A COMPARATIVE STUDY ON ORTHOSTATIC HYPOTENSION IN ELDERLY HYPERTENSIVES AND NON HYPERTENSIVES

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CERTIFICATE

This is to certify that the dissertation titled **"A COMPARATIVE STUDY ON ORTHOSTATIC HYPOTENSION IN ELDERLY HYPERTENSIVES AND NON HYPERTENSIVES"** is a bonafide work carried out by **Dr. Gopinath K G** in the Department of Geriatric Medicine, Madras Medical College, Chennai, and is being submitted to The Tamil Nadu Dr. M.G.R Medical University in partial fulfillment of the requirement for the degree of M.D Branch XVI, Geriatric medicine.

Place:

Date:

Signature of Dean

Signature of HOD

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INTRODUCTION

Orthostatic hypotension is a common and important clinical disorder in the elderly population. The pathogenesis is multi factorial but is probably often due to changes in the autonomic nervous system, as well as to age-related changes in the cardiovascular and endocrine systems. Orthostatic hypotension can be seen as a prototypical clinical disorder of the elderly (1, 6). In addition, the presence of multiple diseases and medications, especially antihypertensives (2, 5) are common contributing factors. Standardized measurement of postural blood pressure and recording of heart rate and assessment of associated symptoms are essential for the clinical diagnosis. Usually elimination of offending medications and treatment of contributing medical disorders are adequate. It reflects impaired homeostasis; its etiology is multi factorial and due to the effects of both age and disease; it is clinically protean. It can result in falls, injury, and progressive decline if not recognized and treated early. Many studies have proved that Orthostatic hypotension is more prevalent among the elderly population, and more so among the elderly hypertensives. Symptoms, as mentioned above can lead to

drug non- compliance as well as a reduced quality of life. High blood pressure, once believed to represent a normal and progressive component of the aging process, is now recognized as a manifestation of structural and physiologic abnormalities of aortic function. Elevated systolic blood pressure and increased pulse pressure unquestionably increase the risk of both fatal and nonfatal cardiovascular events, including stroke, myocardial infarction, and heart failure. Isolated systolic hypertension, defined as a systolic blood pressure > 140 mm Hg with a diastolic blood pressure < 90 mm Hg, affects most individuals aged 60 years and older. Several clinical trials have clearly demonstrated that treatment of hypertension significantly reduces the cardiovascular event rate. However, controversy continues as to the choice of antihypertensive agents and combinations of agents. It is both appropriate and necessary to treat elderly hypertensives aggressively to the same target blood pressures identified for younger patients. It is also appropriate to initiate treatment with lower doses of antihypertensive agents and to bring the pressure down more slowly, monitoring for orthostatic hypotension, impaired cognition, and electrolyte abnormalities.

The prevalence of orthostatic hypotension varies substantially

depending on the groups being studied.

Epidemiological data suggest that the prevalence in community dwelling elderly populations can vary from 4% to 33%. In the Systolic Hypertension in the Elderly Project (SHEP), Orthostatic hypotension was noted in 10.4% at 1 minute after standing and 12.0% at 3 minutes, with 17.3% having hypotension at one or both intervals (6). The Cardiovascular Health study reported a prevalence of 16.2% in individuals aged over 65, which increased with age.

This study makes a comparison between elderly hypertensives and normotensives to find out the prevalence of orthostatic hypotension and other associated factors.

AIMS OF THE STUDY

To find out the prevalence of orthostatic hypotension in elderly hypertensives and non - hypertensives attending the Out – patient clinic.

OBJECTIVES OF THE STUDY

1. To assess the gender difference in the prevalence of orthostatic hypotension among hypertensives and non hypertensives

2. To assess the correlation between orthostatic hypotension and orthostatic symptoms among the elderly population.

3. To assess the relationship between anti hypertensives and Orthostatic hypotension.

4. To assess if control of hypertension has a bearing on the prevalence of orthostatic hypotension

REVIEW OF LITERATURE

Orthostatic hypotension is defined as a fall in systolic or diastolic blood pressure of more than 20 or 10 mm Hg respectively on assuming the upright posture.(Merck manual of Geriatrics). Orthostatic hypotension is not a specific disease but rather a manifestation of abnormal BP regulation due to various causes.

Etiology and Pathophysiology of orthostatic hypotension

The gravitational stress of sudden standing normally causes pooling of blood in the venous capacitance vessels of the legs and trunk. The subsequent transient decrease in venous return and cardiac output results in reduced BP. Baroreceptors in the aortic arch and carotid bodies activate autonomic reflexes that rapidly normalize BP by causing a transient tachycardia. These changes reflect primarily the sympathetic mediated increase in catecholamine levels, which augments vasomotor tone of the capacitance vessels, increases heart rate and myocardial contractility, and thereby enhances cardiac output; arterial and venous vasoconstriction are mediated by similar

mechanisms. Vagal inhibition also increases the heart rate. With continued standing, ADH secretion and activation of the renin-angiotensin- aldosterone system cause Na and water retention and expansion of the circulating blood volume.

When afferent, central, or efferent portions of the autonomic reflex arc are impaired by diseases or drugs, myocardial contractility or vascular responsiveness is depressed, hypovolemia is present, or hormonal responses are faulty, these homeostatic mechanisms may be inadequate to restore the lowered BP. The initial manifestations of decreased tissue perfusion are the effects of impaired cerebral blood flow; however, postural BP changes do not reliably reflect cerebral hypoperfusion.

Hypovolemia is the most common cause of symptomatic orthostatic hypotension. Hypovolemia is often induced by excessive use of diuretics (eg, loop diuretics such as furosemide, bumetanide, and ethacrynic acid); relative hypovolemia is due to vasodilator therapy with nitrate preparations and Ca blockers (verapamil, nifedipine, diltiazem, amlodipine) or with ACE inhibitors. The hypovolemia and

diminished vasomotor tone caused by protracted bed rest are also often a cause of orthostatic hypotension. Orthostatic hypotension is more frequent in diabetic than nondiabetic patients treated with antihypertensive drugs. Orthostatic hypotension is considered the most dramatic clinical manifestation and hallmark of diabetic autonomic neuropathy. While cardiac vagal neuropathy (assessed by higher resting heart rate and decreased respiratory arrhythmia) is the earliest and most common sign of autonomic neuropathy in diabetic patients, it seems that damaged sympathetic vasoconstriction (in the splanchnic bed, muscle and skin) is a prerequisite for postural hypotension and that diminished cardiac acceleration plays a lesser role in the development of such a complication.

A decline of 20 mmHg in systolic blood pressure on standing has been recommended to define a potentially dangerous hypotensive response as it has been shown to be a risk factor for fall and syncope, for symptomatic occlusive cerebrovascular disease and for a higher 5-year mortality rate. Orthostatic hypotension may indeed be worsened after insulin injection (because of the insulin-mediated muscular vasodilatation) and / or in the postprandial phase (because

of the digestion-related vasodilatation of the splanchnic bed). Ageing, certain medications (e.g. psychotropic drugs and antihypertensive agents) and some pathological changes, such as neurological or cerebrovascular diseases, are believed to increase the risk of postural hypotension in the general population, and such associations were also observed in the diabetic population. The appropriate selection and use of antihypertensive drugs in order to minimize the risk of orthostatic hypotension in non-diabetic and, even more important, in diabetic subjects with autonomic neuropathy should be considered as a major objective of the management of arterial hypertension in the future. Good glycemic control is important in the prevention of postural hypotension in subjects with diabetes, as recently reported in 19 poorly controlled Type 2 diabetic patients in whom this complication markedly regressed after insulin therapy and improvement of metabolic control. Orthostatic hypotension cannot be clinically determined just from the presence of postural dizziness and requires specific measurements (3, 7). In the presence of complaint of postural dizziness and in the absence of Orthostatic hypotension, measurements of arterial blood pressure should be repeated several times. Diabetes and (treated) arterial hypertension may all promote

Orthostatic hypotension.

Since life-expectancy of Type 2 diabetic patients is markedly increasing and since greater emphasis is being placed on optimal blood pressure control to prevent both micro- and macroangiopathic complications it is clear that Orthostatic hypotension will become a major problem in the management of increasing numbers of Type 2 diabetic patients (4).

Increased age is not only associated with increasing risk of hypotension, but also with the development of hypertension (HT). HT itself impairs Blood Pressure regulatory mechanisms, further increasing the risk of hypotension. Epidemiological studies of orthostatic hypotension in various populations over 65 years of age show a rather low prevalence of less than seven percent in healthy normotensive or treated hypertensive elderly people (5,6). However, the prevalence has been reported as being 17% in elderly people with HT. HT also increases the risk of cerebral ischemia from sudden declines in BP.

It has been suggested that elderly hypertensives with orthostatic

hypotension may have an elevated risk of developing hypertensive CVA.(5)

Also elderly hypertensives may be more vulnerable to cerebral ischemic symptoms such as syncope or dizziness from relatively small and short-term BP reductions.

Hypertension is defined as systolic BP >= 140 mm Hg or diastolic BP >= 90 mm Hg. Isolated systolic hypertension, a common form of hypertension in the elderly, is defined as systolic BP >= 140 mm Hg and diastolic BP < 90 mm Hg. For most elderly patients, hypertension does not have a reversible cause and is asymptomatic. Evaluation should include detection of other cardiovascular risk factors and end-organ damage and a search for secondary causes when appropriate. Treatment is with lifestyle modifications and drugs, often starting with a thiazide-type diuretic (5).

Physiologic Changes in Elderly

- Reduced cardiac output and myocardial reserve
- Left ventricular hypertrophy
- Reduced aortic elasticity and baroreceptor sensitivity

- Increased susceptibility to orthostatic hypotension
- Increased peripheral vascular resistance and atherosclerosis
- Reduced intravascular volume and plasma renin activity
- Reduced regional blood flow
- Reduced renal and hepatic reserve
- Increased salt sensitivity
- Increased plasma catecholamine levels with decreased beta-adrenergic responsiveness

Geriatric Essentials

- Most people > 65 yr have hypertension. Isolated systolic hypertension (ISH; systolic BP >= 140 mm Hg with diastolic BP < 90 mm Hg) accounts for > 2/3 of cases.
- ISH is caused primarily by an increase in arterial stiffness due to increased collagen deposition and cross-linking, degeneration of elastin fibers, atherosclerotic changes, and age-related endothelial dysfunction (6).
- All elderly people should be screened for hypertension at every health care visit and at least annually.
- BP readings may be falsely elevated in some elderly patients with very stiff, calcified arteries. This phenomenon is called

pseudo hypertension.

- Treatment of hypertension other than ISH reduces incidence of MI, stroke, and heart failure in the elderly.
- Treatment of ISH when systolic BP is >= 160 mm Hg reduces incidence of MI, stroke, and heart failure. Benefits with treatment of ISH when systolic BP is 140 to 160 mm Hg are presumed but unproven(6).
- Most elderly patients ultimately require >= 2 antihypertensive drugs to control BP. Thiazide-type diuretics are especially safe and effective in the elderly.
- In the elderly, all-cause and cardiovascular mortality rates increase linearly as systolic BP increases. All-cause mortality rates increase when diastolic BP is > 80 mm Hg and < 60 mm Hg (producing a J-shaped curve).

If hypertension has not developed by age 55, the probability of developing it is 90% during a person's lifetime. In developed countries, systolic BP tends to increase gradually with age, while diastolic BP tends to stabilize or decrease after age 55 to 60. The high prevalence of hypertension among elderly people in developed

countries might suggest that an age-related increase in arterial pressure is normal, but hypertension is practically nonexistent among elderly people in some developing countries.

The pathophysiology of hypertension in the elderly is complex and usually multifactorial. ISH is caused by an increase in arterial stiffness due to increased collagen deposition and cross-linking, degeneration of elastin fibers, atherosclerotic changes, and age-related endothelial dysfunction. Other possible contributors to hypertension in elderly patients may include genetic factors, classic cardiovascular disease risk factors (eg, tobacco use, diabetes mellitus, obesity, physical inactivity), an imbalance in sympathetic and parasympathetic tone with enhanced vascular reactivity to catecholamines (9), vascular remodeling, and renal microvascular changes.

In most elderly patients, intravascular volume contracts as arterial pressure and total peripheral resistance increase. Plasma renin activity and angiotensin II levels are normal or reduced, suggesting an attenuated relationship between intravascular volume and the renin-angiotensin system. This relationship may explain the

enhanced responsiveness to diuretics and Ca channel blockers in many elderly patients with hypertension, particularly those with systolic hypertension.

Correct measurement technique is especially important for elderly patients. Initially, BP should be measured in both arms (because occlusive atherosclerotic disease of the subclavian or brachial artery may reduce systolic BP in one arm) and with the patient seated (after a 5-min rest). BP should be re measured 1 and 3 min after the patient stands (to document orthostatic falls in BP, which are common among the elderly, particularly after meals).

Regular BP readings measured outside the clinical setting provide more information than a single office reading. In addition, 24-h ambulatory BP monitoring (ABPM) can determine circadian variations in BP and the percentage of all readings that are abnormal. ABPM may better predict end-organ damage than routine office measurements. ABPM is recommended for patients with sporadically elevated BP readings or white-coat hypertension without evidence of end-organ damage. ABPM is also indicated for patients with poor

responses to drugs, hypotensive symptoms during treatment, or autonomic dysfunction.

Pseudohypertension refers to falsely elevated systolic BP readings in elderly patients with very stiff arteries. Pseudohypertension occurs because the BP cuff cannot completely occlude the artery. Osler's sign, the ability to palpate the stiff, thickened radial artery when the sphygmomanometric cuff is inflated to suprasystolic BP, was once thought to suggest pseudohypertension, but more recent studies suggest that Osler's sign is an unreliable marker for this condition. Alternative ways to distinguish true systolic hypertension from pseudohypertension include arm x-rays to document extensive vascular calcification and Doppler flow studies, but neither is routine practice. More commonly, pseudohypertension is diagnosed when elderly patients do not respond to treatment, have markedly elevated systolic BP without signs of end-organ damage, or develop signs of hypotension (eg, fatigue, orthostasis) despite persistently elevated BP measurements.

A study done by Mattace Raso et al. showed that elderly subjects

with Pseudohypertension as measured by direct arterial pressure recordings and stiff arteries had a higher prevalence of orthostatic hypotension than normotensives without stiff arteries.

Acute or subacute severe hypovolemia caused by disease may produce orthostatic hypotension due to a decrease in cardiac output despite intact autonomic reflexes. Hemorrhage, severe vomiting or diarrhea, excessive sweating, or the osmotic diuresis in uncontrolled diabetes mellitus may lead to volume depletion, dehydration, and orthostatic hypotension unless fluid or electrolyte replacement is adequate. Hypokalemia impairs the reactivity of vascular smooth muscle and may limit the increase in peripheral vascular resistance on standing. The adrenocortical hypofunction of Addison's disease may lead to hypovolemic orthostatic hypotension in the absence of adequate salt intake.

Drugs that impair autonomic reflex mechanisms and reduce BP on standing, eg excessive doses of antihypertensive drugs (methyldopa, clonidine, reserpine, ganglionic blocking drugs) and multiple drug use, are also frequent causes. β-Adrenergic blockers are supposedly

a rare cause, but α-adrenergic blockers such as prazosin may be causative, especially at the initiation of therapy (first-dose effect).(8) First dose Orthostatic hypotension is commonly observed with antihypertensive drugs but its incidence may be overstated in clinical trials because of protocol requirements, which may include frequent venesection, fasting, acute withdrawal from caffeine, and frequent changes in posture.(8)

Drugs that provoke Orthostatic hypotension should be initiated in small doses with gradual upward titration. Other drugs that reversibly impair autonomic reflexes and reduce BP on standing (an important adverse effect) include many of those used to treat psychiatric disorders, such as monoamine oxidase inhibitors (isocarboxazid, phenelzine, tranylcypromine) used to treat depression; tricyclic antidepressants (nortriptyline, amitriptyline, desipramine, imipramine, protriptyline) or tetracyclic antidepressants; and phenothiazine antipsychotic drugs (chlorpromazine, promazine, thioridazine). Quinidine, levodopa, barbiturates, and alcohol may also produce orthostatic hypotension

Neurologic disorders that involve the autonomic nervous system interrupt the sympathetic reflex arc and impair normal adrenergic responses to standing. This is common with diabetic neuropathy, parkinsonism, amyloidosis, porphyria, tabes dorsalis, syringomyelia, spinal cord transection, pernicious anemia, alcoholic neuropathy, Guillain-Barré syndrome (postinfectious polyneuropathy), and Riley-Day syndrome (familial dysautonomia). Surgical sympathectomy, vasospastic disorders, or peripheral venous insufficiency (particularly severe varicose veins) may result in orthostatic hypotension.

The postural hypotension of Parkinson's disease may be aggravated by treatment with levodopa. Orthostatic hypotension may be a component of the vasomotor response in the postgastrectomy dumping syndrome.

Shy-Drager syndrome and idiopathic orthostatic hypotension are two possibly related primary neuropathic disorders commonly associated with severe orthostatic hypotension (10). In patients with Shy-Drager syndrome, plasma norepinephrine does not increase on standing; in those with idiopathic orthostatic hypotension, norepinephrine appears

to be depleted from the sympathetic nerve endings. In these conditions, widespread lesions affect the sympathetic and parasympathetic nervous systems, basal ganglia, and spinal tracts, with resultant widespread autonomic dysfunction in addition to failure of arteriolar and venous vasoconstriction; loss of sweating; bowel, bladder, and stomach atony; impotence; decreased salivation and tearing; mydriasis; and impaired visual accommodation. Paradoxically, BP may be elevated in the supine position, even when severe postural hypotension is present, because of loss of parasympathetic, as well as sympathetic, regulation of the cardiovascular system (14).

Orthostatic hypotension dominates the clinical picture of patients suffering from autonomic failure. Paradoxically, about one half of these patients also suffer from supine hypertension, which induces pressure natriuresis, worsening orthostatic hypotension. It also complicates the treatment of orthostatic hypotension. Supine hypertension is mediated by an increase in peripheral vascular resistance. This is due to residual sympathetic tone in patients with multiple system atrophy (Shy-Drager syndrome), but the cause is not known in patients with pure autonomic failure, who have increased

vascular resistance despite very low levels or plasma norepinephrine and renin activity. The recent observation that patients with supine hypertension develop left ventricular hypertrophy suggests they should be treated. During the day, avoiding the supine position is often all that is required. Short-acting vasodilators (e.g., transdermal nitroglycerin) can be used during the night.

Orthostatic hypotension is accentuated in the early morning due to overnight natriuresis and may also be more prominent post prandially and after exercise (16, 17).

In many causes of secondary systemic arterial hypertension, in which BP is not controlled by the usual homeostatic mechanisms, assuming the upright posture may cause orthostasis; this is prominent in most patients with pheochromocytoma and also occurs in patients with primary hyperaldosteronism, who, paradoxically, have hypertension in the supine position as well as orthostatic hypotension.

Cardiac causes of sudden-onset Orthostatic hypotension include unrecognized MI or cardiac arrhythmia. Other cardiac causes reflecting inability to increase cardiac output include severe dilated

cardiomyopathy, aortic stenosis, constrictive pericarditis, and advanced heart failure of any cause.

In the elderly**,** decreased baroreceptor responsiveness, coupled with decreased arterial compliance, accounts for frequent orthostatic hypotension. The decreased baroreceptor responsiveness delays the tachycardic response.

Although Orthostatic hypotension is described in about 20% of unselected elderly persons, its prevalence is much lower in healthy community- dwelling elderly (15). Coexistent multiple abnormalities often impair cardiovascular homeostasis in institutionalized elderly.

Faintness, light-headedness, dizziness, confusion, or visual blurring is evidence of a mild to moderate reduction in cerebral blood flow. With more severe cerebral hypoperfusion, syncope or generalized seizures may supervene (18, 19). Exercise or a heavy meal may exacerbate symptoms. Other associated phenomena usually relate to the underlying cause. Orthostatic hypotension is diagnosed when symptoms suggestive of hypotension and a marked reduction in measured BP are provoked by standing and relieved by lying down.

An underlying cause must be sought based on the patient's presenting circumstances and associated phenomena. Although measurements for orthostatic hypotension are not part of the standard physical examination, they should be taken if a patient's history suggests symptoms of cerebral hypoperfusion or a disease associated with orthostatic hypotension. Because orthostatic hypotension may be symptomatic or asymptomatic, symptoms and blood pressure measurements should be considered.

Diseases not related to orthostatic hypotension can cause similar symptoms (e.g., lightheadedness, dizziness). If a patient has posturally induced symptoms without blood pressure changes, the physician should consider other conditions. If a patient has posturally induced symptoms and a decrease in blood pressure but does not meet the strict definition for orthostatic hypotension, the physician still should consider orthostatic hypotension as a possible problem.

A review by Brian Olshansky states that Orthostatic Hypotension may be symptomatic or asymptomatic. Symptoms of OH are those that develop on assuming the erect posture or following head-up tilt and

usually resolve on resuming the recumbent position. They may include lightheadedness, dizziness, blurred vision, weakness, fatigue, cognitive impairment, nausea, palpitations, tremulousness, headache, and neck ache. If the patient has symptoms suggestive of, but does not have documented orthostatic hypotension, repeated measurements of blood pressure should be performed. Occasional patients may not manifest significant falls in blood pressure until they stand for at least 10 minutes.

Orthostatic hypotension is a risk factor for cerebrovascular accidents (19, 20). Orthostatic hypotension due to hypovolemia or drug excess is rapidly reversed by correcting these problems. Anemia and electrolyte imbalance can be specifically treated. The orthostasis of protracted bed rest can be lessened by having patients sit up each day. Elderly patients should maintain adequate fluid intake, limit or avoid alcohol, and exercise regularly when feasible (21).

The outlook in patients with a chronic underlying disorder is determined by the management of that disease; eg, Orthostatic hypotension appears to indicate a poor prognosis in diabetic patients

with hypertension.

Elderly patients should be encouraged to change posture slowly; sleeping with the head of the bed raised may relieve symptoms by promoting Na retention and reducing nocturnal diuresis. They should also avoid prolonged standing. Regular modest-intensity exercise promotes overall vascular tone and reduces venous pooling. (21)

A study done by Shin C et al. showed that the prevalence of Orthostatic hypotension at 0 and 2 min after standing was 12.3 and 2.9%, respectively. After adjustment for age and other characteristics, hypertension was associated with a 1.7-fold excess in the odds of Orthostatic Hypotension in men and a 1.6-fold excess in women ($P < 0.001$). In contrast, an increase in body mass index (BMI) on the order of 5 kg/m2 was associated with a 20-30% reduction in the odds of Orthostatic Hypotension ($P < 0.001$). Diabetes in women was also associated with a 1.4-fold excess in the odds of Orthostatic Hypotension ($P < 0.05$). (30)

A study done by Saez T et al. assessed the prevalence of orthostatic hypotension in an elderly population and evaluated the influence of

hypertension control and the type of antihypertensive drug used. The overall prevalence of Orthostatic Hypotension was 6.8% among hypertensive patients, 8.1% and 3.4% in normotensives patients ($p < 0.05$). No significant difference was found in the prevalence of Orthostatic Hypotension between those receiving (7.7%) or not (8.6%) antihypertensive medication. No association was found either between the prevalence of Orthostatic Hypotension and the type or number of antihypertensive drugs used. 17.6% of the patients reached hypertension optimal control (SBP < 140 and DBP < 90 mmHg), the prevalence of Orthostatic Hypotension in these patients was 5.3%, among the uncontrolled the prevalence was 8.9% ($p < 0.05$). (32)

A study done by Mattace - Raso et al. investigated whether arterial stiffening, one of the characteristics of the aging vascular system, is associated with orthostatic hypotension. The study included 3362 subjects. Orthostatic hypotension was assessed with blood pressure measurements in supine and standing position. In subjects with higher stiffness a higher drop in blood pressure was observed, but no significant change of heart rate. They concluded that arterial stiffness is independently associated with orthostatic hypotension. The drop in

blood pressure levels and the contemporary attenuated response of heart rate to orthostatic challenge in subjects with stiffer arteries support the hypothesis that arterial stiffness may explain, at least in part, the reduced baroreflex observed in older adults. (23)

A study done by Ejaz AA et al. investigated 100 patients with Orthostatic hypotension and 100 age matched controls. 42% of the cases were females. The most common symptoms complained were light headedness and weakness. During ambulatory blood pressure monitoring, postprandial decreases in blood pressure were noted in 83% of the OH group, supine or sleep hypertension in 84%, and non compensatory heart rate variability in 75%. Findings on autonomic testing were abnormal in 99% of patients, serum creatinine value was increased in 30%, proteinuria was present in 27%, and left ventricular hypertrophy was present in 20%. (29)

A study done by Lagi A et al. showed the incidence of the Hypo-Hyper pattern was 5.5% in the hypertensives studied. Hypo-Hyper was more frequent in the elderly (mean age 58 years), and the affected population exhibited different kinds of underlying pathologies. (31)

A study done by Vara-Gonzalez L et al. to determine the reproducibility of postural changes of blood pressure among hypertensive elderly patients in a primary care setting showed that the reproducibility of the postural changes of blood pressure found in hypertensive elderly patients in primary care was poor. Hence care needs to be taken while diagnosing orthostatic hypotension. (24)

A study done by Fotherby et al. was done to determine the prevalence of, and factors associated with, postural hypotension (PH) in elderly treated hypertensive subjects, to examine the 24h BP profile in those subjects with and without PH and to determine the effects of antihypertensive treatment withdrawal on the prevalence and symptoms of PH. Eighty-six subjects (mean age +/- standard deviation 76 +/- 6 years) on antihypertensive drug therapy for > 6 months had three clinic BP measurements taken in supine and standing positions followed by 24h ambulatory BP monitoring. Forty-seven subjects underwent repeat BP measurement five weeks after withdrawal of antihypertensive medication and institution of standard non pharmacological methods. Twenty-six (30%) of the 86 subjects exhibited orthostatic hypotension within three minutes of standing. In the orthostatic hypotension group, 19 subjects had treatment

withdrawn resulting in a reduction of 58% ($P < 0.001$) in those continuing to demonstrate PH. (25)

A study done by Vargas E et al assessed the relationship between orthostatic hypotension and supine hypertension. Individuals were designated as hypertensive (systolic > 160 and or diastolic > 90 mmHg) or normotensive on the basis of supine blood pressure levels. They concluded that supine hypertension is not related to the development or the degree of orthostatic hypotension in the elderly. (26)

A study by Atli et al. enrolled 61 healthy elderly subjects and 9 out of 61 (14.7%) had Orthostatic hypotension. Fasting plasma insulin level was significantly higher in non- Orthostatic Hypotension group than those in Orthostatic hypotension group ($p<0.05$), left ventricular mass index was significantly higher in subjects with Orthostatic hypotension ($p<0.05$). (28)

A study done by Goldstein et al. showed that 60% of patients with parkinson's disease had Orthostatic hypotension. Also, In Parkinson's Disease, OH can occur early in the disease, occasionally preceding or overshadowing the movement disorder. (27)

A study done by Kazuomi Kario et al. showed that among elderly hypertensive subjects, extreme dippers with marked nocturnal fall in blood pressure (BP) as well as non dippers with absent nocturnal fall in BP are more prone to cerebrovascular disease when compared with those with appropriate nocturnal BP fall. Abnormal diurnal BP variation is closely related to the abnormal postural BP variation in elderly hypertensive patients, with extreme dippers showing orthostatic hypertension and non dippers showing orthostatic hypotension. The upright position during the daytime, which increases the BP in the extreme dippers and decreases it in the non dippers, may in part produce abnormal diurnal BP variation. (33)

A study done by Shimada K et al (34) showed that elderly people, particularly hypertensives, are susceptible to orthostatic hypotension owing to diminished baroreceptor reflexes.

Another study done by Fletcher A et al (35) showed that orthostatic hypotension in hypertensive individuals may be exacerbated by antihypertensive agents, resulting in syncopal episodes and falls thus leading to increased morbidity, mortality and a reduced quality of life.

The NHANES 2 study showed that 6.6% patients showed a reduction in arterial pressure on standing up. (36)

The SHEP study, looking at a population more than 60 years, showed that 10% of hypertensive patients had orthostatic hypotension. (6)

A study done by Viramo et al showed that Orthostatic hypotension affects every fourth person aged 70 years or older (25%). In an unselected population, OH was not associated with cognitive deterioration, nor did it predict cognitive decline during a 2-year follow-up. (38)

A study done by Robertson et al among healthy elderly patients attending a out patient clinic showed that Orthostatic Hypotension was unassociated with age, race, sex, body mass, time since eating, symptoms, or other factors. Orthostatic blood pressure changes were at normally distributed levels with systolic and diastolic pressures dropping an average of 4 mm Hg (standard deviation [SD] =15 mm Hg) and 2 mm Hg (SD =11 mm Hg), respectively. (39)

In a study of 843 independent-living men and women aged between

60 and 87 by Burke et al, postural fall in blood pressure was not significantly related to treatment for hypertension, age, sex, patterns of usual physical activity, tea or coffee drinking, or the diagnosis of diabetes mellitus.

MATERIALS AND METHODS

A convenience sample of 100 subjects aged 60 and above with hypertension under treatment and 100 non - Hypertensives of the same age were selected for the study in the out – patient unit of the department of Geriatric medicine, Madras medical college, Chennai.

Persons included in the study were informed about the aims of the study and consent was obtained.

Inclusion criteria

1. Age > 60 years
2. Hypertensives

Exclusion criteria

1. Age < 60 years
2. Newly detected hypertensives

Demographic data including age, duration of hypertension, details of

drug therapy including drugs used and the dosage were recorded. Patients were enquired about the well known orthostatic symptoms like postural giddiness, unexplained falls or any other features like light headedness, symptoms of cerebral ischemia like syncope, Transient Ischemic Attacks, other symptoms like upper back pain(coat – hanger pain) , shoulder pain. Hypertensive patients on life style modifications as their only therapy were excluded.

Patients in both the groups were enquired about presence or absence of Diabetes mellitus along with other diseases.

A sitting blood pressure was measured. Patients were then made to lie down for ten minutes following which the blood pressure was measured again. After this, patients were made to stand and the blood pressure was checked at 1 and 3 minutes. The apparatus used was a manually operated sphygmomanometer. Patients were enquired if they experienced orthostatic symptoms during the examination. A single apparatus was used and the measurements were made by a single investigator.

Orthostatic hypotension was defined as a drop in either the systolic or

diastolic blood pressure of more than 20 or 10 mm Hg respectively upon standing from a supine position.

There were no drop– outs from the study.

Observations

Among the 100 hypertensives studied, 47 were females and 53 males. Among the non – hypertensive group, 48 were females while 52 were males.

Table 1: *Distribution of cases according to sex*

Group	Male	Female	Total
Hypertensives	53	47	100
Non – hypertensives	52	48	100
	105 (52.5%)	95(47.5%)	200

Figure 1: Sex distribution of Hypertensives

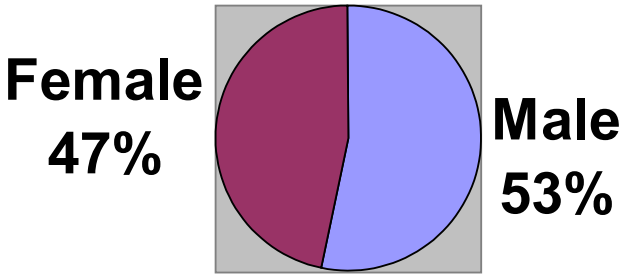
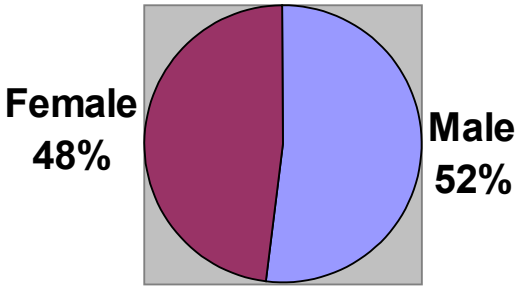


Figure 2: Sex distribution of non hypertesives



Among the hypertensives, 55 were aged 60 – 69 years, 29 were aged 70 –79, while 16 people were above 80 years of age. There were 49 non hypertensives aged 60- 69, 31 in the 70 – 79 age group and 20

were aged above 80 years.

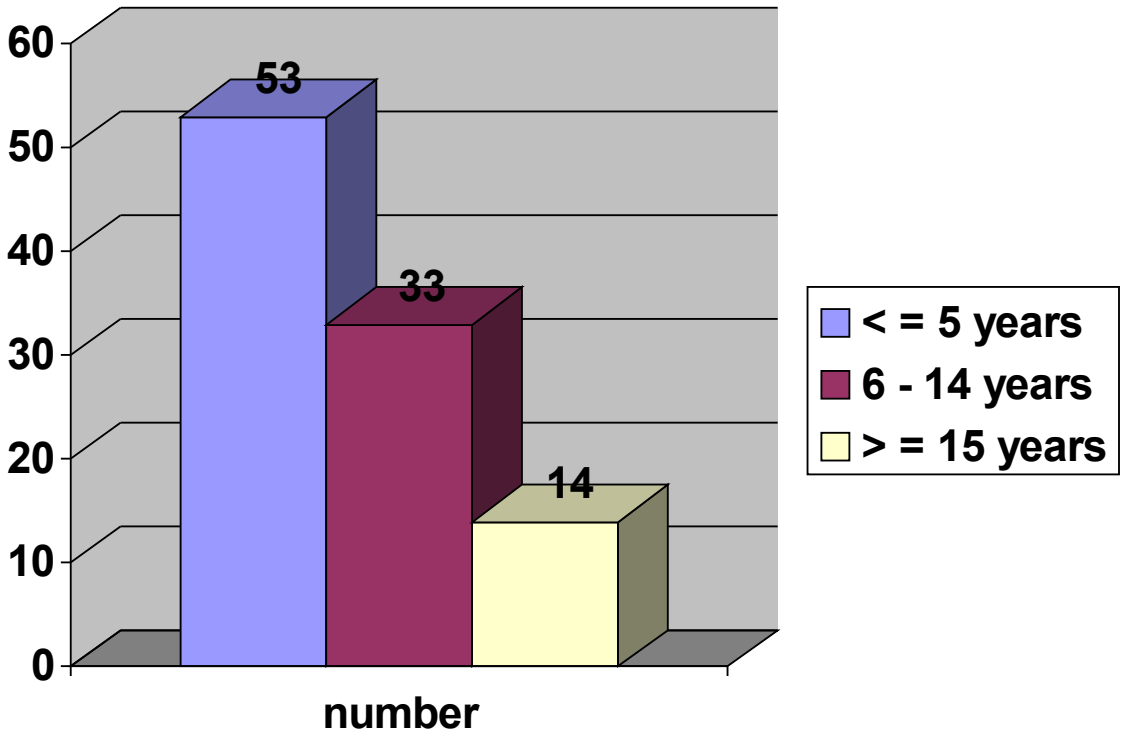
Table 2 : *Frequency distribution of patients according to age*

Age group	Hypertensives	Non hypertensives
60 – 69	55	49
70 – 79	29	31
> 80 years	16	20

The mean age of hypertensives was 70.16 +/ - 8.22 years. The mean age of the non Hypertensives was 72.01 +/ - 9.25 years.

Most of the hypertensives (53) examined were hypertensives for less than or equal to 5 years. 33 people were hypertensives for more than 5 years but less than 15 years while 14 were hypertensive for more than or equal to 15 years.

Figure 3 : Frequency distribution of hypertensives according to duration



While sitting up, in the hypertensive group, mean systolic and diastolic pressures were 139.52 +/- 20.36 mm Hg. In the non hypertensives, it was 117.56 +/ - 11.93 mm Hg.

The mean systolic and diastolic blood pressures in the hypertensive group lying down were 135.42 + / - 19.83 and 83.70 + / - 11.52 mm Hg.

The mean systolic and diastolic blood pressures in the non hypertensive group lying down were 121.36 + / - 13.28 and 76.32 + / - 8.24 mm Hg.

Among the hypertensive subjects, 39 people complained of orthostatic symptoms alone or in combination with a fall. Only one subject of the 39 complained of a history of fall without any symptoms. Orthostatic giddiness was complained by 34 subjects while four complained of giddiness and fall. In the above group, eight had orthostatic hypotension.

61 subjects did not have any symptoms related to orthostatic hypotension. Among this group, 10 had orthostatic hypotension.

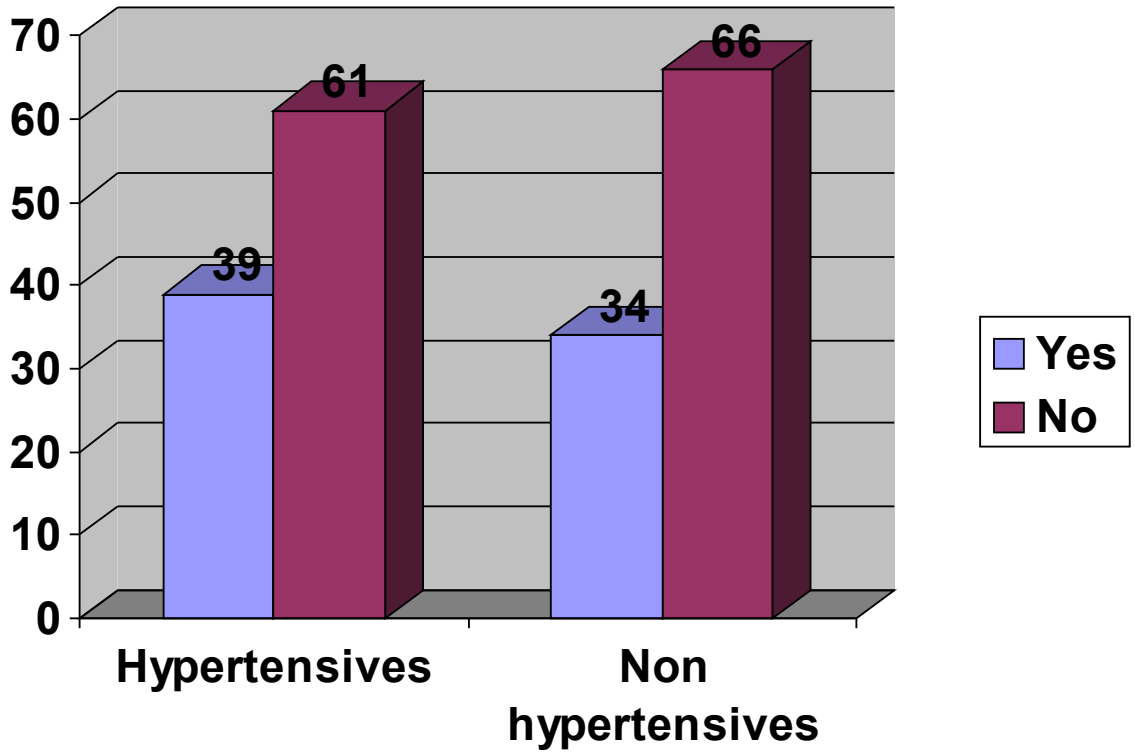
66 people among the non – hypertensives did not complain of orthostatic symptoms and four of them had orthostatic hypotension. 34 subjects did complain of either orthostatic giddiness alone or associated with a fall. Three of them had orthostatic hypotension.

Table 3 : *Symptom – wise distribution of groups*

Orthostatic symptoms	Hypertensives	Non - hypertensives
Yes	39 (8)	34 (3)
No	61 (10)	66 (4)
	P Value > .05	P Value > .05

Note: *Numbers within parentheses denote cases with orthostatic hypotension*

Figure 4 : symptom wise distribution



There were 18 hypertensives and 7 normotensives with orthostatic hypotension among both the groups put together. Hence the prevalence of orthostatic hypotension is 12.5%. The prevalence in the hypertensive and non – hypertensive groups were 18% and 7% respectively.

Table 4: *Distribution of orthostatic hypotension group - wise*

Orthostatic Hypotension	Hypertensives	Non hypertensives	P Value
Present	18	7	P < .05
Absent	82	93	

The mean systolic drop in those with a systolic orthostatic hypotension (Hypertensives) was 24.88 mm Hg. The mean diastolic drop was 16 mm Hg. Similarly it was 17 mm Hg and 13mm Hg in the non hypertensive group.

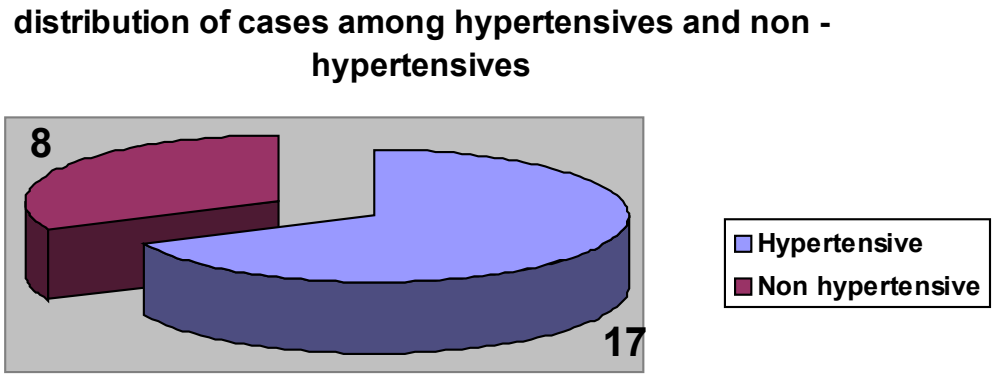


Table 5*: Distribution of orthostatic hypotension group - wise according to time of drop*

	Hypertensives	Non - hypertensives
Drop at 1 min	5(27.8%)	1(14.3%)
Drop at 3 min	13(72.2%)	6(85.7%)

P value > .05

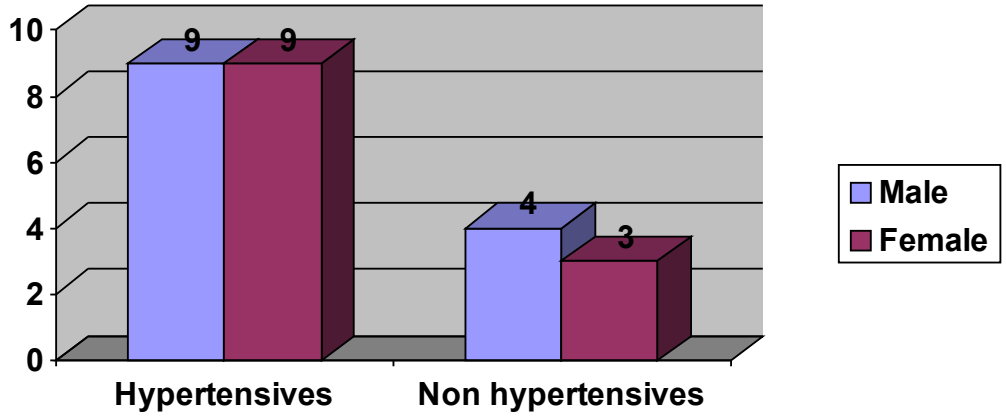
76% of patients showed orthostatic hypotension at 3 minutes after standing while 24% dropped at 1 minute when both the groups were taken together.

Among hypertensives, 9 females and 9 males had orthostatic hypotension. Among the non hypertensives, 3 females and 4 males had orthostatic hypotension.

Table 6 : *Sex – wise distribution of orthostatic hypotension*

	Hypertensives	Non hypertensives
Male	9	4
Female	9	3
	P value > .05	P value > .05

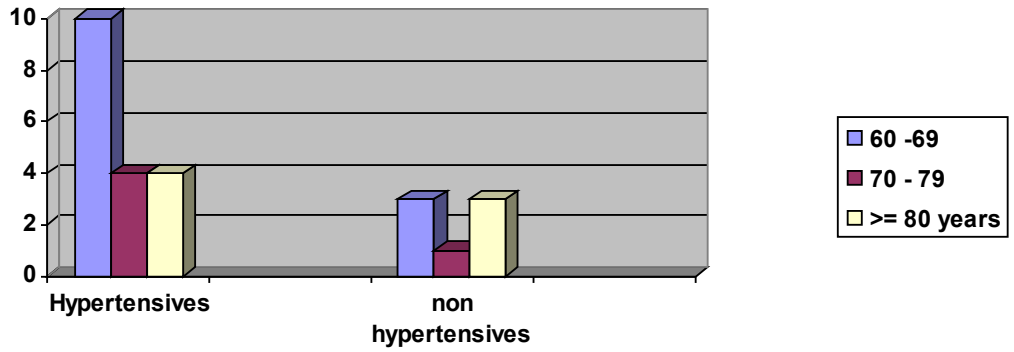
Figure 5: Sex wise distribution of orthostatic hypotension



10 out of the 55 hypertensives (18.2%) aged less than 69 years had orthostatic hypotension. Four out of 29 subjects (13.8%) aged between 70 and 79 had orthostatic hypotension while four were found among 16 (25%) above 80 years of age. In the non hypertensives, three out of 49(6.1%) aged less than 69, one out of 31(3.2%) and three out of 20 (15%) had orthostatic hypotension.

Table 7: *Frequency distribution of orthostatic hypotension according to age*

Age	hypertensives	non hypertensives
60 – 69	10 (18.2%)	3 (6.1%)
70 – 79	4 (13.8%)	1 (3.2%)
> 80	4 (25%)	3 (15%)
	P value > .05	P value > .05



Seven hypertensives out of 53 hypertensive (13.5%) for less than or equal to 5 years had orthostatic hypotension. Ten cases were found among 33 hypertensives (29.4%) with disease duration between 6 – 14 years while there was only 1 case among 14 hypertensives (7.1%) with disease duration more than 15 years.

Table 8: *Distribution of cases according to duration of disease*

Duration of hypertension	No of cases
< = 5 years	7 (13.5%)
6 – 14 years	10 (29.4%)
> = 15 years	1 (7.1%)

10 out of 59 patients on a single drug, 6 out of 31 on two drugs and 2 out of 10 on all three drugs had orthostatic hypotension.

Table 9: *Distribution of orthostatic hypotension according to no of drugs used*

Number of drugs	Orthostatic Hypotension	No Orthostatic Hypotension
1	10	49
2	6	25
3	2	8

There was no significance between the number of drugs used and the prevalence of orthostatic hypotension. (P > .05)

The single most commonly used drug was a calcium channel blocker. 7 out of 30 patients (23.3%) on Calcium channel blockers alone had orthostatic hypotension whereas only 1 out of 13 (7.6%) on beta blocker alone and 1 patient out of 13 (7.6%) on angiotensin converting enzyme inhibitor alone had orthostatic hypotension.

Table 10: *Distribution of cases according to class of drug when used singly*

Class of drug	cases	
Calcium channel blocker	7 (23.3%)	
Beta – blocker	1 (7.6%)	
Angiotensin converting enzyme inhibitor	1 (7.6%)	P Value P > .05

Figure 7: Frequency of orthostatic hypotension in hypertensives using single drugs

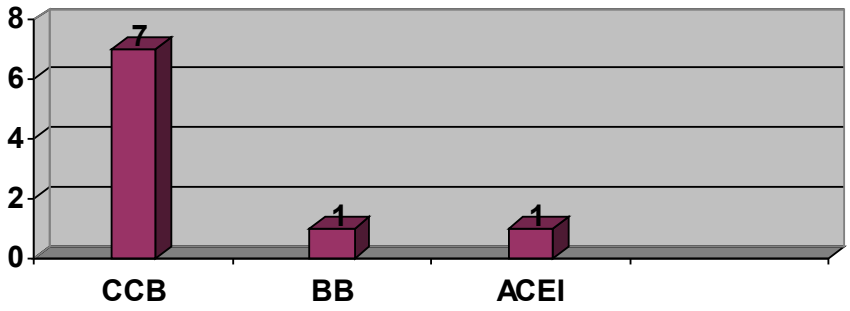


Table 13: *Diabetes Mellitus and Orthostatic hypotension*

Diabetes Mellitus	Hypertensives	Non - hypertensives
Yes	23 (6)	26 (3)
No	77 (12)	74 (4)

Note : Numbers within Parenthesis denote Orthostatic hypotension

6 out of 23 (26.1%) subjects with Type 2 Diabetes mellitus in the hypertensive group had orthostatic hypotension while 3 out of 26 (11%) in the non hypertensive group had orthostatic hypotension. There was no impact of diabetes on the prevalence of orthostatic hypotension (P > .05) between Hypertensives and non hypertensives.

Table 12: *Distribution of orthostatic hypotension according to BP control*

Orthostatic hypotension	Well controlled	Not controlled
Yes	12(18.75%)	6(16.66%)
No	52	30

P value > .05

64 patients were well controlled and 12 (18.75%) out of this had Orthostatic hypotension. Similarly, 36 out of 100 were uncontrolled and 6 (16.66%) had orthostatic hypotension.

DISCUSSION

The population of aged in India is about 7%. This number is bound to increase with better medical facilities offered to the people. Hypertension is more prevalent in the elderly. Age related changes in the neurological and vasomotor systems make the elderly population more prone to develop Orthostatic hypotension. Hypertension, by itself impairs the regulatory mechanisms and antihypertensives used to treat hypertension are important causes of Orthostatic hypotension.

Orthostatic hypotension is an easily identifiable disorder that does not require sophisticated equipment for identification. It is important to identify Orthostatic hypotension as it is associated with many consequences. Orthostatic hypotension can lead to cerebral hypoperfusive states like Transient ischemic attacks, Cerebrovascular accidents, seizures and syncope. It can lead to decreased quality of life by causing falls, fractures, increased hospitalizations and death.

This was a descriptive study of a comparative nature. A convenience sample of 100 was selected for each group. Consecutive cases were selected in both the groups. This study was done to assess the prevalence of orthostatic hypotension among the elderly population attending the out patient unit of a tertiary care hospital in South India.

The overall prevalence of orthostatic hypotension was 12.5%. Among the hypertensive and non – hypertensive groups, the prevalence was 18 % and 7 % respectively. There was a statistically significant difference ($p < 0.05$) in the prevalence of orthostatic hypotension among the hypertensives and non hypertensives.

Shin C et al. showed that the prevalence of Orthostatic hypotension at 0 and 3 min after standing was 12.3 and 2.9%, respectively. After adjustment for age and other characteristics, hypertension was associated with a 1.7-fold excess in the odds of Orthostatic Hypotension in men and a 1.6-fold excess in women ($P < 0.001$).

A study done by Saez et al. showed the overall prevalence of OH was 6.8% among elderly, 8.1% and 3.4% in hypertensives and normotensives patients respectively ($p < 0.05$).

Shimada et al. have proved that elderly hypertensives have a higher chance of developing orthostatic hypotension owing to changes accompanying the disease process itself, drugs used to treat hypertension and age – related changes in related systems. Hence elderly, especially hypertensives need to be followed up regularly.

A study done by Viramo et al showed the prevalence of orthostatic hypotension was 25%.

A study by Lorenzini on 1010 patients showed that orthostatic hypotension was very common(35%) among community dwelling elderly, and the prevalence was higher among elderly aged > 80 years and hypertensive subjects.

There was no statistically significant difference (P value > .05) in the prevalence of orthostatic hypotension among males and females in both the groups of patients.

A study done by Robertson et al showed that Orthostatic Hypotension was unassociated with age, race, sex, body mass, time since eating, symptoms, or other factors.

A study done by Raiha et al showed abnormal postural systolic blood pressure drop (-20 mm Hg or less) after standing for 3 minutes in 28.0% of subjects. There were no sex or age differences between the subjects with postural hypotension. No predisposing factors for postural hypotension other than elevated blood pressure were found. Chronic cardiovascular diseases, disability, body mass index, medication. Diastolic blood pressure drop, in particular after standing for 1 minute, was associated with increased vascular mortality. In the multivariate analysis, however, this association disappeared.

Among both hypertensives and non hypertensives, a higher proportion of patients aged 80 and above had orthostatic hypotension. However, this was statistically insignificant (P value > . 05). The number of patients in this age group was probably insufficient.

There was no relation of patient's complaints to the prevalence of orthostatic hypotension. Many patients who did complain of symptoms actually failed to show a postural drop while many asymptomatic patients had a significant drop. Studies done

previously have shown that few symptomatic patients without a significant drop eventually showed orthostatic hypotension. High frequency of symptoms without a significant drop could also be explained by the increased likelihood of non – specific giddiness, neck pain, shoulder pain among the elderly population. It becomes imperative that all patients be checked for orthostatic hypotension, regardless of symptoms. Moreover, symptomatic patients without orthostatic hypotension should be checked repeatedly.

A review by Brian Olshansky states that Orthostatic Hypotension may be symptomatic or asymptomatic. They may include lightheadedness, dizziness, blurred vision, weakness, fatigue, cognitive impairment, nausea, palpitations, tremulousness, headache, and neck ache. If the patient has symptoms suggestive of, but does not have documented orthostatic hypotension, repeated measurements of blood pressure should be performed. Occasional patients may not manifest significant falls in blood pressure until they stand for at least 10 minutes.

Diabetes Mellitus and Hypertension are common in the elderly.

Hypertensive patients with Diabetes have been shown to have a higher prevalence of orthostatic hypotension. Diabetics alone also have a higher chance of orthostatic hypotension. In this study, Diabetes was apparently not associated with an increased prevalence in both the groups.

The most commonly used drug to treat hypertension was a Calcium channel blocker. 6 out of 23 (26%) cases using a calcium channel blocker only, 1 out of 13 (7.6%) using a beta blocker and 1 out of 13 (7.6%) using a Angiotensin converting enzyme inhibitor had orthostatic hypotension. There was no statistically significant difference ($P > .05$) in the prevalence of orthostatic hypotension among the various single drug users.

A study done by Saez T et al showed no significant difference was found in the prevalence of OH between those receiving (7.7%) or not (8.6%) antihypertensive medication. No association was found either between the prevalence of OH and the type or number of antihypertensive drugs used.

Control of blood pressure was not related to the development of

orthostatic hypotension (P Value > .05). 64 % of hypertensives reached optimum Blood pressure control while 36% were uncontrolled.

The study mentioned above showed that only 17.6% of the patients reached hypertension optimal control (SBP < 140 and DBP < 90 mmHg). The prevalence of OH in the optimally controlled patients was 5.3%, among the uncontrolled the prevalence was 8.9% ($p < 0.05$).

Another study by Burke et al showed Orthostatic hypotension was not significantly related to treatment for hypertension, age, sex, or the diagnosis of diabetes mellitus.

Conclusion

1) Orthostatic hypotension is a common clinical disorder in the elderly. There is no sex difference in the prevalence of orthostatic hypotension.
2) The prevalence of Orthostatic hypotension is more among hypertensives than non – hypertensives.
3) There is no significant relation between orthostatic hypotension and the symptoms of orthostatic hypotension.
4) Orthostatic hypotension is not related to either the type or number of drugs used in this study.
5) Development of Orthostatic hypotension has no relation to control of hypertension.

Annexure I - BIBLIOGRAPHY

1. Robbins AS, Rubenstein LZ: Postural hypotension in the elderly. J Am Geriatr Soc. 1984 Oct; 32(10): 769 - 74

2. H Krum, E L Conway: Postural hypotension in elderly patients given carvedilol . BMJ 1994;309:775-776

3. Wu J-S, Lu F-H, Yang Y-C, Chang C-J. Postural hypotension and postural dizziness in patients with non-insulin-dependent diabetes Arch Intern Med 1999; 159(6): 1350–6

4. UK Prospective Diabetes Study Group. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. Br Med J 1998; 317: 703–13

5. Applegate WB. *Hypertension* in elderly patients. *Ann Intern Med* 1989;110:901-915.

6. The Systolic *Hypertension* in the Elderly Program (SHEP) Cooperative Research Group. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension: final results of SHEP. *JAMA* 1991;265:

3255-3264.

7. Dahlsf B, Lindholm LH, Hanssonh, et al. Morbidity and Mortality in the Swedish Trial in Old Patients with *Hypertension* (STOP-Hypertension). *Lancet* 1991;338:1281-1285.

8. Applegate WB, Phillips HL, Schnaper H, et al. A randomized controlled trial of the effects of three antihypertensive agents on blood pressure control and quality of life in older females. *Arch Intern Med* 1991;151:1817-1823.

9. Bertel O, Buhler FR, Kiowski W, Lutold BE. Decreased beta-adrenoreoeptor responsiveness as related to age, blood pressure, and plasma catecholamine in patients with essential hypertension. *Hypertension* 1980;2:130-138.

10. Morton A. Diamond, Raymond H. Murray. Idiopathic postural hypotension: physiologic observations and report of a new mode of therapy: J Clin Invest. 1970 July; 49(7): 1341–1348

11. Purewal TS, Watkins PJ. Postural hypotension in diabetic autonomic neuropathy: a review. Diabetic Med 1995; 12: 192–200.

12. Lipsitz LA. Orthostatic hypotension in the elderly. N Engl J Med 1989;321:952–7.

13. Doelman CJA, Oude Elberink JNG, Miedema K, Bilo HJG. Orthostatic hypotension in poorly regulated NIDDM [letter]. Diabetes Care1996;19:542.

14. Ooi WL, Barrett S, Hossain M, Kelley-Gagnon M, Lipsitz LA. Patterns of orthostatic blood pressure change and their clinical correlates in a frail, elderly population. JAMA 1997;277:1299- 304.

15. Mader SL, Josephson KR, Rubenstein LZ. Low prevalence of postural hypotension among community-dwelling elderly. JAMA 1987;258:1511-4.

16. Vloet et al. High Prevalence of Postprandial and Orthostatic Hypotension Among Geriatric Patients Admitted to Dutch Hospitals J Gerontol A Biol Sci Med Sci 2005;60:1271-1277.

17. Duncan, James R. Postprandial hypotension in response to duodenal glucose delivery in healthy older subjects; J Physiol 2002 Apr 15;540(Pt 2):673-9

18. Rutan GH, Hermanson B, Bild DE, Kittner SJ, LaBaw F, Tell GS. Orthostatic hypotension in older adults. The Cardiovascular Health Study. CHS Collaborative Research Group. Hypertension 1992;19: 508-19.

19. Eigenbrodt ML, Rose KM, Couper DJ, Arnett DK, Smith R, Jones D. Orthostatic hypotension as a risk factor for stroke: the atherosclerosis risk in communities (ARIC) study, 1987-1996. Stroke 2000;31:2307-13.

20. Weiling W, VanLieshout JJ. Maintenance of postural normotension in humans. Clinical autonomic disorders: evaluation and management. Boston: Little, Brown, 1993:69-77.

21. Robertson D, Davis TL. Recent advances in the treatment of orthostatic hypotension. Neurology 1995;45(4 suppl 5):S26-3

22. Burke V, Beilin LJ. Postural fall in blood pressure in the elderly in relation to drug treatment and other lifestyle factors. Q J Med. 1992 Aug; 84(304):583-91.

23. Mattace-Raso FU, van der Cammen TJ, Knetsch AM, van den Meiracker AH, Schalekamp MA, Hofman A, Witteman JC. Arterial

stiffness as the candidate underlying mechanism for postural blood pressure changes and orthostatic hypotension in older adults: the Rotterdam Study; J Hypertens. 2006 Feb;24(2):339-4

24. Vara-Gonzalez L, Arauzo Alonso S, Gonzalez Fernandez RM, Marin-Gil Vecilla M, Virseda Marin N, Munoz Cacho P; Reproducibility of postural changes of blood pressure in hypertensive elderly patients in primary care. Blood Press Monit. 2006 Feb; 11(1):17-20.

25. Fotherby MD, Robinson TG, Potter JF. Clinic and 24h blood pressure in elderly treated hypertensives with postural hypotension. J Hum Hypertens. 1994 Sep;8(9):711-6

26. Vargas E, Lye M. Clin Auton Res. Is there a relationship between supine systemic blood pressure and orthostatic hypotension in the elderly. 1993 Oct; 3(5):345-9.

27. Goldstein DS Orthostatic hypotension as an early finding in Parkinson's disease Clin Auton Res. 2006 Feb;16(1):46-54

28. Atli T, Keven K. Orthostatic hypotension in the healthy elderly Arch Gerontol Geriatr. 2006 Feb 6(1)

29. Ejaz AA, Haley WE, Wasiluk A, Meschia JF, Fitzpatrick PM Characteristics of 100 consecutive patients presenting with orthostatic hypotension. Mayo Clin Proc. 2004 Jul; 79(7):890-4.

30. Shin C, Abbott RD, Lee H, Kim J, Kimm K. Prevalence and correlates of orthostatic hypotension in middle-aged men and women in Korea: the Korean Health and Genome Study. J Hum Hypertens. 2004 Oct 18(10):717-23.

31. Lagi A, Rossi A, Comelli A, Rosati E, Cencetti S. Postural hypotension in hypertensive patients. Blood Press. 2003;12(5-6):340-4.

32. Saez T, Suarez C, Sierra MJ, Llamas C, Jimenez R, Vega S, Alonso M, Fernandez G, Gabriel R. Orthostatic hypotension in the aged and its association with antihypertensive treatment**.** Med Clin (Barc). 2000 Apr 15; 114(14):525-9.

*33.*Kazuomi Kario et al. Relationship Between Extreme Dippers and Orthostatic Hypertension in Elderly Hypertensive Patients Hypertension. 1998;31:77

34. Shimada K, Iritasumi T, Ogura H, Sadatrane N, Ozawa T.

Differences in age-dependent effects of blood pressure on baroreflex sensitivity between normal and hypertensive subjects. Clin Sci 1986;70:489-94

35. Fletcher A, Amery A, Birkenhager W, et al. Risks and benefits in the trial of the European working party on high blood pressure in the elderly. J Hypertension 1991;9:225-30.

36. Mc dowell et al. plan and operation of the second national health and nutrition examination survey Vital health stat 1.1981; 1(15); 1-144.

37. Jens Jordan, MD ; Italo Biaggioni, MD. Diagnosis and Treatment of Supine Hypertension in Autonomic Failure Patients With Orthostatic Hypotension. *The Journal of Clinical Hypertension*, February, 2002 4;2:139-145.

38. Viramo P, Luukinen H, Koski K, Laippala P, Sulkava R, Kivela SL Orthostatic hypotension and cognitive decline in older people. J Am Geriatr Soc. 1999 May;47(5):600-4. .

39. Robertson D, Des Jardin JA, Lichtenstein MJ Distribution and observed associations of orthostatic blood pressure changes in

elderly general medicine outpatients. Am J Med Sci. 1998 May; 315(5):287-95.

Annexure II - PROFORMA FOR ORTHOSTATIC HYPOTENSION

History

1. Name:

2. Age ------- 60 – 69 / 70 – 79 / > = 80 years

3. Sex ------- Male / Female

4. Duration of hypertension:

   < = 5 years

   6 – 14 years

   > = 15 years

5. Anti - hypertensive Medications:

6. Symptoms ----- Yes / No

   i.
   ii.
   iii.

7. Diabetes Mellitus ------ Yes / No

8. Last 5 blood pressure measurements

   a.

   b.

   c.

   d.

   e.

Examination

Sitting blood pressure:

Lying Blood pressure:

Standing blood pressure

1 min :

3 min :