STUDY ON METABOLIC CONTROL OF HYPERTENSION

By

Major Md. Shamsul Hoque

MBBS (Dhaka), DPH (Dhaka), M.Phil (PSM)

INSTITUTE OF NUTRITION AND FOOD SCIENCE
UNIVERSITY OF DHAKA
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DEDICATED

To

THE MILLIONS OF HYPERTENSIVES

OF THE WORLD

FOR

THEIR SAFE CONTROL &PREVENTION

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PREFACE

Hypertension is very common in most population of the world and is responsible for much morbidity and mortality. Probably half the people with elevated blood pressure are not known to the medical profession and the patients do not know of their condition. Hypertension is a massive health problem affecting at least 8% of the adult population in most countries of the world (WHO 1978)[23]. Because it causes high morbidity, invalidism, death and because it can be identified easily and treated effectively.

The present study provides a guideline on the basis of its findings and the existing state of knowledge of the hypertension problem and the means of assessing and treating patients with metabolic therapy. It makes suggestion for a community approach for prevention and control in countries having varied health care systems, and most important recommendations, to carry out further research for closing major prevailing gaps in knowledge regarding normalisation of blood pressure.

loday with recognition of the risks that accompany elevated blood pressure, millions of hypertensives are receiving pharmacologic treatment that normalizes pressure. Despite gratification with this change there is legitimate concern about the balance between benefit and risk with decades-long use of drugs, on a mass scale, particularly with persons with less severe hypertension.

A reasonable way to cope with this dilema is to attempt, influencing blood pressure favourably, using Metabolic therapies before untreated hypertension has caused damage.

This study and other convincing epidemiologic studies in hypertensive persons confirm that overweight, high salt intake, high fatty diet and physical inactivity have to be controlled effectively.

Combined Military Hospital
Dhaka Cantonment
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Author

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Major Md Shamsul Haque

ABSTRACT

AIM - To ease the suffering of apparently hypertensives, reduce risk factors and unnecessary drug use and give an economically productive and socially desirable life.

OBJECTIVES - To determine the combined antihypertensive effects of dietary changes, exercise, relaxation therapy, estimate the metabolic changes of serum electrolytes, lipid profiles, urea, creatinine and plasma glucose.

DESIGN - A true experimental design that is a pretest and post-test control group design was adopted with common source population, eligibility criteria, data collection and analytic methods. Four week screening period followed by controlled clinical trials with an unmasked design but with blinded measurement of blood pressure as the end point.

SETTING - Subjects were recruited from OPD of Combined Military Hospital, Dhaka, admitted, treated and followed up.

SUBJECTS - A total of fifty six subject, 28 hypertensives as experimental group with diastolic Blood pressure ≤ 110 mmHg and 28 normotensives as control group were matched for age 30-59 years.

INTERVENTIONS - All the subjects have followed a strict regimen after being admitted to the hospital for a duration of three weeks. The combined intervention programmes were walking on a treadmill set with progressive increase in distance and decrease in time, progressive relaxation therapy, deep breathing exercise meditation, low sodium diet (less than 2.5 g NaCl/day) and energy intake less than upto 2000 kcal.

MAIN OUTCOME MEASURES - Subject's baseline resting and pre-programme and post-programme intervention Blood pressure, were the average of many measurements that were taken at different times of day (morning, noon & evening) using mercury sphygmomanometer in sitting posture. The primary changes in diastolic blood pressure from baseline to final follow up, were carefully measured. Secondary changes were in systolic blood pressure, changes in body weight, BMI, P8MI, serum electrolytes, lipid profiles, urea, creatinine, and fasting plasma glucose. The relationship between changes in blood pressure with the changes in other variables were also measured.

RESULTS- Metabolic therapy reduced diastolic blood pressure by 24.75 mmHg and systolic blood pressure 32.82 mmHg in hypertensive group. There was a mean decrease of 5.35 mmHg in the diastolic blood pressure of the control group ($p<.001$) but with only 3.53 mmHg decrease in the mean systolic blood pressure.

The weight loss was ranged from 1 kg to 3 kg in the experimental and 2 kg to 1 kg in the control groups respectively. The mean weight loss was 1.6 kg (± 0.70) and 1.5 kg (± 0.50). In both the groups the weight reduction was highly significant ($p<.001$ CI 95%). Initial mean serum electrolytes for Na, K and NaCl were found to be 140.03 (± 2.35), 4.34 (± 0.41), and 100.45 (± 2.24) mmol/l, while the final level for the same were 138.62 (± 3.39), 4.79 (± 0.61) and 98.96 (± 3.25) mmol/l respectively. The final mean serum cholesterol, LDL, TG, fasting plasma glucose levels were decreased significantly while the mean HDL and serum urea level increased. The mean difference in HDL-cholesterol was 3.49 (± 3.85) and 3.78 (± 3.50) in the hypertensive and the control subjects. The changes of HDL-cholesterol were highly significant ($p<.001$). The effects of statistical concept of regression to the mean on fall of blood pressure were analysed and the fall of blood pressure was confirmed by intervention. After six months follow up, hypertensive subjects had little increase on mean diastolic blood pressure and the change showed limited degree positive co-rrelation ($r=0.8$). All other changes in biochemical variables such as serum sodium chloride, cholesterol, serum TG and fasting plasma glucose were correlated with the fall of blood pressure.

CONCLUSIONS - The findings of the study indicate that combined Metabolic therapy is effective in controlling hypertension in uncomplicated primary essential hypertension. Metabolic therapy is useful in hypertensive persons and is not costly and are beneficial in promoting good health. Metabolic therapy is safe, costless and reduce adverse metabolic effects of some antihypertensive drugs but its long-term consequences for morbidity and mortality remain to be determined.

CHAPTER 1

INTRODUCTION

1.1 BACKGROUND OF THE STUDY:

Hypertension is the most common cardiovascular disease in many countries and thus a great challenge in modern public health[1]. It is a well established risk factors for major cardiovascular diseases. This disease is a secret threat to human health, causes innumerable suffering and death, even in apparently healthy individuals. In 96-99% of cases of hypertension, the cause or causes of blood pressure elevation will not be clearly identifiable[2]. Hypertension is usually symptomless and popularly known as silent killer and major risk factor for coronary, cerebral and renovascular diseases. Prospective studies show that an individual's risk of cardiovascular disease rises with increasing blood pressure, starting from levels so low that they are clinically considered normal[3]. Since many people have "high normal" or "borderline" blood pressure the bulk of the risk attributable to hypertension applies to this large segment of the population[4]. Majority of the patients have diastolic blood pressure ranging from 90-110 mm Hg. It has been observed that many patients do live well without drug and some inspite of drug therapy don't keep well.

Treatment of blood pressure is an important part of prevention and which is indicated when there is end organs damage. The plan of

treatment should include a suitable drug regimen along with regularly reviewing the prognosis of the patients.

No drug is side effect free, so many problems like untowards drug reaction could be avoided by not starting antihypertensive drug treatment. Pharmacological reduction of blood pressure must for several reasons, be restricted to people with definite hypertension; therefore, there is an urgent need for Metabolic control of high blood pressure in this segment of the population[2,4]. Although most patients turned to drugs first, evidence suggests that these can be safely withheld from many hypertensive to allow Non drug therapies, a chance to be effective. Several intervention possibilities for Metabolic control of blood pressure have been documented, for example exercise, weight reduction, stress management, dietary changes, and relaxation technique[1]. Interest in the use of various Non drug therapies for the treatment of hypertension - particularly exercise and dietary changes have risen markedly in the past few years, yet many practitioners either do not use them or use them in a causal, perfunctory manner. This hesitant attitude can be attributed both to the sparseness of firm evidence indicating that these therapies succeed and to the difficulty many have faced in convincing patients to adhere to them. This situation is likely to be changed. The evidence for the effectiveness of these approaches is

growing, techniques for improving adherence are being popularized and patients seem increasingly willing to adopt changes in life style. These changes come at a propitious time when many more people are being identified as hypertensive and are considered in need of lowering their blood pressure.[5-7]

It is important to note here that, the underuse of Metabolic control is due to excitement over newly available potent and synthetic drugs that are being massively promoted by the media services. Furthermore, when physicians decide to treat a condition, they expect almost immediate results with virtual certainty. Such expectations were clearly justify when the majority of patients had fairly severe hypertension; however, as the large number of patients with mild hypertension enter the picture, a more gradual approach to their management seems more appropriate.

It may be further mentioned that in Metabolic control of hypertension if the appropriate type of exercise is performed at the proper intensity, duration, and frequency, sedentary individuals of all ages will achieve significant improvements in physical working capacity. After intervention they will be able to exercise at a greater intensity and for a longer duration than before. Also at the same submaximal exercise intensity they will

experience less fatigue. This increase in functional capacity is due to enhanced metabolic capacity and efficiency of skeletal muscle, increased capacity and efficiency of skeletal muscle, increased capacity for substrate and oxygen delivery to the muscle and changes in autonomic nervous system regulation during exercise. Increases in physical working capacity often are equated inappropriately with improvements in health status or disease prevention. Patients with disorders such as diabetes or hypertension can significantly increase their working capacity through exercise without necessarily changing the severity of their disease or their medical prognosis.

Data from long term controlled studies of a Metabolic therapies document a relatively small but significant reduction in blood pressure both in normotensive[8-9] and in hypertensive, whose therapy has been stopped[5-7]. Of equal if not greater benefit is the concomitant reduction of their risk factors even if the pressure is less than provided by drug therapy[10]-18.

1.2 MAGNITUDE OF THE PROBLEM

Hypertension is one of the most prevalent diseases in the world. It is estimated that 15-20% of people living in developed countries suffer from this condition[19]. Hypertension is also an "iceberg" disease. It became evident in the early 1970s that only about half

of the hypertensive subjects in the general population of most developed countries were aware of the condition, only about half of those aware of the problem were being treated and only about half of those treated were considered adequately treated[18]. If this was the situation in countries with highly developed medical services, in developing countries, the number of treated would be far less. In some industrialized countries, upto 25% of adults have diastolic pressures above 90 mmHg. Prevalence in the developing countries seems to be similar to that in European or other developed societies ranging from 10% to as much as 20% among adult[19].

No single advance in community health would improve the quality of life than the control of hypertension[11,12]. Drug treatment has been recommended even for mild hypertension but implementing this recommendation would mean that majority of the adult population of a country like Bangladesh would require medication for life with substantial problems of cost and compliance and with no effect on primary prevention. An alternative to medication is the metabolic control of hypertension. The beneficial effects of such antihypertensive therapy is the real need for primary prevention of hypertension for a community.

Hypertension is an important public health problem in all countries of the world hence the WHO's commitment to promote

better control, management and treatment[2]. Although virtually symptomless it is an important contributing cause to cerebrovascular, heart and renal disease. It is a chronic disease for which life long drug treatment is required and treatment of hypertension is control, rather than cure. Besides hypertension causes a tremendous financial burden on the patient. Treatment cost of a patient with moderate hypertension will reach taka 10 to 20 per day for which most of the Bangladeshi are unable to afford[12].

Recently doctors have questioned the value of treating certain levels of hypertension. Anomalies exist e.g. drug treatment of hypertension does not invariably reduce coronary heart disease, for which it is an important risk factor. The complex relation between hypertension and other risk factors is only now being teased out, leading to a re-evaluation of values when treatment is justified. Stopping drug treatment, could cause problems. It is important to know the value of any ensuing rise in blood pressure - even to levels that would not justify treatment in new patients. Most doctors have come across patients with normal blood pressure who had previously stopped drug treatment.

Treating hypertension is an important part of preventive medicine, particularly in general practice. Until now patients have been

taught that hypertension means treatment for life, although thousands of them have abandoned treatment without their doctors knowledge or consent. Feeling perfectly well they have made their own judgement about the value of treatment and the acceptability of these side effects[14]. For some patients hypertension is a transitory phenomena linked to other states. When these are removed, time being the biggest healer, the hypertension gets better. Many problems could be avoided by not starting anti-hypertensive drug treatment, before prolonged observation. Attempts should be made to eliminate all possible contributory factors. Patients should no longer be told that treatment is necessary for life[14-15].

Today's drugs aiming to reduce diastolic blood pressure in most cases could all have unpleasant side effects, including headache, sleepiness, flushing, fatigue, drowsiness and impotence[10]. These drug are Beta blockers, diuretics and calcium antagonists and angiotension converting enzyme (ACE) inhibitors.

1.3 WHAT IS HYPERTENSION

Arterial hypertension means the persistent elevation of (arterial) blood pressure[2]. Blood pressure is a continuously distributed variable (Fig 1.1) in population. There is no natural dividing line between "high" and "normal" blood pressure. Since blood pressure in the general population falls on a gaussian curve of normal distribution, it is impossible to define with precision the limits of "normal" blood pressure[18,19]. Furthermore, the blood pressure of a given individual varies widely over time, depending on many variables, including sympathetic nervous system activity, posture, state of hydration and skeletal muscle tone. Accordingly, any definition of hypertension must be arbitrary. The eternal question is at what level pressure should be labeled as hypertension[20-23] ?

The diagnosis of hypertension in adults is made when the average of two or more diastolic blood pressure measurements or at least two subsequent visits is 90 mmHg or higher or when the average of multiple systolic blood pressure readings on two or more subsequent visits is consistently greater than 140 mm Hg. The WHO in its expert committee report has arbitrarily defined hypertension in adults as " a systolic pressure equal to or greater than 160 mm Hg and /or a diastolic pressure (Phase V)

ANNEXURE 1 TO SECTION 1.3

FIGURE 1.1. The continuous distribution of blood pressure values in a population.

(gross.F, Pisa.Z, strasser.T. etal. Management of Arterial Hypertension: A practical guide for the physician and allied health workers. WHO, Geneva : 1984:3.

equal to or greater than 95 mm Hg.[24] The pressure values between elevated and normal are known as borderline. These limits apply to adults of both sexes of any age. Persistent elevation of blood pressure can be ascertained only by repeated measurements over a longer period of observation. A single casual finding of elevated blood pressure does not justify the diagnosis of hypertension. At least there blood pressure readings should be taken on each of at least the two different occasions before declaring that a subject has hypertension.

The definition of blood pressure, therefore using any cut off point is arbitrary[22]. There have been multiple proposed cut off points for the definition of high blood pressure. The simple definition proposed by WHO is based on consensus. In any population, blood pressure values have a continuous bell shaped, unimodal and positively skewed to the upper end. It is not possible to distinguish clearly pathological from "normal" values. Blood pressure is a graded phenomena, associated with graded risk. The higher the arterial pressure - systolic or diastolic- the greater the risk of complications and shorter the life expectancy of the patient. The diagnosis of hypertension alone does not necessarily indicate the need for drug treatment[2,24]. Both transient and persistent elevations of pressure are common when it is taken in the physician's office or hospital[25]. To obviate "white

coat or office hypertension, more wide spread use of out of the office readings either with semiautomatic inexpensive devices[26] or automatic ambulatory recorders,[27] encouraged both to establish the diagnosis and to monitor the patient's response to therapy. Hypertension in as many as half of patients that appears by office readings to be resistant of three or more drugs can be found out-of-office recordings to be well controlled[28]. In one study these results averaged 22/12 mm Hg lower than those recorded in an office by physician[29]. Another observation is that although the logical approach would be to calculate the mean values from multiple reading when deciding whether or not hypertension is present, even a single high measurement should not be disregarded. In large population, single casual measurements have been found to predict a greater likelihood of subsequent cardiovascular disease[30].

1.4 CLASSIFICATION OF HYPERTENSION

The classification of Hypertension is to provide an easy and reliable method for the characterization of each patient. It allows assessment of severity of disease by reference to epidemiological data so that risk can be defined and appropriate treatment instituted. Arterial hypertension may be classified in three separate ways - by etiology, by extent of organs damage & by blood pressure level[28].

1. **CLASSIFICATION BY ETIOLOGY. Essential, primary or idiopathic hypertension** is arterial hypertension of unknown cause. over 95% of all cases of arterial hypertension are in this category.

Secondary hypertension is arterial hypertension of known. Fewer than 5% of all cases of systematic hypertension are in this category[31]. The prevalence of various forms of hypertension in the general population and in specialised referral clinics are shown as under:-

Table 1-1. **Prevalence of Hypertension by type of population**[445]

Diagnosis	General Population %	Speciality Clinic %
Essential hypertension	92-94	65-85
Renal hypertension		
Parenchymal	2-3	4-5
Renovascular	1-2	4-16
Endocrine hypertension		
Primary aldosterorism	0.3	0.4-12
Cushing's syndrome	<0.1	0.2
Pheochromocytoma	<0.1	0.2
Oral Contraceptive induced	2-4	1-2
Miscellaneous	0.2	1

Malignant Hypertension - is the syndrome of markedly elevated blood pressure (diastolic blood pressure usually greater than 140 mmHg) associated with papilloedema.

2. **Classification according to extent of organ damage**[23]

Stages of hypertension

Stage-I No objective signs of organic changes are evident.

Stage-II At least one of the following signs of organ involvement is present - left venticular hypertrophy on physical examination, chest X-ray, E.C.G. ecocardiography etc. Generalised and focal narrowing of the retinal arteries. Proteinuria and/or slight elevation of plasma creatinine concentration (conc).

Stage-III Both symptoms and signs have appeared as a result of damage to various organs from hypertensive disease. These include left venticular failure, cerebral haemorrhage, retinal hemorrhages with or without papilloedema.

9. **Classification according to level.** It has been stated that there is no clear demarcation between "normal" and hypertensive" blood pressure levels. However, according to WHO, normal adult blood pressure is arbitrarily defined as a systolic pressure equal to or below 140 mmHg, together with a diastolic equal to or below 90 mmHg. Hypertension in adults is arbitrarily defined as a systolic pressure equal to or greater than 160 mmHg and/or diastolic (5th phase) equal to or greater than 95 mmHg.

The term borderline hypertension is used to denote blood pressure values between (Fig 1.2) the normal and hypertensive. The terms "mild", "moderate" and "severe" hypertention are used in a

ANNEXURE 2 TO SECTION 1'4

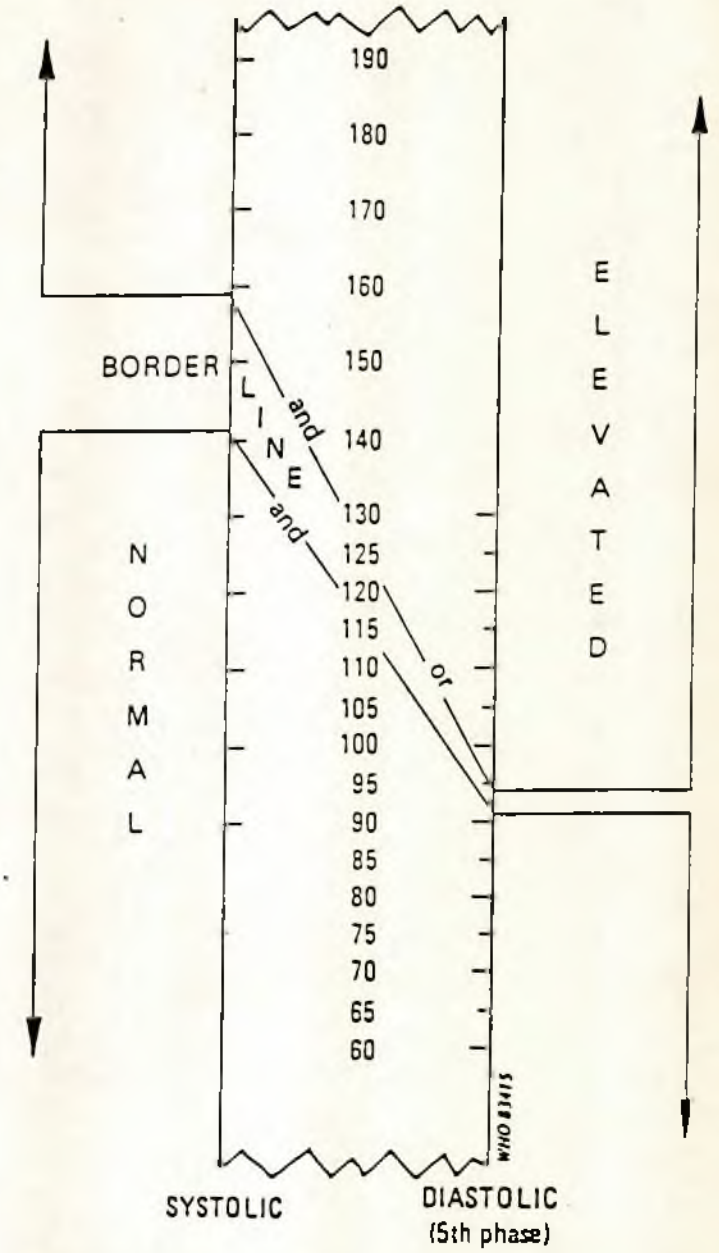


FIGURE 1.2. Normal, borderline, and elevated blood pressure (Mean values of 3 readings on 2 different occasions) (Gross. F,pisa.2 strasser.T, etal. Management of Arterial Hypertension : A practical guide for the Physician and allied health workers. WHO, Geneva:1984:2).

descriptive sense only and not defined precisely. However classification of blood pressure in adults aged 18 years or older is given as under:

Table 1-2 **Classification of BP[10] and follow up criteria for initial measurements**

BloodPressure Range, mmHG	Category
Diastolic Blood Pressure	
<85	Normal blood pressure
85-89	High normal blood pressure
90-104	Mild hypertension
105-114	Moderate hypertension
≥115	Severe hypertension
Systolic blood pressure when Diastolic blood pressure <90 mmHg	
<140	Normal blood pressure
140-159	Borderline isolated Systolic hypertension
≥160	Isolated systolic hypertension

A classification of borderline isolated systolic hypertension or isolated systolic hypertension takes precedence over high normal blood pressure when both occur in the same person. High normal blood pressure takes precedence over a classification of normal blood pressure when both occur in the same person.

In view of the usual variability in blood pressure levels, the term labile is inappropriate for describing diastolic blood

pressure that only occasionally exceed 90 mmHg. The term borderline is more correct.

1.5 INCIDENCE AND PREVALENCE

The concept of incidence has limited value in hypertension because of the variability of consecutive readings in individuals, ambiguity of what is "normal" blood pressure and the insidious nature of the condition[32]. Longitudinal observation of blood pressure reveals a different pattern than cross-sectional data. The reason is obscure. Diastolic pressures are essentially parallel in the sexes, with women's pressures consistently below those of men. Systolic pressures in women, initially lower than those in men, rise more steeply to converge at age 60 with those of men but never exceeding them[33,34]. A progressive and disproportionate rise in systolic pressure with advancing age is presumed to result from loss of arterial elasticity. Blacks have higher blood pressure than whites in most western cultures. The cross over in blood pressure in the sexes appears to occur 10 years earlier in blacks than whites.

According to a survey in the year 1976 to 1980 indicated a hypertension prevalence of 29.7% for persons 18 to 74 years of age in the United States.[35,36] About 60 million persons in the states

have hypertension. The prevalence of blood pressure increases with age in all groups : blacks, whites, men and women. Diastolic blood pressure is roughly twice as common among fifty years-old as among thirty years old and systolic blood pressure increases greatly in prevalence after the age 45. It is a common health disorder in the older population, afflicting about 65% of the population in the 65-74 year old group. The blacks have a higher prevalence of hypertension than white (38% versus 29%) the reason for the increased prevalence of hypertension among blacks is unclear, but it has been attributed to heredity, greater salt intake and greater environmental stress[36]. Men have a higher over all prevalence of hypertension than women (33% versus 27%). Hypertension is more common in men than women up to approximately age 50, after that time, hypertension is more common in women. The increased prevalence of hypertension in postmenopausal women is related to a combination of weight gain and hormonal alternations.

In Bangladesh there is no national epidemiological recording system for hypertension. Nevertheless, several epidemiological studies have been carried out in different groups of population. In 1974-75 a study carried out in and around Dhaka city showed a prevalence of 11 per thousand population[38]. Subsequently study of a rural population showed that 6.7% of the population had diastolic blood pressure equal to or greater than 90 mm Hg with

only 2% having diastolic pressure ≥95 mmHg. As many as 74% of the patients were asymptomatic and 86% undiagnosed before the study. The prevalence was found to increase with age ; only 1.7% of people in the age group 10-19 years were hypertensive whereas 20% people of the age greater than 70 years being hypertensive[37,38]. A study among governmental employees residing in Dhaka showed a prevalence of 13.3% (diastolic blood pressure ≥90 mm Hg) vast majority (71.6%) were not previously diagnosed in recording blood pressure. Diastolic pressure ≥95 mm Hg was found in only 3.7% of the subjects. Prevalence of the disease in a mixed community was 2%[37]. Frequency of the disease among University students was 13.3% (diastolic blood pressure ≥90 mm Hg) of which borderline hypertensive group (diastolic blood pressure between 90 and 95 mmHg) comprised 11.85% of the students. Prevalence of hypertension in Bangladesh appears to be lower than that in the industrialized countries of the world and a good number of cases remain undetected and untreated[39,40].

1.6 BLOOD PRESSURE MEASUREMENT

As far as blood pressure measurement is concerned, discussion continues about its reliability and wide variability in individual subjects. Accurate measurements are essential under standardized conditions for valid comparison between persons or groups over time. Three sources of error have been identified in the recording

of blood pressure : (1) Observer errors e.g hearing acuity, interpretation of korotkoff sounds, (2) Instrumental errors e.g leaking valves, cuffs that do not encircle the arm and too high a reading will be obtained and (3) subject errors e.g the circumstances of examination, these include the physical environment, the position of the subject, external stimuli such as fear, anxiety and so on[41]. Arterial blood pressure can be measured by two methods: (1) direct and (2) indirect. **In direct method**, the artery is exposed and an arterial cannula of which one tapering end is inserted directly in to the lumen of the exposed vessel and the other end is connected to the U shaped mercury manometer that shows the actual blood pressure in mmHg.

In indirect method, the pressure may be measured without any surgical procedure and thus it is very convenient clinically in human being. In this method commonly the pressure of the brachial Artery is measured. The instrument is used is known as sphygmomanometer. Three methods (1) oscillatory (2) palpatory (3) auscultatory are included for indirect measuring.

Oscillatory method: Inspection of oscillation in spring gauge or Hg manometer is the basis of this method. The pressure at which oscillation just becomes smaller or disappears is known as diastolic pressure.

Palpatory method: The instrument is kept at the level of the heart and the cuff is tied round the upper arm. Pressure is raised to 200 mm of Hg and then gradually released. When the pulse just appears at the wrist, the pressure is noted. This is the systolic pressure. This method is not accurate. By this method the diastolic pressure can not be determined.

Auscultatory method: In this method, to obtain an accurate systolic pressure, the cuff should be inflated rapidly to at least 30 mm Hg above the systolic pressure, as determine by the palpation of the radial artery. This inflation is necessary to avoid underestimating the pressure, because of the auscultatory gap, an unexplained disappearance of korotkoffs sound for some interval between systolic and diastolic. The systolic reading is taken as the level of pressure at which clear korotkoff's sound are heard with each heart beat. The diastolic reading is taken at the level when sounds become muffled (korotkoff's phase IV) and when sounds disappear (phase v). The both reading should be recorded. It is not known whether the level of muffling or of disappearance is more accurate reflection of the intra-arterial diastolic pressure, so selection of one over the other as the clinical measurement of diastolic pressure is a matter of convenience and reproducibility. Baseline

blood pressure should be calculated from the average of two separate measurements determined at least 2 weeks apart. However the patients with diastolic blood pressure >115 mm Hg or elevated blood pressure with evidence of on going end organ damage should be started on therapy immediately.[7]

Standard sphygmomano meters and stethoscopes are appropriate for this purpose and are performed over automated indirect blood pressure measuring devices, which are often inaccurate.[42,43,44]

A few salient points need be mentioned about measuring blood pressure. A WHO study group[21] are preferred to the sitting position than the supine position for recording blood pressure.

Uniform policy should be adopted using either the right or left arm consistently. The pressure at which the sounds are first heard (phase 1) is taken to indicate the systolic pressure. Near the diastolic pressure the sounds first become muffled (phase iv) and then disappear (phase v). Most of the studies have used phase v to measure diastolic blood pressure. The systolic and diastolic pressures should be measured at least three times over a period of at least 3 minutes and the lowest reading recorded. The person to be examined should have been seated for at least 5 minutes; relaxing in a comfortable chair. Hg sphymomano meters are

recommended since they are more reliable than the aneroid - type instrument. The dimensions of the cuff are very important; the standard cuffis 12.5 cm wide and sufficiently long to surround the upper arm[45,46]. The instrument should be placed on a horizontal surface and the cuff adjusted firmly, but not too lightly so as not to interfere with blood flow in the arm. As mentioned above blood pressure measurement should take place in a pleasant and reassuring atmosphere. Room temp. should be around 21^{0}c, low temp. causes an elevation of blood pressure and excessive heat provokes tachycardia. The room should be quite to ensure reliable auscultation of the arterial sounds[47,48].

5. **Korotkoff sounds.** When the pressure in the air cuff is lower progressively, after the first tap is heard after release of pressure, the sounds undergo a series of changes known as Korotkoff's sounds. Four phases are heard.

PHASE I Sudden appearance of faint but clear, tapping sound becoming louder for the succeeding 10-14 mmHg fall of pressure.

PHASE II It becomes murmurish, during next 15-20 mmHg fall of pressure.

PHASE III It becomes increasingly clearer and louder during another 5-7 mmHg fall of pressure

PHASE IV The sound becomes muffled during the last 5-6 mmHg fall of pressure, after which it disappears.

PHASE V An additional phase is often mentioned when the sound disappears completely which is usually within 10 mmHg of phase IV. Diastolic pressure measured directly through an intra-arterial needle and external manometer corresponds closely to phase V[94].

6. The auscultatory gap. This is a silence that some times separates the first appearance of the korotkoff sounds from their second appearance at a lower pressure. Mostly in hypertensive, the appearance of the first sound may be followed by a gap of as much as 40 mm Hg before reappearance of sound. This is called the auscultatory gap[44,45], thus one can under estimate the systolic pressure or over estimate the diastolic pressure, unless the auscultatory gap is excluded by palpating or disappearance of radial pulse as the cuff is raised. After disappearance of all sounds the cuff is deflated rapidly not to be reinflated for at least one or two minutes for release of blood trapped in the vein.

1.7 MECHANISMS OF PRIMARY (ESSENTIAL) HYPERTENSION

The cause of elevated blood pressure cannot be identified in more than 95% of cases, these individuals are said to have essential

hypertension. Since, persistence hypertension can develop only in response to an increase in cardiac output or a rise in peripheral resistance, defects may be present in one or more of the multiple factors that affect these two forces. The interplay of various derangements in factors affecting cardiac output (co) and peripheral resistance (PR) may precipitate the disease. looking for a single defect in all patients with essential hypertension may be a mistake[49,50].

1. **Haemodynamic principles.** Under ordinary circumstances, pressure in the Arterial system is maintained within fairly narrow limits in normotensive individuals. The exceptions to this generalization occur during sleep and exercise. During sleep arterial pressure may fall as low as 60/40 mm Hg and during exercise there are marked rises, particularly in systolic pressure. To understand the haemodynamics of hypertension it will be helpful to note shortly, some basic principles of hydrodynamics as they relate to the flow of blood throughout that vascular system.

The flow of liquid in a tube is related to the gradient of pressure along the tube and the resistance the liquid meets as it flows. Resistance (R) can not be measured directly, so, it is expressed as the ratio of the pressure gradient (P) to the flow

rate (F). R=P/F. This equation can be substituting TPR = MAP/CO where

TPR (Total peripheral resistance) = R

MAP (Mean arterial pressure) = P

CO (Cardiac output) = F

MAP is the integrated pressure over one or several cardiac cycles. This integration can be obtained by damping pulsatile pressure or by circulating it as diastolic pressure plus one - third pulse pressure. TPR is the sum of the resistance in all the vascular beds of the body. These are not necessarily equal, nor do they always change in the same direction. Thus for example, a fall in TPR does not mean that vasodilatation has occurred in each vascular bed; in some it may be unchanged, in others even increased, but enough vasodilation has occurred some where to result in an overall decrease[35]. In haemodynamic pattern, the basic equation, Blood pressure = CO x PR that have been measured in patients with hypertension may be considered. The pathogenesis of the disease (Fig 1.3) is probably a slow and gradual process. Regardless of how hypertension begins, the eventual primacy of increased resistance can be shown even in models of hypertension that feature an initial increase in fluid volume and cardiac output[51,52].

ANNEXURE 3 TO SECTION 1'7

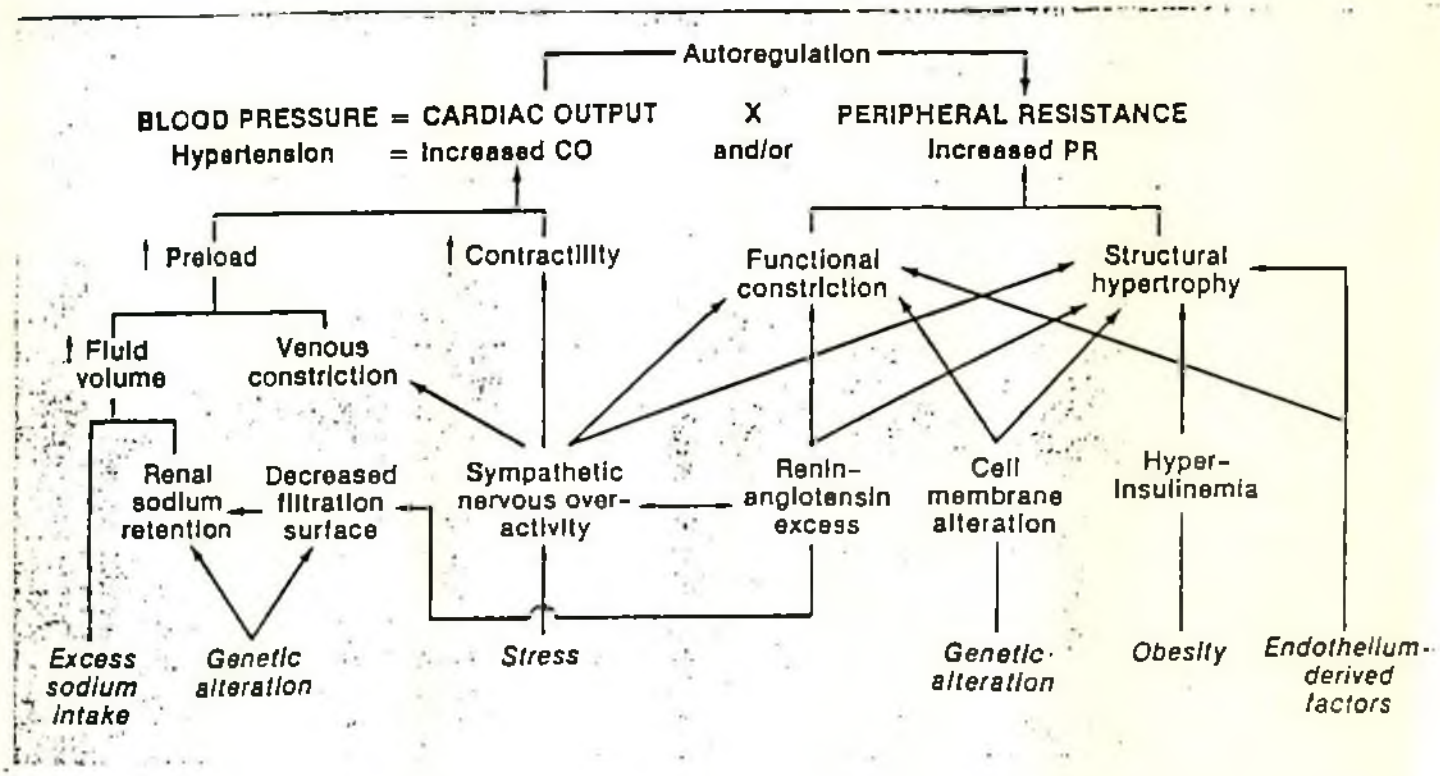


FIGURE. 1.3. Some of the factors involved in the control of blood pressure that affect the basic equation: blood pressure= cardiac outputX peripheral resistance. Cellular hyperplasia may be seen along with hypertrophy. (From Kaplan, N.M: Clinical Hypertension,5th ed, Baltimore,@ by Williams and Wilkins,1990,p.57).

2. **Genetic Predisposition.** Genetic alternations may initiate the cascade to permanent hypertension. Clearly, heredity plays a role, although no discriminatory gene markers are currently available[53] . If heredity does indeed play a role, what is inherited? A number of possibilities have been suggested, including a heightened sympathetic nervous response to stress, a defect in renal excretion of sodium and a defect in the transport of sodium across cell membranes.

3. **Vascular Hypertrophy.** Hypertropy of blood vessels, whether primary or secondary to increases in blood pressure and hence vessel wall tension, plays an important role in the pathogenesis of hypertension. With the knowledge that an increased P R is both necessary and sufficient to perpetuate hypertension even if it starts with an increased cardiac output. Although functional constriction of vascular smooth muscle is portrayed as a possible mechanism, it appears that the high PR in hypertension is mainly determined by structural hypertrophy which in turn, gives rise to a generalised increase in contractility of vascular smooth muscle[54]. Some authorities believe that this is due to an increase in sympathetic nervous activity while other believe that there is a fundamental defect in the vascular smooth muscle.

4. **INSULIN RESISTANCE.** Insulin resistence has been described in essential hypertension. The level of insulin resistance and the

ANNEXURE 4 TO SECTION 1'7

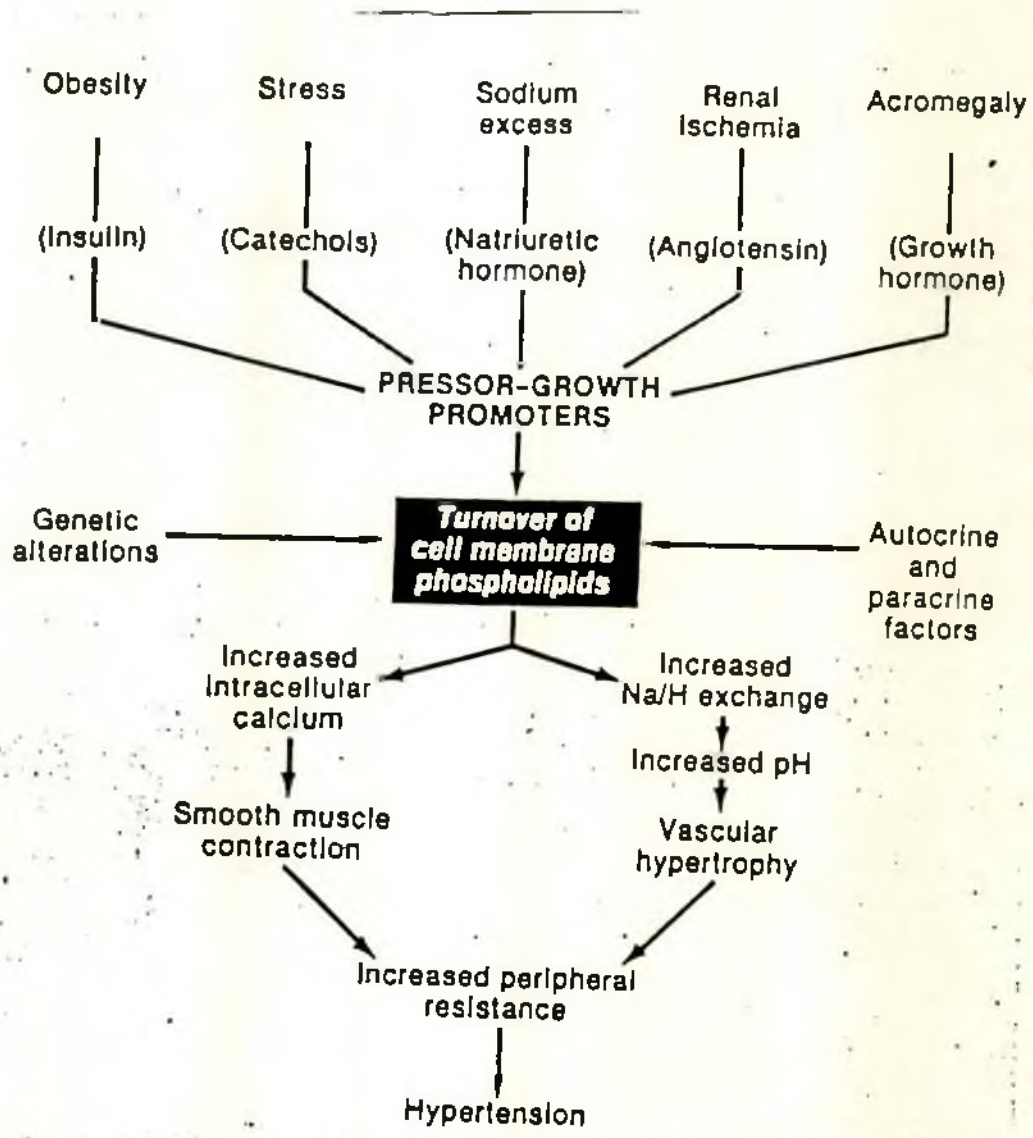


FIGURE.1.4. Scheme for the induction of hypertension by numerous pressor hormones that act as vascular growth promoters.(From Kaplan,N.M: Clinical Hypertension,5th ed, Baltimore,@ by Williams and Wilkins,1990,p.93).

defect in whole body glucose utilization are positively correlated with the severity of the hypertension[55,56]. Insulin resistance in hypertension appears to independent of both obesity and glucose tolerance. Whole body insulin induced glucose uptake and glycogen synthesis and glycolysis are markedly reduced and plasma insulin levels are elevated, presumably as a compensatory mechanism, in insulin resistant subjects. Several mechanisms have been hypothesized to explain the relationship between hyperinsulinemia and blood pressure elevation.

5. **DEFECTS IN CELL TRANSPORT**. It has been observed that many evidences support a causal role for sodium in the genesis of hypertension. This evidence includes an increase in intra cellular sodium in hypertensive animals and people, an increase that extends even to the normotensive children of hypertensive parents[57].

6. **AUTONOMIC FUNCTION**. Elevated blood pressure in essential hypertension is usually initiated and maintained by autonomic nervous system. In patients with early hypertension, there is evidence for diminished resting para sympathetic inhibition and enhanced sympathetic stimulation of the cardio-vascular system. These patients have elevated plasma renin and nerepinephrine levels and enhanced vascular response to stress as consequence of

their increased sympathetic activity. Patients with essential hypertension have an exaggerated pressure response to stress and an exaggerated depressor response to relaxation. Blood pressure falls during meditation and other states of relaxation and rises during exercise. The impressive fall in blood pressure that is frequently seen when a hypertensive patient is removed from his home environment and brought in to the hospital suggests that environmental stress exacerbates hypertension. Stress usually mediates its pressure effect through the sympathetic nervous system.[17]

7. **THE RENIN-ANGIOTENSIN SYSTEM.** Both as a direct pressure and as a growth promoter the renin angiotensin mechanism may also be involved in the pathogenesis of hypertension. All functions of renin are mediated through the synthesis of angiotension II. This system is the primary stimulus for the secretion of aldosterone (Fig 1.7) and hence mediates the mineralocorticoid response to varying sodium intakes and volume loads. When sodium intake is reduced or effective plasma volume shrinks, the increase in renin-angiotension II stimulates aldosterone secretion, and this, in turn, is responsible for a portion of the enhanced renal retention of sodium and water[58-61]. In addition to this primary role in the preservation for normal fluid volume, the renin angiotensin system participates in the control of blood pressure under circumstances of sodium depletion or volume contraction (Fig 1.5).

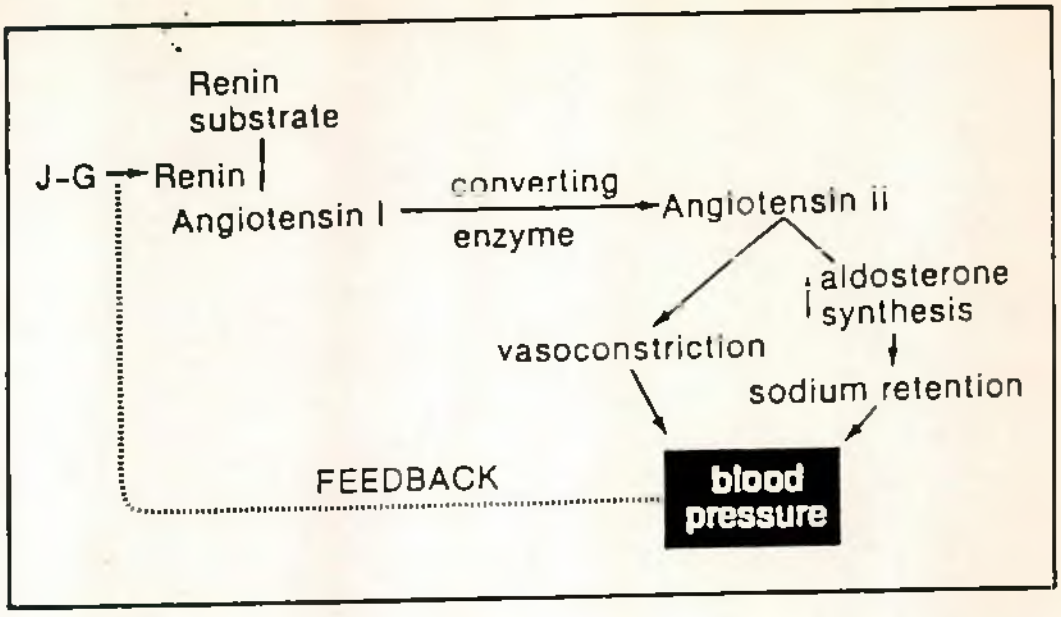


FIGURE.1.7. Overall scheme of the renin-angiotensin mechanism.

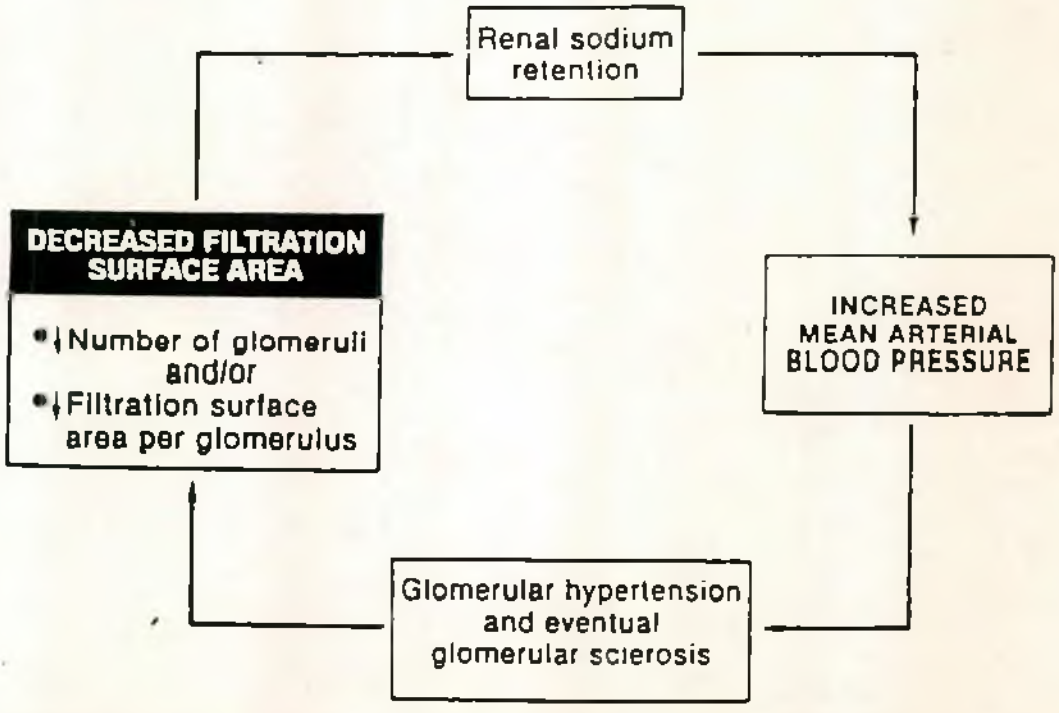


FIGURE.1.5. Relationship between decreased filtration surface area (FSA) and mean arterial pressure (From Brenner ,B.M.Garcia,D.L. and Anderson, S: Glomeruli and blood pressure. Less of one more the other ? Am J Hypertents 1.335,1988.

1.8 RISKS FACTORS OF HYPERTENSION

Blood pressure is a graded phenomena and no clear distinction can be made between "hypertension" and "normal". It is also a graded risk, the risk increasing in proportion to the level of pressure[23]. It is not only one of the major risk factors for most forms of cardiovascular disease, but that it is a condition with its own risk factors. A WHO Scientific group has recently reviewed the risk factors for essential hypertension[19]. This may be classified as:

1. Non-Modifiable risk factors

   a. **Age:** Blood pressure rises with age in both sexes and the rise is greater in those with higher initial blood pressure. Age may represents an accumulation of environmental influences and the effects of genetical endowments in body systems. Some populations have now been identified whose mean blood pressure does not rise with age[21]. These communities are for the most part primitive societies with calorie and often salt intakes at subsistence level.

   b. **Genetic factors.** There is considerable evidence that blood pressure levels are influenced partly by genetic factors and the inheritance is polygenic. The evidence is based on twin and family studies. Twin studies have confirmed the importance of genetic factors in hypertension. The blood

pressure values of monozygotic-twins are usually more strongly correlated than those of zygotic twins. In contrast, no significant correlation has been noted between husbands and wives and between adopted children and adoptive parents[19]. Family studies have shown that the children of two normotensive parents have a 3% possibility of developing hypertension, whereas this possibility is 45% in children of two hypertensive parents. Blood pressure levels among first degree adult relatives has also been noted to be statistically significant[19]. Attempts to find out genetic markers that are associated with hypertension have been largely unsuccessful. The detailed mechanism of heredity i.e. how many genes and loci are involved and their mode of inheritance have not yet been conclusively elucidated[62,63].

2. **Modifiable risk factors**

   a. **Obesity:** Epidemiological observations have detected obesity as a risk factor for hypertension[64]. The greater the weight gain, the greater the risk of blood pressure. Data also indicate that when people with blood pressure lose weight their blood pressure generally decreases[65].

   b. **Salt intake:** There is an increasing body of evidence to the effect that a high salt intake (i.e. 7-8 g/ day) increases blood pressure proportionately. Low sodium intake has been found to lower blood pressure[12,66]. For

instance, the higher the incidence of hypertension is found in Japan where

ANNEXURE 6 TO SECTION 1'7

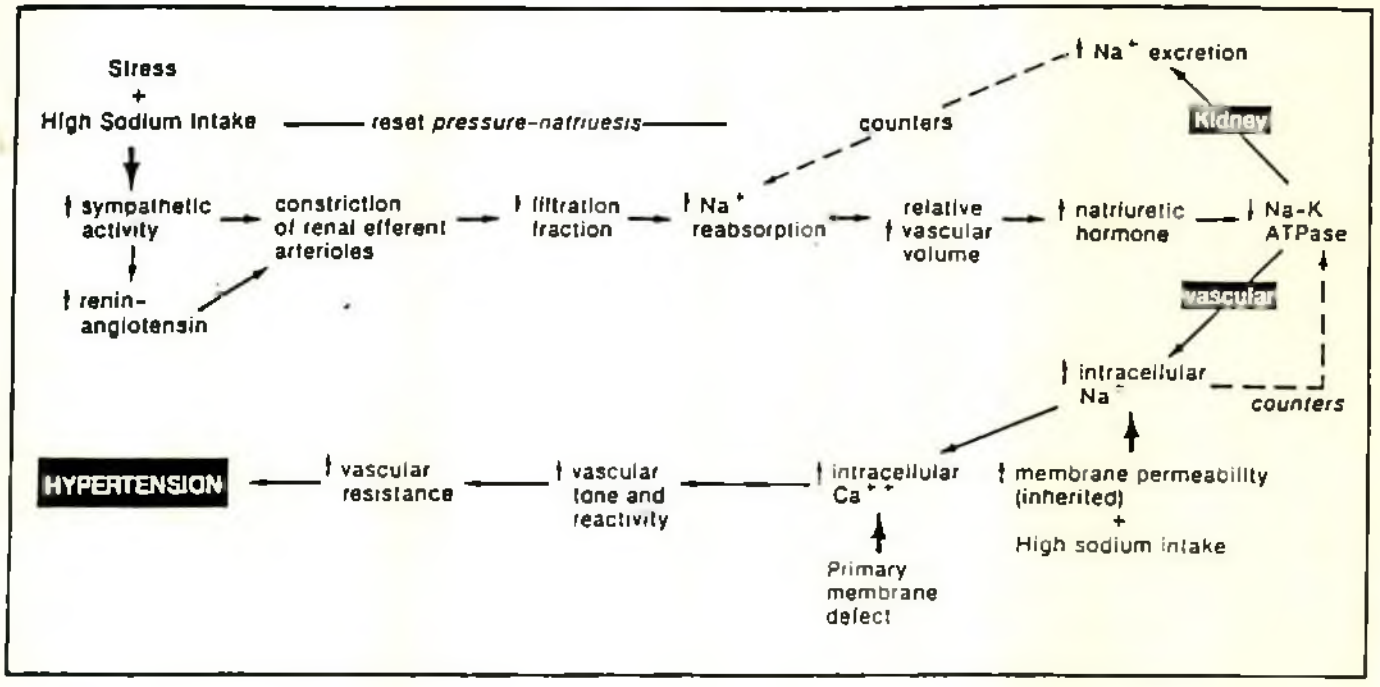


FIGURE.1.6. Hypothesis for the pathogenesis of primary (essential) hypertension, starting from three points, shown as heavy arrows. One starting on the top left is the combination of stress and high sodium intake, which induces an increase in natriuretic defect in sodium transport plus a high sodium intake to induce an increase in intracellular sodium. The third invokes a primary defect in the binding of calcium to cell membranes.

sodium intake is above 400 mmol/day while primitive societies ingesting less than 60 mmol/day have virtually no hypertension[67,68]. It has been postulated that essential hypertensives have a genetic abnormality of the kidney which makes salt excretion difficult except at raised levels of arterial pressure[19]. Besides sodium, there are other mineral elements such as potassium which are determinants as blood pressure. Potassium antagonises the biological effects of sodium, and thereby reduces blood pressure. Potassium supplements have been found to lower blood pressure of mild to moderate hypertensives. Other cations such as calcium, magnesium have also been suggested as of importance in reducing blood pressure levels.

c. **Saturated fat:** Recent evidence suggests that saturated fat raises blood pressure as well as serum cholesterol[1].

d. **Alcohol:** High alcohol intake is associated with an increased risk of hypertension. It appears that alcohol consumption raises systolic blood pressure more than the diastolic[61]. But the finding that blood pressure returns to normal with abstinence suggests that alcohol induced elevations may not be fixed and do not necessarily lead to sustained blood pressure elevation[19].

e. **Physical activity.** Physical activity by reducing body weight may have an indirect effect on blood pressure.

f. **Environmental stress:** The term hypertension itself implies a disorder initiated by tension or stress. Since stress is nowhere defined, the hypothesis is untestable[18]. However, it is an accepted fact that psychosocial factors operate through mental processes, consciously or unconsciously, to produce hypertension. Virtually all studies on blood pressure and catecholamine levels in young people revealed significantly higher nor adrenaline levels in hypertensives than in normotensives. This supports the contention that over activity of the sympathetic nervous system has an important part to play in the pathogenesis of hypertension[19].

1.9 COMPLICATIONS OF HYPERTENSION

With the elevation of blood pressure, the more likely that various cardiovascular diseases will develop prematurely through acceleration of atherosclerosis in uncontrolled hypertension. If untreated, about 50% of hypertensive patients die of coronary heart disease or congestive failure, about 33% of stroke and 10 to 15% of renal failure[69]. Those with rapidly accelerating hypertension die more frequently of renal failure, as do those who are diabetic, once proteinuria or other evidence of nephropathy develops[70]. It is easy to underestimate the role of hypertension in producing the

underlying vascular damage that leads to these cardiovascular catastrophes. Death is usually attributed to stroke or myocardial infarction. Moreover hypertension may not persist after a myocardial infarction or stroke. In general, the vascular complications of hypertension can be considered as either hypertensive or atherosclerotic. The former are more directly caused by the increased blood pressure per se and can be prevented by lowering this level, the later have more multiple causations. Although hypertension may represent the most significant factors of atherosclerosis in qualitative terms, covering of blood pressure may not by itself halt the athorosclerotic process[71]. However the vascular complication of hypertension are given as under:

Table 1-3. Vascular complications of Hypertension[71]

Hypertensive	Atherosclerotic
Accelerated malignant phase	Coronary heart disease
Haemorrhagic stroke	Sudden death
C.C.F	Other arrhythmias
Nephrosclerosis	Atherothrombotic stroke
Aortic disection	Peripheral vascular disease

1.10 METHODS OF TREATMENT

When the diagnosis of hypertension has been firmly established, the need for therapy should be considered. If the diagnosis of

essential hypertension is confirmed, the physician needs to evaluate the severity of the condition. In patients with moderate to severe hypertension the decision whether to treat will be taken without much hesitation[7]. The ultimate goal is to lower the blood pressure to normotensive level or at least to lower the diastolic to below 90 mmHg. Recent trials have shown that an important predictor of risk is the level of blood pressure, particularly systolic blood pressure, achieved during treatment. Reduction in systolic blood pressure should also be an important goal of treatment[72]. However, most patients in whom hypertension is diagnosed for the first time have mild hypertension and it is among this group of patients that is difficult to decide whether to treat or not.

1. **Non-drug therapy.** Several Non drug therapies have been shown to lower the blood pressure in patients with mild hypertension. Weight reduction in over weight subjects, cessation of excessive alcohol consumption, regular exercise in sedentary patients and in some patients, sodium restriction are effective in lowering the blood pressure. Efforts to reduce blood pressure using Non-pharmacological methods should normally precede any decision about the necessity for drug treatment of mild hypertension. Note that some of these measures may take longer duration to become fully effective[72]. If a decision is made to begin drug treatment, a

structured programme of Non-drug therapies remains an essential component of the overall therapeutic programme.

2. **Antihypertensive drugs.** Many drugs are available for the treatment of mild hypertension. These include diuretics, beta-adrenoceptor blocking drugs, alpha-blocking drugs, ACE inhibitors, calcium channel blocking drugs. Serotonin, antagonists, centrally acting drugs and peripheral vasodilator drugs. Most of these drugs are approximately equally effective in reducing blood pressure in patients with mild hypertension, the choice of drug used may be influenced by the individual patient's characteristics and responses[69]. The cost and side effects of drugs are also important consideration[70-72]. Treatment may be started with one of the following categories of drugs.

a. **Diuretic drugs.** Diuretics have been widely used as first line hypertensive therapy and have been shown to be clearly effective in the prevention of stroke. Particularly in large doses, they may cause a variety of unwanted metabolic effects, ventricular ectopics and impotence, so that the dose should be kept as low as possible. Low dose diuretics remain effective and cheap. They are also particularly valuable as ancillary treatment to enhance the effectiveness of other drugs. Combination of diuretics with potassium-sparing drugs including ACE inhibitors, may prevent potassium depletion[72-80].

b. **Beta-adrenoceptor blocking drugs.** Beta-blocking drugs are widely and effectively used to initiate treatment for mild hypertension. Numerous drugs of this type are available. Some with cardio-selective properties, others with partial agonist activity and others with alpha blocking or vasodilator properties. Although beta blockers have been shown to prevent secondary complications of a previous myocardial infarction[72], their role in primary prevention is not established. Beta-blocking drugs should be avoided in patients with reactive airways, obstruction, heart failure, peripheral vascular disease and, possibly, hyperlipidaemia. They are particularly useful in hypertensive patients with angina or rythm disturbances[81-84].

c. **ACE-inhibitors.** Angiotensin-converting enzyme inhibitors are effective in lowering blood pressure. They are generally well tolerated and have been shown not to exert untoward effects on serum lipids and glucose homoeostasis. Among possible adverse effects, persistent cough, angioedema and in patients with vascular disease, deterioration in renal function have been reported[72]. ACE-inhibitors may increase the risk of early fetal death and should be avoided in women considering child bearing. Their safety profile is otherwise good. As with all recent classes of anti hypertensive agents, the ability of ACE-inhibitors to reduce mortality in hypertension has not yet been tested in controlled clinical trials.

d. **Calcium Antagonists.** There are three major classes of calcium antagonists with different characteristics (papaverine, dihydropyridine and benzodiazepine derivatives) but all are effective in lowering the blood pressure. The troublesome side effects may include reflex tachycardia, ankle oedema, flushing and constipatior. Like ACE-inhibitors, they do not have undesirable metabolic effects and their safety profile appears good, but their ability to reduce mortality has not been tested in controlled clinical trials.

e. **Other classes.** Alpha-adrenergic blockers have been used for many years with successful reduction of blood pressure and limited side-effects. Centrally acting drugs are also effective antihypertensive agents and have been used for many years, although the side effect profile is less favourable than for the antihypertensive agents previously mentioned. Others interesting classes of compounds have recently been introduced, such as Serotonin antagonists, but with all new classes and new anti hypertensive compounds any recommendation for general use as a possible first choice has to await the result of extensive clinical experience.

f. **Combination of drugs.** When a single dose is ineffective it is reasonable to substitute a different drug. If a single drug has

been partly effective it may be preferable to add a small dose of a second drug rather than increase the dose of the first[77]. Effective combination include (1) thiazide-diuretic with betablocker or ACE inhibitor (2) beta blocker with dihydropyridine calcium antagonist (3) ACE inhibitor with calcium antagonist. Non drug measures should be continued in order to minimize the required number and dose of drugs and to control other risk factors[85-90].

1.11 JUSTIFICATION OF THE STUDY

High blood pressure is a common disorder of civilization particularly amongst the middle and the old age groups. The detection of high blood pressure and its pronouncement by the physician created anxiety in a patient. In 30 to 40% of the patients[31] whose initial diastolic measurement exceeds 90 mmHg, repeat readings taken soon after will be well below this value. Such patients should be advised that their blood pressure is borderline elevated. What will be done for patients of 30 or 40 years age with diastolic pressure 95-105 mmHg ? The best answer is to have Metabolic therapy[22,24]. Improve the life style, decrease obesity and salt intake and have an exercise programme, stop smoking and alcohol. This is in preference to exposing a symptomless individual to a life time of drugs and the miseries of the complications they bring. Statistics of benefit of drugs in a large group only inform, as how many asymptomatic individuals expose to risk get a disease but will not identify the particular individual requirement of metabolic preventive therapies. On the otherhand drug treatment of hypertension causes a drain in the national economy, metabolic control will enable to reduce such economic consequences for an individual, family and community. Furthermore, drug treatment always need constant supervision and follow up by concerned physician whereas metabolic therapies could

be practised by the patients himself at his own environment. It is worthwhile to note that antihypertensive drugs are not to be stopped if the blood pressure found to come back to normal. Stoppage of the drugs will cause the blood pressure to shot up again probably above the previous level. Non drug therapy if practised by the patients he could very well develop the habits to control his hypertension and therefore, the need for drug treatment and its various problems will substantially be reduced.

The most health related benefit of Metabolic control appear to result from the increase in metabolism required to provide the energy needed for skeletal muscle contraction. This increase in demand for energy triggers a number of adaptation designed to enhance the efficiency and capacity of the skeletal muscle to perform work and minimize fatigue. Adaptations also occur in those systems that support the increased energy requirements of skeletal muscle, including nervous, endocrine, cardiovascular, respiratory and skeletal systems. The area of greatest scientific enquiry regarding the health benefits of exercise has been its potential role in the prevention of heart disease. A frequently unrecognised health benefit of Metabolic control is the effect on carbohydrate metabolism.

Evidence about the effectiveness, mode of action, safety and patient acceptance of the various Non-drug and Metabolic therapies may provide enough antihypertensive effect of lowering blood pressure of many patients with mild hypertension to a safe level without antihypertensive drugs. Therefore, this study in Bangladeshi hypertensive patient is useful in terms of easing patient's suffering, cost, compliance as well as avoidance of side effects of the drugs used in hypertension.

This study also has relevance to National health priorities because hypertension is a very common condition. It is the important public health problems in all countries of the world including Bangladesh, hence the present study will fulfill WHO's commitment to promote better control, management and treatment to the hypertensives.

1.12 GENERAL OBJECTIVES OF THE STUDY

1. To ease the suffering of apparently hypertensives.
2. To reduce the unnecessary drug use for the prevention of adverse metabolic effects.
3. To undertake in the context of overall cardiovascular management of risk factors and overall reduction of risks.
4. To give a economically productive and socially desirable life.

1.13 STATEMENT OF HYPOTHESES

1. Low sodium is associated with low blood pressure.
2. Antihypertensive effects of exercise and/or relaxation therapy can reduce blood pressure in both hypertensive and normotensive groups.
3. Blood pressure do not differ with the changes in blood lipids.

1.14 SPECIFIC OBJECTIVES

1. To decrease the diastolic pressure to below 90 mmHg in the hypertensive group.
2. To determine the combined antihypertensive effects of dietary changes (low sodium and low calorie diet) and to

3. Assess the antihypertensive benefits and value of exercise and relaxation therapies under controlled hospital conditions.
4. To find out the metabolic changes of serum electrolytes, urea, creatinine, lipid profiles and fasting plasma glucose in both experimental and control groups.

1.15 KEY VARIABLES

Concepts	Variable	Indicator	Category
Characteristics of subjects	Age	% of subjects by major age groups	Systolic BP Diastolic BP
	Height	Height in meter	-
	Weight	Ideal, over weight	Initial and changed
	BMI	Ideal, lowest height	Initial and changed
	PBMI	Lean, obese very obese	Systolic BP Diastolic BP
Smoking Habit	History Smoking Cigarette	% of smoker & non smoker	Systolic BP Diastolic BP
Blood Pressure	Systolic BP	160+	Initial & changed
	Diastolic	85-89 90-104 105-109 110	
Occupation	Nature of Job/Trade	% Service % Retired % Others	Army Civilian Entitled
	Physical Inventory	% Sedentary % Hard working	"
Routine Laboratory Investigation	Blood for complete picture	Hemoglobin % Total count RBC WBC Differential Count ESR	Systolic BP Diastolic BP
	Urine Test	For Sugar Albumin Others	

Concepts	Variable	Indicator	Category
Cardiac Involvement	E.C.G	%Ischaemia	"
		% Infarction	"
	X-Ray Chest	Left ventricular hypertrophy	"
Serum electrolytes	Serum Sodium	Normal high or low blood pressure	Initial and changed
	Serum potassium	"	Experimental and control
	Serum chloride	"	Initial and changed
Lipid profiles	Serum Cholesterol	Normal high or low blood pressure	Experimental and control group
	HDL-Cholesterol	"	Initial and changed
		"	Experimental control
	LDL-Cholesterol	Increased or decreased level in relevance to BP	Initial and changed experimental and control
	Serum Triglycerides	"	Experimental and control
	Serum/ ß-lipoprotein	"	Initial and changed
	Serum total lipids	"	Initial and changed
Others	Serum urea	"	
	Serum creatinine		Experimental control
	Fasting plasma glucose		Initial and changed

1.16 DEFINITION OF TERMS & CONCEPTS

1. **Normal Blood Pressure** The normal blood pressure in adults is equal to or below 140 mmHg systolic and 90 mmHg diastolic.
2. **Hypertension:** The hypertension in adults as a systolic pressure equal to or greater than 160 mmHg and/or a diastolic pressure (phase V) equal to and greater than 95 mmHg.
3. **Mild Hypertension:** The term mild hypertension in adult has been defined as a diastolic pressure (phase v) persistently between 90 and 105 mmHg.
4. **Borderline Hypertension:** The pressure values between elevated and normal are known as borderline.
5. **Blood Pressure:** It is the lateral pressure exerted by blood per unit area of vascular walls while flowing through it. It is expressed as millimeters of mercury or dynes per square centimeter. Its function is to maintain a sufficient pressure head, to keep the blood flowing and filtration at capillary bed. The following terms are common in relevance to Blood pressure.

a. **Systolic Pressure:** The maximum pressure during systole. It undergoes considerable fluctuations. excitements, exercise, meals etc, increase it, while

sleep, rest etc., diminish it. The peak systolic pressure is determined by the volume and velocity of the left ventricular ejection, the peripheral arterial resistance, the distensibility of the arterial wall, the viscosity of the blood, and the end diastolic volume in the arterial system.

b. **Diastolic Pressure:** The minimum pressure during diastole at phase v. It undergoes much less fluctuations in health and remains within limited range. Increase of diastolic pressure indicates that the heart is approaching towards its failure. Therefore, the variation of diastolic pressure are of greater prognostic importance than those of systole. Diastolic pressure is the measure of peripheral resistance. It indicates the constant load against which the heart has to work.

c. **Mean Pressure:** It is roughly the arithmetic mean of the diastolic and the systolic pressures. But a close approximation to the mean pressure may be obtained by adding the diastolic pressure with the 1/3rd of pulse pressure.

d. **Basal Blood Pressure:** When an individual is with the least possible amount of strain or stress, basal blood pressure is generally considered.

6. **CARDIAC OUTPUT** : To each minute, certain amount of blood is pumped out by each ventricle into the circulation. This is called cardiac output. Two terms are used:-

   a. **Stroke Volume** or **Systolic Discharge** (output). It means the output per ventitricle per beat.

   b. **Minute Volume** (Minute output) Minute volume means the output per ventricle per minute. Hence minute volume = stroke volume x heart rate.

7. **PERIPHERAL RESISTANCE**. It is the resistance which blood has to overcome while passing through the periphery. The chief seat of peripheral resistance is the arterioles and to a smaller extent the capillaries. It depends on the velocity of blood, viscosity of blood, elasticity of arterials walls & lumen of the blood vessels. Blood pressure = cardiac output x peripheral resistance.

8. **PRIMARY OR ESSENTIAL HYPERTENSION.** This is the type of hypertension in which the underlying cause is not known. This group comprise more than 95% of all hypertensive subjects.

9. **SECONDARY HYPERTENSION.** Secondary hypertensin is hypertension of known aetiology. Less than 5% of all the cases of hypertension belong to this group.

10. **METABOLIC CONTROL OF HYPERTENSION.** The term metabolism is meant by a series of specific biochemical reactions occurring within the living organism (after absorption of product of digestion) into the cell or tissue till its excretion, of which some are concerned with tissue synthesis and others with tissue breakdown what are termed as anabolism and catabolism respectively. The means of achieving metabolic control of hypertension is meant to lower blood pressure by exercise, weight reduction, relaxation and dietary modifications. This increase in functional capacity, adaptation is mainly due to enhanced metabolic capacity and efficiency of skeletal muscle to perform work and minimize a fatigue, increase capacity and energy for substrate and oxygen and nutrients delivery to the muscle including nervous, endocrine, cardiovascular respiratory, skeletal systems and changes in autonomic nervous system regulation and also for the removal of waste products to maintain the internal equilibrium of the cells.

11. **NON DRUG THERAPY OF HYPERTENSION.** The treatment of hypertension which includes, for example, dietary changes, physical exercise, weight reduction, relaxation and meditation either alone or in combined therapies.

12. **BODY MASS INDEX (BMI)**

BMI = Weight (w) in kilogram/[Height(H) in meters]^2 W/H^2

Normal values: MEN= W/H^2 = 20 to 25

WOMEN= W/H^2 = 19 to 24

W/H^2 ratio over 25 is distinctly obese

13. **PERCENT BMI (PBMI)**. When the ratio of BMI and standard BMI (standard BMI for male = 22.1) is multiplied with 100 then it is called PBMI

PBMI < 80	=	Lean (under weight)
PBMI 80-120	=	Normal
PBMI 121-139	=	Obese
PBMI 140+	=	Very obes

14. **BODY ELECTROLYTES**. The predominant cations of the body fluids are Na^+ (140 mmol/L) while K^+ (4.5 mmol/L) Ca^2 (1.2 mmol/L) and Mg^2 (0.7 mmol/L are present in much lower conc. The predominant anions are cl^- (100 mmol./L) and Hco_3^- (26 mmol/L). All the cells whether in health or disease behave as perfect osmometres i.e water moves freely across cell membranes and the cell volumes alter. The body content of Na for a 70 kg adult. as determined by cadaver analysis. is about 4200 mmol. The exchangeable sodium determined by isotope dilution methods is about 3000 mmol of which the greater part (1800 mmol) is in extracellular fluid. The exchangeable potassium content

of the body is about 3200 mmol for a 70 kg man (95% of total). The measurement of cl^- is of secondary importance. Although it is the main anion of extracellular fluid, its conc. is effected by the conc. of other anions and of the main cations . The sum of Na^+ and K^+ conc. on average exceeds those of cl^- and HCO_3 by 17 mmol/L, the so called anion gap.

15. **LIPOPROTEINS** (lipids transport protein)

Lipids are insoluble, but their attachment to proteins of different densities makes them soluble for transport as lipoproteins. All lipids are transported as lipoproteins except FFA which is bound to albumin. The lipoproteins are synthesized and secreted by the intestine and the liver. The plasma lipoprotein estimation therefore, reflect the lipid conc. The various proteins to which lipids are attached are:-

a. **alpha**, a high density protein (HDL), forming alpha lipo proteins

b. **beta**, a low density protein (LDL) forming betalipo protein. This transports about two thirds of the circulating cholesterol that is implicated for CHD.

c. **Pre-beta** a very low density protein (VLDL). This transports triglycerides released from the liver. High conc. of it is usual in hypertriglyceridemia.

d. **Chylomicrons** which mainly transport dietary triglycerides. Thus most of the plasma cholesterol is in low density lipo protein. while most of the triglyceride is in VLDL.

Table 1-4. **Lipoproteins and their major components**[174]

Lipoproteins	Protein %	Major component
Chilomicron	2.3	Ingested triglycerides
VLDL	15.0	Triglycerides
LDL	30.0	Cholesterol
HDL	40-50	Phospholipids

16 **CHOLESTEROL.** Normal humans have cholesterol of about 2g/kg body weight. The daily total loss and replacement is 2 percent of the body pool. The sources of cholesterol are:-

a. **Exogenous.** from food. from acetate. by the liver and the intestines, particularly the ileum.

b. **Endogenous** synthesis of cholesterol is partly regulated by the dietary cholesterol intake. Even on a cholesterol free diet. cholesterol from the body needs will be synthesized by the liver and the intestines. Liver synthesis is reduced with high cholesterol diet, fasting

and increased bile flow. Despite a high cholesterol diet, a major part of serum cholesterol is derived from endogenous synthesis mainly from carbohydrate. The plasma cholesterol level in both men and women remains about the same until the age of 20, and then increases with age to a greater extent in males. A single plasma cholesterol estimation is not conclusive as even with ordinary activity and diet, the level shows significant fluctuations. The cholesterol in atherosclerotic arteries is in a dynamic state and exchanges with that in the blood. Cholesterol is essential to life for the formation of cell membrane. Thus a constant cholesterol supply by endogenous synthesis is essential. A high plasma cholesterol may occur with increased risk of heart disease but not necessarily so. The normal serum values is, suspect > 5.7 mol/L, >220 mg/dl, elevated >6.7, mmol/l, > 260 mg/dl.

17. **HIGH DENSITY LIPOPROTEIN (HDL).** Approximately 20% of the total plasma cholesterol is found in HDL. HDL is presumed to aid esterification of cholesterol. It also scavenges free cholesterol released from the aging cells and then aids cholesterol excretion and decreases atherosclerosis. Thus higher HDL has a beneficial effect in the prevention of CHD HDL cholesterol is higher in those

engaging in physical activity and exercise and in marathon runners and lower with adiposity, cigarette smoking, on eating sugar or starch. The normal values of HDL-cholesterol in health is equal to >1.42 mmol/L, >55 mg/dl.

18. **LOW DENSITY LIPOPROTEIN (LDL).** LDL rises with age. Increase in dietary fat enhances synthesis of LDL whereas in familial hypercholesterolemia there is a genetic defect that prevents breakdown of LDL. LDL transports most of the plasma cholesterol and is therefore, found to be raised with high cholesterol and atherosclerosis. Decreased level of LDL is associated with low incidence of CHD but a higher incidence of stroke. Cholesterol lowering diets apparently reduce HDL and LDL in equal proportion. The normal values of LDL-cholesterol in health is equal to <3.90 mmol/L, <150 mg/dl.

19. **ß LIPOPROTEIN** : These are produced by the further removal of triglyceride from IDL, probably by the action of hepatic triglyceride lipase. In addition to the removal of Triglycerides, the conversion of VLDL to LDL is accompanied by other changes affecting the lipid and apoprotein composition of the particle. LDL or ß lipoprotein are particularly rich in cholesterol which an average forms 40-50% of their weight, with ester cholesterol about three-quarters of this. They are also rich in phospholipids. The normal values : 360-640 mg/dl.

20. **TRIGLYCERIDES (TG).** Chemically, fats and fatty acid esters of alcohol and glycerol. Glycerol has 3 carbon atoms and when 3 fatty acids are attached to it, TG is formed. Sources of TG are as under:-

a. **Exogenous** - derived from ingestion of fatty acids. The absorbed TG raise the plasma level for 3-4 hours after food intake and circulate as chylomicrons.

b. **Endogeneous** - are produced mainly by the liver from the free fatty acids, carbohydrates. If level rises, a few days after ingestion of carbohydrate, sugars stimulate insulin secretion, which enhance TG formation.

TG combining in the liver with phospholipid, cholesterol and protein form VLDL. After an overnight fast, TG are mainly found in the VLDL. Fasting TG level is upto 250 mg/dl but may show marked fluctuation. Their levels rise with age and that is related to obesity. High plasma TG is found in obesity, excessive alcohol intake, antihypertensive drugs such as diuretics, beta blockers etc. Normal TG levels in serum is 0.2 - 2.15 mmol/L, 110-190 mg/dl.

21. **BLOOD LIPIDS.** The plasma lipids are present as complexes with protein molecules, the lipoproteins. The plasma contains 500 mg of

lipids per 100 ml during the post-absorbtive phase. The value tends to rise with increasing age. Lipids are ester like compounds of fatty acids which are insoluble in water but soluble in fat solvents. Since changes in plasma lipid conc. in disease reflect alternations in the conc. of the various types of lipoproteins present,it is most satisfactory to consider lipids and lipoproteins together. The normal lipids level in serum is 4-10 gl, 400-1000 mg/dl.

Average lipid content of blood plasma

Neutral fat (TG)	-	140-225mg/dl
Free cholesterol	-	40-60mg/dl
Cholesterol esters	-	110-190mg/dl
Phospholipids	-	160-200mg/dl

22. BLOOD UREA.It is formed in the body after deamination of amino acids (mainly from exogenous sources). It is formed mainly in the liver. Amount in blood 20-40 mg/dl. Almost equally distributed in plasma and corpuscles. Urea formation- helps to maintain the reaction of blood constant. Urea is the main end product of protein catabolism. Normal values of serum urea - 2.5 - 8.3 mmol/L, 15-50 mg/dl.

23. **CREATININE.** It is the anhydride of creatine. It is mostly formed from break down of creatine phosphate in the body. This

process is not catalysed by any enzymes and is irreversible. It is filtered by the glomeruli and is also actively secreted by the tubular cells in the urine. Normally it is present about in serum - 53-124 mmol/L, 0.6-1.4 mg/dl.

24. **PLASMA GLUCOSE**. There has been an increasing tendency to use plasma or serum which gives a more accurate measurement of the glucose conc. of extra cellular fluid. The glucose conc. in the water of cells and plasma is the same but the water content of cells, 73%, is less than that of plasma, 93%. Plasma glucose is thus higher than the conc. in whole blood. This may be corrected for by multiplying the whole blood glucose conc. by 1.15 and adding 0.33 mmol/L to derive the plasma or serum glucose. This correction is reasonably accurate if the haematocrit is normal. The normal values of fasting plasma glucose is 3.33 - 6.11 mmol/L, 60-110 mg/dl.

25. **STRESS AND PHYSICAL ACTIVITY:** Various response to stress and physical activity were evaluated by statement, obtained through interviewing the subjects of the study. Other questionnaires evaluated other intervention and therapies, psychological general well being and side effects mainly exercise and relaxation programmes. We applied Jacobson progressive Neuromuscular relaxation technique. This technique produces a temporary trophotrophic state, so as to achieve a state of deep relaxation.

1.17 REVIEW OF LITERATURE

Metabolic therapies have received considerable attention in recent years, in the management of essential hypertension both as definitive intervention and as an adjunct to drug therapy. An integral part of this therapeutic approach is weight reduction, dietary management, exercise and relaxation techniques[91-93]. Unlike therapeutic controlled clinical trials with a double blind design, the effectiveness of Metabolic control of hypertension in lowering blood pressure can hardly be demonstrated directly and with full scientific rigour. Many arguments in favour of the measure described below are based on clinical observations. Some of these are of an anecdotal nature, others used " the patient as his own control" in alternating regimens. Further arguments in favour of these measures are based on pathophysiological considerations and reasoning still others on epidemiological findings. However, unlike drug treatment, the Metabolic or Non drug therapeutic measures have no demonstrable harmful effects, except it grossly exaggerated. This is definitely an argument in favour of the Metabolic control of hypertension.

Just as increased awareness of the problem of patient's frequent poor adherence in drug therapy has led to attempts to improve the situation[94-97]. Similar attention toward adherence to Non drug therapies will probably improve their effectiveness. These measures should be introduced gradually and gently. Too many and too drastic

changes in life style may discourage patients from accepting needed care. Eventually, however, all hypertensive patients should benefit from mild restriction of dietary salt, reduction of excess body weight, and moderation of alcohol intake[95,100]. Since the Non-drug therapy useful in hypertensive persons are not costly and are generally beneficial in promoting good health, their gradual introduction should be attempted in all hypertensive patients. Although permanent modifications in diet and life style are difficult to achieve, in motivated patients[17].

The rising enthusiasm for the use of drugs to treat hypertension has not spread to the use of metabolic control, because of diverse interest and very difficult to follow.

It may be noted here that patients enrolled in the Australian trial whose Diastolic blood pressure was between 105 and 109 mmHg after two sets of readings, four weeks apart 11% of those given orally placebo pills had diastolic blood pressure persistently below 90 mmHg for the next four years[99]. There seems no way to provide such evidence on the efficacy of Non-drug therapies, which are less potent, and more difficult to monitor, for the prevention of complications of mild hypertension. For that reason, the use of Metabolic Control must be accepted on the evidence that they work to lower the blood pressure, are safe and are acceptable to many patients.

The following review provides a critical look at the efficacy of Non-drug therapies. Unfortunately, many of the studies reviewed did not provide a suitable run in period to get around the initial non-specific falls in pressure. Several other methodologic problems were common in literatures. Few studies randomly assigned unselected patients to either active or inactive therapies. Few were double or even single blinded. Some compared different numbers of patients with different proportions taking antihypertensive drugs along with the Non drug therapy.

This review focuses mainly on the most recent literature and on the methodologically strongest data. Some therapies, such as relaxation therapy, are considered briefly because recent reviews are not available. For other therapies, such as exercise, as far as entire literature is surveyed because critical reviews are not now available.

1. **Weight reduction**: Body weight and blood pressure have been correlated consistently in several population and clinical studies, but only recently has it been shown clearly that short-term weight loss substantially reduces blood pressure independently of dietary sodium intake[72]. Successful weight reduction is therefore, as effective, cheap, and safe treatment[102]. Forty nine hypertensive patients who were overweight were randomly allocated to one of three strategies for attaining weight reduction and were followed

for one year. Successful weight loss was associated with a highly significant and substantial improvement in blood pressure control and with less frequent increases in antihypertensive treatment[102].

The first identification of blood pressure reduction as a result of weight reduction was published 70 years ago and this has been confirmed subsequently in numerous reports. Out of 16 controlled studies on lowering blood pressure through weight reduction, which were selected for their appropriate methodology and reviewed by the

Table 1-5. Effects of weight loss on Hypertension

Author, year (Reference)	Random Selection	Patients (N)	Diet Cal/d	Duration	Na intake mmol/d	Anti-Hyper drugs	Weight loss	Blood Pressure	
								Initial	Change
Reisin.et al[180] 1978	Yes	24	800-1200	4 mons	165	No	-8.8	157/106	-26/20
Stamler.et al[181] 1980	No	67	Unknown	5 yrs	Unknown	No	-4.5	147/96	-12/9
Fagerberg.et al[111] 1984	Yes	15	1230	9-12 wks	195	No	-8.7	146/98	-7/7
Maxwell.et al[106] 1984	Yes	18	320	12 wks	40	No	-28	152/100	-30/21

world hypertension league. 15 were successful to varying degrees. However, there is no universal agreement on this issue, because of design flaws. Nevertheless, the evidence in favour of the benefits of reducing overweight is by far outweighing the doubts. Weight

control is considered the main stay of metabolic control of hypertension in most patients[69]. This is supported also by the report of the US Joint National Committee[103].

2. **DIETARY SODIUM RESTRICTION.** Recent published studies of dietary sodium restriction in hypertensive patients found only a small reduction in blood pressure and concluded that there is little evidence that reduction of sodium intake has a beneficial effect on blood pressure control[119]. Further, blood pressure increases have been observed in some hypertensive patients when dietary sodium in take is reduced.Eighteen patients with stable mild hypertension (mean blood pressure 144/93 mmHg) restricted their sodium intake for eight weeks while taking part in a double blind randomized cross over or trial of slow sodium and placebo tablets. There was no significant difference in blood pressure. Moderate dietary sodium restriction does not lower blood pressure in patient with this degree of hypertension[120]. Controlled trials in moderately severe essential hypertension have shown a small but worthwhile reduction of arterial pressure by modest, realistic sodium restriction and also by potassium loading[118].

Table 1-6 : **Studies of moderate sodium restriction in mild hypertension**

Author.year (Reference)	Patients	Pre-therapy BP Monitoring	Design	Urinary Na		Initial BP	Change in BP	
				Low Na Diet	High Na Diet			
Mac Gregor.et al[182] 1982	19	2 months	Random crossover blind (4-wk periods)	86	162	156/98	-12/6	-2/1
Watt.et al[183] 1983	13	2 wks	-do-	59	139	150/91	-10/5	-10/5
Silmen.et al[119] 1983 Diet Control	12 16	1 months	Parallel (52 wks)	117	159	165/98 160/98	-29/18	-20/11

The observed heterogeneity in blood pressure response to dietary sodium restriction has given rise to attempts to classify hypertensive patients as salt sensitive or salt resistant and to develop bio-chemical indices of salt sensitivity.

3. **PHYSICAL EXERCISE.** Regular exercise may have a beneficial effect in subjects with mild hypertension. Persons who are physically active and fit may develop less hypertension[123,124] and those who are hypertensive can lower their blood pressure by regular isotonic exercise[125-129]. Data from the Framingham study showed that persons who are more physically active have a lower risk of developing or dying from coronary heart disease[109]. Furthermore, persons who had objective evidence of poor physical fitness, such as more obesity, faster heart rate, and lower vital

capacity. had a higher risk of coronary heart disease mortality. Ten men with uncomplicated essential hypertension and ten normal controls matched for age and weight were studied for the hypertensive potential of moderate exercise. After the tests both the controlled hypertensive groups showed a significant and sustained fall in systolic blood pressure. If a fall in blood pressure after exercise is maintained, then a good walk twice a day might be reasonable treatment for mild hypertension[130].

Table 1-7. Effects of Isotonic Exercise training on selected hypertensive patients

Author,year (Reference)	Patients (n)	Pre-exercise BP monitoring	Anti-hyper drugs	Exercise programme	Duration	Resting BP		Weight change
						Initial	change	
Hagberg,et al129 1983	9	6 months	No	0.5x5	6	141/91	-8/11	None
Roman,et al184 1981	27	None	No	0.573	3-24	182/113	-20/18	None
Krotkiewski et al187 1979	27	None	No	1x3	6	134/67	-9/7	None

Despite widespread interest in Non-drug therapy for hypertension, physical exercise had not been accepted as an effective antihypertensive therapy[130-136]. There have been no well-controlled studies of factor such as age, sex and race and the related mechanisms of antihypertensive effects of exercise remain unclear[137]. From a study the depressor effects of exercise and to analyse changes in hemodynamics was noted[138]. Twenty Japanese

subjects with essential hypertension were clinically observed for 4 weeks, then randomly divided in two groups and agreed to physical training using cycle ergometer exercise. The second group did no particular physical training. Changes in blood pressure, hemodynamics and humoral factors etc. were compared, blood pressure and whole blood and plasma volume indices were significantly reduced in the exercise group compared with controls.

4. DIETARY CHANGES. Some controlled studies have shown that the blood pressure may respond to alternations in the type of macro nutrients consumed. One study examined the effect of a decreased intake of total fat along with an increased intake of poly unsaturated fat[1]. Fifty seven couples were randomly allocated to three different diets for 6 week intervals: one group combined their usual diet, the second reduced their daily sodium intake from 192 to 77 mmol, the third reduced daily total fat intake from 108 to 529 and increased the poly unsaturated to saturated fat ratio from 0.27 to 0.98. In each group, about half were hypertensive. Little changes was seen in body weight or potassium excretion. The systolic and diastolic pressures fell significantly in both normotensive and hypertensive persons on the low-saturated fat diet, whereas only the diastolic pressures fell in the other two hypertensive groups on the control and low-sodium diets.

A few other studies in humans and several in hypertensive animals have shown as antihypertensive effect of increased intake of polyunsaturated fatty acids such as linoleic acid[139,140]. The effect has been assumed to be mediated by an increased synthesis of vasodilatory prostaglandins, but hypertensive rates had a fall in pressure not mediated by changes in prostaglandin synthesis[141].

Dietary manipulation, such as changing to a vegetarian diet, increasing total fibre intake seems to be associated with a lowered blood pressure when a group of 59 healthy, omnivorous persons were randomly assigned to a control group, whose diet for 06 weeks and then crossed over to the other diet for another 6 weeks, falls of 5 to 6 mmHg in systolic pressure and 2 to 3 mmHg in diastolic pressure were seen with vegetarian diet[142].How a vegetarian and diet lowers the blood pressure is unknown. Possibilities include its increased content of polyunsaturated fat, fibre, vegetable protein, potassium and magnesium[143].

5. POTASSIUM SUPPLEMENTATION

Both experimentally and clinically, many of the observed benefits of reduced sodium intake may reflect an increased potassium intake, because whenever sodium is deleted from the diet, particularly by the substitution of natural foods for processed products, potassium intake increases. It has been showed that the high ratio of sodium

to potassium in the diet was important factor in the development of hypertension and the reversal of this ratio back to that consumed by more primitive man would both prevent and relieve hypertension. Some evidence supports this view . Blood pressure was inversely correlated with dietary intake and urinary excretion of potassium in[64], a meticulous analysis of body electrolytes in 91 hypertensive patients found an inverse correlation between plasma exchangeable and total body potassium and blood pressure[65] and the lower blood pressure of vegetarians has been ascribed to their large intake of potassium[68].

Epidemiological studies have demonstrated an inverse relationship between dietary potassium intake and blood pressure[144-147] and several recent controlled studies have demonstrated a small but significant reduction in blood pressure with dietary potassium supplementation[148,154].

Table 1-8. Status of Potassium supplementation in mild Hypertension

Author.year (Reference)	Patient (n)	Pre-therapy BP montioring	Design	Urinary Excretion of K		Blood Pressure	
				Control	Added Kcl	Initial	Change
Mac Gregor.et al150 1982	23	2 months	Random, cross over, double blind	68	118	154/99	-6/4 and +1/0 (Placebo)
Smith.et al151 1983	10	1 wk	Open, 12 days of 96 mmol of Kcl	65	128	156/93	-8/2
Richards.et al117 1984	12	>10d	Random cross over 4-6 wks. each of control, low Na or extra k	60	200	150/92	-2/1

6. CALCIUM SUPPLEMENTATION. Epidemiologic studies have suggested an inverse relationship between dietary calcium intake and blood pressure. The increased peripheral vascular tone of essential hypertension may be the consequence of increased intracellular calcium[155,156]. Calcium is associated with hypertension and that oral calcium supplements may lower, not raise, the blood pressure[157]. A fall, in blood pressure was noted after supplementation of the diet with 1g/d of elemental calcium, both in normal pregnant women and normal young adult[158]. Many authors found that calcium intake, assessed by a single 24-hour diet recall, was 22%, less in 46 hypertensives than in 44 age, race and sex-matched normotensive.

Subsequently, preliminary data on the effects of supplemental calcium in patients with essential hypertension have been presented[159-162]. Investigators gave 15 patients 2g/d of calcium carbonate for 5 months and some gave 32 hypertensive either 1g/d of elemental oral calcium or placebo for 8 weeks, each in a randomized double-blind cross over trial. In both studies, only about half the patients had a significant fall in blood pressure.

7. **RELAXATION/STRESS REDUCTION.** The relaxation and stress management produce only modest blood pressure lowering even in highly motivated patients. It may be noted that some of the antihypertensive effects of exercise could reflect the more relaxed

Table 1-9. Effects of Relaxation therapy on Hypertensive patients

Author, year (Reference)	Patients (N)	Antihyper therapy	Relaxation Programme	Duration	Resting BP	
					Initial	Change
Peled-Ney et al169 1984	30 40	Yes Yes	None Group therapy	6 months 9 months	147/98 152/100	-1/+1 -21/13 -16/11
Engel,et al166 1983	60	Yes	Relaxation and/or biofed back	12 months		-5/7
Lubrosky,et al170 1982	7 15 9 8	Yes No No No	None Relaxation Biofed back Exercise	6 months	142/88 140/87 137/87 143/94	-8/3 -4/1 +2.1 -5/3

feeling that may accompany physical activity. On the other hand, purposeful muscular relaxation has long been noted to lower the

blood pressure in many hypertensives[163,164]. In recent years, considerable evidence from studies in both animals and humans has implicated behavioural stress in the pathogenesis of essential hypertension, often in uncut with a high sodium intake[136]. A long with this evidence, an increasing number of studies, a few well controlled, have shown that reduction of stress by various techniques will reduce the blood pressure, both during the relaxation and for prolonged periods thereafter[137-142]. However, a small number of similarly well controlled studies have failed to show a significant effect in most patients[143-145]. Some reviews of the effectiveness of relaxation therapy that were available before 1980 were optimistic but uniformly described the lack of carefully controlled studies and long-term follow up. The studies shown some additional evidence of a statistically significant fall in blood pressure[165-170], greater than that seen in other patients randomly assigned and followed in parallel without relaxation.

The study by Patel and colleagues[164] remains the best designed: It randomly assigned a group of newly recognized, untreated hypertensive patients to either no therapy or a behavioural modification programme involving 1-hour group by bio-feedback sessions once a week for 8 weeks and twice daily relaxation at home. After the active phase, the treated patients were asked to continue to practice relaxation twice a day, but they were not seen

again until 6 months later. The differences in both systolic and diastolic blood pressure were highly significant both at 8 weeks and 6 months later. In addition patients followed by others[171,173] for at least 3 to 6 months, generally showed continued lowering of blood pressure. These patients were also randomly assigned to relaxation therapy, so these studies to be methodologically strong.

CHAPTER-II

STUDY DESIGN AND METHODOLOGY

2.1 DESIGN OF THE STUDY

A total of 56 subjects: 28 hypertensive and another 28 normotensive control were given metabolic therapy after admitting them into the hospital for three weeks. In this study a true experimental design that is a pretest and post test control group design was adopted with common source populations, eligibility criteria and data collection and analytic methods. The whole study was carried out over a period of eighteen months. Four weeks screening period followed by controlled clinical trials with an unmasked design but with blinded measurement of blood pressure as the end point. Three blood pressure measurements with the use of Mercury sphygmomanometer in sitting posture as baseline and after three weeks the same recording of blood pressure in same condition was obtained as final reading. The subject's baseline, resting and pre-programme and post-programme intervention blood pressure, were the average of many, which were recorded at different times of the day and under various environmental conditions. All other informations were obtained with or without programme intervention as per the objective of the study.

In this study both the experimental (hypertensive) and control (normotensive) group of subjects were received before and after programme measurement as per pertinent selected variables. The follow up period for the life-style change, (weight reduction, salt restriction and stress management) the participants were kept on follow up for a duration of six months, in order to provide a reasonable test of maintenance of blood pressure changes. Since there was a control group of cases, therefore, the threats to validity were substantially reduced. Both the hypertensive and normotensive subjects were given the similar controlled clinical trial with combined metabolic therapies.

2.2 STUDY AREA

This study was conducted at combined Military Hospital Dhaka (CMH), Dhaka Cantonment with proper approval from the authority. The period of subjects selection, programme intervention, data collection and follow up was effective from June 1991 to December 1992. The Medical outpatient department (Med OPD) of this Hospital was the screening place for the selection of the subjects. This Med OPD remains schedule on Saturday, Monday and Wednesday of the week. From different OPD of the Dhaka cantonment, Mirpur cantonment, Gazipur and Postogola area, patients were routinely referred into this OPD for specialised care. Out of these patients,

only uncomplicated cases were screened and an informed consent was obtained from them for inclusion into the study.
Accordingly after finishing all screening procedures, selected patients were admitted in Medical Ward II and Junior Commission Officer (JCO) Ward. Due to non availability of the beds, sometimes patients were also admitted in coronary care unit (CCU).

CMH Dhaka is a largest and most modern military hospital of this country. It is situated towards north of Dhaka city and about 15 km distance from Institute of Nutrition and Food Science, Dhaka University. It is about half km towards west of Dhaka-Tongi Road which passes through the Dhaka Cantonment. CMH Dhaka since its birth as a 50-bedded hospital in the year 1947 has grown today to 700 bedded with the passage of time. It provides Hospital treatment (accn, food, clothing, surgical, medical, dental treatment and other amenities to the entitled persons that is Armed Forces (Army, Navy and Air Force) personnel and their families, retired service personnel, civilians paid from defence estimate and any other person authorised by the govt. It also imparts training in various fields of speciality. This Hospital is capable of extending the following facilities to the entitled persons.

1. Can hold and treat 700 patient at a time.
2. Provide all types of dental treatment including provision of artificial denture.

3. It acts as a referral and Teaching Hospital.
4. It provides specialised care in the field of neurology, neurosurgery, nephrology, urology, cardiology, cardiothoracic surgery, orthopaedic surgery.
5. Renders treatment and rehabilitates war casualties.
6. Provides outpatient treatment to entitled persons.
7. Arranges proplylactic to entitled persons and medical inspection of the troops.
8. Hold medical board for the candidates of Bangladesh Military Academy, enrolment of civilian gazetted officers in the defence services, invalidation and categorisation, resurvey and commutation of pension.
9. Arrangement of medical examination of service pesonnel as and when required, technical training of selected trades.
10. Carry out research on military medicine.

This Hospital has got the following wings and department, for example administrative wing, Family and OPD wing, casualty department, statistical section, surgical wing, intensive care unit, dental wing, cardiovascular and thoracic centre, nephrology and urology centre, neurology centre, medical wing, C.C.U, orthopaedic centre. The present study was carried out under the supervision of medical wing and the selected subjects were admitted in various medical wards including C.C.U. Exercise and

relaxation programmes were performed in the physiotherapy department.

It may be mentioned that station health organisation (SHO) Dhaka an independent health unit which is located within the premises of the CMH Dhaka was responsible to provide all necessary administrative support to implement this research project. Investigator himself has been serving here as an Officer commanding of this Unit. SHO as a preventive medicine unit its role is to promote, health, prevent and control diseases, eradicate mosquitoes and flies, investigate epidemics, carrying out disinfection and supervise general sanitation in a cantonment.

Study on metabolic control of hypertension by dietary changes, exercise, and stress management or in other wards through hygienic and nutritioal means is a relevant research project as per the objectives and principles of the SHO Dhaka Cantonment.

2.3 STUDY POPULATION

In this study, subjects with uncomplicated primary essential hypertension were the study population. Different categories of Army personnel, for example Junior Commission Officer (JCO), Non-commission officer (NCO), Sepoy, Recruits were included in the

study. The members from the Navy and Air Force, all retired service personnel and civilians paid from defense estimate except officers were also included in this project. Socioeconomic status was in order of rich, upper middle, lower middle and poor class that is Class I, Class II, Class III and Class IV employees respectively. The subjects were from Dhaka Garrison, Mirpur, Gazipur and Postogola Cantonment areas who are being normally referred at Med OPD of CMH Dhaka as a case of observation for hypertension were selected. Following four weeks screening period and at four consecutive screening visits at an interval of seven days, all the subjects had a diastolic blood pressure without taking anti hypertensive drugs within 95-110 mmHg. All subjects gave informed consent, following procedures approved by institutional review boards (specimen attached as Appendix-C).

INCLUSION CRITERIA:

2.4 **SELECTION OF SUBJECTS:** IN TO THE EXPERIMENTAL GROUP

1. Age 30-59 years and Sex - only male subjects.
2. Who were not currently taking antihypertensive drugs for the prior 1-2 months.
3. Diastolic blood pressure less than equal to 110 mmHg.
4. Persistent elevation of blood pressure for four weeks following detection of raised blood pressure. At least three blood pressure readings showed persistent elevation that is taken on each of at least two different occasions.

5. Subjects willingness to participate and agree to stay for three weeks in the hospital.
6. Must agree to receive a low-sodium diet, and perform exercises and relaxation and meditation therapies.
7. Entitled to get inpatient facilities in Military Hospital.
8. Must resides within 10 miles of the study area for easy follow up.
9. **For control:** a group of normotensive hospitalized subjects who had diastolic blood pressure, less than equal to 90 mmHg. Sex male, matched for age that is not more than or less than 5 years within the age group of 30-59 years. All others inclusion criteria are similar as per serial No. 1,2,5,6,7, 8.

2.5 EXCLUSION CRITERIA

1. Who were currently taking antihypertensive medication.
2. Subjects with diastolic blood pressure greater than 110 mmHg and age above 59 years.
3. Patients with damaged target organs or isolated systolic hypertension.
4. Patients with secondary hypertension and diabetes mellitus.
5. Subjects with peripheral vascular diseases, orthopedic problems.

6. Subjects were also excluded if they had evidence of unwillingness or inability to comply with intervention.
7. For control subjects, the exclusion criteria are similar as per serial no. 1,2,3,4,5 & 6 respectively.

2.6 TRAINING OF MEDICAL ASSISTANTS AND OTHER PERSONNEL

Qualified medical assistants and trained Dietician and physiotherapist were selected for the project. Medical Assistants having qualified in Class I level (Army Medical Corps training manual) and not below the rank of Naik were trained adequately for one month for collecting personal data, clinical data, data on blood pressure measurement during clincal trial as well as during follow up period. Exhaustive training was given for reproducibility of ausculatory method of blood pressure measurements mainly in sitting posture with the use of mercury sphygmomenometer. Besides, training on relaxation therapies were properly organized to train the physiotherapist as selected for the study. All personnel selected for the study were also trained on how to fill-up questionnaire forms. Sometimes periodical evaluation was carried out to refresh their memory and also to increase their ability. For reproducibility of blood pressure measurement 1-2% observed variation was recorded and accepted for the study.

2.7 BASELINE DATA COLLECTION

Clinical data was obtained regarding persistent elevation of high blood pressure. The three main objectives of clinical examination and baseline data collection in a hypertensive patient are to identify any underlying causes, to recognise risk factors for the development of complication and to detect any complications already present. Clinical history was also recorded regarding urine analysis for protein, serum urea/ creatinine to assess renal function, serum electrolytes, lipid profiles, chest x-ray, ECG. Socio-economic data and dietary information and nature of physical activity were collected. Physical examination was thoroughly done. The initial evaluation was included patient's baseline arterial blood pressure, assessment of degree of end- organ damage. A careful examinatioin of cardiovascular system for evidence of other vascular complications were also performed. Finally all baseline and after programme intervention, data on heart rate, blood pressure, weight etc. were recorded by a trained medical assistants and physiotherapist.

2.8 SAMPLE SIZE

The sample size and selection of sample in respect of experimental and control subjects was as under:-

As already been stated that this study was carried out in a military Hospital of Dhaka cantonment to ascertain whether

Metabolic therapy would reduce both diastolic and systolic blood pressure. All referred patients attending the Med OPD of this Hospital were screened and selected and finally admitted them for receiving controlled programme intervention. The diagnosis of uncomplicated essential hypertension was made beforehand for selecting the experimental subjects. For the control, another group of normotensive (blood pressures≤ 90 mmHg) patients from this Hospital was also selected and admitted to receive similar type of controlled programme i.e intervention. Both the experimental and the control subjects were comparable and matched for age.

The statistical formula as given as under was helpful to us to decide how many experimental and control subjects should be enrolled in each group.

1. **The size of the difference between mean diastolic blood pressure had to detect.**

After much consideration it was decided that a difference of 10 mmHg diastolic blood pressure was an appreciable effect that the study would not like to miss. We, therefore, need to apply the formula with $\mu 1 - \mu 2 = 10$ mmHg

where $\mu 1$= mean reduction of diastolic blood pressure in experimental group (hypertensive).

$\mu 2$ = mean reduction of diastolic blood pressure in normotensive that is control group.

Let $\mu 2$ = 10 mmHg

Therefore, the mean difference= 20-10 = 10 mmHg.

2. **The standard deviation of the distributions of diastolic blood pressure in each group**

It was decided to assume that the standard deviation of the diastolic blood pressure would be the same in the two groups.The past data suggested that it would be about 9 mmHg. In other words it was decided to assume that d_1 = 9 mmHg and d_2 = 9 mmHg.

3. **The power required 95% was agreed on,**

This study therefore, need u = one sided percentage point of the normal distribution corresponding to 100% minus the power, for example if power = 95% (100% - power = 5%) and u = 1.64.

4. **The significant level required**

It was decided that if possible we would like to achieve a result significant at the 1% level. Therefore, v = 2.58, where v = percentage of normal distribution corresponding to the required

(two sided) significant level.

Applying the formula

$$n = \frac{(u+v)^2 \left(d_1^2 + d_2^2 \right)}{(\mu 1 - \mu 2)^2}$$

$$= \frac{(1.64 + 2.58)^2 (9^2 + 9^2)}{10^2}$$

$$= 28.80$$

In view of the above, in order to satisfy the requirement, this study would need to select and admit about 28 hypertensive subjects as experimental in one group and 28 normotensive subjects in another group as control.

2.9 INITIAL EVALUATION

The initial evaluation of the hypertensive patient was to determine the baseline arterial blood pressure following four weeks screening programme at Med OPD. The degree of end organ damages, screening for secondary causes of hypertension, identification of other cardiovascular risk factors were assessed. The characteristics of the patient as per age, sex, life style, concomitant illnesses to facilitate choice of therapy, intervention etc. were also evaluated. Whenhypertension has been confirmed, the study was proceeded as follows:

1. Information to the patient
2. Assessment of severity of the existing hypertension.
3. Assessment of the patients overall cardiovascular risk.
4. Search for possible causes of the hypertension
5. Carry out an extended examination to define and document the causes.

2.10 BLOOD PRESSURE RECORDING

1. POSTURE

a. Prior to the recording of blood pressure the patient was in a relaxed mood in a quiet room.

b. Patient was sitted quietly with back supported for 5 minutes and arm supported at level of heart.

2. CIRCUMSTANCES

a. No smoking or caffine for preceeding hours

b. No exogenous adrenergic stimulants

c. A quiet, warm setting

d. Home readings were taken under varying circumstances.

3. EQUIPMENT

a. Cuff size: The bladder was fully encircled and covered two thirds of the length of the arm.

b. Manometer: Mercury sphygmomanometer was used.

c. The cuff less than 12.5 cm wide was not used.

4. **TECHNIQUE**

Number of readings

a. On each occasion, at least two readings were taken and was prepared by as much as time as was practical. Additional readings were also taken, if the readings vary by more than 5 mmHg.

b. Final diagnosis was made on the basis of 3 sets of readings on different occasions.

c. Initially pressure was measured on both arms. If the pressure differed then, arm with higher pressure was selected.

d. The bachial artery was palpated above the antecubital fossa and then stethoscope applied over it.

e. Diaphargm should be firmly placed with as little a pressure as possible keeping no space between it and the skin.

f. Stethoscope does not touch the clothing or the cuff.

5. **Performance**

a. The bladder was inflated quickly to a pressure 30 mmHg above the systolic pressure, as recognised by the disappearance of the radial pulse.

b. The bladder was deflated 2 mmHg every second.

c. The Korotkoff phase v (disappearance) was recorded as the level of diastolic pressure.

d. The abrupt sharp sound (tapping sound) was recorded to be systolic blood pressure.

2.11 MEDICAL HISTORY (Form attached as appendix J)

A careful, complete history was obtained and a physical examination performed before the intervention was started. The medical history included any previous history of hypertension, including prior and current antihypertensive drug treatment, a history of factor, regarded as predisposing to hypertension, including excessive salt intake, the use of certain drugs that are known to elevate blood pressure, stress, family history of hypertension and its complication, for example, congestive heart failure, coronary artery disease, renal dysfunction and stroke. A history of other cardiovascular risk factors, including diabetes, obesity, cigarette smoking and lipid abnormalities was recorded.

All current medications were assessed and stopped accordingly as per the objective of the study. History taking was also assessed, the patient's understanding of his illness and willingness to participate in experimentation.

2.12 PHYSICAL EXAMINATION

The physical examination began with the patient's general appearance. The next step was to compare the blood pressure and pulses in both upper extremities and in the supine and standing positions. A rise in diastolic pressure when the patients goes from the supine to the standing position was most compatible with essential hypertension. The detail examination of the ocular fundi was carried out. The specific changes in each fundus was recorded. Palpation and ausculation of the carotid arteries were also carried out. While examining the heart and lungs, investigator looked for evidence of left venticular hypertrophy and cardiac decompensation. The examination of the abdomen for bruits; auscultation over all areas were done. The abdomen was also palpated for enlarged kidneys. Finally, examination of the extremities for edema and a search for evidence of previous cerebrovascular accident were also carried out.

2.13 WEIGHT AND HEIGHT MEASUREMENT

All subjects were weighed on a automatic weight machine. Weight was taken without shoes and in minimum clothings and was recorded to the nearest kg. The weights are marked in kg around the dial. Height was measured with Scale's rule. Moreover, the height was taken in a standard erect position without foot wear and recorded to the nearest half-centimeter.

2.14 LABORATORY EXAMINATION: A sample of 5ml blood was drawn aseptically from the antecubital vein for examining the various blood parameters.

1. Blood for complete picture i.e. total count of RBC, WBC, Hb% and ESR. The anaemia associated with renal disease or other forms of anaemia may be detected.
2. Urine for routine examination for albumin and sugar.
3. Electrocardiogram. Resting ECG was performed. ECG was carried out to detect signs of cardiac involvement (left venticular hypertropy, ischaemia infarction) or of cardiac arrhythmias. As per selection criteria we did not include anybody with abnormal ECG finding, stress test was not planned.
4. Serum electrolytes. Serum sodium conc is high in hyperaldosteronism, and it was simultaneously determined with serum K^{+}. Hypokalaemia or Hyperkalaemia may be a feature of pathogenesis.
5. Chest X-ray Information was obtained on cardiac size and contour, on the size and density of the aorta and on the presence of left ventricular failure.
6. Serum cholesterol. This was an important indicator of additional cardiovascular risk associated with hypertension.
7. Serum TG and HDL – Cholesterol Conc.

A plasma lipid profiles was obtained by measuring the fasting levels of TG and HDL-cholesterol, LDL - cholesterol, β-lipoprotein in addition to total cholesterol for predicting cardiovascular disease better than measurement of total cholesterol alone. Elevated TG levels was added a further degree of risk to an elevated plasma cholesterol. The risk of CHD is inversely proportional to the serum level of HDL - Cholesterol.

8. **Serum Urea.** Even though a high level is not as much, a significant CHD risk factor, it influence the choice of therapy and may be affected by therapy.

9. **Serum Creatinine.** This give information on renal glomerular function. It is not affected by water or protein intake and is therefore, a more significant index of renal function than a measurement of blood urea.

10. **Fasting plasma glucose.** Its aim is to detect metabolic disorder - i.e. carbohydrate metabolism. Additional cardiovascular and renal risks associated hypertension.

2.15. LABORATORY METHODS/PROCEDURES

1. SODIUM AND POTASSIUM

Method : Flame photometric methods by 614 Na^{+}/K^{+} Analyzar.

Test Principle. Emission flame photometry into the clinical biochemistry is permitting the simple and rapid determination of

[Na^+]. Modern analyzar incorporate an internal standard, often lithium with simultaneous measurement of Na and K and electric amplification of the flame signal. This permits greater dilution of the sample allowing a smaller volume to be used and also reduces the effect of protein on the viscosity of the diluted sample.

Procedure: A manually operated analyzar 614 Na^+/K^+ analyzar was incorporated an automatic dilator. It was provided with a digital read out of the results when the zero was adjusted by aspirating water and the instrument was calibrated using a single standard. The samples were then aspirated serially into the instrument and diluted by a lithium nitrate solution.

2. **CHLORIDE**

Method: Calorimetric determination of plasma chloride by 925 CL analyzar.

Test Principle. Chloride ions form an undissociated but soluble salt, mercuric chloride, with mercuric ions and this provides a major principle on which Cl^- is determined. In the method of schales and schales (1941), plasma is titrated with mercuric nitrate in acid solution until free mercuric ions can be detected at the end point using diphenylcarbazone.

$Hg(No_3)_2 + 2\ Nacl \text{ -----> } Hg\ Cl_2 = (Undissociated) + 2\ NaNO_3$

The method is modified to provide a colorimetric technique suitable for automated procedures by using an excess of the poorly dissociated mercuric thiocyanate as the primary reagent. In acid solution, this forms mercuric chloride leaving thiocyanate ions in solution which then react with a ferric salt. The colour produced by the red ferric thiocyanate is measured.

$Hg(CNS)_2$ [Undissociated] + 2Cl --->

$Hgcl_2$ [Undissociated] + $2CNS^-$

$3\ CNS^- + Fe^3$ ----> $Fe(CNS)_3$ [red]

In order to avoid too intense a colour and to increase the sensitivity over the required analytical range, it is usual to include mercuric nitrate in the reagent. The Cl^- present converts this entirely to mercuric chloride and only the excess is available for colormetric reaction.

Procedure

Reagents

1. Mercuric nitrate solution. 2. Diphenylcarbazone indicator 3. Standard chloride solution, 10 mmol/L

Technique. 0.2 ml of plasma was added to 1.8 of water, followed by 60 ML of the indicator. The mercuric nitrate solution was titrated by using a microburette calibrated to 0.01 ml. On 2 ml of standard chloride, the titration was repeated, the expected titrant volume was about 2.3 ml. The end point was better with protein-free solutions which gave an intense violet-blue colour on adding the first drop of excess mercuric nitrate. On adding more titrant this becomes pale yellow or colourless until a sharp change to pale violet denotes the end point.

Calculation

$$\text{Plasma chloride (mmol/L)} = \frac{\text{ml titrant needed for unknown}}{\text{ml titrant needed for standard}} \times 100$$

925 Cl^- Analyzar

It covers calibration, operation and shut down procedures in pictorial and multi.

Procedure

1. Analyzar was connected ac supply
2. Investigator waited for zero supply display reading.
3. Sample size was selected and the button pressed.
4. With combined acid buffer it was filled.
5. From step 1, investigator waited for 5 minutes.
6. The beaker was placed on a platform and then raised.
7. 100 mmcl/1 standard was added to beaker (40 µl/200 µl)

8. The condition was depressed and the button pushed.
9. Investigator waited for 000 to be displayed.
10. 100 mmmol cl/1 standard was added to beaker, (20 ml/100 ml)
11. The titrate was depressed and button pushed.
12. Reading was checked.

    100 ml = 100 $\pm$ 2, 20 ml = 100 $\pm$ 4.

3. CHOLESTEROL

Method: Enzymatic determination of cholesterol by cholesterol enzymatique PAP.

Principle: Cholesterol is determined according to the following reaction:

Cholesterol ester (cholesterol estarase)----> cholesterol + fatty acids

Cholesterol (cholesIterol oxidase)-----> cholest -4- en- 3-one + H_2^02

$$2H_2^02 + \text{phenol} + \text{4-amino antipyrine} \xrightarrow{\text{peroxidase}} \text{quinonemine} + 4H_2^02$$

Interpretation. The European atherosclerosis Society has established a relation between the incidence of coronary heart disease and serum cholesterol levels: (From study group, Eruopean Atherosclerosis society, European heart J 1988;9:571-600)

Low risk - <5.2 mmol/L, < 200 mg/dl

Moderate risk - 5.2 - 6.5 mmol/L, 200-250 mg/dl

High risk - > 6.5 mmol/L, >250 mg/dl

Reagents

1 Reagent : (buffer) :	phosphate buffer	0.1 mol/L
	phenol	15 mmol/L
	Sodium cholate	3.74 mmol/L
	Surfactant	
2 Reagent :(enzymes):	4-aminoantipyrine	- 0.5 mmol/L
	Peroxidase	1000 u/l
	Cholesterol oxidase	200 u/l
	Cholesterol esterase	125 u/l

Sample: Serum or plasma collected in heparin or EDTA.

Procedure: Working was prepared by reconstituting the contents of one vial of reagent 2 with the contents of one vial of Reagent 1, using an adoptor. The solution was mixed by inverting and stored it in the reagent 1 bottle.

	Reagent Blank	Standard	Sample
Standard 517 mmol/L	-	10 µl	
Sample	-	-	10 µl
Working solution	1 ml	1 ml	1 ml

- Calculation :

$$\frac{A\ sample}{A\ standard} \times n$$

mmol/L: n = 5.17

mg/100 ml: n = 200

g/l : n = 2

4. HDL-CHOLESTEROL

-Method: Separation of high density lipoproteins and determination of cholesterol bound to these fractions by precipitation.

Principle. The chylomicrons and lipoproteins of VLDL and LDL contained in the sample are precipitated by the addition of phosphotungstic acid in the presence of magnesium ions. The supernatant obtained after centrifugation contains HDL from which the cholesterol can be determined using the cholesterol enzymatique reagents.

Normal values

Men - 1.06 - 1.52 mmol/L, 0.410 - 0.587 g/l

Women - 1.26 - 1.96 mmol/L, 0.486 - 0.750 g/L

Children - 1.34 - 1.86 mmol/L, 0.518 - 0.719 g/L

Reagents

1 Reagent : precipitating agent

Phosphotungstic acid 40 g/l, $Macl_2$ $6H_2^0$ 100 g/l

PH 6.2

2 Reagent : HDL cholesterol calibrating solution

Free + esterified cholesterol - 1.30 mmol/L

Samples : Serum or plasma collected in EDTA.

Procedure: Precipitation by diluting sera containing > 3.5 mmol/L of TG in 9 g/l Nacl.

Serum - 500 µl

Reagent - 50 µl

The solution are mixed and allowed to wait for 10 minutes and centrifuged for 15 minutes at 5,000 rpm

Determination of HDL Cholesterol: The cholesterol enzymatique was reconstituted as specified in the package insert.

	Reagent Blank	Standard	Sample
Distilled water	50 µl	-	-
Reagent 2	-	50 µl	-
Supernatant (Cholestlerol enzymatique)	-	-	50 µL
Working solution	1 ml	1 ml	1 ml

Mixed and incubated for 5 minutes at 37°C and measured

Calculation : $\frac{\text{OD sample}}{\text{OD standard}} \times n$ mmol/L: n = 1.42; g/l : n = 0.55

5. LDL - CHOLESTEROL

Method: Separation of LDL and determination of cholesterol bound to these fractions.

Principle: The various classes of lipoproteins are differentiated according to their density, electrical behaviour and reactivity with specific antibodies. As their differencies in composition cuase widely varying electrostatic and vander waals - type interactions. We studied the influence of amphipathic polymers on lipoproteins isolated by ultracentrifugation. It was observed that addition of certain amphipathic polymers, precipitated certain lipo protein fraction specifically[175]. This was

confirmed on human and animal native sera. A study of the proposed method and the ultracentrifugation method showed good correlation between the levels of cholesterol and phospholipids measured on precipitated fractions and LDL isolated by ultracentrifugation.

Normal values in Serum:

LDL-cholesterol 2.88 mmol/L - 4.87 mmol/L

108 mg/dl - 188 mg/dl

Reagents

1 Reagent : LDL precipitating agent

Polycyclin anionic, surface-active agent	-	0.4 g/l
Polycondensed polycyclic anionic surface-active agent	-	0.8g/l
Poly substituted dioxane	-	12.4 mmol/L
Immidazole buffer PH 6.10	-	25 mmol/L

2 Reagent : LDL solubilizing reagent - trisodium citrate - 0.15 mol/L

Sodium chloride - 0.11 mol/l

3 Reagent: LDL phospholipids calibrating solution choline - 0.5 mmol/l, equivalent to 38.7 mg/dl 0.378 g/l)

Samples: Serum or plasma collected in EDTA.

Procedure

1 ml of reagent 1 was allowed to stand for 5 minutes at 2-8°c Serum ---------- 5 µl and mixed and allowed to stand for 30 minutes at 2-8°c and centrifused for 5 minutes at 4,000 rpm.

Determination of LDL -cholesterol

	Reagent Blank	Standard	Sample
Distilled water	100 µl	-	-
Reagent 3	-	100 µl	-
Cholesterol enzymatique Working solution	1 ml	1 ml	1 ml

Solutions were mixed and incubted for 5 minutes at 37°c & measured.

Calculation : A sample ----> / A standard × n

mmol/l : n = 10
mg/dl : n = 387
gl/l : n = 3.87

6. **β - LIPOPROTEINS**

Method : Colorimetric method, cat. no. 124931 for 25- 50 tests.

Test principle: The β-lipoproteins are precipitated with $cacl_2$/heparin and their core is determined by complexing the cholesterol fraction (30% or average) with sulfuric acid/acetic acid/acetic anhydriole.

- **Normal values in serum** : 360-640 mg/dl, 3.6-6.4 g/l
- **Sample material** : Serum, heparinized plasma.

- **Reagents :**

1 Standard cholesterol glacial acetic acid = 200 mg/dl.

2 Heparin/$cacl_2$ = Heparin-0.0125%, ca cl_2-25 mmol/l Na cl - 3.8 mmol/l

3 Colour reagent, = acetic acid 7.0 mol/l, acetic anhydride - 6.5 mol/l.

Additional reagent : Sulfuric acid, conc.

Sample preparation : Haemolysis interferes with the test, therefore, assay to be performed immediately, precipitation of β-Lipoproteins :

Pipette into 10-ml centrifuse tube	
Sample	0.05 ml
Solution 2	2.00 ml

Solutions are mixed well and allowed to stand for 15 min at 20-25°c, then centrifused for 15 min. Supernatant is poured off carefully and discarded. Then tube stand was inverted to drain on a filter paper for 5 min and any residual supernatant was removed with filter paper. The precipitate was used for cholesterol determination.

Measured against blank. One blank and one standard were sufficient for each assay series.

Pipette into dry test tubes (blank and standard) or on to precipitate in centrifuse tube (sample)			
	blank	standard	sample
Redist water	0.05 ml	-	-
Solution 1	-	0.05 ml	
Solution 3	2.50 ml	2.50 ml	2.50 ml
Mixed and kept blank, standard and sample were stand in a water bath for 5 min at 20-25°c, and added.			
Sulfuric acid	0.50 ml	0.50 ml	.50 ml

It was mixed quickly and left in water bath for a further 10 min. The solutions were poured into dry cuvettes and the absorbances were measursed into dry cuvettes of (A sample) and standard (A standard) against the blank.

If the cholesterol fraction of β lipoprotein exceeds 600 mg/100ml or β-lipoprotein values exceed 1700 mg/100 ml, then 0.2 ml serum was diluted with 0.2 ml of 0.9% Nacl solution and the assay was repeated (result x 2)

Calculation : Of the Conc(c) of β-lipoprotein cholesterol and of the β-lipoproteins in the sample:

Component	Mg/100		ml g/l
Cholesterol C=200X	A sample / A standard	C=2.0 X	A sample / A standard
β -lipoproteins C=570X	A sample / A standard	C=57 X	A sample / A standard

7. **TRIGLYCERIDES**(TG)

Method : Enzymatic determination of triglycerodes by

Enzymatique PAP 150.

Principle : Fully enzymatic determination of triglycerides in serum.

Triglycerides (Lipase) ---> glycerol + fatty acids

Glycerol + ATP (glycerokinase) ----> glycerol-3- phosphate + ADP

Glycerol - 3- phosphate, (glycerol-3 phosphate) ----> dihydxoxyacetone phosphate + H_2O_2 (oxidase)

$2H_2O_2$ + paraclorophenol + 4-amino antipyrine, (peroxidase) ---> quinoneimine + $4H_2^0$

Normal Values

Women : 0.46-1.60 mmol/L

40 - 140 mg/dL

Men : 0.68 -1.88 mmol/L

60-165 mol/100 ml

- **Reagents :**

1 Reagent : Standard - glycerol -2.29 mmol/L equivalent to (200 mg/100 ml -2 g/L) of triglycerides (MW =875)

2 Reagent: Buffer -tris buffer PH 7.6 - 100 mmol/L,

Parachlorophenol ------- 5.4 mmol/L

Magnesium ---------- 4 mmol/L

3 Reagent: Enzymes: amino-antipyrine --- 0.4 mmol\L

lipase ---- ≥ 100,000 U/I

glycerokinase - ≥ 200 U/I

glycerol-3-phosphate oxidase - ≥ 2000 U/I

Peroxidase ≥ 200 U/I, ATP -----0.8 mmol/L

Samples: Serum or plasma collected in heparin, oxalate, citrate, or EDTA. Haemolysis will interfere.

Procedure: Working solution i.e. 1 bottle of reagent 3 was reconstituted with 25 ml of reagent 2 and shaken gently.

	Reagent b	Standard l	Sample a	n	k
Standard (Reagent 1)	-	10 µl	-		
Sample	-	-	10 µl		
Working solution	-	1 ml	1	m	l
Mixed and incubated for 5 minutes at 37°c & then measursed					

Calculation :

$$\frac{\text{A sample}}{\text{A standard}} \times n$$

mmol/L : n =2.29
mg/100 ml : = 200
g/l :n = 2

8. TOTAL LIPIDS

Method : Calorimetric method by sulfophosphovanillin reaction.

Test principle : Lipids react with sulfuric acid and phosphoric acids and vanillin to form a pink colour complex.

Normal values in serum: 400-1000 mg/dl

Sample material: Serum, heparinized plasma.

Reagents : 1 standard total lipids -1000 mg/dL

2 Colour reagent phospheric acid -140 mol/L

vanillin ----- 13 mmol/L

one additional reagent: Sulfuric acid

Sample : Serum can be stored upto 3 days at + 4°c . Conc .A.R.

Procedure : One blank and one standard are sufficient for each assay series.

Pipette in dry test tubes			
	blank	standard	sample
Solution 1	-	0.05 ml	-
Sample	-	-	0.05 ml
Sulfuric acid	-	2.00 ml	2.00 ml

The solutions are mixed well and the tube pluged with cotton wool and allowed to stand in a boiling water bath for 10 min. Then cooled in a cold water bath and again pipetted into dry test tubes.

From above solutions	-	0.10 ml	0.10 ml
Sulfuric acid	0.10 ml	-	-
Solution 2	2.50	2.50 ml	2.50 ml

Thoroughly mixed and allowed to stand at 20-25^{0}c for 30 min. The solutions are poured into dry cuvettes and absorbances of sample (A sample) and standard (A standard) were read against the blank within 30 min.

If the total lipids conc. exceeds 1000 mg/100 ml. 0.1 ml of sample was dilated with 0.9 ml of 0.9% Nacl solution and the assay was repeated (Result X 10)

Calculation : Of the concentration (c) of the total lipids in serum:

$$C = 1000 \cdot X \frac{A\ sample}{A\ standard}\ [\ mg/dl]$$

$$C = 10\ X \frac{A\ sample}{A\ standard}\ [\ g/L]$$

9. UREA

Method : Urea -kits 180, Urea-kits 1000.

Determination of Urea (Urease-modified Berthelot reaction)

Principle: Enzymatic determination of Urea according to the following reaction.

$$Urea + H_2O \xrightarrow{Urease} 2\ NH_2 = + Co_2$$

In an alkaline medium, the ammonium ions react with the salicylates and hypochlorite to form green coloured indophenol (2.2 carboxylindo phenol)

Normal values :

Serum or plasma - 2.5 - 7.5 mmol/L, 15-45 mg/dl

Reagents

1 **Reagent** (standard) - Urea 8.33 mmol/L

2 **Reagent** (enzyme) - Urease 5000 U/L

3 **Reagent** (colour reagent).

Phosphate buffer PH 8 -------- 40 mmol/L

Sodium salicylate --------- 52 mmol/L

Sodium nitroprusside --------- 2.83 mmol/L

EDTA ---------- 1 mmol/L

4 **Reagent** (Alkaline reagent)

Sodium carbonate --- 83 mmol/L

Sodium hypochlorite -- 3.75 mmol/L

Samples: Serum, EDTA, citrated or heparinized plasma. Urine diluted 1:100 in distilled water.

Procedure

Working solution (R2 +R3)

One vial of R2 was added to a bottle of R3 and inverted to mix.

Dilution of R4. The vial of reagent 3 was reconstituted (50 ml) with 200 ml of distilled water (1:5)

	Reagent blank	Standard	Sample
Standard (R1)	-	10 µl	-
Sample	-	-	10 µl
Working solution (R2+R3)	1 ml	1 ml	1 ml

Shaken, incubated for at least 3 min at 37°c for 10 minutes at 20-25°c then measursed

Calculation : $\frac{\text{OD sample}}{\text{OD standard}} \times n$, n = conc of standard.

10. **CREATININE**

Method : Alkaline picrate method with deproteinisation. Reagent kit (SERA-PAK) for the determination of creatinine in serum, plasma and urine.

Test principle : Creatinine in a protein-free alkaline medium reacts with picrate to from red orange compound (Jaffe reaction)

Normal values :

Serum: Males -0.9-1.4 mg/dl, 79-124 mmol/L

Females - 0.8-1.2 mg/dl, 71-106/ mmol/L

Urine: Males - 21-26 mg/kg body weight/24 h

Females 16-22 mg/kg body weight/24 h.

Sample material : Serum or plasma

Reagents

1 Picrate

2 Sodium Hydroxide

Standard : Creatinine 2 mg/dl

Preparation of working solutions :

1 Reagent : Sodium pictrate 44 mmol/L

2 Reagent : Sodium hydroxide 4.o N

One standard and one blank for each series of determinations were prepared. Reagents was at room temp. before use.

Pipette into centrifuge tubes :			
	blank	standard	sample
Trichloroacetic acid	3.00 ml	3.00 ml	3.00 ml
Distilled water	0.50 ml	-	-
Standard	-	0.50 ml	-
Sample	-	-	0.50 ml.

Mixed well and when necessary, the precipitate was dispersed with a glass rod and then centrifuged at 2500 rpm for 10 min.

Pipette in test tube			
Supernatant	2.00 ml	2.00 ml	2.00 ml
Reagent 1	0.30 ml	0.30 ml	0.30 ml
Reagent 2	0.30 ml	0.30 ml	0.30 ml

At room temp solutions were allowed to stand for at least 30 min. The absorbance of the sample (A sample) and standard (A standard) was read against the blank.

Calculation :

$$\text{Serum/Plasma} : \frac{\text{A sample}}{\text{A standard}} \times \begin{matrix} 2 = \text{mg creatinine/dl} \\ 176.8 = \text{mol creatinine/L} \end{matrix}$$

11. **GLUCOSE**

Method : Enzymatic colorimetric method by GOD-PAP (Assay with deproteinization)

Test principle : $\text{Glucose} + O_2 + H_2O \xrightarrow{\text{GOD}} \text{gluconate} + H_2O_2$

$$2H_2O_2 + \text{4- aminophenazone} + \text{phenol} \xrightarrow{\text{POD}}$$

4-(P-benzoquinone-mono-imino) phenozone+$4H_2O$.

Normal values

In blood - 3.89 - 5.55 mmol/L , 70-100 mg/dl

Plasma, serum - 4.22 - 6.11 mmol/L , 74 -110 mg/dl

Sample material

Serum, blood, plasma with fluoride, heparin or EDTA.

Reagents

1 Buffer phenol, 2 reagent strips.

Reagent conc in solutions

tris/phosphate buffer ----- 180 mmol/L, PH 7.8

phenol ---------- 11 mmol/L

3,4 - dichorophenol --------- 2.1 mmol/L

fatty-alcohol-polyglycol ether -- 0.24%

4-aminophenazone ---------- 0.8 mmol/L

peroxidase ---------- $\geq$ 0.9 U/ML

glucose oxidase ---------- $\geq$ 15 U/ml

Additional reagent URAC - deproteinizing solution.

Preparation

One reagent strip was immersed in one bottle of buffer solution and use to stir the bottle contents for 5-10 sec. The buffer solution was allowed to stand in for 5 min, stir once again for 10 sec, and then reagent strip was discarded. Reagent solution from different bottles may be pooled for large series.

Sample preparation

Blood : Immediately deprotenized.

Plasma was separated from cells immediately, if possible, and certainly no later than one hour after collecting the blood specimen.

Pipette in to centrifuge tube :	
URAC :	1.00 ml
Sample :	0.10 ml

Pipette was flushed with the mixture several times. The suspension was centrifused and used 0.10 ml of supernatant for the assay.

The decanted supernatant was stored upto 3 days in a closed vessel at + 4^0c.

Pipette in to test tubes	blank	standard	sample
Redist. water	0.1 ml	-	-
Standard	-	0.1 ml	-
Supernatant	-	-	0.1 ml
Reagent solution	2.0 ml	2.0 ml	2.0 ml

Mixed and incubated at 20-25^0c. Exposure to direct sunlight was avoided. After 30-90 min. absorbances of sample (A sample) and standard (A standard) were read againt the blank.

:ose conc exceeds 800 mg/dl, supernatant 1+2 with 0.9%
on was diluted and the assay repeated (result X 3).

:- of the conc(c) of glucose in blood, serum or plasma:

$$\frac{\text{A sample}}{\text{A standard}} \quad [\text{Mg/dl}]$$

$$\frac{\text{A sample}}{\text{A standard}} \quad [\text{mmol/L}]$$

RVENTIONS - SUBJECTS & METHODS

etion of the screening visits and verification of
, selected subjects get admitted into the hospital for
of three weeks. Various intervention provided by
ɔist, Dietician, and Medical Assistants consisted
f individual training and information followed by
emonstrations and practice. The weight reduction and
iction intervention was achieved by dietary changes by
takes of calories and sodium chloride. The weight
rogramme also encompassed a moderate increase in
enditure, primarily by walking brisk space for 16 to 45
a distance not less than 3 miles on a Treadmill set.
ement involved training and teaching three methods of
(Deep breathing exercise, progressive relaxation
meditation). All the Hospitalised subjects in the

Military hospital had to follow the above regimen strictly as under:

1. **Walking on a treadmill set with progressive increase in distance.**
2. **Progressive relaxation therapy.**
3. **Low sodium diet, less than 2.5g of common salt per day.**
4. **Deep breathing exercise and Meditation.**
5. **Daily calorie intake upto 2000 Kcal.**

1. WALKING ON A TREADMILL SET

This was a brisk and sustained walking programme on a automatic Tread mill machine (Fig 2.1) with gauge. Here is a sample walking programme for someone who is just beginning. This programme was measurable and treadmill machine operated with progressively increase in distance and decrease in time. For easy understanding the programme distances were used in mile instead of kilometre (Table 2.1). Walking was performed on the treadmill set where subjects were allowed to grab the bars as can be seen on figure 2.1.

FIGURE 2.1. Walking on a Treadmill set with progressive increase in distance and decrease in time.

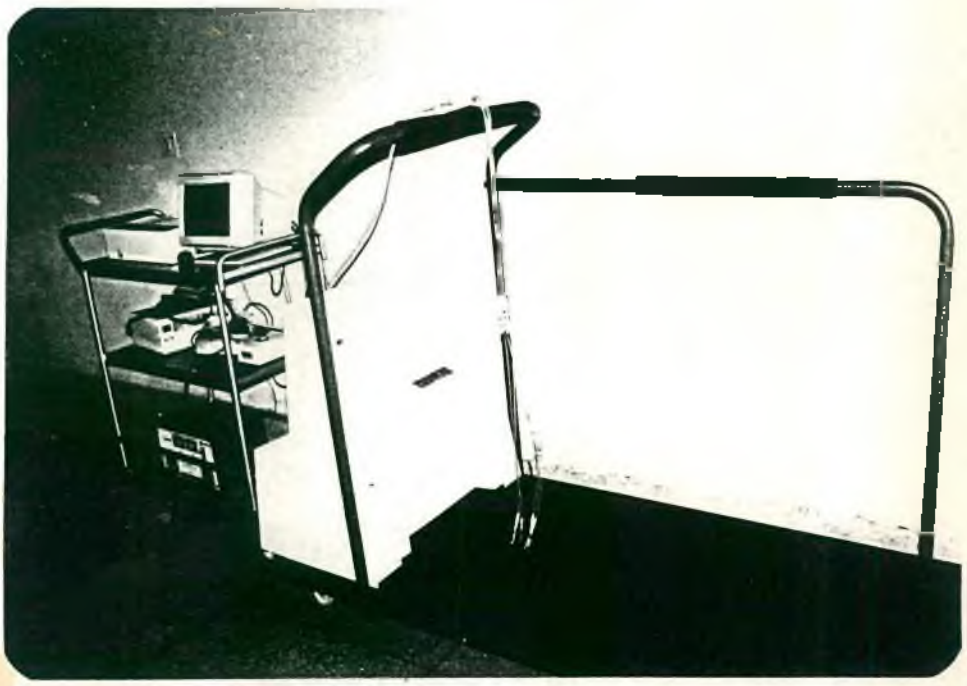


FIGURE 2.2. An automatic Treadmill set (diagnostic) with a E C G Machinine.

Table 2.1 **WALKING PROGRAMME**

Days	Distance (miles)	Time (minute)	Freq/Week (Times)
1	1.0	18:00	6
2	1.0	16:00	6
3	1.5	24:00	6
4	1.5	23:00	6
5	2.0	32:00	6
6	2.0	32:00	6
7	2.5	37:30	6
8	2.5	36:30	6
9	3.0	40:00	6
10	3.0	45:00	6
11	3.0	<45:00	6
12	3.0	45:00	6
13	3.0	<45:00	6
14	3.0	45:00	6
15	3.0	<45:00	6
16	3.0	45:00	6
17	3.0	<45:00	6
18	3.0	45:00	6
19	3.0	<45:00	6
20	3.0	45:00	6
21	3.0	<45:00	6

a. **Intensity of exercise and Heart rate.**

The intensity of the progressive walking exercise on a treadmill set was based on the heart rates expressed as percent of the heart rate in maximal exercise. This was derived from the Karvonen[127] formula with use of the age-adjusted maximum heart rates as shown in the Table 2.2

Table 2.2 Age adjusted maximum heart rate in men				
Age in years	30-39	40-49	50-59	60-69
Maximum heart rate	182	178	167	164

Karvonen's formula is as follows:

Maximum age adjusted heart rate - resting heart rate in beats per minute. For example, the working heart rate at 60% for 45 year-old men was calculated as 178-70 (the resting heart rate) x 60% + 70 (the resting heart rate) = 135. At 70% the working heart rate was 146.

b. Exercise Precautions.

With each exercise session, the condition of each participant was carefully evaluated for the following signs and symptoms:

1. Exercise fatigue following a workout; full recovery was expected within two hours after each session.
2. Musculoskeletal symptoms.
3. Chest pain or pain in teeth, jaw, arm or ear.
4. Light-headedness or dizziness.
5. Any irregularity of the pulse rate.
6. Excessive dyspnea, wheezing or cough.
7. Nausea, vomiting or headache.
8. Any difficulty with co-ordination and
9. Failure of the recovery within two minutes after stopping exercise to fall within 50% to 60% of the exercising heart rate.

Above figure was calculated by dividing the two-minute recovery heart rate by exercise heart rate. For example, a 52 year-old man exercised at a working rate of 138 beats per minute. Two minutes after stopping exercise, his recovery rate was 80 beats per minute, 80 138 = 58%. A 60 year-old man exercised at a working heart rate of 136 beats per minute. His two-minute recovery heart rate was 75 beats per minute, 75 136 = 55%.

by which the subjects systematically contracts/tenses and then relaxes the major muscle groups in the body in the orderly manner so as to achieve a state of deep relaxation. Here, the left foot, leg, arm, shoulder and also the facial muscles were tensed. After systematically tensing and relaxing each muscle group, feelings and sensations of warmth, heaviness and relaxation were observed on subjects, throughout the body. Deepening of the level of relaxation was encouraged by focusing on the breathing, which was smooth and regular with no pauses or jerks and with an even inhalation/exhalation cycle. This therapy was practised 20-30 minutes duration one time daily just after the end of the brisk and sustained walking programme on treadmill set (Fig 2.3).

3. **Deep breathing exercise**

Smooth, regular, diaphragmatic breathing was used also for relaxation. Subjects were taught to check his breathing pattern when practising relaxation[400]. The lower hand was raised on inhalation and fell on exhalation (Fig 2.4), with the top hand barely moving. It was practised on the subjects 20-30 minutes twice daily specially after morning and evening prayer.

4. **Simple Meditation**

In this study a simple type of meditation was applied to lower blood pressure. Here a correct posture (Fig 2.6) by sitting on a chair was maintained. In order to have the proper weight

FIGURE 2.3. Progressive Neuromascu
Relaxation therapy.

FIGURE 2.4. Deep Breathing exercise

distribution, the head, neck and trunk was in a straight line, while the hands were rested in a cradled position on the lap and the feet rest on the ground just beneath the knees[400]. It was practised by the subjects by every day specially in the afternoon with a minimum duration of one hour.

5. Dietary schedule of subjects

The subjects received a diet of 2000 kcal per day for the entire period of study.

Sodium less than 1 gm; common salt 2.5 gm

Break-fast	Quantity	Energy	Remarks
Chapattis/ Bread of wheat	75 gm	255	Small size 3 pieces
Egg (hen)	1	60	
Vegetables	1 cup	20	Cooked
Tea	1 cup		Sugar 0.5 TSF
Mid-morning (At 1100 hours			
Fruit (Banana/orange)	100 gm	98	One
Milk	225 cc	151	One glass with sugar 2 TSF

FIGURE 2.5. Low sodium diet with vegetables & salad Cal up to 2000 kcal.

FIGURE 2.6. Simple Meditation

Lunch			
Rice cooked	100 gm	345	2.5 cup
Fish/Meat (chicken)	60 gm	70	Large size one piece
Vegetables and leafy	1 cup	20	Cooked vegetables
Vegetables salad	1 cup	20	Fresh
Pulses	28 gm	85	One cup
Mid-afternoon			
Tea	1 cup		Sugar 0.5 TSF
Biscuit	14 gm	55	Two (Soya protein fruits, biscuits)
Dinner			
Rice cooked	100 gm	345	2.5 cup
Fish/meat (chicken)	60 gm	70	Large size 1 piece
Vegetables & leafy	1 cup	20	Cooked vegetables
Vegetables salad	1 cup	20	Fresh
Edible oil	35 cc	315	For all cooking
Sugar	15 gm	60	

Dietary precautions

1. No salt or baking soda to be used in cooking
2. No salt permitted at the table
3. Salted biscuit is forbidden
4. No canned products and diet other than schedule are permitted.

5. Diet for the subjects was adjusted to the body weight.

2.17 FOLLOW-UP MEASUREMENTS OF BLOOD PRESSURE

Following discharge from Hospital, data were collected by File card (Appendix-E) at monthly interval for six months from only intervention group by trained Medical Assistant of Army Medical Corps; who were blinded to participants therapies. Blood pressure were measured after the subject sat for 5 minutes at rest. Three readings (first and fifth Korotkoff's sounds) were recorded at each visit and averaged.

Other measurements assessed the success of interventions, as well as possible confounding factors. Participants were weighed on a balance-beam scale at the first of each series of three visits and at each individual visit. Dietary modifications and intermediate response to stress management were inquired about. Other queries evaluated the subjects prescription drug use (if any), psychological general well-being and side effects.

FIGURE 2.7. Follow up measurement of BP at monthly interval. (Rechecked by investigator)

FIGURE.2.8. Supervisor of the project observing a patient on a Treadmill machine.

2.18 DATA COLLECTION PROCEDURE

1. A pretested data collection sheet was prepared (Specimen attached as Appendix-A). The duty medical Assistant was provided with information manual as a guide or a ready reckoner to fill up the data collection sheet efficiently and uniformly. The data collection was consists of two parts, part I and part II respectively. The part I of the sheet comprised of various personal data of the subjects, such as personal number or CS number, name, age, rank or status, name of the unit or institution or organization where the individual had been serving. The nature of job i.e history of occupation and different trades of the services were included in the information sheet. The subjects, height, weight (at baseline and after intervention) smoking habit, routine test of urine, blood for complete picture, ECG and X-ray chest were also included in the data collection instrument.

Part II of the Data Collection Sheet consists of data on clinical signs and symptoms. Clinical data i.e. symptoms is usually symptomless. Symptoms ascribed by subjects to hypertension such as headache griddiness, insomonia, palpitation, fatigue etc. are rarely due to hypertension. Nonetheless the clinical data was yet structured as symptoms present or symptoms absent before and after intervention. The systolic and diastolic blood pressures at baseline and after intervention on daily basis at morning and

evening measurement were incorporated on the sheet. Information on blood chemistry such serum electrolytes, lipid profiles, urea, creatinine and fasting plasma glucose were included for recording after collecting the report from Armed Forces Institute of pathology Dhaka Cantonment.

2. On the day of admission all relevant data regarding personal information was recorded. All baseline measurement for example height, in metre, weight in kg, urine, blood, E.C.G., X-ray chest were carried out and all the results of the investigations were noted on data collection sheet.

All blood pressure measurements at morning and evening were noted by duty Medical Officer of the ward. In addition duty Medical Assistant also obtained regular blood pressure recording on each subject. All subjects had the following diagnostic studies that is a complete medical history and clinical examination and all other relevant laboratory investigations. The results of all examinations/procedures were noted on data collection sheet.

2.19 DATA QUALITY ASSURANCE

Quality Control checks were carried out throughout the study. On the day or prior to Hospital admission all baseline measurement for example height, weight, urine, blood for complete picture,

ECG, X-ray chest were completed accurately. Blood pressure was recorded with a mercury sphygmomanometer. All blood pressure measurements were noted by duty Medical Assistants in their respective wards/ departments and all measurements were checked by investigator.

Physical activity levels were obtained from the statement of the patient and recorded as sedentary and hard working. All efforts were made not to alter the scheduled dietary programme of the Hospitalized subjects. Thus every efforts were made to keep constant those variable such as weight, dietary changes which might have influenced the blood pressure change favourably.

The subjects baseline, resting and pre-programme intervention blood pressure were the average of many, which were taken at different times of the day and under various environmental situation. In the hypertensive subjects the pressure had been persistently elevated as per WHO criteria on repeated examination. Baseline blood pressure were also obtained by duty medical officer on two different occasions with three measurements before the patients started the programmes. All measurements were by auscultatory method. In none of the case was an underlying pathologic cause discovered. All cases were diagnosed as primary essential hypertension.

Other measurements assessed the success of interventions, as well as possible confounding factors. Intermediate response to stress management was assessed by questionnaire. The questionnaire evaluated other intervention and therapies, psychological, general well being and side effects (exercise and relaxation only). Data sheets were reviewed for completeness and consistency daily. Records were coded for computer analysis from Med wards, physiotherapy room of CMH and Armed Forces, Institute of Pathology were analysed. Special inquiries were made in cases of discrepancy and then data was finalized. Since there is considerable variability in Blood Pressure measurement as well as weight reduction, trial average values for each person were used for these variables in the main analyses. In addition comparisons were made based on findings at fixed duration. In this way total trial experience of the entire sample was used.

2.30 DATA PROCESSING AND ANALYSIS

The collected data were processed following the procedures as outlined below:-

a) Filled up data collection sheets were edited using a check list developed earlier.

b) The edited schedules were verified.

c) The data of the verified schedules were then coded as per code plan.

d) The coded data were also verified again in coding the same schedules.

e) The data were then entered into computer and checked for range and logical consistency. Errors thus found were corrected after checking the sheets.

f) The data were analyzed as per the tabulation plans prepared, keeping in view, the objectives of the study. Computer facilities of the Directorate General Medical Services Dhaka Cantt and INFS Dhaka University were utilized.

2.21 STATISTICAL METHODS:

For statistical analysis - a standard computer package that is SPSS PC ± was used to compare blood pressure within and between experimental and Control Groups. For each intervention, baseline characteristics in intervention and control groups were examined for equal allocation of variables including age, blood pressures, nutrient levels and biochemical measurements. The mean difference in blood pressure change between each intervention and control group was assessed by student's t test. Two tailed paired and unpaired t tests were used, based on prior study hypotheses of direction of change. Differences in proportions were compared non-parametrically using the Pearson chi-square test corrected for small numbers and means, and their standard deviation. Standard error, with 95% confidence intervals were reported throughout.

To examine the relationship within the experimental or control groups with their respective variable changes to blood pressure end points, an use was made of calculating co-efficient of correlation. For further analysis, linear regression was also used. Significant tests were carried out as per the objective of the study.

It should be noted here that in Chapter-III (Baseline data) majority of the tables were larger than 2x2 table and also the cell numbers less than 5 where chi-squared test was applied. For our convenience and validity of the test we computed the same by combining rows (or columns) with low expected numbers as recommended by Cochran (1954) (Kirkwood B.R. Essentials of Medical Statistics. Blackwell Scientific Publication, 1988; pp: 91).

CHAPTER-III

RESULTS

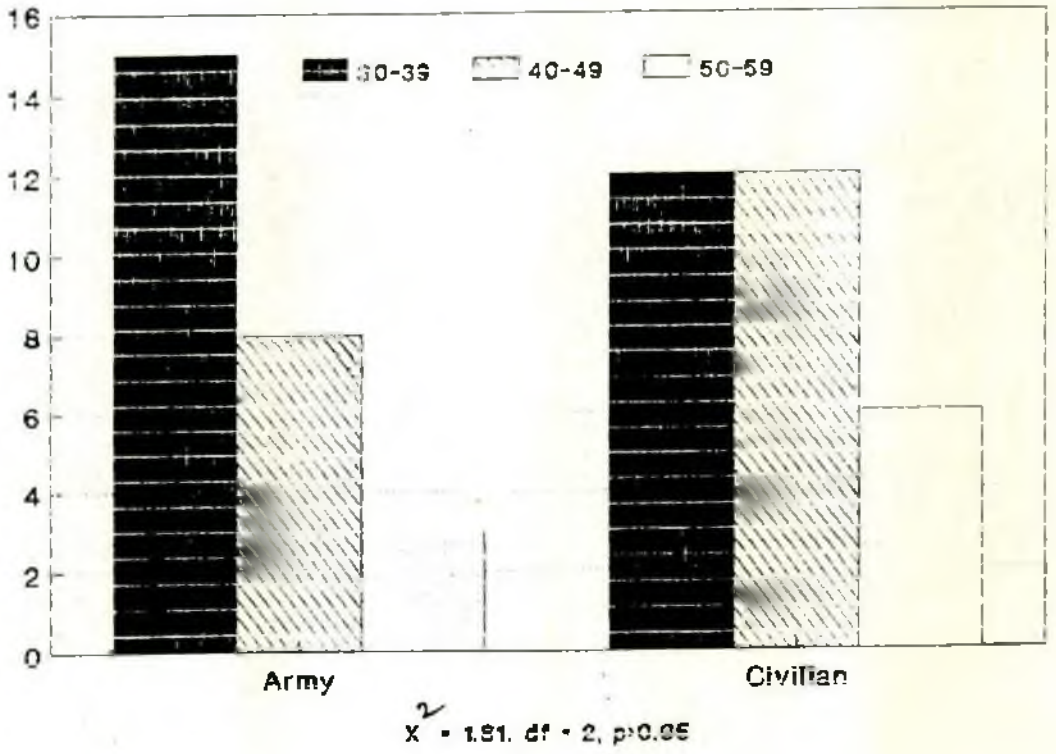
3.1 AGE, CATEGORY OF SUBJECT AND BLOOD PRESSURE

Figure 3.1 shows that 48.2% of the subjects belonged to the age group of 30-39 years. Of them 57.6% were army personnel and 40.0% entitled civilian. In the age group of 40-59 years there were 42.2% army and 60% serving or retired civilian employees respectively (Nonuniform employee.)

Of the total 56 subjects majority (53.5%) were civilian and 46.4% army personnel. However, their difference was not statistically significant.

The variations of blood pressure were observed in different categories of personnel (Table-1). Army personnel having systolic blood pressure equal to 160 mmHg and above were 33.3% whereas in civilian it was 34.2%. Out of 56 subjects 14.6% and 33.9% with systolic blood pressure 140-149 and 160 + mmHg were army personnel and civilian (entitled) respectively.

FIGURE 3.1. Distribution of subjects by age and category



X^2 = 1.81, df = 2, p>0.05

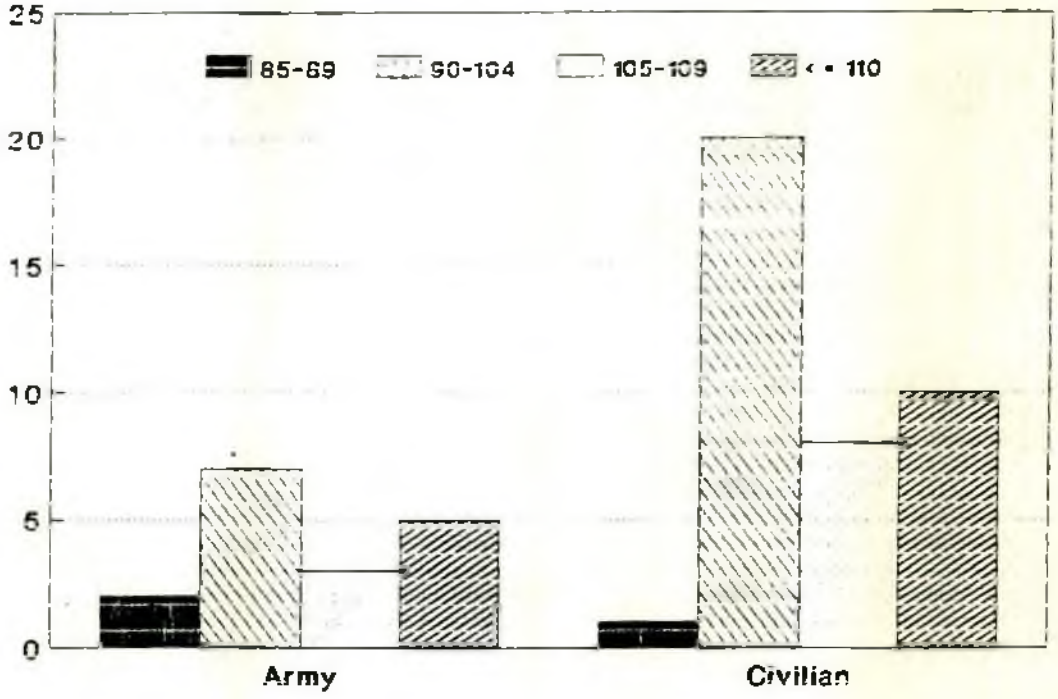
Table 1 **Systolic Blood pressure by category of subjects**

Systolic BP mmHg	Category		
	Army	Civilian	Total
140-149	10 (47.6%)	15 (42.8%)	25 (44.6%)
150-159	4 (19.0%)	8 (22.8%)	12 (21.4%)
160+	7 (33.3%)	12 (34.2%)	19 (33.9%)
Total	21 (37.5%)	35 (62.5%)	

x^2 = 0.14, df = 2, p>0.05

The variations of diastolic blood pressure were analysed in terms of categories of subject enrolled in the study (Fig 3.2). It was noted that 29.4% army and 25.6% civilian had diastolic blood pressure less than equal to 110 mmHg. Diastolic blood pressure within 90-104 mmHg Hg in both the categories i.e. Army and Civilian was 48.2%. Of them majority of the civilian (51.2%) had diastolic blood pressure on that range. This variation of hypertension in different categories of personnel was not statistically significant.

FIGURE 3.2. Initial Diastolic blood pressure by category of subject



Both systolic and diastolic blood pressure as ascertained from various age groups were shown in table 2 and 3. It has been observed that 62% having systolic blood pressure 140-149 mmHg were in the age group of 30-39 years and 50-59 years respectively.

Table 2 Systolic Blood pressure by age

Systolic BP mmHg	Age group in years			
	30-39	40-49	50-59	Total
140-149	18 (62.0%)	7 (36.8%)	5 (62.5%)	30 (53.5%)
150-159	2 (6.8%)	0	0	2 (23.5%)
160 +	9 (31.0%)	12 (61.1%)	3 (37.5%)	24 (42.8%)
Total	29 (51.7%)	19 (33.9%)	8 (14.2%)	

$X^2 = 3.23$, df = 4, $p>0.25$

In 40-49 years of age group systolic blood pressure equal to and greater 160 mmHg was recorded in 63.1% of subjects. Systolic blood pressures were varied in different age groups and the results was not statistically significant ($p<0.25$).

Initial diastolic blood pressure and age group of subjects are shown in Table-3. In age group of 50-59 years 66.6% of the subjects had diastolic blood pressure 90-104 mmHg and 22.2% less than equal to 110 mmHg. 51.8% and 35% with diastolic blood pressure 90-104 mmHg were in the age group of 30-39 years and 40-49 years respectively.

Table 3 **Diastolic Blood pressure by age**

Diastolic BP mmHg	Age group in years			
	30-39	40-49	50-59	Total
85-89	5 (18.5%)	2 (10.0%)	0	7 (12.5%)
90-104	14 (51.8%)	7 (35.0%)	6 (66.6%)	27 (48.2%)
105-109	4 (14.8%)	6 (30.0%)	1 (11.1%)	11 (19.6%)
110	4 (14.8%)	5 (25.0%)	2 (22.2%)	11 (19.6%)
Total	27 (48.2%)	20 (35.7%)	9 (16.0%)	

$X^2 = 2.89$, df = 6, p 0.25

Only 14.8% at the age group of 30-39 were detected to have diastolic blood pressure equal to or less than 110 mmHg. Of the total subject only 48.2% having diastolic blood pressure recorded to be 90-104 mmHg. The variations of diastolic blood pressure in terms of association with different age groups were not statistically significant($p<0.25$).

3.2 PHYSICAL ACTIVITY AND BLOOD PRESSURE

Table-4 shows the variation of blood pressure as per nature of physical activity. Amongst sedentary subjects (57.1%), their systolic blood pressure were recorded to be equal to and above 160 mmHg whereas 52.3% with same systolic pressure were hard working service personnel. This variation of systolic

Table 4 **Systolic Blood pressure by nature of physical activity**

Systolic BP mmHg	Nature of physical activity		
	Sedentary	Hardworking	Total
140-149	7 (20.0%)	3 (14.2%)	10 (17.8%)
149-159	8 (22.8%)	7 (33.3%)	15 (26.7%)
160 +	20 (57.1%)	11 (52.3%)	31 (55.3%)
Total	35 (62.5%)	21 (37.5%)	

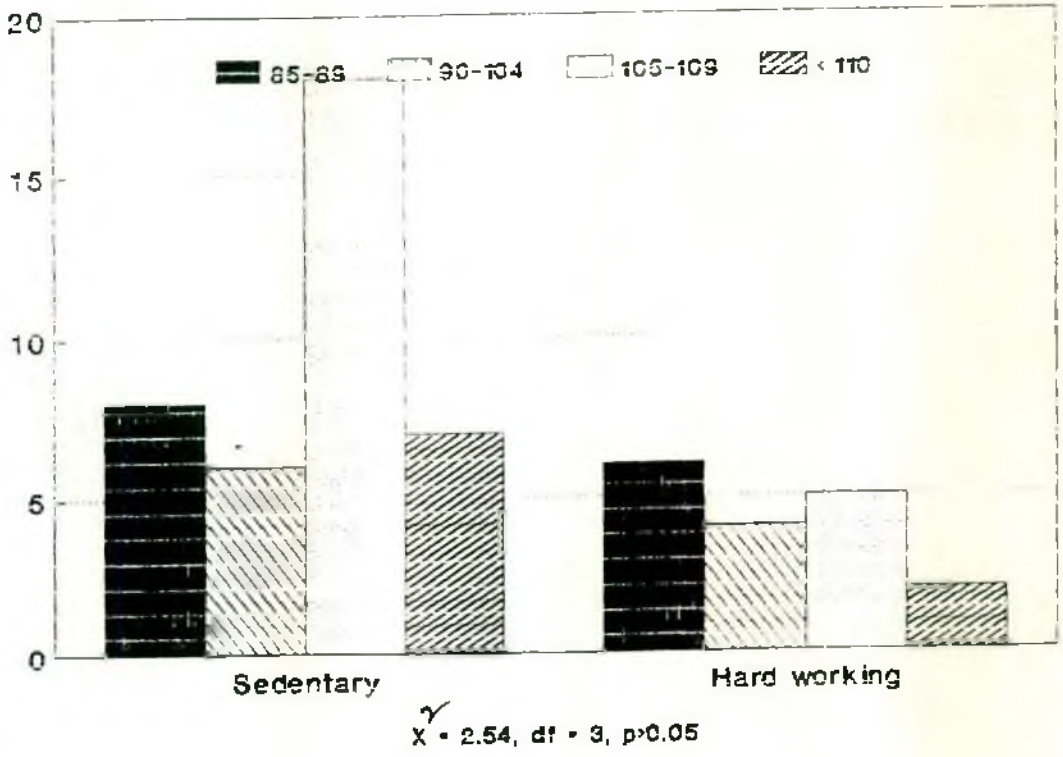
X^2 = 0.82, df = 2, p>0.05

blood pressure as per the nature of physical activity was not statistically significant.

Subjects initial diastolic blood pressure were distributed in Figure 3.3 as per the nature of physical activity. In 35.2%, 29.4%, and 11.7% of the subjects having diastolic blood pressure 85-89, 105-109 and less than equal to 110 mmHg were detected in hard working service employees.

46.1% and 17.9% sedentary subjects had diastolic blood pressure 105-109 and less than equal to 110 mmHg. The major variation of diastolic blood pressure were detected in the sedentary people. This findings were not significant.

FIGURE 3.3. Initial diastolic blood pressure by nature of physical activity



χ^2 = 2.54, df = 3, p>0.05

3.3 SMOKING HABIT AND BLOOD PRESSURE

Initial systolic blood pressure by smoking habit and initial diastolic blood pressure by the same habit were shown in Table 5 and 6. Out of 56 subjects 35 (62.5%) were smoker. Of them 40% were detected to have systolic blood pressure in the range of 140-149 mmHg.

Table 5 Systolic Blood Pressure by smoking habit

Systolic BP mmHg	Smoking habit		Total
	Currently smoker	Never smoked	
140-149	14 (40.0%)	9 (42.8%)	23 (41.0%)
150-159	9 (25.7%)	5 (23.8%)	14 (25.0%)
160 +	12 (34.2%)	7 (33.3%)	19 (33.9%)
Total	35 (62.5%)	21 (37.5%)	56

x^2 = 0.02, df = 2, p>0.05

Only 34.2% had systolic blood pressure equal to and greater than 160 mmHg. 42.8% and 33.3% of the non-smokers had systolic pressure 140-149 mmHg and equal to and greater than 160 mmHg.

Of the total 56 subjects, 36 (64.2%) had habit of smoking (Table 6 whose diastolic blood pressure ranged from 85 to 110 mmHg. Of them 44.0% and 22.2% of the smoker had diastolic blood pressure 105- 109 mmHg and equal to and greater than 110 mmHg. 35.0% and 20.0% non-smokers had diastolic blood pressure 90-104 mmHg.

Table 6 **Diastolic Blood pressure by smoking habit**

Diastolic BP mmHg	Smoking habit		
	Currently smoker	Never smoked	Total
85-89	5 (13.8%)	3 (15.0%)	8 (14.2%)
90-104	7 (19.4%)	7 (35.0%)	14 (25.0%)
105-109	16 (44.4%)	6 (30.0%)	22 (39.2%)
110	8 (22.2%)	4 (20.0%)	12 (21.4%)
Total	36 (64.2%)	20 (35.7%)	56

x^2 = 1.9 , df = 3, p>0.05

and 110 mmHg respectively. Smoking habit and variation of blood pressure both systolic and diastolic did not show statistical association.

3.4 BODY WEIGHT, HEIGHT, BMI, PBMI

Table 7 presents the distribution of body weight in kg with baseline systolic blood pressure. Majority of the subjects (61%) with systolic blood pressure 140-149 mmHg had body weight ranging from 65-84 kg. Out of 24 subjects (42.8%) with high systolic blood pressure (160 and above) only 2 subjects showed weight more than or equal to

Table 7 Systolic Blood pressure by body weight

Systolic BP mmHg	Body weight in kg			
	45-64	65-84	85 +	Total
140-149	9 (45.0%)	21 (61.7%)	0	30 (53.5%)
150-159	0	2 (5.8%)	0	2 (3.5%)
160 +	11 (55.0%)	11 (32.3%)	2 (100.0%)	24 (42.8%)
Total	20 (35.7%)	34 (60.7%)	2 (3.57)	

85 kg. 55.0% of the subjects whose systolic blood pressure was equal to or greater than 160 mmHg, the range of their weight was 45-64 kg. With systolic blood pressure 150-159 mmHg only 5.8% had 65-84 kg body weight.

Figure 3.4 shows the distribution of subjects by body weight and initial diastolic blood pressure. Majority of the subjects (58.9%) show the body weight within the range of 65 to 84 kg, 66.6% of the subjects had equal to or greater than 85 kg body weight whose diastolic blood pressure was 90-104 mmHg.

35.0% and 54.5% had 45 to 84 kg of body weight at 90-104 mmHg diastolic blood pressure. 10.0% and 40.0% of the subjects with 45-64 kg of body weight had 85-89 and 110 mmHg diastolic blood pressure respectively.

FIGURE 3.4 Distribution of subjects by body weight and initial diastolic blood pressure

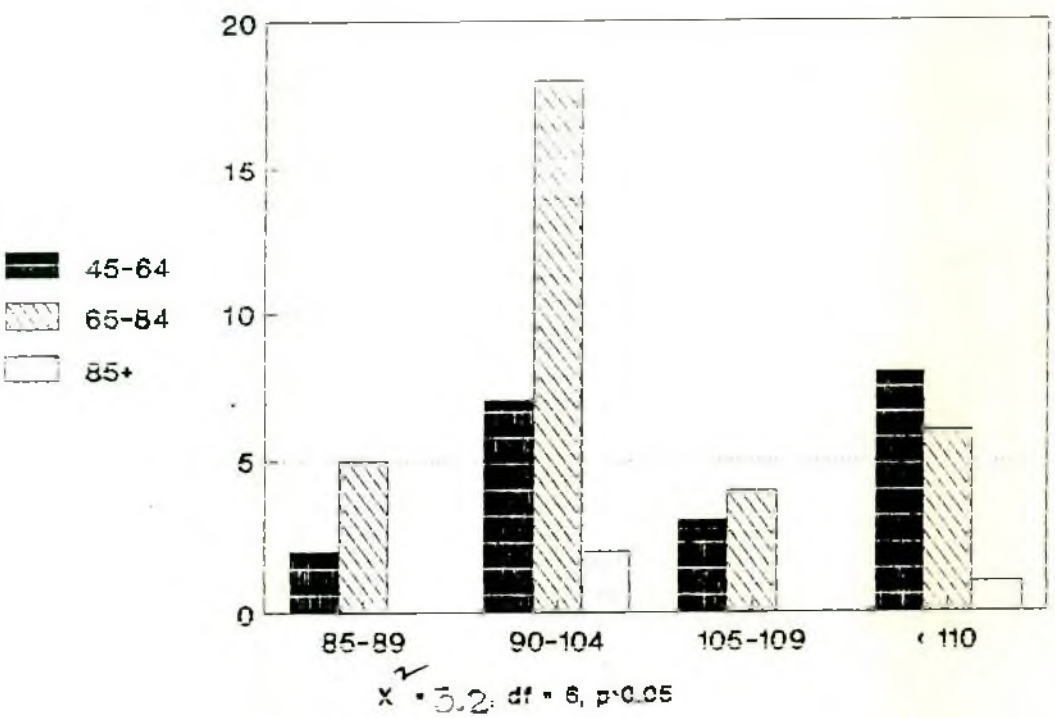


Table 8 and 9 shows the distribution of subjects with initial systolic and diastolic blood pressure by their respective heights. Systolic blood pressure recorded as 140-149 mmHg in 75% of the subjects whose heights were ranging from 1.71 to 1.90 metre.

Table 8 **Initial Systolic Blood pressure by height**

Systolic BP mmHg	Height in metre		
	1.51-1.70	1.71-1.90	Total
140-149	21 (47.7%)	9 (75.0%)	30 (53.5%)
150-159	2 (4.5%)	0	2 (3.5%)
160 +	21 (47.7%)	3 (25.0%)	24 (42.8%)
Total	44 (78.5%)	12 (21.4%)	56

and 25.0% whose heights were 1.51 to 1.70 metre, their systolic blood pressure recorded to be equal to and above 160 mmHg.

On the other hand 83.3% of the subjects whose heights were 1.71 to 1.90 metre their diastolic blood pressure corresponds 90-104 mmHg. On the same range of height no subjects were recorded to have diastolic blood pressure less than and equal to 110 mmHg. 40.9% of the subject with height 1.51 to 1.70 metre, their diastolic

Table 9 **Initial Diastolic Blood pressure by height**

Diastolic BP mmHg	Height in metre		
	1.51-1.70	1.71-1.90	Total
85-89	7 (15.9%)	0	7 (12.5%)
90-104	18 (40.9%)	10 (83.3)	28 (50.0%)
105-109	5 (11.3%)	2 (16.6%)	7 (12.5%)
110	14 (31.8%)	0	14 (25.0%)
Total	44 (78.5%)	12 (21.4%)	56

blood pressure was 90-104 mmHg. In both the table the results of systolic and diastolic blood pressure varies in terms of different height of the subjects.

Body mass index and initial systolic blood pressure is given in table 10. Majority of the subjects (76.7%) had BMI 19-25 kg/m^2. Of them 53.4% and 41.8% had systolic blood pressure, 140-149 mmHg and equal to and greater 160 mmHg respectively.

Table 10 **Systolic Blood pressure by BMI**

Systolic BP mmHg	BMI in kg/m^2		
	19-25	26+	Total
140-149	23 (53.4%)	7 (53.8%)	30 (53.5%)
150-159	2 (4.6%)	0	2 (3.5%)
160 +	18 (41.8%)	6 (46.1%)	24 (42.8%)
Total	43 (76.7%)	13 (23.2%)	

Only 23.2% had BMI 24-30 kg/m^2. Of them 46.1% falls in the systolic blood 140-149 mmHg and 160 mmHg. With systolic blood pressure 140-149 mmHg 53.8% gives BMI 26 and above kg/m^2.

Table 11 shows the proportion of subjects with diastolic blood pressure and BMI. Majority of the subjects (76.7%%) had BMI 19 to 25 kg/m^2. Of them 48.0% had diastolic blood pressure 90-104 mmHg respectively. With BMI 26 and above Kg/M^2, 46.1% had

Table 11 **Diastolic Blood pressure by BMI**

Diastolic BP mmHg	BMI in kg/m^2		
	19-25	26+	T o t a l
85-89	5 (11.6%)	2 (15.3%)	7 (12.5%)
90-104	21 (48.8%)	6 (46.1%)	27 (48.2%)
105-109	5 (11.6%)	2 (15.3%)	7 (12.5%)
110	12 (27.9%)	3 (23.0%)	15 (26.7%)
Total	43 (76.7%)	13 (23.21%)	

diastolic pressure 90-104 mmHg and only in 23% was equal to and greater than 110 mmHg. BMI does not vary with diastolic blood pressure.

Table 12 shows the maximum and minimum BMI and initial systolic blood pressure. Here 53.8% of the subjects with lowest BMI fell in the systolic blood pressure of 140-149 mmHg whereas 41.0% in the high systolic pressure had 160+ mmHg. On the other hand 52.9% of the subjects with systolic blood

Table 12 **Systolic Blood pressure by highest and lowest BMI**

Systolic BP mmHg	BMI in kg/m^2		
	<25	>25	Total
140-149	21 (53.8)	9 (52.9%)	30 (53.5%)
150-159	2 (5.1%)	0	2 (3.5%)
160+	16 (41.0%)	8 (47.0%)	24 (42.8%)
Total	39 (69.6%)	17 (30.3%)	

Pressure 140-149 mmHg had highest BMI. Subjects with high systolic blood pressure (160+mmHg) show the highest BMI in 47.0% of the subjects respectively. Subjects with minimum and maximum BMI do not rise and fall with systolic blood pressure.

Initial diastolic blood pressure with highest BMI and lowest BMI is presented in table 13. Of the total 56 subjects 67,8% had lowest and only 32.1% highest BMI. With diastolic blood pressure 90-104 mmHg, 44.7% and 55.5% had maximum BMI.

Table 13 **Diastolic Blood pressure by lowest and highest BMI**

Diastolic BP mmHg	BMI in Kg		
	<25	>25	Total
85-90	5 (13.1%)	2 (11.1%)	7 (12.5%)
90-104	17 (44.7%)	10 (55.5%)	27 (48.2%)
105-109	5 (13.1%)	2 (11.1%)	7 (12.5%)
$\leq$ 110	11 (28.9%)	4 (22.2%)	15 (26.7%)
Total	38 (67.8%)	18 (32.1%)	

x^2 = 0.54, df = 6, p<0.25

Diastolic blood pressure less than equal to 110 mmHg showed 28.9% and 22.2% at the lowest and highest BMI. This variation of minimum and maximum BMI with different levels of diastolic blood pressure was not associated significantly.

Table 14 gives the proportion of subjects in relevance to their initial systolic blood pressure and PBMI. Out of 56 subjects, 83.6% and 16.0% had normal and obese PBMI. Of them 42.8% and 37.5% with normal range of PBMI had systolic blood pressure, 140-149 mmHg and equal to and greater than 160 mmHg.

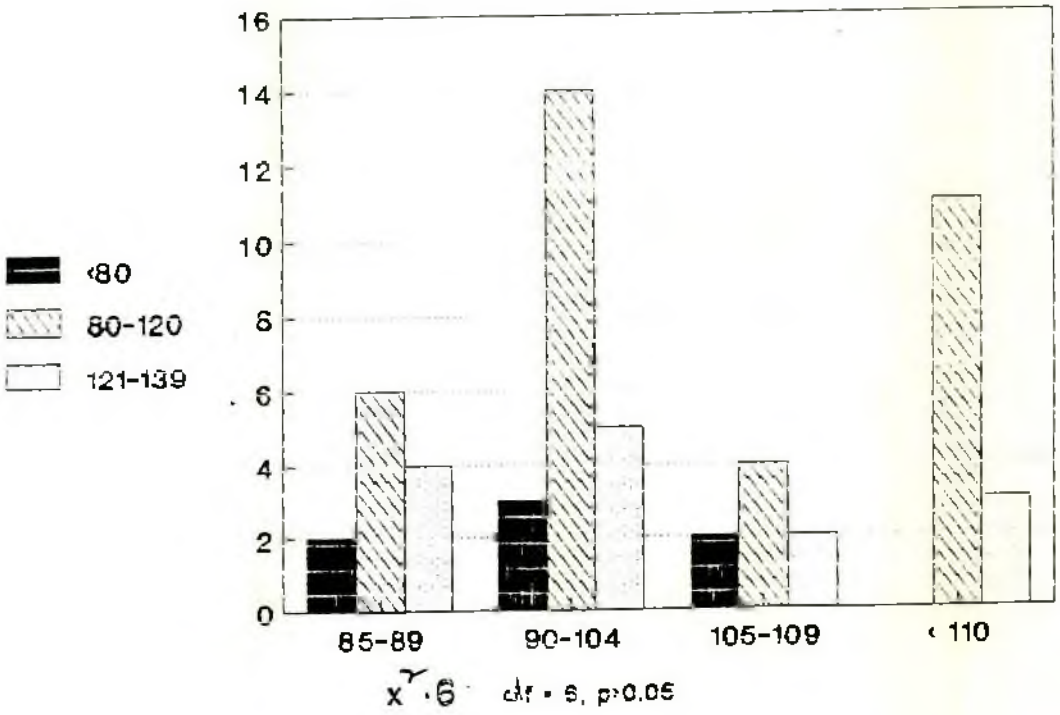
Table 14 **Systolic Blood pressure by PBMI**

Systolic BP mmHg	PBMI in Kg			
	Lean	Normal	Obese	Total
140-149	0	24 (51.0%)	6 (66.6%)	30 (53.5%)
150-159	0	2 (4.2%)	0	2 (3.5%)
160+	0	21 (44.6%)	3 (33.3%)	24 (42.8%)
Total		47 (83.9%)	9 (16.0%)	

Systolic blood pressure within the range of 140 to 160 mmHg, there was no lean subjects i.e., PBMI less 80.

Percent body mass index and the diastolic blood pressure levels are depicted in Figure 3.5. 39.2% and 25.0% of the subjects having diastolic blood pressure 90-104 mmHg and 110 mmHg indicate the varying proportions of PBMI. In PBMI, 42.8%, 40.0% and 35.7% of the subjects with diastolic blood pressure 90-104 mmHg were lean, normal and obese respectively while 31.4% and 21.4% of the subjects with diastolic blood pressure 110 mmHg were normal and obese. It is evident that PBMI does not vary with the rise of diastolic blood pressure.

FIGURE 3.5 Distribution of subjects by Initial diastolic blood pressure and pBMI



3.5 SERUM SODIUM LEVEL AND BLOOD PRESSURE

Baseline serum sodium level was determined in all the subjects and their values were distributed in varying grades of systolic and diastolic blood pressure (Table 15,16). At systolic blood pressure 140-160 mmHg, 62.5% of the subjects had serum sodium level, ranging from 140-144 mmol/L. Of them 48.5% and 45.7% with the same levels of serum sodium had initial systolic blood pressure.

Table 15 Systolic Blood pressure by serum sodium level

Systolic BP mmHg	Na mmol/L		
	135-139	140-144	Total
140-149	11 (52.3%)	17 (48.5%)	28 (50.0%)
150-159	2 (9.5%)	2 (5.7%)	4 (7.1%)
160 +	8 (38.0%)	16 (45.7%)	24 (42.8%)
Total	21 (37.0%)	35 (62.5%)	

x^2 = .05 , df = 2, p>0.05

140-149 and 160+ mmHg. Only 5.7% had systolic blood pressure 150 to 159 mmHg at 140-144 mmol/L of serum sodium level. Majority of the subjects (52.3%) had 135-139 mmol/L of sodium at systolic blood pressure 140-149 mmHg(Table 20).

Table 16 shows the diastolic blood pressure of subjects and their serum sodium. Of the total subjects, 64.2% at varying grades of diastolic pressure showed 140- 144 mmol/L of serum sodium. Of them 47.2% with the same level of serum sodium had diastolic blood pressure 90-104 mmHg. Only 27.7%.

Table-16 **Diastolic Blood pressure by serum sodium level**

Diastolic BP mmHg	Na mmol/L		
	135-139	140-144	Total
85-89	4 (20.0%)	3 (8.3%)	7 (12.5%)
90-104	9 (45.0%)	17 (47.2%)	26 (46.4%)
105-109	2 (10.0%)	6 (16.6%)	8 (14.2%)
≤ 110	5 (27.7%)	10 (27.7%)	15 (26.7%)
Total	20 (35.7%)	36 (64.2%)	

x^2 =,47 , df = 3, p>0.05

of the subject (at 140-144 mmol/L) had diastolic blood pressure equal to and greater than 110 mmHg. 45% and 25% of the subjects with 135-139 mmol/L of serum sodium had diastolic blood pressure 90-104 and equal to and greater than 110 mmHg respectively. The above variation of serum sodium level with different grades of blood pressure was not statistically significant.

3.6 SERUM POTASSIUM LEVEL AND BLOOD PRESSURE

Table 17 and 18 show the distribution of serum potassium level with different levels of blood pressure. At 4.5 to 4.9 mmol/L of potassium the majority (67.8%) of the subject's blood pressure varies from 140-149 mmHg, and 42.1% at equal to and greater than 160 mmHg systolic pressure.

Table 17 Systolic Blood pressure by serum potassium level

Systolic BP mmHg	K m mmol/L			
	3.5-3.9	4.5-4.9	5.5+	Total
140-149	3 (27.2%)	19 (50.0%)	6 (85.7%)	28 (50.0%)
150-159	2 (18.1%)	3 (7.8%)	- 0	5 (8.9%)
160 +	6 (54.5%)	16 (42.1%)	1 (14.2%)	23 (41.0%)
Total	11 (19.6%)	38 (67.8%)	7 (12.5%)	

X^2 = 2.91, df = 4, p 0.25

Potassium level equal to and greater than 5.5 mmol/L had systolic blood pressure 140-149 mmHg in 85.7% of the subjects. No significant association was found in serum potassium and different levels of systolic blood pressure.

It is also shown in Table 18 that 77.7% of the subjects with diastolic blood pressure 90-104 mmHg had serum potassium level equal to and greater than 5.5 mmol/L. Between 4.5 to 4.9 mmol/L majority of subjects had diastolic blood pressure 90-104 mmHg. At 3.5 to 3.9 mmol/L, 50% of the subjects had diastolic blood pressure less than and equal to 110 mmHg.

Table-18 **Diastolic Blood pressure by serum potassium (k) level**

Diastolic BP mmHg	K mmol/L			
	3.5-3.9	4.5-4.9	5.5+	Total
85-89	2 (14.2%)	3 (9.0%)	2 (22.2%)	7 (12.5%)
90-104	3 (21.4%)	17 (51.5%)	7 (77.7%)	27 (48.2%)
105-109	2 (14.2%)	5 (15.1%)	- 0	7 (12.5%)
110	7 (50.0%)	8 (24.2%)	- 0	15 (26.7%)
Total	14 (25.0%)	33 (58.9%)	9 (16.0%)	

Serum potassium level was found to be varied with diastolic blood pressure.

3.7 SERUM CHLORIDE (NACL) AND BLOOD PRESSURE

In all the subjects serum Nacl in undissociated form were determined. Table 19 shows the distribution of chloride (Nacl) with their respective systolic blood pressure levels. 50% of the subjects with 105 mmol/L of chloride had systolic pressure 140-159 mmHg.

Table-19 **Systolic Blood pressure by serum chloride (Nacl)**

Systolic BP mmHg	Chloride mmol/L			
	95-99	100-104	105 +	Total
140-149	12 (60.0%)	16 (47.0%)	1 (50.0%)	29 (51.7%)
150-159	2 (10.0%)	1 (2.9%)	1 (50.0%)	4 (7.1%)
160 +	6 (30.0%)	17 (50.0%)	- 0	23 (41.0%)
Total	20 (35.7%)	34 (60.7%)	2 (3.5%)	

Majority of the subjects whose chloride level, 95-99 mmol/L, their systolic blood pressure was 140-149 mmHg. A certain degree of variation of serum chloride was determined with systolic blood pressure.

Table 20 shows that 50% of subjects having diastolic blood pressure equal to and less than 110 mmHg, whose serum chloride level was 105 + mmol/L 47.0% and 29.4% of the subjects with

Table-20 **Diastolic Blood pressure by serum chloride (Nacl) level**

Diastolic BP mmHg	Chloride mmol/L			
	95-99	100-104	105 +	Total
85-89	3 (16.6%)	4 (11.7%)	0	7 (12.5%)
90-104	9 (50.0%)	16 (47.0%)	1 (25.0%)	26 (46.4%)
105-109	2 (11.1%)	4 (11.7%)	1 (25.0%)	7 (12.5%)
≤ 110	4 (22.2%)	10 (29.4%)	2 (50.2%)	16 (28.5%)
Total	18 (32.1%)	34 (60.7%)	4 (7.1%)	

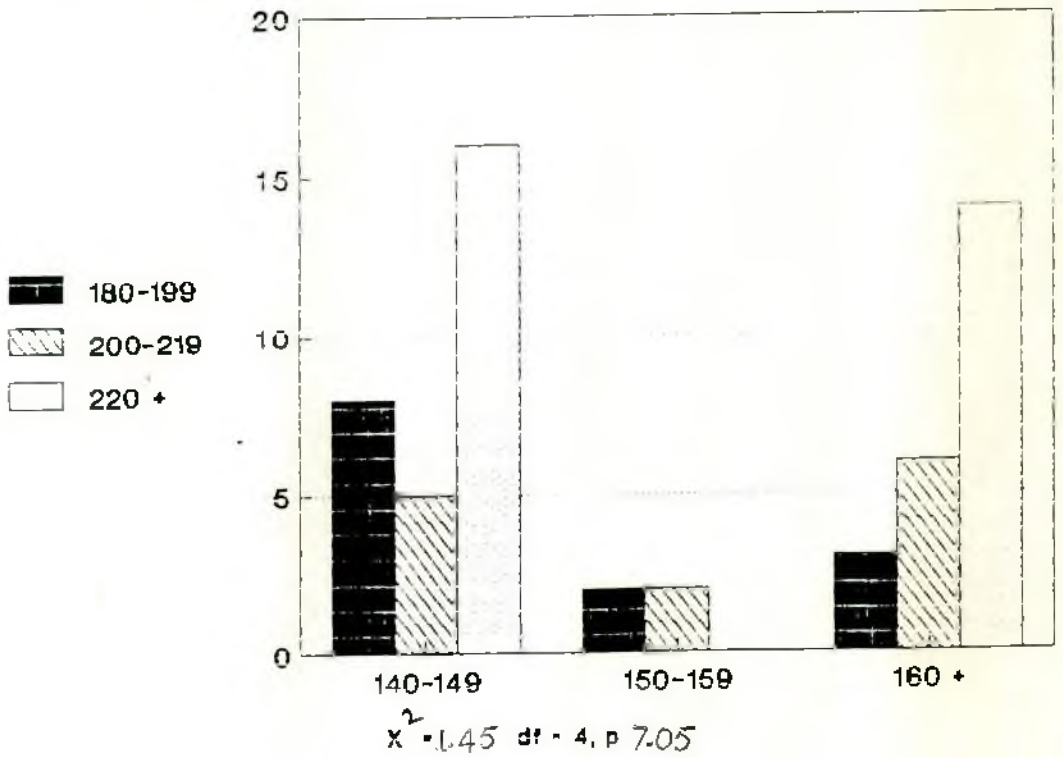
X^2 = 0.66, df = 6, p>0.05

diastolic blood pressure, 90-104 mmHg and 110 mmHg had serum chloride level 100-104 mmol/L. Chloride level between 95-99 mmol/L was found in 50.0% of the subject with diastolic pressure 90-104 mmHg. However, no statistical significant difference between diastolic blood pressure and chloride level was determined.

3.8 SERUM CHOLESTEROL LEVELS AND BLOOD PRESSURE

Subject's serum cholesterol level and their respective systolic and diastolic blood pressure is shown in Figure 3.6 and table 21. Majority of the subjects (53.3%) having systolic blood pressure 140-149 mmHg show serum chloresterol level at equal to and greater than 220 mg/dl. 46.6% of the subjects with high systolic blood pressure (160+ mmHg) had cholesterol at the same level. Subjects with high systolic blood pressure show cholesterol level at 180-199, 200-219 and 220+ mg/dl in 23.0%, 46.1% and 46.6% of the subjects respectively. The serum chloresterol level varies with systolic blood pressure, hence an association is observed in this study.

FIGURE 3.6 Systolic blood pressure by cholesterol level



X^2 = 1.45 df = 4, p 7.05

Majority of the subjects (54.5% & 58.3%) detected to have serum chloresterol level at 180-199 and 200-219 mg/dl with diastolic blood pressure 90-104 mmHg. With the same rise of blood pressure, 42.4% had chloresterol level equal and greater than 220 mg/dl.

Table-21 **Diastolic Blood pressure by serum cholesterol level**

Diastolic BP mmHg	Cholesterol (level mg/dl)			
	180-199	200-219	220+	Total
85-89	2 (18.1%)	2 (16.6%)	3 (9.0%)	7 (12.5%)
90-104	6 (54.5%)	7 (58.3%)	14 (42.4%)	27 (48.2%)
105-109	0	1 (8.3%)	6 (18.1%)	7 (12.5%)
≤ 110	3 (27.2%)	2 (16.6%)	10 (30.3%)	15 (26.7%)
Total	11 (19.6%)	12 (21.4%)	33 (58.9%)	

X^2 = 2.85, df = 6, p>0.05

Hypertension with diastolic blood pressure equal to or less than 110 mmHg shows, 180-199, 200-219, 220+ mg/dl of cholesterol level in 27.2%, 16.6%, and 33.3% of the subjects respectively. However, this variation of chloresterol level at various grades of diastolic blood pressure was not significant at 5% level.

3.9 HDL- CHOLESTEROL AND BLOOD PRESSURE

Serum HDL cholesterol level and its percentage variation with different grades of blood pressure is shown in table 22 and 23. Majority (53.3%) of the subjects having systolic blood pressure 140-149 mmHg showed greater than 55 mg/dl of serum HDL-cholesterol.

Table-22 **Systolic Blood pressure by serum HDL-cholesterol level**

Systolic BP mmHg	HDL cholesterol (mg/dl)		
	<55	>55	Total
140-149	4 (36.3%)	24 (53.3%)	28 (50.0%)
150-159	2 (18.1%)	2 (4.4%)	4 (7.1%)
160 +	5 (45.4%)	19 (42.2%)	24 (42.8%)
Total	11 (19.6%)	45 (80.3%)	

42.2% of the subjects with the same level of serum HDL-cholesterol level had systolic blood pressure equal to and greater than 160 mmHg. Of all the subjects, 80.3% had greater 55 mg/dl serum HDL-cholesterol level whereas 19.6% had less than 55 mg/dl. HDL-cholesterol level varies in different levels of systolic blood pressure.

Table 23 shows the distribution of HDL-cholesterol level and their respective diastolic blood pressure. Majority of the subjects (66.6%) had less than 55 mg/dl serum HDL Cholesterol at 90-104 mmHg diastolic pressure whereas 44.6% showed greater than 55 mg/dl. Out of 56 subjects 47 (83.9%) had greater than

Table-23 **Diastolic Blood pressure by serum HDL cholesterol level**

Diastolic BP mmHg	HDL cholesterol (mg/dl)		
	<55	>55	Total
85-89	1 (11.1%)	6 (12.7%)	7 (12.5%)
90-104	6 (66.6%)	21 (44.6%)	27 (48.2%)
105-109	1 (11.1%)	6 (12.7%)	7 (12.5%)
≤ 110	1 (11.1%)	14 (29.7%)	15 (26.7%)
Total	9 (16.0%)	47 (83.9%)	

55 mg/dl and 16.0% had less than 55 mg/dl of serum cholesterol level. Only 29.7% having diastolic blood pressure 110 mg showed >55 mg/dl HDL cholesterol. No variation of HDL-cholesterol with diastolic blood pressure was detected.

3.10 LDL-CHOLESTEROL AND BLOOD PRESSURE

Table 24 and 25 show the systolic and diastolic blood pressure and serum LDL cholesterol level. At 140-149 mmHg systolic blood

Table-24 **Systolic Blood pressure by serum LDL cholesterol level**

Systolic BP mmHg	LDL cholesterol (mg/dl)		
	<150	>150	Total
140-149	24 (50.0%)	6 (75.0%)	30 (53.5%)
150-159	1 (2.0%)	1 (12.5%)	2 (3.5%)
160+	23 (47.9%)	1 (12.5%)	24 (42.8%)
Total	48 (85.7%)	8 (14.2%)	

pressure, majority of the subjects (75.0%) had greater than 150 mg/dl of LDL-cholesterol whereas at higher level of systolic pressure (160+) only 12.5% showed high level of LDL-cholesterol. 50% and 47.9% of the subjects having systolic blood pressure, 140-149, & 160+ mmHg showed less than 150 mg/dl LDL-cholesterol. The results showed difference in serum LDL-cholesterol and systolic blood pressure.

In table 25, majority of the subjects (71.4%) shows greater than 150 mg/dl of LDL-cholesterol at 90-104 mmHg of diastolic blood pressure. Only 28.2% of the subjects with the same of level of LDL-cholesterol had diastolic blood pressure at 105-109 mmHg. At diastolic blood pressure less than and equal to

Table-25 **Diastolic Blood pressure by serum LDL cholesterol level**

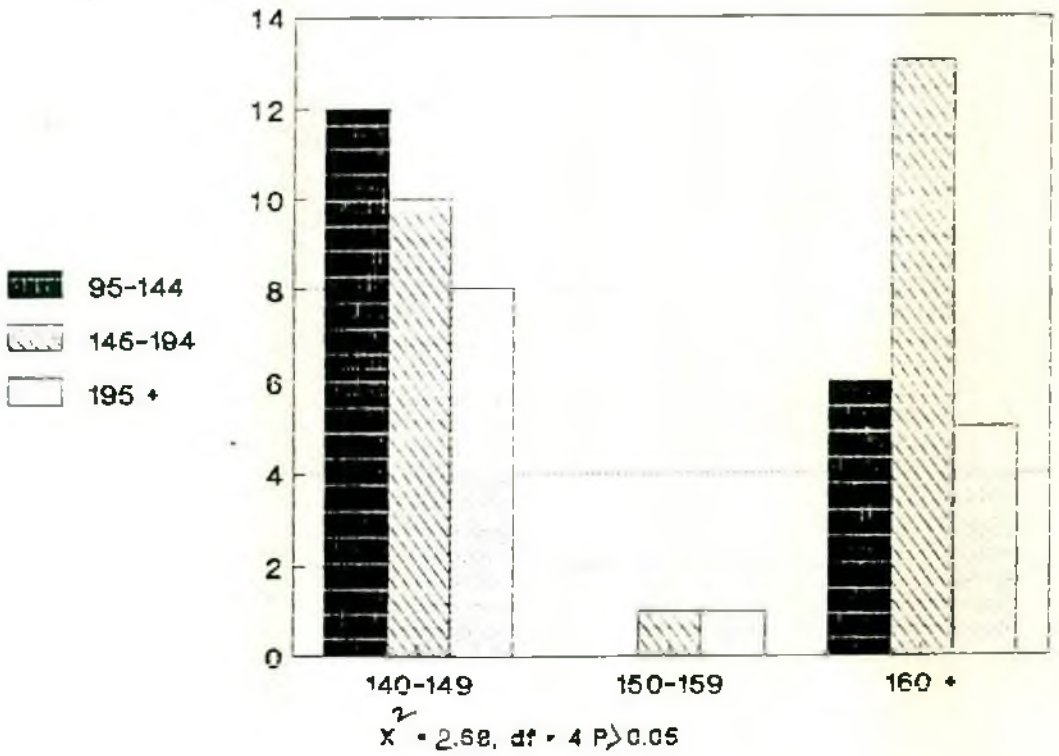
Diastolic BP mmHg	LDL cholesterol (mg/dl)		
	<150	>150	Total
85-89	7 (14.2%)	- 0	7 (12.5%)
90-104	22 (44.8%)	5 (71.4%)	27 (48.2%)
105-109	5 (10.2%)	2 (28.2%)	7 (12.5%)
≤ 110	15 (30.6%)	- 0	15 (26.7%)
Total	49 (87.5%)	7 (12.5%)	

110 mmHg no, subjects were identified having greater than 150 mg/dl of LDL. 44.8% and 30.6% of the subjects had less than 150 mg/dl of LDL-cholesterol whose diastolic blood pressure was 90-104 and 110 mmHg respectively.

3.11 SERUM TRIGLYCERIDES AND BLOOD PRESSURE

Figure 3.7 shows the serum TG and systolic blood pressure. 57.1% of the subjects had equal to and greater than 195 mg/dl of TG at 140-149 mmHg. With the same level of blood pressure, 66.6% and 41.6% of the subjects had 95-144, and 145-194 mg/dl of TG level. At higher level of systolic blood pressure (160+mmHg), 33.3%, 54.1% and 35.7% of the subjects detected to have 95-144, 145-194, 195+ mg/dl of TG respectively.

FIGURE 3.7 Systolic blood pressure by serum Triglycer-ides level



TG level also vary with the diastolic blood pressure (Table-26). Majority of the subjects (57.8%) show 95-144 mg/dl TG at 90-104 mmHg of diastolic blood pressure. 34.7% and

Table-26 **Diastolic Blood pressure by serum Triglycerides level**

Diastolic BP mmHg	Triglycerides (mg/dl)			
	95-144	145-194	195+	Total
85-89	3 (15.7%)	3 (13.0%)	1 (7.1%)	7 (12.5%)
90-104	11 (57.8%)	8 (34.7%)	7 (50.0%)	26 (46.4%)
105-109	1 (5.2%)	3 (13.0%)	3 (21.4%)	7 (12.5%)
≤ 110	4 (21.0%)	9 (39.1%)	3 (21.4%)	16 (28.5%)
Total	19 (33.9%)	23 (41.0%)	14 (25.0%)	

$\chi^2 = 2.89$, df = 6, $P > 0.5$

50.0% of the subjects had 145-194, and 195+ mg/dl TG level at 90-104 mmHg. Hypertension with 110 mmHg, only 21.0%, 39.1% and 21.4% of the subjects showed 95-144, 145- 194 and 195 + mg/dl of TG respectively. No significant difference between TG level and blood pressure was determined.

3.12 SERUM TOTAL LIPIDS AND BLOOD PRESSURE

Subject's total serum lipids and systolic blood pressure are shown in Table 27. From the table 50% of the subject's serum total lipids showed, equal to and greater than 900 mg/dl at the level of high systolic blood pressure (160 + mmHg).

Table 27 Systolic Blood pressure by serum total lipids

Systolic BP mmHg	Total lipids (mg/dl)			
	700-800	800-900	900 +	Total
140-149	5 (45.4%)	18 (58.0%)	5 (35.7%)	28 (50.0%)
150-159	2 (18.1%)	1 (3.2%)	2 (14.2%)	5 (8.9%)
160+	4 (36.3%)	12 (38.7%)	7 (50.0%)	23 (41.0%)
Total	11 (19.6%)	31 (55.3%)	14 (25.0%)	

X^2 = 2.04, df = 4, p>0.05

Subjects whose systolic blood pressure within 140-149 mmHg, 45.4%, 58.0% and 35.7% showed 700-800, 801-900 and 900+ mg/dl of total serum lipids respectively. This variation of total lipids with systolic blood pressure was not significant.

Table 28 shows the level of serum total lipids along with diastolic blood pressure. Majority of the subjects (58.9%) showed the level serum total lipids within 801-900 mg/dl. At diastolic blood pressure 90-104 mmHg, 51.5% and 50.0% of the subjects had serum

Table 28 **Diastolic Blood pressure by serum total lipids**

Diastolic BP mmHg	Total lipids (mg/dl)			
	700-800	800-900	901 +	Total
85-89	2 (22.2%)	4 (12.1%)	1 (7.1%)	7 (12.5%)
90-104	3 (33.3%)	17 (51.5%)	7 (50.0%)	27 (48.2%)
105-109	1 (11.1%)	3 (9.0%)	3 (21.4%)	7 (12.5%)
≤ 110	3 (33.3%)	9 (27.2%)	3 (21.4%)	15 (26.7%)
Total	9 (16.0%)	33 (58.9%)	14 (25.0%)	

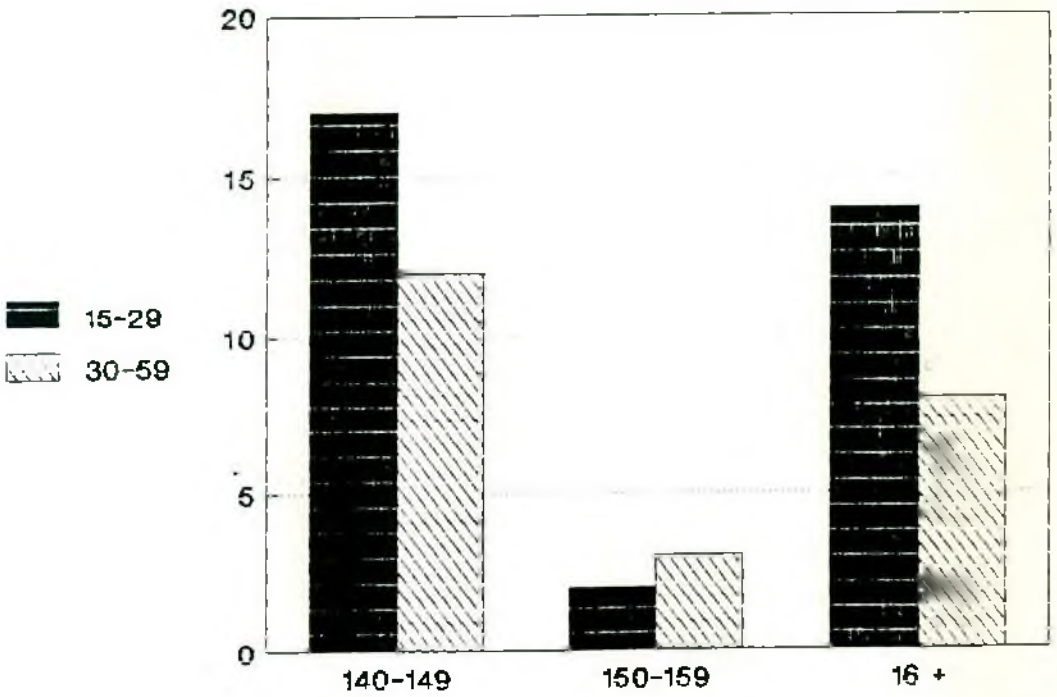
χ^2 = 0.3 , df = 6, p>0.05

total lipids at 801-900 and 901 mg/dl respectively. Hypertensive subjects with diastolic blood pressure 110 mg, only 21.9% showed highest level of (901 + mg/dl) serum total lipids level. Different levels of total lipids with diastolic pressure could not show any statistical significant.

3.13 SERUM UREA LEVEL AND BLOOD PRESSURE

Figure 3.8 shows the serum urea level and systolic blood pressure of the subjects under study. Majority of the subjects (51.5%, 52.2%) serum at 15-59 mg/dl of urea level had systolic blood pressure 140-149 mmHg. 58.9% of the subjects serum urea level was within 15-29 mg/dl. Subjects with raised blood pressure (160 + mmHg) only 42.4% and 34.7% had 15-29 and 30-39 mg/dl of serum urea level. This features of variation of serum urea level were not statistically significant.

FIGURE 3.8 Systolic Blood pressure by serum urea level



The different levels of serum urea in both normotensive and hypertensive in relevance to their diastolic blood pressure is depicted in the table 29. It has been observed that majority of the subjects (52%) with diastolic blood pressure 90-104 mmHg had serum urea level at 30-39 mg/dl.

Table-29 **Diastolic Blood Pressure by serum urea level**

Diastolic BP mmHg	Urea mg/dl		
	15-29	30-59	Total
85-89	3 (9.0%)	4 (17.3%)	7 (12.5%)
90-104	15 (45.4%)	12 (52.1%)	27 (48.2%)
105-109	6 (18.1%)	1 (4.3%)	7 (12.5%)
$\leq$ 110	9 (27.2%)	6 (26.0%)	15 (26.7%)
Total	33 (58.9%)	23 (41.0%)	

X^2 = 2.92, df = 3, p>0.05

At diastolic blood 85-89 mmHg only 9.0% had serum urea level at 15-29 mg/dl. Hypertensive subjects with blood pressure 110 mmHg, their serum urea levels were 27.2% at 15-29 mg/dl and 26.0% at 30-59 mg/dl. However, this variation of serum urea levels was not statistically significant.

3.14 SERUM CREATININE LEVEL AND BLOOD PRESSURE

Majority of subject's (51.7%) creatinine level (Table 30) of 0.5 to 1.4 mg/dl fell in the systolic pressure 140-149 mmHg. Of them 52.3% at 1-1.4 mg/dl and 50.0% at 0.5-0.9 mg/dl respectively.

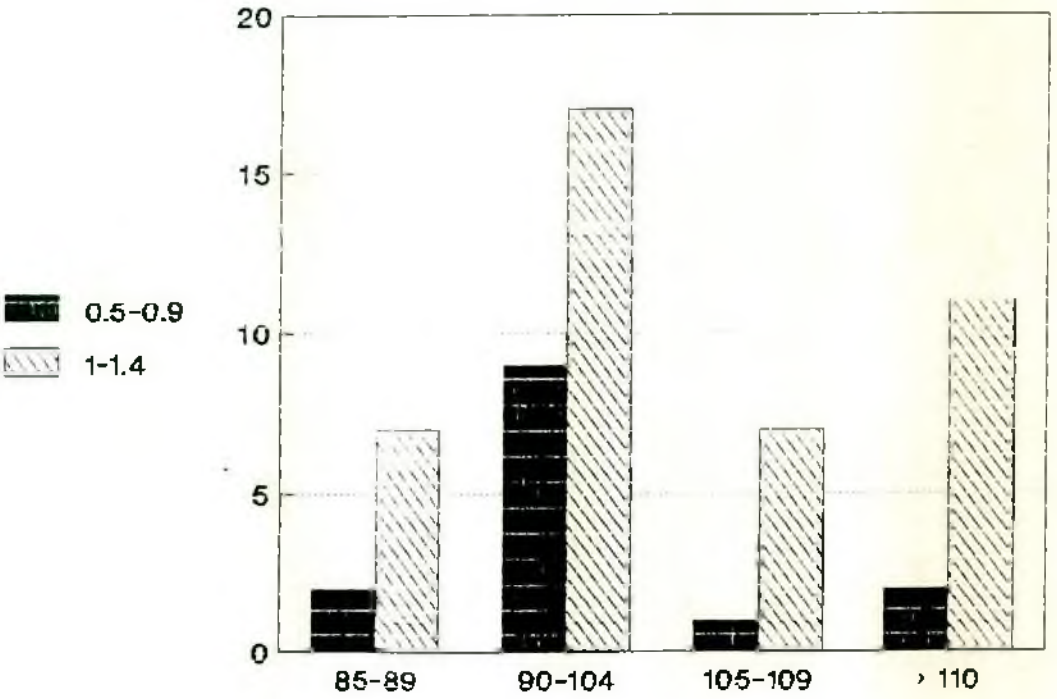
Table-30 **Systolic Blood Pressure by serum creatinine level**

Systolic BP mmHg	Creatinine mg/dl		
	0.5-0.9	1-1.4	Total
140-149	7 (50.0%)	22 (52.3%)	29 (51.7%)
150-159	2 (14.2%)	2 (4.7%)	4 (7.1%)
160+	5 (35.7%)	18 (42.8%)	23 (41.0%)
Total	14 (25.0%)	42 (75.0%)	

41.0% of the hypertensive subjects (systolic blood pressure 160 + mmHg) whose creatinine level were at the level ranging from 0.5 to 1.4 mg/dl. With the same range of creatinine level and systolic blood pressure 160 mmHg equal or above, the proportion of subjects were 35.7% and 42.8% respectively.

Figure 3.9 shows the serum creatinine level and diastolic blood pressure of the subjects. 46.4% of the subject at range of 0.5 to 1.4 mg/dl of serum creatinine had diastolic blood pressure 90-104 mmHg. Of them 64.2% at the level of 0.5 to 0.9 and 40.4% at the level of 1-1.14 mg/dl of serum creatinine level. 23.2% of the subjects had diastolic blood pressure 110 mmHg within the above range of serum creatinine.

FIGURE 3.9 Diastolic Blood pressure by serum creatinine level



3.15 FASTING PLASMA GLUCOSE AND BLOOD PRESSURE

Table 31 shows the subject's fasting plasma glucose and their systolic blood at various level. As ascertained from the table that 50.0% and 41.0% of the subjects had 140-149 and equal to and greater than 160 mmHg systolic pressure respectively.

Table-31 **Systolic Blood Pressure by Fasting plasma glucose**

Systolic BP mmHg	Fasting plasma glucose mg/dl			
	80-89	100-119	120 +	Total
140-149	18 (51.4%)	10 (55.5%)	- 0	28 (50.0%)
150-159	3 (8.5%)	2 (11.1%)	- 0	5 (8.9%)
160+	14 (40.0%)	6 (33,3%)	3 (5.3%)	23 (41.0%)
Total	35 (62.5%)	18 (32.1%)	3 (5.37)	

Only 5.3% with high systolic blood pressure (160+) had equal to and greater than 120 mg/dl of fasting plasma glucose. Majority of the subjects with blood pressure 140-149 mmHg showed fasting plasma glucose level in between 80-119 mg/dl and majority of the subjects (62.5%) had serum fasting plasma glucose within 11.1% and 33.3% of the subjects had 100-119 mg/dl of plasma glucose at 150 to 160 mmHg of systolic pressure.

Table 32 shows the diastolic blood pressure and fasting plasma glucose. As shown in this table 46.4% of the subjects show 90-104 mmHg diastolic pressure. Diastolic pressure from 90 to 110 mmHg only 33.3% of the subjects show fasting plasma glucose level equal to or greater than 120 mg/dl.

Table-32 **Diastolic Blood Pressure by Fasting plasma glucose**

Diastolic BP mmHg	Fasting plasma glucose mg/dl			
	80-99	100-119	120 +	Total
85-89	6 (16.6%)	1 (5.8%)	- 0	7 (12.5%)
90-104	15 (41.6%)	10 (58.8%)	1 (33.3%)	26 (46.4%)
105-109	4 (11.1%)	2 (11.7%)	1 (33.3%)	7 (12.5%)
≤ 110	11 (30.5%)	4 (23.5%)	1 (33.3%)	16 (28.5%)
Total	36 (64.2%)	17 (30.3%)	3 (5.35)	

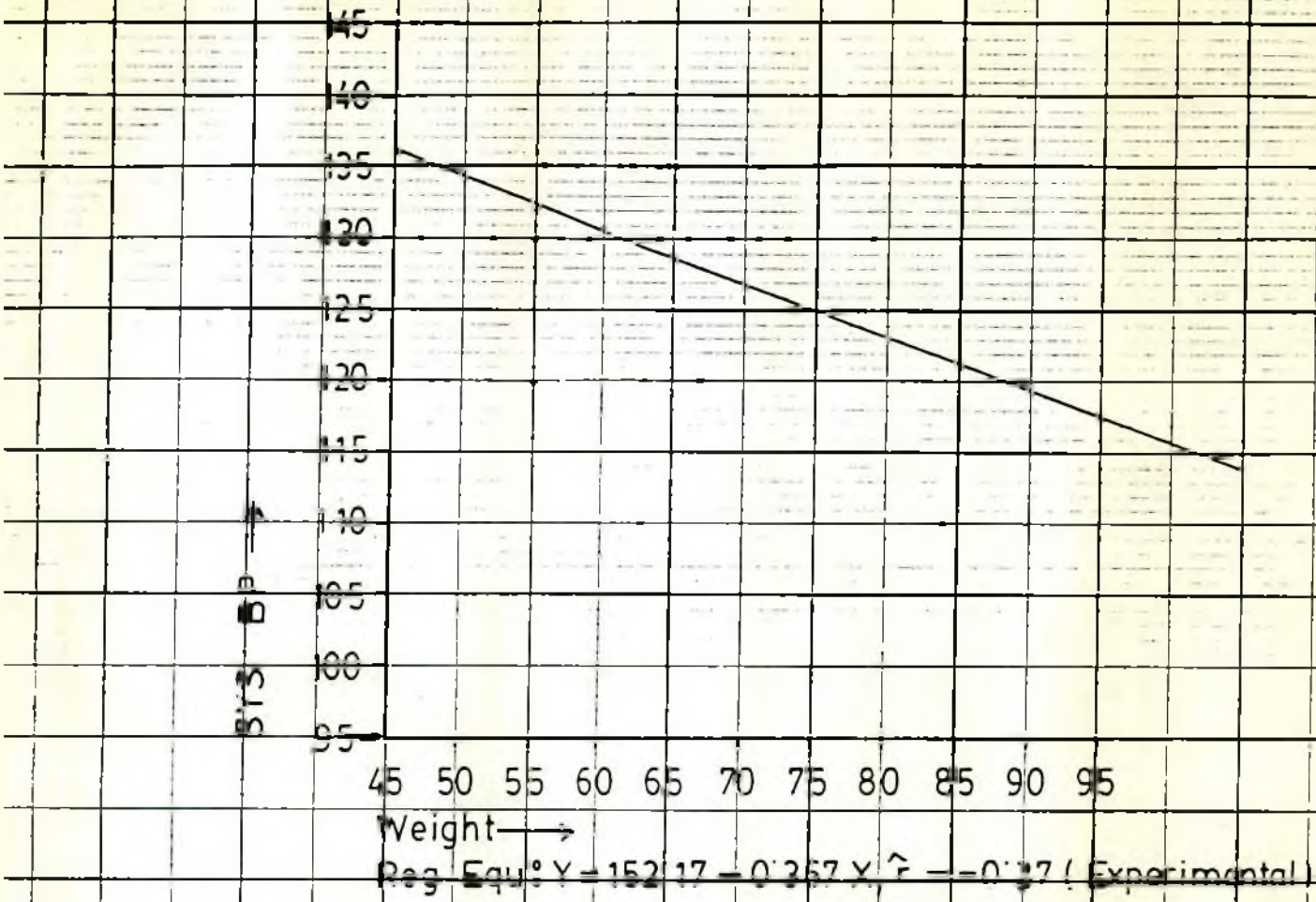
41.6% and 58.8% at 90-104 mmHg diastolic blood pressure had 80 to 119 mg/dl of fasting plasma glucose. At high diastolic blood pressure, 30.5% and 23.5% of the hypertensive subjects show fasting plasma glucose level within 80-99 and 100-110 mg/dl respectively.

3.16 WEIGHT REDUCTION IN PARTICIPANTS

Table 33 presents the difference of weight reduction in experimental and control subjects. The weight loss was ranged from 3kg to 1 kg in experimental and 2 kg to 1 kg in control groups respectively. The mean weight loss was 1.66 kg (0.70) and 1.50 (0.50) kg in hypertensive and normotensive subjects. In both the groups, weight reduction was highly significant ($p < .001$). Figure 3a shows the relation between systolic blood pressure and weight reduction in this scatter diagram. Here final readings of systolic blood pressure were plotted against the final readings of reduced weight. A regression line was drawn in the graph. In the graph there appears to be a downward trend of blood pressure with little increases of body weight in hypertensive ($r = -0.27$) whereas a slight upward trend of systolic blood pressure is noticed (in high normal BP) with the increases of body weight. A moderate degree of positive correlation was noted ($r=0.302$). Fall of systolic blood pressure was slightly correlated with short duration of study. A strong correlation may be observed with weight loss and fall of blood pressure with longer duration of study. Figure 3.b shows the relationship between diastolic blood pressure and and weight reduction in this scatter diagram. Final readings of diastolic blood pressure were plotted against the final readings of reduced weight and lastly a regression line was fitted in the graph. In the diagram, there appears to be a slightly

downward trend of blood pressure with little increases of body weight. The regression equation for experimental is Y = 91.53 - 0.142 x and for control Y = 84.43 - 0.0115 x. The co-efficient of correlation(r) shows slight relationship between the weight loss and fall of diastolic blood pressure and the results showed moderate degree of negative correlation, where estimated r= -0.169 for experimental and r = -0.06 for control groups respectively.

FIGURE 3.a. Relation between changes in systolic BP and Body wt (Kg).



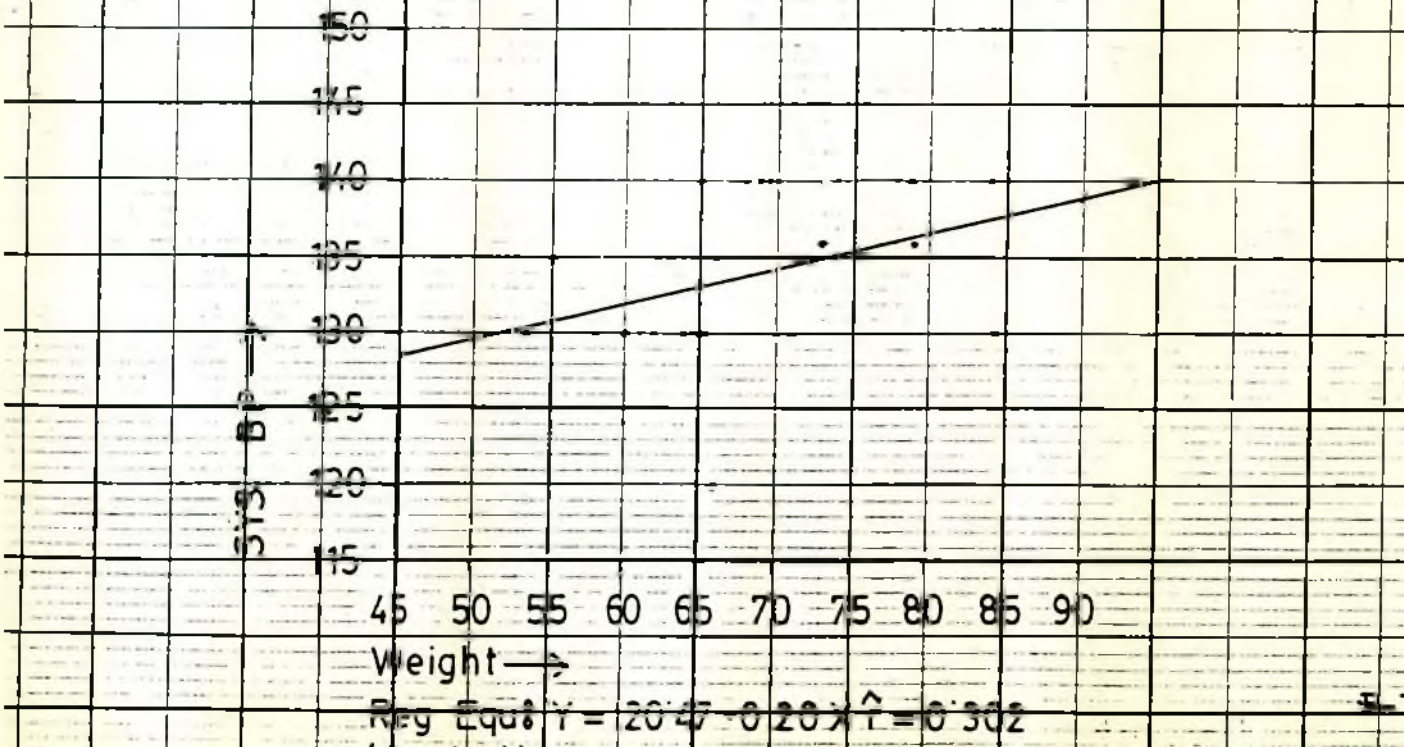
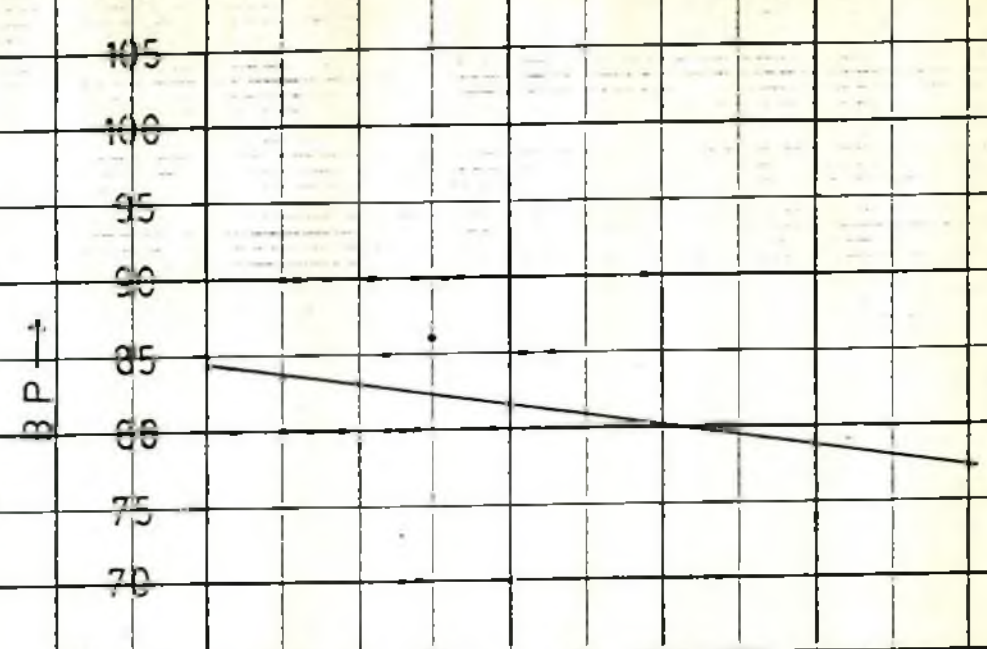
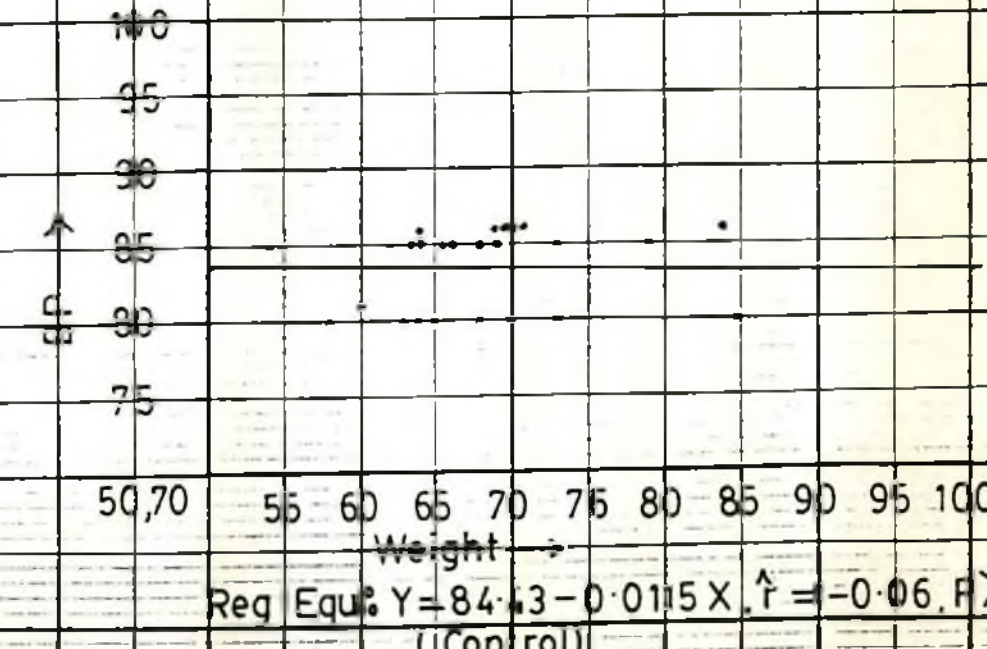


FIGURE 3.b Relation between changes in Diastolic BP (Y) and body weight (X).



Reg Equ: $Y = 91.53 - 0.142X$, $\hat{r} = -0.169$, $P > 0.05$

(Experimental)



Reg Equ: $Y = 84.43 - 0.0115X$, $\hat{r} = -0.06$, $P > 0.05$

(Control)

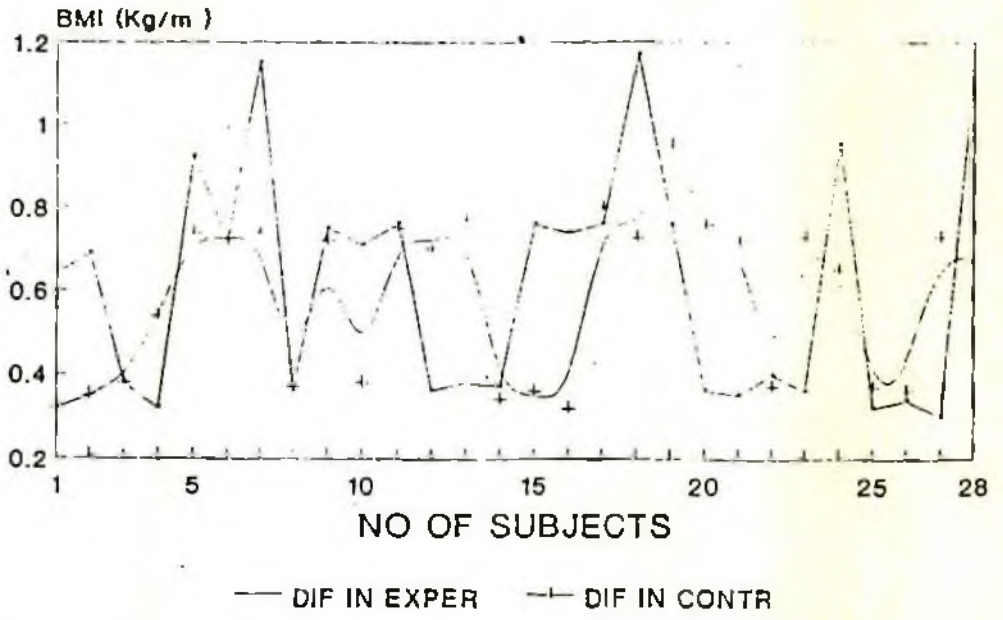
3.17 CHANGES IN BMI AND PBMI

Figure 3.10 presents the difference of BMI in experimental and control subjects. The changes of BMI was ranged from 1.17 kg/m^2 to 0.32 kg/m^2 and 0.95 kg/m^2 to 0.32 kg/m^2 in hypertensive and normotensive participants. The mean fall of BMI was 0.61 kg/m^2 (0.28) and 0.59 kg/m^2 (0.21) in experimental and control groups and their standard error was 0.05 in experimental and 0.03 in control. In both the groups the fall of BMI was highly significant ($p<.001$). A 95% of the confidence limit indicates a similar difference in experimental and control subjects.

Figure 3.11 gives the change of PBMI in hypertensive and normotensive individual. The overall changes of PBMI was renged from 5.29 to 1.45 in experimental and 4.27 to 1.45 in control subjects respectively. The difference of PBMI was 2.77 (1.26) with standard error 0.23 in experimental and 2.69 (0.95) with standard error 0.17 in control. In both hypertensive and normotensive controls the mean difference of PBMI differed significantly ($p<.001$). In PBMI, a 95% of the confidence interval shows no appreciable difference in both the groups.

FIGURE 3.10. CHANGES IN BMI

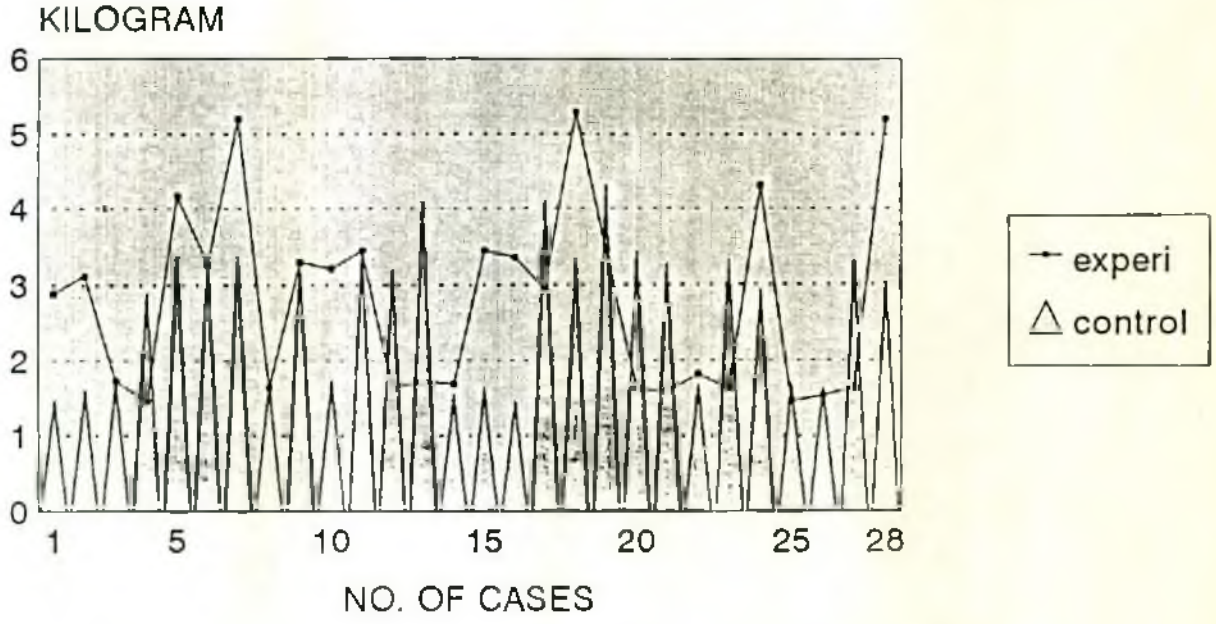
95% CI.FOR EXP(0.51-0.71) CONT(0.51-0.67



$\bar{d}e$=0.51,Ie=11.50,sde=0.28,p<0.001
$\bar{d}c$=0.59,Ic=14.85,sdc=0.27,p<0.001

FIGURE-3.11. CHANGES IN PBMI (kg)

experi= = $\bar{d}$=2.77, t= 11.62
control= =$\bar{d}$=2.29, t= 14.47



experi= = SD= ± 1.26 (p< 0.001), 95% CI= 2.29 to 3.25
control= SD= ± 0.95 (p< 0.001), 95% CI= 2.33 to 3.05

3.18 CHANGES IN SYSTOLIC AND DIASTOLIC BLOOD PRESSURE

Table - 34 & Figure 3.12 give the changes in systolic and diastolic blood pressure in experimental and control. The changes in systolic blood pressure were ranged from 60 to 10 mmHg and 10 to 0 mmHg in experimental and control subjects. The mean difference i.e. mean drop of systolic blood pressure in both the groups was 32.82 and 3.53 mmHg respectively. These difference in drop of systolic blood pressure was statistically highly significant ($p < .001$). The standard deviation and standard error for experimental was 13.66 and 2.58 and for control group, 4.22 and 0.79. The fall of systolic blood pressure in experimental group with 95% confidence interval lies within 27.53 to 38.11 mmHg whereas in control it was within 1.90 to 5.16 mmHg. Thus a large difference in hypertensive group and a very little difference in normotensive group was observed.

Figure 3.12 shows the difference of diastolic pressure in experimental and control groups from their initial and final readings. For experimental group the range of this difference was 40 to 14 mmHg and for control 10 to 4 mmHg. A mean drop of 24.75 mmHg in diastolic pressure in experimental group and a mean drop of 5.35 mmHg in control group were achieved.

In both the trial groups the mean drop in diastolic blood pressure was statistically highly significant ($p < .001$). The standard deviation and standard error in experimental subjects were 13.66 and 2.58 and in control group, 4.22 and 0.79. A 95% of confidence interval for the true difference in the fall of diastolic blood pressure of the two groups was 27.53 to 38.11 mmHg and 1.90 to 5.16 mmHg. Therefore in experimental i.e. in hypertensive group, there may be a rather large difference in the fall of diastolic blood pressure. In the normotensive group there was very little difference in the fall of blood pressure.

Regular exercise has been observed to produce a significant fall in blood pressure in mild hypertension. It has been noted that a fall of 13/12 mmHg in a group of exercised hypertensive men compared to a 0/6 mmHg fall in normotensive control subjects[127]. It has been further suggested that the fall obtained after exercise was maintained for a period of 4-6 hours. In a controlled study of men with uncomplicated essential hypertension (mean standing blood pressure 165/109 mmHg) treadmill exercise was carried out until heart rates of 120 beats/min were obtained and maintained for 5-10 minute periods separated by 3 minutes rest. During exercise the blood pressure rose slightly (175 mmHg systolic) but dropped by approximately 25% after. This fall was maintained for upto 10 hours. The results implied that even " a good walk " twice a day

might be reasonable treatment for mild hypertension. It may be stated that the haemodynamic response to acute dynamic exercise is probably a fall in systemic vascular resistance that reflects a marked vasodilatation of the resistance vessels in the exercising muscle. During sustained dynamic exercise, steletal muscle metabolism is primarily aerobic. Therefore, it is appropriate that the increased demand for ATP generation and utilization is met by a correspondingly large increase in oxygen supply. With acute dynamic exercise, the most striking cardiovascular alternation is an increase in heart rate. The increases in cardiac output and stroke volume are paralleled by a marked increase in systolic blood pressure. Diastolic blood pressure remains unchanged and may even fall slightly[35]. Therefore, mean arterial pressure increase only moderately, reflecting the significant decrease in peripheral vascular resistance.

Table-34 Changes in Systolic Blood Pressure (mmHg) in experimental & control

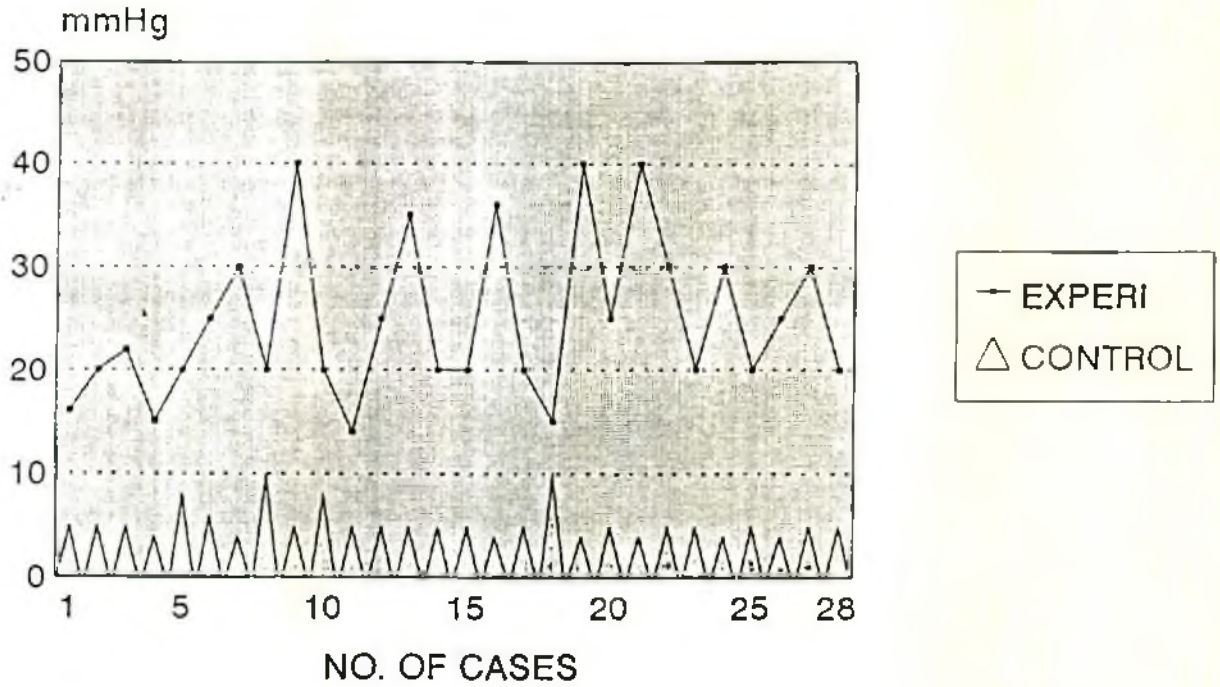
	Experimental			Control		
Subject	Initial	Final	Difference	Initial	Final	Difference
1	160	130	+ 30	140	140	0
2	150	140	+ 10	140	135	+ 5
3	160	130	+ 30	140	140	0
4	145	130	+ 15	140	130	+10
5	160	140	+ 20	140	135	+ 5
6	160	130	+ 30	140	136	+ 4
7	160	130	+ 30	136	130	+ 6
8	160	140	+ 20	140	140	0
9	190	140	+ 50	130	130	0
10	160	140	+ 20	135	135	0
11	170	130	+ 40	130	130	0
12	160	120	+ 40	140	140	0
13	154	130	+ 24	130	130	0
14	160	120	+ 40	120	120	0
15	160	140	+ 20	140	130	+ 10
16	160	100	+ 60	140	136	+ 4
17	160	140	+ 20	136	136	0
18	160	130	+ 30	140	130	+ 10
19	170	110	+ 60	140	140	0
20	190	140	+ 50	140	140	0
21	170	110	+ 60	138	136	+ 2
22	160	130	+ 30	138	135	+ 3
23	140	120	+ 20	140	130	+ 10
24	160	120	+ 40	140	140	0
25	160	130	+ 30	140	130	+ 10
26	170	140	+ 30	140	130	+ 10
27	160	120	+ 40	140	140	0
28	160	130	+ 30	140	130	+ 10

d= 32.82 t = 12.70
SD= ± 13.66 p< .001
SE = 2.58
95% CI = 27.53 to 38.11

d = 3.53 t = 4.42
SD = ± 4.22 p<0.05
SE = 0.79
95% CI= 1.90 to 5.16

FIG-3.12. CHANGES IN DIASTOLIC BLOOD PRESSURE (mmHg)

experi= = $\bar{d}$=24.75, t=16.74
control= $\bar{d}$= 5.35, t=17.04



experi= = SD= ± 7.82 (p< 0.001), 95% CI= 21.72 to 27.78
control= SD= + 1.66 (p< 0.001), 95% CI= 4.71 to 5.99

3.19 CHANGES IN SERUM ELECTROLYTES

Table-35 shows the change of serum sodium level from before and after program intervention in hypertensive and normotensive groups. The changes of serum sodium level were ranged from 11 mmol/L to 1 mmol/L in hypertensive and 7 mmol/L to 1 mmol/L in normotensive. The mean changes in serum sodium was 1.40 mmol/L and 0.82 mmol/L with standard deviation 3.08 and 2.38 and their standard error 0.58 and 0.44 in experimental and control subjects respectively. A 95% of confidence interval was 0.30 to 0.70 in experimental and 0.11 to 0.43 in control. The changes in serum sodium were significant ($p<0.05$) in hypertensive subjects only. Figure 3.C shows the relationship between the differences i.e. changes in diastolic blood pressure and serum sodium level in both hypertensive and normotensive subjects. The differences of diastolic blood pressure were plotted against the differences of serum sodium level in the scatter diagram. In the Graph a regression line was fitted, basing on the scatter diagram. In this diagram there appears to be a slightly upward trend of diastolic blood pressure with little increases of serum sodium level. The regression equation for the hypertensive group is $Y = 23.81 + 0.29 x$ and for normotensive $Y = 5.306 + 0.1046 x$. The estimated co-efficient of correlation (r) shows the moderate degree of positive correlation. Here $r = 0.39$ for hypertensive and $r = 0.158$ for normotensive were calculated.

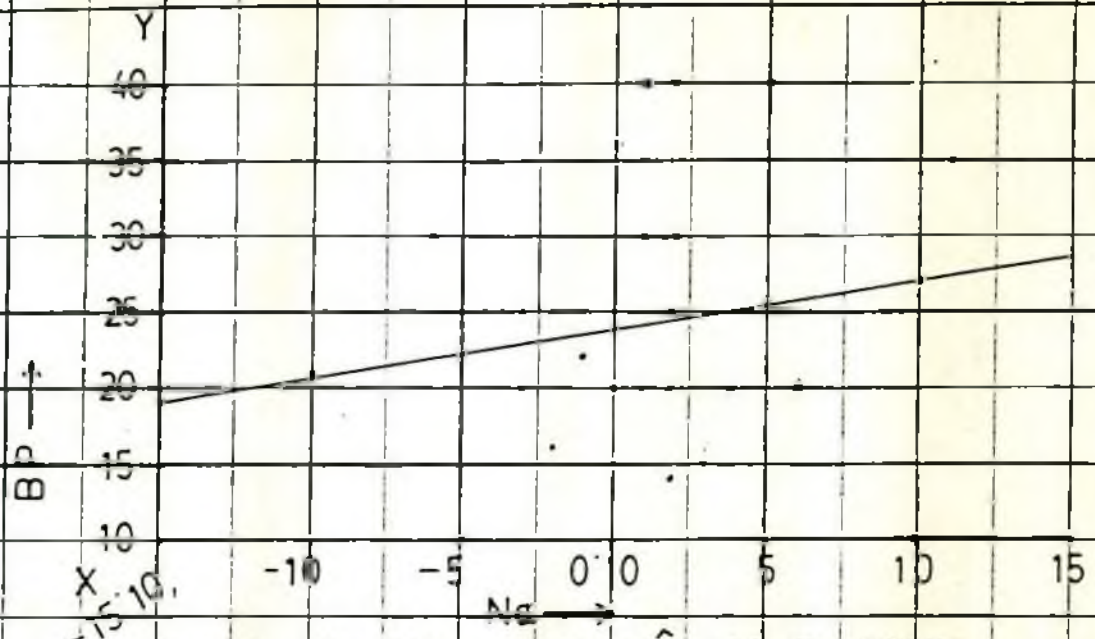
Table-35 Serum Sodium (mmol/L)level of experimental and control at initial and final measurement

	Experimental			Control		
Subject	Initial	Final	Difference	Initial	Final	Difference
1	139	141	+ 2	141	139	+ 2
2	142	142	0	142	142	0
3	140	141	-1	140	141	-1
4	141	141	0	141	141	0
5	141	137	+ 4	141	137	+ 4
6	140	143	-3	140	142	-2
7	137	143	-6	138	140	-2
8	141	135	+ 6	141	140	+ 1
9	141	136	+ 5	140	139	+ 1
10	140	138	+ 2	136	134	+ 2
11	144	142	+ 2	141	140	+ 1
12	140	138	+ 2	138	136	+ 2
13	140	129	+ 11	136	135	+ 1
14	137	140	-3	143	140	+ 3
15	143	142	+ 1	141	140	+ 1
16	140	140	0	135	142	-7
17	142	140	+ 2	136	135	+ 1
18	138	135	+ 3	141	139	+ 2
19	135	134	+ 1	142	140	+ 2
20	144	142	+ 2	135	140	-5
21	135	133	+ 2	139	137	+ 2
22	140	138.30	+ 1.70	145	144	+ 1
23	142	140	+ 2	142	140	+ 2
24	141	140	+ 1	137	133	+ 4
25	138	137	+ 1	135	133	+ 2
26	141	139	+ 2	144	140	+ 4
27	137	135	+ 2	139	138	+ 1
28	142	140	+ 2	142	141	+ 1

d= 1.41 t = 2.41
SD= ± 3.08 p<0.05
SE = 0.58
95% CI = 0.22 to 2.60

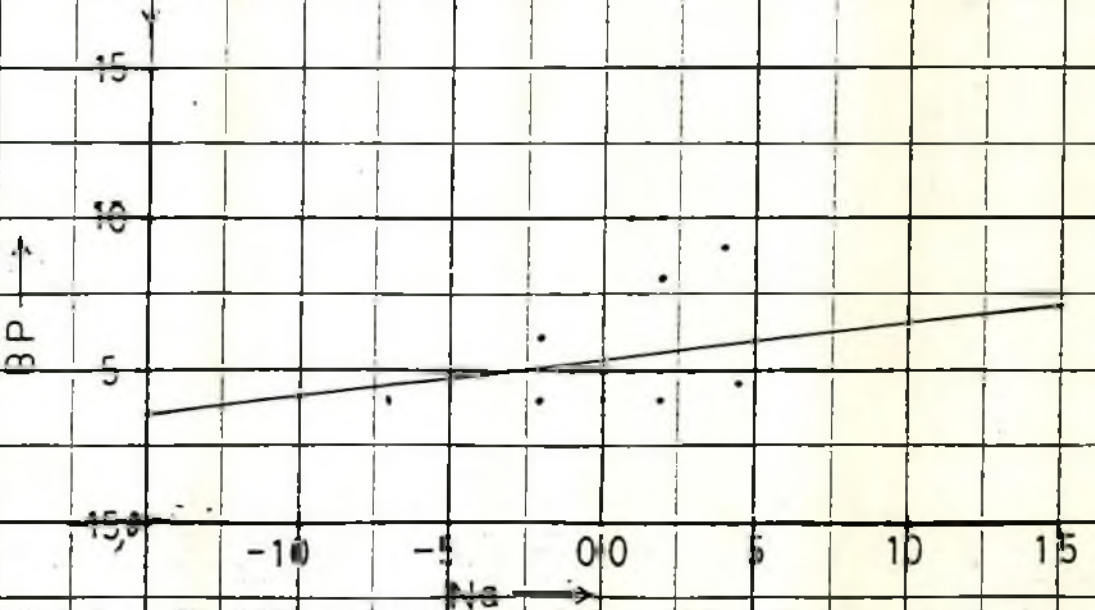
d = 0.82 t = 1.85
SD = ± 2.38 p>0.05
SE = 0.44
95% CI= -0.1 to +1.74

FIGURE 3.c. Relation between changes in serum Na and diastolic BP.



Reg Equ Y = 238 + 0·2946 X $\hat{\gamma}$ = ·39, P < 0·05

(Experimental)



Reg Equ: Y = 5·305 + 0·1046 X, $\hat{\gamma}$ = 0·158, P > 0·05

(Control)

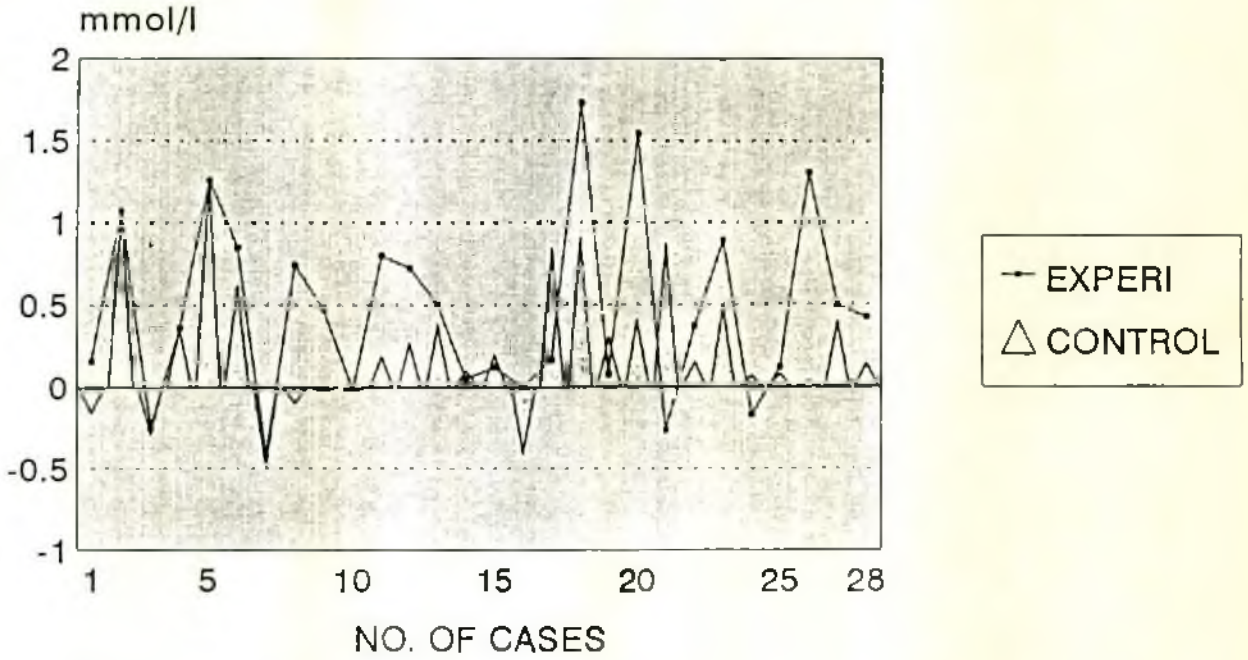
P-20I

Figure 3.13 gives the difference of serum potassium levels from their baseline and final measurement in hypertensive and normotensive subjects. The changes of serum potassium level were ranged from 0-1.73 mmol/L in experimental and 1.07 mmol/L to 0.04 mmol/L in control subjects. The average changes in serum potassium was 0.50 mmol/L and 0.27 mmol/L with standard deviation 0.52 and 0.43 and their standard error 0.09 and 0.08 in hypertensive and normotensive subjects. The mean changes of serum potassium was significant in experimental group only ($p<0.05$). A 95% of confidence interval was 0.03 to 0.70 in experimental and 0.11 to 0.43 in control.

Figure 3.d depicts the relationship between the changes in diastolic blood pressure and serum potassium level in both experimental and control groups. The individual changes of diastolic blood pressure were plotted against the individual changes of serum potassium level as shown in the scatter diagram. A regression line was fitted. From the graph it appears that there is a upward trend of diastolic blood pressure with increase of serum potassium level in experimental group whereas a downward trend i.e. a reverse situation is noticed in control group. The regression equation was $Y = 26.71 + 4.29 x$ for experimental and $Y = 5.08 - 0.97 x$ for control. The co-efficient of correlation shows the limited degree of positive correlation ($r = 0.31$) exist in experimental group and a limited degree of negative correlation exists in control subject ($r = - 0.25$).

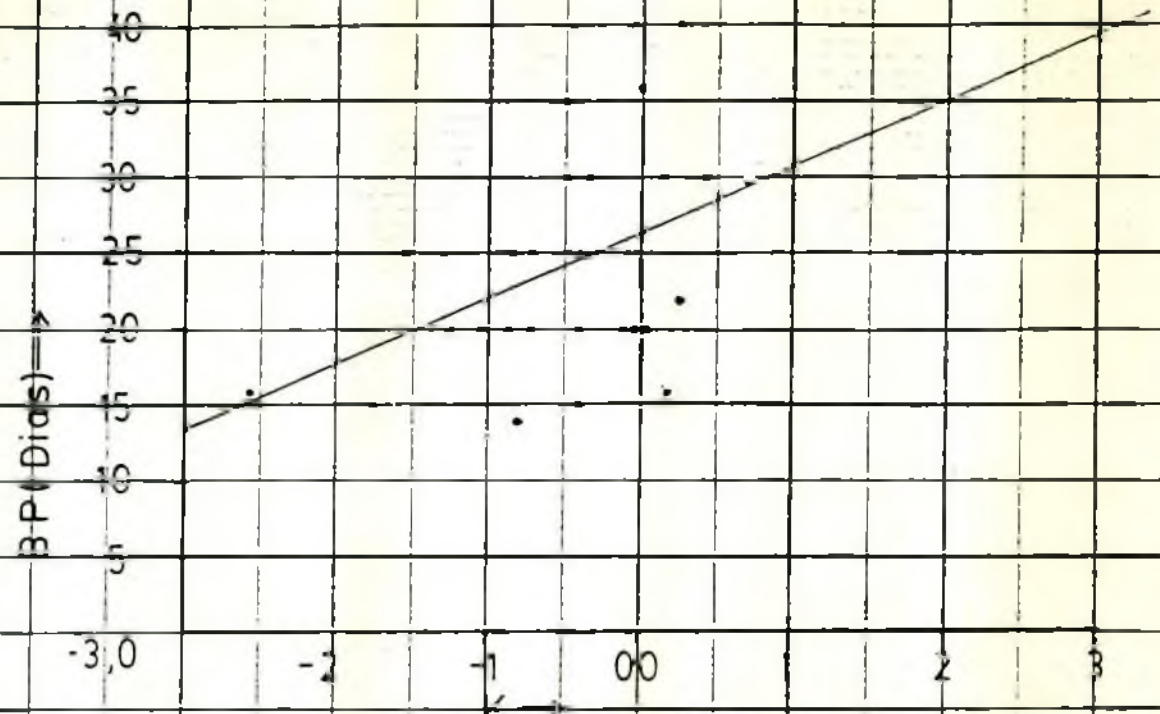
FIG-3.13. CHANGES IN SERUM POTASSIUM LEVEL

experi == $\bar{d}$= 0.50 t= 5.08
control== $\bar{d}$= 0.27, t= 3.30



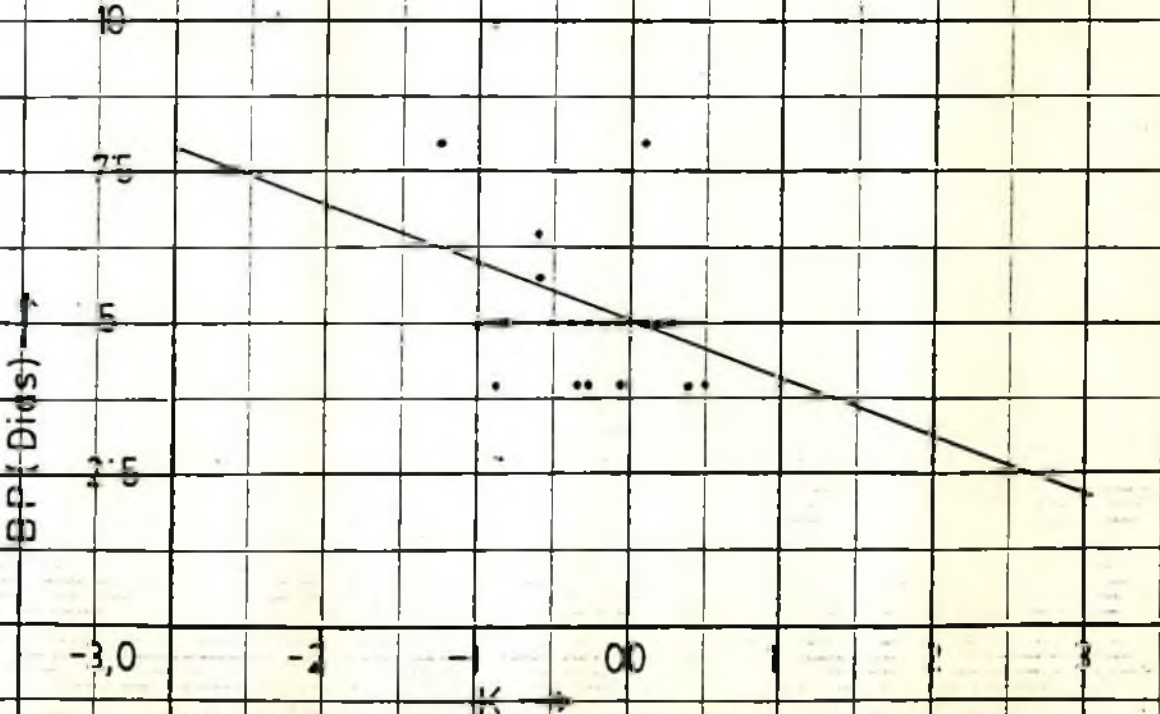
experi== SD= ±0.52 (p< 0.001), 95% CI= 0.30 to 0.70
control==SD= ±0.43 (p< 0.05), 95% CI= 0.11 to 0.43

FIGURE 3.4: Relation between change in serum K and diastolic BP.



Reg Equ: $Y = 26.71 + 4.29X$; $\hat{r} = 0.31$, $P > 0.05$

(Experimental)



Reg Equ: $Y = 5.08 - 0.97X$; $\hat{r} = -0.25$, $P > 0.05$

(Control)

Table 36 presents the difference of serum chloride level i.e. in the undissociated form of Nacl in blood at basline and after programme intervention in hypertensive and normotensive groups. The range of difference of chloride level was 16 mmol/L to 0 mmol/L in experimental and 5 mmol/L to 0 mmol/L in control subjects. The mean difference in serum chloride was 1.91 mmol/L and 0.47 mmol/L with standard deviation 3.72 and 3 and their standard error 0.70 and 0.56 in experimental and control respectively. A 95% of confidence interval shows, a comparatively large difference in hypertensive group (0.47 to 3.35). A significant change of Nacl ($p<0.05$) was found in hypertensive subjects. Figure 3.e shows the relationship between the changes in diastolic blood pressure and serum chloride level in both experimental and control subjects. The subject's changes of diastolic blood pressure were observed against the changes of serum chloride level as shown in the scatter diagram and a regression line was also drawn. From the figure it appears that there is a upward trend of diastolic blood pressure with increases of serum chloride (Nacl) level in hypertensive subjects. On the other hand, a downward trend i.e. a reverse trend is found in control subject. The regression equation is $Y = 23.43 + 0.68x$ for experimental and $Y = 5.39 - 0.09x$ for control. The co-efficient of correlation shows a moderate degree of positive correlation ($r = 0.32$) present in hypertensive and a moderate degree of negative correlation present in normotensive subject ($r = -0.16$).

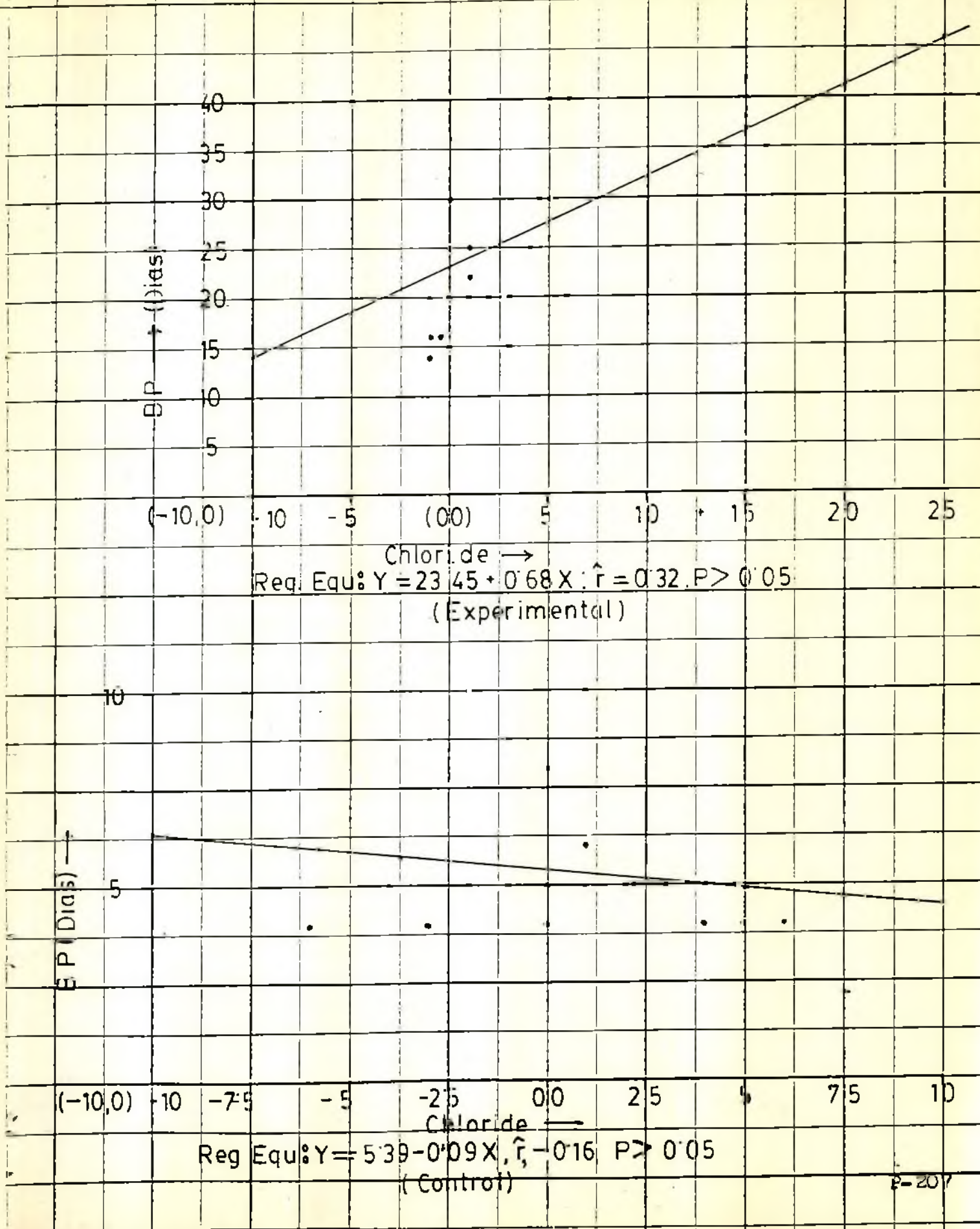
Table-36 Serum chloride (Nacl mmol/L) of experimental and control at initial and final measurement.

	Experimental			Control		
Subject	Initial	Final	Difference	Initial	Final	Difference
1	99	100	-1	99	100	-1
2	102	101	+ 1	102	101	+ 1
3	101	100	+ 1	101	100	+ 1
4	102	102	0	102	102	0
5	100	100	0	100	100	0
6	102	102	0	102	101	+ 1
7	96	101	-5	97	100	-3
8	100	101	-1	103	102	+ 1
9	100	94	+ 6	101	96	+ 5
10	101	95	+ 6	95	96	-1
11	101	102	-1	101	98	+ 3
12	101	97	+ 4	99	95	+ 4
13	106	90	+ 16	98	100	-2
14	100	99	+ 1	102	101	+ 1
15	101	101	0	100	101	-1
16	101	100.85	+ 0.15	96	102	-6
17	102	102	0	97	101	-4
18	99	96	+ 3	100	98	-2
19	96	91	+ 5	101	97	+ 4
20	100	100	0	97	101	-4
21	95	100	-5	98	92	+ 6
22	102.56	98.05	+ 4.51	102	100	+ 2
23	102	100	+ 2	104.22	102	+ 2.22
24	102	102	0	98	93	+ 5
25	100	100	0	94	95	-1
26	102	101	+ 1	101	97	+ 4
27	98	95.11	+ 2.89	98	100	-2
28	101	98	+ 3	101	101	0

d= 1.91 t = 2.71
SD= ± 3.72 p<0.05
SE = 0.70
95% CI = 0.47 to 3.35

d = 0.47 t = 0.82
SD = ± 3 p>0.05
SE = 0.56
95% CI= -0.69 to + 1.63

FIGURE 3.e. Relation between changes in Nacl and diastolic BP.



3.20 CHANGES IN LIPID PROFILES AND BLOOD PRESSURE

Table 37 gives the differences of serum cholesterol level at baseline and after programme intervention in hypertensive and control subjects. The changes of cholesterol levels were ranged from 67 mg/dl to 180 mg/dl in experimental and 47 mg/dl to 3 mg/dl in control. The mean difference in serum cholesterol was 23.23. mg/dl and 15.92 mg/dl with standard deviation 22 and 11.44 and their standard error 4.15 and 2.16 in experimental and control group respectively. The mean changes of serum cholesterol level were highly significant ($p < .001$). A 95% of confidence interval shows a comparatively large gap in hypertensive (14.71 to 31.75) was noted.

Figure 3.f depicts the relationship between the changes in diastolic blood pressure and serum cholesterol level in experimental and control subjects. The individual changes of diastolic blood pressure were plotted against the individual changes of cholesterol level. A regression line was fitted. From the figure it appears that there is a upward trend of diastolic blood pressure with the increase of cholesterol level in both hypertensive and normotensive groups. The regression line is $Y = 22.159 + 0.111x$ for experimental and $Y = 5.007 + 0.0197x$ for control. The co-efficient of correlation shows a limited degree positive correlation exist in experimental ($r = 0.319$) and control ($r = 0.161$).

Table-37 Serum cholesterol (mg/dl) level of experimental and control subjects at initial and final measurement

	Experimental			Control		
Sub	Initial	Final	Difference	Initial	Final	Difference
1	222	217	± 5	230	210	± 20
2	246	200	± 46	247	200	± 47
3	243	202	± 41	243	202	± 41
4	248	245	± 3	248	240	± 8
5	184	180	± 4	188	184	± 4
6	218	200	± 18	218	201	± 17
7	207	188	± 19	210	190	± 20
8	196	190	± 6	245	235	± 10
9	231	202	± 29	230	205	± 25
10	229	201	± 28	187	178	± 9
11	219	152	± 67	225	212	± 13
12	200	194	± 6	229	210	± 19
13	246.32	207.14	± 39.18	218	206	± 12
14	227.55	212.50	± 15.05	186	182	± 4
15	221.43	188	± 33.43	206.89	201	± 5.89
16	306	216	± 90	188	180	± 8
17	238	220	± 18	184	170	± 14
18	236	232	± 4	192	187	± 5
19	181	177	± 4	239	210	± 29
20	205.80	200.52	± 5.28	243	210	± 33
21	288	223	± 65	216	210	± 6
22	238	225	± 13	243	225	± 18
23	222.85	205.22	± 17.63	250	230	± 20
24	257	245.71	± 11.29	226.09	200	± 26.09
25	200	198	± 2	182	180	± 2
26	250	225	± 25	221	210	± 11
27	243.75	220	± 23.75	216	200	± 16
28	217	205	± 12	183	180	± 3

d= 23.23 t = 5.58
SD= ± 22 p<.001
SE = 4.15
95% CI = 14.71 to 31.75

d = 15.92 t = 7.36
SD = ± 11.44 p<.001
SE = 2.16
95% CI = 11.49 to 20.35

FIGURE 3.f. Relation between changes in serum cholesterol and diastolic BP.

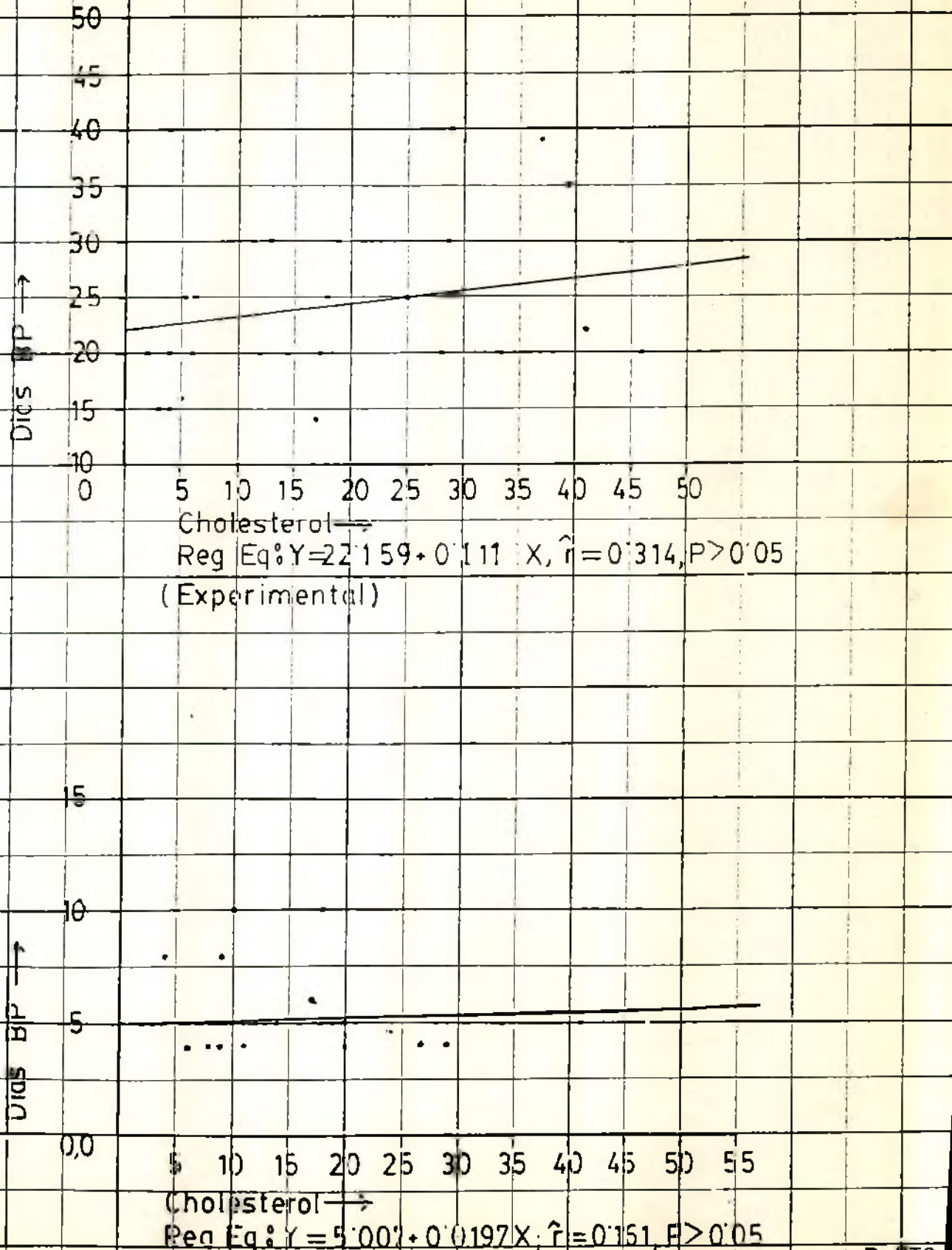


Table 38 shows the change of serum HDL - Cholesterol levels at baseline and after programme intervention in experimental and control subjects. The differences of HDL-cholesterol levels were ranged from 8.40 mg/dl to 0.37 mg/dl in hypertensive and 5 mg/dl to 0.32 mg/dl in control subjects. The mean difference in HDL-cholesterol was 3.49 (3.85) and 3.78 (3.50) and standard error 0.72 and 0.66 in experimental and control groups respectively. The changes of HDL-cholesterol level were highly significant ($p < .001$). A 95% of confidence interval shows almost a similar difference (2 to 4.98 and 2.43 to 5.13) in both the groups.

Figure 3.g presents the relationship between the changes in diastolic blood pressure and HDL-cholesterol level. The changes in HDL- cholesterol level from subject to subject were plotted against the changes of diastolic blood pressure level. Then a regression line was fitted as per the scatter diagram. From the graph it appears that there is a slightly upward trend of diastolic blood pressure with the increases of HDL-cholesterol level. The regression equation was $Y = 26.71 + 0.61x$ for experimental and $Y = 5.5 + 0.05 x$ for control. Co-efficient co-relation shows very little relationship i.e. a limited degree positive correlation exist in experimental ($r = 0.13$) and control subjects ($r = 0.04$).

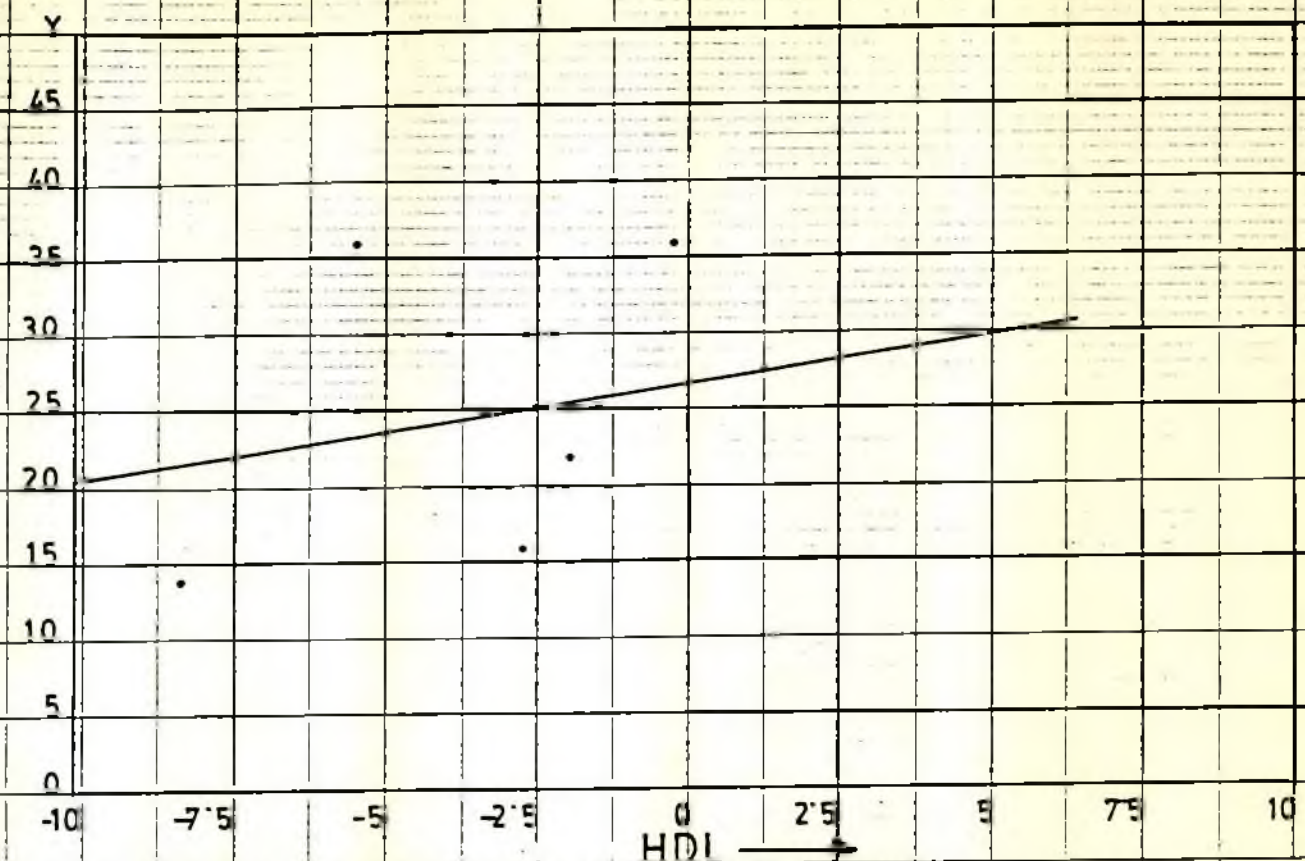
Table-38 HDL-Cholesterol level (mg/dl) of experimental and control at initial and final measurement

	Experimental			Control		
Subject	Initial	Final	Difference	Initial	Final	Difference
1	56.15	59	2.85	54	59	5
2	56	58	2	55	57	2
3	40	60	20	40	60	20
4	56	58	2	56	60	4
5	57	59	2	56.08	59	2.92
6	51.32	57.65	6.33	52.32	58.65	6.33
7	56.99	59.60	2.61	58.90	62.50	3.60
8	59	62	3	60	65	5
9	58	61.37	3.37	59	62	3
10	58	62.38	4.38	59.85	60.50	0.65
11	55	63.40	8.40	60	64	4
12	57.57	60	2.43	57	60	3
13	57.40	57.77	0.37	59	62.70	3.70
14	58.12	56.55	-1.57	58	62	4
15	57.43	59.60	2.17	57.81	61.21	3.40
16	50.50	56	5.50	59.18	59.50	0.32
17	57.61	63	5.39	55	57	2
18	57.60	60	2.40	58.53	60	01.47
19	60.06	56.80	-3.26	56	58	2
20	56.55	62.07	5.52	61.37	62.50	-1.13
21	58.96	60.23	1.27	58	61.78	3.78
22	61	65	4	58	63	5
23	56.55	60	3.45	61	66	5
24	53.80	57.80	4	57.43	60.25	2.82
25	56.55	58.55	2	59.84	60.82	1.08
26	56.55	58	1.45	59	63.07	4.07
27	59.60	62	2.40	57.87	61.78	3.91
28	58.52	62.05	3.53	57	60	3

d= 3.49 t = 4.79
SD= ± 3.85 p<.001
SE = 0.72
95% CI = 2 to 4.98

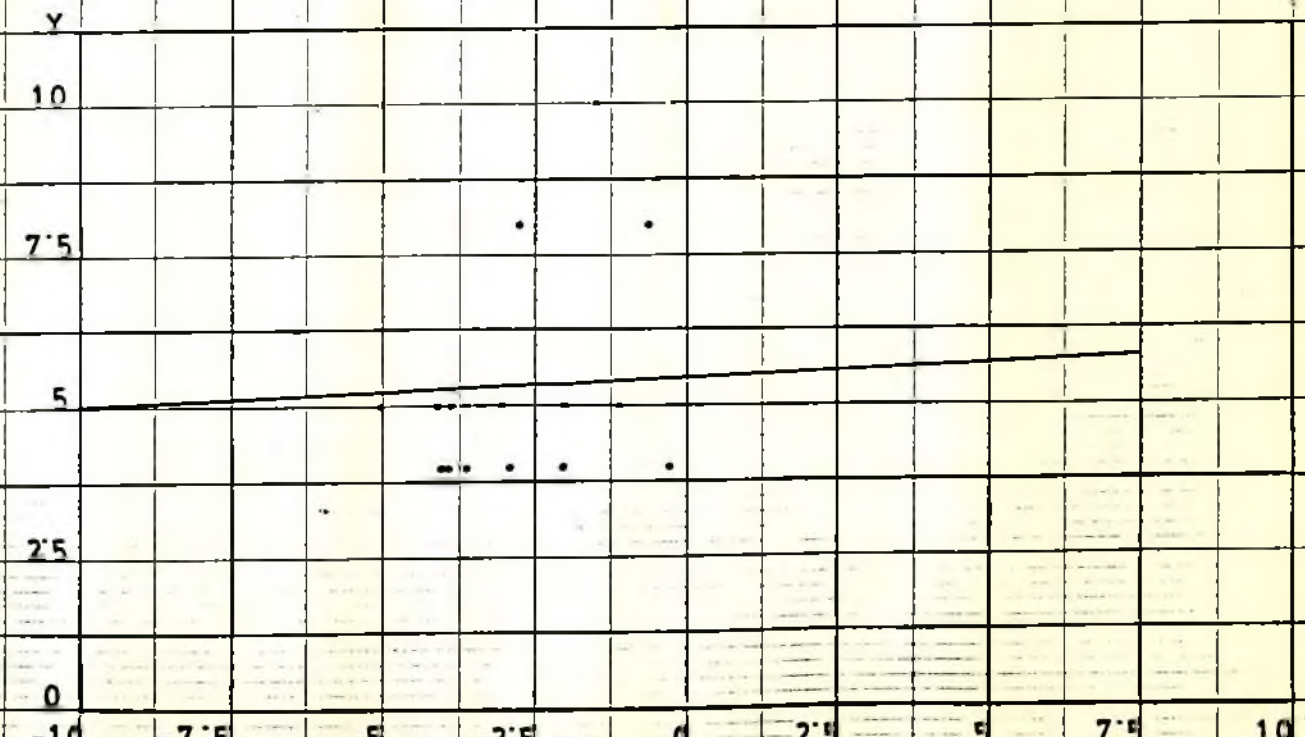
d = 3.78 t = 5.71
SD = ± 3.50 p<.001
SE = 0.66
95% CI = 2.43 to 5.13

FIGURE 3.g. Relation between changes in serum HDL-Cholesterol and diastolic BP.



Reg Eq: $Y = 26{\cdot}71 + 0{\cdot}61X$; $\hat{\delta} = 0{\cdot}13$, $P > 0{\cdot}05$

(Experimental)



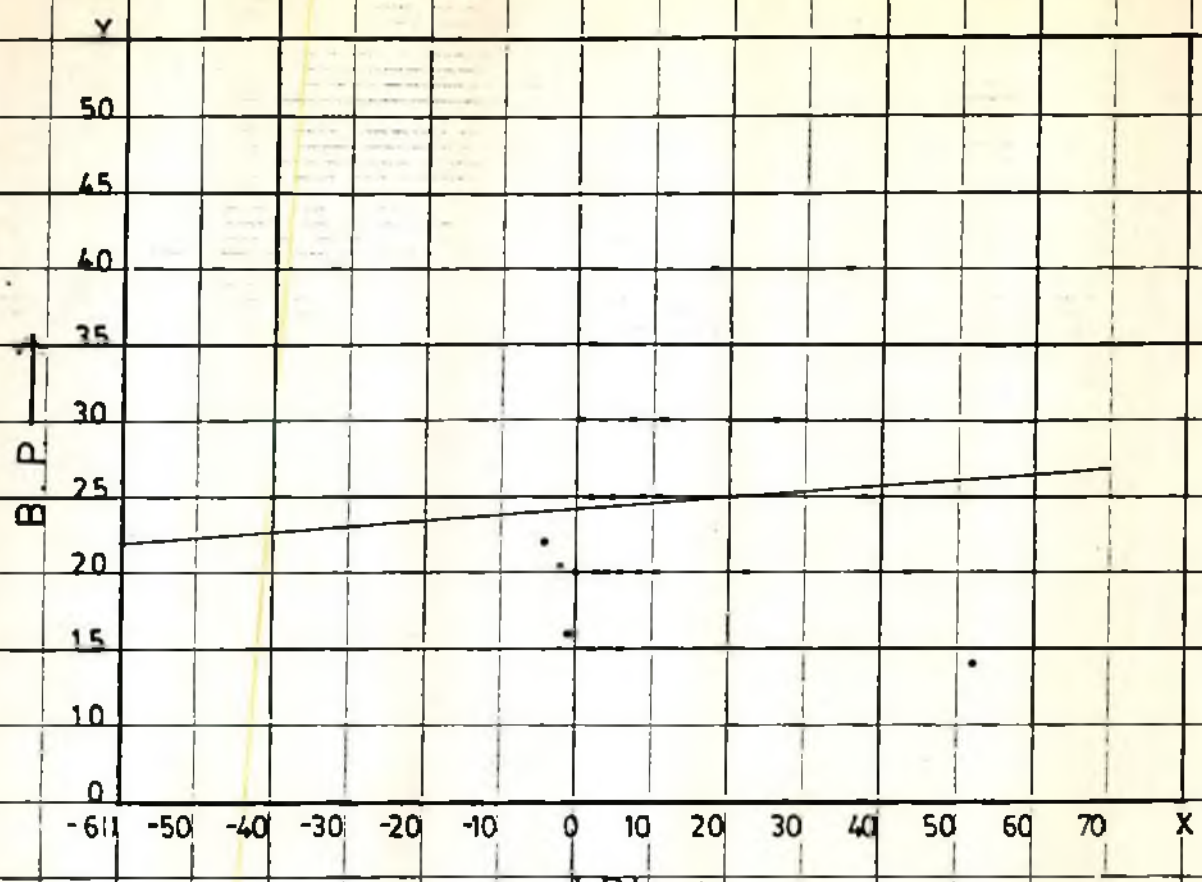
Reg Eq $Y = 5{\cdot}5 + 0{\cdot}05X$, $\hat{\delta} = 0{\cdot}04$, $P > 0{\cdot}05$

(Control)

Figure 3.14 presents the change of serum LDL-cholesterol levels at initial and final measurement in hypertensive and control subjects. From table the differences of LDL-cholesterol level were ranged from 84 mg/dl to 1 mg/dl and 43 mg/dl to 1 mg/dl in experimental and control group. The mean changes in LDL-cholesterol was 12.32 (21.21) and 10.52 (10.66) and standard error 4.00 and 2.01 in experimental and control group respectively. The overall changes i.e. reduction of LDL-cholesterol level were significant ($p < 0.05$ for experiment and $p < .001$ for control). A 95% of confidence interval indicates comparatively a large difference in LDL-cholesterol in hypertensive subjects.

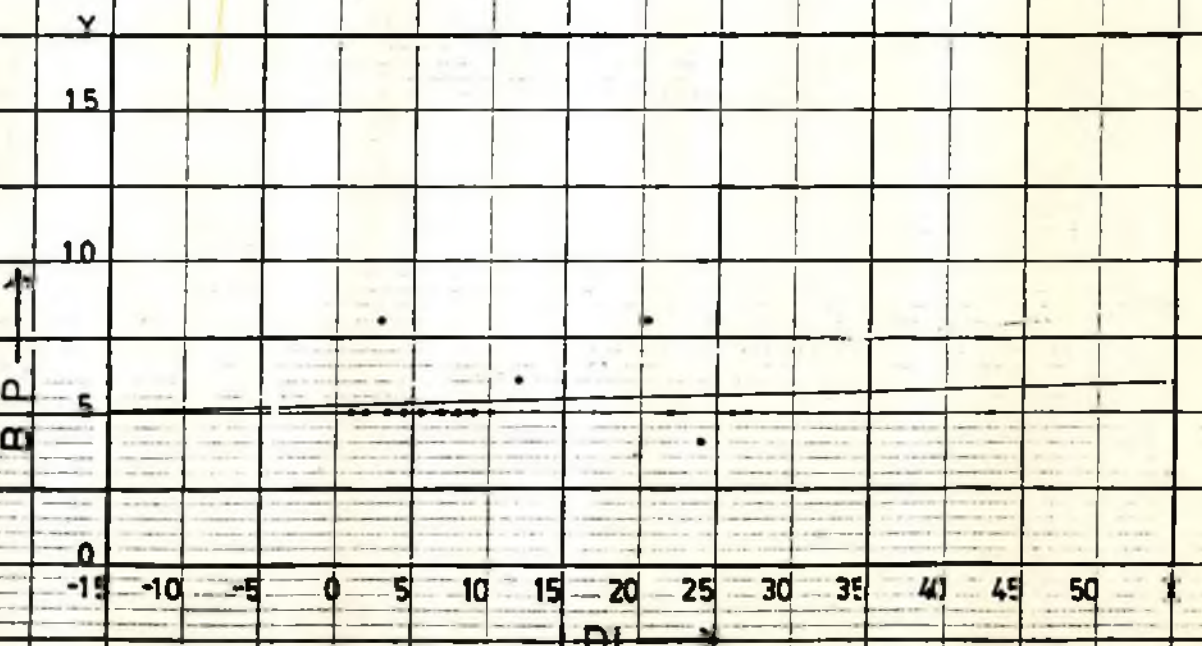
Figure 3.h gives the relationship between the changes in diastolic blood pressure and LDL-cholesterol level from one subject to another were plotted against the changes of diastolic blood pressure level. As per scatter diagram a regression line was drawn. From the graph it appears that there is slightly upward trend of diastolic blood pressure with increases of LDL-cholesterol levels. The regression equation was $Y = 24.28 + 0.037x$ for experimental and $Y = 5.22 + 0.012x$ for control. Co-efficient of correlation indicates a relation of LDL-cholesterol and diastolic blood pressure in relevance to the rise and fall i.e. a limited degree positive co-rrelation exist in experimental group ($r = 0.101$). In control group there also presents a very little i.e. limited degree positive correlation ($r = 0.018$).

FIGURE 3.h. Relation between changes in serum LDL-Cholesterol and diastolic BP



Reg Eq: $Y = 24.28 + 0.037\ X$; $\hat{\gamma} = 0.101$, $P > 0.05$

(Experimental)

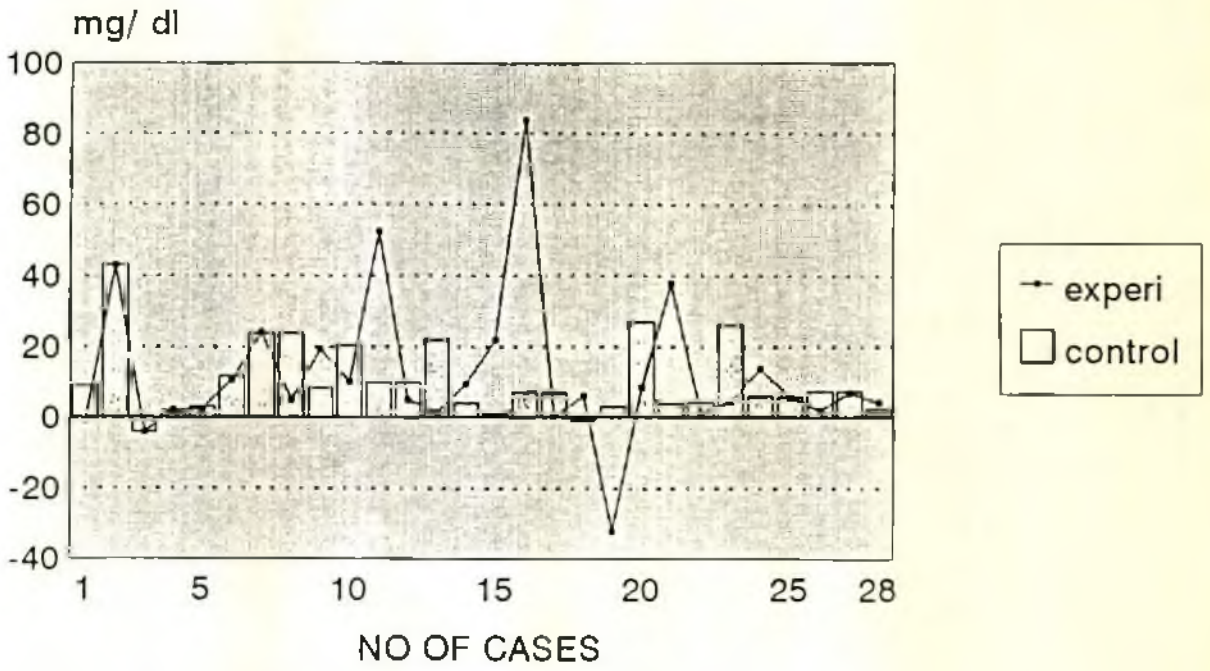


Reg Eq: $Y = 5.22 + 0.012\ X$, $\hat{\gamma} = 0.08$, $P > 0.05$

(Control)

FIG-3.14. CHANGES IN LDL-CHOLESTEROL LEVEL

experi= = $\bar{d}$=12.32, t=3.07
control= $\bar{d}$=10.52, t=5.22



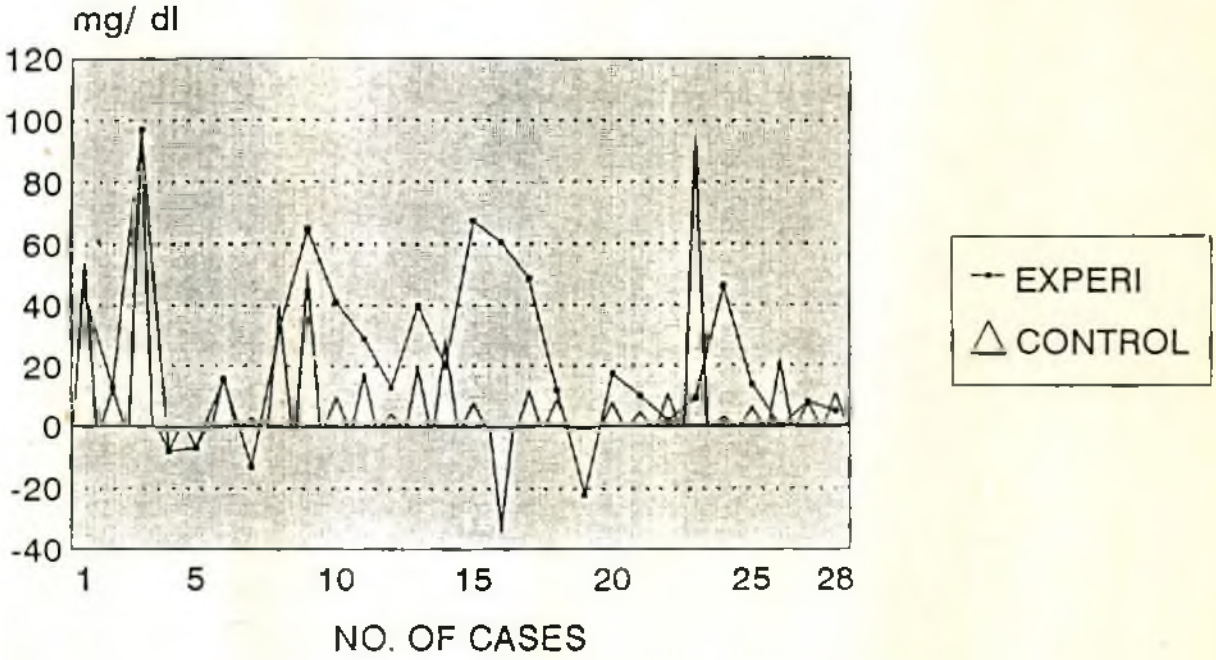
experi= = SD= ± 21.21 (p< 0.05), 95% CI= 4.11 to 20.53
control= SD= ± 10.66 (p< 0.001), 95% CI= 6.39 to 14.65

Figure 3.15 shows the reduction of serum TG levels from initial and final measurement in hypertensive and control subjects. The reduction of serum TG levels (differences) was ranged from 67 mg/dl to 0.07 mg/dl and 95 mg/dl to 3.33 mg/dl in experimental and control respectively. The mean reduction of TG level was 23.58 (27.60) and 18.02 (27/95) and standard error 5.21 and 5.28 in experimental and control. The reduction of TG from baseline to final level of measurement was highly significant ($p<.001$) in experimental subjects. A 95% of confidence interval indicates comparatively a large difference in serum TG level in hypertensive subjects.

Figure 3.j gives the relationship between the reduction of diastolic blood pressure and serum TG level. This reduction of TG level from one subject to another were plotted against the reduction of diastolic blood pressure level. As per scatter diagram a regression line was fitted. Regression line indicates that there appears very little upward trend of diastolic blood pressure with the increases of serum TG level, in hypertensive whereas in control subjects upward trend was more prominent. The regression equation was $Y = 24.77-0.001 x$ for experimental and co-efficient of co-rrelation shows very little relation with the changes of serum TG and diastolic blood pressure in experimental ($r = -0.03$) i.e. a very moderate degree negative co-rrelation was present whereas in control group ($r = 0.08$) a limited degree of positive co-rrelation was found.

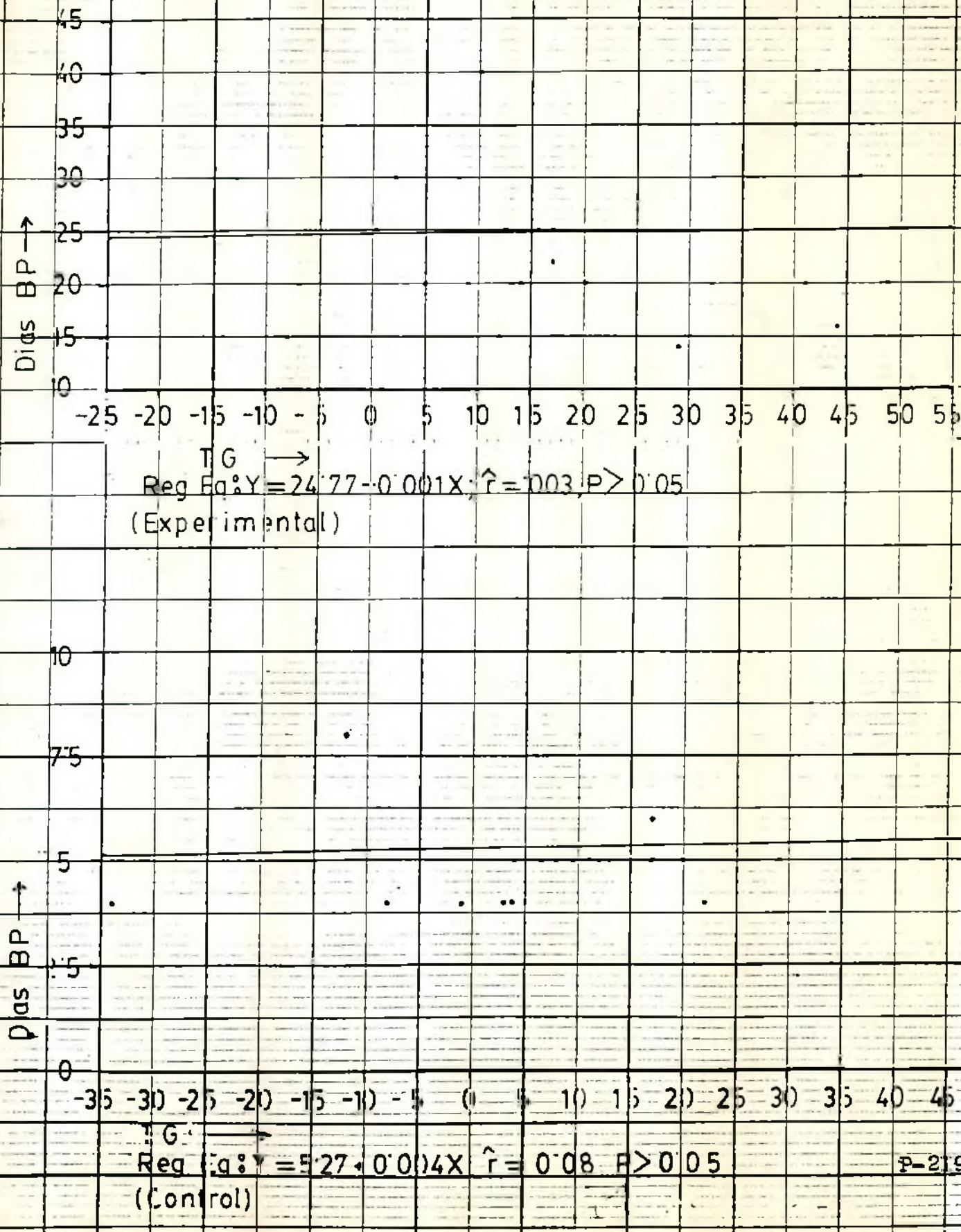
FIG-3.15. CHANGES IN SERUM TRIGLYERIDE LEVEL

EXPERI= = $\bar{d}$= 23.58, t= 4.51
CONTROL= $\bar{d}$= 18.02, t= 3.41



EXPERI= = SD= ± 27.60 (p< 0.001), 95% CI= 12.89 to 34.27
CONTROL= SD= ± 27.95 (p< 0.05) , 95% CI= 7.19 to 28.85

FIGURE 3.j. Relation between changes in serum TG and diastolic BP.



The difference of reduction of serum β lipo-protein levels at baseline and after programme intervention is shown in table-39. This reduction of serum β lipo-protein levels were ranged from 99 mg/dl to 0 mg/dl and 23 mg/dl to 1 mg/dl in hypertensive and control subjects. The mean reduction of β lipo-protein level was 16.37 (± 40.36) and 12.62 (± 7.92) and standard error 7.62 and 1.49 in experimental and control subjects respectively. The changes of β-lipo-protein from before and after programme intervention was statistically significant ($p < 0.05$ for hypertensive and $p < .001$ for control) A 95% of confidence interval indicates comparatively a large difference in serum β lipo-protein in hypertensive subjects.

Figure 3.k shows the relationship between the reduction of diastolic blood pressure and serum β lipo-protein levels. Subject wise reduction of ß lipo-protein levels were plotted against the reduction of diastolic blood pressure. A regression line is drawn. Regression line indicates that there appear downward trend of diastolic blood pressure with the decreases of serum β lipo-protein level in both hypertensive and control groups. The regression equation was $Y = 24.86 + .001x$ for experimental and $Y = 5.03 + 0.026x$ for control. Co-efficient of co-rrelation shows a relation with the reduction of diastolic blood pressure and β lipo-protein levels. A positive correlation is existed in experimental ($r = 0.13$) subjects.

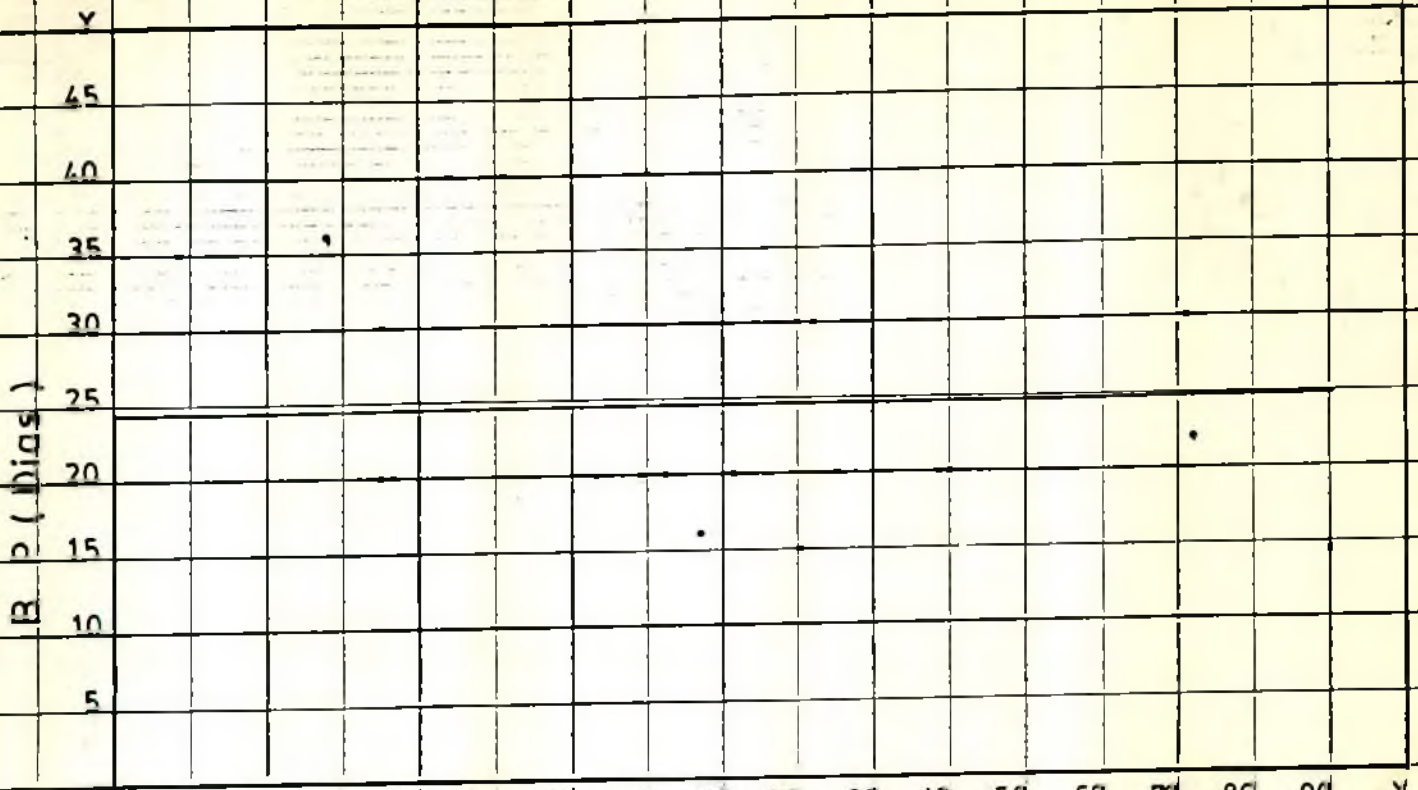
Table-39 Serum ß lipo-protein level (mg/dl) of experimental and control at initial and final measurement

	Experimental			Control		
Subject	Initial	Final	Difference	Initial	Final	Difference
1	463	456	+ 7	464	454	+ 10
2	526	427	+ 99	526	527	-1
3	560	488	+ 72	420	410	+ 10
4	498	456	+ 42	499	476	+ 23
5	418	379	+ 39	420	400	+ 20
6	511	456	+ 55	515	500	+ 15
7	435	470	-35	471	450	+ 21
8	400	401	-1	640	620	+ 20
9	456	456	0	466	455	+ 11
10	456	454	+ 2	420	400	+ 20
11	480	389	+ 91	440	420	+ 20
12	480	423	+ 57	410	400	+ 10
13	402	463.12	-61.12	430	420	+ 10
14	445.31	480	-34.69	340	310	+ 30
15	463.13	470	-6.87	463	450	+ 13
16	498.75	540	-41.25	486	379	+ 07
17	466	500	-34	523	500	+ 23
18	532	512	+ 20	418	417	+ 1
19	437	463	-26	456	450	+ 6
20	463	460	+ 3	532	520	+ 12
21	425.60	445.31	-19.71	427	426	+ 1
22	670	650	+ 20	463	452	+ 11
23	463	452	+ 11	622	600	+ 22
24	498.75	427	+ 71.75	409	400	+ 9
25	445	420	+ 25	425.60	410	+ 15.60
26	391	350	+ 41	437	435	+ 2
27	532	510	+ 22	427	422	+ 5
28	445.32	405	+ 40.32	392	385	+ 7

d= 16.37 t = 2.14
SD= ± 40.36 p<0.05
SE = 7.62
95% CI = 0.73 to 32.01

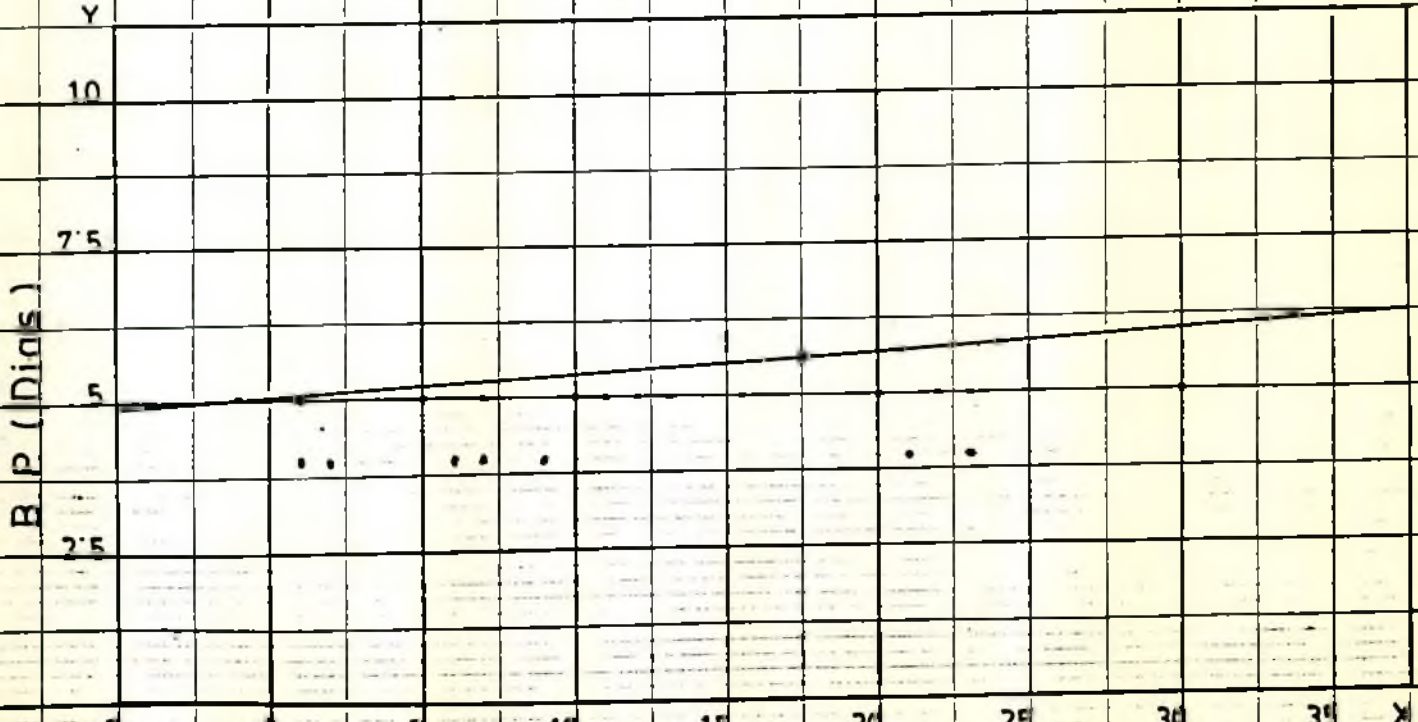
d = 12.62 t = 8.42
SD = ± 7.92 p<.001
SE = 1.49
95% CI = 9.56 to 15.68

FIGURE 3.K. Relation between changes in serum B-Lipo-Protein and Diastolic BP.



Reg. Eq: $Y = 24.86 + 0.001X$; $\hat{\delta} = 0.09$, $P > 0.05$

(Experimental)



Reg. Eq: $Y = 5.03 + 0.026X$; $\hat{\delta} = 0.13$, $P > 0.05$

(Control)

Table-40 shows the change of serum total lipids from initial to final level in both the experimental and control group. The change of serum total lipids was ranged from 98.33 mg/dl to 3 mg/dl in hypertensive and 97 mg/dl to 1 mg/dl in normotensive subjects. The mean reduction of serum total lipids was 54.81 (±29.49) and 42.06 (± 31.64) and standard error 5.57 and 5.98 in experimental and control group respectively. The changes i.e. a reduction of serum total lipids from before and after programme intervention was statistically significant ($p<.001$) in both the groups. A 95% of confidence interval shows no dissimilar differences in serum total lipid level.

The relationship of serum total lipid and diastolic blood pressure is shown in figure 3.L. The changes of total lipid were plotted against the reduction of diastolic blood pressure level. A regression line is fitted. This line indicates that there appear a very little downward trend of diastolic blood pressure with decreases of total lipids level in hypertensive subjects. On the other hand a downward trend of diastolic blood pressure with the decrease of the same was noted. In experimental group there was a very limited degree of negative correlation ($r = -0.27$) whereas in control group a limited degree positive co-relation is present.

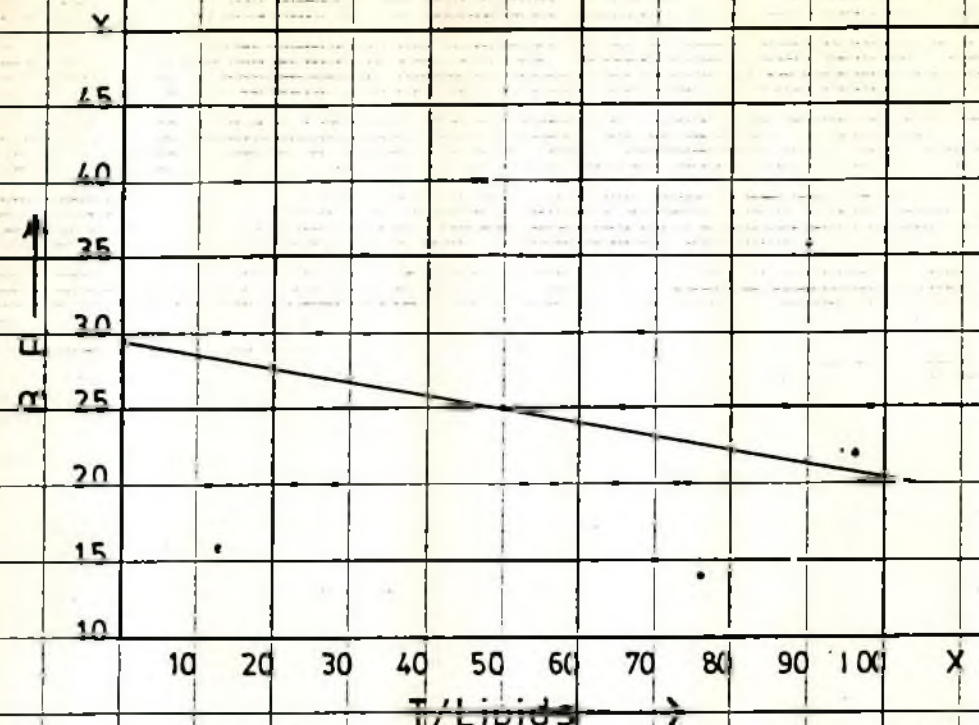
Table-40 **Serum total lipids (mg/dl) of experimental and control subjects at initial and final measurement**

	Experimental			Control		
Subject	Initial	Final	Difference	Initial	Final	Difference
1	813	800	13	850	810	40
2	886	813	73	886	813	73
3	1066	970	96	986	940	46
4	937	867	70	940	869	71
5	786	733	53	788	744	44
6	965	866.67	98.33	967	875	92
7	833	830	3	934	933	1
8	833	810	23	870	800	70
9	867	820	47	877	812	65
10	867	810	57	809	801	8
11	945	867	78	870	840	30
12	843	756	87	840	805	35
13	907	875	32	798	720	78
14	893	865	28	850	820	30
15	875	801	74	843.75	801.22	42.53
16	1065.75	975	90.75	786	800	14
17	862	765	97	883	810	73
18	940	852	88	867	830	37
19	812	766	46	866	800	66
20	875	810	65	910	830	80
21	796.66	812.50	15.84	813	801	12
22	852	848	14	875	821	54
23	875	810	65	866	769	97
24	938.48	912	26.48	812	801	11
25	812.50	801	11.60	713.33	700	13.33
26	750	700	50	866	842	24
27	966	898	68	781	752	29
28	875	810	65	812	800	12

d= 54.81 t = 9.83
SD= ± 29.49 p<.001
SE = 5.57
95% CI = 43.39 to 66.23

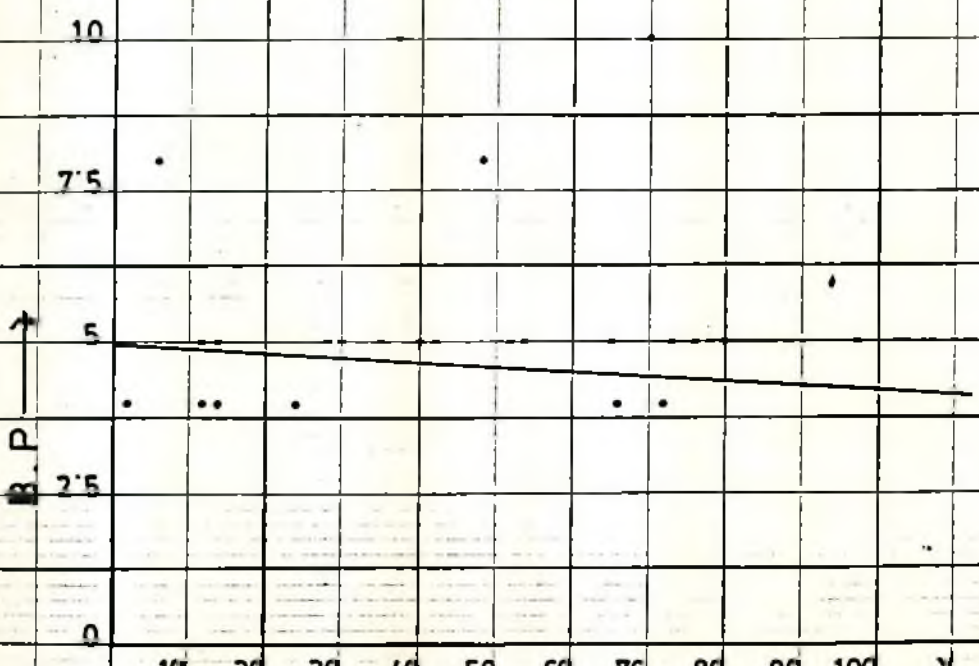
d = 42.06 t = 7.03
SD = ± 31.64 p<.001
SE = 5.98
95% CI = 29.80 to 54.32

FIGURE 4.L. Relation between changes in serum total lipids and diastolic BP.



Reg. Eq: $Y = 28.68 - 0.071X$, $\hat{r} = -0.27$, $P > 0.05$

(Experimental)



Reg. Eq $Y = 5.03 - 0.007X$; $\hat{r} = -0.13$, $P > 0.05$

(Control)

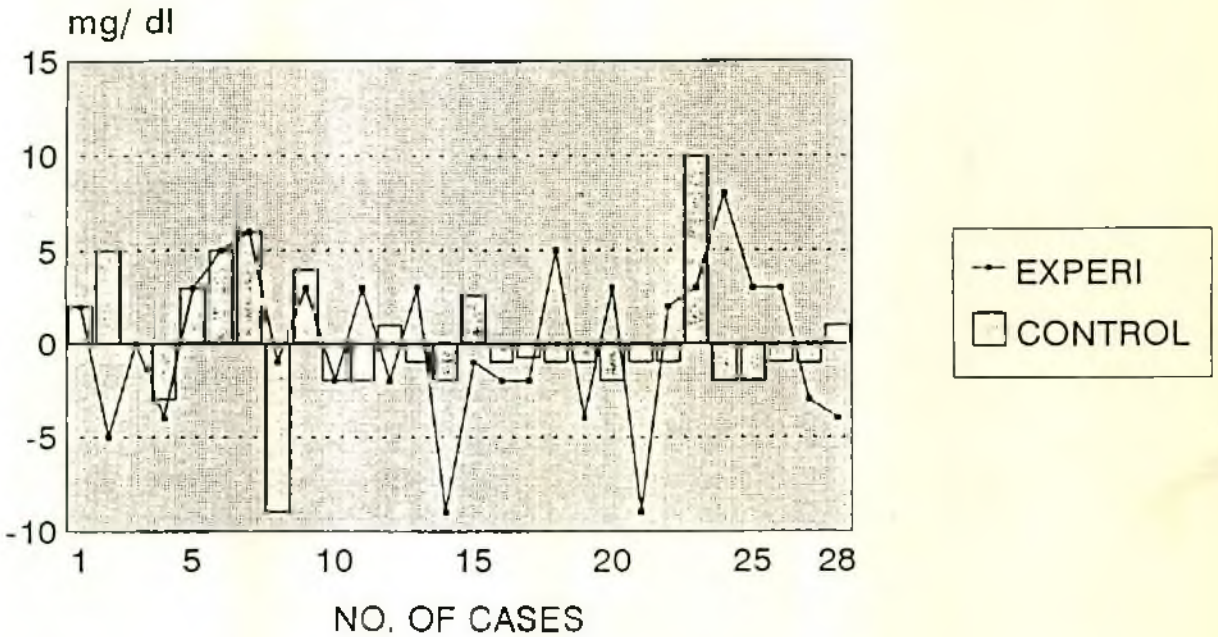
3.21 CHANGES IN SERUM UREA AND CREATININE LEVEL

The changes in serum urea level is shown in Figure 3.16. In both hypertensive and normotensive group the range of difference was estimated to be 9 mg/dl to 1 mg/dl and 10 mg/dl to .75 mg/dl. The mean changes of serum urea level was 0.14 (± 42.6) and 0.24 (± 3.57) with standard error 0.80 and 0.67 in experimental and control. The mean difference was statistically significant in both the groups. ($p < 0.05$). A 95% of confidence interval shows a little difference as per the range (-1.51 to + 1.79 for hypertensive and -1.14 to + 1.62 in control) in hypertensive and control groups.

Figure 3.17 presents the change of serum creatinine level at baseline and after programme intervention in hypertensive and normotensive participants. The change of serum creatinine level was ranged from 0.2 mg/dl to 0 mg/dl in hypertensive and 0.2 mg/dl to .01 mg/dl in normotensive subjects. The mean difference of changes was 0.05 (± 0.18) and 0.07 (± 0.15) with standard error 0.03 and 0.02 in experimental and control group. A 95% of confidence limit shows almost a same difference in both the groups. The difference of creatinine level was significant ($p < 0.05$) in normotensive subjects, however no appreciable significant was observed in hypertensive group.

FIG-3.16. CHANGES IN SERUM UREA LEVEL

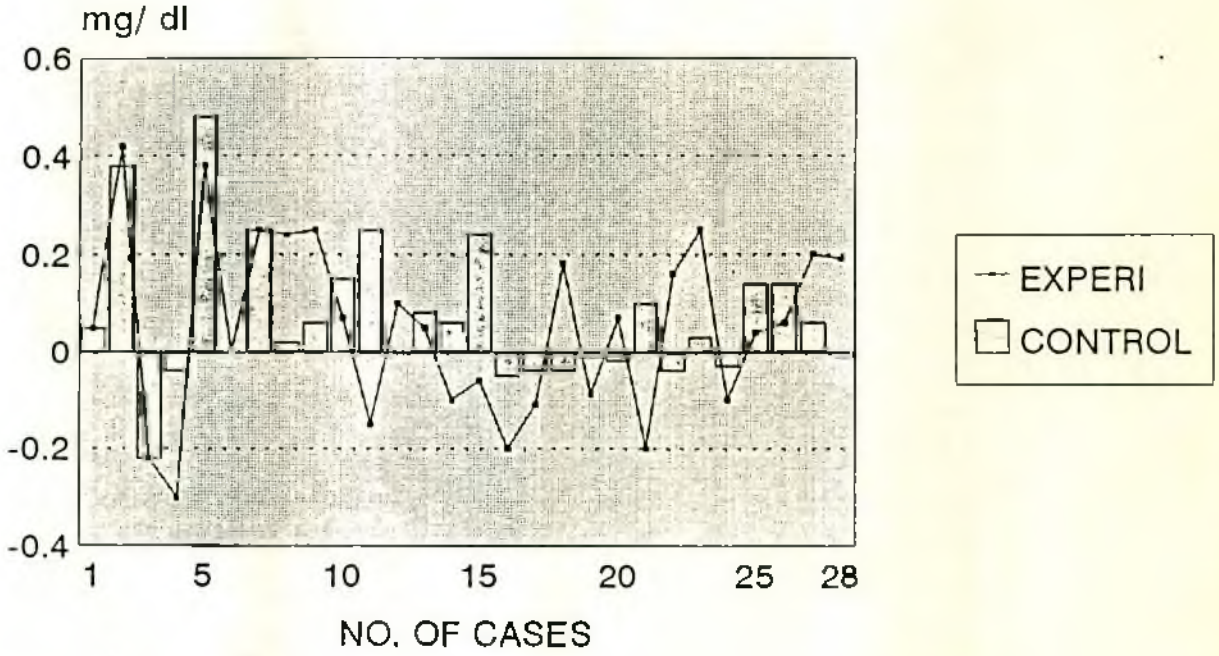
EXPERI == $\bar{d}$=0.14, t= 0.17
CONTROL== $\bar{d}$=0.24, t= 0.35



EXPERI == SD= ± 4.26 (p< 0.05), 95% CI= -1.51 to 1.79
CONTROL== SD= ± 3.57 (p< 0.05), 95% CI= -1.14 to 1.62

FIG- 3.17. CHANGES IN SERUM CREATININE LEVEL

EXPERI == $\bar{d}$= 0.05, t= 1.46
CONTROL== $\bar{d}$= 0.07, t= 2.46



EXPERI == SD= ± 0.18 (p< 0.05), 95% CI= -0.01 to 0.11
CONTROL== SD= ± 0.15 (p< 0.05), 95% CI= 0.02 to 0.12

3.22 REDUCTION IN SERUM FASTING PLASMA GLUCOSE

Table-41 shows the difference i.e. reduction of serum fasting plasma glucose at entry and final point of intervention. The reduction of serum fasting plasma glucose was ranged from 32 mg/dl to 1 mg/dl and 10 mg/dl to 1 mg/dl in hypertensive and control group of subjects. The mean reduction of fasting plasma glucose was 9.32 (± 7.27) and 5.38 (± 3.06) and standard error 1.39 and 0.57. The changes or reduction of serum fasting plasma glucose was highly significant in both the groups ($p < .001$). A 95% of confidence limit indicates a large gap in hypertensive (6.49 to 12.11) subjects than the normotensive.

Figure 3.m - presents the relationship of the fasting plasma glucose and diastolic blood pressure. Changes in fasting plasma glucose value were plotted against the changes of diastolic blood pressure level. A regression line is fitted. This line indicates that there appear upward trend of diastolic blood pressure with the increases of serum plasma glucose level in hypertensive group. While in normotensive group a downward trend is observed with increases of serum plasma glucose. A limited degree of positive co-rrelation ($r = 0.51$) and very little limited degree of negative co-rrelation ($r = 0.90$) in experimental and control subjects was observed.

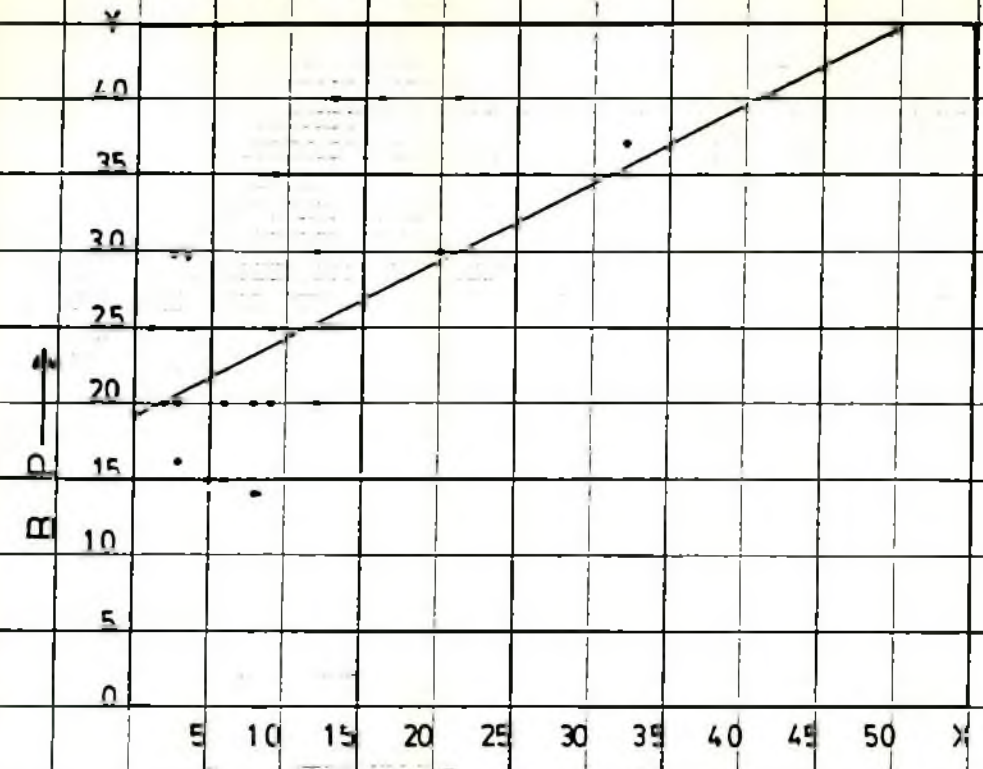
Table-41 Fasting plasma glucose (mg/dl) of experimental and control at initial and final measurement

	Experimental			Control		
Subject	Initial	Final	Difference	Initial	Final	Difference
1	99	96	3	99	96	3
2	98	89	9	98	88	10
3	125	104	21	101	98	03
4	92	87	5	93	87	06
5	96	84	12	96	90	06
6	92	88	4	92	88	4
7	88	76	12	89	80	9
8	83	80	3	100	98	2
9	110	84	16	96	90	6
10	92	83	9	98	88	10
11	98	90	8	86	82	4
12	107	98	9	88	86	2
13	100	90.80	9.20	102	98	4
14	105	97	8	89	86	3
15	88	80	8	102.92	97	5.92
16	132	100	32	104	100	4
17	98	86	12	110	100	10
18	90	84	6	99	96	3
19	115	94	21	85	80	5
20	97	94	3	95	90	5
21	88	75	13	98	90	8
22	102.45	100	2.45	110	100	10
23	79	77	2	101.63	98.70	2.93
24	121	101	20	88	86	2
25	92	86	6	110	100	10
26	87	86	1	84	82	2
27	101	98	3	108	98	10
28	83	80	3	87	86	1

d= 9.30 t = 6.76
SD= ± 7.27 p<.001
SE = 1.39
95% CI = 6.49 to 12.11

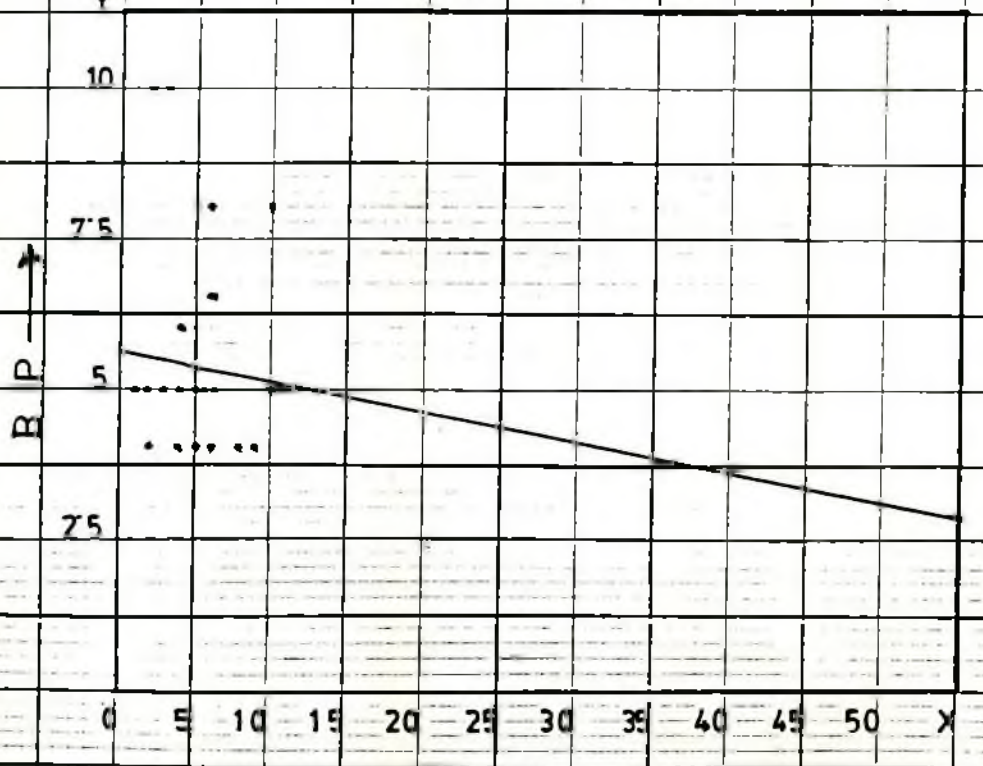
d = 5.38 t = 9.30
SD = ± 3.06 p<.001
SE = 0.57
95% CI = 4.20 to 6.56

FIGURE 3 a. Relation between changes in serum plasma glucose F) and diastolic BP.



Reg Eq: $Y = 19.53 + 0.56X$; $\hat{x} = 0.51$ $P < 0.05$

(Experimental)



Reg. Eq: $Y = 5.63 - 0.05X$ $\hat{x} = -0.09$ $P > 0.05$

(Control)

3.23 FOLLOW UP MEASUREMENT OF BLOOD PRESSURE IN HYPERTENSIVES

Table 42 presents the data on follow up of hypertensive subjects taken at monthly interval over a period of six months. Changes in systolic and diastolic blood pressure were obtained from the final blood pressure reading at the end of the intervention. The changes were ranged from 20 mmHg to 0 mmHg and 10 mg to -5 mmhg in systolic and diastolic blood pressure respectively. The mean difference in systolic blood pressure was 6.82 (± 4.43) and in diastolic blood pressure 4.03 (± 3.96) with standard error 0.83 and 0.74. The changes in blood pressure after six months were highly significant ($p < .001$) in hypertensive subjects. A 95% of the confidence interval shows almost a similar difference (5.11 to 8.83 and 2.50 to 5.56) in systolic and diastolic blood pressure. The relationship between two diastolic blood pressure readings as taken 6 months apart showing little change on average. In Figure 3.11 changes in diastolic blood pressure were plotted against the changes took place after 6 months. A regression line is fitted. The straight line indicates that there appear upward trend of diastolic blood preseure after 6 months apart with the rises of final diastolic blood pressure. A limited degree positive correlation ($r=0.8$) was found (Reg. eq : $35.58 + 0.62y$).

Table-42 Changes in Blood pressure in hypertensives, taken six months apart from final reading

Subject	Systolic B.P mmHg			Diastolic BP mmHg		
	Final B.P	After Six months	Difference	Final B.P	After Six months	Difference
1	130	140	± 10	90	90	0
2	140	140	0	86	90	± 40
3	130	140	± 10	80	86	± 6
4	130	136	± 6	85	86	± 1
5	140	144	± 4	90	90	0
6	130	136	± 6	85	90	± 5
7	130	135	± 5	80	86	± 6
8	140	140	0	90	95	± 5
9	140	142	± 2	80	86	± 6
10	140	142	± 2	90	95	± 5
11	130	135	± 5	90	96	± 6
12	120	130	10	80	90	± 10
13	130	140	10	70	80	± 10
14	120	130	10	90	90	0
15	140	142	2	90	94	± 4
16	100	110	10	70	80	± 10
17	140	142	2	90	90	0
18	130	136	6	90	90	0
19	110	120	10	70	80	± 10
20	140	144	4	85	80	-5
21	110	130	20	70	80	± 10
22	130	135	5	80	84	± 4
23	120	130	10	80	86	± 6
24	120	130	10	80	80	0
25	130	140	10	80	85	± 5
26	140	142	2	80	80	0
27	120	130	10	80	80	0
28	130	140	10	80	85	± 5

d= 6.82 t = 8.14
SD= ± 4.43 p<.001
SE = 0.83
95% C1 = 5.11 to 8.83

d = 4.03 t = 5.384
SD = ± 3.96 p<.001
SE = 0.74
95% C1 = 2.50 to 5.56

3.24 REGRESSION TO THE MEAN

Figure - 3,n, 3.0, 3.p shows the relationship between two systolic and two diastolic blood pressure (Initial and Final) readings taken three weeks and six months apart on 28 hypertensive subjects. It can be seen that apart from random variation there is good agreement between the two readings in both experimental and control. However, when the difference between the two readings is related to the initial reading in both systolic and diastolic blood pressure measuring a different picture is observed in normotensive subjects but no difference in hypertensive (Figure - 3q). It appears that there is a downward gradient and those with a high initial level have a reduced blood pressure three weeks later, while the opposite is true for those with an initial low level. It may be a spurious finding in case of normotensive subjects. It is entirely caused by the fact that there is a mathematical relationship between the difference (Initial BP minus Final BP) and initial blood pressure reading, initial blood pressure reading contributing to both variables. Yet investigator, further wants to be able to assess whether , for example, a metabolic control or Non-drug treatment of hypertension, induced change, is related to initial value.

FIGURE 3.4. Relation between changes in diastolic BP at six months apart.

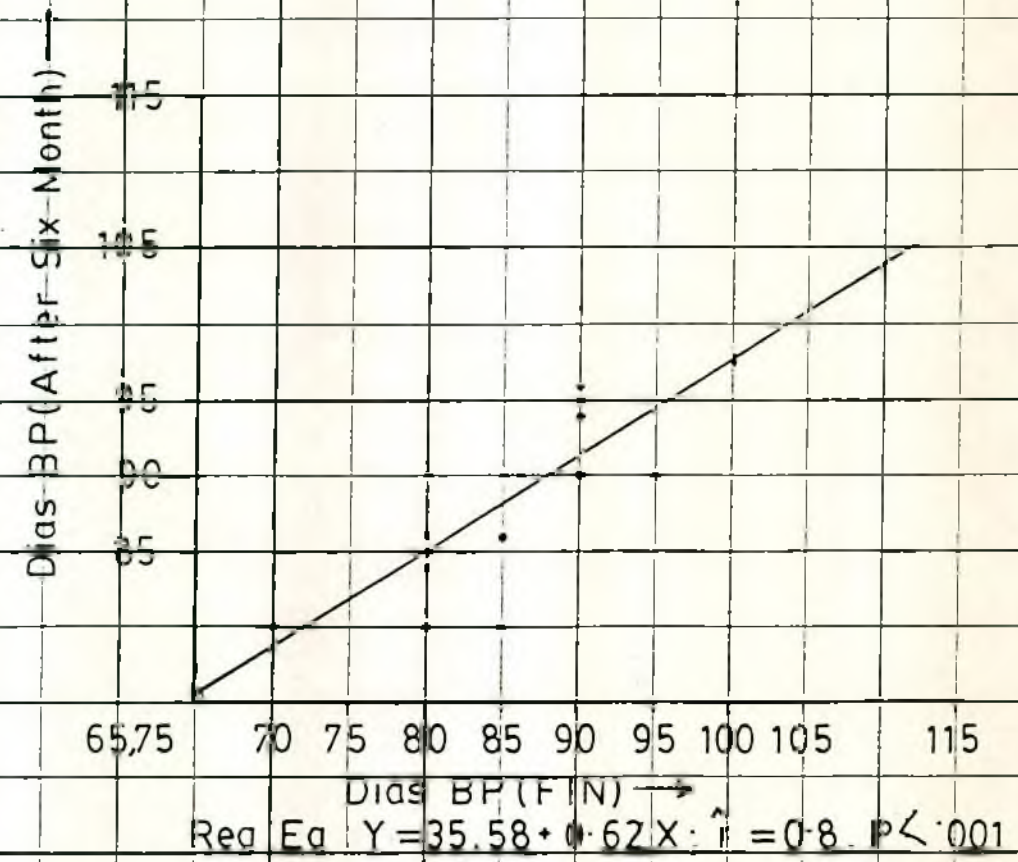
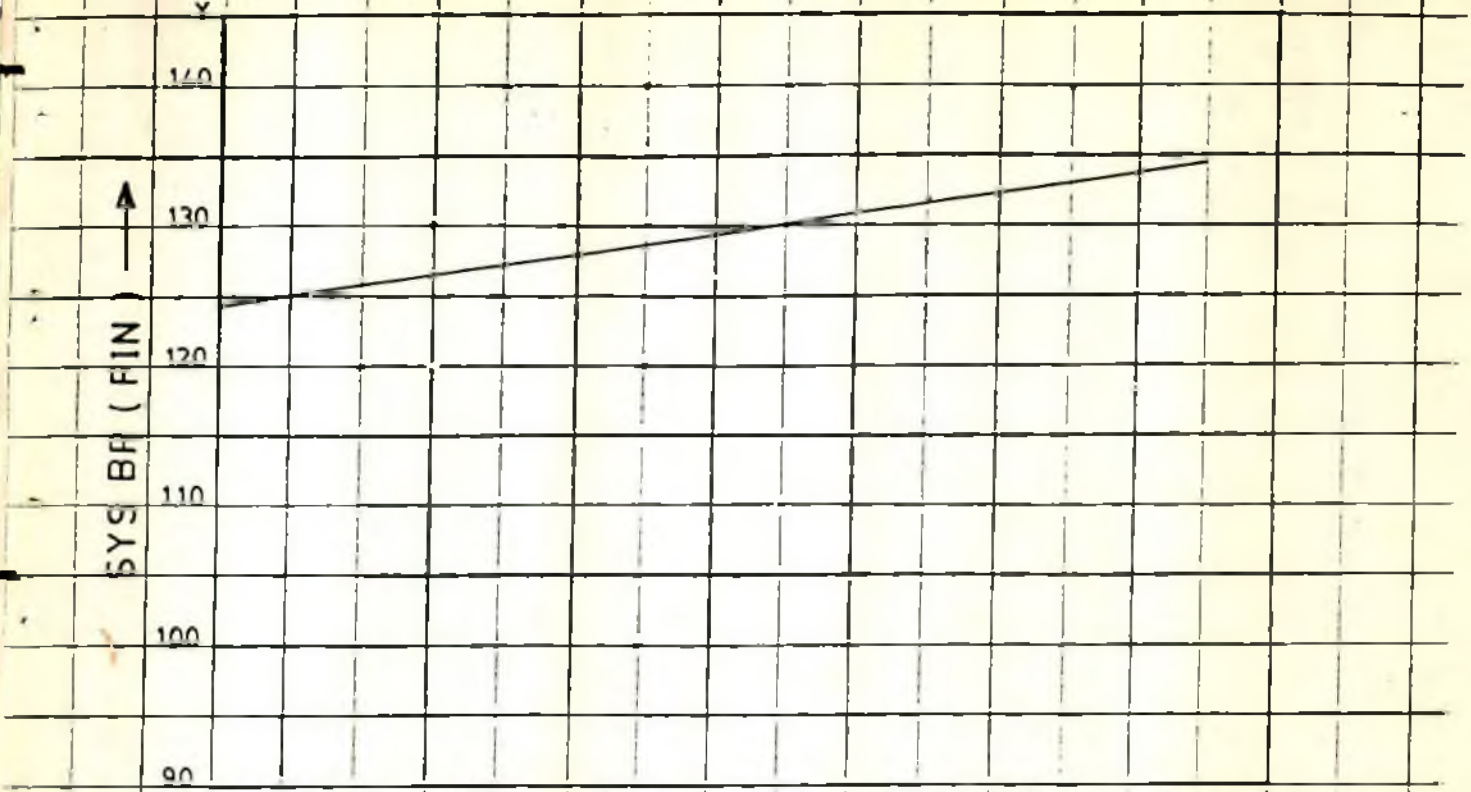
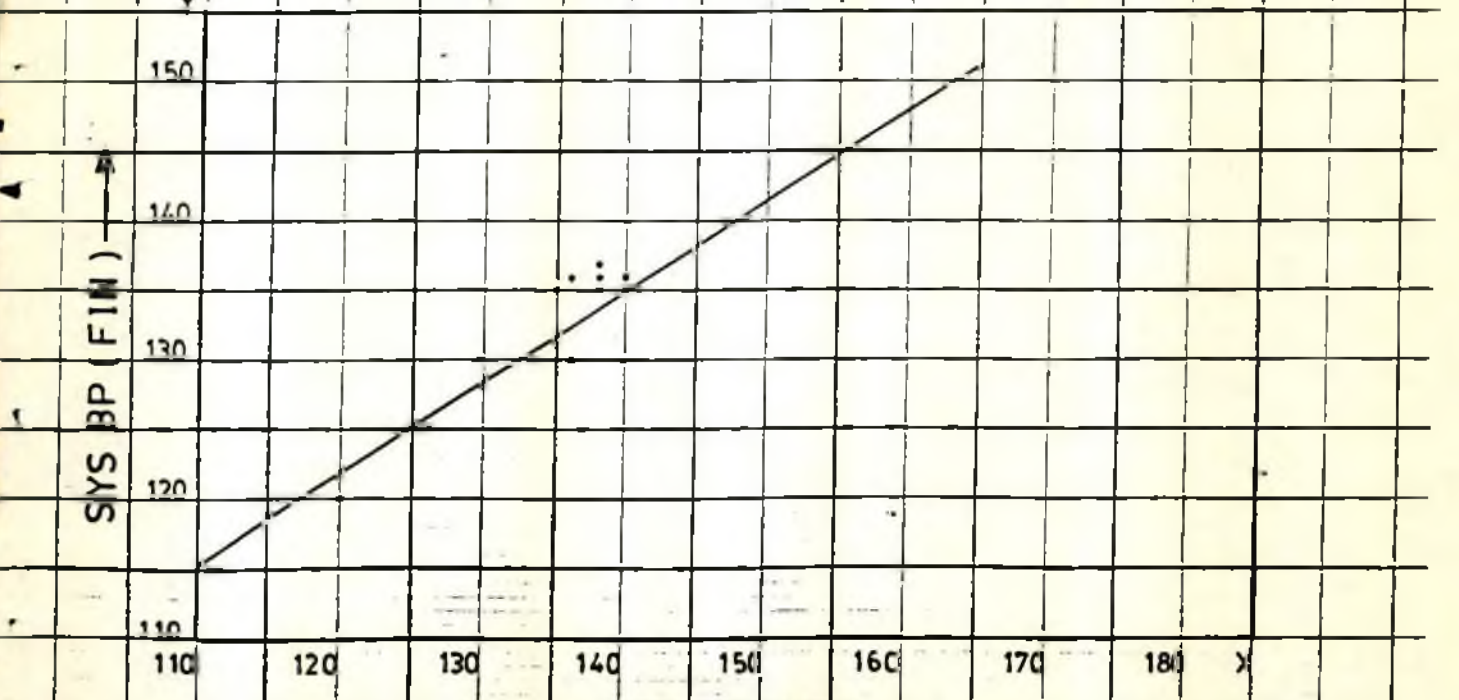


FIGURE 3.o. Relation between changes in Initial and final systolic BP.



Reg Eq Y = 103·47 + ·157 X; $\hat{s}$ = 0·15, P > 0·05

(Experimental)



Reg Eq: Y = 42·63 + 0·66X. $\hat{s}$ = ·63 P < ·001

(Control)

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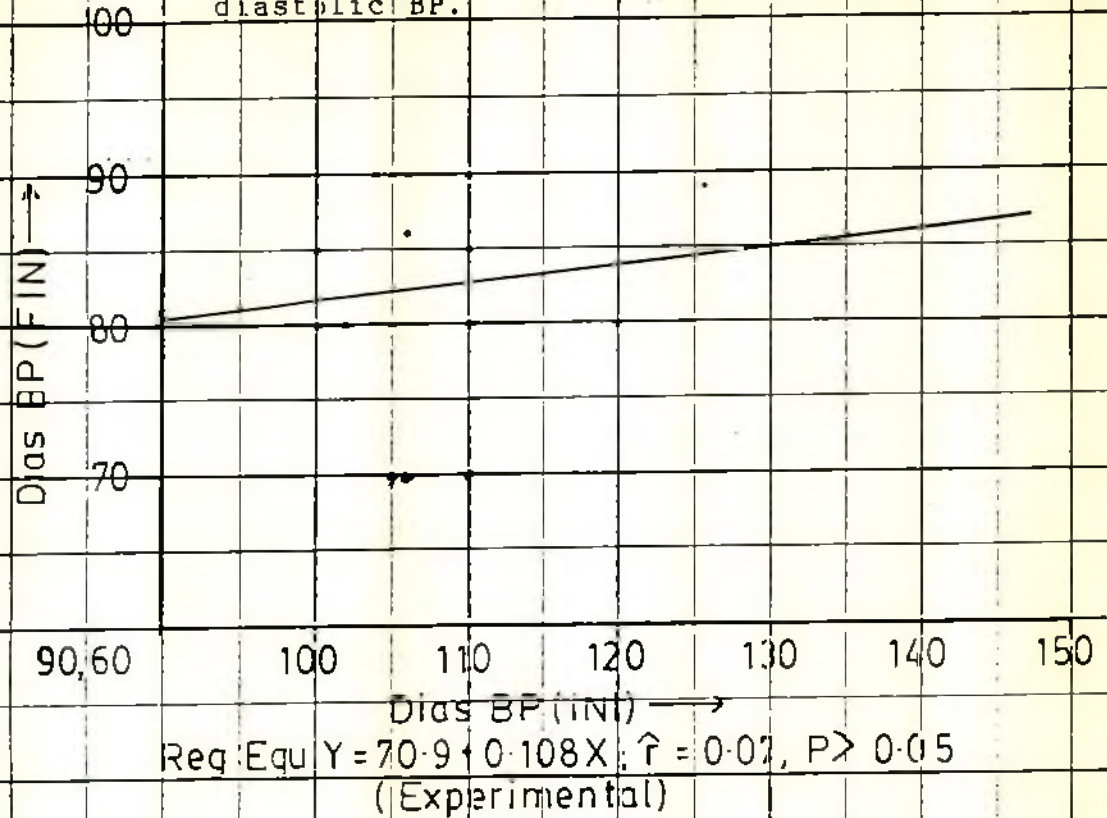
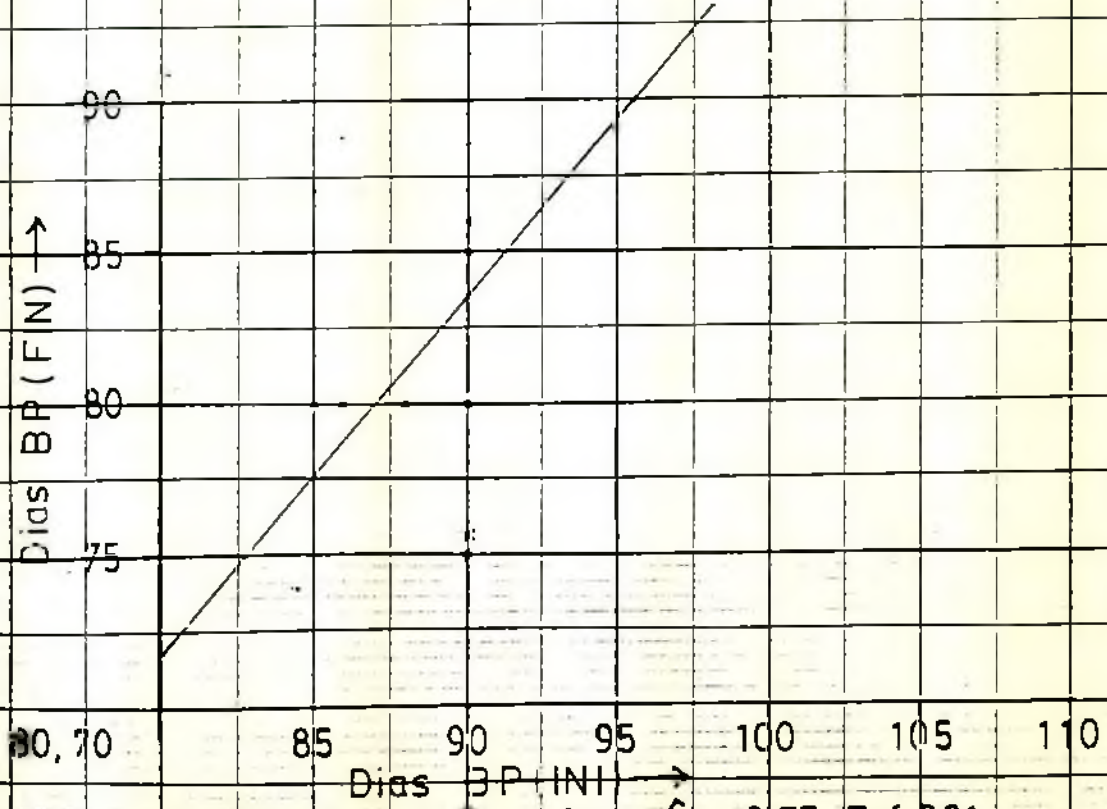
FIGURE 3.P. Relation between changes in Initial and final diastolic BP.

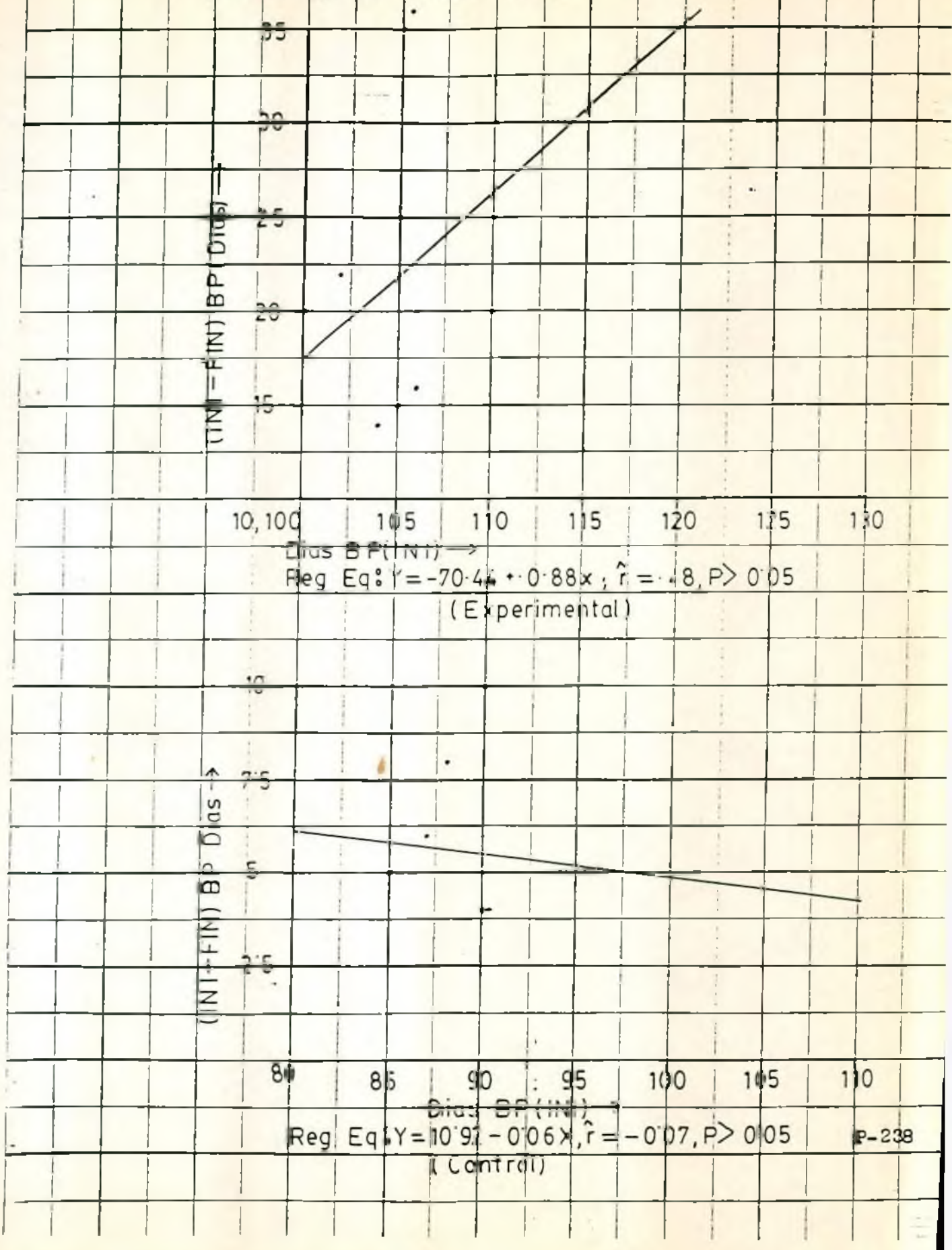


Reg Equ $Y = 70{\cdot}9 + 0{\cdot}108X$; $\hat{r} = 0{\cdot}07$, $P > 0{\cdot}05$
(Experimental)



Reg Eq: $Y = -11{\cdot}81 + 1{\cdot}07X$; $\hat{r} = 0{\cdot}77$, $P < 0{\cdot}001$
(Control)

FIGURE 2.0 Regression differences of diastolic BP, plotted against initial value.

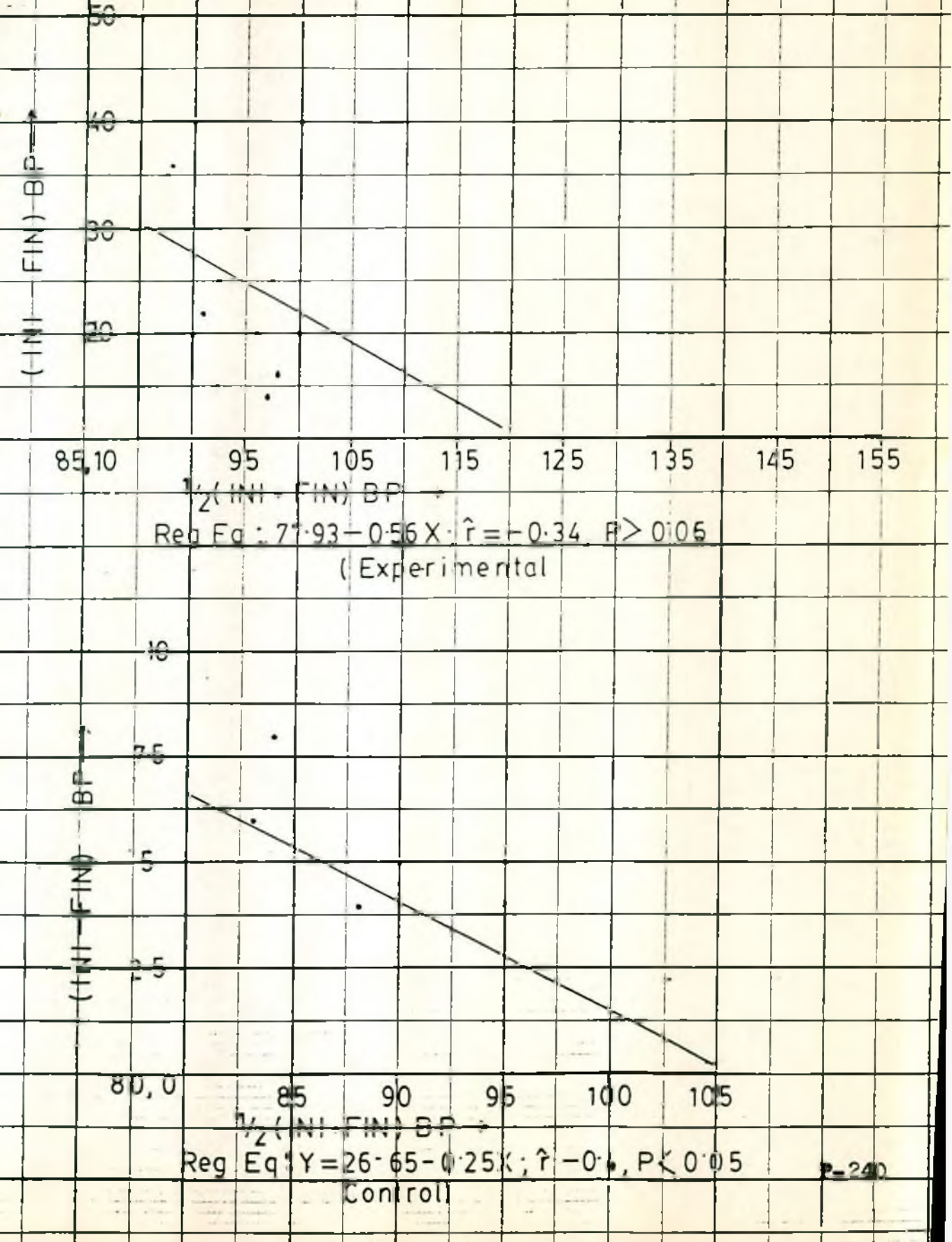


Let us consider the implications of regression to the mean for clinical trials of Nondrug therapies. Such trials are confined to subjects with initial raised blood pressure. It may be seen that if such participants were followed for a period of time and re measured their blood pressures would on average have decreased, even in the absence of any treatment. Thus it is essential to have a control group of persons with initially similar levels of blood pressure and to assess any apparent reduction in mean blood pressure of the treated group by a comparison with the mean reduction observed in the control group.

To assess whether the Nondrug therapeutic approach is related to the fall of blood pressure level a method suggested by Oldham (1968)[177] may be used. The difference (initial BP final BP) is plotted against the average of the initial and final blood pressure readings = $\frac{1}{2}$(Initial BP + Final BP) (Fig 3.r) rather than against the initial reading. A negative correlation of these variables would imply a tendency for subjects with raised blood pressure to show a larger decrease than those with low blood pressure, while a positive correlation would imply the opposite.

Figure - 3.r shows a plot of (initial BP -- Final BP) against $\frac{1}{2}$(initial BP + final BP) for the data in figure 3p. The correlation was not significant. It is therefore, not confirming that Oldham's method has overcome the spurious apparent association between

FIGURE 3.r. Relation between differences of diastolic BP plotted against average of initial value.



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blood pressure reduction and initial blood pressure which was caused by regression. Furthermore in figure 3.q changes in diastolic blood pressure in hypertensive subjects plotted against the initial value. It has therefore, been observed that there appears an upward trend in blood pressure i.e., with high initial reading there will be a high difference in blood pressure. A limited degree positive correlation was noted ($r = 0.48$). ($Y=70.441 + 0.88 X$)

This results indicate that the changes of blood pressure was due to the effect of metabolic therapy but not due to the effect of statistical concept of regression towards mean.

CHAPTER IV

DISCUSSION

4.1 BASE LINE CHARACTERISTICS

1. Age, physical activity, smoking and Blood pressure

The results of this study show that 48.2% of the subjects belonged to the age group of 30-39 years. Of the total 56 subjects majority were civilian. The variations of blood pressure level were observed in terms of different age groups and in different categories of personnel. Various studies have noted: different populations with different levels of blood pressure[188]. It has been observed that both systolic and diastolic blood pressure were found high with the increases of age virtually all surveys. including those from Africa. Latin America, oceania and India. have shown a rise in blood pressure with age in both men and women. In young and older adults the blood pressure increase with age is largely determined by the initial blood pressure levels. However. in connection with aging systolic hypertension deserves special consideration. It may be viewed as a concomitant of aging, being quite infrequent until the mid-fifties, rising further with advancing age and carrying an increase risk of cardiovascular disease[189].

The increase in systolic blood pressure appears to continue throughout life, whereas there is a tendency for diastolic pressure to level of around the age of 55 to 60[190]. This study showed 66.6% of subjects having diastolic blood pressure 90-104 mmHg, were in the age group of 50-69 years. In western societies the average systolic and diastolic blood pressures gradually rise with age. Hypertension is defined arbitrarily at levels above accepted 'normal', for example 140/90 at the age of 20. 160/95 at the age of 50 and 170/105 at the age of 75.[185]. Longitudinal studies have indicated that the increase of blood pressure with age is more marked with higher the blood pressure initially recorded at any age. In young adults in the U.S.A. the increase in pressure with age is greater in blacks that in whites. Several communities, usually isolated and primitive, have by contrast been observed in which there is little or no increase of systolic or diastolic blood pressure with age[177].

The variations of blood pressure level as per nature of physical activity, either sedentary or hard manual labour were observed. The relationship between physical activity and blood pressure has been little studied in large well controlled population samples. In this study no significant association was identified. In the Framingham study, there was also no significant relationship was detected[191]. In studies in Gothenburg relationships were not found between either leisure time or occupational activity and blood

pressure and also there was no association between activity level and change of pressure with time.[192,193]

As per smoking habit, majority of the subjects (62.5%) were smoker and 40% of them had systolic pressure in the range 140-149 mm Hg. Almost a similar pattern of diastolic pressure with smoking habit was also noted but no significant relationship detected. Smoking, though not causally related to high blood pressure, is an important disease risk factor. Since the risk of coronary artery disease is some 50-60% higher in a hypertensive smoker than in a non smoking hypertensive patient. The health hazards of smoking for adults and younger people are well established[194]. The specific relationship of cigarette -smoking to CHD has been documented in population studies, though the detailed mechanisms remain uncertain.

Blood pressure was measured on 253 persons in the age range of 20-70 years comprising randomly selected 198 males and 55 females undertaking different grades of physical activity for at least two years to determine the relationship of physical activity on blood pressure. Variables which are already known to affect the blood pressure were also taken in to account, the study shown that the variables such as age, sex, body weight, smoking habit etc. affect the blood pressure. Regular physical activity inhibits a rise in the blood pressure and specially in diastolic pressure. Such

effect irrespective of other variables was also maintained. The study reveals that both components of blood pressure decreased with increasing grade of physical activity[186].

2. Body weight, Height, BMI and PBMI

From results it indicated that majority of the subjects (61%) with raised systolic blood pressure had body weight ranging from 65-84 kg. This finding was statistically significant. In 66.6% of the subjects had equal to or greater than 85 kg body weight whose diastolic blood pressure was 90-104 mm Hg. This study revealed that subjects with high blood pressure had more body weight.

In a large number of cross-sectional observational studies, it appears that blood pressure and relative weight levels are highly co-rrelated, not only in adults but also at younger ages[33,195,196]. In prospective observational studies, it is found that persons who gain weight show a greater rise in blood pressure over time than those who maintain their weight, while pressure falls with weight loss.[197-200] Among such studies, the most persuasive data come from Georgia, U.S.A., indicating that those who are obese from the start and gain further weight are at 6 times higher risk of becoming hypertensive than this people who stay thin[201]. In

several experimental population studies, it has like wise been observed that weight loss is accompanied by blood pressure reduction.[202,203]

High blood pressure and obesity in relevance to the BMI and PBMI was studied. Majority of the subjects (58.9%) had BMI 18-23 kg/m^2. Of them 57.5% and 42.4% had systolic blood pressure 140- 144 mm Hg and equal or above 160 mm Hg. Diastolic blood pressure also varies with maximum and minimum levels of BMI and PBMI. In evaluating cross-sectional data, the need to distinguish between over weight and obesity has to be kept in mind. In the studies reviewed by the scientific group, the weight indices used largely reflect body fat, though some contribution by body mass can't always be excluded. It is also recognised that the application of insufficiently large sphygmomanometer cuffs to obese arms may lead to an overestimate of blood pressure readings, thus causing fictitiously high correlation between body weight and blood pressure; nevertheless, it is unlikely that this correlation results entirely from such artefacts because the problem has been recognized for some 20 years in epidemiological studies. Furthermore, weight reduction has been shown to cause lowering of arterial pressure. However, in less-well controlled situations and in clinical practice there remains a need to pay attention to the difficulties of making accurate blood pressure measurements.[204]

Height and blood pressure levels vary considerably. Systolic pressure recorded as 140-149 mm Hg in 75% whose heights were ranged from 1.71 to 1.90 metre. 83.3% of the subjects whose heights were 1.71 to 1.90 metre, their diastolic pressure correspond 90-104 mm Hg. The different levels of systolic and diastolic pressure were differed significantly with different heights of the individual. It has recently been observed among INTERSALT subjects that height makes an independent contribution to blood pressure is a model that includes the body mass index[205,206] Contrary to the author's assertion, this finding should not have come as a surprise. A stratified life insurance data on young adult policy holders in to four strata according to the subject's ponderosity index (weight/height^3), without benefit of well-standardized blood pressure measurements or adjustments for other possible effect modifiers. For both men and women aged 30-39 years, they demonstrated that every 3 inch increase in height was associated with a mean systolic blood pressure increase of 1 or 2 mm Hg i.e. for Icm equal to 0.13 or 0.26 mm Hg.

These slope estimates are somewhat higher than the regression coefficients, reported for systolic blood pressure on height, adjusted for body mass index and five other variables, among men and women aged 20-39 years. These findings appear to agree that the height effect on blood pressure is weaker for diastolic

pressure and among older adults.[206,207] From physiologic point of view, the principles of elementary physics require that taller individuals have greater blood pressure. It has been known since the renaissance that the pressure exerted by on fluid on the floor of its container is measured by the vertical height of the fluid. However, it would be useful to see how alternative anatomic approaches to the relation between stature and blood pressure would work in other populations of various ages and aggregations of cardiovascular risk factors.[208]

3. Serum electrolytes level and blood pressure.

Baseline serum sodium, potassium and chloride levels were determined in all the subjects and their values were distributed in varying grades of systolic and diastolic blood pressure. It has been observed that 62.5% had serum sodium level ranging from 140-144 mmoL/L, while systolic pressure recorded 140-160 mm Hg. At high systolic blood pressure (160+mm Hg) 45.7% had 140-144 mmoL/L. On the other hand 64.2% at varying grades of diastolic pressure showed 140-144 mmoL/L of serum sodium. A high level of serum sodium showed proportionately high level of blood pressure. At 4.5 to 4.9 mmoL/L of potassium the majority of the subject's blood pressure varies from 140-149 mmHg and 77.7% with diastolic blood pressure 90-105 mmHg showed equal to or greater than 5.5 mmol/L. Serum potassium was found to be varied with blood pressure.

The result was statistically significant. Serum Nacl in undissociated form shows an association with different levels of blood pressure. In 50% of the subjects with systolic pressure 150-159 mm Hg and diastolic blood pressure ≥110 mm Hg showed equal to and greater than 105 mmoL/L. It was postulated as early as 1904 that hypertension could be caused by an excess sodium chloride in the diet.[208] Further evidence of an association has come from studies in Japanese, American and Eskimo populations.[209] Several communities whose daily intake of sodium chloride has been 33 or less have low average blood pressure levels and show little tendency for blood pressure to rise with age. When people migrate from such communities to areas where the daily salt intake is around 7-8g their blood pressure increases proportionately.[210] Similar findings in Asia have also been published. There are, however, several confounding factors operating, such as social change and altered overall nutritional state. Thus it remains uncertain to what extent salt intake responsible for the blood pressure differences.[211-213] Several epidemiological studies within adult populations in the USA, in Europe, and in New Zealand have failed to demonstrate a clear relationship between salt intake or excretion and blood pressure.[211] However, positive relationships have been found within populations[212,215], often with a relatively high sodium chloride intake.[216] There are well known regional differences in the prevalence of hypertension (or stroke) in relation to salt in take in Japan.[217]

Several reports suggest that potassium intake may be a major factor affecting the blood pressure raising effects of sodium. Walker et al found no relation between sodium excretion and blood pressure, but a significant negative association with potassium.[218] Blood pressure was positively correlated with the urinary sodium/potassium ratio but not with sodium alone[219].

4. **Serum lipid profiles and blood pressure levels.**

Baseline serum cholesterol was measured in both the groups. Majority of the subjects (53.3%) having systolic blood pressure 140-149 mm Hg showed cholesterol level, equal to and greater than 220 mm/dl. Subjects with high systolic blood pressure showed, cholesterol level at 180-199, 200-219 and 220 and above in 23%, 46.1% and 46.6% of the subjects respectively. Serum cholesterol level varies with systolic blood pressure, and an association is observed in this study. Raised blood pressure with diastolic pressure equal to or less than 110 mm Hg showed, 180-199, 200-219, 220+mg/dl of cholesterol level in 27.2%, 16.6% and 33.3% of the subjects. However, their variation of diastolic pressure and cholesterol levels was not associated, with respect, first, to the blood pressure cholesterol CHD relationship. Comparisons of populations show a strong association between average population total cholesterol levels and CHD incidence rates[220,221]. Within population total cholesterol predicts risk up to late middle age, the relationship being continuous over a wide range of cholesterol

levels[220] with respect to diet-blood cholesterol relationships, there is a generally strong correlation between the saturated fat and cholesterol composition of habitual diets of population and their mean cholesterol levels. The distribution of adult cholesterol values in middle-aged men in the USA as at 1975-76, where the mean value of 210 mg/dl compares with a means of about 235 mg/dl that prevailed in the 1950s and early 1960s. It indicated the possibility of achieving a mass change[222]. The new mean and distribution in the USA still relatively high- are similar to or exceeded by those in many affluent cultures in various parts of the the world. Countries with such total cholesterol levels and distribution generally have a significant burden of CHD. In contrast, a population in many areas whose mean cholesterol values are around 160 mg/dl or lower, with a corresponding distribution, manifest little or no clinical CHD or severe atherosclerosis. Moreover, population with mean total cholesterol values ranging between 160 mg/dl and 200 mg/dl are to be found over wide areas,(Mediterranean) these people have intermediate or low CHD rates, are well nourished, show no excess of the cardiovascular disease mortality and have good life expectancy at all ages.

Serum HDL-cholesterol and LDL-cholesterol level and their percentage distribution as per baseline findings were analysed. Majority of the subjects (53.3%) having systolic blood pressure

140-149 mmHg showed greater than 55 mg/dl of serum HDL-cholesterol. This study shows that HDL-cholesterol levels vary in different levels of systolic blood pressure. A total of 66.6% of the subjects had less than 55 mg/dl at 90-104 mm Hg diastolic blood pressure whereas 44.6% showed greater than 55 mg/dl. It may be noted that HDL-cholesterol makes up about 20-30% of total cholesterol. It is strongly and inversely related to individual risk of CHD, but its presence explains little of the differences in rates between population, the new knowledge of lipoprotein roles in transport and metabolism helps to elucidate the mechanisms of genetic- environmental interaction in the causation of atherosclerosis. It extends but doses not weaken evidence of mass behavioural effects on CHD risk factors or population evidence based largely on the simple measurements of total cholesterol.[221]

Blood total cholesterol levels in adults reflect well the levels of LDL-cholesterol, identified as the major source of cholesterol in atherosclerotic plaques. LDL is also mainly responsible for population differences in total cholesterol, for its trends in population and for relation of total cholesterol levels to individual risk. It accounts for between 60% and 80% of the total cholesterol. This study shows that 140-149 mm Hg of systolic blood pressure majority of the subjects (75.0%) had greater than 150 mg/dl of LDL-cholesterol whereas at high level of systolic pressure only 12.5% showed the same level of LDL. Results show

significant association. As per diastolic blood pressure about 71.4% showed greater than 150 mg/dl at 90-104 mm Hg. 44.8% and 30.6% of the subjects had less than 150 mg/dl of LDL-cholesterol whose diastolic pressure was 90-104 mm Hg and ≤ 110 mm Hg respectively. An association LDL and diastolic blood pressure was also observed in this study.

In a large proportion of individual with a raised cholesterol level, the serum conc. of TG is also markedly increased. This study shows 57.1% of the subjects had equal to and greater than 195 mg/dl of TG at 140-149 mm Hg systolic and 57.8% showed 95-144 mg/dl at 90-104 mm Hg diastolic blood pressure. Some workers have indeed claimed that correlation between TG and CHD is as good as that between total serum cholesterol and CHD. Recently it has been shown that TG levels act as a significant independent risk factor for CHD.[222,223] Both cholesterol and TG are associated with specific proteins in the plasma to form lipoproteins. The most important determinant of TG level is the activity of the enzyme, lipoprotein lipase in the endothelial lining of the capillaries and in a variety of tissues. This enzymes removes TG particularly from the VLDL and converts these to the lipoproteins of higher density. However, the present study did not show significant association between TG level and blood pressure. β-lipoprotein reflects the low-density lipoprotein in the blood. 66.6% of the subjects showed

high level of β-lipoprotein (600 mg/dl and above) at 140-144 mm Hg systolic pressure whereas at 90-104 mm Hg diastolic blood pressure the same proportion of subjects showed 600 mg/dl and above. This variation of β-lipoprotein with this level of blood pressures were not statistically significant. Baseline serum total lipids were also measured in all the subjects. About 50% of the subjects serum total lipids showed, equal to and greater than 900 mg/dL at high systolic blood pressure. At diastolic blood pressure 90-104 mm Hg, 51.5% and 50.0% of the subjects had serum total lipids at 801-900 and 901 + mg/dl respectively. No statistical significant difference was detected.

At various types of lipoproteins, it is the level of LDL-cholesterol that is most directly associated with CHD while VLDL has been shown to be associated with premature atherosclerosis.[176] It is more strongly associated with peripheral vascular disease than with CHD. HDL-cholesterol is protective against the development of CHD-the higher its mean level in a group of individuals, the lower the incidence of infarction in that group.[224]

The lipid in atheromatous arterial lesions is principally cholesterol and although the correlation between plasma and tissue conc.of cholesterol is weak. Even so, studies of population in many countries and in individuals have shown a close

correlation between plasma cholesterol levels and occurrence of CHD. Total body cholesterol has been shown to be proportional to body size and weight. The obese tend to have hypercholesterolaemia and levels can be lowered by weight reduction. Recent epidemiological evidence from the Framingham study and continued elsewhere, shows that the negative correlation between HDL conc. and CHD mortality are more important than positive correlations with plasma cholesterol or LDL. With newer techniques, HDL and LDL have been further subdivided into sub-fractions. Recent evidence indicates that levels of plasma apolipoprotein-A-1 (the major HDL protein) and apolipoprotein - B (The major LDL protein) are better predictors of CHD than HDL or LDL cholosterol respectively.

Therefore, measurement of apolipoproteins may replace lipoprotein cholesterol determinations in assessing the risk of CHD224,225.

5. **Baseline Fasting plasma glucose, serum urea and creatinine.**

The mean fasting plasma glucose in hypertensive subjects was 95.55 mg/dl and normotensive 95.77 mg/dl. As ascertained from the results that 50.0% and 41.0% had systolic blood pressure 140-149 mmHg and 160 mmHg and above. 46.6% of the subjects show 90-104 mm Hg diastolic pressure. In 90 to 110 mm Hg only 33.3% of the subjects showed fasting plasma glucose level equal to or greater than 120 mg/dl. No significant difference was established between

blood pressure and fasting plasma glucose. Although this is an imperfect test for diabetes mellitus, its aim is to detect obvious additional cardiovascular and renal risks associated with hypertension. It also serves to identify the patient who might develop diabetes mellitus with diuretic therapy. It is worth mentioning that cardiovascular diseases are lower in populations eating high carbohydrate diets. Support for the hypotheses that consumption of complex carbohydrates may decrease the risk of CHD comes from historical trends of food consumption patterns and mortality rates in US. Neither high carbohydrate nor high sucrose feeding had induced atherosclerosis in animals. An inverse association of fibre intake with the risk of CHD has also been observed.[226] KEMPNER has advocated a rigid rice-fruit-sugar diet for high blood pressure that gives impressive results even in severe cases.[227]

Baseline serum urea levels were estimated in all the subjects. Majority of the subject's (52.2%) serum urea at 15-59 mg/dl had systolic blood pressure 140-149 mmHg. 58.9% of the subjects serum urea level was within 15-29 mg/dl. With 90 to 104 mmHg diastolic blood pressure about 52% of the subjects had serum urea level at 30-39 mg/dl. Hypertensive subjects with blood pressure ≤ 110 mmHg their serum urea levels were 27.2% at 15-29 mg/dl and 26.0% at 30-59 mg/dl. However this variation of serum urea levels was not significant. Mean serum urea level was estimated to be 28 mg/dl

and 29.54 mg/dl in experimental and control subjects. Amount in blood 20-24 mgm (average 30 mg) urea present per 100 ml of blood. Almost equally distributed in plasma and corpuscles and serum urea level of subjects does not vary with normal values. Urea formation helps to maintain the reaction of blood constant. And adult taking normal mixed diet excreates urea through urine and average of 30 mg daily.

Majority of the subjects (51.7%) show creatinine level 0.5 to 1.4 mg/dl with systolic blood pressure 140-149 mmHg. 41.0% of the hypertensive subjects (160 mmHG) whose creatinine were at the level ranging from 0.5 to 1.4 mg/dl. With the same range of creatinine level and systolic pressure 160 mmHg and above, the proportion of subjects were 35.7% and 42.8% respectively. On the other hand 46.4% of the subject at range of 0.5 to 1.4 mg/dl of serum creatinine had diastolic pressure 90-104 mmHg. Of them 64.2% at the level of 0.5 to 0.9 and 40.4% at the level of 1-1.14 mg/dl of serum creatinine level. The mean serum creatinine was 1.13 mg/dl in experimental and 1.15 mg/dl in control groups. Different levels of blood pressure and creatinine levels don't vary significantly. Serum creatinine determination at baseline is a strongly recommended test. It gives information on renal sufficiency. It is not affected by water or protein intake and should therefore, be given preference over the measurement of blood urea nitrogen. Normally it is present about 0.7-2.0 mg/dl.

This level is very constant and it is considered to be pathological when its value increases about 2 mg. Creatinine represents the waste products of creatinine metabolism and it arises in the body from the spontaneous breakdown of creatinine phosphate[228]. It serves practically no function in the body apparently. As its excreatin is not related with food protein so its variations in the excretion indicate some of the metabolic disorders.

4.2 INTERVENTION EFFECTS

1. Effects on body weight and Blood Pressure

The results of this combined Metabolic well clinical trial, among uncomplicated essential hypertensive men provide enough support for antihypertensive effect through weight loss and aerobic exercises. Small but statistically significant reduction in blood pressure and body weight, BMI etc. occurred in both hypertensive and normotensive subjects. The mean drop of systolic blood pressure was 32.8 and 3.5 mmHg and diastolic blood pressure 24.7 and 5.3 mmHg in experimental and control. It has been observed that fall of systolic blood pressure in normotensive was very little. With the reduction of blood pressure, there was changes in weight loss i.e. the mean weight loss was 1.66 kg and 1.50 kg in hypertensive and normotensive subjects. In most published studies, weight loss has been shown to reduce blood pressure[229]. In a review

of adequately controlled intervention studies published through 1985, a 1.0 kg decrease in body weight was accompanied by an average reduction of 1.6/1.3 mmHg in blood pressure. There may be a threshold, around 4 kg to observe an effect[230] and a floor below which further weight loss may not be accompanied by further falls in blood pressure[231]. Use of very low caloric supplement may achieve faster weight loss and marked falls in blood pressure[232] while improving exercise endurance[233]. Moreover, weight loss may reduce the sensitivity of blood pressure to sodium[234], improving further the response to Metabolic therapy. Although the rate of recidivism among obese people may be high, an attempt at weight reduction in all obese hypertensive patients should be made, using whatever level of caloric restriction the patient is able to maintain.

It is important to note here that when people gain weight, their blood pressure usually increases and the frequency of hypertension is about two times higher in obese than in nonobese persons[235]. How weight gain induces hypertension is unknown, but the increased metabolic demand of obese individuals is known to raise the cardiac output (expressed in absolute terms) although the cardiac index, which is a relative measure, may be normal or even decreased.

The increased cardiac output is usually due to an increased in stroke volume; the plasma and total volumes are usually raised. When vascular resistance increases, a high cardiac output related to obesity may act as an important factor contributing to the development of hypertension.

Weight loss and blood pressure loss, these changes tend to go together. Weight loss brings a double benefit to the left ventricle by reducing both preload and after load, along with a fall in plasma norepinephrine levels[236]. If weight loss has an independent antihypertensive effect, a larger question still remains: How often does weight loss occur and persist in clinical practice? For the overall obese population, the results are poor[237]. In all studies from 1966 to 1977, the average weight loss was 5.4 kg and only about 20% of patients lost more than 9 kg. Though more people may have successfully lost weight than those involved in special weight loss programmes, the overall long term success rate is likely no better than 20% even with multidisciplinary programmes. The problem may relate to diminished energy requirements on obese patients after they have lost weight[238-340].

In brief, weight loss, although fairly easy to achieve, seldom lasts, however, even small amounts of weight loss will often lower blood pressure. It seems logical to use the likelihood of a fall

in blood pressure to further motivate obese hypertensive persons to reduce their calorie intake. The observation of a falling blood pressure along with falling weight should help keep these patients on a diet.

2. Physical exercise and changes in blood pressure

All hypertensive subjects showed a decrease in diastolic pressure with a range of 40 to 14 mmHg. A mean drop of 24.25 mmHg diastolic pressure and a mean drop of systolic pressure 32.52 mmHg occurred in hypertensive group. There was a mean decrease of 5.35 mmHg in the diastolic pressure of the normotensive group but no appreciable change occurred in the mean systolic blood pressure. This study results were similar with the following study, where 23 essential hypertensive and 22 normotensive middle-aged men participated in a controlled exercise programme for six months; the effect of the programme had on blood pressures was determined. The exercise medium used was the interval training of the walk jog type, with training intensity based on the actual heart rate expressed as % of working capacities. A drop in mean diastolic pressure of 11.8 mmHg and in mean systolic pressure of 13.5 mmHg occurred in the hypertensive group. There was a mean decrease of 6 mmHg in the diastolic pressure of the normotensive group, but no significant change in the mean systolic pressure[241].

Despite widespread interest in Nonpharmacologic therapy for hypertension, physical exercise had not been accepted as an effective antihypertensive therapy. There have been no well-controlled studies of factors such as age, sex, and race and the related mechanisms of antihypertensive effects of exercise remain unclear. A study was done to confirm the depressor effects of exercise and to analyse changes in hemodynamics in twenty Japanese subjects with essential hypertension. Changes in blood pressure, hemodynamics and humoral factors of the exercise group were compared with that of the control group. Blood pressure and whole blood and plasma volume indices were significantly reduced in the exercise group compared with controls. The change in ratio of serum sodium to potassium was found to correlate positively with the change in systolic pressure[242]. Four previous randomized controlled studies have reported lowered blood pressure associated with aerobic exercise training[243-246]. Another study suggests that a programme of moderate aerobic exercise, without dietary changes, in patients of normal body weight and average level of fitness, appears to offer relatively little benefit. Given the suggestive evidence that the greatest reduction in clinic blood pressure were exhibited by those subjects who achieved the greatest improvements in aerobic power, a more vigorous exercise programme, perhaps over a more extended time period, might be effective[247].

However, this study indicates that essential vascular hypertension is not necessarily a contraindication to controlled interval exercise programme. It is note worthy that the principle change of both hypertensive and control groups is in the diastolic pressure. The known vasodilating effect of endurance exercise is probably responsible for the drop in blood pressure.

During dynamic exercise there is a profound vasodilatation in the working skeletal muscle with a concurrent reduction of perfusion in other vascular beds. Cuteneous vasodilatation will occur, however, when central body temperature rises. Venous return is augmented by the pumping action of the exercising muscles and by the increased abdominothoracic movements of hyperpnoea. During post exercise period, however, venous return falls dramatically owing to the discontinuance of mechanical pumping effects, while skeletal muscle and cutaneous vasodilatation persists. Hence a fall in systemic arterial systolic and diastolic blood pressure is not surprising[128]. The greater fall in blood pressure after exercise might be explained by greater vasodilatation owing to the higher plasma adrenaline conc.[128]

The study also shows that in hypertensives blood pressure can be lowered effectively by treadmill exercise without undue risk if their condition is monitored during exercise and if specific signs and symptoms are noted during each exercise session.

Persons who are physically active and fit may develop less hypertension and those who are hypertensive can lower their blood pressure by regular isotonic exercise. Increasing physical activity is one of the roads towards positive health. Physical inactivity is a substantial risk factor for cardiovascular disease[248-252]. Exercise probable works by increasing physical fitness and modifying other risk factors. Among the other benefits it lessens the risk of stroke and osteoporosis[253-254] and is associated with a lower all cause mortality[256]. Exercise increases the energy needs of exercising human muscle. Therefore working muscles must possess a tremendous capacity for increasing their metabolic rate in order to produce enough ATP to allow increased activity to continue[255]. This capacity relies heavily on the respiratory and cardiovascular systems for the delivery of oxygen and nutrients and for the removal of waste products to maintain the internal equilibrium of the cells. Other central and peripheral changes reported to occur in response to long-term exercise are: a decrease in myocardial catecholanine conc: a greater total blood volume, an increased number of capillaries per skeletal muscle cell, and increase in vital capacity, an increase in lung ventilation and in blood flow to the working muscle with constriction of blood flow to those areas that are inactive. An increase ability to supply blood flow to the skin for cooling. Sweat increases in amount, starts faster, and contains less salt[255,256].

An increase in the conc. of oxidative enzymes, muscle glycogen stores, increased release of free fatty acids. An increased ability to metabolize fat. A more efficient oxygen extraction from the blood at the tissues. An increase in insulin sensitivity and glycogen tolerance and a decrease in recovery heart rate.

Data from Framingham study shows that persons who are more physically active have a lower risk of developing or dying from coronary heart disease. Persons who had objective evidence of poor physical fitness, such as more obesity, faster heart rate and lower vital capacity, had a higher risk of coronary heart disease mortality. Beyond possible prevention, exercise may lower blood pressures that are already elevated, but these studies were poorly controlled.

However, when hypertensives who are not trained or conditioned do isotonic exercise, they usually have hemodynamic changes that are moderately greater than those seen in untrained normotensives. Among normotensives, those who had a marked rise in systolic pressure during exercise stress test had a greater likelihood of developing hypertension compared to those who remained normotensive or had a rise in diastolic pressure, suggesting that the response to isotonic exercise may be a prognostic indicator for the development of hypertension. After training, hypertensives show indication of a lower level of sympathetic activity which may

casual role of sodium in the genesis of hypertension. This evidence includes an increase in intracellular sodium in hypertensive animals and people, an increase that extends even to the normotensive children of hypertensive parents[256]. The evidence for a role of sodium in primary (essential) hypertension are:

a. In multiple populations, the rise in blood pressure with age is directly correlated with increasing levels of sodium intake.
b. Multiple, scattered groups who consume little sodium (less than 50 mmol/L/day) have little or no hypertension when they consume more sodium, hypertension appears.
c. Animals given sodium loads, if genetically predisposed, develop hypertension.
d. Some people, when given large sodium loads over short periods, develop and increase in vascular resistance and blood pressure.
e. An increased conc of sodium is present in the vascular tissue and blood cells of most hypertensives.
f. Sodium restriction, to a level of 60 to 90 mmol per day will lower blood pressure in most people. The antihypertensive action of diuretics requires an initial natriuresis.

There have been a number of observations that hypertension is virtually non existent in primitive societies that consume diets

low in salt[257]. This topic has been the subject of much controversy, but there is some consensus that a diet high in calories, fat and sodium and low in potassium is associated with the development of hypertension[258-263]. People in primitive societies are for the most part physically active throughout life and are lean vegetarians who are not exposed to alcohol. Therefore, it is not clear which dietary component is actually responsible for the development of hypertension[263]. The role of sodium in causing hypertension is difficult to study within a population, because there may not be a sufficiently diverse intake of sodium i.e., the dose-response relationship between sodium and blood pressure may be flat over the range of intake in a population. Studies of migrations and recruitment of primitive people into modern armies, however, lend support to the importance of chronically high sodium intake in increased blood pressure. Major criticisms of the epidemiological data include the difficulty in measuring sodium intake and the lack of standardization of blood pressure measurement[262-264].

Clinical trials of sodium restriction in hypertensive subjects were reviewed and there is reasonable evidence that low sodium diets will lower blood pressure in many patients and reduce their need for antihypertensive drugs[265].

Although the blood pressure response to sodium restriction is quite heterogeneous, there is no known hazard to a moderate sodium restriction and it will benefit the salt-sensitive subset of patients. From a practical stand point reducing sodium intake in the American diet would most likely to also lead to a reduction in dietary fat content, which is considered desirable from several health stand points[266]. If dietary sodium were a major factor in the development of hypertension in industrialized countries, however, one would expect that increased intake would increase blood pressure in normotensive subjects, especially those with strong family histories of hypertension and who are at increased risk of developing it themselves. This has not been found to be the case. In addition, the fact that strict vegetarians or thin non-drinkers can have a low prevalence of hypertension and a high sodium intake suggested that sodium intake alone has a minor role in the development of hypertension[263]. But once hypertension is present, modest salt restriction may help lower the blood pressure. In the review of 24 intervention studies an average reduction in sodium intake of 100 mmol per day produced a decrease in blood pressure of 5.4/6.5 mmHg. Two additional large scale studies published confirm the effectiveness and feasibility of moderate sodium restriction in treating hypertension[267,268]. In a smaller but more controlled study, the fall in blood pressure was shown to be greater at a daily sodium intake of 50 mmol than at 100 mmol per day[182]. However, rigid degrees of sodium restriction

are not only difficult for patients to achieve but may also be counter productive. The marked stimulation of renin-aldosterone that accompanies rigid sodium restriction may prevent the blood pressure from falling and increase the amount of potassium wastage if diuretics are concomitantly used[269]. Not all hypertensives will respond to a moderate degree of sodium restriction to a level of 70 to 100 mmol sodium or approximately 2 gm/day. Blacks tend to be more sodium sensitive[270] and elderly patients may be particularly responsive to sodium restriction perhaps because of their usually lower renin responsiveness[271].

Even if the blood pressure does not fall with moderate degrees of sodium restriction, the patient may still benefit; improved beta-adrenergic responsiveness[272], increased antihypertensive effectiveness of other drugs[737] and less diuretic-induced potassium wastage have all been reported among patients on moderate sodium restriction.

Although there is no assurance that moderate sodium restriction will help, there is no evidence that it will hurt. For example, neither the intake of other vital nutrients[274], nor exercise tolerance in a hot environments are reduced by a lower sodium intake[274]. Therefore, it will be useful for all persons, as a preventive measure in those who are normotensive and more certainly, as partial therapy in those who are hypertensive.

Population wide reduction may be possible[275,276] with considerable potential therapy to reduce cardiovascular mortality[277].

It has been noted that the changes of serum potassium level were ranged from 1.73 mmol/L to 0 mmol/L in experimental and 1.07 mmol/L to 0.04 mmol/L control subjects. The mean changes in serum potassium was 0.50 mmol/L and 0.27 mmol/L in hypertensive and normotensive subjects. The overall changes of serum potassium were statistically significant. In both the groups with the fall of blood pressure, the serum potassium level was slightly increased. The co-efficient of correlation shows a limited degree of negative correlation exists in control subject (r=-0.25). The data indicates that the fall of blood pressure might be due to the effect of increased potassium level.

It should be cleared that how serum potassium levels have been increased in this study. This is either due to dietary changes or low sodium diet with 2000 Kcal per day.

It has been proposed that primitive societies lack hypertension because of their high potassium rather than low sodium intake[278]. As in the case with sodium, the epidemiological data have many limitations. A number of workers, however, believe that the dietary potassium-sodium ratio is an important predictor of the

tendency to blood pressure elevation and studies found an inverse correlation between blood pressure and the ratio of these electrolytes in urine, but no correlation with the excretion of either electrolyte alone[262,263].

Trials of dietary potassium supplementation all have involved small numbers of patients for a short duration and these data have been summarised[279]. Such supplements have no effect upon the blood pressures of normotensive subjects, although it has been shown recently that potassium depletion of normal individuals causes sodium retention and increased blood pressure both at baseline and in response to a saline infusion[280].

The effects of potassium supplements on hypertension individuals also appear to involve a natriuresis that is dependent upon the subjects sodium status. If the patients have low potassium intake and have not had sodium restricted, then additional potassium seems to have a moderate hypotensive effect[279-281].

It is important to note that some of the advantages of a lower sodium intake may relate to its tendency to increase body potassium content both by a coincidental increase in dietary potassium intake and by a decrease in potassium wastage if diuretics are being used. Although potassium deficiency clearly

exerts multiple effects that may increase blood pressure[280] evidence that correction of hypokalemia[282] or addition of dietary potassium to normokalemics will lower the blood pressure in skimpy[283]. Nonetheless diuretic induced hypokalemia may be more of a danger than many suspect, so that for various reasons, hypertensive patients should be protected from potassium depletion. Moreover, certainly in experimental animals[284] and perhaps in humans as well, extra-dietary potassium intake protects against vascular damage and strokes[285]. However, not all of the trials used satisfactory methods and the more rigorous trials tended to show smaller effects. Nevertheless, dietary potassium remains one of the most consistent correlates of blood pressure level in population studies[286-296]. Hypotheses proposed to account for this paradox include the possible importance of the anion given with potassium; i.e., chloride might counter some of the hypertensive effect[295] and an interaction with the level of sodium in the diet[297].

4. Nutritional changes and Hypertension

Both the groups reported some knowledge of the disorder, its risk association with heart disease and the benefits of restricted salt intake, low fat, vegetarian and high-fibre diets. However, information related to the dietary modification required was mainly attributed to the subjects cooperation and hospital dietary

supply. Both groups expressed greater confidence and better awareness of the disorder and its control as a consequence of the information, dietary guidelines, schedule and hospital dietician. Intervention participants were given upto 2000 Kcal per day with a view to achieving both weight loss and improved composition emphasized reduction in total fat, saturated fat and dietary cholesterol. Kempner has advocated a rigid rice-fruit- sugar diet for high blood pressure that gives impressive results even in severe cases. Some authorities point out that the improvement with the above diet is not due only to low protein, low fat and low sodium but also due to weight loss and rest under hospital conditions.

Calorie-restricted diets are usually low in sodium as well, but a number of well-designed trials indicated a significant lowering of blood pressure in hypertensive subjects that was attributable to weight loss[286]. This study shows the similar effect regarding fall of raised blood pressure due to dietary changes, weight reduction etc. It is important that dietary interventions be designed with such effect in mind and that the subject's weights be measured during the study. Because achieving ideal body weight will nearly normalize the blood pressures of many obese male hypertensive patients, weight reduction is a first step in hypertension management. In metabolic control of hypertension dietary intervention was given with a low sodium, low fat and high-fibre

diets. A reduction of total dietary fat and an increase in the ratio of poly unsaturated to saturated fat may lower blood pressure by increasing the synthesis of vasodilatory prostaglandin[287].

It may be mentioned that Lacto-ovovegetarian (LOV) diets successfully reduce blood pressure because of their high potassium content[288]. The lower blood pressure observed among vegetarians has been ascribed to their high potassium intake[289]. Lov diets consist of more than average vegetables protein, whole- grain cereals, poly unsaturated oils, fruits and vegetables and avoid meat, fish and poultry. They also contain significant fibre, vitamin-e, vitamin C, magnesium, calcium and potassium and less total protein, saturated fat, mono saturated fat and vitamin B12 than control omnivore diets[290,291]. Blood pressure changes were significantly and negatively related to individual scores for diets representing an increase in the intake of polyunsaturated fat or fibre or vitamin C or vitamin E or a decrease in intake of protein and vitamin B12. Blood pressure changes were unrelated to change in body weight or sodium intake[290]. Changes in the dietary p:S ratio can influence blood pressure in hypotensives living on Mediterranean diet[292]. The success of a diet high in fibre may be attributed to the fact that dietary fibre may lower sodium absorption[286]. Patients may also benefit from a reduction of dietary protein intake, particularly those with renal disease[294].

Dietary changes and blood pressure have many problems during interpretation of data. It is difficult to select two groups of people eating diets that differ only one dietary component. Most groups eating diets with a high fish content, for example, also have a high intake of salt, which could obscure any hypotensive influence of fish. The consumption of an unusual type of food over many generation may result in genetic adaptation to some components that would therefore have different effects in different population groups. It is possible that a high intake of n-3 fatty acids by Eskimos on a marine diet may have fewer or different long-term effects for them than would the same amounts of these compounds consumed as dietary supplements by American volunteers[281]. Many nutrients have a high degree of association in food stuffs and it is important to remember that people eat food, not specific nutrients. It is therefore, very difficult to isolate the health effects of particular dietary components. Many dietary habits that are believed to influence blood pressure directly also are related to other personal characteristics that are known to have an effect, such as alcohol intake, body size, physical activity and obesity. It has been noted that total energy intake is intimately related to the consumption of all major nutrients and is driven by physical activity level. School children found to have lower blood pressure also were found to have a higher intake of all nutrients, primarily because of their being more active[292].

As a result it would be difficult to interpret any inverse associations found between particular nutrients and blood pressure. An additional problems in population studies is estimating nutrient intake accurately and being rare that standardized methods of blood pressure measurement are used in the different groups being compared[263,298]. Because of these numerous difficulties in interpretation, epidemiological studies are useful in finding associations and generating hypotheses but these hypotheses must be tested in carefully controlled dietary-intervention studies.

5. Effects on Lipids and changes in blood pressure.

Studies on coronary heart disease risk factors have identified hypertension, cigarette smoking and elevated serum cholesterol as the major risk factors to contend with. [299] Effects on lipid profiles summarized changes in risk factors during the intervention period. In the experimental group, mean fall of cholesterol was 23.23 mg/dl and LDL-cholesterol 12.32 mg/dl. These falls occurred even though patients had already receiving reduced fat and low calories and high fiber containing diets. HDL- cholesterol changed significantly in either group. Serum -- lipoprotein fell substantially in both the groups. A high significant changes in serum total lipids exhibit in hypertensive and normotensive subjects. Similar significant results were also observed as far as serum TG level is concerned. As in prior studies, exercise without drug decreased total cholesterol and LDL-cholesterol [300]. After drug run-in, the HDL-cholesterol level of the propranolol group decreased. Exercise induced increases in the HDL-cholesterol levels in the placebo group, did not occur in the drug group. HDL-cholesterol levels of the other group increased in at drug run in. After training, HDL- cholesterol levels in the placebo and other groups were similar. Exercise decreased total and LDL-cholesterol levels in both the groups. Increases in the level of HDL-cholesterol were similar. This study confirms data of other study300' Specific dietary

recommendations were not a part of the present study and as for blood pressure, the slight weight reduction seen in both the groups is an unlikely explanation for the lipid changes. There is adequate evidence that covering serum cholesterol can slow the progression and cause regression of established atheromatous plaques in the coronary arteries. Blood total cholesterol levels in adults reflect well the levels of LDL, identified as the major source of cholesterol in other sclerotic plaques. LDL is also responsible for population differences in total cholesterol. LDL-cholesterol makes up about 20-30% of total cholesterol. It is strongly and inversely related to individual risk of CHD, but its presence explains little of the differences in rates between population. HDL cholesterol has been reported to be higher and TG lower in endurance exercisers than in sedentary individuals [301,302]. Elevation in HDL- cholesterol appears to be largely due to change in HDL_2 cholesterol conc.[295]. Data on the association between physical activity and HDL cholesterol are conflicting.[302] No consistent relation has been found between physical activity and LDL cholesterol, especially when controlling for confounding factors [303]. Lower LDL-cholesterol has however been reported in those participating in very strenuous exercise than in others [304]. In this study a similar effects on LDL cholesterol was noted. Lipid differences are usually less prominent in intra population studies because of the limited variability of physical activity within population[302]. A few population studies have, however, shown

a positive association between more strenuous physical activity and HDL cholesterol [305-307]. Findings on the effect of physical activity on lipids studies are somewhat inconsistent. It has been suggested that HDL-cholesterol even decreases during the first months of training[308]. About half of controlled training studies have, however demonstrated a significant increase in HDL-cholesterol in exercisers. Only a few large scale population studies have investigated the association of physical activity with lipids. Some studies suggest a dose-response relation[309] whereas others suggest a threshold for the amount[310-311] and intensity of physical activity for the favourable impact on lipids[312].

A previous study suggested a dose-response relation between the amount of habitual physical exercise and HDL-cholesterol when comparing physical activity extremes from quadriplegia to marathon running[309]. A weaker gradient for HDL-cholesterol in population studies is likely to be due to the limited range of physical activity within population. It is found, however, a positive linear dose-respone relation between combined metabolic therapy and HDL cholesterol in the study sample of middle aged male, hypertensive and normotensive participants. Some studies have reported a positive [309-313] whereas others an inverse association between physical activity and HDL cholesterol [310-314]. A few studies have shown no relation between physical activity and HDL cholesterol [302]. In the present study a limited degree of positive

correlation was found with the initial and final levels of HDL and LDL cholesterol levels due to the effects of metabolic therapy. The discrepancy may be caused in part by different methods used in the determination of HDL cholesterol subfractions. The separation of HDL_2 cholesterol and HDL_3 cholesterol may be more precise with ultracentrifugation than with precipitation[315]. The inconsistent results concerning the association between HDL cholesterol and exercise therapy may be also due partially to the greater measurement variability for HDL than for other lipids. The separation of HDL-subfractions was beyond the scope of the present study.

A recent study showed that the earlier effect of metabolic therapy is to decrease TG and the author sepeculated that this probably precedes an increase in HDL-cholesterol[316]. A similar finding was also noted in this study. Few studies suggest that the favourables changes in lipids, other than TG, are due to chronic adaptation to regular physical activity rather than acute exercise effect[317]. This study noted that the relation of brisk and sustained walking with serum lipids, was the stronger the shorter was the time frame of the intervention programmes. These data suggest indirectly a short-term rather than a long-term effect of physical activity on lipids. TG related positively with all metabolic therapies in this study. But it is in accordance with

a view that the favourable changes in lipids may persist only for a short duration after exercise sessions[303]. Most of the previous studies have failed to show a relation between physical activity and LDL-cholesterol, independent of age, body adiposity, and dietary factors[303]. In this study, the relationship between LDL cholesterol body weight and the changes in diastolic blood pressure level was observed. It indicates that there is slightly upward trends of diastolic blood pressure with the increase of LDL cholesterol levels. A moderate degree of correlation is detected. LDL-cholesterol however, was associated inversely with vigorous activity for age, race, sex and education, each of which was significantly associated with LDL cholesterol[318]. Even mild, if regular, physical exercise decreased LDL cholesterol after controlling for weight change[314]. LDL- cholesterol was associated also inversely with kilometers of walking, jogging, skiing, bicyclining and swimming per week, when the major confounding factors were controlled. This is consistent with the recent study that demonstrated inverse association between LDL-cholesterol and distance run per week[319]. The present data support the view that walking on a tredmill set with progressive increase of time has a modest LDL cholesterol lowering effect. It has been suggested that the amount rather than the intensity, of physical activity is the primary determinant of HDL cholesterol[320]. A number of cross sectional studies in runners have reported a

positive association between miles run per week and HDL cholesterol[302]. In an exploratory analysis, it has been noted that more than approximately 30 km of walking, jogging, skiing, bicycling or swimming per week may be needed to achieve a detectable increase in HDL cholesterol.

Some studies suggest that a minimum level of energy expenditure is needed to improve the lipid profile. Exercise with an energy expenditure of more than 6 METS was required to influence lipids in middle aged man[307]. No further improvement was observed when comparing this intensity with the highest levels of energy expenditure. Another recent study suggested threshold for HDL-cholesterol at a leisure time energy expenditure of 5 kcal/ minute[305].

According to present data, there may a threshold for an increase in HDL-cholesterol and a decrease in TG. The inconsistency in cross sectional studies concerning an association between physical activity and lipids may be due to the lack of control for confounding factors. General exogenous factors, like dietary cholesterol, saturated, polyunsaturated[322,323], and monounsaturated fat, carbohydrates, body adiposity, coffee[324,325], alcohol, smoking, beta-blockers, thiazide diuretics, androgens and estrogens[322] and indigenous factors, like age, sex, heredity, race, diabetes and

insulin level have been reported to influence blood lipids. It has been observed that several factors confounded the relation between exercise and serum lipids, especially that of physical activity with TG and HDL-cholesterol[326]. Cross sectional studies may be biased by a self selection effect i.e. those with an unfavorable lipid profile may be more in active, because of an existing cardiovascular disease. The relations persisted after controlling for a number of confounding factors, including prevalent symptomatic ischemic heart disease, which supports an independent association between physical activity, hypertension and serum lipids[325,326]. Nutritional counselling, especially when conducted by a dietician or nutritionist, frequent follow up and encouragement to make gradual changes, will produce improved compliance and more effective cholesterol lowering with dietary intervention. It should be noted that dietary modification has been proven to reduce mortality[327-329].

An estimate of the benefit achieved by lowering total serum cholesterol is derived from the lipid Research Clinics Report,[330] which confirmed that a 1% decrease in serum cholesterol yielded a 2% decrease in the risk of CHD. This study conclusion has been re-evaluated[331] through a meta-analysis of up to 16 international trials evaluating reduction in CHD by various intervention. Researcher suggests that for each 1% reduction in serum

cholesterol, the reduced risk of CHD is actually closer to 2.5% [331]. Dietary therapy need not be advanced to severe degrees of fat and cholesterol restriction, since justification for this is lacking. One study showed that both mild and severe fat and cholesterol restricted diets produced similar reduction in LDL cholesterol[331]. Weight loss by exercise or dietary changes might also reduce hepatic lipase activity. Hepatic lipase hydrolyzes the HDL phospholipids. Furthermore, the rate of cholesterol transfer from HDL to hepatocytes is reported to decrease as the phospho lipid/cholesterol ratio of HDL particle is increased. The present findings also coincide with other published data. For equivalent losses of body weight the increase in HDL-cholesterol and decreases in LDL cholesterol in hypertensive and normotensive subjects were almost similar. Weight loss by exercise and dietary changes significantly increases HDL conc. and decreases LDL and serum TG. The changes are also correlated. Although some investigators have ascribed the lipoprotein changes during diet induced weight loss to altered nutrition and cross sectional surveys and experimentation do suggest that dietary composition might affect lipoprotein conc. or distribution, perhaps through adiposity changes. The hypothesis that exercise might accelerate HDL-cholesterol and affect other lipoproteins through weight loss is controvercial. It was noted that the relation of reduced adiposity to HDL conc in 23 published cross sectional comparisons

of long distance runners and sedentary man. The runners and sedentary men's mean HDL-cholesterol differences were largely explained by their adiposity differences. For these results it has been proposed that long distance runners have the lipoprotein metabolism of men who are below their usual weight and not the metabolism of equivalently lean men who are neither exercising nor dieting. The low TG conc of experimental and control subjects might be explained in part, by high lipoprotein lipase activity causing chylomicron and VLDL particles to be catabolized and cleared more rapidly[122].

For the clinician, the relative importance of one disturbance compared to another in an individual patient represents a toubling dilema. Further complicating the clinical situation is the overlap of lipid disturbances, making contrast between the importance of any individual disturbance impossible. In addition one risk factor can modify the importance of another[325]. Today the major emphasis of the clinician must be on the reducing the LDL cholesterol. The present study also gives emphasis to reduce LDL cholesterol and the same was reduced significantly in both experimental and control groups. It should be noted that a positive correlation is observed between LDL-cholesterol and hypertension. Some studies showed that LDL cholesterol blood levels also correlate strongly with the degree of atherosclerosis

progression, and there is substantial justification that lowering LDL cholesterol is associated with a reduced coronary risk[333]. The value of reducing TG levels or independently increasing the HDL-cholesterol achieved by pharmacological intervention in some studies has not been conclusively demonstrated[332,334,335].

The LDL-cholesterol level should be the focus of attention since it is the lipoprotein fraction most closely correlated with coronary risk and it has been shown that reducing LDL cholesterol can significantly reduce the incidence, morbidity and mortality of CHD. Metabolic therapy will provide the similar health benefit in patients with cardio-vascular risk factor i.e. hypertension. The improved CHD risk was perhaps heightened by the simultaneous HDL cholesterol elevations. The HMG COA reductase inhibitors similarly are associated with LDL cholesterol reduction and HDL-cholesterol elevations, as shown in several large scale randomized trial,[336-341].

It may be further mentioned here that the importance of the serum cholesterol level was recognized when one of the earliest studies (The Framingham Heart Study) positively correlated serum cholesterol in 2282 men and 2845 women with the indicence of CHD. The direct positive correlation was present for cholesterol values

of 4-7, 6 mmol/L[342]. A subsequent study of extensive proportion has continued this positive correlation[327].

The multiple risk factor intervention trial related the death rate from CHD over a 6 year period in approximately 360000 middle aged man to the serum cholesterol level, and found an increased mortality to occur at cholesterol levels as low as 5.2 mmol/L[327]. Despite this evidence and that which relates treatment of hypercholesterolaemia to improve coronary health, there is a reduced attentiveness to this risk factor by practicing physicians. Physicians have had an increasing awareness that control of hypertension is associated with a reduction in overall morbidity and mortality from cardiovascular disease and especially from stroke,[327,345]. Unfortunate, and also poorly appreciated , is the finding that the observed reduction in CHD morbidity and mortality is not as convincingly demonstrated when only hypertension is controlled[346,347]. A reduction in the serum cholesterol level has been independently associated with reduction in the incidence of and morbidity and mortality from atherosclerotic cardiovascular and coronary diseases,[347.330].

Physicians and health professionals must be educated with regard to the wealth of information directly relating the coronary risk of their patients to their cholesterol levels. The degree of

physician awareness especially must be heightened, since from this source spring many secondary adductive processes to allied health professionals and the public. It is of course, physician recognition of risk on an individual basis that determines the time of treatment initiation, its goal and success[348-351]. Modifiable risk factors must be controlled. Hypertension must be controlled with agents that do not promote disturbances of lipid levels, insulin resistance or glucose metabolism. Obesity must be minimized and exercise programmes promoted [352-358]. The relevances of the present study have demonstrated a significant effects on primary prevention of essential hypertension.

6. Metabolic Therapy, Effects on Plasma Glucose and Blood Pressure Levels

A frequently unrecognized health benefit of exercise is its effect on carbohydrate metabolism. During large muscle, dynamic exercise of moderate intensity, the glycogen stored in skeletal muscle is used for the production of energy and becomes partially depleted [359]. In this study the metabolic effects on fasting serum plasma glucose were studied on hypertensive and normotensive subjects. The mean reduction of fasting plasma glucose was 9.32 mg/dl and 5.38 mg/dl in experimental and control. These changes or reduction of serum fasting plasma glucose were highly

significant. A relationship was detected between diastolic blood pressure and plasma glucose i.e. with the increased level of plasma glucose, diastolic blood pressure is also increased. It has been detected that a transient rise of blood pressure for about 2 hours was noted after the ingestion of glucose or sucrose 1g/kg [360,361]. At the time the others found that easily digestible carbohydrate is of great help in the dietetic management of high blood pressure and coronary heart disease rates are lowest in population eating high carbohydrate diets[361]. The metabolic effects of exercise on carbohydrate show that after exercise for about 24 to 72 hours, the body glycogen is replaced by the uptake of glucose from the blood. In addition to this acute effect of increased glucose removal, the insulin receptors in skeletal muscle and adipose tissue increase in sensitivity and thus remove glucose more effectively at any given conc. of plasma insulin. This insulin sparing effect of endurance exercise training decreases insulin production and may reduce the risk of insulin deficiency developing with increasing age. While studying the pathogenesis of essential hypertension it has been noted that peripheral resistance to insulin has been explained. The level of insulin resistance and the defect in whole body glucose utilization are positively correlated with the severity of the hypertension. Insulin resistance to hypertension appears to be independent of both obesity and glucose tolerance has measured by

standard glucose tolerance tests. Whole body insulin induced glucose uptake and no oxidative glucose disposal (gycogen synthesis and glycolysis) are markedly reduced and plasma insulin levels are elevated presumably as a compensatory mechanism, in insulin resistant subjects. Insulin resistance may be a key disease state underlying hypertension and ultimately, CHD[362]. Insulin resistance is determined by measuring plasma insulin and glucose. In one study investigator compared 13 men and women with moderate to a severe hypertension who did not have any other reason for insulin resistance, such as obesity or glucose intolerance, with 11 normotensive controls. After a glucose load both plasma insulin and glucose levels were significantly higher in hypertensive compared with normotensive subjects satisfied the criteria for impaired glucose tolerance[363,364]. The present study though does not measure plasma insulin levels but observed a positive correlation with blood pressure and plasma glucose levels but a inverse relation with the normotensive subjects.

Furthermore, there was substantial insulin resistance in these subjects. Stimulation of whole body glucose uptake by insulin was 40% less in the 13 hypertensive subjects compared with the 11 age matched normotensive controls. Peripheral tissue such as muscle, rather than liver appeared to be the site of the insulin resistance. The defect in whole body glucose utilization was

directly related to the severity of hypertension[364-370]. It is known to all that hypertension is more common in the obese and that hyperinsulinemia is a hallmark of obesity. The association are most striking in people with upper body obesity[371], who have the highest prevalence of hypertension and the most pronounced hyperinsulinemia[372,373]. The high level of insulin in subjects with upper body obesity arise both from increased secretion of insulin, common to all forms of obesity and from decreased hepatic removal and degradation of insulin, which appear to be related to the high rate of lipolysis of intra-abdominal fat[374,375]. The excessive quantity of free fatty acids coming from this fat are believed to be responsible for both the hyperinsulinemia and hypertriglyceridemia and low HDL cholesterol levels common in upper body obesity[376]. Insulin can raise the blood pressure either increasing circulating catecholamines and stimulation of renal sodium reabsorption[377,378].

The presence of hyperinsulinemia in obesity and its potential causal role in the hypertension of upper body obesity is rather obvious. Less obvious but of even greater possible importance is the fact that hyperinsulinemia is also common in nonobese patients with primary hypertension. Regardless of how it occurs, insulin resistance with resistant hyperinsulinemia is likely to be pressure growth promoter involved in the pathogenesis of primary

essential hypertension both in the obese and non-obese[364].

Physical activity is known to decrease serum insulin while having little effect on glucose tolerance, which could indicate improved insulin sensitivity through exercise[379,380]. Various data suggest that the beneficial effects of physical activity on serum lipids are mediated in part by a reduced serum insulin conc. Which supports the view that physical exercise affects lipids by improving insulin sensitivity[381].

Exercise has been also reported to improve the lipid profile by reducing body weight[381]. Furthermore, serum insulin associated strongly with BMI and its control weakened the relation of BMI with serum lipids. The present data suggests indirectly that weight loss through physical activity improves the lipid profile partly by reducing the serum insulin conc.

7. Relaxation and Stress Management Effects on Blood Pressure.

Investigator noted the effects on blood pressure changes of three week's daily progressive muscle relaxation, deep breathing exercises, and meditation were identical with those observed in a control group. The relevant changes in blood pressure or its variability were noted either in the day time or during the

evening or the night, when presumably sympathetic activity is reduced. This findings confirm of two recent studies in which modest reduction in blood pressures were found in the clinic but not outside it[382,383]. It is also consistent with those of several other studies reporting sizable reductions in blood pressure at the clinic [384-389].

The problem with behavioral treatments is that several variable that are assumed to be critical for success are difficult to measure[390,391] for example the type of therapy and the clinical skills of the therapist. These complicated and subjective factors make generalization from single behavioral studies difficult, regardless of their outcome.

In combination with other Metabolic therapies, relaxation therapy may be useful for some to enhance well being and compliance but will not in our opinion be more effective or less ineffective than support or reassurance in the traditional sense [392,393,394]. It may be mentioned that relaxation techniques have proved highly successful in the treatment of stress related symptoms and associated clinical syndromes. The most widely used behavioural relaxation techniques include progressive muscular relaxation, meditation, breath control and biofeedbacks[395-400]. The rationale for the use of these techniques and their application within

general practice are discussed, with a brief review of the research findings. Most people normally operate at levels of non-pathological stress which often contribute to improved functioning. However, if the levels of arousal and effort are excessive and prolonged, the individual's ability to cope can suffer, producing a potentially damaging level of neuro physiological stress and symptoms of ill health and disease. A state of healthy equilibrium can be upset by a wide range of psychological, physiological and environmental conditions[400].

When subjected to a mental or physical stress, the autonomic nervous system (ANS) initiates a complex series of neurophysiological and biochemical changes. The two branches of ANS the sympathetic and parasympathetic nervous systems are responsible for the regulation of these changes. The sympathetic nervous system is concerned with arousal and preparing the body for action. For example, it accelerates the heart and pulse rate, constricts involuntary muscles, dilates the pupils increase the respiration rate and activates the secretion of adrenalin and noradrenaline. This fight and flight response, constitutes the body's most comprehensive reaction to a perceived threat. The effects of activation on the para sympathetic system are inhibition, slowing and restorative functions, commonly called the trophotrophic response. This includes slowing the heart and

pulse rate and dilating the blood vessels. When the fight or flight response is prolonged and the individual is unable to take appropriate action by either fighting or fleeing to release the physiological stress, the consequences can be deleterious to health [400,401].

a. Neuromuscular Relaxation

Relaxation techniques produce a temporary trophotrophic state characterized by a generalized state of decreased psychophysiological activity mediated by the para sympathetic nervous system. The state of deep relaxation is highly conducive to positive health since it is the physiological opposite or the sympathetic stress response and facilitates psychophsiological restroration of the body[401,402]. Neuromuscular relaxation refers to a series of exercises which reduce the neural activity and contractile tension in the skeleted muscles. The most widely used procedures are those developed by Edmund Jacobson, termed progressive muscles relaxation[400]. This system consists of a series of exercises by which the person contracts and then relaxes selected muscles and muscle groups so as to achieve a state of deep relaxation. This is based on an enhancement of awareness of of proprioceptive neuromuscular impulses originating at the peripheral muscular levels and increasing with striate muscle

tension. Many authors pointed that progressive muscular relaxation is the most effective treatment for anxiety with a large somatic component[400,403]. Neuromuscular relaxation has been shown to be effective in the treatment of essential hypertension. including insomnia, migraine, tension headaches. However, psychophysiological disorders have a complex relationship with stress and other behavioral factors. Therefore, although relaxation and stress management techniques offer clear therapeutic benefits, they may not be optimally effective unless combined with more complex procedures which attempt more radical alterations in the patients response to stress[404,405].

b. Meditation

Meditation is a technique which has been used for thousands of years within the religious and philosophical traditions of the East, and for only the past 30 years within the medical health care, scientific and psychotherapeutic traditions of the west, where it has been used without religious connotations in the promotion of health. Several hundred papers and books have appeared in the medical literature describes the physiological and psychological effects of meditation, as well as the physical, emotional and spiritual well being of practitioners[406,407].

Meditation represents giving the individual technique which have the potential for inducing an awakened trophotrophic state.

Although there are many types of meditation one element common to all is the object or focal device on which the mediator focuses awareness or attention[402,407]. There are various types of focal devices. In visual conc. the focus is an image, for example a flower, prayer or chanting, and in physical repetition, such activities as dancing, breathing or jogging are the focus of attention. The focal device allow the intuitive, non-ego centered mode of thought processing carried out in the right hemisphere to dominate consciousness in place of normally dominant analytic ego-centred mode of thought. The focal device engages the left hemisphere's neural circuit so that the right hemisphere become a dominant. During meditation, certain physiological changes have been consistently reported reduced heart rate, decreased oxygen consumption, decreased blood pressure, reduction in blood lactate, decreased respiration rate and so on.

However, one author argues that the physiological response pattern found in meditation is not unique to meditation per se, but common to any passive relaxation technique. This view has been supported by a number of studies that suggest few physiological differences between meditation and other self relation stategies, such as biofeedback and relaxation. Most studies have found that the constellation of changes is significantly different between meditation groups and control groups but not between meditation

and other self control treatments. Some researchers have argued though, that the kind of physiological measures being used in meditation research are not sufficiently sophisticated to define the unique aspects of mediation. They also suggests that meditation might be useful for certain types of anxiety. Well controlled research studies on the clinical effectiveness of meditation are few but they indicate a wide range of stress related disorders potentially responsive to meditation [398,402].

A number of research studies have investigated the use of meditation in reducing high blood pressure. These studies consistently show a reduction in blood pressure in the treatment group, a reduction in the use of antihypertensive drugs and a reduction in somatic symptoms[404,407,408]. These studies suggest that meditation may be a promising clinical intervention techniques for stress related disorders[409,411].

B. Effects on Regression towards mean on fall of Blood Pressure.

Treatments are often tried because a manifestation of disease is extreme or unusual for example, a particularly high blood pressure. In this situation, subsequent measurements are likely to show improvement for purely statistical reasons. Similarly when physicians encounter an unexpectedly abnormal test result,

they trend to repeat it. Often the second test result is closer to normal. Patients selected because they represent an extreme value in a distribution can be expected on the average, to have less extreme values on subsequent measurements. This can occur for purely statistical reasons (random variation) and is called regression to the mean in this study. It has been explained by considering the repeated measurement of blood pressure and the assessment of metabolic therapy in reducing blood pressure.

By correlation and regression analysis the over all results showed that the changes of blood pressure were due to the effect of metabolic therapy. Therefore, it eleminates the effect of statistical concept of regression to mean. In a trial on the effect of reducing multiple risk factors on the subsequent incidence of coronary heart disease, high risk patients were selected for study. Elevated blood pressure was one of the risk factors that caused people to be considered for the study. People were screened for inclusion in the study on three consecutive visits. Blood pressures at those visits, before any therapeutic intervention were undertaken. There was a substantial fall in mean blood pressure between the first and the third visits. The authors attributed this fall to regression to the mean, as well as to a tendency for patients to be more relaxed on later visits. There was also a fall in serum cholesterol, which presumably was not affected by moment to moment anxiety[412].

Regression to the mean arose in the following way. Subjects were first selected because their initial blood pressure fell above an arbitrarily selected cut off point in the tail of a distribution of blood pressure for all the patients examined. Some of those subjects would remain above the cut off point on subsequent measurements because their true blood pressures were ordinarily higher than average. But others who were found to have blood pressures above the cut off point during the initial screening usually had lower pressure readings. They were included only because they happened through random variations to have high blood pressures at that point in time. When their blood pressure were retaken they had lower values than in the first visit. This tended to drag downward the mean blood pressure of the sub-group originally found to have readings above the cut off point. Thus, patients who are singled out from others because they posses a laboratory test that is unusually high or low can be expected on the average, to be closer to the center of the distribution if the test is repeated. Moreover, subsequent values are likely to be more accurate estimates of the true value, which could be obtained if the measurement were repeated for a particular patient many times. So the time honored practice of repeating laboratory test that are found to be abnormal and calling the second one, which is often within normal limits, the correct one is not just wishful thinking. It has a sound theoretical basis. It also has an

empirical basis. For example, it has been shown that half of the serum T4 tests found to be outside normal limits on screening were within normal limits when repeated [413,414]. However, the more extreme the initial reading is the less likely it is to be normal if it is repeated. It should be stated that regression to the mean refers to a phenomenon first observed by galton when he noticed that the heights of sons tended to be closer to the over all mean than the heights of their fathers. Thus tall fathers tended to have sons shorter than themselves, while the opposite was true for short fathers[179]. Failure to appreciate this phenomenon can lead to misleading interpretating of findings in two circumstances. The first is if the difference between two repeated measurement is related to the first measurement. The second is, if only individuals in the extreme of a distribution are selected for treatment and follow up.

It may be further mentioned that part of the antihypertensive effect reported with these and other metabolic therapy might be responsible to the non-specific fall in blood pressure so often seen when repeated readings are taken. Such decrease may reflect a statistical regression toward the mean as already discussed above. It may be a placebo effect or a relief of anxiety and stress with time. The same phenomenon is probably also responsible for much of the initial response to drug therapy, so that success may be attributed to both drugs and non drugs when it

is deserved by neither. However, in this study it has been analysed that the fall of blood pressure was due to the effect of metabolic therapy but not due to the effect of statistical concept of regression to mean.

9. The potential of Metabolic and drug therapy

There is an increasing appreciation that the majority of the hypertensive patients i.e. those with mild hypertension (diastolic blood pressure 90-104 mmg Hg), have not been demonstrated to derive a cardiac benefit from the drug therapy[415,416]. Again for whom drug therapy has been ineffective in reducing overall mortality risk or preventing the development of clinical complications such as Ischemic heart disease. Those with diastolic blood pressure between 90-95 mmHg, who constitute 40% of the entire population have not been found to benefit from drug therapy in the controlled trials. Drug treatment though effective, is expensive, frequently accompanied by side effects and minimally cost effective for such patients. Metabolic therapy offers a potential approach and is free from side effects. Dietary and other metabolic therapy, therefore, being avidly sought with the hope of increased benefit for this large number of patients along with decreased risk and cost. Nonetheless, it is important to identify such patients so that metabolic therapies i.e. dietary sodium restriction, weight

control, perhaps exercise conditioning, might be recommended as efforts to reduce cardiovascular risk. However, not all of the trials used statistactory methods, and the more rigorous trials tended to show smaller effects. Nevertheless, dietary potassium remains one of most consistent correlates of blood pressure level in population studies [417]. Hypothesis proposed to account for this paradox include the possible importance of the anion i.e. chloride might counter some of the hypertensive effect and an interaction with the level of sodium in the diet.[418,419]. The magnitude of the blood pressure reductions with changes in body weight and sodium in take could potentially have a substantial benefit in reducing the incidence of hypertension, and on cardiovascular morbidity and mortality, especially if these effects are additive in combination. While a sizable proportion of intervention participants met study goals for reduction of over weight and excess salt. Based on the greater blood pressure response of those who made greater changes it is reasonable to assume that if more substantial improvements in life styles were made, the effect on blood pressure would be more sizable than that observed in the present study. An additional benefit observed within the intervention group was the effect of weight loss on levels of serum cholesterol, serum glucose and serum TG. Over all, study results indicate that a modest programme based on metabolic or nutritional steps to reduce over weight, high salt intake, accompanied by an

increase in moderate exercise can contribute to the control of hypertension.

Recent epidemiologic studies show that high levels of physical fitness are associated with lower blood pressure [420,421]. Considering that there is as yet no unanimity of opinion with respect of drug therapy at these blood pressure levels (90 to 104 mm/Hg), the use of exercise training as a component of metabolic approach is entirely appropriate. An intensification of efforts to further document the efficacy of metabolic therapies in the management of hypertension, including exercise, is timely considering a recent study that shows more than 25% of patients normotensive on medication remain so far at least a year discontinuance of drug therapy [422].

The greater awareness of the dangers of elevated blood pressure along with the availability of safer and more effective antihypertensive agents has led to a therapeutic explosion and at the same time hypertensive has become the most frequent reason for visits to physician as well as the leading indication for prescription drugs [423]. These statistics reflect the extension of treatment to more people with mild hypertension, who make up almost 80% of the hypertensive populations. In patients with mild hypertension the decision to treat is an important issue that has

again come to the fore with the availability of several new techniques for measuring blood pressure. The rise in blood pressure induced by the act of measurement itself, referred to as the "White coat reaction" may lead to a false diagnosis of hypertension [424,425]. This is one reason why 40% of the patients included in large scale hypertension trials subsequently become normotensive with placebo [426].

When all evidence concerning the efficacy and applicability of Metabolic therapies for the treatment of hypertension is considered, the more liberal and optimistic might say. "Everything works at least for a while and patients should be enthusiastically encouraged to use them before, and hopefully instead of, drug therapy." The more conservative and pessimistic might say "Nothing has been shown to work over the long-term and patients should be protected by antihypertensive drugs as soon as their disease is recognized."

A more balanced view is provided in the 1984 of the Joint National Committee, which recommends " Non pharmacologic approaches should be used both as definitive intervention and as an adjunct to drug therapy [427]".

As noted, increasingly long and strong evidence from controlled studies attests to the efficacy of multi faceted metabolic control

programmes to reduce the blood pressure. Whether such success can be achieved by individual practitioners is uncertain. However, because help is available, including various educational materials for patients, professional assistants such as dietitians and psychologists and groups organized for weight reduction exercise, and relaxation therapies, the effort seems both increasingly easy and likely to be successful in lowering blood pressure.

Desptie the uncertainty as to the ability of drug therapy as it has been provided to protect against coronary disease in those with mild hypertension, successful reduction of elevated blood pressure will protect against progression of risk factors[1]. The active therapy of patients with mild hypertension, defined as diastolic blood pressure between 90-104 mmHg has expanded markedly in the past few years as a result of the confluence of three events:

a. even minimally elevated pressures have been shown to increase the overall risk for premature cardiovascular disease;
b. trials have shown that progression of disease and other risk factors can be reduced by drug therapy;
c. medications that are easier to take have become available and intensively marketed.

Because of these factors and the inherent desire for patients and physicians to take direct action against perceived dangers,

millions of asymptomatic persons are now being treated with antihypertensive drugs. The number of people world wide now being continuously treated for hypertension represents the largest use of long term drug therapy. The use of such therapy are more "aggressive" in the United States than any where else. Such aggressive therapy has been vigorously defended and credited with at least some of the reduction in coronary and stroke mortality seen in the United States since 1968[428]. Others question the wisdom of such therapeutic activism. In the words of an English practitioner: "In the US, the threshold for diagnosis is the threshold for treatment. The question to treat or not to treat" need no longer be asked. A free-fire zone has been created above diastolic 90, in which we simply shoot everything that moves[429]. Even in the United States the aggressive treatment of hypertension is now being seriously questioned for numerous reasons including: the awareness that the risks of relatively mild hypertension, although apparent for the aggregation are not shared by all[430], the failure to show clear protection against coronary mortality by drug therapy in nine clinical trial, involving more than 41,000 patients[431-436]. As a result of this apparent inability to protect against coronary disease, concerns have arisen about the bio-chemical changes that may have, increased risk in various ways, such as by lowering potassium levels, raising lipid levels, or altering glucose tolerance, concerns about the adverse effects of

therapy, not upon such bio-chemical markers but also upon the quality of life and the potential for too much of a reduction in blood pressure to induce additional coronary ischemia and the recognition by cost-to-benefit analysis[137].

The sum of current evidence, then, supports the view that the risks of mild hypertension are not so great and the benefits of active therapy are not so large as to mandate that all mildly hypertensive persons be treated. A more aggressive course could be justified if there were no risks of problems associated with active therapy, but in all trials, 20% to 40% of patients given drug therapy experienced some adverse effects. Therefore, useless there is an obvious need for the more immediate institution of drug therapy, such as progressive target organ damage or blood pressures so high as to threaten immediate danger, all the patients should be given the opportunity to practice Metabolic therapeutic measures. As noted earlier, in the preceding chapter. Patient's monitoring logically can be done at home and for some with ambulatory 24-hour monitoring, which may provide, in a condensed manner, better prognestic evidence than multiple blood pressure measurement, taken in the office. While the blood pressure is being monitored, the use of appropriate Metabolic therapies may help lower the pressure even more, without risk and with relatively little inconvenience. Such Metabolic therapies may not only lower the blood pressure but also

reduce overall cardiovascular risk by amelioration of such conditions as hyperlipidemia, glucose intolerance, and alcohol abuse.

The elevations of systolic blood pressure above 170 mmHg at any age, deserve gradual reduction by appropriate Metabolic and drug therapies. In hopes of documenting further the benefits of antihypertensive therapy for the millions with mild to moderate hypertension, additional analysis of the subsequent mortality among MRFT participants and meta-analysis of trials involving more severe hypertensives have been performed. Unfortunately, they have shed little light on the issue of coronary protection for mild-to-moderate hypertension.

Therefore, there is legitimate cause for the disagreement as to the level at which to institute drug therapy, some believing that drug therapy should be given to all with diastolic blood pressure above 90 mmHg, others believing that it should be given only to those with diastolic blood pressure above 100 mmHg. The disagreement is not only of academic interest. As many as 40 million persons in the United States alone are in that 90 to 100 mmHg range. So obviously the issue has great clinical and economic relevance. On the basis of available data, the position adopted by a conference sponsored by the WHO and the International society of Hypertension seems to

be an appropriate compromise. In substance it states that after three to six months of observation, 95 mmHg diastolic blood pressure should be used as the level for institution of active drug therapy.

The benefits of continuous antihypertensive therapy have been extensively documented. However, lack of compliance[438] with the prescribed regimen, excessive cost and adverse effects of drugs led to the consideration of Metabolic therapies as an effective alternative to life long medication. The drug reduction and discontinuation trials have found the best results with mild hypertensives who had, low sodium intake, and achieved weight loss[438-444]. This approach appears to safe provided that blood pressure is monitored frequently and may improve compliance, save treatment costs and reduce adverse effects of certain drugs.

LIMITATION OF THE STUDY

1. A major impediment was investigator's feeling of loneliness, rising criticism and lack of confidence as per the expected outcome, that is overall management and consequences of this research project. This is because of lack of adequate knowledge and prevailing underuse of Metabolic therapies, due to excitement over newly available drugs and the massive advertising campaigns to promote antihypertensive drugs. It may be stated that many physicians or practitioners either donot use Metabolic therapy or use them in a casual or in a neglected manner.

2. The lack of confidence in the effectiveness of Metabolic therapies also represents a reaction by physicians against the overly enthusiastic advocacy of these maneuvers by "true believers" quacks and health gurus who promise major benefits with no scientific basis. Most physicians are suspicious of the claims of long term benefits from Metabolic control and are unwilling to spend the time and effort needed to implement such therapies without proof of their efficacy. As a result investigator had to prescribe 5 mg diazepam during trial on few patients but no antihypertensive drugs were used for hypertensive groups. This was one of the limitation where some patients were occasionally given 5 mg diazepam at bed time.

3. While repeated readings were taken for measuring blood pressure the antihypertensive effect of Non drug therapies might be attributed to the non specific fall in blood pressure. Such decreases may reflect a statistical regression toward the mean, a placebo effect or a relief of anxiety and stress with time. The same phenomena is probably also responsible for much of the initial response to drug therapy, so that success may be attributed to both drugs and Non drugs when it is deserved by neither.

4. A search of the literature shows paucity of data available for comprehensive analysis. Data from long term controlled studies of Non drug therapy document relatively small. Literatures mostly available on various Nondrug therapies in a piece meal way as well in isolated way in different studies. A combined therapies of comprehensive study were also could not be found out. It was another impediment to the overall quality and prospects of this doctoral thesis.

5. All clinical and pathological investigations were carried out in Armed Force institute of pathology, hence the validity and accuracy of the tests was not under the control of the investigator.

6. There was no standard objective measures for success of stress reduction techniques on blood pressure changes. Moveover, there was no laboratory facilities to estimate the urinary excretion of catecholamines and cortisol. The negative results for blood pressure management by relaxation therapies are in general accord with accumulating evidence from other clinical trials.

7. A true experimental study design and other operational problems for example without having any research grant or financial support, all impede good quality of data processing and analysis. However, efforts were taken to delimit all sorts of problems as far as practicable.

CHAPTER-V
SUMMARY

The main findings of the study are briefly tabulated as under:

Parameter		Subjects		P value
		Experimental (n=28) X(SD)	Control (n=28) X (SD)	
Age (yrs)		40.82 (± 8.66)	40.11 (± 14.45)	P>0.05
Height (m)		1.66 (± .05)	1.69 (± .06)	P<.001
Weight (kg)	Initial	64.95 (± 7.95)	69.39(± 7.76)	P>0.05
	Final	63.28(± 8.04)	68(± 7.55)	P>0.05
BMI	Initial	23.76(± 2.51)	24.44(± 2.41)	P>0.05
	Final	23.07(± 2.49)	23.85(± 2.36)	P>0.05
PBMI	Initial	107.17(± 11.31)	110.61(± 10.92)	P>0.05
	Final	104.39(± 11.31)	107.93(± 10.68)	P>0.05
Systolic BP (mmHg)	Initial	161.75(± 10.32)	137.61(± 4.73)	P>0.05
	Final	128.93(± 10.66)	134.07(± 5.02)	P>0.05
Diastolic BP (mmHg)	Initial	107.72(± 4.99)	89.04 (± 1.82)	P<.001
	Final	82.79(± 6.77)	83.64 (± 82.54)	P>0.05

Parameter		Subjects		P value
		Experimental (n=28) X (SD)	Control (n=28) X (SD)	
Serum Sodium (mmol/L)	Initial	140.03(±2.35)	139.68(±2.8)	P>0.05
	Final	138.62(±3.39)	138.86(± 2.85)	P>0.05
Serum potassium mmol/L	Initial	4.34 (± 0.41)	4.62 (± 0.58)	P<.001
	Final	4.79 (± 0.61)	4.89 (± 0.57)	P>0.05
Serum Chloride (Nacl) mmol/L	Initial	100.45 (± 2.24)	99.62 (± 2.51)	P>0.05
	Final	98.86 (± 3.25)	99 (± 2.83)	P>0.05
Serum cholesterol mg/dl	Initial	229.42 (± 27.64)	217.64 (± 23.23)	P>0.05
	Final	206.11 (±20.32)	201.71 (± 17.76)	P>0.05
HDL cholesterol mg/dl	Initial	56.69 (±4.23)	57.19 (± 3.97)	P>0.05
	Final	59.83 (± 2.29)	60.98 (± 2.21)	P>0.05
LDL cholesterol mg/dl	Initial	129.33 (± 25.66)	122.45 (± 30.05)	P>0.05
	Final	117.01 (± 19.64)	116.23 (± 18.78)	P>0.05
Serum TG mg/dl	Initial	170.64(±47.05)	165.49(±50.81)	P>0.05
	Final	138.11 (± 39.75)	147.46 (± 39.48)	P>0.05
Serum Olipoprotein mg/dl	Initial Final	473.60 (±57.12) 437.23(± 62.51)	448.63 (± 102.68) 815.69(± 51.11)	P>0.05 P>0.05
	Final	457.23 (± 62.51)	815.69 (± 51.11)	P>0.05
Serum total lipids mg/dl	Initial	884.87 (± 74.03)	830.4(± 158.44)	P>0.05
	Final	830.11 (± 62.51)	815.69(± 51.11)	P>0.05
Serum urea mg/dl	Initial	28 (± 7.72)	29.54 (± 6.09)	P>0.05
	Final	27.79 (± 8.02)	29.72 (5.92)	P>0.05
Serum creatinine mg/dl	Initial	1.13 (± 0.17)	1.15 (± 0.18)	P<0.05
	Final	1.09 (± 0.12)	1.08 (± 0.13)	P<0.05
Fasting plasma glucose mg/dl	Initial	95.55 (± 2.33)	96.77 (± 7.85)	P>0.05
	Final	88.85(± 8.24)	91.38 (± 6.7)	P>0.05

CONCLUSION

Hypertension is one of the most common cardiovascular diseases in the world including Bangladesh. Majority of the patients though are asymtomatic, the disease is readily detectable and easily treatable and often leads to complications, some of which are fatal if left untreated. The results of this controlled clinical trial of combined Metabolic therapies among men with uncomplicated primary essential hypertension provide a reasonable inference to lower high blood pressure.

The central finding of the present study was the significant reduction in both systolic and diastolic blood pressure, in the experimental group. There was small but also significant decreased of diastolic and systolic blood pressure of the control group. While a majority of the intervention participants met study goal for reduction of over weight, excess salt and less potassium intake, the overall relation with the changes of blood pressure was not uniform. This study showed that final mean serum cholesterol, LDL Cholesterol and serum urea level had increased. A successful changes in Biochemical variables were correlated with the changes of blood pressure. An additional benefit observed within the experimental and control group, was the effect of weight loss on level of serum sodium chloride, cholesterol, fasting plasma glucose

and serum TG. It had already been mentioned that this therapy had a favourable effect on the lipid profiles by decreasing the level of total and LDL-Cholesterol and increasing HDL cholesterol levels.

By statistical analysis, the overall results demonstrated that the fall of blood pressure was due to the effect of Metabolic therapy. Therefore, it eliminates the effect of statistical concept of regression towards the mean a placebo effect or a non specific fall of blood pressure or a relief of anxiety and stress with time. Moreover, after a period of six months follow up, experimental group had small increased on average diastolic blood pressure. In conclusion, overall study results indicate that a combined Metabolic therapy based on nutritional changes, walking on a treadmill set, progressive relaxation therapy, deep breathing exercise and meditation can contribute effectively in controlling hypertension.

Although there was no standard objective measures for success of blood pressure reduction by stress reduction techniques, however, required data on cardiovascular reactivity were collected and which will be reported elsewhere.

Regardless of one's attitude towards the use of antihypertensive drugs, there seems to be no argument that Metabolic therapy is

safer and associated with fewer side effects. Metabolic control even it does not reduce the blood pressure more efficiently, this programme should do no harm and may provide several other benefits including a reduction in most other risk factors for premature cardiovascular disease. The benefits of continuous antihypertensive therapy have been extensively documented. However, lack of compliance with prescribed regimen, excessive cost and troublesome adverse effects of some antihypertensive agents led to the consideration that Metabolic therapy as an effective alternative to lifelong medication. For most patients, the follow up visits mainly for maintaining motivation can be handled by non-physicians. Overall, implementation may be difficult, but the potential for improvement in morbidity status makes the difficulty seem trivial. Moreover, the use of drugs to lower blood pressure does not relieve the hypertensive patient of the need to lose weight, exercise regularly, eat a prudent diet and learn to relax. Therefore, the above combined Metabolic approach appears to be safe, provided that blood pressure is monitored frequently, and may improve compliance, save treatment costs and reduce adverse effects of certain drugs, although its long-term consequences for morbidity and mortality remain to be determined.

Treatment of hypertension is a part of prevention and like preventive strategies its progress on primary prevention should be

regularly reviewed. It may be finally concluded that no desirable advance in primary health care would improve the quality of life than the Metabolic control of hypertension.

RECOMMENDATIONS

Keeping in view, the above findings and other currently available evidences, a practical Metabolic therapy or Metabolic prescription might have the following recommendations:

1. **Weight reduction:** For the over weight, weight reduction should be the primary goal to keep away from chronic disease like coronary heart disease.
2. **Dietary changes:** Since in every case of obesity there is always an imbalance between food intake and energy expenditure, dietary calorie restriction is a pivotal measure in achieving successful weight reduction. The extent of calorie restriction has to be individually determined and should be based on age, body size and physical activity. It is essential that the diet should be balanced.
3. **Salt restriction:** For all hypertensive, dietary sodium intake should be restricted to 2g/d (88 mmol/d) with caution not to reduce the consumption of low fat, low sodium milk and cheese products so as to maintain calcium intake. Additional guidelines include the following:

    a. Encouraged not to add excess salt at table and eat foods with a high sodium content.

b. If a salty taste is desired, use a half sodium and half potassium chloride preparation or a pure potassium chlorides substitute.

c. Avoid or minimize the use of "fast" foods, many of which have high sodium content.

d. Recognize the sodium content of some antacids and proprietary medications.

4. **Fat intake:** The recommended dietary goal is that saturated fatty acids should contribute no more than 10% of the total energy intake, implying that only 20-30% of the total diet should be provided by fats. In this trial the subjects were given a total of 30% fats out of total 2000 kcal intake per day. Of the total, energy intake there were 24% unsaturated fats and 6% saturated fats. Based on total fats intake, there were 80% unsaturated fats and 20% saturated fats.

In practical terms the above recommendation lead to an emphasis on the consumption of foods of plant origin e.g cereals, fruits and vegetables. The increased consumption of fish, poultry and lean meats, non fat and low fat dietary products and the limited fats in food preparation all would help to reduce both the total amount of fat and saturated fats in the diet. The avoidance of a high intake of whole eggs would be a step towards limiting the intake of dietary cholesterol.

5. **Physical exercise:** Regular isotonic exercise is encouraged. Exercise leads to improvements in cardiac functions and glucose metabolism and to psychological benefits such as improved self esteem. A brisk and sustained walking to be carried out (6d/week). Initially it will cover 1.5 to 2.5 miles by 24 to 32 minutes at first and second week and then 3 miles at 45 minutes to be maintained.

6. **Potassium** intake need not be specifically increased, because it will rise with a lowered sodium intake. Supplemental magnesium and calcium should only be given to those who are deficient until additional evidence of their efficacy is available.

7. **Relaxation therapy:** For the control of stress and strain in life, hypertensive patients are advised to perform the following relaxation therapies:

   a. Progressive relaxation : Duration: 20-30 minutes, once daily, 6 d/week.
   b. Deep neuromuscular breathing -do-
   c. Meditation at least for one hour daily.

8. **Primary prevention** : For the control of hypertension, the ultimate goal in general is primary prevention. On both pathological and behavioural grounds, prevention should start in childhood, because this is the time when the atherosclerotic and hypertensive processes start and it is also when life style, habits like smoking, eating physical exercise etc. sets in. Primary prevention, strategically falls into two groups:

   a. **High-risk strategy:** Identification of high risk groups by routine examination of family history, body weight, plasma lipids level, level of blood sugar, history of cigarette smoking, oral contraceptive use, habitual physical activity, ECG abnormalities for timely intervention in the high risk groups. Detection of high-risk subjects should be encouraged by the optimum use of clinical methods. Since hypertension tends to cluster in families, the family history of hypertension and "tracking" of blood pressure from childhood may be used to identify individual at risk.

   b. **Population strategy:** The population strategy is directed at the whole community irrespective of individual risk levels. The goal of population approach is to shift the community distribution of blood pressure towards lower

levels of biological normality. This involves a multifactorial approach based on the following Metabolic interventions.

1. **Nutrition:** (I) Lowering of LDL-cholesterol or total cholesterol: HDL-cholesterol ratio by diet, exercise of both (II) To keep average cholesterol level of the population within normal limit and to prevent further rise (III) Moderate fat intake and avoidance of a high alcohol intake (IV) Restriction of salt intake to an average of not more than 5 g per day. (V) To regulate mandatory levelling of the sodium content of preserved food and commercial arrangements, such as providing the market with low-sodium food articles (VI) restriction of energy intake appropriate to body needs.

2. **Weight reduction :** The prevention and correction of obesity (BMI>25) is a prudent way of reducing the risk of hypertension and indirectly coronary heart disease, it goes with dietary changes.

3. **Exercise promotion:** The evidence that regular physical activity leads to a fall in body weight, blood lipids and blood pressure, goes to suggest that regular physical activity should be encouraged as part of the strategy for risk factor control.

4. **Behavioural changes:** Reduction of stress and smoking, modification of personal life style, food habits, physical exercise, relaxation and meditation could be profitable.

5. **Health education:** The general public require preventive advice on all risk factors and related health behaviour. The whole community must be mobilized and made aware of the possibility of primary care. However, in population with high average salt intake, educational measures should be undertaken immediately to explain the potential importance of reducing excessive salt intake. Similarly in population in which over weight is prevalent, information on the links between overweight and blood pressure elevation should be addressed to general public. Individuals who are obese or accustomed to an excessively high salt intake should be adequately counselled.

6. **Self care :** The patient is taught self care i.e to take his own blood pressure and keep a log book of his readings.

9. **Enhancing compliance:** Subjects found to have hypertension should be followed up and treated by Metabolic therapies and if necessary by prescribed regiments (his compliance) is often deficient, as shown by many studies. It can be improved by patient - education programmes and by improved information and motivation of the practising physician.

10. **Active follow up**: Whether treated by Metabolic measures or drug or if merely on observation, hypertensive patients should be followed up at regular intervals. If a patient misses an appointment a reminder should be sent inviting him to come at another date. In case of further non communication a health visitor should be sent to patients to re-establish the contact.

11. **Metabolic** measures should be introduced gradually and gently. Too many and too drastic changes in life style may discourage patients from accepting needed care. However, Metabolic control of hypertension should be used by all patients either alone or as a combination to drug therapy.

LESSONS LEARNT

1. Metabolic therapy can be practised by the patient himself whereas drug treatment always need constant supervision.

2. Lack of compliance with the prescribed drug, excessive cost and overtly bothersome side effects led to the consideration of Metabolic therapy as an effective alternative.

3. Metabolic approach appears to be safe, may improve compliance, save treatment costs and reduce metabolic disorders of certain drugs.

4. Anti-hypertensive drugs are not to be stopped if the blood pressure is found to come back to normal. Stoppage of the drugs will cause the pressure to shoot up again, most probably above the previous level, whereas sudden stoppage of Metabolic therapy would have no such consequences.

5. Under control situation based on hospital conditions the uncomplicated primary essential hypertension could be controlled without drugs.

6. This study will increase the confidence of the physicians as per the effectiveness of Metabolic therapy and thereby reduce its barrier in their prevailing practising.

7. It will act as a scientific advocacy for other health professionals who promise a great benefit out of Metabolic therapy with no scientific basis.

REFERENCES

1. Puska P, Nissinen A, Vartiainen E, et al. Controlled randomised trial of the effect of dietary fat on blood pressure. The Lancet 1983, 1(8):1-9.
2. Gross F, Pisa Z, Strasser T, et al. Management of Arterial Hypertension - A practical guide for the physician and allied health workers. WHO, Geneva: 1984.
3. The pooling project research group. Relationship of blood pressure serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events. Final report of the pooling project. American Heart association Monograph, no. 60. Dallas. American Heart Association, 1978.
4. Kaplan N M. Systemic hypertension: Mechanism, and diagnosis. Braunwarld Heart disease: A text Book of Cardiovascular medicine, 4th ed; WB Saunders Company; London: 1992: 817- 873.
5. Stamler R, Stamler J, Grimm R, et.al. Nutritional therapy for high blood pressure. Final report of four-year randomized controlled trial- The hypertension control programme. JAMA 1987; 257: 1484.
6. Oberman A, Wassertheil- Smoller, Longford H.G, et al. Pharmacologic and nutritional treatment of mild hypertension in patients on antihypertensive drugs. Results of previous medication, laboratory tests and life quality. J. Intern. Med 1989; 226:39.
7. Aberg H, and Tibblin G. Addition of non-pharmacological methods of treatment in patients on anti hypertensive drugs: Results of previous medication, laboratory tests and life quality J. Intern. Med 1989, 226:39.

8. Stamler R, Stamler J, Gosch F.C, et al. Primary prevention of hypertension by nutritional hygienic means. Final report of a randomized controlled trial. JAMA 1989; 262: 1801-1989.

9. Hypertension prevention trial Research group. The hypertension prevention trial: three-year effects of dietary changes on blood pressure. Arch. Intern. Med. 1990;150:153.

10. Burglund A, Andersson, O.K, Berglund G, et al. Anti- hypertensive effect of diet compared with drug treatment in obese men with mild hypertension. Br. Med. J 1989; 299:480.

11. Haskell WL; Exercise and health, Cecil Text book of medicine, 19th ed, WB Saunders Company, London: 1992;42.

12. Beard TC, Gray WR, Cooke H M, et al. Randomised controlled trial of a no-added-sodium diet for mild hypertension. The Lancet 1982; 28: 455-458.

13. Ahmed Q & Chowdhury SGM. Hypertension, Management, Mismanagement and consequences. Convention, Bangladesh Society of hypertension 1991.

14. Burton R. Withdrawing antihypertensive treatment (editorial) Br. Med J 1991; 6798 (303): 324-325.

15. Aylett M & Ketchin S. Stopping treatment in patients with hypertension. Br Med J 1991;6798 (303): 345-346.

16. Kaplan NM: Systemic hypertension: Therapy. Braunwarld Heart disease: A Text book of cardiovascular Medicine, 4th ed, WB Saunders Company; London; 1992: 852-866.

17. Cecil Text Book of medicine, 19th ed, WB Saunders Company, London: 1992: 253-265.

18. Park's Text Book of preventive and social medicine, 13th ed. M/s. Banarsidas Bhanot Jabalpur India: 1991:242-246.

19. WHO. Techn Rep Ser, No. 686, 1983.

20. WHO. WHO Chronicle, 1974; 28(3):116

21. Marmot M.G. Brit. Med. Bulletin 1984; 40(4): 380.

22. Pickering G. Am. J. Med. 1972; 52: 570

23. WHO. Tech Rep Ser, No. 628, 1978

24. WHO. Bull WHO 1963; 61 (1) 53

25. Pickering T.G., James G.D. Boddice, et al: How common is white coat hypertension? JAMA 1988; 259:225.J

26. Evans CE, Haynes RB, Goldsmith C.H, et al. Home blood pressure measuring devices: A comparative study of accuracy J. Hypertens 1989; 7:133.

27. Prisant L.M, and Carr A.A. Ambulatory blood pressure monitoring and eco cardiographic left ventricular wall thickness and mass. Am J. Hypertens 1990; 3:81.

28. Mejia A.D, Egan B.M, Schor K N. J, et al. Artefacts in measurement of blood pressure and lack of target organ involvement in the assessment of patients with treatment resistant hypertension. Ann. Intern. Med. 1990; 11:270.

29. Saito I, Takeshita E, Hyastri S, et al. Comparison of clinic and home blood pressure levels and role of the sympathetic nervous system in clinic-home differences. Am J. Hypertens 1990;3:214

30. Levey D, Wilson PWF, Anderson K.M, et al. Stratifying the patient at risk from Coronoary disease. New insignts from the Framingham Heart Study. Am Heart J 1990; 119:712.

31. Harrison's principles of Internal Medicine, 12th ed. MCG raw-hill, Inc. New York: 1991: 1002.

32. The 1988 report of the joint National Committee on Detection, evaluation and treatment of high blood pressure. Arch. Intern. Med. 1989; 148:1023. Copyright 1989. American Medical Association.

33. Sarker M.H, Mohibulla M, Sultana, et al. Effects of age, Sex, and body weight on blood pressure in the urban population of Dhaka. J of IPGMR, 1989;4(2);48-52

34. Hypertension Detection and follow up program Co-operative group. Blood pressure studies in fourteen committees. A two stage screen for hypertension. JAMA 1977;237:2385.

35. The Heart-Arteries and veins, 7th ed.(I&H) McGraw-Hill Company, New York: 1990:633.

36. Subcommittee on definition and prevalence of the 1984 Joint National Committee: Hypertension prevalence and status of awareness, treatment and control in the United States. Hypertension 1985;7:457.

37. Waliullah. Hypertension in a mixed community. BMRC Bulletin 1976; 2(2): 95-99.

38. Islam N, Khan M, Latif ZA: Hypertension in the rural population of Bangladesh - A preliminary survey BMRC. Bulletin 1983; 9(1): 11-14.

39. Hypertension detection and follow up programme cooperative group: The effect of treatment in mortality in mild hypertension. N. Egn. J Med 1983;307:976.

40. Islam N, Janan FAj, Chowdhury NA, et al. Hypertension in secretariate population of Bangladesh. BMRC Bulletin 1979; 5(1): 19-24.

41. Hart JT. Hypertension, Library of general practisioner series, Churchill Livingstone;1980.

42. Linfors EW, Feussner JR, Blessing C.L et.al. Spurious hypertension in the obese patient. Effect of sphygmo manometer cuff size on prevalence of hypertension. Arch. Intern. Med 1984; 144: 1482.

43. Manning D M, Kuchirka C, and Kaminski J. Miscuffing. In appropriate blood pressure cuff application circulation 1983; 68:763.

44. Nelson WP and Egbert AM. How to measure blood pressure accurately. Prim. Cardiol 1984; 10:14.

45. Kirkendall W.M, Burton AC, Epstein FH, et al. Recommendation for human blood pressure determination by sphygmo manometers. circulation 1967;36:980.

46. Lendes and Klitzner TS. Ausculatory blood pressure measurement- Effect of pressure on the head of the sthethescope. West J Med 1984; 141:193.

47. Mancia G, Grassi G, Pomidossi G, et al. Effects of blood pressure measurement by the doctor on patient's blood pressure and heart rate. Lancet 1983;2:095.

48. Finnie, K.J.C, Watts, DG and Armstrong P.W. Biases in the measurement of Arterial pressure. Crit Care Med 1984; 12: 965.

49. Editorial catecholamines in essential hypertension. Lancet 1977,1: 1088.

50. Pickering G. Hypertension: Definition, natural histories and consequences. Am J. Med 1972; 52: 570.

51. Lund-Johansen P. Central haemodynamics in essential hypertension at rest and during exercise: A 20 year follow up study. J. Hypertens 1972 7 (Suppl 6) : S 52.

52. Julius S, Mejia A.D, Schork N J, et al. Neurogenic hyperkinetic borderline hypertension (BHT) in Techmsch, Michigan, Circulation 1990; 81:16.

53. Williams R.R. Will gene Markers predict hypertenion: Hypertension 1989, 14:610.

54. Aalkjaer C, Heagerty AM, Bailey I, et al. Studies of isolated resistence vessels from offspring of essential hypertensive patients. Hypertension 1987;9 (Suppl III): III-155.

55. Ferrannini, E. Buzzigoli G, Bonadonna, et al. Insulin resistence in essential hypertension. N. Engl J Med 1987; 317:350.

56. Swislock, ALM, Hoffman BB and Reaven GM. Insulin resistence, glucose intolerance and hyperinsulinemia in patients with hypertension. Am J Hypertens 1989;2: 419.

57. Pedersen KE, Nielsen JR, Klitgaard NA,et al. Sodium content and sodium efflux of mononuclear leucocytes from young subjects at increased risk of developing essential hypertension. Am J. Hypertens 1988; 3: 182.

58. Tang S-S, Stevenson L, and Ozau YJ. Endothelial renin- angiotensin pathway. Adrenergic regulation of angiotensin secretion. Circ. Res 1990;66:103.

59. Ferrario, CM, Barnes KI, Block CH, et al. Pathways of angiotensin formation and function in the brain Hypertension 1990; 15 (Suppl I):1

60. Horiba N, Nomura K and Shizume K. Exogenous and locally synthesized angiotensin II and glomerulosa cell functions. Hypertension 1990;15:90.

61. Hare GMT, and Osmond DH. New renin-angiotensin from plasma protein ? Am J Hyperlens 1990;3: 196.

62. American Heart Association Circulation 1981; 64: 510A

63. WHO. Techn Rep Ser No. 732, 1986

64. Gupta SP,et al. Ind. Heart J 1978; 30: 315

65. Hypertension detection and follow up programme co-operative group. N Eng J Med 1982; 307:976.

66. Stamler, R et.al. JAMA 1978; 240: 1607

67. Beard T.C, et al. Lancet 1982;2:455

68. Oliver MF. Br. Med. Bull 1981; 37(1):49.

69. Kaplan NM. Systemic Hypertension: Mechanisms and Diagnosis. Braunwald Heart Disease. A text Book of cardiovascular medicine, 4th ed. WB Saunders London: 1992;1:824.

70. Chase H.P., Garg S.K, Harris, S et al. High normal blood pressure and early diabetic nephropathy. Arch. Intern Med. 1990;150:639.

71. Smith W.M. Treatment of mild hypertension. Results of a ten- year intervention. Circ. Res. Amer Heart Asso 1977; 25 (Suppl 1): 98.

72. 1989 Guidelines for the Management of mild hypertension: Memorandum from a WHO/ISH meeting. Bull of WHO 1989; 67(5): 493-98.

73. Kaplan N M. Clinical Hypertension, 5th ed. Baltimore. William & Wilkins 1990: 252.

74. Cloher T.P. and Whelton PK. Physician approach to the recognition and initial management of hypertension. Arch. Intern. Med 1986;146:529.

75. McClellan, W., Neel J., and Owen S. Correlates of drug therapy of diastolic blood pressure between 80-89 mmHg by physicians in the community. Am. J. Hypertension 1989;2: 869.

76. Report of the British Hypertension Society Working Party: Treating mild hypertension Agreement from the large from the large trials. Br. Med J. 1969; 298: 694.

77. Bonita R and Beaglehole R. Increased treatment of hypertension does not explain the decline in stroke mortality in the United States, 1970-1980. Hypertension 1989; 13 (Suppl I):1-69.

78. Hart J.T. The practitioner's view. In Gross F., and Strasser, T (eds): Mild Hypertension: Recent Advances. New York, Raven Press, 198;3: 365.

79. Berglund G: Goals of antihypertensive therapy. Is there a point beyond which pressure reduction is dangerous? Am. J. Hypertens 1989;2: 586.

80. Cutler J.A. Mac Mahon S.W and Furbeng Co: Controlled clinical trials of drug treatment for hypertension. A review. Hypertension 1989; 13 (Suppl I) 213: 1143.

81. Veterans Administration Co-operative study group on Antihypertensive Agents: Effects on treatment on morbidity in hypertension II. Results in patients with diastolic blood pressure averaging 90 through 114 mmHg JAMA 1970; 213: 1143.

82. US Public Health Service Hospitals Cooperative study group, Smith, WM. Treatment of Mild hypertension. Results of a ten- year intervention trial. Circ Res 1977; 40 (Suppl-I): 1-98.

83. Helgeland A. Treatment of mild hypertension. A five year controlled drug trial. The oslo study. Am. J. Med. 1980; 69:725.

84. Management Committee. The australian therapeutic trial in mild hypertension. Lancet 1980; 1:1261.

85. Medical Research Council working party: MRC trial of treatment of mild hypertension: principal results. Br. Med. J. 1985; 291:97.

86. Collins, R., Peto, R, MacMahon S, et al. Blood pressore stroke and coronary heart disease. Part II. Short term reductions in blood pressure. Overview of randomized drug trials in their epidemiological context. Lancet 1990; 335:827.

87. Curls J.D. Schneider K, Taylor J.O, et al. Antihypertensive drug side effects in the hypertension detection and follow up program. Hypertension 1988; ii (Suppl-II): 11-51.

88. Cruickshank, JM. Jhorp, J.M, and Zacharias, F.J. Benefits and potential harm of lowering high blood pressure. Lancet 1987,1: 581.

89. Samuelsson, O, Willhemisen L, Anderson O.K, et al. Cardiovascular morbidity in relation to change in blood pressure and serum cholesterol levels in treated hypertension: Results from the primary prevention Trial in Goteberg, Sweden. JAMA 1987; 258: 1768.

90. Cooper S.P, Hardy R.J., Labarthe, D.R, et al. The relation between degree of blood pressure reducation and mortality among hypertensives in the hypertension detection and follow-up program. Am. J. Epidemiol. 1968; 127:387.

91. Berglund G. Goals of antihypertensive therapy. Is there a point beyond which pressure reduction is dangerous? Am. J. Hypertens 1989;2:586.

92. Alderman MH, and Lamport B. Withdrawal of drug therapy in the treatment of hypertension. In Kaplan, NM, Brener, B.M and Largh J.H (eds) New therapeutic Strategies in Hypertension. New York, Raven Press, 1989; 171.

93. V. Seth, and J. Chug. Diet counselling- A integral component of hypertension management. The Ind. J. Nutr. Diet 1990; 27:77-81.

94. Kaplan N.M. Non-drug treatment of hypertension. Ann. Int. Med 1985; 102:359-373.

95. Joint National Committee on Detection, Evaluation and Treatment of High blood pressure. Arch. Int. Med. 1984; 144: 1045.

96. Silman A.J, Locke, C., Mitchelt, P and Hunpherson P. Evaluation of the effectiveness of a low sodium diet in the treatment of mild to moderate hypertension, Lancet 1983;1:1179.

97. Nugret C.A, Cannathan, JE, Sheehan E, and Hyers, C. Salt restriction in hypertension patients. Comparison of advice reduction, and group management. Arch. Int. Med. 1984; 144:1415.

98. Gopalan C, Rama Sastri, B.V, and Balasubramanian, S.C. Nutritive value of Indian Foods, ICMR, Hyderabad, India 1982.

99. Luseher T.F, and Vetter, W. Ahherence to medication. J. Hum. Hypertens 1990; 4 (Suppl 1):43.

100. 1988 Joint National Committee. The 1988 report of the joint National Committee on detection, evaluation, and treatment of high blood pressure. Arch. Inter. Med. 1988; 148:1023.

101. The Australian therapeutic trial in Mild hypertension report by the management committee. Lancet. 1980; 1:1261-7.

102. Ramsay L.E., Ramsay, M.H, HettiaRachchi J, et al. Br. Med J 1978; 244-245.

103. The 1984 Report of the Joint National Committee on detection, evaluation and treatment of high blood pressure. Arch. Int. Med 1984; 144: 1045-57.

104. World Hypertension League. Self-management of blood pressure. Bull of the WHO 66: 1988; 155-159 and J of Hypertension 1988;6: 257-267.

105. Tuck M.L. et al. The effect of weight reduction on blood pressure, plasma renin activity and plasma aldosterone levels in obese patients. New Eng J of Med 1981; 304:930- 933.

106. Maxwell M.H., et.al. Blood pressure changes in obese hypertensive subjects during rapid weight loss comparison of restricted V, unchanged salt intake. Arch. Int. Med 1984; 144:1581-1584.

107. Gillum R.F., et al. Nonpharmacologic therapy of hypertension; the independent effects of weight reduction and sodium restriction in overweight borderline hypertensive patients. Am. H.J. 1983; 105:128-133.

108. Kannel, W.B, et al. Cholesterol in the prediction of atherosclerotic disease. New perspectives based on the Framingham study. Annals of internal medicine 1979, 90:85.

109. Kannel, W.B & Gardon, T. ed. Section 26. Some characteristics related to the incidence of cardiovascular disease and death: Framingham study, 16-year follow-up. Washington, D.C. US Govt. Printing Office, 1970.

110. Haynes R.B. et al. Failure of weight reduction to reduce mildly elevated blood pressure : a randomized trial J of Hypertens 1984; 2: 535-539.

111. Fagerberg B, et al. Blood pressure control during weight reduction in obese hypertensive men: Separate effects of sodium and energy restriction. Br. Med J 1984; 288:11-14

112. Zidek W, et al. Weight reduction and salt restriction in hypertension: effects on blood pressure and intracellular electrolytes. Klinische Wochenschrift 1986; 64:1183-1185.

113. Stamler R, et al. Trial on control of hypertension by nutritional means: three-year results. J. of hypertens 1984; 2 (Suppl 3); 167-170.

114. Imai Y, et al. Effects of weight loss on blood pressure and drug consumption in normal weight patients. Hypertension, 1986;8:223-228.

115. Cohen N & Flamanbaum W. Obesity and hypertension demonstration of a "floor effect". Am. J. Med. 1986; 80:177- 181.

116. MacMahon S.W, et. al. The effect of weight reduction on left ventricularmass - A rendomized controlled trial in young, overweight hypertensive patients. N Eng J Med. 1986; 314: 334-339.

117. Bread T.C, Gray W.R, Cooke H M, et al. Randomised controlled trial of a No-added-sodium diet for mild hypertension. The Lancet 1982; 8296: 455-458.

118. Richards A.M, Nicholls MG, Espiner EA, et al. Blood pressure response to moderate sodium restriction and to potassium supplementation in mild essential hypertension. The Lancet 1984; 757-761.

119. Silman A.J, Mitchel, P, Loeke, et al. Evaluation of the effectiveness of a low sodium diet in the treatment of mild to moderate hypertension. The Lancet 1983; 1179-1182.

120. Watt G.C.M, Edwards C, Hart JJ, et.al. Dietary sodium restriction for mild hypertension in general practice. Br. Med. J. 1983; 432-435.

121. Rouse, I.L, Beilin, L.J, Armstrong BK, et al. Blood pressure lowering effect of a vegetarian diet controlled trial in - Normotensive subjects. The Lancet 1983: 5-9.

122. Williams P.T, Krauss R.M, Vranizian, KM et al. Changes in Lipoprotein subfractions during diet-induced and exercise- induced weight loss in moderately overweight Men. Circulation 1990; 81(4); 1293-1303.

123. Paffernbarger RS, Wing AL, Hyde R.T, et al. Physical exercise and incidence of hypertension in college alumni. Am J epidemiol 1983; 117:245-57.

124. Blair SN, Good year NN, Gibbons LW, et al. Physical fitness and incidence of hypertension in healthy normotensive men and women. JAMA 1984; 252:487-90.

125. Franklin B, Buskirk E, Hodgson J, et al. Effects of physical conditioning on cardio respiratory function, body composition and serum lipids in relatively normal weight and obese middle-aged women. Int J Obes 1979;3:97-109.

126. Johnson WP, Grover JA. Hemodynamic and metabolic effects of physical training in four patients with essential hypertension. Can Med Assoc J; 1967;96:842-7.

127. Boyer JL, Kasch FW. Exercise therapy in hypertensive men. JAMA 1970;211:1668-71.

128. Roman O, Camuzzi AL, Villalon E, et al. Physical training programme in arterial hypertension: a long term prospective follow up cardiology. 1981; 67:230-43.

129. Hagberg JM, Goldring D, EHsani AA, et al. Effect of exercise training on the blood pressure and haemodynamic features of hypertensive adolescents. Am J Cardiol 1983; 52:763-8.

130. Wilcox RG, Bennet J, Brown AM, et al. Is exercise good for high blood pressure. Br Med J (Clin Res) 1982; 285: 767-9.

131. Kannel WB. Cardio vascular consequences of physical inactivity. Primary Cardiol 1984; 10:74-90.

132. Rauramma R, Salonen JT, Kukkonen-Harjula K, et al. Effects of mild physical exercise on serum lipoproteins and metabolites of arachidonic acid: a controlled randomized trial in middle aged men. Br Med J (Clin Res) 1984; 288:603-6.

133. Bogardus C, Ravussin E, Robbins DC, et al. Effects of physical training and diet therapy on carbohydrate metabolism in patients with glucose intolerance and non- insulin dependent diabets mellitus. Diabetes 1984; 33:311-8.

134. Duncan JJ, Hagan RD, Upton J, et al. The effects of an aerobic exercise programme on sympathetic neural activity and blood pressure in mild hypertensive patients (Abstracts) circulation 1983 (4 pt 2): 111-285.

135. Kramseh DM, Aspen AJ, Abramority BM, et al. Reduction of coronary atheroselerosis by moderate conditioning exercise in monkeys on an atherogenic diet. N Engl J Med. 1981, 305: 1483-9.

136. Bjorntor PP. Hypertension and exercise. Hypertension. 1982; 4(5Pt2):111 56-9.

137. Kaplan NM. Joggers may live longer (Editorial). JAMA, 1984; 252:528.

138. Hidenon U, Yoichi T, Akira K, et al (Fukuoka Univ. Japan) Hypertension 1987; 9: 245-252.

139. Kelemen M.H, Effron, M.B, A stephen, et al. Exercise Training combined with antihypertensive drug therapy: Effects on lipids, blood pressure and left ventricular Mass. JAMA 1990; 263: 2766-2771.

140. Smith-Barbano PA, Pucak GT. Dietary fat and blood pressure. Ann Intern Med. 1983; 98(5 pt 2): 828-31.

141. Codde JP, Beilin LJ, Croft KD. Dissociation of effects of dietary fatty acids on blood pressure and prostanoid metabolism in Goldblatt hypertensive rats. J Hypertension. 1984; 2: 65-71.

142. Rouse I L, Beilin LJ, Armotrong BK, et al. Blood pressure lowering effect of a vegetarian diet: Controlled trial in normotensive subjects. Lancet 1983; 1:5-10.

143. Rouse IL, Beilin LJ. Vegetarian diet and blood pressure (Editorial). J Hypertension 1984; 2: 231-40.

144. Hodges RE, Rebello, T. Carbohydrates and blood pressure. Ann inter Med. 1983; 98 (5 pt 2): 838-41.

145. Havlik RJ, Hubert HB, Fabitz RR, et al. Weight and hypertension. Ann Intern Med 1983; 98:855-859.

146. Kurtz TW, Morris RC JR. Dietary chloride as a determinant of "Sodium-dependent" hypertension. Science 1983, 222: 1139-41.

147. Morgan TO. The effect of potassium and bicarbonate ions on the rise in blood pressure caused by sodium chloride. Clin Sci (Suppl) 1982; 63: 4075-95.

148. Hofman A, Hazebrock A, Valkenburg HA. A randomized trial of sodium intake and blood pressure in newborn infants. JAMA 1983; 250:370-3.

149. Ophir O, Peer G, Gilad J, et al. Low blood pressure in vegetarians: The possible role of potassium. Am J Clin Nutr 1983; 37:755-62.

150. MacGregor GA. Smith SJ, Markandu, et al. Moderate potassium supplementation in essential hypertension. Lancet 1982, 2:567-70.

151. Smith S.J, Markanda ND, Sagnella G.A, et al. Does potassium lower blood pressure by increasing sodium excretion ? A metabolic study in patients with mild to moderate essential hypertension. J Hypertension 1983; 1 (Suppl 2): 27-30.

152. Overlack A, Muller HM, Kolloch R, et al. Long term antihypertensive effect of oral potassium in essential hypertension. J Hypertension 1983; 1 (Suppl 2): 165-7.

153. Treasure J, Ploth, D. Role of dietary potassium in the treatment of hypertension. Hypertension 1983, 5: 864-72.

154. Fujita T, Noda H, Ando K. Sodium susceptibility and potassium effects in young patients with borderline hypertension. Circulation 1984; 69, 468-76.

155. Blaustein MP, Hamlyn JM. Role of a natriuretic factor in essential hypertension. An hypothesis. Ann Intern Med 1983; 95 (5 pt 2): 785-92.

156. Erue P, Bolli P, Burgisser E, et al. Correlation of platelet calcium with blood pressure: effect of antihypertensive therapy. N Engl J Med 1984; 310:1084-8.

157. McCarron DA, Morris CD, Henry HJ, et al. Blood pressure and nutrient intake in the United States. Science 1984, 224: 1392-8.

158. Belizan JM, Villar J, Pineda et al. Reduction of blood pressure with calcium supplementation in young adults. JAMA 1983; 249: 1161-5.

159. Macarron DH, Morris CD, Cole C. Dietary calcium in human hypertension. Science 1982; 217: 267-9.

160. Resnick L, Nicholson JP, Laragh, JH. Outpatient therapy of essential hypertension with dietary calcium supplementation (Abstract) J Am Coll Cardiol 1984; 3:616.

161. McCarron DA, Morris C. Oral Ca^{2+} in mild to moderate hypertension: a randomized placebo-controlled trial (Abstract) Clin Res 1984;32:335A.

162. Hurwitz L, McGuffe LJ, Smith PM et al. Specific inhibition of calcium channels by calcium ions in smooth muscle. J Pharmacol exp ther. 1962; 220:382-8.

163. Anderson DE. Interactions of stress, salt and blood pressure. Annu Rev Physiol 1984; 46: 145-53.

164. Patel C, Marmot MG, Terry DJ. Controlled trial of bio feed back aided behavioural methods in reducing mild hypertension. Br. Med. J (Clin Res) 1981; 282: 2005-8.

165. GlassGow MS, Gaarder KR, Engle BT. Behavioural treatment of high blood pressure: 11. Acute and sustained effects of relaxation and systolic blood pressure bio-feedback. Psychosom Med 1982; 44: 155-70.

166. Engel BT, Glassgow MS, Gaarder KR. Behavioural treatment of high blood pressure: 111. Follow-up results and treatment recommendations. Psychosom Med 1983; 45:23-9.

167. Agras WS, Harne M, Taylor CB. Expectation and blood pressure lowering effects of relaxation. Psychosom Med. 1982;44: 389- 95.

168. Wallace RK, Silver J, Mills PJ, Dillbeck MC et al. Systolic blood pressure and long term practice of the transcendental meditation and TM-Sidhi programme: Effects of TM on systolic blood pressure. Psychosom Med 1983;45:41-6.

169. Peled-Ncy R, Silverberg DS, Rosenfeld, et al. A controlled study of group therapy in essential hypertension. ISr J Med Sci. 1984; 20:12-5.

170. Luborsky L, Crits - Christeph P, Brady JP, et al. Behavioural versus pharmacological treatments for essential hypertension- a needed comparison. Psychoson Med 1982; 44:203-13.

171. Hoffman JW, Benson H, Arms PA et al. Reduced sympathetic nervous system responsivity associated with the relaxation response. Science 1982; 215: 190-2.

172. Black HR. Nonpharmacologic therapy for hypertension. Am J Med 1979; 66:837-42.

173. Manlfranz, GA, Karemaker JM, Wieling W, et al. Relaxation therapy and continuous ambulatory blood pressure in mild hypertension: a controlled study Br. Med J 1990; 300: 1368- 1372.

174. Anita FP. Clinical dieletics and Nutrition, 3rd ed. Oxford University Press, Oxford: 1989; 340-344.

175. Varley's practical clinical biochemistry, 6th ed. Heihemann Medical Books: London: 1988: 320-324.

176. Steinberg D. Metabolism of lipoproteins at the cellular level in relation to atherogenesis. In lipo proteins, Atheroselerosis and coronary heart disease, Elseveir-North Holland, 1981; 1(2): 31-48.

177. Zachariah PK, Fla Jacksonville, Steps S.G et al. Age-Related characteristics of Ambulatory blood pressure load and Mean blood pressure in normotensive subjects. JAMA 1992; 265: 1414-1417.

178. Oldham P.D. Measurement in medicine: the interpretation of numerical Data. English Universities Press, London: 1968.

179. Kirkwood B.R. Essentials of Medical Statistics. Blackwell Scientific publications, London; 1988.

180. Reisin E, Abel R, Modan M, et al. Effect of weight loss without salt restriction on the reduction of blood pressure in overweight hypertensive patients. N Engl J Med 1978; 298:1-6.

181. Stamler J, Farinaro E, Mojonnier LM et al. Prevention and control of hypertension by nutritional-hygienic means. Long- term experience of the Chicago Coronary prevention Evaluation Programme. JAMA 1980; 243: 1819-23.

182. Mac Gregor GA, Markandu N, Best F, et al. Double blind randomized crossover trial of moderate sodium restriction in essential hypertention. Lancet 1982; 1: 351-5.

183. Watt GcM, Edwards C, Hart JT et al. Dietary sodium restriction for mild hypertension in general practice. Br Med J (Clin Res) 1983; 286: 432-6.

184. Roman O, Camuzzi AL, Villalon E, et al. Physical training programme in arterial hypertension: a long term prospective follow up. Cardiology 1981; 67:230-43.

185. Davidson's principle and practice of medicine 16th ed; ELBS: Churchill Livingstone; 1991,316-392.

186. Sarker M.H, Hai MA, Khanum FA. Study of Blood pressure in relation with physical activity. Bangladesh Renal J 1988; 7(2): 57-60.

187. Krotkiewski M, Mandroukas K, Sjostrom et al. Effects of long term physical training on body fat metabolism and blood pressure in obesity. Metabolism 1979; 28: 650-8.

188. Kumar PJ, Clark ML. Clinical Medicine- A Textbook for Medical students and doctors, 2nd ed. ELBS, Bailliere Tindall 1990; 614-620.

189. Kannel, W.B et al. Systolic blood pressure, arterial rigidity and risk of stroke. J of Am Med Aoss 1981; 245:1225-1229.

190. Gordon, T. Blood pressure of adults by age and sex: United States, 1960-62. Washington,D.C. US Public Health Service, 1964 (Series II, No.4).

191. Dawber, J.R, et al. Environmental factors in hypertension. In: Stamler J, et al, ed. The epidemiology of hypertension; proceedings of an internatioal symposium New York, Grune & Stratton, 1967.

192. Wilhelmsen L, et al. Physical activity, physical fitness and risk of myocardial infarction. Advances in Cardiology 1976; 18:217.

193. Svardsudd, K & Wilhelmsen L. Change of blood pressure in relation to other variables and to development of hypertensive disease indices in a longitudinal population study. The study of men born in 1913. European Heart J 1980; 1:355.

194. WHO Technical Report Series No. 636, 1979 (Controlling the smoking epidemic: Report of the WHO Expert Committee on smoking control.

195. Chiang, B.N, et al. Overweight and hypertension a review circulation 1969; 34: 403-421.

196. Kotchen T & Havlik R., ed. High blood pressure in the young. Hypertension, 2 (Suppl,1): 1980.

197. Miall, W.E, et al. Relation between change in blood pressure and weight. Br. J of Prev and Soc Med 1968; 22: 73-80.

198. Kannel, W.B. et al. The relation of adiposity to blood pressure and development of hypertension. The Framingham study. Annals of internal medicine 1967; 67: 48-59.

199. Paffenbarger R S, et al. Chronic disease in former college students VIII. Characteristics in youth predisposing to hypertension in later years. Am J of epi 1968; 88:25-32.

200. Svardsudd K, et al. Factors associated with the initial blood pressure level and with the subsequent blood pressure increase in a longitudinal population study. The study of men born in 1913. European H J 1980; 1: 345-354.

201. Tyroler H.A, et al. Weight and hypertension: Evans country studies of blacks and whites. In: Paul, O, ed., Epidemiology and control of hypertension. New York, Stralton 1975: 177- 206.

202. Caggiula A.W, et al. The multiple risk factor intervention trial (MRFIT) iv. Intervention on blood lipids. Preventive medicine 1981; 10: 476-500.

203. Heyden S, et al. Diet treatment of obese hypertensives. Clinical Science 1973; 45:209s-292s.

204. Reisen E, et al. Haemodynamics of weight reduction in obesity hypertension. Proceedings of the Ninth Scientific Meeting of the International Society of Hypertension, Mexico city, Mexico, 21-24, 1982. Clinical Science (in press).

205. Kahn HS. Re:"Body mass index versus height and weight in relation to blood pressure: Findings for the 10,079 persons in the intersalt study: (Letters to the editor). Am J. Epidemiol 1991; 133(5): 511-512.

206. Dyer A R, Elliolt P, Shipley M, for the INTERSALT Cooperative Research Group. Body mass index versus height and weight in relation to blood pressure. Am J Epidemiol 1990; 131:589-96.

207. Voors AW, Harsha DW, Webber LS et al. Relation of blood pressure to stature in healthy young adults. Am J Epidemiol 1982; 115: 833-40.

208. Ambard L & Beaujard E. Causes de 1' hypertension arterielle. Archives of internal medicine 1904; 1: 520.

209. Dahl L.K. Salt and hypertension. Am J of Clinical Nutri. 1972; 25: 231-244.

210. Prior I.A.M, et al. Sodium intake and blood pressure in two polynesian population. New Eng J of med 1968; 279: 515-520.

211. Simpron F.O. Salt and hypertension: a sceptical review of the evidence. Clinical Science 1979; 57:463.

212. Wilhelmsen L. Salt and hypertension. Clinical Science 1979; 57:455.

213. Swales J.D. Dietary salt and hypertension. Lancet 1980: 1:1177.

214. Ellison R.C, et al. Obesity. Sodium intake and blood pressure in adolescents. Hypertension (Suppl.I) 1980; 2:1-78-1-83.

215. Watson R.I, et al. Urinary electrolytes, body weight and blood pressure. Pooled cross-sectional results among four groups of adolescent females. Hypertension (Supp-I) 1980;2:1-93-1-98.

216. Yamori Y, et al. Hypertension and diet: multiple regression analysis in a Japanese farming community. Lancet, 1981; 1: 1204-1205.

217. Sasaki N. High blood pressure and the salt intake of the Japanese. Japanese Heart J 1962; 3: 313.

218. Walker W.G, et al. Relation between blood pressure and renin, renin subtrate, angiolensin II, aldosterone and urinary sodium and potassium in ambulatory subjects. Hypertension 1979; 1: 187-91.

219. Langford, H.G & Watson, R.L. Electrolytes and hypertension. In: Poul, O, ed, Epidemiology and control of hypertension. New York, Stratton 1975: 119-30.

220. Kannel WB, et al. Cholesterol in the prediction of atherosclerotic disease. New perspectives based on the Framingham study. Annals of internal medicine 1979; 90:85.

221. Keys, A. Coronary heart disease in seven countries circulation 1970; 41(20); Suppl.1.

222. Blackburn H, et al. Conference on the health effects of blood lipids. Optimal distributions for populations. Preventive medicine 1979; 8: 612-678.

223. Oliver M.F. Br. Med Bull 1977; 37 (1): 49-58.

224. Superko H.R, et al. Am J Med 1985; 78:826.

225. Schaefer E. J. N. Eng. J Med 1985; 312: 1300.

226. Morris J.N, et al. Brit. Med J 1977;2: 1307-1314.

227. Kempner W. Treatment of hypertensive vascular disease with rice diet. Am J Med 1948; 4: 545-77.

228. McLaren D.S and Meguid M.M. Nutrition and its disorders, 4th ed, Churchill Livingstone London: 1988.

229. Staessen J, Fagard R, Lijnen P, et al. Body weight, sodium intake and blood pressure, J. Hypertens 1989; 7(Suppl 1): 519.

230. Smoller S.W, Blaufox M.D, Oberman A, et al. JAIM study; Adequate weight loss as effective as drug therapy for mild hypertension. Circulation 1990; 81:4.

231. Cohen N, and Flamenbaum W. Obesity and hypertension. Demonstration of a "floor effect Am. J. Med 1986; 80:117.

232. Vertes V, Frolkis, J.P and Martin P.J. Clinical utility of non drug therapy for hypertension. M.E. Sinai J. Med 1988; 55: 296.

233. Krotiewski M., Grimby G., Holm G, et al. Increased muscle dynamic endurance associated with weight reduction on a very low calorie diet. Am. J. Clin. Nutr. 1990; 51: 321.

234. Rocchini A.P, Key J. Bondie D, et al. The effect of weight loss on the sensitivity of blood pressure to sodium in obese adolescents. N. Eng J. Med 1989; 321: 580.

235. Havlik RJ, Hubert HB, Fabsitz RR, et al. Weight and hypertension. Ann Intern Med 1983; 98 (5 pt 2): 855-9.

236. Reisin E, Frohlich ED, Messerli FH, et al. Cardio vascular changes after weight reduction in obesity hypertension. Ann Intern Med 1983; 98: 315-19.

237. Jeffery R.W, Folgom AR, Leupker RV, et al. Prevention of over wieght and weight loss behaviour in a metropolitan adult population: the Minnesota Heart Survey experience. Am J Public Health 1984; 74: 349-52.

238. Palgi A, Bistrin, BR, Blackburn GL. Two to seven year maintenance of weight loss (Abstract) Clin Res 1984; 32: 632A.

239. Leibel RL, Hirsch J. Diminished energy requirements in reduced obese patients. Metabolism 1984; 33: 164-70.

240. Wing RR, Jeffery RW. Out patient treatments of obesity: a comparison of methodolgy and clinical results. Int J obesity 1979; 3: 261-79.

241. Bayer L.J., Kusch, F.W. Exercise therapy in hypertensive men. JAMA 1970; 211(10): 1668-70.

242. Anderson L.T., George, F.J, Tong, S.J. The year book of sports medicine, year book medical pub. Inc. Chicago: 1988:31.

243. Kukkonen K, Rauramma R, Voutilainen E, et al. Physical training of middle-aged men with borderline hypertension. Ann Clin Res 1982; 14 (Suppl 34): 139-145.

244. Duncan JJ, Farr JE, Upton J et al. The effects of aerobic exercise on plasma catecholamines and blood pressure in patient with mild essential hypertension. JAMA 1985; 258: 2609-2613.

245. Urata H, Janabe Y, Kiyonaga A, et al. Antihypertensive and volume-depleting effects of mild exercise on essential hypertension. Hypertension 1987; 9: 245-252.

246. Martin JE, Oubbert, P.M., Chshman WC. Controlled trial of aerobic exercise in hypertension. Circulation 1990; 81: 1560-1567.

247. Blumenthal, AJ, Siegel, C.W, Appelbaum M. Failure of exercise to reduce blood pressure in patients with mild hypertension- Results of a randomized controlled trial. JAMA 1991; 266(15): 2098-2104.

248. Activity and Health Research. Allied dunbar national fitness survey: a report on activity patterns and fitness levels. Main findings, London: Sports Council and Health Education authority BMJ 1992; 304: 1591.

249. Stepens J, Craig CL, Ferris BF. Adult physical activity in Canada: Findings from the Canada fitness survey. Can J Public Health 1986, 77: 285-90.

250. Royal College of Physicians of London. Medical aspects of exercise: benefitis and risks. London: RCP, 1991.

251. Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. Am J Epidemiol 1990; 132: 612-28.

252. Morris J.N, Clayton DG, Evertt MG, et al. Exercise in leisure time. Coronary attack and death rates. Br Heart J 1990; 63: 325-24.

253. Paffenbarger RS JR, Hyde, RT, Wing, AL, et al. Physical activity, all cause mortality and longevity of college alumini. N. Engl J Med 1986; 314: 605-13.

254. Blair, SN, Konl HW, Paffenbarger RS et al. Physical fitness and all cause mortality. A prospective study of healthy men and women. JAMA 1989; 262: 2395-401.

255. Franklin, B.A, Gordon, S, Timmis. Exercise in modern medicine, 1st ed. Williams and Wilkins; Baltimore: 1989: 1- 43.

256. Daphne Gloag. Exercise, fitness and health (editorial), Br. Med J 1992; 305: 377-8.

257. Grossman, E, Oren, S., Garavaglia G E, et al. Desparate hemodynamic and sympatho adrenorgic responses to isometric and mental stress in essential hypertension. Am. J. Cardio 1989; 64: 42.

258. Hagberg J.M, Montain S.J., Martin W.H, et al. Effect of exercise training in 60 to 69 year old persons with essential hypertension. Am J Cardiol 1989; 64: 348.

259. Thompson P.D., Cullinane E.M., Nugent A.M, et al. Effect of atenolol or prazosin on maximal exercise performance in hypertensive joggers. Am J Med 1989; 86 (Suppl 1B): 104.

260. Pedersen K.E, Nielsen, J.R, Klitgaard N.A, et al. Sodium content and sodium efflux of mononuclear leucocytes from young subjects at increased risk of developing essential hypertension. Am J Hypertens 1990; 3: 182.

261. L.K. Dahl. Salt intake and salt need. N. Engl. J. Med. 1958; 258: 1152-1157.

262. Subcommittee on nonpharmacological therapy of high blood pressure. Non pharmacological approaches to the control of high blood pressure. Hypertension 1986; 8: 44-467.

263. L.J. Beilin. Diet and Hypertension: Clinical concepts and controversies. J. Hypertens 1987; 5 (Suppl 5): 5447-5457.

264. F.C. Luft. Salt and Hypertension recent advances and perspectives 1989; J. Lab. Clin. Med 1989; 114: 214-221.

265. R.J. Princes and H. Black burn. Clinical and epidemiologic relationships between electrolytes and hypertension. In: NIH Workshop on Nutrition and Hypertension: Proceedings from a symposium. Biomedical information Corp. New York 1985: 63- 85.

266. R.A. Stallones. Ischaemic heart disease and lipids in blood and diet. Annu. Rev. Nutr 1983; 3: 155-185.

267. Weinberger M, Cohen S.J, Miller, J Z et al. Dietary sodium restriction as adjunctive treatment of hypertension. JAMA 1988; 259: 2561.

268. Australian National Health and Medical Research Council Dietary Salt study Management Committee. Fall in blood pressure with modest reduction in dietary salt intake in mild hypertension. Lancet 1989; 1: 399.

269. Ram C, V.S, Garrett, B.N and Kaplan NM. Moderate sodium restrictioin and various diuretics in the treatment of hypertension. Arch Intern. Med 1981; 141: 1015.

270. Falkner B., and Kushner, H. Effect of chronic sodium loading on cardiovascular response in young blacks and whites. Hypertension 1990; 50:36.

271. Niarchos A.P., Welnstein, D.I, Laragh J.H. Comparison of the effects of diuretic therapy and low sodium intake in isolated systolic hypertension. Am. J. Med 1984; 77: 1061.

272. Feldman R.D. Defective venous beta-adrenergic response in borderline hypertensive subjects is corrected by a low sodium diet. J. Clin. Invest 1990; 85:647.

273. Luft F.C and Weinberger, M.H. Review of salt restrictioin and the response to antihypertensive drugs. Satellite symposium on calcium antagonists. Hypertension 1988; 11 (Suppl): 1-229

274. Nowson, C.A and Morgan T.O. Change in blood pressure in relation to change in nutrients effected by manipulation of dietary sodium and potassium. Clin. Exp. Pharmacol. Physiol 1988; 15: 225.

275. Hargreaves M. Morgan T.O., Snow, R., et al. Exercise tolerance in the heart on low and normal salt intakes. Clin Sci. 1989; 76:553.

276. Fort J.G, Pereira Miguel JM, Pere Miguel M.J, et al. Salt and blood pressure: A community trial J. Hum. Hypertens 1989; 3: 174.

277. Stamler J, Rose G, Stamler R, et al. INTERSALT study findings. Public health and medical care implication. Hypertension 1989; 14: 570.

278. L. Tobian. Potassium and hypertension. Nutr. Rev. 1988; 46: 273-282.

279. W.N. Suki. Dietary potassium and blood pressure. Kidney Int. 1988; 34 (Suppl 25): S175-S176.

280. G.G. Krishna, E. Miller and S Kapoor. Increased blood pressure during potassium depletion in normotensive men. N Engl J Med 1989; 320: 1177-1182.

281. Knapp H.R. Hypertension- in present knowledge in Nutrition, sixth ed. International life sciences institute, Nutrition Foundation, Washington D.C 1990: 355-361.

282. Kaplan N.M., Carnegie A, Raskin P, et al. Potassium supplementation in hypertensive patients with diuretic induced hypokalemia, N. Engl. J. Med 1985; 312: 746.

283. Grimm R.H., Jr. Neaton, J.D Elmer P.J, et al. The influence of oral potassium chloride on blood pressure in hypertensive men on a low sodium diet. N. Engl. J. Med 1990;322:569.

284. Tobian I, Jahner, J.M and Johnson M.A. High K diets markedly reduce alheroseleretic cholesterol ester deposition in cortas of rats with hypercholesterolemia and hypertension. Am J. Hypertens 1990; 3: 133.

285. Khaw K.T and Barrett-Connor E. Dietary potassium and stroke-association mortality. A 12-year prospective population study. N. Engl. J. Med 1987; 316: 235.

286. R.R. Wing, A.W, Cuggiula, MP, Norwal K R et al. Dietary approaches to the reduction of blood pressure: The independence of weight and sodium/potassium interventions. Prev. Med. 1984; 13: 233-244.

287. Pekkanen J, Tinn S, Heiss G, et al. Ten year mortality from cardiovascular disease in relation to cholesterol level among men with and without pre-existing cardiovascular disease. N Engl J Med 1990; 322: 1700-1707.

288. Rouse IL, Beilin LJ, Mahoney DP, et al. Nutrient intake, blood pressure, serum and urinary prostaglandin and serum thromboxane B, in a controlled trial with a lacto- ovavegetarian diet. J Hypertension 1986; 4: 241-250.

289. Ophir, peer G, Gilad J, et al. low blood pressure in vegetarians: the possible role of potassim. Am. J. Clin Nutr 1983; 735-762.

290. Knoop R H. New approaches to cholesterol lowering efficacy and safety Hosp Pract 1988, 23 (Suppl 1): 22-30.

291. Castelli WP. The triglycerides issue: a view from Framingham. Am Heart J 1986; 112:432-437.

292. Strazzullo P, Ferro-luzzi A, siani, et al. Changing the Mediterranean diet: effects on blood pressure. J Hypertenssion 1986; 4:407-412.

293. Anderson JW, plant fibre and blood pressure Ann intern Med 1983; 98: 842-846.

294. Meyer TW, Anderson S, Brenner BM. Dietary protein intake and progressive glomerular sclerosis: the role of capillary hypertension and hyperfusion in the progression of renal disease. Ann intern MEd 1983; 98: 832-838.

295. The Trials of Hypertension prevention collaborative Research group. The effects of Non pharmacologic interventions on Blood Pressure of persons with high normal levels. JAMA 1992; 267(4): 1213-1220.

296. Boegehold MA, kotchen TA. Importance of dietary chloride for salt sensitivity of blood pressure. Hypertension, 1991: 17(suppli): 1-158-1-161.

297. Langford HG. Sodim-potassion interaction in hypertension and hypertensive cardiovascular disease. Hypertension. 1991; 17(suppli): 1-155-1-157.

298. Seedat, Y.K. Nutritional aspects of hypertension. SAMJ 1989; 75: 175-177.

299. Levy, R.I. Feilieb, M, Factors for coronary Artery Disease and their management. In Braunwald, E. ed. Heart Disease. philadelphia, WB Saunders 1984: 1246.

300. Health GW, Ehsani AA, Hagberg JM, et al. Exercise training improves lipoprotein lipid profiles in patients with coronary artery disease. Am Heart J. 1983; 105: 889-895.

301. Williams PT, Krauss RM, Wood PD, et al. Lipoprotein Subfractions of runners and sendertary men, Metabolism 1984; 35: 45-52.

302. Wood PD, Williams PT, Haskell WL. physical activity and heigh density lipo poteins. In: Miller NE, Miller GJ, eds. clinical metabolic aspects of high density lipoproteins. New York: Elsevier science publishers B.V, 1984: 133-65.

303. Goldberg L, Elliot DL. The effect of physical activity on lipid and lipoprotein levels. Med clin North Am 1985; 69: 41-55.

304. Wood PD, Stefanick ML, Exercise, fitness and athesoselerosis. In: Bouchard C, shephard RJ, stephens T, et al. eds. Exercise fitness and health, champaign, IL: Human Kineties Books, 1990: 409-24.

305. Haskell WL, Taylor HL Wood PD, et al. Strenuoms physical activity, treadmill exercise test performance, and plasma high density lipo plretein cholesterol: The lipid Research clinical program prevalenc study. Circulation 1980; 62: 53- 61.

306. Folsom AR, Caspersen CJ, TAylor HL, et al. Leisure time physical activity and its relationship to coronary risk factors in a population- based sample: the Minnessota Heart survey. Am J Epidemiol 1985; 121: 57-9.

307. Leelere S, Allare C, Talbot J, et al. High density lipoprotein cholesterol, habitual physical activity, and physical fitness. Atherosclerosis 1985; 57: 43-51.

308. Allison TG, Iammarino RM, Metz KF, et al. Failure of exercise to increase high density lipoprotein cholesterol. J Cardiac Rehab 1981; 1: 257-65.

309. Laporte R, Brenes G, Dearwater S. HDL-cholesterol across a spectrum of physical activity from quadriplegia to morathor running. Lancet 1983; 1: 1212-13.

310. Wood PD, Haskell WL, Blair SN, et al. Increased exercise level and plasma lipo protein concentration: a one-year, randomized, controlled study in sedentary, midddle- aged men. Metabolism 1983; 32: 31-9.

311. Willams PT, Wood PD, Haskell WL, et al. The effects of running mileage and duration on plasma lipoprotein levels. JAMA 1982; 247: 2674-9.

312. Drygas, Jegler A, Kurski H. study on threshold doses of physical activity in coronary heart disease prevention. Part I Relationship between leisure time physical activity and coronary risk factors. Int J sport Med 1988; 9; 275-8.

313. Robinson D, Ferns GA, Bevan EA, et al. High density lipoprotein subfractions and coronary risk factors in normal men. Arterio-risk sclerosis 1987; 7: 341-6.

314. Rauramaa R, Saloner JT, Kukkonen-Harjula K, et al. Effects of mild physical exercise on serum lipoproteins and metabolites of arachidonic acid: a controlled randomised trial in middle aged men. Br. Med J 1984; 288: 603-6.

315. Daerr WH, Windler EET, Rohwer H-D, et al. Limitations of new double-precipitation method for the determination of high density lipoprotein subsfractions 2 and 3. Atherosclerosis 1983; 49: 211-13.

316. Weintraub MS, Rosen Y, otto R, et al. Physical exercise conditioning in the absence of weight loss reduces fasting and postprandial Triglyceride-rich lipoprotein levels. circulation 1989; 79: 1007-14.

317. Cullinane E, Siconolfi S, Saritelli A, et al. Acute decrease in serum triglycerides with exercise: Is there a threshold for an exercise effect? Metabolism 1982; 31: 844-7.

318. Jacobs DR, Burge GL Tr, Liu K, et al. Relationship of low density lipoprotein cholesterol with age and other factors: a cross sectional analysis of the CARDIAC study 1988, Ann clin Res 1988; 20: 32-8.

319. Willams PT, krauss RM, Varanizan KM, et al. Effects of exercise-induced weight loss on low density lipoprotein sub fractions in healthy men. Arteriosclerosis 1989; 9: 623-32.

320. Cauley JA, laporte Re, Kuller LH, et al. The epidemiology of high density lipoprotein cholesterol levels in post menopousal women. J gerontol 1982; 37: 1D-15.

321. Kavanagh T, Shephard RT, Lindley LJ et al. The influence of exercise and life-style variables upon high density lipoprotein cholesterol after myocardial infraction. Arteriosclerosis 1983; 3: 249-59.

322. Huttumen JK, Lansimies E, voutilainen E, et al. Effect of moderate physical exercise on serum lipo proteins. A controlled clinical trial with special reference to serum high density lipo proteins circulation 1979; 60: 1220-9.

323. Saloner JT. Epidemiology of high density lipoproteins of HDL. London: Smith-Gordon, 1990: 145-54.

324. Grundy SM. Comparison of monousaturater fatty acids and carbo hydrates for lowering plasma cholesterol. W Eng J Med 1986; 314: 745-8.

325. Wood PD, stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in over weight men during weight loss through dieting as compared with exercise. N Eng J Med 1988; 319: 1173-9.

326. Lakka T.A and Salonen J.T. physical activity and serum lipids. Ann. J of epidemiol 1992; 136(7): 806-18.

327. Multiple Risk factor intervention Trial Research group. Multiple risk factor intervention trial: risk factor changes and mortality results. JAMA 1982; 248: 1465-1477.

328. Dayton S, pearce ML, Hashimoto S, et al. A controlled clinical trial of a diet high in unsaturated fat in preventing complications of altherosclerosis. Circulation 1969; 40(suppl II): 1-63.

329. Hjermenn I, velveByre K, Holme I, et al. Effect of diet and smoking intervention on the incidence of coronary heart disease. Report from the oslo study group of a randomized trial is healthy men. Lancent 1981; 2: 1303-1310.

330. Lipid Research Clinics Program. The lipid research clinics coronary primary prevention trials results: I. Reduction in the incidence of coronary heart disease. JAMA 1984; 251: 351-364.

331 Holme I An analysis of randomized trials evaluating the effect of cholesterol reduction on total mortality and cornary heart disease incidence. Circulation 1990; 82: 1961- 1924.

332. Frick MH, Eloo, Haapa K, et al. Helsink, Heart study: Primary prevention trial gemfibrozil in middle-aged men with dys lipidemia. N Eng, J Med 1987; 317: 1237-1245.

333. Lipid Research Clinical Program. The lipid research clinics coronary primary prevention trial results: I. The relationship of reduction in incidence of coronary heart desease to cholesterol lowering. JAMA 1984; 251: 315-364.

334. Hulley SB, Rosermen RH, Barvol RD, et al. Epidemiology as a guide to clinical decisions, the association between triglycerides and coronary heart disease. N Eng. J Med 1980; 302: 1383-1389.

335. Gordon DJ, Rifking BM. High-density lipoprotein-the clinical implication of recent studies. N Eng, J Med 1989; 312: 1311- 1316.

336. Brown G, Albers II, Fisher LD, et al. Regression of coronary artery disease apolipoprotein B. N Eng, J Med 1990; 323: 1289-1298.

337. Kane JP, Malloy MJ, ports TA, et al. Regression of coronary atherosclerosis during treatment of familial hypercholesterolemia with combined drug regimens. JAMA 1990; 264: 3007-3012.

338. The lovastatin study group II-Therapectic response in nonfamilial: a multicenter study. JAMA 1986; 2829-2834.

339. The lovastatin group III. A multicenter comparison of lovastatin and cholestyramin therapy for severe primary hypercholesterolemia. JAMA 1988; 260: 359-366.

340. Lavie CT, Gau GT, Squires RW, Kottke BA. Management of lipids in primary and secondary prevention of cardiovascular disease. Mayo clin proc 1988; 63: 605-621.

341. Lasrosa Jc, ed. New advances in the control of lipid metabolism, Focus on pravastation (International Congress and symposium series, No. 162). London Royal Society of Medicine Services 1989:1-43.

342. Kannel WB, Castelli WP, Gorden T, Mcnamara. Serum cholesterol, lipoproteins and the risk of coronary heart disease: the Framingham study. Ann Intern Med 1971, 74:1-12.

343. Pekkaner J, Linn S, Geiss G, et al. Ten year mortality from cardiovascular disease in relation to cholesterol level among men with and without pre existiing cardiovascular disease. N Eng L J Med 1990; 322: 1700-1707.

344. Veterans Administrative cooperative study group on Antihypertensive agents. Effects of treatment on morbidity in hypertenssion. JAMA 1970; 213: 1143-1152.

345. Hypertension Detection and Follow-up program cooperative group. Five year findings of the hypertension detection and follow-up program: mild hypertension. JAMA 1979; 242: 2562- 2571.

346. Amsterdam EA, Walker N, Ridgeucay M, et al. Physicians recognition of coronary risk factors a disparity between approach to hypertension and hypercholesterolemia in patients hospitalized on a medical service. J Am coll cardiol 1987; 9: 79 A.

347. Samuelsson O, wilhelmsen L, Anderson DK, et al. Cardiovascular mortality in relation to change in blood pressure and serum cholesterol levels in treated hypertension: results from the primary prevention trial in Goteborg Sweden. JAMA 1987; 258: 1768-1776.

348. Houston Mc, New insignts and new approaches for the treatment of essential hypertension: selection of therapy based on coronary heart disease risk factor analysis, haemodynamic profiles, quality of life and subsets of hypertension. Ann Heart J 1989; 117: 911.

349. Grundy SM. Cholesterol and coronary heart disease: a new era. JAMA 1986; 256: 2849-2858.

350. Larosa Jc, Hunninaghke D, Bush D, et al. The cholesterol facts. A summary of the evidence relating dietary fats, serum cholesterol and coronary heart disease. A joint statement by the American Heart Association and the National Heart, lung and Blood institute. circulation 1990; 81: 1721-1733.

351. Schueken B, Wittes JT, Sentonello NC, et al. Change in cholesterol awareness and action. Result from National physician and public surveys. Arch intern Med 1991; 151: 666-673.

352. Grundy SM, Greenland P, Hend A, et al. Cardiovascular and risk factor evaluation of healthy America adults circulation 1987; 75: 1340A-1350A.

353. Kaplan NM. Maximally reducing cardiovascular risk in the treatment of hypertension. Ann intern Med 1988; 109: 36-40.

354. The expert panel. Report of the National cholesterol Education program expert panel on detection, Evaluation and Treatment of high Blood cholesterol in adults. Arch intern Med 1988; 148: 36-69.

355. Study group. European Atherosclerosis society. Strategies for the prevention of coronary heart disease: a policy statement of the European Atherosclerosis Society. Eur Heart J 1987; 8: 77-88

356. Grundy SM, NiX D, whelan MF, et al. Comparsion of the cholesterol-lowering diets in normolipidemic men. JAMA 1986; 256: 2351-2355.

357. Grundy SM. Comparison of mono-unsaturated fatty acids and carbohydrates lowering diets for lowering plasma cholesterol. N Eng J Med 1986; 314: 745-748.

358. Knoop RH. New approaches to cholesterol Lowering: efficacy and safety. Hosp pract 1988, 23 (Suppi): 22-30.

359. Consolazio CF, Johnson RF, Pecora LJ: physiological Measurements of Metabolic functions in Man. New York, Mcgraw Hill, 1963: 7.

360. Woods HF, Alberti KHMM. Dangers of intravenous fructose. Lancet 1972; 2: 1354-7.

361. Hodges RF, Rebello T. Carbohydrate and blood pressure. Ann Intern Med 1988; 98(part 2): 838-41.

362. Seedat Y.K. Hypertension as an insulin resistant state: A newly discovered concept. (paper delivered at a meating on Advances in Cardiology; held is Swaziland in Dec, 1989) Sponsored supp- SAMJ 1990; 1-4.

363. Ferrannini E, Defronzo RA. The association of hypertension, diabetes and obesity: a review. J Nephol 1989; 1: 3-16.

364. Ferrannini E, Hypertension: An insulin- resistant state paper presented at a meeting on the Role of insulin Resistence, Boca Raton, Florida: 1989;3-4.

365. Kaplan NM. Importance of coronary heart disease risk factors in the management of hypertension: an overview. Ann J Med 1989; 86: Supl IB, 1-4.

366. Singer P, Godicke W, voight S, et al. postprandial hyperinsulinacmia in patients with mild essential hypertension, Hypertension 1985; 7: 182-186.

367. Reaver GM. Role of insulin resistence in human disease. Diabetes 1988; 37: 1595-1606.

368. Modan M, Halkin H, Almog S, et al. Hyperinsulinemia: a like between hypertension, obesity and glucose intolerance. J clin invest 1985; 75: 809-817.

369. Ferrannini E, Shieh SM, Fuh MT, et al. Insulin resistence in essential hypertension. N Eng I J Med 1987; 317: 350-357.

370. Swislocki ALM, Hoffmar BB, Reaven GM. Insulin resistence, glucose intolerance and hyperinsulinaemia in patients with hypertension, Ann J Hypertens 1989; 2: 419-423.

371. Kaplan NM. The deadly quartet, upper-body obesity, glucose intolerance, hypertriglyceridemia and hypertension. Arch Inter. Med 1990; 39: 167.

372. Pollare T, Lithell H, and Berne C. Insulin resistance is a chracteristic feature of primary hypertension independent of obesity. Metabolism 1990; 39: 167.

373. Bjorntorp p. Classification of obese patients and complication related to the distribution of surplus fat. Nutrition 1990; 6: 131.

374. Peiris AN, Mueller R.A, Smith G.A, et al. Splanchnic insulin metabolism in obesity: Influence of body fat distribution. J. Clin. invest. 1986; 78: 1648.

375. Stremblad, G, and Bjorntrop, p. Reduced hepatic insulin clearence in rats with dietary-induced obesity. Metabolism 1986; 35: 323.

376. Ostlund, R.F. Jr, stater M, Konrt W.M, et al. The ratio of waist-to-hip circumference, plasma insulin level, and glucose intolerance as independent predictors HDL_7 cholesterol level in older adults. N Engl. J. Med 1990; 322: 229.

377. Rowe J.W, yound J.B, Minaker K.L, et al. Effect of insulin and glucose infusions on sympathieic nervons system activity in normal man Diabetes 1983; 30: 219.

378. Baum M. Insulin stimulates volume absorbtion in the rabbit proximal convoluted tubule. J. clin. invest. 1987; 79: 1104.

379. Bjorntorp p, de Jounge K, Sjostrom L, et al. The effect of physical training on insulin production in obesity. Metabolism 1976; 19: 1631-8.

380. Seals DR. Hagbarg JM, Hurley BF, et al. Effects of endurance training on glucose tolerance and plasma lipids levels in older men and wemen. JAMA 1984; 252: 645-9.

381. Donahue RP, orchard TJ, Becker DJ, et al. physical activity, insulin sensitivity and lipoprotein profile in young adults: The Beaver county study. Ann J Epidemiol 1988; 127: 95-103.

382. Jacob RG, Shapiro AP, Reeves RA, et al. Relaxation therapy for hypertension comparison of effects with concomitant placebo, diuretic and beta- blockers. Arch Intern Med 1986; 146: 2335-40.

383. Cottier C, Shapiro K, Jullius S, Treatment of mild hypertension with progressive muscle relaxation. Predictive value of indices of sympathetic tone. Arch Intern hed 1984; 144: 1954-8.

384. Patel C, Marmot MG, Terry DJ, et al. Trial of relaxation in reducing coronary risk: four year follow up. Br. Med. J 1984; 290: 1103-6.

385. Bali LR. Long term efffect of relaxation on blood pressure and anxiety levels of essential hypertensive males: a controlled study. Psychom Med 1979; 41: 637-46.

386. Patel C. Yoga and biofeedback in the management of hypertension. lancet 1973; 3: 1053-5.

387. Patel C. 12 month follow-up of yoga and biofeedback in the management of hypertension. Lancet 1975; 1: 62-5.

388. Patel C, North WRS. Randomised controlled trial of yoga and biofeedback in the management of hypertension' Lancet 1975; 3: 93-5.

389. Patel C, Marmot M, Can general practitioners use trainiing in relaxation and management of stress to reduce mild hypertension? Br, Med J 1988; 296: 21-4.

390. Shapiro AP, Schwarz GE, Ferguson DCE, et al. Behavioural methods in the treatment of hypertension. A review of their clinical status. An intern Med 1977; 86: 626-36.

391. Patel C, Marmot MG. Stress management, blood pressure and and quality of life. J Hypertension 1987; 5 (Suppi) S 21.

392. Subcommittee on Detection, Evaluation and Treatment of high blood pressure. Nonpharmacological approaches to the control of high blood pressure. Final report. Hypertension 1986; 8: 444-67.

393. Andrews G, Macmahon SW, Austin A. Hypertension: Comparison of drug and non-drug treatments. Br Med J 1984; 284: 1523-6.

394. Mortfans GA, Karemaker JM, Wieling W, et al. Relaxation therapy and continous ambulatory blood pressure in mild hypertension: a controlled study. Br Med J 1990; 300: 1368- 71.

395. Brauer AP, Horlick L, Nelson E, et al. Relaxation therapy for essential hypertension: a veterans Administration out patient study. J Behav Med 1979; 2: 21-9.

396. Jacobson E. Variation of blood pressure. Ann intern Med 1939; 1194: 212.

397. Benson H. Systematic hypertension and the relaxation response. N Engl J Med 1977; 296: 1152-6.

398. Shapiro AP, Schwary GE, Fergusen DCE, et al. Behavioural methods in the treatment of hypertension. A review of their clinical status. An Intern Med 1977; 86: 626-36.

399. Patel C. Meditation in general practice. Br Med J 1981, 282: 528-562.

400. MClean J. The use of relaxation techniques in general practice. The practitioner 1986; 230: 1079-84.

401. Benson H. The relaxation response, collins, London: 1975: 158.

402. Davidson, P.O, schwartz GE. The psychobiology of relaxation and related states-a multiprocess theory, in Mosstates - a multiprocess theory, in Mostofsky DI(ed) Behaviour control and modification of physiological activity, prentice Hall, New York 1979: 399-442.

403. Everly GS, Rosenfeld R. The nature and treatment of the stress response-a practical guide for clinicans, plenum, New York: 1981: 215.

404. Patel C. Twelve month follow up of yoga and biofeedback in the management of hypertension. Lancet 1975; 1: 62-64.

405. Pellitier K. Mind as healer, mind as slayer. A holistic approach to preventing stress disorders, George Allen and unwin, London: 1978; 366.

406. Pietroni P. Holistic living, Dent, london: 1986: 240.

407. Shapiro DH, walsh RNC (eds). Meditation-classic and contemporary perspectives. Aldine, New York: 1984: 722.

408. Steptoe A, Mathews A. Health care and human behaviour, A cadeic press london: 1984: 347.

409. Petal C and Marmot M. Can general practitioners use trainiing in relaxation and management of stress to reduce mild hypertension? Br. Med J 1988; 296: 21.

410. Mc Grady, A and Higgins JT. Prediction of response to biofeedback-assisted relaxation in hypertensives: Development of a hypertensive predictor profile(Hy pp). Psychosomatic Med 1989; 51: 27.

411. Cramer JA, Mattson, R.H, prevey M.T, et al. How often is medication taken as prescribed? A novel assessmant technique. JAMA 1989; 261: 3273.

412. Kuller L, Neton J, Caggiula A, et al. Primary prevention of heart attacks: The multiple risk factor intervention trial. Ann J Epidemiol 1980; 112: 185-199.

413. Estein KA, schneiderman LJ, Bush JW, et al. the "abnormal" screening serum thyroxine (T4): Analysis of physician response, out come, cost and health effectiveness. J chron Dis 1981; 34: 175-190.

414. Feinstein AR: Clinical Epidemiology. The Architecture of clinical Research. Philadelphia, WB Saunders, 1985.

415. Freis E.D. Should mild hypertension be treated? N. Eng 1 J Med. 1982; 307: 306-309.

416. Medical Research council working party. MRC trial of treatment of mild hypertension: principal results. Br. Med. J 1985; 291: 97-104.

417. Intersalt Cooperative Research group. Intersalt: an international study of electrolyte excretion and blood pressure; results for 24 hour urinary sodiam and potassium excretion. BMJ. 1988; 297: 319-328.

418. Boegehold MA, kotchen TA. Importance of dietary chloride for salt sensitivity of blood pressure. Hypertension 1991; 17(Supp I): 1-58-1-161.

419. Langford HG. Sodium-potassium interaction in hypertension and hypertensive cardiovascular disease 1991; 17(Supp I); 1-155- 157.

420. Fraser G. phillips R, Harris R. Physical fitness and blood pressure in school children. Circulation 1983; 67: 405-412.

421. Blair S, Good year N, Gibbons L, et al. Physical fitness and incidence of hypertension in healthy normotensive men and women. JAMA 1984; 252: 487-490.

422. Alderman M, Davis T, Gerber L, et al. Antihypertensive drug therapy withdrawal in a general population. Arch Intern Med 1986; 146: 1309-1311.

423. Bouita R; Beglehole R. Increased treatment of hypertension dose not explain the decline in stroke mortality in the united statee 1990-1980. Hypertension 1989; 13(Supp I)1.

424. Chatellier G, Battaglia C, pagny JY, et al. Decision to treat mild hypertension after assessment by ambulatory monitoring and WHO recomnendations. Br. Med. J 1992; 305: 1062-205.

425. Mancia G, parati G. clinical significance of "White coat" hypertension. Hypertension 1990; 16: 626-6.

426. Management Committee of the Australian therapeutic Trial on Mild Hypertension. Untreated mild hypertension Lancet 1982; 1: 185-91.

427. The 1984 report of the Joint National Committee on Detection, evaluation and treatment of high blood pressure. Arch Intern Med, 1984; 144: 1045-57.

428. Bonita R, and Beaglehole R. Increased treatment of hypertension does not explain the decline in stroke mortality in the United States, 1970-1980. Hypertension 1989; 13 (Suppl 1): 1-69.

429. Hart J. T. The practioner's view. In Gross, F, and Strasser, T (eds). Mild hypertension: Recent Advance. New York, Raven Press, 1983; 365.

430. Bergland, G. Goals of antihypertensive therapy. Is there a point beyond which pressure reduction is dangerous? Am. J. Hypertens 1989;2:586

431. Cutler J.A, MacMahon W, and Furberg CD. Controlled Clinical trial of drug treatment for hypertension. A review. Hypertension 1989; 13 (Supp 1): hypertension. A review. Hypertension 1989; 13 (suppl): 1-36.

432. U S Public Health Service Hospitals co-operative study grop. Smith, W.M. Treatment of mild hypertension. Results of a ten-year intervention trial. Circ. Res 1977; 40 (Suppl 1): 1-98.

433. Perry HM, Goldman, A.I, Lavin MA, et al. Evaluation of drug treatment in mild hypertension: VA. NHLBi feasibility trial. Ann NY Acad. Sci 1978; 304:267.

434. Helgeland A. Treatment of mild hypertension. A five year controlled drug trial. The oslo study. Am. J. Med 1980; 69:725.

435. Management Committee. The Australian therapeutic trial in mild hypertension, Lancet 1980; 1:1261.

436. Amery A, Brikenhager W, Brixko, P et al. Mortality and morbidity results from European working party on high blood pressure in the elderly trial. Lancet 1985; 1:1349.

437. Menard J. Hypertension costs. Source, evaluation and impact of cost-containment measures in various health-cares systems. Clin. Exp. Hypertens 1989, A 11:1149.

438. Roland E, Schmieder MD, Jurgen K, et al. Anti hypertensive therapy: To stop or not to stop. JAMA 1991; 265: 1566-1571.

439. Pickering TG. Predicting the response to nonpharmocologic treatment in mild hypertension (editorial). JAMA 1992; 267: 1256-1257.

440. Longford HG, Davis BR, Blanfox D, et al. Effect of drug and diet treatment of mild hypertension on diastolic blood pressure. Hypertension 1991; 17:210-219.

441. Jacob RG, Chesney MA, Williams DM, et al. Relaxation therapy for hypertension: design effects and stress effects. Ann Behav Med 1991; 13: 5-17.

442. Khaw KT, Barrett Conner E. Dietary potassium and stroke-association mortality: a 12 year prospective population study. N Engl J Med. 1987, 315: 235-240.

443. Burr ML, Fehily AM. Gilbert JF, et al. Effects of changes in fat, fish and fibre intakes on death and Myocardial reinfarction: diet and reinfarction trial (DART) Lancet 1989; 2:757-761.

444. Kawasahi T, Delea CS, Barlter FC, et al. The effect of high-sodium and low sodium intakes on blood pressure and other related variables in human subjects with idiopathic hypertension. Am J Med 1978; 64: 193-198.

445. Harrson's principles of Internal Medicine 12th ed. Mac G Raw Hill, Inc. 1991:1002.

STUDY ON METABOLIC CONTROL OF HYPERTENSION

QUESTIONNAIRE FORM

Instrument - A
Serial No. 1

PART - I

Perrsonal data

Personal Number _____

Name _____

Age _____

Rank/Status _____

Unit _____

Nature of Job/Trade _____

Height (Inches) _____

	Baseline	After intervention
Weight (Kg)		

History of smoking	Nil	I-IO	>IO

Clinical data

Routine test of : Blood, Urine, ECG and X-ray Chest

	Normal	Abnormal
Blood		
Urine		
ECG		
X-Ray Chest		

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PART - II

Clinical Feature

Baseline	Symptoms present

Headache	Griddiness	Insomonia	Palpitation	Fatigue

After Intervention	Symptoms Absent

Headache	Griddiness	Insomonia	Palpitation	Fatigue

History of complications	Yes	No

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Instrument - B
Serial No. 2

Blood Pressure (Auscultatory) Measurement

Systolic in mmHg/Diastolic in mmHg

Baseline ☐ ☐ ☐

After intervention

Day of Recording	Morning	Evening
1		
2		
3		
4		
5		
6		
7		
8		
9		
10		
11		
12		
13		
14		
15		
16		
17		
18		
19		
20		
21		

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Instrument C

Serial no. 3

Blood chemistry

	Baseline		After intervention
Serum Electrolytes			
Serum Na		mmol/L	
Serum K		mmol/L	
Serum Cl (Nacl)		mmol/L	
Lipids profile			
Serum Cholesterol		mg/dL	
HDL-Cholesterol		mg/dL	
LDL-Cholesterol		mg/dL	
Serum Triglycerides		mg/dL	
Serum/β-lipoprotein		mg/dL	
Serum total lipids		mg/dL	
Urea/Creatinine			
Serum Urea		mg/dL	
Serum creatinine		mg/dL	

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Others

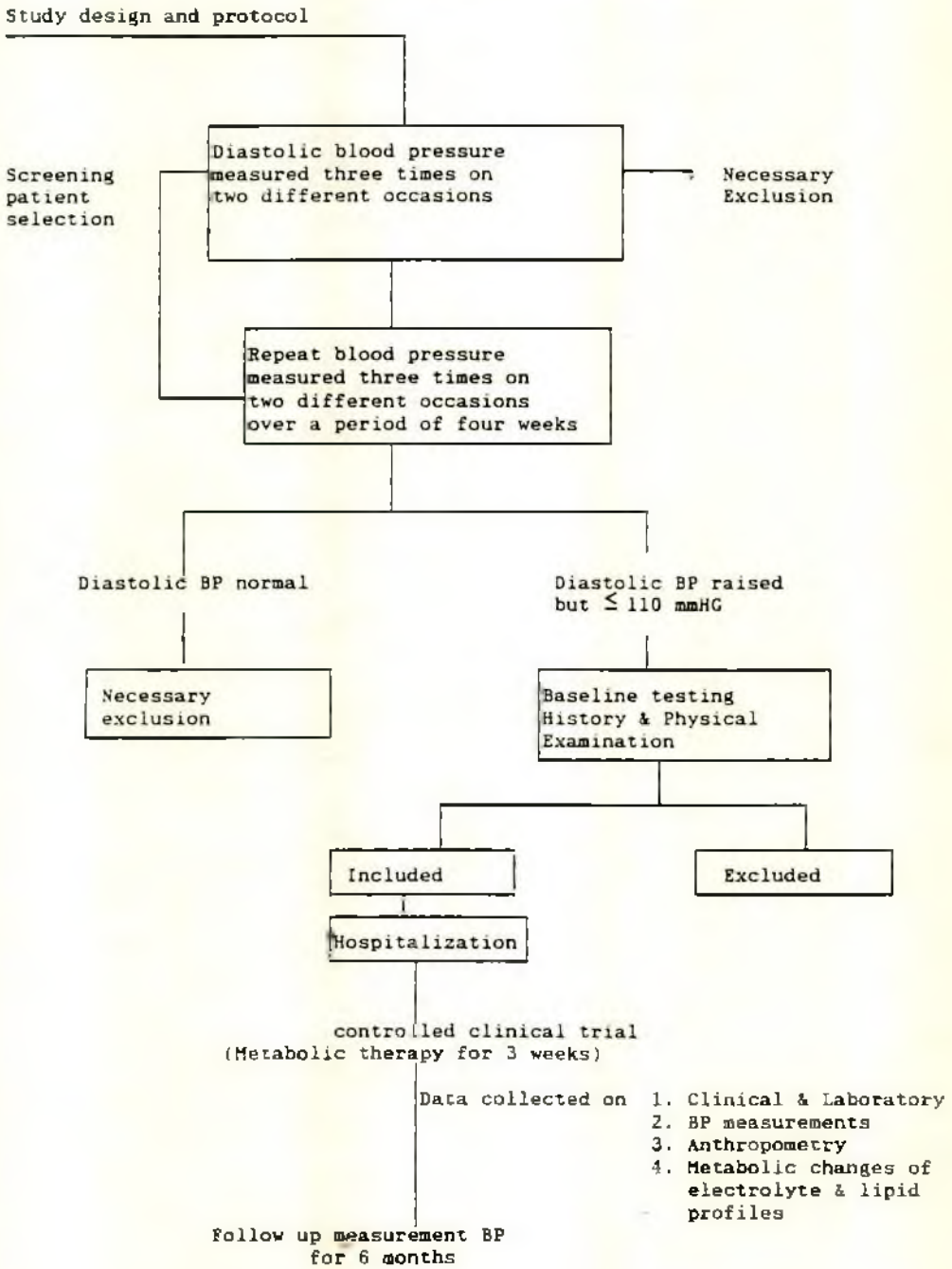
Serum total protein		mg/dL	
Two hours post prandial glucose		mg/dL	
Fasting plasma glucose		mg/dL	

Date:

Signature of the Investigator

P-38I

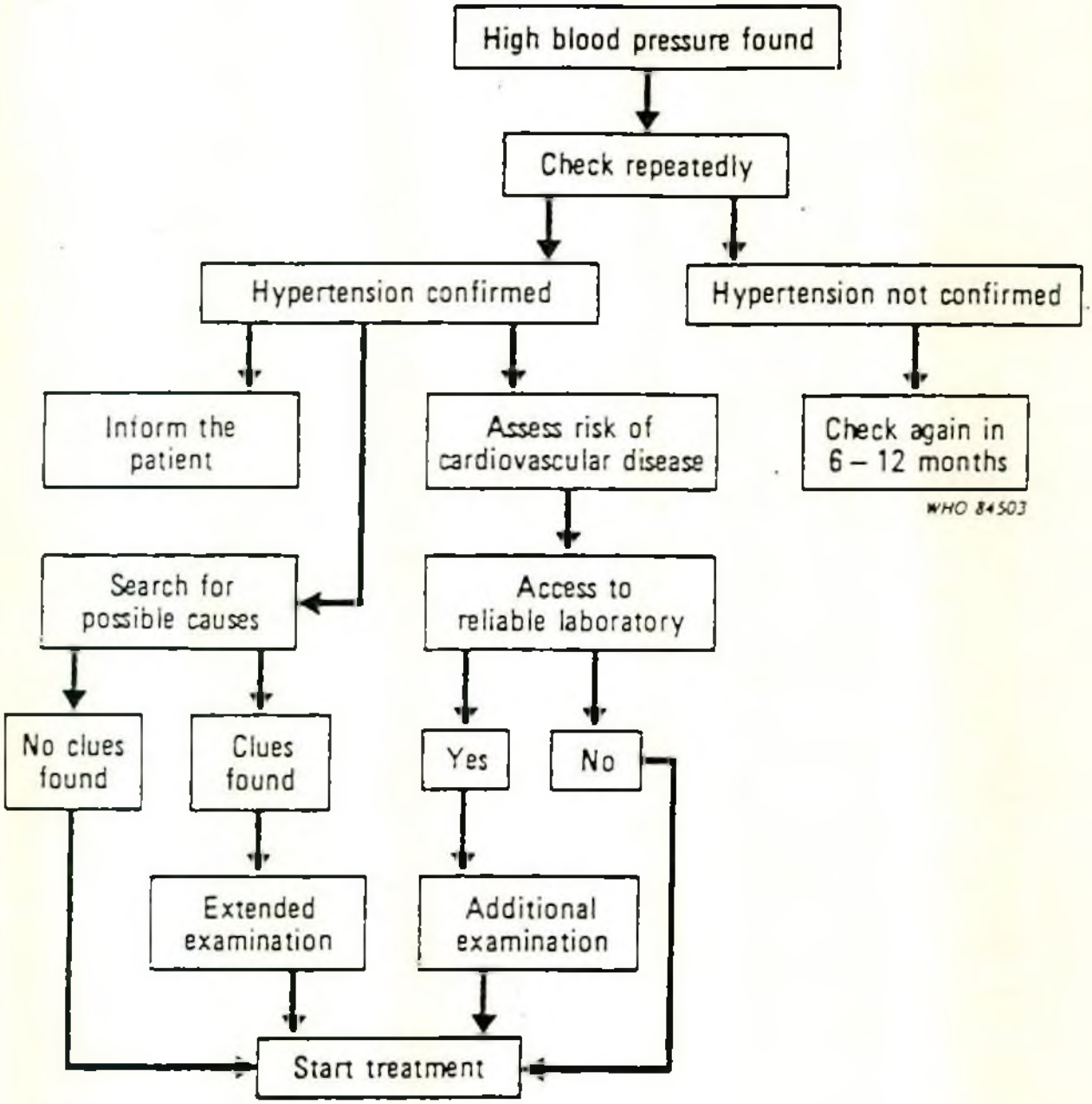
FLOW CHART OF STUDY PROCEDURE



সম্মতি পত্র

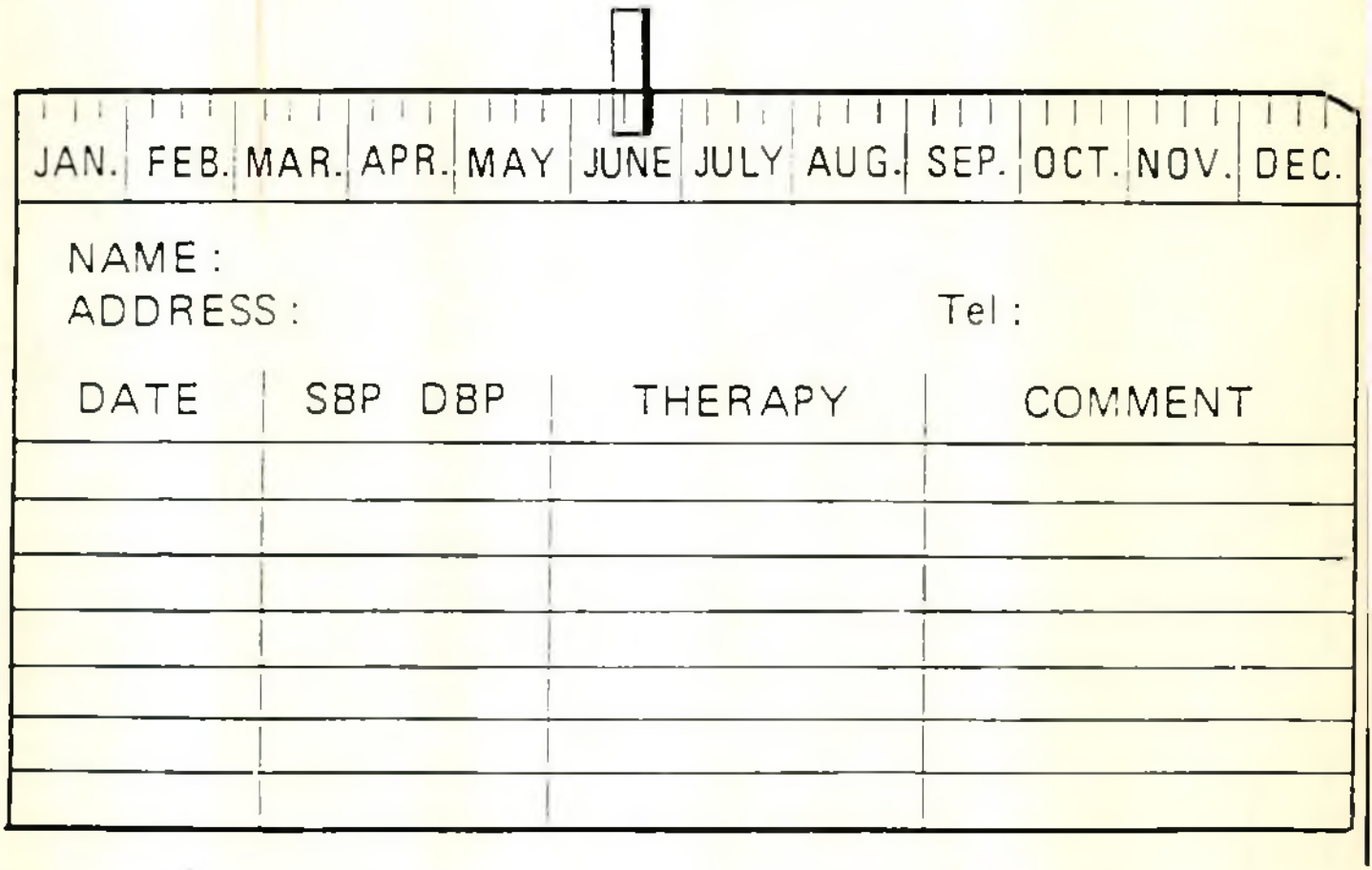
উচ্চ রক্তচাপ একটি মারাত্মক রোগ। এই রোগ সারা জীবন চিকিৎসা করতে হয়। ফলে এই রোগের চিকিৎসার ব্যয়ভার অনেক এবং আজীবন ঔষধ সেবন স্বাস্থ্যের জন্যও ক্ষতিকর। আমরা যদি কিছু কিছু স্বাস্থ্যবিধি যেমন, খাদ্যাভাস পরিবর্তন, লবন কম খাওয়া, প্রচুর বিশ্রাম ও ব্যায়ামের অভ্যাস করি তবে এই রোগ, রোগী নিজেই তার রক্ত চাপ নিয়ন্ত্রনে রাখতে পারবেন। এই উদ্দেশ্যকে সামনে রেখে আমরা আপনাকে ঔষধ ছাড়া স্বাস্থ্য বিধি পালনের মাধ্যমে চিকিৎসা করতে চাই। এই জন্য আপনার সম্মতি ও সহযোগিতা একান্তভাবে কামনা করি। আপনি ইচ্ছা করলে এই চিকিৎসা ব্যবস্থা হতে বিরত থাকতে পারেন।

APPENDIX -D TO
SECTION 2.9



Assessment of the hypertensive patient-sequence of action to be followed

APPENDIX -E TO
SECTION 2.17



File-card with rider indicating next appointment in active follow up programme.

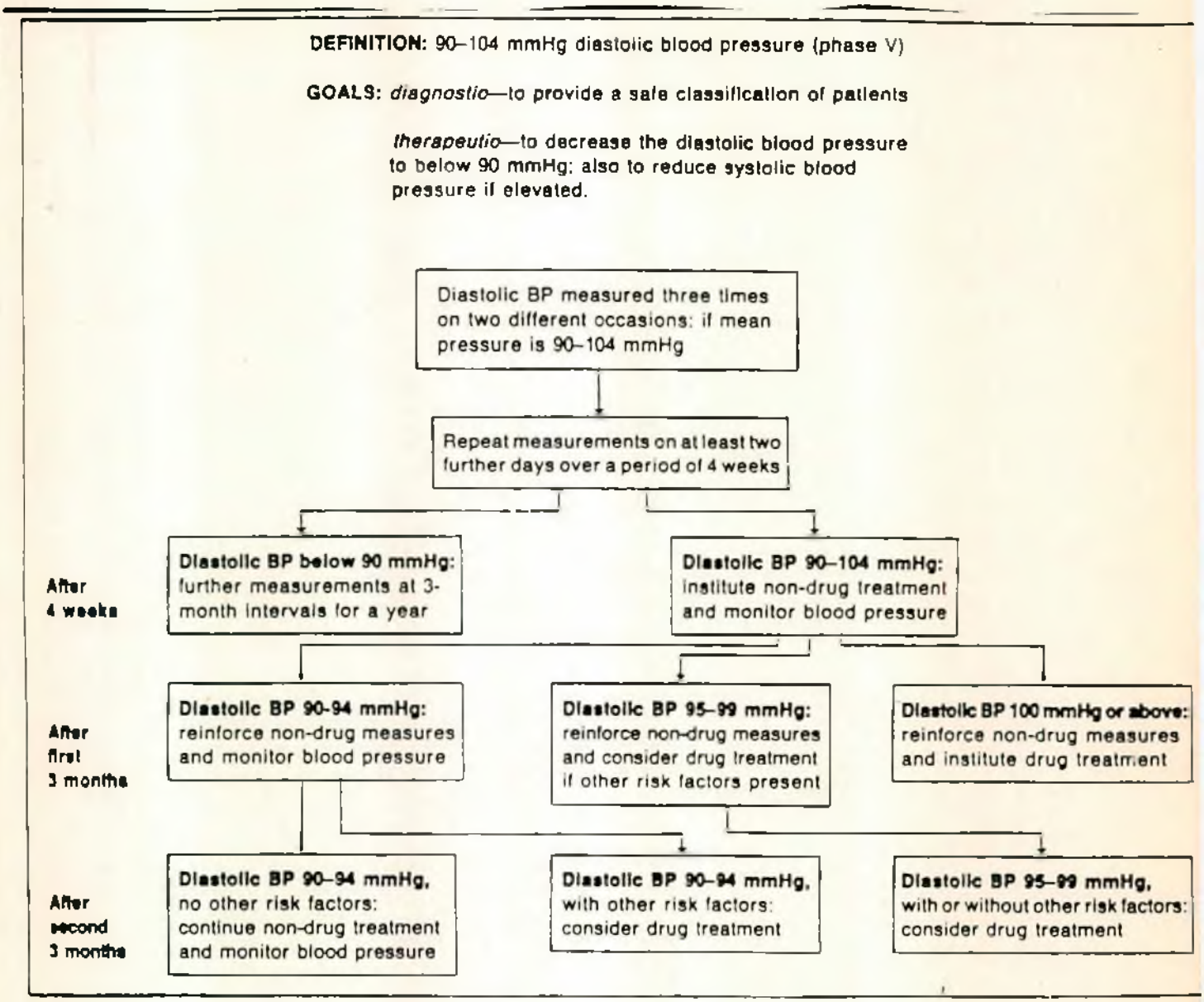
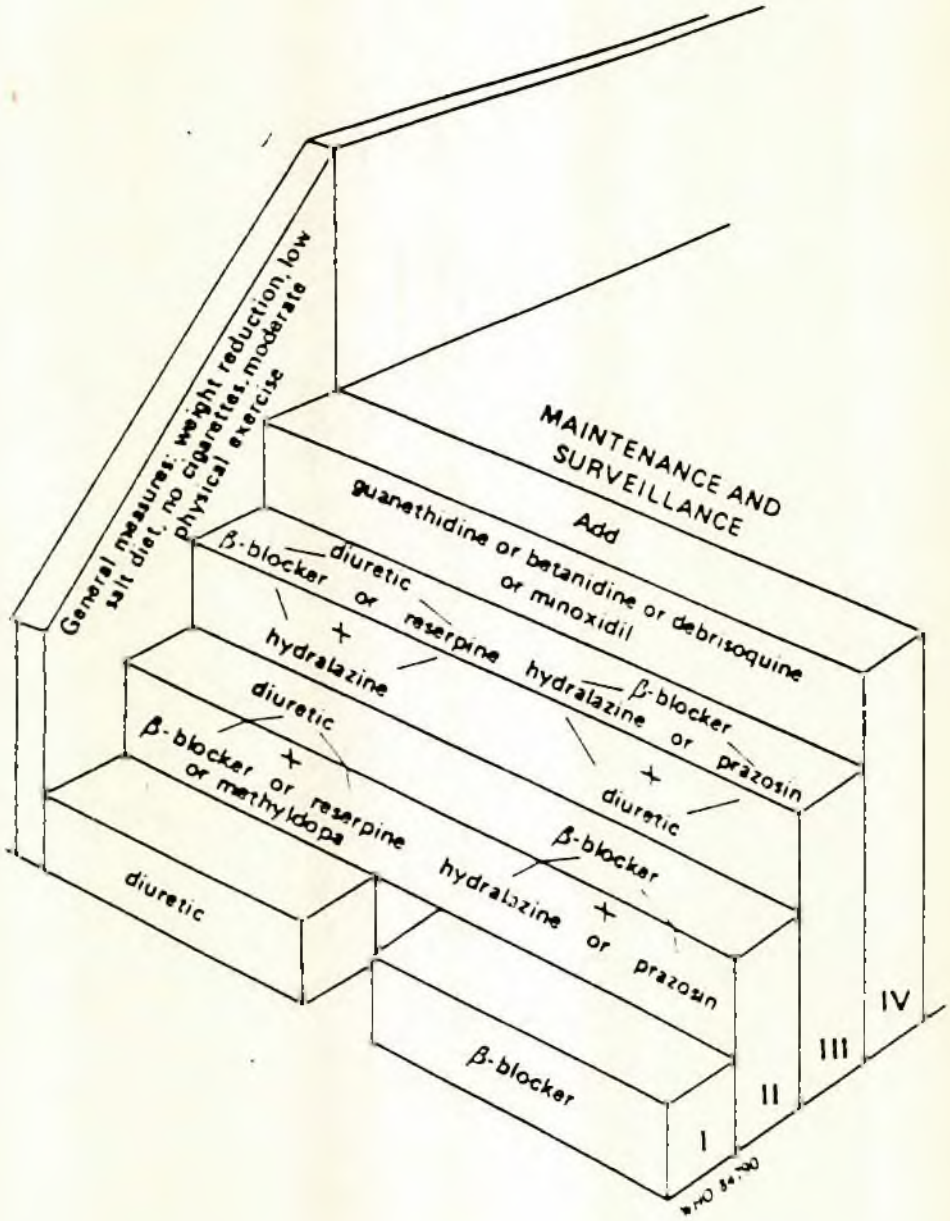


FIGURE.1.10. Definition, blood pressure measurement, and management of hypertension. (From WHO/ISH Mild Hypertension Liaison committee.1989 guidelines for the management of mild hypertension: Memoran dum from WHO/ISH meeting.J. Hypertens,1989;7:689.



A stepped therapeutic progeamme

আমি বিজে-৩৬৪৯৫ নাঃ সুবেদার আতাউর রহমান (অবঃ) পুরকে অর্ডনান্স ডেপো রাজেন্দ্রপুর সেনানিবাস বয়স ৫৯ বৎসর। আমার রোগ বিবরন নিম্নে পেশ করিলাম।

বিগত ১০/১/৯২ ইং হইতে আমার উভয় বাহুতে, বুকের বামদিকে, বাম পিঠে ও ঘাড়ে ব্যথা অনুভব করি। ব্যথা সামান্য অনুভব হওয়াতে বিশেষ গুরুত্ব দেই নি। ১১/১২ দিনপরে ব্যথা এমশঃ বৃদ্ধি পাওয়ায় ক্যান্টনমেন্ট জেনারেল হাসপাতাল হইতে ঔষধ সেবন করিয়া কোন ফল পাই নাই বিধায় বাহিরে একজন প্রবিন চিকিৎসকের নিকট যাইয়া চিকিৎসার অনুরোধ জানাইয়া আমার অসুখের বর্ননা দিলে তিনি আমাকে নানা ভাবে পরীক্ষা করতঃ নিম্ন লিখিত ঔষধ সেবন করিতে পরামর্শ দিলেন :-

১। টেবলেট টেনরমিন

২। টেবলেট ইসরডিন

৩। টেবলেট মিডাকসিক

৪। টেবলেট ভলটারেন

৫। টেবলেট এক্টাসিড

৬। টেবলেট বুডিট

প্রকাশ থাকে যে, আমার বি পি ২০০/১৪০ ছিল, কিন্তু উল্লেখিত ঔষধাবলি খাওয়ার পর তেমন কোন ফল পাই নাই। পরিশেষে সম্মিলিত সামরিক হাসপাতালেস্থ এম আই রুম এ উপস্থিত হইলে আমাকে মেজর মতিন সাহেব মেডিক্যাল স্পেশালিষ্ট সাহেবের কাছে পাঠান। সেখান হইতে মেজর মোঃ শামসুল হক সাহেব আমাকে জেসিও ওয়ার্ডে ভর্তি করিয়া নানারূপ পরীক্ষা নিরিক্ষা করিয়া কি ভাবে ঔষধ ব্যতিরেকে উচ্চ রোগ (উচ্চ রক্তচাপ) নিরাময় হইতে পারে যে বিষয়ে আমাকে উপদেশ দিলেন ও ফিজিওথেরাপি বিভাগের উদ্যোগে মেশিন সহ নানারূপ ব্যায়াম পদ্ধতি করার নির্দেশ দেন এবং খাদ্য তালিকা নিরুপন করিয়া দেন। তাঁহার নির্দেশানুযায়ী আমি ৮/৩/৯২ ইং হইতে তালিকানুযায়ী খাদ্য গ্রহন করিয়া নিয়মিত ব্যায়াম করিতে থাকি। অদ্য ১৫/৩/৯২ ইং হইতে আমি সম্পূর্ন সুস্থ্য বলিয়া মনে করি। অদ্যকার বি পি ১৩০/৮০ এবং ১৮ দিনের ব্যায়াম কোর্স সম্পূন্ন করিলে এ জাতিয় রোগ নির্মুল হইয়া যাইবে বলিয়া মনে করি।

চলমান পাতা ১-২ দ্রষ্টব্য

একজন ভুক্তভোগী রোগী হিসাবে আমি এই সমস্ত চিকিৎসা প্রক্রিয়াকে বর্তমান আধুনিক যোগের অর্থাৎ বিংশ শতাব্দীর এক অত্যাশ্চর্য চিকিৎসা পদ্ধতি বলিয়া মনে করি এবং পরিশেষে বলিতে চাই এই সমস্ত চিকিৎসা পদ্ধতির আবিষ্কারক ও সুযোগ্য চিকিৎসকবৃন্দ ভূয়সী প্রশংসার দাবিদার ও মানব জাতির গৌরব ।

আমি তাঁহাদের দীর্ঘ জীবন কামনা করি ।

ইতি -

(আতাউর রহমান)
[illegible], ১২০০/সি
স: মু: (অব:)
১৮/৩/১৯৯২ ইং

APPENDIX-J

MEDICAL HISTORY FORM

Med Unit : Clinical Clerk

Ward No :

Bed No :

Name of the Patient : _____

Age: _____ Sex : _____ Religion : _____

Occupation: _____ Marital Status: _____

Date of admission :_____

Date of Examination :_____

Address: _____

1.1 Chief complaints:

1. _____

2. _____

1.2 History of present illness:

_____

_____

1.3 History of past illness:

_____

_____

1.4 Family History

_____

_____

1.5 Drug History

_____

_____

1.6 Personal History

_____

_____

GENERAL EXAMINATION

1. Appearance_____
2. Build _____
3. Nutrition _____
4. Skin _____ eruption/pigmentation
5. Anaemia _____
6. Cynosis
7. Jaundice
8. Oedema_____
9. Uk-oilonychia_____
10. Clubbing
11. Neck vein _____
12. Lymphonodes
13. Wasting_____
14. Thyroid_____
15. Body hair _____
16. Obesity_____
17. Respiration rate _____
18. Temperature _____
19 Pulse_____
20 Blood Pressure_____
21 Heart_____
22 Lungs_____
23 Liver _____
24 Spleen _____

CARDIOVASCULAR SYSTEMS

1.1 INSPECTION

1. Dyspnea _____
2. Precordium _____
3. Any pulsation _____
4. Apex beat _____
5. Engorged vein _____
6. Ankle oedema _____
7. Clubbing _____

2.2 PALPATION

1. Apex beat _____
2. Epigastric pulsation _____
3. Thrill _____
4. Pulsatile liver _____
5. Supra sternal pulsation _____

2.3 PERCUSSION

1. Area of cardiac dullness
2. Area of liver dullness
3. Lung resonance

2.4 AUSCULTATION

HEART SOUND

	1st	2nd
1. Mital area		
2. Tricuspid		
3. Pulmonary		
4. Aortic		

Murmur _____
Splitting _____
Triple Rythm _____
Pericardial Friction _____
Basal Crepitation _____

ALIMENTARY SYSTEM

1.1 INSPECTION :_____

1.2 PALPATION :_____

1. Liver _____
2. Kidney _____
3. Spleen _____

1.3 Percussion _____

1.4 Auscultation _____

RESPIRATORY SYSTEM

1.1 INSPECTION :_____

1.2 PALPATION:_____

1.3 PERCUSSION:_____

1.4 AUSCULTATION:_____

NERVOUS SYSTEM

1. Behaviour _____
2. Emotion _____
3. Intelligence _____
4. Co-operation _____
5. Memory _____
6. Sleep _____
7. Speech _____
8. Examination of cranial nerves _____

_____

9. Motor Function _____

Muscles _____

Reflexes _____

Gait _____

Involuntary movement _____