

**EVALUATION OF CORONARY HEART DISEASE RISK
FACTORS AND RISK STRATIFICATION-
A STUDY OF 200 ASYMPTOMATIC SUBJECTS
ATTENDING MASTER HEALTH CHECKUP**

Dissertation Submitted to

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CERTIFICATE

This is to certify that the dissertation titled “**EVALUATION OF CORONARY HEART DISEASE RISK FACTORS AND RISK STRATIFICATION-A STUDY OF 200 ASYMPTOMATIC SUBJECTS ATTENDING MASTER HEALTH CHECKUP**” is the bonafide original work of **DR. A.MURALIDHARAN** in partial fulfillment of the requirements for **M.D. Branch – I (General Medicine)** Examination of the Tamilnadu DR. M.G.R Medical University to be held in September 2006. The Period of study was from January 2005 to December 2005.

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DECLARATION

I, **DR. A.MURALIDHARAN**, solemnly declare that dissertation titled **“EVALUATION OF CORONARY HEART DISEASE RISK FACTORS AND RISK STRATIFICATION-A STUDY OF 200 ASYMPTOMATIC SUBJECTS ATTENDING MASTER HEALTH CHECKUP”** is a bonafide work done by me at Govt. Stanley Medical College and Hospital during January 2005 to December2005 under guidance and supervision of my unit chief **Prof. S.SHIVAKUMAR**, Professor of Therapeutics.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – I) in General Medicine.**

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INTRODUCTION

The recognition of risk factors for coronary heart disease is one of the major achievements of coronary heart disease epidemiology in the 20th century.

Widespread interest in coronary heart disease and its prevention grew from the observation of increased rate of fatal and non-fatal myocardial infarction, particularly in men of relatively young age in westernized societies during the 1950s, 1960s and early 1970s. This led to landmark studies, such as the Framingham heart study initiated in 1948 and including close to 5000 men and women living in town of Framingham, near Boston USA. This cohort study together with several other similar studies, has subsequently demonstrated the importance of a number of risk factors¹.

A major shift in recent thinking has led to a preference for coronary heart disease risk profiling instead of the diagnosis and treatment of individual risk factors such as elevated blood pressure and cholesterol. By the year 2020, the burden of atherosclerotic coronary heart disease in India will surpass that in other regions of the world². Hence the current study has been undertaken to assess the coronary heart disease risk profile and stratify the risk of persons attending the master health checkup in our institution .

AIM OF THE STUDY

1. To evaluate the coronary heart disease risk factors of persons attending the MASTER HEALTH CHECKUP CLINIC of Government Stanley Hospital.
2. To evaluate and stratify the risk according to National cholesterol education programme-Adult Treatment Panel III(NCEP-ATPIII) guidelines.

REVIEW OF LITERATURE

EPIDEMIOLOGY:

PREVALENCE:

Worldwide:

Cardiovascular disease accounts for 18 million deaths per year in the world, coronary heart disease and cerebrovascular diseases being responsible for two thirds of the deaths and for approximately 22% of the 55 million deaths due to all causes³Estimates on mortality due to coronary heart disease according to the region indicate that developing countries contribute with a greater part of the overall burden of mortality due to the disease than developed countries⁴,with a relative excess of 70%.

India:

There have been no large prospective cohort studies to define the incidence of coronary heart disease in indians. The prevalence of coronary heart disease is rapidly increasing in India and is a major contributor to mortality and morbidity in india^{5,6}.Conservative estimates suggest that in 1990 coronary heart disease was responsible for 2.39 million deaths and the nation incurred a loss of 28.59 million disability adjusted life years(DALY's)⁷Epidemiological transition, with increasing life expectancy and demographic shifts of population ,age profile, combined with lifestyle related increases in the levels of coronary heart disease risk factors is

accelerating the coronary heart disease epidemic in india⁸.Recently,the prevalence of coronary heart disease in an urban population in South India was reported to be 11%⁹.

Immigrant Indians:

Indian immigrants in industrialized nations have higher prevalence rates compared to native indians. They are presumably at a more advanced epidemiologic transition. The first report to highlight the high coronary heart disease prevalence among immigrant indians came from an autopsy study from singapore. Coronary heart disease with myocardial infarction was 7 times more common in indians compared to chinese men¹⁰.Other studies from uganda¹¹,singapore¹²,south africa¹³and fiji¹⁴,confirmed a three fold higher coronary heart disease prevalence in indians compared to the respective natives.

CORONARY HEART DISEASE RISK FACTORS

Considerable work has gone into the epidemiology, pathophysiology and clinical implications of coronary heart disease risk factors, and many risk factors have been associated with an incidence of coronary heart disease. Furthermore, these risk factors can be divided into 'modifiable' or 'non-modifiable' depending upon the extent to which they can be altered or not.

MAJOR RISK FACTORS:

1.Modifiable:

- a.Hypertension
- b.Dyslipidemia
- c.Diabetes Mellitus
- d.Smoking
- e.Lifestyle factors such as diet, obesity. physical inactivity.

2.Non-modifiable:

- a.Family history of CHD
- b.Personal history of CHD
- c.Low birth weight
- d.Increasing age
- e.Male sex
- f.Ethnic origin
- g.Familial hyperlipidemia .

Emerging lipid risk factors:

- a. Elevated triglycerides
- b. Lipoprotein remnants
- c. Lipoprotein(a)
- d. Small dense LDL
- e. Apolipoproteins: elevated ApoB , low ApoA1

Emerging non-lipid risk factors:

- a. Homocysteine
- b. Thrombogenic/ hemostatic factors; High fibrinogen, activated factor VII, Plasminogen activator inhibitor(PAI-1)

The important ‘modifiable’ risk factors, which are well documented, include smoking, diabetes mellitus, hypertension and dyslipidemia. Male sex, family history and increasing age are the principal ‘non-modifiable’ major risk factors.

HYPERTENSION:

The Framingham study¹ was the first study to show conclusively that high blood pressure increased the risk of coronary heart disease. Indeed, there almost appears to be a “dose-response” relationship between hypertension and the risk of stroke or coronary heart disease, in that the higher the blood pressure, the greater the risk; conversely the reduction of blood pressure by anti-hypertensive treatment

reduces the risk of stroke and myocardial infarction. Other studies have since confirmed that the increased risk of coronary heart disease extends across racial, gender and age categories. Coronary heart disease also frequently co-exists with other risk factors, such as diabetes mellitus, hyperlipidemia. Furthermore, hypertensive patients have a worse prognosis after myocardial infarction than normotensive patients or those treated for hypertension¹⁵

DYSLIPIDEMIA

Raised total plasma cholesterol levels together with elevated low density lipoproteins(LDL) and lowered high density lipoproteins (HDL) cholesterol levels are associated with a high risk of atherosclerosis and increased risk of coronary heart disease risk^{16,17,18}.Atherogenic dyslipidemia ; elevated triglycerides, small dense LDL and low HDL occurs commonly in persons with premature CHD¹⁹

Many studies have also shown that reducing cholesterol by pharmacological means reduces the risk of eventual coronary heart disease both as primary and secondary prevention^{20,21}

DIABETES MELLITUS:

Diabetes mellitus another well established risk factor for coronary heart disease. Indeed , macro and microvascular complications along with increased atherosclerosis are well documented with diabetes mellitus^{22,23,24}.In particular, diabetics have almost a 50% higher chance of developing coronary heart disease than non-diabetics. It is all the more important as diabetes often co-exists with

other risk factors, such as hyperlipidemia and hypertension. Other coexisting features such as hyperinsulinemia and insulin resistance have also been implicated pathophysiologically in coronary heart disease. Recently, studies such as the UKPDS²⁵, have shown that strict control of blood sugar resulted in lower incidence of coronary heart disease

SMOKING:

Smoking continues to be one of the most important major modifiable risk factors in the development of coronary heart disease²⁶. Smokers have been shown to have almost a twofold increase in the risk of coronary heart disease as compared to non-smokers²⁷, whilst smokers who stop smoking lower their risk of coronary heart disease to that in non-smokers, but the risk reduction may take 2 years..

Cigarette smoking has been shown to have adverse effects on the lipid profile (it lowers HDL, but increases LDL and triglycerides) as well as causing an acute increase in blood pressure levels^{28,29,30,31}. Cigarette smoking also adversely affects platelet function and coagulation parameters, increasing the risk of thrombosis.

OBESITY:

The precise role of obesity as an independent coronary heart disease risk factor remains unclear. The definition of obesity is arbitrary but is often defined as an increase of 20% above ideal body weight. Obesity has a direct relationship with all the risk factors except smoking. Indeed obesity influences the other risk factors

such as dyslipidemia, hypertension and diabetes and thereby increases coronary heart disease risk.^{32,33} It has also been shown that patients with a 'central' fat distribution tend to be at a higher risk as opposed to a generalized obesity^{34,35}

FAMILY HISTORY:

Epidemiological studies have shown that diseases such as diabetes, hypertension, obesity and some forms of hypercholesterolemia have a familial predilection. These would put the individual at a higher risk of coronary heart disease. Other than certain familial forms of dyslipidemias, no familial forms of cardiovascular disease have been described.

AGE:

Increasing age has been shown to be a risk factor for coronary heart disease. The Framingham data¹ have shown a twofold increase in the rates of coronary heart disease in patients over the age of 60. However, this could also be due in part to the increased incidence of other coronary heart disease risk factors with increasing age.

MALE SEX:

Men are more prone to coronary heart disease than women¹. It is likely that oestrogens may play a protective role in women³⁶, as it is postmenopausal women who tend to have incidence rates of coronary heart disease approaching those of men³⁷. Similarly, epidemiological data suggest that women who are on hormone replacement therapy (HRT) tend to have rates similar to those of premenopausal

women³⁸. However the role of HRT in secondary prevention of coronary heart disease is debatable.

ETHNICITY:

Wide differences exist between the different races with regard to incidence of coronary heart disease. The prevalence of coronary heart disease rates in India is four fold higher than in the U.S³⁹. The high rates of coronary heart disease in India are in sharp contrast to low rates of coronary heart disease in china, hongkong, japan, taiwan, malaysia, indonesia, korea and other asian countries⁴⁰

Asian indians have a lower prevalence of conventional risk factors with the exception of abnormalities of glucose metabolism. In general, despite having some of the highest rates of hypertension and cigarette smoking in the world, coronary heart disease rates are low in other asian countries, where stroke is the dominant form of cardiovascular disease. Despite high rates of obesity, diabetes mellitus and low socioeconomic status, coronary artery disease rates are lower in hispanics than white americans. African americans have a higher prevalence of risk factors, especially low socioeconomic status, obesity, diabetes, and malignant hypertension, and yet they have no higher incidence of coronary artery disease than in whites.

**CARDINAL FEATURES OF CORONARY ARTERY DISEASE IN
INDIANS ⁴¹:**

- (a) Greater Prematurity
- (b) Greater Severity
- (c) Higher prevalence of insulin resistance syndrome
- (d) lower prevalence of conventional risk factors; hypertension, obesity, smoking, serum cholesterol levels(similar to whites but higher than other asians)
- (e) Higher prevalence of new and emerging risk factors; elevated lipoprotein(a) homocysteine, plasma fibrinogen

PHYSICAL INACTIVITY:

Physical inactivity is an exceptionally common modifiable risk factor for coronary artery disease. The risk of coronary artery disease in physically inactive people is almost twice that of physically active people⁴²and regular physical activity may protect against death from coronary artery disease.

DIET:

Food intake of different populations can greatly modify the risk factors and can account for some of the differences in coronary artery disease incidence seen among different ethnic groups⁴³.In the GISSI-Prevenzione study⁴⁴,a mediterranean diet along with supplementation with polyunsaturated fats(as

omega3polyunsaturated fatty acids)resulted in a significant decrease in coronary heart disease events post myocardial infarction.

PSYCHOSOCIAL AND BEHVIOURAL FACTORS:

Studies have consistently shown that emotional distress precedes the development of the symptoms of coronary heart disease^{45,46,47}

One large prospective study from Hawaii⁴⁸involving more than 8000 men failed to show any significant correlation between coronary heart disease risk and psychosocial/ behavioural factors.

CLUSTERING OF CORONARY HEART DISEASE RISK FACTORS

THE METABOLIC SYNDROME

The metabolic syndrome is a constellation of interrelated risk factors of metabolic origin that appear to directly promote the development of atherosclerotic coronary heart disease⁴⁹.Patients with metabolic syndrome are also at increased risk for developing type2 diabetes mellitus.

The predominant underlying risk factors for the syndrome appear to be abdominal obesity^{50,51,52} and insulin resistance^{53,54} other associated conditions can be physical inactivity^{51,55}aging⁵⁶and hormonal imbalance⁵⁷

CLINICAL DIAGNOSIS OF METABOLIC SYNDROME:

For the purpose of identification and treatment several organizations have attempted to define the metabolic syndrome.In 2001 U.S National cholesterol education programme-Adult Treatment Panel III(henceforth called ATP III)

recommended that atleast three of the following components are required to confirm the diagnosis of metabolic syndrome-abdominal obesity (men \geq 102cm, women \geq 88cm), increased triglycerides (\geq 150mg/dl), decreased HDL cholesterol (<40mg/dlmen, <50mg/dlwomen), hypertension(\geq 130/85mmHg) and impaired fasting glucose(\geq 110mg/dl)⁴⁹ The recent American Heart Association / National Heart,Lung,Brain Institute (henceforth called AHA/NHLBI) scientific statement on the diagnosis and management of metabolic syndrome has made the following modifications to the ATPIII Criteria⁵⁸

Risk factor	Defining level
Abdominal obesity	Men \geq 90cm,women \geq 80cm
Elevated triglycerides	\geq 150mg/dl or on drug treatment for elevated Triglycerides
Reduced HDL-C	<40mg/dl men,<50mg/dl women or on drug Treatment for reduced HDL-C
Elevated blood pressure	\geq 130 / 85 or on anti-hypertensive treatment
Elevated fasting glucose	\geq 100mg/dl or on drug treatment for elevated glucose

A series of studies ⁵⁹⁻⁶⁹ have found that many middle-aged people with the metabolic syndrome are at increased absolute risk for atherosclerotic coronary

heart disease in the near future. In the Botnia study⁵⁹ an almost threefold increased risk of coronary heart disease was found in those subjects with the metabolic syndrome, compared to those without.

Physical activity and a healthy, low-fat diet are associated with low levels of all risk factors associated with the metabolic syndrome and in patients with diabetes mellitus.

CONCEPT OF GLOBAL RISK

The term risk factor refers to characteristics that are independently related to the risk of subsequent occurrence of coronary heart disease. From the large prospective epidemiological studies such as the Framingham study, the multiple risk factor intervention trial (MRFIT) and the Munster Heart study (PROCAM), it is known that a person's risk of developing coronary heart disease rarely depends on a single risk factor⁷⁰⁻⁷². In most cases, the risk is determined by the interaction of two or more risk factors, each often of only mild degree. It is important to realize that the combined risk conferred by multiple risk factors may be considerably greater than the sum of the individual risks and their joint effect on global risk may be marked. The absolute risk of an individual determines future risk for symptomatic events and sets the indication for action

GLOBAL RISK ASSESSMENT:

In the light of increasing prevalence of coronary artery disease among indians, it is of vital importance to develop specific algorithms to evaluate the global risk of coronary artery disease in all individuals considered to be at risk. At present, there are no specific established guidelines that are widely accepted for calculating the risk of coronary heart disease (CHD) in indians. In the absence of such guidelines, it would be prudent to adopt the guidelines by the Adult Treatment Panel III (ATPIII) based on Framingham Risk Score method⁴⁹

ESTIMATING 10-YEAR CHD RISK FOR MEN AND WOMEN

Risk assessment for determining the 10-year risk for developing coronary heart disease may be carried out using Framingham risk scoring. The risk status in persons without clinically manifest coronary heart disease (CHD) or other clinical forms of atherosclerotic disease is determined by a two step procedure. First, the number of risk factors modifying LDL goal is counted⁴⁹

RISK FACTORS MODIFYING LDL GOALS

CIGARETTE SMOKING

HYPERTENSION (BP \geq 140/90 mm Hg or on anti-hypertensive medication)

LOW HDL CHOLESTEROL (< 40 mg/dl)*

Family H/o Premature CHD (first degree relative men \leq 55, women \leq 65)

AGE (men \geq 45, women \geq 55)

*HDL>60mg/dl counts as a ‘negative’ risk factor;its presence removes 1 risk factor from the total count.

Second, for persons with multiple(2+) risk factors,10 year risk assessment is carried out with Framingham scoring as shown in Annexure 1,2.

Risk factors used in Framingham scoring include age ,total cholesterol, HDL cholesterol, blood pressure and cigarette smoking.

RISK STRATIFICATION

ATP III identifies three categories of risk for CHD that modify goals ⁴⁹and modalities of LDL-lowering therapy: established CHD and CHD risk equivalents, multiple (2+) risk factors, and 0–1 risk factor

(a). Identification of persons with CHD and CHD risk equivalents

Coronary heart disease:. Persons with CHD are at very high risk for future CHD events (10-year risk >20 per-cent). Several clinical patterns constitute a diagnosis of CHD; these include history of acute myocardial infarction, evidence of silent myocardial infarction or myocardial ischemia, history of unstable angina and stable angina pectoris, and history of coronary procedures (coronary angioplasty and coronary artery surgery)

Other clinical atherosclerotic diseases: Persons in this subcategory have a CHD risk equivalent. Included are those with peripheral arterial disease, abdominal aortic aneurysm, carotid artery disease (symptomatic) [e.g., transient ischemic attack or stroke of carotid origin] or >50 percent stenosis on angiography or

ultrasound), and likely other forms of clinical atherosclerotic disease (e.g., renal artery disease).

Diabetes mellitus: ATP III counts diabetes as a CHD risk equivalent.

Multiple risk factors and 10-year risk for CHD >20 percent:

Based on 10-year risk assessment using Framingham scoring, a person in this category can be said to have a CHD risk equivalent.

(b) MULTIPLE (2 OR MORE) RISK FACTORS:

Individuals who fall into the multiple (2 or more) risk factor category may be further subclassified according to their 10-year CHD risk using the Framingham scoring system into those with 10-year CHD risk of more than 20 percent, 10 to 20 percent, and less than 10 percent. This further identifies individuals whose 10-year risk may warrant intensive management despite having a milder risk factor profile and allows better targeting of treatments to those who will benefit most.

(c). Zero to one risk factor:

Most persons with 0–1 risk factor have a 10-year risk <10 percent.

RISK MANAGEMENT

SMOKING:

Efforts to achieve smoking cessation by physicians are worthwhile. Aggressive Urging by physicians will convince some individuals to quit smoking.

DIETARY CHANGES:

Dietary changes have been found to be important in the management of diabetes and hypertension, as well as in treating obesity. Indeed, diets which are higher in fresh fruits and vegetables,⁷³ fish,⁷⁴ fibre and whole grains are associated with lower rates of heart disease.

OBESITY:

It has been shown that even modest(5-10%) reductions in weight are associated with health benefits, with reductions in blood pressure and cholesterol and triglyceride levels⁷⁵.Reduction of waist circumference results in improvement of metabolic coronary heart disease risk factors(blood pressure , lipid and glucose levels),but this is probably dependent on the loss of body weight⁷⁶.A calorie restricted lipid-lowering diet is the central component in weight reduction.Suitable regular physical activity adapted to the individual fitness and health status helps in weight reduction and in the maintenance of reduced body weight.

PHYSICAL INACTIVITY:

Physical inactivity is an exceptionally common modifiable risk factor for Coronary artery disease.Regular aerobic physical activity has favourable effects on body weight,blood pressure,plasma lipids,glucose tolerance,and insulin sensitivity and may also have a direct protective effect against the triggering of acute cardiac events.The duration of physical activity should preferably be 30-40 min, including a 5-10 minute warm-up phase before the 20-30min aerobic phase and a 5-10 min

Cool-down phase at its end, and as frequent as 4-5 times weekly.

HYPERTENSION:

Several lifestyle interventions are known to have a blood pressure-lowering effect. Treatment based on these interventions alone may be sufficient for patients with mildly elevated blood pressure and as emphasized above, it should always be advised for patients who are receiving anti-hypertensive drugs, because the dosage of anti-hypertensive drug needed for good blood pressure control can be reduced by lifestyle measures. Lifestyle interventions include: weight reduction in overweight individuals; reduction in use of sodium chloride to less than 5g/day; restriction of alcohol consumption to no more than 10-30g/day and to no more than 10-20g/day in women; and regular physical activity in sedentary individuals.

Efforts should be made to help hypertensive smokers to stop smoking.

Randomized trials of antihypertensive treatment have demonstrated the benefits of lowering blood pressure by antihypertensive drugs^{77,78}. Evidence from placebo-controlled and comparative trials also makes it clear that coronary heart disease protection can be obtained by treatments based on a variety of antihypertensive drug classes, i.e.; diuretics, beta-blockers, ACE inhibitors, Calcium antagonists and Angiotensin II antagonists. This presumably means that protection is due to a substantial degree, to blood pressure lowering per se^{79,80}

DYSLIPIDEMIA:

Lipid lowering can be undertaken both by pharmacological and non-pharmacological means. Exercise, high fibre diet, calorie reduction and better diabetic control help in lowering lipid levels. Both secondary prevention trials⁸¹⁻⁸⁴ and primary prevention trials⁸⁵⁻⁸⁷ have shown unequivocally the benefits derived from lowering cholesterol, with a 1% reduction in serum cholesterol being associated with a 2% reduction in coronary heart disease, whilst a 1% increase in HDL is associated with a 2-4% reduction in coronary heart disease. Effective lipid Lowering can also slow the rate of progression of coronary heart disease and also Achieve regression of atherosclerosis within 2 years.

The basic principle that guides cholesterol-lowering intervention is that the intensity of treatment is directly related to the degree of risk for CHD events. Both short-term (10-year) risk and long-term risk must be considered for treatment decisions. Persons with existing CHD (or a CHD risk equivalent) are at the highest risk; for this reason, they have the lowest goal level for LDL cholesterol and receive the most intensive treatment. For persons without CHD, classification and treatment goals are based on the category of risk, of which there are two—multiple (2+) risk factors other than LDL, and 0–1 risk factor. Persons with 2+ risk factors have an LDL goal that is not quite as low as that for persons with CHD (or CHD risk equivalents) ATP III⁴⁹ differs from ATP II in that it distinguishes three subcategories of risk among persons with multiple (2+) risk factors: 10-year risk

for hard CHD >20 percent, 10–20 percent, and <10 percent. Among the group with multiple risk factors, those at highest risk receive the most intensive LDL-lowering therapy, and those with the lowest risk receive the least intensive therapy. For persons with 0–1 risk factor, LDL goal levels are not as low as for persons with multiple risk factors, and intensive LDL-lowering therapy is not required unless LDL cholesterol levels are very high.

Therapeutic goals for LDL cholesterol

ATP III⁴⁹ recommends that LDL cholesterol be the primary target of therapy. The LDL cholesterol goals for each risk category are shown in Table 1.

TABLE 1

RISK LEVEL	LDL-C GOAL
CHD and CHD risk equivalent	<100mg/dl
Multiple(2+) risk factors	<130mg/dl*
0-1 risk factor	< 160mg/dl

* LDL-C goal for multiple-risk-factor persons with 10-year risk >20 percent is <100 mg/dl

Persons with CHD or CHD risk equivalent have an LDL cholesterol goal of <100 mg/dL. Those with multiple risk factors have an LDL cholesterol goal of <130 mg/dL; an exception is the patient with a CHD risk equivalent (>20 percent per 10 years) who has an LDL cholesterol goal <100 mg/dL. Finally, those with 0–1 risk factor have a goal LDL cholesterol of <160 mg/dL. These goals are set to

maximize reduction in both short-term and long-term risk. For persons whose LDL cholesterol levels are above the goal for the category, the goal of therapy is achieved through the judicious use of lifestyle and drug therapies. Lifestyle therapy in clinical management is designated. Therapeutic Lifestyle Changes (TLC). TLC includes the following: (a) reduced intakes of saturated fats and cholesterol, (b) therapeutic dietary options to enhance LDL lowering (plant stanols/sterols and increased viscous fiber), (c) weight control, and (d) increased physical activity.

ATP III ⁴⁹ recommends a two-step approach to cholesterol management. Priority goes to attaining the goal for LDL cholesterol; thereafter emphasis shifts to management of the metabolic syndrome and other lipid risk factors. Once the lipoprotein analysis is evaluated, risk factor counting and, if necessary, 10-year risk assessment are carried out to determine risk status. The patient is then started on dietary therapy or discharged with instructions for appropriate life-habit modifications. If the patient has CHD or a CHD risk equivalent, LDL-lowering drug therapy can be started simultaneously with dietary therapy if the LDL level warrants. After an appropriate trial of dietary therapy to reduce LDL cholesterol (~ 3 months), two additional therapeutic decisions may be required. First, if the LDL cholesterol goal has not been achieved, consideration may be given to initiating drug therapy. Second, if the metabolic syndrome is present, additional lifestyle changes (i.e., weight reduction and increased physical activity) will be needed. Later, if lifestyle therapies do not alleviate the metabolic

syndrome, drug therapy for treatment of the metabolic risk factors may be required.

Management of LDL Cholesterol

The following summarizes the ATP III ⁴⁹ approach to management of persons in the three categories of risk.

a. CHD and CHD risk equivalents

For persons with CHD and CHD risk equivalents, the type and intensity of LDL-lowering therapy are adjusted according to baseline LDL cholesterol level, i.e., whether ≥ 130 mg/dL, 100–129 mg/dL, or < 100 mg/dL. Each subcategory of LDL cholesterol is discussed below.

1) Baseline LDL cholesterol ≥ 130 mg/dL:

Persons with LDL cholesterol ≥ 130 mg/dL generally will require an LDL-lowering drug to achieve LDL cholesterol < 100 mg/dL. Therefore, a cholesterol-lowering drug should be initiated simultaneously with TLC and maximal control of other risk factors. If the LDL cholesterol falls to the range of 100–129 mg/dL on cholesterol-lowering therapy, several options are available depending on circumstances;(1)LDL lowering can be intensified with dietary therapy to achieve an LDL cholesterol level < 100 mg/dL(2) .LDL lowering can be intensified with drug therapy to achieve an LDL cholesterol level < 100 mg/dL..If the on-treatment LDL cholesterol level is near the goal of therapy, the physician can maintain

the current LDL-lowering therapy unchanged..If the metabolic syndrome is present, dietary therapy is intensified by increased efforts to reduce excess weight and increase physical activity..If the patient has elevated triglycerides or low HDL, a different lipid-lowering drug can be considered (e.g., nicotinic acid or fibric acid) for combination therapy with an LDL-lowering drug(statin)

2) Baseline LDL cholesterol 100–129 mg/dL:

When baseline LDL cholesterol is 100–129 mg/dL, several therapeutic options likewise are available. All approaches include TLC as initial therapy. Depending on circumstances, the following options are available: Inclusion of therapeutic dietary options (e.g., plant stanol /sterols and increased viscous fiber) can help to achieve the LDL goal..If LDL cholesterol levels remain appreciably above 100 mg/dL after 3 months of maximal dietary therapy, consideration can be given to adding an LDL-lowering drug..If the patient has an elevated triglyceride or low HDL cholesterol level, another lipid-lowering drug can be considered (e.g., nicotinic acid or fibric acid)..If the LDL cholesterol level falls to near the goal on dietary therapy alone, the physician can choose to forgo use of a lipid-lowering drug for the present.Because other risk factors may have contributed importantly to development of CHD in persons with low LDL levels, maximal control of nonlipid risk factors is necessary.

3) Baseline LDL cholesterol <100 mg/dL

If baseline LDL cholesterol is below the goal of therapy, further LDL-lowering

therapy is not currently recommended. Emphasis should be placed on controlling other risk factors and the metabolic syndrome. The TLC diet should be recommended to the person to help maintain a low LDL.

TABLE 2

Subcategory of LDL cholesterol level	LDL cholesterol goal	Level at which to initiate therapeutic lifestyle change(TLC)	Level at which to initiate LDL-lowering drugs
≥130mg/dl	<100mg/dl	>100mg/dl	Start drug therapy simultaneously with dietary therapy
100-129mg/dl	<100mg/dl	>100mg/dl	Consider drug options*
<100mg/dl	<100mg/dl	TLC and emphasize weight control and physical activity	LDL-lowering drugs not required

* Some authorities recommend use of LDL-lowering drugs in this category if an LDL cholesterol <100 mg/dL cannot be achieved by TLC. Others prefer use of drugs that primarily modify other lipoprotein fractions, e.g., nicotinic acid and fibrate. Clinical judgment also may call for withholding drug therapy in this subcategory.

b. Multiple (2+) risk factors:

ATP III distinguishes three subcategories of risk among persons with multiple risk factors, depending on 10-year risk: >20 percent, 10–20 percent, and <10 percent.

Within this category of multiple (2+) risk factors, intensity of therapy is adjusted according to 10-year risk and LDL cholesterol level. The treatment approach for each subcategory is shown in Table 3 .

The following reviews the approach to each subcategory in more detail.

1) Multiple risk factors, and 10-year risk >20 percent:

Persons with multiple risk factors and 10-year risk >20 percent have a CHD risk equivalent and are treated as described in the previous section .

2) Multiple risk factors, and 10-year risk 10–20 percent:

The goal for LDL cholesterol in this risk category is <130 mg/dL. The therapeutic aim is to reduce short-term risk as well as long-term risk for CHD. If baseline LDL cholesterol is ≥ 130 mg/dL, persons are started on TLC for a 3-month trial of dietary therapy, possibly augmented by options for further LDL lowering (plant stanols /sterols and increased viscous fiber). After 6 weeks and again after three months of dietary therapy, lipoprotein analysis is repeated. If LDL remains ≥ 130 mg/dL after three months, consideration can be given to starting an LDL-lowering drug to achieve the LDL goal <130 mg/dL. Should the LDL be less than 130 mg/dL on dietary therapy alone, it can be continued without adding drug treatment. If the metabolic syndrome is present, more attention should be given to weight control and increased physical activity.

3) Multiple risk factors, 10-year risk <10 percent:

The goal for LDL cholesterol in this risk category like-wise is <130 mg/dL. The

therapeutic aim, however, is primarily to reduce longer-term risk. If baseline LDL cholesterol is ≥ 130 mg/dL, persons are started on dietary therapy for reducing LDL cholesterol. Options for enhancing LDL lowering can be employed if needed to achieve the goal of therapy. After three months of dietary therapy, lipoprotein analysis is repeated. If LDL is < 160 mg/dL on dietary therapy alone, the dietary therapy should be continued. LDL-lowering drugs generally are not recommended because the patient is not at high short-term risk. On the other hand, if LDL cholesterol is ≥ 160 mg/dL, drug therapy can be considered to achieve an LDL cholesterol < 130 mg/dL.

TABLE 3

10 year risk	LDL cholesterol Goal	Level at which to initiate therapeutic lifestyle change(TLC)	Level at which to initiate LDL-lowering drugs
$>20\%$	$<100\text{mg/dl}$	$\geq 100\text{mg/dl}$	Treat as CHD equivalent
10-20%	$<130\text{mg/dl}$	$\geq 130\text{mg/dl}$	$\geq 130\text{mg/dl}$
$<10\%$	$<130\text{mg/dl}$	$\geq 130\text{mg/dl}$	$\geq 160\text{mg/dl}$

c. Zero to one risk factor:

Most persons with 0–1 risk factor have a 10-year risk < 10 percent. The goal for LDL cholesterol in this risk category is < 160 mg/dL. The primary aim of therapy is to reduce long-term risk. When baseline LDL cholesterol is ≥ 160 mg/dL,

persons are started on dietary therapy for three months. After 6 weeks, the LDL response is evaluated and dietary enhancers of LDL lowering (plant stanols/sterols and increased viscous fiber) may be added if necessary to reach the LDL goal. After 3 months, lipoprotein analysis is repeated. If LDL cholesterol is <160 mg/dL, dietary therapy is continued. For LDL cholesterol 160–189 mg/dL, drug therapy is optional depending on clinical judgment. Factors that favor use of drugs in this category include: A severe single risk factor (heavy cigarette smoking, poorly controlled hypertension, strong family history of premature CHD, or very low HDL cholesterol)..Multiple life-habit risk factors , emerging risk factors (if measured),10-year risk approaching 10 percent (if measured). If LDL cholesterol is ≥ 190 mg/dL despite dietary therapy in persons with 0–1 risk factor, drug therapy can be considered to achieve the goal of therapy in all adults. For persons with severe elevations of LDL cholesterol (e.g., ≥ 220 mg/dL), drug therapy can be started together with dietary therapy. Most such patients will have genetic forms of hypercholesterolemia that cannot be adequately treated with dietary therapy alone.

TABLE 4

Risk category	LDL cholesterol Goal	Level at which to initiate therapeutic lifestyle change(TLC)	Level at which to initiate LDL-lowering drugs
0-1 risk factor*	<160mg/dl	≥ 160 mg/dl	≥ 190 mg/dl

*Most persons with 0-1 risk factor have a 10-year risk of CHD <10%

d. Management of LDL cholesterol when risk assessment

begins with Framingham scoring :

If clinicians choose to begin risk assessment with Framingham risk scoring, the treatment algorithm is similar to that beginning with risk factor counting. The only difference occurs for persons whose 10-year risk is 10–20 percent and who have 0–1 risk factor; if one begins with risk factor counting, such persons would not have their 10-year risk calculated. This difference occurs in only 2.6 percent of the U.S. population that has 0–1 risk factor.

TABLE 5

10 year CHD risk	LDL cholesterol goal	Level at which to initiate therapeutic lifestyle change(TLC)	Level at which to initiate LDL-lowering drugs
>20% (High risk)	<100mg/dl	≥100mg/dl	Treat as CHD equivalent
10-20% (2+ risk factors)- moderately high risk	<130mg/dl	≥130mg/dl	≥130mg/dl
<10% (2+ risk factors)- moderate risk	<130mg/dl	≥130mg/dl	≥160mg/dl
<10% (0-1 risk factor)- low risk	<160mg/dl	≥160mg/dl	≥190mg/dl

e. Recommendations for persons whose LDL cholesterol levels are below goal

For persons whose LDL cholesterol levels are already below goal levels upon encounter, instructions for appropriate changes in life habits, periodic follow-up, and control of other risk factors are required .For all persons without CHD or CHD risk equivalents whose LDL is below goal, the diet for the general public

and a physical activity regimen should be recommended. For those with CHD or CHD risk equivalent, the therapeutic diet (TLC diet) should be recommended even if the LDL is below goal. Follow-up lipoprotein analysis should be carried out .

Management of atherogenic dyslipidemia and the metabolic syndrome:

After an adequate trial of dietary therapy for LDL lowering, attention should turn to atherogenic dyslipidemia and the metabolic syndrome. Treatment of these conditions usually begins after an initial 3-month period of dietary therapy to lower LDL cholesterol. Therapy for atherogenic dyslipidemia and metabolic syndrome thus begins after the LDL goal has been achieved with TLC alone or simultaneously with initiation of more intensive LDL-lowering therapy with drugs.

a. Atherogenic dyslipidemia

For atherogenic dyslipidemia, treatment strategy focuses on triglycerides. If triglycerides are ≥ 150 mg/dL and HDL cholesterol is < 40 mg/dL, a diagnosis of atherogenic dyslipidemia is made. The patient likely has the metabolic syndrome if triglycerides are ≤ 200 mg/dL, and specific drug therapy to reduce triglyceride-rich lipoproteins (TGRLP) is not indicated. However, if the patient has CHD or CHD risk equivalents, consideration can be given to using a drug to raise HDL cholesterol (fibrate or nicotinic acid), as outlined above under LDL-lowering therapy. On the other hand, if triglycerides are 200–499 mg/dL,

non-HDL cholesterol(T.C-HDL) becomes a secondary target of therapy. Goals for non-HDL cholesterol are 30 mg/dL higher than those for LDL cholesterol. First the LDL cholesterol goal is attained, and if non-HDL remains elevated, additional therapy may be required to achieve the non-HDL goal. Alternative approaches for treatment of elevated non-HDL cholesterol that persists after the LDL goal has been achieved are (a) higher doses of statins, or (b) moderate doses of statins + triglyceride-lowering drug (nicotinic acid or fibrate). If triglycerides are very high (500 mg/dL), attention turns first to prevention of acute pancreatitis, which is more likely to occur when triglycerides are ≥ 1000 mg/dL. Triglyceride-lowering drugs (fibrate or nicotinic acid) become first line therapy; although statins can be used to lower LDL cholesterol to reach the LDL goal, in these patients it is often difficult (and unnecessary) to achieve a non-HDL cholesterol goal of only 30 mg/dL higher than for LDL cholesterol.

b. Metabolic syndrome

Beyond treatment of elevated triglycerides, with drugs if necessary, first-line therapy for the metabolic syndrome is change in life habits, especially reducing weight and increasing physical activity. The approach to treatment of the metabolic syndrome with life-habit modification is presented below.

Management of the metabolic syndrome through life habit changes

a. Weight control

In ATP III⁴⁹, emphasis on weight reduction is delayed until after other dietary

measures are introduced for LDL lowering (reduced intakes of saturated fatty acids and cholesterol and possibly other options for LDL lowering [plant stanols/sterols and increased dietary fiber]). The delay in emphasizing weight reduction is to avoid overloading new patients with a multitude of dietary messages and to concentrate first on LDL reduction. After an adequate trial of LDL-lowering measures, attention turns to other lipid risk factors and the metabolic syndrome. Weight reduction then becomes a major focus of TLC. Indeed, weight control alone, in addition to lowering LDL cholesterol, favorably influences all of the risk factors of the metabolic syndrome.

b. Increased regular physical activity

The recommendation for increased physical activity is introduced when TLC is initiated and the recommendation is reinforced when emphasis shifts to management of the metabolic syndrome.

The purposes of regular exercise are to promote energy balance to maintain healthy body weight, to alleviate the metabolic syndrome, and to independently reduce baseline risk for CHD.

Decision about drug therapy; initiating management of the metabolic syndrome:

If the LDL cholesterol goal has not been achieved after 3 months of TLC, a decision must be made whether to consider adding drug therapy. If drugs are

started, TLC should be continued indefinitely in parallel with drug treatment.

Although the apparent ease of drug use is appealing, the additive effect of TLC to drug therapy in LDL cholesterol lowering is substantial and should not be overlooked.

DIABETES MELLITUS:

Coronary heart disease (macrovascular complications) are the most important Causes of morbidity and mortality in both type1 and type2 diabetes mellitus^{88,89}.The results of UKPDS study do not provide clear support that lowering the Glucose level decreases coronary heart disease risk.⁹⁰.Treatment of hyperglycemia is by both lifestyle modification and pharmacologic treatment.

Studies on risk Factors for Coronary Heart Disease (CHD)in Indians

A rise in the prevalence of CHD in the early half of twentieth century and a subsequent decline in the latter half have been well documented in the industrialized countries. However, the scenario is reversed in developing countries especially India with a steady escalation in prevalence of CHD⁴. Earlier studies on migrant Indians in the UK, USA, Canada and Trinidad showed that migrant Indians had higher rates of CHD compared to the indigenous population. It is consistently observed that Indians have premature CHD and that their risk for CHD was two to four times higher than the white European population.⁹¹ The SHARE study showed a CHD prevalence of 10.7% among South Asians

compared to 4.6% in Europeans⁹². Within the Indian subcontinent also, there has been a rapid rise in CHD prevalence. In 1959, Padmavati⁹³ reported the prevalence of CHD to be 1.0% and this rose to 4.5% in the year 1975⁶ and 7.9% in the year 1996 in subjects aged 20 years and above⁷. In a study of subjects aged 40 years and above by A. Ramachandran et al⁹⁴, The age-adjusted prevalence of probable CHD was 3.9% (3.5% in men and 4.5% in women), the prevalence of risk factors were in the order of central adiposity > dyslipidemia > hyperinsulinemia (2-h) > glucose intolerance > obesity > hypertension.

Women had higher prevalences of increased 2-h insulin (64.2 vs. 49%, p 0.001), glucose intolerance (43.5 vs. 35.6%, p 0.003), and obesity (33.4 vs. 21.4%, p 0.001), but lower central adiposity (56.6 vs. 64.1%, p 0.001) compared with Men⁹⁴

The Chennai Urban Population Study (CUPS)⁹ carried out in 1262 individuals > 20 years of age showed the crude prevalence of CHD to be 11% while the age-adjusted prevalence rate was 9.0%. In CUPS, LDL cholesterol and age were risk factors for CHD but serum triglyceride levels did not come out as an independent variable. The mean HDL was 40mg./dl which is low in our population⁹

Thus the prevalence of CHD appears to be ten times higher in India compared to that reported 40 years ago and the prevalence of CHD in urban Indians is fast approaching the figures reported in migrant Indians.⁹

Data from the Jaipur Heart Watch-2⁹⁵, on 1800 subjects based on a stratified

sampling technique reported an escalation in the prevalence rates of conventional coronary heart disease risk factors like obesity, diabetes and dyslipidemia among North Indians compared to the figures noted in 1990. Hypertension was present in 200 Men (36.4%) and 215 women (37.5%). Diabetes was present in 72 men (13.1%) and 65 women (11.3%). Truncal obesity was widely prevalent. The most common dyslipidemia in both men and women was low HDL-cholesterol (men 54.9%, women 54.2%). Prevalence of high total- and LDL-cholesterol and triglycerides was also seen in significant proportions ⁹⁵

Achari and Thakur ⁹⁶ report on a large retrospective study on 5748 CHD patients and 8103 healthy normals. Serum cholesterol levels, LDL cholesterol levels and total cholesterol to HDL ratio were higher among the CHD subjects compared to normals. The study also makes another interesting observation that there is a lack of association of serum triglycerides levels with CHD ⁹⁶

In the case-control study by Burman *et al*, again LDL cholesterol levels and total cholesterol/HDL cholesterol ratio and Lp(a) levels were higher in CHD patients compared to controls but there was no significant difference in serum triglyceride levels. ⁹⁷ Another large clinic-based study on 17,855 type 2 diabetic subjects looked at the association of isolated hypercholesterolemia and isolated hypertriglyceridemia with CHD ⁹⁸. The prevalence of CHD was significantly higher among patients with isolated hypercholesterolemia, isolated high LDL and

isolated low HDL cholesterol compared with normalipidemic individuals, but not in those with isolated hypertriglyceridemia. There appears to be differences in lipid associations with CHD between native and migrant Indians. In migrant Indians, serum triglyceride levels have been consistently found to be associated with CHD⁹⁹. However in native Indians, LDL cholesterol and total cholesterol/HDL cholesterol Ratio appears to be more important. One factor which is common to all Indians is a low HDL cholesterol levels⁹⁹. In the face of low HDL cholesterol levels, even moderate elevation of LDL cholesterol appears to be sufficient to produce an atherogenic profile. However, the role of triglycerides cannot be completely ruled out as the link between hypertriglyceridemia and CHD has been shown in several studies. Increased production of small VLDL in response to hypertriglyceridemia could contribute to atherosclerosis. This is confirmed by the presence of triglyceride-rich lipoproteins in the human atheroma. Moreover, increase in triglyceride levels are associated with low HDL cholesterol and with small dense LDL molecules (Phenotype B) 'Atherogenic dyslipidemia' is associated with metabolic syndrome and may be responsible for accelerated atherosclerosis⁹⁴. It is opined that although the total cholesterol levels in Asian Indians is similar or lower as compared to caucasians¹⁰⁰ atherogenic dyslipidemia is more common, which may contribute to CHD. Recently an increased prevalence of small dense LDL in migrant Asian Indians has been shown in a study conducted in USA¹⁰¹.

Prevalence of elevated LDL cholesterol defined by the NCEP guidelines levels is only 38.8% among CHD subjects in the study done by Achari ⁹⁶. This suggests that either the cut-off used for elevated LDL cholesterol is not appropriate among Indians or that more than 60% of the CHD is not explained by elevated LDL cholesterol levels. A similar finding was observed in the clinic study ⁹⁸ done on 17,855 type 2 diabetic patients that the prevalence of myocardial infarction was 2.9% in subjects with LDL levels below 100 mg/dl compared to 61% in subjects with LDL above 100 mg/dl. This could mean that in Indians, even those with LDL below the cut-off of NCEP have a high risk for CHD suggesting that aggressive lipid lowering is justified in Indian. In this regard the findings of Heart Protection Study are of great interest. The main message from that study is to “treat high risk patients with high dose-statin irrespective of their baseline cholesterol levels”¹⁰². This seems to be most applicable to Indians, where the threshold of LDL cholesterol for CHD appears to be lower than that demonstrated in Western populations. The main limiting factors of course would be the cost.

Recently, a number of newer coronary heart disease risk factors have been identified. These factors are of great interest in native Indians where more than 60% of the CHD remains unexplained by conventional risk factors. Comparative studies on newer risk factors illustrated that Asian Indians have higher C-reactive protein, plasminogen activator inhibitor (PAI 1) and homocysteine levels ¹⁰³.

Lipoprotein (a) levels have been consistently shown to be elevated among Asian Indians compared to other ethnic groups suggesting a genetic predisposition to coronary artery disease.^{104,105} The study by Burman, et al⁹⁷ done on 20 CHD patients and 20 healthy normals showed that Lp(a) level was associated with CHD. This confirms several earlier reports from India¹⁰⁶.

Given that India is a large country with tremendous urban-rural differences and regional variations large, multicentre prospective long-term follow-up studies addressing non-communicable diseases like diabetes, hypertension and CHD are the urgent need of the hour.

MATERIAL AND METHODS:

This study was conducted at Govt. Stanley Medical College & Hospital, Chennai. This study was conducted during the period of one year Jan 2005-December 2005, 200 asymptomatic subjects attending the MASTER HEALTH CHECKUP CLINIC of Govt. Stanley Medical college & hospital were taken up for the study

INCLUSION CRITERIA:

Men aged ≥ 45 years and women aged ≥ 55 years with no previous history Of coronary heart disease, cerebrovascular disease , peripheral vascular disease, Diabetes mellitus, Hypertension were taken up for the study.

EXCLUSION CRITERIA:

Subjects with known history Of coronary heart disease, cerebrovascular disease ,peripheral vascular disease, Diabetes mellitus, Hypertension.

METHODS:

A total of 200 asymptomatic subjects were taken up for the current study. All were subjected to history taking, thorough clinical examination and investigations that included past history of diabetes, dyslipidemia, hypertension, coronary heart disease, cerebrovascular disease, peripheral arterial disease and renal disorder; family history of hypertension, diabetes, dyslipidemia, premature coronary heart disease, cerebrovascular disease and renal disorder; personal history of physical activity and smoking

The individuals were then subjected to thorough physical examination.

BLOOD PRESSURE:

Two sets of blood pressure reading were taken 5 minutes apart, with patient in sitting position after 5 minutes of rest. The first and the fifth korotkoff's sounds were used to define systolic and diastolic blood pressure respectively. Blood pressure was taken in both arms and the higher of them was taken into consideration. The average of the two readings were taken for diagnosis. Subjects were termed hypertensive when blood pressure reading $\geq 140/90$ according to JNC VII criteria¹⁰⁷.

ANTHROPOMETRIC MEASUREMENTS:

Height

Height was measured by fixing a tape measure to a wall and measuring the height with a movable headboard, with measures to the nearest centimeter. Subjects were asked to stand upright without shoes, with their back against the wall, heels together and eyes directed forward.

Weight

Weight was measured with a traditional spring balance which was kept on a firm horizontal surface. The "Zero" was checked each day and calibration was done with reference to an individual of "known" weight. The subjects were asked to wear light clothing and weight was recorded to the nearest 0.5 kg.

The body mass index(BMI) was calculated using the formula:
 $\text{weight/height}^2(\text{kg/m}^2)$

Subjects with BMI 25-29.9 kg/m² were labelled overweight and BMI \geq 30 as
Obese¹⁰⁸

Waist:

Waist circumference was measured using a non stretchable fibre measure tape.

The subjects were asked to stand erect in a relaxed position with both feet together. One layer of light clothing was accepted. Waist girth was measured at the midpoint between the iliac crest and the lower margin of the ribs. Waist circumference was measured to the nearest centimeter. Waist circumference \geq 90cm in men and \geq 80 cm in women was taken as evidence of abdominal Obesity⁵⁸.

HIP:

Hip girth was recorded at the greatest trochanter (the widest portion of the hip) on both sides.Measures were made to the nearest centimeter.

waist-Hip ratio(WHR) calculated as waist circumference in cm divided by hip circumference in cm..Truncal obesity was diagnosed if Waist-Hip ratio(WHR) $>$ 0.9 in men and $>$ 0.8 in women^{109,49}.

Physical inactivity was defined as less than 30 minutes a day of moderate physical activity on most days of the week.

A thorough physical examination which included peripheral vascular, coronary heart disease, respiratory, abdominal, nervous system and fundus examination was done. An 8 hour fasting blood sample was taken for biochemical investigations. A 2-hour post prandial blood sample was taken after giving 75g oral glucose for postprandial blood sugar estimation.

Biochemical investigations:

The following biochemical investigations were done; fasting(FBS) and 2-hour postprandial blood sugar(PPBS)- Diabetes mellitus was considered if FBS \geq 126mg/dl and/or PPBS \geq 200, Impaired fasting glucose(IFG) FBS \geq 100mg/dl and Impaired glucose tolerance(IGT) PPBS \geq 140 mg/dl¹¹⁰ were noted.

Blood urea and serum creatinine was measured.

Lipid profile was evaluated with Total cholesterol(TC), HDL cholesterol(HDL-C) and triglycerides(TGL). LDL cholesterol (LDL-C) was estimated using Friedwald equation $[LDL-C=TC-(HDL+TGL/5)]^{111}$. Dyslipidemia was considered when TC \geq 200mg/ dl, LDL \geq 130mg / dl, TGL \geq 150mg / dl, HDL <40mg/dl⁴⁹.

Urine examination was done to assess urine sugar and albumin.

A 12-lead ECG was taken to assess the presence of coronary heart disease. Echocardiography was carried out to assess the presence of regional wall motion abnormality. Ultrasonogram was done to assess the renal status.

From these data the following risk factors which modify LDL goal were categorized⁴⁹

RISK FACTORS MODIFYING LDL GOALS

CIGARETTE SMOKING

HYPERTENSION(BP \geq 140/90mm Hg or on anti-hypertensive medication)

LOW HDL CHOLESTEROL(<40mg/dl)*

Family H/o Premature CHD(first degree relative men \leq 55,women \leq 65)

AGE(men \geq 45,women \geq 55)

All the subjects in our study had minimum one risk factor modifying LDL goal which was age(men \geq 45,women \geq 55).

The subjects were divided into the following risk categories according to ATPIII Guidelines⁴⁹

Categories of Risk for Coronary Heart Disease (CHD) Risk Categories

Established CHD & CHD risk equivalents

Multiple (2+) risk factors

0–1 risk factor

Established CHD and CHD risk equivalents:

Coronary heart disease:. Persons with CHD are at very high risk for future CHD events (10-year risk >20 per-cent). Several clinical patterns constitute a diagnosis of CHD; these include history of acute myocardial infarction, evidence of silent myocardial infarction or myocardial ischemia (by ECG), history of unstable

angina and stable angina pectoris, and history of coronary procedures (coronary angioplasty and coronary artery surgery)

Other clinical atherosclerotic diseases: Persons in this subcategory have a CHD risk equivalent. Included are those with peripheral arterial disease, abdominal aortic aneurysm, carotid artery disease (symptomatic [e.g., transient ischemic attack or stroke of carotid origin] or >50 percent stenosis on angiography or ultrasound), and likely other forms of clinical atherosclerotic disease (e.g., renal artery disease).

Diabetes mellitus: ATP III counts diabetes as a CHD risk equivalent.

Framingham risk score:

The Framingham risk score(FRS) for estimating 10-year CHD risk⁴⁹ was utilised for further risk stratification(Annexure1,2). Risk factors used in Framingham scoring include age , total cholesterol, HDL cholesterol, blood pressure and cigarette smoking.

Framingham scoring divides persons with multiple risk factors into those with 10-year risk for CHD of >20%, 10-20%, and <10%.

Subjects were further categorized as

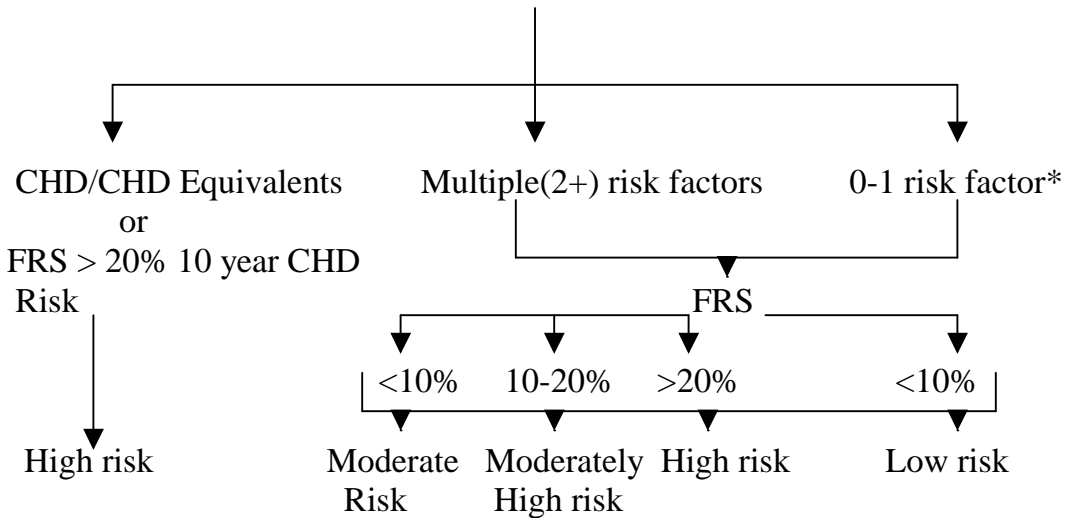
High risk: CHD* or CHD risk equivalents/10-year risk >20%

Moderately high risk: 2 or more risk factors(10-year risk 10% to 20%)

Moderate risk: 2 or more risk factors (10-year risk<10%)

Low risk: 0–1 risk factor*(*Most persons with 0-1 risk factor have a 10-year risk of CHD <10%)

ASSESSMENT OF NO. OF RISK FACTORS MODIFYING LDL GOAL



(*Most persons with 0-1 risk factor have a 10-year risk of CHD <10%)

The clinical features of metabolic syndrome were evaluated using AHA/NHLBI criteria⁵⁸-metabolic syndrome was said to be present if any three of the following risk factors were present

Risk factor	Defining level
Abdominal obesity	Men≥90cm, women≥80cm
Elevated triglycerides	≥150mg/dl
Reduced HDL-C	<40mg/dl men,<50mg/dl women
Elevated blood pressure	≥130/85mmHg
Elevated fasting glucose	≥100mg/dl

STATISTICAL ANALYSIS:

Student's t test was used to analyse quantitative variables and Chi square test for qualitative variables. A p value of <0.05 was considered as significant.

After evaluation of subjects for coronary heart disease risk factors and Categorization into appropriate risk categories according to ATP III guidelines, the individuals were recommended therapeutic life style changes (cessation of Smoking, increased physical activity, dietary changes), anti-hypertensives for control of hypertension, glycemic control by appropriate medications and lipid lowering therapy to achieve the LDL goal for the respective risk categories (Table 6)

TABLE 6

10 year CHD risk	LDL cholesterol goal
>20% (High risk)	<100mg/dl
10-20% (2+ risk factors)- moderately high risk	<130mg/dl
<10% (2+ risk factors)- moderate risk	<130mg/dl
<10% (0-1 risk factor)- low risk	<160mg/dl

RESULTS

200 subjects were taken up for the study out of which 128(64%) were men and 72(36%) were women. The mean age was 52.1 in men and 57.9 in women.

The age structure of subjects in both sexes is shown in Table 7.

AGE –TABLE 7

AGE	MALE (45-78)(n=128)	FEMALE (55-80)(n=72)	OVERALL (n=200)
45-50	70(54.7)*	0	70(54.7)
51-55	19(14.8)	26(36.1)	45(22.5)
56-60	20(15.6)	35(48.6)	55(27.5)
61-65	11(8.6)	8(11.1)	19(9.5)
66-70	5(3.9)	0	5(3.9)
71-75	2(1.6)	2(2.8)	4(2.2)
76-80	1(0.8)	1(1.4)	2(1)
MEAN	52.1	57.9	

*Figures in parentheses denote percentages

The mean, range and standard deviation of the various clinical and biochemical variables of the subjects is shown in Table 8.

Table 8

VARIABLES		MEAN	S.D	MIN	MAX
AGE (Yrs)	M	52.1	7.7	45	78
	F	57.9	4.5	55	80
Systolic BP (mm Hg)	M	133.9	23.4	90	200
	F	143.3	23.5	100	190
Diastolic BP (mm Hg)	M	84.9	11.1	60	120
	F	87.4	12.2	60	120
BMI	M	23.6	4.3	13.75	34.6
	F	24.8	4.96	13.3	35.6
WHR	M	0.92	0.07	0.78	1.1
	F	0.84	0.06	0.72	1.1
Waist Circumference (cm)	M	86.4	10.1	58	109.5
	F	83.4	10.8	55	108
FBS (mg/dl)	M	98.4	31.7	50	250
	F	94.8	24.3	60	185
PPBS (mg/dl)	M	151.6	67.5	72	415
	F	138.6	43.9	84	293
SERUM T.C (mg/dl)	M	205.5	50.6	94	340
	F	204.3	55.2	115	415
LDL (mg/dl)	M	139.6	46.8	55	265
	F	139.3	51.7	66	330
TGL (mg/dl)	M	142.8	58.4	47	396
	F	129.3	37.1	71	279
HDL (mg/dl)	M	37.2	5.5	22	50
	F	39.1	6.3	21	58
VLDL (mg/dl)	M	28.5	11.7	9	79
	F	25.8	8	14	56

The coronary heart disease risk factors analysed in the subjects

are shown in table 9 TABLE 9

RISK FACTORS	MEN-N=128(64)	WOMEN-N=72(36)	TOTAL(N=200)
smoking	52(40.6)*	0	52(26)
Physical inactivity	123(96.1)	72(100)	195(97.5)
H/o prem.CHD	5(3.9)	3(4.2)	8(4)
Overweight (BMI25-29.9)	37(28.9)	23(31.9)	60(30)
Obesity (BMI≥30)	12(9.4)	12(16.7)	24(12)
Truncal obesity (WHR>0.9 men,>0.8 women)	64(50)	43(55.1)	107(53.5)
Abdominal obesity (≥90cmmen≥80 cm women)	50(39.1)	46(63.9)	96(48) p=0.001
Hypertension(≥ 140/90mm Hg)	52(40.6)	38(52.8)	90(45)
IFG/IGT	43(33.6)	18(25)	61(30.5)
DIABETES	20(15.6)	11(15.3)	31(15.5)
High total cholesterol (≥200mg/dl)	67(52.3)	35(48.6)	102(51)
LDL≥130mg/dl	70(54.7)	39(54.2)	109(54.5)
TGL≥150mg/dl	48(37.5)	17(23.6)	65(32.5) p=0.04
Low HDL<40mg/dl	81(63.3)	33(45.8)	114(57) p=0.01
Metabolic syndrome	42(32.8)	36(50)	78(39) p=0.01

* Figures in parentheses denote percentages

The most common overall major coronary heart disease risk factor was physical inactivity present in 97.5% of subjects. Next common risk factor was low HDL Cholesterol, present in 57% of subjects. LDL Cholesterol ≥ 130 mg/dl(54.5%), Total cholesterol ≥ 200 mg/dl(51%), TGL ≥ 150 mg/dl(32.5%) were the dyslipidemias noted. Hypertension was noted in 45% of subjects.

Dysglycemia(IFG/IGT) was seen in 30.5% of subjects. Newly diagnosed Diabetes mellitus was noted in 15.5% of subjects. 30% of subjects were overweight(BMI 25-29.9) and 12% obese(BMI > 30). Truncal obesity(53.5%) and abdominal obesity(48%) were noted. Metabolic syndrome was present in 39% of subjects. Family history of premature CHD(4%) was rare. Abdominal obesity and metabolic syndrome were significantly higher in women.

Hypertriglyceridemia(TGL ≥ 150 mg/dl) and Low HDL < 40 mg/dl were significantly higher in men.(Table 9)

Age wise prevalence of risk factors in men are shown in Table 10

Smoking was common in 61-65 age group(72.7%) and 45-50 age group(42.8%)

The prevalence of Hypertension increased after the age of 50 years in men.

Age wise prevalence of risk factors in men

TABLE 10

RISK FACTOR	45-50 n=70	51-55 n=19	56-60 n=20	61-65 n=11	>65 n=8
SMOKING	30(42.8)*	6(31.6)	5(25)	8(72.7)	3(37.5)
Physical inactivity	68(97.1)	19(100)	17(85)	11(100)	8(100)
Family H/O Prem CHD	2(2.8)	1(5.3)	1(5)	0	1(12.5)
Overweight (BMI 25-29.9)	24(34.3)	8(42.1)	4(20)	1(9.1)	0
OBESE (BMI≥30)	6(8.6)	0	3(15)	2(18.2)	1(12.5)
TRUNCAL OBESITY	39(55.7)	9(47.4)	10(50)	5(45.4)	1(12.5)
ABDOMINAL OBESITY	29(41.4)	7(36.8)	9(45)	2(18.2)	3(37.5)
Hypertension	23(32.8)	10(52.6)	10(50)	5(45.4)	4(50)
IFG/IGT	19(27.1)	8(42.1)	8(40)	3(27.3)	5(62.5)
DM	13(18.6)	2(10.5)	1(5)	4(36.4)	0
TC≥200 mg/dl	41(58.6)	9(47.4)	10(50)	6(54.5)	1(12.5)
LDL≥130 mg/dl	39(55.7)	10(52.6)	11(55)	8(72.7)	2(25)
TGL≥150 mg/dl	31(44.3)	7(36.8)	6(30)	3(27.3)	1(12.5)
HDL<40 mg/dl	44(62.8)	13(68.4)	13(65)	7(63.6)	4(50)
METABOLIC SYNDROME	22(31.4)	7(36.8)	8(40)	3(27.3)	2(25)

*Figures in parentheses denote percentages

Age wise prevalence of risk factors in female are shown in Table 11

Table 11

RISK FACTOR	51-55 n=26	56-60 n=35	61-65 n=8	>65 n=3
SMOKING	0	0	0	0
Physical inactivity	26(100)*	35(100)	8(100)	3(100)
Family H/O Prem CHD	1(3.8)	2(5.7)	0	0
Overweight (BMI25-29.9)	8(30.8)	12(34.3)	2(25)	1(33.3)
OBESE(BMI≥30)	5(19.2)	5(14.3)	2(25)	0
TRUNCAL OBESITY	12(46.1)	25(71.4)	5(62.5)	1(33.3)
ABDOMINAL OBESITY	16(61.5)	21(60)	7(87.5)	2(66.7)
HTN	11(42.3)	17(48.6)	7(87.5)	3(100)
IFG/IGT	9(34.6)	6(17.1)	2(25)	1(33.3)
DM	4(15.4)	5(14.3)	2(25)	0
TC≥200 mg/dl	15(57.7)	15(42.8)	3(37.5)	2(66.7)
LDL≥130mg/dl	16(61.5)	16(45.7)	5(62.5)	2(66.7)
TGL≥150mg/dl	7(26.9)	9(25.7)	1(12.50)	0
HDL<40mg/dl	12(46.1)	14(40)	5(62.5)	2(66.7)
METABOLIC SYNDROME	14(53.8)	14(40)	7(87.5)	1(33.3)

*Figures in parentheses denote percentages

The prevalence of Hypertension increased after the age of 55 years in women.

The Framingham risk score of the subjects are shown in Table 12

TABLE 12

FRAMINGHAM RISK SCORE	MALE n=128	FEMALE n=72	OVERALL n=200
<10% 10-yr CHD risk	42(32.8)*	64(88.9)	106(53)
10-20% 10-yr CHD risk	60(46.9)	8(11.1)	68(34)
>20% 10-yr CHD risk	26(20.3)	0	26(13)

*Figures in parentheses denote percentages

The Risk categories of the subjects are shown in table 13

TABLE 13

RISK CATEGORY	MALE n=128	FEMALE n=72	OVERALL n=200
LOW	14(10.9)*	14(19.4)	28(14)
MODERATE	26(10.7)	36(50)	62(31)
MODERATELY HIGH	46(35.9)	8(11.1)	54(27)
HIGH(CHD EQUIVALENT)	42(32.8)	14(19.5)	56(28)

*Figures in parentheses denote percentages

High risk: CHD or CHD risk equivalents/10-year risk >20%

Moderately high risk: 2 or more risk factors(10-year risk 10% to 20%)

Moderate risk: 2 or more risk factors (10-year risk<10%)

Low risk: 0–1 risk factor

Overall 28% of individuals were in high risk (CHD equivalent) group(men-32.8%,women19.5%).In men 35.9% were in moderately high risk compared to 11.1% in women.50% of women were in moderate risk group compared to 10.7% in men

The multiple risk factor profile modifying LDL goal are shown in Table 14

TABLE 14

RISK FACTORS MODIFYING LDL GOAL	MALE n=128	FEMALE n=72	OVERALL n=200
AGE	20(15.6)*	19(26.4)	39(19.5)
AGE+SMOKING	6(4.7)	0	6(4.7)
AGE+Hypertension(HTN)	7(5.5)	15(20.8)	22(11)
AGE+LOW HDL	28(21.9)	15(20.8)	43(21.5)
AGE+SMOKING+LOW HDL	17(13.3)	0	17(13.3)
AGE+HTN +LOW HDL	24(18.7)	21(29.2)	45(22.5)
AGE+SMOKING+HTN	10(7.8)	0	10(7.8)
AGE+SMOKING+HTN+ LOW HDL	14(10.9)	0	14(10.9)
AGE+SMOKING+LOW HDL+Family H/o prem CHD	1(0.8)	0	1(0.8)
AGE+HTN+HDL+ Family H/o premCHD	1(0.8)	2(2.8)	3(1.5)

*Figures in parentheses denote percentages

Multiple(2+) risk factors that modify LDL goal were present in 84.4% of men and 73.6% of women. Overall the combination of AGE+HYPERTENSION+LOW HDL(22.5%) and AGE+LOW HDL(21.5%) were common.

The risk factor profile comparison between hypertensive and normotensive

Individuals is shown in table 15

Table 15

RISK FACTORS		HTN MEN=52 WOMEN=37	NON-HTN MEN=76 WOMEN=35	
Smoking	M	24(46.1)*	28(36.8)	
	F	0	0	
Physical inactivity	M	49(94.2)	74(97.4)	
	F	37(100)	35(100)	
Family H/o premature CHD	M	2(3.8)	3(3.9)	
	F	2(5.4)	2(5.7)	
Overweight(BMI25-29.9)	M	19(36.5)	17(22.4)	
	F	13(35.1)	10(28.6)	
Obesity (BMI≥30)	M	8(15.4)	4(5.3)	
	F	10(27)	2(5.7)	p=0.02
Truncal Obesity	M	25(48.1)	39(51.3)	
	F	25(67.6)	17(48.6)	
Abdominal obesity	M	25(48.1)	25(32.9)	p=0.03
	F	31(83.8)	14(40)	p=0.01
IFG/IGT	M	16(30.8)	27(35.5)	
	F	9(24.3)	9(25.7)	
DIABETES	M	9(17.3)	11(14.5)	
	F	6(16.2)	5(14.3)	
High total cholesterol (≥200mg/dl)	M	30(57.7)	37(48.9)	
	F	21(56.7)	13(37.1)	
LDL≥130 mg/dl	M	45(86.5)	25(32.9)	p=0.01
	F	24(64.9)	14(40)	
TGL≥150 mg/dl	M	22(42.3)	26(34.2)	
	F	8(21.6)	9(25.7)	
Low HDL<40 mg/dl	M	34(65.4)	47(61.8)	
	F	20(54)	14(40)	
Metabolic syndrome	M	27(51.9)	15(19.7)	p=0.01
	F	25(67.6)	11(31.4)	

*Figures in parentheses denote percentages

The risk factor profile comparison between Normal Glucose Tolerance(NGT) and IFG/IGT/DM subjects is Shown in table 16

TABLE 16

RISK FACTORS	NGT			IFG/IGT			DM		
	M n=65	F n=43	T n=108	M n=43	F n=18	T n=61	M n=20	F n=11	T n=31
SMOKING	25 (38.5)*	0	25(23.1)	16(37.2)	0	16 (26.2)	11 (55)	0	11 (35.5)
PHYSICAL INACTIVITY	61(93.8)	43(100)	107(99.1)	42(97.7)	18(100)	60 (98.4)	20 (100)	11 (100)	31(100)
H/oPRE. CHD	2(3.1)	1(2.3)	3(2.8)	3(7)	1(5.5)	4(6.5)	0	1(9.1)	1(3.2)
OVERW T	19(29.2)	11(25.6)	30(27.8)	12(27.9)	9(50)	21 (34.4)	6(30)	3 (27.3)	9(29)
obesity	6(9.2)	5(11.6)	11(10.2)	4(9.3)	5(27.8)	9(14.7)	2(10)	2 (18.2)	4(12.9)
Truncal obesity	25(38.5)	24(55.8)	49(45.4)	22(51.2)	11(61.1)	33 (54.1)	16 (80)	7 (63.6)	23 (74.2)
									p=0.02
Abdominal obesity	19(29.2)	22(51.2)	41(38)	18(41.9)	16(88.9)	34 (55.7)	10 (50)	8 (72.7)	18 (58.1)
									p=0.03
HTN	27(41.5)	23(53.5)	50(46.3)	16(37.2)	9(50)	25 (41)	9(45)	6 (54.5)	15 (48.4)
TC ≥200 mg/dl	34(52.3)	21(48.8)	55(50.9)	19(44.2)	8(44.4)	27 (44.3)	15 (75)	6 (54.5)	21 (67.7)
LDL ≥130 mg/dl	35(50.8)	23(53.5)	58(53.7)	20(46.5)	8(44.4)	28 (45.9)	15 (75)	8 (72.7)	23 (74.2)
									p=0.04
TGL ≥150 mg/dl	17(26.2)	8(18.6)	25(23.1)	19(44.2)	5(27.8)	24 (39.3)	11 (55)	4 (36.4)	15 (48.4)
									p=0.01
HDL <40 mg/dl	37(56.9)	23(53.5)	60(55.5)	31(72.1)	9(50)	40 (65.6)	13 (65)	2 (18.2)	15 (48.4)
METABOLIC SYNDROME	12(18.5)	14(32.5)	26(24.1)	19(44.2)	13(72.2)	32 (52.4)	11 (55)	9 (81.8)	20 (64.5)
									p=0.01

*Figures in parentheses denote percentages

The individual components of metabolic syndrome in NGT,IFG/IGT/DM are shown in table 17

TABLE 17

METABOLIC SYNDROME COMPONENTS	NGT		IFG/IGT		DM	
	MEN n=12	WOMEN n=14	MEN n=19	WOMEN n=13	MEN n=11	WOMEN n=9
DYSLIPIDEMIA (TGL≥150mg/dl & or HDL<40mg/dl)	0	0	0	0	1(9.1)	2(22.3)
ABD.OBESITY+ DYSLIPIDEMIA	3(25)*	1(7.1)	7(36.8)	4(30.8)	4(36.4)	3(33.3)
ABD.OBESITY + HTN	0	0	0	0	0	0
HTN + DYSLIPIDEMIA	2(16.7)	0	3(15.8)	1(7.7)	2(18.2)	1(11.1)
ABD.OBESITY + HTN + DYSLIPIDEMIA	7(58.3)	13(92.8)	9(47.4)	8(61.5)	4(36.3)	3(33.3)

*Figures in parentheses denote percentages

The risk categorization in individuals with metabolic syndrome

TABLE 18

METABOLIC SYNDROME			
RISK CATEGORY	MALE n=42	WOMEN n=36	OVERALL n=78
LOW	1(2.4)*	3(8.3)	4(5.1)
MODERATE	3(7.1)	18(50)	21(26.9)
MODERATELY HIGH	17 (40.5)	5(13.9)	22(28.2)
HIGH(CHD EQUIVALENT)	21(50)	10(27.8)	31(39.7)

*Figures in parentheses denote percentages

Risk factors and Risk categorization shown in Table 19

TABLE 19

RISK FACTOR	LOW RISK		MODERATE RISK		MODERATELY HIGH RISK		HIGH RISK/ CHD EQUIVALENT	
	M	F	M	F	M	F	M	F
	N=14	N=14	N=26	N=36	N=46	N=8	N=42	N=14
SMOKING	0	0	5 (19.2)	0	21(45.6)	0	26(61.9)	0
Physical inactivity	13(92.8)	14	25 (96.1)	36(100)	44(95.6)	8(100)	41(97.6)	14(100)
Family H/O Prem CHD	0	0	1 (3.8)	1(2.8)	3(6.5)	0	1(2.4)	2(14.3)
Overweight(BMI 25-29.9)	4(28.6)	5 (35.7)	7 (26.9)	11 (30.5)	14(30.4)	3(37.5)	12(28.6)	4(28.6)
OBESE	0	1 (7.1)	1 (3.8)	8 (22.2)	5(10.9)	1(12.5)	6(14.3)	2(14.3)
TRUNCAL OBESITY	6(42.8)	7(50)	12 (46.1)	25 (69.4)	21(45.6)	4(50)	25(59.5)	10(71.4)
ABDOMINAL OBESITY	4(28.6)	8 (57.1)	9 (34.6)	22 (61.1)	18(39.1)	7(87.5)	19(45.2)	9(64.3)
HTN	0	0	7 (26.9)	23 (63.9)	23(50)	7(87.5)	22(52.4)	8(57.1)
IFG/IGT	6(42.8)	7 (50)	9 (34.6)	8 (22.2)	16(34.8)	2(25)	12(28.6)	1(7.1)
DM	0	0	0	0	0	0	20(47.6)	11(78.6)
TC≥200 mg/dl	4(28.6)	6 (42.8)	8 (30.8)	14 (38.9)	24(52.2)	6(75)	31(73.8)	9(64.3)
LDL≥130 mg/dl	5(35.7)	6 (42.8)	9 (34.6)	16 (44.4)	26(56.5)	6(75)	30(71.4)	11(78.6)
TGL≥150 mg/dl	1(7.1)	4 (28.6)	5 (19.2)	8 (22.2)	19(41.3)	1(12.5)	23(54.8)	4(28.6)
HDL<40 mg/dl	0	0	21 (80.8)	23 (63.9)	31(67.4)	6(75)	29(69)	5(35.7)
METABOLIC SYNDROME	1(7.1)	3 (21.4)	3 (11.5)	18(50)	17(36.9)	5(62.5)	21(50)	10(71.4)

figures in parentheses indicate percentages

Newly diagnosed Diabetes mellitus, dyslipidemia, hypertension and smoking were the important risk factors contributing to high risk categorization in men.

DISCUSSION

The epidemic of coronary heart disease has taken deep roots in india and many other developing countries⁴.The burden of death and disability due to coronary heart disease(CHD) continues to increase² and in the absence of suitable preventive efforts is not being controlled¹¹². A large number of studies have highlighted that the primordial prevention strategy of CHD involving control of three lifestyle-related risk factors- smoking, physical inactivity and aberrant diet-is the most cost-effective method¹¹².

Major coronary risk factors: smoking , hypertension , high low density lipoprotein (LDL) Cholesterol , diabetes , obesity , truncal obesity , insulin resistance , low high-density lipoprotein(HDL)-cholesterol are secondary manifestations of these deviant lifestyles and explain more than 90% of the incidence of CHD worldwide¹¹³ Retrospective analysis of previous Indian CHD and risk factor epidemiological studies have reported increasing prevalence of the major coronary risk factors such as smoking , hypertension , diabetes , hypercholesterolemia ,obesity and truncal obesity^{4,7,112}.The increase in these risk factors correlates positively with the increasing CHD in india ⁸

Evaluation of coronary heart disease risk factors can be done by many methods.In this study ATPIII ⁴⁹ guidelines was used to evaluate the coronary heart disease risk factors and for risk stratification.ATP III identifies three categories of

risk for CHD that modify goals and modalities of LDL-lowering therapy: established CHD and CHD risk equivalents, multiple (2+) risk factors, and 0–1 risk factor. Risk assessment for determining the 10-year risk for developing coronary heart disease may be carried out using Framingham risk scoring(Annexure 1,2). The risk status in persons without clinically manifest coronary heart disease(CHD) or other clinical forms of atherosclerotic disease is determined by a two step procedure.First, the number of risk factors modifying LDL goal is counted ⁴⁹

RISK FACTORS MODIFYING LDL GOALS

CIGARETTE SMOKING

HYPERTENSION(BP \geq 140/90mm Hg or on anti-hypertensive medication)

LOW HDL CHOLESTEROL(<40mg/dl)*

Family H/o Premature CHD(first degree relative men \leq 55,women \leq 65)

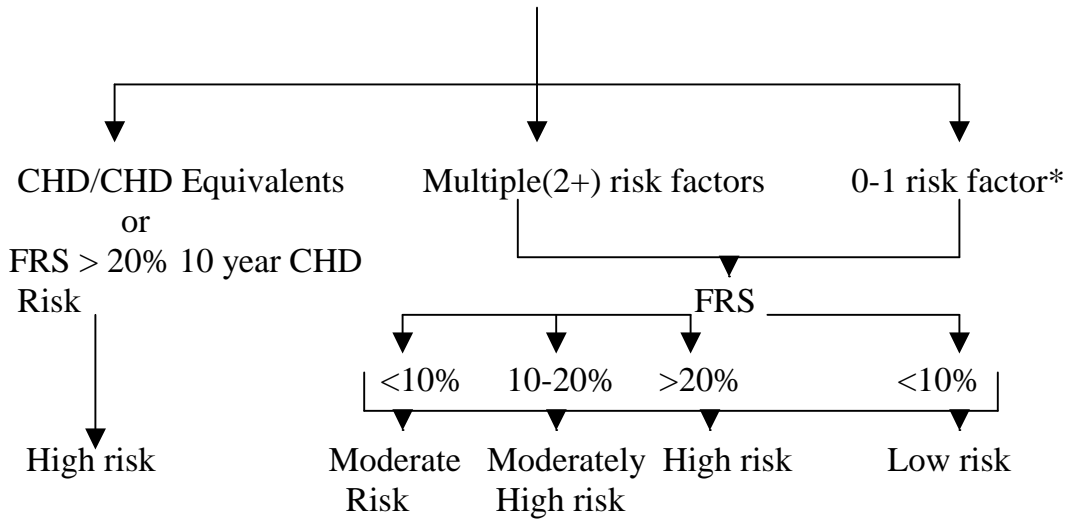
AGE(men \geq 45,women \geq 55)

*HDL $>$ 60mg/dl counts as a ‘negative’ risk factor;its presence removes 1 risk factor from the total count.

Second, for persons with multiple(2+) risk factors,10 year risk assessment is carried out with Framingham scoring as shown in Annexure 1,2.

Risk factors used in Framingham scoring include age ,total cholesterol,HDL cholesterol,blood pressure and cigarette smoking.

ASSESSMENT OF NO. OF RISK FACTORS MODIFYING LDL GOAL



(*Most persons with 0-1 risk factor have a 10-year risk of CHD <10%)

a. Identification of persons with CHD and CHD risk equivalents:

Coronary heart disease: Persons with CHD are at very high risk for future CHD events (10-year risk >20 per-cent). Several clinical patterns constitute a diagnosis of CHD; these include history of acute myocardial infarction, evidence of silent myocardial infarction or myocardial ischemia(by ECG), history of unstable angina and stable angina pectoris, and history of coronary procedures (coronary angioplasty and coronary artery surgery)

Other clinical atherosclerotic diseases: Persons in this subcategory have a CHD risk equivalent. Included are those with peripheral arterial disease, abdominal aortic aneurysm, carotid artery disease symptomatic(e.g., transient ischemic attack or stroke of carotid origin)

Diabetes mellitus: ATP III counts diabetes as a CHD risk equivalent.

Multiple risk factors and 10-year risk for CHD >20 percent:

Based on 10-year risk assessment using Framingham scoring, a person in this category can be said to have a CHD risk equivalent.

(b) MULTIPLE(2+) RISK FACTORS:

Individuals who fall into the multiple (2 or more) risk factor category may be further subclassified according to their 10-year CHD risk using the Framingham scoring system into those with 10-year CHD risk of more than 20 percent, 10 to 20 percent, and less than 10 percent. This further identifies individuals whose 10-year risk may warrant intensive management despite having a milder risk factor profile and allows better targeting of treatments to those who will benefit most.

Further risk stratification of individuals is done by ATP III guidelines as

High risk: CHD* or CHD risk equivalents/10-year risk >20%

Moderately high risk: 2 or more risk factors(10-year risk 10% to 20%)

Moderate risk: 2 or more risk factors (10-year risk <10%)

Low risk: 0–1 risk factor*

(*Most persons with 0-1 risk factor have a 10-year risk of CHD <10%)

In our study 200 asymptomatic subjects (men ≥ 45 , women ≥ 55) attending master health checkup were analysed for major coronary heart disease risk factors. Emerging lipid risk factors; Lipoprotein remnants, Lipoprotein(a), Small dense LDL, Apolipoproteins: ApoB, ApoA1 and non-lipid risk factors; Homocysteine, fibrinogen, activated factor VII, Plasminogen activator inhibitor (PAI), were not evaluated.

There were 128-male, 72-female subjects. Mean age in men was 52.1, women 57.9. The most common overall major coronary heart disease risk factor was physical inactivity present in 97.5% of subjects. Next common risk factor was low HDL Cholesterol present in 57% of subjects. LDL Cholesterol ≥ 130 mg/dl (54.5%), Total cholesterol ≥ 200 mg/dl (51%), TGL ≥ 150 mg/dl (32.5%) were the dyslipidemias noted. Hypertension was noted in 45% of subjects.

Dysglycemia (IFG/IGT)¹¹⁰ was seen in 30.5% of subjects. Newly diagnosed Diabetes mellitus was noted in 15.5% of subjects. 30% of subjects were overweight (BMI 25-29.9) and 12% obese (BMI ≥ 30). Truncal obesity (53.5%) and abdominal obesity (48%) were noted. Metabolic syndrome was present in 39% of subjects. Family history of premature CHD (4%) was rare. (Table 9).

Men:

Physical inactivity (96.1%), Smoking (40.6%), hypertension (40.6%), dysglycemia (IFG/IGT) – 43%, dyslipidemia were the important coronary heart disease risk factors in men. Low HDL < 40 (63.3%) and High LDL ≥ 130 (54.7%) were the important

dyslipidemias. Newly diagnosed Diabetes mellitus (CHD equivalent) seen in 15.6% Obesity (9.4%) and family h/o premature CHD (3.1%) were low. 32.8% had the metabolic syndrome as diagnosed by AHA/NHLBI criteria. Hypertriglyceridemia and low HDL were significantly more in men. (Table 9). ECG evidence of Coronary Heart disease seen in 5.5%

Women:

Physical inactivity (100%), truncal obesity (55.1%) and abdominal obesity (63.9%), hypertension (52.8%) and dyslipidemia were the important coronary heart disease risk factors. In women high LDL (54.2%) was the most common dyslipidemia. Newly diagnosed Diabetes mellitus (CHD equivalent) seen in 15.3%. 50% had the metabolic syndrome as diagnosed by AHA/NHLBI criteria. Smoking was not present in women. Abdominal obesity and metabolic syndrome was significantly more in women (Table 9). ECG evidence of Coronary Heart disease seen in 4.2%.

Prevalences of risk factors:

The overall prevalences of risk factors in our study in descending order of frequency were physical inactivity > dyslipidemia > truncal obesity (WHR > 0.9 Men, > 0.8 men) > Glucose intolerance (IFG / IGT / DM) > Hypertension > obesity (BMI ≥ 30). This is in contrast to the Study done by A. Ramachandran et al⁹⁴ who in a population > 40 years reported the

prevalence of risk factors were in the descending order of frequency
truncal obesity >dyslipidemia> glucose intolerance(IFG/IGT/DM)>obesity >
hypertension.

The overall prevalence of hypertension in our study was 45%.Prevalence of hypertension is higher among women(52.8%) than men(40.6%)(Table 8).This is in contrast to Chennai urban population study(CUPS)¹¹⁴ where overall prevalence was 21% and a higher prevalence among men(22.8%) than women(19.7%).This difference may be because of an older age group in our study .

Abdominal obesity was significantly higher in hypertensive subjects of both sexes than non-hypertensives(Table 15).This is similar to the CUPS¹¹⁴.There was no significant difference in smoking rates between hypertensive and non-hypertensive men which is similar to CUPS¹¹⁴ .LDL cholesterol was significantly higher in hypertensive men similar to CUPS¹¹⁴.Metabolic syndrome was significantly higher in hypertensive groups of both sexes(Table 15)

Table 10 and Table 11 show the age wise distribution of hypertension.The prevalence of hypertension is increasing after the age of 50 years in both sexes.Virtually all studies have shown a rise in blood pressure with age,which was also observed in our study

The most common dyslipidemia in men was low HDL(63.3%) which is similar to Jaipur heart watch 2 study ⁹⁵. In women high LDL was the most

common dyslipidemia. The mean HDL (37.9) was low in our study group which is similar to CUPS¹¹⁴ where the mean HDL was 40.

METABOLIC SYNDROME:

Individuals with IFG/IGT/DM had a clustering of coronary heart disease risk factors and the prevalence of metabolic syndrome as diagnosed by AHA/NHLBI criteria was 56.5% compared to 24.1% in normal glucose tolerance (NGT) individuals which was statistically significant (Table 16). Similar reports have been published in north India which showed a prevalence of metabolic syndrome was 61% in IFG compared to 16.1% in NGT¹¹⁵.

Truncal obesity and abdominal obesity was significantly more in IFG/IGT/DM than NGT individuals in both sexes. Dyslipidemia (LDL \geq 130mg/dl, TGL \geq 150mg/dl) was significantly more in persons with IFG/IGT/DM than NGT individuals. (Table 16).

Among individuals with metabolic syndrome the combination of abdominal obesity+Hypertension+Dyslipidemia was the commonest in both NGT and IFG/IGT/DM individuals. (Table 17)

RISK STRATIFICATION:

Framingham risk score $>20\%$ 10 yr CHD risk was 20.3% in men while 46.9% were in the 10-20% 10 yr CHD risk score range. In women 88.9% individuals were in $<10\%$ 10 yr CHD risk score range, $>20\%$ 10 yr CHD risk – nil. (Table 12)

Overall 28% of individuals were in high risk (CHD equivalent) group(men-32.8%,women19.5%).In men 35.9% were in moderately high risk compared to 11.1% in women.50% of women were in moderate risk group compared to 10.7% in men .(Table 13)

Multiple(2+) risk factors were present in 84.4% men and 73.6% women(Table14).Inspite of only a small difference between the presence of multiple(2+) risk factors in both sexes, moderate risk group(50%) was the commonest risk group in women compared to high risk (32.8%)and moderately high risk(35.9%) being commoner in men.(Table 13)

This can be attributed to the fact that hypertriglyceridemia and low HDL cholesterol which are part of atherogenic dyslipidemia was significantly more in men and smoking was an additional risk factor present only in men(Table9,19)

Diabetes mellitus was the important risk factor contributing to high risk(CHD equivalent) in women since ATPIII considers Diabetes as CHD equivalent.

There was a statistically significant difference in 10 yr CHD risk scores and risk categorization between men and women.Men being more prone for higher 10 yr CHD risk scores and risk categorization.

This study has highlighted the fact that asymptomatic subjects attending our MASTER HEALTH CHECKUP CLINIC had various grades of risk factors necessitating intensive intervention.

It is recommended that evaluation of coronary heart disease risk factors utilizing (a) Multiple risk factor analysis (b) Framingham 10 year CHD risk score and (c) Risk categorization according to ATP III guidelines be done in individuals for primary prevention of coronary heart disease.

SUMMARY

- 1.Total no of subjects-200. Men-128(64%),women-72(36%)
- 2.The overall major risk factors were
 - (a) physical inactivity(97.5%),(men-96.1%,women-100%)
 - (b)Dyslipidemia
 - HDL-C<40mg/dl-57%, (men-63.3%,women-45.8%)
 - LDL-C≥130mg/dl-54.5%(men-54.7%,women-54.2%)
 - Total cholesterol≥200mg/dl-51%(men-52.3%,women-48.6%)
 - TGL≥150 mg/dl-32.5%(men-37.5%,women-23.6%)
 - (c)Truncal obesity-53.5%(men-50%,women-55.1%)
 - (d) Dysglycemia(IFG/IGT)-30.5%(men-33.6%,women-25%)
 - (e) Diabetes mellitus-15.5% (men-15.6%,women-15.3%)
 - (f) Abdominal obesity- 48%(men-39.1%,women-63.9%)
 - (g)Overweight-30%(men-28.9%,women-31.9%)
 - (h) obesity-12%(men-9.4%,women-16.7%)
 - (h)Smoking-26% (only in men)
 - (i)Family H/o premature CHD was rare-4%(men-3.9%,women-4.2%)
- 3.Metabolic syndrome-39%(men-32.8%,women-50%) and abdominal obesity significantly more in women .
- 4.Hypertriglyceridemia and low HDL significantly more in men
- 5.Multiple risk factors modifying LDL goal (2+ risk factors) present in

84.4% men, 73.6% women. Two risk factors(32.1% men,41.6% women)
three risk factors (39.8% men , 29.2% women) , four risk factor
combination (12.5% men , 2.8% women)

6.Framingham 10 year CHD risk score

<10% 10 yr CHD risk-men 32.8%,women 88.9%

10-20% 10 yr CHD risk-men 46.9%,women 11.1%

>20% 10 yr CHD risk-men 20.3%,women 0%

7.Risk categorization

Low risk(0-1 risk factor)-men 10.9%,women 19.4%

Moderate risk(2 or more risk factors+ 10 yr risk<10%)-men 10.7%,

Women 50%

Moderately High risk(2 or more risk factors+10 yr risk 10-20%)

Men-35.9%

Women-11.1%

High risk(CHD or CHD risk equivalents/10 year risk>20%)

Men-32.8%

Women-19.5%

CONCLUSION

- I. Evaluation of coronary heart disease risk factors utilizing (a) Multiple risk factor analysis (b) Framingham 10 year CHD risk score (c) Risk categorization according to NCEP-ATP III guidelines was valuable.

It is essential to utilize all these parameters to evaluate coronary heart disease risk factors.

- II. This study has highlighted that asymptomatic subjects attending MASTER HEALTH CHECKUP CLINIC had various grades of risk factors which required intensive intervention for risk modification utilizing NCEP-ATP III guidelines. This would be very valuable to select asymptomatic high risk individuals for intensive management. It is also essential that moderate and moderately high risk groups be also targeted for risk management.

- III. Metabolic syndrome was present in a significant number of subjects. Evaluation for metabolic syndrome targets a set of multiple risk factor groups who might have high or moderate to moderately high risk for CHD which needs intensive management for risk modification.

BIBLIOGRAPHY

- (1) Kannel W.B, Gordan.T.; Evaluation of cardiovascular risk: The Framingham Study, *Bull N.Y.Acad Med* 1978;54;573-591
- (2) Murray C.J.L, Lopez A.D; Alternative projection of mortality and morbidity by cause 1990-2020; Global Burden of disease study; *Lancet* 1997;349;1498-1504
- (3) Beaglehole R, Saraci et al; Cardiovascular diseases; causes, surveillance and prevention; *Int.J.Epidemiol* 2001;30;1-4
- (4) Reddy K S, Yusuf S; Emerging epidemic of cardiovascular disease in developing countries; *Circulation* 1998;97;596-601
- (5) Lopez A.D; Assessing the burden of mortality from cardiovascular diseases *World health Stat Q* 1993;46;91-96
- (6) Reddy K.S.; Cardiovascular disease in india; *WHO Stat Q* 1993;46;101-107
- (7) . Gupta R, Gupta VP. Meta-analysis of coronary heart disease prevalence in India. *Indian Heart J* 1996; 48: 241–245
- (8) Gupta R, Singhal S. Coronary heart disease in india (Letter) *Circulation* 1997; 96;3785
- (9) Mohan V, Deepa R, Rani SS, Premalatha G. Prevalence of coronary artery disease and its relationship to lipids in a selected population in South India: The Chennai Urban Population Study. *J Am Coll Cardiol* 2001; 38: 682–687
- (10) Danaraj T J, Acker M S et al; Ethnic group differences in CHD Singapore ; an analysis of necropsy records; *Am Heart J* 1959;58;516-526
- (11) Shaper AG et al; Serum cholesterol, diet and coronary heart disease in africans and asians in uganda; *Lancet* 1959;2;534-537
- (12) Chen AJ; Recent trends in the mortality and morbidity of cardiovascular diseases; *Ann.Acad Med Singapore* 1980;9;411-415

(13)Wyndham L H;Trends with time of cardiovascular mortality in the populations of the RSA for the period 1968-1977.S.Afr.Med J 1982;61;987-993

(14)Trumelitito J et al ;Cardiovascular diseases in Fiji;analysis of mortality,morbidity and risk factors;Bull World Health organ 1984;62;133-143

(15)Connolly DC;Coronary heart disease in residents of rochester,Minnesota 1950-1975,effect of hypertension and its treatment on survival of patients with CAD Mayo clinic Proc 1983;58;248

(16)Assman G et al-The emergence of triglycerides as asignificant independent risk factor for CAD Eur.Heart J 1998;19(suppl)M8-14

(17)Anderson K M,castelli WP et al;Cholesterol and mortality,30 years of followup from the Framingham study;JAMA 1987;257(16);2176-2180

(18)Pekkanen J et al;Changes in serum cholesterol level and mortality;A 30 year followup.The Finnish cohorts of the seven countries study ;Am J Epidemiol1994;139(2);155-165

(19) Austin MA, King M-C, Vranizan KM, Krauss RM.
Atherogenic lipoprotein phenotype: a proposed genetic marker for coronary heart disease risk. *Circulation* 1990;82:495-506.

(20)Barringer et al;WOSCOPS;West of Scotland Coronary prevention Group Lancet 1997;349(9049);432-433

(21)Design,rational and baseline characteristics of the Prospective Pravastatin Pooling Project-Combined analysis of three large scale randomized trials LIPID,CARE,WOSCOPS Am J Cardiol 1995;76(12) 899-905

(22)Reddy SS .Health outcomes in Type 2 Diabetes Int.J.Clin Pract Suppl 2000;113;46-53

(23)Kassab E et al Vascular complications in Diabetes and their prevention Vasc med 2001;6(4);249-255

(24)Cyun DA;Diabetes and coronary heart disease; a time for action;Crit care Nurse 2001;21(1);10,12

- (25) Dans TM et al Relationship between ethnicity and glycemic control, lipid profile and BP during first 9 years of type2DM;UKPDS55 Diabetes Care 2001;24(7);1167-1174
- (26)Shaper et al;Risk factors for ischemic heart disease;the prospective phase of the british regional heart study J Epedemiol Community Health 1985;39(3);197-209
- (27)Bartel M;Health effects of tobacco use and exposure Monaldi Arch Chest Dis 2001;56(6);545-554
- (28)Tsuchiya et al;Smoking a single cigarette rapidly reduces combined concentrations of nitrate and nitrite and concentration of antioxidants in plasma circulation 2002;105(10);1155-1157
- (29)Zevin et al ;Cardiovascular effects of carbon monoxide and cigarette smoking J Am Coll.Cardiol 2001;38(6);1633-1638
- (30)Grassi et al Effects of cigarette smoking on systemic hemodynamics Contrib Nephrol 2000;130;21-30
- (31)Dirican et al; Effects of smoking on serum lipid and lipoprotein concentrations and lecithin:cholesterol acyltransferase activity; J Med Invest 1999;46(3-4);169-172
- (32) Sowers et al;Obesity,hypertension and vascular disease ;Curr Hypertens Rep 1999;1(2);140-144
- (33)Beer-Borst S et al;Obesity and other health determinants across Europe;The EURALIM project. J Epedemiol Community Health 2000;54(6);424-430
- (34)Gray RS et al Relation of generalized and central obesity to cardiovascular risk factors and prevalent coronary heart disease in a sample of american indians:the strong Heart study.Int J Obes Relat Metab Disord 2000;24(7);849-860
- (35)Lakka et al;Abdominal obesity is associated with increased risk of acute coronary events in men Eur Heart J 2002;23(9);706-713
- (36)Rossouw et al;Hormones,genetic factors,and gender differences in cardiovascular disease.Cardovasc Res 2002;53(3);550-557
- (37)De Kleijn MJ et al; Endogenous estrogen exposure and cardiovascular mortality risk in post-menopausal women Am J Epedemiol 2002;155(4);339-345

(38)post menopausla women and cardiovascular risk;impact of HRT *Cardiol rev* 2002;10(1);51-60

(39)Janus ED et al ;The modernization of Asia; Implications for CHD ;*Circulation*;1996;94;2671-2673

(40)Mckeigue PM et al; Association of early onset CHD in south asian men with glucose intolerance and hyperinsulinemia;*Circulation* 1993;87;152-161

(41)Enas A enas ;Why is there an epidemic of malignant CAD in young indians? *Asian journal of clinical cardiology* 1998 Vol 1,No 1

(42)Berlin JA et al; A meta analysis of physical activity in the prevention of coronary artery disease.*Am J Epidemiol* 1990;132;612-628

(43) Menotti A et al;food intake patterns and 25 year mortality from CHD;The seven countries study research group;*Eur J Epedemiol* 1999;15(6);507-515

(44)Marchioli R et al;Efficacy of n-3 polyunsaturated fatty acids after myocardial reinfarction;results of GISSI-prevenzione trial *Lipids* 2001;36;S119-126

(45)Tennant C et al;The impact of emotions on CHD risk;*J Cardiovasc Risk* 2001;8(30):175-183

(46)Pickering TG.Mental stress as a causal factor in the development of hypertension and cardiovascular disease.*Curr Hypertens Rep* 2001;3(3):249-254

(47)Jiang W et al;Depression and heart disease;evidence of a link,and its therapeutic implications.*CNS Drugs* 2002;16(2):111-127

(48)Reed DM et al:Occupational strain and the incidence of coronary heart disease.*Am J Epedemiol* 1989;129(3):495-502

(49) National Cholesterol Education Program. Detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *Circulation* 2002; 106: 3143–3421

(50) Lemieux I et al Hypertriglyceridemic waist: a marker of the atherogenic metabolic triad (hyperinsulinemia; hyperapolipoprotein B; small, dense LDL) in men? *Circulation* 2000;102:179-84.

(51) Park YW et al The Metabolic Syndrome; prevalence and associated risk factors found in US persons from the national health and nutritional examination survey 1980-1994. *Arch Intern Med* 2003;163:427-436

(52) Carr DB, Utzschneider KM, Hull RL, Kodama K, Retzlaff BM, Brunzell JD, Shofer JB, Fish BE, Knopp RH, Kahn SE. Intra-abdominal fat is a major determinant of the National Cholesterol Education Program Adult Treatment Panel III criteria for the metabolic syndrome. *Diabetes*. 2004; 53:2087–2094

(53) . Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes*. 1988;37:1595–1607

(54) Ferrannini E, Haffner SM, Mitchell BD, Stern MP. Hyperinsulinemia: the key feature of a cardiovascular and metabolic syndrome. *Diabetologia*. 1991;34:416–422.

(55) Gustat J, Srinivasan SR, Elkasabany A, Berenson GS. Relation of self-rated measures of physical activity to multiple risk factors of insulin resistance syndrome in young adults: the Bogalusa Heart Study. *J Clin Epidemiol*. 2002;55:997–1006.

(56) Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the Third National Health and Nutrition Examination Survey. *JAMA*. 2002;287:356–359.

(57) Apridonidze T, Essah PA, Iuorno MJ, Nestler JE. Prevalence and characteristics of the metabolic syndrome in women with polycystic ovary syndrome. *J Clin Endocrinol Metab*. 2004;90:1929–1935.

(58) An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement Diagnosis and Management of the Metabolic Syndrome: *Circulation* 2005;112:2735-2752

(59) . Isomaa B, Almgren P, Tuomi T, Forsen B, Lahti K, Nissen M, Taskinen MR, Groop L. Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care*. 2001;24:683–689.

(60). Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, Salonen JT. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *JAMA*. 2002;288:2709–2716.

(61). Sattar N, Gaw A, Scherbakova O, Ford I, O'Reilly DS, Haffner SM,

Isles C, Macfarlane PW, Packard CJ, Cobbe SM, Shepherd J. Metabolic syndrome with and without C-reactive protein as a predictor of coronary heart disease and diabetes in the West of Scotland Coronary Prevention Study. *Circulation*. 2003;108:414–419.

(62). Girman CJ, Rhodes T, Mercuri M, Pyorala K, Kjekshus J, Pedersen TR, Beere PA, Gotto AM, Clearfield M; 4S Group and the AFCAPS/TexCAPS Research Group. The metabolic syndrome and risk of major coronary events in the Scandinavian Simvastatin Survival Study (4S) and the Air Force/Texas Coronary Atherosclerosis Prevention Study (AFCAPS/TexCAPS). *Am J Cardiol*. 2004;93:136–141.

(63). Malik S, Wong ND, Franklin SS, Kamath TV, L'Italien GJ, Pio JR, Williams GR. Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States adults. *Circulation*. 2004;110:1245–1250.

(64). Olijhoek JK, van der Graaf Y, Banga JD, Algra A, Rabelink TJ, Visseren FL; the SMART Study Group. The metabolic syndrome is associated with advanced vascular damage in patients with coronary heart disease, stroke, peripheral arterial disease or abdominal aortic aneurysm. *Eur Heart J*. 2004;25:342–348.

(65). Alexander CM, Landsman PB, Teutsch SM, Haffner SM; Third National Health and Nutrition Examination Survey (NHANES III); National Cholesterol Education Program (NCEP). NCEP-defined metabolic syndrome, diabetes, and prevalence of coronary heart disease among NHANES III participants age 50 years and older. *Diabetes*. 2003;52:1210–1214.

(66) Ninomiya JK, L'Italien G, Criqui MH, Whyte JL, Gamst A, Chen RS. Association of the metabolic syndrome with history of myocardial infarction and stroke in the Third National Health and Nutrition Examination Survey. *Circulation*. 2004;109:42–46.

(67) McNeill AM, Rosamond WD, Girman CJ, Golden SH, Schmidt MI, East HE, Ballantyne CM, Heiss G. The metabolic syndrome and 11-year risk of incident cardiovascular disease in the atherosclerosis risk in communities study. *Diabetes Care*. 2005;28:385–390.

(68) Solymoss BC, Bourassa MG, Lesperance J, Levesque S, Marcil M, Varga S, Campeau L. Incidence and clinical characteristics of the metabolic syndrome in patients with coronary artery disease. *Coron Artery*

Dis. 2003;14:207–212.

(69) Turhan H, Yasar AS, Basar N, Bicer A, Erbay AR, Yetkin E. High prevalence of metabolic syndrome among young women with premature coronary artery disease. *Coron Artery Dis.* 2005;16:37–40.

(70) Wilson PWF, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation* 1998;97:1837-47.

(71) Multiple Risk Factor Intervention Trial Research Group. Multiple Risk Factor Intervention Trial: risk factor changes and mortality results. *JAMA* 1982;248:1465-77.

(72) Assman et al ;Simple scoring scheme for calculating the risk of acute coronary events based on the 10 year follow-up prospective cardiovascular munster(PROCAM) study; *Circulation* 2002;105:310-315

(73) Ness AR et al Fruits and vegetables and CHD; a review; *Int J Epidemiol* 1997;26:1-13

(74) Oones CM et al Fish consumption and CHD risk in Finland, Italy and North America ; *Am J Epidemiol* 2000;151(10);999-1006

(75) Goldstein DJ et al; Beneficial health effects of modest weight loss; *Int J Metab* 1992;16;397-415

(76) Wing RR et al; Change in waist-hip ratio with weight loss and its association with change in cardiovascular risk factors; *Am J Clin Nut*; 1992;55;1086-1092

(77) JNC VI. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Arch Intern Med* 1997;157:2413-46

(78) Furberg CD et al; BP and cardiovascular disease; Evidence based cardiology; *BMJ books* 2001; p231

(79) Guidelines Subcommittee. 1999 WHO-ISH guidelines for management of hypertension; *J Hypertens* 1999;17;151-183

(80)ESH guidelines for management of hypertension;J Hypertens 2003;21;1011-1059

(81)Burr ML et al;Effects of change in fat,fish and fibre intake on death and myocardial reinfarction:Diet and reinfarction Trial(DART);Lancet 1989;2(866)757-761

(82)Randomized trial of cholesterol lowering drugs in 4444 patients with CHD:Scandinavian Simvastatin Survival Study(4S);Lancet;1994;344(934);1383-1389

(83)Rubins HB et al;Gemfibrozil for secondary prevention of CHD in men with low HDL;Veteran affairs trial;Nejm;1999;341(6);410-416

(84)Effects of Atorvastatin on early recurrent ischemic events in acute coronary syndromes;the MIRACL study;a randomized controlled trial JAMA2001;285(13):1711-1718

(85)Lipid Research Clinics Program.The Lipid Research Clinics Coronary Primary Prevention Trials;JAMA1984;251;351-364

(86)Frick MH,Elo O,Haapa K et al.Helsinki Heart Study:primary prevention trial with gemfibrozil in middle-aged men with dyslipidemia;NEJM 1987;317(20);1237-1245

(87)Shepherd J et al :Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia.West of Scotland Prevention Study Group;NEJM 1995;333(20);1301-1307

(88)Nishimura R et al;Mortality trends in type1 diabetes.The Allegheny County(Pennsylvania) Registry.1965-1999.Diabetes Care 2001;24;823-827

(89)Nathan DM et al;The epidemiology of cardiovascular disease in type 2 diabetes mellitus:how sweet it is...or is it?Lancet 1998;350(Suppl1):S14-S18

(90)Stratton IM et al;Association of glycemia with macrovascular and microvascular complications of type2 diabetes(UKPDS 35):prospective observational study.Br Med J 2000;321;405-412

(91) Enas EA,Yusuf S,Mehta JL.Prevalence of coronary artery disease in Asian Indians.Am J Cardiol 1992;70:945-49

(92) Anand SS,Yusuf S,Vuksan V,et al.Differences in risk factors,

atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the study of health assessment and risk in ethnic groups (SHARE). *Lancet* 2000;356:279-84.

(93) Padmavati S, Gupta S, Pantulu GVA. Dietary fats, serum cholesterol levels and incidence of atherosclerosis in Delhi. *Circulation* 1959;19:849.

(94) Ramachandran A, Snehalatha C, Latha E, et al. Clustering of cardiovascular risk factors in urban Asian Indians. *Diabetes Care* 1998;21:967-71.

(95) Gupta R, Gupta VP, Sarna M, Bhatnagar S, Thanvi J, Sharma V, et al. Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch-2. *Indian Heart J* 2002; 54: 59-66

(96) Achari V, Thakur AK. Association of major modifiable risk factors among patients with coronary artery disease - A retrospective study. *J Assoc Physicians India* 2004;52:103-8.

(97) Burman A, Jain K, Gulati R, Chopra V, Agarwal DP, Vasisht S. Lipoprotein (a) as a marker of coronary artery disease and its association with dietary fat. *J Assoc Physicians India* 2004;52:99-102

(98) Rajmohan L, Deepa R, Mohan A, Mohan V. Association between isolated hypercholesterolemia, isolated hypertriglyceridemia and coronary artery disease in South Indian Type 2 diabetic patients. *Indian Heart J* 2000;52:400-6.

(99) McKeigue PM, Miller GJ, Marmot MG. Coronary heart disease in south Asians overseas: review. *J Clin Epidemiol* 1989;42:597-609.

(100) Gama R, Elfatih AB, Anderson NR. Ethnic differences in total and HDL cholesterol concentrations: Caucasians compared with predominantly Punjabi Sikh Indo-Asians. *Ann Clin Biochem* 2002;39 (Pt 6):609-11.

(101) Kulkarni KR, Markovitz JH, Nanda NC, Segrest JP. Increased prevalence of smaller and denser LDL particles in Asian Indians. *Arterioscler Thromb Vasc Biol* 1999;19:2749-55.

- (102) Heart Protection Study Collaborative Group. MRC/BHF Heart Protection study of cholesterol lowering with simvastatin in 20536 high-risk individuals: a randomised placebo-controlled trial. *Lancet* 2002;360:7-21.
- (103) Deepa R, Arvind K, Mohan V. Diabetes and risk factors for coronary artery disease. *Current Science* 2002;83:1497-1505
- (104) Bhatnagar D, Anand IS, Durrington PN, et al. Coronary risk factors in people from the Indian subcontinent living in west London and their siblings in India. *Lancet* 1995;345:405-9.
- (105) Enas EA. Lipoprotein (a) is an important genetic risk factor for coronary artery disease in Asian Indians. *Am J Cardiol* 2001;88:201-2.
- (106) Mohan V, Deepa R, Haranath S, et al. Lipoprotein(a) is an independent risk factor for coronary artery disease in NIDDM patients in South India. *Diabetes Care* 1998;21:1819-23.
- (107) National High Blood pressure Education program. The Seventh report of the Joint National committee on prevention, detection, evaluation, and treatment of High blood pressure: JNC VII: Hypertension 2003;42:1206-1252
- (108) Obesity: Preventing and Managing the Global Epidemic. WHO, 1998
- (109) Cannon CP, Battler A, Brindis RG, Cox JL, Ellis SG, Every NR, et al. American College of Cardiology key data elements and definitions for measuring the clinical management and outcomes of patients with acute coronary syndromes. A report of the American College of Cardiology Task Force on Clinical Data Standards (Acute Coronary Syndromes Writing Committee). *J Am Coll Cardiol* 2001; 38: 2114–2130
- (110) The Expert Committee on the Diagnosis and classification of Diabetes Mellitus; *Diabetes Care* 2003;26:3160-3167
- (111) Friedwald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. *Clin Chem* 1972; 18:499-502.

(112) Beaglehole R. Global cardiovascular disease prevention: time to get serious. *Lancet* 2001; 358: 661–663

(113) Grundy SM, Balady GJ, Criqui MH, Fletcher G, Greenland P, Hiratzka LF, et al. Primary prevention of coronary heart disease: guidance from Framingham: a statement for health care professionals from the AHA Task Force on Risk Reduction. American Heart Association. *Circulation* 1998; 97: 1876–1887

(114) Prevalence and risk factors of hypertension in a selected South Indian Population-The Chennai Urban Population Study(CUPS); *JAPI* 2003;51:20-27

(115) Chaturvedi V, Prabhakaran D et al: Cardiovascular risk associated with impaired fasting glucose in urban North India ; *Diabetologia* 2003;46Suppl.2;A142

PROFORMA

NAME:

IP NO:

AGE:

ADDRESS:

SEX:

OCCUPATION

EDUCATION:

EARNING:

PAST HISTORY

1. Hypertension
2. Diabetes mellitus
3. coronary heart disease
4. other illness

PERSONAL HISTORY:

1. Smoking No of cigarettes /day: Duration in years:
2. Alcohol
3. Physical Activity

FAMILY HISTORY OF PREMATURE CHD :

ANTHROPOMETRIC MEASUREMENTS:

- 1.WEIGHT(in Kg) :
- 2.HEIGHT(in cm) :
- 3.BMI(Wt/Ht^2) :
- 4.WAIST CIRCUMFERENCE(in cm) :

5.HIP CIRCUMFERENCE(in cm) :

6.WAIST/HIP RATIO :

CLINICAL EXAMINATION:

Pulse rate :

Blood pressure :

JVP:

CVS:

RS:

ABDOMEN:

CNS:

INVESTIGATION:

1.URINALYSIS:

a.Albumin:

b.Sugar :

2.BLOOD CHEMISTRY:

a.Glucose(mg/dl): FBS

PPBS

b.Urea (mg/dl) :

c. Creatinine(mg/dl) :

d.Lipid Profile :

Total Cholesterol

HDL-C

TGL

VLDL-C

LDL-C

3. 12 LEAD ECG:

4. ECHOCARDIOGRAPHY:

5. ULTRASONOGRAM ABDOMEN:

6. FRAMINGHAM RISK SCORE(10 Yr CHD RISK) :

ANNEXURE-1

Estimate of 10-Year CHD Risk for Men (Framingham Point Scores)

<u>AGE, Yr</u>	<u>POINTS</u>
20-34	-9
35-39	-4
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	11
70-74	12
75-79	13

<u>TOTAL CHOLESTEROL</u>	<u>20-39 Yr</u>	<u>40-49 Yr</u>	<u>50-59 Yr</u>	<u>60-69 Yr</u>	<u>70-79 Yr</u>
<160	0	0	0	0	0
169-199	4	3	2	1	0
200-239	7	5	3	1	0
240-279	9	6	4	2	1
≥280	11	8	5	3	1

<u>SMOKER</u>	<u>20-39 Yr</u>	<u>40-49 Yr</u>	<u>50-59 Yr</u>	<u>60-69 Yr</u>	<u>70-79 Yr</u>
NO	0	0	0	0	0
YES	8	5	3	1	1

<u>HDL cholesterol, mg/dl</u>	<u>POINTS</u>
<40	2
40-49	1
50-59	0
≥60	-1

<u>Systolic blood pressure, mm Hg</u>	<u>If Untreated</u>	<u>If treated</u>
<120	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥160	2	3

Adding up the points

Age _____
T.Cholestorol _____
HDL-C _____
Blood Pressure _____
Smoker _____
Total Points _____

Point Total	10-Year Risk	Point Total	10-Year Risk
<0	<1%	11	8%
0	1%	12	10%
1	1%	13	12%
2	1%	14	16%
3	1%	15	20%
4	1%	16	25%
5	2%	≥17	≥30%
6	2%		
7	3%		
8	4%		
9	5%		
10	6%		

ANNEXURE-2

Estimate of 10-Year CHD Risk for women (Framingham Point Scores)

<u>AGE, Yr</u>	<u>POINTS</u>
20-34	-7
35-39	-3
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	12
70-74	14
75-79	16

<u>T CHOLESTEROL</u>	<u>20-39 Yr</u>	<u>40-49 Yr</u>	<u>50-59 Yr</u>	<u>60-69 Yr</u>	<u>70-79 Yr</u>
<160	0	0	0	0	0
160-199	4	3	2	1	1
200-239	8	6	4	2	1
240-279	11	8	5	3	2
>280	13	10	7	4	2

<u>SMOKER</u>	<u>20-39 Yr</u>	<u>40-49 Yr</u>	<u>50-59 Yr</u>	<u>60-69 Yr</u>	<u>70-79 Yr</u>
NO	0	0	0	0	0
YES	9	7	4	2	1

<u>HDL cholesterol, mg/dl</u>	<u>POINTS</u>
<40	2
40-49	1
50-59	0
≥60	-1

<u>Systolic blood pressure, mm Hg</u>	<u>If Untreated</u>	<u>If treated</u>
<120	0	0
120-129	1	3
130-139	2	4
140-159	3	5
≥160	4	6

Adding up the points

Age _____
T.Cholestorol _____
HDL-C _____
Blood Pressure _____
Smoker _____
Total Points _____

Point Total	10-Year Risk	Point Total	10-Year Risk
<9	<1%	20	11%
9	1%	21	14%
10	1%	22	17%
11	1%	23	22%
12	1%	24	27%
13	2%	≥25	≥30%
14	2%		
15	3%		
16	4%		
17	5%		
18	6%		
19	8%		

s.no	NAME	AGE	SEX	MHC NO	MARI	P/H CAD	P/H DM	P/H HTN	F/Hprechd	H/Oexer	H/Osmok	
1	thangavelu	50		1	1950	2	0	0	0	0	0	1
2	rathinam	57		1	1953	2	0	0	0	0	0	0
3	masthanana	55		2	2320	2	0	0	0	0	0	0
4	Kaniamal	56		2	2330	2	0	0	0	0	0	0
5	murugesha	45		1	2361	2	0	0	0	0	0	2
6	Ansari	63		1	2350	2	0	0	0	0	0	2
7	ranjitham	57		2	2400	2	0	0	0	0	0	0
8	rajendran	46		1	2410	2	0	0	0	0	0	0
9	badusha	45		1	2430	2	0	0	0	0	0	1
10	kanniapan	59		1	2429	2	0	0	0	0	0	0
11	inbavalli	55		2	2431	2	0	0	0	0	0	0
12	subramani	55		1	2446	2	0	0	0	0	0	0
13	veerapathr	60		1	2462	2	0	0	0	0	0	1
14	ashraf	45		1	2475	2	0	0	0	0	0	0
15	abdul kar	45		1	2491	2	0	0	0	0	0	1
16	gnanamoo	55		1	2535	2	0	0	0	0	0	0
17	Jamuna	65		2	2536	2	0	0	0	0	0	0
18	jeychand	50		1	2544	2	0	0	0	3	0	1
19	bose	56		1	2540	2	0	0	0	0	0	0
20	suseela	55		2	2559	2	0	0	0	0	0	0
21	chandra	60		2	2564	2	0	0	0	0	0	0
22	ravi	48		1	2566	2	0	0	0	0	0	1
23	bala	65		1	2562	2	0	0	0	0	0	1
24	saroja	55		2	2563	2	0	0	0	0	0	0
25	amaravaty	55		2	2584	2	0	0	0	0	0	0
26	jeyaraj	45		1	2585	2	0	0	0	0	0	0
27	karunakar	48		1	2596	2	0	0	0	0	0	1
28	meena	56		2	2588	2	0	0	0	1	0	0
29	selvaraj	62		1	2594	2	0	0	0	0	0	0
30	pushpa	80		2	2596	3	0	0	0	0	0	0
31	neelavaty	55		2	2601	2	0	0	0	0	0	0
32	ranganath	62		1	2603	2	0	0	0	0	0	1
33	muniamal	55		2	2605	2	0	0	0	0	0	0
34	prema	55		2	2658	2	0	0	0	0	0	0
35	Dasaratha	45		1	2660	2	0	0	0	0	0	2
36	abdul jaba	54		1	2661	2	0	0	0	0	0	0
37	patchiapa	65		1	2683	2	0	0	0	0	0	1
38	nathan	45		1	2618	2	0	0	0	0	0	0
39	saroja	58		2	2706	2	0	0	0	0	0	0
40	chandra	60		1	2709	2	0	0	0	0	0	0
41	sowdham	55		2	2710	2	0	0	0	0	0	0
42	arumugam	49		1	2737	2	0	0	0	0	0	1
43	jeyaraman	55		1	2735	2	0	0	0	0	0	1
44	Krishnan	49		1	2736	2	0	0	0	0	0	1
45	vaduvamb	60		2	2721	2	0	0	0	0	0	0
46	thiyagaraj	46		1	2734	2	0	0	0	0	0	1
47	baktha	60		1	2738	2	0	0	0	0	0	1
48	prema	56		2	2760	2	0	0	0	0	0	0
49	rajan	46		1	2761	2	0	0	0	0	0	1
50	hema	55		2	2766	2	0	0	0	0	0	0

s.no	NAME	AGE	SEX	MHC NO	MARI	P/H CAD	P/H DM	P/H HTN	F/Hprehd	H/Oexer	H/Osmok
51	gandimath	60	2	2763	2	0	0	0	0	0	0
52	selvam	46	1	2762	2	0	0	0	0	0	0
53	maria	56	2	2768	2	0	0	0	0	0	0
54	nageen	51	1	2785	2	0	0	0	0	0	1
55	jeyalaksmi	64	2	2792	2	0	0	0	0	0	0
56	jabbar	45	1	2833	2	0	0	0	0	0	0
57	ramalingam	46	1	2847	2	0	0	0	0	0	0
58	anbalagan	62	1	2835	2	0	0	0	0	0	1
59	rani	59	2	2830	2	0	0	0	0	0	0
60	thomas	58	1	2858	2	0	0	0	0	0	0
61	lily rani	55	2	2874	2	0	0	0	0	0	0
62	kanthamal	55	2	2875	2	0	0	0	0	0	0
63	murali	45	1	2872	2	0	0	0	0	0	0
64	mustafa	46	1	2863	2	0	0	0	0	0	0
65	ahmed	58	1	2862	2	0	0	0	0	0	0
66	durairaj	45	1	2864	2	0	0	0	0	0	0
67	udaykumr	55	1	2865	2	0	0	0	0	1	0
68	PRASAD	54	1	2872	2	0	0	0	0	0	1
69	komala	65	2	2899	2	0	0	0	0	0	0
70	kuppusami	62	1	2892	2	0	0	0	0	0	1
71	mokamal	55	2	2904	2	0	0	0	0	0	0
72	antoniama	55	2	2911	2	0	0	0	0	0	0
73	madhav	45	1	2912	2	0	0	0	0	0	0
74	kumaran	46	1	2918	2	0	0	0	0	0	0
75	narayan	45	1	2919	2	0	0	0	0	0	1
76	roger	46	1	2920	2	0	0	0	0	0	1
77	anippa	51	1	2922	2	0	0	0	0	0	0
78	baskaran	45	1	2924	2	0	0	0	0	0	0
79	jaikumar	45	1	2930	2	0	0	0	0	0	1
80	thangavelu	56	1	2932	2	0	0	0	0	0	0
81	subramani	47	1	2930	2	0	0	0	0	0	1
82	thirunavu	45	1	2921	2	0	0	0	0	0	2
83	chellaperu	49	1	2933	2	0	0	0	0	0	1
84	sivaji	51	1	2934	2	0	0	0	0	0	0
85	poogavnm	60	1	2923	2	0	0	0	0	0	2
86	abibullah	68	1	2900	2	0	0	0	0	0	0
87	ramakrisna	45	1	2902	2	0	0	0	0	0	0
88	periasamy	46	1	2901	2	0	0	0	0	0	0
89	arunachal	56	1	2903	2	0	0	0	0	0	1
90	venkataiha	61	1	2904	2	0	0	0	0	0	1
91	dhamlingam	45	1	2906	2	0	0	0	0	0	0
92	jeychandr	49	1	2942	2	0	0	0	0	0	0
93	manorama	56	2	2936	2	0	0	0	0	0	0
94	sekar	45	1	2960	2	0	0	0	0	1	0
95	govindan	57	1	2961	2	0	0	0	0	0	2
96	raja	45	1	2935	2	0	0	0	0	0	1
97	gunasekar	47	1	2937	2	0	0	0	0	0	1
98	sankreswari	59	2	2938	2	0	0	0	1,2	0	0
99	thangal	65	1	2943	2	0	0	0	0	0	0
100	vijaybala	55	2	2945	2	0	0	0	0	1	0

s.no	NAME	AGE	SEX	MHC NO	MARI	P/H CAD	P/H DM	P/H HTN	F/Hprechd	H/Oexer	H/Osmok	
101	ahmed	56	1	620		2	0	0	0	0	0	1
102	ahmed jan	68	1	630		2	0	0	0	0	0	1
103	allimuthu	46	1	117		2	0	0	0	0	0	1
104	andal	64	2	302		3	0	0	0	0	0	0
105	annaporni	58	2	294		2	0	0	0	0	0	0
106	babu	45	1	613		2	0	0	0	0	0	1
107	baby	55	2	451		2	0	0	0	0	0	0
108	balakrisnan	52	1	561		2	0	0	0	0	0	0
109	chandrasedr	63	1	516		2	0	0	0	0	0	1
110	chinasamy	67	1	493		2	0	0	0	0	0	1
111	david	75	1	265		2	0	0	0	0	0	0
112	dhanalaxmi	71	2	390		2	0	0	0	0	0	0
113	dillibabu	50	1	154		2	0	0	0	0	0	1
114	egavalli	55	2	540		2	0	0	0	0	0	0
115	farook	55	1	285		2	0	0	0	0	0	1
116	goovardan	53	1	565		2	0	0	0	0	0	0
117	govindamal	56	2	1917		2	0	0	0	0	0	0
118	govindan	55	1	25671		2	0	0	0	0	0	1
119	gowri	56	2	683		2	0	0	0	0	0	0
120	inbavalli	57	2	544		2	0	0	0	0	0	0
121	jamuna	55	2	589		2	0	0	0	0	0	0
122	jarina	57	2	660		2	0	0	0	0	0	0
123	jayalaxmi	60	2	548		2	0	0	0	0	0	0
124	jeyalaksmi	62	2	195		2	0	0	0	0	0	0
125	kaliappan	56	1	438		2	0	0	0	0	0	0
126	kantha	73	2	464		2	0	0	0	0	0	0
127	kasinathan	70	1	489		2	0	0	0	0	0	0
128	kokilam	56	2	376		2	0	0	0	0	0	0
129	kumari	55	2	693		2	0	0	0	0	0	0
130	kupammal	56	2	223		2	0	0	0	0	0	0
131	manikam	78	1	553		3	0	0	0	0	0	0
132	marianayagam	45	1	173		2	0	0	0	0	1	0
133	mathew	56	1	23224		2	0	0	0	0	1	1
134	meenakshi	60	2	399		2	0	0	0	0	0	0
135	muniamal	56	2	312		2	0	0	0	0	0	0
136	murugandi	48	1	39612		1	0	0	0	0	0	0
137	padma	55	2	410		2	0	0	0	0	0	0
138	padmavathy	56	2	184		2	0	0	0	0	0	0
139	panjavarnam	62	2	520		2	0	0	0	0	0	0
140	paramasivam	46	1	229		2	0	0	0	0	0	0
141	patchiamal	58	2	619		2	0	0	0	0	0	0
142	patchiapan	71	1	391		2	0	0	0	1	0	0
143	poosam	55	2	654		2	0	0	0	0	0	0
144	pushpa	56	2	618		2	0	0	0	0	0	0
145	rajakannu	53	1	24313		2	0	0	0	0	0	0
146	rajamani	48	1	404		2	0	0	0	0	0	0
147	ramayee	56	2	216		2	0	0	0	0	0	0
148	ravishankar	45	1	453		2	0	0	0	0	0	0
149	renuka	58	2	470		2	0	0	0	0	0	0
150	sakuntala	62	2	288		2	0	0	0	0	0	0

s.no	NAME	AGE	SEX	MHC NO	MARI	P/H CAD	P/H DM	P/H HTN	F/Hprehd	H/Oexer	H/Osmok
151	alikhhan	45	1	356	2	0	0	0	0	0	0
152	balasubramani	46	1	452	2	0	0	0	0	0	1
153	dinesh	46	1	450	2	0	0	0	0	0	0
154	ravi	47	1	456	2	0	0	0	0	0	1
155	narayan	45	1	567	2	0	0	0	0	0	1
156	krishnan	55	1	555	2	0	0	0	0	0	0
157	babu	45	1	568	2	0	0	0	0	0	1
158	chandra	55	2	510	2	0	0	0	0	0	0
159	muthu	50	1	579	2	0	0	0	0	0	0
160	meenatchi	60	1	580	2	0	0	0	0	1	0
161	trevor	56	1	581	2	0	0	0	1	1	2
162	kutalachangu	55	1	604	2	0	0	0	0	0	0
163	vedachalam	45	1	630	2	0	0	0	0	0	1
164	samutrapandian	45	1	616	2	0	0	0	2	0	0
165	chandrasekr	46	1	626	2	0	0	0	0	0	0
166	pakkirisami	48	1	636	2	0	0	0	0	0	1
167	vasu	49	1	635	2	0	0	0	0	0	1
168	boopathy	65	2	648	2	0	0	0	0	0	0
169	anbu	57	1	649	2	0	0	0	0	0	2
170	ravindran	45	1	650	2	0	0	0	0	0	0
171	jeevanandam	45	1	651	2	0	0	0	0	0	0
172	sargurudas	58	1	655	2	0	0	0	0	0	0
173	aliakbar	48	1	654	2	0	0	0	0	0	1
174	kumari	56	2	657	2	0	0	0	0	0	0
175	indirani	55	2	652	2	0	0	0	0	0	0
176	sudamani	55	1	687	2	0	0	0	0	0	0
177	shenbagavalli	55	2	658	2	0	0	0	0	0	0
178	anandan	45	1	660	2	0	0	0	0	0	0
179	munikrisnan	58	1	662	2	0	0	0	0	0	0
180	rani	55	2	664	2	0	0	0	0	0	0
181	mahendran	45	1	665	2	0	0	0	0	0	0
182	fatimabee	56	2	667	2	0	0	0	0	0	0
183	sankaran	45	1	669	2	0	0	0	0	0	0
184	lakshman	51	1	671	2	0	0	0	0	0	1
185	ajeedadoss	45	1	673	2	0	0	0	0	0	2
186	vijaya	56	2	675	2	0	0	0	0	0	0
187	diilibabu	69	1	677	2	0	0	0	0	0	1
188	saraswathy	58	2	688	2	0	0	0	0	0	0
189	sarojini	55	2	689	2	0	0	0	0	0	0
190	shanthi	56	2	690	2	0	0	0	0	0	0
191	sivagami	56	2	691	2	0	0	0	0	0	0
192	subbiah	62	1	692	2	0	0	0	0	0	1
193	subramani	45	1	693	2	0	0	0	0	0	1
194	kabali	48	1	694	2	0	0	0	0	0	0
195	sriram	46	1	695	2	0	0	0	0	0	1
196	sundaram	55	1	697	2	0	0	0	0	0	0
197	krishnan	50	1	698	2	0	0	0	0	0	0
198	valli	58	2	699	2	0	0	0	0	0	0
199	sekar	45	1	700	2	0	0	0	0	0	2
200	vasanti	58	2	702	2	0	0	0	0	0	0

s.no	BPAv sys	BPAv dias	BMI	Waist circ	WHR	HR	U albumin	U sugar	FBS	PPBS	B urea	
1	120	80	22	85	1.1	74	0	1	217	415	16	
2	120	80	25.4	78	0.9	82	0	0	83	102	26	
3	180	100	22.5	91	0.88	78	0	0	92	112	23	
4	130	80	20	75	0.8	76	0	0	90	120	22	
5	130	80	19.2	76	0.9	75	0	0	67	98	15	
6	140	90	20.8	80	0.9	78	0	0	63	111	18	
7	110	80	17	65	0.76	74	0	0	77	89	18	
8	110	80	18.5	72.5	0.9	76	0	0	50	74	18	
9	120	80	16.8	85	0.9	72	0	0	99	135	25	
10	140	100	21.75	90	0.96	76	0	0	116	132	25	
11	140	90	30.4	91.5	0.8	74	0	0	110	149	33	
12	130	80	29.2	101.5	1	78	0	0	122	173	16	
13	130	80	13.75	67.5	0.8	80	0	0	88	134	18	
14	120	80	21.2	80.5	0.9	78	0	0	72	96	22	
15	110	80	17.3	75	0.9	73	0	0	97	117	16	
16	120	80	17	65.5	0.8	78	0	0	112	150	26	
17	190	100	22.4	88.5	0.9	78	0	0	67	94	53	
18	110	80	21.2	78.5	0.9	76	0	0	105	149	28	
19	130	80	21.2	81.5	0.85	74	0	0	92	149	33	
20	120	80	19.5	74	0.8	72	0	0	86	105	20	
21	130	80	24.4	89.5	0.9	74	0	0	78	125	27	
22	120	80	21.2	82.5	0.9	72	0	0	74	83	19	
23	120	80	21.1	83.5	0.9	74	1	0	96	189	16	
24	130	80	33.2	108	0.8	76	0	0	76	184	20	
25	140	90	20.5	74.5	0.8	72	0	0	70	131	20	
26	120	80	25.2	93	1	76	0	0	53	72	13	
27	130	80	18.75	75.5	0.9	74	0	0	76	117	20	
28	150	90	24.8	93	0.9	78	0	0	79	120	21	
29	90	60	22.6	88.5	0.97	72	0	0	78	112	21	
30	150	90	15.2	62.5	0.8	74	0	0	76	118	21	
31	170	90	25.2	86.5	0.8	72	0	0	86	136	12	
32	130	90	20.3	85.5	0.9	74	0	0	111	242	15	
33	140	90	19.6	73.5	0.8	72	0	2	125	225	26	
34	130	80	29.2	87.5	0.8	76	0	0	117	145	20	
35	130	80	18	91.5	1	72	0	0	103	145	22	
36	120	80	17	66	0.84	76	0	0	61	113	26	
37	140	90	22.7	89	0.9	74	0	1	148	262	22	
38	150	100	29.6	109.5	1	72	0	2	159	316	20	
39	140	90	30.9	82.5	0.74	74	0	2	176	217	18	
40	130	90	27.5	97	0.9	72	0	0	90	120	18	
41	120	80	25.3	79	0.76	76	0	0	80	98	19	
42	140	80	26.9	67	0.8	71	0	0	90	102	24	
43	110	70	17.8	71	0.85	74	0	0	102	173	21	
44	130	90	24.2	89	0.9	76	0	2	173	388	32	
45	120	80	13.3	63	0.84	72	0	0	80	102	23	
46	130	80	29.6	99.5	0.95	76	0	0	82	146	13	
47	120	80	25.3	97	1	71	0	0	56	72	12	
48	170	90	28.7	95	0.9	76	0	0	76	96	12	
49	110	80	24	94.5	0.98	72	0	0	74	112	15	
50	180	120	27.1	91.5	0.9	74	0	0	84	94	23	

s.no	BPAv sys	BPAv dias	BMI	Waist circ	WHR	HR	U albumin	U sugar	FBS	PPBS	B urea	
51	150	90	23.2	82	0.85	71	0	0	0	72	116	18
52	110	70	20.75	84	0.95	74	0	0	0	76	123	25
53	100	60	15	55	0.74	72	0	0	0	86	138	20
54	110	70	18	69.5	0.87	70	0	0	0	86	174	18
55	190	110	35.6	104	0.9	70	0	0	0	86	138	24
56	110	80	21.4	84.5	0.94	75	0	0	0	81	98	22
57	110	80	14.8	58	0.8	72	0	0	0	84	97	23
58	120	80	21.6	88.5	0.97	72	0	0	0	96	154	24
59	120	80	23	79	0.85	71	0	0	0	99	119	15
60	150	90	24.2	84	0.88	70	0	0	0	125	186	24
61	150	100	29.3	90.5	0.84	70	0	0	0	103	182	17
62	110	80	23.1	83	0.88	70	0	0	0	110	230	21
63	130	80	21.1	79	0.9	70	0	0	0	102	166	14
64	130	90	22.9	90	0.94	72	0	0	0	99	120	23
65	110	80	30	105	0.9	74	0	0	0	115	169	23
66	130	80	26.3	84.5	0.8	70	0	0	0	98	139	22
67	140	90	25.2	87	0.9	72	0	0	0	104	114	25
68	110	80	26.4	93	0.96	70	0	0	0	88	112	22
69	160	90	26.6	90.5	0.84	72	0	0	0	126	197	25
70	130	80	30	97.5	0.93	70	0	2	2	112	370	17
71	130	80	27.77	90	0.88	72	0	0	0	112	123	13
72	130	80	18.7	74	0.88	74	0	0	0	71	86	29
73	150	100	28	100	0.96	70	0	0	0	99	133	19
74	120	80	25.22	84.5	0.9	72	0	0	0	96	110	20
75	140	100	22.26	82	0.95	70	0	0	0	104	140	16
76	140	90	26	86	0.85	72	0	0	0	72	164	22
77	150	100	26.9	97	0.96	74	0	0	0	105	145	25
78	160	90	26.6	96	1.1	72	0	0	0	108	144	25
79	140	90	30.2	101	0.97	70	0	2	2	161	282	14
80	110	80	30.1	101	0.93	72	0	0	0	102	144	19
81	140	90	24.5	85.5	0.95	70	0	0	0	90	120	26
82	130	80	27.7	86	0.9	72	0	0	0	76	110	28
83	120	80	22.2	89.5	0.93	72	0	0	0	80	120	17
84	130	80	21.1	84	0.91	70	0	0	0	78	126	19
85	150	90	16.8	71	0.87	74	0	0	0	76	118	15
86	160	90	23.54	96.5	0.96	76	0	0	0	113	147	14
87	130	80	26.8	102.5	1.01	72	0	0	0	109	160	16
88	120	80	22.1	86	0.95	74	0	0	0	101	225	17
89	120	80	22.35	85	0.98	72	0	0	0	97	179	18
90	110	80	21.9	87	0.97	70	0	0	0	84	168	34
91	110	80	21.3	76.5	0.9	74	0	0	0	90	128	22
92	120	80	23	84	0.96	72	0	2	2	134	260	18
93	180	110	35	100	0.86	70	0	0	0	65	85	23
94	120	80	26.4	98.5	0.96	74	0	0	0	76	102	23
95	130	80	21.75	79.5	0.9	74	0	0	0	88	110	25
96	170	110	24.2	91	0.9	72	0	2	2	131	228	19
97	110	80	29.6	99	0.94	70	0	0	0	103	163	15
98	130	80	19.6	79.5	0.83	76	0	2	2	146	198	14
99	130	80	29.6	97	1.1	72	0	0	0	110	216	18
100	150	90	19.6	68	0.8	72	0	0	0	118	168	26

s.no	BPAv sys	BPAv dias	BMI	Waist circ	WHR	HR	U albumin	U sugar	FBS	PPBS	B urea	
101	160	80	22.2	80	0.9	84	0	0	60	88	34	
102	154	100	18.82	81	0.8	74	0	0	64	88	32	
103	128	86	28.65	96	1	82	1	2	190	335	20	
104	180	90	24.11	91	0.9	76	0	0	100	157	17	
105	130	80	22.93	79	0.9	80	0	0	73	118	22	
106	140	90	25.91	81	0.8	82	0	0	96	148	19	
107	167	100	30.22	83	0.8	76	0	0	92	112	34	
108	150	100	27.77	84	0.9	70	0	0	76	99	24	
109	180	110	17.54	69	0.86	84	0	0	98	110	24	
110	130	80	21.5	80	0.85	78	0	0	112	158	28	
111	190	100	22.38	68	0.8	86	0	0	105	118	23	
112	150	83	21.52	80	0.8	74	1	0	90	112	22	
113	158	100	25.14	96	0.91	78	0	0	92	136	32	
114	130	80	30.1	94	0.92	82	0	2	185	242	24	
115	150	90	23.63	84	0.9	80	0	0	58	101	16	
116	158	100	26.7	97	0.92	76	0	2	148	211	24	
117	150	90	29.04	99	0.94	82	0	0	102	153	18	
118	170	100	24.6	85	0.95	76	0	0	96	128	21	
119	150	100	29.74	99	0.93	74	0	0	82	142	21	
120	130	80	22	75	0.72	82	0	0	82	117	28	
121	130	80	22.1	78	0.76	78	0	0	64	92	24	
122	170	110	24.56	85	0.8	76	0	0	80	100	16	
123	170	90	29.28	84	0.82	74	0	0	72	116	24	
124	138	80	24.12	81	0.8	80	0	0	76	188	17	
125	168	90	22.2	78	0.8	82	0	0	98	146	34	
126	170	90	29.09	97	0.91	78	0	0	112	185	26	
127	130	80	22.67	68	0.78	74	0	0	108	146	22	
128	158	89	29.29	83	0.83	78	0	0	86	122	34	
129	180	100	25.39	84	0.9	76	0	0	89	142	19	
130	168	110	30.73	95	0.92	71	0	0	122	154	30	
131	180	100	31.6	92	0.9	78	0	0	102	146	34	
132	130	80	28.95	94	0.86	72	0	0	105	131	36	
133	180	90	30.47	96	1	78	0	0	89	124	22	
134	110	70	29.33	88	0.86	76	0	0	110	168	34	
135	130	80	28.99	72	0.76	72	0	0	82	122	22	
136	180	100	32.84	102	0.9	70	0	0	72	104	22	
137	130	80	24.23	85	0.8	82	0	0	102	145	22	
138	130	80	26.14	81	0.8	78	0	0	103	132	20	
139	140	100	30.17	98	0.91	80	0	0	94	134	36	
140	170	100	34.6	101	1.1	78	0	0	73	100	15	
141	168	100	22.18	76	0.78	82	0	0	88	102	28	
142	110	70	19.47	74	0.8	74	0	0	92	124	26	
143	130	80	20.61	81	0.8	78	0	0	60	84	28	
144	110	70	25.63	77	0.76	82	0	0	80	108	18	
145	170	90	29.66	91	0.84	74	0	0	111	126	16	
146	200	120	30.86	94	0.91	80	0	0	90	147	31	
147	130	80	24.97	83	0.9	76	0	0	124	185	30	
148	190	100	30.48	96	1.1	82	0	0	93	124	26	
149	162	100	30.22	92	0.9	72	0	0	71	86	16	
150	154	90	27.19	84	0.8	78	0	0	64	92	20	

s.no	BPAv sys	BPAv dias	BMI	Waist circ	WHR	HR	U albumin	U sugar	FBS	PPBS	B urea	
151	110	70	23.53	91	0.95	74	0	0	0	100	132	20
152	110	70	18.8	79.5	0.88	72	0	0	0	72	106	23
153	120	80	26.95	94	0.99	78	0	0	0	82	106	26
154	100	70	24.82	94	1	76	0	0	0	98	136	22
155	110	70	21.4	74.5	0.91	72	0	0	0	96	144	24
156	110	70	15.1	67.5	0.96	70	0	0	0	74	93	22
157	150	100	27.6	89	0.94	80	0	0	0	105	125	22
158	130	80	19.4	69	0.85	82	0	1	134	168	20	
159	150	90	24.63	92.5	0.98	78	0	0	0	84	112	24
160	150	90	22.8	90	0.97	78	0	0	0	79	110	20
161	150	100	25.9	101	0.99	76	0	0	0	64	102	30
162	140	90	21.85	85	0.89	74	0	0	0	74	92	28
163	140	100	23.5	83	0.87	70	0	0	0	68	95	25
164	120	80	24.5	90	0.94	72	0	0	0	88	150	22
165	110	70	26.4	96.5	0.99	78	0	0	0	90	253	24
166	140	100	25.48	104	0.98	74	0	2	193	305	22	
167	110	70	17.99	80	0.9	72	0	0	0	85	120	21
168	180	80	21.4	73	0.78	76	0	2	101	206	19	
169	190	120	24.1	88	0.97	78	0	0	0	79	112	25
170	110	70	22.69	86	0.95	76	0	0	0	114	141	18
171	100	70	19.11	75	0.89	78	0	0	0	100	135	19
172	132	80	23.2	97	0.93	72	0	2	250	330	17	
173	150	90	25.78	99	1.05	78	0	0	0	105	171	27
174	130	80	19.48	68	0.83	82	0	0	0	82	116	22
175	100	70	28.1	86	0.86	80	0	0	0	76	118	18
176	130	80	21.13	90	1	78	0	0	0	96	148	22
177	130	80	18.75	74	0.83	72	0	0	0	118	146	24
178	110	70	21.32	82	0.92	76	0	2	99	287	19	
179	110	70	16.6	76	0.97	78	0	0	0	101	184	31
180	110	70	15.83	60.5	0.77	80	0	0	0	82	116	18
181	110	70	21.13	76	0.83	74	0	0	0	67	98	22
182	110	70	26.29	85	0.85	78	0	0	0	69	112	20
183	150	90	26.64	96	0.96	76	0	0	0	98	102	31
184	160	90	22.26	88	0.98	78	0	2	148	256	17	
185	110	70	18.68	78	0.92	76	0	2	120	236	13	
186	110	70	25.45	81	0.84	80	0	2	156	248	17	
187	110	70	18.74	76	0.82	82	0	0	0	71	120	24
188	160	100	22.01	84	0.92	78	0	0	0	128	145	35
189	150	100	30.83	95	1.1	80	0	0	0	86	112	34
190	150	100	16.63	74	0.82	78	0	0	0	92	110	16
191	180	120	25.88	96	0.98	82	0	2	128	293	37	
192	190	100	32.04	101	0.9	80	0	0	0	92	124	29
193	190	100	29.34	89	0.8	82	0	0	0	75	126	17
194	110	70	19.28	78	0.82	78	0	0	0	80	114	20
195	130	80	30.04	91	0.9	80	0	0	0	91	102	22
196	168	100	25.63	80	0.8	82	0	0	0	71	86	15
197	130	80	21.2	84	0.82	78	0	0	0	86	116	18
198	110	70	21.35	78	0.83	84	0	0	0	82	118	16
199	110	79	18.8	78	0.86	80	0	0	0	80	121	20
200	160	90	32.04	101	0.9	76	0	0	0	86	124	23

s.no	S.creat	TC	HDL	TGL	VLDL	LDL	ECG-isch	RWMA	USG	10yr risk
1	0.5	187	40	159	32	115	0	0	0	10%
2	1	241	40	122	24	177	0	0	0	12%
3	0.9	283	38	140	28	217	0	0	0	14%
4	0.9	177	42	81	16	119	0	0	0	2%
5	0.8	207	42	118	24	141	1	0	0	6%
6	0.9	269	37	132	26	206	0	0	0	20%
7	0.9	250	40	139	27	183	0	0	0	2%
8	0.6	176	38	157	31	107	0	0	0	4%
9	0.9	281	32	185	37	212	0	0	0	30%
10	0.8	234	38	171	34	162	0	0	0	16%
11	1	262	42	114	23	197	0	0	0	5%
12	0.5	170	36	183	37	97	0	0	0	12%
13	0.5	141	32	61	12	97	0	0	0	16%
14	0.7	200	38	250	50	112	1	0	0	6%
15	0.5	175	44	98	20	111	0	0	0	10%
16	0.9	200	34	121	24	142	0	0	0	12%
17	0.6	235	41	144	29	165	0	0	0	6%
18	0.9	94	22	85	17	55	1	1	0	8%
19	0.8	137	40	73	15	82	0	0	0	6%
20	0.5	268	38	120	24	206	1	0	0	4%
21	1	212	47	156	31	134	0	0	0	3%
22	0.8	131	28	116	23	80	0	0	0	6%
23	0.9	203	36	110	22	145	0	0	0	20%
24	0.6	152	46	111	22	84	0	0	0	1%
25	1	173	42	71	14	117	0	0	0	2%
26	0.7	239	46	59	12	181	0	0	0	5%
27	0.7	210	40	119	24	136	0	0	0	20%
28	0.7	214	38	139	28	158	1	0	0	5%
29	0.6	197	42	88	18	137	0	0	0	10%
30	0.5	256	40	111	22	194	0	0	0	17%
31	0.6	230	39	156	31	160	0	0	0	11%
32	0.8	217	42	136	27	148	0	0	0	16%
33	0.5	138	46	75	15	77	0	0	0	1%
34	0.7	158	36	158	16	106	0	0	0	1%
35	0.7	250	50	289	58	142	0	0	0	6%
36	0.8	169	46	63	13	110	0	0	0	5%
37	0.6	253	40	100	20	193	0	0	0	25%
38	0.8	122	39	124	26	57	1	0	0	2%
39	0.8	210	42	94	19	149	0	0	0	4%
40	0.8	205	40	103	21	144	0	0	0	12%
41	0.6	192	38	100	20	134	0	0	0	2%
42	0.8	173	45	93	20	108	0	0	0	12%
43	1	184	42	89	18	124	0	0	0	16%
44	1	208	36	87	17	158	1	1	0	25%
45	0.6	115	34	74	15	66	0	0	0	2%
46	0.8	215	36	122	25	154	0	0	0	25%
47	1.1	281	30	131	26	225	0	0	0	25%
48	1	248	48	163	33	167	0	0	0	6%
49	0.9	210	28	298	60	112	0	0	0	20%
50	1	339	40	105	21	278	0	0	0	11%

s.no	S.creat	TC	HDL	TGL	VLDL	LDL	ECG-isch	RWMA	USG	10yr risk
51	0.7	282	42	96	19	221	0	0	0	6%
52	0.7	235	39	71	14	182	0	0	0	6%
53	0.7	210	40	128	26	134	0	0	0	2%
54	0.6	129	32	90	18	79	0	0	1	8%
55	0.8	243	36	135	27	180	0	0	0	14%
56	0.5	214	41	117	23	150	0	0	0	5%
57	0.6	153	39	97	19	95	0	0	0	2%
58	0.7	196	31	155	31	134	0	0	0	16%
59	0.6	217	34	78	16	167	0	0	0	3%
60	0.6	167	27	133	27	113	0	0	0	12%
61	0.8	330	38	135	27	265	1	0	0	11%
62	0.9	202	44	84	17	141	0	0	0	2%
63	0.7	163	49	107	21	93	0	0	0	4%
64	1.1	311	33	151	30	248	0	0	0	16%
65	0.9	250	28	100	20	202	1	1	0	16%
66	0.6	155	40	107	21	94	0	0	0	4%
67	0.8	230	32	174	35	163	0	0	0	16%
68	1.1	143	34	120	24	85	0	0	1	8%
69	0.8	191	36	111	22	133	0	0	1	8%
70	0.6	188	38	96	19	131	0	0	0	20%
71	0.5	326	41	153	51	234	0	0	0	6%
72	0.6	211	53	79	15	143	0	0	0	2%
73	0.8	244	43	86	17	184	0	0	0	8%
74	0.6	175	38	81	16	121	0	0	0	4%
75	0.8	151	45	91	18	88	0	0	0	12%
76	0.9	259	36	151	30	193	0	0	0	30%
77	0.9	174	32	231	46	96	0	0	0	10%
78	1	206	32	304	61	113	0	0	0	10%
79	0.8	210	40	202	41	129	0	0	0	25%
80	1	195	39	154	31	125	0	0	0	10%
81	1	255	38	154	31	186	0	0	0	25%
82	1	256	40	120	24	192	0	0	0	8%
83	0.8	211	36	115	23	152	0	0	0	20%
84	0.9	253	32	206	41	180	0	0	1	12%
85	0.7	153	34	114	23	96	0	0	0	16%
86	0.8	225	39	219	44	142	0	0	0	30%
87	0.8	308	40	189	38	230	1	0	0	12%
88	0.8	241	32	395	79	130	0	0	0	8%
89	0.9	292	36	396	79	177	0	0	0	30%
90	1.2	188	30	200	40	118	0	0	0	16%
91	0.8	237	36	154	31	170	0	0	0	6%
92	0.7	317	42	176	35	230	0	0	0	10%
93	0.9	216	40	165	33	139	0	0	0	5%
94	1	163	38	124	25	100	0	0	0	4%
95	0.9	174	40	131	26	108	0	0	0	10%
96	0.8	217	35	248	50	132	0	0	0	30%
97	0.7	205	38	182	36	131	0	0	1	20%
98	0.8	155	44	182	36	75	0	0	0	1%
99	0.8	326	32	144	29	265	0	0	0	30%
100	0.9	215	42	120	24	149	0	0	0	6%

s.no	S.creat	TC	HDL	TGL	VLDL	LDL	ECG-isch	RWMA	USG	10yr risk
101	1.1	160	40	121	24	96	0	0	0	25%
102	1.3	184	41	126	25	118	0	0	0	25%
103	1.1	230	24	162	32	174	0	0	0	20%
104	0.5	168	35	142	28	105	0	0	0	5%
105	1.2	149	38	89	18	93	0	0	0	2%
106	0.8	162	30	122	24	108	0	0	0	5%
107	0.9	152	36	112	22	94	0	0	0	2%
108	0.8	168	38	101	20	110	0	0	0	8%
109	0.9	164	48	120	24	92	0	0	0	16%
110	0.9	176	32	124	24	120	0	0	0	25%
111	1.2	162	44	119	24	94	0	0	0	25%
112	1	174	36	128	26	112	0	0	0	11%
113	1	186	28	142	28	130	0	0	0	16%
114	0.9	168	45	124	25	98	0	0	0	2%
115	0.9	209	41	99	20	148	0	0	0	25%
116	1.1	162	39	102	20	103	0	0	0	6%
117	0.9	240	21	160	32	187	0	0	0	6%
118	1	202	29	164	33	140	0	0	0	30%
119	1	162	34	132	26	102	0	0	0	3%
120	0.7	172	51	112	22	99	0	0	0	1%
121	0.9	168	37	108	22	109	0	0	0	2%
122	0.8	144	44	103	21	79	0	0	0	2%
123	0.8	136	40	107	21	75	0	0	0	3%
124	1.1	185	58	105	21	106	0	0	0	2%
125	0.8	142	39	92	18	85	0	0	0	10%
126	0.9	202	32	145	29	141	0	0	0	14%
127	1	149	46	102	20	83	0	0	0	16%
128	0.9	165	42	111	22	101	0	0	0	2%
129	0.9	168	30	142	28	110	0	0	0	4%
130	0.9	241	32	168	34	175	0	0	0	8%
131	1	156	34	96	19	103	0	0	0	30%
132	1	178	49	112	22	107	0	0	0	4%
133	0.9	242	34	156	31	177	0	0	0	30%
134	1	182	42	104	21	119	0	0	0	1%
135	0.9	175	40	152	30	105	0	0	0	2%
136	1.2	254	32	198	40	182	0	0	0	12%
137	1	186	34	124	25	127	0	0	0	2%
138	0.8	142	45	102	20	77	0	0	0	1%
139	1.2	163	44	158	32	87	0	0	0	3%
140	0.8	262	33	185	37	192	0	0	0	12%
141	1.1	184	40	124	25	119	0	0	0	3%
142	1	158	35	122	24	99	0	0	0	16%
143	0.6	172	28	136	27	117	0	0	0	2%
144	0.8	130	39	127	25	66	0	0	0	1%
145	1	231	34	168	34	163	0	0	0	12%
146	1.1	194	30	156	31	133	0	0	0	6%
147	1.2	168	42	102	20	106	0	0	0	2%
148	1	201	35	142	28	138	0	0	0	10%
149	0.8	188	28	135	27	133	0	0	0	4%
150	1	239	32	138	28	179	0	0	0	5%

s.no	S.creat	TC	HDL	TGL	VLDL	LDL	ECG-isch	RWMA	USG	10yr risk
151	0.8	169	44	98	20	105	0	0	0	3%
152	0.6	198	43	95	19	136	0	0	0	10%
153	1	212	32	224	45	135	0	0	0	6%
154	0.8	267	42	186	37	188	0	0	0	20%
155	0.8	208	38	154	31	139	0	0	0	20%
156	0.7	198	45	124	25	128	0	0	0	8%
157	0.8	211	33	185	37	141	0	0	0	25%
158	0.7	226	40	163	33	153	0	0	0	3%
159	0.8	172	42	101	20	110	0	0	0	6%
160	0.7	198	44	101	20	134	0	0	0	12%
161	0.9	205	38	174	35	132	0	0	0	16%
162	1.1	221	41	107	21	159	0	0	0	12%
163	0.9	143	46	95	19	78	0	0	0	6%
164	0.9	202	39	146	29	134	0	0	0	8%
165	0.8	315	31	123	25	259	0	0	2	12%
166	0.8	291	35	157	31	225	0	0	0	30%
167	0.5	201	30	218	44	127	0	0	0	20%
168	0.7	193	39	121	24	130	0	0	0	8%
169	0.9	210	42	107	21	147	0	0	0	20%
170	0.7	192	37	149	30	125	0	0	0	4%
171	0.7	134	40	83	17	77	0	0	0	1%
172	0.9	330	38	154	31	261	0	0	0	25%
173	0.7	286	31	252	50	205	0	0	0	30%
174	0.8	220	39	141	28	153	0	0	0	4%
175	0.7	213	45	107	21	147	0	0	0	2%
176	0.7	248	42	143	29	177	0	0	0	16%
177	0.8	324	41	279	56	227	0	0	0	6%
178	0.8	340	40	200	40	260	0	0	0	10%
179	1.1	120	30	81	16	74	0	0	0	6%
180	0.8	415	30	274	55	330	0	0	0	5%
181	0.8	140	30	105	21	89	0	0	0	2%
182	0.7	160	32	110	22	106	0	0	0	1%
183	0.9	265	36	185	37	192	0	0	0	10%
184	1.1	314	36	207	41	237	0	0	0	30%
185	1	181	42	164	33	100	0	0	0	3%
186	0.8	236	40	187	35	161	0	0	0	2%
187	0.9	114	48	47	9	57	0	0	0	12%
188	0.8	204	45	112	22	137	0	0	0	5%
189	0.9	216	21	161	32	163	0	0	0	5%
190	1.2	148	32	102	20	96	0	0	0	2%
191	1.2	210	45	176	35	130	0	0	0	5%
192	0.9	273	34	173	35	204	0	0	0	30%
193	0.9	162	42	102	20	100	0	0	0	16%
194	0.6	200	38	140	28	134	0	0	0	5%
195	0.8	180	32	145	29	129	0	0	0	16%
196	0.8	188	28	135	27	133	0	0	0	16%
197	0.8	193	39	140	28	126	0	0	0	6%
198	1	146	34	120	24	88	0	0	0	1%
199	0.9	143	34	120	24	85	0	0	0	2%
200	1.2	163	37	148	30	96	0	0	0	4%

LEGEND TO MASTER CHART

Sex	1=Male,2=Female
Marital Status	1=Single, 2=Married,3=Widowed
Past H/O CHD	1=Yes, 0=No
Past H/O Diabetes Mellitus	1=Yes,0=No
Past H/O Hypertension	1=Yes,0=No
Family H/O Premature CHD	0=None,1=Father,2=Mother,3=Brother 4=Sister
H/O Exercise	1=Yes,0=No
H/O Smoking	1=Yes,0=No
Urine Albumin	0=None,1=1+,2=2+,3=3+,4=4+
Urine Sugar	0=None,1=1+,2=2+,3=3+,4=4+
ECG-Ischemia	1=Yes,0=No
ECHO-RWMA	1=Yes,0=No
USG-Evidence of nephropathy	1=Yes,0=No

LIST OF ABBREVIATIONS :

CHD : Coronary heart disease

NCEP-ATPIII- National cholesterol education program-Adult Treatment Panel III

AHA/NHLBI-American Heart association/National Heart, Lung,Brain Institute

T.C- Total Cholesterol

H.D.L- High density lipoprotein cholesterol

LDL-Low density lipoprotein cholesterol

TGL-Triglyceride, WHR-Waist Hip ratio, IFG-Impaired fasting glucose

IGT-Impaired glucose tolerance , D.M-Diabetes Mellitus

HTN-Hypertension

FRS-Framingham Risk score