LEFT VENTRICULAR MASS IN HYPERTENSION - CORRELATIVE STUDY OF ELECTROCARDIOGRAPHY & ECHOCARDIOGRAPHY

Submitted By

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In partial fulfillment of the
Requirements for the degree of
MD

in

General Medicine

Under the Guidance of
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2010

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"LEFT VENTRICULAR MASS IN HYPERTENSION – CORRELATIVE STUDY OF ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY**" is a bonafide and genuine research work carried out by me under the guidance of **Dr. S. S. DEVARMANI**$_{MD}$ Professor of Medicine.

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Date:

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LIST OF ABBREVIATIONS USED

ABP.....Ambulatory Blood Pressure

ACE.....Angiotensin Convertase Enzyme

AT1 .....Angiotensin 1

AT2 .....Angiotensin II

BP.....Blood Pressure

Ca++.....Calcium

CHF.....Congestive Cardiac Failure

CO .....Cardiac Output

CV .....Cardiovascular

ECG.....Electrocardiography

ECHO……...Echocardiography

EO .....Endogenous Ouabain

EDV .....Endothelium Dependant Vasodilatation

EH .....Essential Hypertension

gms ……. Grams

K+ .....Potassium

LV .....Left ventricle

LVH .....Left ventricular hypertrophy

LVM.....Left Ventricular Mass

LVIDD……. Left ventricular internal diameter at end diastole

LVP WT-d…….. Left ventricular posterior wall thickness.

LAD …… Left axis deviation

IVS-d …… Interventricular septal thickness at end diastole.

Mg++ .....Magnesium

NCX .....Sodium Calcium Exchanger

Na+.....Sodium

OBP.....Office Blood Pressure

OU.....Ouabain

RWT.....Relative Wall Thickness

PWT……..Posterior wall thickness

TPR .....Total Peripheral Resistance

TGF-B .....Transforming Growth Factor Beta

+ve…… Positive

-ve……. Negative

ABSTRACT

BACKGROUND: Left ventricular hypertrophy {LVH} has been repeatedly shown to be associated with a marked increase in cardiovascular risk. The ECG evidence of LVH is present only in 50% of patients with anatomic LVH. ECG may be entirely normal in around 15% patients with severe LVH. The purpose of this study is to determine the sensitivity of ECG in diagnosis of left ventricular hypertrophy by its correlation with echocardiography of left ventricular mass in hypertensive patients.

MATERIALS AND METHODS: The study was carried out on 130 patients attending the hospital with systemic hypertension diagnosed by JNC VII criteria and managed at B.L.D.E A's Shri B.M Patil Medical College and Hospital. 12 lead ECG and Echo cardiograph examination for assessment of left ventricular function and mass was done using M mode and 2D echocardiography. Left ventricular mass was calculated in each case using the Devereux Formula. LV Mass=1.04 [{IVS (d)+ LVID (D)+ LVPWT (d)}^{3}]- [LVID (d)]^3- 13.6gms where 1.04 is the specific gravity of cardiac muscle. Comparison was done with a control group of 100 patients. The correlation of ECG data and echo cardio graph findings in relation to LV mass was carried out in each case.

OBSERVATIONS: In all the patients in study group ECG evidence of LVH was calculated by Sokolow-Lyon criteria and Romhilt-Estes scoring systems. LVH by Sokolow-Lyon criteria was present in 29 patients (22.31%), and LVH was absent in 101 patients (77.69%), but echocardiograph showed LVH in all 130 patients. Observed LVH by Rhomhilt-Estes scorings system was present in 38 patients (29.23%) and absent in 92 patients (70.77%). But echocardiograph showed LVH in all 130 patients. The mean left

ventricular mass calculated was 179.07±36.62gms in males and 103.78±15.84gms in females in study group. Control group had 112.84±5.88gms in males and 73.36±9.41gms in females. There was significant increase in the left ventricular mass in study group when compared to control group. The sensitivity of Sokolow-Lyon was 22.30% and the sensitivity of Romhilt Estes point scoring system was 29.23%. Sensitivity of echo in detecting LVH in hypertensive patients was 100%.

CONCLUSION: It is concluded in our study that echocardiography is highly reliable and sensitive compared to electrocardiogram in the assessment of LVH in these patients.

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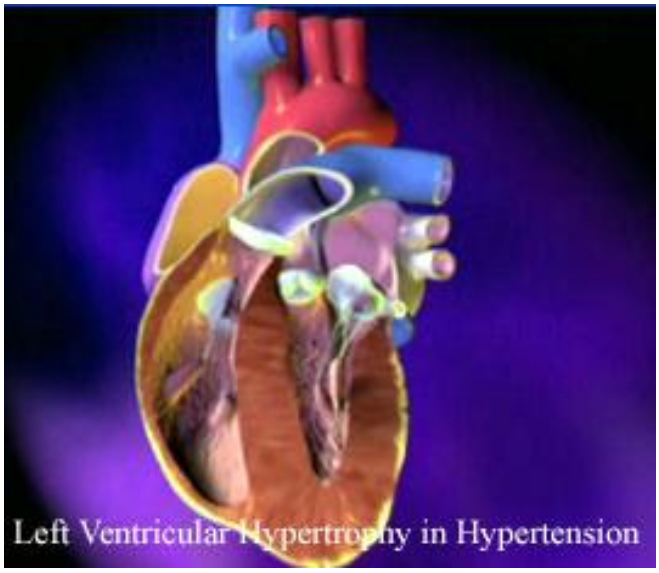


FIGURE .1

The rich heritage of basic cardiovascular medicine particularly physiology of cardiovascular medicine dates back to Sir William Harvey who had provided a framework for understanding cardiovascular diseases and from then onwards to the present development of number of clinical and investigative techniques such as electrocardiogram, echocardiogram and cardiac catheterization.

There is a temptation in cardiovascular medicine as in any other field of medicine to carryout expensive, uncomfortable and sometimes even hazardous procedures to establish diagnosis and to assess the prognosis. It is therefore necessary that the diagnostic tool should be non-invasive so that without torturing the patient one can get the co-operation from the patient for repetitive tests so as to confirm the reliability of the results. Intelligent and appropriate selection of investigative procedures from the ever increasing range of tests now available requires far more

decision making than necessary when the choice is limited to electrocardiogram, roentgenogram and echocardiogram which are the best, simple and basic repeatable investigative procedures.

A variety of cardiovascular disorders are characterized by left ventricular hypertrophy. Left ventricular hypertrophy is not only an important heamo-dynamic compensation for an over burdened heart but also an important independent parameter for predicting morbidity and mortality for various cardiovascular events. Electrocardiogram, echocardiographic and radiographic studies have been found to be indicators of left ventricular hypertrophy and LV mass. However, concept of LV hypertrophy based upon LV mass can only hold ground if the normal distribution of LV mass of the individuals in the population under surveillance is known and used as control.

Left ventricular hypertrophy has been repeatedly shown to be associated with marked increase in cardiovascular risk. The relative risk[1] associated with 100gms increase in left ventricular mass was 2.1 while a 0.1 cm increase in left ventricular posterior wall thickness was associated with a seven-fold increase in the risk of mortality.

Unfortunately conventional electrocardiograph criteria which are primarily aimed at recognizing the presence or absence of LV hypertrophy have a low sensitivity and limited utility.

The correlation of ECG patterns of LV hypertrophy with the anatomical finding offer the firm basis for an appraisal of accuracy of ECG criteria. The ECG

evidence of LVH is present only in 50% of patients with anatomic LVH. ECG may be entirely normal in around 15% patients with severe LVH[43, 25].

LV hypertrophy can be diagnosed by nuclear studies, echocardiography, and angiography. Echocardiograph measurement of LV mass has been shown to correlate closely with its measurements by angiography but has the advantage of being non-invasive and easily repeatable.

Primary echocardiograph parameters of left ventricular hypertrophy are septal hypertrophy and LV posterior wall thickness. LV hypertrophy indicates that mass of left ventricle has increased. Therefore, from equations which permit one to calculate LV mass, a direct estimation of LV mass by echocardiograph is more helpful in determination of cardiac hypertrophy.

The purpose of this study is to determine the sensitivity of ECG in diagnosis of LV hypertrophy by its correlation with the echocardiographic studies of LV mass.

AIMS AND OBJECTIVES

The aim of this study is estimation of left ventricular mass in hypertensive patients by echocardiography.

The results are to be correlated with the following two electrocardiograph criteria detecting left ventricular hypertrophy,

1. Sokolow-Lyon Voltage criterion.
2. Romhilt- Estes Point Scoring System.

REVIEW OF LITRATURE

HISTORICAL REVIEW

Human heart is a muscular pump. While most of the hollow organs of the body are made up of different types of muscular layers, heart is entirely made up of single muscular layer. In most of the other hollow organs of the body muscle layers are composed of smooth muscles and striated muscles but, heart is composed of specialized tissue called cardiac muscle. All muscle types function by contraction causing the muscle cells to shorten. Skeletal muscle cells which make up most of the mass of human body are voluntary and contract when brain sends signals to them making them to react. The smooth muscles surrounding the other hollow organs are involuntary which does not need signals to contract. Cardiac muscles are also involuntary. So functionally cardiac muscles and smooth muscles are similar. Though anatomically, cardiac muscles more closely resemble skeletal muscles. Both skeletal muscles and cardiac muscles are striated. Under medium to high power magnification through the microscope we can see small stripes running crosswise in both types.

Smooth muscles are not striated. Cardiac muscles could almost be said to be a hybrid between skeletal and smooth muscles. Cardiac muscles do have several unique features. Characteristically, in cardiac muscles are intercalated discs which are connections between two adjacent cardiac cells. Intercalated discs help multiple cardiac muscle cells contract rapidly as a unit. This is important for the heart to function properly. Cardiac muscles also can contract more powerfully when they are stretched slightly. When the ventricles are filled they are stretched beyond their normal resting capacity. The result is a more powerful contraction ensuring the

maximum amount of blood can be forced from the ventricles and into the arteries with each stroke. This is most noticeable during exercise when the heart beats rapidly.

There are four chambers in the heart two atria and two ventricles. The atria are responsible for receiving blood from the veins leading to the heart. Contraction of the atria causes blood to be pushed into the ventricles. However, the atria do not really have to work that hard. Most of the blood in the atria will flow into the ventricles even if the atria fail to contract. It is the ventricles that are the real workhorses; for, they must force the blood away from the heart with sufficient power to push the blood all the way to the last point of the arterial tree (this is where the property of contracting with more force when stretched comes into play). The muscles in the walls of the ventricles are much thicker than the atria. The walls of the heart are made up of several spirally wrapped muscle layers. This spiral arrangement results in the blood being pushed out from the ventricles during contraction. Between the atria and the ventricles are valves which are overlapping layers of tissue that allow blood to flow only in one direction. Valves are also present between the ventricles and the vessels leading from them.

Signals from brain can cause the heart to speed up or slow down; however, it does not control the regular beating of the heart. As noted earlier, heart is composed of involuntary muscles. The muscle fibers of the heart are self-excitatory. This means that they can initiate contraction themselves without receiving signals from the brain. This has been demonstrated many times in high school classes of the past by removing the heart of a frog or turtle and then stimulating it to contract. The heart continues to beat with no further outside stimulus, sometimes for hours if bathed in the proper solution. In addition to other properties cardiac muscle fibers also contract

for a longer period of time than do skeletal muscles. This longer period of contraction gives time for the blood to flow out of the heart chambers.

Physicians as well as people of many cultures had their own belief concerning the nature of the heart and circulatory system. The Greeks believed that the heart was the seat of the spirit. The Egyptians believed that the heart was the center of the emotions and the intellect. The Chinese believed the heart was the center for happiness. Even our modern society continues to put emotions under the control of the heart, speaking of having a broken heart when a loved one leaves, or stealing one's heart around Valentine's Day. These beliefs continued to be taught and taken as law until an English physician named William Harvey challenged them in the late 1620's.

Interests in the embryonic development of heart dates back to Aristotelian times and perhaps even earlier. In a developing chick embryo heart appears to be bright, translucent and nearly invisible. Hence early observers concluded quite correctly that the heart was the first organ to be formed during development. However, through many scientific luminaries including Galen, Fabricus, Malphigi and Harvey studied the embryonic heart, a little progress was made towards the understanding of how precisely it is transformed from a single convoluted tube into the four-chambered, four valved adult organ. A significant step was made when the greatest physiologist Galen Albrecht Von Haller in 1758 published a monogram devoted exclusively to the development of the heart.

The concept of cardiac enlargement in the causation of clinical phenomenon of heart failure dates back to 1812. Corvisart considered that the increase in heart size

and the back pressure on the lungs are the prime cause for the clinical phenomenon of heart failure.

In 1830 Hope introduced back pressure theory which has been elaborated and accepted till present day. In this back pressure theory venous congestion of the lungs is of paramount importance in the formation of edema resulting in reduced compliance of the lungs and reduced gaseous exchange.

Starling's work with heart lung preparation also supported the back pressure theory which states that within limits, an augmentation of initial volume of the ventricle which is a function of the initial length of the cardiac muscle results in the increase in the force of ventricular contraction. Beyond certain limit this compensation is lost and back pressure is buildup, resulting in pulmonary congestion. Now, there are many evidences to show that edema formation depends on other factors too.

Mackenzie and Lewis (1913)[34, 41] assumed that inadequate blood supply to the organs was an important abnormality. This theory of forward failure is supported by modern researches but as such, the old theories also couldn't be abandoned. According to these theories, lowered blood flow results in diminished glomerular filtration and greater absorption of salt and water and this retention of water and salt results in hemodilution, hypervolemia, increased extra vascular fluid volume and edema. The resultant increased hydrostatic forces, increased capillary permeability, increased accumulation of tissue metabolites and malnutrition play a part in the development of edema.

Linzbach (1960)[51] studied that in infants there is one capillary for every four or five myocardial fibers, while in the adult heart the ratio is 1:1. In the presence of hypertrophy the adult ratio remains the same. The idea that capillary inadequacy is a cause of hypoxia in hypertrophied hearts does not appear to be valid. In marked hypertrophy the ratio between the coronary artery and coronary ostial diameter decreases. This may restrict the myocardial perfusion in hypertrophied hearts.

Linzbach (1960)[51] also stated that there is an indirect evidence that the number of myocardial cells may increase during hypertrophy in an adult individual. This concept remains controversial as per the work done by Grove et al[50].

Spann et al (1969)[53] had shown that there is progressive decrease in contraction and development of tension in myocardial tissue in non-failing hypertrophied hearts. But later in course of time this will ultimately lead to congestive heart failure.

Badere (1968) pointed out that in the systolic overload or diastolic overload or during increased wall tension as in coronary heart diseases, the mechanical stimuli for hypertrophy require increased energy expenditure per unit mass of myocardium per beat.

Grove et al (1969)[50] put their concept in contrast to Linzbach. There is no equivocal evidence that adult myocardial cells can divide. Embryonic and early postnatal growth of the heart is accomplished by hyperplasia of myocardial cells. It appears that any subsequent myocardial enlargement can only be accomplished by hypertrophy of the individual myocardial cells.

Benzak (1969)[54] showed experimentally that myocardial cells return to their original size when the cardiac workload is returned to normal.

Meerson (1969)[52] had stated that heart has a good capacity to undergo hypertrophy in response to the abnormally increased work load which he termed as compensatory hyper functioning of heart.

Spann and Braunwald (1967)[71] studied in cats that digitalis glycosides have been shown to be capable of protecting against the development of ventricular hypertrophy in response to chronic systolic overload induced by aortic stenosis. Possibly this effect occurs because the drug prevents ventricular dilatation with the increase in intraventricular pressure.

Legato (1970)[72] had provided electron microscopic evidence suggesting that the 'Z' line substance is essential for new sarcomeres production during cardiac hypertrophy.

Rabinowitz and Zak (1972)[72] showed that like other living cells, cardiac fibers also maintain a state of dynamic equilibrium between synthesis and breakdown of cellular elements especially proteins.

Laks et al (1974)[73] showed that during cardiac hypertrophy increase in the cell length is the result of adding more sarcomeres to the cell rather than the result of stretching of the sarcomeres.

Devereveux R. Reichek. N (1986)[48] calculated LV mass by echocardiography using Penn convention method and correlated this work with angiographic studies.

Lewis JF (1990)[47] had explained about the diversity in patterns of left ventricular hypertrophy in patients with systemic hypertension with marked left ventricular wall thickening.

Dahlof B (1992)[46] had shown in a meta analysis of various studies that there is a reversal of left ventricular hypertrophy in the hypertensive patients after regular treatment with antihypertensive drugs which can be studied by ECG and echocardiography.

Levy et al (1990)[43] have studied and stated the prognostic implications of measurements of left ventricular mass determined by echocardiography in essential hypertension and concluded that the estimation of left ventricular mass by echocardiography is superior to any other traditional methods.

Levy et al (1998)[44] stated about the echocardiographic measurements detecting left ventricular hypertrophy in relation to its prevalence and the various other risk factors causing an increase in the mass of left ventricle in hypertensive patients particularly.

Verdechia et al (1998)[45] have measured and estimated serial changes in left ventricular mass in essential hypertension and correlated it with the studies of the electrocardiographic findings of left ventricular hypertrophy. They also proved that increase in left ventricular mass in hypertensive patients is associated with increased mortality.

Missault et al have studied (2002)[42] that there is relationship between left ventricular mass and hypertension once the hypertension is diagnosed and treated. They assessed left ventricular hypertrophy by electrocardiography which was

correlated with left ventricular mass assessed by echocardiography. They found that the echocardiograph parameters of left ventricular mass in hypertensive subjects correlated better when compared with electrocardiograph parameters of left ventricular hypertrophy in patients with hypertension.

ELECTROCARDIOGRAPHY

Waller (1887) was the first person who recorded ECG. While Einthoven (1906) found modern concepts of ECG and he was regarded as the father of ECG. He recorded ECG by using string galvanometer. These were in use till 1930, when it was replaced by amplifier driven oscilloscope with recording facilities. In 1912 Einthoven was the first person to describe different forms of human ECGs and their clinical significance.

The earliest reports available in literature about left ventricular hypertrophy and its ECG diagnosis dates back to 1914 when Lewis T[47] described observations upon ventricular hypertrophy with specific reference to preponderance of one or other chamber. Various investigations have proposed different criteria for LV hypertrophy.

In 1943 Wilson FW[2], Johnson F.D. Rosenbaun U.F[4] published papers on precordial electrocardiogram.

The criteria described for LVH were:

1. Absence of 'r' wave or abnormal small 'r' wave in V_1 with large 'S' wave > 24 mm in V_1.
2. Shift of transitional zone in precordial leads to left.
3. Delay in peak of 'r' wave in V5-V6 (0.05 seconds).
4. Tall 'r' in left precordial lead.

   R > 33 mm in V5

   R > 26 mm in V6
5. T-wave ↓ in V5 or V6
6. QRS interval 0.1 or 0.11 seconds.

In the same year Gubner and Ungerleider[3] proposed ECG criteria of LVH.

The proposed criteria are:

a) Left axis deviation with R1 + S3 > 25 mm.

b) Left axis deviation with depression of ST segment 0.5 mm or more.

c) Left axis deviation with 'T' wave in lead I 1 mm.

In 1946 Katz L.N[4] described his own criteria for LVH under the title 'the electrocardiograph. His criteria were:

1. a) LAD

   b) Small equiphasic QRS or deep 'S' in lead II with normal ST – T wave.

2. a) LAD

   b) Depressed upward convex ST in lead I.

3. a) LAD + (1) + (2)

   b) Concordant QRS wave in standard limb lead.

In 1949 Goldberger[8] described his own criteria for LVH in unipolar leads they are:

1. High voltage QRS complex with R wave

   13 mm in a VL or R wave 20 mm in a VF.

2. Left ventricular strain.

   ST 0.5 mm with T ↓ in VL or a VF (depending on heart position).

   ST 0.5 mm with T ↓ in V5-V6.

In 1949 Sokolow and Lyon[5] TP published their criteria "the ventricular complexes in LV hypertrophy as obtained by unipolar precordial and limb lead system."

The LVH criteria is

1. S wave in V1 + R wave in V5 or V6 =>35 mm or
2. R wave in V5 orV6 =>26 mm.

Scott R.C. Seiwarto V.J. (1955)[6] and his associates correlated accuracy of various ECG criteria with autopsy findings in 100 cases and published their papers.

In studying LVH the accuracy of current ECG criteria when compared to autopsy findings in 100 cases, it was found that sensitivity of various ECG criteria suggested by Scott et al[6] were,

1. Gubner and Ungerleider- 36.2%
2. Katz- 59.00%
3. Schach, Rosenbaun, Katz- 21.00%
4. Goldberger- 68.00%
5. Goulder Kissare- 27.00%
6. Wilson et al- 81.00%
7. Sokolow and Lyon- 85.00%

In 1960 Allenstain, B.J. and Morti[7] evaluated various ECG criteria and correlated with autopsy findings in his article "Evaluation of ECG diagnosis of LVH based on autopsy correlation".

According to Allenstain[7] criteria for the diagnosis of LVH proposed by Gubner and Ungerleider[3], Kartz[4], Schaeh Rosenbaum[4], Goulder and Kissare[8] and

Moth Myers Klien showed the percentage of positive ECGs under 50%. The criteria of Wilson Sokolow and Lyon[5] were relatively high positive but they also had a higher incidence of false positive results.

After well documented reports of comparisons between various ECG criteria for diagnosis of LVH like Allenstain[7] study, Scott[6] et al, Chow and associates[9] and Griep A.H.[10] an effort was made to combine various criteria into a useful system in 1964 by Carter and Estes[11] who re-examined some of the empirical criteria that were proposed for LVH diagnosis in order to weigh these criteria accordingly.

The Romhilt - Estes[12] point score system was described later in 1968.

The Romhilt-Estes Point scoring system is

1. R or S wave in any limb leads > 20mm

   Or S wave inV1 or V2 > 20 mm

   Or R wave in V5 or V6 > 30 mm…… 3 points

2. Left ventricular strain

   ST segment and T wave in opposite direction to QRS complex

   With digitalis……. 3 points

   With out digitalis……... 1 point

3. Left atrial enlargement – terminal negativity of P wave in V1 > 1mm in depth

   or > 0.04 seconds in duration…… 3 points

4. Left axis deviation > -30 degree…… 2 points

5. QRS duration > 0.09 seconds……..…….. 1 point

6. Ventricular activation time > 0.05 seconds…….. 1 point

   TOTAL = 13 POINTS

   PROBABLE LVH = 4 POINTS

LVH = 5 POINTS

Joseph[13] et al, Kenneth[14] et al and Francisco[15] et al have studies the relationship of LV mass and LV hypertrophy by method of 2-D echo and they have compared it with angiography and postmortem studies.

Recently in 1984 M.L Murphy, P.N. Thenebader[41] et al have reported reliability of ECG criteria for ventricular hypertrophy using

(1) Modification of Romhilt Estes point system

(2) S.L criteria of S in V1 + R in V5 or V6 > 35 mm.

They showed improved specificity in diagnosing ventricular hypertrophy with only slight decrease in sensitivity.

ECHOCARDIOGRAPHY

Echo sounding is a technique used in certain birds and animals for distance perception which was first applied by human beings in 1920 for depth recording in oceanographic studies and submarine detection. The principles of diagnostic ultrasound have their roots in navy sonar which use sound impulses to detect objects and measure distance in water.

By definition ultrasound is the sound having a frequency greater than 20,000 cycles /second. The use of ultrasound as a diagnostic tool in medicine is relatively recent but the engineering aspect of ultrasound dates as far back as 1800 when attempts to produce high frequency sounds first began by Galton in 1883 who developed an ultrasound whistle capable of producing vibrations as high as 25,000 cycles /second.

The use of ultrasound became practical with the development of piezoelectric transducer. Firestone is credited with being the first person who started the use of pulsed reflected ultrasound for non-destructive testing.

Keidel was the first person who used ultrasound in examination of heart. The first effect of using pulse reflected ultrasound was initiated by Dr. Hertz.

Edler was the person who went on making vast number of ultrasonic studies of heart. In 1968 Feigenbaum H. Popp R.L[16] and associates measured LV wall thickness by ultrasonic method and published their observation.

While in 1970 Sjogren and Fick published their work on “ultrasonic measurement of LV wall thickness”.

In 1971 Troy, Pombo and Rackley[17] correlated LV internal dimension on ultrasound with the LV cavity volume measured by Biplane angiography. In the following year Troy, Pombo and Rackley[17] published their data in an article called "Measurement of LV wall thickness and mass by echocardiography". Murry J.A Jhonston W, Reid J.W et al[18] determined LV volume, dimension and performance of Echo and correlated them with angiographic studies with good results.

In 1973 Popp R.L[19] described about sources of error in calculation of LV volume by echo.

Devereux.R.[20] Reichek (1976) calculated LV mass by echo using Penn convention method and correlated this work with angiographic studies.

The instrumentation involved in echocardiography has become increasingly complex with the evolution from M-mode to two-dimensional echo-cardiographs to the introduction of Doppler techniques with color coding.

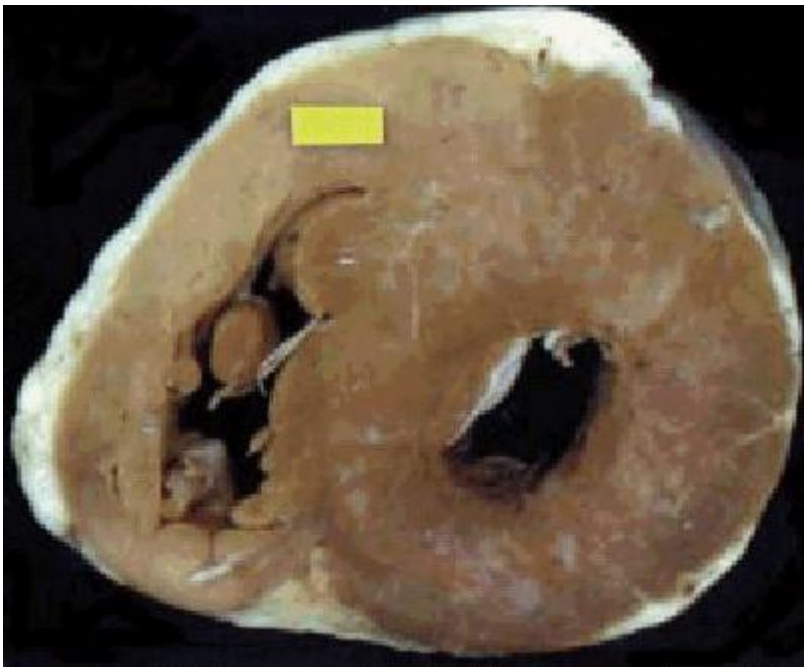


FIGURE 2. LEFT VENTRICULAR HPERTROPHY IN AN AUTOPSY SPECIMEN

Left ventricular hypertrophy, which is defined as an abnormal increase in the mass of the left ventricle is recognized as an important independent risk factor for predicting cardiovascular morbidity and mortality. These include myocardial infarction, congestive heart failure and sudden cardiac death.

Pathologically cardiac hypertrophy is defined as an increase in the volume of the cardiac myocytes[21]. Hypertrophy of the ventricles causes remodeling of the architecture of the heart to normalize the wall stress. The particular pattern of hypertrophy is dependent on the type of load that is imposed on the heart. Increased after load leads to an increase in systolic stress besides addition of number of sarcomeres[21]. This results in increase in the wall thickness but with normal or decreased chamber size. Thus there is increase in relative wall thickness. This pattern has been termed as “Concentric Hypertrophy” and is found in-patients with pressure overload states such as systemic hypertension and aortic stenosis. Increase in the

preload leads to an increase in diastolic stress and the addition of sarcomeres in series resulting in increase in chamber size. This type of ventricular hypertrophy with an increase in the ratio of chamber size to wall thickness has been termed as "Eccentric hypertrophy"[21]. This is common in the volume overload of left ventricle in cases of aortic insufficiency and mitral regurgitation. Eccentric hypertrophy is usually present in the end stages of any dilated cardiomyopathy. In this case the ventricular dilatation is not balanced by the increase in wall thickness. Echocardiography allows the clear distinction between concentric and eccentric left ventricular hypertrophy.

Strenuous physical exercise in athletes also causes increased left ventricular mass but with normal function. Left ventricular hypertrophy that develops in response to pathologic stress on the heart is associated with adverse consequences[21].

The most readily available diagnostic modality for left ventricular hypertrophy has been 12-lead electrocardiogram. Many physicians have relied on electrocardiogram to detect the presence of left ventricular hypertrophy in hypertension[30]. Various combinations of ECG changes have been analyzed and all of them have demonstrated limited sensitivity for the detection of left ventricular hypertrophy. Casale's[22] modified ECG criteria showed the highest sensitivity for left ventricular hypertrophy which was 49% and the specificity of 93%. The insensitivity of electrocardiogram for detecting left ventricular hypertrophy limits its benefits for stratifying risk in hypertensive patients and hence this has suggested critical evaluation of other diagnostic tools[23]. The presence of left ventricular hypertrophy is difficult to detect by ECG but it can be accurately assessed by echo-cardiography[24]. A well represented study by Daniel Savage et al[25] showed that in patients with hypertension echocardiography identified left ventricular hypertrophy in over 50%

while the electrocardiography identified less than 10%. Bart. L. Troy showed that echocardiography offers a reliable and reproducible method for measuring left ventricular thickness and mass[26]. Echocardiography is currently considered as the method of choice for diagnosis of the left ventricular hypertrophy in hypertensive patients[27]. Evaluation of left ventricular hypertrophy by M-mode echocardiography will take into account both wall thickness and cavity size.

Pearson et al[21] in their review on LVH have shown that left ventricular mass determined at autopsy correlated well with that determined by M-mode echocardiography. The same authors have found that M-mode echocardiograph LV Mass greater than 256 Gms had a sensitivity of 88% and a specificity of 84%. M-mode was better than 2D Echocardiography in the above study.

In the Framingham[28] study ventricular hypertrophy was independently associated with age, blood pressures, valvular heart disease and post myocardial infarction. The prevalence of LV hypertrophy ranged from 6.6% in the young women to 33% in older women and fell in between these two values in men.

In the Framingham[28] study men over the age of 45 had greater than 40% mortality rate after 6 years of the appearance of criteria for left ventricular hypertrophy on electrocardiograms. ECG evidences of left ventricular hypertrophy had been more specifically linked to sudden cardiac death[21]. Unfortunately, electrocardiographic left ventricular hypertrophy data are of marginal use in the stratification of risk in the general population and in patients with mild hypertension because of their low sensitivity. However, the high sensitivity of echocardiography to detect left ventricular hypertrophy in these populations makes it an ideal tool for risk stratification.

Casale et al[22] published the first detailed study of the cardiovascular risks of left ventricular hypertrophy. The men with left ventricular hypertrophy in their study did not differ from those without left ventricular hypertrophy in terms of age, blood pressure, smoking or cholesterol, but had a significantly higher rate of cardiovascular morbidity (24% Vs P<0.01).

The patients with echocardiographic evidences of left ventricular hypertrophy had a significantly higher incidence of new cardiac events and of new atherothrombotic brain infarctions. Patients with left ventricular hypertrophy appear to have more frequent and more complex ventricular ectopies than in those without left ventricular hypertrophy. Levy et al [28,43,44] showed that in 6218 patients of Framingham heart study, echocardiographic evidences of left ventricular hypertrophy was over 5 times as prevalent as the E.C.G evidences of left ventricular hypertrophy and was associated with increased risk of complex ventricular arrhythmias in both men and women. The other factor that influenced cardiac mortality rates in patients with left ventricular hypertrophy was reduced coronary flow reserve. Left ventricular hypertrophy is associated with an eight-fold increase in cardiovascular mortality and a six-fold increase in coronary mortality[28].

The electrocardiogram was found to be very specific but insensitive in detection of left ventricular hypertrophy as compared with echocardiogram[21]. There is a paucity of Indian Literature on this subject except for few recent articles[29]. We propose to determine the abnormal ventricular mass in hypertensive patients with echocardiography and compare it with the simultaneous usage of two electrocardiograph criteria for detecting left ventricular hypertrophy. This helps in identifying the high-risk group among them for earlier and effective management.

L.H. Missault et al (2002)[42] studied and evaluated prospectively whether there is a relationship between left ventricular mass and blood pressure once hypertension is treated . Correlation was made between blood pressure values and parameters of left ventricular mass. Electrocardiographic voltage criteria and even more so echocardiographic parameters correlated significantly albeit weakly with blood pressure in treated hypertension. Correlations are consistently higher when systolic or night-time blood pressure is considered. Overall, the best correlations are found between 24-h ambulatory systolic or night-time blood pressure and the Sokolow-Lyon voltage as well as the echocardiograph, age and body mass index adjusted left ventricular mass. In conclusion, once hypertension is treated the relationship between blood pressure and left ventricular mass is low. Parameters of hypertrophy are more closely related to systolic blood pressure than to diastolic blood pressure. In accordance with the finding that dippers have a better prognosis than non-dippers, night-time blood pressure consistently correlates better with left ventricular mass than daytime blood pressure.

Pentti et al[55] studied associations between echocardiographic parameters and electrocardiographic parameters with left ventricular mass values with clinical and sub clinical indices of coronary heart diseases.

Both Echocardiograph and electrocardiograph models demonstrated similar and about equally strong associations with overt and sub clinical disease status and risk factors associated with left ventricular hypertrophy. These observations demonstrated the potential utility of electrocardiograph models for left ventricular mass estimation.

Devereux et al (2002)[48] studied that in hypertensive patients with ECG evidence of left ventricular hypertrophy of mild degree was seen in one eighth of the patients and was associated with male gender and albuminuria. No significant differences were found between patients with global or segmental wall motion abnormalities.

Donna et al (2003)[57] found that epidemiological and clinical studies frequently use echocardiography to measure LV wall thickness and chamber dimension for estimating quantitative measures of LV mass. While echocardiographic M-mode LV images have traditionally been measured using hand-held calipers and strip-chart paper tracings. Digitized M-mode LV images measurements were made directly on the computer screen using electronic calipers which now have become standard practice. They compared if systematic differences in LV mass occur between the two methods by comparing LV mass measured from simultaneously M-mode strip chart recordings and digitized recordings.

Hond et al (2003)[58] studied the relationship between LV mass and systolic blood pressure. Partial correlation coefficients were calculated between electrocardiograph and echocardiograph LV mass at baseline and different types of blood pressure like office blood pressure (OBP), 24-h ambulatory blood pressure (ABP), daytime ABP and night-time ABP.

Electrocardiograph LV mass correlated equally well with all types of systolic blood pressure. The findings were similar for echocardiograph LV mass index. However for echocardiographic mean wall thickness there was a stronger association with ABP than with OBP.

In the combined dataset of the APTH and THOP trials, electrocardiograph and echocardiograph LV mass at baseline correlated significantly with all types of systolic blood pressures. For mean wall thickness the relationship was stronger for ABP than OBP.

Adewole et al (2006)[59] found that left ventricular hypertrophy (LVH) is a well known independent risk factor for cardiovascular events. It has been shown that combination of left ventricular mass (LVM) and relative wall thickness (RWT) can be used to identify different forms of left ventricular (LV) geometry. Prospective studies have shown that LV geometric patterns have prognostic implications with the worst prognosis associated with concentric hypertrophy. The methods for the normalization or indexation of LVM have also recently been shown to confer some prognostic value especially in obese population.

Francesco Perticone, Raffaele Maio, Roberto Ceravolo et al (1999)[15, 60] studied the background of hypertensive patients which are characterized by development of both left ventricular hypertrophy (LVH) and endothelial dysfunction. Their data provided the first evidence that echocardiograph LVM in hypertensive patients is inversely related to the endothelium-dependent vasodilating agent Acetylcholine but it is likely that both endothelium and LVM are damaged by hypertension.

Ferrari et al (2003)[61] studied that primary impairment of the renal sodium excretion has been documented both in hypertensive patients and genetic animal models carrying mutations of the cytoskeleton protein adducin and/or increased plasma levels of endogenous ouabain (EO). Ouabain (OU) itself induces hypertension in rats and both OU and mutated adducin activate the renal Na/K-ATPase function

both in vivo and in cultured renal cells. A new antihypertensive agent PST 2238 able to selectively interact with these alterations has been developed. PST lowers blood pressure (BP) by normalizing the expression and activity of the renal Na-K pump selectively in those rat models carrying the adducing mutation and/or increased EO level at oral doses of 0.1-10 μg/kg of PST to antagonize the OU- dependent activation of growth related genes in the membrane sub domains. In conclusion PST is a new antihypertensive agent that may prevent cardiovascular complications associated with hypertension through the selective modulation of the Na-K pump function.

Julius (1990)[62] studied the evolution of hypertension in healthy population. Borderline hypertensive subjects had significant abnormalities in other coronary risk factors (Cholesterol levels were 0.39 mmol/L higher, triglyceride levels were 0.45 mmol/L higher, high-density lipoprotein levels were 0.08 mmol/L lower, insulin levels were 38 pmol/L higher, and 16.5% more of them were overweight). Borderline hypertension is neither transient nor innocuous. Its association with other predictors of atherosclerosis calls for clinical attention.

Paolo Verdecchia et al (2001)[63] investigated the prognostic value of left ventricular (LV) mass using echocardiography in uncomplicated subjects with essential hypertension. Only a few single center studies supported the prognostic value of LV mass in uncomplicated hypertension. The aim was to explore the prognostic value of LV mass in hypertension. They found a strong, continuous and independent relationship of LV mass to subsequent cardiovascular morbidity. This is the first study to extend such demonstration to a large nationwide multicenter sample of uncomplicated subjects with essential hypertension.

.Richard B. Devereux, MD et al(2004)[64] studied about increased baseline left ventricular (LV) mass predicts cardiovascular (CV) complications of hypertension, but the relation between LV mass and outcome during treatment for hypertension is uncertain.

The conclusion was that in patients with essential hypertension and baseline electrocardiographic LV hypertrophy, LV mass was found to reduce during antihypertensive treatment and was associated with lower rates of clinical end points additional to effects of blood pressure lowering and modifying morbidity and mortality.

L Lind (2008)[65] studied that left ventricular hypertrophy (LVH) is a powerful cardiovascular risk factor and has previously been related to endothelium-dependent vasodilatation (EDV) in hypertensive patients. EDV was mainly reduced in those with left ventricular concentric hypertrophy. In conclusion in a population based sample of elderly subjects EDV was inversely correlated with LVM independent of blood pressure suggesting that EDV in resistance arteries is of more importance for LVH than endothelial vasodilator function in conduit arteries in the elderly.

Manunta et al (2004)[66] have studied that many patients with essential hypertension (EH) exhibit increased left ventricular mass. Elevated circulating levels of an endogenous ouabain (EO) like factor have been found in some patients with hypertension. Moreover ouabain has a hypertrophic influence on isolated cardiac myocytes. They concluded that 50% of patients with uncomplicated hypertension have elevated high circulating ouabain like factor levels. Higher the diastolic blood pressure the greater is the left ventricular mass and stroke volume and reduced heart rate. They proposed that the ouabain like factor affects cardiovascular function and

structure and should be considered as a factor that contributes to the risk of morbid events.

Jin Zhang et al (2005)[67] found a key question in hypertension that is: How is long-term blood pressure controlled? A clue is that chronic salt retention elevates an endogenous ouabain-like compound and induces salt-dependent hypertension mediated by Na^+/Ca^{2+} exchange.

While LVH itself is not a disease, it is usually a marker for disease involving the heart. Disease processes that can cause LVH include any disease that increase the after load on the heart so that the heart has to contract against resistance and some primary diseases of the muscle of the heart. The principle method to diagnose LVH is echocardiography during which the thickness of the muscle of the heart can be measured. The electrocardiogram often shows signs of increased voltage from the heart in individuals with LVH, so this is often used as a screening test to determine who should undergo further testing with an echocardiogram.

2D echocardiography can produce images of the left ventricle. The thickness of the left ventricle as visualized on echocardiography correlates with its actual mass. Normal thickness of the left ventricular myocardium is from 0.6 to 1.1 cm, as measured at the very end of diastole. If the myocardium is more than 1.1 cm thick, the diagnosis of LVH can be made.

There are multiple criteria used to diagnose LVH via electrocardiography. None of them are perfect. However, by using multiple different criteria the sensitivity and specificity are increased.

The enlargement is not permanent in all cases of LVH and in some cases the growth can regress with the reduction of blood pressure.

High BP increases the risk of total mortality; mortality due to heart disease, stroke, chronic kidney disease, and heart failure; and morbidity associated with nonfatal cardiovascular disease events. On the basis of estimates of population, high BP may account for 27% of total cardiovascular disease events in women and 37% in men, 14% of myocardial infarctions in men and 30% in women, 35% of ischemic strokes, 39% of chronic heart failure events in men and 59% in women and 56% of chronic kidney diseases. In the Global Burden of Disease Project, a systolic BP threshold of 115 mm Hg was used to distinguish between optimal and non optimal BP levels. Globally, 62% of strokes, 49% of coronary heart disease and 14% of other cardiovascular diseases was attributable to non optimal BP. Approximately 12.8% of all deaths (7.1 million) and 4.4% of all disability life years lost (64.3 million) in the year 2000 were due to cardiovascular diseases attributable to non optimal BP levels. Clearly high BP is a major cause of mortality and morbidity worldwide.

Randomized controlled trials have provided convincing evidence that BP lowering treatment reduces the risk of total mortality, stroke, coronary heart disease, heart failure, and chronic kidney disease. Consequently clinical practice guidelines have been promulgated to promote detection, treatment and control of high BP.

In essential hypertension the primary defect may be an acquired renal injury rather than a genetic defect. Nevertheless, none of the studies have addressed the question of precisely how salt retention leads to chronic hypertension. Mean arterial BP depends primarily on cardiac output (CO) and total peripheral systemic vascular resistance (TPR): at constant CO, mean BP $\approx$ CO x TPR. Acute plasma volume

expansion elevates BP while CO declines. This condition of high TPR and near-normal CO is commonly observed in humans with essential hypertension.

The shift from high CO to high TPR called as 'whole-body auto regulation' has been attributed to regulation of blood flow to meet metabolic demand. This view is controversial; however, the mechanisms are unresolved. According to one hypothesis salt retention promotes secretion of an endogenous cardio tonic steroid that inhibits Na^+ pumps including those in vascular smooth muscle. By raising the systolic Na^+ concentration this agent would be expected to promote Na^+/Ca^{2+} exchanger (NCX) mediated Ca^{2+} entry into the myocytes. This elevates the cytosolic Ca^{2+} concentration and thus increases TPR by enhancing myogenic tone; the intraluminal pressure induced intrinsic arterial constriction that is prominent in small resistance arteries. Indeed, recent evidence reveals that NCX type-1 in arterial myocytes plays a central role in ouabain induced hypertension and salt dependent hypertension.

The discovery of an endogenous ouabain like compound that is synthesized and secreted by the adrenal cortex supports the hypothesis which states that plasma endogenous ouabain levels are elevated in ~ 45% of patients with essential hypertension and in several animal models of salt dependent hypertension. The endogenous ouabain like compound levels correlate with high BP. Moreover prolonged administration of ouabain like factor which is the Na^+ pump inhibitor from plants induces sustained dose-dependent increase in TPR and BP in normal rats and mice.

Na^+ pumps are expressed as $\alpha\beta$ dimers. Four isoform of the catalytic (α) subunit, the only known ouabain receptor have been identified. But mouse arteries only express Na^+ pumps with the $\alpha1$ and $\alpha2$ isoform. Rodent $\alpha1$ has unusually low ouabain affinity. Mammals have Na^+ pumps with $\alpha2$ subunits and have high ouabain

affinity. Here it is shown that exogenous ouabain at low concentrations approaching circulating endogenous ouabain like factor augments vasoconstriction of pressurized small arteries and plays a role in development of hypertension.

Okin et al (2007)[68] studied about the hypothesis that regression or continued absence of electrocardiographic LVH during antihypertensive therapy is associated with a decreased incidence of atrial fibrillation independent of blood pressure and treatment modality.

New onset atrial fibrillation in relation to electrocardiographic LVH determined at baseline and subsequently was found in many cases. Electrocardiograph LVH was measured using sex-adjusted Cornell product criteria ([R(aVL) + S(V3) [+6mm in women]) x QRS duration).

Lower Cornell's electrocardiographic criteria for LVH during antihypertensive therapy were associated with a lower likelihood of new onset of atrial fibrillation independent of blood pressure lowering and treatment modality in essential hypertension. These findings suggest that antihypertensive therapy targeted at regression or prevention of electrocardiographic LVH may reduce the incidence of new onset AF.

Devereux et al (2004)[64] studied the reduction of electrocardiographic left ventricular hypertrophy (LVH) has been associated with decreased cardiovascular death, stroke, myocardial infarction and atrial fibrillation. However, whether reduction of electrocardiographic LVH is associated with decreased heart failure is unclear.

But it was found that reduction in Cornell's electrocardiographic LVH during antihypertensive therapy is associated with fewer hospitalizations with heart failure independent of blood pressure lowering measures, treatment methods and other risk factors for heart failure.

Okin (2004)[68] again studied that Electrocardiographic left ventricular hypertrophy (LVH) is a strong predictor of cardiovascular morbidity and mortality. However, the predictive value of changes in the magnitude of electrocardiographic LVH criteria during antihypertensive therapy remains unclear.

Less severe electrocardiographic LVH by Cornell product and Sokolow-Lyon voltage criteria during antihypertensive therapy is associated with lower likelihood of cardiovascular morbidity and mortality independent of blood pressure lowering measures and treatment modalities in persons with essential hypertension. Antihypertensive therapy targeted at regression or prevention of electrocardiographic LVH may definitely improve prognosis.

Okin (2006)[69] also studied the ECG strain pattern of ST depression and T-wave inversion is strongly associated with left ventricular hypertrophy (LVH) independent of coronary heart disease and with an increased risk of cardiovascular morbidity and mortality in hypertensive patients. However, whether ECG strain is an independent predictor of new-onset congestive heart failure (CHF) in the setting of aggressive antihypertensive therapy is unclear.

ECG strain identifies hypertensive patients at increased risk of developing CHF and dying as a result of CHF even in the setting of aggressive blood pressure lowering.

Vittorio Palmieri. Peter M. Okin; Jonathan N. Bella Eva Gerdts (2003)[70] assessed the prevalence and the correlates of echocardiographic parameters of global and segmental left ventricular wall motion abnormalities in hypertensive patients with left ventricular hypertrophy enrolled in the Losartan intervention for end point reduction in hypertension (LIFE) echo sub study. In this study, hypertensive patients with ECG evidence of left ventricular hypertrophy had wall motion abnormalities of milder degrees. These wall motion abnormalities occurred only in one eighth of the total patients and were associated with male gender, left ventricular hypertrophy and albuminuria. No significant differences were found between patients with global or segmental wall motion abnormalities.

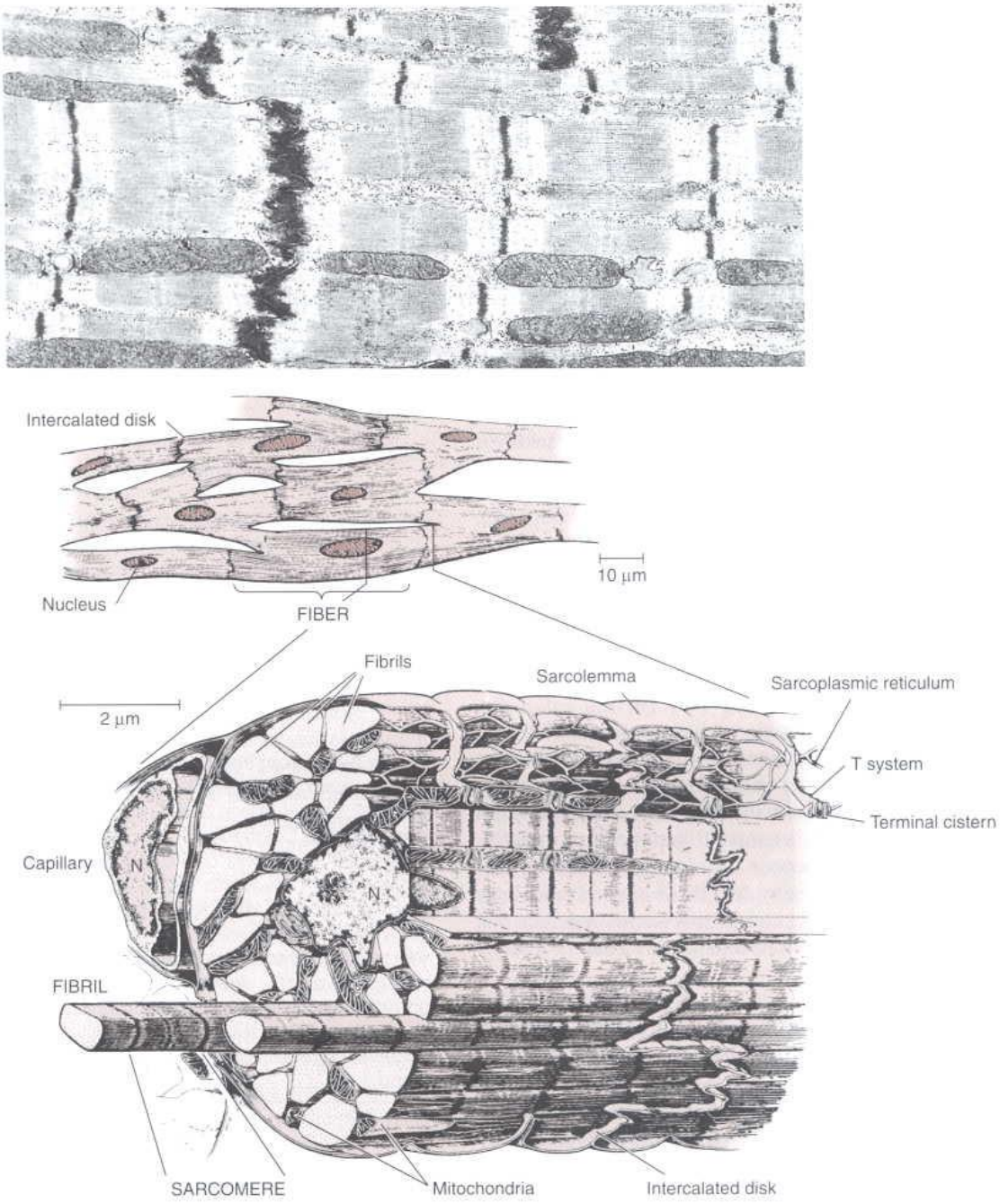


FIGURE 3. STRUCTURE OF CARDIAC MUSCLE FIBERS

A. MYOCARDIAL EXCITATION- CONTRACTION COUPLING:

The myocardial tissue consists of multiple fibers which are again made up of multiple myofibrils. Each myofibril in turn consists of sub-units called the sarcomeres which are separated from each other by horizontal lines called the Z lines and project inside the sarcomeres where it integrates with the thicker filament portion called the myosin.

Myosin has the property to split adenosine tri-phosphate (ATP) and hence acts as ATPase. Interaction between actin and myosin produces actomyosin which has more potent ATPase like activity as compared to myosin alone.

In addition to the contractile proteins, there are two other proteins called the troponin and tropomyosin. They can inhibit the contractile response of the actomyosin. However if calcium (Ca++) ions bind to these inhibitor proteins, the latter are unable to inhibit actomyosin which can contract uninhibitedly. Ca++ ions therefore act as an inotropic factor.

In sarcolemma there are two distinct channel systems; the 'T' system and a membrane bound channel system called the sarcoplasmic reticulum. This acts as a storage site for Ca++ ions.

The cell membrane of sarcolemma maintains a high intracellular concentration of potassium (K+) and low sodium (Na+) and Calcium (Ca++) concentration in diastole by an active process which is enzyme dependent and utilizes energy. With depolarization Na+ enters rapidly into cells and this takes place during the first phase of the action potential. Ca++ enters slowly during the second phase and through separate channels. The entry can be blocked by magnesium ions (Mg+), acidosis or

drugs like verapamil. Methyl xanthine and Catecholamines have exactly opposite effect and therefore increases the Ca++ content of the myocardial cell and produce an inotropic effect. Intracellular Ca++ concentration is also related to intracellular Na+ concentration. When intracellular Na+ is extruded out, three Na+ ions are exchanged for one extra cellular Ca++ resulting in an increased Ca++ concentration inside the cell.

B.THE ROLE OF MUSCLE LENGTH:

The force of contraction depends on initial muscle length. The sarcomere length associated with the most forceful contraction is approximately 2.2 micrometers. At this length the two sets of myo-filaments of the sarcomere are most ideally situated to provide the greater area for their interaction. When sarcomere length increased to 3.65 micrometers the thin filaments are entirely withdrawn from the A band and no tension can be developed. Similarly when the sarcomere are shorter than 2 micro meters, the thin filaments bypass one another producing a double overlap of the thin filaments causing a reduction of sensitivity of the contractile sites to Ca++ and the capacity for force development also declines.

The relation between the initial length of muscle fibers (pre load) and the developed force is of prime importance for functioning of the heart muscle. This forms the basis of the Frank Starlings relation (Starlings Law of Heart) which states that within limits the augmentation of initial volume of the ventricle, which is a function of the initial length of the cardiac muscle, results in an increase in the force of ventricular contraction.

C) THE FORCE VELOCITY CURVE:

The mechanical activity of all muscle may be expressed externally in two ways: shortening and the development of tension. The velocity of shortening is inversely related to the magnitude of tension development which is a fundamental property of every muscle. In simple ways, the greater the load the muscle is called upon to pump, the lower is the velocity of shortening and vice versa. The contractile activity of the myocardium may be readily altered under physiologic conditions by changes in resting muscle fiber length and by changes in the inotropic states (contractility) both of which shift the myocardial force velocity curve.

D) VENTRICULAR WALL TENSION AND THE LAPLACE RELATIONSHIP:

After load is the resistance against which the ventricles contract i.e. aortic impedance, the peripheral vascular resistance, the arterial wall (stiffness) resistance, the mass of the column of blood in the aorta and the viscosity of blood.

Ventricular myocardial tension is related to intraventricular pressure per square centimeter of surface upon which the pressure is exerted. The wall tension begins to decrease after the beginning of ejection and the time of peak systolic pressure and may actually be less than that at the onset of systole. The increased tension in dilated hearts is believed to be an adequate stimulus for myocardial hypertrophy. This accounts for hypertrophy in those abnormal states in which the heart is subjected to a volume overload at normal pressure. An additional influence of changes in after load is manifested by an increase in ventricular performance several beats after aortic pressure is raised. Some studies have indicated that this phenomenon may be due to recovery from transient subendocardial ischemia caused by sudden change in arterial pressure.

E) CONTRACTILITY AND THE IONOTROPIC STATE:

The third major mechanism by which myocardial function is altered is by a change in the inotropic state (contractility) of the muscle independent of a change in preload (fiber length) or after load. This indicates that when contractility is augmented, the myocardium shortens faster at any given length for any given load and also shortens further or generates more force.

F) HEART RATE (BOWDITCH EFFECT):

Fourth major determinant of cardiac function is the heart rate or the frequency of cardiac contraction. This is probably the major mechanism by which most of the individuals increase their cardiac output during periods of modest increased demand or exercise. An increase in heart rate may also increase myocardial contractility and relaxation and improve diastolic performance. The systemic force-interval relationship is known as TREPPE, STAIRCASE PHENOMENON or BOWDITCH effect. There is also a "recuperative effect of a long pause" upon the strength of contraction which is known as the WOODWORTH PHENOMENON or as negative or reverse staircase phenomenon.

G) OTHER FACTORS:

The other factors which influence ventricular contractility are adequacy of ventricular contraction, ventricular diastolic dysfunction, atrial function, nervous control, hormonal control etc.

LEFT VENTRICULAR HYPERTROPHY PATTERNS AND PATHOGENESIS[32]

LEFT VENTRICULAR HYPERTROPHY:

A. Patterns of left Ventricular Hypertrophy:

The development of left ventricular hypertrophy constitutes one of the principle mechanisms by which the heart compensates for increased load. Grossman et al examined systolic and diastolic wall stress in normal subjects and in well compensated patients with chronically pressure overloaded and volume overloaded left ventricles. Left ventricular systolic stress, diastolic pressure and mass were increased approximately equally in both the pressure and volume overloaded groups. There was a substantial increase in wall thickness in volume overloaded ventricles. The latter was just sufficient to counter balance the increased radius so that the ratio of wall thickness to radius remained normal for the volume overloaded patients, while there was disproportionate thickening of ventricular wall. Thus in compensated subjects, the systolic stress are maintained relatively unchanged.

While hypertrophy constitutes a principal compensatory mechanism to an increased load, it may have an adverse effect of slowing relaxation and increasing stiffness (reducing the compliance) of the ventricle. This stiffness can result from

1. An increase in intrinsic myocardial stiffness (Amyloid infiltration, fibrosis, myocardial ischemia).
2. An increase in ventricular mass and wall thickness without alteration in intrinsic myocardial stiffness.
3. Combination of these two mechanisms.

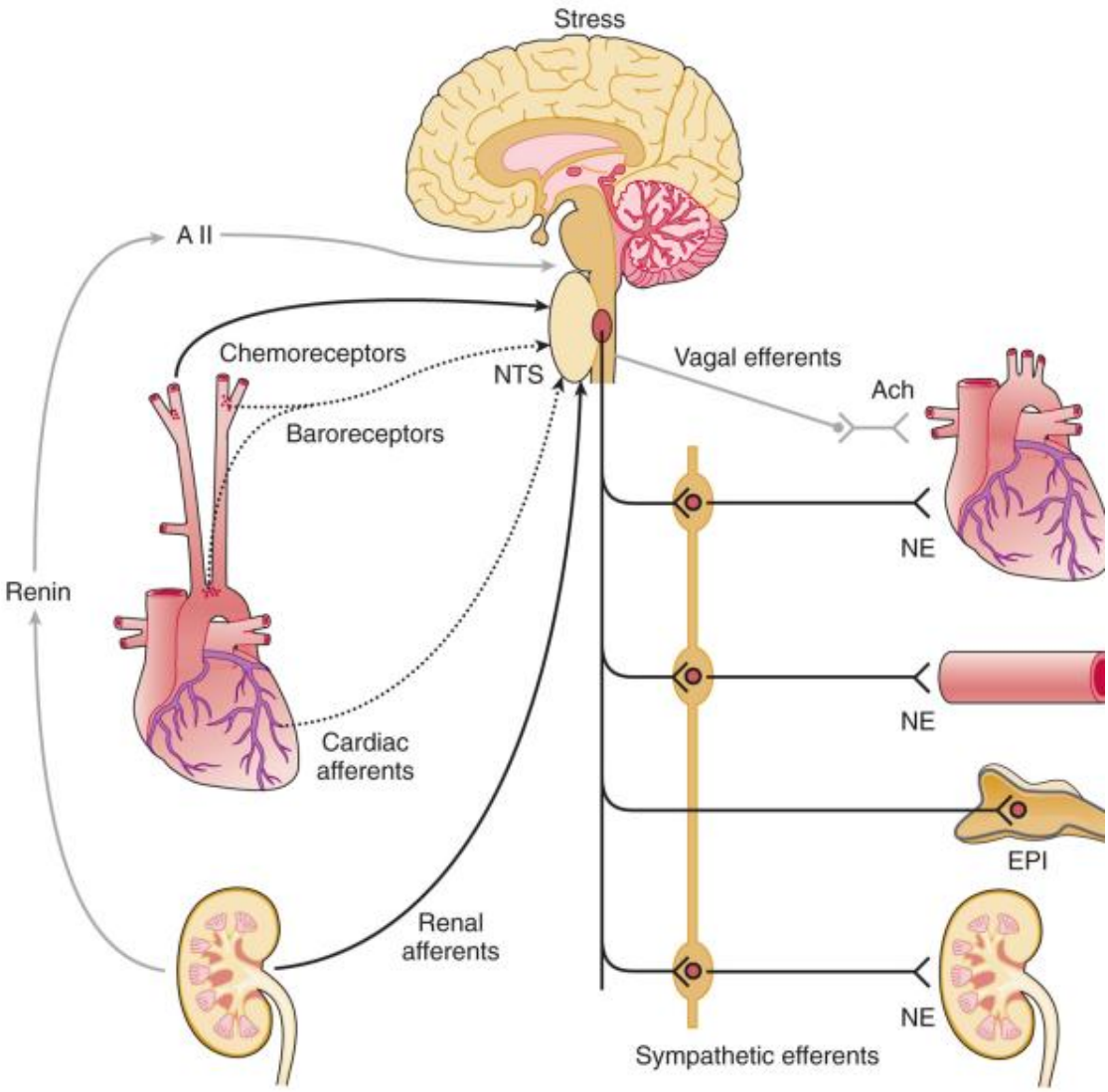
B. Etiopathogenesis of Left Ventricular Hypertrophy in Hypertension:

One of the first cellular changes that occur after the stimulus for hypertrophy is preferential synthesis of mitochondria; presumably the expanded myocardial mass provides sufficient adenosine triphosphate to meet the increased energy demands of hypertrophied cell. Later there is increase in myofibril mass. This can take place only when the DNA in the nucleus of myocytes is depressed, allowing DNA replication to occur.

The following possible stimuli have been suggested:

1. Depletion of ATP
2. Stretch of myocytes caused by a sustained increase in preload or after load (sympathetic nervous system)
3. Accumulation of products of cell degeneration caused by wear and tear.
4. Humoral stimuli such as growth hormone, thyroid hormones, epinephrine, renin-angiotensin etc.

FIGURE 4. SYMPATHETIC NERVOUS SYSTEM



Dotted arrows represent inhibitory neural influences and solid arrows represent excitatory neural influences on sympathetic outflow to the heart, peripheral vasculature and kidneys. EPI = epinephrine; NTS = nucleus tractus solitarius; NE = norepinephrine; Ach = acetylcholine; A II = angiotensin II.

1. Influence of Sympathetic Nervous System on Myocardial Hypertrophy:

Increase in heart rate and myocardial contractility as a result of pressure and volume overload could be mediated through sympathetic innervations of heart. Laks[73] described norepinephrine as a myocardial hypertrophy hormone because chronic infusion of sub hypertensive doses of non epinephrine in the dog led to left ventricular hypertrophy.

Sampson was able to show that norepinephrine stimulates the growth of isolated cultured myocardial cells through alpha receptors. Despite affecting a marked decrease in B.P, diuretics did not lead to regression of cardiac hypertrophy but did stimulate the sympathetic nervous system. This could be responsible for the failure of diuretic therapy to produce reversal of hypertrophy. Trimerzosin substances related to hydrallazine also lead to stimulation of the sympathetic nervous system and hence are unable to induce regression of hypertrophy.

On the other hand decrease in norepinephrine levels in circulation as a result of administration of methyl dopa was paralleled by unrelated reduction in LV mass without a further reduction in blood pressure. Treatment with clonidine was followed by marked decrease in left ventricular muscle mass in relation to reduction in blood pressure. So also the beta adrenergic antagonists who also encourage regression of left ventricular hypertrophy. J Wikman-Coffelt et al[74] however was able to demonstrate very protracted regression of left ventricular hypertrophy after 12 months treatment with metoprolol. Treatment with acebutalol, a B- blocker with intrinsic sympathetic activity did not produce any regression of cardiac hypertrophy. When switched to atenolol, a B-blocker without intrinsic sympathetic activity there was regression of cardiac hypertrophy. The controversial role of B-adrenergic antagonists

in the reversal of cardiac hypertrophy could also be due to the fact that the norepinephrine mediated growth of isolated myocytes can only be prevented by blockade of alpha-receptors and not the beta-receptors in experimental studies.

2. Effect of Renin-Angiotensin System on Myocardial Hypertrophy:

Because treatment with ACE inhibitors in contrast to diuretic treatment leads to regression of cardiac hypertrophy, it is possible that the elevated levels of angiotensin II which is always found during diuretic treatment have a tropic effect. The increase in circulating renin with diuretic treatment is certainly not responsible for the development of left ventricular hypertrophy because regression of cardiac hypertrophy takes place during angiotensin converting enzyme inhibitor treatment despite raised renin levels. Left ventricular hypertrophy as a consequence of clipping abdominal aorta can be completely prevented through treatment with angiotensin converting enzyme inhibitors. This suggests a strong tropic role of angiotensin II. Sahn et al[79] also reported that angiotensin II stimulates myocardial protein biosynthesis; because angiotensin enhances the effects of sympathetic tone due to its peripheral and central action on the autonomic nervous system and stimulates catecholamine secretion from adrenal medulla, the observed tropic action of angiotensin II could be mediated secondarily by catecholamines. A special angiotensin analogue that does not lead to release of catecholamine has no effect on myocardial hypertrophy either.

3. Genetic Determination of Myocardial Hypertrophy:

Asymmetric septal hypertrophy is however seen in about 14% of hypertensive patients hence, asymmetric left ventricular hypertrophy is not specific only for hypertrophic cardiomyopathy with or without obstruction.

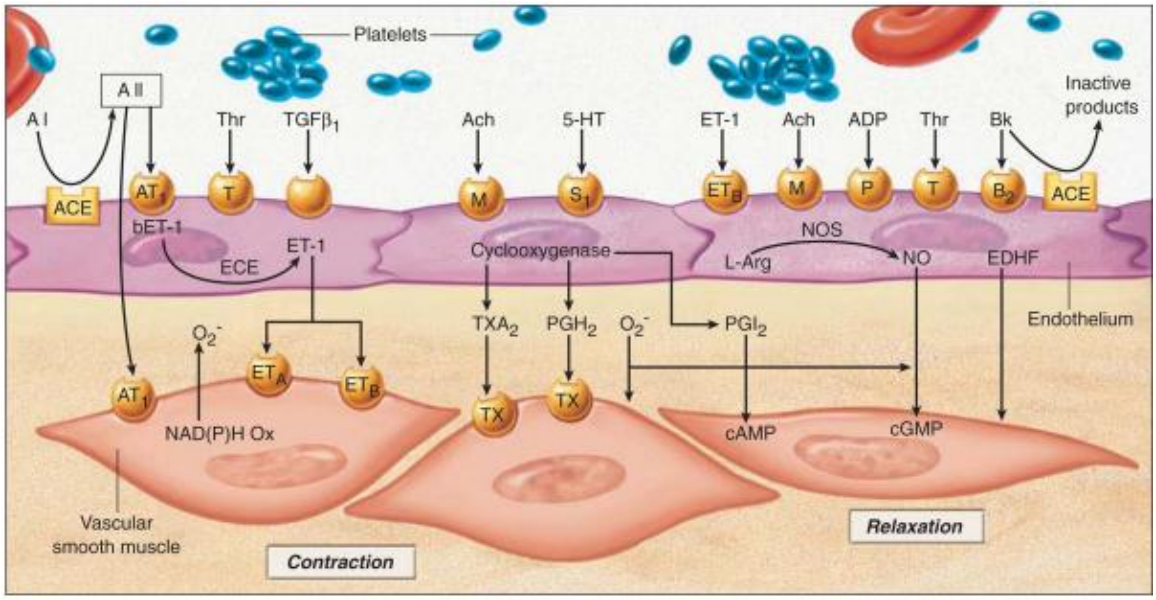
This can occur because of following reasons,

- On account of its longer radius of curvature, the ventricular septum appears to hypertrophy earlier and more markedly in responses to increase in systemic pressure load than does the posterior or anterior wall.
- The higher regional concentration of catecholamines in the ventricular septum might also be a possible explanation for higher prevalence of septal hypertrophy in hypertension.
- Because essential hypertension is genetically determined, the capacity of the heart to respond with hypertrophy might also be genetically determined. Every case of hypertensive cardiac hypertrophy could contain an individually varying genetic component which might help to explain the varying response of myocardium to anti hypertensive treatment in different patients.

4. Endothelial cell dysfunction.

FIGURE 5.

ENDOTHELIUM-DERIVED RELAXING AND CONSTRICTING FACTORS



Various blood- and platelet-derived substances can activate specific receptors (orange circles) on the endothelial membrane to release relaxing factors such as nitric oxide (NO), prostacyclin (PGI_2) and an endothelium-derived hyperpolarizing factor (EDHF). Contracting factors also are released such as endothelin (ET-1), angiotensin (A II) and thromboxane A_2 (TXA_2) as well as prostaglandin H_2 (PGH_2). ACE = angiotensin-converting enzyme; 5-HT = serotonin; Bk = bradykinin; ECE = endothelin-converting enzyme; L-Arg = l-arginine; NOS = nitric oxide synthase; O_2^- = superoxide; $TGF\beta_1$ = transforming growth factor beta-1; Thr = thrombin

The endothelial lining of blood vessels is critical to vascular health and constitutes a major defense against hypertension. Dysfunctional endothelium is characterized by impaired release of endothelial-derived relaxing factors (e.g., nitric oxide, endothelium-derived hyperpolarizing factor) and enhanced release of

endothelium derived constricting, proinflammatory, prothrombotic and growth factors. The latter include endothelin, thromboxane and transforming growth factor beta (TGF-β).

5. Vascular remodelling.

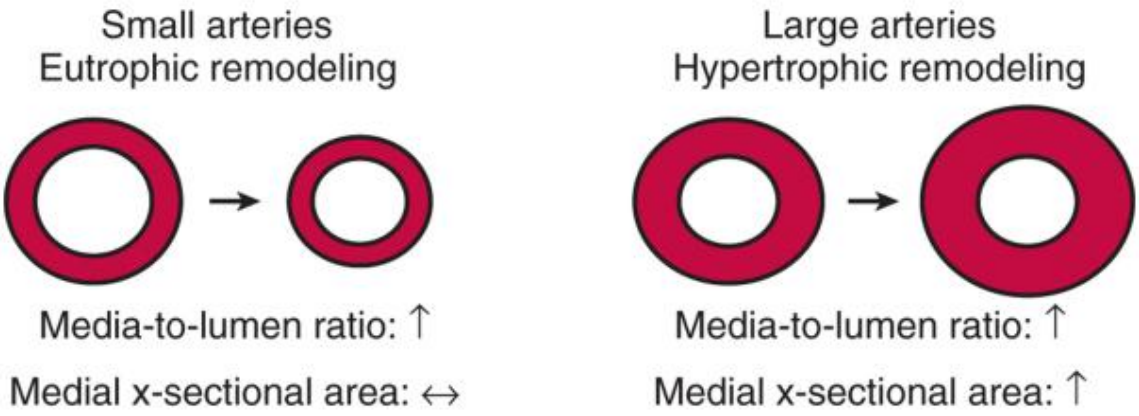


FIGURE 6. VASCULAR REMODELLING

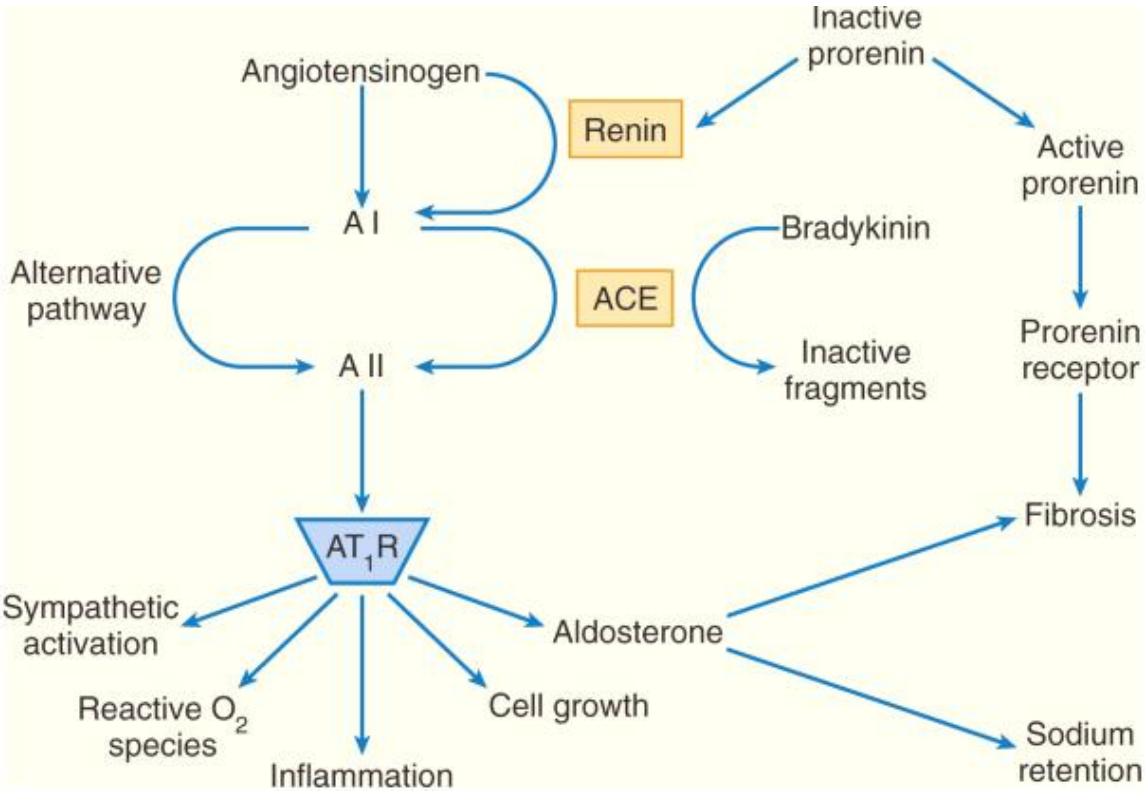
Over time endothelial cell dysfunction, neurohormonal activation and elevated BP cause remodeling of blood vessels, which further perpetuates the hypertension. An increase in the medial thickness relative to lumen diameter (increased media-to-lumen ratio) is the hallmark of hypertensive remodeling in small and large arteries. Small artery remodeling is initiated by vasoconstriction which normalizes wall stress and averts a trophic response. Normal smooth muscle cells rearrange themselves around a smaller lumen diameter; a process termed inward eutrophic remodeling. The media to lumen ratio increases but the medial cross-sectional area remains unchanged. By decreasing lumen diameter in the peripheral circulation inward eutrophic remodeling increases systemic vascular resistance which is the hemodynamic hallmark of diastolic hypertension.

In contrast large artery remodeling is characterized by the expression of hypertrophic genes triggering which increases medial thickness as well as the media to lumen ratio. Such hypertrophic remodeling involves not only an increase in the size of vascular smooth muscle cells but also an accumulation of extra cellular matrix proteins such as collagen and fibronectin because of activation of TGF-b. The resultant large artery stiffness is the hemodynamic hallmark of isolated systolic hypertension.

6. Hormonal Mechanisms: Renin-Angiotensin-Aldosterone System

Activation of the rennin - angiotensin - aldosterone system (RAAS) is one of the most important mechanisms contributing to endothelial cell dysfunction, vascular remodeling and hypertension. Renin, a protease produced solely by the renal juxtaglomerular cells cleaves angiotensinogen (renin substrate produced by the liver) to Angiotensin I which is converted by angiotensin-converting enzyme (ACE) to Angiotensin II .ACE is most abundant in the lungs but is also present in the heart and systemic vasculature (tissue ACE). Chymase, a serine protease in the heart and systemic arteries provides an alternative pathway for conversion of Angiotensin I to Angiotensin II. The interaction of Angiotensin II with G protein–coupled AT1 receptors activates numerous cellular processes that contribute to hypertension and accelerate hypertensive end-organ damage. These include vasoconstriction, generation of reactive oxygen species, vascular inflammation, vascular and cardiac remodeling and production of aldosterone, the principal mineralocorticoid. There is increasing evidence that aldosterone, Angiotensin II and even renin and prorenin activate multiple signaling pathways that can damage vascular health and cause hypertension.

FIGURE 7. THE RENIN ANGIOTENSIN ALDOSTERON SYSTEM



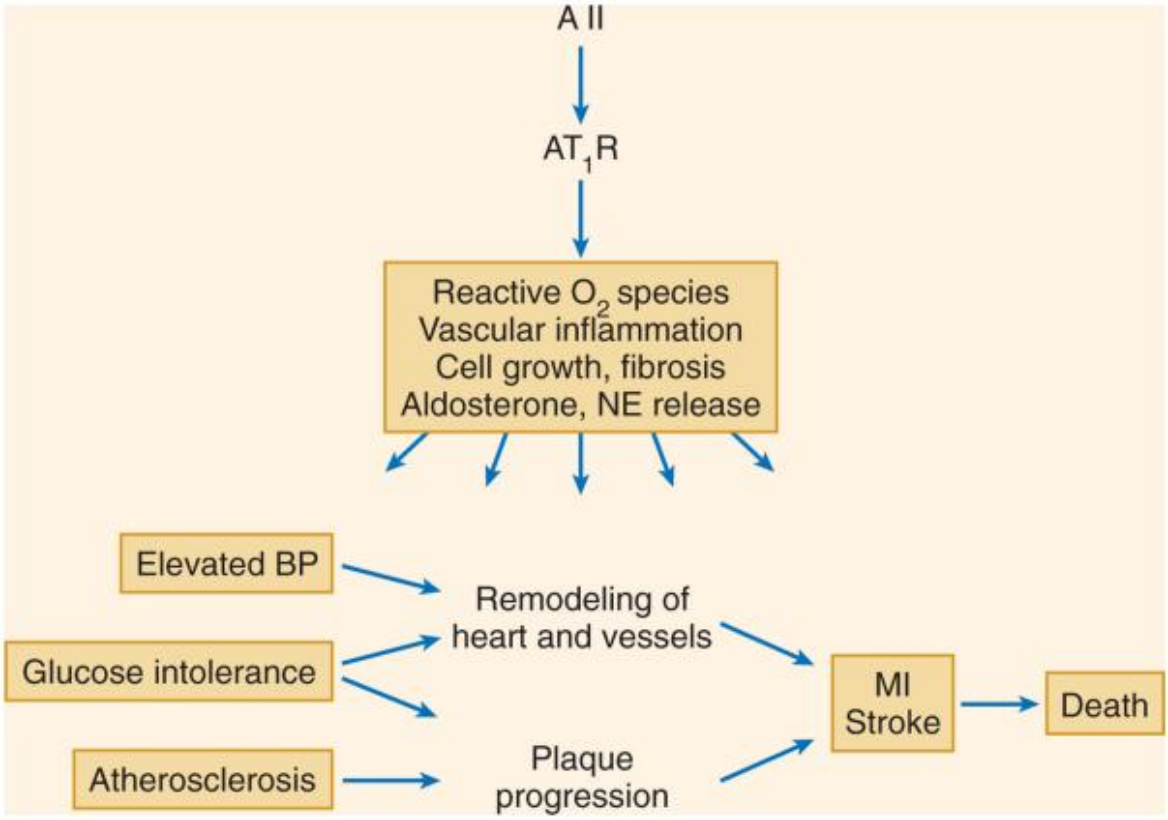
A I = angiotensin I; A II = angiotensin II; ACE = angiotensin-converting enzyme; AT_1R = angiotensin I receptor.

7. Receptor mediated actions of Angiotensin II.

Two main types of angiotensin receptors are known. AT1 receptors are widely expressed in the vasculature, kidney, adrenals, heart, liver and brain. Angiotensin I receptor activation explains most of the hypertensive actions of Angiotensin II. Furthermore enhanced AT1-mediated signaling provides a central mechanism of explanation for the frequent coexistence of elevated BP with insulin resistance and

atherosclerosis and it constitutes a major therapeutic target for interrupting every step in cardiovascular disease progression that is from vascular remodeling, formation of atherosclerotic plaque to stroke, myocardial infarction (MI) and death

FIGURE 8. SCHEMATIC REPRESENTATION OF THE CETRAL ROLE PLAYED BY ANGIOTENSINE 1 RECEPTORS



(AT_1R)= Angiotensin 1 receptor mediated signaling in cardiovascular disease progression. A II = angiotensin II; MI = myocardial infarction

C) Morphology of Cardiac Hypertrophy:

Early Stage: There is increase in number of myofibrils and mitochondria, enlargement of mitochondria and nuclei, muscle cells become longer than normal and cellular organization is largely preserved.

Advanced State: There is preferential increase in size or number of specific organelles such as mitochondria. There is irregular addition of new contractile elements in localized areas of the cell resulting in subtle abnormalities of cellular organization.

Longstanding Hypertrophy: Shows more obvious disruption in cellular organization. It shows markedly enlarged nuclei with highly lobulated membranes and breakdown of normal Z-band registration.

Late Stage of Hypertrophy: Loss of contractile elements with marked disruption of Z bands. It shows:

- Severe disruption of normal parallel arrangement of sarcomeres.
- Deposition of fibrous tissue.
- Dilation and increased tortuosity of T-tubules.

D) Changes in Membrane Proteins in Chronic Mechanical Overload of the Heart:

Cardiac hypertrophy of mechanical origin alters the density and the number of membrane proteins in an equivocal way and two distinct groups of proteins can be distinguished.

1. One group is that which is formed by the Ca ++- ATPase of the sarcoplasmic reticulum, beta one adrenergic and muscarinic receptors and the low affinity isoform of the Na +K+- ATPase. The synthesis of the proteins of this group is not activated by the hypertrophic process and because of this their number per cell does not change. The decrease in the density of Ca ++-ATPase of the sarcoplasmic reticulum reflects the adaptive slowing down of relaxation whereas the down regulation of beta receptors could have protective role.

2. The other group is formed by calcium channels and also by one isoform of Na+K+-ATPase. Their number per cell increases and their synthesis is proportionally activated with the degree of hypertrophy. The gene coding for these proteins is induced during chronic mechanical overload. Its induction could answer to requirements of the cell by having an isoform with lower affinity for sodium. Search in the field of inotropes must take an account of the fact that the keys that these drugs represent must be modified as function of the lock they have to fit into.

Blood Rheology as a Contributing Factor in Reduced Coronary Reserve in Systemic Hypertension:

Relation to blood pressure, blood flow and viscosity is described by the law of Hgen Poiseuille. There is direct relationship of blood flow to pressure gradient and radius of the vessel and inverse relationship to vessel length and blood viscosity.

The possible mechanisms of increase of plasma viscosity in arterial hypertension are,

1. Reduction of intravascular fluid volume by transcapillary shift of fluid into interstitial space (hydrostatic pressure).

2. Local synthesis of fibrinogen degradation products in precapillary vessels due to activated interleukins, thus increase in hepatic synthesis of fibrinogen and other acute phase proteins.

So the cardiac manifestations of arterial hypertension are LVH and reduced coronary vascular reserve (structural) which occurs due to mechanisms as follows:

1. Inadequate vascular capacity.
2. Reduced arteriolar diameter secondary to medial hypertrophy.
3. Reduced distensability of pre-capillary resistance vessels.
4. Some functional alterations such as elevated vasomotor tone.
5. Impaired blood fluidity.

METHODOLOGY

MATERIALS AND METHODS

In this study 130 patients with essential systemic hypertension having blood pressure level 140/90 mmHg and above and managed at SHRI B.M.PATIL MEDICAL COLLEGE AND HOSPITAL were selected.

Left ventricular mass of 130 consecutive patients with hypertension was assessed who attended B.L.D.E A's SHRI B.M.PATIL MEDICAL COLLEGE, BIJAPUR from November 2007 to November 2008 and compared with 130 control subjects of age, sex and risk factors matched.

A sample size of 130 was worked out at 95% level of confidence and with 5% margin of error and prevalence of 10% of hypertension[49] was used for calculating the desired sample size. ($Z\ \alpha = 1.96$, $d = 0.05$, $p = 0.10$, $n = 0.90$).

The formula used is,

$$n = \frac{(1.96)^2\ (p)\ (1-p)}{d^2}$$

After taking a detailed history, clinical cardiovascular examination was carried out to rule valvular, congenital and ischemic heart diseases. The patient's resting blood pressure level was recorded in the right upper limb (after 15 minutes of rest). The recording was repeated at 15 minutes interval and the average of three readings was taken.

Study design selected was case control study.

The following parameters were taken,

1. Age
2. Sex
3. Religion
4. BMI
5. Hypertension both systolic/diastolic
6. Smoking
7. Alcoholism
8. Drug abuse
9. Oral contraception
10. Family history of hypertension in first degree relatives.

The statistical formulas used were,

1. Z list
2. X^2 list
3. Mean
4. Standard deviation
5. Correlation
6. Diagrammatic representations
7. Fischer's exact test (if necessary)

INCLUSION CRITERIA

1. Patients having blood presser of 140/90 mmHg recorded in right upper limb after 15 minutes of rest. The recording was repeated at 15 minutes interval and the average of these readings was taken. Hypertension was diagnosed according to JNC VII criteria.

EXCLUSION CRITERIA

1. Patients with cardiomyopathy.
2. Patients with evidence of arrhythmias.
3. Patients with chronic obstructive lung diseases.
4. Patients with collagen vascular diseases which itself is capable of increasing thickness of myocardium.
5. Patients with aortic stenosis and aortic regurgitation.
6. Patients with well controlled hypertension on treatment.

All the 130 patients having essential hypertension were subjected to following investigation.

1. Blood
   - Hb %
   - TC
   - DC
   - ESR
2. Urine
   - Albumin
   - Sugar
   - Microscopy

3. Biochemical investigations
   - Random blood sugar
   - Blood urea
   - Serum creatinine to detect renal and metabolic causes
   - Lipid profile
4. Chest x-ray PA view to detect cardiomegaly
5. 12 lead electro cardiogram
6. Echocardiography.

All these 130 patients were subjected to 12 lead electrocardiograms with paper speed of 25mm/second.

The ECGs were analyzed with respect to standardization, rate, rhythm, p wave, P.R. interval, Axis, QRS duration and ST-T changes.

The voltage in precordial leads was analyzed according to the following criteria for detecting left ventricular hypertrophy.

1. Sokolow-Lyon Criteria:

S in V1 + R in V5 or R in V6>35mm.

2. Each ECG was scored according of Romhilt- Estes point scoring system.

1. R or S wave in any limb leads > 20mm

   Or S wave inV1 or V2 > 20 mm

   Or R wave in V5 or V6 > 30 mm……...3 points

2. Left ventricular strain

   ST segment and T wave in opposite direction to QRS complex

   With digitalis……..3 points

   With out digitalis……1 point

3. Left atrial enlargement – terminal negativity of P wave in V1 > 1mm in depth or > 0.04 seconds in duration……..…….3 points
4. Left axis deviation > -30 degree…….…….2 points
5. QRS duration > 0.09 seconds……...1 point
6. Ventricular activation time > 0.05 seconds……..1 point

TOTAL = 13 POINTS

PROBABLE LVH = 4 POINTS

LVH = 5 POINTS

Echocardiographic examination for assessment of left ventricular function and mass was done.

The pulse wave Doppler study of the mitral inflow tract was done in all cases. A detailed analysis of mitral valve spectral flow was carried out to detect associated early left ventricular diastolic dysfunction. Measurement of A.E. ratio was carried out.

The following left ventricular dimensions in the M-mode under guidance of left parasternal 2D long axis view just above the tip of the papillary muscle were measured in each case. On an average 3 observations were taken for each reading.

1. Left ventricular internal diameter at end diastole (LVIDD).
2. Interventricular septal thickness at end diastole (IVS-d).
3. Left ventricular posterior wall thickness (LVP WT-d).

Besides these, the left ventricular functions like ejection fraction and cardiac output were estimated in each case.

While measuring interventricular septal thickness, right and left septal endocardial echoes were excluded and so were posterior wall endocardial echoes while measuring posterior left ventricular wall thickness.

For left ventricular internal dimension and measuring both left septal endocardial echo and posterior wall endocardial echo were included.

Left ventricular mass was calculated in each case using the Devereux Formula. LV Mass=1.04 [{IVS (d)+ LVID (D) + LVPWT (d)}^{3}]- [LVID (d)]^3- 13.6gms where 1.04 is the specific gravity of cardiac muscle.

The control group of the study consisted of 100 normal individuals with no evidence of any heart disease between the age group of 40 and 65 years with matching age, sex and risk factors. The correlation of ECG data and echo cardio graph findings in relation to LV mass was carried out in each case.

LV mass more than 131gms in men and more than 90gms in women were considered as abnormal.

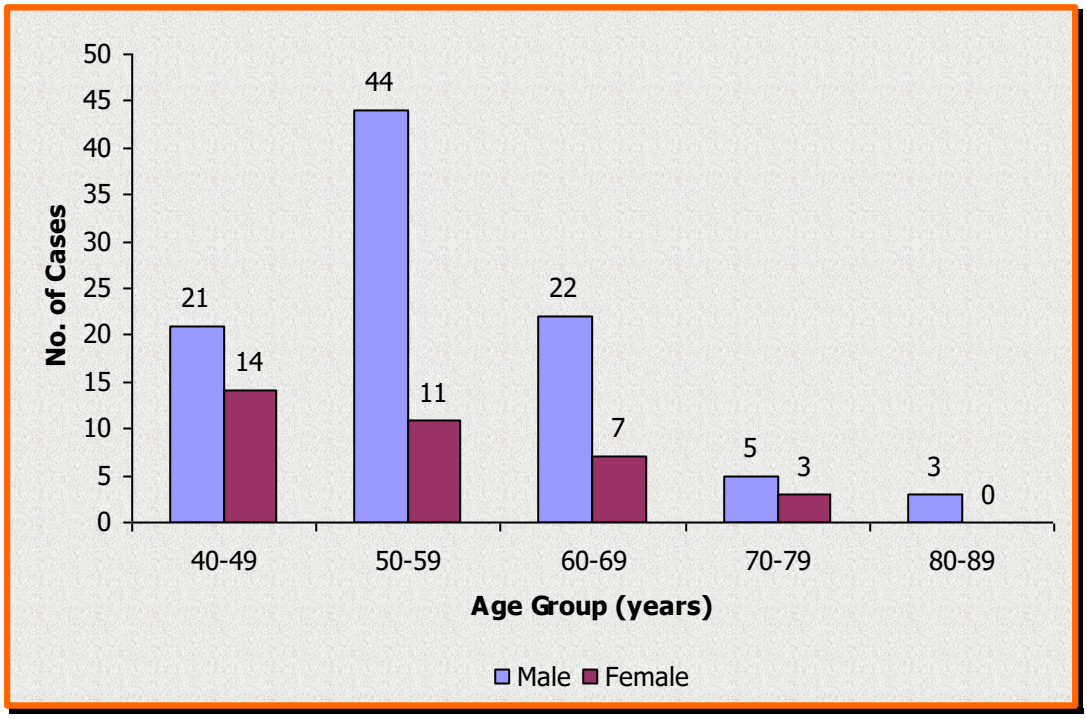
OBSERVATION AND RESULTS

In this study 130 patients with blood pressure value of 140 systolic and diastolic of 90mm Hg and above were selected who attended B.L.D.E A's SHRI B.M.PATIL MEDICAL COLLEGE, BIJAPUR from November 2007 to November 2008 and compared with 100 control subjects of age, sex and risk factors matched.

TABLE-1

AGE AND SEX DISTRIBUTION IN STUDY GROUP

Age Group in Years	Male	%	Female	%	Total No. of Patients	Percent
40-49	21	22.13	14	40.01	35	26.92
50-59	44	46.31	11	31.42	55	42.40
60-69	22	23.15	07	20.00	29	22.45
70-79	05	05.26	03	08.57	08	06.18
80-89	03	03.15	00	00	03	02.05
Total	95	100	35	100	130	100

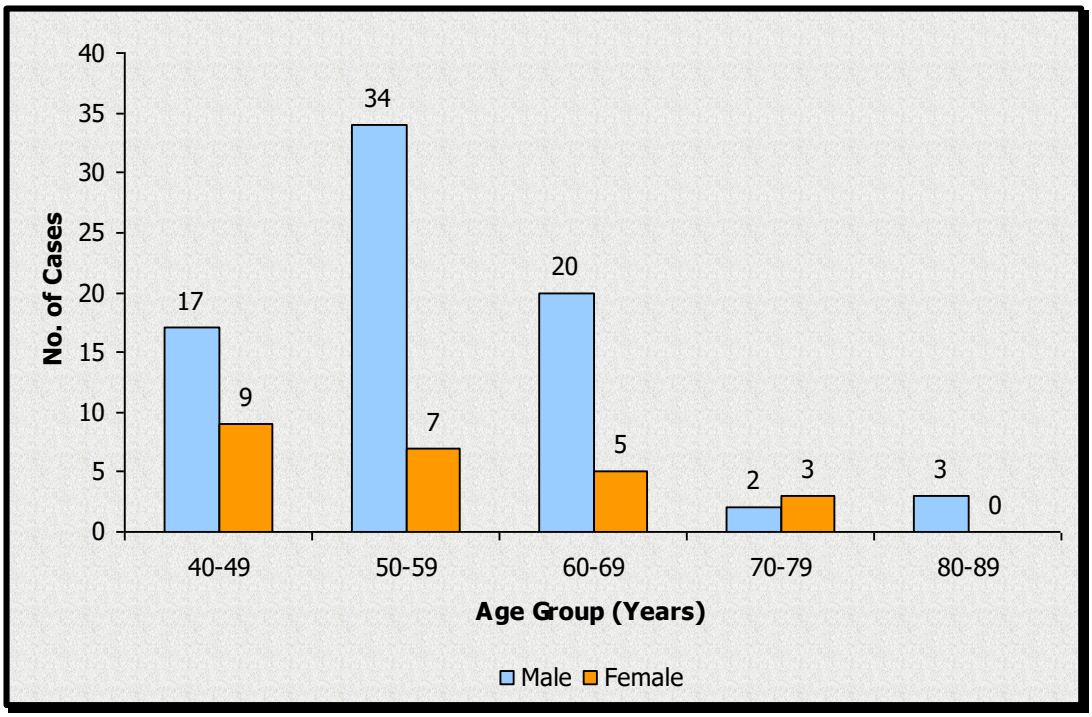


From the above table No.1, we observed that the incidence of hypertension is common in males 95 patients (73%) compared to females 35 patients (27%). The commonest age Group is 50-59 years having 55 patients (42.40%). Least common is 80-89 years age group with 3 patients (2.05%). Among males the commonest age group is 50-59 years 44 patients (46.31%), but in females 40-49 years age group is common 14 patients (40.00%).

TABLE 2

AGE AND SEX DISTRIBUTION IN CONTROL GROUP

Age Group in Years	Male	%	Female	%	Total	Percent
40-49	17	22.36	09	37.50	26	26
50-59	34	44.73	07	29.16	41	41
60-69	20	26.34	05	20.83	25	25
70-79	02	02.63	03	12.51	05	05
80-89	03	03.94	00	00	03	03
TOTAL	76	100	24	100	100	100

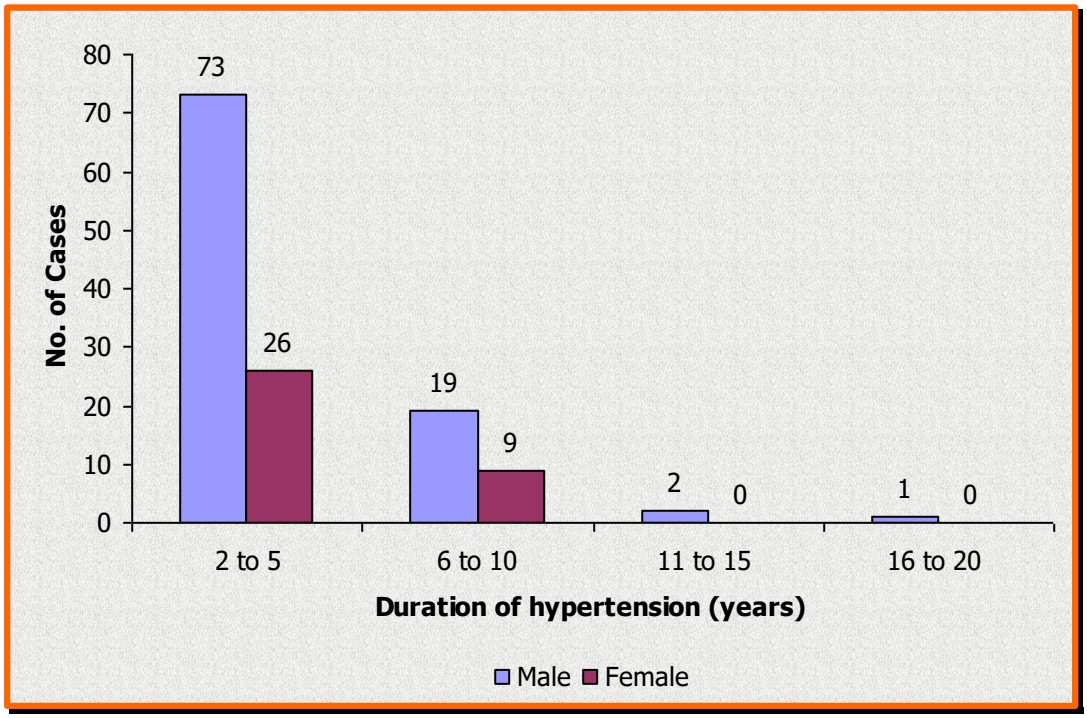


Control group showed commonest age group between 50 to 59 years in males (34 patients).Among females' commonest age group was 40 to 49 years (09 patients). Least numbers of patients were found in age group of 80 to 89 years.

TABLE-3

DURATION OF HYPERTENSION IN STUDY GROUP

Years	Male	Percentage	Female	Percentage
0-5	73	76.84%	26	74.28%
6-10	19	20.00%	09	25.72%
11-15	02	02.11%	00	--
16-20	01	01.05%	00	--
Total	95	100%	35	100%

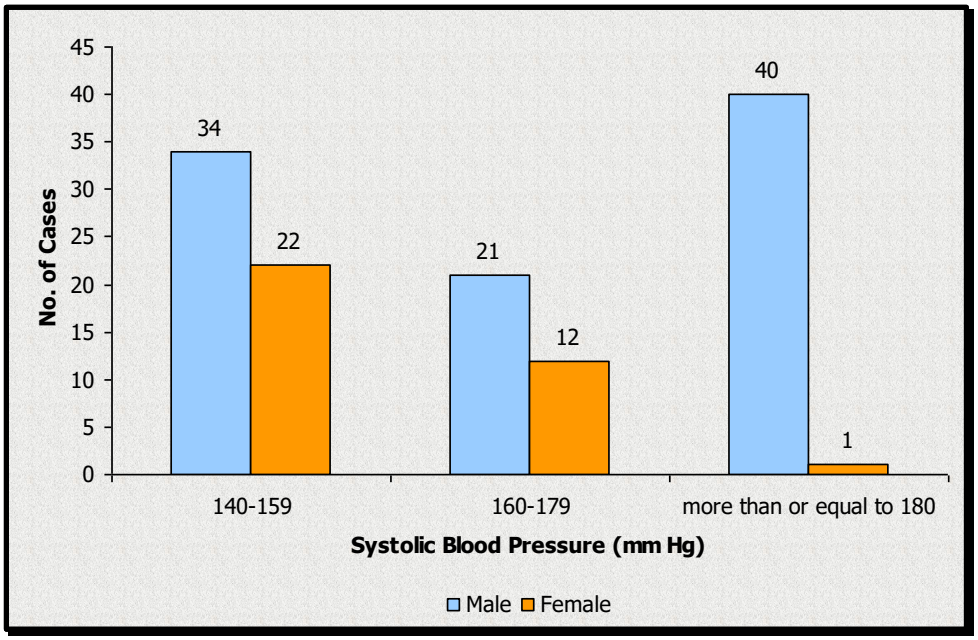


From the above table it is evident that the duration hypertension of 5 years and less in male patients was 73 (76.84%) and in female it was 26 (74.28%) was maximum. Control group had no evidence of hypertension.

TABLE-4

SYSTOLIC BLOOD PRESSURE IN STUDY GROUP

Systolic B.P.(mmHg)	Male	%	Female	%	Total-Percentage
140-159	34	35.78	22	62.85	56- 43.09%
160-179	21	22.12	12	34.28	33- 25.38%
>-180	40	42.10	01	02.87	41- 31.53%
TOTAL	95	100	35	100	130- 100%

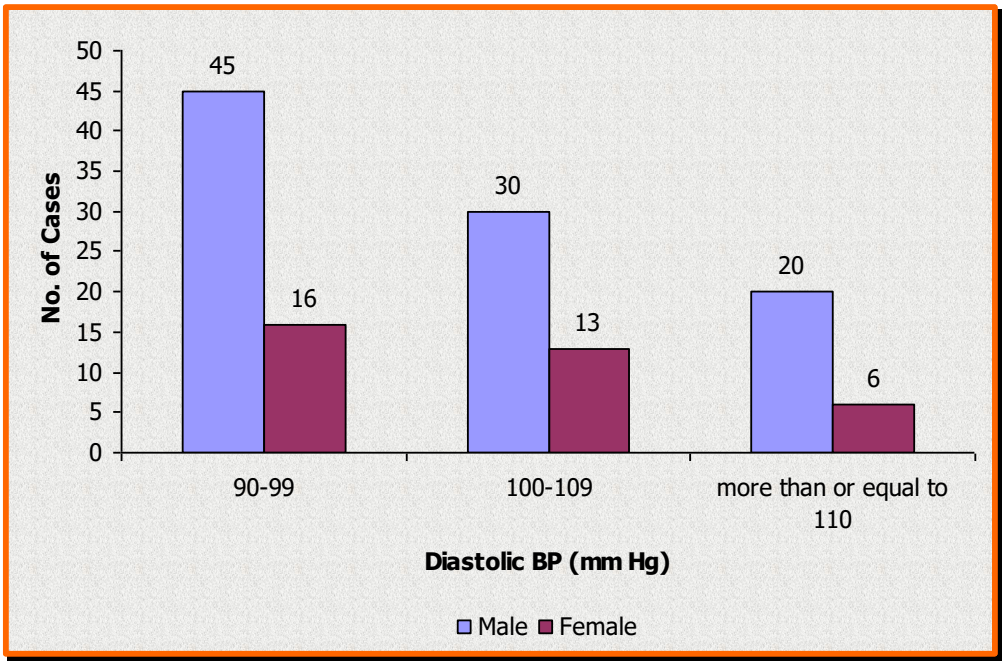


It is evident from table 4 that the maximum number of patients were with systolic blood pressure of 140-159mm Hg was 43.09% (56 patients), least number of patients were in 160-179 Hg group, 25.38% (33 patients) and more than 180 mm Hg group, 31.53% (41 patients). When sex was taken into consideration, blood pressure of 140-159mm of Hg in systolic group was common in both sexes. (34 patients and 22 patients in males and females respectively) and in males least common was 160-179mm of Hg group (21 patients) compared to females who showed least common in > 180mm of Hg in 1 patient. Control group had systolic blood pressure levels of < 140 mm of Hg.

TABLE-5

DIASTOLIC BLOOD PRESSURE IN STUDT GROUP

Diastolic BP	Male	%	Female	%	Total	Percentage
90-99	45	47.36	16	45.71	61	46.92%
100-109	30	31.57	13	37.15	43	33.07%
>- 110	20	21.07	06	17.14	26	20.01%
TOTAL	95	100	35	100	130	100%



It was observed from table 5, that the commonest diastolic hypertension group was 90-99 mm of Hg with 46.92% patients, which was also commonest group among both sexes. In males 45 patients were in 90-90 mm of Hg and in females 16 patients were in 90-99 mm of Hg range.

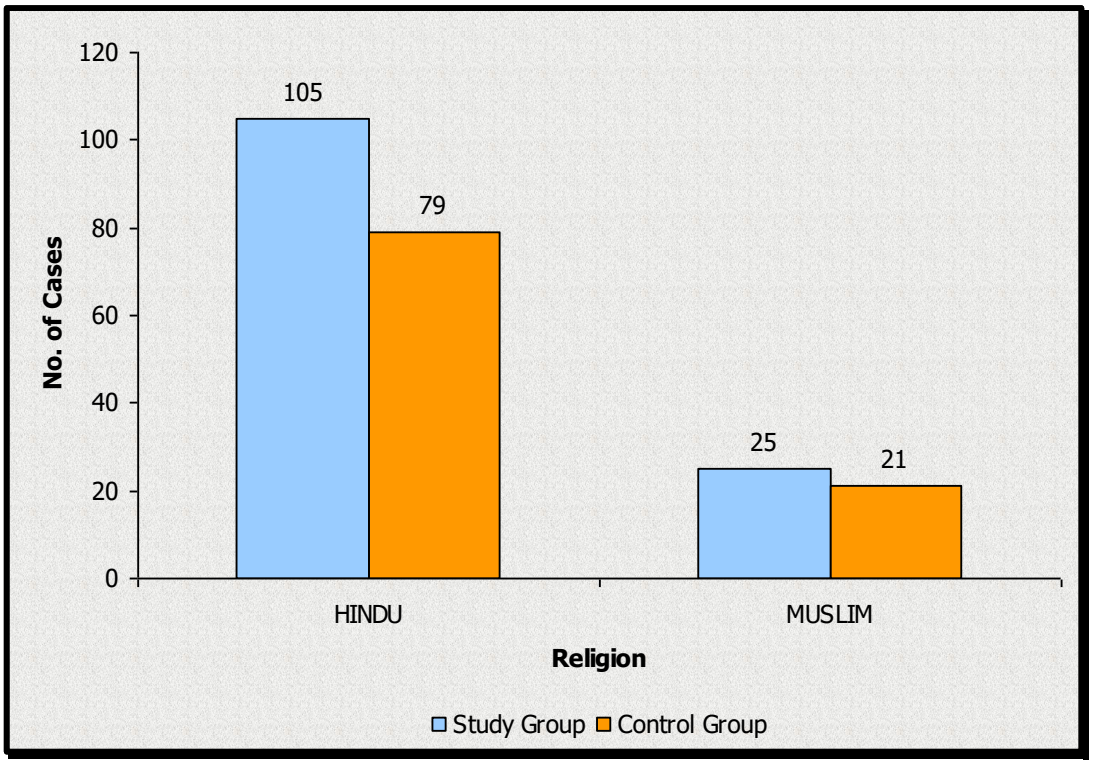
The least common group was > 110 mm of Hg group (26 patients), which was also present in both sexes. In males 20 patients and females 6 patient were in the group of diastolic blood pressure of >110 mm of Hg. The other group 100-109mm of Hg had 43 patients among which males were 30 and females were 13. Control group had diastolic blood pressures of <90 mm of Hg.

TABLE-6

RELIGION WISE DISTRIBUTION OF CASES

Religion	Study Group	Percentage	Control Group	Percentage
HINDU	105	80.76	79	79
MUSLIM	025	19.24	21	21
TOTAL	130	100	100	100

X2 = 0.11 p < 0.05 NOT SIGNIFICANT



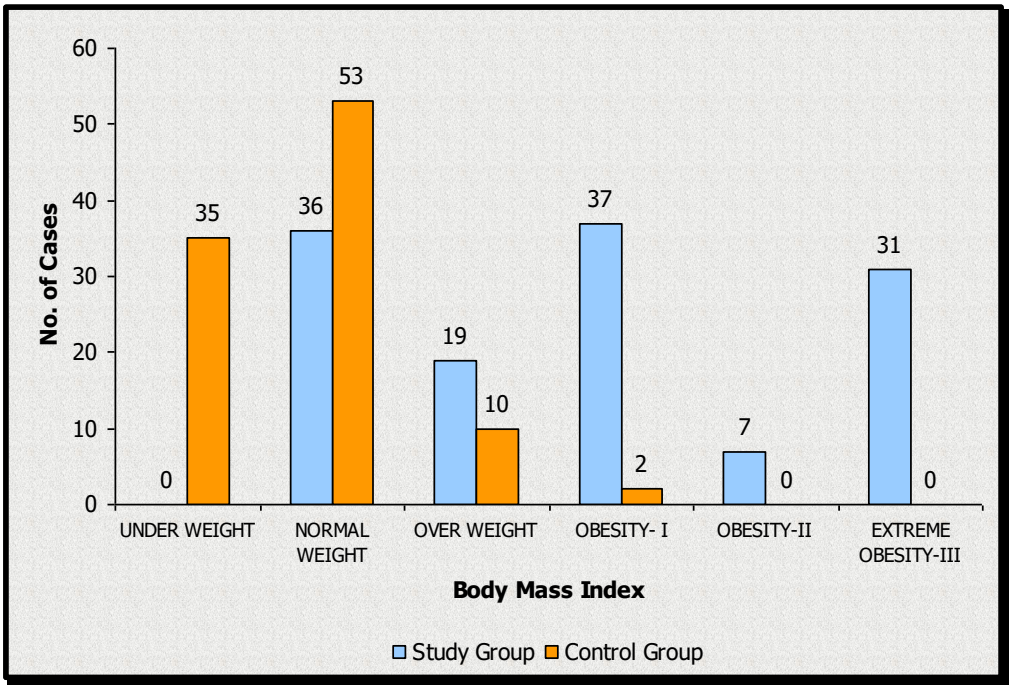
There is no significant difference between study group and control group in the religion wise selection of cases.

From above table it was observed that hypertension was common in Hindu community that is 80.76% (105 patients). In Muslim community it was found to be 19.24% (25 patients). Control group was found to have 79% of Hindus and 12% of Muslims.

TABLE -7

DISTRIBUTION OF CASES ACCORDING TO BMI

	STUDY GROUP	PERCENTAGE	CONTROL GROUP	PERCENTAGE
UNDER WEIGHT	00	00	35	35
NORMAL WEIGHT	36	27.69	53	53
OVER WEIGHT	19	14.63	10	10
OBESITY- I	37	28.46	02	02
OBESITY-II	07	05.38	00	00
EXTREME OBESITY-III	31	23.84	00	00
TOTAL	130	100	100	100



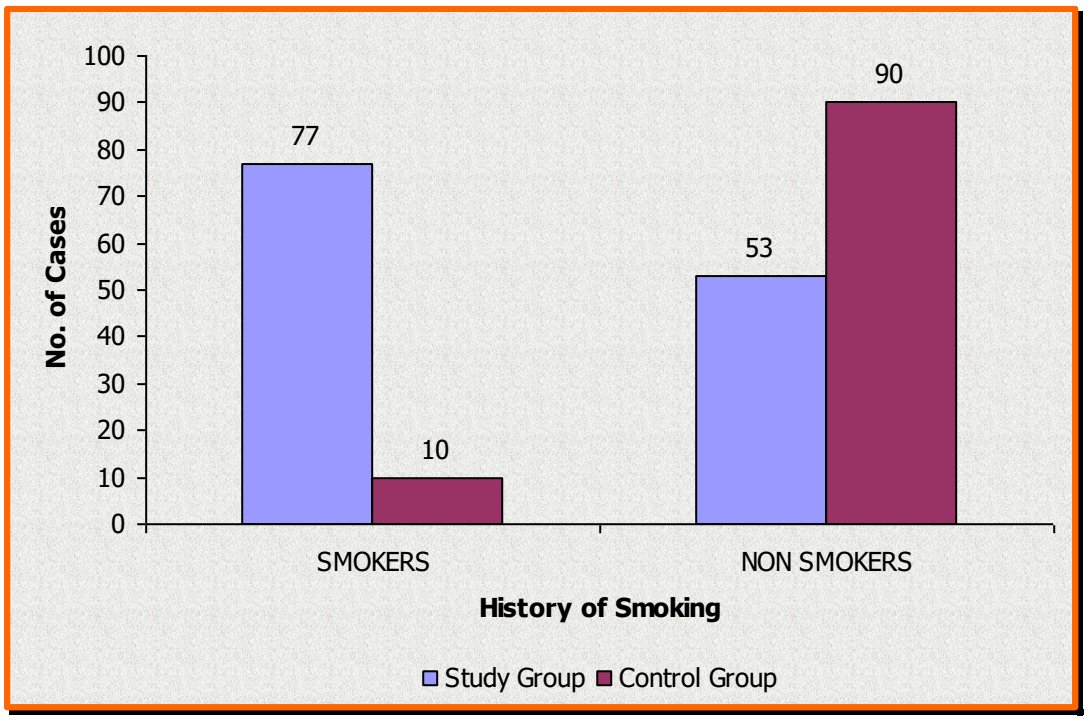
It is evident from above table that hypertension was common in patients with grade I obesity 28.46% (37 patients). The second commonest group was with normal weight that is 27.69% (36 patients).Grade III obesity was found in 23.84% of patients (31 patients). Hypertension was not found in under weight group of patients. In control group it was evident that 53% of patients had normal weight and 35% of patients were in under weight group. Over weight group had 10% of patients.

TABLE-8

SMOKING HISTORY

	STUDY GROUP	PERCENT	CONTROL GROUP	PERCENT
SMOKERS	77	59.24	10	10
NON SMOKERS	53	40.76	90	90
TOTAL	130	100	100	100

X2=58.2 P < 0.001



There is significant difference among smokers and non smokers in study and control group. It is statistically significant.

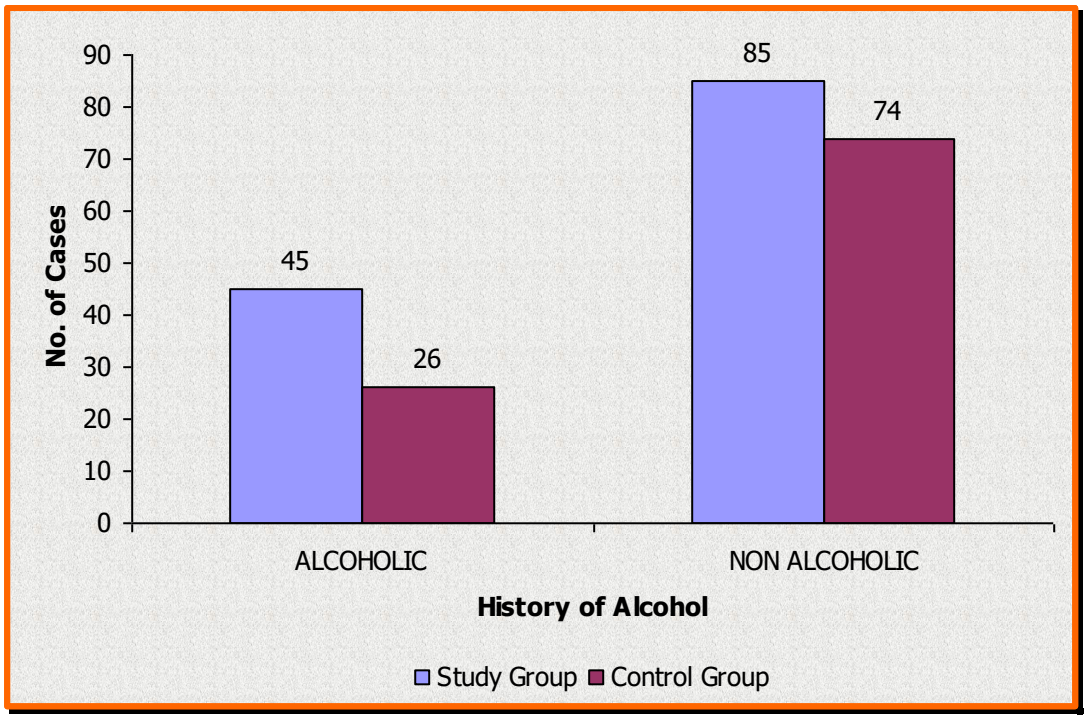
From the above table it is evident that patients having history of smoking had increased evidence of hypertension 59.24% (77 patients). Patients who were non smokers were 53 with 40.76%. In control group 10% were found to be smokers and 90% were found to be non smokers.

TABLE-9

ALCOHOLISM

	STUDY GROUP	PERCENTAGE	CONTROL GROUP	PERCENTAGE
ALCOHOLIC	45	34.62	26	26
NON ALCOHOLIC	85	65.38	74	74
TOTAL	130	100	100	100

X2 = 1.96. P > 0.05



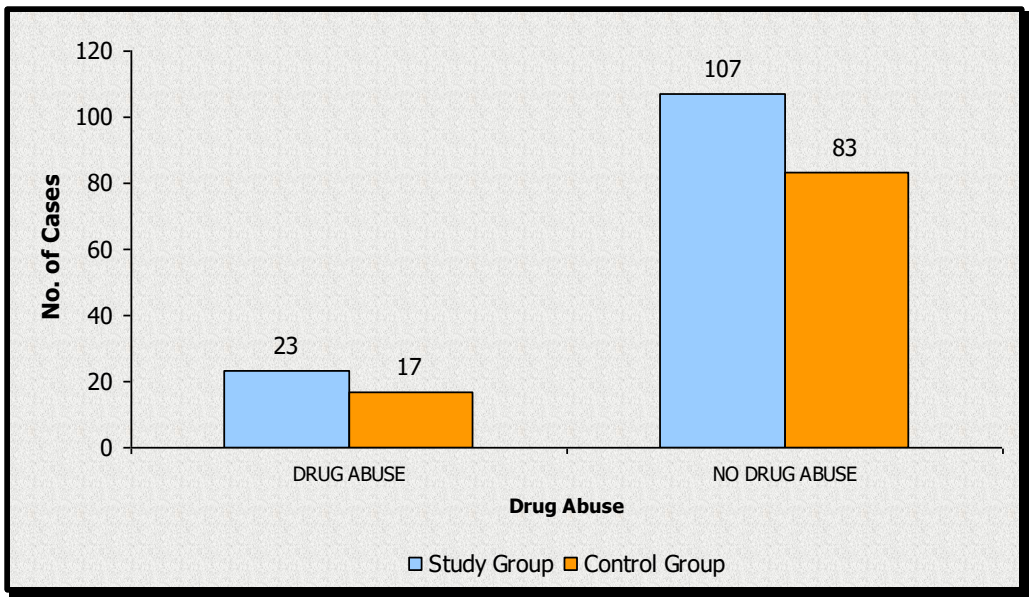
There is no significant difference of alcoholism among study and control groups. It is evident from above table that hypertension was common in non alcoholics that is 65.38% (85 patients). In alcoholic patients hypertension was less common that is 34.62% (45 patients). In control group it was found that 74% were nonalcoholic and 26% were alcoholic.

TABLE-10

DRUG ABUSE

	STUDY GROUP	PERCENT	CONTROL GROUP	PERCENT
DRUG ABUSE	23	17.69	17	17
NO DRUG ABUSE	107	82.31	83	83
TOTAL	130	100%	100	100

X2 = 0.02 P > 0.05



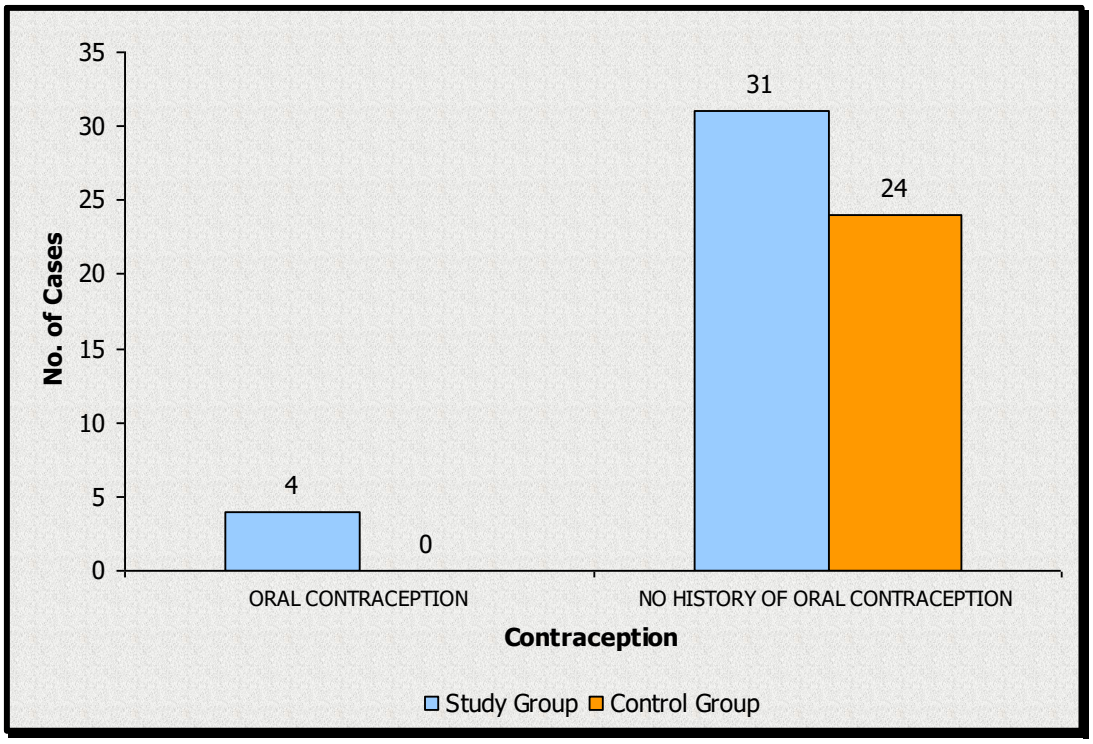
There is no significant difference between study and control group in drug abuse.

From above table it was evident that 17.69% (23 patients) of patients had history of drug abuse. They used analgesics for their headache. 82.31% of patients (107 patients) had no evidence of any drug abuse. In control group 17% of patients were found to have history of drug abuse and 83% of patients were found to have no evidence of drug abuse.

TABLE-11

ORAL CONTRACEPTION IN FEMALES

	STUDY GROUP	PERCENT	CONTROL GROUP	PERCENT
ORAL CONTRACEPTION	04	11.42	00	00
NO HISTORY OF ORAL CONTRACEPTION	31	88.58	24	24
TOTAL	35	100%	24	24

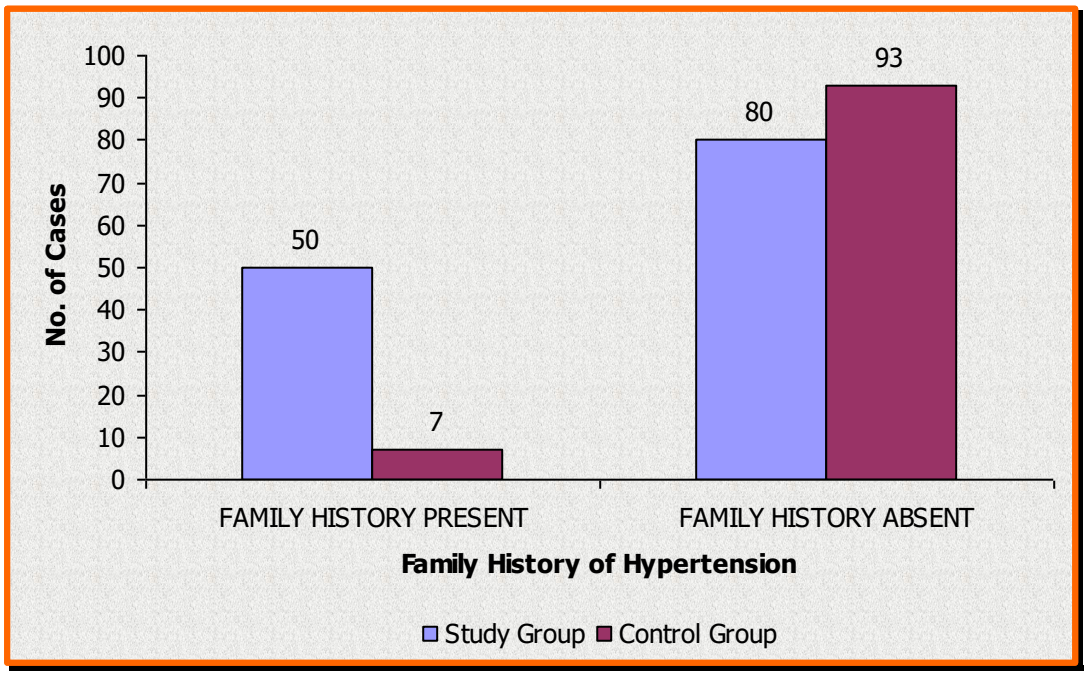


It is evident from the above table that 11.42% (04 patients) of females with history of hypertension had evidence of using oral contraceptive pills for minimum period of more than 3 years. It was observed that 88.58% of females had no evidence of taking oral contraceptive pills. In control group no females have taken oral contraceptive pills.

TABLE-12

FAMILY HISTORY OF HYPERTENSION

	STUDY GROUP	PERCENT	CONTROL GROUP	PERCENT
FAMILY HISTORY PRESENT	50	38.46	07	07
FAMILY HISTORY ABSENT	80	61.54	93	93
TOTAL	130	100%	100	100

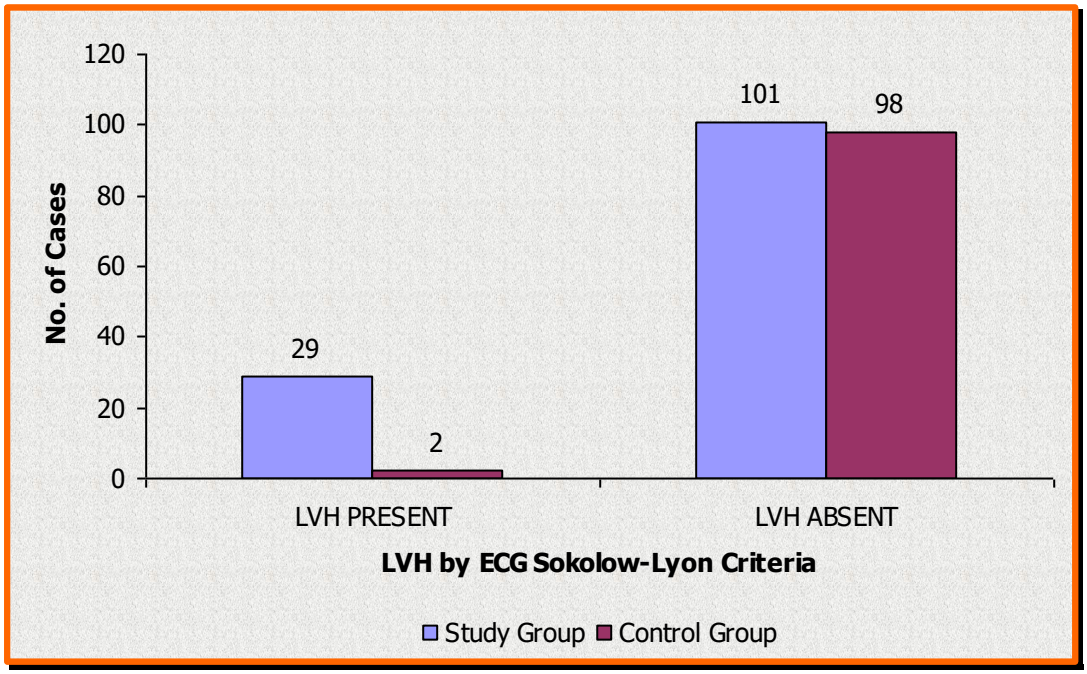


Family history of hypertension was taken in first degree relatives. It was evident from the above table that family history of hypertension was found in 38.46% of patients (50 patients) and there was no evidence of family history in 61.54% (80 patients). There were 7% of patients in control group having a positive family history of hypertension and 93% of patients did not had any family history of hypertension.

TABLE 13

LVH BY ECG SOKOLOW-LYON CRITERIA

	STUDY GROUP	CONTROL GROUP
LVH PRESENT	29	02
LVH ABSENT	101	98

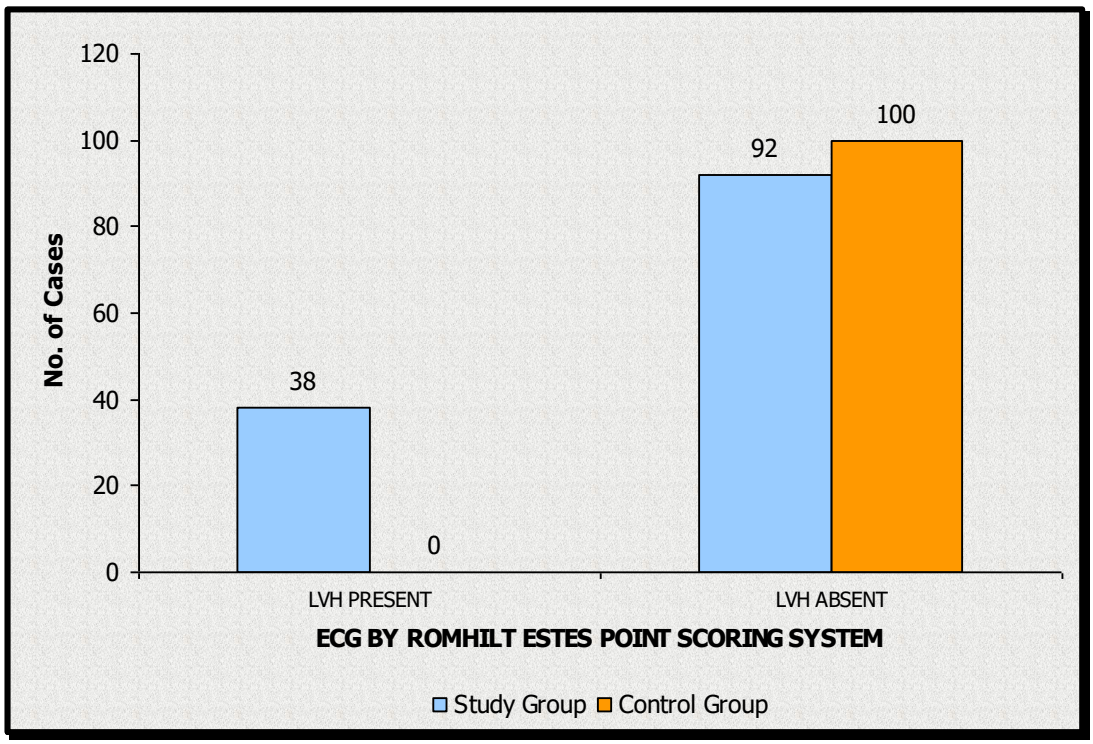


It is evident from the above table that LVH was evident in 29 patients that is in 22.31% of patients in whom 21were males (22.11%) and 8 were female patients (22.11%). LVH was not present in 101 patients that is 77.69%. This group includes 74 males (77.89%) and 27 females (77.14%). Control group had 2 patients showing LVH and 98 showing no evidence of LVH.

TABLE-14

ECG BY ROMHILT ESTES POINT SCORING SYSTEM

	STUDY GROUP	CONTROL GROUP
LVH PRESENT	38	0
LVH ABSENT	92	100



It is evident that LVH was present in 38 patients (29.23%), among which 24 (25.27%) patients were males and 14(40.00%) patients were females. LVH was absent in 92 patients (70.77%) among which males were 71 patients (74.73%) and females were 21 patients (60.00%).Control group had no evidence of LVH.

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TABLE-15

RELATIONSHIP OF ECG LVH BY SOKOLOW-LYON CRITERIA TO ECHOCARDIOGRAPH LVH

	STUDY GROUP		CONTROL GROUP	
ECG BY SOKOLOW LYON CRITERIA	LVH PRESENT	LVH ABSENT	LVH PRESENT	LVH ABSENT
	29	101	02	98
ECHO CARDIOGRAPH	130	00	04	96
PERCENTAGE	ECG- 22.30% ECHO- 100%			

From the above table 15 it is evident that, LVH by ECG criteria was present in 29 patients (22.31%), and LVH was absent in 101 patients (77.69%), but echo cardiograph showed LVH in all 130 patients. Control group had LVH by ECG in 2 patients and by echocardiograph in 4 patients. 98 patients did not have evidence of LVH in ECG.

TABLE-16

RELATIONSHIP OF ECG LVH BY ROMHILT ESTES SCORING SYSTEM TO ECHO CADIOGRAPH LVH

	STUDY GROUP		CONTROL GROUP	
ECG BY ROMHILT ESTES SCORING SYSTEM	LVH PRESENT	LVH ABSENT	LVH PRESENT	LVH ABSENT
	38	92	00	00
ECHOCARDIOGRAPH	130	00	00	00
PERCENTAGE	ECG- 29.23% ECHO- 100%			

From table-16 we observed LVH by Rhomhilt-Estes scorings system was present in 38 patients (29.23%) and absent in 92 patients (70.77%). But echo cardiograph showed LVH in all 130 patients.

TABLE-17

SENSITIVITY AND SPECIFICITY OF ECG CRITERIA

	Sokolow-Lyon Criteria	Romhilt-Estes Scoring System
Sensitivity	22.30%	29.23%
Specificity	100%	100%
Predictive value for positive test	100%	100%
Predictive value for negative test	55.38%	41.54%
Percentage of false positive	0%	0%
Percentage of false negative	77.70%	70.77%

From table-17 it is evident that LVH by Sokolow Lyon criteria showed sensitivity of 22.30% compared to Romhilt Estes Showing 29.23%. Specificity and predictive value for positive test was present in 100% patients, in Sokolow-Lyon criteria and Romhilt Estes, percentage of false positive was 0% in both systems, but percentage of false negative was 77.70% in Sokolow Lyon criteria and 70.77% in Romhilt Estes scoring system. Combined criteria showed sensitivity of 25.76%.

TABLE-18

COMPARISON OF LV MASS IN STUDY AND CONTROL GROUP

	MALE		**FEMALE**		**TOTAL**	
	NO	**MEAN±SD**	**NO**	**MEAN±SD**	**NO**	**MEAN±SD**
STUDY GROUP	95	179.07±36.62	35	103.78±15.14	130	158.79±32..27
CONTROL GROUP	76	112.84±5.88	25	73.36±9.41	100	103.36±6.89

All the 130 patients were detected to have increased left ventricular mass by echo cardiograph.

The mean left ventricular mass was179.07±36.62gms in males and 103.78± 15.14gms in females.

All the 130 patients were detected to have left ventricular diastolic dysfunction on echocardiograph study.

Comparison of both study and control group was done using Z test.

Comparing study group and control group in LV mass,

$Z = 19.11$ $p < 0.01$ HIGHLY SIGNIFICANT.

Comparing males and females of study group,

$Z = 16.73$ $p < 0.01$ HIGHLY SIGNIFICANT.

Comparing males of study and control group,

$Z = 17.45$ $p < 0.01$ HIGHLY SIGNIFICANT.

Comparing females of study and control group,

$Z = 9.5$ $p < 0.01$ HIGHLY SIGNIFICANT.

Comparing males and females of control group,

$Z = 19.35$ $p < 0.01$ HIGHLY SIGNIFICANT.

DISCUSSION

Echo-cardiograph criteria for left ventricular hypertrophy has been shown to have excellent sensitivity, specificity and accuracy when compared with post mortem left ventricular mass and its reliability has also been confirmed by angiographic studies[17]. The population studied in this study permitted the assessment of Sokolow Lyon criteria and Romhilt-Estes scoring criteria in a clearly defined clinical settings. Both electro-cardiograph criteria appeared to have a low sensitivity and specificity.

In our study hypertension is common in the age group of 50-59 years- (43.09%) and age range was from 40-89 years. Similarly Srinivas et al[75] found hypertension common in the age group of 50-59 years (28%), in the age range of 20-69 years, but Alka et al[77] found that hypertension common in the age group of 50-59 (48%) age range to be 16 to 70 years.

It was found that hypertension was common in males (73%) compared to females (26%). In males it was common in the age of 50-59. Similarly Srinivas et al[75] and Alka et al[77] found hypertension to be common in males but age group of 50-59 years was common in both studies. However, Janet F.Lewis et al[34] found hypertension to be common in females compared to males.

When systolic hypertension was considered it was found to be common in 140-159mm of Hg group and diastolic hypertension in 90-99mm of Hg group. Similarly Gopinath Menon et al[78] found similar findings but Srinivas et al[75] found hypertension to be common in mild hypertension group in systolic and diastolic hypertension group.

This study showed that there is significant difference between the people of different religions in study and control group. Hypertension is more common in Hindu group when compared to Muslim group. This can be attributed to the fact that majority of patients visiting the hospital where our study was conducted were Hindus. Levin et al[80] found that there was an inverse relationship between religious commitment and prevalence of hypertension. They also found that the salutary effect of religion on blood pressure can be explained by combination of the following correlates of religion like, the promotion of health-related behavior, hereditary predispositions in particular groups, the healthful psychosocial effects of religious practice, and the beneficial psychodynamics of belief systems, religious rites, and faith. Hence religion does not directly increase the risk of development of hypertension. Multiple social factors play a role in increasing risk of hypertension.

Our study showed that hypertension was common in patients with grade I obesity. The second commonest group of people was with healthy weight (27.69%). The patients included under the study group belonged to lower socio economic group coming from in and around Bijapur. Obesity was not found to be prevalent among them. But Krzysztof Narkiewicz[81] found that obesity is an independent risk factor for the development and progression of hypertension, cardiovascular diseases and chronic kidney disease. There is growing evidence that obesity and associated metabolic abnormalities may induce and accelerate complications in essential hypertension. The clustering of obesity and other features of the metabolic syndrome are considered important implications for prevention of hypertension particularly with regard to interventions targeted at reducing visceral obesity which have beneficial effects on reducing cardiovascular and renal morbidity.

Julius S et al[82] showed that over weight people that is people with grade I obesity have increased risks of developing hypertension when compared to other grades of obesity. This was similar to our study.

Ashish Aneja et al[83] showed that the distribution of body fat is considered important in the genesis of the obesity-hypertension syndrome with a predominantly central distribution being particularly ominous. Hence obesity along with various other factors like hypercholesterolemia and increased waist hip ratio may act together and increase the risk of development of hypertension.

The present study showed that there was a significant increase in the incidence of hypertension in smokers (59.24%) when compared to non smokers (40.76%). It was statistically significant. Kannel WB et al[84] found similar results while correlating smoking and hypertension. They also stated that smoking increases the cardiovascular risk at any level of blood pressure causing coronary heart disease, stroke and cardiac failure. J Levenson et al[85] found that smoking and hypertension may change the flow properties of the blood and the behavior of the arterial wall and this may explain the arterial damage observed in cigarette smokers and hypertensive patients.

Comparison of alcoholism in study group and control group of patients showed that patients in both study and control groups had similar incidence of hypertension. There was no significant difference in number of patients in both groups. Michael et al[86] found similar results in their study. They found that a standard drink of < 10gms of alcohol twice a week was not associated with significant risk of developing hypertension. But increase in the number and quantity of alcohol intake was significantly associated with increase in systolic blood pressure.

Steffens AA et al[87] concluded in their study that individuals with an African ancestry who consumed larger amounts of ethanol were at higher risk of developing hypertension. This risk was not explained by a binge drinking pattern or addiction to alcohol.

The present study also compared the incidence of hypertension in the study group and control group with respect to drug abuse. Many patients used analgesics for headache. But intake of analgesics for various reasons was not associated with increased risk of hypertension. It was also found that there was no significant statistical difference between the study and control group in relation to drug abuse. In the population under our study total number of patients addicted to analgesics is less; hence there may be no statistical difference between the two groups. Ferdinand KC[88] showed that substance abuse and hypertension are an important health concern, especially in adolescent and young adults presenting with elevated blood pressure and associated cardiovascular conditions. Here illicit drugs were found to be associated with hypertension including cocaine, marijuana, amphetamines, and methylenedioxy methamphetamine. These remain potential sources of acute or newly diagnosed hypertension but analgesic abuse was not associated with increase risk of hypertension.

It was found in our study that intake of oral contraceptives was not associated with increased risk of developing hypertension. The present study also showed that there was no significant difference with respect to oral contraceptive intake in both study and control groups. The females under our study were not habituated to take oral contraceptives. This is similar to the study done by Goodlin R C et al[89] who studied 120 female patients after giving them eight types of oral contraceptives and

matched these patients for age with 100 control subjects. They were unable to show any significant rise in blood pressure in treated subjects. Another study done by Woods et al[90] showed that there was a mild increase in systolic blood pressure in patients who have taken oral contraceptives.

It was found during the present study that a positive family history of hypertension was present in 38.46% of patients in the study group when compared to 7% in patients in the control group. Hence it is evident that there is increased occurrence of hypertension among study group with positive family history when compared to control group. Kathleen A[91] proved that a positive family history is definitely associated with increased risk of hypertension. Suurkula MB et al[92] assessed left ventricular function in young men with a positive family history of hypertension for two generations. The findings were compared with three control groups: one age, sex, and weight matched group with a negative family history of hypertension. The group with a positive family history of hypertension, in comparison with the normal blood pressure control group, was heavier, had higher blood pressure, increased left ventricular wall thickness, increased left ventricular mass, and signs of changes in diastolic and systolic left ventricular function. There were no differences in these variables between the group with a positive family history and the other two control groups. Hence this shows that though family history plays an important role in increasing risk of hypertension, increased body weight is also an important factor contributing for the risk of development of hypertension.

TABLE 19

SENSITIVITY AND SPECIFICITY OF VARIOUS STUDIES

Criteria	**Sensitivity**	**Specificity**
Sokolow-Lyon	22	100
Estes	42	96
Cornells	54	97
Nathaniel Reicheke	21	95
Gopinath Menon	18	00
Santosh Kausal	71	81
Present study		
a) Estes	29.23	100
b) Sokolow	22.30	100

After taking ECG the left ventricular hypertrophy was calculated by Sokolow-Lyon criteria and Romhilt-Estes scoring systems. We found that left ventricular hypertrophy by Sokolow Lyon criteria sensitivity of 22.30% and specificity of 100% and by Romhilt-Estes scoring, sensitivity of 29.23% and specificity of 100%. In Santosh Kausal et al[38] study LVH by Ronhilt-Estes[11] criteria found sensitivity of 71% and specificity of 81%. In the original study of Sokolow-Lyon et al[5] sensitivity was 22%. But Alka et al[77] found sensitivity 18% and specificity of 100%; Devereux et al[20, 39, 48] found sensitivity of 60%. Nathaniel Reichek et al[36] found LVH by Romhilt-Estes criteria a sensitivity of 50% and by Sokolow Lyon criteria sensitivity of 20% both had specificity of 95%. In Estes original study sensitivity was 42%, specificity 96% and Cornell et al[40] showed sensitivity 54% and specificity of 97%.

In the present study echocardiograph parameters of left ventricular hypertrophy could be detected in 130 out of 130 patients while both the electrocardiographic criteria taken together could detect only in 67 patients with sensitivity of 25.76%. Similarly Thomas M. McFarland et al[37] found that left ventricular hypertrophy by electrocardiogram in 48% and by echocardiograph in 100% of patients. Daniel et al[25] found that less than 10% of hypertensive patients showed electrocardiographic evidence of left ventricular hypertrophy in contrast to the high prevalence of cardiac abnormalities detected by echo-cardiograph.

Both the Sokolow-Lyon and Romhilt-Estes scoring criterion failed to show any correlation with left ventricular mass as both these criteria were insensitive in several patients with significant increases in left ventricular mass. In this study it was found that mean left ventricular mass was 179.07$\pm$36.62gms in males and 103.78$\pm$15.14gms in females. Francis et al[35] found mean left ventricular mass of 208+18 gms and Alka et al[77] found left ventricular mass of 160.06 gms.

The relationship between clinical measurement of blood pressure and left ventricular hypertrophy in systemic hypertension appears to be weak in most studies. In our study there is no correlation between blood pressure levels and left ventricular mass. In S. Giaconi et al[33] study they showed a direct correlation between left ventricular mass and blood pressure levels. However the Framingham data reported by Levy et al[28] showed modest but statistically significant correlation between systemic arterial blood pressure and left ventricular mass.

TABLE 20

LEFT VENTRICULAR MASS IN GRAMS IN DIFFERENT STUDIES OF NORMAL POPULATION

	Males	Females
Framingham Study	92±19.5	72±14
Devereux et al	84±24	68±20.5
Trivedi et al	118	109
Gopinath Menon	95.23	87.67
Present study	112.84±5.88	73.36±9.41

In the control group which had 100 normal individuals (76 males and 24 females). The mean left ventricular mass in males was 112.84±5.88gms and females 73.36±9.41gms. Corresponding values obtained by Gopinath Menon et al[78] in Indian study was 95.23gms in males. In Trivedi et al[29] study showed mean left ventricular mass in males 118gms and in females 109gms.

The Framingham study[28] showed 92±19.5 gms in males and 72±14gms in females. The corresponding values obtained by Devereux et al[20, 39, 48] in their study were 84.24 gms for men and 68±20.5 gms in females. Our study suggests that in hypertensive patients the reliability of electrocardiographic criteria is poor and the reliability of echocardiographic criteria is better.

CONCLUSION

This study suggests that echo-cardiography is far superior tool in the detection of LVH in hypertensive patients compared to electro-cardiogram.

SUMMARY

This study was done in 130 patients with hypertension and their ECG was recorded and their echocardiography was done. LVH was estimated in ECG by Sokolow-Lyon criteria and Romhilt-Estes point scoring system. Echocardiography was done for all the patients. Left ventricular mass was calculated by using Devereux formula as follows,

LV mass = $1.04\ [(IVS\ (d) + LVID\ (D) + LVPWT\ (d)]^3] - [LVID\ (d)]^3 - 13.6$ grams

Here 1.04 is the specific gravity of cardiac muscle.

The study design was case control study.

The study group of 130 patients was compared with a control group of 100 patients with matching age, sex and risk factors.

The conclusion at which we arrived is that:

Sex-Male: Female Ratio is 2.7:1.

Among systolic hypertension commonest group of patients was in the range of 140-159 mm of Hg (43.09%). Least common group was in the range of 160-179mm of Hg (22.12%) in males and in females it was in the range of > 180 mm Hg (2.87%).

Among diastolic hypertension commonest group was found in the range of 90-99 mm of Hg (46.53%) in both sexes. Similarly least common group was in >110mm Hg (19.10%) in both sexes.

LVH by Sokolow criteria was present in 22.31% of patients and absent in 77.69% of patients.

Similarly in Romhilt-Estes scoring system it was found that LVH was present in 29.23% of patients and it was absent in 70.77% of patients. Comparison of Echo-cardiograph showing LVH was done with ECG showing LVH.

The results obtained were as follows,

Echocardiograph showed LVH in 100% of patients.

Sokolow Lyon criteria showed LVH in 22.31% of patients.

Rohmilt-Estes point scoring system showed LVH in 29.23%of patients.

The mean left ventricular mass was179.07±36.62gms in males and 103.78± 15.14gms in females.

Mean left ventricular mass-158.79±32.27gms.

TABLE 21

SENSITIVITY AND SPECIFICITY OF ELECTRCARDIOGRAPHIC AND ECHOCARDIOGRAPHIC CRITERIAS

	Sensitivity	Specificity	Percentage False-ve	Percentage Falsse +ve	Predictive value for	
					+ve Test	-ve Test
Sokolow-Lyon	22.30%	100%	77.70%	0%	100%	55.38%
Romhilt Estes	29.23%	100%	70.77%	0%	100%	41.54%
Echo cardiograph	100%	100%	0%	0%	-	-

Hence, it is concluded that echocardiograph is highly reliable and sensitive compared to electrocardiogram in the assessment of LVH in these patients. Though this procedure is expensive it is almost available in every institution in our country. In view of the recent reports regarding the prognostic implication of LV hypertrophy, it is desirable that all hypertensive patients should under go echocardiographic studies to rule out left ventricular hypertrophy possibly depending upon cost factor and availability of the facility.

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PROFORMA

LEFT VENTRICULAR MASS IN HYPERTENSION CORRELATIVE STUDY OF ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY

Name Registration no.

Age D.O.A

Sex

Religion D.O.D

Occupation

Address

Present History

1. Giddiness-

    Duration Yes/No

    Any postural variation

    Any diurnal variation

2. Headache -

    Duration Yes/No

    Site

    Type

    Any diurnal variations

    Any aggravating factors

    And relieving factors

3. Blurring of vision Yes/No

4. Chest pain-

Duration Yes/No

Site

Radiations

Type

Any aggravating factors

And relieving factors

5. Palpitation- Yes/No

Duration

Type

Any aggravating factors

And relieving factors

6. Breathlessness-

Duration

Grade

Any aggravating factor

Any relieving factor

H/o Orthopnoea

H/o P.N.D

7. Swelling of lower limbs Yes/No

Duration

Diurnal variation

8. Puffiness of face- Yes/No

Duration

9. Any other symptoms Yes/No

Past History

History of hypertension

- Duration
- Treatment history

H/o MI/ Angina

H/o DM

H/o R.H.D

H/o Drug intake

- Analgesic
- Oral contraceptives

Family history of hypertension

Appetite

Smoking

- Duration
- Number of cigarettes / Beedis pack year smoked

Alcohol

- Duration and Quantity

Sleep

General Physical Examination

Cardiovascular system

Pulse

BP

JVP

Inspection

- Precordial bulge
- Parasternal pulsations
- Epigastric pulsation
- Any engorged veins

Palpation

Apical impulse

- Site
- Character
- Thrill

Palpable A/2 P/2

Percussion of the cardiac borders.

Auscultation

Carotid Bruit

Mitral area

Tricuspid area

Aortic area

Pulmonary area

Respiratory system

- Position of trachea
- Respiratory movements
- Liver dullness
- Breath sounds
- Any added sounds

Per abdomen

Organomegaly

Free fluid

- Shifting dullness
- Fluid thrill

Any other palpable mass.

Any bruit in renal angle/lumbar region.

SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH

CENTRE. BIJAPUR . 586103.

CONSENT FORM

TITLE OF RESEARCH: "LEFT VENTRICULAR MASS IN YPERTENSION – CORRELATIVE STUDY OF ELECTROCARDIOGRAPHY AND ECHOCARDIOGRAPHY"

GUIDE : DR. S. S. DEVARMANI

P.G. STUDENT : DR. Ruqia Asna Rabah

PURPOSE OF RESEARCH:

I have been informed that the purpose of this study is to evaluate the significance of left ventricular hypertrophy in hypertension.

PROCEDURE:

I understand that I will undergo detailed history and clinical examination laboratory investigations and Echocardiography.

RISKS AND DISCOMFORTS:

I understand that there is no risk involved and I may experience mild pain during the above-mentioned procedures.

BENEFITS:

I understand that my participation in this study will help in evaluation of significance of left ventricular hypertrophy in hypertension. This will help in better management of patients with hypertension and also helps in reducing cardio vascular morbidity and mortality.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital record and will be subjected to confidentiality and privacy regulations of hospital. If the data is used for publications the identity of the patient will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may be asked for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or withdraw from study at any time.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me during the study I will get medical treatment but no further compensations.

(Signature of Guardian) **(Signature of patient)**

(If the patient is conscious, well oriented and fully aware)

KEY TO MASTER CHART

BMI	**:**	Body mask index
DPB	**:**	Diastolic blood pressure
ECG	**:**	Electro cardiograph
F	**:**	Female
gms	**:**	Grams
LV	**:**	Left Ventricle
M	**:**	Male
m^2	:	Meter Square
pt	:	Patient
SBP	**:**	Systolic blood pressure
VAT	**:**	Ventricular activation time

MASTER CHART FOR STUDY GROUP

Sl. No.	Name	IP No.	Age	Sex	Systolic BP	Diastolic BP	ECG by Sokolowlyon Criteria	ECG by Romhilt Estes Criteria							LV Mass (gms)	Religion	Smoker/ Non-smoker	BMI (Kg/m²)	Alcoholic/ non-alcoholic	Drug abuse	Contraceptive use	Family History
								Voltage 3 Poins	ST-T chanes 3 points	Left axis deviation 2 points	Left atrial enlargement 3 points	QRS Duration > 0.09 sec 1 point	VAT 1 point	Total								
1	RASHEED AHMED	25172	44	M	160	100	25								163.44	MUSLIM	Yes	34	No	Yes		Present
2	RAFEEQ KHAN	12508	50	M	150	90	30								165.00	MUSLIM	Yes	32	No	Yes		Present
3	BASHEER AHMED	12509	60	M	150	90	18		3					3	221.40	MUSLIM	Yes	26	No	Yes		Present
4	SANGAPPA MUDNAL	13508	56	M	140	90	11			2				2	208.70	HINDU	Yes	26	No	No		Absent
5	MANTAPPA.NAMGO	26211	60	M	170	96	28		3					3	148.38	HINDU	Yes	46	No	No		Present
6	MALLAPPA UKKALI	38573	64	M	140	90	46								228.60	HINDU	Yes	48	Yes	No		Absent
7	SANGAMESH.G	12212	48	M	150	94	18								191.45	HINDU	Yes	34	Yes	No		Absent
8	MALLAPPA UKKALI A PATIL	16292	54	M	150	94	21								148.57	HINDU	Yes	46	Yes	No		Absent
9	SITARAM .W	26208	66	M	200	110	25								152.20	HINDU	Yes	36	No	No		Absent
10	CHANDU.R	32156	54	M	150	90	26								141.98	HINDU	Yes	48	No	No		Absent
11	SHIVAPPA.H	12218	58	M	160	94	5		3					3	177.48	HINDU	Yes	44	Yes	No		Absent
12	DUNDAPPA.A	32029	47	M	170	110	24								270.28	HINDU	Yes	44	Yes	No		Absent
13	LAXMAN UKKALI	33401	55	M	140	90	23			2				2	155.18	HINDU	Yes	46	No	No		Absent

14	SOMANGOUDA	29262	45	M	180	120	35	3	3					6	263.88	HINDU	Yes	34	No	No		Present
15	MALLAPPA PUJARI	16025	49	M	150	100	14		3					3	174.95	HINDU	Yes	26	Yes	Yes		Present
16	SUBHASH METRI	21527	52	M	170	100	15								190.00	HINDU	Yes	26	Yes	No		Present
17	BADRINATH.P	35121	49	M	150	106	19								172.20	HINDU	Yes	27	No	No		Absent
18	SHIVAJI.K	159	80	M	200	100	23		3			1		4	169.91	HINDU	Yes	48	No	No		Present
19	JINNAH SAB	120	67	M	200	100	26		3			1		4	189.86	MUSLIM	No	33	Yes	No		Absent
20	GUNDAPPA.H	12892	45	M	140	100	22		3					3	180.20	HINDU	No	32	Yes	Yes		Present
21	PRABHUGOUDA.P	19004	56	M	210	130	27		3					3	218.95	HINDU	Yes	32	No	Yes		Absent
22	ABDULLAH	36527	58	M	140	94	13								135.95	MUSLIM	Yes	32	No	No		Present
23	SANJEEVE.K	18331	48	M	160	100	36		3					3	218.54	HINDU	Yes	46	No	Yes		Absent
24	GUNDDAPPA.P	18905	68	M	196	110	15			2				2	195.91	HINDU	Yes	46	No	No		Present
25	SANGAPPA.H	18903	54	M	140	96	14								138.80	HINDU	Yes	45	No	Yes		Absent
26	NINGANGOUDA	18907	48	M	180	110	30			2				2	161.00	HINDU	Yes	32	No	No		Present
27	PANDURANGA	4928	65	M	140	90	48	3	3	3				9	161.00	HINDU	No	32	Yes	No		Absent
28	SHRISHAILAPPA.D	21205	47	M	180	100	18		3					3	177.27	HINDU	No	34	Yes	No		Absent
29	GURANNAGOUDA	28312	55	M	140	90	14			2		1	1	4	170.00	HINDU	No	36	Yes	No		Absent
30	GUNDAPPA TELI	12050	55	M	180	110	32		3			1	1	5	136.12	HINDU	Yes	36	No	No		Absent
31	GUNDAPPA MALLAD	29052	69	M	160	96	20								154.76	HINDU	No	34	Yes	No		Absent
32	RUDRAGOUDA PATI	16205	84	M	166	110	52	3	3					6	224.62	HINDU	Yes	36	No	No		Present
33	VITHAL RAO CHAVA	33227	68	M	140	100	21		3					3	139.96	HINDU	No	45	Yes	No		Absent

34	SHABBIR AHMED	16512	56	M	180	110	46								254.76	MUSLIM	Yes	45	No	Yes		Absent
35	SHAFIQ KHAN	32520	58	M	170	90	38		3				1	4	152.86	MUSLIM	No	46	No	Yes		Absent
36	DUNDAPPA ARVAT	64383	65	M	140	94	30		3				1	4	168.50	HINDU	Yes	45	No	No		Present
37	SATISH AMTE	32250	56	M	150	100	28								160.15	HINDU	Yes	34	Yes	No		Absent
38	GAGAPPA TALIKOTI	21265	45	M	220	120	23		3	2			1	6	225.11	HINDU	Yes	34	Yes	No		Present
39	GYASUDDIN	21562	50	M	190	100	52	3					1	4	158.45	MUSLIM	Yes	32	Yes	No		Absent
40	ANIL NAVI	41256	58	M	140	100	30		3	3			1	7	151.04	HINDU	Yes	32	Yes	No		Present
41	MD. QASIMSAB	17586	45	M	140	98	28		3					3	133.66	MUSLIM	No	32	No	Yes		Absent
42	MD.SAMAD	33194	55	M	150	98	54	3	3					6	163.76	MUSLIM	No	32	No	Yes		Present
43	PRABHUGOUDA.P	16285	55	M	190	110	35		3			1	1	5	171.12	HINDU	Yes	34	No	Yes		Present
44	REVU RATHOD	29523	64	M	200	110	14		3					3	170.99	HINDU	Yes	34	No	Yes		Present
45	BASAVARAJ PATIL	42905	53	M	190	100	24								140.80	HINDU	Yes	34	No	No		Absent
46	IBRAHIM SAB	35632	63	M	180	100	36		3				1	4	155.49	MUSLIM	Yes	46	No	No		Absent
47	CHANDRAKANTH BHI	21256	50	M	160	98	29	3						3	163.85	HINDU	No	46	No	No		Absent
48	NINGAPPA PUJARI	12209	55	M	210	130	48	3			3			6	167.07	HINDU	No	46	Yes	No		Present
49	SIDDANGOUDA.P	26121	48	M	180	90	44		3			1	1	5	231.45	HINDU	No	46	No	No		Present
50	RAGHAVENDRA.K	25628	52	M	180	100	38					1	1	2	231.45	HINDU	Yes	32	Yes	No		Absent
51	NARAYAN RAO	16298	62	M	180	100	47	3				1	1	5	142.95	HINDU	Yes	46	No	No		Absent
52	ALLAHBAKSH	33272	66	M	200	110	46	3	3			1	1	8	160.00	MUSLIM	No	34	Yes	No		Absent
53	SIDDAPPA KUMBAR	32582	48	M	200	110	20		3	2		1	1	7	229.41	HINDU	Yes	35	No	No		Present

54	IMAMSAAB	14508	56	M	140	90	25			3				3	143.36	MUSLIM	No	36	Yes	No		Present
55	BASAPPA.D	34507	58	M	180	116	26				3	1	1	5	171.56	HINDU	No	46	No	No		Absent
56	SUBHASH GANNUR	16221	63	M	180	110	29	3	3					6	146.72	HINDU	Yes	45	No	No		Absent
57	BASAPPA.K	34422	59	M	140	94	15			2		1	1	4	237.10	HINDU	No	26	Yes	No		Absent
58	RAMAPPA TULJAPU	34383	54	M	140	90	38					1	1	2	134.36	HINDU	Yes	26	No	No		Absent
59	SIDDRAM GUTTA	34655	61	M	160	90	22	3	3		3	1	1	11	217.36	HINDU	Yes	26	No	No		Absent
60	BASAVARAJ BIRADAR	20256	66	M	160	90	38		3	2	3	1	1	10	156.94	HINDU	Yes	36	Yes	No		Absent
61	KATTEPPA.P	29505	86	M	180	110	17		3		3			6	175.85	HINDU	Yes	32	No	No		Absent
62	SIDDANNA PULKE	46215	59	M	140	90	21								194.94	HINDU	Yes	46	Yes	No		Absent
63	MAHASAAB	26029	67	M	180	100	20						1	1	135.69	HINDU	Yes	43	No	No		Absent
64	MAROVU CHAVAN	28902	65	M	150	90	12								252.18	HINDU	Yes	23	Yes	No		Absent
65	MAGRABI	29625	59	M	170	100	28					1	1	2	135.49	MUSLIM	No	26	No	No		Present
66	RAHMAT K	15265	47	M	140	96	16								163.60	MUSLIM	Yes	24	Yes	No		Present
67	MALLAPPA.KARBUR	4298	57	M	160	90	13								157.72	HINDU	No	24	No	No		Absent
68	SHANKAR.N	4256	74	M	170	90	50	3	3	3		1	1	11	196.90	HINDU	Yes	24	Yes	No		Absent
69	RAMZAN	34700	56	M	180	90	16				3		1	4	233.96	MUSLIM	Yes	32	Yes	No		Absent
70	RAMAPPA.GUTTA	41770	55	M	160	94	16				3			3	169.22	HINDU	Yes	22	No	No		Absent
71	TULGARAM	14271	40	M	150	100	30		3	2		1	1	7	167.78	HINDU	Yes	26	Yes	No		Absent
72	SAMADULLA	26420	46	M	200	100	38								147.37	MUSLIM	Yes	22	Yes	No		Absent
73	NAVEEN RATHOD	39598	56	M	140	90	24			2	3	1		6	166.94	HINDU	Yes	26	Yes	No		Absent

74	LAXMAN NAWAR	39864	75	M	140	90	35				3			3	143.95	HINDU	Yes	19	No	No		Present
75	MALLIKARJUN.K	19025	55	M	140	90	10								130.00	HINDU	Yes	23	Yes	No		Present
76	NINGANGOUDA	38751	64	M	140	94	23								168.98	HINDU	Yes	23	Yes	Yes		Present
77	JINNAH NADAF	15592	45	M	140	98	35	3						3	207.80	MUSLIM	Yes	25	Yes	No		Absent
78	SHIVAJI KUMKANTI	12572	58	M	150	94	30		3					3	147.52	HINDU	No	24	No	No		Absent
79	MD. ISAQ	267	77	M	160	100	28	1					1	2	154.03	MUSLIM	No	24	Yes	No		Absent
80	PANDURANGA.HOSA	228	58	M	200	110	30	3						3	154.34	HINDU	No	32	No	No		Absent
81	SHANTAVEER.B	434	82	M	180	100	31								180.90	HINDU	Yes	22	Yes	No		Absent
82	KRISHNARAJ.R	592	48	M	180	100	22								165.04	HINDU	No	24	Yes	No		Present
83	BASAVARAJ SONNAL	573	62	M	170	90	31	3						3	168.06	HINDU	Yes	26	Yes	No		Present
84	DHARMANGOUDA	490	72	M	180	96	26	3					1	4	195.91	HINDU	Yes	26	Yes	No		Present
85	MALLAGOUDA P	12723	59	M	190	90	28	2			1			3	187.09	HINDU	Yes	26	Yes	No		Present
86	OEGEPPA	1123	54	M	190	100	24						3	3	199.21	HINDU	Yes	26	Yes	No		Present
87	SHARANGOUDA.P	1234	78	M	160	96	26	3						3	204.13	HINDU	No	23	Yes	No		Absent
88	MALLANGOUDA	12568	55	M	200	100	30	3			1		1	5	291.48	HINDU	No	23	No	No		Absent
89	ISHVARAPPA	25125	48	M	160	112	28			1			1	2	189.66	HINDU	No	24	No	Yes		Absent
90	KRISHNAJI	10312	56	M	200	100	27	3						3	264.20	HINDU	No	24	No	Yes		Absent
91	CHANDRAKANTH.P	21558	56	M	200	108	32				3	1	1	5	234.06	HINDU	Yes	24	No	Yes		Present
92	KALLAPPA.ITTENGI	16756	68	M	160	90	19	3					1	4	198.77	HINDU	Yes	23	No	No		Absent
93	RAMAPPA BADIGERE	14625	49	M	192	94	30	3			3		1	7	221.40	HINDU	Yes	22	Yes	No		Present

94	SADANAND HIREMATH	14508	54	M	190	100	24	3						3	174.11	HINDU	Yes	22	No	No		Absent
95	SUDHAKAR VASTRAD	25628	57	M	192	104	34								198.45	HINDU	Yes	23	Yes	No		Present
96	SHATABAI BIRADAR	290532	43	F	148	90	46								120.00	HINDU	Yes	22	No	No	No	Absent
97	ANNAMMA PATIL	25126	45	F	160	100	23								113.20	HINDU	Yes	34	No	No	No	Present
98	PARVATHIBAI	1152	48	F	140	90	34			1		1		2	100.70	HINDU	No	22	No	No	No	Absent
99	CHAND BI	328	58	F	146	108	38	3		1		1		5	117.00	HINDU	No	43	No	No	No	Present
100	SHANTAMMA BHAIRI	230	48	F	148	110	19	3						3	116.00	HINDU	No	43	No	No	No	Present
101	SHANTA BIRADAR	19284	62	F	160	90	25	3		3			3	9	109.11	HINDU	Yes	22	No	No	No	Absent
102	RIYANA MEMON	271	75	F	158	106	32			3				3	111.78	HINDU	No	32	No	No	Oral	Absent
103	SHOBHA BIRADAR	22652	64	F	148	90	28	3			1		1	5	90.30	HINDU	Yes	33	No	No	No	Present
104	HANUMANTAMMA	14295	52	F	180	100	42	3						3	120.30	HINDU	No	44	No	No	No	Present
105	KASTRIBAI	18020	46	F	170	90	31	3			1			4	56.77	HINDU	No	44	No	No	No	Present
106	MALLAMMA BIRADAR	16371	58	F	140	106	32	3						3	113.40	HINDU	No	45	No	No	No	Absent
107	HANUMANTHAMMA	14295	63	F	148	90	31				1		1	2	103.66	HINDU	No	45	No	No	No	Absent
108	NIKHAT KHAN	27248	56	F	140	104	50	3			1		1	5	120.67	MUSLIM	No	25	No	No	No	Absent
109	LAXMIBAI PATIL	49235	49	F	160	102	18						3	3	100.78	HINDU	No	26	No	No	Oral	Absent
110	KOUSHALYA	14628	59	F	170	90	21			1	1	2		4	98.55	HINDU	No	25	No	No	Oral	Absent
111	HALEEMA BI	34025	45	F	160	110	22	3						3	88.74	MUSLIM	No	23	No	No	No	Absent
112	MUMTAZ BI	16629	49	F	148	108	15					1		1	84.99	MUSLIM	No	23	No	Yes	No	Present
113	NIMBAVVA HUGAR	13420	67	F	148	98	18	3				1	1	5	86.00	HINDU	No	23	No	No	No	Present

114	MALLAMMA BANDE	463	53	F	176	116	48	3				3		6	112.77	HINDU	No	23	No	No	No	Absent
115	SEETHA BAI	29241	76	F	140	90	28		1		1	2	1	4	89.73	HINDU	No	23	No	Yes	No	Absent
116	SAHEB BI	43451	57	F	140	104	24	3				3		6	114.23	MUSLIM	No	32	No	No	No	Absent
117	SUDHA PATIL	11292	74	F	140	96	30	3				1	1	5	112.98	HINDU	No	32	No	No	Oral	Absent
118	MANJULA DEVI	16372	49	F	142	106	25	3						3	114.00	HINDU	No	32	No	No	No	Absent
119	SHAILA UPPIN	18290	45	F	146	90	30			1	1	2		4	87.45	HINDU	Yes	32	No	No	No	Absent
120	SHARDA NAGUR	11292	63	F	140	90	48	3		1	1			5	98.00	HINDU	Yes	44	No	Yes	No	Present
121	GANGABAI	41770	58	F	150	104	21	3			2		1	6	78.66	HINDU	Yes	44	No	No	No	Present
122	PARVATAMMA	63451	47	F	140	114	22	3						3	119.36	HINDU	No	33	No	No	No	Present
123	RENUKA DODDAMANI	29241	52	F	146	96	26		2	1		3		6	119.00	HINDU	No	23	No	No	No	Present
124	DYAMMA BHAIRI	27248	56	F	154	106	23		2			3		5	117.45	HINDU	No	25	No	Yes	No	Present
125	SAVITRI PATIL	14867	47	F	158	98	31	3	1		1		1	6	116.79	HINDU	Yes	26	No	No	No	Present
126	DHANAMMA	13427	48	F	160	100	43	3						3	89.23	HINDU	No	23	No	Yes	No	Absent
127	INDUMATI	19284	54	F	160	92	32			1	1	2	1	5	115.00	HINDU	No	45	No	No	No	Absent
128	GIRIJA DEVI	24862	49	F	164	116	24	3						3	92.22	HINDU	No	32	No	No	No	Absent
129	BILQEES BANU	1264	65	F	170	90	36	3		1				4	86.36	MUSLIM	No	32	No	No	No	Absent
130	RAHMAT BEGUM	23117	68	F	168	118	26	3	1					4	116.83	MUSLIM	No	32	No	No	No	Absent

MASTER CHART FOR CONTROL GROUP

Sl. No.	Name	Sex	Age	SBP	DBP	ECG by Sokolowlyon Criteria	ECG by Romhilt Estes Criteria							LV Mass Index (gms/m²)	Religion	Smoker/ Non-smoker	BMI	Alcoholic/ non-alcoholic	Drug abuse	Contraception	Family History
							Voltage 3 pt	ST-T chanes 3 pt	Left axis deviation 2 points	Left atrial enlargement 3 points	QRS Duration > 0.09 sec 1 point	VAT 1 point	Total								
1	ARIF CHIGUPPA	M	44	112	68	12	1	1					2	109.13	MUSLIM	No	19.01	No	No		Absent
2	SANGAPPA MUDNAL	M	50	120	80	14	1		1		1		3	85.23	HINDU	No	15.00	No	No		Absent
3	MANTAPPA.NAMGO	M	60	124	76	10					1		1	78.33	HINDU	No	21.00	No	No		Absent
4	MALLAPPA UKKALI	M	56	110	70	9				1	1		2	112.34	HINDU	Yes	20.00	No	No		Absent
5	SANGAMESH.G	M	60	112	72	12				1			1	110.00	HINDU	No	19.00	Yes	No		Present
6	MALLAPPA UKKALI A PATIL	M	64	114	60	13				1			1	112.00	HINDU	Yes	16.00	No	Yes		Absent
7	SITARAM .W	M	48	126	68	7			1				1	112.00	HINDU	No	17.00	No	No		Absent
8	CHANDU.R	M	54	130	70	8			1				1	115.40	HINDU	Yes	18.00	Yes	No		Absent
9	SHIVAPPA.H	M	66	112	60	9					1		1	113.50	HINDU	No	15.00	No	No		Absent
10	DUNDAPPA.A	M	54	110	68	10	1			1			2	110.23	HINDU	No	21.34	No	No		Present
11	LAXMAN UKKALI	M	58	130	70	11	1	1					2	111.14	HINDU	No	21.00	Yes	Yes		Absent
12	SOMANGOUDA	M	47	110	70	12		1					1	120.21	HINDU	Yes	18.67	No	No		Absent
13	MALLAPPA PUJARI	M	55	112	68	13	1					1	2	112.00	HINDU	No	22.02	No	No		Present

14	SUBHASH METRI	M	45	114	64	14						1	1	112.13	HINDU	No	17.11	No	No		Absent
15	BADRINATH.P	M	49	110	60	15	1		1		1		3	110.00	HINDU	No	18.00	No	Yes		Absent
16	SHIVAJI.K	M	52	110	70	14	1						1	111.65	HINDU	No	13.00	Yes	No		Absent
17	JINNAH SAB	M	49	110	70	11		1					1	120.56	MUSLIM	Yes	18.88	No	No		Absent
18	GUNDAPPA.H	M	80	120	80	10		1	1				2	112.78	HINDU	No	18.59	No	No		Absent
19	PRABHUGOUDA.P	M	67	124	80	10			1				1	111.45	HINDU	No	15.00	Yes	No		Absent
20	ABDULLAH	M	45	122	80	11	1		1				2	112.78	MUSLIM	No	17.56	No	No		Absent
21	SANJEEVE.K	M	56	124	78	12	1						1	115.45	HINDU	Yes	17.44	Yes	Yes		Absent
22	GUNDDAPPA.P	M	58	122	78	19				1			1	114.56	HINDU	No	16.44	Yes	No		Absent
23	SANGAPPA.H	M	48	122	68	12			1				1	112.58	HINDU	Yes	15.00	No	No		Absent
24	NINGANGOUDA	M	68	122	68	11							0	115.89	HINDU	No	18.00	No	No		Absent
25	PANDURANGA	M	54	112	78	10							0	114.43	HINDU	No	17.00	Yes	No		Absent
26	SHRISHAILAPPA.D	M	48	112	68	8							0	106.80	HINDU	No	18.00	No	No		Absent
27	GURANNAGOUDA	M	65	112	64	7							0	114.24	HINDU	Yes	17.22	Yes	No		Present
28	GUNDAPPA TELI	M	47	114	64	9			1				1	112.13	HINDU	No	17.00	No	Yes		Absent
29	GUNDAPPA MALLAD	M	55	114	62	9				1			1	117.89	HINDU	No	18.00	No	No		Absent
30	RUDRAGOUDA PATI	M	55	114	60	8							0	118.20	HINDU	Yes	16.44	No	No		Absent
31	VITHAL RAO CHAVA	M	69	110	60	9					1		1	112.55	HINDU	No	15.22	Yes	No		Absent
32	SHABBIR AHMED	M	84	110	60	8							0	113.65	MUSLIM	No	16.00	Yes	No		Absent
33	SHAFIQ KHAN	M	68	110	62	6						1	1	113.68	MUSLIM	Yes	19.00	Yes	No		Absent

34	DUNDAPPA ARVAT	M	56	110	62	7							0	114.68	HINDU	No	21.00	No	No		Absent
35	SATISH AMTE	M	58	120	70	8							0	114.33	HINDU	No	19.00	No	No		Absent
36	GAGAPPA TALIKOTI	M	65	120	70	11			1				1	112.33	HINDU	No	20.44	No	No		Absent
37	GYASUDDIN	M	56	120	70	12							0	112.87	MUSLIM	No	18.99	No	Yes		Absent
38	ANIL NAVI	M	45	120	68	13		1					1	113.56	HINDU	No	20.00	Yes	No		Absent
39	MD. QASIMSAB	M	50	124	68	14	1						1	112.12	MUSLIM	No	21.00	No	No		Absent
40	MD.SAMAD	M	58	122	70	15							0	112.22	MUSLIM	No	19.00	No	No		Absent
41	PRABHUGOUDA.P	M	45	124	72	16							0	113.24	HINDU	No	23.55	Yes	No		Absent
42	REVU RATHOD	M	55	124	74	9							0	113.50	HINDU	No	24.00	No	No		Absent
43	BASAVARAJ PATIL	M	55	122	74	8							0	117.00	HINDU	No	21.55	No	No		Absent
44	IBRAHIM SAB	M	64	124	72	7	1						1	112.61	MUSLIM	No	24.66	Yes	No		Absent
45	CHANDRAKANTH BHI	M	53	112	68	6		1					1	120.00	HINDU	No	19.89	No	No		Absent
46	NINGAPPA PUJARI	M	63	114	64	9							0	112.71	HINDU	No	16.77	No	No		Absent
47	SIDDANGOUDA.P	M	50	118	60	10							0	110.33	HINDU	No	22.56	Yes	Yes		Absent
48	RAGHAVENDRA.K	M	55	116	78	1213							0	112.22	HINDU	No	20.23	No	No		Absent
49	NARAYAN RAO	M	48	112	60	13		1					1	112.34	HINDU	No	23.00	Yes	No		Absent
50	ALLAHBAKSH	M	52	114	60	12	1						1	112.43	MUSLIM	No	24.00	No	Yes		Absent
51	SIDDAPPA KUMBAR	M	62	116	70	11							0	110.88	HINDU	No	16.44	No	No		Absent
52	IMAMSAAB	M	66	110	70	10							0	110.45	MUSLIM	No	24.45	Yes	No		Absent
53	BASAPPA.D	M	48	110	72	9				1			1	110.00	HINDU	No	18.44	No	No		Absent

54	SUBHASH GANNUR	M	56	110	74				1				1	112.00	HINDU	No	16.89	Yes	No		Absent
55	BASAPPA.K	M	58	124	72	6							0	120.00	HINDU	No	23.00	No	No		Absent
56	RAMAPPA TULJAPU	M	63	124	72	7							0	113.55	HINDU	No	14.00	Yes	Yes		Absent
57	SIDDRAM GUTTA	M	59	122	70	8					1		1	116.31	HINDU	No	23.22	No	Yes		Absent
58	BASAVARAJ BIRADAR		54	124	70	9							0	117.56	HINDU	No	22.00	No	No		Absent
59	KATTEPPA.P	M	61	122	70	11		1					1	118.45	HINDU	No	14.33	No	No		Absent
60	SIDDANNA PULKE	M	66	122	70	10							0	116.25	HINDU	No	22.00	No	No		Absent
61	MAHASAAB	M	86	126	72	12	1						1	117.56	HINDU	No	19.43	Yes	No		Absent
62	MAROVU CHAVAN	M	59	126	72	11							0	114.44	HINDU	No	17.44	No	No		Absent
63	MAGRABI	M	67	118	60	12			1				1	113.76	MUSLIM	No	17.00	No	No		Absent
64	RAHMAT K	M	65	118	60	10							0	114.68	MUSLIM	No	18.00	No	No		Absent
65	MALLAPPA.KARBUR	M	59	118	62	10		1					1	118.00	HINDU	No	21.00	Yes	No		Absent
66	CHANDRAKANTH BHI	M	47	112	62	9				1			1	117.33	HINDU	No	21.66	No	No		Absent
67	NINGAPPA PUJARI	M	57	120	68	8							0	116.98	HINDU	No	21.44	Yes	No		Absent
68	SIDDANGOUDA.P	M	74	120	62	9							0	113.98	HINDU	No	22.56	No	No		Absent
69	RAGHAVENDRA.K	M	56	110	60	8							0	114.00	HINDU	No	21.45	No	No		Absent
70	NARAYAN RAO	M	55	110	60	12							0	116.00	HINDU	No	22.12	Yes	Yes		Absent
71	ALLAHBAKSH	M	40	110	60	9							0	114.00	MUSLIM	No	21.33	No	No		Absent
72	SIDDAPPA KUMBAR	M	46	112	64	11	1			1			2	112.78	HINDU	No	15.45	No	No		Absent
73	IMAMSAAB	M	56	114	66	13				1			1	13.45	MUSLIM	No	18.00	Yes	Yes		Absent

74	BASAPPA.D	M	75	116	66	15							0	115.68	HINDU	No	17.00	No	No		Absent
75	SUBHASH GANNUR	M	55	118	60	12			1				1	114.34	HINDU	No	15.00	Yes	No		Absent
76	BASAPPA.K	M	64	112	60	22							0	115.00	HINDU	No	19.00	No	No		Absent
77	ANNAMMA PATIL	45	F	110	64	21							0	64.33	HINDU	No	21.22			No	Absent
78	PARVATHIBAI	48	F	110	62	18							1	78.44	HINDU	No	22.45			No	Absent
79	CHAND BI	58	F	110	60	20							1	78.55	MUSLIM	No	26.44		Yes	No	Absent
80	SHANTAMMA BHAIRI	48	F	114	72	21							0	86.33	HINDU	No	26.00			No	Absent
81	SHANTA BIRADAR	62	F	116	70	20							1	89.34	HINDU	No	24.00			No	Absent
82	RIYANA MEMON	75	F	118	70	21							0	68.00	MUSLIM	No	27.00			No	Absent
83	SHOBHA BIRADAR	64	F	120	80	22							1	78.23	HINDU	No	25.00			No	Absent
84	HANUMANTAMMA	52	F	130	80	22							0	73.00	HINDU	No	28.00			No	Absent
85	KASTRIBAI	46	F	116	70	20							0	65.33	HINDU	No	19.00			No	Absent
86	MALLAMMA BIRADAR	58	F	110	64	20							0	56.98	HINDU	No	18.99			No	Absent
87	HANUMANTHAMMA	63	F	112	68	20							0	56.90	HINDU	No	21.56		Yes	No	Absent
88	NIKHAT KHAN	56	F	114	60	22							0	65.78	MUSLIM	No	23.55			No	Present
89	LAXMIBAI PATIL	49	F	110	70	10							0	78.56	HINDU	No	24.00			No	Absent
90	KOUSHALYA	59	F	110	80	10							0	88.23	HINDU	No	25.44			No	Absent
91	HALEEMA BI	45	F	130	60	9							0	73.56	MUSLIM	No	23.66			No	Absent
92	MUMTAZ BI	49	F	130	68	8							0	76.45	MUSLIM	No	26.88			No	Absent
93	NIMBAVVA HUGAR	67	F	118	64	8							1	89.67	HINDU	No	30.00			No	Absent

94	MALLAMMA BANDE	53	F	112	64	9							1	68.46	HINDU	No	22.40			No	Absent
95	SEETHA BAI	76	F	114	62	17							1	69.37	HINDU	No	2.45			No	Absent
96	SAHEB BI	57	F	118	68	17							1	72.00	MUSLIM	No	25.00			No	Absent
97	SUDHA PATIL	74	F	118	68	16							1	68.00	HINDU	No	26.00			No	Absent
98	MANJULA DEVI	49	F	118	70	14							1	73.00	HINDU	No	23.77		Yes	No	Absent
99	SHAILA UPPIN	45	F	118	68	15							1	80.55	HINDU	No	30.00		Yes	No	Absent
100	SHARDA NAGUR	63	F	118	64	14							1	56.78	HINDU	No	22.00			No	Absent
101	GANGABAI	58	F	116	62	13							0	86.00	HINDU	No	24.00			No	Present
102	PARVATAMMA	47	F	110	64	18							0	68.77	HINDU	No	22.44			No	Absent
103	RENUKA DODDAMANI	52	F	110	60	22							0	21.34	HINDU	No				No	Absent
104	DYAMMA BHAIRI	56	F	120	70	23							0	22.12	HINDU	No				No	Absent
105	SAVITRI PATIL	47	F	130	70	24							0	23.12	HINDU	No				No	Absent
106	DHANAMMA	48	F		64	25							0	19.22	HINDU	No				No	Absent
107	INDUMATI	54	F		62	22							0	19.78	HINDU	No			Yes	No	Absent
108	RAMAPPA TULJAPU	49	F		68	22							0	23.44	HINDU	No				No	Absent
109	SIDDRAM GUTTA	65	F		70	21							0	34.22	HINDU	No				No	Absent