REVIEW OF LITERATURE
The resultant of interaction between man and his environment is health or disease. Certain ecological, biological and socio-economical conditions of the developing countries of the world favour the greater frequency and severity of certain pathological conditions, foremost among them being the nutritional deficiencies. Infantile malnutrition due to protein and calorie deficiency must have been common in most of the civilizations of the world for centuries, but attention has only been focussed upon it in the early years of this century. Historically marasmus (Greek - Marasmos = wasting) was recognised for ages as being a major contributor to high infant mortality. Proctor (1926-27) and other workers described the physical appearances of the malnourished children but nutrition was not thought to be the cause of such appearance. Williams (1931-32-33) made a great contribution to the subject when she gave a name 'Kwashiorkor', derived from Ga language of Ghana, to a tropical syndrome and maintained that it was nutritional in origin.
and that it was different from pellagra. This was the disease first child got when the second was on the way. It was characterized by skin and hair changes, oedema, moon face, fatty liver, hypoalbuminaemia and psychomotor changes. Waterlow (1948) and Jelliffe et al (1954) used the term 'Sugar baby' to describe, obviously similar condition to Kwashiorkor, found in West Indies, where dermatosis was uncommon though oedema was prominent.

After the second world war (1939-45) the workers were conversant with the fact that marasmus and kwashiorkor were two syndromes of nutritional deficiency. Waterlow (1948) in his monograph on 'fatty liver diseases in infants' recognised that "Kwashiorkor was a nutritional disease probably due to a deficiency of protein and that, in babies dying of under nutrition things were very different, there was complete loss of subcutaneous fat and only small amounts in the liver. " He further added that 'the two syndromes might not be rigidly distinct but that one could be converted into the other by increasing or decreasing the calorie intake.'

Jelliffe (1959) coined the term 'Protein Calorie Malnutrition (PCM) of early childhood' to
include the mild, moderate and severe degrees of malnutrition, which was later on accepted by FAO/WHO Joint Expert Committee on Nutrition (1962) to cover diseases such as marasmus, kwashiorkor and famine oedema. There was a short lived effort through WHO to introduce the term 'Protein Calorie Deficiency Diseases', but this was abandoned by the expert group meeting in 1970 in favour of PEM. Joule being the unit of energy measurement, there were lots of proposals to replace the term calorie by joule and this finally led to a general use of the term 'Protein Energy Malnutrition' (PEM) rather than Protein Calorie Malnutrition.

To emphasize the energy crisis all over the world and the nutritional deficiency being a part of it, some workers have used the term 'Energy Protein Malnutrition' (EPM) - McLaren (1973).

To combat malnutrition grading of PEM was necessary for defining the priorities and to formulate therapy in individual patients. Gomes, et al (1985) are credited with the first ever classification of malnutrition. They used the actual weight expressed as a percentage of standard weight (Boston, 50th percentile) for that age. Children with more than
90% of expected weight for age were classified as normal, between 89% and 75% of expected weight were in mild grade, between 74% and 60% of expected weight were in moderate grade and children of less than 60% of expected weight were classified as severely malnourished. In this classification, presence or absence of oedema was not taken into consideration. The main drawbacks of this classification were that it assumed all children of certain age to have the same weight irrespective of their size, and it also included such children who were underweight as a result of malnutrition in the past.

Jelliffe (1966) modified the Gomez classification and included all cases with nutritional oedema, irrespective of weight in severe degree.

McLaren (1967) introduced a scoring system for classifying the severe forms of malnutrition only (weight of children ≤ 75% of 50th percentile of Boston standard), based on all three methods of assessment viz. clinical, anthropometric and biochemical. He showed that the severe degree of malnutrition in its various clinical forms of marasmus, marasmic kwashiorkor and kwashiorkor formed a spectrum of both clinical signs and biochemical changes, both
being most marked in full blown kwashiorkor and least evident in pure marasmus. In this classification also, the problem of expressing chronicity and stage of disease, however remained unsolved.

Arnold (1969) devised a 'Quacstix method' based upon height and mid arm circumference. The advantages of this classification were; it was age independent, good for field surveys and could be applied by unskilled personnel. Based on this classification, children were divided into two broad categories 'Malnourished' and 'Normal'.

Kanawati and Mc Laren (1970), proposed that the ratio of mid arm circumference and head circumference was independent of sex and age at least from 3 to 48 months. If the ratio was $\geq 0.310$, children were grouped as nutritionally healthy. Children with ratio between 0.310 and 0.280 were classified in mild PCM group, children with ratio between 0.279 and 0.250 were put in moderate PCM group, and children with ratio $\leq 0.249$ were classified in severe PCM group. This classification was again useful for screening large number and could not be used for an individual child.

In 1970, Rao et al suggested an age independent
classification by using weight in kg and height in cms.

Weight in kg

\[ \text{Height in cms} \]

Values less than 0.0016 reflected early PEM.

The classification prepared by 'Wellcome Trust' was approved by FAO/WHO expert committee in 1971. In this classification, children were grouped in 5 grades using their weight as percent of 50th percentile of Harvard standard value.

Underweight - Children having body weight between 80% and 50% of 50th percentile of Harvard standard, without oedema and with minimal deficit in weight for height.

Nutritional dwarf - Children having body weight less than 60% of 50th percentile of Harvard standard, without oedema and with minimal deficit in weight for height.

Marasmus - Children weighing less than 60% of 50th percentile of Harvard standard, without oedema and with '++' deficit in weight for height.

Kwashiorkor - Children weighing between 50% and 60% of 50th percentile of Harvard standard, with oedema and with '+++ ' deficit in weight for height.
Marasmic kwashiorkor - Children weighing less than 60% of the 50th percentile of Harvard standard, with oedema and with '++' deficit in weight for height.

\[ \text{weight for height} = \frac{\text{Weight of patient}}{\text{Weight of normal subject of same height}} \times 100 \]

This was probably the first classification in which and attempt was made to use weight/height as well as weight/age ratios, and included a separate category of nutritional dwarfs'. However, some drawbacks of this classification were; it confused between the type and severity of malnutrition, in this system kwashiorkor appeared to be less severe than marasmus and marasmic kwashiorkor as body weight was between 60% and 80%, gradation of deficit in weight for height by such terms used as 'minimal' or '++' could not be quantitated.


Grade I. Children having weight between 60% and 71% of reference standard.

Grade II. Children weighing between 70% and 61% of reference standard.
Grade III. Children weighing between 60% and 51% of reference standard.

Grade IV. Children having weight less than 50% of reference standard.

Grade I and II were underweight and grade III and IV corresponded to marasmus. When nutritional oedema was present, letter K was suffixed to the grade denoting malnutrition e.g. grade I K, II K, III K etc. I K and II K meant kwashiorkor and grade III K and IV K corresponded to marasmic kwashiorkor.

Waterlow and Rutishauser (1974), published a classification based on weight and height. The present malnutrition was called 'wasting', as measured by loss of weight in relation to height, and past malnutrition was called 'stunting', seen as low weight for age ratios. The children were grouped in following grades:

Healthy children - Weight for height more than 90% and height for age more than 95%.

Grade 1. Weight for height between 90% and 80% and height for age between 95% and 90%.

Grade 2. Weight for height between 80% and 70% and height for age between 89% and 85%.
Grade 3. Weight for height less than 70% and height for age less than 85%.

Waterlow and Rutishauser found that weight for height was independent of age in the age group of 1 to 4 years.

**LIPIDS**

The term lipid is used to describe collectively cholesterol, glycerides (neutral fats), phospholipids, glycolipids, free fatty acids and fat soluble vitamins circulating in the blood. The lipids circulate in the blood in combination with certain proteins as macromolecules, known as lipoproteins. (Hawke, 1976).

Cholesterol is a sterol containing hydrogenated phenanthrene ring. 70 to 80 percent of serum total cholesterol is in ester form and 20 to 30% in form free cholesterol. (Schoenheimer and Sperry, 1934). Gleuk and Tsang (1972), determined the concentration of cholesterol in cord blood samples and found mean level to be 63.8 ± 18.7 mg/dl. Owen and Lubin (1971), found out the mean cholesterol level in the age group
of 1 to 6 years of age, as 150 mg/dl, for 1 to 2 years old children and 165 mg/dl for 2 to 6 years of age.

Glycerides in particular Triglycerides form the main bulk of dietary lipid. About 1 to 2 gm/kg of body weight of glycerides are ingested daily (Henry, 1977). These are the esters of long chain fatty acids and glycerol. During metabolism they are broken into di- and monoglycerides and fatty acids. After absorption, the fatty acids are again converted into triglycerides which are deposited in the liver and adipose tissue (Rex Montgomery, 1977).

Free fatty acids form about 5% of total serum fatty acids. This is the form in which fat enters the blood from the storage depots in adipose tissue to serve as fuel for the tissues (Rex Montgomery, 1977). The concentration of free fatty acids in the plasma is very sensitive to nutritional state. After a meal or a test dose of glucose or an injection of insulin, the level is reduced to half or less (Hadden, 1967) and during starvation free fatty acids level rises (Lewis et al, 1964, 1966).
Nigam et al., 1983 and Sharma et al., 1983, determined the FFA levels in cordblood samples and found the mean level to be between 0.30 and 0.45 meq/l. The normal level of free fatty acids in serum is 400 to 900 μeq/l (Millian Novak, 1965).

Lipo proteins is the form in which most of the lipids are present in plasma. Nearly all the cholesterol, phospholipid and endogenous triglycerides in blood are present as aggregates with various protein moieties (Hawks, 1976). Following are the major lipo proteins groups:

Chylomicrons.

Very Low Density Lipo proteins (VLDL)
Low Density Lipo proteins (LDL)
High Density Lipo proteins (HDL)

Hawks (1976) described phospholipids as a group of compounds, which are composed of glycerol, fatty acids and phosphoric acid. These are found in blood cells, plasma and all tissue cells, usually in combination with proteins and other lipids.
MALNUTRITION AND TOTAL LIPIDS

Van Der Sar (1951) studied 16 hospitalized kwashiorkor children and estimated serum total lipids in them before and after putting them on dietary therapy. He found variable values of serum total lipids on the day of admission. In 6 children the values were between 360 and 635 mg/dl, in 5 children the values were between 667 and 855 mg/dl and in 5 cases, the values were between 930 and 1490 mg/dl. The repeat estimations at the time of discharge from hospital also showed variable results. 4 children showed a rise and 6 children showed a fall in serum total lipid levels.

Schwartz and Dean (1957) investigated 20 hospitalized kwashiorkor children between 15 and 30 months to investigate serum total lipid levels on the day of admission and then after putting them on nutrition therapy, every week. They found a significant rise in the levels, reaching up to 969 mg/dl after 10 days of dietary therapy, as compared to the admission level 532 mg/dl. After 6 weeks time the level fell to 644 mg/dl, which was still higher than the initial level. They divided children into 2 groups,
in one group fat was supplemented in diet, and in other no fat supplementation was done. They found no significant change in the levels. Schwartz and Dean proposed that probably the endogenous fat mobilization led to high levels after the diet therapy.

Cravioto, et al (1959), noted similar changes in the levels of serum total lipids in kwashiorkor cases. They correlated the changes occurring in malnourished children with those of normal children and found a great similarity. They suggested that change in kwashiorkor cases was a biochemical expression of the normal process of growth and development, which arrested during the course of malnutrition, reassumed a rate and pattern comparable to those of normal children.

Mac Donald et al (1963), studied kwashiorkor children between the ages of 13 months and 3 years for the levels of total lipids and its fractions at the time of admission, on 10th day and on 20th day of diet therapy. They found a significant rise in serum total lipid levels after 10 days of treatment followed by a fall in next 10 days period and this rise in the total lipids level was due to an increase in cholesterol, glycerides and phospholipids.
They suggested 2 important features to distinguish kwashiorkor from marasmus; one was very fatty liver, and the other was normal or slightly depleted quantities of depot fat. They proposed that the lipid in those sites most probably arose from the dietary carbohydrate and the changes in lipid levels during treatment reflected a reversal of changes leading to their formation.

Gurson et al (1973), studied serum total lipids and its fractions in marasmus cases with a mean age of 10.5 months and mean weight of 4.50 kg. They estimated serum total lipids on the day of admission and on 30th day after dietary treatment. They noted no significant change in lipid levels as 0 day levels were 647 ± 67 mg/dl and 30 days levels were 645 ± 74 mg/dl, as compared to control levels of 607 ± 74 mg/dl.

MALNUTRITION AND SERUM TOTAL CHOLESTEROL

The workers from Nutrition Research Laboratory, Coonoor, India (1952) studied hepatic cholesterol content in kwashiorkor cases, and found a moderate increase in cholesterol content in liver at the time of admission, which regressed after diet therapy.
Van Der Sar (1951) noted significantly low levels of serum total cholesterol on admission in kwashiorkor, which increased after two to three weeks of diet therapy. The found that rise in serum total cholesterol level was principally due to increased esterified fraction.

Dean and Schwartz (1953) studied total and esterified cholesterol level in kwashiorkor cases and found a decreased cholesterol levels on admission with a tendency to rise and fall after dietary therapy. They could not attribute the changes in cholesterol levels to quality or quantity of diet ingested. Therefore, they suggested that the rapid rise in levels was due to release of preformed fat and cholesterol from stores.

Workers of Nutrition Research laboratory, Cooncoor (1954), found considerably low levels of total cholesterol in kwashiorkor cases which increased significantly after 3 weeks of dietary therapy. They found the average ratio of free to total cholesterol as 0.60 at the time of admission as compared to normal ratio of 0.30.

Ramnathan (1955), studied convalescing kwashiorkor cases and made similar observations as
found by previous workers (Van Der Sar, 1951; Dean and Schwartz, 1953; Cooncor study, 1954).

Schwartz and Dean (1957), in their further study found that the total cholesterol level came down to initial level after about 3 weeks therapy, whereas the esterified cholesterol level remained high.

Cravioto et al (1959), also noted low levels of total cholesterol in kwashiorkor cases, at admission time which rose significantly to a maximum level and then either levelled off or decreased after starting the dietary therapy. They compared the changes with those occurring in normal children from birth to the end of childhood and found a striking resemblance. They suggested that the changes noted during the initial recovery of a malnourished child whose rate of growth and development were decreased to a point at which clinically and biochemically he no longer resembled a child of his own chronologic age but practically a newborn, were similar to changes noted in normal newborns as part of their process or maturation inherent to normal growth and development.

Schendel and Hansen (1961), Mac Donald et al (1963), made similar observations as noted by
previous workers (Van Der Sar, 1951; Dean and Schwartz, 1953; Coonoor study, 1954; Ramnathan, 1955). Schandel Hansen emphasized that the arrested rise or fall was associated with either the onset of complication or inadequate therapy.

Lewis et al. (1964), noted a significant rise in levels in total cholesterol in kwashiorkor cases from $68 \pm 21$ mg/dl to $206 \pm 113$ mg/dl after 2 weeks of dietary therapy, which fell to $152 \pm 29$ mg/dl after 3 weeks of treatment, the later value was still significantly higher than the initial value. In marasmus cases, they found no significant change in total cholesterol levels even after 3 weeks of dietary therapy. The level after therapy was $143 \pm 50$ mg/dl as compared to the admission level $139 \pm 49$ mg/dl.

Jaya Rao and Krishna Prasad (1966), made similar observations in 85 kwashiorkor cases as noted by Lewis et al. (1964).

Taylor (1971), also made similar observations of significant rise in total cholesterol levels after dietary therapy in kwashiorkor cases. In contrast to previous workers he did not notice any significant fall at 3 weeks.
Debnath (1972), studied kwashiorkor and marasmus cases along with complicated kwashiorkor and marasmus cases in the age group of 8 to 60 months to evaluate serum total cholesterol levels on the day of admission, on 10th day and on 30th day of dietary therapy. In uncomplicated kwashiorkor cases, he noted a significant rise in the levels, which rose from 145.9 mg/dl to 189.9 mg/dl on 10th day but on 30th day the level fell back to 155.6 mg/dl which was slightly higher than the initial level. In marasmus cases, in contrast to kwashiorkor he found a less pronounced gradual rise in mean cholesterol level from 135.3 mg/dl at the time of admission to 155.7 mg/dl on 10th day and 169.8 mg/dl on 30th day of dietary treatment. In the complicated group (malnutrition with other diseases) he did not observe any significant change in levels from the time of admission to 30 days of therapy.

Debnath suggested that in face of fatty liver and good depot fat, the low amount of cholesterol in untreated cases of kwashiorkor could be due to lack of appropriate proteins acceptors necessary for discharging lipid from the liver in the form of lipoproteins.
Thus lack of protein acceptors together with the lipogenic tendency, could account for the development of a fatty liver that was responsive to the addition of proteins to the diet.

Gurson, et al (1973), studied marasmic children with mean age of 10.5 months and mean weight of 4.50 kg in the severe and recovery phases to evaluate various lipid fractions. They found the mean level of total cholesterol at admission was 180 mg/dl as compared to level of 172 mg/dl in control cases. After 30 days of therapy, they found the level was 164 mg/dl which was not significantly different from the initial level or the control level. They concluded that similar to mechanisms controlling protein and carbohydrate homeostasis, the marasmic infants probably maintain a balanced source for lipids, as a result of which the plasma levels of lipid and lipid fractions remain within normal limit.

MALNUTRITION AND SERUM FREE FATTY ACIDS

Lewis, et al (1964), studied the levels of free fatty acids in kwashiorkor and marasmus cases and compared the values with normal subjects. The mean age
for kwashiorkor, marasmus and control groups were 22 months, 18 months and 23 months respectively. They found a significantly raised free fatty acid (FFA) values in kwashiorkor cases to $914 \pm 372 \mu\text{eq/l}$, as compared to control values of $367 \pm 306 \mu\text{eq/l}$ ($P < 0.001$) at the time of admission. During treatment rapid fall in FFA level was observed by them and at the end of about 3 weeks time, the values were comparable to those of control group i.e. $402 \pm 336 \mu\text{eq/l}$.

In two of the patients only carbohydrate diet was given for about seven days and the plasma FFA level fell significantly. After introducing proteins in diet in the cases, the levels tended to rise to some extent but eventually showed a fall after five to six days. They concluded that glucose administration depressed FFA levels, whereas prolonged starvation caused elevation in plasma levels. They found the mean level of plasma FFA on admission as $815 \pm 347 \mu\text{eq/l}$ in marasmus, which subsequently fell to $37 \pm 31 \mu\text{eq/l}$ after initiation of dietary treatment, and finally at the time of discharge, it again rose to $466 \pm 104 \mu\text{eq/l}$.

Jaya Rao and Krishna Prasad (1966) studied 85 kwashiorkor children aged 18 to 48 months and weighing
between 6.3 and 14.8 kg. They found that after nutritional therapy with 6 gms proteins and 200 calories per kg per day, there was a significant fall in mean value of non-esterified fatty acids (NEFA) from the pretreatment value of 875.5 ± 47.16 μeq/l to 331.6 ± 94.75 μeq/l on 30th day. The mean concentration of NEFA in their control group was 466 ± 43.16 μeq/l.

They also studied the effect of epinephrine on the levels of NEFA. At the time of admission, after epinephrine stimulation NEFA levels increased from 875.5 ± 47.16 μeq/l to 1,129.9 ± 94.52 μeq/l at 30 min and 1,357.2 ± 273.62 μeq/l at 60 min time. Corresponding levels after therapy were 993.0 ± 164.51 and 490.0 ± 126.4 μeq/l. The mean maximal response to epinephrine administered on admission and after treatment were not statistically different from each other. They suggested that the high circulating NEFA levels could be due to hepatic damage in kwashiorkor cases causing defective uptake of NEFA, or that increased NEFA levels represented an alternative source of energy to tissues.

Plasma FFA concentrations were increased in moderate to severe cases of kwashiorkor (Levis, et al 1964).
This observation was somewhat unexpected in view of the belief that infants developing kwashiorkor habitually consumed an excess of dietary carbohydrates, and it was a well established fact that plasma FFA levels were depressed by the intake of carbohydrates (Lewis, et al., 1964). Lewis, et al. (1966), further studied FFA flux through plasma to assess the quantitative release of FFA into plasma in malnourished children. They studied 4 kwashiorkor and 2 marasmus cases. In the control subjects the flux rate was found to be in the range of 4.9 to 10.9 \( \mu \text{eq/min/kg} \) body weight with the mean value of 6.6 \( \mu \text{eq/min/kg} \), and in the kwashiorkor cases the flux rate was 22.6 to 107.5 \( \mu \text{eq/min/kg} \) with its mean as 64.3 \( \mu \text{eq/min/kg} \) on admission to the hospital. They found that the mean flux in kwashiorkor cases was about 10 times than that of control group.

After treatment for about 10 days, the FFA flux in kwashiorkor had fallen steeply in all patients to 8.1 to 13.4 \( \mu \text{eq/min/kg} \) with the mean value as 10.6 \( \mu \text{eq/min/kg} \) body weight. In the marasmus group, the patients had flux rates above the normal range i.e. 26.6 \( \mu \text{eq/min/kg} \).

In their study, Lewis and other workers found the mean FFA concentrations in kwashiorkor.
marasmus and control cases were 1049.25 ± 296.420, 899.5 ± 362.745 and 287.667 ± 100.271 µg/l respectively. Their study showed that, though the plasma FFA level was 3 to 4 times of the normal in kwashiorkor cases, but the mean FFA flux rate was as much as 10 times higher than the control group. They concluded that production and removal of FFA was increased in kwashiorkor and marasmus.

Hadden (1967) studied the inter relationship between blood glucose, FFA and insulin levels in 24 kwashiorkor cases and 9 marasmus cases, and investigated them on admission and then regularly at an interval of 4 days during the first two weeks of dietary therapy. He observed no significant change in FFA levels in kwashiorkor cases after 2 weeks therapy, as mean level at admission was 1.04 ± 0.08 mEq/l which fell to 0.75 ± 0.06 mEq/l, on the other hand in marasmus, the mean FFA value fluctuated between 0.70 ± 0.16 and 0.91 ± 0.10 mEq/l. He further observed that the kwashiorkor cases showed temporary impairment of carbohydrate tolerance, associated with elevated circulating FFA and a delayed fall in FFA, following a glucose load in the diets. He suggested that their
metabolism could be partially blocked, perhaps due to some enzyme deficiency at the entry to the Kreb-cycle, and stressed that if it were so, this could explain the accumulation of body fat both in depots and in the liver.

Gurson and Saner (1969), studied 14 marasmus cases on admission (within 11 days), after 6 weeks and finally after 14 weeks of dietary treatment. They found no significant change in FFA levels in marasmic cases. In their later study of FFA in marasmic cases (1973), they concluded that marasmic cases probably maintain a balanced source of lipids as a result of which, the plasma level of total lipid and lipid fractions except lipoproteins and phospholipids, remained within normal limit.

Similarly Agedana, et al (1979), found no significant changes in FFA values in untreated marasmic cases, though the values were significantly higher in untreated kwashiorkor cases.
MALNUTRITION AND OTHER LIPID FRACTIONS

Schwartz and Dean (1957), found a significant rise in serum phospholipid levels from 145 mg % to 206 mg % dl at the end of 2 weeks of dietary therapy in kwashiorkor cases, which subsequently fell to 180 mg/dl after 4 weeks therapy. Mac Donald, et al (1963) and Lewis, et al (1964), made a similar observation in kwashiorkor children. However, in marasmus, Lewis, et al observed a rise in serum phospholipid levels after 2 weeks of dietary therapy, though it was not significant.

Taylor (1971), found a gradual rise in levels of phospholipids in kwashiorkor cases after 20 days of therapy after which, he observed a fall on 30th day, though the values were still higher than the initial level. Gurson, et al (1973), studied serum phospholipid levels in marasmic cases, and they found a significant fall in levels after dietary therapy from 150 ± 24 mg/dl to 117 ± 8.9 mg/dl.

Mac Donald, et al (1963), studied serum triglyceride levels at the time of admission and after dietary therapy in kwashiorkor cases and found a significant rise in levels on 10th day of therapy as levels rose from 90 ± 36 mg/dl to a level of 203 ± 46 mg/dl.
and again fell to the initial level of $93 \pm 37$ mg/dl on 20th day of therapy. Lewis, et al (1964) and Jaya Rao and Krishna Prasad (1966), found similar changes in kwashiorkor cases. Whereas in marasmus, Lewis, et al found no significant change on 10th day of therapy, as the levels were $160 \pm 65$ mg/dl as compared to $150 \pm 57$ mg/dl on admission but on 20th day of therapy, they noted a significant fall to a level of $85 \pm 14$ mg/dl. They compared the levels in marasmus and kwashiorkor cases and noted a higher level in marasmus, the cause they suggested, was large production of endogenous plasma triglycerides from circulating FFA, whereas in kwashiorkor cases the lipid accumulated in the liver as liver was unable to dispose of fatty acids adequately.

Truswell and Hansen (1969), and Taylor (1971), noted similar changes in triglyceride levels in kwashiorkor cases as noted by previous workers Mac Donald, 1963; Lewis et al, 1964; Jaya Rao and Krishna Prasad, 1966, but Taylor found that the raised levels persisted up to 20th day of therapy and then fell to initial level. Agbedana (1979), studied
triglyceride levels in kwashiorkor and marasmus cases in the severe stage and found slightly raised levels of 114 ± 19 mg/dl in kwashiorkor as compared to control levels of 90 ± 6.0 mg/dl, but this difference was not significant. In marasmic cases the levels of 98 ± 19 mg/dl were similar to control cases.

Cravioto, et al (1959), studied serum lipoprotein levels in kwashiorkor cases and found significantly low levels of alpha and beta lipoproteins at admission time, which rose after dietary therapy reaching to a maximum and then, either levelled off or decreased. They also found that in two of the cases the initial levels of beta lipoproteins were either normal or higher than normal and after therapy the levels decreased. They further correlated these changes with the changes occurring in normal children from birth to the end of childhood, and noted a similarity. They concluded that the biochemical changes, independent of age, sex and severity of malnutrition were normal finding in much younger well nourished children.

Chatterjee and Chaudhuri (1961) also reported significantly low levels of alpha lipoproteins
in PCM cases at the severe stage of disease. Truswell, et al (1969), reported variable levels of alpha lipoproteins in kwashiorkor cases at admission and commented that the electrophoresis technique, by which the previous workers had reported low levels, was not a reliable method. Gurson, et al (1973), studied marasmic cases and found significantly low fractions of alpha lipoproteins as $24 \pm 8.3\%$ of total lipoproteins, as compared to the normal control fraction of $32 \pm 9\%$. They noted a rise in alpha lipoproteins fraction as recovery occurred. They did not find any change in the beta lipoprotein fractions either at the initial phase or after therapy as compared to the control fraction. They explained the low fraction of alpha lipoproteins in their study were due to significantly low levels of albumin in plasma as compared to the normal control levels.