CAN HEART ATTACKS BE PREVENTED?

Joseph Kurian Chemplavil, 4th year Medical Student
Kottayam Medical College, Kerala, India

Myocardial infarction, “coronary insufficiency” and angina pectoris as a group is a dreaded companion of the modern civilization. Coronary heart disease (CHD), which commonly manifest as a “heart attack” (the coronary thrombosis, the myocardial infarction) is responsible for thirty per cent of all heart diseases and for eighty per cent of all sudden deaths. It may strike any indiscriminately; irrespective of nationality, caste, creed, age, sex; station or status in life; strangers, patients, friends, enemies and ourselves. It usually affects men between the age forty and sixty, striking them down at that period of their lives when they are at their maximum economic productivity, and when they shoulder the maximum social and domestic responsibility. CHD has shown a tremendous rise in incidence during recent years and is so common among the upper and middle strata of urban society, that it may be regarded as having assumed epidemic proportions. There is an increase in the number of middle-aged and elderly individuals, with control of infections; and it can be expected that the number of heart patients will continue to become progressively larger.

But how many are aware that an epidemic of coronary disease has been continuously advancing unchecked? Because its inroads have been made stealthily, year by year rather than week by week, because it does not usually make headlines in news media, it is no less an epidemic, and the devastation in terms of human life and socio-economic productivity, no less catastrophic. The increasing incidence of CHD- the morbidity and mortality that it carries, with possibility of sudden deaths, sometimes before medical or emergency resuscitative care can be made available, the helplessness which a physician experiences in some of acute cases and in chronic cases with an advanced cardiac disease- all these making it all the more important that a concerted drive for preventing the development of coronary atherosclerosis or its effects manifesting as CHD, be made.

Thus, it is high time that whatever preventive knowledge is available at any stage of our existence, should be made
available to the public by proper application. Some of the recent discoveries in preventive medicine could be translated immediately into new cardiac services for citizens, but the discovery of promising “next step” in the development and application of such knowledge, is even more important.

Having realized at least the gravity of the threat that may lie in wait for him, the intelligent layman asks, “Can heart attacks be prevented?” The question is too simple phrased; a categorical Yes! or No! is impossible.; any brief and simple answer would be misleading. Medical science has advanced to a stage when the cardiologists can reply with a qualified “Yes”. Heart attacks cannot be prevented completely, nor can those who have already suffered an attack be entirely safeguarded from the possibility of a second one.

**Risk Factors**

It has been proved that certain factors predispose to the development of heart attacks and the risk developing CHD can be reduced by attention to the controllable risk factors. The factors include **male, high blood pressure, obesity, hyperlipidemia especially very high blood cholesterol, heavy cigarette smoking, sedentary life, occupational and psycho-social stress with little or no physical exercise, diets of high calorific value, especially those rich in animal or saturated fats, history of atherosclerosis occurring prematurely (before the age of sixty) and diseases such as diabetes mellitus and hypothyroidism.** Besides, apparently healthy individuals with premature cessation of ovarian activity, a rapid gain in weight, hyperuricemia and a thrombotic tendency, have a greater incidence of CHD. Certain of the risk factors can be altered significantly and others cannot, e.g. as yet no preventive measures are available for metabolic diseases like hyperuricemia and hyperglycemia. Psychogenic stress may require dissociation from environment for its prevention. Current knowledge does not permit one to state categorically that if the factors are controlled, angina pectoris or myocardial infarction will be prevented; thereby returning the patient to the low risk group. It does seem wise however, to recognize that the facts point very strongly in certain directions; they indicate the ‘possibility of prevention’. Nevertheless, physician’s responsibility to advise his patients cannot be postponed until all the scientific problems are solved and all puzzling questions have been answered.

**Tobacco**

Smoking has long been thought to play some part in the development of peripheral vascular disease, particularly thromboangitis obliterans. Atherosclerosis is directly proportional to age and number of cigarettes smoked per day. The consumption of twenty or more cigarettes daily is associated with at least a three times greater hazard of myocardial infarction than is found in non-smokers. On the other hand, angina pectoris is no more common in cigarette smokers than in non-smokers. This striking difference between angina pectoris and other manifestations of CHD is not easily explained. But frequent clinical observations show that
angina may be precipitated or aggravated by cigarette smoking (Doyle, 1967). Male cigarette smokers are about one and half to two times more likely to die of atherosclerotic heart disease than non-smokers. Among smokers, aged thirty five to forty, heavy smokers have about three times the death rate from atherosclerotic heart disease as light smokers. Data are not available concerning the coronary mortality of people habitually chewing tobacco or using snuff.

The adverse effects are usually attributed to the vasoconstriction action of nicotine though it has been pointed out that the act of deep breathing itself will induce comparable degree of vasoconstriction. It may alter constituents of plasma lipid and the coagulability of blood (B.M.J. Editorial, 1963). Smoking decreases the platelet survival in vivo and increases the platelet stickiness. Much remains to be learnt about the deleterious effects of tobacco on coronary circulation. The evidence is in favor of smoking being a cause of CHD. It is far from unlikely biologically that cigarette smoking should be one of the causes of atherosclerotic heart disease. Therefore it is beneficial to stop smoking.

**Obesity**

Overweight and high fat diets are found to be contributory in one series of study of myocardial infarction in young adults. Overweight alone may not be contributory. It is usually combination of two or more of the aforesaid multiple risk factors. Extreme obesity carries an excess risk. In obese men, both with or without elevation of blood pressure and serum cholesterol, an excess risk of angina pectoris and sudden death, appears to exist, indicating an independent contribution of obesity, to the rate of development of these manifestations or coronary atherosclerosis. But in women obesity appears to play a negligible role. Subjects, with both hypertension and raised serum cholesterol and obesity, have a pronounced increase in risk, greater than that with either factor alone.

**Physical Inactivity Vs-Physical Fitness**

It seems likely that a proper balance between the intake of food and the energy expenditure is much more important than the quantity and quality of the food. During the past fifty years, increasing mechanization has steadily reduced the amount of hard physical work, and this tendency is becoming ever more pronounced. At the same time, the intake of food rich in calories, containing purified carbohydrates and hydrogenated solid fats, is continuously increasing. These are the two most striking changes that have taken place in the life of modern man, who is gradually becoming a soft, sedentary, fat creature; living in an artificial hot house atmosphere.

Investigators interested in the role of exercise in the prevention or treatment of CHD are unable to provide straight forward and conclusive answers, for, exercise cannot be put into a capsule form, the dose is difficult to gauge, a real effort is needed on the part of the physician to persuade the patient, and on the part of the patient, to take to
exercise, the effects of therapy are not easy to measure. Answers to questions—Does physical fitness delay the development of CHD or prolong life? What is the optimal level of fitness? What fact of fitness is most desirable for health and longevity? How is the fitness best achieved?—Are likely to be years away.

The possibility that physical fitness alters morbidity and mortality of coronary sclerosis remains conjectural. Some find it difficult to reconcile the observation on the basis of the current hypothesis that physical activity is a protection against CHD. Taking to physical activity may, in addition to daily exercises, imply using staircase instead of a lift, walking up small distances instead of taking the car to the office door and so on.

Hyperlipidemia

Hypercholesterolemia, mostly genetically determined, is one of the most well documented risk factors. Figures for serum cholesterol levels as low as one hundred fifty mgs percent may probably be an average level for Indian people. Raised serum triglycerides are probably as important as raised cholesterol levels.

At present it seems reasonable to reduce levels of cholesterol and triglycerides in the plasma, particularly if they are abnormally high, and diet is a safe method of achieving this. But the results are insufficiently definite to permit changes in habits of a nation or families on a large scale. It has yet to be proved that reduction of elevated cholesterol levels in patients with Hypercholesterolemia alters the prognosis favorably.

Non dietary Method of Lowering Cholesterol

CPIB with and without androsterone (Atromid and Atromid-S respectively) decreases platelet stickiness and causes fall in serum cholesterol. These changes appear to be independent. In the present state of knowledge, Atromid-S (Clofibrate) can be regarded as an effective and safe drug for lowering elevated lipids without the need for any dietary restrictions.

Cholestyramine, which traps cholesterol in the gut and ensures its excretion in the stool, is not very pleasant to take, not always successful and may cause much dyspepsia. But clofibrate, which lowers the cholesterol by suppressing its synthesis at very early stage, is well tolerated, though a little bulky, and it certainly lowers blood cholesterol in seventy five to eighty per cent of those who take it.

The real purpose of any lipid-lowering regimen is to reduce the prevalence of CHD and this can be demonstrated only through primary prevention trials. Such a trial is going on in Edinburg and Budapest. At the end of five years (which will be completing by now) it should be possible to decide whether correction of hyperlipidemia by the particular method provides any degree of control over incidence of CHD (Oliver, 1967).
**Diet**

There is no evidence that protein as such plays a significant role in the development of atherosclerosis. But usual high protein diet is also rich in saturated fat- mammalian flesh- beef, veal, pork and lamb- with butter and other whole milk products, the chief source of saturated fat in the non-vegetarian diet. There is no evidence to support the theory that dietary fat causes CHD. On the contrary, it is difficult to believe that dietary fat plays any significant role in its causation. Diet does not appear to have any significant predisposing effect, but hypercholesterolemia plays a decisive role in the causation of CHD in young persons. According to some, CHD occurs more commonly among non-vegetarians.

An important factor seems to be the amount of fat in the food and its composition. Animal fats containing mainly saturated fatty acids increase the fat content of the blood to a greater extent than same quantity of unsaturated fatty acids which may even lower the cholesterol content of the blood. However, the problem is by no means straightforward.

Although there is an association between elevated serum lipids and the severity of atherosclerosis in human beings, atherosclerotic lesions do occur in individuals with low serum lipid level. In such patients, the pattern of carbohydrate nutrition may give a clue to the disparity between groups of populations (Kendall 1967).

Therefore, recently more thoughts have been applied to carbohydrate and their relation to CHD. It is a common experience to see diabetes with CHD with a greater frequency and also an abnormal glucose tolerance curve in patients after an attack of myocardial infarction. Symptom less impairment of glucose tolerance may be one of the accompaniments of atherosclerotic disease in the general population, although confirmation of its causal role must await further evidence.

**Psychological Factors**

The coronary patient tends to be an intellectual controller of his behavior and his activity. He is concerned with socially accepted norms. His cerebrally planned activities mostly lack emotional backing. His “action without emotion” does not favor arousal of the autonomic nervous system. Cardiac adaptation to exercise is less efficient in such “civilized pattern of activity”. This maladaptation, by frequent recurrence or in combination with autonomic factors- coronary narrowing – may lead to ischemic phenomenon.

Unexpected domestic trouble, in a family normally trouble free, a sudden calamitous financial disaster, the unexpected loss of a loved one, may contribute to the development of the clinical features of the coronary disease, immediately by increasing the heart rate and cardiac output, in patients with advanced coronary atheroma, and later by accelerating the atheromatous process. Severe emotional strain frequently precedes the onset of CHD in patients under forty years of age.
Patients often admit that during the months preceding the attack, they have been unable to cope with their responsibilities and hence have been exhausted.

**Conclusion**

Until such time that all the vexed problems can be and have been solved, the following instructions regarding prevention of CHD are generally recommended. These include restriction of total calories to reduce the weight to the expected normal for height, build, age and sex. Although diet may not be the major problem or the only factor concerned with CHD, it is one about which one should be able to do something. That is why one needs not only to study the strain of modern life, race, climate and exercise; but also especially the details of the diet itself.

The total ingestion of fats permitted may be about twenty per cent of the total calories, or less. Unsaturated oils, e.g. Cottonseed, safflower, olive, corn and maize oil should form the main oils for frying and cooking. Hydrogenated vegetable oils, e.g. Vanaspati, Dalda, Pakav, margarine, butter, ghee and fat of animal meat, are allowed only in small quantities, if at all. Preferably, they should be avoided. Green vegetables, fresh fruits, cereals, skimmed milk and lean meat should form the bulk of the menu. Three or four smaller meals are preferable to two large meals and each meal should be chewed well before swallowing.

Regular exercise, which should be individualized, is useful and should be carried out at least four times a week. But no exercise may be taken after a meal. Rest after each meal, however small, is useful. Foods rich in cholesterol should be avoided e.g. egg yolk, kidney, liver, brain, sweet bread, fish, roe, meat (especially fatty), poultry, butter and cream. However, two eggs per week may be allowed. Control of high blood pressure when this is present is also equally important.

The most sensible advice seems to be to maintain a balance between food intake and energy expenditure, between physical and mental activity, between work and relaxation, rest and sleep; and try to develop a philosophical outlook in life. In the meanwhile, it would be difficult to improve upon the advice given by Dr. Jean Mayer of Harvard to “return to the simplest virtues, abstinence in diet, enough physical activity: if possible, avoidance of unnecessary psychological stress and smoking”.

Let us wait for the formation of Anticoronary clubs in our cities and towns to help the coronary patients understand their problems, the right perspective and to increase consciousness amongst apparently healthy individuals, but carrying one or more of the risk factors.