PSYCHOLOGICAL AND DEVELOPMENTAL SEQUALAE OF OESOPHAGEAL INJURIES DUE TO CORROSIVES IN CHILREN, COMPARATIVE STUDY IN URBAN AND RURAL AREAS

Thesis

Submitted for Fulfilment of

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By

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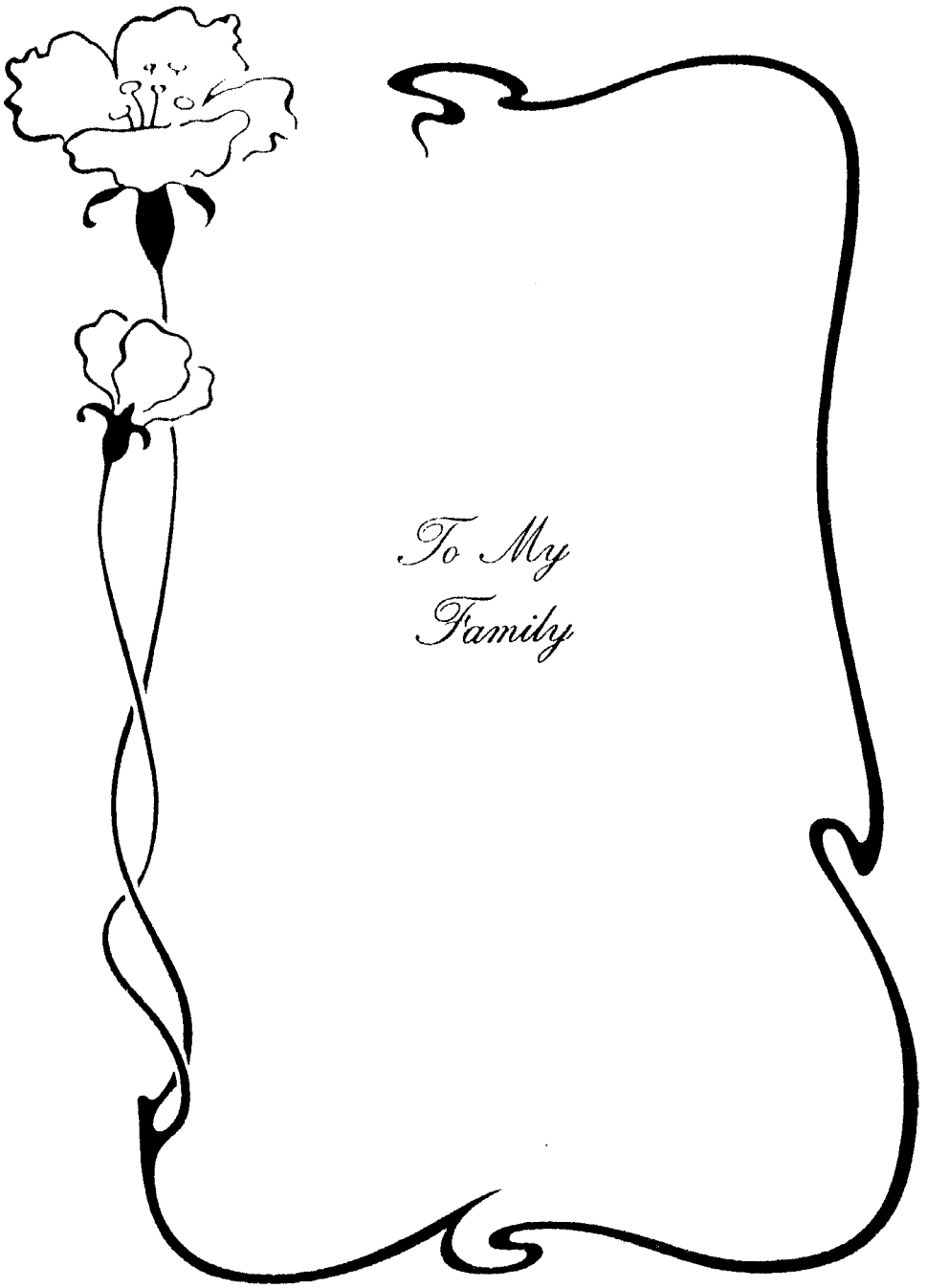

To My
Family

ACKNOWLEDGEMENT

Words can not express how proud I am with the great honour that this thesis has been supervised by one of the greatest professor in the Faculty of Medicine, Ein Shams University. Prof. Dr. Hamed Shatla professor of Paediatrics and Dean of the Faculty.

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Before and after all .... all thanks to God.

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ABSTRACT

Corrosive injuries of the esophagus result from the ingestion of solid or liquid caustic substances. The most frequent victims are children between 1 and 5 years of age, who all too often are tragically enticed by chemical solution that have been carelessly placed in familiar softy drink containers or by crystalline caustics that resemble sugar or sweets exposed in jar or can

Chemicals implicated in corrosive burns of esophagus includes alaline caustics, acids or acid like corrosives, and house hold bleaches, acid corrosives are present in automobile battery acids, toilet bowl cleaners and disinfectants. The alkaline caustics, washing soda, dish washing detergent house hold cleaners. Clinic test tablets or ingestion of small alkaline tablets.

The site and severity of injury depend on the type of caustic agent as well as its consistency, quantity and concentration. Caustic burn from common household bleaches such as clorox are encountered with increasing frequency but rarely produce permanent damage

The caustics that have bad taste or induce pain less affecting the upper GIT tract, and most of the child spit it immediately, however this defense mechanism is absent in the case of liquid caustics which are odourless, colourless and painless may produce serious damage , Acid caustics less affecting the also phagus because of the relative resistance of the epithelium to it and its rapid passage to the stomach. Caustic burns of the aesophagus are superficial manifested by hyperalmia, oedema, blisters and superficial ulceration, or deep burn extended through the full thickness of aesophageal wall and may create tracheoesophageal fistula

The early treatment of esophageal burn are resuscitation of the hemodynamic state, neutralization of the caustics, antibiotics. Corticosteroids and maintenance of the lumen by urgent endoscope an subsequent aesophageal dilatation

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INTRODUCTION

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Corrosive injuries of the oesophagus result from ingestion of solid or liquid caustic substances. The most frequent victims are children between 1 and 5 years of age who were all too often tragically enticed by chemical solutions that have been carelessly placed in familiar soft drink containers or by crystalline caustics that resemble sugar or sweets exposed in Jars or cans *(Krenzelok, EP and Clinton, 1979)*. The most chemicals implicated in corrosive burns of the oesophagus include alkaline caustic acid like corrosive and house hold bleaches. Acid corrosives are present in automobile battery acid toilet bowel cleaners and disinfectants, the alkaline caustics washing soda. "Potash" dish washing detergent. house hold cleaners *(Alford and Harris, 1979; Borja et al., 1969)*.

The site and severity of injury depend on the type of caustic agent as well as its consistency, quantity and concentration.

Caustic burn from common house hold bleaches such as clorox are encountered with increasing frequancy but rarely produce permanent damage *(Kirsh et al., 1978)*.

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oropharyngeal pain are less affecting the oesophagus, and most of the children spit it immediately however the defence mechanism is absent in the case of liquid caustics which are odourles and painles may produce serious damadge ***(Aschraft and Padula, 1974)***, acid caustics less affecting the oesophagus because of the relative resistance of the epithelium to it and its rapid passage to the stomach.

Caustic burns of the oesophagus are superfascial manifested by hyperaemia aedema, blisters and superfascial ulceration or deep burn extended through the full thickness of oesophageal wall and may create tracho-oesophgeal fistula or Mediastinitis ***(Holinger, 1978)***.

Post corrosive oesophageal stricture is a problem of considerable concern in Egypt. The disease occurs most commonly among the age of 4 years and the patients are usualy from large, low class families ***(Abdel wahab Y. Eokby et al., 1991)***. The early treatment of oesophageal burn are resuscitation, neutralization of the caustics antibiotics. corticosteroid and maintenance of the oesophageal lumen by urgent endoscope and subsequent dilatation, ***(Middel Kamp et el., 1969)***. The established oesophageal stricture which doesn't respond to the medical treatement will have a better improvement with surgical treatment ***(Gavriliu, 1975)***.

Oesophageal stricture with inability to have food and diminishing the resistance to diseases developed nutritional deficiency with retardation in growth and delay development in mentality with educational problems Also this will convert the normal child to a chronically ill patient and a handicapped person. Both have their psychological effects on the patients and their relatives ***(Haller, 1964).***

AIM OF THE WORK

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1. To study the incidence of corrosive burns in two different locality unban in Cairo and rural in Shibein El-Kom in relation to age, sex, family state standard of living, education level, and number of children in the family with their rank in the family.

2. To study the effect of early treatment by cortico steroid and endoscopic dilatations to minimizes the state of aesophageal stricture.

3. Anthropometeris study of the effect of stricture on the child growth, weight, height, mentality, and his educational level.

4. To study the morbidity and psychological disturbances of the children developed strictures with the development of a handicapped child.

5. To study the psychological disturbance of the patient's family.

REVIEW OF LITERATURE

Growth and Development

Our understanding of development has undergone major shifts in this century as we have assimilated new information. Freud emphasizes how early emotional experiences determined by the environment shape personality and psychopathology ***(Barnett, 1962).*** Piaget focuses on the predetermined unfolding of cognitive abilities over time in a specific progression that is innate to the child and relatively independent of the environment ***(Gesell and Amatruda, 1947).*** In past decades, however, the influence of the environment has assumed an all-powerful role as people have appreciated how greatly the trauma of dysfunctional families and socioeconomic status affect optimal developmental outcomes. We have now reached a stage of regarding the environmental influences in a context that also takes into account the genetic components of temperament and cognitive abilities in a neurobiologic format. we can now understand development through the interaction of these forces so that the relative importance of any single factor is not inflated unless significant trauma or pathologic circumstances exist.

The newborn

Normal newborns are endowed with a set of reflexes to facilitate survival, including rooting and sucking reflexes, and many sensory abilities. The newborn is no longer considered a blank slate a totally unformed being who gradually gains abilities according to environmental influences. Instead, the newborn is seen as having genetic strengths and weaknesses in neurocognitive organizations that are reflected in temperament, adaptability, responsiveness, and general interaction with the environment. These responses should in turn prompt reciprocal interactions from the parents, which further shape development. The Neonatal Behavioral Assessment scale by Brazelton was developed to measure many of the newborn's characteristics of temperament, including social behavior, orienting responses to stimuli, ability to deal with disturbing stimuli, state of arousal, and motor skills. When these abilities are described to the mother, this assessment can further sensitize her to the unique aspects of her child's behavior and responsiveness. This knowledge may in turn improve their interactions ***(Illingworth, 1960).***

The newborn has significant sensory abilities, which have been better identified in the last decade. Hearing is well developed at birth, and speech sounds are preferred.

The infant becomes alert and oriented to a females voice with high-pitched tones more readily than to a low-pitched male voice. The lower frequency tones of a male voice are more likely to soothe an infant. High-pitched crying is distressing not only to the newborn but also to adults ***(Reynolds and Asakawa, 1951).*** Infants can shut out loud or aversive stimuli and simply not respond. Within the first few weeks of life, they learn to recognize the mother's voice and differentiate it from other female voices ***(Talbot et al., 1959).***

Smell is well-developed at birth and plays a significant role in how infants orient themselves to the environment. Infants turn away from aversive smells and respond positively to pleasant ones. By 1 week of age, breast-fed infants recognize and discriminate the smell of their mother's breast pads. They recognize the smell of their mother, not the smell of milk alone. The infant has definite taste preferences at birth, preferring sweet tastes. Infants have more taste buds than adults do and avoid bitter or aversive tastes ***(Vaughan, 1966).***

At birth, the retina is well-developed, but the lens is rather immobile. Fixation and tracking through the visual field are well developed by 2 months of age Infants prefer to gaze at a human face rather than geometric designs, and they also prefer curved lines, bright colors,

and high contrast. The length of time that an infant on a paired visual stimulus has been interpreted as visual preference and has also been correlated with later cognitive development. Visual fixation tasks are the basis of the "infant IQ" tests marketed recently. Although visual acuity is poor at birth (approximately 20/400), it improves rapidly in the first 6 months of life to 20/40. Strabismus is common after birth but usually resolves by 3 months of age. If it persists, referral to an ophthalmologist is appropriate at 6 months of age ***(Weech, 1954).***

The first year

One of the most distressing features of infants is the amount of time they spend crying in the first few weeks of life. Crying gradually increases during the first 6 to 12 weeks of age because it is the main modality by which infants express responses to stimuli, both aversive and nonaversive. Crying can be a response to a variety of stimuli, including hunger, a wet diaper, fear, fatigue, and overstimulation. Crying gradually decreases after 12 weeks of ages, as the infant develops other responses, such as smiling or reaching, or becomes more adapt at self-soothing, such as by sucking the fingers or thumb. In the first weeks of life, however, crying can become a distressing problem for the parents, and crying associated with irritability is often labeled as colic. particularly

sensitive infants with a low tolerance for stimuli can be irritable and difficult to deal with at home. It is useful to help parents understand their infant's temperament and to teach them techniques for avoiding excessive stimuli (because parents often respond to excessive crying by creating excessive stimuli). Parents should be taught ways to calm the child, such as offering nonnutritive sucking, rocking the child, singing to the child, and walking while holding the child. Perhaps most important for the parents is an understanding of the developmental aspects of crying and the emergence of improved coping skills in the baby after 12 weeks of age ***(Erikson, 1950).***

Piaget describes the first 2 years of life as the sensorimotor period, during which infants learn with increasing sophistication how to link the sensory input from the environment with a motor responses. Infants build on primitive reflex patterns of behavior (termed schema; Sucking is an example) and constantly incorporate or assimilate new experiences to further elaborate their schema. The schema evolve over time as infants accommodate new experiences and as new levels of cognitive ability unfold in a rather orderly sequence. In the first year of life, infants' perception of reality revolves around themselves and what they can see or touch. They follow the trajectory of an object through the

fields of vision, but prior to 6 months, the object does not exist for them once its leaves the field of vision. At 9 to 12 months, infants gradually develop the concept of object permanence, or the understanding that objects exist even when they are not seen. They first apply the concept of object permanence to the image of the mother because of her emotional importance; this realization is a critical part of attachment behavior, discussed below. In the second year, children extend their ability to manipulate objects by using instruments, first by imitation and later by trial and error (***Greulich and Pyle, 1959)*** (Table 1).

Freud describes the firsts year of life as the oral stage because so many of the infant's needs are fulfilled by oral means. Nutrition is obtained through sucking on the breast or bottle, and self-soothing also occurs through sucking on fingers or a pacifier. As Mahler emphasizes, this is a stage of symbiosis with the mother, during which the boundaries between mother and infant are blurred. The baby's needs are totally met by the mother, and the mother has been described as manifesting "narcissistic possessiveness" of the infant. This is a very positive and critical interaction in the bidirectional attachment process known as bonding. The parents learn to be aware of and read their infant's cues, which reflect needs. However, a more sensitive emotional interaction process develops,

Table (1) : Developmental chartes

1 - 2 months

Activities to be observed :
Holds head erect and lifts head.
Turns from side to back.
Regards faces and follows objects through visual field.
Drops toys.
Becomes alert in response to voice.

Activities related by parent :
Recognizes parents.
Engages in vocalizations.
Smiles spontaneously.

3 - 5 months

Activities to be observed :
Grasps cube-first ulnar then later thumb opposition.
Reaches for and brings objects to mouth.
Makes "raspberry" sound.
Sits with support.

Activities related by parent :
Laughs.
Anticpates food on sight.
Turns from back to side.

6 - 8 months

Activities to be observed :
Sits alone for short period.
Reaches with one hand.
First scoops up a pellet then grasps it using thumb opposition.
Imitates "bye-bye."
Passes object from hand to hand in midline.
Babbles.

Activities related by parent :
Rolls from back to stomach.
Is inhibited by the word no.

9 - 11 months

Activities to be observed :
Stands alone.
Imitates pat-a-cake and peek-a-boo.
Uses thumb and index finger to pick up pellet.

Activities related by parent :
Walks by supporting self on furniture.
Follows one-step verbal commands, eg, "Come here," "Give it to me."

1 year

Activities to be observed :
Walks independently.
Says "mama" and "dada" with meaning.
Can use neat pincer grasp to pick up a pellet.
Releases cube into cup after demonstration.
Gives toys on request.
Tries to build tower of 2 cubes.

Activities related by parent :
Points to desired objects.
Says one or two other words .

18 months

Activities to be observed :
Builds tower of 3 to 4 cubes .
Throws ball.
Scribbles spontaneously.
Seats self in chair.
Dumpd pellet from bottle.

Activities related by parent :
Walks up and down stairs with help.
Syas 4 to 20 words.
Understands a 2-step command.
Carries and hugs doll.
Feeds self.

Table (1) : (cont'd) Developmental chartes

24 months

Activities to be observed :
Speaks short phrases, 2 words or more.
Kicks ball on request.
Builds tower of 6 to 7 cubes.
Points to named objects or pictures .
Jumps off floor with both feet.
Stands on either foot alone.
Uses pronounces.

Activities related by parent :
Verbalizes toilet needs .
Pulls on simple garment .
Turns pages of book singly.
Plays with domestic mimicry.

30 months

Activities to be observed :
Walks backward.
Begins to hop on one foot..
Uses prepositions.

Copies a crude circle.
Points to objects descibed by use.
Refers to self as I.
Holds crayon in fist.

Activities related by parents :
Helps put things away.
Carries on a conversation.

3 years

Activities to be observed :
Holds crayon with fingers.
Builds tower of 9 to 10 cubes.
Imitates 3 - cube bridge.
Copies circle.
Gives first and last name.

Activities related by parent :
Rides tricycle using pedals.
Dresses with supervision.

3 - 4 years

Activities to be observed :
Climbs stairs with alternating feet.
Begins to button and unbutton.
"What do you like to do that's fun?" (Answers using plurals, Personal pronoun, and verbs.)
Responds to command to place toy in, on or under table.
Draws a circle when asked to draw a man (girl, boy).
Knows own sex. (Are you a boy or a girl?")
Gives full name.
Copies a circle already drawn. ("Can you make onelike this?")

Activities related by parent :
Feeds self at mealtime.
Takes off shoes and jacket .

4 - 5 years

Activities to be observed :
Runs and turns without losing balance.
May stand on one leg for at least 10 seconds.
Buttons clothes and laces shoes. (Does not tie.)
Counts to 4 by rote.
"Give me 2 sticks." (Able to do so from pile of 4 tongue depressors.)
Draws aman. (Head, 2 appendages, and possibly 2 eyes. No torso yet.)
Knows the days of the week. ("What day comes after Tuesday?")
Gives appropriate answers to : " What must you do if you are sleepy? Hungry? Cold?'
Copies - in imitation.

Activities related by parent :
Self care at toilet. (May need help with wiping.)

Table (1) : (cont'd) Developmental chartes

Plays outside for at least 30 minutes.
Dresses self except for tying.

5 - 6 years

Activities to be observed :
Can catch ball.
Skips smoothly.
Copies a + already drawn.
Tells age.
Concept of 10 (eg, counts 10 tongue depressors).
May recite to higher number by rate.
knows right and left hand .
Draws recognizable man with at least 8 details.
Can describe favorite television program in some detail.

Activities related by parent :
Does simple chores at home. (Taking out garbage, drying silverware, etc.)
Goes to school unttended or meets school bus.
Good motor ability but little awareness of dangers.

6 - 7 years

Activities to be observed :
Copies a Δ .
Defines words by use. ("What is an orange?" "To eat..")
Knows if morning or afternoon.
Draws a man with 12 details. Reads several one - syllable printed words. (My, dog see, boy.).
Uses pencil for printing name.

7 - 8 years

Activities to be observed :
Counts by 2s and 5s.
Ties shoes .
Copies a ◊.
Knows what day of the week it is. (Not date or year.)
Reads paragraph # 1 Durrell :

Reading :
Muff is a little yollow kitten. She drinks milk. She sleeps on a chair. She does not like to get wet.

Corresponding arithmetic :

7	6	6	8
+ 4	+ 7	- 4	-3

No evidence of sound substitution in speech (eg, fr for thr). Adds and subtracts one - digit numbers.
Draws a man with 16 details.

8 - 9 years

Activities to be observed :
Defines words better than by use. (What is an orange?" "A fruit.")
Can give an appropriate answer to the following :
"Waht is the thing for you to do if . . .
- you've broken something that belongs to someone else?"
- a playmate hits you without meaning to do so?"
Reads paragraph #2 Durrell :

Reading :
Alittle black dog ran away from Home. He played with two big dogs. They ran away from him. It began to rain. He went under a tree. wanted to go home, but he did not know the way. He saw a boy he knew. The boy took him home.

Corresponding arithmetic :

	45		
67	16	14	84
+ 4	- 27	- 8	- 36

Table (1) : (cont'd) Developmental chartes

Is learning borrowing and carrying processes in addition and subtraction

9 - 10 years

Activities to be observed :

Knows the month, day, and year.
Names the months in order. (Fifteen seconds, one error) Makes a sentence with these 3 words in it (one of 2. Can use word orally in proper context)
1. Work . . . money . . . men
2. boy . . . river . . . ball
Reads paragraph # 3 Durrell :

Reading :

Six boys put up a tent by the side of a river. They took things to eat with them. when the sun went down, they went into the tent to sleep. In the night, a cow came and began to eat grass around the tent. The boys were afraid. They thought it was abear.

Corresponding arithmetic :

5204	23	837
- 530	x3	x7

Should comprehend and answer question : "What was the cow doing?"
Learning simple multiplication

10 - 12 years

Activities to be observed :

Should read comprehend paragraph # 5 Durrell :
Reading :
In 1807, Robert Fulton took the first long trip in a steamboat. He went one hundred and fifty miles up the Hudson River. The boat went five miles an hour. This was faster than a steamboat had ever gone before. Crowds gathered on both banks of the river to see this new kind of boat. They were afraid that its noise and splashing would drive away all the fish.
Corresponding arithmetic :
420
x 29 9) 72 31) 62
Answer : "What river was the trip made on?"
Ask to write the sentence : "The fishermen did not like the boat."
Should do multiplication and simple diveision

12 - 15 years

Activities to be observed :

Reads Paragraph # 7 Durrell :

Table (1) : (cont'd) Developmental chartes

Reading :

Golf originated in Holland as a game playe on ice. The game in its present form first apeared in 'Scotland. It became unusually popular and kings found it so enjoyable that it was known as "the royal game " James IV, however, thought that people neglected their work to indulge in this fascinating sport so that it was forbidden in 1457. James relented when he found how attractive the game was, and it immediately regained its former popularity. Golf spread gradually to other countries, being introduced in America in 1890. It has grown in =favor until there is hardly a town that does not boast of a private or puplic course.

Corresponding arithmetic :

536 4762

Reduce fractions to lowest forms.
Ask to write sentence: "Goff originated in Holland as a game pjlayed on ice."
Answers questions:
"why was golf forbidden by James IV?"
"Why did he change his mind?"
Does long division, adds and subtracts fractions.

which can be seen in the mirroring of facial expressions by mother and infant and in their mutual engagement in cycles of attention and inattention, which further develops into social play. A mother who is depressed or cannot respond to the baby's expressions and cues has a profound effect on the infant's future development and attachment. Erickson's terms of basic trust versus mistrust are another way of describing the reciprocal interaction that characterizes this stage ***(Lewis et al., (1943).***

Turn-taking games, which occur between 3 and 6 months of age, are a pleasure for both the parents and the infant and are an extensions of mirroring behavior. They also represent an early form of imitative behavior, which is important in later social and cognitive development. More sophisticated games such as peek-a-boo, occur at approximately 9 months. The infant's thrill at the reappearance of the face that vanished momentarily demonstrates the emerging understanding of object permanence ***(Meiks and Green, 1960).***

Eight to nine months is also a critical time in the attachment process because separation anxiety and stranger anxiety become marked. Kagan describes the infant at this stage as able to appreciate discrepant events that match previously known schema only partially ***(Pyle et al., 1959).*** These new events cause uncertainty and

subsequently fear and anxiety. Cognitively, the infant must be able to retrieve the memory of previous schema and integrate the information to previous knowledge over an extended time. These abilities are developed by 8 months of age and lead to the fears that subsequently develop : stranger anxiety and separation anxiety. In stranger anxiety, the infant analyzes the face of a stranger, detects the mismatch with previous schema, and may subsequently respond with fear or anxiety, leading to crying. In separation anxiety, the child perceives the difference between the mother's presence and absence by remembering the schema of her presence. Perceiving the inconsistency, the child becomes uncertain and subsequently anxious and fearful. This begins at 8 months, reaches a peak at 15 months, and disappears by the end of 2 years in a relatively orderly progression because central nervous system maturation facilitates the development of new skills. A parent can put the child's understanding of object permanence to good use by placing a picture of the mother near the child or by leaving an object where the child can see it during her absence. A visual substitute for her actual presence may comfort the child ***(Pyle et al., 1959)***.

Growth in the first 3 years

Fetal growth in length is most rapid at 4 to 6 months'

gestation. However, adipose tissue begins to develop at 7 months, and weight gain accelerates, causing fetal weight to double during the last 2 months in utero. The rate of growth of males in late fetal development and during the first 6 months postnatally is more rapid than the growth rate of females because of a higher level of testosterone. The birth weight of the newborn correlates with the size nutritional state, and general health of the mother and represents the influence of uterine constraints on ultimate size. Newborns may lose up to 10% of their birth weight in the first few days of life, but with normal nutrition birth weight is regained in approximately 10 days. The infant subsequently gains approximately 30 g per day for the first several months ***(Tanner, 1956).***

After the first 6 months of life, genetic factors influencing ultimate height begin to exert their effect. The growth percentile, therefore, may shift significantly in the first 4 to 18 months of life. This shift can be either up or down. An infant who is small for gestational age and has a genetic predisposition to larger stature usually experiences accelerated growth in the first 6 months and by 18 months a relatively stable new growth percentile is established. The downward shift is seen in large infants who have a genetic predisposition to short stature. A fall off in their growth percentiles may often be misconstrued

as failure to thrive, although a stable growth percentile should be achieved by 18 months of age.

By 1 year of age, infants weigh 3 times as much as they did at birth and are 1 1/2 times as long. By 2 year of age, the growth velocity curve has stabilized into the rate for mid childhood, which is a weight gain of 2 to 3 kg/y and a height gain of 5 to 7.5 cm/y. At the second birthday, a child attains approximately one-half of adult height ***(Watson, 1959)***.

The energy requirement during growth also changes dramatically in the first few years of life. Approximately 110 kca/kg/d is necessary in early infancy because up to 40% of this total energy requirement is used for growth. The percentage used for growth gradually decreases to 3% at 2 years of age and remains at this level even during adolescence. After 2 years of age, the overall energy requirement gradually decreases from 90 kcal/kg/d to 60 kcal/kg/d during middle childhood, and the majority of the energy expenditure is accounted for by activity and basal metabolic rate of the tissues. The gradual decrease is secondary to a decline in the relative mass of organs, such as the brain and liver, that have a high requirement for energy compared to resting muscle. The relative energy expended during activity increases in adolescence, particularly for males. The percentage of body weight

that is muscle increases from 22% at 3 months to 35% at 5 years and 40% at maturity in males. In contrast, organ weight is 17% of body weight in the infant, with 75% of organ weight accounted for by the brain. By maturity, only 5.1% of body weight is organ wight. Fat increases during the first year of life from 12% of body weight at birth. After the infant begins to walk and explore, however, the proportion of fat decreases and remains stable throughput childhood. In adolescence, the proportion of body weight that is fat increases with sexual maturation in girls but not in boys ***(Falkner, 1966)*** (Table 1).

Brain Growth

Approximately 100 billion neurons are present in the fully developed brain, and replication of neurons is completed before birth. Most of this growth occurs in the first 3 months of gestation. Cell density subsequently decreases rapidly until birth. After birth, the decrease is slower, and ceases at 15 months. At birth, the head is three-fourths of its adult size and makes up one-fourth of the baby's length. This ratio changes dramatically in time so that by 25 years of age, the head measures one-eighth of the body length (Fig. 1). Postnatally, the brain continues to grow rapidly, completing half of its lifetime growth by the end of the first year. The postnatal growth

is due to an increase in white matter and a proliferation of synaptic connections. After 2 years of age, the head circumference increases only 2 cm/y during middle childhood. By 7 years of age, nine-tenths of brain growth is completed, and many 10-year-olds have the brain weight of an adult ***(Todd, 1937).***

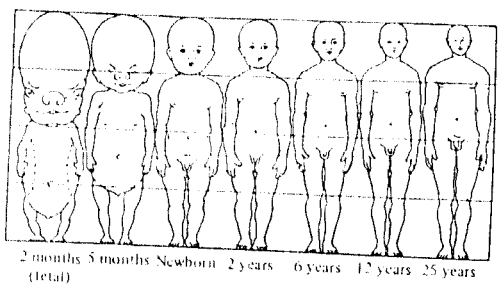

Fig. (1) : Relative proportions of head, trunk, and extremities at different ages.

The cerebellum is the area of the gray matter that develops last. It begins its growth at 30 weeks of gestation and ends at approximately 1 year of age. It is, therefore, particularly vulnerable to trauma, which may occur in late gestation or at birth. The spinal cord extends through the length of the neural canal until the third month of gestation. After this time, the torso of the fetus grows faster than the spinal cord, which is fixed in position superiorly by the brain. The lower end of the spinal cord subsequently rests at a gradually higher vertebral level through later fetal life and, by birth, is located at the third

lumbar vertebra. The spinal cord doubles its weight in the first year of the life and has increased eightfold by adulthood ***(Vogt and Vickers, 1938).***

Myelinization begins in the spinal cord by the fourth month of gestation and begins in the brain during the last trimester. At birth, the autonomic system is matured an myelinated. The cranial nerves, except for the optic and olfactory nerves, are also myelinated. The cortex and most of its connection to the thalamus and basal ganglia are incompetely myelinated. It takes at least 2 years for myelinization of these areas and the spinal cord to the complete ***(Watson and Lowrey, 1967).***

Newborn Reflexes

Reflex movement begins in fetal development as early as 9 weeks' gestation. However, most of the reflexes associated with the newborn develop between 20 and 38 weeks' gestation. Sucking, a basic reflex critical to survival, can first be seen in utero as early as 14 weeks' gestation. The rooting reflex begins by 28 weeks' gestation and is evidenced by the infant's pursing the lips and sucking after turning toward a touch to the cheek. The tonic neck reflex is elicited by forcibly turning the infant's head. In response, the infant extends the arm and leg on the side toward which the head is turned and flexes the opposite side (Fencing position stance). This reflex

disappears by 8 months of age unless myelinization or brain development is pathologic ***(Frankenburg and Dods, 1967).*** The Moro embrace reflex (an embracing movement as a startle response), palmar grasp, and trunk incurving in response to a tactile stimulus to the side of the trunk all develop by 28 weeks' gestation but disappear by 3,4, and 5 months of age, respectively. Babinski's reflex, which develops just prior to birth in a full-term infant, does not normally disappear until 12 to 16 months of age, when adequate myelinization has occurred ***(Stuart, 1946).***

EEG & Sleep

The brain also undergoes rapid maturation in the first 2 years of life. Prior to 26 weeks' gestation, the EEG is disorganized and without periodicity. By 8 months' gestation, however, low amplitude fast waves occur at 16 to 18 cycles per second. At birth, the waking and sleeping cycles can be differentiated, and by 4 months, sleep spindles appear. During this period, the proportion of total sleep time occupied by active or rapid eye movement (REM) sleep decreases from 50 to 20%. The infant's sleep pattern at the onset of sleep also shifts from REM sleep to quiet sleep. The amount of quiet sleep also gradually increases to a maximum of 70 to 80% of total sleep time. These changes are a reflection of significant brain maturation, which has occurred by 4 months.

Infantile reflexes, as previously mentioned, are disappearing, and the infant is becoming more alert and interactive with the environment. The infant is now reaching for and grasping objects, smiling and laughing out loud, anticipating food on sight, and sitting with support ***(Iliff and Lee, 1952).***

Motor Dexterity

The developmental progression of the grasp through the first year illustrates the gradual improvement in motor dexterity. The grasp begins as a raking motion involving mainly the ulnar aspect of the hand at 3 to 4 months. The thumb is used in the grasp just before 5 months, as the focus shifts to the radial side of the hand. The thumb opposes the finger for picking up a cube just before 7 months, but the neat pincer grasp used for smaller objects, such as a pellet, does not develop until approximately 9 months ***(Illingworth, 1960).***

The changes in gross motor skills have a significant impact on the child's exploration of the environment. Sitting alone occurs at 6 months of age, but the onset of walking at 12 months (with a range of 9 - 17 months in the normal child) introduces the major theme of the second year of life, autonomy ***(Illingworth, 1960).***

The second year

Once children can walk independently, they can move away from the mother and explore the environment on their own. Although they use the mother as a home base and return to her frequently to reassure themselves that she is still there and available for them, they have definitely taken a quantum jump in independence and autonomy. These new themes are closely tied to the child's beginning sense of mastery over the environment and an emerging sense of self, These issues lead to the "terrible twos" and the frequent verbalizations "no" as children struggle to develop a better idea of what is under their control. This is a fragile time of ego development. Parents should not crush emerging autonomy but develop appropriate limits that foster independent exploration ***(Brazelton, 1984).***

As children develop a sense of self, they begin to understand the feelings of others and develop empathy. They hug another who is in distress or become concerned when another is hurt. They begin to understand how another child feels when he or she hit or hurt, and this realization helps them to inhibit their own aggressive behavior. They also begin to understand right and wrong and parental expectations. They realize when they have done something "bad" and may signify that awareness with "uhoh!" or distress. They also take pleasure in their

accomplishments and become more aware of their bodies.

The development of these cognitive, emotional, and physical abilities is related to significant brain maturation, which occurs by 2 years of age. Myelinization is reaching its completion and, according to Rabinowicz, all the layers of the cortex reach a similar state of maturation between 15 and 24 months. Before this time, differences exist in the maturation level between cortical layers. These changes set the stage for toilet training after 18 months of age Toddlers have developed the sensory abilities to be aware of a full rectum or bladder and are physically able to control their rectal sphincter. They also take great pleasure in their accomplishments, particularly in appropriate elimination, if it is positively reinforced. children must be given some control over when elimination occurs. If severe restrictions are imposed, the accomplishment of this developmental milestone can become a battle between parent and child, and long-term struggles of control predisposing to encopresis may develop later. Freud terms this period the anal stage because the developmental issue of bowel control is the major task requiring mastery. It basically represents a more generalized theme of socialized behavior and overall body cleanliness, which is begun to be taught or imposed on the child at this age. The child is

encouraged to control impulsive and aggressive behavior by acting in socially appropriate ways. Although Freud describes that by products of anal regularity on personality development, including punctuality, reliability, cleanliness, and conscientiousness, these themes simply represent abilities emerging at the time that toilet is also being mastered *(Brazelton, 1984).*

Language development

1 - 4 year

Communication is important from birth, particularly the nonverbal reciprocal interactions between the infant and caretaker, which have already been described. By 2 months of age, these interactions begin to include vocalizations that involve cooing and reciprocal vocal play between the mother and child. Babbling begins by 6 to 10 months of age, and the repetition of sounds. such as "da-da-da-da," is facilitated by increasing oral muscular control. Babbling reaches a peak at 12 months. The child then moves into a stage of having needs met by using individual words to represent objects or actions. It is common for children of this age to express wants and needs by pointing to objects. There is significant variability in the number of words acquired by 18 months, with an average of 20 to 50 words. The failure of parents or siblings to encourage vocalization and their overuse of

nonverbal communication, such as pointing, slows the development of expressive vocabulary. Recurrent otitis media, which causes a fluctuating conductive hearing loss, may also have a significant impact on the achievement of early language milestones table (2).

Receptive language usually develops more rapidly than expressive language. Word comprehension begins at 9 months; by 13 months, the receptive vocabulary may be as high as 20 to 100 words. After 18 months, there is a dramatic increase in expressive and receptive vocabulary, and by the end of the second year, a quantum leap occurs in language development. This leap represents a major change in cognitive development. The child begins to put words and phrases together and begins to use language to represent a new world, the symbolic world Although the infant begins to use single words to represent objects or people in the latter part of the first year, it is not until the end of the second year that language ability begins to blossom. Children now begin to put verbs into their phrases and focus much of their language on describing their new abilities, often while they are doing them, e.g, "I go out." They incorporate prepositions into speech and ask why and what questions more frequently. They also begin to appreciate time factors and to understand and use this concept in their speech.

Table (2) : Normal speech and language development

Age	Speech	Language	Artlculation
1 month	Throaty sounds		Vowels: /ah/, /uh/, /ee/
2 months	Vowel sounds ("eh"), coos		
2.5 months	Squeals		
3 months	Babbles, initial owels		Consonants: m, p, b
4 months	Guttural sounds ("ah," "go"		Vowels: /o/, /u/
5 months			
7 months	Imitates speech sounds		Syllables: da, ba, ka
8 months			
10 months		"Dada" or "mama" nonspecifically	Approximates names: baba/bottle
12 months	Jargon begins (own language)	One word other than "mama" or "dada"	Understandable: 2-3 words
13 months		Three words	
16 months		Six words	Consonants; t, d, w, n, h
18-24 months		Two-wor phrases	Understandable 2-word phrases
24-30 months		Three-word phrases	Understandable 3-word phrases
2 years	Vowels uttored corroctly	Approximately 270 words; uses pro-nouns	Approximately 270 words; uses phrases
3 years	Some degree of hesitancy and uncertainty common	Approximately 900 words; intelligible 4-word phrases	Approximately 900 words; intelligible 4-word phrases
4 years		Approximately 1540 words; intelligible 5-word phrases or sentences	Approximately 1540 words; intelligible 5-word phrases
6 years		Approximately 2560 words; intelligible 6- or 7-word sentences	Approximately 2560 words; intelligible 6- or 7-word sentences
7-8 years	Adult proficiency		

Piaget describes the 2-to 6-year-old stage as preoperational. This stage begins When language has facilitated the creation of mental images in the symbolic sense. The child begins to learn to manipulate the symbolic world. The child sorts out reality from fantasy imperfectly and may be terrified of dreams, wishes, and foolish threats. Most of the child's perception of the world is egocentric or interpreted in reference to wants, needs, or influence. Cause-and- effect relationships are confused with temporal relationships or interpreted egocentrically. For instance, children often focus their understanding of divorce on themselves; eg, "my father left because I was bad" or "my father left because he didn't love me." illness and the need for medical care are also commonly misinterpreted at this age. The child may make a mental connection between a sibling's illness and a recent argument, a negative comment, or a wish that the sibling be ill. The child may experience significant guilt unless the parents are aware of these misperceptions and take time to sort them out ***(Haith, 1983).***

At this age, children also endow inanimate objects with human feelings. They also assume that humans cause or create all natural events. For instance, when asked why the sun sets, they may respond that "the sun goes to his house" or "it is pushed down by someone else."

Magical thinking blossoms during the ages of 3 to 5 as symbolic thinking incorporates more elaborate fantasy. Fantasy facilitates development of role playing, sexual, identity, and emotional growth. Children test new experiences in fantasy, both in their imagination and in play. In their play, children often create magical stories and novel situations that reflect issues they are dealing with, such as aggression, relationships, fears, and control issues. Children often invent imaginary friends at this time, and nightmares or fears of monsters are common. At this stage, other children become important in facilitating play, such as in a preschool group. Play gradually becomes more cooperative; shared fantasy leads to game playing. Freud describes the Oedipal phase between the ages of 3 and 6, when there is strong attachment to the parent of the opposite sex. The child's fantasies may focus on play acting the adult role with that parent, although by 6 years of age Oedipal issues are usually resolved, and attachment is redirected to the parent of the same sex ***(Kagan, 1982).***

The early school years : 5 - 7

Attendance in kindergarten at 5 years marks an acceleration in the separation/individuation theme initiated in the preschool years. The child is ready to relate to peers in a more interactive manner than

demonstrated by previous parallel play. The brain has reached 90% of its adult weight. At approximately 6 years, a remodeling of the cortex occurs. The Betz cells decrease in length and increase in width. The cortex, in general, shows a decrease in total thickness, with an increase in the number of nerve cells in the different layers. Sensor-moter coordination abilities are maturing and facilitating pencil-and-paper tasks sports, both part of the school experience.

Cognitive abilities are still at the preoperational stage, and children focus on one variable in a problem at a time. However, by 5 1/2 years, most children have mastered conservation of length; by 61/2 years, conservation of mass and weight; and by 8 years, conservation of volume.

By first grade, there is more pressure on the child to master academic tasks, including the recognition of numbers, letters, and works, and the ability to write. Piaget describes the stage of concrete operations beginning after age 6, when the child is able to perform mental operations concerning concrete objects that involve the manipulation of more than one variable. The child is able to order, number, and classify because these activities are related to concrete objects in the environment and because these activities are stressed in

early schooling. Magical thinking diminishes greatly at this time, and the reality of cause-and-effect relationships is better understood. Fantasy and imagination are still strong and are reflected in the themes of play. lists specific developmental abilities through middle childhood and adolescence ***(Yogman et al., 1988).***

Middle childhood years : 7 - 11

Freud terms these the latency years, during which children are not bothered by significant aggressive or sexual drives but instead devote most of their energies to school and peer group interactions. In reality, throughout this period there is a gradual increase in sexual drives, which is manifested by increasingly aggressive play and interactions with the opposite sex. Fantasy still has an active role in dealing with sexuality before adolescence, ad fantasies often focus on movie stars and rock heroes. Organized sports, clubs, and other activities are other modalities that permit preadolescent children to display socially acceptable forms of aggression and sexual interest ***(Karlberg et al., 1987).***

For the 7-year-old, the major developmental task focuses on achievement in school and acceptance by peers. Academic expectations have intensified and require the child to concentrate, attend, and process increasingly complex auditory and visual information.

Children with significant learning disabilities or problems of attention may have difficulty in these tasks and subsequently may receive significant negative reinforcement from teachers and even parents. Such children may develop a poor self-image, which may be manifested as behavioral difficulties. The pediatrician must evaluate potential learning disabilities in any child who is not developing adequately at this stage or who presents with emotional or behavioral problems. Their abilities are not as easily documented as milestones in early development. In the school-age child, the quality of the response, the attentional abilities, and the child's emotional approach to the task can make a dramatic difference in how successful the child is at school. The clinician must consider all of these aspects to appropriately diagnose learning disabilities ***(Falkner and Tanner (1982).***

Pubertal growth

The pubertal growth spurt occurs at approximately 10 years in females and 12.5 years in males. The speed of growth increases, reaching a peak of approximately 9 cm/y in females and 10.3 cm/y in males. Different areas of the skeleton attain their peak growth at different times. This is seen most dramatically in the feet, which first experience a growth spurt. This is followed by a rapid

increase in leg length and subsequently by trunk growth. Facial growth occurs after peak height velocity. The mandible changes most remarkably, demonstrating a 25% increase in height between 12 and 20 years of age, compared to only a 6 - 7 % increase in the size of the cranial base.

Boys have just over 2 more years of preadolescent growth than girls do; during this time leg growth increases more dramatically than trunk growth. Girls have a greater spurt in hip width, related to stature, than boys do, although boys exceed girls in most other areas of bone growth ***(Erikson, 1963).***

Psychological Development

Developmental stages

Anyone who provides for the medical or emotional needs of children understands that children change dramatically throughout the process of growing up. Each period of development presents children and parents its own characteristic set of challenges and problems. Likewise, the manifestations of stress and of disease change as the child changes over time. The planning of interventions by health care providers must therefore be in tune with the developmental level of the child and the corresponding needs of the parents. For these reasons, the chapter begins with a review of some of the highlights of child development that relate particularly to the psychosocial care of children ***(Yogman et al., 1988).*** The struggles that caretakers face in providing a nurturing environment for their children are also noted (Tables 3 and 4).

The Infant

The central developmental challenge of infancy is the development of specific emotional attachments, or bonds, between infant and caretakers. Those attachment bonds, in

Table (3) : Emerging Patterns of Behavior from 1 to 5 Years of Age

	15 Months
Motor :	Walks alone; crawls up stairs
Adaptive:	Makes tower of 2 cubes; makes a line with crayon; inserts pellet in bottle
Language:	Jargon; follows simple commands; may name a familiar object (ball)
Social:	Indicates some esires or needs by pointing
	18 Months
Motor :	Runs stiffly; sits on small chair; walks up with one hand held; explores drawers and waste baskets
Adaptive:	Piles 3 cubes; imitates scribbling; imitates vertical stroke; dumps pellet from bottle
Language:	10 words (average); names pictures
Social:	Feeds self; seeks help when in trouble; may complain when wet or soiled
	24 Months
Motor :	Runs well; walks up and down stairs, one step at a time; opens doors; climbs on furniture
Adaptive:	Tower of 6 cubes; circular scribbling; imitates horizontal stroke; folds paper once imitatively
Language:	Puts 3 words together (pronoun, verb, object)
Social:	Handles spoon well; often tells immediate experiences; helps to undress; listens to stories with pictures
	30 Months
Motor :	Jumps
Adaptive:	Tower of 8 cubes; makes vertical and horizontal strokes, but generally will not join them to make a cross; immitates circular stroke, forming closed figure
Language:	Refers to self by pronoun "I", knows full name
Social:	Helps put things away
	36 Months
Motor :	Goes up stairs alternating feet; rides tricycle; stands momentarily on one foot
Adaptive:	Tower of 9 cubes; imitates construction of "bridge" of 3 cubes; copies a circle imitates a cross
Language:	Knows age and sex; counts 3 objects correctly; repeats 3 numbers or a sentence of 6 syllables
Social:	Plays simple games (in "parallel" with other children); helps in dressing (cubuttons clothing and puts on shoes); washes hands
	48 Months
Motor :	Hops on one foot; throws ball overhan; uses scissors to cut out pictures; climbs well
Adaptive:	Copies bridge from model; imitates construction of "gate" of 5 cubes; copies cross and square; draws a man with 2 to 4 parts besides head; names longer of 2 lines
Language:	Counts 4 pennies accurately; tells a story
Social:	Plays with several children with beginning of social interaction and role-playing; goes to toilet alone
	60 Months
Motor :	Skips
Adaptive:	Draws triangle from copy; names heavier of 2 weights
Language:	Names 4 colors; repeats sentence of 10 syllables; counts 10 pennies correctly
Social:	Dresses and undresses; asks questions about meaning of words; domestic role-playing

After 5 years the Stanford-Binet, Wechsler-Bellevue and other scales offer the most precise estimates of evelopmental level. In order to have their greatest value, they shoul be administered only by an experienced and qualified person.

Table (4) : Highlights of child and family development.

Stage (Age)	Developmental Tasks	Developmental Landmarks	Developmental Concerns	Pitfalls for Caregivers
Intancy (birth - 1 y)	Emotional attachment	1-3 mo: responsive social smile; 7-9 mo: distress in the presence of "strangers.:	Temperamental variations (including colic).	Exhaustion; lack of emotional support; unexpected temperament; unfulfille expectations and fantasies.
Toddler (1 - 3 y)	Beginning of the separation-indicidu ation process	8-14 mo: separation anxiety; 12 mo: locomotion; 15 mo: "no"; 24-36 mos: bowel training.	Sleep disturbances (separation or overstimulation); breath holding and temper tantrums; "terrible two's, ie, oppositional behavior; accident proneness.	Exhaustion; autonomous strivings of child seen as rejection or adversarial relationship; need for parents to set limits seen as parental failure; "permeissivenes" seen as good parenf-child interaction.
Preschooler 3 - 6 y)	Taming of the internal world of fantasy	Symbolic play.	Childhood fears: bed-time, darkness, ghosts and onsters.	Competitive strivings of child seen as personal challenge; egocentricity of child seen as selfishness.
School age (6 - 11 y)	Skills development	7 Y: logical thought processes (cause-and-effect thinking); games and organizations with rules.	Continued childhood fears; school phobia; learning isabilities; primary nocturnal enuresis.	Parental iscomfort with separation from child; high expectations for child's performance; child's performance experienced as parents own selt-esteem.
Adolescence (11 - 20 y)	Continuation of the separation-individuation process: identity formation ("Who am !?"); gaining independence.			
Early adolescence (11 - 15 y)		Puberty; "best friends" of same sex; self-absorption; painful concerns about appearance.	Adolescent "turmoil."	Child's attachment to peer group seen as rejection; self-absorption seen as irresponsible disregard for others; exasperation with the child's changing moods.

turn, form the basis for all meaningful and rewarding human relationships throughout the life span.

This period of forming emotional attachments is now recognized as a time of great reciprocal interaction between the child and the caretakers, with the child being an active participant in the attachment process. Shortly after birth, the child begins to display a powerful attachment behavior-the innate capacity to smile, particularly in response to the visual presentation of the human face. By the age of 2 or 3 months, the child is already beginning to smile preferentially in response to the face and voice of the primary caretakers, and by 6 months of age, a substantial bond has already been formed ***(DeMause, 1974).***

In assessing the process of attachment, one can learn much from observing the reciprocally reinforcing smiling that occurs between the3- t0 6-month-old infant and the primary attachment figure (typically, but not necessarily, the mother). If this process is progressing normally, the infant and the mother experience an obvious mutual pleasure in the interaction of smiling and "talking softly" to one another. In fact, the mutual pleasure in the interaction encourages further smiling and talking, which in turn further strengthens the emotional attachment bonds. Infants aged 3-4 months who do not smile

interactively in response to the slow approach of a smiling, nodding, and cooing human face should be considered at risk for a primary disorder of attachment (for example, a pervasive developmental disorder). Likewise, infants at 4-6 months of age who do not smile preferentially and end enthusiastically at their primary caretaker's smiling and nodding face should be considered at risk for a reactive attachment disorder (for example, maternal deprivation or disabilities in mothering) ***(Ginsburg, 1948) (Ginsbung and Oppers, 19690).***

By 6-8 months of age, the infant can clearly differentiate the primary attachment figure from other individuals. This ability is manifest even before 6 months in the wariness of infants is usually apparent. This developmental landmark of stranger distress heralds the onset of the infant's capacity to recognize and remember the mother's face in contrast to the faces of other human beings. At the same time, beginning at 8-10 months of age, the inability of the infant to evoke a memory of the mother relates to the separation anxiety that is seen with threatened absence of the mother ***(Kanner, 1957).***

An understanding of these developmental landmarks can assist in the clinical assessment of the attachment process; the physician can use information about a child's distress at strangers and the onset of separation anxiety as

early as 7-8 months of age. The infant who is normally attached is at ease and smiling while on the mother's lap and while looking at her face. As the physician approaches the infant and mother, and as the baby notices the physician, the face sobers; the infant then turns to face the mother. The child may cry vigorously when taken off the mother's lap for examination. The child's distress at seeing a strange face approaching and further distress when separated from the mother are signs that the attachmemt process is proceeding satisfactorily ***(Scharfman, 1985).***

By 12 months of age, strong attachment bonds are firmly in place with primary caretakers and, to a somewhat lesser degree, with other prominent persons involved in carind for the child. By 12 months, prolonged separation from attachment figures, particularly in the context of unfamiliar surroundings, such as a hospital, can lead to a predictable series of reactions (described by John Bowlby) involving stages of proest, despair, and, ultimately, emotional detachment ***(Farley, 1985).***

The Toddlers

The toddler stage of development begins at about 1 year of age with the onset of locomotion and the use of a pincer grasp. These 2 developmental milestones allow children first to begin to move away from their caretakers

to explore the world and second to begin to provide for themselves by self-feeding. These landmarks herald the onset of the child's innate thrust toward independence that will unfold more completely during childhood, adolescence, and early adult life. This beginning process of developing independence during the toddler years is referred to as separation-individuation. The developmental challenge of the separation, individuation phase in the child development of a sense of personal mastery and autonomy, particularly in relation to a sense of control over the child's own body. This stage of development provides children with a deep sense of pride in the experience of their own activities and accomplishments and, finally, in the achievement of bowel control ***(Kanner, 1957).***

Mahler describes the young toddler as having a love affair with world. Children at this age are enamored of their own activities and excited by all that they find in the world around them. At the same time, the children are encouraged and reinforced by the delight that they perceive in the expressions of caretakers regarding those wonderful, newly found skills ***(Lowrey, 1986).***

It is important at this point to stress the development of good self-esteem. A central concept is "mirroring," namely, the information that children learn, over time,

about themselves from the expressions on the faces of those around them and, in particular, those people to whom they are emotionally attached. The earliest underpinnings of good self-esteem arise in the reciprocal and interactive smiling that occurs between the infant and the caretakers and then continue in the delight that the caretakers feel in their toddler's emerging skills of independence. The personal pleasure that the caretaker experiences in being with the child and observing the child's newly found skills of independencies is reflected, or mirrored, back to the child as information about the self. Over many years, the facial expression of caretakers become internalized within children as deep-seated feelings and convictions about their own positive self-worth. In short, children learn much about themselves by what the world mirrors back ***(Lowrey, 1948).***

While the toddler is in the midst of a love affair with the world (in the form of physical, exploration, and beginning independence), the developmental process is complicated, at least for caretakers, by 3 conflicting developmental problems: willfulness, poor judgment, and separation anxiety. Regarding willfulness, Rene Spitz pointed out the importance of the child's acquisition and use of the word "no." After "Mama" and "Dada," "no" is frequently the first word in a child's vocabulary.

Children's use of "no," either in verbal expression, or in action through behavioral opposition, signifies the powerful wish on their part to be in control of themselves. This drive for self-control and self-determination is strong and should ultimately result in responsible, self-directed behavior in the future, but in the meantime it is responsible for the temper tantrums, the breath holding, and the oppositional behavior that characterize many toddlers. Because this strong-mindedness (and in particular the use of "no") this period is often called "the terrible two's ***(Robinson, 1957).***

One of the biggest problems regarding children's exuberance and powerful wish for self-determination is that their judgment is poor at this time. For example, running into the street thrusting objects into electrical outlets, and getting into medicine cabinets are clear and distinct dangers for the toddler. These impulses elicit in caretakers a sense of duty to limit their child's behavior-ie, the parents must say "no" themselves, figuratively and literally. The first noticeable conflict between caretaker and child now surfaces, and that state of conflict, although absolutely necessary, is experienced as unpleasant by both child and caretaker. It marks the future, and it places caretakers in the sometimes difficult position of feeling like "the heavy." An important key to

child rearing (in relation to the child's innate thrust toward functioning independently) is to strike a balance: autonomous behavior is enthusiastically rewarded and supported, while dangerous, destructive, or disruptive behavior is consistently and calmly limited ***(Bowlby, 1961).***

The child's delight with activity and exploration is further complicated by the presence of separation anxiety. Separation anxiety begins as early as 8-10 months of age, generally peaks during the middle of the second year of life, and then gradually subsides in the latter part of the second and the beginning of the third year of life. Separation anxiety becomes manifest when the toddler perceives the threatened loss of the support of the primary attachment figures. Signs typically include a distressed expression, with or without crying, and active behavioral attempts on the part of the toddler to become reunited with the parenting figures. Separation anxiety is thought by Bourlby to represent a genetically determined adaptive mechanism that promtes the safety of young chidren by increasing physical proximity with caretakers. The need of toddlers is "emotionally refueled" by intermittent contact with their primary attachment figures during times of exploration of the environment. Depending on the intensity of the separation anxiety, the toddler may need

only brief visual contact with the parent, or the child may need physical contact and even comfort from that parent. Although remnants of separation anxiety can be seen in young school-age children, particularly in times of stress such as the start of school, the intensity and frequency of separation anxiety are thought to wane in the third year of life as the become able to calm themselves by evoking the image of their attachment figures or, at least, the feelings of safety associated with them ***(Metcalf, 1988).***

In addition, the array of factors that come to play in the important achievement of bowel control must be mentioned. The development of bowel control can be viewed as a prototype for the development of self-control in a more general sense. First, adults need to remind themselves that bowel movement. in and of itself, is not inherently unpleaasant or disgusting to young toddlers. Toddlers play happily while soiled or even with feces. In fact, 2-and 3-year-olds often express pleasure in the "wonderful" bowel movement that they have produced. The importance of this concept lies in the fact that the child must accept and take on caretaker's view that a bowel movement is something that belongs only in certain places (ie, in the potty) and does not belong in public (eg, in one's pants).

To incorporate the caretakers' point of view about

bowel movement, the child must have the desire to please them. This desire implies a strong and positive attachment to caretakers, and it implies that the child's needs, eg, appropriate care, emotional responsiveness, and positive mirroring responses from the parents, are being met. In short, the relationship between the child and caretaker is not tinged with the anger that exhausted and disillusioned caretakers feel when dealing with demanding and frustrated children ***(Goldings, 1979).***

Finally, the wish to please the responsive parent must then be reconciled with the toddlers' powerful wish to be in control of themselves. If the child's wish for self-determination is not adequately respected nor the playful explorations encouraged, the child then feels overly controlled or coerced by too many "no's" From caretakers. The child can then develop an attitude that reflect, this view: "One thing that you cannot is where I put my bowel movement." And, in this case, the child is correct.

If, however, the child's developmental thrust toward exploration and independence has not been faced by too many stifling parental "no's," and if the parent-child relationship is such that the child wishes to please those parents, the child will then internalize the parent's attitude about where to put bowel movements (ie, how to behave);

such children experience the personal pleasure and pride associated with their own correct use of the potty. In the end, children who voluntarily elect to use the toilet not only experience the positive reinforcement from their caretakers but also at the same time feel a sense of personal pride in their own accomplishment. An important step toward socially appropriate autonomous functioning has been taken ***(Mercalf, 1988).***

The Preschool Child

By the time children reach 3 years of age, a prominent developmental shift is already occurring: the child no longer behaves simply in relation to caretakers. The focus turns to the mental world of thoughts and feelings within the child. This shift was described by Piaget as a movement from a plane of action (the sensory-motor stage of cognitive development) to the plane of thought (the preoperational stage of cognitive development). While the behavior of the infant is directed primarily toward facilitating and maintaining attachment with primary caretakers and that of the toddler toward expressing the beginning of independence from the parents, the preschool child is mentally aware of thoughts and feedings regarding the caretakers. The 3-and 4-year-old child can begin to think (albeit very illogically), and with that developmental achievement a tremendous new world of

thought, fantasy, and worry opens for the child ***(Barker, 1986).***

The central developmental task of the preschool child is essentially the sorting out of the wondrous and sometimes frightening world that comes with thought. This new cognizance includes awareness of such exciting issues as the perception of differences between the sexes and the ensuing curiosity and worry about those differences. It includes the boastful wish to be big and powerful and the fantasy of being on a par with one's parents and even being able to displace them from their perceived position of power and privilege.

Because preschoolers perceive themselves as being at the center of the universe, they come to believe that everyone around them must be also aware of their thoughts and motives. This perceived exposure makes the mental world of the preschool child filed with unspeakable dangers, leading to worries about caretaker's withdrawal of their love and admiration, and even angry and physical retaliation against the child. Preschool children are caught between exciting and wondrous fantasies on the one hand and prohibitions, dangers, and worries on the other.

The outward behavioral manifestations of this wondrous world of thought and fantasy include "showing

off" behaviors, exhibiting the "private parts," sexual curiosity (exploration and comparison between children), and competitive struggles for favored status with parents. At the same time, children display the content of their mental worlds in symbolic play. The little girl may act out coming home to favoured status with daddy, and the little boy may portray superboy slaying a dangerous dragon ***(Hertz, 1988).***

The frightening and worrisome aspects of the child's mental world become manifest in fears (particularly at night) about ghosts, monsters, dangerous animals, and the dark. In addition, children may even develop transient phobic symptoms that represent the displacement of internal worries onto objects in the outside world. Finally, preschool children are usually greatly concerned about bodily injury; thus, everyday "ouchies" need special care, and trips to the doctor for shots, stitches, and throat cultures are associated with considerable anxiety ***(Lapouse and Monk, 1959).***

The preschool stage of mental development comes to a close as the child learns to moderate, and in a sense give up, the wild fantasies of power and privilege by identifying with the rules and the selfcontrol represented by the child's own loved ones. The internalization of parental rules and values means that children no longer

have to worry so intensely about angering people they love and depend on. Furthermore, despite all the emotional turmoil, the child remains the apple of the parent's eye. Recognizing this fact ultimately leaves the child in a calmer mental state. Generally, the child clearly identifies with an adult model of the same sex and is now ready to focus mental energies on adapting to the challenges at school and in the social community beyond home *(Scharfman, 1985).*

The School-Aged Child

Although the child of gradeschool age may still show residual evidence of earlier developmental challenges in the form of stress-induced separation anxiety (for example, with the start of school) and persistent nighttime fears, the child at this age should generally be prepared to devote mental energies to learning and to expanding social interactions beyond the family. The developmental challenge of this age is the development of confidence in mastering skills, whether athletic, academic, or social.

By the age of 7-8 years, the school-age child has developed the more logical and coherent thought processes that are necessary for more formal academic learning (Piaget's stage of concrete mental operations, during which logical relationships of cause and effect begin to exist). At the same time, the school-age child

becomes more and more involved with organized peer group activities (for example, clubs and teams), during which the rules of the game and acceptable codes of conduct become very important. The child begins to devote considerable time, focused energy, and practice on the development of skills, whether on the athletic field, in hobbies, or in schoolwork. Girls at this age are frequently more developmentally advanced than boys, particularly in the academic work of the classroom. Kindergarten and first-grade boys are much more able to be "developmentally immature" (ie, lacking in impulse control) than are girls of the same age.

Children of this age also begin to experience pleasure in their ability to organize and catagorize information. One may see the development of collections (for example, stamps, baseball cards, and dolls) and, at the same time, the exclusion of other children from activities because they are in some way perceived as different from the rest of the peer group ***(Sours, 1988)***.

During the school-age years, children should develop a sense of confidence about their abilities. The development of skills (whether athletic, academic, or social) is an important source of enhancement to self-esteem. Children with learning disorders, developmental disabilities, or temperamental variations,

including problems with attention or self-control, are at risk for developing deficiencies in self-esteem. Likewise, families that place unrealistically high expectations of performance on their children may in fact emotionally burden them; these children feel they must excel to remain in parental favor ***(Scharfman, 1985) (Campbell, 1986).***

The Adolescent

Adolescence is a stage of transition between childhood, where more emotional and physical dependence on caretakers is the rule, and adulthood, where independent and autonomous functioning is sustained without the continuous support of the family of origin. The stage of adolescence is relatively long in Western society, lasting from the onset of puberty (roughly, 11 years of age) to the early period beyond the high school years (roughly, age 18-20), when the young person makes plans toward an adult identity in the working world. Theoreticians refer to this developmental period as the second period of separation and individuation (a reliving of aspects of the "terrible two's"). For parents, it can be a dreaded period of turmoil and rebellion in their children. In fact, the developmental tasks of adolescence are quite noble: developing a unique identity (answering the question, "Who am I?") and letting go the dependent childhood attachments to primary caretakers. It is more the form of the journey that stirs concern among those responsible for the care of adolescents ***(Sours, 1988).***

Assessments of Growth

1. Assessments of osseousmaturation

The ossification of the skeleton of the fetus begins by about the fifth month and from that time makes considerable demands upon the maternal supply of bone-forming substances. Ossifcation occurs earliest in the clavicle and membranous bone of the skull, and follows rapidly in long bones and spine. The distal femoral and proximal tibial epiphyses are usually ossified in the normal full-term infant. The fusion of the humeral capitellum with the shaft is said to mark the end of the period of most rapid growth in girls and to predict the menarche within the next year.

There is no better index of general growth than bone age as determined from roentgenograms. This is based (1) on the number and size of epiphyseal centers at agiven chronologic age, (2) on the size, shape, density, and sharpness of outline of the ends of bones, and (3) on the distance separating epiphyseal zone of provisional calcification or the degree of fusion between these 2 elements. The information gained from the various epiphyseal areas varies with chronologic age. The hand

and wrist are useful at all ages of childhood; useful information can also be derived from the lower extremity ***(Yogman et al., 1988)***, especially in early infancy. The most widely used standards are those of Todd, of Greulich and Pyle, and of Vogt and Vickers for the hand. Reynolds and Asakawa have provided useful standards for the lower extremity, head of the humerus, and capitellum in early infancy. (Figs. 2 & 3 and Table 5 show expected times of appearance of various ossification centers with normal variabilities for each. Since girls are more all ages, separate standards are necessary.

No interpretation of skeletal age should fail to take into account that 1 normal child in 20 can be expected to have a skeletal age either advanced or retarded by 2 standard deviations from the mean for his chronologic age. Date of Pyle, Reed and Stuart indicate that in boys the standard deviation of bone age (given by the norms of Greulich and Pyle) around chronologic age is about 2 months in the first year of life, and increases to 4 months during the second year, to 6 months during the third year, and to 10 months by the seventh year. Thereafter, for the period rest of the growth period, the standard deviation is about 12 to 15 months. The variability is less for girls than for boys, especially in later childhood. The theoretical percentile points corresponding to such variability can be calculated ***(Dixon and Stein et al., 1987)***.

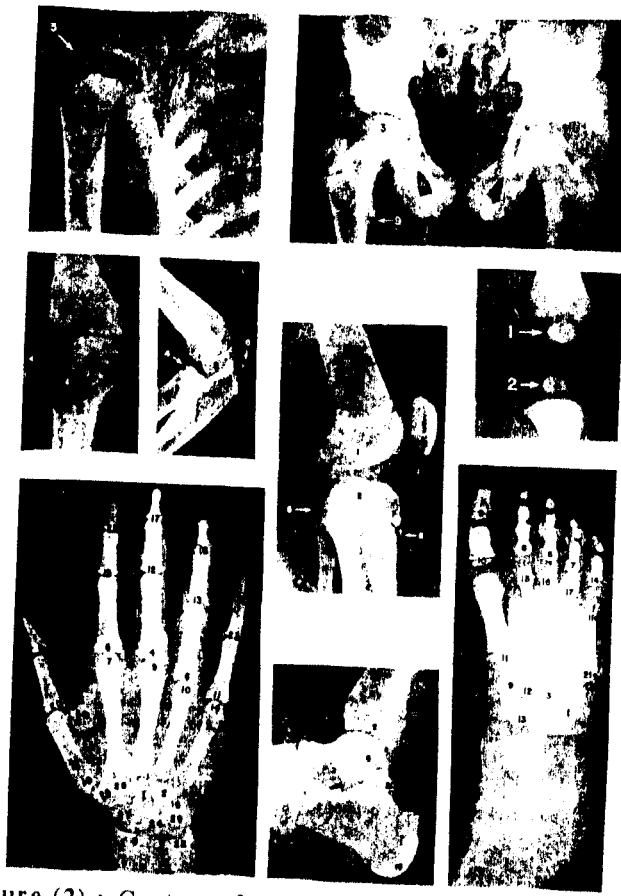

Figure (2) : Centers of ossification in the extremities.

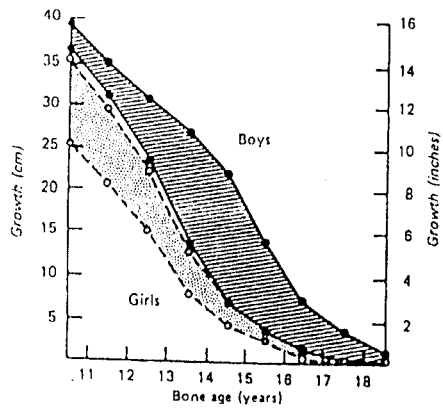

Figure (3) : Growth expectancy at bone ages indicated.

Table (5) : Ages at onset of ossification, recognized by Appearance of centers in roentgenograms, useful as maturity indicators during infancy and childhood

Boys			No. Corresponing to center in fig. 2-9	Bone and ossefication center	Girls		
Mean		Standardd Deviation*			Mean		Standardd Deviation*
Yes.	Mos.	Mos.			Yes.	Mos.	Mos.
				Shoulder and Elbow			
3 weeks					3 weeks		
0	7	4	-	Humerus, head	0	4	-
1	1	7	4	Humerus, capitellum	0	6	2
5	5	15	7	Humerus, greater tuberosity	4	1	3
6	1	15	15	Radius, proximal epiphysis	3	7	14
-	-	-	15	Humerus, medial epicondyle	-	-	12
-	-	-	-	Ulna, olecranon, 1	-	-	-
-	-	-	-	Humerus, trochlea	-	-	-
-	-	-	-	Humerus, lateral epicondyle	-	-	-
-	-	-	-	Ulna, olecranon, 2	-	-	-
				Hand and Wrist			
0	2	2	2	Capitate	0	2	2
0	3	2	2	Hamate	0	2	2
1	1	5	5	Distal epiphysis, raddius	0	10	4
1	4	4	4	Proximal epiphysis, 3rd finger	0	10	3
1	4	4	4	Proximal epiphysis, 2nd finger	0	11	3
1	5	5	5	Proximal epiphysis, 4th finger	0	11	3
1	6	5	5	Epiphysis of metacarpal II	1	0	3
1	7	7	7	Distaal epiphysis, 1st finger	1	0	4
1	8	5	5	Epiphysis of metacarpal III	1	1	3
1	11	6	6	Epiphysis of metacarpal IV	1	3	4
1	9	5	5	Proximal epiphysis, 5th finger	1	2	4
2	0	6	6	Proximal epiphysis, 3rd finger	1	3	5
2	0	6	6	Proximal epiphysis, 4th finger	1	3	5
2	2	7	7	Epiphysis of metacarpal V	1	4	5
2	2	6	6	Middle epiphysis, 2nd finger	1	4	5
2	6	16	16	Triquetral	1	9	14
2	4	6	6	Distal epiphysis, 3rd finger	1	6	4
2	4	6	6	Distal epiphysis, 4th finger	1	6	15
2	8	9	9	Epiphysis of metacarpal I	1	6	5
2	8	7	7	Proximal epiphysis, 1st finger	1	8	5
3	1	9	9	Distal epiphysis, 5th finger	1	11	6
3	1	8	8	Distal epiphysis, 2nd finger	1	11	6
3	3	10	10	Middle epiphysis, 5th finger	1	10	7
3	6	19	19	Lunate	2	10	13
5	7	19	19	Greater multangular	3	11	14
5		15	15	Lesser multangular	4	1	12
5	6	15	15	Navicular (hand)	4	3	12
6	10	14	14	Distal epiphysis of ulna	5	9	13
-	-	-	-	Pisiform	-	-	-
12	8	18	18	Sesamoid in adductor pollicis	10	1	13
				Hand and Wrist			
(Usually at birth)			1	Femur, distal epiphysis	(Usually at birth)		
(Usually at birth)			2	Tibia, proximal epiphysis	(Usually at birth)		
0	4	2	3	Femur, head	0	4	2
3	6	10	4	Femur, greater trochanter	2	5	5
3	9	12	5	Fibula, proximal epiphysis	2	9	11
3	10	11	6	Patella	2	5	7
-	-	-	7	Tibia, tuberosity, 1	-	-	-
-	-	-	8	Tibia, tuberosity, 2	-	-	-

* Standard deviation adjusted to nearest month. The range included between minus 1 and plus 1 standard deviation from the mean for any center will usually include about 68 per cent of a population of healthy children.

Table (5) : (Continued)

Boys			No. Corresponing to center in fig. 2-9	Bone and ossefication center	Girls		
Mean		Standardd Deviation*			Mean		Standardd Deviation*
Yes.	Mos.	Mos.			Yes.	Mos.	Mos.
				Foot and Ankle			
2 weeks		-	1	Cuboid	3 weeks		-
0	4	2	2	Tibia, distal epiphysis	0	4	1
0	4	4	3	Lateral cuneiform	0	4	4
1	1	4	4	Fibula, distal epiphysis	0		3
1	4	16	5	Distal epiphysis, great toe	0	9	3
1	7	5	6	Proximal epiphysis, 3rd toe	0	11	4
1	8	5	7	Proximal epiphysis, 4th toe	1	1	4
1	9	5	8	Proximal epiphysis, 2nd toe	1	1	4
2	1	10	9	Medial cuneiform	1	4	7
2	4	5	10	Proximal epiphysis, great toe	1	6	4
2	5	5	11	Metatarsal I	1	7	3
2	5	9	12	Middle cuneiform	1	7	7
2	7	13	13	Navicular (foot)	1	99	10
2	7	7	14	Proximal epiphysis, 5th toe	1	8	5
2	10	7	15	Metatarsal II	2	0	5
3	5	8	16	Metatarsal III	2	5	5
3	11	8	17	Metatarsal IV	2	9	7
4	5	10	18	Distal epiphysis, metatarsal V	3	2	8
7	5	11	19	Calcaneus, epiphysis, 1	5	0	11
-	-	-	20	Accessory talus	-	-	-
-	-	-	21	Proximal epiphysis, metatarsal V	-	-	-
-	-	-	22	Calcaneus, epiphysis, 2	-	-	-

Age at onset of fusion in skeletal regions useful as maturity indicators during adolescence

Boys		Girls
Modal skeletal age in Years*	Skeletal region	Modal skeletal age in Years*
	Elbow	
13.5 - 13.5	Begins in humerus	11.0 - 11.5
15.0 - 15.5	Completed in ulna	12.5 - 13.0
	Foot and ankle	
14.0 - 14.5	Begins in great toe	12.5 - 13.0
15.5 - 16.0	Cjompleted in tibia and fibula	14.0 - 14.5
	Hand and wrist	
15.0 - 15.5	Begins in distal phalanges	13.0 - 13.5
17.5 - 18.0	Completed in radius	16.0 - 16.5
	Knee	
15.0 - 15.5	Begins in tibial tuberosity	13.5 - 14.0
17.5 - 18.0	Completed in fibula	16.0 - 16.5
	Hip and pelvis	
15.5 - 16.0	Begins in greater trochanter	14.0 - 14.5
after 18.0	Completed in symphysis	17.5 - 18.0
	Shoulder and shoulder girdle	
15.5 - 16.0	Begins in greater tuberosity	14.0 - 14.5
after 18.0	Completed in clavicle	17.5 - 18.0

Table (6) : Chronology of human dentition primary or deciduous teeth

	Calcification		Eruption		Shedding	
	Begins at	Complete at	Maxillary	Mandibular	Maxillary	Mandibular
Central incisors	5th fetal month	18 - 24 months	6 - 8 months	5 - 7 months	7 - 8 years	6 - 7 years
Lateral incisors	5th fetal month	18 - 24 months	8 11 months	7 - 10 months	8 - 9 years	7 - 8 years
Cuspids (canines)	6th fetal month	30 - 36 month	16 - 20 months	16 - 20 months	11 - 12 years	9 - 11 years
First molars	5th fetal month	24 - 30 months	10 - 16 months	10 - 16 months	10 - 11 years	10 - 12 years
Second molars	6th fetal month	36 months	20 - 30 months	20 - 30 months	10 - 12 years	11 - 18 years

Secondary or Permanent Teeth

		Calcification		Eruption	
		Begins at	Complete at	Maxillary	Mandibular
Central incisors	3 - 4 months		9 - 10 years	7 - 8 years	6 - 7 years
Lateral incisors	Max., 10 - 12 months Mand., 3 - 4 months		10 - 11 years	8 - 9 years	7 - 8 years
Cuspids (canines)	4 - 5 months		12 - 15 years	11 - 12 years	9 - 11 years
First premolars	18 - 21 months		12 - 13 years	10 - 11 years	10 - 12 years
Second premolars	24 - 30 months		12 - 14 years	10 - 12 years	11 - 13 years
First molars	Birth		1 - 10 years	6 - 7 years	6 - 7 years
Second molars	30 - 36 months		14 - 16 years	12 - 13 years	12 - 13 years
Third molars	Max., 7 - 9 years Mand., 8 - 10 years		18 - 25 years	17 - 22 years	17 - 22 years

Table (6) : Chronology of human dentition primary or deciduous teeth

	Calcification		Eruption		Shedding	
	Begins at	Complete at	Maxillary	Mandibular	Maxillary	Mandibular
Central incisors	5th fetal month	18 - 24 months	6 - 8 months	5 - 7 months	7 - 8 years	6 - 7 years
Lateral incisors	5th fetal month	18 - 24 months	8 11 months	7 - 10 months	8 - 9 years	7 - 8 years
Cuspids (canines)	6th fetal month	30 - 36 month	16 - 20 months	16 - 20 months	11 - 12 years	9 - 11 years
First molars	5th fetal month	24 - 30 months	10 - 16 months	10 - 16 months	10 - 11 years	10 - 12 years
Second molars	6th fetal month	36 months	20 - 30 months	20 - 30 months	10 - 12 years	11 - 18 years

Secondary or Permanent Teeth

		Calcification	Eruption	
	Begins at	Complete at	Maxillary	Mandibular
Central incisors	3 - 4 months	9 - 10 years	7 - 8 years	6 - 7 years
Lateral incisors	Max., 10 - 12 months Mand., 3 - 4 months	10 - 11 years	8 - 9 years	7 - 8 years
Cuspids (canines)	4 - 5 months	12 - 15 years	11 - 12 years	9 - 11 years
First premolars	18 - 21 months	12 - 13 years	10 - 11 years	10 - 12 years
Second premolars	24 - 30 months	12 - 14 years	10 - 12 years	11 - 13 years
First molars	Birth	1 - 10 years	6 - 7 years	6 - 7 years
Second molars	30 - 36 months	14 - 16 years	12 - 13 years	12 - 13 years
Third molars	Max., 7 - 9 years Mand., 8 - 10 years	18 - 25 years	17 - 22 years	17 - 22 years

eruption prevents such delay from being useful as an index of a growth disorder. In some families the children have conspicuously early or late dentition without other signs of retardation or acceleration of growth.

The first permanent teeth to erupt are the 6- year molars; they are often mistaken for deciduous teeth by the uninformed. The first permanent molars serve focal points in the dental arch and so have a great deal to do with the ultimate shape of the jaw and the orderly arrangement of teeth. Caries or other defects in them should receive prompt attention; these teeth should not be extracted ***(Kagan, 1982).***

3. Assessment of nutrition and metabolism

The infant's and child's nutritional requirements increase with growth in size. The parameter of growth with which many of the nutritional factors bear the most nearly constant relation is body surface, which appears to be as closely related to the body's mass of metabolically active tissue as any other simple measurement. Owing, however, to fundamental differences in the metabolic activity of infants and children at various ages, adjustments may be necessary. This is particularly evident with respect to administration of drugs in the neonatal period.

Measurements of body surface which correspond to given heights and weights are available; reasonably accurate estimates of body surface can be obtained from normograms. Cruder estimates of body surface from weight only can be made for children whose physique is average; Lowe's formula is :

surface area(M^2) = $\sqrt[3]{Wt.^2 (kg.) \times 0.1}$

Another crude estimate for children of average physique is given by the simpler formulas:

Approximation of surface Area (M^2) to weight (kg.)

Weight range	Approximate surface area
1 to 5 kg	$M^2 = 0.05 \times kg. + 0.05$
6 to 10 kg	$M^2 = 0.04 \times kg. + 0.10$
11 to 20 kg	$M^2 = 0.03 \times kg. + 0.20$
21 to 40 kg	$M^2 = 0.02 \times kg. + 0.40$

Examples:

for 7 kg. infant, area (M^2) = $0.04 \times 7 + 0.10 = 0.38\ M^2$

for 17-kg. infant, area (M^2) = $0.03 \times 17 + 0.20 = 0.71\ M^2$

(estimates of o.4 M^2 and o.7 M^2 respectively would be reasonable)

(The formula $M^2 = 0.02 \times kg. + 0.40$ is reasonably accurate from 21 to 70 kg.)

Basal caloric needs, when referred to body surface,

appear to be somewhat lower in premature infants than in full- term ones. They increase during the first year of life from approximately 30 calories per square meter per hour to about 50 by the second year, with a subsequent fall to adult levels of 35 to 40 calories per square meter per hour. The data of Lewis indicate that the rate of fall is slowed during prepubertal and adolescent years, owing to the need for additional energy for accelerated growth.

Needs for water and electrolytes remain roughly constant in their proportion to body surface through most of the growing period; the inevitable variations in intake are met by the capacity of homeostatic mechenisms to adjust to varying conditions of supply and demand. Tallbot, Richie and Crawford have outlined the limits within which the body is equipped to adjust to variations in intake and output ***(KAGAN, 1982).***

4. Assessment of physical growth and development

Appraisal of growth and development in the infant and the child has its greatest usefulness only if it is accurate and continuous in each of the areas in which changes can be observed. In the infant the most useful physical measurements are head circcumference, length and weight (Figs. 3, 4, 5, 6, 7 and 8) and (Tables 7, 8 and 9). note should also be made of the nutritional state, dentiton, and the size or patency of fontanels. In selected

instances measurements of the thickness of subcutaneous tissue or the lengths of body segments may be appropriate ***(Brazelton and Yogman, 1986).***

Table (7) : Percentiles for weight and length - birth to 5 years

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							Birth							
5.80	6.30	6.90	7.50	8.30	9.10	10.10	Weight in Pounds	5.80	6.20	6.90	7.40	8.10	8.60	9.40
2.63	2.86	3.13	3.40	3.76	4.13	4.58	Weight in Kg.	2.63	2.81	3.13	3.36	3.67	3.90	4.26
18.20	18.90	19.40	19.90	20.50	21.00	21.50	Length in Inches	18.50	18.80	19.30	19.80	20.10	20.40	21.10
46.30	48.10	49.30	50.60	52.00	53.30	54.60	Length in Cm.	47.10	47.80	49.00	50.20	51.00	51.90	53.60
							3 Months							
10.60	11.10	11.80	12.60	13.60	14.50	16.40	Weight in Pounds	9.80	10.70	11.40	12.40	13.20	14.00	14.90
4.81	5.03	5.35	5.72	6.17	6.58	7.44	Weight in Kg.	4.45	4.85	5.17	5.62	5.99	6.35	6.76
22.40	22.80	23.30	23.80	24.30	24.70	25.10	Length in Inches	22.00	22.40	22.80	23.40	23.90	24.30	24.80
56.80	57.80	59.30	60.40	61.80	62.80	63.70	Length in Cm.	55.80	56.90	57.90	59.50	60.70	61.70	63.10
							6 Months							
14.00	14.80	15.60	16.70	18.00	19.20	20.80	Weight in Pounds	12.70	14.10	15.00	16.00	17.50	18.60	20.00
6.35	6.71	7.08	7.58	8.16	8.71	9.43	Weight in Kg.	5.76	6.40	6.80	7.26	7.94	8.44	9.07
24.80	25.20	25.70	26.10	26.70	27.30	27.70	Length in Inches	24.00	24.60	25.10	25.70	26.20	26.70	27.10
63.00	63.90	65.20	66.40	67.80	69.30	70.40	Length in Cm.	61.10	62.50	63.70	65.20	66.60	67.80	68.80
							9 Months							
16.60	17.80	18.70	20.00	21.50	22.90	24.40	Weight in Pounds	15.10	16.60	17.80	19.20	20.80	22.40	24.20
7.53	8.07	8.48	9.07	9.75	10.39	11.07	Weight in Kg.	6.85	7.53	8.03	8.71	9.43	10.16	10.98
26.60	27.00	27.50	98.00	28.70	2.20	29.90	Length in Inches	25.70	26.40	26.90	27.60	28.20	28.70	29.20
67.70	68.60	69.80	71.20	72.90	74.20	75.90	Length in Cm.	65.40	67.00	68.40	70.10	71.70	72.90	74.10
							12 Months							
18.50	19.60	20.90	22.20	23.80	25.40	27.30	Weight in Pounds	16.80	18.40	19.80	21.50	23.00	27.80	27.10
8.39	8.89	9.48	10.07	10.80	11.52	12.38	Weight in Kg.	7.62	8.35	8.98	9.75	10.43	11.25	12.29
28.10	28.50	29.00	29.60	30.30	30.70	31.60	Length in Inches	27.10	27.80	28.50	29.20	29.90	30.30	31.00
71.30	72.40	73.70	75.20	76.90	78.10	80.30	Length in Cm.	68.90	70.60	72.30	74.20	75.90	77.10	78.80

Table (7) : (Continued)

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							15 Months							
19.80	21.00	22.40	23.70	25.40	27.20	29.40	Weight in Pounds	18.10	19.80	21.30	23.00	24.60	26.60	29.00
8.98	9.53	10.16	10.75	11.52	12.34	13.33	Weight in Kg.	8.21	8.98	9.66	10.43	11.16	12.07	13.15
29.30	29.80	30.30	30.90	31.60	32.10	33.10	Length in Inches	28.30	29.00	29.80	30.50	31.30	31.80	32.60
74.40	75.60	77.00	78.50	80.30	81.50	84.20	Length in Cm.	71.90	73.70	75.60	77.60	79.40	80.80	82.80
							18 Months							
21.10	22.30	23.80	25.20	26.90	29.00	31.50	Weight in Pounds	19.40	21.20	22.70	24.50	26.20	28.30	30.90
9.57	10.12	10.80	11.43	12.20	13.15	14.29	Weight in Kg.	8.80	9.62	10.30	11.11	11.88	12.84	14.02
30.50	31.80	31.60	32.20	32.90	33.5	34.70	Length in Inches	29.50	30.20	31.10	31.80	32.60	33.30	34.10
77.50	78.80	80.30	81.80	83.70	85.00	88.20	Length in Cm.	74.90	76.80	79.00	80.90	82.90	84.50	86.70
							2 Years							
23.30	24.70	26.30	27.70	29.70	31.90	34.90	Weight in Pounds	21.60	23.50	25.30	27.10	29.20	31.70	34.40
10.57	11.20	11.93	12.56	13.47	14.47	15.83	Weight in Kg.	9.80	10.66	11.48	12.29	13.25	14.38	15.60
32.60	33.10	33.80	34.40	35.20	35.90	37.20	Length in Inches	31.50	32.30	33.30	34.10	35.00	35.80	36.70
82.70	84.20	85.80	87.50	89.40	91.10	94.60	Length in Cm.	80.10	82.00	84.70	86.60	88.90	91.00	90.30
							2.5 Years							
25.20	26.60	28.40	30.00	32.20	34.50	37.00	Weight in Pounds	23.60	25.50	27.40	29.60	31.90	34.60	38.20
11.43	12.07	12.88	13.61	14.61	15.65	16.78	Weight in Kg.	10.70	11.57	12.43	13.43	14.47	15.69	17.33
34.20	34.80	35.50	36.30	37.00	37.90	39.20	Length in Inches	23.30	34.00	35.20	36.00	36.90	37.90	38.90
86.90	88.50	90.20	92.10	94.10	96.20	99.50	Length in Cm.	84.50	86.30	89.30	91.40	93.80	96.40	98.70
							3 Years							
27.00	28.70	30.30	32.20	34.50	36.80	39.20	Weight in Pounds	25.60	27.60	29.60	31.80	34.60	37.40	41.80
12.25	13.02	13.74	14.61	15.65	16.69	17.78	Weight in Kg.	11.61	12.52	13.43	14.42	15.69	16.96	18.96
25.70	36.30	37.00	37.90	38.80	39.60	40.50	Length in Inches	34.80	35.60	36.80	37.70	38.60	39.80	40.70
90.60	92.30	93.90	96.20	98.50	100.5	102.8	Length in Cm.	88.40	90.50	93.40	95.70	98.10	101.1	103.5

Table (7) : (Continued)

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							3.5 Years							
28.50	30.40	32.30	34.30	36.70	39.10	41.50	Weight in Pounds	27.50	29.50	31.50	33.90	37.00	40.40	45.3
12.93	13.79	14.65	15.55	16.65	17.74	18.82	Weight in Kg.	12.47	13.38	14.29	15.38	16.78	18.33	20.55
37.10	37.80	38.40	39.30	40.30	41.10	41.90	Length in Inches	36.20	37.10	38.10	39.20	40.20	41.50	42.50
94.30	96.00	97.50	99.80	102.5	104.5	106.5	Length in Cm.	92.00	94.20	96.90	99.50	102.0	105.4	108.0
							4 Years							
30.10	32.10	34.00	36.40	39.00	41.40	44.30	Weight in Pounds	29.20	31.20	33.50	36.20	39.60	43.50	48.20
13.65	14.56	15.42	16.51	17.69	18.78	20.09	Weight in Kg.	13.25	14.15	15.20	16.42	17.96	19.73	21.86
38.40	39.10	39.70	40.70	41.90	42.70	43.50	Length in Inches	37.50	38.40	39.50	40.60	41.60	43.10	44.20
97.50	99.30	100.8	103.4	106.5	108.5	110.4	Length in Cm.	95.20	97.60	100.3	103.2	105.8	109.6	112.3
							4.5 Years							
31.60	33.80	35.70	38.40	41.40	34.90	47.40	Weight in Pounds	30.70	32.90	35.30	38.50	42.10	46.70	50.90
14.33	15.33	16.19	17.42	18.78	19.91	21.50	Weight in Kg.	13.93	14.92	16.01	17.46	19.10	21.18	23.09
39.60	40.30	40.90	42.00	43.30	44.20	45.00	Length in Inches	38.60	39.70	40.80	42.00	43.00	44.70	45.70
100.6	102.4	104.0	106.7	109.9	112.3	114.3	Length in Cm.	98.10	100.9	103.6	106.8	109.3	113.5	116.2
							5 Years							
33.60	35.50	37.50	40.50	44.10	46.70	50.40	Weight in Pounds	32.10	34.80	37.40	40.50	44.80	49.20	52.80
15.24	16.10	17.01	18.37	20.00	21.18	22.86	Weight in Kg.	14.56	15.79	16.96	18.37	20.32	22.32	23.95
40.20	40.80	41.70	42.80	44.20	45.20	46.10	Length in Inches	39.40	40.50	41.60	42.90	44.00	45.40	46.80
102.0	103.7	105.9	108.7	112.3	114.7	117.1	Length in Cm.	100.0	103.0	105.7	109.1	111.7	115.4	118.8

From Studies of Child Health and Development, Department of Maternal and Child Health, Harvard School of Public Health.

* The figures for the several percentiles of each measurement at 5 years differ slightly from those given in Table 2-8 for this age because they were obtained from a different population of children.

Table (8) : Percentiles for weight and height - 5 to 15 years

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							5 Years*							
34.5	36.60	39.60	42.80	46.50	49.70	53.20	Weight in Pounds	33.70	36.10	38.60	41.40	44.20	48.20	51.80
15.65	16.6	17.96	19.41	21.09	22.54	24.13	Weight in Kg.	15.29	16.37	17.51	18.78	20.05	21.86	23.50
40.20	41.50	42.60	43.80	45.00	45.90	47.00	Length in Inches	9	41.30	42.20	43.20	44.40	45.40	46.50
102.1	105.8	108.3	111.3	114.2	116.7	119.5	Length in Cm.	40.40 102.6	105.0	107.2	109.7	112.9	115.4	118.0
							5.5 Years							
	39.80	42.00	45.60	430	53.10		Weight in Pounds		38.00	40.80	44.00	47.20	51.20	
	17.60	19.05	20.68	22.36	24.09		Weight in Kg.		17.24	18.51	19.96	21.41	23.22	
	42.60	43.80	45.00	46.30	47.80		Length in Inches		42.40	43.40	44.40	45.70	46.80	
	108.3	111.2	114.4	117.5	120.1		Length in Cm.		107.8	110.2	112.8	116.1	118.9	
							6 Years							
38.50	40.90	44.40	48.30	52.10	56.40	61.10	Weight in Pounds	37.20	39.60	42.90	46.50	50.20	54.20	58.70
17.46	18.55	20.14	21.91	23.63	25.58	27.71	Weight in Kg.	16.87	17.96	19.46	21.09	22.70	24.58	26.63
42.70	43.80	44.90	46.30	47.60	48.60	49.70	Length in Inches	42.50	43.50	44.60	45.60	47.00	48.10	49.40
108.5 0	111.2	114.1	117.5	120.8	123.5	126.2	Length in Cm.	108.0	110.6	113.2	115.9	119.3	122.3	125.4
							6.5 Years							
	43.40	47.10	51.20	55.40	60.40		Weight in Pounds		42.20	45.50	49.40	53.30	57.70	
	19.69	21.36	23.22	25.13	27.40		Weight in Kg.		19.14	20.64	22.41	24.18	26.17	
	44.90	46.10	47.60	48.90	50.00		Length in Inches		44.80	45.70	46.90	48.30	49.40	
	114.1	117.2	120.8	124.2	127.0		Length in Cm.		113.7	116.2	119.1	122.6	125.6	
							7 Years							
43.00	45.80	49.70	54.10	58.70	64.40	69.90	Weight in Pounds	41.30	44.50	48.10	52.20	56.30	61.20	76.30
1.50	20.77	22.54	24.54	26.63	29.21	31.71	Weight in Kg.	18.73	20.19	21.82	23.68	25.54	27.76	30.53
44.0	46.00	47.40	48.90	50.20	51.40	52.50	Length in Inches	44.90	46.00	46.0	48.10	49.60	50.70	51.90
114.0	116.9	120.3	124.1	127.6	130.5	133.4	Length in Cm.	114.0	116.8	119.2	122.3	125.9	128.9	131.7

* The figures for the several percentiles of each measurement at 5 years differe slightly from those in Table for this age because they were obtained from a different population of children.

Table (8) : (Continued)

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							7.5 Years							
	48.50	52.60	57.10	62.10	68.70		Weight in Pounds		46.60	50.60	55.20	59.80	65.60	
	22.00	23.86	25.90	28.17	31.16		Weight in Kg.		21.14	22.95	25.04	27.18	29.76	
	47.20	48.60	50.00	51.50	52.70		Length in Inches		47.00	48.00	49.30	50.70	51.90	
	120.0	123.5	127.1	130.9	133.9		Length in Cm.		119.5	122.0	125.2	128.8	131.8	
							8 Years							
48.00	51.20	55.50	60.10	65.50	73.00	79.40	Weight in Pounds	45.30	48.60	53.10	58.10	63.30	69.90	
21.77	43.22	25.17	27.26	29.71	33.11	36.02	Weight in Kg.	20.55	22.04	24.09	26.35	28.71	31.71	
47.10	48.50	49.80	51.20	52.80	54.00	55.20	Length in Inches	46.90	18.10	49.10	50.40	51.80	53.00	
119.6	123.1	126.6	130.0	134.2	137.3	140.2	Length in Cm.	119.1	122.1	124.8	128.0	131.6	134.6	
							8.5 Years							
	53.80	58.30	63.10	68.90	77.00		Weight in Pounds		50.60	55.50	61.00	66.90	74.50	
	24.40	26.40	28.62	31.25	34.93		Weight in Kg.		22.95	25.17	27.67	30.35	33.79	
	49.50	50.80	52.30	53.90	55.10		Length in Inches		49.00	50.10	51.40	52.90	54.10	
	125.7	129.1	132.8	137.0	140.0		Length in Cm.		124.6	127.3	130.5	134.4	137.5	
							9 Years							
52.50	56.30	61.10	66.00	72.30	81.00	89.80	Weight in Pounds	49.10	52.60	57.90	63.80	70.50	79.10	
23.81	25.54	27.71	29.94	32.80	36.74	40.73	Weight in Kg.	22.27	23.86	26.26	28.94	31.98	35.88	
48.90	50.50	51.80	53.30	55.00	56.10	57.20	Length in Inches	48.70	50.00	51.10	52.30	54.00	55.30	
124.2	128.3	131.6	135.5	139.8	142.6	145.3	Length in Cm.	123.6	127.0	129.7	132.9	137.1	140.4	
							9.5 Years							
	58.70	63.70	69.00	76.00	85.50		Weight in Pounds		54.90	60.40	67.10	74.80	84.40	
	26.63	28.89	31.30	34.47	38.78		Weight in Kg.		24.90	27.40	30.44	33.98	38.28	
	51.40	52.70	54.30	55.90	57.10		Length in Inches		50.90	52.00	53.50	55.10	56.40	
	130.6	134.0	127.9	142.1	145.1		Length in Cm.		129.4	132.2	135.8	139.9	143.2	

Table (8) : (Continued)

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							10 Years							
56.80	61.10	66.30	71.90	49.60	89.90	100.0	Weight in Pounds	53.20	57.10	62.80	70.30	79.10	89.70	101.9
25.76	27.71	50.07	32.61	36.11	40.78	45.36	Weight in Kg.	24.13	25.90	28.49	31.89	35.88	40.69	46.22
50.70	52.30	53.70	55.20	56.80	58.10	59.20	Length in Inches	50.30	51.80	53.00	54.60	56.10	57.50	58.80
128.7	132.8	136.6	140.3	144.4	147.5	150.3	Length in Cm.	127.7	131.7	134.6	138.6	142.6	146.0	149.3
							10.5 Years							
	63.70	69.00	74.80	83.40	94.60		Weight in Pounds		59.90	66.40	74.60	84.10	95.10	
	28.89	31.30	33.93	37.83	42.91		Weight in Kg.		27.17	30.12	33.79	38.15	43.14	
	53.20	54.50	56.00	57.80	58.90		Length in Inches		52.90	54.10	55.80	57.40	58.90	
	135.1	138.4	142.3	146.8	149.7		Length in Cm.		134.4	137.5	141.7	145.9	149.7	
							11 Years							
61.80	66.30	71.60	77.60	87.20	99.30	111.7	Weight in Pounds	57.90	62.60	69.90	78.80	89.10	100.4	112.9
28.03	30.07	32.48	35.20	39.55	45.04	50.67	Weight in Kg.	26.26	28.40	31.71	35.74	40.42	45.54	51.21
52.50	54.00	55.3	56.80	38.70	59.80	60.80	Length in Inches	52.10	53.90	55.20	57.00	58.70	60.40	62.00
133.4	137.3	140.5	144.2	149.2	151.8	154.4	Length in Cm.	132.3	137.0	140.8	144.7	149.2	153.4	157.4
							11.5 Years							
	69.20	74.60	81.00	91.60	104.5		Weight in Pounds		66.10	74.00	83.20	94.00	106.0	
	31.39	33.84	36.74	41.55	47.40		Weight in Kg.		29.98	33.57	37.74	42.64	48.08	
	55.00	56.30	57.80	59.60	60.90		Length in Inches		55.00	56.30	58.30	60.20	61.80	
	139.8	142.9	146.9	151.4	154.8		Length in Cm.		139.8	143.1	148.1	152.9	157.0	
							12 Years							
67.20	72.00	77.50	84.40	96.00	109.6	124.2	Weight in Pounds	63.60	69.50	78.00	87.60	98.80	11.5	127.7
30.48	32.66	35.15	38.28	43.55	49.71	56.34	Weight in Kg.	28.85	31.52	35.38	99.74	44.82	50.58	57.92
54.40	56.10	57.20	58.90	60.40	62.20	63.70	Length in Inches	54.30	56.10	57.40	59.80	61.60	63.20	64.80
138.1	142.4	145.2	149.6	153.5	157.9	161.9	Length in Cm.	137.8	142.6	145.9	151.9	156.6	160.6	164.6

Table (8) : (Continued)

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							12.5 Years							
	74.60	80.60	88.70	102.0	116.4		Weight in Pounds		74.70	83.70	93.40	104.	118.0	
	33.84	36.56	40.23	46.27	52.80		Weight in Kg.		33.88	37.97	42.37	47.58	55.52	
	56.90	58.10	60.00	61.90	63.60		Length in Inches		57.40	58.80	60.70	62.60	64.00	
	144.5	147.5	152.3	157.2	161.6		Length in Cm.		145.9	149.3	154.3	159.1	162.7	
							13 Years							
72.00	77.10	83.70	93.00	107.9	123.2	138.0	Weight in Pounds	72.20	79.90	89.40	99.10	111.0	124.5	142.3
32.66	34.97	37.97	42.18	48.94	55.88	62.60	Weight in Kg.	32.75	56.24	40.55	44.95	50.35	56.47	64.55
56.00	57.70	58.90	61.00	63.30	65.10	66.70	Length in Inches	56.60	58.70	60.10	61.80	63.60	64.90	66.30
142.2	146.6	149.7	155.0	160.8	165.3	169.5	Length in Cm.	143.7	149.1	152.6	157.1	161.5	164.8	168.4
							13.5 Years							
	82.20	89.60	100.3	115.5	130.1		Weight in Pounds		85.50	94.60	103.7	115.4	128.9	
	37.20	40.64	45.50	52.39	59.01		Weight in Kg.		38.78	42.91	47.04	52.35	58.47	
	58.80	60.30	62.60	64.80	66.50		Length in Inches		59.50	60.80	62.40	64.00	65.30	
	149.4	153.1	158.9	164.6	168.9		Length in Cm.		151.1	154.4	158.4	162.6	165.9	
							14 Years							
79.80	87.20	95.50	107.6	123.1	136.9	150.6	Weight in Pounds	83.10	91.00	99.8	108.4	119.7	133.3	150.8
36.20	39.55	43.32	48.81	55.84	62.10	68.31	Weight in Kg.	37.69	41.28	45.27	49.17	54.29	60.46	68.40
57.60	59.90	61.60	64.00	66.30	67.90	69.70	Length in Inches	58.30	60.20	61.50	62.80	64.40	65.70	67.20
146.4	152.1	156.5	162.7	168.4	172.4	177.1	Length in Cm.	148.2	153.0	156.1	159.0	163.7	167.0	170.7
							14.5 Years							
	93.30	101.9	113.9	129.1	142.4		Weight in Pounds		94.20	102.5	111.0	121.8	135.7	
	42.32	46.42	51.66	58.56	64.59		Weight in Kg.		42.78	46.49	50.35	55.25	61.55	
	61.00	62.70	65.10	67.20	68.70		Length in Inches		60.70	61.80	63.10	64.70	66.00	
	155.0	159.4	165.3	170.7	174.6		Length in Cm.		154.1	156.9	160.4	164.3	167.6	
							15 Years							
91.30	99.40	108.2	120.1	135.0	147.8	161.6	Weight in Pounds	89.00	97.40	105.1	113.5	123.9	138.1	155.2
41.41	45.09	49.08	54.48	61.23	67.04	73.30	Weight in Kg.	40.37	44.18	47.67	51.48	56.20	62.64	70.4
59.70	62.10	63.90	66.1	68.10	69.6	71.60	Length in Inches	59.10	61.10	62.1	63.40	64.90	66.20	67.6
151.7	157.8	162.3	167.8	173.0	176.7	181.8	Length in Cm.	150.2	155.2	157.7	161.1	164.9	168.1	171.6

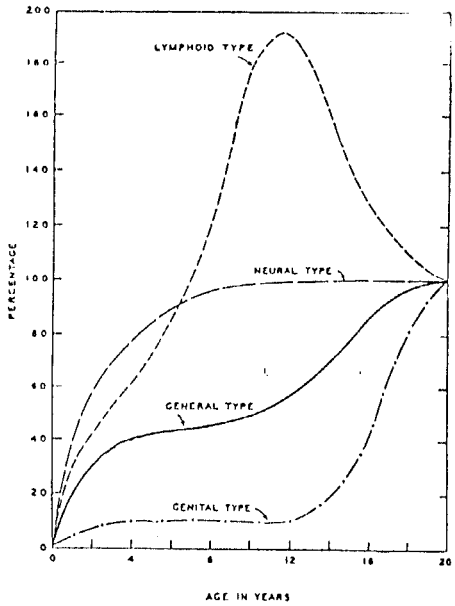

Fig. (4) : Main types of postnatal growth of the various parts and organs of the boy. (After Scammon : The Measurement of the Body in Childhood, The Measurement of Man. University of Minnesota Press.)

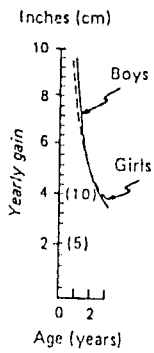

Fig. (5) : Growth rate from birth to age 3 (both sexes).

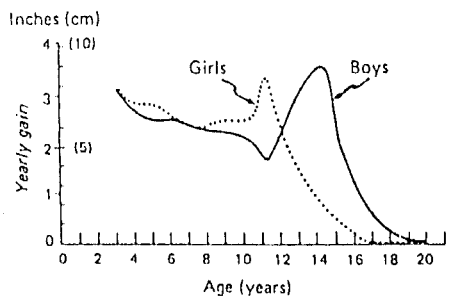

Fig. (6) : Growth rate from age 3 to 20 (both sexes).

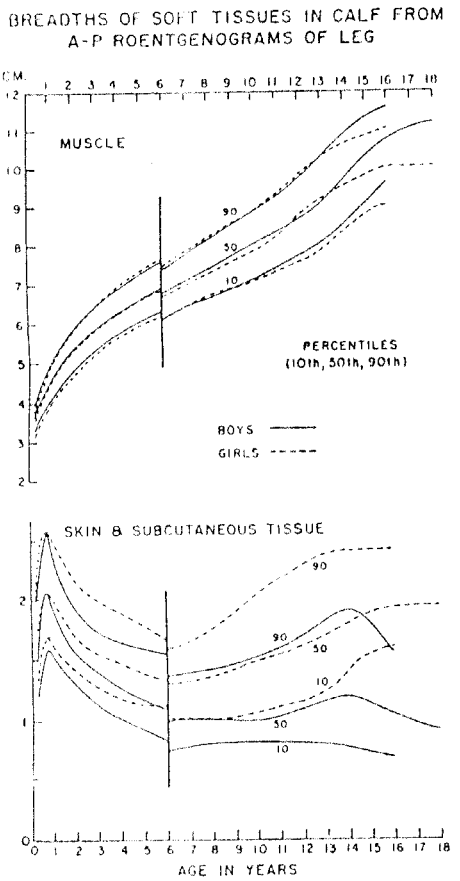

Fig. (7) : Breadths of muscle and of double layers of skin an subcutaneous tissue at greatest width of calf by age and sex from 3 months to 18 years of age.

The graphs reveal the close similarity in pattern of the curves for muscle to those of genral growth, but a unique pattern of increase and decrease and a sex difference in the skin and subcutaneous tissue. (For details, see Stuart and Sobel: *J. Pediat.*, Vol. 28, and ombar: Child Dev., Vol. 21, For distribution of subcutaneous fat in childhood and adolescence, see Reynolds: Monographs Soc. Res. Child Dev., Vol. 15.)

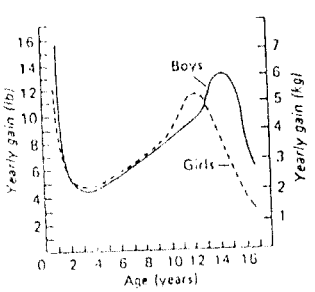

Fig. (8) : Yearly gain in weight, (Rerawn and reproduced, with permission, from Barnett HL: *Pediatrics*, 14th ed. Appleton-Century-Crofts, 1968.)

Techniques of measurement

Height. *Recumbent length* can be more accurately measured than standing height in children under the age of 5years, after which measurement of standing height is generally more convenient. Recumbent length is measured as the child lies on a firm table which has ameasuring stick at least 125 cm. or 50 inches long inserted along one edge. The soles of the feet are held firmly against a fixed upright placed at the zero mark. A movable upright crosses the table above the head and is brought firmly against the vertex. If recumbent length is used after 5 years of age, the value obtained may be reduced by 1 cm. and then considered against the scale for standing height ***(Lowrey, 1986).***

Standing height is measured as the child stands erect, his heels, buttocks, upper part of the back and occiput against a vertical; heels should be close together, and the arms should hang naturally at the sides. the external auditory meatus and the lower border of the orbit should lie in a plane parne parallel with the floor. A wooden head piece having 2 faces at right angles may be placed firmly on the head against a 2-meter or 6-foot measuring scale attached to the vertical surface against which the child is positioned ***(Freud, 1965).***

Head Circumference. This measurement is

particularly valuable in infants; it need not be taken routinely after 3 years of age. The tape is applied firmly over the glabella and supraorbital ridges anteriorly and that part of the occiput posteriorly which gives the maximal circumference. Difficulties with measurement of head circumference will sometimes arise when the head has an abnormal shape, as in hydrocephalus. Under these circumstances serial measurements of the changing size of the head may best be made through positioning the tape over whatever points on the forehead and occiput give the *maximal* circumference (Fig. 9 and 10).

Measurements of circumference should be made with steel, cloth or disposable paper tapes. Cloth tapes may stretch with aging and will need to be checked frequently against wooden or steel standards ***(Dworkin, 1988).***

Chest Circumference. Measurement of chest circumference is made in midrespiration, at the level of the xiphoid cartilage or substernal notch, in a plane at right angles to the vertebral column. Measurement is made recumbent up to the age of 5 years, the child standing thereafter ***(Kagan, 1982).***

Abdominal Circumference. This measurement is taken to 3 years only and will be of value principally in recognizing and following the course of chronic intestinal disturbances. Measurement is made in the plane of the

Table (9) : Percentiles for selected measurements - birth to 5 years - in centimeters

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							Birth							
7.1	7.4	7.7	8.1	8.4	8.7	9.0	Pelvic Breadth	7.0	7.2	7.4	7.7	8.2	8.5	8.9
33.0	33.5	34.4	35.3	36.2	37.0	37.5	Head Circ.	32.5	33.4	33.9	34.7	35.4	36.0	36.6
2.8	30.6	31.8	33.2	34.4	35.7	36.8	Chest Circ.	30.3	30.8	31.8	32.9	34.0	35.0	36.0
							3 Months							
9.8	10.0	10.2	10.6	11.2	11.5	12.1	Pelvic Breadth	9.4	9.6	9.9	10.4	10.9	11.4	12.2
38.7	39.2	40.0	40.9	41.5	42.1	43.2	Head Circ.	37.9	38.5	39.2	40.0	10.8	41.7	42.3
37.6	38.3	39.3	40.6	41.6	42.9	44.1	Chest Circ.	36.5	37.6	38.8	39.8	40.9	42.0	43.0
33.6	35.5	56.8	38.5	399.8	41.4	43.5	Abd. Circ.	32.3	34.4	36.8	38.4	40.4	41.7	42.7
							6 Months							
10.5	10.8	11.2	11.6	12.0	12.4	13.1	Pelvic Breadth	10.3	10.5	10.8	11.3	11.8	12.4	13.2
42.1	42.7	43.3	43.9	44.8	45.4	45.9	Head Circ.	40.9	41.4	42.0	42.8	43.6	44.5	45.4
40.1	41.0	42.5	48.7	45.0	46.3	47.2	Chest Circ.	39.4	40.6	41.8	43.0	44.2	45.4	46.6
36.4	38.4	59.8	41.4	43.2	45.0	46.0	Abd. Circ.	36.2	37.9	39.5	41.4	43.5	45.0	46.2
							9 Months							
11.0	11.5	11.9	12.3	12.7	13.1	13.7	Pelvic Breadth	11.0	11.3	11.5	12.0	12.5	13.1	13.8
43.8	44.5	45.1	46.0	46.5	47.1	47.8	Head Circ.	42.6	43.2	43.8	44.6	45.4	46.3	47.2
42.0	43.7	44.8	46.0	47.5	48.9	49.9	Chest Circ.	41.7	42.7	44.0	45.4	46.6	47.9	49.2
38.1	40.1	41.7	43.4	45.6	47.6	48.4	Abd. Circ.	38.0	39.9	41.3	43.4	45.7	47.7	49.2
							12 Months							
11.4	11.9	12.4	12.8	13.2	13.7	14.2	Pelvic Breadth	11.4	11.7	12.0	12.4	13.0	13.6	14.4
44.9	45.5	46.5	47.3	47.8	48.4	48.9	Head Circ.	43.6	44.3	45.0	45.8	46.7	47.7	48.4
43.5	45.1	45.3	47.6	49.3	50.7	51.9	Chest Circ.	43.1	44.2	45.6	47.0	48.2	49.5	50.9
39.3	41.1	42.9	44.6	47.0	48.9	50.0	Abd. Circ.	38.7	40.9	42.4	44.5	46.9	40.2	51.1
							15 Months							
11.8	12.4	12.8	13.3	13.7	14.2	14.7	Pelvic Breadth	11.6	12.1	12.4	12.9	13.5	14.1	14.8
45.6	46.3	47.1	48.0	48.5	499.2	49.8	Head Circ.	44.3	44.0	45.6	46.5	47.4	48.4	49.1
44.7	46.1	47.3	48.6	50.1	51.7	52.8	Chest Circ.	44.1	45.1	46.5	47.9	49.2	50.5	51.9
40.0	41.7	43.5	45.1	47.4	49.3	50.5	Abd. Circ.	39.3	41.5	43.0	45.0	47.3	49.8	51.8
							18 Months							
12.1	12.8	13.2	13.7	14.2	14.7	15.2	Pelvic Breadth	11.8	12.4	12.8	13.3	13.9	14.5	
46.2	47.0	47.7	48.7	49.2	49.	50.6	Head Circ.	44.9	45.5	46.2	47.1	48.0	49.0	
45.9	47.0	48.2	49.5	50.9	52.6	53.7	Chest Circ.	45.0	46.0	47.3	48.8	50.2	51.4	
40.6	48.2	44.0	45.5	47.8	49.6	50.9	Abd. Circ.	39.8	42.1	43.6	45.5	47.6	50.3	

Table (9) : (Continued)

Percentiles (boys)								Percentiles (girls)						
3	10	25	50	75	90	97		3	10	25	50	75	90	97
							2 Years							
12.8	13.5	13.9	14.4	15.0	15.5	16.1	Pelvic Breadth	12.5	13.1	13.5	14.1	14.7	15.3	16.4
47.0	48.0	48.2	49.7	50.2	51.0	51.7	Head Circ	45.8	46.4	47.2	48.1	49.1	50.1	50.9
47.4	48.4	49.5	50.8	52.2	53.9	54.9	Chest Circ.	46.3	47.4	48.6	50.1	51.8	53.0	54.2
41.6	43.4	44.8	46.2	48.4	50.2	51.5	Abd. Circ	40.7	42.8	44.4	46.3	48.5	51.4	53.5
							2.5 Years							
13.6	14.2	14.6	15.1	15.7	16.2	16.7	Pelvic Breadth	13.2	13.7	14.2	14.8	15.4	16.1	16.9
47.5	48.5	499.2	50.2	50.9	51.6	52.3	Head Circ.	46.3	47.0	47.8	48.8	49.8	50.8	51.5
48.2	49.3	50.3	51.7	53.2	54.9	55.8	Chest Circ.	47.3	48.4	49.7	51.2	52.8	54.3	55.5
42.0	44.0	45.5	46.7	49.1	50.7	52.0	Abd. Circ.	41.7	43.6	45.2	47.0	49.4	52.6	54.7
							3 Years							
14.2	14.8	15.2	15.8	16.4	16.9	17.4	Pelvic Breadth	13.8	14.3	14.8	15.4	16.1	16.8	17.7
47.9	48.9	49.6	50.4	51.3	51.9	52.7	Head Circ.	46.8	47.5	48.4	49.3	50.3	51.1	52.0
48.9	49.9	51.0	52.4	54.1	55.8	57.0	Chest Circ.	47.9	49.3	50.5	51.9	53.5	55.1	56.7
42.1	44.6	46.0	47.2	49.6	51.1	52.7	Abd. Circ.	42.7	44.5	46.0	47.7	50.2	53.6	55.8
							3.5 Years							
14.7	15.3	15.7	16.3	16.9	17.4	17.9	Pelvic Breadth	14.4	14.9	15.4	16.0	16.7	17.4	18.3
49.6	50.5	51.6	53.1	54.9	56.6	58.0	Chest Circ.	48.5	50.1	51.2	52.5	54.1	55.8	58.1
							4 Years							
15.2	15.8	16.2	16.9	17.5	18.0	18.5	Pelvic Breadth	15.0	15.4	15.9	16.5	17.2	17.9	18.9
50.1	51.1	52.2	53.7	55.5	57.2	58.9	Chest Circ.	49.2	50.7	51.7	53.1	54.7	56.5	59.0
							4.5 Years							
15.7	16.2	16.6	17.3	18.0	18.5	19.1	Pelvic Breadth	15.5	15.9	16.4	17.0	17.7	18.5	19.4
50.7	51.7	52.9	54.4	56.3	58.0	59.3	Chest Circ.	49.8	51.3	52.3	53.7	55.4	57.3	59.6
							5 Years							
16.1	16.7	17.1	17.8	18.5	19.0	19.7	Pelvic Breadth	16.0	16.3	16.8	17.5	18.2	18.9	19.8
51.2	52.3	53.5	55.0	57.0	58.8	60.5	Chest Circ.	50.4	15.7	52.8	54.2	56.0	57.9	60.2

From Studies of Child Healtyh and Development, Department of Maternal and Child Health, Harvar School of Public Health.

* The figures for the several percentiles of each measurement at 5 years differ slightly from those given in Table 2 - 10 for this age because they were obtained from a different population of children.

Table (10) : Percentiles for selected measurements - 5 to 18 years - in centimeters

Percentiles (Boys)						Percentiles (Girls)				
10	25	50	75	90		10	25	50	75	90
					5 Years*					
17.0	17.6	18.3	18.9	19.6	Pelvic Breadth	17.0	17.4	18.0	18.7	19.4
51.6	52.8	54.5	56.2	57.5	Chest Circ.	50.2	51.4	52.9	54.6	56.5
21.0	21.7	22.6	23.6	24.6	Leg Circ.	21.1	21.8	22.8	23.8	24.7
					5 1/2 Years			18.4		
17.4	18.0	18.7	19.4	20.1	Pelvic Breadth	17.4	17.8	53.7	19.1	20.0
52.4	53.6	55.3	57.1	58.5	Chest Circ.	50.9	52.2	23.3	24.3	57.4
21.4	22.2	23.1	24.1	25.2	Leg Circ.	21.5	22.3			25.3
					6 Years			18.8	19.5	
17.7	18.4	19.1	19.8	20.5	Pelvic Breadth	17.7	18.2	54.5	56.3	20.5
53.2	54.4	56.1	57.9	59.5	Chest Circ.	51.5	52.9	23.8	24.8	58.2
21.8	22.6	23.6	24.6	25.7	Leg Circ.	21.9	22.7			25.8
					6 1/2 Years					
18.1	18.8	19.5	20.2	21.0	Pelvic Breadth	18.1	18.6	19.2	20.0	21.1
54.1	55.3	57.0	58.9	60.6	Chest Circ.	52.2	53.7	55.3	57.2	59.2
22.2	23.1	27.1	25.2	26.3	Leg Circ.	22.3	23.2	24.3	25.4	26.4
					7 Years					
18.5	19.2	19.9	20.6	21.4	Pelvic Breadth	18.4	18.9	19.6	20.4	21.6
54.9	56.1	57.8	59.8	61.6	Chest Circ.	52.8	54.4	56.1	58.0	60.1
22.6	23.5	24.6	25.7	26.9	Leg Circ.	22.7	23.7	24.8	25.9	27.0
					7 1/2 Years					
18.9	19.6	20.3	21.0	21.9	Pelvic Breadth	18.8	19.3	20.1	20.9	22.1
55.8	57.1	58.8	61.0	62.9	Chest Circ.	53.5	55.1	57.0	59.0	61.2
23.1	24.1	25.2	26.3	27.6	Leg Circ.	23.1	24.2	25.3	26.4	27.7
					8 Years					
19.2	19.9	20.7	21.4	22.3	Pelvic Breadth	19.1	19.7	20.5	21.3	22.6
56.7	58.0	59.8	62.1	64.1	Chest Circ.	54.2	55.8	57.8	59.9	62.3
23.6	24.6	25.7	26.8	28.2	Leg Circ.	23.5	24.6	25.8	26.9	28.3
					8 1/2 Years					
19.6	20.3	21.1	21.8	22.7	Pelvic Breadth	19.4	20.1	20.9	21.8	23.1
57.6	59.0	60.8	63.3	65.4	Chest Circ.	54.9	56.5	58.7	60.9	63.5
24.1	25.1	26.3	27.4	28.9	Leg Circ.	23.9	25.0	26.3	27.5	28.9
					9 Years					
19.9	20.6	21.4	22.2	23.0	Pelvic Breadth	19.7	20.5	21.3	22.2	23.5
58.4	59.9	61.8	64.4	66.7	Chest Circ.	55.5	57.2	59.6	61.9	64.7
24.5	25.6	26.8	28.0	29.5	Leg Circ.	24.2	25.4	26.8	28.1	29.5

Table (10) : Continued

Percentiles (Boys)						Percentiles (Girls)				
10	25	50	75	90		10	25	50	75	90
					9 1/2 Years*					
20.2	21.0	21.7	22.6	23.5	Pelvic Breadth	20.1	20.9	21.8	22.8	24.1
59.3	60.9	62.9	65.5	68.1	Chest Circ.	56.2	58.0	60.5	63.2	66.1
24.9	26.0	27.3	28.5	30.1	Leg Circ.	24.7	25.9	27.3	28.6	30.2
					10 1/2 Years					
20.4	21.3	22.0	22.9	23.9	Pelvic Breadth	20.5	21.2	22.2	23.3	24.6
60.1	61.8	63.9	66.6	69.4	Chest Circ.	56.9	58.7	61.4	64.4	67.4
25.3	26.4	27.7	29.0	30.7	Leg Circ.	25.1	26.3	27.7	29.1	30.9
					10 1/2 Years					
20.8	21.6	22.3	23.2	24.4	Pelvic Breadth	21.0	21.7	22.9	24.0	25.3
60.9	62.8	64.9	67.7	70.7	Chest Circ.	57.8	59.9	62.8	65.8	69.0
25.7	26.8	28.1	29.5	31.4	Leg Circ.	25.6	26.8	28.3	29.9	31.8
					11 Years					
21.1	21.8	22.6	23.5	24.8	Pelvic Breadth	21.4	22.2	23.5	24.6	26.0
61.7	63.7	65.9	68.8	71.9	Chest Circ.	58.6	61.1	64.2	67.2	70.5
26.0	27.1	28.5	30.3	32.0	Leg Circ.	26.0	27.3	28.9	30.6	32.6
					11 1/2 Years					
21.5	22.2	23.1	24.0	25.3	Pelvic Breadth	21.9	22.8	24.2	25.4	26.8
62.5	64.6	66.9	69.9	73.1	Chest Circ.	59.6	62.5	65.5	68.5	72.2
26.4	27.6	29.0	30.6	32.8	Leg Circ.	26.6	27.9	29.5	31.2	33.2
					12 Years					
21.9	22.6	23.5	24.5	25.8	Pelvic Breadth	22.4	23.4	24.9	26.2	27.6
63.3	65.5	67.8	70.9	74.2	Chest Circ.	60.6	63.8	66.7	69.7	73.8
26.8	28.0	29.5	31.2	33.5	Leg Circ.	27.1	28.5	30.1	31.8	33.8
					12 1/2 Years					
22.3	23.1	24.1	25.1	26.5	Pelvic Breadth	23.0	24.0	25.5	26.8	28.3
64.2	66.5	69.1	72.4	75.8	Chest Circ.	61.8	64.9	67.7	70.9	75.3
27.3	28.6	30.1	32.0	34.2	Leg Circ.	27.7	29.1	30.7	32.4	34.3
					13 Years					
22.7	23.6	24.6	25.6	27.2	Pelvic Breadth	23.6	24.6	26.0	27.4	29.0
65.0	67.4	70.3	73.8	77.4	Chest Circ.	62.9	65.9	68.6	72.0	76.7
27.8	29.2	30.8	32.7	34.8	Leg Circ.	28.2	29.7	31.2	32.9	34.8
					13 1/2 Years					
23.2	24.1	25.2	26.4	27.8	Pelvic Breadth	24.2	25.2	26.5	27.8	29.5
66.3	68.8	72.4	75.8	79.4	Chest Circ.	63.8	66.6	69.3	72.9	77.7
28.5	29.9	31.6	33.4	35.3	Leg Circ.	28.7	30.2	31.6	33.4	35.1

Table (10) : Continued

Percentiles (Boys)						Percentiles (Girls)				
10	25	50	75	90		10	25	50	75	90
					14 Years*					
23.6	24.6	25.8	27.1	28.3	Pelvic Breadth	24.8	25.8	26.9	28.1	29.9
67.6	70.2	74.5	77.8	81.4	Chest Circ.	64.6	67.2	69.9	73.7	78.6
29.1	30.6	32.3	34.1	35.8	Leg Circ.	29.2	30.0	32.0	33.8	35.4
					14 1/2 Years					
24.1	25.1	26.3	27.5	28.7	Pelvic Breadth	25.2	26.2	27.2	28.4	30.9
69.4	72.3	76.3	79.6	83.1	Chest Circ.	65.1	67.7	70.4	74.2	79.2
29.8	31.3	32.9	34.6	36.2	Leg Circ.	29.6	30.9	32.3	34.1	35.7
					15 Years					
24.6	25.6	26.7	27.9	29.1	Pelvic Breadth	25.6	26.5	27.5	28.7	30.6
71.1	74.4	78.0	81.3	84.8	Chest Circ.	65.5	68.1	70.9	74.7	79.8
30.4	31.9	33.4	35.1	36.6	Leg Circ.	29.9	31.1	32.6	34.3	35.9
					15 1/2 Years					
25.1	26.0	27.1	28.2	29.4	Pelvic Breadth	25.9	26.7	27.8	29.0	30.8
72.8	75.8	79.4	82.9	86.3	Chest Circ.	65.8	68.4	71.3	75.1	80.2
30.9	32.3	33.8	35.5	37.0	Leg Circ.	30.1	31.4	32.9	34.5	36.1
					16 Years					
25.6	26.4	24.4	28.4	29.6	Pelvic Breadth	26.1	26.9	28.0	29.2	31.0
74.4	77.2	80.7	84.5	87.8	Chest Circ.	66.1	68.7	71.6	75.4	80.5
31.3	32.7	34.2	35.8	37.3	Leg Circ.	30.3	31.6	33.1	34.6	36.3
					16 1/2 Years					
25.9	26.7	27.6	28.6	29.8	Pelvic Breadth	26.2	27.0	28.2	29.3	31.1
75.4	78.1	81.6	85.4	88.8	Chest Circ.	66.3	69.0	71.9	75.7	80.7
31.5	32.9	34.4	36.1	37.6	Leg Circ.	30.5	31.8	33.3	34.8	36.5
					17 Years					
26.1	26.9	27.8	28.7	29.9	Pelvic Breadth	26.3	27.1	28.3	29.4	31.2
76.4	78.9	82.5	86.2	89.7	Chest Circ.	66.4	69.2	72.1	75.9	80.9
31.7	33.1	34.6	36.3	37.8	Leg Circ.	30.6	31.9	33.4	34.9	36.6
					17 1/2 Years					
26.3	27.0	27.9	28.8	30.0	Pelvic Breadth	26.4	27.2	28.4	29.5	31.3
77.0	79.4	83.0	86.7	90.2	Chest Circ.	66.5	69.3	72.2	76.0	81.0
31.8	33.3	34.8	36.5	38.0	Leg Circ.	30.7	32.0	33.5	35.0	36.7
					18 Years					
26.3	27.1	28.0	28.9	30.1	Pelvic Breadth	26.4	27.2	28.4	29.5	31.3
77.5	79.8	83.4	87.1	90.7	Chest Circ.	66.6	69.4	72.3	76.1	81.1
31.9	33.4	34.9	36.6	38.1	Leg Circ.	30.8	32.1	33.6	35.1	36.8

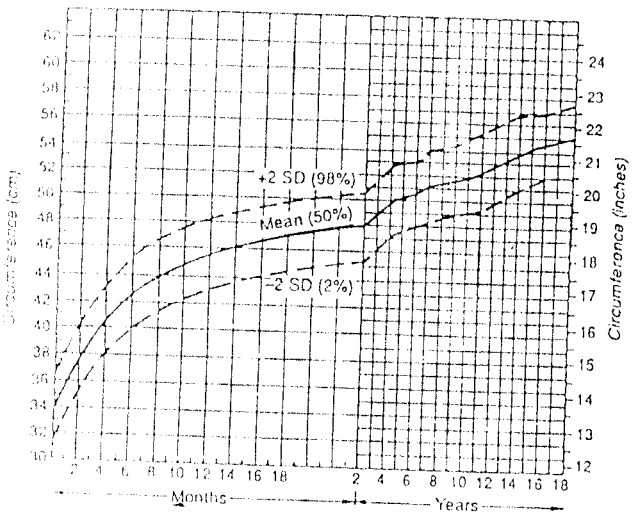

Fig. (9) : Head circumference of girls. (Modified and reprouced, with permission, from Nelinaus *G; Peiatrics* 1968; 41 : 106)

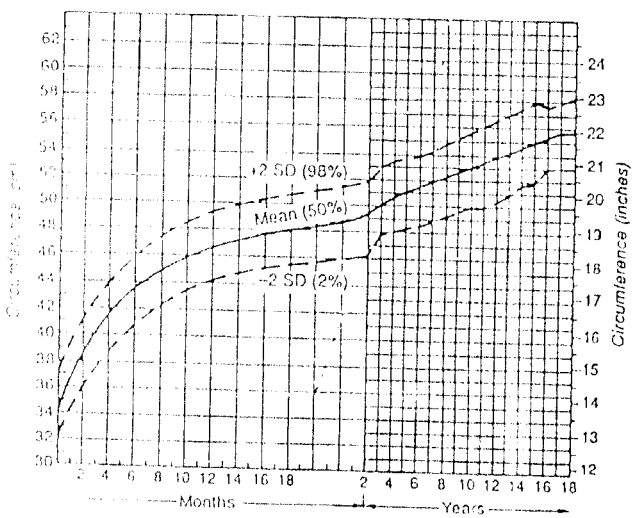

Fig. (10) : Head circumference of boys.

umbilicus when the infant is recumbent ***(Piaget and Inyhelder, 1969).***

Leg Circumference. The maximal girth of the calf is measured with the child standing with his feet several inches apart and his weight equally distributed through both legs ***(Mahler et al., 1975).***

Pelvic Breadth. Pelvic, biiliac or bicristal breadth is the distance between the later almost points of the iliac crest of the pelvis, including the overlying soft tissues. Measurement is made on the recumbent infant or young child by spreading or obstetrical calipers (Table 9). For children 5 years and over, were made with abroad sliding caliper applied over the crest of the ilia, the child standing and facing the measurer (Table 10). The points of spreading calipers should not be pressed deeply into the soft tissue, whereas with sliding capliers the maiximal pressure without causing pain should be applied. If these precautions are followed, measurements taken by the 2 instruments will not differ appreciably; otherwise, measurements by spreading calipers will be somewhat smaller in obese children than those made by sliding calipers ***(Dixon and Stein, 1987).***

Psychological Assessments

The assessment of the functional status of the infant or child is an essential part of each examination, but is all too often uncritical. Only with some knowledge of developmental standards can the physician caring for children be adequately sensitive to deviations which indicate slight or early impairment of development. Moreover, only if he can quickly and confidently compare his observations with the normal developmental schedule will he be able to handle the questions of parents or make appropriate suggestions for further study *(Smith, 1977).*

History

A. Medical History : The medical history should focus on aspects pregnancy, labour, and delivery that are likely to produce damage to the child's central nervous system (eg, use of drugs or X-rays during pregnancy; neonatal infections, asphyxia, and elevated bilirubin levels). Later evidence of central nervous system insults or injury, failure to thrive, chronic illnesses, hospitalizations, or abuse may also contribute significantly to a child's performance at school age. Neonatal records

are often an important source of information, since they may reveal information forgotten by or unknown to parents. Recent studies have also focused on ways of combining psychosocial information about families from birth certificates to assess the risk for later problems in development *(Bakwin and Bakwin, 1963).*

B. Developmental History : The developmental history should give information about the age at which various milestones were passed, especially those pertaining to speech and language. Inability to use meaningful words other than "dada, "mama," "bye-bye," and "hello" by 18 months and inability to speak in short phrases by 24 months have been reported in association with specific learning disability as well as general slow learning and mental retardation. Development of motor skills is also important, particularly in assessing mental retardation, but deviance or delay in motor development may also be present in other conditions such as cerebral palsy and neuromuscular disorders. Information about sleep patterns, problems of temperament such as excessive crying or hyperactivity, and general problems may also be helpful *(Tureeki, S. Tonner L., 1985).*

C. Family History: Specific information regarding central nervous system disorders, mental retardation, epilepsy, or evidence of school problems or specific

learning disabilities in other family members should be included. Details of the mother's pregnancy history, including stillbirths, deaths, and other problems, may also be helpful.

D. Educational and Learning History: In preschool children, considerable information can be obtained from a description of something child has learned in an informal setting. If the child has been placed in a formal preschool setting, information should be obtained regarding the type of preschool and the child's relationships with other children and with teachers. Teachers can often give an excellent description of the child's performance and behavior in the classroom environment, and such assessments, even in the preschool period, are often as good as tests in predicting later problems.

Once a child has reached school age, the educational history should include details of grade placement, special educational evaluations and placement, repetition of grades, and other details of academic performance and participation in special programs. In the absence of a psychologist or educational specialist to provide this information, direct contact with school personnel is imperative for the primary care physician. Telephone conversations with the school nurse,teacher,social worker, or other professionals are very helpful in

obtaining a clear picture of the child's performance and behavior in the school environment. Written reports from teachers using questionnaires that systematically address the common types of learning problems can also be of great assistance.

E. Psychosocial History: Family problems and parental characteristics often interfere with development of both cognitive and social competence and foster deviant behavior in the child. Children of hostile, rejecting, highly authoritarian parents tend to be the most severely affected; these; children often show advanced competence at 1 year of age but a progressive decline in competence beginning around 4 years of age. Children of nurturing parents with highly authoritarian parenting practices often do better; however, children of nurturing parents who are firm and verbal in providing guidance and setting standards without being rigidly authoritarian show advanced competence that increases with age. Parents who provide little nurturance or sense of belonging, who are too lax or too harsh in punishment, or who fail to supervise their children tend to have children who show early evidence of aggressive behavior problems that persist into adolescence and adulthood ***(Offer. D., 1969).***

Because developmental and behavior problems in the child are often provoked by and associated with problems

within the family or seem to be associated with a lack of family support for developing new skills, a good psychosocial history is an essential part of any developmental evaluation. Ideally, this should include assessment of the family's ability to promote cognitive and social development, which includes, as a minimum, information regarding the parents' linguistic and cultural background, quality of verbal interaction, disciplinary peactices (use of positive reinforcement to shape behavior, reliance on physical punishment or limited use of reasoning or verbal explanations in discipline), ability to set standards, neglect, reliance on parenting practices that interfere with or inhibit development, family instability, marital discord, a hostile attitude toward the child, limitations in cognitive and social competence, depression, signs of maladjustment (eg, alcoholism, chronic unemployment, criminal or psychiatric problems), and general stress in the parents and chaos in the family that may contribute to and intensify developmental problems in the child ***(Emde RN, Robinson J., 1979).***

In examining only the child, it is often difficult to distinguish behavior disorders associated with family problems from developmental disorders due to immaturity or alteration development of the neurologic system. This difficulty has resulted in diagnosis by exclusion---ie,

psychosocial assessment is used to determine whether there are social or emotional factors in the child's environment that can account for the observed learning or behavior problems. This is a practical approach in middle-class families, since it is often possible to ascertain that the family is reasonably stable and able to provide adequate support and stimulation to the child, Diagnosis by exclusion is, however, a very unsatisfactory approach for dealing with children from lower socioeconomic families, ie, the majority of children with behavior problems.The need for better assessment of these children is particularly acute, since they often have delays in both social cognitive development and combinations of developmental delay and behavior problems. Ninety percent of the children who later show mental or emotional disorders are normal at birth and appear to be casualties of inadequate or pathologic environments ***(Barker R., 1963) (Brker P., 1986).***

At present, the most commonly used approach to family assessment is a global, clinical history. Some of this information is usually included in the history obtained by the primary care physician. In most instances, however, a social worker will provide the Most thorough and complete analysis. In preschool children, it is often helpful to supplement the usual clinical social history

with the HOME *(home observation for measurement of the environment)* interview assessment of Bradley and Caldwell. This is the most thoroughly studied approach to the systematic evaluation of the growth-promoting aspects of the child's environment. This interview, which requires a home visit, is used to identify economically development in their children. It may be performed by any trained person but is usually done by a social worker or nurse. A shorter, questionnaire version of the HOME interview, the Home Screening Questionnaire (HSQ), provides most of the information obtained from the longer interview version and can be administered and scored by the pediatrician during a clinic or office visit. Although it has not yet been studied widely enough to determine its clinical usefulness, the HSQ appears to be a promising tool for use by the primary care physician. Neither scale is expected to be useful in evaluating children from middle or upper socioeconomic families ***(Barrett et al., 1967).***

Physical & Neurologic

Examination

It is essential that a thorough physical and neurologic examination be performed. A number of children will demonstrate neurologic "soft signs," eg, clumsiness, right-left confusion, disordered temporal orientation, overflow phenomena, choreiform movements, and finger

agnosia. Although "soft signs" are commonly associated with school learning and behavior problems, the significance of these signs is controversial because they are also found in children who have no other problems and because most appear to represent delay in maturation rather than dysfunction. PANESS *(physical and neuralgic examination for soft signs)*, a standardized neurologic examination, has been studied and shows promising results for systematic evaluation of "soft signs".

In addition, recent studies linking minor physical anomalies with behavior disorders in childhood have prompted physicians to examine for the presence of dysmorphic features such as abnormal palmar creases, syndactyly, unruly hair, malformed ears, skin tags, and facial abnormalities. While these features are commonly seen in children with mental retardation, the implications of their presence in nonretarded children are not fully understood ***(Smith, 1977)***.

Sensory Function

All children in whom developmental delay or mental retardation is suspected should be examined for visual and auditory problems. In infants and young children, sensory deficits may be mistaken for retardation. Retarded children often have sensory deficits in addition to their retardation, and this increases the complexity of their

problem. In most nonretarded, school-aged children, vision and hearing can be satisfactorily evaluated by the usual screening methods and referral made to a specialist screening methods and referral made to a specialist for further evaluation of children with abnormal screening results *(Kagan J., 1982).*

A variety of vision problems have been proposed as causes of reading problems, most without substantial research support. Learning to read can be accomplished quite satisfactorily with limited visual acuity. Although it is important that visual defects be corrected to improve the child's overall functioning, it is denerally that learning problems are seldom liked to refractive errors. Difficulty with convergence at near point, however, may interfere significantly with the process of reading and should receive careful evaluation *(Campbell S.B., 1986).*

Hearing loss has a significant impact on language development and may be associated with severe learning and behavior problems. Intermittent hearing loss, such as that due to otitis media, has been implicated in learning disabilities. In the past, deaf children have often been mistakenly labeled retarded. Losses in the high-frequency range may be associated with problems in disciminating speech sounds necessary for school learning. Others may have problems differentiating speech sounds despite

normal hearing *(Becker, 1963) (Hertz M.E., 1988).*

Emotional & Social Behavior

Although some information can be obtained directly from the child through interviews, play, and projective testing, typically one must rely on interviews with parents and reports from school personnel to obtain a picture of the child's social competence. Much of this information is obtained by social workers, psychologists, or psychiatrists. In evaluating reports of problem behavior at home and at school, it is helpful to assess the degree of deviance by comparing an individual child's behavior with children in general. Large studies of normal children indicate that most children show a few signs of deviant dehavior. The truly deviant child, however, usually demonstrates this in a variety of ways. It is especially important to seek information about positive indicators of later maladjustment. Three of the most important positive attributes are school attendance (irrespective of performance), positive peer relations, and nondelinquency *(Thomas A.A., Chess S., 1977).*

General adaptation and development of self-help are often in developmental assessment of preeschool children. Beginning around age 4 years (when abstract reasoning becomes the dominant factor in measures of cognitive development), it becomes increasingly important to

include some assessment of general adaptation in the differential diagnosis of mental retardation. children from minortity caultures who perform poorly on IQ tests often appear less retarded when general adaptation is evaluated ***(Belmont and Birch, 1960).***

Family & Social Resources

The type, Extent, and cost of educational and counseling services to the child and family, the family's ability to support and community resources should be assessed early in the valuation. These factors often limit or modify the treatment plan developed for a child. Sixty percent of families presenting children for evaluation of learning problems have clear-cut social and emotional problems that need to be assessed and addressed as an integral part of treatment plannimg for the child. Social services are usually the principal sources of information in this area, and much of this information will be derived from the psychosocial history. In addition,however, the primary care physician should become familiar with resouces in the community ***(Belmont and Birch, 1966).***

Intelligence

Measures of intelligence attempt to describe a child's general cognitive competence in relation to other children of the same age. The tests provide an increasingly

difficult set of problems, and questions tend to tap general Knowledge, reasoning, judgment, and organization of analytic skills that are expected to develop in the course of experiences encountered by most children in the process of growing up. Where children have grossly diffrent experiences from those in the standard population, their scores may be expected to vary upward or downward. Originally, the IQ score obtained from such tests represented at a given age. This was derived by the formula (mental age ÷ chronologic age) x 100. Scores on most modern tests can still be reported in terms of MA/CA, but the most important tests yield IQ scores that represent a child's relative distance from the average child in standard score units ***(Yogman et al., 1988).***

In the preschool period, the principal diagnostic tests in general use are the Bayley Scales of Infant Development (for children under 30 months of age), the Stanford-Binet Intelligence Scale,the McCarthy Scales of Children's Abilities (for children 3 years of age and older).and the kaufman Assessment Battery for Children (K-ABC). These are all individually administered tests, given by trained personnel. The screening tests used most commonly in the preschool period are the Revised Denvised Developmental Screening Test (Frankenberg); the Revised Developmental Screening Inventory and,

among older preschool children, the Early Screening Inventory (Meisels and Wiske) ***(Frankenbetg et al., 1981).***

In recent years, several new assessment methods have been developed to capitalize on the caretaker's knowledge of the infant and preschool child. Procedures such as the Kent Infant Development Scale (KIDS) for children under 12 months of age and the Minnesota Child Development Inventory for age children over 12 months of age use the caretaker's report to assess the child's general development, including cognitive, gross motor, fine 4 motor, expressive language, self-help, and personal/social development. These procedures have several adantages; They provide for the inclusion of a large number of items at each age, are based on behavior readily observable at home, do not require extensive training for their administration, and can be used to plan and monitor appropriate intervention at home. Disadvantages include potential inaccuracy in the caretaker's reply and bias in reporting ***(Bender and Silver, 1948).***

The Wechsler scales (Wechsler Adult Intelligence Scale [WAIS], Wechsler scales (Wechsler Adult intelligence Scale for Children, Revised [WISC-R], and Wechsler Preschool and Primary Scale of Intelligence [WPPSI] and the Stanford-Binet intelligence Scale are the

most widely used individual intelligence tests for schoolage and older children. The Kaufman Assessment Battery for Children (K-ABC) is a relatively new test that is also gaining wide acceptance. The Stanford-Binet test is a highly verbal test with nonverbal items intermingled; results are reported as a single IQ score. The Wechsler scalcs are subdivded into 6 verbal and and 6 nonverbal tests so that a verbal IQ and performance IQ can both be obtained as well as an IQ based on the full test (full-scale IQ). The Kaufman Battery is designed to assess differences in simultaneous and sequential processind of information as well as differences between aptitude and achievement. It is commonly thought that intelligence tests can reveal the potential for higher functioning, especially when scatter (the pattern of highand low scores) is examined carefully, and both the K-ABC and the Wechsler scales lend the mselves particularly well to this task.

The standardized, individually administered tests of intelligence discussed above require extensive training for proper administration and are usually administered by trained psychologists. The Slosson Intelligence test, however, is an abbreviated version of the stanford-Binet test and is designed for use by nonprofessionals, including office assistants and primary care physicians. It is

probably the most suitable screening test for estimating intelligence in children over 41/2 years of age.

A number of briefer screening tests, such as the Peabody Picture Vocabuiary Test and the Quick Test are also suitable for use in office settings. These measure vocabulary has the best correlation with overall estimates of intelligence. A variety of other shortscreening tools are available, some of which rely on parent reporting or a combination of parent reportingand observation, but these often suffer from limited information regarding standardization data ***(Carroll, 1961).***

Achievement

Achievement usually refers to performance in specific school-related areas where a child has received instruction. In the preschool period, achievement is seldom distinguished from general development. By the time the child enters kindergarten, however, a variety of procedures are available for assessing readiness. In a child with a mental age of 6 years, 2 of the best predictors for readiness to enter the first grade are the ability to engage in sustained taskoriented behavior and the knowledge of letter names and sounds. A number of procedures are available to assess school readiness, and in recent years, testing programs have been specifically designed for early identification of children with learning disabilities.

Although resulty of these programs appear to be about as accurate as teacher assessments of kindergarten performance, many feel that the programs represent an important advance. Most of the early identification testing programs are designed for use in schools or by psychologists. Several procedures are also available for use by primary care physicians in screening for school readiness at the time of well-child visits in the 4 1/2- to 5 1/2- year age range. These include the developmental screening tests described previouslyand the preschool portions of the Wide Range Achievement Test (WRAT); see below).

For school-age children, scores on a variety of achievement tests are often available through routine classroom testing done at school, and results of such testing should be included in the educational history. Where low scores are obtained on group testing of achievement, individual testing should be performed before accepting the results as representative of the child's ability. Many school systems with special education services can administer individual tests of achievement and give the test scores to the primary care physician. The Woodcok-Johnson Psycn-Educational Battery is an individually administered battery of cognitive, achievement, and interest tests often used by

educators to assess achievement and the discrepancy between aptitude and achievement ***(Cowan et al., 1960).***

Unfortunately, many children who are referred to a pediatrician for evaluation have no current achievement test scores in their school records. This is particularly true for children referred because of behaviour problems. When an educational specialist or psychologist is unavailble to provide this information, the primary care physician may wish to make his or her own assessment through use of a screening test such as WRAT/WRAT is rapid screening test for assessing achievement in reading, spelling, and arithmatic. It is suitable for testing children from kindergarten through college, and norms (availble from the Natione; Health Survey, as well as the publisher) are based on results in children from a representative sample of ethnic and economic groups throughout the country. The reading test covers only the accuracy of word reading and tends to yield higher scores than more thorough and comprehensive procedures do. However, WRAT is a simple and easy test to administer, and scores are reported in grade equivatents, percentiles, and standard scores that are comparable to many IQ test scores. In addition, the copying portions of the spelling test can be used to attain a rapid assessment of visual-motor coordnation ***(Cruickshank, 1966).***

Adaptive Behavior

Assessment of adaptive behavior is seldom addressed directly in children unless a child is suspected of being mentally retarded. Because poor performance on a cognitive test may result from many factors, it is essential that the accuracy of low scores be confirmed by assessment of functional level in everyday living. Scales such as the Vineland Adaptive Behavior Scales and the scales of Independent Behavior are questionnaire/structured interview methods for assessing the child's developmental level in areas such as social interaction, communication, personal living, and community living ***(Davis, 1961).***

perceptual-Motor function

In the early school years, a number of children with delays in copying and drawing skils will also demonstrate problems in learning to read. These problems have been variously termed visualperceptual and visual-motor problems, and their presence in children beyond 7-9 years of has often been though to indicate the existence of central nervous system dysfunction, although this inference is controversial. There is a relationship between visual-perceptual problems and reading problems in the early school years; s children get older, however, reading achievement becomes more and more related to intelligence, even when the perceptual problems persist.

Furthermore, perceptual problems are common among children from families in lower socioeconomic groups and even among children whose only history is confinement to bed for more than 2 months during the preschool years.

Because performance on tests of copying skills is highly correlated with intelligence, such teste are most useful when there is a discrepancy between the developmental level demonstrated on visualperceptual tests and general intelligence. Methods for assessing degree of this discrepancy are not as well developed as for assessing the discrepancy between IQ and achievement. At a practical level, however, these problems may be severe enough to interfere with learning the skills of printing and writing. In this latter case, it is usually more helpful to analyze the child,s writing, but several visualperceptual tests, such as the Beery Test of Visual-Motor Integration, are commonly used to examine copying skilld per se *(David, 1969).*

Speech & Language

Speech and language delays are common in mentally retarded children but may also occur in children with average or above-average intelligence. A number of children who appear to have specific learning problems will, on closer evaluation, show evidence of delay in language development or an articulation problem (or

both). These problems can limit academic achievement in Areas that depend on verbal skills (eg, reading). Speech and language delays in preschool children and methods for evaluating them have been discussed previously.

Most children should reach the adult proficiency level in language by 7-8 years of age, at which time evaluation of language skills typically becomes merged with of verbal intelligence. Scores on tests such as the Wechsler scale, the Stanford-Binet test, or the Slosson test will reflect language skills as well as intelligence. Specialized tests for assessing components of recetive and expressive language and language-processing skills are also available *(Cross, 1947).*

Motivation

Clinical assessment of motivation has received little attention despite mounting evidence that motivation is a key factor in determining how a child will use whatever time and help provided for learning new skill. Studies of achievement motivation have suggested that 2 main motivational types can be identified, ie, those who are challenged by moderately difficult tasks and respond to success and those who are primarily motivated to avoid failure and will only attempt very easy tasks, where failure is unlikely, or very difficult ones, where failure carries no stigma. One of the major shifts that occurs in

the age range of 5-7 years is from motivational dependence on social and external rewards to internal motivation for mastery of skills. Some children make this shift poorly or not at and will fail to learn in the usual academic climate, which emphasizes mastery and competition. Often these are the children who seem primarily motivated to avoid failure and who need liberal support from external sources (praise, concrete incentives) just for trying. While motivational immaturity is seldom the principal problem, it is often a major determinant of how well a child will respond or progress in an educational program ***(Brown, 1960).***

Evaluating the discrepancy between intelligence & achievement

The differential diagnosis of competence problems necessitates an evaluation of the significance of the discrepancy between expected achievement based on measures of general competence (intelligence) and actual achievement in specific of academic performace. The following material presents 2 methods of calculating the degree of discrepancy between IQ and achievement, based on grade edquivalents and standard scores.

The United States Office of Education defines the discrepancy between achievement and IQ as significant when achievement is below 50% of expected grade level.

The lowest level of achievement commensurate with age and intelligence is calculated by the following formula:

Age [(IQ/300) + 0.17]- 2.5 = Lowest grade equivalent score wich is commensurate with age and intelligence

For an 8-year-old child with an IQ of 100, this formula indicates that a grade level of 1.5 is the lowest score that would be commensurate with age and in telligence. If this child's achievement tset scores are below grade level 1.5, than there is a significant discrepancy. If they are at grade level 1.5 or higher, the discrepancy is not significant.

The following formula utilizes the direct correlation between intelligece test and achievement test scores to calculate the smallest difference between IQ and achievement test score needed to represent a significant discrepancy (D) :

$D = 1.96\ SD\sqrt{1 - r^2}$ where SD = Standard deviation of scores on achievement test

r = correlation between IQ and achievement test scores

This formula can be used to compare an IQ obtained from a test such as the Slosson test with a standard score obtained from the WRAT. The Slosson test manual

provides the information that the correlation coefficient with the WRAT is 0.72, and the WRAT manual provides the information that the standard deviation of scores on the WRAT is 15. According to this calculation, a difference of 20 points between IQ and achievement test score is needed before a difference between these 2 tests is significant at the P = 0.05 level of confidence or better.

Both of these approaches call attention to the fact that some difference between expected and actual achievement represents normal variation, while a significant discrepancy is defined in terms of an unusual degeree of difference. Individual school districts are free to adopt their own criteria in determining the extent of this discrepancy that must exist for a child to qualify for special services ***(Briggs, 1963).***

Temperamental traits & reactions to developmental crises

During the ages of 5-7 years and 10-13 years, major developmental changes occur in most children. In the 5- to 7- year period, children enter school, begin to develop operational thought, and shift from associattive thinking to use of verbal mediation activity in learning and thinking. The 10- to 13- year period heralds the onset of puberty, entrance into junior high school, and development of formal operational thought. Infections in

the growth curve for intellignce occur during both periods, and there is a dramatic increase in a cluster of behavior problems that appear to be phase-specific reactions to the developmental changes occurring during these periods. These problems include restless sleep, disturbing dreams, physical timidity, irritabilty, overdependence, and jealousy. Emotional turbulence during one of these periods may or may not be associated with turbulence during the other period, and its occurrence in either does not seem to be indicative of serious long-term problems.

Often there is overlap between these phasespecific reactions and temperamental traits, which include characteristics such as shyness, oversensitiveness, and reserve. Both phase specific reactions and temperamental traits may be a source of conflict between parents and children, but they do not in themselves indicate the presence of serious emotional disturbance. They usually resolve with support, and the child does not need treatment ***(Boles, 1956).***

Children's reactions to illness, hospitalization, and surgery

Introduction

In considering disease states, one must also consider the state of health, now recognized as more than the absence of disease. The modern unitary view, as articulated especially by Engel (1960), regards health and disease as phases of life. Health represents the phase of successful adaptation and, in children, of growth and development. Disease represents the phase of failure in adaptation or of breakdown in the attempt of the human organism to maintain an adaptive equilibrium or dynamic steady state; during this phase, disturbances or failures may occur in the growth, development, or adjustment of the organism as a whole or of any of its systems *(Adams, M.S., 1976)*.

Physical, Psychological, and Social Factors

Stressful stimuli of a physical, psychological, or social nature may impinge on the child to bring about a derangement of his adaptive equilibrium and the appearance of illness or a disease state. However, stress is relative, not absolute. The child's genic endowment, his

constitutional characteristics, his developmental capacities, and the nature of his past experience, including experience with disease, all contribute to his adaptive capacity . This capacity, the noxious stimuli, and the current circumstances, including the family's response, determine the degree of stressful significance and the adaptive outcome.

In this unitary view the organism can be said to be made up of three basic levels of organization physiological, psychological, and social. they constitute open systems that communicate, in complex feedback operations, through the neuroendocrine system and its interrelationships with the brain and mental apparatus, as well as the various other organs systems. Stressful stimuli of a physical, psychological, or social nature, impinging originally on the corresponding level of organization, may produce alterations in function that bring about feedback reverberations at the other levels. physiological and psychological defenses, as well as behavioral or social adaptive or coping devices, may be called on to help maintain the adaptive equilibrium. Although the mechanisms are unclear, stressful stimuli of a psychological or social nature, such as competitive pressure or the loss or threat of loss of a parental figure, seem to be involved in the precipitation of various

illnesses, ranging from the common cold and streptococcal infections to the more chronic conditions of diabetes and asthma ***(Azarnolf, P. and Flagel, S.A., 1974).***

The child's adaptive capacity at the time of the stressful experience and the nature. duration, and intensity of the stimulus may bring about a new and more successful adaptive equilibrium representing a state of physiological or psychological immunity. Conversely. temporary decompensation; chronic restriction in growth. development, or function; or serious adaptive breakdown may ensue. Many of the symptoms or signs associated with any disease state, from fever to regressive behavior. may actually be the result of attempts by the organism to maintain adaptation or to achieve compensation, rather than the specific results of the stressful stimuli ***(Bakwin, H., 1951).***

In designating disease states as predominantly physical or somatic or as principally psychological in character, the experienced clinician takes into account the level of organization most heavily involved in the decompensation, restriction of function, or adaptive breakdown. He or she recognizes that multiple causative forces of a physical, psychological, and social nature are involved in a predigesting, contributing, precipitating, and perpetuating fashion in any disease. Rather than adopting

an either or approach, the clinician weighs both the somatic and the psychological components in the clinical picture and arrives at a treatment plan that takes these components and the social factors into consideration ***(Charney, B., 1972).***

The social field of illness includes both the effect of interpersonal forces on the child's adaptive equilibrium, with reverberations at the psychological and physiological levels, and the impact of predominantly physical illness in a child on the most important social unit the family. A child's illness may bring about a family crisis. Families with healthy adaptive patterns may respond with behavior leading to a new and different family adaptive equilibrium, representing a type of family development. In less well - balanced families, parental patterns of handling the ill child may be significantly altered because of more than usual anxiety or guilt, causing further changes in the behavior of the child or occasioning rivalry or other responses in siblings. In seriously disturbed families, the child who falls ill may be made a scapegoat for family tensions, may be treated unrealistically as a chronic invalid, or may be handled in other ways that reflect the unconscious tendency of the parents or other family members to respond to his illness in terms of their own needs, rather than his ***(Bowlby, J. 1951).***

Families from differing socioeconomic and ethnic back grounds may respond quite differently to the illness of a child and may react positively or negatively to the need for dependence on medical personnel or other helping agencies. Certain active children, with inadequates parental supervision or with conflicts over the acceptance of parental supervision or controls, may be involved in frequent accidents or other self injurious situations. Maternal deprivation may lead to failure to thrive or to delayed development or both, as well as to disturbances in behavior. Parental problems in impulse control may produce the battered child syndrome or drug intoxication of the child from repeated use of sedatives.

The importance of all these considerations renders it vital that the clinician accept the family, rather than the child, as the essential unit for the study and treatment of disease. The illness of a child with acute or chronic disease both affects and is affected by the interpersonal family equilibrium in the particular community, society, and culture.

Some difficulty is encountered in separating the reaction to the illness or injury, representing the effects of the stressful stimuli at the physiological level, from the reaction to hospitalization or other treatment measures, representing stressful stimuli at the psychological or social level of organization.

The reactions discussed here may occur with any illness. They are principally situational or reactive disorders, although continuing developmental deviations, structured neurotic disorders, chronic personality disorders, or even psychotic disorders may be touched off or exacerbated by such stresses in biologically or experientially predisposed children. Although these are largely nonspecific responses related to the way in which the environment parental. family, social, and physical helps the child deal with the physical consequences and the symbolic meaning of the illness or injury, there is a generic type of human organismic response to catastrophic illness or injury, drawing on adaptive mechanisms, that involves the phases of impact, recoil, and restitution. Such phasic responses may be seen most clearly in children of school age and beyond; preschool children tend to show similar but less clear-cut patterns. The phase of impact involves initial realistic fears, followed by regression denial, and the use of primitive fantasy. The phase of recoil includes a lessening of regression and denial and the appearance of mourning for the loss of the self, a necessary step if handicaps or other limitations result. In some instances, depression may occur, warded off for a time by eating disturbances or hostile, demanding behavior. The phase of restitution permits beginning adaptation to and attempts at mastery of the situation

during which individual patterns, related to premorbid personality trends, emerge ***(Green, M. 1965).***

The reactions of parents to serious or disabling illnesses in their children tend to parallel the phasic responses in children. Initial realistic fear is often followed, as ***Richmond (1958)*** indicated, by : (1) a phase of denial and disbelief, persisting for weeks or, at times, months; (2) fear and frustration, associated with mourning for the loss of the child that was, as well as depression, guilt, and self-recrimination, with intensified marital strife and even divorce occurring in some families as the parents attempt to deal with such feelings by blaming each other, the physician, or others; (3) rational inquiry and planning, involving the need to live with some uncertainty.

Whether a child's illness or injury produces a deleterious effect on his adaptation or the family equilibrium depends on (1) the developmental level of the child; (2) the child's previous adaptive capacity; (3) the prior nature of the parent-child relationship; (4) the existing family equilibrium; (5) the nature of the illness or injury, including the organ system affected, the degree of prostration or pain, the type of treatment or home care, and any residual defect or handicap; and (6) the meaning of the illness to the child and his family in terms of

immediately antecedent events and their actual or fantasied connection, the previous experience with such illness, the effect on the child's social, academic, and athletic capacities, and the reverberations on siblings ***(Green, M and Solnit, A.J., 1964).***

Acute illness or injury

The immediate response acute illness or injury may vary somewhat according to the organ systems affected and the corresponding reverberations at the psychological or social level. Thus, the local and systemic effects of acute rheumatic fever or a esophageal stricture may have a more severe temporary impact on the psychological and social functioning of the child and produce greater anxiety in the parents than a simple green - stick fracture. Certain broad patterns of response are characteristic of children in general, with some differences related to individual variations and developmental level ***(Engel, G.L., 1960).***

DIRECT EFFECTS

Malaise, discomfort, or pain may produce listlessness, prostration, disturbances in sleep and appetite, and irritability in children, much as in adults. However, restlessness is more common in children, and hyperactivity with mild illness is frequently seen, particularly in preschool children. Anorexia and refusal of food may be

marked in young children; well meaning but anxious parents may urge food, and the child's negativistic responses often lead to feeding problems that persist long after the illness subsides. Difficulties in falling asleep, nightmares, and night-terrors are common; sleeping problems also may continue as struggles for control between young children and parents ***(Freud, A. 1952, Grollman, B.A., 1967).***

Reactive effects

In addition to such direct effects, which may closely resemble or overlap disturbances in behavior from primarily psychological or interpersonal sources, other reactive responses may occur ***(Grollman, B.A., 1967).***

Regression

A ubiquitous pattern in children is that of emotional or behavioral regression, seen most strikingly in older infants and preschool children but encountered also in school-age children and adolescentsand for that matter, in adults to a lesser degree. In infants and young children, this regression may take the form of the reappearance of thumb sucking; a return to the bottle; more demanding clinging, negativistic, or aggressive behavior; and the temporary giving up of recently learned patterns, such as speech, walking, and bowel or bladder control. In older

children, the regression occasions the reappearance of more immature social patterns, including greater dependence on the parents, particularly the mother; demanding or aggressive behavior; limitations in the capacity to share with siblings or other; and difficulties in concentration and learning. Such regression appears to stem partly from the direct effects of the illness on the child's ego and partly from the temporary falling back on earlier, more familiar modes or satisfaction or the giving up of more highly developed functions in an adaptive retreat and regrouping of forces to defend the ego against further disruption.

Depression.

Another pattern of response to illness is depression. which takes different forms in infants, children, and adolescents from those seen in adults. Depression may arise partly from the direct effects of the illness, as in infectious from discouragement or the restriction of activity involved, as well as the separation from the parents resulting from hospitalization. Eating and sleeping disturbances are frequent in young children as depressive equivalents, as are changes in motoric behavior, form hypoactive to hyperactive wide mood swings often appear in older children and adolescents. Other emotional responses. often related to regressive

trends. include the reemergence of primitive fears and feelings of helplessness or inadequacy. Stereotyped behavior of a compulsive or ritualized nature may be seen, as may transient hypochondriacal concerns. Open depression may also occur ***(Bergmann, T. 1945).***

Misinterpretation.

Misinterpretation of the meaning of the illness or injury is a common phenomeneon. It is related to the child's limited capacity for intellectual understanding and testing of reality and his tendency toward magical or animistic thinking, particularly in the young child. Younger preschool children ordinarily view pain or discomfort arising from illness or accident as punishment for real or imaginary transgressions. Older preschool and early school-age children show fears of bodily mutilation related to treatment procedures. Such fears are more intense when sensitive areas, such as the head or the genital organs, are involved; fears of harm to the genital organs. are related to sexual differentiation and psychosocial development ***(MacCarthy, D., 1962).***

Physiological reactions

Psychological conflicts about the meaning of illness, enhanced by regressive trends, may result in the appearance of the physiological concomitants of anxiety.

Tachycardia, palpitation, hyperventilation, diarrhea, or other signs and symptoms may be exhibited. Such physiological changes, ordinarily reversible. may compound the effects of the predominantly physical illness, as in the deleterious effects on diminished cardiac function in congestive failure or the perpetuation of diarrhea originally arising from bacterial infection or parasitic infestation ***(Langford, W.S., 1961).***

Conversions reactions

Other psychological reactions seen frequently in school-age children or adolescents in relation to physical illness include conversion reactions. These disturbances affect the voluntarily innervated striated musculature and the somatosensory apparatus, with their unconsciously symbolic expression of emotional conflict. they are to be distinguished from psychophysiological disorders, which affect involuntarily innervated systems and visceral end organs, with out symbolic significance.

Conversion disorders are manifested in a variety of personality pictures, ranging from the relatively normal child to the classical hysterical personality. Transient, mild to moderate symptoms of this nature are frequently encountered during convalescence from a predominantly physical illness. Such an illness may temporarily block a child's intense developmental need to achieve

scholastically, athletically, or socially. Or the illness may precipitate conflict between a regressive wish to continue to be cared for by the mother and guilt over such wishes as the result of the pressure to return to the competitive arena. Frequently, the symptoms of the physical illness are unconsciously incorporated into the conversion symptoms, as in the continuance of pain, vomiting, headache, and dysphonia or, in a more general sense, weakness and easy fatigability. At times, other symptoms, such as certain types of syncope or disturbances in gait, may appear. Many of these conversion reactions are resolved in a few days or weeks, but they may persist chronically in a child with limited adaptive capacity or one whose family encounters special difficulty with the convalescent phase of the illness ***(Heiscl, J.S., 1973).***

Dissociative reactions.

Dissociative reactions such as amnesia somnambulism, Fugue states, and pseudodelirious states may also occur. these reactions may compound or be compounded by an actual delirium, which is often of a subclinical nature. In children, delirium may arise in response to drug administration, head injury, and metabolic disturbances, or it may accompany systemic diseases, with marked fever or a disturbance in cerebral metabolism, even in the absence of any direct insult to the central nervous system.

Catatonic behavior without the significance of psychosis may be seen temporarily in normal or mildly disturbed children if the physical, psychological, or social circumstances sufficiently stressful ***(Jessner, I. et al., 1952).***

Perceptual-motor lags.

Other effects on behavior may be manifest in the convalescent phase. Meyer and Crothers (1946) described children who exhibited lags in perceptual-motor functions after a systemic illness, such as pneumonia. The lags may persist for several weeks or months without apparent damage to the central nervous system. Such children, who may have experienced a temporary disturbance in cerebral metabolism often with delirium, compounded by regressive tendencies may exhibit learning difficulties after returning to school. If they are not recognized, these difficulties may become chronic and result in a resistance to learning or other behavioral disturbance. Their early recognition can allow the parents and the teachers to help the child return gradually to full academic performance ***(Jackson, K., 1951).***

Injury. Although serious repercussions on the child's personality development and family functioning are frequent, many children with chronic disease or with congenital or acquired handicaps make a surprisingly

adequate adaptation to or compensation for their disabilities. Those variables relating to the child's previous adaptive capacity and the parent-child-family balance appear to be of more importance than is the nature of the specific disease or handicap.

Special challenges to development do exist for children with blindness or deafness. with some differences in responses to congenital versus aequired defects Problems in sexual development are present for children with pseudohermaphroditism or extrophy of the bladder. Difficulties in adaptation arise for the child whose movements are restrained in an orthopedic cast and for the handicapped child who requires special prosthetic devices. The debilitation and discomfort produced by certain diseases certainly have an effect on the child's social or academic functioning and on the parents' responses. Even a small and virtually unnoticeable defect may carry overwhelming significance in certain families for unconsciously overdetermined reasons. Although visible cosmetic defects may be most troubling in some families, in others a hidden metabolic defect may be more mysterious and threatening, without relation to its actual severity. Also, conversion mechanisms or other psychological reactions may lead to invalidism in slightly handicapped children. Experienced clinicians have

observed that marked depression and hopelessness detrimentally affect the course of fatal chronic diseases, such as leukemia, and may even be involved, with other factors, in their precipitation.

However, little correlation appears to exist between extremes of unhealthy parental and family response and particular types of personality disorders in the child, and the earlier concept of specific personality malformations arising from specific illnesses seems no longer valid ***(Korsch, B., 1971).***

Effects on personality

The personality pictures seen in children with various diseases or handicaps appear to fall along a continuum, ranging from overdependent, overanxious, and passive or withdrawn patterns with strong secondary gains from illness to overindependent, aggressive modes of behavior with strong associated tendencies to deny illness, even to markedly unhealthy extremes,. A middle group of children show realistic dependence and acceptance of their limitations, with adequate social patterns and compensatory outlets and with no more denial than is consistent with the maintenance of hope.

parental reactions also fall along a continuum, ranging from overanxiety, overprotectiveness, and overindulgences

often with difficulties in setting limits on the child's demands to problems in acceptance of the child's disability frequently with denial of its extent projection of guilt onto the medical staff, reluctance to accept recommended treatment, and occasional rejection of the child within the family unit. A middle group of parents can, after the initial phases of response, comfortably accept the child's limitations permit him appropriate dependency, and help him constructively exploit his capacities and strengths ***(Korsch, B., 1958).***

Effects on body image

Difficulties in the establishment or maintenance of the body image are present for most children with a chronic illness or handicap, but these difficulties may also be seen in children without physical disfigurement. Size strength, and attractiveness do play some role in the child's confidence and social adjustment. but only in special cases or in individual family situations do these factors appear to be of great importance . The introduction of new immunosuppressive drugs and their prolonged use in children in who have had organ transplants often result in dramatic and, at times. devastating changes in body image. In adolescents who are struggling with issues of dependence. independence, regression, and progression and who need a ccepetance by the peer group. reactions

to a damaged body image may greatly interfere with compliance with in hospital and rehabilitative procedures ***(James, V.L. and Wheeler, W.E., 1969).***

Effects of treatment

Although most children respond positively to the effect of recent drugs and medical procedures, a small group may show paradoxical responses, with psychological decompensation, conversion, and other symptomatic reactions. In an unhealthy family situation in which the child with a chronic disease or handicap has taken on a role as an invalid and this role has become central, the precipitation into health of the child by the treatment may upset the tenuous balance of adaptive forces, making it difficult for the child to return quickly to a healthy role within the family. ***Minuchin et al. (1975)*** showed how some children with chronic psychophysiological disorders have become enmeshed in disturbed families and how the child's illness leads to overprotectiveness, rigidity, and lack of conflict resolution among all the family members.

In adolescents, as in adults, psychotic or severe depressive reactions occasionally develop in response to adrenocorticotropic hormone or cortisone during treatment for rheumatic fever or other diseases. In addition to the direct effect of the drug on the brain, problems in the

realignment of intrapsychic and interpersonal balance may also be involved *(Korsch, B.M., 1968)*.

Hospitalization

Hospitalization, with its separation from home and family and various treatment procedures, may cause various reactions related to the child's developmental level and other factors.

Short-term hospitalization

Most, if not all, infants and children some reaction to the experience of hospitalization. Infants in the first half year of life exhibit temporary and rather global responses, arising from different methods of feeding and handling Infants in the latter part of the first year experience stranger anxiety and separation anxiety, often with some regression and depression.

Children under 4 years of age most vulnerable to separation from the mother. this separation is often misinterpreted as punishment or desertion, resulting in feelings of helplessness or fears of attack related to the child's limited capacities for reality testing. In addition to regression and various symptomatic reactions, the phasic sequence of protest, despair, and detachment the last often associated with withdrawal and depression, is frequently seen after a few days of hospitalization, even

in well - adjusted children, and may be troubling to parents.

The following is an example of a young child's hospital experience in which his behavior appears to be more closely related to the presence of his mother than to the state his physical illness.

For the child from 4 years through the early school - age period, the psychological meaning of the illness and its treatment appear to have greater potential effects than the actual separation from the parents. Fears of bodily mutilation and the tendency to misinterpret painful treatment procedures as punishment often invoke anxiety, regression, and the other manifestations mentioned earlier; boys ordinarily exhibit more aggressive responses than do girls.

Older school-age children, who can comprehend the reality of the hospital experience more fully, may show mild regression and anxiety over the functioning of certain organs; these reactions are related to incomplete body images. Fears of genital inadequacy, muscular weakness, and loss of body control or mastery contribute to the feelings of anxiety and inferiority characteristic of this stage. In adolescents, many of the same trends are seen in more muted fashion. There are also struggles to establish a sense of identity and independence, and these

struggles sometimes interfere with the teenager's cooperation in treatment programs.

Parental reactions show some similarity to the patterns seen during acute illness. Denial and disbelief are less marked with less serious illness, with significant exceptions. Many parents fear criticism from the hospital staff regarding their role in the illness or their effectiveness as parents. Some parents may show strong rivalry with nurses or physicians, misinterpreting professional competence in handling the child as a threat to their own parental capacities. Feeling left out or unwanted is also common among parents. A few may project their own guilt onto the hospital staff and blame them for minor difficulties. Difficulty in accepting recommended treatment occasionally leads to their signing the child out of the hospital against advice or not complying with posthospitalization treatment plans.

Parents who have newborn infants in high - risk nurseries are in a special situation. They have not had a chance to assume responsibility for the care of their infant and must delegate this care to the hospital staff. Modern technical advances which include respiratory and cardiac monitors, with their automatic alarm systems often accentuate parental feelings of helplessness, fear, and frustration. Under these conditions, parents may find it

difficult to ask questions concerning their role in helping to take care of their newborn child. The house staff and nursing staff may inadvertently contribute to this difficulty by investing the majority of their time in direct patient care. Consequently, they sometimes miss important verbal and nonverbal cues that are indicative of the parents' increasing anxiety and guilt.

The need to delegate care and the paucity of communication between parents and staff members may have a significant detrimental effect on the eventual emotional adjustment and bonding between the child and his parents. There is a growing concern that hospital care practices that do not take into consideration the potential importance of the early contacts between infants, parents, and hospital staffs could be contributing to the onset of parent-child relationships that lead to the syndromes of battered child, failure to thrive, and the vulnerable child ***(Loomis, B.A., 1956).***

Posthospitalization

Posthospitalization reactions may occur, even in children who have been able to maintain control through the hospital experience, Regressive tendencies, outbursts of anxiety, and fears of doctors or needles may appear in children after they return home when parents are unprepared for such behavior, overprotective or

overrestrictive tendencies may cause significant interpersonal reverberations within the family unit.

Although some reactions of the types described are virtually universal, the majority of children are able to adapt successfully to the experience of hospitalizations, showing self - limited reactions that ordinarily subside after several weeks to several months. But the possibility of emotionally traumatic reactions is great enough, particularly in preschool children and in previously disturbed children, to require careful thought about the indications for hospitalization and to warrant the use of psychological preventive measures ***(Meyer, E., 1947).***

Long-term hospitalization

With modern treatment measures, a long-term hospitalization or institutionalization is rarely necessary for the chronically ill or handicapped child. The studies of Bakwin (1951)and Bowlby (1951), in particular, indicated the likelihood of serious emotional deprivation for such children. In large institutions especially, children exhibit chronic depression and detachment, often leading to shallow social relationships, distorted time concepts, limited capacities for learning, lowered resistance to disease, and rebellious or antisocial behavior. A few children, because of especially appealing qualities, can reach out to the often limited staff in such settings to meet their emotional needs. but significant

personality distortions have resulted for many from such hospitalization ***(Mellish, R.W.P., 1969).***

Surgery and Transplantation

Many of the observations on hospitalization are pertinent to surgical experience for the child. Specials problems may arise in regard to operations with necessarily mutilating effects, such as amputation. If a young child misinterprets the procedures as punishment or a hostile act, he may, as with certain other procedures, become aggressive in fantasied self-defense; fear, withdrawal, or other types of behavior may result. Phantomlimb phenomena, as seen in adults, are rare in children, but strenuous denial may lead to difficulties with prosthetic devices. In older children and adolescents, particularly those with hysterical personality structures, an unconscious need to suffer, arising from guilt over hostile or sexual feelings, may produce recurrent pain.

Anesthesia may evoke fears of loss of self-control, and older children may have fantasies about what may be done, under narcosis, to various bodily organs. children of differing developmental levels respond differently to preoperative sedation; preschool children are often stimulated, rather than sedated, by certain barbiturates. Marked anxiety over operative procedures may

significantly raise the sedation or anesthetic threshold, at the same time reducing the margin of safety in patients with serious heart disease or other debilitating conditions.

Special surgical and immunological advances have recently made it possible to prolong life through the use of transplanted living donor or cadaver organs. The impact of the transplant procedures on the psychological state of the donor, the recipient. and related family members is an issue presently under rigorous investigation. A major stress precipitated by the transplant routines is the need to make critical decisions in an abbreviated time period. These decisions include the considerations that involved organ can no longer sustain life, that the old organ must be removed, that a new organ will be surgically inserted in its place, and that this new organ may be rejected. The final outcomes for both the recipient and his family depends not only on the proficiency of the surgical and medical team but on the ability of the recipient and his family to make successful psychological adaptations to the total transplant procedure. Tiza (1976) observed that, even under these trying circumstances and under the impact of fear of death and actual life-threatening experiences, adolescent patients were found to be striving toward mastery of the adolescent tasks of increasing independence, self sufficiency, peer-group identification,

and the development of sexual identity ***(Minuchin, S. et al., 1975).***

Treatment and Prevention

In the management of physically ill children, the physician can himself offer effective help of a supportive and preventive nature to child and parents. In addition, the physician has the responsibility to act as the coordinator and integrator of the diagnostic, therapeutic, preventive, rehabilitative, and educational skills of many medical subspecialists and numerous other highly trained professional persons available to the child and his family in the hospital and in the community. In so doing, the physician must draw on the principles of continuity, communication, consultation, collaboration, and coordination to render such help most effective to the child-patient and his family. In hospital settings a regularly scheduled ward management conference attended by representatives of the various disciplines, including mental health professionals, and chaired by the pediatrician in charge of the ward can implement these principles.

Treatment and preventions of unhealthy psychological reactions to illness or injury begin with the diagnosis and are importantly involved in the interpretation of diagnostic findings and treatment recommendations to the

parents. Anxious, confused parents need careful explanations and opportunities to ask repeated questions and to air their misconceptions. The physician must also avoid critical, judgmental attitudes toward the parents and should help them deal with guilt and self recrimination.

At all levels of prevention, the physician can rely on the child's natural need and wish to confide and share his feelings. For young children, the parents' positive relationship to the doctor enables the child to displace his confidence from the parents to the doctor and to communicate openly without experiencing a conflict of loyality. Although older children still use their parents' trusting relationship with the doctor, they can more easily form an independent alliance with him or her. Within this setting of trust and understanding, the doctor, the parents, and the child work together to help prepare the child psychologically for his experience in the hospital ***(Plank, E.N. and Horwood, C., 1961).***

Acute illness and hospitalization

When a child has an acute illness or injury, the physician should carefully explain each procedure in advance to him using terms and concepts appropriate to his developmental level. Preparing the young child for elective hospitalization for tonsillectomy, for example, involves a brief discussion of the concrete details of the

hospital routines and anesthetic procedures and a truthful statement that his throat will be sore afterward. Ideally, the parents should be prepared in order to prepare the child. For the preschool child, his mother's overnight stay in the hospital with him is the most valuable preventive and supportive measure. Her stay may be facilitated by a convertible bed that can be kept in the child's room. For the older child, daily visiting is important, whatever his illness. For children who require extended diagnostic procedures or who are in the convalescent stage of their illness, care by parent units can be established. In such units the majority of care is provided by the parents. Another resourceful adaptation that reduces the need for a full complements of professional staff is to provide motel-like units in close proximity to the hospital. The cost of maintaining these units is significantly less than the cost of a full hospitalization. and they make it possible for the child to remain with his parents.

Special programs should be available at the time of hospitalization to help permit the child to play out his anxieties before and after the medical or surgical treatment procedures.

Before a puppet show, the person performing the show should make an assessment of how much the patients who are going to be present at the show know about why they

are in the hospital, what part of their body is sick, what other members of the family have told them, and what questions they may have about what is going to happen to them. The show may then focus on the issues and questions that seems to be of most concern to the patients. After the show the patients can be asked questions about what they learned and can participate in actively using the puppets and other play material to replay any segments of the show that were unclear or that were especially useful in helping them master their anxiety.

Also valuable is schooling, available on a flexible basis even to the briefly hospitalized child. The combined observations of the physician. the nurse, the recreational or occupational therapist, the teacher, and other personnel on the hospital ward are of great value in assessing the child's total hospital experience and in making specific medical and psychosocial recommendations.

A weekly patient group meeting can help facilitate communication between patients and staff members and between the patients themselves. These meetings provide a setting for the exchange of information about ward policies and medical and surgical procedures. Many of the fears and fantasies surrounding potentially painful procedures and the concerns about anesthesia and preoperative and postoperative experiences can be shared

and more fully understood. A supportive and educational approach is used, and the major focus is on the reactions of the group members to their illness and hospitalization.

Children should be cared for at home whenever possible, and due thought should be given to the contraindications as well as the indications, for hospitalization. When hospitalization is indicated, every effort should be made to cooperate with the parents in a therapeutic alliance, and their concerns and needs, as well as the child's, should be kept in mind during hospitalization. If such an approach is adopted, most parents can make vital contributions to the hospital program through their participation in the care of their own or, at times, other parents' children. The handling of crucial phases in convalescence should be worked out cooperatively with parents. Explanations of regressive or other posthospitalization behavior should be given in advance, so that the parents will see this behavior as a characteristic of childhood and not as a personal failure ***(Prugh, D.G. et al., (1953).***

Chronic illness or handicap

For children with chronic illnesses or handicaps, various treatment measures apply. The use of a supportive psychotherapeutic relationship with the child and his parents underlies all other therapeutic steps. Without such

a positive relationship, other treatment is less than optimally effective. The physician should call on the skills of other members of the rehabilitation team, including the child psychiatrists, the psychologist, hospital nurses, social workers, schoolteachers, recreational and occupational therapists, public health nurses, and other professional persons in the hospital and in the community. In the hospital a social worker familiar with the principles of group work can be effective with groups of chronically ill or handicapped children. Group discussions among parents of children with particular kinds of disabilities can also be helpful.

The chronically ill or handicapped child should be cared for in the home whenever possible. Sometimes he can be handled in a regular class in school, with the addition of special classes when necessary. If placement in an institution is necessary, the small institutions and group foster homes with medical and nursing facilities are best.

For the chronically ill child undergoing surgery, the principles already mentioned are vital. With a very young or seriously disturbed child, special arrangements to have the mother in the child's room when he undergoes preoperative sedation and to have her there when he awakens can afford security and continuity for the child.

This arrangement may also help avoid confusion and battles on the way to the operating room. Opportunities to ask questions before and after such procedures can help children work through their misconceptions and anxieties.

Although ill children need some rest and quiet, their natural tendencies toward activity and the hyperactivity engendered by illness and by the restriction of activity should be taken into account. In the hospital a regimen of moderate activity is helpful. Advice about absorbing but quiet activities can be constructive for the potentially harassed and anxious mother taking care of the child at home. Such an approach can minimize struggles over inappropriately enforced bed rest, because these struggles can spread into other behavioral areas and stimulate hyperactivity of a physically and psychologically detrimental nature ***(Richmond, J.B., 1958).***

Terminal Illness The child's concept of death is not fully developed, in the sense of realistic comprehension, until he is about 10 years old. Therefore, children can quite adequately deny fears of death but show other, more immediate fears of pain or separation. Even in adolescents, fears of death are often expressed in terms of fear of pain or of a need for someone to be with them to help them in difficult moments. Parents may carry out mourning in advance, sometimes seeming somewhat

detached from the child at the end, but then reexperience depression or guilt after the child's death. Physicians and nurses are often disturbed by their own feelings during the child's terminal illness.The important thing is for the child and his parents to have someone to whom they can express their feelings, rather than maintain an unrealistic silence. Psychiatric consultation may be helpful in understanding such situations and in working out a plan for supportive help. parents often require support after the child's death, and opportunities for this support should be routinely available to them. The wish to replace the lost child before adequate time has been invested in the the work of mourning is a frequent issue that can be resolved if it is identified ***(Shran, H., 1965).***

Caustic Injuries to the Esophagus in Children

The esophagus has long been a forgotten organ that is recognized only as conduit to allow food to pass from the mouth to the stomach. One only appreciate the importance of this hollow tube when its function has been disrupted The esophagus is an organ that poorly tolerates trauma; it manifests the complications of injury in a significant and frequently chronic manner. Infants and young children are the true testers of esophageal integrity-they challenge the esophagus with foreign bodies, including coins, jacks and balls, detergents, soaps, cleansers, and other more caustic agents. The ingestion of caustic agents can initiate a progressive and devastating injury to the esophagus and stomach. In the United States, it is estimated that between 5000 and 15,000 caustic ingestions occur annually. As with all other ingestions, the age is bimodal, with the majority occurring in children who are younger than 5 years of age. Another peak occurs in young adults between 20 and 30 years of age; these ingestions usually represent suicide attempts ***(Adam, J.S., and Brick, H.G., 1982).***

With the introduction of concentrated liquid alkaline cleansers in the 1960s, the incidence of severe caustic injury to the esophagus increased sharply. Most accidental ingestions have occurred in children who gained access to strong household cleaners that were stored inappropriately in food and drink containers. Seventy-five per cent of caustic burns the esophagus occur in children who have unknowingly ingested sodium hydroxide or lye. Eighty-three per cent of these children are younger than 3 years of age ***(Applequist, P. and Salno, M., 1980).***

Fortunately, the number of caustic ingestions that occur yearly has decreased over the last decade. This decrease has been accomplished through a nation-wide campaign led by pediatricians to increase public awareness of the devastating consequences of caustic ingestion. Most caustics now are sold in child-resistant containers and legislation requires the manufacturing of less concentrated agents ***(Genieser, N.B., and Becker, M.H., 1969).***

AGENTS THAT INJURE THE ESOPHAGUS

Caustic substances include a wide variety of materials that have different and unique properties; however, they all share the ability to cause direct chemical injuries to tissues . This group of substances includes oxidizing agents, such as chromic acid and potassium permanganate; reducing agents, such as hydrochloric acid and nitric acid;

corrosives, such as phosphorous and lye; and desiccants, such as sulfuric acid ***(Nagy, M. 1973)***. Many caustic substances commonly are found in the home. These products usually are strong alkaline or acidic agents.

Lye is a broad term for strong alkali used in cleansing agents ***(Kulig, K., et al., 1983).*** Sodium and potassium hydroxide in granular paste and liquid form are used for drain and oven cleaners, washing powders, Clinitest tablets, and home soaps. Concentrations of these alkalis vary from 9.5 to 32 per cent in a liquid form up to 100 per cent in a solid form ***(Litovitz, T.L., 1985; Mills, L.J. et al., 1979; Ritter, F., et al., 1968).*** Although current household products are less concentrated (8 to 10 percent) than products sold in previous years, the continued availability of more highly concentrated, industrial- strength alkali products must not be overlooked. Because of their high specific gravity, these products the "ability to sink to the bottom of the clogged toilet bowl or drain" This property increases the risk of injury to the lower esophagus and stomach the recent use of the alkali, sodium carbonate, as a substitute for the noncaustic phosphate detergents is an example of poor manufacturing foresight. Ingestion of this agent often results in significant esophageal injury ***(Sugawa, C. et al., 1981).***

Commercial ammonia or ammonium hydroxide is an alkali that also has an associated vapor production. A large number of ammonia ingestions have been reported in series reviewing caustic ingestions ***(Votteler, T.P. et al., 1983; Oakes, D.D., et al., 1982).*** Ammonia ingestion results in mucosal burns and ulcers. Full- thickness burns of the esophagus can develop after ammonia ingestion. In addition, inhalation of ammonia vapours can cause irritation of the lungs and even frank pulmonary edema ***(Ray, J.F., et al., 1974).***

Children also ingest acidic agents. Acids are commonly available in the home as toilet-bowl cleaners in the forms of sulfuric, hydrochloric, and phosphoric acid. Soldering fluxes, antirust compounds, battery fluids, swimming-pool, and slate cleaners also are acids ***(Litovitz, T.L., 1983).*** Unlike the alkaline solutions, which do not have much taste, acids are bitter. On initial contact to the oropharyngeal mucosa, these agents burn and usually are spit out before much is swallowed.

Detergents and bleach (sodium hypochlorite) are the substances most commonly ingested by children. Hypochlorite bleach is considered an esophageal irritant; it does not cause tissue necrosis ***(Marion, L., et al., 1978).*** Owing to its near neutral pH of 6, it does not produce significant esophageal or gastric injury. In contrast,

industrial- strength detergent containing sodium tripolyphosphate can cause significant mucosal injury and should be approached with the same concern as acid and alkali ingestions.

Another common form of esophageal injury occurs when a child swallows something that is too hot. The burning in the child's chest is a form of thermal injury. Thermal injury to the esophagus can be produced by the ingestion of dry ice, boiling water, or hot foods. The injury usually is superficial and does not cause significant morbidity. On the other hand, Clinitest tablets, which are used commonly diabetics to check their urine, are alkaline agents that cause significant thermal, as well as caustic, injury of the esophagus; the combination causes a devastatingly deep burn that usually progresses to stricture formation ***(Leape, L.L., et al., 1971).***

Recently, the medical literature has alerted physicians to the potentially harmful risks of miniature disc-battery ingestion ***(Friedman, E.M., and Lovejoy, F.H., 1984; Citron, B.P., et al., 1968; Buttross, S., and brouhard, B.H., 1981).*** Due to the limited use of these devices, complications of disc-battery ingestions were not reported prior to the mid-1970s. Since then, reports of disc-battery ingestion have been sporadic, and no individual has had extensive experience with this problem. These electronic

devices are composed of various heavy metals-such as mercury, zinc, silver, nickel, cadmium, or lithium-in concentrated electrolyte solutions of 26 to 45 per cent potassium or sodium hydroxide. Leakage of such caustic solutions from the battery can cause esophageal and gastric burns. In addition, the corrosive effects of mercuric oxide contained within some power cells may injure esophageal mucosa. Serum mercury levels also may be increased after disc-battery ingestion ***(Chung, R., and Denbesten, L., 1971).***

The national button-battery ingestion study was established in order to accumulate data and formulate treatment plans to deal with the increasing incidence of disc-battery ingestion. This study revealed that almost 90 per cent of the ingested button cells successfully traversed the gastrointestinal tract without any complications and were passed in the stool. Symptoms developed in only 10 per cent of button-cell ingestions were minor, except in the single case of a battery lodging in the esophagus. This group recommends that an initial roentgenogram be obtained to determine the location of the battery and to exclude esophageal lodging. If the disc is beyond the esophagus, the patient is sent home. Follow-up roentgenograms should be performed if the battery is not passed within 4 to 7 days. If this roentgenogram

demonstrates a persistent gastric position of the battery, metoclopramide hydrochloride can be used to improve gastric transit. Noting the frequently impeded transit of batteries 23mm in diameter, a 48-hour roentgenogram should be obtained in all cases of ingestion of larger cells. In those patients whom the larger battery remains in the stomach longer than 48 hours, endoscopic retrieval should be attempted. Urgent endoscopic retrieval is indicated if the battery is lodged in the esophagus. If signs or symptoms suggest bowel perforation, operative intervention is necessary. In the absence of these specific clinical manifestations, endoscopic or surgical retrieval is not required and the majority of battery ingestions were benign and could be managed conservatively ***(Hawkins, D.B., et al., 1980).***

PATHOGENESIS AND PATHOLOGY OF CAUSTIC INJURY

Once a caustic agent has been ingested, the extent of injury to the gastrointestinal tract depends upon the type of agent, its concentration, quantity, physical state, and duration of exposure in the esophagus ***(Krey, H., 1952).*** Accidental ingestions sometimes are interrupted by the odor or taste of the ingested substance. Acidic solutions are bitter tasting and cause immediate pain. Unless ingestion is intentional, the agent frequently is expelled

rapidly. On the other hand, liquid alkaline solutions often are tasteless and odorless and are swallowed before protective reflexes can be invoked.

Granular agents often produce focal injury to the oropharynx and proximal esophagus, in comparison to liquid agents, which cause extensive, contiguous damage to the entire esophagus and stomach linings. The extent of injury is limited only by the quantity and concentration of the ingested compound. Smaller quantities at lower concentrations result in less mucosal damage. In contrast, solutions with high specific gravities produce extensive and severe injury. These agents can destroy the full thickness of the esophagus injury adjacent tissues, and cause gastric perforation ***(Lee, J.R., et al., 1972; Daly, J.F., and Cardone, J.C., 1961; Messersmith, J.K., et al., 1970).***

Macroscopically the esophagus seems to be damaged at areas of relative stagnation: the criopharyngeal area, the impression by the aortic arch and left bronchus, and the lower-esophageal sphincter. In addition to contact injury, caustic agents also induce myospasm. Caustic injury to the lower-esophageal sphincter decreases its resting pressure, allowing the caustic to reflux back into the esophagus. The esophagus attempts to propel the caustic back into the stomach. This to-and-fro action can

expose the esophagus to the caustic for a period of 3 to 5 minutes. Violent regurgitation usually follows ***(Middlekamp, J.N., et al., 1969).***

The main difference between alkaline and acidic injury is the rapid penetration into tissue by alkali. Experimentally, it has been shown that the depth of injury is more significant with alkaline ingestion. In cats, 1 ml of 30 per cent sodium hydroxide solution exposed for 1 second can penetrate the full thickness of the esophagus. A 3.8 per cent sodium hydroxide solution, in contact with the esophageal mucosa for 10 seconds, produces necrosis of the mucosa, submucosa, and longitudinal muscle fibers. Using increasing concentrations resulted in deeper injury, to the point of frank perforation ***(Penner, G.E., 1980).***

Alkali do have a potent solvent action on the lipoprotein lining, producing liquifaction necrosis, with intense inflammation and saponification of the mucosal, submucosal, and muscular layers of the esophagus and stomach ***(Rostlethwait, R.W., 1979; Scher, L.A. and Maull, K.I., 1978; Temple, D.M., and Menesses, M.C., 1983).*** During the first several days after an alkaline ingestion, mucosal fats and proteins become saponified, and adjacent blood vessels are thrombosed, leading to cell necrosis and tissue degeneration. Alkali can penetrate deep into the esophageal wall and, at times, into the

paraesophageal and mediastinal tissue ***(Knox, W.G., et al., 1967)***. Bacteria commonly invade the injured site. Necrotic slough occurs 4 to 7 days after the initial ingestion and is followed by the appearance of fibroblasts and deposition of collagen during the next week. Strictures may first appear at about 21 days, whereas complete stricture formation takes 28 to 42 days ***(Johnson, E.E., 1963)***.

Microscopically, acidic agents cause coagulation necrosis, leading to the production of firm eschar. This protective and impedes the penetration of the caustic, limiting the depth of injury. Acidic agents traditionally cause more severe damage to the stomach. When the acid reaches the stomach, it will follow the lesser curvature, along the magenstrasse to the pylorus. Pylorospasm develops, resulting in pooling of the acid in the prepyloric antrum, where the greatest injury occurs. This is usually the site of deep ulcer formation and perforation. Pyloric stenosis may be a late complication. Injury is much more severe when ingestion occurs in an empty stomach ***(Temple, D.M., and Meneese, M.C., 1983)***.

Caustic injuries of the esophagus are classified pathologically in the same manner as burns of the skin. First-degree injury causes hyperemia, with superficial desquamation. Second-degree injury is characterized by

blisters and shallow ulcers limited to the mucosa. Third-degree burns cause ulceration deep into the muscle and may be transmural.

Pathology

Injuries due to chemical burns of the esophagus may be classified in a manner similar to that used to describe thermal burns of skin. A first degree burn signifies minimal involvement, characterized by superficial mucosal hyperemia, mucosal edema and superficial sloughing. A second degree burn is transmucosal, involving all the walls of the esophagus, with exudate, ulceration, loss of mucosa and extension into muscle. A third degree burn is one which in addition to involving the esophageal walls has eroded through the esophagus into the periesophageal tissues, including the mediastinum, or has perforated into the pleural or peritoneal cavities. The course of these injuries begins with an acute inflammatory phase involving the first four days after the injury. Contact of the caustic or corrosive agent with the mucosa results in an intense inflammatory reaction, coagulation of cellular proteins and destruction of cells with necrosis, inflammation of surrounding living tissues, thrombosis of vessels with mucosal hemorrhage and bacterial infiltration. This is followed by a subacute phase, lasting three to 15 days, toward the end which the necrotic tissue sloughs away

leaving a denuded, ulcerated surface. Granulations appear as fibroblasts and new blood vessels develop. The esophagus is considered weakest during this time, from the end of the first week through the second week. Swelling subsides, and often swallowing begins to return to normal, leading to the false conclusion that whatever therapy has been used was effective and need not be continued, Cicatrization, the third phase, begins the third and fourth weeks as the inflammatory reaction subsides and connective tissue contractures begin. Submucosa and muscularis are replaced by dense fibrous tissue, and adhesions involving the esophageal wall as well surrounding structures cause bands with circular or spiral strictures that may result in the obliteration of segments of the lumen. Re-epithelialization is complete by the sixth week, but in deep burns the normal physiology of secretion, elasticity and peristalsis are lost to varying degrees.

Symptoms and Diagnosis

In an acute burn it would seem that the history of ingestion of any of these agents that are so severely destructive, together with even the most cursory examination of the patient's mouth, would make the diagnosis self-evident. There is little doubt about the diagnosis if a child or an hysterical adult is brought to the

emergency room drooling, in severe pain and in shock, by distraught relatives carrying a bottle or jar containing the remnants of material known to have been ingested. Yet a serious question arises if the mother merely surmises the child swallowed something because it was found crying in a kitchen closet with an overturned powdered lye container, or if a depressed adult was seen staring into the medicine cabinet where a half-empty bottle was found spilled on a shelf. In such situations, it is best to urge the family to follow the instructions of the AMA "poison chart" : "Save and give to the physician or hospital the poison container with its intact label and any remaining contents. If the poison is unknown, bring the vomitus for examination". The type of material ingested may be identified by such examination ***(Scher, L. A., and Maull, K.I., 1978).***

In addition to determining the type of poison, it is necessary to attempt to evaluate the extent and degree of the burn or burns. Examination of the lips, tongue, buccal pharyngeal mucosa, and a correlation of the findings with the history, usually give sufficient evidence of chemical ingestion. In severe burns, circumoral and glossal edema, inflammation and bleeding are present, with refusal of food or fluid, dyspnea, and evidence of spill with burns on the clothing, face, hands or other parts of the body. Severe

pain, total lack of cooperation, and shock accompany these local manifestations. Substernal pain, pain in the back, abdominal pain and rigidity are indicative of mediastinal or peritoneal extension. Hoarseness, stridor and dyspnea suggest either associated laryngeal edema or actual epiglottic and laryngeal destruction through aspiration of the chemical during swallowing ***(Holinger, P.H., et al., 1958).***

With lesser involvement, while questions may not arise as to the actual ingestion of a caustic, corrosive or bleach, a major question remains as to the site and degree of damage that has resulted. Opinions vary as to the accuracy of further diagnostic procedures. Fluoroscopy of the esophagus during the acute stage is uninformative unless the destruction is great; but under these circumstances the child or adult will refuse all attempts to force them to swallow barium. Burns of the lips and tongue do not give positive evidence of esophageal burns, and the absence of evidence of burns of the oral mucosa does not exclude esophageal involvement. Furthermore, parents sometimes minimize symptoms if they are not sure of the amount of chemical ingested. An esophagoscopic examination may be made under general anesthesia, preferably within the first 48 hours, in an effort to confirm the diagnosis and locate the most proximal site of

involvement. It is important to avoid additional trauma and possible perforation by not advancing the esophagoscope beyond the upper limit of any encountered. Esophagoscopy is contraindicated, however, in the presence of severe burns of the mouth and hypopharynx when dyspnea and aphonia give evidence of laryngeal edema. Under these circumstances, endotracheal anesthesia would be associated with a considerable risk and possibly might require a tracheotomy. The esophagoscopy in such a burn would reveal nothing more than the cricopharyngeal edema and exudate, since to attempt to enter the esophagus itself would be too hazardous ***(Cleveland, W.W., et al., 1968).***

With lesser involvement of the mouth and larynx, identification of the site and degree of the injury seems indicated. Should no evidence of esophageal burns be detected, it may be concluded that the pathology is limited to the mouth, and therapy is terminated when the burns in the mouth have healed. Under these circumstances a barium swallow should be done in three to four weeks to recognize the occasional stricture.

Symptoms of the late results of ingestion of chemicals are those of gradual dysphagia first for solids and eventually for liquids as scar tissue slowly constricts lumen. Such changes may become apparent within a few

weeks after the accident, but changes sometimes are so gradual and slowly progressive that stricture formation is not realized for months or even years. When these occur in a child, the slow change in eating habits may be misinterpreted as a "feeding problem"; if an adult, voluntary dietary restrictions are sometimes misinterpreted as a personality quirk. Not infrequently in these late changec, diagnosis is first apparent only when a bolus of meat or some small object becomes lodged in the esophagus necessitating endoscopic removal; subsequent diagnostic fluoroscopic studies reveal the stricture ***(Giffin, C.S., 1964).***

Therapy

The AMA "poison chart" gives the following instructions for the immediate management of acute chemical ingestion :

For Alkali or Acid Ingestion: DO NOT INDUCE VOMITING; DO NOT ATTEMPT GASTRIC LAVAGE; DO NOT USE EMETICS.

For Alkali Caustics (Lye, washing soda, household ammonia): Milk, water; citrus fruit juices, dilute vinegar, followed by egg whites, butter, olive or mineral oil. Suggested dose: for patients 1-5 yrs. of age, 1 to 2 cups; for patients 5 yrs. or older, up to 1 quart ***(Poisons and Antidotes, 1963).***

For Acids (Nitric, sulfuric, muriatic, acetic, hydrochloric, carbolic, oxalic, iodine, silver nitrate): Milk, water, milk of magnesia (1 tablespoonful to 1 cup of water), soap solution, aluminum hydroxide gel, followed by egg whites, butter, olive or mineral oil. Suggested dose: for patients 1-5 yrs. of age, 1 to 2 cups; for patients 5 yrs. or older, up to 1 quart ***(Haller, J.A.,Jr., and Bachman, K., 1964).***

For Bleaches: Warm water gastric lavage, emetics, saline cathartics.

Hospitalization for further evaluation of the extent of injury is indicated following the initial emergency measures. There is a wide variation in management of patients with suspected or actual fresh chemical burns of the esophagus. This is apparent in results of experimental studies and from reports of clinical experience. Our own routine varies according to the extent of the injury as evaluated by the history, symptoms, physical findings, and esophagoscopy when indicated ***(Greengard, J., 1966).***

Management of Minimal Involvement to First Degree Burns. The history is vague or questionable; the emergency measures described above are employed and broad spectrum antibiotics are started. In 12 to 24 hours, patients in this category have little or no drooling, begin to accept liquids, and only inflammatory changes may be

present in the oral or phyaryngeal mucosa. Demulcents such as Amphojel or Gelusil will give symptomatic relief and solids may gradually be added to the diet as swallowing function returns. Hospitalization is generally unnecessery ***(Cannon, S., and Chandler, J.R., 1963).***

It had been our practice to make no esophagoscopic examination, and no steroids were used in patients in this category. On the second day Nos. 14 and 16F, or, in older children. Nos. 20 and 22F sized mercury-filled Hurst type bougies; well lubricated, were passed easily and quickly into the stomach,without anesthesia. Dilatations were repeated a week later, increasing the bougies by 2F sizes and eliminating the smallest from the series. Bougienage using the next larger size was repeated in one week, then two weeks, one month and finally three months. The bougies were increased to No. 30F in small children, 36 to 40F in the older children and 44F in adults. It has been our experience that when a history of ingestion has been obtained, with or without visible burns in the mucosa of the mouth, this type of therapy with rubber mercury-filled bougies is simplest, practically atraumatic, requires minimal or no hospitalization and insures against the possibility of late stricture formation. The alternative use of steroids following a diagnostic esophagoscopy under general anesthesia is described below ***(Holinger, P.H., et al., 1954).***

Moderate Involvement : First to Second Degree Burns. Emergency measures described above are employed, the patient is hospitalized, and analgesics are administered because of pain. Antibiotics as above are started intramuscularly or intravenously since pain, dysphagia and lack of co-operation generally prevent oral administration. An esophagoscopic examination under general anesthesia is made to confirm the diagnosis and locate the most proximal site of involvement. It is important to avoid additional trauma and possible perforation by not advancing the esophagoscope beyond the most proximal evidence of the burn. Fluids are given intravenously when oral intake is insufficient. Bougienage as described above was done alternate days for the first week, then twice a week for two weeks. Subsquent bougienage was regulated according to the ability to maintain a normal lumen through a gradual increase in the size of bougies that would pass readily into stomach. Since edema normally subsides in two weeks, and repair through granulation and ultimately healing takes place, the bougienage was repeated in three weeks, a month later, and then in three months. Dilatation was repeated three times the next year, and later only if fluoroscopy or symptoms suggested beginning narrowing ***(Ashcraft, K.W., and Padula, R.T., 1974).***

Much experimental work and many clinical series reports indicate the importance of adequate steroid and antibiotic administration in the treatment of acute chemical burns of the esophagus. This combined management appears to reduce the morbidity of this serious problem, but to be effective the therapy must be initiated as soon after the accident as possible. Esophagoscopy under general anesthesia is done within the first 48 hours after the accident, and the following regimen is initiated if burns of the esophagus are encountered. Antibiotics (Ampicillin, 25 mg/kg/day) and steroids (Prednisone) are administered according to the following dosage schedule: ***(Middelkamp, J.N., et al., 1969)***

20 mgm. q 8 hours for 4 days = 60 mgm. per day
20-15-20 q 8 hours for 4 days = 55 mgm. per day
15-15-20 q 8 hours for 4 days = 50 mgm. per day
15-15-10 q 8 hours for 3 days = 40 mgm. per day
10-10-10 q 8 hours for 3 days = 30 mgm. per day
5-10-5 q 8 hours for 3 days = 20 mgm. per day
5-0-5 q 8 hours for 2 days = 10 mgm. per day
5-0-0 q 8 hours for 2 days = 5 mgm. per day

An esophagram is made three weeks to one month after the accident. Bougienage is indicated if the x-ray study shows narrowing anywhere along the course of the esophagus or if the patient experiences any dysphagia.

Those without evidence of burns on esophagoscopy and those with burns but who are asymptomatic and with negative X-rays will be seen at three months, six months and 12 months post-ingestion for re-evaluation.

If the patient is first seen more than 48 hours after the accident, antibiotics are used together with bougienage but no steroids. There is serious danger of perforation if bougies are used during or immediately following a course of steroid therapy ***(Burrington, J.D., 1975).***

Severe Involvement: Extensive Second and Third Degree Burns. Emergency measures as described; hospitalization, treatment for shock, intravenous fluids, sedation, analgesics and antibiotics. Bougies may be attempted beginning the second day unless clinical and x-ray findings show evidence of erosion into the mediastinum or peritoneum. Steroids may be used as described above, but if so, all bougienage is strictly avoided. The steroid administration may continue for ten days, gradually tapering off the dosage; longer administration results in an increased incidence of esophageal and gastric ulcers and perforation. Esophagoscopy under general anesthesia is done only if pharyngeal, hypopharyngeal and laryngeal destruction do not contraindicate it. However, in these extensive burns esophagoscopy will probably give little additional

information as to the extent of involvement. Burns of this degree almost invariably are apparent in the mouth, hypopharynx and larynx, often with total destruction of the epiglottis which precludes esophagoscopy ***(Ein, S.H., et al., 1973)***. Burns of this severity further down the esophagus are identified by esophagoscopy if there is limited hypopharyngeal involvement and this examination can be made. Patients are fed intravenously or by gastrostomy and may require tracheostomy because of obstructive pharyngeal or laryngeal edema. As soon as liquids can be tolerated orally, attempts are made to have patient swallow a # 4 surgical thread (without bead or knots). It is kept loosely in the mouth, allowing it to advance through the gastrointestinal tract; when possible, a # 14 surgical braided silk is attached and carried down by the advancing fine silk thread. Bougienage is started two weeks after discontinuation of the steroid therapy ***(Gavriliu, D., 1975)***, either by mercury-filed Hurst or Maloney (tapered) bougies or with Tucker or Plummer-type olive tipped bougies the string as a guide. Esophagoscopy under general anesthesia with small diameter esophagoscopes using the string guide may be successful in establishing a lumen and in increasing its size. When a gastrostomy is used for feeding purposes the gastrostomy should be planned for possible future use as a means of prograde or retrograde string-guided esophageal

dilatation. The gastrostomy should be low, into the fundus, directed toward the cardia rather than the pyloris, and proximity to the lower ribs should be avoided. If destruction by the ingested chemical has involved the stomach and pyloris, necrosis and edema prevent a successful gastrostomy and a jejunostomy for feeding purposes is necessary as an alternative. Obviously, string-guided retrograde dilatations are not possible under these circumstances, although prograde dilatations may be accomplished ***(Alford, B.R., and Harris, H.H., 1959).***

Chronic Strictures. The establishment and maintenance of a relatively normal esophageal lumen after disclosure of a tight esophageal stricture or even an atresia is feasible. When this fails, the various surgical replacement procedures may be employed. Some chronic strictures respond rapidly to dilatation with successively larger mercury-filled Hurst or Maloney bougies. Others respond more satisfactorily to string-guided Plummer or olive-tipped bougienage. Tight, firm multiple strictures respond quickest and most safely to gastrostomy and prograde or retrograde dilatation with string-guided metal olive-tipped bougies or to rubber Tucker bougies attached to the thread guide. Some prefer an indwelling esophageal rubber or plastic tube, gradually increasing its diameter, while others advocate heavy metal dilators which are

allowed to dilate the esophagus by gravity. These procedures have been advocated both perorally as well as through a cervical esophagostomy ***(Haller, J.A. et al., 1971) (haller J.A., 1979).***

When the esophagus appears to be atretic, often a lumen may be re-established by careful esophageal dilatation, using radioopaque bougies guided fluoroscopically. The frequency of success is increased if a second esophagoscope or even a cystoscope is introduced into the gastrostomy and a bougie is guided upward from below and finally recovered in the mouth. A thread may then be attached and the bougie carries the thread back into stomach as it it withdrawn, establishing a lumen and guide for subsequent dilatation. It should be noted that often when along segment of esophagus appears atretic, this may be only two web-like strictures with a relatively normal segment of esophagus between, and recannulation and dilatation rataion rapidly restores normal swallowing function ***(Reyes, H.M., et al., 1974).***

Surgical replacement of the esophagus following extensive burns has been feasible only during the past 20 years. Using colon replacements ***Delarue A., et al., 1985,*** or stomach pull up ***Lindahl, H., et al., 1983, Cohen D.H. et al., 1974; Othersen et al., 1988, Gregorie H.B., 1972; Mahmou Baawi et al., 1995***. The re-establishment of oral

deglutition and elimination of the necessity of routine gastrostomy feedings is one of the most satisfying and rewarding accomplishments for patient and surgeon alike, restoring the afflicted person to his place in society ***(Kirsh, M.M. and Ritter, F., 1976).***

MATERIALS AND METHODS

MATERIALS AND METHODS

100 patients were included in this study. They were exposed to corrosive injurey of the aesophagus. They were classified into two groups.

First group

50 patients 34 males and 16 Females with age range 1.5 to 6 years. They were refered to Nasser institutes. Dar El-Sheifa and Ein shams university from the surrounding area (urban)

Second group

50 patients 32 males and 18 Females with age range 1.5 to 7 years they were refered to Menoufia University and Shebien El-Kom educational hospitals from the surrounding area (rural)..

All patients were subjected to epidem iological study of following :

1. Type of corrosives.
2. The amount of the ingested substance.
3. The stage of oesophageal stricture.
4. The degree of oesophageal stricture.
5. Age.
6. Sex.

7. The frequent usage of chemicals in their house.
8. The knowldge of the Family about this type of lesion.
9. The profession of their father.
10. The profession of their mother.
11. The economic state of the family.
12. The Number of their family.
13. The rank of the child in his famihy
14. The father & Mother relation ship.
15. The abscence of one or both parents.
16. The educational level of the parents.
17. The morbidity and mortality..
18. The effect of the morbidity of the child to his parents.
19. Educational study and intelligence using stanford-Bine scale.
20. Behavior changes of the families in relation to their children illnes.

60 patients with recent injuries (30 from each group) were subjected to full clinical examination, anthropometeric, and psychological development (Review page 65, 85) and follow up them 4 years after exposure to oesophageal injury and study the effect of the cortico steroid on the state of the oesophageal stricture in the form of prednisone 2 - 3 Mg/kg/body weight every 24 hours and antibiotics in the form of (ampicilline) 50 - 100

mg/kg/body weight every 24 hours for a total of three weeks, if the patient is initially unable to take orally, hydrocortison 2 - 3 mg/kg/body weight I.M every 24 hours and ampicilline ampules 50 - 100 mg i.M. every 24 hours until the patients can swallow the drugs and then continue the previous oral prednisone Endoscopy was done by flexible fiberoptic endocopy immediately after the injury in the recent referal cases and for the old one at the first visit it was done in the purpose of diagnosis of the stage and degree of the oesophageal injury with subsequent follow up to assesse the progress of the disease, also it was done as therapeutic to do frequent dilatation which was done by pneumatic ballon or by the shaft of the endoscopy it self the dilatation was done under general endotracheal anaesthesia every week for one month, then every 2 weeks for 6 month and then every 1 month for 1 year. beside the endoscopy, X-ray Barium swallow in erect and trendelenburge position to detect the upper and lower border for the impassable endoscopy. Surgical treatements for the patients whome did not respond to the Medical and endoscopic treatement, they were operated upon by colon by pass operation or gastric pull up technique.

All the resulting data were tabulated and statistical analysis.

RESULTS

RESULTS

(I) Epidemiological studies of the caustic oesophageal injuries.

Table (11) : Incidence of caustic oesophageal lesion according to sex. and residance

	No. of patients		Percent.	
	Male	Female	Male	Female
Rural	32	18	64	36
Urban	34	16	68	32
Total	66	34	66	34

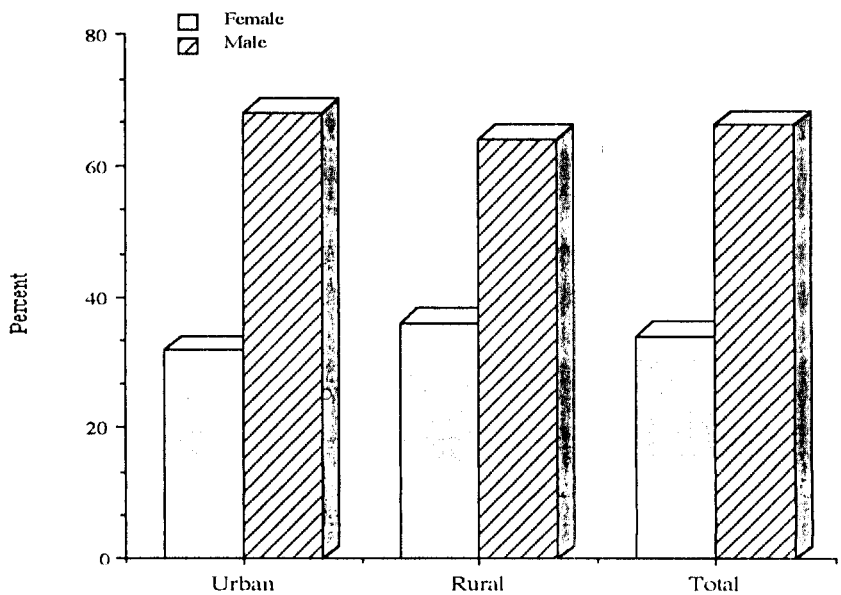

Figure (11) : Incidene of caustic oesophageal lesion in relation to sex and residance.

Table (14) : Incidence of caustic oesophageal stricture in relation to the type of caustic agent

	Potash		Other.	
	No. of patients	Percent	No. of patients	Percent
Rural	46	92	4	8
Urban	47	94	3	6
Total	93	93	7	7

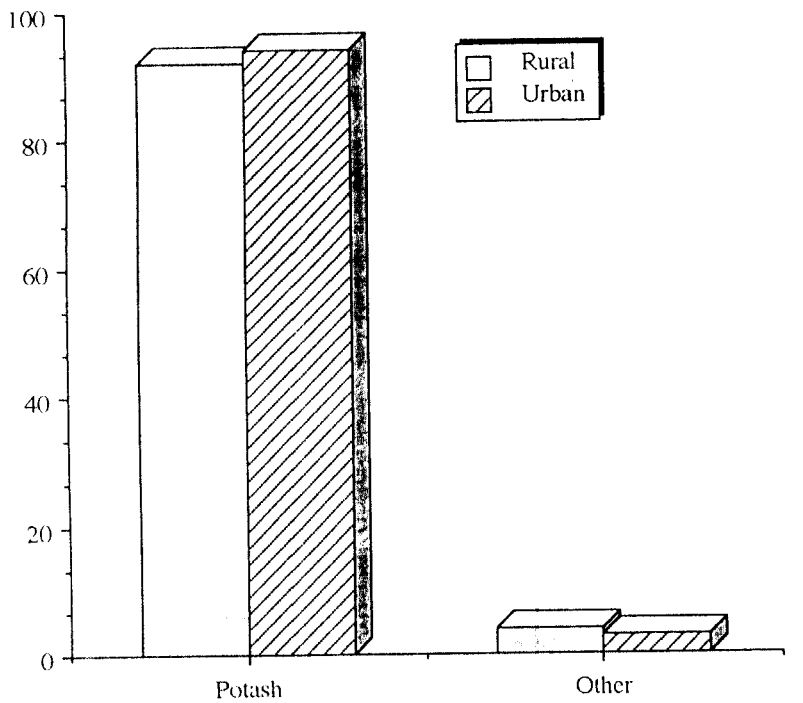

Figure (13) : Incidence of caustic oesophageal stircture in relation to the type of caustic agent.

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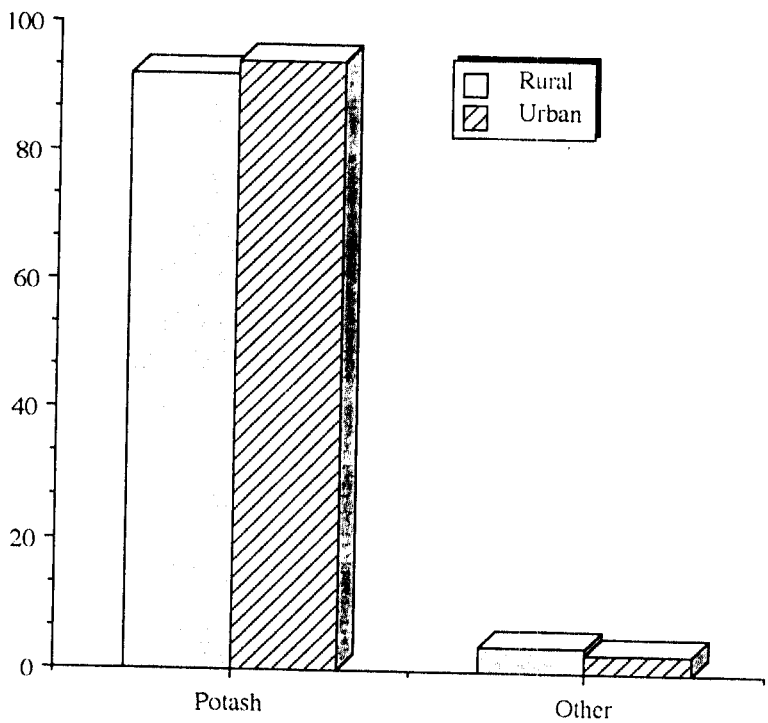

Figure (13) : Incidence of caustic oesophageal stircture in relation to the type of caustic agent.

Table (15) : Incidence of caustic oesophageal stricture in relation to presence of special place far away from children for keeping caustic.

	There is a place		No special place	
	No. of patients	Percent	No. of patients	Percent
Rural	9	18 %	41	82 %
Urban	7	14 %	43	86 %
Total	16	16 %	84	84 %

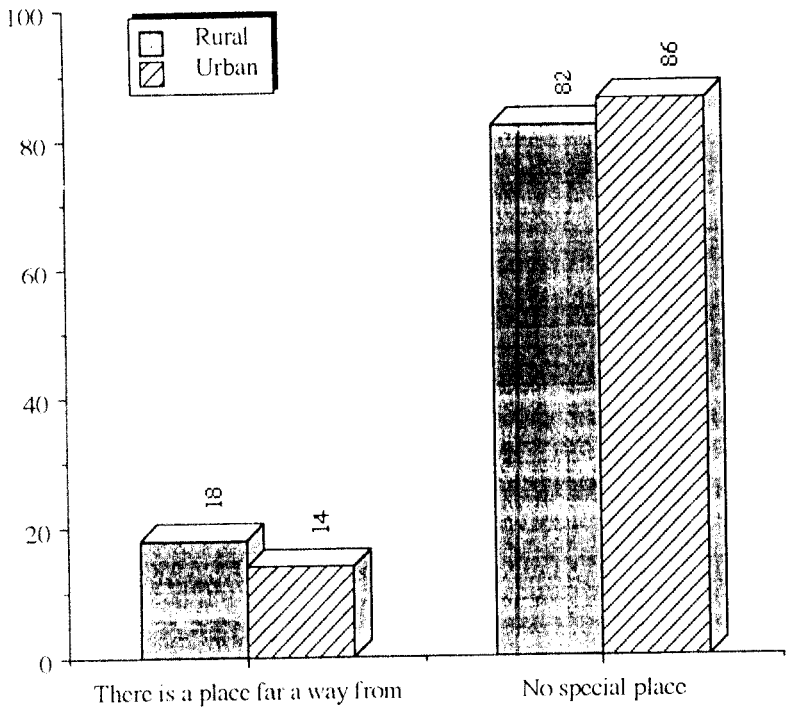

Fig. (14) : Incidence of caustic oesophageal stricture in relation to presence of special place far away from children for keeping caustic.

Table (16) : Incidence of caustic oesophageal stricture in relation to the frequent use of caustic detergent.

	Frequent uses of caustic		No frequent uses of caustic	
	No. of patients	Percent	No. of patients	Percent
Rural	42	84 %	8	16 %
Urban	41	82 %	9	18 %
Total	83	83 %	17	17 %

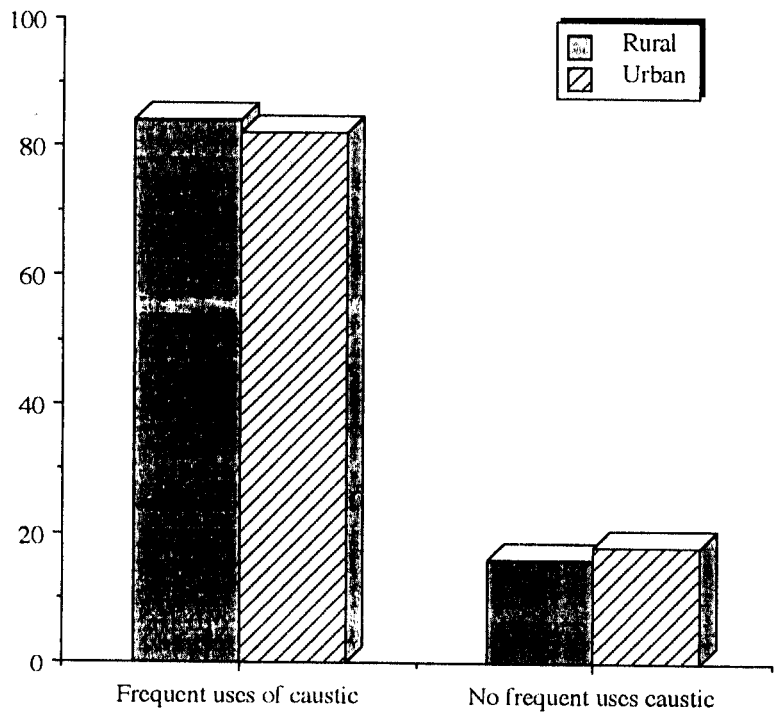

Fig. (15) : Incidence of caustic oesophageal stricture in relation to the frequent use of caustic detergent.

Table (17) : Incidence of caustic oesophageal stricture in relation to the baekground knowldge of the parents about similar conditions.

	Parents have background knowldge		Parents haven't background knowldge	
	No. of patients	Percent	No. of patients	Percent
Rural	14	28 %	36	72 %
Urban	14	28 %	36	72 %
Total	28	28 %	72	72 %

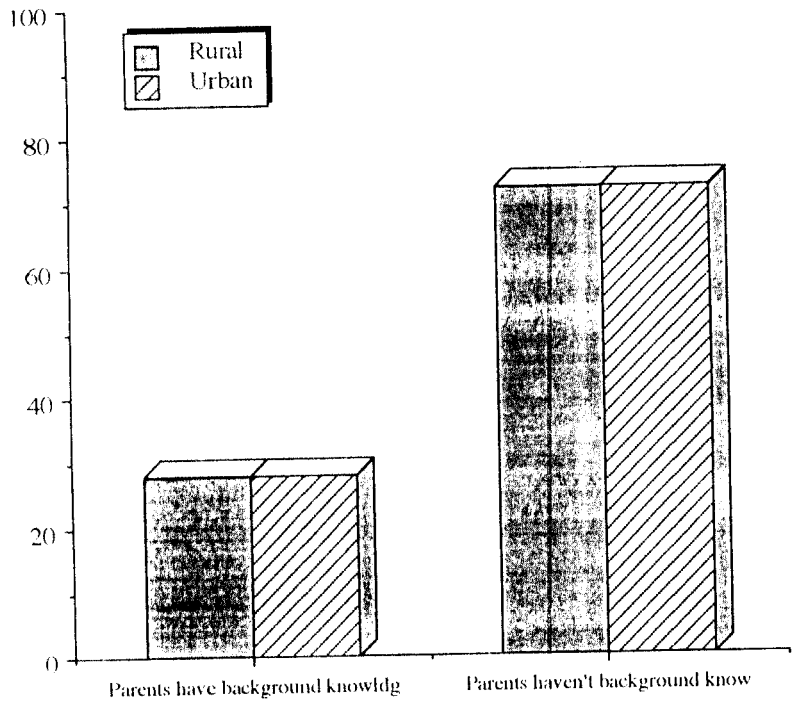

Fig. (16) : Incidence of caustic oesophageal stricture in relation to the baekground knowldge of the parents about similar conditions.

Table (18) : Incidence of oesophageal stricture occurance in relation to father's level of education.

	Hight level		Mid level		Low level		Illuterate	
	No	%	No	%	No	%	No	%
Rural	-	-	14	28%	14	28%	22	44%
Urban	4	8 %	15	30%	14	28%	17	34%
Total	4	4 %	29	29%	28	28%	39	39%

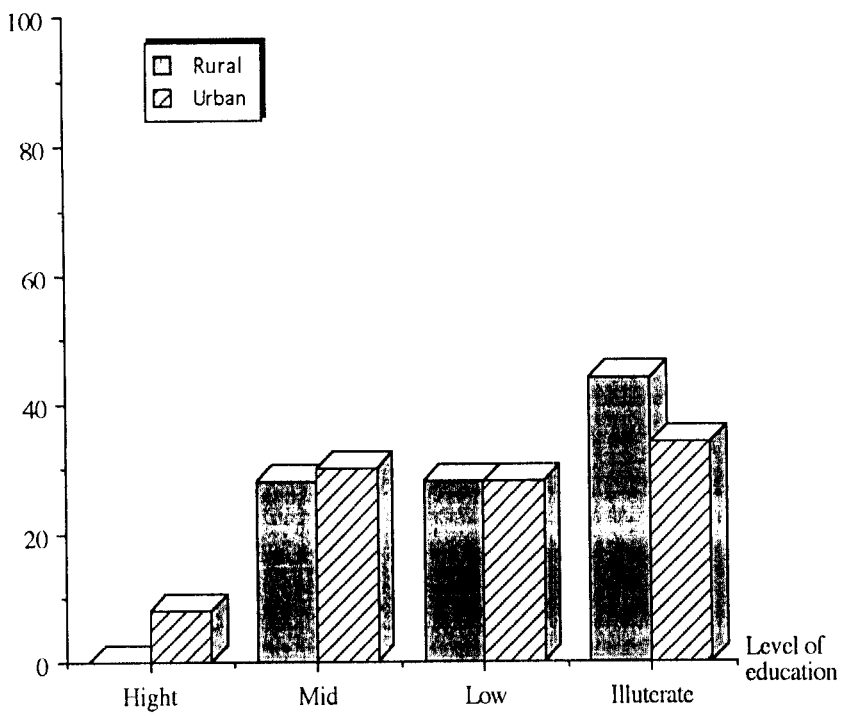

Fig. (17) : Incidence of oesophageal stricture occurance in relation to father's level of education.

Table (19) : Incidence of caustic oesophageal stricture occurance in relation to the mother's profession

	House wife		Private work		Public work	
	No	%	No	%	No	%
Rural	14	28%	1	2%	35	70%
Urban	12	24%	5	10%	33	66%
Total	26	26%	6	6%	68	68%

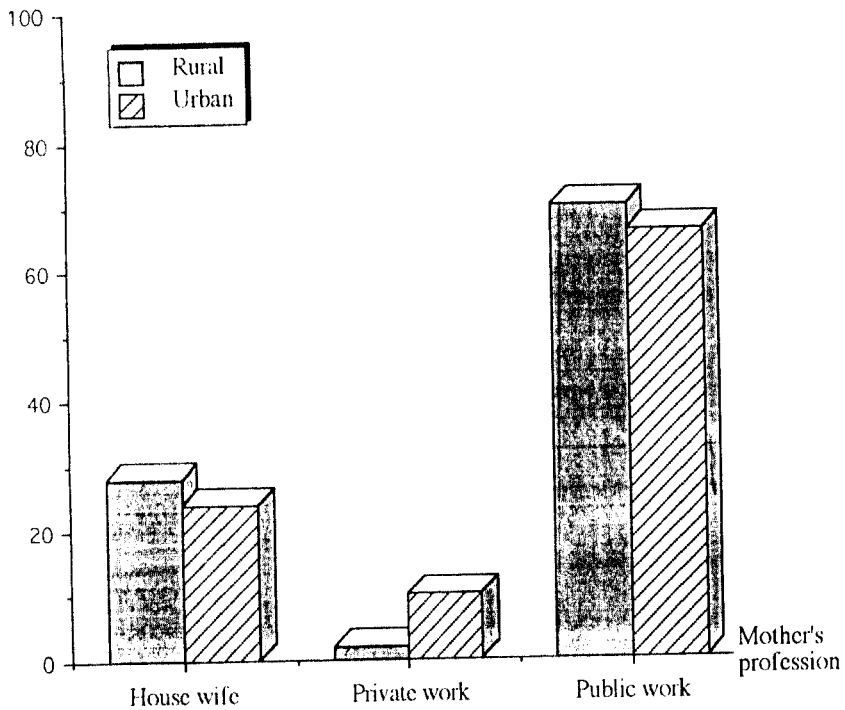

Fig. (18) : Incidence of caustic oesophageal stricture occurance in relation to the mother's profession

Table (20) : Incidence of caustic oesophageal stricture occurance to the Economic level of the family in Egyptian pounds (EP).

	Hight > 300		Moderate upto 300		Low upto 200		Poor upto 150	
	No	%	No	%	No	%	No	%
Rural	5	10%	19	38%	8	16%	18	36%
Urban	9	18%	20	40%	18	36%	3	6%
Total	14	14%	39	39%	26	26%	21	21%

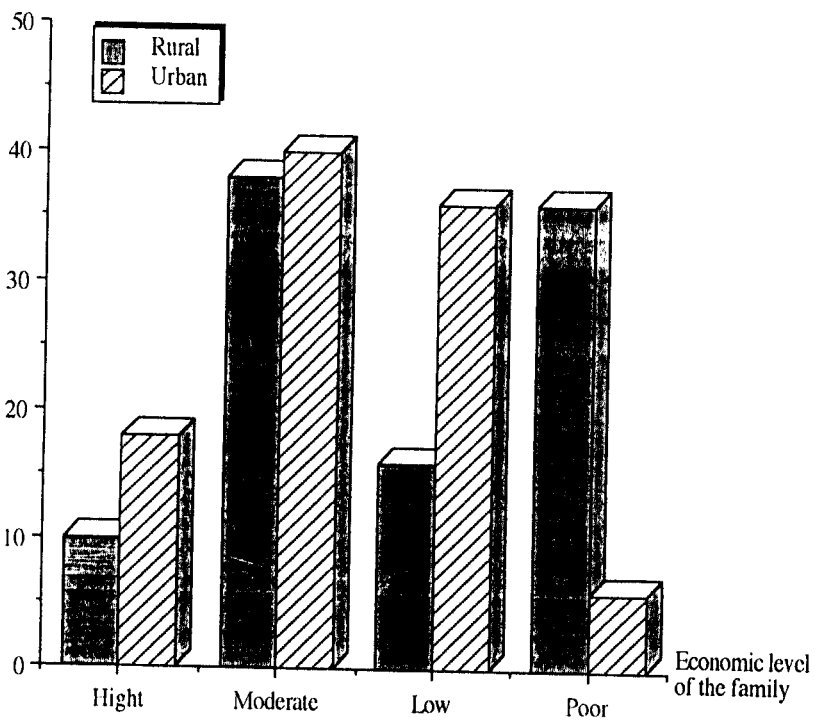

Fig. (19) : Incidence of caustic oesophageal stricture occurance to the Economic level of the family in Egyptian pounds (EP).

Table (21) : Incidence of caustic oesophageal stricture occurance in relation to the numbers of the children in the family.

	Single		up to 3 chidren		More than 3 children	
	No	%	No	%	No	%
Rural	2	4%	17	34%	31	62%
Urban	4	8%	13	26%	33	66%
Total	6	6%	30	30%	64	64%

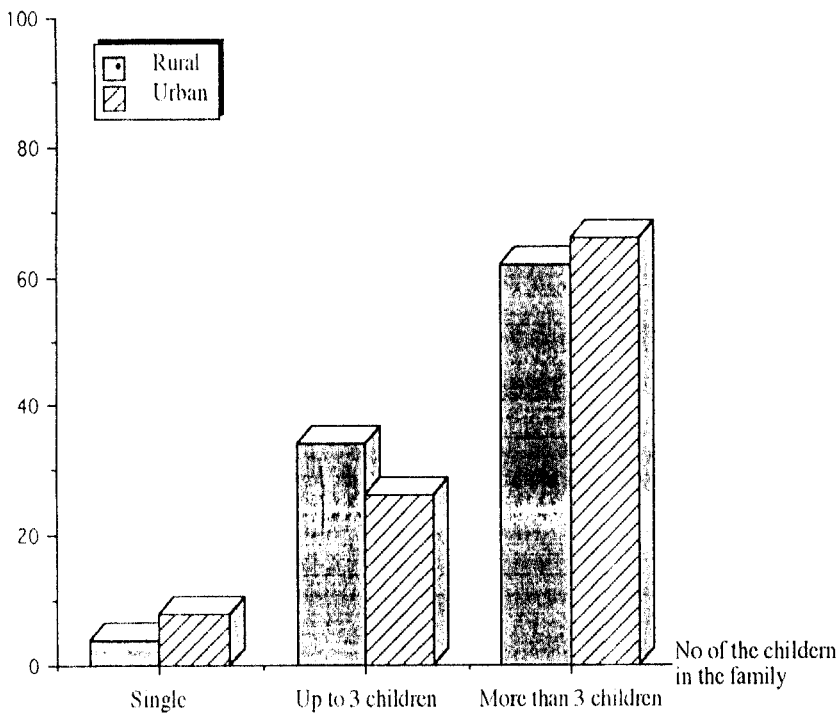

Fig. (20) : Incidence of caustic oesophageal stricture occurance in relation to the numbers of the children in the family.

Table (22) : Incidence of caustic oesophageal stricture occurance in relation to the siborder of the injured child

	The first off spring		The second off spring		Late in the of the family	
	No	%	No	%	No	%
Rural	3	6%	21	42%	26	52%
Urban	7	14%	19	38%	24	48%
Total	10	10%	40	40%	50	50%

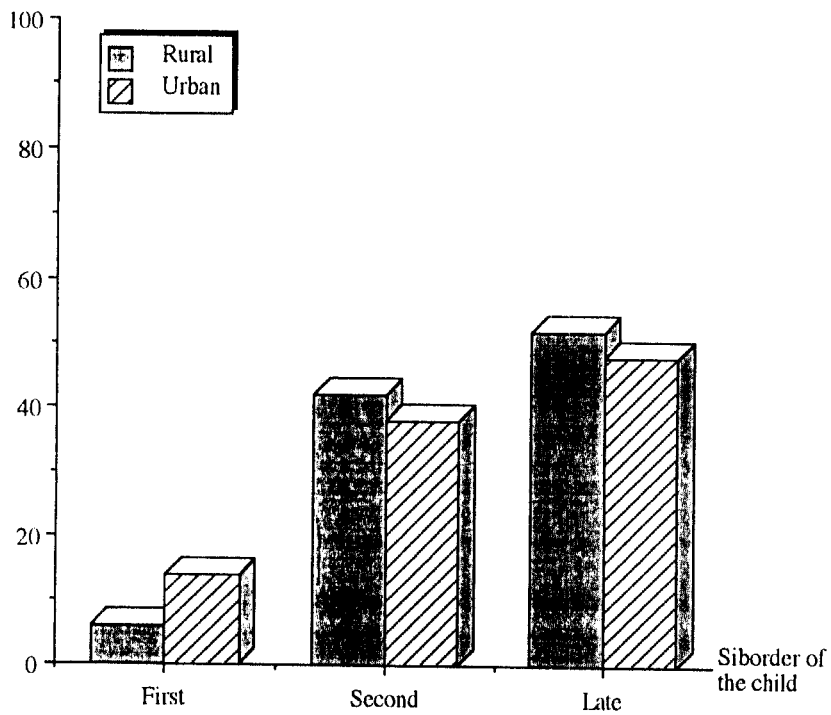

Fig. (21) : Incidence of caustic oesophageal stricture occurance in relation to the siborder of the injured child

Table (23 A) : Incidence of caustic aesophageal stricture occurance in relation to the social state of the family.

	Rural		Urban		Total	
	No	%	No	%	No	%
Father & Mother together	43	86%	36	72%	79	79%
Divorce	2	4%	5	10%	7	7%
Father dead	1	2%	-	-	1	1%
Mother dead	1	2%	1	2%	2	2%
Father & Mother dead	1	2%	1	2%	2	2%
Father abroad	2	4%	3	6%	5	5%
Mother abroad	-	-	1	2%	1	1%
Father in preson	-	-	2	4%	2	2%
Mother in preson	-	-	1	2%	1	1%

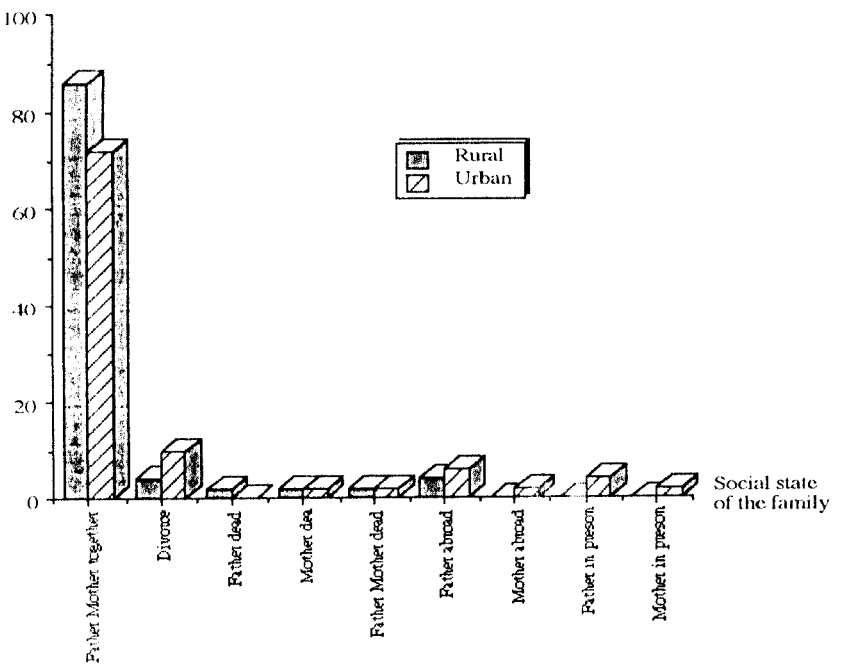

Fig. (22 A) : Incidence of caustic oesophageal stricture occurance in relation to the social state of the family.

Table (23 B) : Incidence of caustic aesophageal stricture occurance in relation to the social state of the family.

	Rural		Urban		Total	
	No	%	No	%	No	%
Child at work nursey	11	22%	9	18%	20	20%
Child at private nursery	6	12%	10	20%	16	16%
Child with relatives or Neighbours during absence of the family.	19	38%	17	34%	36	36%
Child with his family at home	14	28%	17	28%	28	28%

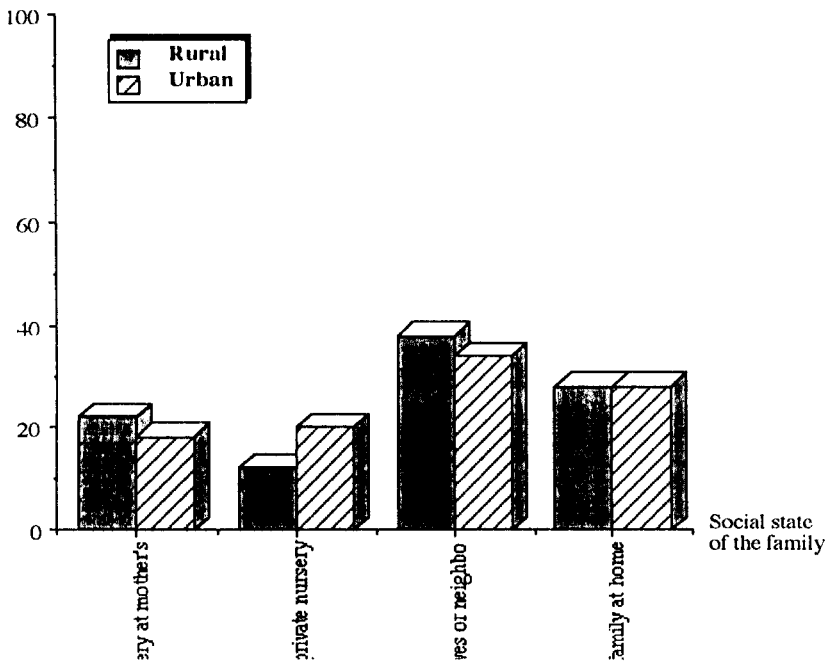

Fig. (22 B) : Incidence of caustic oesophageal stricture occurance in relation to the social state of the family.

II. Pathological study of corrosive oesophageal stricture.

Table (24) : Incidence of the degree of oesaphageal injury in recent injuries according to endoscopic finding.

	Rural		Urban		Total	
	No	%	No	%	No	%
I only segment less than 5 cm and superfascial	6	20.0%	5	16.7%	11	18.3%
II Affected segment more than 5 cm and superficial	16	53.3%	15	50.0%	31	51.68%
III Deep penetration	3	10.0%	5	13.3%	7	11.7%
IV Accompanied perforation and Mediastinits	5	16.7%	5	20.0%	11	18.3%
Total	30	100%	30	100%	60	100%

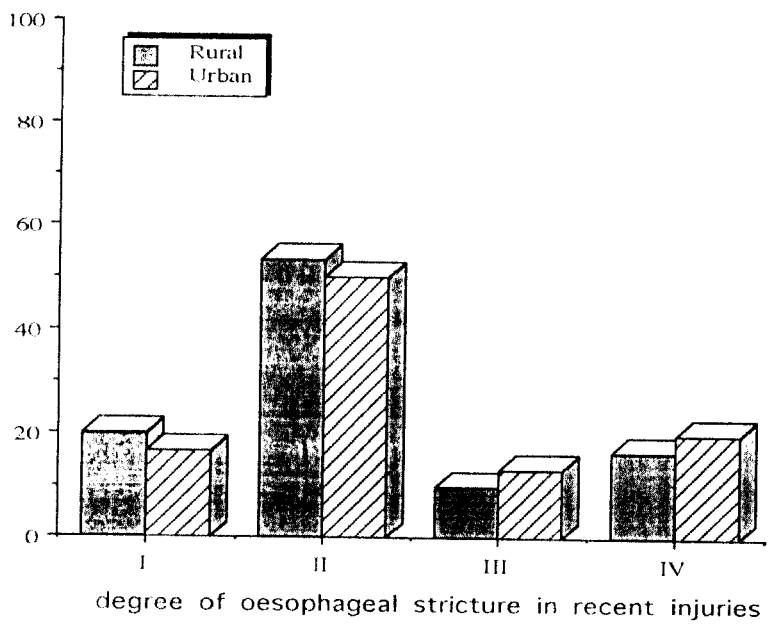

Fig. (23) : Incidence of the degree of oesophageal stricture in recent injuries according to endoscopic finding.

Table (25) : Incidence of the degree of oesophageal stricture in old injuies according to endoscopic finding

Degree	Rural		Urban		Total	
	No	%	No	%	No	%
I Passable short segment < 5	6	30%	3	15%	9	22.5%
II Passable long segment > 5	5	25%	6	30%	11	27.5%
III Impassable short segment < 5	5	25%	7	35%	12	30%
IV Impassable long segment > 5	4	20%	4	20%	8	20%
Total	20		20		40	110%

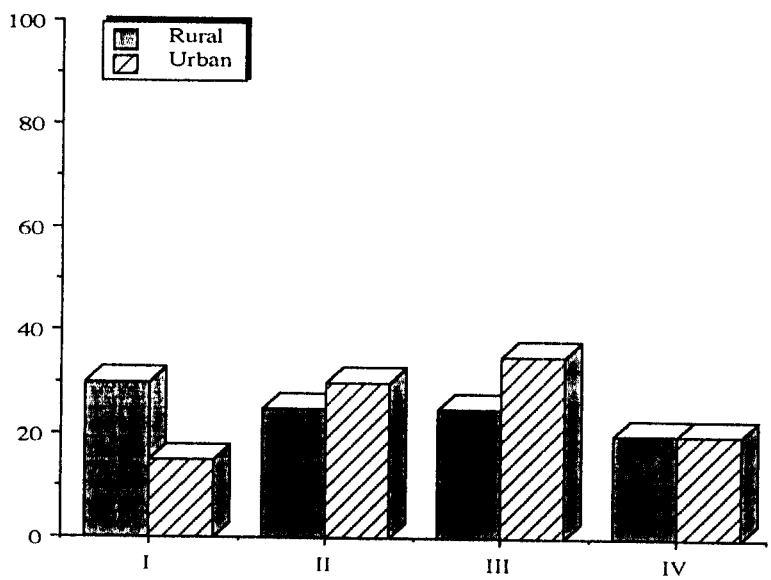

Fig. (24) : Incidence of the degree of oesophageal stricture in old injuies according to endoscopic finding.

Table (26) : Incidence of caustic oesophogeal strictrue in relation to the degree of Dysphagea after establishing the stricture in the 100 patient

Degree	Rural		Urban		Total	
	No	%	No	%	No	%
I Dysphagea to solid	8	16%	4	8%	12	12%
II Dysphagea to semi-solid	19	38%	18	36%	37	37%
III Dysphagea to fliud	10	20%	14	28%	24	24%
IV Dysphagea with regurgetation of saliva	13	26%	14	28%	27	27%
Total	50	100%	50	100%	100	100%

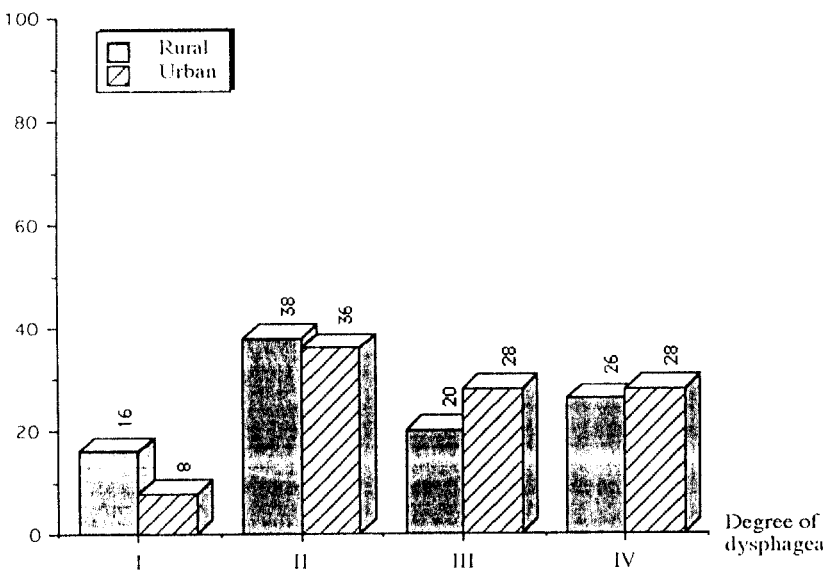

Fig. (25) : Incidence of caustic oesophogeal strictrue in relation to the degree of Dysphagea after establishing the stricture in the 100 patient

Table (27) : Incidence and cause of death in caustic oesophageal.

	Rural 50 patients	Urban 50 patients	Total 100 patients
	No	No	No
A. In recent injury (60 cases)			
1. due to chest complication	3	2	5
2. due to Metabolic disturbance	1	1	2
Total	(4)	(3)	(7)
B. In old injury (40 cases)			
1. due to Malnutrition		1	1
2. due to surgery			
- haemorrhage	1	1	2
- chest infections	1	1	2
- chest fistula	1	1	2
Total	(3)	(4)	(7)

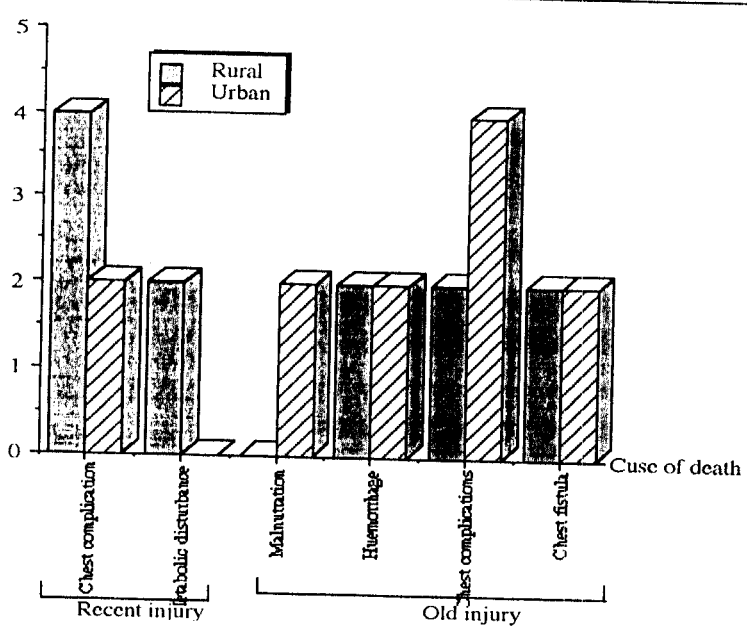

Fig. (26) : Incidence and cause of death in caustic oesophageal injuies.

III. Study the effect of cortisone and endoscopic dilatation on the oesophageal injuries in recent cases.

Table (28) : Effect of cortisone and endoscopic dilatation on the oesophageal injuries in recent cases.

Follow up	Rural				Urban				Total			
	Dilatation + cortisone		No dilatation No cortisone old cases		Dilatation + cortisone		No dilatation No cortisone old cases		Dilatation + cortisone		No dilatation No cortisone old cases	
	No	%	No	%	No	%	No	%	No	%	No	%
- Perforation with dilatation	1	3.3%	-	-	2	6.67%	-	-	3	5%	-	-
- No improvement	5	16.7%	12	60%	6	20%	13	65%	11	18.3%	25	62.5%
- Mild improvement	3	10.0%	5	25%	3	10%	5	25%	6	10%	10	25%
- Moderate improvement	6	20.0%	3		4	13.33%	2	10%	10	16.7%	5	12.5%
- Good improvent	15	50.0%	-	15%	15	50%	-		30	50%	-	-
Total	30	100%	20	100%	30	100%	20	100%	60	100%	40	100%

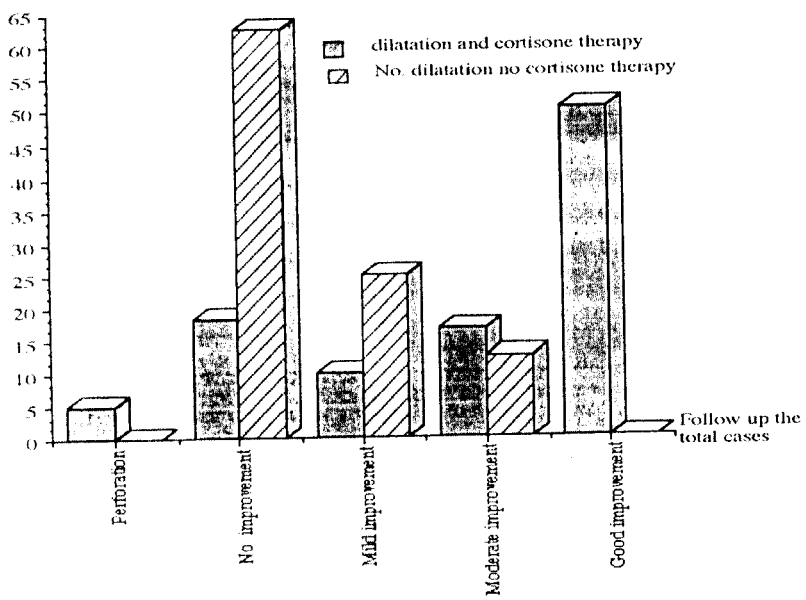

Fig. (27) : Effect of cortisone and endoscopic dilatation on the oesophageal injuries in recent cases.

IV. Anthropometric study of growth and development of the affected children :

Table (29) : Follow up the child weight in the rural locality patients in kilogramms.

No	Sex	Age	Stage	1 st examination		1 M		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F	N	F
1	♂	2	II	12	12	12	7	13	9	14	10	16	12	20	12
2	♂	3	II	14	13	14	5	15	12	16	14	18	16	22	19
3	♀	2 1/2	II	13	13	13	7	14	5	15	7	Death	Death	Death	Death
4	♂	1 1/2	III	11	10	11	5	12	4	13	5	15	7	19	10
5	♀	4	III	16	15	16	10	17	7	18	9	20	10	Death	Death
6	♂	3	II	14	14	14	10	15	10	16	15	18	16	22	18
7	♂	2	II	12	12	12	7	13	5	14	6	16	10	20	16
8	♀	3 1/2	I	15	13	15	10	16	12	17	15	19	19	23	22
9	♂	1 1/2	I	11	10	11	10	12	12	13	15	15	15	19	18
10	♀	3	II	14	14	14	10	15	10	16	12	18	15	22	18
11	♀	4	II	16	15	16	9	17	12	18	16	20	18	24	22
12	♂	2 1/2	I	13	13	13	10	15	12	15	14	17	16	21	19
13	♂	6 1/2	III	21	20	21	5	22	7	23	10	25	12	28	15
14	♂	2	IV	12	10	12	6	13	5	13	6	Death	Death	Death	Death
15	♂	4	III	16	15	16	7	17	8	18	16	20	8	24	10
16	♂	2 1/2	II	13	13	13	8	14	8	15	7	17	8	21	10
17	♂	3 1/2	II	15	17	15	12	16	10	17	10	19	12	23	15
18	♂	1 1/2	III	11	10	11	6	12	5	13	6	15	8	19	10
19	♂	2	IV	12	11	12	5	13	4	14	5	Death	Death	Death	Death
20	♀	2	I	12	12	12	10	13	11	14	12	16	15	20	20
21	♂	3 1/2	II	15	14	15	10	16	9	17	12	19	15	23	20
22	♂	5	II	18	17	18	12	19	10	20	12	22	17	26	22
23	♂	6	I	20	18	20	14	21	15	22	19	24	22	27	25
24	♂	2 1/2	II	13	12	13	7	14	7	15	9	17	10	21	14
25	♂	4	II	16	15	16	10	17	9	18	10	20	12	24	17
26	♀	2	II	12	11	12	6	13	6	14	7	16	9	20	12
27	♀	1 1/2	IV	11	10	11	5	12	4	13	5	15	5	19	7
28	♀	2	I	12	12	12	9	13	9	14	12	16	15	20	17
29	♀	3	IV	14	13	14	5	15	5	16	6	18	7	22	7
30	♀	2	I	12	11	12	9	13	9	14	10	16	12	20	15

N : Standard weight of Normal child in the same age and sex.
F : Follwo up weight of diseased child.

Table (30) : Follow up the child weight in the urban locality patients in kilogramms.

No	Sex	Age	Stage	1 st examination		1 M		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F	N	F
1	♀	2 1/2	II	13	14	13	9	14	10	15	12	17	13	21	14
2	♂	6	III	20	20	20	10	21	8	22	9	24	10	27	16
3	♀	2	II	12	12	12	6	13	7	14	8	16	10	20	12
4	♂	2 1/2	II	13	13	13	8	14	7	15	9	17	12	21	15
5	♂	4	I	16	15	16	10	17	15	18	16	20	20	24	23
6	♂	3 1/2	I	19	13	15	8	16	9	17	12	19	15	23	22
7	♂	3	II	14	12	14	8	15	8	16	9	18	10	22	15
8	♀	1 1/2	IV	11	12	11	6	12	5	13	5	15	5	Death	Death
9	♀	4	I	16	15	16	10	17	10	18	14	20	16	24	18
10	♂	2	II	12	12	12	9	13	8	14	10	16	13	20	15
11	♂	1 1/2	III	11	10	11	4	12	4	13	5	15	6	19	7
12	♀	2	II	12	12	12	7	13	5	14	6	16	7	20	10
13	♂	3	II	14	13	14	6	15	5	16	6	18	8	22	12
14	♂	2 1/2	II	13	12	13	6	14	6	15	5	17	7	21	12
15	♂	3	II	14	13	14	7	15	8	16	8	18	10	22	14
16	♀	2 1/2	II	13	12	13	6	14	5	15	6	17	7	21	13
17	♂	3 1/2	I	15	14	15	10	16	12	17	14	19	16	23	18
18	♂	2	II	12	12	12	9	13	9	14	9	16	12	20	15
19	♂	2 1/2	II	13	12	13	8	14	9	15	9	17	11	21	15
20	♀	2 1/2	IV	13	12	13	4	14	4	15	5	17	6	21	8
21	♀	3	II	14	14	14	6	15	8	16	9	18	12	22	15
22	♂	4	III	16	15	16	7	17	7	18	6	20	8	24	10
23	♀	2	IV	12	12	12	5	13	5	14	4	16	5	20	7
24	♂	3	II	14	14	14	7	15	7	16	9	18	9	22	15
25	♀	3	IV	14	14	14	6	15	5	16	5	18	7	Death	Death
26	♂	3	II	14	15	14	10	15	10	16	12	18	13	22	15
27	♀	4 1/2	III	17	16	17	7	18	6	19	7	21	10	Death	Death
28	♀	6	II	20	20	20	12	21	12	22	13	24	14	27	17
29	♀	3	II	14	14	14	7	15	8	16	8	18	9	22	12
30	♂	4	III	16	16	16	5	17	5	18	5	20	8	24	10

N : Standard weight of Normal child in the same age and sex.
F : Follwo up weight of diseased child.

Table (31) : Follow up the nutritional state of post corrosive aesophageal stricture in relation to surface urea M2 in rural patients.

No	Sex	Age	Stage	1st examination		1 M		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F	N	F
1	♂	2	II	0.56	0.56	0.56	0.38	0.59	0.46	0.62	0.50	0.68	0.56	0.80	0.56
2	♂	3	II	0.62	0.59	0.62	0.30	0.65	0.56	0.68	0.62	0.74	0.68	0.84	0.77
3	♀	2 1/2	II	0.59	0.59	0.59	0.38	0.62	0.30	0.65	0.38	Death	Death	Death	Death
4	♂	2 1/2	III	0.53	0.50	0.53	0.30	0.56	0.25	0.59	0.30	0.65	0.38	0.77	0.50
5	♀	4	III	0.68	0.65	0.68	0.50	0.71	0.38	0.74	0.46	0.80	0.50	Death	Death
6	♂	3	II	0.62	0.62	0.62	0.50	0.65	0.50	0.68	0.65	0.74	0.68	0.84	0.74
7	♂	2	II	0.56	0.56	0.56	0.38	0.59	0.30	0.62	0.34	0.68	0.50	0.80	0.68
8	♀	3 1/2	I	0.65	0.59	0.65	0.50	0.68	0.56	0.71	0.65	0.77	0.77	0.86	0.84
9	♂	1 1/2	I	0.53	0.50	0.53	0.50	0.56	0.56	0.59	0.65	0.65	0.65	0.77	0.74
10	♀	3	II	0.62	0.62	0.62	0.50	0.65	0.50	0.68	0.56	0.71	0.65	0.84	0.74
11	♀	4	II	0.68	0.65	0.68	0.46	0.71	0.56	0.74	0.68	0.80	0.74	0.88	0.84
12	♂	2 1/2	I	0.59	0.59	0.59	0.50	0.65	0.56	0.65	0.62	0.71	0.68	0.82	0.77
13	♂	6 1/2	III	0.82	0.80	0.82	0.30	0.84	0.38	0.86	0.50	0.90	0.59	0.96	0.65
14	♂	2	IV	0.56	0.50	0.56	0.34	0.59	0.30	0.59	0.34	Death	Death	Death	Death
15	♂	4	III	0.68	0.65	0.68	0.38	0.71	0.42	0.74	0.68	0.80	0.42	0.88	0.50
16	♂	2 1/2	II	0.59	0.59	0.59	0.42	0.62	0.42	0.65	0.38	0.71	0.42	0.82	0.50
17	♂	3 1/2	II	0.65	0.71	0.65	0.56	0.68	0.50	0.71	0.50	0.77	0.56	0.86	0.65
18	♂	1 1/2	III	0.53	0.50	0.53	0.34	0.56	0.30	0.59	0.34	0.65	0.42	0.77	0.50
19	♂	2	IV	0.56	0.53	0.56	0.30	0.59	0.25	0.62	0.30	Death	Death	Death	Death
20	♀	2	I	0.56	0.56	0.56	0.50	0.59	0.53	0.62	0.56	0.68	0.65	0.80	0.80
21	♂	3 1/2	II	0.65	0.62	0.65	0.50	0.68	0.46	0.71	0.56	0.77	0.65	0.86	0.80
22	♂	5	II	0.74	0.71	0.74	0.56	0.77	0.50	0.80	0.56	0.84	0.71	0.92	0.84
23	♂	6	I	0.80	0.74	0.80	0.62	0.82	0.65	0.84	0.77	0.88	0.84	0.94	0.90
24	♂	2 1/2	II	0.59	0.56	0.59	0.38	0.62	0.38	0.65	0.46	0.71	0.50	0.82	0.62
25	♂	4	II	0.68	0.65	0.68	0.50	0.71	0.46	0.74	0.50	0.80	0.56	0.88	0.71
26	♀	2	II	0.56	0.53	0.56	0.34	0.59	0.34	0.62	0.38	0.68	0.46	0.80	0.56
27	♀	1 1/2	IV	0.53	0.50	0.53	0.30	0.56	0.25	0.59	0.30	0.65	0.30	0.77	0.38
28	♀	2	I	0.56	0.56	0.56	0.46	0.59	0.46	0.62	0.56	0.68	0.65	0.80	0.71
29	♀	3	IV	0.62	0.59	0.62	0.30	0.65	0.30	0.68	0.34	0.74	0.38	0.84	0.38
30	♀	2	I	0.56	0.53	0.56	0.46	0.59	0.46	0.62	0.50	0.68	0.56	0.80	0.65

N : Standard surface area of Normal child in same age and sex.
F : Follwo up surface are of the diseased child.

Measurments of Nutrational state (surface area) "M2" from body weight in normal child

Kg body weight	M2	Kg body weight	M2	Kg body weight	M2
1	0.10	11	0.53	21	0.82
2	0.15	12	0.56	22	0.84
3	0.20	13	0.59	23	0.68
4	0.25	14	0.62	24	0.88
5	0.30	15	0.65	25	0.90
6	0.34	16	0.68	26	0.92
7	0.38	17	0.71	27	0.94
8	0.42	18	0.74	28	0.96
9	0.46	19	0.77	29	0.98
10	0.50	20	0.80	30	1.00

Table (32) : Follow up the nutirtional state of post corrosine aesophazeal stricture in relation to surface area M2 in urban patients.

No	Sex	Age	Stage	1 st examination		1 M		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F	N	F
1	♀	2 1/2	II	0.59	0.62	0.59	0.46	0.62	0.50	0.65	0.56	0.71	0.59	0.82	0.62
2	♂	6	III	0.80	0.80	0.80	0.50	0.82	0.42	0.84	0.46	0.88	0.50	0.94	0.68
3	♀	2	II	0.56	0.56	0.56	0.34	0.59	0.38	0.62	0.42	0.68	0.50	0.80	0.56
4	♂	2 1/2	II	0.59	0.59	0.59	0.42	0.62	0.38	0.56	0.46	0.71	0.56	0.82	0.65
5	♂	4	I	0.68	0.65	0.68	0.50	0.71	0.56	0.74	0.68	0.80	0.80	0.88	0.86
6	♂	3 1/2	I	0.65	0.59	0.65	0.42	0.68	0.46	0.71	0.56	0.77	0.65	0.86	0.84
7	♂	3	II	0.62	0.56	0.62	0.42	0.65	0.42	0.68	0.46	0.74	0.50	0.84	0.65
8	♀	1 1/2	IV	0.53	0.56	0.53	0.34	0.56	0.30	0.59	0.30	0.65	0.30	Death	Death
9	♀	4	I	0.68	0.65	0.68	0.50	0.71	0.50	0.74	0.62	0.80	0.68	0.88	0.74
10	♂	2	II	0.56	0.56	0.56	0.46	0.59	0.42	0.62	0.50	0.68	0.59	0.80	0.65
11	♂	1 1/2	III	0.53	0.50	0.53	0.25	0.56	0.25	0.59	0.30	0.65	0.34	0.77	0.38
12	♀	2	II	0.56	0.56	0.56	0.38	0.59	0.30	0.62	0.34	0.68	0.38	0.80	0.50
13	♂	3	II	0.62	0.59	0.62	0.34	0.65	0.30	0.68	0.34	0.74	0.42	0.84	0.56
14	♂	2 1/2	II	0.59	0.56	0.59	0.34	0.62	0.34	0.65	0.30	0.71	0.38	0.82	0.56
15	♂	3	II	0.62	0.59	0.62	0.38	0.65	0.42	0.68	0.42	0.74	0.50	0.84	0.62
16	♀	2 1/2	II	0.59	0.56	0.59	0.34	0.62	0.30	0.65	0.34	0.71	0.38	0.82	0.59
17	♂	3 1/2	I	0.65	0.62	0.65	0.50	0.68	0.56	0.71	0.62	0.77	0.68	0.86	0.74
18	♂	2	II	0.56	0.56	0.56	0.46	0.59	0.46	0.62	0.46	0.68	0.56	0.80	0.65
19	♂	2 1/2	II	0.59	0.56	0.59	0.42	0.62	0.46	0.56	0.46	0.71	0.53	0.82	0.65
20	♀	2 1/2	IV	0.59	0.56	0.59	0.25	0.62	0.25	0.65	0.30	0.71	0.34	0.82	0.42
21	♀	3	II	0.62	0.62	0.62	0.34	0.65	0.42	0.68	0.46	0.74	0.56	0.84	0.65
22	♂	4	III	0.68	0.65	0.68	0.38	0.71	0.38	0.74	0.34	0.80	0.42	0.88	0.50
23	♀	2	IV	0.56	0.56	0.56	0.30	0.59	0.30	0.62	0.25	0.68	0.30	0.80	0.38
24	♂	3	II	0.62	0.62	0.62	0.38	0.65	0.38	0.68	0.46	0.74	0.46	0.84	0.65
25	♀	3	IV	0.62	0.62	0.62	0.34	0.65	0.30	0.68	0.30	0.74	0.38	Death	Death
26	♂	3	II	0.62	0.65	0.62	0.50	0.65	0.50	0.68	0.56	0.74	0.59	0.84	0.65
27	♀	4 1/2	III	0.71	0.68	0.71	0.38	0.74	0.34	0.77	0.38	0.82	0.50	Death	Death
28	♀	6	II	0.80	0.80	0.80	0.56	0.82	0.56	0.84	0.59	0.88	0.62	0.94	0.71
29	♀	3	II	0.62	0.62	0.62	0.38	0.65	0.42	0.68	0.42	0.74	0.46	0.84	0.56
30	♂	4	III	0.68	0.68	0.68	0.30	0.71	0.30	0.74	0.30	0.80	0.42	0.88	0.50

N : Standard surface area of Normal child in same age and sex
F : Follwo up surface are of the diseased child

Measurments of Nutrational state (surface area) "M2" from body weight in normal child

Kg body weight	M2	Kg body weight	M2	Kg body weight	M2
1	0.10	11	0.53	21	0.82
2	0.15	12	0.56	22	0.84
3	0.20	13	0.59	23	0.68
4	0.25	14	0.62	24	0.88
5	0.30	15	0.65	25	0.90
6	0.34	16	0.68	26	0.92
7	0.38	17	0.71	27	0.94
8	0.42	18	0.74	28	0.96
9	0.46	19	0.77	29	0.98
10	0.50	20	0.80	30	1.00

Table (33) : Follow up the child length in the rural locality patients in centemeters.

No	Sex	Age	Stage	1 st examination		1 M		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F	N	F
1	♂	2	II	85	85	85	85	90	86	95	89	100	90	112	92
2	♂	3	II	85	80	95	82	97	82	100	85	105	90	120	95
3	♀	2 1/2	II	90	82	90	82	95	82	97	84	Death	Death	Death	Death
4	♂	2 1/2	III	80	78	80	78	87	80	90	80	97	80	107	82
5	♀	4	III	100	95	100	95	102	96	105	98	112	100	Death	Death
6	♂	3 1/2	II	97	95	97	95	100	95	102	96	107	98	130	100
7	♂	2	II	85	80	85	80	90	80	95	84	100	86	112	90
8	♀	3 1/2	I	97	95	97	95	100	95	102	100	107	105	125	115
9	♂	1 1/2	I	80	75	80	80	87	90	90	90	97	90	107	105
10	♀	3	II	95	95	95	95	97	96	100	97	105	100	120	105
11	♀	4	II	100	96	100	96	102	96	105	96	112	100	130	105
12	♂	2 1/2	I	90	95	90	95	95	95	97	95	102	100	115	105
13	♂	6 1/2	III	115	110	115	110	120	110	125	110	135	112	150	115
14	♂	2	IV	85	85	85	85	90	85	95	85	Death	Death	Death	Death
15	♂	3 1/2	III	97	95	97	95	100	95	102	95	107	96	125	95
16	♂	2 1/2	II	90	89	90	89	95	89	97	90	102	92	115	95
17	♂	3 1/2	II	97	99	97	99	100	99	102	100	107	102	125	103
18	♂	1 1/2	III	80	75	80	75	87	75	90	76	97	78	107	83
19	♂	2	IV	87	85	87	85	90	85	95	86	Death	Death	Death	Death
20	♀	2	I	87	85	87	85	90	86	95	188	100	95	112	110
21	♂	3 1/2	II	97	95	97	45	100	48	102	97	107	100	125	110
22	♂	5	II	105	100	105	100	107	101	112	105	120	110	130	115
23	♂	6	I	112	105	112	105	115	108	120	110	130	115	145	120
24	♂	2 1/2	II	90	82	90	87	95	87	97	90	102	95	115	100
25	♂	4	II	100	95	100	95	102	95	105	97	112	100	130	110
26	♀	2	II	85	82	85	82	90	82	95	85	97	90	112	95
27	♀	1 1/2	III	80	75	80	75	87	75	90	75	97	80	107	84
28	♀	2	I	87	85	87	85	90	87	95	90	97	95	112	100
29	♀	3	IV	95	95	95	95	97	90	100	95	105	95	120	98
30	♀	2	I	87	84	81	84	90	84	95	85	97	86	112	100

N : Standard length of Normal child in same age and sex.
F : Follwo length up of the diseased child.

Table (34) : Follow up the child length in the urban locality patients in centemeters

No	Sex	Age	Stage	1 st examination		1 M		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F	N	F
1	♀	2 1/2	II	90	90	90	90	95	92	97	95	102	97	115	105
2	♂	6	III	112	110	112	110	115	110	120	110	125	115	140	120
3	♀	2	II	87	87	87	87	90	89	95	92	100	94	112	100
4	♂	2 1/2	II	90	85	90	85	95	85	97	85	102	90	115	95
5	♂	4	I	100	100	100	100	102	100	105	105	112	110	130	116
6	♂	3 1/2	I	97	93	91	93	100	94	102	96	107	100	125	105
7	♂	3	II	85	95	85	95	97	95	100	98	105	100	120	105
8	♀	1 1/2	IV	80	85	80	85	87	85	90	85	97	85	Death	Death
9	♀	4	I	100	100	100	100	102	102	105	105	112	110	130	115
10	♂	2	II	87	85	87	85	90	87	95	90	100	95	112	105
11	♂	1 1/2	III	80	75	80	75	87	95	90	76	97	77	107	80
12	♀	2	II	87	85	87	85	90	85	95	87	100	89	112	95
13	♂	3	II	95	90	95	90	97	90	100	92	105	95	120	100
14	♂	2 1/2	II	90	85	90	85	95	85	97	87	102	90	115	95
15	♂	3	II	85	90	85	90	97	90	100	92	105	95	120	100
16	♀	2 1/2	II	90	87	90	87	95	89	97	89	102	92	115	95
17	♂	3 1/2	I	97	95	97	95	100	97	102	100	107	105	125	110
18	♂	2	II	87	87	87	87	90	88	95	90	100	94	112	100
19	♂	2 1/2	II	90	85	90	85	92	87	95	89	97	92	115	95
20	♀	2 1/2	IV	90	85	90	85	92	85	95	85	97	87	115	90
21	♀	3	II	85	95	85	95	97	95	100	97	105	100	120	105
22	♂	4	III	100	100	100	100	102	100	105	102	112	104	130	107
23	♀	2	IV	87	85	87	85	90	85	95	85	92	85	112	87
24	♂	3	II	95	95	95	95	97	97	100	100	105	105	120	110
25	♀	3	IV	95	95	95	95	97	95	100	97	105	100	Death	Death
26	♂	3	II	95	95	95	95	97	97	100	100	105	102	120	110
27	♀	4 1/2	III	102	100	102	100	105	100	107	100	115	102	Death	Death
28	♀	6	II	112	110	112	110	115	110	120	112	130	114	145	118
29	♀	3	II	95	95	95	95	97	95	100	96	105	98	120	105
30	♂	4	III	100	100	100	100	102	100	102	102	112	104	130	107

N : Standard length of Normal child in same age and sex.
F : Follwo length up of the diseased child.

Table (35) : Follow up the head circumference in the rural locality patients.

No	Sex	Stage	Age	1st Examination		6 M		1 year	
				N	F	N	F	N	F
1	♂	II	2	48.3	48.0	49.0	48.9	49.8	49.5
2	♂	II	3	49.8	49.9				
3	♀	II	2 1/2	49.0	49.1	49.8	49.7		
4	♂	III	1 1/2	47.6	47.0	48.3	48.1	49.0	49.0
5	♀	III	4						
6	♂	II	3	49.8	49.7				
7	♂	II	2	48.3	48.4	49.0	49.0	49.8	49.7
8	♀	II	3 1/2						
9	♂	I	1 1/2	47.6	47.5	48.3	48.2	49.0	49.0
10	♀	II	3	49.8	49.7				
11	♀	II	4						
12	♂	I	2 1/2	49.0	49.0	49.8	49.7		
13	♂	III	6 1/2						
14	♂	IV	2	48.3	48.4	49.0	48.9	49.8	49.7
15	♂	III	4						
16	♂	II	2 1/2	49.0	49.0	49.8	49.7		
17	♂	II	3 1/2	49.8	49.7				
18	♂	III	1 1/2	47.6	47.5	48.3	48.1	49.0	48.7
19	♂	IV	2	48.3	48.3	49.0	48.9	49.8	49.7
20	♀	I	2	48.3	48.3	49.0	49.0	49.8	49.8
21	♂	II	3 1/2	49.8	49.8				
22	♂	II	5	50.8	50.8				
23	♂	I	6						
24	♂	II	2 1/2	49.5	49.5	49.8	49.7		
25	♂	II	4						
26	♀	II	2	48.3	48.3	49.0	48.9	49.8	49.7
27	♀	IV	1 1/2	47.6	47.5	48.3	48.2	49.0	49.0
28	♀	I	2	48.3	48.2	49.0	49.0	49.8	49.7
29	♀	IV	3	49.8	49.8				
30	♀	I	2	48.3	48.3	49.0	48.9	49.8	49.7

N : Standard head circumference of normal child of same age and sex.
F : Follow up head circumference of the disease of child.
N.B. head circumference measurements is not a parameter of measurement of growth after the age of 3 years old.

Table (36) : Follow up the head circumference in the Urban locality patients.

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year	
				N	F	N	F	N	F	N	F
1	II	♀	2 1/2	49.0	49.0	49.8	49.7				
2	III	♂	6								
3	II	♀	2	48.3	48.2	49.0	49.0	49.8	49.7		
4	II	♂	2 1/2	49.0	48.9	49.8	49.7				
5	I	♂	4								
6	I	♂	3 1/2								
7	II	♂	3	49.8	49.7						
8	IV	♀	1 1/2	47.6	47.6	48.3	48.2	49.0	49.0	49.8	49.7
9	I	♀	4								
10	II	♂	2	48.3	48.2	49.0	49.0	49.8	49.7		
11	III	♂	1 1/2	47.6	47.5	48.3	48.2	49.0	49.0	49.8	49.7
12	II	♀	2	48.3	48.2	49.0	48.9	49.8	49.7		
13	II	♂	3	49.8	49.7						
14	II	♂	2 1/2	49.0	48.0	49.8	49.7				
15	II	♂	3	49.8	49.7						
16	II	♀	2 1/2	49.0	49.0	49.8	49.7				
17	I	♂	3 1/2								
18	II	♂	2	48.3	48.2	49.0	48.9	49.8	49.7		
19	II	♂	2 1/2	49.0	49.0	49.8	49.7				
20	IV	♀	2 1/2	49.0	49.0	49.8	49.7				
21	II	♀	3	49.8	49.7						
22	III	♂	4								
23	IV	♀	2	48.3	48.2	49.0	49.0	49.8	49.7		
24	II	♂	3	49.8	49.7						
25	IV	♀	3	49.8	49.8						
26	II	♂	3	49.8	49.9						
27	III	♀	4 1/2								
28	II	♀	6								
29	II	♀	3	49.8	49.7						
30	III	♂	4								

N : Standard head circumference of normal child in same age and sex.
F : Follow up head circumference of the disease of child.
N.B head circumference measurements is not a parameter of measurement of growth after the age of 3 years old.

Table (37) : Follow up the abdomen circumference in the rural locality patients.

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year	
				N	F	N	F	N	F	N	F
1	II	♂	2	51.5	51.5	52.0	52.0	52.7	52.7		
2	II	♂	3	52.7	52.7						
3	II	♀	2 1/2	52.0	52.0	52.7	52.6			Death	Death
4	III	♂	1 1/2	50.9	50.8	51.5	50.1	52.0	50.1	52.7	49.5
5	III	♀	4							Death	Death
6	II	♂	3	52.7	52.7						
7	II	♂	2	51.5	51.5	52.0	51.0	52.7	51.1		
8	I	♀	3 1/2								
9	I	♂	1 1/2	50.9	50.9	51.5	51.3	52.0	51.8	52.7	52.3
10	II	♀	3	52.7	52.6						
11	II	♀	4								
12	I	♂	2 1/2	52.0	52.0	52.7	52.2				
13	III	♂	6 1/2								
14	IV	♂	2	51.5	51.3	52.0	50.1	52.7	50.1	Death	Death
15	III	♂	4								
16	II	♂	2 1/2	52.0	52.0	52.7	52.6				
17	II	♂	3 1/2								
18	III	♂	1 1/2	50.9	50.8	50.5	50.7	52.0	50.6	52.7	50.1
19	IV	♂	2	51.5	51.3	52.0	50.8	52.7	50.6	Death	Death
20	I	♀	2	51.5	51.5	52.0	51.9	52.7	52.5		
21	II	♂	3 1/2								
22	II	♂	5								
23	I	♂	6								
24	II	♂	2 1/2	52.0	52.0	52.7	52.4				
25	II	♂	4								
26	II	♀	2	51.5	51.5	52.0	50.9	52.7	52.3		
27	IV	♀	1 1/2	50.9	50.8	51.5	50.2	52.0	50.1	52.7	50.1
28	I	♀	2	51.5	51.4	52.0	52.0	52.7	52.6		
29	IV	♀	3	52.7	52.6						
30	I	♀	2	51.5	51.5	52.0	52.0	52.7	52.6		

N : Standard abdomen circumference in normal child of same age and sex.
F : Follow up abdomen circumference of the diseased child.
N.B. abdomen circumference measurements is not a parameter of measurement of growth after the age of 3 years old.

Table (38) : Follow up the abdomen circumference in the Urban locality patients.

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year	
				N	F	N	F	N	F	N	F
1	II	♀	2 1/2	52.0	52.0	52.7	52.6				
2	III	♂	6								
3	II	♀	2	51.5	52.0	52.0	51.9	52.7	52.6		
4	II	♂	2 1/2	52.0		52.7	52.4				
5	I	♂	4								
6	I	♂	3 1/2								
7	II	♂	3	52.7	52.6						
8	IV	♀	1 1/2	50.9	50.8	51.5	50.7	52.0	50.6	52.7	50.6
9	I	♀	4								
10	II	♂	2	51.5	51.5	52.0	52.0	52.7	52.6		
11	III	♂	1 1/2	50.9	50.8	51.5	51.4	52.0	52.0	52.7	52.6
12	II	♀	2	51.5	51.4	52.0	51.8	52.7	52.6		
13	II	♂	3	52.7	52.6						
14	II	♂	2 1/2	52.0	52.0	52.7	52.6				
15	II	♂	3	52.7	52.6						
16	II	♀	2 1/2	52.0	52.0	52.7	52.6				
17	I	♂	3 1/2								
18	II	♂	2	51.5	51.3	52.0	52.0	52.7	52.1		
19	II	♂	2 1/2	52.0	52.0	52.7	52.6				
20	IV	♀	2 1/2	52.0	52.0	52.7	52.5				
21	II	♀	3	52.7	52.6						
22	III	♂	4								
23	IV	♀	2	51.5	51.4	52.0	50.6	52.7	50.4		
24	II	♂	3	52.7	52.6						
25	IV	♀	3	52.7	52.6						
26	II	♂	3	52.7	52.6						
27	III	♀	4 1/2								
28	II	♀	6								
29	II	♀	3	52.7	52.6						
30	III	♂	4								

N : Standard abdomen circumference in normal child of same age and sex.
F : Follow up abdomen circumference of the diseased child.
N.B. abdomen circumference measurements is not a parameter of measurement of growth after the age of 3 years old.

Table (39) : Follow up the chest circumference in the rural locality patients.

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♂	2	54.9	55.0	55.8	55.1	57.0	56.9	58.8	58.6	62.5	60.2
2	II	♂	3	57.0	57.0	58.0	57.0	58.4	56.9	60.5	58.4	64.6	61.2
3	II	♀	2 1/2	55.8	55.6	57.0	55.9	58.0	56.8	Death	Death		
4	III	♂	1 1/2	53.7	53.5	54.9	53.1	55.8	53.2	58.0	54.3	61.5	56.0
5	III	♀	4	58.9	60.0	59.3	58.5	60.5	58.9	62.5	60.1	Death	Death
6	II	♂	3	57.0	57.0	58.0	56.8	58.4	57.0	60.5	57.8	64.6	59.2
7	II	♂	2	54.9	54.6	55.8	54.7	57.0	55.8	58.8	56.2	62.5	58.9
8	I	♀	3 1/2	58.0	58.0	58.9	58.3	59.3	58.9	61.5	59.5	64.6	61.9
9	I	♂	1 1/2	53.7	53.6	54.9	53.7	55.8	55.1	58.0	57.9	61.5	60.9
10	II	♀	3	57.0	56.9	58.0	57.0	58.4	57.9	60.5	58.9	64.6	62.9
11	II	♀	4	58.4	58.2	59.3	56.9	60.5	59.4	62.5	61.9	67.1	63.9
12	I	♂	2 1/2	55.8	55.5	57.0	56.8	58.0	57.9	59.3	59.0	63.6	62.3
13	III	♂	6 1/2	63.6	63.5	64.6	52.1	65.9	62.3	68.4	62.6	73.7	65.0
14	IV	♂	2	54.9	54.8	55.8	54.0	57.0	54.1	Death	Death		
15	III	♂	4	58.9	58.8	59.3	58.8	60.5	59.0	62.5	59.1	67.1	60.3
16	III	♂	2 1/2	55.8	55.7	57.0	55.6	58.0	56.0	59.3	56.9	63.6	58.0
17	II	♂	3 1/2	58.0	85.0	58.9	58.1	59.3	58.2	61.5	59.0	64.6	62.3
18	III	♂	1 1/2	53.7	53.6	54.9	53.0	55.8	53.6	58.0	54.1	61.5	56.0
19	IV	♂	2	54.9	54.8	55.8	54.7	57.0	55.0	Death	Death		
20	I	♀	2	54.9	54.8	55.8	55.0	57.0	55.9	58.8	57.0	62.5	61.2
21	II	♂	3 1/2	58.0	58.0	58.9	58.0	59.3	59.0	61.5	60.9	64.6	62.9
22	II	♂	5	60.5	60.4	61.5	61.0	62.5	61.9	64.6	62.9	69.7	67.9
23	I	♂	6	62.5	62.4	63.6	62.9	64.6	64.2	67.1	67.1	72.4	72.1
24	II	♂	2 1/2	55.8	55.7	57.0	56.5	58.0	57.1	59.3	58.7	63.6	61.9
25	II	♂	4	58.9	58.8	59.3	59.0	60.5	59.5	62.5	61.1	67.1	65.0
26	II	♀	2	54.9	54.9	55.9	55.1	57.0	56.2	58.8	58.1	62.5	61.2
27	IV	♀	1 1/2	53.7	53.7	54.9	53.7	55.8	53.8	58.0	55.2	61.5	58.9
28	I	♀	2	54.9	54.8	55.8	54.9	57.0	56.8	58.8	58.5	62.5	61.8
29	IV	♀	3	57.0	57.0	58.0	56.0	58.4	56.8	60.5	57.0	64.6	58.2
30	I	♀	2	54.9	54.9	55.8	55.0	57.0	56.8	58.8	58.5	62.5	61.9

N : Standard ches circumference of normal child of same age and sex.
F : Follow up chest circumference in the diseased child.

Table (40) : Follow up the chest circumference in the urban locality patients.

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♀	2 1/2	55.8	55.7	57.0	56.0	58.0	56.8	59.3	57.0	63.6	59.2
2	III	♂	6	62.5	62.3	63.6	62.0	64.6	62.9	67.1	64.2	72.4	67.0
3	II	♀	2	54.9	54.8	55.8	54.6	57.0	54.9	58.9	55.5	62.5	58.0
4	II	♂	2 1/2	55.8	55.9	57.0	56.1	58.0	56.9	59.3	57.8	63.6	58.9
5	I	♂	4	58.9	58.9	59.3	58.8	60.5	59.0	62.5	59.9	67.1	65.2
6	I	♂	3 1/2	58.0	58.1	58.9	58.3	59.3	58.9	61.5	56.9	65.9	64.9
7	II	♂	3	57.0	57.1	58.0	57.5	58.9	58.3	60.5	59.1	64.6	62.9
8	IV	♀	1 1/2	53.7	53.8	54.9	53.0	55.8	53.2	58.0	53.9	Death	Death
9	I	♀	4	58.9	58.8	59.3	59.0	60.5	59.9	62.5	61.9	67.1	66.2
10	II	♂	2	54.9	54.9	55.8	55.0	57.0	56.1	58.9	57.1	62.5	61.1
11	III	♂	1 1/2	53.7	53.7	54.9	53.6	55.8	55.1	58.0	56.9	61.6	59.5
12	II	♀	2	54.9	54.8	55.8	55.0	57.0	55.9	58.9	56.3	62.5	61.0
13	II	♂	3	57.0	57.1	58.0	57.0	58.9	57.9	60.5	59.0	64.6	62.9
14	II	♂	2 1/2	55.8	55.7	57.0	56.5	58.0	57.3	59.3	58.9	63.6	62.6
15	II	♂	3	57.0	57.1	58.0	57.5	58.9	58.0	60.5	59.0	64.6	61.8
16	II	♀	2 1/2	55.8	55.8	57.0	56.1	58.0	57.0	59.3	58.0	63.6	60.0
17	I	♂	3 1/2	58.0	58.1	58.9	58.4	59.3	59.0	61.5	60.9	65.9	65.3
18	I	♂	2	54.9	54.9	55.8	55.4	57.0	56.9	58.9	58.9	62.5	62.3
19	II	♂	2 1/2	55.8	55.9	57.0	56.4	58.0	57.4	59.3	59.0	63.6	61.9
20	IV	♀	2 1/2	55.8	55.7	57.0	55.0	58.0	55.1	59.3	55.9	63.6	56.0
21	II	♀	3	57.0	57.0	58.0	57.2	58.9	58.1	60.5	60.0	64.6	64.0
22	III	♂	4	58.9	58.8	59.3	58.9	60.5	59.0	62.5	59.1	67.1	62.3
23	IV	♀	2	54.9	54.8	55.8	54.0	57.0	54.1	58.9	55.1	62.5	57.2
24	II	♂	3	57.0	57.0	58.0	57.0	58.9	57.5	60.5	59.2	64.6	62.9
25	IV	♀	3	57.0	57.0	58.0	57.0	58.9	57.3	60.5	57.8	Death	Death
26	II	♂	3	57.0	57.0	58.0	57.0	58.9	58.1	60.5	60.0	64.6	63.9
27	III	♀	4 1/2	59.3	59.4	60.5	59.3	61.5	59.6	63.5	60.0	Death	Death
28	II	♀	6	62..5	62.5	63.6	62.5	64.6	63.9	65.9	64.8	67.1	65.3
29	II	♀	3	57.0	57.0	58.0	57.5	58.9	58.0	60.5	59.5	64.6	63.8
30	III	♂	4	58.9	58.8	59.3	59.0	60.5	59.3	62.5	59.5	67.1	62.7

N : Standard chest circumference of normal child of same age and sex.
F : Follow up chest circumference in the diseased child.

Table (41) : Follow the size of the calf muscle in the rural locality patients.

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♂	2									25.7	25.6
2	II	♂	3							24.6	24.1	26.9	25.1
3	II	♀	2 1/2							Death	Death	Death	Death
4	III	♂	1 1/2										
5	III	♀	4					24.6	24.1	25.7	24.2	Death	Death
6	II	♂	3							24.6	24.5	26.4	25.3
7	II	♂	2									25.7	25.6
8	I	♀	3 1/2							25.2	25.1	27.6	27.1
9	I	♂	1 1/2									25.2	25.1
10	II	♀	3							24.6	24.4	26.9	26.5
11	II	♀	4					24.6	24.5	25.7	25.6	28.2	27.1
12	I	♂	2 1/2									26.3	25.9
13	III	♂	6 1/2	26.3	26.2	26.9	26.8	27.6	27.5	28.9	28.1	31.4	29.3
14	IV	♂	2							Death	Death	Death	Death
15	III	♂	4					24.6	24.5	25.7	25.1	28.2	27.1
16	II	♂	2 1/2									26.3	25.9
17	II	♂	3 1/2							25.2	24.9	27.6	27.1
18	III	♂	1 1/2										
19	IV	♂	2							Death	Death	Death	Death
20	I	♀	2									27.7	24.9
21	II	♂	3 1/2							25.2	25.1	27.6	27.2
22	II	♂	5	24.6	24.5	25.2	25.1	25.7	25.6	26.9	25.9	29.5	26.9
23	I	♂	6	25.7	25.6	26.3	26.3	26.9	26.7	28.2	27.9	32.7	31.1
24	II	♂	2 1/2									26.3	25.9
25	II	♂	4					24.6	24.2	25.7	26.1	28.2	27.1
26	II	♀	2									25.7	25.2
27	IV	♀	1 1/2									25.2	24.1
28	I	♀	2									25.7	25.6
29	IV	♀	3							24.6	24.5	26.9	24.1
30	I	♀	2									25.2	25.1

N : Standard calf Muscle size of normal child of same age and sex.
F : Follow up calf Muscle size of the diseased child.
N.B. The size of the calf muscle is not a parameter of measurement of growth before the age of 4 years old.

Table (42) : Follow the size of the calf muscle in the urban locality patients.

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♀	2 1/2									26.3	25.4
2	III	♂	6	25.7	25.7	26.3	25.1	26.9	24.9	28.2	25.5	30.7	26.9
3	II	♀	2									25.7	24.9
4	II	♂	2 1/2									26.3	25.5
5	I	♂	4					24.6	24.1	25.7	24.9	28.2	27.2
6	I	♂	3 1/2							25.2	24.5	27.6	25.5
7	II	♂	3							24.6	24.5	26.9	26.1
8	IV	♀	1 1/2									Death	Death
9	I	♀	4					24.6	24.2	25.7	25.5	28.2	26.9
10	II	♂	2									25.7	25.1
11	III	♂	1 1/2									25.2	24.8
12	II	♀	2									25.7	24.9
13	II	♂	3							24.6	24.2	26.9	25.3
14	II	♂	2 1/2									26.3	25.4
15	II	♂	3							24.6	24.1	26.9	25.1
16	II	♀	2 1/2									26.3	25.2
17	I	♂	3 1/2							25.2	24.9	27.6	26.1
18	II	♂	2									25.7	24.9
19	II	♂	2 1/2									26.3	25.8
20	IV	♀	2 1/2									26.3	25.1
21	II	♀	3							24.6	24.2	26.9	25.2
22	III	♂	4					24.6	24.3	25.7	24.5	28.2	24.9
23	IV	♀	2									25.7	25.2
24	II	♂	3							24.6	24.1	26.9	26.1
25	IV	♀	3							24.6	24.1	Death	Death
26	II	♂	3							24.6	24.1	26.9	26.2
27	III	♀	4 1/2					25.2	24.5	26.3	24.9	Death	Death
28	II	♀	6	25.7	25.3	26.3	26.1	26.9	26.1	28.2	21.2	30.7	29.1
29	II	♀	3							24.6	24.2	26.9	26.1
30	III	♂	4					24.6	24.5	25.7	25.1	28.2	27.1

N : Standard calf Muscle size of normal child of same age and sex.
F : Follow up calf Muscle size of the diseased child.
N.B. The size of the calf muscle is not a parameter of measurement of growth before the age of 4 years old.

Table (43) : Follow up the appearance of ossification in the rural locality patient

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♂	2	B	B	C	B	D	C	F	D	J	F
2	II	♂	3	D	D	E	D	F	E	H	F	K	H
3	II	♀	2 1/2	C	C	D	C	E	D	Death	Death	Death	Death
4	III	♂	1 1/2	A	A	B	A	D	A	E	B	I	D
5	III	♀	4	F	E	G	E	H	G	J	G	Death	Death
6	II	♂	3	D	D	E	D	F	E	H	F	K	H
7	II	♂	2	B	B	C	B	D	C	F	D	J	F
8	I	♀	3 1/2	E	D	F	D	G	G	I	I	K	K
9	I	♂	1 1/2	A	A	B	A	D	D	E	E	I	I
10	II	♀	3	D	C	E	C	F	E	H	F	K	H
11	II	♀	4	F	E	G	E	H	G	J	H	L	J
12	I	♂	2 1/2	C	C	D	C	E	D	G		K	L
13	III	♂	6 1/2	K	J	L	J					M	L
14	IV	♂	2	B	B	C	B	D	D	Death	Death	Death	Death
15	III	♂	4	F	F	G	F	H	F	J	G	L	H
16	II	♂	2 1/2	C	C	D	C	E	D	G	E	K	G
17	II	♂	3 1/2	E	D	F	D	G	F	I	G	K	I
18	III	♂	1 1/2	A	A	B	A	D	D	E	B	I	D
19	IV	♂	2	B	B	C	B	D	B	Death	Death	Death	Death
20	I	♀	2	B	B	C	B	D	D	F	F	J	J
21	II	♂	3 1/2	E	E	F	E	G	F	I	G	K	I
22	II	♂	5	H	H	I	H	J	I	L	J	K	I
23	I	♂	6	J	H	K	H	L	L				
24	II	♂	2 1/2	C	C	D	C	E	D	G	E	K	G
25	II	♂	4	F	E	G	E	H	G	J	H	L	J
26	II	♀	2	B	B	C	B	D	C	F	D	J	F
27	IV	♀	1 1/2	A	A	B	A	D	A	E	B	I	D
28	I	♀	2	B	B	C	B	D	D	F	F	J	J
29	IV	♀	3	D	D	E	D	F	D	H	E	K	F
30	I	♂	2	B	B	C	B	D	D	F	F	J	J

N : Standard time of ossification in normal child of same age and sex.
F : Follow up time of ossification of the diseased child.

Cent. of ossification of bone in ♂ and ♀ key for table 46 and 47.

Age in year	Male apperance of oss. centre	Female apperance of oss. centre	Key for the table
1 1/2	epiphysis of II metacarpal proximal epiphysis 4 toe	Epiphysis of I metacarpal proximal epiphysis great toe	A
2	epiphysis of V metacarpal medial cuneiform	Distal epiphysis 5th finger metatarsal toe	B
2 1/2	epiphysis of V metacarpal I Navicular bone	Patella, metatarsal IV	C
3	Distal epiphysis 5th finger metatarsal II	Lunate bone Distal epiphysis metatarsal V	D
3 1/2	Lunate bone, metatarsal III Great trochanter femur	Humerus, medial epicondyle	E
4	Metatarsal IV	Navicular bone (hand) proximal epiphysis raduis	F
4 1/2	Distal epiphysis metatarsal IV	Epiphysis calcaneous	G
5	Navicular bone (hand)		H
5 1/2	Proximal epiphysis (Raduis)	Distal epiphysis ulna	I
6	Medial epicondyle Humerus		J
6 1/2	Distal epiphysis ulna		K
7	Epiphysis calcuneous		L
11		Complete ossification at elbow	M
12			N
12 1/2	Complete ossification at elbow.	Complete ossification at foot	O

Table (44) Follow up the appearance of ossification in the urban locality patient

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♀	2 1/2	C	C	D	C	E	D	G	E	K	J
2	III	♂	6	J	H	K	H	L	J				
3	II	♀	2	B	B	C	B	D	D	F	D	J	F
4	II	♂	2 1/24	C	C	D	C	E	D	G	E	K	G
5	I	♂	3 1/2	F	F	J	F	H	H	J	J	L	L
6	I	♂	3	E	E	F	E	G	F	I	I	K	K
7	II	♂	1 1/2	D	D	E	D	F	F	H	H	K	L
8	IV	♀	4	A	A	B	A	D	B	E	B	Death	Death
9	I	♀	2	F	F	G	F	H	G	J	H	L	J
10	II	♂	1 1/2	B	B	C	B	D	C	F	D	J	F
11	III	♂	2	A	A	B	A	D	B	E	D	I	E
12	II	♀	3	B	A	C	A	D	C	F	D	J	J
13	II	♂	2 1/2	D	D	E	D	F		H		K	
14	II	♂	3	C	C	D	C	E	D	G	E	K	G
15	II	♂	2 1/2	D	D	E	D	F	E	H	F	K	H
16	II	♀	3 1/2	C	C	D	C	E	C	G	F	K	I
17	I	♂	2	E	E	F	E	G	E	I	I	K	K
18	II	♂	2 1/2	B	B	C	B	D	C	F	D	J	F
19	II	♂	2 1/2	C	C	D	C	E	D	G	E	K	G
20	IV	♀	3	C	C	D	C	E	C	G	D	K	E
21	II	♀	4	D	D	E	D	F	E	H	F	K	H
22	III	♂	2	F	F	G	F	H	G	J	G	L	H
23	IV	♀	3	B	B	C	B	D	B	F	D	J	D
24	II	♂	3	D	D	E	D	F	E	H	F	K	H
25	II	♀	3	D	D	E	D	F	E	H	F	Death	Death
26	II	♂	3	D	D	E	D	F	E	H	F	K	H
27	III	♀	4 1/2	G	G	H	G	I	G	K	H	Death	Death
28	II	♀	6	J	J	K	J	L	K				
29	II	♀	3	D	D	E	D	F	D	H	D	K	H
30	II	♂	4	F	F	G	F	H	G	J	H	L	K

N : Standard time of ossification in normal child of same age and sex
F : Follow up time of ossification of the diseased child.

Cent. of ossification of bone in ♂ and ♀ key for table 46 and 47.

Age in year	Male apperance of oss. centre	Female apperance of oss. centre	Key for the table
1 1/2	epiphysis of II metacarpal proximal epiphysis 4 toe	Epiphysis of I metacarpal proximal epiphysis great toe	A
2	epiphysis of V metacarpal medial cuneiform	Distal epiphysis 5th finger metatarsal toe	B
2 1/2	epiphysis of V metacarpal I Navicular bone	Patella, metatarsal IV	C
3	Distal epiphysis 5th finger metatarsal II	Lunate bone Distal epiphysis metatarsal V	D
3 1/2	Lunate bone, metatarsal III Great trochanter femur	Humerus, medial epicondyle	E
4	Metatarsal IV	Navicular bone (hand) proximal epiphysis radius	F
4 1/2	Distal epiphysis metatarsal IV	Epiphysis calcaneous	G
5	Navicular bone (hand)		H
5 1/2	Proximal epiphysis (Radius)	Distal epiphysis ulna	I
6	Medial epicondyle Humerus		J
6 1/2	Distal epiphysis ulna		K
7	Epiphysis calcuneous		L
11		Complete ossification at elbow	M
12			N
12 1/2	Complete ossification at elbow.	Complete ossification at foot	O

Table (45) : Follow up the calcification and eruption of the primary and permenant teeth in the rural locality patient

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♂	2	C	C	D	C					E	D
2	II	♂	3	C	C	D	C					F	D
3	II	♀	2 1/2	C	C	D	C			Death	Death	Death	Death
4	III	♂	1 1/2	A	A	B	A	C	A	D	B		
5	III	♀	4	D	D					E	D	Death	Death
6	II	♂	3	C	C	D	C					E	D
7	II	♂	2	C	C	D	C					E	D
8	I	♀	3 1/2	D	C					E	D	F	D
9	I	♂	1 1/2	A	A	B	A	C	B	D	C		
10	II	♀	3	C	C	D	C					E	D
11	II	♀	4	D	C					E	D	F	E
12	I	♂	2 1/2	C	C	D	C					E	D
13	III	♂	6 1/2	E	D			F	E			H	F
14	IV	♂	2	C	C	D	C			Death	Death	Death	Death
15	III	♂	4	D	D					E	D	F	E
16	II	♂	2 1/2	C	C	D	C					E	E
17	II	♂	3 1/2	D	D					E	D	F	E
18	III	♂	1 1/2	A	A	B	A	C	B	D	C		
19	IV	♂	2	C	C	D	C			Death	Death	Death	Death
20	I	♀	2	C	C	D	C					E	E
21	II	♂	3 1/2	D	D					E	E	F	E
22	II	♂	5	E	E			F	E			G	F
23	I	♂	6	E	E			F	F			H	G
24	II	♂	2 1/2	C	C	D	C					E	E
25	II	♂	4	D	D					E	E	F	F
26	II	♀	2	C	C	D	C					E	E
27	IV	♀	1 1/2	A	A	B	A	C	B	D	C		
28	I	♀	2	C	C	D	C					E	E
29	IV	♀	3	C	C	D	C					E	D
30	I	♀	2	C	C	D	C					E	E

N : Standard calcification and teeth eruption in normal child of same age and sex.
F : Follow up calcification and teeth eruption of the diseased child.

Dentation eruption of primary and pemenant teeth in child according to age key for table 48 and 49

Age in year	teeth eruption	Key for the table
1 1/2	1st molar and canine eruption 1st pre molar calcification	A
2	Lateral and central incisor calcification. 2nd premolar calcification	B
2 1/2	Second molar eruption 1st molar calcification	C
3	Canine and second molar calcification.	D
6	Central incisors, 1st molar permenant eurjption.	E
7 1/2	Lateral incisors pemenant eurption.	F
9 1/2	Calcification 1st and 2nd molar and central incisors calcification.	G
10	Canine and 1st molar permenant eurption.	H
11	2nd premolar permenant eurption.	I
12	2nd molar permenant eruption 1st premolar, canine calcifications.	J

Table (46) : Follow up the calcification and eruption of the primary and permenant teeth in the urban locality patient

No	Stage	Sex	Age	1st Examination		6 M		1 year		2 year		4 year	
				N	F	N	F	N	F	N	F	N	F
1	II	♀	2 1/2	C	C	D	C					E	E
2	III	♂	6	E	E			F	E			H	G
3	II	♀	2	C	C	D	C					E	E
4	II	♂	2 1/2	C	C	D	C					E	E
5	I	♂	4	D	C					E	E	F	F
6	I	♂	3 1/2	D	C					E	E	F	F
7	II	♂	3	C	C	D	C					E	D
8	IV	♀	1 1/2	A	A	B	A	C	A	D	B	Death	Death
9	I	♀	4	D	D					E	E	F	F
10	II	♂	2	C	C	D	C					E	D
11	III	♂	1 1/2	A	A	B	A	C	B	D	C		
12	II	♀	2	C	C	D	C					E	D
13	II	♂	3	D	C					E	D	F	E
14	II	♂	3 1/2	D	D					E	D	F	E
15	II	♂	3	D	D					E	D	F	E
16	II	♀	2 1/2	C	B	D	B					E	E
17	I	♂	3 1/2	D	C					E	D	F	E
18	II	♂	2	C	C	D	C					E	D
19	II	♂	2 1/2	C	C	D	C					E	D
20	IV	♀	2 1/2	C	C	D	C					E	D
21	II	♀	3	D	D					E	E	F	E
22	III	♂	4	D	D					E	D	F	D
23	IV	♀	2	C	C	D	C					E	D
24	II	♂	3	D	D					E	D	F	E
25	IV	♀	3	D	D					E	D	Death	Death
26	II	♂	3	D	C					E	D	F	E
27	III	♀	4 1/2	D	D					E	D	Death	Death
28	II	♀	6	E	D			F	E			H	G
29	II	♀	3	D	D					E	D	F	E
30	III	♂	4	D	D					E	D	F	E

N : Standard calcification and teeth eruption in normal child of same age and sex.
F : Follow up calcification and teeth eruption of the diseased child.

Deutation eruption of primary and pemenant teeth in child according to age key for table 48 and 49

Age in year	teeth eruption	Key for the table
1 1/2	1st molar and canine eruption 1st pre molar calcification	A
2	Lateral and central incisor calcification. 2nd premolar calcification	B
2 1/2	Second molar eruption 1st molar calcification	C
3	Canine and second molar calcification.	D
6	Central incisors, 1st molar permenant eurption.	E
7 1/2	Lateral incisors pemenant eurption.	F
9 1/2	Calcification 1st and 2nd molar and central incisors calcification.	G
10	Canine and 1st molar permenant eurption.	H
11	2nd premolar permenant eurption.	I
12	2nd molar permenant eruption 1st premolar, canine calcifications.	J

V. Study and follow up the educational level of the affected children.

Table (47) : The effect of ocophageal stricture on the level of education.

Level of education	No in Rural		No in urban		Total	
	♂	♀	♂	♀	♂	♀
Ageabove 6 and they didn't registered in school	2	8	1	5	3	13
Registered in school but they didn't continued there education	2	6	1	4	3	10
Continued their school program						
- with normal level	4	2	3	2	7	4
- with moderate level	4	-	4	1	8	1
- with low level	9	2	12	3	21	5
Total	21	18	21	15	42	33

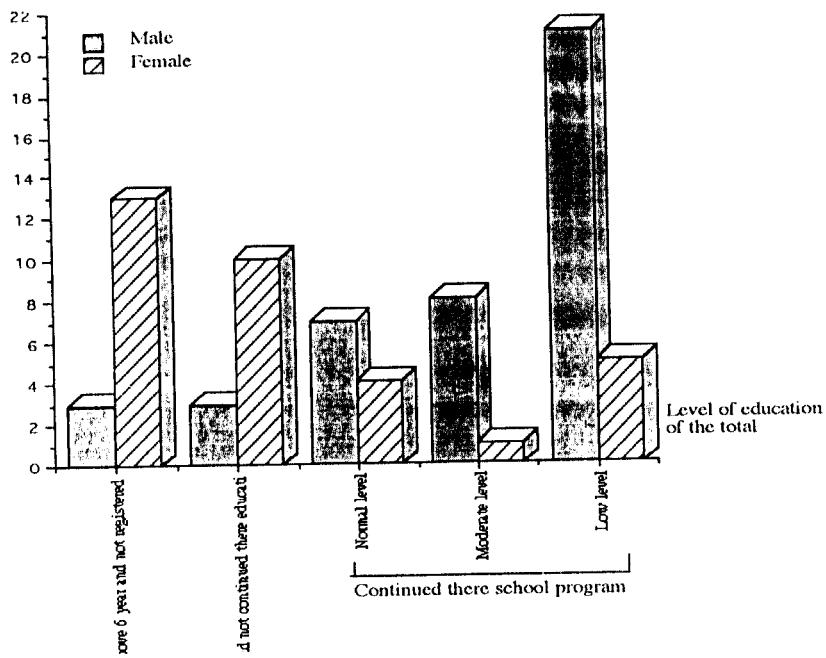

Fig. (28) : The effect of aeophageal stricture on the level of education.

VI. Study the intelligence of the post corrosive oesophageal stricture children.

Table (48) : Intelligence (i.Q.) and its frequance in patients with post corrosive oesophageal stricture patients in relation to sex residance and degree of injury at the end of 4 years follow up.

i.Q.	according to the residance				Total		according to the stage of oesophageal stricture				Total	
	rural		rural									
	♂	♀	♂	♀	♂	♀	I	II	III	IV	No	%
< 70	5	4	6	3	11	7	1	5	6	6	18	21%
71 - 85	8	5	10	1	18	6	8	6	6	4	24	28%
86 - 115	11	2	9	4	20	6	10	8	5	3	26	30%
> 115	5	3	4	6	9	9	3	4	5	6	18	21%

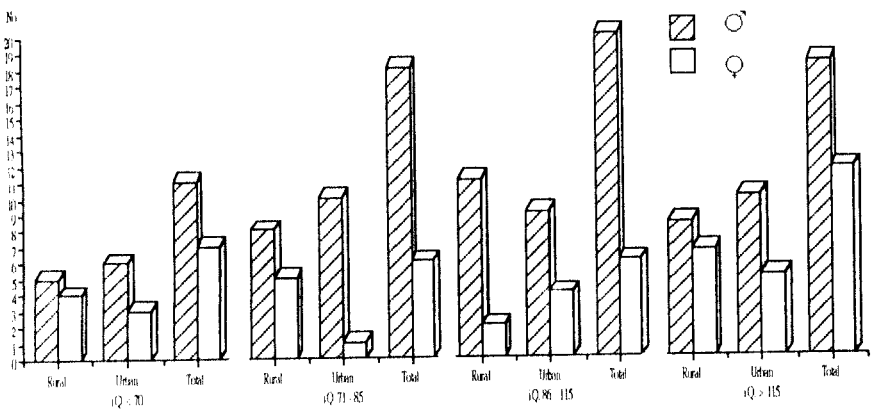

Fig. 29 : i.Q. and its frequance in post corrosive oesophageal stricture patients in relation to sex and residance at the end of 4 years follow up.

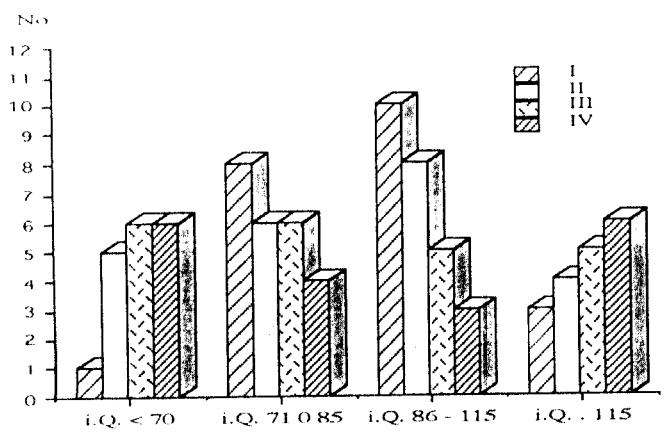

Fig. 30 : i.Q. and its frequance in post corrosive oesophageal stricture patients in relation to the degree of stricture at the end of 4 years follow up.

VII. Study the psychological disturbance of the affected children due to caustic oesophageal stricture.

Table (49) : Study the different psychological disturbance and its frequance in patierts with post corrosive oesophageal stricture in relation to residance, sex sand degree of injury.

Type of psychological disturbance	Rural		Urban		Total		according to the degree of oesophageal injury			
	♂	♀	♂	♀	♂	♀	I	II	III	IV
Listlessness	18	8	15	10	33	18	6	27	8	10
prostration	15	7	13	9	28	16	4	22	8	10
appetite disturbace	21	9	18	12	39	21	11	31	8	10
Anorexia and fear of eating	20	8	18	11	38	19	8	31	8	10
night mears and terrors	12	3	10	4	22	7	2	11	7	9
Anexity	20	9	17	11	37	20	8	31	8	10
fear of hopspital doctors and nurses	18	8	16	10	34	18	8	28	7	9
Interperson reverbation										
aggressive behavier	4	2	3	3	6	6	2	9	1	-
Unshare with others	14	7	13	8	27	15	6	18	8	10
Regression:										
Thumb suckling	6	3	3	2	9	5	5	9	-	-
Return to bottle	3	1	3	3	6	4	7	3	-	-
Uncontolled bowel and bladder	13	5	12	4	25	9	3	12	8	11
Clinging	14	6	15	9	29	15	5	24	6	9
Speech retardation	4	2	3	2	7	4	2	5	5	-
Walk retardation	5	3	5	4	10	7	2	8	4	3
over dependant	16	4	9	8	25	12	5	17	7	8
hypo activets	18	8	16	10	34	18	5	29	8	10
hyper activets	3	1	2	2	5	3	6	2	-	-
hypochondiral concern	13	6	10	7	23	13	4	21	4	7

Table (49) Continue

Type psychological disturbance	Rural		Urban		Total		according to the degree of oesophageal injury			
	♂	♀	♂	♀	♂	♀	I	II	III	IV
Tachycardia	5	7	4	8	9	15	3	5	8	8
Palpitation.	4	6	4	7	8	13	2	6	6	7
hyper ventilation	6	7	3	5	9	12	2	5	6	8
Misinter pretation of the meaning of injury	5	6	7	4	12	10	8	7	4	3
amnesia	3	2	1	4	4	6	2	2	3	3
somnambulism	-	2	1	1	1	3	-	-	2	2
Fugue status	1	3	1	2	2	5	-	2	2	3
delirium	4	3	5	4	9	7	1	5	5	6
Conversion	6	5	4	5	10	10	1	6	6	7
Hysterical attacks	3	2	2	2	5	4	-	3	3	3
Syncopal attocks	-	2	1	1	1	3	-	1	1	2
continuance of pain	5	7	4	8	9	15	1	7	8	8
headache	2	1	-	2	2	3	-	1	2	2
dysphonia	2	2	2	1	4	3	-	2	2	3
effect on bodymage										
looks afraid	16	8	16	12	32	20	3	31	8	10
agonizing	19	9	17	12	36	21	9	30	8	10
unpleasant	20	8	18	11	38	19	10	29	8	10
apathic	8	5	10	7	18	12	4	8	8	10
depressed	15	7	14	16	29	17	8	20	8	10
unattractive	20	9	17	11	37	20	8	31	8	10

VIII. Study the operative procedures in the management of the injuried children.

Table (50) : The operative procederes and its mortality in patients with corrosive oesophageal stricture

Degree of ocsophag eal injury	Occupation	Total No.		colon by pass or replacement		Stomach pull up		Mortility post operative	
		♂	♀	♂	♀	♂	♀	♂	♀
II	Rural	1	1	1	-	-	1	-	-
	Urban	2	1	2	-	-	1	-	1
III	Rural	1	3	1	2	-	1	1	1
	Urban	4	1	2	-	2	1	1	-
IV	Rural	2	3	1	1	1	2	-	1
	Urban	3	1	3	-	-	-	2	-
Total	Rural	4	7	3	3	1	4	1	2
	Urban	9	2	7	-	2	2	3	1

Figure 31:

Stricture upper oesophageal part.

Figure 32:

Stricture middle oesophageal part.

Figure 33:

Stricture lower oesophageal part.

Figure 34:

Middle oesophageal stricture

Figure 35:

Gastric tube replacement the stricture oesphagus.

Figure 36:

Colon replacement of the stricture oesophagus.

IX. Study of the behavior and psychological changes of the families of the corrosive oesophageal injured children.

Table (51) : The reflection of the post corrosive oesophagcal stricture of the children on their families

Type of effect on state of the family / Stage		Unawarness		Over protection		Normal Response	
		F	M	F	M	F	M
Stage I	Rural 12	1	-	2	5	3	1
	Urban 10	2	1	1	2	2	2
Stage II	Rural 27	6	3	-	9	5	4
	Urban 30	5	1	2	8	8	6
Stage III	Rural 6	-	-	2	3	1	-
	Urban 5	1	-	1	3	-	-
Stage IV	Rural 5	-	-	4	1	-	-
	Urban 5	-	-	3	2	-	-
Total	Rural 50	7	3	8	18	9	5
	Urban 50	8	2	7	15	12	8

F = Father M = Mother

DISCUSSION

DISCUSSION

Corrosives oesophageal injury is a problem of considerable concern all over the world especially in developing countries. In Egypt this type of injury is present in both rural and urban areas. The disease almost affects the children with impairments of a very important organ "the oesophagus" due to the chemical destruction induced by corrosives. It leads to inability to swallow with subsequent malnutrition and hyponutrition, infections and severe Psychic trauma due to severe painful swallowing, repeated exposure to instrumentation "endoscopic dilatation, several hospitalisation with possibility of major surgical interferance with its frequent complications. Beside the psychological disturbance, post corrosive oesophageal stricture with inability to have the food requirements leads to deterioration of the child growth and development, becoming a chronically ill child with chronic disability, and the child is considered a handicapped child (like Major causes of handicapped child like major causes of handicapped difficult or impossible to treat it as blindness, deafness, Mental, orthopaedic, cardiac and pulmonary causes) (**Roger Barker, 1963**). Post corrosive oesophageal stricture with the previous mentioned disability leads to serious socio economic

problems, which is the retardation in the educational level of the child even becomes an illitrate without any professional skills so he becomes aburden upon the society in which he lives.

The thesis studied the corrosive oesophageal injuries and its post corrosives sequalae in details including its aetiology, pathology, clinically and with all factors affecting its occurrance and complications.

Also the frequency of the disease in relation to age, sex, injureous substance and socio economic state of the child and his family were studied in both rural and urban areas.

Thesis also assess the type and the degree of the referred cases of oesophageal injuries whether acute "recent" or chronic "old".

The different line of treatment of these cases were evaluated with its efficacy, morbidity and mortality.

Psychological and developmental sequalae were evaluated.

The effect of the disease over the school educational levels of the child and the reflection of this major disability on his family and the environment.

I. Epidemiological study of caustic oesophageal injury:

The studied patients with caustic oesophageal injuries were referred to Ain shams university, Dar El-Sheifa, Nasser institute, sheibein El-Kom, Menoufia University hospitals. They were catagorised into Urban citizen if there residanceisin the city, or Rural citizen if there residance is in the village.

1. Study the incidence in relation to sex. (Table 11 Figure 11) . There are 100 patients 50 from rural area (32♂ and 18♀) and 50 from urban area (34♂ and 16♀). With a higher incidence of male affection 64% in rural and 68% in urban areas over all ♂ percentage 66% and this is similar to that obtained by (**Applequist, P. and Salno, M., 1980**), and the explanation of this is because a male child is more active and tends to be more rebellious and more reluctant to abide by the rules set by his parents, the table also shows alittle bit affection of females in rural area (18%) than females in urban area (16%) due to some girls not being attached to school in rural areas at early age (nursery school), and they have not the chance to play outside home, so they stay longer time at home either with the abscence of mother or the mother's not taking care of the child well during the crowded hours of home duties.

2. Incidence of caustic oesophageal lesion in relation

to age in both rural and urban areas (Tables 12, 13 Figure 12), the higher incidence of injuries occur in age between 1 - 3 years with percentage of 56% in rural and 50% in urban the second peak between 3 - 5 years (34% in rural and 38% in urban, the incidence of injury greatly diminished after the age of 5 years and I didn't detect any case exposed to recent corrosive oesophageal injury after age of 9 years in rural child and 7 years in urban child and the injury never occurred before one year the same result was obtained by old age (**Adam, J.S., and Brick, H.G., 1982**) the explanation of these results is that the injury doesn't occur before one year old age, because the child at that age is in intimate care of his mother and he cann't reach the caustic material also the injury doesn't occur after 9 year old age due to the child's becoming mature enough to be away from the caustic substance and he can determine well it's nature. The explanation of the higher incidence in the age between 1 - 5 years is due to it's being the age of the child's moving around inside his home and he has the desire to explore every thing around him so there is a big chance to reach the caustic substance and he drinks it as he cann't realise its serious effect.

3. Study the incidence of caustic injury in relation to the type of the caustic substance (Table 14 Figure 13) the potash has almost the cause of the oesophageal injury in our country due to its wide use as a detergent for cleaning

at home with a percentage of 93% and only 7% due to other chemicals (4% due to Kerosine 3% due to pattary acid), the same results obtained by (**Nagy, M. 1973, Kulig, K. *et al.*, 1983**).

The explanation of these results is due to the wide use of potash as a cleaning substance in home. Also its properties as it is odourless has platable taste and its colour which is like milk or water .

4. The relationship between the occurrance of the injury and habits of the family to give attention to keep the chemical caustic substance in a locker out of the child's reach. The results in Table 15 Figure 14 show a higher incidence of injury in the families who are careless to keep caustic away from their children with percent age 84% in total studied cases (82% in rural and 86% in urban) the same results obtained by (**Sugawa C. *et al.*, 1981**).

5. The caustic oesophageal injury has a higher incidence in those families frequently using potash detergent in cleaning (Table 16. Figure 15) 83% in the total studied children (84% in rural 82% in urban), with nearly the same results obtained by (**Litovitz, T.L., 1985**) this possibility is due to decrecase in sensation of the hazerdous effects of this dangerous substance due to frequent use forgetting to keep it out of their children's reach.

6. When the family has a back ground about the disease and the catastrophy caused by potash either from the communication (Radio - TV etc.) or the family knows an affected child with this substance, the incidence of injury decreases to be 28% compared to 72% in families have no knowldge about the injury (Table 17 Figure 16), the Same results discussed before by (**Marion, L., *et al.*, 1978**).

7. The level of education of the father plays an important role in the behaviour of his family also more attention to his children to teach them to Keep away from the possible source of dangerous substance (Table 18 Figure 17), in this study the disease is more frequent in the children whose fathers are illiterate (39% of the total no of children, 44% from rural and 34% from urban, then the second frequency in the children whose fathers reached a level of education (Prep. school certificate) 28% and those their fathers are of a mid level of education (seconday school certificate) 29%. The lowest frequency occurs in the children whose fathers are highly educated (18% from urban zero% form rural, these results are similar to that study done by (**Friedman, E.M., and lovejoy, F.H., 1984**).

8. The incidence of caustic oesophageal stricture in relation to the mother's profession (Table 19 Figure 18).

As the mother plays a very important role in taking care of her children when the mother is a house - wife of course the children have more care, comparing them to those under the care of someone else, the frequency of the oesophageal injury is 26% in the children whose mothers are house wives to 74% in the children whose mothers work outside their homes (6% to those who have private work and 68% to those having public. work. The cause of the low frequencey in children whose mothers have a private work is that the child usually accomponies his mother during the working hours, the high frequency in the children whose mothers are governemental workers is because the mother usually leaves her children with her relatives or neighbours, and they of course receive less care and unfortunatly the caustic substance can be reached by the children .

9. The economic level (Table 20 Figure 19) has a reflection on mode of living of the family and the frequency of the oesophageal injury will be low (14%) when the economic level is high and vice versa. unpredicted in the poor family with income lessthan 150 L.E. per month. (21%), this is due to either the percentage of this group of families has decreased in ratio to the general population or when they answer my questionaire they answer in an ironic tone (there is no cloths to search for detergent) the incidence is high in both moderate and

low level with percentage of 39% and 26% consecceuatively the same result was obtained by (**Leape, L.L., *et al.*, 1971**).

10. When the number of children increased in the family, the frequency of aesophageal injury increased (64% over all patients. 62% of rural and 66% of urban), and decreased to be 30 % overall patients. 34 of rural and 26% of urban the incidence becomes very low when the child is single (6% over all patients 4% of rural and 8% of urban areas), Table 21 and Figure 20 this is due to the family take care easily of a single child or two children without big efforts than if the number of children are more than two this also reached by (**Hawkins D.B., *et al.*, 1980**).

11. The siborder of the child also reflected on the frequency of the oesophageal injury as the first off spring usually receives abetter care with frequency of 10% in both groups 6% of rural and 14% of urban(, when The child is late in the family especially if he is of the same sex the incidence is high 50% in both groups (52% of rural and 48% of urban), Table 22 and Figure 21 the same results was obtained by (**Genieser, N.B., and Becker, M.H., 1969**).

12. Table 23, A,B Figure 22 A,B demonestrate the frequency of the occurrance of the oesophageal injury in

relation to the social state of the family, the heigher frequency of the lesion (79% in all groups 86% of rural and 72% of urban) occurrs in the family when father and mother are together is simply due to this being the usual and major manner of life in families, but comparatively to the frequency of social state in the society it is high in the family : divorced parents 7% and 1% if father dead, 2% if mother dead, when the father works abroad the frequence is 5% and if the mother works abroad it is 1%, it is 2%, 1% if father or mother in prison.

The incidence is high 20% if the child in nursery at the mother's work and 16% in case the child in private nursery, the higher frequency occurrs in the children their family leaves them with relatives or neighbours it reached 36% due to the less attention though the frequency of 28% in the children at home with their family looks high that's only due to its being the usual manner of life in our society.

II. Pathological study of corrosive oesophageal stricture :

1. There are 60 patients with recent corrosive aesophageal stricture 30 from rural and 30 from urban areas, they were assessed by endoscopy to state the degree of injury according to the classification of (Johnson, E.E. the study shows in (Table 24 Figure 23)

stage I with superfascial affection of segment less than 5 cm, the percentage is 18.3% in both group of patients 20% of rural and 16.7% of urban stage II with superfascial affection of a segment more than 5 cm has the highest frequency 68% over all 53.32% of rural and 50% of urban and stage III with deep penetration 11.7% of over all 10% of rural and 13.35% of urban lastly stage Iv with perforation and Mediastinitis over all affected children are 18.3% (16.7% of rural and 20% of urban) so the Majority of the pathological degree of the recent caustic aesophageal injuries lies in stage II and III this frequency was obtained also by **(Scher, L.A., and Maull, K.L., 1978).**

2. The degree of oesophageal stricture as it is the sequalae of the acute stage when the child survives, Table 25 Figure 24. It was classified as it was done by **(Johnson, E.E. 1963)** the Majority of cases in this study are of third degree (impassable with short segment less than 5 cm) (30% all over the cases 25% of rural and 35% of urban), and those patients cannot be treated unless surgical treatment was done; unlike the case with passable stricture first and second degree 22.5% and 27.5% consecuatively improved by endoscopic dilatation and cortisone treatment, the 20% of over all cases with impassable segment more than 5 cm those patients are very critically and need major surgical interferance.

3. Dysphagea is the main symptom of aesophageal stricture and injury, this thesis studies all types of dysphagea and its frequency (Table 26 Figure 25), the higher frequency is the dysphagea to semisolid food (stage II) with percentage 37% all over 38% of rural 36% of urban. than dysphagea to fliud with regurgetation of saliva (stage Iv) 27% all over patient 26% of rural and 28% of urban) then the incidence type of dysphagea to fluid stage III with frequency of 24% and only 12% of cases are with dysphagea to solid (stage I), The results were compared to other studies which was done by **(Giffin, C.S., 1964 and Cleveland, W.W. *et al.*, 1968).**

4. The mortality of caustic oesophageal injury and its causes is illustrated in (Table 27 Figure 26)

a. In recent injury out of 60 patients 7 cases died 5 of them due to chest complications and 2 due to Metabolic disorders **(Delarue A.*et al.*, 1985).**

b. In old injury out of 40 cases 7 patients died, 1 case due to malnutrition and 6 cases due to post operative complications (2 due to haemoruhage, 2 due to chest complications and 2 due to chest fistulae, which is high rate of mortality compared to that was obtained by **(Othersen *et al.*, 1988).**

III. Effect of cortisone therapy and frequent oesophageal dilatation as aline of treatment described by **(Middel Kamp, J.N., *et al.*, 1969).**

In recent cases in this study the result (Table 28, Fig. 27) is 3 cases out of 60 patient 5% developed perforation with the dilatation 18.3% of patient showed no improvement compared to 62.5% in the group was n't treated by cortisone and oesophageal dilatation mild improvement in patients takes semi solid fluid occurs in 10% of cases compared to 62.5% in the other group, moderate improvement and patients can takes semisolid diet easily in 16.7% compared to 12.5% in other group without treatment by cortisone and dilatation. the best results with good improvement and patients can have ordinary diet, occurs in 50% of cases in the patients treated by cortisone and dilatation compared to zero% in other group have no treatment with cortisone and dilatation; the same results obtained by **(Ashcraft K.W., and Padula, R.T. 1974).**

IV. Anthropometeric study to assess the growth of the growth of the affected child, All cases with recent injuries 60 children (30 of rural and 30 of urban) were examined immediately after the exposure to corrosives and all parameters of growth were registered and then followed up after 1, 6 month 1, 2 and 4 years. All the

parameters of body growth are tabulated with referance to age, sex and residence, the resulting measurments are put in coloumn opposite that of a normal child with same age and sex the normal measuments are taken from the standard Universal measuments as discussed in the Review. There is deterioration of body weight of the affected child (Table 29 rural 30 urban) after the injury the deterioration occurred very rapidly in the first follow up after one month due the stress and Metabolic response to the trauma **(Kagan, 1982).**

Also due to inability to swallow food requirements due to painful swallowing odynophagea or stablished stricture chest infections induce toxaemia with body loss. In this study when the degree of oesophageal injury is severe there is a rapid and severe as shown in Case No 4, 5, 13, 14, 15, 18, 27, 29 of rural and case No 2, 8, 11, 20, 22, 23, 25, 27, 30 of urban with stage III and IV, the deterioration also continued in the subsequent follow up, there were four dead children in rural patients and 3 cases in urban patients due to chest infection and Metabolic disturbance, as the results obtained by (**Brazelton and Yogman, 1986**) there is slowely regaining of body weight in 15 cases of rura patients No 2, 6, 7, 8, 9, 10, 11, 12, 20, 21, 22, 23, 25, 28, 30) and 15 cases of urban patients (No 1, 4, 5, 6, 7, 9, 10, 15, 16, 17, 18, 19, 21, 24, 26,), with percentage of 50 % of cases this is due to the

improvement and response to cortisone treatment and oesophageal dilatation as discussed by (**Middel Kamp, J.N., *et al.* 1969**).

1. Study of surface area as aparameter of growth which is more sensetive than body weight (**Kagan, 1982**) Tables (31, 32) show the M2 surface area which measured according the quetation page (89) in the review, also to give the same results of deterioration in body weight of the different cases.

2. The effect of corrosive oesopgageal injury on the height of the child (Tables 33,34) the same results in deterioration of child height as that of body weight but to less extent as the body height is less affected then body weight in these cases as said by (**Kagan, 1982**) also the child with improvement of his condition with cortisone and endoscopic dilatation he started to regain the normal standard of height.

3. The head circumference (Tables 35, 36). This parameter is only restored to the child below the age of 3 years as it is unsignificant after that age. (**Dworkin, 1988**). In this study there was no detected difference between the diseased child in the several follow ups and that of the normal child measurments.

4. The abdominal circumference (Tables 37, 38) is

much affected in the child with nutritional defeciency due to enlarged liver, ascites mentioned by (**Piaget and Inhelder, 1969**). Any how in this study there is no difference between the affected child and the standard found results in normal child in the different follow up times.

5. Chest circumference (Tables 39, 40) is better than abdomen circumference as aparameter for growth as it less affected by organomegaly the results of chest cirumference in the affected child follow up is similar to that obtained by measurment of the body weight (**Kagan, 1982**).

6. The size of calf muscle (Tables 41, 42), it' a parmeter of growth and unsignificant before the age of 4 years old (**Mahler *et al.*, 1975**) so its estimation was done till the child reached 4 years old and as its size (let muscle so it is much affected by undernutration of the affected child, it deminshis in size in those patients with stage III and IV more like the body weight affected.

7. The appearance of ossification centre detected by xray is a very accurate Method to estimate the child growth (Tables 43, 44) the results of follow up child was illustrated in (letter) which is an appraviation to sites of bone ossification (underlying the Tables), in the neighbouring coloumn for each follow up there is the

expected appearance of ossification in a standard normal child ♂ and ♀ of the same age of that the follow up diseased child was examined, the Tables showed a delayed time of appearance of ossification due to malnutrilion and vit D, calcium defeciency in diet, (**Yogman *et al.*,1988 Dixon and stein *et al.*, 1987**).

8. Tables (45, 46) shows the follow up of tooth eruption as a parameter for estimation the growth. It is also an accurate method with the same results obtained by study the bone ossifications. it is more simple and rapid method for accurate estimation of growth as said by (**Brazelton and Yogmanb, 1986**) . so assessment of growth which is affected by corrosive oesophageal injury, body weight, body surfac area. child length, tooth eruption, and appearance of ossification center are accurate.

V. Study the follow up of the education level of the affected child (Table 47, Figure 28) the study was done for 75 diseased child reached the age of 6 year or more (The school age), out of total no 16 children reached the age of school but their familes did not rgister them (3 are ♂ and 13, are ♀) the high incidence in females due to the uncorrect idea that the school education is not so important to female especially if she is chronically ill, the study found that there 13 patient (3 ♂, 10 ♀) registered in

school but they can not continue, as the chronically child can not cooperate with the daily regulation of the school programme. Also repeated medical consultation and instrumentation (endoscopics), with the possible surgical treatment and repeated attacks of infection, due to all of these the child can not keep up with his studies, there are 46 children continued their school education with follow up 11 of them (7 ♂, 4 ♀) continue with normal level, 8 children (8 ♂, 1 ♀) continue with moderate level and 26 children (21 ♂, 5 ♀) did not improve at all in their school programme with low level, so the corrosive oesophageal injury (Chronic ill) has a very bad reflection on the child level of education as that was mentioned by **(Perlstein, 1952, Myerson, 1963 Wenar, 1954, Rusk, 1964).**

VI. Study the intelligence of the post corrosive oesophageal stricture patients in relation to sex residance and degree of injury at the end of 4 years follow up table 48 figures 29, 30 by estimating the distibution in i.Q. scores according to the age of the estimated clild in the study there is decrease in the i.Q. scores in the studied cases (86 patients) compared to the normal distribution of i.Q. scores discribed by **Rita wicks-Nelson and Allen C-Israel, 1991** as they Mentioned that the frequance of i.Q. scores in normal population is 3.27 % for i.Q. scores less than 70, 13.59 % for i.Q. scores 71 - 85, 88.26 % for i.Q. scores 86 - 115 and 16.86

% for i.Q. scores more than 115. Compared to the results in this thesis which are 21 % for i.Q. scores less than 70, 28 % for i.Q. scores 71 0 85, 30 % for i.Q. scores 86 - 115 and 21 % for i.Q. scores more than 115; this effect of decrease in i.Q. scores in chronic diseased child was observed also by **Frankenberg *et al.*, 1981; Cowan *et al.*, 1960; Davis, 1963; Diller *et al.*, 1966; Diller, 1970 and Bortner and Birch, 1960** in their studied patients with chronic ill child of Rhematic heart, chronic asthma and poliomyelitis as the patients severaly admitted to the hospitals and usually absent form his school and normal surrounding society that affects also less education and mentality capability.

VII. The psychological disturbance. Patients with corrosive oesophageal injury and its sequalae of stricture were exposed to full psychological assessements (Table 49). Oesophageal corrosives is a major catastrophy to the child as it affects avery vital organ which is the oesophagus, as it is the passway of feeding and saliva. If this important physological demand is deminished due to inability of swallowing or if swallowing becomes painful, the patient does n't get his needs of his nutrients, a state of hypo and malnutration occurs, as the patient stays for along time under treatment and hospitalisation with repeated exposure to the stress of previous instrumentation or surgical interference. All this are reflected to the

psychology of the child as mentioned by (**Minuchin, S. *et al.*, Shranm H., 1965**). The study was done to all psychological and mental disturbance occurred to the injured patients in relation to sex and degree of oesophageal stricture. the psychological findings in the 60 studied patients.

1. Listlessnes : Was observed in 51 patients (33 ♂, and 18 ♀) the majority of then occurred in the patients with second degree oesophageal injury and the results goes with the study done by (**Grollman, B.A., 1967**) (**Murphy and Jellinek, 1988**).

2. Prostration : 44 children developed these symptoms out of them 22 patients with stage II and this agrees with the study done by (**Mellish, R.W.P., 1969**).

3. Appetite disturbance in the form of anorexia and fear of eating 57 patients develops these symptoms, 31 of them with stage II as was detected by (**Jessner, I. *et al.*, 1952**).

4. Nightmares and terrors, developed in 29 children, 11 of them in degree II injury agree with the study was done by (**Force, 1956**) (**Simmons, 1987**) (**thomas, A.A. Chess. S., 1977**).

5. Fear of Doctors, nurses and hospitalisation 52 children developed these symptoms 28 of them with grade

II oesophageal injury, due to the child's developing pain during the instrumentation and injestion also the pain of operation as he can not judje that the procedures are for his benefit as discussed by **(Mac Carthy, D. 1962).**

6. Interperson reverbation in the form of aggressive behaviour towards his brothers, relatives and friends even his parents. It was developed in 12 patients, 9 of them with stage II oesophageal stricture also unshare with others due to his inability to follow their activity due to his chronic illness as it was shown in 42 cases out of them 18 with stage II oesophageal injury both results were obtained by the study was done by **(Green, M. and Solnit, A.J., 1964) (Green M., 1984).**

7. Regression and patient's return to earlier age of life in his manner in the form of thumb suckling which was observed in 14 cases, 9 of them with stage II oesophageal injury, return to bottle feeding if the child can swallow fluid this was detected in 10 cases 7 of them with stage I oesophageal stricture, uncontrolled bowel and bladder, occurred in 34 cases, 12 of them in stag II. Clinging occurred in 44 cases 12 of them with stage II **Jones and Sick 1967.** Speech retardation occurred in 13 cases, 5 of them in stage II. Walk retardation due to the chronicity of the disease and the hypo, malnutration and (development of Rickets in some cases) it occurs in 17

cases 8 of them in stage II. family over dependant and always stuck to his parents there were 37 cases that developed these symptoms 17 of them in stage II, all the previous results also was observed by (**Farber and Jenne 1963**) (**Green, M. and Solinit, A.J., 1964**).

8. Hypoactivity due to the debletation and general weakness. There are 54 cases that developed these symptoms 29 of them in stage II. It was observed also by (**Granosfsky, 1959 and Gofman 1963**).

9. Hypochondrial concern. It occurred in 36 patients, 21 of them stage II oesophageal injury and this sign was observed also by (**Grollman, B.A., 1967**) (**Rutter *et al.*, 1976**).

10. Tachycardia, palpitation, hyper ventilation occurred in 24, 21, 21 cases consecuetively due to fear, chronic anaemia, and chest infections. These symptoms and signs were observed also by (**Langford, W./s., 1961**).

11. Misinterpretation of the meaning of injury occurred in 22 cases of them in stage I as was observed by (**Doman, *et al.*, 1960 Freedman, S., 1968**).

12. Amnesia there are 10 cases 6 of them with stage III, IV sticture. as was descibed by (**Davis, 1963**).

13. Somnambulism : there are only 4 cases that developed this symptoms as discussed by (**Offer D., 1969**).

14. Fugue status there are 7 cases that developed it, as discussed by **(Plank, E.N. and Horwood, C., 1961).**

15. Delirium there are 16 cases 6 of then with stage IV stricture developed it as discussed by **(Force, 1956).**

16. Conversion : 20 cases developed it (7 of them with corrosive stricture stage IV as mentioned by **(Fawcett, 1959, Farber and Jenne 1963).**

17. Hysterical and syncopal attacks occurred in 9 and 4 cases consecuatively as observed by **(Heiscl, J.S., 1973).**

18. Continuances of pain it was a symptom occurred in 24 cases 16 of them occurred in cases of stage III and IV also phageal Stricture, this was mentioned by **(Heiscl, J.S., 1973).**

19. Headache was observed in 5 cases, as descriped by **(Heiscl, J.S., 1973).**

20. Dysphonia : Occurred in 7 cases it was observed also by the study that was done by **(Heiscl, J.S., 1973).**

21. The effect on body image was observed in children in the form of looks : a frear, agony, Unpleasantness, apathy, depression and unattraction. The previous signs were observed in cases 52, 57, 57, 30, 46 and 57 consecuatively as mentioned by the study of

(James, V.L., and Wheeler, W.E., 1969) (Davis, 1963, Diller *et al.*, 1966, Diller, 1970, Bortner and Birch 1960).

VIII. Study the operative procedures "Table 50, Figures 31, 32, 33, 34, 35, and 36".

The effect, morbidity and mortality was studied in 22 cases (equal number in both rural and urban areas) to whom surgical operations were done, colon by pass or replacement was done in 13 patients 6 of them from rural area(3 ♂ and 3 ♀) and 7 from urban area all were ♂,

Stomach pull up was done in 9 cases 5 of them from rural areas (1 ♂ and 4 ♀) and 4 from urban areas (2 ♂ and 2 ♀)with mortality of 7 cases about 32% which is high due to the operation's of a major type in already. chronically ill patient, the same results also described by **Abdel wahab Y. El-Ok by *et al* 1991, LindahL, H. *et al.* 1983, Cohen, D.H., *et al* 1974** This study also discussed the operative procedures in relation to the degree of oesophageal stricture. 5 patients were operated upon in cases of stage II with mortality of one case and a cases were operated upon in cases of stage III with mortality of 3 cases. Lastly 8 patients were operated upon in cases of stage IV with mortality of 3 cases. From this the mortality was increased when the stage of oeso phageal stricture is advanced the same results were obtained by

SUMMARY

Corrosive oesophageal injury is considered one of the most dangerous injuries in children affecting one of the most important organs which is the oesophagus causing it to lose its function which is food swallowing. As this injury leads to death of the child or affection of one of his vital organs and as it occurs due to ingestion of chemical substances available in most houses in our society especially potash (Na OH) used in cleaning processes. This thesis studies all reasons and factors of occurance of this injury, it also studies the percentage of its occurance in relation to age and sex, the degree of injury in acute "recent" cases and chronic "old" ones, the percentage of deaths, side effects due to injury, effects of oesophageal stenosis on physical and mental growth of the child and psychological effect of the injury on the child and his family through the study of cases in Menoufia University Hospitals, Sheibien El-Kom Hospital, "rural area" Ain Shams University, Dar El-Sheifa and Nasser Institute with the consideration of the child's residence whether in villages or cities - 100 patients affected by caustic oesophagsl injury were put under study. They were divided into 2 groups of 50 patients each. Each group includes 30 acute cases and 20 chronic ones. By

epidemilogical studies, it was found that the incidences of the injury : 66 ♂ and 34%♀. The majority of injuries occures before the age of 3 years with a percentage of 50% in urban area and 56% is rural ones and seldomly occurs after the age of 5 years, as the age of the child between 2 and 5 years is accompanied by movement and locomotion of the child inside the house and the increase in his desire to explore everything around him ignorant to the dangerous effects of the surrounding objects. Potash is considered the main cause of caustic injuries as it is always available in egyptian houses (used as a detergent), its properties help the child to drink it as it's odourless, looks like milk and doesn't cause the child any pain while drinking it. The study assures that families don't pay enough attention to keep the substance out of the children's reach which increases the incidence of the injury to 84%. Also the frequency of the use of the injury is considered an important factor, as the percentage of occurance reaches 83% in children of families who frequently use it, also the ignorance of the dangerous effect of this chemical and not knowing any one affected by it increases the percentage to 72% in both societies. Another important factor is the educational level of parents as the percentage of incidence shows remarkable drop when the parent is a university graduate as he knows the effect of such a dangerous substance and it increases

to 39 % (44% rural, 34% urban) when the parent is ignorant. The percentage of occurnce also increases when the mother is working to 68% (70% rural, 66% urban) as less care and attention is paid to the child. It is also affected by the economic level of the family as it increases in midlevel families and as the number of children increases especially if the no of children reaches more than 3 children reaching 64% (62% rural, 66% urban). The same happens when the rank of the child increases as the latter the child, the higher the probability of incidence reaching 50% (62% rural, 48% urban). By studying the social conditions of the family, it was found that the occurance of divorce, death of one of the parents, his working abroad or being in prison increases the percentage of incidence putting into consideration the frequency of occurance of such cases in the Egyptian society. If the mother is working, she puts her child in a private kindergarten leading to drop in percentage due to absence of such substance, but if the child is left with relatives, the percentage of occurance increases due to lack of care to 36%, (38% rural, 34% urban). As to percentage of incidence in children who stay at home, it's 28% in both societies, which is considered a small percentage as this is the common and normal case.

The pathological study of diseased oesophagus states

that the percentage of second degree injury is 51.68% in recent cases. While in old cases, the frequency of incidence is higher, with a percentage of incidence of 30% in rural, 35% in urban in the third degree. They all showed difficulty in swallowing which increases directly with the increase in degree of injury. The percentage of difficulty in swallowing according to nature of substance was 12% to solids, 37% to semi solids 24% to fliuds and 27% showed dysphagea with regurgitation of saliva which shows how dangerous this disease is and its side effects on the nutritional process with the improbability of finding substitutes for natural nutrition in most cases.

There were 14 death cases : 7 in recent injuries and 7 in old ones. In the recent cases : 5 were due to chest complication and 2 were due to metabolic disturbances. As for the old cases : 1 was due to malnutrition and its complications, while in the other 6, it was due to surgery.

The research also studied the effect of cortisone treatment and endoscopic dilatation on recent cases and the following results were obtained : 5% dangerous, due to perforation 50% good improvement, 16 - 6% moderate improvement.

Those results were compared to the old cases that were not treated by cortisone or endoscopic dilatation showing no improvement in swallowing which shows the

importance of early cortisone treatment and endoscopic dilatation decreasing the percentage of oesophagael stricture making treatment and nutrition processes easier.

It also discussed the effect of oesophagael injury by corrosives on body growth though studying and following his measurements. The results showed general growth through following the child's body weight, height, nutritional conditions, and his head, abdomen, chest, calf muscle circumfernce in addition to appearance of deciduous and permanant teeth and bone ossification. The degree of injury is considered an important factor affecting the child's growth, as growth is inversly proportional to the degree of injury which might prevent the child in school age from education with a percentage of 13% in females and 3% in males or lead to the child's not continuing his studies with a percentage of 10% in females and 3% in males or not keeping up with the level of his collegues with a percentage 5% in females and 21% in males which shows the dangerous effect of this injury as it doesn't only delay the child's physical growth but also it affects his mental growth and educational ability which is highly reflected on the society in which we live, as it leads to the increase in number of ignorante and illitrate citizens unable to work due to strong physical weakness in addition to long duration of curing period, that might take a lifetime.

The thesis also studied the child's psychological condition which was highly affected due to the child's feeling continous pain while swallowing also due to long and various modes of currance in hospitals in addition to affection by different diseases subjecting him to chronic depression causing him to become an antisocial person limiting his relations ships which in turn creates anger and hatred inside the child towards the society in which he lives. There's a strong relation ship between the degree of injury and the psychological condition where the higher the degreee of injury, the worse the psychological condition especially in the second degree due to its high frequency.

The study also followed cases subjected to surgical interferance and they were 22 cases 11 rural (4 males, 7 females) and 11 urban (9 males, 2 females). As for the type of surgery, it was as follows 13 cases (replacement of oesophagus by colon and 9 cases replacement of stricture by the stomach) and 7 of those cases died which shows how dangerous the surgery is, as injured cases are very weak making feeding very difficult in addition to frequent accompaning diseases like recurrent chest infection.

The thesis also studied the psychological effect of injury on the child's family due to mental and economical burdon, besides taking care of the injured child causes

their deley in work and neglecting their other children which causes great mess in the family with a percentage of 48%.

The study instructs the following recommendations :

1. Paying attention to corrosive injuries and their complications considering it a chronic disease and considering injured children handicapped children giving them medical and social attention given to other handicapped children.

2. Increasing the citizens' understanding of the dangerous effects of this disease and way of avoiding it by national campaigns through different mass media.

3. Recommending factories producing these chemicals to add warning signs on bottles and adding any substance that makes potash unplatable without changing its chemical propeties.

4. Paying good attention to family matters *e.g.* no of childern, family planning. Strengthening family bonds, good relationships between parents.

5. Increasing the no of kindergartes in mothers working places to avoid leaving them home alone.

6. In case of injury, citizens and doctors in medical centers should be aware that after first aid treatment, the

patient should be sent to specialized centers so that endoscopic treatment and dilatation would be given to the child and also surgical interferance without delay.

7. Paying social service to families having injured children and aiding them in the financial expenses of their injured child.

8. Paying good care to psychological and educational services to injured child by the help of specialists and arranging field visit to their study places and training teachers to know how to deal with them.

9. Arranging suitable jobs for those patients, so that they would not be a burdon on their families and giving them the chance to be responsible for them selves.

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الملخص العربى

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تعتبر الإصابات الناتجة عن تناول المواد الكاوية من أخطر الإصابات التى تصيب الأطفال وتسبب ضررا بالغا لواحد من أهم أجزاء الجهاز الهضمى الا وهو المرئ مما يؤثر تأثيرا مباشرا على عملية بلع ووصول الغذاء إلى المعدة حيث أن الإصابه تترك آثارا شديدة تؤدى إلى حدوث ضيق فى المرئ يصعب علاجه مما ينتج عنه إما وفاة الطفل أو حدوث عجز لأحد الأجزاء الحيويه من جسده .

وحيث أن أحد هذه المواد الكيماويه - الا وهو البوتاس - يوجد بصورة شبه دائمه فى أغلب بيوت مجتمعنا حيث يستخدم فى عمليات الغسيل والنظافه فإن هذه الرساله تقوم بدراسة جميع الأسباب والعوامل المحيطه بحدوث الإصابه .

* نسبة الإصابه فى الأطفال بالنسبه للسن والجنس .
* درجة الإصابه فى الحالات الحاده "الحديثه" والحالات المزمنه (القديمه) .
* وسائل التشخيص والعلاج وجدواها
* نسبة الوفيات والآثار الجانبيه الناتجه عن حدوث الإصابه مثل :
  - تأثير ضيق المرئ على النمو الجسمانى والعقلى للطفل
  - التأثير النفسى للمرض على الطفل وأسرته

وذلك من خلال الدراسه التى أجريت فى كل من :

١ - المجموعه الأولى :

بمستشفيات جامعة المنوفيه والمستشفى التعليمى بشبين الكوم - كمثال لمجتمع ريفى .

٢ - المجموعه الثانيه :

بمستشفيات جامعة عين شمس ودار الشفاء ومعهد ناصر - كمثال لمجتمع حضرى .

مع الأخذ فى الاعتبار مكان ومعيشة الطفل فى أى من الحضر أو الريف .

– وقد شملت الدراسه ١٠٠ مريض أصيب كل منهم بحروق كاويه بالمرئ وقد تم تقسيمهم الى مجموعتين :

المجموعة الأولى :

٥٠ مريضا منهم ٣٤ من الذكور ، ١٦ من الإناث يمثلون ٣٠ حاله حاده – ٢٠ حاله مزمنه.

المجموعة الثانيه :

٥٠ مريضا منهم ٣٢ من الذكور ، ١٨ من الإناث يمثلون ٣٠ حاله حاده ، ٢٠ حاله مزمنه .

وبدراسة الأصابه من الناحيه الوبائيه وجد أن :

* نسبة الإصابه فى الأطفال الذكور ٦٦٪
* نسبة الإصابه فى الأطفال الإناث ٣٤٪.

الغالبيه العظمى للإصابه فى الجنسين قبل سن الثالثه وتقل النسبه بصوره ملحوظة بعد سن الخامسه حيث أن السن مابين الثانيه والخامسه هى سن حركة الطفل داخل المنزل مع مايميزه من حب الإستكشاف والإطلاع وعدم المعرفه الكافيه بخطورة الأشياء .

وتمثل مادة البوتاس أكثر المواد تسببا لحدوث الإصابه نتيجة لوجودها فى المنزل بصفة دائمه مع عدم وجود رائحه لها كما أنها لاتسبب آلاما شديده فور ابتلاعها بالإضافه لكونها شبيهه باللبن .

كما أكدت الدراسه عدم حرص الأسره على إبعاد مثل هذه الكيماويات عن متناول الأطفال مما يزيد نسبة الإصابه بنسبه ٨٤٪ . كما أن جهل الأسره بخطورة هذه المواد وعدم معرفتها بآثارها المدمره أو بحدوث حالات مماثله فى أحد الأقارب أو الجيران يزيد نسبة الإصابه بنسبة ٧٢٪ من كلا من المجتمعين .

كذلك تعود الأسره على إستخدام هذه الكيماويات يزيد الإصابه بحروق المرئ بنسبة ٨٣٪ : ٨٤٪ فى الريف ، ٨٢٪ فى الحضر .

كان للمستوى التعليمى تأثير كبير فى درجة حدوث المرض حيث أن نسبته تقل بدرجه شديده عندما يكون رب الأسره متعلما تعليما عاليا حيث أنه يكون على درايه بخطورة مثل هذه المواد فى حين تزيد النسبه فى الأطفال لآباء غير متعلمين بنسبة ٣٩٪ : ٤٤٪ فى الريف ، ٣٤٪ فى الحضر .

كما تبين أن نسبة الإصابه تكثر حينما تكون الأم تعمل خارج المنزل حيث يقل الاهتمام بالطفل وتكون نسبة حدوث الإصابه ٦٨٪ : ٧٠٪ ريف ، ٦٦٪ حضر .

كما أن للمستوى الإقتصادى للأسره أثر كبير فى حدوث الإصابه حيث تزيد فى الأسر ذات المستوى الإقتصادى المتوسط وكلما زاد عدد الأطفال فى الأسره حيث تقل الرعايه وتزيد نسبة الإصابه خاصه إذا زاد عدد الأطفال عن ٣ أطفال بنسبة ٦٤٪ : ٦٢٪ حضر ، ٦٦٪ ريف ويحدث نفس الشئ إذا تأخر ترتيب الطفل فى الأسره حيث تكثر الإصابه إذا كان ترتيب الطفل متأخرا .

ومن دراسة الحاله الإجتماعيه للأسره فإن حدوث طلاق أو وفاة أحد الآباء أو أحدهم بالخارج أو بالسجن يزيد من نسبة الإصابه .

أما بالنسبه لوضع الطفل فى دور الحضانه فهذا يختلف تبعا لنوع هذه الدار فاذا كانت تابعه للعمل أو مؤهله لهذا الغرض فتقل نسبة الإصابه لعدم وجود مثل هذه المواد الكاويه . أما اذا كانت هذه الدار فى منزل يتم استخدام جزء منه كحضانه فتكثر الإصابه لإحتواء هذا المنزل على هذه المواد وكذا فان ترك الطفل عند الجيران أو الأقارب حيث يقل الاهتمام والرعايه ، تزيد نسبة الإصابه بنسبة ٣٦٪ : ٣٨٪ ريف ، ٣٤٪ حضر .

أما بالنسبه للأطفال الذين يظلوا بالمنزل مع ذويهم فإن نسبة الإصابه تكون ٢٨٪ فى المجتمعين وهى نسبة قليله بالمقارنه بنسبة تكرار هذا النمط داخل المجتمع .

وقامت الرساله بدراسة الناحيه المرضيه الباثولوچيه ، وكانت درجة الإصابه فى الحالات الحاده من الدرجة الثانية بنسبة ٥١٫٦٨٪ .

أما فى الحالات القديمه المزمنه التى تؤدى إلى حدوث ضيق فى المرئ فكانت درجة الإصابه

وتقدم الرساله بالتوجيهات التاليه :

١ - الإهتمام بالإصابات الناتجة عن تناول المواد الكاويه ومايتتج عنها بإعتبارها من الأمراض المزمنه وإعتبار المصابين بها من الأطفال المعاقين المطلوب شملهم بالرعايه الطبيه والإجتماعيه التى تشمل باقى إعاقات الطفوله .

٢ - تكثيف التوعيه من خلال وسائل الإعلام المختلفه من خلال حمله قوميه للتعريف بخطورة هذه الإصابه ومضاعفاتها والأهم الوقايه منها من خلال تعميق مفاهيم السلوك الشخصى للأطفال وتجنب الإصابه بهذا المرض من خلال المدارس وأيضا الجامعات .

٣ - التوصيه للشركات والمصانع التى تنتج مثل هذه المواد الكيمائيه بأن تضع علامات شديدة التحذير على العبوات وإضافة لون أو طعم أو رائحه منفره لمادة البوتاس المستعمله فى المنازل بحث لا تؤثر على خواصها التى تستخدم من أجلها .

٤ -- الإهتمام بالأسره ككيان إجتماعى من ناحية عدد الأفراد وتنظيم النسل وتقوية الروابط الأسريه والعلاقه السوه بين الآباء والأمهات .

٥ - نشر إنشاء دور الحضانه فى أماكن عمل الأمهات لتجنب تركهم فى المنازل فترة غياب الأم .

٦ - فى حالة الاصابه يجب توعية الأفراد وكذلك الأطباء فى الوحدات بعد عمل الإسعافات الأوليه بضرورة تحويل المريض الى مراكز متخصصه والضغط على الأسره فى ذلك حتى يتم عمل المناظر والتوسيع للمرئ وكذا التدخل الجراحى فى المراحل الأولى .

٧ - توفير الرعايه الإجتماعيه للأسره التى يكون بها طفل مصاب وتحمل الجهات الحكوميه الإجتماعيه والأهليه الخيريه لنفقات الانتقال والاعاشه والإقامه للطفل الذى ينقل للمراكز المتخصصه .

٨ - الاهتمام بالرعايه النفسيه والتعليميه والمهنيه للطفل المعاق بضيق المرئ من خلال المتخصصيين مع زيارات ميدانيه لأماكن دراستهم وتدريب المشرفين والمدرسين على وسائل الرعايه والمعامله الخاصه لهم .

٩ - توفير العمل المهنى المناسب للطفل المعاق بما لايؤثر على صحته وحتى لايكون عبئا على اسرته وكى يستطيع إعالة نفسه مع تقدم العمر .

جامعة عين شمس

معهد دراسات الطفولة

الدراسات الطبية

مستخلص

عن الرسالة المقدمة من

الطبيبة / نجوى محمد المغاورى ابراهيم نصار

للحصول على

درجة دكتوراه الفلسفة فى دراسات الطفولة

قسم الدراسات الطبية

عنوان الرسالة

دراسة التأثير النفسى والنمو للحروق الكاوية للمرئ فى الأطفال ودراسة مقارنة بين الريف والحضر

تدور الرسالة حول تاثير المواد الكاوية وخاصة البوتاس الدي يستخرم بكثرة فى البيوت المصرية على الاصابات الخطيرة التى بسببها وخاصة ضيق المرئ مما يسبب صعوبة شديدة في البلع وتناول الغذاء اللازم للطفل وكذا تأثير ذالك علي النمو الجسمانى والعقلى والنفسى ودرجة الذكاء والتقدم الدارسى فى المدرسة ، حيث اثبتت الدراسة من أن هذه الاصابة تؤدى إلى عجز لصعوبة علاج مثل هذه الحالات وقامت الرسالة بدراسة أسباب وعوامل الاصابة وذلك فى جميع حالات البحث التى تشمل ١٠٠ مريض ٥٠ مريض من الحضر و٥٠ مريض من الريف وذلك بالنسبة للسن والنوع ودرجة الاصابة وأسباب كثرة الاصابة في مختلف الأسر من حيث دراسة مستوى معيشة الأسرة وكذلك الحالة الاجتماعية والاقتصادية والتعليمية للأسرة وكذلك كثرة استخدام الأسرة للمواد الكاوية فى الأغراض المنزلية واثبتت الدراسة أن الاصابة تكثر فى الأسر ذات المستوى الاقتصادى المنخفض وكذلك عدم استقرار الحالة الاجتماعية للأسرة.

ثم قامت الباحثة بدراسة عدد ٦٠ مريض ٣٠ من المجتمع الحضري ، ٣٠ من المجتمع الريفي بدراسة الحالة المرضية للاصابة وطرق العلاج المبكر بالكورتيزون والتوسيع بالمناظير وكذلك تاثير الاصابة على النحو الجسمانى والنفس والعقلى للطفل حيث اثبتت الدراسة تأخرهما لعوامل الاصابة المزمنة التى تسبب اعاقة للطفل ، واكدت الدراسة اهمية العلاج المبكر بالكورتيزون

والتوسيع بالمناظير وكذلك تأثير الاصابة على النمو الجسمانى والنفسى والعقلى للطفل حيث اثبتت الرداسة تأخرهما لعوامل الاصابة المزمنة التى تسبب اعاقة للطفل ، وأكدت الدراسة أهمية العلاج المبكر بالكورتيزون والتوسيع بالمناظير فى تقليل درجة الاصابة وسرعة الشفاء

وقد أوصت

الرسالة بالتوجيهات التالية :

١- الاهتمام بالإصابات الناتجة عن تناول المواد الكاوية وما ينتج عنها باعتبارها من الأمراض المزمنة واعتبار المصابين بها من الأطفال المعاقين المطلوب شملهم بالرعاية الطبية والاجتماعية التى تشمل باقي اعاقات الطفولة.

٢- تكثيف التوعية من خلال وسائل الاعلام المختلفة من خلال حمله قوميه للتعريف بخطورة هذه الإصابة ومضاعفاتها والأهم الوقاية منها من خلال تعميق مفاهيم السلوك الشخصى للأطفال وتجنب الإصابة بهذا المرض من خلال المدارس وأيضا الجامعات.

٣ ـ التوصية للشركات والمصانع التى تنتج مثل هذه المواد الكيمائية بأن تضع علامات شديدة التحذير على العبوات وإضافة لون أو طعم أو رائحة منفرة لمادة البوتاس المستعملة فى المنازل بحيث لا تؤثر على خواصها التى تستخدم من أجلها.

٤ ـ الإهتمام بالأسرة ككيان إجتماعى من ناحية عدد الأفراد وتنظيم النسل وتقوية الراوبط الأسرية والعلاقة بين الأباء والأمهات.

٥ ـ نشر إنشاء دور الحضانة فى أماكن عمل الأمهات لتجنب تركهم فى المنازل فترة غياب الأم.

٦ ـ فى حالة الإصابة يجب توعيةالأفراد وكذلك الأطباء فى الوحدات بعد عمل الإسعافات الأولية بضرورة تحويل المريض الى مراكز متخصصة والضغط على الأسرة فى ذلك حتى يتم عمل المناظيروالتوسيع المرئ وكذا التدخل الجراحى فى المراحل الأولى.

٧ ـ توفير الرعاية الإجتماعية للأسرة التى يكون بها طفل مصاب وتحمل الجهات الحكومية الإجتماعية والأهلية الخيرية لنفقات الإنتقال والإعاشة والإقامة للطفل الذى ينقل للمراكز المتخصصه .

الكلمات الدالة :

١- المواد الكاوية ، ٢- وسائل الاعلام ٣- الاهتمام بالمناظير

" جامعة عين شمس "
الكليـة :

شكـــــــــــــر

أشكـــر الســادة الأســاتذة الــذين قامــوا بالاشــــراف وهــم :

١) . . أ.د/ حامد محمود شعلة
٢) . [illegible]
٣) . . . [illegible]
٤) . . . .

ثـم الأشـخـاص الذيــن تعــاونوا معــي فــي البحــث وهــم :

١) . . [illegible]
٢) . . . [illegible]
٣) . . . . [illegible]

وكـــذلك الهيئـــات :

١) . . [illegible]
٢) . [illegible]
٣) . . . [illegible]

" جامعة عين شمس"
الكليـــة:

رسالـــة ماجســـتير / دكتـــوراه

أسم الطـــالب:.....
عنوان الرسالة:.....

أســم الدرجـــة : (ماجستيـــر / دكتـــوراه)

لجنـــة الإشـــراف

١- الاسم/ .....
٢- الوظيفة/.....
١- الاسم/ .....
٢- الوظيفة/.....
١- الاسم/ .....
٢- الوظيفة/.....

تاريخ البحـــث : / / ١٩٩٦

الدراســـات العليـــا
ختـــم الإجـــازة :
/ / ١٩٩

أجيزت الرسالـــة بتاريخ ٢ / ٥ / ٩٦

موافقة مجلس الكلية
/ / ١٩٩٦

موافقة مجلس الجامعة
/ / ١٩٩

"جامعة عين شمس"

الكليـــة:

صفحـــة العنـــــوان

أســـم الطالــــب : نجوى محمد عبد القادر الصابر

الدرجـــة العلميـــة : دكتوراه الفلسفة

القسم التابع لـــــه : القسم الطبي

أســـم الكليـــــة : معهد دراسات العلوم

الجامعــــــــــــة : عين شمس

سنـــة التخــــــرج : ١٩٧٣

سنـــة المنـــــــح : ١٩٩٩

شــــروط عامـــــه

يوضع شعار الجامعة على الغلاف الخارجي

بسم الله الرحمن الرحيم

قالوا

سبحانك

لاعلم لنا إلا ماعلمتنا

إنك أنت العليم الحكيم

جامعة عين شمس
معهد الدراسات العليا للطفولة
قسم الدراسات الطبية

دراسة التأثير النفسى والنمو للحروق الكاوية للمرىء فى الأطفال ودراسة مقارنة بين الريف والحضر

رسالة مقدمة من

الطبيبة/ نجوى محمد المغاورى الشيخ

لنيل درجة دكتوراه الفلسفة

فى دراسات الطفولة

معهد دراسات الطفولة – الشعبة الطبية

جامعة عين شمس

المشرفون

أ.د. / حامد شتله

أستاذ طب الأطفال

كلية الطب – جامعة عين شمس

جامعة عين شمس

١٩٩٦