PHYSICAL GROWTH IN ASTHMATIC CHILDREN

Thesis

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Presented by

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CONTENTS

INTRODUCTION AND AIM OF THE WORK

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Bronchial asthma is a major respiratory disease affecting approximatly 6 millions of children below the age of 12 years in the United States and has a significant morbidity and mortality. However, the true incidence of asthma is unknown and can be only estimated indirectly (Speizer,1968).

Asthma is responsible for a significant proportion of school days lost because of chronic illness.

Asthma can lead to severe psychosocial disturbances in the family. With proper treatment, however, much relief can be provided.

Underweight, understature and retardation of bone age have been reported in association with bronchial asthma (Von Metre et al.,1960 ; Falliers et al.,1961 ; Spock,1965).

Aim of the work :

This study aims to :

1) Assess the physical growth of children suffering from bronchial asthma.

2) Compare the physical growth of asthmatic children with that of non asthmatic ones of the same sex, age and socio-economic status.

BRONCHIAL ASTHMA

DEFINITION OF BRONCHIAL ASTHMA

Bronchial asthma is a complex disorder,which cannot be defined adequately in terms of a single pathophysiological mechanism, and there is no universal agreed definition of the word asthma (Proter & Birch,1971).

In 1962,the Committee of the American Thorathic Society defined asthma as a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli and manifsted by widespread narrowing of the airways that change in severity either spontaneously or as a result of treatment.

Scadding (1976) suggested that asthma is a disease characterized by wide variations ,over short periods of time, in resistance to flow intrapulmonary airways.

The basic problem in asthma is hyper-reactivity or "twitchiness" of the airways, causing the subject to develop bronchospasm in response to variety of stimuli (Burrows, 1979).

Bronchial asthma can be also defined as a recurrent generalized airway obstruction which ,at least in the early stages, is paroxysmal and reversible (Crofton & Douglas , 1981).

INCIDENCE OF BRONCHIAL ASTHMA

The true incidence of asthma is unknown and can only be estimated indirectly (Speizer,1968).

The prevalence figures for asthma are very crude,and comparison between different surveys subjects to wide margin of error (Clark & Godfrey,1977). The published figures suggest an asthma prevalence of 2.5% in primary achool children (Smith,1961). In Egypt, the incidence of asthma among diseased children presenting to out-patient clinics of Children's Hospital,Cairo University, was found to be 2.2% (El Hefny, 1966).

Age of Onset:

Most serveys in Britain,North America and Australia have found that, in at least 30% of patients asthma began before the age of 10 years. In Scandinavia,India and Nigeria a childhood onset has been much less common (Clark & Godfrey, 1977).

Sex Incidence:

The great majority of surveys have found a male excess of asthma in childhood 1.5 : 1, and tendency to decrease as adolescence is approached (Blair,1977). In adults there is little differences between sexes (Derrick,1971).

In general, up to the age of 15 years,about 2.3% of boys and 1.2% of girls have asthma (Rhyne,1974).

PATHOLOGY AND PATHOGENESIS OF ASTHMA

The main factors contributing to bronchial obstruction are the following :

(1) Bronchospasm :

bronchial muscle contraction is the most important component in the attack of the paroxysmal asthma (Crofton & Dougla,1981). Contraction of the bronchial muscle in response to a specific allergen has been shown experimentally in human lungs resected from patients with allergic asthma (Schild et al.,1951).

(2) Swelling of the mucous membrane :

At autopsy, the bronchial epithelium of patients dying from status asthmaticus was found shed, thaugh the basement membrane was thickened with submucosal oedema and infiltration with eosinophils (Spencer,1977).

(3) Plugging with viscous mucous :

Wanner and co-workers (1975) found an increase in the mucous glands and goblet cells, with plugging of the peripheral bronchi with viscid mucous in patients dying from status asthmaticus. This may be due to inability of the bronchial muscles to relax or replacement of ciliated cells by goblet cells. Less effective ciliary movement may also interfere with clearing mechanism,

(4) **Invagination of posterior mucous membrane** between the tips of semilunar cartilages of the intrathoracic trachea and the large bronchi on expiration, may play a rule in bronchial obstruction (Groen, 1976).

The pathogenesis of asthma is poorly understood. It may be due to: immunologic mechanisms, release of chemical mediators, pharmacological abnormalities or reflex pathways. All acting singly or in combination (Hinshaw, 1980).

I **Immunologic Mechanisms** :

Hypersensitivity reaction may be regarded as an exaggeration or distortion of a protective immunological process resulting in adverse manifestations in the individual (Kaltreider, 1976). (Bellanti 1985 a)

Bellanti 1985 a

Gell & Coombs (1963) classified hypersensitivity reactions into 4 distinct types ; 2 of them mainly are concerned in asthma, namely type I and type III.

Type I hypersensitivity reaction :

This is an immediate or anaphylactic reaction, starting 10 to 20 minutes after exposure to the allergen in atopic response. It may be local reaction (atopy) as in skin e.g. dermatitis or in lung e.g. bronchial asthma , or it may be a generalised reaction leading to true anaphylaxis. This type of hypersensitivity reactions is mediated by immunoglobulin E (Ig E), which is synthesized in response

to exposure to a specific allergen, and gets attached to the surface of the mast cells. On subsequent exposure, the allergen combines with its specific IgE on the cell surface, causing the release of mediators from the granules of the sensitized mast cells (Hinshaw, 1980).

Mathews 1982 (6)

Stenius & co-workers (1971) reported a highly significant correlation between the presence and the amount of specific IgE against common allergens, such as grass pollens, and reaction they provoke in an inhalation test and clinical history of asthma. They stated that type I response can explain the majority of short lived attacks of asthma.

Johanson (1967) reported significantly raised serum levels of IgE in 63% of patients with allergic, as compared to 5% with non allergic asthma.

Sharaf El-Din (1982) found a significantly higher level of serum IgE in asthmatic than normal control children, with no significant difference between atopic and non atopic asthma.

Type III hypersensitivity reaction :

This is also called immune complex or arthus reaction. Immune complexes are aggregates of antigen and antibody with or without complement. The antibodies concerned are IgG and IgM. Antigens that evoke type III asthmatic reactions are numerous e.g. fungus Aspergillus fumigatus,

bacterial infections, drugs, wood dust, vapors and fumes (Colen et al., 1964).

Hargreave & Pepys (1972) suggested a role of type III reaction in late asthma when they observed a disproportionate fall in the forced expiratory volume in the first second (FEV_1), 4-6 hours after challenge, during the course of bronchial challenge studies on asthmatic patients.

II Chemical Mediators :

The physiologic consequences of exposing IgE-sensitized mast cells to antigen, against which the IgE molecule is directed, result from secretions of mast cell granules, from which derived chemical mediators of anaphylaxis (Metcalf et al., 1981). These chemical mediators may be preformed mediators, which are contained in the granule matrix (as histamine) and are released into the tissue fluid immediatly after reaction ; or they may be secondrily formed mediators, generated by interaction of primary mediators and nearby cells and tissues (e.g. prostaglandins).

Among the mast cell-derived mediators, those which are capable of causing bronchial smooth muscle contraction are :

1- Histamine :

Two cellular receptors for histamine have been identified, designated H_1 and H_2. Histamine-induced airway obstruction occures through stimulation of H_1

receptors on muscle fibers. In addition, there may be a vagally-mediated reflex parasympathetic action. Histamine also dilates the small vessels of pulmonary vascular tree, through an H_1 response, thus increasing the distance between endothelial cells of the venules, thereby increasing the potential for transudation of plasma for extravasation of leucocytes (Rosenthal et al., 1977).

2- Slow-reacting substances of anaphylaxis (SRS-A) :

SRS-A are though to be important mediators in man. The maxium effect on bronchial muscle is reached more slowely and is much prolonged than that of histamine.

SRS-A are composed of leukotreines (LT) C,D and E. These are pipridolipids derived from arachidonic acid, through lipoxygenase pathway. Studies on human bronchial muscle indicate that LTC and LTD are 1000 times more potent than histamine, and 500 times more potent than prostaglandin F_2 (Dahlen et al., 1980).

Weiss et al., (1982) stated that in normal persons LTC and LTD are the most potent bronchoconstrictor substances yet described, and that their prolonged duration of action is consistant with a possible role in mediation of IgE-mediated bronchoconstriction.

3- Prostaglandins (PGs) :

These are complex interacting group derived from products of arachidonic acid metabolism via a cycloxygenase-dependant pathway. The exact role of PGs in asthma is still unclear. Hyman (1978) reported that PGs form a complex interacting group, some members of which (PGE series) relax the bronchial muscle, while others (PGF series, specially $PGF_{2\alpha}$, PGD and Thromboxan A_2) are bronchoconstrictors.

Serum levels of PGF_2 and its metabolites have been shown to be higher in asthmatic attacks, and also an increase in PG metabolites have been found in urine (Green, 1974).

PGs are generated during the course of anaphylaxis. Through stimulation of H_1 receptors, histamine is responsible for about 50% of PG generated during anaphylaxis. On the other hand, bronchoconstriction selectively causes PGE generation, irrespective to the cause of the muscle contraction (Platshon & Kaliner, 1978).

III Pharmacological Abnormalities :

Szentivang (1968) suggested that the basic abnormality in asthma was impaired beta adrenergic responsiveness. This was termed the beta adrenergic theory. This theory was supported by Smith et al. (1980), who reported β_2 adrenergic hyporesponsiveness in atopic subjects if compared to control group.

The beta adrenergic theory had led to considerable investigations of the possible role of cyclic 3,5-adenosine monophosphate (cAMP), which is one of the major intracellular massengers, and its balance with cyclic guanidine monophosphate (cGMP) is of an atmost importance. Lowering of cAMP relative to cGMP contracts bronchial smooth muscles and releases histamine and other mediators from the mast cells ; on the other hand, raising of cAMP has the converse effect (Kaliner,1977). (Weiss et al 1982 ⑥)

Cyclic AMP is formed from adenosine triphosphate (ATP) by the action of adenyl cyclase enzyme, which constitutes a part of the beta receptors. The increase of adrenaline and the action of adrenergic drugs and certain prostaglandins (PGE), increase the adenyl cyclase and consequently cAMP with relaxation of smooth muscles and inhibition of release of histamine and other mediators from the mast cells. On the other hand, phosphodiestrase is another enzyme which is responsible for the breakdown of cAMP. It is through inhibition of that enzyme originated the bronchodilating effect of xanthines (Linchtenstein & Austen,1977). (Leff 1982 ⑥)

Calcium ions are another type of cell massengers through interaction with cAMP. Release of histamine from mast cells is mediated by an influx of calcium ions. This process is inhibited by cAMP. Moreover, calcium ion transport may be involved in the nervous stimulation of mucous glands (Morley,1977). Leff 1982 ⑥

IV Reflex Mechanism :

The vagus nerve plays an important role in regulating airway muscle tone in normal subjects, as well as asthmatics. Histamine causes both direct and reflex airway narrowing as a result of stimulation of irritant receptors. Chemicals, dust and mechanical irritation cause bronchospasm, via a vagal reflex, in subjects with bronchial hyperreactivity (Gold et al., 1972). Nadel 1983 (R)

Triggering Stimuli Causing The Attacks Of Asthma :

A wide variety of stimuli may be responsible for induction of asthmatic attacks including :

1) Allergic factors :

These factors are important, specially in children with a history of other allergic diseases. Inhalents, such as pollens, house dust, feathers, animal hair or dander, and fungi are the commonest precipitating factors.

House dust mite (Dermatophagoides) is the commonest allergen and the most important allergic component of house dust. The mites feed on human skin peelings and are found in mattresses, pillows, blankets and carpets. Prick tests and bronchial challenge test are both positive in many asthmatic to house dust mite (Warner, 1978). Bellanti 1985 (R)

Sensitivity to food allergens such as eggs, wheat, fish, meat, caw's milk or chocolate are common accusants for asthma by lay people. However, food as a sole precip-

(Malkhou 1983 (6))

itating factor in asthma is rare (Chobot et al.,1951).

2) **Infection** :

Viral infection , specially rhinovirus, predisposes to wheezy attacks in asthmatic children. In adults,viral infection may be associated with asthmatic attacks. The significance of bacterial infection is still uncertain, but both may have a non apecific effect in sensitizing irritant receptors (Gregg,1977). (Loff 1982 (6))

3) **Physical factors** :

Exercise-induced asthma is a well known clinical type in which exercise aggrevates or induces bronchoconstriction in many asthmatics. (Kid 1983 (6)) Jones et al.(1962) reported bronchoconstrition together with decreased expiratory flow rates, few minutes after stopping exercise and persisting for 10 minutes with gradual recovery in 30 to 60 minutes.

4) **Reflex factors** :

(Malkison 1985 (6))

These are not well established. Swineford (1962) stated that an attack of asthma was induced by inflating a balloon inserted into a maxillary sinus through an antrostomy opening.

5) **Psychological factors** :

Emotional stress can alter airways caliber in subjects with asthma. Attacks of asthma can be precipitated by psychological upset, althaugh it is doubtful whether

psychological upset alone is ever the only factor responsible for a patient having asthma (Dekker & Groes, 1956).

6) **Parasitic infestations** :

These have been long linked with asthmatic attacks in endemic areas, yet there is no solid evidence that infestation may cause asthmatic attacks. Sami (1951) stated that asthma in bilharzial subjects is an associated condition and not causally related.

COMPLICATIONS OF BRONCHIAL ASTHMA

1) Thoracic deformity :

Chronic asthma in the very young children draws the sternum inward, or there may be a Harrison's sulcus. Older children tend to develop a pigeon chest (Derbes et al.,1951).

2) Pneumonia and atelectasis :

Segmental pneumonic or atelectatic shadows may be found in the X-ray of the chest during excerbations, specially those caused by infection (Miller et al.,1960).

3) Bronchitis :

This is the commonest complication of asthma ; it is associated with a much poorer prognosis (Ogilnic,1962).

4) Spontaneous pneumothorax :

This is relatively a rare complication, but does occure from time to time.

5) Interstitial pulmonary or mediastinal emphysema :

These complications may occasionally occure, and are sometimes associated with subcutaneous emphysema.

6) Cardiac complications :

Increased pulmonary pressure has been noted in occasion (Bates et al.,1971). Derbes et al.,(1951) reported that right ventricular hypertrophy is present in most patients at

autopsy. Cardiac failure usually occures only in very severe and prolnged status asthmaticus.

7) Psychological troubles :

Emotional disturbance is manifested in the child's behaviour in a number of ways : excessive anxiety, immaturity, lack of self confidence, dependancy and depression (Pilling, 1979).

CLASSIFICATION OF BRONCHIAL ASTHMA

From the immunological point of view, two subgroups of asthmatics (extrinsic and intrinsic) can be identified (Rackemann, 1947).

I Extrinsic Asthma :

Due to a specific external allergen. It can be further subdivided according to whether the subject is atopic (i.e. manifested by demonstrable type I skin reaction to a standard range of common allergens) or not.

1) Atopic extrinsic asthma :

Symptoms of this type tend to appear early in life (Dees, 1957). Many of these cases remit spontaneously (Rackmann & Ewards, 1952). Williams & Mc Nicol (1969) found that nine out of ten childern with atopic extrinsic asthma have greatly diminished attacks or are cured by the time they reach their teens. The fact of natural remission in childhood asthmatics can be explained by the theory of transient immune defect, which allows IgE to be formed in early years, but with a gradually lessening tendency to do so later on. The fact of lessening atopic responsiveness with age is also seen in data relating age of onset of symptoms of asthma to the number of skin test responses to common allergens. Patients with later onset of asthma, in general, having fewer positive skin tests (Handrick et al., 1975).

2) **Non atopic extrinsic asthma** :

In this type,patients develop their symptoms in relation to some particular agents, offten after exercise. These patients have negative skin tests to the standard range of common allergens, but may show an immediate, late or dual response to specific sensitizing agents. Specific IgE and IgG antibodies may be demonstrable, but whether these are observed or not in an individual case largely depends upon how for suitable and purified antigen have been prepared (Stenius et al.,1971). patients with extrinsic non atopic asthma tend to develop their symptoms later in life (Pepys,1969).

II **Intrinsic (Cryptogenic) Asthma** :

This group have been suggested by Rackmann (1974), when he noticed that in some patients presenting with typical asthma, no evidence of extrinsic allergen is obtained from the clinical history and prick testing with a standard range of common allergens.

There are conflicting views as to whether cryptogenic asthma has a different immunological and/or genitic bases from extrinsic asthma. Nasal polypi and aspirin sensitivity are traditionally regarded as more common in cryptogenic asthma (Samter and Beers,1967).

DIAGNOSIS OF BRONCHIAL ASTHMA

The most important clinical manifestations of bronchial asthma are dyspnea and wheeze, althaugh in severe asthma the obstruction may be so great that there is no audible wheeze (Crofton,1981).

In a trial to study the relationship between wheezing and the degree of bronchial obstruction, Shim & William (1983) found that expiratory wheezing was usually accompanied by inspiratory wheezing ; this biphasic wheezing was associated with a lower peak expiratory flow rate (PEFR) than only expiratory wheezing. Loudness and high pitch of wheezing were associated with more severe obstruction. Tney also found that expiratory or inspiratory wheezing of high pitch, moderate to severe intensity and spanning the entire phase of breath was associated with lower PEFR than wheezing without those characteristics. Pulmonary function tests specially vital capacity and FEV/FVC studies are important.

Increased airway resistance is the basic physiologic abnormality in bronchial asthma, chronic bronchitis and emphysema. Intravenous aminophylline causes significant decrease in pulmonary resistance,and early increase in the dynamic compliance were characteristic of patients with asthma and bronchitis (Pande,1970). Patients with bronchial asthma showed greater decrease in inspiratory resistance than patients with chronic bronchitis, and greater decrease in both inspiratory and expiratory resistance than patients with chronic

bronchitis and emphysema. Morton & Turnbull (1968) reported a significantly greater improvement in maximal breathing capacity (MBC) after a bronchodilator in asthmatic subjects compared to patients having chronic bronchitis with or without emphysema.

However, Brown (1984) reported that asthma alone can cause irreversible airway obstruction and that the degree of obstruction is a function of the duration and severity of previous asthma.

DRUG TREATMENT OF BRONCHIAL ASTHMA

The aim of drug treatment is to reduce the symptoms and also to reverse , at least in part, the disease process towards normality. Many drugs are used for this purpose and each has a certain mode of action.

Drugs used in treatment of bronchial asthma can be classified in various ways, but the following groups of agents were readily identified by Zeiment (1978):

I **Bronchodilators** :

A) **β_2 adrenergic receptor stimulators** :

1- **Selective β_2 agonists** : Which include salbutamol (ventoline), terbutaline (bricanyl) and hexoprenaline.

2- **Non selective β receptor agonists** : As adrenaline, isoprenaline and orciprenaline (alupent).

B) **Phosphodiesterase inhibitors** :

1- **Theophylline** and its derivatives (Methyl xanthines).

2- **Quazodine** : A drug related to papavereine, and has been shown to be 18 times as active as theophylline.

C) **Anticholinergic drugs** :

As atropine and related drugs as intratropium.

D) **Prostaglandins** :

Bronchodilatation by PGE_1 and PGE_2 is probably mediated through cAMP mechanism ; the prostaglandin

receptors being in some ways linked to the beta adrenergic receptors.

E) **Other bronchial muscle relaxants** :

As khellin , pitutary extracts , L-dopa , glucagon and hexamethonium. These drugs are of little value in asthma compared to their side effects.

II **Antimediator Drugs** :

A) **Glucocorticoids.**

B) **Antihistaminics.**

C) **Bischromones** :

As khellin and cromolyn.

D) **Immunosuppressive drugs** :

Cytotoxic drugs which have attained some success in the treatment of asthma ,when other mrasures fail, include the purine analogues azathioprine (imuran) , 6-mercaptopurine and thioguanozine (Mc Combs,1972).

E) **Anti-inflamatory agents** :

As asprin (acetyl salicylic acid) and other analgesic antipyretic agents as phenazone, aminophenazone, phenylbutazone and flufenamic acid. Althaugh asprine and related analgesics have achieved notoriety as aetiologic agents in some forms of atopic asthma, there are asthmatic patients who appear to benefit from the prophylactic use of asprin. Since salicylates have profound

effect on immunologic processes and interfere with prostaglandin production, it is concievable that in some patients asprine has relatively more effect on the activity of $PGF_{2\alpha}$, and thereby interfere with prostaglandin-induced bronchospastic process.

F) **Diethyl carbamazine (Heterazan)** :

It is an antihelminthic drug related to piberazine. It appears to be comparable to cromolyn, both in its clinical effectiveness and its mechanism of action as an antimediator.

III Agents Affecting α Receptors :

A) α-adrenergic receptor stimulants :

Stimulation of alpha receptors activates cGMP production, which causes bronchospasm and increases the airway resistance. However, stimulation of α receptors supplying pulmonary blood vessels results in vasoconstriction ; if the mucosa is inflamed and boggy, the resulting mucosal shrinkage may outweigh the α-adrenergic bronchospasm and results in a balance that decreases the airway resistance. Thus these drugs may improve the airway dynamics in bronchial asthma, but they are mainly of benifit in treatment of upper respiratory tract mucosa. Examples of these drugs are ephedrine, methoxamine, cyclopentamine and naphazoline.

B) **α- adrenergic blockers** :

They decrease the α- adrenergic tone in bronchial muscle, and this leads to decrease in airway resistance. This action may be due to a decrease in ATPase activity which leads to decreased cGMP production. Examples of these drugs are phentolamine, tolazoline and phenoxybenzamine.

IV **Miscellaneous Drugs** :

A) Mucokinetic agents.

B) Disodium chromoglycate (Intal).

C) Antimicrobial agents.

D) Cough medications.

E) Tranquillizers.

F) Gases: Oxygen and Helium.

GROWTH AND DEVELOPMENT

DEFINITION OF GROWTH AND DEVELOPMENT

Growth and denelopment represent a continuous interaction of biologic processes that are iniated at conception & terminate at death (Hughes et al.,1980).

Growth implies changes in size resulting from multiplication of cells or enlargement of pre-existing ones, and it can be accurately measured (El Behairy,1977).

Development is the maturation of organs in their quality than quantity or size. It implies learning ability, acquisition of skills and adaptability to stress (El Behairy, 1977). It encompasses aspects of differentiation of form or function including those emotional or social changes preeminently shaped by interaction with the environment (Tanner, 1984). Measurement of development is much more difficult than that of growth.

Growth and development are continuous dynamic processes. In normal human being the increase in size of organs and tissues is associated with their functional maturation, i.e.growth and development run parallel to each other (Nassar,1975).

Different tissues and different regions of the body mature at different rates (Tanner,1984).

PARAMETERS OF PHYSICAL GROWTH

Physical growth is evaluated by measuring the indeces of the growth of the body mass i.e. weight and height or length. Other measurements are head circumference, chest circumference, upper arm circumference and various body proportions.

Height :

Growth in height ,like all other measurements, is not uniform throughout life. During foetal life, the rate of growth is extremely rapid. The newborn infant is found to be growing at a fast rate. The velocity slows gradually with time. In most children around the age of 3 years, the velocity curve decreases and comparatively a steady period of growth occurs. There is evidence of slight acceleration in the middle of this period in some children (Silver et al., 1975). Then, before puberty there is a further slight deceleration to prepare for the drammatic eminent period of great velocity and acceleration at adolescence, called the adolescent growth spurt. The growth spurt is followed by a period of slowing of growth. Girls gain 98 % of their final weights by the average age of 16.5, whereas boys reach the same stage by the age of 17.75 years. There is wide variations around this mean (Tanner et al.,1966).

Weight :

Body weight is the best index of growth and maturation

(Silver et al.,1976). The weight at birth reflects the maternal environment more than the heredity of the infant and is more variable than height (Sinclair,1973).

The greatest increase in the body weight occurs in the first year of life. Weight gain averages approximately 20 gram per day for the first five months of life, and approximately 15 gm/day for the remainder of the first year, the fullterm infant will generally double his birth weight by five months and triple it in one year (Nelson et al.,1983).

The weight increases in a relatively slower rate to be quadripled by the end of the second year, then the gain is relatively steady untill the onset of adolescent spurt. During the spurt, boys may add 20 kgm and girls 16 kgm to their weights (Tanner et al.,1966).

Head Circumference :

Measurment of head circumference is an essential part of paediatric examination, it is a good indicator for skull size and brain size. Growth of skull is much more accurate index for growth of brain than the presence or size of fontanels (Silver et al.,1975).

EVALUATION OF PHYSICAL GROWTH

Standard charts give the bases for comparison to know the normal from the abnormal, as the normal in growth is an area with illdefined boundaries and not a figure or a sharp line.

1) Standard deviation curve :

To show the degree of dispersion of observations around the mean. Mean ± 1 S.D. includes about 67 % of total number of observations, while mean ± 2 S.D. includes about 95 % of it.

2) Percentile growth curve :

Measurements are arranged in ascending or descending manner. The smallest measure corresponds to first percentile, while the greatest corresponds to the 100th percentile. The 50th percentile represents the median. Values below the 3rd percentile and above the 97th percentile are considered abnormal.

FACTORS AFFECTING GROWTH AND DEVELOPMENT

Growth is the outcome of interaction of genetic and environmental factors.

I **Hereditary Factors** :

1) **Age** : The greatest rate of growth is during intrauterine life, first year and adolescence.
2) **Sex** : At birth, males are slightly bigger than females (Abbassy et al.,1972). Females mature earlier starting adolescence at about 8 years, while males start it at about 12 years (Ellis,1966).
3) **Race** : There are racial differences in rate and pattern of growth, some of these differences are genetically controlled while others depend on nutritional and climatic factors (Watson & Lawery,1967).
4) **Family** : Some families tend to be short or tall.
5) **Inborn errors of metabolism** : e.g. in galactosaemia, vomiting, diarrhea and dehydration in infants after milk ingestion lead to malnutrition and growth retardation (Bockus et al.,1976).
6) **Chromosomal abbrations** : e.g. Down syndrome (trisomy 21) and Turner syndrome are associated with short stature (Reisman,1971).

II **Environmental Factors** :

1) **Socio-economic** : The lowest percentiles of size, skeletal and dental development are from the poorest families

(Garn & Clark,1974).

2) **Nutrition** : It is essential for optimal rate of growth and development, both prenatally and postnatally. Faulty nutrition during pregnancy may be a cause of still birth, prematurity and small for date babies (Holt,1968).

3) **Diseases** : Minor and relatively short illness as measles, middle ear infection and even pneumonia cause no discernible retardation of growth rate in the great majority of well-nurished children. Major diseases which take the child to the hospital for a month or more or keep him in bed for several months, may cause considerable slowing down of growth followed by catch-up when the disease is cured. The mechanism of retardation varies from one disease to another (Forfar & Arnil,1978).

4) **Endocrine glands** : They have an important effect on growth and development from physical, mental and emotional points of view. Hormonal imbalance, specially of growth hormone, insulin, glucocorticoids and thyroid hormones affect growth markedly. Hypothyroidism as well as hyperthyroidism cause growth retardation (Andersen,1975).

5) **Season** : The greatest increase in height is in spring, while weight gain is fastest in autum. Seasonal variation may be due to variation in diet, sleep and incidence of infections (Tanner & Whitehouse, 1966).

GROWTH PATTERN IN BRONCHIAL ASTHMA

Falliers et al.(1961) demonstrated statistically signigicant differences in height between asthmatic children and normal controls. They also stated that, some children with severe asthma were underweight for age even when they were not given corticosteroid therpy.

On the other hand, Cohen & Abram (1948) reported that, asthmatic symptoms of moderate degree did not seem in general to affect growth. Moreover, other investigators reported lack of any significant growth impairment in children with prolonged history of asthma (Mc Nicol et al.,1970).

The pathogenesis of growth retardation in asthmatic children is neither clear nor generally acceptable. Some investigators demonstrated that it is related to the severity of the asthmatic attack (Falliers et al.,1961). Some others considered that corticosteroid therpy of childhood asthma is the major factor responsible for growth retardation in these children (Boldgett et al.,1956 ; Von Metre et al.,1960 ; Spock,1965).

MATERIALS AND METHODS

MATERIALS AND METHODS

A) Choice of Samples :

This work was carried out on 100 children aging from 6 months to 12 years, all of them were of the same socio-economic status. This sample was divided into 2 groups :

1- Experimental group :

Included 50 children, 29 males and 21 females. Half of this group was below 6 years old, while the other half aged from 6 to 12 years. These children were chosen from the out-patient allergic unit of the New Children's Hospital, Cairo University. All these children were well-known to be asthmatics on the bases of full history, clinical examination and skin test which is done routinely in the out-patient allergic unit.

The cases of asthma were graded,in relation to severity, on the bases of history, measures used to relieve the asthmatic attacks as well as the need of hospitalization of the patient into 2 grades :

Grade I : Response to simple measures, no hospitalization.

Grade II : Only relieved by corticosteroids and/or hospitalization.

2- Control group :

Included 50 children of the apparently healthy, non asthmatic ones. One half of these children aged below

6 years, and were chosen from El Amal Nursary at El Monira. The other half aged from 6 to 12 years, and were chosen from El Shahied Abdel Hafez Primary School at El Monira.

B) **Method** :

All children studied were subjected to the following :

1- **Full history** :

Including the complaint, duration of illness, age of onset, course of the disease, frequency of the attacks, lines of treatment used during the attack, history of other allergic manifestations, dietary history, family incidence, residence and socio-economic status.

2) **Full clinical examination.**

3) **Routine investigations** :

a- Group of investigations to confirm clinical diagnosis of asthma e.g. skin test.

b- Group of investigations to exclude associated conditions which may interfere with growth and development like anaemia, parasitism and urinary tract infection. Such investigations included urine analysis, stool examination and full blood picture.

4) **Specific investigations** :

Evaluation of physical growth was done by obtaining the following measurements:

a- Height or length.

b- Weight.

c- Head circumference.

C) **Techniqual Procedures** :

1- **Length** :

The crown-heel measurement of infants and young children below the age of 5 years was taken in the recumbent position (recumbent length). This was done by the use of special horizontal measuring device, a plank with two trisquares for measuring the distance between the crown of the head and the soles of the feet. The baby was placed on his back on this plank and the head trisquare was fixed touching the crown of the head ; the baby's legs were straightened out and pressed flat against the plank, while the feet were flexed at right angles to the plank, the other trisquare was pressed to the soles. The distance between the two squares was the baby's length.

2) **Height** :

The crown-heel measurement in older children aged 5 years or more was obtained in the standing position (standing height). ready-made measuring boards were employed. It is an upright rack with centimeter graduation on it, and a small horizontal board in the form of a muff moving up and down freely.

The subject was placed so that the back of his

head, shoulder blades and buttocks touched the upright rack. The head was held, so that the tragus of the ear and the outer corner of the eye were on a horizontal level. The horizontal board fitted closely to the head but did not press on it. It goes without saying that the child was bare feet,

3) **Weight** :

Any system of scales is suitable for weighing infants, provided that the baby can be placed on them in a comfortable position. Infants and young children were weighed on a small scale in the recumbent position while older children were weighed on a larger scale in the standing position. All children were weighed in identical conditions e.g.naked and fasting. A prelimminary checking of the accuracy of the scales used was done.

4) **Head circumference** :

The head circumference was measured by a non stretchable tape from the farthest point on the forehead to the farthest point on the occiput passing just above the auricles of the ear.

The results obtained from control and asthmatic children were subjected to statistical analysis following the student (t) test of Fischer (Fischer & Yales,1946).

RESULTS

RESULTS

The results are shown in tables from 1 to 5 and figures from 1 to 3 .

Cases in which retardation of growth was found to be due to other lesions like mongolism, parasitism or nutritional deficiency were excluded before tabulating our results. This is to make certain that the growth retardation in the cases studied is purely due to the bronchial asthma.

Data obtained from asthmatic and control children were compared to the growth charts provided by Hamill, (1976). The percentage of these data to the expected 50th percentile of those growth charts were calculated.

Physical growth in all asthmatic & control children:-

The results are shown in tables 1,2 & 3 and fig.1.

(1) Physical growth in control children:-

The results are shown in table 1. This group comprised 50 children , 27 males and 23 females. Their ages ranged from 15 months to 12 years. One

half of this group (25 children) were below 6 years, while the other half ranged from 6 to 12 years old.

a. Height :

The percentages of the obtained data to the expected 50^{th} percentile of the same age and sex ranged from 90.33% to 105.56%; with the mean value of 98.14% ± 4.5%.

b. Weight:

The percentages of the obtained data to the expected 50^{th} percentile ranged from 73.44% to 116.28% with the mean value of 99.5% ± 10.74%.

c. Head circumference:-

Because of lack of growth curves for children beyond the age of 3 years, standardization of head circumference could be achieved only in 16 out of the 50 children studied. It was found that the percentages of the obtained data to the expected 50^{th} percentile ranged from 95.05% to 112.07% with the mean % of 100.4% ± 4.92%.

(2) Physical growth in asthmatic children :-

The results are shown in table 2. This group included 50 children suffering from bronchial asthma, 29 males and 21 females. Their ages ranged from

6 months to 12 years. One half of this group were below 6 years, while the other half aged from 6 to 12 years.

a. Height:-

The percentages of the obtained data to the expected 50^{th} percentile ranged from 79.65% to 106.19% with the mean value of 91.24% ± 5.31%.

b. Weight :-

The percentages of the obtained data to the expected 50^{th} percentile of the same age and sex ranged from 66.3% to 113.79% with the mean value of 85.24% ± 10.88%.

c. Head Circumference :-

Only 18 out of the 50 cases studied could be standardized. The percentages ranged from 83.16% to 101.52% with the mean value of 95.14% ± 5.49%.

Comparison between the percentages obtained from asthmatic children and those obtained from control children was shown in table 3 and fig.1. It can be seen that there was a significant decrease in the height ($P < 0.001$), weight ($P < 0.001$) and head circumference ($P < 0.01$) of asthmatic children in comparison to the control ones.

Physical growth in control and asthmatic children below 6 years and from 6 to 12 years old:-

The results are shown in tables 1,2,4 and fig.2. Because of lack of growth curves for head circumference of children beyond to age of 3 years, comparison between the 2 age groups could be achieved only for height and weight.

I- Physical growth of the age group of children below 6 years :-

(1) Control children :

The results are shown in tables 1 and 4. This group comprised 25 children,13 males and 12 females.

a. Height :-

The percentages to the expected 50^{th} percentile ranged from 90.33% to 105.56% with the mean value of 98.28% $\pm$ 4%.

b. Weight :-

The percentages to the expected 50^{th} percentile ranged from 80.58% to 116.28% with the mean value of 100.14% $\pm$ 10.32%.

(2) Asthmatic children:-

The results are shown in tables 2&4. This group included 25 children, 17 males and 8 females.

a. Height:-

The percentages ranged from 79.65% to 106.19% with the mean value of 90.23% ± 5.65%.

b. Weight:-

The percentages ranged from 67.79% to 104.4% with the mean value of 86.16% ± 9.91%.

II- Physical growth in the age group of 6 to 12 years old :-

(1) Control children:

The results are shown in tables 1 and 4. This group included 25 children 14 males and 11 females.

a. Height:

The percentages to the expected 50th percentile ranged from 93.07% to 105.51% with the mean value of 98% ± 3.4%.

b. Weight:

The percentages ranged from 73.44% to 115.64% with the mean percentage of 98.86% ± 11.33%.

(2) Asthmatic children:-

The results are shown in tables 2 and 4. This group comprised 25 children 12 males and 13 females.

a. Height:

The percentages to the expected 50^{th} percentile ranged from 82.18% to 101.89% with the mean % of 92.25% ± 4.87%.

b. Weight:

The percentages ranged from 66.3% to 113.79% with the mean value of 84.31% ± 11.79%.

Comparison between the percentages in asthmatic and control children in both age groups was shown in table 4 and fig.2. It can be seen that there was a significant decrease in the height and weight of the asthmatic children in comparison to the corresponding control children of the same age group ($P < 0.001$). There was no significant change in the height or weight between the 2 age groups neither in control ($P < 0.8$, $P < 0.7$) nor in asthmatic ($P < 0.2$, $P < 0.6$) respectively.

Physical growth in mild and severe cases of bronchial asthma :

The results are shown in tables 2,5 and fig.3. The cases of asthma were graded in relation to severity into 2 grades (I & II) on the basis of history, measures used to relieve the asthmatic attacks and the need of hospitalization of the patient.

(1) Physical growth in grade I bronchial asthma (mild cases):

They responded to simple measures with no history of hospitalization. This group included 27 children; 18 males and 9 females.

a. Height:

The percentages of the obtained data to the expected 50th percentile ranged from 88.19% to 106.19% with the mean value of 93.31% $\pm$ 4.72%.

b. Weight:

The percentages ranged from 68.9% to 113.79% with the mean value of 89.79% $\pm$ 10.19%.

c. Head circumference:-

Only 12 out of the 27 cases studied could be standardized. The percentages ranged from 95.05% to 100.44%with the mean value of 98.33% $\pm$ 1.72%.

(2) Physical growth in grade II bronchial asthma (sever cases):

Only relieved by corticosteroids and/or history of hospitalization. This group comprised 23 children, 11 males and 12 females.

a. Height:

The percentages to the expected 50^{th} percentile ranged from 79.65% to 100.36% with the mean value of 88.81% ± 5.01%.

b. Weight:

The percentages ranged from 66.3% to 101.78% with the mean value of 80.28% ± 9.1%.

c. Head circumference:-

Only 6 out of the 23 cases studied could be standardized. The percentages of the obtained data to the expected 50^{th} percentile ranged from 83.16% to 101.52% with the mean value of 90.27% ± 6.55%.

Copmarison between the percentage height, weight and head circumference of control children, grade I and grade II asthmatic children was shown in table 5 and fig.3. It can be seen that there were significant decreases in all measures of asthmatic children

of both grade I and grade II in comparison to the control children ($P < 0.001$). Also there was a significant decrease in height ($P < 0.005$), weight ($P < 0.005$) and head circumference ($P < 0.001$) of grade II in comparison to grade I asthmatic children.

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Table (1):- Critria of physical growth in control children.

No	Age	Sex	Height (Cm)			Weight (Kgm)			Head Circum. (Cm)		
			Actual	50th percentile	%	Actual	50th percentile	%	Actual	50th percentile	%
1	12 y	F	141	151.5	93.07	30.5	41.53	73.44	54.5		
2	18 M	M	82	82.4	99.51	12	11.47	104.62	50	48.4	103.31
3	12 y	M	142	149.7	94.86	46	39.78	115.64	55		
4	15 M	F	77	77.4	99.48	10.4	10.17	102.26	52	46.4	112.07
5	20 M	M	82	84.2	97.39	13	11.32	114.84	47.4	48.7	97.33
6	11.5y	F	141	148.2	95.14	37	39.23	94.31	54.5		
7	2.25y	F	80	88.4	90.5	15.5	13.4	115.67	47	48.4	97.11
8	9.5 y	F	133	135.2	98.37	27.7	30.45	90.97	53.9		
9	10.5y	M	139	140.3	99.07	36	33.3	108.11	54.2		
10	3 y	F	93	94.1	98.83	13	14.1	92.2	48.7	49.3	98.78
11	2 y	M	84	86.8	96.77	11	12.34	89.14	48.5	49.2	98.58
12	10 y	M	136.8	137.5	99.49	33	31.44	104.96	55.1		
13	2 y	M	82	86.8	94.47	11	12.34	89.14	52.5	49.2	106.71
14	7 y	F	115	120.6	95.36	21	21.84	96.15	53.3		
15	2.5 y	F	95	90	105.56	10.5	13.03	80.58	47	48.8	97.11
16	2.5 y	M	87	90.4	96.24	15	13.52	110.95	48.2	49.9	96.59
17	7 y	F	116	120.6	96.19	20	21.84	91.57	52.4		
18	9 y	M	125	132.2	94.55	26	28.13	92.43	35		
19	3 y	M	90	94.9	94.84	17	14.62	116.28	49	50.5	97.03
20	8.5 y	M	128	129.5	98.77	28	26.66	105.03	53.4		

Table(1):- Cont.

No.	Age	Sex	Height (Cm)			Weight (kgm)			Head Circum. (Cm).		
			Actual	50th percentile	%	Actual	50th percentile	%	Actual	50th percentile	%
21	7 y	F	116	120.6	96.19	22	21.84	100.73	53.3		
22	2.5y	F	95	90	105.56	15	13.03	115.12	51	48.8	104.52
23	6 y	F	118	114.6	102.97	15.5	19.52	79.4	52.7		
24	3 y	M	93	94.9	98	12.5	14.52	86.09	48	50.5	95.05
25	10 y	M	134	137.5	96.75	33	31.44	104.96	54.1		
26	3.5y	M	102	99.1	102.93	16	15.68	102.04	50.9		
27	3 y	F	88	94.1	93.52	12	14.1	85.11	48	49.3	97.36
28	12 y	M	145	149.7	96.86	44	39.78	110.61	55.2		
29	4.5y	M	107.5	106.6	100.84	17	17.69	96.1	51.3		
30	8 y	M	134	127	105.51	28	35.3	79.32	53		
31	4 y	F	105	101.6	103.35	16	15.96	100.25	50.7		
32	3 y	F	85	94.1	90.33	13	14.1	92.2	51.5	49.3	104.46
33	5 y	M	110	109.9	100.1	20	18.67	107.12	50.9		
34	9 y	M	127	132.2	96.07	26.5	28.13	94.21	52.7		
35	6 y	M	118	116.1	101.64	23	20.69	111.16	51.8		
36	6 y	F	109	114.6	95.11	20	19.52	102.46	51.6		
37	7 y	M	124	121.7	101.89	25	22.85	109.41	52.1		
38	6.5y	M	121	119	101.68	23	21.74	105.8	52		

Table(1):- Cont.

No.	Age	Sex	Height (Cm)			Weight (Kgm.)			Head Circum.(Cm).		
			Actual	50th percentile	%	Actual	50th percentile	%	Actual	50th percentile	%
39	12 y	F	141	151.5	93.07	33.5	41.53	80.66	55.2		
40	4 y	F	101	101.6	99.41	16	15.96	100.25	49.9		
41	6 y	F	109	114.6	95.11	20	19.52	102.46	51.3		
42	2.5y	M	88.5	90.4	97.9	14.5	13.67	107.07	52.5	49.9	105.21
43	5 y	M	105.5	109.9	96	19.5	18.67	104.44	50.3		
44	8.5y	F	132	129.3	102.9	27	26.58	101.58	52.2		
45	3 y	F	90	94.1	95.64	13	13.98	93.32	47	49.3	95.33
46	9.5y	M	136	134.8	100.89	32.5	29.73	109.32	52.6		
47	4.5y	F	104	105	99.05	16.5	16.81	98.15	50.3		
48	10.5y	M	139.5	140.3	99.43	35.5	33.3	106.61	53.7		
49	4 y	M	106	102.9	103.01	15.5	16.69	92.87	49.8		
50	5.5y	F	109	111.6	97.67	20	18.26	107.76	51.1		
Mean Percentage Standard Deviation					98.14% ± 4.5			99.5% ±10.74			100.41% ±4.92

Table (2):- Criteria of physical growth in asthmatic children.

No.	Age	Sex	Grade	Height(Cm)			Weight (Kgm)			Head Cirumference(Cm)		
				Actual	50th percentile	%	Actual	50th percentile	%	Actual	50th percentile	%
1	12 y	F	II	143	151.5	94.39	29	41.53	69.83	54		
2	12 y	F	II	141	151.5	93.07	31	41.53	74.64	54		
3	10 y	F	II	128	138.3	92.55	25	32.85	76.8	52		
4	4.5y	F	I	111.5	105	106.19	15	16.81	88.23	51		
5	3 y	M	II	83	94.9	87.46	13	14.62	88.92	43	50.5	85.15
6	10.5y	F	II	131	141.5	92.58	28	34.72	80.65	53.5		
7	11 y	M	II	134	143.3	93.51	32	35.3	90.65	50		
8	9 y	M	II	121	132.2	91.53	24	28.13	85.32	52		
9	4.8y	M	I	102.5	108.1	94.82	19	18.2	104.4	47.5		
10	10 y	F	II	121	138.3	87.49	24	32.55	73.73	53		
11	8 y	M	I	112	127	88.19	20	25.3	79.05	52		
12	5 y	M	I	114	109.73	19	19	18.67	101.77	50		
13	7 y	F	II	112	120.6	93.73	19	21.84	87	51		
14	7 y	M	II	110	121.7	91.16	20	22.85	87.53	51		
15	12 y	M	I	140	149.7	93.52	40	39.78	100.55	53		
16	2 y	M	I	77	86.8	88.71	10.5	12.34	85.09	48	49.2	97.56
17	4 y	F	II	90	101.6	88.58	13	15.96	81.45	48		
18	18 M	M	I	74	82.4	89.81	9.5	11.47	82.82	48.5	48.5	100

Table (2):- Cont.

No.	Age	Sex	Grade	Height (Cm).			Weight (Kgm)			Head Circum (Cm).		
				Actual	50th percentile	%	Actual	50th percentile	%	Actual	50th percentile	%
19	2 y	M	I	79	86.8	91.86	10.5	12.34	85.09	47	49.2	95.53
20	4 y	F	II	92	101.6	90.55	14	15.96	87.72	50		
21	9 y	M	II	66	72.3	91.29	06.5	9.18	70.81	42	45.8	91.7
22	18 M	M	I	74	82.4	89.8	11.5	11.47	100.26	48	48.4	99.17
23	8 y	M	I	119	127	93.7	23.5	25.3	92.89	50		
24	2 y	M	I	79	86.8	91.01	11.5	16.69	68.9	49	49.2	99.59
25	11 y	F	II	120.5	144.8	82.87	24.5	36.95	66.3	52		
26	10 y	F	I	130	138.3	94	29	32.55	89.09	54		
27	9 M	M	I	66	72.3	91.29	8	9.18	87.15	46	45.8	100.44
28	1 y	F	I	66.5	74.3	89.5	9	9.53	94.44	44.5	45.6	97.59
29	6 M	F	I	59	65.9	90.07	7	7.21	97.09	42	42.4	98.59
30	3 y	M	I	85	94.9	89.57	13.5	14.62	92.34	48	50.5	95.05
31	2.5y	M	II	75	90.4	82.96	11	13.52	81.36	43.5	49.5	87.88
32	5 y	M	II	94.5	109.9	85.99	16	18.67	85.7	50.5		
33	2 y	F	II	71.5	86.8	82.37	8	11.8	67.79	40	48.1	83.16
34	7 y	M	I	124	121.7	101.89	26	22.85	113.79	51		
35	10 y	M	I	130	137.5	94.55	23.5	31.44	74.75	54		

Table (2):- Cont.

No.	Age	Sex	Grade	Height (Cm).			Weight (Kgm).			Head Circum. (Cm)		
				Actual	50th percentile	%	Actual	50th percentile	%	Acutal	50th percentile	%
36	8 y	F	I	119.5	126.4	94.54	23	24.84	92.59	50		
37	2 y	M	I	82	86.8	94.47	9.5	12.34	76.98	48	49.2	97.56
38	11 y	F	II	119	144.8	82.18	24.5	36.95	66.31	52.5		
39	10 y	F	I	129	139.3	93.28	26.5	32.55	81.41	53		
40	9 M	M	II	62	72.3	85.75	6.5	9.18	70.81	46.5	45.8	101.52
41	11 M	F	I	65	73.3	88.68	7.5	9.33	80.39	45	45.1	99.79
42	6 M	F	I	59.5	65.9	90.29	6.5	7.21	90.15	42	42.4	99.06
43	5 y	M	I	100.5	109.9	91.45	15	18.67	80.34	50		
44	11 y	M	I	133	143.3	92.81	31	35.3	87.82	53		
45	10 y	M	II	138	137.5	100.36	32	31.44	101;78	54		
46	10 y	M	I	139	137.5	101.09	30	31.44	95.42	55		
47	2.5 y	M	II	72	90.4	79.65	11.5	13.52	85.06	46	49.9	92.18
48	8 y	F	I	114.5	126.4	90.59	25	24.84	100.64	50		
49	10 y	F	II	120	138.3	86.77	26.5	32.55	81.41	52		
50	8 y	M	II	109	127	85.83	21.5	25.3	84.98	51		
Mean Percentage						91.24			85.41			95.14
Standard Deviation						± 5.31			± 10.88			±5.49

Table (3):-
Physical growth in all asthmatic children in comparison to the control children.

	No.	Height		Weight		Head Circumf.	
		Mean %	S.D.	Mean %	S.D.	Mean %	S.D.
Control	50	98.14	± 4.5	99.5	± 10.74	100.41	± 4.92
Asthmatics	50	91.24*	± 5.31	85.24*	± 10.88	95.14*	± 5.49
		(P< 0.001)		(P< 0.001)		(P< 0.01)	

* Significant decrease compared with the corresponding control value.

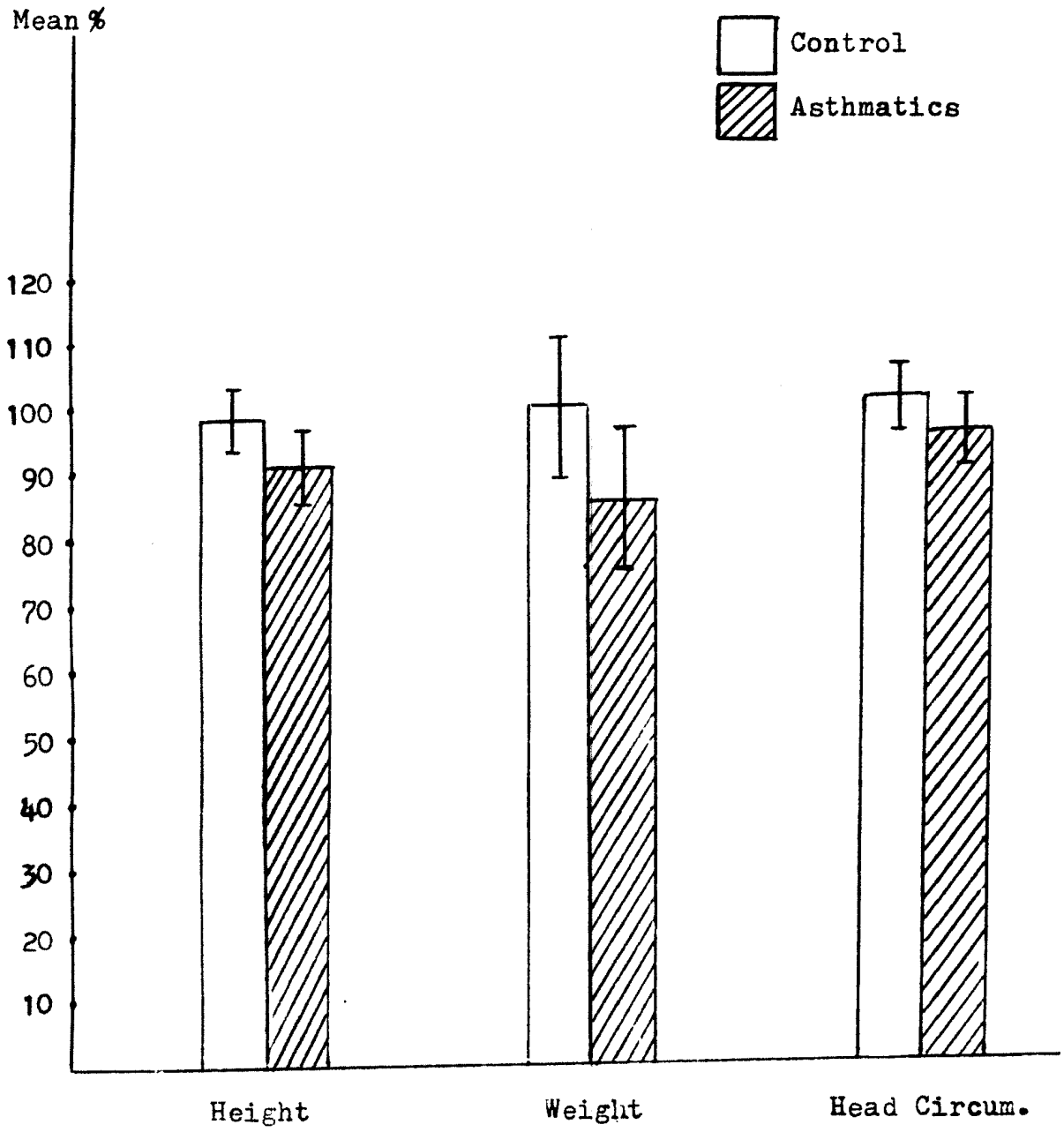

Fig. (1): Physical growth in all asthmatic and control children.

Table (4):- Physical growth in control and asthmatic children below 6 years in comparison to those aging 6-12 years.

		No.	Height		Weight	
			Mean %	S.D.	Mean %	S.D.
below 6 years	control	25	98.28	± 4	100.14	± 10.32
	Asthmatics	25	90.23*	±5.65	86.16*	± 9.19
6-12 years	Control	25	98	± 3.4	98.86	± 11.33
	Asthmatics	25	92.25*	± 4.87	84.31*	± 11.79

* Significant decrease compared with the corresponding control value ($P < 0.001$).

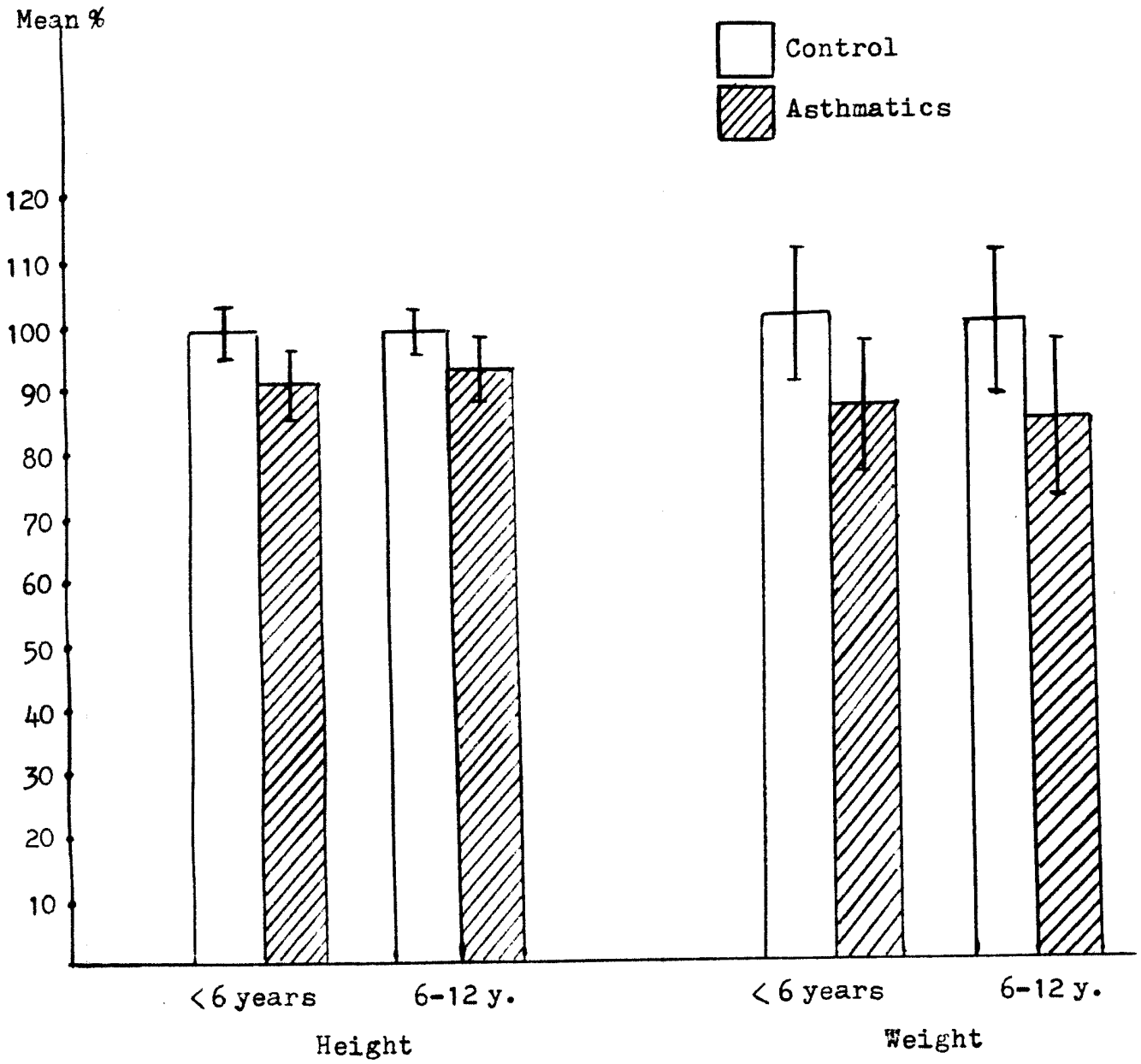

Fig. (2) ;Physical growth in control and asthmatic children aging below 6 years and 6 to 12 years.

Table (5):- Physical growth in mild and severe cases of bronchial asthma in comparison to control children.

	No.	Height		Weight		Head Circum	
		Mean %	S.D.	Mean %	S.D.	Mean %	S.D.
Control	50	98.14	± 4.5	99.5	± 10.74	100.41	± 4.92
Grade I	27	93.31*	±4.72	89.79*	± 10.19	98.33	± 1.72
Grade II	23	**88.81*	± 5.01	** 80.28*	± 9.1	** 90.27*	± 6.55

*(P < 0.001) *(P < 0.001) *(P < 0.001)

**(P < 0.005) **(P < 0.005) **(P < 0.001)

* Significant decrease compared with the corresponding control value.

** Significant decrease compared with mild cases of bronchial asthma.

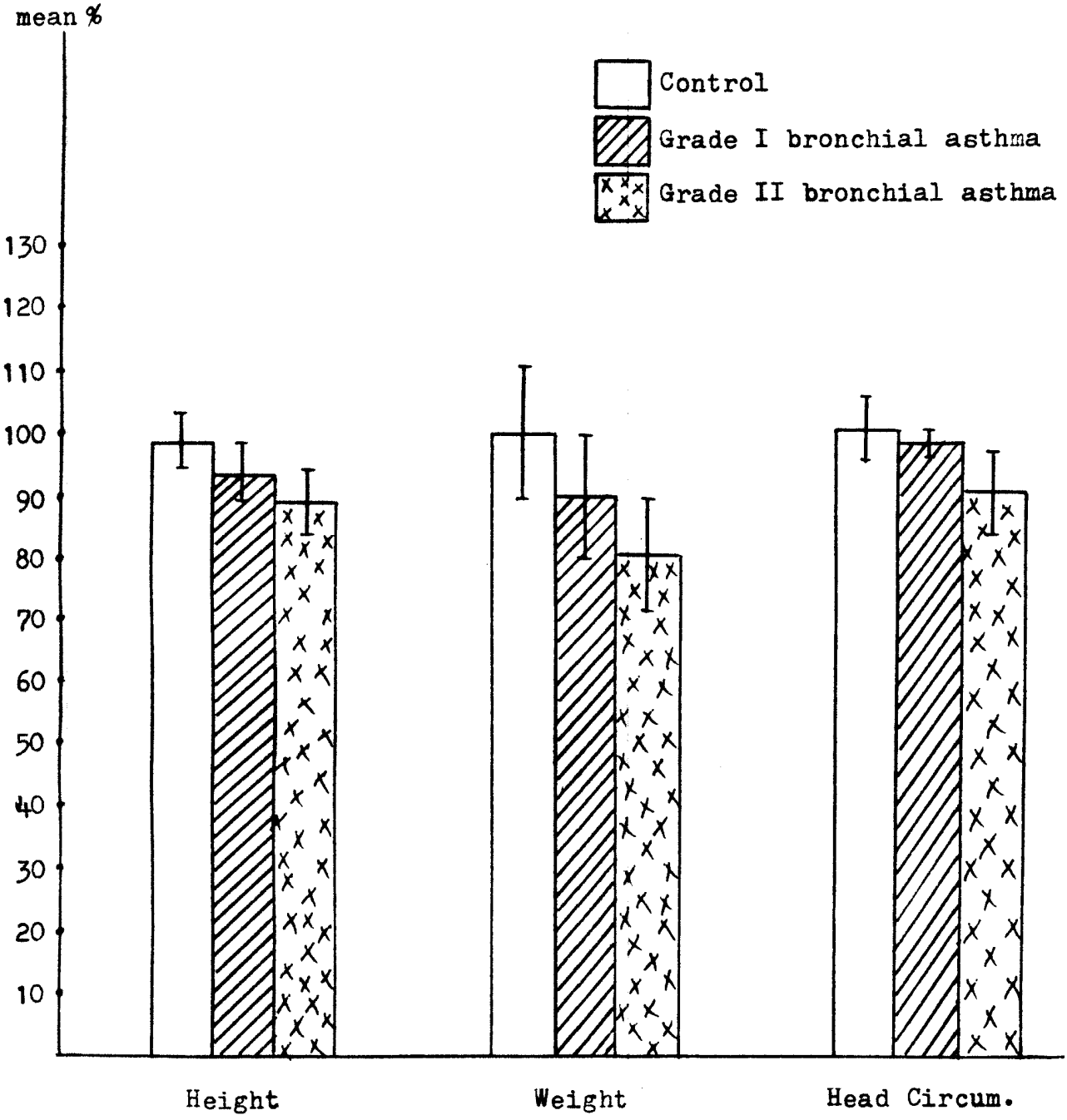

Fig. (3) : Physical growth in control children and mild and severe cases of bronchial asthma.

DISCUSSION

DISCUSSION

It has been mentioned before that bronchial asthma is a common disease of childhood (El-Hefny, 1966). It has also been shown that asthma is not considered as a major cause of death, but it is one of the leading causes of chronic illness in children (Inman & Adelstein, 1969).

Bronchial asthma in children is considered as one of the handicapping diseases of which retardation of physical growth is considered an important aspect (Von Metre et al., 1960; Falliers et al., 1961 and Spock, 1965).

Several reports concerning retardation of physical development in children suffering from bronchial asthma have been already discussed before.

The present study aims to investigate this possible presence of retardation of physical growth in asthmatic children by comparing the heights, weights and head circumferences of asthmatic children up to the age of 12 years with control children of the same age and sex.

The results of our work showed clearly that in asthmatic children retardation of physical growth occurs in height (91.24% ± 5.31% of the expected 50th percentile of the same age and sex), weight (85.24% ± 10.88%) and head circumference (95.14% ± 5.49%) in comparison to the control children (98.14% ± 4.5%, 99.5% ± 10.74% and 100.41% ± 4.92% respectively).

The mechanisms by which bronchial asthma affects physical growth are various, including respiratory insufficiency, dietary factors, chronic or recurrent infection, suppression of normal activity and the effect of certain drugs (Falliers et al., 1961).

The degree of retardation of physical growth in bronchial asthma also varied greatly according to many other factors including, the severity of the disease, duration of illness and age of the patients.

The severity of asthma is one of the most important factors that affect the rate and degree of growth retardation. In our study a growth retardation was found in height (88.81% ± 5.01), weight

(80.28% ± 9.1%) and head circumference (90.27% ± 6.55%) in severe cases compared to 93.31% ± 4.72%, 89.79% ± 10.19% and 98.33% ± 1.72% in mild cases respectively . This is in accordance with the results of Dawson et al., (1969), who showed that both heights and weights tended to be below the mean for age and sex, and that this trend was most evident in the clinically severely affected patient.

The age of the patient seems also to be one of the important factors that affect the rate and degree of growth retardation in children with bronchial asthma. Hypoxia due to respiratory insufficiency results in slowing of normal cellular multiplication. This was reported by Naeys (1967) when he found that some children with congenital heart disease had subnormal number of skeletal muscle cells. He did a quantitative study for 220 individuals dying with congestive heart failure: body size, organ size and cellular structure. He found that infants and children aged one month to 8 years had organ and cellular abnormalities resembling those usually found with chronic under-nutrition. On the other hand older individuals were found to have organ

and cellular abnormalities which were related to chronic hypoxia.

The role of insufficient food intake is difficult to assess because a minor deficit over a prolonged period of time can cause growth failure when this deficit occurs during a period of rapid growth (Krieger, 1970).

Intake may be insufficient because of psychological factors related to the presence of chronic or life threatening disease. A fearful and insecure mother, not given proper dietary instructions, who does not feed the infant unless it cries and demandsto be picked up. Such abnormal eating behavior may not be reversible in other patients (Krieger, 1970).

Histories and direct observation of patients indicated that development of growth retardation was related to frequent infections and episodes of anorexia and minor illnesses during which nitrogen retensions decreased.

Mitchell et al., (1975) studied the incidence of viral infections in children over the age of one year admitted to hospital with asthma and wheezy bronchitis. Viral infection was found in 17.2% of the patients investigated. The most common viruses cultured were rhinovirus and respiratory syncytial virus. Children studied during second or subsequent admission had higher incidence of viral infection than those admitted for the first time.

Falliers (1960) reported that presence of thyroid hypofunction, which may follow the combined use of iodides and steroids for asthma without clinical signs of thyroid abnormality. This conclusion was based on the reported observation that a suppression of thyroidal I^{131} uptake during iodotherapy was followed by a rebound avidity of the thyroid gland for I^{131} after discontinuance of iodides, an effect similar to that of known antithyroid compounds.

Friedman & Strang (1966) compared the effect of prolonged adminstration of corticosteroids and corticotrophin on growth in a group of children with asthma, and showed that the corticosteroids

inhibit growth and lead to considerable stunting, but when corticotrophin is given in doses sufficient to control the signs and symptoms of the disease, the growth rate increase.

Pharmacological doses of corticosteroids retard or interrupt the growth of children, indicating an adverse effect on the epiphyseal cartilage. Inhibition of growth is a rather widespread effect of the glucocorticoids. For example, they inhibit cell division or symthesis of DNA in thymocytes (Dougherty & White, 1944), fibroblasts (Pratt & Aronow, 1966), normal developing and regenerating liver (Howard, 1964; Loeb & Sternchein, 1973), gastric mucosa (Loeb & Sternschein, 1973), developing brain (Howard, 1964), developing lung (Carson et al., 1973) and epidermis (Fisher & Maibach,1971). Nevertheless, this effect is somewhat selective and corticosteroids do not characteristically produce the bone marrow depression or the enteritis that follow the exposure to non specific antimitotic agents . The mechanisms of these effects of steroids are not known. It has been reported that cortisone treatment rapidly decreases the activity of DNA polymerase in rat liver (Henderson and Loeb,1970).

To conlude, growth retardation secondary to bronchial asthma is multifactorial. It also affects the prognosis of the case and its future course. So, every effort should be done to prevent or ameliorate such undesirable secondary complication of bronchial asthma.

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SUMMARY AND RECOMMENDATIONS

SUMMARY AND RECOMMENDATIONS

The aim of the present study was to detect the possible presence of physical growth retardation in children suffering from bronchial asthma in comparison to healthy control children.

The work was carried out on 2 groups of children :

A) **Control group** :

Included 50 children of the apparently healthy non asthmatic ones ; 27 were males and 23 were females. Half of this group was below the age of 6 years, while the other half ranged from 6 to 12 years old.

B) **Experimental group** :

Included 50 children, 29 males and 21 females, all of them were well known to be asthmatics. This group was classified as follow :

1- According to the age :

-below 6 years : 25 children.

-6 to 12 years : 25 children.

2- According to the degree of asthma :

-mild cases : 27 children.

-severe cases : 23 children.

Evaluation of physical growth was done by obtaining the height, weight and head circumference. The obtained values were compared with the expected 50 th percentile of the same age and sex obtained from the international growth charts.

The percentages of the growth parameters to the expected 50 th percentiles were compared in asthmatic and control children.

The results showed that :

1) There were significant decreases in the height ($P < 0.001$), weight ($P < 0.001$) and head circumference ($P < 0.01$) of the asthmatic children in comparison to the control children.
2) The retardation in height and weight was significant between asthmatic and control children of the same age group in both age groups (below 6 years and 6 - 12 years), while there was no significant change between the different age groups neither in asthmatic nor in control children.
3) The physical growth in the severely affected asthmatics (height, weight and head circumference) was significantly retarded from that in the moderately affected children ($P < 0.005$, $P < 0.005$ and $P < 0.001$ respectively).

The pathogenesis of physical growth retardation in bronchial asthma could be related to many factors including respiratory insufficiency, dietary factors, chronic or recurrent infections, suppression of normal activity, psychological factors and the effect of certain drugs.

Recommendations :

It is recommended that more attention should be paid to the development of sound eating habits and to the provision of sufficient caloric intake. Respiratory infection

should be dealt with properly and preventive treatment should be performed as early as possible. It is also recommended that, during the growing period of life, corticotrophin should be used in preference to corticosteroids.

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ARABIC SUMMARY

ملخص الرسالة

كان الغرض من هذا البحث هو تبين امكانية حدوث تأخر فى النمو الجسمانى فى الاطفال المصابين بمرض الربو الشعبى عن الاطفال الطبيعيين .

وقد اجرى البحث على مجموعتين من الاطفال :-

١- المجموعة الضابطة :

وتتكون من ٥٠ طفلا (٢٧ من الذكور و٢٣ من الاناث) يتمتعون بصحة جيدة ظاهريا ومن غير المصابين بالربو الشعبى . ونصف هذه المجموعة كانت اعمارهم اقل من ٦ سنوات بينما النصف الاخر تراوحت اعمارهم من ٦ الى ١٢ سنة .

٢- المجموعة التجريبية :

وكانت تتكون من ٥٠ طفلا (٢٩ من الذكور و ٢١ من الاناث) من المصابين بالربو الشعبى . وقد قسمت هذه المجموعة وفقا لدرجة المرض الى ٢٧ حالة معتدلة و٢٣ حالة شديدة كما قسمت وفقا لسن المريض الى ٢٥ حالة اقل من ٦ سنوات و٢٥ حالة من ٦ الى ١٢ سنة .

وقد تم تقييم النمو الجسمانى بقياس الوزن والطول ومحيط الرأس ومقارنة تلك القياسات بالنسبة المئوية الخمسينية المتوقعة فى نفس السن والجنس والمستخرجة من منحنيات النمو الطبيعية .

وقد اظهرت نتائج البحث ما يلى :-

١- كان هناك نقص فى الطول والوزن ومحيط الرأس فى الاطفال المصابين بالربو عن الاطفال الطبيعيين . وهذا النقص له دلالة احصائية .

٢- كان هناك نقص ذو دلالة احصائية فى الاطفال المصابين عن الاطفال الطبيعيين فى نفس المجموعة السنيه وذلك فى كلتا المجموعتين السنيتين (اقل من ٦ سنوات ، من ٦ الى ١٢ سنة) بينما لم يكن هناك تغير يذكر بين الاطفال فى المجموعات السنيه المختلفه فى اى من الاطفال الطبيعيين او الاطفال المصابين بالربو الشعبى .

٣- فى الاطفال المصابين بالربو كان هناك نقص ذو دلالة احصائية بين الحالات الشديده والحالات المعتدله فى الطول والوزن ومحيط الرأس .

- * ***** -

النمو الجسمى لدى الاطفال المصابين بالربو الشعـــــــى

رسالة مقدمة مـــن

الطبيـــــب / وائـــل عرفـــات المنســـــى

توطئـة للحصـــول علـى درجة الماجستير

فـــــى

الطفولـــــــــــــــة

تحـــت اشـــراف

الاستـــاذ الدكتـــور
ضيائـــى محمـــد حســـــين
استـــاذ طـــب الاطفـــــال
الاكاديميـــة الطبيـــة العسكريـــــة

معهـــــد الدراسات العليا للطفولة
جامعـــــة عين شمس
١٩٨٦