Valsalva's maneuver is rarely used in the evaluation of patients with heart failure. Yet this test is simple to perform and carries one of the best combinations of specificity (91 percent) and sensitivity (69 percent) for the detection of left ventricular systolic and diastolic dysfunction in patients with heart failure. [25]

Valsalva's maneuver is performed with the blood pressure cuff inflated 15 mm Hg over the systolic blood pressure. While the physician auscultates over the brachial artery, the patient is asked to perform a forced expiratory effort against a closed airway (the Valsalva's maneuver).

A normal response would be an initial rise in systolic blood pressure at the onset of straining (phase I) with Korotkoff's sounds heard. While the maneuver is maintained (phase II), a decrease in the blood

pressure occurs with loss of Korotkoff's sounds. Release of the maneuver (phase III) is followed by an overshoot of blood pressure and the reappearance of heart sounds (phase IV). Abnormal responses occurring in patients with heart failure are maintenance of beats throughout Valsalva's maneuver (square wave) or lack of reappearance of Korotkoff's sounds after release of the maneuver (absent overshoot).

Laboratory Findings

Most patients with heart failure have normal electrolyte levels. However, extended use of kaliuretic diuretics can lead to hypokalemia, and the use of potassium-sparing diuretics and ACE inhibitors may result in hyperkalemia. Blood urea nitrogen and creatinine levels may become elevated, reflecting prerenal azotemia. Hyponatremia may be present in patients with advanced heart failure.

When the liver becomes congested, serum transaminase and bilirubin levels may become elevated, and jaundice may be present. With chronic congestive hepatomegaly, cardiac cirrhosis may occur and cause hypoalbuminemia, hypoglycemia and an increased prothrombin time.

The prognosis is worse in patients with hyponatremia or abnormalities secondary to congested hepatomegaly.

Anemia may contribute to worsening heart failure. When severe, anemia may even cause heart failure.

In all patients with newly diagnosed heart failure, thyroid function tests should be performed to rule out hypothyroidism or hyperthyroidism.

It may soon be possible to routinely obtain serum measurements of two plasma enzymes secreted by the overloaded heart. Plasma atrial natriuretic peptide is secreted in response to increased intra-atrial pressure, and brain natriuretic peptide (BNP) is secreted by the failing ventricle. Levels of these enzymes, but specifically BNP, are elevated in patients with dyspnea resulting from heart failure. In one study, elevated BNP levels had more than a 90 percent specificity and sensitivity for heart failure. [26]

Diagnostic Tests

Patients with heart failure and atrial fibrillation, atrial tachycardia, ventricular tachycardia or left bundle branch block have a worse prognosis than patients with heart failure who do not have these electrocardiographic findings.

Electrocardiography

An electrocardiogram (ECG) should be obtained in all patients who present with heart failure. A normal electrocardiogram suggests that the diagnosis of Heart failure should be carefully reviewed. The negative predictive value of normal electrocardiogram to exclude LV systolic dysfunction exceeds 90%.[27] On the other hand, the presence of

anterior Q waves and a left bundle branch block in patients with ischemic heart disease are good predictors of decreased Ejection fraction.[28] Electrocardiographic signs of left atrial overload or left ventricular hypertrophy may be associated with systolic as well as isolated diastolic dysfunction, but they have a low predictive value. A QRS width of more than 120 milliseconds suggests that cardiac dyssynchrony may be present and a target for therapy. The electrocardiogram is crucial for detecting atrial fibrillation or flutter, and sometimes ventricular arrhythmia, all of which are causative or contributive factors for heart failure. The diagnostic contribution of the electrocardiogram increases if completed with clinical signs and symptom of cardiac failure.

Chest Radiography

Chest radiographs can be helpful in the diagnosis of heart failure. Cardiomegaly is usually manifested by the presence of an increased cardiothoracic ratio (greater than 0.50) on a posteroanterior view. However, patients with predominantly diastolic dysfunction may have normal heart size, one of the distinguishing markers of diastolic versus systolic dysfunction. Similarly Cardiomegaly is frequently absent in patients with acute heart failure of any cause. Right ventricular enlargement is suggested by the loss of free space between the cardiac silhouette and the sternum on a lateral view.

Signs of increased pulmonary venous pressure seen on chest radiographs may progress from redistribution of blood flow from the bases of the lungs to the apices to linear densities reflecting interstitial edema (Kerley's lines) to a hazy appearance concentrated mostly around the hila of the mediastinum and presenting a butterfly pattern.

Echocardiography

Transthoracic two-dimensional echocardiography with Doppler flow studies is the investigation of choice for all patients with heart failure.[29] This test helps in the assessment of left ventricular size, mass and ejection fraction.

The ejection fraction is defined as the ratio of the stroke volume to end diastolic volume. It is most often computed as follows

$$EF = EDV - ESV / EDV \times 100\ (\%)$$

The ejection fraction can be calculated by several methods, including visual estimation, which has good correlation with ejection fractions obtained by angiography[30] or radionuclide cineangiography.[31] Regional wall motion and valvular integrity, can also be evaluated.

Transesophageal echocardiography offers higher quality images than transthoracic studies. However, this technique is invasive and is best reserved for use when the quality of the two-dimensional echocardiogram is unacceptable.

Angiography

Radionuclide angiography is another noninvasive method for assessing systolic and diastolic function. This imaging technique is used when two-dimensional echocardiography is not diagnostic because adequate images could not be obtained or the findings do not agree with the clinical picture. Radionuclide angiography provides a reliable and quantitative measurement of the left ventricular ejection fraction and the regional wall motion. Left ventricular angiography can be used to assess the ejection fraction, the left ventricular volume and the severity of valvular regurgitation or stenosis.

Other Techniques

Magnetic resonance imaging (MRI) and ultrafast or cine computed tomography (CT) [31] can measure the ejection fraction and assess regional wall motion. However, assessment of cardiac function using these studies is only performed in a limited number of centers, and the superiority of the studies to echocardiography and angiography has not been proved.

In patients with known coronary artery disease and heart failure but no angina, coronary arteriography or noninvasive testing (i.e., a thallium stress test or stress echocardiogram), followed by coronary arteriography in those patients with ischemia, should be considered. The intensity of the search for ischemic heart disease in patients with heart

disease depends on the patient's probability of having coronary artery disease.

The electrocardiogram

An electrocardiogram or ECG (also known as EKG - abbreviated from the German word Elektro-Kardiographie), is surface measurement of the electrical potential generated by electrical activity in cardiac tissue. It is the product of a series of technological and physiological advances pioneered over the past two centuries.[32] British physiologist Augustus D. Waller was the pioneer of electrocardiography and in 1887 published the first human electrocardiogram. Yet in 1911 Waller said, "I do not imagine that electrocardiography is likely to find any very extensive use in the hospital. It can at most be of rare and occasional use to afford a record of some rare anomaly of cardiac action." However, the invention of the string galvanometer in 1901 by Dutch physiologist, Willem Einthoven provided a reliable and direct method of registering electrical activity of the heart. 13 years later, the Nobel Prize in Medicine was awarded to Willem Einthoven.

Basic principles

The electrocardiogram is a graphic recording of the electric potentials generated by the heart. The signals are detected by means of

metal electrodes attached to the extremities and chest wall and are then amplified and recorded by the electrocardiograph. ECG leads actually display the instantaneous differences in potential between these electrodes. The clinical utility of the ECG derives from its immediate availability as an invasive, inexpensive and highly versatile test.

In addition to its use in detecting arrhythmias, conduction disturbances and myocardial ischemia, electrocardiography may reveal other findings related to life threatening metabolic disturbances (eg. Hyperkalemia) or increased susceptibility to sudden cardiac death (e.g. QT prolongation syndromes)

The standard clinical Electrocardiogram includes recording from 12 leads. These 12 leads includes 3 bipolar (leads I, II and III), 6 unipolar precordial leads (leads V1 through V6) and 3 modified unipolar limb leads (the augmented leads aVR, aVL and aVF).

Einthoven's Law

The electrical connections for these leads are such that the potential in lead II equals the sum of potentials sensed in leads I and III

$$I + III = II$$

This relationship is known as Einthoven's Law or Einthoven's equation.

ECG waveforms and intervals

The standard 12 lead electrocardiogram is composed of waves complexes, intervals and segments. Waves are positive and negative deflection in the electrocardiograph baseline. The waves are labeled alphabetically beginning with the letter P. Interval refers to the length of a wave plus the isoelectric line that follows it. The length of an interval ends when another wave begins. They are named by using the letters of both waves on either side. Intervals contain waves. Segments refer to the baseline between the end of one wave and the beginning of the next wave. Segments are the lines between waves.

These include in the normal sinus rhythm the P wave, PR Interval, PR Segment, QRS Complex, ST Segment, QT Interval, T wave and occasionally U wave.

The P wave is caused by atrial depolarization. Electrical impulses originating in the SA node trigger atrial depolarization. The normal P wave is no more than 0.1 second in duration and 2.5mm high. The direction of electrical activity is from SA to AV node. The P wave is a representation of the time it takes for atrial depolarization. It is viewed normally as small and curved with a positive deflection. Seen at it's tallest on lead II.

The PR interval is the portion of the electrocardiogram wave from the beginning of the P wave (onset of atrial depolarization) to the

beginning of the QRS complex (onset of ventricular depolarization). It is normally 0.12 - 0.20 seconds.

The PR segment is the portion on the electrocardiogram wave from the end of the P wave to the beginning of the QRS complex. The PR segment corresponds to the time between the end of atrial depolarization to the onset of ventricular depolarization. It is an isoelectric segment.

The QRS complex represents the time it takes for depolarization of the ventricles. - Due to ventricular depolarization. It consists of three waveforms. The normal complex begins with a downward deflection known as the Q wave, followed by an upward deflection called the R wave. The next downward deflection will be the S wave. All ventricular complexes are known as QRS complexes even if every wave is not present in all complexes. The normal QRS is 0.04 to 0.12 seconds measured from the first deflection to the end of the QRS complex.

QT Interval is the beginning of the QRS complex to the end of the T wave. In the presence of a U wave the measure should be from the beginning of the QRS complex to the end of the U wave.

ST Segment is the length between the end of the S wave of the QRS complex and the beginning of the T wave. It is electrically neutral.

The ST Segment represents the period of ventricular muscle contraction before repolarization. The ST segment is normally isoelectric.

The QT interval begins at the onset of the QRS complex and to the end of the T wave. It represents the time of ventricular depolarization until ventricular repolarization.

The T wave is due to ventricular repolarization. The polarity of the T wave is generally the same as the net polarity of the preceding QRS complex. T waves are usually upright in leads I, II, aVL, aVF and the lateral precordial leads.

The U wave is a low amplitude wave that may follow the T wave. It is largest in mid precordial leads at slow heart rates. It is of the same deflection as T Wave and similar to shape to P Wave. The U Wave is thought to represent late repolarization of the Purkinje fibers in the Ventricles and is more often not shown on a rhythm strip.

ECG in left ventricular systolic dysfunction

Systolic dysfunction of the left ventricle not only results in an mechanical failure where in the left ventricle is not able to pump a stroke volume that is adequate tissues, but also leads to a disarray of the myocytes which lead to a change in their electric potential. This is reflected in the surface electrocardiogram. A dilated left ventrical which occurs in all cases of long standing systolic dysfunction produces an

electrocardiographic picture of chamber hypertrophy and more commonly an increase in QRS duration

Numerous studies have shown that left ventricular systolic dysfunction is unlikely to be present if the electrocardiogram is normal (or shows only minor abnormalities). Conversely, there is usually a major electrocardiographic abnormality in the presence of increasing left ventricular systolic dysfunction.[33]

Prolongation of QRS (120 ms) occurs in 14% to 47% of heart failure (HF) patients. Left bundle branch block is far more common than right bundle branch block.[34] In a study done by A P Davie et al in 1996, Western General Hospital, Edinburgh, a total of 534 patients aged 17-94 were assessed. Ninety six had impaired left ventricular systolic function. Of these, 90 had major electrocardiographic abnormalities (atrial fibrillation, previous myocardial infarction, left ventricular hypertrophy, bundle branch block, or left axis deviation); none had a normal electrocardiogram. Of 438 patients with normal left ventricular systolic function, 169 had major electrocardiographic abnormalities.[5]

In another study conducted by Xiao HB, Roy C, Fujimoto S, Gibson DG et al., 1996, 58 patients with dilated cardiomyopathy were followed for 4 years. A QRS duration over 160 ms was found in 8 out of the 10 patients who died, 6 of 9 who had a pacemaker and only in 5 out of the 39 stable patients ($P < 0.001$).[40]

A prolonged QRS duration **AND** the presence of LBBB as a marker of significant left ventricular systolic dysfunction was confirmed by several studies.[35,36]

Apart from wide QRS duration and bundle branch blocks, up to 50% of heart failure patients have minor intraventricular conduction delays that result in abnormal electrical depolarisation of the heart and mechanical asynchrony of the ventricles. Hence examination of a surface ECG would pick up these changes.

Amplitude of the QRS complex also predicts left ventricular dysfunction. In a study done by Wilensky RL et al., 1998, progressive electrocardiographic changes were common in patients with idiopathic dilated cardiomyopathy and QRS amplitude criteria were accurate in the prediction of left ventricular dysfunction.[37]

There are also data which suggest that in more advanced stages of congestive heart failure, power spectral analysis of heart rate variability allows identification of a subgroup of patients with higher sympathetic activation and poorer clinical status who are at major risk of adverse events.[38, 39]

Methods and materials used.

Study population

The study was conducted among 50 randomly selected patients referred to the Cardiology Department of Government Royapettah Hospital for cardiac evaluation. Another 50 patients who had normal left ventricular systolic function were used as controls.

Study duration

The period of study was from July 2004 to December 2005.

Inclusion criteria

1. Adult patients between ages 18 to 80
2. Left ventricular systolic function as defined by an EF< 50 % in ECHO (the study population)

Exclusion criteria

1 Patients with myocarditis.

2. Recent Myocardial infarction.
3. Presence of pericardial effusion.
4. Patients in Cardiogenic Shock.

Study protocol

The patients included in the study were evaluated clinically. An experienced cardiologist performed transthoracic two-dimensional

echocardiography with Doppler flow. Left ventricular systolic function was quantified in terms of fractional shortening derived from M mode. Regional wall motion and valvular integrity were also evaluated.

Normal ejection fraction was defined as an ejection fraction of 0.50 or more (50%). Left ventricular systolic dysfunction was defined as an ejection fraction less than 0.50 (50%), an arbitrary definition based on criteria used in studies such as the SOLVD study.

The patients were classified into study population (EF<50%) and control population (EF > 50%). The study population was furthered categorized into those with mild, moderate and severe left ventricular systolic dysfunction. The classification used in our study is shown below.

	Mild	Moderate	Severe
Ejection fraction (%)	40- 49	30- 39	<30

Both the control and study population were evaluated clinically. A standard 12 lead electrocardiogram along with a rhythm strip was analyzed in detail and the study performa was completed.

The following points were noted in the electrocardiogram – normal or abnormal, rate, rhythm, axis, presence of atrial or ventricular ectopics, conduction defects, left atrial Abnormality, ventricular enlargement and QRS duration.

The presence of certain electrocardiographic criteria such as the Q waves, ST Segment and T wave abnormalities were not taken into account as majority of the patients had ischemic heart disease.

For the purpose of the study the following criteria were observed when interpreting the electrocardiogram.

Rate

A rate of more than 100 –Tachycardia

A rate of less than 60 – Bradycardia

Normal sinus rhythm

Equal R-R, PR, PP intervals

Each p wave followed by a QRS complex

P waves with normal morphology

P wave rate 60 - 100 bpm with <10% variation

Axis

Normal Axis – 30 to +100 degrees

Left Axis deviation – 90 to – 30 degrees

Right Axis deviation +100 to +180 degrees

Left atrial abnormality

Broad, notched P waves in II and AVF (greater than 2.5 millimeters wide)

A negative component in V1 or V2 (that exceeds one millimeter by one millimeter, e.g., 40 milliseconds by 0.1 millivolt)

Right Bundle Branch Block

Wide QRS, more than 120 ms (3 small squares)

An rSR' or rsR' in right-sided lead V1

Prominent, delayed and wide terminal S in V5 or V6

Left Bundle Branch Block

Wide QRS, more than 120 ms (3 small squares)

An upright (monophasic) QRS complex in leads I and V6

A predominantly negative QRS complex in lead V1

Left anterior hemiblock

QRS axis more left than -30 degrees

Initial R wave in the inferior leads (II, III and aVF)

Absence of any other cause of left axis deviation

Normal QRS complex

< 0.12 s duration (3 small squares)

LV Hypertrophy

Sokolow’s criterion (S wave in lead V1 [SV1] + R wave in lead V5 or V6 [RV5 or RV6] > 35 mm)

RV hypertrophy

Dominance of R in right-oriented leads

Right axis deviation.

Using the above criteria each electrocardiogram was read and the results were tabulated. These were then analyzed with the data obtained from the electrocardiograms of the controls.

Computation of results

Of the Electrocardiograms studied in the study group (with left ventricular systolic dysfunction) only 2 (4%) were absolutely normal. Among the control group 70% of the electrocardiograms were normal.

	Control	Study
Normal ECG	35	2
Abnormal ECG	15	48

Age distribution

The age distribution of the study group was tabulated. The youngest person was 24 years old and the oldest patient was 84years old. The average age of the study group was 54.7 years.

	Study	Control
18-29	2	4
30-39	6	13
40-49	9	11
50-59	12	10
60-69	15	9
70-79	5	2
80-89	2	1
Average	54.7	47.7

Sex distribution

Majority of the patients were male both in the control population and in the study population.

	Study	Control
Male	30 (60%)	30 (60%)
Female	20 (40%)	20 (40%)
Total	50	50

Etiology of Heart failure

In our study majority (68%) of the patients had ischemic heart disease. The other causes included hypertensive heart disease, valvular heart disease and cardiomyopathies.

Etiology	Study
Coronary artery disease	32
Hypertensive heart disease	5
Valvular heart disease	7
Cardiomyopathies	6

Variation in heart rate

The average resting heart rate in the study group was 99.8 with a range from 72 to 124. Whereas the average heart rate in the control population was 80.16 with a range from 60 to 110.

	Control	Mild	Moderate	Severe
Normal rate (60 to 100)	47	15	8	2
Sinus tachycardia (>100)	3	4	10	4
Average heart rate	80.16	93.3	103.6	110.5

Rhythm abnormalities

As the Ejection fraction decreased, then incidence of atrial and ventricular ectopics increased. 94% of the control population had normal sinus rhythm. This decreased to 42% among those with left ventricular systolic dysfunction.

	Control	Mild	Moderate	Severe
Normal sinus rhythm	47	15	9	2
Atrial ectopics	3	3	4	4
Ventricular ectopics	4	4	5	5
Atrial fibrillation	0	5	0	2

Axis deviation

Normal axis was present in 96% of the control population, whereas only 68% of the study population had normal axis. The mean axis in control population was 37.5 (range -40 to 70) and in the study was 20.9 (range -60 to 120).

	Control	Mild	Moderate	Severe
Normal Axis	48	17	14	3
Left axis deviation	2	4	3	5
Right axis deviation	0	3	1	0
Extreme axis	0	0	0	0
Average axis	37.5	36.2	19.16	-21.2

Chamber hypertrophy

12% of the control population had left ventricular hypertrophy. Among the study population 36% had LV hypertrophy and 4% had RV hypertrophy.

	Control	Mild	Moderate	Severe
LV hypertrophy	6	11	4	3
RV hypertrophy	0	2	0	0
No hypertrophy	44	11	14	5

Conduction Abnormalities

Normal conduction was present in 46 (92%) persons of the control population. This decreased to 33 (66%) in the study population. The most common form of conduction defects was Left Anterior Fascicular block.

	Control	Mild	Moderate	Severe
Normal conduction	46	20	11	2
LAFB	2	4	1	3
RBBB	1	1	2	2
LBBB	1	1	0	4

Left Atrial Abnormality

Abnormal wide P waves were present in 48% of the persons with left ventricular systolic dysfunction, but occurred only in 10% of them with normal left ventricular systolic function.

	Study	Control
Normal p	26	45
Abnormal p (LA Abnormality)	24	5

QRS Duration

Average QRS duration in the control population was 86.6 milliseconds (range 75 to 110). In the group with left ventricular systolic dysfunction it was 107.4 milliseconds (range 80 to 140). None of the patients in the study population had a QRS duration more than 120 milliseconds. On the other hand 30% of the study population had QRS duration more than 120 milliseconds

QSR duration	Control	Mild	Moderate	Severe
70-79	1	0	0	0
80-89	27	6	1	0
90-99	19	7	3	0
100-109	2	4	5	0
110-119	1	4	4	1
120-129	0	1	4	1
130-139	0	1	1	2
140-149	0	1	0	4
Average	86.6	99.1	107.5	131.85

Combination of criteria

Combination 1: Heart rate more than 90, left atrial abnormality and QRS duration >110 milliseconds

Combination 2: Heart rate more than 90, Axis < 0 or > 90 and QRS duration > 110 milliseconds

Combination 3: Heart rate more than 90, and QRS duration >110 milliseconds

	Control	Study	Mild	Moderate	Severe
Combination 1	0	11 (22)	5 (20)	3 (16.6)	3(37.5)
Combination 2	0	12 (24)	2 (8)	4 (22.2)	6 (75)
Combination 3	0	22 (44)	7 (29)	8 (44.4)	7(87.5)

None of the control population had more than one criteria present. Hence none of the combination criteria was present. A combination of Heart rate more than 90, and QRS duration >110 milliseconds (combination 3) was present in 87.5 % of patients with very low ejection fraction. However all 3 three combinations were not present in majority of patients with mild LV dysfunction.

Summary

Table showing in percentage the difference between the heart rate, left atrial abnormality (LAA), ectopics, ventricular hypertrophy, and increased QRS duration between the control population and the study population.

	Control	Study
Tachycardia	6	34
L. Atrial Abnorm	10	48
LAFB	4	16
Bundle blocks	4	20
LVH/RVH	12	40
QSRD > 120 ms	0	30

Discussion

A total of 100 patients were analyzed in this study of which 50 were patients with left ventricular systolic dysfunction (EF < 50%) and 50 patients with normal left ventricular function. Analysis of the data shows us that majority of the patients were male (60%) both in the study and control population.

The most common cause of heart failure in this study was coronary artery disease (64%). Similar findings were observed in other studies.[13]

48 patients in the study population had ECG abnormalities (96%). In contrast only 30% had abnormal electrocardiograms among the control population. Hence the negative predictive value of a normal electrocardiogram is very high.

Our study showed that as the Ejection fraction decreased the resting heart increased. The average heart rate in the control population was 80.1 per minute. It increased to 93.3 per minute in the control population. In patients with severe left ventricular systolic dysfunction it was still higher (110.5 per minute). 34 % of the control population had sinus tachycardia and 14% had atrial fibrillation. In strong contrast only 6% of the control population had sinus tachycardia and none had atrial

fibrillation. Hence an increasing heart rate is associated with low EF but should be interpreted with the help of associated findings.

Axis deviation was more in patients with left ventricular systolic dysfunction (32% compared to 4% among control population). Among this left axis deviation was more common (24%) than right axis deviation (12%). However axis deviation cannot be taken as a significant ECG criterion as majority of the patients even with left ventricular systolic dysfunction (i.e. 68%) had normal axes.

In our study the QRS duration was the single most important ECG criterion strongly associated with left ventricular systolic dysfunction. As the QRS duration increased the EF decreased. The average QRS duration in the control population was 86.6 milliseconds (range 75 to 110). This increased to 107.8 ms in the study population. Moreover among patients with severe left ventricular systolic dysfunction the average QRS duration was 131.85 milliseconds. Looking at the incidence, none of the patients in the study group had QRS duration more than 120 milliseconds. This rose to 30 % among the control group. Among patients with severe left ventricular systolic dysfunction 87.5% had a QRS duration more than 120 milliseconds.

These results are similar to the conclusions of several studies which have proved that intraventricular conduction delay is associated

with more advanced myocardial disease including those by A P Davie, Amir Kashani, Das MK, Cheripambil K, Bedi A, et al.[33, 34, 35]

Left atrial abnormality as evidenced by abnormal P waves is another ECG criterion closely associated with left ventricular systolic dysfunction. In our study 48 % of the patients with left ventricular systolic dysfunction had left atrial abnormality, while it was present in only 10% of the control population. Thus in the absence of systemic hypertension and mitral valve disease, left atrial abnormality can be taken as an indicator to the presence of left ventricular dysfunction.

34% of our patients in study population had conduction abnormalities, compared to only 8 % of the control population. The most common abnormality was Left anterior fascicular block. However, in patients with severe left ventricular systolic dysfunction 50 % had a left bundle branch block. Studies done by Das MK, Cheripambil K, Bedi A, et al and Murkofsky RL et al also confirms similar observation that LBBB is a marker of significant left ventricular systolic dysfunction.[35, 36]

12% of the control population had left ventricular hypertrophy. Among the study population 36% had LV hypertrophy and 4% had RV hypertrophy. Although incidence of LV hypertrophy is more in the study population, chamber hypertrophy is both non specific and non sensitive in detecting left ventricular systolic dysfunction.

Finally in this study combination of several ECG criteria were used. A combination of heart rate more than 90, left atrial abnormality and QRS duration >110 milliseconds was present in 37.5% of patients with severe LV dysfunction but in only 20% of the mild LV dysfunction group. Another combination of Heart rate more than 90, and QRS duration >110 milliseconds was present in majority (87.5%) of patients with severe LV dysfunction but in only 29% of the mild LV dysfunction group. However none of the control population had more than single ECG abnormality. Hence use of combination criteria would only detect patients with severe LV dysfunction.

Limitations of our study

1. The etiology of heart failure in most of the patients in the study population was coronary artery disease. The electrocardiographic changes produced by ischemia in those patients could be a source of error.
2. In view of the high incidence of coronary artery disease, certain criteria such as Q waves, ST segment and T wave changes were not used for analysis.
3. This study had certain confounding factors such as age, emphysema, obesity, electrolyte imbalance, drugs etc. which could alter the surface electrocardiogram.
4. The study population is small and the design was retrospective. A larger study population and a prospective study may have produced different results.

Conclusions

1. Coronary artery disease is the most common cause of left ventricular systolic dysfunction.
2. There is a consistent association between the standard 12 lead electrocardiogram and left ventricular systolic dysfunction.
3. A **norma**l electrocardiogram virtually excludes chronic heart failure due to left ventricular systolic dysfunction.
4. There are usually major electrocardiographic abnormalities as the severity of left ventricular systolic dysfunction increases.
5. The resting heart rate increases consistently with decreasing left ventricular systolic function.
6. Ectopic activity (Both Atrial and ventricular) increases with decreasing left ventricular ejection fraction.
7. Conduction defects increases with worsening Left ventricular systolic function. Left anterior fascicular block is the most common conduction abnormality. However a Left bundle branch block is more common in those with severe left ventricular systolic dysfunction.
8. Increasing QRS duration is the single most specific criteria which correlates well with decreasing ejection fraction. A QRSD duration more than 120 milliseconds especially in the absence of a typical

conduction defect signifies significant left ventricular systolic dysfunction

9. Among the electrocardiographic abnormalities a combination of sinus tachycardia, left Atrial abnormalities and QRS duration were found to be most specific but loses sensitivity.
10. The electrocardiogram is not a substitute for echocardiography, as an abnormal electrocardiogram does not accurately predict the presence of left ventricular systolic dysfunction.
11. An abnormal electrocardiogram does not mean that the patient has chronic heart failure but is an indication for an echocardiogram.

Master Chart – study population - I

S.No.	Name	AGE	Sex	Cause	EF	Rate	Rhythm	Axis	LAA	LAFB	QRSD	APC	VPC	LVH	RVH	RBBB	LBBB
1	Raghu	35	M	CM	25	120	ST	-40	+	+	140	--	--	--	--	+	--
2	Vaidyanathan	50	M	CAD	35	124	ST	-45	+	--	85	+	--	--	--	--	--
3	Revathi	56	F	CAD	40	78	NSR	30	--	--	110	--	--	+	--	--	--
4	Muhtulaxmi	84	F	CAD	42	120	AF	25	NA	--	120	NA	+	--	--	--	--
5	Varadhan	65	M	Valv	44	98	NSR	40	+	--	110	--	--	--	--	--	--
6	Destagiri	58	M	CAD	40	102	ST	-50	+	+	130	+	+	--	--	--	+
7	Padma	40	F	CAD	48	90	NSR	30	--	--	110	--	--	+	--	--	--
8	Devika	25	M	Valv	40	75	AF	120	+	--	90	+	--	+	--	--	--
9	Jeyaraman	80	M	CAD	45	110	ST	50	--	--	80	--	--	--	--	--	--
10	Mary	32	F	CM	35	98	NSR	-35	+	+	125	--	--	--	--	+	--
11	Yunus	63	M	CAD	28	72	NSR	-60	+	+	110	+	+	+	--	--	--
12	Thulukanam	52	M	Valv	39	110	ST	45	+	--	100	--	--	+	--	--	--
13	AbdulMazzed	75	M	CM	30	98	NSR	60	--	--	120	--	+	--	--	--	--
14	Ulaganathan	51	M	CM	25	100	NSR	20	--	--	140	--	--	--	--	--	--
15	Ahamed	65	M	CAD	32	110	ST	35	--	--	95	--	+	+	--	--	--
16	Annaraj	46	M	CAD	42	75	NSR	35	+	--	85	--	--	--	--	--	--
17	Samuel	24	M	Valv	49	100	AF	110	NA	--	90	NA	+	+	+	--	--
18	Aarthi	42	F	CAD	38	98	NSR	60	--	--	90	--	--	--	--	--	--
19	Devika	70	F	CAD	42	110	ST	55	+	--	95	--	--	--	--	--	--
20	James	40	M	HHD	49	86	AF	30	NA	--	100	--	--	+	--	--	--
21	Allah Basha	58	M	CAD	30	120	ST	-35	--	--	125	+	--	--	--	+	--
22	Parvathi	60	F	CAD	28	122	ST	-45	+	--	130	+	--	+	--	--	+
23	Laxmi	62	F	CAD	39	98	NSR	100	+	--	105	--	--	+	--	--	--
24	Mohammed	55	M	HHD	45	74	NSR	30	+	--	85	--	--	+	--	--	--
25	Prabu	65	M	CM	26	112	AF	-35	NA	+	140	+	+	--	--	+	--

Master Chart – study population - II

S.No.	Name	Age	Sex	Cause	EF	Rate	Rhythm	Axis	LAA	LAFB	QRSD	APC	VPC	LVH	RVH	RBBB	LBBB
26	Prasanna	48	M	CAD	45	86	NSR	50	--	--	85	--	--	--	--	--	--
27	Anandhi	54	F	CAD	38	86	NSR	40	--	--	100	--	+	--	--	--	--
28	Kavya	66	F	CAD	32	110	ST	30	+	--	110	+	--	--	--	--	--
29	Rajan	47	M	Valv	45	100	NSR	30	+	--	110	--	--	+	--	--	--
30	Vijay Anand	68	M	CAD	25	120	ST	-50	--	--	140	--	+	--	--	--	+
31	Meghala	58	F	CAD	40	82	NSR	55	--	--	85	+	--	--	--	--	--
32	Hameedha	53	F	CAD	39	86	NSR	40	+	--	95	--	--	+	--	--	--
33	Hareesh	60	M	CAD	45	92	NSR	50	--	--	80	--	+	--	--	--	--
34	Vijay Kanna	58	M	CAD	30	112	ST	10	--	--	120	--	+	--	--	--	--
35	Armugam	31	M	HHD	49	92	NSR	-50	+	+	90	--	--	+	--	--	--
36	Barathan	65	M	CAD	31	98	NSR	15	--	--	110	--	+	--	--	--	--
37	Vasanthi	75	F	CAD	49	100	NSR	50	--	--	100	--	--	+	--	--	--
38	Sundeep	72	M	CAD	34	112	ST	-20	--	--	105	--	--	--	--	--	--
39	Rohini	39	F	Valv	45	96	AF	120	NA	--	95	+	--	--	+	--	--
40	Karpagam	48	F	HHD	41	98	NSR	-40	+	+	90	--	+	+	--	--	--
41	Kannan	62	M	CAD	32	112	ST	00	--	--	115	--	+	--	--	--	--
42	Satheesh	64	M	CAD	47	98	ST	10	+	--	90	--	--	+	--	--	--
43	Vidya	48	F	CAD	21	120	AF	60	NA	--	130	NA	+	--	--	--	+
44	Hariprasad	52	M	CAD	42	86	NSR	55	--	--	110	--	--	--	--	--	--
45	Shakunthala	68	F	CAD	39	110	ST	35	+	--	115	--	--	--	--	--	--
46	Guruprasath	35	M	CM	25	118	ST	-20	+	--	125	+	+	+	--	--	+
47	Prakash	72	M	CAD	49	94	NSR	70	+	--	100	--	--	--	--	--	--
48	Ravindar	42	M	HHD	40	98	NSR	-35	+	+	140	--	--	+	+	+	--
49	Kalpana	32	F	Valv	38	112	ST	00	+	--	100	--	--	--	--	--	--
50	Geetha	65	F	CAD	35	82	NSR	10	--	--	120	+	--	--	--	--	--

Master Chart – control population - I

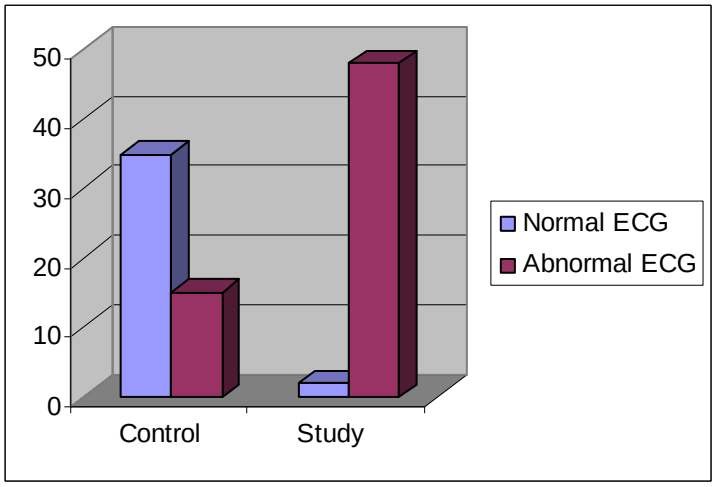
S.No.	Name	AGE	Sex	EF	Rate	Rhythm	Axis	LAA	LAFB	QRSD	APC	VPC	LVH	RVH	RBBB	LBBB
1	Prasanna	48	M	55	72	NSR	25	--	--	85	--	--	--	--	--	--
2	Radha	54	F	65	68	NSR	35	--	--	85	--	--	--	--	--	--
3	Kiran joseph	66	M	50	88	NSR	40	--	--	90	--	--	--	--	--	--
4	Shyam	47	M	60	62	NSR	-10	--	--	80	--	+	--	--	--	--
5	Prathap	68	M	65	85	NSR	55	--	--	90	--	--	--	--	--	--
6	Vidya	58	F	70	74	NSR	10	+	--	80	--	--	+	--	--	--
7	Hareesh	53	M	65	70	NSR	50	--	--	90	--	--	--	--	--	--
8	Sunil	40	M	52	84	NSR	60	--	--	100	--	--	--	--	--	--
9	Preethi	58	F	54	72	NSR	65	--	--	90	--	--	--	--	--	--
10	Laxmi	31	F	68	74	NSR	70	--	--	85	--	--	--	--	--	--
11	Arun rathnam	65	M	63	85	NSR	65	--	--	85	+	--	--	--	--	--
12	Shanthakumar	35	M	70	69	NSR	00	--	--	80	--	--	--	--	--	--
13	Joel	50	M	58	93	NSR	54	--	--	75	--	--	--	--	--	--
14	Anuradha	56	F	56	73	NSR	40	--	--	80	--	--	--	--	--	--
15	Gnanam	70	M	50	80	NSR	10	--	--	85	--	--	--	--	--	--
16	Anjana	65	F	63	85	NSR	70	--	--	90	--	--	--	--	--	--
17	Bennett	58	M	67	70	NSR	15	--	--	90	--	+	--	--	--	--
18	Ponammal	40	F	62	94	NSR	56	--	--	85	--	--	--	--	--	--
19	Sandhya	25	F	68	98	NSR	50	--	--	85	--	--	--	--	--	--
20	Ahmed	80	M	60	104	ST	-35	+	+	100	--	--	+	--	--	--
21	Kalyan	32	M	55	74	NSR	35	--	--	90	--	--	--	--	--	--
22	Nimbi	63	M	55	79	NSR	40	--	--	80	--	--	--	--	--	--
23	Preethi	52	F	54	73	NSR	42	+	--	90	--	--	+	--	--	--
24	Sangeetha	45	F	60	72	NSR	40	--	--	80	--	--	--	--	--	--
25	Narayanan	35	M	68	60	NSR	40	--	--	90	+	--	--	--	--	--

Master Chart – control population - II

S.No.	Name	Age	Sex	EF	Rate	Rhythm	Axis	LAA	LAFB	QRSD	APC	VPC	LVH	RVH	RBBB	LBBB
26	Fernandez	42	M	52	86	NSR	-10	--	--	85	--	--	--	--	--	--
27	Gayathri	39	F	57	86	NSR	64	--	--	90	--	--	--	--	--	--
28	Yunus khan	48	M	68	90	NSR	65	--	--	90	+	--	--	--	--	--
29	Prem kumar	62	M	72	79	NSR	62	--	--	85	--	--	--	--	--	--
30	Robert	64	M	64	60	NSR	-40	--	+	110	--	--	+	--	--	+
31	Seethalaxmi	48	F	65	82	NSR	52	--	--	90	--	--	--	--	--	--
32	Bharath	52	M	62	86	NSR	58	--	--	80	--	--	--	--	--	--
33	Amrutha	68	F	58	92	NSR	50	--	--	90	--	--	--	--	--	--
34	Ravikumar	35	M	52	68	NSR	60	--	--	80	--	+	--	--	--	--
35	Dinesh	32	M	58	73	NSR	30	+	--	90	--	--	+	--	--	--
36	Hameedha	42	F	50	90	NSR	63	--	--	85	--	--	--	--	--	--
37	Kandan	32	M	60	73	NSR	20	--	--	85	--	--	--	--	--	--
38	Menon	65	M	66	79	NSR	65	--	--	90	--	--	--	--	--	--
39	Keerthi	52	M	63	65	NSR	10	--	--	85	--	--	--	--	--	--
40	Tarakeshwari	39	F	70	65	NSR	68	--	--	90	--	--	--	--	--	--
41	Ajay	28	M	65	78	NSR	25	--	--	90	--	--	--	--	--	--
42	Srividya	34	F	54	90	NSR	35	--	--	85	--	--	--	--	--	--
43	Deepa	70	F	68	110	ST	40	--	--	80	--	+	--	--	--	--
44	Lawerance	31	M	52	86	NSR	42	--	--	80	--	--	--	--	--	--
45	Chandran	45	M	58	100	ST	15	--	--	85	--	--	--	--	--	--
46	Shakunthala	28	F	70	70	NSR	40	--	--	85	--	--	--	--	--	--
47	Nandhini	30	F	53	94	NSR	20	+	--	90	--	--	+	--	--	--
48	Radhika	41	F	50	98	NSR	40	--	--	80	--	--	--	--	+	--
49	Guru Prasad	29	M	60	68	NSR	45	--	--	90	--	--	--	--	--	--
50	Jeyakumar	35	M	65	82	NSR	35	--	--	80	--	--	--	--	--	--

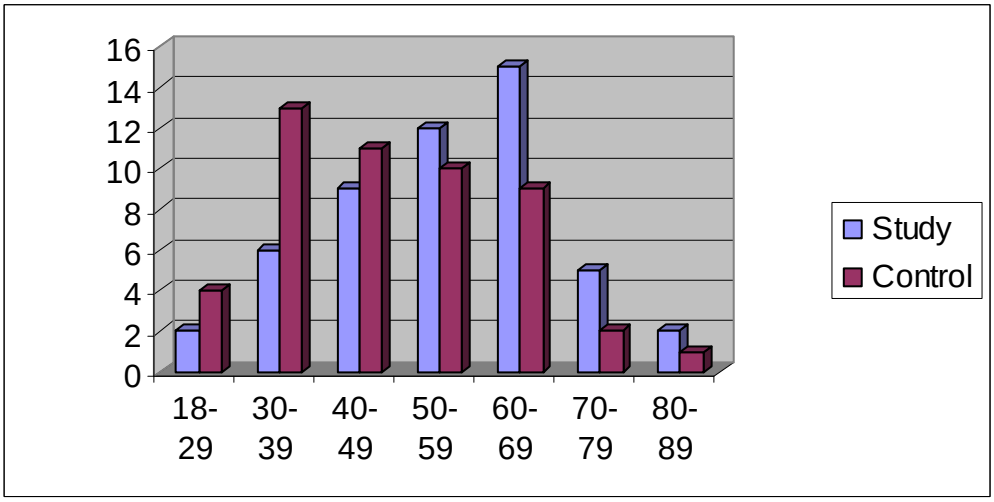
Bar diagram showing number of normal and

abnormal ECGs between the populations



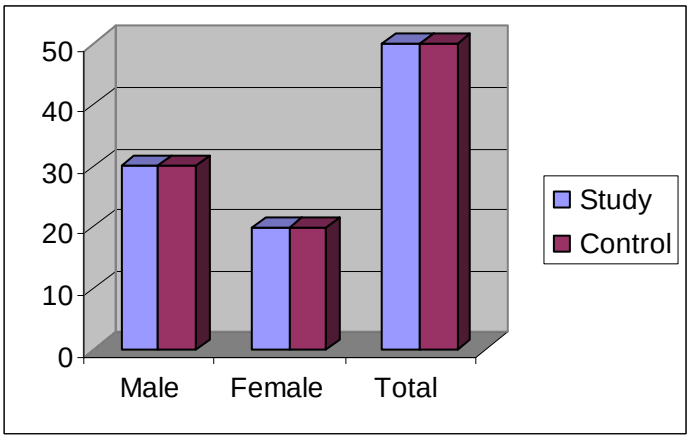
Bar diagram showing Age distribution

among the Study and control populations



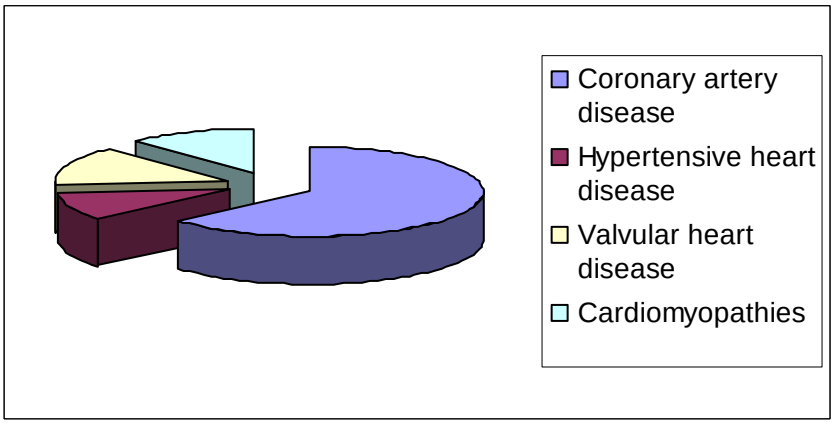
Bar diagram showing sex distribution

among the Study and control populations



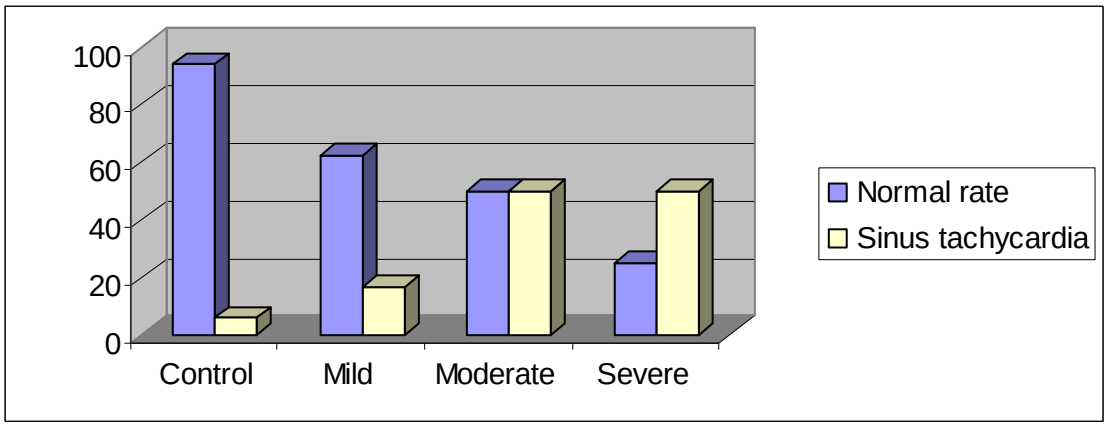
Bar diagram showing etiology of

LV dysfunction among the Study population



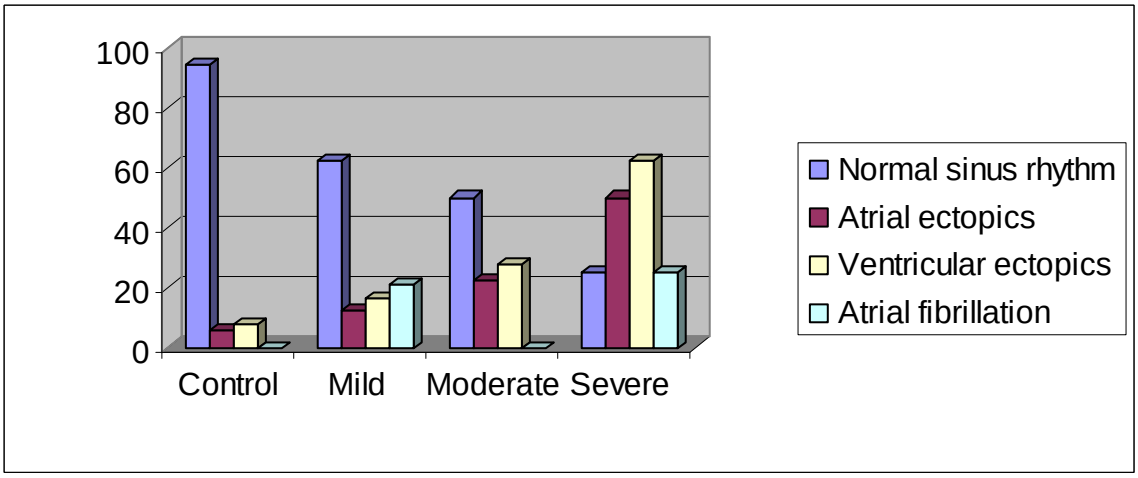
Bar diagram showing normal rates and tachycardia

among the Study and control populations in percentage



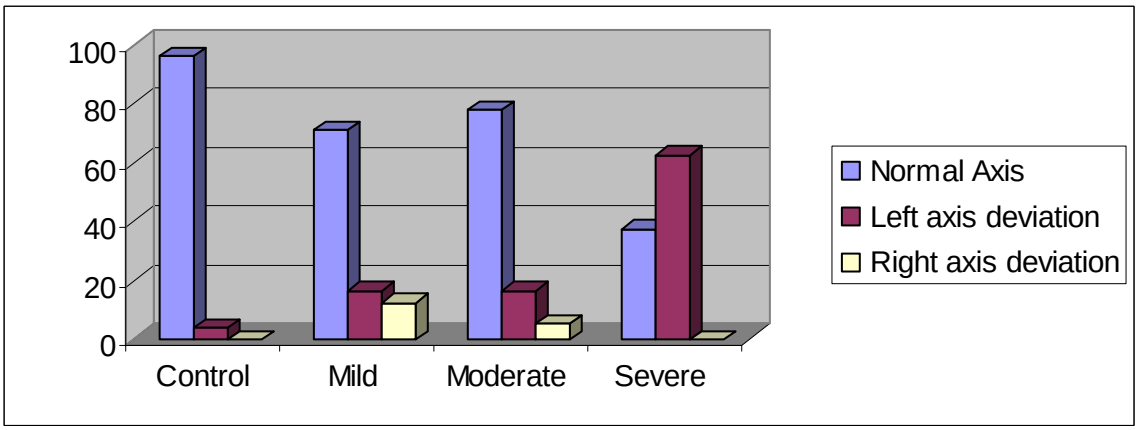
Bar diagram showing various rhythms (percentage)

among the Study and control populations



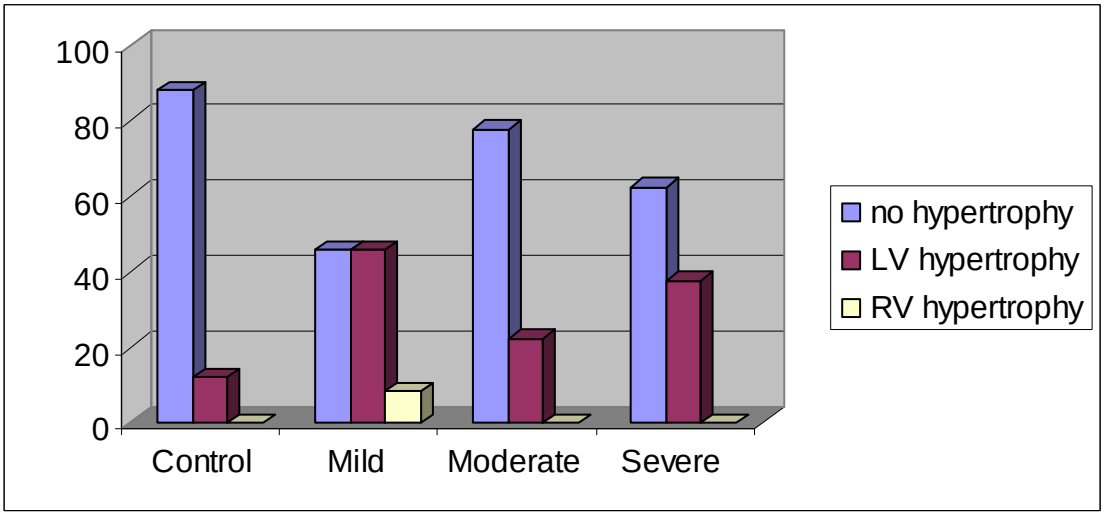
Bar diagram showing normal and abnormal axis

among the Study and control populations in percentage



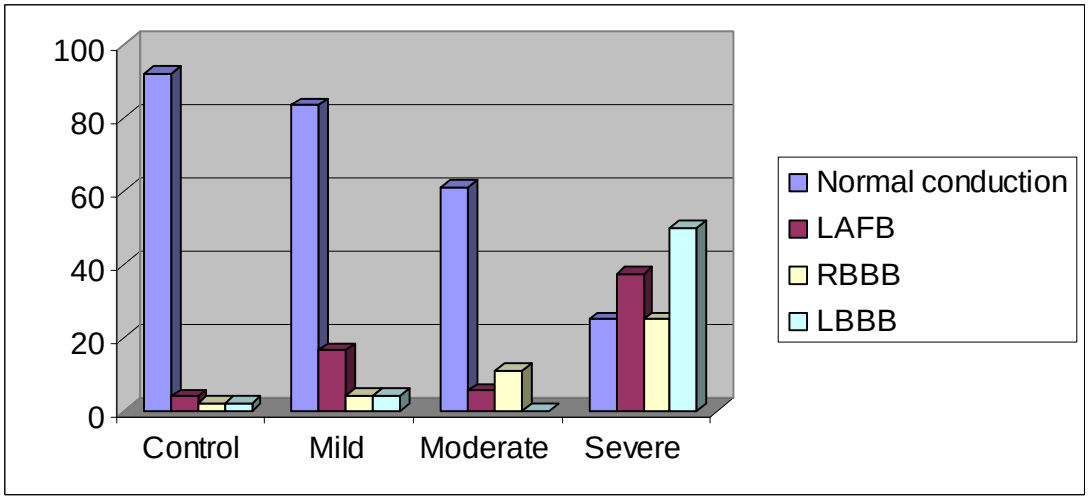
Bar diagram showing ventricular hypertrophy

among the Study and control populations in percentage



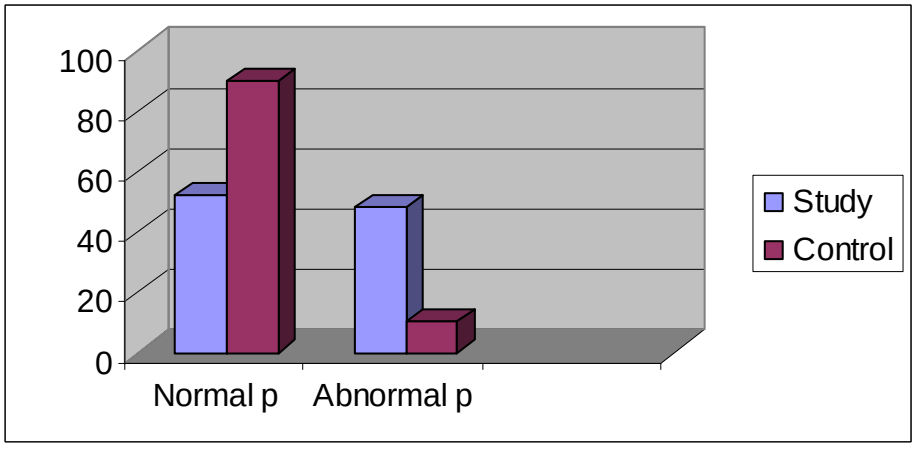
Bar diagram showing conduction defects (percentage)

among the Study and control populations



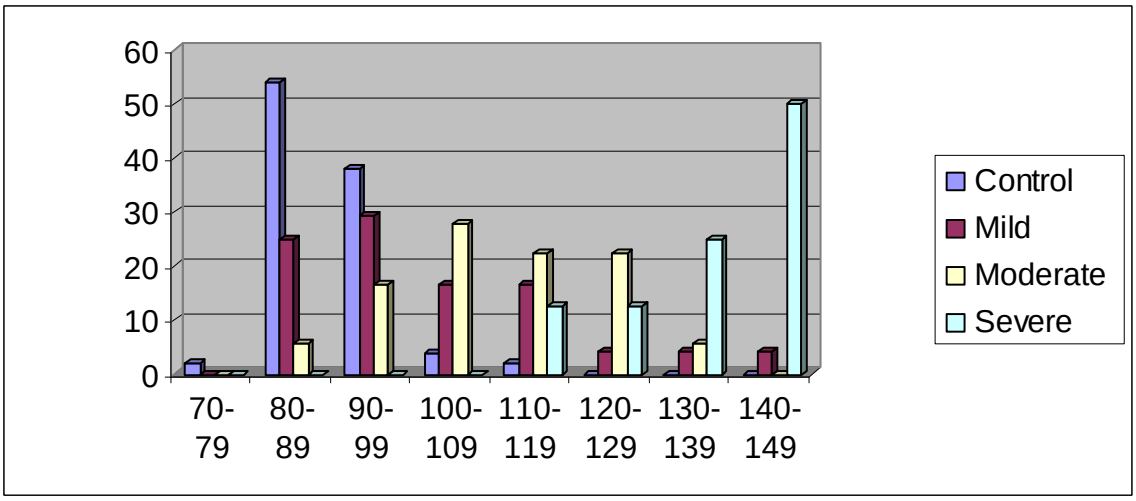
Bar diagram showing Left atrial abnormality (Abnormal P)

among the Study and control populations



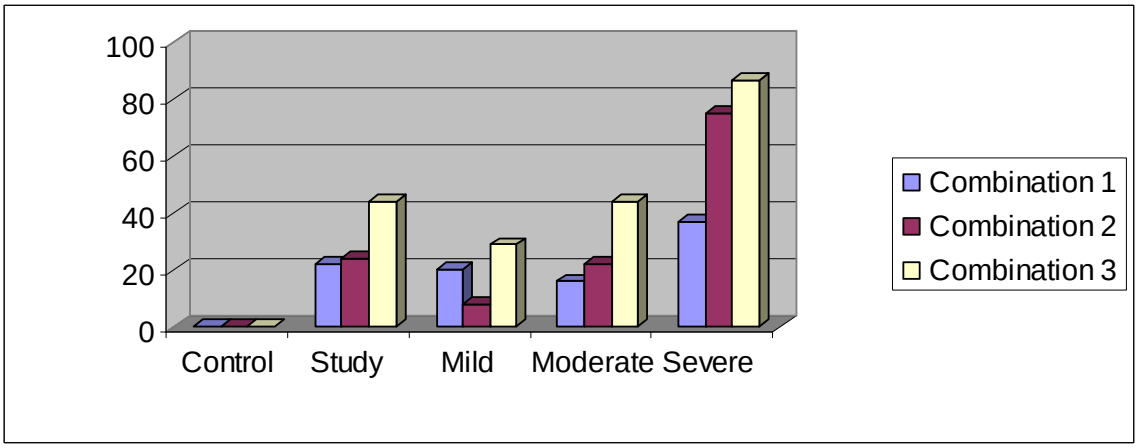
Bar diagram showing QRS duration in milliseconds

among the Study and control populations



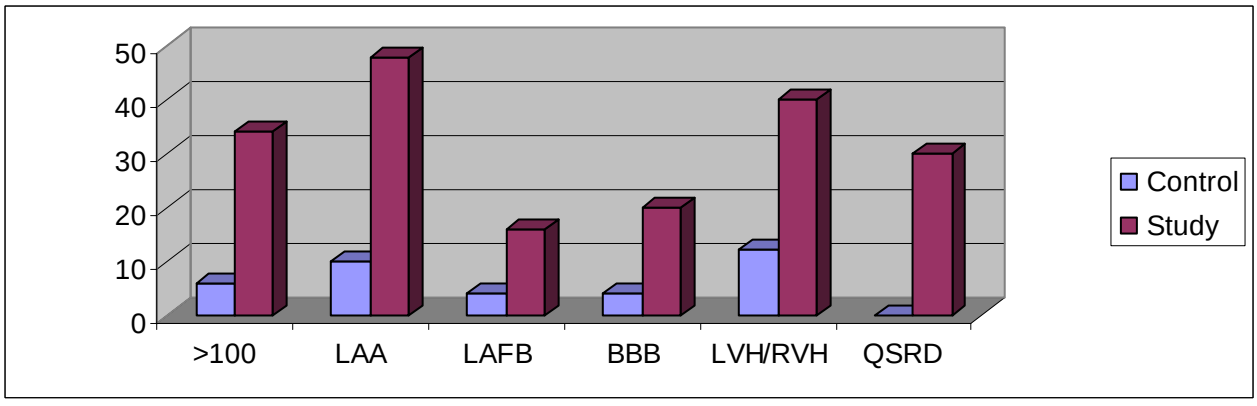
Bar diagram showing various combination of ECG criteria

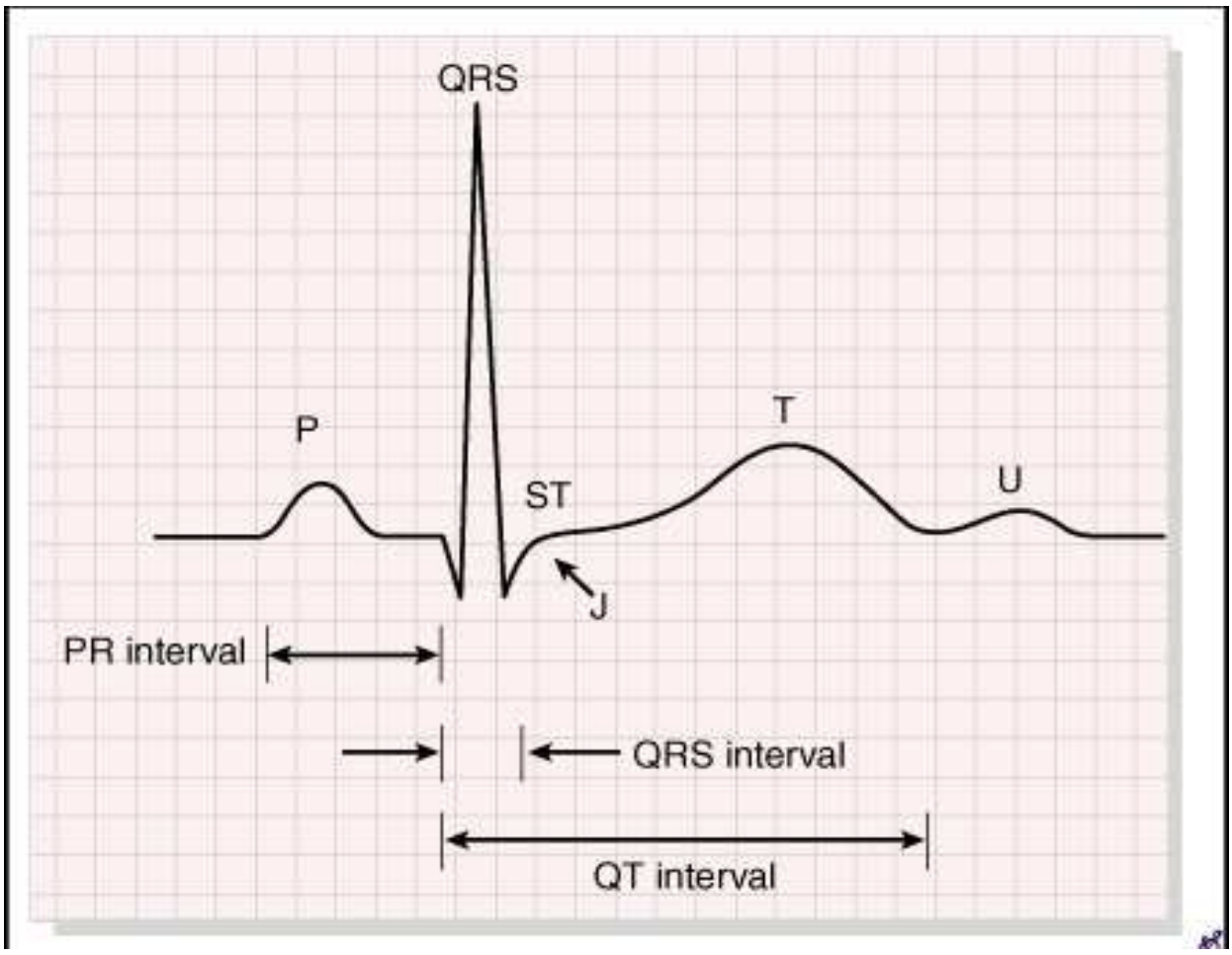
among the Study and control populations



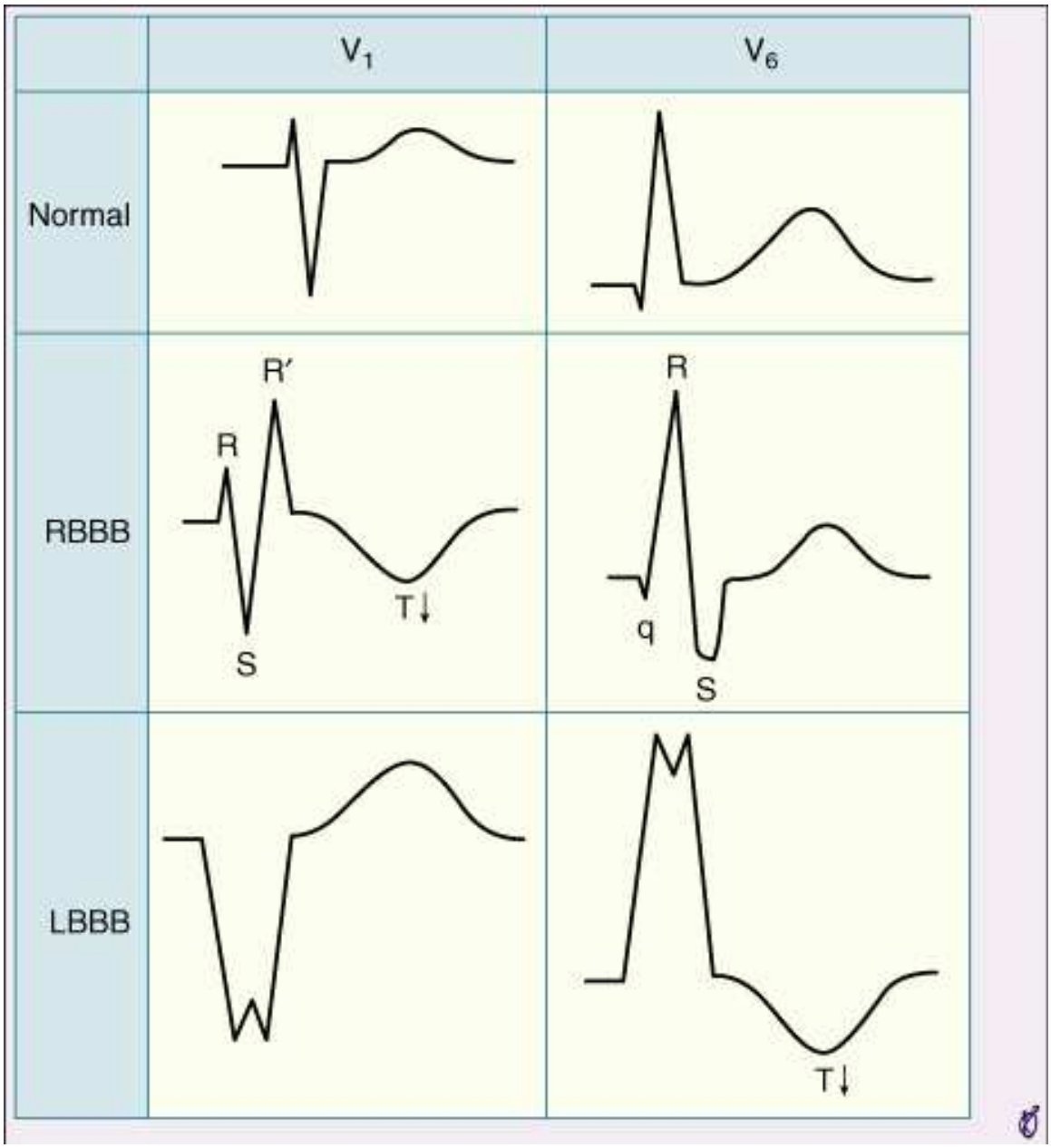
Bar diagram showing various ECG criteria (in %)

among the Study and control populations

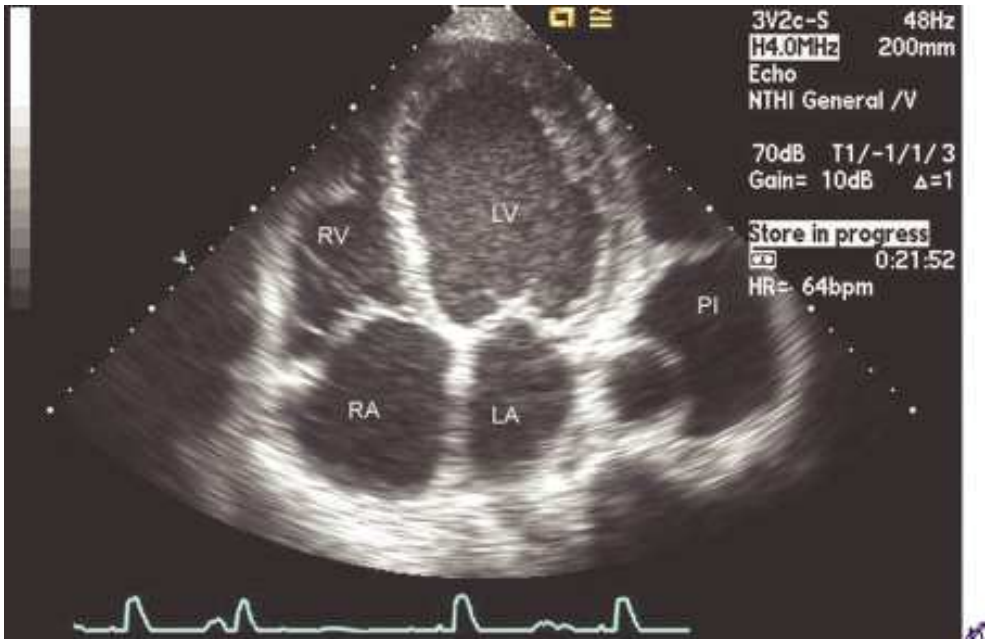




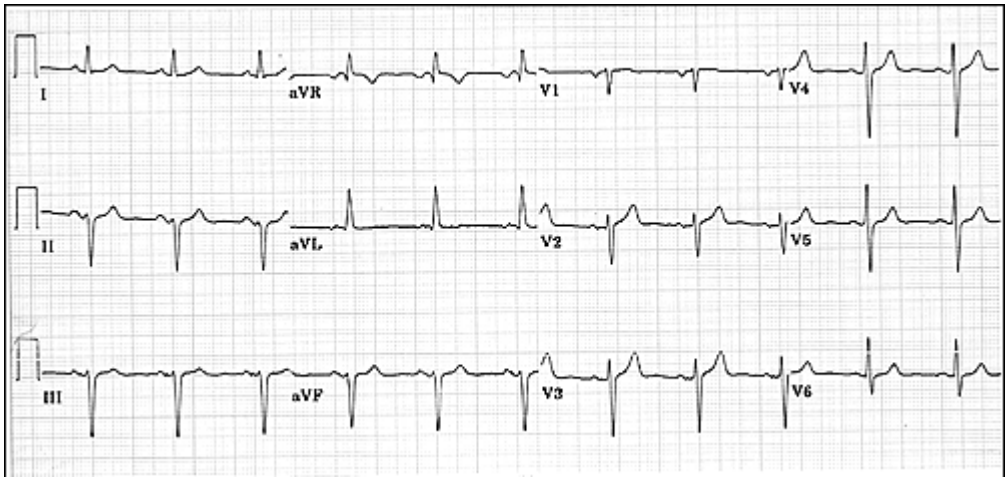
A diagram showing basic ECG waves and intervals



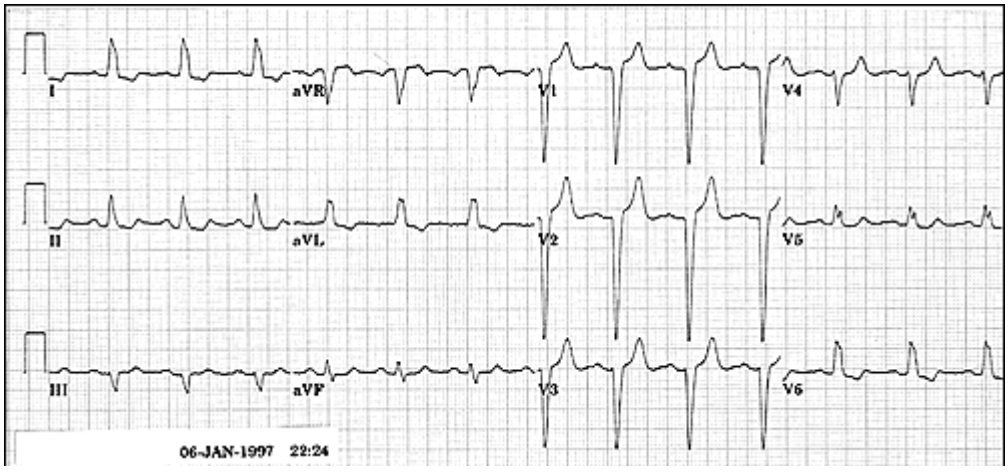
An illustration depicting right and left bundle branch blocks



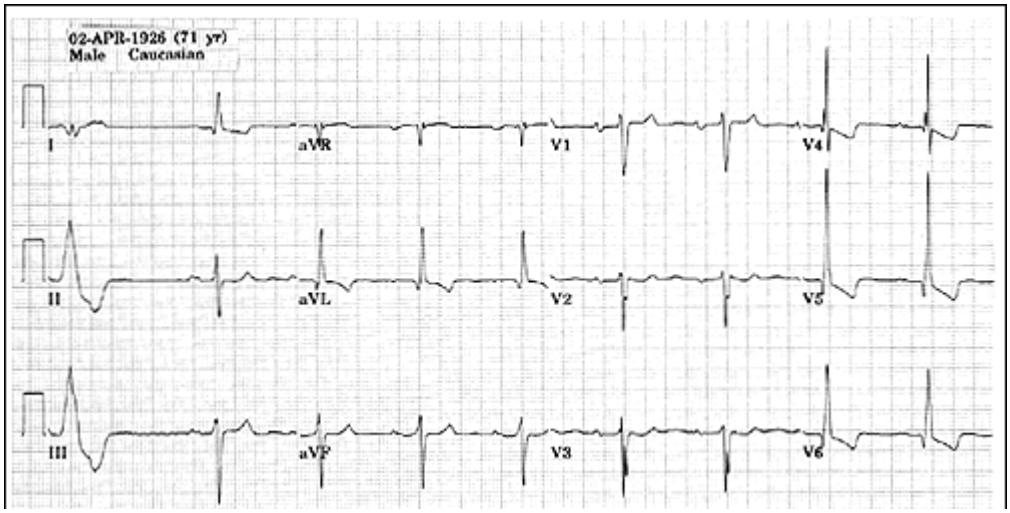
Apical four-chamber view of a patient with a dilated cardiomyopathy as visualize by Echocardiography.



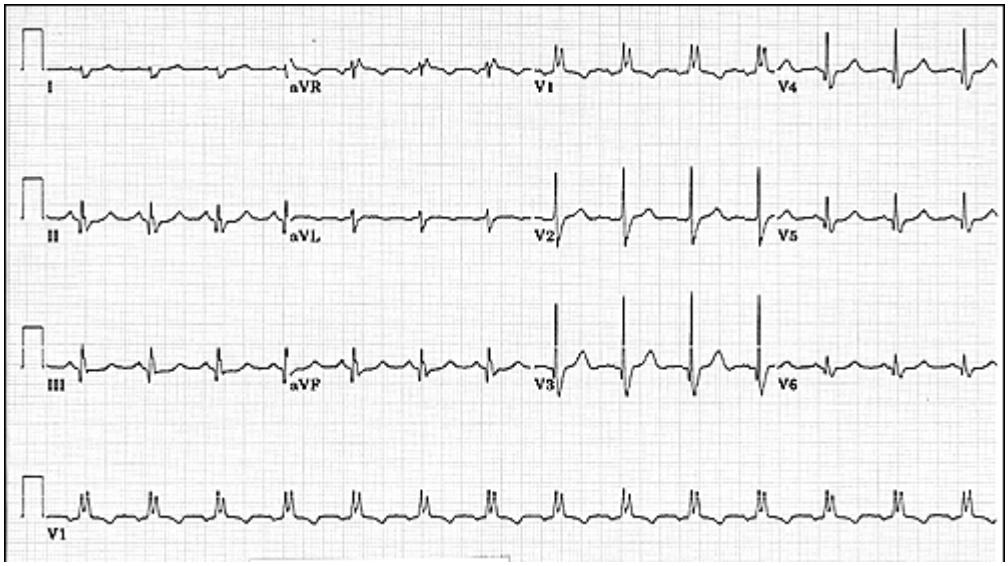
This ECG shows left anterior fascicular block



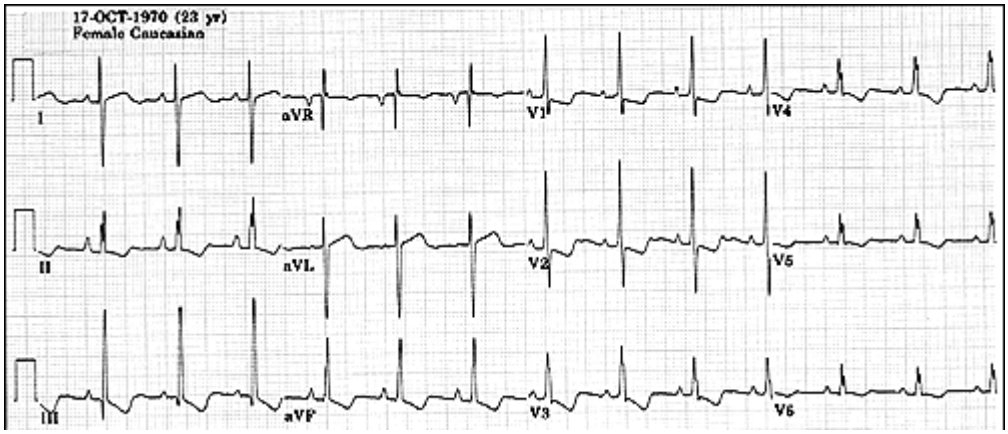
This ECG shows left bundle branch block



This ECG shows left ventricular hypertrophy, left atrial abnormality and a ventricular ectopic



This ECG shows right bundle branch block



This ECG shows right axis deviation and right ventricular hypertrophy

Study Performa

Name

Age

Sex

O.P. No.

Current NYHA Class

Ejection Fraction

Etiology of Heart Failure

Rate

- Normal
- Bradycardia
- Tachycardia

Rhythm

- Normal Sinus
- Atrial Ectopics
- Ventricular Ectopics
- Atrial Arrhythmias
- Ventricular Arrhythmias

Axis

- Normal

Left Axis Deviation

Right Axis Deviation

P Wave Morphology

Normal

Left Atrial Abnormality

QRS duration

<120 ms

>120 ms

Conduction Defects

None

Right Bundle Branch Block

Left Bundle Branch Block

Left Anterior Fascicular Block

Others

Left Ventricular Hypertrophy

Present

Absent

Right Ventricular Hypertrophy

Present

Absent

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