Chapter I - I

Introduction
Cardiovascular disease rank number one in the United States in causing morbidity and mortality. This disease account for about one half of all deaths in the United States. Approximately 1.5 of million myocardial infarction and 5,20,000 deaths occur per year. Although cardiovascular disease is considered an acquired problem of industrialised western world, the improvement in the socio-economic industrial base in the developing countries has brought this problem to the doorstep of third world countries. In a press report it was revealed that about 40 million people in Indian subcontinent suffer from heart disease. The statistics in our country shows that after the age of 30, 33 out of 1000 suffer from CAD and in high risk group 22 out of 1000 sustain strokes.

Experience from Vellore Medical Mission Hospital, revealed an increase in proportion of patients with CAD from 4% to 33% during a thirty year period from 1960 to 1989. A study conducted by Marthur from Agra revealed that about 1.05% persons being have coronary heart disease. Padmavathi in a random population survey in Delhi, identified 1.03% of persons have this disease.
Large scale epidemiological studies have demonstrated that coronary artery disease and its complications have been associated with a variety of risk factors. Coronary risk factors refer to conditions which have been demonstrated by statistical procedures to increase susceptibility of coronary atherosclerosis. Personal attributes like high serum cholesterol, high blood pressure, hypertriglyceridemia and obesity were found to increase frequency of disease. Personal habits such as Type A behaviour, cigarette smoking, lack of exercise and dietary habits were also considered as risk factors for coronary artery disease.

It is believed that diet and lifestyle has a significant role in the development of atherosclerosis. Lipid is closely related to atherogenesis. Subject with high cholesterol levels are at greater risk of developing coronary artery disease. Low density lipoprotein in high amount causes myocardial infarction or angina. On the other hand HDL level is positively associated with exercise, on moderate ingestion of alcohol and inversely related to smoking and obesity. Atherosclerosis of coronary arteries and larger blood vessels results from lipid deposition.
Before 1950, saturated fat content in the diet of people of U S A were very high. Great progress against specific aspects of coronary artery disease has been made over past 40 years. For example in 1950 the United States age adjusted mortality rate from myocardial infarction was 226.4/1,00,000 people by 1987 the rate had dropped to 124.1 per 1,00,000. These decline was related to life style changes, specifically to serum cholesterol and cigarette smoking. Since the 1950’s Americans have been eating less saturated fat and red meat and fewer dairy products while eating more polyunsaturated fats.

Compared to U S population, intake of dietary fat were low in Indians. Percentage of smoking were also lower. The traditional Indian diet contain 10 - 20% Kilocalorie/day energy from fat including 3 - 5 % kilocalorie /day from saturated fat in rural area, which do not require any advice for saturated fat intake and cholesterol. But the prevalence of CAD increased from 1% in 1960 to 9.6%.
A better understanding of the process of coronary atherosclerosis and etiological factors led to several primary and secondary prevention studies.

A number of secondary prevention studies have provided strong evidence that reduction in the serum cholesterol level would actually decrease the risk. In USA, 24% CAD reduction were attained by smoking cessation. Regular physical exercise and low fat diet may retard progression of CAD and improved myocardial perfusion. Some studies conducted in India proved that modification of diet and lifestyle reduced the incidence of CAD.

This study is being taken up because objectively assessed studies relating the effect of a modified diet and lifestyle and coronary artery disease are limited especially in third world countries.
1. To know how the local diet and life style is related to coronary artery disease.

2. To know whether a modified diet and life style decreases coronary artery disease.

3. To know whether optimum physical activity modifies coronary artery disease.

4. To find the role of coconut oil in causing atherosclerosis
Chapter I - II

Objectives
Chapter I - III

Review of Literature
Coronary heart disease is a major health problem in many countries including India. In spite of tremendous amount of research work going on a global scale, there are controversies regarding basic cause of Ischemic heart disease.

Large scale epidemiological studies have demonstrated that coronary artery disease and its complications were associated with a variety of risk factors. The Framingham study in the United States was one of the first to describe the primary and secondary risk factors for coronary artery disease. This project started in 1948. At the outset 5,209 men and women living in Framingham entered in the study. Individuals have been examined every 2 years and have been evaluated for different factors that might be related to the subsequent development of disease. (Kannel, Gordon 1978).

Over the years data emerging from this project have demonstrated that age, male sex, elevated blood cholesterol level, high blood pressure, cigarette smoking, obesity, diabetes mellitus were characteristics that increases the probability for heart attack and stroke.
Studies conducted after this have viewed the above factors as risk factors and conducted to reveal the relation of one or more particular risk factors to CAD. There have been many studies which have given the evidences about the relation between diet and lifestyle. (Shekelle 1976).

The diet has a great role in the development of coronary heart disease. In 1909 Ignatowski found that dietary proteins can induce experimental atherosclerosis. The first linking relationship between diet and heart disease came by the observation that persons who suffered heart attack almost always had above normal level of blood lipoproteins.

In 1957 Irvine and Page summarised the evidence on the relation between diet and atherosclerosis. This study concluded that (1) diet was important in the pathogenesis of atherosclerosis. (2) Fat and total caloric content of diet were dominant contributing factor. (3) The type of fat or the balance between saturated and unsaturated fats may also be important (Page 1957).
Another international study after Framingham study was the U.S. Western Electric study consisting of 1900 men aged 40 to 55 years, who were employed at Western Electric company, in Chicago in 1957. This study concluded the dietary relation to coronary artery disease. Dietary information was obtained by interviews and questionnaires by nutritionist at baseline and after one year. Serum cholesterol concentration was positively associated with dietary saturated fatty acids and cholesterol and inversely associated with polyunsaturated fatty acids. The risk of death from CAD during 19 years of study was positively related to the baseline levels of dietary scores. It was inversely related to intake of polyunsaturated fatty acids and positively related to the intake of dietary cholesterol. Saturated fatty acid in the diet was not significantly associated with coronary heart disease (Shekelle 1976).

The Zutphen study started in 1960, investigated relationship between diet and other risk characteristics such as serum cholesterol, hypertension, smoking and anthropometric measures and incidence of CAD. This study group consisted of 871 men aged 40 - 59 years living in Zutphen, in the Netherlands. Dietary intake was collected using a
cross check dietary history method. After 10 years 37 men had died from CHD, 30 of these men were free of CHD at the onset of study. Those men who were initially free of disease at the onset of study, but died from CHD during the study consumed 273 Kcal/day less than survivors. (Kromhout 1984).

The Honolulu heart study was a prospective study of cardiovascular disease in men of Japanese ancestry who resided in the island of Oahu in 1965. More than 8,000 men undergone complete physical examination and dietary survey using 24 hour recall method. Men who developed CHD had lower mean intake from carbohydrates, starch and vegetable proteins and had higher caloric intake from proteins, fats, saturated fatty acids and polyunsaturated fatty acids. This group had significantly lower mean percentage of calories from carbohydrates and higher mean ingestion of cholesterol per 1000 calories (McGee 1984).

The Puerto Rico Heart Health Program was a long term prospective epidemiologic study started in 1965 to investigate the relationship of CHD risk factors to coronary heart disease in urban and rural area. Approximately 8000 men aged 45 to 64 years were involved.
in this study. Puerto Rican had previously been noted to have roughly one half of the incidence of CHD, as those in the Continental United States. Dietary cholesterol intake averaged less than 450mg/day in urban dwellers and 330mg/day for those living in rural areas. Both urban and rural men who developed myocardial infarction or CHD had lower average caloric intake and lower intake of total carbohydrate. Dietary carbohydrate and starch intake was inversely related to baseline serum cholesterol in urban men and to a lesser degree in rural cohort. Particularly carbohydrate intake from legumes was inversely related to CHD incidence especially for urban residents. (Garcia Palmieri 1980)

The seven country study was a prospective study using 16 Cohorts of men aged 40-59 yrs who lived in seven different countries. Dietary analysis were done. After 15 years approximately 20% of men who were healthy at entry died. Death rate was related to proteins in the diet, but was believed to the rate of monounsaturated fatty acid to saturated fatty acid in the diet. Incidence of CHD and death rate were low in cohorts with olive oil as main fat source in their diet. (Key 1979) This study revealed that unsaturated fat does not causes heart disease.
The Ireland - Boston Diet Heart study was a prospective epidemiologic study of 1001 middle aged men of 30–69. (Kushi and Lew 1985). Boston brothers cohort was found to have the highest mortality rate for CHD compared with other cohort, although difference was not statistically significant. The Boston brothers had the highest dietary cholesterol levels, the highest dietary lipid score, animal food score with lowest vegetable protein intake and total carbohydrate intake.

The Belgium Army study and Belgium Inter-university research on nutrition and health study confirmed the relationship between butter consumption, serum cholesterol and CHD mortality (Kesteloot - 1984).

Ni - HO - San study showed inter-population relationship of CHD. Positive coefficients were noted for dietary cholesterol, saturated fat and animal protein as well as for percentage of calories from total protein, animal protein, total fat and saturated fat. Caloric intake, body weight, complex carbohydrate intake and total carbohydrate intake exhibited a negative correlation with serum cholesterol and incidence of CHD (Kutto Tillotson 1975).
Among the various risk factors including behaviour or traits or lifestyle factors, that increase the risk of coronary heart disease (CHD), diet appears to have a central risk (Helen Gutrie 1989). Four dietary factors raising the danger for development of coronary artery disease were dietary cholesterol, cholesterol raising fatty acid, excess caloric intake leading to obesity, increased salt intake, that further rise the blood pressure. (Grundy and Friedman 1995)

The potential importance of dietary cholesterol in the causation of atherosclerosis was realised many years ago from cholesterol feeding experiments in laboratory animals. Virchow in 19th century identified cholesterol crystals in atherosclerotic lesion. In a series of studies a definite effect of dietary cholesterol on plasma cholesterol level was observed, for example Framingham study and Western Electric study. In these studies dietary cholesterol showed a positive relationship with incidence of coronary heart disease.
Since plasma cholesterol correlates significantly with incidence of atherosclerosis and CHD, the relationship of dietary cholesterol to death rate of CHD has also been described. (Conner 1979)

A significant positive association exists between saturated fat and coronary artery disease. (McGee 1984). Higher dietary intake of saturated fat and transunsaturated fat was associated with an increased risk of coronary disease whereas higher intake of monounsaturated and polyunsaturated fat was associated with a decreased risk (Frank et al 1997). In 1995 an expert panel assembled by the International Life-Science Institute extensively reviewed the literature on transfats and concluded that they raised cholesterol, when substituted for polyunsaturated or cismonounsaturated fat. In that same year European Antioxidant Myocardial Infarction and Cancer study, a large case control study in Europe showed no overall association between levels of transfat in adipose tissue and biopsy specimen and incidence of myocardial infarction. (Aso et al 1995) Diets high in saturated fat and cholesterol and low in polyunsaturated fatty acid increase the risk of coronary heart disease. The diets that increases the blood cholesterol concentration was related to risk of coronary disease.
A study conducted in human beings compared the association between intake of saturated fat and risk of coronary heart disease. This study started in 1986 and 51529 health professionals aged 40 - 75 participated. The analysis was based on the hypothesis that saturated fat and cholesterol intake affect the risk of coronary heart disease. This study also supported a specific preventive effect of linoleic acid intake. (Alberto Ascheria Eric Rimm 1996) There were evidences that hypercholesterolemia can inhibit secretion of endothelial dependent relaxant factor and predisposes the blood vessels to thrombosis and atherosclerosis. Ahrens clearly demonstrated hypercholesterolemic effect of saturated fat in man (Ahrens 1937)

Dietary pattern can cause atherosclerosis was established by many experimental studies conducted in laboratory animals. Meeker and Kesten compared the effect of animal protein and plant protein. One group of rabbits were given casein in their diet and another group with plant protein. They found former to be more atherogenic. (Meker and Kesten - 1940)
There were some studies in human volunteers also. A diet containing mixed protein was hyperlipidemic for human volunteers than one containing only vegetable protein. (Hodges1967) This study was in agreement with studies conducted by Meeker and Kesten in 1940. In another intervention trial subjects in one group with type II hyperlipoproteinemia, were given a diet rich in soya protein and another group with a similar diet containing meat. It was found that soya protein were hyperlipidemic. (Sister1977).

The amount of carbohydrate ingested has a role in the development of coronary heart disease. For understanding this, at first, we have to think about metabolism of carbohydrates. Excess glucose is converted into glycogen and after liver glycogen concentration reaches to 6%, additional glucose is converted into acetyl CoA which is converted into fatty acids and then synthesised within liver itself and used to form triglycerides. (Guyton1995) Influence of dietary carbohydrate on blood lipids depends not only on type of carbohydrate and its amount ingested but also on whether the subjects were normal, diabetic or hyperlipidemic. Increased intake of carbohydrates to 55% and 10% of total calorie intake
per day may be associated with hypertriglyceridemia. (Den Besten - 1973)

Earlier studies showed that an increase in dietary carbohydrate causes a rise in fasting triglyceride which was significant only in persons with abnormal lipid levels (Mahley 1976)

Yudkin in his studies revealed that sucrose consumption was central to etiology of CHD. (Yudkin 1966) A study conducted in rabbits by feeding different types of carbohydrate such as fructose, sucrose, starch, glucose and lactose for 10 months for showing atherogenic effect of carbohydrate. Fructose fed rabbits exhibited high serum cholesterol and liver cholesterol level and also high level of atherosclerosis. Lactose fed rabbits exhibited low serum and liver cholesterol level and low level of atherosclerosis. (Kritchevsky 1968) Lactose has no significant effect in baboons fed a semi-purified cholesterol free diet but adding 0.1% Cholesterol to diet renders it very atherogenic. (Kritchevsky 1980)

A study conducted to reveal the effect of dietary cholesterol and fatty acids on HDL and LDL in 20 young men. This study concluded that both the cholesterol contents and the P/S ratios of diets were important in
determining LDL levels. The lipid and apoprotein compositions, molecular weights, and binding by cellular receptors of LDL were virtually unchanged by the addition of cholesterol to the diets high in saturated fat. These diets caused an increase in the number of LDL particles of virtually unchanged physical and biological properties. On the diet with low P/S ratio, HDL-2 rose, whereas this effect was absent on diets with high P/S ratio. (Gustav Schonfeld 1982).

High carbohydrate diet intake for 4 days has been found to cause striking changes in VLDL composition and in distribution of apo-C protein in normal subjects. (Schonfeld 1976) Another study showed that a lacto-vegetarian diet can decrease blood lipids and blood pressures with lack of decrease in HDL cholesterol (Kestinon 1989).

Hypercholesterolemia which can lead to atherosclerosis was usually pointed out as an important predisposing factor for cardiovascular diseases. Research in the field of human lipid metabolism was strongly motivated by the relationship between blood lipoprotein and pathogenesis of atherosclerosis which may later lead on coronary heart disease. The critical role of blood cholesterol in the pathogenesis of CAD was well
established. Framingham study proved that total cholesterol to be reliable predictor of CHD under 50 years. Serum lipoprotein proved to be the best predictor of heart disease in elders. Pioneering lipoprotein studies had been conducted at the Donner Laboratory at the University of California under the direction of John Gofman. Gofman’s group separated the lipoprotein fraction as chylomicrons, VLDL, LDL, and HDL. (John Gofman 1949)

Report of Adult Treatment Panel II (1994) reaffirmed that increased blood cholesterol level especially high LDL cholesterol increases risk of CHD, while lowering total cholesterol and LDL cholesterol reduces CHD risk. LDL was the major risk factor. Oxidative modification of LDL alters both lipid and protein components of lipoprotein. Increased uptake of modified LDL within arterial wall can cause accumulation of cholesteryl ester leading to foam cell formation and development of fatty streak. (Mori et al 1996) The effect of dietary cholesterol may also be modulated by fatty acid content and composition of diet. (Schonfeld 1982) Alteration in LDL contribute to the development of atherosclerotic plaque. Chemical modification of LDL particles by oxidation, glycosylation makes LDL ligand for an alternative scavenger receptor
pathway found on endothelial cells and macrophages. Small dense LDL are more atherogenic than normal sized LDL (Austin 1988)

On the other hand, High Density Lipoprotein was smallest of lipoprotein particles. They exert an inverse relationship with atherosclerosis (Miller 1975, Gordon 1977, Gold bast 1985). HDL particles are classified in several ways and commonly known classification is HDL 3 and HDL 2. Jayakumari et al. in 1993 found that low level of HDL-c is a characteristic feature of patients with coronary artery disease.

Framingham study proved the protective effect of HDL against CHD (Gorden, Cestil et al. 1977). A study conducted by Lipid Research Clinic in 1984 also established that HDL has an inverse relation to CHD. European society of Atherosclerosis and Hypertension in 1987 established that elevated levels of Total and LDL cholesterol and low levels of HDL were important risk factors for CHD. Total cholesterol is taken away from cells by the HDL and delivered to liver cells for catabolism to bile acids resulting into loss from the body. A study
conducted by Chen and Anderson in 1979 found that plant fibre may increase HDL-C concentration.

National Cholesterol Education Programme, Adult Treatment Panel II has defined an HDL cholesterol value < 35 mg/dl as a major and independent risk factor of CHD and a value > 60 mg/dl as being protective against CAD (NCEP 1994). In the Norwegian study 11% decline in coronary mortality and 0.6% decline in cardiovascular mortality was found with 1 mg/dl increased in HDL cholesterol in men without a cardiovascular history (Yudkin 1966)

Many epidemiologic surveys document a relation between serum total triglyceride levels and CHD rates. In most case control and prospective studies the level of triglycerides correlates positively with rates of CHD by univariate analysis. (Davis 1990) A case control study conducted by Albrink and Man showed that fasting triglyceride level were increased among patients with coronary artery disease, compared with control subjects. (Albrink and Man 1959) An epidemiological study conducted by Halley established the positive association between triglyceride and coronary artery disease. (Halley 1980) But triglyceride is not an
independent risk factor because in the multivariate analysis when level of total cholesterol and HDL cholesterol were taken into account, triglyceride may lose their statistical power to predict CHD rate. (Grundy and Friedman 1995)

Beyond dietary factors, lifestyle factors plays an important role in the development of CAD. In 1960 the evidences relating cigarette smoking to various diseases including cardiovascular disease were arise. This led the United States Surgeon General to urge the American public to reduce their smoking. Analysis of relation between cigarette smoking and serum lipid showed that cigarette smoking may be associated with roughly a rise in serum cholesterol. (Crag 1989) Another study revealed that smoking may decrease HDL cholesterol and endothelial dependent relaxant factor and increases other serum lipids. (Parrot 1988) Smoking may be associated with a rise in concentration of several hormones including increased release of nor-epinephrine and dopamine from brain tissue. The incidence of myocardial infarction and mortality from heart disease increases progressively with extent of smoking. (Rosenbery 1985) Subjects who smoke more than 25 cigarettes a day have lower levels of HDL and higher levels of VLDL cholesterol than non smokers, ex-
smokers and those who smoke less than 25 cigarettes/day (Brischetto 1980) Exposure of non smoking subjects to second hand smoke breaks serum antioxidant defence leading to accelerated lipid production, LDL modification and accumulation of LDL cholesterol in human macrophages (Mila Valkonen 1998) These data also provided patho-physiological background for recent epidemiological evidence about increased CHD risk among passive smokers. Several studies have demonstrated decreased levels of serum ascorbic acid in smokers, hyperlipidemia and in acute MI (Dawnuy 1990).

Rabbits which had been given a high lipid diet, when exposed to tobacco smoke accelerated the development of atherosclerosis (Zhu 1993) In another study lipid fed rabbits who were exposed to tobacco smoke expressed the adverse effects of tobacco smoke on endothelial function. (Sun et al1994)

Carbon monoxide levels increased in smokers and those exposed to tobacco smoke. This reduces the oxygen carrying capacity of blood and this may have adverse effect on exercise tolerance in patients with ischemic heart disease (Dawyer 1985) If the smokers discontinue their...
smoking, the risk of subsequent myocardial Infarction was lowered, until it approaches that of a non-smokers after two years. (Kannel 1978)) In Western countries tobacco was mostly used in the form of cigarettes, while in India 50% of tobacco was consumed in smokeless form such as chewing, snuff etc and in smoking also only 10% was sold as cigarettes while the rest of 40% was sold as beedies. (Uton 1994)

There were evidences about increased CHD mortality in communities with excess intake of alcohol. Alcohol consumption in high amounts may be associated with modification of pattern of fatty acid in liver and blood lipids. (Lieberies 1984). When alcohol is available in blood it becomes primary fuel for liver leading to accumulation of fat and lipogenesis. There was an increased synthesis of triglycerides and lipoproteins in liver. Increased consumption of alcohol causes a decrease in linoleic acid content of liver and blood. (Allinge et al 1979) Further evidence showed that low linoleic acid content in alcoholics may be related to increased incidence of CHD (Kittner 1983).

There were significant evidences showing lowest CHD mortality in moderate drinkers and highest mortality in nonalcoholic and heavy
drinkers (Marmot 1984). Alcohol enhances the concentration of HDL in the blood which provides protection due to increased fibrinolytic activity or inhibition of platelet aggregation. (Clark 1984) In the Framingham study, Honolulu, Chicago and Yugoslavian studies, person who drank less than 2 drinks/day or greater than one drink/day showed a protective effect of alcohol against CHD, but had more incidents of stroke, accidents and cancer (Gordon 1986). Cabot in 1904 first observed a low incidence of atherosclerotic lesions in autopsies from patients with a history of alcoholism. (More 1986)

Hypertension was a major modifiable risk factor for the development of CHD. High blood pressure increases the risk of development of cardiovascular disease. (Kannel 1974) Hypertension was frequently associated with other risk factors such as abnormalities of lipoproteins and hyperinsulinemia. (Stamler et al 1985) Systolic hypertension was a recognised risk factor in cardiovascular and cerebrovascular disease. (Samulson - 1987) Since atherosclerotic lesions seldom occur in low pressure segments of circulation such as pulmonary artery and veins blood pressure was a critical factor in atherogenesis. There was linear rise in subsequent mortality with increasing level of both systolic and
diastolic pressure. (Samullson 1986) Hypertension, one of the most important risk factor of CHD, may be a response of our body which manifests due to lack of adaptation against increased intake of calories, obesity, physical inactivity, increased dietary sodium, alcoholism, sugar, saturated fatty acid, cholesterol, stress, smoking. (Alan Chait et al 1993)

Build and Blood Pressure study published in 1959 reported that overweight may be a risk factor of cardiovascular disease. The relationship between obesity and plasma lipids in normal subjects has been demonstrated in several studies. A positive correlation has been shown between relative body weight and plasma concentration of cholesterol, triglycerides, phospholipids and beta lipoproteins. (Rifkind and Begg 1966) In the Framingham study obesity was a significant risk predictor for CHD especially among females. (Kanell 1974).

Rapid changes in diet and lifestyle due to urbanisation have been associated with obesity. The relationship between obesity and plasma lipids in normal subjects has been demonstrated in several studies. Jajoo in 1988 showed that prevalence coronary artery disease was much lower in subjects taking physical exercise than those in those who were largely
sedentary. A study conducted in obese females showed that obesity was found to be associated with changes in lipoproteins, especially with VLDL and LDL. In obese women cholesterol in VLDL was found to be higher in relation to that in LDL. (Gandhi 1987) A study conducted by World Health Organisation found that body fat is deposited in the trunk and abdomen (WHO 1990) Recent studies indicated that total adiposity as well as body fat distribution can contribute an independent relation to the risk of insulin resistance which may be associated with diabetes, hypertension and coronary artery disease (Sing et al 1996). Indians Women’s Health Study showed that body mass index, sedentary lifestyle and family history of excess intake of fat were significant risk factors for central obesity. (Saraswathy Ghosh 1998)

Seven countries study consisting of 15 cohorts of Southern European Men between 40 to 59 years showed no significant relationship between obesity and coronary death rate. The strongest evidence for the relationship between obesity and atherosclerosis came from patients who were extremely obese, for example more than 30% above normal body weight (Key et al 1984) In several studies upper body obesity has been found to have a more consistent relation with CHD than total degree of
obesity, while body mass index and skin-fold thickness showed no relation in a 13 year study in Sweden. (Larson et al 1984). A recent study showed that obese people with upper body obesity behave differently, metabolically as reflected by their glucose tolerance, insulin resistance, lipids and apo-lipoprotein (Van Gal - 1989).

Reports of several published studies showed the relationship between depression, anxiety and neuroticism and condition of CHD. These studies provided strong evidence that emotional distress precedes the development of symptoms of CHD. Subjects with high socio-economic status and education showed greater modification of risk factors such as lifestyle changes like heavy smoking and frequent alcohol consumption. Men with little or no physical activity in leisure time were also associated with higher prevalence of mental distress which in turn showed association with current lifestyle. (Mori et al 1996)

Type A behaviour is characterised by competitiveness, aggressiveness, especially expressed anger, ambition and sense of time urgency. Type A behaviour had been studied in great detail by Friedman. He found that Type A behaviour had a role in the
pathogenesis of coronary arteries. (Friedman 1969) In a study comprising of 592 patients with myocardial infarction, there was a significant reduction in type A behaviour in 44% of experimental subjects after 3 years associated with significant decrease in re-infarction compared to control group. (Johnston 1980)

Western Collaborative study showed that Type A men had 22 times higher prevalence than Type B men (Roseman 1964). A study conducted in India showed that the patients experienced higher number of stressful life events in the year before their infarction as compared to controls. This study concluded that type A behaviour and stressful life events were significantly associated with risk of myocardial infarction. (Bhatia et al 1990) A prospective study of worry and coronary heart disease in Normative Aging study suggested that high levels of worry in specific domains may increase the risk of CHD in old men. (Laruen 1997) Recent research has suggested that anxiety was related to incidence of CHD (Kawachi 1997). The relationship of Type A personality and CAD has been evaluated in several large studies including Framingham study. In response to repeated episodes of emotion and stress, adrenaline is repeatedly liberated from adrenal.
medulla which mobilises free fatty acids from adipose tissue and causes fat deposits in the wall of blood vessels. (Guyton 1995.)

The Boston Collaborative Drug Surveillance Programme showed that intake of greater than 6 cups of coffee per day was positively associated with a greater than 2 fold increase in risk of myocardial infarction (BCDS 1971). In Framingham study a correlation was found with an increase in risk of coffee intake in relation to death from all causes although CHD showed no relation with coffee consumption. (Dawber 1974) A prospective study on the effect of coffee consumption and CAD showed that drinking 5 or more cups of coffee/day increased the risk of CHD including sudden cardiac death, about 2 - 3 fold than non coffee drinkers. (Lacroix 1986).

One experimental study in Japan showed that addition of caffeine or theobromine to diet cause hypercholesterolemia. Coffee intake in association with high cholesterol diet enhanced the serum levels of cholesterol phospholipid and triglycerids whereas in association with
cholesterol free diet, intake of coffee caused a decrease in plasma cholesterol, phospholipid and triglycerides (Wessner 1982)

A study conducted by Trough showed that Vitamin C may be important in controlling cardiovascular disease (Trough 1991). The Vit.E has a great role in cardiovascular disease. This was first described by Vogetsany in 1946. A study conducted by Rimerson showed that plasma concentration of vitamin C, E and carotene were significantly inversely related to risk of angina. (Reimerson 1991).

Dietary Vitamin E administration was found to decrease susceptibility of LDL isolated from hypercholesterolemic men to oxidative modification. LDL isolated from a single subject, after vitamin E supplementation had a higher level in lipoproteins and was protected against macrophage mediated oxidation compared to LDL, before administration of dietary vitamin E. (Jessup 1990) Dietary supplementation with vitamin C and E causes resistance of lipoprotein to oxidation by more complex mechanism than simply increasing lipoprotein associated vitamin E. (Vincent and Avedis 1993)
In a recent study, male smokers who received Vitamin E supplements experienced lower mortality from IHD, while betacarotene supplementation was associated with more ischemic heart disease death. (AlphaTocopherol Beta-carotene Cancer prevention group:1994) The CARDIA study concluded that dietary antioxidant was associated with HDL -C in some subsets of population, although these associations may be operating in conjunction with other lifestyle behaviour. (Martha and Slattery 1995) A report in Medical Times 1995, revealed that antioxidants should be taken in their natural taste. (green, yellow and orange fruits and vegetables.) A study conducted in women to determine the effect of ascorbic acid supplement on plasma lipid and lipoproteins showed that increase in ascorbic acid intake favourably alter lipoprotein profile in young women. (Liss 1996) A recently conducted study among the coronary artery diseased patients found that long term ascorbic acid treatment reverses the endothelial vasomotor dysfunction. (Noyan Gokce 1999)
Diabetes can be considered as a risk factor for CAD. Experimental studies showed that insulin may have a direct effect on arterial wall leading to atherosclerosis (Duff et al 1949). Diabetes is a strong and independent risk factor for the presence of coronary artery disease, increasing the risk, particularly in women, between two fold to four fold. (Kannel 1974) In patients with diabetes, coronary disease is more extensive and severe. Cardiovascular death is 3 times more common in diabetes, than in non diabetic patients for the MRFIT. Most studies of coronary artery surgery have shown that risk of death, myocardial infarction and stroke are increased in patients with diabetes than with non diabetic patients. (Herlitz et al 1997) In another study it was revealed that an increased incidence of myocardial infarction resulted from increase in fasting level of plasma insulin (Sorge 1995)

Positive Family History is considered as an independent risk factor. (Sinaiko-1990) The family at high risk for the development of CAD usually has at least one member who has hyperlipidemia, low HDL, hypertension, a positive family history of coronary disease, or a positive family history premature CAD. A study by Becker and associates, examined the siblings free of clinically evident coronary disease by
questionnaire and interview. This wide-scale screening revealed that 48% of brothers 22% of sisters had a lipid abnormality. (Becker 1988) Another study established a Mendelian dominant inheritance for the atherogenic lipoprotein pattern associated with small dense LDL, VLDL, IDL, and small dense LDL were increased while HDL and HDL2 were decreased. (Austin 1990) It should be especially important to reduce all appropriate risk factors in individuals who have a strong family history of CHD.

CHD has been considered to affect people of high socio-economic status. Epidemiologic studies conducted in Agra, Delhi, Coonor have suggested that about 6% of high income people had CHD while less than 1% of low income group had the disease. (Padmavathy 1959) A report from Amritsar suggested that 52% of CHD patients belonged to middle income group, whereas 34% was from upper income group and 14% from low income category. (Haughst 1990) Study conducted in Trivandrum district showed that this disease was affecting in a similar fashion in both high and low income group. (Vardhan et al 1990)

In a study it was found that morbidity and mortality in patients without risk factors was found to be lower compared with that found in
snorers with risk factors. This study concluded that snoring worsened prognosis of patients with risk factors for cardiovascular disease, but did not represent an independent or predictive risk factor in itself. (Augusto Zanirelli 1991)

Total levels of plasma homocysteine were associated with a high incidence of coronary disease in adolescents. (Veland 1992) Several studies have shown a correlation between fibrinogen concentration and risk of developing Ischemic heart disease. There was a strong suggestion that platelet activation also plays an important role in the development of acute events in patients with underlying CHD. (Yarnell 1991) Elevated plasma levels of homocysteine was often associated with low plasma levels of folate and pyridoxal phosphate, a form of vitamin B6. (Ubbink 1993) Elevated homocysteine levels can be lowered or normalised by supplementation with vitamin B6 and Folate. (Selhub et al 1995) In a prospective study conducted to reveal the role of Folate and Vitamin B6 among U.S. Physicians revealed that low dietary intake of folate or vitamin B6 contribute to risk of M.I. The highly significant inverse correlations exist between folate, vitamin B6 and level of homocysteine. (Lisa 1996)
Studies have shown that elevated lipoprotein(a) \([\text{Lp(a)}]\) concentration were associated with IHD. (Schaefer 1994, Sigurdsson 1992) A study conducted to reveal the independent role of \(\text{Lp(a)}\) in the causation of CAD showed that it was not an independent risk factor, but appeared to increase the risk associated with other lipid risk factors. (Bernad Centin1998) But results of another study indicate that elevated plasma \(\text{Lp(a)}\) is an independent risk factor for angiographic CAD in chronic stable angina and may have particular significance in women. (Paul et al 1998)

Hypercalcemia was another risk factor. Calcium overload in the arterial wall may play a role in the pathogenesis of atherosclerosis. Recent studies using Ultrasound computed tomographic scanning have pointed out that presence of increased coronary calcification puts individuals at higher risk for developing CHD. Studies already showed that extent and severity of coronary calcification appear to provide information through the eventual development of coronary disease. (Fallavovitta 1994) Coronary artery calcification should not necessarily be viewed as an additional risk factor apart from other risk factors, since all
the other traditional risk factors presumably contribute to premature calcification.

In nonhuman primates, vasectomy increased the severity of diet induced atherosclerosis. (Alexander 1978) Several studies have shown a statistical association between elevated levels of uric acid and hypertriglyceredemia. The Framingham study found that the concentration of uric acid also correlates with systolic and diastolic blood pressure values. (Brand 1985)

Atherosclerosis was known as a progressive disease of aging, and accordingly age plays an important role in the development of CHD. (Balarajan 1991) In India CHD occurs in people of relatively younger age group. A report from Madras showed that about 10% of patient undergoing coronary angiography for angina pectoris were 27 to 40 years old with a mean age of 38 years. (Jayachandran 1987) The deficiency of copper or excess of zinc, predisposes to secondary hypercholesterolemia. (Klevay 1984)
An analysis of monthly death certificate data from the Los Angeles showed that there were seasonal variations in the development of coronary artery death, with more deaths occurring in December and January than in June to September. Monthly cardiac death correlated inversely with monthly temperature. Other factors including overindulging or the stress related to holiday season could be important. (Kloner et al 1999)

The risk factors stated above were very few compared to existing risk factors. CHD risk factors were more than additive in their joint efforts. A male smoker who has a total cholesterol of 240 mg or more and diastolic blood pressure over 90 mm Hg was at 14 times higher risk over 6 year period of attack, than a male non-smoker with lower cholesterol and blood pressure. (Medical Times 1995) The risk factors identified till now have reached 246 ranging from dietary to demographic. (Hopkins 1981) Among the various risk factors some are prevalent in some areas. The population surveys and risk factor analysis conducted in India reveal the prevalence of CAD is increasing in India.

In India intake of dietary fat was lower compared to Western population. Occurrence of other risk factors like smoking and alcoholism...
were also lower. But the prevalence of CAD was increasing day by day. A few population studies were reported from India in the 1960s. A study conducted among the general population of Agra in the age group of 25 years or more. It was a field survey of 1046 individuals from different parts of Agra. Eleven person's were identified as having CHD (1.05%) (Marthur1960). A study conducted by Padmavathi in 1962 in adults over 20 year of age in the general population of Delhi showed a prevalence rate of coronary artery disease of 5.5/1000 in the high income group and 3.3/1000 in the low income group. (Padmavathi 1962).

A population survey reported from urban population of Chandigarh identified that prevalence of probable and possible CAD was 66/1000, among 1331 persons. (Sarvotham and Berry 1968) Another population survey conducted in Hariyana compared the prevalence of CHD in rural and urban community in Rohtak (Hariyana) and its adjoining areas. The number of people examined in the rural and urban setting was 1,504 and 750 respectively with a CHD prevalence rate of 4.53/1000 and 17.2/1000 respectively. (Gupta and Malhotra 1975)
The above available data represents low prevalence of coronary heart disease in Indian population. But surveys conducted in 1990s showed higher prevalence of CHD. A well designed study with a sample size of 13,723 men and women aged between 25 - 64 years was reported from urban areas of Delhi. The overall prevalence of CHD using both clinical and ECG data was 96.7/1000, the prevalence was estimated as 31.9/1000 (39.5/1000 in men and 25.3/1000 in women). Electrocardiographic evidence of CHD in asymptomatic males was 56.7/1000 and in females was 75.1/1000. (Chaddha et al 1990).

A study conducted in urban people of Jaipur district. The sample included 2,212 persons aged 20 years or more. (men 1,415 and women 797) CHD was diagnosed by ECG changes. The overall prevalence was 76/1000 (men 60/1000 and women 104/1000). ECG evidence of CHD was exhibited more by women (84/1000) than men (35/1000) (Gupta et al 1995).

Prevalence of CHD in the rural population of Thiruvananthapuram district in Kerala was studied in 1253 villages. Age of the people was more than 25 yrs. The estimated prevalence was 75/1000 persons with
probable CHD. On the other hand definitive CHD prevalence was only 14/1000. The study reported that prevalence of CHD in this urbanised rural area was 7/1000 or less. They also suggested that women had a higher or equal prevalence of CHD as men at ages 25 - 54 years (408 men/487 women) (Ramankutty et al 1993).

The coronary risk factors in the Indian subjects has been revealed by many studies. A community based survey of CHD carried out on random urban sample of 13,723 adults aged 25 - 46 years in Delhi showed hypertension had the strongest association with CHD. Chadha et al identified smoking, obesity, hypertension and family history of CHD and diabetes mellitus as major risk factors in descending order of frequency in males. In females obesity was the commonest observed factor followed by hypertension, a family history of CAD, smoking and diabetes mellitus. Risk factors identified from Jaipur were smoking, hypertension, and obesity with a very low prevalence of known diabetes. On the other hand women had more hypertension followed by smoking, obesity and very low level of diabetes mellitus. (Gupta et al 1995).
A study conducted in the 80's to evaluate and compare the total cholesterol, triglyceride and lipoprotein fraction in different age groups of healthy subjects of North India. Total triglyceride and total cholesterol showed marked increase with age. LDL cholesterol increased up to the age of 50 years. There was not much changes in values of VLDL cholesterol and HDL cholesterol. Total cholesterol, LDL cholesterol, HDL cholesterol values were lower in Indians than Western population while total triglycerides and VLDL cholesterol values were higher. (Gandhi 1982)

Prevalence of CAD and coronary risk factors in rural and urban populations of North India showed that prevalence of CAD was 9% and coronary risk factors were two or three times higher among urban subjects as compared with rural subjects. Indian Social class and Heart study examined whether social class is associated with coronary artery disease and coronary risk factors. (Singh et al 1996) This may be due to greater sedentary behaviour and alcohol intake among urban people. It was possible that some percentage of Indian population can benefit by reducing serum cholesterol, blood pressure, central obesity increasing by physical activity. (Singh et al 1998)
Contrary to Western world, lipid profile level was lower in Indians. In Madras 75% of people with M.I. had plasma cholesterol level less than 200mg/dl. (Krishnaswamy 1970) In another study, even lower level of plasma cholesterol (<150mg/dl) in patients with CHD have been reported. (Chaudhuri 1966) Total plasma cholesterol level has been found to be lower among the expatriate Indians living in the UK. (McKeigue 1988) Among the expatriate Indians living in USA the total and low density lipoprotein cholesterol were similar to that of European Americans, while the high density lipoprotein cholesterol was lower in former. (Enas 1992)

Studies conducted in India revealed that coronary artery disease is seen in people with low serum cholesterol levels. In Madras 75% of people with myocardial infarction had plasma cholesterol levels less than 200mg/dl (Krishnaswamy 1970) In another study from India, even lower level of plasma cholesterol levels less than 150mg/dl have been reported in patients with CHD. (Guptha 1985) Study conducted in Kerala also found that subjects having low cholesterol level, have this disease. (Jayakumari et al 1993)
Risk factors identified from Thiruvananthapuram district in Kerala was following. Prevalent risk factors in men were smoking, hypertension, diabetes, and obesity while in women obesity, hypertension, diabetes and smoking (Ramankutty et al 1993).

In Kerala coconut oil was considered as a risk factor, which is the common cooking medium in this area. During 1950’s there arose a controversy about consumption of coconut oil and coconut kernel and development of coronary artery disease. Being a saturated fat, coconut oil was suspected to cause hypercholesterolemia and development of atherosclerosis. But studies conducted in human beings and animals found that consumption of coconut oil does not produces any atherogenic effect. Human beings were supplied with coconut oil, coconut oil + coconut kernel, ground nut oil, ground nut oil + ground nut kernel. The results revealed that coconut oil was more advisable than groundnut oil and moderate consumption of coconut kernel does not produce any undesirable effect. It produced lower serum cholesterol, triglycerides, higher HDL-c and lower LDL-c/HDL. (Rajmohan 1997)
A study conducted by Gopinath and associates found the mean level serum cholesterol was 215 mg/dl in individuals with CHD and 206 mg/dl in CHD free controls. In Rajmohans study mean level of serum cholesterol was 204mg/dl in normal healthy volunteers aged 40-60. A study conducted in SCTIMS - Thiruvananthapuram selected 214 male patients with CAD from cardiology clinics. The patients were divided into 2 groups according to their age. There were also 100 healthy male volunteers. The lipid level was higher in the older age group. Higher levels of lipid profile except HDL was observed in the diseased subjects. In this study it was also confirmed that HDL-C, more specifically its HDL2 was the only parameter, which was independently associated with presence CAD irrespective of age and extent of disease. (Jayakumari et al 1993)

Coronary artery disease can be reversed. This was established by many secondary prevention trials between 1968 and 1976. The age adjusted mortality from CAD in the United States progressively declined by 20% (Braunwald1990) during this period. The Goldman and Cook in 1984 estimated that more than half of that decline was related to lifestyle changes specifically to decrease in serum cholesterol and cigarette
smoking. They attributed as much as 24% of CAD reduction was due to smoking cessation. American have been eating less saturated fat and red meat fewer dairy products while eating more PUFA. Public concern about heart healthy eating has grown stronger with each other. U.S. Anti-hypertension Campaign of 1970’s also cited this as a possible explanation for declining CAD mortality during recent years.

Appropriate prevention and management of CHD requires an integrated approach to reduction of risk factors. These principally include reduction of elevated lipids, control of blood pressure, and cessation of smoking. In addition to this, appropriate exercise, diet and weight reduction were also important. Control of diabetes and stress management was also helpful. Lipoprotein abnormalities, smoking and hypertension were generally considered the most important correctable risk factors while positive family history, male gender and older age are non correctable, but important risk factors. (Werlin 1997)

There were primary and secondary prevention trials which have proved that atherosclerosis can be reversed. Trials over the last several years have demonstrated the benefit of cholesterol lowering in secondary
prevention and producing regression of coronary lesions. Cholesterol lowering effectively induces regression and delays the progression of coronary atherosclerosis. (Levine 1995) Cholesterol lowering may stabilise the vulnerable plaque by modifying its content mostly through a reduction of the oxidised lipids in its core, which renders it susceptible to rapture. (Fuster 1996) Cholesterol reduction also improves coronary, systemic and endothelial function. Another potential benefit of cholesterol reduction could be through an effect on haemostatic factors. (Lacote 1995)

A recent study conducted among patients with mild to moderate hypercholesterolemia, revealed that lipid lowering therapy in early stages of coronary atherosclerosis would prevent further disease progression. (Detlev Baller 1999) In another recent study, it was showed that aggressive lipid lowering delays progression of atherosclerosis in SVG,s irrespective of gender, age and certain other risk factors for CHD. That study also showed that aggressive LDL-c lowering was most beneficial in higher risk patients with HDL-c levels <35mg/dl or triglyceride levels ≥200mg/dl or patients having other associated risk factors. (Campeau et al 1999)
Cholesterol Lowering Atherosclerotic Study was a 27 year random angiographic study, that examined the hypothesis that reduction in LDL and increase in HDL would arrest or reverse development of atherosclerotic lesions in a population that had demonstrated a good compliance in the past. (Blankenhorn 1987). 188 non smoking men aged 40 to 50 years who had a coronary artery bypass grafts were treated with diet and niacin. Marked reductions in total cholesterol by 22%, LDL by 39% and increase in HDL/LDL ratio was observed. This study showed that aggressive measure to reduction of lipids results in less progression and more regression of atherosclerosis. At least 50% of decrease in coronary disease risk has been attributed to an increase in HDL-C and approximately 18% reduction in LDL-C. (Langer 1990)

A screening conducted during primary prevention studies showed that 75% of individuals with elevated cholesterol can be controlled with diet, weight reduction and exercise and 25% will require some form of pharmacotherapy in primary prevention. (Sempson 1993) Familial Atherosclerosis Treatment Study, examined men younger than 62 years with a family history of premature coronary disease, elevated lipid levels.
angiographic coronary artery disease and no history of CABG for 2.5 years. This trial demonstrated that high risk patients treated with aggressive lipid reducing combination therapy, compared with conventional treatment demonstrated a drastic decrease in progression and increase in regression of atherosclerotic lesion. (Brown, Albers and Fisher 1990)

In the Leiden Intervention Trial (1978 - 1982) 39 patients with stable angina pectoris and more than 50% narrowing of at least one coronary artery were administered experimental diet for 2 years. Experimental diet included less than 100mg/day of cholesterol and linoleic acid enriched diet with a P:S ratio of 2. After 2 years there was a significant reduction in body weight, serum total cholesterol, total HDL cholesterol and systolic blood pressure (Arntzenius - 1985).

Lifestyle heart trial was designed to determine whether comprehensive lifestyle characters consisting of low fat vegetarian diet, cigarette smoking cessation, and stress management after progression of coronary atherosclerosis, after 1 year. This trial demonstrated that
aggressive dietary therapy in conjunction with relaxation technique can have a marked impact on atherosclerotic coronary disease. (Ornish 1990)

Several studies have cleared that specific theories aimed at coronary risk factors can decrease CHD death rates (Manninen 1988). Lipid Research Clinics Primary Prevention Trials stated risk of coronary artery disease was lower in people with low fat diet, physical activity and persons without smoking. (Lipid Research clinics programme - 1984) Lipid reducing trial, as well as multifactorial approaches to risk modification, have confirmed that CHD events can be favourably arrested through prevention strategies (Rossouw 1990). In a meta-analysis of primary prevention studies, Law et al concluded that a 10% reduction in LDL cholesterol after 5 years can result up to a 50% decrease in CHD. (Law et al 1990)

The National Heart Lung and Blood Institute (NHLBI) Type II Coronary Intervention study proved that there was a significant fall in serum total cholesterol in the group treated with diet and drug. (Levi 1984)
St. Thomas Atherosclerotic Regression study was a study conducted among British men with coronary heart disease and cholesterol level greater than 232 mg/dl randomised to either conventional case, a low fat diet or diet plus cholestyramine. Cholesterol level decreased by 11% and 20% in the diet and cholestyramine groups compared with usual care group (Watts 1992). In Heidelberg Diet and Exercise study, men with CHD were randomly assigned to a diet with a variable fat content in conjunction with a exercise programme (both group and home exercise). Both groups received a dietary therapy with a cholesterol intake less than 20mg/day. The intervention group exhibited no overall change in either LDL or severity of coronary stenoses. However patients with regression had lower levels of cholesterol after therapy than group showing no change or progression. (Sctiuler 1992) The new coronary biology suggests that cholesterol reduction and vigorous risk factor modification can improve coronary artery endothelial function, impede coronary artery disease progression, after atherosclerotic plaque formation (Grundy and Friedman 1995)
A study conducted in Germany tested the applicability and effects of intensive physical exercise and low fat diet on progression of coronary atherosclerotic lesions and stress induced myocardial ischemia. Eighteen patients in this programme consumed a low fat low cholesterol diet and exercised for less than 1 hr per week. In the Intervention group there was a significant reduction in stress induced Myocardial Ischemia. Regular physical exercise and low fat diet retarded the progression of CAD. There was a significant improvement of myocardial perfusion achieved by regression of stenotic lesions. (Gerhard et al. 1993)

Stanford Coronary Risk Intervention Programme (SCRIP) was designed to determine whether a comprehensive cardiac intervention programme would reduce the risk of CHD. The treatment group consumed a low fat diet and home based aerobic exercise and stopped smoking with goal of lipid lowering, compared to a group receiving usual care. The SCRIP trial demonstrated that Multiple Risk Factor Intervention can cause dietary progression and decrease hospitalisation for cardiac events. (Quinn and Haskel. 1994). This project also demonstrated that multiple risk factor intervention can delay angiographic progression of coronary stenoses and reduce hospitalisation for cardiac events in patients.
with baseline LDL cholesterol level in the borderline high range. (Haskel - 1994).

Another secondary prevention study proved that increased intake of poly unsaturated fatty acid reduced the serum total cholesterol level in dietary intervention group. This in turn reduced CHD events in intervention group than control group. (Leren - 1966)

The Seven Countries Study has shown that, the risk of CHD rises progressively with increase in serum cholesterol levels above 150mg/dl. Serum cholesterol level in the low risk population of India, China and Japan are around 170mg/dl which may be considered desirable serum cholesterol levels for Indians. (WHO study group 1990) This was in agreement with other experts who suggested desirable level of <170mg/dl, borderline high 170-184mg/dl and high if >185 mg/dl. The concerned values for LDL cholesterol was above 90mg/dl, 90-104mg/dl, and above 105mg/dl. The limits for fasting triglycerides was above 140mg/dl and of HDL-C was above 40mg/dl in urban and above 35mg/dl in rural area. (Chadha et al 1994) The upper limit of triglyceride was 150mg/dl. (Singh 1995)
A scientific statement of the International College of Nutrition stated that the limit for total energy from fat intake should be below 21% Kcal/day (7% each from PUFAs, saturated and monounsaturated fatty acids). Carbohydrate intake should be above 65% Kcal/day mainly from complex carbohydrates. Eating 400-600g/day of fruits, vegetables and legumes and mustard or soyabean oil in place of hydrogenated fat, coconut oil or ghee in conjunction with moderate physical activity and mental relaxation as well as cessation of tobacco intakes may be protective against CHD in Indians. (Singh et al 1996)

Certain studies revealed the disadvantage of polyunsaturated fatty acids. PUFA were highly susceptible to free radical attack and hydrogen abstraction reactions generating lipid peroxides and more free radicals which give rise to vicious cycle in which free radical attack on fatty acid side chains and hydrogen abstractions give rise to more free radicals which were able to carry on and magnify the damage. The polyunsaturated fatty acids is a very rich source of omega 6 fatty acids. In addition, omega 6 fatty acids are found freely in most foods. Ghee, mustard oil, coconut oil are traditional cooking media which have been used in India and other
developing countries for thousands of years and they are poor sources of omega 6 fatty acids. Decreased intake of polyunsaturated fatty acids which are potent source of lipid peroxides and free radicals significantly decreases the omega 6 intake. (Sadikot 1996) Anderson has stated that high fibre intake alters absorption rate of fats and sugars. He also stated that fibre intake lowers colonic intraluminal pressure and decreases the residence time of faeces in the colon. (Anderson 1981) There was consistent evidence that common Indian foods such as onion, garlic, guava, goose berry, mushroom, black, red and bengal gram, trichonanthes, bitter gourd, soya beans, groundnut, sunflower oil, almonds, fenugreek seeds and walnuts can modulate concentration of serum lipids and fibrinolytic activity leads to reduction in atherosclerosis (Arntzenius 1985). The clinical effect of diet rich in these local diet may be the same as that of intake of aspirin daily. (Ram Singh et al 1991)

One study in which diet rich in fibre, antioxidant vitamins and minerals given to patients with acute myocardial infarction, significantly reduced cardiovascular events over six weeks. (Singh 1992) Another study proved that comprehensive dietary changes in conjunction with weight loss, immediately after acute myocardial infarction may modulate
blood lipoprotein and significantly reduced complications and mortality after one year. (Singh 1992)

There was evidence that inclusion of high fibre diet such as oats, fruits and vegetables in the diet can decrease fat intake and modulate blood lipids. This was confirmed by a study in which one group of patients were advised to take gauva fruit daily (0.521 Kg/day) while group B served as control. Adding moderate amounts of gauva fruit in the usual diet resulted changes in dietary fatty acids and carbohydrates and it provided, significant amount of soluble dietary fibre and antioxidant vitamins and minerals without any adverse effects. There was greater decrease in lipoprotein metabolism and blood pressure. (Rastogi - 1992). Individualised dietary advice for reducing cholesterol concentration was modestly effective in free living subjects. More intensive diets achieve greater reduction in serum cholesterol level. (Tang et al 1998)

Many common foodstuffs, such as seeds, cereals, berries and some vegetables, contain plant lignan. Lignan has a role in the protection against cancer and coronary artery disease. (Adlercreutz 1997) Mammalian Lignans were produced when plant lignans were modified by
intestinal bacterial flora. (Setchell 1981) The most abundant plant lignan is enterolactone. A recently conducted study among healthy men showed that high serum concentrations of enterolactone had a lower risk of acute coronary events than men with lower concentrations. The findings of that study support the hypothesis that plant dominated fibre rich diet lowers the risk of CHD. (Meri Vancharanta et al 1999)

The protective effect of Mediterranean dietary pattern was and found that this dietary pattern decreases the cardiovascular morbidity and mortality. (Michael 1999) Fish consumption appeared to protect coronary artery disease. Fish oil contain abundant omega - 3 fatty acids such as eicosapentanoic acid and docosahexaenoic acid. (Dyerberg et al 1978) A study conducted by Anne Nafaziger proved that fish oil diets can reduce triglycerides. Fish oil supplements in daily doses of 8 - 15 g reduce triglyceride levels by lowering VLDL production. In addition this study also revealed that exercise lowers triglyceride concentrations, increase insulin sensitivity and improves glucose tolerance. (Anne Nafziger 1995)
Modification of lifestyle along with diet reduces the incidence of coronary artery disease. The Dean Ornish in his book "A Programme for Reversing Heart Disease" has described about stopping of alcoholism, smoking cessation, physical activity and exercise. Moderate exercise and quitting of smoking will reduce the incidence of coronary artery disease. (Dean Ornish 1990) Within 2-3 years of stopping smoking, CHD risk decreases to the level of people who have never smoked. (Uton1994).

Exercise has a wide range of beneficial effects for heart health. It lowers heart rate, blood pressure, level of plasma triglycerides, lowers body weight and increases HDL cholesterol. (Medical Times 1995). The pioneering studies by Morris et al from England and Paffenbarger et al from United States were the first to strongly suggest that increased physical activity either at work or during the leisure time was a deterrent to the development of CHD. In 1953, Morris et al studied transportation workers in London, and found that sedentary bus drivers had a greater incidence of CHD than more active conductors on double decker buses. Later this group showed that vigorous leisure time activity also decreased incidence of CHD. (Morris et al 1990)
In 1970 Paffenbarger et al examined the prevalence of CHD in San Francisco, longshoremen, according to their levels of physical activity. They found that coronary death rate were lower in the middle and high activity groups, than in low activity groups. Paffenbarger et al used a questionnaire to determine activity index of large sample of Harvard University and found a progressive decline in both fatal and nonfatal coronary events with an increasing activity index group upto 2000 Kcal/wk. Further activity had little additional effect on incidence of CAD. (Paffenbarger et al 1978)

In 1992, the American Heart Association published a position statement on exercise that stated “There is a relationship between Physical inactivity and cardiovascular mortality and inactivity is a risk factor for the development coronary artery disease.” In 1995 Centres for Disease Control and Prevention (CDC) and American College of Sports Medicine (ACSM) came to a similar conclusion that increased physical activity had a protective effect against the development of CHD.
Study conducted by Ram Singh established that moderate physical activity in conjunction with dietary changes in patients with coronary artery disease may cause substantial reduction in central obesity and associated disturbances corresponding to a significant decrease in cardiac events and mortality during the follow up of 3 years (Ram Singh 1996) A study conducted among the Asian Indians to know the role of physical exercise, found that serum triglycerides, systolic and diastolic pressure were lower in non sedentary subjects than sedentary subjects. (Dhawan 1997) A study conducted to reveal the effect of regular physical activity and coronary risk factor in Japanese men showed that those who engaged in regular physical activity > 3 days per week had fewer CHD risk factors than sedentary individuals. Those who engaged in physical activity once per week had fewer CHD risk factors than sedentary individuals. (Shijun Dong Hsiech et al 1998).

Finding from the Honolulu Heart Program which targeted physically capable elder men, suggested that an active lifestyle reduces the risk of cardiovascular disease in younger and more diverse groups, this
suggests that important health benefits could be derived by encouraging
the elderly to walk. (Amy Hakkim et al 1999)

Thus epidemiological, clinical, and experimental studies indicate
that coronary artery disease can be reversed. Such a study was not
undertaken in Kerala where the coconut oil is a suspected risk factor.
Hence we conducted this study to know whether coronary artery disease
could be reversed by modifying diet and lifestyle, at the same time
continuing this traditional local cooking medium in our area.
Chapter I - IV

Methodology
234 patients with coronary artery disease were selected from outpatient department of Little Flower Hospital. Their height, weight, blood pressure, heart rate and lipid profile were taken. Dietary pattern was assessed by questionnaire. Habits of smoking, alcoholism and other risk factors were noted by interviewing them. 100 subjects free of CAD were selected for analysing the lipid profile for control group.

After analysing the risk factors in our local area we conducted a “Diet Intervention” trial. For this study, 72 TMT positive patients were recruited. Their height, weight, blood pressure, heart rate and ECG were taken.

Patients with Valvular heart disease, Congenital heart disease, Diabetes, Tuberculosis, and Piles were excluded. (because they cannot follow the modified. diet and life style) Normal blood pressure was considered as 120/80. Essential hypertension was diagnosed if blood pressure was above 140/95mm Hg. Hypercholesterolemia and hypertriglyceridemia were classified based on WHO suggestion.
Consumption of more than 10 cigarettes or beedies per day was considered as heavy smokers. Alcohol intake by consumption of above 80ml or more of alcohol per day and physical inactivity by < 1km/day of walking during daily activities.

Among the 234 subjects, 72 TMT positive patients who were willing to participate for 2 years were recruited for the study. All participants were admitted 7 days not only to assess dietary pattern but also to identify their personal risk factors of CAD. During this time they were closely observed, their dietary pattern was assessed by a questionnaire. They were given a knowledge about the risk factors of coronary artery disease and that disease could be reversed by changing diet and lifestyle. They were advised to follow a modified diet schedule based on Diet Manual. The modified diet included more vegetables and fruits, with moderate amount of rice and pulses. (Patients were allowed to taken 100 grams of fish thrice in a week and 100g of skin removed chicken twice per month.) They were advised to keep away from the possible risk factors like alcohol, smoking. Cooking medium continued was coconut oil. The advises given was walking exercise for 30 minutes at morning and evening.
MODIFIED DIET AND LIFESTYLE

Modified diet :-

Bed coffee : 1 glass tea with skimmed milk.

Break fast : 50gm of cereal + 25 gm of legumes + 250gm of vegetables (like salad or curries).

Lunch : 50g of cereals + 50 g of leafy vegetables + 100g of other vegetables.

4 o' clock : 1 glass of any type of juice + any types of fruits 100gm.

Supper : 2 Chappathees with vegetable curry or equivalent.

(Patients were supplied with a dietary guide describing various choices of food).

If patients like to take non vegetarian food, they were allowed to take 100 g of fish thrice in a week, 100 g of skin removed chicken twice in a month. They were also allowed to take egg white, but not egg yolks.
Modified lifestyle :-

Participants were advised to abandon alcoholism, smoking and beef intake. They were asked to do walking exercise of 30 minutes in the morning and evening. Counselling have been given to reduce their tension and Type A behaviour.

After seven days they were discharged. They were given index card and printed diet schedule. They were asked to visit again after 2 weeks, along with their spouses. blood pressure and heart rate were taken. They were asked whether they could follow the modified diet schedule and could live without cigarettes or stimulants and patients were asked whether they could do the walking exercise.
Fifty subjects could follow the modified diet and lifestyle and they were included in the intervention group. Twenty two subjects were not followed modified diet and lifestyle, but they reduced their fat intake and smoking habits. They were included in the control group.

The participants were given a date after 1 month for checkup. They were asked whether they could follow the changed dietary pattern with more vegetables and fruits. Advises were given about the advantages of modified diet and lifestyle (quitting of smoking and doing walking exercise) and they were attracted by saying about secondary prevention studies conducted to reduce the risk of CAD in world wide.

After this counselling they were given a date after 3 months. Periodic check up were conducted at 3 months interval. Lipid profile values were taken at 8 months interval. Stress ECG were taken after 1 year.
For testing lipid profile, fasting blood was collected at 8.00am. Serum was separated and total cholesterol was measured by Carr-Drekter method. After separating the HDL cholesterol with phosphotungstic acid magnesium method, HDL was measured by Carr-Drekter method. Triglyceride was measured by using Tryglyceride Enzokit. (GPO - PAP) method. LDL and VLDL were calculated.

Treadmill Bruce Protocol was used for stress ECG. Machine used was CARDIOVIT Cs-6/12.
Test to measure Total Cholesterol

*Carr - Drekter method :*

(i) Principle : Cholesterol in serum is extracted into acetic anhydride in the presence of acetic acid. On treatment with a modified Liebermann - Burchard reagent (acetic anhydride/acetic acid/sulphuric acid) a green colour is produced, the intensity of which is measured at 630nm in a spectrophotometer, or using the orange filter (Illford 607 -600nm) or red filter (Illford 608 - 680nm). comparing it with a standard cholesterol solution similarly treated.

(ii) Test samples : The test was done on fresh serum.

(iii) Reagents : All reagents should be of analytical reagent (AR) grade.

(a) Acetic anhydride
(b) Glacial acetic acid

(c) Acid reagent: 100ml of glacial acetic acid were transferred into a 500ml flat at 25 - 30 C. bottom flask. This were kept in a basin containing ice. Slowly add 100ml concentrated sulphuric acid into the above flask. Mixed by gentle rotation and allowed to cool for 30 minutes. This reagent is stable for at least 6 months if kept in a well - stoppered bottle and stored.

(d) Dehydrating reagent: 10ml of the acid reagent were mixed with 10ml of glacial acetic acid. This reagent were also stable for at least 6 months if kept in a well stoppered bottle at 25-30C.

(e) Cholesterol standard (200mg/dl): 200mg pure cholesterol were mixed in glacial acetic acid and made up to 100 ml with glacial acetic acid. Stored at 25.-30C. This is stable for 6 months.

(iv) **Procedure:** Test tubes were labelled for blank, standard, and for test samples. Following reagents were added in the order and amount indicated.
<table>
<thead>
<tr>
<th>Reagents</th>
<th>Blank</th>
<th>Standard</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distilled water (ml)</td>
<td>0.2</td>
<td>0.2</td>
<td>-</td>
</tr>
<tr>
<td>Standard (200mg/dl)(ml)</td>
<td>-</td>
<td>0.2</td>
<td>-</td>
</tr>
<tr>
<td>Serum (ml)</td>
<td>-</td>
<td>-</td>
<td>0.2</td>
</tr>
<tr>
<td>Glacial acetic acid (ml)</td>
<td>0.8</td>
<td>0.6</td>
<td>0.8</td>
</tr>
</tbody>
</table>

Mixed gently and left for 2 minutes

Acetic anhydride (ml) 4.0 4.0 4.0

Mix each tube by gentle rotation. Centrifuge only the test (patient serum) tube for 10 minutes at 3,500 rpm, and then decant completely the supernatant into an 18 x 150 mm dry tube. Labelled that tube as ‘Test’. 
Set up a 25 °C water bath by adding ice water to tap water. To each tube add 1 drop of dehydrating reagent and rotate to mix. Immediately placed tubes in water bath at 25 °C. After 5 minutes when the tube have cooled to 25 °C, 1 ml of acid reagent added to each tube. Allow acid reagent to flow in freely without touching the wall of the tube. Mixed well by tapping the tubes. Replace the tube in 25 °C water bath and kept for further 20 minutes.

Zero the calorimeter with blank at 630nm orange filter and read the absorbance of other tubes. Cholesterol concentration were calculated by using the formula

$$\text{Cholesterol (mg/dl)} = \frac{\text{Absorbance of test} \times 202}{\text{Absorbance of standard}}$$

Factor 202 were used for correcting for small amount of supernatent fluid which remained in the original test tube, after decanting supernatent into second test tube.
Test to measure High Density lipoprotein

Phosphotungstate/Mg method

(a) Phosphotungstic acid (PH 6.15)

4 g of phosphotungstic acid were weighed, add 50 ml of water and adjust the PH to 6.15 with 1m NaOH. Then made upto 100 ml with distilled water.

(b) Magnesium chloride 2.5m/litre.

5.075 gram of magnesium chloride were weighed and made upto 10 ml with distilled water.

10 microliter of magnesium chloride + 50 microliter of phosphotungstic acid + 500 microliter of serum were taken. Kept for 10 minutes.
Supernatent were taken for cholesterol estimation as in Carr Drekter method.

Multiply the result by HDL cholesterol = \text{absorbance of test} \times 1.11 \\
\text{absorbance of standard.}
Test to measure Triglycerides.

Principle

Tryglycerides + H₂O (Lipoprotein lipase) → Glycerol + Fatty acids

Glycerol + ATP (Glycerokinase) → Glycerol - 3-phosphate + ADP

Glycerol 3-Phosphate + O₂ (Glycerol-3-phosphate oxidase) → H₂O₂ + Dihydroxyacetone phosphate

H₂O₂ + 4 Aminoantipyrine + ESPAS (Peroxidase) → Quinomine + H₂O

The red quinonemine dye formed due to peroxidase reaction is measured at 540nm and the intensity of the colour formed is directly proportional to the concentration of triglycerides in the sample.
Procedure

<table>
<thead>
<tr>
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<th>Blank</th>
<th>Standard</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pipette into Test Tubes</td>
<td>1ml</td>
<td>1ml</td>
<td>1ml</td>
</tr>
<tr>
<td>Working reagent</td>
<td>-</td>
<td>10ul</td>
<td>-</td>
</tr>
<tr>
<td>Standard</td>
<td>10ul</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Sample</td>
<td>10ul</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

Mixed and incubate at 37°C for 5 minutes. Mix and read absorbance of the test (AT), standard (As) and reagent blank (AB) at 540 nm in a calorimeter.

Calculations:

Triglycerides (mg/dl) = \(\frac{AT - AB \times 300}{As - AB}\)

@ LDL and VLDL cholesterol can be calculated using following formula.

\[\text{LDL} = \text{HDL} + \text{Triglycerides}/5 - \left(\chi - \left(\text{HDL} + \frac{\text{Triglycerides}}{5}\right)\right)\]

\[\text{VLDL} = \frac{\text{Triglycerides}}{5}\]
Blood pressure:

Blood pressure is the pressure exerted by blood, while flowing through vessels. Systolic pressure is the pressure exerted during systole and diastolic pressure is the pressure exerted during diastole. Normal blood pressure is 120/80. Pressure was measured by sphygmomanometer.

Electrocardiogram:

ECG is graphical representation of electrical activity of heart. The electrocardiograph is a sophisticated galvanometer, sensitive electromagnet which can detect and record changes in the electromagnetic
potential. The wire extension from these poles have electrodes at each end, i.e; a positive electrode at the end of extension from positive pole and a negative electrode at the end of extension from negative pole.

An electrocardiographic lead can be placed on the body in any three dimensional relationship to heart. There are 12 conventional leads which may be physiologically divided into 2 groups depending upon their orientation of heart.

(1) Frontal plane leads: - These are oriented in frontal or coronal plane of body and consist of standard leads I, II and III and leads AVR, AVL, AVF.

(2) The horizontal plane leads: - These are in transverse or horizontal plane of body and are formed by precordial leads; leads V1 to V6.

Standard Lead I: This lead is derived from placement of negative electrode on right arm and positive electrode on left arm.
Standard lead II: This lead is derived from placement of negative electrode on left arm and positive electrode on left foot.

Standard limb III: This lead is derived from placement of negative electrode on left foot.

The lead axes of three leads form a triangle and as electrodes of these leads are regarded as equidistant from heart. These lead axes thus form an equilateral triangle with heart at centre called Einthoven's triangle.

The unipolar limb lead:

The electrode of unipolar limb lead constitute exploring electrodes and in effect positive electrode of lead. The negative electrode is so constructed that is considered to be at zero potential. The exploring electrode reflects true potential. All unipolar leads are termed V leads and consist of extremity or limb leads and precordial or chest leads. Extremity leads are of low electrical potential and are instrumentally augmented.
Lead AVR is the augmented unipolar right arm lead. All the deflection P, Q, R, S and T deflection are normally negative.

Lead AVL is augmented unipolar left arm lead. Lead AVF is augmented unipolar left leg lead.

Precordial leads constitute V1, V2, V3, V4, V5 and V6

Basic Electrocardiographic deflections :-

Electrocardiographic deflections are termed P, Q, R, S, T and U. P wave represents atrial activation. QRS complex represents ventricular activation. T wave represents ventricular recovery or repolarisation. The T and U wave together represent total duration of ventricular recovery. (Leo Schamroth 1990)

ECG machine used here were CARDIART 40s. Before taking ECG the patients skin were cleared with alcohol at the site of electrode. A spot of cardiojelly were applied to skin and rubbed. Cardiojelly were
spread thickly on the surface of limb electrode. Then electrodes were placed on the skin and the patient cable were connected firmly to the corresponding electrode. Patients cable were connected as like.

Right arm       Red tip at black cord end
Left arm        yellow tip at black cord end
Left leg        Green tip at black cord end
Right leg       Black tip at black cord end
Chest V1        White tip at black cord end
Chest V2        White tip at black cord end
Chest V3        White tip at black cord end
Chest V4        White tip at black cord end
Chest V5        White tip at black cord end
Chest V6        White tip at black cord end

Then machine were started and ECG were recorded.
Treadmill test

Exercise testing is an important diagnostic and prognostic procedure in the assessment of patients with ischemic heart disease. The diagnostic utility of ECG was recognised by Feil and siegel in 1928. Here we used Treadmill Bruce Protocol for stress ECG. The treadmill protocol should be consist with patients physical capacity and purpose of test.

The machine used was CARDIOVIT CS-6/12. The main element of CARDIOVIT CS-6/12 comprise a thermal printer, a tray for printer paper an LCD display and key board divided into a control panel on the left and alphanumeric key board is used for the input of data.

Kit of electrocardiograph includes lead and patient cable. 4 precordial section electrodes and 6 chest electrodes. Patient chest were shaved. After cleaning with alcohol, a spot of cardiojelly were applied on the electrode, placed on respective portion.
The leads were connected as follows

R      Rt. arm
C1     Rt. axillary
C2     Front median Presternal
C3     45 Ventral from A
C4     left axillary
C5     near median over spinal cord
C6     neck
L      LA left arm
F      LF left leg.
After connecting leads resting ECG were taken. Blood pressure were measured by Sphygmomanometer. Age, sex and name were noted. Before each test, heart rate limit has to be noted. These were done by reducing age from a constant value 220.

Exercise test were started by pressing key B. Treadmill Bruce protocol consist of 7 stages. Each stages carries 3 minutes speed can be increased after 3 minute. If patient were showed any type of discomfort like chest pain, dizziness or dyspnoea, stress ECG were stopped.
Chapter I - V

Results
**Statistical Analysis:**

234 patients with coronary artery disease were selected for analysing risk factors from outpatient department of Little Flower Hospital. 100 subjects free of CAD were selected for analysing lipid profile for control group. Among 234 CAD patients, 72 TMT positive patients were selected for *Diet Intervention Trial*. 50 patients who followed the modified diet and lifestyle were included in the intervention group. 22 patients did not follow the modified diet and lifestyle strictly and they were included in the control group. Analysis of lipid profile showed that there were significant differences between coronary artery diseased patients and healthy subjects. Lipid profile analysis of *Diet intervention trial* showed that there were significant differences in the intervention group and control group. Paired ‘T’ test was used for testing the significance. (P < 0.5)
Average height of subjects was 151.70cm and weight was 60kg. Analysed data revealed that 49.56% of people were smokers. Among this, 29% started their smoking during early ages (i.e.-between the ages of 18 to 24 yrs. Most of early smokers became heavy smokers during their later life. Among the smokers 50% were heavy smokers and 50% were occasional smokers.

Lipid profile analysis of healthy subjects and diseased subjects showed differences. Total cholesterol level of healthy subjects was 184.28 ± 28.8mg/dl while that of CAD patients was of 216.93 ± 42.53. Mean value of LDL-C in healthy subjects was of 110.93 ± 30.77 and of experimental group was of 136.61 ± 41.67. The HDL level in healthy subjects was 43.02 ± 9.57 and in CAD subjects was 43.86 ± 9.84. Triglyceride level in the diseased subjects was higher (184.15 ± 85.50) while that of healthy subjects was lower (146.11 ± 58.5). TC/HDL ratio was higher in the CAD patients (5.02 ± 1.6) than in healthy subjects (4.53 ± 1.29).
Alcoholism was very common and most of these cardiac patients used alcohol occasionally. About 15% of people were using 80 or 100ml of alcohol per day. 13.86% of people were alcohol abusers. Among alcohol abusers 5% of people were taking more than 500ml daily.

Analysis of dietary habits showed that rice, tubers, pulses, fish, vegetables, beef were most common items. People were taking one plate of rice with a very little amount of vegetables. Root tubers were taken by low income group. Coconut kernel was used for making curries and sweets. Coconut oil was the cooking medium. Consumption of fresh vegetables was very rare. Consumption of fruits depended on availability.

90% of selected subjects were non vegetarians. 70% of people consume fish 4 or 5 days a week. Beef was the most commonly used meat (at least one or two days per week). Chicken and pork were eaten frequently. Mutton was taken very rarely. Among the diseased subjects about 15% were using 3 or 4 eggs per week. Analysis showed that 50% of non vegetarians were taking fish thrice or four times per week, beef once or twice in a week, chicken 1 or 2 times in a month, pork and mutton on rare occasions and egg once or twice in a week. 20% of people were
taking high amount of meat and fried items. 40% were consuming high carbohydrate diet with very little amount of fruits and vegetables. 50% people were taking about 4500 calories/day. Most of the vegetarians were taking ghee and fried items.

Intake of tea and coffee was higher. 20% of people were taking more than 8 cups of coffee or tea per day. 30% of people were taking 5 - 8 cups of tea per day. Rest of people were taking 2 - 4 cups of tea or coffee per day.

Data showed that 40% people were heavy workers, 30% were moderate workers and 30% were sedentary. The subjects had tensions from worry about their disease, future of children and economic status.

Mean Blood Pressure was 128/80. 15% of people exhibited hypertension. 27.08% of people had systolic pressure of about 130-150mm Hg, 23.75% of people had systolic pressure below 120.

In the Diet Intervention Trial, lipid profile values changed both in intervention group and control group. But significant change was observed.
only in the intervention group (table II, graph II - VI), while changes in the control group was not significant. (table III, graph VII- XII)

After 2 years TMT became negative in 12 subjects and exercise tolerance rate increased in 16 subjects. In the control group no patients exhibited negative TMT, but exercise tolerance rate increased in 7 patients.

Blood pressure also changed, but there was no significant difference. (Table VII, Graph VIII)

In the intervention group, total cholesterol reduced significantly (P<.001). HDL cholesterol which is known as good cholesterol increased more in the intervention group (P<.05) than control group. LDL which increases the risk of CAD, decreased significantly in the intervention group (P<.05) but not in the control group. Level of triglyceride also reduced to a significant level (P<.05) in the intervention group.
Table I: Comparison of lipid profile levels of healthy subjects and diseased subjects

<table>
<thead>
<tr>
<th></th>
<th>Healthy Subjects</th>
<th>n = 100</th>
<th></th>
<th></th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>TC</td>
<td>HDL</td>
<td>LDL</td>
<td>VLDL</td>
<td>TRI</td>
<td>TC/HDL</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>184.28</td>
<td>43.02</td>
<td>110.93</td>
<td>30.17</td>
<td>146.11</td>
<td>4.53</td>
<td></td>
</tr>
<tr>
<td>S.D.</td>
<td>± 28.8</td>
<td>± 9.5</td>
<td>± 30.77</td>
<td>± 15.4</td>
<td>± 50.51</td>
<td>± 1.29</td>
<td></td>
</tr>
<tr>
<td>CAD subjects</td>
<td>n = 234</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>216.93</td>
<td>43.86</td>
<td>35.10</td>
<td>36.64</td>
<td>184.15</td>
<td>5.02</td>
<td></td>
</tr>
<tr>
<td>S.D.</td>
<td>± 42.53</td>
<td>± 10.04</td>
<td>± 41.09</td>
<td>± 20.06</td>
<td>± 85.50</td>
<td>± 1.60</td>
<td></td>
</tr>
</tbody>
</table>
Table II: Changes in the lipid profile level of intervention group.

\[ n = 50 \]

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>8month</th>
<th>16month</th>
<th>24month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (T.C.)</td>
<td>224.62</td>
<td>205.54</td>
<td>191.72</td>
<td>184.34</td>
</tr>
<tr>
<td>SD</td>
<td>±40.58</td>
<td>±30.01</td>
<td>±30.02</td>
<td>±26.78</td>
</tr>
<tr>
<td>Mean (HDL)</td>
<td>41.28</td>
<td>42.98</td>
<td>42.55</td>
<td>47.46</td>
</tr>
<tr>
<td>SD</td>
<td>±11.51</td>
<td>±9.13</td>
<td>±9.15</td>
<td>±6.06</td>
</tr>
<tr>
<td>Mean (LDL)</td>
<td>138.85</td>
<td>125.24</td>
<td>119.34</td>
<td>111.04</td>
</tr>
<tr>
<td>SD</td>
<td>±35.76</td>
<td>±27.43</td>
<td>±25.90</td>
<td>±26.20</td>
</tr>
<tr>
<td>Mean (VLDL)</td>
<td>31.3</td>
<td>36.22</td>
<td>33.34</td>
<td>27.84</td>
</tr>
<tr>
<td>SD</td>
<td>±16.71</td>
<td>±14.73</td>
<td>±13.62</td>
<td>±7.51</td>
</tr>
<tr>
<td>Mean (Tri)</td>
<td>195.76</td>
<td>167.88</td>
<td>156.09</td>
<td>139.90</td>
</tr>
<tr>
<td>SD</td>
<td>±64.41</td>
<td>±51.94</td>
<td>±49.50</td>
<td>±37.51</td>
</tr>
</tbody>
</table>
Table III: Changes in the lipid profile level in the control group.

\[ n = 22 \]

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>8month</th>
<th>16month</th>
<th>24month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (T.C)</td>
<td>223.54</td>
<td>210.59</td>
<td>208.21</td>
<td>203.90</td>
</tr>
<tr>
<td>S.D.</td>
<td>+42.71</td>
<td>+34.37</td>
<td>+35.10</td>
<td>+37.02</td>
</tr>
<tr>
<td>Mean (HDL)</td>
<td>45.22</td>
<td>44</td>
<td>43.13</td>
<td>39.68</td>
</tr>
<tr>
<td>S.D.</td>
<td>+9.86</td>
<td>+6.91</td>
<td>+7.54</td>
<td>+5.74</td>
</tr>
<tr>
<td>Mean (LDL)</td>
<td>145.68</td>
<td>131.18</td>
<td>137.77</td>
<td>136.31</td>
</tr>
<tr>
<td>S.D.</td>
<td>+15.57</td>
<td>+11.27</td>
<td>+7.87</td>
<td>+30.01</td>
</tr>
<tr>
<td>Mean (VLDL)</td>
<td>32.13</td>
<td>30.13</td>
<td>27.72</td>
<td>27.90</td>
</tr>
<tr>
<td>S.D.</td>
<td>+15.57</td>
<td>+11.27</td>
<td>+7.87</td>
<td>+7.02</td>
</tr>
<tr>
<td>Mean (Tri)</td>
<td>73.90</td>
<td>154.13</td>
<td>147.77</td>
<td>144.27</td>
</tr>
<tr>
<td>S.D.</td>
<td>+88.20</td>
<td>+55.43</td>
<td>+47.75</td>
<td>+39.30</td>
</tr>
</tbody>
</table>
Graph I Differences in the serum lipid profile of CAD patients and Subjects free of CAD
Graph II: Changes in the serum Total cholesterol level in the Intervention group and control group.
Graph III Changes in the serum HDL level in the Intervention group and control group.
Graph IV: Changes in the serum LDL level in the Intervention group and control group.
Graph V: Changes in the serum VLDL level in the Intervention group and control group.
Graph VI: Changes in the serum Triglyceride level in the Intervention group and control group.
Graph VII

Changes in Systolic Pressure through an Interval of 3 months each

Graph VIII

Changes in Diastolic Pressure through an Interval of 3 months each
Chapter I - VI

Discussion
Smoking was a major risk factor. This was in agreement with the studies conducted by Ramankutty in 1993 and Chaddha 1990. Nicotine stimulate sympathetic nervous system and causes the body to produce more thromboxane and less prostacyclin. These changes increases heart rate and blood pressure and as a result arteries, throughout the body will constrict. Nicotine damages the lining of arteries causing blockages to form more readily decreasing blood flow. (Ornish 1990)

A large number of evidences relates high serum cholesterol to development of atherosclerosis and CAD (Kannel and Gordon 1976). Formerly it was established that total cholesterol level up to 250mg/dl was normal. According to this International standard, hypercholesterolemia was observed only in 10.5% of people. NCEP suggested that total cholesterol level below 200 are desirable. In our study average value of total cholesterol was below 200 in subjects without CAD and above 200 in CAD subjects. WHO in 1993 suggested that the level of total cholesterol below 170 was desirable for Indians. The mean level of total cholesterol, HDL, low density lipoprotein and triglycerides were
comparable with earlier published studies in India (Krishnaswamy 1989 and Jayakumari 1993).

Low HDL cholesterol was considered as a major risk factor for CHD. Mean value of HDL was about $40.065 \pm 1.65$. Only 10.5% of people had HDL cholesterol concentration below the desirable level. Analysed data showed that only 7% of people exhibited the HDL cholesterol level above 60mg/dl which was considered as negative risk factor. (NCEP1994)

Increase in concentration of LDL cholesterol increases the risk of CAD. Most studies strongly support a close relation between LDL and CAD. According to international standard, the mean level of LDL was normal in our study. 41% of people had LDL level above the normal in CAD subjects. In the healthy subjects 8% had LDL level above 150. According to WHO suggestion desirable LDL level was observed only in 16.5% of people in CAD subjects and 23% of people in healthy subjects. Level of LDL was higher in 76.5% of people in diseased subjects. LDL values in our study was in agreement with studies conducted by Rajmohan 1995, Jayakumari 1993 and Chaddha 1990.
Increased carbohydrate diet increases the triglyceride concentration with genetic forms of hypercholesterolemia. Many epidemiologic surveys document a relation between serum triglyceride levels and CAD rates (Halley 1980). According to international standard, high level of triglycerides was observed only in 39% of people. But according to Indian standard, high level of triglycerides was observed in 58% of CHD people. Mean level of triglyceride in healthy subjects was in borderline and high level in diseased subjects.

Analysis of lipid profile of healthy subjects and diseased subjects showed that all lipid fractions except HDL were higher in most of people. Level of lipid profile was comparatively lesser in disease free subjects than diseased subjects. Though lipid profile appeared to be higher (according to WHO suggestion) in this study, it agrees with results previously published studies by Rajmohan 1997, Jayakumari 1993 and Chaddha 1990.
High intake of alcohol was observed as a risk factor here. About 5% of alcoholics was without any other risk factors. Alcoholism was observed as a risk factor in studies conducted by Wallace 1961. Blood pressure was higher in the alcoholics than people without alcoholism. The alcoholics had a tendency to intake high amount of diet. Intake of high calorie can cause the development of CAD. High intake of beef which contains saturated fat and cholesterol was a risk factor. It can cause the development of atherosclerosis. Kritchesky in 1973 found the atherogenic effect of beef.

Anxiety and type A behaviour were observed in these patients, there was no patient having only these risk factors. In this study, these did not act as an independent risk factor.

Risk factor analysis showed that smoking, high level of serum lipid profile, alcoholism, and beef intake was the important risk factors in our area. This lead us to think that how the modification of diet and lifestyle affects serum lipid level and coronary artery disease.
In the diet intervention trial modified diet and lifestyle reduced the incidence of coronary artery disease. Modified diet and lifestyle simply meant that increased consumption of fruits and vegetables with little amount of fat and oil along with stopping of smoking and alcohol intake and moderate exercise. By following this diet and lifestyle risk of coronary artery disease decreased. This was achieved by decreasing the level of total cholesterol, LDL, VLDL and triglyceride and increased level of HDL. The changes in the lipid profile values were comparable with previous secondary prevention studies published in India. (Singh 1992) The changes in the lipid profile may be attained by the increased intake of fruits and vegetables with little amount of carbohydrate. Along with this, stopping of smoking and moderate physical activity may be a factor reducing the risk of CAD. During each counselling session advice was given for avoiding competitive mentality.

Dietary cholesterol was a significant independent explanatory variable for serum cholesterol. The reduction in fat intake reduces serum cholesterol level. (Ornish 1990, Gerhard et al 1992) In this study, patients were advised to reduce their fat intake. Patients were asked to reduce the consumption of carbohydrate. Increased intake of carbohydrates from
55% to 80% of total calorie increases serum cholesterol. (Girsberg 1976)

Excess carbohydrate after converting glycogen will convert to fatty acid in liver. (Guyton 1995)

In general, eating 400g/day fruits, vegetables and legumes was advisable for primary prevention and 600g/day was advisable for secondary prevention of CAD. (Ram Singh 1996) In our study we advised the subjects to take more fruits and vegetables. The modified diet was rich in antioxidants, vitamins A, E, C and beta-carotene and soluble dietary fibre which were protective against CAD. (Riemerson 1991)

Cooking medium used by the study group was coconut oil. By changing the diet and lifestyle along with this cooking medium, cholesterol level and risk of CAD reduced.

Though coconut oil was a saturated fat, it contains short chain and medium chain fatty acids. These fatty acids undergo metabolism and produce energy and does not deposit in the adipose tissue. Small and medium chain fatty acids containing triglycerides (SCT and MCT) do not require prolonged digestion. As the digestion is rapid, medium chain fatty acids and medium chain triglycerides will not suppress gastric emptying.
time as in the case of long chain fatty acids. Lingual lipase is effective on them and so a fraction is hydrolysed into free fatty acid in the stomach itself. Medium chain fatty acids diffuse directly into portal circulation where they bound to serum albumin. Hence they are immediately oxidised in the peripheral cells. Short chain fatty acids and medium chain fatty acids are preferentially oxidised and are not deposited in adipose tissues. Long chain fatty acids are passed to lymphatics, then to thoracic duct and systemic circulation carried by chylomicrons and deposited in the adipose tissue leads to hypercholesterolemia and atherosclerosis. (Vasudevan 1991) Coconuts also contain antioxidants and isokines which prevent oxidation of LDL (Raheena Beegam 1996)

Dietary supplementation with Vitamin A and C causes resistance of LDL to oxidation (Vincent 1993). Dietary Vitamin E administration found to decrease susceptibility of LDL to oxidative modification. (Jessup 1990) This modified diet supplies enough antioxidants through food. Reduction in the utilisation of high amount of carbohydrate may reduce the formation of triglycerides which has a positive relation with CAD.
Stopping of smoking and moderate physical activity may be factors that increased the HDL cholesterol in this study. Smoking can induce the atherosclerotic changes in LDL. Stopping of smoking can reduce the incidence of CAD and in our study this may be a factor for reducing the incidence of CAD. This finding agrees with studies conducted by Ornish.

Increased physical activity may be a factor for reducing the incidence of CAD. Some reports suggested that even a single session of moderate to long duration exercise can reduce blood pressure, triglyceride level and can increase HDL cholesterol level. This was comparable with studies conducted by Ram Singh 1996 and Shijun Dony 1998.

Though many studies revealed that moderate alcohol consumption decreases the risk of CAD, our modified lifestyle advised to abandon alcohol. The reason was that alcohol consumption with hypertriglyceridemia (Den Besten 1973) causes a rise in triglyceride (Moore 1986) The predominant increase in HDL3 cholesterol, instead of HDL-2 was caused by ingestion of alcohol. Increased incidence of liver disease, stroke, cancer and hypertension also limits therapeutic usefulness of alcohol. (Dean Ornish 1990)
During each visit patients were closely observed and advice were given to reduce their tension, competitive mentality and depression resulted by disease. Also this may have a little role in reduction of coronary artery disease.
Chapter I - VII

Conclusion
This study revealed that smoking, high level of lipids, high caloric diet, alcoholism, diabetes and beef intake were the risk factors of CHD for the subjects in this local rural setting. This study proved that coconut oil does not produce any atherogenic effect in human beings. Modification of diet and lifestyle along with this local cooking medium reduced the incidence of CAD. Changes in the dietary habits, quitting of smoking, stopping of alcoholism and reducing the tensions were the factors, which played their role in the risk reduction of coronary artery disease. But this study had certain limitations, that it cannot prove the independent role of diet or independent role of coconut oil in the development of coronary artery disease. So we conducted another study in rabbits to know the effect of cholesterol and coconut oil in the development of atherosclerosis.