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Many studies conducted over the decades have demonstrated the association of Biochemical, Physiological factors and atherosclerosis, a disorder that underlies most the ischaemic heart disease and cerebrovascular disorders which are most common diseases in the developed countries accounting for more than 50% of all deaths. Atherosclerosis of coronary arteries and its complications particularly thrombosis is responsible for epidemic of premature ischaemic heart disease and that has reached enormous proportion, striking more and more at younger subjects. It will result in coming years in the greatest epidemic mankind has faced unless we are able to reverse the trend by concentrated research into its cause and prevention (W.H.O. Board, 1969).

The lesion of atherosclerosis first appears as fatty streaks, then as fibrous plaques and finally as complicated lesions with calcification, subintimal haemorrhage and obstruction with or without thrombosis.

Studies comparing different populations and interpopulations studies have demonstrated etiologically significant independent association between dietary saturated fat, cholesterol and atherosclerosis. The decisive effect of cholesterol in diet of human upon the
plasma cholesterol and phospholipid level was demonstrated in a series of metabolic ward experiments (Connor et al 1961, Mattson et al, 1972). Atherosclerotic lesions develops mainly from flux of lipids into arteries. Based on above studies Katz et al 1958 formulated well known concept "Nutritional metabolic cholesterol lipid lipoprotein theory of atherosclerosis". It is not a matter of one factor to the exclusion of other but multiple factors interacting to influence atherogenesis and plasma cholesterol level.

A major problem of coronary heart disease exhibits in those nations and communities in which serum cholesterol determination of middle aged man average 220 mg/100 ml. or above. The Framingham study (1959) has shown that in comparison with middle aged man, whose plasma cholesterol level was 210 mg/100 ml. or less, men with a serum cholesterol level over 244 mg/100 ml. have more than 3 times incidence of coronary heart diseases (Dawber et al, 1959). The concept of feed back regulatory mechanism in setting the homeostatic level of plasma cholesterol was suggested by Gould and Popjack (1957).

Inspite of strong association of high cholesterol diet with raised plasma cholesterol there are studies which showed no relationship between dietary cholesterol and plasma cholesterol and this applies to children (Lauer et al, 1975) as well as adults (Connor and
Connor, 1972). Similarly insignificant difference between fasting and post prandial plasma cholesterol is reported by several workers (Schilling et al, 1964 and Heyden, 1969). But most of postprandial studies were done up to 24 hours while Biggs et al,(1952) demonstrated peak radioactivity of ingested C¹⁴ cholesterol in plasma within 36-72 hours.

Recently Quintao et al (1971) observed variable regulatory feed back mechanism and stressed that particular response of an individual may be an important factor in determining the cholesterol content of plasma. Thus when large number of individuals are presented with high cholesterol diet, there is wide distribution of plasma cholesterol response. Plasma cholesterol level of some individuals are high with increased susceptibility to atherosclerosis. The level of others are low and therefore without much risk for atherosclerosis.

It was therefore of interest to study the response of a person before assuming that plasma cholesterol will increase with high cholesterol diet, such test will eliminate uncertainty and would produce a wider choice of food items. It may be advantageous to have more than one cholesterol determination in order to obtain a reliable information on specific subject response to dietary cholesterol because result indicate a simple change in diet regimen per re-influence the plasma cholesterol level.
Keeping the above facts in mind the present study was carried out with following aims and objects.

(1) To study response of cholesterol/fat ingestion in healthy volunteers and diseased subjects.

(2) To study response of cholesterol/fat ingestion in subjects having normolipoproteinaemia or hyperlipoproteinaemia.

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