CHAPTER - III

R
E
S
U
L
T
S

AND

D
I
S
C
U
S
S
I
O
N

STUDIES ON FETAL TISSUE STORES OF IRON IN RELATION TO MATERNAL IRON STATUS, GESTATIONAL AGE AND FETAL GROWTH STATUS.

Blood hemoglobin levels at birth are found to be of the order of 20-22 g/dl (Toverud et al, 1950; Lanzkowsky, 1960; Bhatt et al, 1969), but this value declines to about 11 g/dl by the age of six months even in well nourished populations (Sturgeon, 1954; Guest and Brown, 1957). This decline in hemoglobin concentration is compensated by an increase in total hemoglobin from an estimated 62g at birth to 71g at six months because of an increase in blood volume from 285 ml to 480 ml. This represents an increase in total circulating iron from 210 mg to 240 mg. Assuming a milk secretion of 700 ml, the total exogenous supply of iron would be of the order of 188 mg for six months and much less than this if the availability of iron which is not more than 20% even under very favourable conditions is taken into account. This means that a substantial portion of the iron needed for the hemoglobin synthesis would have to be met mainly from the body stores acquired during fetal life by placental transfer. Practically all the iron used for hemoglobin synthesis during the first 3-4 months of life was found to be acquired before birth (Smith et al, 1955).

Most of the storage iron present at birth, mainly in the liver, is found to be depleted during the suckling period in order to maintain hemoglobin synthesis in a variety of mammalian species (Bruckmann and Zondek, 1939; Widdowson and Spray, 1951; Smith et al, 1955; Ramage et al, 1975) suggesting the importance of acquiring adequate iron stores before birth.

It has also been suggested that stores acquired before birth are not likely to be adequate in infants born of anemic mother, as such infants show an earlier onset of anemia of infancy (Strauss, 1933; Woodruff and Bridgeforth, 1953; Lanzkowsky, 1961; Bhatt et al, 1969). However, no data are available on the effects of maternal anemia on the amount of iron transferred to the fetus at birth, a question of some importance as the transplacental delivery of iron is replaced before birth by a smaller and less consistent dietary supply after birth.

The present studies were undertaken in this context to study the relation between the maternal iron status and fetal iron stores. Comparative studies were made in women belonging to low ~~k~~ and high income groups and consequently differing in their nutrition status.

The data on fetal weights and liver weights in the low and high income groups are presented in Table-16 and those of spleen and kidney are presented in Tables-17 & 18. As may be expected, the weights of the whole fetus and of the liver, kidney and spleen increased with the progress of gestation. The fetal weights are less than those reported in the west for corresponding gestational age by other investigators (Widdowson, 1968; Warwick and Williams, 1973; Montreewasuwat and Olson, 1979) but the value for 18 weeks were similar to those reported by Abravomich (1969) and Apte and Iyengar (1972). When fetal weights were considered as percentages of expected weights for gestational age using Widdowson's norms (1968), the values for the two groups were found to be comparable in early pregnancy but some deceleration of growth was observed in low income group around mid-pregnancy. The deficits in fetal weight as compared to Widdowson's norms (1968) are consistent with differences in birth weights (Thomson and Hytten, 1966; Rajalakshmi, 1971). The social class differences are in accordance with the higher proportion of small for gestational infants among poor (Achar and Yankauer, 1962; Udani, 1963; Rajalakshmi, 1971). The deceleration of fetal growth in the low income group around mid-pregnancy is consistent with other observations (Bhatt, 1982; Iyengar, 1984). In this

TABLE-16 : Fetal liver iron stores in relation to gestational age and fetal weight in low (LIG) and high (HIG) income groups.

Weeks of gestation		Fetal weight (gm)	Liver weight (g)	Liver iron concentration (mg/g)	Content (mg)
		Mean ± S.E., with no. of subjects in bracket			
∠ 18	LIG	122.0 ± 13.3 (27) (73)†	4.06 ± 0.41 (27)	0.40 ± 0.03 (27)	1.49 ± 0.17 (27)
	HIG	177.0 ± 13.2* (21) (78)†	7.00 ± 0.45* (21)	0.44 ± 0.03 (21)	3.20 ± 0.36* (21)
>18	LIG	433.0 ± 33.0 (47) (64)†	17.38 ± 1.35 (47)	0.36 ± 0.02 (47)	5.86 ± 0.59 (47)
	HIG	344 ± 62.2 (14) (68)†	13.26 ± 2.42 (14)	0.46 ± 0.05 (14)	5.76 ± 0.88 (14)

† Fetal weight as % expected weight as given by Widdowson (1968)

Values significantly different from LIG values, *P ∠0.001.

TABLE-17 : Fetal spleen iron stores in relation to gestational age and fetal weight in low (LIG) and high (HIG) income groups.

Weeks of gestation		Fetal weight (g)	Fetal weight as % expected	Spleen weight (g)	Spleen iron	
					Concentration (mg/g)	Content (mg)
Mean ± S.E.; with no. of subjects in parentheses						
< 18	LIG	133.0 ± 14.3 (17)	73.7 ± 4.13 (17)	0.146 ± 0.024 (17)	0.90 ± 0.30 (17)	0.087 ± 0.022 (17)
	HIG	175.0 ± 27.9 (8)	70.1 ± 4.50 (8)	0.175 ± 0.032 (8)	0.85 ± 0.37 (8)	0.086 ± 0.015 (8)
>18	LIG	619.8 ± 96.6 (38)	64.9 ± 2.10 (38)	0.950 ± 0.236 (38)	0.31 ± 0.031 (38)	0.258 ± 0.070 (38)
	HIG	312.5 ± 68.6 (4)	67.3 ± 4.99 (4)	0.289 ± 0.053 (4)	0.60 ± 0.094** (4)	0.157 ± 0.011 (4)

Values are significantly different from LIG values, ** $P < 0.01$.

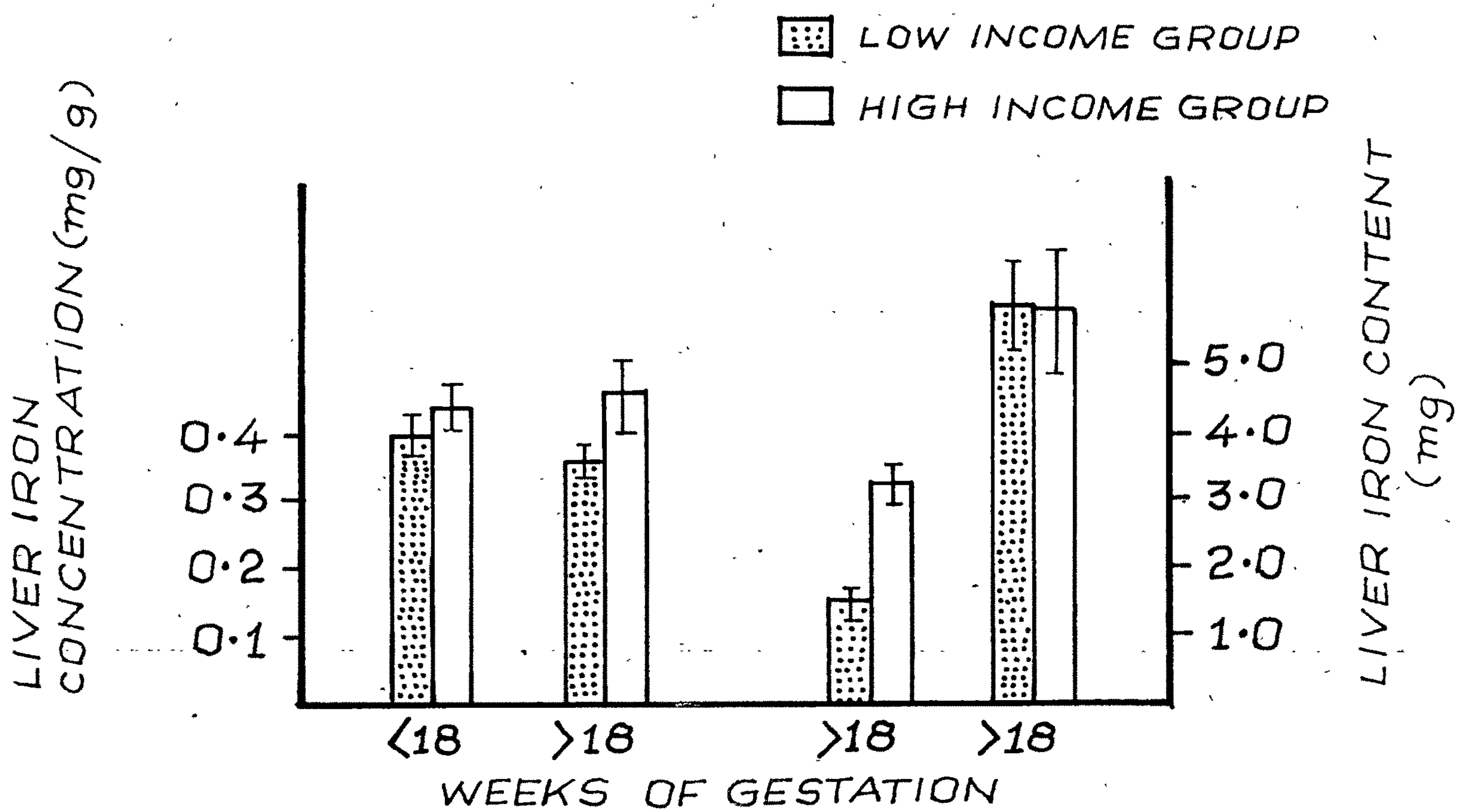
TABLE-18 : Fetal kidney iron stores in relation to gestational age and fetal weight in low (LIG) and high (HIG) income groups.

Weeks of gestation		Fetal weight	Fetal weight as % expected	Kidney weight	Kidney iron: Concentration (mg/g)	Kidney iron: Content (mg)
		Mean ± S.E.; with no. of subjects in parentheses				
< 18	LIG	128.2 ± 18.7 (12)	79.3 ± 4.91 (12)	0.936 ± 0.084 (12)	0.165 ± 0.019 (12)	0.153 ± 0.024 (12)
	HIG	231.7 ± 12.2 (3)	77.7 ± 2.41 (3)	2.308 ± 0.320 (3)	0.123 ± 0.024 (3)	0.269 ± 0.033 (3)
> 18	LIG	334.1 ± 35.5 (16)	62.5 ± 3.27 (16)	2.500 ± 0.229 (16)	0.124 ± 0.012 (16)	0.300 ± 0.032 (16)
	HIG	305.2 ± 53.5 (5)	65.8 ± 4.12 (5)	2.639 ± 0.959 (5)	0.134 ± 0.022 (5)	0.282 ± 0.047 (5)

connection,the concentration of several nutrients such as vitamin A, calcium in fetal tissues and overall growth is accelerated after mid-pregnancy when nutritional requirements for the growing fetus are much more than at early stages. It is not surprising that fetal growth is more affected in later stages of pregnancy by maternal malnutrition.

Liver iron concentration did not show much variation with gestational age and no significant difference was observed between the two groups with regard to liver iron concentration but a significant difference in content was found because of fetal size, and consequently liver size (Table-16)(Figure-7). Spleen and kidney iron concentration decreased with the progress of gestation but content increased and no difference was observed between the two groups with regard to their concentration in early pregnancy but the difference was significant in later pregnancy in case of spleen,whereas total amount differed because of difference in fetal size, and consequently spleen and kidney size (Tables-17 & 18). These observations are supported by Apte and Iyengar (1972) who found concentrations of 0.31, 0.33 and 0.29 mg/g for gestational ages 24-28, 28-32 and 32-36 weeks respectively. However, for the newborn infant it is the amount of iron and not the concentration that is critical. Even with regard to

FIGURE-7 FETAL LIVER IRON STORES IN RELATION TO GESTATIONAL AGE IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.



concentration in liver and kidney, a trend in favour of the high income group is found in late pregnancy but the difference falls short of significance perhaps because of small sample size in the upper class who tend to terminate their pregnancy as soon as possible after conception. In this connection, Bothwell et al. (1958) on the basis of ferrokinetic studies in the rabbit, suggested that the amount of iron going to the fetus is proportional to the increase in weight of the fetus. The ratio of iron to body weight remains relatively constant through out pregnancy at an average level of 75 mg/kg according to Widdowson and Spray (1951). When the present data were calculated in terms of liver iron per kg of body weight social class differences were more apparent, specially in later pregnancy (Table-19).

The values for maternal hemoglobin, serum iron and total iron binding capacity are well in agreement with the values reported by several investigators and previous studies in this laboratory (Dave, 1980). Maternal blood hemoglobin, serum iron and total iron binding capacity did not differ significantly in the two categories (Tables-20, 21 & 22). However, the value of blood hemoglobin and maternal serum iron tended to be higher in the case of the high income group, reflecting differences in iron stores.

TABLE-19 : Fetal liver iron stores expressed as mg/kg body weight in relation to gestational age in low (LIG) and high (HIG) income groups.

Weeks of gestation		Fetal weight (g)	Liver weight as % body weight	Liver iron content (mg)	Liver iron as mg/kg body weight
		Mean ± S.E., with no. of subjects in parentheses			
$\angle$ 18	LIG	122.0 ± 13.3 (27)	3.7 ± 0.24 (27)	1.49 ± 0.17 (27)	15.6 ± 1.86 (27)
	HIG	177.0 ± 13.2 (21)	4.1 ± 0.16 (21)	3.20 ± 0.36*** (21)	18.6 ± 1.84 (21)
>18	LIG	433.0 ± 33.0 (47)	4.1 ± 0.13 (47)	5.86 ± 0.59 (47)	13.1 ± 0.91 (47)
	HIG	344.0 ± 62.2 (14)	3.9 ± 0.16 (14)	5.76 ± 0.88 (14)	18.4 ± 2.46** (14)

Values significantly different from LIG values, *** P $\angle$0.001; ** P $\angle$0.05.

TABLE-20 : Fetal liver iron stores in relation to gestational age and maternal hemoglobin in low (LIG) and high (HIG) income groups.

Gestational age (weeks)		Fetal weight (gm)	Liver weight (g)	Liver iron concentration (mg/g)	Liver iron content (mg)	Maternal blood hemoglobin (g/dl)
		Mean ± S.E., with no.of subjects in bracket				
<18	LIG	105.0 ± 11.09 (20) (72)*	4.07 ± 0.54 (20)	0.41 ± 0.04 (20)	1.48 ± 0.22 (20)	10.9 ± 0.49 (20)
	HIG	162.0 ± 14.86†† (13) (82)*	6.08 ± 0.55† (13)	0.43 ± 0.04 (13)	2.95 ± 0.49 (13)	11.1 ± 0.55 (13)
LIG as % HIG		(65)	(67)	(95)	(50)	
>18	LIG	358 ± 43.5 (21) (64)*	14.11 ± 1.81 (21)	0.34 ± 0.02 (21)	4.61 ± 0.64 (21)	10.6 ± 0.46 (21)
	HIG	359 ± 75.9 (5) (74)*	14.45 ± 9.07 (5)	0.45 ± 0.04 (5)	6.10 ± 0.67 (5)	11.1 ± 0.77 (5)
LIG as % HIG		(100)	(98)	(76)	(76)	

* - fetal weight as % expected weight as given by Widdowson (1968).

Values significantly different from LIG values, † $P < 0.02$, †† $P < 0.01$.

TABLE-21 : Fetal liver iron stores in relation to gestational age and maternal serum iron in low (LIG) and high (HIG) income groups.

Gestational age (weeks)		Fetal weight (g)	Liver weight (g)	Liver iron concentration (mg/g)	Liver iron content (mg)	Maternal serum iron (μg/dl)
		Mean ± S.E., with no. of subjects in parentheses				
$\angle$ 18	LIG	107 ± 12.76 (16) (71)*	4.33 ± 0.63 (16)	0.42 ± 0.04 (16)	1.64 ± 0.26 (16)	104 ± 12.99 (16)
	HIG	156 ± 15.31†† (11) (84)*	6.56 ± 0.64† (11)	0.44 ± 0.05 (11)	3.01 ± 0.59 (11)	104 ± 10.93 (11)
>18	LIG	325 ± 30.3 (21) (63)*	13.09 ± 1.48 (21)	0.35 ± 0.03 (21)	4.54 ± 0.67 (21)	108 ± 11.71 (21)
	HIG	365 ± 97.8 (4) (74)*	15.54 ± 3.71 (4)	0.44 ± 0.05 (4)	6.36 ± 0.81 (4)	138.0 ± 31.8 (4)

* Fetal weight as % expected weight as given by Widdowson (1968)

Values significantly different from LIG values, † P $\angle$0.02, †† P $\angle$0.01

TABLE 22 : Fetal liver iron stores in relation to gestational age and maternal serum total iron binding capacity in low (LIG) and high (HIG) income groups.

Weeks of gestation		Fetal weight (g)	Liver weight (g)	Liver iron concentration (mg/g)	Liver iron content (mg)	Maternal TIBC (μg/dl)
		Mean ± S.E., with no. of subjects in parentheses				
< 18	LIG	128 ± 11.3 (10)	4.95 ± 0.54 (10)	0.38 ± 0.05 (10)	1.84 ± 0.31 (10)	404 ± 18.4 (10)
	HIG	151 ± 21.1 (8)	5.95 ± 0.74 (8)	0.37 ± 0.03 (8)	2.23 ± 0.37 (8)	470 ± 44.2 (8)
> 18	LIG	321 ± 33.3 (19)	12.62 ± 1.50 (19)	0.35 ± 0.02 (19)	4.55 ± 0.69 (19)	448 ± 22.0 (19)
	HIG	280 ± 54.7 (2)	12.42 ± 1.13 (2)	0.51 ± 0.0 (2)	6.36 ± 0.70 (2)	571 ± 42.4 (2)

When the values for liver (total) iron were calculated as percentages of the value at term given by Widdowson and Spray (1951) in the two groups, a much slower acquisition of iron in the low income group is suggested from the data of Table-23. The pattern of accretion in liver is similar to that observed for the accretion of total iron in the body with the progress of gestation reported by Widdowson and Spray (1951).

Since the two groups differed with regard to liver, spleen and kidney iron content, the data on iron content and concentration and fetal weights were analysed as shown in Tables-24, 25 & 26 (Figure-8). Social class differences were found with regard to liver iron content but not with regard to concentration. Social class differences are suggested before mid-pregnancy in iron content even for fetuses of comparable growth status.

The differences in the pattern of accretion of liver iron in the two groups suggests that the preterm infant in the low income group may be more vulnerable to iron deficiency, specially, if postnatal nutrition is also unsatisfactory. This may also affect blood hemoglobin levels at birth and consequently its respiratory capacity. In this connection, poor iron stores in premature infants are reported by Widdowson and Spray (1951). This might

TABLE-23 : Fetal liver iron content as per cent of values at term* in low (LIG) and high (HIG) income groups.

Weeks of gestation	12-16	16-20	20-24	24-28	>28
LIG	3.6	6.3	14.9	19.2	34.2
HIG	7.4	11.0	21.9	-	35.8

Reported values for accretion of iron in fetal body as % of term value†

G.A.	16	20	24	28	32	36	40
%	1.8	6.3	16.5	32.8	50.7	74.8	100

*† Based on value at term given by Widdowson and Spray (1951).

TABLE-24 : Fetal liver iron stores in relation to fetal size and gestational age in low (LIG) and high (HIG) income groups.

Weight as % of expected weight@		Weeks of gestation < 18 Concentration (mg/g)	Weeks of gestation < 18 Content (mg)	Weeks of gestation > 18 Concentration (mg/g)	Weeks of gestation > 18 Content (mg)
		Mean ± S.E., with no. of subjects in bracket			
LIG	< 70	0.46 ± 0.06 (12)	1.38 ± 0.26 (12)	0.38 ± 0.03 (34)	5.71 ± 0.73 (34)
	> 70	0.36 ± 0.04 (14)	1.59 ± 0.26 (14)	0.31 ± 0.03 (13)	6.25 ± 0.94 (13)
HIG	< 70	0.41 ± 0.03 (7)	1.95 ± 0.19 (7)	0.52 ± 0.08 (8)	5.05 ± 1.01 (8)
	> 70	0.46 ± 0.04 (14)	3.82 ± 0.45 (14) **†††	0.38 ± 0.04 (6)	6.71 ± 1.58 (6)

@ Weight as given by Widdowson (1968).

Values significantly different from < 70 values ** $p < 0.01$.

Values significantly different from LIG values ††† $P < 0.001$.

FIGURE-8 FETAL LIVER IRONSTORES[95] IN RELATION TO GROWTH STATUS IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

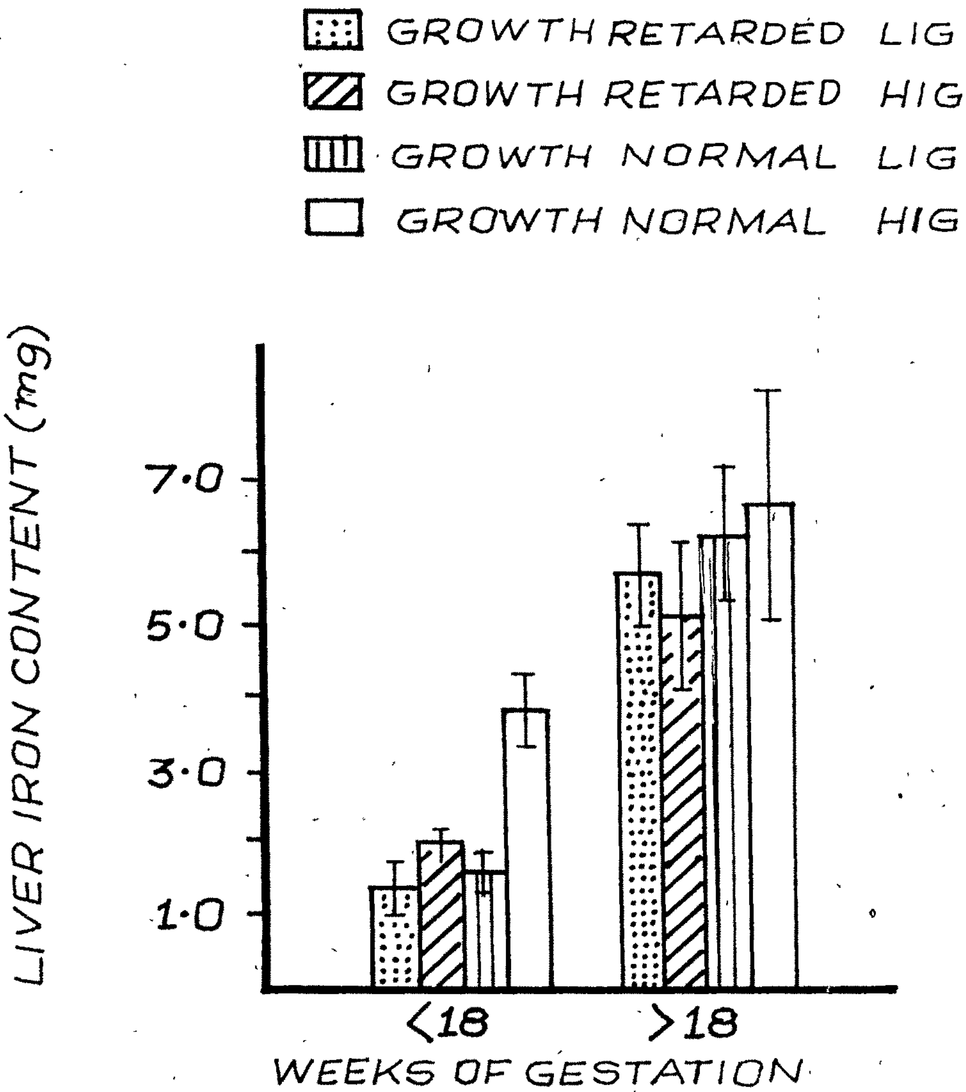


TABLE-25 : Fetal spleen iron stores in relation to fetal size and gestational age in low (LIG) and high (HIG) income groups.

	Fetal weight as % expected@	Weeks of gestation < 18: Fetal spleen iron Concentration (mg/g)	< 18: Content (mg)	> 18: Concentration (mg/g)	> 18: Content (mg)
		Mean ± S.E.; with no. of subjects in parentheses			
LIG	< 70	0.64 ± 0.090 (6)	0.085 ± 0.030 (6)	0.29 ± 0.03 (29)	0.150 ± 0.020 (29)
	> 70	1.05 ± 0.470 (11)	0.089 ± 0.030 (11)	0.40 ± 0.09 (9)	0.590 ± 0.270 (9)
HIG	< 70	1.44 ± 0.970 (3)	0.047 ± 0.009 (3)	0.64 ± 0.120 (3)	0.148 ± 0.008 (3)
	> 70	0.45 ± 0.090 (4)	0.100 ± 0.020** (4)	0.57 ± 0.110 (2)	0.166 ± 0.020 (2)

@ Weight as given by Widdowson (1968).
Values significantly different from < 70 values, ** $P < 0.05$.

TABLE-26 : Fetal kidney iron stores in relation to fetal size and gestational age in low (LIG) and high (HIG) income groups.

	Fetal wt. as % expected@	Weeks of gestation			
		< 18		> 18	
		Fetal kidney iron			
		Concentration (mg/g)	Content (mg)	Concentration (mg/g)	Content (mg)
		Mean ± S.E.; with no. of subjects in parentheses			
LIG	< 70	0.16 ± 0.010 (4)	0.09 ± 0.030 (4)	0.12 ± 0.010 (13)	0.29 ± 0.040 (13)
	> 70	0.16 ± 0.030 (8)	0.17 ± 0.030 (8)	0.14 ± 0.005 (3)	0.37 ± 0.06 (3)
HIG	< 70	-	-	0.15 ± 0.02 (4)	0.24 ± 0.03 (4)
	> 70	0.15 ± 0.010 (2)	0.29 ± 0.040** (2)	0.07 ± 0.004 (2)	0.33 ± 0.11 (2)

@ Weight as given by Widdowson (1968).

Values significantly different from LIG values, ** $P < 0.05$.

also apply to the full term infants subjected to intra-uterine growth retardation as suggested by the data in Table-24. This is consistent with the observation of Hussain (1968) who found no significant difference between the concentration of iron in livers of 26 small and 56 large babies at birth but the total amount of liver iron in small babies was significantly less than that in the livers of large babies. A similar pattern is observed with regard to total body iron at birth which is found to be less in the low birth infant, although, the ratio of total iron to body weight is not affected (Worwood, 1977). Since low birth weight infants (including premature infants) have a more rapid rate of postnatal growth under satisfactory conditions, they are likely to exhaust their iron stores at an earlier age, so tha t the requirements for exogenous iron in such infants may be greater (Shculman and Smith, 1954; Gorten and Cross, 1964; Lundstrom <u>et al</u>, 1977; Committee on Nutrition, 1976) so that the dependence on dietary iron begins earlier (Layrisse <u>et al</u>, 1974; Bezwoda <u>et al</u>, 1979). In these infants, the nadir in concentration of hemoglobin is much lower than in the normal weight term infant and is followed by a much higher rise in reticulocytes count.

A further analysis of data showed that variations in maternal blood hemoglobin levels had no impact on liver iron with regard to either concentration or content (Table-27). This was also true of maternal serum iron and serum total iron binding capacity in the low income group (Tables-28 & 29). But in the earlier stages of pregnancy in the high income group liver iron concentration was affected by maternal serum total iron binding capacity (Table-29).

These observations are consistent with those of Lanzkowsky (1976) who found no effect of maternal iron status on fetal iron stores and number of others (Bothwell and Finch, 1962; Bothwell, 1966; Hunter, 1978; Murray et al, 1978; Murray and Stein, 1971) who found no effect on neonatal iron stores. It is also reported that the transfer of iron across the placenta is unidirectional and an active process, independent of maternal iron status and may even induce iron deficiency in the mother to accomodate the demands of the fetus (Lanzkowsky, 1961; Lanzkowsky, 1961a; Shott et al, 1972). Ahmed et al. (1983) have reported a correlation between maternal hemoglobin levels and total fetal stores (expressed as mg/kg body weight) but fetal age is not specified. This trend was not found in the present studies.

TABLE-27 : Fetal liver iron stores in relation to maternal blood hemoglobin levels and fetal weights in low (LIG) and high (HIG) income groups.

Weeks of gestation	Maternal blood hemoglobin (g/dl)	Fetal weight as % expected		Fetal liver iron			
				Concentration (mg/g)		Content (mg)	
		LIG	HIG	LIG	HIG	LIG	HIG
		Mean ± S.E.; with no. of subjects in parentheses					
< 18	< 11	71 ± 4.56 (9)	80 ± 4.52 (5)	0.43 ± 0.05 (9)	0.42 ± 0.08 (5)	1.44 ± 0.22 (9)	3.21 ± 0.73 (5)
	> 11	72 ± 4.59 (9)	76 ± 7.57 (7)	0.34 ± 0.08 (9)	0.39 ± 0.03 (7)	1.32 ± 0.40 (9)	2.16 ± 0.34 (7)
> 18	< 11	64 ± 3.88 (14)	72 (1)	0.36 ± 0.04 (14)	0.51 (1)	4.74 ± 0.93 (14)	5.10 (1)
	> 11	69 ± 5.73 (11)	71 ± 11.48 (3)	0.33 ± 0.03 (11)	0.48 ± 0.03 (3)	6.57 ± 2.90 (11)	5.74 ± 0.74 (3)

TABLE-28 : Fetal liver iron stores in relation to maternal serum iron and gestational age in low (LIG) and high (HIG) income groups.

Weeks of gestation	Mat. serum iron (ug/dl)	Fetal weight as % expected		Fetal liver iron			
				Concentration (mg/g)		Content (mg)	
		LIG	HIG	LIG	HIG	LIG	HIG
		Mean ± S.E.; with no. of subjects in parentheses					
< 18	< 90	73 ± 6.95 (7)	92 ± 8.23 (4)	0.46 ± 0.06 (7)	0.33 ± 0.01 (4)	1.62 ± 0.45 (7)	1.68 ± 0.24 (4)
	>90	69 ± 3.91 (9)	70 ± 3.84 (6)	0.38 ± 0.06 (9)	0.46 ± 0.07 (6)	1.65 ± 0.33 (9)	3.19 ± 0.65 (6)
> 18	< 90	59 ± 2.96 (10)	93 (1)	0.38 ± 0.04 (10)	0.52 (1)	4.07 ± 1.07 (10)	7.07 (1)
	>90	68 ± 5.20 (12)	60 ± 4.51 (2)	0.32 ± 0.04 (12)	0.46 ± 0.04 (2)	7.48 ± 2.65 (12)	5.08 ± 0.57 (2)

TABLE-29 : Fetal liver iron stores in relation to maternal serum total iron binding capacity in low (LIG) and high (HIG) income groups.

Weeks of gestation	Mat. serum TIBC (ug/dl)	Fetal weight as % expected		Fetal liver iron			
				Concentration (mg/g)		Content (mg)	
		LIG	HIG	LIG	HIG	LIG	HIG
		Mean ± S.E.; with no. of subjects in parentheses					
∠ 18	∠ 450	71 ± 6.56 (7)	69 ± 5.3 (4)	0.45 ± 0.05 (7)	0.47 ± 0.05 (4)	1.98 ± 0.37 (7)	2.62 ± 0.74 (4)
	>450	75 ± 6.90 (3)	94 ± 7.00 (4)	0.23 ± 0.09 (3)	0.29 ± 0.03* (4)	1.51 ± 0.66 (3)	1.86 ± 0.10 (4)
>18	∠ 450	64 ± 4.82 (11)	-	0.37 ± 0.04 (11)	-	5.25 ± 1.12 (11)	-
	>450	62 ± 4.81 (8)	74 ± 19.1 (2)	0.35 ± 0.04 (8)	0.51 ± 0.01 (2)	3.60 ± 0.48 (8)	6.36 ± 0.71 (2)

Values significantly different from ∠450 values, * P ∠0.05.

In conclusion, liver, spleen and kidney iron stores in human fetuses were found to increase with the progress of gestation, the accretion of the same being poor in the low income group mainly because of the smaller size of the fetus. It also appears that fetal size rather than maternal iron status is a critical determinant of liver, spleen and kidney iron stores, and that, even with anemia in the mother, iron is preferentially transferred to the fetus. However, premature infants and full term infants with low birth weights are at greater risk of earlier onset of anemia because of a smaller size of their liver stores.

However, it must be pointed out that the present studies were confined to tissue iron and no attempts were made to determine hemoglobin in fetal blood or estimate blood volume. It is quite conceivable that maternal iron status has an impact on circulating levels of iron in the form of hemoglobin as differences in fetal size can be expected to be associated with differences in blood volume and possibly blood hemoglobin levels.

STUDIES ON NEONATAL IRON STATUS IN RELATION TO MATERNAL IRON STATUS, GESTATIONAL AGE AND BIRTH WEIGHT

Maternal iron deficiency and anemia have been reported to influence iron metabolism in the infant and to be associated with a high incidence of preterm and low birth weight babies (Widdowson and Spray, 1951; Evers, 1966; Roszkowski et al, 1966; Whiteside et al, 1968; Fleming, 1974; Wintrobe, 1974; Bogden et al, 1978; Singla et al, 1978) and poor iron stores at birth resulting in an earlier onset of anemia of infancy (Strauss, 1933; Woodruff and Bridgeforth, 1953; Lanzkowsky, 1961; Bhatt et al, 1969). Such anemia also accounts for a major proportion of maternal mortality, about 20% in regions such as Gujarat. On the other hand, many pregnant women with hemoglobin concentrations in the range of 3-5 g/dl pass through labour without any apparent ill effects (Tasker et al, 1956). The results of studies on the correlations between iron levels of the maternal and cord sera are conflicting with the few investigators showing a very high correlation (Sanchez-Medal, 1969; Nhonoli et al, 1975; Singla et al, 1978; Zittoun et al, 1983) and others showing no such correlation (Vahlquist and Serumeisen, 1941; Sturgeon, 1959; Shott et al, 1972; Murray et al, 1978a; Jansson et al, 1979; Bartlid and Moe, 1980; Vobecky et al, 1982). Previous studies in this laboratory on cord and maternal blood hemoglobin levels suggested a significant correlation between the two (Rajalakshmi and Ramakrishnan, 1969).

Thus present studies were undertaken in this context on the iron status of the neonate in relation to gestational age, body weight and maternal iron status. Iron status in both neonates and mothers was assessed by blood hemoglobin, serum iron and total iron binding capacity.

The mean birth weights (kg) of full term infants were 2.70 $\pm$ 0.06 in the low income group and 2.94 $\pm$ 0.06 in the high income group (Tables 30 & 31). The mean birth weights of infants born in low and high income groups are similar to those reported by others (Kulkarni, 1959; Achar and Yankauer, 1962; Udani, 1963; Rajalakshmi et al, 1978). The social class differences are in accord with the higher proportion of small-for-gestational age infants among the poor (Achar and Yankauer, 1962; Udani, 1963; Rajalakshmi et al, 1978). The difference was greater in the premature infants. The mean birth weights (kg) in this category being 2.23 $\pm$ 0.14 and 2.55 $\pm$ 0.14 respectively in the two groups although the mean gestational ages was somewhat greater in the former (35 weeks) than in the latter (33 weeks). The possibility of some catch up growth

TABLE-30 : Hemoglobin and serum iron concentrations in maternal and cord blood in low (LIG) and high (HIG) income groups.

Weeks of gestation		Birth weight (kg)	Blood hemoglobin (g/dl)		Serum iron (µg/dl)	
			Cord	Mother	Cord	Mother
		Mean ± S.E.; No. of subjects in parentheses				
< 36 (34.6 ± 0.28)	LIG	2.16 ± 0.11 (9)	15.5 ± 0.85 (9)	11.4 ± 0.72 (9)	176.8 ± 13.5 (9)	109.1 ± 8.95 (9)
< 36 (33.6 ± 0.59)	HIG	2.56 ± 0.13** (9)	14.6 ± 0.63 (9)	11.8 ± 0.62 (9)	166.2 ± 12.8 (9)	85.1 ± 6.46 (9)
> 36 (38.3 ± 0.33)	LIG	2.70 ± 0.06+++ (56)	14.8 ± 0.48 (56)	10.5 ± 0.43 (56)	185.9 ± 7.32 (56)	89.7 ± 5.01 (56)
> 36 (38.0 ± 0.28)	HIG	2.94 ± 0.06***+++ (26)	15.5 ± 0.38 (26)	15.5 ± 0.41** (26)	190.2 ± 11.7 (26)	99.1 ± 9.35 (26)

Values significantly different from premature values +++ $P < 0.001$,

Values significantly different from LIG ** $P < 0.05$, *** $P < 0.01$.

TABLE-31 : Serum iron levels in maternal and cord blood in low (LIG) and high (HIG) income groups.

Weeks of gestation		Birth weight (kg)	Serum iron (ug/dl)		Serum TIBC (µg/dl)	
			Cord	Mother	Cord	Mother
		Mean ± S.E.; No. of subjects in parentheses				
$\angle$ 36	LIG	2.23 ± 0.14 (9)	161.2 ± 10.6 (9)	102.4 ± 7.84 (9)	317.5 ± 26.3 (9)	538.0 ± 26.1 (9)
	HIG	2.55 ± 0.15 (7)	169.0 ± 14.2 (7)	90.6 ± 6.91 (7)	304.0 ± 18.2 (7)	521.0 ± 52.5 (7)
>36	LIG	2.70 ± 0.05^{+++} (62)	181.7 ± 6.1 (62)	88.4 ± 4.35 (62)	338.4 ± 10.0 (62)	495.0 ± 11.5 (62)
	HIG	2.96 ± 0.07^{***++} (20)	191.4 ± 13.42 (20)	103.5 ± 11.72 (20)	323.0 ± 16.7 (20)	518.0 ± 19.1 (20)

Values significantly different from LIG *** P $\angle$ 0.01.

Values significantly different from premature values +++ P$\angle$0.01, ++ P $\angle$0.05.

in late pregnancy is suggested by a greater social class differences with regard to birth weights in premature infants as compare to full term infants.

Maternal blood hemoglobim, serum iron and serum iron binding capacity were analysed in relation to gestational age and cord blood levels (Tables 32,33,34). The values are similar to those found in previous studies in this laboratory (Rajalakshmi and Ramakrishnan, 1969). Cord blood hemoglobin and serum iron levels were found to be higher than those in maternal blood. The higher level of hemoglobin in the infant at birth is a well-known phenomenon. Similar observations have been made with regard to serum iron by Beal in 1980. This is attributed to the release of iron in the form of transferrin at the maternal end of placenta and its rapid transfer to the fetus in the form of fetal transferrin. Contrary to reports obtained elsewhere (Alvarez et al, 1974; Karotia et al, 1976; Haridas and Acharya, 1982), no increase was found in cord blood hemoglobin levels with the progress of gestation, but a slight decline, although not a significant decline was observed in maternal hemoglobin levels in the low income group, but not in the high income group. A greater decline with the progress of gestation has been found in previous studies in this department.

TABLE-32 : Hemoglobin levels in mother and cord blood in relation to gestational age in low (LIG) and high (HIG) income groups.

Weeks of gestation		Birth weight (kg)	Blood hemoglobin (g/dl)	
			Cord	Mother
Mean ± S.E.; No. of subjects in parentheses				
$\angle$ 36	LIG	2.17 ± 0.10 (10)	15.5 ± 0.77 (10)	11.4 ± 0.65 (10)
	HIG	2.45 ± 0.11 (13)	14.6 ± 0.51 (13)	12.4 ± 0.56 (13)
> 36	LIG	2.70 ± 0.05 (65)	14.7 ± 0.38 (65)	10.6 ± 0.39 (65)
	HIG	2.94 ± 0.04 (40)	15.3 ± 0.31 (40)	11.6 ± 0.32 (40)

TABLE-33 : Serum iron levels of mother and cord in relation to gestational age in low (LIG) and high (HIG) income groups.

Weeks of gestation		Birth weight (kg)	Serum iron (µg/dl)	
			Cord	Mother
Mean ± S.E.; No. of subjects in parentheses				
< 36	LIG	2.23 ± 0.12 (10)	170.8 ± 13.5 (10)	107.0 ± 8.3 (10)
	HIG	2.56 ± 0.13 (9)	166.2 ± 12.8 (9)	85.1 ± 6.5 (9)
> 36	LIG	2.72 ± 0.05 (67)	184.8 ± 6.10 (67)	89.8 ± 4.1 (67)
	HIG	2.93 ± 0.06 (23)	184.3 ± 14.3 (23)	97.7 ± 7.8 (23)

TABLE-34 : Serum total iron binding capacity (TIBC) of mother and cord in relation to gestational age in low (LIG) and high (HIG) income groups.

Weeks of gestation		Birth weight (kg)	Serum TIBC ($\mu g/dl$)	
			Cord	Mother
Mean ± S.E.; No. of subjects in parentheses				
< 36	LIG	2.29 ± 0.13 (10)	315.7 ± 23.6 (10)	537.1 ± 23.4 (10)
	HIG	2.55 ± 0.15 (7)	315.5 ± 10.6 (7)	521.2 ± 52.5 (7)
> 36	LIG	2.71 ± 0.05 (64)	339.7 ± 10.4 (64)	496.8 ± 11.5 (64)
	HIG	2.93 ± 0.06 (14)	319.4 ± 21.7 (14)	526.9 ± 19.0 (14)

This could be due to iron-folate supplementation during the later stages of pregnancy which is part of community health care at present and a majority of these women were beneficiaries under this programme. The present studies are cross sectional with appreciable intra-group variation and perhaps this has obscured the small differences which were found in the longitudinal studies carried out earlier supplemented by cross sectional studies with a larger sample size. Social class differences were not observed either in premature infants or full term infants with regard to these parameters but maternal blood hemoglobin levels in high income group were somewhat higher than in low income group in both categories although the differences fell short of significance. This small difference was, however, associated with a higher proportion of women with hemoglobin levels less than 11 g/dl in the low income group (47% in low income group as compared to 34% in the high income group).

Maternal serum iron levels were further analysed in relation to birth weights (Table 35). In the low income group, the correlation between maternal hemoglobin and birth weight as per cent of those expected for gestational age as reported by Widdowson (1968) was 0.272 ($P < 0.001$) and the corresponding value for maternal serum was 0.229 ($P < 0.02$). The corresponding values for the high income group

TABLE-35 : Significance of correlations (product-moment 'r') between different variables in low (LIG) and high (HIG) income groups.

Parameters	'r'		'p'	
	LIG	HIG	LIG	HIG
Gestational age Vs Birth weight	0.327	0.414	< 0.001	0.01
Mat. Hb. Vs. Birth weight as % exp.	0.272	0.125	< 0.001	NS
Mat. Fe Vs Birth weight as % exp.	0.229	0.236	< 0.02	NS
Mat. hemoglobin Vs Cord hemoglobin	0.409	0.707	< 0.001	< 0.001
Mat. iron Vs Cord iron	0.485	0.184	NS	NS
Mat. iron Vs Cord hemoglobin	0.258	0.218	NS	NS
Mat. hemoglobin Vs Mat. iron	0.532	0.236	< 0.001	NS
Cord hemoglobin Vs Cord iron	0.207	0.239	NS	NS

were 0.125 (NS) and 0.236 (NS). This is consistent with the greater variability of all the three in the low income group which includes a greater proportion of women with low levels of blood hemoglobin as mentioned earlier.

Thus maternal iron status seemed to have more perceptible effect on infant iron status in the low income group. Similar observations have been made with regard to other nutrients such as vitamin A (Shah and Rajalakshmi, 1984) and vitamin E (Shah and Rajalakshmi, unpublished). Also, Fleming (1974) and Singla et al. (1978) showed that moderate anemia in the mother (hemoglobin less than 10 g/dl) may be associated with fetal growth retardation. However, no correlation was found when absolute weights were used for the analysis, presumably because the differences in gestational age would obscure the picture. Product-moment correlations for gestational age and birth weights were found to be 0.275 in low income group ($P<0.01$) and 0.414 ($P<0.01$) in high income group. However, this could be due to individual differences in the stage when the 'catch up' growth which is found to occur towards term in the low income group, specially in view of the observation that in other studies, a wide variation is found in the pattern of maternal body weight changes during this period.

A high correlation between maternal and cord blood hemoglobin was observed in both premature and full term infants in both low and high income groups (Table 36). The values for premature and full term infants were therefore combined and the product-moment correlation was found to be 0.41 in the low income group and 0.71 in the high income group (Table 35). In the case of premature infants taken as a separate category, however, the correlation was higher in the high income group (0.90) than in the low income group (0.57). This is perhaps consistent with the greater maturity of the pre-term infants in the high income group as compared to the low income group as judged by several criteria such as, body weight, serum levels of vitamin A and E (Shah, 1986). Similarly, cord serum iron levels were found to vary with the concentration of maternal serum iron (Table 37). The correlation between the two were significant for both premature and full term infants in the low income group but only for the premature infants in the high income group. The values for the two categories combined were 0.485 for low income group and 0.184 for high income group. A less clear cut association was found with regard to serum total iron binding capacity (Table 38). These significant correlations between maternal and cord levels of blood

TABLE-36 : Maternal and cord blood hemoglobin in relation to maternal blood hemoglobin in low (LIG) and high (HIG) income groups.

Weeks of gestation	Maternal blood hemoglobin (g/dl)	Blood hemoglobin (g/dl)					
		Mother			Cord		
		LIG	HIG	LIG + HIG	LIG	HIG	LIG + HIG
		Mean ± S.E.; with no. of subjects in parentheses					
∠ 36	∠ 11.0	9.0 ± 1.26 (3)	9.8 ± 0.15 (3)	9.4 ± 0.59 (6)	13.1 ± 1.08 (3)	12.5 ± 0.35 (3)	12.8 ± 0.52 (6)
	>11.0	12.5 ± 0.26 (7)	13.2 ± 0.49 (10)	12.9 ± 0.31 (17)	16.5 ± 0.70** (7)	15.2 ± 0.52*** (10)	15.7 ± 0.44**** (17)
>36	∠ 11.0	8.4 ± 0.36 (36)	9.6 ± 0.25 (15)	8.8± 0.27 (51)	13.6 ± 0.49 (36)	13.8 ± 0.41 (15)	13.6 ± 0.36 (51)
	>11.0	13.4±0.29 (29)	12.8 ± 0.27 (25)	13.1 ± 0;20 (54)	16.1 ± 0.49**** (29)	16.2 ± 0.32**** (25)	16.2 ± 0.30**** (54)

Values in both gestational ages significantly different from ∠11.0.
** P ∠0.05; *** ∠ 0.002 ; **** P ∠ 0.001.

TABLE-37 : Maternal and cord serum iron levels in relation to gestational age and maternal serum iron in low (LIG) and high (HIG) income groups.

Weeks of gestation	Maternal serum iron range	Serum iron (μg/dl)					
		Mother			Cord		
		LIG	HIG	LIG + HIG	LIG	HIG	LIG + HIG
		Mean ± S.E.; with no. of subjects in parentheses					
< 36	< 90	78.8 ± 3.3 (4)	73.9 ± 3.8 (6)	75.9 ± 2.6 (10)	166.7±20.0 (4)	149.3±13.6 (6)	156.3±11.1 (10)
	> 90	125.7 ± 5.1 (6)	107.5 ± 7.3 (3)	119.7 ± 4.9 (9)	173.5±19.5 NS (6)	200.0±13.6 ** (3)	182.3±13.9 NS (9)
> 36	< 90	66.6 ± 2.6 (39)	72.4 ± 2.6 (18)	68.4 ± 2.0 (57)	169.0±7.3 (39)	167.0±13.5 NS (18)	167.9±6.6 (57)
	> 90	122.2 ± 4.4 (28)	139.8 ± 12.2 (13)	127.8 ± 5.0 (41)	206.8±9.0 **** (28)	196.6±18.7 NS (13)	203.5±8.5 *** (41)

Values significantly different from < 90 values, ** P < 0.02, **** P < 0.01.

TABLE-38 : Maternal and cord serum total iron binding capacity (TIBC) in relation to gestational age in maternal serum total iron binding capacity in low (LIG) and high (HIG) income groups.

Weeks of gestation	Maternal serum TIBC range	Maternal TIBC (µg/dl)			Cord TIBC (µg/dl)		
		LIG	HIG	LIG + HIG	LIG	HIG	LIG + HIG
		Mean ± S.E.; No. of subjects in parentheses					
< 36	< 450	-	239.4 (1)	239.4 (1)	-	291.7 (1)	291.7 (1)
	> 450	537.1 ± 23.4 (10)	568.2±28.0 (6)	548.8±17.9 (16)	315±23.6 (10)	319±11.6 (6)	317.1±15.0 (16)
> 36	< 450	396.9±9.9 (20)	398.4±18.8 (5)	397.2±8.6 (25)	315.1±18.8 (20)	274.8±16.0 (5)	307.0±15.6 (25)
	> 450	537.7±11.7 (44)	557.3±13.2 (15)	546.1±8.5 (59)	350.8±12.2 (44)	339.0±20.3** (15)	347.8±10.4* (59)

Values significantly different from less than 450 ug/dl. * $P<0.05$, [illegible] hemoglobin as well as serum iron suggest that maternal iron status has a greater deleterious effect on the circulating iron in the neonate possibly because of differences in maternal iron store and transport mechanism. Thus making the infant born of anemic mothers appear to be at greater risk of prematurity and earlier and more severe anemia of infancy. This is consistent with the observations made by number of investigators (Sanchez-Medal, 1969; Nhonoli et al, 1975; Singla et al, 1978). Similar observations have been made in case of vitamin A (Shah and Rajalakshmi, 1984).

A positive correlation was also found between blood hemoglobin and serum iron in both mothers and infants in the low income group but not in the high income group (Table 35). A similar pattern is also reported by Zittoun et al, (1983).

The observation that blood hemoglobin is affected by anemia in the mother is of great importance as hypoxia and respiratory distress are critical factors associated with neonatal morbidity and mortality and both may be aggravated by low levels of hemoglobin in the infants.

Thus the present studies suggest that maternal blood hemoglobin and serum iron levels have a significant influence on circulating iron levels of hemoglobin in the newborn suggesting that hemoglobin synthesis is affected whereas studies on fetal liver iron showed no correlation between either maternal blood hemoglobin and serum iron levels, on one hand, and fetal liver iron concentration, on the other. These differential effects on storage and circulating iron may represent a biological adaptation as a satisfactory concentration of iron in liver at birth sustains the infant in the postnatal period when the diet (milk) is poor in iron and the demand for iron is very high because of the rapid increase in blood volume from 285 ml at birth to 480 ml at 6 months and in total circulating iron from 210 mg to 240 mg. Moreover, although liver iron concentration is not affected in the fetus, total liver iron is affected as fetal weight and liver weight are less in the low income group than in the high income group.

The results of these studies considered together with fetal studies suggest even if liver iron concentration is not affected, maternal anemia may be a critical determinant for the size, iron status and vitality of the new born. Should the deficit in blood hemoglobin, serum iron levels be combined with a deficit in blood volume, the consequences would be even more serious. These observations emphasize the importance of treating and preventing anemia of pregnancy and emphasize the relevance of public health measures taken in this country in the form of iron-folate supplementation to the pregnant women, particularly in the low income group, the need for more extensive and effective implementation of this programme.

FETAL AND NEONATAL MAGNESIUM STATUS IN RELATION TO MATERNAL NUTRITIONAL STATUS

The adult human body contains 21-28g of magnesium or about 43 mg/100g of fat free tissue (Widdowson et al, 1951), half of it being in the bone and the other half being distributed between muscle and other soft tissues.

During development, the proportionate distribution of various organs to the body varies at different ages. For example, the brain accounts for a larger proportion of body weight in the fetus or the newborn than in the adult. Similarly, fetal heart muscle, liver and kidneys reach adult composition before the skin and the skeletal muscle suggesting differences in their pattern of development consistent with ontogenetic priority (Widdowson and Dickerson, 1960). Skeletal muscle accounts for about 25% of the weight of the newborn infant and 43% of the weight of adult man (Wilmer, 1940). In addition, the composition of muscle changes more with respect to magnesium, creatine, etc., than that of any other soft tissue, and muscle is the first to be affected by nutritional stress (Widdowson and Dickerson, 1964).

It is well known that during development, the water content of skeletal muscle decreases and that this is accompanied by a fall in the concentrations of the chief extra cellular ions, namely, sodium and chloride (Needham, 1931; Mannet and Darrow, 1938; Hines and Knowlton, 1939; McMeckan, 1940; McCance and Widdowson, 1956a; Widdowson and Dickerson, 1960). This is associated with an increase in protein (Needham, 1931; McCance and Widdowson, 1956b) and intracellular constituents such as nitrogen, potassium and magnesium. These changes are suggestive of a decrease in the percentage of extracellular fluid and an increase in cell mass.

In fact, it has been known for at least 100 years that fetuses and newborn animals contain a higher proportion of water than do adults of the same species (Von Bezold, 1857). This is to some extent accounted for by an increasing proportion of fat in the body, but even on a fat-free basis the percentage of water in the whole body falls from the embryonic period to some time in postnatal life. This percentage then becomes approximately constant at an age which varies from species to species. This 'steady state' was termed by Moulton (1923) as 'Chemical maturity'.

As mentioned earlier, the sequence of ontogenetic development of different tissues varies with the nervous system, the skeleton and the heart developing earlier and faster than other tissues such as the skin and skeletal muscle. This is also reflected in their biochemical maturation. This is particularly true of embryonic and fetal stages of development.

A progressive increase in total magnesium content with fetal development, a large proportion of which is present in muscle has been reported by McCance and Widdowson (1956a). On the other hand, Apte and Iyengar (1972) concluded from their studies on 30 fetuses (gestational age - 20-40 weeks) that total body magnesium increased with body weight upto 1.7 kg but not thereafter (Table-39).

Also, studies in this laboratory on fetal development and its relation to maternal nutritional status have shown that the concentration of some of the nutrients in the fetal tissues are affected by a low plane of maternal nutrition (e.g. vitamin A) (Gal et al, 1972; Underwood, 1974; Shah, 1986), whereas others are not (as discussed in an earlier section on iron) and bone composition (Satya Prasad, unpublished) but size is affected.

TABLE-39 : Fetal magnesium stores in relation to body weight[†]

Body weight(g) Range	n	Magnesium content g	Magnesium content g/kg fat free tissue
		Mean ± S.D.	
200 - 500	2	0.062 ± 0.034	0.158 ± 0.037
500 - 800	3	0.128 ± 0.006	0.199 ± 0.039
800 - 1000	6	0.198 ± 0.006	0.204 ± 0.004
1100 - 1400	7	0.216 ± 0.076	0.180 ± 0.056
1400 - 1700	5	0.241 ± 0.083	0.203 ± 0.048
1700 - 2000	3	0.377 ± 0.036	0.225 ± 0.006
2000 - 2300	5	0.436 ± 0.039	0.221 ± 0.074
2300 - 2600	4	0.530 ± 0.009	0.261 ± 0.0009
2600 - 2900	4	0.562 ± 0.079	0.234 ± 0.032
3200 - 3500	2	0.728 ± 0.63	0.256 ± 0.0736

† Apte and Iyengar (1972).

n = number of observations.

Since muscle is one of the tissues more vulnerable to nutritional status, the question arises as to whether the magnesium content of fetal soft tissues is affected by a low plane of maternal nutrition. This is particularly relevant for the muscular vigour of the newborn infant which is critical for its postnatal development. For instance, postnatal nutrition is derived entirely from breast milk among the poor, and it is well-known that lactation performance is influenced by the vigour with which the newborn can suckle at the breast, which, in turn depends on the strength of the facial muscle (Montagu, 1962, 1979). Similarly, vigorous crying by the infant helps in strengthening lung function.

The present studies were undertaken in this context to determine the relation between fetal and neonatal magnesium, levels and maternal nutritional status, using as criteria the magnesium concentration of selected tissues in the case of the fetus, and that of the cord and maternal serum levels of magnesium in the case of the neonate and the mother.

Fetal weights and tissue magnesium concentrations at various stages of fetal development are shown in Table-40. Fetal weights increased with gestational age as may be expected. The fetal weights were less than those reported in the west for corresponding gestational ages

TABLE-40 : Tissue magnesium concentrations at different gestational ages in low income group fetuses.

		Weeks of gestation					
		12-16	16-20	20-24	24	< 20	>20
		Mean ± S.E.; no. of subjects in parentheses					
Body weight (g)		106 ± 12.6 (18)	266 ± 16.5 (33)	424 ± 57.6 (6)	1240 ± 270.0 (10)	210 ± 16.0 (49)	912 ± 200.6 (16)
Liver	Conc. (mg/g)	0.106 ± 0.03 (15)	0.102 ± 0.03 (29)	0.062±0.02 (6)	0.093±0.01 (9)	0.098±0.02 (44)	0.080±0.01 (15)
	Content (mg.)	0.37±0.07	0.95±0.24	1.25±0.34	5.09±1.43	0.75±0.17	3.56±0.99
Spleen	Conc. (mg/g)	0.751±0.33 (8)	0.326±0.19 (22)	0.107±0.03 (5)	0.096±0.01 (10)	0.439±0.17 (30)	0.099±0.01** (15)
	Content (mg)	0.06 ± 0.02	0.06±0.02	0.08±0.03	0.29±0.10	0.06±0.02	0.22±0.07
Heart	Conc. (mg/g)	0.184±0.04 (16)	0.115±0.02 (28)	0.185±0.02 (5)	0.122±0.03 (9)	0.143±0.02 (44)	0.126±0.06 (14)
	Content (mg)	0.09±0.01	0.14±0.02	0.48±0.04	0.70±0.20	0.120±0.02	0.62±0.16

TABLE-40 (Contd.)

		Weeks of gestation					
		12-16	16-20	20-24	24	$<$ 20	$>$20
Kidney	Conc. (mg/g)	0.138±0.02 (15)	0.100±0.01 (28)	0.116±0.02 (6)	0.097±0.02 (10)	0.111±0.01 (44)	0.103±0.01 (16)
	Content (mg)	0.09±0.02	0.18±0.02	0.35±0.06	0.89±0.20	0.147±0.02	0.69±0.14
Muscle	Conc. (mg/g)	0.174±0.07 (14)	0.114±0.03 (31)	0.131±0.03 (5)	0.104±0.02 (9)	0.132±0.03 (45)	0.114±0.01 (14)

Mean values different from those for less than 20 weeks values, ** $P < 0.05$.

(Widdowson, 1968; Warwick and Williams, 1973; Montreewasuwant and Olson, 1979) but the values for 20 weeks are similar to those reported by Abravomich (1969) and Lakshminarayana et al. (1974). The deficits in fetal weight as compared to Widdowson's norms are consistent with differences in birth weights (Thomson and Hytten, 1966; Rajalakshmi, 1971). Although the total amount of magnesium increased in all the tissues, magnesium concentration (mg/g) did not show a consistent trend with development, although some suggestion of a decline was found especially in the case of the spleen. This trend was more evident when the values for below and above 20 weeks were pooled together. However, except in the case of the spleen, the differences were not statistically significant. This is consistent with the observations of Widdowson and Dickerson (1960) for muscle and liver. This is also true of other nutrients such as iron, as already reported, and vitamin A (Shah, 1986). Widdowson and Dickerson (1960) have also pointed out that the increments in mineral at each stage of development are partly the result of the increasing size of the body and partly the result of changes in chemical composition, as for example, the decrease in water content and an increase in the amount of solids, associated with the increase in cell mass (Table-41).

TABLE-41 : Changes in chemical composition during development.

Constituent		Fetus	Newborn	Adult
Body weight (kg)		0.175	3.5	70
			Per kg body weight	
Water	(kg)	0.88	0.69	0.60
Total N	(g)	14.0	18.9	28.6
Na	(meq)	104.0	69.4	73.6
K	(meq)	42.3	42.9	57.9
Cl	(meq)	78.3	45.7	42.0
Ca	(g)	3.4	8.1	18.9
P	(g)	2.4	4.6	10.6
Mg	(g)	0.14	0.22	0.39
Fe	(mg)	52.9	91.4	62.1
Cu	(mg)	3.3	3.9	1.4
Zn	(mg)	20.0	15.1	23.4

Source : Widdowson and Dickerson (1964)

Values for tissue magnesium concentration are in agreement with the only available values in the literature reported by Widdowson and Dickerson (1960), except for the liver where the present series showed lower values in comparison to the western values. In addition, the total amount of magnesium in all tissues are much lower. This is consistent with the only Indian study of Apte and Iyengar (1972) who found total body magnesium stores to be 6-9% less than reported in the West for fetuses of corresponding weights. This is because of lower fetal weight as compared to western norms at each gestational age and therefore tissue weights. The study also suggested that magnesium content increased with body weight upto 1700g of fetal weight and levelled off thereafter.

Tissue magnesium levels were analysed in relation to fetal growth status as judged by weight appropriate for gestational age. For this purpose, the weights were expressed as per cent of expected weight for gestational age (Table-42). Fetal growth status did not have any impact on magnesium concentration except in the case of the muscle before mid-pregnancy (Figure 10), but no difference was observed thereafter. Total magnesium stores in all the tissues were related to fetal size (Figure 9). This is accounted for by the fact that muscle mass and

TABLE-42 : Tissue magnesium concentrations in relation to grwoth status and gestational age in low income group fetuses.

		Weeks of gestation					
		< 20			>20		
		Weight at per cent of expected weight[@] for gestational age					
		< 60 I	>60 II	I as % II	< 60 I	>60 II	I as % II
		Mean ± S.E.; with no. of subjects in parentheses					
Fetal weight (g)		108 ± 26.4 (7)	226 ± 16.7 (42)	48	594 ± 230.3 (6)	1137 ± 268.4 (10)	52
Liver	Conc. (mg/g)	0.078±0.01 (7)	0.102±0.02 (37)	76	0.079±0.02 (5)	0.081±0.01 (10)	98
	Content (mg)	0.270±0.05*** (7)	0.840±0.19 (37)	32	1.58 ± 0.74* (5)	4.55 ± 1.36 (10)	35
Spleen	Conc. (mg/g)	0.204 ± 0.17 (4)	0.475 ± 0.19 (26)	43	0.093 ± 0.03 (5)	0.103 ± 0.01 (10)	90
	Content (mg)	0.035 ± 0.02 (4)	0.067 ± 0.02 (26)	52	0.095 ± 0.06 (5)	0.290 ± 0.10 (10)	33
Heart	Conc. (mg/g)	0.170 ± 0.06 (6)	0.135 ± 0.02 (38)	126	0.099 ± 0.02 (5)	0.140 ± 0.06 (9)	71
	Content (mg)	0.07 ± 0.03 (6)	0.127 ± 0.02 (38)	55	0.500 ± 0.33 (5)	0.720 ± 0.17 (9)	69

TABLE-42 (Contd.)

		Weeks of gestation					
		< 20			> 20		
		Weight at per cent of expected[@] weight for gestational age					
		< 60 I	> 60 II	I as % II	< 60 I	> 60 II	I as % II
Kidney	Conc. (mg/g)	0.111 ± 0.03 (7)	0.134 ± 0.02 (37)	83	0.105 ± 0.02 (6)	0.103 ± 0.01 (10)	102
	Content (mg)	0.060 ± 0.01**** (7)	0.170 ± 0.02 (37)	35	0.410 ± 0.16 (6)	0.850 ± 0.20 (10)	48
Muscle	Conc. (mg/g)	0.076 ± 0.009* (5)	0.134 ± 0.032 (42)	57	0.126 ± 0.03 (5)	0.107 ± 0.02 (9)	118

Mean values significantly different from more than 60 values;

* P < 0.10; *** P < 0.01; **** P < 0.001.

@ Fetal weights as given by Widdowson (1968).

FIGURE - 9 FETAL TISSUE MAGNESIUM STORES (mg) IN RELATION TO GROWTH STATUS

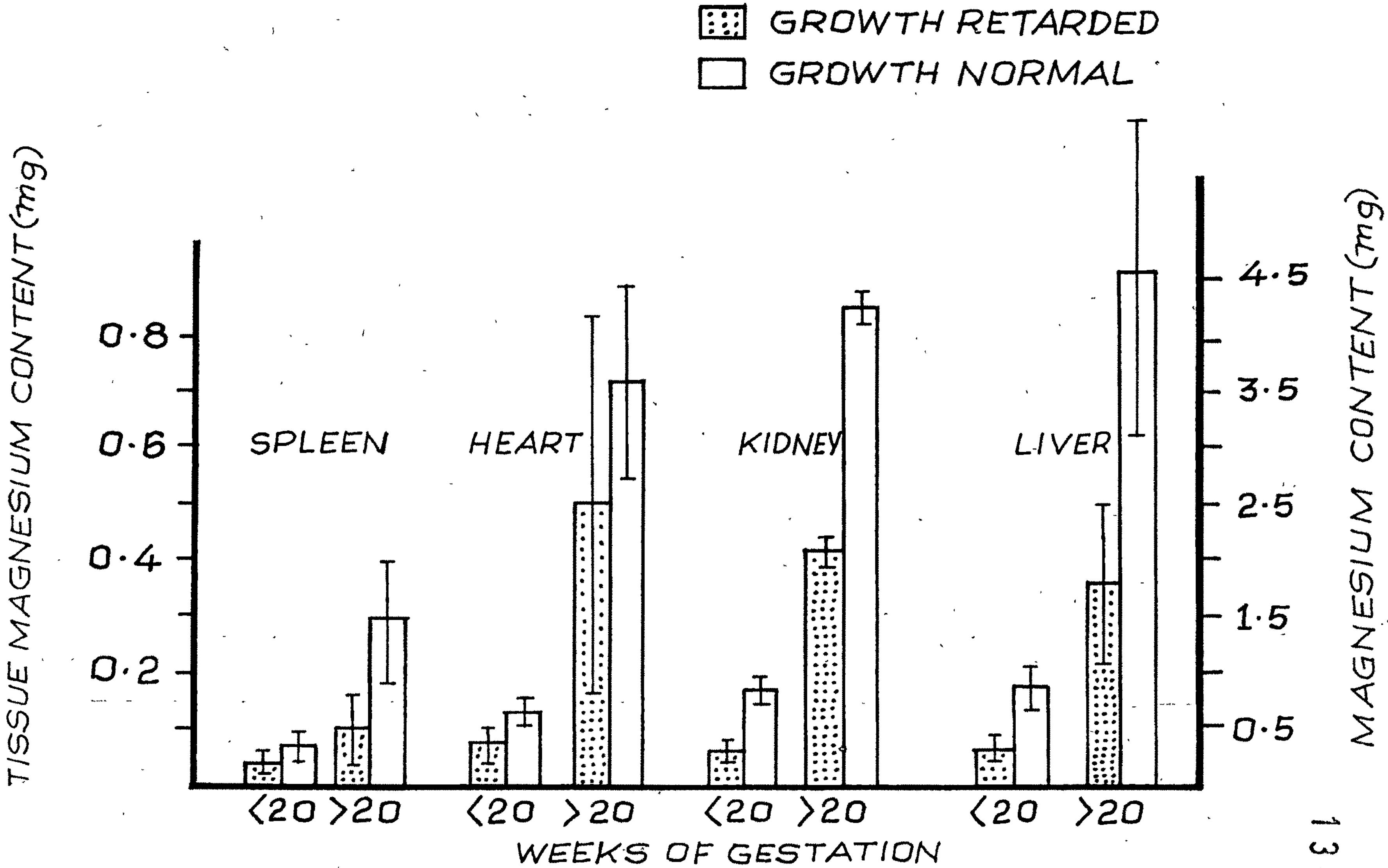
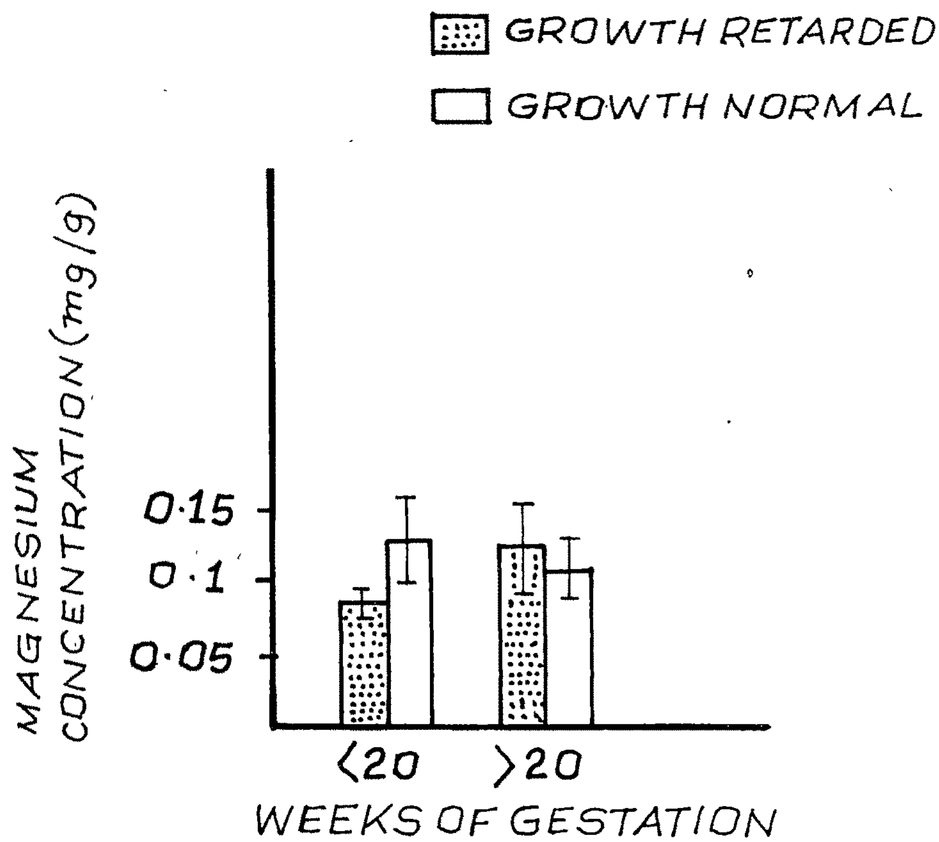


FIGURE-10: FETAL MUSCLE MAGNESIUM CONCENTRATION (mg/g) IN RELATION TO GROWTH STATUS



composition change to a greater extent with the progress of gestation than any other tissues (Widdowson and Dickerson, 1964). Muscle atrophies the most as ~~xxx~~ a result of prolonged undernutrition (Vincent and Radnermeeker, 1959; Widdowson and Dickerson, 1964; Waterlow and Mendes, 1975; Heymsfield et al, 1982; Lopes et al, 1982).

In this connection, it has been suggested that magnesium plays an important metabolic role during exercise other than as a cofactor for many enzymes and neuro-muscular function (Lusaski et al, 1983). Further, the reduction in Vo2 max (approximately 80% in the study of Barac-Nieto et al. (1978) observed in severely malnourished subjects is accountable in terms of difference in muscle-mass (Viteri, 1971; Barac-Nieto et al, 1978; Spurr et al, 1983; Desai et al, 1984). It is also known that in protein-calorie malnutrition, serum as well as muscle magnesium levels are decreased (Metcoff, 1960; Montgomery, 1960; Caddell, 1965; Mehta et al, 1972).

As mentioned earlier, wherever possible, attempts were also made to correlate tissue magnesium levels with maternal magnesium status. In spite of a lack of statistical difference between tissue magnesium concentration and maternal magnesium levels, a significant trend seems to be observed in early pregnancy (Table-43), tissue stores of magnesium in human fetuses seem to be affected by fetal size rather than maternal status suggesting that magnesium is preferably transferred to the fetus. However, low magnesium stores at birth may pose a risk for muscle-development and activities. As mentioned earlier, the vigour of facial muscles at birth are important, as it may be expected to determine the vigour with which the infant can cry and suckle at birth which are both important for stimulating and development of the vocal chords and respiratory functions. In this connection, the importance of breast feeding for the development of facial, dental and articulation in children have been pointed out by several investigators (Pottenger, 1946; Pottenger and Krohn, 1950; Bertrand, 1968; Broad, 1972a, 1972b, 1975).

As mentioned earlier, cord serum was analysed for magnesium in relation to gestational age, maternal magnesium levels and infant size (Table-44). The mean birth weight (kg) of full term infants were of the order of 2.76 and 2.87 in low and high income groups respectively (Table-44). The differences was greater in premature infants, the mean birth weight (kg) in this category being 2.18 ± 0.14 and 2.34 ± 0.14 respectively in the two groups. The mean birth

TABLE-43 : Tissue magnesium concentrations in relation to maternal serum magnesium levels during early pregnancy ($<$20 weeks).

		Maternal serum magnesium (mg/dl)			
		$<$ 2.0		$>$2.0	
		Mean ± S.E.; with no. of subjects in parentheses			
Maternal serum magnesium levels (mg/dl)		1.78 ± 0.03	(8)	2.42 ± 0.09	(10)
Liver	Conc. (mg/g)	0.050 ± 0.005	(8)	0.078 ± 0.010	(7)
	Content (mg)	0.52 ± 0.140	(8)	0.34 ± 0.110	(7)
Spleen	Conc. (mg/g)	0.138 ± 0.090	(4)	0.090 ± 0.030	(5)
	Content (mg)	0.02 ± 0.008	(4)	0.20 ± 0.005	(5)
Heart	Conc. (mg/g)	0.06 ± 0.010	(8)	0.15 ± 0.060	(9)
	Content (mg)	0.07 ± 0.020	(8)	0.05 ± 0.008	(9)
Kidney	Conc. (mg/g)	0.106 ± 0.007	(6)	0.134 ± 0.030	(9)
	Content (mg)	0.14 ± 0.030	(6)	0.08 ± 0.020	(9)
Muscle	Conc. (mg/g)	0.077 ± 0.010	(8)	0.100 ± 0.020	(8)

TABLE-44 : Cord and maternal serum magnesium (mg/dl) levels in relation to gestational age in low (LIG) and high (HIG) income groups.

Weeks of gestation		Birth weight (kg)		Serum magnesium (mg/dl)			
				Cord		Maternal	
		Mean ± S.E.; with no. of observations in parentheses					
∠ 36	LIG	2.18 ± 0.14	(10)	2.43 ± 0.19	(10)	2.13 ± 0.10	(10)
	HIG	2.34 ± 0.14	(8)	2.54 ± 0.18	(8)	2.04 ± 0.10	(8)
36-38	LIG	2.74 ± 0.06	(48)	2.55 ± 0.07	(48)	2.22 ± 0.06	(48)
	HIG	2.94 ± 0.06	(21)	2.59 ± 0.10	(21)	2.12 ± 0.08	(21)
> 38	LIG	2.77 ± 0.10	(13)	2.38 ± 0.11	(13)	2.21 ± 0.09	(13)
	HIG	2.80 ± 0.14	(7)	2.79 ± 0.10	(7)	2.49 ± 0.08	(7)
All groups combined	LIG	2.66 ± 0.05	(71)	2.50 ± 0.06	(71)	2.20 ± 0.04	(71)
	HIG	2.78 ± 0.07	(36)	2.64 ± 0.07	(36)	2.18 ± 0.06	(36)

weights of infants born in low and high income groups are similar to those reported by others (Kulkarni, 1959; Achar and Yankauer, 1962; Udani, 1963; Rajalakshmi et al, 1978). The social class differences are in accord with the higher proportion of small-for-gestational age infants among the poor (Achar and Yankauer, 1962; Udani, 1963; Rajalakshmi and Ramakrishnan, 1969; Rajalakshmi, 1971; Rajalakshmi et al, 1978). In the present studies, birth weights were consistently greater in high income group but the differences fell short of significance. In other studies, social class differences in this regard were found to be quite significant (mean birth weights of premature infants being 2.16 ± 0.11 and 2.56 ± 0.13 in low income group and high income group and of full term infants being 2.70 ± 0.06 and 2.94 ± 0.06 in low income group and high income group respectively). This may be because of small sample size and greater variability in birth weights among the infants studied. The greater differences in the premature infants suggests some catch up growth in late pregnancy. This has been observed in other studies and it has been suggested that the period of gestation may be extended to more than 40 weeks in poorly nourished women to allow for such growth (Reinhardt et al, 1978).

When serum magnesium levels were analysed in relation to gestational age, they did not vary either with gestational age or social class. The levels of cord serum magnesium were found to be higher than those in the maternal serum and a consistent trend was found in favour of high income group. When maternal serum magnesium levels were analysed in relation to cord serum levels, no correlation was found between the two (Table-45). Similarly, the data presented in Table-44 showed no correlation between maternal and infant serum magnesium levels either in premature or full term infants. The value for the cord and maternal serum magnesium levels are in agreement with reported values (Salmi, 1954, 1955; Hillman et al, 1977; Bogden et al, 1978; Vobecky et al, 1982).

However, in parallel studies done in this laboratory in Kerala, where diets were marginally deficient in magnesium, serum magnesium levels were found to be lower in premature and small for date infants. Such a differential pattern is perhaps accounted for by the fact that the diets in Baroda are not deficient in magnesium. The diets of the poor in Kerala are composed of rice and tapioca supplemented mainly with 25-50g of fish and only occasional consumption of vegetables whereas those of the poor in Baroda are composed of wheat, bajra and rice supplemented with legumes and vegetables. The typical diets of the poor in the two regions provide respectively 100-150 and 200-230 mg of

~~magnesium~~

TABLE-45 : Cord serum magnesium levels in relation to maternal serum magnesium levels in low (LIG) and high (HIG) income groups.

Maternal serum magnesium levels (mg/dl)		Serum magnesium (mg/dl)					
		Birth weight (kg)		Cord		Maternal	
		Mean ± S.E.; no.of observations in parentheses					
∠ 2.0	LIG	2.63 ± 0.11	(20)	2.48 ± 0.13	(20)	1.77 ± 0.04	(20)
	HIG	2.75 ± 0.12	(12)	2.57 ± 0.17	(12)	1.82 ± 0.05	(12)
2.0-2.3	LIG	2.57 ± 0.09	(20)	2.26 ± 0.10	(20)	2.13 ± 0.02	(20)
	HIG	2.63 ± 0.14	(9)	2.54 ± 0.10	(9)	2.13 ± 0.03	(9)
> 2.3	LIG	2.73 ± 0.08	(31)	2.63 ± 0.06	(31)	2.53 ± 0.03	(31)
	HIG	2.91 ± 0.10	(15)	2.77 ± 0.08	(15)	2.53 ± 0.07	(15)
All groups combined	LIG	2.66 ± 0.05	(71)	2.50 ± 0.06	(71)	2.20 ± 0.04	(71)
	HIG	2.78+_0.07	(36)	2.64 ± 0.07	(36)	2.18 ± 0.06	(36)

magnesium. The dietary patterns in the two regions are shown in Table-46.

In this connection, serum magnesium levels are found to be low in severely malnourished children in Kerala whereas they were found to be normal in children with an apparently similar degree of malnutrition in Madurai (Prathapkumar, 1983).

In this connection, it is also relevant to point out that although low serum magnesium levels are definitive of magnesium deficiency normal levels may not be indicative of the absence of magnesium deficiency on the basis of the finding that children suffering from protein-energy malnutrition and manifesting magnesium deficiency on the basis of low urinary response to magnesium supplementation may some time have normal levels in the serum (Montgomery, 1960; Montgomery, 1961; Linder et al, 1963). However, while normal serum levels may not be indicative of adequacy, low levels are positive indicators of deficiency so that some difference in the magnesium status of severely malnourished children in Kerala and Madurai must be presumed.

In conclusion, tissue magnesium concentration decreased with the progress of gestation in the case of spleen. However, total content increased because of a

TABLE-46 : Nutritive value of the diets consumed in Trivandrum and Baroda in high (HIG) and low (LIG) income groups.

Region		Total calories (Kcal)	Protein (g)	Magnesium (mg)	Type of water used
Trivandrum					
HIG	V	1850	43	100 - 150	Soft
	NV	1800	46	100 - 150	Soft
LIG		1580	30	100 - 150	Soft
Baroda					
LIG		1570	38	200 - 230	Hard

Source: Peramma, (1985)

V and NV, Vegetarian and Non-vegetarian.

greater increase in organ size. It also appears that fetal size rather than maternal magnesium status is a critical determinant of fetal magnesium status. Muscle magnesium levels were found to be affected by fetal size apart from the overall decrease in total magnesium stores. Cord serum magnesium levels were not found to be influenced by either maternal status, gestational age or fetal size.

EFFECTS OF SUPPLEMENTATION ON PREGNANCY WEIGHT GAIN AND OUTCOME.

As mentioned earlier, the diets of the poor in India and other similar regions are often inadequate in food energy, protein, calcium, vitamin A and riboflavin. This is especially true of women because they get less than their due share of the nutritious foods because of the prevailing food sharing practices. Clinical symptoms of deficiency of these nutrients are seen in an appreciable proportion of women and are aggravated during pregnancy because of increased requirements and no change in the quality or quantity of diets. Iron deficiency anemia is also common even when the diets are adequate in iron because of poor availability. Maternal anemia accounts for an appreciable proportion of maternal mortality (15%) and is higher in Gujarat (20%). The problem is aggravated by the high prevalence of nausea and anorexia during early pregnancy. It is possible that this is further aggravated by a possibly poor condition of liver (since malnutrition affect liver function to some extent). It also appears to be reinforced by psychological and cultural factors as the young women who has to assume a self-effacing role ordinarily does get some degree of attention during pregnancy and delivery.

The net result is decreased intakes. Whatever the factors involved, weight loss is found in many women during the first trimester of pregnancy (Rajalakshmi et al, 1978). More and more data are available on the consequences of poor pregnancy weight gains for placental and fetal growth (Love and Kinch, 1965; Eastman and Jackson, 1968; Singer et al, 1968; Bergner and Susser, 1970; Rajalakshmi et al, 1978). Although mean weight gains during pregnancy (7.0 kg) and placental weights (490 and 470g) are not markedly different between poor and middle class women in this country (Rajalakshmi and Ramakrishnan, 1969; Rajalakshmi, 1971), the prevalence of gains below 2 kg in the last trimester and placental weights below 400g is quite appreciable among the poor. Both these factors are associated with low weights. Evidence for a relationship between maternal weight gain and fetal growth as judged by birth weight has also been obtained from epidemiological studies which show that birth weights are lower in times of food restriction (Antonov, 1947; Smith, 1947; Stein et al, 1975, 1978; Prentice, 1980). Nutritional supplementation of women is found to be associated with increase in birth weights (Table 47) (Srikantia and Iyengar, 1972; Brozek et al, 1977; Lechtig et al, 1979; Mora et al, 1979; Prentice, 1983; Adair et al, 1984). Although reports contrary to this have also been made (Williams and Fralin, 1942; Mcganity et al, 1954, 1955), the conflicting results

TABLE-47 : Summary of studies on the impact of maternal nutrition interventions during pregnan

	Dutch famine study 1,2	Bogota Colambia 2, 4	Guatemala/ INCAP 2,3,5,6	India 7, 8
Description of subjects	Dutch Cities under Nazi occupation. Well fed upto 1940 Food rationing 1941-44 Near starvation 1944-45. Rehabilitation 1945-46.	Urban Slums Pregnant women having previous malnourished child. Average daily intake 1600 cal. Protein 35.5g.	Rural pregnant women from 4 villages. Average daily intake 1400 cal. Protein 45g.	Urban subjects. Average daily intake 1600-1800 cal. Protein 40g.
Dietary modification	Starvation due to stoppage of external food supplied (1944-1945); then restored to high dietary intake.	Selected foods provided for entire family. Net increment for pregnant women 133 cal and 20g protein.	Liquid supplements: 1) Fresco-low cal, no protein, vitamin mineral fortified. 2) Atole-Protein plus calories, vitamin-mineral fortified Ad-lib intake.	Regular hospital foods (2000 cal) plus up to 500 cal in additional foods or supplements.

TABLE-47 (CONTD.)

	Dutch famine study 1,2	Bogota Colambia 2, 4	Guatemala a/ INCAP 2, 3, 5, 6	India 7, 8	Mexico 3, 9
Influence of :					
(a) Gestational age on response to dietary change.	Rehabilitation during 3rd trimester suffi- cient to restore birth weight to prefamine level.	Last trimester or more showed effect.	No effect.	Treatment limited to last trimester.	None reported.
(b) Sex of infant on response to dietary change.	At height of famine decre- ment in birth weight greater for males than for females.	Increment in males (100g) greater than in females (12g).	None reported.	None reported.	None reported.
Infant morbidity and mortality.	Starvation associated with increased mortality.	Supplementat- ion reduced perinatal mortality.	No relation- ship.	None reported.	None reported.

seem to be due to differences in the nutritional status of the women receiving the supplements. Moreover, in most of these studies, supplementation is usually done after mid-pregnancy or in the third trimester of pregnancy but benefits from supplementation initiated earlier in pregnancy have also been reported (Habicht et al, 1973). But no data appear to be available on the comparative response to supplementation at different stages of pregnancy in terms of weight gain and the outcome of pregnancy.

The present studies were undertaken to study the course of pregnancy weight gains and their response to food supplementation at different stages of pregnancy on weight gain, the outcome of pregnancy and postnatal development of the infant. Because of difficulties in getting the cooperation of the subjects in blood collections, the studies had to be restricted to these aspects.
The general dietary pattern of these women is shown in Table 48. The average food intake at home of both the supplemented and control groups was found to be of the order of 1200-1400 Kcal. The supplement in the form of 'Dhokla' a fermented food, provided about 275 Kcal.
The home diet and supplement provide 35g and 8.8g protein, 28g and 4g fat, 130 mg and 434 mg of calcium, 20 mg and 7.5 mg of iron and 500 ug and 1188 ug of carotene.

TABLE-48 : Typical meal pattern of the pregnant women in Nava Yard.

Morning	Tea + Roti
Lunch	Roti + Vegetable or Dal
Evening	Tea
Dinner	Kichri + Vegetable

The intake at home remained practically unchanged during the progress of pregnancy except for a reduction in early pregnancy in only few cases and compared with intakes in the non-pregnancy state (Table 49).*** Similar findings have been made in previous studies in this laboratory and elsewhere (Darby et al, 1953; Bagchi and Bose, 1962; Rajalakshmi and Ramakrishnan, 1969; Rajalakshmi, 1971; Srikantia and Iyengar, 1972).

The data on initial nutritional status and clinical status in the control and supplemented groups at different stages of pregnancy are presented in Tables 50 & 51. As mentioned earlier, no difference was found with regard to initial weight, height and clinical symptoms in the two groups. Subjects were matched for their age and parity.

The data are anlysed according to the period of supplementation and the results are presented in Table 52. In the case of the group supplemented throughout pregnancy, the supplemented group showed a greater gain than the control group in the first trimester of pregnancy (2.52±0.49 and 2.08±0.67), but lost this advantage with the progress of pregnancy so that the mean values for net gains did not vary significantly between the two groups. The supplemented group had an initial advantage but a 'catch up' phenomenon was observed in the case of controls. This is supported by the

*** A few individuals were found to have dietary intakes less than 1000 k.cals/day. They were of smaller physical stature and a repeat investigations yielded the same results.

TABLE-49 : Calorie intake at home of control and supplemented group at different stages of pregnancy.

Stage of pregnancy (Trimester)		Supplemented	Control
I	M	1200	1180
	R	710 - 1940	850 - 1600
	n	32	37
II	M	1140	1200
	R	830 - 1625	880 - 1400
	n	31	60
III	M	1315	1300
	R	840 - 1680	960 - 1730
	n	13	47

M, R and n; Mean, Range and no. of observations.

TABLE-50 : Cross-sectional data on weight, height, age and parity of control and supplemented groups at different stages of pregnancy.

Stage of pregnancy (Trimester)	Weight (kg)	Height (cms)	Age (years)	Parity
	Mean ± S.E; with no. of subjects in parentheses			
Supplemented				
I	40.6 ± 1.07 (32)	150.0 ± 0.69 (32)	24.1 ± 0.55 (32)	3.4 ± 0.29 (32)
II	44.0 ± 0.76 (31)	152.0 ± 0.97 (31)	22.8 ± 0.17 (31)	2.9 ± 0.28 (31)
III	44.4 ± 1.16 (13)	153.0 ± 1.61 (13)	20.0 ± 1.86 (13)	2.8 ± 0.47 (13)
Control				
I	41.5 ± 0.87 (37)	151.0 ± 0.75 (37)	22.2 ± 1.07 (37)	2.2 ± 0.19 (37)
II	42.2 ± 0.70 (60)	151.0 ± 0.59 (60)	22.4 ± 0.70 (60)	2.6 ± 0.21 (60)
III	43.7 ± 0.75 (47)	152.0 ± 0.67 (47)	24.0 ± 0.69 (47)	2.9 ± 0.29 (47)

TABLE-51 : Incidence of clinical symptoms in control and supplemented groups at different stages of gestation (cross-sectional).

Clinical symptoms	Stage of pregnancy (Trimester)					
	I		II		III	
	S	C	S	C	S	C
	Per cent incidence					
Nausea	59	59	28	36	23	23
Vomiting	56	53	21	22	8	12
Anorexia	54	58	31	42	25	35
Edema	0	0	0	5	8	23
Body ache	46	53	48	47	31	42
Night blindness	0	3	7	10	8	12
Insomnia	12	11	31	19	8	9
Dizziness	29	42	34	36	23	23
Weakness	42	44	59	49	38	49
Vitamin B_2 deficiency	4	14	6	8	8	9

S and C, Supplemented and Control groups.

TABLE-52 : Weight gain during pregnancy in supplemented and control groups (longitudinal data)

Gestation period at start (weeks)		Weeks of gestation				
		Upto 16	16 - 24	24 - 32	Upto 32	32 - 40
		Mean ± S.E.; with no.of subjects in parentheses				
Around 6 weeks	S	2.52 ± 0.49 (14)	2.20 ± 0.37 (14)	1.49 ± 0.39 (14)	6.22 ± 0.97 (14)	1.54 ± 0.35 (8)
	C	2.08 ± 0.67 (12)	2.25 ± 0.34 (12)	2.03 ± 0.39 (12)	6.32 ± 0.91 (12)	1.38 ± 0.69 (4)
At 16 weeks	S	-	3.33 ± 0.46 (12)	1.98 ± 0.30 (12)	5.32 ± 0.44 (12)	1.19 ± 0.24 (14)
	C		2.07 ± 0.51* (11)	1.68 ± 0.38 (11)	3.75 ± 0.69 (11)	0.50 ± 0.16** (8)
At 24 weeks	S			1.87 ± 0.57 (7)		1.94 ± 0.16 (10)
	C	-	-	1.25 ± 0.25 (2)	-	1.69 ± 0.43 (8)

S and C, Supplemented and Control groups.

The supplement given was 'Dhokla' made of wheat and bengalgram and provided 275 Kcal and 8.8g of protein.

Values significantly different from supplemented group, * P ∠0.05; ** P ∠0.02.

the observation that birth weights also did not differ between the two groups. In this connection, in simultaneous studies in this laboratory on fetuses derived from medical termination of pregnancy a similar 'catch up' phenomenon is observed in the case of fetal weight and liver vitamin A stores which showed a greater association with maternal vitamin A status in the earlier stages of pregnancy than in the later stages (Shah, 1986). As there were some drop-outs because of various reasons, mainly, going to the parental home for delivery in late pregnancy, comparisons were made till 32 weeks of pregnancy. Those who were supplemented upto 32 weeks of gestation showed a gain of 6.22 $\pm$ 0.77 (n=14) whereas the control group gained 6.32 $\pm$ 0.91 (n = 11). The corresponding values when the supplement was given during 16-32 weeks (second trimester) were 5.32 $\pm$ 0.44 (n=12) and 3.75$\pm$0.69 (n=11) for the period of 16-32 weeks. Although the difference found in weight gain between supplemented and control groups in the second case striking, it fell short of significance presumably because of the small sample size and large intra-group variation. This was also true of birth weights which did not differ in two groups (Table 53). However, the birth weight in all the supplemented groups combined together was found to be significantly greater than those in the controls.

TABLE-53 : Birth weight in relation to pregnancy weight gain at different stages of gestation in supplemented and control groups.

Period of supplementation	Birth weight (kg)	Weight gain (kg)		
		I	II	III
	Mean ± S.E.; with no. of subjects in parentheses			
Through out pregnancy	2.69 ± 0.09 (19)	1.09 ± 0.29 (19)	3.79 ± 0.42 (18)	2.22 ± 0.33 (17)
Second and third trimester	2.72 ± 0.07 (22)	-	2.77 ± 0.42 (19)	2.65 ± 0.51 (18)
Third trimester	2.70 ± 0.10 (13)	-	-	3.18 ± 0.50 (13)
Combined value for all the supplemented groups	2.70 ± 0.05** (54)	-	-	-
Control	2.55 ± 0.06 (47)	0.35 ± 0.26 (23)	3.32 ± 0.29 (34)	2.34 ± 0.26 (37)

Values significantly different from control, ** $P < 0.05$.

The most interesting observation that emerged from the data is that supplementation throughout pregnancy does not represent an advantage over supplementation after 16 weeks although a few individuals in the early supplemented group seemed to have benefited. Thus the prevention of weight loss in the first trimester does not seem to have a measurable favourable effect in terms of net weight gain or birth weight if the weight gain during the rest of pregnancy is satisfactory. This is an important observation in the light of the enormous difficulties in monitoring women soon after conception whereas it is much easier to identify pregnant women after the first trimester. In fact, Metcoff et al. (1982) suggest screening of women at mid-pregnancy and supplementation thereafter.

In this context, it is a tendancy in India to consider some weight loss in first trimester to be normal and not take too much notice of it. It is also interesting to note that this weight loss had its origin in the first month of pregnancy even before many women in the group were either not aware or not sure of their being pregnant suggesting that it is to some extent physiological. In this connection, other similar changes such as fall in serum triglyceride levels during the second quarter of pregnancy with a subsequent rise have been noted and await a satisfactory explanation. The globulin levels tend to fall during early pregnancy and register a rise during late pregnancy. Weight loss in the first month was of the order of 0.3 kg in the control group with a range - 1.0 to 0.5 kg and the supplementation was started only around 6 weeks of pregnancy. The variability in weight gain in the control group seemed greater than in the supplemented group, the coefficients of variation being 46 and 29 for the groups from early pregnancy till 32 weeks and from 16-32 weeks. The corresponding values for controls were 50 and 60, suggesting that the smaller variability in the supplemented groups might be due to the benefits derived by a few women who might otherwise have shown very low weight gains in supplemented group. This is supported by the data in Table-54.

When the data on weight gain were analysed in relation to the prevalence and duration of nausea, the supplemented group was shown to have a lower prevalence of nausea with reduced severity and duration as compared to controls. Also, in the supplemented groups, the gains in the groups with and without nausea were comparable whereas this was not true of the controls. In the controls, weight gains in the first trimester differed between those with and without nausea (Table-55).

Further, women with nausea and anorexia were able to take as much 'Dhokla' as (265g being mean value) those without these symptoms.

TABLE-54 : Frequency distribution of weight gain during pregnancy (cross-sectional).

Period of supplementation		Weight gain (kg)					
		∠1.0	1.0-2.0	2.0-3.0	3.0-4.0	4.0-5.0	>5.0
		No. of subjects					
S Through out pregnancy	I	13	12	6	1	-	-
	II	2	3	5	10	5	6
	III	3	10	4	6	-	2
S Second and third trimester	II	3	8	9	4	4	3
	III	4	10	4	2	2	5
S Third trimester	III	1	4	4	2	-	2
C Control	I	25	11	1	-	-	-
	II	4	16	18	7	7	8
	III	6	21	9	5	5	3

S and C, Supplemented and Control groups.

TABLE-55 : Severity of nausea and vomitting Vs pregnancy weight gain and birth weight.

Clinical symptoms (Nausea and vomiting)	Weight gain (kg)						Birth weight (kg)	
	First trimester		Second trimester		Third trimester		S	C
	S	C	S	C	S	C		
	Mean ± S.E.; with no. of subjects in parentheses							
Absent	0.99±0.34 (14)	0.45±0.40 (10)	3.25±0.34 (26)	3.25±0.44 (20)	2.75±0.34 (31)	2.31±0.23 (22)	2.74±0.06 (31)	2.56±0.10 (21)
Present	0.97±0.24 (15)	0.16±0.19** (25)	3.26±0.30 (29)	2.90±0.25 (43)	2.60±0.33 (32)	2.59±0.28 (32)	2.76±0.07 (23)	2.61±0.05 (37)

S and C, Supplemented and Control groups.

Values significantly different from supplemented group, ** P $\angle$0.01.

The nausea and vomiting associated with pregnancy seem to be among the factors responsible for the low gains observed in the first trimester specially in the controls. Nausea was less prevalent and its severity and duration less in the supplemented group than in controls. Also, the observation that the gains in the groups with and without nausea were comparable in supplemented group but not in controls suggest that in spite of the nausea, the supplemented women were able to maintain their food intakes or avoid a reduction in the same below critical levels.

Seven of the supplemented women who joined the study group in the middle of the first trimester and who were in very poor shape (and whose weight gains were not available for first trimester) reported a distinct improvement in their home food intake after taking the supplement. The main factor involved in the prevention of weight loss in the first trimester seems to be their being able to avoid reduction in food intake below critical levels because of the supplement provided. The food supplement provided is a fermented food with a slightly sour taste which is found to be more acceptable specially during such conditions. In this connection, a distinct preference for slightly sour foods and drinks is shown by pregnant women. The nutritional quality of 'Dhokla' which is rich in 'B' vitamins, ascorbic acid, might also have contributed to better intakes.

The data on birth weights were further analysed in relation to pregnancy weight gain at different stages (Table-56). Those for each trimester were analysed according to whether they were less than or greater than a cut off point which was decided in accordance with the distribution of gains for that trimester.

The cut-off points so chosen for the first, second and third trimesters were 0.5, 2.0 and 1.5 kg. A consistent trend was found indicating a relation between weight gain and birth weight. This association was more clear-cut in the case of male infants (Table-57). The association between pregnancy weight gains and birth weights are consistent with other reports (Beilly and Kurland, 1945; Love and Kinch, 1965; O'Sullivan et al, 1965; Singer et al, 1968; Thomson et al, 1968; Bergner and Susser, 1970; Niswander, 1972; Rajalakshmi, 1978, 1980). In previous studies in this laboratory (Dave, 1980), pregnancy weight gain appeared to be somewhat greater with male infants although the difference fell short of significance. The data were therefore analysed from this point of view (Table-58). Neither net pregnancy weight gains nor birth weights differed with the sex of the progeny when the supplemented and control groups were combined together. However, in the supplemented group, male infants were

TABLE-56 : Weight gain Vs birth weight at different stages of gestation in control and supplemented groups combined.

Weight gain (kg)	Birth weight (kg)	
	Mean ± S.E.; with no. of observations in parentheses.	
I trimester		
< 0.5	2.65 ± 0.10	(13)
0.5 - 1.5	2.66 ± 0.08	(24)
> 1.5	2.64 ± 0.14	(5)
II trimester		
< 2.0	2.59 ± 0.11	(15)
2.0 - 3.5	2.67 ± 0.08	(30)
> 3.5	2.73 ± 0.05	(31)
III trimester		
< 1.5	2.55 ± 0.10	(18)
1.5 - 3.0	2.71 ± 0.05	(42)
> 3.0	2.72 ± 0.07	(27)
Total		
< 5.0	2.40 ± 0.19	(18)
5.0 - 7.0	2.73 ± 0.08	(13)
> 7.0	2.80 ± 0.08	(17)

TABLE-57 : Weight gain Vs birth weight at different stages of gestation in control and supplemented groups combined in relation to the sex of the infant.

Weight gain (kg)	Birth weight (kg) Male		Birth weight (kg) Female	
	Mean ± S.E.; with no. of observations in parentheses.			
I trimester				
∠ 0.5	2.57 ± 0.20	(6)	2.71 ± 0.10	(7)
0.5 - 1.5	2.65 ± 0.15	(11)	2.67 ± 0.09	(13)
>1.5	2.77 ± 0.27	(3)	2.56 ± 0.17	(5)
II trimester				
∠ 2.0	2.58 ± 0.18	(8)	2.60 ± 0.13	(7)
2.0 - 3.5	2.71 ± 0.13	(17)	2.61 ± 0.07	(13)
> 3.5	2.82 ± 0.08	(14)	2.66 ± 0.06	(17)
III trimester				
∠ 1.5	2.48 ± 0.17	(10)	2.64 ± 0.08	(8)
1.5 - 3.0	2.69 ± 0.10	(18)	2.72 ± 0.06	(24)
> 3.0	2.83 ± 0.10	(14)	2.60 ± 0.09	(13)
Total				
∠ 5.0	2.05 ± 0.26	(4)	2.74 ± 0.15	(4)
5.0 - 7.0	2.89 ± 0.05	(6)*	2.60 ± 0.12	(7)
>7.0	2.78 ± 0.14	(8)*	2.81 ± 0.09	(9)

Values significantly different from ∠5.0 values, * P ∠0.01.

TABLE-58 : The response to food supplementation during pregnancy in relation to the sex of the progeny.

Stage of pregnancy	Female		Male		S/C x 100	
	Supplemented (S)	Control (C)	Supplemented (S)	Control (C)	Female	Male
	Mean ± S.E.; with no. of observations in parentheses					
	Maternal weight gain					
Trimester I	1.18 ± 0.32 (13)	0.0 ± 0.41 (11)	1.16 ± 0.52 (7)	0.54 ± 0.29 (13)	118	215
II	3.64 ± 0.43 (20)	3.41 ± 0.40 (18)	2.84 ± 0.39 (18)	3.18 ± 0.36 (21)	107	89
III	2.68 ± 0.25 (28)	2.07 ± 0.30 (17)	2.22 ± 0.46 (20)	2.60 ± 0.33 (22)	130	85
	Infant birth weight					
	2.66 ± 0.05 (32)	2.58 ± 0.08 (22)	2.89 ± 0.08 (21)	2.53 ± 0.10** (25)	103	114

Values significantly different from supplemented group, ** $P < 0.01$.

found to be heavier than those in the control group. The greater beneficial effects of supplementation on both birth weights of male infants are consistent with the findings of Mora et al.(1979), who reported an increase of 100g of birth weight with food supplementation in case of male infants as against the 12g in female infants. Similarly, the Dutch famine study (Stein et al, 1975, 1978) showed that with relief conditions, birth weights were greater for males than for females. This is consistent with several findings suggesting that males are more vulnerable to certain types of nutritional stress than females. Similar sex differences have been observed with regard to skeletal retardation which is more evident in boys than in girls (Shah, 1983). Similarly, in rats, females subjected to food restriction were not found to show much growth retardation till the amount fed was restricted to 2/3 of ad lib intakes in the control group whereas the corresponding figure for males from the same litter was 80% of ad lib intakes. The degree of growth retardation was also greater in males (Mittal, 1982).

Similarly, the premature infants in the supplemented group showed less evidence of fetal growth retardation than those in the controls (Tables-59 & 60). This observation that premature infants in the supplemented group

TABLE-59 : Infant birth weight in relation to gestational age.

		Gestational age (weeks)			
		∠ 36	36 - 38	38 - 40	40 & more
		Mean ± S.E.; with no.of subjects in parentheses			
Supplemented					
	Male	2.80 ± 0.12 (3)	2.82 ± 0.07 (9)	3.00 ± 0.18 (4)	2.76 ± 0.27 (4)
	Female	2.59 ± 0.15 (7)	2.66 ± 0.07 (11)	2.62 ± 0.13 (6)	2.80 ± 0.13 (5)
	Combined	2.65 ± 0.11 (10)	2.74 ± 0.05 (20)	2.77 ± 0.12 (10)	2.78 ± 0.13 (9)
Control					
	Male	2.30 ± 0.30 (5)	2.69 ± 0.08 (9)	2.56 ± 0.20 (8)	
	Female	2.55 ± 0.12 (5)	2.72 ± 0.10 (6)	2.60 ± 0.21 (3)	2.8 ± 0.30 (2)
	Combined	2.43 ± 0.16 (10)	2.70 ± 0.06 (15)	2.57 ± 0.15 (11)	2.8 ± 0.30 (2)
		Percentage comparisons			
Male Vs Female					
	S	108	106	115	99
	C	90	99	98	-
Control Vs supplemented					
	Male	82	95	85	-
	Female	98	102	99	100

S and C, Supplemented and Control groups.

TABLE-60 : Distribution of birth weights in relation to gestational age and sex.

Weeks of gestation		Male		Female	
		$\angle$2.5	> 2.5	$\angle$2.5	> 2.5
		No. of subjects			
$\angle$ 36	S	-	3	3	4
	C	2	4	1	5
> 36	S	1	17	1	24
	C	3	16	3	12

S and C, Supplemented and Control groups.

fared better than those in the controls is consistent with the finding that social class differences with regard to birth weights are greater than in premature infants than full term infants suggesting some 'catch up' growth towards term in the infants of poorly nourished women. Mean weights of full term infants in the low and high income groups are found to be 2.70 ± 0.06 and 2.94 ± 0.06 as compared to 2.00 ± 0.09 and 2.45 ± 0.07 in premature infants matched for gestational age. This phenomenon may not be evident in the supplemented group because of the differential pattern of weight gains possibly associated with differences in the patterns of fetal growth.
Thus premature infants of poorly nourished mothers may be more liable to consequences including poor subsequent growth, poor neuromotor development and mental retardation (Davies and Stewart, 1975; Winick, 1976). In studies done on fetuses in this laboratory, premature infants are found to have lower levels of serum vitamin A (Shah, 1986), vitamin E (Shah, 1986) than full term infants and these difference are more marked in the low income group in which the prevalence of prematurity is also higher (Iyengar, 1984). Premature infants, who are also small-for-gestational age are in double jeopardy. It would appear that food supplementation can minimize these risks, specially among the poor. Moreover, the frequency distribution of the

gestational age of infants at birth in both groups (Table-61) suggests the incidence of prematurity to be higher in among the control group and female infants in the supplemented group as compared to male infants. This is consistent with the observation that male infants showed greater increment and benefited more as a result of supplementation than female infants. Mean gestational age was higher in the supplemented group as compared to controls. The average figures being 38.0 and 37.3 weeks in supplemented and control groups respectively.

Information on prenatal and neonatal mortality was also obtained. Food supplementation was associated with a significant decrease in the mortality to about one half. The figures were 3.9% in supplemented group (4 out of 102) and 6.7% in control group (10 out of 148). Similar finding have also been reported elsewhere (Brozek et al, 1977; Stein et al, 1978; Mora et al, 1979; Lechtig et al, 1979). The observation that mean birth weights did not differ between the two groups is perhaps not surprising as only about 10% of infants born of poorly nourished women have birth weights below 2.0 kg, and 29% below 2.5 kg. The remaining 60% or so are unlikely to be affected by supplementation. The mean values would not be sensitive indicators because of this. An important consideration that tends to be obscured in such studies is the feeling

TABLE-61 : Variations in gestational age at birth.

Weeks of gestation	Supplemented			Control		
	Male (M)	Female (F)	M + F	Male (M)	Female (F)	M + F
	No. of observations with percentages in parentheses					
∠ 36	4 (17.0)	7 (22.0)	11 (19.6)	6 (23.0)	7 (32.0)	13 (27.1)
36 - 38	10 (42.0)	13 (41.0)	23 (41.0)	9 (35.0)	9 (41.0)	18 (37.5)
38 - 40	5 (21.0)	6 (19.0)	11 (19.6)	11 (42.0)	4 (18.0)	15 (31.3)
> 40	5 (21.0)	6 (19.0)	11 (19.6)	0 (0.0)	2 (9.0)	2 (4.2)
Total	24	32	56	26	22	48
Mean gestational age	38.0 ± 0.49	37.4±0.44	(38.0)	36.6 ± 0.56	37.3 ± 0.48	(37.3)

of well-being in the mothers, more often reported in the supplemented group, is likely to have on her attitude towards pregnancy and towards other members of the family, specially towards young children who continue to need medical care.

***

Weights of the infants in supplemented group were not different from control group at the age of 3 months or 6 months (Table-62). Also, the mean age at which the birth weight doubled or tripled also did not vary in the two groups. This is also observed in the only study published in literature on postnatal growth of supplemented infants in New York City (Rush et al, 1980). This observation is consistent with the usually excellent lactation performance of poorly nourished women (Rao et al, 1958; Venkatachalam et al, 1967; Rajalakshmi and Ramakrishnan, 1969). In previous studies in this laboratory, weights of infants born of poor and upper class women were not significantly different at six months of age. Even infants born with birth weights of about 2 kg achieved a weight of 6 kg by this age (Table-63).

It is relevant to point out that an improvement in birth weight produced by prenatal supplementation will not necessarily be followed by a corresponding improvement in mortality or postnatal growth, if infection is highly prevalent in the population studied.

*** The birthweights of the infants monitored for growth did not vary between the ~~two~~ groups presumably because of dropouts so that there was reduction in numbers.

TABLE-62 : Postnatal growth pattern in supplemented and control infants.

Age of infants (months)	Weight (kg)	
	Supplemented	Control
	Mean ± S.E.; with no.of observations in parentheses	
At birth	2.62 ± 0.07 (23)	2.61 ± 0.15 (15)
1	3.23 ± 0.11 (22)	3.43 ± 0.18 (15)
2	3.90 ± 0.14 (24)	4.25 ± 0.20 (15)
3	4.62 ± 0.20 (21)	5.24 ± 0.17 (16)
4	5.00 ± 0.17 (21)	5.72 ± 0.20 (16)
5	5.63 ± 0.19 (18)	6.00 ± 0.23 (12)
7	6.76 ± 0.40 (8)	6.59 ± 0.19 (13)

TABLE-63 : Weight gain of infants in relation to birth weight (kg).

Range of birth weight (kg)	Mean birth weight (kg)	Weight at 6 months (kg)	Weight gain (kg)	% increase in 6 months
1.5 - 2.0	1.7	6.1	4.4	260
2.1 - 2.5	2.4	6.1	3.7	150
2.6 - 3.0	2.8	6.8	4.0	140
Above 3.0	3.3	6.9	3.6	110
IOWa norms	3.4	8.0	4.6	140

Source : Subbulakshmi (1970).

In conclusion, these studies suggest that supplementation helped to alleviate the nausea and loss of appetite in early pregnancy and to improve gains in the first trimester. It had a favourable outcome on the neonatal status of premature infants as reflected in the weight for gestational age as well as full term male infants. The frequency distribution of weight gains and birth weights suggest low weight gains and birth weights might have been prevented in atleast a few women.

The promotion of a feeling of well-being in atleast a few of the supplemented mothers also suggests the possibility that the supplementation might have had other benefits. This aspect remains to be investigated. It has also been suggested that for such supplementation individuals prone to the risk of a fetal growth retardation should be identified by multiple biochemical and other indices. Also, extensive analysis are needed on the meaning of birth weight in terms of maternal status and the subsequent health and development of the child. Such monitoring is not practicable in the field situation. Also, in the case of poor women the supplements ensure that atleast minimal levels of food intake are maintained and no adverse effects whatever are likely to follow supplementation of this type. Apart from this, the supplement might have other beneficial effects on the mother, and consequently her family members.

The main aim from a public health point of view should be to reduce the risk of fetal growth retardation not only for the sake of improving growth but, also because such retardation is associated with an increased postnatal morbidity and mortality due to risk factors such as hypothermia, hypoglycemia, respiratory distress syndrome, etc. Moreover, low birth weight infants are also born with poor stores of nutrients and poor organ growth and are at greater risk of subsequent growth retardation and malnutrition if postnatal nutrition is not satisfactory. In this connection, in studies carried out in this laboratory in Trivandrum and Madurai in severely malnourished children, marasmus was found more often in the case of infants who were reported to have been small at birth and not adequately breast fed. In girls pre and postnatal growth retardation is likely to be associated with a higher risk of suboptimal growth and development during adolescence with continued chronic undernutrition and this result in a poor pre-pregnant nutritional state of the next generation of prospective mothers.

It is increasingly recognised that antenatal care as well as the care of the infant in the immediate post-partum

period play a crucial role in the promotion of lactation
performance as well as mother-infant relations. Of particular importance are factors such as the feeding of colostrum, physical contact between the mother and infant and the type and amount of the first few feeds given to the infant, the mode of feeding, dietary practices pertaining to the mother, place of delivery, etc. During the course of present studies systematic information on all these aspects was obtained. The major practices observed are described below :

Perinatal practices

Mothers:

(1) Generally, the foods avoided during late pregnancy are also avoided during the first three months of lactation. These includes brinjals, cluster beans, ladies fingers (Okra), dahi (yoghrut) and sour foods and wheat.

(2) After delivery, women consume gum, sheera (made out of wheat flour, ghee and jaggery), piperimol, dry ginger and methi seed powder. Usually bland foods composed of kodri and mung dal (green gram) are consumed for first fifteen days.

(3) Women are not allowed by their mother-in-low or elders to come out of the house for 40 days after delivery. No one is allowed to touch them.

(4) In home delivery cases, usually the 'Dayan' or the older relative attends on the delivery.

The cord is cut by a sharp object, viz. knife, scissor or blade which are not sterilized. It is clamped using ordinary thread, in cases, where septisis occurs, 'sindoor' is applied. The placenta is buried with a coin and pinch of salt and turmeric.

(5) The mother bathes with 'hot' water to which some 'Sheppu' seeds are added. The child is bathed immediately after birth, after applying oil on the whole body.

Infants :

(1) Among 118 women studied, only 4 put the child to the breast within 8 hours of delivery, and 31 within 24 hours of delivery, whereas 50 were breast fed on day 2 and 37 on day 3.(Table-64a).

The pre-lacteal feeds used are either glucose water or jaggery water or occasionally, honey. This is administered using a spoon or cotton wool. This practice is carried out even in the hospitals. Usually, other fluids including water are not given.

TABLE-64 : Perinatal practices.

(a) Interval between birth and first breast feed.

Interval in hours	Number with percentage in parentheses	
∠ 8	4	(33.8)
8 - 24	27	(22.9)
24 - 48	50	(42.4)
48 - 72	35	(29.7)
>72	2	(1.7)

(b) Place of delivery

Place	Number with percentage in parentheses	
Home	31	(24.8)
S.S.G. Hospital	23	(18.4)
Missionary Hospital	50	(40.0)
Railway Staff Hospital	21	(16.8)

(c) Duration of labour pain

Period in hours	No.with percentage in parentheses	
∠ 2	16	(14.5)
2 - 4	23	(20.9)
4 - 8	25	(22.7)
8 - 16	19	(17.3)
16 - 24	8	(7.3)
>24	16	(14.5)
Ceaserean	3	(2.7)

TABLE-64 (Contd.)

(d) Duration of post-partum amenorrhea.

Duration in months	Number with percentage in parentheses
< 3	41 (19.1)
3 - 6	25 (11.6)
6 - 9	19 (8.8)
9 - 12	72 (33.5)
> 12	58 (27.0)

(2) Two out of 118 women failed to establish lactation. About 75% of the women delivered in the hospital and 25% of deliveries were home deliveries either by untrained 'Dais' or traditional midwifes with some modern training (Table-64b).

(3) In about 80% of the cases, child was given to the mother within 2 hours of delivery whereas in 20% of cases, child was given within period ranging from 2 hours - 8 days. Reasons being either the child was born prematurely or mother underwent cessarean section. In these cases, child was given glucose water till mother was allowed to breast fed the child by hospital authorities.

(4) Duration of labour pain varied from 2 hours to 48 hours. Multiparous women showed a tendancy to have shorter duration of labour pain (Table-64c).

(5) About 68% of women had post partum bleeding for more than a week. A delayed resumption of menstruation was observed in the present studies (Table-64d). About 61% had not resumed menstruation when the child was 9 months of age. Similar studies have been made in previous studies in this laboratory (Subbulakshmi, 1970). In contrast, in the upper class the mean age of the child at the resumption of

menstruation was six months. These differences have been attributed to prolonged lactation in the lower class although an adaptation to a low plane of nutrition could also be a factor as even in the case of women of whose infants are entirely breast fed a wide variation is found among the poor women and between poor and upper class women. Moreover, other factors such as the introduction of weaning foods, the rate of fall in prolactin levels, etc. have been implicated. Similar findings have also been made in the Gambia by the cambridge group (Prentice, 1980) and similar differences found between women studied in the Gambia and Cambridge.

(6) In instances, when the child is found to be passing green stools, mother omits fat from her diet.

(7) In the present studies, the average inter pregnancy interval was found to be two years, although a longer interval of three was found in previous studies (Rajalakshmi and Ramakrishnan, 1969; Bhalla et al, 1974). The present studies indicated the persistance of traditional antenatal and post partum practices such as lack of antenatal medical care, avoidance of some nutritious foods during pregnancy, with-holding of breast milk for 2-3 days after delivery, dietary restrictions for the mother and unhygenic conditions with regard

to severing and bandaging of umbilical cord in home delivered cases. Information on resumption of menstruation after delivery indicates a considerable delay in such resumption in poor women.

THE PATTERN OF FETAL GROWTH AND NEONATAL GROWTH STATUS IN RELATION TO GESTATIONAL AGE AND THE PLANE OF MATERNAL NUTRITION.

SECTION-A.:

The pattern of organ growth during human fetal development in relation to maternal gestational age, nutritional ~~STX~~ status and fetal growth status

The body is made up of many different organs and tissues, each with a different chemical composition, and each contributing a different proportion to the body as a whole. Moreover, the proportionate contribution of these various organs to the body varies with development. Also, nutrients that reach the fetus are distributed among various fetal organs according to genetically and environmentally conditioned schedules (Zamenhof and Marthens, 1982). The differential nutrient supply to various developing organs depend on the metabolic rates of these organs at that particular time, the tissue with highest metabolic rate receiving highest priority. For example, the brain accounts for a large proportion of the body in early life than in the adult whereas the reverse is true of skeletal muscle.

As part of the studies described earlier, attempts were made to study the rate at which the different organs of the body increase in weight and effect of the plane of maternal nutrition as well as fetal growth status on this pattern.

Fetal weights and tissue weights in both low and high income groups are presented in Table-65. As may be expected, all parameters increase with the progress of gestation both in low and high income groups. As pointed out earlier, fetal weights in the low income group were less than those of the high income group and those reported in the west (Widdowson, 1968; Warwick and Williams, 1973; Montreewasuwat and Olson, 1979), but the value for early pregnancy were similar to those reported by Apte and Iyengar (1972).

When fetal weight was expressed as percentage of expected weight for gestational age using Widdowson's norms (1968), the values in the two groups were comparable in early pregnancy but some deceleration of growth was observed in low income group around mid-pregnancy (Table-65). This deceleration of growth continued till the age of 32 weeks after which a 'catch up' is observed. The deceleration of growth in the low income group around mid-pregnancy is consistent with other observation (Bhatt, 1982; Iyengar, 1984). The growth of the organs studied, namely the liver, TABLE-65 : Fetal and tissue weights in relation to gestational age in low (LI

Weeks of gestation	12-16		16-20		20-
	LIG (A)	HIB (B)	LIG (A)	HIG (B)	LIG (A)
			Mean ± S.E.; with no. of observatic		
Gestational age (weeks)	13.4±0.35 (20)	14.0±0.41 (11)	17.6±0.23 (34)	18.1±0.16 (35)	21.7±0.24 (25)
Weight of whole fetus (g)	73.8±9.70 (20)	107.8±16.7 (11)	228±15.7 (34)	230±10.9 (35)	381±31.7 (25)
Whole fetus wt. as % expected wt.	71 ± 2.69 (20)	84 ± 6.84 (11)	72 ± 2.71 (34)	73 ± 2.41 (35)	65 ± 3.74 (25)
A as % B	68		99		80
Spleen weight (g)	0.076±0.02 (14)	0.089±0.02 (10)	0.251±0.03 (31)	0.228±0.02 (35)	0.620±0.18 (23)
as % body weight	0.106	0.08	0.11	0.10	0.16
A as % B	85		110		141
Liver weight (g)	3.02±0.48 (20)	4.91±0.87 (9)	8.28±0.67 (32)	8.54±0.44 (34)	16.26±1.53 (24)
as % body weight	4.1	4.6	3.6	3.0	4.3

spleen, kidney, heart, lung and brain as well as the whole body showed a linear growth, but the actual pattern varies with the tissue studies (Figure 11). This is observed by Potter (1961). Organ weights and fetus weights were comparable in early pregnancy in both income groups but differences set in after mid-pregnancy (Table-66). This is probably due to the much more rapid growth rate during this period so that differences in growth pattern associated with those in nutrient supplies are accentuated.

When organ weights are presented as percentages of body weight, the proportion of body weight contributed by liver, kidney and heart was substantially the same from 12 weeks onwards. This was also observed by Southgate and Hey (1976). The contribution of liver, heart and kidney were 4%, 0.6% and 0.8% to the total body weight. The figures are in agreement with the values reported by Widdowson and Dickerson (1960) of 4%, 0.6% and 0.7% respectively during fetal life. On the other hand, the contribution of spleen, lung and brain to total body weight declined with the progress of gestation indicating that these are the tissues which enjoy ontogenetic priority as might be expected because of their vital metabolic role. In the case of adrenals, no change was observed whereas placental weight in relation to body weight showed a decline where the reverse is true of spleen. Gruenwald (1974)

FIGURE-11 A-ORGAN GROWTH PATTERN IN LOW (LIG) AND HIGH (HIG) INCOME GROUP.

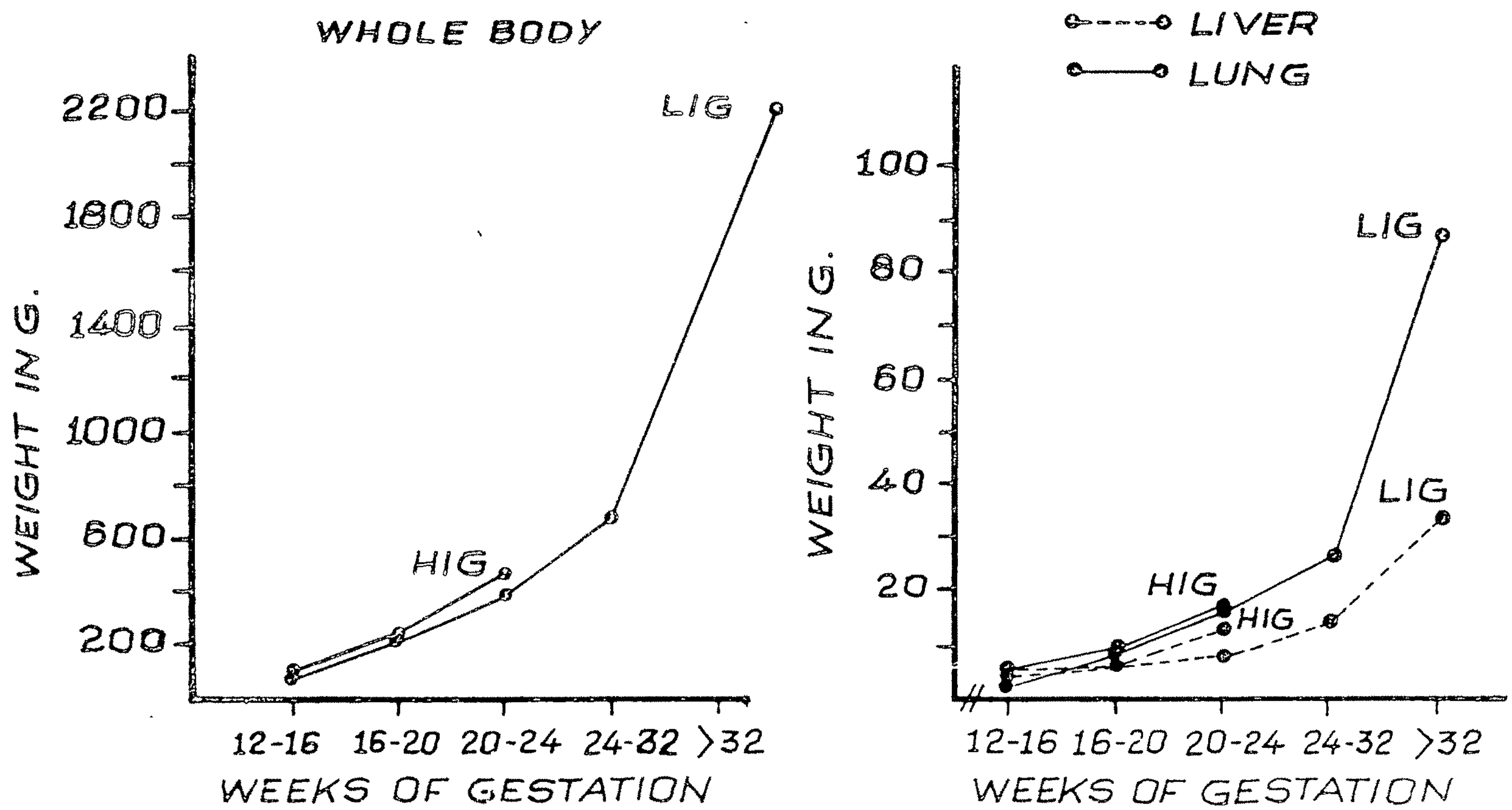


FIGURE-11 B-ORGAN GROWTH PATTERN IN LOW (LIG) AND HIGH (HIG) INCOME GROUP.

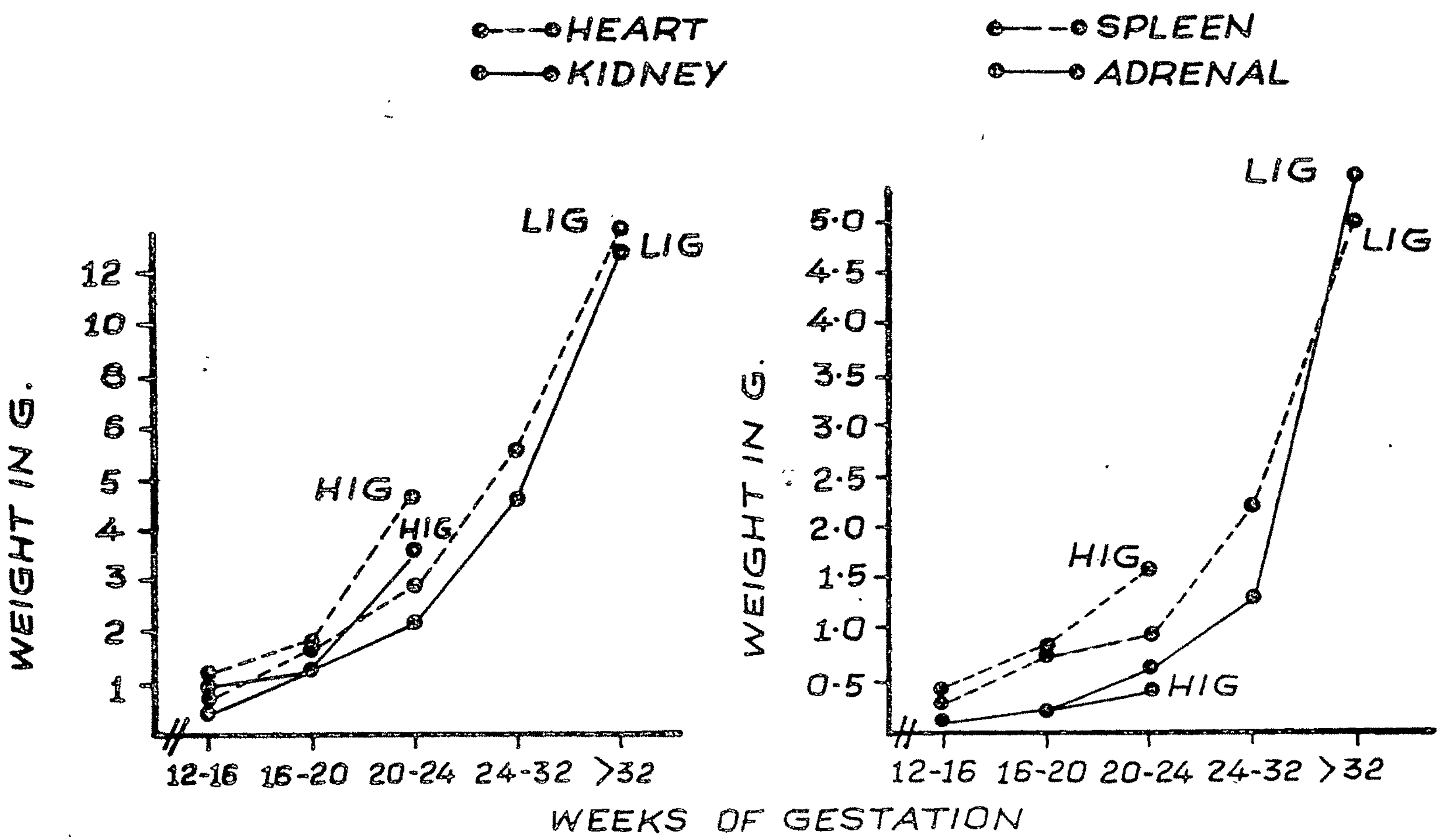


FIGURE-11 C- ORGAN GROWTH PATTERN IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

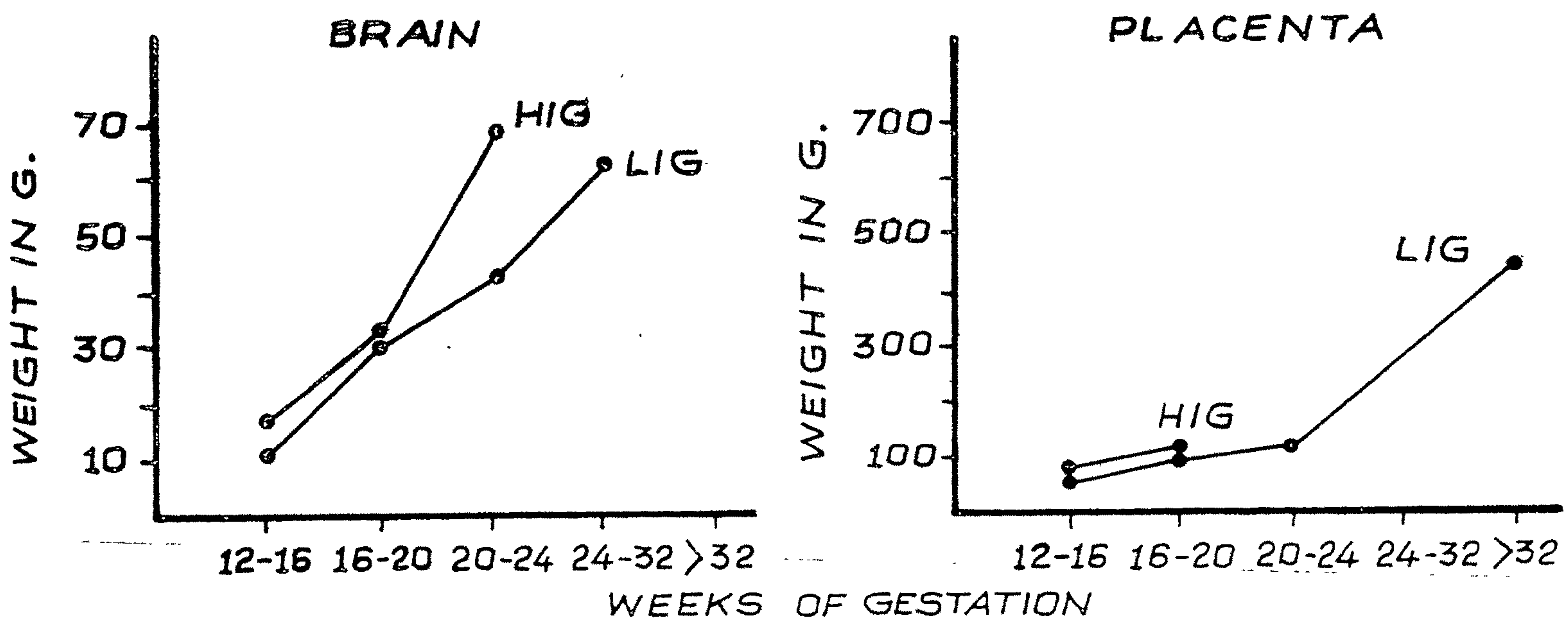


TABLE-66 : Fetal and tissue weights in low (LIG) and high (HIG) income groups as per cent values at 32 weeks*.

Parameter	Weeks of gestation							
	12-16		16-20		20-24		24-32	> 32
	LIG	HIG	LIG	HIG	LIG	HIG	LIG	LIG
	Values as percentage							
Fetal weight	3.3	4.9	10.3	10.4	17.3	21.5	30.8	100
Spleen weight	1.4	1.7	4.8	4.3	11.8	8.4	24.4	100
Liver weight	3.4	5.6	9.4	9.7	18.4	20.8	32.6	100
Lung weight	7.2	11.9	20.1	22.2	27.9	39.4	45.4	100
Kidney weight	5.2	8.6	12.9	14.1	22.6	36.7	43.7	100
Heart weight	3.3	7.2	10.6	10.5	18.2	28.9	37.2	100
Adrenal weight	5.3	9.1	15.2	16.4	19.6	31.9	44.1	100

* 32 weeks value taken is for LIG fetus.

showed the ratio of the weight of the fetus to that of the placenta to be 5:1 during third trimester to change to 7:1 suggesting that the fetus outgrows placenta at term. Further, Flexner (1948) clearly demonstrated a reduced transfer function of the placenta towards term. The weights of these organs in the low income group were lower than high income group but the proportion of them to body weight did not differ in two categories. (Figure 12). Further, the weight of the organs in growth retarded and growth normal fetuses classified on the basis of weight as per cent of expected weight for gestational age according to Widdowson's norms and using a cut-off point of 60% and their proportionate contribution to whole body weight are shown in Table-67. Although, growth retarded fetuses had smaller body weight and smaller organ weights (Figure 13), their contribution to total body weight did not differ in two categories. Thus, the organs of fetuses that are small-for-gestational age are also small as may be expected. In this connection, their smaller size may also be associated with retarded biochemical maturation. This has been found to be the case in the previous studies in this laboratory on rats in the case of lung (Khanna and Reddy, 1983), intestine (Arockiadoss, 1982) and brain (Rajalakshmi, 1980). Similar observations have been made by other investigators with regard to the lung (Hallman and Gluck, 1977; Lafeber <u>et al</u>, 1979; Schulte, 1981),

FIGURE:12 - FETAL TISSUE WEIGHTS AS PERCENT OF BODY WEIGHT IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

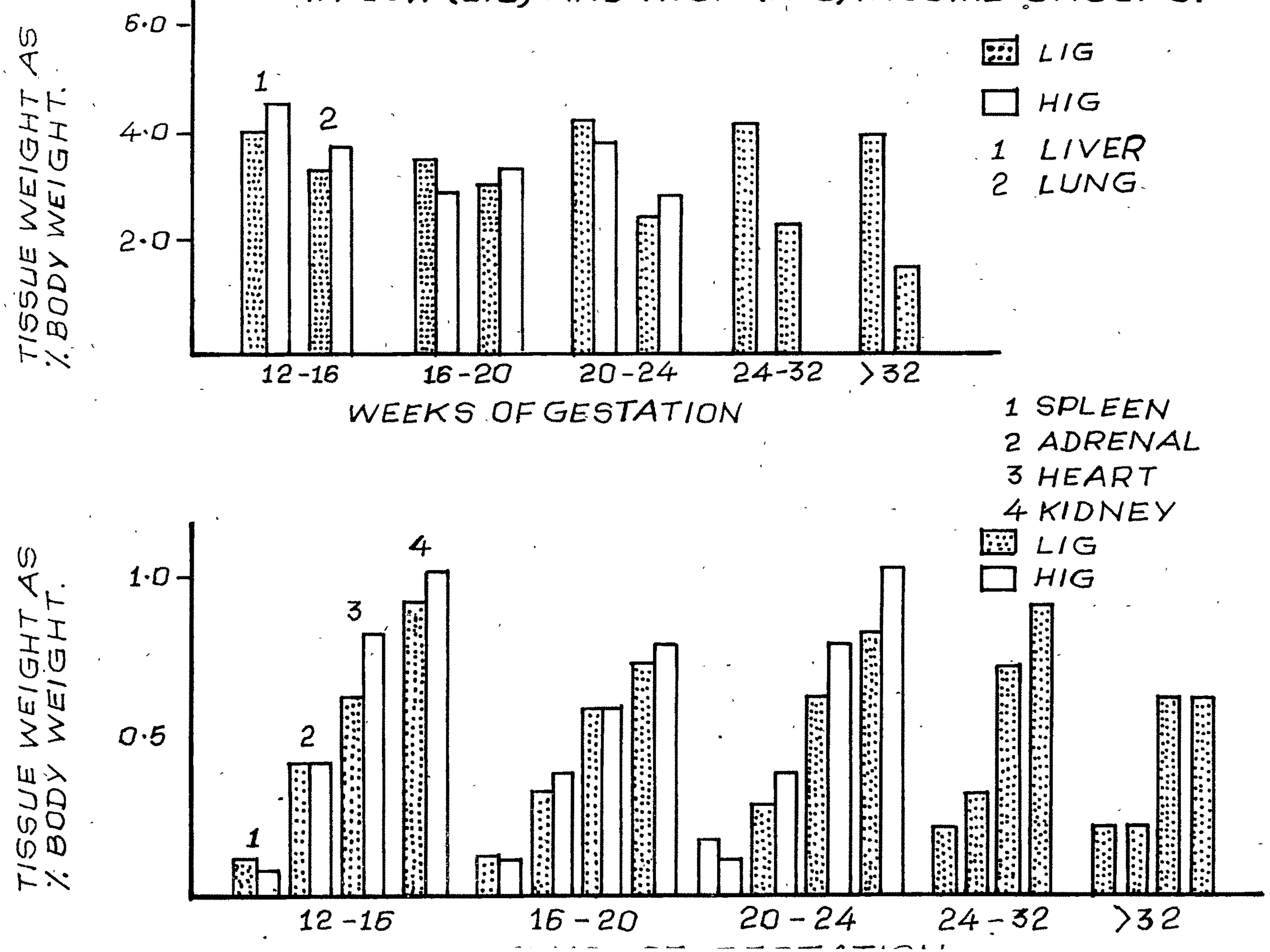
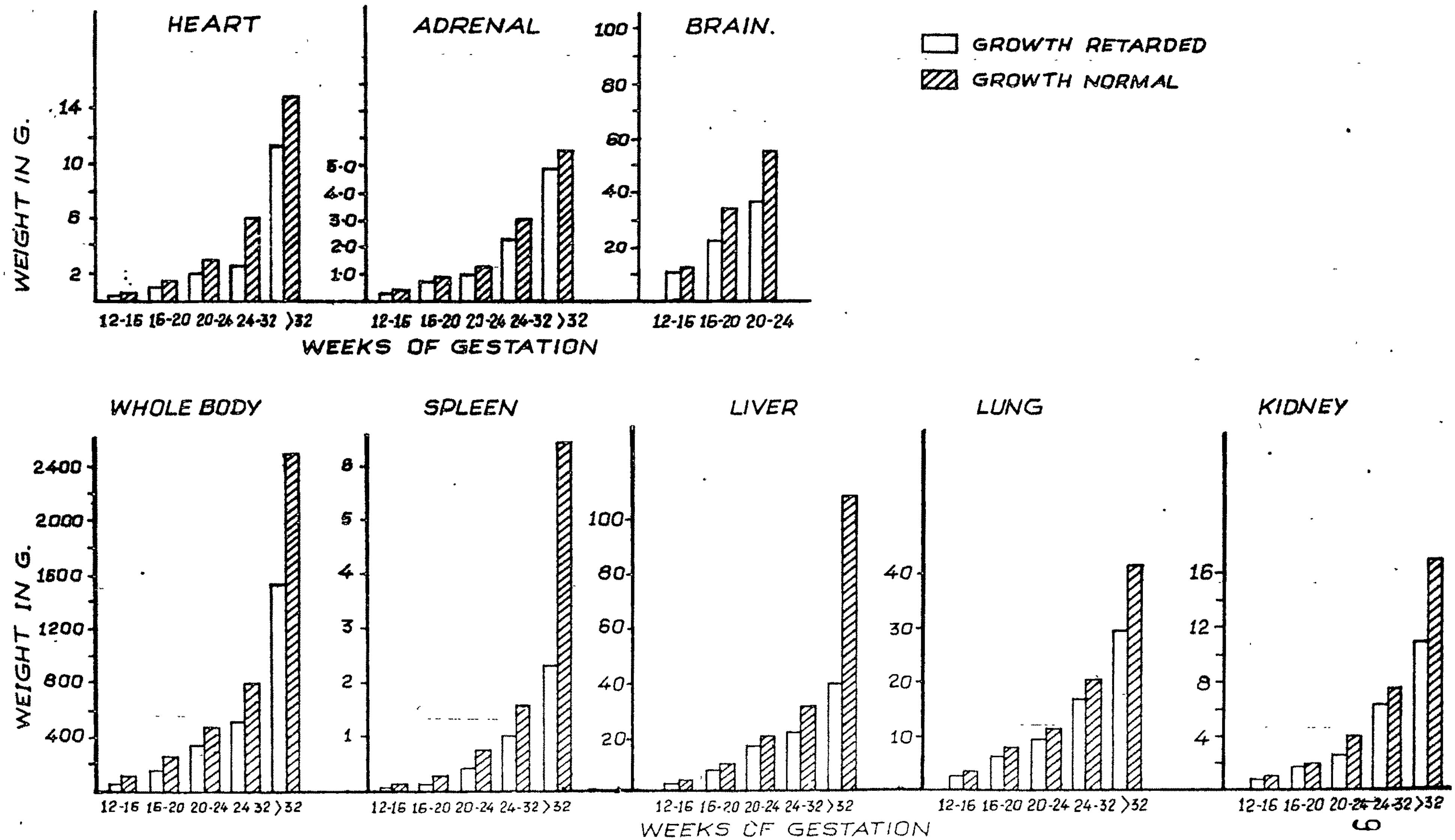


TABLE-67 : Fetal and tissue wrights in relation to fetal weight in growth-retarded and gro

Weeks of gestation	12-16		16-20		20-24		24-32
Weight as % expected wt.	< 60 (A)	> 60 (B)	< 60 (A)	> 60 (B)	< 60 (A)	> 60 (B)	< 60 (A)
	Mean ± S.E.; with no.of observations in pa-ren						
Weeks of gestation	14.0±0.05 (3)	14.0±0.30 (26)	17.6±0.34 (11)	18.0±0.15 (57)	21.6±0.29 (12)	22.2±0.52 (18)	26.6±0.76 (8)
Whole fetus weight (g)	51.3±3.67 (3)	89.5±10.1 (27)	151±11.90 (11)	245±9.88 (57)	335±22.9 (12)	474±54.8 (18)	511±105.1 (8)
as % expected wt	53 ± 1.52 (3)	79 ± 3.12 (27)	50±2.30 (11)	76±1.66 (57)	53±1.58 (12)	75±4.06 (18)	48±5.50 (8)
Spleen weight (g)	0.049±0.02 (2)	0.086±0.02 (21)	0.136±0.02 (11)	0.264±0.02 (54)	0.399± 0.05 (12)	0.749±0.26 (16)	0.744± 0.16 (7)
as % body wt.	0.095	0.096	0.090	0.110	0.120	0.160	0.150
A as % B	57		52		53		47
Liver weight (g)	2.699± 0.23 (3)	3.702± 0.52 (25)	6.42±0.74 (11)	8.82±0.43 (55)	15.04± 1.65 (12)	18.89± 2.39 (17)	20.10± 4.60 (8)
as % body wt.	5.3	4.1	4.3	3.6	4.5	4.0	3.9
A as % B	73		73		80		64
Lung weight (g)	2.113±	3.233±	5.88±0.55	7.41±0.45	8.96±0.67	11.05±	6.72±3.43

FIGURE-13 WEIGHTS OF ORGANS IN FETUSES IN RELATION TO GROWTH STATUS



the intestine (Brown, 1962; Shrader and Zeman, 1969; Vollrath, 1969; Kumar and Chase, 1971; Loh *et al*, 1971; Younoszai and Ranshaw, 1973), and the brain (Culley *et al*, 1966; Rajalakshmi and Nakhasi, 1974; Geel and Dreyfus, 1975; Reddy and Sastry, 1978). Further it is also reported that fetally growth retarded infants exhibits features such as poor pulmonary function (Schulte, 1981), and poor intestinal digestion and absorption (Koldovsky, 1982). The fetally growth retarded infant is also at risk of poor neuromotor development and mental retardation (Drillien, 1970; 1972; Davies and Stewart, 1975; Lubchenco, 1976; Winick, 1976; Vohr *et al*, 1978; Commey and Fitzhardinge, 1979).

Section B : SOMATIC MEASUREMENTS OF HUMAN FETUS AND NEONATE IN RELATION TO GESTATIONAL AGE, GROWTH STATUS AND PLANE OF MATERNAL NUTRITION.

The previous section was concerned with the pattern of organ growth during the course of human fetal development. The present section is concerned with different body measurements of the whole fetus and the neonate in relation to gestational age, growth status of the fetus or neonate and the social class of the mother which is found to influence food intake as it is well known that different somatic measurements are affected differentially in growth-retarded children (Gurney, 1969; Kondakio, 1969; McKay, 1969; McLaren and Pollit, 1970; Vishweswara Rao and Singh, 1970; Seone and Lathem, 1971; Kanawati and McLaren, 1972). For instance, height is less affected than weight (Jelliffe,1969; Seone and Lathem, 1971). Similarly, head circumference is less affected than arm circumference (Kanawati and McLaren, 1970). The indices used for these studies were the same as those used in young children with a few measurements added, such as total arm length, total leg length, thigh circumference and foot length.

As mentioned earlier, fetal growth retardation has been categorised into two classes by Gruenwald and others (Gruenwald, 1963; Ounsted and Taylor, 1971; Dubowitz, 1971;

Cook, 1977). They differ in their pathogenesis and postnatal course of development. In the first type, the infant has a normal crown-heel length but is deficient in subcutaneous fat and skeletal muscle and has also reduced weights of liver, spleen, adrenals and thymus glands. In the second type, the infant is symmetrically small at birth for gestational age as judged by external body dimensions but is not necessarily underweight for height. The former type has its origin in late pregnancy whereas the latter has its onset much earlier.

Most of the data on fetal growth have been obtained on the basis of birth weight. Somatic measurements have been widely studied in children (e.g. weight, height, head, chest and arm circumference) but only a few studies on crown heel length, head circumference, abdomen circumference and foot length have been carried out on newborn infants (Widdowson, 1968; Usher and McLean, 1969; Gruenwald, 1974; Lakshminarayan _et al_, 1974; Colaneri and Correa, 1977; James _et al_, 1979; Bhatia _et al_, 1981) whereas a few studies on weight, crown heel length, crown rump length, chest circumference, biparietal diameter and foot length have been made on human fetuses (Streter,1920; Widdowson, 1968; Campbell, 1970; Lakshminarayan _et al_, 1974; Birkbeck _et al_, 1975; Campbell and Thomas, 1977; Robinson, 1979) and their predictive value for future growth indicated by a few investigators (Gruenwald, 1974; Campbell and Thomas, 1977; Campogrande et al, 1977; James et al,1979). No such studies appear to have been carried out on human fetuses in relation to growth retardation and plane of maternal nutrition.

Thus it seemed worthwhile to obtain data on weight and somatic measurements in the case of fetuses and neonates in both low and high income groups at different stages of gestation.

The present studies on somatic measurements were carried out as part of the studies already described. The indices used were absolute weight, weight as compared to weight appropriate for gestational age, crown heel length, crown rump length, circumference of head, chest, abdomen, arm and thigh, total arm length, total leg length and foot length. They were studied in relation to gestational age, and the social class of the mother which is found to influence nutritional status markedly in this country.

Fetal growth in relation to gestational age, growth status and plane of maternal nutrition :-

The data on fetal weight and somatic measurements are presented in Table-68. As may be expected, all parameters increased with the progress of gestation in both groups

TABLE-68 : Human fetal weight and somatic measurements at different stages of gestation in low (LIG) and high (HIG) income groups.

Weeks of gestation		12-16	16-20	20-24	24-28	28-32	> 32
		Mean ± S.E.; with no. of observations in parentheses					
Weeks of gestation	LIG	13.5 ± 0.26 (36)	17.6 ± 0.16 (58)	21.5 ± 0.26 (32)	25.0 ± 0.23 (17)	30.2 ± 0.48 (9)	36.1 ± 1.00 (9)
	HIG	14.0 ± 0.39 (11)	18.1 ± 0.16 (37)	22.2 ± 0.69 (14)	-	-	-
Weight (g)	LIG	79.9 ± 7.23 (36)	206 ± 9.64 (58)	332 ± 17.5 (32)	615 ± 37.01 (17)	1026 ± 86.2 (9)	2156 ± 248.3 (9)
	HIG	97.7 ± 15.35 (11)	244 ± 9.29 (37)	485 ± 66.85 (4)	-	-	-
LIG as % HIG		82	84	68			
Weight as % expected weight@	LIG	75 ± 3.27 (36)	68 ± 1.92 (58)	57 ± 2.09 (32)	67 ± 3.46 (17)	60 ± 2.58 (9)	72 ± 5.8 (9)
	HIG	89 ± 6.00 (11)	77 ± 2.54 (37)	76 ± 3.59 (4)	-	-	-
LIG as % HIG		84	88	75			
Crown-heel length (cm)	LIG	15.9 ± 0.60 (34)	22.6 ± 0.40 (58)	25.9 ± 0.48 (30)	31.6 ± 0.60 (17)	36.9 ± 0.80 (9)	47.1 ± 1.87 (9)
	HIG	17.4 ± 1.36 (10)	23.9 ± 0.29 (37)	30.3 ± 1.69 (4)	-	-	-
LIG as % HIG		91	95	85			

TABLE-68 (CONTD.)

Weeks of gestation		12-16	16-20	20-24	24-28
				Mean ± S.E.; with no.of observat	
Shoulder to elbow length (cm)	LIG	2.9 ± 0.14 (32)	4.2 ± 0.09 (57)	4.9 ± 0.11 (30)	5.8 ± 0.16 (16)
	HIG	3.3 ± 0.25 (10)	4.5 ± 0.07 (36)	5.8 ± 0.32 (4)	-
LIG as % HIG		88	93	84	
Elbow to finger tip length (cm)	LIG	3.9 ± 0.20 (32)	5.7 ± 0.11 (57)	6.8 ± 0.16 (30)	8.6 ± 0.22 (16)
	HIG	4.2 ± 0.32 (10)	6.3 ± 0.14 (36)	7.9 ± 0.55 (4)	-
LIG as % HIG		93	90	86	
Hip to knee length (cm)	LIG	3.0 ± 0.16 (32)	4.4 ± 0.13 (57)	4.9 ± 0.13 (30)	6.2 ± 0.21 (16)
	HIG	3.4 ± 0.30 (10)	4.6 ± 0.10 (36)	5.6 ± 0.31 (4)	-
LIG as % HIG		88	96	88	

(Figure 14A & 14B). Fetal weights were less than those reported in the West for corresponding gestational ages (Montreewasuwat and Olson, 1979; Widdowson, 1968) but the values for early pregnancy were similar to those reported by Abravomich (1969) and Lakshminarayan et al, (1974) in this country. When weight was expressed as percentages of expressed as percentages of expected weights for gestational age using Widdowson's norms, the values in the two groups were comparable in early pregnancy but some deceleration of growth was observed in the low income group around mid-pregnancy. This deceleration continued till the age of 32 weeks after which the data suggest a trend for some degree of catch up growth. The deficits in fetal weights as compared to Widdowson's norms are consistent with x differences in birth weights (Thomson and Hytten, 1966; Rajalakshmi and Ramakrishnan, 1969; Rajalakshmi, 1971). The deceleration of fetal growth in the low income group around mid-pregnancy is consistent with other observations (Bhatt, 1982; Iyengar, 1984). The possibility of some catch up in late pregnancy is further confirmed by the data on frequency distribution where the proportion of low body weights was lower in a group of fetuses of greater than 24 weeks of gestation (Table-69).

FIGURE : 14-A : FETAL SOMATIC MEASUREMENTS AT DIFFERENT STAGES OF GESTATION IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

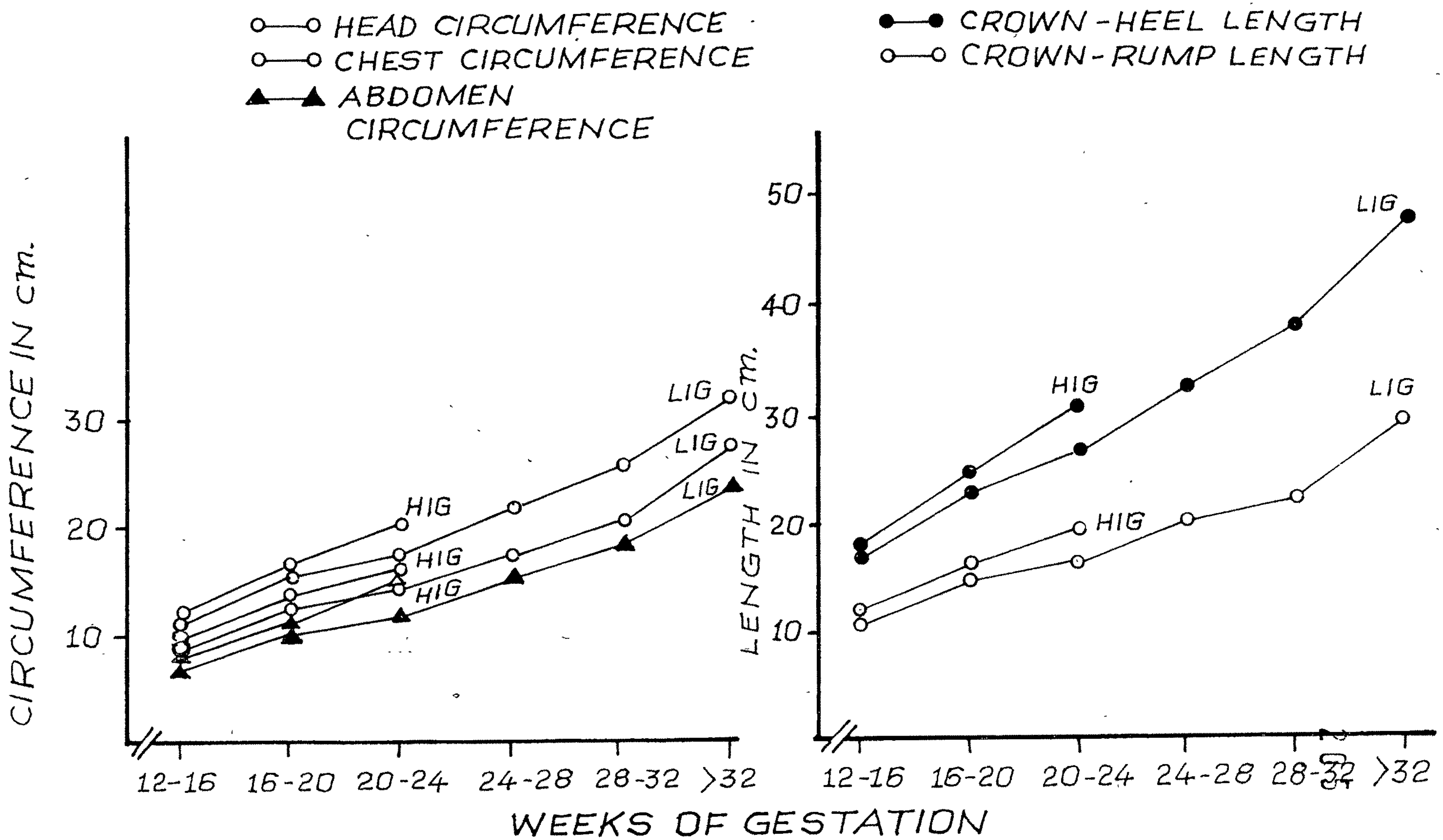


FIGURE -14 B: FETAL SOMATIC MEASUREMENTS AT DIFFERENT STAGES OF GESTATION IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

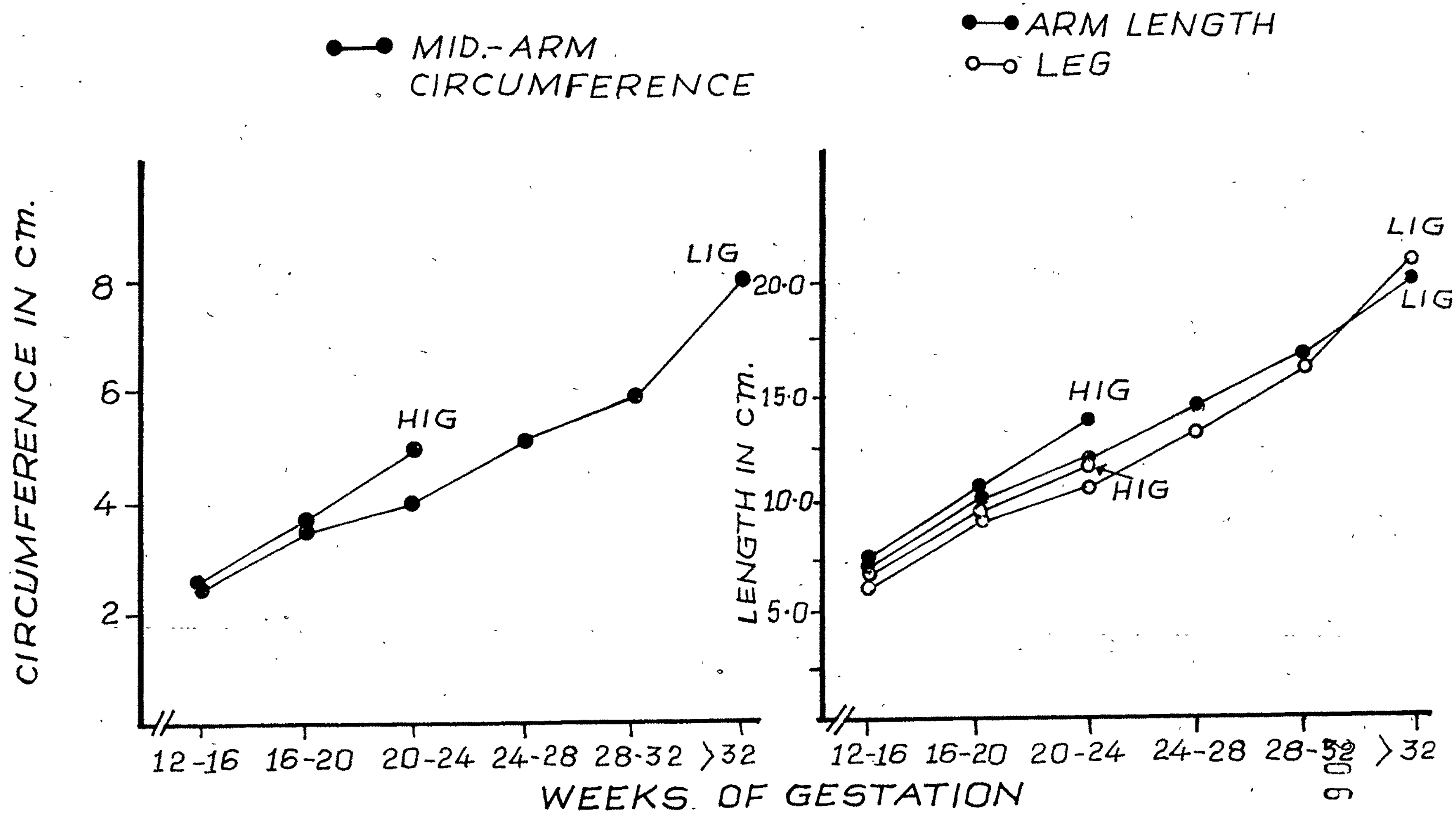


TABLE-69 : Frequency distribution of fetal weights as per cent expected weight for gestational age.

Fetal weight as % expected weight	< 70		70-90		>90	
Weeks of gestation	LIG	HIG	LIG	HIG	LIG	HIG
	Number with percentages in parentheses					
12 - 16	14 (39)	2 (18)	19 (53)	4 (36)	3 (8)	5 (45)
16 - 20	35 (60)	12 (32)	17 (29)	19 (51)	6 (10)	6 (16)
20 - 24	29 (88)	1 (25)	4 (12)	3 (75)	-	-
>24	22 (63)	-	11 (31)	2 (100)	2 (6)	-

All somatic measurements increased linearly with gestational age (Table-68). The values for fetuses in the two socio-economic groups were comparable for all parameters in early pregnancy but the differences were greater during mid-pregnancy (Figure-14A & 14B). When the values for the low income group were expressed as per cent of those for the high income group values, essentially the same pattern in growth was observed.

The linear increase in all other somatic measurements with gestational age are consistent with expectation and the observations of several others. However, all parameters were not studied simultaneously by other investigators. Streeter (1920) studied weight, sitting height and foot length in relation to menstrual age. Robbinson (1979) studied crown-rump length and biparietal diameter in relation to gestational age. Widdowson (1968) gave norms for crown-rump length and weight of fetuses at different stages of development. Birkbeck _et al_. (1975) showed a linear relationship between different linear dimensions and crown heel length.

The data were further analysed according to growth status. Grown retarded fetuses were smaller in size as compared to normal ones (Table-70). The results also suggest that weight and mid-arm circumference were affected to a greater extent as compared to all other

(Figure 14A & 14B). Fetal weights were less than those reported in the West for corresponding gestational ages (Montreewasuwat and Olson, 1979; Widdowson, 1968) but the values for early pregnancy were similar to those reported by Abravomich (1969) and Lakshminarayan et al, (1974) in this country. When weight was expressed as percentages of expressed as percentages of expected weights for gestational age using Widdowson's norms, the values in the two groups were comparable in early pregnancy but some deceleration of growth was observed in the low income group around mid-pregnancy. This deceleration continued till the age of 32 weeks after which the data suggest a trend for some degree of catch up growth. The deficits in fetal weights as compared to Widdowson's norms are consistent with a differences in birth weights (Thomson and Hytten, 1966; Rajalakshmi and Ramakrishnan, 1969; Rajalakshmi, 1971). The deceleration of fetal growth in the low income group around mid-pregnancy is consistent with other observations (Bhatt, 1982; Iyengar, 1984). The possibility of some catch up in late pregnancy is further confirmed by the data on frequency distribution where the proportion of low body weights was lower in a group of fetuses of greater than 24 weeks of gestation (Table-69).

TABLE-70 : Fetal somatic measurement in relation to growth status and gestational age in low (LIG) income groups.

Parameters		Weeks of gestation: 12-16		16-20		20-24		24-28		28-32	
		Weight as % expected weights									
		<90	>90	<90	>90	<90	>90	<90	>90	<90	>90
		Mean ± S.E.									
No.of subjects	LIG	33	3	51	5	32	-	17	-	9	-
	HIG	5	5	31	6	4	-	-	-	-	-
Weight (g)	LIG	74.8±6.89	142±17.7	193.0±8.42	337±39.3	332±17.6	-	615±37.2	-	1026±98.6	-
	HIG	71.2±10.7	132±24.1	228±8.60	324±10.3	485±70.2	-	-	-	-	-
Weight as % expected weight@	LIG	69±1.17	126±16.1	67±1.55	101±2.98	57±2.06	-	67±3.47	-	61±2.44	-
	HIG	73±4.24	105±7.13	71±1.79	102±6.09	77±3.56	-	-	-	-	-
Length (cm)	LIG	15.8±0.61	16±3.13	22.7±0.40	26.6±1.16	25.9±1.36	-	31.6±0.63	-	36.9±0.90	-
	HIG	15.1±1.76	19.7±1.68	23.7±0.30	25.1±0.34	30.3±1.79	-	-	-	-	-
Crown rump length (cm)	LIG	10.3±0.41	14.3±0.25	14.0±0.25	16.8±0.91	16.0±0.33	-	19.7±0.52	-	21.9±0.89	-
	HIG	9.7±1.13	13.0±1.21	14.9±0.24	16.1±0.49	18.9±1.66	-	-	-	-	-
Head circumference (cm)	LIG	10.1±0.47	13.0±0.50	14.6±0.22	18.2±1.03	17.4±0.38	-	21.4±0.74	-	24.5±0.77	-
	HIG	9.9±0.51	13.1±0.80	15.6±0.28	17.3±0.17	20.0±0.92	-	-	-	-	-

parameters. This is also observed in the case of growth-retarded children (Gurney, 1969; Kondakia, 1969; McKay, 1969; Rutishauser, 1969).

When ratios for different somatic measurements were calculated, all ratios except weight/head circumference and weight/crown heel length in low income group were comparable to high income group values (Table-71). This is because of the differences in weight in the two categories. These ratios did not change with the progress of gestation. Only head circumference and abdomen circumference tended to decrease from 20 weeks onwards. This is consistent with the observations of Campbell and Thomas (1977). This is because the contribution of brain weight to body weight declines with the progress of gestation.

Neonatal growth in relation to gestational age, growth status and plane of maternal nutrition :-

The data on birth weight and somatic measurements are presented in Table-72. As may be expected, all parameters increased with the progress of gestation. Birth weights were low in the low income group as compared to those in the high income group. The mean birth weights of infants born in the low and high income groups are similar to those reported by others (Kulkarni et al, 1959; Achar and Yankauer,

TABLE-71 : Ratios for different somatic measurements in fetuses in low (LIG) and high (HIG) income groups.

Weeks of gestation		12-16	16-20	20-24	24-28	28-32	> 32
		Mean ± S.E., with no. of observations in parentheses					
Weight/Head circumference (kg/cm)	LIG	7.76 (34)	13.8 (57)	19.1 (30)	28.6 (16)	40.4 (9)	68.4 (9)
	HIG	8.50 (10)	15.4 (36)	24.3 (4)	-	-	-
Mid-arm circumference/Head circumference	LIG	0.23 (34)	0.23 (57)	0.23 (30)	0.24 (16)	0.23 (9)	0.26 (9)
	HIG	0.22 (10)	0.23 (36)	0.25 (4)	-	-	-
Chest circumference/head circumference	LIG	0.82 (34)	0.80 (57)	0.79 (30)	0.80 (16)	0.83 (9)	0.86 (9)
	HIG	0.81 (10)	0.82 (36)	0.79 (4)	-	-	-
Chest circumference/Abdomen circumference	LIG	1.22 (34)	1.21 (57)	1.17 (30)	1.15 (16)	1.17 (9)	1.16 (9)
	HIG	1.18 (10)	1.26 (36)	1.17 (4)	-	-	-
Mid-arm circumference/Chest circumference	LIG	0.29 (34)	0.29 (57)	0.29 (30)	0.30 (16)	0.28 (9)	0.30 (9)
	HIG	0.27 (10)	0.28 (36)	1.17 (4)	-	-	-
Crown rump length/Crown heel length	LIG	0.66 (34)	0.63 (57)	0.61 (30)	0.56 (16)	0.59 (9)	0.61 (9)
	HIG	0.65 (10)	0.63 (36)	0.62 (4)	-	-	-

TABLE-71 (Contd.)

Weeks of gestation		12-16	16-20	20-24	24-28	28-32	>32
Weight/ Crown heel length (Kg/Cm)	LIG	5.03 (34)	9.12 (57)	12.8 (30)	19.5 (16)	27.8 (9)	45.8 (9)
	HIG	5.61 (10)	10.20(36)	16.0 (4)	-	-	-
Head circumference/Abdomen circumference	LIG	1.49 (34)	1.52 (57)	1.49 (30)	1.44 (16)	1.40 (9)	1.35 (9)
	HIG	1.46 (10)	1.53 (36)	1.48 (4)	-	-	-

TABLE-72 : Somatic measurements of newborn infants in relation to gestational age in low (LIG) and high (HIG) income groups.

Parameters		Gestational age of the infant at birth (weeks)		
		$\angle$ 36	36-38	> 38
1		2	3	4
		Mean ± S.E.; Values in parentheses are LIG values as % of HIG values.		
No. of subjects	LIG	43	84	64
	HIG	15	54	49
Birth weight (kg)	LIG	2.24 ± 0.06**** (88)	2.54 ± 0.04**** (85)	2.64 ± 0.04**** (86)
	HIG	2.56 ± 0.06	2.98 ± 0.05	3.08 ± 0.05
Birth weight as % expected weight@	LIG	89 ± 1.89* (94)	82 ± 1.21**** (85)	74 ± 1.20**** (88)
	HIG	95 ± 2.62	96 ± 1.69	84 ± 1.48
Crown-heel length (cm)	LIG	46.0 ± 0.49*** (95)	48.3 ± 0.26**** (96)	49.1 ± 0.24**** (96)
	HIG	48.6 ± 0.72	50.5 ± 0.29	50.9 ± 0.29
Crown-rump length (cm)	LIG	26.8 ± 0.44** (96)	27.8 ± 0.21**** (95)	27.6 ± 0.50*** (94)
	HIG	27.8 ± 0.21	29.4 ± 0.26	29.5 ± 0.26

TABLE-72 (Contd.)

1		2		3		4	
Head circumference (cm)	LIG	31.3 ± 0.24**	(96)	32.4 ± 0.14****	(96)	32.7 ± 0.15****	(96)
	HIG	32.6 ± 0.52		33.8 ± 0.15		33.9 ± 0.15	
Chest circumference (cm)	LIG	28.9 ± 0.33 NS	(97)	30.6 ± 0.23****	(96)	30.9 ± 0.22****	(96)
	HIG	29.9 ± 0.52		32.0 ± 0.21		32.1 ± 0.27	
Abdomen circumference (cm)	LIG	28.1 ± 0.35 NS	(101)	29.0 ± 0.24****	(96)	29.6 ± 0.25	(98)
	HIG	27.9 ± 0.70		30.3 ± 0.29		30.3 ± 0.31	
Mid-arm circumference (cm)	LIG	8.9 ± 0.13**	(94)	9.5 ± 0.09****	(93)	9.6 ± 0.09****	(94)
	HIG	9.5 ± 0.22		10.2 ± 0.13		10.2 ± 0.13	
$\frac{\text{Weight}}{\text{crown-heel length}}$ $\left(\frac{kg}{cm}\right)$	LIG	0.049 ± 0.0009**	(96)	0.053 ± 0.0006****	(90)	0.054 ± 0.0007****	(90)
	HIG	0.051 ± 0.0010		0.059 ± 0.0009		0.060 ± 0.0009	
$\frac{\text{Crown rump length}}{\text{Crown-heel length}}$	LIG	0.57 ± 0.004***	(97)	0.58 ± 0.003 NS	(100)	0.57 ± 0.002 NS	(98)
	HIG	0.59 ± 0.004		0.58 ± 0.003		0.58 ± 0.003	

TABLE-72 (Contd.)

1		2		3		4	
Shoulder to elbow length (cm)	LIG	9.5 ± 0.14 NS	(97)	9.9 ± 0.09 ****	(93)	10.1 ± 0.09 **	(97)
	HIG	9.8 ± 0.15		10.6 ± 0.11		10.4 ± 0.12	
Elbow to finger tip length (cm)	LIG	12.9 ± 0.14 ****	(93)	13.3 ± 0.09 ****	(94)	13.4 ± 0.10 ****	(92)
	HIG	13.9 ± 0.16		14.2 ± 0.14		14.5 ± 0.14	
Total arm length (cm)	LIG	22.4 ± 0.24 ****	(95)	23.3 ± 0.16 ****	(94)	23.6 ± 0.15 ****	(95)
	HIG	23.7 ± 0.26	24x8	24.8 ± 0.18		24.9 ± 0.22	
Hip to knee length (cm)	LIG	9.0 ± 0.14 NS	(100)	9.5 ± 0.10 NS	(98)	9.5 ± 0.11 NS	(98)
	HIG	9.0 ± 0.18		9.7 ± 0.09		9.7 ± 0.12	
Knee to heel length (cm)	LIG	11.7 ± 0.18 ****	(90)	12.2 ± 0.10 ****	(90)	12.5 ± 0.12 **	(95)
	HIG	13.0 ± 0.23		13.5 ± 0.12		13.2 ± 0.27	
Total leg length (cm)	LIG	20.7 ± 0.24 ***	(94)	21.7 ± 0.15 ****	(94)	21.9 ± 0.15 ****	(90)
	HIG	22.0 ± 0.36		23.2 ± 0.15		24.4 ± 0.25	

TABLE-72 (Contd.)

1		2	3	4
Foot length (cm)	LIG	7.7 ± 0.13*** (93)	8.2 ± 0.10**** (94)	8.3 ± 0.10*** (95)
	HIG	8.3 ± 0.12	8.7 ± 0.07	8.7 ± 0.06
Thigh circumference (cm)	LIG	13.1 ± 0.29*** (90)	14.0 ± 0.23**** (90)	14.3 ± 0.27* (94)
	HIG	14.5 ± 0.38	15.5 ± 0.19	15.2 ± 0.41

@ Values given by Widdowson (1968)

Values significantly different from high income group

**** P ∠0.001; *** P ∠0.01; ** P ∠0.05; * P ∠0.10.

1962; Udani, 1963; Rajalakshmi and Ramakrishnan, 1969; Rajalakshmi, 1971; Rajalakshmi et al, 1978). These social class differences are in accordance with the higher proportion of small-for-gestational age infants among the poor (Achar and Yankauer, 1962; Udani, 1963; Rajalakshmi, 1971). The possibility of some catch up growth in late pregnancy is suggested by differences in the magnitude of the social class differences with regard to birth weights in premature infants as compared to full term infants.

All the linear measurements increase with the progress of gestational age as might be expected (Figure-15A, 15B, 15C & 15D). The rate of growth decrease after the age of 38 weeks. A distinctive fall in the rate of growth after 36 weeks onwards have been reported by number of investigators (Gruenwald, 1966; Usher and McLean, 1969). This was true of all parameters (Figures-15A, 15B, 15C & 15D). This deceleration in the rate of growth is possibly associated with deterioration in placental function (Gruenwald, 1966). Other patterns are also observed. In the series of Thomson et al.(1968), the weights continued to increase even after 36 weeks i.e. upto 38 weeks and in the series of Lubchenko et al. (1963), the weights were lower after 35 weeks. Colaneri and Correa (1977) showed two peaks in birth weights at 31 and 38 weeks of gestation.

FIGURE:15-A-SOMATIC MEASUREMENTS IN NEONATES AT DIFFERENT GESTATIONAL AGES IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

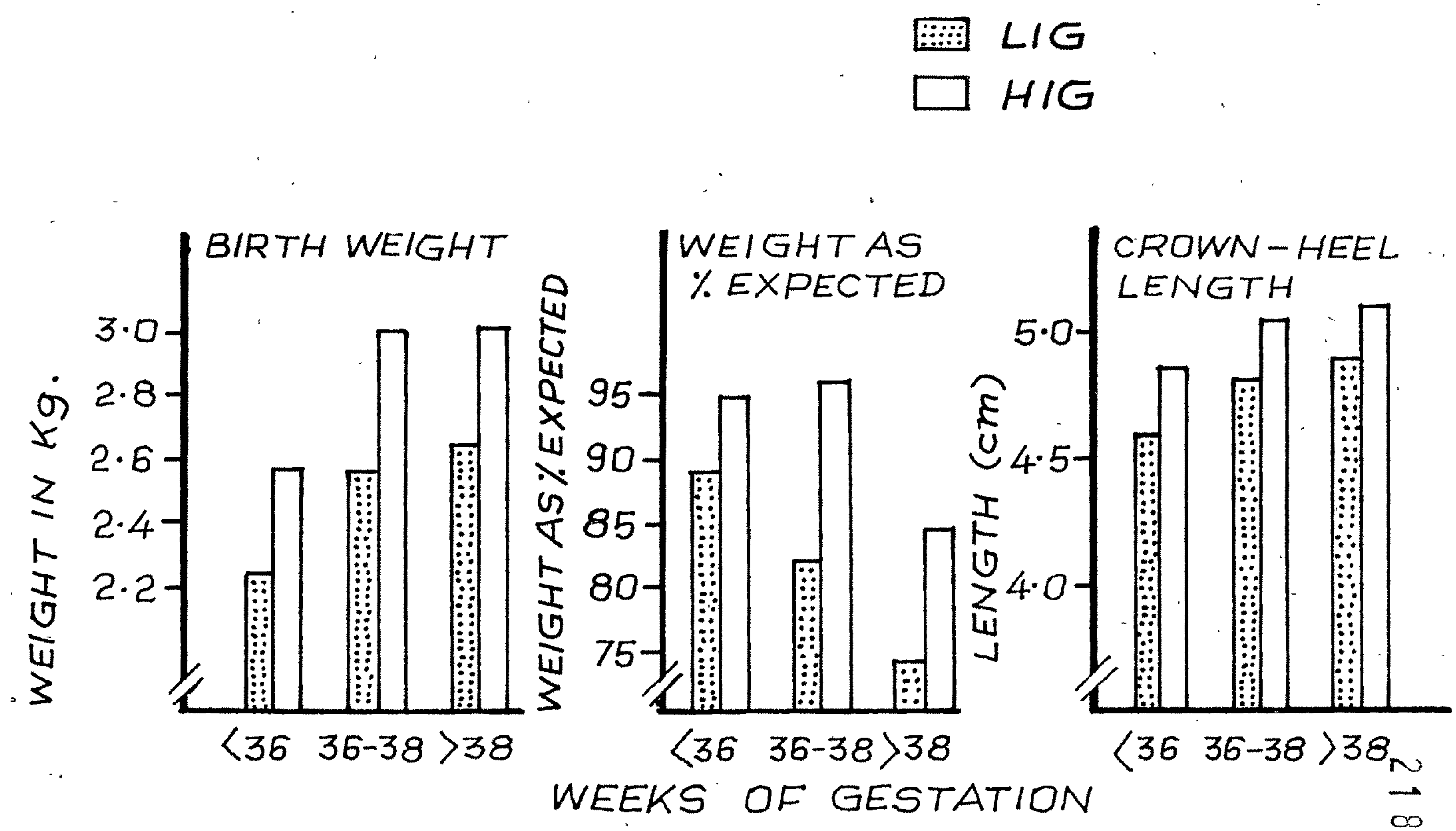


FIGURE:15-B: SOMATIC MEASUREMENTS IN NEONATES AT DIFFERENT GESTATIONAL AGES IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

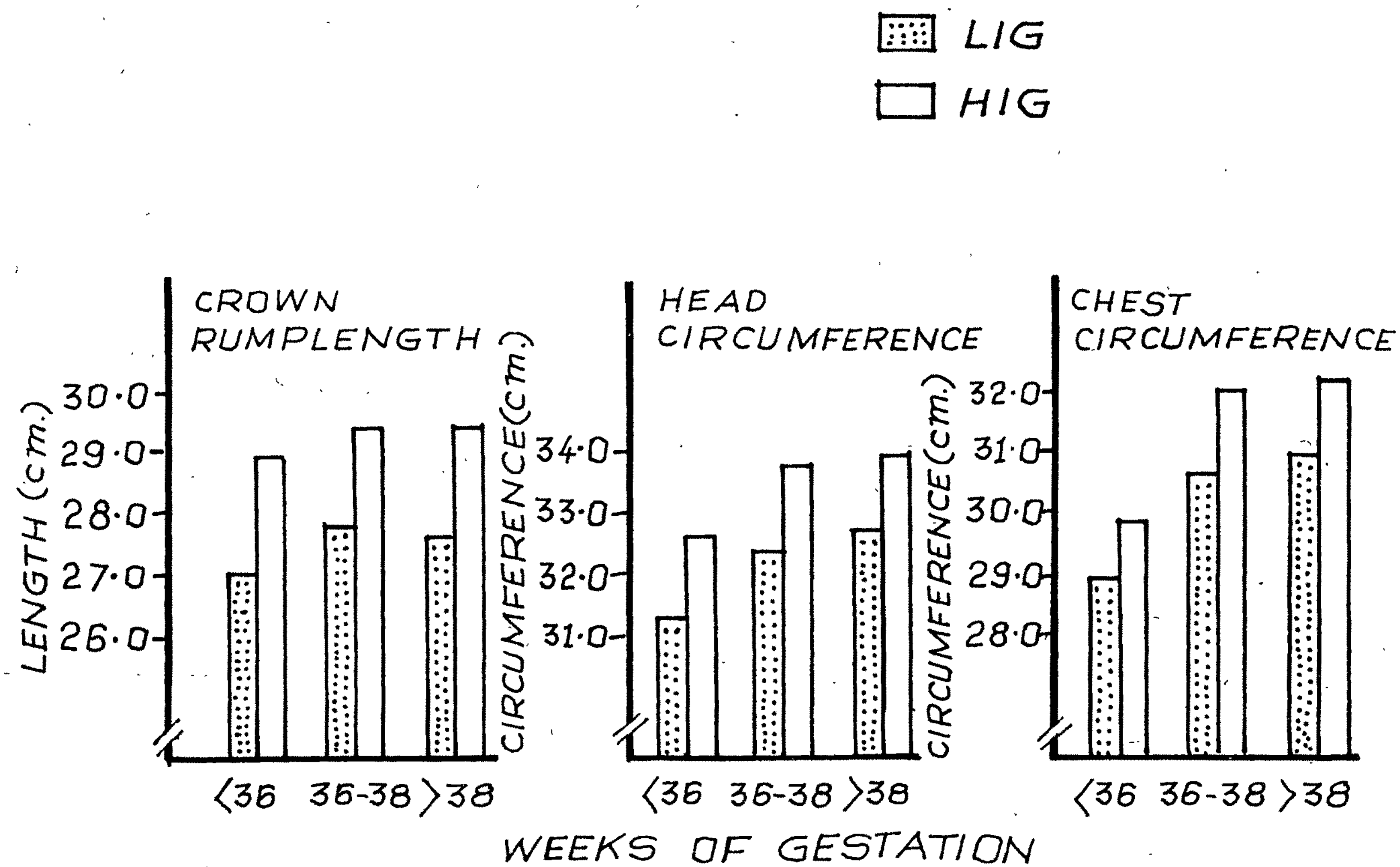


FIGURE:15-C: SOMATIC MEASUREMENTS IN NEONATES AT DIFFERENT GESTATIONAL AGES IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.

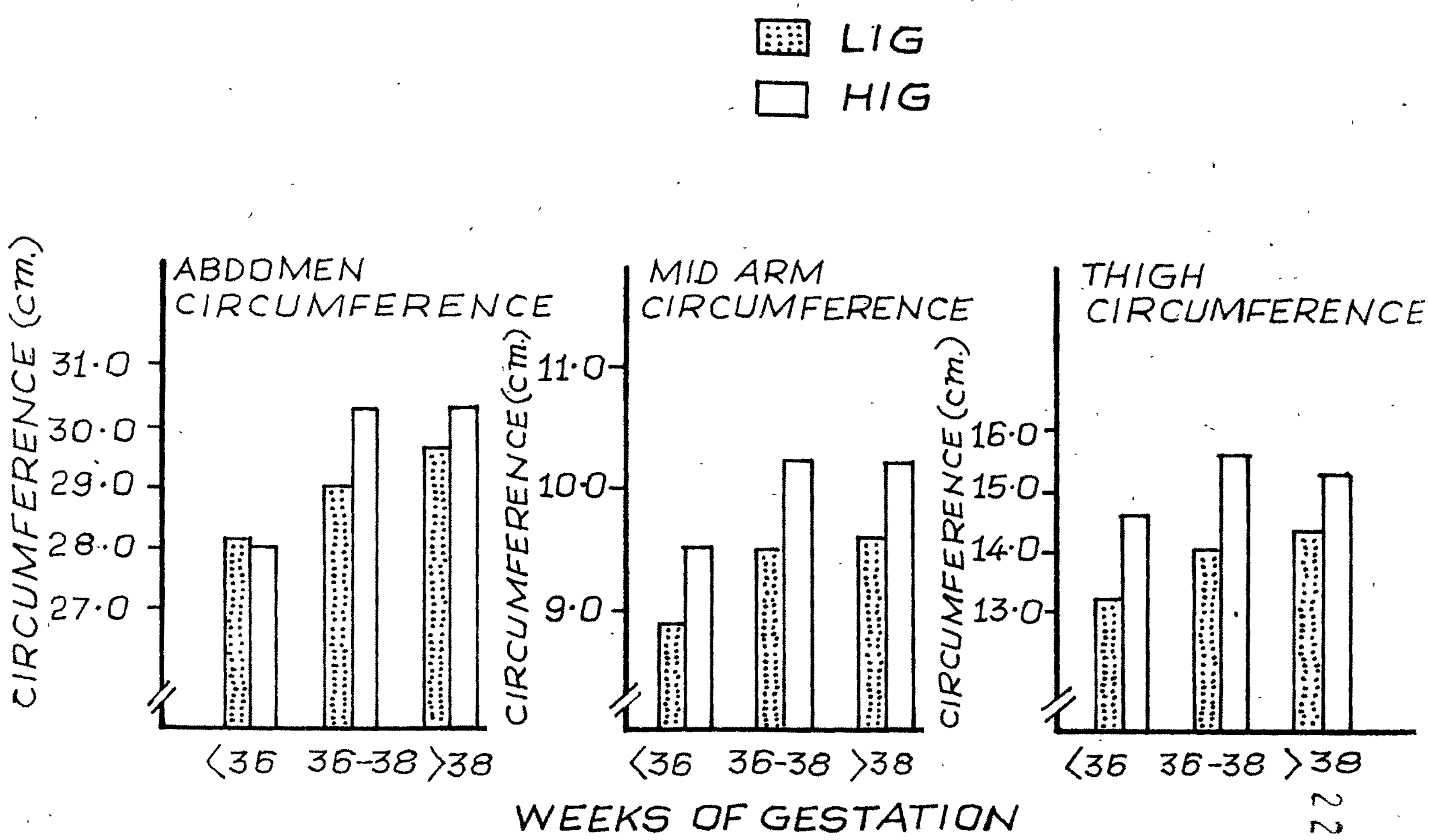
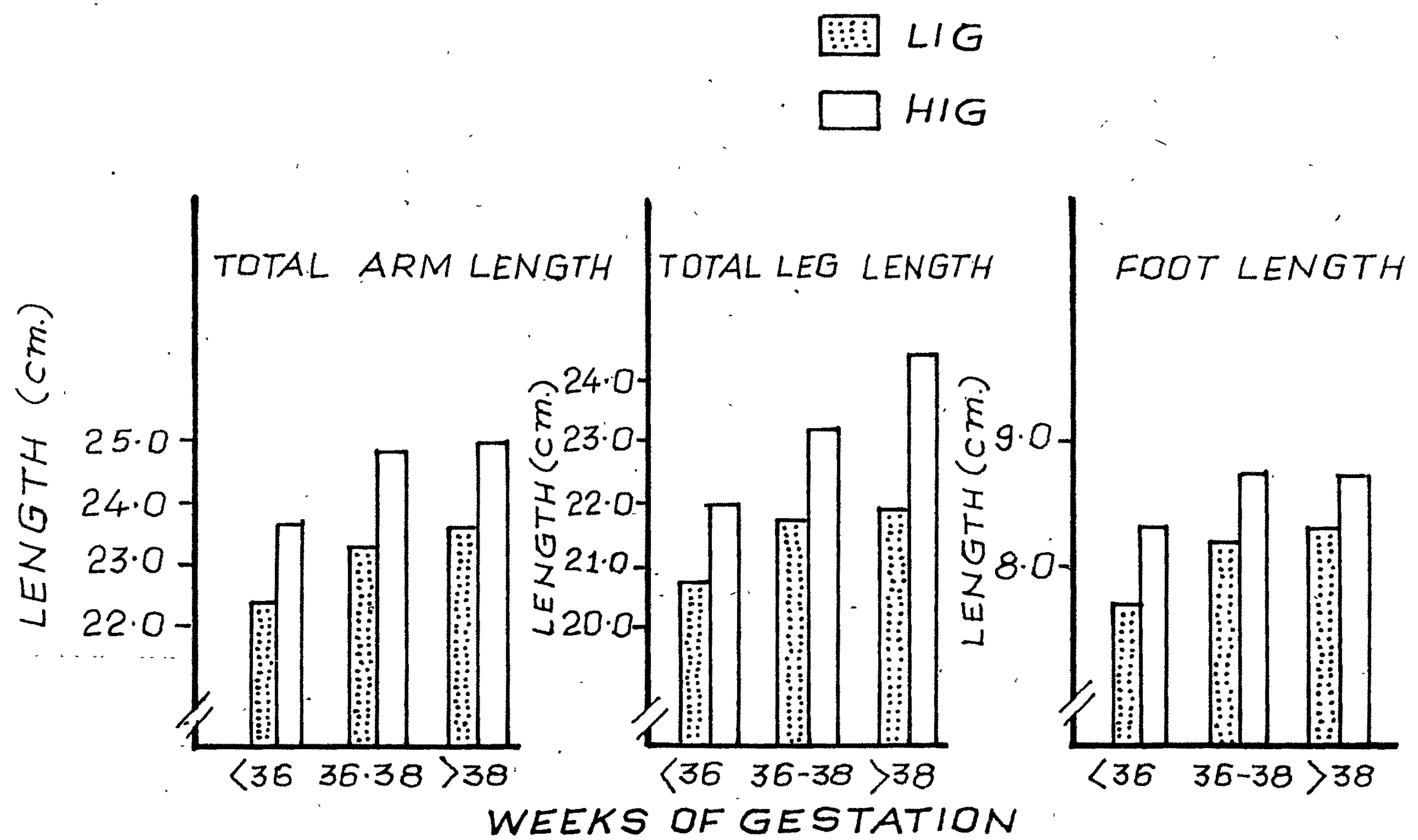


FIGURE :15-D: SOMATIC MEASUREMENTS IN NEONATES AT DIFFERENT GESTATIONAL AGES IN LOW (LIG) AND HIGH (HIG) INCOME GROUPS.



The linear increments in all other somatic measurements with gestational age are consistent with expectation and other reports (Widdowson, 1968; Usher and McLean, 1969; Bhatia et al, 1981).

The data were further analysed according to birth weights. Neonates with lower body weights were smaller in all dimensions in both groups in both premature and full term infants (Tables-73 & 74).

The data on full term and premature infants in low and high income groups are presented in Table-75. The values for all parameters were higher in the high income group as compared to the low income group in both categories. Birth weights of premature infants in the high income group were similar to those of full term infants in the low income group. This suggests the higher maturity of premature infants in high income group as compared to low income group. Their greater maturity has also been observed with regard to body stores of vitamin A (Shah, 1986). Further, the data suggested that weight and mid-arm circumference were affected to a greater extent as compared to all other parameters. This is clearly seen when infants with weights appropriate for gestational age were compared with small-for-gestational age infants and premature infants (Table-76). Also, all

TABLE-73 : Somatic measurements of premature infants in low (LIG) and high (HIG) income groups according to birth weight.

Parameters		Weight (kg) of premature infants at birth		
		∠2.0	2.0 - 2.5	2.5 - 3.0
			Mean ± S.E.	
No.of subjects	LIG	12	14	17
	HIG	0	9	7
Birth weight (kg)	LIG	1.76 ± 0.03	2.15 ± 0.03	2.64 ± 0.03
	HIG	-	2.30 ± 0.05	2.82 ± 0.05
Birth weight as percentage expected@	LIG	76 ± 2.8	87 ± 2.45	98 ± 1.4
	HIG	-	87 ± 2.28	103 ± 2.6
Crown-heel length (cm)	LIG	41.7 ± 0.36	46.3 ± 0.43	48.9 ± 0.35
	HIG	-	46.8 ± 1.05	49.8 ± 1.07
Crown-rump length (cm)	LIG	23.6 ± 0.24	26.0 ± 0.57	28.2 ± 0.30
	HIG	-	27.5 ± 0.71	29.7 ± 0.91
Head circumference (cm)	LIG	29.8 ± 0.29	31.2 ± 0.54	32.5 ± 0.21
	HIG	-	32.2 ± 0.80	33.1 ± 0.61

TABLE-73 (Contd.)

1		2	3	4
Chest circumference (cm)	LIG	26.8 ± 0.48	28.4 ± 0.28	30.7 ± 0.37
	HIG	-	29.5 ± 0.79	30.5 ± 0.61
Abdomen circumference (cm)	LIG	26.5 ± 0.44	27.7 ± 0.58	29.5 ± 0.52
	HIG	-	26.6 ± 0.94	29.6 ± 0.71
Mid-arm circumference (cm)	LIG	8.0 ± 0.18	8.4 ± 0.19	9.4 ± 0.15
	HIG	-	9.1 ± 0.32	9.9+_0.37
$\frac{\text{Crown heel length}}{\text{crown-rump length}}$	LIG	0.56 ± 0.01	0.56 ± 0.01	0.58 ± 0.004
	HIG	-	0.58 ± 0.004	0.59 ± 0.008
Elbow to finger length (cm)	LIG	11.9 ± 0.18	13.0 ± 0.19	13.2 ± 0.20
	HIG	-	13.7 ± 0.23	14.1 ± 0.20
Shoulder to elbow length (cm)	LIG	8.8 ± 0.25	9.2 ± 0.19	10.1 ± 0.15
	HIG	-	9.8 ± 0.18	9.8 ± 0.26
$\frac{\text{Weight}}{\text{Crown heel length}}$ (kg/cm)	LIG	0.043 ± 0.006	0.046 ± 0.0008	0.054 ± 0.0006
	HIG	-	0.049 ± 0.0005	0.054 ± 0.0002

TABLE-73 (Contd.)

1		2	3	4
Total arm length (cm)	LIG	20.7 ± 0.39	22.3 ± 0.27	23.3 ± 0.27
	HIG	-	23.6 ± 0.38	23.9 ± 0.42
Hip to knee length (cm)	LIG	8.7 ± 0.27	8.8 ± 0.24	9.4 ± 0.25
	HIG	-	9.0 ± 0.23	9.1 ± 0.30
Knee to heel length (cm)	LIG	10.6 ± 0.23	11.7 ± 0.28	12.5 ± 0.21
	HIG	-	12.8 ± 0.18	13.6 ± 0.55
Total leg length (cm)	LIG	19.3 ± 0.33	20.5 ± 0.37	21.9 ± 0.24
	HIG	-	21.8 ± 0.34	22.5 ± 0.75
Foot length (cm)	LIG	7.3 ± 0.20	7.8 ± 0.07	7.7 ± 0.25
	HIG	-	8.2 ± 0.12	8.5 ± 0.22
Thigh circumference (cm)	LIG	11.8 ± 0.94	12.9 ± 0.30	13.6 ± 0.36
	HIG	-	13.9 ± 0.50	15.5 ± 0.40

@ Weight as given by Widdowson (1968).

TABLE-74 : Somatic measurements of full term infants in low (LIG) and high (HIG) income groups according to birth weight.

Parameters		Weight (kg) of full term infants at birth				
		< 2.0	2.0 - 2.5	2.5 - 3.0	3.0 - 3.5	> 3.5
1		2	3	4	5	6
		Mean ± S.E.				
No.of subjects	LIG	5	37	83	25	0
	HIG	0	1	44	52	7
Birth weight (kg)	LIG	1.73 ± 0.04	2.24 ± 0.03	2.64 ± 0.01	3.05 ± 0.02	-
	HIG	-	2.1	2.70 ± 0.02	3.22 ± 0.02	3.72 ± 0.05
Birth weight as % expected@	LIG	53 ± 3.56	69 ± 1.20	79 ± 0.88	92 ± 1.45	-
	HIG	-	64	82 ± 1.38	96 ± 1.64	101 ± 2.66
Crown-heel length (cm)	LIG	43.5 ± 1.36	46.9 ± 0.31	49.3 ± 0.19	50.6 ± 0.35	-
	HIG	-	49.0	49.6 ± 0.25	51.4 ± 0.28	52.7 ± 0.59
Crown-rump length (cm)	LIG	25.0 ± 1.53	27.3 ± 0.22	28.3 ± 0.16	28.9 ± 0.34	-
	HIG	-	29.0	29.1 ± 0.25	29.8 ± 0.29	30.1 ± 0.40

TABLE-74 (Contd.)

1		2	3	4	5	6
Head circumference (cm)	LIG	31.8 ± 0.56	31.5 ± 0.19	32.8 ± 0.13	33.7 ± 0.14	-
	HIG	-	33.5	33.4 ± 0,12	34.2 ± 0.16	34.4 ± 0.23
Chest circumference (cm)	LIG	27.0 ± 0.76	29.0 ± 0.26	31.2 ± 0.14	32.6 ± 0.24	-
	HIG	-	32.0	31.0 ± 0.18	32.7 ± 0.17	34.5 ± 0.57
Abdomen circumference (cm)	LIG	26.0 ± 0.79	27.7 ± 0.34	29.7 ± 0.16	31.2 ± 0.27	-
	HIG	-	30.0	29.1 ± 0.25	31.0 ± 0.24	33.2 ± 0.70
Mid-arm circumference (cm)	LIG	7.6 ± 0.19	9.1 ± 0.13	9.7 ± 0.07	10.0 ± 0.15	-
	HIG	-	9.0	9.4 ± 0.13	10.5 ± 0.10	11.1 ± 0.24
Weight / Crown heel length (kg/cm)	LIG	0.040 ± 0.001	0.048 ± 0.0005	0.054±0.0003	0.060±0.0006	-
	HIG	-	0.043	0.054±0.0004	0.063±0.0006	0.071±0.0008
Crown rump length / Crown heel length	LIG	0.59 ± 0.023	0.58 ± 0.004	0.58 ± 0.002	0.56 ± 0.006	-
	HIG	-	0.59	0.59 ± 0.003	0.58 ± 0.004	0.58 ± 0.01
Shoulder to elbow length (cm)	LIG	9.1 ± 0.10	9.8 ± 0.13	10.0 ± 0.14	10.4 ± 0.16	-
	HIG	-	10.0	10.3 ± 0.14	10.7 ± 0.09	10.9 ± 0.14

TABLE-74 (Contd.)

1		2	3	4	5	6
Elbow to finger tip length (cm)	LIG	12.5 ± 0.16	12.9 ± 0.12	13.6 ± 0.09	13.9 ± 0.16	-
	HIG	-	15.0	13.9 ± 0.15	14.6 ± 0.13	15.3 ± 0.18
Total arm length (cm)	LIG	21.6 ± 0.10	22.7 ± 0.21	23.6 ± 0.16	24.3 ± 0.25	-
	HIG	-	25.0	24.1 ± 0.18	25.3 ± 0.20	26.1 ± 0.26
Knee to hip length (cm)	LIG	8.5 ± 0.32	9.0 ± 0.13	9.7 ± 0.08	10.1 ± 0.16	-
	HIG	-	9.0	9.6 ± 0.08	9.7 ± 0.10	10.4 ± 0.49
Knee to heel length (cm)	LIG	11.2 ± 0.25	12.0 ± 0.13	12.8 ± 0.12	12.5 ± 0.20	-
	HIG	-	14.0	13.4 ± 0.13	13.5 ± 0.14	13.9 ± 0.32
Total leg length (cm)	LIG	19.7 ± 0.46	21.0 ± 0.19	22.5 ± 0.12	22.6 ± 0.21	-
	HIG	-	23.0	23.0 ± 0.17	23.2 ± 0.15	24.3 ± 0.24
Foot length (cm)	LIG	7.3 ± 0.19	8.0 ± 0.12	8.3 ± 0.10	9.0 ± 0.11	-
	HIG	-	9.4	8.5 ± 0.07	8.8 ± 0.06	8.8 ± 0.12
Thigh circumference (cm)	LIG	11.5 ± 0.50	13.3 ± 0.33	14.5 ± 0.17	15.9 ± 0.23	-
	HIG	-	14.0	15.0 ± 0.15	16.0 ± 0.18	17.5 ± 0.89

@ Values given by Widdowson (1968).

TABLE-75 : Somatic measurements of premature and full-term infants in low (LIG) and high (HIG) income groups.

Parameters	Full term (FT)		Premature (PM)		LIG as % HIG		PM as % FT	
	LIG	HIG	LIG	HIG	FT	PM	FT	PM
			Mean ± S.E.					
Birth weight (kg)	2.59±0.03	3.0 ± 0.04	2.24 ± 0.06	2.56 ± 0.06	86	88	86	85
Birth weight as % expected weight@	78 ± 0.95	90 ± 1.30	89 ± 1.89	95 ± 2.62	87	94	114	106
Crown-heel length (cm)	48.7 ± 0.19	50.7 ± 0.20	46.0 ± 0.49	48.6 ± 0.72	96	95	94	96
Crown-rump length (cm)	28.0 ± 0.14	29.5 ± 0.19	26.8 ± 0.44	27.8 ± 0.21	95	96	96	94
Head circumference (cm)	32.6 ± 0.11	33.9 ± 0.10	31.3 ± 0.24	32.6 ± 0.52	96	96	96	96
Chest circumference (cm)	30.6 ± 0.25	32.1 ± 0.17	28.9 ± 0.33	29.9 ± 0.52	95	97	94	93
Abdomen circumference (cm)	29.3 ± 0.17	30.4 ± 0.22	28.1 ± 0.35	27.9 ± 0.70	96	101	96	92
Mid-arm circumference (cm)	9.6 ± 0.07	10.1 ± 0.14	8.9 ± 0.13	9.5 ± 0.22	95	94	93	94
$\frac{\text{Weight}}{\text{Crown heel length}}$ $(\frac{\text{Kg}}{\text{cm}})$	0.053±0.0005	0.060±0.0006	0.049±0.0009	0.051±0.0010	88	96	92	85

TABLE-75 (Contd.)

Parameters	Full term (FT)		Premature (PM)		LIG as % HIG		PM as % FT	
	LIG	HIG	LIG	HIG	FT	PM	FT	PM
	mean ± SE							
Crown rump length / Crown heel length	0.58 ± 0.002	0.58 ± 0.002	0.57 ± 0.004	0.59 ± 0.004	100	97	98	102
Shoulder to elbow length (cm)	10.0 ± 0.07	10.5 ± 0.08	9.5 ± 0.14	9.8 ± 0.15	95	97	95	93
Elbow to finger tip length (cm)	13.4 ± 0.07	14.3 ± 0.10	12.9 ± 0.14	13.9 ± 0.16	94	93	96	97
Total arm length (cm)	23.4 ± 0.12	24.8 ± 0.14	22.4 ± 0.24	23.7 ± 0.26	94	95	96	96
Hip to knee length (cm)	9.5 ± 0.07	9.7 ± 0.07	9.0 ± 0.14	9.0 ± 0.18	98	100	95	93
Knee to heel length (cm)	12.4 ± 0.08	13.5 ± 0.09	11.7 ± 0.18	13.0 ± 0.23	92	90	94	96
Total leg length (cm)	21.9 ± 0.11	23.2 ± 0.11	20.7 ± 0.24	22.0 ± 0.36	94	94	95	95
Foot length (cm)	8.2 ± 0.08	8.7 ± 0.05	7.7 ± 0.13	8.3 ± 0.12	94	93	94	95
Thigh circumference (cm)	14.1 ± 0.18	15.6 ± 0.14	13.1 ± 0.29	14.5 ± 0.38	90	90	93	93

@ Weight as given by Widdowson (1968).

TABLE-76 : Low birth weight infants as per cent of appropriate for gestational age infants in low income group.

Parameters	AGA	SGA	PM	Values as % AGA	
	Mean ± S.E.			SGA	PM
No. of subjects	121	26	43		
Birth weight (kg)	2.70 ± 0.02	2.06 ± 0.04	2.24 ± 0.06	76	83
Birth weight as % expected weight@	81 ± 0.85	63 ± 1.60	89 ± 1.89	78	110
Crown-heel length (cm)	49.2 ± 0.17	46.1 ± 0.42	46.0 ± 0.49	94	93
Crown-rump length (cm)	28.2 ± 0.14	26.9 ± 0.33	26.8 ± 0.44	95	95
Head circumference (cm)	32.8 ± 0.11	31.5 ± 0.23	31.3 ± 0.24	96	95
Chest circumference (cm)	31.1 ± 0.28	28.4 ± 0.34	28.9 ± 0.33	91	93
Abdomen circumference (cm)	29.7 ± 0.17	27.4 ± 0.41	28.1 ± 0.35	92	95
Mid-arm circumference (cm)	9.8 ± 0.07	8.7 ± 0.16	8.9 ± 0.13	89	91
$\frac{\text{Weight}}{\text{Crown-heel length}}$ $(\frac{KG}{Cm})$	0.055±0.0004	0.045±0.0007	0.049±0.0009	82	89
$\frac{\text{Crown rump length}}{\text{Crown heel length}}$	0.57 ± 0.002	0.58±0.006	0.57±0.004	102	100
Shoulder to elbow length (cm)	10.1 ± 0.07	9.4 ± 0.14	9.5 ± 0.14	93	94
Elbow to finger tip length (cm)	13.6 ± 0.07	12.8 ± 0.13	12.9 ± 0.14	94	95

TABLE-76 (Contd.)

Parameters	AGA	SGA	PM	Values as % AGA	
				SGA	PM
		Mean $\pm$ SE			
Total arm length (cm)	23.7 $\pm$ 0.12	22.3 $\pm$ 0.23	22.4 $\pm$ 0.24	94	95
Knee to hip length (cm)	9.7 $\pm$ 0.07	8.7 $\pm$ 0.14	9.0 $\pm$ 0.14	90	93
Knee to heel length (cm)	12.5 $\pm$ 0.08	11.8 $\pm$ 0.17	11.7 $\pm$ 0.18	94	94
Total leg length (cm)	22.2 $\pm$ 0.10	20.5 $\pm$ 0.23	20.7 $\pm$ 0.24	92	93
Foot length (cm)	8.3 $\pm$ 0.09	7.8 $\pm$ 0.15	7.7 $\pm$ 0.13	94	93
Thigh circumference (cm)	14.5 $\pm$ 0.18	13.0 $\pm$ 0.38	13.1 $\pm$ 0.29	90	90

@ Weight as given by Widdowson (1968)

AGA - Appropriate for gestational age

SGA - Small for gestational age

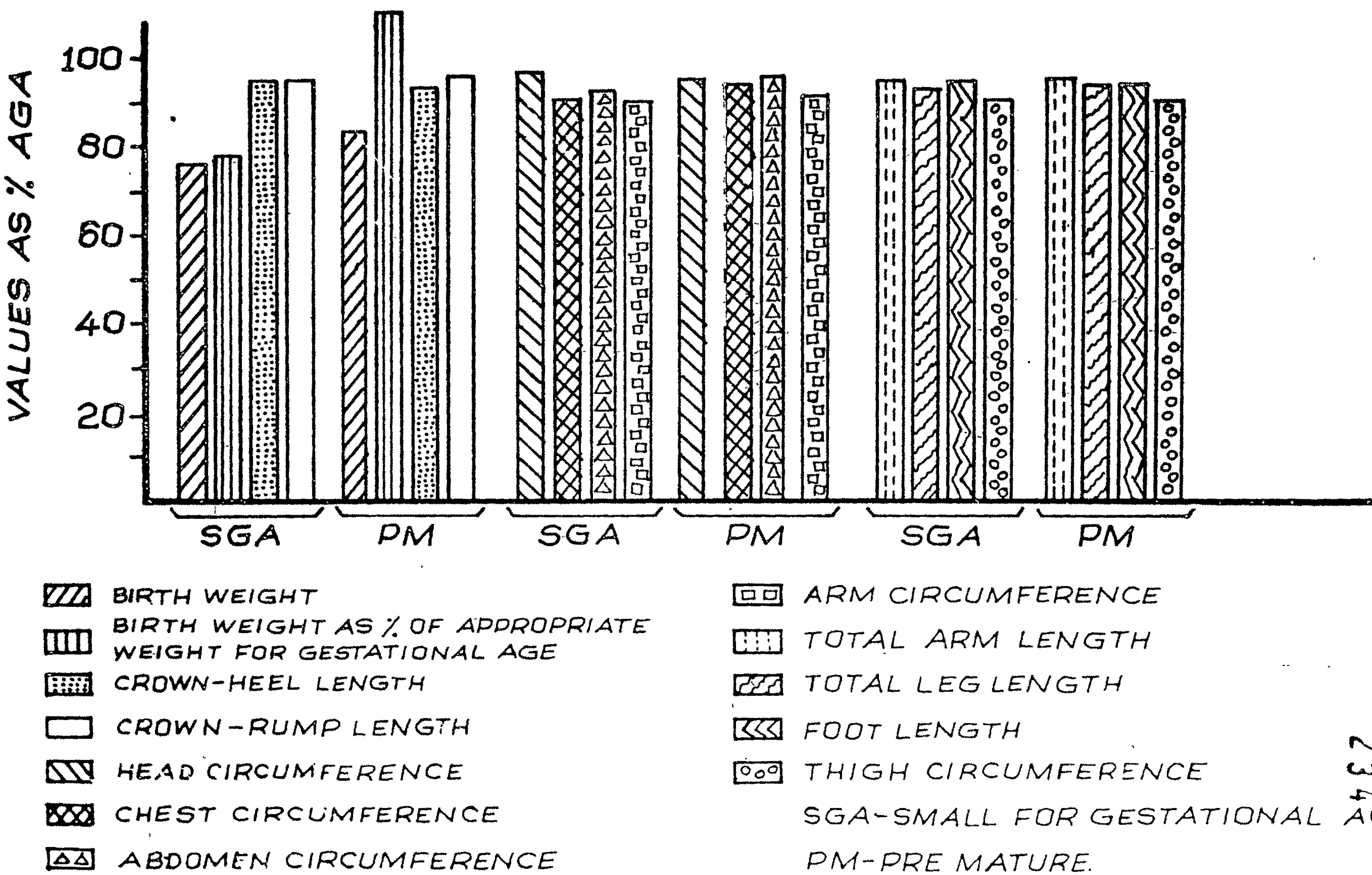
PM - Premature infants.

other parameters in small-for-gestational age and premature infants were smaller in comparison to appropriate for gestational age infants in low income group (Figure-16).

In this connection, it is well known that fetally growth retarded infants have normal subcutaneous fat for their body weight but lower as compared to their full term normal counterpart (Nut Rev., 1975, 1978). Moreover, weight is readily affected while height and head circumference are also affected with continued nutritional adversity (Stoch and Smythe, 1963). Even in chronic undernutrition, height is found to be less affected than weight where the height deficit is as low as 10% compared to the weight deficit which is 30% (Cravioto, 1966), and the effects of this in the later life are long lasting resulting in a stunted individual.

Among the premature infants two fairly distinct categories are seen, one with weights small-for-gestational age and the second with appropriate-weight for gestational age (Table-73). Infants in the later category are believed to exhibit some catch up or accelerated growth followed by a rate that parallels that of a full term infant, after a period of adjustment following birth (Jackson, 1968; Birch *et al*, 1970;

FIGURE: 16 SOMATIC MEASUREMENTS OF SMALL FOR GESTATIONAL AGE (SGA) AND PRE MATURE (PM) INFANTS EXPRESSED AS PERCENT APPROPRIATE FOR GESTATIONAL AGE (AGA) IN LOW INCOME GROUP.



Butler, 1972). However, longitudinal studies done in this laboratory suggest that in spite of their low weights they have a good potential for growth and development as judged by extensive anthropometric measurements and neuromotor development (Desai,1980; Savkur, 1980).

However, the very fact that such babies occur with more frequency among the poor where postnatal conditions may be unsatisfacoty may make the realisation of this potential difficult. Inadequate lactation in the mothers of such babies is associated with poor growth. The small size of the baby itself may contribute to inadequate lactation as lactation performance is influenced by many factors such as the vigour of the suckling reflex in the infant, and mother child bonding which in turn is influenced by the size and sex of the infant, vocalization and responsiveness of the mother and infant, physical contact, face to face interaction etc. These latter factors may also be influenced by the size of the infant (Cortiol and Lezine, 1974; Claire et al, 1975). In addition, the proportion of infants failing to achieve a satisfactory weight at the weaning age of six months is much higher in this group (Rajalakshmi and Ramakrishnan, 1969).

TABLE-77 : Ratios for different somatic measurements in premature and full term infants in low (LIG) and high (HIG) income groups.

Parameters		Weeks of gestation		
		∠ 36	36 - 38	> 38
		mean ± S.E.		
No. of subjects	LIG	43	84	64
	HIG	15	54	49
Birth weight / Head circumference	LIG	0.071	0.078	0.081
	HIG	0.079	0.088	0.091
Mid-arm circumference / Head circumference	LIG	0.28	0,29	0.29
	HIG	0.29	0.30	0.30
Chest circumference / Head circumference	LIG	0.92	0.94	0.94
	HIG	0.92	0.95	0.95
Chest-circumference / Abdomen circumference	LIG	1.03	1.06	1.04
	HIG	1.07	1.06	1,06
Mid-arm circumference / Chest circumference	LIG	0.31	0.31	0.31
	HIG	0.32	0.32	0.32
Head circumference / Abdomen circumference	LIG	1.11	1.12	1.10
	HIG	1.17	1.12	1.12

When ratios for different somatic measurements were calculated in newborns (Table-77), all ratios except weight/head and weight known heel length in low income group were comparable to the values for the high income group. These ratios did not change with the gestation.