Epidemiology and Prevention of Coronary Heart Disease in India

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Introduction

Coronary heart disease (CHD) is the commonest variety of heart disease that occurs as a result of critical blockages in coronary arteries, manifesting as angina pectoris, acute myocardial infarction (heart attack), or sudden death. Like other living tissues, heart muscle also requires adequate blood supply for nourishment. Coronary arteries supply blood to the heart muscle. A critical obstruction to blood flow through one or more major coronary arteries compromises blood supply to the heart muscle. At birth, major arteries of the body are smooth, open and elastic conduits for blood circulation. These expand and contract, as blood flows through them. With ageing, arteries lose their flexibility and fatty substance(s) circulating in the blood may clog them up at places. This gradually compromises blood flow through the arteries. These degenerative changes constitute, what is known as, atherosclerosis.

Atherosclerosis is inevitable with ageing. However, atherosclerosis may occur rapidly with certain diseases, characteristics or behaviours called coronary risk factors. Detecting a risk factor does not mean that CHD is present. It only means that there is a risk of CHD. Controlling risk factor(s) may reduce the risk or slow down the progression of CHD.

Coronary Risk Factors

Advancing age, male gender, and occurrence of CHD in a first degree relative before 55 years of age are the most important coronary risk factors. We have no control over these factors. Hence, these are called non-modifiable risk factors. Then, there are factors, which may be prevented, or controlled. The major ones in this category are high blood cholesterol, tobacco abuse, high blood pressure (hypertension), diabetes mellitus and physical inactivity. Other coronary risk factors are marked overweight (obesity), and psychosocial factors (Table 1).

Table 1
Risk Factors for Coronary Heart Disease

Factors Which Cannot be Modified

- Age more than 55 years (Males), more than 65 years (Females).
- Male gender (Males are more prone to atherosclerosis).
- Family history of CHD before 55 years of age (Occurrence of CHD in a parent or grandparent before 55 years of age).

Factors Which May be Modified

- High blood cholesterol.
- Smoking or tobacco chewing (Present/Past).
- High blood pressure.
- Diabetes mellitus
- Physical inactivity (Sedentary lifestyle).
- Psychosocial factors
- Obesity, especially abdominal fat deposition (Central obesity).
- (Waist circumference >90 cm in males, >80 cm in females; Waist to hip girth ratio > 0.90 in males; > 0.80 in females)

CHD in India

Of all the major ethnic groups in the world, Indians run the highest risk of CHD. The risk of CHD in Indians is 3 to 4 times higher than white Americans, 6 times more than Chinese and 20 times greater than Japanese people. The prevalence of CHD in urban adults is estimated to be 7 to 10 percent in North India, and as high as 14 percent in South India. CHD prevalence in India is rising, though it is not so common in rural regions.

In white populations, the average age at which the first heart attack occurs is 55 years. Indians generally fall prey to a heart attack around 50 years of age. It also seems that the risk of heart attack in young adults, in 3rd and 4th decades of life, is rising in India. Classical risk factors alone fail to explain this.

The prevalence of hypertension in Indians is not extraordinarily high. Smoking is more prevalent among Westerners. The average level of cholesterol in Indians is also lower. Still, they are prone to CHD at a young age. This so called, "The Asian Paradox," is probably due to many factors, such as:

- High prevalence of diabetes mellitus. The prevalence of non-insulin dependent diabetes mellitus (Type 2 Diabetes Mellitus) is 3 percent in North India, 7 percent in South India and rising.
- Raised blood triglycerides and low HDL levels. Though the average blood cholesterol level of Indians is not as high as Westerners, they commonly have raised blood triglycerides and low HDL levels, which also increase the risk of CHD.
- Abdominal obesity. Indians have a tendency for fat deposition around abdomen - the abdominal, or central obesity. Abdominal obesity is an important risk factor for CHD. It results from an excess of insulin in blood and resistance to its actions. Insulin regulates blood glucose level. Insulin resistance leads to high blood triglyceride levels, low HDL levels, abdominal obesity, and increase in the risk of hypertension and diabetes mellitus, all of which increase the risk of CHD. The constellation of these clinical and biochemical abnormalities is referred to as metabolic syndrome.
- Physical inactivity. Regular exercise is not a popular past time with Indians.
- Excessive consumption of saturated fat. Desi ghee and coconut oil. These may increase the risk of CHD by increasing blood cholesterol level. High prevalence of CHD in Kerela could be due to coconut oil consumption.
- Consumption of hydrogenated fats like vanaspathi. Hydrogenated fats contain trans-fatty acids, which, in some ways, behave worse than animal fats like desi ghee.
- Lp(a). Lp(a) is a special variety of cholesterol, which has recently been recognized to be an important CHD risk factor. Lp(a) levels have been found to be 3 times higher in Indians, compared to Caucasian and Chinese populations. Lp(a) level is largely determined by genes.

Hypercholesterolemia and Hypertriglyceridemia

Cholesterol is a fat like substance of soft and waxy consistency, circulating in minute quantities in blood. It serves many important functions. It is a component of animal cell membranes. Cholesterol is also a precursor of bile acids, vitamin D and several hormones. Excess of blood cholesterol increases the risk of CHD.

Atherosclerosis is a slow process which begins as early as second decade of life. Thereafter, it keeps on progressing for decades, till it is severe enough to cause symptoms. One of the early steps in atherosclerosis is deposition of circulating cholesterol in arterial walls. Such deposits are visible as fatty streaks. Gradually, fatty streaks grow into hard plaques, which narrow down the arterial passages by bulging into them. Calcium deposition in the plaques may harden the arteries further. Critical obstructions compromise blood flow through the affected artery. This reduces oxygen supply to vital organs. Sometimes, an acute increase in plaque size leads to complete cessation of blood flow. This commonly follows ulceration of the plaque surface, which promotes abnormal blood clotting within the artery. Atherosclerosis may also involve major arteries supplying blood to the brain, the kidneys and the legs.

Triglyceride is the fat, present in cooking oil and dairy products. Both cholesterol and triglycerides are lipids. Lipids are insoluble or poorly soluble in water. Therefore, cholesterol and triglycerides don't mix well with blood. So, lipid transportation in blood relies on carriers called lipoproteins, which are complexes of cholesterol and triglycerides with blood proteins. Lipoproteins escort cholesterol and other fats in the blood from one place to another. Based on their density, the important lipoproteins are:

- Low density lipoprotein (LDL) Accumulation of LDL cholesterol in arteries gradually results in their narrowing and eventual blockage. Therefore, LDL cholesterol is nicknamed bad cholesterol.
- High density lipoprotein (HDL) HDL scoops cholesterol from arteries, and takes it back to liver for disposal. This property has earned HDL cholesterol the tag of good cholesterol. A low HDL cholesterol level increases the risk of CHD.
- Very low density lipoprotein (VLDL) VLDL carries fat and cholesterol from liver into circulation. VLDL cholesterol also behaves like LDL cholesterol.

Cholesterol is present in flesh foods and milk and milk products. Plants are devoid of cholesterol. Cholesterol is also synthesized in the liver. Liver forms about 1500 mg of cholesterol every day. Like cholesterol, blood triglycerides are also derived from dietary fats and internal production in liver from fats and sugars.

Blood cholesterol, LDL, HDL, VLDL, and triglyceride levels are estimated by a blood test called "Lipid profile." Ideally, everybody should have a lipid profile at the age of 20 years. This is to assess the future risk of CHD. It also helps in detection of genetic abnormalities of cholesterol or triglyceride levels. Lipid profile is interpreted by referring to

standard guidelines. If lipid profile is normal at 20 years, it should be repeated after 5 years to assess age related changes in lipid levels. Lipid profile in young age is especially indicated in the situations listed in table 2.

Table 2
Indications of Lipid Profile in Healthy, Young Persons

- Family history of CHD or stroke, especially before the age of 55 years.
- Family history of high blood cholesterol.
- Lipid deposits as cream coloured elevated patches over eyelids.
- Persons with obesity, hypertension, diabetes or thyroid problem.
- History of excessive drinking.
- History of chronic oral contraceptive pill intake.

There is no threshold level of cholesterol below which CHD cannot occur. Thus, the risk of CHD with blood cholesterol level is "continuous." Nevertheless, it may be possible to halt the progression of coronary atherosclerosis in established CHD by reducing total cholesterol to < 150 mg/dl. LDL-C, VLDL-C, and triglycerides increase the risk of CHD. Therefore, their blood level should be low. HDL cholesterol is an anti-risk factor. Its level should be high. Desirable levels of LDL-C, HDL-C, VLDL-C and triglycerides are given in table 3.

Table 3
Desirable Blood Levels of Cholesterol Subfractions and Triglycerides

LDL - C	<100 mg/dl
HDL – C	> 40 mg/dl for men
	> 50 mg/dl for women
VLDL-C	< 28 mg/dl
Triglycerides	< 150 mg/dl

Leading causes of LDL Cholesterol level elevation are excessive cholesterol formation in the liver, and high saturated fat intake. The latter suppresses the ability of liver to remove cholesterol from the blood. Other causes are cholesterol rich diet, and medical disorders like hypothyroidism. High blood triglyceride levels may be due to excessive formation of triglycerides in the liver. Other reasons are inefficient storage of dietary triglycerides into fat deposits, high fat diet, excessive intake of sugar and refined carbohydrates, too much of alcohol, and medical disorders like diabetes, obesity and kidney failure. Very low HDL cholesterol level may be due to genetic problem. There is also a reciprocal relationship between blood levels of triglycerides and HDL cholesterol. Other reasons are lack of physical activity, undesirable weight gain, tobacco abuse, diabetes mellitus, oral contraceptive pill intake and excessive intake of PUFA rich oils, sugar and refined carbohydrates. Lipid abnormalities due to medical disorders are known as secondary dyslipidaemia. Common causes of secondary dyslipidaemia are diabetes mellitus, hypothyroidism, obesity, and kidney failure.

Strategies for cholesterol management

Healthy eating and desirable lifestyles are the most important strategies. Medicines are required in some cases. Principles of healthy eating are: Eat a variety of foods, eat in moderation, restrict total fat intake, minimize saturated fat intake, avoid hydrogenated fats, reduce cholesterol intake, ensure adequate fibre intake, restrict simple sugar and refined carbohydrates, and limit alcohol, caffeine, and salt intake. To achieve these goals, eat:

- Plenty of cereals, pulses, vegetables and fruits.
- Moderate amount of low fat dairy products, meat, poultry and eggs.
- Restricted amount of fats, especially animal fats, oils and sweets.

Cholesterol is found only in animal foods. Most of the animal foods are rich in both cholesterol and saturated fat. Some like egg yolk, liver, brain, kidney and sweetbread are especially rich in cholesterol but their fat content is not so high. Fish is low in fat and cholesterol. Is it all right to have a "zero cholesterol" diet? As such, a "zero" cholesterol diet does not have any untoward effect, since cholesterol needed by the body is manufactured internally. To eliminate cholesterol from the diet, it is necessary to stop all non-vegetarian food, including dairy products, except may be a cup of skimmed milk in a day. In practice, such harsh measures are neither necessary, nor desirable. Vegans are at a risk of vitamin B12 and calcium deficiency. Standard advice for reducing cholesterol and fat intake is:

- Minimize the frequency, and servings of flesh foods.
- Prefer "white meat" over "red meat." Breast portion of chicken flesh contains the least amount of fat and cholesterol. Choose poultry without skin. Light chicken should be preferred over dark chicken. Avoid fried chicken.
- Red meat may be taken occasionally. Choose lean cuts of red meat over fatty cuts. Children can eat meat, especially lean meat. For teenage girls, who are more likely to get iron deficiency anaemia, lean meat is an especially important source of iron.
- Egg yolks should be eaten in moderation, or not at all. With mild elevation of cholesterol, 3-4 egg yolks can be eaten in a week. Don't forget to count the egg yolks used in processed and baked foods. Egg white is all right, as it is cholesterol free. Two egg whites can be substituted for 1 whole egg in egg preparations.
- Avoid organ meat like liver, sweetbread, kidney, and brain, except rarely.

- Fish may be eaten 2-3 times a week. It is also all right to take shellfish like clams, mussels, and scallop. Avoid crabs, prawns, lobsters and shrimps.
- Consume skimmed milk and skimmed milk products like low fat yogurt, cottage and low fat cheeses, and butter milk. Avoid whole milk, whole milk yogurt, high fat cheeses, cream, ice-cream and non-dairy creamer. Dairy products are often added to foods like casserole, pizza, cookie, and sauce.
- Eat plenty of fresh fruits and vegetables.
- Choose low fat sweets and snacks. Avoid fried sweets like jalebi, gulabjamun and imerti.
- Low fat cooking methods are very helpful in reducing overall fat content of diet. Desirable methods of cooking are steaming, grilling, boiling, broiling, baking, stir frying, barbecueing and micro-waving. Flesh foods can be roasted or grilled. Grilling and roasting also allow ex-cess fat to drip away.

Most natural fats are mixtures of saturated and unsaturated fats, which behave differently. Based on relative predominance, a fat is recognized as a source of either saturated or unsaturated fat. Saturated fats tend to solidify if kept in refrigerator. They raise blood cholesterol more than any-thing else we eat. In fact, the blood cholesterol elevating potential of dietary saturated fat is even more than that of dietary cholesterol. Animal fats are rich in saturated fatty acids (SFA). Edible oil seeds are the chief source of unsaturated fat. There are 2 varieties of unsaturated fats: polyunsaturated fats (PUFA), and monounsaturated fats (MUFA). Unsaturated fats tend to remain liquid during refrigeration. Unlike SFA, both PUFA and MUFA reduce LDL cholesterol. Cholesterol reducing action of PUFA and MUFA rich vegetable oils make them natural choices to keep blood cholesterol levels under check. Sunflower, safflower, corn, cottonseed, and soyabean oils are PUFA rich. MUFA rich oils are olive, groundnut, sesame, mustard and Canola (rapeseed, toria) oils. Though both PUFA and MUFA reduce LDL cholesterol, excessive PUFA intake may be detrimental. It may reduce HDL cholesterol. Therefore, oils with very high PUFA content alone are not so desirable. It is better to strike a balance between PUFA and MUFA intake, while minimizing saturated fat intake. In addition, adequate omega 3 PUFA intake should also be ensured.Omega 3 fats are a special variety of PUFA, which decrease blood triglyceride levels, and also reduce the tendency of abnormal blood clotting. Fish and fish oils are rich in omega 3 PUFA. To ensure adequate PUFA, MUFA, and omega 3 fat intakeselecting correct cooking media is important:

- Choose a variety of vegetable oils instead of a single source.
- Mix 2 or more oils with different composition. A good choice is an equal mixture of PUFA rich sunflower, safflower or corn oil, with MUFA rich groundnut oil, supplemented with mus-tard oil, to ensure adequate omega 3 PUFA intake.
- A blend of rice bran oil with sunflower oil, Canola oil and sesame oils are also some good choices.
- Mustard oil or Rapeseed (Canola) oils have similar fatty acid composition, except that erucic acid content of genetically engineered rapeseed is low. In recent years, these oils are increasingly getting accepted as good cooking media because of their high MUFA, and particularly high omega 3 fatty acid content. These oils are also relatively stable at high temperatures, and thus should be preferred over high PUFA oils like safflower and sunflower oil for frying.

A commonly asked question is whether it is alright to have plenty of vegetable oils? Well, no, oils of plant origin provide as much energy as animal fats. When taken in excess, vegetable oils also promote weight gain. This nullifies their beneficial effects. On the other hand, small amounts of butter and desi ghee are not detrimental. Another common question is why not a fat free diet? Very low or fat free diets are generally tasteless, ineffective and may even be harmful. Fat is a normal constituent of human body. Very low fat or fat free diets may cause deficiency of essential fats, as well as fat soluble vitamins i.e. vitamins A, D, E and K. None of these can be manufactured by the body. Also, there is some evidence that very low fat diet may reduce HDL cholesterol. Therefore, diet should not be fat free.

One should remember that neither crude, nor processed (refined) oils are ideal; the best oil produced by cold extraction from an organically grown crop of seeds. In the absence of organic farming on a large scale, it is preferable to use refined oils to avoid contaminants and toxins. Secondly, oil should be stored in a cool, dry, and dark place, avoiding contact with heat, moisture, and light. And of course, only branded oils should be purchased from a reliable outlet, noting the date of manufacture carefully.

Hydrogenation increases the saturated fat content of oils, and also results in formation of unnatural “trans-fatty acids.” Trans-fatty acids raise LDL cholesterol, decrease HDL cholesterol, and increase the tendency of abnormal blood clotting. Thus, even though hydrogenated fats are devoid of cholesterol, they are detrimental for health. For these reasons, vanaspathi, margarine, and low fat butter should be avoided. If the total amount of fat in diet is low, butter is a better choice than low fat butter.

Eating fish may be beneficial for the heart. Fish provide high class protein, calcium, omega 3 PUFA, and other nutrients. Saturated fat and cholesterol content of most fish is also low. Regular consumption of fish reduces the risk of death from CHD. So, it is a good choice. Eating fish is preferable to fish oils because fish oils are unpalatable, it is difficult to swallow a large number of fish oil capsules, and fish oils are lightly hydrogenated to increase their shelf life.

Nuts like almonds, cashew nuts, groundnuts and walnuts are alright. Most of the fat present in nuts is unsaturated. Nuts are also devoid of cholesterol. However, overweight people should avoid eating too much of nuts due to their high fat content.

Dietary fibre is the indigestible carbohydrate in our diet, derived from plant foods. Fibre is of 2 types, water insoluble and soluble. Soluble fibre reduces blood cholesterol level. Liver is the chief site of cholesterol synthesis in our body. A part of cholesterol synthesized by the liver gets incorporated into bile acids and bile salts. Bile is secreted in the gut to help digestion and absorption of food. It is reabsorbed and taken up by the liver after digestion of food. Dietary fibre interrupts this cycle by preventing reabsorption of bile salts from the gut. The latter are excreted with undigested food. Vegetables, fruit peel, and outer coating of unmilled grains contain insoluble fibre. Rich sources of soluble fibre are barley, jowar, ragi, oats, kalachana, rajmah, soyabean, peas, guava, pomegranate, grapes, and apple. Wheat, bajra, corn, kabulichana, redgram, peaches, and pears also have good quantity of soluble fibre. Refined cereals like rice, suji, maida, noodles, vermicelli, potato, and fruit juice are poor sources of fibre. Guar gum powder, isabgol, oat bran and fenugreek (methi) seeds are good sources to supplement the dietary fibre. These supplements are not required, if dietary fibre content is adequate.

It is important to avoid excessive sugar intake, even if a person is not a diabetic. Too much of sugar promotes undesirable weight gain and tends to increase blood triglyceride level and reduce HDL-cholesterol level.
There is some evidence that more than 5-6 cups of coffee, especially unfiltered, boiled coffee may increase LDL cholesterol by 9-14 percent. This is not seen with filtered coffee. Coffee, tea and most aerated drinks also contain caffeine. These should be taken sparingly. Habitual coffee or tea drinkers may limit tea or coffee intake to half a cup at a time. Decaffeinated coffee also reduces caffeine consumption.

Some years back, it was observed that atherosclerosis is greatly accelerated by oxidation of LDL. This gave rise to speculation that antioxidants might prevent arterial clogging. Pursuing this hypothesis, vitamin C, beta carotene, and vitamin E supplements were tried. Vitamin C and beta carotene did not show much promise, but vitamin E did. Nevertheless, clinical trials of large doses of vitamin E supplements did not show reduction in heart disease or even showed some increase in risk:

- Supplements contain a high dose of vitamin E, to the tune of 20-40 times the recommended allowance. Vitamin E is fat soluble. High doses of vitamin E may accumulate in body, with unpredictable side effects.
- Vitamin E supplements may interfere with action of concurrent medication. A notable interaction is with oral anticoagulant drugs, which may increase bleeding tendency.
- The consensus at the moment seems to be in favour of keeping a good antioxidant status by eating plenty of vitamin E rich natural foods, rather than pill supplements. Ample vitamin E is present in natural foods. Rich sources of vitamin E are seed oils, nuts, green vegetables, grains, garlic, and onions.

Clinical trials indicate that various garlic preparations cause small reductions in serum cholesterol and triglyceride levels. These effects are short lasting. While chemical compounds in garlic are reported to have significant antioxidant properties, the evidence that they have important antioxidant properties that protect from cardiovascular disease is weak. The majority of trials have shown that garlic has inhibitory effects on platelet function and may also enhance spontaneous clot lysis. This aspect seems to hold promise for a beneficial effect of garlic. Most trials do not show a significant difference in blood pressure between garlic treatment and placebo. Finally, although is possible that garlic or its constituents may have some benefit in reducing atherosclerotic progression or even causing regression of existing atherosclerotic disease, there are insufficient data from which to draw useful conclusions. Except for body odour, there are no studies demonstrating detrimental effects of garlic consumption. Therefore, while the cardiovascular benefits of garlic and garlic supplements may be questionable, consumption of garlic supplements does not appear to be dangerous. Unless medical conditions dictate otherwise, the decision to consume garlic or a garlic supplement for cardiovascular health benefits is best left to individual discretion.

There is general agreement that the consumption of soy protein reduces the serum cholesterol. Because of the strong clinical evidence supporting this diet-disease relationship, the US FDA now allows the labels of certain soy foods to bear a health claim. A soy protein diet that reduces TC level by 10 percent can be anticipated to decrease the risk of CHD by 20 percent. Although the specific component of soy responsible for the lipid-lowering effect is unknown, isoflavones present in soy per se are not likely to be responsible. However, isoflavones may have other cardiovascular benefits apart from their lipid-lowering effects, including their ability to reduce the oxidation of LDL and enhancement of blood vessel elasticity.

Medication may be required in addition to dietary measures for high blood cholesterol when:

- There is a strong genetic predisposition for an abnormal lipid profile.
- Blood cholesterol or triglyceride level is severely elevated.
- Dietary and other lifestyle measures do not suffice.
- There is evidence of clinical CHD or other manifestations of atherosclerosis.

In the past, medication for cholesterol lowering was associated with considerable expense and troublesome side effects. Current medications are more effective, and with less frequent side effects. Safety and efficacy of drug therapy should reviewed by periodic lipid profile and monitoring of side effects. Drug therapy should be considered a lifetime commitment. Generally, if medication is stopped, cholesterol returns to higher levels.

Research has shown that aggressive LDL lowering with diet and medication stabilizes heart disease, stops further cholesterol deposition in arteries, and may even result in some regression of established lesions.

Physical Activity

Regular exercise prevents CHD and premature death. Investigations have shown that physically fit men and women live longer than physically unfit people. A middle aged sedentary person should undergo medical evaluation and a stress test before embarking on regular exercise. This is to detect unsuspected hypertension and diabetes mellitus, and to determine the safety of exercise. It is not necessary to become an athlete. Modest exercise for 30 minutes, 5-7 times a week is enough. There is no need to test the limit of endurance, to sweat or to develop marked breath-lessness during exercise. Excessive effort may be injurious for the muscles, and the joints, without additional benefits for the heart. The level of physical effort may always be increased gradually, as one becomes more and more fit. Exercise should be preceded by warm-up. Warm-up is a brief session of slow, gentle, rhythmic and repetitive movements of the major joints of the body before exercise. Warm-up improves the flexibility of joints and prevents stretch injury. A warm-up also ensures a smooth and gradual rise in pulse rate, and blood pressure before exercise. For exercise, free movements like brisk walking, swimming, and cycling, or light games like badminton and table tennis are good. Heavy weight lifting is undesirable. Some cool-down exercises should always be performed after exercise. Cool-downs are vigorous stretches, performed after exercise. This is to prevent muscle and joint stiffness, and abrupt fall in blood pressure after cessation of exercise. A few minutes of physical and mental relaxation after cool-down is also rewarding. Muscle strengthening exercises are also not contraindicated for heart patientsunless the patient has uncontrolled hypertension or heart failure. Light weight training in stable heart disease is beneficial provided it is begun under medical supervision and does not lead to extreme fatigue or undue increase in blood pressure. Safety rules for exercise are given in table 4.

Table 4
Safety Rules for Exercise

- Exercise either before, or 2 hours after major meals.
- Avoid exercise when unwell.
- Avoid a steaming hot shower after exercise.
- Don't discontinue exercise for more than 2 weeks at a stretch.
- Have any fresh symptom evaluated.
- Execute all movements gently and rhythmically.
- Avoid difficult and unenjoyable exercises.
- Avoid breath holding.
- Avoid early morning walks in winter. Don't walk against cold wind. Avoid heavy woollens, and wear a face scarf in extreme cold.
- Wear loose and light coloured cotton clothes in summer season. Exercise under the shade. Drink water before, during, and after exercise. Slow down the pace during extremely hot weather. Be alert for signs of heat injury. These are headache, dizziness, faintness, nausea, cramps and palpitations.
- Elderly should avoid high impact activities. They should step up the pace of exercise very gradually, over a period of time.
- Obese people should perform low intensity, low impact, exercises like brisk walking. They should exercise at least 5 days in a week.
- Hypertensive patients should avoid vigorous exercise if blood pressure is not well controlled. They should not hold their breath, and avoid weight training.

Alcohol Consumption and CHD

Several studies have shown that regular alcohol consumption in moderate amounts may reduce the risk of death from fatal heart attacks. Beneficial effects of alcohol on heart, if any, may be due to a rise in HDL cholesterol, which is an anti-risk factor for atherosclerosis. Even if light drinking protects against CHD, it cannot be recommended for this purpose. At the same time, social drinkers need not stop drinking. Small amounts of alcohol are not harmful. Alcohol in excess of 60 ml of whiskey/rum, 180 ml. of wine or 360 ml. of beer should be avoided. Excessive consumption of alcohol may raise blood triglyceride levels, weight and blood pressure and damage the liver. Regular drinkers may miss their drink badly after recovering from heart attack. An occasional drink is all right for them. However, alcohol should be avoided at all costs, if heart attack leaves behind a poorly pumping heart or heart rhythm abnormalities.

Alcohol depresses heart muscle contractions, even in social drinkers. This goes unnoticed as long as heart muscle is healthy. Overindulgence for more than 10 years may lead to heart muscle damage, called alcoholic cardiomyopathy, manifested by progressive heart enlargement, deterioration of pumping efficiency and eventually, frank heart failure.Rapid, and irregular heart rhythms may be precipitated after heavy drinking. Sometimes, these are life-threatening. This is called Holiday Heart Syndrome, as many such cases reach emergency during weekends and holidays, when overindulgence in alcohol is especially common.

The primary determinant of alcohol induced problems is the quantity of alcohol. The type of bev-erage does not matter. Occasionally, contaminants like cobalt, arsenic and lead may contribute to heart damage. Rarely, alcohol induced deficiency of thiamine (vitamin B1) may precipitate heart failure in a chronic, malnourished alcoholic.

Tobacco Abuse

Chances of a smoker dying in a given period of time are 70 percent more than that of a non-smoker. The average decrease in life span of a smoker is 4.6 years if one pack is smoked daily from 25 years of age. The decrease in life span is 8.3 years if two packs are smoked daily. Smoking is a major risk factor for CHD. The prevalence of CHD in smokers is 3-5 times more than in non-smokers. People who smoke 20 cigarettes a day are twice as likely to have a heart attack as non-smokers. The risk is increased 10 times if 40 cigarettes are smoked. The most dramatic result of smoking is unanticipated sudden death which is 4 times more common in smokers. With an increase in the prevalence of smoking, more and more young people are getting heart attacks and dying suddenly. Smoking is, in fact, the major or the only risk factor for CHD in people below 40 years of age. Studies have revealed that three out of four young victims of heart attack are smokers. Though men are more adversely affected by smoking, the risk of heart disease increases 10 fold in women taking birth control pills.

Besides CHD, tobacco abuse increases the risk of occlusion or ballooning (aneurysm formation) of the major arteries. This may lead to stroke (Cerebrovascular accident, brain haemorrhage or clotting of a brain artery), foot gangrene (loss of viability of the foot, requiring amputation) or rupture of a major artery, leading to serious or fatal bleeding in internal organs or body cavities. One out of five instances of stroke is attributed to smoking. Smoking per se does not cause hypertension but may aggravate it. It may also accelerate the complications of hypertension.

Tobacco contains about four thousand chemicals which fall in three categories: Nicotine, carbon monoxide and other organic chemicals. Nicotine leads to an increase in heart rate and blood pressure and narrowing of blood vessels. Carbon monoxide is a deadly gas which kills on acute or heavy exposure. Compared to oxygen, carbon monoxide has a better capacity to bind with hemoglobin, the carrier molecule of oxygen in the blood. Thus, it interferes with oxygen transport and release of oxygen from hemoglobin in tissues of the body. An aggregate of particulate matter in cigarette smoke after removing nicotine and moisture is termed tar. Majority of the chemicals in tobacco smoke, implicated in cancer, are present in tobacco tar.

Smokers not only take inadvertently high risks with their health, but also expose others to the same risk who are in their close contact when they smoke. Passive inhalation of tobacco smoke is known as passive smoking. Studies indicate that a non-smoker smokes equivalent of three cigarettes in a room where 40 cigarettes have been smoked. Side-stream smoke comes from tip of the burning cigarette. Second-hand smoke is that exhaled by smokers. Passive smokers are exposed to side stream and second hand smoke. Side stream smoke contains the same chemicals as mainstream smoke but in much higher concentrations. Both side stream smoke and second hand smoke increase carbon monoxide level of air in a closed space.

Risks of smoking begin to decrease within 1 year of quitting even in life long smokers. The risk continues to fall further with abstinence. Those who continue to smoke after a heart attack or heart surgery have a poor long term prognosis. They are at increased risk of recurrence of angina, heart attack and sudden death. After coronary artery bypass surgery, grafts are more likely to block if smoking is resumed. Despite withdrawal symptoms associated with cessation of smoking,as a rule, it can only be given up abruptly. A gradual reduction is not very successful in giving up smoking.

Obesity and CHD Risk

Obesity (Severe overweight) increases the risk of fatal heart attack, when the weight exceeds 30-40 percent of ideal body weight. Obesity is associated with nearly all CHD risk factors, except smoking. Blood cholesterol, triglycerides, and glucose levels, as well as blood pressure tend to rise with weight gain. HDL (good) cholesterol also falls with undesirable weight gain. It also increases the risk of type 2 diabetes mellitus, previously called non-insulin dependent diabetes mellitus (NIDDM).

Breathing insufficiency in marked obesity may precipitate heart failure, the so called “Cor-pulmonale.” A less common cause of heart failure in marked obesity is cardiomyopathy, characterized by weakness of heart muscle contractions.

A good measure of obesity is, what is called, “body mass index,” or B.M.I. This is calculated as, weight in kg., divided by square of height in meters. For example, B.M.I. of a person, who is 1.66 meters tall, and weighs 90 kg., is, 90/(1.66)2 = 90/2.75 = 32.7. Normal and abnormal ranges of B.M.I. are given in table 5.

Table 5
Body Mass Index (B.M.I.)

Normal	<23.0
Overweight	23.0 – 26.9

Obese	27.0 – 29.9
Very Obese	≥30

An important measure of health risks of obesity is distribution of fat in body. Abdominal or central obesity is more risky than generalized obesity. Central obesity, also called apple type is often seen in males, while so called pear shape is more characteristic of females. Abdominal obesity is present when waist circumference exceeds 90 cm in males and 80 cm in females. Another way of finding whether abdominal obesity is present is to calculate waist to hip ratio. Abdominal obesity is present when waist to hip girth ratio exceeds 0.90 in males and 0.80 in females.

Weight reduction often reduces blood pressure to normal levels in obese people. Weight reduction also reduces frequency and severity of angina, risk of heart attack, and improves the pumping efficiency of the heart. Weight is a net outcome of average energy intake and energy expenditure. Therefore, the basic principles underlying obesity control are restriction of energy intake and physical activity. This may be achieved by setting up definite targets, keeping a track of energy intake, and regular exercise. Unless these measures are implemented for life, weight gain occurs rapidly once again.

Metabolic Syndrome

Metabolic syndrome is defined as presence of 3 or more of the following inter-related risk factors in a person: abdominal obesity, high serum triglycerides, low HDL cholesterol, elevated blood pressure, and elevated fasting blood glucose. The average relative risk of T2 DM is increased by about 5-fold in metabolic syndrome. Nevertheless, there are at least some patients who don't qualify for a clinical diagnosis of metabolic syndrome, yet they are at high risk of diabetes mellitus. Examples include those with history of impaired fasting glucose, gestational diabetes or family history of type 2 diabetes. Therefore, metabolic syndrome is not a profoundly better way of predicting future diabetes than fasting glucose alone. The average relative risk of CVD is increased by about 2- fold in metabolic syndrome. On theoretical grounds, identification of metabolic syndrome is better likely to assess future long-term risk than simply enumerating classical risk factors. Nevertheless, several studies have shown that the risk of CVD associated with the syndrome is no greater than that explained by the presence of its components. Moreover, metabolic Syndrome does not take into account the risk attributable to age, gender, family history, health behaviours, smoking, and LDL cholesterol. Therefore, it is not surprising that metabolic syndrome is does not predict CHD as well as other methods of risk prediction like Framingham Risk Scoring. In diabetes, UKPDS risk engine is considered to be a better predictor of risk. Another more robust tool for risk prediction is ADA Diabetes PHD that predicts 30- year risk of heart attacks, stroke, diabetes, and kidney failure, taking into account a large number of variables including current drug therapy. Thus, the utility of metabolic syndrome as a risk predictor is limited and better tools are available for this purpose. Nevertheless, the concept is popular and further research may lead to development of new therapeutic agents that specifically target the fundamental abnormality underlying the metabolic syndrome.

Psychosocial Factors and CHD Risk

A number of studies have identified an association between depression or depressive symptoms with coronary heart disease, stroke, and hypertension. At the same time, there are also studies have failed to find these associations. Overall, major depressive disorder, current depressive symptoms, and a history of depression all seem to be associated with increased risk of cardiovascular morbidity and mortality. In a recent analysis, the overall relative risk of developing CHD in patients who were initially depression free was 1.64, implicating depression as an independent CHD risk factor. During long-term follow up, depression was associated with 1.6-6.0 times greater risk of recurrence of MI or fatal heart attack. Chronic anxiety, particularly phobic anxiety also appears to be a risk factor for development of CHD. Anxiety may also increase the risk of sudden death in CHD.

Type A person is one who is exceedingly hard-driving, ambitious, competitive, excessively involved in job, time-urgent, unusually quick-tempered, hostile, impatient, exhibiting vigorous speech and psychomotor activity. It had been suggested that people with this action-emotion complex, prompted by certain environmental events and fostered by western culture, which "re-wards" those who can think, perform, communicate, and in general, live more rapidly and aggressively than their peers, have higher cholesterol levels and higher CHD risk, compared with those who are "Type B" with relatively low motivational levels. Current evidence suggests that TABP neither predicts the risk of CHD nor an adverse outcome in CHD. However, specific components of TABP, i.e., hostility and anger may increase the risk of CHD but may not increase recurrent coronary events or CHD mortality. The evidence linking hostility and CHD is less consistent than that for depression, anxiety and lack of social support.

A number of psychosocial stressors are associated with increased risk and progression of cardiovascular disease. Most studies have examined chronic stressors in the form of work stress or care-giving and produced fairly consistent results. Occupational stressors appear to influence outcomes for men, whereas in women, marital life stress and care-giving seem to be more important.

Studies also indicate that lack of social support increases the risk of CHD though the evidence is not unequivocal. However, lack of social support does increase long-term mortality in post MI pa-tients. The type and quality of support seem to confer additional protection over and above the effects of simple social contacts. Mortality risk is lower in people with good social networks pre-sumably because such people are more likely to seek medical care.

Biological effects of psychosocial factors that increase the risk of cardiovascular disease are beginning to be understood. The primary effects seem to be activation of hypothalamo-pituitary-adrenal axis and sympathoadrenal system. Serotonergic dysfunction is present in certain disor-ders, notably major depression. These effects lead to changes in circulatory dynamics, blood clotting, biochemical mileau, blood vessel wall, and endocrine and immune system that in turn promote atherothrombosis, heart rhythm disorders, and vasospasm, thus producing varied clini-cal manifestations.

Unwarranted stress can be removed to some extent, by critical examination of personal acts and beliefs. Self-introspection may identify internal causes of stress, which may be removed by taking corrective measures. When stress is beyond personal control, Yoga, meditation and other techniques of stress management may help.

Aspirin

Daily aspirin in healthy middle aged men may reduce the risk of a heart attack by 44 percent. At the same time, it may increase the risk of hemorrhagic stroke, bleeding peptic ulcer, and sud-den death. Aspirin does not increase the survival of healthy people. For these reasons, aspirin should not be taken for CHD prevention, except when there is a high risk of CHD or when CHD is already present. High risk middle aged and old men are most likely to benefit from aspirin prophylaxis. Aspirin should not be taken, without physician's advice who is able to identify, whether there are any contraindications. The efficacy of aspirin prophylaxis in women is un-known.

Hormone Replacement after Menopause

Female hormone replacement, improves lipid profile, and decreases abnormal blood clotting. At the same time, it increases the risk of endometrial and breast cancer. Large scale studies have failed to show protective effect of hormone replacement therapy against coronary heart disease. Therefore, the case for hormone replacement is strong only when there is evidence of post-menopausal decrease in bone density too. All women on hormone replacement therapy should have access to long term surveillance for endometrial and breast cancer.