To Our Readers

We are happy with the positive feedback and renewed interest displayed by the subscribers to our Update Series "Nurtition in Disease management'. The interest displayed by specialists in various disciplines of Medicine and Surgery has been particularly encouraging.

The current issue contains the latest concepts regarding nutritional support in hepatic failure. This article also provides simple guidelines for nutritional management from a practical standpoint, with protocols.

The Second article highlights the very important fact (aptly brought out in the article) that modern fast-paced life in the urban setting makes us pay a heavy price. It draws our attention to some interesting observation regarding breakfast eating patterns of Indian school children and their impact.

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Nutritional Support In Hepatic Failure

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Nutritional support (NS) in hepatic failure (HF) poses a major challenge. HF is a power failure. All nutrients administered either enterally or parenterally have to be metabolised eventually in the liver in order to produce nutritional repletion. The liver has multiple functions, many independent of one another. Laboratory tests cannot be used to predict a specific function of the liver.

This outline summarises the current concepts of HF and hepatic encephalopathy (HE) and provides practical guidelines on Nutrition Suuport (NS) in these conditions.

HF presents as

- acute liver injury (hepatitis, toxins) sometimes leading to fulminant HF,
- hepatic encephalopathy (HE), or
- chronic liver failure.

Mortality in fulminant HF is over 80 per cent without liver transplantation and a beneficial role for NS is unproven. However, NS should not be delayed if other therapeutic modalities are continued. Renal failure and hypoglycaemia require appropriate modifications in the formula. In chronic liver failure, empiric restriction of any specific nutrient, including protein and fat, is justified.

METABOLIC ALTERATIONS IN HF

HF is associated with changes in all substrates due to cytokine mediators and hormonal changes. Hormonal changes are:

- Hyperinsulinaemia due to decreased hepatic clearance (secondary to hepatic dysfunction or portosystemic shunting) or increased pancreatic synthesis. Sustained Hyperinsulinaemia results in peripheral insulin resistance, producing hyperglycaemia, which again stimulates production of insulin. Eventually beta cell exhaustion occurs.
- A disproportionate hyperglucagonaemia due to diminished hepatic clearance and increased pancreatic secretion, resulting in an elevated glucagon:insulin ratio.
- Decreased synthesis of insulin-like growth factor-1 (IGF-1), resulting in increased growth hormone. Low levels of IGF-1 cause protein catabolism and decreased glycogen synthesis. Alterations in substrate metabolism that occur as a result of the above are:
- **Carbohydrate**: Decreased hepatic and skeletal muscle glycogen synthesis; increased gluconeogenesis glucose intolerance and insulin resistance. Impaired glucose tolerance and insulin resistance is seen in a majority of cirrhotics, of whom 40 per cent are diabetic. However, in severe HF, hypoglycaemia occurs as a consequence of impaired gluconeogenesis, increased insulin levels and inability to metabolise glycogen.
- **Fat**: Increased lipolysis, but also increased reesterification. Fat metabolism in HF is a state of accelerated starvation resulting in depletion of fat stores and essential fatty acid deficiency1. Mild to moderate hypertriglyceridaemia can occur due to lipolysis and some decrease in hepatic lipoprotein lipase activity.
- **Protein:** While reports vary, generally protein synthesis is decreased. Protein catabolism is always increased in HF and cirrhosis. Peripheral release of aromatic aminoacids is increased and ammonia production is increased, contributing to hepatic encephalopathy (HE) (see below). Muscle utilisation of branched chain aminoacids (BCAA) increases with typically low plasma BCAA levels.

PATHOGENESIS OF HEPATIC ENCEPHALOPATHY

The Unified Theory of Fisher involving both ammonia and false neurotransmitters is the best explanation so far2.

- **Ammonia:** The majority of ammonia comes from glutamine oxidation by the small intestine; bacterial contribution is much smaller3. The main site of ammonia metabolism (to urea and back to glutamine) is the liver and secondarily the muscle. Due to HF and muscle wasting, ammonia metabolism is diminished and the excess crosses the blood brain barrier. There is no urea cycle in the brain; so it is metabolised by alternative routes. Ammonia combines with glutamate (in the presence of glutamine synthetase) to form glutamine. Glutamine causes cerebral edema4. Thus, glutamine-enriched enteral feeds should be avoided in HF.
- **False neurotransmitters:** AAA (phenylalanine, tyrosine and tryptophan) and their amines (phenylethylamine, octopamine, tryptamine) are produced in the gut during digestion and normally cleared by the liver. In HF, these monoamine 'false neurotransmitters' are shunted around the liver and reach the brain.

MALNUTRITION IN HEPATIC FAILURE

Protein-calorie malnutrition (PCM) is common in patients with chronic liver diseases. With a history of alcoholism, the incidence is 100 per cent5. PCM is an independent predictor of complications such as variceal bleeding. A majority of patients with HF already have PCM and there should be no delay in initiating NS.

NUTRITIONAL ASSESSMENT IN HEPATIC FAILURE

Standard anthropometric measurements are of no value, especially due to oedema. Short-term weight gain or loss is also of no clinical significance. Visceral protein markers are unreliable as the production of albumin and other proteins is diminished in HF. In the presence of renal failure or hepato-renal syndrome, the use of nitrogen balance also becomes unreliable. In short, there is no reliable marker or prognostic indicator of malnutrition in HF.

WHEN SHOULD NS BE INITIATED IN HF?

HF patients with pre-existing PCM (and often with insufficient oral intake after admission) are malnourished by definition, and appropriate nutritional intervention should be planned urgently. As in other critical care situations, some form of NS should be initiated at least within two days of admission, preferably after attempts at hemodynamic stabilisation and correction of major fluid and electrolyte imbalances. Early initiation of enteral nutrition (EN) prevents the onset of ileus.

WHAT ROUTE SHOULD BE USED?

The decision regarding the optimal route follows standard teaching: if the gut works use it! The advantages of enteral nutrition are well known. Intolerance to intragastric feeding is not a contraindication for enteral feeding as nasojejunal tubes can be easily positioned with endoscopy or under fluoroscopy.

A combination of routes are often used: enteral (or oral) in combination with parenteral (central vein or peripheral vein).

Target Requirements for NS in Liver Failure

Guidelines for NS in HF were recently provided by the European Society for Parenteral and Enteral Nutrition (ESPEN)7. Unwarranted iatrogenic protein and fat restriction, common in India, is often a cause of morbidity and mortality in HF. Requirements below are given as units per kg of ideal body weight (IBW).

Protein

Contrary to previous teaching, protein requirement in HF is actually increased8,9,10. There have been no controlled studies on protein restriction in HF. Mental status often improves with standard tube feeding11,12. Lactulose and neomycin can be continued with enteral feeding. Neomycin actually lowers ammonia production by reducing mucosal glutaminase activity (and thus reducing ammonia production from glutamine) rather than by its antibacterial action13.

In grade 1-2 HE: protein is initiated at 0.5-0.6 g/kg/day and increased rapidly by 0.25 g/kg/day until the target is reached or HE worsens significantly. It is reasonable to reach a target of 1-1.5 g/kg/day even in HE (ACCP and ESPEN guidelines strongly support this recommendation).

In the protein intolerant patient, the first step is to rule out precipitating events such as sepsis or gastrointestinal bleeding before arbitrarily decreasing protein intake. If none are found, rather than decreasing protein intake, a portion of the intake is substituted by branched chain aminoacid (BCAA)-enriched enteral or parenteral products. These contain 40-45 per cent BCAA compared to 20-25 per cent BCAA in standard preparations.

At these concentrations, 0.5 to 1.2 g/kg/d of proteins can be provided as BCAA. The BCAAs - leucine, isoleucine and valine - are essential aminoacids. Only if the condition worsens after 48 hours of use of BCAA should protein intake be reduced. Specialised products for use in HF also have decreased content of aromatic and sulfour-containing amino acids.

In grade 4-5 HE: Initiate protein at doses mentioned above but use BCAA-enriched formulas. The target is 1.3 g/kg/d. Many patients with HE improve by one grade within 48-72 hours after initiation of BCAA-enriched formulas. Other advantages include normalisation of plasma aminoacid levels, increased protein synthesis and improvement in nitrogen balance. There is no clear data that BCAA improves survival, but there is definitely no deleterious effect. In view of the increased cost, we recommend that its use should be limited to 48-72 hours in the absence of improvement.

The use of specialised HF products in situations other than isolated HF, for example, multiple organ system failure in the absence of cirrhosis, is not recommended.

Calories

Calorie requirements approximate 25-30 non-protein kcal/kg IBW/d. The Harris-Benedict equation and stress factor corrections are unreliable but is the best clinical tool available as indirect calorimetry is not available easily. Sixty-five to 50 per cent of non-protein calories are provided as carbohydrates and 35 to 50 per cent as fat.

In PN only glucose should be used as the carbohydrate source.

The administration of intravenous lipids is safe; in humans, the lipoprotein lipase activity in capillary endothelium is more important that in the liver. A moderate fat load (<1 g/kg/d) is adequately cleared14.

Combinations of medium chain triglycerides (MCT) and long chain triglycerides (LCT) have some advantages over the usual LCT preparations. Omega-3 enriched fat emulsions have been shown to be advantageous by their positive effects on immunomodulation.

In EN, as fat malabsorption is common in HF, providing fat as MCT oil decreases steatorrhea.

Electrolytes

Mild Na restriction (to 120 mEq per d) is recommended. (Note: 1 g of elemental Na contains 43.5 mEq of Na; 1 g of NaCl contains 17.5 mEq of Na). Adequate K, Mg and P should also be provided.

KCl should not be bolused through enteral feeding tubes as it causes diarrhoea due to high osmolality.

MgS04. 7H20 (Epsom salt) can be added to enteral feeding in 5 g doses (1 g MgS04. 7H20 contains 8 mEq or 4 mMoles of Mg). The parenteral requirement for Mg is 15 mEq/d.

P is not locally manufactured for IV use. Physicians involved in critical care should arrange to procure this lifesaving product from other sources. The daily requirement of P is 10-15 mMol/1000 carbohydrate calories. Enterally, P can be replenished by using certain 'tonics' containing glycerophosphates.

Vitamins

Deficiencies of water-soluble vitamins are common, especially in alcoholics. Fat-soluble vitamin deficiencies occur in several patients with liver diseases. Many multivitamin preparations have inadequate levels or absence of vitamin K, folic acid and biotin. Standard doses should be given enterally or parenterally.

Vitamin K is given as 10 mg doses parenterally, initially daily and later every alternate day. There is no storage form of vitamin K in the body.

Thiamine deficiency is especially common in HF and produces lactic acidosis due to inadequate conversion of lactate to pyruvate. Acute thiamine deficiency can occur especially when a high carbohydrate intake is provided; wet beri beri manifesting as congestive heart failure can occur15. A dose of 50 mg/d is recommended, parenterally for three days and then enterally.

Trace Elements

Zinc (Zn) deficiency is especially common in HF16 and may contribute to HE. Supplements are given either enterally or parenterally. Various commercial preparations are available for enteral use (for example, Zn sulfate, 600 mg/d) and absorption from the proximal bowel is generally good. Zn requirement in PN is 5 mg/d; doses of up to 20 mg/d are safe.

Selenium (Se) deficiency causing cardiomyopathy and myositis has also been described in HF.

Copper and manganese are excreted in the bile and their addition to PN is avoided in HF. Appropriate multiple trace element additives should therefore be used.

Prevention of Refeeding Syndrome

With the ready availability of various parenteral and enteral products, physicians are sometimes over-aggressive. Too much nutrition is sometimes lethal! Mechanisms are ill defined but include decreased serum levels of P, Mg and K and micronutrient deficiencies. The refeeding syndrome is common in patients with preexisting PCM, common in HF. The greater the degree of PCM, and the longer the period of inadequate oral intake, the more severe are the manifestations of the refeeding syndrome.

One avoids this serious complication by reaching the target requirements over a period of several days.

Initially only 15 to 20 non-protein calories/kg/d (and correspondingly lower protein intake with a non-protein calorie: nitrogen ratio of approximately 100:1) are provided. Serum levels of Na, K, Mg, P and glucose are carefully monitored and the calorie intake is increased by not more than 20 per cent each day. Adequate micronutrients (trace elements and vitamins) are provided.

Monitoring

In addition to the routine monitoring (glucose, electrolytes, P and Mg) hepatic and renal parameters are also checked periodically. Prothrombin time is a good prognostic indicator of hepatic function. Serum triglycerides should also be monitored; levels less than 350 mg/dl (3.95 mmol/L) are acceptable18. The addition of 4,000-5,000 units of heparin per day to parenteral nutrition (PN) admixtures will stimulate lipoprotein lipase activity and decrease serum triglyceride levels.

CONCLUSION

Providing nutritional support to patients with hepatic failure is challenging, in terms of assessment, fine-tuning requirements, monitoring and trouble-shooting. Unnecessary restriction of protein and fat should be avoided. The need for electrolytes (including Mg and P) and micronutrients should not be forgotten. While 'liver failure is power failure', the clinician can attempt to replenish this power with the products and technology available.

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REMINDER

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Breakfast Eating Patterns Of School Children And Their Impact On Nutritional Status

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INTRODUCTION

'You are what you eat' is an ancient saying that motivates health professionals to be concerned with what people eat especially at the start of the day. A nutritionally adequate breakfast, therefore, is considered important for achieving and maintaining physical and mental health. This is a fact borne out and based on several controlled studies that have been carried out to determine the effect of different breakfast habits on the physiological responses, attitudes and scholastic achievements of subjects under study1-3 .

The New Harvard Research School Breakfast Programme focussed on breakfast eating habits of children and found that this meal gives children a good start in terms of energy and performance in various tasks. Thus, ensuring that children eat a nutritious breakfast every day is one of the most important contributions that parents can make to their children's health, well-being and success.

A link between hunger and a large number of behavioural problems exhibited by children such as fighting, stealing, indiscipline, having problems with teachers, and so on has been established4

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NUTRITIONAL SIGNIFICANCE OF BREAKFAST

The nutritional significance of breakfast can hardly be undermined. Eating after a night's long rest ensures that the blood glucose levels are maintained through the activity period of the morning and till the next meal. Since the brain derives its energy only from glucose, an interruption in the supply can lead to

lethargy, muscle weakness and often fatigue and exhaustion. The hunger sensation is, therefore, triggered off when glucose levels fall below 70 mg per cent. When the levels fall below 65 mg per cent, a craving for sweets so common among children is noticed. When blood glucose levels fall continuously thinking gets confused and reactions become slow often leading to accidents5. A good breakfast is therefore vital for providing a continuous source of energy for growth, play and work.

BREAKFAST AND GROWTH

Skipping breakfast may hinder growth of children, because the body is forced to call upon body stores of protein for meeting energy requirements.

Skipping breakfast has become the norm in modern India because of lifestyle changes in family life, and when this happens largely among children, it can result in sub optimal growth and development - a factor important to the future human resource development of the country.

BREAKFAST HABITS OF CHILDREN

Studies have reported that millions of children attend school on an empty stomach7,8. In a Connecticut study, it was found that over half the children in grades five to eight missed breakfast9. The well known Iowa breakfast studies suggested that half to two-third of school children had poor breakfasts10.

Most of the research, however, has been reported from the developed countries. Only one Indian study has reported that 38 per cent of children from government schools came without breakfast, 44 per cent with only milk and only 18 per cent had cereal (chappati) with tea11.

A study of breakfast eating patterns of nine to 10 year olds was therefore conducted on 220 Central School children of West Delhi, and their impact on growth was studied using dietary recalls and suitable anthropometric measurements.

METHODS

- The breakfast eating habits of subjects were determined through questionnaires designed for children and their parents.
- 24-hour dietary recalls were used to assess dietary intake on any one school day. The nutritional contribution of their diets were then calculated using the food composition tables12, and the nutritional adequacy ratio calculated.
- Anthropometric measurements such as weight, height, MUAC and triceps skinfold, were used to assess their current nutritional status.

The results obtained were then correlated with the breakfast eating habits of the children to determine if missing breakfast had any impact on their growth and development.

RESULTS

The data revealed that only 50 per cent of the children ate breakfast regularly every day. Of the other 50 per cent, two-thirds ate breakfast three days a week while one-third skipped it altogether. The reasons for skipping breakfast were listed as:

- waking up late
- not hungry
- dislike the food offered
- busy doing homework
- no one to eat with
- feel sick.

A larger number of boys gave the first two reasons and none of them reported that they were busy doing homework, while more girls gave the last five reasons for missing breakfast.

Of the breakfast eaters, however, 37 per cent said that they enjoy the meal and overall gave the following reasons for eating it.

- most important meal
- prevented head and stomach aches
- to gain weight
- habit.

While 46.7 per cent subjects considered it to be the most important meal, 33 per cent stated the second reason while only 10 per cent ate breakfast to gain weight.

The perceptions of the subjects' parents with respect to breakfast eating were also recorded and it was found that:

- 48 per cent believed that eating this meal leads to better thinking and work efficiency
- 23 per cent said it kept the child active throughout the day
- 19 per cent reported no complaints about head and stomach aches
- 10 per cent felt that it was the most nutritious meal of the day.

CONSUMPTION PATTERNS

The foods consumed varied from bread-based items such as sandwiches, toast, burgers to dalia, cornflakes, parantha, milk, coffee, fruits and juices. It was noted that 65 per cent children never took fruit, and 75-80 per cent consumed sandwiches or burgers and coffee more often. Only 7-8 per cent ate parantha with plain milk. The frequency of consumption is indicated in Figure I.

Children's food preferences as reported by mothers indicated that 35 per cent preferred noodles, 32 per cent burger and sandwich, 19 per cent burgers and 14 per cent french fries.

NUTRITIONAL INTAKE

The mean nutrient intake of Breakfast Eaters (BE) and Breakfast Skippers (BS) diets from 24-hour recalls were calculated using food composition tables. The values are presented in Tables I and II, which indicate that they were inadequate in all respects when compared to the recommended values of nutrients for nine to 10 year olds. The BE group showed deficient intakes of iron, b-carotene and niacin whereas the BS subjects had distinctly lower values than breakfast eaters. Similar results have been reported by researchers from developed countries13-15. The mean energy intake of BS was significantly lower than BE (t = 5.86). The values indicated a deficit of 650-750 kcal as against that for BS which was 650-900 kcals per day. Similar findings have been reported in the literature16,17. The trend was the same for other nutrients although in the case of b-carotene and iron, both the eaters and skippers were found short of the recommended values for their age. This was attributed to the low intake of green leafy vegetables in the day's diet and the low bioavailability of iron from cereal based diets. Except for B-vitamins and vitamin C the diets of breakfast skippers fell short of the RDA for all nutrients in contrast to those of breakfast eaters.

Table I: Comparison of mean daily nutrient intake of 9-10-year-old boys and girls (BE)

Nutrients	9 years			10 years			
	RDA*	Boys	Girls	RDA*	Boys	RDA	Girls
Energy (kcal)	1950	1979	1642	2190	1970	1970	1789
Protein (g)	41	58	52.7	54	57	57	56
Calcium (mg)	400	757	751.3	600	764	600	676
Iron (mg)	26	13.7	12.5	34	12	19	11
b-carotene (mg)	2400	489.5	263.3	2400	935	2400	538
Vitamin C (mg)	40	68.5	98.9	40	66	40	46
Thiamin (mg)	1.0	1.4	1.4	1.2	1.3	1.0	1.1
Riboflavin (mg)	1.2	1.3	1.1	1.5	1.4	1.2	1.2
Niacin (mg)	13	11.2	10.4	16	10.9	13	8.2
*RDA as suggested by ICMR (1990)							

Table II: Comparison of mean daily nutrient intake of 9-10-year-old boys and girls (BS)

Nutrients	9 years			10 years			
	RDA*	Boys	Girls	RDA*	Boys	RDA	Girls
Energy (kcal)	1950	1350	1217	2190	1286	1970	1204
Protein (g)	41	38.8	38.3	54	30.9	57	30.7
Calcium (mg)	400	36.7	416	600	351.6	600	360.2
Iron (mg)	26	9.4	10	34	7.3	19	8.3
b-carotene (mg)	2400	215	184.6	2400	345	2400	218.3
Vitamin C (mg)	40	34	25.8	40	15.2	40	18.7
Thiamin (mg)	1.0	1.0	0.99	1.2	1	1.0	0.93
Riboflavin (mg)	1.2	0.86	0.69	1.5	0.78	1.2	0.83
Niacin (mg)	13	8.2	8.11	16	9.09	13	7.29
*RDA as suggested by ICMR (1990)							

An NAR value of 0.66 and above reflects adequate dietary intake of a particular nutrient. The NAR was calculated for the nutrients in the diets of subjects and the values obtained are presented in Table III.

Table III: NAR values of diets of subjects

	NAR					
Nutrients	9 years		10-year boys		10-year girls	
-	BE	BS	BE	BS	BE	BS
Energy (kcal)	0.93	0.65	0.90	0.59	0.91	0.6
Protein (g)	1.33	0.62	1.06	0.57	0.97	0.54
Calcium (mg)	1.36	0.63	1.27	0.59	1.13	0.60
Iron (mg)	0.49	0.38	0.38	0.22	0.58	0.44
b-Carotene (m-g)	0.81	0.33	1.56	0.58	0.90	0.36
Vitamin C (mg)	0.97	0.42	1.66	0.38	1.17	0.47
Thiamin (mg)	1.04	1.01	1.21	0.91	1.07	0.93
Riboflavin (mg)	0.98	0.65	1.06	0.60	0.99	0.69
Niacin (mg)	0.81	0.63	0.73	0.61	0.63	0.56

The values indicate that the BE could meet the NAR for all nutrients except iron and b-carotene while the BS showed values which were significantly lower for all essential nutrients, energy and protein reflecting inadequate nutrient intake.

NUTRIENTS FROM BREAKFAST

The nutrient composition of breakfasts eaten by the subjects (BE) was calculated. The mean nutrient values for breakfast alone are presented in Table IV, which indicates that eaters meet one-fourth to one-third their total daily energy and protein requirements.

The nine-year-olds consumed more protein and fat than the 10-year-olds. The difference being statistically significant (t = 4.92). The breakfast contributed 55 to 57 per cent of the energy from carbohydrates, 12-13 per cent from protein as against the 20 per cent recommended, and 28 to 33 per cent from fat in the two age groups. The Iowa breakfast studies defined a basic breakfast as one that provides one-fourth the total daily requirement for energy and protein.

Table IV: Mean breakfast intake of the subjects

Nutrients	Boys		Girls	
-	9 Years	10 years	9 years	10 years
Calories (kcal)	677	730	722	581
Protein (g)	22	21.9	21.7	18.6
Fat (g)	21.4	27.5	22.5	19.6
CHO (g)	100.1	100.8	108.4	83.1

ANTHROPOMETRY

Growth and physical development of children are widely used as indicators of overall health and nutritional status. Anthropometric measurements of the subjects were therefore recorded with respect to weight, height, MUAC and triceps skinfold. The mean values of BE and BS are presented in Table V.

Table V: Anthropometric measurements of subjects

Age(yrs)	Height(cm)		Weight(kg)		MUAC(cm)		Triceps(mm)	
-	BE	BS	BE	BS	BE	BS	BE	BS
9+Boys	133.8 ±4.2	132.1 ±7.9	30.7 ±7.83	26.6 ±4.47	19.73 ±3.52	18.44 ±1.51	12.1 ±7.86	8.89 ±2.17
9+Girls	134.5 ±4.4	128.7 ±3.21	31 ±4.93	25.7 ±3.59	20.1 ±2.27	17.7 ±1.21	10.4 ±1.98	8.8 ±1.4
10+ Boys	137.8 ±4.8	135.3 ±3.2	30.8 ±2.87	26.9 ±2.13	19.9 ±1.12	18.9 ±1.88	11.38 ±2.16	9.4 ±1.84
10+Girls	134.4 ±5.2	135.6 ±4.25	30.4 ±2.6	29.4 ±3.85	20.0 ±1.93	18.8 ±1.28	9.74 ±1.96	9.6 ±1.15

Anthropometric data revealed that the mean weight and height of breakfast eaters did not show any significant difference between the sexes although between the ages the difference was highly significant in response to the t-test (t = 3.23). When compared by percentiles of NCHS standards for weight for age, 60 per cent BE were above the 50th percentile and a minority of BS (13 per cent) were above 50th percentile. The low weight of BS may be partly attributed to their low energy intake and deficiency of essential nutrients, which were otherwise provided through the breakfast. A positive correlation was found between energy and weight (r=0.493) and also protein and weight (r= 0.429) in case of breakfast eaters.

When compared by percentiles of NCHS standards for height for age, 46.7 per cent were above the 50th percentiles. Only 36.7 per cent BS, on the other hand, had height for age above the 50th percentile. Lower height gain in Indian children may partly be due to genetic factors and partly to the fact that their diets are predominantly cereal-based and rich in phytates leading to poor bioavailability of calcium from them.

Weight for height is a sensitive index of current nutritional status of children and is independent of age13-16. When comparison was made with percentiles of NCHS standard only two girls and two boys exceeded the 95th percentile even among the BE, whereas only one girl and no boys reached the 90th percentile among the skippers.

The relationship between weight at birth to present weight was found to be significant. It was, however, assumed that the BE subjects were of normal weight while BS group birth weights, were recorded from parents through questionnaires. Although all the subjects showed incremental growth patterns and were born with normal birth weights the BS subjects failed to reach the desirable weight gain. This can partly be attributed to omission of breakfast. The data tends to establish the importance of breakfast as an important meal, as the nutrients obtained through it are rarely compensated by any other meal of the day.

The MUAC measurements were statistically significant when BE and BS subjects were compared (t = 2.96). The mean MUAC of BS was lower than the reference mean values (Jelliffe), but there was no significant difference between the sexes. However, a positive correlation was found between weight and MUAC for both BE (r = o.899) and BS (r = 0.776). The mean TSF of BE was 10.9 mm as compared to 9.0 mm for skippers. There was a significant difference between BE and BS subjects (t = 2.06), especially among the boys.

BREAKFAST AND HUNGER

About half the breakfast skippers experienced hunger on reaching school which varied in intensity from moderate to severe against only 25 per cent of breakfast eaters who felt the same. A comparison is shown in Figure II.

BREAKFAST AND SCHOOL PERFORMANCE

School performance was judged by using the attendance, class test performance and participation in extra-curricular activities of the subjects as indicators.

More than 50 per cent of breakfast eaters (BE) reported that they never missed school as against 23 per cent breakfast skippers. The skippers (76.7 per cent) reported missing school frequently due to sickness (60.9 per cent), fear of test (8.7 per cent) and did not feel like going to school (17.4 per cent) in contrast to the percentages for BE which were 46.7 per cent, 0.0 and 26.9 per cent, respectively.

BE (80 per cent) did not miss class tests whereas only 33 per cent skippers appeared regularly. BE (56.7 per cent) scored good marks as compared 36.7 per cent skippers who showed poor performance. These findings were confirmed by verifying their performances in the previous annual exams in which more than half the BE scored 75 per cent and above as compared to 37.7 per cent skippers who scored less than 60 per cent.

Results of participation in extra-curricular activities showed 65 per cent eaters and 54 per cent skippers took active part in extra-curricular activities. BE performed higher in sports (69 per cent) as compared to skippers (34 per cent).

CONCLUSIONS

1. The study revealed that over half the subjects skipped breakfast frequently or altogether, the main reason being getting up late indicating better attention to time management, both by parents and children, was needed avoiding late nights on school days.

2. Omission of breakfast is one of the major contributing factors leading to inadequate nutrient intake during the growth period of the subjects. This deficiency cannot be made up through other meals of the day.

3. Breakfast skipping has shown a relationship to varied behaviour in children as well as in their hunger patterns. These result in eating whatever snacks are available in school when they are hungry.

4. Hungry children suffered from head and stomach aches frequently, also affecting their concentration in class.

5. Anthropometric measurements revealed that skippers had a lower growth profile than breakfast eaters. The growth rate of eaters corresponded to the growth pattern of their counterparts in developed countries.

6. Skipping breakfast can affect physical and mental development in children.

The study also confirms the importance of breakfast to overall dietary quality and adequacy especially for school children and the need for parents to adjust their lifestyles to ensure that every child eats a good breakfast before going to school.

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