

**"INSTRUCTIONAL AND EDUCATIONAL PROGRAM FOR TREATMENT
OF CHILDREN WITH BRONCHIAL ASTHMA AND THEIR FAMILIES"**

Thesis

Submitted For Fulfilment Of

Ph.D. Degree

In

Childhood Studies

By

283

Aboul Magd Ahmed Abdel Reheim Farghaly
M.B.B.CH. , M.Sc. (Childhood Studies)

Supervised By

Prof. Dr. Saadia M. Bahader
Prof. of Developmental Psychology
Dean of Institute of Post Graduate
Childhood Studies
Ain Shams University

Prof. Dr. Anissa EL-Hefny
Prof. of Pediatrics and Allergy
Faculty of Medicine
Cairo University

and

Prof. Dr. Olweya Mohammed Abdel Baky
As. Prof. of Medical Childhood Studies
Medical Department
Institute of Post Graduate
Childhood Studies
Ain Shams University

Ain Shams University
1994



**"INSTRUCTIONAL AND EDUCATIONAL PROGRAM FOR TREATMENT
OF CHILDREN WITH BRONCHIAL ASTHMA AND THEIR FAMILIES"**

Thesis

Submitted For Fulfilment Of

Ph.D. Degree

In

Childhood Studies

By

Aboul Magd Ahmed Abdel Reheim Farghaly
M.B.B.CH. , M.Sc. (Childhood Studies)

Supervised By

Prof. Dr. Saadia M. Bahader
Prof. of Developmental Psychology
Dean of Institute of Post Graduate
Childhood Studies
Ain Shams University

Prof. Dr. Anissa EL-Hefny
Prof. of Pediatrics and Allergy
Faculty of Medicine
Cairo University

and

Prof. Dr. Olweya Mohammed Abdel Baky
As. Prof. of Medical Childhood Studies
Medical Department
Institute of Post Graduate
Childhood Studies
Ain Shams University

Ain Shams University

1994

DISCUSSION AND JUDJMENT COMMITTEE

The vice-president for higher studies and research of Ain-Shams University has approved to form the following committee for the discussion of Mr. *Aboul. M. ogd Ahmed Alidd kha*

1. *Prof. Dr. Saadia M. A. Bahadar*.....
..... Chairman. *[Signature]*

2. *Prof. Dr. Anissa M. EL-Hefny*.....
..... Member. *[Signature]*

3. *Prof. Dr. Daisy M. H. Hussein*.....
..... Member. *[Signature]*

4. *Prof. Dr. Hanan Hafez Hathout*.....
..... Member. *[Signature]*

بِسْمِ اللّٰهِ الرَّحْمٰنِ الرَّحِیْمِ

" قَالُوا سُبْحٰنَكَ لَا عِلْمَ لَنَا اِلَّا مَا عَلَّمْتَنَا اِنَّكَ اَنْتَ الْعَلِیْمُ الْحَكِیْمُ "

صدق الله العظيم
(سورة البقرة آية ٣٢)

Acknowledgement

I would like to express my deepest appreciation and profound gratitude to **Professor Dr. Saadia Mohamed Bahader** , Professor of developmental Psychology , and Dean of Institute of Post Graduate Childhood Studies , Ain Shams University for her continuous encouragement , valuable advices , and sincere help that enabled me to complete this work in its present form .

It is my honour to express my sincere feelings of gratitude and highest appreciation to **Professor Dr. Anissa EL-Hefny** , Professor of Pediatrics and Allergy , Faculty of Medicine , Cairo University , for her continuous guidance , generous help , and precious advices that enabled me to finish this work in the present form .

My deepest thanks and sincere respect to **Professor Dr. Olweya Mohamed Abdel Baky** , Assistant Professor of Medical Childhood Studies. Institute of Post Graduate Childhood Studies , Ain Shams University , for her careful guidance , constructive advices , and keen interest to accomplish this work in its present form .

I would like to express my deepest appreciation and much thanks to **Professor Dr. Mohamed Hafez Hathout** Director of the Institute of Research for Tropical Medicine for his efforts and kind assistance during coduction of the study .

Grateful acknowledgement and deep thanks to **Dr. Mona Moustafa EL-Falaky** lecturer of Pediatrics , Faculty of Medicine , Cairo University , for her efforts and assistance during clinical sessions .

TO MY PARENTS , MY WIFE AND CHILDREN

CONTENTS

| | <u>Page</u> |
|--|-------------|
| List of tables | i |
| List of figures | vi |
| List of abbreviations | ix |
| Introduction and Aim of Work | 1 |
| Review of Literature | 3 |
| Definitions | 3 |
| Prevalence of Asthma | 5 |
| Genetics of Asthma | 16 |
| Prenatal and Perinatal Risk Factors | 18 |
| Development of Wheezing in Infancy and Toddler years | 19 |
| Development of Wheezing and Asthma in Childhood | 27 |
| Development of Asthma in Later Childhood | 28 |
| Risk Factors For Triggering Attacks | 56 |
| Risk Factors For Hospitalization | 75 |
| Risk Factors For Asthma Deaths | 79 |
| Pathophysiology of Bronchial Asthma | 84 |
| The Role of The Nervous System In Asthma | 92 |
| Programs in medicine | 95 |
| Subjects and Methods | 110 |
| Results and Analysis | 125 |
| Discussion | 213 |
| Recommendations | 231 |
| Summary | 233 |
| References | 236 |
| Appendix | 269 |
| Arabic Summary | |

LIST OF TABLES

| Table No. | Page |
|---|------|
| 1 - Association between age and sex at initial interviews ——— | 130 |
| 2 - Age of onset of first attack of wheezing according to sex — | 131 |
| 3 - Educational level of enrolled families ————— | 132 |
| 4 - Occupational level of enrolled families ————— | 133 |
| 5 - Residence of families ————— | 134 |
| 6 - Birth order of asthmatic child ————— | 134 |
| 7 - Period of breast feeding ————— | 136 |
| 8 - Seasonal variations of asthma episodes ————— | 136 |
| 9 - Diurnal variations of asthma episodes ————— | 138 |
| 10 - Presence of nocturnal enuresis ————— | 138 |
| 11 - Presence of other types of allergy in the child ————— | 140 |
| 12 - Presence of family history of asthma ————— | 140 |
| 13 - Presence of family history of allergy ————— | 142 |
| 14 - Restriction of certain types of food because of asthma ——— | 142 |
| 15 - Foods prevented from asthmatic child ————— | 144 |
| 16 - Child's sport practice ————— | 144 |
| 17 - Presence of smoking persons in the flat ————— | 146 |
| 18 - Relation of smoking persons to asthmatic child ————— | 146 |
| 19 - Whether they smoke inside the flat ————— | 148 |

| Table No. | Page |
|---|------|
| 20 - Presence of dogs ,cats ,domestic animals or birds in the house _____ | 148 |
| 21 - Assessment of asthma severity by medical score _____ | 150 |
| 22 - Assessment of asthma severity by PEFr measurements _____ | 150 |
| 23 - Knowledge of caretakers about triggers of asthma attacks _____ | 152 |
| 24 - Assessment of knowledge of caretakers about triggers of asthma attacks _____ | 153 |
| 25 - Practice of caretakers to prevent asthma attacks _____ | 154 |
| 26 - Assessment of practice of caretakers to prevent asthma attacks _____ | 154 |
| 27 - Knowledge of caretakers about early warning signs of attack _____ | 156 |
| 28 - Assessment of knowledge of caretakers about early warning signs of attacks _____ | 157 |
| 29 - Practice of caretakers at start of the attacks _____ | 158 |
| 30 - Assessment of practice of caretakers at start of attacks _____ | 159 |
| 31 - Knowledge of caretakers about symptoms during attacks _____ | 161 |
| 32 - Assessment of knowledge of caretakers about symptoms during attacks _____ | 162 |
| 33 - Practice of caretakers during asthma attacks _____ | 163 |
| 34 - Assessment of practice of caretakers during attacks _____ | 163 |
| 35 - Knowledge of caretakers about tiggers of asthma attacks , before and after follow up _____ | 164 |
| 36 - Assessment of knowledge of caretakers about triggers of attacks (comparison group) _____ | 167 |

| Table No. | Page |
|--|------|
| 37 - Assessment of knowledge of caretakers about triggers of attacks (intervention group) _____ | 168 |
| 38 - Practice of caretakers to prevent asthma attacks , before and after follow up _____ | 170 |
| 39 - Assessment of practice of caretakers to prevent attacks , (comparison group) _____ | 171 |
| 40 - Assessment of practice of caretakers to prevent attacks , (intervention group) _____ | 172 |
| 41 - Knowledge of caretakers about early warning signs of asthma attacks , before and after follow up _____ | 174 |
| 42 - Assessment of knowledge of caretakers about early warning signs of attacks (comparison group) _____ | 175 |
| 43 - Assessment of knowledge of caretakers about early warning signs of attacks (intervention group) _____ | 176 |
| 44 - Practice of caretakers at start of asthma attacks , before and after follow up _____ | 178 |
| 45 - Assessment of practice of caretakers at start of attacks (comparison group) _____ | 179 |
| 46 - Assessment of practice of caretakers at start of attacks (intervention group) _____ | 180 |
| 47 - Knowledge of caretakers about signs of asthma attacks , before and after follow up _____ | 182 |
| 48 - Assessment of knowledge of caretakers about signs of asthma during attacks (comparison group) _____ | 183 |
| 49 - Assessment of knowledge of caretakers about signs of asthma during attacks (intervention group) _____ | 184 |
| 50 - Practice of caretakers during asthma attacks , before and after follow up _____ | 186 |

| Table No. | Page |
|---|------|
| 51 - Assessment of practice of caretakers during attacks (comparison group) _____ | 186 |
| 52 - Assessment of practice of caretakers during attacks (intervention group) _____ | 187 |
| 53 - Feelings of caretakers about usefulness of drug treatment _____ | 188 |
| 54 - Restriction of child's activity because of asthma _____ | 190 |
| 55 - Prevention of child from going to school for one day or more because of asthma _____ | 191 |
| 56 - Child school performance _____ | 192 |
| 57 - Child sport practice _____ | 194 |
| 58 - Restriction of certain types of food because of asthma , before and after follow up _____ | 194 |
| 59 - Presence of smoking persons in the flat , before and after follow up _____ | 196 |
| 60 - Whether they smoke inside the flat , before and after follow up _____ | 197 |
| 61 - Use of child's room for other purposes (smoking , cooking , or as a store) _____ | 199 |
| 62 - Use of sprays in the flat as an insecticides _____ | 199 |
| 63 - Presence of plants inside the flat _____ | 200 |
| 64 - Presence of dogs , cats , domestic animals or birds in the house , before and after follow up _____ | 201 |
| 65 - Presence of asthma attacks last year _____ | 203 |
| 66 - Classification of degree of asthma according to medical score , before and after follow up _____ | 204 |
| 67 - Assessment of severity of asthma by medical score , (comparison group) _____ | 205 |

| Table No. | Page |
|--|------|
| 68 - Assessment of severity of asthma by medical score , (intervention group) ----- | 206 |
| 69 - Classification of degree of asthma according to PEFR measurements , before and after follow up ----- | 208 |
| 70 - Variables of the program , before and after follow up ----- | 210 |

LIST OF FIGURES

| Fig. No. | Page |
|---|-------------|
| 1 - Site of residence _____ | 135 |
| 2 - Birth order of asthmatic child _____ | 135 |
| 3 - Period of breast feeding _____ | 137 |
| 4 - Seasonal variations of asthma episodes _____ | 137 |
| 5 - Diurnal variations of asthma attacks _____ | 139 |
| 6 - Presence of nocturnal enuresis _____ | 139 |
| 7 - Presence of other types of allergy in the asthmatic child _____ | 141 |
| 8 - Family history of asthma _____ | 141 |
| 9 - Family history of allergy _____ | 143 |
| 10 - Food restriction because of asthma _____ | 143 |
| 11 - Foods prevented from the asthmatic child _____ | 145 |
| 12 - Sport practice _____ | 145 |
| 13 - Smoking persons in the flat _____ | 147 |
| 14 - Relation of smoking persons to asthmatic child _____ | 147 |
| 15 - Whether they smoke inside flat _____ | 149 |
| 16 - Presence of domestic animals or birds in the house _____ | 149 |
| 17 - Severity of asthma by medical score _____ | 151 |
| 18 - Severity of asthma by PEFr measurements _____ | 151 |
| 19 - Knowledge of triggers of asthma _____ | 155 |

| Fig.No | Page |
|---|------|
| 20 - Practice to prevent asthma attacks _____ | 155 |
| 21 - Knowledge of early signs _____ | 160 |
| 22 - Practice at start of asthma attacks _____ | 160 |
| 23 - Knowledge of signs during attacks _____ | 165 |
| 24 - Practice during asthma attacks _____ | 165 |
| 25 - Knowledge of triggers (comparison group) _____ | 169 |
| 26 - Knowledge of triggers (intervention group) _____ | 169 |
| 27 - Practice to prevent attacks (comparison group) _____ | 173 |
| 28 - Practice to prevent attacks (intervention group) _____ | 173 |
| 29 - Knowledge of early signs (comparison group) _____ | 177 |
| 30 - Knowledge of early signs (intervention group) _____ | 177 |
| 31 - Practice at start of attacks (comparison group) _____ | 181 |
| 32 - Practice at start of attacks (intervention group) _____ | 181 |
| 33 - Knowledge of signs of attacks (comparison group) _____ | 185 |
| 34 - Knowledge of signs of attacks (intervention group) _____ | 185 |
| 35 - Practice during attacks (comparison group) _____ | 189 |
| 36 - Practice during attacks (intervention group) _____ | 189 |
| 37 - Child school performance (comparison group) _____ | 193 |
| 38 - Child school performance (intervention group) _____ | 193 |
| 39 - Child sport practice (comparison group) _____ | 195 |
| 40 - Child sport practice (intervention group) _____ | 195 |

| Fig.No . | Page |
|---|------|
| 41 - Food restriction because of asthma (before and after PGM)- | 198 |
| 42 - Smoking inside flat (before and after PGM) _____ | 198 |
| 43 - Use of sprays in the flat (before and after PGM) _____ | 202 |
| 44 - Presence of domestic animals or birds in the house , before and after PGM _____ | 202 |
| 45 - Asthma severity by medical score (comparison group) _____ | 207 |
| 46 - Asthma severity by medical score (intervention group) _____ | 207 |

LIST OF ABBREVIATIONS

- ARI** : Acute Respiratory Infections
- BHR** : Bronchial Hyperresponsiveness
- EIA** : Exercise Induced Asthma
- ETA** : Environmental Tobacco Smoke
- FEV1** : Forced Expiratory Volume In 1 second
- ICD8** : International Classification of Diseases 8
- ICD9** : International Classification of Diseases 9
- ICU** : Intensive Care Unit
- IHCA** : Isocapnic Hyperventilation of Cold Air
- LTD4** : Leukotrienes D4
- OKY-048** : Thromboxane Synthetase Inhibitor
- PAF** : Platelet Activating Factor
- PEFR** : Peak Expiratory Flow Rate
- PGD2** : Prostaglandin 2
- PGM** : Program
- URTI** : Upper Respiratory Tract Infection

INTRODUCTION

INTRODUCTION

Acute episodes of bronchial asthma represent one of the most common respiratory emergencies observed in the practice of medicine .Frequently when patient is first observed for care , s/he appears intensely ill (Mc Fadden , 1989) .

Health-care professionals who care for children are being challenged with an increasing prevalence , increasing severity , and perhaps increasing mortality .This is despite an ever-increasing understanding of the mechanisms of asthma and an increasing pharmacologic armamentarium to combat this disease. Many of the problems besetting us in our battle against childhood asthma are related to delay in diagnosis , lack of patient compliance , and socio-economic factors (Stillwell , 1993) .

Educational programs for patients with asthma offer them the opportunity to increase their knowledge and help them manage the condition more effectively by participating as active partners with their physician in controlling the disease .For patients who are receiving good medical care ,structural education can maximize the benefits this care can provide ; for patients who are receiving poor or less-than-optimal medical care , the program can make them more knowledgeable health care consumers .

AIM OF THE WORK

The aim of the study is to assess knowledge and practice of families of asthmatic children toward asthma ,increase knowledge ,improve practice of asthmatic children and their families against asthma , reduce asthma severity ,better home environments of asthmatic children ,and increase their participation in normal childhood activities .

**REVIEW
OF
LITERATURE**

DEFINITION OF ASTHMA :

Asthma has been recognized since the time of Hippocrates (Thompson and Thompson , 1985) . Osler ,1892 , almost century ago pointed out asthma as it presents with distressing sense of want of breath and a feeling of great oppression in the chest .Soon the respiratory efforts become violent , and all of the accessory muscles are brought into play . In a few minutes the patient is in a paroxysm of the most intense dyspnea .

In 1959 the Ciba Guest Symposium clearly distinguished bronchial asthma from asthma cardiale as a disease characterized by acute attacks of dyspnea and / or cough associated partly or totally with reversible airway obstruction including episodic , seasonal , perennial , and chronic variable wheezing . Historically asthma was used as a synonym of dyspnea , shortness of breath or simply difficulties in breathing (Matthys , 1990) .

Asthma is a respiratory disease characterized by intermittent or chronic , usually reversible airway obstruction . With optimal therapy it does not cause permanent lung damage, nor should it interfere with normal childhood activities (Alexander et al ,1981) .

Macklem , 1989 , defined asthma as an abnormal condition in which the

airways narrow excessively in response to a variety of different triggering factors that have little or no effect on normal lung . According to this definition the process of developing an asthmatic attack can be divided into two components , the triggers and the response. Although more is known about the former than the latter and although a trigger is clearly a risk factor for the development of an asthmatic attack, it is less clear that triggers are risk factors for the development of asthmatic condition. Similarly , attacks may be prevented by avoidance of triggers , but little , is known about prevention of asthma .

Warner et al , 1989 , defined asthma as ' episodic wheeze and / or cough in a clinical setting where asthma is likely and other rarer conditions have been excluded'. Later on , Warner et al , 1992, stated that : the vague definition outlined in the first document has not been changed . there has been a strong feeling that the concept of eosinophil mediated airway inflammation should be incorporated into the definition. As we have no information on the pathology in the airway of all but the most severe childhood asthmatics it was not included. However , the need to introduce prophylactic treatment at a very early stages remains a firm recommendation.

The Expert Panel Report of National Asthma Education Program , 1991 , concluded that : a widely accepted definition of asthma remains elusive. The

clinician , physiologist , immunologist , and pathologist all have different perspectives of asthma , and these perspectives are difficult to merge into a comprehensive definition sufficiently specific to exclude other disease entities that may share one or more of the characteristics of asthma . In light of our current knowledge , the generally agreed -on working definition of asthma recognizes that : Asthma is a lung disease with the following characteristics : (1) airway obstruction that is reversible (but not completely so in some patients) either spontaneously or with treatment ; (2) airway inflammation ; and (3) increased airway responsiveness to a variety of stimuli.

PREVALENCE OF ASTHMA :

Reports of increased deaths from asthma have drawn attention to a problem many people thought did not exist . The introduction and widespread use of new and specific asthma medications over the past two decades have induced a sense of control over asthma . Further evidence for increased prevalence and hospitalizations for asthma has caused the sense of control to be replaced by a sense of crisis in asthma care .

" How common is asthma ? " and " Is there more asthma now than before ? " . Epidemiologically these questions are answered by prevalence

rates - the number of children with asthma per defined population over various times of study . Attempts to answer these questions are complicated by lack of uniform diagnostic criteria for asthma as well as differences in methodologies from study to study (Bloomberg and Strunk , 1992) .

In the second National Health and Nutrition Examination Survey , conducted by the National Center for Health Statistics from 1976 to 1980 , in the United States , asthma was present in 3.6% of the children 3 to 17 years of age when determined by the presence of physician-diagnosed asthma , but in 5.3% when determined by the presence of trouble with wheeze . Either criterion was present in 7.6% of the children , and this figure was used as the overall prevalence ..There was an increase in prevalence in the 6 - to 11- year - old group compared with the previous surveys (Gergen et al , 1988) .

Buist , 1989 concluded that there are several ways in which the number and proportion of individuals in the population who carry the diagnosis of asthma may increase . *First* , there may be a true increase in prevalence and incidence . This may be a result of an increase in initiating factors (e.g., specific air pollutants in the environment or occupational setting) , because the pool of genetically susceptible individuals has increased , because there is increased survival of infants and children with severe respiratory impairment , or because there is increased case finding . *Second* , the increase may be in part an artifact resulting from increased labeling of individuals who previously

would have been diagnosed as having another condition (diagnostic shift) or who would previously have been undiagnosed . In the United States , for children aged 6 to 11 years , the prevalence of asthma (defined as disease currently diagnosed by a physician and or frequent trouble with wheezing during past 12 months) increased from 4.8% (according to First National Health and Nutrition Examination Survey carried out between 1971 to 1974) to 7.6% (according to the second survey carried out between 1976 to 1980) . The data confirm that asthma occurs more commonly in boys than in girls and that the prevalence decreases during childhood .

Hill et al , 1989 , stated that :Asthma is the commonist chronic illness among school children and an important cause of absence from school and of reduced participation in sport and other activities . The prevalence of wheezing , ' asthma ' , and school absence as a result of wheezing in Nottingham in England was calculated from a questionnaire survey of parents of 4750 children in a random sample of primary school .A response was achieved for 3805 (80%)children of whom 438 (11.5%) had episodes of wheezing in the last year and 224 (5.9%) had been diagnosed as having asthma .

Braback and Kalvesten , 1988 , reported that in a Swedish survey of asthma among school children , a questionnaire was sent to the parents of 10527 children aged 7- 16 years . A group of 420 children with asthma was

identified . Morbidity due to asthma in childhood is reflected in absence from school , nocturnal symptoms and restriction of normal activity .

Nonspecific disorders , such as asthma and chronic bronchitis , are the most common and the most important chronic airways diseases. They are the major cause of chronic respiratory insufficiency in Africa as well as on other continents .Data collected in 10 African countries showed that asthma prevalence ranges between 2 and 5% , as shown by questionnaire surveys among representative samples of school children. Large regional differences can be observed : such as in Tunisia , where the national survey yielded a mean prevalence of 2.3% ; but the prevalence was 2.3% in the north , 1.9% in the center , and 3.1 in the south (Chaulet , 1989) .

Bahna , 1970 , showed that the prevalence of asthma among school children in Egypt was 1.03%..El-Hefny et al , 1991 , reported that in Egypt , asthma affects approximately 8.2% of children aged 3 - 14 years .

Brook , 1991 , found that the prevalence of bronchial asthma among adolescents living in central Israel is 9 per cent . The number of children who had a history of recurrent wheezing episodes was 15 per cent . The prevalence of asthma mentioned in the pediatric and respiratory literature varies between 0.8 and 13.5 per cent . This great difference is probably the result of varying definitions , as well as the long list of differential diagnosis of

wheezing in childhood . Many studies based their data of prevalence upon the number of hospitalizations of asthmatic children .

Rate Of Hospitalization For Asthma :

Unlike prevalence and morbidity , for which change over time has been difficult to measure , hospitalization rates have increased dramatically in the past two to three decades (Gergen and Weiss , 1990) .

The increase in hospitalization for children with asthma in the United States , 112 per 100,000 in 1970 to 279 per 100,000 in 1980 , contrasts sharply with the stable rate of hospitalization for all other diseases in children . Children less than 4 years of age has been disproportionately affected , as this age group accounted for 46% of childhood asthma admissions in 1979 and 54% in 1987 . Black children under the age of 4 years have a rate of admission fourfold higher than white children of the same age (National Center for Health Statistics , 1990) . All studies indicate a large increase in cost per admission , the cost of a hospitalization has increased by 30% from 1972 to 1983 . In a recent study , children under 18 years represented 35% of hospitalizations for asthma annually and 24% of the hospital expenditure (Weiss et al , 1992) .

The increase in hospitalization have not been fully explained . Possible factors that have been considered include changes in disease classification ,

increase prevalence of asthma , increase severity of individual cases , increased numbers of children readmitted rather than an increase in the total number of children admitted , and changes in criteria for admission (Halfon and Newacheck , 1986)..

The other major issue to be considered in the increase rate of hospitalization is criteria for admission .There appears to be a trend for patient to present to the emergency room later in their exacerbation . Factors that have been considered in this change include longer waits during exacerbations because of lack of access to regular asthma care , lack of health insurance , and unavailability of medication because of expense (Gergen and Mullally , 1988). Unwillingness of the staff to continue treatment in the emergency room , knowledge that hospital care is effective in producing rapid return to a normal routine for the family , and a general availability of beds could contribute to the tendency to admit once the patient is in the emergency room (Bloomberg et al , 1992).

In Egypt A Five- Governorate Health Facility Survey , for children under five , was conducted during January to March 1991 . The revision of the inpatient and outpatient records for children admitted to the inpatient departments or attended the outpatient clinics from the 1st of January to the 31 of December , 1990 , revealed that :

In Fawzy Moaz Children Hospital (in the center of Alexandria) 764 child with bronchial asthma out of 1276 child with Acute Respiratory Infectins (ARI) were admitted to the inpatient department , the ratio reaches 59.9% . If we add children admitted with the diagnosis of asthmatic bronchitis (25 child) the ratio rises up to 61.8% of all children with ARI .

In Sahel Selim District Hospital in Assuit Governorate in Upper Egypt , the ratio of children with bronchial asthma to children with ARI admitted was 2%. While the ratio of children with bronchial asthma to all children admitted was 1.02% .*In Bany Mohammediat Rural Hospital ,* the ratio of children with asthmatic bronchitis reaches 8.7% of cases with ARI admitted to the hospital . While the ratio of children with asthmatic bronchitis to all children admitted was 3.5% . *In Dalrout District Hospital ,* the ratio of children with bronchial asthma reaches 4.3% of all cases of children with ARI admitted , and 0.5% of all cases of children admitted .

In Shebin El Koum Fever Hospital situated in El Menoufia Governorate (part of Lower Egypt) , the ratio of children with asthmatic bronchitis to children with ARI admitted to hospital was 11.2%. While the ratio of children with asthmatic bronchitis to all children admitted was 6.9% .*In Ashmoun District Hospital ,* the ratio of children with bronchial asthma or asthmatic bronchitis to children with ARI admitted was 5.4% . While the ratio is 1.1% for

all children admitted . *In Berket El Sabe District Hospital* , the proportion of children with bronchial asthma or asthmatic bronchitis to children with ARI admitted was 27.4% . The proportion reaches to 6.5% for all children admitted to hospital . In the outpatient department of the same hospital , children with bronchial asthma or asthmatic bronchitis accounted for 2.4% of children with ARI , and 1.2% of all children attended the outpatient clinic .

In El Khazendar General Hospital (in the center of Cairo) , the proportion of children with bronchial asthma or asthmatic bronchitis to children with ARI admitted to hospital reaches upto 73.75% . *In Fifteenth of May General Hospital* (a new extension south to Cairo) , the ratio of children with bronchial asthma or asthmatic bronchitis to children with ARI admitted was 17.6%. While the ratio reaches to 3% of all children admitted to the hospital (Khallaf and Farghaly et al , 1992).

Recent therapeutic advances have made it possible to better control the symptoms of childhood asthma than has been possible in the past . It is paradoxical that despite these advances , the hospital admission rate for asthma and asthma mortality have reportedly increased in recent years .

The experience at the Childrens Hospital of Los Angeles has been similar , with a doubling of the hospital admission rate for asthma between 1973 and 1987 . The causes of the observed changes in hospitalization are not

known , but several factors have been implicated : (1) the nature of the disease may be changing with regard to severity or natural history , (2) improved diagnosis , (3) changes in vital statistics reporting , (4) untoward effects of treatment , (5) environmental factors , (6) increased prevalence of asthma , and (7) an increased tendency for patients to use emergency room facilities as the primary provider . It was noted that hospitalizations and asthma mortality appear to be increasing faster than prevalence , suggesting that increasing severity of asthma may be more important than increased incidence . The increased incidence of asthma occurring among boys and black children is striking . Reports concerning the association of asthma and socioeconomic status factors such as poverty index ratio , gross family income , and education of the head of the household have been conflicting (Richards , 1989) .

Respiratory tract infections has been shown to be an important trigger of acute asthma and was present in a large percentage of patients : otitis , sinusitis , and pneumonitis was found in 45% of study patients . Results of studies do suggest that successful treatment of the sinusitis and asthma management appear to benefit the clinical course of the patient's asthma . A significant number of patients with severe and / or disabling symptoms appear to have been undertreated with respect to the use of inhaled adrenergic agents , continuous therapy for chronic symptoms , and avoidance measures for allergens and irritants (Richards , 1989) .

Trends In Asthma Mortality :

Generations of health professionals have been taught that death from asthma essentially never occurs . It is true that death from asthma is a very rare event and that individual physician is unlikely to have a patient with asthma who dies of the condition . Nevertheless , it is time to acknowledge that patients do die of asthma , as well as , with asthma , and that we appear to be in a period in which the death rate from asthma is gradually but steadily increasing in North America and in many other Western countries . This increase is of particular concern because it comes at a time when mortality from most other causes is on the decline (Buist , 1989) .

In 1988 , 119 children under the age of 15 years died of asthma in the United States (Sly , 1991) . The number of deaths from asthma in this age group in the United States had reached a low of 54 in 1977 , increasing to a peak of 125 in 1985 . Deaths due to asthma in all age groups rose from 1674 in 1977 (0.8 per 100,000 population) to 4597 in 1988 (1.9 per 100,000) . As with the prevalence and hospitalisation data , black have shared disproportionately in the increases in deaths (Sly , 1991) . In several studies done since 1940s , the death rate in patients with severe asthma has ranged from 1% to 3% (Strunk et al , 1985) .

In Fawzy Moaz Children Hospital in Alexandria 2 children died out of 764 children under five years with bronchial asthma admitted to the inpatient department , with death rate of 0.26% during 1990 , these informations obtained by revising the inpatient records of the indicated year (Khallaf and Farghaly et al , 1992) .

Possible explanations for the increase in asthma mortality can be summarized as follows : (1) More frequent diagnosis of asthma due to real increase in prevalence , increased case finding , spectrum of asthma broadened to include chronic cough , increased survival of infants and children with respiratory treatment , diagnostic shifts from other categories of disease to asthma (2) Increasing severity and case fatality of asthma (3) Coding changes from ICD-8 to ICD-9 (4) Treatment is harmful (Buist , 1989) .

Although deaths from asthma are essentially preventable , and despite the resources devoted to the treatment of asthmatics , many countries appear to be experiencing an increase in reported asthma mortality . Inappropriate self treatment with bronchodilators is likely to delay the initiation of corticosteroid and oxygen therapy in acute severe attacks , and the major cause of preventable deaths in the New Zealand epidemic in the late 1970 was related to the delays in receipt of potentially life-saving care in emergencies . This commonly occurred in association with inadequate maintenance therapy and long term management . Increased awareness among doctors and their

patients of the potential dangers of poor acute and long-term care may have contributed to the subsequent decline in deaths (Jackson et al , 1988)

Genetics of Asthma :

The study of twins and of the familial incidence of the disease have contributed significantly to the understanding of the contribution made to asthma by heredity . These studies have shown that there is a strong genetic component in atopic diseases including asthma , as demonstrated by the fact that most studies have found a great concordance of asthma in monozygotic than in dizygotic twins reared apart or together . Evaluation of twins for presence of atopic disease also has revealed no differences for twins raised apart compared with those raised together (Hanson et al , 1990) .

The high degree of discordance in clinical allergy between monozygotic twins (up to 80% in some studies) strongly suggests that environmental factors play a pivotal role in the clinical expression of atopic illness . The patterns of inheritance of total serum IgE have been extensively studied . Family and twin studies have shown that total IgE production has a heritability in the range of 50% to 84% . The production of specific IgE against precise allergens appears to be influenced more by environmental than genetic factors (Wutrich et al , 1981) .

In a recent study of the familial occurrence of atopy , Cookson and Hopkin , 1988 , suggested that atopy is inherited as an autosomal dominant trait and that the clinical expression of the disease depends on environmental and developmental factors . There has been considerable debate on the possible independent inheritance of asthmatic trait from that of atopy .

Sibbald et al , 1980 , studied intrinsic and extrinsic (i.e. , allergy skin test positive and allergy skin test negative) subjects with asthma . They found no differences between these two groups in the proportion of those with at least one affected first-degree relative . They concluded from these findings that asthma and atopy were inherited independently .

There is some evidence suggesting that bronchial hyperresponsiveness may have an important genetic component , Hopp et al , 1987 , studied bronchial responsiveness in nonasthmatic parents of children without asthma and in nonasthmatic parents of children with asthma . They suggested that a familial component of bronchial hyperresponsiveness may exist that is transmitted from one generation to the next . A study by Young et al , 1991 , in 64 normal infants showed that bronchial responsiveness measured early in life was increased in infants with a family history of asthma , but IgE levels in infants were unrelated to their level of bronchial responsiveness , suggesting that the latter may be inherited independently from atopy . There was no relation between the level of parental responsiveness to histamine airway

challenge and that of the infants .

Although it is well established that asthma has a significant familial component , the mode of inheritance is not well understood . The relation between the genetics of asthma and that of allergy and bronchial hyperresponsiveness remains to be elucidated (Morgan and Martinez , 1992) .

Prenatal and Perinatal Risk Factors :

Other risk factors seem to have a strong effect on the prevalence of asthma . Children of low birth weight are significantly more likely to have asthma than those of higher birth weight , and this relation does not seem to be explained by differences in race or socioeconomic status among children of different birth weights (Schwartz et al , 1990) .

It has been shown that children of low birth weight have a higher prevalence of bronchial hyperresponsiveness than controls of normal birth weight . It is possible that intrauterine conditions that predispose to low birth weight and prematurity may affect lung development and enhance bronchial responsiveness (Chan et al , 1988) .

Children of younger mothers (< 20 years of age) are almost twice as likely to have asthma than those of older mothers , and this was not due to differences in birth weight , socioeconomic status , or race between younger

and older mothers (Schwartz et al , 1990) . Younger mothers tend to be poorer and may not seek prenatal care until late during pregnancy , thus increasing the risk of perinatal complications . They may provide a lower quality of care for their children than older mothers . The mechanisms by which these factors increase the risk of asthma are not known , but it has been suggested that stress during the neonatal period may possibly increase the risk for the development of allergy (Morgan and Martinez , 1992) .

Development of Wheezing in Infancy and Toddler Years : . . .

Both exogenous factors deriving from the child's life experience and endogenous (congenital) factors may increase the risk of wheezing in infancy

Exogenous Risk Factors :

Exposure To Viral Infection : because most wheezing lower respiratory tract illnesses in infancy are viral associated , social factors play a leading role in their development , and risk of exposure to viral contagion is a major determinant of illness . Thus , crowding , number of siblings , and daycare have been shown to be associated with increased risk for lower respiratory tract illness . Lower socioeconomic status , has been related of wheezing , perhaps because of poorer air quality and increased risk of exposure to viral agents (Morgan and Martinez 1992) .

Parental Smoking : Parental , particularly maternal , cigarette smoking has been clearly associated with an increased risk of wheezing , respiratory symptoms , lower respiratory tract illness , and hospitalization in exposed infants (Wright et al 1991). The effect of maternal smoking has been assumed to be due to passive inhalation of sidestream tobacco smoke by the infant . This result in airway inflammation and other alterations favoring both viral infection and the development of clinical wheezing illness or pneumonia .

Recently , preliminary results from several studies have suggested that this relationship may be due to alteration of the developing lung by maternal smoking , leading to greater risk for wheeze with infection (Hanrahan et al 1990) . Maternal smoking during pregnancy results in fetal stress secondary to both intrauterine hypoxia and nicotine exposure . Whereas the growth retardation associated with maternal smoking during pregnancy is well known , lung-specific effects may occur , including a reduction in lung elastin content (Collins et al 1985) .

Breastfeeding : Breastfeeding in early infancy help to reduce the risk for wheezing lower respiratory tract illness . In developing countries , breastfeeding has been shown to be protective against respiratory illnesses (Kovar et al 1984) . Studies of breastfeeding in economically advanced societies have had more equivocal results (Bauchner et al 1986) .

Wright et al 1989 , demonstrated that breastfeeding was most protective against wheezing lower respiratory tract illness early in life . The impact of breastfeeding was most apparent when the risk of exposure to infection was high .

Endogenous Risk Factors :

Although no clear genetic basis for wheezing lower respiratory tract illness has been described , several factors endogenous to the infant play a role in wheezing . First , young infants (<2months) are less likely to develop severe wheezing . Second , older infants and children are much less likely to develop wheezing or lower respiratory tract disease with viral infection than are infants . Gender seems to play a role , with male infants bearing a greater burden of severe lower respiratory tract illness . The reasons for these age and gender relationships are not known , but they may relate to differences in lung function between sexes and with growth (Tepper et al 1986) .

Premature infants have a greater risk for wheezing and rehospitalization (Greenough et al 1990) . Infants who have acquired early lung damage because of mechanical ventilation have a further increase in their risk for wheezing , and infants with bronchopulmonary dysplasia may develop life-threatening bronchiolitis (Groothuis et al 1988) .

Immunologic Factors : Infants whose parents have a history of atopy have a higher risk for wheezing in some studies (Liang et al 1982) . Zweiman et al 1971 have shown that infants hospitalized with bronchiolitis have increased skin test reactivity , an increased family history of allergy , and a greater rate of eczema . In contrast , several epidemiologic studies have suggested that no strong relationship exists between familial atopy and wheezing lower respiratory tract illness in infancy. Halonen et al 1990 , have shown that cord blood immunoglobulin E (IgE) levels are inversely related to the risk of wheezing . In another recent study , Wilson et al 1992 , concluded that fifty children with at least one hospital admission for acute lower airway obstruction in the first 2.5 years of life were assessed at 3 years of age to determine the relationship between atopy , bronchial responsiveness , and the pattern of their symptoms . The findings of the study suggest that in this hospital based group of children , acute wheeze associated with colds in the first three years of life is independent of the finding of atopy and that bronchial responsiveness in this age group may have a different pathogenesis from that in older subjects . Thus , the role of familial predisposition to allergy in infantile wheezing remains controversial and seems unlikely to be of major importance .

Airway Reactivity : Studies of both normal infants and those with a history of wheezing have shown that wheezing reduce airway conductance and forced expiratory flow after exposure to a variety of bronchial challenges . This

response can both be blocked and reversed by B-agonist aerosol administration (Prendiville et al 1987) .

Initial Lung Functions : The importance of baseline lung function as a risk factor for wheezing has been demonstrated in a study of infants from Tucson , Arizona . Lung function was measured early in life prior to any lower respiratory tract illness . Infants with initial lung function suggesting diminished airway conductance had a three to six times greater risk for wheezing in the first year of life . More importantly , this risk of wheezing persisted for the first three years of life (Martinez et al 1991) .

Respiratory Illness In Infancy And The Development of Asthma :

It has been suggested that early damage to the lung secondary to viral infection in infancy and the toddler years may lead to an irreversible decrease in lung function and an increase in the risk of chronic lung disease (Burrows et al 1977) . Studies of children between 7 and 10 years of age have found that a history of hospitalization for bronchiolitis was associated with an approximate of 50% risk for recurrent wheezing as compared with 20% of the control group (Mok and Simpson , 1982) .

More recently , Murry et al , 1992 , reported that as a part of a long term prospective study , 73 children who had been admitted to hospital with viral bronchiolitis as infants were reviewed 5.5 years later and compared to carefully matched control group . In the postbronchiolitis group , there was a

highly significant increase in respiratory symptoms including wheezing . Although atopy in the family was not significantly increased in the index group , personal atopy was more prevalent . Bronchiolitis had a significant predictor of wheezing after adjusting for potential confounding variables , including atopy . Bronchial responsiveness to histamine was significantly increased in the index group . The long term prospective study of bronchiolitis has shown that 75% experienced wheezing in the first two years after the illness and 59% were still having wheezing episodes 3-5 years later . What remains unclear is whether viral bronchiolitis per se causes increased airways lability or whether those children with a genetic predisposition to atopy are more likely to develop bronchiolitis and /or postbronchiolitis symptoms . The findings have been contradictory on the role of atopy , and have suggested that environmental factors such as family size , parental smoking , breast feeding , and neonatal respiratory illness may be contributing to either the initial illness , subsequent symptoms , or both . At follow up 42.5% of postbronchiolitis children reported wheezing episodes over the previous year compared with 15.1% of the controls . Coughing episodes were reported in 65.8% of the index children and 37% of the controls . Upper respiratory tract infections (URTI) were reported as precipitants of wheezing in all symptomatic children (index and control) . In addition , 24.7% of the index group and 9.6% of controls reported exercise induced wheezing ($p < 0.05$) . Allergen associated wheezing provoked by

animals , grasses or food were reported in 6.8% of the index and 2.7% of the controls . There was a highly significant difference in the number of index children who used bronchodilator therapy over the previous year compared to the control group . Significant differences were also seen in the numbers who were prescribed antibiotics for respiratory symptoms . More of the index children were on asthma prophylactic therapy (sodium cromoglycate or beclomethasone dipropionate) , 9.6% compared with 4.1% , but this difference failed to reach significance . Children in the index group who had smoking mothers had a relative risk of 4.1 for wheezing compared with 1.8 for those with non-smoking mothers . This suggest that maternal smoking may be influencing the rate of wheezing after bronchiolitis . The data showed that acute bronchiolitis in infancy have a highly significant effect on the incidence of wheezing , once maternal smoking , breast feeding , personal atopy , family size , and social class , had been taken into account . A further analysis showed that bronchiolitis was acting as an independent factor , strongly supporting the contention that acute bronchiolitis is generating a state of abnormal bronchial reactivity in the airways rather than acting as a marker for children who have an inherent asthma tendency .

On the other hand Morgan and Martinez , 1992 , concluded that a significant proportion of children wheeze before 3 years of age , but only a minority go on to develop asthma . This remission in wheezing illness may

occur in some children because of lung growth and development . In other children , the presence of allergy and other factors leads to the development of asthma independent of prior wheezing lower respiratory tract illness history .

Wilson et al 1992 , reported that the findings of their study were compatible with the proposal that the varying interaction of three factors – bronchial hyper-responsiveness , atopy , and an increased susceptibility to lower respiratory manifestation of viral infections – gives rise to the differing patterns of asthma seen during childhood . The changing pattern of wheezing during early childhood could be explained by the following hypothesis : The early development of lower airway obstruction could occur in those with a combination of poor airway function (a probable determinant of increased bronchial responsiveness) and a particular susceptibility to viral infections . With age the size of airways increases and viral infections become less frequent , so the tendency to wheeze in response to viral infections will diminish , unless bronchial responsiveness is perpetuated by airway inflammation associated with atopy . In addition , the results of this study suggest that although the number of acute wheezing episodes related to clinical viral infections in the first three years of life is independent of atopy , the tendency to wheeze between viral infections was associated with atopy . The finding of increased bronchial responsiveness in the non-atopic 3 years olds contrasts with the reverse situation reported in older subjects with asthma and lends

weight to the idea of a different pathogenesis for bronchial responsiveness in the two age groups .

Development of Wheezing and Asthma in Childhood :

The study of asthma risk factors is complicated by their multiplicity, and by the difficulty of defining the outcome variables . Asthma is a heterogenous condition that varies substantially in presentation , severity , and clinical course among individuals . The pattern and severity of an individual's asthma may wax and wane over his or her life span . To further cloud the picture , infants and toddlers develop a high rate of wheezing lower respiratory tract illness triggered by viral infection . The investigation of risk factors for asthma is confused by these early wheezing illnesses , which resemble clinical asthma , but may not bear any direct relationship to its ultimate development . Recent evidences , suggest that it is most rewarding to view infantile wheezing lower respiratory tract illness and asthma as related but separate conditions .

Although immune response , viral infection , passive smoking , and lung function are risk factors for both of these conditions , their relative importance differs between each condition (Martinez et al , 1988) .

The risk for wheezing lower respiratory tract illness in infancy and toddler years appears to be closely related to initial lung function and chance of exposure to viral infection . In contrast , asthma in older children is closely

associated with the development of allergy . Although many infants and toddler wheeze ,most do not go on to develop asthma (Morgan and Martinez , 1992) .

Wheeze-associated lower respiratory tract illness occurs in 20% of all children and accounts for more than 60% of all lower respiratory tract illness . Wheezing lower respiratory tract illness is of concern not only because of its acute morbidity but also because it has been suggested that children and adults with respiratory problems in early childhood may have suffered irreversible airway damage leading to long-term lung dysfunction and a higher risk for clinical respiratory disease , including asthma (Samet et al 1983) .

Development Of Asthma In Later Childhood :

The onset of asthma may be an end-product of the summative or synergic effects of various factors (Teirama , 1979) . There is an interaction between medical and psychosocial factors leading to varying levels of individual adaptation to asthma . The dynamic interactions of medical and psychosocial issues in asthma allows an explanation and an understanding of the outcome in individual patients . It also emphasizes the interrelationship and connections between an individual's past and present experiences and his or her personality style as well as the biologic , psychologic , and social factors of importance in the production and perpetuation of asthmatic symptoms (Yellowlees and

Kalucy , 1990) . Bronchial asthma exert a definite influence on the entire family and psychosocial factors are regarded as important in the most severely ill 5-10% of all asthmatic children (Connors , 1983) . The concept of asthma as an inflammatory disease of the airways has gained considerable support . The role of allergy as a determinant of persistent bronchial inflammation in asthma recently has been the object of extensive studies . New attention has been paid to the factors that determine and maintain the bronchial hyperresponsiveness that is characteristic of most subject with asthma (Morgan and Martinez , 1992) .

Asthma and Allergy : The fact that many subjects with asthma are not obviously atopic (do not have a wheal - and - flare reaction to allergy skin tests) led to the conclusion that the condition could be divided into "extrinsic" (apparently allergic) and " intrinsic " (apparently non allergic) disease . Many of these subjects with apparently non allergic asthma had demonstrable bronchial hyperresponsiveness . It was thus believed that atopy and bronchial hyperresponiveness could be independent risk factors for asthma . The relation between allergic sensitization and asthma appeared to be age dependent . It has been reported that 75- 85%of patients with asthma have positive immediate skin test reactions to common inhalant allergens (Nelson , 1985) .

El-Hefny et al ,1991 , in their study " Egyptian house dust versus foreign house dust as an important inhalant allergens " demonstrated that the mean

total serum IgE was significantly higher in asthmatics compared to controls .The most common inhalant allergen found to cause asthma in this study was house dust insect allergens (cockroach ,house fly ,and mosquito) 76.4% followed by house dust (foreign 52.8% and Egyptian 46.8%) and house dust mite 34.4% . Children with positive skin tests to house hold insects were further studied and the results revealed sensitivity to cockroach in 64%, house fly 54.8% and mosquito 47.2% of cases .

El-Hefny et al , 1992 , in another study "Egg and milk allergy in Egyptian asthmatic children - Evaluation of their nutritional pattern " reported that skin sensitivity to common environmental allergens was as follows : house dust (20.6%) , mixed moulds (7%) , and dust mite (15.8%) .The percentage and degree of skin test sensitivity to house dust , dust mite ,and mixed moulds are significantly higher in asthmatics compared to controls .The allergic reaction in the airways is significant for two reasons : (1) It can cause an immediate reaction , with bronchial obstruction , (2) It can precipitate a late bronchial obstructive reaction several hours after the initial exposure . The delayed bronchial response is associated with an increase in airway hyperresponsiveness to non immunologic stimuli and can persist for several weeks or more after a single allergen exposure . The basis for the late bronchial response and increased airway hyperresponsiveness is thought to be inflammation and secondary epithelial damage in the airways (Cockcroft,1987)

In infancy , allergens play a less important role than at other ages because it takes time for allergic sensitivity to develop . Although allergic reactions to food may occur in infants , foods are not common triggers of asthma . An elimination diet is not routinely recommended because it only rarely will reveal a previously unsuspected food as a cause of asthma (Expert Panel Report of National Asthma Education Program , 1991) .

Studies in children with asthma suggest that allergy influences the persistence and severity of the disease , the severity of childhood asthma correlates with the number of positive immediate skin tests (Martin et al ,1981) . Children with multiple positive skin tests are more likely to have daily rather than intermittent asthma , possibly because of the presence of a chronic allergic inflammatory process (Zimmerman et al 1988) .

In Egypt El-Hefny and Moustafa , 1987 , in their study " The role of Schistosomiasis and Ascariasis in extrinsic atopic asthma in Egyptian children " found that hypersensitivity to Ascaris or Schistosoma antigens , may play a role in the aetiology of allergic asthma . Parasites may potentiate a non-specific IgE response to common environmental antigens which might influence the development of unrelated hypersensitivity , to such allergens , at least in generally predisposed individuals . The prevalence of asthma and immediate hypersensitivity reactions to common environmental allergens in patients who have IgE-mediated anti-Ascaris and anti-Schistosoma skin responses suggest

that their mast cells participate actively in allergic response . Mast cell receptors are not necessarily surfacted by parasite specific IgE .

Asthma and Airway Inflammation : It is now widely believed that airway inflammatory processes are in some way responsible for asthma . The availability of animal models and the use of bronchoalveolar lavage in experimental studies in humans has allowed to better understanding of the nature of this inflammation . Histopathologic studies from asthmatic patients who have died during asthma attacks show that there is marked inflammation in the airways , with infiltration of inflammatory cells , particularly eosinophils , disruption of airway epithelium and mucus hypersecretion (Dunnill , 1960) . Similar pathologic changes have been found in bronchial biopsies from asthmatics who are relatively mild and suggest that airway inflammation may underlie bronchial hyperresponsiveness (Laitinen et al 1985) . By bronchoalveolar lavage , several cell types were found , especially after provocation ; these cells are eosinophils , neutrophils , mast cells , and lymphocytes . It seems that several different cells are involved in the pathogenesis of asthma , and that these cells produce a variety of mediators that interact in a complex way to produce a number of pathologic effects that contribute to bronchial hyperresponsiveness (Neijens , 1990) . Macrophages are present throughout the respiratory tract . They have the potency to produce and release a variety of mediators . Macrophages from asthmatics

release increased amounts of mediators , such as thromboxane , prostaglandins , and platelet-activating factor (PAF) (Neijens , 1990) .

Histologic studies suggest that eosinophil infiltration is a feature of asthmatic airways and differentiates asthma from other inflammatory conditions . Allergen inhalation results in marked increase in eosinophils in bronchoalveolar lavage fluid at the time of the late reaction , and there is a relationship between peripheral eosinophilia and bronchial hyperresponsiveness (Frigas and Gleich , 1986) . Eosinophils release a variety of mediators , including leukotriene C₄ and PAF , and basic proteins such as major basic protein , eosinophil cationic protein and eosinophil-derived neurotoxin , that are toxic to airway epithelium and other structures . Many different mediators have been implicated in asthma , such as histamine , prostaglandins , and leukotrienes that contract airway smooth muscle , increase microvascular leakage , increase mucus secretion , and attract other inflammatory cells . Interaction between inflammatory mediators might account for bronchial hyperresponsiveness (Neijens , 1990) . PAF , like antigen , stimulates accumulation of eosinophils in lung and in the skin of atopic subjects . Since eosinophils themselves are a rich source of PAF , they can attract further eosinophils and there is the potential for a continued inflammation . PAF is a potent inducer of airway microvascular leak , being the most potent agent so far described . This lead to airway edema and plasma extravasation which

may be involved in the creation of bronchial hyperresponsiveness (Persson , 1986) .

Bronchial Hyperresponsiveness (BHR): Although very little is known about the risk factors for the development of asthma , probably the most important is BHR (Macklem , 1989) . It is possible to define BHR as a fall in FEV1 of 20% or more provoked by a cumulative dose of histamine or methacholine of 8.0 micro mole or a concentration (noncumulative) of 16 mg/ml or less (Salome et al , 1987) . BHR is a condition in which the airways narrow excessively in response to a dose of histamine or methacholine that has a smaller or no effect on normal lungs . Approximately 35% of children with BHR were not previously diagnosed as having asthma , nor did they have respiratory symptoms . Almost half of the children previously diagnosed as having asthma did not have BHR . It seems clear , that it is possible to have BHR without asthma , as well as asthma without BHR (Peat et al , 1987) .

Thus , BHR is neither perfectly sensitive nor perfectly specific , although the sensitivity is probably greater than specificity (Macklem , 1989) . Peat et al , 1987 , have shown that 3 factors - atopy , respiratory illness early in life , and a history of asthma in either parent - were the most important risk factors for BHR . The odds ratio for either moderate or severe BHR doubled if atopy was coupled with one of the other 2 risk factors and was increased 6-fold if all

3 were present . Studies of twins fail to indicate that a genetic factor by itself is important . Thus , genetic-environmental interactions that influence gene expression presumably account for the hereditary effect (Macklem , 1989) . A high salt intake has been reported as risk factor for BHR (Burney , 1986) . Exposure to plicatic acid can induce BHR and asthma with repeated exposures and sometimes when the worker is removed from the work place BHR is permanent . In those in whom BHR is reversible , it reverses very slowly . Thus , " healing " of BHR , if it takes place at all , occurs very slowly . The data suggest that exposure to allergens can induce BHR . Further , allergen exposure or avoidance has a clear cut-effect on the severity of BHR (Macklem , 1989) .

The majority of asthmatic patients are found to have bronchial hyperreactivity (BHR) and this phenomenon is clearly a central feature of the pathophysiology of asthma . BHR is related to the occurrence and severity of asthmatic symptoms in general and to the bronchial reaction after various stimuli such as allergens , exercise , and cold air . A wealth of information has been accumulated about clinical aspects of BHR , but still relatively modest knowledge is present about factors that influence BHR and about basic regulating processes (Neijens , 1990) . Recent epidemiologic evidence suggest that allergic sensitization is a major determinant of bronchial responsiveness in childhood and that it is independent of the presence of

symptoms . Sears et al , 1991 , obtained information on respiratory history and measured both bronchial responsiveness and serum IgE levels in 562 11-year-old children . They confirmed the finding that no cases of asthma were present among subjects who had low levels of serum IgE for their age (< 32 IU / mL) . They found that there was a strong linear relationship between prevalence of BHR and serum IgE levels . This was also true for the 198 children who had no history of current or past asthma , wheezing symptoms , rhinitis , or eczema . BHR was not found in any asymptomatic child with serum IgE of <32 IU / mL . These results strongly suggest that , at least in children , BHR is closely linked to the serum IgE level , even in subjects with no clinical expression of allergy . Because both allergy and BHR may predispose for the development of asthma , measures that avoid or delay allergic sensitization may play an important role in preventing asthma in children . These studies show that although atopy may be a necessary cause for the development of asthma and BHR , it is certainly not a sufficient cause capable of acting on its own . Clearly , other genetic and environmental factors play an important role as determinants of asthma .

Psychosocial Factors : A variety of psychological factors have been described as playing a role in bronchial asthma . Clinical and experimental observations have suggested that aggression is involved in the development and precipitation of some cases of asthma (Straker and Tamerin , 1974) .

Clinically , it has been noted that aggressive conflicts which threaten

separation from a maternal figure are prominent in patient with asthma (Jessner et al , 1955) . Asthmatic children have been reported to repress aggression as a means of defending against fears of separation , and aggressive behavior is often increased during the psychotherapy of children with asthma (Sperling , 1968) . In a study of intractable asthmatic children on a child psychiatric in-patient unit , clinical improvement in asthmatic symptomatology correlated highly with a concurrent increase in aggressive behavior , suggesting an inverse relationship between symptomatology and the overt expression of aggression (Straker and Tamerin , 1974) . Mathe and Knapp , 1971 , in an experimental study design to provoke aggression , demonstrated that perennial asthmatic subjects manifested a relative inability to express aggression as compared to control subjects , while physiologically having increased bronchial resistance . The authors reported that asthmatics as compared to controls had significantly less manifest hostile emotion prior to and during the stress . Extensive interviews revealed that " asthmatics showed a trend toward more anxiety , depression , guilt , and disgust-shame ; hostility ratings were much higher in healthy controls " . In addition , they reported that asthmatics had significantly lower urinary epinephrine values during both stress and control periods . Hahn , 1966 , using the mental arithmetic design , concluded that 18 asthmatic children's response to criticism was " more timid , self-reproachful , and less manifestly angry " , as compared to the controls .

The asthmatic subjects physiological response resembled the initial stages of an asthmatic attack . The investigators of both these experimental studies suggested that the inhibition of aggressive impulses seems concomitant to and possibly etiologically significant to the physiological response of bronchial asthma .

Straker and Tamerin , 1974 , concluded that a study of 42 perennial childhood asthmatics in the natural setting of a summer camp revealed a statistically significant relationship between the severity of asthmatic symptomatology and aggressive behavioral expression . The treated , or symptomatic group whose bronchial function was significantly more impaired than the untreated , or asymptomatic group were shown to be less expressive of manifest aggression . These findings support other experimental studies which suggest the inhibition of aggressive impulses as playing a role in the etiology of bronchial asthma . Purcell et al , 1961 , reported that asthmatic children who underwent a rapid remission after admission to hospital had "neurotic" symptoms such as anxiety , depression , and headache more often than those who continued to be dependent on corticoids even after admission

Teirama , 1978 , concluded that in his study with adult asthmatics , a large sub-group of corticoid-dependent patients showed strong psychological defences which in part may prevent or delay the onset of neurotic symptoms . Although the group lacked 'phobias' as a diagnostic label , the patients showed

various psychic disturbances , particularly obsessions depression and anxiety . On the other hand, neurotic and psychosomatic symptoms were reported less often by corticoid patients than by the asthmatics receiving heavy medication but no regular corticoid treatment .Psychic and psychosocial factors probably effect both the long-term trend and severity of asthma (Teiramaa , 1978 , a)

Van Der Valk , 1960 , found in his study that the asthmatics had less hobbies , memberships of societies and social contacts in general than did healthy control subjects or coronary patients . Introversion was a common trait to asthmatics showing no improvement in their condition (Teiramaa , 1978 , b) and to patients with a negative family history of atopic disorders (Teiramaa , 1979 , a) or depending on corticoids . In case of allergic asthma , a psychic stress or infection may precipitate asthma by upsetting the balance between the person and his allergen-containing environment . Long et al , 1958 , reported that home dust caused dyspnetic attacks in asthmatic children , but no such attacks were experienced in hospital even though dust collected from the children's homes was sprayed into the hospital room . Teiramaa , 1979 , b , directed attention to the probability of certain combinations of factors being prevalent in a potential or manifest instance of asthma , he concluded that one purpose of his study was to determine those psychic and psychosocial factors which may operate against or towards the inception of asthma . He reported that the asthmatics had a small 'number of hobbies when of school age ' more

frequently than the case among the controls . He concluded that his results indicate that psychic and psychosocial factors could be important in " potential asthma " . A high degree of extraversion and behavioural activities could help the subject to resist the onset , whereas introversive tendencies and an inability to feel or express psychic difficulties , depression in particular , may have the opposite effect . Teiramaa , 1981 , reported that particular psychosocial stress factors may increase the person's vulnerability as regards both the onset of asthma and its acute-subacute-insidious status in the early phase . The diagnosis of phobias , depressive illness or symptoms , anxiety neurosis or paranoid characteristics were about two and half times more frequent among the patients with certain disappointments before asthma than among the remaining asthmatics . The author concluded that his findings suggest that the early evaluation of an asthmatic's psychic characteristics and his psychosocial environment is obviously important at the outset of the disease , even in cases in which asthma is only suspected . This could in many cases help in understanding of why a particular person has contracted asthma under the given circumstances , and could give clues for possible psycho-therapeutic intervention , in addition to the usual allergologic and pneumological methods (Teiramaa , 1981) .

Psychosomatic research in the field of the asthmatic child and his environment has been underway for many years . The results so far , indicate

a complicated picture involving anxiety , rejection , and difficulty in communication (Aronsson and Koivunen , 1985) . A study made by Miller and Baruch , 1948 , indicate that asthmatic children had been exposed early to rejection from parents . Rees , 1963 , concluded that rejection with consequent feelings and compensating overprotection is often found . The parent's attitude is often perfectionist and rejecting . French and Alexander , 1941 , examined 27 asthmatics who underwent psychoanalytic treatment . They found the central problem to be anxiety at being separated from their mother . The asthma attack was a suppressed cry for a ' lost ' mother . In clinical work with asthmatic children , Wikran et al , 1978 , stated three characteristics of the asthmatic child's family situation : 1- Constant emotional repression ; 2- Difficulty in communicatin ; 3- Rewarding of their symptoms . Difficulty in communication will particularly lead to the avoidance of open discussions around a conflict . Liebman et al , 1974 , stated that severe and recurrent asthma , in spite of adequate medical treatment , is due above all to unsolved conflicts . Pinkerton , 1967 , examined this type of case and found that only 10% of the families had an adequate parental attitude with intellectual understanding and emotional insight . Around 50% of the children had overprotective parents , 20% had rejecting parents and 20% had parents who were ambivalent between overprotection and a rejecting attitude . The number of families studied was 25 . Garner and Wenar , 1959 , found in a retrospective

study that mothers of asthmatic children found no pleasure in bringing up their children but were ambitious and controlling . Their children felt persecuted . It is important to note that according to mothers , their ambitions and controlling personalities preceded the birth of their children and was not the result of motherhood . According to Garner and Wenar , 1959 , asthmatic children and their mothers do not function in harmony with each other . The mother cannot meet the child's needs in an adequate way . The interaction between mother and child is both intimate and frustrating . It is characterized by competition , especially on the mother's part . The authors believe that the child is still a part of the ' self ' of the mother and has not yet learned to differentiate himself from the mother .

Aronsson and Koivunen , 1985 , concluded that the parents of asthmatic children have been described in different studies as being rejecting , as having more guilt feelings with consequent overprotective attitudes , and as being more perfectionist , than parents of non asthmatic children . The authors conducted a study its purpose was to investigate the degree to which these attitudes exist in parents of asthmatic children , compared to a matched control group of parents of non-asthmatic children . The authors concluded that the results of this study indicates that the parents of asthmatic children are more aggressive and less exhibitionist (which can be an expression of compulsive character neurosis) than parents of non-asthmatic children . Whether the

personality characteristics in the asthmatic group have developed in the parent's interaction with their child or were pre-existing , it is hard to say . The authors reported that the psychoanalytic interpretation of the results indicates that the problem and conflict is often the relationship between the mother and unwanted child . The mother experiences anger over the unwanted child she has borne , maybe she even wishes the child dead . The aggression that she initially directs towards the child will soon lead to guilt feelings . If the child after some time gets asthma , the mother's guilt feeling may be fortified . She may take it as her fault that the child has developed asthma . This may lead to the mother becoming overprotective and experiencing a strong need to nurse and control the child . The mother easily develops a dominating role in the family , ' it is she who has the big responsibility for the child and who is responsible for the child's illness ' . All this is done by the mother to compensate (unconsciously) for the aggression initially directed toward the child . This development lead to the mother making the child more dependent on her than is healthy child . The other personality difference between the two groups appearing in the results is that the asthmatic group was less exhibitionist than the control group . The hysterical character is described as exhibitionist , theatrical , dramatic , a person who seeks the limelight and who finds it easy to get in contact with people . The opposite pole on the continuum of character neurosis is the compulsive neurotic , who is rigid , overcontrolling

and not very emotional . The whole personality is characterized by diligence , order and conformity . Persons with a compulsive character not only control themselves , but also control and dominate other people around them . The emotional and exhibitionist traits which mark the hysterical character are completely missing in the compulsive neurotic character . The asthmatic group in this study had a more compulsive neurotic character than the control group (Aronsson and Koivunen , 1985) .

Gustafsson et al , 1985 , reported that an unstable respiratory tract which readily reacts to many stimuli , including psychogenic factors , is probably the most important feature of bronchial asthma . The precipitating factors have an additive effect and the total load of stimuli is important . The relative significance of psychological factors varies among the patients and even in the same individual under different circumstances (Lask , 1979) .

Previous studies have demonstrated disturbed family relations in the families of children with severe asthma (Liebman et al , 1976) and family therapy has been shown to decrease asthma symptoms in these children (Lask and Matthew , 1979) . Gustafsson et al , 1985 , conducted a controlled family therapy study to children with severe bronchial asthma to evaluate the effects of family therapy upon ten clinical parameters focusing on the functional impact of the illness . They concluded that family therapy may represent a valuable therapeutic tool in the management of severe asthma . Their

hypothesis was that the parents of children with severe asthma would be overinvolved (enmeshed) and show little flexibility (rigid) and that the asthmatic child would have the role of detouring conflict in the family .

Whenever conflict in the family (e.g. between spouses) reached a certain threshold limit , the child would unconsciously develop symptoms distracting attention from the conflict . In this way the child could change the source of tension from the parents to himself or herself . The various pedagogical methods such as breathing exercises , practice in inhalation techniques , and teaching about allergic factors , would have little effect on a child under such psychological stress . If the stress could be reduced by family therapy , the child could start to learn to manage his or her illness . Psychological strain can also influence the balance of autonomic nervous system and other mediating systems , and thus have a direct effect on asthma symptoms . The authors reported that the therapists noted very few open conflicts in the families and usually one of the parents (most often the mother) had organized her daily life in order to help sick child . Because of this the parents spent less time together and conflicts were unusual , i.e. the way the families were organized around the disease resulted in the sick child detouring conflicts continuously . There was also a fear among the parents (and siblings) that strong emotions like screaming or crying could provoke attacks . This restrained the parent's executive functions and often made them too permissive . Siblings often had to

make much responsibility and parents were unable to give them optimal attention . In the family therapy a primary goal is to improve family function and competence. The authors suggest that the more competent and secure the parents and the children feel in handling the asthma symptoms at home , the less anxiety will be added to other precipitating factors in an ongoing attack . As a result , the risk for severe attacks demanding hospitalization will be less . The children in this study had been ill for a long time and most of the families were of the opinion that this form of support would have been even more effective if given earlier . They concluded that family therapy seems to improve severe bronchial asthma in children and should be considered in the treatment of these cases .

Yellowlees and Kalucy , 1990 , reported that recent research suggest that anxiety disorders are more common in asthmatic patients than in the population as a whole . There are a variety of biologic , psychologic , and social factors that suggest that the disorder of asthma may in itself be anxiogenic and that simply having asthma may give patients an increased vulnerability toward the development of anxiety disorders . There are a variety of indicators that suggest that biologic factors associated with psychiatric disorders may be of significance in asthma . Miller , 1987 , has postulated a physiologic link between depression and death from asthma via cholinergic

pathways . Yellowlees and Kalucy , 1990 , reported that tricyclic antidepressants not only improve mood disorders of asthmatic patients but in certain cases have also markedly improved their functional asthmatic status . Goldfarb and Venutolo , 1963 , used imipramine in an uncontrolled trial in patients with chronic allergic asthma , they concluded that imipramine was a useful and safe drug in the treatment of chronically ill depressed allergic patients . Sugihara et al , 1965 , concluded that amitriptyline was helpful in the treatment of patients either in status asthmaticus or who suffered recurrent attacks of asthma . Meares et al , 1971 , noted that amitriptyline could ameliorate symptoms of asthma . Steen , 1976 , concluded that in general , tricyclic antidepressants are safe , with little or no effect on respiratory center , as long as prescribed appropriately . Yellowlees and Kalucy , 1990 , demonstrated that even a patient with a disastrous developmental background , major medical problem , and strong biological propensity to develop both asthma and psychiatric disorders may still adapt well if he has a good insight into his problems and complies appropriately with an effective therapeutic regimen organized by a physician with whom he has a good and trusting relationship . Conversely a patient with a reasonable upbringing , a relatively mild degree of asthma , and a very dependent personality with few family and social supports , being treated by a physician who does not appreciate the patient's psychological needs , and who consequently gets angry and

frustrated , may spend inordinate amounts of time in the hospital and be placed on a regimen of excessive dosages of corticosteroids leading to a markedly increased level of disability .

Childhood Asthma and Gender : Age and sex are risk factors for asthma , but the role they play is small . Asthma is more prevalent in children than in adults and in boys than in girls (Macklem , 1989) . Most surveys of asthma in children have found that the prevalence of asthma is higher in boys than in girls , with a ratio of approximately 2 : 1 . The causes of this difference in prevalence are not well understood . Most epidemiologic studies of bronchial responsiveness in children have found no significant differences in prevalence of bronchial hyperresponsiveness between genders (Sears et al , 1991) . There is a consensus , on the fact that total serum IgE levels are higher in boys than in girls , and the discrepancies in gender specificity of allergy skin tests between studies may be due to the number and type of allergen extracts chosen .

Childhood Asthma , Poverty and Race : In the United States , results of a large survey on health and nutrition showed that asthma is 2.5 times more prevalent among black children than among white children (Schwartz et al , 1990) . In the same study , race-specific prevalence of asthma was found to be inversely related to family income . What causes the increased risk of

asthma among black and poor American children is not well understood .

Morgan and Martinez , 1992 , suggest that the factors that determine the increased risk of asthma among black children may be different from those that are responsible for the increase in asthma among poor children . It is possible that poor children may be exposed to and become sensitized to aeroallergens that are more likely to enhance bronchial responsiveness and asthma (e.g. , house dust mites and molds) , whereas children of higher socioeconomic level may be sensitized to allergens that are less likely to enhance bronchial hyperresponsiveness (e.g. , pollens) . There are important geographic differences , the countries with the greatest prevalence are Australia and New Zealand , with rates of 11 - 17% . Among Eskimos and Gambia , the prevalence is claimed to be zero . How these variations relate to genetic and environmental factors , or to differences in definition of asthma and thus to diagnostic criteria is unknown . There is evidence that a move from a rural to an urban community increases risk of asthma . Xhosa children living in their homelands have a prevalence of asthma of 0.1% , whereas in Capetown the prevalence in these children is 3.2% . Similarly , in Tokelau islanders the prevalence of asthma in the island is 1.3% , whereas among those who moved to New Zealand it is 6.9% (Macklem , 1989) . Sprik et al , 1990 , suggested that early exposure to house dust mite antigens may increase sensitization to these allergens . Schwartz et al , 1990 , reported that asthma is 1.5 times

more prevalent in the inner city than outside of it . The authors concluded that aeroallergens in New York inner-city apartments have shown that antigens such as *Alternaria* , cockroach dust , and mouse urine are common in these settings . House dampness and molds are common in poor areas and have been associated with increased obstructive respiratory morbidity in children (Brunekreef et al , 1989) .

Asthma and Passive Smoking : Environmental tobacco smoke (ETS) is a combination of mainstream smoke (ms) , the smoke inhaled by the smoker , and the stream smoke (ss) , the smoke that comes from the burning end of the cigarette . Side stream smoke represents the major component of ETS . The central issue for health effects relates to quantitative differences in exposure between an active and an involuntary smoker , such as an infant or a child . Factors such as the number of active smokers in a given environment and the size and ventilation characteristics of that environment further , modify the exposure potential for the involuntary smoker (Tager , 1989) . The contribution of cigarette smoke to indoor air pollution and adverse health consequences of passive smoking have recently recognized as major health problem . Estimates vary , but children living in temperate climates spend 60% to 80% of their time indoors and approximately 70% of all children in the United States live in homes where there is at least one adult smoker (Fielding and Phenow , 1988) . Several lines of evidence suggest that exposure of infants

States live in homes where there is at least one adult smoker (Fielding and Phenow , 1988) . Several lines of evidence suggest that exposure of infants and children to ETS , especially that generated by mothers , plays a role in the occurrence and severity of asthma . Epidemiologic studies have demonstrated an increased occurrence of asthma and persistent wheezing in children of smoking mothers . In one such study , 18 to 34% of asthma was attributed to maternal smoking . Epidemiologic studies have demonstrated that bronchial responsiveness to carbachol or cold air is increased in children with smoking parents and that this observation is due largely to a greater of responsiveness in asthmatic patients with smoking mothers vs those with nonsmoking mothers . A large study of inner-city asthmatic children showed that asthmatic subjects with smoking parents appear to have more emergency room visits for asthma , even after adjustment for asthma severity and asthma self-management practices . Cigarette smoking has been shown to increase levels of IgE and enhance the occurrence of a specific IgE response to aeroallergens . Cord blood IgE levels have been found to be increased in infants whose mothers smoked during pregnancy . Infants of these mothers were found to have a four-fold excess risk of developing clinically manifest allergic disease state . Thus , exposure to ETS may function as an environmental agent that enhances biologic mechanisms that relate to bronchial responsiveness and asthma (Tager , 1989) .

It was demonstrated in a series of studies that maternal smoking is associated with diminished lung size and decreased pulmonary functions as measured by forced expiratory volume in 1 second , forced vital capacity , or forced expiratory flow , mid-expiratory phase among older children , suggesting long-term negative effects on children's pulmonary function (Murray and Morrison , 1986) . Several studies have suggested that children exposed to environmental tobacco smoke in their homes may be at increased risk of developing asthma . Weitzman et al , 1990 , studied the relationship between maternal smoking and 1- the prevalence of childhood asthma among children aged 0 to 5 years , 2- the likelihood of taking asthma medications prescribed by a physician , 3- the age of onset of children's asthma , and 4- the numbers of hospitalizations . Their results indicate that maternal cigarette smoking is associated with higher rates of asthma , an increased likelihood of using asthma medications , and an earlier onset of the disease among children 0 to 5 years of age . They reported that children aged 0 to 5 years whose mothers smoked 10 or more cigarettes per day were 2.1 times as likely to have asthma than children of non-smoking mothers , and are four times as likely to use asthma medications as are children whose mothers do not smoke . Martinez et al , 1992 , studied children at ages 0 to 5 years and then again several times during childhood and up to the age of 11 years . they found that among children who did not have asthma at ages 0 to 5 and whose mothers had 12 or

less years of education , incidence of new cases of asthma was 2.5 times higher if the mother smoked 10 or more cigarettes per day than if the mother did not smoke . No relationship was observed between maternal smoking and asthma incidence among children of mothers with more than 12 years of formal education . It seems that exposure to cigarette smoke may cause asthma in susceptible children . The mechanisms by which environmental tobacco smoke may cause asthma are not well understood . Ronchetti et al , 1990 , showed increased sensitization to aeroallergens , increased IgE levels , and increased prevalence of eosinophilia in children of smoking parents . Ronchetti et al , 1992 , hypothesized that parental smoking is a factor that , together with specific allergenic exposure , may enhance allergic sensitization in children and increase the risk of asthma particularly in their sons . The mechanism by which ETS enhances sensitisation to aeroallergens is unknown .

The studies by Zetterstrom et al , 1981 , in rats suggest that a direct contact between the allergen and the bronchial mucosa is necessary for this increased sensitization to occur . This support the hypothesis that a disruption of the bronchial epithelium by tobacco smoke with increased permeability to antigens may be involved . It is also possible that the disruption of the epithelium may be the consequence of an inflammatory process occurring in the mucosa , which alter the mechanisms by which the aeroallergens are handled and presented to immuno-competent cells in the lung . Active cigarette smoke

has been found to be associated with changes in T lymphocyte function and it is possible that these changes may alter the complex immunological mechanisms involved in the regulation of IgE production (Ronchetti et al,1992).

Asthma and Air Pollution : Rapidly increasing , and often uncontrolled , urbanization , as well as expanding mining and processing industries , have multiplied the environmental factors encouraging the development of nonspecific airways disease . These diseases cause a real public health problem because of their frequency and their medical and social cost . Air pollution is the first factor to be put forward to explain the differences observed between urban and rural areas . In the cities , such as Algiers , differences have been observed in prevalence rate of asthma among students living in highly polluted areas , 4.1% compared with 3.1% among students from less polluted areas . Similarly , in Southern Africa , African Xhosa schoolchildren living in rural areas have a 0.15% prevalence , whereas those living in the suburbs of Cape Town have a prevalence of 3.2% (Chaulet , 1989) .

There is evidence that a move from a rural to an urban community increases risk for asthma , in Tokelau islanders the prevalence of asthma in the island is 1.3% , whereas among those who moved to New Zealand it is 6.9% (Macklem , 1989) . Wire reclamation incineration is a source of environmental contamination , Wang et al , 1992 , compared the bronchial responsiveness of

primary school in the polluted area to those living in a non polluted area , this is to investigate if long term exposure to air pollution resulting from wire reclamation incineration might have harmful effects on the airway reactivity . In this study , all the children in polluted areas have suffered from long term exposure to airpollution caused by outdoor wire reclamation incineration since their birth . Their results showed that children in the exposed area had a greater incidence of pulmonary function abnormality than the control children . They found that the bronchial responsiveness was increased in children living in the exposed area compared with those of the controls .

Avol et al , 1990 , concluded that atmospheric pollution by smoke , sulphur dioxide , and nitrogen dioxide provoke progressive impairment of ventilatory function and airflow obstruction in control subjects and in asthmatic patients . Tsing and Li , 1990 , found that prolonged exposure to low concentrations of atmospheric sulphur dioxide might induce airway inflammation and bronchial hyperreactivity and predisposes to episodes of childhood asthma . Sultz et al , 1970 , reported a significant correlation between levels of air pollution and the number of children hospitalised for asthma .

There is a need for close and thorough investigation of all risk factors for asthma in every country and in every region to determine the conditions of everyday life and of the particular social evolution of various human populations

, the respective role played by each of these risk factors on one hand and the influence of urbanization , general health education of the community , and medicalization of its health problems on the other hand (Chaulet , 1989) .

Risk Factors For Triggering Attacks :

Several studies have suggested that viral infections can precipitate wheezing , others have demonstrated a role for passive smoking exposure , still others have emphasized a genetic predisposition to asthma . An association between allergens and asthma has been suggested for many years . In a large number of instances , the pollen of grass and other flowering plants has been held to be the most active and efficient of all causes . Over the past few years evidence has accumulated that sensitization to inhalant allergens is common in children with asthma over the age of 4 years . In most countries the commonest source of allergen appears to be the dust mite , and study of exposure has become easier with the development of simple techniques for measuring exposure in houses (Duff and Mills , 1992) .

Viral Respiratory Infections and Asthma : A number of studies have provided evidence for a temporal relationship between respiratory infections and acute episodes of wheezing in children with asthma . McIntosh et al , 1973 , prospectively correlated acute episodes of wheezing in 32 hospitalized children with asthma aged 1 to 5 years with clinical , microbiologic , and serologic

evidence of respiratory infection . Over a 2-year time period , 139 episodes of wheezing were identified , of which 58 (42%) were associated with documented viral infection . The viruses isolated most frequently were respiratory syncytial virus , parainfluenza virus , and coronavirus ,. Bacterial pathogens also were identified , but their presence did not correlate with asthma severity . Minor et al , 1974 , performed a similar prospective study in 16 nonhospitalized , nonatopic children with asthma aged 3 to 11 years with a history of four or more respiratory infection-induced exacerbations in the preceding year . Acute episodes of wheezing were correlated with clinical , microbiologic , and serologic evidence of respiratory infection . Over 7 months , 61 episodes of wheezing occurred , 42 associated with clinical evidence of viral respiratory infection , of which 23 (38%) were documented by culture or serologic studies , rhinovirus and influenza A were the most frequently identified viruses in this slightly older cohort . Only one episode of wheezing occurred in association with a bacterial respiratory infection .

Minor et al 1976 , performed another study using an older cohort of 41 children (aged 3 - 17 years) identified 71 episodes of wheezing , 17 (24%) with corresponding evidence of viral infection . Rhinovirus and influenza A were again the most frequently isolated organismus . Roldaan and Mansural 1982 , performed a prospective study of 32 older children (aged 9 - 15 years) with atopic asthma and correlated acute exacerbations with clinical , microbiologic ,

and serologic evidence of infection . Over a study period of 3 to 36 months , 58 episodes of respiratory illness were identified clinically . Thirty-eight of these episodes were associated with wheezing , of which 19 (54%) were accompanied by tissue culture or serologic evidence of viral infection .

Influenza A was the most frequent viral isolate . A similar prospective study on subjects with atopic asthma was performed by Carlsen et al , 1984 , on 169 wheezing occurred , 73 (29%) with evidence of viral infection . Rhinovirus and respiratory syncytial virus were the most frequent isolates .

Most recently , Mertsola et al , 1991 , prospectively followed 54 children (aged 1 - 6 years) with a history of two or more attacks of "wheezy bronchitis " and one or more hospitalizations for wheezing . One hundred fifteen upper respiratory were identified clinically , 76 associated with wheezing and 30 (39%) with a documented viral etiology. Coronavirus and rhinovirus were implicated in more than one half of these infections . Together these studies indicate an overall 24% to 54% incidence of acute exacerbations of asthma associated with viral infections in both atopic and nonatopic patients . An age-dependent relationship between wheezing and viral infections seems to exist : respiratory syncytial virus and coronavirus are isolated more frequently with wheezing in younger children , influenza A in older children , and rhinovirus in children of all ages (Cypcar et al , 1992) .

Although viral respiratory infections are important triggers of acute

exacerbations of asthma in children, the role of bacterial respiratory infections is less clear. At the present time, there is little evidence to suggest that bacterial respiratory infections (other than sinusitis) can influence asthmatic symptoms (Cypcar et al, 1992). Rachelefsky et al, 1984, treated 48 children with asthma with active sinusitis and found dramatic clinical improvement in clinical symptoms (cough and wheezing), decreased bronchodilator use, and improved pulmonary function.

Food Allergy and Asthma : The role of food allergens in asthma historically has been controversial subject and difficult to prove, possibly because of the lag time between consumption and manifestation of symptoms (Duff and Mills, 1992). Food allergy is believed to play a greater role in early infancy, when the intestinal barrier is immature and relative deficiency of IgA allow absorption of large dietary antigens, leading to sensitization in susceptible individuals (Walker, 1987). The prevalence of food sensitivity in the pediatric population is from 0.5% to 8% and can be manifested by atopic dermatitis, gastrointestinal disturbance, rhinitis, anaphylaxis, urticaria, and asthma (Bock, 1987). The percentage of children who wheeze because of food hypersensitivity is small. Bock, 1987, has performed more than 1000 double-blind placebo food challenges in children with a history of suspected food reactions. He found that 3.5% of all challenges had asthma in

combination with skin or gastrointestinal symptoms . Chiaramente and Altman , 1991 , found that only 1% had wheezing as the sole manifestation of a positive food reaction . Sampson and Scanlon , 1989 , in evaluating children with atopic dermatitis , some of whom had concurrent asthma , found that after a year of specific allergen avoidance , 31% of food hypersensitivities were no longer evident . Wheezing in response to food antigens is not uncommon as a parental complaint and , occasional cases of asthma primarily related to food sensitivity do occur . The evidence for the relationship is largely restricted to case reports (Duff and Mills , 1992) .

El-Hefny et al , 1992 , studied the incidence of egg and milk allergy in asthmatic Egyptian children , they found that most of the cases developed their first attack within the first two years of life . The mean of total serum IgE was significantly higher in asthmatic s compared to controls and was higher in egg sensitive compared to milk sensitive group .Skin test sensitivity to food allergens was highest to milk whole (21.2%) followed by egg yolk (17%) ,casein (15.8%) , egg whole (13.9%) ,egg white (11.5%) and a-lact albumin (10.9%) . A significant correlation was found between dietetic history to egg induced asthma and skin test values to egg allergens , a significant correlation was found between dietetic history to milk induced asthma and skin test values to milk allergens . They concluded that egg and milk allergy play a role in the aetiology of asthma in young Egyptian children and good dietetic

history is probably the most important factor in suggesting the presence of cow's milk or egg allergy. Skin test , prick method by itself can not be a diagnostic test for food allergy , but in association with good history , better results can be achieved .

Aeroallergens and Asthma : The evidence establishing a causal relationship between inhalant allergy and asthma is extensive . Specific sensitization occurring in genetically susceptible individuals and leading to signs of allergic disease has been demonstrated with many different indoor and outdoor allergens . Sensitization has been demonstrated by skin testing , serum IgE antibodies , and bronchoprovocation challenges . In 45% to 85% of patients with asthma , IgE antibodies to one or more inhalant allergens can be demonstrated (Duff and Mills , 1992) .

A - Indoor Allergens :

1 - House Dust Mites : Dust mites are probably the best studied of indoor allergens and the most common sensitizing allergen . They are 0.3 mm sightless eight-legs arthropode that are closely related to scabies mites , ticks and the species of mites that eat stored food . There are 47 species of mites in 17 genera in the family Pyroglyphidae . Different species predominate in different parts of the world . The most common species in North America are *Dermatophagoides farinae* and *D . pteronyssinus* . Other common species in

temperate and tropical regions include those from the genera Euroglyphus , Hirstia , Malayoglyphus , Pyrglyphus , Stumophagoides , and Blomia . Mites thrive in humid environments , which is the decisive factor for mite growth (Mills and Weck , 1989) . Indoor humidity is important and mites inhabit areas in the home that hold moisture , such as mattresses , carpets , and stuffed furniture . A single mattress can contain > 100,000 mites , whereas dust samples may contain > 100,000 mite fecal particles / gram of dust . Mites use human skin scales or the fungal flora growing on skin as their predominant food source , but they probably use a wide range of other foods (Mills and Weck , 1989) . Changes in home building construction in the last 25 years have been conducive to greater mite growth . Homes are more " energy efficient " and insulated , which maintains optimal temperatures and allows indoor humidity to rise . The increased use of wall to wall carpets and more plush furnishings provide an extensive haven for mites . The introduction of cool wash detergents may have improved conditions for mite growth , because mites are killed at 130 deg F but not at 70 deg F (Duff and Mills , 1992) .

In Egypt , El-Hefny et al , 1991 , found that the most common inhalant allergen found to cause asthma was house hold insect allergens (Cockroach , house fly , and mosquito) followed by house dust and house dust mite .

2 - Cockroaches : Cockroach debris is a common source of indoor allergen in patients , particularly those of lower socioeconomic status (Gelber

et al , 1991) . Morris et al , 1986 , reported that 47% of inner city children seen for allergic rhinitis and asthma were found to be cockroach sensitive .

Cockroach allergen appears to accumulate in the kitchen , but significant levels can be found on other floor surfaces in infested houses (Pollart et al , 1989) .

In Egypt El-Hefny et al , 1992 , in their study " The role of some household insects as inhalant allergens in Egyptian asthmatic children : Efficacy of immunotherapy in Cockroach asthma " demonstrated that the percentage of positive skin tests to common environmental allergens in children was as follows : house dust (64.5%) , house dust mite (46%) , *Alternaria* (33%) , grass pollens (32%) , *cladosporium* (27.5%) , *phoma* (20.5%) , *Helminthosporium* (17.5%) , *penicillium* (7%) , and *Aspergillus* (6.5%) . The percentage of positive skin prick tests to some household insects were as follows : cockroach representing (32.5%) followed by housefly (30.5%) , and mosquito (29.9%) of the total number of cases . The relation between cockroach and house dust allergens on basis of skin tests were as follows : 19.5% were positive reactor to both types , 45% to house dust only and 13% to cockroach only .

3 - Other Indoor allergens : Domestic cats are a major source of allergen in the home . Approximately 3% of the general population is allergic to cats ; of those who are allergic , one third may live in a house with a cat (Blay et al , 1991) . Cat allergen is found in cat saliva and on the pelts . Allergen can

be measured in the air and in house dust . Stuffed furniture and carpet are excellent reservoirs for cat allergen . Because of these reservoirs , it may take 16 to 24 weeks after a cat has been removed from the home to see a reduction in cat allergen levels below that of a home without a cat (Wood et al , 1989) . Dog allergen , like cat , can be detected in public places , including schools . Dog saliva and dog dander are the main sources of dog allergen (Duff and Mills , 1992) . Rodents as pets can be a source for indoor inhalant allergy , although they are less common than cats or dogs . The major source of rodent allergy is protein in their urine , some inner-city children may have difficulty with urinary allergen of rodents (Mills et al , 1987) . Molds (fungi) are found throughout the indoor and outdoor environments , with multiple species adapting to most climates . Molds can be a source for indoor perennial exposure , often growing on shower curtains , damp basements , and on indoor plants . The most commonly identified indoor molds are Aspergillus , Penicillium , and Rhizopus species (Duff and Mills , 1992) .

B - Outdoor Allergens : The sources of outdoor allergens include trees , shrubs , grasses , weeds , herbs , and molds . Most of the outdoor allergens exhibit a seasonal pattern as well as a geographic distribution . Most aeroallergens are in the form of pollen grains or spores . Pollen grains are part of the male plant's reproductive structures , and the pollen grains of each

species are distinct (Duff and Mills , 1992) . In Northern California there is a dramatic grass pollen season with high pollen counts starting in the first week of May . This peak is paralleled by a sharp increase in the number of patients presenting to the hospital at Travis Air Force base with asthma . Serum assays on the patients presenting with asthma show that sensitization to rye grass pollen is a major risk factor for asthma during that period (Pollart et al , 1988) .

Many children with asthma are allergic to common inhalant allergens , particularly dust mite , cat and cockroach . Continued exposure of sensitized children is considered to be an important cause of the ongoing inflammation in their lungs , which correlates with asthma . Clearly , if exposure to common inhaled allergens is a major cause of asthma , then reducing exposure is a logical treatment for the disease . It is possible that redesigning bedrooms for young children (i.e. , no carpets , covered mattresses and pillows , hot-washed bedding) would reduce the prevalence of asthma in the community . Currently the focus is on the correct management of the large numbers of children presenting with asthma . The evidence suggests that allergen avoidance should be the primary anti-inflammatory treatment for asthma in these children . The experience of many physicians is that the combination of inhaled anti-inflammatory drugs (i.e , cromolyn or low-dose inhaled steroids) and specific allergen avoidance procedures is the optimum treatment to control symptoms and minimize side effects in children with asthma (Duff and Mills ,

1992) .

Exercise-Induced Asthma (EIA) : Wheezing with exercise is considered a universal feature of asthma if the exercise challenge is sufficiently intense and conducted under the optimal conditions of temperature and humidity (McFadden , 1987) . This response to exercise can affect individuals of all ages and consequently becomes a significant limitation to a normal lifestyle for the patient with asthma . EIA is mainly caused by a transient hyperosmolarity due to loss of water and to cold air arising from mouth breathing during exercise . EIA can be prevented by avoiding exertions as far as possible . But daily life of children should not be impaired (Pierson , 1988) .

The clinical pattern of EIA is very characteristic . During the initial few minutes of exercise , there is usually a brief episode of bronchodilation , probably secondary to endogenous catecholamine release . If , exercise continues during the next 5 minutes and then stopped , bronchial obstruction begins and reaches its peak approximately 10 minutes later . Occasionally , EIA becomes progressively more severe for 30 to 60 minutes after exercise before it improves . In contrast , symptoms in adults often begin later and last longer than they do in children (Anderson et al , 1975) . Interestingly , many patients indicate that they can continue to exercise despite experiencing symptoms in the first minutes of exertion and , in fact , "run through" their asthma . EIA is less frequent with interrupted exertion or less severe exercise (

Busse , 1991) . The type of exercise often determines the probability of wheezing . Running is more "asthmagenic" than cycling or swimming , even though both involve similar work loads . The time since the last episode of EIA plays a role in the development of subsequent wheezing to exertion .

Consequently , there appears to be a refractory period that follows the initial episode of EIA , and this period can be protective for subsequent episodes of wheezing with exercise (Busse , 1991) .

Patients with asthma commonly recount that preliminary " warm-up " exercise may prevent or reduce symptoms of EIA and that they may be able to " run through " their asthma (Reiff et al , 1989) . A study by Schnall and Landau , 1980 , described the protective effects of repeated short sprints on a subsequent 6-minute treadmill run . Reiff et al , 1989 , reported a similar protective effect provided by a 30-minutes period of submaximal running on a treadmill . They concluded that a warm-up period of exercise can induce refractoriness to EIA without itself inducing marked bronchoconstriction .

Tal et al , 1984 , reported that exercise and cold air-induced bronchospasm have different physiologic mechanisms , and that cold air testing can be used as a routine challenge to identify airway hyperreactivity in children . They concluded that isocapnic hyperventilation of cold air (IHCA) can be used as a routine challenge test to identify airway hyperreactivity in children , and that recovery occurs faster with IHCA than with the exercise test .

Psychosocial Factors as Triggers for Bronchial Asthma : A basic state of " primary hypersensitivity " of bronchial airways seems to be a common denominator in asthmatic individuals (Khan , 1977) . Beale et al , 1952 , have shown that the bronchial tubes of most asthmatics are in a constant state of mild to moderate constriction . In experimental animals , bronchial irritation by catheter , inhalation of bronchoactive chemicals , or inhalation of dust cause reflex bronchoconstriction , mediated by efferent vagal pathways (Widdicombe et al , 1962) . Normal humans also respond to various physical and chemical irritants with some bronchoconstriction , but the asthmatics manifest a highly exaggerated response to these substances . Reflex bronchoconstriction may be one of the underlying factors in the production of asthma precipitated by respiratory infection or exposure to sensitive allergens (Gold , 1973) . A " secondary hypersensitivity " to a large number of physical , chemical , mechanical , and psychological stimuli has been described (Khan et al , 1974) . This type of hypersensitivity , which causes a significant proportion of asthmatic attacks , appears to be acquired through the process of conditioning during the usual course of the illness . Both types of conditioning , instrumental as well as classical , seem to be involved in its acquisition . For example , hypersensitivity to a large number of environmental irritants , such as cold air , cold water , sulphur dioxide , hydrocarbons

ammonia , etc ; seems to be acquired through the process of classical conditioning . There is no evidence that these substances cause bronchospasm through an allergic reaction . The conditioned reflexes are usually established either during or immediately after an asthmatic attack which may have been initiated by some allergic reaction or an infection . After some repetition of this association , these substances alone may precipitate an asthmatic attack . It is concluded that the stimulation of the sensory nerve endings in the bronchial mucosa reflexly causes the constriction of bronchial musculature through the efferent link of the vagus nerve . Association of asthma with certain emotions appears to be established through the process of operant conditioning . For example , a child who has a great dependency need and fears separation , may learn (in operant manner) that asthmatic attacks bring him closer to his mother and relieve his fear . Later on , these fears or feelings , whenever aroused by the environment , may themselves trigger bronchospasm on the paradigm of operant conditioning (Khan , 1977) . Attempts to desensitize asthmatics to multiple conditioned stimuli have been only mildly successful because of an infinite number of possible precipitants and the difficulty in determining them from the reports of the patients (Moore , 1965) . Khan , 1977 , designed a study to assess the effectiveness of conditioned bronchial dilatation in the treatment of asthma in children . The treatment procedure involved a combination of biofeedback training and counter- conditioning . In

summary , the experimental treatment helped reduce the frequency , duration and severity of asthmatic attacks in both the experimental groups .

Yellowlees and Kalucy , 1990 , reported that among the most common accepted trigger factors of asthma are allergens , infections , physical irritants , chemical irritants , reflex reactions (eg , to cold temperatures) , exercise , and psychogenic factors , including hyperventilation . The relatively common occurrence of anxiety disorders in asthmatics has been well documented (Yellowlees et al , 1987) as the production of asthma attacks by suggestion (Luparello et al , 1968) , and the association between mood and pulmonary function (Steptoe and Holmes , 1985) . More recently , Hibbert and Pilsbury , 1988 , showed that hyperventilation preceded the exacerbation of asthma in an anxious patient . In this case teaching the patient to control his breathing after hyperventilation led to his being able to avoid attacks of asthma . The family issues in asthma have received wide attention , particularly with respect to asthmatic children where abnormally family functioning has been described as a risk factor for death due to asthma (Strunk et al 1985) . Abnormal family functioning has been noted in families of asthmatics who have survived a life-threatening episode of acute asthma where mutual anger and distrust , as well as mutual overinvolvement has been described (Yellowlees and Ruffin , 1989) .

Mothers of asthmatic children have been reported to be cold and unloving , unconsciously rejecting , overtly rejecting , hostile and punitive ,

loving but only in condition that the child meets her demands and overprotective , in a series of studies . It stands to reason that certain mother characteristics may lead to prolonged stress for her child and thus decrease resistance to disease (Marx et al , 1986) . Parental denial or overprotectiveness , inappropriate handling of the asthma , family tensions or conflict , and the child's emotional reactions to asthma can each exacerbate the illness as a result of , emotional arousal , suggestion , conditioning , or hyperventilation (Lask , 1992) . During the acute attack of asthma , the patient experiences physical symptoms such as breathlessness and wheezing and also accompanying emotional symptoms such as anxiety and arousal . On the basis of conditioning theory , it may be postulated that this heightened arousal level becomes associated with asthma so that a vicious circle is established . The rationale of relaxation therapy is based on the theory that emotional stress can act either as a precipitator and / or exacerbator in acute and chronic asthma . Relaxation is seen as the anti-thesis to stress and may interrupt the continuing cycle between physical and emotional symptoms (Erskine and Schonell , 1979) . It is reported that relaxation is more effective with emotional or " suggestible " non-severe asthmatics (Phillip et al , 1972) . Teiramaa , 1978 , b , reported that the results of his study showed a strong interdependence between the course of asthma and psychic and psychosocial factors . The results suggest that a low level of introversion and / or lack of

psychic symptoms as indicated in a psychiatric interview have an especially beneficial effect on the prognosis of asthma . Plutchik et al ,1978 , stated that the result of their study confirms that treatment of the asthma patient should be directed not only towards improving indices of lung functioning but should also be directed towards the improvement of emotional factors .

Other Possible Agents As Triggers For Asthma Attack : There are components of indoor air other than allergens that may be harmful for asthmatic child such as : Tobacco smoke : An increased incidence of asthma has been reported in children who live in a home where the mother smokes (Gortmaker et al , 1982) . In addition children with asthma who are exposed to maternal smoking have poorer pulmonary function , a higher requirements for medication especially during the winter months , and more frequent emergency department visits (Murray and Morrison , 1988) . Unlike some aeroallergens , tobacco smoke consists of very small particles that tend to remain air-borne for long periods (Expert Panel Report , 1991) . Wood smoke : Although smoke from wood-burning heating stoves is not an allergen , it has been reported to increase lower respiratory symptoms in children (Honicky et al , 1985) . Strong odors or sprays produced by cosmetics (e.g . , perfume , talcum powder) , room deodorizers , cooking (especially frying) , household cleaning products , and fresh paint , these may irritate patient's airway and trigger asthma symptoms . Those affected by such odors should avoid them (

Expert Panel Report , 1991) . Air pollutants : Exposure to oxidants such as ozone and sulfur dioxide has been associated with worsening pulmonary function and increased airway hyperresponsiveness in patients with asthma . These environmental exposure may interact with allergens and other triggers in the pathogenesis of clinical asthma (Expert Panel Report , 1991) . Rhinitis , Sinusitis , and Nasal Polyps : The nose prepares air for the lungs by adding moisture and by removing both particulate matter and gases . Maintenance of nasal patency and function will contribute to asthma control . Of particular current interest is the possible relation between sinusitis and activation of asthma . It is possible , but unproven , that sinus infection may lead to aggravation of asthma through reflex mechanisms . Consequently , treatment of sinusitis may lead to more effective control of asthma (Expert Panel Report , 1991) . Aspirin Sensitivity : From 5 to 20% of adults with asthma experience severe and even fatal exacerbations of bronchoconstriction after ingestion of aspirin or certain nonsteroidal anti-inflammatory drugs . The prevalence increases with increasing severity of asthma . It is recommended that all patients with asthma be counseled to avoid this group of medications and to employ safe alternatives as acetaminophen or sodium salicylate (Expert Panel Report , 1991) . Sulfite Sensitivity : Sulfiting agents have been used to preserve foods and beverages since ancient times . They maintain the crisp and fresh appearance of foods , prevent browning , and control microbial

growth and spoilage (Simon , 1989) . The agents employed include sulfur dioxide as well as the sodium and potassium salts of sulfite , bisulfite , and metabisulfite . All these agents release sulfur dioxide gas under suitable conditions of warmth and acidity . Exposure to sulfites , particularly in the setting of restaurant salad bars , has been incriminated in many severe and even fatal asthma exacerbations . Studies have incriminated sulfur dioxide released from sulfites in the mouth and perhaps in the stomach as the precipitant of asthma in the vast majority of patients (Bush et al , 1986) . Major sources of exposure to sulfites are processed potatoes , shrimp , dried fruits beer and wine . Another source of sulfite exposure for patients with asthma is medication . Sulfites are employed to prevent oxidation of beta-adrenergic agonists . For this purpose , sulfites are contained in some nebulizer solutions , injected epinephrine , and injected local anesthetics containing epinephrine . Except in the rare individuals with true allergy to sulfites , the amount in the injected solutions is inconsequential . However , the amount in the nebulizer solutions is sufficient to cause paradoxical bronchoconstriction or at least blunted bronchodilator response in some individuals and should be avoided in the sulfite-sensitive patient (Taylor et al , 1988) . Tartrazine Sensitivity : A number of reports linked the yellow dye tartrazine , commonly employed in food and medication , with the occurrence of acute bronchoconstriction . This association was noted in those patients with

asthma who reacted adversely to aspirin . With more carefully controlled studies , it became apparent that these reports grossly overestimated the occurrence of tartrazine sensitivity in asthma patients . The incidence of tartrazine-induced asthma must be very low and may be limited to those rare individuals who have an immunologically mediated sensitivity to the dye (Expert Panel Report , 1991) .

Gastroesophageal Reflux : The presence of gastroesophageal reflux is increased at least threefold in both children and adults with bronchial asthma . Most of these patients have a demonstrable hiatal hernia . The relation of asthma to gastroesophageal reflux remains a matter of debate . In some studies , medical and surgical treatment the reflux resulted in improvement in symptoms of oesophagitis and decrease in asthma symptoms , particularly at night . Other studies have failed to document similar beneficial effect on asthma . When reflux lead to wheezing , the most probable mechanism is reflex vagal bronchoconstriction secondary to stimulation of sensory nerve fibers in the lower esophagus (Expert Panel Report , 1991) .

Risk Factors For Hospitalization :

Recent therapeutic advances have made it possible to better control the symptoms of childhood asthma than has been possible in the past . It is paradoxical that despite these advances , the hospitalization admission rate for

asthma and asthma mortality have reportedly increased in recent years (Halfon and Newacheck , 1986) .

Richards , 1989 , demonstrated the data concerning 100 children admitted to Childrens Hospital of Los Angeles because of status asthmaticus between February and June , 1988 . He reported that the patients tended to be young (median age 4 years , age range 7 months to 16 years) , there were more boys than girls (2.4 to 1) , black children outnumbered Hispanic and White children 1.5 to 1 and 3.5 to 1 respectively . More than half of the study children came from single-parent families . Positive family history of atopy was present in 83% of cases . Seventy percent of patients received public assistance vs 53.4% for general admission patients . 68% of patients received care by a community or the hospital pediatricians and 21% by allergists . Only 6% used emergency rooms and 3% used family physicians as their primary care providers . All who had private physicians had seen them within 3 months prior to hospitalization and on several occasions during the previous year . Three children had never wheezed previously and one had not wheezed for 12 years prior to admission . 94% required at least one emergency room or physician visit for treatment of acute asthma in the previous year . 70% had ever been hospitalized , 69% had been hospitalized within the previous 2 years and 34% were hospitalized more than three times during that period . 14% had previously required intensive care unit admission

because of asthma . A significant number were disabled , as manifested by frequent school absences , compromised physical activities , and disturbed sleep . Significant number never had adrenergic aerosols in any form prescribed for them . For those who were prescribed adrenergic agents with metered-dose inhaler without a spacer device , many of them their technique of administration was severely flawed so as to interfere markedly with delivery of medication . On the other hand , all patients using a spacer device exhibited proficiency in administering the sprays . 33% of children who had symptoms more frequently than once a month and who were cared for by non-allergist , no continuous use asthma medications had ever been prescribed , 63% recieved continuous use oral bronchodilators , and only 4% received cromolyn sodium or inhaled steroids . The technique of administering inhaled medication using a metered-dose inhaler has found to be wanting in most cases , particularly in children , because synchronization between delivery of the aerosol by actuating the spray and inhalation is critical but difficult . The use of devices has been found to be of significant value in delivering inhaled medications to the lower respiratory tract by reducing the need for synchronization . Non of the patients using a metered-dose inhaler without a spacer device demonstrated an adequate technique of administration , whereas those using these devices did , attesting to the efficacy of these inhalation aids (Richards , 1989) .

Stein et al , 1989 , reviewed retrospectively , the management of children with severe acute asthma who required admission to the intensive care (ICU) , during 1982 to 1988 . Findings from history and clinical examinations indicated that preceding respiratory infection was precipitating cause of acute asthma in 74% of patients admitted . In the remaining patients , allergen exposure , weather changes , and poor compliance were responsible , and the precipitating cause was unclear in some patients . Among the patients who had received oral theophylline preparations at home , 57% of serum theophylline concentrations at the time of admission to the hospital were in the therapeutic range (10 to 20 mg / L) , 40% were sub-therapeutic , and 3% were in the toxic range . Twice as many boys as girls required admission for asthma . The majority of the acute asthma episodes were triggered by respiratory viral infections . The seasonal trend in ICU admissions for asthma during October , June , April and September . All patients had previously been documented as having asthma , indicating that underdiagnosis did not contribute to the need for ICU admission . 21 of the 89 children had more than one ICU admission during the study period , confirming that children whose asthma has caused one episode of respiratory failure are at high risk for the subsequent development of further episodes of respiratory failure . Although a significant proportion of patients were treated with prophylactic agents such as cromolyn sodium and inhaled steroids , more preventive treatment might have reduced morbidity .

The observation that the children had been symptomatic for a mean of 48 hours prior to admission to the ICU , suggests that parents and / or physicians have underestimated the severity of symptoms . The resulting delay in seeking hospital care have contributed to the need for ICU admission , because prolonged asthma episodes are generally more resistant to treatment . Despite this long prodromal period , only 23% of children received oral corticosteroids at home . It is possible that underuse of systemic corticosteroids have been a factor that influenced the necessity for ICU admissions . The authers concluded that delay in seeking medical care and underuse of oral corticosteroids at home have contributed to the need for ICU admissions . The mortality and morbidity for children with severe asthma who required ICU admissions are small , provided that bronchodilators and IV steroids are used optimally and patient who require mechanical ventilation are carefully selected .

Risk Factors For Asthma Deaths :

Death due to asthma can occur suddenly or after a prolonged attack that has not treated optimally (Carswell , 1985) . Since the 1960s most deaths have occurred outside of hospitals , although some continue to occur during hospitalization of asthma (Eason and Markowe , 1987) . Thorough reviews of cases have identified potentially avoidable factors contributing to deaths in a large percentage of cases . This was true both for deaths outside

as well as inside the hospitals . Issues most often cited were poor utilization of corticosteroids and bronchodilator drugs , poor cooperation of the patient with management of asthma , failure to recognize the severity of the attack . Patients , relatives and physicians associated with delays in starting appropriate treatment , and inadequate responses of medical services to emergency calls (Sears et al , 1986) . Circumstances surrounding deaths due to asthma were first investigated by Fraser et al , 1971 , who focused on bronchodilator and corticosteroid therapy . Excessive inhalation of bronchodilators was thought to have contributed to death in 37% of the patients , primarily by delaying medical advice . Johnson et al , 1984 , found four sets of circumstances in the fatal attack contributing to deaths : patient failure to recognize severity of the attack , rapid progression in attack severity , misjudgement in management , and delay from many causes .

Rea et al , 1987 , found many of those same factors . They noted that some of those who died were unusually fearful or anxious and some had premonitions of death 1 or 2 days before the event . The British Thoracic Society , 1987 , concluded that allergy play a role in the deaths of some patients . Miller and Strunk , 1989 , compared the courses in 12 children who died of an acute attack of asthma with courses in 12 children of comparable age and sex who had a life threatening attack of asthma but survived . The analysis of the information during the 6 months before the attacks showed that

the study children had a greater frequency of respiratory failure requiring intubation , a decrease in steroid use in the month before the attack , history of family disturbance , abnormal reaction to separation or loss , and expressed hopelessness and despair . During the period immediately surrounding the acute attack , study patients more often had attacks starting during sleep , but less frequently experienced vomiting during the course of the attacks .

Treatment of the attack by the parents was poor (primarily because of delays) in 7 out of the 12 children who died , but was also a factor in 6 of the 12 controls . The authors suggest that certain characteristics of asthmatic children place them at greater risk for death due to their asthma . They postulate that there may be inherent differences in the mechanisms of the acute attacks between the children who died and those who survived .

Birkhead et al , 1989 , stated that factors implicated in deaths from asthma include : inadequate recognition of asthma severity by the patient , family and medical care provider ; overreliance on B-adrenergic inhalant therapy leading to underuse of other medications or to delay in seeking medical treatment during an asthma attack ; psychologic factors , such as behavioral disorders or depression ; in addition , race and socioeconomic status have been related to higher mortality . The authors investigated a cluster of five adolescent deaths from asthma during a three month period in 1987 . They stated that despite the lack of evidence of common exposure , the

decedents shared many personal and medical characteristics . All were black patients and of lower socioeconomic status . All were adolescents and responsible for regulating their own medication schedules . Lack of appreciation of the severity of their asthma by medical personnel and the patient's families was evident . For example , two patients with severe asthma had not been prescribed inhaled corticosteroids . Four decedents tested and had markedly subtherapeutic or zero serum theophylline levels measured at the time of the fatal episode eventhough appropriate amounts of theophylline had been prescribed . Their theophylline levels were lower than levels in patients of the same age observed at the emergency room or hospitalized for asthma during the same time period . They concluded that continued efforts to educate adolescent patients , their families and medical care providers about the treatment of asthma are warranted .

Fletcher et al ,1990 , investigated 35 asthma deaths in children aged 1 to 16 years , they stated that a high proportion of childhood asthma deaths are preventable , 6 cases (17%) had experienced relatively mild and infrequent symptoms before the onset of the final illness . Thus , the widely held belief that only children with chronic severe asthma are at risk of dying is not true . Half the children in the study were obviously at risk of dying but this fact was insufficiently acknowledged at that time . Hospital discharge letters did not emphasize the life-threatening nature of previous attacks , nor were parents

properly instructed . Chronic undertreatment of asthmatic children is well documented and this , in association with poor supervision , was present in half of the cases . Closer attention should be paid to long term management , poor compliance may have influenced control of asthma . Over-reliance on inhaled bronchodilator was common . Non of the deaths appeared related to a direct adverse effect of nebulized treatment , but nebulizer over-reliance in one case had led to fatal delay in seeking further medical help . While they are extremely useful in the home management of children with severe asthma , nebulisers should not be issued without proper education of parents and child and meticulous attention to long term control . As for the emergency management , the most important finding was that 20 children who died more than six hours after the onset of the fatal attack had not the access to optimal emergency treatment . The parents consistently described very grave clinical signs but they had not been warned by professionals of the danger of such signs . Some families had become tolerant to severe symptoms while others , despite their anxiety , had been reluctant to call general practitioners at night or weekends . Deshpande and McKenzie , 1986 , showed the efficacy of oral corticosteroids in speeding recovery and preventing dangerous deterioration in childhood asthmatic attacks .

Fletcher et al , 1990 , stated that the use of self-initiated courses of oral corticosteroids is accepted practice in the management of adult

asthmatics but some pediatricians have tended to be reluctant to apply this to children . Such treatment does not generally cause significant long term hypothalamo-pituitary-adrenal axis suppression . In their analysis the authors estimated that a parent or patient initiated course of oral corticosteroids might have altered the outcome in at least 20 cases . They concluded that , if mortality is to be reduced , families of asthmatic children must be educated to recognize severe symptoms and be given an appropriate " crisis plan " . Hospitals should permit free access and have a clear protocol for the management of children with severe asthmatic attacks .

Pathophysiology of Bronchial Asthma :

The development of airway obstruction is responsible for the clinical manifestations of asthma . In mild asthma , there may be no obvious clinical evidence of airflow obstruction or any changes detectable during routine pulmonary function testing . More sensitive laboratory assessment may reveal airway hyperresponsiveness and abnormalities in peripheral airway function (Wagner et al ,1990) . In moderate and severe asthma , bronchial reactivity increases , and evidence of airway obstruction will be apparent upon physical examination and during pulmonary function testing (spirometry and peak expiratory flow rate measurements) (McFadden et al , 1973) . There is considerable lability in the responsiveness of asthmatic airways . Airway

narrowing may worsen gradually and persist despite therapy , but it can also develop abruptly and produce acute respiratory insufficiency . The changes associated with airway obstruction in asthma are thought to be initiated by the inflammatory events in the airways (Busse , 1989) . The airways of asthma patients are infiltrated by inflammatory cells , have epithelial disruption , and show evidence of mucosal edema . Airway inflammation is thought to be a primary mechanism responsible for airway hyperresponsiveness in asthma (Holgate et al , 1987) .

Airway Hyperresponsiveness (HPR) : Asthma is characterized by airway HPR , a condition manifested by an exaggerated bronchoconstrictor response to many physical changes and chemical and pharmacologic agents (Boushey et al , 1980) . Asthma patients develop clinical symptoms as wheezing and dyspnea after exposure to allergens , environmental irritants , viral infections , cold air , or exercise . Airway HPR is important in the pathogenesis of asthma , as it is ubiquitous in the disease . The level of airway HPR usually correlates with the clinical severity of asthma and medications requirements (Cockcroft , 1987) .

The level of airway HPR can be measured in the laboratory by standard inhalation challenge testing with methacholine or histamine as well as after exposure to nonpharmacologic stimuli as hyperventilation with cold dry air ,

inhalation of hypo- or hypertonic aerosols , or after exercise (Boushey et al , 1980) . Fluctuations in morning (a.m.) and evening (p.m.) peak expiratory flow rates (PEFR) reflect airway HPR and serve as a measure of airway HPR in asthma (Ryan et al , 1982) . Several mechanisms have been proposed to explain airway HPR in asthma , including airway inflammation , abnormalities in bronchial epithelial integrity , alterations in autonomic neural control of airways , changes in intrinsic bronchial smooth muscle function , and baseline airflow obstruction (Cockcroft , 1987) .

Airway Inflammation : The mechanisms contributing to airway inflammation in asthma are multiple and involve a number of different inflammatory cells . Asthma results from complex interactions among inflammatory cells , mediators , and cells and tissues resident in the airways (Beasley et al , 1989) . An initial trigger in asthma may be the release of inflammatory mediators from bronchial mast cells , macrophages and epithelial cells . These substances cause the direct migration and activation of other inflammatory cells (eosinophils and neutrophils) which produce alterations in epithelial integrity , abnormalities in autonomic neural control of airway tone , changes in mucociliary function , and increased airway smooth muscle responsiveness (Kowalski et al , 1989) . Evidences suggesting the presence of airway inflammation in all asthma patients , indicate that airway inflammation is a key factor . Therapeutic interventions that reduce bronchial inflammation in

asthma patients appear to decrease the degree of airway HPR (Bleeker , 1985) .

Inflammatory Cells and Asthma : Numerous leukocytes found in the circulation and within the lungs are important in the generation , amplification , and persistence of the inflammatory condition of the airways that is seen in asthma . Involvement of mast cells , eosinophils , and lymphocytes is important to varying degrees (Hill et al , 1992) . Mast cells are found throughout the walls of the respiratory tract , and increased numbers of these cells have been described in the airways of allergic patients with asthma . Once binding of the allergen to cell-bound IgE occurs , mediators such as histamine , eosinophil and neutrophil chemotactic factors , leukotrienes (C-4 , D-4 and E-4) , prostaglandins , platelet activating factor , and others are released from mast cells (Kaliner , 1989) . Decreased numbers of granulated mast cells are observed on histologic examinations of the airways of patients who have died from acute asthma attacks , suggesting that mast cell degranulation is a contributing factor in the progression of the disease (Holgate et al , 1986) . There has been an association between asthma and peripheral eosinophilia . The airway HPR has been related to the number of eosinophils in peripheral blood and bronchoalveolar lavage fluid (Wardlaw et al , 1988) . Major basic protein , a constituent of eosinophil granules , can produce direct damage to airway epithelium and is found in high quantities in the sputum of patients with

asthma . The damage to airway epithelium caused by eosinophil degranulation and the subsequent disruption of normal physiology contribute to airway HPR (Gleich et al , 1988) . Glucocorticoids decrease the eosinophil migratory response to an inflammatory stimulus in vitro and lower the number of circulating eosinophils and the number of eosinophils accumulating at allergen challenged sites in the skin and presumably airways (Zweiman et al , 1976) . Data obtained from an animal model of allergic inflammation in the airways revealed an accumulation of T lymphocytes in bronchial mucosa , corresponding to allergen-induced late asthmatic response (Frew et al , 1990) . Corrigan et al , 1988 , revealed that T lymphocytes and their activation markers in peripheral blood were increased in severe acute asthma and decreased with treatment and clinical improvement . Activated T lymphocytes from patients with allergic and nonallergic asthma are capable of secreting lymphokines that promote eosinophilia and prolong eosinophil survival (Walker et al , 1991) .

Important Mediators In Asthma : Many substances have been implicated in the pathogenesis of asthma . In the following part , mediators that are clinically relevant or having increasing importance are discussed .

Histamine : Associated with asthma for many years , histamine is capable of inducing smooth muscle contraction and is believed to play a role in producing mucosal edema and mucus secretion . Lung mast cells are important

source of histamine , its release can be stimulated by exposure of the airway to variety of factors , including physical stimuli (such as exercise) and allergen exposure (Kliner , 1989) .

Membrane-derived Lipid Mediators : Phospholipids occur in the membranes of the inflammatory cells involved in asthma pathogenesis . Several classes of important mediators , including arachidonic acid and its metabolites , prostaglandins , leukotrienes , and platelet activating factor are derived from these membrane phospholipids (Hill et al , 1992) .

Prostaglandins : When the enzyme cyclooxygenase breaks down arachidonic acid , the result is the formation of prostaglandins . Prostaglandins relevant to asthma include PGD₂ . PGD₂ is a potent bronchoconstrictor but has never been associated with the ability to produce sustained effects on airway caliber or be involved in amplifying an inflammatory response . The extent of its actions in asthma pathology remains to be determined (Hill et al , 1992) . Another bronchoconstricting prostanoid , PGF₂ alpha , enhances the effects of histamine on airway caliber (Fish et al , 1984) .

Thromboxanes : Thromboxane A-2 is produced by most cell types in the lung and have several properties , including bronchoconstriction , involvement in late asthmatic response , and involvement in the development of airway inflammation and hyperreactivity (Smith and Henson , 1987) . Development of thromboxane synthetase inhibitors has been encouraging . One such compound

, OKY-046 , has demonstrated the ability to attenuate airways reactivity for several days after inhalation (Fujimura et al , 1990) .

Leukotrienes : The most relevant group of arachidonic acid metabolites to asthma is the leukotrienes . The 5-lipoxygenase pathway produces leukotrienes C-4 , D-4 and E-4 . These sulfipeptide leukotrienes , formerly known collectively as slow reacting substance of anaphylaxis , are liberated during inflammatory processes in the lung and have significant effects on airway smooth muscle (bronchoconstriction) , mucociliary function , microvascular permeability , and airways edema (Piacentini and Kaliner , 1991) . Patients with asthma have greater airway HPR to inhaled LTD-4 than normal individuals , and these results paralleled those found with inhaled histamine (Bisgaard et al , 1985) .

Platelet Activating Factor : It is involved in the mediation of important steps during the development of asthmatic response (Smith , 1991) . These steps include immediate bronchoconstriction and induction of airway HPR , edema accumulation , and cellular changes associated with generalized inflammatory responses , including chemotaxis of eosinophils (Barnes et al , 1988) . Specific PAF antagonists have been developed , but their clinical effects have not been clearly established (Hill et al , 1992) .

Pathology of The Airways In Asthma : Damage to the airways epithelium and associated inflammatory changes are universal findings in

asthma in patient with mild to moderate disease (Laitinen et al , 1985) . Similar but more severe changes have been seen in patients who have died from severe asthma attacks . A correlation between the degree of epithelial denudation and airways reactivity suggests that patients with the most reactive airways have the least amount of normal bronchial epithelium (Jeffery et al , 1989) . Subepithelial fibrosis has been described in the bronchi of patients with mild asthma (Roche et al , 1989) . Epithelial damage is reversible , and favorable changes have been observed with long-term treatment with inhaled glucocorticoids (Lundgren , 1977) .

Microvascular Leakage : An important component of inflammation is the induction of microvascular leakage of plasma constituents from the tracheobronchial circulation . Vascular leakage is a component of a general inflammatory response and important feature in the pathogenesis of asthma (Erjefalt and Persson , 1989) . Vascular leakage of plasma proteins and the attendant passage of fluid occur within minutes after local allergen challenge in human airways . This passage of proteins through the subepithelial space toward the airway lumen , associated with mucosal edema and subsequent narrowing of airway caliber , contribute to the development of airway HPR (Persson , 1986) . Low to moderate doses of inhaled glucocorticoids (400 microgram / day) administered for one month has been demonstrated to lower protein concentrations in bronchoalveolar lavage (Van De Graaf et al , 1991) .

The Role Of The Nervous System In Asthma :

The most important categories of neural control of airway tone in humans are the parasympathetic and sympathetic nervous systems . Other types of innervation and mechanisms for neural control such as the nonadrenergic , noncholinergic (NANC) nervous system are present in humans , but their roles in asthma remain unclear . Local release of neuropeptides such as substance P may activate neural reflex mechanisms , resulting in increased microvascular leakage with mucosal edema and increased mucus secretion (Hill et al , 1992) .

Parasympathetic Nervous System : The parasympathetic nervous system is involved in the pathogenesis of asthma . Sensory nerve endings along the airway travel through the afferent vagus nerve to the central nervous system . Nerve impulses return to the lung by the efferent vagus and arrive at ganglia located throughout the lung . Postganglionic nerve release acetylcholine , which binds to cholinergic receptors located in airway smooth muscle resulting in bronchoconstriction . It is at this postganglionic cholinergic receptors that the anticholinergic bronchodilator atropine and its derivatives such as ipratropium bromide have their site of action (Hill et al , 1992) . In humans , three muscarinic subtypes have been identified with relevance to neural control of the respiratory system (Barnes , 1989) . Located on

autonomic ganglia , M1 receptors mediate vagal tone , and antagonism of these receptors results in bronchodilation . M2 receptors have a negative feedback effect on the cholinergic presynaptic junction , and antagonism of these receptors increase acetylcholine release . M3 receptors are located on postsynaptic receptors . Antagonism of M3 receptors results in bronchodilation (Barnes , 1989) . A pure M3 antagonist or a combination of M1 - M3 antagonists have a more desirable effects on the vagally mediated bronchoconstriction observed in asthma than any of the currently available nonspecific antagonists such as ipratropium or atropine derivatives (Hill et al , 1992) .

Sympathetic Nervous System : In humans , the sympathetic nervous system does not have direct innervation of airway smooth muscle (Richardson , 1979) . The bronchodilating responses seen in asthma are due to rich supply of B-adrenergic receptors in most airway smooth muscle . In normal individuals , the sympathetic nervous system is relatively passive in controlling airway caliber . B-adrenergic antagonists have little or no effect on airway tone in individuals without asthma , but can cause profound bronchoconstriction in individuals with asthma (Skinner et al , 1976) . B-adrenergic agonists have little effect on normal individuals yet are potent bronchodilators in those with asthma . The sympathetic nervous system is not highly active in maintenance

of airway tone but is important in reversing bronchoconstriction in response to adverse stimuli (Hill et al , 1992) . In lung , as in cardiac tissue , there is equal distribution of B-1 and B-2 receptors (Hedberg et al , 1985) . The functional significance of pulmonary B-1 receptors has not been fully established , but they may regulate mucus secretion . Pulmonary B-2 receptors are located in the lung from the trachea to the alveolus and mediate the profound bronchodilator response to B-2 agonists in patients with asthma . Other functional aspects of B-2 receptor stimulation include mucociliary clearance inhibition of antigen-induced release of mediators from mast cells , and prevention of microvascular leakage induced by histamine (Barnes , 1987) .

PROGRAMS IN MEDICINE :

The term Program (Programme) means plan of intended proceedings ; descriptive notice , list , of series of events , items , etc ; series of coded instructions for computer (Coulson , 1973) .

Examples of programs mentioned in the medical literatures are :

Establishment of a collegiate nursing education program . A lifelong dream of Anna D. Wolf was to establish nursing education in a university . Her first step toward this goal occurred in China in 1922 at the Peking Union Medical College to bring the best of scientific western medicine to China and to make nursing an acceptable occupation for educated Chinese women (Allison , 1993) .

A family physician -counselor program for medical students to identify and manage student needs was conducted at the Eastern Verginia Medical School . The program was helpful in identifying problems and implementing treatment strategies for medical students (Davies et al , 1993) .

Recovering staff and smoking in chemical dependency program is another program to solve the problem of cigarette smoking among those who

are recovering from alcohol dependence in rural Nebraska (Bobo and Davis , 1993) .

A comprehensive evaluation and treatment program for high-risk and human immune deficiency (HIV) virus positive adolescents was developed in New York city in 1987 , to address the unique manifestations of the disease among adolescents aged 13 to 21 years . Futterman et al , 1993 , stated that with the epidemic of HIV infection increasing nationwide among adolescents , specialized , comprehensive programs are needed to counsel and treat HIV-infected adolescents and youth in high-risk situations .

In Denmark , pregnant women are offered antenatal care in a nationwide programme . This programme is organized around health examinations in general practice , hospital outpatient departments and at midwives centers (Lindenkov et al , 1993) .

In Egypt , the child survival project of the ministry of health developed the acute respiratory infection control program . The main objectives are : to reduce mortality from acute respiratory infections in particular pneumonia , to reduce the severity and complications of acute upper respiratory infection , to reduce inappropriate use of antibiotics and to reduce the incidence of acute lower respiratory infections , in particular pneumonia (Khailaf and Lamb , 1991) .

Asthma Self-Management Educational Programs :

Educational self-management programs for children with asthma and their families have proliferated in recent years.

Several educational programs for children with asthma have produced improved skills in management, increased knowledge about asthma for both patients and parents, and fewer emergency room visits and days of hospitalization. Testing theories of health education is difficult in practice, especially in the case of asthma, which is an unpredictable illness often requiring complex treatment regimens. However, more than a dozen studies of self-management programs for patients with asthma have been reported in the literature; nearly all of these have been applied to children. A number of these studies either were uncontrolled or did not include morbidity assessments as outcome measures (Rachelefsky , 1987) .

Educational programs for asthma are commonly termed asthma self-management education . Self-management education provides the patient with educational experiences specifically designed to promote changes in behaviour that will lead to an improvement in health status and ultimately enhance the quality of life (Wilson-Pessano and McNabb , 1985) .

Educational programs for patients with asthma offer them the opportunity to increase their understanding and help them manage the

condition more effectively by participating as active partners with their physician in controlling the disease . These programs are educational programs ; they are not intended as treatment for severe behavioral or psychiatric disorders nor as the sole basis for treatment of asthma (Wilson-Pessano and Mellins , 1987) .

Examples of pediatric asthma-self management programs including the name of the program , the institute and the references are listed below :

ACT (Asthma Care Training) : University of California , Los Angeles . USA .

(Lewis et al , 1984) .

Air-Power : American Institutes for Research in the Behavioral Sciences , Palo Alto , California . USA .

(National Heart , Lung and Blood Institute , 1984 a) .

Air-Wise : American Institutes for Research in the Behavioral Sciences , Palo Alto . California . USA .

(National Heart , Lung and Blood Institute , 1984 b) .

Living with asthma : National Asthma Center , Denver , Colo. USA .

(National Heart , Lung and Blood Institute , 1985) .

Open Airways / Respiro Abierto : Columbia University , New York . USA .

(National Heart , Lung and Blood Institute , 1984 c) .

Family Asthma Program : State University of New York - Buffalo . USA .

(Hindi-Alexander and Cropp , 1984) .

Superstuff (self-help) : ALA (American Lung Association) .

(American Lung Association , 1982) .

Teaching My Parents About Asthma (self-help) : University of Texas ,

Galveston . USA .

(Parcel et al , 1980) .

You Can Control Asthma : Georgetown University , Washington , D.C.

(Parker , 1987) .

Educational / self-management programs to help children with asthma have been described in outpatient clinics in hospitals, medical office settings, school settings, computer-oriented instructions, camp programs, short-term community agency programs, and as isolated one-time interventions (Rachelefsky , 1987) .

CHILDHOOD SELF_MANAGEMENT PROGRAMS IN OUTPATIENT

MEDICAL FACILITIES :

Clark et al , 1986 , evaluated their health education program (Open Airways) targeted for children from low-income urban environments followed in a hospital setting. Their study was designed to evaluate whether the health education program would reduce emergency room visits and hospitalization,

and whether the effect of education would be greater among those with high baseline health care use than among those with low use. Two hundred ninety families with 310 children (between 4 and 17 years of age) were evaluated; 207 participated in the health education program and 103 received regular clinic care. The basic program involves six 1-hour sessions (in English and Spanish). Ten to 15 families are taught at one time, with areas covered including asthma attack, taking medicine, communicating with physician, improving school performance, maintaining a health home environment, and establishing guidelines for the child's physical activities.

Health care use 1 year before program delivery (baseline) and 1 year after program delivery (follow-up) were determined. The overall results demonstrated no significant decreases in health care when the experimental group was compared with the control group. However, decreases in health care use were observed among children who had made use of health care facilities before the program. In evaluating cost savings for the health care program, the authors estimated a saving of \$11.22 for every \$1.00 spent to deliver health education to the children with one or more hospitalizations.

In another study the Clark et al , 1984 , evaluated the effects of their program on school performance and on the adjustment of children with asthma . They observed a favorable impact on school-related problems. Although not significantly different, program group children did show increased attendance as compared with controls. Children in the treatment group were

significantly more able to maintain their grade levels than were those in control group.

Creer et al , 1985 ,developed a program (Living with Asthma) at the National Asthma Center in Denver. They evaluated 147 children. Living with Asthma is an 8-week, health education program . Parents and children are taught the same material,which includes physiology of asthma,trigger factors,treatment(s), and self-management skills.Patients monitor their disease with a daily diaries and peak flowmeters. The authors noted a significant increase in knowledge in the treatment group.In addition,there was a decrease in the number of attacks per month and a reduction in school absences when the year before the intervention was compared with the year after the intervention.

Hindi Alexander and Cropp , 1984 , developed the Family Asthma Program as a 6-week,12-hour program that reviews the clinical features,pathophysiology,prognosis, and treatment of asthma. The children (6 to 14 years old) in this program were also involved in an activity program that included games,swimming,and relaxation training. The investigators evaluated 92 children over a 1-year follow-up period with patients acting as their own controls. Daily diaries were kept for 3 to 6 months before and for 12 months after the intervention,covering drugs,morbidity, and disability. There was a significant increase in knowledge about asthma and a significant change in

attitude toward asthma . In addition, total childhood activities increased, and school absences decreased.

McNabb et al , 1985 , developed an individualized self-management program. Airwise, was evaluated to determine whether those who had not been compliant would benefit from self-management education that could be tailored to their identified educational and behavioral needs. Designed to be used in a clinical setting, Airwise consists of four 45-minute weekly sessions. The intervention is conducted in a standardized manner, although it is adapted to the individual child's needs. Fourteen subjects between 9 and 13 years old were paired and were randomly assigned to either an experimental group or a control group of seven children each. In a comparison of the 12-month posteducation period with the previous 12-month period, the frequency of experimental group emergency visits was less than those of the control group. There was no difference in posttreatment nonemergency visits or drug scores between the groups. The authors estimated an immediate cost savings of \$ 507 per year per child following their program.

Lewis et al , 1984 , evaluated their asthma care training (ACT) for kids with a randomized controlled trial. The ACT program uses the analogy of " you're in the driver's seat." To facilitate learning, an analogy is drawn between maintaining health and maintaining and safely driving an automobile. A key concept in safe driving is recognizing and obeying traffic signals-green for go,

yellow for caution, and red for stop. Daily control of asthma symptoms means taking medications that are color-coded green to keep going and prevent symptoms. When mild symptoms develop, it is time to slow down and to take a more cautious approach, which involves taking medications that are colored yellow. When more symptoms occur, it is time to take red medication to stop the episodes. In addition, children color code their symptoms and their degree of sensitivity to various allergens and irritants. The ACT program consists of five 1-hour sessions.

Seventy-six children between 8 and 12 years old with chronic asthma were randomly assigned to control and experimental groups. The control group received 4.5 hours of lecture presentations on asthma and its management. The experimental groups underwent the ACT program. Results included equivalent increases in knowledge and changes in beliefs in both groups, and significant reductions in emergency room visits and days of hospitalization among those receiving the experimental treatment as compared with the control group. These changes represent an estimated savings of approximately \$ 180 per child per year for those in the experimental group.

Fireman et al , 1981 , reported a controlled trial of health education based in a physician's office with 26 children with asthma (13 study group, 13 control group) 2-14 years old. Children and their parents were given 4 hours of

individual instruction in all aspects of asthma management by a trained nurse educator, followed by regular group discussions and telephone access to the educator over the course of 1 year. A nonphysician educator was used because a physician's office routine makes it difficult for patients to ask questions of their doctors.

The education curriculum provided by the nurse educator to the patient and the family included a description of the anatomy of the lung, review of elementary pulmonary physiology and pathophysiology, and an explanation of asthma trigger factors. The importance of avoidance and control of the patient's environment was stressed.

The outcome, as measured by school and medical records, symptom diaries, and telephone interviews, was a reduction in school absence and significantly fewer hospital attendances and acute attacks of asthma in the treatment group. Estimated hospital and emergency room costs were much less in the treatment group. It appeared that the educational program, along with access to a trained nurse, was successful because of improved comprehension and compliance, more use of prescribed medications, and earlier initiation of asthma therapy. The educated group knew how to prevent the development of asthma by earlier recognition of symptoms along with their earlier initiation of drug therapy.

OUTPATIENT COMPUTER-ORIENTED PROGRAMS :

Rubin et al , 1986 , devised an asthma-specific computer game (asthma command) to convey asthma information to children 7 to 12 years old. Information presented includes symptoms recognition, aggravators, medications, appropriate use of health providers, and encouragement to attend school. The game tests knowledge and is based on a point system. Fifty-four children with moderately severe asthma were studied in a randomized controlled design. Twenty-five subjects played Asthma Command for 40 minutes while 29 played routine computer games. Children were seen every 6 weeks for 10 months.

Compared with children in the control group, experimental subjects showed improvement in knowledge ($p < 0.001$), in behavior related to the management of asthma ($p < 0.008$). However, they failed to demonstrate differences in morbidity data in terms of visits to physicians, emergency rooms, and hospitals.

SELF-ADMINISTERED PROGRAMS :

Weiss , 1981 , conducted Superstuff which was developed as a self-administered self-management package for children with asthma who are of elementary school age, who are receiving medical care, and whose disease is severe enough to disrupt their lives. It is designed to be mailed to parents of children with asthma between 7 and 12 years old and to be administered without professional supervision. The parent's magazine and the children's kit

(cost \$ 7.00) focus on asthma facts and fallacies, internal and external signals and triggers, relaxation techniques, and personal control and decision making.

The purpose of the study was to assess the degree of improvement in the management of asthma and asthma-related behaviors as a result of Superstuff. Forty-three children with asthma between 7 and 12 years old either were "treated" with Superstuff (20 children) or entered a no-treatment control group (23 children).

Superstuff enhanced self-management skills ($p < 0.02$), especially in the older children. The reports of both parents ($p < 0.04$) and physicians ($p < 0.05$) demonstrated a significant decrease in interruptions due to the progression of asthma.

SCHOOL-BASED PROGRAMS :

Parcel et al , 1980 , developed a program that teachers and children can use in a school setting that also involves parents and health providers. The primary teaching tool for the program is a patient education book, 'Teaching My Parents About Asthma'. The book was designed to develop the skills of observation, discrimination, communication, decision-making, and self-reliance. The material is appropriate through the fifth grade. Instruction includes a 40-minute class once a week for 24 weeks conducted by a voluntary team of teachers, or school nurses.

Fifty-three children with asthma participated in the educational program and 51 children who did not participate during the first year served as a control group. After the first year, the control group received the intervention. Children in the treatment group increased their knowledge about asthma significantly, and those in the control group did not. The increase in knowledge was replicated in the control group after they received the educational program. The greatest differences between groups were in the areas evaluating measures to prevent an asthma attack. Frequency of asthma attacks and health services utilization were not significantly affected by this program.

Whiteman et al , 1985 , evaluated an asthma self-care program (Self Care Rehabilitation in Pediatric Asthma) for both preschool (2 to 5 years) and school-aged (6 to 14 years) children. The curriculum was designed to present information and skills to allow children to control their asthma, and includes physiology of asthma, medications, recognition of triggers and symptoms of asthma. Proper breathing, relaxation skills, chest physiotherapy, and physical conditioning are all emphasized. The preschool program consists of six 1-hour classes scheduled twice a week for 3 weeks. The school-aged curriculum consists of eight 90-minute classes for both child and parents scheduled twice a week for 4 weeks.

Twenty-one preschool children, serving as their own controls, and 38 school-aged children randomly assigned to a study or control group were evaluated before and 3 months after intervention. Results demonstrated a

statistically significant decrease in the number of asthma episodes but no change in the severity in all three groups. The school-aged group in pre- and posttesting demonstrated an increase in knowledge and skills in the study group as compared with the control group.

Kubly and McClellan , 1984 , evaluated the effects of a self-care asthma education program on the asthma self-care activities in children with asthma. Twenty-eight second,third, and fourth grade students were evaluated, 15 in the control group and 13 in the experimental group. Parents and children were taught by one of the authors or a nursing student in 3- to 4-week sessions (60 to 90 minutes each). The control and experimental groups were lectured on asthma facts , while the latter were also taught self-medications and breathing exercises . The experimental group demonstrated a more asthma control, but no significant difference was found between the groups in asthma self-care activities.

As stated by the authors, school nurses are in an ideal position to conduct such programs because they deal with the daily management of the child with asthma within the family and community. They can also contribute to the maintenance of self-care behaviors through daily follow-up and acting as an accessible resource to parents and children.

Because of increased recognition of the patient's important role in

chronic disease management , asthma education has emerged as an essential component in the medical treatment of childhood asthma . Asthma management requires enhanced communication and close cooperation among child , parent and physician , and considerable independent decision-making by both the child and family . The asthma educational programs that have achieved successful outcomes have all maintained a close link between health education and the medical treatment of the child's asthma (Feldman , 1987) .

Each of these self-management programs stresses the need for a regular systematic approach to care of patients with asthma including the consistent use of medications as prescribed by the physician , the need to avoid triggers of asthma when these are known , and to initiate early drug therapy when avoidance is not possible (Clark , 1989) .

SUBJECTS

AND

METHODS

SUBJECTS AND METHODS

Before starting the clinical study, the questionnaire was developed meeting the requirements of the program and revised. A pilot test was conducted on 10 families, readjusted, modified and became suitable to be applied in the study. It has a direct, easily understandable questions. A trial was performed to eliminate some variables at the start of program, but we were convinced that all the variables included in the questionnaire has its relative importance for evaluation of the program. Another follow up questionnaire was developed to measure the changes in certain variables that can be changed by the program, revised, tested and implemented. A sample of the initial interview questionnaire and follow up questionnaire are present in the appendix.

The interview period started from March 1992 to December 1993 and conducted in allergy outpatient clinic of Abo El Reish Children Hospital and the families were followed up for one year.

Several trials were performed to apply the program in other health facilities like Imbaba Chest and Allergy clinic, El Giza Chest Hospital, Aum El

Masrien Urban Health Center and Wasat El Giza Primary Schools Health Clinics .

In Imbaba Chest and Allergy Clinic , the manager was met and informed about the program , accepted to conduct the program in the clinic , a permission from the undersecretary of ministry of health in El Giza governorate was obtained . Later on , there was internal troubles in the clinic that lead to change the manager , the manager refused to check the files of the asthmatic children , the place of the interview was not suitable for the conduction of the program and the trial was terminated .

In EL Giza Chest Hospital , a trial was performed to conduct the program in the outpatient clinic , but most of cases were adults and suffering from T.B. or pneumonia , there was no follow up system and there was no enough cases of asthmatic children , so the trial was terminated .

In Aum EL Masrien Urban Health Center , a trial was performed to conduct the program in the outpatient clinic ,the number of asthmatic children was very low (2 asthmatic children in 2 weeks) and the follow up rate was very low , so the trial was terminated .

In Wasat EL Giza Primary Schools , a trial was performed to conduct the program in its health clinics , after permission from the undersecretary of ministry of Education , a list of primary schools was obtained , after several settings with the school physicians , it was clear that the number of children suffering from bronchial asthma was low , and the trial was terminated .

Abo El Reish Children Hospital was the most suitable place for conduction of the program for the following reasons :

- Cooperation of the physicians in the outpatient allergy clinic .
- Suitable number of asthmatic children attending the clinic .
- Presence of case history files and follow up system .
- Availability of investigations .
- Presence of highly qualified specialists .

The original sample consisted of 89 asthmatic children with their parents, aged 5 to 12 years, they were assigned into one of two groups intervention and the comparison group. The basis of assignment depends on random selection categorized according to severity of asthma into

equal sized groups. When first met , the children were confirmed to be suffering from bronchial asthma , the degree of asthma severity was assessed using medical score , and then randomly assigned into intervention and comparison group according to severity . So that the two groups will be of the same grades of asthma severity .

All children were examined by a pediatrician and

proved to be suffering from bronchial asthma after confirmation by several tests including complete medical history, complete clinical examination, chest x ray, complete blood picture, sputum examination, stool analysis and sensitivity tests if required. Children suffering from wheezing due to other causes (eg . inhaled foreign body, chest TB) were excluded from the study . The medical history , severity of asthma and current medications were revised from the child's record in the hospital . The parents were interviewed and informed about the program , their consent was obtained to be included in the study.

Out of the 89 initial children enrolled in the study, 13 were living in distant places (other governorates) and only interviewed for few times . Out of the remaining 76 families, 58 completed the program, 28 of them were of the intervention group and 30 of the comparison group. Possible causes for not completing the follow up period for one year are : living in distant areas, leaving the original places to other new places (after the earthquake in October 1992), improvement in cases, and unavailability of freely administered drugs.

For the intervention group, the educational sessions started

by filling the data of the questionnaire from the caretaker, verifying knowledge of the caretaker and child about causes of asthma , triggers of asthma attacks, signs and symptoms of asthma attacks, medications used for treatment of asthma , what the family do to prevent the attacks,at start of the attack and during the attack and their knowledge about healthy home environments.

The following points were covered :

- Answering questions of the caretaker
- Educating the family that asthma is a chronic disease

characterized by temporary airflow obstruction that leads to difficulty in breathing , swelling of the airways and increased sensitivity of the airways to a variety of triggering factors associated with viscid mucus secretions that cause breathing difficulties.

- Triggers of asthma attacks may differ from one child to other and it is important to identify the triggers of child's asthma .

- Possible asthma triggering factors are : viral respiratory infections , exposure to cold direct air currents (usually after bathing), exposure to dust , exposure to smoke and irritant fumes, inhalation of house dust mites , pollen grains inhalation, exposure to

wastes of cockroaches , animal danders and bird feathers, vigorous physical exercise , exaggerated child emotions and some types of food in few cases.

-The PGM stress on healthy home environments : to avoid triggering factors healthy home environments include:living in areas away from factories that produces air pollution (e.g cement factories) ,avoid smoking inside the house , avoid viral respiratory tract infections, avoid direct air currents specially in winter seasons , avoid irritant fumes , avoid use of aerosols that has irritant components as an insecticides, avoid use of mocket or carpets in the child's room and if used should be cleaned and washed regularly , avoid exaggerated emotions avoid cleaning of the ground with manual sweepers in front of the child, avoid keeping animals or birds in close contact to child , avoid use of child's room as stores or for smoking .

- The PGM encourage the following points : good nutrition and diet containing proteins,fats and carbohydrates, suitable chest exercises as blowing balloons, blowing air bubbles , sport practice as swimming and cycling, regular picnics to clean healthy areas , good family

relations, regular cleaning of mattresses and pillows by washing and ironing , encasing of pillows by nylon cases, regular cleaning of the flat specially kitchen and bathroom to get rid of cockroaches ,rats ,molds ,and fungi.

- Adherence to the drug treatment plan prescribed is of prime importance , according to the condition of each child , correct method of administration ,correct doses ,correct frequency ,and duration of time

- Early recognition of signs and symptoms of asthma : symptoms vary from one child to other , the following are the most common :

Symptoms related to respiratory system: cough , rapid breathing , nasal symptoms (runny nose) , wheezing , sneezing.

General symptoms : pale faces, stopped feeding and drinking well, bluish discoloration around eyes , itching, tearing, pale lips and sense of weakness.

Behavioral symptoms : nervousness, irritability, easily excited , restlessness, sleep interruption due to recurrent cough .

- The PGM instruct the family to adopt the following at start of attacks : following the individualized tailored plan according to severity of asthma and child's condition as regards type of drug,

dose, frequency and for how long. Giving warm fluids to help expelling mucus secretion , calming and resting the child to avoid fears and exacerbation of the attack, and putting the child under observation , after removal of the triggers if identified . Early treatment within minutes with medications ,it is easier to stop an episode in early phase than later.

- Recognition of signs and symptoms of severe attacks which are the following : increase of severity of cough, mouth breathing , interrupted talking, sweating, moving alae nasae during respiration , suprasternal retraction , intercostal retraction , subcostal retraction , bluish extremities or the general condition becomes worse. Presence of one or more of the above signs and symptoms indicates severe attack and the child should be urgently transmitted to the hospital.

Techniques used for teaching families include asking questions , repetition , illustrative drawings , written instructions and role play .

Because a considerable number of families attending the outpatient allergy clinic in Abo El Reish Children Hospital were illiterate , as manifested from the pilot study , there was a need for illustrative drawings to help families understand the triggers of asthma and requirements of healthy home environments . The main ideas were discussed and informed to professionals in the art of drawings working in the Medical Education Technology Center of the Ministry of Health . Several sessions were held on several occasions to settle the appropriate drawings , Examples of these illustrations are present in the appendix .

Slides to be seen by the projector were developed for the program in the Medical Education Technology Center of the Ministry of Health for educating asthmatic children and their families about asthma . Unfortunately , there was no suitable place to demonstrate such slides .

A trial was performed to involve some of the pharmaceutical companies to help in supply of the educational materials , but it was time consuming and the trial was terminated .

- The family received a *written instructions* a sample of which is present in the appendix , *demonstration and drawings* of the possible initiating factors and how to avoid are present in the appendix.

- An individualised tailored plan suitable for each child's condition according to medical history, clinical examination and home environments was instructed to the caretakers.

-Assessment of asthma severity according to medical scores :

The following grades of asthma severity ,quoted from the Expert Panel Report,1991 , for asthma education :

1 - *Characteristics of mild asthma* : exacerbations of cough and wheezing no more often than 1-2 times / week . Few clinical signs or symptoms of asthma between exacerbations. Good exercise tolerance ,but may not tolerate vigorous exercise ,specially prolonged running . Symptoms of nocturnal asthma occur no more often than 1-2 times /month. Good school attendance .

2 - *Characteristics of moderate asthma* :exacerbation of cough and wheeze on a more frequent basis than 1-2 times / week ,could have history of severe exacerbations but infrequent , urgent care treatment in hospital emergency department or physician's office < 3 times/year . Cough and low grade wheezing between acute exacerbations often present , symptoms of nocturnal asthma present 2-3 times/week. School

attendance may be affected.

3 - *Characteristics of severe asthma* : daily wheezing ,exacerbations frequent ,often severe , tendency to have sudden severe exacerbations ,urgent visits to hospital emergency departments or physician's office > 3 times / year. Continuous low-grade cough and wheezing almost always present .Very poor exercise tolerance with marked limitation of activity . Considerable , almost nightly sleep interruption due to asthma , chest tightness in early morning . Poor school attendance .

- Measuring Peak Expiratory Flow Rate : it was recorded for each child in the intervention and the comparison groups , using Mini Flow Meter (low range model for children) range from 50 - 280 Litre / Minute .

The technique adopted was as follows :

- The child was familiarized with the instrument and one or two practice blows were attempted before results were recorded.The child should be standing, the child holds the PEFM, pointer should be at zero position .

- The child is instructed to take deep breath as deep as possible, put his lips tightly around the mouth piece and blow out as hard as as fast as possible in a short sharp blast (the expiration should

not be prolonged ,since the peak expiratory flow occurs within the first tenth of a second of expiration).

- The position of the pointer is recorded according to the scale.

- The test is repeated at least three times ,the highest reading is recorded and considered PEFR . Adequate period of rest between attempts permitted if child has moderate to severe airway obstruction.

- The mouth piece was disinfected using alcohol after each child.

- The height of the child was measured while barefooted with measurement scale , while the child standing upright and his head ,back and soles adjacent to the wall.

- The PEFR was compared to the predicted normal for the child's height according to Godfrey et al,1970 scale .

- *Assessment of severity of asthma according to PEFR* : according to the following :

- * If the value of the PEFR is 100% or more of the predicted normal , then the child is asymptomatic

- * If the value of the PEFR is more than 80% of the predicted normal, then the child is considered as having mild asthma.

- * If the value of the PEFR is between 60 to 80% of the predicted

normal, the child is considered to have moderate asthma.

* If the value of PEFr is less than 60% of the predicted normal , this child is considered to have severe asthma.

Assessment of knowledge and practices of families :

As we have the information regarding knowledge and practices of caretakers , there was a need for assessment of those knowledge and practices appropriate to the study . The system of assessment was adopted as there was no other assessment system mentioned in the other programs suitable for the study . Assessment system will be illustrated in the chapter of the results .

- For the comparison group , the same steps were adopted except that they did not receive the Program's instructions and drawings. They received the routine advice of the attending physician in the clinic.

- At the end of the sessions , a promotional materials as rulers, pencils or blocknotes were given to the children .

Statistical Analysis

Since most of the results were qualitative data, statistical significance was measured using the Chi Square Test (χ^2), the formula used was as follows:

$$\chi^2 = \frac{[(a * b) - (c * d)]^2 * g}{e * f * h * i}$$

Where:

a = exposed and improved

b = exposed and not improved

c = not exposed and improved

d = not exposed and not improved

e = the sum of a + c

f = the sum of b + d

g = the sum of a + b + c + d

h = the sum of c + d

i = the sum of a + b

If the result is less than 3.84, then P value is > 0.05 , the change is not significant

If the result is more than 3.84 ,then P value is < 0.05 , the change is significant .

If the result is more than 5.88 ,then P value is < 0.01 , the change is highly significant.

The results were calculated using IBM compatible computer , with EPI info program version 5 , for statistical calculations . Made by the CDC (Center of Disease Control) , Atlanta , Georgia , USA . In cooperation with WHO (World Health Organization) , Geneva , Switzerland . October , 1990 .

RESULTS

RESULTS AND ANALYSIS

At the beginning of the program and before educational sessions ,we tried to know as much as we can about the environments and living conditions of the asthmatic children and their families ,so that we can assess each child's conditions and focus the instructions to certain areas where the defect is most probably present e.g presence of domestic animals in the flat .So that each child can receive his own tailored instructions ,where there is stress on particular points of special importance to him in addition to the general instructions present in the appendix .

A summary of these informations on the 76 families are present below :

1 - Basic data about children and their families :

mean age of children at first interviews = 7.33 years ,of them 33 = 43.4% females ,and 43 = 56.6% males .61% = 80.3% accompanied by their mothers ,12 = 15.8% by their fathers .1 = 1.3% by his sister ,1 = 1.3% by her aunt ,and 1 = 1.3% by his grandmother .22 child = 28.9% came for the first time to the clinic ,and 54 = 71.1% had recurrnt times .68 = 89.5% living in urban areas ,while 8 = 10.5% living in rural areas .

Concerning the birth order of the child ,25 = 32.9% were the first child ,21 = 27.6% the second child ,11 = 14.5% the third child 7 = 9.2% the fourth ,9 = 11.8% the fifth ,and 3 = 3.9% the sixth child .AS for the number of siblings for the child ,5 = 6.6% have no siblings ,40.8% have two siblings ,9 = 11.8% have three siblings ,14 = 18.4% have four siblings ,4 = 5.3% have five siblings ,and 3 = 3.9% have six siblings .

As for breast feeding early in life ,6 =7.9% did not breast fed ,13 = 17.1% breast fed less than six months ,11 = 14.5% breast fed from 6 to 12 months ,and 45 = 59.2% for more than one year .5 = 6.6% have incomplete vaccination ,and 71 = 93.4% completed their vaccination .

As for seasonal variations of asthma episodes ,44 = 57.9% most commonly during winter ,8 = 10.5% during summer ,23 = 30.3% all round year equally .and 1 = 1.3% could not assess .Regarding diurnal variations of asthma attacks 59 = 77.6% most commonly during night .6 = 7.9% during day ,10 = 13.2% day and night equally ,and 1 = 1.3% could not assess .

2 - Presence of associated conditions :

25 children= 32.9% suffered from nocturnal enuresis : high proportion of nocturnal enuresis may be due to dependent child or due to hypoxia that influence bladder control mechanisms

during night) .25 children = 32.9% suffered from other kinds of allergy ,of them 21 = 84% skin allergy ,and 4 = 16% other kinds of allergy e.g nasal or gastrointestinal allergy .

Positive family history of asthma in 44 = 57.9% ,of them 7 = 15.9% was the father ,6 = 13.6% was the mother ,2 = 4.5% the sibilings ,and 29 = 65.9% other relatives including grandfather ,grandmother ,uncles and aunts .25 children = 32.9% have positive family history of allergy ,of them 7 = 28% have allergic father 5 = 20% allergic mother ,7 = 28% allergic sibling ,and 6 = 24% other relatives .

3 - Feelings of child and families related to asthma :

In between the attack 10 children felt relaxed ,36 = 47.4% worried about the attack ,10 = 13.2% scared from the attack ,1 = 1.3% afraid of dying ,and 18 = 23.7% felt normal .Family relations were good in 59 = 77.6% ,bad in 9 = 11.8% ,1 = 1.3% separate parents ,and 7 = could not assess .As for effect of asthma on family relations 6 = 7.9% mentioned no effect ,12 = 15.8% increase sympathy with child , and in 58 = 76.3% families caused family troubles .

4 - Restrictions of child's life because of asthma :

71 children = 93.4% had restricted activities because of asthma ,and 5 = 6.6% had not .64 were attending school of them 57 = 90.5% prevented from going to school for one day or more because of asthma .29 children= 38.2% do not practice sport because of asthma ,38 = 50% sometimes practice ,8 = 10.5% practice regularly ,and 1 = 1.3% not assessed .53 children = 69.7% were restricted from certain types of food because of asthma .Of them 42 = 79.2% were prevented from eating egg ,33 = 62.2% from eating fish ,25 = 47.2% restricted from milk .19 = 35.8% from banana ,11 = 20.8% from strawberries ,23 = 43.4% from other foods including lentils and chocolates .

5 - Home environments of asthmatic children :

The mean of persons living in the flat = 5.91 persons / flat .Mean of rooms in the flat = 3.16 rooms / flat (including hall) .47 children = 61.8% have smoking persons living in the flat ,of them 39 = 83% have persons smoke inside the flat ,6 children = 12.8% have more than one smoking persons ,38 children = 80.9% have smoking father .1 = 2.1% smoking mother ,3 = 6.4%

smoking siblings ,and 5 = 10.6% have other smoking relatives e.g grand father or uncle .

70 children = 92.1% sleep on mattresses and pillows made of cotton ,6 = 7.9% sleep on mattresses and pillows made of foam .25 children = 32.9% their room's floor is covered by carpets ,31 = 40.8% covered by hasser ,10 = 13.2% covered by terrazzo ,5 = 6.6% by mocket ,4 = 5.3% by cement ,and 1 = 1.3% by kanaltex .72 children = 94.7% their flat's ground is cleaned by manual hand sweeper ,and 4 = 5.3% by electric vaccume cleaner .

The roof of the flat is made of concrete in 73 = 96.1% ,and made of wood in 3 = 3.9% of the families .31 families = 40.8% use kerosine for cooking ,and 45 = 59.2% use butagas .14 families = 18.4% use kerosine for heating during winter ,5 = 6.6% use electric heaters ,3 = 3.9% use crop residue e.g corn stalks or rice stalks ,1 = 1.3% use burning wood ,1 = 1.3% use gas heaters ,and 52 = 68.4% use nothing .32 families = 42.1% mentioned that cockroaches and / or rats are present in the flat .40 families = 53.3% use sprays as an insecticides .10 = 13.2% have plants ,and 27 families have dogs or cats or birds or domestic animals in the house .

Other data will be demonstrated in the next tables :

Table 1 :

Association between age and sex at initial interviews before implementation of the PGM :

| Age in years | Females | | Males | | Total | |
|--------------|---------|---------|--------|---------|--------|---------|
| | Number | Percent | Number | Percent | Number | Percent |
| 5 to < 6 | 9 | 11.8 | 8 | 10.5 | 17 | 22.4 |
| 6 to < 7 | 8 | 10.5 | 8 | 10.5 | 16 | 21.0 |
| 7 to < 8 | 6 | 7.9 | 8 | 10.5 | 14 | 18.0 |
| 8 to < 9 | 1 | 1.3 | 7 | 9.2 | 8 | 10.5 |
| 9 to < 10 | 5 | 6.6 | 2 | 2.6 | 7 | 9.2 |
| 10 to < 11 | 2 | 2.6 | 4 | 5.3 | 6 | 7.8 |
| 11 to < 12 | 2 | 2.6 | 6 | 7.9 | 8 | 10.5 |
| Total | 33 | 43.4 | 43 | 56.6 | 76 | 100 |

As shown from the table ,22.4% of children aged from 5 to < 6 years ,21% from 6 to 7 years ,18% from 7 to < 8 years ,10.5% from 8 to < 9 years ,9.2% from 9 to < 10 years ,7.8% from 10 to < 11 years ,and 10.5% from 11 to < 12 years .Most of children (43.4%) aged from 5 to < 7 years .Of them 22.3% were females and 21% were males .

Table 2 :

Age of onset of first attack of wheezing according to sex , study population at initial interviews :

| Age of onset in years | Females | | Males | | Total | |
|-----------------------|---------|------|-------|------|-------|------|
| | No. | % | No. | No. | No. | % |
| < 1 | 13 | 17.1 | 22 | 28.9 | 35 | 46 |
| 1 to < 2 | 4 | 5.2 | 5 | 6.6 | 9 | 11.8 |
| 2 to < 3 | 4 | 5.2 | 4 | 5.2 | 8 | 10.5 |
| 3 to < 4 | 3 | 3.9 | 3 | 3.9 | 6 | 7.9 |
| 4 to < 5 | 4 | 5.2 | 2 | 2.6 | 6 | 7.9 |
| 5 to < 6 | 2 | 2.6 | 2 | 2.6 | 4 | 5.3 |
| 6 to < 7 | 2 | 2.6 | 1 | 1.3 | 3 | 3.9 |
| 7 to < 8 | 0 | 0 | 0 | 0 | 0 | 0 |
| 8 to < 9 | 1 | 1.3 | 3 | 3.9 | 4 | 5.3 |
| 9 to < 10 | 0 | 0 | 1 | 1.3 | 1 | 1.3 |
| Total | 33 | 43.4 | 43 | 56.6 | 76 | 100 |

As shown from the table ,46% of children had their first attack of wheezing before the age of one year ,11.8% aged from 1 to < 2 years. Most of children (57.8%) had their first attack of wheezing before completion of 2 years .

Socio-economic level of the enrolled families :

Table 3 :

Educational level of the enrolled families :

| Educational level | Father | | Mother | |
|-----------------------|--------|------|--------|------|
| | No. | % | No. | % |
| Illiterate | 22 | 27.6 | 39 | 51.3 |
| Just can read & write | 22 | 28.9 | 10 | 13.2 |
| Primary | 3 | 3.9 | 2 | 2.6 |
| Preparatory | 5 | 6.6 | 7 | 9.2 |
| Secondary | 17 | 22.4 | 15 | 19.7 |
| University | 8 | 10.5 | 3 | 3.9 |
| Total | 76 | 100 | 76 | 100 |

As shown from the table ,27.6% of fathers were illiterate ,and 28.9% just can read and write .51.3% of mothers were illiterate , and 13.2% just can read and write .

Table 4 :

Occupational level of the enrolled families :

| Occupational level | Father | | Mother | |
|----------------------------|--------|------|--------|------|
| | No. | % | No. | % |
| Unemployment or house wife | 6 | 7.9 | 64 | 84.2 |
| Unskilled | 13 | 23.7 | 3 | 3.9 |
| Skilled manual | 38 | 50 | 3 | 3.9 |
| Skilled unmanual | 9 | 11.6 | 3 | 3.9 |
| Managerial | 4 | 5.3 | 3 | 3.9 |
| Professional | 1 | 1.3 | 0 | 0 |
| Total | 76 | 100 | 76 | 100 |

Unskilled e.g servants

Skilled manual e.g carpenters ,mechanics

Skilled unmanual e.g secretar ,clarks

Managerial e.g lawyers

Professional e.g physicians ,engineers ,university staff.

According to Parker and Parker , 1979 .

As shown from the table ,7.9% of fathers were unemployed ,23.7% were unskilled ,and 50% were skilled manual .84.2% of mothers were house wives ,3.9% were unskilled ,and 3.9% were skilled

Table 5 :

Residence of the families :

| Site of living | Number | Percent |
|----------------|--------|---------|
| Urban | 68 | 89.5 |
| Rural | 8 | 10.5 |
| Total | 76 | 100.0 |

As shown from the table , 89.5% of families were living in urban areas ,and 10.5% were living in rural areas .This is represented in the pie diagram figure number 1 .

Table 6 :

Birth order of asthmatic child :

| Birth order | Number | Percent |
|--------------|--------|---------|
| First child | 25 | 32.9 |
| Second child | 21 | 27.6 |
| Third child | 11 | 14.5 |
| Fourth child | 7 | 9.2 |
| Fifth child | 9 | 11.8 |
| Sixth child | 3 | 3.9 |
| Total | 76 | 100.0 |

As shown from the table ,32.9% were the first child ,27.6% were the second child ,this account for 60.5% of all children. The table is represented in figure 2 .

Fig 1 : Site of Residence
Urban = 89.5% Rural = 10.5%

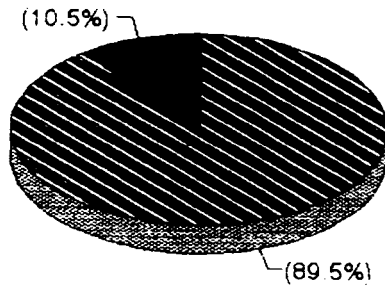


Fig 2 : Birth order of asthmatic child

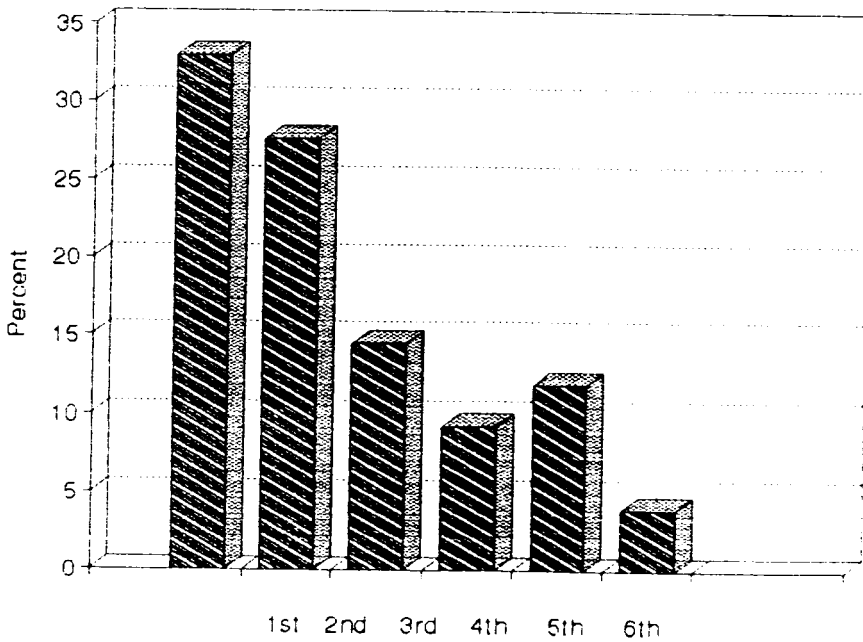


Table 7 :

Period of breast feeding early in life :

| Period in months | Number | Percent |
|---------------------|--------|---------|
| Did not breast fed | 6 | 7.9 |
| Less than 6 months | 13 | 17.1 |
| From 6 to 12 months | 11 | 14.5 |
| More than 12 months | 45 | 59.2 |
| Not assessed | 1 | 1.3 |
| Total | 76 | 100.0 |

As shown from the table ,7.9% were not breast fed ,17.1% breast fed < 6 months ,14.5% from 6 < 12 months ,59.2% > 12 months , and 1.3% were not assessed .The table is represented in figure 3 .

Table 8 :

Seasonal variations of asthma episodes :

| Season | Number | Percent |
|------------------------|--------|---------|
| Winter | 44 | 57.9 |
| Summer | 8 | 10.5 |
| All round year equally | 23 | 30.3 |
| Not assessed | 1 | 1.3 |
| Total | 76 | 100.0 |

As shown from the table ,57.9% of children had their attacks most commonly during winter ,10.5% during summer .30.3% all round year and 1.3% not assessed .The table is represented in figure 4 .

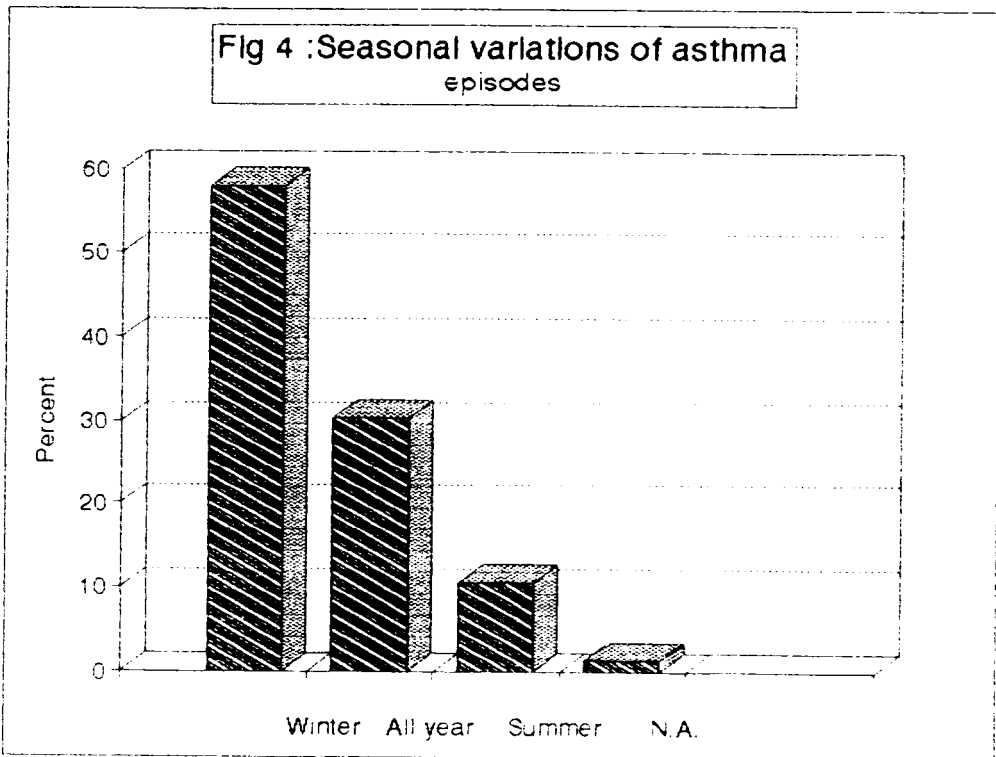
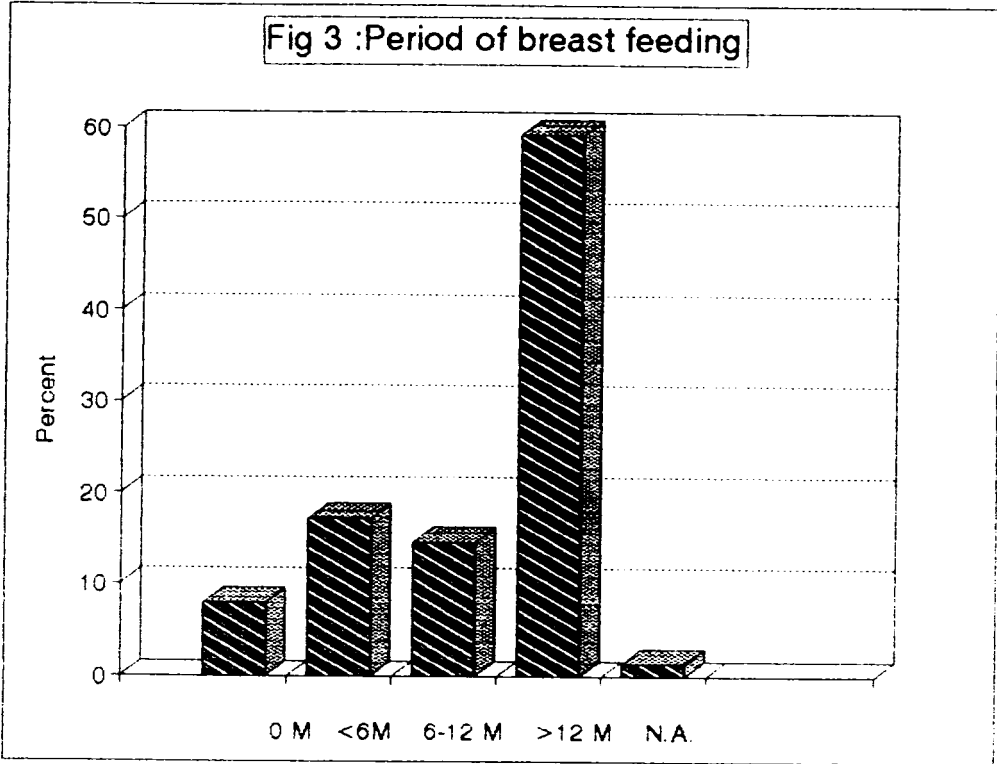


Table 9 :

Diurnal variations of asthma episodes :

| Timing | Number | Percent |
|-----------------------|--------|---------|
| Night | 59 | 77.6 |
| Day | 6 | 7.9 |
| Day and night equally | 10 | 13.2 |
| Not assessed | 1 | 1.3 |
| Total | 76 | 100.0 |

As shown from the table ,77.6% had their attacks most commonly during night ,7.9% during day ,13.2% day and night equally ,and 1.3% was not assessed .The table is represented in figure 5 .

Table 10 :

Presence of nocturnal enuresis :

| Nocturnal enuresis | Number | Percent |
|--------------------|--------|---------|
| Present | 25 | 32.9 |
| Not present | 51 | 67.1 |
| Total | 76 | 100.0 |

As shown from the table ,32.9% suffering from nocturnal enuresis and 67.1% were not .This is represented in figure 6 .

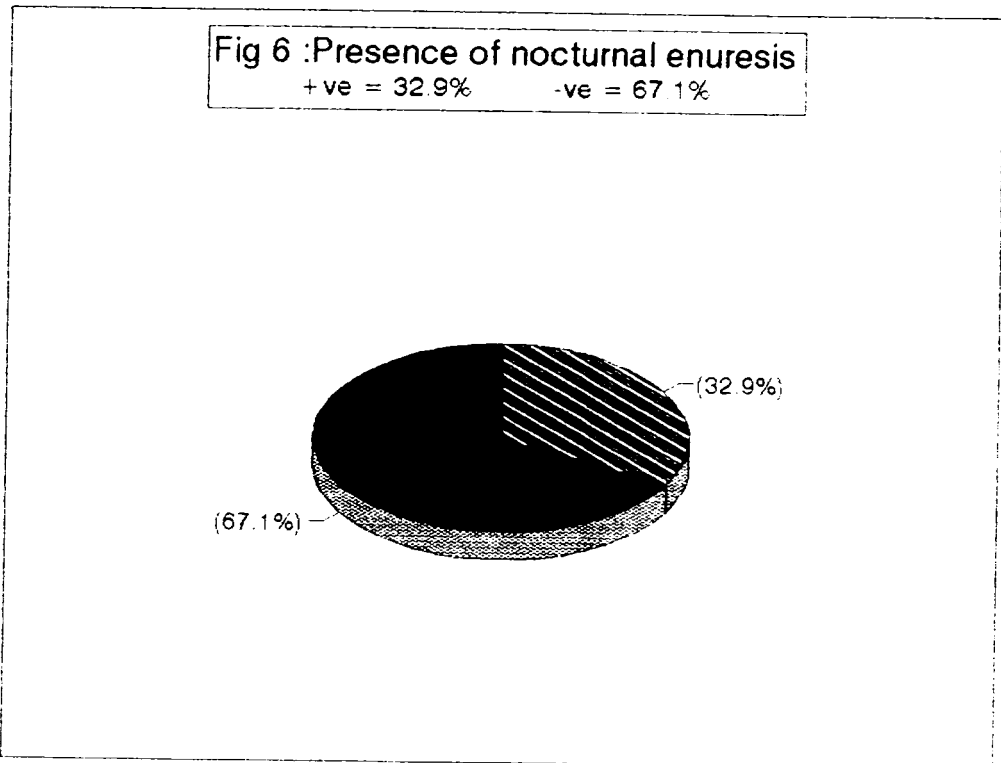
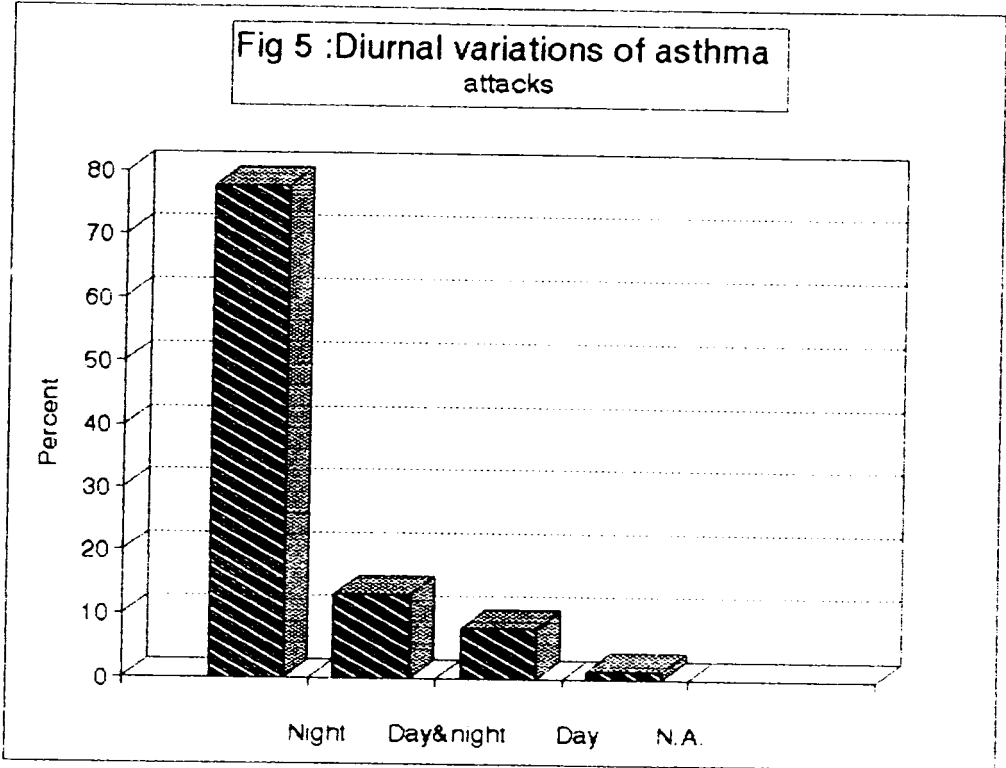


Table 11 :

Presence of other types of allergy in the child :

| Presence of allergy | Number | Percent |
|---------------------|--------|---------|
| Present | 25 | 32.9 |
| Not present | 51 | 67.1 |
| Total | 76 | 100.0 |

As shown from the table ,32.9% have other types of allergy ,and 67.1% have not .The table is represented in figure 7.

Table 12 :

Presence of family history of asthma :

| Family history | Number | Percent |
|----------------|--------|---------|
| Present | 44 | 57.9 |
| Not present | 32 | 42.1 |
| Total | 76 | 100.0 |

As shown from the table ,57.9% of children have family history of asthma ,and 42.1% have not .Figure 8 represent the table .

Fig 7 : Presence of other types of allergy in the asthmatic child

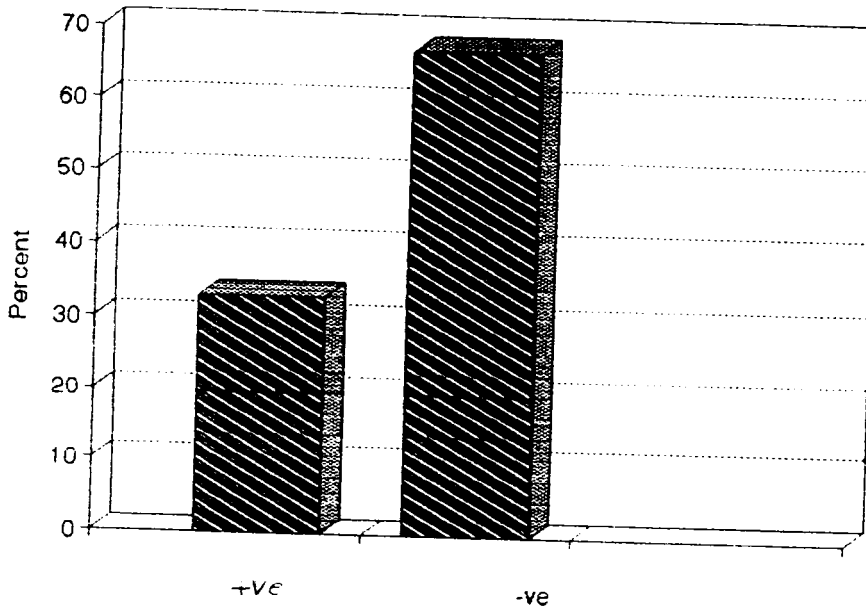


Fig 8 : Family history of asthma in the asthmatic child

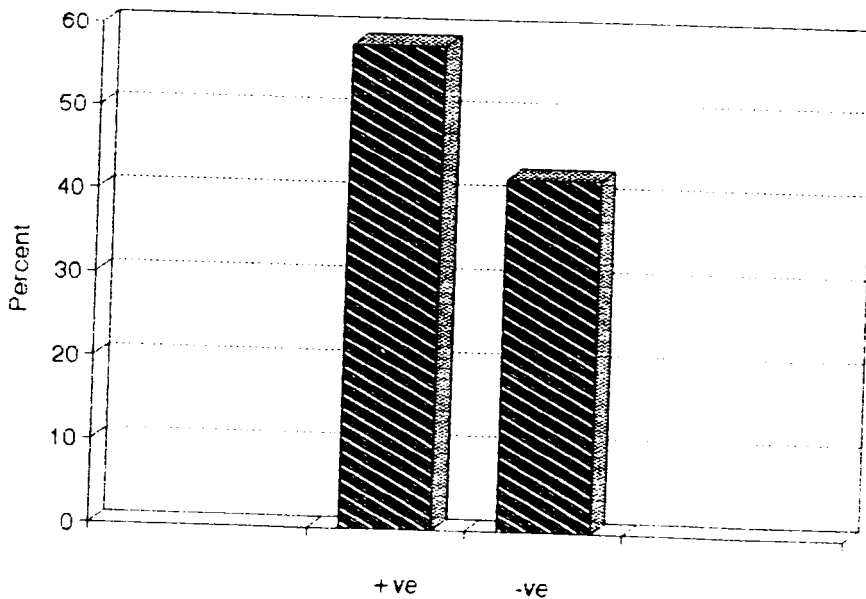


Table 13 :

Presence of family history of allergy :

| Family history | Number | Percent |
|----------------|--------|---------|
| Present | 25 | 32.9 |
| Not present | 51 | 67.1 |
| Total | 76 | 100.0 |

As shown from the table ,32.9% of children have family history of allergy ,and 67.1% have not .Figure 9 represent the table .

Table 14 :

Restriction of certain types of food because of asthma :

| Food restriction | Number | Percent |
|------------------|--------|---------|
| Restricted | 53 | 69.7 |
| Not restricted | 23 | 30.3 |
| Total | 76 | 100.0 |

As shwn from the table .69.7% of children were restricted from certain types of food because of asthma ,and 30.7% were not .The table is represented in figure 10 .

Fig 9 :Family history of allergy in the
in the asthmatic child

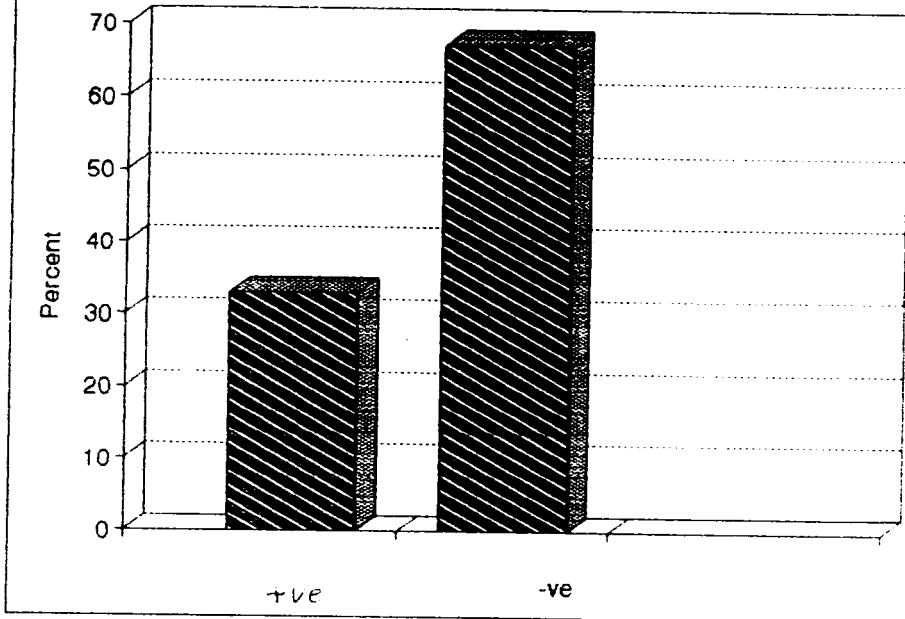


Fig 10 :Food restriction because of
asthma

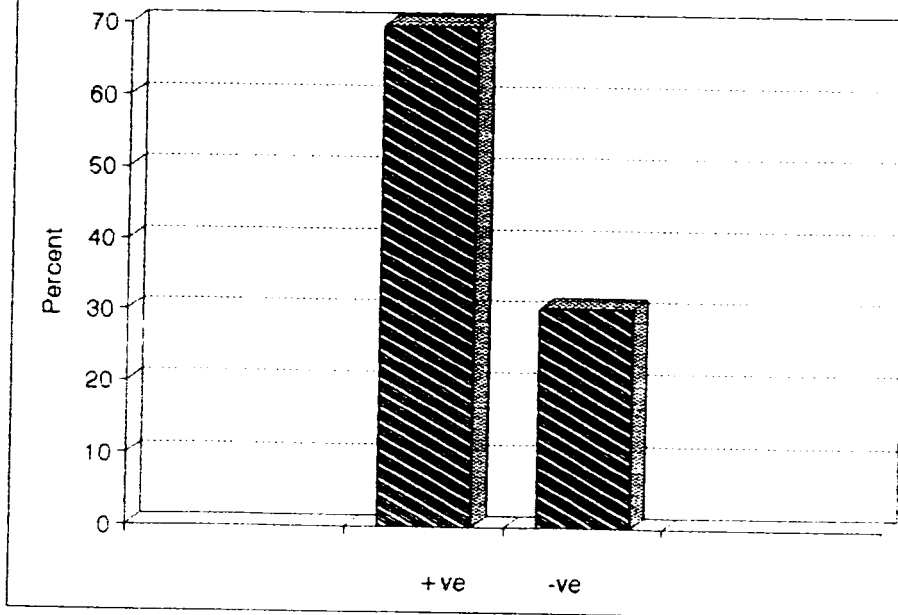


Table 15 :

Types of foods prevented from the child because of asthma :

| Foods prevented | Number | Percent |
|-----------------------|--------|---------|
| Egg | 42 | 79.2 |
| Fish | 33 | 62.2 |
| Milk | 25 | 47.2 |
| Banana | 19 | 35.8 |
| Strawberries | 11 | 20.8 |
| Chocolates or lentils | 23 | 43.4 |

As shown from the table, 79.2% of children were prevented from eating egg, 62.2% from eating fish, 47.2% from drinking milk, 35.8% from eating banana, 20.8% from eating strawberries, and 43.4% from eating chocolates or lentils. See figure 11.

Table 16 :

Child's sport practice :

| Sport practice | Number | Percent |
|----------------|--------|---------|
| Regular | 8 | 10.5 |
| Sometimes | 38 | 50.0 |
| None | 29 | 38.2 |
| Not assessed | 1 | 1.3 |
| Total | 76 | 100.0 |

10.5% of children practice sport regularly, 50% sometimes, 38.2% none, and 1.3% was not assessed. Refer to figure 12.

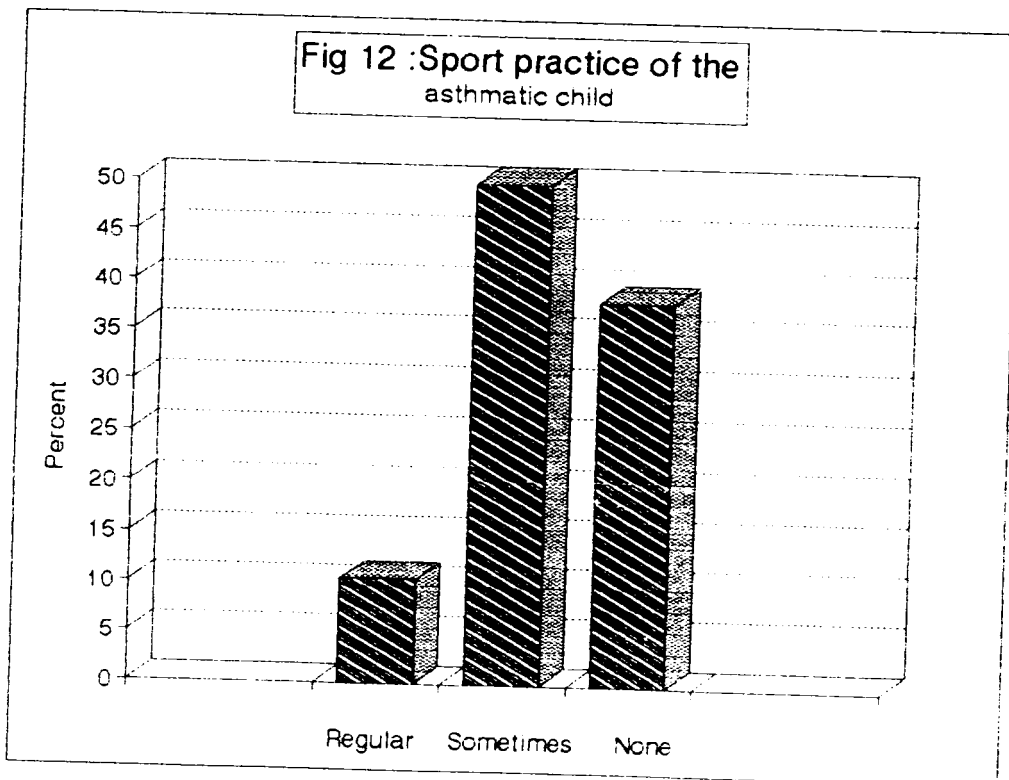
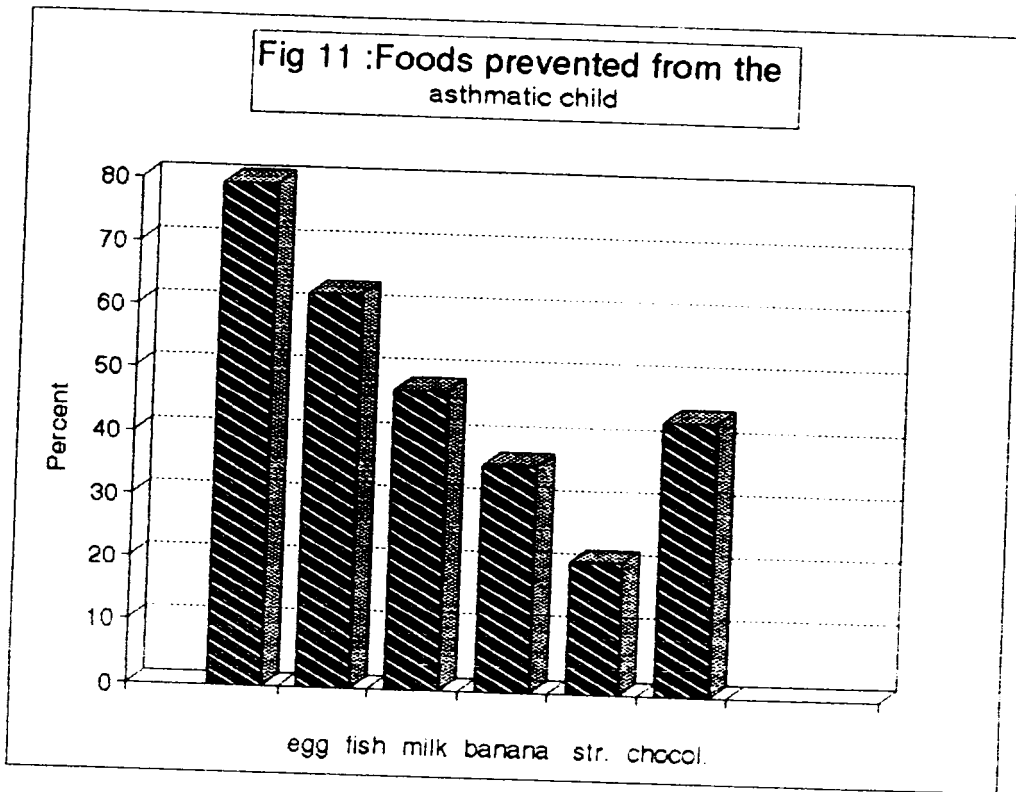


Table 17 :

Presence of smoking persons in the flat :

| Smoking persons | Number | Percent |
|-----------------|--------|---------|
| Present | 47 | 61.8 |
| Not present | 29 | 38.2 |
| Total | 76 | 100.0 |

As shown from the table ,61.8% of children have smoking person living in the flat ,and 38.2% have not .Figure 13 represent the table.

Table 18 :

Relation of smoking persons to asthmatic child :

| Relation | Number | Percent |
|--------------------------|--------|---------|
| Father | 38 | 80.9 |
| Mother | 1 | 2.1 |
| Siblings | 3 | 6.4 |
| Others e.g. grandfathers | 5 | 10.6 |
| Total | 47 | 100.0 |

As shown from the table ,80.9% out of 47 children that have smoking person in the flat ,have smoking father ,2.1% smoking mother ,6.4% smoking siblings ,and 10.6% have other relatives as grandfather or uncle .Figure 14 represent the table .

Fig 13 :Smoking person in the flat
61.8% = +ve 38.2% = -ve

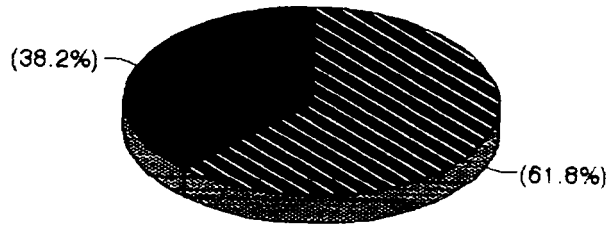


Fig 14 :Relation of smoking persons to asthmatic child

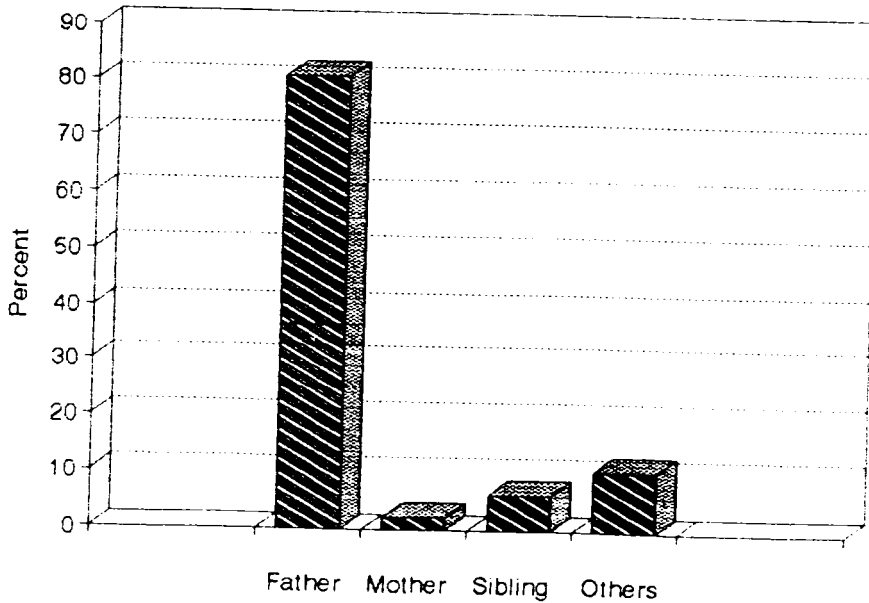


Table 19 :

Whether they smoke inside flat :

| Smoking inside flat | Number | Percent |
|---------------------|--------|---------|
| Yes | 39 | 83 |
| No | 8 | 17 |
| Total | 47 | 100 |

As shown from the table ,83% of smoking persons smoke inside flat ,and 17% smoke outside flat .Figure 15 represent the table .

Table 20 :

Presence of dogs ,cats ,domestic animals or birds in the house :

| Presence of domestics | Number | Percent |
|-----------------------|--------|---------|
| Present | 27 | 35.5 |
| Not present | 49 | 64.5 |
| Total | 76 | 100.0 |

As shown from the table ,35.5% of families have domestic animals or birds in the house ,and 64.5% have not .This is represented in figure 16 .

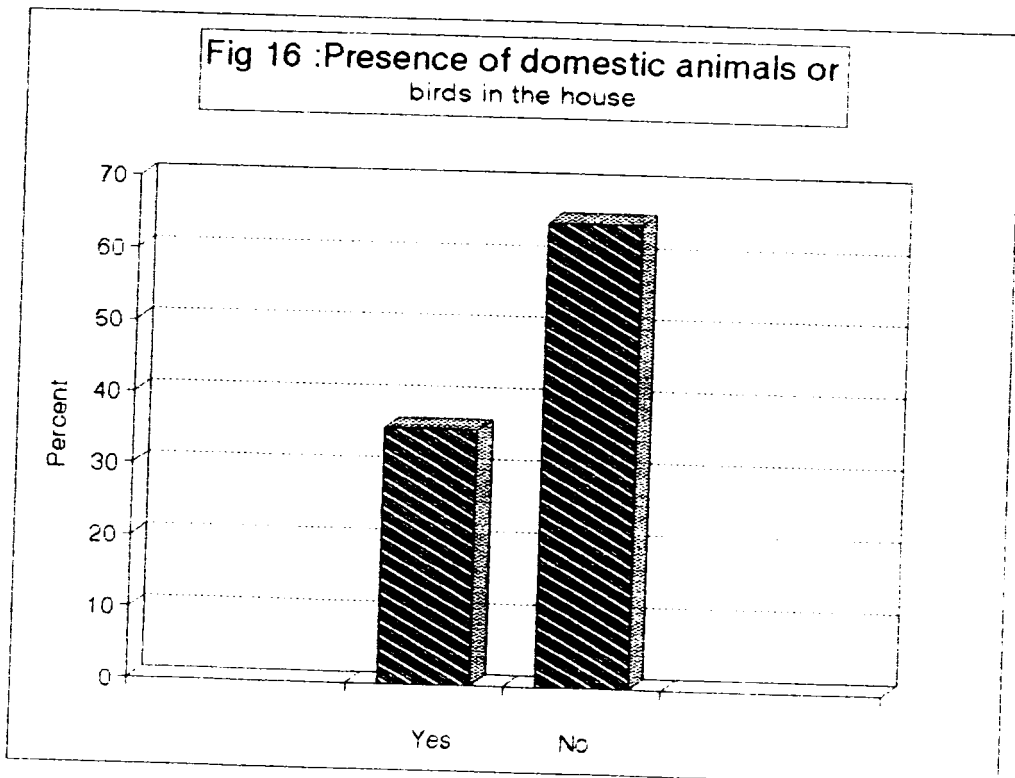
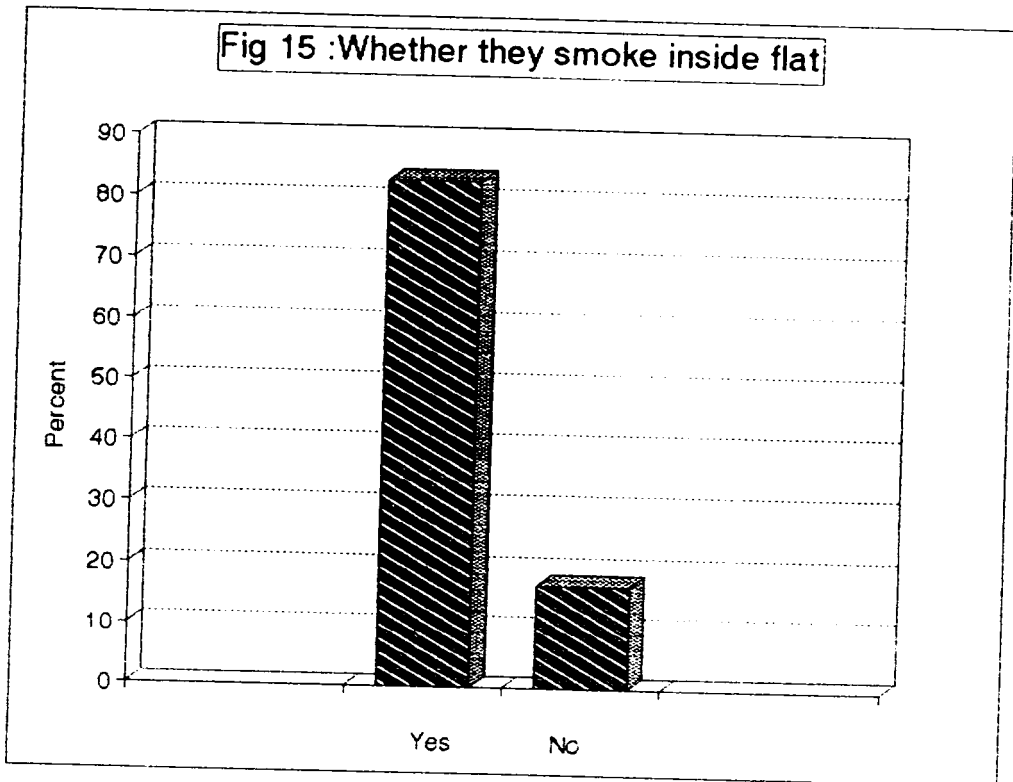


Table 21 :

Assessment of asthma severity by medical score :

| Severity of asthma | Number | Percent |
|--------------------|--------|---------|
| Mild | 16 | 21.1 |
| Moderate | 37 | 48.7 |
| Severe | 23 | 30.2 |
| Total | 76 | 100.0 |

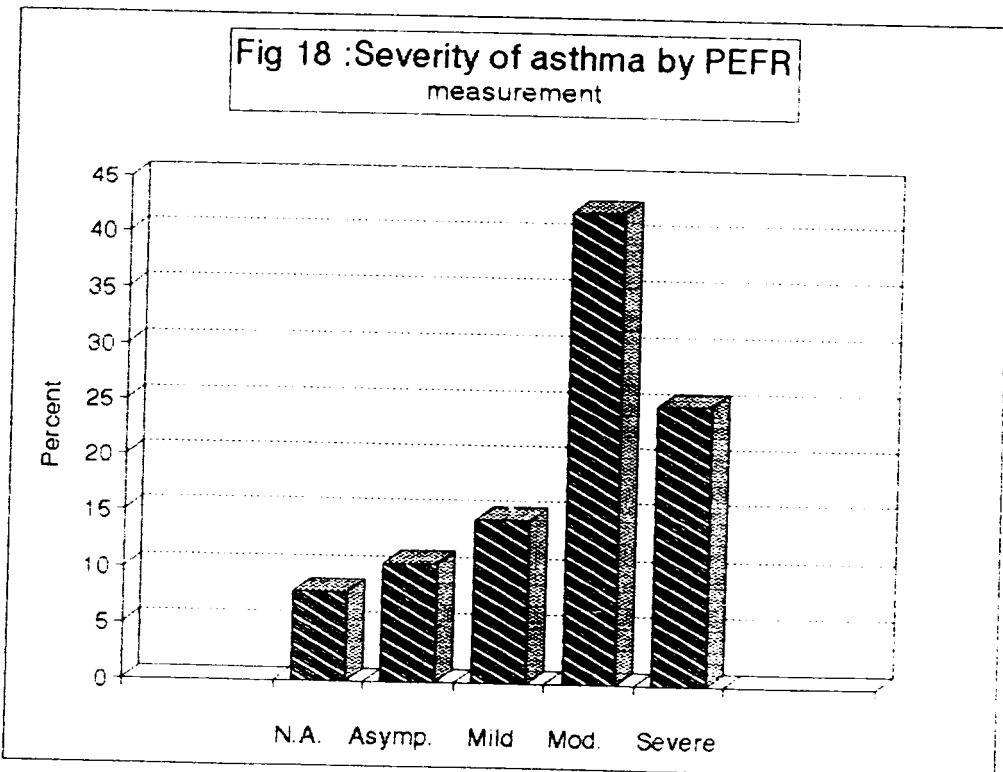
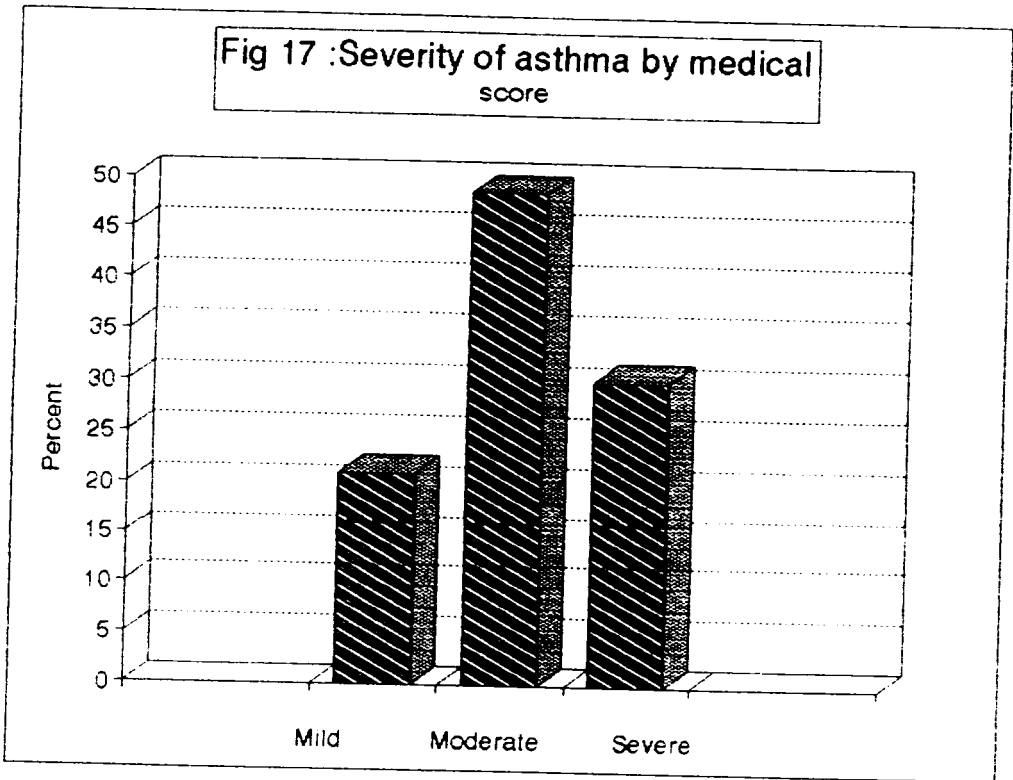
As shown from the table ,21.1% of children were suffering from mild asthma ,48.7% from moderate ,and 30.2% from severe asthma . Figure 17 represent the table .

Table 22 :

Assessment of asthma severity by PEFr measurements :

| Severity of asthma | Number | Percent |
|--------------------|--------|---------|
| Not assessed | 6 | 7.6 |
| Asymptomatic | 8 | 10.5 |
| Mild | 11 | 14.5 |
| Moderate | 32 | 42.1 |
| Severe | 19 | 25.0 |
| Total | 76 | 100.0 |

As shown from the table ,7.6% were not assessed ,10.5% were asymptomatic ,14.5% were mild ,42.1% were moderate ,and 25% were suffering from severe asthma .Figure 18 represent the table .



Knowledge and practice of families toward asthma at initial interviews (76 families) :

Table 23 :

Knowledge of caretakers about triggers of asthma attacks :

| Knowledge | Number | Percentage |
|--|--------|------------|
| Infection (colds) | 8 | 10.5% |
| Exposure to cold (direct air current ,change in temperature) | 29 | 38.1% |
| Physical exercise (physical exertion) | 25 | 32.9% |
| Exposure to smoke | 17 | 22.4% |
| Exposure to dust | 24 | 31.6% |
| Eating certain types of food | 32 | 42.1% |
| Does not know | 24 | 31.6% |

As shown from the table ,31.6% of caretakers mentioned that they do not know asthma triggers ,infection or colds were mentioned by 10.5% ,exposure to cold or direct air currents were mentioned by 38.1% ,physical exertion was mentioned by 32.9% ,exposure to smoke or irritant fumes were mentioned by 22.4%,exposure to dust was mentioned by 31.6% ,eating certain types of food was mentioned by 42.1% .Other causes mentioned by caretakers which are irrelevant were excluded .

Table 24 :

Assesment of knowledge of caretakers about triggers of asthma attacks :

| Assessment | Number | Percentage |
|-------------|--------|------------|
| Complete | 4 | 5.4% |
| Incomplete | 48 | 63.1% |
| Do not know | 24 | 31.6% |
| Total | 76 | 100.0% |

Complete means knowing five correct causes or more

Incomplete means knowing less than five correct causes

Do not know means that caretaker did not mention any correct cause ,or stated that s\he do not know .

As shown from the table ,5.3% knew five correct causes or more , 63.1% knew incomplete knowledge ,and 31.6% did not know any correct cause. These results encourage implementation of the PGM. The table is represented in figure 19 .

Table 25 :

Practice of caretakers to prevent asthma attacks :

| Practice | Number | Percentage |
|--------------|--------|------------|
| Avoid causes | 39 | 51.3% |
| Use of drugs | 31 | 40.0 |
| Do nothing | 18 | 23.7% |

As shown from the table ,51.3% avoid the causes ,40% use drugs , and 23.7% do nothing .

Table 26 :

Assessment of practice of caretakers to prevent asthma attacks :

| Assessment | Number | Percentage |
|------------|--------|------------|
| Complete | 12 | 15.8% |
| Incomplete | 46 | 60.5% |
| Do nothing | 18 | 23.7% |
| Total | 76 | 100.0% |

Complete means avoid causes and use of drugs

Incomplete means avoid causes only or use of drugs only

Do nothing means no action to prevent attacks

As shown from the table ,15.8% were assessed as having complete action , 60.5% were assessed as having incomplete action ,and 23.7% were assessed as having no action to prevent asthma attacks
The table is represented in figure 20.

Fig 19 :Knowledge of triggers of asthma
5.4%=comp. 63.1%=incomp. 31.6%=D't know

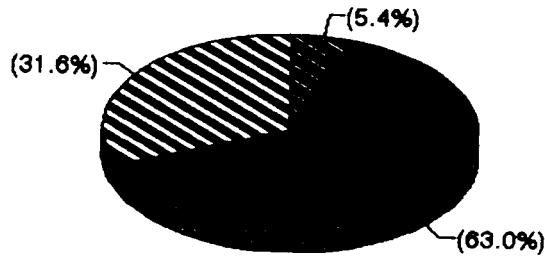


Fig 20 :Practice to prevent asthma attacks

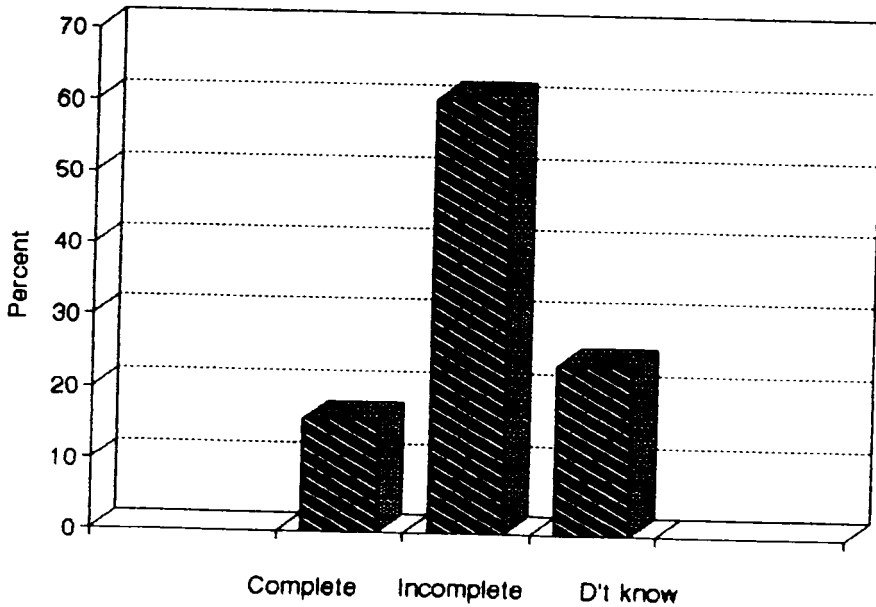


Table 27 :

Knowledge of caretakers about early warning signs of the attacks:

| Knowledge | Number | Percent |
|---------------------|--------|---------|
| Cough | 53 | 69.7% |
| Rapid breathing | 19 | 25.0% |
| Runny nose | 11 | 14.5% |
| restless | 7 | 9.2% |
| wheezing | 13 | 17.1% |
| Difficult breathing | 14 | 18.4% |
| tearing ,pale face | 17 | 22.5% |

As shown from the table ,69.7% mentioned cough ,25% mentioned rapid breathing ,14.5% mentioned runny nose ,9.2% mentioned restless ,17.1% mentioned wheezing ,18.4% mentioned difficult breathing ,and 22.5% mentioned tearing (watery eyes) and pale face .Other signs mentioned by caretakers which were irrilevant were excluded .

Table 28 :

Assessment of knowledge of caretakers about early warning signs of the attacks :

| Assessment | Number | Percent |
|-------------|--------|---------|
| Complete | 10 | 13.2% |
| Incomplete | 51 | 67.1% |
| Do not know | 15 | 19.7% |
| Total | 76 | 100.0% |

Complete means knowing 4 correct signs or more

Incomplete means knowing two or three correct causes

Do not know means knowing one correct cause or less.

As shown from the table ,13.2% were considered having complete knowledge ,67.1% having incomplete knowledge ,and 19.7% did not know .Figure 21 represent the table .

Table 29 :

Practice of caretakers at the start of the attack :

| Practice | Number | Percent |
|-----------------------------|--------|---------|
| Use drugs | 47 | 61.8% |
| Go to hospital or physician | 29 | 38.2% |
| Give warm fluids | 3 | 3.9% |
| Calm child | 3 | 3.9% |
| Keep child in rest | 0 | 0.0% |
| Do nothing | 5 | 6.6% |

As shown from the table ,61.8% use drugs ,38.2% go to hospital or physician directly ,3.9% give warm fluids ,3.9% calm child ,0% keep child in rest ,and 6.6% have no action at start of the attack.

Table 30 :

Assessment of practice of caretakers at start of asthma attacks :

| Assessment | Number | Percent |
|------------|--------|---------|
| Complete | 0 | 0.0% |
| Incomplete | 71 | 93.4% |
| Do nothing | 5 | 6.6% |
| Total | 76 | 100.0% |

Complete means use of drugs as prescribed ,give warm fluids ,keep child calm & relaxed ,and keep child in rest.

Incomplete means missing one of the above practices.

Do nothing means no action.

As shown from the table ,0% have complete action ,93.4% have incomplete action ,and 6.6% have no action. The table is represented in figure 22 .

Fig 21 :Knowledge of early signs
Comp. = 13.2% Incomp. = 67.1% 19.7% = D'tknow

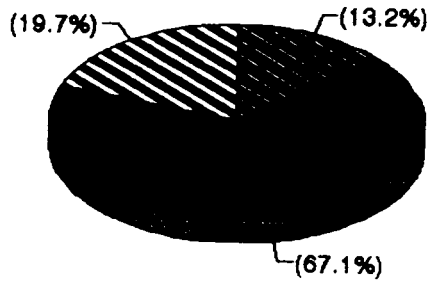


Fig 22 :Practice at start of asthma attack

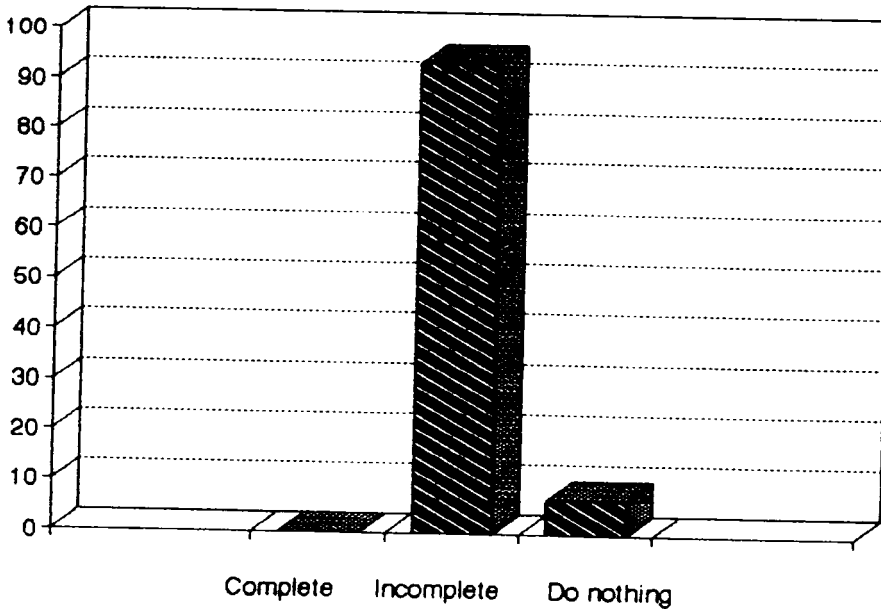


Table 31 :

Knowledge of caretakers about signs during attacks :

| Knowledge | Number | Percent |
|--------------------------|--------|---------|
| Cough | 51 | 67.1% |
| Wheezing | 18 | 23.7% |
| Rapid breathing | 22 | 28.9% |
| Difficult breathing | 37 | 48.7% |
| Grunting | 5 | 6.6% |
| Supra sternal retraction | 1 | 1.3% |
| Inter costal retraction | 0 | 0.0% |
| Sub costal retraction | 1 | 1.3% |
| Headache | 3 | 3.9% |
| Restless | 18 | 23.7% |
| Tired | 25 | 32.9% |
| Bluish exremeties | 13 | 17.1% |
| Do not know | 1 | 1.3% |

As shown from the table ,67.1% mentioned cough ,23.7% mentioned wheezing ,28.9% mentioned rapid breathing ,48.7% mentioned difficult breathing ,6.6% mentioned grunting ,1.3% mentioned indrawing of the lower part of the neck (supra sternal retraction) ,no one mentioned inter costal retraction ,1.3%

mentioned indrawing of lower part of the chest (subcostal retraction) ,3.9% mentioned headache ,23.7% mentioned restlessness ,32.9% mentioned tiredness ,17.1% mentioned bluish extremities ,and 1.3% did not know .

Table 32 :

Assessment of knowledge of caretakers about signs during attacks :

| Knowledge | Number | Percent |
|-------------|--------|---------|
| Complete | 0 | 0.0% |
| Incomplete | 34 | 44.7% |
| Do not know | 42 | 55.3% |
| Total | 76 | 100.0% |

Complete means mentioning seven correct symptoms or more.

Incomplete means mentioning from four to six correct symptoms.

Do not know means mentioning three correct symptoms or less.

As shown from the table ,no one have complete knowledge ,44.7% have incomplete knowledge ,and 55.3% were considered as do not know . The table is represented in figure 23 .

Table 33 :

Practice of caretakers during asthma attacks :

| Practice | Number | Percent |
|-------------------|--------|---------|
| Use drugs | 18 | 23.7% |
| Go to physician | 32 | 42.1% |
| Go to hospital | 35 | 46.1% |
| Irrelevant action | 3 | 3.9% |

As shown from the table ,23.7% use drugs ,42.1% go to physician , 46.0% go to hospital ,3.9% do irrilevant action .

Table 34 :

Assessment of practice of caretakers during asthma attacks :

| Assessment | Number | Percent |
|-------------------|--------|---------|
| Complete | 12 | 15.8% |
| Incomplete | 61 | 80.3% |
| Do useless action | 3 | 3.9% |
| Total | 76 | 100.0% |

Complete means use prescribed drugs and go to physician or hospital directly .

Incomplete means either use drugs only or go to physician &

hospital without using drugs .

Do useless action means doing irrelevant action .

As shown from the table ,15.8% have complete action ,80.3% have incomplete action ,and 3.9% have useless or irrelevant actions during asthma attacks .Figure 24 demonstrate the table .

Knowledge and practice of families toward asthma categorized into comparison and intervention group ,before and after program :

Table 35 :

Knowledge of caretakers about triggers of asthma attacks :

| Knowledge | Comparison group | | | | Intervention group | | | |
|-----------------------|------------------|-----|-------|----|--------------------|----|-------|----|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Infection (colds) | 2 | 6.6 | 4 | 13 | 2 | 7 | 13 | 46 |
| Expoure to cold | 14 | 46 | 19 | 63 | 11 | 39 | 26 | 93 |
| Physical exertion | 15 | 50 | 19 | 63 | 9 | 32 | 24 | 88 |
| Exposure to smoke | 8 | 27 | 11 | 37 | 2 | 7 | 18 | 64 |
| Exposure to dust | 10 | 33 | 15 | 50 | 4 | 14 | 24 | 87 |
| Certain types of food | 12 | 40 | 10 | 33 | 10 | 35 | 11 | 39 |
| Do not know | 7 | 23 | 5 | 16 | 13 | 46 | 0 | 0 |

Comparison group composed of 30 families ,were followed for one year receiving regular physician care ,intervention group

Fig 23: Knowledge of signs during attack
Comp. =0% Incomp. =44.7% D't know=55.3%

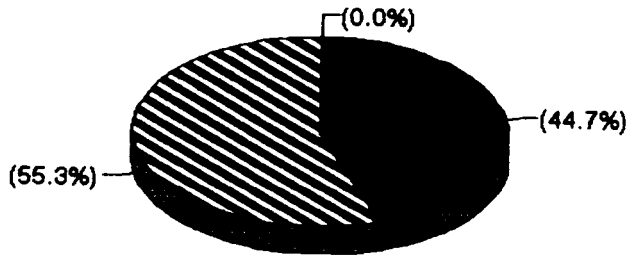
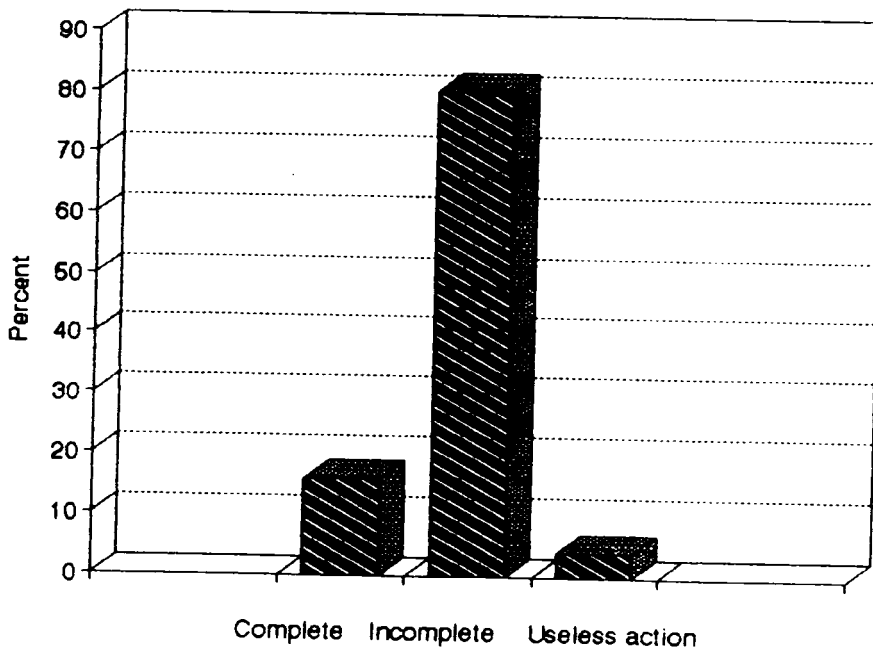


Fig 24 :Practice during asthma attack



composed of 28 families ,were exposed to PGM activities and followed for one year .As shown from the table ,knowledge of colds as triggers of asthma attacks increased in the comparison group from 6.6% at start of follow up to 13% at the end of follow up while in the intervention group increased from 7% before PGM to 46% after PGM .The same for physical exertion as a trigger increased in the comparison group from 50% to 63% while in the intervention group increased from 32% to 88% .Exposure to smoke as a trigger increased from 27% to 37% in the comparison group while in the intervention group increased from 7% to 64% ,exposure to dust as a trigger increased from 33% to 50% in the comparison group while in the intervention group increased from 14% to 87% , certain types of food as a triggers for attacks decreased from 40% to 33% in the comparison group ,while increased from 35% to 39% in the intervention group , caretakers who do not know decreased in the comparison group from 23% to 16% while in the intervention group decreased from 46% to 0% .

Table 36 :

Assessment of knowledge of caretakers about triggers of asthma attacks ,comparison group before and after follow up :

| Assessment | Comparison group | | | | Chi Squ. | P value | Sig |
|-------------|------------------|------|-------|------|----------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 2 | 6.6 | 4 | 13.3 | 0.74 | .389 | NS |
| Incomplete | 21 | 70 | 21 | 70 | 0.0 | 1.0 | NS |
| Do not know | 7 | 23.3 | 5 | 16.6 | 0.42 | 0.518 | NS |
| Total | 30 | 100 | 30 | 100 | | | |

Complete means knowing five correct causes or more

Incomplete means knowing less than five correct causes

Do not know means that caretakers did not know any correct cause or stated that s\he does not know .

As shown from the table ,there was no significant change in knowledge of triggers of asthma attacks in the comparison group before and after follow up .Figure 25 demonstrate the table .

Using Chi Square :

$P < 0.05$ = Significant (S) , $P < 0.001$ = Highly Significant (HS) ,

$P > 0.05$ = Non Significant (NS) .

Table 37 :

Assessment of Knowledge of caretakers about triggers of asthma attacks ,intervention group before and after PGM :

| Assessment | Intervention group | | | | Chi Squ | P value | Sig. |
|-------------|--------------------|------|-------|------|---------|---------|------|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 1 | 3.6 | 13 | 46.4 | 13.7 | .0002 | HS |
| Incomplete | 14 | 50 | 15 | 53.6 | 0.07 | 0.78 | NS |
| Do not know | 13 | 46.4 | 0 | 0 | 16.93 | 0.003 | HS |
| Total | 28 | 100 | 28 | 100 | | | |

As shown from the table ,there was statistically significant improvement of knowledge of caretakers about triggeres of asthma attacks before and after implementation of the PGM .Figure 26 demonstrate the table .

Using Chi Square (X²) =

$P < 0.05$ = Significant (S) , $P < 0.001$ = Highly Signficant (HS) ,and $P > 0.05$ = Non Significant (NS) .

Fig 25 :Knowledge of triggers
(comparison group)

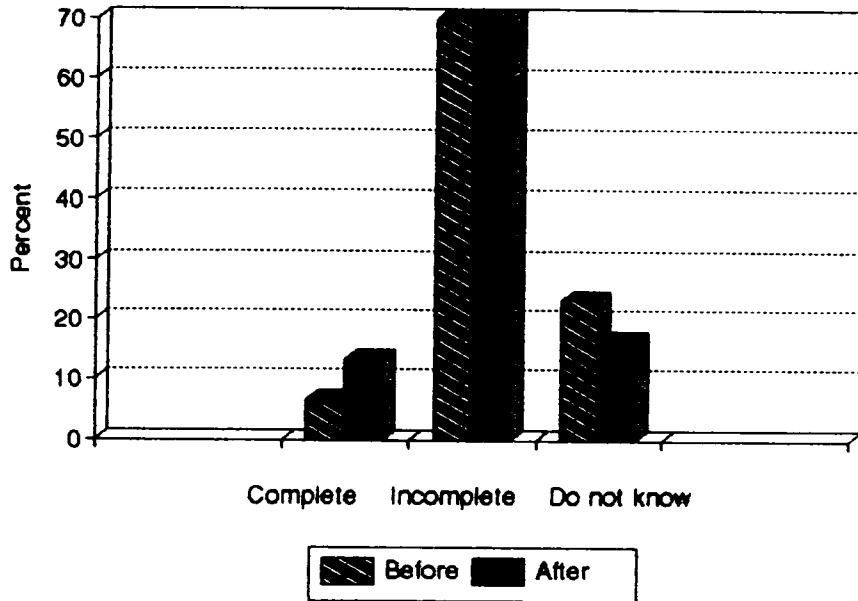


Fig 26 :Knowledge of triggers
(Intervention group)

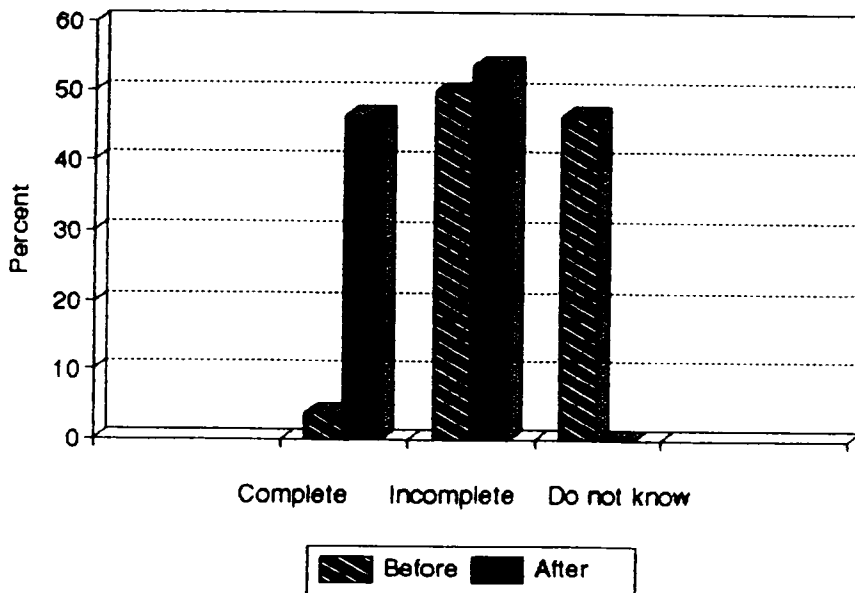


Table 38 :

Practice of caretakers to prevent asthma attacks :

| Practice | Comparison group | | | | Intervention group | | | |
|--------------|------------------|----|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Avoid causes | 18 | 60 | 19 | 63.3 | 9 | 32.1 | 28 | 100 |
| Use drugs | 12 | 40 | 13 | 43.3 | 8 | 28.5 | 13 | 46.4 |
| Do nothing | 6 | 20 | 5 | 16.6 | 11 | 39.2 | 0 | 0 |

As shown from the table ,there was improvement of practice of caretakers to prevent asthma attacks ,the improvement is noticeable in the intervention group than in the comparison group regarding avoiding the causes ,use of drugs .Caretakers that had no active participation for prevention of attacks decreased from 20% to 16.6% in the comparison group while decreased in the intervention group from 39.2% to 0% .

Table 39 :

Assessment of practice of caretakers to prevent asthma attacks ,
comparison group before and after follow up :

| Assessment | Comparison group | | | | Chi Squ | P value | Sig |
|------------|------------------|-----|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 6 | 20 | 7 | 23.3 | 0.1 | 0.75 | NS |
| Incomplete | 18 | 60 | 18 | 60 | 0.0 | 1 | NS |
| Do nothing | 6 | 20 | 5 | 16.7 | 0.11 | 0.73 | NS |
| Total | 30 | 100 | 30 | 100 | | | |

Complete means avoidance the causes and use of prescribed drugs

Incomplete means avoidance the cause only or use of drugs only

Do nothing means that there is no active participation for prevention of attacks .

As shown from the table ,there was no statistically significant improvement in practice of caretakers to prevent asthma attacks in the comparison group before and after follow up .Figure 27 demonstrate the table .

Table 40 :

Assessment of practice of caretakers to prevent asthma attacks , intervention group ,before and after PGM :

| Assessment | Intervention group | | | | Chi Squ | P value | Sig |
|------------|--------------------|------|-------|------|---------|---------|-----|
| | Befor | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 2 | 7.1 | 13 | 46.4 | 11.0 | 0.009 | HS |
| Incomplete | 15 | 53.6 | 15 | 53.6 | 0.0 | 1 | NS |
| Do nothing | 11 | 39.3 | 0 | 0 | 13.7 | 0.002 | HS |
| Total | 28 | 100 | 28 | 100 | | | |

Complete means avoidance the causes and use of drugs

Incomplete means avoidance the causes only or use of drugs only

Do nothing means that there was no active participation to prevent asthma attacks .

As shown from the table , there was a statistically significant improvement in practice of caretakers for prevention of attacks in the intervention group before and after implementation of the PGM. Figure 28 demonstrate the table .

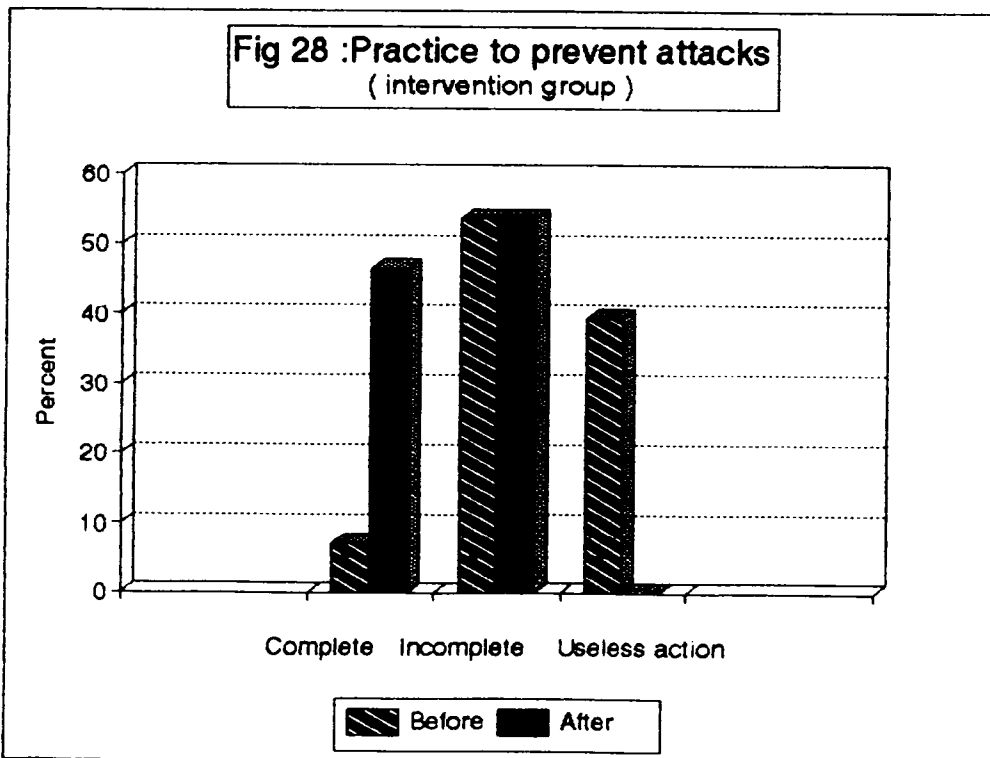
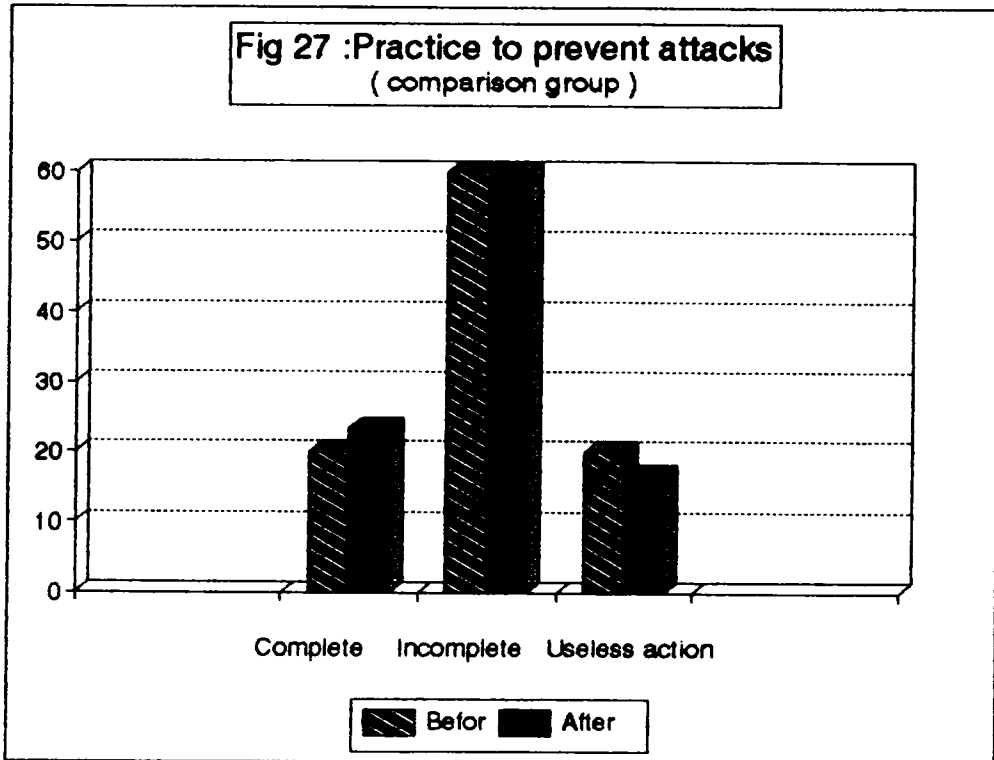


Table 41 :

Knowledge of caretakers about early warning signs of asthma attacks :

| Knowledge | Comparison group | | | | Intervention group | | | |
|-------------------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| cough | 18 | 60 | 20 | 66.6 | 21 | 75 | 27 | 96.4 |
| rapid breathing | 6 | 20 | 11 | 36.6 | 7 | 25 | 27 | 96.4 |
| runny nose | 4 | 13.3 | 9 | 30 | 3 | 10.7 | 8 | 28.5 |
| wheezing | 5 | 16.6 | 11 | 36.6 | 4 | 14.2 | 22 | 78.5 |
| tearing | 6 | 20 | 9 | 30 | 8 | 28.5 | 13 | 46.4 |
| tired | 0 | 0 | 2 | 6.6 | 0 | 0 | 8 | 28.5 |
| change in face colour | 0 | 0 | 3 | 10 | 0 | 0 | 9 | 32.1 |
| dark circles under eyes | 0 | 0 | 3 | 10 | 0 | 0 | 7 | 25 |

As shown from the table ,there was improvement in the knowledge of caretakers regarding early warning signs of asthma attacks in both groups before and after PGM .

Table 42 :

Assessment of knowledge of caretakers about early warning signs of asthma attacks ,comparison group,before and after follow up :

| Assessment | Comparison group | | | | Chi Squ | P Value | Sig |
|-------------|------------------|------|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 4 | 13.3 | 5 | 16.7 | 0.13 | 0.717 | NS |
| Incomplete | 17 | 56.7 | 22 | 73.3 | 1.83 | 0.175 | NS |
| Do not know | 9 | 30 | 3 | 10 | 3.75 | 0.052 | S |
| Total | 30 | 100 | 30 | 100 | | | |

Complete means knowing four signs or more

Incomplete means knowing from two to three signs

Do not know means knowing one sign or that the caretaker stated that s\he does not know .

As shown from the table ,there is improvement in knowledge specially in category who did not know before follow up , this improvemnt reach a statistically significant change ,other categories did not reach significant improvement .Figure 29 demonstrate the table .

Table 43 :

Assessment of knowledge of caretakes about early warning signs of asthma attacks ,intervention group ,before and after PGM :

| Assessment | Intervention group | | | | Chi Squ | p value | Sig |
|-------------|--------------------|------|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 4 | 14.3 | 13 | 46.4 | 6.84 | 0.008 | S |
| Incomplete | 16 | 57.1 | 15 | 53.6 | 0.07 | 0.788 | NS |
| Do not know | 8 | 28.6 | 0 | 0 | 9.33 | 0.002 | HS |
| Total | 28 | 100 | 28 | 100 | | | |

Complete means knowing four signs or more

Incomplete means knowing from two to three signs

Do not know means knowing one sign or that the caretaker states that s\he does not know

As shown from the table ,there is statistically significant improvement in knowledge of caretakers in the intervention group in the category who knew four signs or more ,and highly significant in the category who did not know . Figure 30 demonstrate the table .

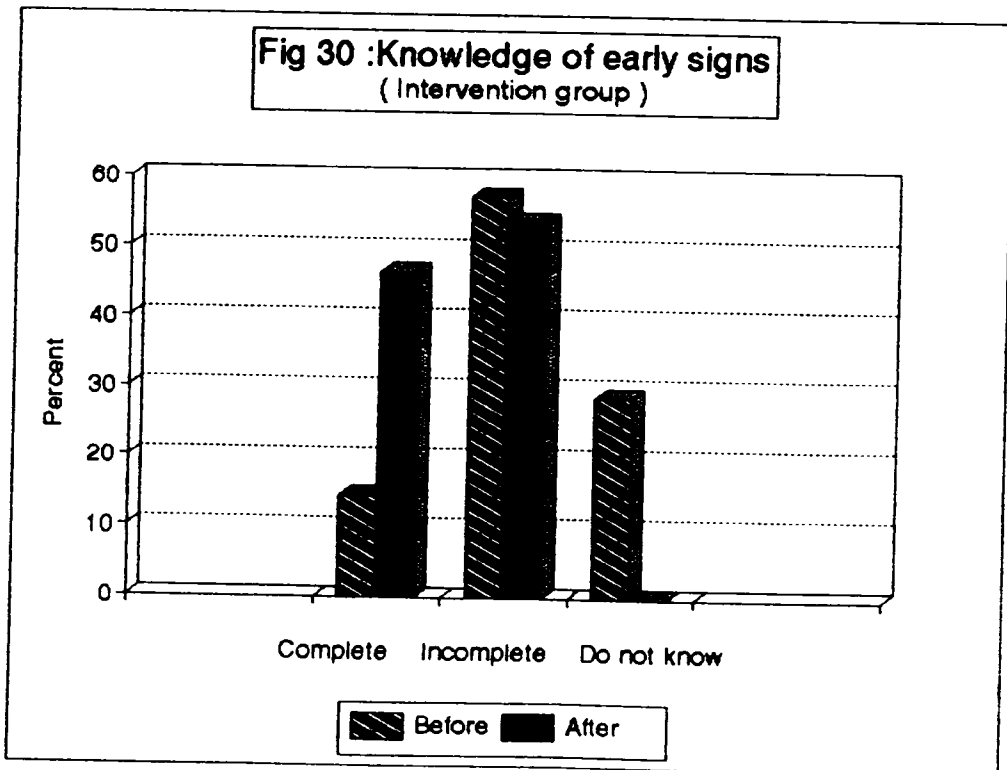
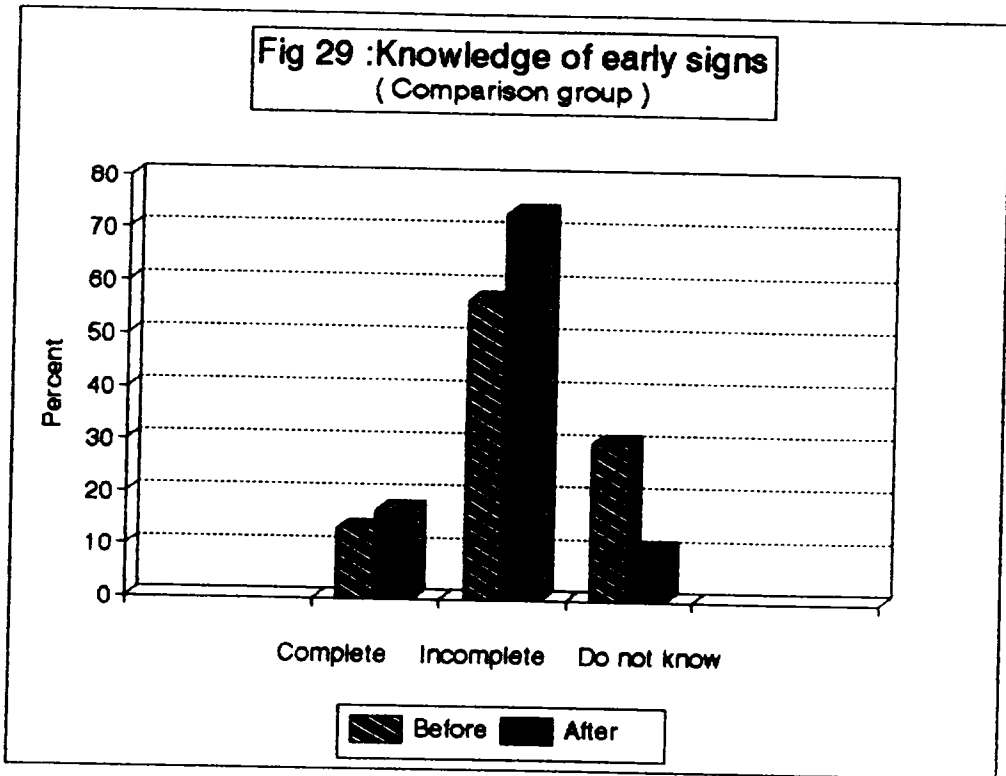


Table 44 :

Practice of caretakers at start of asthma attacks ,categorized into comparison and intervention group ,before and after PGM :

| Practice | Comparison group | | | | Intervention group | | | |
|------------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Use drugs | 19 | 63.3 | 20 | 66.6 | 17 | 60.7 | 26 | 92.8 |
| Go to hospital | 13 | 43.3 | 12 | 40 | 9 | 32.1 | 5 | 17.8 |
| Give warm fluids | 1 | 3.3 | 1 | 3.3 | 2 | 7 | 19 | 67.8 |
| Keep calm &relax | 0 | 0 | 0 | 0 | 3 | 10.7 | 17 | 60.7 |
| Keep in rest | 0 | 0 | 1 | 3.3 | 0 | 0 | 13 | 46.4 |

As shown from the table ,there is improvement in practice of caretakers ,this improvement is remarkable in the intervention than the comparison group as reagard giving warm fluids ,keep child calm and relaxed and keep resting the child .

Table 45 :

Assessment of practice of caretakers at start of asthma attacks , comparison group before and after follow up :

| Assessment | Comparison group | | | | Chi Squ | P value | Sig |
|------------|------------------|-----|-------|-----|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 0 | 0 | 0 | 0 | | | |
| Incomplete | 27 | 90 | 30 | 100 | 3.16 | 0.075 | NS |
| Do nothing | 3 | 10 | 0 | 0 | 3.16 | 0.075 | NS |
| Total | 30 | 100 | 30 | 100 | | | |

Complete means use of drugs as prescribed ,giving warm fluids , keep calm & relaxed and keep child in rest and under observation

Incomplete means missing one of the above points

Do nothing means no active action

As shown from the table ,there was no significant change in the comparison group before and after follow up .Figure 31 demonstrate the table .

Table 46 :

Assessment of practice of caretakers at start of asthma attacks ,
intervention group ,before and after PGM :

| Assessment | Intervention group | | | | Chi Squ | P value | Sig |
|------------|--------------------|------|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 0 | 0 | 11 | 39.3 | 13.69 | 0.002 | HS |
| Incomplete | 26 | 92.8 | 17 | 60.7 | 8.11 | 0.004 | HS |
| Do nothing | 2 | 7.2 | 0 | 0 | 2.07 | 0.149 | NS |
| Total | 28 | 100 | 28 | 100 | | | |

Complete means use drugs as prescribed ,giving warm fluids ,keep child calm & relaxed and keep child in rest under observation

Incomplete means missing one of the above points

Do nothing means no active action

As shown from the table ,there was statistically significant improvement in practice of caretakers at start of asthma attacks after compared to before implementation of the PGM .Figure 32 demonstrate the table .

Fig 31 :Practice at start of attacks
(Comparison group)

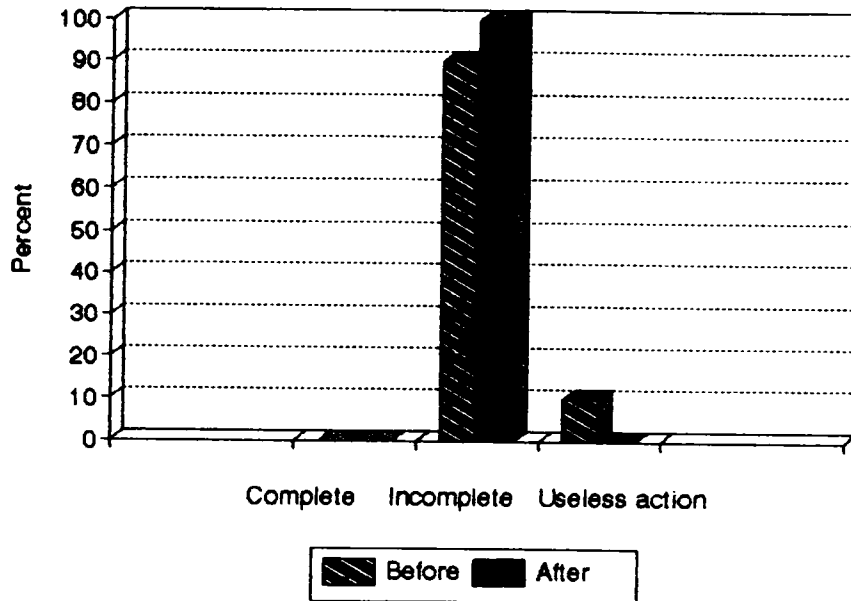


Fig 32 :Practice at start of attacks
(Intervention group)

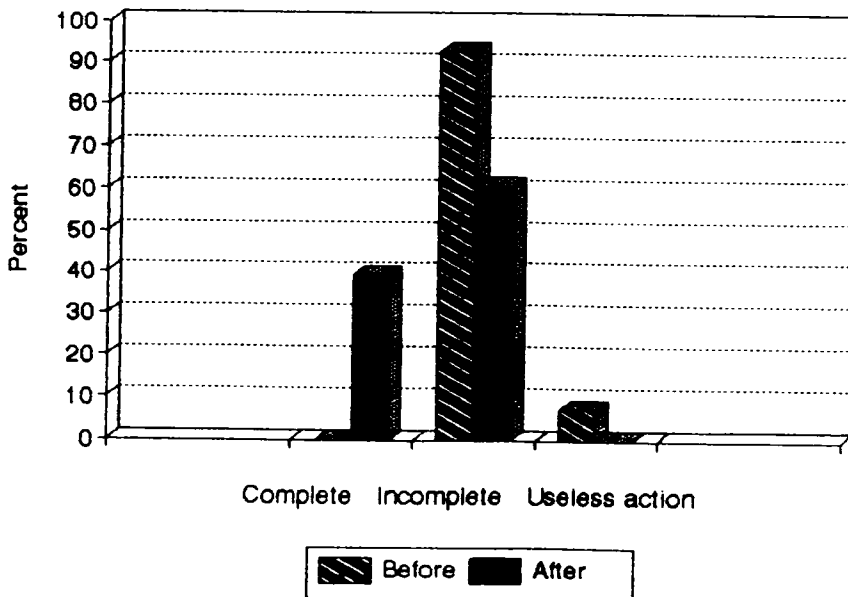


Table 47 :

Knowledge of caretakers about signs of asthma attacks :

| Knowledge | Comparison group | | | | Intervention group | | | |
|--------------------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No | % | No. | % | No. | % | No. | % |
| Cough | 19 | 63.3 | 20 | 66.6 | 19 | 67.0 | 27 | 96.4 |
| Wheezing | 9 | 30 | 16 | 53.3 | 5 | 17.8 | 27 | 96.4 |
| rapid breathing | 8 | 26.6 | 15 | 50 | 8 | 28.5 | 26 | 92.8 |
| difficult breathing | 13 | 43.3 | 17 | 56.6 | 11 | 39.2 | 26 | 92.8 |
| grunting | 1 | 3.3 | 4 | 13.3 | 2 | 6.6 | 17 | 60.7 |
| Suprasternal retraction | 0 | 0 | 3 | 10 | 1 | 3.5 | 13 | 46.4 |
| intercostal retraction | 0 | 0 | 3 | 10 | 0 | 0 | 12 | 42.8 |
| subcostal retraction | 0 | 0 | 3 | 10 | 0 | 0 | 7 | 25 |
| headache | 3 | 10 | 10 | 33.3 | 0 | 0 | 13 | 46.6 |
| restless | 9 | 30 | 12 | 40 | 7 | 28.5 | 17 | 60.7 |
| weakness | 9 | 30 | 10 | 33 | 8 | 28.5 | 11 | 39.2 |
| grey or blue extremities | 4 | 13 | 6 | 20 | 5 | 17.8 | 15 | 53.5 |

As shown from the table ,there was improvement in knowledge for both group before and after implementation of the PGM .

Table 48 :

Assessment of knowledge of caretakers about signs of asthma during attacks ,comparison group ,before and after PGM :

| Assessment | Comparison group | | | | Chi Squ | P value | Sig |
|-------------|------------------|------|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 0 | 0 | 7 | 23.3 | 7.92 | 0.004 | S |
| Incomplete | 13 | 43.3 | 19 | 63.3 | 2.41 | 0.12 | NS |
| Do not know | 17 | 56.7 | 4 | 13.4 | 12.38 | 0.004 | S |
| Total | 30 | 100 | 30 | 100 | | | |

Complete means knowing seven signs or more

Incomplete means knowing from four to six signs

Do not know means knowing three signs or less

As shown from the table ,there was significant improvement in the complete and do not know groups . Figure 33 demonstrate the table .

Table 49 :

Assessment of knowledge of caretakers about signs of asthma during attacks ,intervention group before and after PGM :

| Assessment | Intervention group | | | | Chi Squ | P value | Sig |
|-------------|--------------------|------|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 0 | 0 | 18 | 64.3 | 26.53 | 0.000 | HS |
| Incomplete | 10 | 35.7 | 9 | 32.1 | 0.08 | 0.77 | NS |
| Do not know | 18 | 64.3 | 1 | 3.6 | 23.02 | 0.000 | HS |
| Total | 28 | 100 | 28 | 100 | | | |

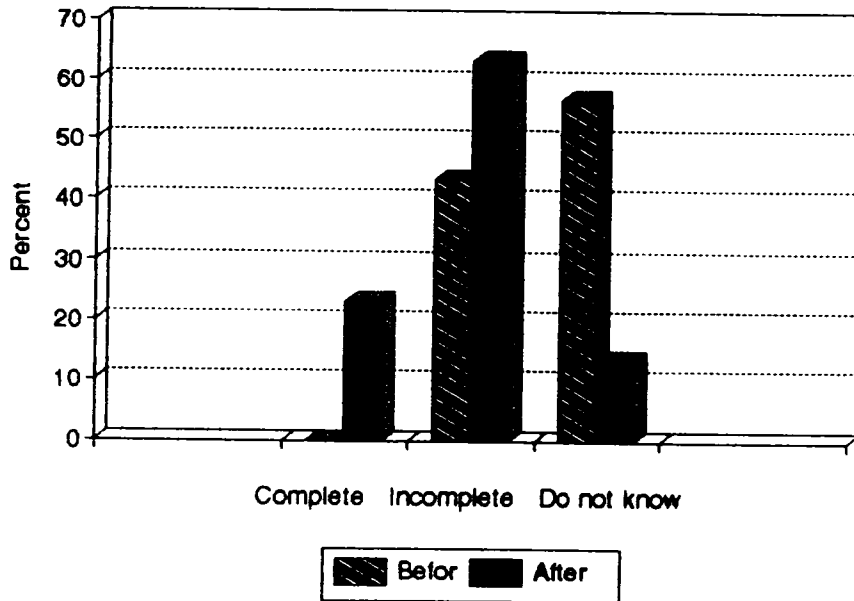
Complete means knowing seven signs or more

Incomplete means knowing from four to six signs

Do not know means knowing three signs or less .

As shown from the table ,there was highly significant improvement in the categories of complete and do not know ,this improvement is more than in the comparison group. Figure 34 demonstrate the table .

**Fig 33 :Knowledge of signs of attacks
(Comparison group)**



**Fig 34 :Knowledge of signs of attacks
(Intervention group)**

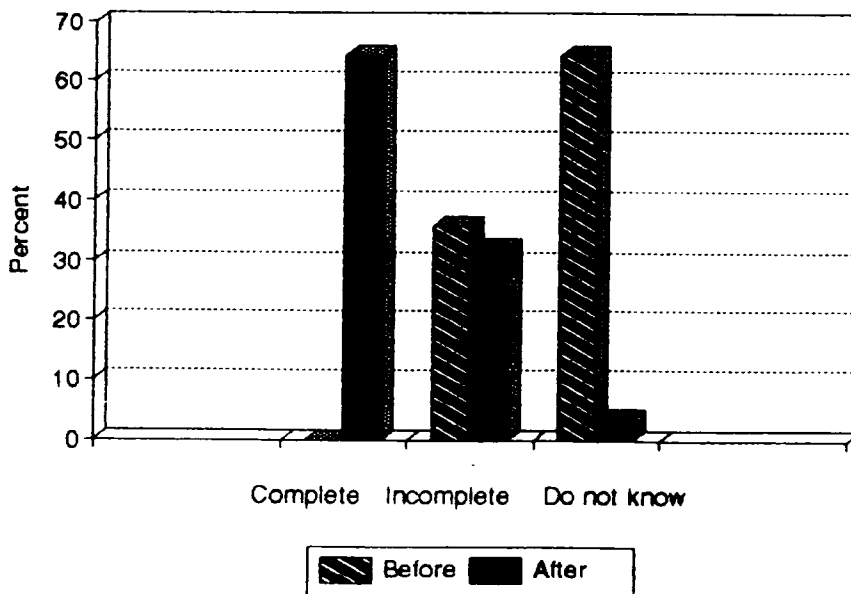


Table 50 :

Practice of caretakers during asthma attacks :

| Practice | Comparison group | | | | Intervention group | | | |
|-----------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Use drugs | 10 | 33.3 | 15 | 50 | 4 | 14.2 | 20 | 71.4 |
| Go to physician | 12 | 40 | 7 | 23.3 | 12 | 42.8 | 8 | 28.5 |
| Go to hospital | 13 | 43.3 | 13 | 43.3 | 12 | 42.8 | 20 | 71.4 |

As shown from the table ,there is decrease in the caretakers who go to private physicians during the attacks in both groups ,and increase dependence on hospitals .

Table 51 :

Assessment of practice of caretakers during asthma attacks , comparison group before and after follow up :

| Assessment | Comparison group | | | | Chi Squ | P value | Sig |
|------------|------------------|------|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 7 | 23.3 | 5 | 16.7 | 0.42 | 0.518 | NS |
| Incomplete | 21 | 70 | 25 | 83.3 | 1.49 | 0.22 | NS |
| Do nothing | 2 | 6.7 | 0 | 0 | 2.07 | 0.15 | NS |
| Total | 30 | 100 | 30 | 100 | | | |

Complete means use of prescribed drugs and go to hospital or

physician directly

Incomplete means use prescribed drugs only or go to hospital or physician only

Do nothing means no action or doing useless action

As shown from the table ,there was no statistically significant change in practice before and after follow up .Figure 35 demonstrate the table .

Table 52 :

Assessment of practice of caretakers during asthma attacks , intervention group before and after PGM :

| Assessment | Intervention group | | | | Chi Squ | P value | Sig |
|------------|--------------------|------|-------|------|---------|---------|-----|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Complete | 1 | 3.6 | 20 | 71.4 | 27.5 | 0.000 | HS |
| Incomplete | 27 | 96.4 | 8 | 28.6 | 27.5 | 0.000 | HS |
| Do nothing | 0 | 0 | 0 | 0 | 0 | 0 | |
| Total | 28 | 100 | 28 | 100 | | | |

Complete means use prescribed drugs and go to hospital or physician directly .

Incomplete means use of drugs only or going to hospital or physician only .

Do nothing means no action or do useless actions

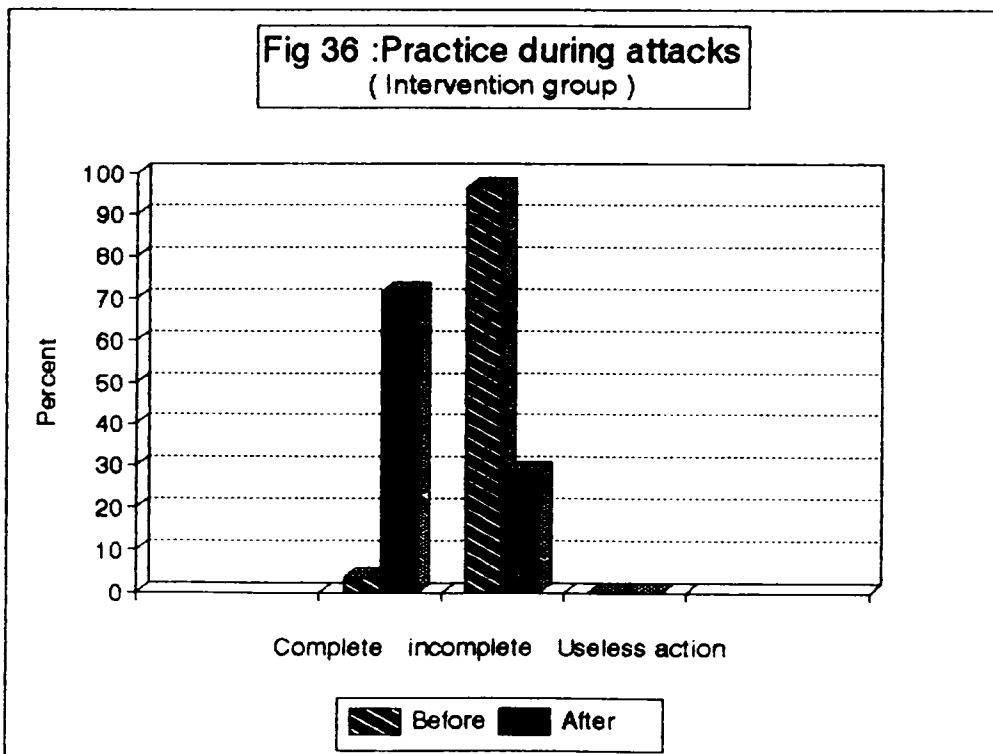
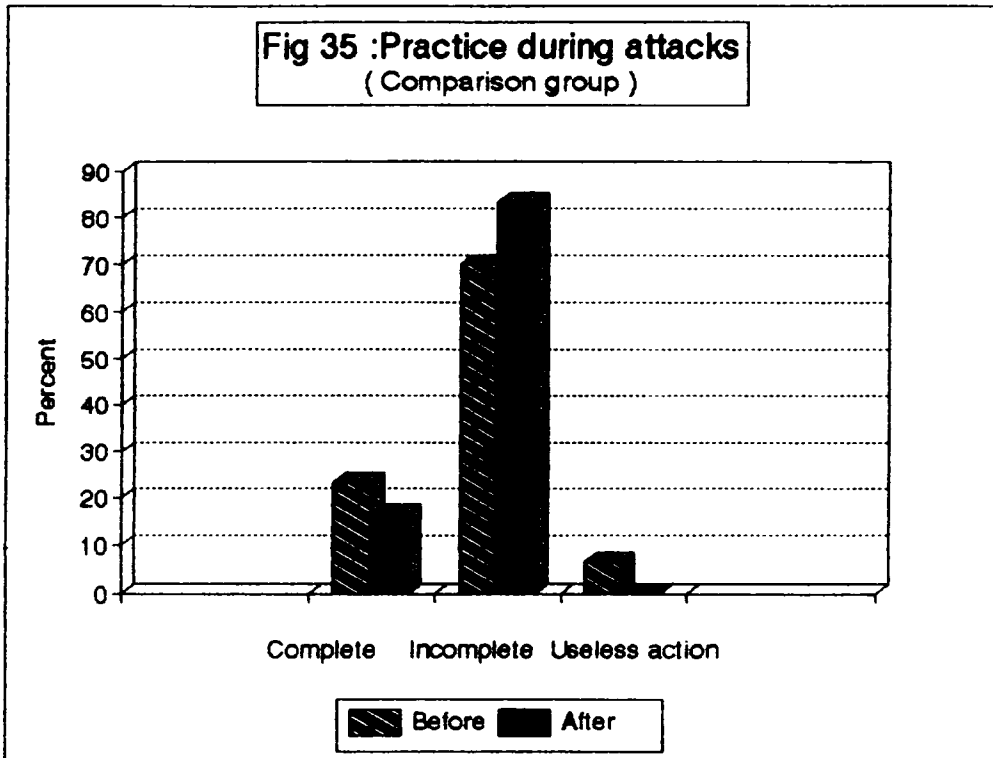
As shown from the table ,there was statistically significant improvement in practice of caretakers in the intervention group , before and after PGM .Figure 36 demonstrate the table .

Table 53 :

Feelings of caretakers about usefulness of drug treatment :

| Feelings of caretakers | Comparison group | | | | Intervention group | | | |
|-----------------------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Useful | 17 | 56.6 | 22 | 73.4 | 15 | 53.3 | 25 | 89.2 |
| Useless or sometimes useful | 13 | 43.4 | 8 | 26.6 | 13 | 46.5 | 3 | 10.8 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,perception of caretakers about usefulness of drug treatment was improved in the comparison group but statistically insignificant $X^2 = 1.83$, $P = 0.17$ (> 0.05) , in the intervention group the improvement is statistically significant $X^2 = 8.75$, $P = 0.03$ (< 0.05).



Restrictions of child's life because of asthma :Table 54 :

Restriction of child's activity because of asthma (not going outside flat for some days because of asthma) last year :

| Activity | Comparison group | | | | Intervention group | | | |
|----------------|------------------|-----|-------|------|--------------------|------|-------|-----|
| | Before | | After | | Befor | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Restricted | 30 | 100 | 17 | 56.6 | 27 | 96.4 | 7 | 25 |
| Not restricted | 0 | 0 | 13 | 43.4 | 1 | 3.6 | 21 | 75 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,there was increase in activity after PGM in both group ,in the comparison group the activity increased from 0% to 43.4% while in the intervention group increased from 3.6% to 75% .

Table 55 :

Prevention of child from going to school for one day or more because of asthma last year :

| School absence | Comparison group | | | | Intervention group | | | |
|----------------|------------------|-----|-------|------|--------------------|-----|-------|------|
| | Before | | After | | Befor | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Absent | 24 | 100 | 12 | 46.2 | 18 | 100 | 5 | 22.7 |
| Not absent | 0 | 0 | 14 | 53.8 | 0 | 0 | 17 | 77.3 |
| Total | 24 | 100 | 26 | 100 | 18 | 100 | 22 | 100 |

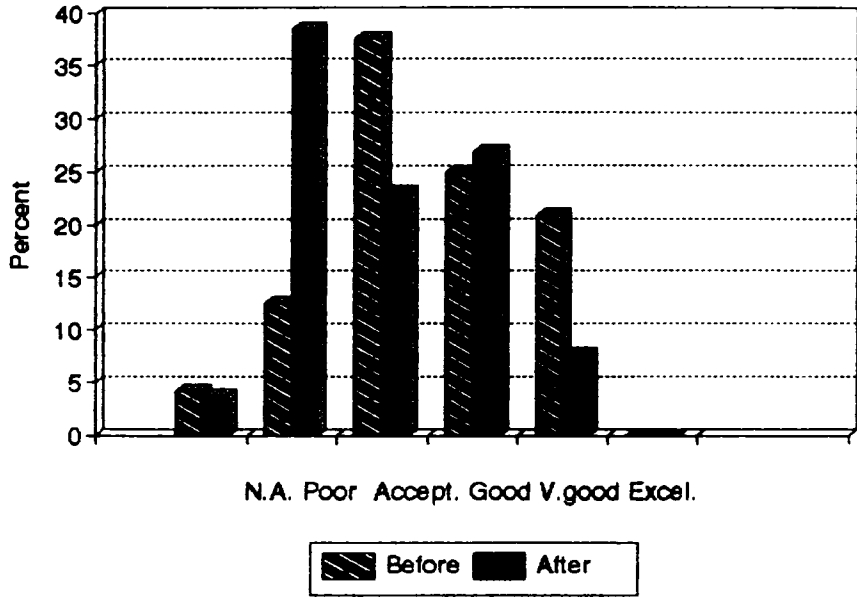
As shown from the table ,school absence decreased in both groups in the comparison group ,100% of the twenty four children attending school missed one day or more at the start of the follow up ,at the end of follow up 46.2% of the twenty six children attending school missed one day or more .In the intervention group ,100% of the eighteen children attending school missed one day or more at the start of the PGM ,at the end of the PGM 22.7% of the twenty two children attending school missed one day or more.

Table 56 :**Child school performance :**

| Performance | Comparison group | | | | Intervention group | | | |
|--------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Not assessed | 1 | 4.2 | 1 | 3.8 | 3 | 16.7 | 0 | 0 |
| Poor | 3 | 12.5 | 10 | 38.5 | 3 | 16.7 | 3 | 13.6 |
| Accepted | 9 | 37.5 | 6 | 23.1 | 6 | 33.3 | 6 | 27.3 |
| Good | 6 | 25 | 7 | 26.9 | 3 | 16.7 | 11 | 50 |
| Very good | 5 | 20.8 | 2 | 7.7 | 2 | 11.1 | 0 | 0 |
| Excellent | 0 | 0 | 0 | 0 | 1 | 5.5 | 2 | 9.1 |
| Total | 24 | 100 | 26 | 100 | 18 | 100 | 22 | 100 |

As shown from the table ,there is no improvement in child school performance in the comparison group on the contrary poor school performance increased from 3 (12.5%) to 10 (38.5%) which is statistically significant $X^2 = 4.41$, $P = 0.035$ (< 0.05) .In the intervention group poor school performance decreased from 16.7% to 13.6% ,and good school performance increased from 3 (16.7%) to 11 (50%) which is statistically significant $X^2 = 4.87$, $P = 0.02$ (< 0.05) .Figure 37 demonstrate the comparison ,and figure 38 demonstrate the intervention group .

**Fig 37 :Child school performance
(Comparison group)**



**Fig 38 :Child school performance
(Intervention group)**



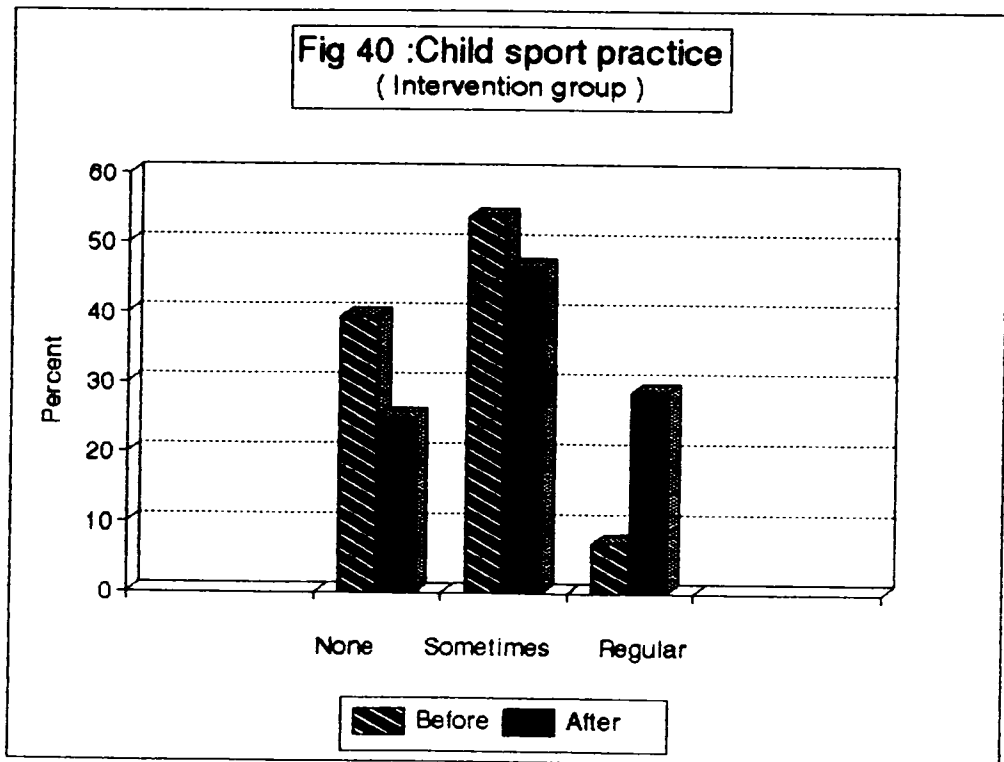
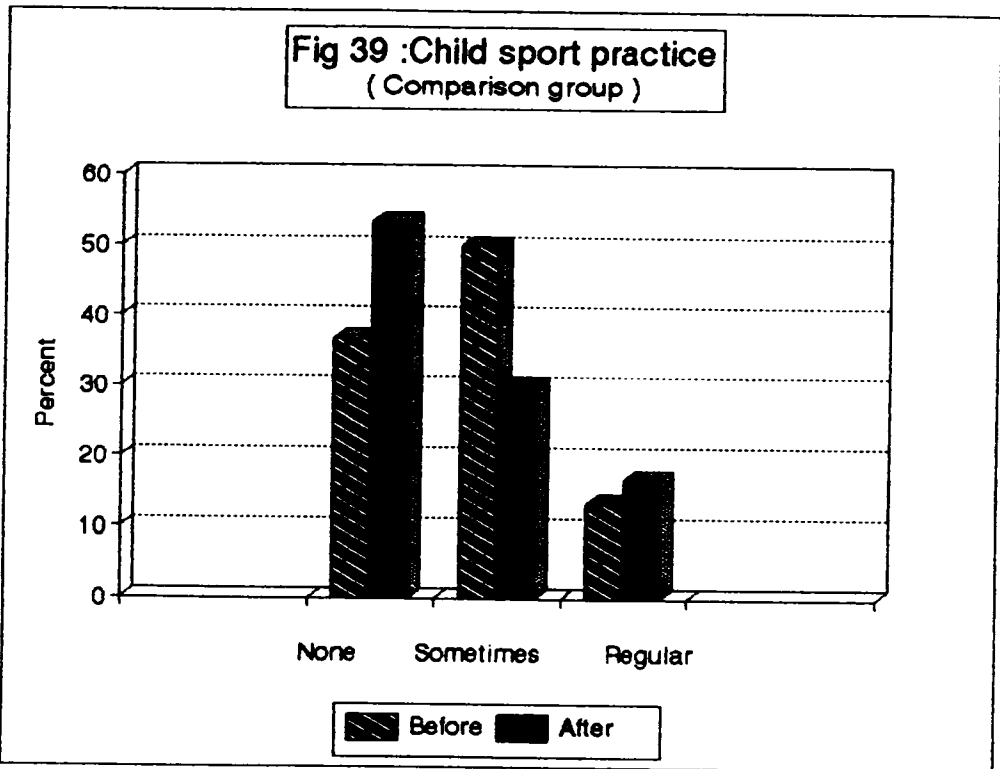
Table 57 :***Child sport practice :***

| Practice | Comparison group | | | | Intervention group | | | |
|-----------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| None | 11 | 36.7 | 16 | 53.3 | 11 | 39.3 | 7 | 25 |
| Sometimes | 15 | 50 | 9 | 30 | 15 | 53.6 | 13 | 46.4 |
| Regular | 4 | 13.3 | 5 | 16.7 | 2 | 7.1 | 8 | 28.6 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,regular sport practice improved in the comparison group from 4 (13.3%)to 5 (16.7%) ,while in the intervention group improved from 2 (7.1%) to 8 (28.6%) which reaches a statistically significant improvement Chi Square = 4.38 ,P = 0.03 (< 0.05) .Figures 39,and 40 demonstrate the table .

Table 58 :***Restriction of certain types of food because of asthma:***

| Restriction of food | Comparison group | | | | Intervention group | | | |
|---------------------|------------------|-----|-------|------|--------------------|-----|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 18 | 60 | 13 | 43.3 | 21 | 75 | 11 | 39.3 |
| No | 12 | 40 | 17 | 56.7 | 7 | 25 | 17 | 60.7 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |



As shown from the table ,restriction of food in the comparison group changed from 18 (60%) at start of follow up to 13(43.3%), while in the intervention group changed from 21 (75%) at start of PGM to 11 (39.3%) at the end of PGM .The change in the comparison group is statistically insignificant $X^2 = 1.67$, $P = 0.19$ (>0.05). In the intervention group ,the change is significant $X^2 = 7.29$, $P = 0.006$ (< 0.05) .Figure 41 demonstrate the table .

Home environments of the asthmatic children :

Table 59 :

Presence of smoking persons in the flat :

| Presence of smoking person | Comparison group | | | | Intervention group | | | |
|----------------------------|------------------|-----|-------|------|--------------------|-----|-------|-----|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 24 | 80 | 22 | 73.3 | 14 | 50 | 14 | 50 |
| No | 6 | 20 | 8 | 26.7 | 14 | 50 | 14 | 50 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,in the comparison group 80% of children had smoking persons in their flat at start of follow up and 73.3% at the end ,while in the intervention group 50% of children had somking persons in the flat at start of PGM and 50% at the end . There was no significant change before and after PGM .

Table 60 :***Whether they smoke inside the flat :***

| Smoking inside flat | Comparison group | | | | Intervention group | | | |
|---------------------|------------------|------|-------|------|--------------------|-----|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 20 | 83.3 | 21 | 95.5 | 14 | 100 | 10 | 71.4 |
| No | 4 | 16.7 | 1 | 4.5 | 0 | 0 | 4 | 28.6 |
| Total | 24 | 100 | 22 | 100 | 14 | 100 | 14 | 100 |

As shown from the table ,in the comparison group 83.3% out of 24 somking persons were smoking inside the flat at start of follow up and 95.5% out of 22 smoking persons were smoking inside the flat .In the intervention group 100% of the 14 smoking persons were smoking inside the flat at start of the PGM while at the end of the PGM dropped into 10 (71.4%) out of 14 smoking persons. This improvement is statistically significant improvement in the intervention group ,Chi Square = 4.67 ,P = 0.03 (< 0.05) . Figure 42 demonstrate the table .

Fig 41 :Food restriction because of asthma

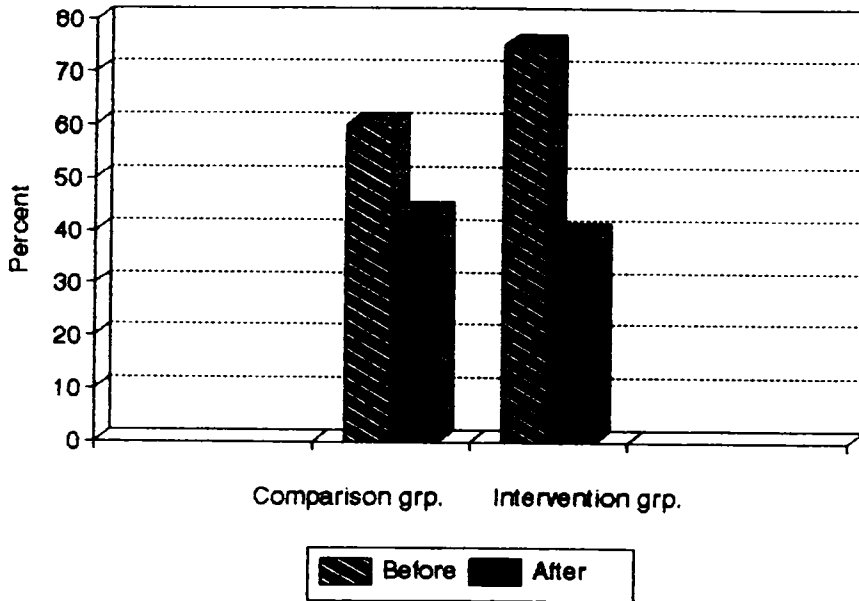


Fig 42 :Smoking inside flat

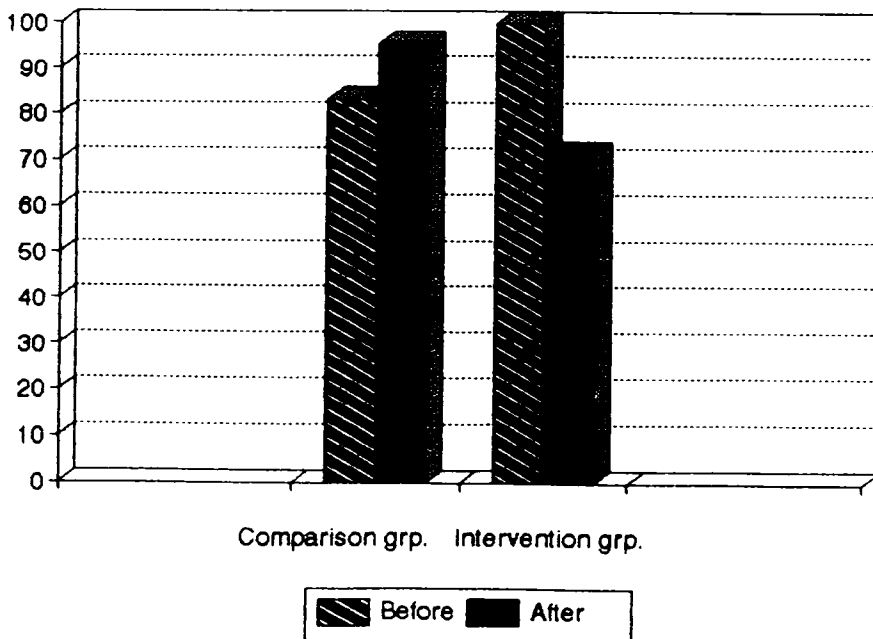


Table 61 :

Use of child's room for other purposes (smoking ,cooking or as store) :

| Use of child's | Comparison group | | | | Intervention group | | | |
|----------------|------------------|-----|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 12 | 40 | 11 | 36.7 | 12 | 42.9 | 6 | 21.4 |
| No | 18 | 60 | 19 | 63.3 | 16 | 57.1 | 22 | 78.6 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,in the comparison group there was slight change 12 (40%) before to 11 (36.7%) after .In the intervention group the change is bigger from 12 (42.9) before to 6 (21.4) but it did not reach statistically significant change , $\chi^2 = 2.95$, $P = 0.08$ (> 0.05) .

Table 62 :

Use of sprays in the flat as an insecticides :

| Use of sprays | Comparison group | | | | Intervention group | | | |
|---------------|------------------|------|-------|------|--------------------|------|-------|-----|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 13 | 43.3 | 14 | 46.6 | 17 | 60.7 | 7 | 25 |
| No | 17 | 56.7 | 16 | 53.3 | 11 | 39.3 | 21 | 75 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,in the comparison group there was an increase in use of sprays in the flat from 43.3% before to 46.6% after follow up ,in the intervention group there was a statistically significant decrease in use of sprays $X^2 = 7.3$, $P = 0.006$ (<0.05).Figure 43 demonstrate the table .

Table 63 :

Presence of plants inside the flat :

| Plant present | Comparison group | | | | Intervention group | | | |
|---------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 2 | 6.7 | 8 | 26.7 | 3 | 10.7 | 3 | 10.7 |
| No | 28 | 90.3 | 22 | 73.3 | 25 | 89.3 | 25 | 10.7 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,in the comparison group there was an increase in presence of plants from 6.7% before to 26.7% after . in the intervention group ,there was no change 10.7% before and after PGM .

Table 64 :

Presence of dogs ,cats ,domestic animals or birds in the house :

| Present | Comparison group | | | | Intervention group | | | |
|---------|------------------|------|-------|-----|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 2 | 6.7 | 6 | 20 | 8 | 28.6 | 2 | 7.1 |
| No | 28 | 93.3 | 24 | 80 | 20 | 71.4 | 26 | 92.9 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,in the comparison group there was an increase in domestic animals or birds in the house from 6.7% before to 20% after follow up ,in the intervention group there was a decrease from 8(28.6%) before to 2(7.1%) after PGM ,this decrease is statistically significant $X^2 = 4.38$, $P = 0.038$, (< 0.05) . Figure 44 demonstrate the table .

Fig 43 :Use of sprays in the flat

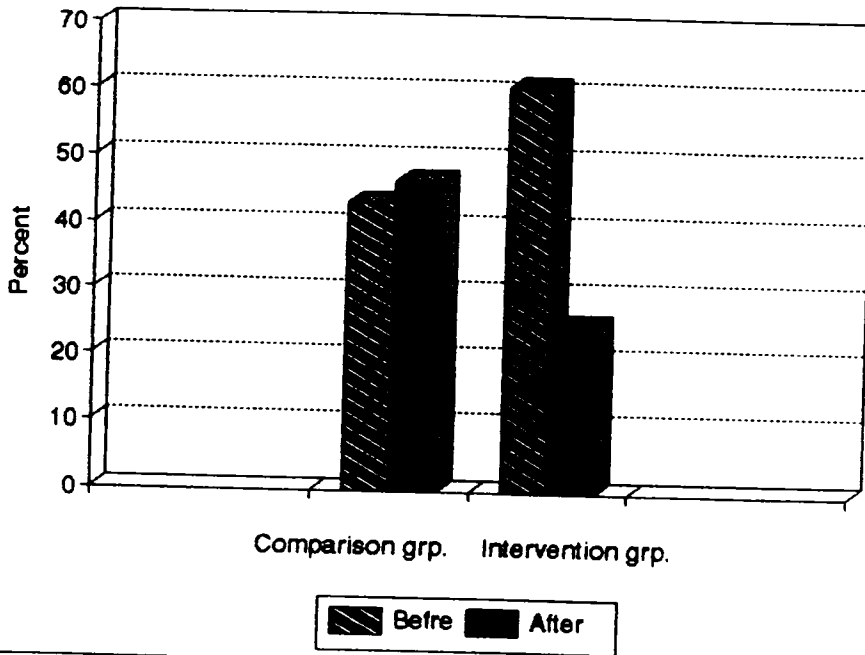
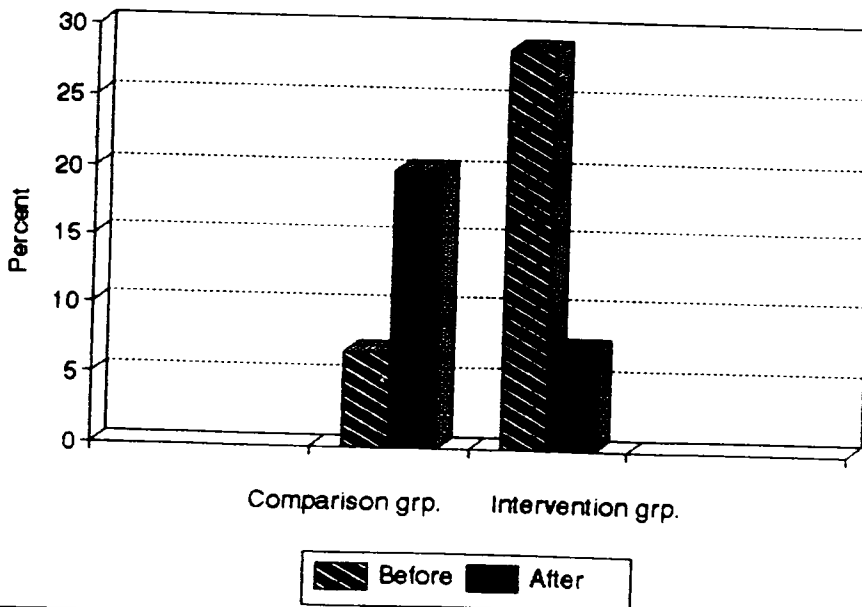


Fig 44 :Presence of domestic animals or birds in the house



Information related to severity of asthma :Table 65 :*Presence of asthma attacks last year :*

| Present | Comparison group | | | | Intervention group | | | |
|---------|------------------|------|-------|-----|--------------------|-----|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Yes | 29 | 96.6 | 27 | 90 | 28 | 100 | 19 | 67.9 |
| No | 1 | 3.4 | 3 | 10 | 0 | 0 | 9 | 32.1 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,in the comparison group a decrease in presence of asthma attacks from 96.6% before to 90% after ,which is statistically insignificant $X^2 = 1.07$, $P = 0.3$,in the intervention group a decrease in presence of asthma attacks from 28 (100%) before to 19 (67.9%) after PGM is statistically significant $X^2 = 10.7$, $P = 0.001$ (< 0.05) .

Table 66 :**Classificatin of degree of asthma according to medical score :**

| Degree of asthma | Comparison group | | | | Intervention group | | | |
|------------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Mild | 7 | 23.3 | 6 | 20 | 7 | 25 | 16 | 57.1 |
| Moderate | 16 | 53.4 | 13 | 43.3 | 13 | 46.4 | 10 | 35.7 |
| Severe | 7 | 23.3 | 11 | 36.7 | 8 | 28.6 | 2 | 7.2 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

As shown from the table ,in the comparison group there was a decrease in mild cases from 7 (23.3%) to 6 (20%) ,decrease in moderate cases from 16 (53.4%) to 13 (43.3%) ,and increase in severe cases from 7 (23.3%) to 11 (36.7%) , there is no statistically significant change in mild cases before and after PGM , $X^2 = 0.1$, $P = 0.75$.In the intervention group there was an increase in mild cases from 7 (25%) to 16 (57.1%) ,decrease in moderate cases from 13 (46.4%) to 10 (35.7%) ,decrease in severe cases from 8 (28.6) to 2 (7.2%) ,there is statistically significant improvement in mild cases before and after PGM , $X^2 = 5.98$, $P = 0.014$ (< 0.05) .

Table 67 :

Assessment of severity of asthma by medical score in the comparison group ,before and after follow up :

| Degree of severity | Comparison group | | | | Chi Squ | P value | Sig. |
|--------------------|------------------|------|-------|------|---------|---------|------|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Mild | 7 | 23.3 | 6 | 20 | 0.1 | 0.75 | NS |
| Moderate | 16 | 53.3 | 13 | 43.3 | 0.6 | 0.43 | NS |
| Severe | 7 | 23.4 | 11 | 36.7 | 1.27 | 0.25 | NS |
| Total | 30 | 100 | 30 | 100 | | | |

As shown from the table ,there was no significant changes in degree of severity before and after follwo up . Figure 45 demonstrate the table .

Table 68 :

Assessment of severity of asthma by medical score in the intervention group ,before and after program :

| Degree of severity | Comparison group | | | | Chi Squ | P value | Sig. |
|--------------------|------------------|------|-------|------|---------|---------|------|
| | Before | | After | | | | |
| | No. | % | No. | % | | | |
| Mild | 7 | 25 | 16 | 57.1 | 5.98 | 0.01 | S |
| Moderate | 13 | 46.4 | 10 | 35.7 | 0.66 | 0.41 | NS |
| Severe | 8 | 28.6 | 2 | 7.2 | 4.38 | 0.03 | S |
| Total | 28 | 100 | 28 | 100 | | | |

As shown from the table ,there was significant improvement in degree of asthma before and after PGM in mild and severe cases. Figure 46 demonstrate the table .

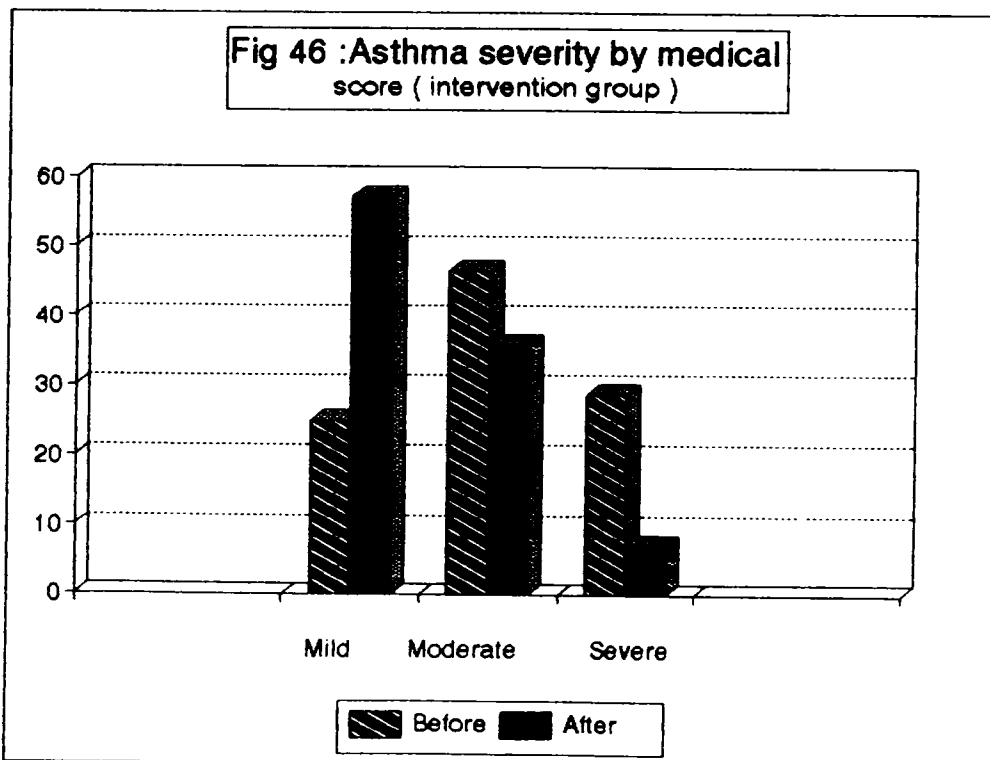
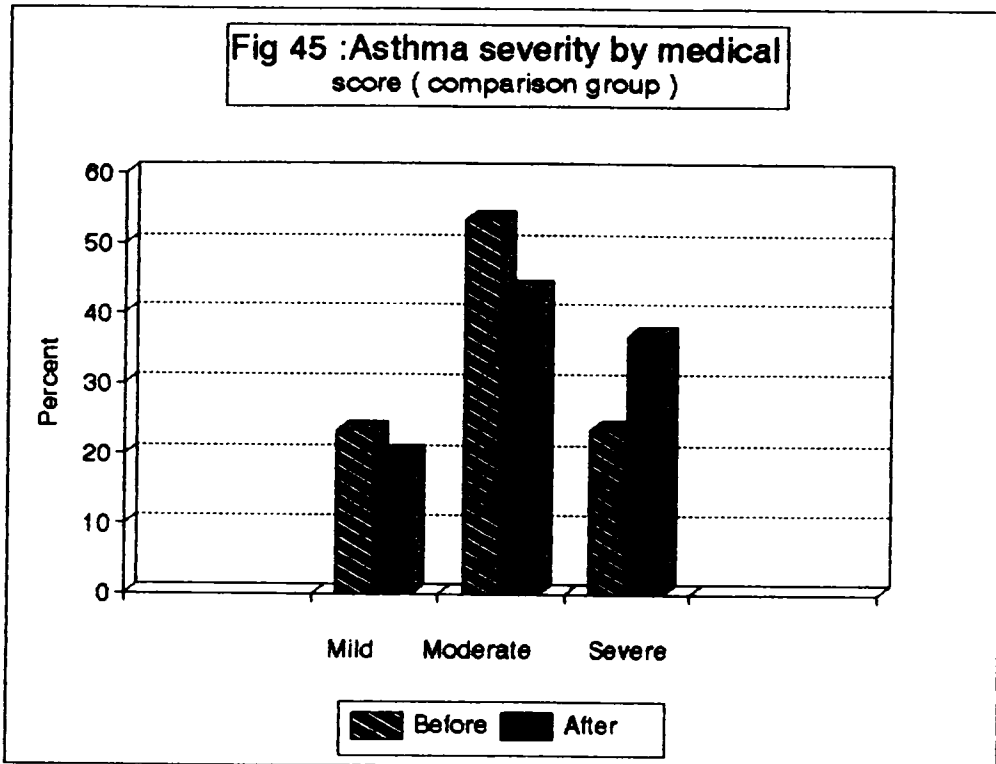


Table 69 :

Classification of degree of asthma according to PEFR measurements:

| Degree of asthma | Comparison group | | | | Intervention group | | | |
|------------------|------------------|------|-------|------|--------------------|------|-------|------|
| | Before | | After | | Before | | After | |
| | No. | % | No. | % | No. | % | No. | % |
| Not assessed | 2 | 6.7 | 1 | 3.3 | 2 | 7.1 | 1 | 3.6 |
| Asymptomatic | 2 | 6.7 | 1 | 3.3 | 5 | 17.9 | 8 | 28.6 |
| Mild | 4 | 13.3 | 12 | 40 | 6 | 21.4 | 8 | 28.6 |
| Moderate | 17 | 56.7 | 9 | 30 | 9 | 32.2 | 7 | 25 |
| Severe | 5 | 16.6 | 7 | 23.4 | 6 | 21.4 | 4 | 14.2 |
| Total | 30 | 100 | 30 | 100 | 28 | 100 | 28 | 100 |

Not assessed means that PEFR could not be measured

Asymptomatic means that PEFR is 100% or more of the predicted normal.

Mild means that PEFR is > 80% of the predicted normal

Moderate means that PEFR is from 60% to 80% of predicted normal

Severe means that PEFR is < 60% of the predicted normal.

As shown from the table ,in the comparison group asymptomatic cases decreased from 2 (6.7%) to 1 (3.3%) ,mild cases increased

from 4 (13.3%) to 12 (40%) ,moderate cases decreased from 17 (56.7) to 9 (30%) ,severe cases increased from 5 (16.6%) to 7 (23.4%) ,there is no statistically significant changes before and after follow up .In the intervention group ,asymptomatic cases increased from 5 (17.9%) to 8 (28.6%) ,mild cases increased from 6 (21.4%) to 8 (28.6%) ,moderate cases decreased from 9 (32.2%) to 7 (25%),and severe cases decreased from 6 (21.4%) to 4 (14.2%) ,there is tendency for improvement in the intervention group than the comparison group .There is no statistically significant improvement before and after PGM .

Table 70 : demonstrates variables of the program before and after intervention . The first column demonstrates the variable , the second column demonstrates differences between the comparison and intervention groups before program , the third column demonstrates differences between the same groups after the program :

| Variable | Before program | After program |
|--|---|--|
| Knowledge of caretakers about triggers of asthma attacks | No significant differences between the two groups: Complete P=0.59 ,NS incomplete P=0.11,NS d'not know P=0.06,NS | Sign. improvement in the intervention group: complete P=0.005, S. d'not know P=0.023, S. |
| Practice to prevent asthma attacks | No sign. differences between the two groups: Complete P=0.15, NS incomplete P=0.62,NS d'not know P=0.10,NS | Sign. improvement in the intervention group: d'not know P=0.023,S. |
| Knowledge about early warning signs of asthma attacks | No sign. differences between the two groups: complete P=0.91,NS incomplete P=0.97,NS d'not know P=0.90,NS | Sign. improvement in the intervention group: complete P=0.014.S |
| Practice at start of asthma attacks | No sign. differences between the two groups: complete NS incomplete P=0.69,NS d'not know P=0.69,NS | Highly sign. improvement in the intervention group: complete P=0.0001,HS |
| Knowledge about signs of asthma during attacks | No sign. differences between the two groups: complete NS incomplete P=0.55,NS d'not know P=0.53,NS | Highly sign. improvement in the intervention group: complete P=0.001.HS |
| Practice during asthma attacks | Sign. better practice in the comparison group: complete P=0.02,S. incomplete P=0.007,S d'not know P=0.16,NS | Highly sign. improvement in the intervention group: complete P=0.0001,HS |

| | | |
|--|--|---|
| Feelings about usefulness of drug treatment | No sign. differences between the two groups: P=0.81 ,NS. | No sign. differences between the two groups: P=0.27 ,NS. |
| Restriction of child's activity because of asthma | No sign. differences between the two groups: P=0.29 ,NS. | Sign. improvement in the intervention group: P=0.014 ,S. |
| Prevention of child from going to school because of asthma | No sign. differences between the two groups: All children missed one day or more because of asthma. | Improvement in the intervention group , but do not reach sign. difference: P=0.09 ,NS |
| Child school performance | No sign. differences between the two groups. | Sign. improvement in the intervention group, in grade good: P=0.053 ,S. |
| Child sport practice | No sign. differences between the two groups: None P=0.83,NS sometimes P=0.78,NS regular P=0.34,NS | Sign. improvement in the intervention group: P=0.027 ,S. |
| Restriction of certain types of food because of asthma | No sign. differences between the two groups: P=0.22, NS | No sign. differences between the two groups: P=0.75 , NS. |
| Presence of smoking persons in the flat | Sign. increase in smoking persons in the comparison group: P=0.016,S. | No sign. differences between the two groups: P=0.067 ,NS. |
| Smoking inside the flat | No sign. differences between the two groups: P=0.10, NS | Sign. improvement in the intervention group: P=0.042 ,S. |
| Use of child's room for other purposes | No sign. differences between the two groups: P=0.95 ,NS | No sign. differences between the two groups: P=0.20 ,NS. |
| Use of sprays in the flat as an insecticides | No sign. differences between the two groups: P=0.18 ,NS | Improvement in the intervention group ,but do not reach sign. improvement P=0.086 ,NS. |

| | | |
|---|--|--|
| Presence of plants inside the flat | No sign. differences between the two groups: P=0.58 ,NS | No sign. differences between the two groups: P=0.12 ,NS. |
| Presence of dogs ,cats,domestic animals or birds in the house | Sign. increase in the intervention group: P=0.027 ,S | No sign. differences between the two groups: P=0.155 ,NS. |
| Presence of asthma attacks last year | No sign. differences between the two groups: P=0.32 ,NS | Significant improvement in the intervention group: P=0.037 .S. |
| Severity of asthma by medical score | No sign. differences between the two groups. | Highly improvement in asthma severity in the intervention group: Mild cases:P=0.003,HS Severe cases:P=0.007, HS. |

DISCUSSION

Discussion

During the past decade , significant advances have been made in researches concerned with the self management of childhood asthma. These include a number of research programs aimed at developing model health education programs for children with asthma and their families .By 1982 ,at least seven major asthma self-management research programs had been developed either in controlled or uncontrolled settings .It is possible to conclude from the currently available asthma-self management research that these programs improve knowledge and practice toward asthma and increase the self-management behaviours taken by families (Park ,1987) .

Although new diagnostic and therapeutic modalities for asthma have improved the quality of life for many children with asthma ,there is still significant morbidity and mortality . Important reasons include lack of patient compliance ,lack of appreciation of adjustment problems ,and the impact of asthma on family dynamics .Although some patients are not receiving proper medical management ,the level of control and functioning of those who are receiving appropriate care are often not optimal (Rachelefsky , 1987) .

For both adults and children , key areas in the self-management of asthma require the following decision making skills : *Triggers* : the identification of individual triggers followed by the decision to avoid ,eliminate ,or

premedicate before exposure .*Symptoms control* : recognition of one's prodromal or early warning signs and the decision to medicate early ,continue medicating according to a predetermined protocol until symptoms subside .
If symptoms worse : Call the physician or seek emergency care at a predetermined place .These factors form the essence of self-management of asthma ,which when achieved reduces morbidity ,fear ,anxiety ,feelings of helpless ,and mortality as well (Hindi-Alexander ,1987) .

There are four recognized main categories of risk factors that contribute to death from asthma :1 - delays in getting / receiving appropriate care ,
 2 - medication misuse (underuse , overuse , interaction , and toxic effects),
 3 - inappropriate care (sedation , insufficient corticosteroids) ,4 - other risk factors as nocturnal asthma , labile and hyperreactive airways , and infection .
 All are amenable to preventive measures . Delays are one of the major causes of asthma deaths (Hindi-Alexander and Middleton , 1986) .

The program achieved a significant improvement in knowledge and practices of caretakers as regard knowledge of asthma triggers ,
 practice in between the attacks ,knowledge of early warning signs of asthma attacks ,practice at beginning of asthma attacks , knowledge about signs of asthma attacks ,and practice during asthma attacks ,this will be discussed in the following points :

Knowledge of caretakers :In the intervention group showed statistically

significant improvement about triggers of asthma attacks .Knowledge of caretakers in the comparison group did not change significantly before and after follow up .The program stressed to identify possible triggers of asthma episodes for each child .Some families were knowledgeable about triggers of their child's asthma ,the majority were not . Possible triggers of asthma attacks were discussed and demonstrated through written instructions as well as drawing pictures , a sample of which is present in the appendix . These results are comparable to those published by Whiteman et al , 1985 ,who evaluated an asthma self-care program (self care rehabilitation in pediatric asthma)where 38 school aged children were randomly assigned to a study and control group , results demonstrated an increase in knowledge and skills in the study group as compared with the control group .

Practice of caretakers to prevent asthma attacks : in the comparison group did not change significantly ,while in the intervention group there was statistically significant improvement as regard avoiding the cause and use of prescribed drugs . During the educational sessions , the families were taught that avoiding the triggers is one of the important factors to reduce symptoms, A series of studies indicated that avoidance of specific allergens can both reduce asthma symptoms and decrease non-specific bronchial hyperreactivity (Platts-Mills , 1993) . Depending on the specific factors which provoke attacks ,children may have to avoid particular foods ,stay away from dusty or smoky

environments , and be protected against catching colds .Gelber et al .1993 , concluded that the dominant allergens associated with chronic asthma are found indoors ,i.e .mites ,cats ,dogs and cockroaches .

Knowledge of caretakers about early warning signs of asthma attacks

did not change significantly in the comparison group before and after follow up ,except in those who did not know any sign before follow up (30%) .,decreased to 10% .Other categories did not reach significant changes .In the intervention group , there was statistically significant improvement before and after PGM in the categories who did not know four signs ,and highly significant in those who did not know any sign before PGM .

Practice of caretakers at start of asthma attacks : In the comparison group did not change significantly before and after follow up ,in the intervention group there was significant improvement before and after PGM as regard use of prescribed drugs ,giving warm fluids ,keep child calm ,and keep child in rest and under observation .Fireman et al, 1981, in a controlled trial of asthma education based in a physician's office with 26 children with asthma , reported more use of prescribed medications ,and earlier initiation of asthma therapy, the educated group knew how to prevent the development of asthma by earlier recognition of symptoms along with earlier initiation of therapy .

Knowledge of caretakers about signs of asthma during attacks :There was significant improvement in the comparison group before and after follow

up ,in the intervention group there was highly significant improvement ,this improvement is higher than those of the comparison group . The improvement in the comparison group is related to the favourable impact of repeated follow up visits and stress of physicians on health educaion and general public awareness .

Practice of caretakers during asthma attacks : the comparison group did not show significant improvement ,the intervention group showed significant improvement .The families were taught to seek medical care in hospitals once the symptoms become worse in spite of use of prescribed medications .Hindi-Alexander and Cropp , 1984 ,in their family asthma program ,reported a significant increase in knowledge about asthma and a significant change in attitude toward asthma , indicating an interest to assume more responsibility for self-management .

Perception of caretakers about usefulness of drug treatment : In the comparison group there was no significant change before and after follow up ,in the intervention group , there was significant change toward usefulness of drug treatment before and after PGM . At the start of the program ,40 (53.0%) of caretakers felt that the drug treatment is useful ,34 (4.4.4%) reported not useful or sometimes useful. The widely used cough mixtures containing antihistamines ,sedatives and antitussives were discouraged during the attacks .Specific asthma treatment drugs according to individualized tailored plan were recommended.

Restrictions of child's activity : It was decreased in the comparison as well as the intervention groups ,increase in child's activity was more in the intervention group than the comparison group .At the start of the program 71 (94.7%) of children had restricted activity ,and 4 (5.3%) were not .Restricted activity means not going outside the flat for somedays last year for fear of asthma .The instructions delivered to caretakers stress on participation of children in ordinary physical activities as playing with his peers and friends and participation in exercise classes in the school according to individual adjustments .The increase in child activities is a significant point because these children were frequently prevented from participating in many activities especially in the school for fear of triggering an asthma episode. Within school itself , some children experience problems if they have to reduce activities that

provoke attacks or if they are not allowed to participate in normal physical activities (Richards , 1986) .

School absence : It was decreased in the comparison group , 100% of the 24 children attending school at start of follow up missed one day or more ,this figure decreased to 46.2% of the 26 children attending school at the end of follow up .In the intervention group ,100% of the 18 children attending school at start of the PGM missed one day or more ,this figure decreased to 22.7% of the 22 children attending school at the end of the PGM .School absence decreased in the intervention than the comparison group after the end of PGM. The decrease in school absence noticed in the comparison group can be explained by the fact that older children assume more responsibility for school attendance ,and due to newly introduction of school health insurance system during conduction of the study . The decrease in school absence in the intervention group is related to the same reasons ,in addition to improvement in asthma severity .Speight et al ,1983 , found that since starting school ,one in three 7 year old children who suffered from asthmatic symptoms had missed more than fifty days schooling as a direct result of asthma ,this represented more than three times the usual number of absences .It is in their early years that children with asthma miss most days schooling ,younger children are more likely to pick up respiratory infections which precipitate attacks .School absence can affect academic performance ,and frequent short absences are

generally more harmful in this respect than an occasionally long one (Douglass and Ross , 1965) .

Child school performance : In the comparison group poor school performance increased from 3 (12.5%) to 10 (38.5%) which is statistically significant $X^2 = 4.41$, $P = 0.035$ (< 0.05) , good school performance increased from 25% to 26.9% . In the intervention group , bad school performance decreased from 16.7% to 13.6% , and good school performance increased from 3 (16.7%) to 11 (50%) which is statistically significant $X^2 = 4.87$, $P = 0.02$ (< 0.05) . The increase in the group of poor school performance in the comparison group in spite of decrease in school absence can be related to increased asthma severity , and to other factors e.g bad family relations or low socioeconomic level . The increase in the group of good school performance in the intervention group is explained by better school attendance , improvement in asthma severity , and that the children who could not be assessed in the intervention group at the beginning of the PGM (16.7%) could be of better school performance . Other grades including accepted , very good and excellent did not show significant changes in both groups before and after PGM .

Clark et al , 1984 , evaluated the effects of their program on school performance and the adjustment of children with asthma . They observed a favourable impact on school related problems and showed increased

attendance as compared with controls . Children in the treatment group were significantly more able to maintain their grade levels than those in the control group .

Child sport practice : regular sport practice in the comparison group changed from 13.3% to 16.7% which is insignificant statistically ,while in the intervention group increased from 7.1% to 28.6% which is statistically significant. The PGM emphasized on regular participation of sport activities in school and with friends . During initial interviews , 29 child (38.2%) did not practice sport at all ,38 (50%) practiced sport sometimes ,and 8 (10.5%) practiced sport regularly . In order to avoid exercise induced asthma ,in addition to the use of drugs ,the child instructed to breathe via the nose as long as possible ,to perform a warm-up period (in exertion intervals of 1 - 2 minutes) with the aim to induce refractoriness to exercise induced asthma ,the child should play appropriate kind of sports as swimming ,cycling ,and physical activities with short breaks as in the case of ball games (Lindemann , 1990) .Donnally et al , 1987 , reported that 61% of the parents of asthmatic children said their children's participation in sporting activities was restricted .Coughlin , 1988 , concluded that 22% of a sample of 111 children with asthma had been advised to avoid some sports ,25% had been unable to complete a game involving exertion ,and 39% had occasionally missed sport due to their asthma .A regular and reasonable performance of exercise improves physical fitness

,enhances skills ,and improves child's psychological state (Pierson , 1988) .

Restriction of certain types of food : in the comparison group decreased from 18 (60%) to 13 (43.3%) ,while in the intervention group decreased from 21 (75%) to 11 (39.3%) .The decrease in the comparison group is insignificant statistically $X^2 = 1.67$, $P = 0.19$ (> 0.05) .The decrease in the intervention group is statistically highly significant $X^2 = 7.29$, $P = 0.006$ (< 0.050).

Most of children with bronchial asthma were restricted from eating certain types of food e.g. egg ,milk ,lentils ,fish ,all of these foods are important for growth and nutrition of the growing children ,in most cases restriction has no scientific bases (e.g documented skin test sensitivity and history of asthma attacks after ingestion of the suspected food and within few hours of ingestion) .

At the initial interviews 53 (69.7) of the 76 children were restricted from certain types of food .Of these restricted children 20 (37.7%) because of physician's instructions ,27 (50%) the caretakers mentioned that it casuses the attacks.42 children out of 53 (79.2%) were restricted from egg, 33 (62.2%) were restricted from fish , 25 (47.2%) were restricted from milk ,19 (35.8%) were restricted from banana , 11 (20.8%) restricted from strawberries ,and 23 (43.4%) from other foods e.g chocolates and lentils .By asking mothers if they prepare other meals containing these restricted food and eaten by the child (cake) ,42 out of 53 (79.2%) answered yes .By asking mother if asthma

develops after eating these other meals , 25 answered that asthma did not develop after eating these meals,while 22 answered that asthma develops after eating these meals .

El Hefny et al , 1992 ,in their study "egg and milk allergy in Egyptian asthmatic children - Evaluation of their nutritional pattern" ,concluded that in our country food allergy as a cause of asthma is overdiagnosed .Many asthmatic children are deprived from egg and milk ,yet their asthma is not controlled .Pediatricians and general practitioners give mothers instructions to eliminate egg and milk products from diet of their children without proper diagnosis .The study reported that the incidence of egg and milk induced asthma by dietetic history was 22.4% and 12.1% respectively among asthmatic children.

Elimination and challenge is the most important tool in the diagnosis of food allergy but it is not easy to be done as most patients were uncooperative .They recommended that for patients with egg and or milk induced asthma to omit these elements from their diet . Doing this ,one must be sure that it is the real cause and not to forget that an asthmatic child is a growing one and needs to receive protein of high biological value.

Home environments of the asthmatic children :

Presence of smoking persons in the flat : in the comparison group , 80% of children at start of follow up had at least one smoking person in their flat, this proportion decreased to 73.3% at the end of follow up .In the

intervention group 50% of children at start of the PGM had at least one smoking person in their flat , this proportion remained the same at the end of the PGM .The PGM stressed stop smoking of the smoking persons, but there was no favourable response and the comparison group improved in this respect than the intervention group ,this improvement did not reach statistically significant change .During the initial interviews 47 families (61.8%) out of the 76 enrolled families had at least one smoking person in the flat .Of them 6 (12.8%) had more than one smoking persons in the flat .40 out of 47 (85.1%) were smoking both inside and outside the flat .Of the 47 smoking persons ,38 (80.9%) were the fathers , 1 (2.1%) was the mother , 3 (6.4%) were the siblings ,and 5 (10.6%) were other relatives to the child including grandfathers and uncles . The improvement in the comparison group could be related to awareness of hazards of smoking or due to economic factors . The results as regard presence of smoking persons in the flat between the two groups before program revealed significant increase in smoking persons in the comparison group ($P=0.016$) . This increase is changed to insignificant difference between the two groups at the end of the program ($P=0.067$), as shown from table 70 .

Whether the smoking person smoke inside flat : in the comparison group 83.3% out of the 24 smoking persons were smoking inside the flat at start of follow up ,at the end of follow up 95.5% out of 22 smoking persons were smoking inside the flat .In the intervention group ,100% out of the 14

smoking persons were smoking inside the flat at the start of the PGM ,this proportion dropped into 71.4% (10 out of the 14 smoking persons) .This improvement in the intervention group is statistically significant $\chi^2 = 4.67$, $P = 0.03$ (<0.05).Lewis et al , 1984 , in their program " Asthma care training " reported changes in smoking behaviour in eight parents in the experimental group and one from the control group . In all cases ,this involved the smoking habit of parents .parents did not stop smoking but no longer smoked in the house or in areas where it could affect the child .Ronchetti et al ,1990 , studied the effects of environmental tobacco smoke in a sample of 166 nine year old children ,the relationship between parental smoking and degree of bronchial responsiveness in males was significant .Also .prick skin tests reactivity to allergens was significantly increased in children with smoking parents . The results concerning smoking inside the flat between the two groups at the start of the program revealed insignificant difference ($P=0.10$) . This is changed to significant improvement in the intervention group at the end of the program ($P=0.042$) , as shown from table 70 . The program could not reduce the number of smoking persons , but could change the smoking behaviour of the smoking persons .

Use of child's room for other purposes (smoking ,cooking or as a store) :

in the comparison group there was a decrease in use of child's room for other purposes from 12 (40%) to 11 (36.7%) , in the intervention group there was a decrease in use of child's room from 12 (42.9%) to 6 (21.4%), this decrease is statistically insignificant $\chi^2 = 2.95$, $P = 0.08$ (>0.05) .The program instructed parents to avoid smoking ,cooking or use of child's room as a store .During initial interviews 27 families out of 76 (35.5%) were using child's room for other purposes than sleeping , of them 22.2% used child's room for cooking , 51.9% for smoking ,11.1% as stores for old furnitures ,and 14% for other purposes as sewing .The limited improvement as regard use of child's room could be explained by the limited number of rooms / flat . The mean of persons living in the flat = 5.91 person / flat ,while the mean of rooms in the flat = 3.16 room / flat (including the hall) .This means that approximately every three persons will share one room .

Cooking deprive the room from fresh oxygen ,accumulates irritant oil fumes and increase gas production resulting from fuel combustion .Old furnitures and newspapers help accumulation of dust and breeding of insects which exposes the child to its debris and excreta ,with increased humidity and without regular cleaning this can enhance growth of molds .El Hefny et al ,1988 , in their study " common inhalant allergens in extrinsic atopic asthmatic children in rural area in Egypt " concluded that the most common inhalant allergens

were house dust (44%) followed by house dust mite (41%) ,mixed moulds (33%) ,hay dust (31%) ,and straw dust (26%) .

Use of sprays in the flat as an insecticides : in the comparison group there was an increase in use of sprays from 43.3% to 46.6%.

In the intervention group there was a significant decrease in use of sprays inside the flat from 17 (60.7%) to 7 (25%) , $X^2 = 7.3$, $P = 0.006$.At the start of the program ,40 (52.6%) of families used sprays as an insecticides inside the flat .The program discourage the use of sprays that contain irritant fumes and gases in presence of the child .These fumes and gases can predispose the hyperreactive airways to an asthma episode .

Presence of plants inside the flat : in the comparison group ,there was an increase in presence of plants inside flats from 2 (6.7%) to 8 (26.7%), in the intervention group ,there was no change 3 (10.7%) before and after PGM .At the start of the program , 10 (13.2%) of families had plants inside flats .Grass Pollen grains are of the known common triggers of asthma ,in addition ,molds and fungi commonly grows on the plants which are one of the most common inhalant allergens .

Presence of dogs , cats , domestic animals or birds in the house:

In the comparison group there was an increase in presence of domestic animals or birds from 2 (6.7%) to 6 (20%) ,in the intervention group there was a decrease from 8 (28.6%) to 2 (7.1%) ,this decrease is statistically significant $X^2 = 4.38$, $P = 0.03 (< 0.05)$. As regard the results of the two groups before the program , there was a significant increase in the intervention group ($P=0.027$) . The program succeeded to reduce the number of domestic animals in the intervention group , that lead to insignificant difference between the two groups at the end of the program ($P=0.15$) .Anderson et al ,1983 , in their study of asthmatic children found that 20% of families had to get rid of pits .

Presence of asthma attacks last year : in the comparison group there was a decrease in presence of asthma attacks from 29 (96.6%) to 27 (90%) ,,which is statistically insignificant . In the intervention group presence of asthma attacks decreased from 28 (100%) to 19 (67.9%) which is statistically significant improvement $X^2 = 10.7$, $P = 0.001 (< 0.05)$. The decrease in the presence of asthma attacks last year in the intervention group could be explained by better knowledge and better avoidance measures concerning triggers of asthma attacks .

Assessment of asthma severity by medical scores : In the comparison group there was no significant change in degree of asthma in mild,moderate and severe cases ,in the intervention group there was significant improvement

in degree of asthma in mild and severe cases , mild cases increased from 7 (25%) to 16 (57.1%) $\chi^2 = 5.98$, $P = 0.01$,and severe cases decreased from 8 (28.6%) to 2 (7.2%) $\chi^2 = 4.38$, $P = 0.03$ (< 0.05) .At the initial interviews ,18 (23.7%) of children had mild asthma ,35 (46.1%) had moderate asthma ,and 23 (30.3%) had severe asthma .

Assessment of asthma severity by measuring Peak Expiratory Flow

Rate : There is tendency for improvement in the intervention group than the comparison group ,but there was no statistically significant difference .At the initial interviews ,4 (6.9%) of children could not be assessed ,7 (12.1%) were asymptomatic ,10 (17.2%) were mild ,26 (44.8%) were moderate ,and 11 (19%) suffered from severe asthma ,as shown from table 69 .

Problems encountered during conduction of the program :

1 - Some of the caretakers ,at the beginning of the program , were reluctant to use metered dose inhalers ,they preferred syrups or tablets.Most of them were convinced with the use of metered dose inhalers at the end of the program .

2 - The terminology of " asthma " and labelling child as asthmatic developed a sense of worry among caretakers and consequently on the child ,they prefer the term allergic child .

3 - Considerable number of caretakers were illiterate or just can read

and write ,written instructions delivered to them could be read by the child or his sibling ,use of illustrative drawings helped to solve this problem

4 - Considerable number of families were of low socio-economic level ,this class can not easily afford to buy prescribed drugs if it is not freely available from the clinic (which occurred in sometimes) .

5 - Minimal involvement of school personnel as school physician ,teachers ,coaches ,and nurses .Their participation can help compliance and adherence to instructions .

6 - Some families were outreached and observed to be living in unhealthy environments ,where exposed to dust and vehicle waste products as well as damp humid environments specially in the ground floors with under developed infra structures .

7 - Polluted environments of certain urban areas ,where the sample came from e.g Helwan ,Dar El-Salam ,Shoubra ,and center of town ,where cement factories ,pollution producing factories ,under-developed infra structures of certain districts with dusty roads and unavailability of pipe water .

We hope we could alleviate pain and relief suffering of some of those asthmatic children and their families and help them to live better life .

RECOMMENDATIONS

Recommendations

1 - The majority of children with asthma have not received the benefit of such education ,and the majority of physicians are unaware of such programs .The program should be implemented in the private physician's office ,large urban clinics ,schools ,and specialised centers for asthma care.

2 - Participation of school personnel e.g school physician,nurses, teachers ,and coaches in the future asthma education programs .Involvement of school personnel will improve the environments during school time and will share in the process of management by encouraging participation in sport and by avoiding exposure to triggers such as chalk dust after sweeping of classboard ,exposure to tobacco smoke or sweeping ground of classes during school time .Involvement of school physician and nurse will help child detect early signs and permitting use of medication early and,more importantly, prescribing free affordable asthma medications . Involvement of coaches will help child premedicate ,and stress the importance of warm-up and good pacing skills .

3 - Freely prescribed asthma medications should be available for each asthmatic child ,whether through school health insurance system or outpatient and inpatient hospital clinics .

4 - The program can be implemented with assistance of nurses and

interested social workers .For the program to be effective accurate and appropriate informations should be instructed .The instructors should be enthusiastic ,well qualified for teaching skills and actively involve asthmatic children with their caretakers in the learning process .

5 - Choice of places well equiped with audio-visual materials ,will increase the efficacy of educational process.

6 - Prevention of evironmental pollution and raising standard of living of these families on the long term basis can help them for better asthma management .

7 - Production of T.V. Programs for patients and caretakers about signs and symptoms of asthma , how to manage , when to ask advice and when to go to the hospitals .

8 - Camps for asthmatic children or diabetic children to share experience and playing sports under supervision of trained staff .

9 - Influenza vaccine should be given obligatory to asthmatic children to guard against influenza infections .

SUMMARY

Summary

Asthma is a respiratory disease characterized by intermittent or chronic, usually reversible airway obstruction. With optimal therapy it does not cause permanent lung damage, nor should it interfere with normal childhood activities in the majority of children.

In children between the ages of six and 11, the prevalence of asthma has increased from 4.8% in the late 1970s to 7.6 percent in 1980 in the United States. In Egypt, asthma affects approximately 8.2% of children aged 3 to 14 years.

The aim of the study is to assess and increase knowledge about asthma and its treatment among the asthmatic children and their families, to assess and improve practice as regard prevention and treatment, to reduce severity, improve home environments of asthmatic children and increase their participation in normal childhood activities.

The program included 76 asthmatic children and their families, aged between 5 to 12 years, 43 males (56%) and 33 females (43%). It was conducted in the outpatient allergy clinic of Abo El Reish Children Hospital, Cairo University. Starting from March 1992 to December 1993 on a random sample categorized according to severity. Each family were followed up for one year. Families who completed the follow up

period were 58 (76.3%) .Of the 58 families completed the follow up 28 were of the intervention group ,while 30 were of the comparison group . Child's severity of asthma level was determined after the initial interview using medical score and peak expiratory flow rate measurements.

The intervention group received one hour of instructions every two weeks for one year. Instructions included asthma precipitating factors ,signs and symptoms at the beginning of the attacks ,how to deal with child at start of the attack,signs and symptoms of asthma during attacks ,what to do during attacks ,and when to go to the hospital .

Informations about home environments and children activities were collected through a revised questionnaire at start of the program. Another follow up questionnaire was developed to assess the impact of instructions and education on regular basis .During the sessions questions of the families were answered and corrections of believes , written instructions and illustrative drawings were delivered to the families .Peak expiratory flow rate was measured. The comparison group received the routine clinic care .

Data analysis revealed a significant improvement in knowledge and practice of caretakers towards asthma ,significant reduction of severity of asthma,increase in sport practice and reduction of missed school days that resulted in significant impact on child school performance .Home

environments were better controlled as regard significant reduction in persons smoking in the flat and removal of asthma triggering factors e.g. domestic animals or birds.

Distribution of such asthma education program will have its favourable impact on asthma severity ,knowledge ,practice and better control of home environments of families of asthmatic children.

REFERENCES

REFERENCES

- Alexander M.H., Gerd J.A., and Cropp. (1981) :**
Community and family programs for children with asthma
Ann. Allergy 46:143.
- Allison , S.E. (1993) :**
Anna Wolf 's dream . Establishment of a collegiate nursing
education program
Image. j. Nurs. Sch. 25: 127
- American Lung Association (1982) :**
Superstuff - a pediatric asthma self-management program . New
York .
American Lung Association , 1982
- Anderson H.R., Bally P.A., Cooper J.S., Palmer J.C. and West S.(1983):**
Morbidity and school absence caused by asthma and wheezing
illness.
Arch.Dis.Child. 58:777
- Anderson S.D., Silverman M., Konig P., and Godfrey S. (1975) :**
Exercise-induced asthma
Br. J. Dis. Chest 69:1.
- Aronsson G., and Koivunen E. (1985) :**
Differences in personality between parents of asthmatic children.
J. Psychosom. Res. 29:177.
- Avol E.L, Linn W.S., and Shamod D.A. (1990) :**
Respiratory responses to young asthmatic volunteers incontrolled
exposures to sulfuric acid aerosol.
Am. Rev. Resp. Dis. 142:343.
- Bahna S. (1970) :**
Ph. D. thesis.
University of Alexandria. Quoted from : Chaulet P.(1989):Asthma and
chronic bronchitis in Africa .Evidence from epidemiologic studies .
Chest 96 :335

Barnes P.J. (1986) :

Asthma as an axon reflex.
Lancet 1:242

Barnes P.J. (1987) :

Airway receptors .In Jenne J.W, Murphy S. (eds) : Drug therapy for asthma : Research and clinical practice.
New York, Marcel Dikker. pp 67-95.

Barness P.J.(1989) :

A new approach to the treatment of asthma.
N.Engl.J.Med. 321:1517

Barnes P.J. (1989) :

Muscarinic subtypes : Implications for lung disease.
Thorax 44:161

Barnes P.J., Chung K.F.,and Page C.P. (1988) :

Platelet activating factor as a mediator of allergic disease.
J. Allergy Clin. Immunol 81:919

Baucher H.,Leventhol J.,and Shapiro E. (1986) :

Studies of breast-feeding and infections :How good is the evidence?
J A M A 256:887

Beale H.D.,Fowler W.S.,and Comroe J.M. (1952) :

Pulmonary function studies in 20 asthmatic patients in the symptom-free interval
J. Allergy 23:1

Beasley R.,Roche W.R.,Roberts J.A.,and Holgate S.T. (1989) :

Cellular events in the bronchi in mild asthma and after bronchial provocation.
Am Rev Respir Dis 139:806

Birkhead G., Attaway N.J., Strunk R.C., Townsend M.C., and Teutsch S. (1989) :

Investigation of a cluster of deaths of adolescents from asthma :
Evidence implicating inadequate treatment and poor adherence
with medications

J. Allergy Clin Immunol 84:484

Blay D.F., Chapman M., and Mills P.T. (1991) :

Airborne cat allergen (Fed d 1)

Am Rev Resp Dis 143:1334

Bleecker E.R. (1985) :

Airways reactivity and asthma : Significance and treatment

J Allergy Clin Immunol 75:21

Bloomberg G.R., and Strunk R.C. (1992) :

Crisis in asthma care

Ped Clin North Am 39:1225

Bobo J. K., and Davis C.M. (1993) :

Recovering staff and smoking in chemical dependency programs in
rural Nebrasks

J. Subst. Abuse. Treat. 10:221

Bock S.A. (1987) :

Prospective appraisal of complaints of adverse reactions to foods in
children during the first 3 years of life

Pediatrics 79:683

Boushey H.A., Holtzman M.J., Sheller J.R., and Nadel J.A. (1980) :

Bronchial hyperreactivity

Am Rev Respir Dis 121:389

British Thoracic Society (1987) :

Comparison of atopic and non-atopic patients dying of asthma

Br J Dis Chest 81:30

Broback L., and Kalvesten L. (1988) :

Asthma in school children : Factors influencing morbidity in Swedish survey
Acta Ped Scand 77:826

Brook U. (1991) :

The prevalence of bronchial asthma among high school pupils in Holon (Israel) .
J Trop Ped

Brunekreef B., Dockery D., and Speizer F. (1989) :

Home dampness and respiratory morbidity in children
Am Rev Resp Dis 140:1363

Buist A.S. (1989) :

Asthma mortality : What have we learned ?
J Allergy Clin Immunol 84:275

Burney P.G.J. (1986) :

Asthma mortality in England & Wales : Evidence for a further increase
Lancet 2:323

Burrows B., Knudson R., and Lebowitz M. (1977) :

The relationship of childhood respiratory illness to adult obstructive airway disease
Am Rev Resp Dis 115:751

Bush R.K., Taylor S.L., and Busse W. (1986) :

A critical evaluation of clinical trials in reactions to sulfites
J Allergy Clin Immunol 78:191

Busse W.W. (1989) :

The role of inflammation in asthma : A new focus
J Resp Dis 10(11):72

Busse W.W. (1991) :

Exercised-induced asthma : A role for the eosinophil ?
J Allergy Clin Immunol 88:697

Carlsen K.H., Orstavik I., and Leagaard J (1984) :

Respiratory virus infections and aeroallergens in acute bronchial asthma
Arch Dis Child 59:310

Carswell F. (1985) :

Thirty deaths from asthma
Arch Dis Child 60:25

Chan K., and Nable-Jamieson C.B. (1988) :

Prevalence of bronchial hyperresponsiveness in low birth weight children and their mothers
Arch Dis Child 63:905

Chaulet P. (1989) :

Asthma and chronic bronchitis in Africa : Evidence from Epidemiologic studies
Chest 96:334 S

Chiaramente L., and Altman D. (1991) :

Food sensitivity in asthma : Perception and reality
J Asthma 28:5

Ciba Guest Symposium Report (1959) :

Terminology , definitions and classification of chronic pulmonary emphysema and related conditions
Thorax 14:286

Clark N.M. (1989) :

Asthma self-management education , research and implications for clinical practice .
Chest 95: 1111

- Clark N.M., Feldman C.H., and Evans D. (1984):**
Changes in children's school performance as a result of education for family management of asthma.
J. Occup. Safety Health 54:143
- Clark N.M., Feldman C.H., and Evans D. (1986) :**
The impact of health education on frequency and cost of health care use by low income children with asthma
J. Allergy Clin. Immunol. 78:104
- Coakson W., and Hopkin J. (1988) :**
Dominant inheritance of atopic immunoglobulin-E responsiveness
Lancet 1:86
- Cockcroft D.W. (1987) :**
Airway hyperresponsiveness : Therapeutic implications
Ann Allergy 59:405
- Collins M., and Moessinger A (1985) :**
Fetal lung hypoplasia associated with maternal smoking :
A morphometric analysis
Pediatr Res 19:408
- Connors C.K. (1983) :**
Psychological management of the asthmatic child
Clin Rev Allergol 1:163
- Corrigan C.J., Hartnell A., and Kay A.B. (1988) :**
T-lymphocyte activation in acute severe asthma
Lancet 1:1129
- Coughlin S.P.(1980):**
Sport and the asthmatic child:A study of exercise-induced asthma and the resultant handicap.
J.R.Coll.Gen.Pract. 38:253
- Coulson J. (1973) :**
In " The little Oxford Dictionary of Current English " . Edited by Coulson J. , Fourth Edition , P. 423 . Oxford University Press . Delhi .
- Creer T.L., Backiel M., Ultman S., and Leung P. (1985) :**

Living with asthma , NIH publication no. 84-2364 . Bethesda , Md :
Nationa Heart , Lung and Blood Institute , 1985 .

Cypcar D., Stark J., and Lemanske R.F. (1992) :

The impact of respiratory infections on asthma
Ped Clin North Am 39:1259

Davis S.M., Rutledge C.M., and Davis T.C. (1993) :

A family physician-counselor program for medical students.
Fam. Med. 25:327

Deshpande A., and Mc Kenzie S.A. (1986) :

Short course of steroids in home treatment of chidren with
acute asthma
Br Med J 293:169

Donnelly J.E., Donnelly W.J., Thong Y.H.(1987) :

Parental perceptions and attitudes toward asthma and its treatment
: a controlled study .
Soc. Sci. Med 24 : 431

Douglas J.W.B. and Ross J.M.(1965) :

The effects of absence on primary school performance
Br.J.Educ.Psychol. 35:29

Duff A.L., and Mills T.A.E.P. (1992) :

Allergens and asthma
Ped Clin North Am 39:1277

Dunnil M.S. (1960) :

The pathology of asthma with special reference to changes
in bronchial mucosa
J Clin Pathol 13:27

Eason J., and Markowe H.L.J. (1987) :

Controlled investigation of deaths from asthma in hospitals in the North East Thames region
Br Med J 294:1255

EL-Hefny A.M. ,and Moustafa N. (1987) :

The role of Schistosomiasis and Ascariasis in extrinsic atopic asthma in Egyptian Children
Thesis ,Cairo University

EL-Hefny A.M.,Aboul-Hassan A.,Nour S.,EL-Beleidy A.,and Abdel-Alim S.M. (1992) :

The role of some household insects as inhalant allergens in Egyptian asthmatic Children : Efficacy of immunotherapy in cockroach asthma
Thesis ,Cairo University

EL-Hefny A.M.,EL-Baroudy R.,Nour S.,EL-Beleidy A.,and Abou-El-Ezz A.A. (1991) :

Egyptian house dust versus foreign house dust as an important inhalant allergens
Thesis ,Cairo University

EL-Hefny A.M.,EL-Heneidy F.,and Awad M.F. (1989) :

Epidemiologic study of the incidence of bronchial asthma among school children between 6-12 years in Tanta
Thesis ,Cairo University

EL-Hefny A.M.,EL-Heneidy F.,and Fadel A.A. (1988) :

Common inhalant allergens in extrinsic atopic asthmatic children in rural area in Egypt
Thesis ,Cairo University

EL-Hefny A.M.,Haddad H.,Ekladlous E.M,Nassar S.K.,EL-Heneidy F.M,Nour S.,and EL-Sayed M.S.C. (1991) :

Bronchial asthma in Egyptian children .An epidemiological, environmental ,clinical and immunological study .
Final Progress Report ,Vol III
Faculty of Medicine ,Cairo University

EL-Hefny A.M.,Morcos S.R.,Nour S.,Tapouzada S.,and Abdel-Al-Aziz

- A.M. (1992) :**
Egg and milk allergy in Egyptian asthmatic children : Evaluation of their nutritional pattern .
Thesis ,Cairo University
- Erjefalt I.,and Persson C.G.A. (1989) :**
Inflammatory leakage of plasma macromolecules into airway tissue and lumen .
Pulmonary Pharmacology 2:93
- Erskine J.,and Schonell M. (1979) :**
Relaxation therapy in bronchial asthma
J Psychosom Res 23:131
- Expert Panel Report Of National Asthma Education (1991) :**
Sheffer A.L.,Bailey W.C., et al In " Guidelines for the diagnosis and management of asthma : Definition and diagnosis " .National Institute of Health .Bethesda , Maryland.Publication 91-3042 .
August 1991 .
- Feldman C.H. (1987) :**
Asthma education : General aspects of childhood programs .
J. Allergy Clin. Immunol. 80:494
- Fielding J.E.,and Phenow K.J. (1988) :**
Health effects of involuntary smoking
N Engl J Med 22:1452
- Fireman P., Friday G.A., Gira C. , et al (1981) :**
Teaching self-management skills to asthmatic children and their parents in an ambulatory care setting .
Pediatrics 68:341
- Fish J.E.,Jameson L.S., and Albright A. (1984) :**
Modulation of the bronchomotor effects of chemical mediators by prostaglandin F2-alpha in asthmatic subjects
Am Rev Respir Dis 130:571
- Fletcher H.J.,Ibrahim S.A.,and Speight N.C. (1990) :**
Survey of asthma deaths in the Northern region,1970-85

Arch Dis Child 65:163

Fraser P.M., Speizer F.E., Waters S.D.M., Doll R., and Mann N.M. (1971):
The circumstances preceding death from asthma in young people
in 1968-1969
Br J Dis Chest 65:71

French T., and Alexander F. (1941) :
Psychogenic factors in bronchial asthma
Psychosom Med.; Monograph 4

Frew A.J., Moqbel R., and Azzawi M. (1990) :
T-lymphocytes and eosinophils in allergen-induced late - phase
asthmatic response in the guinea pig
Am Rev Respir Dis 141:407

Frigas E. and Gleich G.J. (1986) :
The eosinophil and the Pathology of asthma
J Allergy clin Immunol 77:527

Fujimura M., Nishioka S., Kumabashiri I., et al (1990):
Effects of aerosol administration of a thromboxane synthetase
inhibitor (OK Y-046) on bronchial responsiveness to acetyl choline
in asthmatic subjects
Chest 98:276

Futerman D., Hein K., Reuben N., Dell R., and Shaffer N. (1993) :
Human Immune deficiency virus-infected adolescents : the first 50
patients in a New York City Program
Pediatrics 91:730

Garner A.M., and Wenar C. (1959) :
The mother-child interaction in psychosomatic disorders
Urbana : University of Illinois Press , 1959

**Gebber L.E., Seltzer L.H., Pollart S.H., Chapman M.D., Bouzoukis J.K. and
Platts-Mills T.A.E. (1993):**
Sensitization and exposure to indoor allergens as risk factors
for asthma among patient presenting to hospital.
Am.Rev.Resp.Dis. 147:573

Gelber L., Seltzer L., Pollart S., et al (1991)

Specific Ig E ab and exposure to cat and cockroach allergens as risk factors for acute asthma
 J Allergy Clin Immunol 87:233

Gergen P.J., and Weiss K.B. (1990) :

Changing patterns of asthma hospitalization among children : 1979 to 1987
 J A M A 264:1688

Gergen P.J., Mullally D.J., and Evans R.:(1980):

National survey of prevalence of asthma among children in the United State, 1976 to 1980
 Pediatrics 81:1

Gleich G.J., Flavahan N.A., and Fujisawa T. (1988) :

The eosinophil as a mediator of damage to respiratory epithelium. A model for bronchial hyperreactivity
 J Allergy Clin Immunol 81:776

Godfrey S., et al (1970) :

Quoted from National Heart, Lung, and Blood Institute .Natal Asthma Education Program ,Expert Panel Report. Washington ,DC :US Government Printing Office ;1991. US Dept of Health and Human Services publication 91 : 3042.

Gold W.M. (1973) :

Vagally-mediated reflex bronchoconstriction in allergic asthma
 Chest 63:(Suppl.)115

- Goldfarb A.A., and Venutolo F. (1963) :**
 The use of an antidepressant drug in chronically allergic individuals: A double blind study
 Ann Allergy 21:667
- Gortmaker S.L., Walker D.K., Jacobs F.H., and Ruch-Rass H. (1982) :**
 Parental smoking and the risk of childhood asthma
 Am J Public Health 72:574
- Greenough A., Maconochie I., and Yuksel B. (1990) :**
 Recurrent respiratory symptoms in the first year following preterm delivery
 J Perinat Med 18:489
- Groothuis J., Gutierrez K., and Laver B.A. (1988) :**
 Respiratory syncytial virus infection in children with bronchopulmonary dysplasia
 Pediatrics 82:199
- Gustafsson P.A., Kjellman M., and Cederblad M. (1985) :**
 Family therapy in the treatment of severe childhood asthma
 J Psychosom Res 30:369
- Hahn W. (1966) :**
 Autonomic responses of asthmatic children
 Psychosom Med 28:323
- Halfon N., and Newacheck P.W. (1986) :**
 Trends in the hospitalization for acute childhood asthma, 1970-84
 Am J Public Health 76:1308
- Hanson B., Mc Gue M., and Johanson R.B. (1991) :**
 Atopic disease and immunoglobulin E in twins reared apart and together
 Am J Hum Genet 48:873
- Harnahan J., Tager I., Segal M., et al (1990) :**
 Effect of prenatal smoking on infant lung function
 Am Rev Res Dis 141:282
- Hedberg A., Kempf F., and Josephon M. (1985) :**

Coexistence of beta-1 and beta-2 adrenergic receptors in human heart ·Effects of treatment with receptor antagonists or calcium entry blockers.
J.Pharmacol Exp Ther 234:561

Hibbert G.,and Pilsburg D. (1988) :

Demonstration and treatment of hyperventilation causing asthma
Br J Psychiatry 153:687

Hill R.A.,Standen P.J.,and Tattersfield A.E. (1989) :

Asthma,wheezing and school absence in primary schools.
Arch Dis child 64:246

Hill M.,Szeffler S.J.,and Lorsen G.L. (1992) :

Asthma pathogenesis and the implications for therapy in children.
Ped clin North Am 39:1205

Hindi-Alexander M.C. (1987):

Asthma education programs:Their role in asthma morbidity and mortality.
J.allergy Clin.Immuno. 80:492

Hindi-Alexander,MC,and Cropp G.J.A. (1984):

Evaluation of a family asthma program
J. Allergy.Clin.Immunol 74:505

Hindi-Alexander MC ,and Hiddleten E. (1986):

Asthma deaths:are they preventable?
N. Engl. Allergy Proc. 7:482

Holgate S.T.,Beasley R.,and Twentymen O.P. (1987) :

The pathogenesis and significance of bronchial hyperresponsiveness in airways disease.
Clin Sci 73:561

Holgate S.T., Hardy C., Robinson C. et al (1986) :

The mast cell as a primary effector cell in the pathogenesis of asthma.

J.Allergy Clin Immunol 77:274

Hopp R., Bewtra A., and Nair N. (1987) :

Bronchial reactivity patterns in non-asthmatic parents of asthmatics.

Ann Allergy 61:184

Jackson R., Sears M.R., Beaglehole R., and Rea H.H. (1988) :

International trends in asthma mortality : 1970 to 1985

chest 94(5) : 915

Jeffery P.K., Wardlaw A.J., and Nelson F.C. (1989) :

Bronchial biopsies in asthma. An ultrastructural ,quantitative study and correlation with hyperreactivity

Am Rev Resp Dis 140:1745

Jessner L., Lamont J., Long R., Rollins N., Whipple B., and Prentice N.(1955) :

Emotional impact of nearness and separation for the asthmatic child and his mother

Psychoanal Study Of The Child 10:353

Johnson A.J., Sommer A.R., Stable-forth D.E. and Stewart C.J (1984) :

Circumstances of death from asthma

Br Med J 288:1870

Kabiner, M. (1989) :

Asthma and mast cell activation

J Allergy clin Immunol 83:510

Khallaf N., and Lamb M. (1991) :

In " Case management of acute respiratory infection in children " . A manual for primary health care physicians . 2nd edition . ARI program . Child Survival Project . Ministry of Health . Egypt .

- Khallaf N., Farghaly A., Harrison L. et al (1992) :**
 Egyptian Acute Respiratory Infections Control Programme : Five Governorate Health Facility Survey . Child Survival Project.
 Ministry of Health (Egypt) .with cooperation with AID.In press
- Khan A.U. (1977) :**
 Effectiveness of biofeedback and counter-conditioning in the treatment of bronchial asthma .
 J Psychasom Res 21:97
- Khan A., Borck C. and Steark M. (1974):**
 Non-allergic asthma and conditioning
 J Allergy 32:245
- Kovar M., Serdula M., and Marks J. (1984):**
 Review of the epidemiologic evidence for an association between infant feeding and infant death
 Pediatrics 74:615
- Kowalski M.L., Diddier A. and Kaliner, M.A. (1989) ;**
 Neurogenic inflammation in the airways
 Am Rev Respir Dis 140 : 101
- Kubly L.S., and McClellan M.S. (1984) :**
 Effects of self-care instruction on asthmatic children .
 Issues Comp. Pediatr. Nurs. 7:121
- Laitinen L.A., Heino M., Laitinen A., Kava T. and Haahtela T. (1985):**
 Damage of the airway epithelium and bronchial reactivity in patients with asthma.
 Am. Rev. Resp. Dis 131:599
- Lask B. (1979):**
 Emotional considerations in wheezy child
 J R Soc Med 72:56
- Lask B. (1992):**
 Psychological treatments for childhood asthma
 Arch Dis Child. 66:458
- Lask B. and Matthew D. (1979);**

Childhood asthma .A controlled trial of family psychotherapy.
Arch Dis Childhood 55:116

Lewis C.E., Rachelesky G., Lewis M.A., La Sota M.A. and Kaplan M. (1984):

A randomized trial of A.C.T. (asthma care training) for kids.
Pediatrics 74:478

Liang I., Reidel F., and Yap P. (1982):

Atopy predisposing to acute bronchiolitis during an epidemic of respiratory syncytial virus.

Br Med J 284:70

Liebman R., Minnuchin S., and Baker L. (1974):

The use of structural family therapy in the treatment of intractable asthma

Am J Psychiat 131:535

Liebman R., Minnuchin S., and Rosman B. (1976) :

The role of the family in the treatment of childhood asthma. In Family therapy theory and practice .Edited by Guerin T.J., P 30 .
New York : Gardner .

Lindemann H. (1990):

Prevention of bronchial hyperreactivity in children.

Lung (Suppl):249

Lundgren R. (1977) :

Scanning electron microscopic studies of bronchial mucosa before and during treatment with beclomethazone dipropionate inhalations.
Scand. J. Resp. Dis. 101(suppl.) :179

Lindenskov L., Andersen A.M., Andersen K.V., Herman N., Kristensen F.B., Knudsen V.W., and Nielsen H.K. (1993) :

Preventive health examinations of pregnant women in Denmark .

Ugeskr - Laeger 155:302

- Luparello T., Lyons H.A., Bleeker E.R., and McFadden E.R. (1968) :**
Influence of suggestions on airway reactivity in asthmatic subjects.
Psychosom. Med 30 :819
- Mackdem P.T. (1989) :**
Risk factors and prevention strategies for asthma.
Chest 96:361
- Martin A.J., Landau L.I., and Pheldan P.P. (1981) :**
National history of allergy in asthmatic children followed to adult life
Med. J. Aust. 2:470
- Martinez F., Morgan W., and Wright A. (1988) :**
Diminished lung function as a predisposing factor for wheezing respiratory illness in infants.
N. Engl. J. Med. 319:1112
- Martinez F., Morgan W., Wright A. et al (1991) :**
Initial airway function is a risk factor for recurrent wheezing respiratory illness during the first three years of life.
Am. Rev. Resp. Dis. 143:312
- Marx D., Zofel C., Linden U., Bonner H., Franzen U., and Florin I. (1986) :**
Expression of emotion in asthmatic children and their mothers.
J. Psychosom. Res. 30:609
- Mathi A.A. and Knapp P.H.(1971) :**
Emotional and adrenal reactions to stress in bronchial asthma.
Psychosom. Med. 33:323
- Matthys H. (1990) :**
Definition and assessment of Asthma.
Lung 168 :(Suppl) 51

- Mc Fadden E.R. (1987):**
Exercise-induced asthma :assessment of current etiologic concepts.
Chest 91:515
- MC Fadden E.R. (1989):**
Therapy of acute asthma
J.Allergy Clin.Immunol 84:151
- Mc Fadden E.R.,Kiser R., and Groot D.W.J. (1973) :**
Acute bronchial asthma : relations between clinical and
physiologic manifestations
N. Engl. J. Med 288 : 221
- McIntosh K, Ellis E.F., and Hoffman L.S. (1973) :**
The association of viral and bacterial respiratory infections with
exacerbations of wheezing in young asthmatic children.
J.Pediatr. 82 : 578
- McNabb W.L., Wilson-Pessano S.R., Hughes G.W., and Scamogas P.
(1985) :**
Self-management of children with asthma : Airwise .
Am. J. Public Health 75:1219
- Meares R.A., Mills J.E. and Harvath T.B. (1971) :**
Amitriptaline and asthma.
Med. J. Aust. 2:25
- Mertsola J., Ziegler T., Ruuskanen O. et al (1991) :**
Recurrent wheezy bronchitis and viral respiratory infection
Arch.Dis.Child. 66:124
- Miller B.D. (1987) :**
Depression and asthma : a potentially lethal mixture
J.Allergy Clin. Immunol. 80:481
- Miller B.D., and Strunk R.C. (1989) :**
Circumstances surrounding the deaths of Children due to Asthma :
A case - control study
A J D C 143:1294
- Miller H., and Baruch D.W. (1948) :**

Psychosomatic studies of children with allergic manifestations,
maternal rejection, study of 63 cases
Psychosom. Med. 10:275

Mills P.T.A.E. and Weck D.A.L. (1989):

Dust mite allergens and asthma : A world wide problem
J. Allergy clin. Immunol 79:781

Mills P.T., Longbottom J., and Edwards J. (1987) :

Occupational asthma and rhinitis related to laboratory rats : serum
IgG and IgE antibodies to rat urinary allergen
J. Allergy. clin. Immunol 79:505

Minor T.E., Baker J.W., Dick E.C. (1974) :

Greater frequency of viral respiratory infections in asthmatic
children as compared with their nonasthmatic siblings
J. Pediatr. 85:472

Minor T.E., Dick E.C., Baker J.W. (1976) :

Rhinovirus and influenza type A infections as precipitants of
asthma.
Am. Rev. Respir. Dis. 113:149

Mook J. and Simpson H. (1982):

Outcome of acute lower respiratory tract infection in
infants preliminary report of seven year follow up study
Br. Med.J. 285:333

Moore N. (1965) :

Behavior therapy in bronchial asthma : a controlled study
J. Psychosom. Res. 9:257

Morgan, W.J. and Mortinez F.D. (1992) :

Risk factors for developing wheezing and Asthma in childhood.
Ped. Clinics of N. America 39:1185

Murray A.B. and Morrison B.J. (1986) :

The effect of cigarette smoke from the mother on bronchial responsiveness and severity of symptoms in children with asthma.
J.Allergy.Clin.Immunol 77:575

Murray A.B. and Morrison B.J. (1988) ;

Passive smoking and the seasonal difference of severity of asthma in children
Chest 94:701

Murry M. ,Webb M.S.C.,Callaghan C.,Swarbrick A.S. and Milner A.D.(1992):

Respiratory status and allergy after bronchiolitis.
Arch.Dis.Child. 67:482

National Center for Health Statistics (1990):

Asthma : United States ,1980 -1987
M M W R 39:493

National Heart , Lung and Blood Institute (1984 a) :

Air Power : self management of asthma through group education .
NIH Publication no. 84-2362 . Bethesda , Md : National Institute of Health , 1984 .

National Heart , Lung and Blood Institute (1984 b) :

Airwise : management of asthma through individual education . NIH
Publication no. 84-2336 , Bethesda , Md : National Institute of Health ,
1984 .

National Heart , Lung and Blood Institute (1984 c) :

Open airways / respiro abierto : Asthma self-management program .
NIH Publication no. 84-2365 . Bethesda , Md : National Institute of
Health , 1984 .

National Heart , Lung and Blood Institute (1985) :

Living with asthma : Manual for teaching families the self-management
of childhood asthma . NIH Publication no. 85-2364 . Bethesda , Md :
Nationa Institute of Health , 1984 .

Neijens H.J.(1990):

Determinants and regulating processes in bronchial hyperreactivity.

Lung Suppl. :268

Nelson H.S. (1985):

The atopic diseases
Ann. Allergy 55:441

Ogilvie A. (1962):

Asthma : a study in prognosis of 1000 patients
Thorax 17:183

Osler W. (1892):

The principles and practice of medicine:designed for the
use of practitioners and students of medicine.
New York:D.Appleton and Lo,1892:499 .Quoted from :McFadden E.R
(1989): Therapy of asthma .J. Allergy Clin. Immunol. 84 : 151 .

Parcel G.S. , Nader P.R. and Tierman K. (1980) :

A health education program for children with asthma .
Dev. Behav. Pediatr. 1:128

Parker S.R. (1987);

The future role of asthma self-management
J.Allergy Clin.Immunol 80:511

Parker J. E. and Parker K. (1979) :

Assessment of the socioeconomic status . In textbook of preventive
and social medicine . 7th edition . Ed. Park J.E. and Park K. ,
Hessero . Barnarsides . Bhanat Publishers 1268 . Napier Town ,
P 81.

Peat J.K.,Britton W.J.,Salome C.M., and Woolclock A.J. (1987) :

Bronchial hyperresponsiveness in two populations of Australian
school-children :II.Relative importance of associated factors.
Clin Allergy 17:283

Persson C.G.A. (1986):

Role of plasma exudation in asthmatic airways
Lancet 2:1126

Persson C.G.A. (1986);

The role of microvascular permeability in the pathogenesis of

asthma.

Eur.J.Respir Dis 68 (Suppl 144):190

Pierson W.E.(1988):

Exercise-induced bronchospasm in children and adolescents.
Pediatr. Clin. N. Am. 35:1031

Pinkerton P.(1967):

Correlating physiologic with psychodynamic data in study and management of childhood asthma
J.Psychosom. Res. 11:11

Phillip R.L., Wilde G.J.S., and Day J.H.(1972):

Suggestion and relaxation in asthmatics
J.Psychosom. Res. 16:193

Platts-Mills T.A.E.(1993):

Allergen-specific treatment of Asthma:III
Am.Rev.Resp.Dis. 148:553

Plutchik R., Williams M.H., Jerrett I., Karasu T.B., and Kane C.(1978):

Emotions, personality and life stresses in asthma
J.Psychosom.Res. 22:425

Pollart S.M., Chapman M.D., Fiocco G.P. et al (1989):

Epidemiology of acute asthma: IgE antibodies to common inhalant allergens as a risk factor for emergency room visits.
J.Allergy clin.Immunol 83:875

Pollart S.M., Reid M.J., and Fling J.A. (1988):

Epidemiology of emergency room asthma in northern California: Association with IgE antibody to ryegrass pollen.
J.Allergy clin Immunol 82:224

- Prendville A., Green S., and Silverman M. (1987):***
 Airway responsiveness in wheezy infants. Evidence for functional beta adrenergic receptors.
 Thorax 42:100
- Purcell K. (1963):***
 Distinctions between subgroups of asthmatic children: children's perceptions of events associated with asthma
 Pediatrics 31:486
- Purcell K., Bernstein C., and Bukantz S.C. (1961):***
 A preliminary comparison of rapidly remitting and persistently steroid dependent asthmatic children
 Psychosom. Med. 23:305
- Rachelefsky G.S. (1987):***
 Review of asthma self-management programs.
 J. Allergy Clin. Immunol 80:507
- Rachelefsky G.S., Katz R.M. and, Sigel S.C. (1984):***
 Chronic sinus disease with associated reactive airway disease in children.
 Pediatrics 73:526
- Rea H.H., Sears M.R, and Beaglehole R. (1987):***
 Lessons from the national asthma mortality study: circumstances surrounding death.
 NZ. Med. J. 100:10
- Rees L. (1963):***
 The significance of parental attitudes in childhood asthma.
 J. Psychosom. Res. 7:181
- Reiff D.B., Choudry N.B., Pride N.B., and Ind P.W. (1989):***
 The Effect of prolonged submaximal warm-up exercise on exercise-induced asthma.
 Am. Rev. Respir. Dis. 139:479
- Richards W. (1986):***
 Allergy, asthma and school problems

Journal of School Health 56:151

Richards W. (1989):

Hospitalization of children with status Asthmaticus :A Review
Pediatrics 84:111

Richardson J.B. (1979):

Nerve supply to the lung
Am.Rev.Resp.Dis. 119:758

Roche W.R., Beasley R., Williams J.H. et al (1989):

Subepithelial fibrosis in the bronchi of asthmatics
Lancet 1:520

Roldaan A.C., and Manswral N. (1982):

Viral respiratory infections in asthmatic children staying in a
mountain resort.
Eur.J.Respir.Dis. 63:140

**Ronchetti R., Bonci E., Cutrera R., De Castro G., Indinnime L., Medulla
F., Toncredi G. and Martinez F.D. (1992):**

enhanced allergic sensitization to parental smoking
Arch.Dis.child. 67:496

Ronchetti R., Bonci E. and Martinez F.D. (1990):

Passive smoking in childhood-tobacco smoke.
Lung Suppl.:313

Ronchetti R., Marci F., Ciofetta G. et al (1990):

Increased serum immunoglobulin E (IgE) and increased prevalence
of eosinophilia in nine-year old children of smoking parents
J.Allergy Clin.Immunol 86:400

Rubin D.H. , Leventhal J.M. , Sadock R.T. et al (1986) :

Educational intervention by computer in childhood asthma : a
randomized clinical trial testing the use of a new teaching intervention
in childhood asthma .
Pediatrics 77:1

Ryan G., Latimer K.M., Dolovich J. and Hargreave F.E.(1982):

Bronchial responsiveness to histamine :relationship to

diurnal variation of peak flow rate, improvement after bronchodilator, airway caliber
Thorax 37:423

Salome C.M., Peat J.K., Britton W.J. and Waalcock A.J. (1987):

Bronchial hyperresponsiveness in two populations of Australian school-children: I. Relation to respiratory symptoms and diagnosed asthma
Clin. Allergy 17:271

Samet J., Tager I. and Speizer F. (1983):

The relationship between respiratory illness in childhood and chronic air-flow obstruction in adulthood
Am. Rev. Resp. Dis. 127:508

Schnall R.P., and Landau L.I. (1980):

Protective effects of repeated short sprints in exercise-induced asthma
Thorax 35:828

Schwartz J., Gold D., and Dockery D. (1990):

Predictors of asthma and persistent wheeze in a national sample of children in the United States. Association with social class, perinatal events and race.
Am. Rev. Resp. Dis. 142:555

Sears M.R. (1987):

Are deaths from asthma really on the rise?
J. Resp. Dis. 8(8) :39

Sears M., Burrows B., Flannery E. et al (1991):

Relation between airway responsiveness and serum IgE in children with asthma and in apparently normal children.
N. Engl. J. Med. 325 :1067

Sears M.R., Rea H.H., and Fenwick J. (1986):

Deaths from asthma in New Zealand
Arch. Dis. child. 61:6

Sibbald B., Horn M., and Brain A. (1980):

Genetic factors in childhood asthma

Thorax 35:671

Simon R.A.(1989):

Sulfite challenge for the diagnosis of sensitivity.
Allergy Proc. 10:357

Sireman P.,Friday G.A., and Gira C. (1981):

Teaching self-management skills to asthmatic children and their parents in an ambulatory care setting.
Pediatrics 68:341

Sly R.M.(1991):

The disquieting data on asthma morbidity and mortality.
Current Issues in Allergy and Immunology 2:14

Smith L.J.(1991):

The role of platelet activating factor in asthma
Am.Rev.Resir.Dis. 143:100

Smith H.R. and Henson P.M.(1987):

Mediators of asthma.
Seminars in Respir.Medicine 8:287

Speight A.N.P.,Lee D.A. and Hey E.M. (1983):

Underdiagnosis and undertreatment of asthma in childhood
B. M. J. 286:1253

Sperling M.(1968):

Asthma in children - An evaluation of concepts and therapies.

J.Am.Acad.child.Psychiat. 7:44

Sporik R., Holgate S., Platts-Mills T. et al (1990):

Exposure to house-dust mite allergen and the development of asthma in childhood
N.Engl.J.Med. 323:502

Steen S.N. (1976):

The effects of psychotropic drugs on respiration.
Pharmacol.Ther. 2:717

Stein R., Canny G.J., Bohn D.J., Reisman J.J., and Levison H.(1989):

Severe acute asthma in a pediatric intensive care unit: six year's Experience
Pediatrics 83:1023

Stephoe A., and Holmes R.(1985):

Mood and pulmonary function in adult asthmatics: a pilot self-monitoring study
Br.Med.Psychol. 58:87

Stillwell P.C.(1993):

Keeping ahead of childhood asthma
Clin. Pediatr. 32:97

Straker N. and Tamerin J. (1974):

Aggression and childhood asthma: a study in a natural setting
J.Psychosomatic Research 18:131

Strunk R.C., Mrazek D.A., Wolfson S., Fuhrmann G.S., and La Breque J.F.(1985):

Physiologic and psychological characteristics associated with deaths due to asthma in childhood.
J.A.M.A. 254:1193

Sugihara H., Ishihara K. and Noguchi H.(1965):

Clinical experience with amitriptyline (Tryptarol) in the treatment of bronchial asthma.
Am.Allergy 23:422

- Sultz H., Feldman J.G., Schlesingen E.R. and Mosher E.M. (1970):**
An effect of continued exposure to air pollution on the incidence of chronic childhood allergy disease.
Am.J.Public Health 60:891
- Tager I.B. (1989):**
Health effects of "Passive smoking in children".
Chest 96:1161
- Tal A., Pasterkamp H., Serrette C., Leahy F. and Chernick V. (1984):**
Response to cold air hyperventilation in normal and in asthmatic children.
J.Pediatr. 104:516
- Taylor S.L., Bush R.K., Selner J.C. et al (1988):**
Sensitivity to sulfited foods among sulfite-sensitive subjects with asthma.
J.Allergy Clin.Immunol 81:1159
- Teiramaa E. (1978) a :**
Psychic disturbances and severity of asthma.
J.Psychosom.Res. 22:401
- Teiramaa E. (1978) b :**
Psychosocial and psychic factors in the course of asthma.
J.Psychosom.Res. 22:121
- Teiramaa E. (1978) c :**
Psychic disturbances and duration of asthma.
J.Psychosom.Res. 22:127
- Teiramaa E. (1979) a :**
Asthma, psychic disturbances and family history of atopic disorders
J.Psychosom.Res. 23:209

Teiramaa E. (1979) b :

Psychic factors and the inception of asthma.
J.Psychosom.Res. 23:253

Teiramaa E.(1981):

Psychosocial factors, personality and acute-insidious asthma.
J.Psychosom.Res. 25:43

Tepper R., Morgan W., Cota K. et al (1986):

Physiologic growth and development of the lung during the first year of life.
Am.Rev.Resp.Dis. 134:513

Thompson W.L., and Thompson T.L.H. (1985):

Psychiatric aspects of asthma in adults.
Adv.Psychosom.Med. 14:330

Tseng R.Y.M., and Li C.K. (1990):

Low level atmospheric sulphur dioxide pollution and childhood asthma.
Ann.Allergy 65:379

Usherwood T.P., Scrimgeour A., and Harber J. H. (1990) :

Questionnaire to measure perceived symptoms and disabilities in asthma .
Arch. Dis. Child. 65:779

Van De Graaf E., Out T.A., Roos C.M. et al (1991):

Respiratory membrane permeability and bronchial hyperreactivity in patients with stable asthma.
Am.Rev.Respir.Dis. 143:362

Van Der Volk J.M. (1960):

Comparison of the social setting and behaviour of patients with bronchial asthma, Coronary occlusion and healthy subjects.
Adv.Psychosom.Med. 1:284

Wagner E.M., Liu M.C., Welmann G.G., Permutt S., and Bleecker E.R. (1990):

Peripheral lung resistance in normal and asthmatic subjects
Am. Rev. Respir. Dis. 141:584

Walker A. (1987):

Pathophysiology of intestinal uptake and absorption of antigens in food allergy.
Ann.Allergy 59:7

Walker C., Virchow J.C., Bruijnzeel P.L.B. et al (1991):

T-cell subsets and their soluble products regulate eosinophilia in allergic and non-allergic asthma.
J.Immunol 146:1829

Wang J.Y., Hsiue and , Chen H.I. (1992):

Bronchial responsiveness in an area of air pollution resulting from wire reclamation.
Arch.Dis.Child. 67:488

Wardlaw A.J., Dunnette S., and Gleich G.J. (1988):

Eosinophils and mast cells in bronchoalveolar lavage in subjects with mild asthma: Relationship to bronchial hyperreactivity.
Am.Rev.Respir.Dis. 137:62

Warner J.O., Gotz M., Landau L.I., Levison H., Milner A.D., Pedersen S., and Silverman M. (1989):

Management of asthma: a Consensus statement.
Arch.Dis.Child. 64:1065

Warner J.O., Neljens H.J., Landau L.I. et al (1992):

Asthma: a follow up statement from an international pediatric asthma consensus group.
Arch.Dis.Child. 67:240

Weiss J.H. (1966):

Mood states associated with asthma in children.
J.Psychosom.Res. 10:267

Weiss J.H. (1981) :

Superstuff : self-management educational program for childhood

asthma , Vol 2 . Bethesda , Md : National Institute of Allergy and Infectious Diseases 2 :273

Weiss K.B., Gergen P.J. and Hodgason T.A. (1992):

An economic evaluation of asthma in the United States.
N.Engl.J.Med. 326:862

Weitzman M., Gortmaker S., Kleinwalker D. et al (1990):

Maternal smoking and childhood asthma.
Pediatrics 85:505

Whiteman N., West D., Brough F.R. and Welch M.C. (1975):

A study of a self-care rehabilitation in Pediatric asthma
Health Educ. Q 12:333

Widdicombe J.G., Kant D.C. and Nadel J.A. (1962):

Mechanism of bronchoconstriction during inhalation of dust.
J.Appl.Physiol. 17:613

Wikran R., Faleide A., Blakor R.M. (1978):

Communication in the family of the asthmatic child.An
experimental approach.
Acta Psychiat.Scand. 57:11

Wilson-Pessano S.R. and McNabb W.L. (1985) :

The role of patient education in the management of childhood asthma
Prevent Med 14:670

Wilson-Pessano S.R. and Mellins R.B. (1987) :

Workshop on asthma self-management
J. Allergy Clin. Immunol 80:487

Wilson N.M., Phagoo S.B. and Silverman M. (1992):

Atopy, bronchial responsiveness, and symptoms in wheezy 3 year olds.
Arch.Dis.child 67:491

Wood D.L., Hawyard R.A., and Corey C.L. (1990):

Access to medical care for children and adolescents in the United States.
Pediatrics 86:666

Wood R., Chapman M., and Atkinson N. (1989):

The effect of cat removal on allergen content in household dust samples.
J.Allergy Clin.Immunol 83:730

Wright A., Halberg C., and Martinez E. (1989):

Breastfeeding and lower respiratory tract illness in the first year of life.
Br.Med.J.299:946

Wright A., Taussig L., Ray C. et al (1991):

Relationship of Parental smoking to wheezing and nonwheezing lower respiratory tract illness in infancy.
J.Pediatr. 118:207

Wutrich B., Baumman E., and Fries R. (1981):

Total and specific IgE (RAST) in atopic twins.
Clin.Allergy 11:147

Yellowlees P.M. and Kalucy R.S. (1990):

Psychobiological aspects of asthma and the consequent research implications.
Chest 97:686

Yellowlees P.M. and Ruffin R.E.(1989):

Psychological defences and coping styles in patients following a life threatening attack of asthma.
Chest 95:1298

Yellowlees P.M, Alpers J.H., Bowden J.J. and Ruffin R.E. (1987):

Psychiatric morbidity in patients with chronic airflow obstruction.
Med.J.Aust. 146:305

Yorkston N.J.,McHugh R.B.,Brady R.,Serber M. and Sergeant H.G.S. (1974):

Verbal desensitization in bronchial asthma.
J.Psychosom. Res. 18:371

Young S.,Le Souf P.,Geelhoed G. et al (1991):

The influence of a family history of asthma and parental smoking on airway responsiveness in early infancy.
N.Engl.J.Med. 324:1168

Zimmerman B.,Chambers C.,and Forsyth C. (1988):

Allergy in asthma II.The highly atopic infant and chronic asthma.
J.Allergy Clin.Immunol 81:71

Zweiman B.,Glott R.I. and Atkins P.C. (1976):

Histologic studies of human skin test responses to ragweed and compound 48/80.III.Effects of alternate-day steroid therapy.
J.Allergy Clin.Immunol 58:657

Zweiman B.,Schoenwetter W., and Pappano J. (1971):

Patterns of allergic respiratory disease in children with a past history of bronchiolitis.
J.Allergy Clin.Immunol. 48:283

APPENDIX

بسم الله الرحمن الرحيم

- لرشادات و نصائح لامهات و نياء الاطفال الذين يعانون من ازمات ربوية :
- أ- العمل على حماية الطفل من العوامل المسببة للحساسية و حدوث الازمة بصفة عامة، مثال ذلك :
- تجنب نخان السجائر و منع تدخين الاب او الام او الضيوف امام الطفل او فى حجرة يدخلها الطفل حيث ان نخان السجائر يعلق على الحوائط .
 - تجنب التهابات الحلق و الانف و علاجها مبكرا فى حلة حدوثها و تجنب تقبيل الاطفال اثناء دور البرد او الاتفلونزا خاصة فى الوجه و تجنب مخالطة الاطفال المصابين بالبرد او الاتفلونزا .
 - منع تعرض الطفل للتيارات الهوائية المباشرة خاصة بعد خروجه من الاستحمام و تجنب الاستحمام بماء بارد لعدم تعرضه لنزلات البرد و عدم فتح شباك الحجره امام وجه الطفل مباشرة خاصة فى الصباح البارد اثناء الشتاء و تجنب فتح الثلجة و الوقوف امامها لفترة طويلة .
 - تدريب الطفل المصاب بالبرد على وضع منديل على الانف و الفم اثناء السعال او العطس .
 - تجنب للتعرض للروائح النفاذة مثل البويات و عند بياض او دهان الشقة او الدوكو او عوادم السيارات او الايروسول او المواد المضادة للحشرات بصفة عامة و كذلك بياور للجاز و دفايات الفحم او الخشب او استنشاق الاتربة او رائحة زيت للقلى بالمطبخ .
 - تجنب لمسك بالقرب من مصانع الاسمنت او المسابك او قمان الطوب الاحمر او حظائر الحيوانات او مزارع للطيور .
 - تجنب للتعرض للعواصف و الرياح الترابية و حبوب اللقاح التى تزداد خلال شهرى مارس و ابريل و يمكن وضع قطعة من الشاش على الانف عند الضرورة .
 - عدم استعمال موكيت للارضية (على الاقل فى غرفة الطفل) وعدم نوم الطفل على الارض او السجاد او على اثاث الصالون و يفضل استعمال مئتمع للارضية او حصير للعب الطفل مع تنظيفها باستمرار .
 - يفضل استخدام المكثس للكهربائية الحديثة و اذا لم يمكن فيجب عدم الكنس او التنفيض او اثاره الغبار او الاتربة اثناء وجود الطفل و يمكن رش بعض المياه على الارضية لتجنب تصاعد الاتربة .
 - تنظيف للشبابيك و الستائر و غسلها باستمرار لعدم تراكم الاتربة و ازالة للكراميب او الكتب او المجلات للقديمة و التى تحتوى على كميات كبيرة من التراب او تنظيفها باستمرار .
 - تجنب وجود الفراء او الحيوانات المنزلية مثل القطط او الكلاب و عدم تربية الحمام او البط او الاوز او الدجاج او الارانب و التى قد يتسبب ريشها او فرانها فى حدوث الازمات الربوية .
 - منع للقفز على السرير او اللعب تحت السرير لتجنب للتعرض للتراب و العناية بتهوية غرفة الطفل لتجنب حشرة الفرائش او عة للفرائش .
 - تجنب المجهود العضلى الشديد مثل الجرى بشدة او اللعب على المراحيج فى ارضية ترابية وتجنب صعود السلم بسرعة خاصة الادوار العليا و المرتفعة او تعرضه للاماكن المغلقة للرطوبة فى الادوار الارضية .

ب - على الام الاهتمام بالنقاط الآتية :

- الاهتمام بتغذية الطفل التغذية السليمة بتوافر العناصر الأساسية فى الغذاء مثل البروتينات و النشويات و ادهنيات بالنسب الصحية لكن عنصر للمساعدة على النمو و التشجيع على شرب السوائل المفيدة مثل عصائر الليمون و البرتقال و الحلبة و الينسون و التليو و الكركديه بالاضافة الى اللبن و العسل الابيض و الزبادى .
- يجب ملاحظة بعض الاطعمة التى قد تؤدى الى حدوث الازمة و تجنبها ، مثال ذلك الاطعمة ذات الالوان الصناعية المحفوظة و مكسبات الطعم للون و للنكهة و الرائحة الصناعية و التى يقبل عليها كثير من الأطفال . و كذلك بعض الاطعمة الاخرى مثل الفراولة او السمك او الشيكولاتة و ليس بالضرورة منع الطفل من هذه الاطعمة اذا لم تلاحظى حدوث الازمة بعد تناولها .
- اتعرف على قدرات الطفل على القيام بالتمارين الرياضية و تشجيعه على القيام بها فى جو صحى سليم و ممارسة التمرينات الصدرية بغرض زيادة كفاءة الرئتين عن طريق الآتى :
لعبة الفقاع الهوائية ، نفخ البالونات و اللعب بها ، نفخ كرة التنج بنج على السفرة .
- تجنب الانفعالات الشديدة مثل الخوف او الفزع او الكاء بشدة و معايشة الطفل فى جو من السعادة و شهو العائلى و عدم تعرضه لمشاجرات او خلافات حادة بين الاب و الام ، و الحرص على تجديد نشاط الطفل و تحسين حالته النفسية بالنزهة فى مكان هادئ نظيف خالى من الاتربة .
- يجب على الام الاهتمام بالتواحي الآتية :
اعطاء الطفل كفايته من النوم ، الاهتمام بالتغذية السليمة ، ممارسة الرياضة الملائمة ، الراحة التامة أثناء الازمة ، تحليل لسبب الازمة السابقة و محاولة معرفة اسبابها و تجنبها ، و معرفة العلاجات التى سفرت عن نتيجة طيبة و كذلك العلاجات التى لم يتحسن بعدها الطفل .
- على الام مراعاة الآتى :
تهوية البطاطين و المخدات باستمرار و وضعها فى الشمس يوميا تقريبا .
غسل ملابس الطفل بعد تخزينها لفترة طويلة و الوقاية من عتة الملابس .
غسل اكياس المخدات و ملايات السرير و غليها بصفة دورية و الاهتمام بكيها و يفضل ان تكون قطنية .
تجنب استخدام الملابس الداخلية الصوفية و التى قد تسبب الحساسية و يفضل استعمال ملابس قطنية .
فرش مشمع او مفرش بلاستيك بين ملاية السرير و المرتبة التى ينام عليها الطفل .
شرب الطفل ولو مقدار بسيط من الماء قبل نزوله من الشقة خاصة فى الصباح و الجو بارد و فى الشتاء
- الاهتمام بنظافة الشقة بصفة عامة و خاصة الحمامات و المطابخ و الارضيات و يجب تهويتها و تنظيفها باستمرار ، و التخلص من للحشرات المنزلية مثل الصراصير و التى قد تكون من مسببات الحساسية للطفل خاصة فضلاتها او بعد تحللها .

الاكتشاف المبكر لحالة الازمة و كيفية التعامل معها :

- يمكنك ان تجنبى طفلك حدوث الازمة عن طريق التعرف على علامات الازمة و التعامل معها مبكرا ، و تختلف العلامات المبكرة من طفل لآخر ، و على الام ان تتعرف على العلامات الخاصة بابنها ، و من الاعراض المبكرة لحدوث الازمة ما يلى :
- اعراض خاصة بالجهاز التنفسى مثل :
الكحة (وهى من اهم الاعراض) ، العطس ، سيلان الانف ، النهجان ، زيادة سرعة التنفس .
- اعراض عامة تظهر على الطفل مثل :
فقدان الشهية ، شحوب و تغير فى لون الوجه ، ظهور زرقان حول العينين ، الهرش او حكة . الم بلبطن حرقان بالحلق ، الشعور بالثعب و الازهاق ، دموع بالعين ، ارتفاع درجة الحرارة ، زيادة ضربات القلب ، جفاف الفم .
- الاعراض السلوكية و الانفعالية مثل :
ظهور النرفزة او العصبية او القلق او اللامبالاه ، من السهل استثارة الطفل ، اضطراب النوم نتيجة لكحة خاصة اثناء الليل ، الشعور بالضيق و الزهقان و عدم الراحة .

فى حالة ظهور الاعراض المبكرة ، يجب عمل الاتى :

- لراحة التامة و محاولة طمأنة الطفل و تهدئته نفسيا .
- شرب سوائل دافئة كالتي نكرت من قبل .
- محاولة لتحكم فى عضلات التنفس ، والعمل على ارتخاء العضلات تدريجيا لمحاولة التحكم فى الازمة .
- الاهتمام بتحثة النفسية المستقرة و الظهور بمظهر هادئ امام الطفل لتعطيه الطمأنينة و الهدوء ، و تجنبى الانفعال او ظهور مشكل او مشاحنات بين الاب و الام .
- اعطاء العلاج كما تم وصفه من قبل الطبيب .
- تذكرى ان معظم الازمات تاتى بصورة تدريجية ، و الخطوات التى تتخذ مبكرا لها اثر فعال فى منع الازمة .
- فى حثه عدم تحسن الحثه او ظهور اى من الاعراض الاتية :
- لزياد الكحة مع تحرك فتحتى الانف مع التنفس ، زيادة للنهجان ، زيادة سرعة التنفس ، للتنفس من الفم ، ظهور تزييق بالصدر ، اهراز العرق ، تداخل ما بين الضلوع ، زيادة المجهود اثناء الشهيق ، او سوء الحثه العامة .
- عند ظهور اى من الاعراض السابقة ، فيجب للتوجه فورا لى المستشفى او الطبيب لان طفلك من الممكن ان يعانى من ازمة ربوية شديدة .

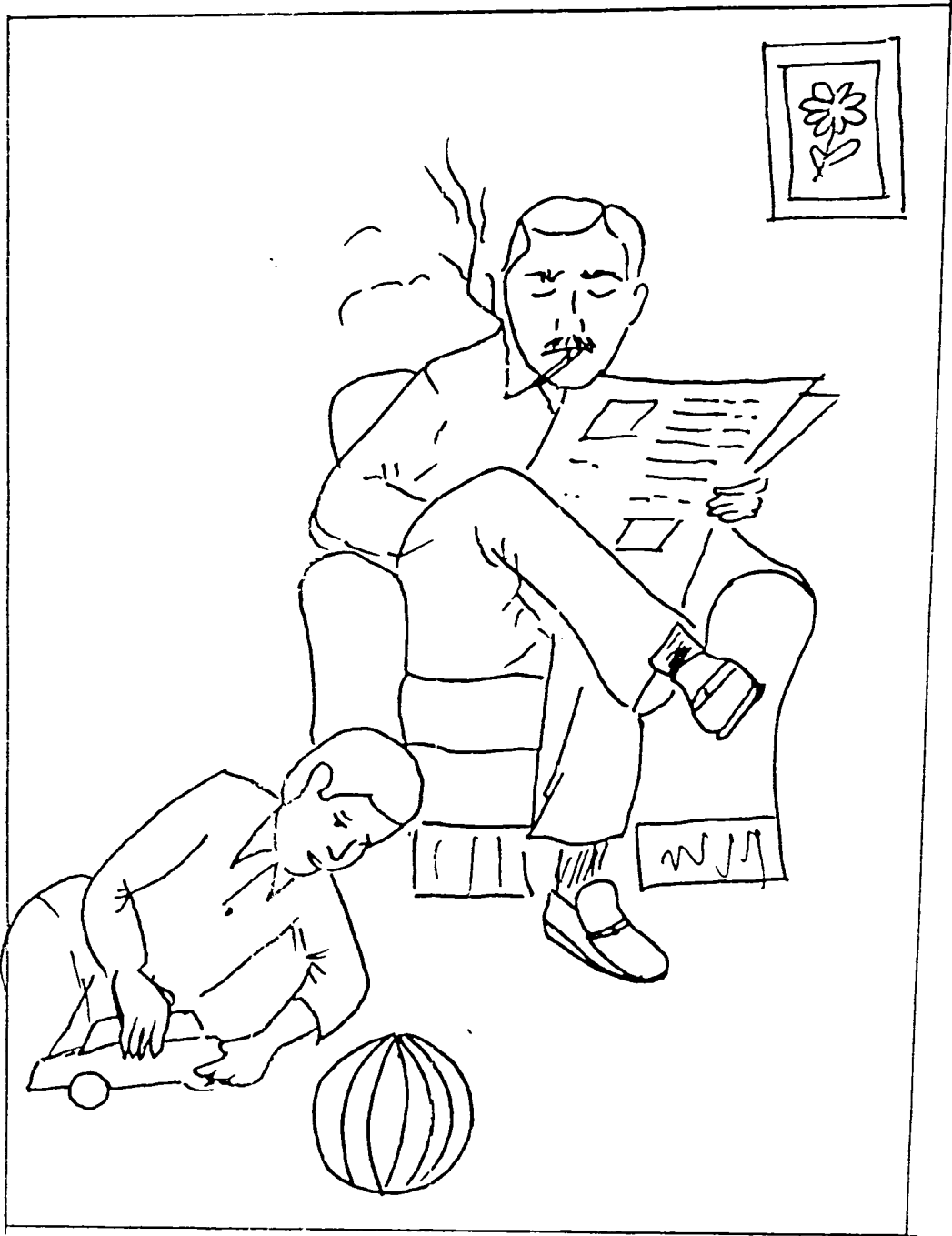
مع خالص الامنيات بتمام الشفاء

Examples of the drawings and illustrations

shown to the asthmatic children and their families :



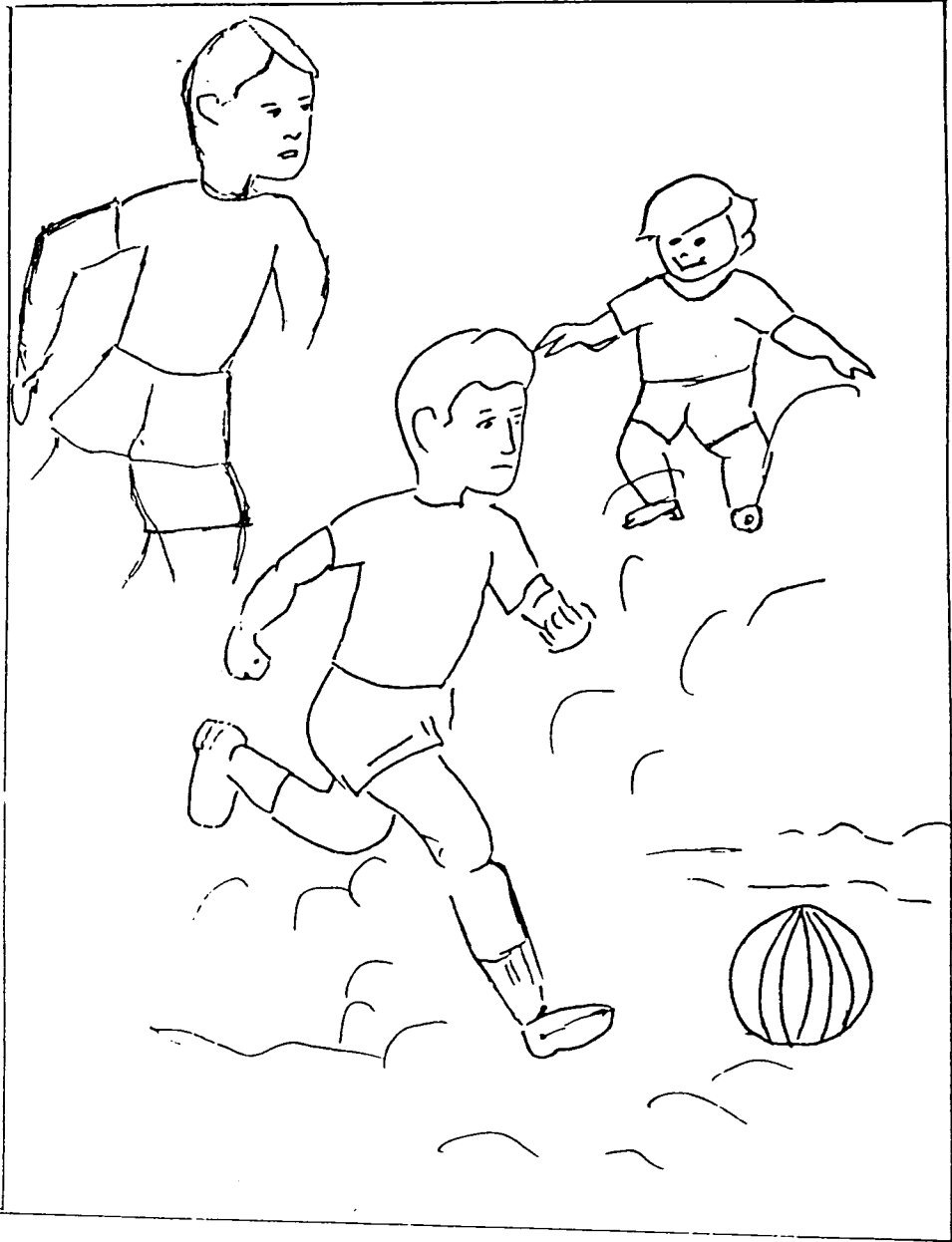
تجنب التعرض لتيار هواء مباشر خاصة في الصباح اثناء الشتاء



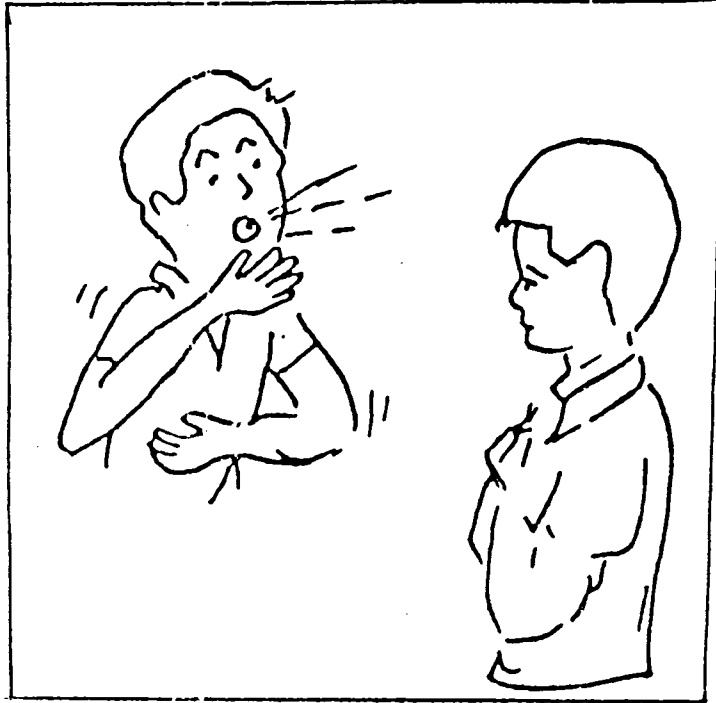
تجنب التدخين بالشقة أو امام الطفل حيث ان دخان السجائر يعلق بالجو لفترة طويلة و يؤدي الى ظهور الازمة



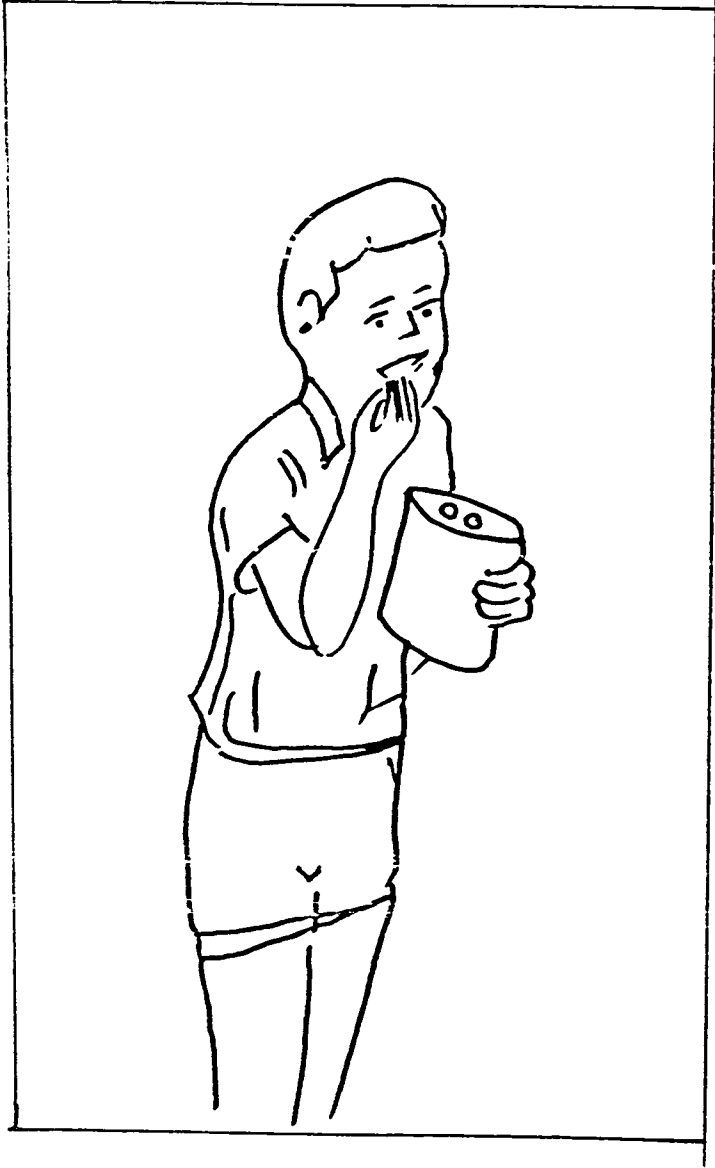
تجنبى كنس أرضية الشقة امام الطفل و يمكن كنسها بعد خروجه
 او استعمال مكاتس كهربائية شافطة للترية



تجنب اللعب بالجهاد في ملاعب ترابية و يفضل عمل فترة تسخين و اللعب
في ملاعب نجيلية خالية من التراب



تجنب ملاصقة الأشخاص المصابين بالبرد أو الانفلونزا



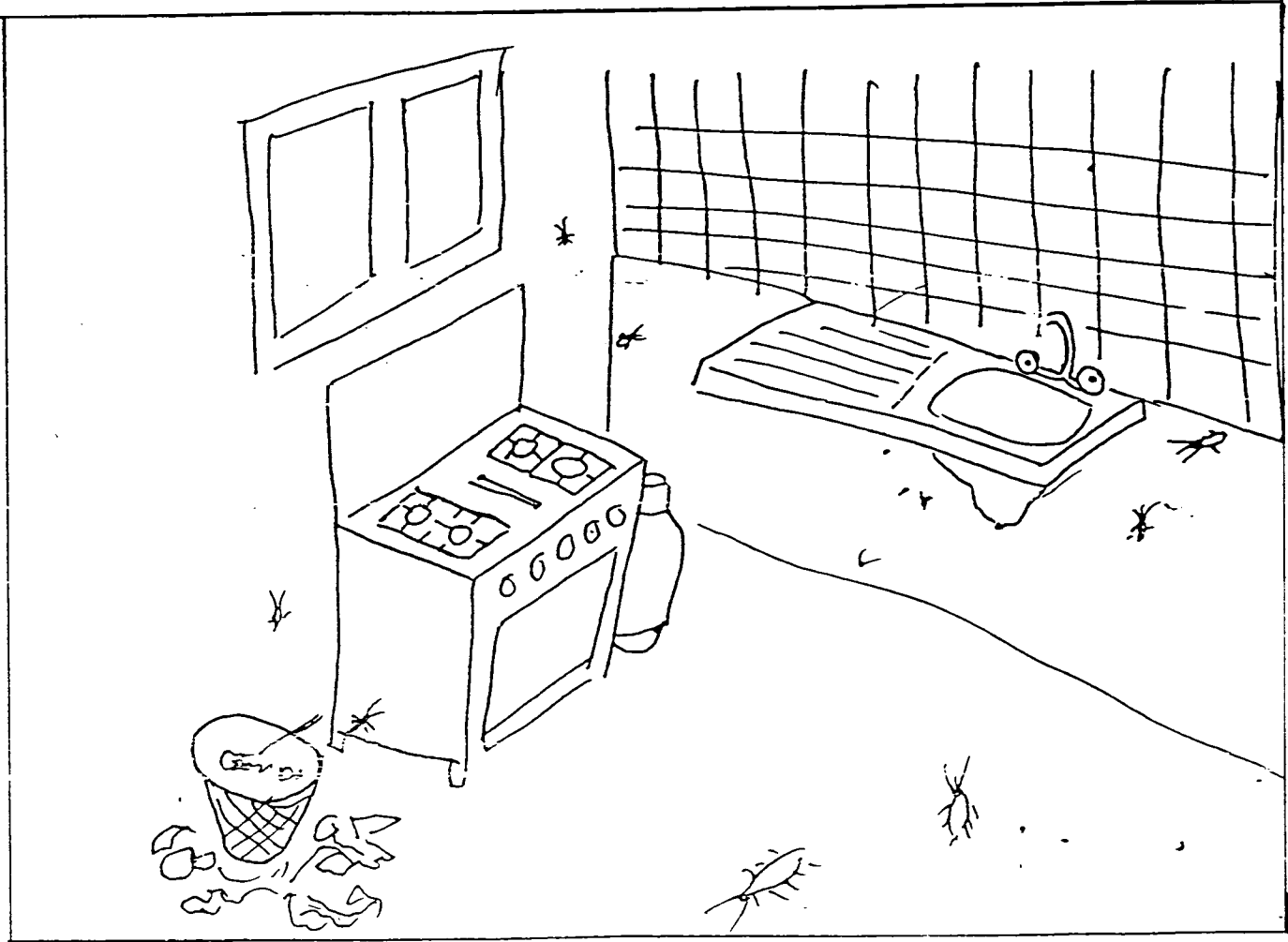
تجنب الاطعمة المحفوظة والتي تحوى مضافات النكهة و الرائحة و مواد
حافطة صناعية تساعد على ظهور الازمة ويفضل تناول الخضروات
والفواكه الطازجة



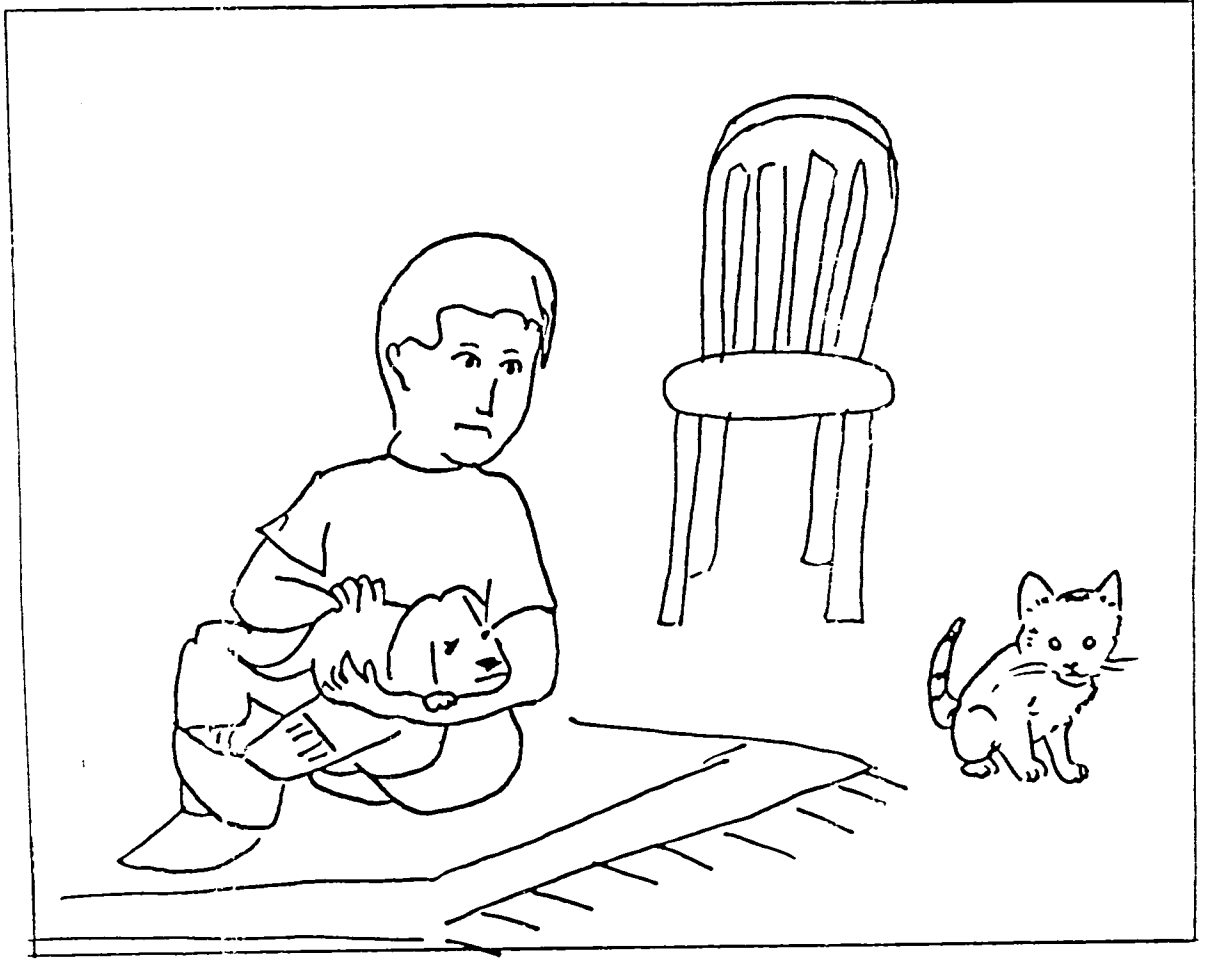
تجنبي المشاحنات امام الطفل و توبيخه عند حدوث الازمة ووفرى له جو عاقل
هادئ و نظيف



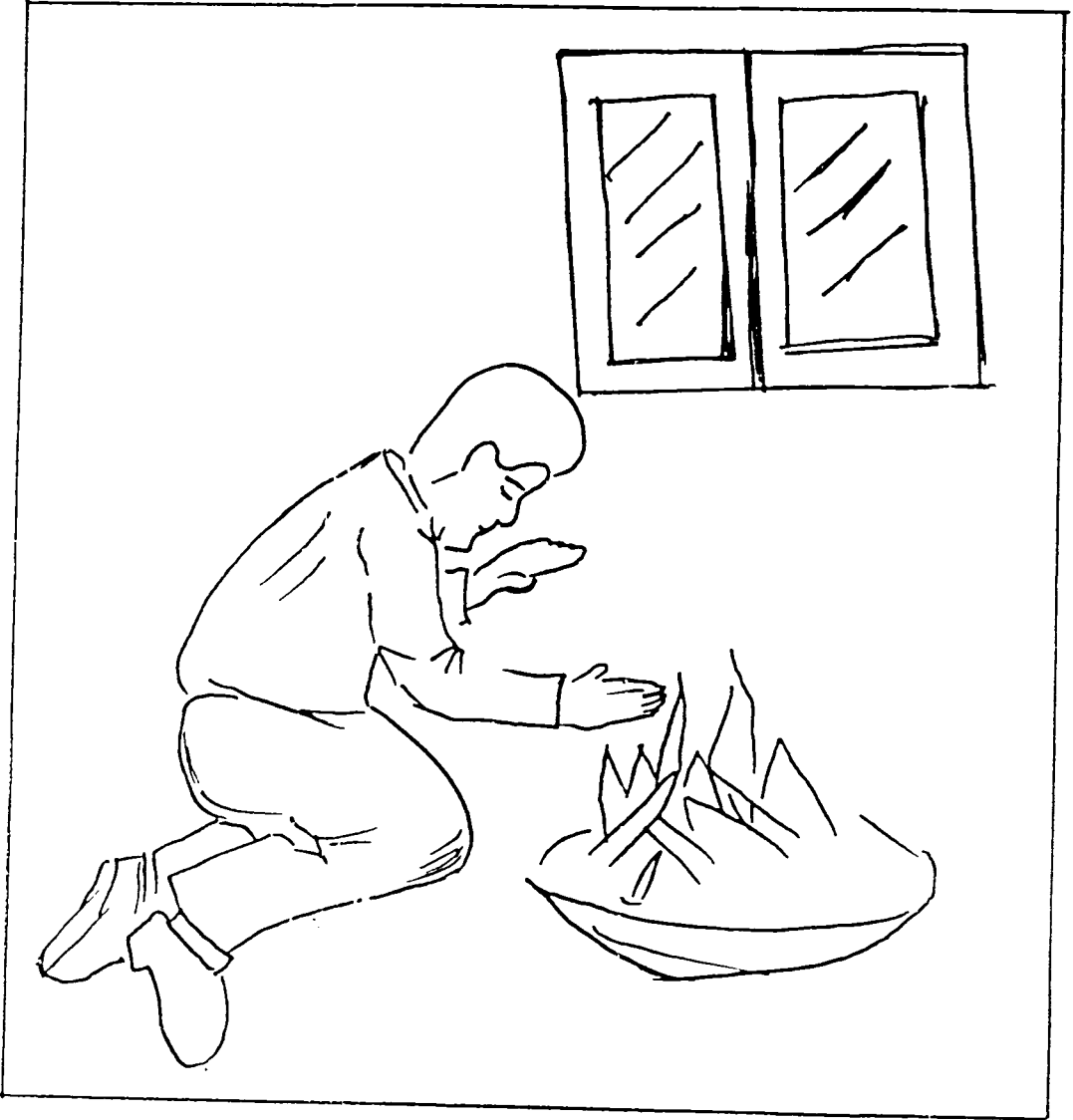
اعتنى بتهوية الشقة خاصة المطبخ واثناء قلي الزيت فالروائح و العادم
الناتج من القلي تساعد على ظهور الازمة



اعتنى بنظافة الشقة خاصة المطبخ و الحمام و تجنبي و جود الصراصير
او الفئران حيث انها تؤدي الى حدوث دور الازمة



تجنب تربية الحيوانات كالقطط او الكلاب خاصة القطط السيامي بالمشقة
حتى لا تسبب نور الازمة



تجنب التعرض للدخان و الروائح النفاذة و البويات خاصة أثناء دهان الشقة



يفضن و ضع شاش او منديل امام الانف اثناء رياح الخمسين او في حالة
وجود اتربة علقه بالجو

QUESTIONNAIRE FOR ASTHMATIC CHILDREN
AND THEIR PARENTS

PATIENT ID _____ DATE OF FIRST INTERVIEW <dd/mm/yy>
 PLACE OF INTERVIEW _____ NAME OF INFORMANT _____
 NAME OF CHILD _____ AGE AT INTERV. ##.## YEARS
 SEX <A> _____ BIRTHDAY <dd/mm/yy>
 PATIENT TYPE : THIS VISIT IS THE PRIMARY OR RECURRENT VISIT ? <A>
 ADDRESS _____ TEL NO.: <phonenum>
 ARE YOU LIVING IN URBAN OR RURAL AREA ? <Y>
 HEIGHT : ###.# CM WEIGHT : ##.# KG
 BIRTH ORDER : # SIBLINGS : #
 PERIOD OF BREAST FEEDING : #
 1- 0-- MONTH 2- 0--6 MONTH
 3- 6--12 MONTH 4- MORE THAN ONE YEAR
 VACCINATION : #
 1-NONE 2-NOT COMPLETED 3-COMPLETED
 FATHER EDUCATION : #
 1- ILLETRATE 2- CAN READ AND WRITE
 3- PRIMARY 4- PREPARATORY
 5- SECONDARY 6- UNIVERSITY
 FATHER OCCUPATION : #
 1- PROFESSIONAL 2- MANAGERIAL
 3- SKILLED UNMANUAL 4- SKILLED MANUAL
 5- UNSKILLED 6- UNEMPLOYMENT
 NAME OF MOTHER : _____
 MOTHER EDUCATION : #
 1- ILLETRATE 2- CAN READ AND WRITE
 3- PRIMARY 4- PREPARATORY
 5- SECONDARY 6- UNIVERSITY
 MOTHER OCCUPATION : #
 1- PROFESSIONAL 2- MANAGERIAL
 3- SKILLED UNMANUAL 4- SKILLED MANUAL
 5- UNSKILLED 6- HOUSEWIFE
 IS THE CHILD IN SCHOOL <Y> IF YES :
 NAME OF SCHOOL : _____
 STAGE OF EDUCATION : _____
 CHILD SCHOOL PERFORMANCE : #
 1- POOR 2- ACCEPTABLE
 3- GOOD 4- VERY GOOD
 5- EXCELLENT
 HIS ORDER IN SCHOOL : #
 1- FIRST 2- OF FIRST FIVE
 3- OF FIRST TEN 4- OTHERS
 SPORT PRACTICE : #
 1- NONE 2- SOMETIMES
 3- REGULAR 4- OTHERS

FAMILY RELATIONS : #

- | | |
|-------------|-------------|
| 1- GOOD | 2- BAD |
| 3- DIVORCED | 4- SEPARATE |
| 5- OTHERS | |

WHAT IS THE EFFECT OF CHILD'S ASTHMA ON FAMILY RELATIONS ? #

- | | |
|--------------------|----------------------|
| 1- NO EFFECT | 2- INCREASE SYMPATHY |
| 3- CAUSES TROUBLES | 4- OTHER SPECIFY |

IS THE CHILD SUFFERING FROM NOCTURNAL ENURESIS ? <Y> IF YES :

- | | |
|--|-----------|
| 1- NO. OF BED WETTING ## / W , IF LESS THAN ONCE/W.NO./MONTH = # | |
| 3- DOES NOT KNOW | 4- OTHERS |

IS THERE PERSONS IN THE FLAT SMOKING ? <Y> IF YES : HOW MANY? NO. #

- | | | |
|-----------------|-----------------|---------|
| DO THEY SMOKE # | | |
| 1- INSIDE FLAT | 2- OUTSIDE FLAT | 3- BOTH |

WHO ARE THOSE PERSONS ? #

- | | |
|------------|------------------|
| 1- FATHER | 2- MOTHER |
| 3- BROTHER | 3- OTHER SPECIFY |

IF THE MOTHER WAS SMOKING, DID SHE SMOKE DURING PREGNANCY ? <Y>

IS THE CHILD SUFFERING FROM OTHER KINDS OF ALLERGY ? <Y> IF YES : #

- | | |
|-----------------|-------------------|
| 1- SKIN ALLERGY | 2- FOOD ALLERGY |
| 3- DRUG ALLERGY | 4- OTHERS SPECIFY |

IS THERE FAMILY HISTORY OF ASTHMA ? <Y> IF YES . WHOM ? #

- | | |
|-------------|-------------------|
| 1- FATHER | 2- MOTHER |
| 3- SIBLINGS | 4- OTHERS SPECIFY |

IS THERE FAMILY HISTORY OF ALLERGY ? <Y> IF YES . WHOM ? #

- | | |
|-------------|-------------------|
| 1- FATHER | 2- MOTHER |
| 3- SIBLINGS | 4- OTHERS SPECIFY |

WHAT KIND OF ALLERGY ? #

- | | |
|-----------------|-------------------|
| 1- SKIN ALLERGY | 2- FOOD ALLERGY |
| 3- DRUG ALLERGY | 4- OTHERS SPECIFY |

HOW DID YOU KNOW THAT THE CHILD IS SUFFERING FROM ASTHMA ? #

- | | |
|--------------|-------------------|
| 1- PHYSICIAN | 2- OTHERS SPECIFY |
|--------------|-------------------|

WHAT WAS THE AGE OF THE CHILD DURING THE FIRST ATTACK DIAGNOSED BY PHYSICIAN ?

- | | |
|----------------------|-----------|
| 1- AGE = ##.## YEARS | 3- OTHERS |
| 2- DOES NOT REMEMBER | |

WAS THE CHILD HOSPITALISED DUE TO CHEST INFECTION DURING FIRST YEAR LIFE ? <Y>

HOW MANY ASTHMA ATTACKS THE CHILD HAD DURING LAST YEAR ? 1- NO. ## AT WHERE DID YOU TREAT THE CHILD FROM ASTHMA DURING LAST YEAR . WHAT WAS THE COSTS ?

- | | | | | | | |
|------------|-----|-----------|----|------|-----|--------|
| HOSPITALS | <Y> | FREQUENCY | ## | COST | ### | POUNDS |
| PHYSICIANS | <Y> | FREQUENCY | ## | COST | ### | POUNDS |
| HOME | <Y> | FREQUENCY | ## | COST | ### | POUNDS |

WAS THE CHILD HOSPITALISED DURING LAST YEAR DUE TO ASTHMA ? <Y> IF YES HOW MANY HOSPITALISATIONS ? AND ITS COSTS ?

- | | | | | |
|---------------------------|----|------|-----|--------|
| NO. OF HOSPITALISATIONS : | ## | COST | ### | POUNDS |
|---------------------------|----|------|-----|--------|

IN YOUR OPINION WHAT ARE THE CAUSES WHICH INITIATE ASTHMA ATTACK ?

- | | |
|--------------|---------------------|
| 1- INFECTION | 2- EXPOSURE TO COLD |
|--------------|---------------------|

- 3- EXPOSURE TO DUST 4- PHYSICAL EXERCISE
 5- EXPOSURE TO SMOKE 6- FAMILY TROUBLES
 7- DOES NOT KNOW 8- OTHERS SPECIFY

NO. #, #, #, #, #, # WERE MENTIONED.

WHAT DO YOU USUALLY DO TO PREVENT THE DEVELOPMENT OF ASTHMA ATTACK ?

- 1- AVOID CAUSES 2- USE DRUGS
 3- DO NOTHING 4- OTHERS SPECIFY

NO. #, # WERE MENTIONED

IF YOU USE DRUGS TO PREVENT THE DEVELOPMENT OF ATTACK , WHAT DRUGS DO YOU USE REGULARLY ? AND ITS COST ?

| DRUG | FORM | COST/MONTH |
|-------|-------|------------|
| _____ | _____ | ### POUN |
| _____ | _____ | ### POUN |
| _____ | _____ | ### POUN |

WHAT ARE THE EARLY WARNING SIGNS FOR ASTHMA ATTACK ?

- 1- COUGH 2- RAPID BREATHING
 3- NASAL SYMPTOMS 4- IRRITABILITY
 5- WHEEZING 6- DIFFICULT BREATHING
 7- DOES NOT KNOW 8- TEARING, PALE FACE
 9- OTHERS

NO. #, #, #, # WERE MENTIONED

WHAT DO YOU USUALLY DO WHEN YOU FEEL THAT ASTHMA ATTACK WILL START ?

- 1- USE DRUGS 2- GO TO PHYSICIAN
 3- DO NOTHING 4- GIVE WARM FLUID
 5- CALM CHILD 6- OTHERS SPECIFY

NO. #, #, #, # WERE MENTIONED

IF YOU USE DRUGS AT THE START OF THE ATTACK , WHAT ARE THESE DRUGS AND ITS COST ?

| DRUG | FORM | COST/ATTACK |
|-------|-------|-------------|
| _____ | _____ | ### POUN |
| _____ | _____ | ### POUN |
| _____ | _____ | ### POUN |
| _____ | _____ | ### POUN |

WHAT ARE THE SYMPTOMS WHICH OCCUR DURING AN ASTHMA ATTACK ?

- 1- COUGH 2- WHEEZING
 3- RAPID BREATHING 4- DIFFICULT BREATHING
 5- GRUNTING 6- WORKING ALAE NASAE
 7- SUPRA STERNAL RETRACTION 8- INTER COSTAL RETRACTION
 9- SUBCOSTAL RETRACTION 10- HEADACHE
 11- CHANGE OF BEHAVIOR 12- IRRITABILITY
 13- WEAKNESS 14- CYANOSIS
 15- DOES NOT KNOW 16- OTHERS SPECIFY

NO. #, #, #, #, ##, ##, ##, ## WERE MENTIONED

WHAT DO YOU USUALLY DO DURING AN ASTHMA ATTACK ?

- 1- USE DRUGS 2- GO TO PHYSICIAN
 3- GO TO HOSPITAL 4- OTHERS SPECIFY

NO. #, # WERE MENTIONED

IF YOU USE DRUGS DURING AN ASTHMA ATTACK . WHAT ARE THESE DRUGS AND ITS COST ?

| DRUGS | FORM | COST/ATTACK |
|-------|-------|-------------|
| _____ | _____ | ### POUND |
| _____ | _____ | ### POUND |
| _____ | _____ | ### POUND |
| _____ | _____ | ### POUND |

SHOW ME HOW YOU CAN GIVE THE DRUGS ?

FORM OF DRUG CORRECT METHOD OF ADM. CORRECT FREQ. CORRECT DURATION

_____ : <Y> : <Y> : <Y>
 _____ : <Y> : <Y> : <Y>

IN YOUR OPINION WHAT HAPPENS INSIDE THE CHEST OF THE CHILD WHEN ASTHMA OCCURS ?

- | | |
|----------------------------|--------------------|
| 1- BRONCHOCONSTRICTION | 2- MUCOSAL OEDEMA |
| 3- VISCID MUCOUS SECRETION | 4- HYPERREACTIVITY |
| 5- DOES NOT KNOW | 6- OTHERS SPECIFY |

NO. #, #, #, # WERE MENTIONED

IN YOUR OPINION WHAT ARE THE ROLES OF DRUGS IN TREATING ASTHMA ?

- | | |
|-------------------|----------------------|
| 1- BRONCHODILATOR | 2- ANTI INFLAMMATORY |
| 3- DOES NOT KNOW | 4- OTHERS SPECIFY |

NO. #, # WERE MENTIONED

DO YOU THINK THAT DRUG TREATMENT OF ASTHMA IS USEFUL ?

- | | |
|--------------|------------------|
| 1- YES | 2- NO |
| 3- SOMETIMES | 4- DOES NOT KNOW |

NO. # WAS MENTIONED

IN BETWEEN THE ATTACK THE CHILD FEELS :

- | | |
|-----------------------|-----------------------------|
| 1- RELAXED | 2- WORRIER ABOUT THE ATTACK |
| 3- WORRIED ABOUT SELF | 3- FRIGHTENED |
| 4- AFRAID OF DYING | 5- SCARED |
| 5- DOES NOT KNOW | 6- OTHERS |

NO. #, # WERE MENTIONED

MOST COMMONLY . ASTHMA ATTACKS THE CHILD DURING :

- | | |
|---------------------------|-----------|
| 1- WINTER | 2- SPRING |
| 3- SUMMER | 4- AUTUMN |
| 6- ALL ROUND YEAR EQUALLY | 7- OTHERS |

NO. # WAS MENTIONED

THE ATTACKS MOST COMMONLY START DURING :

- | | |
|--------------------------|-----------|
| 1- NIGHT | 2- DAY |
| 3- DAY AND NIGHT EQUALLY | 4- OTHERS |

NO. # WAS MENTIONED

DO YOU RESTRICT CHILD'S ACTIVITY BECAUSE OF ASTHMA ? <Y> IF YES :

HOW MANY DAYS DID YOU RESTRICT CHILD'S ACTIVITY DUE TO ASTHMA LAST YEAR ? ### DAYS (NOT GOING OUTSIDE THE FLAT)

DID YOU PREVENT THE CHILD FROM GOING TO SCHOOL DUE TO ASTHMA? <Y> IF YES

HOW MANY DAYS THE CHILD WAS ABSENT FROM SCHOOL DUE TO ASTHMA LAST YEAR

- | | |
|---------------------|----------------------|
| 1- NO. OF DAYS = ## | 2- WAS NOT IN SCHOOL |
|---------------------|----------------------|

3- DOES NOT REMEMBER
NO. # WAS MENTIONED

4- OTHERS

DO YOU RESTRICT CERTAIN TYPES OF FOOD FROM CHILD'S DIET ? <Y> IF YES

WHAT TYPES OF FOOD DO YOU RESTRICT ?

1- EGG

2- MILK

2- FISH

4- STRAWBERRIES

5- BANANA

6- OTHERS SPECIFY

NO. #, #, #, # WERE MENTIONED

WHY DO YOU RESTRICT THESE FOODS ?

1- PHYSICIAN INSTRUCTIONS

2- IT CAUSES THE ATTACK

3- IT CAUSES ALLERGY

4- OTHERS

NO. # WAS MENTIONED

DO YOU USE THESE FOODS IN PREPARING OTHER MEALS EATEN BY THE CHI
(FOR EXAMPLE :CAKE) ? <Y> IF YES :

WAS ASTHMA ATTACK DEVELOPED AFTER EATING THESE FOODS (WITHIN
HOURS) ? <Y>

QUESTIONS RELATED TO THE ENVIRONMENT :

CROWDING INDEX (NO. OF PERSONS ##/NO. OF ROOMS #) = #.# /ROOM

SPECIFY THE TYPE OF BEDDING (MATTRESS, PILLOWS) USED FOR CHILD'S SLEEPING

1- FOAM

2- COTTON

3- FEATHERS

4- RAGS

5- GRASS OR STRAW

6- OTHERS

NO. # WAS MENTIONED

THE FLOOR OF THE FLAT IS COVERED WITH :

1- CARPETS

2- MOCKET

3- CEMENT

4- TERRAZZO

5- KANALTEX

6- OTHERS

NO. # WAS MENTIONED

HOW DO YOU CLEAN THE GROUND ?

1- MANUAL CLEANING WITH SWEEPER 2- ELECTRIC VACCUME CLEANER

NO. # WAS MENTIONED

THE ROOF OF THE FLAT IS MADE OF :

1- CONCRETE

2- WOOD

3- STRAW

4- OTHERS

NO. # WAS MENTIONED

IS THE ROOM WHERE THE CHILD SLEEP USED FOR OTHER PURPOSES ? <Y> IF YES

WHAT PURPOSES ?

1- COOKING

2- SMOKING

3- STORE

3- OTHERS

NO. # WAS MENTIONED

WHAT KIND OF FUEL IS USED FOR COOKING ?

1- KEROSENE

2- BUTAGAS

3- FIREWOOD

4- CORN STALK

5- RICE STALK

5- OTHERS

NO. # WAS MENTIONED

WHAT KIND OF FUEL IS USED FOR HEATING ?

- 1- CROP RESIDUE
- 2- COAL
- 3- ELECTRIC
- 4- GAS
- 5- WOOD
- 6- NONE

NO. # WAS MENTIONED

ARE RATS OR COCKROACHES PRESENT IN YOUR HOUSE ? <Y>
 DO YOU USE SPRAYS AS AN INSECTICIDES OR FOR SPRAYING CROPS ? <Y>
 DO YOU HAVE PLANTS INSIDE YOUR HOUSE ? <Y>
 ARE DOGS OR CATS OR BIRDS OR OTHER DOMESTIC ANIMALS PRESENT N YOUR HOU
 OR KEPT BY YOUR FAMILY ? <Y> IF YES COMPLETE THE FOLLOWING :

| TYPE OF ANIMAL | NUMBER | CHILD IN CLOSE CONTACT TO ANIMA |
|----------------|--------|---------------------------------|
| _____ | # | <Y> |
| _____ | # | <Y> |
| _____ | # | <Y> |

DO YOU HAVE A REFRIGERATOR ? <Y>
 DO YOU HAVE A TELEVISION ? <Y> IF YES , IS IT COLOURED <Y>
 DO YOU HAVE AN ELECTIC WASHER ? <Y> IF YES , IS IT FULL AUTOMATIC ? <Y>

INVESTIGATIONS : (FROM MEDICAL RECORDS)

- 1- COMPLETE BLOOD PICTURE
- 2- SEROLOGICAL EXAM.
- 3- SPUTUM EXAM
- 4- SPIROMETRY
- 5- CHEST X RAY
- 6- THROAT EXAM.
- 7- SENSITIVITY TESTS
- 8- IMMUNOTHERAPY

NO. #.#,#.#,#.# WERE DONE

PEAK EXPIRATORY FLOW RATE = ###.# LITRE/MINUTE

DEGREE OF ASTHMA :

- 1- MILD
- 2- MODERATE
- 3- SEVERE

NO. # WAS MENTIONED

TYPE OF ASTHMA :

- 1- EXTRENSIC
- 2- INTRENSIC
- 3- MIXED
- 4- UNSURE

NO. # WAS MENTIONED

- 1 - FATHER
- 2 - MOTHER
- 3 - BROTHERS
- 4 - GRANDFATHERS
- 5 - UNCLES
- 6 - OTHERS

DID THE CHILD HAD ASTHMA ATTACKS SINCE FIRST INTERVIEW IN <dd/mm/yy>?

<A> IF YES , HOW MANY ? NO. ##

WHERE DID YOU TREAT THE CHILD FROM ASTHMA SINCE FIRST INTERVIEW AND WHAT WAS THE COSTS ?

| | | | | | | |
|------------|-----|-----------|----|------|-----|--------|
| HOSPITALS | <A> | FREQUENCY | ## | COST | ### | POUNDS |
| PHYSICIANS | <A> | FREQUENCY | ## | COST | ### | POUNDS |
| HOME | <A> | FREQUENCY | ## | COST | ### | POUNDS |

WAS THE CHILD HOSPITALIZED DUE TO ASTHMA SINCE FIRST INTERVIEW ? <A>

IF YES , HOW MANY HOSPITALIZATIONS AND ITS COSTS ?

NUMBER OF HOSPITALIZATIONS ## ITS COSTS ### POUNDS

IN YOUR OPINION WHAT ARE THE CAUSES WHICH INITIATE ASTHMA ATTACKS ?

- 1 - INFECTION
- 2 - EXPOSURE TO COLD (BATHING)
- 3 - EXPOSURE TO DUST
- 4 - PHYSICAL EXERCISE
- 5 - EXPOSURE TO SMOKE
- 6 - FAMILY TROUBLES
- 7 - EXAGGERATED CHILD EMOTION
- 8 - EATING SOME TYPES OF FOOD
- 9 - HOUSE DUST MITE
- 10 - WITHOUT APPARENT CAUSE
- 11 - CUTTING HIS HAIR
- 12 - OTHERS

NUMBER # # # # # # ## ## WERE MENTIONED

WHAT DO YOU USUALLY DO TO PREVENT THE DEVELOPMENT OF ASTHMA ATTACK ?

- 1 - AVOID CAUSES
- 2 - USE DRUGS
- 3 - GIVE WARM FLUIDS
- 4 - MAKE CHILD BREATHE FRESH AIR
- 5 - DO NOTHING
- 6 - OTHERS

NUMBER # # # WERE MENTIONED

IF YOU USE DRUGS TO PREVENT THE DEVELOPMENT OF ATTACK . WHAT DRUGS DO YOU USE REGULARLY ? AND ITS COSTS ?

| DRUGS | FORM | COST/MONTH |
|-------|------|------------|
| <A> | <A> | ### POUNDS |
| <A> | <A> | ### POUNDS |
| <A> | <A> | ### POUNDS |

WHAT ARE THE EARLY WARNING SIGNS FOR ASTHMA ATTACK ?

- 1 - COUGH
- 2 - RAPID BREATHING
- 3 - NASAL SYMPTOMS
- 4 - WHEEZING
- 5 - STOP FEEDING.DRINKING
- 6 - PALE FACE
- 7 - TEARING
- 8 - INCREASED SALIVATION OR SPUTUM
- 9 - TENDENCY TO VOMIT
- 10 - GENERAL WEAKNESS
- 11 - SWEATING
- 12 - BLUISH LIPS OR FACE
- 13 - SUNKEN EYES
- 14 - FEVER
- 15 - PALE LIPS
- 16 - SNEEZING
- 17 - OTHERS

NUMBER # # # # # # ## ## ## ## ## WERE MENTIONED

WHAT DO YOU USUALLY DO WHEN YOU FEEL THAT ASTHMA ATTACK WILL START ?

- 1 - USE DRUGS
- 2 - GO TO PHYSICIAN
- 3 - GO TO HOSPITAL
- 4 - GIVE WARM FLUIDS
- 5 - CALM CHILD
- 6 - MAKE CHILD IN REST

7 - OTHER SPECIFY

NUMBER # # # # # # WERE MENTIONED

IF YOU USE DRUGS AT THE START OF THE ATTACK , WHAT ARE THESE DRUGS AND ITS COSTS ?

| DRUGS | FORM | COST/MONTH |
|-------|------|------------|
| <A > | <A > | ### POUNDS |
| <A > | <A > | ### POUNDS |
| <A > | <A > | ### POUNDS |

WHAT ARE THE SYMPTOMS WHICH OCCUR DURING AN ASTHMA ATTACK ?

- | | |
|-----------------------------|-------------------------------------|
| 1 - COUGH | 2 - WHEEZING |
| 3 - RAPID BREATHING | 4 - DIFFICULT BREATHING |
| 5 - GRUNTING | 6 - WORKING ALAE NASAE |
| 7 - SUPRASTERNAL RETRACTION | 8 - INTERCOSTAL RETRACTION |
| 9 - SUBCOSTAL RETRACTION | 10 - HEADACHE |
| 11 - CHANGE OF BEHAVIOR | 12 - IRRITABILITY |
| 13 - WEAKNESS | 14 - BLUISH FACE |
| 15 - SWEATING | 16 - ABDOMINAL PAIN |
| 17 - VOMITING | 18 - INCREASED SALIVATION OR SPUTUM |
| 19 - COLD EXTREMITIES | 20 - FEVER |
| 21 - TREMORS | 22 - DO NOT KNOW |
| 23 - OTHER SPECIFY | |

NUMBERS # # # # # # # ## ## ## ## ## ## ## WERE MENTIONED

WHAT DO YOU USUALLY DO DURING ASTHMA ATTACK ?

- | | |
|---------------------|--------------------|
| 1 - USE DRUGS | 2 - GO TO HOSPITAL |
| 3 - GO TO PHYSICIAN | 4 - DO NOTHING |
| 5 - OTHERS SPECIFY | |

NUMBERS # # # WERE MENTIONED

IF YOU USE DRUGS DURING AN ASTHMA ATTACK , WHAT ARE THESE DRUGS AND ITS COSTS ?

| DRUGS | FORM | COST/MONTH |
|-------|------|------------|
| <A > | <A > | ### POUNDS |
| <A > | <A > | ### POUNDS |
| <A > | <A > | ### POUNDS |

DO YOU THINK THAT DRUG TREATMENT OF ASTHMA IS USEFUL ?

- | | |
|---------------|-----------------|
| 1 - YES | 2 - NO |
| 3 - SOMETIMES | 4 - DO NOT KNOW |

NUMBER # WAS MENTIONED

IN BETWEEN THE ATTACKS . THE CHILD FEELS :

- | | |
|--------------------------|------------------------------|
| 1 - DOES NOT WORRY | 2 - WORRIED ABOUT THE ATTACK |
| 3 - AFRAID OF THE ATTACK | 4 - DO NOT KNOW |
| 5 - OTHERS SPECIFY | |

NUMBER # # # WERE MENTIONED

IN BETWEEN THE ATTACKS . THE FAMILY FEELS :

- | | |
|--------------------------|------------------------------|
| 1 - DOES NOT WORRY | 2 - WORRIED ABOUT THE ATTACK |
| 3 - AFRAID OF THE ATTACK | 4 - WORRIED ABOUT THE CHILD |
| 5 - SENSE OF FEAR | 6 - OTHERS SPECIFY |

NUMBER # # # WERE MENTIONED

THE ATTACKS MOST COMMONLY START DURING :

- | | |
|---------------------------|--------------------|
| 1 - NIGHT | 2 - DAY |
| 3 - DAY AND NIGHT EQUALLY | 4 - OTHERS SPECIFY |
- NUMBER # WERE MENTIONED

DID YOU RESTRICT CHILD'S ACTIVITY BECAUSE OF ASTHMA SINCE THE INTERVIEW IN <dd/mm/yy> ? <A> IF YES :

HOW MANY DAYS DID YOU RESRICT CHILD'S ACTIVITY SINCE THIS INTERVIEW ?

NUMBER OF DAYS = ## DAYS (NOT GOING OUTSIDE THE FLAT)

DOES THE CHILD GO TO SCHOOL ? <A> IF YES :

DID YOU PREVENT THE CHILD FROM GOING TO SCHOOL DUE TO ASTHMA SINCE THE INTERVIEW IN <dd/mm/yy> ? <A> IF YES :

HOW MANY DAYS THE CHILD WAS ABSENT FROM SCHOOL DUE TO ASTHMA SINCE THIS INTERVIEW ?

1 - NUMBER OF DAYS = ## DAYS 2 - DOES NOT REMEMBER

NUMBER # WAS MENTIONED

DO YOU RESTRICT CERTAIN TYPES OF FOOD FROM CHILD'S DIET ? <A> IF YES

WHAT TYPES OF FOOD DO YOU RESTRICT ?

- | | |
|-------------------------|------------------|
| 1 - EGG | 2 - MILK |
| 3 - FISH | 4 - STRAWBERRIES |
| 5 - BANANA | 6 - MANGO |
| 7 - COLD DRINKS OR FOOD | 8 - LENTILS |
| 9 - CHOCOLATE | 10 - LIVER |
| 11 - OTHERS SPECIFY | |

NUMBERS # # # # # # ## WERE MENTIONED

WHY DO YOU RESTRICT THESE FOODS ?

- | | |
|------------------------------|---------------------------------|
| 1 - PHYSICIAN'S INSTRUCTIONS | 2 - IT CAUSES THE ASTHMA ATTACK |
| 3 - IT CAUSES SKIN ALLERGY | 4 - OTHERS SPECIFY |

NUMBERS # # WERE MENTIONED

DO YOU USE THESE FOODS IN PREPARING OTHER MEALS EATEN BY THE CHILD (FOR EXAMPLE CAKE) ? <A> IF YES :

WAS ASTHMA ATTACK DEVELOPED AFTER EATING THESE FOODS (WITHIN 24 HOURS) ? <A>

QUESTIONS RELATED TO THE ENVIRONMENT :

THE FLOOR OF THE CHILD'S ROOM IS COVERED WITH :

- | | |
|--------------------|--------------|
| 1 - CARPET | 2 - MOCKET |
| 3 - CEMENT | 4 - TERRAZZO |
| 5 - KANALTEX | 6 - HASSER |
| 7 - OTHERS SPECIFY | |

NUMBER # # WERE MENTIONED

HOW DO YOU CLEAN THE GROUND ?

- | | |
|----------------------------------|------------------------------|
| 1 - MANUAL CLEANING WITH SWEEPER | 2 - ELECTRIC VACCUME CLEANER |
|----------------------------------|------------------------------|

NUMBER # WAS MENTIONED

IF YOU CLEAN USING MANUAL CLEANING WITH SWEEPER . DO YOU CLEAN THE GROUND IN PRESENCE OF THE CHILD ? <A>

IS THE ROOM WHERE THE CHILD SLEEP USED FOR OTHER PURPOSES ? <A> IF

YES : WHAT PURPOSES ?

- | | |
|----------------------------|-----------------------------------|
| 1 - COOKING | 2 - SMOKING |
| 3 - STORE OF OLD FURNITURE | 4 - STORE OF OLD PAPERS AND BOOKS |
| 5 - OTHERS SPECIFY | |

NUMBER # # WERE MENTIONED

ARE RATS OR COCKROACHES PRESENT IN YOUR FLAT ? <A>

DO YOU USE SPRAYS AS AN INSECTICIDES ? <A>

DO YOU HAVE PLANTS INSIDE YOUR FLAT ? <A>

ARE DOGS OR CATS OR BIRDS OR OTHER DOMESTIC ANIMALS PRESENT IN YOUR FLAT OR KEPT BY YOUR FAMILY ? <A> IF YES , COMPLETE THE FOLLOWING :

| TYPE OF ANIMAL | NUMBER | CHILD IN CLOSE CONTACT TO ANIMAL? |
|----------------|--------|-----------------------------------|
| <A > | # | <A> |
| <A > | # | <A> |
| <A > | # | <A> |

WHAT IS THE PERCENT OF EXPENSES ON ASTHMA (INCLUDING DRUGS , PHYSICIAN VISITS , TRANSPORTS AND OTHERS) TO THE FAMILY MONTHLY INCOME ?

ANNUAL AVERAGE EXPENSES = ## % OF THE TOTAL FAMILY INCOME

INVESTIGATIONS :

- | | |
|----------------------------|----------------------------|
| 1 - CLINICAL EXAMINATION | 2 - COMPLETE BLOOD PICTURE |
| 3 - SENSITIVITY TESTS | 4 - IMMUNOTHERAPY |
| 5 - SPUTUM EXAM. | 6 - CHEST X RAY |
| 7 - STOOL & URINE ANALYSIS | 8 - OTHERS SPECIFY |

NUMBERS # # # # # # # # WERE MENTIONED

SUBJECTIVE ASSESSMENT OF THE DEGREE OF ASTHMA : #

MEASURED ACCORDING TO USE OF MEDICATIONS , NUMBER OF ACUTE ATTACKS , NUMBER OF DAYS OF RESTRICTED ACTIVITIES OR SCHOOL ABSENCE DURING LAST YEAR.

- | | |
|------------|--------------|
| 1 - MILD | 2 - MODERATE |
| 3 - SEVERE | 4 - OTHERS |

SCALE FOR MEASUREMENT OF PERCEIVED DAY TIME SYMPTOMS (QUESTIONS FROM 1 TO 4) , PERCEIVED DISABILITY (QUESTIONS FROM 5 TO 12) . PERCEIVED NOCTURNAL SYMPTOMS (QUESTIONS FROM 13 TO 15) . QUOTED FROM : T.P.USHERWOOD , A.SCRIMGEOUR AND J.H.BARBER : QUESTIONNAIRE TO MEASURE PERCEIVED SYMPTOMS AND DISABILITY IN ASTHMA. ARCH. DIS. CHILD. 1990,65 :779-881

MEASUREMENT OF PERCEIVED FAMILY DISABILITY : THE INFORMER IS #

- | | |
|--------------------|------------------|
| 1 - FATHER | 2 - MOTHER |
| 3 - BROTHER | 4 - SISTER |
| 5 - GRAND FATHER | 6 - GRAND MOTHER |
| 7 - OTHERS SPECIFY | |

EACH QUESTION IS PRECEDED BY THE SENTENCE : SINCE THE INTERVIEW IN <dd/mm/yy> . THE QUESTIONS ARE ANSWERED BY ONE OF THE FOLLOWING :

- | | |
|----------------|----------------|
| 0 - NOT AT ALL | 1 - A FEW DAYS |
| 2 - SOME DAYS | 3 - MOST DAYS |

4 - EVERY DAY
 THE HIGHER THE SCORES , THE GREATER THE PERCEIVED SYMPTOMS AND
 DISABILITIES .

QUESTIONS

ANSWERS

A - DAY TIME SYMPTOMS SCORES :

- 1 - YOUR CHILD HAS BEEN WHEEZY DURNG THE DAY #
- 2 - YOUR CHILD HAS BEEN COUGHED DURING THE DAY #
- 3 - YOUR CHILD HAS COMPLAINED OF BEING SHORT OF BREATH #
- 4 - EXERTION (EG. RUNNING) HAS MADE YOUR CHILD BREATHLESS #
- ** TOTAL DAY TIME SYMPTOM SCORES = ## OUT OF 16, THE PERCENT = ##.##%

B - DISABILITIES SCORES :

- 5 - YOUR CHILD HAS STAYED INDOORS BECAUSE OF WHEEZING OR
 COUGHING #
- 6 - HIS/HER ASTHMA HAS STOPPED YOUR CHILD FROM PLAYING WITH
 HIS/HER FRIENDS #
- 7 - DURING TERM TIME , YOUR CHILD'S EDUCATION HAS SUFFERED
 DUE TO HIS/HER ASTHMA #
- 8 - ASTHMA HAS STOPPED YOUR CHILD FROM DOING ALL THE THINGS
 THAT A BOY OR GIRL SHOULD DO AT HIS/HER AGE #
- 9 - YOUR CHILD'S ASTHMA HAS INTERFERRED WITH HIS/HER LIFE #
- 10 - ASTHMA HAS LIMITED YOUR CHILD'S ACTIVITY #
- 11 - YOUR CHILD'S ASTHMA HAS LIMITED YOUR ACTIVITY #
- 12 - YOU HAVE HAD TO MAKE ADJUSTMENTS TO FAMILY LIFE
 BECAUSE OF YOUR CHILD'S ASTHMA #
- ** TOTAL DISABILITY SCORES = ## OUT OF 32, THE PERCENT = ##.##%

C - NOCTURNAL SYMPTOMS SCORES :

- 13 - YOUR CHILD HAS COUGHED AT NIGHT #
- 14 - YOUR CHILD'S SLEEP HAS BEEN DISTURBED BY WHEEZING
 OR COUGHING #
- 15 - YOUR CHILD HAS BEEN WAKEN UP BY WHEEZING OR COUGHING #
- ** TOTAL NOCTURNAL SYMPTOMS SCORES = ## OUT OF 12,THE PERCENT= ##.##%

** TOTAL SCORES OF PERCEIVED SYMPTOMS AND DISABILITIES = ## OUT OF 60
 THE PERCENT = ##.## %

*** THE DEGREE OF PERCEIVED SYMPTOMS AND DISABILITIES OF THE CHILD'S

ASTHMA = #

- 1 - NO PERCEIVED SYMPTOMS OR DISABILITIES 0 %
- 2 - MILD PERCEIVED SYMPTOMS AND DISABILITIES 0-33 %
- 3 - MODERATE PERCEIVED SYMPTOMS AND DISABILITIES 34-66 %
- 4 - SEVERE PERCEIVED SYMPTOMS AND DISABILITIES 67-100%
- 5 - OTHERS NOT ASSESSED

ARABIC SUMMARY

ملخص الرسالة

الربو الشعبي مرض من أمراض الجهاز التنفسي يتميز بانتسداد متكرر أو مزمن و بطريقة عكسية للشعب الهوائية، بالعلاج الامثل لا يؤدي الى ضرر دائم بالرئة ولا يجب أن يحد من الانشطة المعتادة للطفل في معظم الاطفال.

في الاطفال في المستويات العمرية من ٦ الى ١١ سنة ازداد انتشار الازمة من ٨ , ٤% في أواخر السبعينات الى ٨ , ٧% في الولايات المتحدة الامريكية. في مصر يصيب الربو الشعبي ٢ , ٨% تقريبا من الاطفال من سن ٣ الى ١٤ سنة .

الهدف من الدراسة هو زيادة المعلومات عن الازمات الربوية و علاجاتها في الاطفال المصابين بالربو الشعبي و عائلاتهم , تحسين الممارسة فيما يختص بالوقاية والعلاج،التقليل من شدة الاصابة،تحسين البيئة (الأجواء) المنزلية المحيطة للاطفال المصابين بالربو وازدياد ممارستهم (اشتراكهم) في الانشطة المعتادة.

شمل البرنامج ٧٦ طفلا مصابا بالربو الشعبي من سن ٥ الى ١٢ سنة مع عائلاتهم منهم ٤٣ طفل يمثلون ٥٦% و ٣٣ طفلة يمثلون ٤٤% .

تم تنفيذ البرنامج بالعيادة الخارجية للحساسية بمستشفى أبو الريش للأطفال بجامعة القاهرة بداية من مارس ١٩٩٢ وحتى ديسمبر ١٩٩٣ على أساس عينة عشوائية مقسمة حسب شدة الازمة، ثم متابعة العائلات لمدة سنة. الذين اكملوا المتابعة من العائلات وصلوا الى ٥٨ عائلة (٧٦%)، منهم ٢٨ عائلة في المجموعة التجريبية ، و ٣٠ في المجموعة الضابطة.

تم تحديد شدة الازمة بعد المقابلة الاولى باستخدام القياس الطبى و قياس أعلى قوة اندفاع زفيرى باستخدام جهاز خاص .

المجموعة التجريبية تم مقابلتهم لمدة ساعة كل أسبوعين لمدة عام.

أما التعليمات فشملت العوامل المسببة للأزمة، العلامات والأعراض فى بداية الأزمة، كيفية التعامل مع الطفل فى بداية الأزمة، ما يجب عمله أثناء حدوث الازمة، ومتى يجب التوجه الى المستشفى.

معلومات بصدد البيئة المحيطة بالأطفال و العلاقات الأسرية تم جمعها من خلال استبيان تم مراجعته فى بداية البرنامج. استبيان آخر تم تصميمه للمتابعة لقياس تأثير التعليمات و الارشادات بصورة منتظمة. خلال الجلسات تم مناقشة أسئلة الأسرة و تصحيح المفاهيم الخاطئة، و تم تعليم الأسرة ارشادات ونصائح مكتوبة و تم اطلاعهم على رسومات توضيحية وتم قياس أعلى اندفاع زفيرى.

تحليل البيانات أوضح تحسن ملحوظ فى المعلومات وممارسة الأسرة لمواجهة الأزمة وانخفاض ملحوظ فى شدة الأزمة، وكذلك زيادة ممارسة الرياضة وتقليل عدد أيام الغياب من المدرسة والذي ادى الى تحسن فى الأداء المدرسى. البيئة المنزلية تم تحسينها فيما يتعلق بتخفيض عدد المدخنين داخل الشقة. كما ادى الى ازالة العوامل المهيجة للازمات مثل الحيوانات و الطيور المنزلية .

نخلص الى أنه يجب تعميم هذه البرامج التعليمية للأطفال المصابين بالربو الشعبي و عائلاتهم
لما لها من أثر فعال على شدة الأزمة,المعلومات,الممارسة وتحسين الأجواء المنزلية لعائلات
الأطفال المصابين بالربو الشعبي.

بيان بحالة الباحث

الاسم : الجواد احمد الهه عبدالرحيم نزيه

مسمى : دراسات طب

موضوع الرسالة : برنامج تعليمي وتثقيفي لعدوى الدفتار الصائغ بالربو العبد ر

للحصول على درجة : الدكتوراه

الوظيفة : اخصائى طب الدفتار

مكان العمل : معهد اجائ طب البدر الحارة. الرية- الهامة للمستعميات والعاقد العقل

الشهادات الحاصل عليها الطالب:

١ - دكتوراه في طب وجرارة

٢ - ماجستير الدراسات العليا للطفولة - جامعة عين شمس

٣ - ماجستير طب الدفتار - جامعة القاهرة

٤ -

تاريخ التسجيل: ٢٩ مارس ١٩٨٩

تاريخ المناقشة: ١٨٩٤ / ٧ / ٢٠

التقدير:

لجنة المناقشة والحكم

وافق الاستاذ الدكتور / نائب رئيس الجامعة لشئون الدراسات العليا

والبحوث بتاريخ ١٤ / ٢ / ١٩٩٤ ، على تشكيل لجنة لمناقشة الطالب /

البحر الجوراء محمد عبد الرقيب نزعلي

من السادة الاساتذة :

- ١ - د. محمد بن محمد علي بن احمد
رئيسا
- ٢ - د. بن محمد بن محمد
عضوا
- ٣ - د. صبيح بن محمد بن محمد
عضوا
- ٤ - د. محمد بن محمد بن محمد
عضوا

جامعة عين شمس
معهد للدراسات العليا للطفولة
القسم الطبى

" برنامج تعليمى و تثقيفى لعلاج الاطفال المصابين بالربو الشعبى و عائلاتهم "

رسالة مقمة من

للطبيب / ابوالمجد احمد عبدالرحيم فرغلى
بكالوريوس الطب و الجراحة
ماجستير للدراسات العليا فى الطفولة

للحصول على درجة الدكتوراه فى الفلسفة

فى

دراسات الطفولة

تحت اشراف

الاستاذة للدكتوراة / أنيسة الحفنى
استاذ طب الاطفال و الحساسية
كلية الطب
جامعة القاهرة

الاستاذة للدكتوراة / سعادى محمد على بهادر
استاذ علم نفس النمو
عميدة معهد الدراسات العليا للطفولة
جامعة عين شمس

الاستاذة للدكتوراة / علوية محمد عبد الباقى
استاذ مساعد دراسات الطفولة الطبية
القسم الطبى
معهد الدراسات العليا للطفولة
جامعة عين شمس

جامعة عين شمس

١٩٩٤

جامعة عين شمس
معهد الدراسات العليا للطفولة
القسم الطبى

" برنامج تعليمى و تثقيفى لعلاج الاطفال المصابين بالربو الشعبى و نائلاتهم "

رسالة مقدمة من

الطبيب / ابوالمجد احمد عبدالرحيم فرغلى
بكالوريوس الطب و الجراحة
ماجستير الدراسات العليا فى الطفولة

للحصول على درجة الدكتوراه فى التلمفة

فى

دراسات الطفولة

تحت اشراف

الاستاذة الدكتورة / أنيسة الحفنى
استاذ طب الاطفال و الحساسية
كلية الطب
جامعة القاهرة

الاستاذة الدكتورة / سعاد محمد على بهادر
استاذ علم نفس النمو
عميدة معهد الدراسات العليا للطفولة
جامعة عين شمس

الاستاذة الدكتورة / علوية محمد عبد الباقى
استاذ مساعد دراسات الطفولة الطبية
القسم الطبى
معهد الدراسات العليا للطفولة
جامعة عين شمس

جامعة عين شمس

١٩٩٤