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The rapidly sweeping pandemic of ischaemic heart disease has led to a search for newer strategies to prevent atherosclerosis in its early stages. Because of a greater risk of developing premature accelerated coronary atherosclerosis early detection of children with familial hyperlipoproteinaemia with subsequent dietary intervention seems to be an attractive approach.

Recently, interest in cord lipid has increased because serum lipids disorders have their roots in childhood and atherogenic changes are postulated to originate in early life. So much so, atherosclerosis is being thought of as a pediatric problem. It has been well documented that the initial change of atherosclerosis (fatty streaks) are visible in aorta & coronary arteries of very young children and increased in the aorta at puberty whether or not these lesions progress to advanced lesions at particular site depends largely on hemodynamic forces and atherogenic lipoproteins.

Cord blood lipid levels are much less likely to be influenced by extraneous factor as compared with that in any other period of life. A general dissimilarity between the cord blood and maternal cholesterol and triglyceride
levels has been described at the time of parturition suggesting that maternal lipids and lipoprotein do not cross the placental barrier (Kaplan and Lee, 1965). However, some evidence have been gathered to suggest that ante-partum factors such as maternal hypertension, antepartum haemorrhage, foetal anoxia or intrapartum compromise such as prolonged labour and leaking per vaginal, muconeous stained liquor amni and post maturity may be associated with hyperlipidaemia (Tsang et al, 1974; Cress et al, 1977).

It has been suggested that hyperlipoproteinaemia can be diagnosed at birth by elevated levels of umbilical cord cholesterol, although opinion to the contrary have been offered and babies with elevated cholesterol at birth had values distributed through normal range when re-examined at one year age (Dermayan et al, 1972).

In small for date babies with intra-uterine malnutrition which favours adipose tissue break-down liberating free fatty acids. The portion of free fatty acids which escapes oxidation for energy is converted in the liver into triglyceride, resulting into rise in blood triglyceride levels. Full term baby on the contrary are in receipt of ready placental supply of nutrient so there is little need of lipolysis in utero (Haridas et al, 1984). Pre-term delivery is not a physiological phenomenon and it involves some amount of stress to fetus which may or may not be manifest clinically (Kumar et al, 1989). Stress
in any form has been shown to raise serum triglyceride levels (Cress & Shabah, 1977).

Before birth fetus utilizes carbohydrates as the major fuel. After birth, with cutting off of the nutrients from maternal circulation and before milk feeding established, the new born has to depend on its own endogenous sources of nutrients for survival. As the carbohydrate store of body are meagre and protein metabolism can account for only a fraction of the total energy requirement, body fat become a major source of energy for the newly born infants. Increased mobilization of lipid from stores and increased lipolysis in the immediate post-natal period have been shown in the normal newborn infants by demonstrating a rise in the level of total lipids. Cholesterol, phospholipid and free fatty acid after birth (Persson B, Gents J, Keele DK, Kay JL, 1966).

The assessment for hyperlipidemia can be done by serum cholesterol, serum triglyceride, serum lipoprotein, serum free fatty acid assays. But simple cholesterol and triglycerides estimation is still retained as preliminary screening tests and it is possible that quantitation of cord blood cholesterol and triglycerides might provide a rapid, easily available and inexpensive measures that could be used prospectively to forecast hyperlipidemia and atherogenic problem in later life.