SOME NUTRITIONAL AND PSYCHOLOGICAL ASPECTS OF FAILURE TO THRIVE

Thesis
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To my dear parents

To my lovely wife

To my precious son

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LIST OF ABBREVIATIONS

ADHD	Attention deficit hyperactivity disorder
AN	Anorexia nervosa
AVP	Arginine vasopressin
BDLS	Brachmann-de-Lange syndrome
BMI	Body mass index
CAS	Child assessment schedule
CHD	Congenital heart disease
CHO	Carbohydrate
CIIP	Chronic idiopathic intestinal pseudo-obstruction
CP	Cerebral palsy
DI	Diabetes insipidus
DSM-IIIR	Statistical Manual of Mental disorders
e.g.	Example
EPDS	Edinburgh Postnatal Depression Scale
FTT	Failure to thrive
GH	Growth hormone
GHRH	Growth hormone releasing hormone
GQ	Growth quotient
HAP	Height / Age percentile
HAM	Height / Age median
HAZ	Height / Age z-score
Hb	Hemoglobin
Hct	Hematocrite
Ht	Height
IQ	Intelligence quotient
Kcal	Kilocalorie
Kg	Kilogram
KWO	Kwashiorkor
MAC	Midarm circumference
MSBP	Munchausen syndrome by proxy
NCHS	National Center for Health Statistics
NI	Nutritional index
NOFTT	Nonorganic failure to thrive
NREM	Non-rapid-eye-movement
OFTT	Organic failure to thrive

OSAS	Obstructive sleep apnea syndrome
PCC	Primary care clinic
PEM	Protein energy malnutrition
RADI	Reactive attachment disorder of infancy
RBCs	Red blood corpuscles
SD	Standard deviation
SEE	Sleep energy expenditure
SRIF	Somatotrophin releasing inhibitory factor
TSF	Triceps skin fold
UNICEF	United Nations International Children Emergency Fund
WAM	Weight / Age median
WAP	Weight / Age percentile
WAZ	Weight / Age z-score
WBCs	White blood corpuscles
WHM	Weight / Height median
WHO	World Health Organization
WHP	Weight / Height percentile
WHZ	Weight / Height z-score
WSIS	Whiplash shaken infant syndrome
Wt	Weight

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INTRODUCTION

Failure to thrive (FTT) describes a failure of expected growth (usually weight). It occurs most often in infants but also is observed later in childhood. Initial recognition of this problem is usually based on relating growth (weight, height and head circumference) to appropriate anthropometric charts and noting those below the fifth percentile, or those who have deviated significantly from a previously acceptable pattern of growth ***(Accordo, 1982).***

Traditionally, the causes of FTT were classified as organic failure to thrive (OFTT) or nonorganic failure to thrive (NOFTT). OFTT accounting for 30% of the total, was ascribed to a major illness or system dysfunction thought to be sufficient to account for growth failure. These may include neurological, gastrointestinal, endocrinal, pulmonary, renal, chronic infections, chromosomal abnormalities and congenital syndromes due to maternal drug intake and infections. On the other hand, NOFTT was attributed to maternal deprivation. Emotional deprivation and neglect or abuse including the withholding of food, are commonly associated with this condition ***(Rathbun & Peterson, 1987).***

Theoretic advances in pediatrics and child development are pointing that all cases of NOFTT, and in many cases of OFTT, the primary biologic common factor is malnutrition. Stresses and disturbed parent-child relationship, create the context in which malnutrition leading to failure to thrive occurs, and exacerbate the developmental and behavioral status of the child ***(Frank & Zeisel, 1988).***

As the child progresses from complete dependence to independence, he needs a stable and secure family background, with a consistent pattern of emotional warmth, acceptance, help, and constructive discipline. Prolonged separation from or loss of parents can have a profound effect on psychological development in infancy and childhood. Poor relationships in the family may have similar adverse effects ***(Graham & Rutter, 1968).***

Therefore, evaluation of a child with FTT needs to take into consideration, nutritional, medical, psychological and developmental factors. By managing these factors simultaneously in the child with FTT, the vicious circle of psychological, nutritional and developmental disturbances will be circumvented and abolished ***(Rathbun and Peterson, 1987).***

Aim of Work

The aim of this work is to:

1. Identify the children with FTT.
2. Evaluate the different etiological factors of FTT such as nutritional, medical, psychological and developmental factors.
3. Assess the behavioral and psychological affection children with FTT.
4. Evaluate the maternal depression in children with FTT.
5. Investigate the relationship between maternal depression and child behavioral affection in cases of FTT.

Review of Literature

GROWTH AND DEVELOPMENT

Definition of growth

Growth is a term used to describe the process of growing, the increase in size and development of a living organism, from a simple to a more complex form, or from its earliest stage of being to maturity.Growth can be accomplished by an increase in the number of cells (hyperplasia), by an increase in the size of cells (hypertrophy), or by an increase in the amount of intercellular material (accretion). Growth is not simply a uniform process of becoming taller and larger, however, it involves changes in shape and body composition, and may involve replacement of tissues (the ductus arteriosus), tissue substitution (cartilage/bone), and alteration of specific tissues (puberty) ***(Bower, 1977).***

Growth is a dynamic process; not all tissues of body grow at the same rate, nor stop growing simultaneously. Some tissues continue to grow throughout our lifetime, while others reach full development early and remain static thereafter ***(Abbassy et al., 1972).***

Factors affecting growth

The harmony of growth lies in the intimate orchestration of growth process, so that each tissue is found in the right amount at the right time, and that differentiation of each tissue is accomplished as needed, so that each function of the growing organ can be served ***(Walker & Hendricks,1985).***

(1) Local growth stimulation and inhibition

Specific humoral substances in particular tissues have been described to play a role in cell division and differentiation. A specific humoral protein within liver cells seems to be responsible for regulation of mitosis occuring during repair of liver tissues. When the optimal number of liver cells is present, a substance termed "chalone" accumulates to inhibit further mitotic growth. These tissue specific substances act to keep the liver at its optimal size for the organism. Similar feed back systems to regulate growth of spleen and kidney have been suggested ***(Bower, 1977).***

Some tissue specific factors have been also described to enhance growth. Somatomedins, produced in the liver and other tissues primarily under stimulation of human growth hormone (GH), promote cellular proliferation and accelerate cartilagenous growth. Other factors promote growth of the nerve cells (nerve growth factor) and epiphyseal cells (epiphyseal growth factor) and fibroblasts (fibroblast growth factor) ***(Rosenblith & Sims-Knight, 1985).***

(2) Genetic factors affecting growth

The growth of a child is the result of complex interactions of genetic and environmental factors.Certain persons are taller than others, largely the consequence of multiple genetic differences, although nutrition may play some role as well. The pigmies of the Congo, one of the world's smallest races, appear to have a genetically determined inability to respond to somatomedins. Black children tend to be large and more advanced in skeletal

maturation than white children. Asiatic children tend to be smaller than black or white children ***(Collins & Gelehrter, 1990).***

(3) Neural control of growth

The hypothalamus has been suggested to be responsible for keeping the child in his or her genetically determined growth curve. If a child deviates from a growth pattern for a period of time because of malnutrition or illness, a period of accelerated or "catch up" growth brings him back to the predetermined curve. Peripheral nerves may also play some part in control of growth. Many structures, including muscles, nails and taste buds undergo atrophy when denervated ***(Berwick et al., 1982)***

(4) Hormonal influence on growth

Most of the endocrine glands influence growth in some way, but some have greater influence than others. Human GH produced by the anterior pituitary gland is necessary for normal growth throughout infancy and childhood. GH promotes synthesis of fat and carbohydrate, and is necessary for the proliferation of cartilage, leading to bone growth. As such, it is a major determinent of height achievement ***(Philips, 1992).***

GH as most other hormones is secreted in an intermittent pulsatile pattern, its synthesis and release from the pituitary somatotrophs are under the regulation of GH-releasing hormone (GHRH) and somatostatin (SRIF, somatotropin release inhibitory factor). GHRH promotes synthesis and

release of GH from anterior pituitary somatotrophs, SRIF inhibits GH release from somatotrophs ***(Schwartz & Bercu, 1992).***

In addition GH depends on numbers of factors, including nutritional status, sleep stage, stress and exercise together with a number of neurogenic, metabolic and hormonal influences ***(Thorner et al., 1992).***

Insulin appears to be a major growth regulating hormone and together with somatomedins influences the complex growth of the fetus ***(Arky, 1984).***

Thyroid hormone is essential for normal growth and development of the skeleton and the central nervous system, and is essential for complete expression of human growth hormone's effect on cartilage and bone formation ***(Rezvani & DiGeorge, 1977).***

Testosterone and other androgenic hormones mediate the growth spurt of puberty, by increase in muscle mass, acceleration of bone growth, maturation of male sexual organs, and appearance of male secondary sexual characteristics ***(Marvin & Rallison,1986).***

Table (1)
Factors influencing normal GH secretion
(Thorner et al., 1992)

Factor	++GH secretion	- -GH secretion
Neurogenic	.Stages III and IV sleep	.REM sleep
	.Stress	.Emotional deprivation
	.Alpha-adrenergic agonist	.Alpha-adrenergic antagonist
	.Beta-adrenergic agonist	.Beta-adrenergic antagonist
	.Acetylcholine agonist	Acetylcholine antagonist
Metabolic	.Hypoglycemia	.Hyperglycemia
	.Falling fatty acid level	.Rising fatty acid level
	.Amino acids	.Obesity
	.Uncontrolled diabetes mellitus	
	.Uraemia	
	.Hepatic cirrhosis	
Hormonal	.GHRH	.Somatostatin
	.Estrogens	.Hypothyroidism
	.Glucagon	.High glucocorticoid levels

(5) Nutrition in growth.

Adequate food to provide substrate for energy and synthesis of protein is essential for normal growth. In the diet suitable for normal growth, there must be an adequate supply of protein and appropriate amounts of amino acids. Absence of any of the essential amino acids will result in disordered or stunted growth. Nutritional factors are the most important and most common factors affecting growth in Egypt ***(Abbassy et al., 1972).***

Specific nutritional deficiencies may also interfere with growth. A lack of Zinc, a constituent of many enzymes involved in protein metabolism, has been associated with growth failure. Formation of bone requires an adequate supply of calcium, phosphorus and trace amounts of magnesium and manganese. Iron is required for hemoglobin, iodine is needed for thyroxine formation and fluoride for proper formation of tooth enamel and bone. Vitamin C deficiency results in deficient formation of intercellular substance blood vessels leading to fragility. Vitamin D deficiency results in inadequate calcification of the bones, leading to rickets ***(Walker & Hendricks, 1985).***

(6) Miscellaneous factors affecting growth.

Oxygen is required for optimal growth. Children with cyanotic heart disease may experience interference with growth until lesion is corrected ***(Braden & Strong, 1990).***

The lower the socioeconomic status of the mother, the smaller the baby and child. Growth in height of children is faster in spring than in autumn, whereas growth in weight is faster in autumn ***(Alleyne, 1981).***

Generalized diseases, such as tuberculosis or kidney disease, may affect growth in about the same manner as malnutrition. This is due to the associated anorexia, malabsorption and increased catabolism ***(Lowerey, 1973).***

GROWTH ASSESSMENT

The growth of an individual can be assessed by dietary history, clinical examination, anthropometric assessment and biochemical investigations. The clinical and biochemical methods require highly skilled personnel or techniques, while anthropometry requires only simple instruments, so it is the most practical. The anthropometric measures selected should be the simplest, quickest to take and the easiest to reproduce in order to give the maximal information concerning the growth state ***(Radheshyam et al., 1985).***

Basic measurements recommended for children were those made to assess :

1. Body mass, as judged by weight (Wt).
2. Linear dimensions, especially height (Ht).

3. Body composition and reserve of calories and protein, as judjed by subcutaneous fat and muscles ***(Jelliffe,1969).***

Weight (Wt)

Body weight is surely the commonest and best measure made of human being. The child must be weighed naked or with minimal clothes, and not after meals. Theoretically, the bladder should be emptied prior to measurements. In fact, this is rarely done and has little practical significance ***(Vaughan & Litt,1990).***

Approximately 95% of infants weigh between 2500 and 3500 grams at birth. Loss of excess fluid during the first few days, accounts for a weight loss of about 5-10% of birth weight, but the later has usually been recovered by about 10 days of life. Thereafter, the infant gains about 20 grams / day for the first 5 months, at which point, the infant has doubled his birth weight. For the remainder of the first year, the infant gains about 15 grams / day to triple his birth weight by one year ***(Tanner & Davies, 1985).***

During the second year of life, further deceleration in the rate of growth occurs. There is a weight gain of only 2.5 kilograms. During the third, fourth and fifth years of life, gain in weight is relatively steady at approximately 2 kilograms per year. The average gain in weight during school years is about 3 - 3.5 kilograms / year, ending in a preadolescent growth spurt by the age of 10 in girls, and 12 in boys ***(Vaughan & Litt, 1990).***

Table (2)
Average weight of normal infants and children
(Tanner & Davies, 1985)

Weight	Kilogram	Pounds
At birth	3.25	7
3 -12 months	(age in month + 9) / 2	age in month + 11
1 - 6 years	(age in year x 2) + 8	(age in year x 5) +17
7 -12 years	(age in year x 7 - 5) / 2	(age in year x 7) +5

Table (3)
Reference values for growth of children in developing countries
(Maurice, 1982)

Age in month	Healthy child's weight in Kg
Birth	3.5
4 month	6.3
6 month	7.5
8 month	8.4
10 month	9.3
12 month	10.0
18 month	11.3
24 month	12.5
36 month	14.5
48 month	16.5
60 month	18.5

When Wt. is unknown, but age (A) is known, we can calculate (Wt) from the prediction formula (for 2 - 6 years) : Wt = A - 2.4 / 2.8 . And the reverse occurs, we can calculate the age from the following formula, if we know the weight : A = Wt + 0.94 / 0.36 ***(Ogunrant, 1986).***

Height (Ht)

Ht is a good indicator of human growth in general. It is a stable measure, as compared with body Wt. The latter can fluctuate markedly in health disease as opposed to length ***(Tanner & Davies, 1985).***

The average length at birth is between 45 and 55 cm. Length increases by 25 - 30 cm during the first year. During the second year of life, the child will gain about 12 cm. During the third, fourth and fifth year of life, the child will gain about 6 - 8 cm / year during the school years, the average length gain is 6 cm / year. The length of an individual is composed of four components: legs, pelvis, spine and skull. For detailed studies of body proportions, all these measurements may be required, but usually only the total length is sufficient ***(Knobloch et al., 1980).***

Weight / Height indices (Wt / Ht)

The Wt / Ht ratio is an index that describes the current health status, expressing a child's Wt as the percentage of the expected Wt for the child's Ht.

$$\text{Wt/Ht ratio} = \frac{\text{Wt of child}}{\text{Wt of standard child of the same Ht}} \times 100$$

According to this index, normal children are those whose Wt / Ht ratio is 110%-90% of expected value. Mildly, moderately and severely malnourished children are those whose Wt / Ht ratio are between 90%- 85%, 85%-75% and below 75% of the expected respectively ***(Mclaren & Read, 1975).***

Indices based on Wt / Ht have the advantage that the measurements themselves can be made easily, quickly and with a fair degree of accuracy. The disadvantage of Wt / Ht index is that it includes the abnormal Wt contributed by a swollen abdomen, in a measurement which is assumed to represent healthy Wt, thus making the malnourished child appear better than actual condition ***(Zeitlin,1986).***

Midarm-circumference (MAC)

Next to Wt, the most widely used measurement for the assessment of nutritional status is the arm circumference. It has been recommended as an

alternative to Wt in the epidemiological assessment of protein-energy malnutrition (PEM) ***(Shakir, 1975).***

The MAC represents a summation of bones, muscles and fat components of the arm. It increases rapidly in the first year of life, but then remains fairly constant between the ages of 1 - 5 years. It increases 5.4 cm during the first year of life. From 1 - 6 years, the increase is only 1.5 cm ***(Alleyne, 1981).***

Simplicity of MAC measurement makes it an important diagnostic tool as the measuring tape is the only equipment that is required, and even untrained personel or paramedical staff may be utilized in surveying large number of children under field condition ***(Indira Bai & Sastry,1976).***

MAC of 13.5 cm is commonly accepted as the threshold indicating malnutrition. If the circumference is between 12.5 - 13.5 cm, the child is considered to be moderately malnourished, and measurements below 12.5 cm indicate severe malnutrition ***(Shakir & Morley, 1974).***

Table (4)
Average MAC of normal infants & children 1-6 years old
(Jelliffe, 1966)

Age in month	Boys	Girls
0	8.62 cm	8.16 cm
6	11.76	11.36
12	12.67	12.46
18	12.97	12.70
24	13.26	12.93
36	13.58	13.35
48	13.78	13.40
60	14.14	13.95

Table (5)
Correlation between nutritional status and MAC
in chidren 1 - 6 years old
(Shakir & Morley, 1974)

Degree of malnutrition	MAC	% of standard
Normal child	16 cm	
Mild malnutrition	13.5 cm	85%
Moderate malnutrition	13.5 - 12.5 cm	75 - 85%
Severe malnutrition	< 12.5 cm	< 75%

Triceps Skin fold (TSF)

Subcutaneous fat begins to be laid down in the foetus at about 30 weeks gestational age, increasing from then till birth and from birth onwards till about 9 months old, this is the usual in the average child. The peak may be reached as early as 6 months or as late as 12 - 15 months. Female values are higher than males ***(Forfar & Arneil, 1978).***

Since subcutaneous adipose tissue is a major component of body fat, skin fold thickness has been proposed as a useful index, not only of the amount of subcutaneous fat (about 50% of body fat), but also of relative fatness of the body ***(Goodhart & Medalie, 1974).***

For measurement of TSF, a mark is placed at the left acromion and olecranon processes. This distance is measured and the midpoint is marked. The skinfold is grasped by the examiner 1 cm superior to the previously marked midpoint. The skinfold thickness at a certain age is expressed as percentage of the standard skinfold thickness of the same age, using standard charts ***(Owen & Lippman, 1977).***

Table (6)
Correlation between nutritional status and TSF
(Malina, 1972)

Degree of malnutrition	TSF
Obese	110% - 120%
Normal	90 - 110%
Mildly malnourished	80% - 90%
Moderately malnourished	60% - 80%
Severely malnourished	< 60%

There is a high correlation between skinfold thickness and body Wt. So, the skinfold thickness is affected markedly by the loss or gain of Wt *(Malina, 1972)*

The main problems in measurement of skinfold thickness are :

1. It needs an expensive caliper and well trained anthropometrist
2. Standardizing the pinching effect of the calipers.
3. The distribution of fat on the upper arm is not uniform , and great care must be taken to select exactly the prescribed site.
4. In children at risk to kwashiorkor (KWO), the presence of edema can also influence the measurement *(Sann et al., 1988).*

Growth curves

Growth curves are constructed to fit, and thus, summarize human growth data. Fitting a curve to the individual values, is the only way of extracting the maximum information for the individual's measurement ***(Roche & Himes, 1980).***

Types of growth curves :

1. Distance curves :

It represents the size attained at each age. It shows the three phases of rapid growth of the infant, gradual accelerating growth of prepubertal child and the growth spurt of adolescence ***(Roche & Himes, 1980).***

2. Centile curves :

These curves describe the distribution of characteristics in a population. The various physical parameters may be expressed in term of percentiles for chronological age, from the fifth to the ninety fifth percentiles. The 50th percentile for length means that there is at each age a length above and below which 50% of infants lie, and thus 5% of infants have a length which falls below the 5th percentile ***(Roche & Himes, 1980).***

3. Velocity curves:

The rate of growth per unit of time is expressed as velocity in cm per year. Velocity curves truely reflect the pattern of growth in stature ***(Roche & Himes, 1980).***

Use of growth charts

Anthropometry is an essential component of infant health supervision. Therefore, use of the charts in public health clinics, nutrition survey studies and physician's offices, can assist identification of individuals with growth or nutritional abnormalities. Plotting anthropometric values for an infant on the charts, graphically indicates where the infant ranks relative to other infants of the same age and sex ***(Frankenburg et al., 1981)).***

The National Center for Health Statistics (NCHS) has conducted a large survey of characteristics of the growth of children in the United States. The children studied represented a cross - section of ethnic and economic groups; accordingly, some genetic, ethnic and socioeconomic differences are imbedded in the data. The data and the derived charts are best regarded, therefore, as *reference standards* rather than as a descriptive of any particular group of children ***(Hamill et al., 1979).***

THE DIET AND NUTRITIONAL REQUIREMENTS

I. Food

1. Food of vegetable origin

(a) ***Cereals:*** the common cereals are wheat, rye, oats, rice and maize. *Wheat* is the most universally important cereal, and is composed mainly of starch (70-75%). Gluten constitutes up to 90% of its protein which represents 7-15%. *Rice* is second to wheat in global importance, and although the proportion of protein is relatively less (6-8%) this is of high quality. *Rye* is still used to make bread which is rich in fibre and B vitamins. *Oats* contain more protein and oil than other common cereals. *Maize* is an important cereal in parts of Central America and Africa. The principal protein is zein which lacks lysine and tryptophan. This is important in the relationship between maize and pellagra ***(Paul & Southgate, 1978).***

(b) ***Legumes:*** it includes peas, beans, soya beans, and lentils. They are a rich source of protein, the methionine content of which is relatively low, and they complement cereals. This combination provides a cheap and nutritious protein diet ***(Passmore & Eastwood, 1986).***

(c) ***Root vegetables:*** each 100g of potato includes 70-80g of water, 20g of starch and only 2g of protein. Less starch is found in carrots, onions and radishes ***(Passmore & Eastwood, 1986).***

(d) ***Leaves, fruits and nuts:*** *leafy vegetables* are an important source of beta carotene, Vitamin C and folate. *Fruits* are major source of Vitamin C most of which is available as they are frequently eaten uncooked. Both fruits and vegetables supply fibre. *Nuts* have a high fat and protein content, but they are eaten in relatively small quantities and so they make an insignificant dietary contribution ***(Paul & Southgate, 1978).***

2. Food of animal origin

(a) ***Meat:*** protein in meat is of high biological value, being rich in essential aminoacids. The energy content depends on the amount of associated fat. Meat is an important source of iron, nicotinic acid and riboflavin, but it contains little calcium, Vitamin A or Vitamin C. Most meats contain a moderate amount of Vitamin B12 which is concentrated in the liver ***(Reina, 1983).***

(b) ***Fish:*** fish protein is also of high biological value and fish is an important source of iodide and fluoride. Fish oils contain high concentrations of VitaminA and VitaminD ***(Reina, 1983).***

(c) ***Eggs:*** egg protein, much of which is in the form of albumin in the egg white, has a very high biological value and is used as a standard by which

other proteins are compared. Eggs contain significant amounts of thiamin, nicotinic acid and riboflavin, and are also rich in cholesterol. The iron content is poorly absorbed because of binding by protein ***(Gaull, 1989).***

(d) Milk, cheese and butter: in addition to high- quality protein, of which there is more in cow's milk than human milk, *milk* contains carbohydrate in the form of lactose and a variable amount of fat. The calcium content is high and milk also contains small amounts of iron, Vitamin D and Vitamin C. *Cheese* contains a variable amount of fat and other nutrients such as calcium and VitaminA in addition to milk protein. *Butter* has a high fat content with fat soluble vitamins particularly Vitamin A ***(Barker & Bender, 1980).***

3. Miscellaneous foods

Thy include refined ***Sugar and honey***. These provide energy without other nutrients, and excessive consumption contributes to the problems of obesity and dental caries. ***Coffee and tea*** both contain caffeine, a mild stimulant and diuretic, and the potassium content of coffee can be considerable. ***Fruit juices*** also provide potassium, and ***soft drinks*** contain much sugar and added phosphate ***(Owen & Lippman, 1977).***

II. Dietary components

1. Proteins

Protein contains nitrogen, sulphate and carbohydrate. It is made up of chains of amino acids which are linked by peptide bonds. Nine amino acids cannot be synthesized and must be supplied, these are known as essential amino acids: histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan and valine. High quality proteins such as meat and egg are rich in these amino acids ***(Goodhart & Shils, 1980).***

2. Carbohydrates

Dietary carbohydrates may be ingested in several forms. ***Monosaccharides:*** include glucose, fructose, galactose and mannose. ***Disaccharides:*** lactose which consists of galactose and glucose is the most important disaccharide, and is the carbohydrate energy source in milk. Sucrose which consists of fructose and glucose is a major component of the Western diet. ***Polysaccharides:*** are polymers of glucose. They occur in two forms, starch and glycogen. Plants store carbohydrate as starch; the animal equivalent of starch is glycogen ***(Anderson, 1982).***

3. Fats

The majority of ingested fat is in the form of ***triglycerides, esters of glycerol and fatty acids.*** Fatty acids of animal origin are saturated, and those

of plant origin are unsaturated. Other dietay fats include ***phospholipids and sterols*** such as cholesterol. The latter is found in high concentration in eggs and dairy products ***(Goodhart & Shils, 1980).***

4. Vitamins

These are essential micronutrients. Table (7) summarizes physical properties, food sources and effects of deficiency of different vitamins.

5.Minerals

Dietary ***calcium*** is mostly obtained from dairy products such as milk and cheese. Root vegetables contribute significant amounts but there is very little calcium in potatoes. ***Phosphate*** is similarly obtained from dairy products and also meat. ***Magnesium*** is mostly obtained from foods of vegetable origin. ***Potassium*** is rich in chips, dried fruits, spinach, as well as beverages such as coffee and orange juice. ***Sodium*** content is high in processed food such as white bread, beef sausages and cheese. Up to one third of dietary salt is added during cooking ***(Goodhart & Shils, 1980).***

6.Trace elements

The essential trace elements, their dietary sources and the effect of their deficiency are summarized in table (8)

Table (7)
Physical properties, food sources and effects of deficiency of the Vitamins (Barber & Bender, 1980)

Vitamin	Physical property	Food sources	Deficiency
Vitamin A	Fat-soluble	Carrots,green leaf vegetables,liver, fish liver oils	Skin changes, nyctalopia,keratomalacia,photphobia
Thiamin (B1)	Water-soluble	Liver, meat and pork, whole grain	Beriberi (neuritis and heart failure)
Riboflavin (B2)	Water-soluble	Liver, dairy products, meat fish	Ariboflavinosis, cheilosis,angular stomatitis, photophobia, eye burn
Niacin (nicotinic acid)	Water-soluble	Meat, fish, poultry, green vegetables, whole grain	Pellagra (dermatitis, dementia and diarrhea
Pyridoxine (B6)	Water-soluble	Meat, liver,nuts, whole grain cereals, soya beans	Irritability,convulsions peri pheral neuritis
Cobalamine (B12)	Water-soluble	Liver, meat, eggs, cheese and fish	Juvenile pernicious anemia
Folacin	Water-soluble	Liver, cheese, cereals, spinach	Megaloblastic anemia (usually 2ry to malabsorption disease)
Vitamin C	Water-soluble	Fruits, tomatoes, green vegetables	Scurvy and poor wound healing
Vitamin D	Fat-soluble	Vitamin D-fortified milk and margarine, fish liver oils, exposure to sunlight	Rickets, infantile tetany, osteomalacia
Vitamin E	Fat-soluble	Vegetable oils, nuts, eggs, butter	Muscle weakness and hemolysis in premature infants
Vitamin K	Fat-soluble	Green vegetables, liver	Hemorrhagic manifestations

Table (8)
Food sources and effect of deficiencies of the essential trace elements (Mertz, 1981)

Trace element	Food sources	Deficiency
Iron	Liver, meat, egg yolk, green vegetables, nuts	Iron deficiency anemia
Zinc	Meat, fish, grain, nuts	Teratogenic effect in humans, poor growth, alterations in taste
Manganese	Legumes, nuts, fruits, whole grain cereals	Skeletal abnormalities and ataxia in offsprings of deficient mothers
Copper	Liver, meat, fish, nuts, whole grain cereals	Neutropenia, leukopenia bone demineralization, failure of erythropoiesis and finally death
Fluoride	Water, sea foods	Dental caries
Iodide	Sea food	Simple goiter

III.Nutritional requirements

1. Energy

The energy needs of a child are determined by his or her basal metabolism, rate of growth, body size, age and activity. Enough calories must be provided to ensure growth and to spare protein from being used as energy, yet they cannot be so excessive that obesity results. Of the total energy intake, a suggested proportion is 50-60% as carbohydrate, 25-35% as fat and 10-15% as protein ***(Owen & Lippman, 1977).***

Table (9)
Recommended dietary allowances of energy (kcal) for children

Age (years)	Daily mean (males)	Per kg (males)	Daily mean (females)	Per kg (females)
1-3	1300	100	1200	90
4-6	1700	85	1600	80
7-10	2400	86	2300	81

From Food and Nutrition Board, National Research Council: Recommended Dietary Allowances, 9th ed., Washington, D.C., National Academy of Sciences, 1980.

2. Proteins

The minimum protein requirement values refer to high-quality dairy protein. More protein will be needed when the diet is based on foods which contain low-quality protein, with a lower essential amino acid content. Furthermore, the absorption of amino acids from protein of vegetable origin may be less complete than that from protein of animal sources ***(Contento, 1981).***

Table (10)
Recommended dietary allowances of protein for chidren

Age (years)	Daily mean	Per Kg
1 - 3	23	1.8
4 - 6	30	1.5
7 - 10	34	1.2

From Food and Nutrition Board, National Research Council: Recommended Dietary Allowances, 9th ed. Washington. D.C., National Academy of Sciences, 1980.

FAILURE TO THRIVE (FTT)

Definition

FTT has been defined in a number of ways, but most definitions include a Wt less than the 5th percentile on the growth chart or a decreasing rate of Wt gain. Non-organic FTT (NOFTT), i.e. FTT not due to organic disease, is the most common category of FTT in the United states, and is associated with delayed growth and development and abnormal behaviors. Factors extrinsic to the infant are primarily responsible for NOFTT. That acute undernutrition may be a cause of the poor Wt gain is suggested by anthropometric studies, and by the observation that NOFTT infants often gain Wt when food is supplied. Yet, decreased caloric intake has been documented in only a few infants, and not all infants immediately gain Wt when given adequate calories. Current thinking attributes lack of Wt gain in NOFTT to probably mixed interacting causes, including decreased nutrition, and abnormal hormonal mechanisms associated with abnormal behavior ***(Powell, 1988).***

The term "failure to thrive" evokes a pathetic image, but do all those who care for children have a similar concept of what it means? Perhaps the biggest gap is between those who dig deep for biochemical or metabolic causes ("organic" FTT) and those for whom the social history reveals all the necessary clues ("non-organic" FTT). Batchelor and Kerslake now suggest that this distinction is artificial, that much FTT is missed, and that the feeding process is the root cause ***(Batchelor & Kerslake, 1990).***

The maximum weight centile achieved by a child between 4 and 8 weeks of age, was found to be a better predictor of the centile at 12 months than the birth weight centile. Children whose weight deviated two or major centiles below this maximum weight centile for a month or more, showed significant anthropometric differences during the second year of life, from those who showed no such deviation. It is suggested that this leads to a logical and practical definition of FTT ***(Edwards et al., 1990).***

Causes of FTT

To determinate the different characteristics of growth and development that could categorize the etiology of FTT, 174 patients hospitalized because of FTT were analyzed, 48% had organic causes and 52% had non organic causes ***(Morice et al., 1989).***

An approach to FTT should allow the practitioner to detect and classify this problem into NOFTT or OFTT , with minimal observation and testing. In the first step, we should evaluate adequacy of calories offered. Inadequate calories may be offered because of poverty, poor feeding technique or child neglect, which focuses the attention on social and environmental issues. If adequate calories are offered, they may be refused, vomited or retained. Refusal may direct attention to neuromuscular disorders, malignancy or aneroxia nervosa. Vomiting will direct attention to intestinal obstruction, gastro-esophageal reflux, and so on. If calories are retained, they may be lost as diarrhea; consumed by excessive energy requirement as in

chronic respiratory diseases, cardiac diseases or hyperthyroidism; or be ineffective because of end organ unresponsiveness. Excess fecal loss should direct attention to malabsorption disorders ***(Marvin & Rallison, 1986).***

Non organic failure to thrive (NOFTT)

NOFTT is called " reactive attachment disorder of infancy " (RADI). After exclusion of organic causes of Ht and Wt retardation below the fifth percentile (head circumference is usually normal), the diagnosis is based on the family history and on observation of the child-parent relationship. Studies demonstrate a variety of maladaptive mother-child interactions, in most cases impaired mothering is due to chronic depression, drug dependence, and poor day-to-day functioning. Fathers in these studies were often described as ineffective with respect to the mother-child relationship. There is a big role of postnatal intervention, in the prevention of the negative consequences of postpartum depression on developing mother-infant relationships ***(Milgrom, 1994).***

Adequate nutrition, especially for the infant, requires a healthy interaction between mother and child. The development of abnormal maternal-child interaction may lead to NOFTT. Factors involved in NOFTT include poor parental interaction with the child and inadequate caloric intake ***(Broughton, 1989).***

This form of FTT may be seen in a variety of psychological situations ranging from improper feeding technique to physical and psychological abuse,

and has potentially serious effects on child development, behavior and cognitive skills ***(Bithoney et al., 1991).***

Barbero and Shaheen in 1967, reported the following criteria by which NOFTT may be recognized:

1. The child has a low Wt which improves noticably with appropriate nurturing.
2. There is no evidence of systemic disease or an organic cause for the FTT.
3. Developmental retardation improves with appropriate stimulation and feeding.
4. Signs of social or intellectual deprivation improve with better nurturing in a new environment.
5. The family environment shows signs of psychological disruption ***(Barbero & Shaheen, 1967).***

Causes of NOFTT

I- Abnormal maternal - child interaction. (Psychological deprivation)

Psychological deprivation represents a more serious disruption of the maternal-child bond, and has been variously termed maternal deprivation, emotional deprivation or environmental deprivation. All of these terms imply a gross disturbance of the normal maternal-child interaction, resulting in disordered growth and development ***(Sell et al., 1991).***

(1) Inadequate mother - child bonding:

Bonding between mother and child, appears to be a basic need for normal growth and develpoment of the child. If the baby is placed with the mother even for a short time immediately after birth, bonding and subsequent growth and development are obviously improved.The adverse effect of abnormal maternal-child interaction on growth and nutrition represents the most common cause of FTT, with the exception of world protein-calorie malnutrition ***(Green et al., 1979).***

Infants develop a sense of self, a secure attachment to caregivers and a positive view of the world from experiencing appropriate and immediate responses. There is also evidence that there are continuities between interactional difficulties in the first six months of life and subsequent disturbances in childhood emotional, social and intellectual development.

Newborns capabilities to engage in social interaction, are apparent from the first few hours of life. Alert newborns will imitate the facial expressions of the first person they see ***(Lier, 1988).***

(2) Impaired mothering :

Adolescent drug addiction and alcohol abuse became a widespread serious problem. Teenagers of all races, socioeconomic groups and both genders have become avid experimenters with a full spectrum of substances, including alcohol, marijuana, stimulants, cocaine and hallucinogens. A diagnosis of addiction requires familiarity with the normative trends of adolescent behavior, as well as deviations from these trends. Moreover, an adolescent mother with a drug addiction problem confronts the additional burden of fostering adaptive developmental patterns in her infant. Treatment techniques such as previewing, which empowers through the representation of future outcomes, have helped addicted teenage mothers relinquish drug use and adopt more mature caregiving behaviors ***(Trad, 1993).***

FTT with severe congenital disorders or acquired diseases may be due, in part, to a breakdown of mothering caused by the mother's attitude towards the affected child. This was described in a child with a small congenital heart defect, who failed to thrive until the mother became convinced that the child was not going to die ***(Barbero, 1967).***

It is often postulated that a mother's past experiences influence her ability to function as a parent. If those past experiences involve her as a

victim of abuse, what lies ahead for her offspring ? A study was done on 59 mothers of children referred for NOFTT to the university of Colorado Health Sciences Center, it compared their abuse history with a group of 131 mothers of children with normal growth. The mothers of NOFTT children were younger but of the same socioeconomic groups as the comparison group. A surprising 80% of mothers of NOFTT children reported they were victims of abuse ***(Weston et al., 1993).***

(3) Postnatal Maternal Depression

Postnatal maternal depression is a psychiatric illness that affects 8-23% of women in the postnatal period. It impairs the woman's ability to function in her many roles, and has also been shown to increase her child's risk of later emotional, cognitive and social problems. The finding that the children of such depressed mothers may show behavioral disturbances at the age of 3 years, or cognitive defects at the age of 4 years, suggests that postnatal depression may have a long-term negative impact on the family ***(Schaper et al., 1994).***

II- Inadequate calories offered

1. Protein - Energy malnutrition (PEM)

Worldwise, the most common cause of FTT is starvation or PEM, a consequence of unavailability of adequate food. This is more obvious in developing countries due to poverty and low socioeconomic level ***(Cupoli et al., 1980).***

Malnutrition is the primary biologic insult in most cases of FTT ***(Frank & Zeisel, 1988).***

Preschool children particularly in tropics, are more susceptible to PEM. Protein and energy requirements of children are substantially higher per unit weight, in addition unhygienic habits and immaturity the of immune system increase susceptibility to infection. Gastrointestinal infections in particular constitute a major precipitant of PEM in infants because such illness results in altered feeding habits, vomiting, decreased intestinal absorption, increased metabolic needs and increased metabolic losses ***(Mason & Rosenberg, 1991).***

Nutrition of children on severe or strict vegeterian diet, sometimes lacking dairy products and eggs, is not suitable for babies or infants. Serious deficiency states have been described after such regimens, as rickets, anemia and FTT ***(Lentze, 1992).***

Wellcome classified PEM in 1970 into: a. marasmus with Wt less than 60% of Boston median for age, b. marasmic kwashiorkor with Wt below 60% and edema present, c. kwashiorkor with Wt greater than 60% and edema present, d. underweight with Wt 60 - 80% and no edema, the 80% of Boston median Wt is about the 3rd centile ***(Barltrop, 1992).***

Precise evaluation of nutritional status is difficult. Severe disturbances are readily apparent, but mild disturbances may be overlooked, even after careful physical and laboratory examinations. The diagnosis of malnutrition rests on an accurate dietary history; on evaluation of present deviations from average Ht, Wt, HC, and past rates of growth; on comparative measurements of MAC and skinfold thickness; and on chemical and other tests. Decreased skinfold thickness suggests PEM ***(Hegsted, 1978).***

Jeffrey et al., compared the results of three methods of categorizing undernutrition in a group of children referred for evaluation of "FTT". There was broad variation between the number of children classified as malnourished (75% to 98%) and the degree of undernutrition, depending on the method used. Jeffrey recommended that categorization systems should be used only to define a child as having a risk of possible adverse effects of undernutrition, not as being malnourished ***(Jeffrey et al., 1994).***

Table (11)
Comparison of three methods to categorize undernutrition in children (Jeffrey et al., 1994)

Degree of undernutrition	Method 1 (Gomez): % median Wt for age	Method 2 (Waterlow): % median Wt for Ht	Method 3 (McLaren Read) %median Wt/Ht for age ratio
None	> 90	> 90	> 90
Mild	75 - 90	80 - 90	85 - 90
Moderate	60 - 74	70 - 79	75 - 84
Severe	< 60	< 70	< 75

(a) Marasmus:

Occurs in infants who are severely deprived of adequate calories. Initially, there is failure to gain Wt, followed by loss of Wt until emaciation results, with loss of turgor in skin that becomes wrinkled and loose as subcutaneous fat disappears. Because fat is lost last from the sucking pads of the cheeks, the infant's face may retain a relatively normal appearance for some time before becoming shrunken and wizened. The abdomen may be distended or flat, and the intestinal pattern may be readily visible. Atrophy of muscle occurs, with resultant hypotonia. The infant is usually constipated, but the so-called starvation type of diarrhea may appear, with frequent, small stools containing mucous ***(Cupoli et al., 1980).***

(b) <u>Kwashiorkor:</u> (KWO)

It is another PEM disorder that occurs due to insufficient intake of protein of good biologic value. There may also be impaired absorption of protein, such as in chronic diarrheal states, abnormal losses of protein in proteinuria (nephrosis), infection, hemorrhage or burns, and failure of protein synthesis, such as in chronic liver disease. KWO is a clinical syndrome that results from a severe deficiency of protein and an inadequate caloric intake. It is the most serious and prevalent form of malnutrition in the world today, especially in industrially underdeveloped areas. It may become evident from early infancy to about 5 year of age, usually after weaning from the breast ***(Zain et al., 1977).***

Early clinical evidence of KWO is vague but does include lethargy, apathy, or irritability. When well advanced, it results in inadequate growth, loss of muscular tissue, increased susceptibility to infections, and edema. The liver may enlarge early or late, fatty infiltration is common. Edema usually develops early, failure to gain Wt may be masked by edema, which is often present in internal organs before it can be recognized in the face and limbs. Dermatitis is common. The hair is often sparse and thin and loses its elasticity. Infections and parasitic infestations are common ***(Sleisenger, 1979).***

2. Improper feeding techniques

The mother may not understand simple fundamentals in infants feeding, and may be offering too much or too little, of a formula which is too rich or too poor, through a nipple with too large or too small holes ***(Dunitz & Scheer, 1991).***

3. Improper breast feeding

(a) Inadequate breast milk supply:

A study was done to determine whether FTT in a breast fed infant could be attributed to altered milk production or composition from a mother who consumed a self-imposed energy and protein restricted diet. The study was based on the dietary intakes by food records, milk production by the test weighing procedure, and milk composition by proximate analysis. This study showed a marked decline in the infant's linear and ponderal growth rates, when the mother consumed an energy restricted diet. Maternal milk production showed positive relationships with maternal dietary energy and protein intakes. The conclusion of this study was that FTT in a breast fed infant could be attributed to reduced milk production in conjunction with maternal dietary energy and protein restriction, and that an assessment of maternal dietary intakes is essential in an evaluation of the breast fed infant with FTT ***(Motil el al., 1994).***

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(b) Prolonged breast feeding:

A study on the determinants of nutritional marasmus and KWO was conducted using Sudanese children aged 6-36 months. Mothers were interviewed in hospital, and information on duration of breast feeding, age at introduction of supplementary foods, and weaning foods was obtained. The results suggest a positive association between prolonged breast feeding without introduction of supplementary feeding between the ages of 6 and 24 months, and PEM ***(Abdel Sayed, 1995).***

(c) Ineffective suckling:

FTT in breastfed babies frequently is attributed to inadequate lactation. In some cases, the origin of inadequate lactation may be ineffective suckling that obstructs milk flow. To support this conclusion, an infant who presented with FTT was studied at the breast with magnetic resonance imaging ***(Morton, 1992).***

4. Parental misconceptions and health beliefs

Parental misconceptions and health beliefs concerning what constitutes a normal diet for infants is reported as a cause of NOFTT. The children's caloric intake had been restricted by their parents. They were concerned that their chidren would become obese, develop atherosclerosis, become junk food dependent, and/or develop eating habits that the parents believed were unhealthy. The parents instituted diets consistent with health beliefs currently

in vogue and recommended by the medical community for adults who are at risk for cardiovascular disease. These diets caused the infants to experience inadequate Wt gain and have a decreased linear growth rate. With nutritional counseling, all food restrictions were removed, so the Wt gain rate and the linear growth rate increased significantly ***(McCann et al., 1994).***

5. Excess fruit juice consumption

Eight patients, 2 years old, referred for FTT were evaluated by medical history, physical examination, and biochemical assessment. All children exhibited weights <5th percentile, and five also had lengths <5th percentile. Organic disease was ruled out in all cases. The children's diets were hypocaloric, providing 78% to 92% of recommended energy intake for age and Wt. Dietary intake included considerable juice consumption (12 to 30 oz/day). Fruit juice, primarily apple juice, contributed 25% to 60% of daily energy intake. As a result, food consumption was reduced, lowering dietary protein, fat and micronutrient intakes. Excessive juice was consumed for various reasons including children's preferences, parental health beliefs, behavioral feeding difficulties and financial considerations. Breath hydrogen testing revealed malabsorption of fructose and/or sorbitol. After nutritional intervention, dietary intake increased to 96% to 116% of recommended intakes and Wt gain increased significantly. So, these findings indicate that large intakes of fruit juices may displace more caloric and nutrient dense foods. Additionally, fructose and sorbitol malabsorption may occur. Excess fruit juice consumption may present a contributing factor in NOFTT ***(Smith & Lifshitz, 1994).***

III- Child abuse .

About 1 million children are abused or neglected each year in the United States, and about 2 thousand die from nonaccidental injury. Child abuse is more likely to occur in the younger children, 40% of victims are under 5 years of age. It is more common than appendicitis, and the mortality is many times higher *(Levin, 1987).*

1. Physical abuse

Physical abuse accounts for 60 - 65% of reported cases. The abused child is a victim of his family. ***The parents*** are often poorly prepared for child rearing. They have unrealistic expectations of the child, are frequently impulsive, and tend to use corporal punishment. These parents usually have little social support and often misuse drugs and alcohol. ***The child*** is often single out in a family. ***The trigger*** that often predisposes to abuse, may be the loss of a job, an illness, or a death in the family. Crowded housing also plays a role. No social class is immune *(Levin,1987).*

Diagnosis and assessment of physical abuse needs the following:

* A detailed history must be taken from all persons involved. Suspicious findings include any discrepancy between the history and the physical findings, different stories given by different people, delay in seeking medical help, child brought to the hospital by a parent who was not present

when the trauma occured, and repeated accidents involving the same child ***(Kessler & New, 1989).***

* A complete physical examination to detect signs of neglect, such as poor growth, poor hygiene, bruises, burns, frenulum injury which may result from forced feeding, skeletal injuries and brain injury. The latter is the primary cause of death in most cases of physical abuse, it can result from direct trauma such as directly inflicted subdural hemorrhage or indirect trauma due to shaking. The whiplash shaken infant syndrome (WSIS) should be suspected in any young infant with an unexplained decrease in level of consciousness ***(Kessler & New, 1989).***

2. Neglect

Neglect is not so obvious as in abuse. Accordingly, it is more difficult to recognize. Situations that should arouse suspicion include FTT, developmental delay (if there is no obvious medical explanation), poor hygiene, and delayed immunizations ***(Levin, 1987).***

Physical neglect, which must be differentiated from the effects of poverty, will be present even after the provision of adequate social services to families in financial need. Neglectful parents appear to have an inability to recognize the physical or emotional states of their chidren, they respond differently to their children. A baby's cry may be perceived as an expression of anger at the parent, which can then be ignored. If hunger cries are

misinterpreted in this way, the infant may not get adequate food, leading to FTT ***(Belsky & Rovine, 1988).***

3. Sexual abuse

Child sexual abuse is the involvement of developmentally immature children and adolescents in sexual activities that they do not fully comprehend. Sexual abuse is the most frequent form of abuse, one in four females and one in ten males have been sexually abused by the age of 18 ***(McCann,1990).***

The offender is rarely criminal or psychotic. He often has poor family relationships and low self-esteem, is sexually insecure, and engages in sexual activities with children to gain a feeling of power and control. *The mother* of the child is often passive and dependent, and is unable to protect the child. She may be aware of the problem but is afraid to say anything for fear of disrupting the family. *The child* or the victim is often angry and afraid and feels trapped. In many cases the abuse continues for years ***(McCann, 1990).***

Diagnosis and assessment of sexual abuse needs the following :

* A detailed history should be taken carefully. Interviewing the parents should be alone and before dealing with the child, if possible. A minimum number of people should be present. Interviewing the child should be in simple terms that he or she can understand. Children may retract their statements, so record what they say carefully; they may tell their story only

once. Other relevant data in the history include the presence of dysuria, pain on defecation, constipation, vaginal or anal bleeding, previous trauma to the perineal area. The child must be assured that the story is believed and that he or she will not be punished ***(McCann, 1992).***

* Physical evidence of abuse. Acute signs include redness, bruising, edema, and hymenal abrasions. Chronic signs include hymenal scarring and attenuation. Evidence of anal assault should be examined. Acute signs include anal fissure and redness. Chronic signs may include anal laxity and healed scars ***(Herman-Giddens & Frothingam, 1987).***

* Collection of specimens. Take saline-moistened swabs from the vagina and rectum for gonorrhea and Chlamydia, and throat swab for gonococcus. Use a moistened swab or feeding tube to obtain a vaginal sample for sperm detection ***(Cowell, 1981).***

* Behavioral indicators depend on the chid's age. Examples of such signs are the use of sexual terminology inappropriate for age, regressive behavior, secondary enuresis or encopresis, sleeping and eating disorders, poor school performance and running away from home. The more sexual the behavior, the more concerning it is for sexual abuse ***(McCann, 1992).***

4. Munchausen syndrome by proxy (MSBP)

MSBP is a form of child abuse in which a parent falsifies illness in a child by fabricating or producing symptoms, and presenting the child for

medical care while disclaiming knowledge as to the cause of the problem ***(Stevenson & Alexander, 1990).***

The following "diagnostic pointers" have been recommended as indicators that the presenting symptoms may be fabricated:

- Symptoms and signs that are unusual or bizarre and inconsistent with known pathophysiology.
- Observations and investigations inconsistent with parental reports or the condition of the child.
- Treatments which are ineffective or poorly tolerated.
- Parents who are unusually calm for the severity of illness.
- Parents who have a history of unusual illness ***(Samuels & Southall, 1992).***

Unthinking use of such indicators without realizing their non-specific nature, may lead to mothers being falsely accused. Nevertheless the presence of several criteria does, if nothing else, raise the chances that the child is at risk ***(Morley, 1995).***

IV-Anorexia nervosa.

The incidence of anorexia nervosa (AN) and bulimia has increased over the last 2 decades. It is estimated that 1:100 females, 16-18 yr old, has anorexia nervosa. Affected females exceed males by 10 to 1. The Diagnostic and Statistical Manual of Mental Disorders (DSM-IIIR) criteria for the diagnosis of AN include :

a. Intense fear of becoming obese, which does not diminish as Wt loss progresses.

b. Disturbance in the way in which one's body Wt, size, or shape is experienced (e.g. claiming to "feel fat" even when one is emanciated.

c. In females, absence of at least three consecutive menstrual cycles when otherwise expected to occur ***(Bruch, 1978).***

Patients who have AN are subdivided into the restrictor and bulimia subgroups, according to their method of caloric reduction. Restrictors severely limit their intake of carbohydrate and fat-containing foods, whereas bulimics tend to eat in binges and then to purge themselves of food by self-induced vomiting or the use of cathartics. The death rate in AN is approximately 10% and is usually caused by severe electrolyte disturbance, cardiac arrhythmia, or congestive heart failure in the recovery phase ***(Palla & Litt, 1988).***

Organic failure to thrive (OFTT)

I- Refusal of Adequate calories

Conditions in which adequte calories are offered but are not accepted or are refused, range from purely mechanical inability to accept food, to emotional rejection of nutrients ***(Marvin & Rallison, 1986).***

1. Congenital anomalies of upper digestive system

(a) Cleft lip and palate :

The incidence of cleft lip with or without cleft palate is about 1:1000 birth, the incidence of cleft palate alone is about 1:2500 birth. The most immediate problem is feeding; a plastic obturator is fitted soon after birth to aid in control of fluids and provide a reference plane of suction. Soft artificial nipples with large openings are beneficial to patients with cleft palate. Patients with isolated cleft lip may be breast fed ***(Ross, 1987).***

(b) Esophageal atresia and tracheoesophageal fistula :

Esophageal atresia occurs in 1:3000-4500 live births, about one third of the affected infants are born prematurely. In more than 85% of cases, a fistula between the trachea and distal esophagus accompanies the atresia. Diagnosis of esophageal atresia is ideally made in the delivery room, when the catheter used for ressuscitation cannot be inserted into the stomach. Contrast medium used for X-Rays should be water soluble, less than 1 ml given under fluoroscopic control is sufficient to outline the blind upper pouch. The contrast medium should then be withdrawn to prevent overflow into the lungs and development of chemical pneumonitis ***(Puntis et al., 1990).***

Clinically, esophageal atresia may present with dysphagia, regurgitation, vomiting and excessive oral secretions. When a fistula connects the proximal esophagus to the trachea, the first attempt at feeding may lead to

massive aspiration and cyanosis. If a fistula connects the trachea and distal esophagus, air usually enters the abdomen which often becomes tympanitic and distended ***(Reyes, 1989).***

2. Macroglossia of congenital hypothyroidism

Congenital hypothyroidism is twice as common in girls as in boys. Feeding difficulties especially sluggishness, lack of interest, somnolence, and choking spells during nursing, are often present. These manifestations progress leading to retardation of physical development, in addition to the mental retardation which increases with age ***(Fisher, 1991).***

3. Neurological causes

(a) Cerebral palsy: (CP)

Feeding difficulties, vomiting and recurrent chest infections associated with poor growth and nutrition are common in children with CP. Symptoms consistent with gastroesophageal reflux (severe feeding difficulties, failure to thrive, anemia and recurrent chest infections) are often present. However, the role of gastroesophageal reflux as a possible cause has been little studied ***(Reyes et al., 1993).***

MAC and TSF were significantly smaller on the affected side compared with the unaffected side, in children with hemiplegic CP. These results suggest that non-nutritional factors related to disease severity have a

significant influence on the growth of children with CP, even in the absence of malnutrition ***(Stevenson, 1995).***

(b) Duchenne muscular dystrophy:

Patients with Duchenne muscular dystrophy present with FTT in infancy. Length growth is always affected, while growth in head circumference remains unaffected. FTT in Duchenne muscular dystrophy is neither widely recognized nor understood ***(Rapisarda, 1995).***

4. Genetic causes

(a) Smith-Fineman-Myers syndrome :

Five cases of Smith-Fineman-Myers syndrome were reported with mental retardation, short stature, cryptorchidism, asplenia in one, and dramatic FTT ***(Ades et al., 1991).***

(b) Brachmann-de-Lange syndrome : (BDLS)

Gastroesophageal abnormalities occur with increased frequency in patients with BDLS, and contribute to problems with feeding, emesis and FTT. Findings in these patients included tracheal aspiration, esophageal dismotility, gastroesophageal reflux, hiatal hernia, and esophagitis. Medical treatment was instituted where appriopriate, and surgical treatment was performed if the problems did not resolve with medical treatment.

Improvement in Wt centiles occured in all patients fed by nasogastric or feeding gastrostomy tube, but only one patient appeared to experience increase in rate of linear growth ***(Bull et al., 1993).***

(c) Trisomy 21 : (Down syndrome)

The Down syndrome is a cytogenetic disease, the fundamental abnormality is an extraquantity of chromosomal material of chromosome 21. In most of the patients, there are 47 chromosomes and a trisomy chromosome No.21 (94%), this results from non disjunction during meiosis in the formation of the ovum, which is highly correlated to the increased maternal age ***(Nyhan & Saketi, 1976).***

The major clinical features of Down syndrome are mental retardation, FTT, oblique palpebral fissures, epicanthal folds, protruding tongue, congenital heart disease mainly septal defects, small penis, short broad hands with Simian crease, and clindactyly of the 5th finger ***(Thompson,1986).***

(d) Wiedemann-Rautenstrauch syndrome:

All affected children have had intrauterine growth retardation with subsequent failure to thrive and short stature. At birth and during infancy children have a progeroid appearance consisting of apparent macrocephaly, sparse hair, prominent scalp veins, entropion, greatly widened anterior fontanelles and generalised lipoatrophy. Over time, the nose assumes a beaked appearance and caudal fat accumulation. Mental retardation is usually

present to some degree, ranging from mild to severe impairment. In 1977 Rautenstrauch and Snigula reported on two sisters with a previously unreported, progeria-like syndrome. In 1979 Wiedemann described two unrelated males with the same condition ***(Toriello, 1990).***

(e) Imerslund-Grasbeck syndrome:

It is a rare inherited disorder characterized by a megaloblastic anemia due to a selective vitamin B12 malabsorption, in association with a mild proteinuria. Usually recurrent infections, gastrointestinal complaints, and pallor are presenting symptoms ***(Wulffraat et al., 1994).***

5. Malignancies

In the United States, cancer causes more deaths than any other disease of children between the ages of 1 and 15 yr. The incidence rate of malignant tumors in children under 15 yr of age is estimated to be 14:100,000/yr for the years 1986-1987 ***(Fearon & Vogelstein, 1990).***

Malignancies may be subtle evidences of widespread nutritional deficiency, such as anemia, osteopenia, and loss of subcutaneous tissue. The anorexia accompanying the tumor and increased metabolism by the tumor are important factors leading to FTT ***(Skuse & Rowley, 1989).***

6. Diabetes Insipidus (DI)

Diabetes insipidus, characterized by polyuria and polydipsia, results from lack of the antidiuretic hormone, arginine vasopressin (AVP). Vasopressin deficiency may be total or partial with varying degrees of polyuria and polydipsia ***(Richman et al., 1981).***

Polydipsia and polyuria are the outstanding symptoms of DI. Hyperthermia, rapid loss of Wt, and collapse are common in infancy. Vomiting, constipation, and growth failure may be observed. Dehydration in early infancy may result in brain damage and mental impairment. Anorexia is common ***(Czernichow et al., 1985).***

Any lesion that damages the neurohypophyseal unit may result in DI. Tumors of the suprasellar and chiasmatic regions are common causes; the symptoms of increased intracranial pressure may accompany those of DI or may follow years later. Encephalitis, sarcoidosis, tuberculosis, and leukemia are occasional causes. Injuries to the head, especially basal skull fractures, may produce DI immediately or after a delay of several months ***(Sklar et al., 1985).***

II- Loss of calories by chronic vomiting

Vomiting is a prominent symptom of disorders of esophagus and stomach, of bowel obstruction at any level, and of systemic illness which can cause reflex vomiting ***(Marvin & Rallison, 1986).***

1. Esophageal stenosis

It has been discussed before (Page 55).

2. Gastroesophageal reflux (Chalasia)

When the lower esophageal sphincter is not competent, excessive and passive reflux of gastric contents may cause significant symptoms. The term chalasia describes free reflux across a dilated sphincter ***(Nakayama et al., 1987).***

The signs and symptoms relate directly to the exposure of the esophageal epithelium to refluxed gastric contents. In 85% of affected infants excessive vomiting occurs during the 1st week of life; an additional 10% have symptoms by 6 weeks. Symptoms abate without treatment by the age of 2 years as the child assumes a more abrupt posture and eats solid foods, but the remainder continue to have symptoms until at least 4 years of age. Patients with cerebral palsy, Down syndrome, and other causes of developmental

delay have an increased evidence of reflux. Growth and Wt are adversely affected in about two thirds of patients ***(Azizkhan et al., 1980).***

3. Chronic intestinal obstrucion

(a) Hirschsprung's disease : (congenital megacolon)

It is the most common cause of chronic intestinal obstruction in children. The disease results from lack of ganglion cells in a part of the bowel wall, which results in abnormal peristalsis and functional obstruction. Episodes of constipation and diarrhea alternate with periods of apparently normal bowel function. Chronic constipation, abdominal distention and growth failure are common complaints in the older child ***(Schofield et al., 1990).***

(b) Tumors of the bowel :

Tumors of the bowel, including mesenteric cysts and polyps, may be obstructive. Vomiting and abdominal distention may occur with either mechanical obstruction or ileus; severe colicky periumbilical pain and hyperactive bowel sounds are almost invariably found in the former ***(Schuffler,1990).***

(c) Chronic idiopathic intestinal pseudo-obstruction : (CIIP)

CIIP is a condition of impaired intestinal propulsion producing persistent or recurrent episodes of nonmechanical gastrointestinal obstruction, resulting from hyperplasia of the Schwann cells and neurons. It involves the small intestine and colon, and rarely the stomach or esophagus. The male to female ratio is equal, and overall 20% are familial ***(Glassman et al., 1989).***

CIIP is manifested by abdominal distention , vomiting, constipation, and FTT. The onset is often in the neonatal period; 50% of patients manifest signs before the age of 1 month. The mortality may approach 30% during early childhood ***(Schuffler, 1990).***

4. Celiac syndrome
(Gluten sensitive enteropathy)

Celiac syndrome is said to be a rare cause of chronic malabsorption, although its frequency may have been underestimated. It is caused by a hereditary intolerance for gluten fraction of wheat, rye and oats. The gliadin fraction of glutens is injurious to the surface epithelium leading to gradual disappearance of the villi. The mechanism of injury appears to involve an immunologic reaction ***(Swinson et al., 1983).***

The diagnosis is easy in most cases, if not obscured by an inconsiderate and inconsequent approach. The most frequent mistake is prescribing gluten free diet on trial before proving the diagnosis by small bowel biopsy. The basic prerequisite for the diagnosis of celiac disease is the

finding of subtotal villous atrophy in the small bowel mucosa. The only absolute contraindication against small bowel suction biopsy is a blood clotting defect. FTT, deficiency states and abnormal stools are the leading symptoms of celiac disease ***(Granditsch, 1990).***

The clinical features of celiac disease range from generalized severe intestinal malabsorption to normal or nearly normal health. The stools are pale and foul, the child underweight and perhaps short, with wasted muscles, particularly in the proximal groups. The most constant features are decreased rates of Wt gain and linear growth, which may persist without obvious gastrointestinal symptoms ***(Swinson et al., 1983).***

III- Loss of Calories (Excessive fecal loss).

1. Malabsorption syndromes

Malabsorption is characterized by increased fecal loss of nutrients, abdominal distention and FTT. The stools may be watery, bulky and fatty, or even normal, depending on the cause of the malabsorption. In infants this problem is especially critical, because not only the infant is deprived of necessary nutrients, but he also tends to lose large amounts of fluid as well, and can become profoundly dehydrated. Since damaged mucosal cells are not replaced as rapidly as they are in the adult, attempts to refeed the infant may actually worsen the malabsorption state and lead to many secondary aspects of malabsorption, including intractable diarrhea ***(Ament, 1972).***

(a) Cystic fibrosis of the pancreas :

Cystic fibrosis is an inherited multisystem disorder of children and adults, characterized chiefly by chronic obstruction and infection of airways, and by maldigestion and its consequences. It is characterized by, failure to clear mucous secretions, paucity of water in mucous secretions, elevated salt content of sweat and other serous secretions, and chronic infection limited to the respiratory tract ***(Johansen et al., 1991).***

In cystic fibrosis of the pancreas, the lack of pancreatic digestive enzymes lead to maldigestion of all classes of food, most notably protein and fat. The stools are frequent, bulky, greasy, foul smelling and characteristically contain readily visible droplets of fat. FTT may be primarily due to nutritional disorder, but pulmonary infection and insufficiency may contribute to growth failure as well ***(Bines & Israel, 1991).***

(b) Digestive enzyme deficiencies :

Specific digestive enzyme deficiencies, such as disaccharidase, enterokinase or lactase, can cause malabsorption of a specific class of food and lead to diarrhea and FTT ***(Flats, 1987).***

(c) Celiac syndrome :

It has been discussed before in causes of loss of calories by chronic vomiting (page 63).

(d) Gastrointestinal allergy :

Many foods are capable of provoking an allergic response in the gastrointestinal tract, but milk is the most common offender among children who fail to thrive. In some cases an IgE mechanism has been established. In others, however, even with antibodies to milk proteins (particularly alpha-lactalbumin, beta-lactoglobulin, and casein) present in sufficient quantities to be demonstrable by gel diffusion methods, no immunologic mechanism has been established ***(Hill et al., 1984).***

Diarrhea occurs often with vomiting and abdominal pain, beginning within the first few weeks or months of life. The stools are watery and contain excessive mucous, and frequently blood. The diagnosis of milk allergy is based on clinical criteria. Symptoms should subside within 48 hours after removal of milk from the diet, and recur within 48 hours after a trial of feeding ***(Atkins & Metcalfe, 1984).***

2. Inflammatory bowel syndrome

(a) Crohn disease :

Crohn disease is a slowly progressive and persistent inflammatory disorder of the gastrointestinal tract, characterized by monocytic inflammation of the mucosa and submucosa of the bowel wall. Granulomas, composed of epithelial and giant cells, are found in mucosa and in lymph nodes ***(Ament, 1975).***

Pain is the most common complaint accompanied by Wt loss, short stature, abdominal tenderness, anemia and clubbing of fingers. Watery diarrhea, sometimes with blood, is present in 90% of patients at the time of diagnosis. Many simultaneous processes lead to FTT in Crohn disease. The inflammatory process may produce anorexia and malaise while increasing metabolic needs. The thick inflammed portion of the bowel interferes with motility and produces a functional bowel obstruction, causing postprandial pain and anorexia. Protein may be lost in large amounts from the inflammed mucosa ***(Mashako et al., 1989).***

(b) Ulcerative colitis :

In its active state, ulcerative colitis may interfere temporarily with growth and be a cause of FTT, but it only occasionally produces permanent FTT. The disease is rare in infancy when milk protein allergy is a leading cause of chronic colitis. It always involves the distal segment (rectum) and

extends to a variable extent proximally. Children with severe disease have a bloody diarrhea, abdominal cramps, fever, anemia, Wt loss and peripheral edema *(Michener & Wyllie, 1990).*

3. Biliary tract disease

FTT in biliary atresia or chronic liver diseases may stem from diminished food intake due to lack of appetite, nausea and vomiting, or from malabsorption and diarrhea due to faulty fat absorption. In addition to the impairment of fat absorption, there may be also deficiencies of fat soluble vitamins A,D,E and K *(Mieli-Vergani et al., 1989).*

IV- Excess energy expenditure

It is logical to assume that an infant or child will fail to gain Wt if energy expenditure exceeds that of intake, but it is difficult to demonstrate excess energy expenditure as the probable cause of FTT. In many circumstances in which excess energy expenditure is suspected, anorexia and decreased food intake may contribute to the FTT *(Marvin & Rallison, 1986).*

1. Recurrent or prolonged fever

Recurrent or prolonged fever may require excess energy, however, the disease state which produces the fever may also cause malaise and anorexia, leading to decreased food intake, or may interfere with food utilization by vomiting or diarrhea. Any disease state characterized by prolonged fever is

capable of interfering with growth, and only correction of the fever producing disease will assure resumption of normal growth. Excess caloric utilization may be combined with anorexia or failure to utilize nutrients in repeated acute or chronic infections ***(Marvin & Rallison, 1986).***

2. Inflammatory bowel disease

Inflammatory bowel disease can contribute to FTT in several ways, there may be excess fecal loss, anorexia and excessive energy expenditure. It has been discussed before in causes of loss of calories (page 66)

3. Arthritis and other inflammatory disorders

A similar wasting of energy and failure to gain Wt can be seen in the inflammatory arthritis such as juvenile rheumatoid arthritis, especially during periods of active disease ***(Harris, 1990).***

4. Congenital heart diseases (CHD)

CHD occurs in approximately 8 of 1000 live births. The incidence is higher among premature infants. FTT in congenital heart diseases is multifactorial. It occurs in infants with congestive heart failure or cyanotic heart defects for which cellular hypoxia seems to be the common denominator. In addition, ther is a reduction in food intake and a relative or absolute hypermetabolism ***(Hoffman, 1990).***

FTT in infants with CHD can be attributed to their low energy intakes and high resting energy expenditures. Energy intake, energy expenditure and growth were studied in infants with CHD on normal formula feeds, and then on feeds supplemented with glucose polymer to see whether supplementation improved energy retention and growth. Mean gross energy intakes increased by 31.7% on high energy feeding and mean Wt gain improved from 1.3 g/kg/day on normal formula feeds to 5.8 g/kg/day on high energy feeding. It is recommended that infants with CHD known to be associated with FTT are fed on high energy diets from the time of diagnosis in order to optimize growth ***(Jackson & Poskitt, 1991).***

5. Obstructive sleep apnea syndrome (OSAS)

FTT is a common complication of childhood OSAS. To further evaluate its cause, a study was done including anthropometric measurements, and measurements of energy expenditure during sleep (SEE) in children with OSAS before and after tonsillectomy and adenoidectomy, in 14 children. In this study, the SEE in children with OSAS decreased after resolution of the OSAS. This decrease in SEE was accompanied by an increase in Wt. Caloric intake remained constant before and after tonsillectomy and adenoidectomy. Therefore it is unlikely that anorexia or dysphagia are major factors influencing growth in children with OSAS. These data indicate that growth is decreased in children with OSAS because of increased caloric expenditure, presumably resulting from increased work of breathing during sleep ***(Marcus et al., 1994).***

6. Hyperthyroidism

Hyperthyroidism represents the protype for failure to thrive due to hypermetabolism. Wt loss and restlessness or emotional lability may be the presenting complaints, and unless proptosis draws attention to the possibility of Graves disease, the FTT may become quite severe before the hyperthyroidism is suspected. Children with hyperthyroidism may have advanced skeletal maturation in addition to their Wt loss ***(Mihailovic et al., 1980).***

7. Diabetes mellitus

Insulin deficiency in diabetes mellitus, leads to catabolic state, producing rapid Wt loss in addition to dehydration from the polyuria ***(Rosenbloom et al., 1981).***

V- End organ unresponsiveness

FTT which may have its origin in end organ unresponsiveness, is the most varied and most nebulous of the causes of FTT ***(Marvin & Rallison, 1986).***

1. Chronic lung diseases

FTT in chronic lung disease is attributed to tissue anoxia, although infection, anorexia, increased metabolic needs of muscles of respiration, and acid-base disturbances may also contribute to end organ unresponsiveness and FTT. Although recent interest in the growth of asthmatic children has centered on growth failure as a potential side effect of inhaled corticosteroid treatment, it has long been recognized that asthma itself can impair growth ***(Price, 1993).***

There are several possible explanations for the association between asthma and poor growth:

(a) ***Impaired GH secretion:*** it is tempting to speculate that asthmatic children, who commonly suffer from night time symptoms with consequent sleep disturbance, might have impaired nocturnal GH secretion. ***(Russel, 1993).***

(b) ***The severity of the asthma:*** it may influence growth through hypoxaemia and impaired pulmonary function ***(Ninan & Russel, 1992).***

(c) ***Decreased appetite:*** asthma, in common with most systemic disorders, may affect appetite ***(Zeitlin et al., 1992).***

(d) ***Increased energy demands:*** it would not be surprising if the diversion of energy resources to maintain the enhanced metabolic demands of

increased work of breathing, were to result in growth impairment ***(Zeitlin et al.,1992).***

2. Renal diseases

Children with chronic renal failure often fail to ingest adequate amounts of food or protein due to anorexia, leading to PEM. However, even with correction of dietary deficiencies, normal growth may not be achieved. Acidosis has a retarding effect on growth and appears to be the major cause of FTT in chronic renal insufficiency ***(Klahr et al., 1988).***

3. Chronic anemia

(a) Iron deficiency anemia:

Tissue hypoxia is the main cause of FTT in chronic anemia. There is a close relationship between iron deficiency and Wt gain. The Wt, stature, alertness and learning of both infants and adolescents are usually affected in iron deficiency anemia ***(Nathan & Oski, 1987).***

(b) *Thalassemia major:*

Body growth retardation in children with thalassemia major constitutes one of the significant medical and social problems of this disease, and has been studied by several investigators in recent years. Most authors believe that the retardation of body growth must be associated with the toxic effect of

iron overload, and in an early publication a negative correlation was found between ferritin levels and Ht. Another possibility could be that the growth retardation is due to the chelating agent itself. GH estimations in thalassemic patients with growth retardation, either by stimulation tests or by a 24-hour profile of GH secretion, have in general been reported as normal ***(Pantelakis, 1994).***

4. Fetal environmental toxic syndromes

In utero, insults from viral infections or from maternal ingestion of alcohol, drugs, or maternal cigarette smoking, seem to affect fundamental growth regulating processes and render tissues thereafter incapable of normal growth ***(Manning et al., 1982).***

5. Endocrine disorders

(a) Congenital hypothyroidism :

Development is usually retarded. Hypothyroid infants appear lethargic and are late in learning to sit and stand. The voice is hoarse, and they do not learn to talk. The degree of physical and mental retardation increases with age. Sexual maturation may be delayed or may not take place at all ***(Fisher, 1991).***

Early recognition and treatment of newborns and infants with central hypothyroidism of hypothalamic origin, is important to maximize the potential for growth and development ***(Jain et al., 1994).***

(b) Congenital hypopituitarism :

It is characterized by growth failure, microphallus and hypoglycemia ***(DiGeorge, 1968)*** .

(c) Congenital thymic aplasia : (DiGeorge's Syndrome)

DiGeorge's syndrome is one of the few immunodeficiency disorders associated with symptoms immediately following birth. The complete syndrome consists of the following features :

- Hypoparathyroidism, which is diagnosed by the demonstration of low serum calcium levels, elevated serum phosphorus levels, and an absence of parathyroid hormone.

-Abnormal facies, consisting of low-set ears, fish-shaped mouth, hypertelorism, notched ear pinnae, micrognathia, and an antimongoloid slant of eyes.

- Congenital heart disease.

- Cellular immunodeficiency, lymphopenia reflects a decreased number of T-cells. Patients who survive the immediate neonatal period may then develop recurrent or chronic infection with viral, bacterial, fungal, or protozoal organisms. Pneumonia, chronic infection of the mucous membranes with Candida, diarrhea and FTT ***(Carbonari, 1990).***

6. Mucopolysaccharidoses

Mucopolysaccharidoses are a group of inherited disorders caused by incomplete degradation and storage of acid mucopolysaccharides. The clinical manifestations result from the accumulation of mucopolysaccharides in various organs. Specific degradative lysosomal enzyme deficiencies have been identified for all the mucopolysaccharides. FTT is a common feature of the children affected with this disease ***(Matalon, 1983).***

DIAGNOSIS OF FAILURE TO THRIVE

1. History.

(a) Medical history :

The diagnosis of FTT is complex, because of the many factors that affect a child's growth. History may provide clarification of whether inadequate intake, increased losses from vomiting and diarrhea, or disturbed food utilization is the mechanism leading to the growth failure. Frequently, the mechanism is unclear, but information gathered from other observers and through repeated interviews may reveal unsuspected adverse factors in the child's environment ***(Barbero & shaheen, 1967).***

(b) Family history :

The heights of parents and grandparents, and heights and weights of siblings may indicate genetic growth aberration or some factor common in several family members ***(Powell et al., 1987).***

(c) Social history :

Social history may be helpful in determining not only economic status and availability of resources, but also allows an estimation of time and family interest given to the child. Since in most recent series, NOFTT is the most common cause of FTT, it is prudent to seek historical evidences of abnormal

maternal-child interaction as an affirmative diagnosis, rather than relegating it to a diagnosis of exclusion. Children with NOFTT tend to be the youngest in the family, born within 18 months of the next oldest sibling and manifest growth failure before their first birthday. They often come from single parent, low income homes, and present with vomiting and diarrhea ***(Freyer, 1988).***

(d) Dietary history :

Caloric intake from a careful one day feeding is estimated. A history of massive intake with no growth usually indicates that the informant is fabricating or is ignorant of intake, although excessive losses cannot be excluded. It is important to recognize that parents will seldom admit that they feed an infant infrequently, or that they restrict severely the amount offered at each feeding ***(Powell et al., 1987).***

2. Physical examination.

(a) Growth charts :

Constructing and studying both a growth chart and a developmental flow sheet may identify when the child began failing to thrive, and may help uncover the environmental or physical factors responsible ***(Barbero & Shaheen, 1967).***

(b) Anthropometric measurements :

Accurate Ht and Wt measurements are essential. Some parents complain that their children are not growing properly. If the history and physical examination are within normal limits, and the child falls within the lower percentiles of growth charts for Ht and Wt, explanation of normal growth patterns may reassure parents and the child. Some parents who themselves are short may expect excessive growth of their normal children. Explanation of genetic factors and normal growth rates leads to realistic expectations of growth without excessive investigations ***(Drotar, 1984).***

In the young child, HC can give valuable clues to diagnosis. In caloric deprivation, Wt is lost first, then linear growth slows, and finally HC is small for age, this indicates either severe malnutrition or primary failure of development of the skull or brain ***(Berwick et al., 1982).***

(c) Systemic physical examination :

A careful systemic physical examination further leads to detection of diseases in body systems, anyone of which can cause FTT. Blood pressure should not be forgotten as a screening test for acute or chronic renal disease ***(Drotar, 1984).***

3. Observation.

An initial period of observed nutritional rehabilitation, usually in a hospital setting, is often helpful in the diagnosis of FTT. Hospitalizing the child provides an opportunity for quantitating factors governing the net caloric intake (food intake, vomiting, stools) and for observing the child's interactions, especially during feeding and play, with parents, health personnel, and other children. Hospitalization frequently leads to dramatic improvement in Wt gain and in social responses and thus provides evidence that environmental factors are causative, eliminating the need for searching further for underlying organic disease ***(Freyer, 1988).***

4. Laboratory investigations.

(a) Initial methods :

Initial laboratory investigations at the time of admission might be limited to blood, urine, stool examination and tuberculin testing. For ***blood***, we do complete blood count and sedimentation rate. For ***urine***, complete analysis should be done including *pH*, which when high in the presence of acidosis suggests renal tubular insufficiency; *specific gravity*, which if low may indicate diabetes insipidus; *glucose*, which usually indicates diabetes. As regards the ***stools***, it should be examined for fat, parasites and occult blood. Stool culture is done to detect infections. ***Tuberculin test*** can detect tuberculosis which might be unexpected ***(Berwick et al., 1982).***

(b) Screening tests :

Table (12)
Some causes of Failure to Thrive and screening tests
(Barbero, 1967)

Cause	Screening test
NON-ORGANIC	
-Inadequate caloric intake	History; observation in hospital.
-Emotional deprivation	History; observation in hospital.
-Gastroesophageal reflux	History; observation in hospital.
-Anorexia nervosa & bulimia	History; examination.
ORGANIC	
-Central nervous system (abnormalities & infections)	Neurodevelopmental assessment; brain scan.
-Gastrointestinal system (malabsorption, cystic fibrosis, inflammatory bowel disease, megacolon, liver disease)	Stool examination; sweat test; liver functions; barium swallow; ESR.
-Chronic heart failure	Physical examination; chest X-rays; echocardiography.
-Endocrine disorders	Growth chart; thyroid function; blood tests; bone age.
-Pulmonary disease (bronchopulmonary dysplasia, bronchiectasis)	Physical examination; chest X-rays; tuberculin test; pulmonary function tests.
-Renal disease (anomalies, infection, renal failure)	Urine analysis; urine pH; ultrasound; blood urea nitrogen.
-Chromosomal disorders (Down syndrome)	Chromosomal analysis; identification of peculiar facies & cong. defects.
-Chronic infection (tuberculosis, congenital)	Tuberculin test; specific laboratory tests to identify infectious agent.
-Chronic inflammation (juvenile rheumatoid arthritis)	Physical examination; ESR.

MANAGEMENT OF FAILURE TO THRIVE

FTT is categorized as organic (OFTT) or nonorganic (NOFTT). Traditionally, it has been taught that children with OFTT are unable to grow well in spite of adequate care (calories, nurturance, medical supervision), whereas NOFTT children will grow well when given adequate care. A study was done to determine if NOFTT and OFTT children could grow at similar rates when treated by a specialized multidisciplinary team that provided concrete, individualized therapies including psychological support, medical care and hypercaloric diets. OFTT children grew just as well as NOFTT children when given adequate calories for catch-up growth. These data indicate that Wt gain alone cannot reliably differentiate OFTT from NOFTT, as has been traditionally taught. The study suggests that every attempt should be made to maximize caloric intake in FTT children and that a multidisciplinary team consisting of a pediatrician, child psychiatrist, nutritionist, nurse clinician, and social worker may be succesful in managing FTT children ***(Bithoney et al., 1989).***

NON-ORGANIC FAILURE TO THRIVE

A study was done to determine the effect of two interventions, ***Caloric Management and Socioemotional Growth Fostering***, on (a) the Wt of children aged 1 to 3 years with NOFTT and (b) the interaction behaviors of 10 mother-child dyads. Caloric management, focusing on nutritional intake, seemed to enhance current mother-child conflicts. The Socioemotional Growth Fostering Intervention, focusing on healthy mother-child interaction

behaviors, produced a greater Wt gain in the children and growth enhancing behaviors in the mothers ***(Sullivan, 1991).***

FTT is a chronic symptom accounting for 1% of all patients admitted to pediatric hospitals. FTT, which is traditionally attributed to OFTT and/or NOFTT causes, results in undernutrition. Undernutrition has potentially serious effects on child development, behavior, and cognitive skills. ***Bithoney et al. (1991),*** undertook a study of children with FTT to determine whether ***multidisciplinary team treatment*** resulted in improved Wt gain compared with children treated in a ***primary care clinic (PCC)***. Children followed in the multidisciplinary team clinic grew better than did children in the PCC ***(Bithoney el al., 1991).***

Cognitive-behavioral treatment of health-impairing food phobias in three boys (ages 6 to 8 years) was reported. The three boys were hospitalized because of Wt loss and malnutrition, caused by severe dietary restriction and/or refusal to eat solid food. Psychological, behavioral, and medical assessments indicated that the boys were of average intelligence, without other significant psychological or medical disorders. Their eating disturbances were conceptualized as phobic disorders maintained by family factors reinforcing the children's avoidant behaviors. Treatment positively affected overall caloric intake, Wt gain, number of solid foods accepted, and incidence of emesis ***(Singer et al., 1992).***

Mothers of children with severe defects or disease should be directed and assisted in providing maternal care in as normal and full manner as possible ***(Barbero & Shaheen, 1967).***

Treatment of protein-energy malnutrition requires gradual increase in calories and gradual introduction of protein. Realimentation in a marasmic child may be accompanied by no Wt gain for as long as 6 weeks, presumably due to shifting of water between intracellular and extracellular spaces. During the phase of rapid Wt gain, the liver may enlarge due to fat accumulation, and signs if increased intracranial pressure may occur. These are usually temporary and require no treatment ***(Powell, 1988).***

The process of refeeding to promote catch-up growth must be undertaken with caution. If high intakes are provided at the beginning of nutritional resuscitation, severely malnourished children may develop vomiting, diarrhea and circulatory decompensation. To minimize these complications, such children should be restricted to the normal dietary intake for age for the first 7 to 10 days of treatment. Feeding during this period should consist of small, frequently offered portions. Over the next week, the child may be gradually advanced to a diet that meets the calculated requirements for catch-up growth ***(Peterson et al., 1984).***

$$\text{Daily caloric needs/kg} = \frac{120/\text{kg} \times \text{median weight for current height}}{\text{current weight (kg)}}$$

Underfeeding because of ***calorically inadequately formula*** is treated by increasing fluid and caloric intake and/or instructing the mother about infant feeding skills will produce gratifying results ***(Barbero & Shaheen, 1967).***

Breast feeding should be promoted via pediatrician, obstetrician, hospital staff, radio, television programmes, and the use of posters in government hospitals and clinics. Confidence is the single most important determinant of succesful breast feeding. If the mother thinks she will succeed, she most likely will. The mother's fears and anxieties associated with breast feeding should be discussed, and a psychological push to the mother will definitely promote the process of breast feeding ***(Cunningham, 1977).***

Inadequate breast milk supply is corrected by complementation or supplementation. Complementary feeding is particularly needed in the mid-day feeds, the bottle must be given after the breast and never before, this ensures that the breast is completely emptied and hence can be refilled again. Complementary feeding should never be too sweet otherwise the infant will refuse the breast. In supplementary feeding, we replace a breast feed by animal milk ***(Gaull, 1989).***

Anorexia nervosa is a devastating illness for the patient and family, and requires psychotherapy. Before proceeding with psychiatric therapy, it is important to exclude the possibility of hypopituitary cachexia, chronic infection or malignancy, which may produce emaciation and the symptom complex of anorexia. If the emaciation is extreme and life-threatening,

hospitalization will be required to reverse the Wt loss. The treatment may include behavioral modification, forced nasogastric tube feeding, hyperalimentation and psychopharmacotherapy with antidepressants, chlorpromazine, cyproheptadine or diphenylhydantoin ***(Palla & Litt, 1988).***

ORGANIC FAILURE TO THRIVE

Congenital stenosis, atresia of the esophagus or external compression can be treated by surgical interference. Excellent response is manifested by disappearance of vomiting, improvement in dysphagia and Wt gain. Pneumatic dilatation may produce similar results as surgical esophagomyotomy without the immediate operative morbidity, cost and potential long-term side effects ***(Nakayama et al., 1987).***

Gastroesophageal reflux is a common cause of chronic vomiting and FTT in infants. Without treatment, symptoms in a majority of children ameliorate by age two as they assume an upright position for eating. Keeping the child upright during and after feedings, and burping carefully may be sufficient treatment for mild cases. Most of the remaining children improve spontaneously by four years. Thickened foods are also helpful. Use of antacids for esophagitis may be necessary ***(Jolley et al., 1980).***

Table (13)
Treatment of congenital anomalies of the digestive tract (Nakayama et al., 1987)

Congenital anomaly	Treatment
-Esophageal duplication & cysts	Surgical removal.
-Gastroesophageal reflux	Maintaining the infant in an upright position during feeding & medical treatment.
-Hiatal hernia	Feeding thickened food & surgery
-Vascular rings	Surgical correction.
-Esophageal atresia and tracheo-esophageal fistula	Surgical repair.
-Congenital pyloric stenosis	Correction of dehydration and associated electrolyte disturbances, followed by pyloromyotomy.
-Congenital duodenal obstruction	Nasogastric suction, electrolyte replacement & surgical correction.
-Malrotation	Surgical correction.
-Congenital jejunoileal obstruction	Surgical correction.
-Gastrointestinal duplication	Resection and primary anastomosis.
-Congenital megacolon	Colostomy proximal to the aganglionic segment.

In ***Celiac disease***, wheat, rye, and barley should be eliminated from the diet; many patients tolerate small quantities of oats. Although disaccharidase activities in the mucosa are diminished during active celiac disease, significant disaccharide intolerance is rare. A few patients who have definite lactase deficiency will benefit from a short period of disaccharide restriction. During the early months of therapy extra fat soluble vitamins are advisable, and for those who are iron-or folate-deficient appropriate supplements should be given. Lifelong dietary treatment is a major undertaking and best carried out with the help of an experienced nutritionist and ample written instructions and recipes ***(Swinson et al., 1983).***

Four children with ***cystic fibrosis***, ranging in age from 10 to 40 months, were admitted to a specialized pediatric unit for evaluation and treatment of malnutrition. All were below the fifth percentile for Wt despite appropriate pancreatic enzyme replacement and outpatient nutritional counseling. Dietary evaluation revealed oral intake of 48% to 62% of that required for growth. Standardized nursing and psychological assessments of feeding behaviors during meals indicated a low acceptance rate of foods and a high rate of maladaptive feeding behaviors. Treatment consisted of behavioral management using positive reinforcement of food acceptance, extinction of negative behaviors, and parent training. Mean percentage of caloric intake increased from 54% to 92% for the four patients. At long-term follow up, the patients who continued the program demonstrated substantial and persistent catch-up growth. Behavioral feeding disorders may contribute to FTT in patients with cystic fibrosis, and must be considered when growth failure occurs despite correct medical management ***(Singer et al., 1991).***

In case of ***gastro-intestinal allergy***, the offending allergen should be removed from the diet. Calcium supplements may be required for an infant who must use milk substitutes indefinitely ***(Atkins & Metcalfe, 1984).***

No treatment is entirely effective in ***Crohn's disease.*** Corticosteroids are most effective for treating active disease. Once begun, systemic corticosteroid therapy should be given, in a full dose for 6 weeks, then tapered by 5 mg/24hr each week and replaced with sulfadiazine in a dose of 50-75 mg/kg/24hr ***(Kleinman et al., 1989).***

In patients who fail to respond to medication, total parenteral nutrition may be necessary. Surgery must be performed for massive uncontrolled bleeding or toxic megacolon, but elective surgery for removal of affected bowel offers limited relief. New medications such as cyclosporine, azathioprine, and 6-mercaptopurine continue to be subjected to clinical trial but have no proven benefit ***(Michener & Willie, 1990).***

Growth retardation may be expected from corticosteroid administration, but catch-up growth should occur once the colitis is controlled and the steroids discontinued. Total colectomy generally restores the growth to normal, and may allow catch-up growth if performed before pubertal events limit achievable growth ***(Korelitz, 1990).***

FTT in infants with ***congenital heart disease*** can be attributed to their low energy intakes and high resting energy expenditures. It is recommended

that infants with CHD known to be associated with FTT, are fed on high enegy diets from the time of diagnosis in order to optimize growth ***(Jackson & Poskitt, 1991).***

To determine an effective nutritional regimen for management of growth failure in infants with CHD and congestive heart failure, ***Schwartz et al. (1990),*** reported that continuous 24-hour nasogastric alimentation is a safe and effective method for achieving both increased nutrient intake and improved overall nutritional status in these infants ***(Schwatz et al., 1990).***

The catabolic state of ***diabetes mellitus*** is corrected by the use of insulin injections, together with nutritional management. The caloric mixture of the child should comprise approximately 55% carbohydrates, 30% fat, and 15% protein. In general, it is recommended that approximately 70% of the carbohydrate content be derived from complex carbohydrates such as starch, and that intake of sucrose and highly refined sugars must be avoided ***(Menon & Sperling, 1988).***

Hypothyroidism is another example of FTT due to endocrine disorders. Whatever the cause of hypothyroidism, replacement therapy with thyroid hormone is indicated and effective. Levels of both thyroxine and thyroid stimulating hormones should be monitored and maintained within the normal range ***(Fisher, 1991).***

Of the effective preparations available for symptomatic treatment of ***diabetes insipidus*** is desmopressin (1-desamino-8-D-arginine vasopressin; dDAVP), a highly effective analogue of vasopressin. It is given by a nasal tube delivery system that delivers precise amounts to the nasal mucosa. The usual dose ranges from 5 - 15 ***u***g given either as a single dose or divided into two doses. Children under two years of age require smaller doses (0.15 - 0.5 ***u***g/kg/24hr ***(Lee et al., 1976).***

Inborn errors of metabolism are important causes of FTT due to end organ unresponsiveness. A significant number, of once hopeless cases, can now be treated as shown in table 13 ***(Scriver et al., 1989).***

Table (14)
Some errors of metabolism susceptible to treatment
(Scriver et al., 1989)

Disease	Treatment
Aminoacid metabolism:	
- Phenylketonuria	- Phenylalanine restricted diet.
- Maple syrup urine disease	- Diet restricted in leucine, isoleucine, and valine.
- Homocystinuria	- Vitamin B and cystine supplement, diet restriction in methionine.
- Histidinemia	- Histidine restricted diet.
- Tyrosinemia	- Diet restricted in phenylalanine and tyrosine.
- Cystinosis	- Diet restricted in methionine and cystine.
- Cystinuria	- Alkali, high fluid intake, D-penicill-amine.
Carbohydrate metabolism:	
- Galactosemia	- Galactose - free diet.
- Fructosemia	- Fructose - free diet.
- Malabsorption of diet and monosaccharides	- Monosaccharide - free or disaccha-ride - free diet.
Other metabolic errors:	
- Wilson's disease	-D-penicillamine, potassium sulfide, copper - restricted diet.
- Primary hemochromatosis	- Removal of excess iron stores by re-peated phlebotomy, desferroxamine.
- Pyridoxine deficiency	- High doses of pyridoxine.
- Familial hyperlipoproteinemia	- Fat restriction, use of medium chain fatty acids, cholesteramine.
- Familial defective synthesis of thyroid hormone (familial goiter)	- L-thyroxine or dessicated thyroid.
- Adrenogenital syndrome	- Cortisone, mineralocorticoids in pa-tients subject to salt loss.

CHILD PSYCHIATRY

The practice of child psychiatry differs from that of adult psychiatry in several important ways. It is seldom that the child initiates the consultation with the clinician. Instead he is brought by adults (usually the parents) who think that some aspect of behavior or development is abnormal. Much depends on the attitudes and tolerance of these adults, and how they percieve the child's behavior. Healthy children may be brought to the doctor by over-anxious parents or teachers, while in other circumstances severely disturbed children may be left to themselves ***(Illingworth, 1980).***

The practice of child psychiatry differs from adult psychiatry in two other ways. First, children are generally less able to express themselves in words. Evidence of disturbance is therefore based more on observations of behavior made by parents, teachers, and others. The assessment of these accounts requires skills in taking a developmental history, assessing behavior, evaluating the emotional involvement of informants, and understanding the home and school background. Second, in the treatment of children, less use is made of medication or other methods of individual treatment than in adult psychiatry. Instead the main emphasis is on changing the attitudes of parents, reassuring and retraining the child, and co-ordinating the efforts of others who can help him especially at school ***(Rutter, 1980).***

Normal development

The practice of child psychiatry calls for knowledge of the normal process of development from a helpless infant to an independent adult. In order to judge whether any observed emotional, social or intellectual functioning is abnormal, it has to be compared with the corresponding normal range for the age group ***(Illingworth, 1980).***

The first year of life

This is a period of rapid development of motor and social functioning. Three weeks after birth, the baby smiles at faces; selective smiling appears by six months, fear of strangers by eight months, and anxiety on separation from the mother shortly after. Although bonding to the mother is most significant, important attachments are also made to the father and other people who are close to the infant. Recent research has stressed the reciprocal nature of this process and the probable importance of the very early contacts between mother and newborn infant in initiating bonding ***(Boddy & Skuse, 1994).***

By the end of the first year, the child should have formed a close and secure relationship with the mother. There should be an ordered pattern of eating and sleeping. By the end of the first year, the child enjoys making sounds and may say "mama", "dada", and perhaps one or two other words ***(Bowlby, 1980).***

Year two

This is a period of rapid development. The child begins to wish to please the parents, and appears anxious when they disapprove. He begins to learn to control his behavior. By now, attachment behavior should be well established. Temper tantrums occur, particularly if exploratory wishes are frustrated. These do not last long, and should lessen as the child learns to accept constraints. By the end of the second year he should be able to put two or three words together as a simple sentence ***(Rutter, 1980)***

Preschool years (2 to 6 years)

This phase brings a rapid increase in intellectual abilities, especially in the complexity of language. Social development occurs as the child learns to live within the family. He begins to identify with the parents and to adopt their standards in matters of conscience. Temper tantrums continue, but diminish and should disappear before the child starts school. At this age, the child has much curiosity about the environment, and may ask a great number of questions. The child aged 2 to 6 begins to learn about his own sexual identity. He realizes the differences between males and females in their appearance, clothes, behavior, and anatomy. Sexual play and exploration are common at this stage ***(Zeitlin, 1986).***

Middle childhood (6 to 10 years)

By the age of 6 years, the child should understand his or her identity as boy or girl, and his position in the family. He has to learn to cope with school, and to read, write, and acquire numerical concepts. The teacher becomes an important person in the child's life. At this stage, the child gradually learns what he can achieve and what are his limitations. Defense mechanism, conscience, and standards of social behavior develop further. According to psychoanalytic theory, this is a period in which psychosexual development is quiescent (the latent period). This notion has been questioned and it now seems that in the 6 to 10 years old period, sexual interest and activities are present, although they may be concealed from adults ***(Rutter, 1971)***

Child psychiatry in developing countries

Significant growth in child psychiatry in developing countries has occured only in the last 20 years, despite the fact that children under 15 years of age constitute 40-50% of the population of these countries. Evidence of the substantial morbidity arising from mental health problems, and of the significance of the psychological factors to childhood morbidity has, however, only recently become available ***(Olawatura, 1978).***

Developing countries are less industrialized than developed countries, with limited resources for service development. Populations are relatively young and often predominantly rural. Many countries have established traditional cultures which affect beliefs and life-styles. Changes in traditional

values due to economic, political and educational influences, lead to considerable diversity within and between countries ***(Minde, 1976).***

Epidemiological issues

The importance of epidemiology is related to demonstrating presence of, and need for services for, mental health problems, the revelance of these problems to major causes of mortality and morbidity such as malnutrition and infections, and the identification of causal links and preventive measures. There are few data from community-based epidemiological studies compared with developed countries. World Health Organization (WHO) case studies done in a number of countries which included Egypt, Nigeria, India, Indonesia, Thailand and SriLanka illustrated that child psychiatric symptomatology does not differ to a significant extent across cultures ***(Nikapota, 1991).***

Factors affecting child psychiatric disorders

(a) Organic factors:

The association between levels of cognitive development and conditions such as chronic malnutrition, anemia and mental retardation, highlights the need for further study on the effect of organic factors on the child's ability to interact with and make demands from its natural environment ***(Agarwal et al., 1989).***

(b) Individual and environmental differences:

The higher rates observed in urban samples may indicate that influences of urban living increase the vulnerability of children, despite possible advantages such as easier access to health care and educational facilities. Differences in family life-style and employment patterns; changes in parental expectations, and attitudes to and tolerance of behavior, educational and academic pressures; and an increased level of family stress, are all factors related to urban living that have been implicated as causing increased rates of disturbance in children ***(Minde, 1975).***

(c) Family factors and child-rearing practices:

There has always been an assum that the extended family is protective with regard to emotional and behavior problems in children, particularly where surrogate parenting is required due to separation from parents, or marital disruption. On the other hand, intergenerational conflict of values can be associated with a higher rate of problems ***(Kong et al., 1988).***

Marital conflict, separation and divorce are associated with increased problems in children. Separation or divorce may lead to social isolation of a mother or disruption of extended family networks. Clinically, these mothers are often observed to be depressed, leading to poor levels of care for their children ***(Sartotius & Graham, 1984).***

It is important for developing countries to identify facilitatory as well as maladaptive mechanisms within current child care patterns. Strengthening potentially facilitatory practices and community education to alter potentially maladaptive ones is a part of primary prevention. Close physical contact with mother from infancy is cited as one positive factor ***(Cederblad, 1988).***

Recent work on the relevance of caring styles and feeding practices, not only to early child development, but also to important physical health problems in the young child as malnutrition, has high-lighted the importance of bonding and a responsive carer-child interaction ***(Heptinstall et al., 1987).***

Carers often do not appreciate the degree to which children react to events within a family, or the child's need to express these feelings. Families may behave in a way that denies, rather than aknowledges, the events and the feelings aroused, to reduce distress in the child. This practice is clinically associated with problems in children. For example, sibling rivalry is aknowledged by parents but children are often actively discouraged from expressing these feelings, which are considered socially inappropriate. Sibling rivalry leading to behavior problems is commonly encountered clinically ***(Yu-Feng et al., 1989).***

(d) Parental education:

Higher educational levels among parents was one of the factors significantly associated with greater understanding of a child's problem ***(Resnick et al., 1988).***

Risk groups

Child abuse is an issue of concern, which in developing countries has received relatively little attention. There is often a belief that abuse does not occur. This is not so, although the availability of the extended family as well as the relative lack of privacy within families may serve as a protection against physical abuse of the baby or young child. Deliberate neglect of unwanted children because of illegitimacy or severe disability has been known. There is some evidence from previous studies that the group at risk of abuse are older children ***(Lieh-Mak et al., 1983).***

A number of developing countries are currently faced with situations of violence and conflict, due to repressive political systems or terrorist action. The cost to children of these situations is immense ***(UNICEF, 1989).***

Services, clinical strategies and interventions

Parents may find it difficult to discuss family problems with a professional whom they view as an outsider, or in front of their children.

Where cultural values or beliefs lead to resistance, clinicians need to develop strategies to overcome them, since imposition of an alien approach will only lead to loss of contact with the family. Children may require help to express feelings about family factors, particularly in cultures where the verbalizing of such opinions by children is considered very inappropriate ***(Minde & Musisi, 1990).***

There are certain factors which influence the choice of treatment and outcome. Parents often expect a particular treatment such as medication, as this is associated with quick results. Compliance with therapy which involves behavior change, may be affected if adequate explanation and reassurance is not given by the clinician. Many parents may find frequent clinic attendances difficult to maintain, due to economic factors; families may have to travel considerable distances and cope with difficult transport conditions ***(Krahl et al., 1981).***

The identification and evaluation of effective strategies in child psychiatry is far of satisfactory. There are few effective treatments for specific conditions, or criteria developed to guide clinicians in the selection of the best mode of treatment ***(Van Engeland, 1989).***

Training in child psychiatry

The potential role of the child psychiatrist and the need for child psychiatric expertise at various levels of health care, have implications for training at an undergraduate and postgraduate level. Most undergraduate

schools have some psychiatric input, though not always specific content with regard to child psychiatry. There may be a reluctance among the traditional specialities of medicine and surgery to give equal weighting to pediatric and psychiatric courses ***(Barlett, 1989).***

Training in child psychiatry for clinicians working in developing countries is an issue of some importance. Child psychiatric work in the very near future, in most developing countries, will be done not only by child psychiatrists but by pediatricians, general psychiatrists, and psychologists. Many countries have postgraduate training in psychiatry and pediatrics, which may therefore require review and revision with regard to child psychiatric content. There is a place for regional initiatives with inputs from specialised centers in other countries, and this would be a useful form of collaboration between developed and developing countries ***(Checkoway & Orley, 1990).***

Conclusion

Child psychiatry in developing countries is now an established field. These countries are subject to rapid socio-cultural and political changes which affect the life-styles of children and their families, and hence their physical and emotional well-being. The need for child psychiatry is highlighted by established links between psychological factors and physical health, and existing morbidity among children due to mental health problems. Child psychiatric skills and expertise cannot therefore be restricted to sparse centers at a secondary or tertiary level. Child psychiatry can also contribute to changes in social policy about children ***(Nikapota, 1991).***

The DSM-III-R classification of psychiatric disorders in children and adolescents

- **Mental retardation.**
- **Pervasive developmental disorders.**
    * Childhood autism
- **Specific developmental disorders.**
    * Language and speech disorder
    * Motor skill disorder
    * Other developmental disorders
- **Disruptive behavior disorders.**
    * Conduct disorder
    * Attention deficit-hyperactivity
- **Emotional disorders.**
    * School refusal (school phobia)
    * Overanxious disorder
    * Separation anxiety disorder
- **Eating disorders**
    * Anorexia nervosa
    * Bulimia nervosa
- **Gender identity disorder.**
- **Other disorders of infancy, childhood and adolescence.**
    * Dementia
    * Schizophrenia
    * Sleep disorders

Child autism

This condition was described by Kanner (1943) who suggested the name infantile autism, which is still widely used. The prevalence of autism is probably about 30-40 per 100 000 children. It is four times as common in boys as in girls ***(Rutter, 1985).***

Etiology

The cause of autism is unknown. Genetic influences are probable since the condition is more frequent in the families of affected persons than in the general population. Organic brain disorder is suggested by an increased frequency of complications of pregnancy and childbirth, and by an association with epilepsy ***(Hermelin & O'Connor, 1983).***

Clinical features

Autistic aloneness is the inability to make warm emotional relationships with people. Autistic children do not respond to their parent's affectionate behavior by smiling or cuddling. Instead they appear to dislike being picked up or kissed ***(Wing, 1976).***

Speech and language disorder is another important feature. Speech may develop late or never appear. Occasionally, it develops normally until about the age of two years, and then disappears in part or completely. This

lack of speech is a manifestation of a severe cognitive defect, which affects non-verbal communication as well ***(Schopler & Mesikov, 1988).***

Obsessive desire for sameness, for example, autistic children may prefer the same food repeatedly, insist on wearing the same clothes, or engage in repetitive games ***(Schopler & Mesikov, 1988).***

Bizarre behavior and mannerisms are commonly found. Some autistic children engage in odd motor behavior such as whirling round and round, twiddling their fingers repeatedly, flapping their hands, or rocking. Others do not differ obviously in motor behavior from normal children. Autistic children may suddenly show anger or fear without apparent reason ***(Rutter & Lockyer, 1967).***

Treatment

A developmental-educational-psychological assessment of the child and the family is indicated. Skilled special education is needed in a structured and supportive environment. Behavioral treatment has been a useful adjunct for social responses, communication and self - abusive behavior. Neuroleptics (such as chlorpromazine) have been useful in agitation ***(Hermelin & O'Connor, 1983).***

Language and speech disorder

Half of all children use words with meaning by 12.5 months and 97% do so by 21 months. Half form words into simple sentences by 23 months. Vocabulary and complexity of language develop rapidly during the pre-school years. However, when they start school, 1% of children are seriously retarded in speech and 5% have difficulty in making themselves understood by strangers ***(Neligan & Prudham, 1969).***

Speech delay

Etiology

The most common cause of delay in the development of normal speech is mental retardation. Other important causes are deafness and cerebral palsy. Social deprivation can cause mild delays in speaking. Infantile autism is an important but infrequent cause ***(Bishop, 1987).***

Diagnosis

Two categories are recognized in DSM-III-R: receptive and expressive. The former have difficulty in understanding language. The latter appear to understand language but their own speech is hard to understand because the words are ill-formed. Serious speech delay is often accompanied by other problems of development. It has obvious and important consequences for education and social development. Early investigation is

essential and should include both a detailed assessment of speech and language, and a search for one of the causes mentioned above ***(Cantwell & Baker, 1985).***

Treatment

It depends partly on the cause, but usually includes a programme of speech training carried out through play and social interaction. In milder cases, this help is best provided at home by the parents who are given information on what to do ***(Cantwell & Baker, 1985).***

Elective mutism

In this condition, a child refuses to speak in certain circumstances, although he does so normally in others. Usually speech is normal at home but lacking at school. There is no defect of speech or language, only a refusal to speak in certain situations. The condition usually begins between three and five years of age, after normal speech has been acquired ***(Kolvin & Fundulis, 1981).***

Stammering (Stuttering)

Stammering (or stuttering) is a disturbance of the rhythm and fluency of speech. It may take the form of repetitions of syllables or words, or of blocks in the production of speech. Stammering is four times more frequent in boys than girls. It is usually a brief disorder in the early stages of language

development. However, 1% of children suffer from stammering after they have entered school. The cause of stammering is not known, although many theories exist. Genetic factors, brain damage and anxiety may all play a part in certain cases. Stammering is not usually associated with a psychiatric disorder, even though it can cause distress and embarassment. Most children improve whether treated or not. The usual treatment is speech therapy ***(Kolvin & Fundudis, 1981).***

Specific motor disorder

Some children have delayed motor development, which results in clumsiness in school-work or play. In DSM-III-R this condition is called "developmental co-ordination disorder". It is also known as "clumsy child syndrome" or "specific motor dyspraxia". The children can carry out all normal movements, but their co-ordination is poor. They are late in developing skills such as dressing, walking and feeding. They tend to break things and are poor at handicrafts and organized games. They may also have difficulty in writing, drawing and copying. IQ testing usually shows good verbal but poor performance scores. These children are sometimes referred to a psychiatrist because of a secondary emotional disorder. An explanation of the nature of the problem should be given to the child, the family and the teachers. Special teaching may improve confidence. It may be necessary to exempt the child from organized games or other school activities involving motor co-ordination ***(Henderson, 1987).***

Specific reading disorder

In DSM-III-R this condition is called "developmental reading disorder". It is defined by a reading age well below the level expected from the child's age and IQ ***(Yule & Rutter, 1985).***

Etiology

Etiology is unknown. The frequent occurrence of other cases in the family suggests a *genetic cause*, but evidence is lacking. Because children with cerebral palsy and epilepsy have increased rates of specific reading disorder, it has been suggested that children with specific reading disorder but no obvious neurological disease, might have *minor neurological abnormalities*. *Social factors* may add to these psychological problems among children brought up in a large family or poor school, where they receive little personal attention ***(Yule & Rutter, 1985).***

Clinical features

Specific reading disorders should be clearly distinguished from general backwardness in scholatic achievement owing to low intelligence or inadequate education. The child presents with a history of serious delay in learning to read, sometimes preceded by delayed acquisition of speech and language. Writing and spelling are also impaired, but development in other areas is not ***(Gittelman, 1985).***

Treatment

It is important to identify the disorder early. Assessment is carried out by an educational or clinical psychologist. Treatment is educational unless there are additional medical or behavioral problems requiring separate intervention ***(Gittelman, 1985).***

Specific arithmetic disorder

In DSM-III-R this condition is called "developmental arithmetic disorder". It is probably the second most common specific disorder. Little is known about it. Although it causes less severe handicap in everyday life than reading difficulties, it can lead to secondary emotional difficulties while the child is at school. The causes are uncertain. The existence of mathematical prodigies suggests that some of the abilities needed for mathematics might be inherited. Treatment is by remedial teaching, but it is not known whether it is effective ***(Quay & Werry, 1986).***

Conduct disorders

Diagnosis

Problematic parent-child relationships of varying severity are involved in these cases. The parents or outside agency (e.g. school) usually present complaints of lying, fighting, truancy, stealing and/or fire setting. The

behaviors, occuring more frequently in boys than in girls, are persistent, troublesome, and socially disapproved. Learning disabilities are frequently associated, along with a history of temperament extremes ***(Graham & Stevenson, 1987).***

Treatment

Prevention is important. Early prenatal education about the temperamentally difficult infant may be helpful. Counseling and advice should be offered around firm and consistent discipline techniques. Referral to a child psychiatrist is recommended for the child who is persistently out of control. Treatment is most helpful when the problems are responses to emotional difficulties ***(Graham & Stevenson, 1987).***

Attention deficit hyperactivity disorder (ADHD)

Etiology

The cause is thought to be related to *abnormalities in neurotransmitters* (norepinephrine, dopamine), but the exact nature of these abnormalities remains elusive. The most obvious characteristic of these children is inattention to appropriate stimuli. Some, but not all, of these children are hyperactive ***(Richman et al., 1982).***

Diagnosis

Developmentally inappropriate degrees of inattention, impulsiveness and hyperactivity. Parent and teacher questionnaires are helpful in diagnosis ***(Richman et al., 1982)***

Treatment

Cognitive therapy aimed at teaching these children approaches to increasing attention span, and decreasing distractability and impulsivity, has appeared beneficial. *Stimulant medications*, such as methylphenidate (Ritalin), have been shown to be beneficial in approximately 75% of the children ***(Taylor, 1986).***

School phobia

Etiology

It includes separation anxiety or fear of school, which may reflect real school problems, such as a " class bully", a "difficult teacher" or "difficulty with new school material". School refusal may be a partial manifestation of a more pervasive social withdrawal in all spheres ***(Bernstein & Garfinkel, 1986).***

Diagnosis

The history should emphasize earlier separation problems, previous undefined illnesses with prolonged school absence, absence of symptoms on holidays and weekends or specific school problems that might be contributing to absences. The longer the sustained absence and the older the child, the more serious and urgent the problem is ***(Bernstein & Garfinkel, 1986).***

Treatment

Reassure the child and parents after the physical examination and necessary laboratory tests, that the symptoms do not represent serious disease. Help the family *organize a specific plan* for getting the child to school. Try to ease the child's way back into school by speaking to the principal or teacher about *decreasing any complicating stresses during the first few days* ***(Hersov, 1985).***

Overanxious disorders

Etiology

Excessive states of anxiety may relate to temperamental variation, or chronic environmental stress, or both ***(Vaughan & Litt, 1990).***

Diagnosis

The predominant disturbance is generalized and persistent anxiety or worry lasting at least 1 month, as manifested by at least four of the following:

1. Unrealistic worry about future events.
2. Overconcern about competence (e.g. school, social, athletic).
3. Somatic complaints, such as headaches or stomachaches, with no established physical basis.
4. Excessive need for reassurance about worries.
5. Marked self-consciousness.
6. Marked feelings of tension or inability to relax; shaking, trembling or restlesness.
7. Autonomic hyperactivity such as sweating, heart pounding, dry mouth, diarrhea, high resting pulse and respiratory rate ***(Kovacs et al., 1984).***

Treatment

Anxiety may be responsive to *reassurance* of the child and the parents. *Psychiatric consultation* may be needed if the child is unresponsive to the supportive relationship. Acute anxiety may be helped by *medications* such as benzodiazepines, hydroxyzine or diphenhydramine. The child (usually 7 years or older) can be instructed in the use of *relaxation techniques* such as arranging a quiet and comfortable place, closing the eyes and thinking about "doing nothing", relaxing the head and neck slowly and repeating a single word such as "calm" or "relax" after each exhalation and feel relaxation ***(Vaughan & Litt, 1990).***

Separation anxiety disorder

Etiology

It usually occurs after *death of a parent*, *hospitalization* and its strange environment, *divorce* or maternal deprivation in case of *working mothers* ***(Fine, 1980).***

Diagnosis

Unrealistic and persistent worries of possible harm befalling primary caregivers, reluctance to go to school or to sleep without being near the parents, persistent avoidance of being alone, nightmares involving themes of separation, and numerous somatic symptoms and complaints of subjective distress ***(Beeghly, 1986).***

Treatment

Children are referred for *psychiatric therapy* when the usual supportive approaches have failed to return the child to school or to reduce the symptoms. *Parent training* is often necessary to delineate underlying motivations, and to teach appropriate ways to help the child fulfill reasonable expectations regarding school attendance. A judicious use of *medications* such as antidepressants or antianxiety, is often necessary to facilitate treatment goals ***(Beeghly, 1986).***

Anorexia nervosa and Bulimia nervosa

It has been discussed previously (Page 53).

Gender identity disorders

Some boys prefer to dress in girls' clothes and to play with girls rather than boys. Some have an obvious effeminate manner and say they want to be girls. There is no evidence of any endocrine basis for these behaviors. Possible family influences include the encouragement of feminine behavior by the parents, a lack of an older male with whom the child can identify and a lack of boys as companions in play. Associated emotional disturbance in the child may require help, and it may be useful to investigate and discuss any family behaviors which seem to be contributing to the child's behavior. Effeminate behavior in early childhood may proceed in adult life to homosexuality or bisexuality, transvestism or personality problems ***(Green, 1985).***

Tomboyishness in girls

In girls the significance of marked tomboyishness for future sexual orientation is not known. It is usually possible to reassure the parents, and sometimes necessary to discuss their attitudes to the child and their responses to her behavior ***(Zuger, 1984).***

Dementia

Dementing disorders are rare in childhood. They result from organic brain diseases such as lipoidosis, leucodystrophy, or subacute sclerosing panencephalitis. Some of the causes are genetically determined and may affect other children in the family. Many cases are fatal, others progress to profound mental retardation ***(Graham & Stevenson, 1987).***

Schizophrenia

Schizophrenia is almost unknown before seven years of age, and seldom begins before late adolescence. When it occurs in childhood, the onset may be acute or insidious. Before symptoms appear, many of these children are odd, timid or sensitive, and show delayed speech development. Early diagnosis is difficult particularly when these non-specific abnormalities precede the characteristic symptoms. Antipsychotic drugs are used as in the treatment of adult schizophrenia, with appropriate reductions in dosage. The child's educational needs should be met and support given to the family ***(Tanguay & Cantor, 1986).***

Sleep disorders

The commonest sleep difficulty is ***delayed sleep initiation and maintenance*** which is most frequent between the age of one and two years. About a fifth of children of this age take at least an hour to get to sleep, or are wakeful for long periods during the night. Management depends on a detailed

assessment of the problem and any other difficulties. When wakefulness is an isolated problem and not over-distressing to the family, it is enough to reassure parents about the prognosis. However, if treatment is needed because the sleep problem is seriously exhausting or distressing to the family, the most effective approach is behavioral. Hypnotic medications may be useful for special occasions, but is unlikely to be effective in the long term ***(Richman et al., 1985).***

Other difficulties such as ***nightmares and night terrors*** are quiet common among healthy toddlers, but they seldom persist for long. The etiology is unknown, but there is frequently a positive family history. Episodes occur in the first third of the night, during a transition from deep (stage 3/4) non-rapid-eye-movement (NREM) sleep to another sleep stage. The child is often confused and unresponsive to his or her environment, exhibits autonomic features (tachycardia, sweating, pupillary dilatation), and has retrograde amnesia of the episode. As these episodes are self-limited, often no specific intervention is necessary other than giving an explanation and reassuring the child and the parents. When arousals are disruptive , pharmacologic therapy (benzodiazepines) can be used. Scheduled awakening before the time that the event typically occurs, has been found to be successful in some cases ***(Campbell & Spencer, 1988).***

Material & Methods

MATERIAL AND METHODS

Subjects of the study

One hundred preschool children suffering from FTT, were recruited from the outpatient sections of the medical school of Ein Shams University in Cairo, and from the health clinic in El Katta village in Giza Governorate, where a multidisciplinary rural development survey was conducted there by the National Research Center.

FTT cases were determined by chosing children whose Wt was less than 5th percentile. Fourty normal children free of any symptoms or signs of FTT, have been also recruited from relatives or friends of the diseased children, and were used as control. Age of all children ranged between 2- 6 years.

All cases were subjected to the following:

(I) CLINICAL ASSESSMENT that covered:

. Complaint.

. Symptoms pointing to organic disease.

. Family history.

. Social history to check for the major social "risk factors" that are often associated with family disfunction such as financial difficulties, psy-

chiatric illness or problems with drugs or alcohol, single parent, marital problems, excessive crowding or death in the family.

. Physical examination is an essential screen for organic disease.

(II) ANTHROPOMETRIC MEASUREMENTS that included weight, height, midarm circumference and triceps skinfold.

Wt: the child was weighed in light clothing without shoes. The scale was adjusted to read zero before the child was placed on it. The reading was taken to the nearest 0.01 kg.

HT: the child was standing erect and barefoot, his feet put together with the heels against the wall. A horizontal bar was put to rest on the top of the head. The reading was taken to the nearest 0.5 cm.

MAC: using a flexible non stretchable tape, measurement was taken midway between the acromion process of the scapula and the tip of the elbow. The reading was taken to the nearest 0.1cm.

TSF: using a special caliper, measurement was taken at a point over the triceps muscle midway between the acromion and olecranon processes, on the posterior aspect of the arm; the latter being held vertically with the skinfold running parallel to the length of the arm.

BMI: (Body mass index) it relates Wt and Ht to each other in an attempt to develop a ratio that indicates appropriate Wt for Ht. It is the most common and well accepted ***(Keys, 1972).***

$$\text{BMI} = \frac{\text{Wt (in kilograms)}}{\text{Ht (in cm2)}} \times 100$$

(III) DETERMINATION OF BLOOD PICTURE AND SERUM ALBUMIN

3 ml was withdrawn from each child in a tube containing EDTA. The blood was used to perform the routine complete blood count, and then the plasma was separated by centrifugation and was stored at -20C, until it was used for determination of serum albumin.

Blood picture

It was done using Coulter Counter that included red blood corpuscles (RBCs), hemoglobin concentration (Hb), hematocrite (Hct) and total leucocytic count (WBCs).

Determination of serum albumin (Doumas et al, 1971)

Principle:

At a certain pH value, albumin is specifically combined with bromcresol green, to produce a coloured complex, which is photometrically measured.

Reagent:

Bromocresol green solution, composed as follows:

. Succinate buffer pH 4.2 (0.05 M)

. Bromocresol green

. Surfactants

. Preservatives and stabilizers

Procedure:

	Blank (BL)	Standard (ST)	Sample (SA)
Sample	------	------	10 ml
Standard	------	10 ml	------
Reagent	2500 ml	2500 ml	250

Mix and let stand for 5 minutes at room temperature.

Read immediately at wavelength 630 nm.

Calculations:

$$\frac{\text{SA O.D}}{\text{ST O.D}} \times \text{ST concentration in g\%}$$

Normal range:

3.9 - 5.0 g/dl

(IV) DIETARY ASSESSMENT

The purpose of the questions was carefully explained to the mother so that they respond with validity. A food frequency questionnaire and 24-hour recall was used (Appendix 2). Two days later, the mother was seen again to record a 24-hour recall of the nutrition intake of her child in the two successive days following the 1st 24-hour recall information. Adequately collected food intake data were evaluated by a skillfull nutrition specialist, who categorised the food intake into carbohydrate (CHO), protein and fat intake, and then the total caloric intake per day was estimated for each of the 3 days. Finally an average total caloric intake, in addition to CHO, protein and fat caloric intake was evaluated per 24 hours.

Recommended dietary allowances were determined from the Food and Nutrition Board, National Research Council, as shown in table 9 (page 33).

Evaluation of the extent to which the child's nutritional requirements are being met, was done by using the "nutritional index" (NI). The nutritional

index quantifies the extent to which the child's actual intake of a nutrient meets the expected or desirable intake defined for that child *(Wade,1977).*

$$NI = \frac{\text{Actual intake of nutrient-Desirable intake}}{\text{Desirable intake}} X\ 100$$

If the actual daily intake exceeds the desirable intake, the nutritional index is stated as a positive percentage. If actual intake equals desirable intake, the index is stated as +1 per cent to avoid an index of zero. If the actual intake is less than the desirable intake, then the nutritional index is stated as a negative percentage *(Wade, 1977).*

(V) STUDY OF SOCIAL BEHAVIOR IN CHILDREN 2-3 YEARS OLD

Assessment of the sociability of the children 2-3 years old, was based on a semi standardized procedure devised by Stevenson and Lamb in 1979 (Appendix 4), to assess the child's initial responses to a stranger in the mother's presence *(Stein et al., 1991).*

The test lasted 3-5 minutes for each child. It began with the child sitting on the mother's lap, the examiner offered the child a toy, and then tried to initiate a " give and take" exchange of the toy. Next, an assessment was made of the child's response to being placed on the floor. Finally, the examiner moved to the floor, and again tried to start a " give and take " exchange of the toy. The infant response to each initiative was rated on a 1-5 scale, ranging from withdrawn/distressed to outgoing/friendly.

Table (15)
Scoring of child's sociability and behaviorto a stranger (Stevenson & Lamb, 1979)

Score	Sociability and Behavior
1 - 5	Quite unfriendly, unsocial, fearful
5 -10	Generally unfriendly, serious, worried
10 - 15	Neutral, neither friendly nor unfriendly
15 - 20	Friendly, positive reaction
20 - 25	Very friendly, outgoing, smiling

(VI) BEHAVIORAL AND PSYCHOLOGICAL ASSESSMENT IN CHILDREN 3-6 YEARS OLD

The child assessment schedule (CAS) was developed by Hodges et al., in 1982 (Appendix 5), to address the need for a diagnostic interview which is appropriate for children. The CAS has questions and responses that are standardized, and the format was designed to facilitate the development of a good rapport with the child. In addition, questions and response items were chosen such that information, which is relevant to making a diagnosis according to DSM-III-R (page 103), is obtained from the child. The interview consists of two parts. In the first part, the child is asked a series of questions about several topics, including school, friends, activities and hobbies, family, fears, worries, mood, somatic concerns, expression of anger and thought disorder symptomatology. The second part of the CAS provides a format for the interviewer to record observations and judgements after the completion of the interview ***(Hodges et al., 1982).***

(VII) EVALUATION OF MATERNAL POSTNATAL DEPRESSION

The Edinburgh Postnatal Depression Scale (EPDS) is a validated instrument developed specifically to identify women experiencing postnatal depression. It consists of 10 statements related to symptoms of depression. Four possible responses are provided for each question, indicating the level of severity experienced. The items are scored from 0 to 3, according to increased severity of the symptom, and when totaled, can provide immediate identification of the likelihood of depression (Appendix 6). Validation studies advocate using a score of > 13, to identify women experiencing postnatal depression. The scale is simple to complete, and does not require the health worker to have any specialist knowledge of psychiatry ***(Cox et al., 1987).***

(VIII) STATISTICAL METHODS

Statistical Analysis System (SAS) was used for data management and analysis and Harvard Graphics package was used for the figures.

Quantitative data were simmarized as means and standard deviations. Qualitative data were summarized as percentages. Analysis of variance procedures (ANOVA) or the nonparametric analysis of variance, Kruskal-Wallis test was used to compare more than 2 groups. ANOVA is an extension to the Student's t-test used when comparing more than 2 groups. For each

significant test, pairwise comparisons between the groups were performed using Sheffe's multiple comparisons test. Comparisons between percentages were done using the chi-square test or Fisher's exact test for small samples ***(Armitage & Berry, 1987).***

P-values < or = 0.05 is considered significant. All reported p-values are two-sided.

Results

Results

100 children suffering from failure to thrive (FTT) were classified into nonorganic failure to thrive (NOFTT) group composed of 56 children and organic failure to thrive (OFTT) group composed of 44 children (table 16)

NOFTT group was subclassified into 3 subgroups: nutritional, environmental and constitutional (table 17)

Organic causes of FTT such as gastrointestinal, neurological, pulmonary, cardiac, renal and others are shown in (table 18)

Age difference between the 3 groups was *highly significant* (P-value <0.001). Children in NOFTT group were younger than those in OFTT group, while sex distribution in the 3 groups was *not significant* (table 19 and figure 1)

Family size comparison between the 3 groups was *highly significant* (p-value = 0.002). Family size was bigger in OFTT than in NOFTT group (table 20 and figure 2).

Family income study was a *highly significant* factor (P-value = 0.001) in the socioeconomic evaluation of the cases of FTT. It was lower in both affected groups (NOFTT and OFTT groups) than in controls, and it was lower in NOFTT than in OFTT group (table 21 and figure 3).

Father and mother occupation and education was *not a significant factor* in the evaluation of the socioeconomic status of the parents of FTT children (tables 22and 23).

Comparison of the socioeconomic status in the 3 groups was *not significant* (table 24).

Comparison of the marital status between the 3 groups was *not significant* (table 25).

Mean weight (Wt), weight-for-age Z-score (WAZ), weight-for-age percentile (WAP) and weight-for-age median (WAM) values were *significantly lower* (P-value <0.001) in both affected groups compared to controls, but there was no difference between the 2 affected groups (table 26 and figure 4).

Mean height (Ht), height-for-age Z-score (HAZ), height-for age percentile (HAP) and height-for-age median (WAM) values were *significantly lower* (P-value <0.001) in both affected groups compared to controls, but there was no difference between the 2 affected groups (table 27 and figure 5).

Mean weight-for-height Z-score (WHZ), weight-for-height percentile (WHP), weight-for-height median (WAM) and body mass index (BMI) values were *significantly lower* (P-value <0.001) in both affected groups than in controls, but there was no difference between the 2 affected groups (table 28 and figure 6).

Midarm circumference (MAC) and triceps skinfold (TSF) values were *significantly lower* (P-value <0.001) in both affected groups than in controls, and were lower in NOFTT than in OFTT group (table 29).

Correlation between MAC and WAZ in the 3 groups was *not significant* (table 30)

The number of children suffering from anemia (hemoglobin lower than 10 g/dl) was *significantly higher* (P-value = 0.001) in both affected groups than in controls, but there was no difference between the 2 affected groups (tables 31&32 and figures 7&8).

Serum albumin was *significantly lower* (P-value = 0.008) in both affected groups than in controls, but there was no difference between both the 2 affected groups (table 33).

Estimation of protein caloric intake in the 3 groups was *highly significant* (P-value <0.001). It was lower in both affected groups than in controls, but there was no difference between the 2 affected groups (table 34 and figure 9).

Estimation of carbohydrate caloric intake in the 3 groups was *highly significant* (P-value <0.001). It was lower in both affected groups than in controls, and it was lower in NOFTT than in OFTT group (table 34 and figure 10).

Estimation of fat caloric intake in the 3 groups was *highly significant* (P-value <0.001). It was lower in both affected groups than in controls, and it was lower in OFTT than in NOFTT group (table 34 and figure 11).

Estimation of the mean total caloric intake in the 3 groups was *highly significant* (P-value <0.001). It was lower in both affected groups than in controls, and it was lower in NOFTT than in OFTT group (table 35 and figure 12).

Nutritional index was *significantly lower* (P-value <0.001) in both affected groups than in controls, but there was no difference between the 2 affected groups (table 36 and figure 13).

Eighty one percent of children in both affected groups and in controls were breast fed. Those who were breast fed for more than 1 year represented 61% in NOFTT group, 28% in OFTT group and 18% in control group (table 37).

Frequency of anorexia was *significantly higher* (P-value = 0.001) in both affected groups than in controls, but there was no difference between the 2 affected groups (table 38 and figure 14).

The number of mothers suffering from depression was *significantly higher* (P = 0.008) in both affected groups than in controls, and was slightly higher in OFTT than NOFTT group (table 40 and figure 15).

Frequency of child behavioral and psychological affection was *significantly higher* (P-value <0.01) in both affected groups than in controls, but there was no difference between the 2 affected groups (table 41 and figure 16).

Conduct disorders, mental retardation, speech problem, attention deficit hyperactivity disorder, autism, sleeping disorders, anxiety and eating

disorders are different child behavioral and psychological disorders detected in the 3 groups (table42).

There was a ***significant*** positive relation between maternal depression and child behavioral and psychological affection (table 43 and figure 17).

Table (16) Classification of children With Failure to thrive

Causes	*No of children*
Nonorganic etiology	56
. Nutritional	46
. Environmental deprivation	4
. Constitutional	6
Organic etiology	44

Table (17) Etiological classification of children with nonorganic failure to thrive

Cause of NOFTT	*No of children and %*	
Protein-energy malnutrition	**46**	**82%**
. Marasmus	37	65%
. Kwashiorkor	9	17%
Environmental deprivation	**4**	**7.3%**
. Child abuse	1	1.8%
. Separation anxiety	1	1.8%
. Alcoholic parents	1	1.8%
. Addictive parents	1	1.8%
Constitutional	**6**	**10.7%**

Table (18) Etiological classification of children with organic failure to thrive

Causes	*No of patients and %*
Gastrointestinal	**6 (13%)**
. Post-corrosive esophageal stricture	3
. Chronic diarrhea with E.Coli	2
. Celiac disease	1
Central nervous system	**7 (16%)**
. Mental retardation and cerebral palsy	5
. Microcephaly	1
. Multiple congenital anomalies	1
Pulmonary	**4 (9%)**
. Tuberculosis	3
. Bronchiectasis	1
Cardiovascular	**6 (13%)**
. Fallot's tetralogy	3
. Rheumatic heart disease	2
. Atrial septal defect with pulmonary stenosis	1
Endocrine	**5 (12%)**
. Diabetes mellitus	2
. Growth horrmone deficiency	2
. Cretinism	1
Chromosomal	**3 (7%)**
. Down's syndrome	3
Renal	**4 (9%)**
. Bilateral renal hypoplasia	2
. Chronic renal failure	1
. Polycystic disease of the kidney	1
Orthopedic	**1 (3%)**
. Pott's disease	1
Hematological	**4 (9%)**
. Thalassemia Major	2
. Sickle cell anemia	2
Neoplastic	**4 (9%)**
. Acute lymphocytic leukemia	2
. Hodjkin lymphoma	1
. Wilm's tumour	1

Table (19) Mean age and sex distribution in the different groups

Groups	*Number of cases*	*Age (months)*		*Sex*	
		Mean+/-sd	*Range*	*Male*	*Female*
Control	40	41.6+/-15.2b	24-72	20 (50.0)	20 (50.0)
Nonorganic	56	33.8+/-13.8c	24-72	35 (62.5)	21 (37.5)
Organic	44	49.7+/-14.8a	24-72	29 (65.9)	15 (34.1)
P-value		*< 0.001*		*N.S.*	

* P-values < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

Table (20) Family size distribution in the different groups

Number of children	*Control (n=40)*		*Nonorganic (n=56)*		*Organic (n=44)*	
	n	*(%)*	*n*	*(%)*	*n*	*(%)*
5 or more	2	(5)	1	(1.8)	5	(11.4)
3 or 4	13	(32.5)	32	(57.1)	29	(65.9)
1 or 2	25	(62.5)	23	(47.1)	10	(22.7)
P-value	*0.002*					

* P-values < or = 0.05 is considered significant, N.S. = not significant.

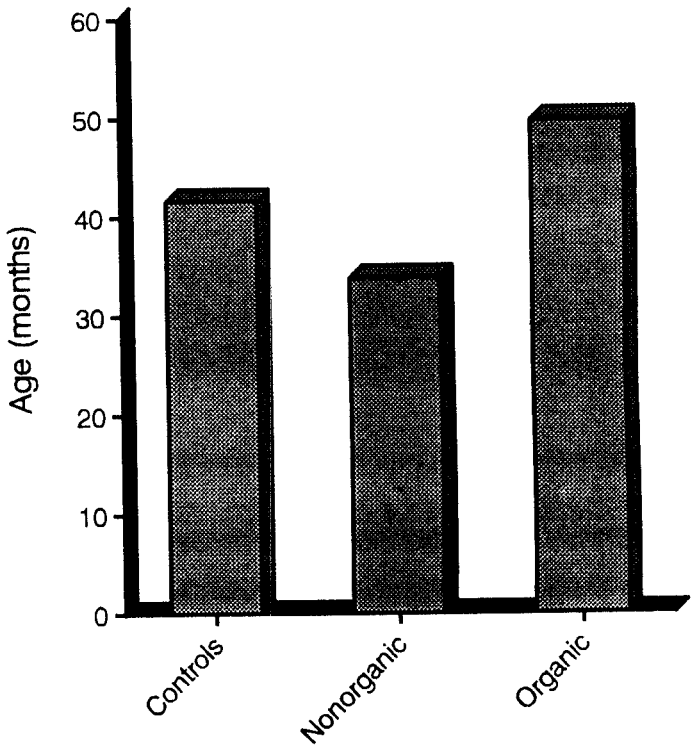

Fig (1): Mean Age in the Different Groups.

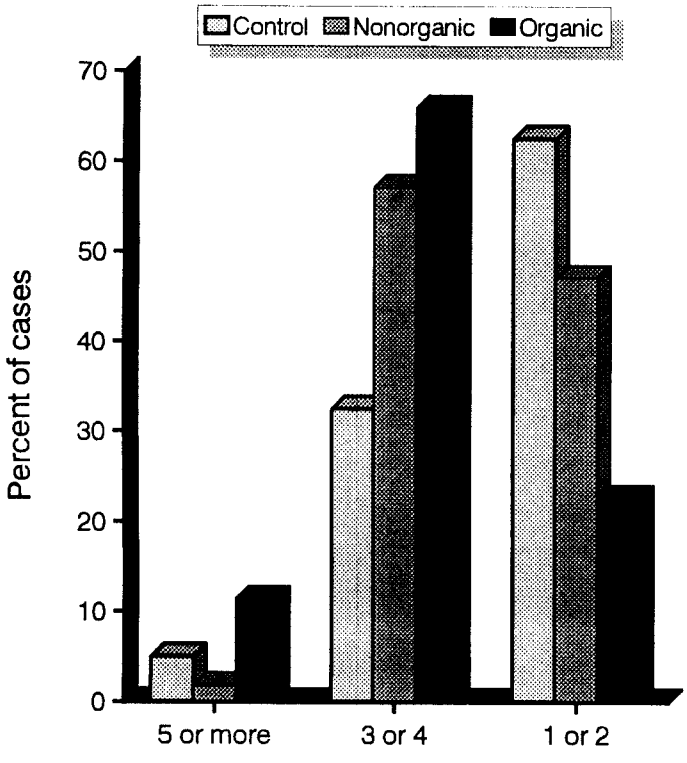

Fig (2): Family Size Distribution in the Different Groups.

Table (21) Family income distribution in the different groups

Family income	*Control (n=40)*		*Nonorganic (n=56)*		*Organic (n=44)*	
	n	*(%)*	*n*	*(%)*	*n*	*(%)*
100 LE	13	(32.5)	46	(82.1)	25	(56.8)
100-200 LE	26	(65)	10	(17.9)	19	(43.2)
> 200 LE	1	(2.5)	0	(0.0)	0	(0.0)
P-value	***0.001***					

* P-values < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

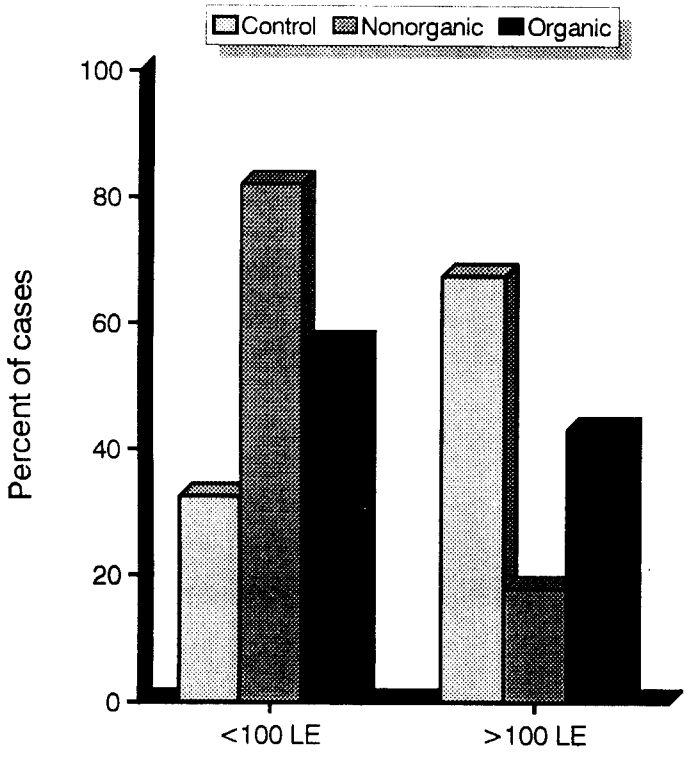

Fig (3): Family Income Distribution in the Different Groups.

Table (22) Father occupation and education distribution in the different groups

Ocupation & education	*Control (n=40)* n (%)	*Nonorganic (n=56)* n (%)	*Organic (n=44)* n (%)
Manual worker, did not complete 1ry education, or illiterate	31 (77.5)	41 (73.2)	27 (61.4)
Skilled laborer or 2ry school	9 (22.5)	13 (23.2)	17 (38.6)
Government employee or university graduate	0 (0.0)	2 (3.6)	0 (0.0)
P-value	***N.S.***		

* P-values < or = 0.05 is considered significant, N.S. = not significant.

Table (23) Mother occupation and education distribution in the different groups

Occupation & education	*Control (n=40)* n (%)	*Nonorganic (n=56)* n (%)	*Organic (n=44)* n (%)
Manual worker, did not complete 1ry education or illiterate	30 (75)	43 (76.8)	29 (65.9)
Skilled laborer or 2ry school	10 (25)	13 (23.2)	15 (34.1)
Government employee or university graduate	0 (0.0)	0 (0.0)	0 (0.0)
P-value	***N.S.***		

* P-values < or = 0.05 is considered significant, N.S. = not significant.

Table (24) Socioeconomic status in the different groups

Socioeconomic status	*Control (n=40)* n (%)	*Nonorganic (n=56)* n (%)	*Organic (n=44)* n (%)
Low	26 (65)	44 (78.6)	29 (65.9)
Middle	14 (35)	12 (21.4)	15 (34.1)
High	0 (0.0)	0 (0.0)	0 (0.0)
P-value	***N.S.***		

* P-values < or = 0.05 is considered significant, N.S. = not significant.

Table (25) Marital status in the different groups

Marital status	*Control (n=40)* n (%)	*Nonorganic (n=56)* n (%)	*Organic (n=44)* n (%)
Married	31 (77.5)	39 (69.6)	30 (68.2)
Divorced	6 (15.0)	7 (12.5)	9 (20.4)
Widow	0 (0.0)	1 (1.8)	0 (0.0)
Separated	3 (7.5)	9 (16.1)	5 (11.4)
P-value	***N.S.***		

* P-values < or = 0.05 is considered significant, N.S. = not significant.

Table (26) Mean weight, weight/age Z-score, weight/age percentile and weight/age median of the three groups in reference to the NCHS standards

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Weight (kgs)	14.7 +/- 2.5 10.5 - 20.0	10.5 +/- 1.7 9.0 - 15.5	14.3 +/- 12.6 8.8 - 95.0
Weight for age Z-score	-0.5 +/- 1.3 **a** -7.9 - 1.1	-2.4 +/- 0.4 **b** -3.4 - -1.5	-2.3 +/- 0.4 **b** -3.2 - -1.4
P-value <0.001			
Percentile	39.0 +/- 19.9 **a** 5.1 - 93.9	1.3 +/- 1.5 **b** 0.0 - 6.3	1.4 +/- 1.6 **b** 0.1 - 8
P-value <0.001			
Median	96.5 +/- 6.0 **a** 85.1 - 113.1	75.7 +/- 4.0 **b** 63.9 - 85.0	74.4 +/- 3.9 **b** 67.7 - 84.1
P-value <0.001			

Values are mean +/- standard deviation.
P-values < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

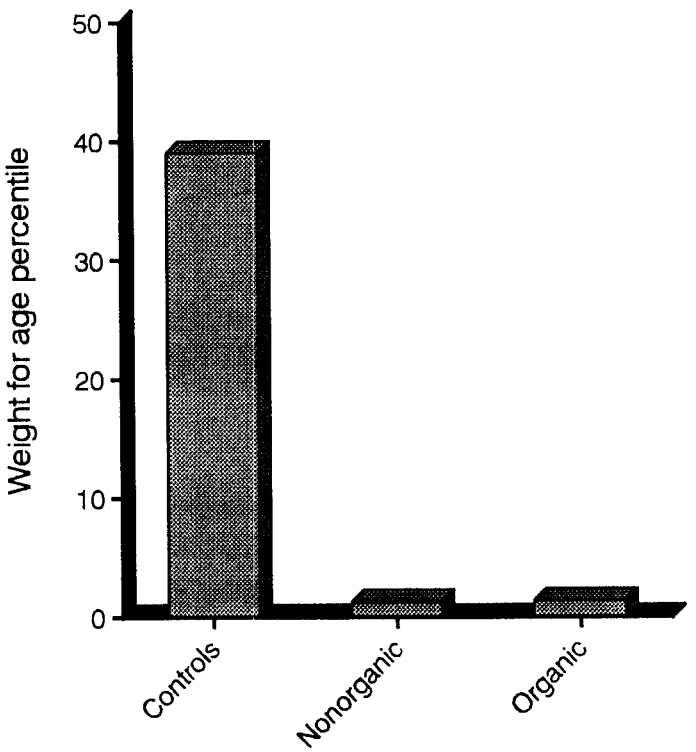

Fig (4): Mean Weight for Age Percentiles of the Different Groups in Reference to the NCHS.

Table (27) Mean height, height/age Z-score, height/age Percentile and height/age Median of the three groups in reference to the NCHS standards.

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Height (cms)	92.7 +/- 9.9 84.0 - 116	86.7 +/- 7.4 78.0 - 109	93.3 +/- 15.6 81.0 - 112.0
Height for age Z-score	-0.0 +/- 0.7 **a** -1.3 - 1.5	-1.5 +/- 0.9 **b** -3.5 - -0.6	-1.7 +/- 0.9 **b** -3.3 - -1.3
P-value <0.001			
Percentile	48.8 +/- 23.1 **a** 10.2 - 92.7	13.1 +/- 15.6 **b** 0.0 - 71.9	7.6 +/- 10.2 **b** 0.1 - 42.0
P-value <0.001			
Median	99.8 +/- 2.7 **a** 95.0 - 106.1	94.0 +/- 3.8 **b** 85.5 - 102.2	92.5 +/- 3.3 **b** 86.4 - 99.2
P-value <0.001			

Values are mean +/- standard deviation and range.
P-values < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

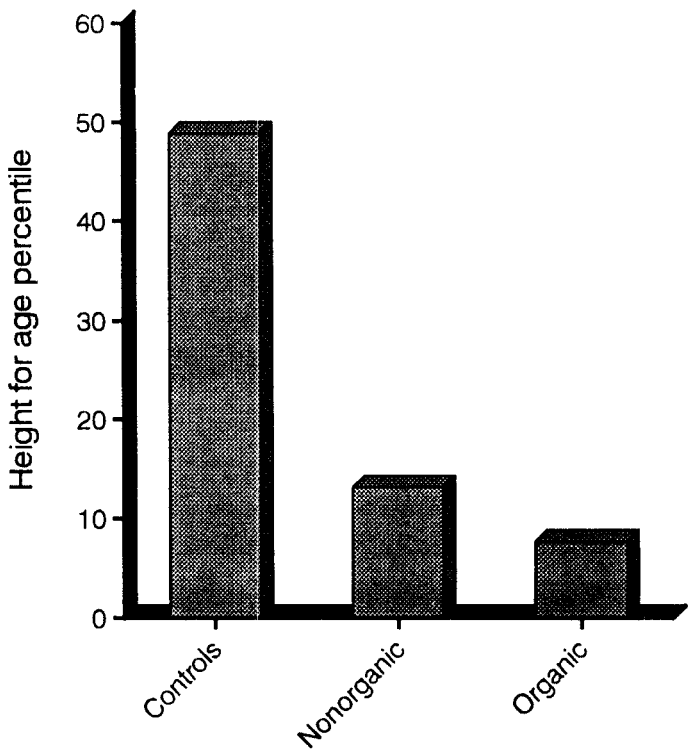

Fig (5): Mean Height for Age Percentiles of the Different Groups in Reference to the NCHS.

Table (28) Mean weight/height Z-score, weight/height Percentile and weight/height Median and body mass index of the three groups in reference to the NCHS standards

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Weight for height			
Z-score	-0.2 +/- 0.4 **a**	-1.7 +/- 0.5 **b**	-1.6 +/- 0.6 **b**
	-1.3 - 0.4	-2.7 - -0.2	-2.6 - 0.8
P-value <0.001			
Percentile	42.4 +/- 13.9 **a**	6.6 +/- 7.5 **b**	7.5 +/- 7.3 **b**
	9.7 - 66.2	0.4 - 34.9	0.5 - 30
P-value <0.001			
Median	98.0 +/- 4.1 **a**	84.5 +/- 4.6 **b**	85.6 +/- 4.5 **b**
	88.3 - 104.6	75.7 - 96.6	77.7 - 95.4
P-value <0.001			
Body mass index	15.6 +/- 0.7 **a**	14.0 +/- 0.8 **b**	13.7 +/- 0.9 **b**
	14.4 - 17.4	11.8 - 15.9	12.0 - 15.6
P-value <0.001			

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

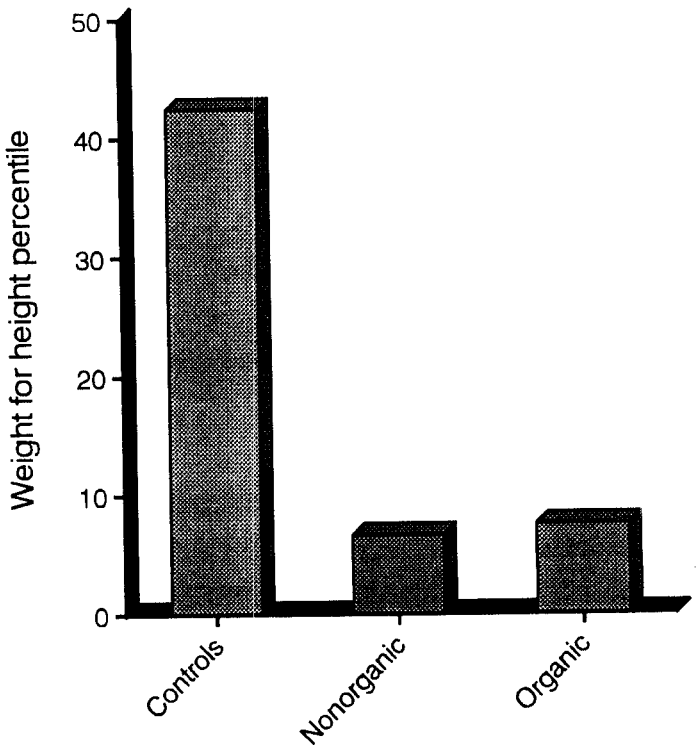

Fig (6): Mean Weight for Height Percentiles of the Different Groups in Reference to the NCHS.

Table (29) Mean midarm circumference and triceps skinfold of the three groups in reference to the NCHS standards

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Midarm circumference (cms)	15.4 +/- 1.5 **a** 12.9 - 18.5	11.6 +/- 1.2 **c** 10.0 - 14.0	12.9 +/- 1.2 **b** 10.0 - 14.8
P-value <0.001			
Triceps skinfold (cms)	9.2 +/- 1.0 **a** 7.8 - 11.5	6.8 +/- 1.0 **c** 5.0 - 9.8	7.4 +/- 1.7 **b** 4.5 - 13.2
P-value <0.001			

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

Table (30) Weight for age in relation to midarm circumference

Midarm circumference	*Weight for age Z-score*				*P-value*
	<2		*2*		
	n	*(%)*	*n*	*(%)*	
Control					
Total number	1		39		***N.S.***
< 12.5 cm	0	(0.0)	4	(10.3)	
Nonorganic					
Total number	49		7		***N.S.***
< 12.5 cm	20	(40.8)	2	(28.6)	
Organic					
Total number	37		7		***N.S.***
< 12.5 cm	10	(27.0)	4	(57.1)	

* P-values < or = 0.05 is considered significant, N.S. = not significant.

Table (31) Mean level of the hematological parameters of the three groups

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Hemoglobin (g/dl)	11.6 +/- 1.3 **a** 9.7 - 15.2	9.9 +/- 1.0 **b** 8.2 - 12.7	9.8 +/- 1.3 **b** 6.5 - 12.4
P-value <0.001			
Hematocrit (%)	33.6 +/- 3.3 **a** 29.0 - 45.0	31.5 +/- 2.1 **b** 28.0 - 36.0	31.0 +/- 2.4 **b** 26.0 - 36.0
P-value <0.001			
Red blood cells	4.3 +/- 0.4 **a** 3.5 - 5.0	3.9 +/- 0.5 **b** 2.9 - 5.0	3.7 +/- 0.8 **b** 0.4 - 4.9
P-value <0.001			
White blood cells	8.6 +/- 2.3 3.3 - 13.4	7.7 +/- 3.0 1.3 - 17.6	8.2 +/- 4.1 0.0 - 21.0
P-value = N.S.			

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

Table (32) Frequency of anemia in the three groups

	Control (n=40) *n (%)*	*Nonorganic (n=56)* *n (%)*	*Organic (n=44)* *n (%)*
Anemia (Hemoglobin <10 g/dl)	12 (30.0)	44 (80.0)	34 (77.3)
P-value=0.001			

P-values < or = 0.05 is considered significant, N.S.= not significant.

Table (33) Mean level of serum albumin in the three groups

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Albumin (g/dl)	3.5 +/- 0.4 **a**	3.2 +/- 0.6 **b**	3.3 +/- 0.5 **b**
	2.9 - 4.3	2.0 - 4.6	2.5 - 4.0
P-value=0.008			

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

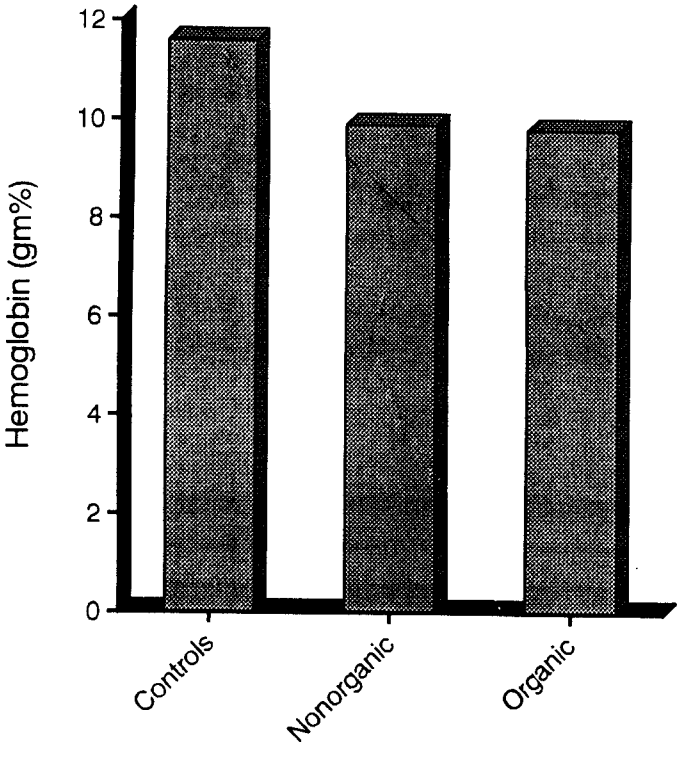

Fig (7): Mean Hemoglobin in the Different Groups.

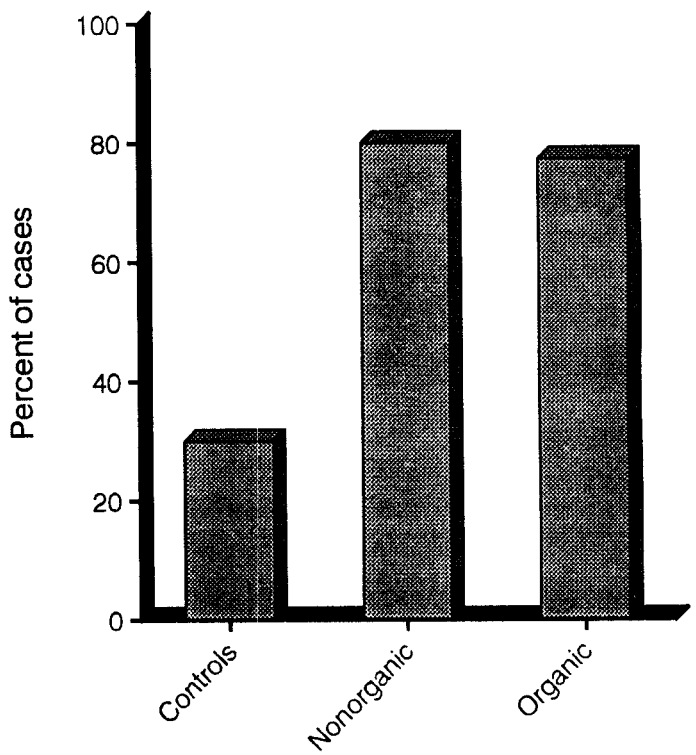

Fig (8): Frequency of Anemia in the Different Groups.

Table (34) Mean protein, carbohydrate and fat caloric intake of the three groups

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Protein	276.8 +/- 89.5 **a**	161.0 +/- 77.6 **b**	191.7 +/- 61.3 **b**
	107.0 - 475.0	40.0 - 362.0	60.0 321.0
P-value <0.001			
Carbohydrate	731.4 +/-119.6 **a**	385.6 +/- 162.1 **c**	468.4+/-174.1 **b**
	555.0 - 1050.0	134.0 - 860.0	163.0 - 875.0
P-value <0.001			
Fat	234.2 +/- 71.6 **a**	153.2 +/- 97.3 **b**	200.3 +/- 66.4 **c**
	100.0 - 384.0	32.0 - 466.0	95.0 - 365.0
P-value <0.001			

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

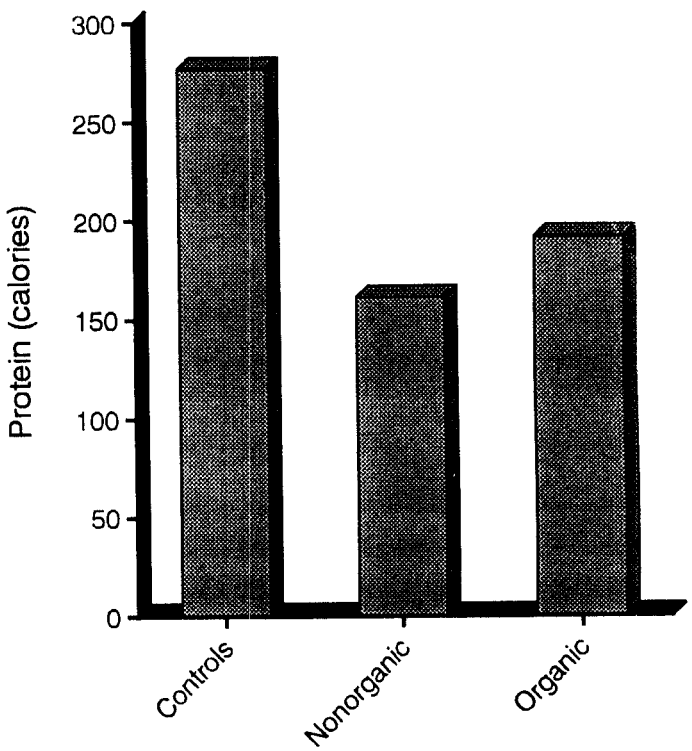

Fig (9): Mean Protein Caloric Intake in the Different Groups.

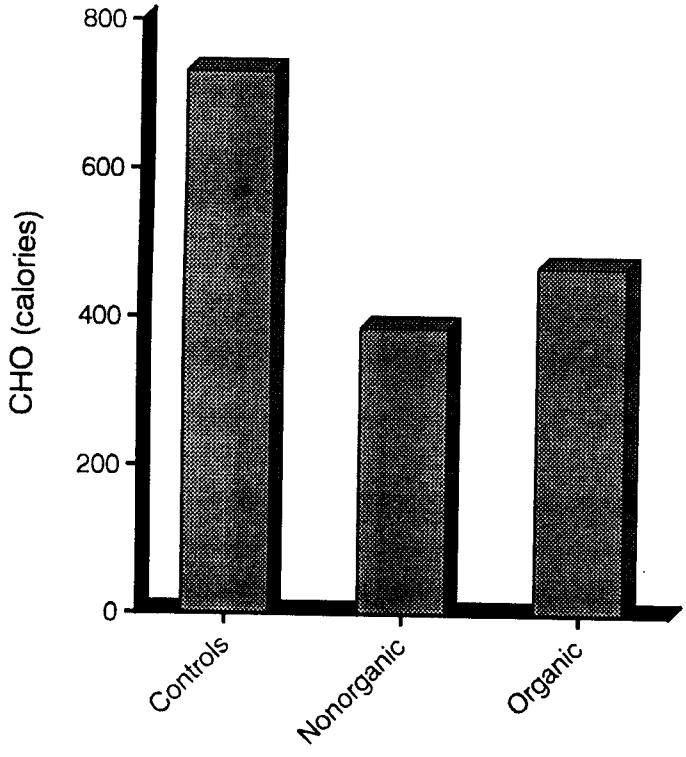

Fig (10): Mean Carbohydrate Caloric Intake in the Different Groups.

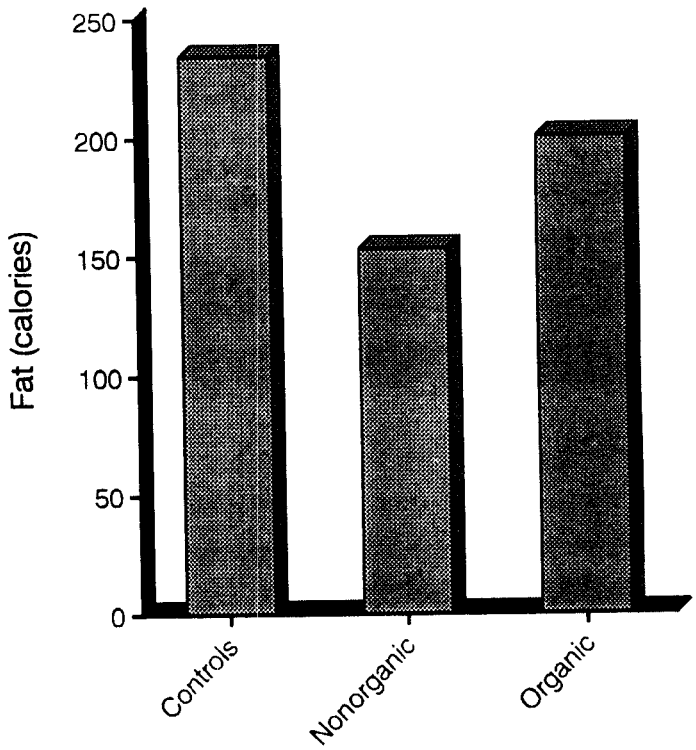

Fig (11): Mean Fat Caloric Intake in the Different Groups.

Table (35) Mean caloric intake, expected caloric intake and caloric deficit of the three groups

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Caloric intake	1237 +/- 163.1 **a**	700.7 +/- 264.8 **c**	860.3 +/- 202.8**b**
	900.0 - 1520.0	400.0 - 1400.0	500.0 - 1300.0
P-value <0.001			
Expected caloric intake	731.4 +/-119.6 **b**	1298.2+/-148.0 **c**	1498.9+/-180.0 **a**
	1200.0 - 1750.0	1150.0 - 1750.0	163.0 - 875.0
P-value <0.001			
Caloric deficit	166.5 +/- 105.1 **b**	597.7+/- 181.5 **a**	646.6+/- 184.9 **a**
	0.0 - 350.0	98.0 - 894.0	240.0 - 907.0
P-value <0.001			

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

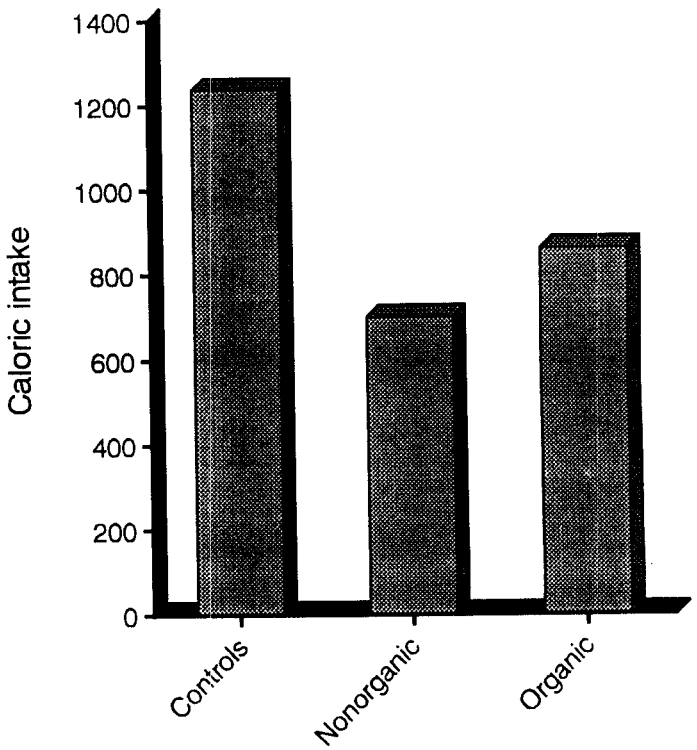

Fig (12): Mean Caloric Intake in the Different Groups.

Table (36) Mean level of the nutritional index of the three groups

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Nutritional index	-11.2 +/- 7.6 **a** -28.0 - 1.0	-46.5 +/- 14.7 **b** -65.0 - -10.0	-42.7 +/- 11.5 **b** -61.0 - -16.0
P-value	***< 0.001***		

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

Table (37) Frequency and duration of breast feeding in the three groups

	Frequency	*6 months*	*6-12 months*	*> 1 year*
Control	32 (80%)	6	20	6 (18%)
NOFTT	48 (85%)	6	10	32 (61%)
OFTT	35 (80%)	7	18	10 (28%)

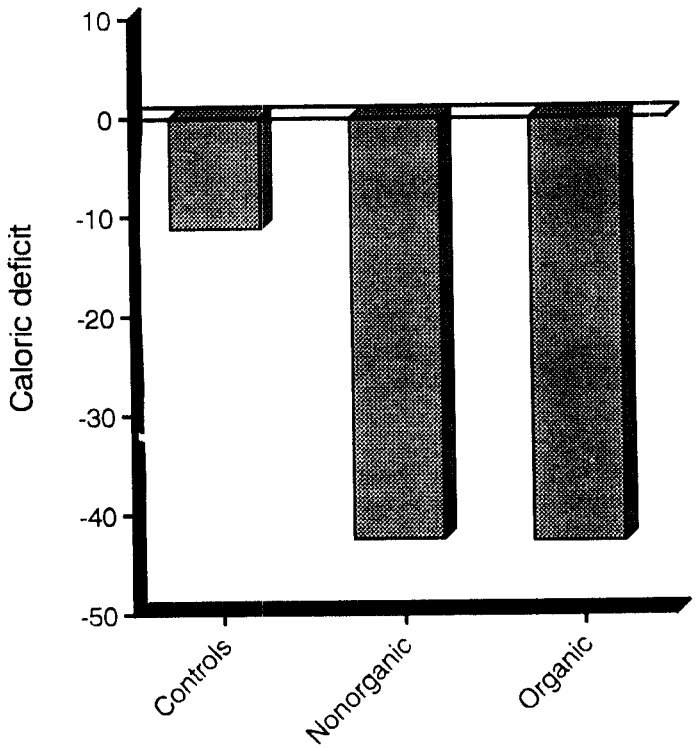

Fig (13): Mean Nutritional Index in the Different Groups.

Table (38) Frequency of anorexia in the three groups

	Control (n=40)		*Nonorganic (n=56)*		*Organic (n=44)*	
	n	*(%)*	*n*	*(%)*	*n*	*(%)*
Anorexia	18	(45.0)	47	(83.9)	31	(70.5)
P-value=0.001						

P-values < or = 0.05 is considered significant, N.S.= not significant.

Table (39) Assessment of maternal depression score in the three groups

	Control (n=40)	*Nonorganic (n=56)*	*Organic (n=44)*
Maternal depression score	10.9 +/- 3.2 **b** 7.0 - 17.0	13.2 +/- 4.2 **a** 7.0 - 20.0	14.6 +/- 3.2 **a** 6.0 - 19.0
P-value	***< 0.001***		

Values are mean +/- standard deviation and range.
P-values* < or = 0.05 is considered significant, N.S. = not significant. For each significant test group, means sharing same letter are not significantly different from each other.

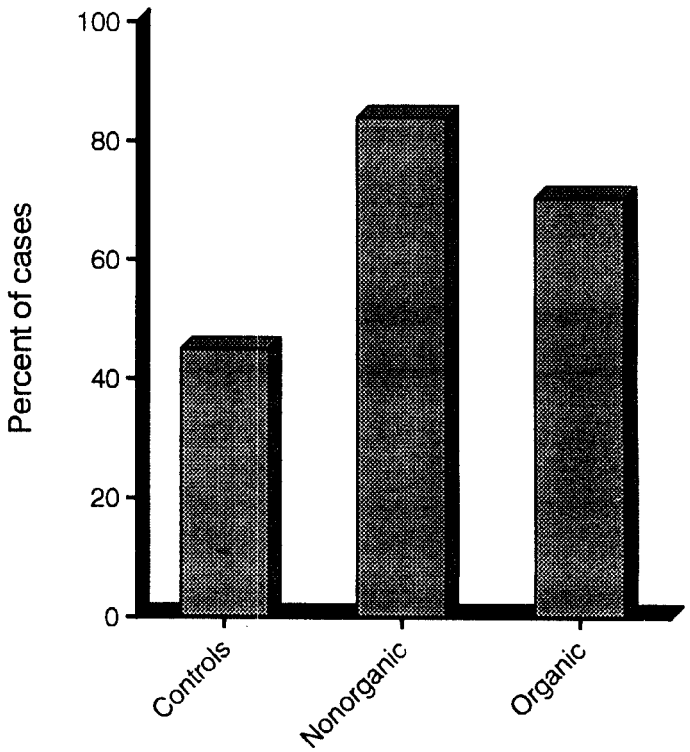

Fig (14): Frequency of Anorexia in the Different Groups.

Table (40) Frequency of maternal depression in the three groups

	Control (n=40) *n (%)*	*Nonorganic (n=56)* *n (%)*	*Organic (n=44)* *n (%)*
Maternal depression	11 (27.5)	30 (53.6)	26 (59.1)
P-value	***= 0.008***		

P-values* < or = 0.05 is considered significant, N.S. = not significant.

Table (41) Frequency of child behavioral and psychological affection in the three groups

	Control (n=40) *n (%)*	*Nonorganic (n=56)* *n (%)*	*Organic (n=44)* *n (%)*
Child behavioral and psychological affection	6 (15.0)	29 (52.1)	26 (59.1)
P-value	***= 0.001***		

P-values* < or = 0.05 is considered significant, N.S. = not significant.

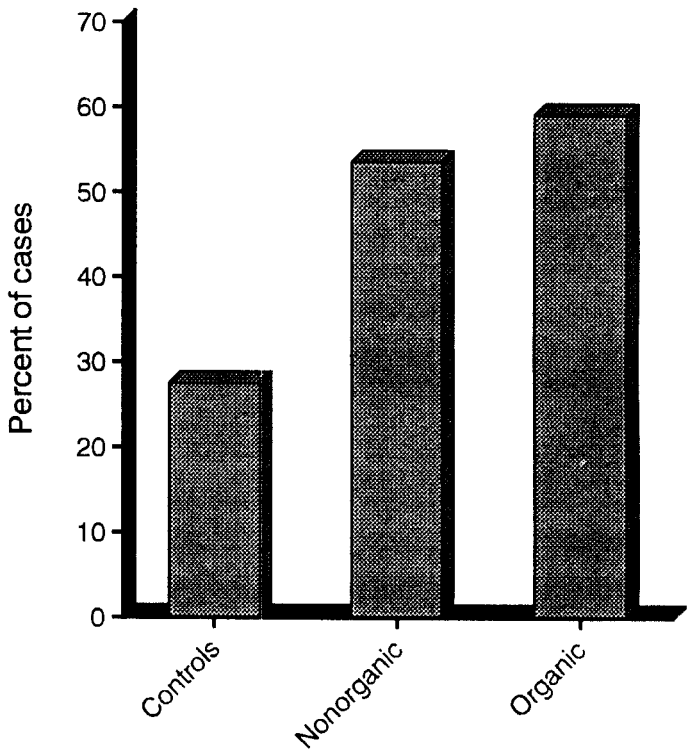

Fig (15): Frequency of Maternal Depression in the Different Groups.

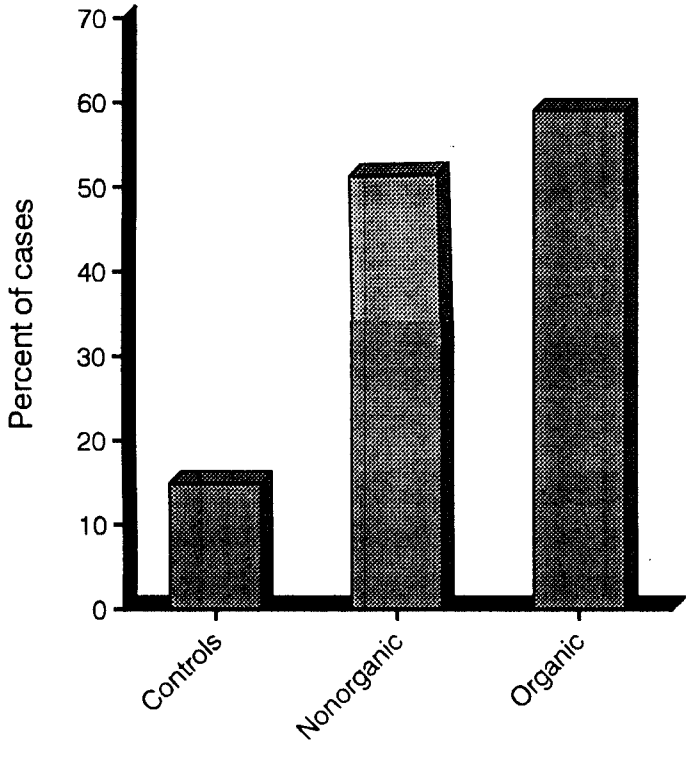

Fig (16): Frequency of Child Behavioral and Psychological Affection in the Different Groups.

Table (42) Type and frequency of child behavioral and psychological affection inNOFTT and OFTT groups

Type of affection	*NOFTT (29 cases)*	*OFTT (26 cases)*
(children 2-3 years old) ***Quite unfriendly, unsocial, fearful (19)***	.11 marasmus .2 Kwashiorkor .2 constitutional FTT (30 months old)	.1 atrial septal defect .1 microcephaly .3 esophageal stricture
(children 3-6 years old) ***Mental retardation (11)***	0	11
Autism (1)	.1 marasmus (44 months old boy)	0
Speech problem (3)	.1 autism .1 separation anxiety	.1rheumatic heart disease (RHD) (6 years old boy)
Conduct disorder (18)	.1 alcoholic father (stealing) .2 constitutional FTT (lying) (3 & 5 years old girls) .1 addictive father(insulting) .1 child abuse(lying,spitting) .1 separation anxiety (lying) .3marasmus(fighting, spitting) (3years old boys)	.1tuberculosis (6years old boy) (setting fire) .1 diabetes mellitus (fighting and insulting) .2 cerebral palsy (post. infl. and post. traumatic) (aggressive &destroying) .1 Wilm's tumor(spitting) .1 RHD 5 years old girl(lying) .1 sickle cell anemia(lying) .1thalassemia (disobeying & arrugant) (3 years old boy) .1 growth hormone deficiency (stealing)
Hyperactive child (3)	.1 separation anxiety	.1 thalassemia(3 years oldboy) .1 Down syndrome
Anxiety disorder (4)	.1 separation anxiety .2 constitutional FTT (2 brothers)	.1 tuberculosis (4 years old boy)
Eating disorder (1)	.1 Food allergy (psychological anorexia)	
Sleeping disorder (8)	.1 autism .1 separation anxiety .2 constitutional FTT (2 brothers) .1 marasmus (3 years old girl) .1 alcoholic father .1 addictive father	.1 tuberculosis (4 years old boy)

Table (43) Maternal depression in relation to child behavioral affection

Child behavioral affection	*Maternal depression*				*P-value*
	Yes		*No*		
	n	*(%)*	*n*	*(%)*	
Control					
Total number	11		29		***0.020***
Affected children	4	(36.4)	2	(6.9)	
Nonorganic					
Total number	30		26		***0.001***
Affected children	25	(83.0)	4	(16)	
Organic					
Total number	26		18		***0.001***
Affected children	25	(96.2)	1	(5.6)	

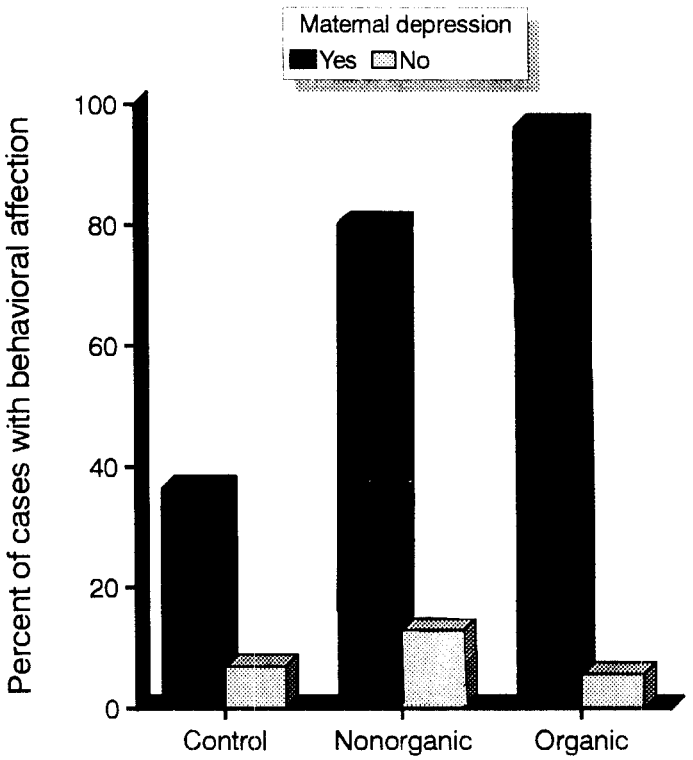

Fig (17): Frequency of Child Behavioral and Phychological Affection in Relation to Maternal Depression in the Different Groups.

Discussion

Discussion

Classification of failure to thrive

This study was done on 100 preschool children, aged between 2 and 6 years old, suffering from FTT. They were recruited from the admitted cases in the inpatient sections of Kasr El Eini and Ein Shams Universities in Cairo, some of the cases were as well recruited from the health office clinic in El Katta village, near El Kanater in Giza where a survey study for rural development was going on there by the National Research Center. FTT cases were assessed by chosing children whose Wt/Age was less than 5th percentile.

In this study classification of failure to thrive into organic failure to thrive (OFTT) and nonorganic failure to thrive (NOFTT) was based on positive findings, not on exclusion. Positive findings resulted from the performance of several investigations as follows:

- A ***careful history*** that included symptoms of different systems of the body, to detect any organic cause for FTT.

- ***Physical examination*** included abdominal, cardiac, pulmonary and neurological systems. Physical examination proved to be a sensitive indicator of an organic component. ***Laboratory and radiographic investigations*** rarely produce findings in favour of an organic etiology that were not anticipated by clinical evaluation ***(Homer & Ludwig, 1981).***

- ***Anthropometric measurements*** included the child's height (Ht), weight (Wt), midarm circumference (MAC) and triceps skinfold (TSF).

- Careful evaluation of ***caloric intake*** to detect underfeeding (Appendix 2).

- A careful questionnaire should be taken to detect any ***psychiatric disturbance,*** behavioral affection of the child and any social stress on the parents which may affect their ability to function, such as friction in the marital relationships, low family income and big family size (Appendix 7).

- Critical observations for the ***interactions between children who are failing to thrive and their caretakers*** were done during the first visit of the mother. Children suffering from growth retardation in our study, with no physical cause detected from the clinical assessment, evidence of problems in the mother-child relationship have been sought. Infants who were friendly and affectionate with us during examination, had been suspected to be emotionally rejected and socially deprived within their families. Normally, mothers in this unfamiliar setting, are sensitive to signs of their children's anxiety and prompt them with verbal and physical contact to reduce stress. Low visual attention, persistent ignoring of approaches, or frequent critical remarks were considered as merely pointers to a heightened risk of emotional neglect or abuse.

Careful awareness in this study was taken by the interviewer, that abusing and neglectful parents may be perfectly pleasant and cooperative with the examining doctor. This may serve to direct one's attention from the actual mother-child relationship ***(Skuse, 1985).***

To define a case as NOFTT, the following criteria that were previously suggested by ***Barbero & Shaheen (1967),*** were considered:

. Wt/Age below the 5th percentile, according to the data of the National Center for Health Statistics (NCHS).

. Developmental retardation detected by history and anthropometry.

. No evidence of systemic disease or abnormality of laboratory findings, to account for the growth failure.

. Significant environmental or psychological disruption within the family, such as alcoholism, severe financial problems or serious parental illness.

Criteria used to assess cases of OFTT were as follows:

. Wt below the 5th percentile, according to the data of the NCHS.

. Symptoms revealing dysfunction in a certain organic system of the body.

. Evidence of signs of systemic disease or abnormality of laboratory findings, to account for the growth failure.

In our study done on 100 preschool children with FTT, NOFTT (56%) was more common than OFTT (44%).

Ambuel & Harris (1963) carried a study on 100 preschool children of average age 4 years old, suffering of FTT. They found on the other hand that

the OFTT (68%) was more common than NOFTT (32%). Similarly ***Riley et al. (1968),*** studied 83 children of average age 3 years old, suffering of FTT, and found that OFTT (47%) was more common than NOFTT (33%) and 20% were undiagnosed. These authors were relying in their assessment entirely on Wt/Age, history and clinical evaluation.

Our study was similar to the studies done by ***Hannaway (1970), Sills (1978), Homer & Ludwig (1981) and Morice et al. (1989)***, in which NOFTT was more common than OFTT. We conclude therefore that the previous workers in 1963 and 1968, have reported higher frequencies of the diagnosis of organic disease, since they have undervalued the psychological investigation of the affected family, which entered lately into the area of differential diagnosis.

Batchelor & Kerslake (1990), Wittenberg (1990) and Budd et al. (1992) suggested that distinction between NOFTT and OFTT is artificial, and that the feeding process is the root cause in both groups. They refused to categorize FTT as an organic and nonorganic dichotomy, instead, they suggested an approach which recognizes both organic and nonorganic influences, and named it " Mixed FTT".

We agree with the point of view of previous authors suggesting a new category of FTT called "Mixed FTT", because both OFTT and NOFTT children in our study had anorexia and a significant diminish in caloric intake. In addition, children with OFTT in our study, displayed behavioral affection (59%) and their mothers suffered from depression (59%) as those with

NOFTT group. This supports the needs for more detailed classification including mixed OFTT and NOFTT instead of an organic-nonorganic dichotomy.

The NOFTT group (56 children) in this study was further subclassified into the following:

1. ***Malnutrition group*** ***(46cases)*** represented 82% of the NOFTT group. This group according to clinical manifestations 37 marasmic cases and 9 Kwashiorkor cases. Decreased caloric intake in this group was attributed to one of the following causes:

(a) Scanty breast milk (8 cases / 17%)

Mothers of those children insisted to breast feed their children aged between 2 and 3 years old, and considered their scanty breast milk as the main source of feeding without the introduction of supplementary feeding. In addition, the maternal dietary energy and protein intake could contribute to altered milk production or composition. Accordingly we suggest that assessment of maternal dietary intake is essential in an evaluation of the breast-fed infant with failure to thrive.

Motil et al. (1994), and ***Abdel Sayed (1995),*** showed that FTT in a breast fed infant could be attributed to reduced milk production, in conjunction with maternal dietary energy and protein restriction.

Serventi et al. (1995), confirmed that early cessation of breast feeding before the age of 2 years old, is associated with higher prevalence of malnutrition and a drastic drop of the growth curve in children. This contrasts with our findings which indicate that prolonged breast feeding is associated with higher prevalence of malnutrition in children.

(b) Poverty and ignorance (36 cases / 78%)

The nutritional problems of developing countries are conditioned by poverty and inadequate parental education regarding nutritional requirements of the child, normal growth and nutritional values of the food they offer to him.

(c) Restriction of children's diet by the mother (2cases / 5%)

A 4 years old boy was admitted to the pediatric section of Kasr El Eini Children's Hospital with FTT and history of food allergies to milk, cheese, tomatoes, fish, chocolate, strawberries and eggs. The child was slender, active and irritable. Stools were mixed with fresh blood. Colonoscopy and biopsy showed evidence of colitis characterized by a marked eosinophilic infiltrate. There was a history of allergy to different foods in both parents. Growth difficulties in this child was due to a long term restriction of many foods, leading to lack of growth.

This coincides with a study done by ***Roesler et al. (1994),*** which concluded that parental beliefs about food allergies can lead to dietary restrictions severe enough to cause FTT in their children.

Another case was that of a 30 months old girl suffering from FTT. The mother was very obese (90 Kg), and did not want her child to be overweight. She started to restrict the child's diet, such as sweets, biscuits, chocolate, fried food, meat and bread, as she considered this type of food to be unhealthy and fattening. Despite the child's Wt being below 5th percentile, she believed her child to be of normal Wt.

2. *Environmental deprivation group* ***(4 cases)*** represented 7% of the NOFTT group. We realized in this study, different forms of deprivation:

(a) Child abuse (1 case / 1.8%)

A 4 years old boy presented to us with symptoms of gastro-enteritis and severe anorexia. He looked anxious and irritable. Examining him, we discovered scattered areas of cigarette burns in his body, and a wide area of 1st degree burn with hot water on the buttocks area. This was done byhis father's wife, pretending that this was the best way to teach him potty training. Clinical examination revealed no organic cause for his FTT. Adequate nutrition, especially for the infant, requires a healthy interaction between the parents and the child. This child suffering from psychological deprivation, has a severe reduced appetite and refusal to feed. An additional factor could be that his father's wife does not offer him adequate calories.

Goldson et al., (1976) studied 140 children of average age 3 years old, exposed to nonaccidental trauma. This study revealed that 12% of these children had FTT. Bruises constituted 58% of the accidents, fractures 20%, burns 7% and drowning 3%.

(b) Separation anxiety (1 case / 1.8%)

A 5 years old girl with FTT due to severe anorexia since the death of her father 8 months ago. She was constantly loosing weight during this time. Physical, laboratory (blood picture, ESR, stool and urine tests, liver function tests, renal functions and thyroid functions) and radiological examination (chest X-rays), excluded any organic etiology for FTT. Jejunal biopsy was done and revealed no abnormalities.

(c) Alcoholic parents (1 cases / 1.8%)

A 6 years old girl whose father was severely alcoholic, had recurrent somatic symptoms such as headache, abdominal pain and anorexia. " She was depressed most of the time and socially isolated " said her mother. She saw her father beating her mother so many times, in order to take the money she gains from selling some vegetables every morning. Consequently, the child was neglected from her parents; the father being an alcoholic, and the mother being depressed. Severe anorexia resulted in severe diet restriction and inadequate caloric intake.

Nordbey et al. (1994), found that 12% of the 532 families included in their study were suffering from alcoholism. Children of alcoholic parents had poor growth development, retarded mental development and showed more behavioral problems than controls. Boys were found to be more vulnerable than girls.

(d) *Parent addiction* *(1 case / 1.8%)* A 5 years old girl, whose father was an addict, had a history of refusal to go to school, irritability and crying all the time. She had persistent nausea and severe anorexia. Child's hygiene was grossly inadequate reflecting evidence of neglect. The father was 70 years old, "hash" addict and did not participate in the house problems. The mother was 37 years old. She was socially isolated, depressed and felt insecure because of the lack of support she received from her husband. This is a case of frank breakdown in the parent-infant interaction.

In the maladapted parent-infant interaction, the parent does not read and respond to the infant appropriately, and the infant has difficulty in eliciting attention and appropriate care from the parent. This bidirectional problem, ultimately results in nutritional deficiencies which by time result in deviation from normal growth and development ***(Homer & Ludwig, 1981).***

3. ***Constitutional*** ***(6 cases)*** represented 11% of the NOFTT group.

Two brothers (5 and 6 years old) and two girls (3 and 5 years old) had both Wt and Ht below the 5th percentile. Their parents' height did not exceed 150 cm. The caloric intake of these children was satisfactory, with a

nutritional index -10%. These children were stunted as shown by HAZ, but their WHZ was little affected (Appendix 9). Physical examination of these children did not show any organic disease.

While two other children (a boy and a girl 30 months old) had both Wt and Ht below the 5th percentile. Their parents' height did not exceed 150 cm. Their caloric intake was low, with a nutritional index -55%. Stunting was shown by low HAZ values and wasting was shown by low WHZ values. Medical history and examination of these children did not show any organic or psychological disturbances. These children could also be classified in the malnourished group, but we included them in the constitutional group, because we were convinced that even if adequate calories are offered, this will not affect the genetic growth limitation.

OFTT in this study was categorized according to the organic lesion present, as shown in table (18). The predominant organic factors were neurological (16%), gastrointestinal (13%), cardiac (13%) and endocrinal (12%).

In the study done of ***Morice et al. (1989),*** the predominant organic factors were diseases of the central nervous system (41%) and gastrointestinal pathology (24%).

Age, sex and failure to thrive

In our study, there was no significant relationship between sex and FTT. This is similar to a study done by ***Altemier et al. (1985).*** On the other hand, ***Richman et al. (1975) and Herman-Staab (1992),*** found that there was a male predominance in preschool children suffering from FTT.

Regarding the age distribution in our study, we found that children in NOFTT group were younger (mean age 33.8 months) than those in OFTT group (mean age 49.7 months), the difference was highly significant ($P<0.001$). This could be explained on the basis that towards the end of the first year of life and during the second year, because of the constant decelerating rate of growth, there is a gradual reduction in the infant's caloric intake per unit of body weight. In addition, there is temporary periods of lack of interest in certain foods or even in food in general. Feeding difficulties between the ages of 2 and 3 years frequently result from excessive parental insistence on eating. Protein-energy malnutrition (PEM) occurs usually from early infancy to about 4 years of age, usually after weaning from the breast, due to reduction of caloric intake, resulting from poverty and ignorance of the mothers and their unawareness of the principles of adequate nutrition. All these factors explain the fact that children with NOFTT are younger than those with OFTT.

Socioeconomic factors affecting FTT

1. Socioeconomic level

During the 20th century, social, political and economic changes, influenced the growth and development of public health nutritional services. Such changes included the high rates of diseases and deaths among mothers and children, the continuing presence of poverty and hunger, lack of parental education, low parental occupational status (70% manual workers), increasing number of children in the families leading to a poor hygienic and socioemotional life, especially with the low family incomes, and finally the lack of a high quality public health nutritional services.

In our study, comparison of the socioeconomic level between control, NOFTT and OFTT groups was not significant. Collection of the FTT cases were based on weight-for-age (WA) and not on a socioeconomic base. Around 70% of the children in the three groups were of a low socioeconomic level and the rest was from middle socioeconomic level. Socioeconomic deprivation is associated with poverty, ignorance, unhealthy living conditions, large family size, family instability (divorce or separation) and insufficient nutrient intake which may lead to FTT.

Alleyne (1981), reported that the lower the socioeconomic state of the mother, the higher the risk of having a child with FTT.

2. Family size

In our study, we found that the effect of family size was a highly significant factor affecting FTT (P-value 0.002). Big family size (3-4 children) and (5 or more children) were more frequent in OFTT group than NOFTT group, while small family size (1-2 children) was more common in NOFTT group than OFTT group. Asking the mothers why do they keep on having children when they have so little to give them, 20% of the mothers refused to answer, 50% said it was God's will, 20% disagreed with the means of contraception and 10% wanted to be sure that at least some of their children will survive to contribute to the family income.

Our study coincides with the studies done by ***Richman (1977) and Ballard & Neumann (1995).*** They reported that there is a tendency for larger families to be more likely to have a child with increased risk of FTT and behavioral problem.

3. Marital status

In our study, comparison of marital status in NOFTT, OFTT and control groups was not significant.

Our study differed from that done by ***Richman (1977),*** who found a significant association between a poor marital relationship and the presence of FTT and behavioral disturbance in the child. The marital relationship of FTT parents has been described as dysfunctional or nonexistent by many

studies including that of ***Kerr et al. (1978), Mitchell et al. (1980), Homer & Ledwig (1981) and Altemeier et al. (1985).*** However our study coincides with that done by ***Casey et al. (1984),*** which found no difference in maternal marital status between FTT and control group.

4. Family income

In our study, low family income was a significant factor affecting FTT, mainly through malnutrition (P-value <0.001). It was also lower in OFTT than in NOFTT group, putting the parents in a difficult situation even to obtain basic medical services for their sick children.

Our study coincides with other studies showing that poverty is the most important single social risk factor for FTT, because of the close association between poverty and childhood malnutrition (***Casey et al. 1984, Listernick et al. 1985, Groenewold & Tilahun 1990 and Newacheck, 1994).***

5. Father and mother occupation and education

In our study, comparison of the parental occupation and level of education between NOFTT, OFTT and control groups was not significant. Around 75% of parents in NOFTT group and 63% of parents in OFTT group were non educated (did not complete primary education or illiterate), which is cosidered a high percentage. These uneducated parents lacked the knowledge about the nutritional requirements of children, nutritional value of foods, immunizations, hygiene, oral rehydration and diarrhea. They did not realize

the economic limitation of trying to rear and educate an over-large family. Seventy three percent of the fathers in NOFTT group were manual workers with low income and were illiterate, 23% did desk works and completed 2ry school, while 4% only were graduated from university and were government employees. So, the big majority of the fathers were poor and ignorant, which definitely affects the development and education of their outcomes. Nearly 77% of the mothers in NOFTT group were illiterate and housewives, 23% completed 2ry school (15% of them were manual workers). Results were nearly similar in both OFTT and NOFTT groups.

Other studies showed a strong association between FTT and the educational level of both parents (***Gupta et al. 1991, Bouvier et al. 1995).***

Drotar et al. (1990), found that families of children with FTT have lower occupational status, lower intellectual and cultural orientation than that of families of healthy children.

Nutritional assessment and failure to thrive

1. Anthropometric assessment

The ability to assess the anthropometric results depends essentially on having valid reference values to which actual measurements may be compared. The use of the National Center of Health Statistics (NCHS) reference values, has been accepted by WHO to be internationally used as

reference standards ***(WHO, 1986).*** Accordingly in this study, evaluation and analysis of the results of the anthropometric measurements were done in relation to NCHS standards, by using the "Anthro" computer program, version 1.01.

Because by definition, most children identified as having FTT have Wt or Ht below the fifth percentile on the NCHS charts, additional calculations were necessary to determine the severity of nutritional risk, as a guide to clinical care. Evidence of growth failure was determined in this study by the analysis of weight-for-age (WA), height-for-age (HA) and weight-for-height (WH) indices. Each of these indices was related to NCHS reference standards, by either its position within the centile distribution "percentiles (P)", or as standard deviation score "Z score" or as percentage of the reference "median (M)". Categorizing growth according to standard deviation units (Z scores) is the most accurate technique for classifying growth deficits for research purposes ***(Peterson et al., 1984).***

WA was analyzed in children of the three groups (NOFTT, OFTT and controls) by the three procedures. Results demonstrated that **WAZ** (-2.4 in NOFTT and -2.3 in OFTT), **WAP** (1.3 in NOFTT and 1.4 in OFTT) and **WAM** (75.7% in NOFTT and 74.4% in OFTT) were significantly reduced in the NOFTT and OFTT children than those of the control groups (Table 26). In both affected groups, WAZ was lower than -2 ,WAP was lower than the 5th percentile and WAM was considerably lower than the cut-off (80% of the median).

HA is a determinant of chronic, ongoing malnutrition which ultimately leads to a status known as "stunting". Thus, when constitutional causes can be ruled out, depressed HA is considered as evidence of chronic malnutrition ***(Tenore & Vargus, 1993).*** Results demonstrated that each **HAZ** (-1.5 in NOFTT and -1.7 in OFTT), **HAP** (13.1 in NOFTT and 7.6 in OFTT) and **HAM** (94% in NOFTT and 92.5% in OFTT) were significantly reduced in the NOFTT and OFTT groups (Table 27), but were above the cut-off values. This means that chronic malnutrition is an important factor leading to FTT.

WH is an index that describes the current health status. Results demonstrated that each **WHZ** (-1.7 in NOFTT and -1.6 in OFTT), **WHP** (6.6 in NOFTT and 7.5 in OFTT) and **WHM** (84% in NOFTT and 85.6% in OFTT) were significantly reduced in the NOFTT and OFTT groups (Table 28), but were above the cut-off values. However, there was no difference between the NOFTT and OFTT groups (Table 28). In controls, all the three WH indices were away above the cut-off values, thus indicating an adequate current nutritional status of this group, satisfactory to be used for comparison in the rest of the study. In some cases correct ages of children were not easy to obtain. Since Wt / Ht index is independent of age, and takes into consideration weight in relation to height, the index may be considered to have advantage over using Wt / age ***(Visweswara & Prathad, 1975).***

WA, HA and WH measurements were similarly affected in both OFTT and NOFTT groups, so, they could not be considered as points of differentiation between the 2 groups. This confirms the importance of the

clinical examination and the psychosocial history in determination of the 2 groups.

Midarm circumference (MAC) is a method that is currently widely used for detecting malnutrition. Taking 12.5 cm as a cut-off point below which the child is identified as malnutrition ***(Pust et al., 1992),*** our results showed that in NOFTT the value were far lower than 12.5 cm. In the control group, the mean was significantly higher. Previous studies suggested that MAC measurements are unreliable for determining the severe malnourished state, and that the use of Wt was preferable in determining the nutritional status ***(Anderson et al., 1990).*** The same observation was shown in this study by using MAC versus WAZ (table 30). Children with WAZ below the cut-off values (-2) were 87% in NOFTT group and 84% in OFTT group, while those with MAC below the cut-off values (-12.5 cm) were 40% in NOFTT group and 27% in OFTT group.

Mody mass index (BMI) is an important noninvasive clinically convenient manner of assessing chronic energy deficiency in children above 1 year of age. BMI was significantly reduced in both NOFTT and OFTT groups, this means that chronic malnutrition is an important factor leading to FTT.

Triceps skinfold (TSF) measurements were significantly lower in both NOFTT and OFTT groups than in controls (P-value <0.001). Expressing TSF as a percentage of the standard skinfold thickness of the same age, according to Frisancho's standards (Appendix 8), it was 68% in NOFTT,

8o% in OFTT and 92% in controls. Estimation of the nutritional status of our cases according to the classification done by ***Malina (1972)*** shown in (table 6), NOFTT and OFTT children were considered having "moderate malnutrition" while controls were considered "normal".

2. Biochemical assessment: (Serum albumin)

Plasma albumin level in all the three groups were greater than 2.5 g%, except 9 children in NOFTT group who had plasma albumin level less than 2.5 g%, they were diagnosed as Kwashiorkor. Carbohydrate intake (in the form of rice, potatoes and sugared water) constituted the major part of the caloric intake of these children (70%), proteins (8%) and fats (12%). Low plasma albumin levels has been attributed to adequate energy in the form carbohydrates but deficient proteins in diet of these children, while when both carbohydrates and proteins were deficient (e.g. marasmic children), albumin level was within the normal range ***(Lunn & Austin, 1983).*** There was no difference in serum albumin level between NOFTT and OFTT groups, but it was lower in both affected groups than that in control group. This is due to the increased protein intake in control group than in both affected groups. Hypoalbuminemia can lead to anemia and affects the level of immunoglobulins, increasing the risk of infections.

3. Dietary intake

A single food item cannot possibly provide all of the essential nutrients in the amounts required. The best way to insure an adequate diet is to choose a wide variety of foods from all food groups that are available. Children

require sufficient calories for growth and the maintenance of body functions. Ingestion of unsaturated fatty acids and consumption of carbohydrates, minerals, proteins are essential for optimal growth and development. Approximately 9-15% of the calories required are derived from protein, 45-55% are derived from carbohydrates and 35-45% are derived from fat ***(Food and Nutrition Board, 1980).***

Carbohydrates are the essential fuel in the first days of life, to prevent hypoglycemia. Subsequent delivery of protein and fat help rectify reduced muscle and fat stores, and promote weight gain.

Fat is necessary in the diet of infants and young children because of their extraordinary energy needs and limited dietary capacity. In addition, deficiencies in the amounts of long-chain fatty acids in the diet during infancy may affect the maturation of the central nervous system, including visual development and intelligence. Therefore fat should not be restricted in the diet of infants and young children.

Proteins supply amino acids for growth and repair of tissue cells. It is an important component in the formation of hemoglobin, enzymes, hormones and antibodies. In addition it is one of the main sources of energy.

In our study, the mean total caloric intake was less in both NOFTT and OFTT children than in controls, and was less in NOFTT than in OFTT group. The caloric deficit was more in both affected groups than in controls. The actual caloric intake was much lower than the nutritional requirements of

children in both NOFTT and OFTT groups. The nutritional index was estimated in the three groups and was found to be -46.5% in NOFTT group, -42.7% in OFTT, while it was only -11.2% in controls. Malnutrition in NOFTT is due to impoverishment, inadequate education and dysfunctional relationships in the family, while malnutrition in OFTT may be related either to decreased energy intake from anorexia accompanying most of the organic diseases, and/or to increased energy requirements such as the increased respiratory rate accompanying pulmonary disease or congestive heart failure. This finding is similar to the study done by ***Batchelor & Kerslake (1990),*** concluding that undernutrition is a common factor in both affected groups, and that a child having an organic disease might as well have an additional problem with feeding.

In our study, 80% of children in both affected groups and in controls were breast-fed, so breast feeding was not a significant factor differentiating between the 3 groups. On the other hand, the duration of breast feeding varied from one group to another, especially those who were breast-fed for more than 1 year (61% in NOFTT, 28% in OFTT and 18% in controls). Prolonged breast feeding might enhance the maternal-child attachment, but may also be an important factor affecting the caloric intake, especially if the child is depending mainly on breast milk, without any food supplementation.

Our study contradicts a study done by ***Serventi et al. (1995),*** which proved that a history of early weaning from the breast before 2 years, was followed by a drastic drop of the growth curve, due to recurrent attacks of gastro-enteritis resulting from poor hygiene. In another study done by

Herman-Staab (1992) on a NOFTT group of children, breast fed infants were 41% in NOFTT and only 7% in controls. This finding was surprising in comparison to a study done by ***Drotar et al. (1990)*** that considered breast feeding to enhance maternal-child attachment. ***Nelson (1993)*** commented on the statement of Herman-Staab (1992) regarding the high percentage of NOFTT babies that were breast-fed, that the growth pattern for weight of breast-fed infants differed from current U.S. National Center of Health Statistics (NCHS) reference data, and was less than that of their formula-fed peers. Breast-fed and formula-fed infants had similar Wt gain for the first 3 months, then the Wt gain decreased in breast-fed infants. Due to this slower rate of growth, breast fed infants appear to be faltering when compared to NCHS charts, even if they are healthy and thriving. The NCHS reference data are based on informations collected from 1929 to 1975, from infants who were primarily bottle-fed. Nelson (1993) recommends news charts to be done, that reflect breast-fed infants' actual growth patterns.

In our study, onset of most of the cases of PEM (78%) followed an acute episode of gastro-enteritis. Mothers believed that restriction of food was the ideal way to stop the diarrhea, leading to severe malnutrition, enough to produce growth failure. It may also impair immune function. With each infection, the child's appetite and nutrient intake decrease , while nutrient requirements are increased by fever, diarrhea and vomiting. Unless attention is paid to restore the child's nutritional status as well as treating the acute illness, cumulative nutritional deficits occur, leaving the child increasingly vulnerable to more severe and prolonged infections and to even less adequate growth.

A similar study to ours was done by ***Jeffrey et al. (1994),*** and found that diarrhea is a leading cause of failure to thrive in children below the age of 5 years. The peak incidence is during the weaning period and is obviously related to the introduction of weaning foods which are contaminated.

4. Anorexia

Anorexia is a highly significant factor associated with FTT (P-value = 0.001). It was more common in both NOFTT and OFTT children than in controls.

As mentioned before, most cases of NOFTT due to PEM followed an acute episode of diarrhea. Diarrheal diseases have a well recognized negative effect on children's growth, probably due in part to severe anorexia during illness which is worsened by restriction of diet by the mother, impaired digestion and absorption, nutrient losses and altered immune responses. Acidosis and dehydration are most closely associated with anorexia of children with diarrheal diseases. Clinical trials done by ***Brown (1994),*** indicated that children should be fed continuously during diarrheal episodes with their usual diets. Breastfed infants should continue to nurse at the breast during diarrhea. However, children with more severe diarrhea and dehydration, may have a slightly increased rate of complications and would, therefore, benefit from a modification of their diet.

In children suffering from NOFTT due to environmental deprivation or constitutional FTT, anorexia is a very common complaint and is due to psychological disturbances in these children.

In OFTT, anorexia is also a frequent symptom in most of the organic diseases, leading to severe malnutrition and FTT. This was present in our study, in the group of children with neoplastic diseases such as leukemia, lymphoma and Wilm's tumor. Growth failure in children with respiratory infections such as tuberculosis and bronchiectasis, was due to severe anorexia and increased energy expenditure. ***Grantham et al. (1993)***, reported that respiratory infection and undernutrition in young children act synergistically.

A study was done by ***Bryant et al. (1992),*** to determine whether doctors recognize anorexia in children as a part of eating disorders, especially anorexia of psychological origin. A group of pediatricians, general practitioners and school medical officers was approached to participate in this study. The response rate was 64.5%. These results suggest that doctor's awareness of childhood onset eating disorders, especially anorexia, remain limited.

5. Anemia

Anemia (hemoglobin <11g%) was a highly significant factor in failure to thrive (P-value <0.001). Both NOFTT and OFTT groups were similarly affected. It is a major problem among children with anorexia, malnutrition and growth failure. Anemia is an important factor affecting intellectual

performance and schooling. Iron deficiency anemia is the commonest type of anemia in cases of failure to thrive and malnutrition, due to insufficient iron intake and prolonged breast feeding. It is also expected in malnourished infants during the period of catch-up growth if sufficient supply is not provided.

Our study is similar to a study done by ***Van Den Broeck et al. (1993),*** reporting that anemia is one of the commonest factors affecting FTT and malnutrition.

Psychological assessment and failure to thrive

1. Maternal depression

In our study, we used the Edinburgh Postnatal Depression Scale (EPDS) to identify mothers suffering from depression. Taking a score of > or = 13 as a cut-off point, below which the mother is identified as being depressed, we found that 59.1% of mothers in NOFTT group and 53.6% of mothers in OFTT group were depressed , while depressed mothers in control group represented 27.5% only, which is significant (p-value = 0.008). Most of the mothers were depressed because of the frequent arguments with their husbands, and low satisfaction with the amount of help they received from their husbands, whether it was moral or financial support. The low family income was also an important factor, especially in families with a large number of children. Depression in mothers of children with OFTT was due to the organic disease of their children. The mother is the first and most effective

person in giving care and affection to her child, but a depressed mother would never be able to express her feelings of love, or to be patient and tolerable with her infant, if he is difficult to feed. Fifteen per cent of the depressed mothers did not give their child any physical affection like cuddles or caressing, because they did not feel like they wanted to do it or because they did not have time. Effect of maternal physical interaction and physical affection on FTT, was also proved by ***Polar & Ward, (1994).***

Brown et al. (1975), reported a striking finding regarding the high rate of depression in mothers of 3 years old children. It may be that having a preschool child is in itself particularly stressful, and increases the risk of developing depression in the vulnerable mother because of the physical and emotional demands.

An infant's mother may be depressed and this may result in a tense child who consequently does not feed well. The mental state of the mother may render her intolerant of the behavior of her child who does not eat, and as a result she habitually stops the feed prematurely, leaving her child angry and hungry. A vicious cycle then develops, to which both infant and caregiver contribute ***(Skuse, 1985).***

Schaper et al. (1994), evaluated postnatal depression in 287 mothers randomly selected from the total population completing EPDS, and they identified 17.4 % of these women having an EPDS > or = 13, indicating significant depression and a potential risk of having children with growth retardation and social behavioral affection.

Hay & Kumar (1995), interpreted the effects of mother's postnatal depression on FTT children's intelligence, and found that there was an association between maternal postnatal depression, FTT and impairement of children's cognitive abilities.

Previous studies done by ***Brown et al. (1975), Skuse (1985), Schaper et al. (1994) and Hay & Kumar (1995),*** including our study, they all share the same idea which is the close association between maternal depression and the risk of having children with growth retardation and social behavioral affection.

2. Behavioral and psychological affection of the child

In psychological and behavior study of the children, informations were gathered from the child and his mother, following the usual clinical practice in child psychiatry. At the age of 2-3 years, the child is unlikely to be able to describe his emotions and relationships, but one can however observe how the child interacts with his caretakers, his play, his speech, his activity level and his relationship with the interviewer in a particular social context. Care should be taken, that the assessment of the psychological behavior of the infants has been based mostly on the point of view of their mothers, who may have little opportunity to see their child in a wider social context. In addition that these mothers who have a high incidence of depression, might consider their infants as being difficult and unpleasant. These children may also be subjected to a lack of appropriate emotional and intellectual stimulation in

their homes. A study of the emotional status of 24 children (average age 5 years old) was done by ***Hufton & Oates (1977)*** and found that 90% of the children were emotionally abnormal if the interview was completed by the children's mothers, while 50% were emotionally affected if the interview was completed by the children's teachers.

In our study, 59.1% of OFTT children and 52.1% of NOFTT children had behavioral affection, while 15% of controls were affected. So, both NOFTT and OFTT groups were significantly affected (P-value = 0.001). Preschool children with behavioral affection may have problems in socioemotional development and learning.

Richman et al. (1975), found the prevalence of behavioral problems in preschool children to be 17-18%. Our results were different from the studies done by ***Powell et al. (1987), Budd et al. (1992) and Reifsnider (1995),*** in which children with NOFTT were more psychologically affected than those with OFTT. On the other hand, our study was similar to that done by ***Singer et al. (1990),*** reporting that FTT children were more stressful, less adaptable, more inconsolable and more unhappy than the healthy children, and was also similar to the research done by ***Drotar & Sturm. (1992),*** which demonstrated more behavioral deficits in NOFTT children.

Table (43) shows the types of behavioral affection in our study:

Mental retardation was found in 11 cases of OFTT distributed as follows: post-traumatic cerebral palsy (2 cases), post-inflammatory cerebral palsy (2 cases), Down syndrome (3 cases), congenital cerebral palsy (1 case), cretinism (1 case), microcephaly (1 case) and multiple congenital anomalies (1 case).

Autism was found in a 44 months old boy, suffering from NOFTT due to malnutrition. He was diagnosed and followed up in the psychiatric outpatient clinic of Kasr El Eini children's hospital. The mother complained that her son failed to smile or to respond to the smiling of others, never showed her any sort of affection like kissing or hugs, wanted to play with the same toys and refused to share other children in their play. He had delayed speech and had an expressive vocabulary difficult to understand. His intellectual potential was below average as shown by his IQ which was 85.

Speech problem was found in 3 children, 2 of them had NOFTT (a 5 years old girl with separation anxiety "page 177" and a 44 months old boy with autism). They had delayed speech. They appeared to understand language, but their own speech was hard to understand because the words were ill-formed. The third child had OFTT (a 6 years old boy with rheumatic heart disease). He had severe stuttering.

Conduct disorders were present in 9 children with NOFTT and 9 children with OFTT, mainly in the form of lying, stealing, spitting, insulting

people and fighting with other children. Only a 6 years old boy, suffering from tuberculosis, had the pleasure of setting fire in newspapers, tissues and even flies after catching them (Table 42).

Attention deficit hyperactivity disorder (ADHD) was found in 3 children, (a case of NOFTT in a 5 years old girl suffering from separation anxiety, and 2 boys having OFTT "a 4 years old boy with Down syndrome and a 3 years old boy with thalassemia major"). The most common characteristics of these children were inattention, disobeying, touching everything and hyperactivity to the extent of disturbing surrounding people.

Anxiety disorders were present in 4 children with FTT. First one was a case of NOFTT in a 5 years old girl, suffering from separation anxiety (discussed before, page 177). Her father died 8 months ago, and since that time she keeps crying, she became very attached to her mother and wants to be with her all the time. She wakes up every night from nightmares and keeps asking her mother about her father and the reason he died. She also has severe anorexia.

The second and third cases suffering from anxiety were 2 brothers (5 and 6 years old) with short stature (discussed before, page 178). These two brothers always refused to go to school. They feel most of the time that they are socially isolated, and that they are at risk both of being teased and of being treated as the "baby". They are hypersensitive to criticism and have a low self-esteem. Our findings regarding the psychological behavior of children with short stature are similar to many other studies which proved that

these children are often socially isolated and have poorer attention than the controls ***(Young-Hyman, 1986 and Skuse et al., 1994). Holmes et al., (1984)*** found a tendency for exceptionally short children to be kept down a grade at school in comparison with their chronological age, in an attempt to match the height age, to avoid them from being socially isolated.

The fourth case was OFTT in a 4 years old boy, suffering from tuberculosis. He avoided being alone and had frequent nightmares.

A study was conducted to assess quality of mother-infant attachment in preschool children of different nutritional status. Around 43 nutritionally healthy and 42 chronically underweight children were seen with their mothers. They were classified in the "secure" and " anxious" attachment patterns. Results showed a greater proportion (93%) of anxious attachments in the underweight group as compared to the group of children without a history of nutritional deficits (50%). Children classified as "anxious" presented the most serious Wt deficits within the underweight group, indicating an association between severity of the nutritional deficits and insecure/disorganized attachments ***(Valenzuela, 1990).***

Eating disorder was seen in a 4 years old boy, having an allergy to most foods (Page 175). One day he had an attack of angioneurotic edema following chocolate ingestion. The parents convinced their son that he could die if he eats anything without the permission of his mother, to prevent him from eating the types of food he was allergic to. So, the boy was afraid of eating, resulting into a "psychological anorexia".

Sleeping disorders were present in 8 children in the form of nightmares. The 44 months old old boy with autism (page 198) and the 4 children discussed before in anxiety disorders (page 199) had nightmares. A 3 years old girl with NOFTT due to nutritional marasmus had a frank history of nightmares almost every night. A 6 years old girl, neglected by her alcoholic father and depressed mother (page 177) and a 5 years old girl whose father is an addict (page 178) had also very frequent nightmares. Anxiety and sleeping disorders were more frequent in NOFTT than OFTT children.

It was difficult to apply the Child Assessment Schedule (CAS) in children between 2 and 3 years old, because of their young age and their inability to answer the questions. Instead, we tried to assess their sociability by using the procedure devised by Stevenson & Lamb (1979). Fifteen children in NOFTT group and 5 children in OFTT group were quite unfriendly, unsocial and fearful.

Environmental and psychological deprivation were the major etiological factors leading to NOFTT in 4 children of our study (Table 17). The different types of behavioral and psychological affection shown in table (42) were detected during the psychological evaluation of the FTT cases. We may consider that FTT might be a precipitating factor for different forms of behavioral disturbances to occur in these children, in addition to the disturbance of the mother-child relatioship in some cases.

Finally, table (43) shows a highly significant positive relation between maternal depression, behavioral and psychological affection of their children. In NOFTT group, behavioral and psychological affection were present in 83% of children with depressed mothers, while they were present in only 16% of those with non depressed mothers. In OFTT group of children, behavioral affection was present in 96.2% of children with depressed mothers, while it was only present in 5.6% of those with non depressed mothers.

In conclusion, our study shows that both NOFTT and OFTT represent a major social problem, that requires special attention from the care takers. FTT has multiple causes which include malnutrition, poverty, ignorance and infections. Therefore, a general developmental programme is needed, which aims at raising family incomes, nutritional education and protecting family health. It has to be well planned between pediatricians, nutritionists, psychiatrists and social workers.

Summary

Summary

Failure to thrive (FTT) in infancy and early childhood challenges the diagnostic and therapeutic skills of the most experienced pediatricians. In broad terms, FTT refers to infants and children whose weight is below the 5th percentile for age. This common condition is of great concern to clinicians, because FTT in early life identifies children who are at high risk for lasting deficits in growth, cognition and socioemotional functioning.

The aim of this work was to evaluate the different etiological factors of FTT, assess the social behavior of the child, evaluate the maternal psychological status and study the effect of these different factors on FTT. The study comprised 100 preschool children between 2 and 6 years old.

In this study, the causes of FTT are dichotomized as organic (44%) and nonorganic (56%) failure to thrive. Organic failure to thrive (OFTT) is ascribed to a major illness or organ system dysfunction thought to be sufficient to account for growth failure, such as gastro-intestinal, neurological, cardiovascular, pulmonary, renal, endocrinal and chromosomal diseases. In contrast, nonorganic failure to thrive (NOFTT) is ascribed to environmental deprivation, simple feeding problems and constitutional growth delay.

Malnutrition was identified as the critical biologic factor in all cases of FTT. It was the result of poverty, low family income, ignorance of the mother about the basics of child care and feeding, anorexia affecting nearly 80% of children with FTT, very young mothers who were not at the level of being responsible of a child, and finally the psychological state of the mothers suffering of depression (55%) which impairs the mother's ability to function in her many roles.

The concept of maternal deprivation was put into consideration as a framework for understanding the psychological correlates and socioeconomic consequences of FTT. Behavioral affection of the child and the psychological state of the mother were studied, and there was a marked association between family disturbance (especially maternal depression) and the behavioral or emotional problems of the child with FTT.

So, a knowledge and awareness of the way in which social and familial factors affect very young children, is of importance for the furtherance of health and social policies.

Conclusion

Conclusion

1. Clinical examination and careful psychological history are the main determinants in the classification of failure to thrive (FTT).

2. Non-organic failure to thrive (NOFTT) is more common than organic failure to thrive (OFTT).

3. Diminished caloric intake is the root cause in both NOFTT and OFTT cases.

4. FTT in a breast-fed infants could be attributed to reduced milk production in conjunction with maternal dietary energy and protein restriction.

5. Low family income and big family size are important factors contributing to FTT.

6. Anemia is a major problem that requires a prompt attention in children with FTT.

7. There is a significant positive association between maternal depression and behavioral affection of children with FTT.

Recommendations

Recommendations

1. There is a critical need for early detection of children with FTT, and early aggressive nutritional intervention in such infants. A significant increase in caloric intake will lead to a prompt improvement in growth percentiles.

2. Increased attention to the importance of the psychological evaluation of cases with FTT, to detect any socioemotional defect in parent-child relationship.

3. The nutritional problems of developing countries are conditioned by poverty and ignorance. The educational level and nutrition knowledge of parents influence the nutritional quality of the diet offered to their children. Child care programs require systematic diagnosis of the existing situation and appropriate intervention such as improving economic conditions, promoting breast feeding, delaying introduction of nonhuman milk and providing parents with important information about the nutrients children need and the foods that provide these nutrients.

4. The tremendous health impact of diarrhea on both morbidity and mortality in children of our community must be recognized and controlled, along with correction of food shortages in order to improve the nutrition, growth and survival of impoverished children.

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Appendix

Appendix 1

Socioeconomic record

(A) Father data

. Age: (in years)
. Ocupation & education:
(1) Manual worker or did not complete 1ry education or illitrate.
(2) Skilled labourer or 2ry school.
(3) Government employee or university graduate.

(B) Mother data

. Age: (in years)
. Ocupation & education:
(1) Manual worker or did not complete 1ry education or illitrate.
(2) Skilled labourer or 2ry school.
(3) Government employee or university graduate.

(C) Family size

(1) 5 or more children.
(2) 3-4 children.
(3) 1-2 children.

(D) Family income

(1) <100 LE
(2) 100-200 LE
(3) >200 LE

(E) Crowdness of the house

(1) >4 members/room.
(2) 3-4 members/room.
(3) 1-2 members/room.

(F) Total score

(1) High = score 13-15
(2) Middle = score 9-12
(3) Low = score below 9

Appendix 2

Nutrition screening questionnaire for young preschool children (Fomon,1976)

1. Does the child drink milk? (1. Yes 2. No)

If yes

What type of milk? (1.Breast milk 2.Whole milk 3. Half cream 4.Powder milk 5.Breast & artificial)

If yes

Amount of milk if artificial? (1.<240ml 2. 240-1000ml 3.>1000ml)

2. How many times a day does the child usually eat? .....

3.What kind of fluids besides milk does the child usually drink?
(1.water 2.juices 3.none)

4.Please indicate which (if any) of these foods the child eats?

	Day1	Day2	Day3
-Cheese	.....	.....	.....
-Yogurt	.....	.....	.....
-Eggs	.....	.....	.....
-Beans	.....	.....	.....
-Meat, fish, poultry	.....	.....	.....
-Bread	.....	.....	.....
-Rice	.....	.....	.....
-Cereals	.....	.....	.....
-Pasta	.....	.....	.....
-Potatoes	.....	.....	.....
-Fruits	.....	.....	.....
-Vegetables	.....	.....	.....
-Others	.....	.....	.....

5. Does the child take vitamins or iron? (1.Yes 2.No)

6. How would you describe the child's appetite? (1.Good 2.Poor)

7. Is the child on a special diet? (1.Yes 2.No)

If Yes

What is the reason? (1.Allergy 2.Wt reduction 3.Other)

8. Does the child eat clay, paint chips, dirt, or anything else that is not usually considered food? (1.Yes 2.No)

If Yes

What? .....

Appendix3

Clinical history

1. Respiratory: (a.cough b.runny nose c.wheezes d.NAD)

2. Cardiac: (a.dyspnea b. cyanosis c.palpitations d.NAD)

3. Gastro-intestinal: (a.vomiting b.diarrhea c.vomiting & diarrhea d.blood in stools e.constipation f.NAD)

4. Nervous: (a.drowsiness b.convulsions c.headache d.paralysis e. abnormal gait f.NAD)

5. Loss of appetite: (a.yes b.No)

6. Family history: (a.similar condition b.no similar condition)

Clinical examination

1. General appearance: (a.edematous b.growth failure c.normal appearance)

2. Mucous membrane: (a.pallor b.cyanosis c.NAD)

3. Skin: (a.loss of subcutaneous fat b.petichae c.macerated d.intact)

4. Hair: (a.altered colour b.NAD)

5. Nails: (a.Pallor b.cyanosis c.clubbing d.koylonichia e.NAD)

6. Teeth: (a.caries b.NAD)

7. Cardiac examination: (a.murmur b.arrhythmia c.NAD)

8. Chest examination: (a.crepitations b.wheezes c.bronchial breathing d.NAD)

. Abdominal examination: (a.hepatomegaly b.splenomegaly c.hepatosplenomegaly d.ascites e.NAD)

. Neurological: (a.hemiplegia b paraplegia c.hypertonia d.hypotonia e. NAD)

Appendix 4

The procedure for assessing the child's sociability to a strangrer (Stevenson & Lamb,1979)

Each response by the child is rated on a 1-5 scale as shown and then an overall impression is rated.

(a) Baby's initial reaction to being offered a toy by stranger on mother's lap:

(1) Cries.
(2) Refuses toy by looking away or pushing it away.
(3) Looks at toy without reaching for it.
(4) Reaches for or touches it.
(5) Accepts toy without hesitation.

(b) Baby's reaction to the stranger's initiation of a give-and-take exchange on mother's lap:

(1) Cries.
(2) Refuses to join.
(3) Initially reluctant, then participates.
(4) Immediately joins and participates.
(5) Actively participates by smiling or acting playfully.

(c) Baby's behavior when given floor freedom:

(1) Touches mother or requests to return to lap.
(2) Approaches or turns towards mother.
(3) Stays where he is or moves away from mother to play.
(4) Approaches stranger.
(5) Touches stranger or requests to be picked up.

(d) Baby's initial reaction to being offered a toy by stranger on floor:

(1) Cries.
(2) Refuses toy by looking away or pushing it away.
(3) Looks at toy without reaching for it.
(4) Reaches for or touches it.
(5) Accepts toy without hesitation.

(e) Baby's reaction to the stranger's initiation of a give-and-take exchange on the floor:

(1) Cries.
(2) Refuses tojoin.
(3) Initially reluctant, then participates.
(4) Immediately joins and participates.
(5) Actively participates by smiling or acting playfully

Appendix 5

Child Assessment Schedule
Diagnostic interview
(Hodges et al., 1982)

Questions	No	Yes	No respose	Not aplicable
PART I				
Sample from "Fears and Anxieties"				
1. Most people are afraid of something?	.....	.....	.....	.....
Do you also fear of something?				
2. Do you take long time to initiate sleeping?				
3. Do you get nightmares?	.....	.....	.....	.....
4. Do you see quarrels between your father and mother?	.....	.....	.....	.....
5. Do you like going to school?	.....	.....	.....	.....
6. Do you have a lot of friends?	.....	.....	.....	.....
7. Do you cry every day?	.....	.....	.....	.....
8. Do you think about death?	.....	.....	.....	.....
9. Do you like playing with others?	.....	.....	.....	.....
10. Do you prefer playing alone?	.....	.....	.....	.....
Sample from "Expression of Anger"				
11.Do you often get angry?	.....	.....	.....	.....
12.Do you have trouble following rules at school?	.....	.....	.....	.....
13.Do you have trouble following rules at home?	.....	.....	.....	.....
14.Have you ever been thought of as a troublemaker?	.....	.....	.....	.....
15.If yes, what type of troublemaker?				
-Stealing?	.....	.....	.....	.....
-Physical violence?				
-Verbal fighting?	.....	.....	.....	.....
-Persistent lying?				
-Physical violence?	.....	.....	.....	.....
Part II				
Impressions about quality of interpersonal interactions				
1. Stubborn?	.....	.....	.....	.....
2. Argumentative?	.....	.....	.....	.....
3. Not interested in rapport?	.....	.....	.....	.....
4. Shye?	.....	.....	.....	.....

Appendix 6.

Edinburgh Postnatal Depression Scale (EPDS) (Cox et al., 1987)

After delivery:

1. Have you been able to laugh and see the funny side of things?
0 As much as I always could.
1 Not quite so much now.
2 Definitely not so much now.
3 Not at all.

2. Do you enjoy life as you did before delivery?
0 As much as I ever did.
1 Rather less than I used to.
2 Definitely less than I used to.
3 Hardly at all.

3. Have you blamed yourself unnecessarily when things went wrong?
3 Yes, most of the time.
2 Yes, some of the time.
1 Not very often.
0 No, never.

4. Have you been anxious or worried for no good reason?
0 No, not at all.
1 Very rare.
2 Yes, sometimes.
3 Yes, very often.

5. Have you felt scared or panicky for no very good reason?
3 Yes, quite a lot.
2 Yes, sometimes.
1 No, not much.
0 No, not at all.

6. Do you feel like things have been getting on top of you?
3 Yes, most of the time I haven't been able to cope at all.
2 Yes, sometimes I haven't been coping as well as usual.
1 No, most of the time I have coped quite well.
0 No, I have been coping as well as ever.

7. Have you been so unhappy that you have had difficulty sleeping?
3 Yes, most of the time.
2 Yes, sometimes.
1 Not very often.
0 No, not at all.

8. Have you felt sad or miserable?
3 Yes, most of the time.
2 Yes, sometimes.
1 Not very often.
0 No, not at all.

9. Have you been so unhappy that you have been crying?
3 Yes, most of the time.
2 Yes, sometimes.
1 Only occasionally.
0 No, never.

10. Have you thought of harming yourself?
3 Yes, quite often.
2 Yes, sometimes.
1 Hardly ever.
0 Never

Appendix 7

Psychiatric sheet

1. Physical complaints: (a.headaches b.stomachaches c.hearing defect d.blurring of vision e.seizures)

2. Eating disorders:

3. Sleeping disorders: (a.difficulty to start sleep b.nightmares c.NAD)

4. Level of activity: (a.active b.lethargic)

5. Mood: (a.happy b.sad c.depressed d.anxious e.fearful)

6. Antisocial behavior: (a.aggressive b.stealing c.truancy)

7. Speech disorders: (a.delayed speech b.stuttering c.NAD)

8. Family structure: (a.married b.divorced c.widow d.separated)

9. Quality of parental relationships: (a.mutual affection b.Quarrels c.addiction d.alcoholism)

10. Quality of parent-child relationship: (a.positive interaction b.rejection)

11. Schooling history: (a.easy attendance b.retarded progress in school work c.does not go to school)

Appendix 8

Percentiles of triceps skinfold in male preschool children
Health and Nutrition examination survey
Frisancho, (1981)

Age (years)	5	10	25	50	75	90	95
2-3	6	7	8	10	12	14	15
3-4	6	7	8	10	11	14	15
4-5	6	6	8	9	11	12	14
5-6	6	6	8	9	11	14	15

Percentiles of triceps skinfold in female preschool children
Health and Nutrition examination survey
Frisancho, (1981)

Age (years)	5	10	25	50	75	90	95
2-3	6	8	9	10	12	15	16
3-4	7	8	9	11	12	14	15
4-5	7	8	8	10	12	14	16
5-6	6	7	8	10	12	15	18

Appendix 9

Height/Age Z-score and Weight/Height Z-score in children with constitutional Failure to Thrive

Age (years)	Sex	HAZ	WHZ
30	Male	-1.2	-1.7
30	Female	-1.8	-1.9
36	Female	-1.4	-0.2
60	Female	-3.4	-0.2
60	Male	-3.3	-0.2
72	Male	-3.0	-0.3

(ملخـــــص)

تمثل مشكلة القصور النموى (FTT) فى الطفوله والطفوله المبكره تحديا للمهارات العلاجيه والتشخيصيه لدى أعلى أطباء الاطفال خبره وبمعناها الشامل تعبر مشكلة القصور النمو (FTT) عن الاطفال الذين تقل معدلات اوزانهم عن منحني (5th Percentile) عن معدلاتهم العمريه ، وتمثل هذه الحاله الشائعه أهمية قصوى للمعالجين ، وذلك لأن القصور النموى المبكر فى حياة الاطفال يعتبر مؤشر للاطفال المهددين بخطر العجز فى النمو الجسدى والمعرفى والوظائف النفسيه والاجتماعيه .

وهدف هذا العمل تقييم العوامل المسببه للمرض (القصور) النموى ،حيث يقوم بتقييم السلوك الاجتماعى للطفل وتقييم الحاله النفسيه للامهات ودراسه العوامل المختلفه المسببه للقصور النموى. ويدرس العمل حالات مئه طفل من مرحله ما قبل الدراسه فيما تتراوح اعمارهم بين الثانيه والسادسه .

وتنقسم اسباب القصور النموى الى ٤٤٪ اسباب عضوية و٥٦٪ لاسباب غير عضويه ، ويعزى القصور النموى العضوي لخلل وظيفى فى معظم حالاته المرضيه مثل الخلل المعدى - المعوى والعصبى والاوعيه الدمويه والكلوى أو الغدد او الامراض الخاصه بالكروموسومات.

فى حين أن القصور النموى الغير عضوى يعزى اسبابه لعوامل الحرمان البيئى ومشكلات التغذيه والتأخر فى النمو الناتج عن اسباب معيشيه .

وتعتبر سوء التغذيه أهم الاسباب البيولوجيه المسببه للقصور النمو (FTT) حيث انها نتاج الفقر وانخفاض مستوى دخل الاسره وجهل الامهات بأساسيات تغذيه الاطفال والعنايه بهم ويعانى ٨٠٪ من الاطفال المصابين بالقصور النموى بفقدان الشهيه ، كما يعزى المرض لصغر سن الامهات الذين يعتبرون تحت مستوى المسؤوليه المطلوبه لرعاية الطفل ، واخيراً الحاله النفسيه للامهات الذين يعانون من الاكتئاب (٥٥٪) والذى يؤدى لتقليص الدور الذى تقوم به الام.

وقد تم وضع مفهوم "فقد الام" كأحد أهم العوامل المترابطه مع العوامل السيكلوجيه والاجتماعيه - الاقتصاديه المتسببه فى القصور النموى.

وتم هنا دراسة السلوك الاجتماعى للطفل وحالة الام النفسيه وقد وجد ترابط كبير بين اضطراب الاسره (وخاصه اكتئاب الام) وبين المشاكل السلوكيه او العاطفية للطفل المصاب بالقصور النموى

ولهذا فان المعرفه والوعى بالعوامل الاجتماعيه والاسريه التى يتعرض لها الاطفال تعد على قدر كبير من الاهميه لوضع سياسات صحيه واجتماعيه سليمه .

جامعة عين شمس
معهد الدراسات العليا للطفولة
قسم الدراسات الطبية

أسم الباحث : ناصر جمال فخرى

\عنوان الرسالة : الظواهر الغذائية والنفسية للقصور النموى

تمت هذه الدراسة على مائة حالة من الاطفال الذين يعانون من القصور النموى وتتراوح أعمارهم بين سنتين و٦سنوات وتعريف القصور النموى هو الطفل الذى يكون وزنة أقل من المنحنى الخامس وقد تم دراسة أسباب القصور النموى والظواهر الغذائية والنفسية فى الاطفال الذين يعانون من هذا المرض وتأثير حالة الام النفسية عليهم وقد نتجت هذه الدراسة أن ٤٤٪ من أسباب القصور النموى عضوية و٥٦٪ غير عضوية وأن سوءالتغذية يمثل أهم الاسباب البيولوجية المسببة للقصور النموى حيث انها نتاج الفقر وأنخفاض مستوى دخل الاسرة كما أن هذه الدراسة أثبتت وجود ترابط كبير بين اضطراب الاسرة وخاصة أكتئاب الام وبين المشاكل السلوكية والعاطفية للطفل المصاب بالقصور النموى .

الكلمات المفتاحية :

-القصور النموى
- الظواهر النفسية
_ سؤ التغذية
- القياس الانثروبومترى
-أكتئاب الام
-الحرمان من الام
- فقدان الشهية
- المشاكل السلوكية
- الدخل الاسرى
- حجم الاسرة
- المستوى التعليمى للاسرة .

" جامعة عين شمس "
الكلية :

شكـــــــــــــــر

أشكـــر الســـادة الأســـاتذة الـــذين قامـــوا بالاشـــــراف وهـــم :

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تـم الأشـخـاص الذيـن تعـاونوا معـى فـى البحـث وهـم :

١) د. أمينة حافظ - استاذ مساعد طب الاطفال - المركز القومى للبحوث
٢) د. عفاف كامل - استاذ مساعد الأمراض الجلدية - المركز القومى للبحوث
٣) د. كمال شاروبيم - استاذ

وكـــذلك الممرضـــات :

١) . . . . . .
٢) . . . . . .
٣) . . . . . .

"جامعة عين شمس"
الكلية: معهد الدراسات العليا للطفولة

صفحة العنوان

أسم الطالب : ناجية جمال فخري
الدرجة العلمية : دكتوراه
القسم التابع له : قسم الدراسات الطبية
أسم الكلية : معهد الدراسات العليا للطفولة
الجامعة : عين شمس
سنة التخرج : ١٩٨٩
سنة المنح : ١٩٩٦

" جامعة عين شمس"
الكليـــة:

رسالـــة ماجســـتير / دكتــــوراه

أسم الطـــالب: ناصر جمال فخري
عنوان الرسالة: الظواهر الغذائية والنفسية للقصور الكلوي

أســم الدرجـــة : (~~ماجستير~~ / دكتـــوراه)

لجنــــة الإشــــراف

١- الاسم/ أ. د. رمزي الدرديري — ٢- الوظيفة/ أستاذ طب الأطفال جامعة القاهرة
١- الاسم/ أ. د. سعيد بهادر — ٢- الوظيفة/ أستاذ علم النفس معهد الدراسات العليا للطفولة
١- الاسم/ أ. د. سلوى الحسيني — ٢- الوظيفة/ أستاذ الكيمياء الحيوية المركز القومي للبحوث

تاريخ البحـــث : ٢٥ / ٣ / ١٩٩٠

الدراســـات العليـــا
ختـــم الإجـــازة :
/ ١٩٩

أجيزت الرسالـــة بتـاريخ ٢٠/٥/٩٦

موافقة مجلس الكلية
١٩٩٦ / ٥ / ٢٢

موافقة مجلس الجامعة
/ / ١٩٩

جامعة عين شمس

معهد الدراسات العليا للطفولة

قسم الدراسات الطبيه

الظواهر الغذائيه والنفسيه للقصور النموى

رساله مقدمه من

الطبيب/ ناصر جمال فخرى

للحصول على درجة الدكتوراه فى دراسات الطفوله

تحت اشراف

أ.د. رمزى البارودى

استاذ طب الاطفال

جامعة القاهرة

أ.د. سعدية بهادر

استاذ علم نفس النمو

معهد الدراسات العليا للطفولة

أ.د. سلوى الحسينى

استاذ الكيمياء الحيويه

المركز القومى للبحوث

د. محمد الجندى

مدرس طب الأطفال

المركز القومى للبحوث

١٩٩٦