A STUDY ON THE CLINICAL PROFILE AND DETERMINANTS OF ISOLATED SYSTOLIC HYPERTENSION

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CERTIFICATE

This is to certify that **A STUDY ON THE CLINICAL PROFILE AND DETERMINANTS OF ISOLATED SYSTOLIC HYPERTENSION** is bonafide work done by **M.SHARMILA** Post Graduate Student, Department Of **INTERNAL MEDICINE, KILPAUK MEDICAL COLLEGE,** Chennai -10 under my guidance and supervision in partial fulfillment of regulations of the Tamilnadu **Dr. M.G.R. MEDICAL UNIVERSITY** for the award of **MD DEGREE BRANCH I, PART II GENERAL MEDICINE** during the academic period from May 2005 To March 2008.

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INTRODUCTION

ISOLATED SYSTOLIC HYPERTENSION

INTRODUCTION

Until the mid 20th century, clinicians' concern was directed mainly to the systolic component of blood pressure[1]. Later, however, when systolic blood pressure was found to be elevated with advancing age and decreased compliance of the arterial wall,[1,2] it began to be considered an inevitable consequence of aging[1-3]. However, there is now compelling evidence from cross sectional, longitudinal, and randomized controlled trials that show that isolated systolic hypertension confers a substantial cardiovascular risk.[4,5] Despite this, it remains under diagnosed and largely untreated.[6] The roots of this lie in a century of over reliance on the importance of diastolic pressure and largely unjustified concerns about the potential adverse consequences of treating systolic pressure.

After the mercury sphygmomanometer was introduced, convention dictated that diastolic pressure was a better determinant of cardiovascular risk than systolic pressure. Systolic pressure was thought to vary considerably throughout the day, and a high pressure was believed to reflect a “strong” left ventricle. This view was perpetuated by the reliance of life

assurance companies on diastolic pressure and the use of diastolic pressure in the early studies of lowering blood pressure. The use of diastolic pressure was further supported by the discovery that essential hypertension is characterized by increased peripheral vascular resistance and therefore raised mean arterial pressure, which more closely correlates with diastolic than systolic pressure. Evidence that systolic pressure is equally, if not more, important than diastolic, particularly in people over 50, was largely ignored.

Although the use of diastolic pressure for risk prediction may be reasonably effective for younger people and people with essential hypertension, data from cohort and intervention studies indicate that it is inappropriate for the over 50s, particularly those with isolated systolic hypertension[7]. Nevertheless, isolated systolic hypertension is not a benign condition. The latest data from the Framingham study, showing, at least in the over 50s, that arterial stiffness is a key determinant of cardiovascular risk.[8] Despite continued reluctance to accept isolated systolic hypertension as a discrete pathological entity, the benefits of treatment are established.[4,5] The relative risk reduction of cardiovascular events in elderly people with isolated systolic hypertension, reported in the latest Cochrane review, is similar to that in younger people.[9] However, as elderly people are at much higher absolute risk of such events, they stand to benefit more from

treatment than younger people. Moreover, elderly people tolerate antihypertensive drugs with few side effects.[9] Yet patients with isolated systolic hypertension remain under-recognized and undertreated.[6]

The latest World Health Organization and Inter- national Society of Hypertension guidelines for the management of hypertension emphasize the importance of arterial stiffness and pulse pressure as predictors of cardiovascular risk and call for further investigation of the prognostic relevance of other indices of arterial stiffness.[10] The enemy today is no longer arterial pressure taken in isolation, but a collection of factors, of which age and doctors' conservatism are among the most important.[11] It is high time that we recognize Isolated Systolic Hypertension as an important clinical condition and update our practicing guidelines accordingly.

AIM

AIM OF THE STUDY

- To study the clinical profile of Hypertensives with Isolated Systolic Hypertension in terms of Symptomatology, History, Clinical features and Laboratory data.

- To study the determinants of Isolated Systolic Hypertension.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

EPIDEMIOLOGY AND RISK

Hypertension is present in more than half of all persons over 60 years of age, regardless of race[10]. The majority of hypertensive patients in this age-group have ISH[11,12]. Isolated systolic hypertension(ISH) has been identified as an entity since long[13]. About 20% of the elderly suffer from ISH[14]. ISH leads to three fold risks of cardiovascular accidents, and 2.2 fold rise in the risk of myocardial infarction[14]. Recent evidence that treating ISH leads to a lowering of cardiovascular morbidity and mortality has aroused keen interest in this entity[13]. There is a paucity of reliable estimates of burden of diseases and distribution of cardiovascular risk factors. Disaggregated data about the burden of disease and risk factors in the community is required for the prevention of cardiovascular disease.

Both the SBP and DBP increase with age in men and women until the early 50s. Between the ages of 54 and 59 years, DBP plateaus and there after falls modestly for the remainder of life. The prevalence of elevated DBP(that is, 90 mm Hg or more), therefore, increases until the mid-50s.Elevated SBP(for example,140 mm Hg or more) is infrequent before the age of 50, begins to raise in prevalence about age 55, and continuous to

increase well beyond the age of 80. Further, the national health and nutrition examination survey (NHANES)-III data demonstrate that for Americans between the ages of 55 and 74. Women have slightly higher prevalence of elevated SBP than men and African-Americans have a higher prevalence than Caucasians. African-American females as a race-sex group have the highest prevalence at 11.3%.

Since 1993 in the United States, systolic BP has been given equal weight to diastolic BP in the diagnostic scheme for hypertension. Elevated systolic BPs have been identified as a major public health problem, for several reasons[15]. The lifetime risk in Framingham for 55 or 65 year old men or women to develop hypertension is 90 percent[16]. Most importantly, for people over age 50 or 60 years, systolic BP is a much better predictor of TOD and future CV and renal events than diastolic BP[15,17] . Overall, each 20 mmHg increase in systolic BP doubles cardiovascular risk[18].

The number of elderly among the populations of the United States and many other countries is rising rapidly. At the beginning of the 20th century, only 4% of the US population was older than 65 years of age. By 2040, the comparable figure is estimated to be 21%. Clearly, therefore, ISH will be an important issue for practitioners in the years to come.

HISTORICAL REVIEW

Diastolic Blood Pressure (DBP), as opposed to Systolic Blood Pressure (SBP) or the combination, became the focus of cardiovascular risk assessment relatively early in the 20th century and of hypertensive treatment trials in 1970s. Clinical trials focusing on diastolic hypertension showed that associated deaths from all causes and strokes could be reduced by vigorous treatment.

A renewed interest in SBP, its elevation, and associated risks was generated first by an analysis of the Build and Blood Pressure study in 1959. That and other studies demonstrated unequivocally that an elevation of SBP was associated with an increase of morbidity and mortality, especially among older people. Some analyses were done with adjustments made for other risk factors. As Fisher points out, in every study where the effect of elevations of DBP and SBP have been compared, elevations of SBP have consistently shown greater associated risk for stroke Coronary Heart Diseases (CHD), and mortality from all causes. Further, the data showed that an elevation of SBP in the presence of normal DBP (that is, Isolated Systolic Hypertension) [ISH] was associated with an increased risk of stroke, cardiovascular disease, and mortality from any cause. Systolic Hypertension

in the Elderly Program (SHEP) was the first clinical trial of antihypertensive therapy to focus on SBP and specifically ISH.

SHEP Trial

SHEP[19] is a multicenter, randomized, double-blind, placebo-controlled trial of treatment for ISH in 4736 persons aged >/=60 years in a community based ambulatory population in tertiary care centers. In the Systolic Hypertension in the Elderly Program (SHEP), treatment with the diuretic agent chlorthalidone for an average of 4.5 years in patients with systolic blood pressure of 160 mm Hg or greater and diastolic pressure below 90 mm Hg resulted in impressive reductions in the incidence of stroke (–36%), coronary heart disease (–27%), and congestive heart failure (–55%), as compared with placebo[20].

Syst-Eur Trial

The Syst-Eur trial[21] was a randomized, double-blind, placebo-controlled trial in elderly patients with ISH aged >/=60. ISH in Syst-Eur was defined as an SBP of 160-219 mm Hg and a DBP of <95 mm Hg. The patients received the dihydropyridine nitrendipine or a placebo (n=2398 vs. 2297). Nitrendipine was supplemented with enalapril, and further with hydrochlorothiazide, if needed, to achieve blood pressure control. The study

was prematurely stopped after the second interim analysis showed a significant decrease in occurrence of strokes in the active treatment group.

In the European Trial in Systolic Hypertension and in the Systolic Hypertension in China Trial, treatment was associated with decreases in the incidence of stroke (–42 and –38%, respectively), coronary heart disease (–30 and –6%), and congestive heart failure (–29 and –58%).[22]

Syst-China Trial

The Syst-China trial[23] was a study of 2394 Chinese patients aged >/=60 with ISH in which an alternative assignments approach of titrated drug therapy or placebo was used. As in Syst-Eur, ISH in Syst-China was defined as an SBP of 160-219 mm Hg with a DBP of <95 mm Hg. The blood pressure goal in the active treatment group was to lower the SBP to <150 mm Hg and to achieve a change in sitting SBP of >/=20 mm Hg.

Nitrendipine was used as initial therapy in the active treatment group and was supplemented, if needed, with captopril, hydrochlorothiazide, or both. The incidences of stroke and other cardiovascular diseases were the main outcome measures. The blinded end point committee reviewed and validated all end points, which were defined as in Syst-Eur.

A meta-analysis of eight trials involving several drug regimens in patients 60 years of age or older with systolic pressure of 160 mm Hg or greater and diastolic pressure below 95 mm Hg showed that antihypertensive therapy administered for an average of 3.8 years reduced total mortality by 13% and mortality due to cardiovascular disease by 18%. In addition, all complications of cardiovascular disease were reduced by 26%, stroke by 30%, and coronary heart disease events by 23%[24].

PATHOPHYSIOLOGY OF ESSENTIAL HYPERTENSION

Systemic Hypertension is a disorder of BP regulation from multitude causes[25]. Control of BP involves complex interactions among the kidneys, the central nervous system (CNS) and peripheral nervous system (PNS), and the vascular endothelium throughout the body as well as a variety of the other organs, such as the adrenal and pituitary glands. The heart is the organ that responds to many of the changes mediated by these systems. It also secretes hormones locally and systemically that help regulate BP. In people genetically predisposed to develop hypertension, an imbalance occurs among the various systems that modulate BP.

The sympathetic nervous system (SNS), the renin angiotensin-aldosterone (RAA) system, vasopressin (VP), nitric oxide (NO), and a host

of vasoactive peptides, including endothelin, adrenomedullin, and others produced by heart and many different cells (endothelial and vascular smooth cells), modulate the responses of the systems and help maintain BP over a range commensurate with optimum physical and mental activity.

PATHOPHYSIOLOGY OF ISOLATED SYSTOLIC HYPERTENSION

Factors that may play a role in the high prevalence of ISH include increased body fat, sedentary lifestyle, and increased sodium intake. A decreased distensibility of the aorta and other large arteries, or the loss of the Windkessel-function., is known to be the main pathophysiologic feature of ISH. Interestingly, systolic blood pressure itself is one of the determinants of aortic distensibility. This may lead to the hypothesis of a vicious circle of high systolic blood pressure decreasing aortic distensibility which in itself increases systolic blood pressure: systolic hypertension begets systolic hypertension.

Increased cardiac output may play a role in ISH. In addition, elderly hypertensive patients tend to have relatively low plasma volume and relatively low levels of rennin and aldosterone. Renal excretion of salt tends to be decreased in these patients, and this probably accounts for relatively

greater salt sensitivity compared with their younger counterparts. Decreased calcium levels resulting from increased calciuria and poor dietary intake may also increase peripheral resistance, leading to hypertension.

MECHANISMS OF VASCULAR STIFFNESS

Vascular stiffening develops from a complex interaction between stable and dynamic changes involving structural and cellular elements of the vessel wall. These vascular alterations are influenced by hemodynamic forces[26] as well as by "extrinsic factors" such as hormones, salt, and glucose regulation. Stiffness is not uniformly disseminated throughout the vascular tree but is often patchy,[27] occurring in central and conduit vessels while sparing the more peripheral arteries.[28]

STRUCTURAL COMPONENTS OF ARTERIAL STIFFENING

The stability, resilience, and compliance of the vascular wall are dependent on the relative contribution of its 2 prominent scaffolding proteins: collagen and elastin. The relative content of these molecules is normally held stable by a slow, but dynamic, process of production and degradation. Dysregulation of this balance, mainly by stimulation of an

inflammatory milieu, leads to overproduction of abnormal collagen and diminished quantities of normal elastin, which contribute to vascular stiffness.[29] Increased luminal pressure, or hypertension, also stimulates excessive collagen production.[30] On gross pathologic vascular specimens, these molecular changes manifest as a doubling to tripling of intima-medial thickness between ages 20 to 90,[31] as well as a hypertrophied vascular smooth muscle layer.[32]

Histological examination of the intima of stiffened vessels reveals abnormal and disarrayed endothelial cells, increased collagen, frayed and broken elastin molecules, infiltration of vascular smooth muscle cells, macrophages and mononuclear cells, and increased matrix metalloproteinases, transforming growth factor (TGF)-ß, intracellular cell adhesion molecules, and cytokines.[33] In addition to vessel wall thickening, aging is associated with a gradual increase in central artery lumen diameter (9% per decade from 20 to 60 years in the ascending aorta),[34] although some recent studies have suggested this does not occur.

CELLULAR ROLE IN VASCULAR STIFFENING

In addition to structural changes, arterial stiffness is strongly affected by endothelial cell signaling and vascular smooth muscle cell (VSMC) tone.

VSMC tone can be modified by mechanostimulation, itself, in part because of cell stretch and changes in calcium signaling, and by paracrine mediators such as angiotensin II,[35] endothelin, oxidant stress, and nitric oxide. Endothelial dysfunction is evidenced clinically by an impaired vasodilatory response to acetylcholine.[36] This stems, in part, from an imbalance between nitric oxide and endothelial-derived hyperpolarizing factor and constricting hormones, and oxygenases (eg, cyclooxygenase, NADPH, and xanthine oxidase).[37] Nitric oxide expression may itself be reduced, and increased expression of a natural nitric oxide synthase (NOS) inhibitor, asymmetrical dimethylarginine, has been linked to vascular stiffening. Bioavailability of nitric oxide is also reduced by activation of reactive oxygen species caused by stress, hormones, and likely AGEs.[38] The formation of peroxynitrite and other highly reactive species results in abnormal vascular tone.

NEUROENDOCRINE SIGNALING AND SALT

Many hormones are known to modulate vascular stiffness. Angiotensin II (AII) stimulates collagen formation, triggers matrix remodeling and vascular hypertrophy, depresses nitric oxide-dependent signaling, increases oxidant stress, and reduces elastin synthesis.[35] In addition, AII stimulates cytokines and growth factors in the matrix that

contribute to an increased inflammatory response. Many of these changes are transduced by AII-stimulated NADPH oxidase and NOS uncoupling.[39] Aldosterone (ALDO) synthesis is primarily controlled by the action of AII on the angiotensin type I receptor, and also promotes vascular stiffness and hypertension by stimulating VSMC hypertrophy, fibrosis, and fibronectin.[40] The action of ALDO is closely tied to endothelin-1; infusion of ALDO increases endothelin-1 production, which has vasoconstrictive and "fibrotic" effects on the vasculature itself.

GLUCOSE, INSULIN, AND VASCULAR STIFFENING

Hyperinsulinemia itself has proliferative effects, because insulin resistance impairs PI3-kinase–dependent signaling responsible for the acute metabolic effects of insulin, yet activity of growth-promoting mitogen activated kinase pathways remains relatively preserved.[41] Impaired glucose tolerance also enhances nonenzymatic glycation of proteins with covalent cross-linking of collagen (AGEs) and alters the mechanical properties of interstitial tissue of the arterial wall.[42] Stiffness is further increased by endothelial dysfunction caused by high LDLs, free fatty acids, endothelin-l, inadequate vasodilatory effects of insulin, or decreased levels of adiponectin and natriuretic peptides.[43] Importantly, increased arterial stiffness in the

metabolic syndrome is not the consequence of fully established diabetes, but rather caused by subtle hormonal and metabolic abnormalities present from the very beginning of an insulin-resistant state.

GENETICS OF VASCULAR STIFFENING

Given the involvement of numerous proteins and hormones in vascular stiffening, it is perhaps not surprising that genetic polymorphisms have been identified that are associated with increased arterial stiffening. In a recent genome-wide scan of the Framingham Heart Study population, DeStefano et al report that having chronically increased arterial pulse pressure has moderate inheritability (0.51 to 0.52). There appears to be minimal overlap between linkage peaks of pulse pressure (PP) versus systolic or diastolic pressure,[44] suggesting that genes contributing to PP variability are separate. Several highly suggestive regions have been identified, some in concordance with genome scans in different cohorts, such as 122 cM region of 15 chromosome, 164 cM region of 8 chromosome (in proximity of ALDO synthase gene), and 70 cM region of 7 chromosome.[45]

VASCULAR STIFFENING PATHOBIOLOGY

Vascular stiffening results in widening of the arterial pulse pressure, which can profoundly influence blood vessel and heart biology. In arteries, the impact is primarily related to changes to mechanical vascular stimulation caused by increased pulsatile shear and pressure.[46] Local regions near bifurcations have more turbulent flow and experience a higher amplitude of oscillatory shear stress with elevated stress, magnifying endothelial dysfunction and vascular disease.[47] In compliant arteries, increased pulsatile perfusion can augment vasodilation, a change linked to enhanced nitric oxide production as well as activation of calcium-sensitive K^+ channels linked to endothelial-derived hyperpolarizing factor. This is further amplified when PP is enhanced in vascular beds dilated by local stimulation of ATP-sensitive K^+ channels,[48] a common mechanism regulating regional flow in the coronary arteries and peripheral vasculature. However, this augmentation of flow by pulse perfusion may require normal vascular distensibility, because reduction of wall compliance appears to block key signaling involved with this response.

CLINICAL IMPLICATIONS OF VASCULAR STIFFENING

Isolated systolic hypertension (defined as systolic blood pressure >140 and diastolic blood pressure <90 mm Hg) and elevated pulse pressure (PP=systolic blood pressure–diastolic blood pressure) are 2 clinical manifestations of decreased vascular distensibility.[49] The prevalence of hypertension increases with age such that >60% of people older than age 65 years are hypertensive with systolic blood pressure >140 mm Hg and/or a diastolic blood pressure >90 mm Hg; older blacks have a higher prevalence of hypertension than do whites in all age groups.[50] However, unlike younger hypertensive subjects in whom systolic blood pressure, diastolic blood pressure, and MAP are all risks for cardiovascular events,[51] Isolated Systolic Hypertension and elevated PP pose more significant risks for strokes, myocardial infarctions, heart failure, and overall mortality in older adults.[52] This difference in risk implies a different pathophysiological mechanism for hypertension in younger versus older individuals and perhaps a different therapeutic approach.[53] "In fact, it is reported that every 2-mm Hg increase in systolic blood pressure increases the risk of fatal stroke by 7% and fatal coronary heart disease event by 5%."[54]

DIAGNOSIS OF HYPERTENSION

Objective measurements were made easier by the instruments of Janeway and Korotkoff, who characterized the sounds heard when the stethoscope was placed over the compressed artery in 1905. The terminology introduced by Korotkoff is still used today: systolic BP is recognized when clear and repetitive tapping sounds are heard; diastolic BP is recorded when the sounds disappear. An exception is recognized among patients who have audible sounds even down to zero millimeters of mercury; the "muffling" of the sounds (Korotkoff phase IV) is then recorded before the zero[55].

TECHNIQUES OF MEASURING BLOOD PRESSURE

To accurately measure BP, the deflation rate of the column of mercury should be 2 to 3 mm Hg/s. the lower rate of deflation should be used in for persons with heart rate less than 72 beats per minute(bpm); the more rapid deflation is appropriate only for those with resting tachycardia. If the precision of measurement is to be atleast 2mm Hg, the observer should have the opportunity to hear atleast one Korotkoffs sound at each 2mm Hg gradation of the mercury column. Thus, the proper deflation rate depends on the heart rate of the subject and is unlikely to be more than 3mm Hg/s if a precise BP measurement is desired.

It is usual for a single BP measurement to be an accurate indicator of future CV risk; multiple measurements made on different occasions are more likely to be helpful in deciding whether a particular person ought to have his or her BP lowered.

HOME BLOOD PRESSURE MEASUREMENTS

Home BP readings are typically lower (by an average of about 12/7 mm Hg) than measurements taken in the traditional medical environment, even in normotensive subjects[56]. Home readings tend to be better correlated with both the extent of TOD and the risk of future mortality than are readings taken in the physician's office[57]. Home readings can be helpful in evaluating symptoms suggestive of hypotension, especially if the symptoms are intermittent or infrequent. Home BP readings should be interpreted cautiously, carefully and conservatively[58]. There are no long-term clinical trials that based all treatment decisions solely on home readings, but several reports show benefit from supplementing office BP measurements with home readings[59].

AMBULATORY BLOOD PRESSURE MONITORING(ABPM)

ABPM makes it possible to measure BP routinely during sleep and has reawakened interest in the circadian variation of HR and BP. Most

normotensives and perhaps 80 percent of hypertensives have at least a 10 percent drop in BP during sleep compared with the daytime average. Although there may be some important demographic confounders (blacks and the elderly have less prominent "dips"[60]), several prospective studies have shown an increased risk of CV events (and proteineria in type 1 diabetics[61]) among those with a nocturnal "nondipping" BP or pulse pattern[62]. However, there is concern, based on several Japanese studies, that elderly persons with more than a 20 percent difference between nighttime and daytime average BPs ("excessive dippers") may suffer unrecognized ischemia in "watershed areas" (of the brain and other organs) during sleep of their BP declines below the autoregulatory threshold[63-65].

During the last 20 years, research has demonstrated an important correlation between ABPM readings and the prevalence and extent of TOD in hypertensives. Compared with "casual" BP measurements (obtained in the health care provider's office), ABPM measurements clearly are a better predictor of LVH, cardiac function, and overall scores summing optic, carotid, cardiac, renal, and peripheral vascular damage resulting from elevated BP. Ambulatory BP monitoring may also be useful in identifying "white coat normotensives". In the first published study of outcomes in central Italy, ABPM was the best predictor of future CV events; "nondipper

hypertensives" had approximately three times the risk of hypertensives whose BP was ≥10 percent lower at night compared to daytime ("dippers"). Continued follow-up and refinements in these analyses come to the same conclusions.

WHITE COAT HYPERTENSION

The name white coat hypertension has been given to the situation in which BP measurements outside the health care setting are considerably lower than those in it, even though the "White Coat" itself is unlikely to be the only factor that increases BP. Even in the largest and longest experience, the risk of future CV events did not differ between white coat and sustained hypertensives when both were treated with antihypertensive medications[66].

PSEUDO HYPERTENSION

Osler's manoeuver, the sign of cuff artifact due to arterial stiffness described by Messerli *et al*[67] and so named because Osler, in his 1892 text, indicated that he mistrusted the BP reading in patients with stiff arteries, in whom the radial artery was still palpable even though the cuff had been inflated above the systolic pressure. Messerli *et al* called patients with that finding 'Osler manoeuver positive' (OM^+), and found discrepancies between

the cuff and intra-arterial diastolic pressure in such patients, ranging from 10 to 54 mm Hg. In the cohort of patients being screened for SHEP (the Systolic Hypertension in the Elderly Program) studied by Wright and Looney, 243 of 3387 patients (7.2%) were OM^+. They suggest that Osler's manoeuver Osler manoeuver could be used to identify patients with pseudohypertension.

Pseudohypertension is a problem in some elderly patients with stiff arteries. Its prevalence is still unclear; the problem occurs in approximately half of elderly patients with diastolic pressure > 100 mm Hg but no end-organ disease. Belmin *et al*[68] found that 5.8% of elderly geriatric in-patients were OM^+, and Wright and Looney[69] found that 7.2% of patients screened for SHEP at their centre were OM^+; however, not all those patients will have pseudohypertension.

Osler's manoeuver may be used to raise clinical suspicion of a large cuff artifact, but further evaluation is required to establish the true BP level; new approaches to sorting this out are ultrasound determination of arterial closing pressure, and BP measurement with a finger cuff. The diagnosis should be suspected in elderly patients with resistant high diastolic pressures and no end-organ disease, who complain of light-headedness when the BP is treated to levels that do not explain the symptoms.

EVALUATION OF THE HYPERTENSIVE PATIENT

Six key issues must be addressed during the initial office evaluation of a person with elevated BP readings:

- Documenting an accurate diagnosis of hypertension.
- Defining the presence or absence of TOD related to hypertension.
- Screening for other CV risk factors that often accompany hypertension.
- Stratifying risk for CVD.
- Assessing whether the person is likely to have an identifiable cause of hypertension (secondary hypertension) and should have further diagnostic testing to confirm or exclude the diagnosis.
- Obtaining data that may be helpful in the initial choice and subsequent choice of therapy.

ROUTINE EVALUATION IN ALL HYPERTENSIVE PATIENTS

The recommendations of JNC 7 and other national and international expert panels limit the number of initial tests and the expense related to the for the routine evaluation of hypertensive patients[70]. Those that are used in assessing the presence or absence of TOD include physical examination,

blood urea nitrogen (BUN)/creatinine, electrolytes, urinalysis, and an electrocardiogram (ECG). Assessing the number of CV risk factors can be accomplished with the medical history, chemistry panel (glucose, lipid profile) and urinalysis.

BLOOD PRESSURE CLASSIFICATION IN ADULTS (JNC-7)

By Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure

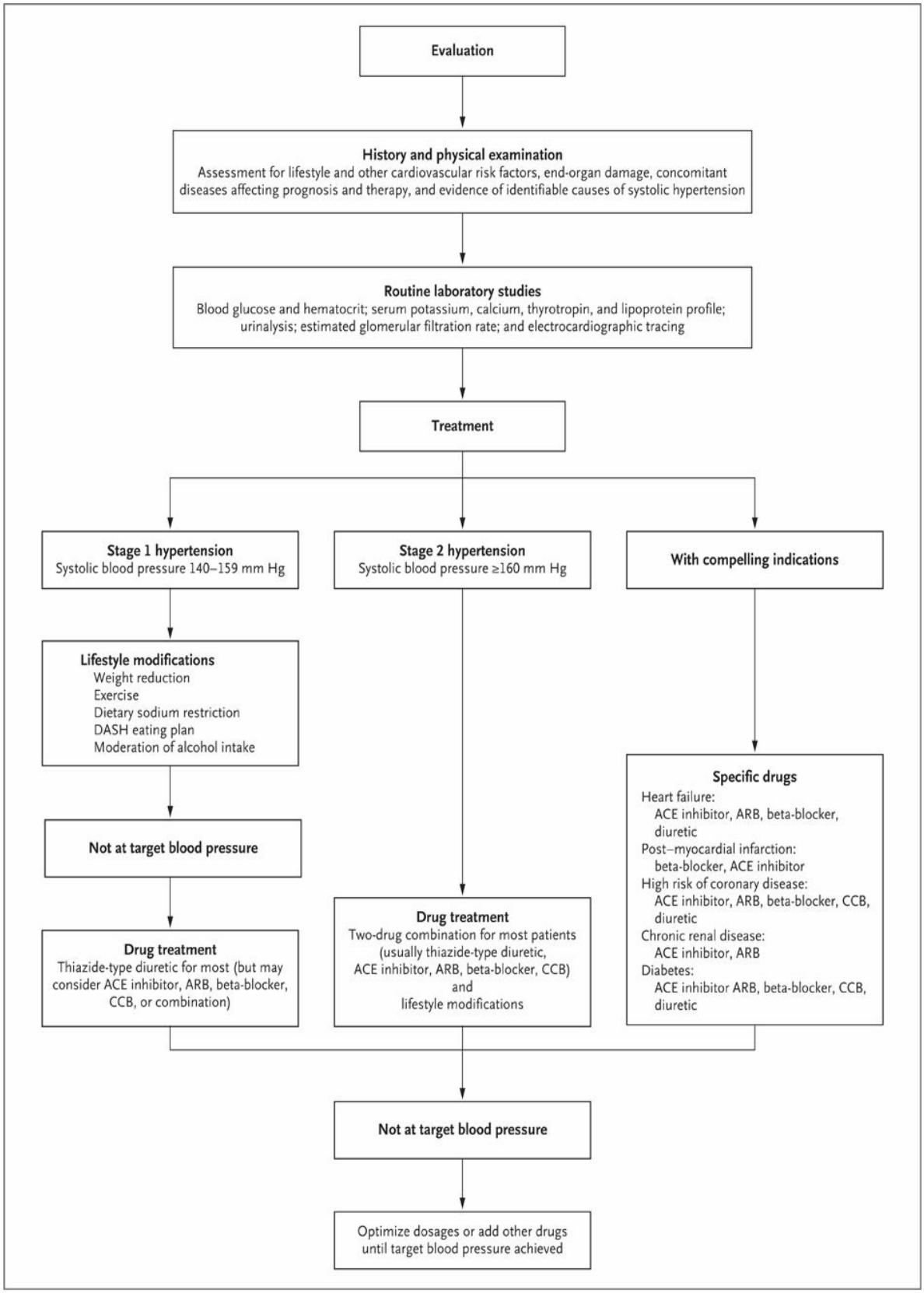
BP Classification	SBP mm Hg	DBP mm Hg
Normal	<120	<80
Pre hypertension	120-139	80-89
Stage I hypertension	140-159	90-99
Stage II hypertension	≥160	≥100

EVALUATION IN ISOLATED SYSTOLIC HYPERTENSION

The initial evaluation of the patient with systolic hypertension should include an assessment for the presence of other cardiovascular risk factors, end-organ damage, concomitant diseases affecting prognosis and treatment, identifiable causes of hypertension (e.g., hyperthyroidism) and potentially contributing lifestyle factors (diet and exercise).[70]

MANAGEMENT

The therapeutic approach and goals for isolated systolic hypertension are similar to those recommended for most other types of hypertension. The recommended target level of blood pressure is below 140/90 mm Hg, except in patients with diabetes or chronic renal disease, for whom a lower goal (130/80 mm Hg or lower) is advised.


Evaluation
History and physical examination
Assessment for lifestyle and other cardiovascular risk factors, end-organ damage, concomitant diseases affecting prognosis and therapy, and evidence of identifiable causes of systolic hypertension
Routine laboratory studies
Blood glucose and hematocrit; serum potassium, calcium, thyrotropin, and lipoprotein profile; urinalysis; estimated glomerular filtration rate; and electrocardiographic tracing
Treatment
Stage 1 hypertension
Systolic blood pressure 140–159 mm Hg
Stage 2 hypertension
Systolic blood pressure ≥160 mm Hg
With compelling indications
Lifestyle modifications
Weight reduction
Exercise
Dietary sodium restriction
DASH eating plan
Moderation of alcohol intake
Not at target blood pressure
Drug treatment
Thiazide-type diuretic for most (but may consider ACE inhibitor, ARB, beta-blocker, CCB, or combination)
Drug treatment
Two-drug combination for most patients (usually thiazide-type diuretic, ACE inhibitor, ARB, beta-blocker, CCB) and lifestyle modifications
Specific drugs
Heart failure:
ACE inhibitor, ARB, beta-blocker, diuretic
Post–myocardial infarction:
beta-blocker, ACE inhibitor
High risk of coronary disease:
ACE inhibitor, ARB, beta-blocker, CCB, diuretic
Chronic renal disease:
ACE inhibitor, ARB
Diabetes:
ACE inhibitor ARB, beta-blocker, CCB, diuretic
Not at target blood pressure
Optimize dosages or add other drugs until target blood pressure achieved

LIFESTYLE CHANGES

The lifestyle modifications recommended for patients with isolated systolic hypertension are the same as those for patients with other forms of hypertension, including weight reduction, restriction of dietary sodium, adoption of the Dietary Approaches to Stop Hypertension (known as DASH) eating plan (a diet rich in fruits, vegetables, and low-fat dairy products and low in saturated and total fat), increased physical activity, and moderation of alcohol intake (no more than the equivalent of two drinks per day for men and one for women). These interventions not only reduce blood pressure but also favorably affect other risk factors for cardiovascular disease, such as dyslipidemia, abdominal obesity and diabetes that characterize the metabolic syndrome.[71]

DRUG TREATMENT

Five major classes of antihypertensive drugs are most useful: diuretics, β-adrenergic blockers, angiotensin-converting–enzyme (ACE) inhibitors, angiotensin-receptor blockers, and calcium-channel blockers. Each has been shown in clinical trials to reduce cardiovascular events.[72] when used in recommended dosages; their mean effects on blood pressure are similar,[73] although individual patients may have different responses to

each drug. In approximately two thirds of patients with hypertension, two or more drugs will be required to achieve target blood-pressure levels.

The current Joint National Committee guidelines[70] recommend thiazide diuretics as initial drug therapy for most patients with hypertension, on the basis of their proven efficacy in reducing blood pressure and cardiovascular complications in clinical trials and their low cost. Other antihypertensive medications are preferred initially when there are certain coexisting conditions. For example, in patients with hypertension and chronic kidney disease, compelling evidence from clinical trials supports the use of either an ACE inhibitor or an angiotensin-receptor blocker,[74] and for patients who have had myocardial infarction or heart failure, a beta-blocker and an ACE inhibitor are preferred.[75] Elderly men with both hypertension and benign prostatic hypertrophy are often treated for urinary symptoms with an α-1–receptor antagonist, which can help control the hypertension but may increase the risk of orthostatic hypotension. Nevertheless, despite some important differences between antihypertensive medications, the major benefits of therapy are related to the reduction of blood pressure rather than to other specific drug actions.

Thiazide-type diuretics can induce carbohydrate intolerance and diabetes,[76] effects that are greater in patients in whom hypokalemia develops.[77] However, the clinical importance of such adverse effects is uncertain, given clinical trial data showing that thiazides are at least as effective as other drug classes in reducing the risk of complications from cardiovascular disease.[78] The current debate over initial drug use notwithstanding, most patients with hypertension should end up receiving a diuretic as part of their regimen, since more than one drug is usually required to achieve blood-pressure control and since diuretics complement the action of the other drugs so well.

The use of beta-blockers as first-line therapy for elderly patients with hypertension has been questioned recently. A meta-analysis of intervention trials for hypertension showed a 16% higher incidence of stroke among patients treated with traditional beta-blockers (primarily atenolol) than among those treated with other antihypertensive medications.[79] The lesser benefit from beta-blockers could be related to a smaller reduction in blood pressure. In a recent study of patients treated with atenolol, blood pressure measured by standard cuff techniques overestimated the pressure reduction by 4.5 mm Hg as compared with aortic pressure calculated from applanation tonometry and radial-artery waveforms[80]; in contrast, with a calcium-channel

blocker, ACE inhibitor, or diuretic agent, the effects on central aortic- and brachial-artery pressures were similar.[81]

Initial therapy with beta-blockers in elderly patients should probably be limited to those with compelling indications, such as coronary heart disease, myocardial infarction, congestive failure or certain arrhythmias. No data is available yet on whether such restrictions should apply to the newer beta-blockers with peripheral vasodilator properties.

STRATEGIES FOR IMPROVING BLOOD-PRESSURE CONTROL

Inertia on the part of physicians and a reluctance to treat systolic hypertension are important factors limiting optimal control of blood pressure.[82] Many physicians do not give adequate doses of antihypertensive medications or do not use a combination of drugs to achieve the target pressure. Factors that adversely affect adherence to treatment include inadequate patient education; lack of physician empathy and social support; the presence of coexisting diseases; complex dose regimens; problems with transportation of the patient and the cost of medications. Participation by ancillary staff, including nurse clinicians, physicians' assistants, and pharmacists, has been shown to be effective in improving blood-pressure

control.[83] Most elderly patients tolerate antihypertensive medications well, although a low starting dose and a gradual rate of increase in the dose (e.g., every 2 to 4 weeks) is prudent, particularly in frail and relatively immobile patients and in patients with diabetes, since both groups are at increased risk for orthostatic hypotension and associated falls.[84]

GUIDELINES

The Joint National Committee guidelines, which have been endorsed by several professional organizations, including the American Medical Association, the American Heart Association, and the American Society of Hypertension, recommend thiazide-type diuretics as initial drug therapy for most patients with isolated systolic hypertension unless there are specific contraindications for their use. Compelling indications discussed above warrant initiation of therapy with an ACE inhibitor, angiotensin-receptor blocker, calcium-channel blocker, or beta-blocker. The addition of a drug from another class is required if the target blood pressure is not achieved.

The joint guidelines of the European Society for Hypertension and the European Society of Cardiology do not give preference to diuretics and recommend any of the five major classes of antihypertensive drugs for first-line therapy.[85] Recent guidelines from Great Britain argue against the use of

both diuretics and beta-blockers for initial therapy and favor ACE inhibitors, angiotensin-receptor blockers, or calcium-channel blockers.[86] Despite some differences in recommendations, all of these guidelines emphasize that the major benefits of therapy are related to lowering blood pressure and controlling hypertension.

MATERIALS AND METHODS

MATERIALS AND METHODS

Materials–Patients with Isolated Systolic Hypertension who attended Hypertension OP under institution of Kilpauk Medical College and Govt. Hospital were taken up.

Study population included patients belonging to low socioeconomic urban or semi urban city of Chennai .

Isolated Systolic Hypertension was defined as per JNC-7 guidelines

The study group included

1. Newly detected hypertensives with Isolated Systolic Hypertension.
2. Control group with essential hypertension (Systolic and Diastolic Hypertension).

Following group were excluded from the study.

1. Patients with secondary form of hypertension
2. Patients with Pre hypertension.

Detailed evaluation of patients in terms of symptomatology, History, Clinical examination, and Laboratory data were carried out. The proforma used for the same is attached

Once diagnosed, they were put on appropriate management. The treatment and outcome is not included in the study.

DEFINITIONS

DEFINITIONS

Essential Hypertension

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure(2003) defines Essential Hypertension as Systolic Blood Pressure consistently 140 mmHg or greater, and diastolic blood pressure consistently 90 mmHg or greater in the absence of any evident cause.

Isolated Systolic Hypertension

Isolated Systolic Hypertension (ISH) is defined as elevated Systolic Blood Pressure above 140 mm Hg in conjunction with Diastolic Blood Pressure below 90 mm Hg.

Systolic Blood Pressure

Systolic Blood Pressure is defined as the maximum arterial pressure during contraction of the left ventricle of the heart.

Diastolic Blood Pressure

Diastolic Blood Pressure is defined as the minimum arterial pressure during relaxation and dilatation of the left ventricle of the heart.

Pulse Pressure

Pulse Pressure is defined as the change in blood pressure seen during contraction of the heart. It is the **Systolic Pressure minus the Diastolic Pressure.**

Mean Arterial Pressure

The Mean Arterial Pressure (MAP) is defined as the average arterial pressure during a single cardiac cycle. It is the **sum of Diastolic Blood Pressure and one-third of Pulse Pressure**.

RESULTS

RESULTS AND ANALYSIS

- 115 patients with isolated systolic hypertension were diagnosed among patients attending hypertension clinic and were included in study group during the period Aug 2006 - Jul 2007
- 50 patients with essential hypertension group were attending hypertension clinic were included in the study to compare and analyze the determinants of isolated systolic hypertension.

ANALYSIS OF CLINICAL PROFILE OF ISOLATED SYSTOLIC HYPERTENSION

- Mean age of study population - **61.87 yrs**.
- Mean BMI – **25.39 kg/m^2**
- Mean waist circumference – **85.57 cm**

Anthropometric analysis states that most of the patients were over weight individuals. Most of them had increased waist circumference putting them to increased cardiovascular risks per modified ATP III criteria (≥85 cm) appropriate for Indians.

Commonest Symptoms	**No of Patients**	**%**
Dyspnoea	42	36.5%
Giddiness	22	19.1%
Angina	16	13.9%
Edema	11	9.5%
Headache	9	7.8%
Oliguria	5	4.3%

37 Patients **(32.17 %)** had h/o DM

13 Patients **(11.3 %)** had family history of Hypertension

13 Patients **(11.3 %)** had pedal edema

63 Patients **(54.7 %)** were overweight BMI (25-29.9)

4 Patients **(3.4 %)** were obese (>30)

11 Patients **(9.5 %)** had I fundus changes

1 Patients **(0.8 %)** had II fundus changes

34 Patients **(29.5 %)** had LVH and Hypokinetic changes in echocardiogram

10 Patients **(8.69 %)** had abnormal kidney echoes

37 Patients **(32.17 %)** had Hb % < 10 gm/day

37 Patients **(32.17 %)** had casual glucose > 200

9 patients **(7.8 %)** had serum calcium level >10.5 mg/dl

6 patients **(5.2 %)** had serum uric acid level >6 mg/dl

2 patients **(1.7 %)** had serum sodium level >144 Meq/dl

2 patients **(1.7 %)** had serum potassium level >5.5 Meq/dl

Total Cholesterol	**n - no. of patients**	
Borderline (200-239 mg/dl)	n = 42	36.5%
Undesirable >240 mg/dl	n = 34	29.56%

LDL	**n - no. of patients**	
Borderline (130-159 mg/dl)	n = 30	26.08%
Undesirable >160 mg/dl	n = 40	34.78%

HDL	**n - no. of patients**	
Borderline (40-60 mg/dl)	n = 82	71.30%
Undesirable >40 mg/dl	n = 30	26.08%

TGL	**n - no. of patients**	
Abnormal (>160) mg/dl)	n = 35	30.43%

ISOLATED SYSTOLIC HYPERTENSION

SUMMARY STATISTICS FOR CONTINUOUS VARIABLES

Sl. No	ITEMS	MEAN	MEDIAN (RANGE)
1	AGE	62.41	62 (42-77)
2	BMI	25.39	26.5 (17.32)
3	SYS BP	168.92	168 (144-280)
4	DBP	79.87	80 (70-90)
5	PULSE PRESSURE	88.38	88 (56-126)
6	MAP	109.31	109.3 (94.6-123.3)
7	HB	11.05	10.5 (7.4-12.8)
8	ESR	17.42	14 (5-92)
9	BL SUGAR	161.43	125 (71-312)
10	UREA	30.45	28 (15-112)
11	CREATININE	0.95	0.9 (0.4 - 3.0)
12	NA+	137.53	138 (130-147)
13	K+	4.2	4.2 (3.0 - 5.7)
14	TOTAL CHOLES	218.9	219 (126-316)
15	TGL	139.66	132 (60-240)
16	HDL	46.20	46 (33-66)
17	VLDL	27.84	27 (12-46)
18	LDL	35.01	142 (42-220)
19	SE CALCIUM	9.46	9.4 (8.3-10.8)
20	SE URIC ACID	3.73	3.4 (2.0 - 11.0)

ANALYSIS OF THE DETERMINANTS OF ISOLATED SYSTOLIC HYPERTENSION

TABLE – 1: AGE DISTRIBUTION - ANALYSIS

Age	ISH	EHT
<50 yrs	6(5.2%)	4(8%)
51-60 yrs	27(23.4%)	23(46%)
61-70 yrs	69(60%)	20(40%)
>71	13(11.3%)	3(6%)
	115	50

TABLE – 2: AGE DISTRIBUTION – ANALYSIS

Category	No. of Patients	Mean	S.E of Mean
ISH	115	62.41	0.5857
EHT	50	59.26	0.94450

P = 0.00411 'P' value is significant

Interpretation:

Among the 115 patients with Isolated Systolic Hypertension studied, the age incidence was highest in the 61- 70 year age group (60%). This was followed by 51- 60 year age group (23.4%).

TABLE – 3: SEX DISTRIBUTION – ANALYSIS

Category	**ISH** (no. of patients) %	**EHT** (no. of patients) %
Male	(39) 33.9%	(26) 52%
Female	(76) 66.08%	(24) 48%
	115	50

P = 0.028877 'P' value is significant

Interpretation:

Among the 115 patients with Isolated Systolic Hypertension studied, females form the majority. This is also true when compared with 50 patients with Essential Hypertension

TABLE – 4: SYMPTOM – ANALYSIS

Isolated Systolic Hypertension

SYMPTOMS	PERCENTAGE
Shortness of Breath	36.5%
Giddiness	19.1%
Angina	13.9%
Edema	9.5%
Headache	7.8%

Interpretation:

Among the 115 patients with Isolated Systolic Hypertension studied, 36.5% complained of shortness of breath followed by giddiness (19.1%).

TABLE – 5: SMOKING – CORRELATION

Category	ISH No. of Patients	EHT No. of Patients
Non-Smokers	88	39
Smokers	27	11
	115	50

P = 0.140682 'P' value is not significant

Interpretation:

Among the 115 patients with Isolated Systolic Hypertension studied, 27 (23.4%) cases have history of smoking

Among the 50 patients with Essential Hypertension 11 (22%) cases have history of smoking when compared smoking is not a major factor in the occurrence of Isolated Systolic Hypertension.

TABLE – 6: BODY MASS INDEX (BMI) – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	25.39913	0.30117
EHT	50	24.862	0.45719

P = 0.327816 'P' value is not significant

Interpretation:

The mean BMI in the ISH group is 25.39913

The mean BMI in the EHT group is 24.862

The baseline BMI is not a major factor in the occurrence of Isolated Systolic Hypertension.

TABLE – 7: SYSTOLIC BLOOD PRESSURE (SBP) – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	168.9217	1.708647
EHT	50	176.64	2.205329

P = 0.010277 ‘P’ value is significant

Interpretation:

Among the 115 patients with Isolated Systolic Hypertension studied, the mean SBP is 168.9217. Among the 50 patients with Essential Hypertension, the mean SBP is 176.64.

On analyzing, the Systolic Blood Pressure has a positive correlation with the incidence of Isolated Systolic Hypertension

TABLE – 8: DIASTOLIC BLOOD PRESSURE (DBP) – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	79.87	0.6294
EHT	50	103.72	1.093222

P = 0.0000 ‘P’ value is significant

Interpretation:

The mean DBP in the ISH group 79.87

The mean DBP in the EHT group 103.72

Thus, the diastolic pressure has a definite correlation in the occurrence of Isolated Systolic Hypertension.

TABLE – 9: PULSE PRESSURE (PP) – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	88.382	1.6368
EHT	50	72.92	2.4771

P = 0.00001 'P' value is significant

Interpretation:

The mean pulse pressure is increased in the Isolated Systolic Hypertension group compared to the Essential Hypertension group and is statistically significant. Hence, the Pulse Pressure determines the occurrence of Isolated Systolic Hypertension.

TABLE – 10: MEAN ARTERIAL PRESSURE (MAP) – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	109.31	0.5997
EHT	50	128.19	1.0441

P = 0.0000 'P' value is significant

Interpretation:

The mean MAP (Mean Arterial Pressure) is reduced in Isolated Systolic Hypertension group when compared to the Essential Hypertension group and is statistically significant. Hence, the MAP determines the occurrence of Isolated Systolic Hypertension.

TABLE – 11: BLOOD GLUCOSE – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	161.4348	6.96
EHT	50	188.9	1212716

P = 0.039595 'P' value is significant

Interpretation:

The mean blood glucose level in the ISH group is 161.4348

The mean blood glucose level in the EHT group is 188.9

Thus, the increase in blood glucose level favors the occurrence of Essential Hypertension rather than Isolated Systolic Hypertension.

TABLE – 12: TOTAL CHOLESTEROL – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	218.9043	4.9858
EHT	50	229.14	5.5650

P = 0.225345 'P' value is not significant

Interpretation:

The mean total cholesterol in the ISH group is 218.9043

The mean total cholesterol in the EHT group is 229.14

Thus, the baseline cholesterol level does not correlate with the occurrence of Isolated Systolic Hypertension.

TABLE – 13: SERUM SODIUM – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	137.539	0.4143
EHT	50	137.6	0.6546

P = 0.936452 'P' value is not significant

TABLE – 14: SERUM POTASSIUM – CORRELATION

Category	No. of Patients	Mean	95% C.I	
			LCL	UCL
ISH	115	4.201	4.077	4.326
EHT	50	4.148	3.948	4.347

P = 0.642373 'P' value is not significant

Interpretation:

Thus, the baseline serum sodium and serum potassium level does not correlate with the occurrence of Isolated Systolic Hypertension.

TABLE – 15: SERUM CALCIUM – CORRELATION

Category	No. of Patients	Mean	95% C.I	
			LCL	UCL
ISH	115	9.466	9.360	9.572
EHT	50	9.772	9.598	9.945

P = 0.002368 'P' value is significant

Interpretation:

The increase in serum calcium level favors the occurrence of Essential Hypertension rather than Isolated Systolic Hypertension.

TABLE – 16: SERUM URICACID – CORRELATION

Category	No. of Patients	Mean	S.E of Mean
ISH	115	3.737	0.1440
EHT	50	4.226	0.3031

P = 0.100521 'P' value is not significant

Interpretation:

Thus, the baseline serum uricacid level does not correlate with the occurrence of Isolated Systolic Hypertension.

DISCUSSION

DISCUSSION

Isolated Systolic Hypertension is a common disorder in the elderly, carrying with it a high risk of cardiovascular morbidity and mortality

Analyzing age distribution ISH is more common in 61-70 years age group (60%). ISH incidence increases from 50 years of age and maximum incidence is 61-70 years age group. This suggests that increasing age determines the occurrence of Isolated Systolic Hypertension. This is similar to the Framingham Heart Study analysis by Van B Welking, Al Belanger MA which shows ISH in 57.4% in men > 65 years[87]. This study also represents that ISH is the frequent form of hypertension among older individuals.

Among the 115 patients with Isolated Systolic Hypertension studied females (66.08%) form the dominant group then the males (33.9%). This is again confirmed by a comparative study of patients with Essential Hypertension and was statistically significant using a paired 't' test with a 'P' value of 0.028877. The Chennai Urban Rural Epidemiology Study (CURES-52) states that women had a higher prevalence of isolated systolic blood pressure compared to men.

The SHEP (Systolic Hypertension in the Elderly Program) similarly shows a high prevalence of ISH in older adults especially in older women[88] . Moreover, after menopause, there is a sharp increase in the prevalence of hypertension in women to levels that equal or surpasses that of men. This is because of the protection afforded by the ovarian hormones to the pre-menopausal women[89] . These may be the reasons for higher prevalence of Isolated Systolic Hypertension among older women in our study.

The chief symptom in the Isolated Systolic Hypertension in our study is shortness of breath (36.5%) followed by giddiness (19.1%). Christopher J. Bulpitt, Astrid E. Fletcher in a study based on the SYST-EUR Trial also states that unsteadiness, nocturia and headache occur in excess in untreated Isolated Systolic Hypertension.

The base line mean BMI in the ISH group is 25.39913 and mean BMI in the Essential Hypertension group is 24.862. When analyzed statistically using paired 't' test, it is not significant. The lack of association of baseline BMI with the incidence of ISH in our study is supportive of a cross-sectional Mexican survey,[90] which failed to show a significant association of excess body weight with ISH. Jose R. Pio, BS; Nathan D. Wong, PhD in a study also quotes that BMI at baseline was not a predictor of ISH.

Analyzing the Systolic Blood Pressure (SBP) in the Isolated Systolic Hypertension group and in the Essential Hypertension group it was found that SBP is positively related to the development of ISH. This is also statistically significant using a paired 't' test with a 'P' value of 0.010277.

Studies of elderly subjects with isolated systolic hypertension showed

.that increased input impedance (large artery stiffness and early pulse wave reflection) 'predominated' over increased vascular resistance.[91] In addition, a computer simulation of a modified Windkessel model for geriatric isolated systolic hypertension indicated that vascular resistance increased by only 25%, whereas there was a 50% to 75% increase in input impedance secondary to large artery stiffness and early wave reflection.[92] These conclusions are further supported by the observed decrease in DBP and increase in SBP after age 60 in the Framingham subjects.

The mean DBP in the Isolated Systolic Hypertension group is 79.87 and in the Essential hypertension group is 103.72 which is statistically significant with the 'P' value of 0.000. The decline in DBP seen in the elderly is probably the result rather than the cause of the disease process. Age-related stiffening of the aorta is associated with a decreased capacity of the elastic reservoir and hence a greater peripheral runoff of stroke volume during systole. The exaggerated fall in DBP seen in elderly hypertensive

subjects suggests a process of transmural pressure-induced arterial wall damage resulting in large artery stiffness.[93] The most likely explanation, therefore, for the fall in DBP after age 60 years is increased large artery stiffness.[94] Our study also supports the concept of an interaction between aging and hypertension in the progressive fall of DBP and rise of SBP.

The mean pulse pressure is increased in the Isolated Systolic Hypertension group compared to the Essential Hypertension group in our study. This is statistically significant using paired 't' with a 'P' value of 0.00001. This is similar to the study quoted by Nichols WW, O'Rourke MF. based on the Framingham Heart Study. The most plausible explanation given by them for both the late rise in Pulse Pressure and fall in Diastolic Blood Pressure is an increase in the large artery stiffness caused by intrinsic structural abnormalities[95].

The mean MAP (Mean Arterial Pressure) is reduced in Isolated Systolic Hypertension group when compared to the Essential Hypertension group in our study. This is similar to the study quoted by Messerli FH, Sundgaard-Risse K, Ventura HO state that the leveling off of MAP after age 50 to 60 years in all SBP groups in the above study suggests that vascular resistance is underestimated in older persons, since there is firm evidence that vascular resistance continues to rise with aging.[96].

Baseline heart rate, total cholesterol, blood glucose, and smoking were not predictive of ISH incidence as quoted by Stanley S. Franklin, MD; William Gustin, IV, BS. This is similar to our study when analyzed statistically between the Isolated Systolic Hypertension group and the Essential Hypertension group.

Thus, our study shows that Isolated Systolic Hypertension is a definite clinical entity and not a benign consequence of aging.

CONCLUSION

CONCLUSION

1. Isolated Systolic Hypertension is not an inevitable consequence of aging; rather it is the endpoint of several contributing factors.
2. Increasing Age and Female Preponderance are the most significant variables in the evolution of Isolated Systolic Hypertension.
3. The Blood Pressure components namely Systolic and Diastolic Blood Pressure, Pulse Pressure and Mean Arterial pressure influence the occurrence of Isolated Systolic Hypertension.
4. Baseline cholesterol and Baseline Blood glucose were not predictive of Isolated Systolic Hypertension incidence but may influence and contribute in due course.
5. Similarly Smoking and BMI were also not predictive of Isolated Systolic Hypertension incidence.
6. Since Isolated Systolic Hypertension is a definite clinical entity, it is important to study the determinants and pay more attention to the diagnosis and treatment of the same.

   Limitations of the study

   - Small Sample Size
   - Baseline Parameters were only used to assess the determinants of Isolated Systolic Hypertension.

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ANNEXURES

ABBREVIATIONS

ISH	:	Isolated Systolic Hypertension
EHT	:	Essential Hypertension
SBP	:	Systolic Blood Pressure
DBP	:	Diastolic Blood Pressure
PP	:	Pulse Pressure
MAP	:	Mean Arterial Pressure
TOD	:	Target Organ Damage
CV-RISK	:	Cardiovascular Risk
AGE	:	Advanced Glycation End products

PROFORMA

CLINICAL PROFILE OF ISOLATED SYSTOLIC HYPERTENSION

No… **HTN Clinic No…**

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

Address: **Education/Occupation**

Phone.No.:

COMPLAINTS:

Head ache	**:**	**Y/N**
Giddiness	**:**	**Y/N**
Palpitations	**:**	**Y/N**
Angina	**:**	**Y/N**
Dyspnoea	**:**	**Y/N**
Syncope	**:**	**Y/N**
Edema	**:**	**Y/N**
Oliguria	**:**	**Y/N**
Limb weakness	**:**	**Y/N**
Epistaxis	**:**	**Y/N**

PAST HISTORY: DM/Smoking/Alcohol/Drug intake

FAMILY HISTORY OF HYPERTENSION:

EXAMINATION:

Wt (kg): **Waist circumference (cm):**

Ht (cm): **BMI(kg/m^2):**

Pallor/Juandice/Cyanosis/Clubbing/Edema/Lymphadenopathy

Pulse (per min): **Peripheral Pulse:**

BP **UL(R):** **mm/Hg** **UL(L):** **mm/Hg**

Supine: **mm/Hg** **Standing:** **mm/Hg**

JVP :

CVS : **S_1 S_2 Added sounds**

RS : **Breath Sounds**
Crepts/Rhonchi

ABD : **Aortic Pulsations**
Bruit

CNS : **FUNDI :**

INVESTIGATIONS:

Hb(%)
ESR
RBS
UREA
CREATININE
Na^+
K^+
Total Cholestrol
LDL
HDL
TGL
VLDL
Se.Calcium
Se.Uric acid

Urine ALB:
ECG:
ECHO:
USG ABD:
Others:

COMMENTS:

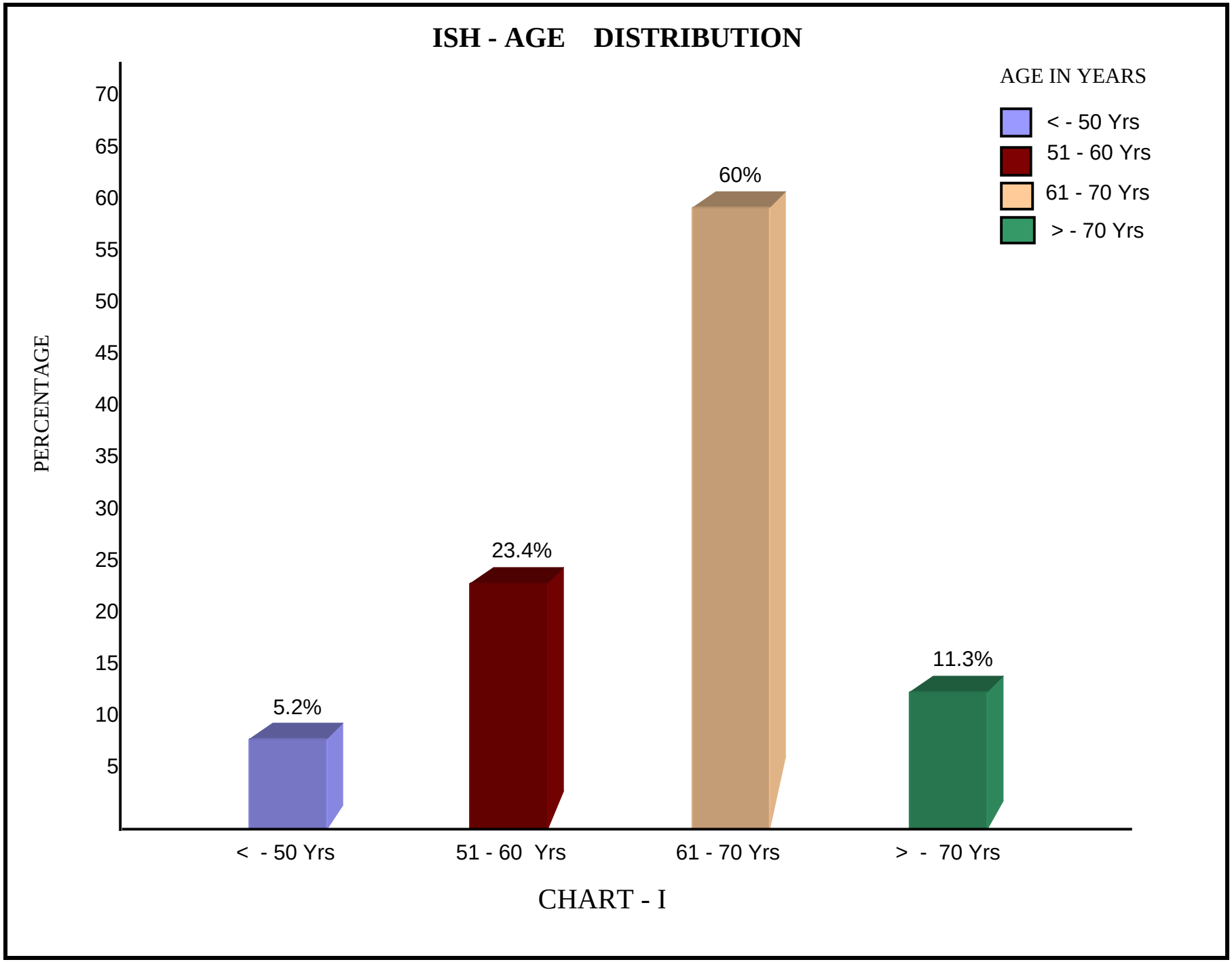
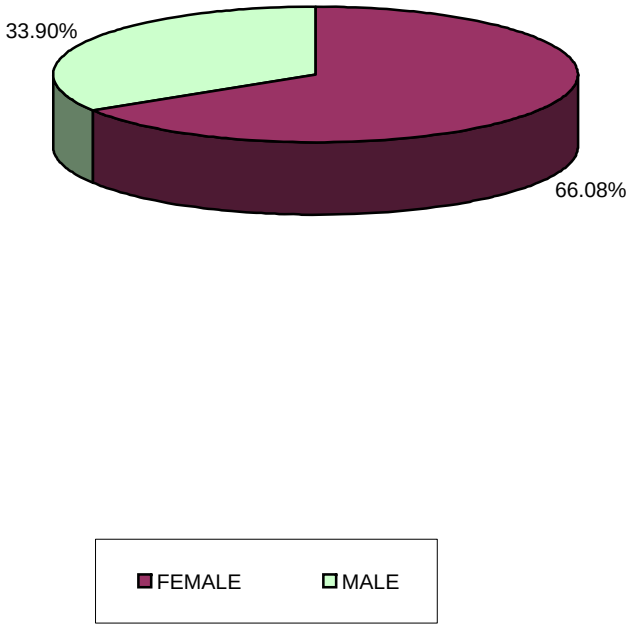

ISH - AGE DISTRIBUTION
AGE IN YEARS
< - 50 Yrs
51 - 60 Yrs
61 - 70 Yrs
> - 70 Yrs
PERCENTAGE
70
65
60
55
50
45
40
35
30
25
20
15
10
5
5.2%
23.4%
60%
11.3%
< - 50 Yrs
51 - 60 Yrs
61 - 70 Yrs
> - 70 Yrs

CHART - I

SEX DISTRIBUTION - ISH



SEX DISTRIBUTION - EHT

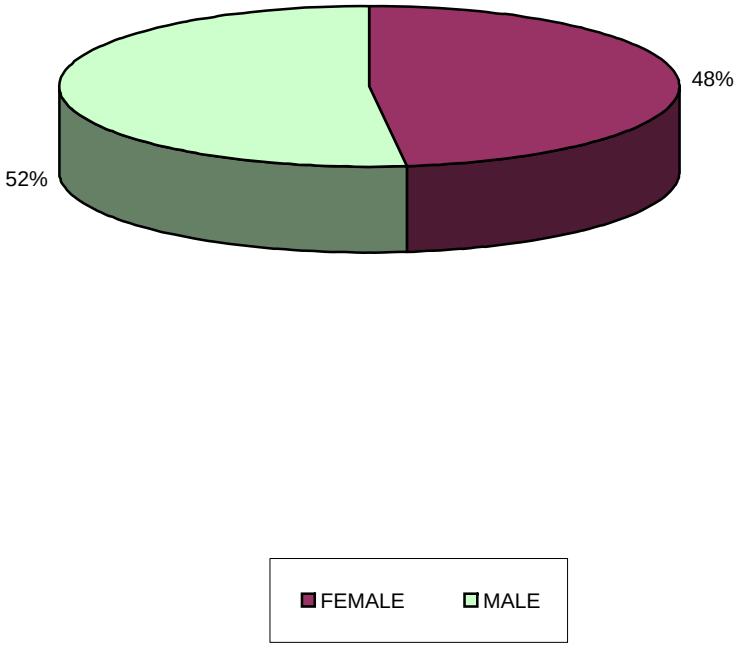


CHART - II

SYMPTOM - ANALYSIS

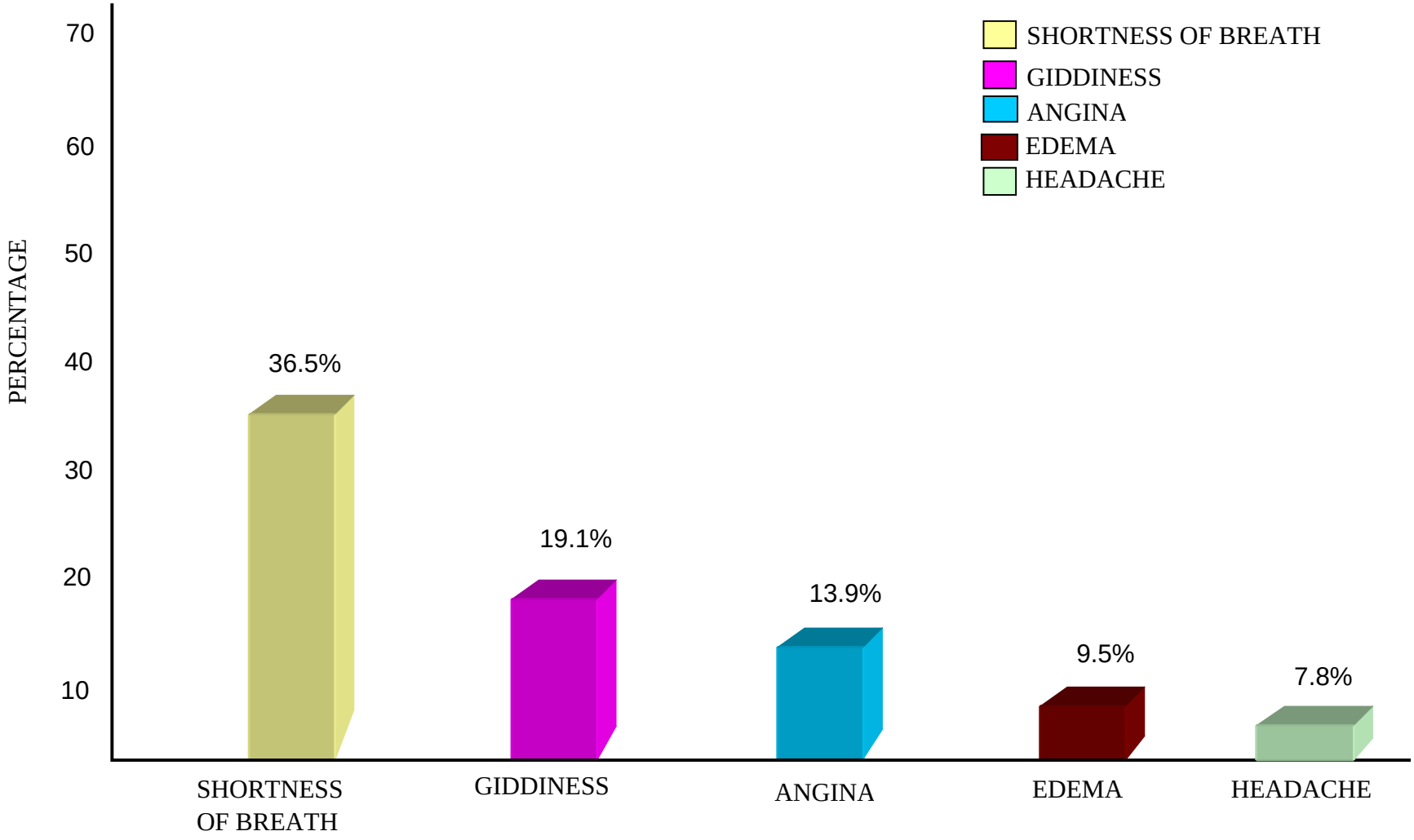


CHART - III

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	NAME	HTN No.	AGE (yrs)	GENDER	SYMPTOMS								FAMILY H/O HTN		
					HEADACHE	GIDDINESS	PALPITATION	ANGINA	DYSPNOEA	EDEMA	OLIGURIA	SMOKING	MOTHER	FATHER	BOTH
1	Mariammal	4207/06	60	F	-	-	-	+	-	-	-	-	-	-	-
2	Lakshmi	4322/06	62	F	+	+	-	-	+	-	-	-	-	-	-
3	Muthu Krishnan	5016/07	67	M	-	-	-	-	+	-	-	+	-	-	-
4	Prema	4262/06	62	F	+	-	-	-	-	-	-	-	-	-	-
5	Nagaraj	5112/06	64	M	-	+	-	-	-	-	-	+	-	-	-
6	Manonmani	4587/06	53	F	-	+	-	-	-	-	-	-	-	-	-
7	Annal	5011/06	61	F	-	-	-	+	+	-	-	-	-	-	+
8	Ramanujaya	4628/06	60	M	-	-	-	+	+	-	-	+	+	-	-
9	Arasalli	4325/07	62	F	+	-	-	-	+	-	-	-	-	-	-
10	Gloria	4901/07	68	F	-	-	-	-	+	+	-	-	+	-	-
11	Saradha	4100/06	68	F	-	-	-	-	-	-	-	-	-	-	-
12	Ellappan	4801/06	63	M	+	-	-	+	-	-	-	+	-	-	-
13	Kanaga	4315/06	66	F	-	-	-	-	-	-	-	-	-	-	+
14	Murugesan	5207/07	53	M	-	+	-	-	-	-	-	+	-	-	-
15	Kannama	4208/06	66	F	-	-	-	-	-	-	-	-	-	-	-
16	Sundari	4626/07	61	F	-	-	-	-	-	-	-	-	-	-	-
17	Rajalakshmi	4173/07	64	F	-	-	-	-	+	-	-	-	-	-	-
18	Banumathi	4386/06	62	F	-	-	-	-	-	-	-	-	-	-	-
19	Thangaraj	4426/07	44	M	-	-	-	-	-	-	-	+	-	-	-
20	Vadevelu	4465/06	66	M	-	-	-	-	-	-	-	+	-	-	-
21	Kamatchi	4109/07	54	F	-	-	-	-	+	-	-	-	-	-	-
22	Kannagi	4300/07	52	F	-	+	-	-	-	-	-	-	-	-	-
23	Madhavan	4269/06	68	M	-	+	-	+	-	-	-	+	+	-	-
24	Chandra	4613/07	69	F	-	-	-	+	-	-	-	-	-	-	-
25	Sivagami	5316/06	51	F	-	-	-	-	+	-	-	-	-	-	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	NAME	HTN No.	AGE (yrs)	GENDER	SYMPTOMS								FAMILY H/O HTN		
					HEADACHE	GIDDINESS	PALPITATION	ANGINA	DYSPNOEA	EDEMA	OLIGURIA	SMOKING	MOTHER	FATHER	BOTH
26	Kanthal	4529/06	61	F	-	-	-	-	+	+	-	-	+	-	-
27	Lakshmanan	4209/07	57	M	-	-	-	-	-	-	-	+	-	-	+
28	Muniyama	4258/07	64	F	-	-	-	-	+	-	-	-	-	-	-
29	Leelavathy	4297/06	47	F	+	-	-	-	+	-	-	-	-	+	-
30	Sekar	4397/06	61	M	-	-	+	-	-	-	-	+	-	-	-
31	Mani	4912/07	73	M	-	-	-	-	-	-	+	+	-	-	-
32	Mrs. Begam	4656/07	63	F	-	+	-	-	-	+	-	-	-	-	-
33	Magadevi	4928/07	62	F	-	-	-	-	-	-	-	-	-	-	-
34	Vasuki	4971/06	60	F	-	-	-	-	-	+	-	-	+	-	+
35	Meenakshi	5125/06	65	F	-	-	-	-	+	-	-	-	-	-	-
36	Srinivasan	4375/07	56	M	-	+	-	+	+	-	-	+	-	-	-
37	Prema	5248/07	64	F	-	-	-	-	-	-	-	-	-	-	-
38	Ellamma	5341/06	66	F	-	-	-	-	+	-	-	-	-	-	-
39	Kamala veni	4791/07	67	F	-	-	-	+	-	-	-	-	-	-	-
40	Lakshmi	5314/06	72	F	-	-	-	-	+	-	-	-	-	-	-
41	Andal	4257/07	62	F	-	-	-	-	-	-	-	-	-	-	-
42	Narmadha	4199/06	71	F	-	-	-	+	-	-	-	-	-	-	-
43	Ragubai	4766/06	62	F	-	+	-	-	+	-	-	-	-	-	-
44	Nagaraj	4329/07	60	M	-	+	-	-	+	-	-	+	-	-	-
45	Lazar	4587/07	62	M	-	-	-	-	-	+	-	-	-	-	-
46	Kanthimathy	4352/06	55	F	-	-	-	-	+	+	-	-	+	-	-
47	Rani	4365/06	66	F	-	-	+	-	-	-	-	-	+	-	-
48	Logasan	5128/06	64	M	+	+	-	-	+	+	-	+	+	-	-
49	Ellappan	5397/07	77	M	-	-	-	-	-	-	-	-	-	+	-
50	Eswari	5122/07	67	F	+	-	-	-	-	+	-	-	-	-	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	NAME	HTN No.	AGE (yrs)	GENDER	SYMPTOMS								FAMILY H/O HTN		
					HEADACHE	GIDDINESS	PALPITATION	ANGINA	DYSPNOEA	EDEMA	OLIGURIA	SMOKING	MOTHER	FATHER	BOTH
51	Muniyamal	4813/07	71	F	-	-	-	-	-	-	-	-	-	-	-
52	Indirani	4355/06	62	F	-	+	-	+	-	-	-	-	-	-	+
53	Lakshmi	4566/07	63	F	-	+	-	-	-	-	+	-	-	-	-
54	Kalavathi	5312/06	67	F	-	-	-	-	+	-	-	-	-	-	-
55	Sudha	4379/06	65	F	-	-	-	-	+	-	-	-	-	-	-
56	Eswaran	4987/06	56	M	-	-	-	-	+	-	-	+	-	-	-
57	Marudhu	4123/0	63	M	-	-	-	-	-	-	-	+	-	-	-
58	Sakunthala	5513/07	62	F	-	-	-	-	-	-	-	-	-	-	-
59	Arasalli	5397/06	60	F	-	-	-	-	-	-	-	-	-	-	-
60	Annammal	4923/06	58	F	-	-	-	-	+	-	-	-	-	-	-
61	Muthu	4377/06	62	M	-	-	-	-	-	-	-	-	-	-	-
62	Krishnan	4982/07	60	M	-	+	-	-	-	-	-	+	-	-	-
63	Chandra	4568/06	72	F	-	+	-	-	+	-	-	-	-	-	-
64	Arunadevi	4123/06	47	F	-	-	-	-	+	-	-	-	-	-	-
65	Gloria	4378/07	62	F	-	-	-	-	-	-	-	-	-	-	-
66	Ganthimathy	5379/06	60	F	-	-	+	-	+	-	-	-	-	+	-
67	Lakshmipriya	4987/06	61	F	-	-	-	-	-	-	-	-	-	-	-
68	Sarojini	4569/07	57	F	-	-	+	-	-	-	+	-	-	+	-
69	Sundram	4658/07	63	M	-	-	-	-	-	+	-	-	-	-	-
70	Muralidharan	4172/06	64	M	-	-	-	-	-	-	-	-	-	-	-
71	Thangaraj	4293/07	66	M	-	+	-	-	-	+	-	+	-	-	-
72	Chinnamal	4168/06	62	F	-	+	-	-	+	-	-	-	+	-	-
73	Balammal	4912/06	72	F	-	-	-	-	-	-	+	-	-	-	-
74	Kamatchi	4567/07	52	F	-	-	-	+	+	-	-	-	-	-	-
75	Parvathy	4321/06	72	F	-	-	-	+	-	-	-	-	-	-	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	NAME	HTN No.	AGE (yrs)	GENDER	SYMPTOMS								FAMILY H/O HTN		
					HEADACHE	GIDDINESS	PALPITATION	ANGINA	DYSPNOEA	EDEMA	OLIGURIA	SMOKING	MOTHER	FATHER	BOTH
76	Kasthuri	4654/06	73	F	-	+	-	-	+	-	-	-	-	-	-
77	Lakshmi	4789/06	62	F	-	-	-	-	-	-	-	-	-	-	-
78	Vadivelu	4895/06	55	M	+	-	-	-	+	-	-	+	-	-	-
79	Chinathai	5378/07	63	F	-	-	-	-	-	-	-	-	-	-	+
80	Rajendran	5198/07	72	M	-	-	-	-	+	-	-	-	-	-	-
81	Shankaran	5346/07	63	M	-	-	-	-	-	-	-	+	-	-	-
82	Pattammal	4912/06	63	F	-	-	-	-	+	-	-	-	-	-	-
83	Jaya	4852/07	64	F	-	-	-	-	-	-	-	-	-	-	-
84	Yasodha	4951/06	72	F	-	-	-	-	+	-	-	-	-	-	-
85	Murugan	5375/06	60	M	-	+	-	-	-	-	-	-	-	-	-
86	Saradha	5295/07	62	F	-	-	-	-	-	-	-	-	-	-	-
87	Vijayalakshmi	5468/07	61	F	-	-	-	-	+	-	-	-	-	+	-
88	Marimuthu	4288/07	65	M	-	+	-	-	-	-	-	+	-	-	-
89	Chinnaiyan	4333/07	66	M	-	-	-	-	-	-	+	-	-	-	-
90	Kannambal	5247/06	53	F	-	-	-	-	+	-	-	-	-	-	-
91	Kanaga	4287/06	68	F	-	-	-	-	+	-	-	-	-	-	-
92	Vasuki	4255/06	60	F	-	-	+	-	+	-	-	-	-	-	-
93	Sadhasivam	4978/06	62	M	-	-	-	-	-	-	-	-	+	-	-
94	Balan	4567/07	68	M	-	-	-	-	-	-	-	-	+	-	-
95	Duraimurugan	4458/07	54	M	-	-	-	+	-	-	-	+	-	-	-
96	Valli	4192/06	60	F	-	-	-	+	-	-	-	-	-	-	-
97	Thenmozhi	4668/06	74	F	-	-	-	-	-	-	-	-	-	-	-
98	Vanitha	5138/06	62	F	-	-	-	-	-	+	-	-	-	-	-
99	Kanagasabai	5299/06	42	M	-	-	-	-	-	-	-	+	-	-	-
100	Naga lakshmi	4511/06	68	F	-	-	-	-	-	-	-	-	-	-	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	NAME	HTN No.	AGE (yrs)	GENDER	SYMPTOMS								FAMILY H/O HTN		
					HEADACHE	GIDDINESS	PALPITATION	ANGINA	DYSPNOEA	EDEMA	OLIGURIA	SMOKING	MOTHER	FATHER	BOTH
101	Shankaran	4311/06	66	M	-	-	-	-	+	-	-	-	-	-	-
102	Ganesh	4655/06	62	M	-	-	-	-	-	-	-	+	-	-	-
103	Lakshmi	4243/06	68	F	-	+	-	-	+	-	-	-	-	-	-
104	Kamakshi	5100/07	52	F	-	-	-	-	-	-	-	-	-	-	-
105	Vaasugi	4352/06	61	F	-	-	-	-	-	-	-	-	+	-	-
106	Maariammal	5210/07	68	F	-	-	-	+	-	-	-	-	-	-	-
107	Murugesan	4265/06	51	M	-	-	-	-	-	-	-	+	-	-	-
108	Chinna ponnu	4987/07	64	F	-	+	-	+	+	-	-	-	-	-	-
109	Valliammal	4253/06	58	F	+	-	-	-	-	-	-	-	-	+	-
110	Manikam	4761/06	65	M	-	-	-	-	-	-	-	-	-	-	-
111	Kasturi	5302/07	65	F	-	-	-	-	-	-	-	-	-	-	-
112	Saraswati	4523/06	63	F	-	-	-	-	+	-	-	-	-	-	-
113	Bala krishnan	4130/06	72	M	-	-	-	-	-	-	-	+	-	-	-
114	Meenakshi	4634/06	60	F	-	-	-	-	-	-	-	-	-	-	-
115	Kanniappan	4967/06	66	M	-	-	-	-	-	-	-	+	-	-	-

MASTER CHART

ESSENTIAL HYPERTENSION GROUP

Total no of patients 50

Sl. No.	NAME	HTN No.	AGE (yrs)	GENDER	SYMPTOMS								FAMILY H/O HTN		
					HEADACHE	GIDDINESS	PALPITATION	ANGINA	DYSPNOEA	EDEMA	OLIGURIA	SMOKING	MOTHER	FATHER	BOTH
1	Nagaraj	4212/07	65	M	-	-	-	-	-	-	-	+	-	-	-
2	Kandhan	4294/07	62	M	+	-	-	-	+	-	-	-	-	-	-
3	Mariappan	4256/06	65	M	-	+	-	-	-	-	-	+	-	-	-
4	Chinnathai	4975/06	53	F	-	-	-	+	-	-	-	-	-	-	-
5	Muniammal	4263/07	60	F	+	+	-	-	+	-	-	-	-	+	-
6	Velayudham	4350/06	58	M	-	+	-	-	+	-	-	+	-	-	-
7	Paremeswari	4259/06	62	F	-	-	+	-	-	+	-	-	-	+	-
8	Koteeswaran	4213/07	66	M	-	+	-	-	-	-	-	-	-	-	-
9	Valliammal	4358/07	57	F	-	+	-	-	+	-	-	-	-	-	-
10	Shanmugam	4453/07	42	M	+	-	-	-	-	-	-	+	+	-	-
11	Meenakshi	4718/06	65	F	-	-	-	-	-	-	+	-	-	-	-
12	Vijaya	4968/07	58	F	-	+	-	-	+	-	-	-	-	+	-
13	Kamakshi	4720/06	64	F	-	-	-	-	-	-	-	-	-	+	-
14	Eswarammal	5200/07	60	F	-	+	-	+	+	-	-	-	-	-	-
15	Kondammal	4281/06	57	F	+	-	+	-	-	+	-	-	-	-	-
16	Annamal	5145/07	62	F	-	+	-	-	-	-	-	-	+	-	-
17	Deenadayalan	4265/06	48	M	-	+	-	-	-	-	-	-	-	-	-
18	Saradhamal	5051/07	67	F	-	-	-	-	+	-	-	-	-	-	-
19	Sugumari	4214/06	58	F	+	-	-	-	+	-	-	-	+	-	-
20	Jagadeswari	4390/07	36	F	-	-	-	+	+	-	-	-	-	-	-
21	Sundaram	4512/06	65	M	-	+	-	-	-	-	-	-	-	-	-
22	Saranya	4554/07	59	F	-	+	-	-	-	-	-	-	-	+	-
23	Ellappan	4359/06	56	M	-	-	-	-	-	-	-	+	-	-	-
24	Munian	4225/06	52	M	-	-	-	-	-	-	-	+	+	-	-
25	Senthamarai	4455/07	52	F	+	-	-	-	-	-	-	-	-	-	-

MASTER CHART

ESSENTIAL HYPERTENSION GROUP

Total no of patients 50

Sl. No.	NAME	HTN No.	AGE (yrs)	GENDER	SYMPTOMS								FAMILY H/O HTN		
					HEADACHE	GIDDINESS	PALPITATION	ANGINA	DYSPNOEA	EDEMA	OLIGURIA	SMOKING	MOTHER	FATHER	BOTH
26	Kesawan	4348/07	66	M	-	+	-	-	-	-	-	+	-	-	-
27	Annakili	5015/07	53	F	+	-	-	-	+	-	-	-	-	-	-
28	Mangammal	4549/07	65	F	-	+	-	-	-	-	-	-	+	-	-
29	Balakrishnan	4910/06	62	M	-	+	-	-	+	-	-	-	-	-	-
30	Govendammal	4215/06	60	M	+	-	+	-	-	-	-	-	-	-	-
31	Rajalakshmi	4350/07	64	F	-	-	-	-	-	-	-	-	-	-	-
32	Shanmugam	4141/07	63	F	-	-	-	-	+	-	-	-	-	-	-
33	Shankaran	4345/07	67	M	-	+	-	-	+	-	-	+	-	-	-
34	Parijadham	4040/07	55	F	-	-	-	-	-	-	-	-	+	+	+
35	Vadivelu	4559/06	60	M	-	+	-	-	-	-	-	+	-	-	-
36	Mallayan	4916/06	65	M	-	-	-	+	-	-	-	-	-	-	-
37	Kannaiyan	4725/06	59	M	+	-	-	-	-	-	-	-	+	-	-
38	Sadasivam	4216/07	55	M	-	-	-	-	+	-	-	-	-	-	-
39	Frameis	4347/06	50	M	-	+	-	-	+	-	+	-	-	-	-
40	Leelavathi	4475/06	68	F	+	-	-	-	-	-	-	-	-	+	-
41	Lakshmiammal	4550/06	55	F	-	+	-	-	-	-	-	-	-	-	-
42	Thangaraj	4549/07	58	M	-	+	-	-	-	-	-	-	-	-	-
43	Nagammal	4963/07	60	M	-	-	-	-	-	-	-	-	-	-	-
44	Vasuki	4723/07	65	F	+	-	-	-	+	-	-	-	-	-	-
45	Chennappan	4545/06	63	M	-	-	-	-	-	-	-	-	-	-	-
46	Velmurugan	4716/07	58	M	-	+	-	-	+	-	-	-	+	+	+
47	Iyyanar	4226/07	57	M	-	-	-	-	-	-	-	-	-	-	-
48	Pandian	4964/07	65	M	+	-	-	-	-	-	-	-	-	-	-
49	Rajarathnam	4612/06	62	M	-	-	-	-	+	-	-	+	-	-	-
50	Ganesan	4925/06	64	M	-	-	-	-	-	-	-	+	-	-	-

ABBREVATIONS:

(+) - POSITIVE
(-) - NEGATIVE
HK - HYPOKINESIA (ECHO)
LVH - LEFT VENTRICULAR HYPERTROPHY (ECHO)
↑ K ↕ KIDNEY ECHOES
↓ K ↕ KIDNEY ECHOES
FL - FATTY LIVER
CL - CHOLELITHIASIS
WT - WEIGHT
HT - HEIGHT
BMI - BODY MASS INDEX
JVP - JUGULAR VENOUS PRESSURE
HB - HAEMOGLOBIN
ESR - ERYTHROCYTE SEDIMENTATION RATE
Na + - SERUM SODIUM
K + - SERUM POTASSIUM
CHOLES - CHOLESTEROL
TGL - TRIGLYCERIDES
HDL - HIGH DENSITY LIPOPROTEIN
VLDL - VERY LOW DENSITY LIPOPROTEIN
LDL - LOW DENSITY
ALB - ALBUMIN

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	WT (kg)	HT (cm)	BMI (kg/m2)	WAIST (cm)	JVP	EDEMA	PULSE (per min)	SBP (mmHg)	DBP (mmHg)	PULSE PRESSURE (mmHg)	MAP (mmHg)	FUNDUS
1	69.1	160	27	89	-	-	78	156	80	76	105.3	-
2	48	152	20.7	78	-	-	72	180	72	108	108	-
3	64	155	26.6	87	-	-	82	148	80	68	102.6	-
4	63	162	24	84	-	+	86	172	80	92	110.6	I
5	61	153	26	78	+	-	88	150	70	80	96.6	-
6	65	151	28.5	91	-	-	74	190	80	110	116.6	-
7	68	160	26.5	80	-	-	78	150	82	68	104.6	-
8	60	158	24	88	-	+	80	158	70	88	99.3	-
9	66	153	28.1	86	-	-	90	160	70	90	100	-
10	60	160	23.4	86	-	-	60	186	72	114	110	-
11	35.7	145	17	79	-	-	80	200	80	120	120	-
12	52	157	21	81	-	-	72	170	90	80	116.6	-
13	68	166	24.6	92	-	-	78	144	70	74	94.6	I
14	67	154	28.2	85	-	-	64	164	84	80	110.6	-
15	55	162	21	88	-	-	78	150	74	76	99.3	-
16	70	156	28.7	83	-	-	72	170	90	80	116.6	-
17	78	170	26.9	79	-	-	64	184	76	108	112	-
18	52	161	20	88	-	-	74	170	82	88	111.3	-
19	65	155	27	90	-	-	76	168	80	88	109.3	-
20	74	167	26.5	85	-	-	82	180	70	110	106.6	-
21	59	157	23.9	85	-	-	82	156	72	84	100	-
22	70	156	28.7	90	-	-	64	172	88	84	116	-
23	60	165	22	81	+	-	84	170	76	106	111.3	-
24	72	158	28.8	89	-	-	78	150	90	60	110	-
25	47	146	22	91	-	-	66	180	80	100	113.3	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	WT (kg)	HT (cm)	BMI (kg/m2)	WAIST (cm)	JVP	EDEMA	PULSE (per min)	SBP (mmHg)	DBP (mmHg)	PULSE PRESSURE (mmHg)	MAP (mmHg)	FUNDUS
26	67	152	28.9	90	-	+	80	164	80	84	108	I
27	51	160	19.9	80	-	+	90	158	90	68	112.6	-
28	69	164	25.6	79	-	-	76	162	82	80	108.6	-
29	48	159	19	86	-	-	84	170	70	100	103.3	I
30	73	164	27.1	92	-	-	92	154	80	74	104.6	-
31	49	157	20	87	-	-	72	180	80	100	113.3	-
32	67	155	27.8	84	-	-	76	176	80	96	112	-
33	70	163	26.3	88	-	-	86	154	84	70	107.3	-
34	52	147	24	88	-	+	78	152	76	76	101.3	-
35	53	155	22	85	-	-	92	182	78	104	112.6	-
36	75	165	27.5	82	-	-	88	148	90	58	109.3	-
37	54	150	24	78	-	-	78	190	90	100	123.3	-
38	80	171	27.3	89	-	-	100	150	80	70	103.3	-
39	68	156	27.9	76	-	-	80	186	70	116	108.6	-
40	48	151	21	87	-	-	94	280	80	120	120	-
41	55	162	21	83	-	-	90	160	82	78	108	-
42	46	148	21	81	-	-	76	146	90	56	108.6	I
43	66	155	27.4	85	-	-	98	176	80	96	112	-
44	70	160	27.3	84	-	-	72	174	70	104	104.6	-
45	63	162	24	88	-	-	74	152	76	76	101.3	-
46	72	161	27.7	89	-	-	88	156	84	72	108	-
47	58	159	23	85	-	-	68	158	80	78	106	-
48	74	154	31.2	90	-	+	96	168	80	88	109.3	-
49	67	153	28.6	86	-	+	73	148	90	58	109.3	I
50	72	163	27	78	-	-	84	170	80	90	110	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	WT (kg)	HT (cm)	BMI (kg/m2)	WAIST (cm)	JVP	EDEMA	PULSE (per min)	SBP (mmHg)	DBP (mmHg)	PULSE PRESSURE (mmHg)	MAP (mmHg)	FUNDUS
51	50	158	20	92	-	-	82	150	90	60	110	-
52	82	172	27.7	80	-	-	80	180	90	90	120	-
53	66	158	26.4	87	-	-	76	152	80	72	104	-
54	68	154	28.6	79	-	-	86	200	82	118	119.3	-
55	51	149	23	86	-	+	76	198	72	126	114	-
56	50	159	19.7	80	-	-	78	164	90	74	114.6	-
57	63	149	28.9	87	-	-	80	150	80	70	103.3	-
58	66	154	27.8	82	-	-	80	170	80	90	110	-
59	67	150	29.7	84	-	-	82	176	70	106	105.3	-
60	53	154	22.3	89	-	-	78	180	90	90	120	-
61	66	151	28.9	86	-	-	90	148	74	74	98.6	-
62	50	162	19	84	-	-	70	168	78	90	108	-
63	49	153	21	82	-	-	86	170	80	90	110	-
64	54	161	20.8	87	-	+	74	152	84	60	106.6	-
65	71	160	27.7	80	-	-	76	180	70	110	106.6	-
66	55	158	22	79	-	-	82	158	90	68	112.6	-
67	80	170	27.6	88	-	-	90	190	70	120	110	-
68	66	152	28.5	82	-	-	88	144	80	64	101.3	-
69	53	155	22	88	-	-	82	192	70	122	110.6	-
70	66	150	29.3	90	-	-	70	180	86	94	117.3	-
71	50	158	20	86	-	-	78	172	90	82	117.3	-
72	74	163	27.8	81	-	-	75	156	80	76	105.3	-
73	65	153	27.7	87	+	-	82	158	70	88	99.3	-
74	50	152	21.6	78	-	-	96	200	82	118	121.3	-
75	68	160	26.5	89	-	-	80	150	90	60	110	I

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	WT (kg)	HT (cm)	BMI (kg/m2)	WAIST (cm)	JVP	EDEMA	PULSE (per min)	SBP (mmHg)	DBP (mmHg)	PULSE PRESSURE (mmHg)	MAP (mmHg)	FUNDUS
76	53	151	23.2	92	-	-	74	192	86	106	121.3	-
77	69	153	29.4	82	-	+	80	170	80	90	110	-
78	59	162	22.4	83	-	-	76	160	76	84	104	-
79	67	152	28.9	91	-	+	94	180	90	90	120	-
80	84	173	28	85	-	-	82	156	90	66	112	-
81	55	154	23.1	81	-	-	87	152	70	82	97.3	-
82	54	150	24	78	-	-	72	172	72	100	105.3	-
83	55	153	23.4	86	-	-	84	164	70	94	101.3	-
84	65	151	28.5	94	-	-	90	148	80	68	102.6	-
85	56	157	22.7	85	-	-	88	190	74	116	112.6	-
86	63	155	26.2	88	+	-	82	152	80	72	104	-
87	64	160	25	96	-	-	76	180	80	100	113.3	-
88	61	153	26	79	-	-	74	176	76	100	109.3	I
89	62	153	26.8	90	-	-	86	160	80	80	106.6	-
90	70	161	27	89	-	-	80	190	70	120	110	-
91	63	152	27.2	92	-	-	90	182	80	102	114	-
92	64	163	24	82	-	-	73	176	72	104	106.6	I
93	70	160	27.3	90	-	+	86	150	88	62	108.6	-
94	72	162	27.4	87	-	-	72	200	80	120	120	-
95	74	167	26.5	83	-	-	96	190	90	100	123.3	-
96	60	155	25	98	-	-	78	158	70	88	99.3	-
97	70	162	26.6	84	-	-	92	186	70	116	108.6	-
98	74	164	27.5	80	-	-	82	160	90	70	113.3	-
99	56	158	22.4	88	-	-	80	170	76	94	107.3	-
100	56	156	23	94	-	-	90	156	80	76	105.3	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	WT (kg)	HT (cm)	BMI (kg/m2)	WAIST (cm)	JVP	EDEMA	PULSE (per min)	SBP (mmHg)	DBP (mmHg)	PULSE PRESSURE (mmHg)	MAP (mmHg)	FUNDUS
101	72	158	28.8	88	-	-	82	164	72	92	102.6	-
102	80	160	31.5	83	-	-	90	156	84	72	108	-
103	54	150	24	92	-	-	94	190	82	108	118	I
104	64	155	26.6	90	-	-	96	150	72	78	98	-
105	55	153	23.4	87	-	+	80	168	88	80	114.6	-
106	85	163	32	82	-	-	96	160	90	70	113.3	II
107	53	154	22.3	102	-	-	88	180	84	96	116	-
108	68	160	26.5	79	-	-	90	160	80	80	106.6	-
109	73	156	30	83	-	-	80	170	88	82	115.3	I
110	58	159	23	96	-	-	84	158	74	84	102	-
111	64	151	28	78	-	-	92	174	80	94	111.3	-
112	60	158	24	90	-	-	88	152	74	78	100	-
113	81	162	31	80	-	-	82	162	90	72	114	-
114	75	165	27.5	88	-	-	98	200	84	116	122.6	-
115	70	156	28.7	80	-	-	84	182	76	106	111.3	-

MASTER CHART

ESSENTIAL HYPERTENSION GROUP

Total no of patients 50

Sl. No.	WT (kg)	HT (cm)	BMI (kg/m2)	WAIST (cm)	JVP	EDEMA	PULSE (per min)	SBP (mmHg)	DBP (mmHg)	PULSE PRESSURE (mmHg)	MAP (mmHg)	FUNDUS
1	67	150	29.7	85	-	-	82	170	98	72	122	-
2	69	153	29.4	87	-	-	84	156	92	64	113.3	-
3	48	159	19	79	-	-	64	188	110	78	136	-
4	50.5	155	21	90	-	-	78	220	100	120	140	I
5	66	154	27.8	92	-	-	82	176	100	76	125.3	-
6	60	158	24	88	-	+	84	172	96	76	121.3	-
7	63	149	28.9	80	-	-	73	170	100	70	123.3	-
8	68	156	27.9	83	-	-	90	190	94	96	126.0	-
9	82	172	27.7	86.5	+	+	86	160	94	66	116	II
10	47	160	18.3	78	-	-	88	180	120	60	140	-
11	63	162	24	84	-	-	78	174	110	64	131.3	-
12	56	156	23	88	-	-	90	184	104	80	130.6	-
13	68	166	24.6	82	-	-	100	186	106	80	132.6	-
14	47	146	22	80	-	+	86	152	114	38	126.6	-
15	58	159	23	87	-	-	88	188	100	88	129.3	I
16	53	152	23	84.5	-	-	92	160	110	50	126.6	-
17	59	157	23.9	90.5	+	+	94	170	100	70	123.3	-
18	67	154	28.2	79	-	-	90	150	106	44	120.6	-
19	52	147	24	86.5	-	-	92	176	110	66	132	-
20	67	152	28.9	81	-	-	82	180	110	70	133.3	I
21	53	155	22	84	-	-	84	200	100	100	133.3	-
22	80	171	27.3	88	-	-	86	164	120	44	134.6	-
23	53	154	22.3	82	-	-	85	190	104	86	132.6	-
24	67	153	28.6	87	-	-	78	186	106	80	132.6	-
25	66	154	27.8	88	-	-	88	200	110	90	140	-

MASTER CHART

ESSENTIAL HYPERTENSION GROUP

Total no of patients 50

Sl. No.	WT (kg)	HT (cm)	BMI (kg/m2)	WAIST (cm)	JVP	EDEMA	PULSE (per min)	SBP (mmHg)	DBP (mmHg)	PULSE PRESSURE (mmHg)	MAP (mmHg)	FUNDUS
26	53	154	22.3	78	-	-	76	150	104	46	119.3	-
27	66	153	28.1	86	-	-	80	178	92	86	120.6	-
28	74	163	27.8	79	-	-	72	182	102	80	128.6	-
29	67	152	28.9	83	-	-	78	210	104	106	139.3	I
30	50	159	19.7	88	-	-	74	170	106	64	127.3	-
31	67	152	28.9	80	-	-	82	152	100	52	117.3	-
32	70	162	26.6	91	-	-	85	170	100	70	123.3	-
33	59	162	22.4	90	-	-	84	180	94	86	122.6	-
34	56	156	23	81	-	-	90	200	98	102	132	I
35	70	161	27	80	-	-	82	184	106	78	132	-
36	50	152	21.6	87	-	-	88	180	120	60	140	-
37	55	162	21	84	-	-	90	172	114	58	133.3	-
38	64	163	24	89	-	-	84	160	108	52	125.3	-
39	48	152	20.7	82	-	-	92	184	100	84	128	-
40	54	150	24	84	-	-	86	180	94	86	122.6	-
41	50	158	20	82.5	-	-	80	188	110	78	136	II
42	60	158	24	85	-	-	90	148	96	52	113.3	-
43	55	158	22	87	-	-	88	182	110	72	134	-
44	74	164	27.5	83	-	-	85	170	94	76	119.3	-
45	56	157	22.7	88	-	-	82	160	100	60	120	-
46	67	152	28.9	86.5	-	-	78	186	98	88	137.3	-
47	68	166	24.6	82	-	-	84	176	92	84	120	I
48	66	151	29	87	-	-	73	158	110	48	126	-
49	60	160	23.4	89	-	-	86	190	100	90	130	-
50	70	156	28.7	80	-	-	90	180	120	60	140	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	HB (gm)	ESR (mm)	SUGAR (mg/dl)	UREA (mg/dl)	CREATININE (mg/dl)	NA+ (Meq/L)	K+ (Meq/L)	TOTAL CHOLES (mg/dl)	TGL (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	SE CALCIUM (mg/dl)	SE URIC ACID (mg/dl)	URINE ALB	ECG	ECHO	USG ABD
1	11	15	102	28	0.8	140	3.5	134	109	52	22	166	9.6	2.3	+	+	LVH	-
2	9	11	91	26	1.4	143	3.4	242	200	54	14	112	8.6	3.6	-	-	-	-
3	10.4	50	125	22	0.5	134	3.7	200	74	36	26	220	9.3	3.3	-	+	-	-
4	10.2	7	112	20	2	140	4.7	206	158	55	32	152	9.8	4.2	-	-	-	-
5	8.2	16	118	26	0.6	137	3.3	194	64	48	33	134	10	4	-	+	LVH	-
6	10.7	41	240	50	1.5	139	5.0	236	188	56	27	142	10.2	2.6	-	+	LVH	-
7	12	14	132	28	0.9	142	3.9	144	143	48	25	182	9.6	5	-	-	-	-
8	12.7	12	129	24	0.4	132	3.8	294	68	35	23	138	8.3	7.2	-	+	-	-
9	11.8	10	234	25	0.7	131	3.8	211	75	42	20	137	9.2	3.3	++	+	-	↑K
10	9.4	20	81	16	1.3	141	4.8	128	111	46	32	83	9.6	3.6	-	-	-	-
11	10.5	6	119	18	1.1	137	4.7	310	172	36	36	203	9.3	2.6	-	-	HK	-
12	10.8	37	270	64	1.5	134	3.7	243	121	50	29	56	9.2	3.4	-	+	LVH	-
13	11.9	14	116	38	1.4	135	5.0	207	76	37	21	120	9.6	4	-	-	-	-
14	8.6	12	90	40	1.1	147	3.6	263	182	58	41	138	9.7	5.2	-	-	-	CL
15	11.2	19	214	25	+	141	3.0	222	122	50	16	48	10.8	3.8	+	-	LVH	-
16	8.8	12	103	23	0.8	135	4.5	128	232	66	25	136	9.3	2.6	-	-	-	-
17	11.6	25	144	22	0.5	138	4.8	152	98	42	44	200	9.8	2.4	-	-	-	-
18	9.2	13	302	28	1.3	135	4.1	280	82	49	37	55	10.7	4.2	-	-	HK	-
19	11.4	20	111	24	0.7	144	5.0	224	168	39	15	96	9.7	3.3	-	-	-	-
20	10.9	15	292	27	1.2	142	4.6	162	60	53	23	88	9.6	3.8	-	-	-	-
21	11.5	14	93	24	1.3	141	3.1	156	117	42	40	82	9.3	2.6	-	-	-	-
22	9.2	46	101	23	0.9	130	3.9	300	108	48	37	90	8.4	3.2	-	-	-	-
23	12.5	13	286	31	0.4	143	3.2	210	111	35	15	172	9.7	3.4	-	-	LVH	-
24	12.0	11	113	28	1.1	131	4.4	160	200	40	41	144	8.8	2.8	-	-	-	-
25	9.0	40	105	21	0.7	144	4.1	188	112	51	31	66	9.6	4.4	+	+	-	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	HB (gm)	ESR (mm)	SUGAR (mg/dl)	UREA (mg/dl)	CREATININE (mg/dl)	NA+ (Meq/L)	K+ (Meq/L)	TOTAL CHOLES (mg/dl)	TGL (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	SE CALCIUM (mg/dl)	SE URIC ACID (mg/dl)	URINE ALB	ECG	ECHO	USG ABD
26	11.7	16	80	50	1.3	140	4.8	230	152	58	12	162	9.1	5.0	-	-	-	-
27	8.0	13	243	30	0.4	137	5.0	143	177	50	33	57	9.8	2.4	-	+	-	-
28	10.6	10	100	22	0.5	132	3.1	206	110	34	29	136	8.9	2.6	-	+	-	-
29	8.8	16	89	26	0.8	145	5.0	307	210	57	37	140	9.2	8.8	++	+	-	-
30	10.2	32	310	30	1.4	133	5.7	236	124	50	27	152	9.0	3.2	-	-	HK	-
31	11.3	12	111	38	1.0	131	3.3	286	113	45	23	170	9.3	3.6	-	-	-	-
32	10.8	9	262	40	1.8	144	3.2	138	68	44	19	144	8.3	4.2	-	+	-	-
33	10.5	14	117	27	1.2	134	4.1	210	165	62	20	114	9.4	2.8	-	-	LVH	↑K
34	8.7	18	82	22	0.5	136	4.6	272	129	42	22	122	10.7	5.0	+	-	-	-
35	10.7	26	104	25	1.4	143	4.0	224	89	37	25	168	9.7	6.0	-	+	-	-
36	10.3	11	306	20	1.2	132	4.2	214	220	46	21	68	9.6	3.3	-	+	-	-
37	7.6	13	92	62	0.6	144	3.3	192	158	40	44	53	9.8	5.7	-	-	-	↑K
38	12.6	10	274	23	1.2	133	4.2	256	172	45	20	180	9.0	2.5	+	+	LVH	-
39	9.8	17	83	29	1.5	139	3.3	169	147	39	26	166	10.8	3.3	-	+	LVH	-
40	11.4	15	114	31	0.4	142	4.9	134	146	41	30	188	9.4	3.5	-	-	-	-
41	9.8	10	286	48	1.5	144	3.6	136	116	36	22	120	8.6	3.2	-	-	-	-
42	11.7	32	88	33	0.9	141	4.2	312	69	52	31	126	9.4	2.6	-	+	LVH	-
43	10.5	18	300	21	1.0	133	4.9	132	180	42	43	130	9.6	2.2	-	+	-	↑K
44	10.8	17	95	19	1.1	135	5.0	272	122	39	27	192	9.4	2.8	+	+	-	-
45	8.6	13	110	35	0.6	141	4.8	207	144	54	41	131	9.5	3.2	-	+	-	-
46	12.4	11	84	40	0.9	143	3.4	128	142	50	43	55	10.6	3.6	-	-	LVH	-
47	10.9	30	224	34	1.0	138	4.0	148	112	37	13	168	9.3	3.6	-	+	-	-
48	9.2	7	120	37	0.6	140	3.5	281	200	42	28	136	8.4	3.5	-	+	LVH	-
49	10.5	18	99	20	1.0	136	3.8	229	119	50	30	200	10.2	4.6	-	-	-	-
50	10.2	40	236	32	1.1	134	4.6	173	100	44	26	100	10.0	5.2	++	-	-	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	HB (gm)	ESR (mm)	SUGAR (mg/dl)	UREA (mg/dl)	CREATININE (mg/dl)	NA+ (Meq/L)	K+ (Meq/L)	TOTAL CHOLES (mg/dl)	TGL (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	SE CALCIUM (mg/dl)	SE URIC ACID (mg/dl)	URINE ALB	ECG	ECHO	USG ABD
51	8.0	16	94	27	1.5	142	4.7	212	188	34	15	150	10.2	6.8	-	+	HK	-
52	11.5	8	106	20	0.7	138	3.5	237	152	41	25	48	9.6	3.3	-	+	-	-
53	8.4	14	244	18	0.8	132	3.6	164	118	43	16	182	9.4	3.5	-	-	-	-
54	12.8	25	115	32	0.6	137	3.9	214	166	58	21	133	8.3	2.8	-	+	-	FL
55	11.5	10	286	28	0.7	135	4.3	265	84	56	28	46	9.3	2.6	+	-	LVH	-
56	10.3	17	87	29	0.9	133	4.1	232	178	64	25	166	10.6	8.4	-	-	-	-
57	9.4	8	123	36	1.0	139	4.6	171	152	48	38	150	9.6	3.0	-	-	-	-
58	10.7	16	212	21	1.2	144	4.5	226	88	51	18	42	9.8	3.3	-	+	HK	-
59	7.4	92	71	112	3.0	138	3.4	186	192	35	41	160	9.4	4.4	-	+	-	↑K
60	10.6	9	118	33	0.9	130	3.8	270	150	52	33	148	9.0	5.2	-	-	-	-
61	11.2	19	110	36	0.8	130	4.6	307	89	49	26	152	9.3	2.3	-	-	LVH	-
62	10.4	7	242	19	0.5	137	3.7	181	97	43	30	88	9.6	2.6	-	+	-	-
63	9.6	9	128	35	0.9	130	5.0	298	98	39	16	212	8.6	3.5	-	+	-	-
64	11.7	20	111	19	0.7	135	3.2	211	210	53	28	101	9.4	11.0	+	+	-	-
65	12.3	9	210	46	0.9	144	4.5	220	146	59	24	144	9.1	3.6	-	-	HK	↓K
66	11.0	26	180	29	1.3	138	4.4	190	96	46	24	67	9.8	3.0	-	-	LVH	-
67	9.2	9	162	35	1.2	136	3.6	224	176	34	30	200	9.0	2.8	++	-	-	-
68	10.2	19	306	18	0.7	137	4.8	188	150	47	17	48	10.7	2.2	-	-	-	-
69	11.6	5	97	39	0.4	135	3.1	312	148	51	26	140	9.6	5.0	-	+	LVH	-
70	8.2	60	290	17	1.1	134	3.4	211	168	45	42	42	9.6	2.5	-	+	-	-
71	10.4	18	80	56	1.4	131	4.4	148	88	34	28	210	8.8	3.6	-	-	-	↑K
72	11.5	6	146	34	0.8	139	5.7	220	117	50	46	58	10.2	4.2	-	-	-	-
73	9.0	18	232	18	1.5	141	4.5	296	182	52	43	186	10.0	4.0	+	+	HK	-
74	10.3	8	119	34	1.2	137	3.1	230	110	37	20	62	9.2	5.2	+	+	-	FL
75	8.3	32	124	16	0.6	138	4.7	164	149	49	27	142	9.0	2.0	-	+	-	-

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	HB (gm)	ESR (mm)	SUGAR (mg/dl)	UREA (mg/dl)	CREATININE (mg/dl)	NA+ (Meq/L)	K+ (Meq/L)	TOTAL CHOLES (mg/dl)	TGL (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	SE CALCIUM (mg/dl)	SE URIC ACID (mg/dl)	URINE ALB	ECG	ECHO	USG ABD
76	10.6	7	300	36	1.3	143	4.3	214	176	45	41	88	10.2	2.5	-	-	LVH	-
77	10.8	40	126	38	0.8	136	3.8	284	90	44	16	196	10.6	2.6	-	+	-	-
78	9.1	8	256	30	1.0	144	4.7	218	112	36	37	158	9.4	3.2	-	-	-	-
79	10.5	6	109	37	1.1	132	4.3	182	180	52	45	201	9.2	3.8	-	+	-	-
80	11.3	20	122	33	0.8	140	3.3	300	114	56	33	132	9.6	4.0	-	+	-	-
81	9.1	12	118	38	1.1	130	5.0	307	126	48	41	138	10.0	2.2	-	+	-	-
82	11.9	5	240	16	0.6	142	3.6	224	116	35	25	73	9.3	2.8	-	+	-	-
83	10.7	30	152	32	0.7	137	4.1	126	100	54	33	192	9.6	3.2	+	-	-	-
84	8.2	6	200	29	1.3	133	4.4	138	112	56	37	142	9.8	3.0	-	-	-	-
85	11.4	5	276	39	0.4	142	4.9	208	200	60	23	148	9.4	4.2	-	-	LVH	-
86	10.3	15	108	17	0.8	133	4.5	162	96	38	13	156	9.8	3.5	-	+	-	↓K
87	12.2	25	86	21	1.4	140	3.9	223	140	47	24	80	9.0	2.6	-	+	-	-
88	11.4	20	98	40	0.6	131	3.8	260	186	45	38	186	9.2	3.3	+	+	-	-
89	8.9	38	140	23	0.5	142	3.3	238	156	36	32	46	9.0	3.7	-	-	-	-
90	11.6	6	312	17	1.2	144	5.0	194	88	41	30	57	9.8	10.6	-	-	HK	FL
91	13	44	157	19	0.9	131	3.5	219	164	46	28	62	8.3	4.3	-	+	LVH	-
92	11.6	17	96	37	1.4	139	5.5	132	89	55	44	188	9.6	4.0	-	-	-	-
93	11.8	5	226	40	0.4	138	4.1	152	192	57	28	66	9.4	5.2	-	+	-	-
94	10.5	19	74	58	1.5	134	3.5	301	117	38	16	133	9.3	2.8	-	+	-	-
95	8.1	11	212	31	1.0	132	3.2	227	178	44	23	80	10.8	2.5	++	-	LVH	-
96	11.0	13	85	17	0.5	141	4.8	181	140	47	38	220	9.2	2.7	-	+	-	-
97	12.0	5	107	38	1.3	132	4.0	230	118	60	32	92	9.0	2.3	-	+	HK	-
98	8.6	8	192	39	1.0	143	3.7	272	93	38	40	172	9.1	3.6	-	-	-	-
99	10.2	7	232	16	0.4	134	5.0	201	132	52	12	110	9.6	2.6	-	-	LVH	FL
100	9.3	20	76	79	1.5	136	4.2	129	98	48	46	168	9.8	2.4	-	+	-	FL

MASTER CHART

ISOLATED SYSTOLIC HYPERTENSION GROUP

Total no of patients 115

Sl. No.	HB (gm)	ESR (mm)	SUGAR (mg/dl)	UREA (mg/dl)	CREATININE (mg/dl)	NA+ (Meq/L)	K+ (Meq/L)	TOTAL CHOLES (mg/dl)	TGL (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	SE CALCIUM (mg/dl)	SE URIC ACID (mg/dl)	URINE ALB	ECG	ECHO	USG ABD
101	11.2	13	150	30	0.9	136	3.5	310	202	55	16	165	9.6	3.4	-	+	-	-
102	9.6	19	173	18	0.6	144	3.8	235	156	58	35	123	8.8	2.8	-	-	-	-
103	12	20	250	33	0.8	142	3.6	288	207	46	38	203	9.2	4.4	++	-	-	-
104	9.2	9	178	23	0.8	131	4.8	220	140	48	17	200	10	5	-	+	HK	-
105	10.5	11	165	15	1.1	142	5	288	210	56	14	172	9	2.4	-	-	-	↑K
106	11	15	120	40	0.7	135	5.3	301	120	58	16	135	8.6	2.8	++	-	-	-
107	9	10	265	35	0.5	139	4.1	316	240	38	25	180	10.2	3.8	-	-	LVH	-
108	11.5	20	128	17	+	136	4.7	216	122	36	33	142	9.4	5	-	-	-	-
109	9.3	16	134	16	0.9	140	4.6	212	232	45	12	192	9.2	2.8	-	-	-	-
110	11	19	126	21	0.5	138	5.2	242	110	33	44	188	8.3	2.5	-	+	-	-
111	11.4	15	290	15	0.8	138	5.4	232	98	35	18	166	9.4	5.7	+	-	LVH	↑K
112	10.4	8	130	18	0.5	143	5	280	225	44	22	172	9.1	2.3	-	-	-	-
113	12	12	115	28	0.7	130	4.8	210	106	34	15	220	9.6	3.5	-	-	-	-
114	9.2	10	300	33	0.8	136	4.6	268	216	40	18	170	9	2.6	-	-	-	FL
115	10	14	116	38	0.6	142	5.2	228	238	36	20	200	9.4	5.2	-	-	HK	-

MASTER CHART

ESSENTIAL HYPERTENSION GROUP

Total no of patients 50

Sl. No.	HB (gm)	ESR (mm)	SUGAR (mg/dl)	UREA (mg/dl)	CREATININE (mg/dl)	NA+ (Meq/L)	K+ (Meq/L)	TOTAL CHOLES (mg/dl)	TGL (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	SE CALCIUM (mg/dl)	SE URIC ACID (mg/dl)	URINE ALB	ECG	ECHO	USG ABD
1	10.8	12	114	37	0.80	130	3.7	196	78	34	19	182	9.6	4.2	-	-	-	-
2	12.4	14	220	22	0.40	137	5.0	186	120	62	44	144	10.0	3.8	+	+	-	-
3	8.6	10	160	28	1.0	135	3.2	200	112	37	22	75	9.2	3.2	-	-	-	-
4	9.2	30	142	40	1.1	144	4.5	222	200	42	30	176	9.3	2.4	+	-	LVH	-
5	10.5	46	312	68	1.2	138	3.6	263	130	36	19	132	10.7	8.8	-	+	HK	↑K
6	8	32	112	25	1.0	136	4.8	230	164	48	32	162	8.4	4.8	-	-	-	-
7	11.5	9	78	39	0.8	135	3.4	192	108	46	15	200	8.6	2.3	+	+	-	-
8	12.6	11	280	24	0.6	131	4.4	236	92	35	12	157	10.4	2.8	-	-	LVH	-
9	8.4	16	88	21	0.5	134	5.7	240	225	43	24	64	9.3	4.2	-	+	-	-
10	9.2	13	300	60	2.2	139	4.5	280	115	46	36	133	9.9	3.5	+	-	HK	↑K
11	12.8	14	180	30	0.5	141	3.1	194	230	39	32	105	9.4	11.0	-	-	-	-
12	7.6	09	310	38	0.8	137	4.7	224	110	44	15	142	8.8	5.8	+	-	HK	-
13	9.4	11	112	27	1.2	143	4.3	284	136	36	19	198	10.0	2.6	-	+	-	-
14	8.7	17	90	32	0.6	136	3.8	306	168	52	23	58	9.5	3.8	-	+	-	-
15	10.6	10	280	112	3.6	138	4.3	198	102	45	26	168	9.4	5.6	+	+	LVH	↓K
16	12.5	18	86	36	0.6	144	4.9	210	80	53	29	154	10.2	4.0	-	-	-	-
17	12.5	15	320	19	0.9	132	3.5	188	100	45	43	52	10.8	2.8	-	+	-	FL
18	10.7	32	104	23	1.1	141	4.1	258	96	38	18	102	9.8	4.2	+	-	-	-
19	12.4	07	94	29	1.0	133	3.6	216	176	47	31	64	9.6	3.8	-	+	-	-
20	9.8	17	275	33	1.2	136	4.5	230	82	46	13	134	9.6	10.6	-	-	LVH	-
21	10.3	18	130	36	1.0	140	3.2	186	115	58	38	210	9.8	3.0	-	+	HK	-
22	12.0	08	340	20	1.2	134	4.4	272	212	48	18	146	9.7	4.4	-	-	-	-
23	11.2	40	142	26	1.1	138	5.0	228	102	38	14	78	9.8	2.8	++	+	HK	-
24	12.5	25	160	31	0.8	142	3.2	296	116	46	18	133	10.0	3.0	-	-	-	-
25	12.5	14	114	35	0.7	142	4.2	216	202	44	23	152	10.2	2.5	-	+	-	-

MASTER CHART

ESSENTIAL HYPERTENSION GROUP

Total no of patients 50

Sl. No.	HB (gm)	ESR (mm)	SUGAR (mg/dl)	UREA (mg/dl)	CREATININE (mg/dl)	NA+ (Meq/L)	K+ (Meq/L)	TOTAL CHOLES (mg/dl)	TGL (mg/dl)	HDL (mg/dl)	VLDL (mg/dl)	LDL (mg/dl)	SE CALCIUM (mg/dl)	SE URIC ACID (mg/dl)	URINE ALB	ECG	ECHO	USG ABD
26	11.7	16	120	18	+	143	3.4	196	146	38	23	116	9.2	3.0	-	-	-	-
27	10.3	13	170	24	+	130	3.7	188	196	53	42	120	9.6	5.2	-	+	-	-
28	11.0	10	280	16	0.8	144	4.7	236	142	49	34	138	10.4	2.6	-	-	LVH	-
29	9.4	15	124	27	0.5	137	5.0	312	122	44	13	98	9.4	4.8	-	+	HK	-
30	8.2	32	162	30	0.9	132	3.5	192	178	36	25	182	9.1	4.0	++	-	-	↑K
31	9.6	7	220	22	+	145	4.5	234	120	57	45	58	9.6	11.0	-	-	-	-
32	12.8	12	152	56	+	131	3.3	220	150	56	27	210	10.3	3.2	-	+	-	-
33	9.2	9	242	25	0.4	136	3.8	196	164	34	15	176	10.0	2.1	-	+	-	-
34	13.0	26	126	18	0.6	144	3.6	230	68	52	16	102	9.9	3.5	-	+	-	-
35	10.5	14	290	78	1.1	131	4.7	310	110	50	39	136	9.3	2.6	-	-	-	↑K
36	11.5	18	320	17	1.2	133	3.0	216	148	37	17	124	9.4	2.8	-	-	-	-
37	11.3	15	144	34	0.5	140	4.8	190	185	58	26	148	10.6	2.4	-	+	HK	-
38	10.5	11	230	31	0.8	142	4.1	234	185	38	14	166	8.6	2.2	-	-	-	-
39	8.6	50	400	20	1.1	131	5.0	236	104	57	12	210	10.8	3.0	-	-	-	FL
40	11.4	13	160	19	0.9	139	4.8	186	190	55	33	112	9.8	4.2	+	-	-	-
41	8.1	10	312	72	1.2	147	5.0	216	106	56	44	140	10.2	4.8	-	+	-	↑K
42	13	17	140	32	0.6	135	4.6	188	1722	51	12	106	9.8	2.3	-	+	LVH	-
43	9.3	14	146	21	0.8	138	3.1	288	92	33	41	45	9.6	4.0	-	-	-	↑K
44	12.0	11	84	26	1.1	136	3.3	220	130	36	28	150	10.8	5.3	-	-	HK	-
45	10.2	16	288	28	0.7	145	5.7	190	196	55	17	190	8.6	5.4	++	+	-	-
46	11.0	30	222	32	0.7	142	4.1	200	98	54	35	175	10.1	4.8	-	+	-	FL
47	9.0	14	98	23	0.8	132	4.0	300	78	35	16	94	10.8	2.5	-	-	-	-
48	10.2	20	122	35	1.2	140	4.6	194	84	60	40	156	10.3	5.6	-	+	-	-
49	10.6	12	136	29	1.8	133	3.3	316	130	34	22	88	10.6	3.5	-	-	-	-
50	9.2	25	214	33	0.8	138	4.2	218	96	37	14	182	9.8	6.6	-	+	-	-