"CLINICAL AND ECHOCARDIOGRAPHIC STUDY OF ELDERLY HYPERTENSIVE PATIENTS"

By

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Dissertation submitted to

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In partial fulfillment of the requirements for the degree of

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In

General Medicine

Under the guidance of

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2013

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LIST OF ABBREVIATIONS

AHA- American Heart Association

BMI- Body Mass Index

BP- Blood Pressure

CAD-Coronary Artery Disease

CHD- Coronary Heart Disease

CHF- Congestive Heart Failure

CKD- Chronic Kidney Disease

CV- Cardio Vascular

DBP- Diastolic Blood Pressure

ECG- Electrocardiography

ECHO- Echocardiography

eGFR- estimated Glomerular Filtration Rate

HDL- High Density Lipoprotein

IHD- Ischemic Heart Disease

ISH- Isolated Systolic hypertension

JNC- Joint National Committee

LBBB- Left Bundle Branch Block

LDL- Low Density Lipoprotein

LV- Left Ventricle

LVDD- Left Ventricular Diastolic Dysfunction

LVH- Left Ventricular Failure

MI- Myocardial Infarction

PP- Pulse Pressure

RBBB- Right Bundle Branch Block

RWMA- Regional Wall Motion Abnormality

SBP- Systolic Blood Pressure

SHEP- Systolic Hypertension In Elderly

TIA- Transient Ischemic Attack

ABSTRACT

INTRODUCTION: Isolated systolic hypertension(ISH) is the commonest cause of raised blood pressure in the older population. As the age progresses more and more persons will be hypertensive. It is a disease, which is definitely the most prevalent, remediable risk factor for cardiovascular diseases and cerebrovascular diseases. Hence the present study is undertaken to study the clinical profile of hypertension in elderly, to find out any associated risk factors, especially on cardiac through echocardiography.

METHODS: A total number of 60 elderly hypertensive patients (i.e. ≥ 60 years) with systolic blood pressure ≥ 140 and diastolic blood pressure ≤ 90 mmHg were included in this study. A detailed history with general and systemic examination was carried out. Complete blood count, urine analysis, blood sugar, renal profile, lipid profile, chest x-ray, electrocardiogram, and echocardiogram and fundus examination were done in all patients

RESULTS: Sixty patients were evaluated, and the mean age was 67.72±7.004 years of which 35 (58.33%) were males and 25(41.67%) were females and the common age group was 60-65 years. Chest pain (45%) and breathlessness (38.33%) were commonest chief complaint. Family history, smoking history and total cholesterol were associated risk factors of hypertension. Hypertensive retinopathy was observed in 38.33% patients. Ischemic changes (31.67%) followed by left ventricular (LV) hypertrophy (20%) were most common electrocardiographic (ECG) findings. The echocardiographic evaluation showed LV dysfunction (63.33%) was common entity in the form of LV hypertrophy (30%), LV dilatation (15%) and LV diastolic dysfunction (48.33%). Ischemic heart disease was diagnosed in 55% patients. Out of

these 18 (54.55%) had anterior, 4 (12.12%) lateral, 3 (9.09%) inferior, and 4 (12.12%) septal infarcts. 1 patient had global hypokinesia, and 3 patients had more than one segment involvement. There was strong co relation between LV hypertrophy, Ischemia and SBP.

CONCLUSION: The elderly hypertensive patients tend to have ISH. Smoking history, family history and dyslipidemia are important determinants of hypertension. LVH and other associated risk factors are responsible for coronary artery disease.

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INTRODUCTION

Ageing is a natural process. As age progresses the blood pressure also increases gradually. Every 3 of 4 adults will be hypertensive by the age of 50 years[01].Epidemological studies suggest that by the age of 60 years 55% of the whole population will be hypertensive and 65% over age 0f 70 years. Out of these 65% of them are suffering from isolated systolic hypertension[02].

The biology of ageing includes age related decline in individual's capacity in various metabolic activities. Morbidity occurs due to the changes occurring during the ageing process either due to intrinsic and extrinsic or self-induced factors in the different sphere of life. Majority of the events are progressed rapidly due to inherited lifestyle by the individuals[03]. With the increase in life expectancy and modification of lifestyle, cardiovascular disease primarily hypertension is emerging as a major health problem in elderly population

AIMS AND OBJECTIVES

To study the clinical profile of hypertension in elderly (above the age of 60 years), to find out any other associated risk factors, end organ complications, especially on cardiac through echocardiography.

REVIEW OF LITERATURE

DEFINITION

Isolated systolic hypertension (ISH) is defined as elevated systolic blood pressure (SBP) $\geq$ 140 mm Hg in conjunction with normal diastolic blood pressure (DBP) $\leq$ 90 mm Hg[04].

Earlier in 1990's hypertension was defined using only the criteria of elevated DBP[01]. Until then, there was no prospective clinical trial data using SBP entry criteria to define whether treatment of hypertension based on SBP level was beneficial or not. Various epidemiological studies now have consistently demonstrated that SBP is a strong predictor of risk than DBP[05] and increasing levels of SBP correlate directly with risk of developing cardiovascular events and mortality in elderly[01].

The importance of isolated systolic hypertension was first mentioned in 1993 in report by joint national committee (JNC) V[06]. Earlier it was defined as SBP between 140 to 160 and DBP < 90 as borderline ISH and SBP more than 160mmHg and DBP <90 mmHg as ISH. This definition of ISH was changed in JNCV report by as any SBP above 140 mmHg together with DBP below 90mmHg[07].

GUIDELINES FOR HYPERTENSION

Based upon the various epidemiological and observational datas, different societies and committees have classified blood pressure for adults. The most widely accepted and implemented guideline is JNC VII guidelines[04]

JNC – VII GUIDELINES

(Joint National Committee – Seventh report 2003) Category	Systolic BP mm Hg	Diastolic BP mm Hg
Normal	<120	<80
Pre Hypertension	120-139	80-89
Stage 1 Hypertension	140-159	90-99
Stage 2 Hypertension	≥160	≥100
Isolated Systolic Hypertension(ISH)	>140	<90

EPIDEMOLOGY

An age associated rise in SBP was earlier considered as a normal process. Many physicians used to consider 100+age in years as a proper SBP. Indeed, hypertension was earlier treated only on criterion of elevated DBP until the fifth report of joint national committee on detection and evaluation and treatment of high blood pressure in 1993(JNC V)[08].

A number of studies, including the Framingham study, demonstrate that SBP rises gradually till the age of 80 years and then tends to be plateau thereafter[09]. In contrast, DBP initially rises along with SBP till the age of 50 to 55 years and then gradually decreases after age 60 to 65 years. Hence the pulse pressure defined by the difference between peak SBP and end DBP becomes important predictor of the risk factor in elderly.

After the 6th decade of life, increasing PP and decreasing DBP represents the central artery stiffening. Indeed fall in SBP is due to peripheral run off of stroke volume

through stiffened artery and fall in DBP is due to decrease amount of blood in the aorta at the beginning of diastole, with diminished recoil[08].

Framingham study showed that untreated hypertension have accelerated stiffening of elastic arteries leading to decrease in DBP and increase in PP as compared with normotensive group[10].

PREVALENCE

ISH is strongly age dependent. The Framingham study and the NHANES III (National Health and Nutrition Examination Survey) study showed that SBP progressively increases throughout adult life in untreated individuals[08]. It is estimated that in America, a person with history of hypertension, there is 69% risk of myocardial infarction (MI), 77% risk of stroke and 74% risk of heart failure (HF) [11]. It is also estimated that 90% of normotensive participants at the age of 55 years will develop hypertension eventually[11]. Data from systolic hypertension in elderly program(SHEP) [12]revealed that 8% of those aged 60-70 years, 11% aged 70-79 years, and 22% of those over 80 years had ISH. When compared with older studies like SHEP and SYST-EUR trial, Framingham study reported higher frequency of ISH of 35-40% in age group of 50-59 years and65-70% in age group of more than 60 years[05].

Franklin et al[05] analysis based on NHANES III, concluded that ISH was commonest subtype of hypertension in adults,54% had ISH between 50-59 years, 87% had ISH after 6th decade of life. Stage I SBP was more common (75%) when compared with stage II (25%) and ISH was predominant in females (58%).

Maddens et al[13] reported that among untreated hypertensive patients aged 60 years and older, 76% of men with stage I isolated systolic hypertension will progress to

stage II or higher, as will 47% of men whose SBP is less than 140 mm Hg at baseline. Among women, the numbers are 80% and 59%, respectively.

According to Indian hypertension guidelines[14] studies shows that there are 34 million hypertensives in urban and 31.5 million in rural population accounting for 25% urban and 10% rural subjects in India. A total of 70% of these would be Stage I hypertension (systolic BP 140-159 and/or diastolic BP 90-99 mmHg).

According to Gupta et al[15], a review on "the global burden of hypertension", the prevalence of hypertension in India in 2000 was 20.6% among males and 20.9% among females and is estimated to increase to 22.9% and 23.6% respectively in 2025.

NC Hazarika et al[03]showed 11.94% had ISH in elderly hypertensive population of Assam. Vrinda et al[16]showed ISH in 56.6% of the elderly hypertensive patients studied. Gupta et al review article on trends in hypertension prevalence in India showed more than 50% had ISH

PATHOPHYSIOLOGY[01, 09]

Pulse pressure is determined not only by arterial stiffness, but also by early pulse wave reflection, heart rate, and left ventricular ejection. In contrast mean arterial pressure is determined by cardiac output and total peripheral vascular resistance.

The main cardiovascular pathophysiologic changes associated with aging are decrease in large arterial compliance along with arterial dilation, because of loss of elastin fibers and proliferation of collagen and deposition of calcium. This occurs mainly in aorta and its main branches.

The central wave form produced by ventricular ejection travels faster due to stiffening of large arteries and the reflected pressure wave returns to the ascending aorta earlier

during late systole rather than in diastole due to peripheral vascular resistance. This results in augmentation of central SBP, and fall in DBP and hence increase pulse pressure. The increase in systolic blood pressure puts excess mechanical strain on left ventricle, leading to concentric left ventricular remodeling. This results in increase myocardial demand and decrease coronary perfusion and results in atherosclerosis due to intimal damage and an increased likely hood of thrombosis.

In addition to arterial stiffening the left ventricle also stiffens due to left ventricular hypertrophy and there is increase in late systolic overload leading to increased work load on cardiac and impaired diastolic relaxation. The loss arterial distensibility leads to decrease in release of nitric oxide leading to compromised vasoprotection and decrease in baroreceptor sensitivity due to old age, both leads to increase in blood pressure. Combining all above changes there is increased atherosclerosis and rupture of unstable plaque, leading to acute coronary syndrome.

CLINICAL APPROACH AND DIAGNOSIS

Diagnostic of hypertension should aim at: [17]

1) Establishing blood pressure levels;
2) Identifying secondary causes of hypertension;
3) Evaluating the overall cardiovascular risk by searching for other risk factors, target organ damage and concomitant diseases or accompanying clinical conditions.

The diagnostic procedures comprise:

– Medical history

– Physical examination

– Repeated blood pressure measurements

– Laboratory and instrumental investigations

Symptoms and Signs

Most patients with hypertension are asymptomatic. Although, most commonly headache occurs in patients with severe hypertension. Characteristically, a "hypertensive headache" occurs in the morning and is localized to the occipital region. Other nonspecific symptoms that may be related to elevated blood pressure include dizziness, palpitations, easy fatigability, and impotence. When symptoms are present, they are generally due to hypertensive cardiovascular disease or due to manifestations of secondary hypertension.[18]

Symptoms caused by secondary hypertension include

(a) Headaches, pallor, and diaphoresis (pheochromocytoma);

(b) Weight loss, tremor, anxiety, tachycardia, fatigue, weakness (hyperthyroidism);

(c) Confusion, anorexia, weakness, myalgia, constipation, depression (hypothyroidism);

(d) Urinary frequency, nocturia, hematuria, fatigue (renal parenchymal disease);and

(e) Muscle cramps, muscle weakness, nocturia, hypokalemia (primary aldosteronism).

History

The purpose of the history is to determine potential symptoms associated with or suggesting causes of hypertension, evaluate the presence of target organ damage, determine other cardiovascular risk factors, assess concomitant diseases that might interfere with the treatment of hypertension, seek clues suggestive of secondary hypertension, catalogue all medications used, assess resources, assess mental status, and determine general ability to implement the activities of daily living.

Physical Examination

In addition to blood pressure, heart rate should be carefully measured because the repeated finding of values above normal may be an indication of greater risk, increased sympathetic or decreased parasympathetic activity, or of heart failure. Physical examination should search for evidence of additional risk factors, for signs suggesting secondary hypertension, and for evidence of target organ damage. Waist circumference should be measured with the patient standing and body weight and height should be obtained to calculate body mass index by a standard formula.

Physical examination for secondary hypertension, target organ damage and visceral obesity[17]

Signs suggesting secondary hypertension and target organ damage

- Features of Cushing syndrome
- Skin stigmata of neurofibromatosis (phaeochromocytoma)
- Palpation of enlarged kidneys (polycystic kidney)
- Auscultation of abdominal murmurs (renovascular hypertension)
- Auscultation of precordial or chest murmurs (aortic coarctation or aortic disease)
- Diminished and delayed femoral pulses and reduced femoral BP (aortic coarctation, aortic disease)

Signs of target organ damage[17]

- Brain: murmurs over neck arteries, motor or sensory defects
- Retina: fundoscopic abnormalities

- Heart: location and characteristics of apical impulse, abnormal cardiac rhythms, ventricular gallop, pulmonary rales, peripheral oedema
- Peripheral arteries: absence, reduction, or asymmetry of pulses, cold extremities, ischaemic skin lesions
- Carotid arteries: systolic murmurs

Evidence of visceral obesity

- Body weight
- Increased waist circumference (standing position) M:>102 cm; F:> 88 cm
- Increased body mass index [body weight (kg)/height $(m)^2$]
- Overweight $\geq$ 25 kg/m^2; Obesity $\geq$ 30 kg/m^2

Laboratory investigations[17]

Laboratory investigations are directed at providing evidence for additional risk factors, searching for secondary hypertension and looking for the absence or presence of target organ damage. Investigations should progress from the most simple to the more complicated.

Routine tests

- Fasting plasma glucose
- Serum total cholesterol
- Serum LDL-cholesterol
- Serum HDL-cholesterol

- Fasting serum triglycerides
- Serum potassium
- Serum uric acid
- Serum creatinine
- Estimated creatinine clearance (Cockroft-Gault formula) or glomerular filtration rate (modification of diet in renal disease-MDRD formula)
- Haemoglobin and haematocrit
- Urinalysis (complemented by microalbuminuria via dipstick test and microscopic examination)
- Electrocardiogram

Recommended tests

- Echocardiogram
- Carotid ultrasound
- Quantitative proteinuria (if dipstick test positive)
- Ankle-brachial BP Index
- Fundoscopy
- Glucose tolerance test [if fasting plasma glucose >5.6 mmol/L (100 mg/dL)]
- Home and 24 h ambulatory BP monitoring
- Pulse wave velocity measurement

Extended evaluation

- Further search for cerebral, cardiac, renal and vascular damage. Mandatory in complicated hypertension

- Search for secondary hypertension when suggested by history, physical examination or routine tests: measurement of renin, aldosterone, corticosteroids, catecholamine's in plasma and/or urine; arteriographies; renal and adrenal ultrasound; computer-assisted tomography; magnetic resonance imaging.

CLASSIFICATION OF HYPERTENSION[18]

Primary (Essential) ---80-95%

Secondary ---5-20%

1. Renal

a) Chronic pyelonephritis

b) Acute and chronic glomerulonephritis

c) Polycystic renal disease.

d) Reno vascular stenosis or renal infarction.

f) Renin producing tumors

2. Endocrine

a) Oral contraceptives

b) Cushing's disease and syndrome

c) Primary hyperaldosteronism

d) Congenital or hereditary adrenogenital syndromes

e) Pheochromocytoma

f) Myxedema

g) Acromegaly

3. Neurogenic

a) Psychogenic

b) Diencephalic syndrome

c) Familial dysautonomia

d) Polyneuritis

e) Increased intracranial pressure

f) Acute spinal cord section

4. Miscellaneous

a) Coarctation of aorta

b) Increased intravascular volume

c) Poly arteritis nodosa

d) Hypercalcemia

e) Medications, e.g., glucocorticoids, cyclosporine

f) Toxemia of pregnancy

g) Acute intermittent porphyria.

Causes of Isolated Systolic Hypertension[18]

1. Decreased vascular compliance

2. Increased cardiac output.

a. Aortic regurgitation

b. Thyrotoxicosis.

c. Hyperkinetic heart syndrome.

d. Fever.

e. Arteriovenous fistula

f. Patent ductusarteriosus

Factors influencing risk

Before initiating therapy, patients' overall risk should be assessed considering the presence or absence of additional risk factors; extent of target organ damage and other associated clinical conditions.

Risk factors[17]

- Age > 55 years
- Male sex
- Post-menopausal women
- Smoking and tobacco use
- Diabetes mellitus

- Family history of premature CAD (Males < 55 years ,Female < 65 years)
- Increased Waist hip ratio
- High LDL or total cholesterol, Low HDL cholesterol and High triglycerides
- High sensitivity C-reactive protein (HS-CRP) and homocysteine levels might evolve as markers for high risk of vascular damage
- Estimated GFR <60 mL/min

Target organ damage (TOD) [17]

- Left ventricular hypertrophy detected by ECG and/or echocardiogram
- Microalbuminuria / proteinuria and/or elevation of serum creatinine (1.2-2.0 mg/dl)
- Urinary ACR (albumin creatinine ratio)
- Ultrasound or radiological evidence of atherosclerotic plaques in the carotids
- Hypertensive retinopathy

Associated clinical conditions

- Cerebrovascular disease
    - Ischemic stroke
    - Cerebral hemorrhage
    - Transient ischemic attack
- Heart disease
    - Myocardial infarction
    - Angina
    - Coronary revascularization
    - Congestive heart failure

- Renal disease
    - Diabetic nephropathy
    - Renal failure (serum creatinine > 2.0 mg/dl)
- Vascular disease
    - Peripheral arterial disease including non-specific aortoarteritis
    - Aortic dissection
- Retinal disease
    - Advanced hypertensive retinopathy
    - Hemorrhages or exudates
    - Papilledema

RISK FACTORS

Age, race, sex, smoking, alcohol intake, serum cholesterol, glucose intolerance, and weight all may alter the prognosis of hypertensive vascular disease. The younger the patient when hypertension is first noticed, the greater is the reduction in life expectancy if the hypertension is left untreated.

In the United States, urban blacks have about twice the prevalence of hypertension as whites and more than four times the hypertension induced morbidity rate. At all ages and in both white and nonwhite populations, females with hypertension far better than males up to the age of 65, and the prevalence of hypertension in premenopausal females is substantially less than that in age-matched males or postmenopausal women. Yet compared with their normotensive counterparts, females with hypertension run the same relative risk of a morbid cardiovascular event as do males

Obesity

Obesity is associated with an elevation in blood pressure. For each unit of increase in BMI, there is 1.2mmHg rise in SBP and 0.7mmHg rise in DBP. The risk of obesity-related hypertension declines with age, there being a threefold increase in

hypertension in middle aged obese (20- to 45 years) compared with a 1.5 increase in elderly obese (> 65year). Interestingly, as BMI increase, CV relative risk increases from 1.8 to 2.9for elderly hypertensive men, whereas the reverse is true for women. According to the European Working Party on Hypertension in the Elderly (EWPHE) study, there was decrease in cardiovascular mortality in study group with a BMI of 26 to 27 and there was decrease in total mortality and CV events in the moderately obese group with a BMI of 28 to 29 kg/m^2.Truncal obesity (reflected in an increased waist to hip ratio) is more strongly related to hypertension and is a better predictor for coronary heart disease and stroke than BMI alone.[09]

Barry R. Davis et al., [19] showed that BMI had a significant quadratic relationship with hemorrhagic stroke those with low and high BMI had an increased risk. NC-Hazarika et al.,[03] has observed increased body mass index to be associated with increased prevalence of ISH and its complications. High prevalence of hypertension in elderly patients having higher body mass index (BMI) associated with family history has been reported in a recent study in Taiwan city[20].

Smoking

Cigarette smoking raises blood pressure, probably through the nicotine-induced release of norepinephrine from adrenergic nerve endings. In addition, smoking causes an acute and marked reduction in radial artery compliance independent of the increase in blood pressure. The relative risk of stroke among older hypertensive smokers is five times that of normotensives but 20 times that of normotensive nonsmokers.[09]Vrinda et al., [16]observed that smoking and tobacco chewing as important determinants of hypertension in elderly. Barry R. Davis et al., [19] observed diabetes and smoking were significantly related to lacunar stroke risk.

Alcohol

Alcohol even in small quantity may raise blood pressure there is a linear progressively increasing level of blood pressure with increase in consumption of alcohol. Multiple studies show a clear association between excess alcohol intake and the development of hypertension. Patients who have more than two drinks per day have a 1.5 to 2 fold increase in the incidence of hypertension compared to nondrinkers; this effect is dose-related and is most prominent when intake exceeds five drinks per day appears to have a cardioprotective effect, even in patients with preexisting hypertension.

A meta-analysis of 15 randomized trials reported that decreased alcohol consumption lowered systolic and diastolic BP by 3.3 and 2.0 mm Hg, respectively.[08]

N C Hazarika et al., [03] found a direct association between alcohol consumption and increase in blood pressure in rural population of Assam. Barry R .Davis et al., [19] found that for atherosclerotic strokes, there appeared to be an association with moderate alcohol intake.

Diabetes

Co-existence of hypertension and diabetes is about increasingly 30-35%. The prevalence of hypertension is 1.5 to 2 times greater in patients with diabetes mellitus compared with matched non-diabetic individuals.[14] Diabetes doubles the risk of developing coronary heart disease and stroke in those aged 65 to 94 years. Like total cholesterol, however, its impact on CV events falls with age: women remain slightly more at risk than men, though the absolute risk from diabetes is greater in the elderly than the young[09].

Vrinda et al.[16], observed the positive association of cardiovascular complications in elderly diabetic hypertensives.

In the SHEP trial[12]the percent reduction in events related to coronary heart disease was greater in the treated diabetic patients, than in treated non-diabetic patients.

UKPDS (United Kingdom Prospective Diabetes Study) trial reported randomized tight control of blood pressure vs. less tight control of blood pressure over a median of 8.4 years, reduction in risk were 24%. In diabetes related end points, 32% in death related to diabetes, 44% in strokes and 37% in microvascular end points. HOT (hypertension optimal treatment) study observed that, cardiovascular events to be significantly lowered in diabetic patients, whose hypertension was aggressively treated.

Dyslipidemia

According to Multiple Risk Factor Intervention Trial, higher cholesterol level will increase the risk of coronary heart disease (CHD). When compared with cholesterol of 200,250 and 300 mg/dl, there was 2 and 3 fold increase in risk of CHD in this trial. Lower level of HDL is also related with increase in risk of CHD. In a 12 year follow up of Framingham study incidence of MI was directly related with increase in cholesterol levels and inversely related to low HDL levels in women. Similar results were obtained for men in Copenhagen male study[08].

Vrinda et al [16] observed cardiovascular complication in 19.1% patients having dyslipidemia and hypertension.

EFFECTS OF HYPERTENSION

Adverse effects of hypertension principally involve the major organ systems like the cardiovascular system, central nervous system, retina and the kidneys; also called "TARGET ORGAN DAMAGE".

CARDIOVASCULAR SYSTEM

Hypertension induced complications of cardiovascular system are:

- Left ventricular hypertrophy as a result of pressure overload.
- Left ventricular failure.
- Coronary artery disease.
- Aortic aneurysm.
- Aortic dissection.

Left Ventricular Hypertrophy (LVH)

The high prevalence of Isolated Systolic Hypertension (ISH) in the elderly represents a major public health issue. Hypertension is usually characterized by concentric hypertrophy of left ventricle `with circumferential hypertrophy of myofibrils, normal or increased contractility, increased relative wall thickness, normal or low end-diastolic volumes, and at times impaired relaxation (diastolic dysfunction). In population-based samples, 30% to 50% of individuals with stage 1 and 2 hypertension have impaired left ventricular (LV) relaxation, and in more severe forms of hypertension, about two-thirds have abnormal LV relaxation. In untreated or poorly treated individuals, LVH becomes a major risk factor for dilated cardiomyopathy and HF.[04]

During four years of follow up in Framingham heart study each 50g/m^2 increase in LV mass was associated with 1.49 increase in relative risk of cardiovascular disease for men and 1.57 increase for women. The effect on cardiovascular mortality was even more striking with a 1.73 relative risk for each 50 g/m2 for men and each 2.12 g/m^2 for women[22].

The long term follow up of 10 years also predicted that LV mass was better predictor of cardiovascular morbidity and mortality than SBP, smoking or cholesterol levels[21].

According to Framingham heart study, echocardiography derived LVH to height ratio offers prognostic information beyond that provided by cerebrovascular disease risk factors and hence facilitates individual at a high risk for strokes and TIA[10].Verdecchia et al[22], and Koren et al[21], showed that LV geometry has prognostic significance in ISH[21].Barry R. Davis et al[19] reported ECG signs of LVH were associated with increased risk of stroke. Virinda et al[16] showed LVH to be the commonest ECG manifestation (36.8 percent) of ISH. Gohel et al[23] showed incidence of left ventricular failure (59.7%)

ECG in Left ventricular hypertrophy

- **Sokolow-Lyon voltage criteria**

  SV1 + RV5 or RV6 $\geq$ 3.5 mV (35 mm)

  RaVL$\geq$ 1.1 mV (11 mm)

- **Cornell voltage criteria**

  SV3 + RaVL$\geq$ 2.0 mV (28 mm) in men

  SV3 + RaVL$\geq$ 2.8 mV (20 mm) in women

  (Some variations use a lower cutoff value in men)

- **Cornell product criteria**

  SV3 + RaVL (+8 in women) x QRS duration ≥ 2,440 mm × ms

- **Romhilt-Estes point score system**

  (a score $\geq$ 5 is diagnostic of LVH, a score of 4 is “probable” LVH)

  Voltage criteria (3 points):

  Any S or R in limb leads $\geq$ 20 mm

SV1, SV2, RV5, or RV6 ≥ 30 mm

ST-T wave changes of LVH

(3 points, 1 point on digitalis)

Left atrial abnormality (3 points):

Terminal component of the P wave in V1 ≥ 1 mm

And ≥ 40 ms

Left axis deviation (2 points):

QRS axis of –30 degrees or more negative

Prolonged QRS duration (1 point): ≥ 90 ms

Delayed intrinsicoid deflection time (1 point):

≥ 50 ms in V5 or V6

In LVH due to diastolic overload, there are deep narrow q-waves in left oriented leads. There may be tall and symmetrical T-waves in left precordial leads.

Echocardiography in Left ventricular hypertrophy

A two-dimensional echocardiography - Trans thoracic-echocardiogram (TTE) is useful in the diagnosis of Left ventricular hypertrophy, seen as an increase in wall thickness, often with the septum 1.3 or more times the thickness of the high posterior left ventricular free wall.

The septum may demonstrate an unusual “ground glass” appearance, probably related to its abnormal cellular architecture and myocardial fibrosis. Systolic anterior motion (SAM) of the mitral valve is found in patients with pressure gradient. The left ventricular cavity typically is small with vigorous posterior wall motion but reduced septal excursion.

A 2D echocardiogram with color flow Doppler should be obtained promptly in patients with suspected cardiogenic shock or pulmonary edema which demonstrates LV dysfunction in the form of left to right shunt with ventricular septal rupture and mitral regurgitation. Pulmonary embolism may be visualized in the form of proximal aortic dissection with aortic regurgitation or tamponade.

The clinical uses of 2D Echocardiography are

1. Assessing Chamber (left ventricular) size and function, Left Ventricular Hypertrophy (LVH), Regional Wall Motion Abnormality (RWMA) and valve morphology and motion.

2. Assessing pericardium—Effusion, in the form of black echolucent ovoid structure surrounding the heart and Tamponade, in the form of right ventricular collapse, right atrial collapse, and dilated inferior vena cava are seen respectively.

3. Any solid masses appear as echo dense structures like LV thrombus, atrial myxomas, lipomatous infiltrations of the atrial septum and calcified mitral annulus.

4. Great vessels like Aorta, where aortic dissection can be diagnosed when an intimal flap is visualized on a Transthoracic echocardiogram. Definitive diagnosis of an aortic dissection usually requires a TEE (Transesophageal echocardiogram).

There is a discrepancy in the incidence of LVH by ECG and 2D ECHO. This is due to the fact that the latter is the most sensitive predictor of LVH[24].

SHEP trial shows active therapy to be associated with a reduction in left ventricular mass index (LVMI). After a minimum follow up of three years LVMI fell by 13% in the actively treated patients and increased by 6% in the placebo group. Vrinda et

al[16]observed cerebrovascular complications in 15.4% elderly hypertensive patients with LVH. Gohel et al[23] observed sensitivity to detect left ventricular hypertrophy by electrocardiography was 37% and echocardiography and 50.5%.

Coronary Artery Disease (CAD)

Data from the Framingham study[25] and multiple risk factor intervention trial[26] indicated the importance of isolated systolic hypertension in the development of coronary artery disease. The SHEP trial noted that lowering blood pressure yielded a statistically significant reduction in adverse events related to coronary artery disease, congestive heart failure and overall cardiovascular disease[12]. Most elderly patients with isolated systolic hypertension have other risk factors for coronary disease. In the SHEP trial antihypertensive therapy reduced coronary heart disease events by 31% in patients with cholesterol levels below 216 mgs/dl, 29% in those with cholesterol levels 216 – 250 mgs/dl and by 24% in those with levels above 251 mgs/dl[08]. The Brisighella heart study[27] demonstrated a consistent, strong, gradual association between systolic blood pressure and cardiovascular events. In Syst-Eur trial significant reduction was noted in fatal and non-fatal cardiac end points. Further analysis of this trial found that the mortality benefit increased with a higher systolic blood pressure at study entry, lessened with age. Virinda et al.[16] showed 28.5% elderly hypertensive patients with ISH to have IHD, angina and LVF.

Congestive Heart Failure (CHF)

Data from Framingham heart study indicate that hypertension is a major factor in the development of congestive heart failure[28]. A review of major systolic hypertension trials in elderly demonstrated a highly statistically significant reduction (48%) in the

occurrence of congestive heart failure as a result of treatment for a relatively short period[29].

The development of heart failure occurs as a result of depressed systolic function (e.g. - EF < 45%) or with preserved systolic function (e.g. - with impairment in diastolic function seen in LVH). Heart failure in elderly with preserved function is probably due to age related fibrosis and thickening of myocardium.[11]

In a cross sectional study of patients with hypertension > 65 years with LV ejection > 45%, HF was observed in 22.6% and diastolic dysfunction in 25.8%[11].

CENTRAL NERVOUS SYSTEM

STROKE

Hypertension remains the major treatable risk factor for stroke, although the attributable risk for increasing BP levels decreases with age. According to Barry R. Davis et al[19], 5 year cumulative risk for stroke in an active treatment group were 5.1/100 and, 7.9/100 for placebo group. Old age, high SBP, heart rate, lower HDL cholesterol, smoking, diabetes mellitus, history of stroke were the important risk factors for ischemic stroke, Among them diabetes and smoking were significantly related to lacunar stroke, whereas carotid bruit and age were important risk factors for atherosclerotic and embolic stroke respectively in older patients with isolated systolic hypertension. Lacunar strokes accounted for 56% among ischemic stroke and there was 36% reduction in stroke incidence among active treatment group.

Another similar study from the Melbourne risk factor study noted hypertension was the most important risk factor for intracerebral hemorrhagic risk. The Framingham

study has shown that increased blood pressure levels impose an escalating risk for stroke. Virinda et al[16]observed strokes in 30.25% patients with ISH.

The relative risk of cerebral infarction varies depending on the hypertension type in older age groups. Isolated systolic hypertension (ISH) is a bigger risk factor (ratio 2.3) than is combined systolic and diastolic hypertension (ratio 1.5).The population attributable risk for stroke in those aged 70to 79 years with ISH is about 21% for women and 17% for men, whereas for those aged 50 to 59 years the figures are5% for women and 4% for men. A reduction of 9/5 mm Hg can be expected to produce about a 30% reduction in stroke incidence, whereas a fall of 18/10 mm Hg halves the risk; these expectations are irrespective of baseline BP levels.

HYPERTENSIVE RETINOPATHY

The primary response of the retinal arterioles to systemic hypertension is narrowing.

Pathogenesis

The vascular retinopathy is associated with raised blood pressure and with pronounced degenerative changes in the retinal vessels. The primary response of the retinal arterioles to systemic hypertension is narrowing. However the degree of narrowing is dependent on the amount of pre-existing replacement fibrosis. For this reason, hypertensive narrowing is seen in its pure form only in young individuals. In older patients, the rigidity of retinal arterioles caused by involutional sclerosis prevents the same degree of narrowing that is seen in young individuals. In sustained hypertension the blood retinal barrier is disrupted in small areas, resulting in increased vascular permeability.

The fundus picture of hypertensive retinopathy is characterized by:

a) Vasoconstriction

b) Leakage

c) Arteriosclerosis

Funduscopic findings provide one of the best indications of the duration of hypertension and of prognosis.

A useful guide is the **Keith-Wagener-Barker classification** of funduscopic changes:

Grade-I: Mild generalized arteriolar narrowing.

Grade-II: More narrowing of the arterioles and also focal arteriolar attenuation. Arterio venous crossing changes present.

Grade-III: Grade-II changes with hemorrhage, cotton wool spots and exudates.

Grade-IV: All the changes of Grade-III with papilledema including neuro-retinal edema.

HYPERTENSIVE RENAL DISEASE

Persistent exposure of renal circulation to elevated intraluminal pressures results in development of intrinsic lesions of the renal arterioles (hyaline arterio sclerosis) that eventually lead to loss of function (Nephrosclerosis).

Renal excretory function is represented by glomerular filtration rate (GFR). The filtration rate starts declining in third or fourth decade of life. By the sixth decade GFR declines by 1 to 2ml/min. if SBP is not controlled it may accelerate and GFR deteriorates to 4 to 8 ml/min.[04]Chronic kidney disease (CKD) itself is an independent risk factor for cardiovascular disease (CVD). There is 16% increase in mortality with

eGFR < 60 and it further increases to 30% with eGFR < 30. CVD also exhibit a continuous relationship with albuminuria, there is 50% increase in risk in the presence of micro albuminuria, when compared with macroalbuminuria the risk increases to 300%.[04]

MATERIALS AND METHODS

The study was conducted on inpatient of department of General Medicine in B.L.D.E.U's SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTER, BIJAPUR.

Period of Study:

This study was conducted from. OCTOBER 2010 to JULY 2012

Inclusion Criteria:

Patients above the age of 60 years having hypertension with systolic > 140 and diastolic $\leq$ 90 mm Hg according to JNC–VII report

Exclusion Criteria:

1. Patients below the age of 60 years.
2. All Diabetes Mellitus patients
3. Patients with Renal disorders.
4. Other causes of Secondary hypertension

Design:

Blood pressures were recorded for the patients and 60 elderly patients, male as well as females of age 60years and above who had hypertension were included.

Blood Pressure Measurement:

The following technique were adopted

1. Patients were seated in a chair or lying in bed.
2. Measurement of BP only after 5 minutes of rest.

3. Appropriate cuff size was used to ensure accurate measurement. The bladder within the cuff encircled attest 80% of the arm circumference.
4. The measurement of BP was taken with a mercury sphygmomanometer.
5. Both systolic and diastolic blood pressure was recorded. The appearance of sound (Phase I) was used to define SBP. The disappearance of sound (Phase V) was used to define DBP. Pulse pressure was calculated as SBP – DBP.
6. Hypertension was defined as per JNC VII criteria.

Diagnostic Criteria Employed:

JNC – 7 Criteria was employed for the diagnosis of Hypertension.

JNC – 7 GUIDELINES 2003

(Joint National Committee – Seventh report 2003) Category	Systolic BP mm Hg	Diastolic BP mm Hg
Normal	<120	<80
Pre Hypertension	120-139	80-89
Stage 1 Hypertension	140-159	90-99
Stage 2 Hypertension	≥160	≥100
Isolated systolic hypertension	>140	<90

METHODOLOGY

A detailed history and clinical examination was carried out in all the subjects who gave informed consent to participate in the study and necessary investigations were recorded. A detailed proforma of this study is enclosed in this book.

The following investigations were conducted on all these patients:

1. Blood Routine- Hb%, TLC, DLC & ESR
2. Fasting Blood Sugar (FBS) and Post Prandial Blood Sugar (PPBS).
3. Chest X-ray PA view
4. Electrocardiogram (ECG) in all leads
5. Echocardiogram (2D ECHO, Trans thoracic)
6. Blood Urea, Serum Creatinine
7. Lipid profile
8. Urine – Albumin, Sugar, Microscopic
9. Fundus Examination and
10. Thyroid Profile (if necessary).

Method of Statistical Analysis

Chi-Square test of significance:

In this study, the investigator has used to find the association between Systolic Blood Pressure (SBP) – Stage 1 and Stage 2, with several variables.

Student t-test of significance:

This test is used to test the difference between two means. In this study the following variables were tested against Systolic Blood Pressure (SBP) – Stage 1 and Stage 2.

Factor Analysis:

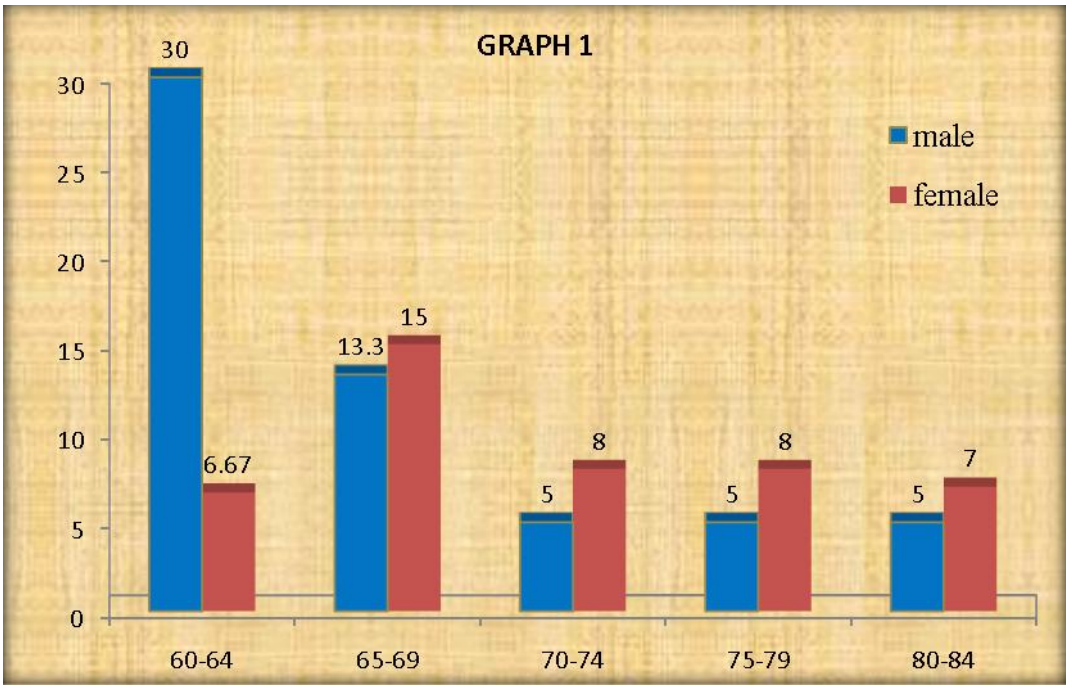
It is a powerful method of multivariant technique. It aims at explaining several correlated variables in terms of relatively few factors. In this study, this technique is used to reduce the data namely variables into smaller number of factors. The method used is Principle Component Analysis.

OBSERVATIONS AND RESULTS

A total number of 60 geriatric hypertensive patients of age group ≥ 60 years, who were admitted in SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL AND RESEACRH CENTER, BIJAPUR were selected

TABLE-1: AGE AND SEX DISTRIBUTION IN STUDY GROUP

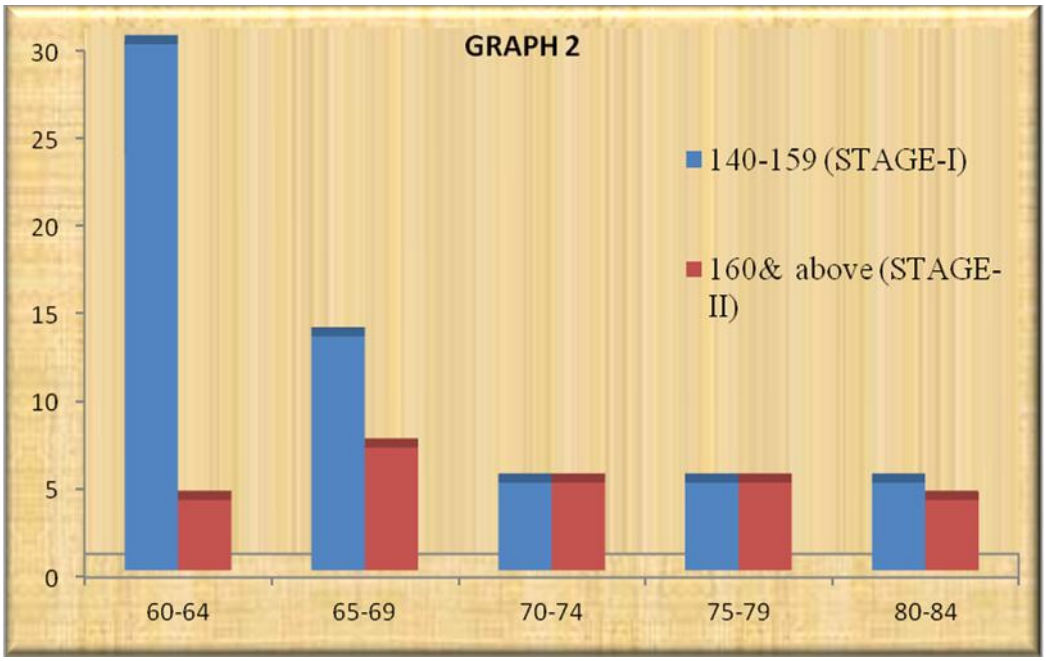
Age in years	male	%	female	%	Total no of patients	percent
60-64	18	30	04	06.67	22	36.67
65-69	08	13.3	07	11.67	15	25.00
70-74	03	05	05	08.33	08	13.3
75-79	03	05	05	08.33	08	13.3
80-84	03	05	04	06.67	07	11.67
Total	35	58.33	25	41.67	60	100



- From the above table, we observed that hypertension is more common in males having 35 patients (58.33%) compared to females having 25 patients (41.67).
- The commonest age group is 60-64 years having 22 patients (36.67%) followed by 65-69 years having 15 patients (25%). Least common age group is 80-84 having 07 patients (11.67%).
- Among males commonest age group is 60-64 years having 18 patients (30%), and in females 65-69 years is common having 07 patients (11.67%).

TABLE-2: DISTRIBUTION OF SYSTOLIC BLOOD PRESSURE IN STUDY GROUP

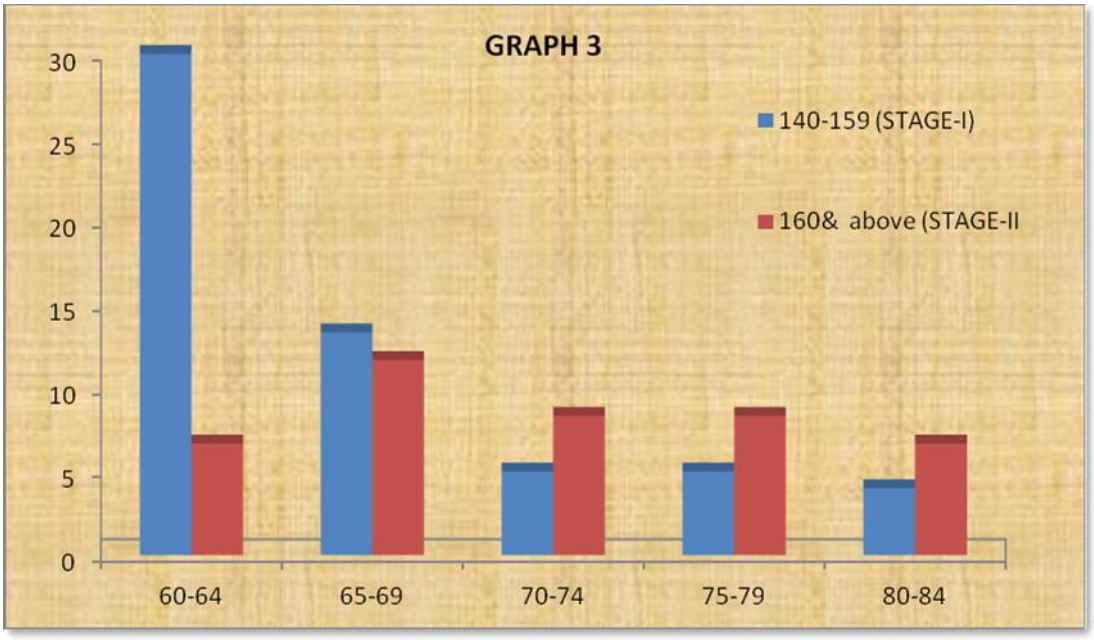
SBP(mmHg)	Male	%	Female	%	Total	%
140-159 (STAGE-I)	23	38.33	14	23.33	37	61.67
160 and above (STAGE-II)	12	20	11	18.33	23	38.33
Total	35	58.33	25	41.67	60	100



- From the above table we observed that stage-I SBP is common with 37 patients (61.67%) when compared with grade-II SBP comprising of 23 patients (38.33%).
- Stage-I SBP was common in both sexes with 23 patients (38.33%) and 14 patients (23.33%) in male and females respectively.
- When stage-II is taken into consideration there are 12(20%) male patients and 11(18.33%) female patients.

TABLE-3: DISTRIBUTION OF AGE WITH SYSTOLIC BLOOD PRESSURE IN STUDY GROUP

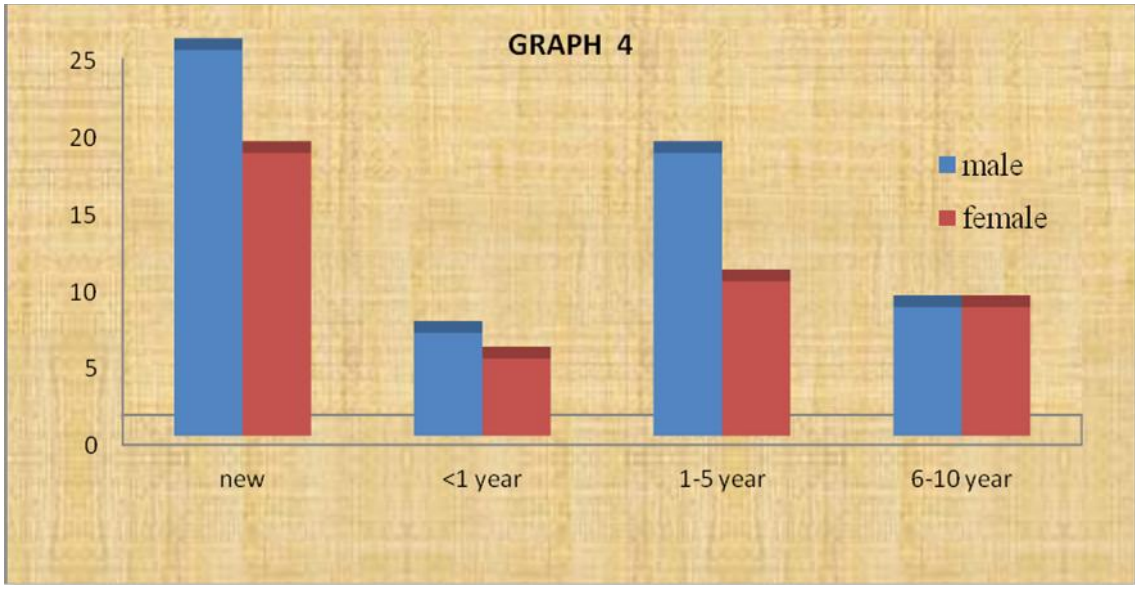
Age in years	140-159 (STAGE-I)	%	160& above (STAGE-II)	%
60-64	18	30	04	06.67
65-69	08	13.33	07	11.67
70-74	03	05	05	08.33
75-79	03	05	05	08.33
80-84	03	05	04	06.67
Total	35	58.33	25	41.67



From the above table it is evident that stage-I SBP is common in 60-64 yearswith 18 patients (30%) and stage-II is common in 65-69 years with 07 patients (11.67%).

TABLE-4: DURATION OF HYPERTENSION IN STUDY GROUP

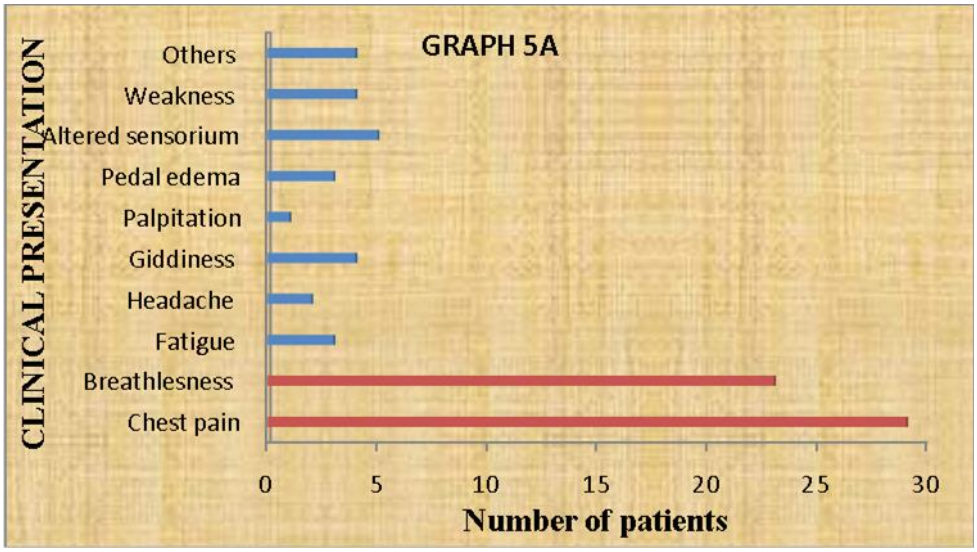
Years	Male	%	female	%	Total	%
New	15	25	11	18.33	26	43.33
< 1yr	04	6.67	03	05	07	11.67
1-5yr	11	18.33	06	10	17	28.33
6-10yr	05	08.33	05	08.33	10	16.67
Total	35	58.33	25	41.67	60	100



- From the above table we observed that there are 26(43.33%) newly detected hypertensive patients. Out of them 15(25%) are male patients and 11(18.33%) are female patients.
- Newly detected hypertension is more common in males when compared to females.
- 7(11.67%) patients had history of hypertension less than 1 year, out of them 5 patients were not on treatment.
- 17(28.33%) patients had history of hypertension between 1-5 years, and 10(16.67%) patients between 6-10 years.

TABLE-5: SYMPTOMATOLOGY IN STUDY GROUP

Symptom	Number	%
Chest pain	29	48.33
Breathlessness	23	38.33
Fatigue	3	05
Headache	2	03.33
Giddiness	4	06.67
Blurring of vision	0	0
Syncope	0	0
Palpitation	1	01.67
Pedal edema	3	05
Convulsions	0	0
Altered sensorium	5	08.33
Weakness	4	06.67
Others	4	06.67



- In our study, the most common presentation was chest pain- 29 patients (48.32%), followed by dyspnea -23 patients (38.37%). On detailed examination 28 46.67%) patients had IHD; out of 33 patients 19 (57.58%) patients had the chest pain; 9 (27.27%) patients had breathlessness on exertion.

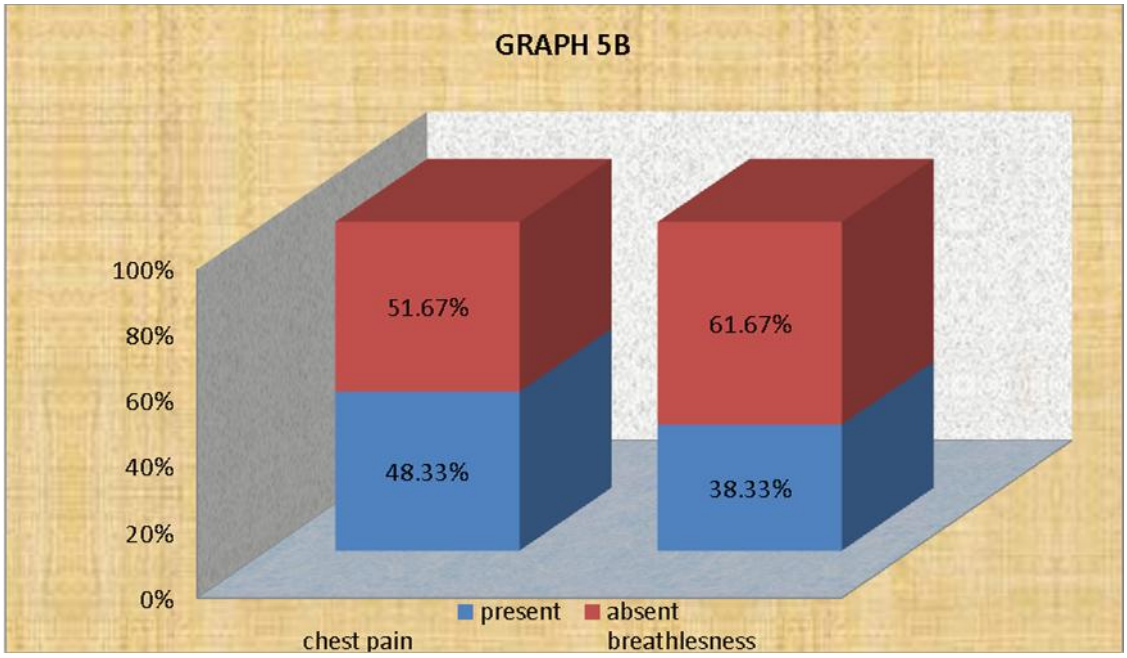
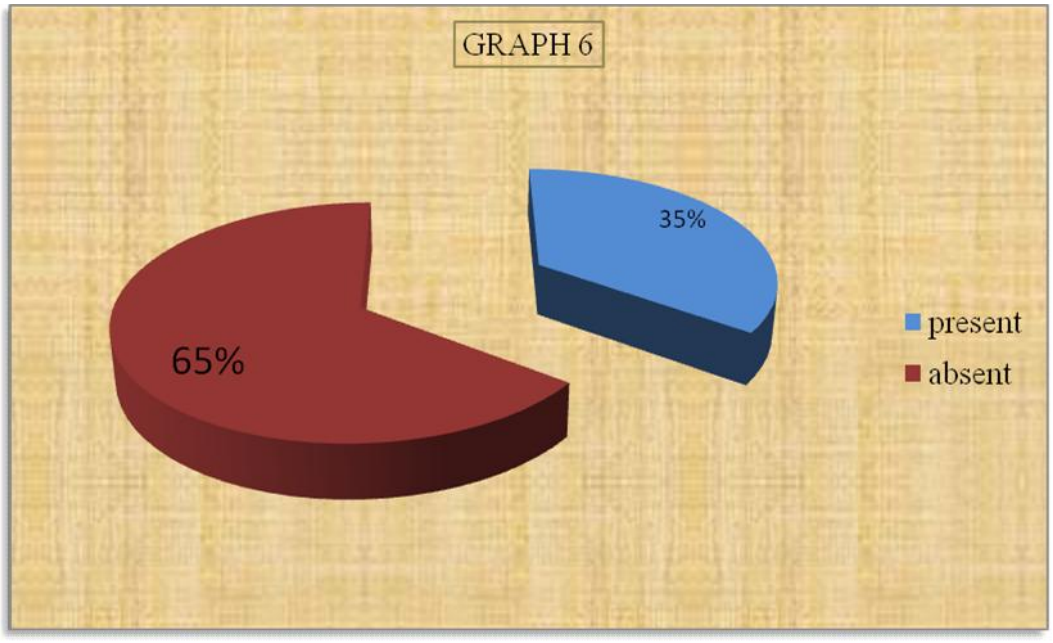


TABLE-6: OTHER RELEVENT HISTORY IN STUDY GROUP

	Number	%
Past history	08	13.33
Family history	22	36.67
Smoking history	21	35
Alcohol history	16	26.67

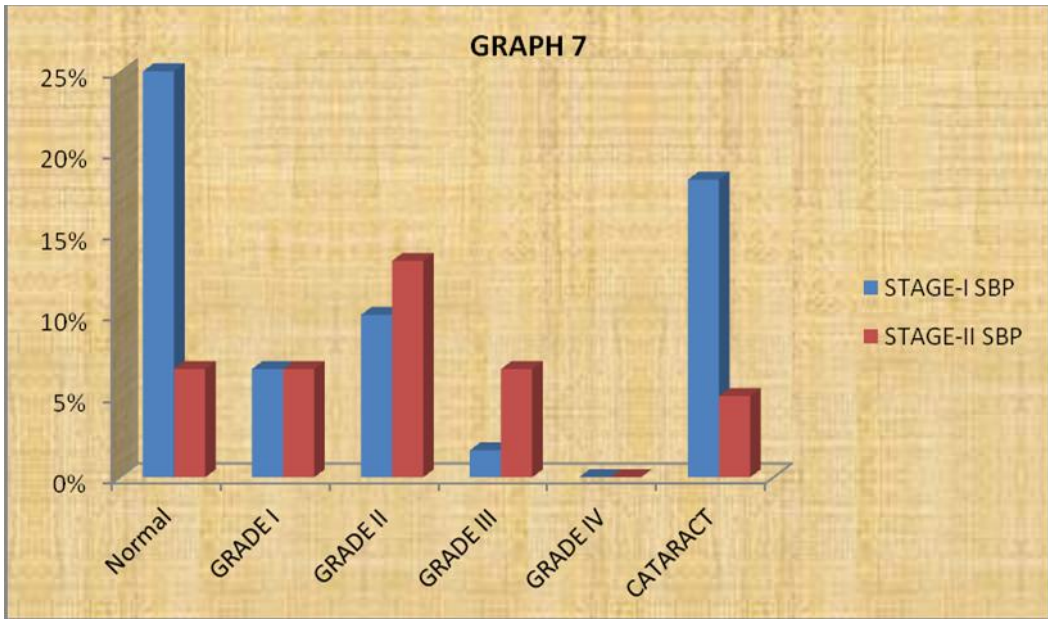


In our study 8 (13.33%) patients had past history of IHD and 22 patients (36.67%) had family history of hypertension in which 14 patients had family history of Diabetes mellitus and hypertension in first degree relatives.

21 (35%) patients had smoking history**,** 16 (26.67%) patients had alcohol history, and 13 (21.67%) patients had both smoking and alcohol history

TABLE-7: FUNDUS EXAMINATION IN STUDY GROUP

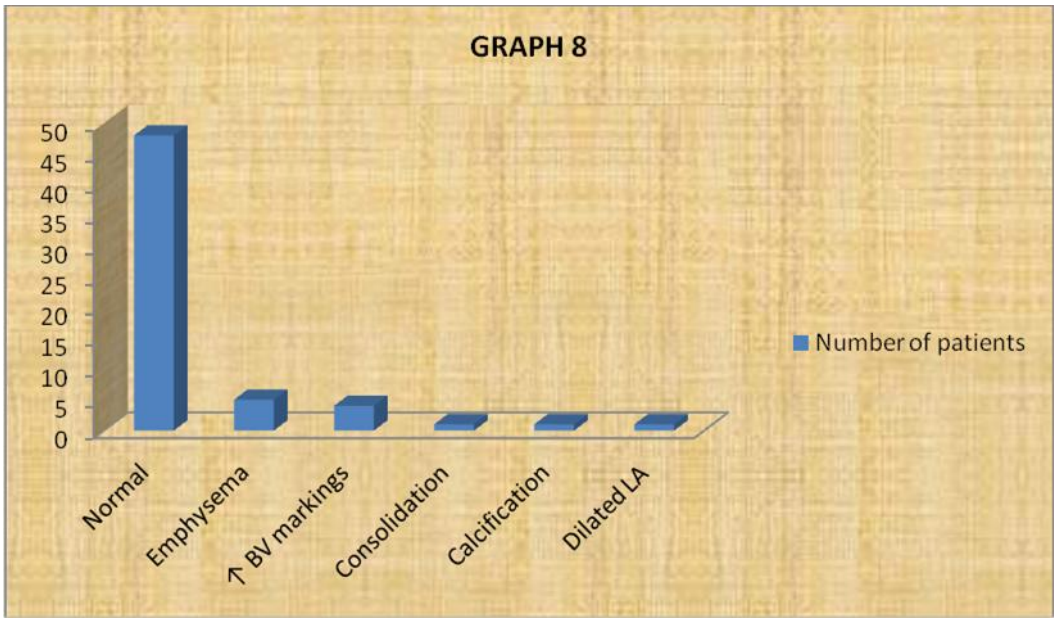
Fundus	STAGE-I SBP		STAGE-II SBP		TOTAL	
NORMAL	15	25%	04	6.67%	19	31.67%
GRADE I	04	6.67%	04	6.67%	08	13.33%
GRADE II	06	10%	08	13.33%	14	23.33%
GRADE III	01	1.67%	04	6.67%	05	8.33%
GRADE IV	00	0.00%	00	0.00%	00	0.00%
CATARACT	11	18.33%	03	05%	14	23.33%



- Ophthalmic fundus examination is done for all the patients. Keith Wagner barker classification was used.
  - 19 patients (31.67%) had normal fundus;
  - 14 patients (23.33%) had cataract;
  - 08 patients (13.33%) had Grade I Hypertensive Retinopathy;
  - 14 patients (23.33%) had Grade II Hypertensive Retinopathy;
  - 05 patients (8.33%) had Grade III Hypertensive Retinopathy.
- Grade II hypertensive retinopathy was more common in both stage I and II SBP.
- Hypertensive retinopathy changes were more common in Stage II hypertension.

TABLE-8: CHEST X-RAY FINDINGS IN STUDY GROUP

X-ray	Number of patients	%
Normal	48	80
Emphysema	05	8.33
Increased bronchovesicular markings	04	6.67
Consolidation	01	1.67
calcification	01	1.67
Dilated LA	01	1.67

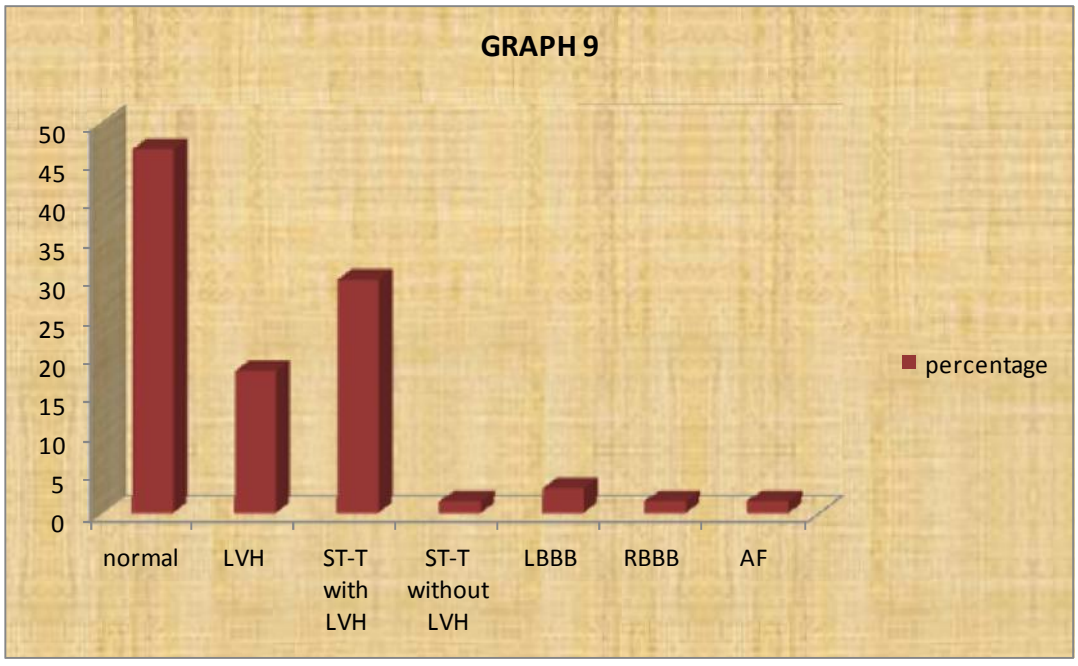


In this study, 48 patients had normal chest X-ray,12 patients had abnormal chest x-ray in the form of

- Emphysematous changes- 05
- Increased bronchovesicular markings-04
- Consolidation-01
- Calcification-01
- Dilated LA -01

TABLE-9: ECG FINDINGS IN STUDY GROUP

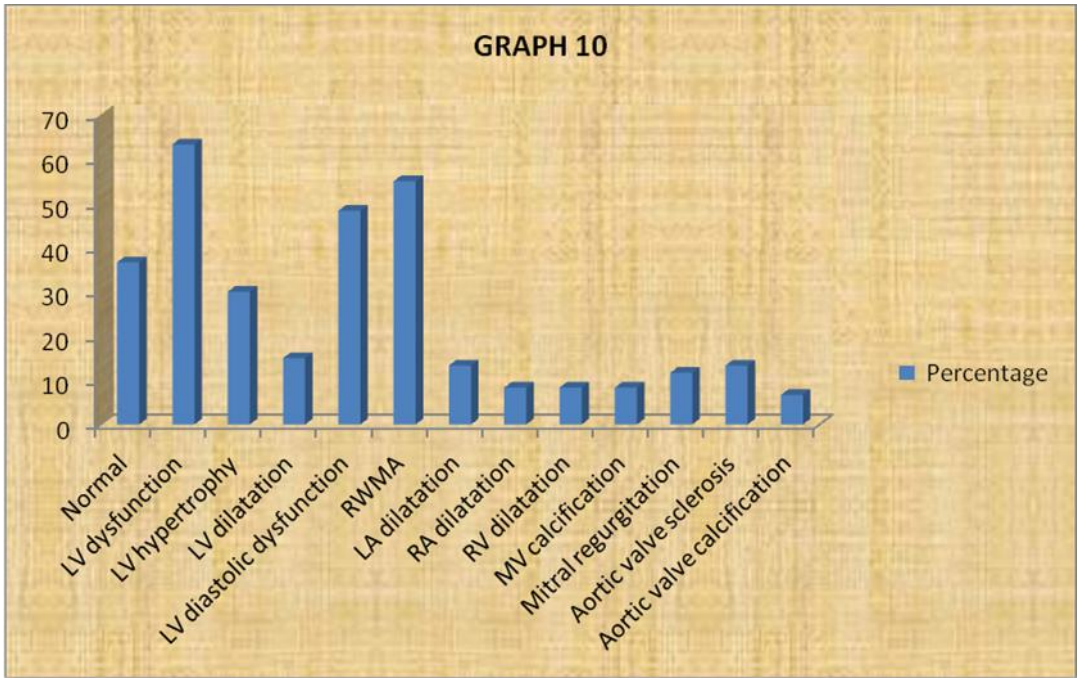
ECG	Number of patients	%
Normal	28	46.67
Left ventricular hypertrophy	11	18.33
ST-T changes without LVH	18	30
ST-T changes with LVH	01	1.67
LBBB	02	3.33
RBBB	01	1.67
Atrial fibrillation	01	1.67



From above table it is evident that ischemic changes (31.67%) were most common ECG findings followed by left ventricular hypertrophy (20%).

TABLE-10: ECHOCARDIOGRAPHY FINDINGS IN STUDY GROUP

Echocardiogram	Number of patients	%
Normal	22	36.67
LV dysfunction	38	63.33
LV hypertrophy	18	30
LV dilatation	09	15
LV diastolic dysfunction	29	48.33
RWMA	33	55
LA dilatation	08	13.33
RA dilatation	05	8.33
RV dilatation	05	8.33
MV calcification	05	8.33
Mitral regurgitation	07	11.67
Aortic valve sclerosis	08	13.33
Aortic valve calcification	04	6.67



In this study, echocardiography showed left ventricular dysfunction in 38 patients (63.33%) in the form of

-LV hypertrophy in 18 patients (30%)

-LV dilatation in 09(15%)

-LV diastolic dysfunction in 29(48.33%)

Another common finding was 33(55%) patients had regional wall motion abnormality

- Anterior-18 patients (54.55%)
- Lateral- 4 patients (12.12%)
- Inferior- 3 patients (9.09%)
- Septal- 4 patients (12.12%)
- Global hypokinesia- 1patient

And 3 patients had more than one segment involvement

Posterior and lateral

Inferior and lateral

Anterior and inferior

Statistical Analysis and Significance

In this study, we have tried to find the association between Systolic Blood Pressure (SBP) with several variables.

CHI-SQUARE TEST

VARIABLE	P-VALUE	SIGNIFICANCE
Sex	>0.05	Not significant
Symptom	<0.05	Significant
Chest pain	<0.05	Significant
Breathlessness	<0.05	Significant
Fatigue	<0.05	Significant
Giddiness	>0.05	Not significant
Palpitation	>0.05	Not significant
Pedal edema	>0.05	Not significant
Altered sensorium	>0.05	Not significant
Weakness	>0.05	Not significant
Smoking	<0.05	Significant
Alcohol	>0.05	Not significant
Proteinuria	<0.05	Significant
ECG	<0.05	Significant
LV hypertrophy	<0.05	Significant
LVDD	>0.05	Not significant
Ischemia	<0.05	Significant

Chi-square was applied to the above variables, and P value less than 0.05 was considered significant. The following variables were found to have association with systolic blood pressure in both stage I and II - symptom, chest pain, breathlessness, fatigue, smoking, proteinuria, ECG changes, LV hypertrophy and ischemia.

STUDENT T-TEST

VARIABLE	P-VALUE	SIGNIFICANCE
Age	<0.05	Significant
Creatinine	>0.05	Not significant
Cholesterol total	<0.05	Significant
Serum triglyceride	>0.05	Not significant
High density lipoprotein	>0.05	Not significant
Low density lipoprotein	>0.05	Not significant
Left ventricular mass	<0.05	Significant
Ejection fraction	>0.05	Not significant

Student T-test was applied for above variables and mean age, mean cholesterol total and mean LV mass was found statistically significant between Stage I and II systolic blood pressure.

FACTORIAL ANALYSIS:

SL. NO.	VARIABLE	FACTOR LOADING	COMMUNALITIES
1	ST	+0.872	0.676
2	CT	+0.814	0.453
3	LVIDS	-0.077	0.809
4	LV M	+0.240	0.705
5	ISCHEMIC	+0.159	0.574
6	IVSD	+0.071	0.883
7	LV	+0.073	0.599
8	CHEST PAIN	-0.148	0.418
9	LVIDD	-0.004	0.593
10	DYSPNEA	-0.401	0.733
11	PALPITATION	+0.016	0.767
12	GIDDINESS	-0.111	0.441
13	FATIGUE	-0.005	0.722
14	LVPWD	+0.241	0.591
15	ECG	+0.095	0.729

Factorial analysis is done for important variables, 64.62% of the problem are explained by first 6 variables only. The variables (1, 2, 5, 7 and15) are the significant clinical findings, the variables (08, 10) are the chief complaints of the patients (3, 9, 11, 12, 13) have the negative values indicating buckling load has exceeded.

DISCUSSION

Isolated systolic hypertension is the commonest cause of raised blood pressure in older population. A common misconception among patients and practitioners is that elevated diastolic blood pressure is more important than elevated systolic pressure. In fact one of the key message of JNC VII is in persons older than 50 years, systolic blood pressure of more than 140 mmHg is a much more cardiovascular risk factor than DBP.

The present study aims at detecting end organ complications of ISH mainly on cardia through ECG & ECHO in term of left ventricular hypertrophy, regional wall abnormalities, reduced ejection fraction, which may result /manifest as CAD, MI & CCF

Age:

Age is the important risk factor for development of complications in elderly hypertensive patients. Isolated Systolic Hypertension is strongly age dependent. This age dependent pattern of increasing rates of ISH is observed in Framingham Heart Study and (NHANES) III.

Anne-Sophie Rigaud[30] reported increased frequency of ISH in elderly hypertensive patients in the age group of 65-74 years. S. Franklin et al[05]reported 87% frequency for ISH in elderly hypertensive patients in the age of 60-69 years. Vrinda et al[16], N C Hazarika et al[03], Wilfred F. Heesen et al[31]all have considered 60 years and above as a cut off point for studying ISH in elderly.

In our study 60 patients with hypertension were studied with mean age 67.72 7.004 years which is similar to N C Hazarika et al[03]Anand et al[32.] The common age group in our study was 60-65yr which is similar to Vrinda et al.[16]

Sex:

In our study of 60 patients, there were 35 males and 25 females. Gupta et al[15], comparing rural and urban population showed that prevalence of ISH was 59.9 per 1000 males and 69.9 per 1000 females, respectively in urban population and 35.5 and 35.9 per 1000 in males and females respectively in rural population. Wilking S.B. et al[33]showed female predominant of ISH. N C Hazarika et al[03]detected Isolated Systolic Hypertension in 13.2% males and 10.31% females among elderly hypertensives. Vrinda et al[16]has observed male to female ratio of 1.3:1. Anand et al[32] found male to female ratio to be 1.6:1, which correlates with our study. This could be due to male dominance in our society. This could also be confirmed by the fact that the percentage of males coming as outpatients and inpatients are higher than that of female.

Clinical Presentation:

In our study, the most common presentation was chest pain 48.33%, followed by breathlessness 38.37%. On detailed examination 33 (55%) patients had IHD; out of 33 patients 16(48.48) patients had the classical angina; 08(25%) patients had breathlessness on exertion. Both these symptoms were statistically significant and were important predictors of cardiovascular complications.

Gupta et al[15]found hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths in India. Holay et al[34] observed in his study that Atypical chest pain, sweating, breathlessness and giddiness were common presentation in the elderly group. In contrast to our study Vrinda et al[16] found headache as common symptom. Brisighella Heart Study[27] also concluded that there was increased cardiovascular risk as systolic blood pressure increases.

Duration of hypertension:

In our study 32 patients had hypertension history less than 1 year, out of them 26 were newly detected and 7 were having history less than 1 year, out of 7 patients 5 were not taking treatment.17 patients had hypertension since 1-5 years and 11 patients had hypertension since 6-10 years. From the above study it is evident that there is lack of knowledge in patients about hypertension and its complications.

Family History:

In our study, 22 patients had family history of hypertension and this confirms family history of hypertension as important risk factor of hypertension. Recent studies have shown family history of hypertension as an important determinant of hypertension. Vrinda et al[16] report 55.4% patients having family history of hypertension.

Fundus Examination:

Increasing severity of hypertension is associated with focal spasm and progressive general narrowing of arterioles as well as the appearance of hemorrhages, exudates and papilloedema. In our study, 08 patients had Grade I Hypertensive Retinopathy;

14 patients had Grade II Hypertensive Retinopathy; 05 patients had Grade III Hypertensive Retinopathy, 14 patients had cataract changes, 19 patients had normal fundus.

Out of 27 patients, 16 of them were of Stage-2 systolic blood pressure. This shows that increasing systolic blood pressure also increases retinopathy along with cardiovascular risks and cerebrovascular risks. Vrinda et al[16] found Grade II hypertensive retinopathy in 50.5% patients.

In the Framingham Heart Study it has been demonstrated that hypertension seldom occurs in isolation of other atherogenic risk factors with which it tend to cluster. This clustering with other metabolically linked risk factors has been shown to reflect,

insulin resistance promoted by weight gain and abdominal obesity which was shown to be the major determinant of hypertension in general population.

Dyslipidemia:

Dyslipidemia is as important factor for atherogenesis. Accelerated atherosclerosis is an invariable companion of hypertension. In our study 36 patients (60%) had dyslipidemia and Cholesterol total was found statistically significant.This is similar to study done by Vrinda et al[16]where dyslipidemia is 55.9%. In Framingham study incidence of MI was directly related with increase in cholesterol total levels and inversely related to low HDL levels in women. Similar results were obtained for men in Copenhagen male study[08] which supports our present study.

According to AHA guidelines[11] for hypertension in elderly, it is observed that dyslipidemia is concomitant in elderly patients. it was observed in PROSPER and HYVET trial dyslipidemia was common.

Systolic Blood Pressure:

Systolic blood pressure is a continuous variable and its associated risks increases from the lowest to the highest values. This had been shown by the analysis of data from multiple risk factor intervention trial[26].

In our study 37 patients had Systolic Blood Pressure (SBP) between 140-159 mm Hg and 23 patients had SBP 160 mm Hg and above. Stage I SBP was common and male predominance was seen in our study. Common age group was between 60 to 70 years. SBP was associated with complaints like chest pain and breathlessness, smoking history, proteinuria, hypertensive retinopathy, dyslipidemia, left ventricular hypertrophy and ischemia. Alex Sagie et al.[25], found that patients with borderline ISH are at an increased risk of cardiovascular disease. The increased frequency of

cardiovascular risk was even more striking when compared with those with optimal blood pressure. Chicago stroke study found SBP to be a greater risk factor for stroke. Stamler J. Neaton et al [26] and Brisighella heart study[27] pointed to and SBP as a key determinant of mortality. However, Kannel et al.[02] using multivariant analysis demonstrated that there is no evidence that liability of pressure independently influences cardiovascular risk in subjects with isolated systolic hypertension.

Coronary Heart Disease:

Coronary heart disease is the major cause of morbidity and mortality in both elderly men and women. Hypertension either systolic/diastolic or isolated systolic hypertension is considered as a major risk factor for Coronary heart disease. In our study ischemic heart disease was diagnosed in 55% patients. The clinical profile of these patients was 60 patients out of 48.33% had chest pain and 38.33% of them also had breathlessness. Out of these 33(55%) patients, 18 (54.55%) had anterior, 4(12.12%) lateral, 3(9.09%) inferior, and 4(12.12%) septal infarcts. 1 patient had global hypokinesia, and 3 patients had more than one segment involvement. Vrinda et al[16] reported 32.3% incidence of ischemic heart disease. Brisighella heart study[27] reported 22.82% incidence of ischemic heart disease. Kannel WB et al[02] and Neaton et al[26] indicated the importance of isolated systolic hypertension in the development of Coronary artery disease.

The prevention of congestive heart failure and progression from less severe to more severe hypertension is probably more significant in the elderly. Moser M. et al[29] demonstrated a highly statistically significant reduction (48%) in the occurrence of congestive heart failure as a result of treatment for relatively short period in elderly hypertensive.

ST-T changes suggesting myocardial ischemia was the commonest ECG manifestation seen in our study. Among 60 patients, 32 patients had abnormal findings. Out of these 18(30%) had ischemic changes, 11(18.33) had LV hypertrophy changes, 01 had both ischemia and LVH. Vrinda et al[16] observed LVH in ECG in 36.8% patients and cardiovascular complications 19%. Welfred Hessen et al[31] found LVH in 47% patients. Gupta et al[15] commented Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths in India. Gohel et al[23] found Incidence of cardiac disease was 15.7% out of which incidence of left ventricular failure -59.7% myocardial infarction -24.3%, angina pectoris was16.2%. . Sensitivity to detect left ventricular hypertrophy by electrocardiography was 37%.

In our study Echocardiogram (ECHO) was done for all 60 patients. Out of 60 patients:-60% had Left Ventricular Dysfunction in the form of Left Ventricular Hypertrophy in 18 (30%) patients. Left Ventricular Dilatation in 09 (15%) patients. Left Ventricular Diastolic Dysfunction in 29 (48.33%) patients and RWMA (Regional wall motion abnormality) in 33 (55%) patients.

Various trials and their results

	% Risk Reduction					
Trial name	CVA	MI	CHF	Total CVD	All-cause mortality	CV mortality
ACCOMPLISH	16	NR	↑4	17*	10	20*
ALLHAT	7	ND	↑38*	↑38*	↑4	NR
EWPHE	36	20	22	29	9	27*
HYVET	30	NR	64*	34*	21	23
SHEP	36*	25	55	32	13	20
STONE	57*	6	68	60*	45	26
STOP-HTN	47	13	51	40	43	50
SYST-EUR	42	26	36	31	14	27
SYST-CHINA	38*	33	38	37*	39*	39*

SUMMARY

60 elderly patients having Isolated Systolic Hypertension (ISH) were studied in B.L.D.E.U's SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTER, BIJAPUR from a period of October 2010 to July 2012.

The study was conducted to know the clinical profile of hypertension in elderly (above the age of 60 years), to find out any other associated risk factors, any end organ complications, especially on cardiac through echocardiography.

1. Isolated Systolic Hypertension is a disease of elderly and related to ageing and arterial stiffness.
2. The mean age was 67.72±7.004 years and the common age group in our study was 60-65 years
3. Out of 60 cases 35 were males and 25 were females
4. SBP stage I was common with 37 (61.67%) patients
5. Most common clinical presentation was chest pain (48.33%) and breathlessness (38.33%). Both were statistically significant.
6. Newly detected hypertensive patients were 26 (18.33%) and they presented with complications.
7. 61.67% of patients had SBP of 140-159 (stage1) and 38.33% of patients had SBP>160(stage 2). There was significant correlation between SBP (1 & 2) and cardiovascular end organ complications were noted.
8. 22 patients (36.67%) had family history of hypertension and 21 (35%) had smoking habits which were statistically significant in our study.

9. 27 patients (45%) had hypertensive retinopathy with grade II as common presentation 14 patients (50.5%)
10. Dyslipidemia was seen in 36 patients (60%) with cholesterol as independent predictor of risk in ISH.
11. Most common ECG abnormality was ischemia followed by LV hypertrophy and both had strong correlation with SBP in our study.
12. The echocardiographic evaluation showed LV dysfunction (63.33%) was common entity in the form of LV hypertrophy (30%), LV dilatation (15%) and LV diastolic dysfunction (48.33%). There was strong co relation between LV hypertrophy and SBP.
13. In our study ischemic heart disease was diagnosed in 55% patients. Out of these 18 (54.55%) had anterior, 4(12.12%) lateral, 3(9.09%) inferior, and 4(12.12%) septal infarcts. 1 patient had global hypokinesia, and 3 patients had more than one segment involvement. There is association between SBP and ischemia

CONCLUSION

- In this study of elderly patients of 60 years and above, mean systolic blood pressure (SBP) increases with age until the 8th decade and may be beyond.
- Chest pains, Breathlessness are the commonest clinical presentations. Smoking, family history and Dyslipidemia are associated risk factors.
- ECG and ECHO evidence of significant LVH, and LV dysfunction and ischemia respectively, are the commonest investigative findings.
- So the goal of treatment of hypertension in Geriatric patients should be to reduce the blood pressure to less than 140/90 mm Hg and to maintain at the Pre-hypertension level (120-139 SBP and 80-89 DBP).

RERERENCE

1. Franklin SS. Hypertension in Older People: Part 1. The Journal of Clinical Hypertension. 2006; 8(6):444-9.
2. Kannel WB, Gordon T, Schukartz MJ; Systolic vs. diastolic blood pressure and risk of coronary heart disease: the Framingham Study, Am J Cardiol 27 1971 335-345
3. Hazarika NC, Biswas D, Mahanta J. Hypertension in the elderly population of Assam. *J Assoc Physicians India.* 2003;51:567- 73.
4. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, et al. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003;42(6):1206-52.
5. Franklin SS, Jacobs MJ, Wong ND, L'Italien GJ, Lapuerta P. Predominance of Isolated Systolic Hypertension Among Middle-Aged and Elderly US Hypertensives. Hypertension. 2001 March 1, 2001;37(3):869-74.
6. Gupta R, Kasliwal R. Understanding systolic hypertension in the elderly. JAPI. 2004;52..
7. JAIN, PK, and SHAILESH KUMAR SRIVASTAVA. "Isolated Systolic Hypertension." Medicine Update: chapter 92:538-540
8. Black HR, Elliott W. Chapter 14, Chapter 38 Hypertension: A companion to Braunwald's heart disease: Saunders; 2006. p .169-174,457-468.
9. Potter JF. CHAPTER 43 - Hypertension. :Brocklehurst's Textbook of Geriatric Medicine and Gerontology (SEVENTH EDITION). Philadelphia: W.B. Saunders; 2010. p. 300-11.
10. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. The New England journal of medicine. 1990 May 31;322(22):1561-6. PubMed PMID: 2139921. Epub 1990/05/31. eng.; N.E.J.M. 1990; 332: 1561-1566.
11. Aronow WS, Fleg JL, Pepine CJ, Artinian NT, Bakris G, Brown AS, et al. ACCF/AHA 2011 expert consensus document on hypertension in the elderly: Journal of the American College of Cardiology. 2011 May 17; 57(20):2037-114. PubMed PMID: 21524875. Epub 2011/04/29. eng.

12. Implications of the systolic hypertension in the elderly program. The Systolic Hypertension in the Elderly Program Cooperative Research Group. Hypertension. 1993 Mar;21(3):335-43. PubMed PMID: 8478043. Epub 1993/03/01. eng..
13. Maddens M, Imam K, Ashkar A. Hypertension in the elderly. Primary care. 2005 Sep; 32(3):723-53. PubMed PMID: 16140125. Epub 2005/09/06. eng.
14. Indian Hypertension Guidelines-II;JAPI; http://www.apiindia.org, 5-39
15. Gupta R. Trends in hypertension epidemiology in India. Journal of Human Hypertension. Feb 1, 2004;18, 73–78.
16. Kulkarni V, Bhagwat N, Hakim A, Kamath S, Soneji SL: Hypertension in the Elderly. J Assoc Phy India (JAPI) 2001, 49:873-876.
17. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G, et al. 2007 Guidelines for the management of arterial hypertension European Society of Cardiology (ESC). European heart journal. 2007; 28(12):1462-536.
18. Fauci AS. Chapter 247. Hypertensive Vascular Disease, Harrison's principles of internal medicine: (18th edition) McGraw-Hill Medical New York; 2008 .p. 2042-2059.
19. Davis BR, Vogt T, Frost PH, Burlando A, Cohen J, Wilson A, et al. Risk Factors for Stroke and Type of Stroke in Persons With Isolated Systolic Hypertension. Stroke. 1998 July 1, 1998; 29(7):1333-40.
20. Lu FH, Tang SJ, Wu JS, Yang YC, Chang CJ. Hypertension in elderly persons: its prevalence and associated cardiovascular risk factors in Tainan City, southern Taiwan. The journals of gerontology Series A, Biological sciences and medical sciences. 2000 Aug; 55(8):M463-8. PubMed PMID: 10952370. Epub 2000/08/22. eng.
21. 1. Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of Left Ventricular Mass and Geometry to Morbidity and Mortality in Uncomplicated Essential Hypertension. Annals of Internal Medicine. 1991;114(5):345-52.
22. Verdecchia P, Schillaci G, Borgioni C, Ciucci A, Battistelli M, Bartoccini C, et al. Adverse prognostic significance of concentric remodeling of the left ventricle in hypertensive patients with normal left ventricular mass. Journal of the American College of Cardiology. 1995; 25(4):871-8.
23. Gohel DR, Dalal K, abstract ; Prevalence of Medical Disorders in Elderly Patients with Special Emphasis on Geriatic Hypertension; japi 2003.

24. Vijan SG, Manning G, Millar-Craig MW. How reliable is the electrocardiogram in detecting left ventricular hypertrophy in hypertension? Postgraduate Medical Journal. 1991 July 1, 1991; 67(789):646-8.
25. Sagie A, Larson MG, Levy D. The Natural History of Borderline Isolated Systolic Hypertension. New England Journal of Medicine. 1993; 329(26):1912-7..
26. Neaton JD, Wentworth DN, Cutler J, Stamler J, Kuller L. Risk factors for death from different types of stroke. Multiple Risk Factor Intervention Trial Research Group. Annals of epidemiology. 1993 Sep; 3(5):493-9. PubMed PMID: 8167825. Epub 1993/09/01. eng.
27. Borghi C, Dormi A, L'Italien G, Lapuerta P, Franklin SS, Collatina S, et al. The Relationship Between Systolic Blood Pressure and Cardiovascular Risk—Results of the Brisighella Heart Study. The Journal of Clinical Hypertension. 2003;5(1):47-52.
28. Levy D, Larson MG, Vasan RS, Kannel WB, Ho KK. The progression from hypertension to congestive heart failure. JAMA: the journal of the American Medical Association. 1996 May 22-29; 275(20):1557-62. PubMed PMID: 8622246. Epub 1996/05/22. eng.
29. Moser M. Hypertension treatment and the prevention of coronary heart disease in the elderly. American family physician. 1999 Mar 1;59(5):1248-56. PubMed PMID: 10088879. Epub 1999/03/24. eng.
30. Rigaud AS, Forette B. Treatment of Hypertension in the Elderly. Geriatrics and Ageing. 2002; 5(1):27-9.
31. Heesen WF, Beltman FW, May JF, Smit AJ, de Graeff PA, Havinga TK, et al. High prevalence of concentric remodeling in elderly individuals with isolated systolic hypertension from a population survey. Hypertension. 1997 Feb;29(2):539-43. PubMed PMID: 9040435. Epub 1997/02/01. eng.
32. Wilking SV, Belanger A, Kannel WB, D'Agostino RB, Steel K. Determinants of isolated systolic hypertension. JAMA : the journal of the American Medical Association. 1988 Dec 16;260(23):3451-5. PubMed PMID: 3210285. Epub 1988/12/16. eng.
33. Gupta M, Anand J, Singh H, Aggarwal R, Verma R, Gupta G. Echocardiographic Assessment of a Healthy Geriatric Population. Journal, Indian Academy of Clinical Medicine. 2004; 5(1).

34. Holay M, Janbandhu A, Javahirani A, Pandharipande M, Suryawanshi S. Clinical profile of acute myocardial infarction in elderly (prospective study). JAPI. 2007; 55.
35. Burt VL, Whelton P, Roccella EJ, Brown C, Cutler JA, Higgins M, et al. Prevalence of hypertension in the US adult population results from the Third National Health and Nutrition Examination Survey, 1988-1991. Hypertension. 1995; 25(3):305-13.
36. Franklin SS, Khan SA, Wong ND, Larson MG, Levy D. Is pulse pressure useful in predicting risk for coronary heart disease?: The Framingham Heart Study. Circulation. 1999; 100(4):354-60.
37. L.M. Prisant, M. Moser, Hypertension in the elderly. Can we improve results of therapy? Arch. Intern.Med. 160 (2000) 283–289.
38. Prisant LM, chapter 2; Clinical Hypertension and Vascular Diseases. Hypertension in the Elderly: Humana Press; 2005.p. 12-21.

ANNEXURE I

CONSENT FORM

TITLE OF RESEARCH: "CLINICAL AND ECHOCARDIOGRAPHIC STUDY OF ELDERLY HYPERTENSIVE PATIENTS''

GUIDE : DR. BADIGER SHARANABASAWAPPA

P.G. STUDENT : DR. SHRIHARISH B PUJARI

PURPOSE OF RESEARCH:

I have been informed that the purpose of the research is to study hypertension in elderly with reference to clinical profile and echocardiography.

PROCEDURE:

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

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 to know clinical and echocardiographic changes in elderly hypertensive patients.

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 ask 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 for study at any time.

INJURY STATEMENT:

I understand 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)

ANNEXURE II

"CLINICAL AND ECHOCARDIOGRAPHIC STUDY OF ELDERLY HYPERTENSIVE PATIENTS"

Name: IP. No:

Age: Date of Admission:

Sex: Date of Discharge:

Occupation:

Address:

Chief complaints:

Presenting Complaints

1. Headache
2. Giddiness
3. Fatigue
4. Chestpain
5. Breathlesness
6. Blurring of Vision
7. Syncope
8. Convulsions
9. Palpitation
10. Swelling of Legs
11. Altered Sensorium
12. Any others- mention

History of Hypertension

Age of onset

Duration

Family history of Hypertension

Complication (If any)

Treatment

Past History: (Significant)

History of : Myocardial infarction /Angina

History of : Diabetes

History of : Drug intake

Personal history:

Diet
Appetite
Sleep
Bladder and bowel habits:
Habits

Family history:

GENERAL PHYSICAL EXAMINATION

Pallor:

Icterus:

Cyanosis:

Clubbing:

Lymphadenopathy:

Oedema:

VITAL SIGNS:

Pulse rate:

Blood pressure:

Temperature:

Respiration rate:

Systemic Examination:

- Cardiovascular System
- Respiratory System
- Per abdomen
- Nervous System

PROVISIONAL DIAGNOSIS

INVESTIGATIONS:

BLOOD ROUTINE:

URINE ROUTINE:

ROUTINE BIOCHEMISTRY:

LIPID PROFILE:

ECG:

CHEST XRAY:

FUNDUS EXAMINATION:

ECHOCARDIOGRAPHY:

KEY TO MASTER CHART

Sym	- Symptom
SBP	-Systolic blood pressure
DBP	- Diastolic blood pressure
Htn/h	- Hypertension history
rx/h	- Treatment history
Creat	- Serum creatinine
Na	- Sodium
K	- Potassium
CT	- Cholesterol total
ST	- Serum triglyceride
HDL	- High density lipoprotein
LDL	- Low density lipoprotein
VLDL	- Very low density lipoprotein
LVIDd	- Left ventricular internal diastolic dimension
LVIDs	- Left ventricular internal sysstolic dimension
IVSD	- Inter vernicular septum in diastole
LVPWD	- Left ventricle posterior wall in diastole
LVM	- Left ventricular mass
LA	- Left atrium
LV	-Left atrium
RA	-Right atrium
RV	-Right ventricle
MV	-Mitral valve
AV	-Aortic valve

TV	-Tricuspid valve
PV	-Pulmonary valve
LVDD	-Left ventricular diastolic dysfunction
RWMA	- Regional wall motion abnormality
EF	- Ejection fraction
Cal	- Calcified
Scl	- Sclerosed
Lvh	- Left ventricular hypertrophy
dil	- Dilated
Ant	- Anterior
Inf	- Inferior
Lat	- Lateral
Pos	- Posterior
Sep	- Septal
Emp	- Emphysema
Inc BV	- increased bronchovesicular markings
MR	- Mitral regurgitation
AR	- Atrial regurgitation
TR	- Tricuspid regurgitation
PR	- Pulmonary regurgitation

MASTER CHART

Sl.no	I.P no	AGE	SEX	Sym	SBP	DBP	Htn/h	Rx/h	Smoking	Alcohol	urine	creat	Na	k	CT	ST	HDL	LDL	VLDL	FUNDUS	C-Xray	ECG	LVIDd	LVIDs	IVSD	LVPWD	LVM	LA	LV	RA	RV	MV
1	14365	82	M	+	156	80	6yr	yes	-	-	-	0.9	142	4.3	158	130	45	99	16.4	cataract	normal	LVH	4.65	3.78	0.1	0.1	171.6	dil	lvh	-	-	-
2	22247	75	F	+	200	90	new	no	-	-	-	0.8	140	3.5	216	129	45	145.2	25.8	stage 2	normal	MI	5.99	4.35	1.16	1.16	250	-	dil	-	-	-
3	2274	75	M	+	140	82	7yr	irreg	-	-	-	1.2	137	3.5	145	72	40	96	14.4	stage 2	normal	LVH	6.8	5	1.12	0.8	305.99	dil	dil	-	-	MR
4	4454	70	F	+	182	78	new	no	-	-	1+	1.4	144	3.7	145	78	32	97.4	15.6	stage 1	normal	LVH	4.07	2.73	1.63	1.22	200	dil	lvh	-	-	cal
5	11644	60	M	-	152	80	2yr	yes	+	+	-	1	138	3.4	192	220	30	118	44	normal	normal	LVH	6.69	3.55	1.28	1.28	409.97	-	lvh	-	-	-
6	13505	62	F	+	156	86	new	no	-	-	-	0.9	136	3.9	216	159	38	146	31.8	normal	normal	normal	4.4	3	1.16	1	184.6	-	lvh	-	-	-
7	13622	82	F	-	180	84	new	no	-	-	-	0.9	140	4.1	175	111	3.7	115.8	22.2	stage 1	normal	LVH	3.2	2	1.1	1.3	100	-	lvh	-	-	-
8	14268	80	M	+	144	90	new	no	+	+	-	1.2	141	4.1	123	75	47	61	15	cataract	normal	LVH	5.5	4.4	1	1	213.18	-	dil	-	-	-
9	23894	65	F	+	142	74	5yr	irreg	-	-	-	1.8	129	3.5	145	79	49	80	15.8	stage 2	normal	normal	3.25	2.17	1.33	1.39	149.07	-	lvh	-	-	-
10	24454	72	F	+	210	90	new	no	-	-	1+	1.1	137	4.7	159	89	39	102.2	17.8	stage 2	normal	normal	3.67	2.42	1.16	1.4	150.5	-	lvh	-	-	-
11	26548	64	M	+	150	80	new	no	-	-	1+	0.8	130	3.8	154	61	37	104	12.2	normal	empysema	normal	4.7	4.54	1.1	1.1	187.53	-	lvh	-	-	-
12	27075	70	F	+	162	76	new	no	-	-	-	0.9	140	3.7	161	91	48	94.8	18.2	stage 3	normal	MI	3.52	1.86	1.06	1	100.5	-	-	-	-	-
13	27781	65	M	+	144	70	new	no	+	-	-	1	139	4.6	155	68	32	109	13.6	normal	normal	normal	4.2	3	0.96	0.9	144.12	dil	-	dil	dil	cal
14	15	60	M	+	200	88	1.5yr	irreg	+	+	3+	0.9	138	4.2	180	92	43	118	18.4	stage 1	normal	LVH	5.8	4.7	1.06	1.16	162.5	-	lvh	-	-	-
15	48	65	F	+	152	90	new	no	-	-	1+	1	139	4	170	136	35	107	27.2	stage 1	normal	normal	4.6	4.4	0.96	1	154.49	-	-	-	-	-
16	124	68	F	+	160	76	8yr	yes	-	-	1+	1.2	132	3.8	150	90	44	90	24	stage 3	normal	normal	4.15	3.09	0.37	1.16	93.6	-	-	-	-	-
17	328	63	M	+	152	70	6m	no	+	-	1+	1.2	138	4.2	172	104	40	100	18.2	normal	normal	normal	6.18	5	0.77	1.2	254.62	-	-	dil	dil	-
18	528	70	F	+	146	82	2yr	yes	-	-	1+	1	145	3.5	172	186	45	124	30	cataract	normal	MI	5.8	4.8	0.77	1.06	207.83	-	-	-	-	-
19	598	61	M	-	150	80	new	no	-	-	1+	0.9	141	4.3	201	113	34	144.4	22.8	normal	normal	normal	4.58	3.25	1	1.06	174.23	-	-	-	-	-
20	696	80	F	+	144	78	10yr	yes	-	-	2+	0.7	141	4.5	140	84	30	93.2	16.8	cataract	empysema	MI	4.25	3.48	1.06	1	145.76	-	-	-	-	-
21	759	60	F	+	148	72	1yr	yes	-	-	1+	1.1	134	4.2	160	84	46	90.8	20.2	stage 1	normal	MI	4.18	3.72	1	1	136.2	-	-	-	-	-
22	916	75	M	+	162	84	2yr	yes	-	-	-	1.1	123	6.2	136	78	46	82.4	15.8	stage 2	normal	normal	4.25	2.11	1.26	1.45	215.5	dil	lvh	-	-	-
23	858	80	M	+	146	82	2yr	yes	+	+	1+	1.4	142	5.8	260	248	49	129	49	cataract	normal	MI	6	4.8	1.1	1.26	307	-	dil	-	-	-
24	957	62	F	+	156	74	5yr	yes	-	-	-	1.2	142	3.6	226	164	34	146	36.4	stage 2	empysema	normal	4.5	2.9	0.68	8.7	129.03	-	-	dil	dil	-
25	987	65	M	+	170	80	6m	yes	+	-	1+	1.1	138	5.5	138	154	27	80.2	30.8	normal	normal	MI	5	4.15	1.16	1.66	204.63	-	-	-	-	-
26	1063	65	F	+	170	86	8yr	yes	-	-	-	0.9	133	5	150	69	50	86.2	13.8	stage 3	normal	normal	5.4	3.9	0.8	0.6	121.2	-	-	-	-	MR
27	1129	66	M	+	152	84	1.5yr	yes	-	+	-	1	138	3.7	198	176	40	122.8	35.2	normal	normal	MI	5.6	4.2	0.87	0.87	173.3	-	-	-	-	-
28	1138	79	F	+	172	90	new	no	-	-	1+	1	134	4	165	102	48	96.6	20.4	cataract	inc BV	LVH	4.2	2.5	1.16	1.06	149.5	-	lvh	-	-	-
29	1194	64	M	+	142	88	1m	no	-	-	1+	1	141	4.5	191	208	43	106.4	41.6	cataract	normal	normal	4.54	2.61	1.06	1.16	179.76	-	-	-	-	-
30	1273	60	M	+	144	86	2yr	yes	+	+	-	0.8	136	3.6	139	102	38	80.6	20.2	normal	normal	normal	31.9	2.3	1.16	1.26	120.48	-	lvh	-	-	-
31	1325	70	F	+	146	90	new	no	-	-	1+	1.2	141	4	164	94	42	98	20.4	cataract	inc BV	normal	4.7	3.38	0.77	0.58	125.03	-	-	dil	dil	-
32	1343	60	M	+	162	72	new	no	+	+	-	0.8	136	3.6	140	84	32	96	18	stage 1	normal	normal	3.67	2.9	1.35	1.28	157.5	-	-	-	-	-
33	1832	62	M	-	166	84	4yr	yes	-	-	1+	0.9	140	4	204	186	28	140	28	stage 2	LL con	normal	5.99	4.15	1.06	1.06	255.5	-	-	-	-	-
34	1883	62	M	+	144	88	new	no	-	-	-	1.1	141	4.7	168	135	33	108	27	normal	calcified	MI	4.7	3.8	1.26	1.5	259.63	-	-	-	-	-
35	2119	60	M	+	152	80	2yr	irreg	+	+	2+	1.2	138	4	163	170	43	88	34	stage 2	normal	normal	5.8	4.8	1	0.8	203.5	-	dil	-	-	MR
36	2268	60	F	+	146	82	4m	yes	-	-	-	1.4	140	4.1	174	164	44	98	36	normal	normal	normal	5.8	4.25	0.77	0.87	171.33	-	-	-	-	-
37	2446	65	M	+	142	90	new	no	-	+	-	0.9	138	4.4	140	112	34	60	15	stage 2	inc BV	MI	5.6	4.25	0.87	1.16	150.12	-	-	-	-	-
38	2669	60	M	+	162	72	new	no	+	+	1+	0.8	138	4	115	70	43	58	14	normal	normal	normal	4.48	4.15	0.8	0.9	112.17	-	-	-	-	-
39	2731	65	F	-	144	84	8yr	yes	-	-	-	1	139	4.2	166	150	45	117	30	stage 3	normal	LBBB	4.35	2.8	0.77	0.77	224.07	-	-	-	-	-
40	2737	65	M	+	142	86	new	no	+	-	1+	1.4	136	4.6	185	67	39	132.6	13.4	normal	inc BV	MI	4.9	4.24	1.2	1.06	208.11	-	lvh	-	-	-
41	3202	80	F	-	148	82	new	yes	-	-	1+	0.9	156	3.5	150	168	40	92	32	cataract	empysema	normal	3.9	2.3	0.97	1	168.88	-	-	-	-	cal
42	3432	75	F	-	162	74	6yr	yes	-	-	2+	1.4	141	3.7	115	62	38	64.6	12.4	cataract	LA dil	MI	5.4	4.2	0.8	0.8	134.5	dil	dil	-	-	cal
43	3496	69	M	+	192	88	10yr	yes	+	+	-	1.1	140	4.4	268	184	50	138	38	stage 2	normal	normal	4.15	2.71	0.87	0.77	102.63	-	-	-	-	-
44	4567	65	M	-	168	70	new	no	+	-	-	1	142	3.9	194	201	39	123	38.8	normal	normal	normal	4.01	2.44	1.1	1.22	147.17	-	lvh	-	-	-
45	4574	60	M	+	152	74	new	no	-	-	1+	1.1	132	5.3	204	196	42	128	44	normal	normal	normal	4.65	2.91	0.87	1.05	152.9	-	-	-	-	-
46	4587	61	M	+	142	90	3yr	yes	+	-	-	0.9	133	5.3	118	141	29	60.8	28.2	stage 1	normal	MI	4.7	3.86	1.06	1.16	182.64	-	-	-	-	-
47	4608	68	M	+	190	86	4yr	yes	-	+	-	1.1	143	3.5	146	210	36	86	26	stage 2	normal	normal	4.4	2.62	1	1	137.83	-	-	-	-	-
48	4742	80	F	+	192	78	new	no	-	-	1+	1.4	138	3.9	178	194	40	130	46	cataract	normal	LVH	3.55	2.21	1.1	1.16	116.55	-	lvh	-	-	-
49	5060	74	M	+	146	90	new	no	+	-	1+	1.4	132	5.3	204	196	38	128	44	cataract	normal	MI	3.57	2.42	1.55	1.16	168.81	-	-	dil	dil	-
50	6134	65	F	+	162	76	1yr	irreg	-	-	-	1.3	142	4.7	238	182	40	176	21	stage 2	normal	normal	4.24	3.31	1.22	1.34	188.7	dil	lvh	-	-	MR
51	7210	75	F	+	146	80	6m	irreg	-	-	1+	0.8	139	4.2	217	65	28	176	13	cataract	normal	MI	6.28	5.41	0.87	1.06	254.95	-	dil	-	-	MR

52	7255	60	M	+	142	76	new	no	+	+	-	1.2	140	3.8	146	182	30	108	24	stage 2	normal	MI	50	4.2	0.7	0.9	135.79	-	dil	-	-	-
53	7337	75	F	+	146	72	new	no	-	-	-	0.6	133	4.2	154	104	38	84	28	cataract	normal	AF	3.4	2.2	0.8	0.8	120.5	dil	-	-	-	c/MR
54	7460	70	M	+	154	82	new	no	+	-	-	0.9	138	3.9	258	170	46	122	34	normal	empysema	MI	5.2	4.3	0.8	0.8	145.22	-	dil	-	-	-
55	7487	60	M	+	164	90	10yr	yes	+	+	1+	0.9	142	3.7	180	141	34	118	28	normal	normal	RBBB	4.3	2.6	0.9	0.8	114.16	-	-	-	-	-
56	7635	65	F	+	146	84	3yr	yes	-	-	1+	1.3	142	3.8	192	158	42	128	30	stage 1	normal	LBBB	4.8	3.3	0.9	0.8	137.07	-	-	-	-	-
57	11057	60	M	+	150	90	new	no	+	+	-	1	144	4.3	134	116	40	82	24	normal	normal	normal	4.8	3.2	1	0.8	147.78	-	-	-	-	-
58	12008	60	M	+	142	82	1m	no	-	-	-	0.7	145	4	156	88	42	78	36	normal	normal	normal	3.9	2.4	0.8	0.8	120.5	-	-	-	-	-
59	13706	75	M	+	174	74	8yr	yes	-	+	3+	1.2	148	4.4	202	186	44	118	34	stage 3	normal	LVH,MI	4.8	3.8	1.2	0.8	170.19	-	lvh	-	-	MR
60	2492	72	M	+	184	76	4yr	yes	-	-	1+	0.9	138	3.7	168	78	29	123.4	15.6	stage 2	normal	LVH	5	3.5	0.87	0.9	154.76	-	lvh	-	-	-

AV	TV	PV	LVDD	ISCHEMIA	RWMA	EF	chestpain	dyspnea	fatigue	headache	giddiness	bl.vision	syncope	palptn	p.edema	covulsion	alt.sense	weaknes	others
scl	-	-	-	+	ant	50	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	+	ant	40	-	-	-	-	+	-	-	-	-	-	-	-	-
scl	TR	PAH	+	+	-	50	-	-	-	-	-	-	-	-	-	-	+	+	-
scl	-	-	+	-	-	60	-	-	-	-	-	-	-	-	-	-	+	-	-
-	-	-	+	-	-	60	-	-	-	-	-	-	-	-	-	-	-	+	-
-	-	-	+	+	lat	60	-	-	-	-	-	-	-	-	-	-	-	+	-
cal	-	-	+	-	-	68	+	-	-	-	-	-	-	-	-	-	-	-	-
scl	-	-	+	+	lat	30	+	+	-	-	-	-	-	-	+	-	-	-	-
-	-	-	+	-	-	65	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	-	-	60	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	-	-	65	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	79	+	-	-	-	-	-	-	-	-	-	-	-	-
AR	TR	PR	+	-	-	50	-	-	-	-	+	-	-	-	-	-	+	-	-
-	-	-	+	+	lat	40	-	-	-	-	-	-	-	-	+	-	-	+	-
-	-	-	-	-	ant	60	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	50	-	+	-	-	-	-	-	-	-	-	-	-	-
-	TR	-	-	+	ant	40	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	ant	40	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	55	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	ant	35	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	inf	35	+	-	-	-	-	-	-	-	-	-	-	-	-
c/AS/AR	-	-	+	-	-	50	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	global	30	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	-	-	60	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	ant	35	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	pos,lat	52	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	inf,lat	40	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	-	-	65	+	-	-	-	-	-	-	+	-	-	-	-	-
-	-	-	-	-	-	60	-	-	-	-	+	-	-	-	-	-	-	-	-
-	-	-	+	+	ant	50	+	-	-	+	-	-	-	-	-	-	-	-	-
-	TR	-	+	-	-	55	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	-	-	45	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	57	-	-	+	-	-	-	-	-	-	-	-	-	-
-	-	-	+	+	ant	40	+	-	-	-	-	-	-	-	-	-	-	-	-
-	TR	PAH	-	+	ant	30	+	-	-	-	-	-	-	-	+	-	-	-	-
-	-	-	+	-	-	50	-	+	+	-	-	-	-	-	-	-	-	-	-
-	-	-	+	+	ant	45	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	ant	70	-	+	-	-	+	-	-	-	-	-	-	-	-
-	-	-	-	+	lat	65	-	-	-	-	-	-	-	-	-	-	-	-	+
-	-	-	-	+	ant	55	+	-	-	-	-	-	-	-	-	-	-	-	-
cal	-	-	-	+	sep	50	-	-	+	-	-	-	-	-	-	-	-	-	-
scl	TR	-	+	+	ant	30	-	-	-	-	-	-	-	-	-	-	-	-	+
-	-	-	+	+	sep	64	-	-	-	-	-	-	-	-	-	-	+	-	-
-	-	-	+	-	-	70	-	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	60	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	ant	40	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	+	ant,inf	70	-	-	-	-	-	-	-	-	-	-	+	-	-
scl/AS	TR	-	+	-	-	55	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	inf	60	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	-	-	50	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	sep	30	-	+	-	-	-	-	-	-	-	-	-	-	-

-	-	-	+	+	ant	30	+	-	-	-	-	-	-	-	-	-	-	-	-
scl/AR	TR	PAH	-	-	-	60	-	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	+	ant	25	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	60	-	-	-	+	-	-	-	-	-	-	-	-	-
-	-	-	+	+	sep	50	+	+	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	60	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	-	-	-	60	+	-	-	-	-	-	-	-	-	-	-	-	-
scl/AR	-	-	-	+	ant	30	+	-	-	-	-	-	-	-	-	-	-	-	-
-	-	-	+	+	inf	55	+	-	-	-	-	-	-	-	-	-	-	-	-