

**DISSERTATION ON**

**“CLINICAL ASSESSMENT AND METABOLIC PROFILE OF NEWLY  
DIAGNOSED HYPERTENSIVES AND THEIR SIGNIFICANCE”**

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## CERTIFICATE

This is to certify that this dissertation entitled “**CLINICAL ASSESSMENT AND METABOLIC PROFILE OF NEWLY DIAGNOSED HYPERTENSIVES AND THEIR SIGNIFICANCE**” submitted by **Dr. ANBU SELVAN. P** appearing for Part II M.D. Branch I General Medicine Degree examination in March 2010 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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## DECLARATION

I solemnly declare that the dissertation titled “**CLINICAL ASSESSMENT AND METABOLIC PROFILE OF NEWLY DIAGNOSED HYPERTENSIVES AND THEIR SIGNIFICANCE**” is done by me at Madras Medical College & Government General Hospital, Chennai during May 2009 to October 2009 under the guidance and supervision of **Prof.T.Manivel,M.D.**, Professor of Medicine, Madras Medical College and Government General Hospital, Chennai.

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical university towards the partial fulfillment of requirements for the award of M.D. Degree(Branch-1) in General Medicine.

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## ABBREVIATIONS

ABPI	-	Ankle brachial index
AT	-	Angiotensin
ATP	-	Adult Treatment Panel
CHD	-	coronary heart disease
CKD	-	Chronic Kidney Disease
CURES	-	Chennai Urban Rural epidemiological Study
DBP	-	diastolic blood pressure
ED	-	erectile dysfunction
ESRD	-	end stage renal disease
HT	-	hypertension
HDL	-	high density lipoprotein
IDF	-	international diabetic federation
IIEF	-	international index for erectile function
ISH	-	isolated systolic hypertension.
JNC	-	joint national committee
MD	-	macula densa
MS	-	metabolic syndrome
Na	-	sodium
NO	-	nitric oxide
NCEP	-	national cholesterol education programme
RAAS	-	rennin angiotensin aldosterone system
PAD	-	peripheral arterial disease
SBP	-	systolic blood pressure

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## INTRODUCTION

Hypertension has long been called as the silent killer as because when its symptoms are apparent the damage is already done to the system and to add to the worse they are mostly irreversible.

The phenomenon of hypertension was first characterized at the turn of the previous century, when Riva-Rocci<sup>[6]</sup> developed the prototype of the modern sphygmomanometer and so allowed the routine measurement of BP. Korotkov<sup>[7]</sup> then perfected the sphygmomanometric technique by describing the sounds heard over the brachial artery as the pressure in the cuff is reduced. In general, the upper limits of normal BP in older persons had been considered to be a systolic value of 140 mm Hg and a diastolic value of 90 mm Hg. These figures have been adjusted downward to the point that readings in excess of 120/80 mm Hg may be considered abnormal.<sup>[8]</sup> Population studies suggest that BP is a continuous variable, with no absolute dividing line between normal and abnormal values.<sup>[14]</sup> A recent report on the global burden of hypertension indicates that nearly 1 billion adults (more than a quarter of the world's population) had hypertension in 2000, and this is predicted to increase to 1.56 billion by 2025<sup>(2)</sup>. Subjects with hypertension are known to have a two-fold higher risk of developing coronary artery disease (CAD), four times higher risk of congestive heart failure and seven times higher risk of cerebrovascular disease and stroke compared to normotensive subjects<sup>(3)</sup>



Hypertension has been identified as one of the leading risk factors for mortality, and is ranked third as a cause of disability adjusted life-years.<sup>(4)</sup> Existing data suggests that the prevalence of hypertension has remained stable or has decreased in economically developed countries during the past decade, while it has increased in developing countries.<sup>(5)</sup> However, the increase in the prevalence rates of hypertension needs to be quantified so as to plan for effective prevention strategies which are urgently needed in developing countries.

However, in spite of its proven association with all clinical associations of atherosclerosis including peripheral vascular diseases hypertension is one disease which is more commonly mismanaged. The awareness about the disease and its complications are poor as only less than 30% of even known hypertensives have a good optimal control even in western population.

Less studies are there in newly diagnosed hypertensives and the prevalence of complications and risk factors among them. Being the diabetes capital of the world already India is also projected to become the Hypertensive capital of the world by 2025. In this context this study is aimed in the evaluation of newly detected hypertensives in a tertiary care centre at Chennai and the prevalence of associated risk factors for future cardio vascular incident among them.

## **AIMS AND OBJECTIVES OF THE STUDY**

- To study the complete profile of newly diagnosed hypertensives.
- To stage them
- To study the prevalence of end organ damage among them.
- To study the prevalence of metabolic syndrome, erectile dysfunction and coronary heart disease equivalents among them.
- To calculate Framingham risk score for all of them

## REVIEW OF LITERATURE

### PREVALENCE:

Hypertension is common among the population. In a Population study conducted in suburban Chennai the prevalence of hypertension was 20% <sup>(1)</sup>. However its prevalence varies with population and geographical area studied.

Similarly the rate of complications and associated risk factors also vary with population studied. In the CURES study Mohan et al observed that in the HT population the rate of cardio vascular risk factors was twice that of non hypertensives on diagnosis.

### PHYSIOLOGY OF BLOOD PRESSURE

Before proceeding with a discussion of the pathophysiology of hypertension, it is necessary to briefly review the factors that control BP homeostasis.

BP is defined as  $BP = CO * TVR$

$$= SV * HR * TVR$$

where BP = blood pressure, CO = cardiac output, HR = heart rate, SV = stroke volume, and TPR = total peripheral resistance.

Body volume varies directly with total body Na content because Na is the predominant extracellular solute that retains water within the extracellular space. One primary function of the kidneys is to regulate Na and water excretion, and consequently, they also provide a dominant role in the long-term control of BP. This phenomenon has been referred to as *pressure natriuresis*.<sup>[9] [10]</sup> The second mechanism employs the RAAS, which directly controls peripheral vascular resistance and renal reabsorption of Na and water. Accordingly, the renin system normally functions as a long-term regulator of BP homeostasis.<sup>[12] [11]</sup>

### **Pressure Natriuresis**

Pressure natriuresis is the increase in urinary excretion of Na and water that occurs when arterial pressure increases. As a consequence of this compensatory renal response, BP is maintained within the normal range.<sup>[14]</sup> Accordingly, the kidney functions as a servocontroller of arterial pressure and exhibits an infinite negative feedback gain for the long-term regulation of arterial pressure by adjusting blood volume.<sup>[13]</sup>

### **Renin-Angiotensin-Aldosterone System**

The ability to maintain normal BP at Na intakes ranging from levels well below to those far above normal is a direct effect of the circulating levels of renin-Ang II. The kidneys secrete the enzyme renin from the juxtaglomerular cells in response to a variety

of normal or abnormal phenomena that reduce arterial BP, renal perfusion, or Na chloride load to the macula densa (MD).<sup>[12]</sup> These include changes in posture or effective circulating fluid volume (i.e., Na depletion, hemorrhage, HF, nephrotic syndrome, cirrhosis). Baroreceptors in the afferent arterioles, chloride-sensitive receptors in the MD and juxtaglomerular apparatus, and efferent renal sympathetic nerve activity all participate in this feedback control.<sup>[15] [16]</sup> In this way, circulating renin levels are tightly regulated and subject to constant physiologic adjustment.<sup>[17] [18]</sup>

The kidney secretes renin into the peripheral circulation and, thus, it has characteristics of both an enzyme and a hormone.<sup>[19]</sup> The half-life of renin in the circulation is about 15-20 minutes, with its metabolism occurring primarily in the liver. Under normal circumstances, changes in the biosynthesis and renal secretion of renin are the primary determinants of plasma Ang II formation. Thus, renin secretion is the rate-limiting step in the regulation of the renin-angiotensin system<sup>[19]</sup>. Renin cleaves the inactive Ang I, from angiotensinogen Ang I is then converted to the octapeptide, Ang II by ACE, located in the circulation.

Ang II is the first effector hormone of the system. It increases BP in several different ways, each of which is mediated by the AT1. First, it exerts powerful, direct, and immediate vasoconstriction and, thus, increases peripheral vascular resistance<sup>[20] [21]</sup>  
<sup>[22]</sup> Second, it rapidly stimulates Na reabsorption via proximal nephron and at the medullary thick ascending limb<sup>[23] [24]</sup>. Third, and at a slower pace, Ang II stimulates

aldosterone biosynthesis and secretion by the adrenal zona glomerulosa.

Aldosterone stimulates electrogenic reabsorption of Na by principal cells in the collecting duct and by mineralocorticoid responsive tissue in the colon and sebaceous and salivary glands. Retained Na is responsible for increased extracellular fluid volume that increases BP hydraulically and also heightens vascular sensitivity to Ang II and other vasoconstrictors.<sup>[25]</sup> Together, these multiple effects combine to raise BP and to restore fluid volume to the point at which the initial signals for renin release (i.e., low BP and renal perfusion pressure) are attenuated or abolished.

In the normotensive individual, when salt intake is increased, circulating renin-Ang II levels decrease and BP remains within the normal range. Conversely, when Na intake decreases, renin-Ang II levels increase without a significant deviation in BP.<sup>[26]</sup> Thus, BP is kept relatively constant, even when Na intake is varied from 10 to 1500 mEq/day, because of the reciprocal change in Ang II levels and body Na content<sup>[27]</sup>.

### **Cytochrome P-450–Dependent Metabolites of Arachidonic Acid**

Arachidonic acid is metabolized primarily by CYP enzymes in the brain, lung, kidney, and peripheral vasculature to 20-hydroxyeicosatetraenoic acid (20-HETE) and epoxyeicosatrienoic acids (EETs).<sup>[28]</sup> EETs, which are derived from endothelial cells, promote vasodilatation by activating K<sup>+</sup> channels and hyperpolarizing vascular smooth muscle cells. By contrast, 20-HETE vasoconstricts vascular smooth muscle cells by

reducing the open-state probability of  $\text{Ca}^{2+}$ -activated  $\text{K}^+$  channels. Both EETs and 20-HETEs play an important role in renal vascular tone and Na excretion, and thus, it has been proposed that they participate in the pressure natriuresis relationship

## **Nitric Oxide**

Renal medullary blood flow plays a central role in the maintenance of BP homeostasis. NO regulates medullary renal vascular resistance, natriuresis, and diuresis and, therefore, contributes importantly to the pressure natriuresis relationship. <sup>[29]</sup> <sup>[30]</sup> There is considerable evidence that NO serves an important counterregulatory role within the renal medullary circulation.

## **SYSTEMIC REGULATION BY THE NERVOUS SYSTEM**

### **Neural**

Although the arterioles and the other resistance vessels are most densely innervated, all blood vessels except capillaries and venules contain smooth muscle and receive motor nerve fibers from the sympathetic division of the autonomic nervous system. The fibers to the resistance vessels regulate tissue blood flow and arterial pressure..

## **Vessel innervation**

Noradrenergic fibers end on vessels in all parts of the body<sup>(31)</sup>. The noradrenergic fibers are vasoconstrictor in function. In addition to their vasoconstrictor innervation, the resistance vessels of the skeletal muscles are innervated by vasodilator fibers, which, although they travel with the sympathetic nerves, are cholinergic (the sympathetic vasodilator system.)

There is no tonic discharge in the vasodilator fibers, but the vasoconstrictor fibers to most vascular beds have some tonic activity.

## **Cardiac innervation**

Impulses in the noradrenergic sympathetic nerves to the heart increase the cardiac rate (chronotropic effect) and the force of cardiac contraction (inotropic effect). They also inhibit the effects of vagal stimulation, probably by release of neuropeptide Y, which is a cotransmitter in the sympathetic endings. Impulses in the cholinergic vagal cardiac fibers decrease the heart rate. There is a moderate amount of tonic discharge in the cardiac sympathetic nerves at rest, but there is a good deal of tonic vagal discharge (vagal tone).



## **VasomotorControl**

Spinal reflex activity affects blood pressure, but the main control of blood pressure is exerted by groups of neurons in the medulla oblongata that are sometimes called collectively the vasomotor area or vasomotor centre.

When vasoconstrictor discharge is increased, there is increased arteriolar constriction and a rise in blood pressure. Heart rate and stroke volume are increased because of activity in the sympathetic nerves to the heart, and cardiac output is increased. There is usually an associated decrease in the tonic activity of vagal fibres to the heart. Conversely, a decrease in vasomotor discharge causes vasodilation, a fall in blood pressure, and an increase in the storage of blood in the venous reservoirs. There is usually a concomitant decrease in heart rate, but this is mostly due to stimulation of the vagal innervations of the heart

## **Baroreceptors**

The baroreceptors are stretch receptors in the walls of the heart and blood vessels. The carotid sinus and aortic arch receptors monitor the arterial circulation. The baroreceptors are stimulated by distention of the structures in which they are located, and so they discharge at an increased rate when the pressure in these structures rises. increased baroreceptor discharge *inhibits* the tonic discharge of the vasoconstrictor nerves and *excites* the vagal innervation of the heart, producing vasodilation,

venodilation, a drop in blood pressure, bradycardia, and a decrease in cardiac output.

### **Carotid Sinus & Aortic Arch**

The carotid sinus is a small dilation of the internal carotid artery just above the bifurcation of the common carotid into external and internal carotid branches . Baroreceptors are located in this dilation. They are also found in the wall of the arch of the aorta.. The carotid sinus nerves and vagal fibers from the aortic arch are commonly called the buffer nerves.

### **Buffer Nerve Activity**

At normal blood pressure levels, the fibers of the buffer nerves discharge at a low rate . When the pressure in the sinus and aortic arch rises, the discharge rate increases; and when the pressure falls, the rate declines.

The carotid receptors respond both to sustained pressure and to pulse pressure<sup>(32)</sup>. A decline in carotid pulse pressure without any change in mean pressure decreases the rate of baroreceptor discharge and provokes a rise in blood pressure and tachycardia. The receptors also respond to changes in pressure as well as steady pressure; when the pressure is fluctuating, they sometimes discharge during the rises and are silent during the falls at mean pressures at which if there were no fluctuations, there would be a steady discharge.

From the foregoing discussion, it is apparent that the baroreceptors on the arterial side of the circulation, their afferent connections to the vasomotor and cardioinhibitory areas, and the efferent pathways from these areas constitute a reflex feedback mechanism that operates to stabilize the blood pressure and heart rate. Any drop in systemic arterial pressure decreases the inhibitory discharge in the buffer nerves, and there is a compensatory rise in blood pressure and cardiac output. Any rise in pressure produces dilation of the arterioles and decreases cardiac output until the blood pressure returns to its previous normal level.

## **PATHOPHYSIOLOGY OF HYPERTENSION**

Several lines of evidence further support the conclusion that the kidney plays an essential role in the pathogenesis of hypertension. First, in human hypertension and virtually all experimental models of hypertension, the ability to excrete Na is impaired at normal BP.<sup>[9] [10] [33]</sup> This phenomenon has been demonstrated in renal artery stenosis, aortic coarctation, mineralocorticoid hypertension, surgical reduction of renal mass, glomerulonephritis, long-term infusion of vasoconstrictors,<sup>[34]</sup> and all genetic rat models of hypertension. Second, when renal excretory function is dampened by infusion of Na- and water-retaining hormones (e.g., vasopressin, Ang II, aldosterone), an increase in renal perfusion pressure is required to restore Na and volume homeostasis. Third, all effective antihypertensive drugs shift the pressure natriuresis relationship back to control levels.<sup>[34]</sup> Fourth, the BP level in a human or experimental animal renal transplant

recipient is directly related to the BP of the kidney donor. For example, to maintain a similar BP level in renal transplant recipients without familial hypertension, a kidney received from a hypertensive donor determines a 10-fold larger increase in the required dose of antihypertensive therapy than the transplantation of a kidney from a normotensive donor.<sup>[35]</sup>

### **Vascular Remodeling and Pathologic Changes**

In primary hypertension, the column of blood in the arterial tree between the aortic valves and the capillaries moves at abnormally high pressure throughout the cardiac cycle of contraction and relaxation. However, cardiac output is usually normal or close to normal. Thus, the main determinant of the sustained elevated BP is an increase in peripheral resistance. The increase in vascular resistance, a cardinal characteristic of diastolic hypertension, is commonly related to excessive vasoconstriction of arteriolar smooth muscle, although it can also result, at least in part, from structural changes in these arterioles, from increased blood viscosity, or even perhaps from increased extravascular (interstitial) pressure.<sup>[36]</sup>

Renal vein catheterization data also indicate that the earliest physiologic lesion of essential hypertension is vascular. Under normal circumstances, peripheral resistance is determined predominantly by the precapillary vessels with a lumen diameter of approximately 100 to 300  $\mu\text{m}$ .<sup>[37] [38]</sup> In human hypertension and in experimental animal models of hypertension, structural changes in these resistance vessels are commonly

observed. In primary hypertension, the outer diameter and lumen of these vessels are smaller, the media-to-lumen ratio is greater, but the cross-sectional area of the media is not different from that of age- and sex-matched normotensive subjects. This pathologic alteration is termed *eutrophic remodeling*.<sup>[39]</sup>

The LaPlace relationship illustrates that these characteristic vascular changes provide an adaptive function by reducing wall tension as follows:  $T = P * (r/w)$

where T = tension per unit wall layer, P = transmural pressure, r = radius, and w = wall thickness.

It is apparent from this relationship that, when the P increases, T remains constant only if the ratio of radius to wall thickness (r/w) decreases proportionately by w thickening and/or r decreasing. When this alteration of vascular structure occurs within the resistance axis, located from the aortic valve up to and including the glomerular capillary membrane, then it contributes to the long-term elevation in BP.<sup>[14]</sup>

In human essential hypertension, increasing evidence supports the view that vascular remodeling, rather than growth, is the predominant change occurring in resistance vessels. The increase in media-to-lumen ratio of the resistance vessels occurs by the addition of material to either the outer or the inner surfaces of the blood vessel wall.<sup>[40]</sup> For this to occur, a reduction in the external diameter of the blood vessel is required. Restructuring of the vessel wall is the consequence of several events, which

appear to include increased vasoconstriction, increased matrix deposition, increased apoptosis in the periphery of the vessel with enhanced growth toward the lumen, and changes in motility of smooth muscle cells.<sup>[41][42][43]</sup>

Ang II stimulates vascular smooth muscle cell hypertrophy and hyperplasia, extracellular matrix production, and collagen degradation, which contribute to the remodelling of resistance vessels.<sup>[39]</sup> Ang II, via NAD(P)H oxidase, also has significant pro inflammatory actions in the vascular wall, stimulating the production of reactive oxygen species, such as superoxide ( $O_2^{2-}$ ) and  $H_2O_2$ , cytokines, adhesion molecules, and activation of redox-sensitive inflammatory genes.<sup>[44][45]</sup> Vascular  $O_2^{2-}$  and  $H_2O_2$  influence redox-sensitive signaling molecules that regulate the vascular smooth muscle cell responses involved in remodeling.<sup>[39]</sup>

The significance of Ang II in the pathogenesis of vascular remodeling was reinforced in studies of human hypertension in which treatment with an ACE inhibitor or ARB, but not a  $\beta$ -blocker, corrected the structure and improved the function of small arteries.<sup>[39]</sup> This response appeared to be independent of the BP, which was equivalent in each treatment group. Activation of peroxisome proliferator-activated receptors (PPARs), which participate in the regulation of cell growth and migration, oxidant stress, and inflammation in the cardiovascular system<sup>[46]</sup> may also attenuate Ang II-mediated vascular remodeling.<sup>[47][48]</sup>

## **Impaired Pressure Natriuresis**

Disruption of the pressure natriuresis relationship is a fundamental aspect of human hypertension and all experimental models of hypertension.

## **Sympathetic Nervous System**

Several studies suggest that patients with primary hypertension have impaired circulatory homeostasis with abnormal vascular reactivity. When monitored for 24 hours, these patients generally show a resetting of their diurnal BP profile to a higher level, with somewhat wider than normal fluctuations in BP.<sup>[49]</sup> They may also have a wider BP response to various psychic or physical stimuli. They may exhibit abnormal responses (fainting) to venous occlusion of the legs and can exhibit such other vasomotor phenomena as increased flushing, tachycardia, and sweating in response to various stimuli.<sup>[50]</sup> These phenomena are not necessarily or consistently related to the hypertension itself. They may reflect a relative instability of the individual's circulation compared with that of normotensive people, whose BP level is closer to the midpoint of defensive buffering systems that protect against assaults on the circulation.

## **COMPLICATIONS OF HT**

### **Cardiovascular Disease**

Life insurance statistics have established that hypertensive individuals, as a population, have shortened survival and that this vulnerability correlates broadly with

increasing levels of arterial BP.<sup>[53]</sup> The Framingham Heart Study<sup>[51] [52]</sup> confirmed these findings and demonstrated that high BP is a leading risk factor predisposing to stroke, HF, heart attack, and kidney failure. Whether and how soon these complications occur in a specific hypertensive patient appears to be strongly determined by the concurrence of other risk factors, such as LVH, diabetes, smoking and hypercholesterolemia.

## **Heart Failure**

Hypertension is the most common condition antedating HF, with a two- to threefold higher risk than for normotensive subjects and with a graded increase in risk at higher pressure.<sup>[55] [56]</sup> In the Framingham population,<sup>[54]</sup> more than 90% of patients with symptomatic HF had a history of hypertension. Systolic, diastolic, and pulse pressures were related to the risk of HF in the Framingham cohort,<sup>[55]</sup> but the relation was strongest for systolic and pulse pressures. This risk is amplified further when ECG-LVH or echo-LVH are present

## **Kidney**

Primary renal disease is the most common etiology of secondary hypertension. Conversely, hypertension is a risk factor for renal injury and ESRD. The increased risk associated with high blood pressure is graded, continuous, and present throughout the entire distribution of blood pressure above optimal. Renal risk appears to be more closely related to systolic than to diastolic blood pressure, and black men are at greater



risk than white men for developing ESRD at every level of blood pressure.

## **Microalbuminuria**

*Microalbuminuria*, defined as urinary albumin excretion between 30 and 299 mg/mg creatinine, is the earliest sign of nephropathy.<sup>[57]</sup> It occurs in approximately 6% to 40% of individuals with primary hypertension, with the prevalence increasing with age and duration of hypertension.<sup>[58] [59]</sup> Microalbuminuria is a well-defined marker for increased risk of CVD in patients with diabetes . In a study of more than 11,000 nondiabetic individuals with hypertension, the presence of microalbuminuria was associated with a significantly higher prevalence of LVH, coronary artery disease, MI, hyperlipidemia, and peripheral vascular disease.<sup>[60]</sup> These findings relating albumin excretion with adverse outcomes in nondiabetic subjects have been reported in other large-population studies.<sup>[59]</sup>

## **Brain**

Hypertension is an important risk factor for brain infarction and hemorrhage. Approximately 85% of strokes are due to infarction and the remainder are due to hemorrhage, either intracerebral hemorrhage or subarachnoid hemorrhage. The incidence of stroke rises progressively with increasing blood pressure levels, particularly systolic blood pressure in individuals >65 years. Treatment of hypertension convincingly decreases the incidence of both ischemic and hemorrhagic strokes.

Hypertension is also associated with impaired cognition in an aging population, and longitudinal studies support an association between mid-life hypertension and late-life cognitive decline. Hypertension-related cognitive impairment and dementia may be a consequence of a single infarct due to occlusion of a "strategic" larger vessel or multiple lacunar infarcts due to occlusive small vessel disease resulting in subcortical white matter ischemia. Several clinical trials suggest that antihypertensive therapy has a beneficial effect on cognitive function, although this remains an active area of investigation.

## **PERIPHERAL ARTERIES**

In addition to contributing to the pathogenesis of hypertension, blood vessels may be a target organ for atherosclerotic disease secondary to long-standing elevated blood pressure. Hypertensive patients with arterial disease of the lower extremities are at increased risk for future cardiovascular disease. Although patients with stenotic lesions of the lower extremities may be asymptomatic, intermittent claudication is the classic symptom of PAD. This is characterized by aching pain in the calves or buttocks while walking that is relieved by rest. The ankle-brachial index is a useful approach for evaluating PAD and is defined as the ratio of noninvasively assessed ankle to brachial (arm) systolic blood pressure. An ankle-brachial index  $< 0.90$  is considered diagnostic of PAD and is associated with  $>50\%$  stenosis in at least one major lower limb vessel. Several studies suggest that an ankle-brachial index  $< 0.80$  is associated with elevated

blood pressure, particularly systolic blood pressure.

## **RETINOPATHY**

Retina is an end organ which is frequently damaged in hypertensives<sup>(67)</sup>

Retina is a window to the vascular remodelling happening in the pathogenesis of hypertension as described previously. There are various classification System proposed for hypertensive retinopathy. In our study we have applied the most widely used Keith wagner's system of grading hypertensive retinopathy.

### **KEITH WAGNER'S GRADING**

<b>STAGE</b>	<b>FEATURES</b>
GRADE 1	Arteriolar narrowing, tortuosity, silver/copper wiring
GRADE 2	AV nipping
GRADE 3	Flame shaped haemorrhages,cotton wool spots and hard exudates
GRADE 4	PAPILLOEDEMA

The prevalence of retinopathy varies among the population studied. In a Study in India chowta et al<sup>(62)</sup> the prevalence was 33% among the hypertensives

## DEFINING HYPERTENSION

Table below provides a classification of BP for adults 18 years and older<sup>(8)</sup>. The classification is based on the average of two or more properly measured, seated, BP readings on each of two or more office visits.

Prehypertension is **not** a disease category<sup>(8)</sup>. Rather, it is a designation chosen to identify individuals at high risk of developing hypertension, so that both patients and clinicians are alerted to this risk and encouraged to intervene and prevent or delay the disease from developing.

<b>BLOOD PRESSURE CLASSIFICATION</b>	<b>SYSTOLIC BP</b>	<b>DIASTOLIC BP</b>
NORMAL	<120	and <80
PREHYPERTENSION	120-139	Or 80-99
STAGE 1 HT	140-159	Or 100-109
STAGE 2 HT	≥160	Or ≥ 110

### JNC-7 CLASSIFICATION

Impressive evidence has accumulated to warrant greater attention to the importance of SBP as a major risk factor for CVD<sup>(63)</sup>. Changing patterns of BP occur with increasing age. The rise in SBP continues throughout life in contrast to DBP, which rises until approximately age 50, tends to level off over the next decade, and may remain the same or fall later in life. Diastolic hypertension predominates before age 50, either alone or in combination with SBP elevation. The prevalence of systolic hypertension

increases with age, and above 50 years of age, systolic hypertension represents the most common form of hypertension. DBP is a more potent cardiovascular risk factor than SBP until age 50 thereafter, SBP is more important<sup>(63)</sup>. If the rise in BP with age could be prevented or diminished, much of hypertension, cardiovascular and renal disease, and stroke might be prevented. A number of important causal factors for hypertension have been identified, including excess body weight; excess dietary sodium intake, reduced physical activity; inadequate intake of fruits, vegetables, and potassium; and excess alcohol intake<sup>(64)</sup>

### **Essential Hypertension**

Essential hypertension tends to be familial and is likely to be the consequence of an interaction between environmental and genetic factors. The prevalence of essential hypertension increases with age, and individuals with relatively high blood pressures at younger ages are at increased risk for the subsequent development of hypertension. It is likely that essential hypertension represents a spectrum of disorders with different underlying pathophysiologies. In the majority of patients with established hypertension, peripheral resistance is increased and cardiac output is normal or decreased; however, in younger patients with mild or labile hypertension, cardiac output may be increased and peripheral resistance may be normal.

When plasma renin activity (PRA) is plotted against 24-h sodium excretion, ~10–15% of hypertensive patients have high PRA and 25% have low PRA. High-renin

patients may have a vasoconstrictor form of hypertension, whereas low-renin patients may have a volume-dependent hypertension. Inconsistent associations between plasma aldosterone and blood pressure have been described in patients with essential hypertension. The association between aldosterone and blood pressure is more striking in African Americans, and PRA tends to be low in hypertensive African Americans. This raises the possibility that subtle increases of aldosterone may contribute to hypertension in at least some groups of patients who do not have overt primary aldosteronism.

## **METABOLIC SYNDROME**

The IDF criteria for diagnosing metabolic syndrome has been used for this study

(65)

Metabolic syndrome is defined in the presence of waist circumference of more than 90cm for male and 80 cm for female with any of the following 2 criteria.

1. A triglyceride level of more than 150mg/dl or specific medication
2. a fasting plasma glucose of >100mg/dl or a known type2 diabetic
3. a known HT or BP>135/85mm of HG
4. HDL cholesterol<40 and <50mg/dL for men and female respectively.

## **ASSOCIATED DISEASES WITH METABOLIC SYNDROME**

## **Cardiovascular Disease**

The relative risk for new-onset CVD in patients with the metabolic syndrome, in the absence of diabetes, averages between 1.5- and threefold. In an 8-year follow-up of middle-aged men and women in the Framingham Offspring Study (FOS), the population attributable risk for patients with the metabolic syndrome to develop CVD was 34% in men and 16% in women. In the same study, both the metabolic syndrome and diabetes predicted ischemic stroke with greater risk for patients with the metabolic syndrome than for diabetes alone (19% vs 7%), particularly in women (27% vs 5%)<sup>(66)</sup> Patients with metabolic syndrome are also at increased risk for peripheral vascular disease.

## **Type 2 Diabetes**

Overall, the risk for type 2 diabetes in patients with the metabolic syndrome is increased three- to fivefold. In the FOS's 8-year follow-up of middle-aged men and women, the population-attributable risk for developing type 2 diabetes was 62% in men and 47% in women.

## **Etiology**

### **Insulin Resistance**

The most accepted and unifying hypothesis to describe the pathophysiology of the metabolic syndrome is insulin resistance, caused by an incompletely understood defect in insulin action. The onset of insulin resistance is heralded by postprandial hyperinsulinemia, followed by fasting hyperinsulinemia and, ultimately, hyperglycemia.

An early major contributor to the development of insulin resistance is an overabundance of circulating fatty acids . Plasma albumin-bound free fatty acids (FFAs) are derived predominantly from adipose tissue triglyceride stores released by hormone-sensitive lipase. Fatty acids are also derived through the lipolysis of triglyceride-rich lipoproteins in tissues by lipoprotein lipase (LPL). Insulin mediates both antilipolysis and the stimulation of LPL in adipose tissue. Of note, the inhibition of lipolysis in adipose tissue is the most sensitive pathway of insulin action<sup>(67)</sup>. Thus, when insulin resistance develops, increased lipolysis produces more fatty acids, which further decrease the antilipolytic effect of insulin. Excessive fatty acids enhance substrate availability and create insulin resistance by modifying downstream signaling. Fatty acids impair insulin-mediated glucose uptake and accumulate as triglycerides in both skeletal and cardiac muscle, whereas increased glucose production and triglyceride accumulation are seen in liver.

The oxidative stress hypothesis provides unifying theory for aging and the



predisposition to the metabolic syndrome. In studies carried out in insulin-resistant subjects with obesity or type 2 diabetes, in the offspring of patients with type 2 diabetes, and in the elderly, a defect has been identified in mitochondrial oxidative phosphorylation, leading to the accumulation of triglycerides and related lipid molecules in muscle. The accumulation of lipids in muscle is associated with insulin resistance.

## **OBESITY**

Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health. A crude population measure of obesity is the body mass index (BMI), a person's weight (in kilograms) divided by the square of his or her height (in metres). A person with a BMI of 25 or more is generally considered obese. A person with a BMI equal to or more than 23 is considered overweight<sup>(68)</sup>.

Overweight and obesity are major risk factors for a number of chronic diseases, including diabetes, cardiovascular diseases and cancer. Once considered a problem only in high income countries, overweight and obesity are now dramatically on the rise in low- and middle-income countries, particularly in urban settings.

Recent guidelines have reduced the cut-off for Indian population based on various studies. The new WHO Asia Pacific guidelines defines generalised obesity as BMI>25 and abdominal obesity as a waist circumference of more than 90 for males and 80 for females.

<b>BMI</b>	<b>CLASSIFICATION</b>
<18.5	UNDERWEIGHT
18.5-22.9	NORMAL
23-24.9	OVERWEIGHT
25-29.9	OBESE 1
30 AND ABOVE	OBESE 2

classification of weight by BMI for adult asians

## **ERECTILE DYSFUNCTION**

Erectile dysfunction (ED) is defined as the persistent inability to achieve and maintain an erection sufficient to permit satisfactory sexual intercourse<sup>(69)</sup> Incidence and prevalence of ED are considerable and awareness is growing that the condition is treatable. Despite the increasing demand for clinical services and the potential impact of ED and other sexual disorders, on interpersonal relationships and quality of life, epidemiological data are relatively scarce.

The prevalence of ED depends on the population studied and the definition and methods used. Since ED often accompanies aging and is associated with chronic illness, such as diabetes mellitus, heart disease, hypertension, and a variety of neurological diseases, very few studies have been carried out to establish the incidence and prevalence of this condition in a healthy population. In a community-based survey of men between the ages of 40 and 70 y, 52% of the respondents reported some degree of erectile difficulty

In the present study the abridged 5-item version of the International Index of Erectile Function (IIEF-5)<sup>(70)</sup> as a diagnostic tool for establishing the prevalence of ED is used in the Evaluation of newly diagnosed males.

In one study it was conclusively proved that HT patients had not only more incidence of ED but also more severe form of ED<sup>(71)</sup>.

In another study the prevalence of ED among patients with metabolic syndrome was proven to be higher. At present it has been accepted that HT and MS are independent risk factors for the development of ED.

Various questionnaire has been used to identify ED among the population. The widely used International Index of Erectile Function-5 questionnaire<sup>(70)</sup> has been used in our study.

The use of simplified version of the IIEF questionnaire has been proved to be useful and reliable in the evaluation of ED in large number of men. <sup>(70)</sup>

## IIEF-5 scoring system

	Score				
Over the past six months:	1	2	3	4	5
How do you rate your confidence that you could get and keep an erection?	Very low	Low	Moderate	High	Very high
When you had erections with sexual stimulation, how often were your erections hard enough for penetration?	Almost never or never	Much less than half the time	About half the time	Much more than half the time	Almost always or always
During sexual intercourse, how often were you able to maintain your erection after you had penetrated (entered) your partner?	Almost never or never	Much less than half the time	About half the time	Much more than half the time	Almost always or always
During sexual intercourse how difficult was it to maintain your erection to the completion of intercourse?	Extremely difficult	Very difficult	Difficult	Slightly difficult	Not difficult
When you attempted sexual intercourse, how often was it satisfactory for you?	Almost never or never	Much less than half the time	About half the time	Much more than half the time	Almost always or always
The IIEF-5 score is the sum of questions 1 to 5. The lowest score is 5 and the highest score 25.					

**severe (5-7), moderate (8-11), mild to moderate (12-16), mild (17-21), and no ED (22-25).**

## LIPID ABNORMALITIES

Dyslipidemias are more common in patients with metabolic syndrome and in HT. However the distribution of lipid abnormalities among newly detected hypertensives is not well studied. Dyslipidemias constitute one the major risk factor for coronary heart disease as proven by the Framingham heart studies.

Current lipid abnormalities are defined by Adult Treatment Panel 3(ATP3) guidelines published by the National Cholesterol Education Programme (NCEP) Of United states<sup>(72)</sup>

### **ATP III Classification of LDL, Total, and HDL Cholesterol (mg/dL)**

LDL CHOLESTEROL	
<100	OPTIMAL
100-129	NEAR OPTIMAL/ABOVE OPTIMAL
130-159	BORDERLINE HIGH
160-189	HIGH
>_190	VERY HIGH
TOTAL CHOLESTEROL	
<200	DESIRABLE
200-239	BORDERLINE HIGH
>240	HIGH
HDL	
<40	LOW
>60	HIGH

From NCEP guidelines.

## FRAMINGHAM RISK SCORE

### **Estimating 10-Year Risk for Men and Women**

Risk assessment for determining the 10-year risk for developing CHD is carried out using Framingham risk scoring<sup>(72)</sup>. The risk factors included in the Framingham calculation of 10-year risk are: age, total cholesterol, HDL cholesterol, systolic blood pressure, treatment for hypertension, and cigarette smoking. The first step is to calculate the number of points for each risk factor. For initial assessment, values for total cholesterol and HDL cholesterol are required. Because of a larger database, Framingham estimates are more robust for total cholesterol than for LDL cholesterol. Total cholesterol and HDL cholesterol values should be the average of at least two measurements obtained. The blood pressure value used is that obtained at the time of assessment, regardless of whether the person is on anti-hypertensive therapy. However, if the person is on antihypertensive treatment, an extra point is added beyond points for the blood pressure reading because treated hypertension carries residual risk. The average of several blood pressure measurements, as recommended by the Joint National Committee (JNC), is needed for an accurate measure of baseline blood pressure. The designation “smoker” means any cigarette smoking in the past month. The 10-year risk for myocardial infarction and coronary death (hard CHD) is estimated from total points, and the person is categorized according to absolute 10-year risk

## Risk Assessment Tool for Estimating 10-year Risk of Developing Hard CHD (Myocardial Infarction and Coronary Death)

The [risk assessment tool](#) below uses recent data from the Framingham Heart Study to estimate 10-year risk for “hard” coronary heart disease outcomes (myocardial infarction and coronary death). This tool is designed to estimate risk in adults aged 20 and older who do not have heart disease or diabetes. Use the calculator below to estimate 10-year risk.

Age:  years

Gender:  Female  Male

[Total Cholesterol:](#)  mg/dL






[HDL Cholesterol:](#)  mg/dL

[Smoker:](#)  No  Yes

[Systolic Blood Pressure:](#)  mm/Hg

Currently on any medication to treat high blood pressure.  No  Yes

Calculate 10-Year Risk

-  **TOP Total cholesterol** - Total cholesterol values should be the average of at least two measurements obtained from lipoprotein analysis.
-  **TOP HDL cholesterol** - HDL cholesterol values should be the average of at least two measurements obtained from lipoprotein analysis.
-  **TOP Smoker** - The designation “smoker” means any cigarette smoking in the past month.
-  **TOP Systolic blood pressure** - The blood pressure value used is that obtained at the time of assessment, regardless of whether the person is on antihypertensive therapy (treated hypertension carries residual risk).
-  **TOP More Information** - Determining 10-year (short term) risk for developing CHD is carried out using Framingham risk scoring. The risk factors included in the Framingham calculation are age, total cholesterol, HDL cholesterol, systolic blood pressure, treatment for hypertension, and cigarette smoking. Because of a larger database, Framingham estimates are more robust for total cholesterol than for LDL cholesterol. Note, however, that LDL cholesterol remains the primary target of therapy. The Framingham risk score gives estimates for “hard CHD” which includes myocardial infarction and coronary death.

# **MATERIALS AND METHODS**

## **SETTING**

This study is conducted in the Hypertension clinic of Madras Medical College and Government General Hospital in collaboration with the Institute of Biochemistry.

## **ETHICAL APPROVAL**

Obtained

## **STUDY DURATION**

This study was conducted over a period of 6 months from May2009 to October2009.

## **STUDY POPULATION**

Patients attending the hypertensive clinic who are yet to be started on any Drugs.

## **TYPE OF STUDY**

It's a cross sectional study of patients attending hypertension clinic.

## **INCLUSION CRITERIA**

1. Age greater than 18 years.



2. patients newly diagnosed as hypertensives in out patient department

## **EXCLUSION CRITERIA**

1. Age less than 18yrs
2. known hypertensives who have discontinued drugs and have now come for treatment or with complications
3. Known DM patient under antiproteinuria therapy
4. Known CKD/ hypothyroid
5. Known case of secondary HT or during evaluation found to have secondary causes.
6. Patients who are already under anti hypertensive drugs for other indications like CAHD,etc.

## **SAMPLE SIZE**

100 patients who came for the registration were taken for the study.

## **METHODS**

All patients underwent CBC,GTT, serum electrolytes, urea, creatinine, LFT, fasting lipid profile, serum calcim, uric acid, ecg, echo, USG abdomen,urine for microalbuminuria after giving written consent. A detailed examination to ruleout

secondary causes were done. BP was measured and staged. The classification is based on the average of two or more properly measured, seated BP readings on each of two or more office visits.

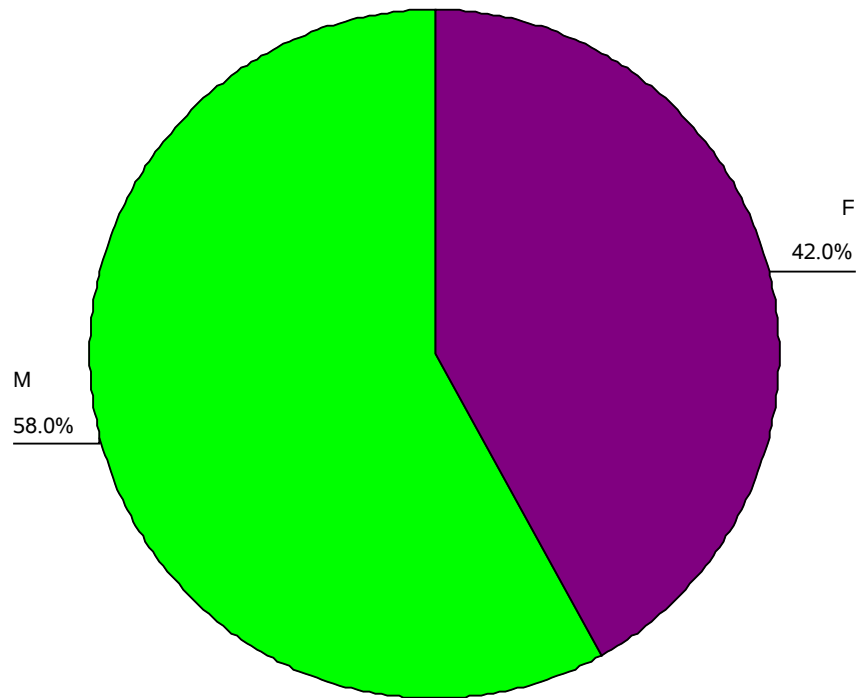
A standard proforma was used to record all variables for every individual patient.

## **STATISTICAL ANALYSIS**

Data analysis was done with use of SPSS software version 10. Descriptive statistics were used to calculate frequency. To examine the linear trend of proportions, trend chi-square was used and to find the test of association chi-square was computed.

# RESULTS

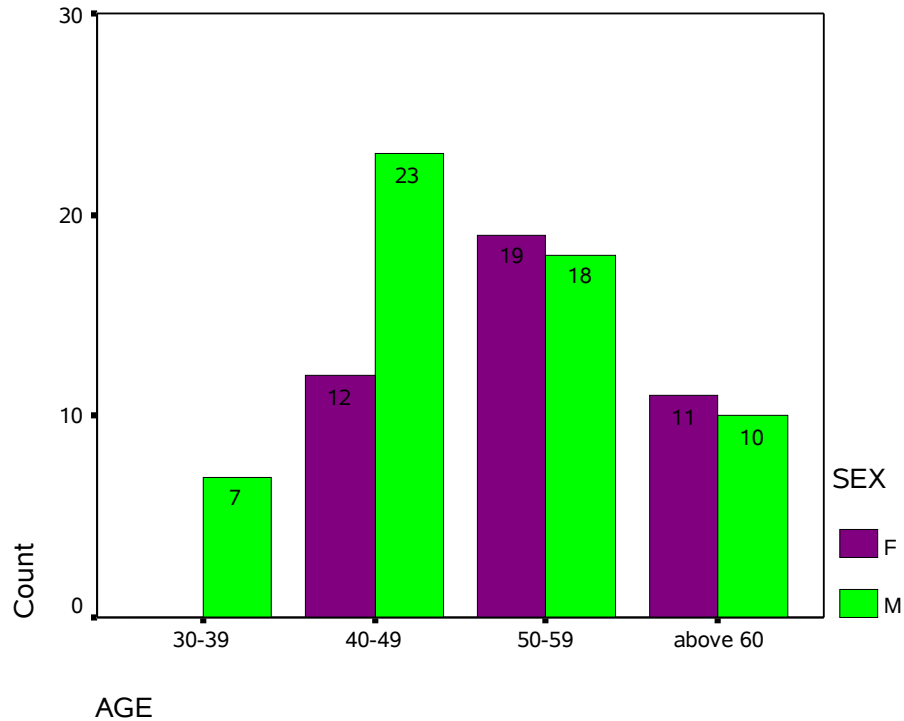
## SEX DISTRIBUTION OF THE HYPERTENSIVE PATIENTS



In our study the male to female ratio is 1.38:1

SEX	NO OF PATIENTS
FEMALE	42
MALE	58

**AGE GROUP & NEWLY DIAGNOSED HYPERTENSIVES**

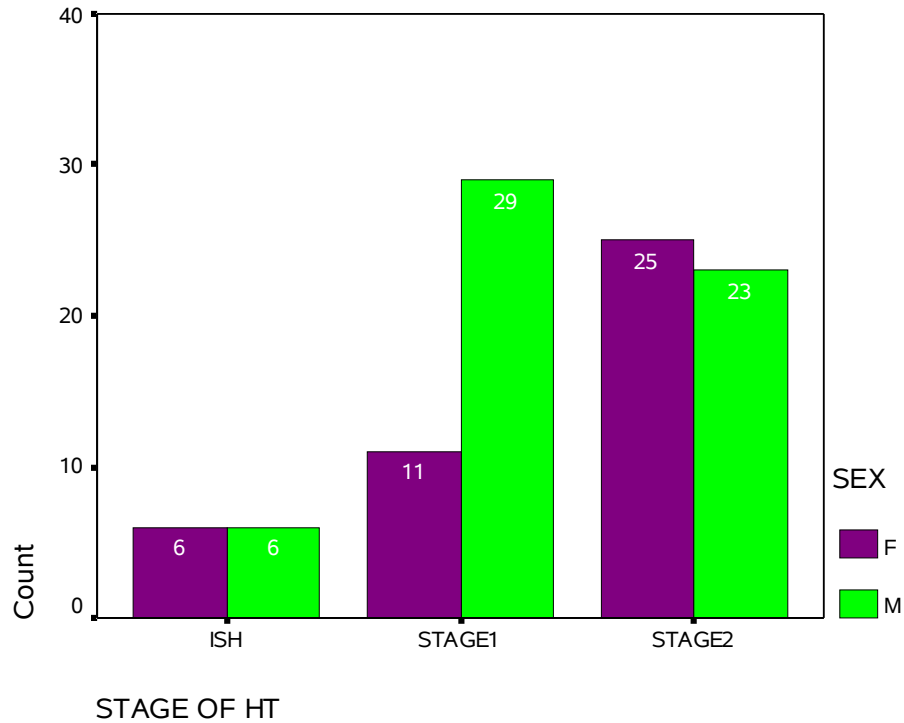


**TABLE SHOWING THE AGE DISTRIBUTION IN NEWLY DIAGNOSED HYPERTENSIVES**

AGE	30-39	40-49	50-59	>60	TOTAL
F	-	12	19	11	42
M	7	23	18	10	58
RATIO	-	1.9:1	0.9:1	0.9:1	1.38:1

In our study 45% of female are in age group of 50 to 59 whereas 39% of males are in age group of 40 to 49. The p value was <0.05 significant.

## STAGES OF HYPERTENSION AMONG THE SEXES

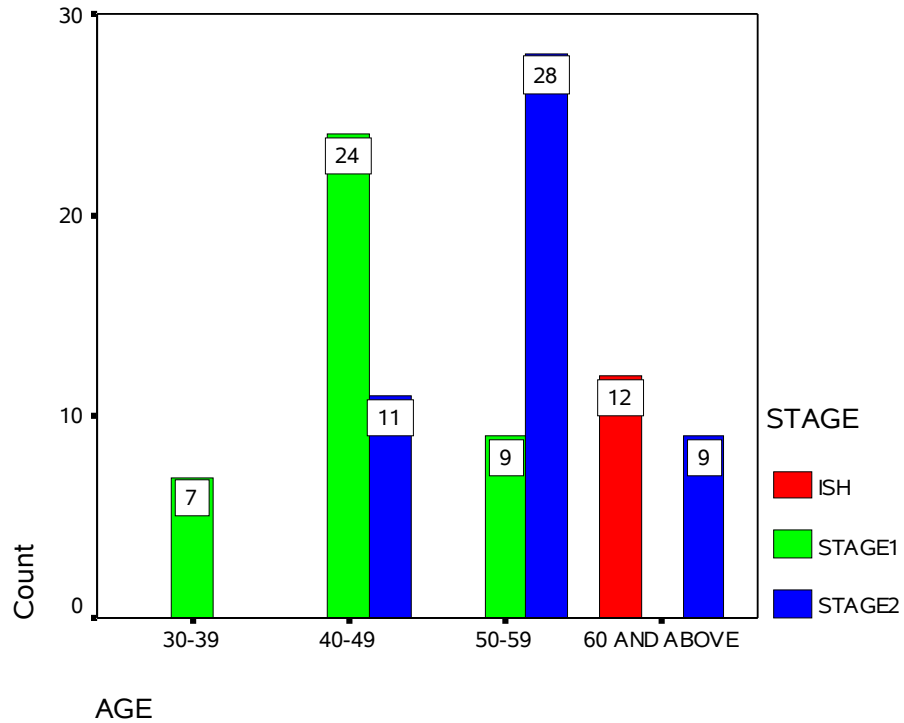


**TABLE SHOWING THE DISTRIBUTION OF HT STAGE AMONG THE SEXES**

	ISH	STAGE1	STAGE2	TOTAL
F	6	11	25(59.5%)	42
M	6	29(50%)	23	58

In our study females tend to present in stage 2 HT( 59.5%) , whereas males tend to present a little earlier in stage 1 HT (50%). The P value was <0.05.

**AGE WISE DISTRIBUTION OF HT**

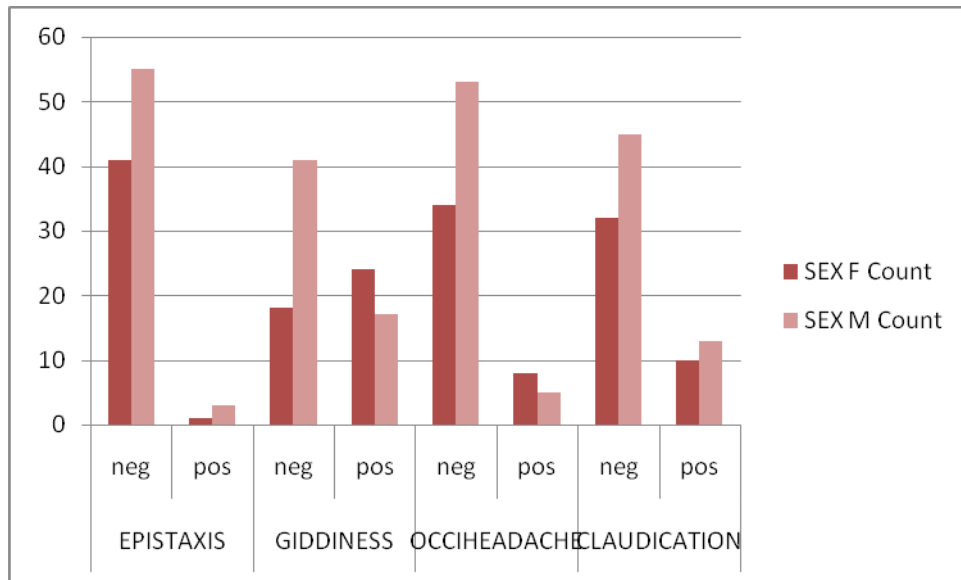


**TABLE SHOWING THE AGE WISE DISTRIBUTION OF STAGE OF HT**

STAGE	AGE			
	30-39	40-49	50-59	60& ABOVE
ISH	0	0	0	12
STAGE1	7	24	9	0
STAGE2	0	11	28	9

In this study the prevalence of ISH was 12% and almost all exclusively occurred in patients of more than 60 yrs

## SYMPTOM DISTRIBUTION AMONG THE HYPERTENSIVES

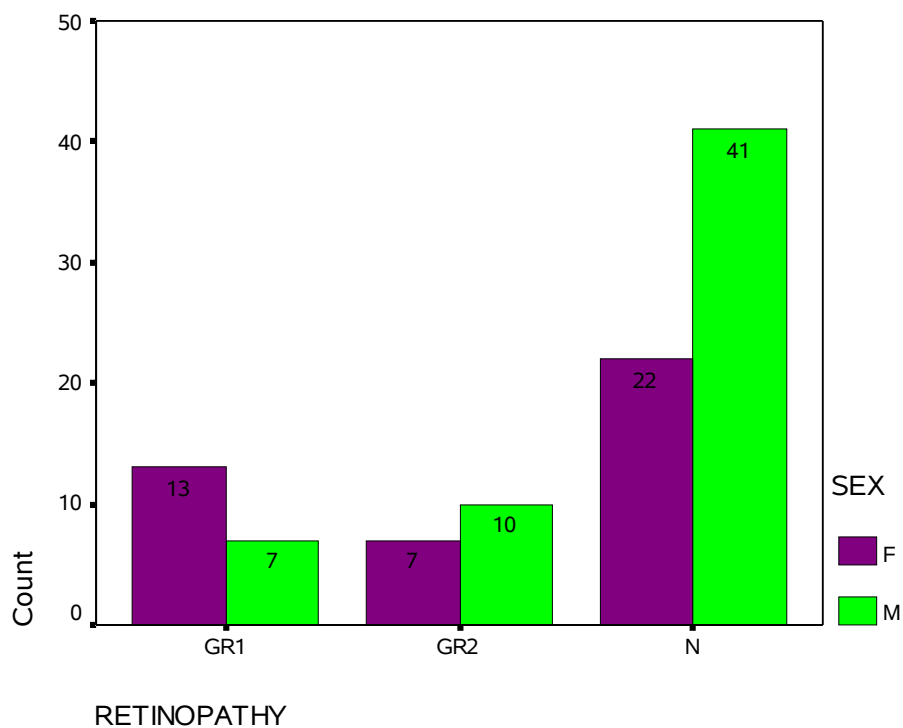


**TABLE SHOWING THE DISTRIBUTION OF SYMPTOMS**

		SEX			
		F	%	M	%
EPISTAXIS	No	41		55	
	yes	1	3%	3	5%
GIDDINESS	No	18		41	
	yes	24	57%	17	29%
OCCIPITAL HEADACHE	No	34		53	
	yes	8	19%	5	9%
CLAUDICATION	No	32		45	
	yes	10	24%	13	22%
ASYMPTOMATIC		13	31%	30	52%

In our study the predominant symptom was giddiness but however males tend to present asymptomatic more.

## RETINOPATHY IN NEWLY DETECTED HYPERTENSIVES



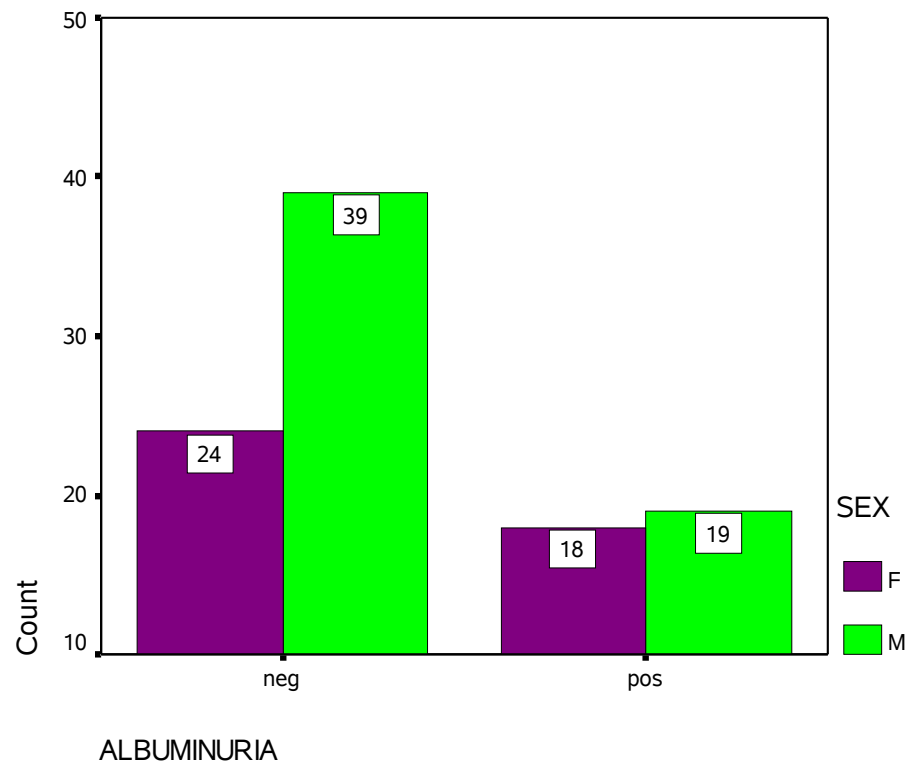
**TABLE SHOWING THE DISTRIBUTION OF RETINOPATHY**

SEX	RETINOPATHY	TOTAL	PERCENTAGE
M	17	58	29%
F	20	42	47.6%

In our study the prevalence of retinopathy among the females was 47.6% and among the males was 29%. The P value was  $<0.05$  and it is statistically significant.



## MICROALBUMINURIA AMONG NEWLY DETECTED HYPERTENSIVES

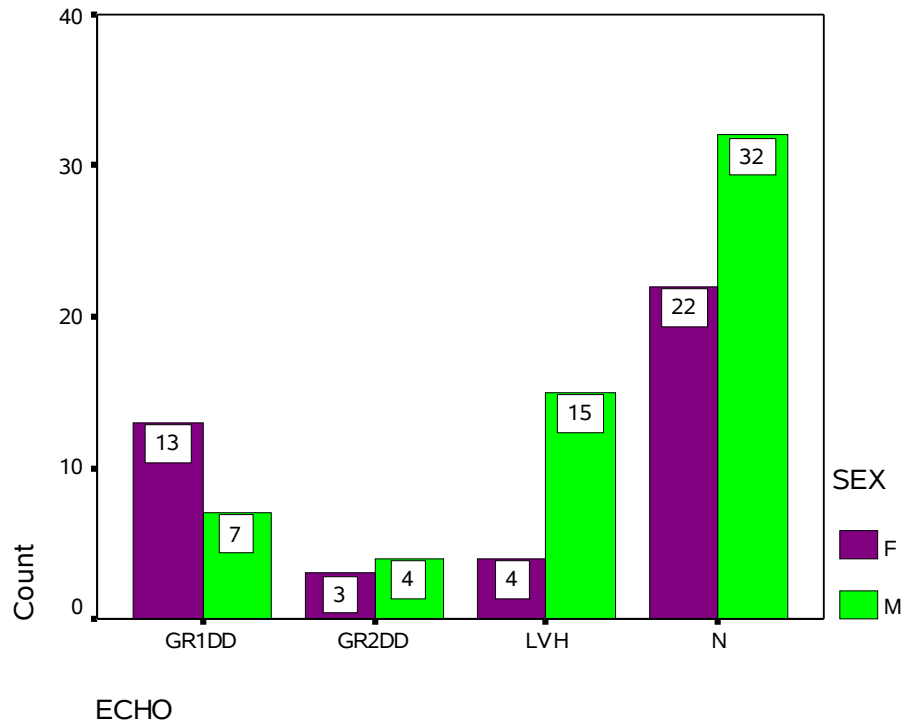


### DISTRIBUTION OF MICRO ALBUMINURIA

MICRO ALBUMINURIA	Nil	present	PERCENTAGE	TOTAL
F	24	18	42.90%	42
M	39	19	32.70%	58

In our study the prevalence of albuminuria was 42.9% among females and 32.7% in males with a p value of  $>0.05$  not statistically significant.

## CARDIAC INVOLVEMENT IN NEW HT

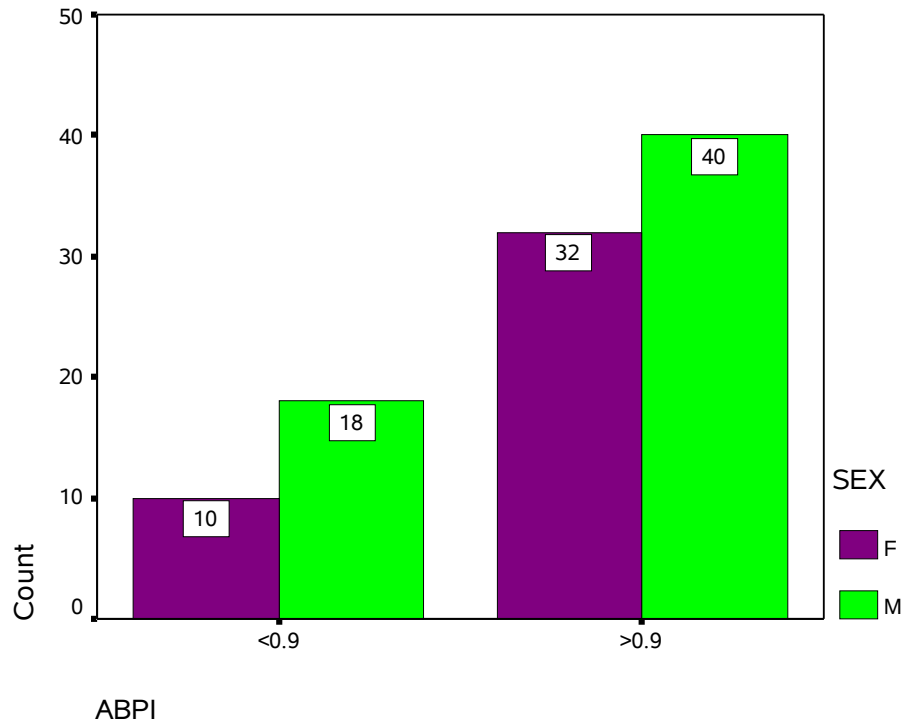


## DISTRIBUTION OF CARDIAC INVOLVEMENT

	GR1DD	GR2DD	LVH	N	TOTAL	PERCENTAGE
F	13	3	4	22	42	47.6%
M	7	4	15	32	58	44.8%

In this study the prevalence of cardiac changes was 47.6% among females and 44.8% among males with a p value of <0.05 which is statistically significant.

## PERIPHERAL VASCULAR DISEASE IN NEW HT

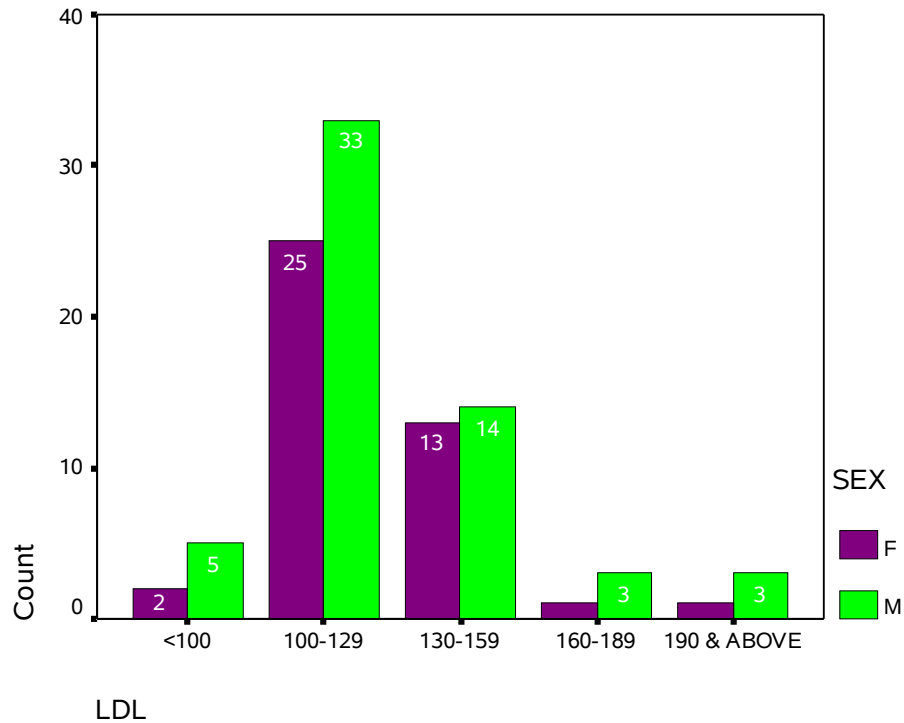


## DISTRIBUTION OF ABPI IN HT

ABPI	<0.9	>0.9	TOTAL	PERCENTAGE
F	10	32	42	23%
M	18	40	58	31%

In this study the prevalence of PVD among females was 23% and 31% among males with a p value of  $>0.05$  not significant.

## DYSLIPIDEMIA AMONG THE HT

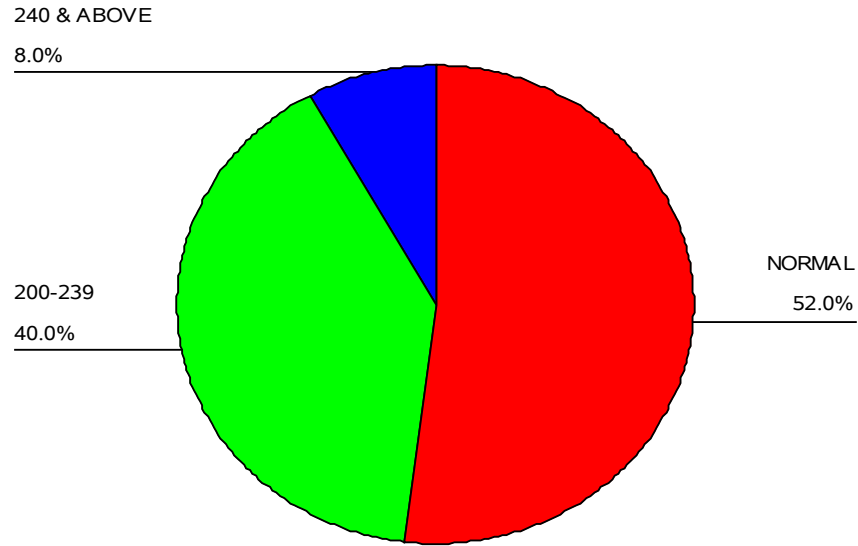


**TABLE SHOWING THE LDL ABNORMALITIES**

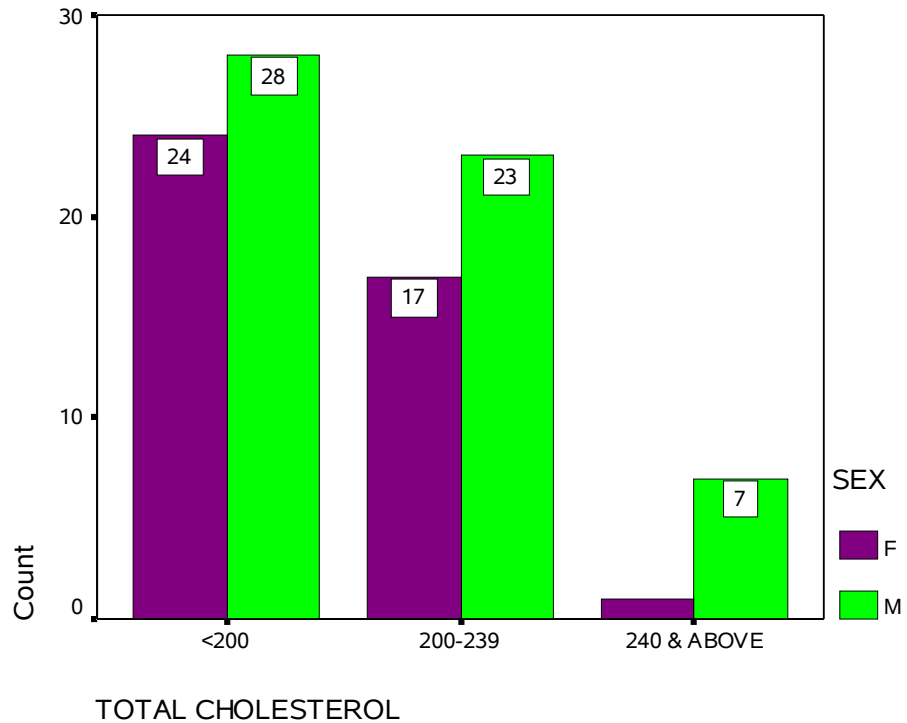
LDL	<100	100-129	130-159	160-189	190& ABOVE	%
F	2	25	13	1	1	35.7%
M	5	33	14	3	3	34.48%

In this study 35.7% of females and 34.48% of males had dyslipidemia that is a LDL level of more than 130mg/dL.

## TOTAL CHOLESTEROL AMONG THE HT



## SEX WISE DISTRIBUTION OF HYPERCHOLESTEROLEMIA

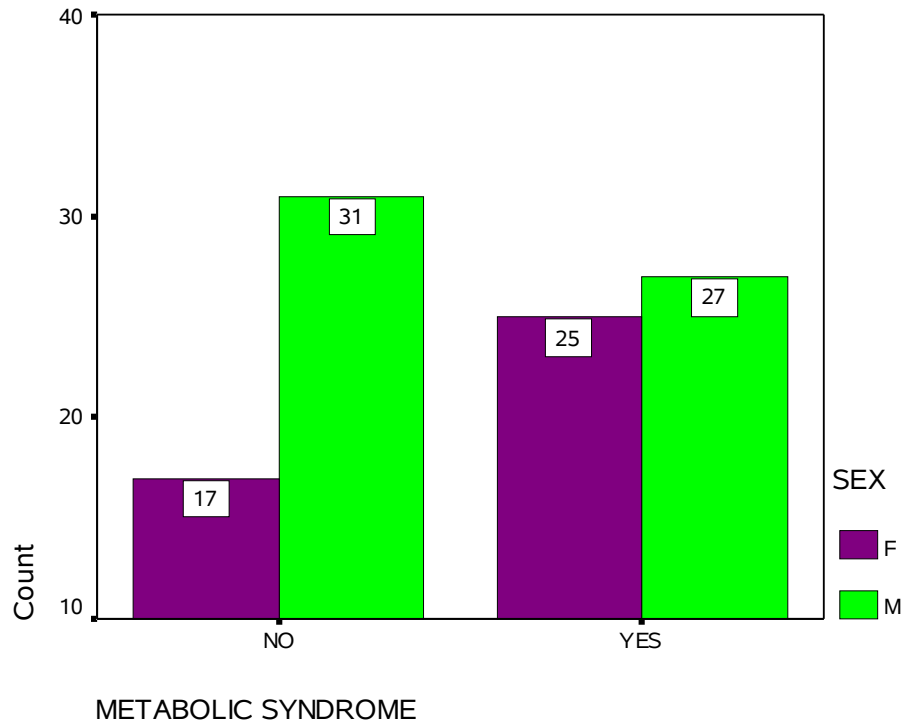


**CHOLESTEROL DISTRIBUTION AMONG THE HT**

<b>TOTAL CHOLESTEROL</b>	<b>&lt;200</b>	<b>200-239</b>	<b>240&amp; ABOVE</b>	<b>%</b>
F	24	17	1	42.8%
M	28	23	7	51.7%

In this study 48% of newly detected HT had hypercholesterolemia. 42.8% of females and 51.7% of males had hypercholesterolemia.

## METABOLIC SYNDROME AMONG THE HT

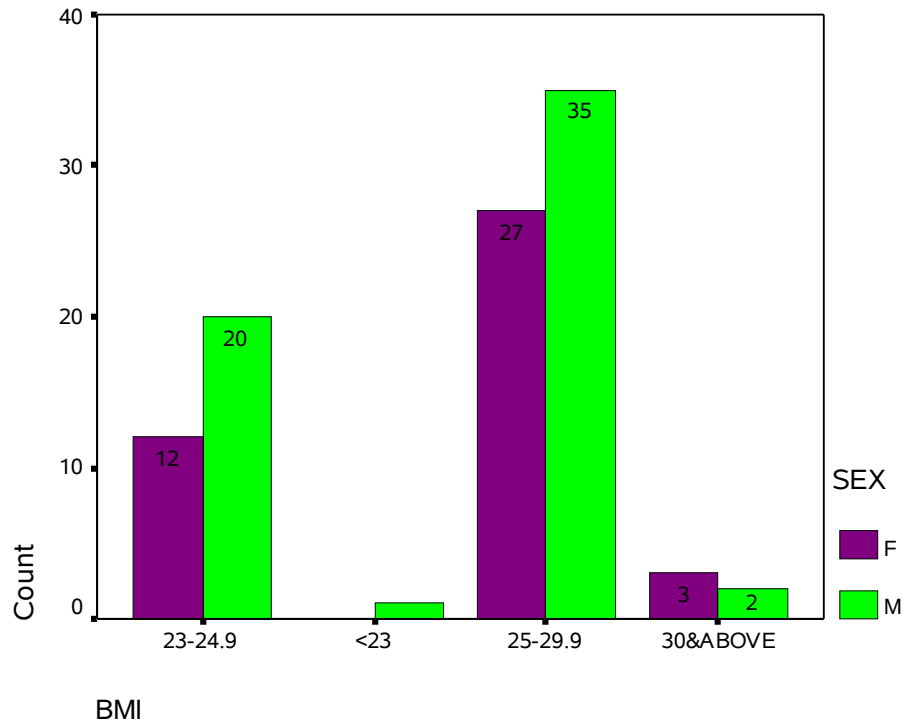


## DISTRIBUTION OF METABOLIC SYNDROME

		NO MS		MS PRESENT	
		Count	%	Count	%
SEX	F	17	40.50%	25	59.50%
	M	31	53.40%	27	46.60%

In our study 59.5% of females 46.6% of males had metabolic syndrome. The p value was  $<0.05$  and statistically significant.

## BODY MASS INDEX AMONG THE HT



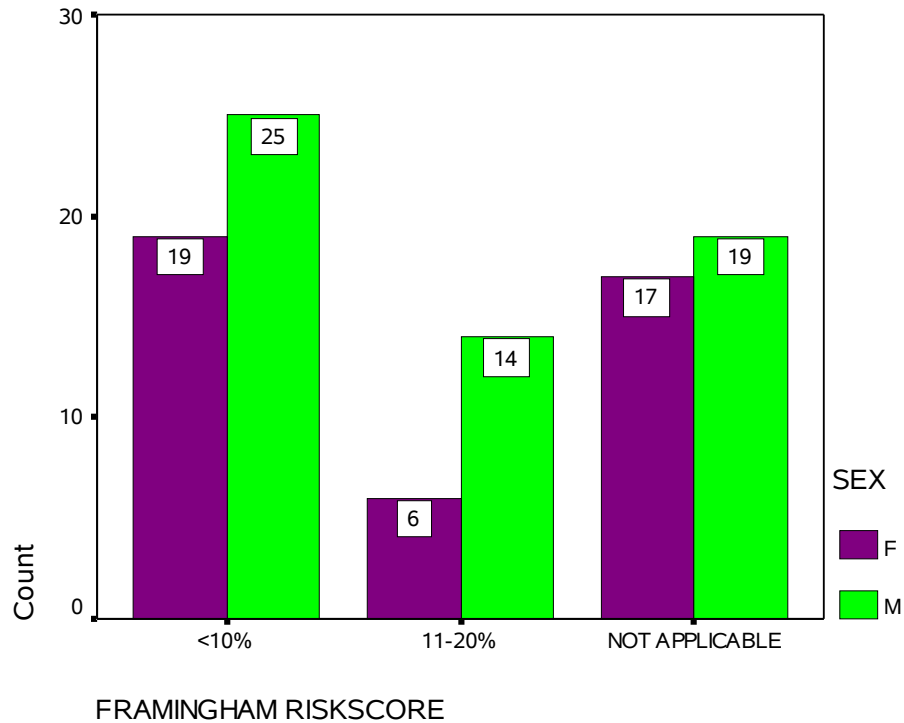
## DISTRIBUTION OF BMI GENERALISED OBESITY

	<23	23-24.9	25-29.9	30 & ABOVE	PERCENTAGE
F		12	27	3	71%
M	1	20	35	2	63%

In our study the prevalence of obesity was 67% and 71% of females and 63% of males were found to be obese.



**FRAMINGHAM RISK SCORE AMONG THE NEW HT**

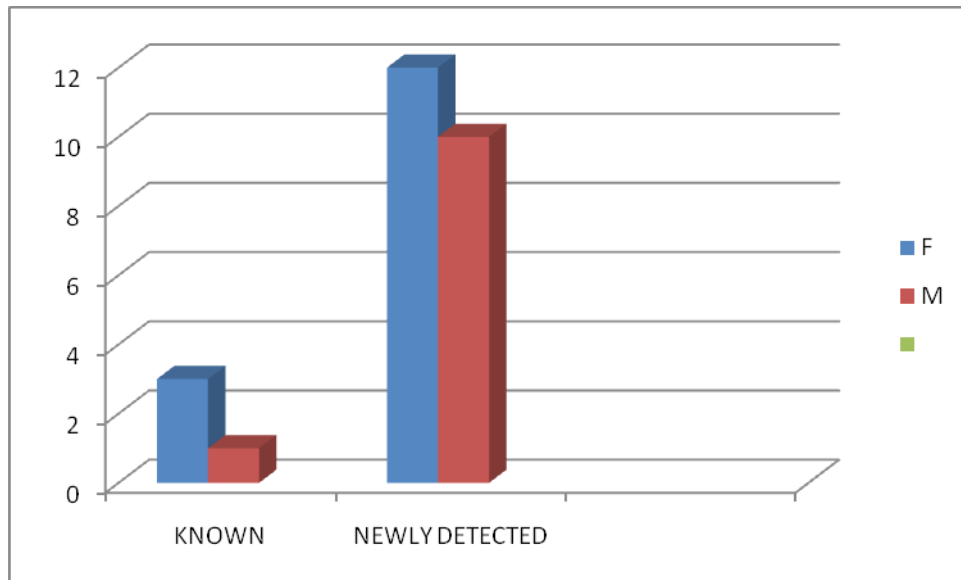


**DISTRIBUTION AMONG THE SEXES**

	<b>&lt;10%</b>	<b>11-20%</b>	<b>CHD EQUIVALENT</b>	<b>PERCENTAGE &gt;10%</b>
F	19	6	17	54.70%
M	25	14	19	56.80%

In our study 54.7% of females and 56.8% of males had a 10 years Framingham risk score of more than 10%.

## DIABETES MELLITUS AMONG THE NEW HT

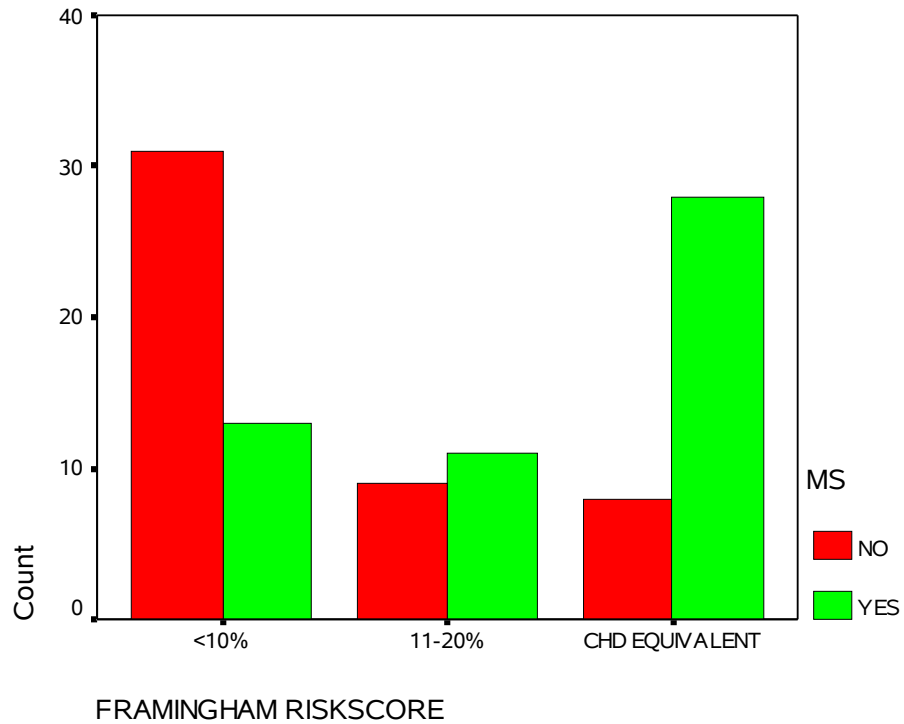


**TABLE SHOWING THE DISTRIBUTION**

SEX	KNOWN DM		NEWLY DIAGNOSED DM		TOTAL DM	%
	neg	Pos	neg	pos		
F	39	3	30	12	15	35.7%
M	57	1	48	10	11	19%

In this study 26% were diabetic out of which 22% were newly diagnosed. 35.7% of females and 19% of males were diabetic. The p value was < 0.05 and Significant.

## **METABOLIC SYNDROME WITH FRAMINGHAM RISK SCORING**



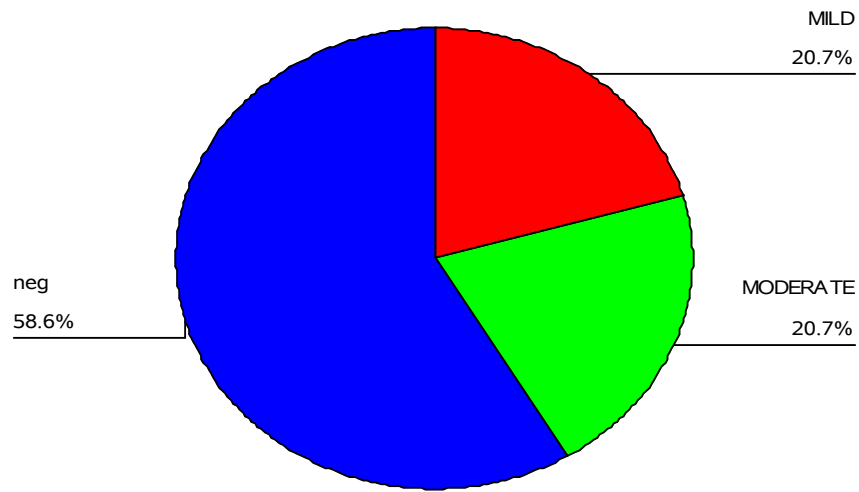
**CHD EQUIVALENT PREVALENCE AMONG PATIENTS WITH MS**

MS	RISKSCORE		CHD EQUIVALENT	>10%	TOTAL
	<10%	10-20%			
NO	31	9	8	35.4%	48
YES	13	11	28	75%	52

In our study 75% of persons with CVS risk score >10% had metabolic syndrome .

The p value was<0.01 statistically significant.

## ERECTILE DYSFUNCTION

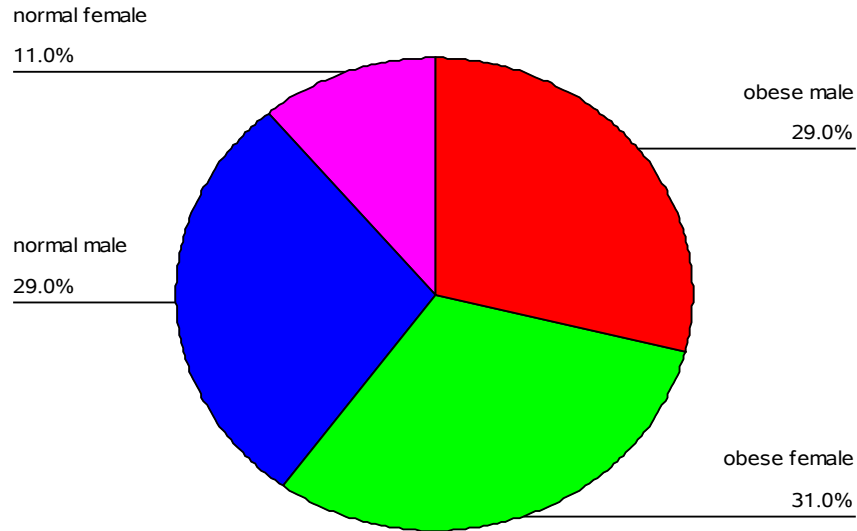


## DISTRIBUTION OF ED AMONG MALES

	MILD	MODERATE	ABSENT	PERCENTAGE
M	12	12	34	41.3%

In this study 41.3% of males had some form of erectile dysfunction.

## ABDOMINAL OBESITY



**TABLE SHOWING THE DISTRIBUTION AMONG SEXES**

WAIST CIRCUMFERENCE	ABNORMAL	NORMAL	%
F	31	11	73.8%
M	29	29	50%

In our study the prevalence of abdominal obesity was 60% of the new HT . 73.8% of females and 50% of males had abdominal obesity with a p value of  $<0.05$  which is significant.

## **DISCUSSION**

In our present study applying the JNC-7 criteria 100 newly diagnosed hypertensive patients were included after excluding secondary hypertension.

### **SEX DISTRIBUTION AND HYPERTENSION**

There were 58 males and 42 females in our study with a male to female ratio of 1.38:1. In the CURES study<sup>(1)</sup> conducted by Mohan et al in the suburban population around Chennai, after screening 2,350 individuals they found that the male to female ratio was 1.35:1. This is almost similar to our study.

### **AGE GROUP AND HYPERTENSION**

The mean age of our patient group was 52 yrs. The mean age of diagnosis for females is 54.3 and for males it is 50. However the sex ratio was not the same in different age groups. None of the females were below 40yrs of age. Similarly 45% of females presented between 50 to 59yrs of age whereas 39% of males diagnosed were between 40-49yrs of age. The p value was <0.05 and significant implying that females tend to present late.

### **STAGES OF HT AMONG THE NEWLY DETECTED PATIENTS**

In our study 40% of patients had stage 1 HT, 48% had stage 2 HT and 12% presented with isolated systolic HT. However 59.5% of females presented in stage 2 HT whereas 50% of males presented with stage 1 HT. p value was <0.05 and significant

implying, female tend to present late.

In the CURES<sup>1</sup> study by Mohan et al the prevalence of ISH among persons more than 60yrs was 25.2%. In our study the prevalence of ISH among patients aged more than 60 was 57%

#### **PREVALENCE OF ISH AMONG THE ELDERLY(>60yrs AGE)**

<b>Stage of HT</b>	<b>PRESENT STUDY</b>	<b>CURES STUDY</b>
Isolated systolic HT	57%	25.2%

#### **SYMPTOMS AMONG THE HYPERTENSIVES**

43% of our patients were completely asymptomatic implying that routine screening is necessary to identify hypertension. Among the symptomatic 57% complained of giddiness. Males (52%) tend to be asymptomatic more than females(31%). p value was <0.05 and significant. To the best of our knowledge no study compared the symptoms of HT with sexes.

#### **RETINOPATHY AND HYPERTENSION**

In our study 37% of patients had retinopathy.20% had Gr1 and 17% had Gr2 retinopathy. 47.6% of females and 29% of males presented with retinopathy.

The p value was <0.05, statistically significant. This implies that females are

diagnosed more after the onset of complication. In a study conducted by Chowta et al<sup>(62)</sup> the prevalence of retinopathy among the south Indians was 33% which is almost similar to our study.

### **MICROALBUMINURIA AND HYPERTENSION**

In our study the prevalence of microalbuminuria was 37%. The difference between the sexes in our study was not statistically significant. In the study by Biachi et al<sup>(73)</sup> about microalbuminuria among patients with HT the authors found that the incidence of microalbuminuria was 62% which is more than our study.

### **CARDIAC INVOLVEMENT IN HT**

In our study 46% of patients showed cardiac involvement. 19 showed LVH, 20 Gr1 DD and 7 showed Gr2 DD. The p value between the sexes was  $<0.05$  and significant again implying female tend to present more with complications compared to males. In the study by Chowta et al<sup>(62)</sup> the prevalence of cardiac involvement was 49.5% almost near to our study.

### **ABPI IN NEW HYPERTENSIVES**

28% of patients had a ABPI of  $<0.9$  signifying the presence of peripheral vascular disease among the newly diagnosed hypertensives. 31% of male and 28% of females had a ABPI of  $<0.9$ . However the p value was  $>0.05$  and not significant. The observed difference could be due to the prevalence of smoking among males. To the best of our



knowledge we couldn't find studies comparing ABPI in newly detected hypertensives.

### **LDL ABNORMALITIES IN NEW HYPERTENSIVES.**

In our study the dyslipidemia was present in 35% of patients. 35% among females and 35.7% among males. However p value was not significant. In the CURES<sup>(1)</sup> study by Mohan et al 38% had dyslipidemia which is almost similar to our study.

### **HYPERCHOLESTEROLEMIA IN NEW HYPERTENSIVES**

48% of our patients had hypercholesterolemia. 42.8% of females and 51.7% of males showed abnormal cholesterol values. However the p value was  $>0.05$  and not statistically significant. In the CURES<sup>(1)</sup> study by Mohan et al the prevalence of hypercholesterolemia was 29.3% .

### **METABOLIC SYNDROME IN NEW HYPERTENSIVES**

52% of our patients had metabolic syndrome as defined by the International Diabetic federation criteria. Of this 59.5% were females and 46.6% were males and the p value was  $<0.05$  statistically significant. Corriea et al<sup>(74)</sup> showed that the prevalence of metabolic syndrome among hypertensives was 48% which is near to our study.

### **BODY MASS INDEX IN NEW HYPERTENSIVES**

67% of patients in our study had a BMI of more than 25 which is defined as the general abdominal obesity. 71% of females and 63% of males were obese.

However p value was  $> 0.05$  and not significant. In the CURES<sup>(1)</sup> study by Mohan et al the prevalence of generalised obesity by following a similar criteria was 28.5%.

### **FRAMINGHAM RISK SCORE IN NEW HYPERTENSIVES**

56% of our patients had a cardio vascular risk score for hard incidents in the next 10 years of more than 10%. 54.7% of females and 56.8% of males had a risk of more than 10%.

36% of our new hypertensives had coronary heart disease equivalents. Out of this 36, 26 had diabetes, 6 had peripheral vascular disease and 4 had a risk score Of more than 20%. This again stresses the importance of early screening.

### **DIABETES IN NEW HYPERTENSIVES**

26% of our patients were found to have diabetes. 35.7% of females and 19% of males were found to be diabetic. The p value was significant  $<0.05$ . in the CURES<sup>(1)</sup> study by Mohan et al the prevalence of diabetes was 31.8% among the hypertensives which is almost near to our study.

### **ABDOMINAL OBESITY IN NEW HYPERTENSIVES**

60% of our patients had abdominal obesity. 73.8% of females and 50% of males had abdominal obesity as per the revised WHO Asian Pacific guidelines.

The p value was  $<0.05$  and significant. In the CURES <sup>(1)</sup> study by Mohan et al The prevalence of abdominal obesity was only 26.1%

### **ERECTILE DYSFUNCTION IN NEW HYPERTENSIVE MALES**

41.3% of males had mild to moderate erectile dysfunction in our study. Burchardt et al<sup>(71)</sup> in a study found that the prevalence of erectile dysfunction among hypertensives was 68.3%. However this difference might be because this study was done in patients who were under treatment for HT and probably the higher incidence might be due to the beta blockers or diuretics which those patients may be taking. To the best of our knowledge we couldn't get studies on ED in newly detected hypertensives in India.

### **METABOLIC SYNDROME WITH CVS RISK SCORING**

In our study 75% of patients with metabolic syndrome had a CVS hard risk score of  $>10\%$ . The p value was  $<0.01$  highly significant. This implies that metabolic syndrome is a strong predictor of future coronary events.

## LIMITATIONS OF THE STUDY

1. This study is done at a tertiary care centre and not at the primary level.
2. The population studied is very small compared with other studies.
3. Endocrine abnormalities which may cause secondary HT were ruled out only clinically and not biochemically.
4. There was no control population for the results to be compared.
5. Carotid and vertebral Doppler studies were not done.
6. Renal Doppler was not done to rule out reno vascular HT.

## CONCLUSION

1. **59%** of newly diagnosed hypertensives had some form of end organ damages.
2. **52%** of newly diagnosed hypertensives had metabolic syndrome and 75% among them had a CHD risk score of more than 10%.
3. **41.3%** of males had erectile dysfunction among them. So when an antihypertensive is chosen for a male patient this should be taken into consideration.
4. 67% of patients had a BMI >25 that is they had obesity.
5. **56%** of newly detected hypertensives have a CHD risk score of more than 10% . So aggressive risk reduction is necessary.
6. Females tend to present late with more complications compared to males.
7. As diabetes, hypertension is also a multisystem disease and each hypertensive patient has to be individualised and managed.
8. Finally it's high time that a routine screening programme is initiated at Community level to identify HT at early stages itself so that complications are averted.

## BIBLIOGRAPHY

7. V.Mohan, M. Deepa, S. Farooq, M. Datta, R. Deepa-- Prevalence, awareness and control of hypertension in Chennai – the Chennai urban rural epidemiological study (CURES) study- JAPI
8. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. *Lancet* 2005;365:217–23.
9. Stamler J. Blood pressure and high blood pressure: Aspects of risk. *Hypertension* 1991;18 (suppl.):I.95–I.107.
10. Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. Selected major risk factors and global and regional burden of disease. *Lancet* 2002;360:1347-60.
11. Kearney PM, Whelton M, Reynolds K, Whelton PK, He J. Worldwide prevalence of hypertension: a systematic review. *J Hypertens* 2004;22:11–19.
12. Riva-Rocci S: Un nuovo sfigmomanometro. *Gaz Med Torino* 1896; 47:981.
13. Korotkov N: A contribution to the problem of methods for the determination of the blood pressure. *Izv Imperatorskoi Voennomeditsinskoy Akad* 1905; 11:365
14. Chobanian AV, Bakris GL, Black HR, et al: Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* 2003; 42:1206-1252.

15. Evans RG, Majid DS, Eppel GA: Mechanisms mediating pressure natriuresis: What we know and what we need to find out. *Clin Exp Pharmacol Physiol* 2005; 32:400-409.
16. McDonough AA, Leong PK, Yang LE: Mechanisms of pressure natriuresis: How blood pressure regulates renal sodium transport. *Ann N Y Acad Sci* 2003; 986:669-677.
17. Granger JP, Alexander BT, Llinas M: Mechanisms of pressure natriuresis. *Curr Hypertens Rep* 2002; 4:152-159.
18. Laragh JH, Sealey JE: *Renin-angiotensin-aldosterone system and the renal regulation of sodium, potassium, and blood pressure homeostasis*. In: Windhager EE, ed. *Handbook of Physiology, Renal Physiology, Vol II*. New York: Oxford Press; 1992:1409-1541
19. Cowley Jr AW, Roman RJ: The role of the kidney in hypertension. *JAMA* 1996; 275:1581-1589
20. Guyton A, Hall J, Coleman T, et al: *The dominant role of the kidneys in long-term arterial pressure regulation in normal and hypertensive states*. In: Laragh JH, Brenner BM, ed. *Hypertension: Pathophysiology, Diagnosis, and Management, Vol 2*. New York: Raven Press; 1995:
21. Osborn J, DiBona G, Thames M: Beta-1 receptor mediation of renin secretion elicited by low-frequency renal nerve stimulation. *J Pharm Exp Ther* 1981; 216:265-269

22. DiBona GF: Peripheral and central interactions between the renin-angiotensin system and the renal sympathetic nerves in control of renal function. *Ann N Y Acad Sci* 2001; 940:395-406.
23. Schweda F, Kurtz A: Cellular mechanism of renin release. *Acta Physiol Scand* 2004; 181:383-390.
24. Kim SM, Mizel D, Huang YG, et al: Adenosine as a mediator of macula densa-dependent inhibition of renin secretion. *Am J Physiol Renal Physiol* 2006; 290:F1016-F1023
25. Sealey JE, Laragh JH: *The renin-angiotensin-aldosterone system for the normal regulation of blood pressure and sodium and potassium homeostasis*. In: Laragh JH, Brenner BM, ed. *Hypertension: Pathophysiology, Diagnosis, and Management*, Vol 2. New York: Raven Press; 1995:1763-1797.
26. Ghose RP, Hall PM, Bravo EL: Medical management of aldosterone-producing adenomas. *Ann Intern Med* 1999; 131:105-108
27. Morganti A, Lopez-Ovejero JA, Pickering TG, Laragh JH: Role of the sympathetic nervous system in mediating the renin response to head-up tilt. Their possible synergism in defending blood pressure against postural changes during sodium deprivation. *Am J Cardiol* 1979; 43:600-604.
28. Goodfriend TL, Elliott ME, Catt KJ: Angiotensin receptors and their antagonists. *N Engl J Med* 1996; 334:1649-1654



29. Carey RM, Wang ZQ, Siragy HM: Role of the angiotensin type 2 receptor in the regulation of blood pressure and renal function. *Hypertension* 2000; 35:155-163.
30. Carey RM: Update on the role of the AT2 receptor. *Curr Opin Nephrol Hypertens* 2005; 14:67-71
31. Meneton P, Jeunemaitre X, de Wardener HE, MacGregor GA: Links between dietary salt intake, renal salt handling, blood pressure, and cardiovascular diseases. *Physiol Rev* 2005; 85:679-715.
32. Laragh JH: The renin system and new understanding of the complications of hypertension and their treatment. *Arzneimittelforschung* 1993; 43:247-254.
33. Guyton A, Hall J, Coleman T, et al: *The dominant role of the kidneys in long-term arterial pressure regulation in normal and hypertensive states*. In: Laragh JH, Brenner BM, ed. *Hypertension: Pathophysiology, Diagnosis, and Management*, Vol 2. New York: Raven Press; 1995:1311-1326.
34. Roman RJ: P-450 metabolites of arachidonic acid in the control of cardiovascular function. *Physiol Rev* 2002; 82:131-185
35. Tigerstedt R, Bergman P: Niere und kresilauf. *Scand Arch Physiol* 1898; 8:223.
36. Goldblatt H, Lynch R, Hanzai R: Studies on experimental: Production of persistent elevation of systolic blood pressure by means of renal ischemia. *J Exp Med* 1934; 59:347.
37. William f. ganong review of medical physiology 2005

38. Guyton physiology of buffer nerves and their mechanism of action in normal individuals
39. Garcia-Estan J, Roman RJ: Role of renal interstitial hydrostatic pressure in the pressure diuresis response. *Am J Physiol* 1989; 256:F63-F70.
40. Cowley Jr AW, Guyton AC: Baroreceptor reflex effects on transient and steady-state hemodynamics of salt-loading hypertension in dogs. *Circ Res* 1975; 36:536-546
41. Guidi E, Menghetti D, Milani S, et al: Hypertension may be transplanted with the kidney in humans: A long-term historical prospective follow-up of recipients grafted with kidneys coming from donors with or without hypertension in their families. *J Am Soc Nephrol* 1996; 7:1131-1138.
42. Chabanel A, Chien S: *Blood viscosity as a factor in human hypertension*. In: Laragh JH, Brenner BM, ed. Hypertension: Pathophysiology, Diagnosis, and Management, Vol 1. New York: Raven Press; 1995:365-376.
43. Norrelund H, Christensen KL, Samani NJ, et al: Early narrowed afferent arteriole is a contributor to the development of hypertension. *Hypertension* 1994; 24:301-308
44. Mulvany MJ: Effects of angiotensin converting enzyme inhibition on vascular remodelling of resistance vessels in hypertensive patients. *J Hypertens Suppl* 1996; 14:S21-S24.

45. Schiffrin EL, Touyz RM: From bedside to bench to bedside: Role of renin-angiotensin-aldosterone system in remodeling of resistance arteries in hypertension. *Am J Physiol Heart Circ Physiol* 2004; 287:H435-H446.
46. Mulvaney M: *Structural changes in the resistance vessels in human hypertension*. In: Laragh JH, Brenner BM, ed. *Hypertension : Pathophysiology, Diagnosis, and Management*, Vol 1. New York: Raven Press; 1995:503-513.
47. Tracy RE: The heterogeneity of vascular findings in the kidneys of patients with benign essential hypertension. *Nephrol Dial Transplant* 1999; 14:1634-1639
48. Mitchell KD, Navar LG: Enhanced tubuloglomerular feedback during peritubular infusions of angiotensins I and II. *Am J Physiol* 1988; 255:F383-F390
49. Skott O, Briggs JP: Direct demonstration of macula densa-mediated renin secretion. *Science* 1987; 237:1618-1620
50. Mulvany MJ: Effects of angiotensin converting enzyme inhibition on vascular remodelling of resistance vessels in hypertensive patients. *J Hypertens Suppl* 1996; 14:S21-S24.
51. Luft F, Grim C, Willis L, et al: Natriuretic response to saline infusion in normotensive and hypertensive man: The role of renin suppression in exaggerated natriuresis. *Circulation* 1977; 55:779-784.
52. Schiffrin EL: Peroxisome proliferator-activated receptors and cardiovascular remodeling. *Am J Physiol Heart Circ Physiol* 2005; 288:H1037-H1043.

53. Olsen ME, Hall JE, Montani JP, et al: Mechanisms of angiotensin II natriuresis and antinatriuresis. *Am J Physiol* 1985; 249:F299-F307.
54. Olsen ME, Hall JE, Montaini JP, Guyton AC: Angiotensin II natriuresis and anti-natriuresis: Role of renal artery pressure in anaesthetized dogs. *J Hypertens Suppl* 1984; 2:S347-S350.
55. Pickering TG, Harshfield GA, Kleinert HD, et al: Blood pressure during normal daily activities, sleep, and exercise. Comparison of values in normal and hypertensive subjects. *JAMA* 1982; 247:992-996.
56. Rozanski A, Blumenthal JA, Kaplan J: Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation* 1999; 99:2192-2217
57. Lloyd-Jones DM, Leip EP, Larson MG, et al: Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. *Circulation* 2006; 113:791-798.
58. Kannel WB, Wilson PWF: *Hypertension, other risk factors, and the risk of cardiovascular disease*. In: Laragh JH, Brenner BM, ed. *Hypertension: Pathophysiology, Diagnosis and Management*, Vol 1. New York: Raven Press; 1995:99-114.
59. Lew E: *High blood pressure, other risk factors and longevity: The insurance viewpoint*. In: Laragh JH, ed. *Hypertension Manual*, New York: Yorke Medical Books; 1974.

60. Levy D, Larson MG, Vasan RS, et al: The progression from hypertension to congestive heart failure. *JAMA* 1996; 275:1557-1562.
61. Haider AW, Larson MG, Franklin SS, Levy D: Systolic blood pressure, diastolic blood pressure, and pulse pressure as predictors of risk for congestive heart failure in the Framingham Heart Study. *Ann Intern Med* 2003; 138:10-16.
62. Moser M, Hebert PR: Prevention of disease progression, left ventricular hypertrophy and congestive heart failure in hypertension treatment trials. *J Am Coll Cardiol* 1996; 27:1214-1218.
63. Jones CA, Francis ME, Eberhardt MS, et al: Microalbuminuria in the US population: Third national health and nutrition examination survey. *Am J Kidney Dis* 2002; 39:445-459
64. Romundstad S, Holmen J, Kvenild K, et al: Microalbuminuria and all-cause mortality in 2089 apparently healthy individuals: A 4.4-year follow-up study. The Nord-Trondelag Health Study (HUNT), Norway. *Am J Kidney Dis* 2003; 42:466-473.
65. Roest M, Banga JD, Janssen WM, et al: Excessive urinary albumin levels are associated with future cardiovascular mortality in postmenopausal women. *Circulation* 2001; 103:3057-3061.
66. Agrawal B, Berger A, Wolf K, Luft FC: Microalbuminuria screening by reagent strip predicts cardiovascular risk in hypertension. *J Hypertens* 1996; 14:223-228
67. Kohara K, Hiwada K. End organ damage in essential hypertension in the elderly. *J Human Hyperten*, 1995;9:717-21.

68. Chowta Kn, chowta MN, sandeep s- study of clinical profile of hypertension among the young and elderly-Indian journal of practising doctor vol5 no6
69. Staessen JA, Thijs L, Fagard R, O'Brien ET, Clement D, de Leeuw PW, et al. Predicting cardiovascular risk using conventional vs ambulatory blood pressure in older patients with systolic hypertension. Systolic Hypertension in Europe Trial Investigators. *JAMA* 1999;282:539-46.
70. Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, et al. Primary prevention of hypertension: Clinical and public health advisory from The National High Blood Pressure Education Program. *JAMA* 2002;288:1882-8
71. The IDF Epidemiology task force consensus group. The metabolic syndrome- a new world wide definition. *Lancet* 366:1059,2005
72. ECKEL RH et al The metabolic syndrome. *Lancet* 365:1415,2005.
73. Lyon CJ, Law RE, Hsueh WA. Minireview: adiposity, inflammation and atherogenesis. *Endocrinology* 2003;144:2195–2200.
74. World Health Organization, Western Pacific Region. The Asia Pacific Perspective. Redefining obesity and its treatment. World Health Organization. International Association for the Study of Obesity and International Obesity Task Force. Melbourne: International Diabetes Institute
75. **NIH Consensus Development Panel on Impotence.** Impotence. *JAMA* 1993; **270:**

76. **Rosen RC *et al.*** Development and evaluation of an abridged, 5-item version of the International Index of Erectile Function (IIEF-5) as a diagnostic tool for erectile dysfunction. *Int J Impot Res* 1999; **11**: 319-326
77. HYPERTENSION IS ASSOCIATED WITH SEVERE ERECTILE DYSFUNCTION MARTIN BURCHARDT *et al* international journal of impotence volume 4 issue 164 oct 2000
78. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 2001;285:2486-97.
79. Biachi.S. Bigazzi R, Baldari G. microalbuminuria in patients with essential HT. *Am J of medicine* 1992;93 525-529
80. Corriea. Basher N. gafar S. *Journal of human HT* 21 Jun 2007

# PROFORMA

Name: Age: Sex:

Occupation:

Place of living: village/town/city:

annual income:

## **SYMPTOMS:**

Epistaxis:	occipital headache:
Giddiness: yes/no	Chest pain: yes/no
Pedal edema: yes/no	TIA: yes/no
Episodic Headache/palpitations/perspiration	CVA: yes/no
Weight gain: yes/no	hyperpigmentation: yes/no
Visual disturbance:	claudication: yes/no

## **HISTORY:**

Sexual dysfunction:

Physical activity:

Drug abuse: cocaine/steroids/NSAID/ephedrine

Diabetes: yes/no Renal disease: yes/no

Thyroid disease: yes/no CAHD: yes/no

Smoker: yes/no Alcoholism: yes/no

TIA/CVA: yes/no

Family history: HT: DM:  
CKD: premature CVS:

## **O/E:**

Facies:	pallor:
Xanthoma:	Xanthelasma:
Purple striae:	Thyroid gland:
Waist circumference:	Hyperpigmentation:
Pedal edema:	Built:
JVP:	
PR:	
Carotid bruit:	
Radiofemoral delay:	



Peripheral pulses:

BP in Rt UL:

Stage:

Lt UL:

CVS:

Apical impulse position:

Heart sounds:

Added sounds:

Murmurs:

P/A

Organomegaly:

pulsations:

Renal artery bruit:

RS: rales/rhonchi

CNS:

FUNDUS:

**INVESTIGATIONS:**

Hb: Htc: Tc:

Dc: Platelets:

Urea:

creatinine:

Creatinine clearance:

Calcium:

Na+:

K+ :

Lipid profile:

Total cholesterol:

Triglycerides-

HDL:

LDL:

VLDL:

ECG:

ECHO:

BMI:

WAIST:

USG ABDOMEN:

URINE COMPLETE:/ MICROALBUMINURIA:

ABPI:

WAIST CIRCUMFERENCE:

BMI:

METABOLIC SYNDROME: YES/NO

VHD RISK SCORE:

CHD EQUIVALENT:

ERECTILE DYSFUNCTION:

**LIVER FUNCTION RESULT:**

