DISCUSSION
The present study was carried out on 36 malnourished and 10 control infants and children. Children suffering from primary liver disorders of diseases like Diabetes mellitus, Primary hypertension, Myxoedema, Renal disorders and Malaria etc. affecting the total lipids or its fractions, were not included in the control as well as the study group. Observations have been presented in Table 1 to 7. A critical analysis of our data with tangible inference is dealt herewith.

In the undernutrition group, which comprises the biggest group of malnourished population, only 2 cases were admitted for the study, as it was difficult to convince the parents for the admission and the sick children of this group admitted were suffering from diseases affecting the lipid status, so an appreciable number could not be included in the present study. These cases, though investigated, were not included for statistical analysis.
The mean age of marasmus group in our study was 23.1 months, similar to the observation made by Lewis et al (1964). Gurson and Saner (1960), Rao et al (1969), Agbedana (1979) have reported the mean age in marasmus to be between 1-2 years, but Mclaren (1966) and Hijazi (1974) reported the mean age as less than 1 year. Mclaren (1966) suggested that early and abrupt weaning associated with diluted and unhygienic formulae, repeated infections and starvation therapy leads to nutritional marasmus in infancy. In our study besides other social factors, prolonged breast feeding (upto 16 months, range 4-36 months) and delayed weaning could be the important causes of the higher mean age of marasmus group.

In our study, the mean age of the kusahaan group was 31 months, which was similar to the finding of Agbedana (1979). The range of age noted by Ramnathan (1955), Schwartz and Dean (1957), MacDonald et al (1963), Lewis et al (1964, 1966), Jaya Rao and Krishna Prasad (1966), Mclaren (1966), Rao et al (1969), Agbedana (1979) was 1-3 years. The probable explanation of higher mean age being that kusahaan is a nutritional imbalance due to low
protein and rich carbohydrate diet frequently as a result of prolonged breast feeding and weaning on to traditional starchy family diet prevalent in this area.

During follow up of the marasmic group after starting the dietary therapy, the mean weight rose significantly from the pretreatment level to the level on 1st follow up and continued to rise significantly till 2nd follow up (Table-5). Brooke and Wheeler (1976) recorded a more pronounced rise in weights, but their follow up weights were recorded on 2nd week and 4th week after dietary therapy.

In the kwashiorkor group, after starting the dietary therapy, the weight rose significantly from the pretreatment stage to 2nd follow up in each case. One of the 5 cases of kwashiorkor left against medical advice after 1st follow up, therefore the mean weight of remaining 4 cases, as shown in Table-5, appears to be apparently lower than the initial mean weight of 5 cases. Brooke and Wheeler (1976) in their study, noted an initial fall in the weight on 1st follow up, which was due to loss of oedema, then the weight started rising as noted on 2nd follow up after 4 weeks of dietary therapy. In our study,
it took on an average 7 days time for oedema to disappear and then the kwashiorkor cases started gaining weight.

In the present study, in the marasmic kwashiorkor group, the weight started rising significantly from the time of admission to the 1st follow up and continued till 2nd follow up. The pattern of weight gain in our study was similar to the pattern noted by Brooke and Wheeler (1976) and Kumar et al (1983).

In marasmic group, the rate of weight gain in our study was significantly higher than those in kwashiorkor and marasmic kwashiorkor groups (Table-6). Waterlow (1961) and Ashworth (1968) have also reported similar finding in their studies on weight gain. In the kwashiorkor group, the rate of weight gain in our study was not significantly different from that in the marasmic kwashiorkor group. Suraj Gupta (1979) reported that after initial 10-14 days of dietary therapy, the rate of weight gain in both the marasmic and kwashiorkor group is same i.e. 10-15 gm/day. Lily Philip et al (1982) observed a difference in weight gain in
marasmic children if Nicotinic acid was added to the diet. In the children without Nicotinic acid in the diet, rate of weight gain was 171.81 gm/kg/month, whereas in those children, in whom Nicotinic acid was added, in the diet, rate of weight gain was 231.05 gm/kg/month.

In the present study serum total lipid levels of the control group were higher than those of all the malnourished groups (Table-2 Fig.1). Schwartz and Dean (1957), Cravioto et al (1959) and MacDonald et al (1963) reported low levels of serum total lipids in kwashiorkor cases at the time of admission, whereas van Der Sar (1951) reported variable levels in kwashiorkor. Contrary to our findings Hansen (1948), Monckberg (1968) and Gunson et al (1973) reported normal levels of total lipids in marasmus groups at the time of admission.

In kwashiorkor, prolonged poor nutrition results in reduction in the intestinal absorption of different fat fractions (Viteri, Flores and Bahar, 1966) which is further reduced by attacks of diarrhea. These children also have an abnormally high content of free bile acids throughout the small intestine and show distorted intestinal villi with a change in
structure from finger like to leaf and convoluted villi (Schneider and Viteri, 1972). Both malnutrition and diarrhea also produce a decrease in concentration of conjugated bile acids, which are essential for solubilization of lipids in the intestinal lumen and their absorption through formation of lipid micelles (Schneider and Viteri, 1974a). The transport of fat from the intestine to the liver is not primarily altered (Viteri and Schneider, 1974) but the transport of fat from the liver to the tissues as low density lipoproteins is markedly reduced, because of impaired production of the protein moiety of β lipoproteins.

The fatty liver, characteristic of kwashiorkor is perhaps due to increased fat transport from the adipose tissues to liver (Lewis et al, 1964) to decreased β lipoproteins synthesis (Flores et al, 1970) and possibly due to increased liver lipogenesis (Netoff, 1975).

Schwartz and Dean (1957), Cravioto et al. (1959) and MacDonald (1963) suggested that the low levels of total lipids were due to reduced levels of triglycerides, cholesterol, phospholipids and
lipoproteins. During the active stage of disease in kwashiorkor group, Sriniwasan and Patwardhan (1952) observed decreased activity of plasma esterase, lipase and amylases, which results in low levels of total lipids.

In our study, the serum total lipid levels at the time of admission in marasmic group were found to be significantly higher than in the kwashiorkor group (Table-2). This observation was consistent with the finding reported by Schwartz and Dean (1957), Cravioto et al. (1959), MacDonald et al. (1963), Hansen (1968), Monokberg (1968) and Gurson et al. (1973). Gurson et al. (1973) suggested that similar to mechanisms controlling proteins and carbohydrate homeostasis, the marasmic infants probably maintain a balanced source for lipids as a result of which the plasma levels of total lipids remain within normal limits.

Serum total lipid levels in the marasmic kwashiorkor group were found to be, as expected, in between the marasmic and kwashiorkor levels. In our study these levels were significantly different from the control and marasmic levels, but were insignificant when compared to the kwashiorkor group (Table-2 Fig-1). This marasmic kwashiorkor group
forms a major group of the malnourished population in this region. Serum total lipid levels in this group have not been described separately by other workers. Malnutrition cases with oedema have been either described as Kwashiorkor (Williams, 1933; Davies, 1948; van der Sar, 1951; Dean and Schwartz, 1953; Schwartz and Dean, 1957; etc.) or Nutritional oedema syndrome (Gopalan and Patwardhan, 1951; Venkatachalam, Sri Kantia and Gopalan, 1954).

In the marasmus group, the serum total lipid levels showed a significant rise on 1st follow up and then there was an insignificant fall on 2nd follow up as compared to the 1st. Prathum (1968), Konickberg (1968) and Gurson et al. (1973) have reported no significant change in the follow up levels in marasmus group. It is to be recalled that pretreatment total lipid values were also not different from the controls. Gurson et al. (1973) have reported low levels of phospholipids at admission as compared to controls, in follow up they found further decrease in the phospholipids. Reduction of this fraction of total lipids may be cause of reduced total lipid levels in our study.
In the present study in the kwashiorkor, the serum total lipid level at the time of admission was significantly low as compared to controls, which rose significantly on the 10th day of therapy and then fell insignificantly on 20th day, but the level still remained significantly higher than the level at admission time. Our observation was consistent with the observations made by Schwartz and Dean (1957), Cravioto et al (1959) and MacDonald et al (1963). Schwartz and Dean (1957) explained the rise in total lipid levels after dietary therapy to be due to sudden increase in neutral fat and esterified cholesterol levels which was to some extent brought by the diet provided, but mainly it was due to the release of preformed fat and cholesterol from the stores. The fall in the level of total lipids that succeeded the initial rise presumably represented utilisation of fat in a normal manner. Cravioto et al (1959) compared the changes occurring in the lipids during recovery from kwashiorkor to those of the new born babies, and found a striking resemblance. They further observed that these changes being independent of the amount and the kind of the diet consumed and also of the age and sex of the patients. MacDonald et al (1963) suggested that the increase in the total lipid levels was due to the increase in
cholesterol, glycerides and phospholipids, and the fall in the total lipid levels after 10 days therapy was almost entirely due to the sharp fall in glyceride fraction.

In the marasmic kwashiorkor group, in the present study, the total lipids rose significantly from the initial level to reach to the control level on 10th day and then showed a fall though insignificant. The observed values were in between the marasmus and kwashiorkor group values.

In the present study serum cholesterol levels in all the malnourished groups were lower than the control group. Our finding was consistent with the observations made by Van Der Sar (1951), Dean and Schwartz (1953), Workers from Nutrition Research lab. Coonoor South India (1954), Ramnathan (1955), Schwartz and Dean (1957), Cravioto et al (1959), Schendel and Hansen (1961), MacDonald et al (1963), Lewis et al (1964), Jaya Rao and Krishna Prasad (1966), Taylor (1971), Debnath (1972) and Agbadana (1979) in kwashiorkor cases. Our findings were similar to the findings noted by Truswell and Hansen (1969) and Debnath (1972) in marasmus cases. The various explanations suggested for the decreased cholesterol levels in kwashiorkor are decreased activity of plasma

Truswell and Hansen attributed decreased levels of plasma lipoproteins to be the cause of low level of cholesterol in marasmus. Debnath (1972) suggested that poor nutrition results in reduced amount of body fat, reduced endogenous fat metabolism leading to hypocholesterolaemia. Agbedana (1979) suggested a defective mobilization of liver lipids to be a cause of decreased cholesterol levels in plasma and thought that decreased hepatic lipoprotein lipase may be responsible for it. Lewis et al (1964) and Gurson et al (1973) found normal levels of serum cholesterol in the marasmic group. Gurson et al (1973) suggested that marasmic cases probably maintain a balanced source for lipids as a result of which the plasma levels of cholesterol remain within normal limit.
The serum cholesterol levels at admission in marasmic group were found to be significantly higher than in the kwashiorkor and marasmic kwashiorkor groups. In our study, our finding was similar to the findings reported by Van Der Sar (1951), Dean and Schwartz (1953), Workers from Nutrition Research lab. Coonoor (1954), Ramnathan (1955) etc.

The serum cholesterol level in the kwashiorkor group and marasmic kwashiorkor group were insignificantly different. The serum cholesterol level in the marasmic group was significantly lower than the level in control group (Table-3, Fig.2), similar observation was made by Debnath (1972). After 10 days of dietary therapy the levels increased and came to the control levels and then persisted at this level even on 20th day of therapy. The pattern of changes in cholesterol levels in marasmic group, observed in our study, was consistent with the pattern observed by Debnath (1972), Lewis et al (1964) and Gurson et al (1973) on the other hand, noted no significant changes in the serum cholesterol levels before or after therapy.

In the kwashiorkor group, the serum cholesterol level, before starting the dietary therapy was significantly low as compared to the level in control group. Our finding was similar to the findings
noted by all the previous workers. After initiating the dietary therapy, the cholesterol levels increased significantly on 10th day and crossed the control levels and remained so till our 2nd follow up. Similar observations have been noted by Dean and Schwartz (1953), Nutrition Research Lab. Coonoor (1954), Schwartz and Dean (1957), Schendel and Hansen (1961), MacDonald et al (1963), Lewis et al (1964), Jaya Rao and Krishna Prasad (1966), Debnath (1972) and Agbedana (1979).

Schendel and Hansen (1961) reported that the rise in cholesterol level appeared to be maximal during the first two weeks of adequate dietary treatment and they suggested that the serum concentration of cholesterol reflect the rate at which liver fat is cleared from stores during recovery. They further observed that an arrested rise or fall was associated with the onset of complications, or inadequate therapy. Contrary to the explanation given by Schwartz and Dean (1957), Jaya Rao and Krishna Prasad (1966) attributed the rise in cholesterol level solely to the caloric intake. Debnath (1972) suggested that the protein in the diet helped in the discharging lipids from the liver in the form of lipoproteins.

Schwartz and Dean (1957), MacDonald et al (1963), Lewis et al (1964), Jaya Rao and Krishna
Prasad (1966) and Debnath (1972) reported a return of serum cholesterol levels to the control level on 20th day of therapy, whereas similar to our findings, Ramnathan (1955), Schendel and Hansen (1961), Taylor (1971), noted persistently higher levels even on 20th day of therapy, thus showing a delayed fall in the cholesterol levels.

In the marasmic kwashiorkor group the serum cholesterol level was significantly lower than the control group level in the pretreatment phase and it was not significantly different from the level in kwashiorkor group. On 1st follow up the cholesterol level rose significantly to a level higher than the control level and then on 2nd follow up the level fell and came to the control group level.

In the present study, serum free fatty acid levels in the kwashiorkor and marasmic kwashiorkor groups were found to be significantly higher than in the control group. The free fatty acid levels in the marasmic group were insignificantly different from the control levels. Higher levels of serum free fatty acids in kwashiorkor group before starting the therapy, have been noted by Lewis et al (1964, 1966) Jaya Rao and Krishna Prasad (1966) Hadden (1967), Agbedana (1979), Hadden (1967), Gurson and Saner (1969)
Gurson et al (1973) and Agbedana (1979) reported normal levels of serum free fatty acids in marasmic group. Lewis et al (1964, 66) reported higher levels of free fatty acids in serum in marasmic children and suggested that the rate of release of free fatty acids from adipose tissue is reciprocally related to the extent of carbohydrate utilization. Glucose administration depresses the free fatty acid production and plasma free fatty acid levels, whereas prolonged starvation is associated with elevated plasma levels.

They further suggested that in kwashiorkor calorie deficiency is responsible for the rise in free fatty acid levels. They divided the course of kwashiorkor into two phases—a chronic phase characterized by low protein, relatively adequate carbohydrate diet, advancing protein depletion and reasonable caloric balance and a later acute phase in which reduced intake and malabsorption produced a state of general undernutrition. In acute stage there is little fat synthesis from carbohydrate which is otherwise seen at adipose organs in early phase. Lewis et al (1964) concluded that increased flux of fatty acids from adipose tissue to the liver was responsible for the elevated plasma free fatty acid levels. Jaya Rao and Krishna Prasad suggested that the high circulating levels of non-esterified fatty acids could be due to hepatic damage
causing defective uptake of nonesterified fatty acids or that nonesterified fatty acid levels represented an alternate source of energy to tissues.

Hadden (1967) suggested a block at the point of entry of short chain of fatty acyl coenzyme A into the Kreb cycle and temporary impairment of carbohydrate tolerance as responsible for the high levels of free fatty acids. Agbedana (1979) proposed the cause for high levels of free fatty acids in serum to be due to effect of growth hormone and insulin in kwashiorkor.

Serum free fatty acid levels in marasmic group were found to be significantly lower than in the kwashiorkor and marasmic kwashiorkor group. Our observation was similar to the findings reported by Hadden (1967) and Agbedana (1979). It could be because the children of kwashiorkor and marasmic kwashiorkor group face more stressful situation than marasmus and probably because of the same reason, the free fatty acid levels in kwashiorkor group were significantly higher than in the marasmic kwashiorkor group.

In the present study the serum free fatty acid level at the time of admission in marasmic group was not significantly different from the control level.
(Table-4, Fig. 3). After starting the dietary therapy, on 10th day and on 20th day the levels remain unchanged. A similar pattern was observed by Hadden (1967), Gurson and Saner (1969), Gurson et al (1973). As in kwashiorkor, high levels of free fatty acids were reported in marasmus before starting dietary therapy, by Lewis et al (1964), which came down drastically on 1st follow up and then rose to the control level on 2nd follow up.

In the kwashiorkor group, the free fatty acid levels in serum were significantly higher than the control group which after instituting the dietary therapy, fell significantly to a level lower than the control on 1st follow up and then rose insignificantly on further follow up. Similar observation was noted by Lewis et al (1964), Jaya Rao and Krishna Prasad (1966) and Hadden (1967). Lewis et al (1964) proposed that the calorie mainly in the form of carbohydrate provided in the kwashiorkor cases was responsible for the sudden fall in free fatty acid levels. After 20 days therapy the levels were insignificantly different from the control levels. Jaya Rao and Krishna Prasad (1966) suggested that after starting the therapy the defective uptake of free fatty acids by the liver might have corrected.
thus resulting in sudden fall in nonesterified fatty acid levels.

In the marasmic kwashiorkor group before starting the dietary therapy, in our study the free fatty acid levels were significantly higher than the control group levels and were in between the marasmic and kwashiorkor group levels. After 10 days of dietary therapy the levels fell significantly and then showed an insignificant rise to come to the control levels. The pattern was similar to the pattern observed in kwashiorkor cases.

In our study, serum albumin levels in the control group were found to be significantly higher than those in all the malnourished groups (Table-7, Fig.4). Our finding was consistent with the findings of Lewis et al (1966) and Debnath (1972). In kwashiorkor group, Ramnathan (1955) and MacDonald et al (1963) have reported low serum albumin levels in the pretreatment phase. Gunson et al (1973) have noted low levels of serum albumin in marasmic groups.

Hypoalbuminaemia has been attributed as a basic change in kwashiorkor. Cohen and Hansen (1962) suggested that the total albumin pool is decreased, the intravascular compartment being less affected than the extravascular. The changes in albumin concent-
ration were supposed to be the consequence of decreased rate of synthesis, which occurs before the rate of catabolism becomes reduced. James and Hay (1968) reported that in kwashiorkor the albumin turnover is lower than normal and the half life is prolonged. The other important causes of low albumin levels in kwashiorkor are—the protein turnover rate in muscles is low, synthesis rate is decreased while that of catabolism is low when carbohydrate intake is enough to suppress gluconeogenesis and normal if energy deficiency is there (Felig et al. 1966; Waterlow, 1975) and the low levels of plasma amino acids.

In our study the serum albumin levels in pretreatment stage of marasmic group, was found to be significantly higher than the levels in kwashiorkor and marasmic kwashiorkor group (Fig.4). Our finding was similar to the finding noted by Lewis et al. (1964) and Debnath (1972). Serum albumin levels though higher in marasmic kwashiorkor group, were not statistically different than the levels in kwashiorkor group.

In the present study the serum albumin levels in marasmic group, after starting the therapy, rose significantly to reach the control levels on 1st follow up and then continued to rise. When paired
T-test was applied the difference between the levels on 1st and 2nd follow up were significantly different. Lewis et al (1964), Debnath (1972) and Gurson (1973) also noted a progressive rise in the serum albumin levels in marasmic group. Lewis et al (1964) and Debnath (1972) did not compare their values of serum albumin with the control levels, and Gurson et al (1973) studied the albumin levels before starting the therapy and after completing the therapy (21 to 37 days).

In our study in the kwashiorkor group, the mean serum albumin level observed before starting the therapy were comparable with the findings of Ramnathan (1955), MacDonald et al (1963) and Debnath (1972). These serum albumin levels in our study rose significantly on 1st follow up and continued to rise till reached to a level, insignificantly different from the control level on 2nd follow up. The rising pattern of serum albumin in our study was similar to the patterns observed by Ramnathan (1955), MacDonald et al (1963), Lewis et al (1964) and Debnath (1972). The level achieved on 2nd follow up in our study was similar to the level observed by MacDonald et al (1963), but was higher than those observed by Ramnathan (1955).
Lewis et al (1964) and Debnath (1972).

In the marasmioc kwashiorkor group in our study, the serum albumin levels rose progressively and significantly from the pretreatment level to the control level in 20 days after starting the dietary therapy. The rising pattern in this group was similar to the pattern in kwashiorkor group.