DIETARY FATS & CORONARY DISEASE

SQN. LDR. J. K. SEHGAL

Assistant Director of Health, Medical Directorate, Air Headquarters, New Delhi.

Introduction

Coronary disease is the most common cause of death due to heart disease in most countries today and claims immediate attention. It is true that the reported statistical increase in coronary artery disease, is partly due to the higher proportion of older people, better methods of diagnosis and the greater interest in the disease. Another 40% of the increase in the crude death rate can be ascribed directly to the changes in procedures and classification adopted with the sixth revision of the International Causes of Death. Taking the above factors into consideration at least 15% of the increase in the death rate can be attributed to coronary artery disease.

Role of Dietary Fats in Heart Disease

One of the first things that occurs to the lay mind, is to put blame on changes in diet for the increased incidence of any chronic disease. Frequently, this reasoning is applied to conditions in which diet is of little or no importance. This results in a flood of diet fads and quackery. In the case of coronary artery disease, however, there is evidence that diet may be of considerable importance. Surveys carried out in different countries like the U.K., U.S.A. and Australia have shown that coronary disease has a multifactorial etiology. The important factors involved, are heredity, high fat diet, lack of physical exercise and occupational stress and strain. The observations of Rosenman and Freidman have shown that mounting tension at work is associated with not only elevated blood cholesterol levels, but also with an increased tendency of the blood to clot. Immoderate ingestion of fat may thus be superimposed on neurogenic disturbance of cholesterol metabolism and blood coagulability.

Biochemical studies have indicated the importance of a disordered state involving the fatty constituents of blood and other tissues, partly at least due to dietary factors. This has resulted in 'fat-phobia' in a large number of people. They want to know whether they are eating themselves into premature heart disease and are entitled to an unequivocal answer. Unfortunately there is no evidence available to permit a rigid stand on what the relationship is between nutrition (particularly the fat content of the diet and type of fat) and coronary artery disease. The ingestion of a large amount of animal fats steadily over many years is suspected to be hazardous but clear proof is still lacking. The type of fat appears important; vegetable fats, especially the unsaturated ones, are probably harmless. According to Ancel Keys, present prevalence of hypercholestrolaemia and coronary disease would be much altered if we simply reduced the general level of fat in the diet. The association of fats with atherosclerosis has been investigated in nearly every part of the world.

Epidemilogical, clinical and experimental data are being collected to arrive at a definite conclusion.

Animal Vs Vegetable Fats

Metabolic studies, such as those reported by E. H. Ahrens have indicated that the substitution of unsaturated fats in the diet leads to reduced serum lipid level. These studies have shown that relatively saturated fats of vegetable origin and saturated animal fats tend to increase serum cholestorol. The reported effects of vegetable fats may be a function of their degree of unsaturation or of their essential fatty acid (E.F.A.) content. The mechanism, by which unsaturated fats lower serum lipids, is not known. Is it by influencing absorption, excretion, transport or utilisation? It has not been demonstrated as yet, whether unsaturation per se, the content of poly-unsaturated fatty acids or some other factors in fats (such as situsterol), is responsible for the decrease in serum lipids which follows administration of unsaturated fat. According to Beveridge, Connell and Mayer, animal and vegetable fats contain some unidentified cholesterol increasing and cholesterol depressing factors respectively. Sinclair postulates that cholesterol is normally esterified with unsaturated fatty acids. Vegetable oils rich in E.F.A. are hardened by hydrogenation and during this process much of the E.F.A. are destroyed and unnatural trans-fatty acids are formed. In their absence, cholesterol in the blood becomes esterified with abnormal or unusually saturated fatty acids and these abnormal esters are less readily disposed of and so may cause atheroma. Phospho-lipids containing abnormal or unusually saturated fatty acids are less readily disposed of, are retained in plasma and increase the coagulability of blood thereby contributing to coronary thrombosis. The exact proof for this hypothesis is still lacking because atheroma has not been described or associated clinically or experimentally with fatty acid deficiency.

The work of Bronte Stewart et al have shown that any dietary factor that raises or lowers the level of β —lipo-proteins in the blood will also by implication raise or lower the liability to coronary disease. In their experiments on volunteers it was shown that ingestion of beef drippings, butter, hydrogenated ground nut oil and eggs in large numbers was followed by a rise in β-lipoproteins levels in the plasma. On the other hand ingestion of olive, ground nut, sunflowers, arachis, seal or pilchard oils was associated with a fall in or low levels of serum β- lipoproteins. Foods, fats and oils thus seem to fall into two classes whose effects on plasma lipids are antagonistic to each other. Although these metabolic studies show a relationship between dietary fat and serum lipids, it does not necessarily follow that there is a cause and effect relationship between serum lipids and atheroselerosis. These may rather be related to some other common cause. It is more likely that atheroselerosis is the end result of a number of factors, some operating locally in the blood vessels, others systemically through the blood stream and nervous system, Heredity, mechanical factors such as blood pressure, anatomy of the arterial wall, hormones, serum lipids and dietary factors including substances other than fats may all be involved. Unfortunately research is handicapped in that atherosclerosis in man cannot be diagnosed at an early stage. It may be pointed out here that an increase in the consumption of hydrogenated fats in the recent years does not necessarily mean a decrease in the intake of E.F.A.

The hydrogenated vegetable oils have replaced the use of animal fats and their use has, if anything slightly increased the amount of E.F.A. in the diet. During hydrogenation the contents of normal linoleate of an oil, while reduced, is not abolished.

Conclusion

It is obvious that much has been learned about fat metabolism in recent years but much remains unknown. Pathologists are not agreed as to the manner in which the lesions of atherosclerosis develop. The normal or desireable levels of cholesterol and other lipids in blood are unknown. Serum lipid levels may not reflect changes in the total fat pool in the body. Lipids, other than cholesterol, may be important in the atheroclerosis problem. In man, the effects of dietary factors other than fats or serum lipid level remains largely unexplored.

In our present state of knowledge with many hypotheses and few established facts, it seems obvious that there is insufficient evidence to recommend any basic change in the diet of the average healthy person. We do not yet know either the kinds of fat or the amounts which are desirable for good nutrition. A diet containing adequate amounts of all the known essential nutrients and furnishing enough calories to maintain normal weight should be recommended. The fat content of the diet should be sufficient only to meet caloric and E.F.A. demands i.e. about 25–30% of the total calories should be from fats. For persons with coronary artery disease or with a strong familial tendency to this disease or for persons with elevated serum lipid level, the physician may prescribe some change in dietary fat intake, as an experimental measure, to be carefully followed up and evaluated.

Finally in spite of the gaps in our present knowledge there is enough evidence to suggest that dietary fat and serum lipid levels may be implicated in the development of atherosclerosis and coronary heart disease to justify intensive exploration of all aspects of this field especially with regards to the role of fat in human nutrition.

References

- 1. Brown, R. G., McKeown, T and Whitfield, A. G. W. Lancet II; 1073-1957,
- 2. Fats and Disease, Leading Article. Lancet 1: 557, 1956.
- 3. Goldsmith, G. A. Fats in Nutrition and Health.
- Keys, A: Deficiency of Essential Fatty Acids. Letter to the Editor, Lancet: 576, 1956.
- 5. Morris, J. N.: Fats and Disease. Letter to the Editor Lancet: 687, 1956.
- Page, I. H., Stare, F. J., Coreoran, A. C.: Pollack, H and Wilkinson, C. F. Atherosclerosis and the Fat Content of the Diet. Report presented to the American Heart Association and American Society for the Study of Arteriosclerosis, Nutrition Committee of the Council on Community Service and others,
- Rosenman, R. H., and Freidman, M.: Presented at Ann. Meeting of American Heart Association Chicago Oct., 27, 1957.

- Russek, H. I. and Zohman B. L: American Journal of Medical Sciences 3: 266, 1958.
- Sinclair, H. M.: Deficiency of Essential Fatty Acids & Atheroselerosis. Letter to the Editor, Lancet 1: 381, 1956.
- Southwood, A. B.: Prevention of Heart Disease, Melroy lecture delivered at the Royal College of Physicians of London on Feb. 5, 1959.